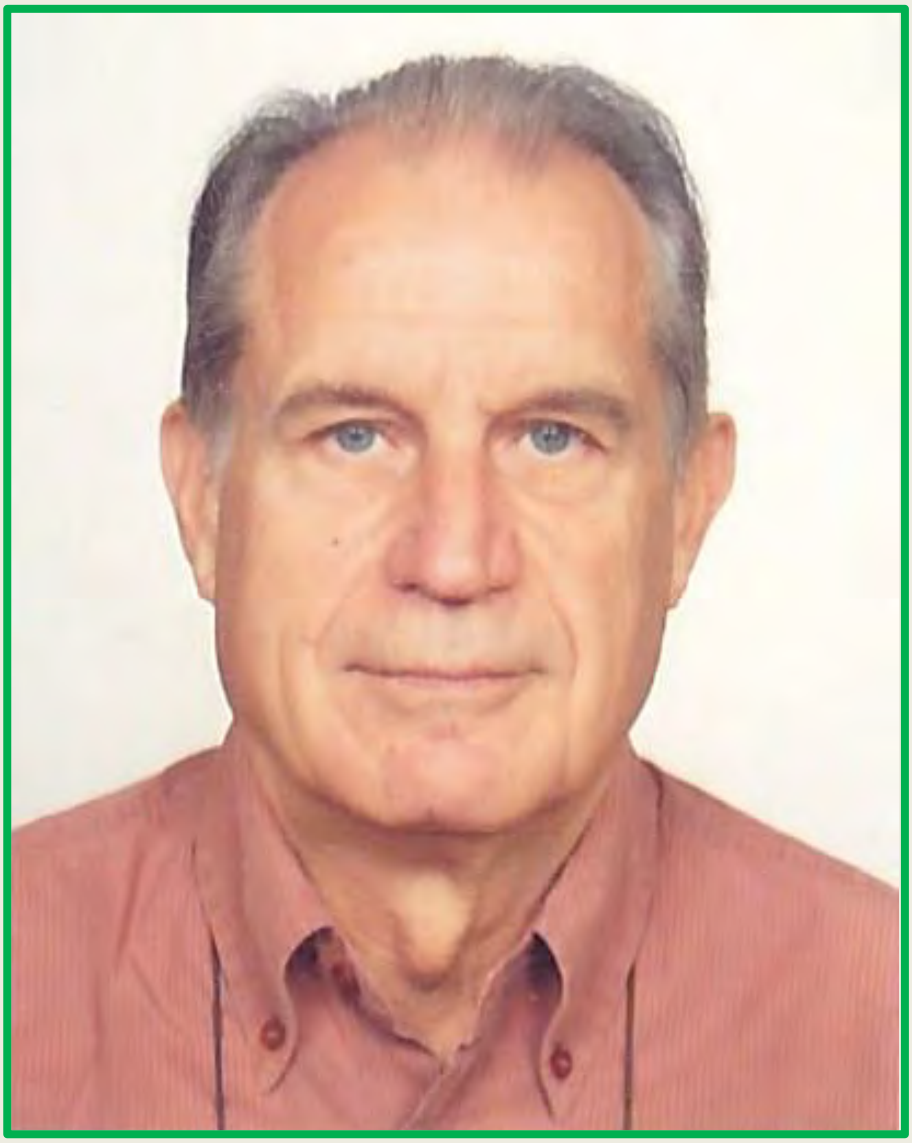
TRANSLATIONAL MOBILITY MEDICINE

Dreams, hopes, frustrations



Ugo Carraro

Preface

I summarized in this book my experiences (good and bad) as a teacher and scientist, who spent more than 60 years at the University of Padua, Italy. Enrolled to medical studies to become a doctor in hotels of my Carraro family in Thermae of Euganean Hills (Padua) Italy, after graduation with honors, I choose the University career, with the approval of my parents and the encouragement of my fiancée and then wife Annalisa Bossi.

At the time, the end of 60s', the enrollments in the Italy university exploded and then the need of teachers made for me very easy to become first an assistant (voluntary and very soon paid) and then an Associate Professor of General Pathology at the University of Padua. Student teaching and exams were very heavy, but more than fifty percent of my activities were devoted to basic research in myology, the study of structural and molecular characteristics of skeletal muscles and their strict dependence to the nervous system for their functions (but not their survival and regeneration, as we will see). The readers will find in the following Chapters, the beginning of my research activities and the serendipitous events of my dedication to denervation-reinnervation of muscles and their electrical stimulation in animal models and later on in patients. After years of basic research I was engaged in human trials to apply those results in human mobility disorders, including those very frequent in aging. Some of my projects were successful, more often frustrating. My contributions have been optical and ultrastructural microscopy and molecular approaches, particularly on isomyosins and other muscle-type markers, but the most important things were attracting bright young collaborators, along with some decisions to transfer to skeletal muscle those approaches which had proven to be useful for clinical cardiology. On the way, other scientists and clinicians (specifically, Physical Medicine and Rehabilitation Specialists) with my own interests contacted me. Some collaborations provided exciting results, the majority frustrations.

Nevertheless, this is the normal ratio in Translational Studies from Basic Science to Medicine: many preliminary exciting results end in failure, in particular those more original and promising. The majority of my dreams ended in disappointments, but I continue to think that it is more than enough to have dreams and the great fortune to test them by rigorous scientific approaches. This is why the book will end, though I will be 80-years-young the February 23, 2023, with a series of my unending dreams.

Here I thank the many young and old persons who inspired, supported and collaborated with me, including the editorial assistants of PAGEPress, the Italian publisher of the European Journal of Translational Myology. Some of the supporters, collaborators and pupils have been kind enough to send me their CVs upon my invitation. But many others I have not been able to contact them or have had very little to share with them other than unpleasant memories.

The list is very long, but I hope that CLEUP will accept a few more pages. I apologies for any missing names. They weren't deliberate omissions.

I'll try to add the names in chronological order, but apologies for any missing names. They weren't deliberate omissions: Margreth A, Salviati G, Catani C, Biral D, Vascon M, Zanella G, Lodolo R, Morale D, Lucke S, Noventa D, Zrunek M, Scabolcs M, Gruber H, Streinzer W, Belluco S, Marchioro L, Mussini I, Favaro G, Caroli M, Pessina AC, Angelini A, Tessari F, Saggin L, Szabolcs M, Streinzer W, Mayr W, Thoma H, Pauletto P, Nascimben L, Piccolo D, Secchiero S, Scannapieco G, Pessina AC, Dal Palù C, Kordowska J, Lotta S, Scelsi R, Alfonsi E, Saitta A, Nicolotti D, Epifani P, Kirillina VP, Borovikov IuS, Szczepanowska J, Velussi C,

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Finally, thanks to CLEUP. I am very happy to publish this book with this Publisher who printed the Proceedings of the first International Scientific Conference that I organized in 1985 in Abano Terme, my hometown, a long time ago.

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Padua (Italy), January 15, 2022.

About the Author


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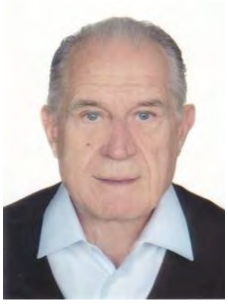
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Prof. Ugo Carraro was born on 23 February 1943 in Abano Terme (Padova), Italy. In 1968, he earned his medical degree from the University of Padua, Italy. He is a Senior Scholar at Padua University and past-Scientific Consultant at IRRCS Fondazione Ospedale San Camillo in Venice-Lido, Italy. Prof. Ugo Carraro was a Professor at the University of Padua, Italy in the Institute of General Pathology (now Department of Biomedical Sciences) and a pioneer in skeletal muscle structural and molecular analyses. Prof. Carraro founded and served as first head the Interdepartmental Research Center of Myology of the University of Padova, Italy. He founded and continue to organize annually the international Conference “Padua Muscle Day”, recently renamed “Padua Days on Muscle and Mobility Medicine” to stress the applications of basic results on many aspects (prevention, diagnostics, managements and rehabilitation) of muscle and neuromuscular diseases of human and veterinary interests.

Prof. Carraro is a world-class expert in structural and molecular investigations of skeletal muscle, having received numerous national and international grants. He applied bidimensional gel electrophoresis for myosin light chains, particularly the embryonic isoform, and was the first to separate mammalian muscle myosin heavy chain isoforms by SDS-gel electrophoresis. He demonstrated the long-term ability of denervated muscle to survive denervation by non-compensatory myofiber regeneration, as well as the beneficial impacts of an athletic lifestyle on muscle reinnervation. Expert in histochemical and ultrastructural morphometry of human skeletal muscle biopsies, he is utilizing his experience in translational myology by analyzing denervation-reinnervation and ageing skeletal muscle. Prof. Ugo Carraro's primary research interests are in the fundamentals of muscle plasticity and their translational applications to medical research (the roles of regenerative myogenesis and apoptosis in exercise-induced muscle damage and in genetic and acquired muscle diseases; Translational myology for Demand Dynamic Cardiomyoplasty; Functional electrical stimulation of denervated human muscle; Functional electrical stimulation of aging human muscle).

Prof. Carraro is currently validating non-invasive blood analyses to monitor (anti- and pro-inflammatory) Cytokines and Myokines via saliva and sweat sampling, a very promising approach that will increase acceptability by volunteers and their frequency, both of which are critical factors in evaluating the numerous transient effects of training and rehabilitation in early ageing and ageing.

Prof. Carraro found and continue to serves to date, as Editor-in-Chief, the European Journal of Translational Myology (EJTM), formerly known as Basic Applied Myology (BAM), published by PAGEpress, Via Antonio Cavagna Sangiuliani, 5 - 27100 Pavia, Italy.

Publications of Ugo Carraro listed in PubMed®

From 1973 to 2022: 165 - Citations > 8000
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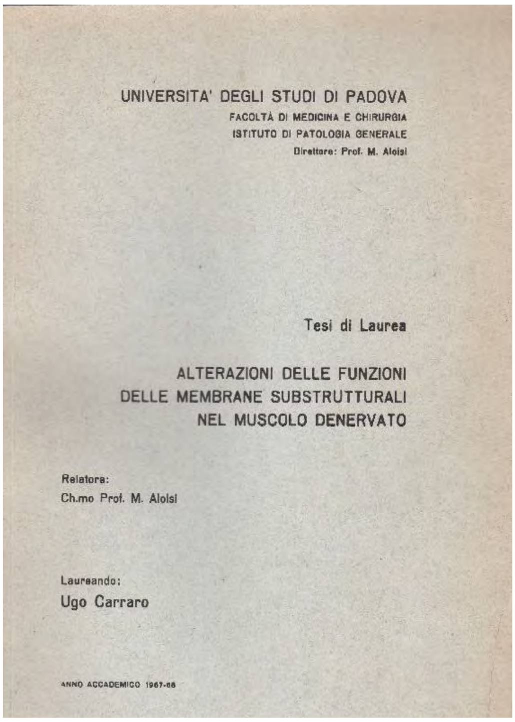
Chapter 1
My family, a key Professor of General Pathology and the first years after M.D. graduation

1.1 My family

One of the most common Italian surnames in the north of Italy, but particularly in Veneto, is the one of the Carraro family. The etymology of this surname is most likely linked to the Latin *carrarius*, which meant the person who built or who had to drive the wagons. But there is also another hypothesis, it is imagined that the family probably took its name from a castle in the vicinity of Padua, the castle of Carrara precisely and in fact the Carraros are nobles from the Veneto region. The meaning and origins of the surname are therefore, as usual in Italy, very ancient. The history of the name has very ancient roots, precisely, the noble family has origins that date back to the early Middle Ages and in particular, to Ugozio who arrived in Italy in 774 following the emperor of the Holy Roman Empire, Charlemagne. This lineage, so distant in time, is historically confirmed by Gumberto, who died in 970. The Carraro family therefore has a very noble origin of the surname, so much so that the title in question was granted thanks to particular merits for completed works in favor of the homeland who were rewarded with the title of noble which announced the beginning of the noble ascent. In fact, the nobles, unlike the bourgeois, had the opportunity to have their own personal coat of arms to represent the important family. All this, of course, was true for one unique or few families in the *Alto Medio Evo*, but is not true for the thousands of Carraro living now in the Province of Padua, where there are more than one thousand persons listed in the telephone book. It was also true for my family living in Abano Terme (Padua), now one of the most important Balneotherapy Station in Europe with more than 100 Hotels offering "*Cure Termali*", that is, warm mud therapies, but in 1943, when I was born, had less than ten. My father's family was of common unknown origin. Of my grandfather, dead many years earlier than me birth, I know only the nick name "*Lustro*", something probably related with his life style, in particular to be always well dressed. I have some good memory of my grandmother, but not of her name [I always called her *Nonna* (Grandmother)], while I remember that she was running an *osteria* (a tavern with a few chambers for guests). Despite the poor conditions of my father's family, he was able to become an accountant for a bank in Abano Terme and from 1946 co-owner with my mother Carmela Mioni Carraro of a new Hotel, increasing year after year the old tavern, where I was born, to a Hotel with "*Cure Termali*", named "*Albergo Sana*t"up to 300 rooms. My mother Carmela Mioni-Carraro was the 16th daughter of my grandparents Mioni-Pezzato, who had decided to leave the care of fields to become hoteliers, together with their numerous sons and daughters, of various Thermal Hotels in Abano Terme and Montegrotto Terme, now collectively better known as "*Terme Euganee*" (Thermae of Euganean Hills, Padua, Italy).



UNIVERSITA' DEGLI STUDI DI PADOVA
FACOLTÀ DI MEDICINA E CHIRURGIA
ISTITUTO DI PATOLOGIA GENERALE
Direttore: Prof. M. Aloisi

Tesi di Laurea

ALTERAZIONI DELLE FUNZIONI
DELLE MEMBRANE SUBSTRUTTURALI
NEL MUSCOLO DENERVATO

Relatore:
Ch.mo Prof. M. Aloisi

Laureando:
Ugo Carraro

ANNO ACCADEMICO 1967-68

Fig. 1. M.D. Thesis of Ugo Carraro, Prof. Massimo Aloisi, Relator. Alterations in the functions of substructural membranes in denervated muscle.
University of Padua, Italy

Those were the roots of the decision, solicited, but not imposed, to enroll in the Faculty of Medicine and Surgery of the University of Padua, Italy, just 12 kilometers from my native Abano Terme. The plan was to become an M.D. and then a Specialist in Physical Medicine and Rehabilitation, to work in the Thermal Hotels of the Mioni, Pezzato and Carraro families in Abano and Montegrotto Terme (Padua, Italy).

After seven years of study (I had to repeat the first year having not reached the mandatory number of practical activities of the Physics course) I graduated with honors in Medicine and Surgery on July 17, 1968 with a Thesis on: Alterazioni nelle funzioni delle membrane substrutturali nel muscolo denervato (Alterations in the functions of substructural membranes in denervated muscle), Relator Professor Massimiliano Aloisi (Figure 1). [166]

My destiny to become an expert in biology, physiopathology and treatment of denervated muscles was sealed!

Fig 2. Wedding of Ugo and Annalisa

In 1969 I enrolled in the Physical Medicine and Rehabilitation Specialty at the University of Padua Medical School, but I never went to lessons, practical medical activities, nor the first year examination.

During the previous years, I had met two key persons and they heavily influenced my decisions: Massimiliano Aloisi, Professor of General Pathology in 1963 (see Chapter 1.2) and in 1969 Annalisa Bossi a student in Mathematics, then my wife in 1973 (Figure 2).

Indeed, I spent six months of 1969 five days a week at the Institute of General Pathology, as a volunteer assistant for bench teaching and research, and the weekends as the young Doctor in my family's hotel. That heavy week job load was the consequence of the fact that the two weeks of stay of the guests / patients at the Thermae of Euganean Hills usually start on Saturday or Sunday. Working seven days a week is a normal lifestyle for hoteliers, my self-included, but it was not acceptable to my fiancée.

After discussing with my parents and Annalisa, I resigned as a doctor of the family hotels to pursue a university career as teacher and basic scientist in the medical fields.
It has been an easy, quick and winning decision.

The reasons will be clear if anyone keeps reading this book:

1. I haven't lost a girlfriend;
2. Our family is growing up in London where our only son Alessandro lives with his wife and two children (Figures 3 and 4);

Fig 3. London, UK. August 24, 2021. Sushi dinner at Alessandro's home. From left: Antonio Carraro, Monika Carraro, Annalisa Bossi Carraro (Nonna Isa), Cristina Carraro, Alessandro Carraro.

Fig 4. Padua, Italy. October 15, 2022. Pizza dinner at the Marechiaro Restaurant in Via Manin, Padua, Italy. From left: Cristina Carraro, Alessandro Carraro, Ugo Carraro (Nonno Ugo), Monika Carraro, Antonio Carraro.

3. I am happy to write this book in Padua (Italy), September 2022 after more than fifty years of dreams, hopes and frustrations.
In fact, I was very lucky to have avoided potential family conflicts, as inevitable as those that occurred during my career as a lecturer and researcher at the University of Padua. But only careful readers will find traces of them in this book, because I have decided to minimize the dedicated pages.

Only conflicts that have opened up new opportunities are reported.

1.2 A key Professor of General Pathology

As a student enrolled at the 2nd year of the curriculum of Medicine of the University of Padua, Italy, I was obliged to follow the teaching hours of Anatomy, Chemistry, Physiology and General Pathology. Being a lazy student after a few boring lectures of other professors, I decided to follow only the lectures of Professor Massimiliano Aloisi and his collaborators. A few words on his carrier and an abstract (taken from an Issue of the European Journal of Translational Myology [167] that report the contents of a Padua Muscle Days organized in 2008 to honor his legacies nine years after his departure in 1999) explain, much better than I can, why I decided to: i) follow his lectures in 1962 and 1963; ii) enroll as an Internal Student at the Institute of General Pathology in 1963, where I spent almost all my afternoon (five days a week, excluding August) to learn histopathology and clinical biochemistry; iii) graduate in Medicine in 1968; and iv) remain at the Institute of General Pathology as a Voluntary Assistant of the Prof. Massimiliano Aloisi.

Massimiliano Aloisi

Massimilano Aloisi - 1990
Emeritus Professor of General Pathology at the University of Padua, Italy.
Born in Florence on December 19, 1907; Died in Rome on October 22, 1999.

Massimiliano Aloisi graduated in Medicine and Surgery at the University of Florence in 1932 and qualified for the profession at the University of Pavia. He was assistant from 1932 to 1934 at the Florentine Anatomical Institute directed by the famous Giulio Chiarugi, in which he prepared his degree thesis on the distribution of glycogen in the guinea pig embryo. He then passed as Assistant to the Institute of Pathology of Rome, directed by G. Vernoni. He remained there as Assistant and Help until 1948 dealing with teaching and research activities of the same Institute and of the Study Center for Physiopathology of the *Consiglio Nazionale delle Ricerche* (Italian National Research Council), directed by G. Vernoni. In 1940 he obtained the free teaching in General Pathology. He was able to perfect his technical and scientific training by going first to the *Institute fur Zellphysiologie* (Kaiser-Wilhelm-Gesellschaft) in Berlin-Dahlem, then directed by Otto Warburg, and then to the biochemistry department of the Postgraduate Medical School (Hammersmith Hospital), then directed by E.J. Re. In 1948, first of the three in the relative competition, he became Extraordinary Professor of General Pathology at the University of Ferrara, from which, after three years, he passed to the Medical Faculty of Modena where he remained for another eight years. Finally he was called by the Faculty of Padua where he was Full Professor of General Pathology until 1978, non-permanent professor until November 1983 and then professor emeritus. From 1954 he was Director of the Center for the Study of Physiopathology of the National Research Council, until its restructuring, in 1971, in the Center then called Center for the Study of Muscular Biology and Physiopathology. He was President of the Scientific Council of the same Center from 1971 to 1975. From 1945 to 1947 he was a member of the Superior Council of Public Education as Professor and University Assistant. From 1968 to 1978 he was a member of the Advisory Committee for Biological and Medical Sciences of the Italian National Research Council. Corresponding member of the Accademia Nazionale dei Lincei since 1956, he became National Member in 1959. Effective member of the Patavina Academy of Sciences, Letters and Arts and of the International Academy of Pathology, Honorary Member of the Academie Royale de Medicine de Belgique, Foreign Correspondent of the IV Division of the National Academie de Médicine de France, Corresponding Member of the Society of Biology of France. In 1954 he won the "Feltrinelli" National Prize of the National Academy of the Lincei. Died in Rome on 22 October 1999. He left his library and the archive of papers to the Veneto Institute of Sciences, Letters and Arts. M. Aloisi is the author of over 100 publications on topics of embryology, normal and pathological histology, bacterial biochemistry and muscle biology, biochemistry and physiopathology. Numerous are those devoted to theoretical questions of biology and philosophy of science. [168]

Massimiliano Aloisi departure on October 22, 1999 took away a model for biomedical students and scientists, leaving an indelible sorrow for many in Pathology, Neurosciences and Italian Universities. He was known for his studies on damage and muscle regeneration after Vitamin E deprivation and supplementation. Those studies developed into an Italian-USA collaboration in the 60s of last century, obtained founds from the Muscle Dystrophy Association of America and opened to his young fellows the doors of many International Laboratories and Universities. Some of his students returned to Italy with precious knowledge, others succeeded to become Professor of Neurology or Myology Leaders in the States. Other pupils succeeded as brave and innovative surgeons. Massimiliano Aloisi had also strong friendly relationships with East-Europe Scientists, and managed to help them when in troubles for their political choices. One of his many roles was to encourage the development of culture systems for muscle cells (in vivo and in vitro). Furthermore, he opened in Padua a muscle Electron Microscopy laboratory with many good fellows. Charismatic, and often controversial, he was Professor of General Pathology in Ferrara, Modena and Padua, but maintained for all his life his Roman roots, participating to the Italian National Research Council (C.N.R.) organization and management. We remember the three goals of his life: i) Promotion of the scientific method and of "the scientific Systems Medicine", ii) Internationalization of research, and iii) Strong mentorship of young students and scientists. Proud of his Roman roots and of being a Professor in one of the University in which Galileo Galilei taught and the Normal Anatomy and Medical Pathology had been developed, he was a fascinating "Maestro". Some of his pupils, which are here present, remind his presentations of histopathological cases by projecting his own beautiful color slides. He always taught raising questions and patiently driving to replies a crowd of students. His Socratic teaching influenced many young Italian doctors, not enough, unfortunately, considering the bad decisions that the majority of them took in "reforming" the Italian Medical School during recent years! The University of Padua is proud to honor the memory of one of his Professors, sponsoring the Meeting on "Skeletal muscle in denervation, aging and cancer, Padua and Terme Euganee, Padua (Italy), March 15 – 17 and May 2, 2013". Many experts in muscle damage and functional recovery are participating, in particular those fond of rehabilitating paraplegics by FES, but other world-class experts in Cell Stemness for the recovery of muscle and other soft tissues are among

Lectures and Speakers. Padua is notoriously a city of great cultural prestige. Thanks to its ancient University, it has been a landmark for arts, philosophy, law, medicine and science during the centuries. Testimonies of its prestigious past remain also near us. You may today visit the anatomical theatre where Fabrizio d'Aquapendente performed the first autopsies with scientific spirit and where five hundred years ago William Harvey discovered how the heart pumps blood toward the body. Chair of Galileo Galilei, the great supporter of the experimental method, is over the next door. In the past, and today, great personalities (painters, among others) converged in Padua. The Scovegni's Chapel, painted by Giotto, is a Renaissance's jewel. We would like to remind and witness to people, who did not meet Massimiliano Aloisi, his enthusiasm for research, his pragmatism and perseverance to overcome difficulties, and above all his high motivation as a fundamental message for young generations.
[Taken from the Proceedings of the 2008 Spring Padua Muscle Days, in the European Journal of Translational Myology/Basic Applied Myology 2008;18(1):4-5.][167].

1.3. The first years after graduation in Medicine and Surgery

After I graduated in Medicine and Surgery, I decided to pay my obligation to have military service as a young doctor spending September to November 1968 in Florence at the Military Academy. When I returned home, I had to wait several months to be certified as a M.D. During the winter of 1968-1969 I was able to often go to the Institute of General Pathology of the University of Padua.
During the University of Padua Dancing Celebration of February 8, 1969 I meet a young student of Mathematics, Annalisa Bossi. She decided I was the right man for her, so we fell in love and got married in 1973. In July 1969 I was aggregated as military doctor to the "Italian Lagunari Squadron" at sea side east of Venice. It was a wonderful summer, but in late autumn 1969 returning from Padua (where I had spent a long evening with my girlfriend Annalisa) to the Lagunari barracks of Ca' Vio (Venice), I had a serious car accident whose consequences lasted up to the late autumn of 1970.

During recovery from the car accident, I had the possibility to work at the Institute of General Pathology with one of the senior helpers of Professor Aloisi (See Chapter 1.2). As a voluntary and then recruited assistant of Professor Aloisi, I worked on the research projects of Alfredo Margreth during the following 10 years.
Because Giovanni Salviati, the first choice and one year older than me, also had to comply his military obligation, I had the opportunity to join the Margreth's group substituting Giovanni in the current research projects. Thus, I was included as coauthor in two interesting publications on neural control of the activity of the calcium-transport system in sarcoplasmic reticulum of rat skeletal muscle (Margreth A, Salviati G, Carraro U. Nature. 1973 [1]; Margreth A, Carraro U, Salviati G. Biochem J. 1974) [2].
Unfortunately, I was also asked to work on other topics in a rabbit model. Being the results of the experiments never as expected, we repeated the experiments for tens and tens of times, never attaining the desired results.
Beside this, I have a gap from 1974 to 1979 in my list of publication also because the 1975 to 1978 were very turbulent years in Italy. The "Brigate Rosse [the Red Brigates]"were killing people up to the Prime Minister Aldo Moro.
I remember a day when I was in the Library of the Institute of Physiology when young males and females, perhaps pre-university students, came in with incendiary bottles to burn the library. Fortunately, due to their inexperience, they failed their goal.
The police watched over the university canteens to avoid infiltration of students, or pseudo-students, willing to eat without paying, intimidating the staff and calling their behavior proletarian expenditure. It was not easy to go to university buildings to teach and carry out bench research.
Anyhow, the molecular analyses of protein synthesis also abruptly exploded in those years. So we moved to the new topic with the help of an England colleague, who spent his sabbatical at the University of Padua. He taught me how to denervate the left rat hemidiaphragm and to prepare skeletal muscle ribosomes from three-day denervated and the right contralateral innervated hemidiaphragm that supported pulmonary ventilation in experimental animals. Despite the results of those studies never attained the level to allow publication in international journals (with the exception of a single paper: Carraro U, Catani C. 1980) [4], a group of animals forgotten for six-month in the animal house offered me the occasion to study effects of long-term denervation of muscle and to submit

and have accepted my first first-name publication in a decent journal (Carraro U, Catani C, Biral D. Exp Neurol. 1979) [3].

Having established an original research program, I was then able to publish more than one good paper per year up today on skeletal muscle biology and physiopathology both in experimental animals and in human diseases' cases, the two stages of biomedical research that were then identified as Translational Medicine, in my case Translational Mobility Medicine.

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Chapter 2
Studies of long term denervation of skeletal muscles in rodents: the emidiaphragm model

In the long term denervated rat diaphragm, specific neurotrophically controlled proteins are maintained

Skeletal muscles could contain different type of muscle fibers, characterized by their speed of contraction (fast or slow), histochemical and immunohistochemical pattern of staining and presence of genetically determined molecular properties, including those of the types of isomyosins. Skeletal muscles generally contain a mixture of fiber types, but small leg muscle of rodents may be very reach of one type of them either the fast or the slow fiber types. This peculiar distribution of fiber types is strictly under the control of different motor neurons that innervate them. In both fast and slow muscles, denervation causes a variety of distinct alterations. On the 70s of the last century fast and slow muscle fibres rigidly treated to the same mechanical stimuli have not yet been compared following long-term denervation, nevertheless. The unilateral denervated rat diaphragm (a mixed muscle) provided a good chance to investigate the effects of denervation and stretch brought on by the rhythmic contractions of the contralateral intact hemidiaphragm on the diverse population of fiber types. After unilateral phrenic nerve transection, the myosin of the denervated hemidiaphragm was examined for ATPase activity and light chain composition every month. After 6 months of denervation, the denervated hemidiaphragm (a mixed muscle) changed into a fast-type muscle in both parameters. This observation is in favor of the hypothesis that motor neurons control the properties of the different type of muscle fibers trought the amount of activity they impose to the muscle fibers [3]. For details, see Chapter 14.1.1. Gerta Vrbová [154] and in Dirk Pette discussion of the significance of Gerta Vrbová's low-frequency stimulation experiment. Eur J Transl Myol. 31 (1): 9585, 2021 doi: 10.4081/ejtm. 2021.9585 [169,170].
On the other hand our observations suffered the criticism that the chronic

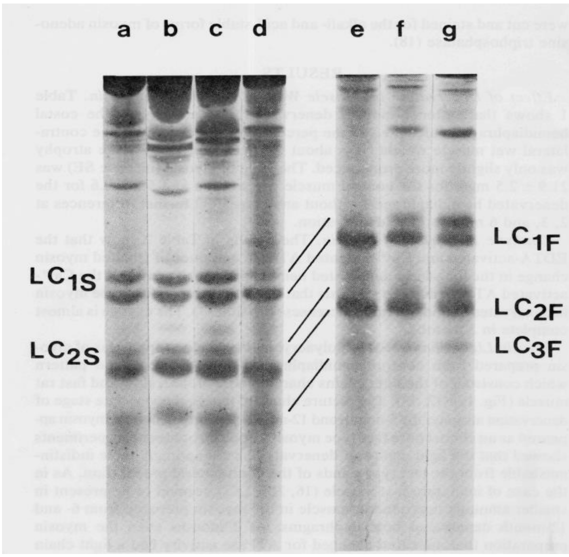

Fig 1. SDS-polyacrylamide gel electrophoretograms of myosin prepared from control and denervated hemidiaphragms. Pattern of muscle myosin light chains of: a—normal hemi-diaphragm, b—muscles denervated 2 months, c—muscles denervated 3 months, d—muscles denervated 6 months, e—muscles denervated 12 months, f—muscles denervated 12 months and contralateral coelectrophoresis, g—contralateral normal hemidiaphragm at 12 months. LC1S, LC2.S—myosin light chains that distinguish the slow muscles, LC1F, LC2F, LC3F—myosin light chains that distinguish the fast muscles

stretching could be an important factor in determining the observed isomyosin transitions. Thus we extended our control muscles by transecting also the sciatic nerve of the rats and thus studying long term denervation in almost resting muscles of the legs, i.e., with minimal passive movements.

Rat gastrocnemius and diaphragm myosin light and heavy chains analyses during chronic denervation or reinnervation confirm that specific neurotrophically controlled proteins are maintained after long term denevation even in absence of passive stretching

The myosin, a protein characterized by different types of heavy chains and light chains, of rat muscles that had been chronically denervated and re-innervated was investigated. The 6-month denervated hemidiaphragm and gastrocnemius muscles (severely atrophic) almost exclusively contained fast-type isomyosin, whereas in reinnervated muscles (who attained almost normal size) the normal two types of myosins were present, as in normal muscles, as shown by two-dimensional gel electrophoretic pattern of light chains and the tryptic mapping of the native molecule [5]. Myosin heavy chains (MHC) that had been electrophoretically isolated were subjected to chymotryptic peptide mapping under denaturing conditions, which demonstrated that the modifications noticed affected both the myosin light and heavy chains [8]. The results of reinnervation tests lead one to draw

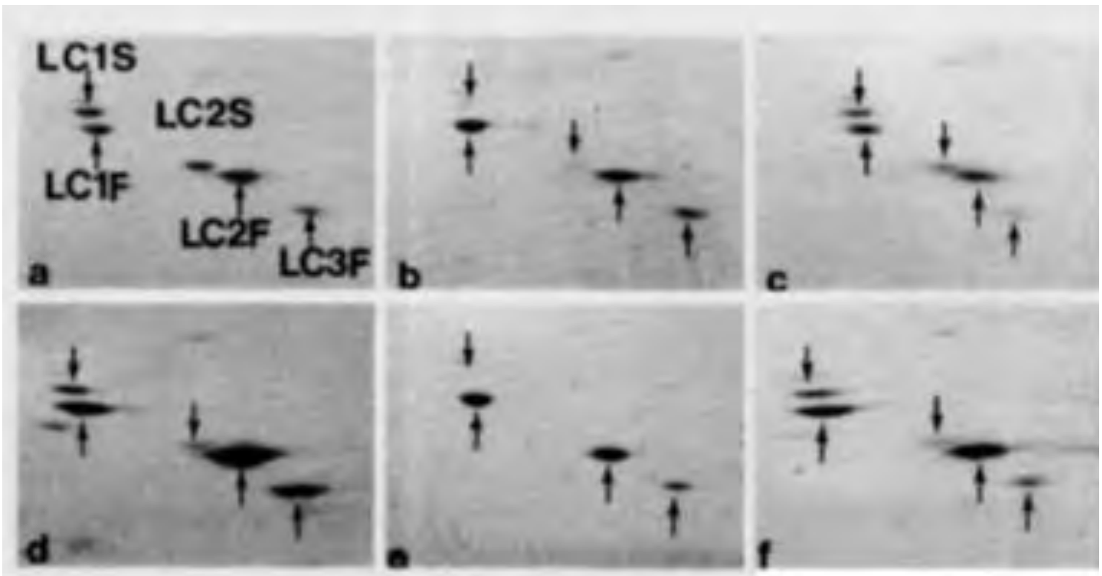

Fig 2. Two-dimensional gel electrophoresis of myosin light chains from control, chronically denervated, and reinnervated diaphragm and gastrocnemius rat muscles. Patterns of muscle myosin light chains of: a—control hemidiaphragm, b—6-month denervated hemidiaphragm, c—6-month reinnervated hemidiaphragm, d—control gastrocnemius, e—6-month denervated gastrocnemius, f—6-month reinnervated gastrocnemius. LC,S, LC2S—myosin light chains that distinguish the slow muscles, LC1F, LC2F, LC3F—myosin light chains that distinguish the fast muscles. Downward vertical arrows indicate slow-type chains, upward vertical arrows indicate fast-type light chains. Only the light chains region of the slabs are presented. About 40 μg of myosin per slab were used. After 6-month denervation the slow-type components that are clearly visible in reinnervated muscles, have almost completely disappeared in both diaphragm and gastrocnemius muscles.

the conclusion that spontaneous reinnervation events cannot account for the preferential maintenance of fast-type myosin in chronically (severely atrophic) denervated muscles, supporting our conclusion that slow-type characteristics of normal muscle are related to a continuos activitation of the muscle contractions.
By these experiments we also explored the eventual differential intrinsic molecular stability of the different types of isomyosin. A peculiar quality of skeletal muscle myosin appears to be the length of time needed to demonstrate adaptive responses to neural and non-neural stimuli. Not only in denervation tests but also following cross innervation, inactivity, or electrical stimulation of the supplying nerve, a significant amount of time is required to cause a shift in muscle fiber-types. It was proposed that this distinctive time course may be connected to myosin's apparent longer half-life than glycolytic enzymes or membrane proteins in muscle. Even though such a transformation occurs a long time (months) after the muscle has reached the maximum atrophy, it's possible that the faster half-life of fast-type myosin, compared to that of the slow type, is significant in the interpretation of molecular events induced in a mixed-fiber population by long-term denervation. It is tempting to hypothesize that the inertness of myosin metabolism may be to blame for the challenges in getting myosin-type alterations in directly stimulated muscles after denervation. In any case, we extended again our analyses to other neuroregulated contractile proteins, the tropomyosins.

Differences in tropomyosin subunit distribution between fast and slow rat muscles and their changes on the long term denervated hemidiaphragm confirm that specific neurotrophically controlled proteins are maintained or lost

The rod-shaped protein known as skeletal tropomyosin, which is found in the grooves of the double-stranded F-actin filament, is a component of the calcium control mechanism for muscle contraction. The native molecule is a dimer in which the two tropomyosin subunits are differentially assembled in slow and fast mammalian muscles, the ratio of molar amounts of tropomyosin subunits differs, with one subunit being more represented in the slow type and the other subunit being more represented in the fast type muscle fibers. By using electrophoretic analysis of highly purified tropomyosin and immunohistochemistry analyses, the distribution of tropomyosin subunits has been investigated in a number of mammalian species. Electrophoresis in two dimensions has been used to gather the results. Tropomyosin subunit positions have been determined in a two-dimensional electrophoretogram of tropomyosin purified using conventional methods, using crude myofibrils from rat muscles. Actomyosin was electrophoresed in two dimensions from both healthy and permanently denervated rat muscles. Thus, in the same preparation, the tropomyosin subunit pattern and the myosin light chain pattern were examined. According to electrophoresis, adult rat soleus, a muscle that is primarily of the slow type, has nearly exclusively thee-subunit of tropomyosin, whereas adult fast and juvenile muscles both include the subunits in roughly equal amounts. This is another proof that, despite having a distribution specific to each mammalian species, the tropomyosin subunit ratio in fast and slow skeletal muscles differs. This study of tropomyosin subunits and myosin light chains reveals that the long-term denervated hemidiaphragm has a lack of slow type components [6].

Rat hemidiaphragm after chronic denervation: preservation of fiber heterogeneity and corresponding rise in fast type myosin isoform by analyses of single muscle fibers, electron microscopy and 2D gel electrophoresis

Rat mixed muscles lose slow myosin over the course of several months of denervation, though the rate of loss varies between animals. Observations could be extended to the analyses of single fibers isolated from the experimental muscles. All the single fibers analyzed of the denervated hemidiaphragm reacted with an anti-fast myosin, but many also reacted with anti-slow myosin, according to immunocytochemical investigations [15]. This raises the question of whether different myosin variants coexist within individual fibers or if only one distinct myosin, presumably an embryonic variant that shares epitopes with both fast and slow myosins, is present. Another question to consider is whether the re-expression of embryonic myosin in the pre-existing fibers and cell regeneration are connected in chronically denervated muscle. We used SDS PAGE to examine the myosin heavy chains from single fibers of the denervated hemidiaphragm and performed a morphological search for regeneration events in the long-term denervated muscle in order to provide answers to these issues. The severely atrophic fibers of the hemidiaphragm revealed either fast or a combination of fast and slow myosin heavy chains three months after denervation. Despite the absence of the selective distribution of fast and slow characteristics, typical of normally innervated muscle fibers, structural analysis of proteins sequentially isolated from muscle cryostat sections revealed that slow myosin was still present 16 months after denervation. If they are denervated during development and differentiation, muscle fibers can express adult fast myosin, but they can also do so after the slow programme has been active for a while. Both light and electron

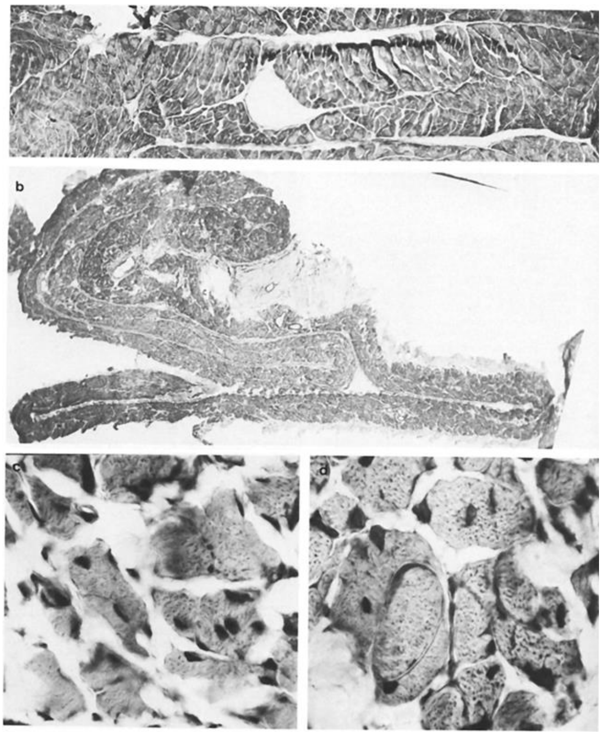

Fig 3. 16-mo-denervated hemidiaphragm. (a) A portion of a cryostat section of the contralateral innervated hemidiaphragm. (b) At the same magnification, the cross-section of the whole denervated hemidiaphragm. The folded muscle is made up by so much atrophic fiber that each layer appears to be about one fourth the size of the contralateral innervated muscle. The fibers are hardly distinguishable from each other . (c) At higher magnification the smaller fibers are shown to be angulated in shape and separated by wide interfiber spaces. (d) An example of the numerous clusters of round fibers wich are scattered among the atrophic fibers. Fibers with central nuclei are also present. X 60 (a and b); x 1,120 (c and d)

microscopy revealed that the muscle that had been long-term denervated remained atrophic throughout the rat's life. A few morphological characteristics suggest that aneural regeneration events continue to take place and may contribute to the increasing uniformity of myosin gene expression in long-term denervated diaphragm. This conclusion stands on a series of molecular analyses we will discuss in Chapter 4: Post-damage muscle regeneration is independent from motor neuron innervation; Macrophage-myoblast interactions and beyond.
In the next Chapter 3: Muscle fiber types and their modulation by electrical stimulation in rodents, we will discuss the effects of contratile patterns in denervated rat muscles induced by chronic electrical stimulation performed using implantable electrodes connected to an external electrostimulator able to mimic the discharge pattern of either the fast or the slow motoneurons. Complementary descriptions of this experimental approach could be found in the Chapter 13.1.1. Gerta Vrbová [154].

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Chapter 3
Molecular approaches to study isomyosins and modulation of muscle fiber types in rodents, sheep, canines and humans

The study of the different molecular characteristics of the contractile proteins, in particular of the myosin isoenzymes of the different types of muscle and muscle fibers, was a tedious and laborious work until the introduction of SDS Gel electrophoresis, which allowed us to confirm results collected by histochemistry on cryostatic section of skeletal muscles. Giovanni Salviati's groups (Romeo Betto, Daniela Danieli-Betto and Donatella Biral) and my groups (Claudia Catani, Luciano Dalla Libera, Corrado Rizzi, Katia Rossini, Marco Sandri and myself) equally contributed to optimization of methods that appeared serially in the international literature, but we also designed and implemented original protocols. Notably, this was the case with the separation of myosin heavy chain isoforms by gel electrophoresis. During a stage I spent in Boston in the laboratory of John Gergely and Frank A. Sreter, I first observed that in the gel plate it was possible to see by light diffraction that the myosin heavy chains sometimes splitted in the upper region of the gel plates. Then in Padua the era of the gel gradient concentration plates began, which required the addition of 25% glycerol to one of the gel buffers. This was the trick that allowed the separation of three or more different types of heavy muscles from fast and slow muscles in a gel plate allowing the comparison of dozens of samples under the same conditions. The sensitivity of the silver stain introduced in the coloration of the plates made it possible to determine the nature of the fiber types by analysis of single cryostat sections of small rodent muscles or of individual muscle fibers. All those developments have allowed to gather information on skeletal muscle plasticity also in human muscle micro biopsies.

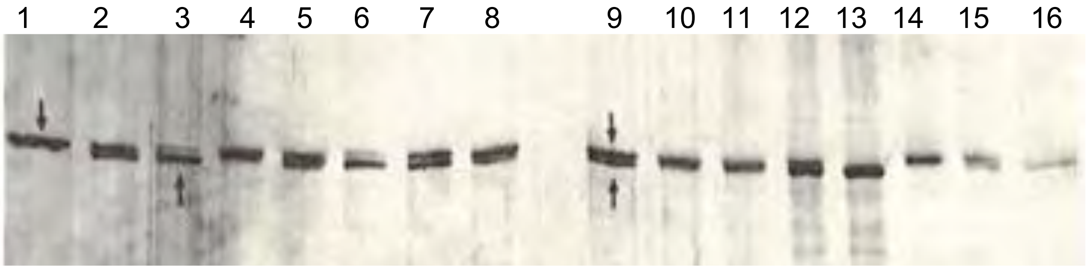
3.1 Improving molecular approaches to study isomyosins from milligram to nanogram amounts of protein muscle samples

The first evidence that myosins were molecularly different in fast/slow (also known as white/red) muscles of mammals and bird was based on enzymology approaches showing that the activity of the fast myosin ATPase was higher than that of slow myosin [171]. Furthermore their resistance to either acid or basic pretreatment allowed to demonstrate by histochemistry that the vast majority of the muscles contain a mixture of fast and slow myofibers [172]. The enzymatic and structural study of isolated myosins require tissue grams, tissue grinding, serial centrifugation and ultracentrifugation of the samples to isolate the different substructures of muscle fibers, i. e., contractile proteins, mitochondria, plasmalemma and sarcoplasmic reticulum, from the final supernatant devoid of substructures. To isolate the actomyosin complex, a complementary approach is to solubilize the first pellet with a highly saline solution followed by a dialysis against water to precipitate myosin, the main contractile protein constituting the thick filaments of skeletal muscles. When isolated myosin is subjected to electrophoresis on Sodium Dodecyl Sulphate Poly Acrylamide Gel Electrophoresis (SDS-PAGE) it is possible to separate the heavy chains from the light chains, and the different subunits present in embryonic, developing and adult muscles. The light chains present in the predominantly fast muscles are different from those found in the slow muscles of adult animals. The demonstration that heavy chains also have a peculiar differential composition was initially based on the electrophoretic analysis of the products of proteolytic digestion of myosin or its isolated heavy chains. We were able to obtain all this information (including a typical embryonic light chain 1, i.e., LC Emb 1) by SDS PAGE of proteins present in single cryostatic section of the rat leg muscles in experiments of myotoxic damage and regeneration (see Chapter 4). Others were studying single myofibers from skeletal muscles of mammals, birds and fishes. This was possible also thanks to adaptation to gel slab of an old histological method based on silver staining and then by using immuno-staining. Thus, the required amount of proteins analyzed by SDS PAGE decreased from milligrams to nanograms and the initial amount of tissue decreased from grams to milligrams of skeletal muscle, an amount easily collected by muscle biopsy in animal models or sick patients. As an example I report below one of our publication (Carraro U, Catani C. 1983) [12] on a sensitive SDS-PAGE method that separates the myosin heavy chain isoforms of rat skeletal muscle revealing the heterogeneous nature of embryonic myosin. To achieve those results we also optimized methods to separate the myosin heavy chains by preparative SDS gel electrophoresis or after eluting them from gel slabs. Complementary methods to recover proteins from very diluted solutions were also major contributions to improvements of those methods, [31,35,38,68] in particular thanks to commitment of Corrado Rizzi to solve the emerging problems (see also Chapter 14.3.4).

A sensitive SDS-PAGE method separating myosin heavy chain isoforms of rat skeletal muscles reveals the heterogeneous nature of the embryonic myosin

Myosin isoforms are used as markers of heterogeneity and plasticity of skeletal muscle fibers and motor units. Tedious and time-consuming methods, needing microgram or milligram amounts of myosin are widely used to characterize the heavy subunits. We described a sensitive procedure that separates in nanogram or microgram amounts the heavy chains of immature, fast and slow adult rat muscles in complex mixtures of isomyosins. Though the method was assembled from published procedures (SDS-PAGE, peptide mapping in the presence of SDS, silver stain) for the logical extensions introduced the end-product is a powerful tool to separate and characterize those high molecular weight biopolymers until then inseparable in complex mixtures. The method revealed the heterogeneous nature of the embryonic myosin heavy chains (MHC). Skeletal muscles are composed of a large number of fibers with different physiological and biochemical properties that under neuronal control can respond to a variety of stimuli in a plastic manner. Although the differences are mainly quantitative in nature, some are qualitative, implicating a switch in the control of gene expression in single fibers [173].

Electrophoresis of myosin in SDS-5% polyacrylamide slab gels.



The heavy chains of myosin from adult EDL (essentially pure fast fibers) and soleus (predominantly slow fibers) were compared by direct electrophoresis in Laemmli slab gels. When 250 ng of soleus myosin were electrophorized in 5% polyacrylamide two bands appeared when stained with the silver method. The predominant band (MHCS) of the soleus (i. e., that of the slow fibers) migrated faster than the heavy chains (MHCF) of the EDL. The smaller band of the soleus myosin comigrated with the MHCF band of the EDL, and could have constituted the heavy chain complement from the fast fibers in the soleus (usually between 5 to 8 % in ATPase stained histochemistry of rat soleus muscle). The difference in mobility between MHCF and MHCS seemed to disappear upon coelectrophoresis of myosin from fast and slow muscles, if the amount of protein applied was higher than 1000 ng. The use of low

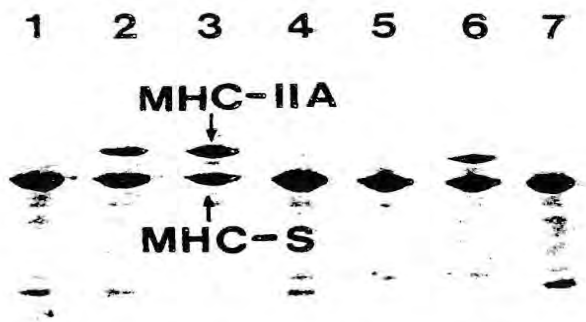


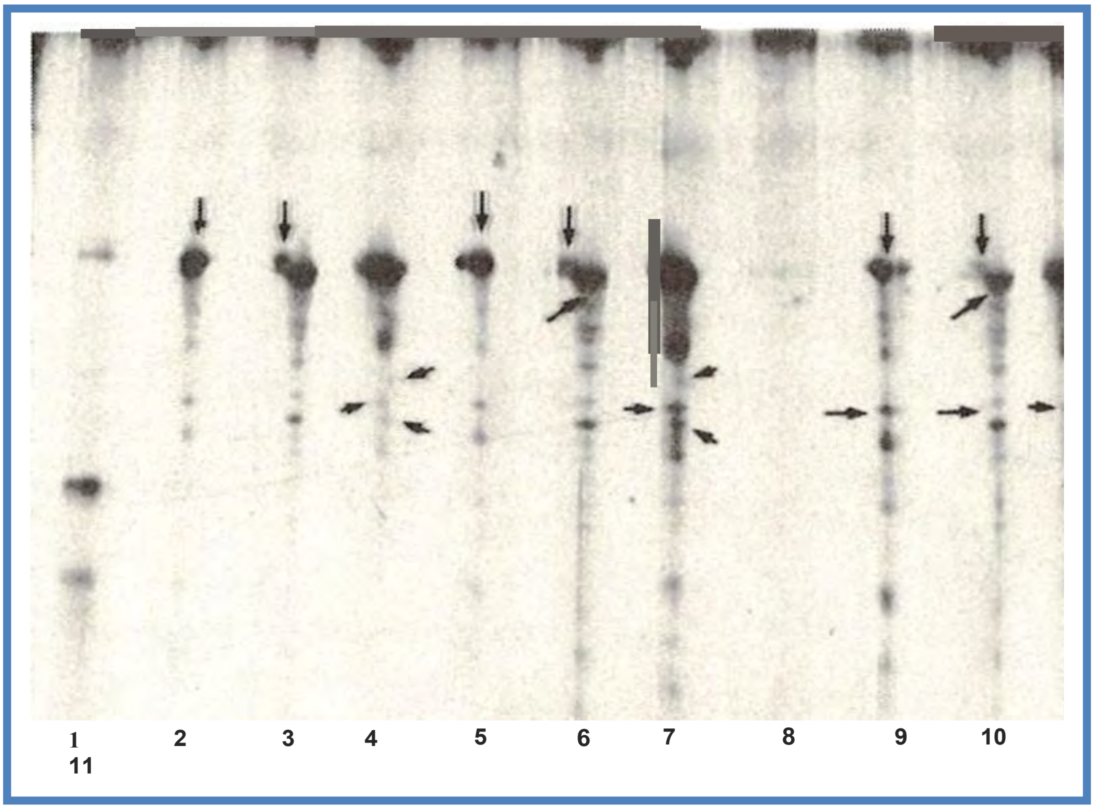
Figure 2. SDS PAGE of Myosin Heavy Chains from soleus muscles of rat, mouse and sheep. Heavy chains of myosin from: 1, rat; 2, 1+3; 3, mouse; 4, 1+5; 5, sheep; 6, 3+5; 7, rat.

percentage of polyacrylamide (5%) was not a necessary condition to separate MHCF from MHCS. On electrophoresis in a 7.5% gel the difference was smaller but detectable. Furthermore SDS of different sources had been used without influence on the mobility of MHC. Thus a critical amount of protein and the presence of 25% glycerol in the buffer were the only requirements needed to separate fast and slow

myosin heavy chains (Ugo Carraro, personal communication).
The large myosin load required to detect light chains in standard myosin SDS PAGE electrophoresis explains why this difference was overlooked. In fact, only a 10% share of polypeptides could be attributed to isolated myosin light chains (Ugo Carraro, personal communication). The only previous report of myosin heavy chains separation with the Laemmli system has been presented in a study of myosin heavy chains from normal and dystrophic chicken muscles [174]. However this interesting observation surprisingly has been neglected.
The fast migrating band (MHCS) from soleus is not a proteolytic product of the true heavy chains of the slow fibers because three independent preparations of soleus myosin have the two MHC bands in the same proportion and as shown below they have different proteolytic patterns in orthogonal peptide mapping. Furthermore the tibialis anterior (essentially pure fast fibers) showed MHCF, while the diaphragm which is a natural mixture of fast and slow myosins, the fast type being in slightly major proportion showed a mixed fast population of adult rat muscle fibers.
Interestingly, Figure 2 (taken from Carraro U, Catani C. 1986 Proceedings of the Vienna FES) shows that the myosin heavy chains of three different species of mammals comigrate in the 7% SDS PAGE gel electrophoresis. Notice that the percentage of fast MHC is larger in mouse soleus, as well known from ATPase histochemical analysis of mouse muscles.

Orthogonal peptide mapping (SDS-OPM) of MHC isoforms from EDL, soleus and embryonic muscles

For orthogonal proteolytic mapping (SDS-OPM), the MHC were electrophoretically purified in 150 mm width, 100 mm height, 0.5 mm thick, 5% polyacrylamide gel slabs. The peptide mapping was performed essentially according to [175], using S. Aureus protease. The MHC bands separated from 1000 ng of myosin were briefly stained with Coomassie blue and equilibrated in 125 mM Tris-Hcl , pH 6.8 - 1 mM EDTA - 1 mM 2-mercaptoethanol. The gel slices were introduced into the slots of a 0.75 mm thick, 7.5% polyacrylamide gel slab with the long axis of the bands orthogonal to the surface of the slabs. The stacking gel 15 mm height was 4% polyacrylamide. After the bromophenol tracking dye migrated 5 mm into the stacking gel a solution of S. Aureus protease containing bromophenol blue was poured into the slots. After the tracking dye of the protease fused with the front dye near the interfaces between the stacking and the separating gels, the run was interrupted for 15 minutes. Under these conditions the digestion of MHC may be estimated to be of about 20 min at the selected protein to protease ratio. The running buffer was 25 mM Tris-Hcl - 192 mM glycin- 0.9 % SDS , pH 8.3. The power supply was setted at 50 V during the stacking of the proteins, then at 50 V. The gels with picograms amounts of myosin were stained with Coomassie blue. When myosin was applied in nanograms amounts the silver stain of [40], as modified by R. Betto (personal communication) was used to visualize proteins. A detailed description of the methods has been published as Rapporto Interno n. 1/82 of the C.N.R. Unit for Muscle Biology and Physiopathology-Research theme 01. The report is available on request.
The below figure shows the SDS-OPM of the MHC from adult EDL (lanes 2, 5 and 9), adult soleus (lanes 3, 6 and 10), embryonic muscles (lanes 4, 7 and 11). Under the conditions used the digestion of MHC may be estimated to have been of about 20



min at a protein to protease ratio of 20:1 in the lanes 2, 3 and 4 , 10:1 in the lanes 5, 6 and 7, 5:1 in the lanes 9 , 10 and 11. The EDL and soleus MHC displayed peculiar peptide patterns consistent at three different amounts of S. Aureus protease. The heavy chains of soleus myosin showed a trace amount of the MHCF. Indeed under this spot the major peptide (indicated by rightward arrows) of the MHCF pattern is present. The orthogonal peptide map of embryonic MHC (lanes 4,7,11) showed that the spots do not extended for the entire span of the MHC band, some being present under the right-hand portion, others under the left-hand portion of the MHC band. Thus at least two bands ran unresolved one in front of the other in 5% SDS-PAGE. The MHCF present in soleus myosin displayed a pattern similar to that of EDL MHC.
Therefore the method we have here described separates nanogram amounts of heavy chains of immature, fast and slow adult rat muscles in complex mixtures of myosins. Though the method is assembled from published procedures for the logical extensions introduced the end-product is a powerful tool to separate and characterize high molecular weight biopolymers until then inseparable from complex mixtures. The method is very sensitive, allowing to detect a 2% "contamination" in a mixture of proteins. The standard peptide mapping of Cleveland et al. [175] hardly recognize a single component when it is present in amounts less than 20%.
Besides the ability to separate MHC of adult fast and slow rat muscles we have shown that it is possible to distinguish different heavy chain isoforms in the myosin of embryonic muscles.

Acknowledgments
We thank Dr R. Betto and L. Dalla Libera for helpful discussions. The authors gratefully acknowledge the skilled technical assistance of Mr. Silvio Belluco.

3.2. Modulation by electrical stimulation of muscle fiber types in rodents, sheep and canines

I previously introduced the idea that skeletal muscles are not as an uniform tissue as they appear in butchers' frigos. In fact, the muscle can be both white (in rabbit, chicken and fish) and red (in cattle, sheep and goats). Their dynamic contractile properties are fast or slow. Finally, all these characteristics are strongly dependent on kind of innervation of motor units, being the entire spectrum dependent on their gene expression. Interestingly, those properties changes during development and also in adult animals under the control of involuntary changes in use, disuse and neuromuscular disorders. That is, muscles are very plastic not only because of their size, but also because of the patterns of gene expression in adulthood and aging. A simplistic hypothesis is that all this plasticity is under the control of the central and peripheral nervous systems that dictate the behaviors of the different muscle units of which an adult muscle is typically composed. Due to the dependence of the activities of neurons and muscle fibers on their own generated electrical events, it is not surprising that electrical stimulation can be mimicked by external currents applied through the skin (surface stimulation) or by implantable devices, the cardiac pacemaker being the most effective clinical application.
As we will see again in Chapter 11, electrical stimulation (ES) of skeletal muscle was used from the beginning of the electrical physics and engineering. Current accepted management of muscles are in cases of atrophy due to muscle disuse, particularly during or after bone recovery from fracture cast immobilization. More debated is its use as a pain reliever and, even more, after muscle denervation. In the last case, the optimistic opinion is that ES is ineffective, while many experienced physiatrists and physiotherapists are against its use, because, in their opinion, ES could hinder or at least delay muscle reinnervation.
I will try to convince readers of this book that a more optimistic view is possible, if not mandatory, based on published solid experimental and clinical evidence, mainly on those papers in which I am present as a co-author (we will discuss again them in Chapter 10).
Here, I will discuss about ES as an experimental tool to mimic the discharge patterns of fast and slow motor neurons. We will refer to four applications in rat, sheep and canine models. Extensive discussion of the effects of different patterns of ES in animals and in patients are described in Chapter 13.1.1. Gerta Vorbová.

The frustrating aspect of this topic is that it appears to be rare or impossible to replicate some of our results. Do not ask me why.

3.2.1 Modulation by electrical stimulation of muscle fiber types in rodents

Slow-like electrostimulation switches on slow myosin in denervated fast muscle

Adult fast and slow skeletal muscles are composed of a large number of fibers with different physiological and biochemical properties that under neuronal control can respond in a plastic manner to a variety of stimuli. Although muscle cells synthesize muscle-specific contractile proteins in absence of motoneurons, after innervation the neuron controls the particular set of isoforms subsequently synthesized. However, agreement has not been reached on the mechanism, either chemotrophic or impulse-mediated, by which the nerve influences gene expression in the muscle. Here we report the effect on isomyosins of continuous, low-frequency (a protocol mimicking the discharge pattern of the slow motoneuron) direct electrical stimulation of a permanently denervated fast muscle, the extensor digitorum longus of adult rat [21]. After several weeks, unlike sham-stimulated muscle, the stimulated muscle showed a dramatic increase of the slow myosin light and heavy chains. Myosin light chains were identified by two-dimensional gel electrophoresis. The slow myosin heavy chain was clearly distinguished from fast and embryonic types by one-dimensional sodium dodecyl sulfate-polyacrylamide gel electrophoresis and orthogonal peptide mapping. The myosin change could be restricted to a portion of the muscle by the position of the stimulating electrodes [21]. Taking into account the morphologic appearance of the electrostimulated muscle and the large body of evidence demonstrating the absolute dependence of slow myosin on specific innervation, our observations indicate that at least the slow motoneuron influences the isomyosin genes' expression by the kind of activity it imposes on developing muscle fibers.

3.2.2. Modulation by electrical stimulation of muscle fiber types in sheep

Isomyosin changes after functional electrostimulation of denervated sheep muscle
Isomyosin analyses by biochemical, immunochemical, and histochemical investigations have been carried out in five sheep following unilateral recurrent laryngeal nerve paralysis and direct functional electrostimulation of the denervated cricoarytenoid posterior muscle [25]. Myosin light chains were identified by two-dimensional gel electrophoresis. Myosin heavy chains were analyzed by one-dimensional SDS-polyacrylamide gel electrophoresis. Slow myosin heavy chain was identified by orthogonal peptide mapping and immunochemistry. The stimulation effect at cellular level was determined using adenosine triphosphatase (ATPase) histochemistry. A dramatic increase of the type 1 fiber area (slow, fatigue-resistant fibers) could be seen after many weeks of an increasing regime of low-frequency direct electrical stimulation. Biochemically, the amount of slow myosin was always higher than in normal muscles. Some muscles were transformed almost completely to the slow type. At the time they were studied and with the methods employed, the expression of embryonic isomyosin was not observed. In conclusion, after numerous weeks of maintained functional activity, elicited by direct electrostimulation, the denervated muscle regionally showed areas of hypertrophy or at least lack of atrophy of slow myofibers without major signs of muscle damage [25].

Activity-Rest Regimen of Latissimus Dorsi Stimulation for Cardiomyoplasty: Isomyosins and Sustained Power of Sheep LD up to One Year
A prudent explanation of the clinical effect of dynamic cardiomyoplasty is that a minimal systolic assistance enhances the chronic elastic girdle effect of the transposed Latissimus Dorsi (LD). Slowness of the contraction-relaxation cycle and reduced power output of a fully conditioned LD limit its systolic support. Steady partial transformation of LD could increase power output by taking advantage of a faster contraction-relaxation cycle. To avoid full fast-to-slow transformation of LD, we chronically tested a daily activity-rest regimen of muscle stimulation in a simplified experimental sheep model. To mimic loss of

resting tension which occurs in cardiomyoplasty, sheep LD after tenotomy of distal aponeurosis were resutured in shortened position [176], and ITREL neurostimulators (Medtronic) connected to intramuscular electrodes were implanted according to the Medtronic Protocol. From two weeks after surgery shortened LD were burst-stimulated either 10 or 24 hr per day, the stimulators being programmed to the settings that elicited just fatiguing contractions in the shortened LD. Full-day activated LD were stimulated six months and then left unstimulated for additional six months, while the half-day activated muscles were stimulated up to one year. Two weeks after surgery and two, four, six and twelve months after stimulation, fusion frequency of tetanic contraction, power output, and fatigue resistance of LD were assessed [56]. To allow histological and molecular characterization of the two groups of stimulated muscles, LD were biopsied at six months of stimulation, and sheep sacrificed at twelve months to collect macrosopic anatomical records and perform molecular and histological analyses. After one year of 10 hr/day electrostimulation the gross anatomy of the LD were substantially conserved in comparison with contralateral, normal muscles (about 10% atrophy accompanied by minor fat infiltration and fibrosis). Isomyosin analysis shown that even after one year of stimulation the 10 hr/day stimulated LD contained large amounts of fast type myosin, in particular MHC2A, the isoform of fast-oxidative fibers, less prone to fatigue than the type 2B fibers of which normal LD of adult sheep is very rich. Though after six months of 24 hr/day stimulation LD were fully converted to type 1 myosin, after additional six months of resting these LD were white in appearance, atrophic (about 40%), fibrotic, and their isomyosin pattern as mixed as the LD stimulated 10 hr/day for twelve months. After four and six months of stimulation the frequency of tetanic fusion was higher (i.e., the contraction-relaxation cycle was faster) in 10 hr/day stimulated LD than in 24 hr/day stimulated LD; the difference disappeared at one year since the fusion frequency of the rested LD recovered to values of the one-year 10 hr/day stimulated LD. Of foremost importance is the fact that from two-month up to one-year of stimulation the sustained power output per muscle of the 10 hr /day stimulated LD (that is of the daily rested muscle) is three to four times higher than that of the 24 hr/day activated LD. From two and at least up to twelve months of stimulation the sustained power of the "daily-rested" LD become higher than that of the heart at rest. In conclusion, results of our activity-rest daily regimen are encouraging: sheep LD loses very low contractile mass, and its power is equal or bigger than that of the left ventricle, since it seems to achieve a stable intermediate state of fast-to-slow transformation when stimulated for ten hours a day.
After such encouraging evidence, I was brave enough to accept a request of cardiac surgeons of Padua University to test the Demand Dynamic Cardiomyoplasty in patients. The clinical results of end-stage cardiac failure patients will be discussed in Chapter 6.

3.2.3 Modulation by electrical stimulation of muscle fiber types in canines

Chronic intermittent stimulation of the thyroarytenoid muscle maintains dynamic control of glottal adduction

Patients with laryngeal motor control disorders need improved dynamic glottal closure for speech and swallowing. To evaluate the functional outcome of intermittent chronic thyroarytenoid muscle stimulation in an animal model, 6 canines were implanted with bilateral Medtronic Xtrel systems containing Peterson-type electrodes in the inferior and

superior portions of the thyroarytenoid muscle [58]. Stimulation was on one side only at 60 Hz, for 5 s on and 5 s off, over 8 h, 5 days per week, up to 8 months. Monthly video recordings were done under anesthesia to measure the voltage threshold for detectable movement on each side, and vocal fold displacement and velocity during maximal stimulation of each side. Movement thresholds were lower in the inferior portion of the thyroarytenoid muscle ($P \leq 0.0005$). Movement velocity was greater on the stimulated than on the nonstimulated side after 3 to 8 months ($P = 0.039$). No differences in the percentage distribution of different myosin heavy chain types were found between the stimulated and nonstimulated muscle samples. Sustained dynamic glottal adduction with no alteration in thyroarytenoid muscle function or fiber type was achieved with intermittent stimulation over 8 months.
The results suggested that chronic intermittent thyroarytenoid stimulation has good potential for improving airway protection in dysphagia

3.3 Towards applications of research on isomyosins: the collaboration with hypertension specialists of the University of Padua

The publications we will discuss in this sub-chapter are the result of my previous interest in developing molecular methods based on nanograms of myosin, but the greatest merit goes to Luciano Dalla Libera, very active in applying analyzes of the proteolytic products of myosin and of purified heavy chains as tools to study adaptation of the heart ventricle and skeletal muscle in different experimental models, from rodents to turkey and finally human patients. As soon as our publications demonstrated in animal models (more often muscle denervation) that the analytical methods proved reliable, colleagues from the medical faculty of the University of Padua contacted us to study their clinical problems. The first application, thanks to Luciano's friendship with medical specialists in arterial hypertension, was in fact the analysis of rat cardiac ventricles subjected to chronic pressure overload.
It was for those collaborations, which began in 1986, but lasted until Luciano's retirement in 2003, that Barbara Ravara began to collaborate with him.

Isomyosin redistribution in chronic pressure overload: comparison between peptide mapping and electrophoresis under non-denaturing conditions
Chronic pressure overload induces a redistribution in myosin isoenzymes of rat cardiac ventricles, as demonstrated by Ca^{2+}-activated ATPase activity, electrophoresis under non-denaturing conditions, and immunohistochemistry. They compared, in groups of renal hypertensive rats and control rats, the isoenzymatic pattern of rat cardiac ventricles obtained by electrophoresis under non-denaturing conditions with those observed after digestion of heavy chains with S. Aureus V8 protease [17]. In hypertensive animals in which a shift towards the "slow" isomyosins V2 and V3 was evident, the peptide mapping always gave rise to a band that was not present in controls. We consider this peptide a marker of redistribution towards "slow" isoforms. Thus mapping of the Cleveland peptide appears to be a simple and useful method for evaluating differences in isomyosin composition, at least between hypertrophic, pressure overloaded and normal rat ventricles. In our experience this technique was simple, the patterns obtained from highly purified substrates very reproducible and the digestions allowed easy and clear comparisons.

Ventricular myosin pattern of spontaneously hypertensive turkeys is unaffected by labetalol treatment.

In most animal species, left ventricular hypertrophy due to pressure overload is associated with a beneficial "slow" rise in isomyosin V3. In contrast, in spontaneously hypertensive turkeys, the development of left ventricular hypertrophy is associated with the synthesis of a "fast" V1-like isomyosin, with a high incidence of heart failure. This could be related to the high levels of catecholamines present in these animals. For this reason we have studied the ventricular myosin pattern after the lowering of blood pressure and the regression of cardiac hypertrophy obtained by means of labetalol, and the alpha and beta-blocking drug that inhibits the effects of catecholamines. From 2 to 32 weeks of age, 22 turkeys were treated with increasing doses of labetalol (20 to 35 mg / kg body weight per day) and 16 other turkeys were given placebo. Blood pressure and heart rate were periodically measured by an indirect method. After sacrifice, degree of cardiac hypertrophy was assessed by body weight / biventricular weight ratio, ventricular myosin was purified, Ca^{2+}-activated ATPase activity evaluated, and ventricular myosin pattern determined by gel electrophoresis of myosin heavy chains on two-dimensional gel. Plasma and cardiac catecholamines were measured by high performance liquid chromatography. Throughout the study period, blood pressure and heart rate were significantly reduced in labetalol-treated animals compared to untreated ones. At the end of the study period, the ventricular mass was significantly lower in the labetalol group. However, no differences were observed in ventricular myosin pattern and levels of Ca^{2+} activated ATPase activity between the two groups. An increase in plasma catecholamines and only a slight, but not significant, increase in cardiac catecholamines was found in the labetalol group. These data indicate that in spontaneously hypertensive turkeys, the synthesis of "fast" V1-like isomyosin is not affected in cardiac hypertrophy by known pathophysiological stimuli such as blood pressure and catecholamines [24].

Specific changes in skeletal muscle myosin heavy chain composition in cardiac failure: differences compared with disuse atrophy as assessed on microbiopsies by high resolution electrophoresis.

In congestive heart failure (CHF), the muscles of the lower limbs develop a myopathy with atrophy and transformation of slow fibers into the fast ones. We wanted to test the hypothesis that this myopathy is specific and not just due to detraining, comparing patients with different degrees of CHF with patients with severe muscle atrophy due to disuse [45]. From needle biopsies of 50-150 micrograms of gastrocnemius muscle we separated the three isoforms of myosin heavy chains (MHC) with an electrophoretic micromethod. We studied five patients confined to bed for more than a year due to stroke, with severe disuse atrophy, but normal ventricular function and nineteen CHF patients. Seven were the age-matched controls. We determined the percentages of MHC1 (slow isoform), MHC2a (fast oxidative) and MHC2b (fast glycolytic) by densitometric scanning, correlating them with heart failure severity indices. Ejection fraction was 42.5 (SD 15.2)% in CHF, 59.5 (1.0)% in disuse atrophy, and 60.3 (1.4)% in controls ($P < 0.001$ versus both of them). The degree of muscle atrophy, calculated from the body mass index / cross-sectional area of the gastrocnemius, showed a profound degree of atrophy in patients with muscle disuse [0.94 (0.39)]. This value was worse than controls and patients with CHF. Atrophy in patients with CHF was also greater than in controls. MHC1 was lower

in CHF than in disuse atrophy while MHC2b was higher. There was a similar trend for MHC2a. Within the CHF group there was a positive correlation between NYHA class and MHC2a and MHC2b and a negative correlation between NYHA and MHC1 class. Significant correlations were found for ejection fraction, diuretic consumption score, exercise test tolerance, and degree of muscle atrophy.
In conclusion, CHF myopathy appears to be specific and unrelated to detraining. The extent of MCH redistribution correlates with the severity of the disease. The electrophoretic micromethod is very sensitive and reproducible. The biopsies are well tolerated since pain is equivalent to that of a normal intramuscular injection of drugs, so that they can be repeated allowing a complete follow-up.

Vescovo G, Ceconi C, Bernocchi P, Ferrari R, Carraro U, Ambrosio GB, Libera LD. Skeletal muscle myosin heavy chain expression in rats with monocrotaline-induced cardiac hypertrophy and failure. Relation to blood flow and degree of muscle atrophy. Cardiovasc Res. 1998 Jul;39(1):233-41. doi: 10.1016/s0008-6363(98)00041-8. PMID: 9764203

In congestive heart failure (CHF), the skeletal muscle of the lower extremities develops a myopathy characterized by atrophy and transition from slow to fast fibers. The mechanisms responsible for those changes are still unclear. We investigated the influence of blood flow and degree of muscle atrophy on the myosin heavy chain (MHC) composition of the soleus and EDL of rats with hypertrophy and right ventricular failure. CHF was induced in 16 rats by injecting 30 mg / kg of monocrotaline. Eight animals had the same dose of monocrotaline, but resulting in compensated right ventricular hypertrophy. Two groups of age- and diet-matched control animals (nine and five respectively) were also studied. The relative percentage of MHC1 (slow isoform), MHC2a (fast oxidative) and MHC2b (fast glycolytic) was determined by densitometric scanning after electrophoretic separation. The relative weights of soleus and EDL (muscle weight / body weight) were taken as an index of muscle atrophy. Skeletal muscle blood flow was measured by injecting fluorescent probes. The CHF and Control rats showed a similar degree of atrophy in both soleus and EDL. In CHF rats these two muscles showed statistically significant redistribution of MHC to fast-type isoenzymes. Similar changes were not found in the muscles of animals with compensated hypertrophy. No correlation was found between the MHC pattern and relative muscle weight in animals with CHF. The blood flow of the soleus in CHF rats was significantly lower than that of control, while differences were not found in EDL. Skeletal muscle myopathy characterized by a shift of MHCs to fast-type isoforms occurs in rats with CHF. The magnitude of the displacement correlates neither with the degree of atrophy nor with the blood flow of skeletal muscle, suggesting that these two factors do not play a main role in pathogenesis of the myopathy.

Skeletal muscle abnormalities in rats with experimentally induced heart hypertrophy and failure.

In congestive heart failure (CHF), skeletal muscle function and metabolism are abnormal. To evaluate whether the reduced oxidative capacity of skeletal muscles in CHF is due to an altered use of O_2, CHF was induced in rats by injecting 50 mg / kg of monocrotaline. Several animals received the same dose of monocrotaline, but only compensated for right ventricular hypertrophy and no signs of congestion were found. Two groups of age- and diet-matched control animals were also studied. In the soleus and EDL, we studied the blood flow of the skeletal muscle, the oxidative capacity, and the respiratory function of the skinned muscle fibers. In CHF, we observed a decrease in muscle blood flow (statistically significant in the soleus, $p < 0.05$ compared to controls). In compensated rats, a similar trend in blood flow was observed. A significant reduction in high-energy phosphate and a shift in redox potential towards the accumulation of reducing equivalents was observed in both the soleus and the EDL [177]. The reduction in energy charge was not related to the decrease in blood flow. In skinned myofibers, the ratio of O_2 used in the presence and absence of ADP (a phosphorylating efficiency index) was reduced in soleus and EDL. The activity of the various complexes of the respiratory chain was studied using specific inhibitors, highlighting important anomalies at the level of complex I. In fact, the inhibition of VO_2 by rotenone was decreased both in soleus and in EDL. In rats with CHF, abnormalities of oxidative phosphorylation of muscles occur and respiratory chain I complex appears to be primarily affected. The metabolic alterations of skeletal muscle in CHF can be explained, at least in part, by a reduced use of O_2.

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Chapter 4
Post-damage muscle regeneration is independent from motor neuron innervation, macrophage-myoblast interactions and beyond

4.1. Post-damage muscle regeneration is independent from motor neuron innervation
4.2. Macrophage-myoblast interactions and beyond

What I will describe in this chapter are studies that are fullly dependent on my collaborators, although I have contributed concepts and new strategies, usually in informal discussions with them. The better examples are the results of a study on macrophage-myoblast interactions, which revealed for the first time in the literature that macrophages, in addition to being the essential scavenger cells that remove debris from damaged muscle fibers in muscle regeneration, actively contribute to activation of the satellites cells and to proliferation of myoblasts. Without the expertises of Marcello Cantini, the father of muscle cell culture at the University of Padua, none of the following results could have been achieved [18,19,21,25,28,35].
But I will start with a topic that had equal if not more important applications for human neuromuscular disorders, specifically the demonstration in an in vivo experimental rat model that early phases of post-damage muscle regeneration is independent from motor neuron innervation.

CHAPTER 4.1. Post-damage muscle regeneration is independent from motor neuron innervation

Myosin light and heavy chains in rat muscle regenerating in absence of the nerve in both fast and slow muscles: transient appearance of the embryonic light chain, but later on accumulation of fast-like myosin

To test whether chronically denervated muscle is able to regenerate after a myotoxic focal lesion (Injection of anesthetics into a low blood circulation muscle tissue) we examined myosin in fast and slow rat skeletal muscles (EDL and soleus respectively) after the injection of bupivacaine into permanent denervated limbs rendered transiently ischemic [9]. Histological analyzes confirmed the myotoxic damage and after four days the presence of small myofibers with central nuclei (regenerating myotubes/myofobers). Four days after the injury, two-dimensional gel electrophoresis revealed the presence of the embryonic light chain in the portions of muscle that showed a homogeneous population of new small fibers on histological examination. Two weeks after the myotoxic lesion this subunit was absent, while the two light chains, LC1F and LC2F characteristic of myosin of the fast muscles in adult rats, became prominent both in the rapid muscle (EDL) and in the slow muscle (soleus). One month after the injury, the soleus muscle was still denervated and with a pattern of light chains typical of fast-twitch muscles (i.e. those observed in the myosin from normal adult EDL). Native myosin gel electrophoresis and

peptide mapping of the electrophoretically purified heavy chains have confirmed that the muscle regenerating in the absence of the nerve accumulated a myosin that had the general characteristics of a fast myosin, not even slow in a muscle almost homogeneously formed by slow fibers likes the soleus is, but contained well-defined differences from the first, on whose nature we could not pronounce in the discussion of the work [9].
In conclusion, these observations are in favor of the hypothesis that regenerative events in permanent denervated muscle may contribute to endow it with fast-, or rather fast-like myosin, while it is confirmed that the slow myosin decrease abruptly up to disappear also in almost pure slow muscles (soleus).

Early stages of maturation, followed by degenerative alterations, and ongoing regeneration of skeletal muscle fibers occur in absence of innervation

We looked at muscle regeneration brought on by bupivacaine in the permanently denervated soleus muscle of adult rats to find out how much skeletal muscle regeneration depends on input from peripheral nerve We investigated the degree of maturity obtained by regenerated myofibers and their capacity to regenerate once again following recurrent bupivacaine injury using light and electron microscopy. Morphometric investigations revealed that the regenerated denervated fibers increased in diameter, matured, and then became atrophic within the first two weeks following damage [22].
The morphological properties of mature fibres are visible by electron microscopy, although complete classification into adult fibre types is not achieved. This is consistent with earlier reported biochemical findings. Repeating the bupivacaine treatment on regenerated muscle resulted in a new fresh phase of regeneration, proving that myofibers that have recovered from injury but lack innervation retain their natural capacity to repair themselves, i.e., also satellite cells populate the regenerating tissue. In the later stages, when the atrophy and degeneration of the fibers became evident, the regenerated denervated muscle also underwent spontaneous, albeit poor, regeneration. The proportion of myosatellite cells remained high at the same time. These morphological findings lend even more credence to the idea that spontaneous fiber regeneration helps maintain muscles that have been chronically denervated rich in severely atrophic muscle fibers [22].

CHAPTER 4.2. Macrophage-myoblast interactions and beyond

Macrophage – Myoblast interactions

Myofibers are reconstituted by the proliferation and fusion of muscle precursor cells, i.e., of so called satellite cells, when skeletal muscle is injured. One of the critical events is the peak accumulation of macrophages after 48 hours at the damage site preceding satellite cell activation and proliferation. Macrophage-muscle cell interactions are complex, and the majority were unknown, beside the obvious scavenger role of leukocytes. The persistence of inflammatory cells in skeletal muscle could be critical for the viability of myofibers [35]. On the other hand, macrophages release factors that increase myoblast number and fusion to form myotubes thus helping muscle regeneration after traumatic, myotoxic or neuromuscular diseases' lesions. I would like to emphasize that we were pioneers in this field and that our in vitro observations, with the main contribution of Marcello Cantini, continue to be cited in the international literature [21,48]. Marcello was also the key person to explore also related research topics, specifically, in vitro and in vivo gene transfer into satellite cell from regenerating muscle [18], Functional in vivo gene transfer into the myofibers of adult skeletal muscle [40], Effects of beta 1-integrin antisense phosphorothioate-modified oligonucleotide on myoblast behaviour in vitro [19].

Impressed by the clinical implications of our findings, Francesco Mazzoleni, Head of the Plastic Surgery Unit of the University General Hospital of Padua, contacted me to explore the potential of a cell therapy approach to reconstruct the ablated or transposed rectus abdominis muscle in an in vivo model in rodents [39]. In fact, plastic surgeons use the rectus abdominis muscle to repair or rebuild lost tissue, but its useful and acceptable transplant for the benefits it offers to the patient can compromise the function of the abdominal wall. It was the beginning of a long collaboration [117], which still exists even after his retirement with his helpers and pupils, in particular between Vincenzo Vindigni and my students Sandra Zampieri and Barbara Ravara [178].

Inhibition of FasL sustains phagocytic cells and delays myogenesis in regenerating muscle fibers.

In a 2001 paper [35], we demonstrated that FasL plays a role in the resolution of muscle inflammation. We analyzed inflamed muscles of normal mice treated from day 3 to day 8 with a FasL inhibitor (Fas-Ig) or with control immunoglobulins (Ig). Treated muscles were collected at 3, 5, and 10 days. The treatment with recombinant Fas-Ig protein induced a severe persistence of inflammatory cells at 5 days and 10 days from injury. Myofiber regeneration was highly impaired. Apoptosis of phagocytic cells was absent during Fas-Ig treatment, but apoptotic, mononucleated cells appeared at day 10, 2 days after the suspension of Fas-Ig administration. The time course of FasL expression during muscle inflammation, at mRNA and protein level, reveals a peak during myoblast proliferation. The peak of FasL expression coincides with the peak of apoptosis of phagocytic cells. In situ hybridization shows the co-expression of FasL and MyoD mRNA in mononucleated cells, i.e., myoblasts. Experiments on myoblast cell culture confirmed the expression of FasL in myoblasts. The findings shown here indicate one of the pathways to control myoblast-macrophage interaction and might be relevant for the control of inflammatory cells in muscle tissue. Perhaps altering FasL expression with recombinant proteins could ameliorate inflammation in degenerative myopathies and up-regulate muscle regeneration.

Macrophage-released factor stimulates selectively myogenic cells in primary muscle culture.

In addition to their well-known role as a scavenger cell, there is now direct evidence of a mitogenic role of macrophages in regenerating muscle. We have utilized an in vitro model to directly investigate and prove that macrophages increase myoblast growth not only of satellite cells, but also of primary myoblasts [21]. Rat muscle cells were cultured in the presence or absence of exudate macrophages obtained by peritoneal washing after thioglycollate broth injection. Macrophage coculture increases several times the myoblasts/myotubes yield. This effect is particularly evident in muscle culture conditions in which fibroblast growth is predominant over myoblast proliferation, suggesting a myoblast selective mitogenic effect of macrophages. The results are confirmed by quantitative analyses of both DNA and skeletal muscle-specific-contractile proteins by gel electrophoresis and immunocytochemistry. Experiments with macrophage-conditioned media show this effect is mediated by soluble factors. This growth factor-like activity, which has been shown to be acid-stable and heat-labile, exerts its effects not only on specialized satellite cells during muscle regeneration, but also has a broader mitotic activity on all myogenic cells. In view of the role of muscle regeneration in muscle diseases and of the perspectives offered by gene therapy via myoblasts, we strongly believe that our results opened new opportunities in removing many of the clinical constraints associated with repair and cell transplantation.

ED2+ macrophages increase selectively myoblast proliferation in muscle cultures

We have previously shown by coculturing myoblasts and macrophages that myotube formation is strongly increased in vitro by the presence of an acid stable, heat-labile, soluble growth factor(s) secreted by macrophages [21]. In a following paper we obtained macrophages from peritoneal washing which also contained limited amounts of other cells such as lymphocytes and mesothelial cells. We demonstrated that an ED2-positive (ED2+) macrophage subpopulation is responsible for myoblast enhanced proliferation [48]. ED2+ macrophages were separated by a magnetic-activated cell sorter (MACS) using a monoclonal antibody against ED2, a membrane antigen peculiar to macrophages. Both ED2+ macrophages and their conditioned medium increased myotube formation when added to primary muscle cultures. Furthermore we demonstrated that muscle growth induced by macrophages is mainly the consequence of an increased myoblast proliferation by showing the presence of an increased number of MyoD-positive (MyoD+) myonuclei [48].

Gene transfer into satellite cell from regenerating muscle: bupivacaine allows beta-Gal transfection and expression in vitro and in vivo.

A large bulk of experimental evidence suggests that myogenic cell transfer can be regarded as a promising therapeutic approach in the cure of inherited pathologies. In particular, it has been shown that primary myoblasts obtained from embryonic or neonatal muscles allows the recovery of the normal phenotype in defective muscle tissues. The utilization of this approach in clinical settings still bears heavy limitations. Apart from the legal and ethical difficulties, the use of muscles obtained from aborted fetus is challenged by a large risk of rejection, due to the incompatibility between donor and recipient. In this context based on the genetic alteration and reimplanting of the patient's own satellite cells, appears an approach attractive. Myoblasts derived from satellite cells are the obligate candidates for experiments, but the production of sufficient cell numbers is a major problem. Local anesthetics [Bupivacaine (1-n-butyl-DL-piperidine-2-carboxylic acid-2, 6-dimethyl anilide hydrochloride) and related molecules] had been used to induce myofiber damage (and thus satellite cells proliferation) and thereby may represent a tool for increasing the yield of myoblasts from adult muscles. We have shown that satellite cells obtained from adult muscles after bupivacaine injection can be transfected in vitro and that the transfected gene is expressed in vitro and in vivo, after reimplantation of the modified myoblasts in recipient muscles [18].

Functional in vivo gene transfer into the myofibers of adult skeletal muscle

The postmitotic nature and longevity of skeletal muscle fibers permit stable expression of any transfected gene. Direct in vivo injection of plasmid DNA, in both adult and regenerating muscles, is a safe, inexpensive, and easy approach. We presented an optimized electroporation protocol based on the use of spatula electrodes to transfer cDNA in vivo into the adult myofibers of an anatomically defined muscle, which could be functionally characterized. In our hands, about 80% of adult myofibers were transfected in vivo by different plasmids for GFP fusion proteins or for beta-galactosidase [40]. The luciferase activity increased several orders of magnitude when compared to standard DNA delivery. In an anatomical defined muscle, the wide gene transfer was comparable to or better than that of retrovirus delivery, that recently has been shown to be prone to severe side-effects in human clinical studies. Furthermore, with our method the tissue damage was greatly decreased. Thus, the present work describes in vivo functional electrotransfer of genes in adult skeletal muscle fibers by a protocol that is of great potential for gene therapy, as well as for basic research.

Telthon Meeting . Neaples, Italy 1993

Effects of beta 1-integrin antisense phosphorothioate-modified oligonucleotide on myoblast behaviour in vitro.

Myoblasts genetically modified in vitro and then injected in vivo are safe and efficient options for gene therapy. Although satellite cell isolation is achieved routinely, their proliferation potential in vitro remains a limiting factor for cell transplantation under clinical conditions. We investigated the role of reversible inhibition of gene expression by antisense oligonucleotides on myogenic cell proliferation. The addition of antisense oligonucleotides to myoblast cultures was used to specifically inhibit the expression of the beta 1-integrin subunit gene. We have demonstrated that the effects of multiple pulses of an antisense phosphorothioate oligodeoxynucleotide on substrate attachment and myoblast proliferation were dose-dependent [19]. The addition of antisense to the rat myoblasts caused cell rounding and most of the cells detached after several days of culture. A single pulse did not show consistent effects, while in the presence of continuously administered antisense, the relative number of myoblasts in the treated muscle culture increased. We have no evidence of inhibition of myoblast fusion under these conditions. On the other hand, [3H] -TdR incorporation, total DNA and total cell number decreased in antisense-treated cultures, thus demonstrating an inhibitory effect of phosphorothioate oligonucleotides on DNA synthesis. These side effects could be overcome by replacing the phosphorothioate with unmodified oligonucleotides, thus decreasing the half-life of the antisense, but also its toxicity. The overall results suggest a potential role of the integrin antisense strategy in modulating the proliferation potential of myoblasts.

Reconstruction of ablated rat rectus abdominis by muscle regeneration.
Skeletal muscle regeneration is a powerful, naturally occurring process of tissue reconstruction that follows myofiber damage secondary to myotoxic injury that does not normally affect the tissue circulation and scaffold. The ablated tissue, in traumatology and free muscle grafts, is frequently replaced by scars. The final outcome is poor even after *in situ* myoblast seeding of the harvested muscle. The goal of one of our studies was to identify protocols to reconstruct muscle tissue, even in such adverse environments. We applied a step-by-step approach to identify factors favoring the survival of autologous satellite cells and, thus, muscle regeneration. In a rat model of full-thickness rectus abdominis muscle ablation, autologous myoblasts were isolated from the explanted rectus abdominis and seeded in a homologous acellular matrix immediately after wall reconstruction (group 1, five animals). In group 2 (five animals), the ablated rectus abdominis was autografted in situ. In a third group of five rats, Marcaine was injected into both the autograft and the surrounding abdominal wall muscle. Three weeks after surgery, serial cross-sections of the reconstructed abdominal wall were stained with hematoxylin and eosin or embryonic myosin antibody, a well-characterized molecular marker of early myogenesis in development and regeneration. Percentages of the patch area covered by regenerated myofibers were determined by morphometry. When autologous myoblasts were seeded in a homologous acellular matrix, the only myofibers observed to regenerate were those along the border of the patch. Autografting of the middle third of the rectus abdominis muscle similarly resulted in scar formation. The few muscle cells in the graft core were scanty myoblasts that could be detected only by monoclonal embryonic myosin antibody. Although negative for myofiber regeneration, the results in both cases confirmed the mechanical patency of the patches with regard to abdominal organ support. Myofibers were successfully regenerated in the graft by injecting Marcaine into both the autograft and the surrounding muscles. Three weeks after surgery, the patch was paved with young, centrally nucleated myofibers intermixed with young myofibers and myotubes expressing embryonic myosin. The difference in percentage of patch area covered by regenerated myofibers in group 3 (Marcaine injection around the patch, 81.6 +/- 3.0 percent) (mean +/- SD) versus either group 1 (Myoblast-seeded acellular patch, 18.0 +/- 3.0 percent) or group 2 (Autograft, 25.8 +/- 7.0 percent) was statistically significant on independent t test analysis ($p < 0.0001$). Even an acellular matrix showed some myofiber regeneration after surrounding muscles had been injected with Marcaine. Our was the first successful evidence of muscle reconstruction after full-thickness ablation of the middle third of the rectus abdominis. Muscle regeneration seems to be the result of successive waves of migration of angioblasts, of activation of satellite cell and then myoblasts from the muscles surrounding the patch.

The results strongly suggest that revascularization and successive coordinate proliferation of the seeded cells are required for myoblasts to be able to migrate into the patch [39].

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Chapter 5
Muscle apoptosis: a debated issue

In my research life I have sometimes found myself on the wrong side of the stream of internationally published results. Sometimes I was wrong and I was wasting my time and my resources and those of my collaborators, but in a few cases we were on the right side. Either way, surfers say it's exciting to go against the tide.
This chapter is one example proving that sometimes I was right. In fact, it led to a change of direction towards an interesting area of research: muscle apoptosis, also known as programmed cell death. For a recent general reveiw of cell apoptosis sees: Kopeina GS, Zhivotovsky B. Programmed cell death: Past, present and future. Biochem Biophys Res Commun. 2022 Dec 10;633:55-58. doi: 10.1016/j.bbrc.2022.09.022. PMID: 36344162.[179].
Rejected by leading myologists, we used electron microscopy and molecular analyzes to show that accepted markers of apoptosis are present in the mouse muscle two days after a night of voluntary running (up to 5 km during the first night). This observation in a few year has been extended by us to other experimental models in vitro and in vivo and in human cases of muscular dystrophies [43,46,47,51,52,55,59,62.]
But I have to start from how this story started, but point out that Marco Sandri and Marzena Podhorska-Okolow have the greatest long-term merits.
Prof. Claudio Franceschi, an immunologist specialized in the study of centenaries, spent trhee years at the University of Padua, working in a small lab together with Paola Arslan, Marcello Cantini and a few pupils. One day he entered my lab asking support to demonstrate that both myocardiocytes and skeletal muscle fibers may undergo apoptosis, a process that occurs every day in the cells that die and regenerate continuously (labile tissues), as the cells of the epidermis, of the intestine and those of blood. The strange name describe the fall of leaves that occur every autumn in the decidual trees, whose leaves fall donw to the soil to nurtur the trees in the following spring. Apoptosis is indeed the greek word that describe the fall from the top of the tree (apo-ptosis means from high-fall). Despite we know well now that the skeletal muscles (but not the cardiac cells, at least in large mammals) may death and regenerate, it was obvious in my mind that a kind of normal event must occur unrecognized in the tissue to explain the impressive potential of regeneration of the skeletal muscle tissue.
Why not, unrecognized cycles of death by apoptosis and regeneration?
On the other hand, only severe trauma and ischemia or genetic muscle diseases were accepted as to causing death and regeneration of the muscle fiber of our muscles [180,181], not functional events like those occurring in runners.
However, we had previously studied the impressive damage and regeneration that occurs in a mouse, to which a free wheele is offered after months or years of sedentary life in a small cage. They run the first night up to 5 km and show extensive muscle regeneration during the next week. It was in my mind the best experimental setting to study skeletal muscle apoptosis during the first two days after running [43].
I was right.
Starting from that observation we demonstrated in vitro and in vivo that the muscle tissue may present the well known ultrastructural and molecular markers that allow to

quantitate apoptosis in labile tissues (blood cells, cells of the skin and of the internal mucoses) [46,47,51,52,55,59,62].
In 1995 the first world meeting on the role of apoptosis in development, damage and repair of skeletal muscle and heart was held in Abano Terme (Padua). Participation included molecular and cellular myologists and clinicians and there we presented our findings on skeletal muscle apoptosis in dystrophic mice after a night of spontaneous running in free wheel [182]. We subsequently published two reviews [47,59] that helped establish muscle apoptosis as an important topic for at least the next twenty years [183].

A dream had come true.

Sudden Spontaneous Exercise Increases Myonuclear Ubiquitination and Apoptosis of Dystrophin Deficient Muscle
Apoptosis or programmed cell death is an active multi-step process characterized by morphological, biochemical and molecular events, which requires coordinated regulation of specific genes [184]. This program of cell suicide plays a major role in development, in tissues with high cellular turnover and contributes to the pathogenesis of several human diseases [185]. In vitro experiments on normal and dystrophin deficient myoblasts [43,186] add information on regulation of myoblast proliferation, differentiation and death during regeneration of skeletal muscle, but few information is available on the role of apoptosis in adult muscles. Some observations come from studies on myocardium, since it can display apoptosis after ischemia and reperfusion [187, 188]. Recently we shown in line with other published results apoptosis in skeletal muscle of adult mdx mice in vivo [189, 190,43, 186,191].
One of the first genes up-regulated during programmed cell death is the ubiquitin gene [192]. In mammalians, different conditions of muscle wasting reveal an increased expression of ubiquitin [193]. To determine whether ubiquitin plays a role in progressive damage of dystrophic muscle we studied myofibers of mdx mice after a mild spontaneous exercise. Sedentary mdx mice and congenit BALB/c mice were used as controls.
Light mycroscopy of muscles of dystrophic mice, both al rest and exercised, shows foci of muscle injury with inflammatory cells, small regenerating myofibers and myofibers with centrally located myonuclei, while muscles of sedentary BALB/c mice present homogeneous well-defined fibers with peripheral myonuclei. After immunoreaction with an anti-ubiquitin antibody BALB/c myofibers appeared poorly reacting due to low level of ubiquitin expression in physiological conditions, while some cytoplasmic stain distinguishes slow and fast fibers. Myofibers of sedentary mdx mice present a positive reaction in some peripherally-located nuclei, and in small regenerated myofibers.
On the other hand, in mdx muscle after exercise many centrally located myonuclei are positive both in small regenerating and in mature myofibers, while foci of inflammation are negative. The high turnover of ubiquitin and the 24 hr of rest after exercise exclude that ubiquitin is induced in parallel with Heat Shock Proteins by the stress due to exercise per se. On the other hand, it is well documented that ubiquitin is tightly bound to histones or to some other proteins of the nuclear matrix after DNA damage.
When the slides were processed for in situ analysis of DNA fragmentation, numerous myonuclei in exercised muscles of mdx mice were positive for apoptosis. As we recently described , muscles of sedentary mdx mice show 2-3% of apoptotic myonuclei while BALB/c muscles are negative [15]. The increase of the percentage of positive myonuclei

for ubiquitin in mdx muscles after exercise correlates with the increased number of apoptotic myonuclei.

When DNA analysis by pulsed field gel electrophoresis is performed on isolated myonuclei the results reveal that: i) No DNA fragments are detectable in BALB/c muscles; ii) some fragments at 200 kb and at 50 kb are present in muscles of sedentary mdx mice in good correlation with the 2-3% of apoptotic myonuclei found with Apo-Tag kit; and iii) an increased amount of DNA fragments are detected in muscles of mdx mice after sudden spontaneous exercise together with a smeared pattern of DNA, which suggests a complete digestion of DNA typical of the necrotic process.

The possibility that inflammatory cells contributed to DNA fragmentation is not excluded, but a myonuclear origin of the DNA fragments is suggested by the presence in myonuclei of apoptotic features detected by in situ nick-end labelling and by electron microscopy. Normal myofibrillar fields around apoptotic nuclei distinguish myonuclei from nuclei of satellite cells, endothelia, fibroblasts and eventual invading macrophages. In 15 % of nuclei of mdx muscles after exercise electron microscopy documents typical features of apoptosis with condensed chromatin around myonuclear membrane.

Massive activation of proteases is one candidate in triggering cell apoptosis and it is implicated in nuclear proteins catabolism and in lamin-DNA fragmentation. Which is the protease system associated is still unknown, one candidate could be ubiquitin. Ubiquitin binding proteins for successive degradation, influences life of several important proteins for apoptosis such as p53, c-myc, BAG-I, and a relationship between ubiquitin and DNA fragmentation was clearly shown. When the distribution of ubiquitin and ubiquitin-conjugated proteins was investigated by SDS-PAGE and Western blot in supernatants and myofibrills of muscle homogenates, low level of free ubiquitin is constantly shown in all studied muscles, in good agreement with published data. This observation could be related to the ceased expression of stress proteins two days after exercise, since shock and other stress cause only transiently increase free ubiquitin. In the soluble fraction of exercised mdx muscle we detect an increased content of ubiquitin-conjugated proteins compared with muscles of both mdx and BALB/c mice at rest: the exercised mdx muscles contain at least ten times of the amount present in the muscles of sedentary mice. Similar results are obtained in the myofibrillar fractions. The highest level of ubiquitination is detected in mdx mice after exercise. Densitometry of ubiquitin-reacting bands shows that ubiquitin linked to contractile proteins increased two-three times in comparison with the ubiquitin amount of the mdx and BALB/c sedentary mice muscles. On the other hand, in situ analysis suggests that exercise-induced ubiquitin is preferentially linked with nuclear proteins. This has been related with DNA damage and could be important for fragmentation of histones or nuclear matrix proteins, as lamin, or for changes of nuclear structure during the apoptotic process. Also, some myoplasm proteins were labelled indicating that proteinase activity is generalized. The widespread expression of ubiquitin and its capacity to link with multiple nuclear and cytoplasmic proteins suggests a major role in regulating apoptosis and other mechanisms of muscle damage. Recent in vitro studies underlay the role of cell death in regulating myoblast proliferation and fusion and this could be relevant in regenerating myofibers of mdx mice, in particular after exercise. On the other hand, in vivo apoptotic myonuclei were found in mature myofibers indicating a pathogenetic role of the mechanisms of programmed cell death in exercise-induced muscle damage in dystrophynopathies. The secondary pathogenetic processes by which a lack of dystrophin/dystrophin associated glycoproteins leads to progressive muscle degeneration in muscular dystrophies is an open issue. A number of possible mechanisms have received attention: changes in plasma membrane permeability, a specific defect in muscle intracellular free calcium homeostasis, and a decreased mechanical stability of the muscle plasma membrane and of the sarcomers. It is general expectation that exercise-induced damage plays a role in the myodystrophic process and that modifications of the training programs of muscles may have some importance in influencing muscle degeneration in patients with muscular dystrophies. It is well known that exercise in an unaccustomed muscle provokes mild injury, soreness and lactic acid accumulation. Our observations that a sudden spontaneous running in unaccustomed animals increases the number of apoptotic myonuclei in differentiated muscle fibers of adult mdx mice shed a light on the pathogenesis of the post-exercise muscle injury. We suggest that exercise-induced damage or fatiguing exercise itself activates the program of cell suicide in mdx muscle possibly because of unbalanced calcium homeostasis or because of an increased generation of reactive oxygen species during reperfusion. Muscle cells initiate the apoptotic process activating the process of DNA fragmentation and the protease system. Only some myofibers reach the final steps of apoptosis, i.e., chromatin condensation and apoptotic body formation. In spite of the clear difference between sedentary and exercised mdx mice observed, myonuclei showing apoptotic features by electron microscopy were one/half·of positive myonuclei for both ubiquitin and in situ DNA end-labeling.

In conclusion, exercise-induced muscle damage in mdx mice suggests new roles of ubiquitin related to nuclear events, and it provides evidence for a new and provoking pathogenesis in dystrophinopathies, which could open new pharmacologic strategies in managements of exercise-induced muscle damage and muscle dystrophies.

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Apoptotic myonuclei in human Duchenne muscular dystrophy.

The view that apoptosis precedes necrosis in the death of dystrophin-deficient muscle fibers of the mdx mouse, an animal model presenting mild muscular dystrophy, has been well substantiated [47,59]. Additionally, apoptotic myonuclei have been reported to increase in dystrophin-deficient mice 2 days after sudden spontaneous runn [43,46,182]. To investigate the role of apoptosis in human muscular dystrophy, the muscles of 11 patients of different ages with Duchenne muscular dystrophy were analyzed for apoptosis [51]. Muscle apoptosis was evaluated by terminal deoxynucleotidyl transferase test and expression of bcl-2 and bax was examined by immunohistochemistry. Very rare in normal muscles of age-matched controls (less than 0.1%), apoptotic nuclei have been detected in dystrophic muscles, particularly at the interstitial level. Furthermore, dystrophin-deficient myofibers with centrally located nuclei (regenerating myofibers?) showed a positive reaction for DNA fragmentation. A mosaic pattern of bcl-2 / bax-positive myofibers characterized the dystrophic muscles, so the relative proportion of pro- and anti-apoptotic proteins differs between muscle fibers in correlation with the presence of apoptotic myonuclei. In the interstitium, apoptotic cells were identified as macrophages and activated satellite cells. This was the first worldwide study to show an apoptotic process in the adult muscle fibers of patients with Duchenne muscular dystrophy [51]. It added an additional pathogenetic mechanism, shedding new light on muscle damage and its progression in dystrophinopathies.

Caspase 3 expression correlates with skeletal muscle apoptosis in Duchenne and facioscapulo human muscular dystrophy. A potential target for pharmacological treatment?

Apoptosis has been detected in several muscle diseases, including severe dystrophin deficiency [47], but apoptotic mechanisms are not fully described in diseases of adult skeletal muscle [59]. Studying patients with Duchenne muscular dystrophy (DMD) and facio-scapulohumeral dystrophy (FSHD) we have shown an increase of apoptotic myofibers and of bax and bcl-2-positive myofibers [62]. A positive correlation was found between apoptotic nuclei and bax expression. Caspase expression was analyzed by RNase protection. DMD muscles expressed caspase 8, 3, 5, 2, 7 and Granzyme B mRNAs. Low transcription levels of caspase 6, 3 and Granzyme B were detected in FSHD patients. Tissue levels of the caspase 3 protein were significantly correlated with apoptotic myonuclei and with bax expression. Caspase 3 activity was increased in all DMD cases, while FSHD samples were heterogeneous. Caspase transcription was not detected in normal skeletal muscle.

These data indicate that human skeletal muscle fibers during the dystrophic process modulate the expression of caspases and that caspase 3 is involved in the death and progression of myofibers, opening new perspectives in pharmacological treatments of dystrophinopathies, such as the use caspase inhibitors.

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Chapter 6
Hopes and frustrations of the Demand Dynamic Cardiomyoplasty

Permanent cardiac assistance from skeletal muscle: the Demand Dynamic Cardiomyoplasty

From 1980 I was part of an European Union-supported effort to use skeletal muscle power to provide cardiac assistance to patients suffering with a drugs' intractable cardiac failure. The hopes of cardiologists, plastic and cardiac surgeons were nurtured by the basic results of a small group of Myologists that had collected strong evidence that the skeletal muscle is plastic, not only as its size is concerned, but also for its functional and molecular characteristics. I previously discussed the evidence that those characteristics develop and are maintained under the control of the innervating motoneurons, but also of the central nervus system that dictates the daily pattern of use (see Chapter 3).

Major findings had been collected by mimicking the patterns of fast and slow motorneuron discharges, that may be simplified to the two extremes of a fast pattern delivered at high frequency (up to 100 Hz), but for short periods during the day and the slow patterns that typically were delivered 12 or 24 hours per day at low frequency (10 Hz) in rodents (rat and mouse), rabit and sheep. Fast muscle fibers are typically prone to fatigue in few minutes, while slow muscle produce much less potent tetanic contractions all the day long.

I discussed some of these basic, preliminary, information in Chapter 3. Complementary descriptions of these experimental approaches could be found also in Chapter 13.1.1. Gerta Vrbová [154]. As for the surgeons, they have played their part in the project from the beginning by designing and building, in preliminary experimental models in sheep and canines, surgical models of cardiomyoplasty, aortomyoplasty and skeletal muscle ventricles to find surgical solutions for pharmacologically intractable heart failure.

Unfortunately, the heart has to work 24 hours daily with a frequency of at least 60 contractions per minute, i.e. 76400 (60 x 60 x 24 = 76400) tetanic contractions per day. If you try to do this work with your hands, squeezing a hard rubber ball, you will suffer from fatigue in five to ten minutes. Only by squeezing a thin rubber balloon filled with air can you do this for hours, i.e. muscle fatigue strongly depends on the required muscle strength. Compression of a heart ventricle is often very challenging for a thin flat muscle, typically the latissimus dorsi (LD) wrapped around the aorta (aortomyoplasty), or the heart (cardiomyoplasty), or alone when a skeletal muscle ventricle is used in parallel to support a weak heart.

The problem of muscle fatigue, encountered very early by surgeons, hasn't stopped related research. The first compromise was to provide skeletal muscle contraction every two or more heart beats, but this did not prevented the complete transformation of a powerful but fatigue-prone LD muscle into a fatigue-resistant, but much less strong muscle. Basic myologists (My self in 1995 [194]1997 [176] and 1998 [56] in collaboration with Giorgio Arpesella]; Stanley Salmons and Jonathan Jarvis, Department of Human Anatomy and Cell Biology, University of Liverpool, UK in 1999 [195], and 2001[196]), but

also in 1999 a cardiac surgeon (James A Magovern of Allegheny General Hospital, Drexel University College of Medicine, Pittsburgh, Pennsylvania, USA [197]) found that it was possible to maintain a faster and most powerful contraction of the LD and avoiding muscle fatigue, either by drastically decreasing the number of its tetanic contractions per day (down to 30) (Salmons)[195] or by implementing an activity- rest pattern of contractions of 12 hours ON and 12 hours OFF per day (Myself in collaboration with Giorgio Arpesella of Alma Mater University of Bologna, Italy [194,176,56]; Magovern, Pittsburgh, Pennsylvania, USA [197]).
My proposal, for clinical application in patients, was to stimulate the LD only when the patients were very active (i.e., their heart rate was above 75 per minute), avoiding the stimulation of the LD when the heart rate was lower than the one set in the pourpose-modified Medtronic pacemaker. I called this approach Demand Dynamic Cardiomyoplasty when the heart surgeons of Padua asked me to implement my proposal in a group of Italian patients including those operated in Padua by Juan C. Chachques, a heart surgeon based in Paris, according to the surgical protocol validated by him and Prof. Alain Carpentier.
As I described in Chapter 3, Giorgio Arpesella, a cardiac surgeon at the University of Bologna, and I carried out a project proposed by an eminent cardiologist at the University of Pisa. The idea was to produce in Italy an animal model that would imitate the then famous surgical approach of Dynamic Cardiomyoplasty proposed by the French cardiac surgeons Alain Carpentier and Juan C. Chachques. This was tested in a simplified sheep model. The results of a year-long experiment on six sheep were more than encouraging. So, when cardiac surgeons at the University of Padua asked me to implement the Demand Dynamic Cardiomyoplasty protocol in a group of Italians suffering with farmacologic intractable cardiac failure, including new patients operated in Padua, I was confident that the approach had solid preliminary experimental foundations, as explained below . The results had been interesting in the opinions of the doctors who had followed the patients. Specifically, Dr. Gianluca Rigatelli, collecting Doppler flow wire data in the group of Italian patients, provided direct evidence of support for the cardiac systole of the LD which had led to faster, fatigue resistant tetanic contractions [50,56,60,65,66,69-74,91] .
However, despite all the evidence, the Medtronic company decided to discontinue the commercialization of the new pacemaker needed for this procedure because sales did not covered costs.
A dream to alleviate suffering and promote life-saving treatments of end-stage cardiac failure ended in great frustration due to lost profits!

Activity-rest stimulation of latissimus dorsi for cardiomyoplasty: 1-year results in sheep.

A prudent explanation of the clinical effect of dynamic cardiomyoplasty is that a minimal systolic assistance enhances the chronic elastic girdle effect of the transposed Latissimus Dorsi (LD). Slowness of the contraction-relaxation cycle and reduced power output of a fully conditioned LD limit its systolic support. Steady partial transformation of LD could increase power output by taking advantage of a faster contraction-relaxation cycle. To avoid full fast-to-slow transformation of LD, we chronically tested a daily activity-rest regimen of muscle stimulation in a simplified experimental sheep model. To mimic loss of resting tension which occurs in cardiomyoplasty, sheep LD after tenotomy of distal aponeurosis were resutured in shortened position [176], and ITREL neurostimulators

(Medtronic) connected to intramuscular electrodes were implanted according to the Medtronic Protocol. From two weeks after surgery shortened LD were burst-stimulated either 10 or 24 hr per day, the stimulators being programmed to the settings that elicited just fatiguing contractions in the shortened LD. Full-day activated LD were stimulated six months and then left unstimulated for additional six months, while the half-day activated muscles were stimulated up to one year. Two weeks after surgery and two, four, six and twelve months after stimulation, fusion frequency of tetanic contraction, power output, and fatigue resistance of LD were assessed [56]. To allow histological and molecular characterization of the two groups of stimulated muscles, LD were biopsied at six months of stimulation, and sheep sacrificed at twelve months to collect macrosopic anatomical records and perform molecular and histological analyses of proximal, intermediate and distal muscle specimens. After one year of 10 hr/day electrostimulation the gross anatomy of the LD were substantially conserved in comparison with contralateral, normal muscles (about 10% atrophy accompanied by minor fat infiltration and fibrosis). Isomyosin analysis shown that even after one year of stimulation the 10 hr/day stimulated LD contained large amounts of fast type myosin, in particular MHC2A, the isoform of fast-oxidative fibers, less prone to fatigue than the type 2B fibers of which normal LD of adult sheep is very rich. Though after six months of 24 hr/day stimulation LD were fully converted to type 1 myosin, after additional six months of resting these LD were white in appearance, atrophic (about 40%), fibrotic, and their isomyosin pattern as mixed as the LD stimulated 10 hr/day for twelve months. Accordingly, after four and six months of

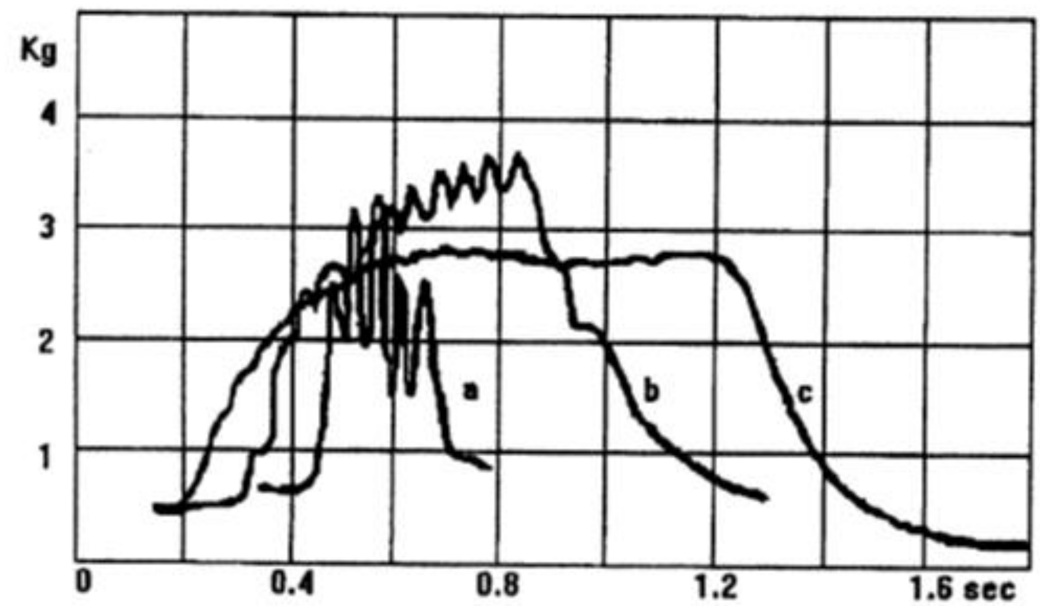

Fig 1. Contractile characteristics of conditioned LD. Tetanic fusion. The Figure shows that stimulation at 20 Hz produces a clone in normal LD (trace a, 0.25 sec), a non-fused tetanus in a half-day electrostimulated LD (trace b, 0.5 sec), and a fused tetanus in a full-day conditioned LD (trace c, 1 sec). After half-day electrostimulation sustainable power output is similar to that extractable from a 24 hrs per day electrostimulated LD (about 0.5 watts per LD, i.e., more than 2.5 watts per kg).

stimulation the frequency of tetanic fusion was higher (i.e., the contraction-relaxation cycle was faster) in 10 hr/day stimulated LD than in 24 hr/day stimulated LD; the difference disappeared at one year since the fusion frequency of the rested LD recovered to values of the one-year 10 hr/day stimulated LD (Fugure 1). Of foremost importance is the fact that from two-month up to one-year of stimulation the sustained power output per muscle of the 10 hr /day stimulated LD (that is of the daily rested muscle) is three to four times higher than that of the 24 hr/day activated LD. From two and at least up to twelve months of stimulation the sustained power of the "daily-rested" LD become higher than that of the heart at rest. In conclusion, results of our activity-rest daily regimen are encouraging: sheep LD loses very low contractile mass, and its power is equal or bigger than that of the left ventricle, since it seems to achieve a stable intermediate state of fast-to-slow transformation when stimulated for ten hours a day. After such encouraging evidence, I was brave enough to accept a request of cardiac surgeons of Padua University to test the Demand Dynamic Cardiomyoplasty in patients.

Permanent cardiac assistance from skeletal muscle: a prospect for the new millennium

This paper looks at the prospects for new surgical solutions to the problem of end-stage heart failure based on cardiac assistance from skeletal muscle [195]. The mechanical properties and myosin isoform composition changes of rabbit tibialis anterior muscles were studied after continuous stimulation at 2.5 Hz for up to 12 wk. The effects of stimulation at 2.5 Hz were less profound than those observed for the same duration of stimulation at 10 Hz. Stimulation at 10 Hz for 12 wk induced in a fast-contracting rabbit muscle complete transformation to a slow-contracting muscle homogeneous in slow myosin isoforms. Stimulation for the same period at 2.5 Hz resulted in moderate changes in contractile speed and a very small increase in the synthesis of slow myosin isoforms. On the other hand, the fatigue resistance of muscles stimulated at 2.5 Hz was as great, in both isometric and dynamic fatigue tests, as that of the muscles stimulated at 10 Hz. Thus entire fast skeletal muscles can be transformed to a state in which fast myosin isoforms continue to be synthesized, but the oxidative capacity is sufficient to support sustained working at a higher power output than that associated with slow muscle.

Intermittent stimulation enhances function of conditioned muscle

Skeletal muscle is highly adaptable in that its metabolic and contractile characteristics are largely regulated by its pattern of use. It is known that muscle can be manipulated via chronic electrical stimulation to enhance fatigue resistance. Type 2A fibers are fatigue resistant, powerful, and considered most desirable for cardiac assist purposes. We have found that 12-wk of intermittent-burst stimulation produces a high percentage of 2A fibers and increases fatigue resistance and power in rabbit latissimus dorsi muscle [197]. Fixed-load endurance tests were used to quantify fatigue resistance among normal and trained muscle groups. Control muscles were found to fatigue completely within 10-20 min. Muscles stimulated continuously for 6 wk retained 35% (71.5 +/- 19.5 g. cm) of their initial stroke work at 40 min. Muscles stimulated 12 h/day for 12 wk had the highest initial stroke work (449.7 +/- 92.4 g. cm) and the highest remaining stroke work (234.7 +/- 50.1 g. cm) at 40 min. Results suggest that employing regular resting periods during conditioning preserves strength in a fatigue-resistant muscle.

Induction of a fatigue-resistant phenotype in rabbit fast muscle by small daily amounts of stimulation

We have shown that fatigue resistance can be induced in rabbit tibialis anterior (TA) muscles without excessive power loss by continuous stimulation at low frequencies, such as 2.5 or 5 Hz, and that the same result is obtained by providing a 10 Hz stimulation in equal periods on / rest. Here we ask whether the same phenotype could be produced with daily amounts of stimulation that would be more appropriate for clinical use [195]. We stimulated rabbit TA muscles for 6 weeks, alternating fixed periods of 30 min of stimulation at 10 Hz with rest periods of different lengths. All models transformed fast glycolytic fibers into fast oxidative fibers. The muscles had fatigue-resisting properties, but maintained higher contractile velocity and energy production than fully transformed muscles of the slow oxidative type. We concluded that a single 30-min stimulation period in 24 hours could result in a substantial increase in muscle resistance to fatigue in the rabbit.

Demand dynamic cardiomyoplasty: mechanograms prove incomplete transformation of the rested latissimus dorsi.

In dynamic cardiomyoplasty, standard stimulation produces high fatigue resistance but also undesirable dynamic characteristics of the latissimus dorsi (LD), that isthere is a large loss of contractile strength and power. Based on results of intermittent stimulation in animals [56], we introduced demand stimulation, a lighter regimen of LD activity-rest stimulation [50], and the mechanogram, a noninvasive method to determine the contractile characteristics of the LD wrap. Surgery and standard stimulation was according to the technique of Carpentier and Chachques, demand stimulation and LD wrap mechanogram were as we previously described [60]. The LD contraction is synchronized to heart systole by mechanogram and echocardiography, and extent of transformation by tetanic fusion frequency analysis. A total of 22 patients were studied to date. Data for the 8 subjects who attained 6-month follow-up are reported. Four of them were lightly stimulated from the conditioning period, whereas 4 others were converted to light and then demand stimulation after years of standard stimulation. Patients were followed up with respect to survival, functional class, hospital admission rate, medication used, cardiopulmonary exercise testing, and LD wrap mechanography.

Latissimus dorsi wrap slowness reverses by the activity-rest regimen, even after years of standard stimulation (Tetanic fusion frequency of 11 +/- 2 Hz after standard stimulation vs 30 +/- 3 Hz after demand regimen, $p < 0.0001$). After demand dynamic cardiomyoplasty there are no deaths. Quality of life is substantially improved with significant reduction of heart failure symptoms (New York Heart Association class: preoperative 3.0 +/- 0.0, post-demand dynamic cardiomyoplasty 1.5 +/- 0.2, $p < 0.0001$). In the subgroup of patients lightly stimulated from LD conditioning, exercise capacity tends to increase over preoperative values more than 2 years after operation (VO2 max: preoperative 12.3 +/- 0.7 vs 16.6 +/- 1.7 post-demand dynamic cardiomyoplasty, $p = 0.05$). In conclusions, Demand stimulation and mechanography of the LD wrap are safe procedures that could offer long-term benefits of dynamic cardiomyoplasty to patients with pharmacologically intractable heart failure.

New advances in dynamic cardiomyoplasty: Doppler flow wire shows improved cardiac assistance in demand protocol

To our knowledge, there are no published data on effective cardiac assistance in dynamic cardiomyoplasty. We tested the utility of a Doppler flow wire in the beat-to-beat measurement of aortic flow velocity and in the assessment of cardiac support in cardiomyoplasty patients [60]. The technique was tested on seven patients enrolled in the Italian Demand Dynamic Cardiomyoplasty Trial. Measurements were taken using a 0.018 inch peripheral Doppler flowwire advanced through a 5 French femoral arterial sheath. Three 1-minute periods with pacer off and three 1-minute periods with clinical pacing were recorded. We measured peak aortic flow velocity over all beats. The mechanogram of Latissimus Dorsi (LD) was recorded at the same time. Comparison of preoperative and follow-up data showed significantly higher values of tetanic fusion frequency and follow-

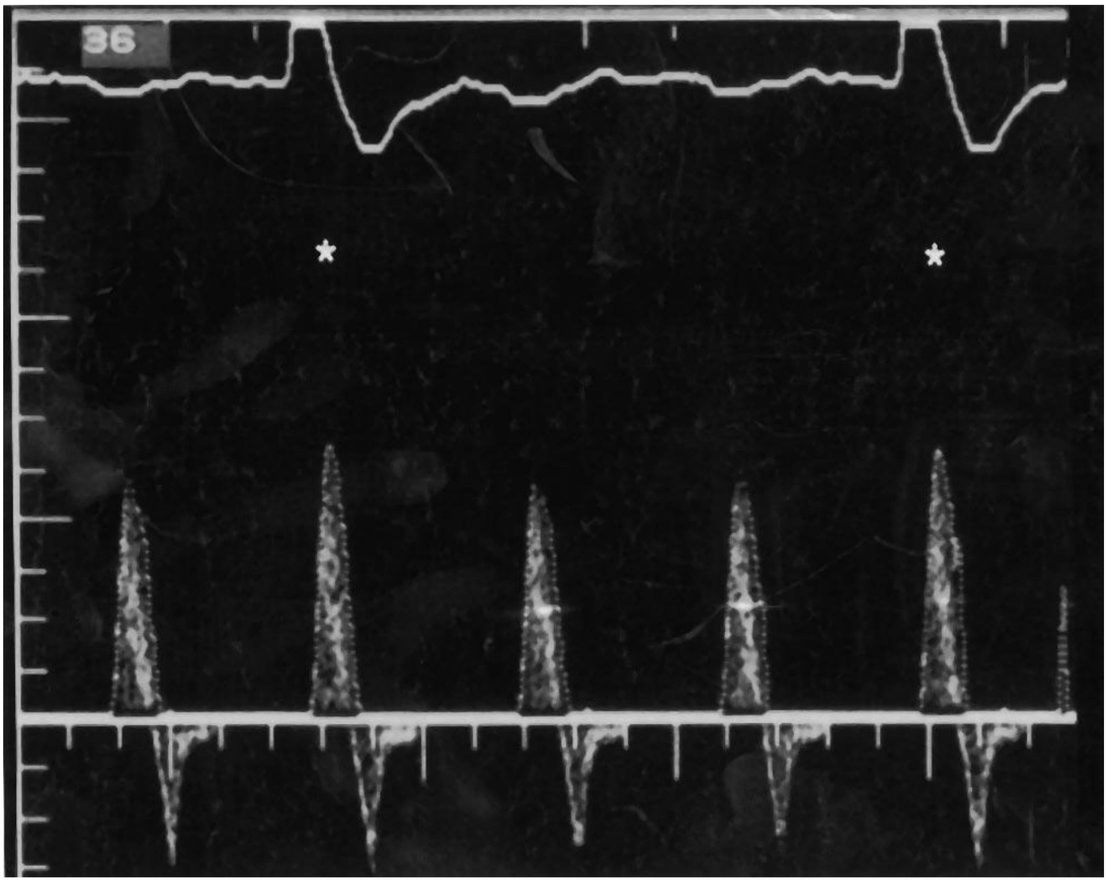

Fig 2. Aortic flow velocity spectrum during unassisted and assisted beats (asterisks) in patient number 1: the aortic flow peak increases during the assisted beat due to the increase in cardiac output during latissimus dorsi wrap contraction.

up ejection fraction, while mean NYHA class was significantly lower. Statistical analysis showed an increase in aortic flow velocity not only in the assisted versus resting period, but also in assisted versus unassisted beats (8.42+/-6.98% and 7.55+ /-3.07%). A linear correlation was found between the increase in flow velocity and the rate of tetanic fusion of the LD sheath (r2 = 0.53). In conclusion, in Demand Dynamic Cardiomyoplasty systolic assist is significant and correlates with LD contraction speed.Thus, a demand stimulation protocol maintains LD muscle properties and increases muscle performance.

Maintained benefits and improved survival of dynamic cardiomyoplasty by activity-rest stimulation: 5-year results of the Italian trial on "demand" dynamic cardiomyoplasty

Latissimus dorsi (LD) degeneration related to continuous electrical stimulation has been the main cause of poor results of dynamic cardiomyoplasty (DCMP) and of its exclusion from the recent international guidelines on heart failure. To avoid full transformation of the LD improving results, a new electro stimulation protocol was developed; fewer impulses per day were delivered, providing the LD wrap with daily periods of rest (stimulation on demand), based on a heart rate cut-off. We here report results at 5 years of follow-up of the Italian Trial of Demand Dynamic Cardiomyoplasty, discussing their impact on the destiny of this type of cardiac assistance. Twelve patients with dilated myocardiopathy were submitted during 1993-1996 to DCMP and at different intervals to demand protocol. Clinical, echocardiographic, mechanographic and cardiac invasive assessments were scheduled before initiating the demand protocol and during the follow-up at 0, 6 and every 12 months. The mean duration of follow-up was 40.2+/-13.8 months (range 18-64). There were no perioperative deaths. The demand stimulation protocol showed a decrease in 5 years in New York Health Association (NYHA) class (3.17+/-0.38-1.67+/-0.77, P=0.0001), an improvement of left ventricular ejection fraction (22.6+/-4.38-32.0+/-7.0, P<0.001), a 5-year actuarial survival of 83.3%. In conclusions, Demand DCMP maintains over time LD muscle properties, enhances clinical benefits and improves survival, thus reopening the debate whether this type of treatment should be considered in patients with end-stage heart failure. [69]

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Chapter 7
Helmut Kern and his request for collaborations
The EU Program RISE

I met Helmut Kern at one of the Vienna Workshops on Functional Electrical Stimulation (FES), perhaps in 1998, and then in 1999 when he came to see me at the Department of Biomedical Sciences of the University of Padua during one of his stays at the Hotel Continental in Montegrotto Terme (Padua), Italy. He was in fact a fanatic of the warm pools and the ups and downs of the Euganean Hills where he pedaled under clear sky avoiding the bad weather of Vienna, Austria. Helmut had brought with him his rehabilitation thesis, which described experiences of a very young Rehabilitation Specialist who had treated the first cases of implantation of Vienna FES stimulators in paraplegic patients.

He asked for my opinions and offered me the possibility of collaborating to collect more solid evidence of muscle improvements even in the worst cases, that is those of patients with permanent denervation of the lower limbs due to serious lesions of the conus and cauda equina, both for original trauma and/or for ischemic / hemorrhagic / infectious complications.

My immediate response was: Dear Helmut, could you take biopsy samples from the

EU-RISE Plenary Meeting

Padova/Montegrotto, Italy -March 5th-8th, 2003

Thursday, March 6, 2003 (Montegrotto)

09.00 – 09.30	**Carraro, Rossini:** ***Myogenesis, rat experiments***
09.30 – 10.30	**Mayr, Rafolt, Sauermann*: Rabbit experiments Vienna: study, electro- and muscle physiological measurements.***
10.30 – 11.30	**Gruber, Bittner, Rossmanith:** ***Rabbit experiments Vienna: histological, biochemical and metabolic investigations***
11.30 – 12.30	**Salmons, Jarvis, Ashley:** ***Rabbit experiments Vienna: histological, biochemical and metabolic investigations***
14.00- 15.00	**Kern et al,:** ***Patient study: current status*** **Hofer:** ***Current stimulation technology***
15.00 –16.30	**Carraro et al., Gruber et al.: P*atient study: histological, biochemical and metabolic investigations***
16.30 – 17.30	**Hufgard, Serrat, Maier, Rupp, (Kaps, Exner), Cerrel-Bazo, Lotta:** ***Project status in clinical partner sites***
17.30 – 18.30	**General discussion of current project status**

Friday, March 7, 2003 (Padova)

10.00 – 11.00	**Carraro: Guided tour of the Dept. of Experimental Biomedical Sciences**
11.00 – 12.00	**Salmons, Jarvis, Ashley:** ***Rabbit experiments Liverpool: plans and strategies***
12.00 – 12.30	**Discussion: questions to be answered by the pig experiments**
14.00 - 14.30	**Dimitrijevic:** ***Patient assessment***
14.30 – 15.00	**Kern:** ***Clinical study - plans and strategies***
15.00 – 16.00	**Mayr, Hofer, Rafolt, Gallasch:** ***Stimulation and measurment equipment, concepts and development work.***
16.00 – 17.00	**General discussion, forthcoming project activities**

muscles of rehabilitated legs? Because he was very optimistic, a long-standing partnership began almost immediately, [199-201,79-81,86,90,93,97,101,111,123,144] and continues to date [164, 165].
A series of papers were published in BAM 16. 1, 2022 with the preliminar results for the application to an European Cooperative Project: RISE (Use of electrical stimulation to restore standing in paraplegics with long-term denervated degenerated muscles).
A dedicated Meeting was held in Padua in 2003 (see previous Figure).
Then the project was approved as the EU Commission Shared Cost Project RISE (Contract no. QLG5-CT-2001-02191).
The first article listed in PubMed was published in 2004.
The following subchapters of this book detail what happened during the next 20 years of collaborations and pubblications in decent scientific journals, starting from the very successful Eu Program RISE.
RISE is not an acronym, but the aim of the project: i. e., to allow paraplegic patients stand up from their wheel chairs, a goal that was luckely scored!

A second dream that had become reality

Home-Based Functional Electrical Stimulation for Long-Term Denervated Human Muscle. The Vienna Rehabilitation Strategy's History, Foundations, Eu Project RISE Results, and Future Perspectives

Here, we'll go over the issues with home-based functional electrical stimulation of denervated degenerating muscles (hbFES for DDM), which is a therapeutic option for people whose leg muscles have been permanently denervated [111]: i) Muscle atrophy/hypertrophy versus processes of degeneration/regeneration, and recovery of muscle twitch and tetanic contractility by hbFES; ii) clinical effects of hbFES using the protocol of the "Vienna School"; iii) damage to the lower spinal cord's upper (UMN) and lower (LMN) motor neuron neurons; iv) Limitations and viewpoints.
The main justifications for implementing the Vienna hbFES protocol are: i) Histological and electron microscopic evidence that two years of hbFES return muscle fibres to a state typical of two weeks denervated muscles with respect to atrophy, disrupted myofibrillar structure, and disorganised Excitation-Contraction-Coupling system; ii) Increased muscle size in both legs; iii) Improved tetanic force production after 3-5 months of skin surface electrical stimulation using long stimulus pulses (> 150 msec) of high amplitude (> 80 mAmp).
It is vital to encourage these individuals to engage in lifelong chronic stimulation, preferably standing against their own weight rather than sitting, but only younger, light weight patients can be expected to be able to stand up and perform those exercises on their electrostimulated limbs. For decades, the patients must continue their hbFES training. The use of big surface electrodes and the length of time patients are ready to devote to such muscle training are two factors that clearly allow the treatment.

Dr. Kern is currently expanding the advantages of hbFES to participants who experience the effects of muscular weakening for a variety of reasons, from the gradual but persistent ageing process to the devastatingly quick advancement of muscle atrophy in cancer patients. Furthermore, the Vienna principles were applied to the seemingly simplest cases

of partial peripheral denervation of the arms and legs by a multidisciplinary research team at the Interdepartmental Research Center of Myology of the University of Padua, Italy [107,108].
Despite the successes of heart pacing and mini-implants for the deaf, two very successful clinical trials, and despite the effectiveness of lifelong high-level physical activity in delaying the effects of ageing and of physical approaches in peripheral and central neural repair, functional electrical stimulation in paraplegics by implanted electrodes and neuromodulators has been almost abandoned by commercial producers, only surface stimulators are easily found also in Internet.
On the other hand, hbFES for denervated muscles deserves to be re-evaluated according to precise scientific guidelines, but keeping in mind its low costs and the legal rights of patients to have their burdens lightened. Luckily, after so many years in which the only published articles were ours, articles by independent clinical researchers are starting to appear in journals listed in PubMed [202,203].

Home-based Functional Electrical Stimulation for long-term denervated human muscle: History, basics, results and perspectives of the Vienna Rehabilitation Strategy

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Abstract

We will here discuss the following points related to Home-based Functional Electrical Stimulation (hbFES) as treatment for patients with permanently denervated muscles in their legs: 1. Upper (UMN) and lower motor neuron (LMN) damage to the lower spinal cord; 2. Muscle atrophy/hypertrophy versus processes of degeneration, regeneration, and recovery; 3. Recovery of twitch- and tetanic-contractility by hbFES; 4. Clinical effects of hbFES using the protocol of the “Vienna School”; 5. Limitations and perspectives. Arguments in favor of using the Vienna protocol include: 1. Increased muscle size in both legs; 2. Improved tetanic force production after 3-5 months of percutaneous stimulation using long stimulus pulses (> 100 msec) of high amplitude (> 80 mAmp), tolerated only in patients with no pain sensibility; 3) Histological and electron microscopic evidence that two years of hbFES return muscle fibers to a state typical of two weeks denervated muscles with respect to atrophy, disrupted myofibrillar structure, and disorganized Excitation-Contraction Coupling (E-CC) structures; 4. The excitability never recovers to that typical of normal or reinnervated muscles where pulses less than 1 msec in duration and 25 mAmp in intensity excite axons and thereby muscle fibres. It is important to motivate these patients for chronic stimulation throughout life, preferably standing up against the load of the body weight rather than sitting. Only younger and low weight patients can expect to be able to stand-up and do some steps more or less independently. Some patients like to maintain the hbFES training for decades. Limitations of the procedure are obvious, in part related to the use of multiple, large

surface electrodes and the amount of time patients are willing to use for such muscle training.

Key Words: SCI, FES, skeletal muscle permanent long-term denervation, recovery of function, 2D and 3D Color TAC, biopsy, histology, electron microscopy

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The series of e-published issues of the European Journal of Translational Myology (EJTM) started in 2014 with the Specials on "The long-term denervated muscle" to resound "The denervated muscle", a book edited in 1962 by one of our virtual mentors, Ernest Gutmann, a true pioneer of nerve-muscle inter relations [1].

To explain why you are now reading this chapter, we need a tremendous amount of details that we cannot include in this review. Indeed, looking to his roots, one of us rediscovered a few months ago (emptying the office for retirement) that his M.D. Thesis was on muscle denervation [2].

Ugo Carraro: Pioneering studies

Prof. Carraro would like to remember that in the early 1960s he was a young student at the School of Medicine of the University of Padova, just admitted in 1964 to Internship of the Institute of General Pathology directed by Prof. Massimiliano Aloisi. When in Padua our full Professor almost every day took a tea cup with the fellows, discussing muscle research and his hope to develop in vitro muscle mimics, despite the difficulties to obtain motoneuron-myotubes cultures. Thus, he started lab training doing histology and discussing of the muscle and of its dependence from the motor neuron. How to study this topic, if not by denervation experiments? Four years later he defended his M.D. Thesis on: "Impairments of the functions of substructural membranes of the denervated muscle (Alterazioni delle funzioni delle membrane substrutturali nel muscolo denervato) [2]. Now, after 45 years he is trying to convince experts in aging that sparse, but incremental denervation is one of the many mechanisms that worsen muscle performances and quality of life of seniors, and that a long-term high-level physical activity may defer the unavoidable decay of aging [3,4]. Mosole et al. [3], indeed, comparing muscle biopsies from sedentary and very physically active seniors observed reduced numbers of denervated fibers and higher percentages of throphic and slow-type groups of reinnervated fibers in the active group. The observations suggest that long-term physical activity promotes reinnervation of muscle fibers undergoing age-related denervation [3].

Here he may only list the topics he would like to describe in a future book. He will need to start with his mentors (Aloisi, Zatti and Margreth), and describe the importance for his ability to design and perform independent research of his younger or older colleagues Catani, Mussini, Cantini, Salviati and Schiaffino. The explanation of why in Padua there was and there is such a strong tradition of Myology will end the first chapter. It will be a funny story related to fever and burning of toxins in the muscle.

He will explain why he moved from General Pathology to Muscle Biology and Physiopathology, from Basic to Applied Myology, organizing the Interdepartmental Research Center of Myology of the University of Padova, in which clinical colleages and biomedical scientists are almost equally present, from organizing the PaduaMuscleDays Meetings and editing the journal Basic and Applied Myology (BAM) to the European

Journal of Translational Myology (EJTM).
He will mention the inter-relationships among his students (Donatella Biral, Donatella Morale, Giorgio Vescovo, Corrado Rizzi, Gianluca Rigatelli, Marco Sandri, Marzena Podhorska-Okolov, Katia Rossini, Massimo Donà, Nicoletta Adami, Sandra Zampieri and Simone Mosole), the visits and lab periods spent in international laboratories (in particular those of John Gergely and Alfred Goldberg in Boston), the Italian and International friends with which he has published papers (Anna Jakubiec-Puka, Claudio Franceschi, Giorgio Arpesella, Mike V. Dodson, Stanley Salmons, Winfried Mayr, Simona Boncompagni, Feliciano Protasi, Antonio Musarò, Giorgio Fanò, Vincenzo Vindigni, Franco Bassetto, Francesco Mazzoleni, Dan Graupe, Amber Pond, Marina Marini, Fabio Francini, Paolo Gargiulo, Thordur Helgason, Tiziana Pietrangelo, Nejc Sarabon, and last but not least Helmut Kern) and of course the many others he met during International Conferences.
His "first" Meeting, as a young fellow of myology was organized in Switzerland by Marcus C. Schaub, but how to forget the International Conferences where he and his students had the chance to know Bruce M. Carlson, John Faulkner, Zipora Yablonka-Reuveni, Eric Monnet, Miranda Grounds, Winfried Mayr and many other Vienna friends or the Conference he organized in Thermae of Euganea Hills, Padua: where he met Juan Carlos Chachques, a young Surgeon from Argentina working in Paris with Alain Carpentier, Carlo Reggiani, now full professor of Physiology in Padua University, Terje Lomo, Dirk Pette, Salvatore Di Mauro, Clara Franzini-Armstrong, Tessa Gordon, Victor Dubowitz, Terry Partridge, Ryoichi Matsuda, Stanley Salmons, Jonathan C. Jarvis, Dario Coletti, Werner Lindenthaler and many others. Of Gerta Vrbova he will remember that she was one of the first invited speakers he personally met in 1979 in the Margreth's Lab, as a young fellow who presented to her his first independent publication on "selective maintenance of neurotrophically regulated proteins in long-term denervated hemidiaphragm"[5]. Finally, he will identify the main research topics he worked on during 45 years of research activity. He started in 1966 to prepare the M.D. Thesis, but he is not yet ready to "retire" from Myology: as a Senior Scholar of the University of Padova, he think that he has a lot to translate to clinical colleagues.
The first topic of his long career was: Contractile protein isoforms identified by several electrophoretic methods [6,7] and their transitions as tools to study modulation and pathology of muscle fiber units and motoneurons. He will remember his first publication on "Neural control on the activity of the calcium transport system in sarcoplasmic reticulum of rat skeletal muscle" by Margreth, Salviati, Carraro in Nature 1973 [8] and six years later "denervation-induced isomyosin transitions" by Carraro, Catani, Biral. Exp Neurol 1979 [5] and by Carraro et al. 1985 [9]. Some years afterwards, inspired by Terje Lomo and Stefano Schiaffino [10-19], he independently collected corroborating results on a rat model of continuous electrical stimulation of denervated muscle, achieving a yet unexplained and infrequently cited high increase of slow muscle fibers properties in the rat denervated fast muscle [20]. A system analysis with flow charts may summarizes all the interactions among old and recent topics, Carraro's mentors - students - Padua colleagues with Italian and International Scientists/Clinicians involved in animal and human muscle biology, pathology, therapy and rehabilitation, but he belive he will need months if not years to complete his book project.
He has to describe: Muscle damage and regeneration via myoblast's proliferation, differentiation and fusion [21,22] including exercise-induced muscle fiber apoptosis in normal and dystrophic animal and human muscles [23,24]. He studied: Isomyosins in

hypertension and heart failure [25] and introduced the concept of "Demand Dynamic Cardio-myoplasty," first in a sheep model with Giorgio Arpesella [26] and then in patients with Gianluca Rigatelli [27]. Corroborating evidence of effectiveness of the intermittent stimulation strategy was collected on other sheep models [28,29].

The main preliminary observations that support the Vienna Strategy for recovery of permanent denervated human muscles stand on an experimental study of rat muscles, showing that "A Subpopulation of Rat Muscle Fibers Maintains an Assessable Excitation-Contraction Coupling Mechanism After Long-Standing Denervation Despite Lost Contractility" by Squecco R, Carraro U, Kern H, Pond A, Adami N, Biral D, Vindigni V, Boncompagni S, Pietrangelo T, Bosco G, Fanò G, Marini M, Abruzzo PM, Germinario E, Danieli-Betto D, Protasi F, Francini F, Zampieri S. A subpopulation of rat muscle fibers maintains an assessable excitation-contraction coupling mechanism after long-standing denervation despite lost contractility. J Neuropathol Exp Neurol. 2009 Dec;68(12):1256-68. doi: 10.1097/NEN.0b013e3181c18416. PMID: 19915489 [93], whose abstract follow below.

To define the time course and potential effects of electrical stimulation on permanently denervated muscle, we evaluated excitation-contraction coupling (ECC) of rat leg muscles during progression to long-term denervation by ultrastructural analysis, specific binding to dihydropyridine receptors, ryanodine receptor 1 (RYR-1), Ca2+ channels and extrusion Ca2+ pumps, gene transcription and translation of Ca2+-handling proteins, and in vitro mechanical properties and electrophysiological analyses of sarcolemmal passive properties and L-type Ca2+ current (ICa) parameters. We found that in response to long-term denervation: 1) isolated muscle that is unable to twitch in vitro by electrical stimulation has very small myofibers but may show a slow caffeine contracture; 2) only roughly half of the muscle fibers with Bvoltage-dependent Ca2+ channel activity are able to contract; 3) the ECC mechanisms are still present and, in part, functional; 4) ECC-related gene expression is upregulated; and 5) at any time point, there are muscle fibers that are more resistant than others to denervation atrophy and disorganization of the ECC apparatus. These results support the hypothesis that prolonged "resting" of [Ca2+] may drive progression of muscle atrophy to degeneration and that electrical stimulation-induced [Ca2+] modulation may mimic the lost nerve influence, playing a key role in modifying the gene expression of denervated muscle. Hence, these data provide a potential molecular explanation for the muscle recovery that occurs in response to rehabilitation strategies developed based on empirical clinical observations [93].

Finally, the strong leadership of Helmut Kern convinced Engineers in Vienna and then myologists in Italy (Carraro's team in Padua, Antinio Musarò of Sapienza University of Rome and Feliciano Protasi with Simona Boncompagni in Chieti) to implement two pilot trials supported by the EU Project RISE, the first a cross-sectional study [30-36] and then a longitudinal-study [37-40] demonstrating that a home-based strategy of Functional Electrical Stimulation (hbFES) recovers muscle mass and functions of permanently denervated human muscle even after years of permanent denervation.

With EU-support, Helmut Kern and his European collaborators, Carraro's team included, are now translating this strategy to the more frequent cases of muscle deterioration due to aging and cancer. [3,41-43] Further, in Padua he is extending the EU RISE results to partially reinnervating muscle, developing dedicated monitoring strategies. [39,44,45]. To objectivize results of these researches, he is proud to have revitalized the clinical use of ultrasound muscle approaches, adding dynamic analyses of contractile properties in clinical evaluation of denervated and reinnervating muscles. Further, he suggested to Paolo Gargiulo and Helmut Kern to add false color to "Monitoring of muscle and bone recovery in spinal cord injury using three-dimensional imaging and segmentation techniques", to allow doctors and their patients to read much easy-to-interpret Computer Tomography analyses of their deteriorating or recovering muscles [38,39,46] That is why after almost 50 years, he is still fond of the effects of denervation and of the modulation by electrical stimulation of skeletal muscle fibers, of their adaptation/damage/apoptosis/regeneration potentials by reciprocal interactions with inflammatory cells and nerve, hoping to identify further clues worth to be translated into clinically relevant therapy and rehabilitation strategies.

Helmut Kern: Pioneering research

In 1990 dr. Helmut Kern achieved his Habilitation for M.D. Rehabilitation with a thesis that has been published in German in the Oesterreichische Zeitschrift fuer Physikalische Medizin 1995; 5: Heft 1, Supplementum [47]. The thesis is now reprinted in the special issues "The long-term denervated muscle". The English abstract is provided in the following paragraph.

Functional Electrical Stimulation on Paraplegic Patients.

We report on clinical and physiological effects of 8 months Functional Electrical Stimulation (FES) of quadriceps femoris muscle on 16 paraplegic patients. Each patient had muscle biopsies, CT-muscle diameter measurements, knee extension strength testing carried out before and after 8 months FES training. Skin perfusion was documented through infrared telethermography and xenon clearance, muscle perfusion was recorded through thallium scintigraphy. After 8 months FES training baseline skin perfusion showed 86 % increase, muscle perfusion was augmented by 87 %. Muscle fiber diameters showed an average increase of 59 % after 8 months FES training. Muscles in patients with spastic paresis as well as in patients with denervation showed an increase in aerob and anaerob muscle enzymes up to the normal range. Even without axonal neurotropic substances FES was able to demonstrate fiber hypertrophy, enzyme adaptation and intracellular structural benefits in denervated muscles. The increment in muscle area as visible on CT-scans of quadriceps femoris was 30 % in spastic paraplegia and 10 % in denervated patients respectively. FES induced changes were less in areas not directly underneath the surface electrodes. We strongly recommend the use of Kern`s current for FES in denervated muscles to induce tetanic muscle contractions as we formed a very critical opinion of conventional exponential current. In patients with conus-cauda-lesions FES must be integrated into modern rehabilitation to prevent extreme muscle degeneration and decubitus ulcers. Using FES we are able to improve metabolism and induce positive trophic changes in our patients' lower extremities. In spastic paraplegics the functions „rising and walking" achieved through FES are much better training than FES ergometers. Larger muscle masses are activated and an increased heart rate is measured, therefore the impact on cardiovascular fitness and metabolism is much greater. This effectively addresses and prevents all problems, which result from inactivity in paraplegic patients.

The 325 references added at the end of the Thesis are a remarkable collection of the pioneering work on FES in paraplegics that ended up with the first world implant of a device performed in Vienna in 1983 [49]. Since then, an enormous amount of new work has been necessary to establish a clinically accepted strategy for recovery of contractile function of long-term denervated muscle, but the work in the 1970's and 1980's has provided a firm and accurate basis for the current understanding of the recovery process in human muscles.

Collaboration of Austrian and Italian researchers

In 1998 Helmut Kern went once again in Terme Euganee to cycle on the Euganei Hills. A late morning he went to the Padua Institute of General Pathology with his Habilitation Thesis to meet Ugo Carraro and to express is strong willingness to collaborate in a scientific study of a series of *Conus Cauda* sufferers he was training with hbFES since several years. The reply of Ugo was outspoken: "Helmut harvest a muscle biopsy and we will show to skeptics that the astonishing functional improvements in muscle contractility you achieved with your elegant training strategy will be supported by evidence of improved muscle fiber size and ultrastructural features". The first biopsy from muscles treated in this way is described in two articles that report the characteristics of the muscle fibers from the Quadriceps of a person after 26 months of denervation and hbFES Training [30,32].

From the second article, submitted years before acceptance (and only after a cross-sectional study published in a prestigious journal provided stronger evidence of the effectiveness of the hbFES for denervated muscles [32]) we here republish the clinical description of this first, very successful case.

"V. Z., a 47-year-old man, had suffered a traumatic cauda equina *lesion at T12. One year later, his quadriceps femoris muscles were severely wasted on both sides. Voluntary movement, sensation, and reflexes were all absent, consistent with total denervation. After a further 6 months, findings at neurological examination were unchanged. Absence of volitional activity on needle electromyography (EMG) and of evoked activity using surface EMG with transcranial and lumbosacral magnetic stimulation confirmed permanent and complete loss of motor functions of spinal nerves L1 to L4. Direct electrical stimulation, which in a normal muscle would elicit a response with a chronaxie of 0.1–0.7 ms, required a chronaxie of more than 20 ms, constituting further evidence of complete loss of innervation. A computerized tomography (CT) scan of the thighs revealed marked atrophy of muscle tissue with replacement by fat; the cross-sectional areas of the quadriceps muscles were 36.0 cm2 (right) and 36.1 cm2, representing 58.9% (right) and 59.1% (left) of the corresponding areas in a typical healthy individual. No detectable knee extension torque could be elicited by stimulation under isometric conditions with the subject sitting with the knee flexed at 90°. Eighteen months after his injury, V. Z. commenced a training program which, after appropriate instruction, he was able to carry out at home. Two pairs of large electrodes, each having an area of 200 cm^2, were strapped to the anterior surface of the thighs in proximal and distal positions. Twitch contractions were elicited by biphasic rectangular current pulses having duration of 120 ms and amplitude of 200 mA, delivering impulse energy of 1.92 Joules, to recruit fibers throughout the quadriceps femoris muscles. The long duration of the impulses needed for stimulation precluded the use of frequencies that would elicit tetanic contractions; training was therefore initiated with single twitches at 2 Hz and delivered for 15 min per day, 5 days per week. After 4 months, excitability of the muscle fibers had recovered sufficiently for pulses of shorter duration to be used. At this stage, the protocol was augmented with an additional tetanic pattern consisting of pulses of 40 ms delivered at 20 Hz for 2s on, 2 s off for 15 min daily, 5 days per week. The total amount of stimulation was then 30 min daily for each muscle. The additional tetanic stimulation pattern produced more rapid and more forceful contractions, resulting in a progressive increase in knee extension torque. After 26 months of stimulation V. Z.'s thighs came to resemble*

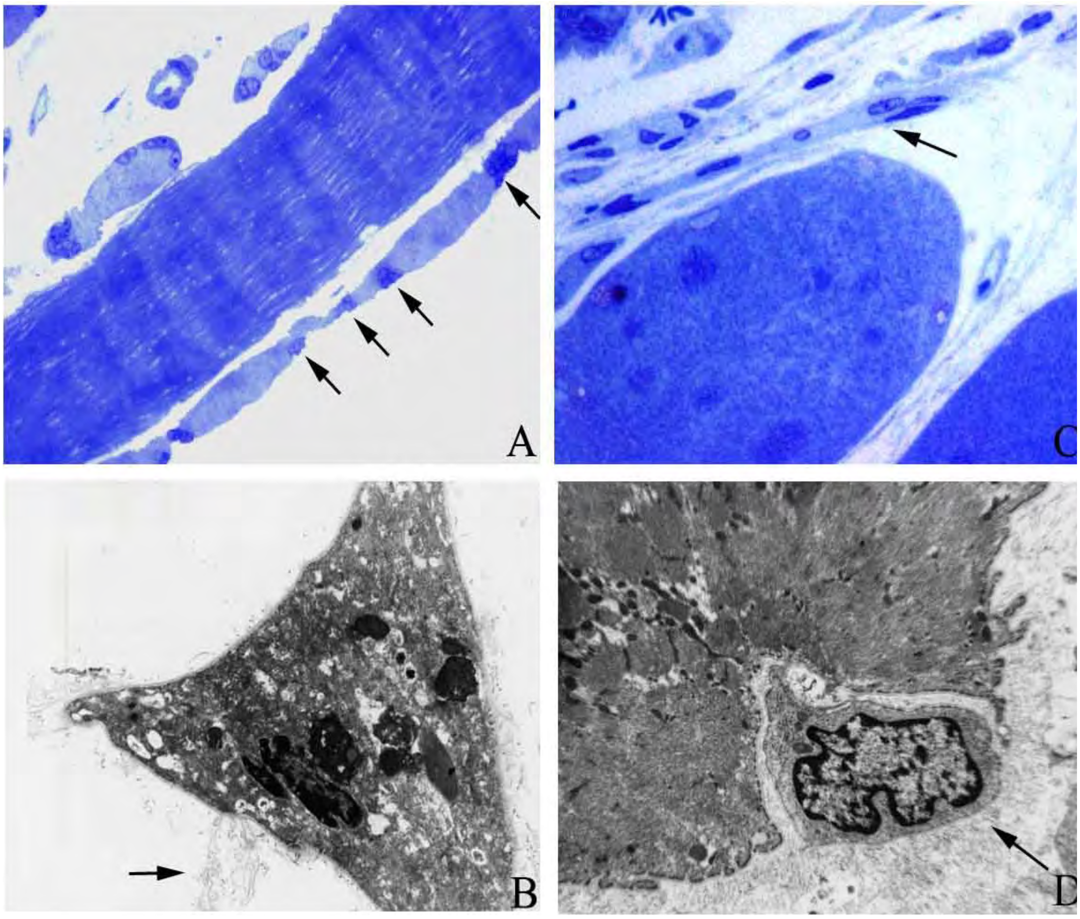

Fig 1. Semi-thin longitudinal and transverse electron microscopy sections from 4-year human denervated skeletal muscle biopsy after 2-year FES. A, Semi-thin section of eutrophic or severely atrophic myofibers. Arrows indicate clusters of myonuclei in severely atrophic myofibers. B, Electron microscopic cross section of a small myofiber: the angular appearance and redundant layers of the basal lamina are hallmarks of severe atrophy. C, Semithin section of a eutrophic myofiber and myotube (arrow). D, Electron microscopy of a myotube. Serrated sarcoplasm caracterize an aneural regenerated myofiber undergoing "denervation" atrophy.

those of a healthy sedentary subject; although the external appearance was not entirely normal, it was certainly more acceptable cosmetically to the patient. CT scan showed that the cross-sectional areas of the quadriceps muscles at the same level had increased on the right side from 36.0 to 57.9 cm2 and on the left side from 36.1 to 52.4 cm^2; these figures represent 94.7% (right) and 85.7% (left) of the areas typical of a healthy subject. Muscle density, expressed in Hounsfield Units, had risen from 11.0 to 26.4 on the right side and from 10.7 to 24.1 on the left. Stimulation of the quadriceps muscles elicited a knee extension torque of 12.0 Nm on the right and 10.5 Nm on the left. Despite the marked restoration of muscle cross-sectional area, this was less than 10% that of a normal subject. Nevertheless, this stimulation-induced torque enabled V. Z. to extend the knee from a sitting position and to maintain a standing posture without the support of the upper extremities.

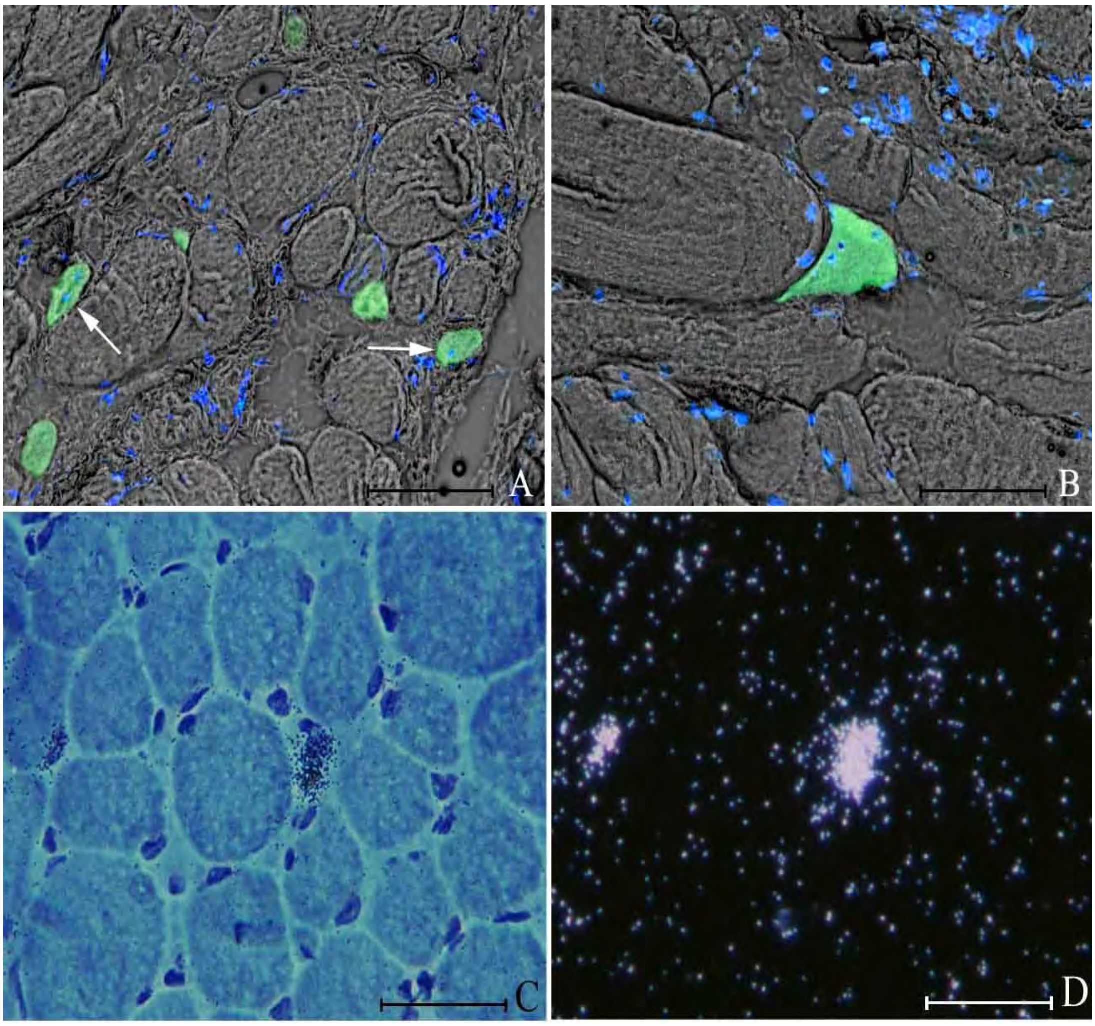

Fig 2. Markers of myogenic events in long-term denervated human muscle. A and B, anti-MHCemb positive myofibers in skeletal muscle biopsy of 4-year human flaccid paralysis after 2-year FES. Arrows point to small centrally nucleated myofibers. C and D, Myogenin positive cells (myoblasts) [from 51].

Biopsies were taken from the right and left vastus lateralis muscles and frozen sections were stained with hematoxylin and eosin and with a monoclonal antibody (NCL-MHCd; Novocastra Laboratories Ltd, Newcastle upon Tyne, United Kingdom) to the embryonic myosin heavy chain isoform (MHCemb).The sections consisted mainly of large round myofibers with a mean diameter of 37.2 +/-24.8 μm (right) and 40.5 +/- 24.9 μm (left). There was very little fat or fibrous connective tissue. Small myofibers (<10 μm diameter) were also present. Some appeared to be severely atrophic (Figure 1); others, intensely basophilic with several large internal nuclei, we interpret as undergoing regeneration. The latter stained positively with anti-MHCemb, providing evidence of their recent formation (Figure 2). The antibody also reacted with some larger myofibers (> 30 μm diameter) with subsarcolemmal myonuclei; we have seen similar fibers in permanently denervated rat muscles in which regeneration had been induced by myotoxin treatment. MHCemb-positive myofibers constituted 8.7% (right) and 2.3% (left) of the identifiable muscle fibers present in the biopsies.

Paralysis and denervation were demonstrated clinically in this patient at 12 months and 18 months post-injury and again after 26 months of stimulation. We conclude that the injury was stable and that no recovery could have occurred spontaneously during the period of treatment. Nonetheless, the intensive regime of electrical stimulation was associated with an increase in excitability, size, and force-generating capacity of the denervated quadriceps muscles, and this was sufficient to allow knee extension to be induced by electrical stimulation. There was histologic evidence of an accompanying reduction in fat and connective tissue, of growth in diameter of surviving myofibers, and also of regenerative phenomena resulting in the formation of new myofibers.
Previous studies on denervated muscles in both animals and humans have shown that electrical stimulation can induce a small increase in muscle mass and recovery from atrophy of the denervated muscle fibers. The present case is unusual in the extent of the changes produced in the long-term denervated and stimulated muscle rationale to plan research aimed to recover long-lasting denervated muscle.
Permanent denervation of leg muscles due to LMN injury can occur after trauma to the spinal cord, roots, and peripheral nerves. When proximal denervation occurs, reinnervation can take more than a year, during which time severe atrophy and fibrosis of the affected muscle tissue can impair synaptic reorganization.Early effect of SCI is rapid loss of contractile force and mass of the affected muscles. Atrophy of leg muscles is particularly severe when the injury destroys the LMN and, hence, the contacts between motor neurons and muscle fibers. In such cases, within a few weeks the atrophied and fibrillating muscles become unable to sustain tension during tetanic contractions induced by electrical stimulation. Within a few months the denervated leg muscles are no longer excitable by normal commercial electrostimulators because they have undergone a serious disorganization of the contractile elements (myofibrils) and of the excitation-contraction coupling apparatuses [31]. Finally, after years of denervation, muscle fibers are replaced with adipose and fibrous tissues [30,31,37,38,58].
Those severe functional and structural changes of denervated muscle tissue are not detectable in patients with complete upper motor neuron (UMN) lesions even 20 years after thoracic SCI [59] On the other hand, larger trauma of the lumbar and ischiatic regions, complicated by ischemic and infection necrosis of the spinal cord, may extend the damage to large segments of the medulla and of the nerve roots. In these latter cases, the diagnostic problems are related to completeness of the LMN denervation, while the absence of sensation of the legs and of the pelvic sphincters grants completeness of the transverse spinal cord lesion (ASIA grade A of SCI).
To avoid problems in interpreting clinical findings related to residual innervation or reinnervation, we first designed and implemented a cross-sectional study [31] followed by a 2-year prospective longitudinal study [37,38] that recruited 25 paraplegic patients specifically selected for complete LMN denervation of the quadriceps muscle. In the longitudinal study, the same group of patients was evaluated before and after two years of hbFES using clinical, functional, imaging, and muscle biopsy analyses [37,38,60] Protocols have been designed and implemented to test for "completeness" of LMN denervation of right and left quadriceps muscles before and during the two years of the study [38,61]. By such tests, in particular the electrical stimulation test by bidirectional rectangular pulses of 1 ms, 40 Hz, 100 mA amplitude for thigh muscle contraction, complete and permanent denervation of the quadriceps before hbFES and after two years of training was fully granted. Indeed, the stimulated muscle improved its excitability by recovering tetanic contractility, but never responded to settings that are

capable of eliciting contraction of the innervated muscle tissue [38]. If no electrical stimulators are available that provide the high-level stimulation parameters, electrical stimulation with bidirectional rectangular pulses of approximately 1 ms, a frequency of 40Hz and an intensity of 100mA can be used for the first evaluation of the paralyzed muscle. These parameters can be provided by most commercially available devices and are sufficient for a first diagnosis, if the stimulated muscle shows signs of denervation. Overall, the behaviors described above leave a significant time window for intervention to avoid denervated LMN muscle degeneration using home electrical stimulation.

From the first biopsy to the end of the European Project RISE: Use of electrical stimulation to restore standing in paraplegics with long-term denervated degenerated muscles (Contract no. QLG5-CT-2001-02191)

From early 2000 to August 2004 more than 130 biopsies of Conus Cauda Patients were analyzed in Padua (by morphometry and immunostaining) and in Chieti (electron microscopy). Further, muscle biopsies from spastic paraplegics (i.e., those with lesion of the upper motoneuron) were also analyzed to described the differential behaviors of truly disconnected muscle fibers to those severely atrophic (but never degenerated) due to severe unloading [59].

Aim of the EU Commission Shared Cost Project RISE (Contract no. QLG5-CT-2001-02191) was to confirm previous results of the cross-sectional study [31] by a longitudinal prospective study in 25 paraplegic patients specifically selected because of complete LMN denervation of the quadriceps muscles. The overall conclusions, taken from Kern et al. 2010 NNR article [38] of all these studies may be summarized as follows:

"Atrophy of skeletal muscle groups is particularly severe when SCI involves all the lower motor neurons (LMNs). After such a complete injury, the peripheral endings of motor neurons quickly degenerate whereas LMN denervated muscles undergo progressive decay, which can be roughly divided in the following chronological steps: (a) in days denervated muscle starts to spontaneously activate action potentials (fibrillations); (b) in weeks, muscles become unable to sustain tension during tetanic contractions induced by electrical stimulation; (c) within months, muscles are unexcitable with standard commercial electrical stimulators[62-66], undergoing ultrastructural severe disorganization of the E-C Coupling and of the contractile apparatuse; and (d) after years, the myofibers are replaced by adipocytes and collagen.

To counteract the progressive changes that transform muscle into an unexcitable tissue, over the past 20 years we have developed a novel therapy concept for paraplegic patients with bilateral and complete LMN denervation of the lower extremity due to complete lesions of the conus and cauda equina. This new training strategy became possible because of the development of a new generation of stimulation equipment specifically designed for home-based functional electrical stimulation (hbFES). These new stimulators and the large surface electrodes necessary to cover the denervated muscles were developed by the Center of Biomedical Engineering and Physics at the Medical University of Vienna and by the Wilhelminenspital, Vienna (Austria), to reverse long- standing and severe atrophy by delivering high- intensity and long-duration impulses that can directly elicit contraction of denervated skeletal fibers in the absence of nerve endings. Our data, indeed, show that hbFES can be an effective home therapy to counteract muscle atrophy and degeneration after complete LMN denervation due to *conus-cauda* lesions. The hbFES device stimulates muscle fibers in the absence of nerve

endings and after prolonged denervation, enabling: i) recovery of muscle mass and fiber size; ii) recovery of tetanic contractility, by restoration of muscle fiber ultrastructure.
Up to now, the muscles of affected extremities in these paraplegic patients are commonly not treated with FES because it is widely accepted that long-term and completely denervated muscles cannot be effectively stimulated. On the other hand, studies in animal models and humans indicate that: i) severe atrophy does not occur in rats for at least 3 to 4 months; ii) in rabbit, the degeneration of muscle tissue does not appear during the first year of denervation; and iii) in humans, muscle tissue degeneration starts from the third year onward. Our recent findings that the long- term denervated rat muscle maintains L-type Ca^{2+} current and gene expression of the related proteins longer than functional contractile machinery [35], provide the molecular, structural, and functional rationale for rehabilitation training of permanently denervated muscles, consistent with clinical observations. This leaves a window of opportunity to initiate muscle stimulation and avoid muscle degeneration and adipose and fibrous tissues accumulation. Our light microscopy results suggest a window for intervention in patients up to 2 years after injury, because fibers maintain at least 30% of their initial size and the extracellular matrix is still evolving. EM analyses, on the other hand, indicate that the structure of the sarcotubular system (reputed to deliver action potential to the fiber interior) and myofibrils decays quite quickly, suggesting that it is best to start hbFES training as soon as possible after SCI, possibly not later than six months. The poor excitability/contractility of human long-term denervated fibers is likely attributable to ultrastructural changes that affect the EC coupling apparatus and contractile elements and precede severe atrophy and degeneration. The reorganization of T- tubules and Ca^{2+} release units and myofibrils that follows hbFES likely plays a role in the recovered ability of LMN denervated muscles to be stimulated and to respond with tetanic contractions.
Because the progression of recovery in hbFES- trained LMN denervated muscle is inherently slow (Figure 3), in part due to exercise training for only 30 minutes per muscle group, 5 times a week, patients were clinically evaluated every 12 weeks by physiatrists, who progressively modified the stimulation parameters and training protocol according to the patient's improvements. During the first few months of hbFES training, the initially poor excitability of the denervated muscle was improved by twitch-contraction training. Three to 6 months later, electrical stimulation induced tetanic contractions against loads that were progressively increased, accompanied by a significant increase in the mass of the quadriceps muscles (24% at the midterm evaluation) and by improvement in limb appearance and muscle cushioning. None of the subjects that reached 1 year hbFES training (n = 20) declined in terms of their muscle properties, and 20% reached the ability to perform stand-up exercise assisted by electrical stimulation of quadriceps muscles.
At 2 years, 90% (n = 20) of hbFES trained subjects recovered or increased tetanic contractions, and 25% stood during electrical stimulation in parallel bars. Minimal functional improvements were associated with long time elapses between SCI and initiation of hbFES and possibly lower compliance with training. In single case reports, low compliance substantially decreased the effects of training, yet in the same subjects the mass of thigh muscles increased when the patient resumed hbFES.
The likelihood that the lower extremities of these patients were completely denervated before initiation of hbFES training and remained denervated during and after the 2 years of training was indicated by several assessments (e.g., test electrical stimulation, needle

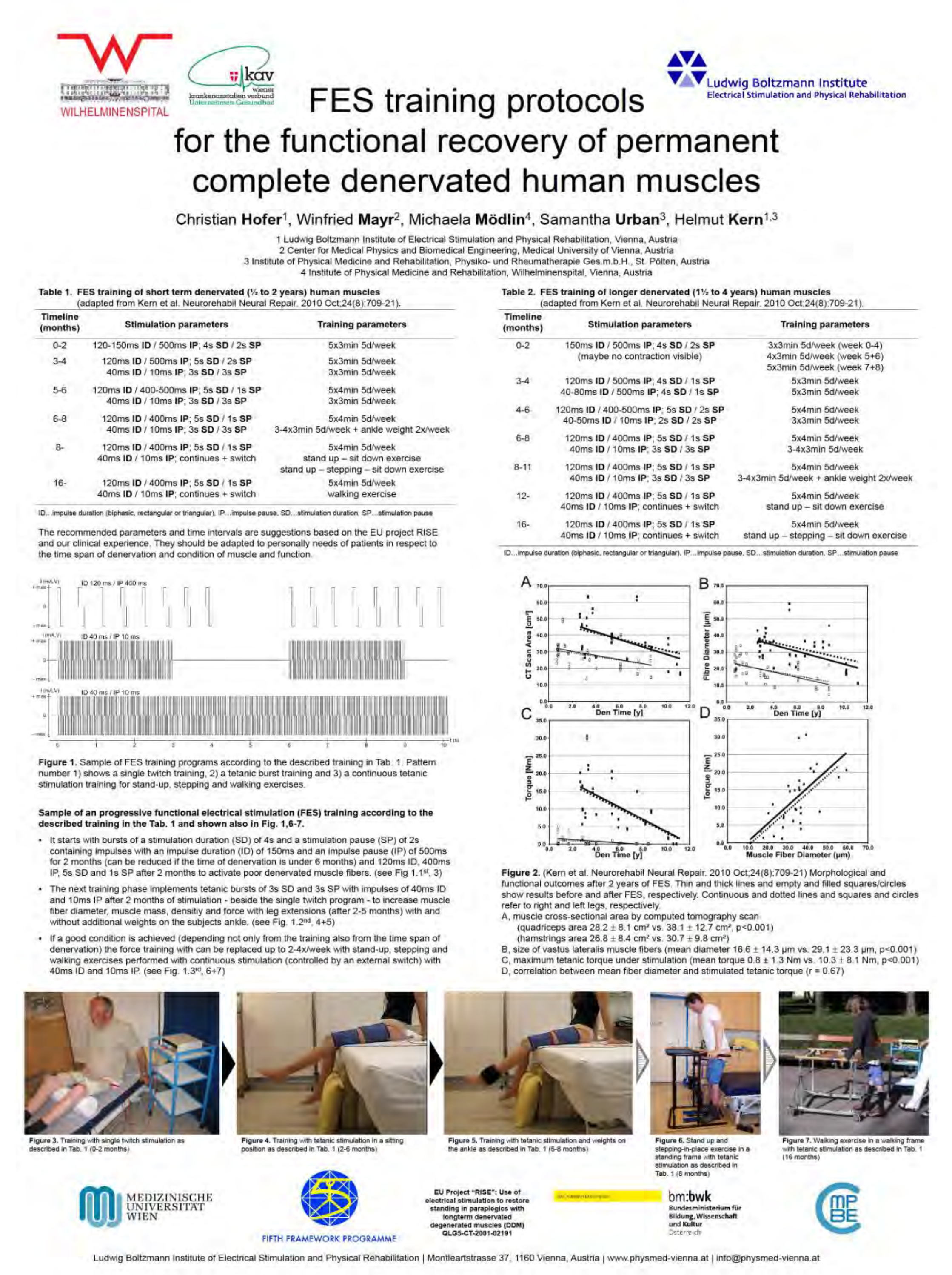

Fig 3. 2017 Spring Padua Muscle Days, Thermae of Euganean Hill (Padua), Italy. Poster of Christian Hofer et al. March 14, 2017. Vienna progressive hbFES strategy for long-term complete denervation of quadriceps muscles

electromyography, transcranial and lumbosacral magnetic stimulation). In particular, the threshold of excitability of the quadriceps muscles never increased to a level that allowed them to respond to standard commercial electrical stimulators (impulse duration about 0.5-2 ms), which elicit a muscle contraction through the nerve. The severity of

postdenervation atrophy (and the extent of hbFES- induced recovery) was similar in the left and right quadriceps muscles of the same patient. In incomplete denervation (or some re-innervation), we would have expected greater variability. Finally, patients did not describe pain during surface stimulation with high current (1000-3000 times higher energy [2.4 J at 120 ms and 200 mA] than that delivered by standard commercial stimulators [0.8 mJ at 0.7 ms and 50 mA]), implying complete sensory loss.
In conclusion, our findings strongly support the RISE rehabilitation protocol as a method to improve the mass and contractility of LMN denervated muscles, although we found a limited “measurable” knee torque changes in hbFES trained muscles. These benefits could be extended to patients with similar lesions, especially to determine whether hbFES can reduce secondary complications related to disuse and impaired blood perfusion (reduction in bone density, risk of bone fracture, decubitus ulcers, and pulmonary thromboembolism).

On the other hand, the Authors share the following suggestions of Gerta Vrbova, which was so kind to attract our attention on the intrinsic limiting factors that will never allow long term denervated muscle to reach by Electrical Stimulation (as it is feasible in clinical settings) the stage of a fully normal muscle. Indeed, our main evidence for muscle denervation even after years of hbFES is the fact that the trained muscles never attain the ability to respond to the much lower currents that stimulate curarized or the denervated muscle fibers early after degeneration of the peripheral nerve stump.
While there is no doubt that impulse activity has a decisive role in determining muscle properties [67], it cannot entirely replace the effect of innervation on denervated muscle. Whether this is due to a trophic influence of nerve on muscle or other factors has not yet been resolved. There are several possible reasons why electrical stimulation cannot entirely mimic the effect of innervation on skeletal muscle: 1. Denervated muscles are stimulated in a manner that causes synchronous contraction of all muscle fibers in the stimulated muscle. This differs greatly from the activity that the nerve is imposing onto the muscle it innervates. During nerve induced movement different motor units are activated asynchronously, and never at the same time [68]. Thus the recruitment order of different muscle fibers is completely different from electrically induced muscle stimulation. 2. The synchronous activity of denervated muscles cannot mimic that which occurs during natural movement and as a consequence the mechanical conditions of different muscle fibers within the stimulated muscle will be far from normal. The amount of load during contraction affects slow muscle fibers more than fast ones; indeed they degenerate if they contract in the absence of load [69]. 3. The simple interpretation of the effect of whole muscle stimulation is therefore limited for synchronous stimulation of all muscle fibers in denervated muscles is very different from nerve induced activity during normal movement.
3. Apart from the superbly organized recruitment order of motor units during normal movement that seems to be necessary for the integrity of the different types of muscle fibers there could be an additional trophic effect of the nerve on muscle but there is little evidence for such an influence that is independent on muscle activity or the mechanical conditions.

We are aware, indeed, that the clinical results may appear poor or very poor to "normal people", but, please, reader consider them from the point of view of a disabled person at risk of serious complications. The increase in mass (cushioning effect) and the anti-gravitational pumping of leg blood are muscle "functions" that are fully lost after denervation, but are substantially recovered during long-term daily electrical stimulation.

Devices and Vienna Stimulation Strategy for hbFES of large denervated human muscles in SCI

To counteract the progressive changes that transform muscle into an unexcitable tissue unable to generate force with standard commercial stimulators (from six months onward), in the past 20 years Clinicians and Engineers developed in Vienna novel rehabilitation concepts for paraplegic patients with bilateral and complete LMN denervation of the lower extremity due to complete lesion of the Conus Cauda [70]. new rehabilitation protocol became possible due to the development and optimization of new stimulation equipments for FES. The devices have been specifically designed to reverse longstanding and severe atrophy of LMN denervated muscles by delivering high-intensity and long-duration impulses that can directly elicit contraction of denervated skeletal fibers in absence of nerve endings. These new stimulators and the large surface electrodes needed to cover the denervated muscles were developed by the Center of Biomedical Engineering and Physics at the Medical University of Vienna, Austria [71,73]. In parallel, specific clinical assessments and training settings were developed at the Wilhelminenspital Wien, Austria [61,74,75]. The rehabilitation progressive training strategy for LMN denervated muscles (see the Figure 3) are validated by the clinical results, strongly supported by those obtained from light and electron microscopy muscle biopsies' analyses performed in Padua and Chieti Universities (Italy), respectively, as described by Kern et al. in the longitudinal prospective study [38,59]. Patients were provided with stimulators and electrodes in order to perform stimulation at home for five days per week. The large (180 cm2) electrodes (Schuhfried GmbH, Mödling, Austria) made of conductive polyurethane, were placed on the skin surface using a wet sponge cloth (early training) and fixed via elastic textile cuffs. As soon as the skin was accustomed to the necessary high current density, gel was used under the polyurethane electrodes to achieve minimal transition impedance. A special design feature was a non conductive bulge along the entire edge of the electrode that prevents potential skin burns that presumably can occur where a conductive edge gets in electrical contact with skin surface and causes local current density hot-spots (Mayr W, 2007; Patent -Surface Electrode, EP2021068, WO/2007/131248). The electrodes were flexible enough to maintain evenly distributed pressure to the uneven and moving skin, thus providing homogeneous current distribution throughout the entire contact area.

Stimulation needle electromyography (SNMEG) was used to study the electrophysiological properties of single muscle fibers. We measured the muscle fiber conduction velocity (MFCV) and the shortest interstimulus interval (ISI) still eliciting a response to the second stimulus delivered to the fiber [76]. MFCV recorded in the denervated patients before and after hbFES therapy showed a significant increase in conduction velocity (fastest and mean CV) and reduced refractory periods (shortest ISI). This suggests that electrical stimulation training is effective to improve the electrical properties of the muscle fibre and SNEMG could serve as an additional measurement technique to specify the status of the denervated muscle. The training strategy consisted of two combined stimulation programs [31,38]. All applicable rules concerning the ethical use of human volunteers were followed during the course of this research (Approval of

Ethical Committee, Vienna, Austria: EK- 02-068-0702). For a multilingual translation of the Work Packages, ethical and safety issues link to: http://www.bio.unipd.it/bam/bam18-2&3.html

At the beginning of the treatment, biphasic stimulation impulses of very long-duration (120-150 ms, 60-75 ms per phase) at high intensity (up to ±80V and up to ±250 mA) were applied (Training Program 1). Then the subjects underwent clinical assessment and stimulated knee torque measurement every 12 weeks by physiatrists, who progressively modified the stimulation. The routine daily training consisted of combined twitch and tetanic stimulation patterns (Training Programs 2, 3 and 4) in consecutive sessions lasting up to 30 min for each group of muscles (gluteus, thigh and lower leg muscles on both sides). After tetanic contractility was achieved and the subject achieved full extension of the leg, the ankle was progressively loaded (Figure 3). Finally, the more compliant patients became able to stand and perform step-in-place and (if young and light) walking exercise [37,38].

Perspectives

In collaboration with his international partners, Dr. Kern is now extending the benefits of hbFES to those subjects, which for different reasons, from the mild but unrelenting process of aging to the devastating fast progression of muscle atrophy in cancer patients, suffer the consequences of muscle weakness. Further, a multi-disciplinary research team of the Interdepartmental Research Center of Myology of the University of Padua is applying the Vienna principles to the apparently easier cases of peripheral incomplete denervation of arms and legs.

Examples in literature of the effectiveness of life-long high-level physical activity in postponing effects of aging [3,4,77], and of physical approaches in peripheral and central neural repair, seems to open new perspectives to an approach, home based Functional Electrical Stimulation in paraplegics by implanted electrodes and neuromodulators, that has been abandoned twenty or more years ago despite the successes of heart pacing and mini-implants for deaf, two very successful cases of Functional Electrical Stimulation of human tissues. HbFES is worth to be reassessed under strict scientific rules, balancing its costs against the needs and legal rights of patients to see alleviated their burdens.

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History of skin results 20 years after conclusion of the RISE project: Plain hematoxylin-eosin histology allows publications in high-level journals

As described in her CV (see Chapter 14), I meet Giovanna (Figure 4) at the Human Anatomy Section of the Department of Neuroscience of the University of Padua, Italy. I don't remember why I was there, but it was the beginning of a research collaboration based on a research tool that I find essential in any muscle study: use results collected by light microscopy.

In time of MiRNA and LNC-RNA research, I know people can't believe that such an approch could be relevant, but it is and could be enough to publish in decent scientific journals. In her CV Giovanna explain much better than me why it is possible. But I must add that without the expert help of Prof. Mauro Alaibac, Head of the Dermatology Section of the Medicine Department of the University of Padua, nothing would have been published. Thank you Giovanna and thank you Mauro for having accepted the invitation to collaborate and even more for the last words of the presentation of Giovanna's CV, below reported!

Fig 4. 2018 Autumn Padua Muscle Days - October 15, 2018 Montegrotto Terme (Padua), Italy. Discussing skin results of the RISE patients more than 20 years after collecting the biopsies. From left: Helmut Kern, Ugo Carraro, Feliciano Protasi, Sandra Zampieri and **Giovanna Albertin, the key researcher for the hbFES skin project.**

I met prof. Ugo Carraro as a colleague in the Faculty of Medicine when he was still officially a professor at the University of Padua, but we became research colleagues only a few years ago. It was one morning in January 2016 when I met prof. Ugo Carraro in the corridors of the Institute of Human Anatomy. I had had the opportunity to read his full-body in-bed gym gymnastics program and since my dad had to be stimulated to do a physical activity to get him back in shape, I immediately found the availability to give me a hand and the desire to give himself do and test his studies and projects. We did some gymnastics sessions with my dad that I then tried to carry on for a while, and I must say that they were very stimulating. At the same time Ugo wanted to involve me in a research that I had understood was in his mind for a long time. It concerned the skin biopsies included in formalin, they were parts of the muscle biopsies of the RISE, European project of prof. Helmut Kern with whom Ugo was been collaborating for several years. The skin biopsies collected between 2002 and 2007 had been put aside because they were not involved in the research of that European project.
With Ugo, and the collaboration of technicians of the Institute of Human Anatomy, we analyzed those biopsies in their epidermal thickness, dermal papillae and Langerhans cells and the results were published on a series of papers, highlighting that regular and continuous hbFES led to an improvement in the thickening of the epidermis in subjects suffering from SCI for different time from neurological injury. The epidermis improved in the formations of the dermal papillae and the Langerhans cells did not differ significantly in number between before and after hbFES such as to allow us to say that the electrostimulation did not involve a statistically significant activation of the Langerhans cell, seen as "sentinel cell of the skin's immune system".
With Ugo and his friend Paolo Gava we also started a collaboration to analyze data from marathon runners and put them in relation to physiological decay, and for this reason I hope to continue to collaborate with Ugo despite his angular and not very patient character, small defects that take second place if one observes his passion for research on different fronts.

Two-years of home based functional electrical stimulation recovers epidermis from atrophy and flattening after years of complete Conus-Cauda Syndrome.

To evaluate progression of skin atrophy during 8 years of complete Conus-Cauda Syndrome and its recovery after additional 2 years of surface Functional Electrical Stimulation a cohort study was organized and implemented. Functional assessments, tissue biopsies, and follow-up were performed at the Wilhelminenspital, Vienna, Austria; skin histology and immunohistochemistry at the University of Padova, Italy on 13 spinal cord injury persons suffering up to 10 years of complete conus/ cauda syndrome. Skin biopsies (n. 52) of both legs were analyzed before and after 2 years of home-based Functional Electrical Stimulation (hbFES) delivered by large anatomically shaped surface electrodes placed on the skin of the anterior thigh. Using quantitative histology in hematoxylin-Eosin slides we analyzed: 1. Epidermis atrophy by thickness and by area; 2. Skin flattening by computing papillae per mm and Interdigitation Index of dermal-epidermal junctions; 3. Presence of Langerhans cells (Figure 5). Linear regression analyses show that epidermal atrophy and flattening worsen with increasing years post- spinal cord injury and that 2 years of skin electrostimulation by large anatomically shaped electrodes reverses skin changes (pre-Functional Electrical Stimulation vs post-Functional Electrical

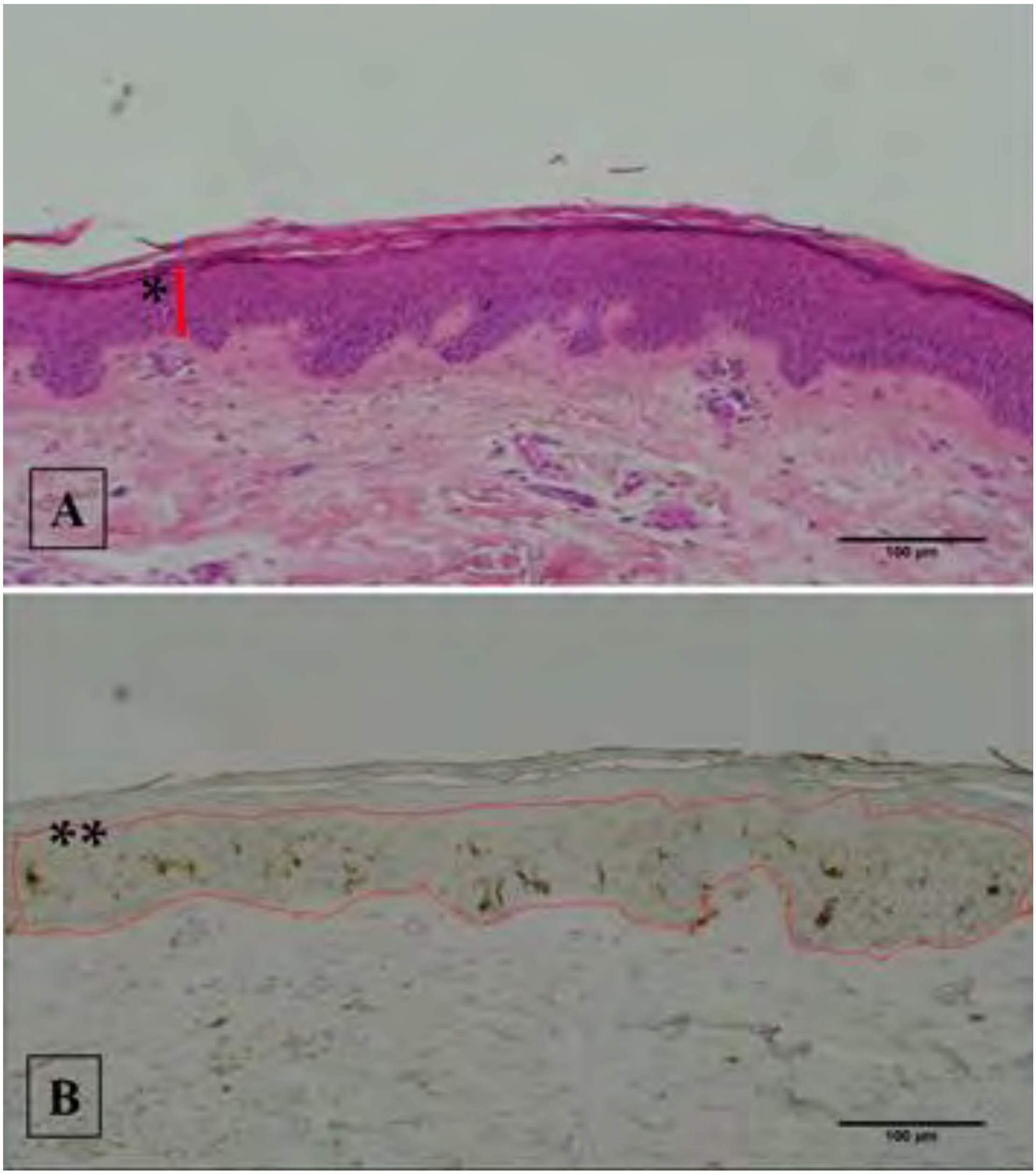

Fig 5. Histological sections of 5 µm thickness were collected and stained by (A) standard Hematoxilin-Eosin (H-E) stain protocol and (B) with a standard immunohistochemical proce-dure to analyze CD1a, a specific marker of Langerhans Cells (LCs); * = the distance between the outermost surface of the epidermis (excluding the stratum corneum) and the dermo–epidermal junction; ** = outline of total area of epidermis.

Stimulation: thickness 39%, $P < .0001$; area 41%, $P < .0001$; papillae n/mm 35%, $P < 0.0014$; Interdigitation index 11%, $P < 0.018$), producing a significant recovery to almost normal levels of epidermis thickness and of dermal papillae, with minor changes of Langerhans cells, despite 2 additional years of complete Conus-Cauda Syndrome. In conclusion, in complete Conus-Cauda Syndrome patients, the well documented beneficial effects of 2 years of surface home-based Functional Electrical Stimulation on strength, bulk, and muscle fiber size of thigh muscles are extended to skin, suggesting that electrical stimulation by anatomically shaped electrodes fixed to the skin is also clinically relevant to counteract atrophy and flattening of the stimulated skin. Mechanisms, pros and cons are discussed in [164].

Trauma of Peripheral Innervation Impairs Content of Epidermal Langerhans Cells.

Langerhans cells represent the first immune cells that sense the entry of external molecules and microorganisms at the epithelial level in the skin. In this pilot case-study, we evaluated Langerhans cells density and progression of epidermal atrophy in permanent spinal cord injury (SCI) patients suffering with either lower motor neuron lesions (LMNSCI) or upper motor neuron lesions (UMNSCI), both submitted to surface electrical stimulation. Skin biopsies harvested from both legs were analyzed before and after 2 years of home-based Functional Electrical Stimulation for denervated degenerating muscles (DDM) delivered at home (hbFES) by large anatomically shaped surface electrodes placed on the skin of the anterior thigh in the cases of LMNSCI patients or by neuromuscular electrical stimulation (NMES) for innervated muscles in the cases of UMNSCI persons. Using quantitative histology, we analyzed epidermal thickness and flattening and content of Langerhans cells. Linear regression analyses show that epidermal atrophy worsens with increasing years of LMNSCI and that 2 years of skin electrostimulation reverses skin changes, producing a significant recovery of epidermis thickness, but not changes in Langerhans cells density. In UMNSCI, we did not observe any statistically significant changes of the epidermis and of its content of Langerhans cells, but while the epidermal thickness is similar to that of first year-LMNSCI, the content of Langerhans cells is almost twice, suggesting that the LMNSCI induces an early decrease of immunoprotection that lasts at least 10 years. All together, these are original clinically relevant results suggesting a possible immuno-repression in epidermis of the permanently denervated patients [165].

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Chapter 8
Paolo Gargiulo, a very successful collaboration. Dreams have come true

I meet Paolo Gargiulo in Vienna, Austria, where he was a PhD student in Medical Engeneering with Prof. Winfried Mayr of the University of Vienna, after his graduation in Medical Enegeneering at the University of Reykjavik, Icaland.
It was very easy to spend time with him and start collaborating speaking Italian!
I visited Reykjavik and the friends I had there, in particular Thordur Helgason. Every time with Paolo we exchanged ideas for collaborating, but the key discussion we had was during the drive to the airport for my back-fly to Italy. Talking about his methods to quantify density by CT scan of bone, cartilage and MUSCLE, I suggested to use false colors to distinguish normal from denervation-degenerating muscle tissue (using colors mimicking histology slides), something Cardiologists were initiating to publish to indentify infarcted cardiac tissue: the 3D Quantitative Color Skeletal Muscle Clinical Imaging was impleneted in few days by Paolo and a very successful and clinical relevant method was established.
Paolo was able to develop 3D Color Skeletal Muscle Imaging to bone and cartilage, further extending it in recent years by Artificial Inteligence approaches obtaining even more clinical significant powerful methods.
But I will start by reproducing below an open access paper from EJTM 25(2) 2015 summarizing the work of the early years.

3D False Color Computed Tomography for Diagnosis and Follow-Up of Permanent Denervated Human Muscles Submitted to Home-Based Functional Electrical Stimulation

European Journal of Translational Myology
Basic Applied Myology - BAM On-Line
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PMID: 26913154

3D False Color Computed Tomography for Diagnosis and Follow-Up of Permanent Denervated Human Muscles Submitted to Home-Based Functional Electrical Stimulation

Ugo Carraro,[1] Kyle J. Edmunds,[2,3] and Paolo Gargiulo[2,3]

Abstract

This report outlines the use of a customized false-color 3D computed tomography (CT) protocol for the imaging of the *rectus femoris* of spinal cord injury (SCI) patients suffering from complete and permanent denervation, as characterized by complete *Conus* and *Cauda Equina* syndrome. This muscle imaging method elicits the progression of the syndrome from initial atrophy to eventual degeneration, as well as the extent to which patients' quadriceps could be recovered during four years of home-based functional electrical stimulation (h-b FES). Patients were pre-selected from several European hospitals and functionally tested by, and enrolled in the EU Commission Shared Cost Project RISE (Contract n. QLG5-CT-2001-02191) at the Department of Physical Medicine, Wilhelminenspital, Vienna, Austria. Denervated muscles were electrically stimulated using a custom-designed stimulator, large surface electrodes, and customized progressive stimulation settings. Spiral CT images and specialized computational tools were used to isolate the *rectus femoris* muscle and produce 3D and 2D reconstructions of the denervated muscles. The cross sections of the muscles were determined by 2D Color CT, while muscle volumes were reconstructed by 3D Color CT. Shape, volume, and density changes were measured over the entirety of each *rectus femoris* muscle. Changes in tissue composition within the muscle were visualized by associating different colors to specified Hounsfield unit (HU) values for fat, (yellow: [-200; -10]), loose connective tissue or atrophic muscle, (cyan: [-9; 40]), and normal muscle, fascia and tendons included, (red: [41; 200]). The results from this analysis are presented as the average HU values within the *rectus femoris* muscle reconstruction, as well as the percentage of these tissues with respect to the total muscle volume. Results from this study demonstrate that h-b FES induces a compliance-dependent recovery of muscle volume and size of muscle fibers, as evidenced by the gain and loss in muscle mass. These results highlight the particular utility of this modality in the quantitative longitudinal assessment of the responses of skeletal muscle to long-term denervation and h-b FES recovery.

Key Words: 3D Color Computed Tomography, False-color, permanent denervation diagnosis, human skeletal muscles, Functional Electrical Stimulation, Three-dimensional reconstruction, Modeling, Tissue composition

The ever-expanding field of medical imaging utilizes a wide variety of techniques and processes to produce non-invasive images of various internal and external tissue morphologies. In the clinical context, medical imaging remains a vital tool for diagnostic and clinical investigations. Of the many facets of the field, most current research aims to improve aspects of instrumentation design, data acquisition methodology, image processing software, and computational modeling. Indeed, three-dimensional (3D) visualization of the internal anatomy provides valuable information for the diagnosis and surgical treatment of many pathologies, but every modality has its inherent limitations.[1,2]
For the purposes of clinical assessment in particular, visually simplistic imaging methods that can optimize the noninvasive, high-resolution assessment of diseased or damaged tissues have readily been identified as a strategic priority in clinical research, and extant imaging modalities have certainly been identified as preferential.[3-11] However, their employment via standard methodology may not be optimal for various avenues of translational myology research. The implementation of traditional imaging modalities, in the context of a variety of novel case studies, can significantly impact this process of methodology optimization.

Here, we review the results of one such case study, involving the use of a customized false-color 3D computed tomography (CT) protocol for the imaging of the *rectus femoris* of spinal cord injury (SCI) patients suffering from complete and permanent denervation, as characterized by complete *Conus* and *Cauda Equina* syndrome.[12-18] Home-based functional electrical stimulation (h-bFES) was applied in these persons using customized electrical stimulators with large electrodes that covered nearly the entirety of the thigh ventral surface.[14-26] The use the reported false-color 3D CT imaging protocol highlights its utility in both confirming the diagnosis of long-term denervation and in quantifying the progression of muscle atrophy to degeneration.[27-33] Furthermore, this method illuminates the importance of h-b FES training compliance with respect to the *rectus femoris* recovery in such patients.

Effects of long-lasting complete denervation of human muscles

Long-standing, complete denervation of human muscles is most often the result of permanent lower motor neuron (LMN) death in patients with either acute damage, such as spinal cord injury (SCI), or pathological conditions, such as peripheral nerve lesions or neuromuscular disorders that result in severe muscular atrophy, apoptosis and fat degeneration of muscle tissue.[34-42] In either case, the progression of denervation in muscle fibers begins with spontaneous activation, i.e. fibrillation, and leads to myofiber atrophy, ultrastructural changes of excitation-contraction coupling, and the gradual loss of excitability in response to external electrical stimulation using standard commercial stimulators.[36] Finally, muscles enter severe atrophy, wherein myofibers undergo the internalization of subsarcolemmal myonuclei, resulting in the regrouping of tens of myonuclei within the center of myofibers and the complete disappearance of long segments of the contractile apparatus.[13] Eventually, muscle fibers all but completely disappear, while fibrous and adipose tissue accumulates.[14] Additionally, based on results of our world-unique CIR-Myo-Bank of LMN-denervated human muscle biopsies, the time course of human muscle atrophy and degeneration has been shown to occur over the order of years – not months, as was common belief based upon rodent models.[42-44] Furthermore, both mid and late phase denervation present three very contrasting myofiber populations: beside those which are severely atrophic with internalized groups of myonuclei, there are large fast-type muscle fibers that continue to be present four-to-six years after SCI,[45,46] and embryonic myosin heavy chains-positive myotubes and myofibers. The last events are evidence of muscle fibers regenerated through satellite cell activation, proliferation and fusion during the last month before muscle biopsy have been harvested, regardless of the time from SCI.[12,47,48] Up to recent years, the LMN-denervated muscles of affected legs in paraplegic patients were not commonly treated with functional electrical stimulation (FES), because it was widely accepted that long-term and completely denervated muscles could not effectively respond to commercially available electrical stimulators due to the assumptive disappearance of a majority of muscle fibers in severely atrophic legs. On the other hand, studies in animal models and humans indicate that denervation-induced severe atrophy and degeneration may occur several months or years later than generally believed. For example, in rats, severe atrophy was shown not to occur for at least four months.[49-51] Likewise, in rabbits degeneration of muscle tissue did not appear during the first year of denervation.[52-55] Indeed, our own recent findings show that rat muscle maintains L-type Ca^{2+} current and gene expression of the related proteins longer than functional contractile apparatuses.[37] Additional investigations in human patients showed that irrecoverable muscle tissue degeneration can be delayed up to three years, and indeed light microscopy analysis showed that myofibers retain at least 30% of their initial size over this duration, confirming at least a two-year window for possible FES intervention.[12-26] However, electron microscopy investigations on patients biopsies indicated that the decay of excitation-contraction contractile apparatus, i.e., of t-tubules and triads, starts within one year of denervation, suggesting that for the best results the h-b FES treatment must start as early as possible.[14,15,17]

Investigating h-b FES treatment of LMN-denervated muscles

Until recently, the mid-term progression of irreversible lower motor neuron denervation was comparatively much less studied that the long-term progression of muscle atrophy in upper motor neuron lesion patients. Hundreds of reports published in the literature describe studies of muscle properties up to three years following upper motor neuron spinal cord injury. Within the first month of injury, the thickness of the muscle bulk, measured by ultrasound, has been shown to decrease by up to 40%.[56] However, after the first few months of significant muscle mass loss, studies show that the atrophy process slows down considerably.[57-58] Indeed, a 50% stable atrophy in spastic incomplete paralysis is well documented up to two years after SCI.[59-63] Information regarding patients up to 20 years after SCI was relatively unavailable before our published results showed stable muscle atrophy in long-term paraplegics with complete upper motor neuron lesion 3 to 20 years after SCI.[64]. FES is also applied to complete thoracic SCI patients to activate their leg muscles under the SCI level.[59-63] However, it should be noted that almost all of these studies were related to FES in thoracic-level paraplegics that responded with tetanic muscle contraction when stimulated with commercial electrical stimulators.[65] All these features are in sharp contrast to the type of spinal cord injury that involves lower motor neurons lesion, because this causes actual denervation of the affected muscles. The disease process is particularly severe when a complete transverse SCI involves all the lower motor neurons of the affected muscles. This is precisely the case of complete, irreversible *Conus* and *Cauda Equina* syndrome, wherein patients exhibit complete LMN denervation of the leg muscles.[66] Complete LMN denervated muscles are, indeed, soon unable to sustain tension during tetanic contractions induced by electrical stimulation, as the long-lasting severe atrophy of the permanently inactive muscles is worsened by the replacement of myofibers with adipocytes and collagen.[13-18,67-69]

As previously mentioned, there is a clear rationale for the existence of a window of opportunity to initiate muscle stimulation that may not only delay or avoid muscle degeneration in LMN-denervated patients, but could also recover contractility of the atrophic muscles. While several stimulators and rehabilitation strategies are commercially available for performing FES on patients with upper motor neuron damage due to SCI, for patients suffering from complete LMN damage, the protocols and home-based system developed and tested by the EU program "RISE" (Vienna, Austria) is the clear gold standard.[17,18] This h-b FES system utilizes large surface electrodes fabricated from conductive polyurethane and custom-designed muscle stimulators to provide a homogeneous current distribution throughout the entire contact area. Two years of h-b FES training results in: 1) a significant increase of thigh muscle size and of the muscle fibers, with striking improvements of the ultra-structural organization of contractile structures and E-C coupling mechanisms;[14-18] 2) a significant increase in muscle force output during electrical stimulation that is sufficient to perform h-b FES-assisted standing and in-place stepping exercises;[14-18] 3) evidence that the number and the size of recovered muscle fibers is inversely proportional to the elapsed time between a SCI event and the beginning of h-b FES.[14-18] The reorganization of T-tubules, Ca^{2+} release units, and myofibrils that follows h-b FES likely plays a role in the recovery of LMN denervated muscles, as well as their ability to be stimulated and to respond with tetanic contractions.[14-18]

Patients were provided with stimulators and electrodes in order to perform stimulation at home for five days per week. The large (180 cm2) electrodes (Schuhfried GmbH, Mödling, Austria), made of conductive polyurethane, were placed on the skin surface using a wet sponge cloth (early training) and fixed via elastic textile cuffs. As soon as the skin was accustomed to the necessary high current density, gel was used under the polyurethane electrodes to achieve minimal transition impedance. The electrodes were flexible enough to maintain evenly distributed pressure to the uneven and moving skin, thus providing homogeneous current distribution throughout the entire contact area. The particular h-b FES training strategy consisted of four combined stimulation programs.[14-20] Additionally, since the progression of recovery by h-b FES is inherently slow, patients were clinically evaluated every 12 weeks by physiatrists who progressively modified their training protocols according to the patient's improvements.[16-18] It should likewise be noted that all applicable rules concerning the ethical use of human volunteers were followed during the course of this research (Approval of Ethical Committee, Vienna, Austria: EK-02-068-0702). For a multilingual translation of the work packages, ethical, and safety issues, visit: http://www.bio.unipd.it/ejtm/ejtm18-2&3.html.[25,26]

Muscle tissue segmentation and visualization using false-color 3D Computed Tomography

Discrimination of tissue types, densities, and volumes is of particular interest in the medical imaging research community. The post-processing of 3D CT images via colorization of these morphologies holds particular utility with regards to the segmentation of skeletal muscle. Specific attenuation values are assigned to individual volumetric elements (voxels), based upon the degree to which CT X-rays of given energies transmit through the volume element.[17,27-33] The degree of attenuation depends on the energy spectrum of the incident X-rays, as well as the average atomic number of the tissues of the patients. Since most computers utilize hardware that requires integer values, linear attenuation coefficients are rescaled to an integer range that encompasses 4096 values, between -1000 and +3095. This scale is known as the CT number, which may likewise be readily converted to Hounsfield units (HU). Comparatively dense tissues, such as bone, have large attenuation coefficients and thereby large, positive CT numbers; conversely, large, negative CT numbers are typical for low-density tissues, such as lung and adipose tissue. Muscles are normally displayed with CT number between 50 and 100 Hounsfield units, although within a normal muscle belly, other tissue elements are typically present, such as connective tissue and fat, which have much lower CT numbers. If a singular tissue type occupies a particular voxel, that element will possess a readily-identifiable CT number corresponding singularly to that tissue type; however, typical voxels simultaneously contain several tissue types and thereby express an average CT number whose value is proportional to the ratio of the tissue types. This phenomenon explains the wide range of values present within a particular dataset and suggests the necessity for increased voxel resolution and the development of novel intervoxel segmentation methods to optimize the study of muscle structural changes, in particular for 3D Color Muscle CT.[27-33]

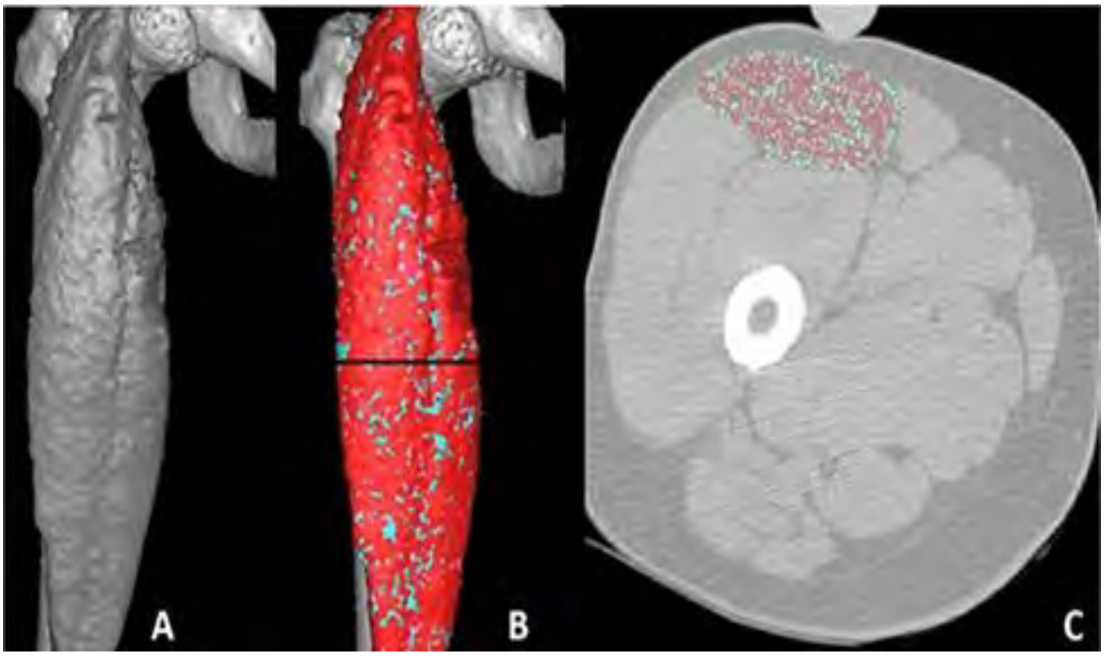

Fig 1. *3D false-color CT reconstruction of a rectus femoris muscle from a healthy patient, including the epimysium at the cortical level in gray (A) and false colors (B), along with the corresponding cross-section at mid belly (C). The total volume is 4.2×105 mm3, and from this volume, 3% is fat [-200 to -10] HU, 28.7% is loose connective tissue and low-density (atrophied) muscle [-9 to 40] HU, and 68.3% is normal muscle, if fascia and tendons are included [41 to 200] HU.*

Protocol for 3D Color CT of long lasting muscle denervation

Despite being widely used as an imaging modality in cardiology,[1,2] the false color approach to spiral CT is typically ignored in clinical imaging-based evaluations of skeletal muscle tissue. As is typical of any spiral CT protocol, 3D data are gathered by scanning the patient's lower limbs with a spiral CT machine. For the purposes of this study, the imaging software MIMICS (http://www.materialise.com) was utilized to isolate and monitor the *rectus femoris* within the quadriceps muscle. 3D reconstruction is possible in the *rectus femoris* – even when severely degenerated due to long-term denervation, as the muscle remains readily recognizable despite severe atrophy of thigh muscles.

As evidenced by Figure 1, a typical leg scan starts above the head of the femur and continues down to the knee joint, covering both legs with one scan. Slice increments are set to 0.625mm, resulting in a total of about 750–900 CT slices, depending on the patient's size. Each slice consists of 512×512 pixels, and each pixel has a gray value in the HU scale of 4096 grayscale values, meaning that pixels may be represented by a 12 bit value. This dataset is effectively a complete 3D description of the particular morphological structure, including all tissue types. The size of the volumetric element (voxel) in the dataset is about 0.7 mm ; therefore, as previously alluded to, the CT number assigned to these voxels often represented an average of different tissue elements. For instance, in the case of normal muscle tissue, such a voxel would contain the transverse section of 20 to 50 muscle fibers with their inter-myofiber elements (endomysium, nerve terminals, adipocytes, and, in

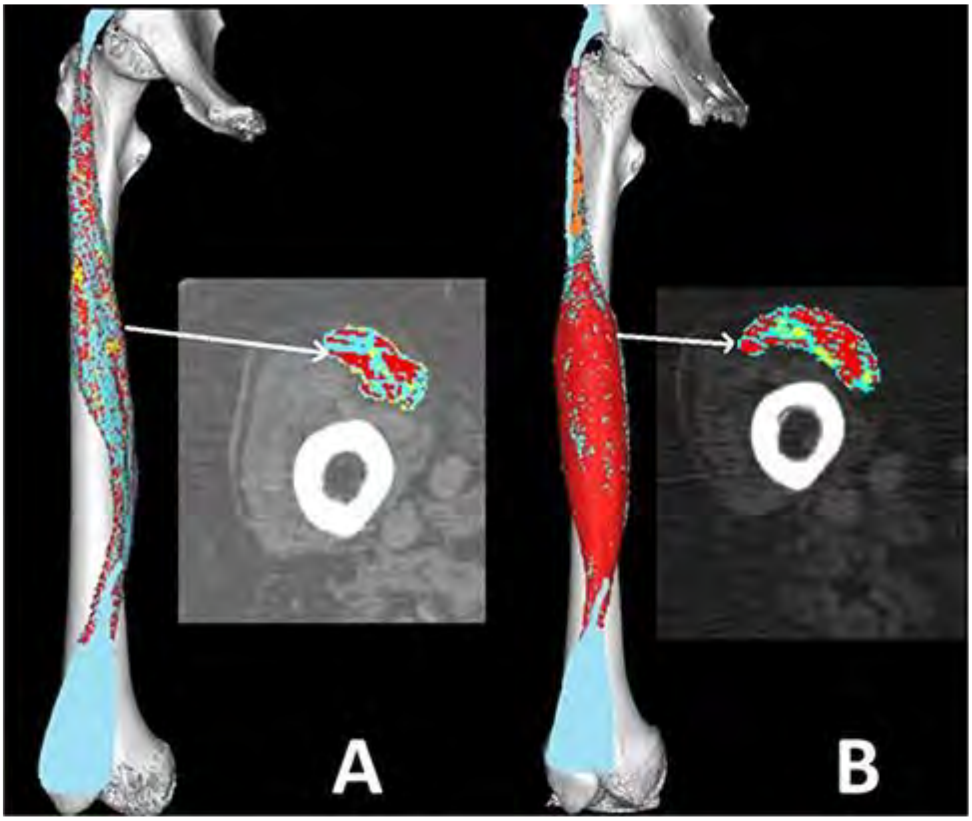

Fig 2. *3D reconstructions of rectus femoris from the representative SCI patient at the following three time points: A) four years after SCI, B) four years after starting h-b FES, and C) four years after discontinuing treatment. The topmost curve outlined as a, b, and c illustrates the increase and decrease of muscle volume at these time points, from 1.0×105 mm3, 1.7×105 mm3, and 0.72×105 mm3, respectively. The primed curve below (a', b', and c') shows the increase and decrease of HU-based densities at these time points, from 42.5, 47.5, and 31.7, respectively. Note that the volumes are calculated identifying the epimysium, i.e. the cortical level of muscle.*

particular, capillaries, arterioles, and venulae). It is important to note that the volume of a voxel corresponds approximately to one tenth of the volume accessed by a typical muscle needle biopsy, and that this slicing size allows for the microstructural evaluation of muscle quality via volume and density. The results of this microstructural analysis are presented as a 3D reconstruction containing the percentage of different tissues within the total volume of the *rectus femoris* muscle (Figure 1). In this reconstruction, only the first cortical layer of voxels describing the muscle epimysium is depicted. It should be stressed that these images depict the tissue distribution at the level of the muscle surface (epimysium), but do not coincide with the distribution through the entirety of the muscle volume. Therefore, to monitor and estimate the tissue composition of the stimulated muscle volume, the computed tomography number present within the segmented volume were subdivided into three HU intervals and displayed with different colors as follows: [-200 to -10], [-9 to 40], and [41 to 200] representing respectively: fat (yellow), loose connective tissue and atrophic muscle (cyan), and, if fascia

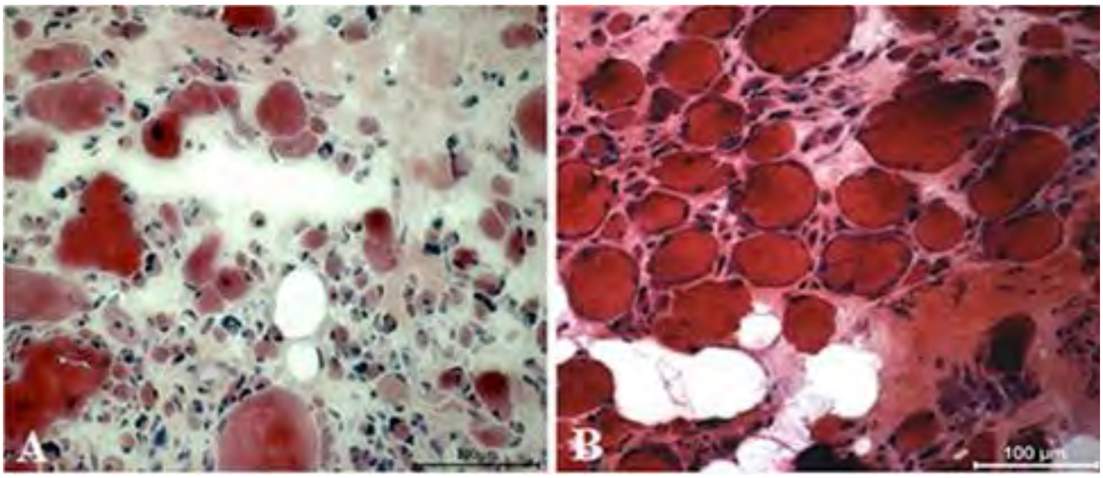

Fig 3. *Histological analysis of the muscle biopsies harvested before (panel A) and after three years of h-b FES (panel B). Hematoxilin-Eosin stain. Interestingly the area of the cryosections covered by muscle fibers profile in before and after h-b FES specimens are 15% and 37%, respectively, a behavior that is in good agreement with the results of the total volume of the rectus femoris before and after h-b FES, respectively. Accordingly the sparse muscle fibers present before h-b FES have mean diameter of less than 10 µm, while the muscle diameter increase to 37 µm in the after h-b FES biopsy. These results are in good agreement with the overall correlation tests among muscle strength, transverse CT area of the quadriceps muscle and diameter of the muscle fibers before and after h-b FES in the series of LMN denervated person enrolled in the EU Project RISE.16,17Bar = 100 µm.*

Evaluation of the impact of h-b FES

This section details the results from the 3D segmentation and false-color analysis of both a healthy, active subject and a SCI patient with complete LMN denervation who was subjected to four years of h-b FES as previously described, but starting four years from SCI. Firstly, the mean HU value of normal leg muscle, collected from the healthy, young sportsman, was found to be between the mean density of the sub-patellar tendon and the skin. The tendon density may, thus, be taken as the upper limit reference of the range of values for an healthy muscle tissue, as both exhibit a comparatively high protein content. Figure 2 illustrates the results from the false-color analysis of the SCI patient's *rectus femoris* after four years of denervation (Figure 2, panel A) and additional four years of h-b FES therapy, i.e., at eight years from SCI. Accordingly, Figure 2A shows that in a longitudinal study of our SCI patient, four years of denervation elicited a mean HU value of 42±20, which is comparatively much less than that of the healthy sportsman (59±36). Additionally, the volume of the *rectus femoris* was a full order of magnitude less than a typical healthy subject (1.0×10^5 mm^3 for the SCI patient, compared to 4.2×10^5 mm^3 computed previously in Figure 1). However, after four years of h-b FES, the SCI patient's muscle volume nearly doubled to 1.7×10^5 mm^3. In addition, the three measured tissue compositions changed dramatically over the period of treatment: fat composition went from 8% to 4%, loose connective tissue and atrophied muscle went from 47% to 36%, and

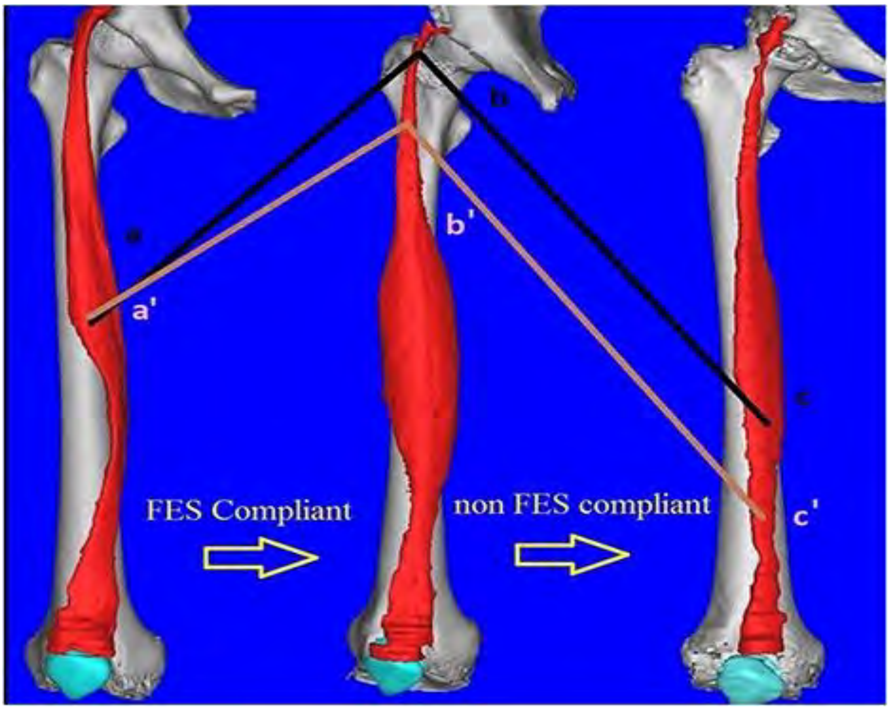

Fig 4. *hbFES-induced recovery of LMN denervated human muscles from atrophy/degeneration is fully dependent from patient's compliance. 3D reconstructions of rectus femoris from the SCI patient at three time points: a, a') four years after SCI, b, b') after four years of h-b FES, and c, c') four years after discontinuing h-b FES training. The topmost curve outlined as a, b, and c illustrates the increase and decrease of muscle volume at these time points, from 1.0×105 mm3, 1.7×105 mm3, and 0,72×105 mm3, respectively. The primed curve below (a', b', and c') shows the increase and decrease of HU-based densities at the same time points, from 42.5, 47.5, and 31.7, respectively. Note that the volumes are calculated identifying the epimysium, i.e. the cortical level of the muscle.*

normal muscle went from 45% to 60%. After four years of treatment, these muscular composition percentages were nearly recovered to those of the healthy adult example depicted in Figure 1 (3%, 27.8%, and 68.3% for fat, loose connective tissue and atrophied muscle, and normal muscle, respectively).

Figure 3 shows the results of the histological analysis of the muscle biopsies harvested before (panel A) and after three years of h-b FES (panel B). Interestingly, the area of the cryosections covered by muscle fibers profile in before and after h-b FES specimens are 15% and 37%, respectively a behavior that is in good agreement with the results of the total volume of the *rectus femoris* before and after h-b FES, respectively. Accordingly the sparse muscle fibers present before h-b FES have mean diameter of less than 10 µm, while the muscle diameter increase to 37 **µ**m in the after h-b FES biopsy. These results are in good agreement with the overall correlation tests among muscle strength, transverse CT area of the quadriceps muscle and diameter of the muscle fibers before and after h-b FES in the series of LMN denervated person enrolled in the EU Project RISE.[16,17] To better appreciate the extent of dependency of muscle recovery and its reversibility on compliance with h-b FES treatment, it may be useful to compare the h-b FES recovery of the four-year-denervated muscle to its degeneration after four years of discontinued treatment (Figure 3). Intriguingly, the degree to which the atrophied *rectus femoris* recovered muscle volume was mirrored by the subsequent reversal of recovery after the discontinuation of h-b FES (Figure 4).

As is evident in Figure 4, average HU values dropped from 47.5 to 31.7 HU after discontinuation of h-b FES treatment. Likewise, muscle volume dropped from 1.7×10^5 mm^3, and 0.72×10^5 mm^3; both HU values and muscle volumes ended up substantially lower than they were prior to h-b FES treatment. The reported mean HU values are heavily influenced by the capillaries (open or closed) and content of fat (mean value -100 HU), but also by the ratio of muscle sarcoplasm to extracellular fluid, which usually increases due to the absence of muscle contractions in the legs. Table 1 depicts the comparison between measured mean HU values of various tissues in the healthy, young sportsman and the SCI patient.

To begin discussing the utility of the h-b FES therapy, it must first be stressed that the amplitude of electrical current needed to extend the knee still remained more than 100-1000 times larger than that which is required for normally-innervated human muscles, demonstrating that the recovered function is not the results of reinnervation – a phenomenon which is not known to occur at such an extended time following SCI.[63] In general, the case study highlights the utility of h-b FES for SCI patients with LMN denervation. Indeed, the muscle recovery in Figure 4 clearly illustrate an increase in *rectus femoris* volume and quality during four years of h-b FES training despite four years of prior LMN denervation. Likewise, after discontinuing therapy, our results clearly show worsening of *rectus femoris* degeneration, highlighting the importance of life-long sustaining h-b FES treatment in SCI patients. Important additional benefits for these patients include the improved cosmetic appearance of lower extremities, the enhanced cushioning effect for seating, and the overall reduction of leg edema.[16] These benefits may additionally be extended to patients with similar lesions (incomplete LMN denervation of skeletal muscles) – especially with regards to determining whether h-b FES can reduce secondary complications related to disuse and impaired blood perfusion (reduction in bone density, risk of bone fracture, decubitus ulcers, and pulmonary thromboembolism). Hence, it is of special interest to monitor and study the electrophysiology of the *rectus femoris* – specifically with regards to the modeling and measurement of electrical stimulation effects. Indeed, our previous works in assessing segmentation techniques and related computational tools were used to isolate the *rectus femoris* from other proximal muscle bellies and analyze the muscle in a novel way.[27-33]

In general, it may be recommended that h-b FES should be applied to all denervated leg muscles (from the gluteus to the foot muscles), but the main target muscle group for this therapy should be the quadriceps femoris, which consists of the four muscles on the front of the thigh: *rectus femoris*, *vastus lateralis*, *vastus medialis* and *vastus intermedius*. The muscle volume in the quadriceps is very large, and the muscles are not uniformly activated by external electrical stimulation. In fact, surface electrodes are by definition non-selective, and enormous amounts of energy is used to deliver electrical stimuli to denervated muscles. The *rectus femoris* occupies the middle of the thigh and covers most of the other three quadriceps muscles; hence, it is closer to surface electrodes and, therefore, more exposed to external stimulation – a notion which was evidenced in a study using T2 mapping.[70,71]

Advantages and disadvantages of 3D Color CT imaging, segmentation, and reconstruction

The particular computational method depicted herein is founded upon critical thresholding criteria and CT numbers, which are in-turn used to define different tissues within the muscle. The main advantage of using segmentation technique and 3D modeling is that it provides the option to analyze the whole muscle and not a small part of it. This notion is particularly valuable with respect to the presented case study, as we were able to readily isolate the *rectus femoris* as our muscle belly of interest. A further advantage of this method is that by adjusting the dimensions of the voxel size utilized, one may investigate various tissue compositions at the microscopic level. With this approach, the characteristics observed on the macroscopic level can be compared and correlated to measurements made on much smaller samples, and these results can serve as a validation of specific details from muscle biopsies. However, the main limitation for the use of this technique is related to the radiation dose, which the patient absorbs during the scanning process, and the overall reliability of CT numbers to discriminate tissue composition, as various physical factors can influence CT number representation during a scanning session. The parameter which mostly affects the accuracy and the spatial distribution of CT numbers is the applied voltage across an X-ray tube; this amplitude is measured in kilovolts and dictates the maximal X-ray energy and, therefore, the attenuation coefficient. CT number distributions are also influenced by phantom (or patient) orientation within the scan aperture. Therefore, it is necessary to recognize and account for these variabilities when CT numbers are employed for tissue characterization and comparison. Therefore, to avoid or at least limit possible discrepancies in CT number assignments, in the beginning of the clinical trial, a scanning protocol should be established and calibration tools employed during every measurement (a rule of thumb that was followed in the presented study). Additionally, to limit the patient dose, spiral CT technology should be used to gather 3D data and volumes of interest in order to collect necessary information in the shortest possible period of time. We are confident that this case study, in accordance with other related works from the EU Program RISE, may help to convince clinicians to extend h-b FES observation to a larger cohort of cases. Specifically, the case study vividly highlights the use of 3D false-color CT as a non-invasive imaging procedure designed and implemented to objectively demonstrate the improvements in muscle mass and contractility, despite complete LMN denervation. The reported method describes a perspective opportunity for generating a multidisciplinary approach to monitor myopathies, based on this and other pre- and end-point analyses, such as muscle biopsy and functional muscle ultrasound analyses.[72] All together, these methods may reopen the possibility for testing therapeutic rehabilitation approaches in shorter time frame studies. In conclusion, the study presented herein outlines the particular utility of this modality in quantitative longitudinal assessments of the responses of skeletal muscle to long-term denervation and to h-b FES induced recovery. Ongoing research is aimed to select other muscle views (multiple or mid-belly transverse section, multiple or mid-belly vertical section, etc.) to identify a panel of the most descriptive presentations. Additional analytical methods of muscle tissue volume and density may be found in other published clinical imaging articles,[74-75] in studies of aging[76] and are described in detail in other reviews and articles of the EJTM Special “News on clinical imaging of muscle tissue”.[77-81]

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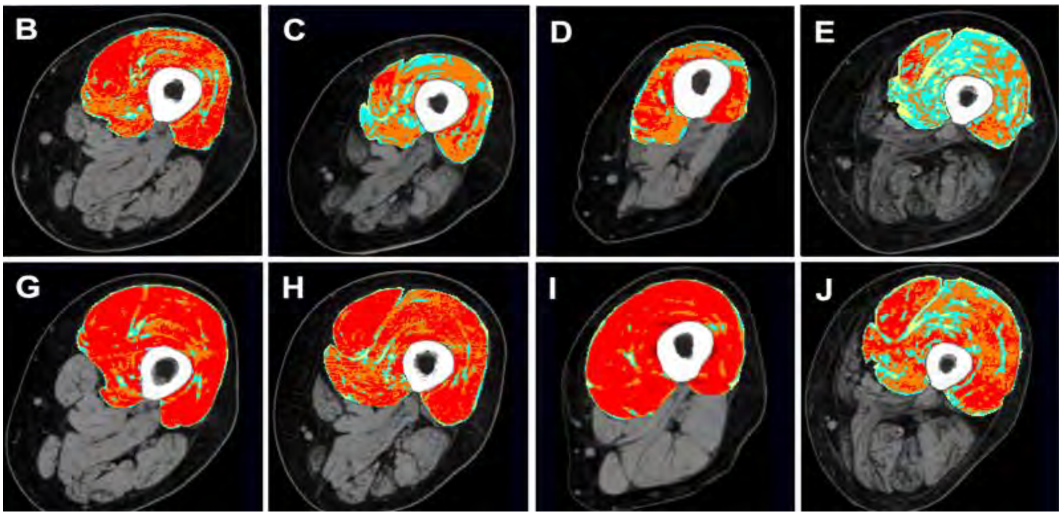
Additional evidence of the usefulness of the 2D Color Computed Tomography were published in: Kern H, Carraro U, Adami N, Biral D, Hofer C, Forstner C, Mödlin M, Vogelauer M, Pond A, Boncompagni S, Paolini C, Mayr W, Protasi F, Zampieri S. Home-based Functional Electrical Stimulation (h-bFES) recovers permanently denervated muscles in paraplegic patients with complete lower motor neuron lesion. Neurorehab Neur Rep 2010;24:709-21.

From that key paper I here describe the most intriguing Figure, as redrow for one of my favorite slides. I use it to teach this topic to Specilists of Physical Medicine and Rehabilitation of the University of Padua, Italy and beyond.

2 D Color CT at 1 (B), 2 (C), 4 (D) and 6 (E) years from SCI and after two additional years of h-b FES (G, 3-ys; H, 5-ys; I, 6-ys; J, 8-ys from SCI)



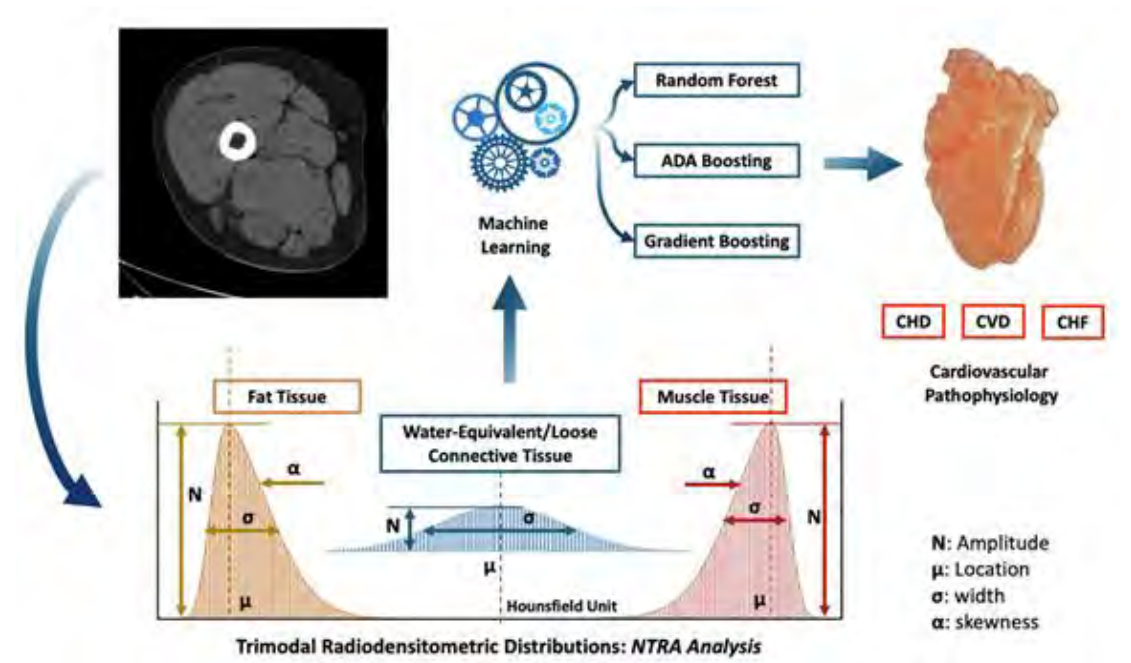
Color CT scans of thigh muscles before and after 2 years of home-based functional electrical stimulation (h-bFES).
The cross-sectional area of quadriceps muscles in patients starting h-bFES at different time points after denervation (B, 1.2; C, 1.7; D, 3.2; E, 5.4 years) increased after 2 years of home training (G, H, I, J, respectively). Moreover, the interstitial tissues that increase with the denervation time (compare yellow, green, and blue areas in panels B, C, D, and E) decreased in the respective patient after 2 years of h-bFES (G, H, I, J, respectively)

I find this panel a compelling evidence that any paraplegic patient can understand: muscle tissue gets worse with years of denervation, but hbFES is able to not only delay, but reverse the process!

A third dream become reality.

Paolo Gargiulo continues to produce excellent results with his group of international collaborators, including brilliant young Biomedical Engineers from Reykjavik University research group and his native University of Naples. In recent years he continued to work on medical images moving forward to muscle profiling.

Paolo group developed a novel technology to assess radiological images based on nonlinear trimodal regression analysis (NTRA) parameters in characterizing changes in soft tissue radiodensity as a quantitative construct for sarcopenia in the longitudinal, population-based cohort of the AGES-Reykjavík study. NTRA features were firstly introduced in [303] and they consist in 11 patient-specific radiodensitometric parameters extracted from the Hunsfield Unit distribution of fat, muscle and connective tissue extracted from a mid tigh CT-Scan. Healthy elderly volunteers from the AGES-Reykjavík cohort underwent mid-thigh X-ray CT scans along with a four-part battery of LEF tasks: normal gait speed, fastest-comfortable gait speed, isometric leg strength, and timed up-and-go. These data were recorded at two study timepoints which were separated by approximately 5 years: AGES-I (n = 3157) and AGES-II (n = 3098). Participants in AGES-I were likewise administered a survey to approximate their weekly frequency of engaging in moderate-to-vigorous physical activity (PAAGES-I). Kyle Edmunds researcher at Paolo institute contributed dramatically in the development and validation of the NTRA methodology. Using a multivariate mediation analysis framework, linear regression models were assembled to test whether NTRA parameters mediated the longitudinal relationship between PAAGES-I and LEFAGES-II; all models were covariate-adjusted for age, sex, BMI, and baseline LEF, and results were corrected for multiple statistical comparisons. Our first series of models confirmed that all four LEF tasks were significantly related to PAAGES-I; next, modelling the relationship between PAAGES-I and NTRAAGES-II identified muscle amplitude (Nm) and location (μm) as potential mediators of LEF to

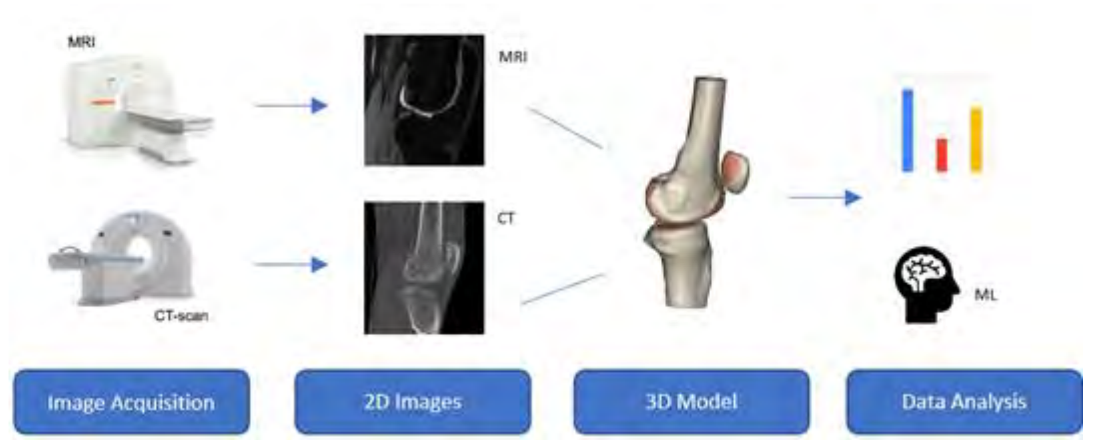


Wokrflow of the study published on Scientific Report [304]: from a mid-thigh CT scan, 11 radiodensitometric distributions parameter from fat, muscle and connective tissues, are extracted and used as features for assessing cardiovascular risks through three tree-based algorithms.

test. Finally, adding these two parameters into our PAAGES-I → LEFAGES-II models attenuated the prior effect of PAAGES-I; bootstrapping confirmed Nm and μm as significant partial mediators of the PAAGES-I → LEFAGES-II relationship, with the strongest effect found in isometric leg strength. This work describes a novel approach toward clarifying the mechanisms that underly the relationship between physical activity and LEF in aging individuals. Identifying Nm and μm as significant partial mediators of this relationship provides strong evidence that physical activity protects aging mobility through the preservation of both lean tissue quantity and quality [306].
Further development on the NTRA technology were introduced by the PhD candidate Marco Recenti who started his Phd program under Paolo's supervision in January 2020 after his master degree in ICT engineering at Politecnico di Torino. His researches focus mainly on Artificial Intelligence, in particular Machine Learning and prediction models for assessment and diagnostic in advanced Digital Helth Engineering. Marco work was recently awarded at the IEEE Metroxraine Conference in Rome. Together with me, Paolo, and other Reykjavik University engineers he published multiple studies with important scientific relevance also on EJTM [205,206].
The most impactful results were obtained from the AGES Reykjavik dataset study where the value of nonlinear trimodal regression analysis (NTRA) parameters has been recognized in characterizing changes in soft tissue radiodensity as a quantitative construct for sarcopenia in the longitudinal, elderly population-based cohort of the AGES-Reykjavík studyThey were used as input for multiple Machine Learning models to classify various comorbidities using the two AGES study timepoints which were separated by approximately 5 years: AGES-I and AGES-II.
A first preliminary work, to understand the prediction potential of NTRA, was published in EJTM [205,207]: NTRA were here able to predict body mass index and isometric length strength using tree-based algorithms with a regression coefficient of determination of 0.83. With these results NTRA were proved to be efficient with a regression approach. The following study used NTRA to assess cardiovascular risks on AGES database and was published on Scientific Report [208] using always a Machine Learning approach but switching from regression to classification models. Fig 301 shows the workflow of the research: it is possible to graphically see the 11 different features from the fat, connective tissue and muscle HU distributions used here to classify coronary heart disease (CHD), cardiovascular disease (CVD), and chronic heart failure (CHF) with multivariate logistic regression and three tree-based ML algorithms. The best algorithm results random forests using a K-Fold cross validation. It generated the highest classification performance obtaining excellent metrics for all three conditions: CHD (AUCROC: 0.936); CVD (AUCROC: 0.914); CHF (AUCROC: 0.994). Longitudinal assessment for modelling the prediction of CHF incidence between AGES-I and AGES-II was likewise significantly robust (AUCROC: 0.993). Furthermore, a tissue-based feature importance analysis was also performed, highligting a noteworthy relevance of the connective tissue in the classification process.
The following study that used the same NTRA features was published on IEEE Journal of Biomedical and Health Informatics [305]. It was focused on the classification of Diabetes (DM) and Hypertension (HTN) and the influence of past and present lifestyle of the elderly population of AGES-I and II. The approximately 3 thousands subjects were arranged into a binary-tree structure composed of three levels: the first was about lifestyle factors based on smoking and self-reported physical activity; the second focused on the presence of HTN or DM; finally the third included the cardiac pathophysiology classified in [304].

Differences were evaluated at each binary-tree level, and subsequently the same tree-based machine learning (ML) models, already proved to be highly efficient, were performed to classify subjects with DM or HTN. Classification metrics for predicting HTN or DM based on the first level of the binary-tree (lifestyle factors) were superb (AUCROC: 0.978 and 0.990, respectively). Moreover, the soft tissue importance analysis underlined again the elevated impact of the connective tissue parameters. Furthermore, a five-year longitudinal model to predict DM from AGES-I to AGES-II gave an excellent classification accuracy of 94.9%. A forth study relative to the relation between NTRA, age, and physical activity was published on EJTM [206].
Here a lifestyle index similar to the one of [305] is classified splitting the AGES data according to three different age groups. The classification accuracy is never less than 80 and sensitivity can reach more than 96 for the older group (80-93 ys)

Finally Paolo Gargiulo team has been involved in research on cartilage 3D modeling and development of novel evaluation metrics. This work aims to find a new solution to assess cartilage condition based on new-investigated features. It is part of the European projects RESTORE (https://restoreproject.eu/) and SINPAIN (https://www.osteoarthritis-sinpain.eu/). RESTORE aims to develop and validate solutions for personalized knee cartilage regeneration, while SINPAIN aims to improve the quality of life for those affected by OA and reduce the substantial costs linked with the disease by developing a new generation of OA therapeutics. In this frame, our objective is to develop a patient-specific multiscale profile capable of accurately describe cartilage and bone conditions. The profile will be used to support the decision-making process and the customizing of tissue engineered solutions.
A key person in this research is Federica Kiyomi Ciliberti, graduated in Digital Health and Bioinformatic Engineering at the University of Salerno, Italy, and currently working in Paolo group. Federica research has been focusing on the role of bone mineral density and cartilage volume to predict knee cartilage degeneration [204].
Knee Osteoarthritis (OA) is a highly prevalent condition affecting knee joint that causes loss of physical function and pain. Clinical treatments mainly focuses on pain relief and



Support Vector Machine (SVM) and Logistic Regression (LR) were used to assess the capability of the extracted features to distinguish the two classes – D versus ND. The models were validated using LeaveOne-Out (LOO), 5- and 10-fold cross validation (CV) limitation of disabilities; therefore, it is crucial to find new paradigms assessing cartilage conditions for detecting and monitoring the progression of OA. [211]

Forty-seven subjects were enrolled for this study, divided into two groups: Degenerative (D) group, presenting OA pathology, and Non Degenerative (ND), used as control class. Each patient underwent both a CT and a MRI acquisition. We used the medical 3D modeling software Materialise MIMICS to segment bones and cartilages from CT and MRI respectively. The considered bones are femur, tibia and patella, while the cartilages are femoral, lateral and medial tibial and patellar. The segmented part were then converted

***Table 1**. ML algorithm scores.*

Algorithm	Validation	Accuracy			Sensitivity			Specificity		
		Mean	±	STD	Mean	±	STD	Mean	±	STD
LR	K-fold (k=5)	0.85	±	0.10	0.87	±	0.11	0.85	±	0.13
	K-fold (k=10)	0.84	±	0.19	0.88	±	0.18	0.82	±	0.24
	LOO	0.81	±	0.40	0.75	±	0.49	0.87	±	0.50
SVM	K-fold (k=5)	0.92	±	0.04	0.89	±	0.09	0.97	±	0.07
	K-fold (k=10)	0.83	±	0.18	0.86	±	0.18	0.83	±	0.31
	LOO	0.83	±	0.38	0.83	±	0.49	0.83	±	0.49

LOO: leave-one-out; LR: Logistic Regression; STD: Standard Deviation

into 3D objects and combined together, in order to have a personalized and realistic three-dimensional representation of the knee compartment. From this processed image and 3D model, we computed: all cartilages volumes, all cartilages surfaces, patella volume and surface, average radiodensity (HU) of all cartilages with relative standard deviations and average bone mineral density (BMD) of all the bones with relative standard

***Table 2**. Feature Importance (top 5 features) for Logistic Regression (k=5).*

Feature	Importance (%)
FemCartVOL	24.22
StdDensTibCartMed	15.90
AvDensTibCartLat	14.29
AvBMDTibia	10.25
AvBMDPatella	10.16

AvBMDPatella: Average BMD of patella bone; AvBMDTibia: Average BMD of tibia bone; AvDensTibCartLat: Average density of lateral tibial cartilage; FemCartVOL: Volume of femoral cartilage; StdDensTibCartMed: Standard Deviation of density distribution of medial tibial cartilage.

deviations. For femur and tibia bones, we only considered the region closed to the cartilage, while patella is acquired in its entirety.[212]
Feature importance was performed to find out how (and whether) each feature affects the prediction of the degeneration of the knee cartilages. Results are shown in the tables. Degenerated cartilages usually present a greater amount of water with respect to healthy ones, because of the tears of the collagen matrix tissue cartilage is composed of.[213] This could explain both the relevance of cartilage volume and density in the presented study: the presence of water leads to a change in density and to a swelling that increases the volume.[214,215]
In conclusion, the presented findings have shown that volumes and densities of cartilages can be relevant to predict cartilage degeneration with good performances results; therefore, these features can be part of a set of new features to look at in case of suspected cases OA or knee and hip related problems.

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It goes without saying that Paolo is Editor of the EJTM Section: “Biomedical Engineering for Translational Myology“ and convinced scientific supporter of the Padova Muscle Days, for which he annually organizes the Session on Clinical Quantitative Muscle Imaging.

He published in EJTM some of his key papers that were not accepted by other journals. Despite this he continued to move forward in his academic career.
Paolo Gargiulo is now full Professor and works at center of Medical Technology Center - Reykjavik University /University Hospital Landspitali. Paolo interests and expertise are mostly in: Medical Image processing, Neuroengineering, 3-D printing and Medical technologies. He developed at Landspitali a 3D-Printing service to support surgical planning with over 200 operation planned with a significant impact on the Icelandic health care system and he currently cooperate with institutions in Italy and UK to establish similar infrastructures. He has been a consultant for MedEl (from 2010 to 2016) for the development of larynx pacemaker.
Since December 2013 Paolo Gargiulo is the director of the Institute of Biomedical and Neural Engineering and the Icelandic center of Neurophysiology and manages the center of Medical Technology at the University Hospital Landspitali/ Reykjavik University. Thanks to domestic cooperation’s with Össur, University of Iceland, Decode and the Icelandic Heart association and the support of infrastructure grants from RANNIS, Paolos lab currently include the following facilities: high density Electroencephalographic system (256-EEG), Postural control platform and Virtual reality system, polyjet 3D printer and multimetric Biosignal platform.

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Chapter 9
Aging Muscle: Studies of Master Athletes and lifelong active seniors

After the strong evidence that it is possible to reverse by hbFES muscle atrophy even in to worst case of long term denervated human muscles, Helmut Kern decided to move his international cohort of collaborators to study "Aging Decay and Related Countermeasures", against my trivial opinion that the topic was over-studied by hundreds of thousand (if not millions, accounting the Indian and Chinese doctors) of Gerontologists, Geriatrics and Rehabilitation Specialists (nothing to say, Sport Medicine and Physical Science Specialists).
Luckely, Helmut ignored my opinion and with the strong supports of Simona Boncompagni, Feliciano Protasi and Giorgio Fanò-Illic he started a new series of studies with the muscle biopsies of himself and of several of his friends with which he had cycled and continued to cycle several times a week since twenty and more years.
Indeed the benefit of an active life-style as an anti-aging measure is accepted without exception by all experts and the general opinion, despite the process of aging decay is inexorable, even in the most active persons, that is, the World Recordmen of the Master Athlete Classes.
Anyhow the stronger support to the expectations of Helmut Kern was presented in a preliminary study of the University of Chieti Group showing that some structural and biochemical characteristics of long-term denervated human muscles could be observed in ordinary healthy old persons.
Wheter those observations were also evidence that some denervation events occur in healthy elderly people will be discussed later on.

9.1 Aging Decay of Master Athletes

I am indebet to Paolo Gava for introducing me in this very interesting topic: the ineludible decay of old people even after a life of physical exercise at the higher possible level.
We were once ready to dinning in my home in 2013 in Padua waiting with a glass of Prosecco for the meals. The chat went to our problems of aging persons. When I said that soon or later we will enter an accelerated phase of our decay, Paolo interrupted me with a excellent observation: this is thrue, he sed, only if a disease will hit you, normal persons steadly (and almost linearly) decay from their 30s to 110 years!
This was the beginning of research activities that included the Group of Helmu Kern, my Vienna Friend and Giovanna Albertin, a colleague of the Human Anatomy Section of the Department of Neuroscience of the University of Padua.

Unfortunately Paolo died two years ago. I can't find better words to acknowledge his brilliant activity as an amateur sportsman, an amateur researcher and a lifelong friend than summarizing below the obituary I, Helmut and Giovanna published in the European

Journal of Translationa Myology: Carraro U, Kern H, Albertin G. Paolo Gava, a professional engineer, who has become a Master athlete, an amateur scientist and a lifelong friend. Eur J Transl Myol. 2021 Nov 5. doi: 10.4081/ejtm.2021.10260. Online ahead of print (*Reprinted with permission)* [160].

Paolo Gava, a professional engineer, who has become a Master athlete, an amateur scientist and a lifelong friend

Paolo Gava (Conegliano, Treviso, Italy September 1, 1946 – Stra, Venezia, Italy, July 19, 2021) was an engineer specialized in sustainable energy resources. He worked in Venice (Italy), Montecarlo (France), The Hague (Netherlands) and London (England), leading research programs well before the current international interest in fight against global warming. Passionate about Tango, Paolo kept himself in shape for many decades by running, pedaling and roller-skating, after years of training as a semi-professional athlete, who competed and winned Italian and European short distance races in the Master classes.

After retirement from the races, Paolo applied his engineering skills to optimize comparisons between the results of the different Categories of Master Athletes, questioning the rules used by Italian and World Master Sports Associations.

Friendly discussing during an after-dinner, he shocked us claiming that, in absence of diseases and trauma (i.e., of Early Aging), **the aging decay is an almost linear process from 30 to 110 years**. Under our friendly pressure he was able to publish his first biomedical article, detailing his mathematical approaches and results, in a 2015 issue of Experimental Aging Research, titled: "*Age-associated power decline from running, jumping and throwing male master world records*".[1]

Unfortunately Paolo died less than two years ago and I find easier to honor his other legacies during his last six years of life, by taking substantial text from the Obituary Giovanna, Helmut and I published in the European Journal of translational Myology,[2] adding further examples of Paolo's scientific studies and of his relationships with senior colleagues and young students of Aging and Sports Curricula.[3-8]

***Fig 1**. Paolo Gava as a Master Athlete in 1989.*

Paolo Gava as a Master Athlete

Figure 1. depicts Paolo when, in his forties, competed in short-run Master classes. He participated to several Italian and European races, winning the 100 meters Italian race in 1988. He also competed in the 400 meters race up to 1991.

Paolo Gava as an amateur scientist

As stated in the Abstract of his 2015 seminal first paper: Gava P, Kern H, Carraro U. Age-associated power decline from running, jumping, and throwing male masters world records. Exp Aging Res. 2015;41(2):115-35. doi: 10.1080/0361073X.2015. 1001648.[1]

Absolute male world records of 16 events were collected along with world records of male Masters categories. Performance was normalized with respect to the absolute record; the *performance of various age groups is consequently represented by a number ranging from 1 (world absolute records) to 0 (null performance). Throwing events are further normalized for the decreasing weight of the implements with the increasing age of the Masters athletes. Male world records of track and field events were taken from the websites of the International Association of Athletics Federation (IAAF) for absolute world records (http://www.iaaf.org/) and the World Masters Athletics (WMA) for Masters world records (http://www.world-masters-athletics.org/).* The main unxpected result was summarized by Paolo stating: "*Human power decline in Masters athletes was analyzed,*

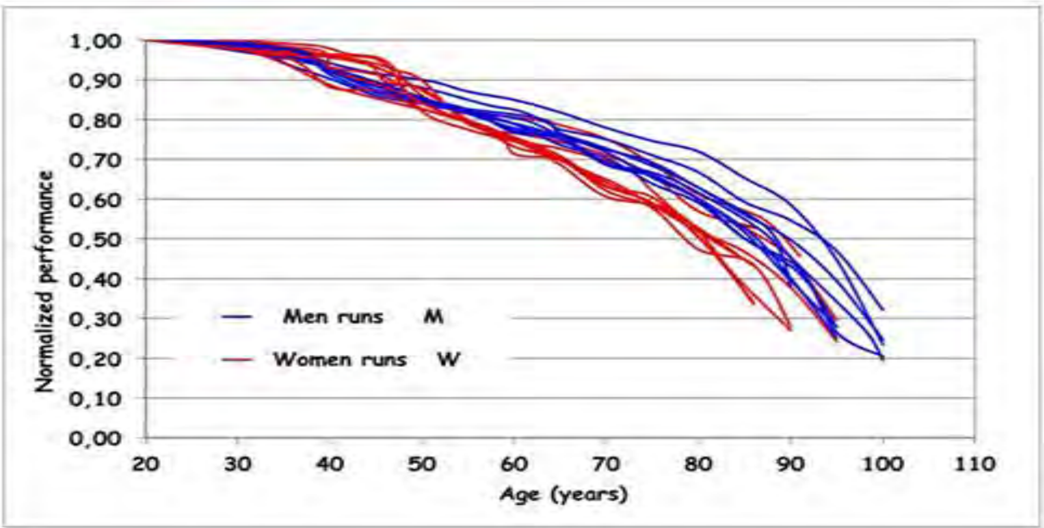

***Fig 2**. Comparison of normalized running parameters of male (blue) and female (red) Masters Athletes. Decline trends of World Records of the running disciplines are very similar. They are very gentle from 30 to 50 years, almost linear from 50 to 70 years and then the decays are progressively steeper.*

adopting a coherent approach based on an extended database. Skeletal muscle power starts declining after the age of 30. The various trend lines point to 0 at the age of 110 years, which is in line with the present human survival age".

Figure 2 shows his latest results, published in 2020, i. e., the comparison of the normalized decay rate for age-dependent performance in the men's and women's world records of Masters categories. Once again, the expected gender differences that characterize absolute values in sports activities did not occurred, something fully unexpected in gender-related sports behaviours. Implications may have important influences on biology, physiopathology and managements of aging.

Paolo Gava and the Venicemarathon

The marathon is the most classic Olympic running event, but in several cities around the world it has become very popular with increasing participation over the past 20 years. The growing popularity of the marathon has led to a significant increase in participants, especially Master athletes.[9-12] There is evidence that long-distance running could provide long term health benefits for older runners.[13] As a former Master Athlete, Paolo had the opportunity to contact the president and organizers of the Venicemarathon and convince them to provide a long series of data with the aim of analyzing them with his dimensionless analysis method. In collaboration with him, some of us have analyzed data of different editions of this famous Italian race that attracts people from all over the world. The race starts outside Venice, usually near Stra (Venice) then runs along the Brenta Riviera into the city, crossing there the canals through floating bridges set up for the race. So far, a student from the Sports Curriculum discussed the Venice Marathon data in his graduation thesis.[6] Furthermore, he analyzed the data to describe the gender distribution of participants in 17 editions from 2003 to 2019, but for age groups and nationality only in the 13 editions from 2007 to 2019. Data published as an EJTM Communication show that there has been a steady increase of female participation, from 12.35% in 2003 to 22.27% in 2019.[14] Using Paolo's approaches we will further compare the performance of different athletes by category, highlighting the trends over the years and the physiological decline regardless of the absolute values of performances.

Paolo Gava as an engineer who loved cycling training and cycle tourism

Paolo as an amateur cyclist loved cycling the Brenta Riviera that connects Padua to Venice along running waters and embankments. Annoyed by the kilometers shared with the risky car traffic, he studied and developed detailed plans to avoid those busy roads, proposing to connect Venice to Padua along a reserved cycle path. He also presented his detailed plans to the municipal and regional authorities, but unfortunately to date without success. We are confident that sooner or later with support of amateur cycling associations and the growing value of cycle tourism, his pioneering project will be implemented following his pioneering path.

Unfortunately, in the last year Paolo has suffered from an incurable disease. He died in his beloved house in Stra (Venice) on the night of July 19th, 2021. Paolo was a sensitive and intellectuall person who loved and was loved by his family, colleagues and friends.

We will never forget Paolo's generosity and exceptional positive approach to life.

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9.2 Lifelong active seniors

9.2.1 Progressive Disorganization of the Excitation–Contraction Coupling Apparatus in Aging Human Skeletal Muscle as Revealed by Electron Microscopy: A Possible Role in the Decline of Muscle Performance

Aging is a physiological process associated with a significant decline in neuromuscular functions, which can affect the quality of life of older people and which cause a dramatic increase in health care costs. Today, where the number of elderly people continues to grow, an understanding of the aging mechanisms is mandatory to prevent secondary disability and maintain autonomy of the elderly. One of the effects of aging is the inevitable reduction in muscle mass. Muscle atrophy that accompanies aging is the result of a variety of changes: loss of motor units, shifting to slower fiber types, impaired Ca2+ homeostasis, mitochondrial alterations, and oxidative stress. Although the impact of aging on skeletal muscle has been extensively studied, the precise mechanisms are not yet fully understood. Among orther mechanisms, it has been proposed that a reduction in the supply of Ca2+ ions available to trigger muscle contraction may be one of the key factors in explaining age-related muscle weakness. Thus an impairment of the mechanisms controlling the release of calcium from internal stores (excitation–contraction [EC] coupling) may contribute to the age-related decline of muscle performance that accompanies aging (EC uncoupling theory). EC coupling in muscle fibers occurs at the junctions between sarcoplasmic reticulum and transverse tubules, in structures called calcium release units (CRUs). In their paper [Boncompagni S, d'Amelio L, Fulle S, Fanò G, Protasi F. Progressive disorganization of the excitation-contraction coupling apparatus in aging human skeletal muscle as revealed by electron microscopy: a possible role in the decline of muscle performance. J Gerontol A Biol Sci Med Sci. 2006 Oct;61(10):995-1008. doi: 10.1093/gerona/61.10.995. PMID: 17077192.] Simona Boncompagni et al. studied the frequency, cellular localization, and ultrastructure of CRUs in human muscle biopsies from male and female participants with ages ranging from 28 to 83 years. Their results show significant alterations in the CRUs' morphology and cellular disposition, and a significant decrease in their frequency between control and aged samples: 24.4/100 lm2 (n ¼ 2) versus 11.6/100 lm2 (n ¼ 7). These data indicate that in aging humans the EC coupling apparatus undergoes a partial disarrangement and a spatial reorganization that could interfere with an efficient delivery of Ca^{2+} ions to the contractile proteins.

9.2 Lifelong active seniors

Age-related muscle reinnervation is promoted by sustained high-intensity exercise

The histologic characteristics of ageing muscle point to denervation as a factor in atrophy, immobility as an accelerant, and regular exercise as a potential defence against the loss of motor units and muscle tissue. Here, we compared the muscle biopsies of sedentary and physically active seniors and discovered that seniors with a long history of intense recreational activity up to the time of the muscle biopsy had the following characteristics: 1) less decrease of muscle strength in comparison to young men (32% loss in physically active seniors vs. 51% loss in sedentary seniors); 2) fewer small angulated (denervated) myofibers; 3) a higher percentage of fiber-type groups (reinnervated muscle fiber)
Old, physically active seniors' muscle biopsies ranged from scant fiber-type groupings to nearly totally changed muscle, indicating that coexpressing myofibers seem to fill in the gaps. The evidence suggests that decades of intense exercise enable the body to adapt to age-related denervation by sparing otherwise lost muscle fibres by selective recruitment to slow motor units. Long-term physical activity appears to promote reinnervation of muscle fibres. These modifications of myofiber size and shape may prevent functional deterioration in late ageing.
The level of recreational training documented in this study is something that the general public may attain, especially if properly encouraged by experts in the field, even though the subjects were not masters athletes. We demonstrate that recreational levels of activity are highly beneficial at improving functional performance and changing the fiber type of muscles in seniors. These levels of activity, in particular, appear to have positive effects on muscle fiber reinnervation, which preserves muscle growth, function, and structure and delays the functional decline and loss of independence that are frequent in late ageing.

Reinnervation of Vastus lateralis is increased significantly in seniors (70-years old) with a lifelong history of high-level exercise

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Abstract
In 2013 we presented results showing that at the histological level lifelong increased physical activity promotes reinnervation of muscle fibers in aging muscles. Indeed, in muscle biopsies from 70-year old men with a lifelong history of high-level physical activity, we observed a considerable increase in fiber-type groupings (F-TG), almost exclusively of the slow type. Slow-type transformation by denervation-reinnervation in senior sportsmen seems to fluctuate from those with scarce fiber-type transformation and groupings to almost fully transformed muscle, going through a process in which isolated fibers co-expressing fast and slow Myosin Heavy Chains (MHCs) seems to fill the gaps. Taken together, our results suggest that, beyond the direct effects of aging on the muscle fibers, changes occurring in skeletal muscle tissue appear to be largely, although not solely, a result of sparse denervation-reinnervation. The lifelong exercise allows the body to adapt to the consequences of the age-related denervation and to preserve muscle structure and function by saving otherwise lost muscle fibers through recruitment to different, mainly slow, motor units. These beneficial effects of high-level life-long exercise on motoneurons, specifically on the slow type motoneurones that are those with higher daily activity, and on muscle fibers, serve to maintain size, structure and function of muscles, delaying the functional decline and loss of independence that are commonly seen in late aging. Several studies of independent reserchers with independent analyses confirmed and cited our 2013 results. Thus, the results we presented in our paper in 2013 seem to have held up rather well.
Trial Registration: ClinicalTrials.gov: NCT01679977
Key Words: Aging; human skeletal muscle; lifelong physical exercise; senior sportsmen; denervation and reinnervation; fiber-type grouping; training.
Eur J Transl Myol 32 (1): 10420, 2022 doi: 10.4081/ejtm.2022.10420

It has long been accepted that histological changes seen in aging muscle suggest that denervation significantly contributes to tissue atrophy.(1,2) Corroborating evidence of a progressive loss of α - motoneurons has been described with aging.(3) Electrophysiological studies have confirmed a decrease in the number of motor units with some increase in their size, suggesting reinnervation events.(4) Further evidence supporting rounds of denervation and reinnervation is based on the observation that in young humans, fiber types appear randomly distributed across the muscle but become increasingly clustered or grouped together with age.(5) Therefore, it has been proposed that apoptosis of motoneurons in the spinal cord (with subsequent incomplete reinnervation of fibers by surviving motoneurons) contributes to the loss of muscle strength and mass that occurs with age.(6) All of these processes are accompanied by a progressive increase in slow muscle fibers, although the literature provides some contradictions (see a recent review).(7) Some of this discrepancy has been dispelled by comparisons of muscle from active and immobile patients: the immobile elderly have a shift toward fast isoform expression, as is common in “unloaded” muscle (e.g., during spaceflight or limb immobilization), whereas muscle wasting is accompanied by a shift toward a fast twitch phenotype.(8) Thus the actual expression pattern of myosin isoforms in the elderly is modulated by complex factors because it depends upon the conflicting influences of both aging and reduced activity tending to shift toward slow and fast isoforms, respectively.(9) To further complicate the situation, conflicting results regarding fast to slow myosin transition arise in endurance training studies using animal models and in clinical trials of humans involving either voluntary exercise or electrical stimulation (directly to muscle or indirectly through nerve stimulation).(8,10-15) Whether these shifts are under neural control or the direct effect of use/disuse on muscle fibers remains to be clarified. In the presents study, we analyzed muscle biopsies harvested from the Vastus lateralis of senior (65 to 79 years) amateur sportsmen (i.e., subjects who routinely practice sport activities usually more than three times a week, up to the time of biopsy). In agreements with some previous studies of master athletes,(16-18) we show that

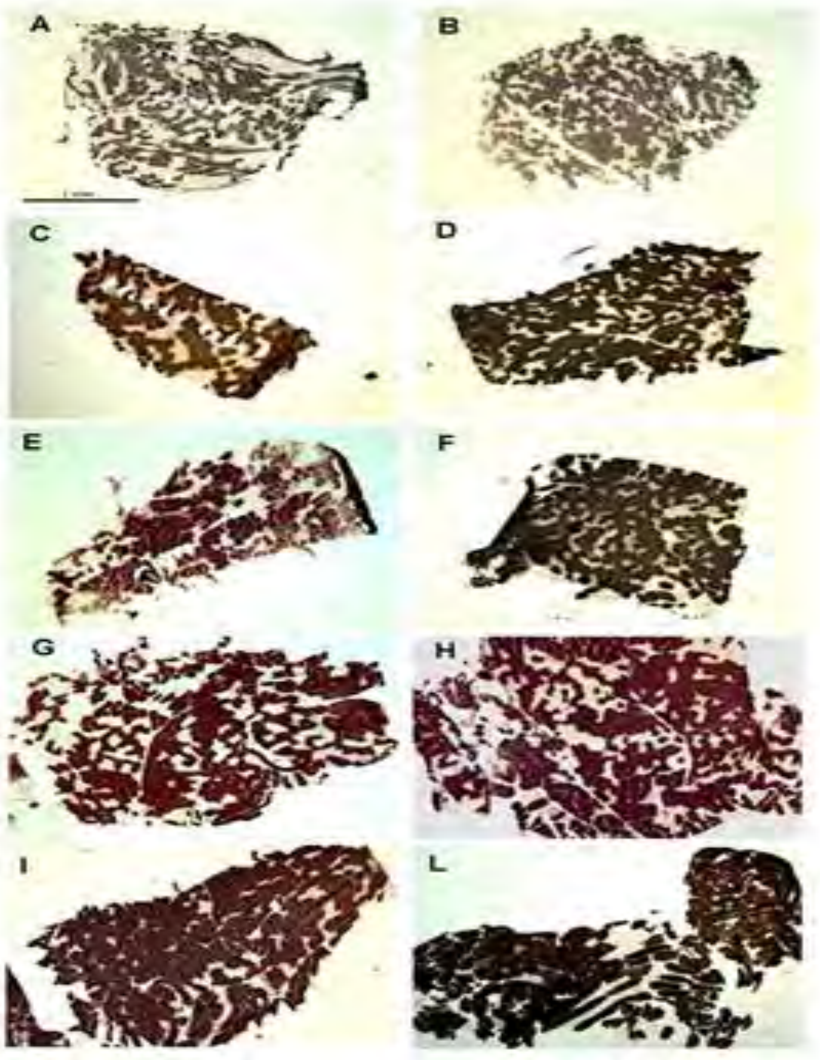

Fig 1. *Fiber type distribution by ATPase staining (pH 4.35) in 70-year sportsmen shows a high occurrence of slow type fibers (dark stained myofibers). Biopsies are ordered from panel A to panel L according to their increasing percentage of slow fibers. The majority has around 70% of slow type, ranging from 51% (panel A), to 92% (panel L). See also Table 3. All panels are at the same magnification, bar = 1 mm.*

lifelong high-level physical activity considerably increases the percentage of slow-type myofibers and the number of muscle fiber-type groupings (F-TG). Slow-type transformation by reinnervation in senior sportsmen appears to be a clinically relevant mechanism because, despite the facts that the biopsies from our subjects vary in the degree to which they have undergone slow-type transformation and that numerous factors can affect fiber type transition, the analyses of our data demonstrate that the senior sportsmen have a significantly greater level of slow type fiber groupings, demonstrating that their muscle has undergone significant reinnervation. Indeed, in recent meetings, we have reported that muscle properties of these senior amateur sportsmen are more similar to those of active young men than to those of sedentary seniors.(19,20) Thus our studies support the concept that lifelong high-level exercise has a beneficial effect on the motoneurons and, through them, on the muscle fibers, resulting in maintainance of muscle size, structure and function, thereby delaying the functional decline and loss of independence that are commonly seen in aging adults.

Materials and Methods

All subjects recruited for the study were volunteers who received detailed information and all signed an informed consent. Approval from the national committee for medical ethics was obtained before study onset (EK08-102-0608). Groups of young men (n=16), seniors with normal life style (sedentary, n=16) and seniors with a lifelong history of high-level recreational sport activities (n=16) were enrolled. All subjects were healthy and declared not to have any specific mobility impairment or disease. Upon enrollment in the study, needle muscle biopsies were harvested through a small skin incision (6 mm) from the right and left Vastus lateralis muscles of each patient and then frozen for light microscopy as described.(12)

Serial cryosections (8 μm) from frozen muscle biopsies were mounted on polysine™ glass slides, air-dried and stained either with Hematoxylin and Eosin (H&E) or using conventional techniques for myofibrillar ATPases to evaluate muscle fiber types.(21) In the latter method, slow-type muscle fibers are dark while the fast-type fibers are lightly stained following preincubation at pH 4.35. The reverse is true after preincubation at pH 9.4. Morphometric analyses of the fiber diameter and of the fiber type distribution were performed on cryosections using Scion Image for Windows version Beta 4.0.2 (2000 Scion Corporation) as previously describe.(12,19,21-24)

ANOVA tests were performed with statistics algorithms of Origin™, OriginLab Corporation, USA. The level of statistical significance was set at $p<0.05$.

Results

From our previous studies on skeletal muscle biopsies of paraplegic patients we know that muscle disuse resulting from decades of years of denervation (after upper motor neuron lesion) induces at most a 50% decrease in size (i.e., from a myofiber diameter of approximately 70 μm to 35 μm),24 while lower motor neuron denervated skeletal muscle (one year. after denervation) shows muscle fibers with a diameter less than 30 μm.(12,22,23,25) Based upon these findings, we are confident in defining those muscle fibers having a diameter smaller than 30 μm as denervated. This interpretation is strengthened by the fact that several small myofibers have angular aspects.(20) In the biopsies analyzed here, small angular muscle fibers have the size and the morphology of denervated muscle fibers and they are more frequent in sedentary septuagenarians than in young men and septuagenarians with a lifelong history of high-level exercise.

Muscle fibers with a diameter less than 30 μm are seldom observed (< 0.5 %) in the muscle biopsies of young men, while biopsies harvested from the sedentary seniors contain the highest percentage (6.9 %) of denervated muscle fibers among the three groups. When muscle fibers with diameters less than 25 μm are counted the percentages decrease by approximately 50% for each group, however, the sedentary seniors still maintain the highst values. ANOVA tests on these data confirm that the higher percentages of small angular fibers in sedentary seniors relative to both young subjects and senior sportsmen are statistically significant. This is not the case when young subjects and senior sportsmen are compared.

Analyses of Fiber-Type Groupings demonstrate that, although not statistically significant, the percentage of fast fiber types is markedly higher in the sedentary seniors than in either the senior sportsmen or the young men. The percentage of slow type fibers, however, is significantly higher in the senior men (both sedentary and sportsmen) than in the younger men. Most interestingly, the percentage of slow-type fibers in the senior sportsmen is signficantly higher than in the sedentary seniors.

Fiber-type grouping is identified on the basis that one myofiber is completely surrounded by fibers of the same phenotype. Because two or more slow type fibers were not always easily distinguished one from another in alkaline-resistant ATPase specimens, we confirmed our fiber border delineations with the less ambiguous method of acid resistant ATPase staining of specimens following preincubation at pH 4.35. In figure 1 some examples of ATPase staining of muscle biopsies harvested from high-level recreational sportsmen are shown, beginning with one which contains a one-to- one proportion of slow-to-fast fibers (as in normal adult muscles) and escalating to a sample in which almost all the muscle biopsy is covered by very large slow F-TG.

Some fast F-TG were present in the biopsies harvested from sedentary seniors: the central fibers characterizing fast F-TG were 3.6% of the total muscle fibers, while those of slow-type were around 0.5%. Even more evident is the fact that, in the biopsies harvested from senior sportsmen, the slow type fibers are grouped in larger areas (mean 8.4 %), almost reaching the 92% in the extreme cases.

It has long been recognized that the histological changes seen in aging muscle suggest that denervation significantly contributes to muscle decay,(1,26,27) and that immobility accelerates the deterioration process,(9) while running activity sustained for decades (as that performed by master athletes) protects against the age-related loss of motor units,(28-30) and, thereby, protects lean muscle mass.(31) However, the degree to which denervation causes muscle fiber transformation and loss of myofibers is an open issue in humans, since reinnervation events may compensate long-term for motor neuron loss in spinal cord and/or axonal abnormalities in peripheral nerves.(4,5,32,33)

Discussion

In the present study we used histochemical ATPase methods to analyze muscle biopsies harvested from septuagenarian sportsmen and compared their relative amount of: i) small angular myofibers (denervated muscle fibers), ii) fast and slow muscle fibers (muscle plasticity), and iii) central muscle fibers of fiber-type clusters (reinnervated muscle fibers) with those in muscle biopsies of sedentary septuagenarians and young men. The main results are: i) biopsies from young men seldom contain denervated and reinnervated muscle fibers or transforming myofibers; ii) biopsies from sedentary seniors contain both denervated and a few reinnervated clustered myofibers of the fast type; and iii) senior sportsmen present with a larger percentage of slow myofibers, up to 90%, which appear clustered in slow F-TG.

Our data suggest that slow-type transformation by reinnervation in senior sportsmen is a clinically relevant mechanism despite the facts: i) that subject biopsies vary from those with scarce fiber-type transformation and groupings to those with almost fully transformed muscles in which isolated fibers co-expressing fast and slow MHCs fill in the gaps (Mosole et al.);(34,35) and ii) there are potential confounding factors such as the sampling of a heterogeneous muscle, individual genetic backgrounds, difference in kind and extent of the high level activities.
Indeed, in recent meetings we reported that the muscle properties of this group of senior sportsmen are more similar to that of active young men than those of sedentary seniors. Specifically, the results indicate that relative to their sedentary cohorts, senior sportsmen have greater muscle maximal isometric force and function and better preserved muscle morphology and ultrastructure.(19,20) Taken together our results suggest that, beyond the direct effects of aging on the muscle fibers, changes occurring in skeletal muscle tissue appear to be largely, although not solely, a result of sparse incremental denervation.
In senior sportsmen the increase in slow clustered fiber percentage is conceivably the result of the positive effect of lifelong physical activity on the motoneuron pool, which has spared the slow motoneurons from age related lesion/death, increasing the chance that peripheral reinnervation occurs due to sprouting of slow axons. Lifelong exercise seems to allow the body to adapt to the consequences of age- related denervation and to preserve muscle structure and function by saving otherwise lost muscle fibers through recruitment to different, mainly slow, motor units. Regular physical activity is a good strategy to attenuate muscle functional decline and ultrastructural abnormalities associated with aging. Certainly other mechanisms contribute to lifelong muscle health, however, our present data support the concept that lifelong high-level exercise has a beneficial effect on the motoneurons, specifically on the slow type motoneurones that are those with higher daily activity, and, through them, on muscle fibers.
In conclusion, the beneficial effects of lifelong high-level exercise on motor neurons and, of course, muscle fibers serve to maintain muscle fiber size, structure and function, delaying functional decline and loss of independence commonly observed in progressive aging. Several confirmatory studies by independent researchers with independent analyzes have been published to date citing our 2013 results.(36) Those results, here presented, seem to have held up quite well.

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Chapter 10
Muscle aging decay: Countermeasures by FES and Full-Body in-Bed Gym

10.1. Countermeasures by FES

The use of functional electrical stimulation as a countermeasure for skeletal muscle atrophy and dysfunction has been a major topic during my research career. Here I will summarize what the Group led by Dr. Helmut Kern, Vienna, Austria, has published over the past decade to prevent and reverse the decay of aging.
We can start reading the editorial that was published in Ejtm 25 (4), 2015, an issue dedicated to Mobility in Elderly. We will then add the Abstract of a 2016 paper, one of Kern's Group's most important publications on this topic. His authorship, which follows immediately, offers a good representation of the network of Researchers and Clinicians who have taken part in a number of related studies.

The EJTM Special "*Mobility in Elderly*"

This Issue of the European Journal of Translational Myology/Basic Applied Myology Vol. 25 (4), 2015 belongs to the series of *EJTM Specials on "Mobility in Elderly".* The issue collects reviews and articles. Their contents were presented at the "MOBIL project completion congress" held at the Wilhelminenspital Wien, Austria the November 8, 2014 to discuss results of the Project: *MOBIL (Mobility in Elderly 2008-2014)*, supported by European Regional Development Fund - Cross Border Cooperation Programme Slovakia – Austria 2007–2013 (Interreg-IVa), project Mobilität im Alter, MOBIL, N_00033 (partners: Ludwig Boltzmann Institute of Electrical Stimulation and Physical Rehabilitation, Austria, Center for Medical Physics and Biomedical Engineering, Medical University of Vienna, Austria, and Faculty of Physical Education and Sports, Comenius University in Bratislava, Slovakia).
Since many years the Ludwig Boltzmann Institute of Electrical Stimulation and Physical Rehabilitation, directed by one of us (Prof. Helmut Kern) is counting on the collaboration not only of Austrian and Slovakian Colleagues but also of several research teams of other European and Transatlantic countries, as it is witnessed by the Authors of the reviews and articles of this *EJTM Special on "Mobility in Elderly"* and the 150 coauthors of the 50 papers published in international journals listed in PUBMED that the Ludwig Boltzmann Institute of Electrical Stimulation and Physical Rehabilitation, Vienna, Austria published from 2004 to 2015.
From 2000 that a fruitful collaboration leaded by Prof. Helmut Kern succeeded to be granted by the EU to respond to the needs of a peculiar group of Spinal Cord Injury persons with complete *Conus* and *Cauda Equina* lesion that completely disconnect the leg muscles from their innervating spinal cord motor neurons. If irreversible, the complete lesion results in degeneration of the muscle tissue, if not counteracted by a purpose-developed Functional Electrical Stimulation (FES) strategy designed and developed in

Vienna starting from the 1990s. The final evidence of the effectiveness of the Vienna rehabilitation strategy, was collected thanks to the support of the EU Commission Shared Cost Project RISE (Contract no. QLG5-CT-2001-02191) leaded by Prof. W. Mayr, Dr. C. Hofer (engineering part), Prof. H. Kern (clinical part) with the expert collaboration of C. Forstner, M. Mödlin, M. Vogelauer, S. Löfler, P. Drewniak, H. Stöhr, C. Rossini, S. Zampieri of the LBI, with the partnership of M. Bijak, D. Rafolt, E. Unger, Center of Biomedical Engineering ad Physics, Vienna, Austria; H. A. Cerrel Bazo, Neuromotor Rehabilitation, Cernusco, Milan, Italy; M. R. Dimitrijevic, Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, TX, USA; G. Exner, Spinal Cord Injury Center, Hamburg, Germany; E. Gallasch, Physiology, Graz, Austria; H. J. Gerner and R. Rupp, Orthopedics, Heidelberg, Germany; W. Girsch, Orthopedics, Speising, Vienna, Austria; T. Helgason, P. Ingvarsson, S. Yngvason, Landspitali-University Hospital, Reykjavik, Iceland; J. Hufgard, M. Obrovsky, Rehabilitation, Klosterneuburg, Austria; H. P. Jonas, Rehabilitation, Bad Häring, Tirol, Austria; S. Lotta, Villanova sull'Arda (PC), Italy; D. Maier and M. Potulski, Murnau, Spinal Cord Injury, Murnau, Germany and the scientific support of Italian Research Teams leaded by U. Carraro, University of Padova and F. Protasi, University of Chieti. Prof. S. Salmons and J. Jarvis, Liverpool University were also engaged in animal experiments, mainly performed in rabbit.

The final report of the EU RISE trial, published in 2010 was the happy-end of a long debated basic and clinical topic, which first studies may be found in the literature of the Nineteenth Century,(1) though it was in the 1940s that the study of events occurring in denervated muscle fibers emerged as a topic distinct from the more clinical relevant studies of nerve regeneration and muscle reinnervation (2-4). During next twenty years, the reports increased in numbers year after year. Finally in 1962 the book edited by Ernest Gutmann summarized previous knowledge from biology to rehabilitation by electrical stimulation and opened the modern era of "*The Denervated Muscle*".(5)

Three pioneers of the modern studies on muscle denervation contributed to the 2014 EJTM Special on "*The Long-Term Denervated Muscle*" and/or lectured at the 2014 Spring PaduaMuscleDays: 1. Bruce M. Carlson, author of several papers with Ernest Gutmann on regeneration of transplanted muscles, opens the EJTM Special with the review "*The biology of long-term denervated skeletal muscle*".(6) He offers to researchers the basic concepts and the results to understand problems and actual or future solutions that continue to nurture Translational Myology; 2. Terje Lømo was the first in 1972 to electrically stimulate denervated rat muscle to test the hypothesis that induced activity modifies muscle properties and indeed he demonstrated that it suppresses one of the hallmarks of muscle fiber denervation, i.e., ACh sensitivity spreading from the synaptic area to the whole sarcolemma.(7) Prior to 1972, it was believed that neurotrophic factors, not related to excitatory impulse transmission, played a major role in spontaneous fibrillation, another functional marker of muscle denervation, whose appearance is inversely related to the length of the degenerating nerve stump. Lømo and co-workers demonstrated, instead, that chronic electrical stimulation of denervated rat muscles caused ACh-sensitivity to disappear from denervated muscles already ACh supersensitive. Further, he showed that the passive electrical properties and the contractile characteristics that distinguish fast and slow fiber types are under the control of the patterns of activity.(8,9) In a report at the First Abano Terme Meeting on Rehabilitation (1985), there reprinted,(10) Lømo et al. defended the hypothesis against the criticisms of authoritative neuroscientists. In his 2014 Commentary, (11) he states "*While reports favoring the existence of neurotrophic factors were numerous before 2000, they have now essentially disappeared from the literature, including original research papers, textbooks and handbooks, which suggest that the hypothesis is no longer arguable. Thus, the results that I presented in our paper in 1985 seem to have held up rather well*".

We hope that the EJTM Special on long term denervated muscle, rising again the interest of clinicians and scientists (Terje Lømo, included) on rehabilitation of denervated muscle may add to his merits the pioneering evidence that activity, anyhow imposed, strongly modulate trophism and characteristics of denervated muscles; 3. Clara Franzini-Armstrong lectured at the 2014Spring PaduaMuscleDays on "*Structure-function relationships in skeletal muscles. Lessons from ultrastructure*".(13) She remembered to us that "*Muscle fibers have a stereotyped organization of contractile myofibrils and membrane systems best defined by their ultrastructure. The sliding filament model (in 1945) established currently accepted principles of most cell motility"*. Her many contributions to the study of the muscle membrane systems and ability to attract young brilliant scientists to electron microscopy are well known and demonstrated also by two speakers of the 2014Spring PaduaMuscleDays, Feliciano Protasi and Simona Boncompagni of Chieti University.(14,15) They have been and are strongly contributing to the success of FES for permanently denervated muscles.(16) We would like to add to Clara's many merits, the pioneering electron microscopy study in the field of muscle denervation: her (first …) 1963 article *"An electron microscope study of denervation atrophy in red and white skeletal muscle fibers". (17)*

Standing on the shoulders of these giants, two of us contributed to the EJTM Special three articles that describe history and results of an application of the concepts and discoveries of Bruce M. Carlson, Terje Lømo and Clara Franzini-Armstrong, namely the Vienna Rehabilitation Strategy by home-based Functional Electrical Stimulation (hbFES) for permanently denervated muscles (Kern H, Carraro U. "*Home-based Functional Electrical Stimulation for long-term denervated human muscle: History, basics, results and perspectives of the Vienna Rehabilitation Strategy")*.(18) Analytical tools and devices, designed and implemented to diagnose, treat and follow up the *Conus Cauda* complete syndrome that paralyze large muscles of human legs are also described.(18) Among the new analytical tools, Gargiulo P, Helgason T, Ramon C, Jónsson H jr, Carraro U describes "*CT and MRI assessment and characterization using segmentation and 3D modeling techniques: applications to muscle, bone and brain*".(19) Recently, a multi-disciplinary team of the Interdepartmental Research Center of Myology of the University of Padua is applying the Vienna principles to the apparently easier cases of peripheral incomplete denervation of limbs. To support the project, denervated muscle fibrillation analyses are revisited in the article of Pond A, Marcante A, Zanato R, Martino L, Stramare R, Vindigni V, Zampieri S, Kern H, Masiero S, Piccione F "*History, mechanisms and clinical value of fibrillation analyses in muscle denervation and reinnervation by Single Fiber Electromiography and Dynamic Echomyography*".(20) Further, in collaboration with his international partners, Dr. Kern is extending the benefits of hbFES to those subjects, which for different reasons suffer the consequences of muscle weakness, from the mild but unrelenting process of aging, (21-24) to the devastating fast progression of muscle cachexia in cancer patients (25,26).

The present 25 (4), 2015 EJTM Special "*Mobility in Elderly*" is dedicated to the results of his recent studies, in particular to the Aging Topic. Supported by European Regional Development Fund - Cross Border Cooperation Programme Slovakia – Austria 2007–2013 (Interreg-Iva), project Mobilität im Alter, MOBIL, N_00033 the International group of Prof. Helmut Kern collaborators have produced remarkable results published in International impacted journals that are listed below. Here we would like to list and thanks all the 150 authors of the 50 papers in international journals listed in PubMed that the Ludwig Boltzmann Institute of Electrical Stimulation and Physical Rehabilitation, Vienna, Austria published from 2004 to 2015. (27-76).

Without their strenuous scientific, translational and clinical work and brilliant intellectual contributions nothing would have been achieved: Abruzzo PM, Adami N, Ambrosio F, Ashley Z, Baraibar MA, Barberi L, Bassetto F, Belia S, Bijak M, Bily W, Biral D, Blaauw B, Boato N, Boncompagni S, Bosco G, Bottinelli R, Burggraf S, Canepari M, Carnio S, Carraro U, Coletto L, Corbianco S, Cvecka J, Danieli-Betto D, Danner SM, De Rossi M, di Tullio S, Dimitrijevic MR, Doria A, Fanó G, Fend M, Ferrero M, Forstner C, Francini F, Franz C, Friguet B, Fruhmann H, Fulle S, Gallasch E, Gargiulo P, Gava P, Germinario E, Ghirardello A, Gobbo V, Grim-Stieger M, Gudmundsdóttir V, Haller M, Hamar D, Haslinger W, Helgason B, Helgason T, Hendling M, Hoellwarth U, Hofer Ch, Hofstoetter US, Ingvarsson P, Jäger H, Jarvis JC, Jernej R, Kaider A, Kern H, Khan MM, Kinz G, Knútsdóttir S, Kovarik J, Krenn M, La Rovere R, Lanmuller H, Lapalombella R, Lenaz G, Li F, Löfler S, Longa E, LoVerso F, Maffei M, Mancinelli R, Mandl T, Marcante A, Marchionni C, Marini M, Masiero S, Mayr W, McKay WB, Merigliano S, Meyerspeer M, Mildner E, Minassian K, Mödlin M, Moser E, Mosole S, Müller L, Musarò A, Nejc S, Nori A, Paolini C, Paternostro-Sluga T, Pelosi L, Persy I, Piccione F, Pietrangelo L, Pietrangelo T, Pinter MM, Podhorska- Okolow M, Pond A, Protasi F, Rafolt D, Rakos M, Rampudda ME, Raschka D, Raschner C, Rattay F, Ravara B, Reichel M, Reischl M, Reynisson PJ, Richter W, Romanello V, Rosker J, Rossini K, Rudolf R, Rupp R, Salmons S, Sandri M, Sarabon N, Sarzo G, Sbardella S, Schils S, Scordari A, Sedliak M, Sgarbi G, Squecco R, Stramare R, Strohhofer M, Sutherland H, Tansey KE, Tirpakova V, Trimmel L, Unger E, Valente M, Vatnsdal B, Vecchiato M, Vindigni V, Vogelauer M, Yngvason S, Zampieri S, Zanato R, Zanin ME.

Finally, the conclusive remarks of Prof. Helmut Kern at the 2014 Vienna MOBIL project completion conference: *I think the presentations were fantastic and they showed that we have done a lot of work in the past 4 years. Every topic here is special and helping our goal: keeping seniors more mobile and prevent them from falling. This may even be the main task for the future. How to train, measure and correlate the outcomes are the most important things. Further, it is very important that when we stimulate we will not avoid the nerve regeneration process. We need precise training procedures. Prof. Hamar's collaborators are the specialists to work this out. We look forward to our future collaboration and our next project (and preparation of a grant proposal) will start immediately after this conference. One thing only I left to say, thanks to all, and in particular to Mike Willand, whose travel from home to Vienna was the longest.*

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Physical exercise in aging human skeletal muscle increases mitochondrial calcium uniporter expression levels and affects mitochondria dynamics

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Age-related sarcopenia is characterized by a progressive loss of muscle mass with decline in specific force, having dramatic consequences on mobility and quality of life in seniors. The etiology of sarcopenia is multifactorial and underlying mechanisms are currently not fully elucidated. Physical exercise is known to have beneficial effects on muscle trophism and force production. Alterations of mitochondrial Ca^{2+} homeostasis regulated by mitochondrial calcium uniporter (MCU) have been recently shown to affect muscle trophism in vivo in mice. To understand the relevance of MCU-dependent mitochondrial Ca^{2+} uptake in aging and to investigate the effect of physical exercise on MCU expression and mitochondria dynamics, we analyzed skeletal muscle biopsies from 70-year-old subjects 9 weeks trained with either neuromuscular electrical stimulation (ES) or leg press. Here, we demonstrate that improved muscle function and structure induced by both trainings are linked to increased protein levels of MCU Ultrastructural analyses by electron microscopy showed remodeling of mitochondrial apparatus in ES-trained muscles that is

consistent with an adaptation to physical exercise, a response likely mediated by an increased expression of mitochondrial fusion protein OPA1. Altogether these results indicate that the ES-dependent physiological effects on skeletal muscle size and force are associated with changes in mitochondrial-related proteins involved in Ca^{2+} homeostasis and mitochondrial shape. These original findings in aging human skeletal muscle confirm the data obtained in mice and propose MCU and mitochondria-related proteins as potential pharmacological targets to counteract age-related muscle loss.
Keywords: Aging skeletal muscle; electrical stimulation; mitochondria Ca2+ uptake.

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10.2. Countermeasures by Full-Body in-Bed Gym

Mobility-impaired persons, either very old or younger but suffering with systemic neuromuscular disorders or chronic organ failures, spend small amounts of time for daily physical activity, contributing to aggravate their poor mobility by resting muscle atrophy. Sooner or later the limitations to their mobility enforce them to bed and to more frequent hospitalizations. We include among these patients at risk those who are negative for the SARS-COV-2 infection, but suffering with COVID-19 pandemic syndrome. Beside managements of psychological symptoms, it is mandatory to offer to the last group physical rehabilitation approaches easy to learn and self-managed at home. Inspired by the proven capability to recover skeletal muscle contractility and strength by home-based volitional exercises and functional electrical stimulation, we suggest also for chronic COVID-19 pandemic syndrome a 10–20 min long daily routine of easy and safe physical exercises that can activate, and recover from weakness, the main 400 skeletal muscles used for every-day mobility activities. Persons can do many of them in bed (Full-Body in-Bed Gym), and hospitalized patients can learn this light training before leaving the hospital. It is, indeed, an extension of well-established cardiovascular-respiratory rehabilitation training performed after heavy surgical interventions. Blood pressure readings, monitored before and after daily routine, demonstrate a transient decrease in peripheral resistance due to increased blood flow to many muscles. Continued regularly, Full-Body in-Bed Gym may help maintaining or recovering independence of frail people, including those suffering with the COVID-19 pandemic syndrome.

Key Words: skeletal muscle weakness; home-based Full-Body in-Bed Gym; older olds; borderline mobility impaired persons; COVID-19 pandemic syndrome.
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There are about 700 named skeletal muscles in the human body, including 400 that are important only for medical specialists. Better known are the roughly 200 skeletal muscles that are serious bone-movers, plus another 100 little muscles of hands, feet, and face. The aim of this report is to convince persons-in-need, and their practitioners, to counteract muscle atrophy-sarcopenia-cachexia, maintaining at their best function and shape of the majority of their body muscles.(1) Geriatric subjects, due to advanced age and/or associated diseases, spend only a short amount of time for daily physical activity. The consequent disuse muscle atrophy contributes to limit their independence ultimately enforcing them to bed and to hospitalization for long periods. Low mobility-related muscle atrophy is associated with neuromuscular weakness, functional limitations, thromboembolism, and high costs.(2-4) All progressive muscle contractile impairments need permanent management. Besides eventual pharmacological treatment, a home-based physical exercise approach is helpful in counteracting muscle atrophy. Awaiting development of implantable devices for muscle stimulation, as effective as pacemakers for cardiac arrhythmias,(5) implantable stimulators for ventilatory supports,(6,7) or cochlear implants for hearing loss,(8,9) education of sedentary patients to perform home physical exercises could be an effective low-cost alternative during and after hospitalization.(10-12)
Cardiovascular and respiratory physical rehabilitation protocols of surgical patients are well established approaches, whose main goal is to reverse muscle

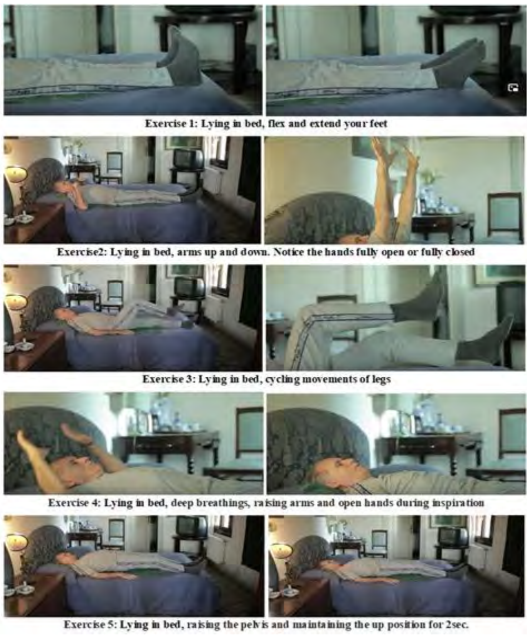

Fig 1. The routine will sequentially contract all major skeletal muscles, alternating exercises that will mobilize arms, legs, spine, neck, diaphragm and accessory ventilation muscles.

Fig 2. The routine will sequentially contract all major skeletal muscles, alternating exercises that will mobilize arms, legs, spine, neck, diaphragm and accessory ventilation muscles.

weakness/atrophy.(13,14) We extended those routines to a daily short (10–20 min) sequence of easy-to-learn and safe volitional physical exercises to be performed in bed (Full-Body in-Bed Gym) to improve muscles and, hence, mobility of impaired persons.

Chronic COVID-19 pandemic syndrome is characterized by the psychological response to the global problem of COVID-19 pandemic, and often by muscle weakness that negatively influences the quality of life of persons for weeks or months before or after resolution of the infection.(15-17) This syndrome is believed to affect up to 10% of the population, because it could already be observed as an acute stress reaction to the spread of the SARS-CoV-2 infection. Certainly it changes in people the ordinary lifestyle for the forced lockdown measures imposed to control the epidemics.(18,19) However, the most severe responses are expected later on after recovering COVID-19. In this case the pandemic syndrome is similar to post-traumatic stress disorders. The problem is that pandemic syndrome will affect the working capacity of population even when economic recovery will be possible and essential. Adequate prophylaxis and management of the syndrome in high-risk groups are important for maintaining global mental health and economy. Beside pharmacological support and psychotherapy in the acute phases, it will be mandatory to prevent and control the mild cases by general prophylactic measures and healthy lifestyle, i.e., by normalization of sleep-wake schedule, by controlling dietary intake of vitamins and microelements and by inducing moderate physical activity. All these measures are important to maintain a good physical condition that improves body adaptive potentials and the immune system. Here our contribution is to convince practitioners,(20-23) and the population at large that Full-Body in-Bed Gym is an option to be taken seriously, despite its apparent minimal requested effort.(10-12)

Fig 3. The routine will sequentially contract all major skeletal muscles, alternating exercises that will mobilize arms, legs, spine, neck, diaphragm and accessory ventilation muscles.

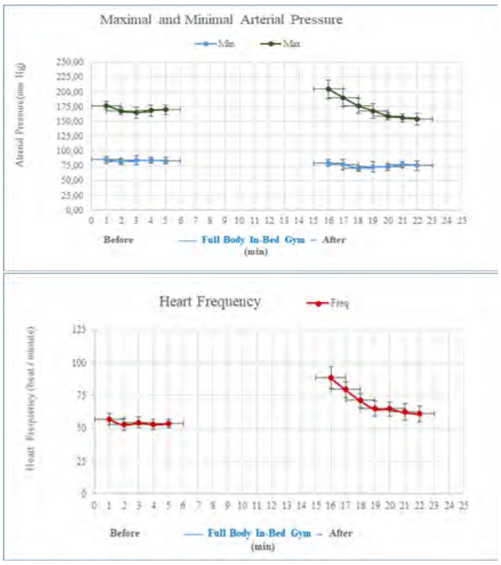

Fig 4. *Arterial pressure and cardiac frequency before and after 10 min. of Full Body in-Bed Gym. Mean +/- SD. Cardiac frequency increased with the maximal, but not the minimal arterial pressure.*

Suggested workout In Figures 1, 2 and 3, the Exercises 1 to 14 show the routine that could be a seasonal warm up also for active persons (typically at early spring after a long winter to recover fitness for demanding physical activities), i. e, those able to make at least 20 consecutive push-ups in 3 min (Figure 3, Exercise 15). After advice of his/her family physician to avoid the very low risks of exercise pain and eventual muscle and joint damage, any sedentary people may start with five repetitions of each exercise. After one-two weeks of training, they may add groups of five additional repetitions, up to 30, every additional week. If compliant, even older olds will progressively increase their muscle strength, if they reach and maintain 15 or 20 daily repetitions. It is safer to start performing the exercises at very slow speed, but when the maximum number of each exercise is reached (15 or 20 repetitions), improving effects will be obtained by speeding up each exercise and thus increasing intensity. The daily routine may last from 10 min (in the beginning) to 30 min (for complete session in accustomed persons). Figures 1, 2 and 3, show the exercises and the captions provide some details. A video describes them dynamically (24): https://www.youtube.com/watch?v=pcHKmxCLYFs&t=336s

If sedentary persons with rest-related muscle weakness, but without major comorbidities, challenge themselves avoiding much stress in a few weeks of Full-Body in-Bed Gym they may increase their muscle strength, fatigue resistance and independence in daily life activities. In particular, cautious Full-Body in-Bed Gym may help patients to recover earlier after hospitalization, decreasing the risk of thromboembolism after surgical interventions, and concurring to reduce eventually present arterial hypertension.(25) Indeed, after a routine that challenge personal fitness, i. e., inducing sweat to the forehead, increasing cardio-respiratory rates and maximal, but not minimal blood pressure, in a few minutes the increases return to the pre-exercise values, as do the minimal arterial pressure.

One example of those behaviors of the cardiovascular ststem to a challenging series of a week of trainings are exemplified in Figure 4. There is strong evidence that peripheral arterial resistance decreased during the series of challenging exercises because blood perfusion is increased by relaxation of the perforating arteries of the main skeletal muscles of the body, i.e. for the systemic functional hyperemia of the main body muscles.(12)

Furthermore, Full-Body in-Bed Gym routine mitigates the bad mood that is usually associated to mobility limitations,(26,27) strengthening confidence of patient in recovering partial or total independence, and it reduces risks of accidental falls. Eventually, during hospitalizations the monitoring of the responses to challenging trainings could include oxygen saturation and many more fitness variables. Furthermore to speed-up positive changes, the trainings could be performed twice a day to improve fatigue resistance and cardio-respiratory reserve.(28,29) Wearable devices are an emerging and cost-effective technology that allows to monitor several biometric data,30 and have been tested in many diseases.(31) It might be interesting to add one of these devices (e.g. smartwatches, fitbands, smartphones, etc.) which could represent a guide for the patient during workout (heart rate monitoring and oxyhemoglobin saturation, reminder to perform exercises every day, stopwatch for timing her/his workout.(32) In any case, during the initial learning period of Full Body In-bed Gym, all seniors, if not hospitalized, must be supervised by at least one trainer, if not a health professional to avoid harmful exercise. These, in fact, are linked to their fitness and, nothing to say, to comorbidities often present in elderly population. If elderly persons cannot, or are reluctant to perform volitional physical rehabilitation protocols, functional electrical stimulation may mimic those exercises and be almost equally effective.(33-41) As detailed in Kern et al., in 2014,36 old persons may be exposed to regular neuromuscular electrical stimulation training. Stimulators for surface electrical stimulation (ES) that are especially suited for elderly people requirements were designed and implemented in Vienna, Austria.42 These constant voltage stimulation devices can be safely applied during home use. Starting two times a week, for a total amount of 24 training sessions (3 × 10 minutes for each session) ES is safe and effective. The subjects ought to be instructed to increase the stimulation intensity until their maximal tolerance is reached. Using this approach a full knee extension is achieved in all subjects. The outcome is a significant increase in muscle strength, associated with an increase of fast muscle fibers, which are the first to respond to ES and are well related to the power of skeletal muscle. ES significantly increased the size of fast type muscle fibers, and the number of Pax7- and NCAM-positive satellite cells. Moreover, analyzed muscle biopsies did not presented signs of muscle damage and/or cellular inflammation.(36,43) Altogether, previous results demonstrated that physical exercise, either voluntary or induced by electrical stimulation, improves the functional performance of skeletal muscles, including those essential for ventilation, a main problem in COVID-19 patients. Indeed, it is worth noting that one of the most successful clinical application of skeletal muscles ES is the ventilatory support to person-in-need by pacing of a conditioned diaphragm in quadriplegia and beyond.(6,7,44-47) In conclusion, it is never too early and it is never too late to increase daily levels of volitional or FES-induced muscle contractions in aging and early-aging syndromes.19-22 Full-Body in-Bed Gym could help patients suffering with mild cases to prevent chronic COVID-19 syndrome and to recover from weakening of skeletal muscles.

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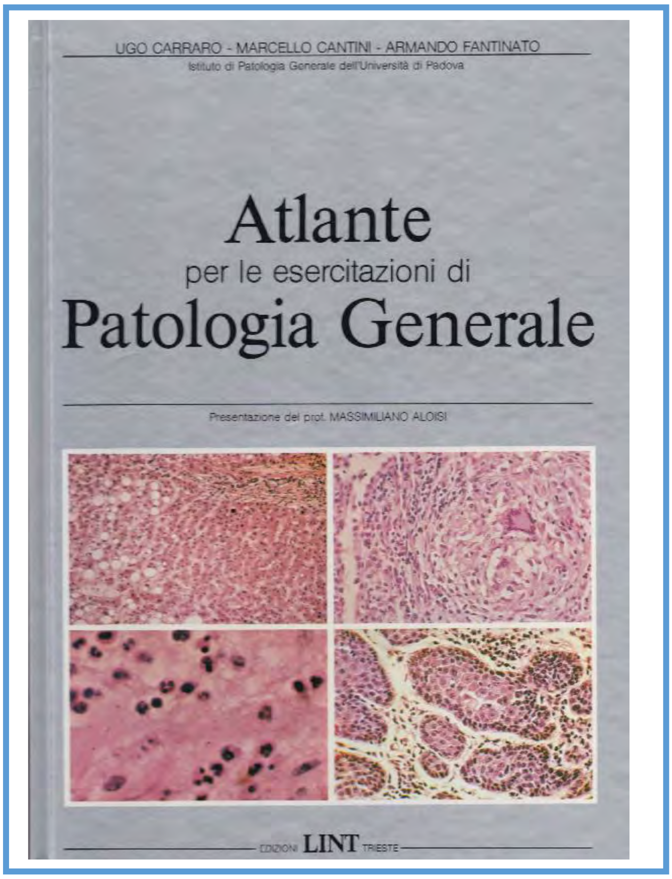
Chapter 11
51 Years of teaching General Pathology and 38 Years of Padua Muscle Days

Chapter 11.1. Fifty-one years of teaching General Pathology

Fascinated by two years of lessons of General Pathology by Prof. Massimiliano Aloisi during the second and third year of the Medicine Course at the University of Padua, I collected personal notes of his “Socratic teaching”. Indeed, he usually spent more time in posing questions and insisting in waiting reply from the audience than lecturing. Furthermore, He spent many weeks in projecting a wonderful collection of microscopic slides of pathological cases, insisting at the beginning of the projection on details that could allow the audience to identify the site of the eventual pathological cases by identifying details not present in normal specimens of the organ. After graduation in Medicine, I had his offer to remain as a voluntary assistant and then paid teaching assistant, in particular to help him, participating as young partner in examinations of students at the end of the General Pathology course. During long morning and afternoon, I had the opportunity to listen thousands of time his questions and the reply of brilliant or poor students. Part of the examination was also based on observation of a microscopic slide of pathology cases.



All those information allowed me, when I became Associate Professor of General Pathology in the Faculty of Medicine of the University of Padua, Italy to write and publish both an “ATLANTE per le “ESERCITAZIONI DI PATOLOGIA GENERALE (Atlas for the practical examinations of pathologic microscopic slides) by Ugo Carraro, Marcello Cantini, Armando Fantinato, Lint, Trieste, Italy and three editions of the book of PRINCIPI DI PATOLOGIA GENERALE (Principles of General Pathology), published by UNIPRESS, Padua, Italy, dedicated to Dentistry and Nursing University Courses.

Together with the short preface written for the Atlas by Prof. Massimiliano Aloisi, an example of a “Socratic” presentation of a pathological sample will follow.

ATLAS FOR PRACTICAL EXERCISES OF GENERAL PATHOLOGY

Curated by Ugo Carraro, Marcello Cantini and Armando Fantinato
2007 Edition, Lint, Trieste, Italy

To Prof. Massimiliano Aloisi hoping that
He recognizes himself if not in the quality
of the realization at least in the inspiring idea.

Forewords

Histopathological iconographies are now overflowing and this could have been a good reason not to add another one. But instead there are valid reasons for this publication intended for second and third year students of the Medicine course. One of these reasons is the goodness of the reproductions, but others are no less important:

a) presenting only elementary and general case studies suitable for students not yet engaged in specific pathological histology, which would create confusion in those who are preparing to understand the first morphological derivatives of human nosology;
b) limiting oneself to even formally common samples, that is, not different or selected with respect to those that should in fact be shown during the exercises, including the defects of the preparations, which too must be, and in fact are, explained;
e) constantly following a logical method of presentation of the various images, at different levels of magnification, in order to avoid that the conclusion of the exercise done under the microscope with the help of this iconography results in only visual memorization, which is the worst vice that a student can face, abdicating his own logical abilities; it is always better to be mistaken for a logical jatus than to guess for a stereotypical memorized image;
d) being completed by someone who is now very familiar with student exercises.

The efforts of the young authors therefore deserves the attention of those who do not claim to have an iconographic treatise on hand, but a guide for the first contacts with a pathology laboratory, a guide not so much for the wealth or peculiarity of the things shown, but for the way they are shown.

MASSIMILIANO ALOISI

ATLAS FOR PRACTICAL EXERCISES OF GENERAL PATHOLOGY
Curated by Ugo Carraro, Marcello Cantini and Armando Fantinato
2007 Edition, Lint, Trieste, Italy

SHEET 8. Rat: Cavity wall, Hematoxylin – Eosin stain

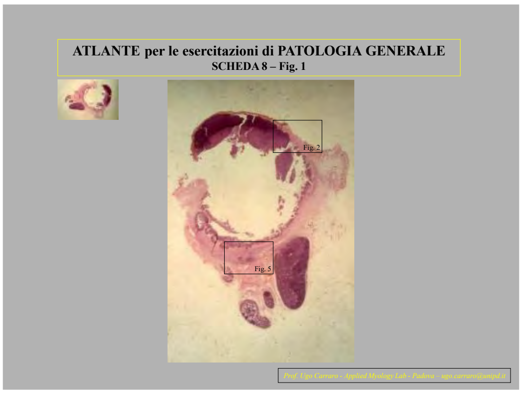


Fig. 8.1 [4 x]

Fig. 8.1 [4x] - The preparation has an annular section, interrupted by an artefact interruption of a cavity wall. Irregular basophilic areas (purple) are present in the lumen and along the inner contour of the cavity. On the external contour of the preparation, the basophilic areas are instead structured and with clearly defined limits. The further observation does not allow to draw conclusions about the exact nature of the piece. We proceed to the systematic examination of the preparation, starting at a small magnification from the outer contour in the upper part of the figure.

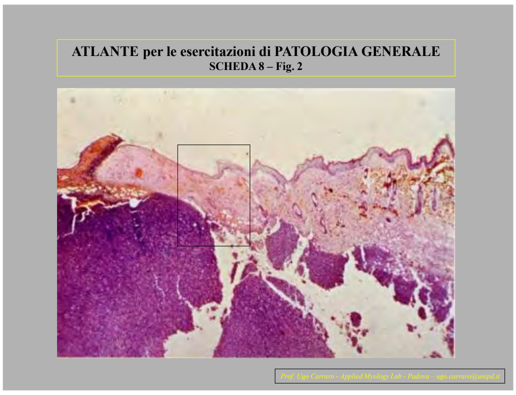


Fig. 8.2 [50 x]

Fig. 8.2 [50 x] - Even at small magnification the external contour of the preparation presents, at least in part, the characteristics of a natural limit. A thin, intensely eosinophilic (orange) border rests on a more basophilic, wavy ribbon, but congruent with the free limit. In the underlying eosinophilic tissue (pink) there are small oval or tubular structures. It is, in fact, the transverse section of the skin, of which the keratinized epidermal lining and some hair bulbs can be recognized in the dermis. The cavity, the intensely basophilic content of which is visible in the lower part of the microscopic field, is therefore located in the subcutaneous. Towards the left half of the figure, the basophilic material is in contact with the dermis, which continues with an intensely eosinophilic material (pink-orange), devoid of nuclei and structures (necrosis).

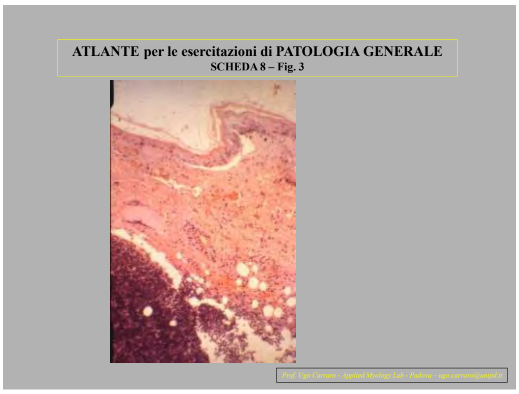


Fig. 8.3 [50 x]

Fig. 8.3 [200 X] - Detail of the previous image in which the cellular nature of the basophilic masses that partially occupy the cavity is recognized. It is also confirmed that the epidermis from right to left becomes hypoplastic (decrease in the number of cell layers) and therefore becomes confused in an eosinophilic material poor in nuclei and devoid of structures (necrosis).

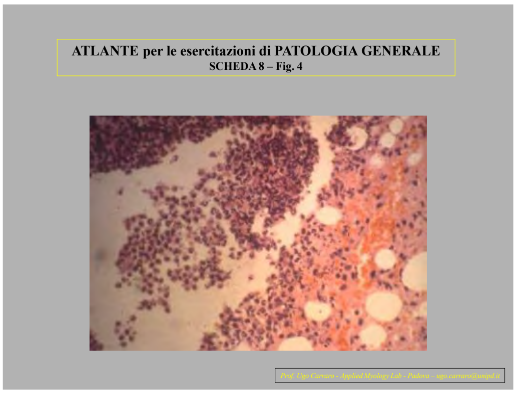


Fig. 8.4 [505 x]

Fig. 8.4 [500 x] - At a further magnification we can recognize the peculiar polymorphism of the multi-lobed and apparently fragmented nuclei of the cells that occupy the cavity: these are neutrophil poly-morphonuclear granulocytes (neutrophil leukocytes). A purulent exudate, of which the cellular part can be seen here (the liquid component was lost during the preparation of the preparation), then fills the cavity present in the subcutaneous. Taking into account the site, the size and the absence of a natural lining (epithelial or endothelial), it is concluded that the lumen is the site of collection or drainage (fistula) of the purulent material of an abscess. The round, optically empty areas are subcutaneous fat cells. The vessels are instead filled with red blood cells (the various orange round areas). Note that numerous leukocytes are also present in the subcutaneous loose connective tissue.

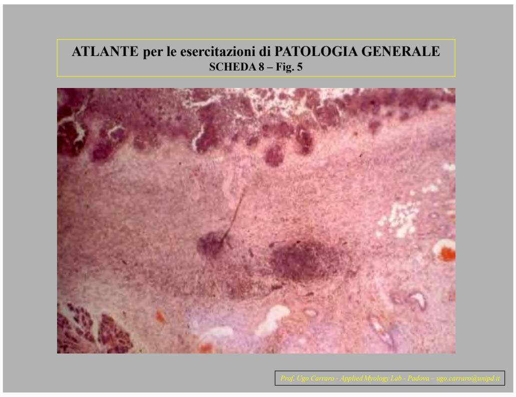


Fig. 8.5 [500 x]

Fig. 8.5 [500 x] - At small magnification the wall of the abscess towards the deep subcutaneous planes. Above, the abscess cavity limited by purulent exudate. Below the connective tissue infiltrated by cell nuclei. On the right a group of fat cells, below numerous vessels with well-defined walls (arterioles) or which are confused with the surrounding connective tissue (venules). Lower left part of a gland. The line in the center of the photo field is a fold that formed during the preparation of the slide: it is a common artifact. Note the sinuosity and the infiltrating trend of the pus that digs new paths in the periascessual connective tissue.

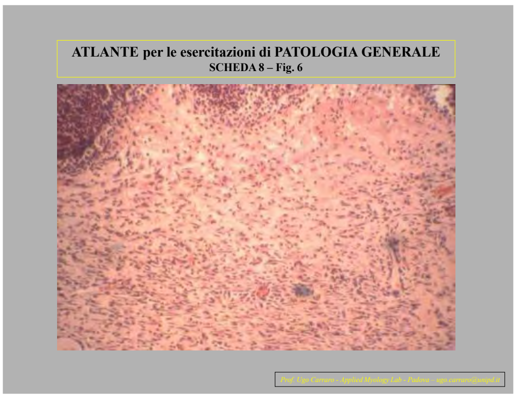


Fig. 8.6 [200 X]

Fig. 8.6 [200 X] and Fig. 8.7 [200 X] At higher magnification the various layers of the cavity wall can be better identified: a) purulent exudate also present in the cavity; b) the thin layer of eosinophilic fibrillar material (the fibrinous component of the exudate); e) the mesenchymal connective tissue infiltrated with cells and rich in vessels, mainly capillaries. This layer is the granulation tissue that in processes like

Fig. 8.7 [500 x]

abscess have been in place for some time to repair the necrotic lesion. Tissue with lytic and necrotizing activity of purulent exudate. A competitive process is thus established between extension and repair of the lesions whose outcome is determined from time to time by the influence of many factors related both to the host and to the cause of the purulent inflammation.

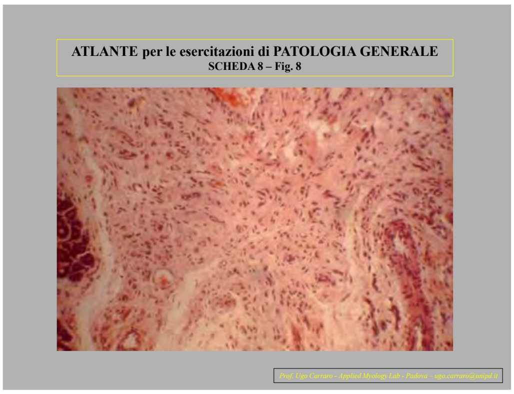


Fig. 8.8 [200x]

Fig. 8.8 [200x] - At lower magnification the granulation tissue in an area a little further away from the lumen of the abscess cavity. The newly formed connective tissue is still very rich in capillaries, but less infiltrated by inflammatory cells. The further evolution will be towards fibrosis due to the accumulation of collagen fibers and the disappearance of the vessels. Bottom right, a tangentially dissected arteriole at the level of smooth muscle; since the lumen is highlighted at both ends, the arteriole must have described a broad curve.. On the left we can see the round lumens of two vessels of the same caliber as the arteriole: the upper one, whose wall is formed by a single layer of endothelial cells, is a venule. On the left, the annular or tubular sections lined with epithelium, mixed with small epithelial areas, are the excretory tubules of an acinar gland. The size, structure and proximity to the skin (see Figures 8.1 and 8.5) suggest that it is a salivary gland. The abscess cavity was therefore located in the anterior cervical region.

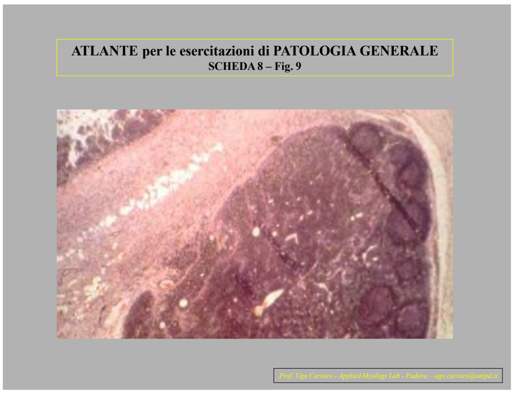


Fig. 8.9 [50 x]

Fig. 8.9 [50x] - At small magnification the ellipsoidal basophilic area in the subcutaneous. Above left, the lumen of the abscess cavity. On the right, the adipose tissue that represents the artificial limit of the piece (see figure 8.1). The large basophilic area that occupies the central part of the figure is a hyperplastic lymphoganglion. The slightly lighter round areas lined up along the right side are hyperplastic follicles (germinal centers). This is therefore the cortical portion of the lymphoganglion. The medullary part is instead to the left of the basophilic area. The image of a lymph node, not normal, but less hyperplastic, is obtained by comparing in figure 8.1 the area we are examining with that of the smaller kidney located in the lower center. In this the clear lights of the sinuses of the medulla are clearly visible.

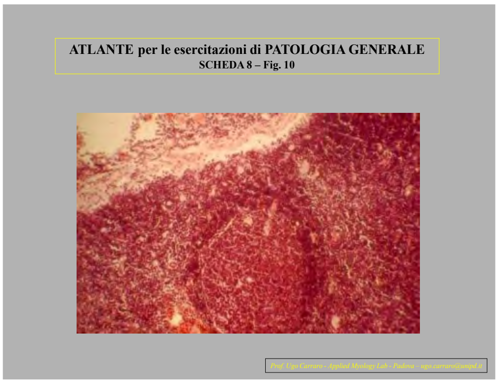


Fig. 8.10 [200 x]

Fig. 8.10 [200 X] - Detail of the lymphoganglion cortex. From right to left: the subcutaneous connective; the thin rectilinear strip richest in cells is the fibrous capsule of the lymphoganglion; the thin lighter strip parallel to the previous one is the cortical sinus, into which the lymph coming from the tributary lymphatics draining the surrounding tissue enters; finally, in the cortex three hyperplastic lymphatic nodules (follicles). At this magnification, while recognizing the cellular nature of the whole basophilic area, the nature of the cells that constitute it cannot be defined. The crowding of the cells and the supracellular structures, however, allow it to be recognized as lymphoid tissue.

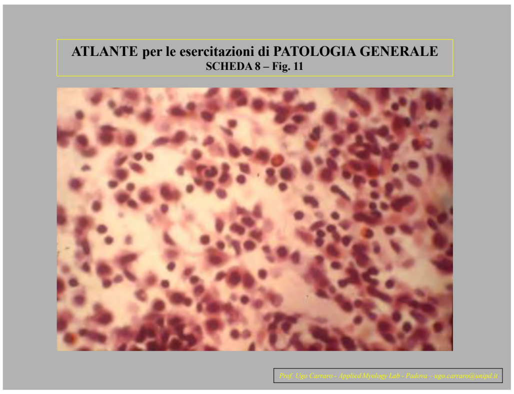
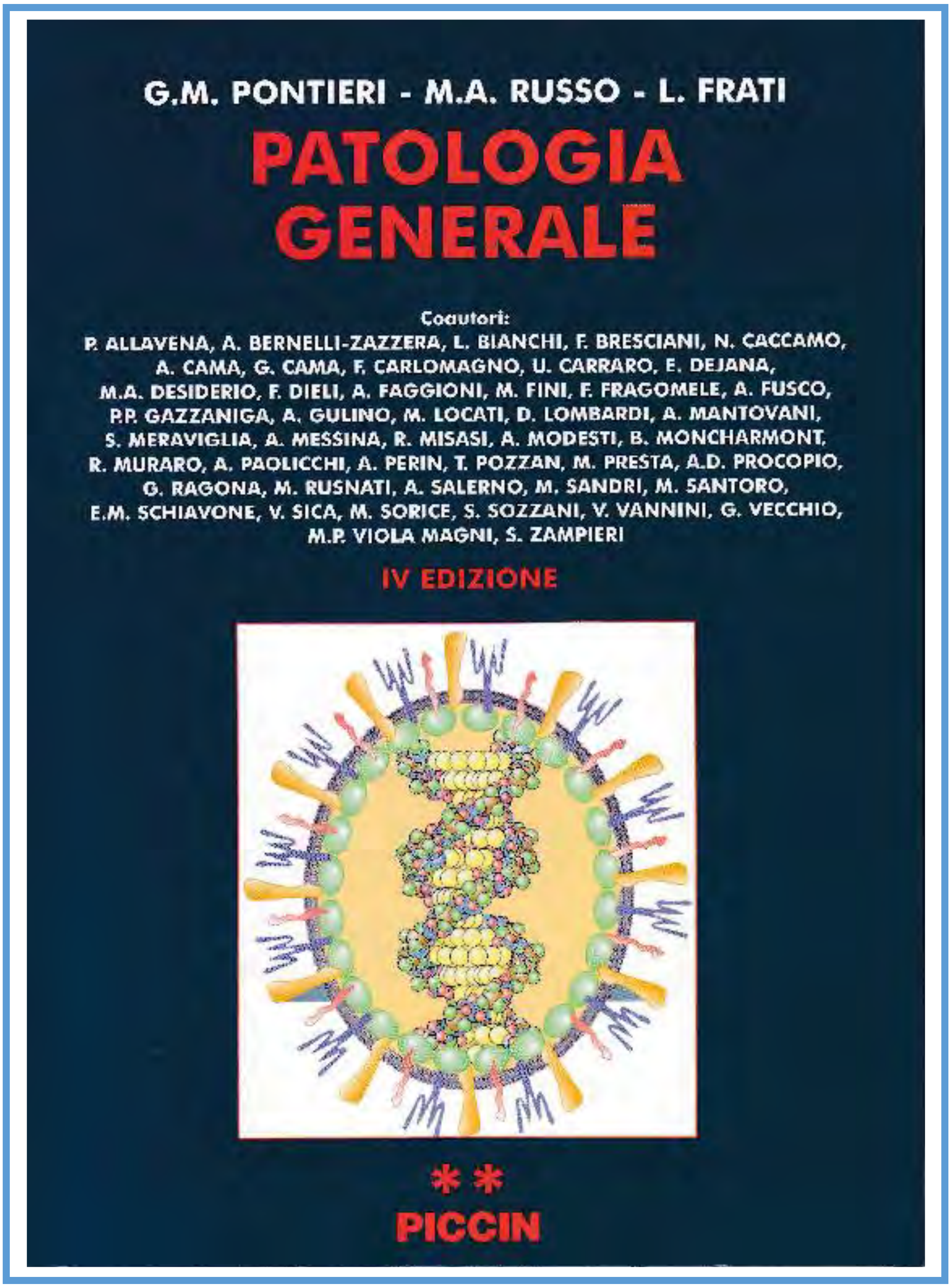


Fig. 8.11 [800 x]

Fig. 8.11 [800 x] - Very enlarged detail of the lymphoganglion medulla. The lymphatic sinuses contain a much higher number of cells than normal, in particular polymorphonuclear neutrophils (the small cells recognizable by the multilobed shape of the nucleus, see again Figure 8.4). Polymorphonuclear cells are present for lymphatic drainage of exudate. There are also numerous lymphocytes, cells that are normally found in the lymph and that make up a large part of the lymphatic tissue with fibroblasts and macrophages that limit the paranasal sinuses. Lymphocytes are small cells with very little cytoplasm with a round nucleus, intensely basophilic. The cells with a larger oval nucleus and abundant cytoplasm are instead macrophages (partly floating in the fluid of the sinuses, partly fixed to form the walls - entothelial cells with macrofacial activity of the reticulo-endothelial system). The inflammation affecting the tributary tissue of the lymphatic gland then induces the activation of both the immune system (cortical lymphocytes) and the phagocytic cells of the sinuses, causing hyperplasia of the lymphogagus as lymphoid tissue.

In conclusion, the slide n. 8 presents a paradigmatic case of "chronic purulent exudative inflammation", characterized by: i) cellular exudative phenomena causing colliquative necrosis of the tissue; ii) reparative reactions of connective tissue (granulation tissue) and iii) non-specific and specific immune reactions of the lymphatic system. These concurrent processes, identifiable in different parts of the organ involved (the skin), allow us to conclude that a chronic abscess is in progress that is not susceptible to spontaneous healing.



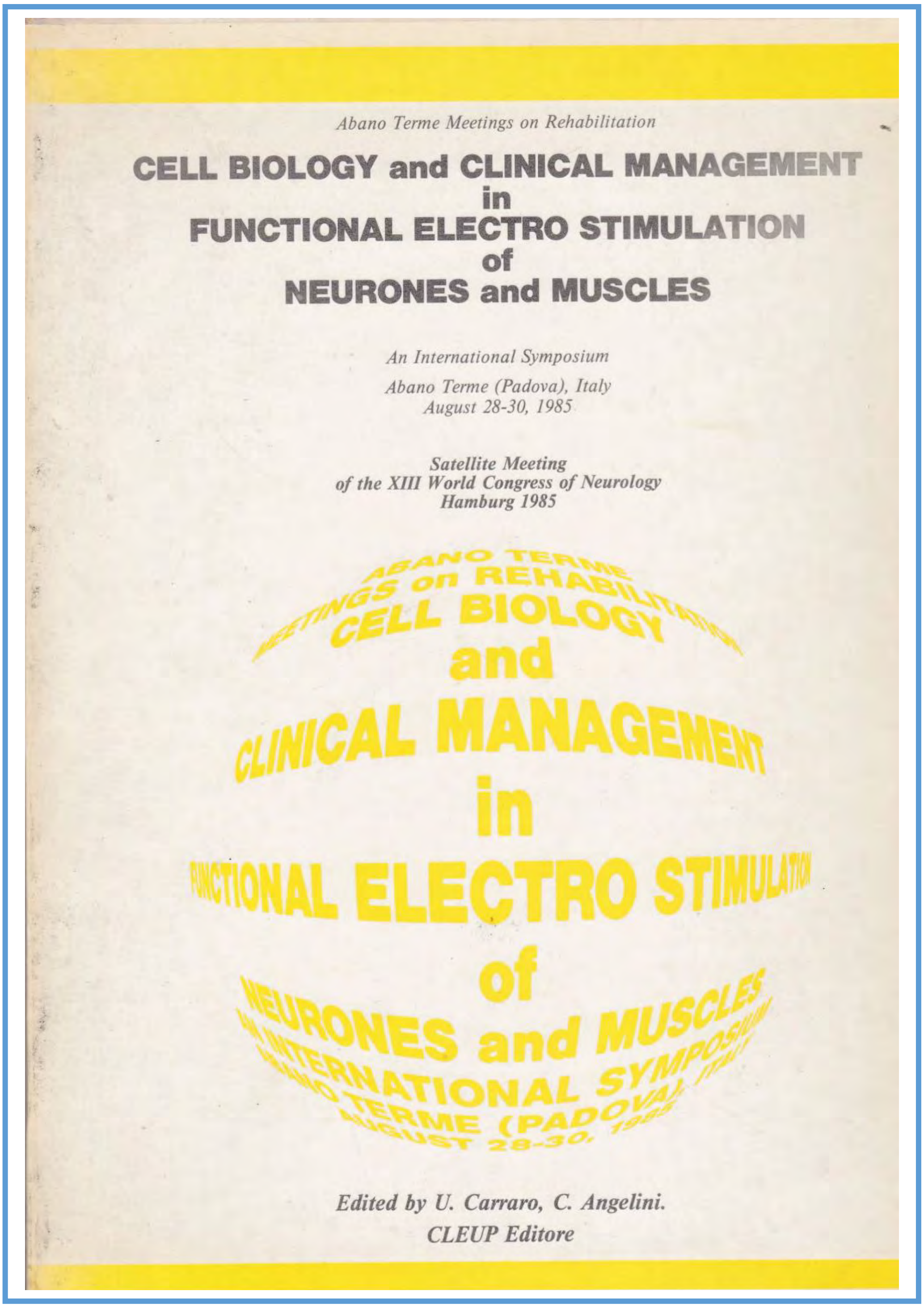
More recentely, I published the Chapter 60 on Muscle Pathology in an Italian Text book for students of Medicine, together with Marco Sandri and Sandra Zampieri.

Capitolo 60. PATOLOGIA GENERALE DEL MUSCOLO SCHELETRICO. Carraro U, Sandri M, Zampieri S. Tomo II PATOLOGIA GENERALE. G.M. Pontieri, M.A. Russo, L. Frati, Eds. IV Edizione, 2010. Piccin, Padua, Italy ISBN 978-88-299-2036-5. Pag. 1523-1542.

Chapter 11.2. Thirty-eight years of Abano Terme Meetings on Rehabilitation, Padua Muscle Days (PMD) and Padua Days on Muscle and Mobility Medicine (PDM3)

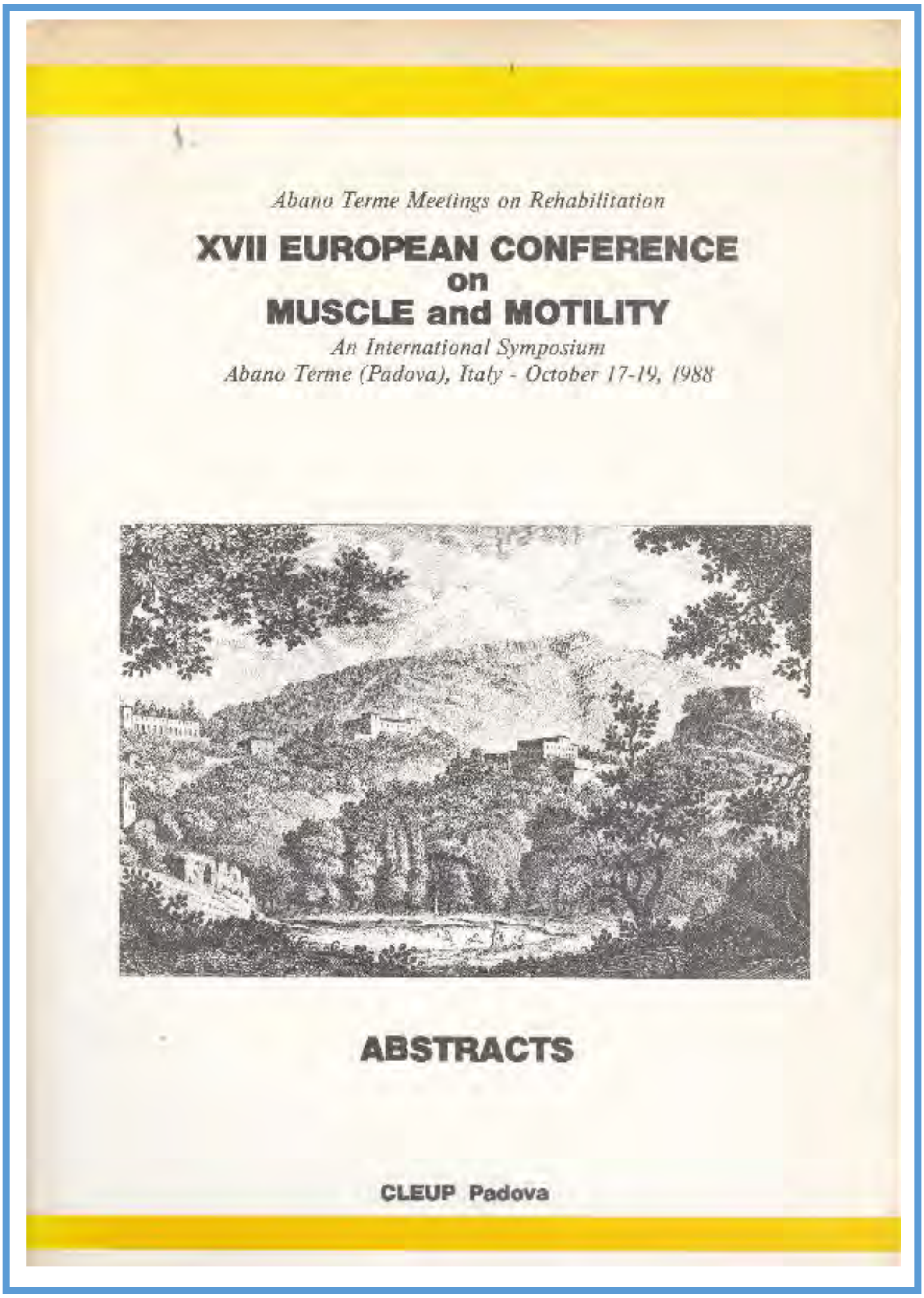
As a very young researcher mainly interested in Myology, I had the opportunity to partecipate to the second Meeting of a new establisched "European Muscle Club (EMC)," organized in 1973 in Lenzburg, Switzerland by Marcus Schaub. Marcus Schuab was leading the EMC with the help of a Committee of 6 to 8 members from 1971 up to 1995 with the help of an informal Committee Board that was preferably composed of club members who had organized former conferences.
The "Journal of Muscle Research and Cell Motility" (JMRCM) founded by Richard Tregear and Chris Ashley (Oxford) started in 1980. From then onward the abstracts of the yearly EMC conferences were officially published in the JMRCM. With that the "Club" gained some wider international visibility and started to attract participants from all over the world. By that time, the term "Muscle Club" became a little ill-reputed; Marcus Schaub was repeatedly asked by the bank with EMC account what kind of body building club He was managing. Actually, at the EMC 1985 in Ulm (Germany) the organizer Reinhardt Rüdel invited a group of professional body builders (men and women) who presented a breathtaking performance in applied myology. Perhaps inspired by this fascinating show, a few years later, in 1991, Ugo Carraro (Padova, Italy) started a new muscle journal appropriately called "Basic and Applied Myology" (BAM). With its 20th volume appearing in 2010 BAM changed its name to "European Journal of Translational Myology" and continues to publish original research articles under "Basic Applied Myology" and expert reviews in a second line called "Myology Reviews" (for details see, http://www.bio.unipd.it/bam/bam.html). As times changed, it got more and more difficult to raise financial support from National Science Foundations and other grant-giving organizations for the "Muscle Club Meetings". Finally, 1988 at the meeting organized by Ugo Carraro (Padova) in Abano Terme the name "European Society for Muscle Research" (ESMR) was adopted. The term EMC was kept with its logo as label for the yearly muscle conferences. Ugo Carraro was an early enthusiast of the "Club" and the ESMR. As a young muscle researcher (working with Alfredo Margreth in Padova) he attended most of the EMC meetings beginning with Lenzburg in Switzerland 1973 and served for well over 20 years on the Committee Board.

After those information taken from an editorial that Marcus Schaub wrote in 2010 for the European Journal of Translational Myology - Myology Reviews 20 (3): 77-82, 2010, I may do not need to recapitulate my role in ECM, but I may add a few words to explain my passion to organize Muscle Meetings in Padua under the patronage of the University of Padua.
I started my hobby in 1985 organizing with the help of Corrado Angelini, a neurologist of the Padua University, the First Abano Terme Meetings on Rehabilitation: CELL BIOLOGY and CLINICAL MANAGEMENT in FUNCTIONAL ELECTRO STIMULATION of NEURONES and MUSCLES, an International Symposium held in Abano Terme (Padova), Itaty - August 28-30, 1985 as a Satellite Meeting of the XIII Wortd Congress of Neurology, Hamburg 1985.
The Proceedings were printed by CLEUP, a publisher related to the University of Padua, Italy (see following figure).



After that experience I accepted the proposal of Marcus Schaub to organize in Abano Terme the XIV ECM Meeting, held from 17 to 19 October, 1988. The Meeting was a successful even with young and old Myologists from all over the world. Once again the Collection of the Abstracts was printed by CLEUP, Padova, Italy

Under pressure of a few friends, three years later I organized the “**Basic and Applied Myology: Perspectives for the 90's”** a Conference held in Abano Terme, Padova (Italy), May 30 - June 1, 1991. This time the Proceedings were published by UNIPRESS Padova, (Italy), a new publisher organized by Prof. Gialuigi Borgato, a philologist of the University of Padua, Italy


Abano Terme Meetings on Rehabilitation

XVII EUROPEAN CONFERENCE on MUSCLE and MOTILITY

An International Symposium

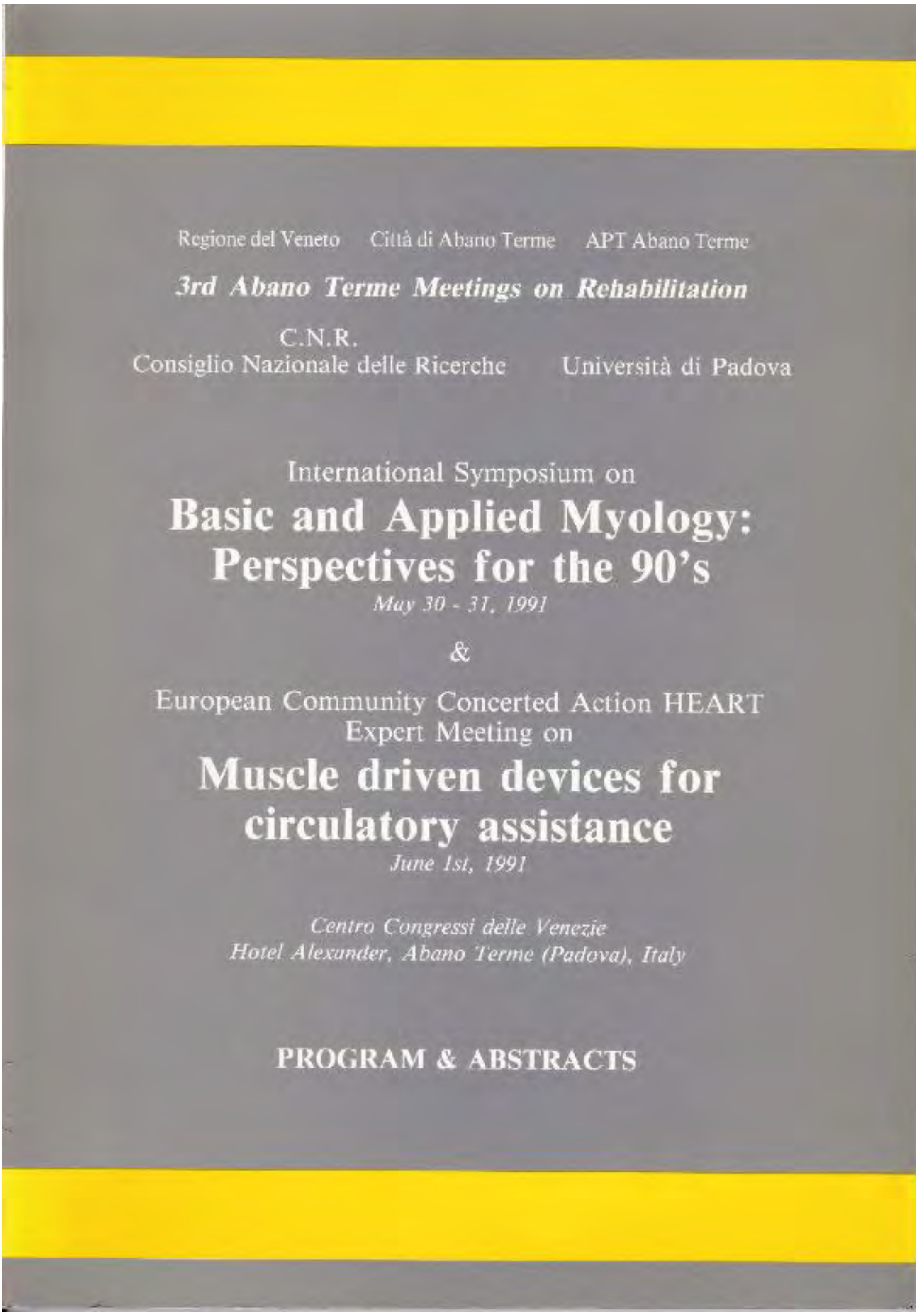
Abano Terme (Padova), Italy - October 17-19, 1988

ABSTRACTS

CLEUP Padova

In 1991 I also started the publication with UNIPRESS, Padova (Italy) of a new Journal: Basic and Applied Myology (BAM). For details, see Chapter 13.3.

A few years later I had the opportunity to invite Prof. Leonardo Vecchiet for a seminar in Padua. Lenardo was a professor of sports medicine and rehabilitation, who had led the medical supervision of the Italian football team that had won the 1982 World title in Spain. It was for that occasion that for the first time I used in the announcement of his seminar the formula: Padua Muscle Day (PMD).
Then when a few international Colleagues asked me to meet in the Thermae of Euganean



Hills to discuss future projects and to present preliminary results, I used for those workshops the Title: Padua Muscle Days (PMD). The meetings followed almost yearly (and often twice a year) up to 2018, when the conference name was modified to Padua Days of Muscle and Mobility Medicine (PDM3) to stress the extended interests of the European Journal of Translational Myology and Mobility Medicine (EJTM3), the new name of BAM from 2010 published by PAGEpress Scientific Publications, via A. Cavagna Sangiuliani, 5 - 27100 Pavia, Italy.

Due to the COVID-2019 pandemic the 2020 and the 2021 Padua Days of Muscle and Mobility Medidcine were organized as virtual meetings.

Luckely in 2022 we were able to held the Padua Days of Muscle and Mobility Medicine on-site from March 30 to April 3, 2022 in Padua and theThermae of Euganean Hills (Padua), Italy (2022 On-Site PDM3).

For a few days it was the first International Conference held on-site in the world, after massive vaccinations in Italy and beyond relaxed restrictions due to the COVID-2019. The 2022 On-Site PDM3 was followed a few days later by the 2022 On-Site “Experimental Biology Meeiting” held in Philadelphia, USA, April 2-5, 2022.

To provide evidence of the continuous success of the Padua Muscle Days / Padua Days of Muscle and Mobility Medicine three Programs will follow:

1. **Basic and Applied Myology: Perspectives for the 90's** published by UNIPRESS Padova, Via C. Battisti 231, I-35121 Padova (Italy), Proceedings of a Conference held in Abano Terme, Padova (Italy), May 30 - June 1, 1991;
2. **BAM’2000 Meeting - Basics&Applications of Muscle Plasticity**: Foundations of Muscle Plasticity – Cell&Gene Muscle Therapy – Dynamic Cardiomyoplasty & Cardiac-Bio-Assists, Abano Terme, Padova (Italy), June 11-13, 2000;
3. **2022 On-site Padua Days on Muscle and Mobility Medicine**: *2022 Padua Days on Muscle & Mobility Medicine Hosts the University of Florida Myology Institute and Wellstone Center, Thermae of Euganean Hills and Padua (Italy), March 30 - April 3.*

Basic and Applied Myology: Perspectives for the 90's

published by UNIPRESS Padova, Via C. Battisti 231, I-35121 Padova (Italy)

Proceedings of a Conference held in Abano Terme, Padova (Italy), May 30 - June 1, 1991.
Edited by Ugo Carraro and Stanley Salmons

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2003. Left: Pier A. Grandjean of Bakken Research Center, Maastricht, The Netherlands; Right: Ugo Carraro

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Mechanical power and isomyosins in transplanted soleus muscle of mice. *P. Moens,* G. *Beckers-Bleukx,* G. *Marechal*

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Skeletal muscle ventricles in circulation: 24-month update. *R. Ruggiero, D.R. Anderson, H. Niinami, A. Pochettino, T.L. Hooper, R.H. Hammond, S. Lavine, A.D. Spanta,L. Huiping and L.W. Stephenson*

Skeletal muscle ventricles: bioenergetic and biomechanical considerations. *A. Pochettino, T.L. Hooper, H. Lu and L.W. Stephenson*

A cardiac assist device which takes advantage of the power output of conditioned skeletal muscle. G. *Arpesella, P. Mikus, M. Cirillo, M. Parlapiano, L. Gianessi, A. Pierangeli, L. Miracoli, B. Mambrito,* C. *Rizzi,* C. *Catani, and U. Carraro*

BAM'2000
Basics & Applications of Muscle Plasticity

Foundations of Muscle Plasticity - Cell&Gene Muscle Therapy - Dynamic Cardiomyoplasty&Cardiac-Bio-Assists

Abano Terme, Padova (Italy), June 11-13, 2000

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Aim

Reactions of normal muscles to unusual requests and of diseased muscles to normal demands

BAM'2000 Abstracts and Manuscripts Publication

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PROGRAM

June 10, 2000 (Saturday)

Morning: Tour to Venice.

Late Afternoon: Opening Ceremony and BAM'2000 Gala Dinner at the Locanda Cipriani in the beautiful historical island of Torcello, Venice Laguna.

June 11, 2000 (Sunday)

9.00 - 13.30 Foundations of Muscle Plasticity (I) (II)

9.00 – 11.00 Foundations of Muscle Plasticity*: (I) Signalling Pathways*

Chairpersons: H Blau, S Schiaffino

9.00 – R Sanders Williams, University of Texas South-western Medical Center, Dallas, TX, USA*: Calcineurin-dependent signalling events in muscle plasticity*

9.40 – S Hughes, The Randall Institute, King's College, London: *Hedgehog and the control of muscle cell diversification*

10.10 – S Schiaffino, Department of Biomedical Sciences and C.N.R. Unit for Muscle Biology and Physiopathology, University of Padova, Italy: *Nerve Activity-dependent Muscle Gene Regulation: A New Role for RAS*

10.40 - RW Tsika, University of Missouri-Columbia: *Muscle-specific transcription of beta-myosin heavy chain transgene requires A/T-rich and NF-AT elements*
11.00 - Coffee Break
11.30 - 13.00 Foundations of Muscle Plasticity: *(II) The Muscle Transcriptome*
Chairpersons: S Williams, S Schiaffino
11.30 - TA Prolla, University of Winsconsin, Madison, WI, Usa: *The Transcriptional Profile of the Aging Process in Skeletal and Cardiac Muscle*
12.10 - G Lanfranchi, Department of Biology, University of Padova, Italy: *Gene expression profiling of human skeletal muscle during ageing and differentiation using an arrayed collection of 3'-end muscle cDNAs*
12.30 - G Valle, Department of Biology, University of Padova, Italy: *A data base of transcripts expressed in human skeletal* muscle
13.00 - 14.30 Lunch and Posters
14.30 - 18.00 Apoptosis of Sarcomeric Muscles (Myocardium and Skeletal Muscle)
14.30 - 16.00 *Apoptosis of Sarcomeric Muscles (I)*
Chairpersons: RA Gottlieb and U Carraro
14.30 - RA Gottlieb, Division of Hematology, Department of Molecular & Experimental Medicine, The Scripps Research Institute, La Jolla, CA, USA: *Mitochondria:The Ignition Chamber for Apoptosis*
15.00 - R Sabbadini, Heart Institute and Department of Biology, San Diego State University, San Diego California, USA: *G Protein-coupled receptors, calcium deregulation and apoptosis in the heart*
15.30 - P Bernardi, Department of Biomedical Sciences, University of Padova, Italy*: Mitochondria and muscle cell death*
16.00 - G Vescovo, Division of Internal Medicine, Adria General Hospital, Ro, Italy: *Skeletal muscle apoptosis: a determinant of muscle atrophy in CHF?*
16.30 - Coffee Break
17.00 - 18.00 *Apoptosis of Sarcomeric Muscles (II)*
Chairpersons: R Sabbadini and G Vescovo
17.00 - M Sandri, Department of Biomedical Sciences and C.N.R. Unit for Muscle Biology and Physiopathology, University of Padova, Italy: *Apoptosis and muscular dystrophies*
17.15 - R Matsuda, Department of Life Sciences, University of Tokyo, Japan: *Dystrophic processes can be separated into two distinct stages in mdx skeletal muscle*
17.30 - A Jakubiec-Puka: *Sarcolemma damage in extended-stimulated muscles*
17.40 - K Rossini, Department of Biomedical Sciences and C.N.R. Unit for Muscle Biology and Physiopathology, University of Padova, Italy: *Activity-induced apoptosis in mdx mice*
17.50 - C Destro, Department of Biomedical Sciences and C.N.R. Unit for Muscle Biology and Physiopathology, University of Padova, Italy: *Fas/FasL system regulates apoptosis of macrophages and myoblasts during muscle regeneration*
18.00 – TELETHON Lecture: Helen Blau, Department of Molecular Pharmacology, Stanford, CA, USA
BAM'2000 Keynote: *Gene Expression and Signal Transduction*
19.00 - Concert

June 12, 2000 (Monday)

9.00 - 13.00 - Dynamic Cardiomyoplasty

9.00 - 11.00 *Dynamic Cardiomyoplasty (I)*

Chairpersons: WP Santamore and V Chekanov

9.00 - A Carpentier & J Chachques, Broussais Hospital, Paris, France: *Present and future of Dynamic Cardiomyoplasty*

9.30 - JK Kirklin, Division of Cardiothoracic Surgery, University of Alabama, Birmingham, USA: *Cardiomyoplasty-skeletal muscle assist randomized trial (C-SMART): 6 month results*

10.00 - C Werling, Department of Cardiac Surgery, Herzzentrum Ludwigshafen, Germany: *Dynamic Cardiomyoplasty : Clinical experience after seven years*

10.15 - J Trainini, Hospital Peron, Buenos Aires, Argentina: *Chronic aortic counterpulsation with latissimus dorsi: clinical follow-up. Cardiomyoplasty comparison*

10.30 - R Lorusso, Cardiac Surgery, Spedali di Brescia, Italy: *Cardiomyoplasty and implantable defibrillator in heart failure patients: positive impact on patient survival*

10.40 - R Scelsi, Department of Human Pathology, University of Pavia, Italy: *Pathological findings in LD wrap after short- and long-term dynamic cardiomyoplasty*

10.50 - V Chekanov, Milwaukee Heart Institute, Heart Care Associates, Milwaukee, Wisconsin, USA: *A new alternative for the use of electrical stimulation – atherosclerosis prevention*

11.00 Coffee break

11.30 - 13.00 *Dynamic Cardiomyoplasty (II)*

Chairpersons: JK Kirklin, A Carpentier

11.30 - WP Santamore, Philadelphia, PA, USA*: Vascular delay and intermittent stimulation: Keys to success in Cardiomyoplasty*

11.50 – D Casarotto & C Muneretto, Cardiac Surgery, Universities of Padova and Brescia, Italy: *Three-year experience of Demand Dynamic Cardiomyoplasty*

12-00 R Riccardi, Fondazione Maugeri, IRCCS, Centro Medico Montescano, Pavia, Italy: *Demand stimulation after long-term Dynamic Cardiomyoplasty*

12.10 - GL Rigatelli & M Barbiero, Legnago General Hospital, Legnago (Vr), Italy: *Systolic assistance by Demand Dynamic Cardiomyoplasty? The answer from aortic peak flow determined by intravascular Doppler flow wire*

12.20 - M Trivellato, on behalf of the Registry partners, Ospedale Geriatrico, Padova, Italy*: The Registry of Demand Dynamic Cardiomyoplasty*

12.30 - VS Chekanov, Milwaukee Heart Institute, Heart Care Associates of Sinai Samaritan Medical Center, Milwaukee, Wisconsin, USA: *Novel functions (work-rest and day-night regimens) in a new cardiomyostimulator for cardiac bioassist*

12.40 - F Monese & F Di Gregorio, MEDICO spa, & U Carraro, Padova, Italy: *Demand Dynamic Cardiomyoplasty and LD wrap mechanography: new characteristics of a myostimulator (demand LD-cardio pacer) to physiologically activate LD wrap and clinically determine its dynamic characteristics*

12.50 – U Carraro, Department of Biomedical Sciences and C.N.R. Unit for Muscle Biology and Physiopathology, University of Padova, Italy: *Dynamic Cardiomyoplasty: It is time to wrap!*

13.00 Lunch and posters

14.15 – 17.30 – Cell and Gene Therapy in Sarcomeric Muscles: Basics and Applications

Chairpersons: F Rossi and GS Butler-Browne

14.15 - GS Butler-Browne, UFR Biomedicale des St Pères, Paris, France: *Satellite cells: life-span and telomeres*
14.35 - L Gorza Department of Biomedical Sciences and C.N.R. Unit for Muscle Biology and Physiopathology, University of Padova, Italy: *Cell-surface localization of the glucose-regulated protein GRP94 in skeletal myoblasts is involved in myotube formation*
14.50 - M Shiozuka, Department of Human Sciences, Waseda University, Japan: *A possible role of SPARC (secreted protein-acidic and rich in cysteine) in myogenesis*
15.00 - J-P Jin, Department of Physiology & Biophysics, Case Western Reserve, Cleveland, OH, USA*: Troponin T gene regulation during muscle development and adaptation, and functional significance*
15.10 - Y Atomi, Department of Life Science, University of Tokyo, Tokyo Japan*: Role of stress protein* ⍰B-crystallin for stabilization of tubulin/microtubule in vitro and in myoblast cells
15.20 - T Obinata, Department of Biology, Chiba University, Japan: *Actin filament organization is regulated by cofilin, a small G-actin-binding protein, during myofibril assembly*
15.30 - I Dell'Aica, Department of Biomedical Sciences and C.N.R. Unit for Muscle Biology and Physiopathology, University of Padova, Italy: *Telomerase activity in skeletal muscles of the mdx mice*
15.40 Coffee Break
16.00 - F Rossi, Department of Molecular Pharmacology, Stanford, CA, USA : *Myoblast-mediated delivery of tightly regulated therapeutic genes*
16.20 - J Huard, Growth and Development Lab, Children's Hospital of Pittsburg, PA, USA: *The use of muscle derived cells expressing stem cell antigens to improve the efficiency of cell transplantation to dystrophic muscle*
16.40 - A Wernig, Dept. Physiology Univ. Bonn and Klinikum Karlsbad-Langensteinbach, Germany: *Human muscle tissue grown in mice*
17.00 - L Vitiello, Department of Biology, University of Padova, Italy*: Gene transfer into skeletal muscle by means of lipopolyplexes*
17.10 - M Cantini CRIBI Center and Department of Biomedical Sciences, University of Padova, Italy: *J774 macrophage cell line secretes a specific myogenic factor capable to inducing myoblast proliferation and to delaying their differentiation*

17.20 - Shuttle bus to Padova University

Palazzo del Bò, Università di Padova
18.10 - Visit to historical Anatomical Theatre

Myology Lectures
18.40 - G Marechal, Department of Physiology and Pharmacology, UCL, Brussels, Belgium*: The effects of nitric oxide on the contraction of skeletal muscle*
19.00 - A Margreth, CNR Unit for Muscle Biology and Physiopathology c/o Department of Biomedical Sciences, University of Padova, Italy: *Species-specific biochemical properties and adaptive changes of the sarcoplasmic reticulum of human skeletal muscle under pathological conditions*

20.00: BAM Friends, Get Together

June 13, 2000 (Tuesday)

9.00 - 10.00 - Cellular Mechanisms and Progression of Muscular Dystrophies

Chairpersons: L Larsson and C Angelini

9.00 - C Angelini, Neuromuscular Center, Department of Neurology, University of Padova, Italy: *Clinico molecular correlations in dystrophinopathies and carriers*

9.20 - A Stracher, Stony Brooks, New York, USA: *Ca++/calpain hypothesis for neuromuscular degeneration*

9.30 - R Betto, CNR Unit for Muscle Biology and Physiopathology, University of Padova, Italy: *Role of purinergic receptors in the pathogenesis of sarcoglycanopathies*

9.40 - G Siciliano, *Metabolic and muscle adaptation to aerobic training in mitochondrial myopathies*

9.50 - M Bonifati, Neuromuscular Center, Department of Neurology, University of Padova, Italy: *A multicenter double-blind randomized trial of deflazacort versus prednisone in Duchenne muscular dystrophy: analysis after 2 years*

Coffee Break

10.30 - 13.30 - Mechanisms of Cellular Adaptation to Workload and Exercise-induced Muscle Damage

Chairpersons: Y Mounier and C Reggiani

10.30 - L Larsson, Noll Physiological Research Center & Department of Cellular and Molecular Physiology, School of Medicine, Hershey Medical Center, The Pennsylvania State University, USA: *Effects of aging on the regulation of muscle contraction at the motor unit, muscle cell, and molecular levels*

11.00 - C Reggiani, Department of Human Anatomy and Physiology, University of Padova, Italy: *Disuse induced-atrophy and contractile impairment of human skeletal muscle fibres*

11.15 - Y Mounier, Laboratoire de Plasticité Neuromusculaire, UST Lille, France: *Troponine C plasticity in unloading conditions*

11.30 - H Kern, University of Vienna: *Denervated muscles in human: First results of training with electrical stimulation*

11.45 - M Falempin, Laboratoire de Plasticité Neuromusculaire, UST Lille, France: *Sole plantar stimulation: a countermeasure for rat soleus atrophy observed during unloading*

12.00 - A Wernig, Dept. Physiology Univ. Bonn and Klinikum Karlsbad-Langensteinbach, Germany: *Locomotor (Laufband) therapy in SCI persons*

12.15 - W Mayr, Dept. of Biomedical Engineering and Physics, and Dept. of Plastic and Reconstructive Surgery, University of Vienna Medical School, Austria: *Functional electrostimulation as a countermeasure against muscular atrophy in long-term space flights*

12.30 - A Martinuzzi, *Walking energy cost in children with neurogenic motor impairment*

12.40 - C Chisari: *Impaired muscle oxidative metabolism in polymyositis and dermatomyositis evaluated in vivo through lactic acidaemia assay*

12.50 - O Rossetto, Department of Biomedical Sciences, University of Padova: *Recovery of human neuromuscular junction after botulinum neurotoxin therapy*

13.00 - A Megighian, Human Physiology, University of Padova: *Effects of tenotomy on rat slow muscle regeneration*

Lunch and posters

14.30 - 18.00 - Cardiac-Bio-Assists
Chairpersons: J Jarvis and NW Guldner
14.30 - J Jarvis, Department of Anatomy and Cell Biology, University of Liverpool, UK: *Skeletal muscle ventricles in the pig: left ventricular off-loading demonstrated by observation of pressure-volume loops*
15.00 - NW Guldner, Clinic of Cardiac Surgery, Medical University of Lubeck, Germany: *Biomechanical Hearts Performed in a One-Step Operation and Trained Dynamically under Support of Clenbuterol*
15.30 - B Voss, Department of Cardiac Surgery, Deutsches Herzzentrum München, Germany: *Dynamic Cardiomyoplasty in a growing organism*
15.45 - R Lorusso, Cardiac Surgery, Spedali di Brescia, Italy: *Muscle power after vascular delay in a sheep model of muscle transposition.*
16.00 - V Chekanov, Milwaukee Heart Institute, Heart Care Associates, Milwaukee, Wisconsin, USA: *Pharmacological support of angiogenesis using deferoxamine in biological glue for cardiomyoplasty*
16.15 - A Shafy/JC Chachques, Department of Cardiac Surgery, Broussais Hospital, Paris, France: *Association of cellular cardiomyoplasty with multisite cardiac pacing*
16.30 - E Monnet, Department of Clinical Sciences, Colorado State University, Fort Collins CO, USA: *Is injury to the toracodorsal nerve present after long-term dynamic cardiomyoplasty in goats?*
16.45 - P Klapproth, Clinic of Cardiac Surgery, Medical University of Lubeck, Germany: *Methods to evaluate dynamic training of skeletal muscle ventricles and biomechanical hearts*
17.00 - E Giardini, Institute of Plastic Surgery, University of Padova, Italy: *Vascular delay of a LD in an experimental rat model for dynamic cardiomyoplasty*
17.15 - U Carraro, Department of Biomedical Sciences, University of Padova, Italy*: Myoblast cell therapy to augment muscle mass in Cardiac-Bio-Assists*

POSTERS

1. MYOTUBES OVER-EXPRESSING Fas L PROTEIN UNDER MCK PROMOTER ARE RESISTANT TO Fas-Fas L-INDUCED PAPOOSES, A Bon, M Sandri, C Radu, S Tiozzo, E Sartori, L Vitiello, M Cantini, CRIBI Center, and Departments of Biomedical Sciences and Biology, University of Padova, Italy
2. PROPOSAL OF VALUTATIVE AND REHABILITATIVE PROTOCOL IN FACIOSCAPULOHUMERAL DYSTROPHY, Chisari C, Giannini E, Mussini F, Simonella C, Bresci M, Rossi B, Neurorehabilitation Unit, Dept. of Neuroscience, University of Pisa
3. SURFACE EMG TO STUDY MUSCLE FUNCTION IN ASYMPTOMATIC DIABETIC NEUROPATHY, Chisari C, Piaggesi A, Baccetti F, Rizzo L, Giannini E, Simonella C, Rossi B, Neurorehabilitation, Dept. of Neuroscience, and Chair of Metabolic Diseases, Dept. of Endocrinology and Metabolism, University of Pisa
4. IMPAIRED MUSCLE OXIDATIVE METABOLISM IN POLYMYOSITIS AND DERMATOMYOSITIS EVALUATED *IN VIVO* THROUGH LACTIC ACIDAEMIA ASSAY, Chisari C, Stampacchia G, Giannini E, Neri R, Mosca M, Rossi B, Neurorehabilitation Unit, Dept. of Neuroscience, and Reumatology Unit, Dept of Medicine, University of Pisa
5. CORRELATIVE ^{31}P-MRS - M WAVE ANALYSIS OF HIGH AND LOW FREQUENCY ELECTRICALLY-INDUCED FATIGUE IN RABBIT *TIBIALIS ANTERIOR* MUSCLE, J-L Darques,

D Bendahan , M Roussel , Y Le Fur , S Confort-Gouny , F Tagliarini , PJ Cozzone , Y Jammes, Laboratoire de Physiopathologie Respiratoire (UPRES EA 2201), Faculté de Médecine and Centre de Résonance magnétique biologique et médicale, UMR CNRS 6612, Univ. Méditerranée, Marseille, France.

6. SOLE PLANTAR STIMULATION: A COUNTERMEASURE FOR RAT SOLEUS ATROPHY OBSERVED DURING UNLOADING, L De Doncker, F Picquet, M Falempin, Laboratoire de Plasticité Neuromusculaire - SN4 - UST Lille 1, Villeneuve d'Ascq, Lille, France
7. TELOMERASE ACTIVITY IN MUSCLE OF NORMAL AND *MDX* MICE AND IN MUSCLE-REGENERATION, I Dell'Aica, M Sandri, K Rossini, C Destro, U Carraro. C.N.R. Unit for Muscle Biology and Physiopathology, Department of Biomedical Sciences, University of Padova, Italy.
8. Fas/FasL SYSTEM REGULATES APOPTOSIS OF MACROPHAGES AND MYOBLASTS DURING MUSCLE REGENERATION, C Destro, M Sandri, K Rossini, C Sandri, I Dell'Aica, U Carraro, C.N.R. Unit for Muscle Biology and Physiopathology, Department of Biomedical Sciences, University of Padova, Italy.
9. VASCULAR DELAY OF LD IN AN EXPERIMENTAL RAT MODEL FOR DYNAMIC CARDIOMYOPLASTY, E Giardini, F Mazzoleni, K Rossini (1), A El Messlemani (1), A Donà (1), U Carraro (1), Institute of Plastic Surgery, and (1) C.N.R. Unit for Muscle Biology and Physiopathology, Department of Biomedical Sciences, University of Padova, Italy
10. METHODS TO EVALUATE DYNAMIC TRAINING OF SKELETAL MUSCLE VENTRICLES AND BIOMECHANICAL HEARTS, P Klapproth, NW Guldner, M Grssherr (1), HH Sievers, Clinic of Cardiac Surgery, and (1) Clinic of Anaesthesiology, Medical University of Lubeck, Germany
11. SORTING AND DYNAMICS OF MYOSIN ALKALI LIGHT CHAIN EXPRESSED IN CULTURED CARDIOMYOCYTES AND CARDIAC FIBROBLASTS, MM Khan, M Komiyama, Y Shimada, Department of Anatomy and Cell Biology, School of Medicine, Chiba University, Japan
12. IDENTIFICATION OF MUSCLE DERIVED STEM CELLS CAPABLE OF DIFFERENTIATING INTO OSTEOGENIC LINEAGES AND IMPROVING BONE HEALING, JY Lee, Z Qu-Petersen, B Cao, S Kimura, R Jankowski, D Musgrave, P Bosch, C Gates, P Robbins, A Wernig, J Huard, Growth and Development Laboratory, Department of Orthopaedic Surgery, University of Pittsburg and Children's Hospital of Pittsburgh, PA, Department of Molecular Genetics and Biochemistry, University of Pittsburgh, PA, USA and Department of Physiology, Neurophysiology, University of Bonn, Germany
13. WALKING ENERGY COST IN CHILDREN WITH NEUROGENIC MOTOR IMPAIRMENTS, A Martinuzzi, E Trevisi, P Zamparo (1), PE Di Prampero (1), IRCCS "E. Medea" Polo Regionale di Conegliano, (1) Dip. Scienze e Tecnologie Biomediche, Università di Udine.
14. EFFECTS OF TENOTOMY ON RAT SLOW MUSCLE REGENERATION, A Megighian, D Danieli Betto, E Germinario, M Midrio, Department of Human Anatomy and Physiology, University of Padova, Italy
15. DEMAND DYNAMIC CARDIOMYOPLASTY AND LD WRAP MECHANOGRAPHY: NEW CHARACTERISTICS OF A MYOSTIMULATOR (DEMAND LD-CARDIO PACER) TO PHYSIOLOGICALLY ACTIVATE LD WRAP AND CLINICALLY DETERMINE ITS DYNAMIC CHARACTERISTICS, F Monese, F Di Gregorio, U Carraro (1), MEDICO s.p.a., Rubano (Padova), (1) Department of Biomedical Sciences, University of Padova, Italy

16. GENE TRANSFER INTO SKELETAL MUSCLE BY MEANS OF LIPOPOLYPLEXES, F Pampinella, M Pozzobon, E Zanetti, P Gamba, GL Lukacs, M Cantini, L Vitiello, CRIBI Center and Departments of Biomedical Sciences, Biology and Pediatrics, University of Padova, Italy, Cell and Lung Biology, Research Institute, Hospital for Sick Children, Toronto, Canada
17. THE TRANSDUCTION OF SKELETAL MUSCLE WITH ADENO-ASSOCIATED VIRUS IS FIBER DEPENDENT, R Pruchnic, BH Cao, Z Qu, X Xiao, J Li, RJ Samulski, M Epperly, J Huard, Department of Orthopaedic Surgery, Department of Molecular Genetics and Biochemistry, University of Pittsburgh, PA, University of North Carolina, Chapel Hill, NC, USA
18. EXERCISE LACTATE ANAEROBIC THRESHOLD IN HEREDITARY SPASTIC PARAPLEGIA, L Pasquali, ML Manca, E Pastorini, FM Santorelli, G Siciliano, Department of Neurosciences, Neurological Clinics, University of Pisa, Italy
19. J774 MACHROPHAGE CELL LINE SECRETES A SPECIFIC MYOGENIC FACTOR CAPABLE TO INDUCING MYOBLAST PROLIFERATION AND TO DELAYING THEIR DIFFERENTIATION, C Radu, S Tiozzo, A Bon, G Zaniolo, E Sartori, M Cantini, CRIBI Center and Departments of Biomedical Sciences and Biology, University of Padova, Italy
20. TIME-COURSE OF EXERCISE AND APOPTOSIS IN THE MDX MICE, K Rossini, A Donà, M Sandri, U Carraro, C.N.R. Unit for Muscle Biology and Physiopathology, Department of Biomedical Sciences, University of Padova, Italy.
21. IS A HIGH INTRACELLULAR LIPID CONTENT AN ADAPTATIONAL RESPONSE TO A LOW CAPILLARY SUPPLY IN SKELETAL MUSCLE?, AM Saenger, Institute of Zoology, University of Salzburg, Austria
22. ENDURANCE TRAINING AFFECTS WHITE AXIAL MUSCLE IN THE CYPRINID SPECIES CHALCALBURNUS CHALCOIDES MENTO (AGASSIZ, 1832), CYPRINIDAE, TELEOSTEI, AM Saenger, U Putscher, Institute of Zoology, University of Salzburg, Austria
23. ASSOCIATION OF CELLULAR CARDIOMYOPLASTY WITH MULTISITE CARDIAC PACING, A Shafy, JC Chachques, P Argyriadis, L Shen, C Rajnoch, A Berrebi, JN Fabiani, A Carpentier, Department of Cardiac Surgery, Broussais Hospital, Paris, France
24. A POSSIBLE ROLE OF SPARC (SECRETED PROTEIN-ACIDIC AND RICH IN CYSTEINE) IN MYOGENESIS, M Shiozuka, Department of Basic Human Sciences, School of Human Sciences, Waseda University, Saitama, Japan
25. DYNAMIC CARDIOMYOPLASTY IN A GROWING ORGANISM , B Voss, M Thielmann, M El-Mehsen, PA Schnabel (1), S Hagl (1), R Lange, Department of Cardiac Surgery, Deutsches Herzzentrum München, and (1) Departments of Cardiac Surgery and Pathology, University Hospital Heidelberg, Germany
26. DEVELOPMENTAL RELATIONSHIP OF MYOSIN BINDING PROTEINS (CONNECTIN, MYOMESIN and C-PROTEIN) TO MYOSIN IN CHICKEN SOMITES AS STUDIED BY CONFOCAL MICROSCOPY, Y Yang, T Obinata, Y Shimada, Department of Anatomy and Cell Biology, School of Medicine, Chiba University, Japan

2022 PDM3 On-site, March 30 to April 3, PROGRAM

ABSTRACTS Link: https://www.pagepressjournals.org/index.php/bam/article/view/10440

2022 Padua Days on Muscle & Mobility Medicine
Hosts the University of Florida Myology Institute and Wellstone Center
Padua & Thermae of Euganean Hills, Padua (Italy),
Hotel Petrarca, Montegrotto, Piazza Roma 23, Montegrotto Terme, Euganean Hills, (Padua), 35122 Italy
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Organizers

Ugo Carraro, Raffaele De Caro, Paolo Gargiulo, Russell T. Hepple, Andrew R. Judge, Helmut Kern, Christiaan Leeuwenburgh, Stefano Masiero, Marco Narici, Feliciano Protasi, Marco Sandri, H. Lee Sweeney

WEDNESDAY March 30, 2022

Aula Magna Galileo Galilei, Padua University (Padua), Italy

9:00 AM *Openings*

Daniela Mapelli*, Rettrice Magnifica* of the Padua University
H. Lee Sweeney*,* Head of the University of Florida Myology Institute
Marco Narici*,* Head of the Padua University IRC - Myology

09:20* AM *Lecture: ***The crucial role of force transmission in muscle function: Focus on cytoskeleton and myotendinous junction***

A: Stefano Schiaffino*, University of Padua, Italy: Cytoskeletal specialization and mechanoprotection in fast and slow skeletal muscles. Insights from single-fiber proteomics and comparison with cardiac muscle.*

B: Abigail Mackey*, University of Copenhagen, Denmark: The myotendinous junction: the weakest link of the muscle-skeleton force transmission chain?*

10:00 AM - SESSION I: Molecular mechanisms regulating muscle mass

Marco Sandri, Andrew R. Judge, Chairs

10:00-10:20 AM Daniel Taillandier: *Mechanisms of muscle atrophy: from UPS implication in rodent models to human biomarkers*

10:20-10:40 AM Pascal Maire: *A fast Myh super enhancer dictates adult muscle fiber phenotype through competitive interactions with the fast Myh genes*

10:40-11:00 AM Marco Sandri: *Novel players in muscle mass regulation*
11:00-11:10 AM Daria Neyroud et al.: *Loss of MuRF1 prevents skeletal muscle wasting and weakness, and slows the rate of tumor growth, in mice bearing pancreatic tumors*
11:10 Open Bar
11:20-11:40 AM Marcus Ruegg: *Insights into the mechanisms of muscle wasting at high age*
11:40-12:00 AM Andrew R. Judge: *Molecular mechanisms of cancer-induced muscle wasting*
12:00-12:20.AM Marco Narici: *Neuromuscular basis of disuse muscle atrophy and weakness*
12:20-12:30 AM Bert Blauw: *Activation of Akt-mTORC1 signaling reverts cancer-dependent muscle wasting*

2:00 PM - SESSION II: Muscle metabolism, mitochondria
Andrew R. Judge, Christiaan Leeuwenburgh, Chairs

2:00-2:20 PM Antonio Zorzano: *Mitochondrial fusion proteins and their role in metabolism and in inflammation*
2:20-2:40 PM Jorge Ruas: Dynamic regulation of muscle mass by dark matter DNA
2:40-3:00 PM Antonio Musarò: *The role of IL-6 signaling in muscle growth, atrophy, and wasting*
3:00-3:20 PM Ashley J. Smuder et al.: *Diaphragm ABCB6 overexpression preserves respiratory function following doxorubicin chemotherapy treatment*
3:20-3:40 PM Mattia Scalabrin et al.: S*keletal muscle homeostasis in an experimental model of hind limb ischemia*
3:40-4:00 PM Feliciano Protasi: *Exercise-dependant remodelling of the sarcotubular system: the role of temperature and pH.*
4:00 PM Open Bar

2:00 PM - SESSION II: Muscle metabolism, mitochondria, continue –
Christiaan Leeuwenburgh, Andrew R. Judge, Chairs

4:15-4:35 PM Elisabeth Barton: *Form vs. function: strategies to deliver IGF-I for muscle therapeutic*
4:35-4:55 PM Leonardo Ferreira: *Metabolic link between mitochondrial and contractile abnormalities*
4:55-5:15 PM Christiaan Leeuwenburgh: *Higher abundance of deletions and strand break damage within specific mitochondrial ETC genes are associated with functional performance in older adults*
5:15-5:30 PM Massimo Ganassi: *Molecular Antagonism between DUX4 and DUX4c Highlights a Potential Pathomechanism in Facioscapulohumeral muscular dystrophy*
5:30-5:45 PM Rosanna Piccirillo et al.: *The p97/Nploc4 ATPase complex plays a role in muscle atrophy during cancer and amyotrophic lateral sclerosis*
5:45 - 6:00 PM Roberta Sartori et al.: *Deciphering the cachexia-inducing signature*

THURSDAY March 31, 2022

Hotel Petrarca, Montegrotto, Euganean Hills (Padua), Italy

9:0 AM - Session III: Muscle diseases

H. Lee Sweeney, Gillian Butler-Browne, Chairs

9:00-9:20 AM Francesco Muntoni: *The challenges of AAV gene therapy in DMD*
9:20-9:40 AM Kevin Flannigan: *Alternative gene therapy approaches for DMD*
9:40-10:00 AM H. Lee Sweeney: *Limitations to micro-dystrophin gene therapy for DMD*
10:00-10:15 AM: Young il Lee et al.: *Micro-dystrophin-mediated utrophin displacement from cardiomyocyte sarcolemma in the D2.mdx mouse model of DMD*
10:15-10:30 AM David W. Hammers: *NOX4 inhibition reduces skeletal muscle fibrosis in a severe murine model of Duchenne muscular dystrophy*
10:30-10:45 AM David Israeli et al.: *The co-administration of simvastatin does not boost the benefit of gene therapy in the mdx mouse model for Duchenne muscular dystrophy*
10:45 Open Bar
10:55-11:10 AM Tanja Taivassalo et al.: *Therapeutic potential of combined cycling and isometric strength training in patients with DMD: preliminary findings*
11:10-11:25 AM Piera Smeriglio et al.: *Uncovering the epigenetic control of paracrine crosstalk between motor neurons and skeletal muscles in SMA*
11:25-11:40 AM Alexis Boulinguiez et al.: *Targeting ER stress to resolve aggresome accumulation in oculopharyngeal muscular dystrophy.*
Lunch

2:00 PM - Session IV: Trainee Data Blitz Session

H. Lee Sweeney, Gillian Butler-Browne, Chairs

2:00-2:09 PM Cora C. Hart et al.: *D2.mdx mice undergo a transient period of left ventricular*
restriction prior to heart failure
2:09-2:18 PM Alessandra Norris et al: *Uncovering a novel mechanism for intramuscular fat formation*
2:18-2:27 PM Christopher A. Wolff et al.: *Muscle clocks change with age: A potential contributor to sarcopenia?*
2:27-2:36 PM Chandler Callaway et al.: *Cancer cell-derived IL-8 and CXCL1 mediate cachexia in mice bearing human pancreatic tumors*
2:36-2:45 PM Vinicius M. Mariani et al.: *Succinate impairs skeletal muscle isometric and isotonic contractile function*

2.45 PM - Session IV: Trainee Data Blitz Continued,

Andy Judge, Marco Sandri, Chairs

2:45-2:50 PM Miguel A. Gutierrez-Monreal et al.: *Skeletal muscle specific rescue of Bmal1 is sufficient to extend the lifespan of the Bmal1 KO mouse*
2:50-2:59 PM Chih-Hsuan Chou et al.: *The role of muscle IGF-I after a single bout of exercise on AMPKα in mouse skeletal muscle*
2:59-3:08 PM Giulia Trani et al.: *Peroxisomal-mitochondrial interaction impinging on muscle homeostasis*
3:08-3.17 PM Miriam Mistretta et al.: *Dysregulation of heme synthesis-export axis in*

skeletal muscle reshapes energetic metabolism and results in impaired motor performance
3:17-3:26 PM Davide Steffan et al.: *Identification of a novel TFEB and exercise dependent gene*
3:26-3:37 PM Camilla Pezzini et al.: *Understanding BMP signaling in cancer cachexia*
3:37-3:46 PM Hui Jean Kok et al.: *IGF-I from satellite cells is critical for skeletal muscle growth and regeneration*
4:50 Open bar

4:50 PM - Session V: Other European Contributions to Muscle Diseases,
Corrado Angelini, Marija Meznaric, Chairs

4:50-5:10 PM Francesco Girardi: *Video killed the Imaging star*
5:10-5:23 PM Sonia Albini et al.: *A Dual-AAV gene therapy strategy for Duchenne Muscular Dystrophy*
5:23-5:36 Roberta Costa et al.: *Pathogenetic mechanism of Limb Girdle Muscular Dystrophy D2: functional characterization of Transportin 3 in cellular and animal models of disease*
5:36 - 5:49 Sara Missaglia et al.: *Neutral Lipid Storage Diseases: a patient clinical follow-up and presentation of two novel cases*
5:49-6:02 PM Eylem Emek Akyürek et al.: *Human Brody disease and novel therapeutic approaches of its animal model cattle pseudomyotonia*
6:02-6:15 PM Martina Scano et al.: *The strength and the broadness of CFTR correctors for the treatment of sarcoglycanopathies*
6:15-6:28 Mark Viggars et al.: *The timecourse of adaptive change in gene expression across 30d of daily programmed resistance exercise in rats*
6:28-6:41 PM Stephen Gargan et al.: *Proteomic profiling of the aged diaphragm from the mdx-4cv model of dystrophinopathy*
6:41-6:52 PM Raphael S. Bonadio, Stefano Cagnin: *New molecular network identified in Amyotrophic Lateral Sclerosis reveals microRNAs involved in the neuromuscular junction development*
6:52-7:05 PM Marija Meznaric, Corrado Angelini: *Differential dysferlin expression in human fast and slow skeletal*
7:05-7:18 PM PM Valentina Pegoraro et al.: *Two brothers with X-linked Charcot Marie Tooth disease and different lifestyle*
7:20 PM Adjourn

FRIDAY April 1, 2022

Hotel Petrarca, Montegrotto, Euganean Hills (Padua), Italy

9.00 AM - SESSION VI: The curse of inactivity
Marco Narici, Roberto Bottinelli, Chairs

9:00 – 09:20 AM Gianni Biolo: *Energy balance and skeletal muscle in microgravity*
9:20 – 09:40.AM Bruno Grassi: *Peripheral impairments of oxidative metabolism during exercise following inactivity*
09:40 – 10:00 AM Roberto Bottinelli: *Metabolic dysfunction and exercise preconditioning in disuse*
10:00 – 10:20 AM Giuseppe De Vito: *Alterations in the behavior of individual motor units with inactivity*
10:20 AM Open Bar

10:20 AM - SESSION VII: Subclinical denervation in aging skeletal muscle
Russell T. Hepple, Raffaele De Caro, Chairs

10:20-10:40 AM Gregorio Valdez: *A tripartite view of NMJ aging: parsing out the contribution of motor neurons, muscle fibers and synaptic Schwann cells*
10:40- 11:00 AM Richard Robitaille: *Changes of neuromuscular innervation during aging in healthy males*
11:10-11:20 AM Russell T. Hepple: *Mechanisms of exacerbated denervation in aging muscle*
11:20-11:40 AM Dario Coletti, Ugo Carraro: *Mosole's evidence of transforming muscle fibers coexpressing fast and slow myosin heavy chains in slow type muscle-groupings of life-long active seniors*

11:40 AM - SESSION VIII: Masters Athletes as a model for Healthy Aging
Russell T. Hepple, Ugo Carraro, Chairs

11:40 – 12:00 AM Matthew Piasecki: *Motor unit adaptations in Masters Athletes*
12.00 – 12:20 AM Russell T. Hepple: *Insights to mechanisms of healthy muscle aging in octogenarian track and field athletes*
12:20 – 12:40 AM Giovanna Albertin et al.: *The Venice Marathon 2007-2019 as a model for analyses of Master Athletes*
12:40 – 12.50 AM Ugo Carraro et al.: *Master Athletes' Studies in Padua honor the legacy of Paolo Gava*
13:00 PM Break for Lunch

2:00 PM - Session IX-: Basic and clinical muscle imaging
Simona Boncompagni, Shantanu Sinha, Chairs

2:00 – 2:20 PM Usha Sinha: *Fiber Strains and Strain Tensor Mapping of Medial Gastrocnemius at Sub-Maximal Isometric Contraction at Different Ankle Angles*
2:20 – 2:40 PM Shantanu Sinha: *Calf Muscle 3D Strain Imaging and Initial Results on Correlation with Histology*
2:40 – 3:00 PM Simona Boncompagni: *New intracellular junctions: The calcium entry units*
3:30 - 3:20 PM Marcus Kruger: *Looking at the proteome landscape in single muscle fibers*
3:20 – 3:40 PM Jonathan C. Jarvis: *PCM1 labelling reveals myonuclear and nuclear dynamics in skeletal muscle across species*
3:40 –4:00 PM Amber L. Pond: *The HERG K+ Channel Increases Intracellular Calcium Concentration in Myotubes by Modulation of IP3 Signaling*
4:00 – 4:20 PM Lorenzo Marcucci et al.: *Cytosolic calcium as intracellular signal: local and average concentrations and their variations in relation to release from SR.*
4:20 PM Open Bar

4.30 PM - SESSION X: Artificial lntelligence for myopathology diagnosis & management
Paolo Gargiulo, Michael J. Fischer, Chairs

4:30 – 4:55 PM Paolo Gargiulo: *New paradigms for 3D modelling and surgical planning*
4:55 – 5:20 PM Vincent Grote, Michael J. Fischer: *Prospects for translational research on outcome measures in musculoskeletal rehabilitation: the search for critical success factors*

5:20 – 5:35 PM Carlo Ricciardi, Marianna Amboni: *Gait analysis for the detection of non-motor mental symptom in Parkinson's disease*
5:35 – 5:50 PM Marco Recenti et al.: *A Novel Knee Bone and Cartilage Osteoarthritis Index Extracted from a Patient-Specific Image Feature Analysis*
5:50 – 6:05 PM Andrea Colacino et al.: *Postural Control Assessment through Visual Induced Motion Sickness and a Moving Force Platform*
60:05 – 6:20 PM Maria Laura Gatto et al.: *Multi-scale Bone Remodeling Prediction in Patients Undergoing Total Hip Arthroplasty*
6:20 – 6:35 PM Magnús Kjartan Gíslason et al.: *Neuromuscular control in the neck muscles in patients suffering from whiplash associated disorders and traumatic brain injury*
6:35 – 6:50 PM Eva Kohlscheen, Tito Brambullo, Vincenzo Vindigni: *A new CT analysis of abdominal wall after DIEP flap harvesting*

7:00 PM Adjourn

SATURDAY April 2, 2022

Hotel Petrarca, Montegrotto, Euganean Hills (Padua), Italy

9.00 AM - SESSION XI: Active aging and early rehabilitation management
Helmut Kern, Feliciano Protasi, Chairs

9:00 – 9:20 AM Helmut Kern: *Centre of Active Ageing (Austria): current status*
9:20 – 9:40 AM Stefan Loefler: *AMB-REMOB - study protocol of an early outpatient rehabilitation program*
9:40 – 10:00 AM Jan Cvecka: *Exercise intervention in elderly: a novel system within the Centre of Active Aging in Bratislava*
10:00 - 10:20 Nejc Sarabon: *The potential of eccentric training in older adults*
10:20 - 10:40 AM Feliciano Protasi: *Mimicking disuse and rehabiliation in a mouse model*
10:40 - 11:00 AM Sandra Zampieri: *The effects of exercise on skeletal muscle atrophy and denervation in ageing and disuse*

Coffee Break

11:00 AM- SESSION XII: Climate changes and heat strokes - The role of muscle
Feliciano Protasi, Chair

11:00 - 11:00 AM Piero di Carlo: *Heatwaves in a warming climate: overview and impacts*
11:20 - 11:40 AM Feliciano Protasi: *Environmental Heat Stroke: the role of skeletal muscle*
11:40 - 12:00 AM Matteo Serano et al.: *High-fat diet increases the risk of environmental heatstroke in mice*
12:00 - 12:20 AM Barbara Girolami et al.: *Exertional Heat Stroke: the possible role of external Ca^{2+}* 12:30 Break for Lunch

2:00 PM - SESSION XIII: Basics of neuromechanics & motor control
Laura Schaefer, Alessandro Del Vecchio, Chairs

2:00 - 2:20 PM Alessandro Del Vecchio: *Integration of Motion, Forces, and the Central Nervous System*
2:20 - 2:40 PM Stéphane Baudry: *On the role of proprioception in the sense of force*
2:40 – 3:00 PM Frank Bittmann, Laura Schaefer: *Adaptive Force in Patients with Long-COVID*
3:00 – 3:15 PM Leonardo Cesanelli et al: *The role of age on neuromuscular performance decay induced by a maximal intensity sprint session in a group of competitive athletes*
3:15 – 3:30 PM Caterina Fede et al.: *Connections between hyaluronan properties and fascial health*

3:30 p.m. - SESSION XIV: Muscle stimulation in rehabilitation
Winfried Mayr, Thordur Helgason, Chairs

3:30 - 3:50 PM Winfried Mayr: *How far can electrical stimuli recruit specific neurons: mechanisms, realistic options and limits*
3:50 - 4:10 PM Ines Bersch-Porada: *The effect of direct muscle stimulation on denervated gluteal muscles and tissue composition in people with chronic spinal cord injury*
4:10 - 4:30 PM Thordur Helgason, et al.: *Spinal cord stimulation review and recent progress*
4:30 – 4:50 PM Sara Kristinsdóttir, et al.: *Development of an experimental setup for exact measurement of time in event chain of patellar reflex test, transcutaneus spinal cord stimulation (tSCS) and H-reflex analysis in healthy, spinal cord injured and brain insulted individuals*
4:50 – 5.10 PM Serafina Pacilio et al.: *Skeletal muscle tissue restoration using functionalized biomaterials*
5:10 - 5:30 PM Janine Tomasch et al.: *Establishment of models for mechanical and oxidative stress based on tissue-engineered skeletal muscle*

5:30 PM - SESSION XV: Myo-rehabilitation in dentistry and beyond
Elena P. Ivanova, Riccardo Rosati, Chairs

5:30 - 5.50 PM Elena P. Ivanova, Riccardo Rosati: *Dental rehabilitation from a muscular point of view*
5.50 - 6:10 PM Claudia Dellavia et al.: *Head and neck functional analysis: the Functional Anatomy Research Center experience*
6:10 - 6:30 PM Giacomo Begnoni: *Masticatory muscles guided orthodontic treatments*
6:30 - 6:50 PM Andrey Rachin: *Topical aspects of neuro-rehabilitation*
6:50 – 7:10 PM Ekaterina V Makarova et al.: *Back muscles training and balance therapy in rehabilitation of patients with osteoporotic vertebral fractures*

SUNDAY April 3, 2022

Hotel Petrarca, Montegrotto, Euganean Hills, (Padua), Italy

9:00 AM - SESSION XVI: Translational rehabilitation
Carla Stecco, Stefano Masiero, Chairs

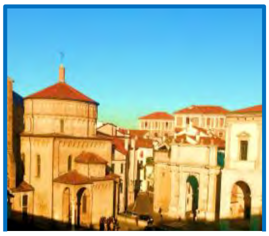
9:00 - 9:20 AM Carla Stecco et al.: *Fascia lata alterations in Hip Osteoarthritis*
9:20.-.9:40 AM Daniele Coraci et al.: *Rehabilitation of peripheral nerve disorders by physical agents. A multiperspective literature evaluation*
9:40 - 10:00 AM Lucrezia Tognolo: *Extracorporeal Shock Wave Therapy (ESWT) in muscular pathologies*
10:00 - 10:20 AM Maria Chiara Maccarone et al.: *Can home-based rehabilitation be effective to counteract skeletal muscle atrophy and to ameliorate physical functioning of elderly patients?*
10:20 - 10:40 PM Kirill V. Terentev et al.: *Early rehabilitation of ischaemic stroke with medicinal acupuncture: A clinical study*
10:40 - 11:00 AM Irina A. Grishechkina et al.: *Can aqua exercises in fresh water improve the gait stereotype function in patients with a neurological disease?*

11:00 AM - SESSION XVI: Translational rehabilitation, continue
Carmelo Chisari, Giuseppe Messina, Chairs

11:00.-.11:20 AM Anatoly D. Fesyun, Maxim Yu. Yakovlev*: Sanatorium and spa resort treatment of patients who have recovered from COVID-19*
11:20.-.11:40 AM Valentina Azzollini et al.: *Focal Muscle Vibration and Action Observation: a novel approach for muscle strengthening*
11:40 - 12:00 AM Carmelo Chisari et al.: *A combined treatment protocol for postural instability in Pisa Syndrome*
12:00 - 12:20 AM Giuseppe Messina et al.: *Study of correlations between neuromuscular occlusion and posturographic parameters in the elderly for falls prevention: a pilot study*
12:20 - 12:40 AM Patrizia Proia et al.: *Improvement of upper limb muscle strength and balance in patients with multiple sclerosis, through a proprioceptive rehabilitation protocol combined with the application of photon emission device*
12:40 - 12:50 AM Igor Reverchuk et al.: *Mental health disorders of relatives of oncohematological patients*
12:50 – 12:59 AM Anna Zavertyaeva et al.: *The state of helplessness in preschool children with mental retardation and its correction*

01:00 PM See you to the 2023 Padua Days of Muscle and Mobility Medicine (2023 PDM3) *Ugo Carraro*

Despite worsening times in Europe, I am confident that the PDM3 will attract international speakers for the years to come.
The Program of the 2023 Pdm3 at January 6, 2023 follow




2023 Padua Days on Muscle & Mobility Medicine

Hotel Petrarca, Piazza Roma 23, Montegrotto Terme, Euganean Hills, (Padua), 35122 Italy
Phone +39 049 891 1744 - Email: petrarca@hotelpetrarca.it -
https://www.hotelpetrarca.it/

WEDNESDAY March 29, 2023

08:00 AM ***Complimentary Bus Transfer to Padua***

Guariento Hall, Galilean Academy of Arts and Science of Padua, Italy

09:00 AM ***Openings*** **Raffaele De Caro, Marco Narici, Marco Sandri:**
Greetings and thanks to lecturers, speakers, audience and sponsors

09:20 AM ***Lecture of Carlo Reggiani:*** **Single muscle fibers as a tool in aging research**

10:00 AM ***Session I: Adaptations to Physical Exercise in Aging: from cell to functioning -*** *Marco Narici, Ugo Carraro, Chairs*

10:00 AM ***Russell Hepple, University of Florida, USA*****: Skeletal Muscle Plasticity to Resistance Training in Pre-frail/Frail Elderly Women**

10:20 AM ***Charlotte Suetta, University of Copenhagen, DK:*** **Rehabilitation for life: the effect on physical function of rehabilitation and care in older adults**

10:40 AM ***Julian Alcázar Caminero, Universidad de Castilla-La Mancha, Toledo, Spain:*** **Improving muscle power through exercise training in old age**

11:00 AM ***Simone Porcelli, University of Pavia, Italy*****: Home-based aerobic exercise training improves skeletal muscle oxidative metabolism in old people**

11:20 AM ***Martino Franchi, University of Padua, Italy:*** **Differential muscle adaptations to concentric and eccentric resistance training in older people**

11.40 AM ***Marco Narici, University of Padua, Italy:*** **The neuroprotective effects of exercise in older age**

12:00 AM ***Gianni Biolo, University of Trieste, Italy:*** **Nutrient-exercise interaction on muscle mass and function in aging**

12:20 AM ***Maria Chiara Maccarone, Barbara Ravara, Walter Giuriati, Stefano Masiero, Ugo Carraro, University of Padua, Italy:*** **Combating muscle weakness in bed-ridden eldery with Home-based Full-Body in-Bed Gym (hbFBiBG): Basics, Implementation and Preliminary Results of the Padua Initiative**

12.45 AM ***Lunch in Piazza Duomo, Padua, Italy***

02:00 PM SESSION II: ***Skeletal Muscle Epigenetics and the dark side of the genome***
Piera Smeriglio, Marco Sandri, Chairs

02:00 PM ***Marco Sandri et al., University of Padua, Italy:*** **Discovering novel longevity genes by looking at the dark side of the genome**

02:20 PM ***Chiara Lanzuolo, Istituto Nazionale di Genetica Molecolare, Milan, Italy*****: Chromatin conformation of muscle stem cells in physiological and pathological muscular aging**

02:40 PM ***Isabella Scionti, INMG, Lyon, France:*** *To be announced*

03:00 PM ***Piera Smeriglio, Sorbonne Université, Paris, France:*** *To be announced*

***03:20 PM Giuseppina Caretti et al., Milan University, Italy:* Epigenetic targeting of BET proteins rewire metabolism in the aged skeletal muscle**

03:40 PM Silvere M. van der Maarel, Leiden University Medical Center, the Netherlands, *To be announced*

04:00 PM Break

04:00 PM SESSION III: Adaptations in Aging: from molecules to functioning
Amber L. Pond, Rosanna Piccirillo, Chairs

***04:00 PM Amber L. Pond, Southern Illinois University School of Medicine in Carbondale, IL, USA:* The HERG K+ channel increases intracellular calcium in myotubes by modulation of Calsequestrin**

***04:20 PM Rosanna Piccirillo et al., IRCCS - "Mario Negri", Milan, Italy*: MyoRep: a novel reporter system to detect early muscle atrophy in vivo**

04.40 PM Paolo Grumati, Telethon Institute of Genetics and Medicine, Naples, Italy: *To be announced*

***05:00 PM 05:40 PM Bert Blaauw, University of Padua, Italy*: The role of Akt-mTORC1 signaling in regulating muscle mass and function**

***05:20 PM Hans Hoppeler, University of Bern, Switzerland:* Fascia, Facts and Fantasies**

***05:40 PM Stefano Schiaffino, University of Padua, Italy:* Who is Terje Lømo, a 88-year-YOUNG scientist still fully active!**

***06:00 PM Lecture of Terje Lømo, University of Oslo, Norway:* Body temperature regulation by muscle tone**

06:45 PM Complimentary Bus Transfer to Hotel Petrarca, Thermae of Euganean Hills (Padua), Italy

08:00 PM *Dinner in Hotel Petrarca*

THURSDAY March 30, 2023

Conference Hall, Hotel Petrarca, Thermae of Euganean Hills (Padua) Italy

09:00 AM SESSION IV: Acquired muscle diseases: FES managements
Ines Bersch-Porada, Helmut Kern, Chairs

09:00 AM Ines Bersch-Porada, Functional Electrical Stimulation Center, Notwill, Switzerland: Electrical stimulation in lower motoneuron lesions , from scientific evidence to clinical practice – a successful transition

09:20 AM Winfried Mayr, University of Vienna, Austria: To Be Announced

09:40 AM Ugo Carraro, A&C M-C Foundation, Padua, Italy: Main findings of my 40 years of research on electrical stimulation of permanently denervated muscles summarized in 20 slides

10:00 AM Giovanna Albertin, University of Padua, Italy: Skin improvements by home-based DDM FES for permanent peripheral denervation of Conus-Cauda in SCI patients

10:20 AM To Be Announced

10:40 AM Giovanni Pegoraro, Neuromuscular Rehabilitation Center, Fondazione Borghi, Varese, Italy: Hand NMES in post-COVID syndrome

11:00 AM Lecture of H. Lee Sweeney, University of Florida, USA: Improving upon AAV.micro-dystrophin gene therapy for DMD

11:40 AM SESSION V: Genetic muscle diseases - Elisabeth R. Barton, H. Lee Sweeney, Chairs

11:40 AM Kay Ohlendieck, Maynooth University, Maynooth, Co. Kildare, Ireland: Proteomic profiling of reactive myofibrosis in the aged and dystrophic diaphragm

12:00 AM Elisabeth R. Barton, University of Florida, USA: To Be Announced

12:20 AM Marina Bouchè DAHFM, Sapienza University of Rome, Italy: To unravel immune response in Duchenne Muscular Dystrophy

12:40 AM Philippe Perrin et al., Development, Adaptation and Handicap, University of Lorraine, France: Postural control impairments in Fabry disease

13:00 PM Lunch

02:20 PM - SESSION VIa: Twenty Years of AIM *– Daniela Tavian, Corrado Angelini, Chairs*

***02:20 PM Giovanna Lattanzi, IRCCS Rizzoli Orthopedic Institute, Bologna, Italy*: Laminopathies**

***02:40 PM Gabriele Siciliano, University of Pisa, Italy*: Phenotype variabilities of laminopathies**

***03:00 PM Gulia Ricci, Gabriele Siciliano et al., University of Pisa, Italy:* New avenues for treatment of facioscapulohumeral MD (FSHD)**

03:20 PM Giovanna Cenacchi et al., Alma Mater Studiorum University of Bologna, Italy: LGMD D2 TNPO3-Related: Clinical Spectrum

03:40 PM Giovanna Cenacchi et al., Alma Mater Studiorum University of Bologna, Italy: LGMD D2 Pathogenetic Mechanism

04:00 PM Break

04:20 PM SESSION VIb: Twenty Years of AIM *– Corrado Angelini, Daniela Tavian, Chairs*

Conference Hall, Hotel Petrarca, Thermae of Euganean Hills (Padua) Italy

***04.20 PM Massimiliano Filosto, NeMO-Brescia Clinical Center for Neuromuscular Diseases, University of Brescia, Italy*: Clinical and genetic characterization of a novel NLSDM patient**

***04:40 PM Elena Pennisi, Sara Missaglia, Daniela Tavian et al., Milan, Italy*: Exploring triheptanoin as treatment for neutral lipid storage disease with myopathy**

***05:00 PM Ester Tommasini, Daniela Tavian et al., Milan, Italy*: Irisin and sarcopenia: salivary irisin is induced by strenuous exercise and correlates with circulating irisin**

***05:20 PM Giovanna Cenacchi et al., Alma Mater Studiorum University of Bologna, Bologna, Italy*: LGMD D2 Pathogenetic Mechanism**

05:40 PM PatriziaProia, Giuseppe Messina, University of Palermo, Italy: *To Be Announced*

05:50 PM Angelo Iovane et al., University of Palermo, Italy: *To Be Announced*

***06:00 PM Alessia Geremia, Sabrina Zorzato, Roberta Sartori, Martina Baraldo, Leonardo Nogara, Hendrik Nolte, Jorge L. Ruas, Bert Blaauw:* Activation of muscle-specific Akt1 reverts cancer-dependent muscle wasting and reduces tumor mass**

07:30 PM Dinner

FRIDAY March 31, 2023

Conference Hall, Hotel Petrarca, Thermae of Euganean Hills (Padua) Italy

09:00 AM SESSION VII: Senescence & Rejuvenation

Nathan K. LeBrasseur, Christiaan Leeuwenburgh, Chairs

***09:00 AM Nathan K. LeBrasseur, Mayo Clinic, Rochester, MN, USA*: Cellular senescence as a driver of skeletal muscle aging**

***09:20 AM David Hood, York University, Canada*: Impact of age and sex on lysosomes and mitophagy during muscle use and disuse**

***09:40 AM Christiaan Leeuwenburgh, University of Florida, Gainesville, FL, USA:* Inflammation,**

mitochondrial dysfunction senescense in skeletal muscle with aging and in peripheral artery disease

10:00 AM ***Maira Rossi, University of Pavia, Italy:*** **Nitrate supplementation promotes an anabolic response and attenuates neuromuscular alterations in 24-months old male mice**

10.40 ***Break***

ROOM B, Hotel Petrarca, Thermae of Euganean Hills (Padua) Italy

09:30 AM Practical Course on functional analysis of the stomatognathic system

Claudia Dellavia, Riccardo Rosati, Chairs

9.30-11.00: Riccardo Rosati, Milan, Italy: **Instrumental evaluations of the stomatognathic apparatus: static tests**

11.00-11.15 Break

11.15-12.30: Claudia Dellavia, Milan, Italy**: Instrumental evaluations of the stomatognathic apparatus: dynamic tests,**

For dentists who want to review/expand the functional analysis protocols of the stomatognathic system developed at the Laboratory of Functional Anatomy of the Stomatognathic Apparatus (LAFAS) of the University of Milan, Italy

Conference Hall, Hotel Petrarca, Thermae of Euganean Hills (Padua) Italy

10:20 AM SESSION VIII: Muscle Fascia, biology and pathology

Carla Stecco, Alessandro Martini, Chairs

10:20 AM Carla Stecco, University of Padua, Italy: **Fascia and aging**

10:40 AM Alessandro Martini, University of Padua, Italy**: Tensor Tympani and Stapedius: two unknown muscles**

11:00 AM Carmelo Pirri, University of Padua, Ital**y: Fascia and Ultra Sounds**

11:20 AM Caterina Fede, University of Padua, Ital**y: How sex hormones can affect the fasciae: Implication for pain**

11:40 AM Giovanna Albertin, University of Padua, Italy**: Lymphatic drainage of the subcutaneous tissue**

12:00 AM Lucia Petrelli, University of Padua, Italy**: Innervation and vascularization of the superficial fascia**

12:20 AM Lorenza Bonaldi, University of Padua, Italy: **Deep learning applied to fascial alteration**

12:40 AM Lunch

02:00 PM SESSION IX: Non-invasive Assessments in Myology *- Paolo Gargiulo, Ugo Carraro, Chairs*

02:00 PM Riccardo Forni, Paolo Gargiulo, University of Rejkyavik, Iceland: **3D Printing and radiodensity characterization of cardiac tissue: virtual histology and age dependency**

02:20 PM D Jacob, et al., University of Rejkyavik, Iceland: **Towards defining biomarkers to evaluate concussions using virtual reality and a moving platform (BioVRSea)**

02:40 PM Valentina Betti, Halldór Jónsson Jr, Luca Cristofolini, Magnús Kjártan Gíslason, Paolo Gargiulo, University of Rejkyavik, Iceland**: In silico 3d approach to evaluate bone remodelling after total hip arthroplasty: a six years longitudinal study**

03:00 PM Federica Ciliberti, Halldór Jónsson Jr, Paolo Gargiulo, University of Rejkyavik,

Iceland: **Novel strategies for cartilage assessment, interplay between bone and muscle**

03:20 PM ***Carlo Ricciardi, Alfonso Maria Ponsiglione, University of Federico II, Naples , Italy:*** *Interplay between the age and the asymmetry of NTRA in elderly people".*

03:40 PM ***Rosanna Piccirillo et al., IRCCS - "Mario Negri", Milan, Italy:*** **A novel reporter technology, MyoRep, to follow in vivo skeletal muscle wasting**

04:00 PM ***Ettore Rocchi, et al. University of Urbino Carlo Bo, Urbino, Italy:*** **Exploring myofibrillar alignement in muscular tissue**

04:20 PM ***Break***

Conference Hall, Hotel Petrarca, Thermae of Euganean Hills (Padua) Italy

04:30 PM SESSION X: Muscle Rehabilitation in Dentistry, *Riccardo Rosati, Elena P. Ivanova, Chairs*

04:30 PM ***Gaia Pellegrini, University of Milan, Italy:*** **Standardised protocols for sEMG of the masticatory muscles in oral rehabilitation**

04:45 PM ***Elena P. Ivanova, Rehabilitation and Balneology Center, Moscow, Russia:*** **Innovative methods of full dental rehabilitation**

05:00 PM ***Francesca Ferrante, University of Pavia, Italy*: Facial morphology and masticatory muscle rectruitment**

05:15 PM ***Roberto Rongo, University of Naples Federico II, Italy*: Masticatory muscles pain management**

05:30 PM ***Maria Dimova, University of Bulgaria: Bulgaria*: Diagnosis and treatment of cranio-mandibular disorders**

05:45 PM ***Mauricio Gonzales Balu, Mexico: Centro Ortodoncico Especializado, Mexico City, Mexico :*** **Definite Orthodontic treatment for patients with Craniomandibular Dysfunction and/or TMD**

06:00 PM ***Bazar Amarsaikhan, Mongolia:*** **Chewing hard food and its importance for general health**

06:15 PM ***Avtandil Bakradze, Tbilisi State Medical University, Tbilisi, Georgia:*** **Peculiarities of the chewing muscles electrophysiological activity in mouth breathing individuals**

06.30 PM ***Francesco Mantia, et al., University of Palermo, Italy*: Intra-articular ultrasound-guided injection with Hyaluronic Acid and corticosteroid in retrodiscal tissue for TMD**

06.45 PM ***Francesco Mantia, et al., University of Palermo, Italy*: Effects of Platelet-Rich-Plasma injection in association with therapeutic exercise in the management of Medial Epicondylitis**

07:30 PM *Dinner*

09:00 PM ***AFTER DINNER ACTIVITIES***

SATURDAY April 1, 2023

Conference Hall, Hotel Petrarca, Thermae of Euganean Hills (Padua) Italy

09:00 AM Session XI: LBI workshop on muscle rehabilitation - from mouse to elderly

Sandra Zampieri, Feliciano Protasi, Chairs

09:00 AM ***Feliciano Protasi, University of Chieti, Italy:*** **Mimicking disuse and rehabiliation in a mouse model**

09:30 AM ***Antonio Musarò, University Sapienza of Rome, Italy*: Molecular biological basis and effects of immobility and training in young and aging.**

10:00 AM *Sandra Zampieri, University of Padua, Italy*: **C-Terminal Agrin Fragment as a biomarker of muscle wasting and weakness in aging and disuse**

10:30 AM *Break*

10:40 AM *Jan Cvecka, University of Bratislava, Slovakia*: **Exercise intervention in elderly: a novel system within the Centre of Active Aging in Bratislava**

11:10 AM *Nejc Sarabon, University of Primorska, Slovenia*: **Relationship between 24-hour movement behaviour and physical performance in older adults: A cross-sectional insight into the Centre of active ageing data**

11:40 AM *Stephan Loefler, Helmut Kern, LBI Rehabilitation Research, Vienna, Austria*: **AMB-REMOB – results of an early outpatient rehabilitation program**

12:10 AM *Vincent Grote, Michael Fischer, LBI Rehabilitation Research, Vienna, Austria:* **Outcomes of early rehabilitation in elderly patients**

12.40 AM *Lunch*

12:45 AM *Complimentary Transfer to Medical Hotel Ermitage (*restricted to 15 Attendees*)*

Medical Hotel Ermitage, Abano Thermae (Padua) Italy

01:00 PM *Working Lunch in Medical Hotel Ermitage*

01:30 PM ***Marco Maggia, Director of the Medical Hotel Ermitage***

Balneotherapy, Mud, Physiotherapies for Prevention & Rehabilitation in Medical Hotel Ermitage, a pioneering 15-year successful example

Conference Hall, Hotel Petrarca, Thermae of Euganean Hills (Padua) Italy

03:00 PM SESSION XII: European Medical Thermalism and the World Federation Hydrotherapy (FEMTEC) *- Marcus Coplin, Umberto Solimene, Chairs*

03:00 PM *Marcus Coplin, Balneology Association of North America, Naturopathic Healthcare, Pagosa Springs, Colorado, USA*: **A case study of balneotherapy in Fibromyalgia**

03:30 PM *Helmut Kern, LBI Rehabilitation Research, Vienna, Austria:* **Underwather physiotherapy after knee replacement**

04:00 PM *Umberto Solimene, Center Integrative Medicine, State University Milan, Italy:* **World Thermal Clusters**

04:30 PM *Maria Chiara Maccarone, Ugo Carraro, Stefano Masiero, University of Padua, Italy*: **Balneology and Health Resort Medicine and rehabilitation in the Euganean Hills Thermae: building the future**

05:00 PM *Daniele Coraci, et al. University of Padua, Italy:* **Technological transition of different rehabilitation approaches: challenges and answers**

05:20 PM *Andrey Rachin, Rehabilitation and Balneology Center, Moscow, Russia*: **Modern methods of neuro-rehabilitation**

05:40 PM *Irina A. Grishechkina, Rehabilitation and Balneology Center, Moscow, Russia:* **Outcomes of rehabilitation programs in patients with post-COVID-19 syndrome**

06:00 PM *Stefano Masiero, University of Padua, Italy:* **Final remarks**

06:30 PM ***Ugo Carraro: Adijo, Adiós, Arrivederci, Auf Wiedersehen, Au revoir, Bayartai, Do pobachennya, Do svidaniya, Dovizhdane, Goodbye, Tot ziens, Zbohom to the 2024 Padua Days on Muscle and Mobility Medicine***

07:30 PM *Dinner*

09:00 PM ***AFTER DINNER ACTIVITIES***

Chapter 12

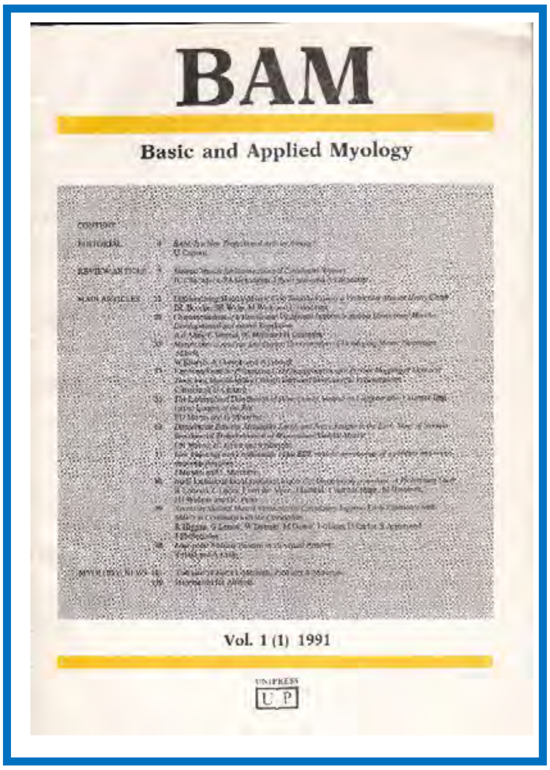
32 Years of European Journal of Translational Myology, 20 Years of the CIR-Myology of the University of Padua, Italy and 5 Years of the Armando and Carmela Mioni-Carraro Foundation for Translational Myology, Padua, Italy

12.1 Thirty-two years of Basic and Applied Myology (BAM), renamed in 2010 European Journal of Translational Myology (EJTM).

12.2 Twenty years of CIR-Myology of the University of Padova, Italy

12.3 Five years of the "Armando and Carmela Mioni-Carraro Foundation for Translational Myology (A&C M-C Foundation)" and EASY AGING

Chapter 12.1 Thirty-two years of Basic Applied Myology (BAM), renamed in 2010 European Journal of Translational Myology (EJTM).



Born in 1991 as a focused journal on skeletal muscle reports, *Basic Applied Myology* (BAM), from 2010 was re-titled *European Journal of Translational Myology* (EJTM), and mainwhile contents extended to *Mobility Medicine*, a relevant neologism to include the interdependencies of skeletal muscle biology, physiology and pathology with nervous system and metabolism of animal and human bodies, including prevention, management and rehabilitation from cradle to the inevitable decay of aging skeletal muscles.

For details see the EJTM Editorial: *Carraro U. EJTM3 is also covering Mobility and Medicine at large, an update. Eur J Transl Myol. 2018 Sep 17;28(3):7814. doi: 10.4081/ejtm.2018.7814. eCollection 2018 Jul 10.*

From 2014 the contents of the ***European Journal of Translational Myology (EJTM)*** *are listed in PubMed, a prestigeous data-base of the National Institue of Health, National Library of Medicine, 8600 Rockville Pike, Bethesda, MD 20894, USA. PubMed is a free resource supporting the search and retrieval of biomedical and life sciences literature with the aim of improving health–both globally and personally. The PubMed database contains*

more than 34 million citations and abstracts of biomedical literature. It does not include full text journal articles; however, links to the full text are often present when available from other sources, such as the publisher's website or PubMed Central (PMC). Available to the public online since 1996, PubMed was developed and is maintained by the National Center for Biotechnology Information (NCBI), at the U.S. National Library of Medicine (NLM), located at the National Institutes of Health (NIH) of Bethesda, USA. Citations in PubMed primarily stem from the biomedicine and health fields, and related disciplines such as life sciences, behavioral sciences, chemical sciences, and bioengineering. PubMed facilitates searching across several NLM literature resources: MEDLINE is the largest component of PubMed and consists primarily of citations from journals selected for MEDLINE; articles indexed with MeSH (Medical Subject Headings) and curated with funding, genetic, chemical and other metadata.
Citations for PubMed Central (PMC) articles make up the second largest component of PubMed. PMC is a full text archive that includes articles from journals reviewed and selected by NLM for archiving (current and historical), as well as individual articles collected for archiving in compliance with funder policies. The final component of PubMed is citations for books and some individual chapters available on Bookshelf. Bookshelf is a full text archive of books, reports, databases, and other documents related to biomedical, health, and life sciences.

Beside **PubMed** the articles of EJTM are listed in other scientific Data-Bases, specifically **SCOPUS** of Elsevier.com and **ESCI** of Clarivate.com
SCOPUS** is an abstract and citation database of peer-reviewed literature including scientific journals, books, and conference proceedings. Scopus provides a comprehensive overview of worldwide research output in the fields of science, technology, medicine, social sciences, and arts and humanities.* ***EJTM has a SCOPUS – CiteScore 2021 = 3.6
CLARIVATE's Impact Factor of 2022 EJTM will be released June 2023

Finally, I would like to thank the friends who have agreed to be included in the 2022 EJTM Advisory Board reported below. Some of them are very active, respond promptly to the request for advice on typescripts submitted and organize special issues. Others, overwhelmed with daily commitments, have been kind enough to spend their names to make sure EJTM is a serious scientific journal that deserves to be on the list of those that Clarivate will publish the 2022 Impact Factor next June 2023.
This is much more than I expected and probably deserve.

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Below are 395 titles of EJTM articles published from March 31, 2014 to date, excluding those with my name only, while typescripts in collaboration with my pupils and other international authors are included.
Clarivate will release in June 2023, once again after 20 years, the 2022 Impact Factor of EJTM. I hope it will be a memorable gift to EJTM Section Editors, Advisers, Authors and Readers and to me.

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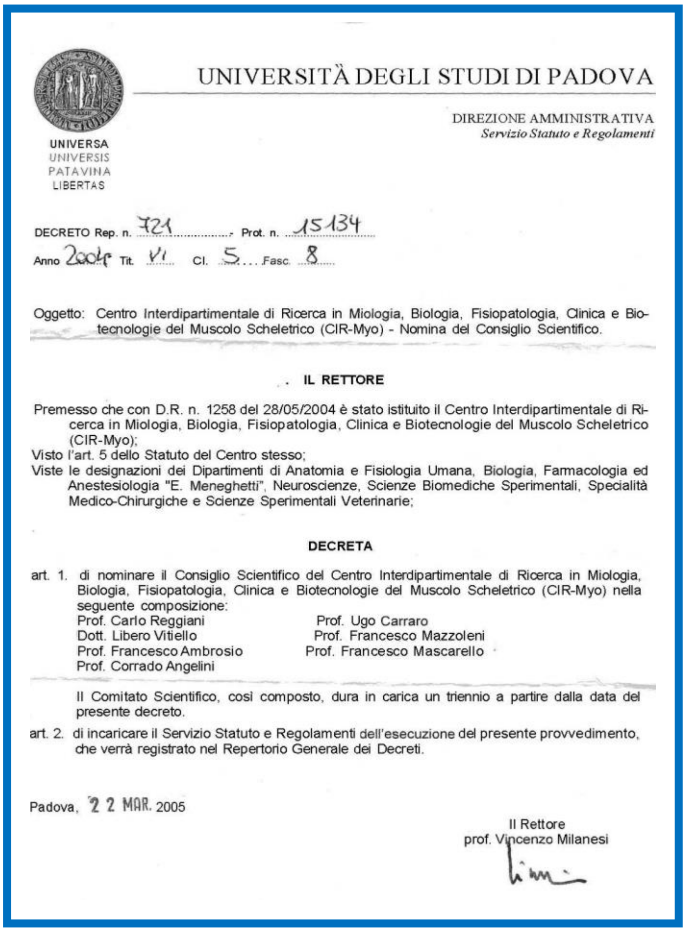
Chapter 12.2 Twenty years of CIR-Myology of the University of Padova, Italy

When Prof. Massimiliani Aloisi moved from the University of Modena to the University of Padua in the early 1960s, he brought with him some collaborators and the great financial support of a Center of the Italian National Research Council (CNR - National Research Council) . After some discussions with colleagues from the fledgling Biochemistry (formerly part of Physiology) and Neurology, the big money was split into three independent Centers. One of these (with the largest portfolio) has become the Center for Biology and Pathophysiology of the Skeletal Muscle (Center for Musclean Biology and Pathophysiology). With the Italian CNR support (and the personal support of an USA Telethon Grant), Prof. Aloisi equipped a traditional histopathology laboratory with an electron microscopy, centrifuges, ultracentrifuges, a laboratory for radioisotopic labeling of chemical compounds, a well-equipped photographic laboratory and a stabularium for small mammals.His most important decision was that five researchers and ten laboratory technicians were paid with a scholarship untill they became regular employees of the University of Padua, when the Italian government decided to invest in undergraduate public school and in university, including scientific research.
Abruptly around 1980, the golden age of Italy ended and a decreasing number of resources had to cover the growing needs of an expanding university system. Part of the solution to responding to the financial crisis has been to group several competing Centers into larger Departments. The prestige of the Skeletal Muscle Research Group was first diluted in a Department of Neuroscience, the next step was to provide resources through a large Department of Medicine of the Italian CNR. A tradition of high-level muscle research was at risk.



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DIREZIONE AMMINISTRATIVA
Servizio Statuto e Regolamenti

DECRETO Rep. n. 721 Prot. n. 15134
Anno 2004 Tit. VI Cl. 5 Fasc. 8

Oggetto: Centro Interdipartimentale di Ricerca in Miologia, Biologia, Fisiopatologia, Clinica e Biotecnologie del Muscolo Scheletrico (CIR-Myo) - Nomina del Consiglio Scientifico.

IL RETTORE

Premesso che con D.R. n. 1258 del 28/05/2004 è stato istituito il Centro Interdipartimentale di Ricerca in Miologia, Biologia, Fisiopatologia, Clinica e Biotecnologie del Muscolo Scheletrico (CIR-Myo);
Visto l'art. 5 dello Statuto del Centro stesso;
Viste le designazioni dei Dipartimenti di Anatomia e Fisiologia Umana, Biologia, Farmacologia ed Anestesiologia "E. Meneghetti", Neuroscienze, Scienze Biomediche Sperimentali, Specialità Medico-Chirurgiche e Scienze Sperimentali Veterinarie;

DECRETA

art. 1. di nominare il Consiglio Scientifico del Centro Interdipartimentale di Ricerca in Miologia, Biologia, Fisiopatologia, Clinica e Biotecnologie del Muscolo Scheletrico (CIR-Myo) nella seguente composizione:
Prof. Carlo Reggiani — Prof. Ugo Carraro
Dott. Libero Vitiello — Prof. Francesco Mazzoleni
Prof. Francesco Ambrosio — Prof. Francesco Mascarello
Prof. Corrado Angelini

Il Comitato Scientifico, così composto, dura in carica un triennio a partire dalla data del presente decreto.

art. 2. di incaricare il Servizio Statuto e Regolamenti dell'esecuzione del presente provvedimento, che verrà registrato nel Repertorio Generale dei Decreti.

Padova, 22 MAR. 2005

Il Rettore
prof. Vincenzo Milanesi

Fortunately, the University of Padua decided to organize its research activities also through self-financed Interdepartmental Centers for Multidisciplinary Research.
I took that opportunity and with a few interested colleagues of the Departments of Biomedical Sciences, Biology, Neurosciences and Rehabilitation, Special Surgery, in particular Plastic Surgery and Veterinary and Food Sciences (meat research), we started the process and finally obtained in 2004 the approval of the "Interdepartmental Center of Myology (CIR -MYO) of the University of Padua", officially recognized with the "D.R. n. 1258 of 28/05/2004".
Among the goals of CIR-MYO, the Statute enlists recruitment of

young researchers, support for organizing Seminars, Conferences and publications dedicated to Myology, in its broadest sense.
I was elected Director of CIR-MYO for two four-year terms, favoring contributions of basic scientists to medical and non-medical applications, introducing “Translational Myology” concept to stress that aim. Then Marco Sandri followed for the next five years. Now Prof. Marco Narici of the Physiology Section of the Department of Biomedical Sciences leads CIR-Myo.

A 60-year long tradition of Myology at the University of Padua is in good hands and CIR-Myo will continues to attract the colleagues, among others, specialists in Sports Sciences and from Pediatrics to Medicine of Aging.

12.3 Five years of the “Armando and Carmela Mioni - Carraro Foundation for Translational Myology (A&C M-C Foundation)” and EASY AGING

Almost ten years ago I was obliged to retire from the duties of an Associate Professor of General Pathology, but the University of Padova invented the title of “Senior Scholar” for those retired persons willing (but only after consent of the Council of a Department of the University of Padova) to continue to have good relations with ex-colleagues, maybe a desk generosusly provided by younger colleagues, the right to maintain and use the institutional email (in my case: ugo.carraro@unipd.it) and to teach without compensation as Special Expert, in particular in the medical and non-medical Specialization Schools. Indeed, I continue to teach General Pathology to Rehabilitation Specialists, looking for young doctors willing to collaborate in clinical reasearch trials. Though, I am also spending time accepting invitation to serve as referee for several muscle-rehabilitation-related journals and editing the European Journal of Translational Myology (EJTM), my wife accepted five years ago that I added to the list one more senil hobby: the “Armando and Carmela Mioni-Carraro Foundation for Translational Myology”.

My major excuse was to thanks my parents that allowed me to avoid my obbligations as a Doctor in two Hotels of the Thermae of Euganean Hills, Padua, Italy, but the thrue is that by the Foundation it would be easier to contact senior colleagues and test some tricks to delay the inavoidable decay of aging. To implement this plan I established also “Easy Aging” a club for seniors dedicated to organise visits to the monuments, museums and Special Art Events in Padova and surrounding cities, usually once a month on Thuesday afternoon. The meetings were also occasions to talk about the value and the possible modalities of physical activity for old and oledest older.

Of course, secondary reasons were to attract support for the same aims I cultivated as founder director of the CIR-MYO: i) attract money to cover grants for young researchers; ii) organise Conferences; iii) support publications of young researchers and editing of EJTM. The Covid-2019 pandemic has heavily interfered with my plans/dreams. I had to interrupt the meeting with senior colleagues and their families, but resuming and continuing those events and activities of Easy Aging and A&C M-C Foundation for Translational Myology are among my endless dreams.

Chapter 13
Inspirers & supporters

CHAPTER 13. Inspirers & Supporters
Gerta Vrbová, Clara Farnzini-Armstrong, Giorgio Fanò-Illic, Carlo Reggiani, Sergio Adamo, Zipora Yablonka-Reuveni, Terence Partridge, Ines Bersch-Porada, Christiaan Leeuwenburgh, Marco Narici, Gabriele Siciliano, Guglielmo Sorci

13.1. Gerta Vrbová

In Memoriam: Gerta Vrbová, May 2015

I meet Gerta for the first time in 1980, when she was visiting Prof. Massimiliano Aloisi at the Institute of General Pathology of the University of Padova, Italy. She was so kind to talk with a very young Ugo presenting to her is first first-name paper.

Our relations continued for the following decades, but were strengthened after we discovered (getting out from a night bus after a dinner in Central London) that she was living in Muswell Hill just 10 walking-minutes from the house of my son's family.

Sadly, Gerta's passed on October 2nd, 2020.

Gerta Sidonová-Vrbová, Trnava (Slovakia) November 28, 1926 – London (UK) October 2, 2020.

Gerta Vrbová was a key neuroscientists who for more than half-century contributed results and hypotheses on the mutual relations between motoneurons and skeletal muscle fibers, i.e., about differentiation and maintenance of the characteristics of the motoneurons and of the muscle fiber types of mammalian muscles. Implication and transfer of her personal conclusions to managements of neuromuscular disorders were her second main interest. Gerta Vrbová made a career out of studying nerves, though her own were made of steel. Twice she escaped brutal regimes: once by jumping from a window to flee the Nazis, and later by crossing from Czechoslovakia to Poland on foot with two young children in tow to escape the communists. Her troubles began in her home town of Trnava, in western Slovakia, in 1939. Jewish people faced discrimination and the 12-year-old was excluded from school. Rudi Vrba, an old school friend who was two years her senior, helped with her studies. She recalled a bicycle trip one summer day in 1939 with another friend, Marushka, who announced that they could no longer meet because Gerta was Jewish. We leave to others to stress the courage and determination of Gerta to achieve scientific results and to overcome tremendous personal obstacle along her long life (1,2). The issue 31 (1), 2021 of the European Journal of Translational Myology (EJTM) opens with the personal obituary authored by Dirk Pette who remember his lifelong collaboration with Gerta, describing the many molecular and metabolic events that occur by changing the pattern of activation of adult muscle fibers through neuromuscular low frequency electrical stimulation (3). To honor Gerta Vrbová and her scientific legacy, I add below my own memories.

I meet Gerta for the first time in 1980, when she was visiting Prof. Massimiliano Aloisi at the Institute of General Pathology of the University of Padova, Italy. As the last younger fellow of Prof. Aloisi, I was invited to present my first-name paper on long term denervatation of rat hemi-diaphragm (4)

She was very pleased to hear that in the six-month denervated hemi-diaphragm (a very mixed muscle) only fast-type Myosin Light Chains were present, a molecular result fully in agreement with the Gerta's seminal observation that denervation, depriving the slow muscle fibers of the continuous stimulation of the slow-type motoneurons, shortened the contraction time of the slow-type muscles (5-7).

Her warm attention to my observations was the main support to my commitment to continue those studies during the following decades, independently from other international researchers, but heartened from many concordant results of prestigious groups, including, beside Gerta and Dirk Pette (8-10), Stanley Salmons (11-13) and Terje Lomo (14-20).

Specifically, Terje had pioneered, often in collaboration with Stefano Schiaffino (21), the experimental model of low-frequency full-day electrical stimulation of denervated muscle in the rat model, to avoid the criticism that, stimulating the nerve, an antidromic adaptation of the motoneurons may occur before transformation of the innervated muscle fibers of the motor units.

Further strong evidence negating that option was convincingly presented by Terje Lømo during his Lecture at the 2014 Padua Muscle Days (22).

The criticism of course remained that my observations were restricted to the denervated hemi-diaphragm, a peculiar experimental model in which the denervated muscle fibers continued to be passively stretched by the innervated contralateral hemi-diaphragm. Encouraged by the suggestion of Gerta that that was further evidence that the denervated muscle fibers were able to respond to induced passive-stretch in absence of direct contact with the motoneuron terminals and by the accumulating evidence that the muscle fibers may develop and partly differentiate in vitro in the absence of neural contacts [see for a recent short account of the history of this topic (23,24) soon after the visit of Gerta to Padova, we firstly extended our observations to long term denervated and reinnervated leg muscles (25). Then in a conclusive paper we published in the Journal of Cell Biology [(1985),(26)] we demonstrated a substantial slow to fast transformation of the denervated rat hemi-diaphragm by electron microscopy analyses (evidence of severely decreased mitochondria, pathological features of membranes of sarcoplasmic reticulum (SR) and regeneration of muscle fibers), by single fibers analyses of myosin heavy chains (MHC) (evidence that MHC of fast type accumulate at the expense of the slow type) and by 2D SDS Gel electrophoresis of myosin light chains (MLC) and parvalbumin (again, clear

prevalence of the fast type characteristics).
With our surprise, we were able to analyze a large numbers of muscle fibers up to 16 months after phrenectomy (26). All those results indicated that, after reaching a severe atrophic status 3 months after denervation, all types of denervated muscle fibers: i) maintain a residual 10% mass; ii) resting fast fibers continue to show their features, while iii) previously slow fibers acquire partially or almost completely fast type molecular characteristics (26).
In 1986, under the influence of results of Terje Lømo, we developed an independent experimental model of electrical stimulation of denervated rat leg muscle, showing that the fast muscle fibers of the fast-type extensor digitorum longus (EDL) rat muscle, submitted to continuous slow-like electrostimulation, switch-on the genes of slow myosin in denervated fast-type muscle fibers (27) Thus, adult fast and slow skeletal muscles are composed of a large number of fibers with different physiological and biochemical properties that under neuronal control can respond in a plastic manner to a variety of stimuli.
Although muscle cells synthesize muscle-specific contractile proteins in the absence of motoneurons, after innervation the type of motoneuron controls the particular set of isoforms subsequently synthesized. However, agreement had not been reached on the mechanism, either chemotrophic or impulse-mediated, by which the nerve influences gene expression of the skeletal muscle fibers. In that study (27) we reported the effect on isomyosins of continuous, low-frequency (a protocol mimicking the discharge pattern of the slow motoneuron) direct electrical stimulation of a permanently denervated fast muscle, the extensor digitorum longus of adult rat. After several weeks, unlike sham-stimulated muscle, the stimulated muscle showed a dramatic increase of the slow myosin light and heavy chains. Myosin light chains were identified by two-dimensional gel electrophoresis. The slow myosin heavy chains were clearly distinguished from fast and embryonic types by one-dimensional sodium dodecyl sulfate-polyacrylamide gel electrophoresis and orthogonal peptide mapping. The myosin changes could be restricted to a portion of the muscle by the position of the stimulating electrodes (27).
Taking into account the morphologic appearance of the electrostimulated muscle and the large body of evidence demonstrating the absolute dependence of slow myosin on specific innervation, our observations indicated that at least the slow motoneuron influences the isomyosin genes' expression by the kind of activity it imposes on developing muscle fibers (27). I am still wondering if the protocol we used in Padova that was able to induce in the denervated fast-type EDL of rats high levels of expression of slow-type light and heavy chains found their rational in the fact that we increased the current duration of each impulse from 0.4 to 4.0 milliseconds (27). The relevance of this hypothesis will be clear if the readers will have the patience to read the Chapter dedicated to the hb(FES) of permanently denervated human muscles.
Meantime Gerta Vrbová and Stanley Salmons proposed the use of electrical stimulation to increase resistance to fatigue of skeletal muscles for different managements of neuromuscular disorders, among others Duchenne Muscular Dystrophy (28,29) and for the support of insufficient circulation by Cardiomyoplasty (30) or of sphincter muscles (31) Meanwhile, electrical stimulation of the diaphragm has become an accepted clinical approach for patients with respiratory paralysis and intact phrenic nerves. Indeed, continuous simultaneous pacing of both hemi diaphragms with low-frequency stimulation and a slow respiratory rate is a satisfactory method of providing full-time ventilation

support (32-36).
In collaboration with cardiac surgeons and engeneers of the University of Bologna (Italy), we have been involved in a ten-year research project on cardiomyoplasty, testing the concept of dynamic cardiomyoplasty on demand. We were inspired by the differential effects on the contractile properties, population of fibers, myosin light chains and enzymes of energy metabolism of different periods of intermittent or continuous electrical stimulation of fast-twitch muscles [(see Gerta Vrbová and Dirk Pette (37)]. Both in normal sheep (38) and in patients suffering with chronic heart failure, the latissimus dorsi responded to daily intermittent electrical stimulation reaching intermediate contractile characteristics during its fast to slow transition induced be the "Demand (intermittent) stimulation protocol (39). The partially maintained fast type contractions of the patient muscle wrapped around the falling heart allowed to synchronize the pace-maker-induced tetanic contractions with the heart systole, avoiding interference with the diastolic function. Specifically, during assisted systolic contractions cardiac ejection fractions of heart failure patients were increased (40-42)
Always supported by interest and suggestions of Gerta, from 2000, in collaboration with Prof. Helmut Kern of Vienna (Austria) we were able to show in a study supported by the EU Commission [(RISE - Use of electrical stimulation to restore standing in paraplegics with long-term denervated degenerated muscles (QLG5-CT-2001-02191)] that a home-base protocol using long currents (up to 150 milliseconds) is able to reverse severe atrophy of permanent denervated human muscles, up to a level to allow stimulation-induced standing and pacing-in-place exercise.43-49
For an example see the supplementary material in Kern H, Carraro U (50): Home-based Functional Electrical Stimulation (hbFES) assisted stand-up exercise (51)
One of the effects of spinal cord injury (SCI) is rapid loss of contractile force and muscle mass, but atrophy of leg muscles is particularly severe when the injury destroys the soma of the lower motoneurons and, hence, the contacts between skeletal muscle fibers and motoneurons are permanently lost. Within weeks after SCI, muscles become unable to sustain tension during tetanic contractions induced by electrical stimulation (52-54) Within months after a complete injury of the conus medullaris and cauda equina, the muscles are no longer excitable by commercially available electrical stimulators (55) This is because they have undergone severe disorganization of contractile elements (i.e., of the myofibrils) and of the excitation-contraction coupling (ECC) apparatuses. Finally, after several years of LMN denervation, human muscle fibers are almost completely replaced by adipose and fibrous tissues (44-46) This severe degeneration of muscle tissue does not occur in patients with upper motoneuron lesions even 20 years after thoracic-level SCI (56) To substantiate the functional and molecular mechanisms that allow muscle fibers to survive long-term denervation, we meantime performed experiments in a rat model of long-term denervation by analyses not possible in humans for obvious ethical concerns. The results are summarized in the abstract of a paper published by Squecco R, et al. (2009) (57).
To define the time course and potential effects of electrical stimulation on permanently denervated muscles, we evaluated ECC of rat leg muscles during progression to long-term denervation by ultrastructural analysis, specific binding to dihydropyridine receptors, ryanodine receptor 1 (RYR-1), Ca2+ channels and extrusion Ca2+ pumps, gene transcription and translation of Ca2+-handling proteins, in vitro mechanical properties and electrophysiological analyses of sarcolemmal passive properties and L-type Ca2+

current (ICa) parameters. We found that in response to long-term denervation: i) isolated muscle that is unable to twitch in vitro by electrical stimulation has very small myofibers but may show a slow caffeine contracture; ii) only roughly half of the muscle fibers with voltage-dependent Ca^{2+} channel activity are able to contract; iii) the ECC mechanisms are still present and, in part, functional; iv) ECC-related gene expression is upregulated; and v) at any time point, there are muscle fibers that are more resistant than others to denervation atrophy and disorganization of the ECC apparatus. These results support the hypothesis that prolonged "resting [Ca^{2+}]" may drive progression of muscle atrophy to degeneration and that electrical stimulation-induced [Ca^{2+}] modulation may mimic the lost nerve influence, playing a key role in modifying the gene expression of denervated muscle. Hence, these data provide a molecular explanation for the muscle recovery that occurs in RISE SCI patients in response to the rehabilitation strategies developed on the grounds of empirical clinical observations (44-51).
Gerta maintained her interest for the myology activities of the Interdepartmental Research Center of Myology of the University of Padova (CIR-Myo), even after she was more than 85 years old.
She joined several times the PaduaMuscleDays, a meeting mainly devoted to translational research for skeletal muscle biology, management and rehabilitation.
Last time it was in 2017, when she went together with Dirk Pette. During that meeting she accepted also to be interviewed on the importance for old people to stay physically and mentally active.
She was, indeed, a witness (and herself a strong evidence, being in her ninety years) of the value of a very active life for the oldest olds.
Readers may follow her advices at the YOUTUBE link:
https://www.youtube.com/watch?v=NJ9BPLquPWw (58).

During her career Gerta Vrbová published more than 270 scientific papers, but her main role was to inspire a generation of successful researchers. Some were her postdoctoral fellows Maggie Lowrie (59), Angela Connold (60), Linda Greensmith (61), Roberto Naverrete (62), Antal Nógrádi (63), and Katarzyna Sieradzan (64), but independent scientists, among which I mention researchers studying nerve regeneration after partial or complete nerve injuries [(Tessa Gordon, Canada)(65)], electrical stimulation and muscle plasticity [(Dirk Pette, Germany (3,8,9,66), electrical stimulation of denervated muscle in animal models, e.g., Terje Lomo, Norway (67) and myself in Italy, working in both animal models and humans (68) and finally aging human muscles [Helmut Kern, Austria (69)] Many of her pupils were brilliant enough to continue independent scientific careers and make major contributions to the fields of neuron diseases and injury, including amyotrophic lateral sclerosis and spinal muscular atrophy [Linda Greensmith in London 61)] Parkinson's, Huntington's and Pompe diseases and epilepsy [Katarzyna Sieradzan in Bristol (64)] ventral root avulsion [Antal N Nógrádi in Szeged, Hungary (63,70)] and in the field of locomotion [Urszula Slawinska in Warsaw, Poland) (1)]
Her medical background explains her interest in translating experimental results into possible treatments for childhood genetic diseases of muscular dystrophy and spinal muscular atrophy, in collaboration with neurologists, Victor Dubowitz in London, UK (72), Milan Dimitrijevic in Houston, USA (73) and Irena Hausmanova-Petrusewicz in Warsaw, Poland (74)
Most of the latest opportunities found grounds on just two of her publications that

inspired her a working hypothesis that changed the perspectives of interactions between skeletal muscle fibers and motoneurons, starting 50 years of studies still in need of further investigations.

As is often the case in science and even more in translational research, there are now more open questions and hypotheses than before. Firm conclusions for some of the above discussed topics remain open to further researches, worth of significant founding by international sponsors. What is certain is that Gerta's legacy remains among the key preliminary results for supporting those grant applications.

More information on Gerta’s legacy and also on her personal experience of the Holocaust and its aftermath in Czechoslovakia may be found in Pette D. The significance of Gerta Vrbová's low-frequency stimulation experiment. Eur J Transl Myol. 31 (1): 9585, 2021 doi: 10.4081/ejtm. 2021.9585 (75) and in her Obituary published by the Journal of Physiology (London): Tessa Gordon and Ugo Carraro. IN MEMORIAM A stimulating life and career – an obituary for Professor Gerta Vrbová. J Physiol 599.10 (2021) pp 2763–2767 2763 (76).

I will never forget Gerta’s friendship and support.

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75 Pette D. The significance of Gerta Vrbová's low-frequency stimulation experiment. Eur J Transl Myol. 31 (1): 9585, 2021 doi: 10.4081/ejtm. 2021.9585 (75)
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CHAPTER 13. Inspirers & Supporters

13.2. Clara Farnzini-Armstrong

Clara Franzini-Armstrong 2020

I had the honor and the good fortune to meet Clara Franzini-Armstrongat the University of Chieti, Italy on occasion of a visit to her students Feliciano Protasi and Simona Boncompagni.
She was kind enough to show interest in contributions of Feliciano and Simona to the study of long-term denervated muscles undergoing home electrical stimulation according to the Vienna protocol. She appeared surprised when I reminded that her first article was on electron microscopy of denervated muscles (see below).
Perhaps this attracted her to delve into the results of our research, and she was kind enough to accept invitation to being listed as a BAM / EJTM Advisor. Then she contributed papers to the journal and edited in 2015 an EJTM Issue on:

3D Structure of the Dihydropyridine Receptor of Skeletal Muscle.

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2. Samsó M. 3D Structure of the Dihydropyridine Receptor of Skeletal Muscle.Eur J Transl Myol. 2015 Jan 7;25(1):4840. doi: 10.4081/ejtm.2015.4840. eCollection 2015 Jan 7.
3. Wagenknecht T, Hsieh C, Marko M. Skeletal Muscle Triad Junction Ultrastructure by Focused-Ion-Beam Milling of Muscle and Cryo-Electron Tomography. Eur J Transl Myol. 2015 Jan 15;25(1):4823. doi: 10.4081/ejtm.2015.4823. eCollection 2015 Jan 7.
4. Baker MR, Fan G, Serysheva II. Single-Particle Cryo-EM of the Ryanodine Receptor Channel in an Aqueous Environment.Eur J Transl Myol. 2015 Jan 12;25(1):4803. doi: 10.4081/ejtm.2015.4803. eCollection 2015 Jan 7.
5. Jayasinghe ID, Clowsley AH, Munro M, Hou Y, Crossman DJ, Soeller C. Revealing T-Tubules in Striated Muscle with New Optical Super-Resolution Microscopy Techniquess. Eur J Transl Myol. 2014 Dec 24;25(1):4747. doi: 10.4081/ejtm.2015.4747. eCollection 2015 Jan 7.
6. Wagenknecht T, Hsieh C, Marko M. Skeletal muscle triad junction ultrastructure by Focused-Ion-Beam milling of muscle and Cryo-Electron Tomography. Eur J Transl Myol. 2015;25(1):49-56. doi: 10.4081/ejtm.2015.4823.
7. Baker MR, Fan G, Serysheva II. Single-particle cryo-EM of the ryanodine receptor channel in an aqueous environment.Eur J Transl Myol. 2015;25(1):35-48. doi: 10.4081/ejtm.2015.4803.

I have no doubts that her contributions to the Journal were decisive for convincing PubMed to include in its prestigious database the papers of EJTM.
https://pubmed.ncbi.nlm.nih.gov/?term=Eur.+J.+Transl.+Myol.&sort=date&size=200

Directly, or indirectly through her pupils Feliciano Protasi and Simona Boncompagni, Clara Franzini-Armstrong inspired me and will inspire my future dreams.

Curriculum of Clara Franzini-Armstrong

Education 1956-60 Laurea, Biological Sciences, University of Pisa, Italy

Post-graduate training and fellowship appointments

1960-61 Perfezionanda, Scuola Normale Superiore, Pisa. Italy
1961-63 Post-doctoral fellow, Biological Laboratories (advisor: Keith R. Porter)
1963-64 Research Assistant, NIH, NIAMD (advisor: Richard J. Podolsky)
1964-66 Hon. Research Assistant, University College, London. (advisor: Andrew F. Huxley)

Appointments

1960-62 Assistant Professor in Pathology, University of Pisa, Italy
1967-69 Research Associate, Associate in Physiology, Duke Univ. Durham, NC
1969-72 Associate in Physiology, University of Rochester, Rochester, NY
1972-75 Assistant Professor in Physiology, Univ. of Rochester, Rochester, NY
1975-81 Associate Professor in Anatomy, University of Pennsylvania, Philadelphia PA
1981-2007 Professor in Cell Developmental Biology (formerly Anatomy), University of Pennsylvania, Philadelphia PA.
1990-2006 Director, Medical Histology Course
2000-present Member, Collegio Docenti del Dottorato in Fisiopatologia del Muscolo, Universita' G. D'Annunzio, Chieti Italy
2007-present Professor Emerita/Associate dept. Cell Developmental Biology, University of Pennsylvania, Philadelphia PA.

Awards and Honors

1956-60 Fellow, Scuola Normale Superiore, Pisa, Italy
1960-61 Post-doctoral fellow, Scuola Normale Superiore, Pisa
1989 K.S. Cole Award, Biophysical Soc. (with Dr. W.K.Chandler)
1995- Member, National Academy of Sciences, USA
1997 Honorary MD, University of Pisa, Italy
2001- Foreign Member, Royal Society, London, UK
2003 NIH MERIT Award
2005- Member, European Academy of Sciences
2007 Founder's Award, Biophysical Society

2011 Foreign Member, Accademia Nazionale dei Lincei, Roma, Italy

Books

Engel, A.G. and Franzini-Armstrong, C. Eds. Myology, second Edition. Mc Graw Hill, 1994.
Engel, A.G. and Franzini-Armstrong, C. Eds. Myology III Edition. McGrawHill NY, 2004.

Publication of refereed articles

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7. Franzini-Armstrong, C. Natural variability in the length of thin and thick filaments in single fibers from a crab Portunus depurator. J. Cell Sci. 6:559-592, 1970.
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11. Franzini-Armstrong, C. Studies of the triad: III Structure of the junction in fast twitch fiber. Tissue and Cell 4:469- 478, l972.
12. Franzini-Armstrong, C. Studies of the triad: IV Structure of the junction in frog slow fibers. J. Cell Biol. 56:120-128, l973.
13. Franzini-Armstrong, C. The structure of a simple Z line. J. Cell Biol. 58:630-642, l973.
14. Franzini-Armstrong, C., R. Venosa and P. Horowicz. Morphology and accessibility of the "transverse" tubular system in frog sartorius muscle after glycerol treatment. J. Membr. Biol. 14:197-212, 1973.
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16. Franzini-Armstrong, C., I. Landmesser and G. Pilar. The size and shape of transverse tubule openings in frog twitch muscle fibers. J. Cell Biol. 64:493-497, l975.
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CHAPTER 13. Inspirers & Supporters

13.3. Giorgio Fanò-Illic

2010 Giorgio Fanò-Illic 2022

I meet for the first time Giorgio, a Physiologist from Perugia, but at that time Full Professor of Physiology at the University of Chieti, Italy during the 2003 Meeting of the *Associazione Italiana di Miologia* (AIM) in Torino, Italy, where Italian Neurologists, Pediatricians, Geneticists and Basic Scientists caring and studying genetic muscle diseases were gatering for their Annual Meeting.

At the end of a long afternoon of scientific reports, Giorgio had the opportunity to present a proposal for a new Scientific Consortium: The Interuniversity Institute of Myology (IIM), with the hope to extend the role of Multidisciplinary Basic Scientists and alltogether find recognition and Institutonal support from the Italian Government and others. I was among the few people present that supported his proposal. Despite the minimal interest of Clinical Colleagues, his proposal during the next few months was accepted by some Italian Universities and during a Meeting organized by Giorgio in Chieti and the near National Park of Central Italy the IIM was born and officially recognized.

As one of the hentusiast supporter, I organized together with Giorgio Fanò the First Meeting of the Interuniversity Institute of Myology in Montegrotto Terme - Padova (Italy), October 16 – 18, 2004 at the Continental Hotel, Via Neroniane, 8 - 35036 Montegrotto Terme (Padova).

With our surprise, the Meeting attracted many more Colleagues than expected and was a very successful event as the following Program testify. The IIM is still alive and during succcessive years allowed to start and/or strenghten collaborations among Italian and International ”Myologists”, and the word itself was accepted.

From the EJTM paper celebrating ten years of IIM I add here two images. In the first, interested readers may find himself 10-year younger.

Interuniversity Institute of Myology (IIM) European Journal Translational Myology - Basic Applied Myology 2013; 23(4): 199-203 - Fig 2. The foto group.

European Journal Translational Myology - Basic Applied Myology 2013; 23(4): 199-203 - Figure 4.

Navigating the Tyrrenian sea toward Palermo and Stromboli for the prerelease of the IIM's birth, two of the future opinion leaders: Antonio Musarò (on the left) and Ugo Carraro discuss how to kill the "boss-in-charge" Giorgio Fanò-Illic

After the First Meeting (see below), often the Proceedings of the Annual IIM Meetings were printed in the European Journal of Translational Myology.
Recent examples are:

1. Sorci C, Gabellini D. Report and Abstracts of the 17th Meeting of IIM, the Interuniversity Institute of Myology: Virtual meeting, October 16-18, 2020. Eur J Transl Myol. 2020; 30 (4), 9485. doi: 10.4081/ejtm.2020.9485.
2. Gabellini D, Musarò A 16th Meeting of the Interuniversity Institute of Myology (IIM) - Assisi (Italy), October 17-20, 2019: Foreword, Program and Abstracts.. Eur J Transl Myol. 2020 Sep 15;30(3):9345. doi: 10.4081/ejtm.2020.9345. eCollection 2020 Sep 30.
3. Gabellini D, Musarò A. Report on Abstracts of the 15th Meeting of IIM, the Interuniversity Institute of Myology - Assisi (Italy), October 11-14, 2018. Eur J Transl Myol. 2018 Nov 30;28(4):7957. doi: 10.4081/ejtm.2018.7957. eCollection 2018 Nov 2.

Hopefully also the 2022 18th Meeting of IIM will appears in the European Journal of Translational Myology 32 (4), 2022.

First Meeting of the Interuniversity Institute of Myology

Montegrotto Terme - Padova (Italy), October 16 – 18, 2004
Continental Hotel Terme
Via Neroniane, 8 - 35036 Montegrotto Terme (Padova)

Saturday October 16, 2004

14.30 Welcome Address, G Fanò and U Carraro

STRUCTURAL, FUNCTIONAL AND REGULATORY MUSCLE MOLECULES
Chair: P Bruni and C Reggiani

14.45 IDENTIFICATION OF TWO SITES IN OBSCURIN THAT MEDIATE THE INTERACTION WITH ANKYRIN 1.5 Armani A, Galli S, Sorrentino V

15.00 CROSSBRIDGE PROPERTIES STUDIED BY FAST STRETCHES IN ACTIVATED FROG MUSCLE FIBRES Colombini B, Bagni MA, Cecchi G

15.15 MYOSIN ORIENTATION IN SKELETAL MUSCLE REVEALED BY X-RAY DIFFRACTION STUDIES DURING SARCOMERE LENGTH OSCILLATIONS Colombini B, Griffiths PJ, Bagni MA, Amenitsch H, Bernstorff S, Ashley CC, Cecchi G

15.30 THE MOLECULAR MOTOR OF MUSCLE STUDIED BY X-RAY INTERFERENCE Reconditi M, Linari M, Lucii L, Piazzesi G, Stewart A, Sun Y-B, Narayanan T, Irving T, Irving M, Lombardi V

15.45 MECHANICAL AND KINETIC PROPERTIES OF PURE ISOFORMS OF SKELETAL MYOSIN FRACTION S1 STUD- IED BY A SINGLE MOLECULE MECHANICAL APPROACH Capitanio M, Canepari M, Maffei M, Cicchi R, Pavone FS, Bottinelli R

16.00 ENDOGENOUS TROPONIN COMPLEX REPLACEMENT IN SINGLE SKELETAL AND CARDIAC MYOFIBRILS Belus A, Piroddi N, Scellini B, Tesi C, Poggesi C

16.15 THE IK-B-HOMOLOGUE CACTUS IS NECESSARY FOR NORMAL NEUROMUSCULAR FUNCTION IN DROSO- PHILA MELANOGASTER Peron S, Beramendi A, Megighian A, Reggiani C, Cantera R

16.30 REGULATION OF GLYCOGEN SYNTHASE BY SARCOPLASMIC RETICULUM-BOUND CAMKII IN RABBIT FAST-TWITCH SKELETAL MUSCLE Sacchetto R, Bovo E, Damiani E

16.45 TRANSFER OF PLASMID DNA AND OLIGONUCLEOTIDES INTO SKELETAL MUSCLE BY MEANS OF CATI- ONIC LIPID-BASED VECTORS Ditadi A, Malerba A, Occhi G, Gamba PG, Scambi I, McLachlan I, Baroni V, Vitiello L

17.00 Break

MOTOR CONTROL, AGING AND DENERVATION OF SKELETAL MUSCLE

17.30 AGONIST-ANTAGONIST ACTIVATION DURING ELBOW FLEXION AND EXTENSION AT DIFFERENT ANGU- LAR VELOCITIES. Bazzucchi I, Marzattinocci G, Felici F

17.45 EFFECTS OF INHIBITORS OF CELL MEMBRANE CALCIUM CHANNELS ON HIGH-FREQUENCY FATIGUE OF FAST AND SLOW SKELETAL MUSCLES. GerminarioE, Esposito A, Midrio M, Palade PT, Betto R, Danieli D

18.0 DIFFERENCES IN FORCE/ENDURANCE RELATIONSHIP BETWEEN YOUNG AND OLDER MEN Bazzucchi I, Marchetti M, Rosponi A, Fattorini L, Castellano V, Sbriccoli P, Felici F

18.15 EFFECTS OF SPHINGOMYELIN DERIVATIVES ON INNERVATED AND DENERVATED RAT SOLEUS MUSCLE ZaninM, Germinario E, Betto R, Danieli D

18.30 LONG-TERM DENERVATION OF RAT MUSCLE: A TIME COURSE STUDY Adami N, Biral D, Kern H, Carraro U

18.40 LONG-TERM LOWER-MOTONEURON DENERVATION OF HUMAN MUSCLE: A TIME COURSE STUDY Caccavale S, Rossini K, Adami N, Kern H, Carraro U

18.50 MYOREGENERATION IN HUMAN DENERVATED DEGENERATED MUSCLE DECREASES AFTER MUSCLE RE- COVERY INDUCED BY FES TRAINING Rossini K, Caccavale S, Adami N, Kern H, Carraro U

19.00 RESTITUTION OF LONG-TERM DENERVATED MUSCLE BY FES Boncompagni S, Kern H, Mayr W, Carraro U, Protasi F

19.15 HELMUT KERN, Guest Speaker: Functional Electrical Stimulation of Skeletal Muscle: Clinical Results of the EU Project RISE

20.00 IIM Social Dinner

Sunday October 17, 2004

REGENERATIVE PATHWAYS AND MUSCLE DISEASES

Chair: A. Musarò and R Bottinelli

9.00 SKELETAL MUSCLE OF MICE OVEREXPRESSING FRG1 SHOWS HYSTOLOGICAL AND FUNCTIONAL SIGNS OF MUSCULAR DYSTROPHY Brocca L, Pellegrino MA Moggio M, Green M, Tupler R, Bottinelli R

9.15 TRANSGENIC MOUSE MODELS OF MUSCLE WASTING AND REGENERATION Dobrowolny G, Giacinti C, Pelosi L, Nicoletti C, Barberi L, Molinaro M, Rosenthal N, Musarò A

9.30 HYALURONIC ACID BASED DRESSING AS ANTI-FIBROSIS AGENT IN THE TREATMENT OF MUSCLE INJURY Vindigni V, Mazzoleni F, Carraro U

9.45 STRESS GENE EXPRESSION IN SKELETAL MUSCLES AFTER MODERATE EXERCISE Marini M, Lapalombella R, Scordari A, D'Aloia C, Margonato V, Esposito F, Veicsteinas A

10.00 GENE EXPRESSION MODIFICATIONS IN RAT HEART FOLLOWING MODERATE PHYSICAL EXERCISE Margonato V, Veicsteinas A, Samaja M, Ventura C, Lapalombella R, Scordari A, Carinci P, Marini M

10.15 CONTRACTILE PROPERTIES OF SINGLE MUSCLE FIBERS FROM NORMAL DOGS AND DOGS AFFECTED BY GOLDEN RETRIEVER MUSCULAR DYSTROPY Rinaldi C, Pansarasa O, Bottinelli R, Blot S, D'Antona G

10.30 Break

MUSCLE DISEASES 2

Chair: A Uncini and V Sorrentino

11.00 IDENTIFICATION AND FUNCTIONAL STUDIES OF MUTATION IN THE RYR1 GENE IN PATIENS WITH MALIG- NANT HYPERTHERMIA Rossi D, De Smedt P, Galli L, Orrico A, Franci D, Petrioli F, Lorenzini S, Tegazzin V, Sorrentino V

11.15 PROTEIN AND MOLECULAR DIAGNOSIS IN LGMD2A Nascimbeni AC, Fulizio L, Spinazzi M , Fanin M, Angelini C

11.30 LIMB-GIRDLE MUSCULAR DYSTROPHIES TYPE 2A AND 2B: CLINICAL AND RADIOLOGICAL ASPECTS Borsato C, Padoan R, Stramare R, Fanin M, Angelini C

11.45 AN ENDOCRINOLOGICAL AND NEUROPSYCHOLOGICAL INVESTIGATION IN MYOTONIC DYSTROPHY TYPE 1 (DM1) Romeo V, Squarzanti F, Mongiat M, Gasparoni P, D'Ascenzo C, Pegoraro E, Angelini C

12.00 FAMILIAL IDIOPATHIC HYPERCKEMIA Capasso M, De Angelis MV, Pace M, Zuccarini F, Di Muzio A, Uncini A

12.15 FAMILIAL IDIOPATHIC HYPERCKEMIA: A POSSIBLE PREDISPOSITION TO STATIN-INDUCED MYOPATHY Capasso M, Di Muzio A, De Angelis MV, Uncini A

12.30 MUSCLE INFECTION IN CHRONIC HEPATITIS B Capasso M, Di Muzio A, Comar M, Campello C, Robuffo I, Gambi A, De Angelis MV, Uncini A

14.00 – 15.00 Meeting of the IIM Council

REGULATORY MECHANISMS OF MYOGENESIS 1

Chair: F. Protasi and G Cecchi

15.00 TNFalpha AND AVP EXERT OPPOSITE EFFECTS ON MUSCLE REGENERATION Moresi V, Adamo S, M Molinaro, Coletti D

15.15 CHARACTERIZATION OF MYOGENIC FACTORS DERIVED FROM A STABLE MACROPHAGE CELL LINE Malerba A, Scambi I, Segat D, Frigo M, De Coppi P, Gamba P, Boldrin L, Cavallini L, Bellomo R, Fanò G, Vecchiett L, Vi- tiello L, Baroni D

15.30 3D-CULTURE OF ISOLATED CELLS AND TISSUE EXPLANTS IN RELATIVE MICROGRAVITY: NEW PERSPEC- TIVES AND POSSIBLE APPLICATIONS Steimberg N, Rovetta F, Boniotti J, Mazzoleni G

15.45 MUSCLE TISSUE ENGINEERING USING SINGLE FIBRE ISOLATION TECHNIQUE: IN VITRO AND IN VIVO PROSPECTS Boldrin L, Flaibani M, Malerba A, Slanzi E, Pozzobon M, Baroni D, Messina C, Zanesco L, Gamba PG, Vitiello L, Elvasso- re N, De Coppi P

16.00 THE OXIDATIVE DAMAGE INDUCES MODIFICATIONS OF Ca2+ TRANSPORT SYSTEM IN HUMAN SATELLITE CELLS Beccafico S, Belia S, Puglielli C, Pietrangelo T, Fulle S

16.15 SPHINGOSINE 1-PHOSPHATE AFFECTS THE ELECTRIC PROPERTIES OF THE PLASMAMEMBRANE IN C2C12 MYOBLASTS Squecco R, Formigli L, Sassoli C, Chellini F, Quercioli F, Zecchi S, Tiribilli B, Francini F

16.30 ORGANIZATION OF THE SARCOPLASMIC RETICULUM DURING MUSCLE

DEVELOPMENT Cusimano V, Giacomello E, Sorrentino V

16.45 MHC ISOFORM EXPRESSION IN BOVINE SINGLE FIBRES STUDIED AT RNA AND PROTEIN LEVEL Toniolo L, Maccatrozzo L, Patruno M, Mascarello F, Reggiani C

17.00 MYOSIN HEAVY CHAIN ADULT ISOFORMS EXPRESSION IN DIFFERENT SKELETAL MUSCLES OF CATTLE Maccatrozzo L, Patruno M, Toniolo L, Reggiani C, Mascarello F

17.15 FIBRE TYPES, MHC ISOFORM EXPRESSION AND SINGLE FIBRE CONTRACTILE PROPERTIES IN FELINE SKELETAL MUSCLES Pavan E, Toniolo L, Maccatrozzo L, Patruno M, Mascarello F, Reggiani C

17.30 INSIDE MUSCLE –TENDON UNIT BY SURFACE MECHANOMYOGRAM (MMG) AND FORCE SIGNAL Orizio C, Gobbo M

18.15 Open Convention of the IIM

Monday October 18, 2004

REGULATORY MECHANISMS OF MYOGENESIS 2

Chair: G Fanò and U Carraro

9.00 SARCOLEMMAL IONIC CONDUCTANCE IN CULTURED MUSCLE FIBRES OF mdx, COL VI KO, dy/dy AND WT MICE Canato M, Pavan E, Vassanelli S, Megighian A, Reggiani C

9.15 CALCIUM COMPARTMENTS AND GENE EXPRESSION ACTIVATION IN L6 MYOGENIC CELLS Naro F, De Arcangelis V, Coletti D, Canato M, Molinaro M, Adamo S, Reggiani C

9.30 THE HETEROLOGOUS EXPRESSION OF SARCOGLYCANS Gastaldello S, Sandonà D, Maddaloni C, D'Angelo S, Martinello T, Chan Yi-umo, Betto R

9.45 DYSTROPHIN-ASSOCIATED-GLYCOPROTEIN AND VINCULIN-TALIN-INTEGRIN-COMPLEXES DURING MYO- GENESIS. TIMING APPEARANCE AND LOCALIZATION IN NORMAL HUMAN SKELETAL MUSCLE CULTURE Di Mauro D, Magaudda L, Mancinelli R, Trimarchi F

10.00 EXPRESSION OF VASOPRESSIN RECEPTORS IN MYOGENESIS Alvisi M, Ciccone L, Naro F, Adamo S

10.15 EXTRACELLULAR NUCLEOTIDES AFFECT MYOGENESIS Martinello T, Sandonà D, Gastaldello S, Betto R

10.30 THE ROLE OF EXTRACELLULAR GTP ON DIFFERENTIATION OF C2C12 MYOBLASTS Pietrangelo T, Mancinelli R, Fulle S, Fanò G

10.45 ROLE OF cAMP SIGNALLING IN MYOGENIC CELLS De Arcangelis V, Némoz G, Molinaro M, Adamo S, Naro F

11.00 SPHINGOSINE 1-PHOSPHATE REGULATES MYOGENIC DIFFERENTIATION. A MAJOR ROLE FOR S1P2 RECEPTOR Donati C, Meacci E, Nuti F, Becciolini L, Farnararo M, Bruni P

11.15 SPHINGOSINE 1-PHOSPHATE INDUCES CYTOSKELETAL REORGANIZATION IN C2C12 MYOBLASTS Chellini F, Sassoli C, Nosi D, Meacci E, Bruni P, Formigli L, Zecchi-Orlandini S

11.30 INVOLVEMENT OF PHOSPHATIDIC ACID IN ACTIN FIBER FORMATION IN DIFFERENTIATING L6 MYOBLASTS Komati H, De Arcangelis V, Adamo S, Némoz G, Naro F

11.45 A QUANTITATIVE STUDY ON REGULATORY MOLECULES INVOLVED IN THE POST-NATAL GROWTH OF PIG Caliaro F, Maccatrozzo L, Toniolo L, Reggiani C, Mascarello F, Patruno M

AUTHORS OF ABSTRACTS

Curriculum of Giorgio Fanò Illic (Alias Giorgio Fanò), PhD

1970, University of Perugia, Degree in Biological Sciences
1971-1973 Didactic-scientific fellowship in Human Physiology (PhD equivalent)
2015-2016, Quiescence

CURRENT SITES:
Free University of Alcatraz Santa Cristina di Gubbio (ITALY);
Laboratory of Functional Assessment, Department of Neuroscience, Imaging and Clinical Sciences, University 'G. d'Annunzio', Chieti-Pescara.
Tel: +39 348 089 7627, E-mail: fanoillic@gmail.com

LATEST ACADEMIC POSITIONS:

1. Former Contract Professor (2018-2019) for General Physiology (M-Z) of the Bachelor of Biological Sciences, Polytechnic University of Marche, Ancona
2. Former Full Professor of Physiology (BIO-09) and Coordinator of the Physiology and Biophysics Course, Faculty of Medicine and Surgery - 'G. d'Annunzio' University, Chieti- Pescara.
3. Former Coordinator of the PhD in "Basic and Applied Medical Sciences", consortium with the University of Brescia in collaboration with University of Philadelphia,USA (Prof. C. Franzini-Armstrong), University of South Florida Tampa, USA (Prof. V. Camporesi).
4. Former Deputy Director of the Department of Neuroscience & Imaging - 'G. d'Annunzio' University, Chieti-Pescara.

PREVIOUS ACADEMIC POSITIONS:
1978-1986 Full Assistant for General Physiology at the Faculty of MM.FF.NN. Sciences, University of Perugia
1986-1993 Associate Professor of General Physiology, Faculty of SS. MM. FF. e NN., University of Perugia
1994-1998 Associate Professor of General Physiology, Faculty of Pharmacy, University 'G. d'Annunzio' Chieti-Pescara
1994-1997 Lecturer in Human Physiology, Faculty of Medicine and Surgery, University 'G. d'Annunzio' Chieti-Pescara
1994- 1998 Professor of General Physiology, Faculty of SS. MM. FF. e NN., University of Perugia
1999- 2014 Full Professor of Physiology, Faculty of Medicine and Surgery, University 'G. d'Annunzio' Chieti-Pescara
2002-2008 Lecturer in Applied Physiology, Faculty of Physical Education, University 'G. d'Annunzio' Chieti-Pescara
2004-2007 Founder and Director of the Interuniversity Institute of Myology (IIM): Consortium between the Italian universities of Chieti, Florence, Messina, Milan, Perugia, Siena, Brescia, Padua, Rome1

SCIENTIFIC EXPEDITIONS
1. 2008 Coordinator of the inter-university project: INTERAMNIA 8000- MANASLU EXPEDITION (NEPAL- HIMALAYA).
2. 2012 Coordinator of the inter-university project: TREK GOKIO CUMBU/AMADABLAM ((NEPAL- HIMALAYA)): GENDER DIFFERENCES IN PHYSIOLOGICAL RESPONSES TO HYPOBARIC HYPOXIA.
3. 2014 Head of Unit in MEDICAL RESEARCH ON HYPOXIA (MERHY): MAN AT ALTITUDE from molecular level to man, in healthy and pathological conditions. Coordinators C. Marconi and P. Cerretelli

EDITORIAL BOARD
- European Journal of Translational Myology (Senior editor for the Section of History and Future of Mobility Medicine)
- Sport Science for Health
- pH (from physics to Philosophy)
- International Journal of molecular science (IJMS)

FUNDED RESEARCH PROJECTS:
1983-1986 Subcontract for GMNCE-CNR Project
1985-1988 Subcontract for MPI-COFIN national project
1988-1989 Subcontract Strategic-CNR project
1989-1997 Subcontract for MPI-COFIN national project
2000 Co-holder (with M.P. Rathbone) of United States Provisional Patent Application No. 38,003/11230-1
2001- 2002 Principal Investigator for MPI-COFIN national project
2003-2006 Principal Investigator for MATT national project
2008- Subcontract for MPI-COFIN national project
2008-2010 Subcontract (Work -Package No. 1.5.02 GMP-A.S.I.)

SCIENTIFIC INTERESTS

A) Regulation of muscle trophism: production and role of free radicals

B) Neurons and muscle cells: role of local growth factors

C) Physiological & pathophysiological aspects of myogenesis and muscle senescence

D) Effects of magnetic fields on the vital capacity of different cellular systems

A- Emotional papers

1. Mecocci P, Fanó G, Fulle S, MacGarvey U, Shinobu L, Polidori MC, Cherubini A, Vecchiet J, Senin U, Beal MF. Age-dependent increases in oxidative damage to DNA, lipids, and proteins in human skeletal muscle. Free Radic Biol Med. 1999 Feb;26(3-4):303-8.
2. Fanò G, Biocca S, Fulle S, Mariggiò MA, Belia S, Calissano P. The S-100: a protein family in search of a function. Prog Neurobiol. 1995 May;46(1):71-82. Review.
3. Fanò G, Orlacchio A. beta-N-acetyl-D-glucosaminidase activity levels in atrophic gastrocnemius muscle of Rana esculenta. Comp Biochem Physiol B. 1982;73(2):399-403.
4. Bosco G, Verratti V, Fanò G. Performances in extreme environments: effects of hyper/hypobarism and hypogravity on skeletal muscle. Myology Reviews 2010. 20(3): 83-90.
5. Musarò A, Fulle S, Fanò G. Oxidative stress and muscle homeostasis. Curr Opin Clin Nutr Metab Care. 2010. May;13(3):236-42. Review.
6. S Fulle, G Di Tano, G Fano The prescription of physical exercise in the prevention and treatment of neuromuscular degeneration. Italian journal of sport medicine. 2006. 59 (4), 453-456
7. Verratti V, Falone S, Fanò G, Paoli A, Reggiani C, Tenaglia R, Di Giulio C. Effects of hypoxia on nocturnal erection quality: a case report from the Manaslu expedition. J Sex Med. 2011 Aug;8(8):2386-90.
8. M. Bizzarri, G. Fanò-Illic Some Inshights into biological complexity, pH, 2013. 1-2013, 48-62
9. G Fanò-Illic, S Belia, G Cocchia, V Verratti THE PHYSIOLOGICAL BASIS OF DORIAN GRAY'S PORTRAIT Journal of the Siena Academy of Sciences 2013. 5 (1), 41-48
10. Pietrangelo T, Di Filippo ES, Mancinelli R, Doria C, Rotini A, Fanò-Illic G, Fulle S. Low Intensity Exercise Training Improves Skeletal Muscle Regeneration Potential. Front Physiol. 2015 Dec 24;6:399.
11. Giammarco E, Di Sano S, Aureli T, Cerratti P, Fanò-Illic G, Pietrangelo T. Psychological and Physiological Processes in Figure-Tracing Abilities Measured Using a Tablet Computer: A Study with 7 and 9 Years Old Children. Front Psychol. 2016 Oct 18;7:1528.
12. G.Abate, G. Fanò-Illic How to get rid of the scientific fraud - and saving the scientists at the same, pH, 2016. 2-2016, 51-65.
13. G. Fanò-Illic GOING BEYOND 'There are awards that give recognition to the success of those who "make people laugh first and then make them think"', pH, 2017. 2-2017, 49-58
14. T. Pietrangelo, S. Fulle, F. Coscia,P.V. Gigliotti, G. Fanò-Illic. Old muscle in young body: an aphorism describing the Chronic Fatigue syndrome. European Journal of Translational Miology: 10.4081/ejtm.2018.7688

15. Giorgio Fanò-Illic, Rosa Mancinelli Critical reflections on the physiology of marriage published in July - December 2020 - pH - issue no.2; 2020

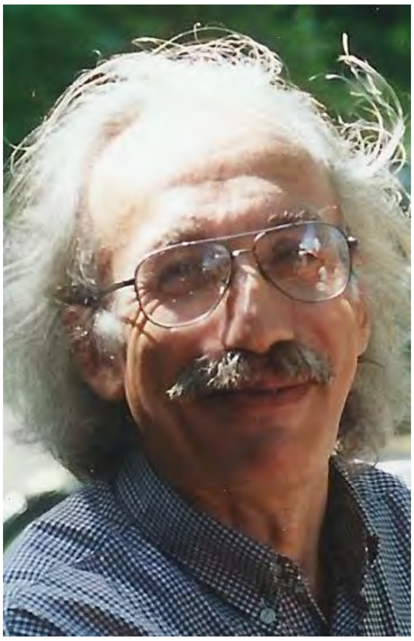
B- Scientific articles (short selection)

1. Fanò-Illic G, Fulle S, Mecocci P. Editorial for the Special Issue 'Molecular Bases of Senescence'. Int J Mol Sci. 2021 Nov 2;22(21):11873. doi: 10.3390/ijms222111873.
2. Mancinelli R.; Checcaglini F.; Coscia F.; Gigliotti P; Fulle S.; Giorgio Fanò-Illic G. Biological aspects of selected myokines in skeletal muscle: fo-cus on the aging process. Int. J. Mol. Sci.(ISSN 1422-0067) on 04 August 2021
3. Mancinelli R, Fanò-Illic G, Pietrangelo T, Fulle S. Guanosine-Based Nucleotides, the Sons of a Lesser God in the Purinergic Signal Scenario of Excitable Tissues. Int J Mol Sci. 2020 Feb 26;21(5):1591. doi: 10.3390/ijms21051591.
4. Fulle S, Belia S, Fanò Illic G. The Ariadne thread: the matching of S-100 family with the RyR's muscle receptor. Eur J Transl Myol. 2020 Apr 1;30(1):8839. doi: 10.4081/ejtm.2019.8839.
5. Doria C, Verratti V, Pietrangelo T, Fanò-Illic G, et al Changes in energy system contributions to the Wingate anaerobic test in climbers after a high altitude expedition. Eur J Appl Physiol. 2020 Jul;120(7):1629-1636. doi: 10.1007/s00421-020-04392-8. Epub 2020 Jun 3. PMID: 32494861.
6. Coscia F, Gigliotti PV, Piratinskij A, Pietrangelo T, Verratti V, Foued S, Diemberger I, Fanò-Illic G. Effects of a vibrational proprioceptive stimulation on recovery phase after maximal incremental cycle test. Eur J Transl Myol. 2019 Aug 27;29(3):8373. doi: 10.4081/ejtm.2019.8373.
7. Verratti V, Letta F, Paulesu L, Romagnoli R, Ceccarelli I, Doria C, Fanò Illic G, Di Giulio C, Aloisi AM. Physiological effects of high-altitude trekking on gonadal, thyroid hormones and macrophage migration inhibitory factor (MIF) responses in young lowlander women. Physiol Rep. 2017 Nov;5(20).
8. Giammarco E, Di Sano S, Aureli T, Cerratti P, Fanò-Illic G, Pietrangelo T. Psychological and Physiological Processes in Figure-Tracing Abilities Measured Using a Tablet Computer: A Study with 7 and 9 Years Old Children. Front Psychol. 2016 Oct 18;7:1528.
9. Pietrangelo T, Di Filippo ES, Mancinelli R, Doria C, Rotini A, Fanò-Illic G, Fulle S. Low Intensity Exercise Training Improves Skeletal Muscle Regeneration Potential. Front Physiol. 2015 Dec 24;6:399.
10. Mancinelli R, La Rovere RM, Fulle S, Miscia S, Marchisio M, Pierdomenico L, Lanuti P, Procino G, Barbieri C, Svelto M, Fanò-Illic G, Pietrangelo T. Extracellular GTP is a Potent Water-Transport Regulator via Aquaporin 5 Plasma-Membrane Insertion in M1-CCD Epithelial Cortical Collecting Duct Cells. Cell Physiol Biochem. 2014;33(3):731-46.
11. Guarnieri S, Morabito C, Paolini C, Boncompagni S, Pilla R, Fanò-Illic G, Mariggiò MA. Growth associated protein 43 is expressed in skeletal muscle fibers and is localized in proximity of mitochondria and calcium release units. PLoSOne. 2013;8(1):e53267. oi: 10.1371/journal.pone.0053267.
12. Morabito C, Bosco G, Pilla R, Corona C, Mancinelli R, Yang Z, Camporesi EM, Fanò G, Mariggiò MA. Effect of pre-breathing oxygen at different depth on oxidative status and calcium concentration in lymphocytes of scuba divers. Acta Physiol (Oxf). 2011 May;202(1):69-78.

13. Bosco G, Yang ZJ, Di Tano G., Faralli F, Savini F, Landolfi A, Doria C, Fanò G. Effect of in-water oxygen prebreathing at different depths on decompression-induced bubble formation and platelet activation. J Appl Physiol. 2010 May;108(5):1077-83.
14. Pietrangelo T, Puglielli C, Mancinelli R, Beccafico S, Fanò G, Fulle S. Molecular basis of the myogenic profile of aged human skeletal muscle satellite cells during differentiation. Exp Gerontol. 2009 Aug;44(8):523-31.
15. Pietrangelo T, Fioretti B, Mancinelli R, Catacuzzeno L, Franciolini F, Fanò G, Fulle S. Extracellularguanosine-5 '-triphosphatemodulatesmyogenesis via intermediate Ca(2+)-activated K+ currents in C2C12 mouse cells. J Physiol. 2006 May 1;572(Pt 3):721-33.
16. Fulle S, Protasi F, Di Tano G, Pietrangelo T, Beltramin A, Boncompagni S, Vecchiet L, Fanò G. The contribution of reactive oxygen species to sarcopenia and muscle ageing. Exp Gerontol. 2004 Jan;39(1):17-24. Review.
17. Fulle S, Mecocci P, Fanó G, Vecchiet I, Vecchini A, Racciotti D, Cherubini A, Pizzigallo E, Vecchiet L, Senin U, Beal MF. Specific oxidative alterations in vastus lateralis muscle of patients with the diagnosis of chronic fatigue syndrome. Free Radic Biol Med. 2000 Dec 15;29(12):1252-9.
18. Mecocci P, Fanó G, Fulle S, MacGarvey U, Shinobu L, Cherubini A, Vecchiet J, Senin U, Beal MF. Age-dependent increases in oxidative damage to DNA, lipids, and proteins in human skeletal muscle. Free Radic Biol Med. 1999 Feb;26(3-4):303-8.

CHAPTER 13. Inspirers & Supporters

13.4. Carlo Reggiani



Carlo Reggiani 1996

Carlo Reggiani 2020

In 2000 Prof. Menotti Midrio, full professor of Physiology at the University of Padova with the support of Stefano Schiaffino, a long term collaborator of Terje Lomo and Carlo Reggiani, was able to create the conditions for attracting to Padova Carlo as the Full Professor of Physiology. It was a major chance for the Padua Physiology and Myology tradition. Indeed, he brought to Padova all his international links opening a new era for a very traditional Institute of Human Physiology of the University of Padua. His very soft but determined touch allowed survival of the best physiologists previously working in Padova, adding year after year new young brilliant researchers and teachers of Human Physiology. After his retirement in 2018, he supported the Deptment of Biomedical Sciences to invest in one of his international collaborators and Marco Narici, at that time Professor of Physiology at the Medical School of Nottingham (UK) became full professor of Physiology in Padova bringing from UK not only his expertise but also a network of international collaborations and a few young collaborators. I am honored that Carlo accepted invitation to join this book. Though we never directly interacted in research activities (we have not scientific paper in common) his support to CIR-MYO, Padua Muscle Days and BAM/EJTM was always the key to obtain academic support from the University of Padova and in the international arena.

Short Curriculum of Carlo Reggiani

Born in Pavia 1948, MD 1972, specialized in cardiology 1977
Working as physician (general practitioner (medico condotto) in Nibbiano (PC) 1973-4, emergency doctor in Pavia 1974-1979, factory doctor in Pavia, Necchi factory, 1975-76)
Stay in Lund Department of Pharmacology 1979-1984 in Paul Edman's laboratory: from a project on new glycosides and experimental heart failure (in rats) to experiments on skeletal single muscle fibers.
Associate Professor in Physiology, University of Pavia 1984-1991,
Full Professor in Physiology, University of Pavia 1991-2000 and University of Padova 2000-2018
Professor Emeritus University of Padova, 2019 to present
Director of the School of Specialization in Sport Medicine in Pavia and later in Padova.
President of the undergraduate program in kinesiology (Corso di laurea in Scienze Motorie) from 2008 to 2016
President of the Italian Society of Physiology SIF from 2009 to 2012.
Feltrinelli Prize of "Accademia dei Lincei "(2014)

Research lines of Carlo Reggiani

Cardiac physiology, heart failure, regulation of contraction-relaxation (1970-1990)
Skeletal muscle physiology, single fiber mechanics, myosin isoforms, intrafiber calcium dynamics, plasticity and aging (from 1980 to now)

Personal memories and reflections on Ugo and the Myology in Padova

I got to know Ugo Carraro at the XVII EMC (European Muscle Conference) held in Abano in 1988. At that time, my lab in Pavia was still working on cardiac muscle and the transition to skeletal muscle was only at the beginning. A short paper "Shortening velocity, myosin and myofibrillar ATPase activity related to myosin isoform composition in rat ventricular myocardium" was published in the book collecting the presentation at the conference.

Even without a direct collaboration with Ugo, I followed, appreciated and participated to several projects of Ugo:
-- I attended and contributed to most if not all "PADUA muscle days"
-- I published and served as reviewer to the journal BAM from 1990 and then EJTM from 2010, see list below (possibly not complete)
-- I joined and supported the activity of the CirMYO, where I represented the department of belonging

With Ugo I shared the attendance at the EMC (European Muscle Conference) and the presence as Italian representative in the steering committee.

To my best knowledge, Myology, study on skeletal muscle, was born in

Padova in the Institute of General Pathology. Massimo Aloisi, professor of General Pathology was very likely the initial promoter of this particular field of study in “60s. Then, Margreth and Schiaffino brought it to international recognition.

Ugo Carraro not only continued and further implemented that field of study, but gave it a structure, flexible but resilient to the difficult times experienced by academic world in Italy. Without discussing his scientific contribution which are collected in 175 papers (more than 9000 citations), three initiatives of Ugo have been extremely relevant to the development, growth and maturation of the muscle studies in Padova.

Without the proposals of Ugo, myology in Padova was only the field of study of isolated researchers, with little exchange among them and no links with clinicians. The three proposals listed here below have created the milieu for productive exchanges and collaborations.

-- A journal, first with the sexy name of BAM and then under the most serious name of EJTM was founded in 1991 and reaches now the 32nd year.

-- A series of annual meetings “Padua Muscle Days” provided an occasion of discussion among the workers of the field, first with only a local appeal, but more recently with a wide and qualified international participation.

-- The foundation of the Interdipartmental Center CirMYO of the University of Padua gave structural bases to the activity of basic scientists and clinicians working on skeletal muscle.

On BAM/EJTM I’ve published the following full papers

Carlo Reggiani and Truus te Kronnie. Hyperplasia in Exercise-Induced Muscle Growth? Basic Applied Myology. 9 (6): 289-292, 1999

Marco Dal Maschio, Marta Canato, Filippo M. Pigozzo, Alberto Cipullo, Gianantonio Pozzato, Carlo Reggiani. Biophysical effects of high frequency electrical field (4-64 MHz) on muscle fibers in culture Basic Applied Myology 19 (1): 49-56, 2009

Francesco Pacelli, Antonio Paoli, Valfredo Zolesi, Aleandro Norfini, Alessandro Donati, Carlo Reggiani. Implementation and ground validation of a facility for functional and structural analysis of proximal upper limb muscles in microgravity Basic Applied Myology 19 (2): xx-yy, 2009

Reggiani C, Schiaffino S. Muscle hypertrophy and muscle strength: dependent or independent variables? A provocative review. Eur J Transl Myol. 2020 Sep 9;30(3):9311. doi: 10.4081/ejtm.2020.9311. PMID: 33117512; PMCID: PMC7582410

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CHAPTER 13. Inspirers & Supporters

13.5. Sergio Adamo

In Memoriam: Sergio Adamo (1950 – 2022)

Sergio Adamo, sadly we have to use the verb in the past tense, was a pioneer in Rome of modern embryology and histology, but I am ashamed to add he was also a strong supporter of my dreams and projects in Translational Myology and Mobility Medicine.
It is easier for me to remember him by giving the words to his pupils Bianca M. Scicchitano, Marina Bouchè, Clara Nervi, Dario Coletti, who asked to publish his obituary in the European Journal of Translational Myology (EJTM) 32 (1): 10434, 2022 doi: 10.4081 /ejtm.2022.10434.
I just have to add that I can not forget that he had agreed to be the editor for EJTM reviews. It is a great honor for me to have the opportunity to remember him in this book as well.

A tribute to Professor Sergio Adamo, Full Professor of Histology and Embryology at Sapienza University, Rome

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Abstract

Sergio Adamo prematurely left us on January 7th 2022, just one year after his retirement, leaving his family, friends and colleagues deeply sad and grieving. Sergio was a full Professor of Histology and Embryology at the Sapienza University of Rome. Since the foundation of the Institute of Histology and Embryology more than 50 years ago, he dedicated himself to the institution, research, and teaching with integrity, generosity, and a great sense of teamwork. Sergio's main research interests have been the mechanisms of myogenesis, muscle homeostasis and regeneration under normal and pathological conditions. Most relevant results obtained by Sergio and his collaborators indicate novel functions for the neurohypophyseal hormones, vasopressin and oxytocin, upon striated muscle differentiation, trophism, and homeostasis. Here we like to give the proper tribute to a mentor, a colleague and a sincere friend. He left an indelible mark on the professional and personal lives of all of us and his absence provokes a profound sense of emptiness.

"The trouble with the world is that the stupid are cocksure and the intelligent are full of doubt."
Bertrand Russell

Key Words: Skeletal muscle; Neurohypohyseal hormones; cachexia.

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Sergio Adamo, full Professor in Histology and Embryology at the Sapienza University of Rome, passed away on January 7th, 2022 leaving his family, friends, and colleagues with a profound sense of sadness. He had retired just one year earlier, but he was still an active participant within the Department and the academic community. He was a well-known and highly respected scientist and teacher. Since the foundation of the Institute of Histology and Embryology more than 50 years ago, he dedicated himself to the institution, research, and teaching, always with integrity, generosity, and a great sense of teamwork. He joined the Institute when he was still a Medical student, when Valerio Monesi, the father of the Histology discipline in Rome, moved there to establish an innovative and active research center. Sergio started his research activity within the myogenesis group, coordinated by Mario Molinaro, who was developing a line of research aimed to study cell differentiation, among the few, at that time, establishing primary cell cultures as experimental strategy.1,2 After a period at the NIH in Bethesda, where Sergio improved his expertise in cellular biochemistry, he returned to Rome in 1978, and established his own laboratory, making his contribution towards the understanding of the regulation of in vitro myoblasts differentiation.3,4 Since then, he continued his research in muscle biology, using both in vitro and in vivo experimental models. As a full Professor of Histology and Embryology since 1990, and for many years as a coordinator of the Ph.D. program in Morphogenesis and Engineering Tissue, he was a scientific guide and an inspiration for all the students and colleagues as a person of the highest integrity, honesty, and sincerity. Likewise, as a Director of the Histology Department from 2007 to 2010, he worked tirelessly in improving the academic standing not only for his lab but also for the entire community. Sergio's main research interests were focused on muscle homeostasis and regeneration under normal and pathologic conditions.[1-16] The most relevant results obtained by Sergio and his collaborators indicate novel functions for the neurohypophyseal hormones, vasopressin (AVP) and oxytocin (OT), which regulate skeletal muscle differentiation, trophism, and homeostasis. In particular, Sergio's group demonstrated for the first time that AVP promotes muscle differentiation, hypertrophy, and regeneration through the combined activation of the calcineurin and Calcium/Calmodulin-dependent Protein Kinase (CaMK) pathways.[5–9] Sergio's studies revealed that the AVP system is impaired in several neuromuscular diseases, suggesting that AVP may act as a physiological factor in skeletal muscle. This prompted Sergio to investigate the role of AVP in vivo demonstrating in a murine model that local over-expression of the AVP receptor V1a enhances the regeneration of atrophic muscle. Indeed, by upregulating the regeneration and differentiation markers, modulating the inflammatory response, and attenuating fibrogenesis, the stimulation of AVP-dependent pathways creates a favorable environment for efficient and sustained muscle regeneration and repair even in the presence of elevated levels of the inflammatory cytokine TNF.[10–15] All these studies performed by Sergio's group highlight a novel in vivo role for AVP-dependent pathways, which may represent an interesting strategy to counteract muscle decline in aging or in muscular pathologies.[13] All of the above prompted to verify whether neurohypophysial hormones might be proposed as a hormonal treatment to counteract cancer-induced muscle wasting, as well. Indeed, circulating concentration of OT was found reduced in cancer patients, and its administration in a pre-clinical model of cancer cachexia appeared to be promising: these results are currently submitted for publication and represent a cue strongly suggesting that the use of neurohypophysial hormones has potential clinical applications. Additional contributions to counteract cachexia come from his group's studies on the mechanisms underlying the impairment of muscle stem cells in cachexia and countermeasures, such as physical exercise.[16] The area of interest of Sergio's group recently spread also in the mechanisms underlying muscle denervation and the pathophysiology of Duchenne Muscular Dystrophy (DMD), focusing in both cases on the role of HDAC4 in muscle fiber atrophy and on mechanisms of sarcolemma repair.[16]

We all had the opportunity to work with Sergio, either enjoining his laboratory or closely collaborating in research and teaching. He has been an extraordinary support for all of us, and we all established a profound friendship together with a working relationship. His passion for science was truly contagious and he was a generous, open-minded, and helpful mentor and collaborator, always willing to listen and truly pleased to see the progress of our achievements and success. We also had a lot of fun with him, pleased by his sharp humor, always searching for new jokes to share. Do you remember his mustache bended by the ironic but always friendly smile? This is the way we evoke our Sergio, professor at Sapienza, scientist, and friend: gentlemanly but funny, exciting but calm, pleasant but strict, upstanding. A honest person. His teaching will remain in all of us, students and colleagues, spread around the world, forever. Many people will miss him, we will certainly miss him sorely.

Bianca, Marina, Clara, Dario

When I joined Monesi's group in 1969 as assistant professor, Sergio Adamo together with Marco Conti, two young students at the Medical School, were already there, the first to be present in Monesi's lab. Indeed, when Valerio Monesi was appointed as professor of Histology in 1968, his lab was an empty small space inside the Institute of Anatomy, and Sergio and Marco represented the founder germ of the new histological institution, helping Monesi in moving furniture, mounting shelves, and planning about the future. I was immediately attracted by Sergio's curiosity, his desire of knowledge and experimental ability. Sergio was promptly involved in setting up a model of myogenesis in primary cultures, laying the foundation the future studies on satellite muscle cell differentiation and their role in muscle growth and repair. Since then, we never stopped our long-lasting collaboration. Many qualities were harmoniously fused in Sergio's person. His sudden death left a sense of sadness for the loss of a friend, a colleague, a teacher, a just man.

Mario Molinaro

List of acronyms

AVP - Arginine Vasopressin
CaMK - Calcium/Calmodulin-dependent Protein Kinase
DMD - Duchenne Muscular Dystrophy
OT - Oxytocin
TNF - Tumor Necrosis Factor

Contributions of Authors

All contributors approved the manuscript and agreed with publication.

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CHAPTER 13. Inspirers & Supporters

13.6. Zipora Yablonka-Reuveni


Left panel: The first satellite cell meeting, Boston, 1998**. From left, Drs. Ryuichi Tatsumi, Ron Allen, Richard Bischoff, Bruce Carlson, David Yaffe, **Zipora Yablonka-Reuveni**, Oicheng Ng. (Photo credit Dr. Judy Anderson; found and contributed by Dr. Orna Halevy). **Right panel: Zipora Yablonka-Reuveni, 2022

I first met Prof. Zipora Yablonka-Reuveni, now Prof Emerita, in Tiberias, Israel, for an European Muscle Club Meeting in 1987 where she presented the Abstract: Yablonka-Reuveni Z, Nameroff M. Satellite cells from chicken skeletal muscle exhibit distinct properties which are not expressed by embryonic myoblasts. Joint Meeting of the European Club for Muscle and Motility and the European Cytoskeletal Club on Cellular Dynamics. Tiberias, Israel. J Muscle Res Cell Motil. 1988; 9: 105. We were both young and unknown to each other, but satellite cells and muscle regeneration were a good link to start and then continue discussions up to-date by exchanging e-mails, when this had become the easy way to maintain distant friendships. I still remember her presentation in Tiberias of the first antibody against satellite cells. The pictures were impressive and I had the opportunity to stress it. I also remember that we danced during an evening party. At least on that I was better than her.
After having published some articles on BAM (sometimes criticizing the reproduction of her beautiful figures ...) I experienced her desired for perfectionism and attention to details, also recently when working with her on the recent EJTM article in memory of Professor David Yaffe. Having accepted to act as a BAM / EJTM Advisor, Zipora every two years promised to join the Padova Muscle Days, but this only happened in 2020 when, due to COVID-2019 epidemics, we were forced to organize it by Zoom Sessions. Her SESSION ON SATELLITE CELLS is one of the best in 30 years of Padova Muscle Days, with many excellent speakers and even more audience discussions that can be partially followed at the YOUTUBE link: https://www.youtube.com/c/EASYAGINGinITALYtheMyologyway/videos
Despite the difficult period in Eastern Europe, I hope she will join the 2023 On-site Padua Days on Muscle and Mobility Medicine (2023 PDM3), which will be held from 29 March to 1 April, 2023 at the Thermae of Euganean Hills, Padua, Italy.
In any case, we will remain in contact via e-mail and Zoom Sessions!

Curriculum of Zipora Yablonka-Reuveni

Education

B.Sc., The Hebrew University, Jerusalem, Israel. Awarded with Distinction, 1972, Biology
M.Sc., Feinberg Graduate School, The Weizmann Institute of Science, Rehovot, Israel. Awarded with Distinction. 1975, Life Sciences Ph.D.
Univ. of Windsor, Windsor, Ontario, Canada. Awarded with Distinction. 1979, Biology

Present Position

Professor Emerita 10/2020 to present, Department of Biological Structure, University of Washington School of Medicine, Seattle, WA, USA.

Previous Positions

Graduate Research, Department of Cell Biology, The Weizmann Institute of Science, Rehovot, Israel. In the laboratory of Dr. D. Yaffe; Gene Expression During Differentiation of Muscle Cells, 1973-1976

Graduate Research, Department of Biology, University of Windsor, Windsor, Ontario, Canada. In the laboratory of Dr. A.H. Warner; Control of Protein Synthesis During Development of the Brine Shrimp Artemia salina, 1976-1979

Postdoctoral Research Associate, Department of Zoology, University of Washington, Seattle, WA, USA. In the laboratory of Dr. M.B. Hille; Regulation of Protein Synthesis in Eggs and Embryos of the Sea Urchin, 1979-1982

Postdoctoral Research Associate, Department of Biological Structure, University of Washington School of Medicine, Seattle, WA. In the laboratory of Dr. M. Nameroff, Myogenic Lineage and the Differentiation of Muscle Cells, 1982-1984

Faculty Research Associate, Department of Biological Structure, School of Medicine, University of Washington, Seattle, WA, 1984-1987

Research Assistant Professor, Department of Biological Structure, School of Medicine, University of Washington, Seattle, WA, 1987-1990

Research Associate Professor, Department of Biological Structure, University of Washington School of Medicine, Seattle, WA, 1990-2001

Adjunct Professor, Department of Oral Health Sciences, University of Washington School of Dentistry, 12/2010-present
Member, Graduate School Faculty, University of Washington. 1988-present

Research

Our research focuses on the regulation of myogenic stem cell function in adult life. Our long-term goal is to identify means to ameliorate age-related muscle deterioration (sarcopenia) and combat muscle wasting in muscular dystrophy. We investigate satellite cells, classically defined tissue specific myogenic stem cells that reside beneath the myofiber basal lamina, as well as non-myogenic progenitors associated with the microvasculature that may contribute to myogenesis by myogenic reprogramming.

The following research areas have been pursued

Mechanisms involved in supporting myogenic commitment and renewal of satellite cells. The role of FGF-FGFR system in regulating satellite cells. The role of Klotho genes in the balance between myogenicity and adiposity of skeletal muscle.

Origin and cellular/molecular distinctions of satellite cells from extraocular muscles (EOM) that contribute to enhanced stem cell performance and sparing from muscular dystrophy. Origin and significance of unconventional progenitors that may function to replace myonuclei during myofiber maintenance. Emphasis is given to the role of cells associated with the microvasculature, in particular the pericytes.
Current funding: National Institutes of Health

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49. Shefer, G., and Yablonka-Reuveni, Z. Reflections on lineage potential of skeletal muscle satellite cells: Do they sometimes go MAD? Crit. Rev. Eukaryot. Gene Expr. 2007;17: 13-29.
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posthatch chicken myogenesis. Dev. Dynamics. 2009;238: 1013-1021.

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Invited Reviews & Chapters In Books/Series

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87. Yablonka-Reuveni, Z., Warner, A.H. 1979. Characterization of elongation factor 2 in dormant cysts and developing embryos of Artemia salina. In: Biochemistry of Artemia Development. J. C. Bagshaw and A. H. Warner, Eds., University Microfilm International, Ann Arbor, pp. 22-41.
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89. Yablonka-Reuveni, Z., Nameroff, M. 1986. Immunocytochemical studies on the expression of desmin by dividing cells from skeletal muscle. In: Molecular Biology of Muscle Development, C. Emerson, D.A. Fischman, B. Nadal-Ginard and M.A.Q. Siddiqui, Eds. UCLA Symposia on Molecular and Cellular Biology, New Series 29: 47-60. Alan R. Liss, Inc., New York.
90. Yablonka-Reuveni, Z. 1989. Application of density centrifugation and flow cytometry for the isolation of myogenic and fibroblast-like cells from embryonic and adult skeletal muscle. In: Cellular and Molecular Biology of Muscle Development. L.H. Kedes and F. E. Stockdale, Eds. UCLA Symposia on Molecular and Cellular Biology, New Series 93: 869-879. Alan R. Liss, Inc., New York.
91. Yablonka-Reuveni, Z., Bowen-Pope, D.F., Hartley, R.S. 1990. Proliferation and differentiation of myoblasts: The role of platelet-derived growth factor and the basement membrane. In: The Dynamic State of Muscle Fibers. D. Pette, Ed. Walter de Gruyter & Co., Berlin, pp. 693-706.
92. Yablonka-Reuveni, Z. 1993. Patterns of proliferation and differentiation of adult myoblasts define a unique myogenic population. Prog. Clin. Biol. Res. 383B: 575-585.
93. Grounds, M.D., Yablonka-Reuveni, Z. 1993. Molecular and cell biology of muscle regeneration. In: Molecular and Cell Biology of Muscular Dystrophy. T. Partridge, Ed. Mol. and Cell Biol. Human Diseases Ser. 3: 210-256. Chapman and Hall, London.
94. Yablonka-Reuveni, Z. 2004. Isolation and culture of myogenic stem cells. In: Handbook of Stem Cells - Vol 2: Adult and Fetal Stem Cells, pp. 571-580. R. Lanza, H. Blau, D. Melton, M. Moore, E.D. Thomas, C. Verfaillie, I. Weissman and M. West. Eds. Elsevier: Academic Press, San Diego. ISBN 9780124366435
95. Shefer, G., Yablonka-Reuveni, Z. 2008. Ins and outs of satellite cell myogenesis: the role of the ruling growth factors. In: Skeletal Muscle Repair and Regeneration (Advances in Muscle Research, volume 3), S. Schiaffino and T. Partridge, eds. Springer Netherlands, Chapter 6, pp. 107-144. ISBN: 9781402067679
96. Yablonka-Reuveni, Z., Day, K. 2011. Skeletal muscle stem cells in the spotlight: the satellite cell. In: Regenerating the Heart: Stem Cells and the Cardiovascular System (Stem Cell Biology and Regenerative Medicine Series) I. Cohen and G. Gaudette, eds. Springer, Humana Press. pp. 173-200, chapter 11. http://www.springer.com/life+sciences/book/978-1-61779-020-1

Other Short Publications

97. Greenlee, A.R., Dodson, M.V., Yablonka-Reuveni, Z., Kersten, C.A., Cloud, J.G. 1992. Isolation and culture of trout muscle precursor cells: Application for growth studies. Proc. Idaho Aquaculture Assoc., pp. 27-28.

98. Dodson, M.V., McFarland, D., Bandman, E., Dayton, W.R., Yablonka-Reuveni, Z., Green, E., Doumit, M., Bergen, W., Merkel, R., Vierck, J., Velleman, S., Koumans, J. 1995. Status of satellite cell research in agriculture. Basic and Applied Myology BAM 5: 5-10. Issue on "Satellite cell regulation in agriculturally important animals". Editorial. http://www.bio.unipd.it/bam/bam5-1.html

99. Dodson, M.V., Yablonka-Reuveni, Z., Bandman, E., Grounds, M. 1997. Basic and Applied Myology: A Reflection of our roots and vision for the immediate future. Basic and Applied Myology (BAM) 7: 295-298. Issue on "Satellite cells and myoblast transfer therapy". Commentary. http://www.bio.unipd.it/bam/bam7-3&4.html

GenBank

100. Kwiatkowski, B.A., Fullerton, H.E., Yablonka-Reuveni, Z. 2006. Mus musculus fibroblast growth factor receptor 4 (Fgfr4) mRNA, complete cds. Accession: DQ388428.1

101. Kwiatkowski, B.A., Fullerton, H.E., Yablonka-Reuveni, Z. 2003. Mus musculus fibroblast growth factor receptor 4 minus 16 form mRNA complete cds, alternatively spliced. [FGFR4(-16), mouse FGFR4 form that lacks exon 16]. Accession: AY493377.2

102. Phelps, M., Yablonka-Reuveni, Z. 2013. Mus musculus klotho beta transcript variant 1 (Klb) mRNA, complete cds. Accession: KC810035.1

Theses

103. Yablonka, Z. 1975. Synthesis of myosin subunits directed by RNA from muscle cell cultures. M.Sc. Thesis, pp. 1-58, The Weizmann Institute of Science, Rehovot, Israel.

104. Yablonka-Reuveni, Z. 1979. Characterization of elongation factor 2 during early development of the brine shrimp Artemia salina. Ph.D. Thesis, pp. 1-130, University of Windsor, Windsor, Ontario, Canada (University Microfilm International, Ann Arbor).

INVITED LECTURES & CONFERENCE PRESENTATION

2011-present

- The 2nd Batsheva Seminar on "Integrative Perspectives on the Development of the Musculoskeletal System", Ein Gedi, Israel, February 27 - March 3, 2011
- Forum on Aging and Skeletal Health, American Society for Bone and Mineral Research (ASBMR) and NIH. Bethesda, Maryland, March 21-23, 2011
- The 2nd Polish Joint Congress of Biochemistry and Cell Biology, Krakow, Poland, September 5-9, 2011
- The 2012 Wingate Congress of Exercise and Sport Sciences, The Zinman College, Netanya, Israel, March 15-18
- University of Connecticut Health Center, Farmington, CT, Lawrence G. Raisz Endocrine Scholar Lecture Series, April 17, 2012
- 2012 FASEB Conference on Muscle Satellite and Stem Cells (invited speaker and chair), Il Ciocco, Barga, Italy, August 2012
- 8th European Congress of Biogerontology, Healthy Ageing and Regenerative Medicine, Ben Gurion University, Beer Sheva & Dead Sea, Israel, March 10-13, 2013
- Dept. of Biological Regulation, Weizmann Institute of Science, Rehovot, Israel, March 2013
- MDA (Muscular Dystrophy Association) Scientific Conference, Washington DC, April 21-

24, 2013

- Nationwide Children’s Hospital/OSU/Wellstone Myology Course, Columbus, OH, August 2013
- Stem Cells and Aging: The 2013 San Antonio Nathan Shock Center Aging Conference – Mayan Ranch, Bandera, Texas, October 17-20, 2013
- San Diego Skeletal Muscle Research Center, P30 National Seminar, Depts. of Orthopaedic Surgery and Bioengineering, University of California, San Diego, School of Medicine, December 10, 2013
- FASEB Conference on Muscle Satellite and Stem Cells, Steamboat Springs, Colorado, July 2014
- Eggmeat 2015 Symposium, Nantes, France, May 2015
- International Conference on Skeletal and Cardiac Myogenesis, "Batsheva de Rothschild Workshop on Skeletal and Cardiac Myogenesis", Weizmann Institute of Science, Rehovot, Israel, March 6-11, 2016
- FASEB Conference on Skeletal Muscle Satellite and Regeneration, Keystone, Colorado, July 24-29, 2016
- Fusion Conferences, “2nd Fibroblast Growth Factors in Development and Repair Conference”, Cancun, Mexico, March 8-11, 2017
- University of Minnesota Medical School, Stem Cell Institute Seminar Series, Minnesota, April 29, 2017
- 6th Central European Congress of Life Sciences Eurobiotech, Krakow, Poland, September 11-14, 2017
- Children’s Cancer Therapy Development Institute, Beaverton, Oregon, August 30, 2018

2006-2010

- Gordon Conference on “Fibroblast Growth Factors in Development & Disease”, Ventura, CA, 5/2006
- ”Stem Cells & Regenerative Medicine”, Symposium, Tel Aviv University, Tel Aviv, Israel, 3/2006
- ”Frontiers in Myogenesis meeting”, Society of Muscle Biology, Callaway Gardens in Pine Mountain, Georgia, April 2006
- Workshop on Stem Cells and Aging (Kenneth Day presented), National Institute on Aging, Maryland, May 2006
- Center for Genetic Medicine Research, Children's National Medical Center, Washington, DC, 6/2006
- Boston Biomedical Research Institute, Watertown, Massachusetts, December 2006
- 3rd Seattle Muscular Dystrophy Conference, International meeting, June 13-14, 2007
- Growth and Development Symposium, American Society of Animal Science Annual Meeting, July 2007, San Antonio, Texas
- 2007 FASEB Conference on Muscle Satellite and Stem Cells (invited chair), Indian Wells, California, July 2007
- Age-Related Atrophy: Causes and Mechanisms, Nathan Shock Center, the Barshop Institute for

  Longevity and Aging Studies and the National Institute on Aging. Annual San Antonio Nathan Shock

Center Conference on Aging, October 2007, Mayan Ranch, San Antonio, Texas

- Dept. of Cell and Structural Biology and Barshop Institute for Longevity and Aging Studies, The
  University of Texas Health Science Center at San Antonio, February 2008
- Bioengineering Dept. and Berkeley Stem Cell Center, University of California, Berkeley, April 2008
- Sticht Center on Aging, Wake Forest University Medical Center, Winston-Salem, May 2008
- Understanding Aging: Biomedical and Bioengineering Approaches, hosted by the Methuselah Foundation, UCLA, Los Angeles, California, June 2008
- Division of Molecular Medicine, Wake Forest University Medical Center, Winston-Salem, Sept. 2008
- 2008 American Physiology Intersociety Meeting: The Integrative Biology of Exercise V, Hilton Head, South Carolina, September, 2008
- EMBO Conference: The Molecular and Cellular Mechanisms Regulating Skeletal Muscle Development and Regeneration, Sant Feliu de Guixols, Spain, September, 2008 (Ricardo Almuly presented)
- Dept. of Biological Regulation, Weizmann Institute of Science, Rehovot, Israel, November 2008
- ”Making Muscle in the Embryo and Adult” - Joint meeting of the Society for Muscle Biology (”Frontiers in Myogenesis”) and FASEB (”Skeletal Muscle Satellite and Stem Cells”), Columbia University, New York City, May-June 2009
- 1st International and Interdisciplinary Conference on Skeletal Muscle, Tel Aviv University, Israel, January 26, 2010
- Tulane University HSC - Heart and Vascular Institute, Grand Rounds, April 14, 2010
- 2010 FASEB Summer Conference on Skeletal Muscle Satellite and Stem Cells, Carefree, Arizona,
  July 18-23, 2010
- Children's Hospital Boston, Division of Genetics and Program in Genomics, Boston, MA, October 8, 2010

2001-2005

- Gordon Research Conference on Myogenesis, Il Ciocco, Italy, (invited participant), April 2001
- Department of Animal Science, Faculty of Agriculture, Food and Environmental Quality Sciences, The Hebrew University of Jerusalem, Rehovot, Israel, May 2001
- FASEB Conference on Muscle Satellite and Stem Cells, Tucson, Arizona July 2001
- Symposium on “Adult Stem Cells: Origin and Differentiation”, The 6th Joint Meeting of The Japan Society of Histochemistry & Cytochemistry and The Histochemical Society, Seattle, Washington, July 2002
- Dept. of Molecular Cell Biology, Weizmann Institute of Science, Rehovot, Israel, Sept. 2002
- EMBO Workshop on The Molecular Genetics of Myogenesis and Muscular Diseases,

Churchill College, Cambridge, UK, September 2002

- Workshop on Stem Cells in Aging, (invited discussant), Nathan Shock Center, University of Michigan, Ann Arbor, Michigan, May 2003
- Workshop on Stem Cells and Aging, (invited discussant), National Inst. on Aging, Maryland, May 2003
- "Molecular Biology of Muscle Development and Regeneration", Society of Muscle Biology, The Banff Centre, Banff, Alberta, Canada, April 2003
- Boston Biomedical Research Institute, Watertown, Massachusetts, October 2003
- 13th Conference of the International Society of Differentiation, Honolulu, Hawaii, Sept. 2004
- Workshop on Stem Cells and Aging, National Institute on Aging, Maryland, November 2004
- FASEB Conference on Muscle Satellite and Stem Cells, Tucson, Arizona, June 2005
- Dept. of Marine Biology and Biotechnology, Israel Oceanographic & Limnological Research, Tel-Shikmona, Haifa, Israel, September 2005
- EMBO Workshop on "Molecular and Cellular Mechanisms underlying Skeletal Muscle Formation and Repair", Fontevraud, France, September, 2005
- "From Satellite Cells to Gene Therapy", Clinical Sciences Centre Symposium, The Zoological Society of London, September 2005
- Genentech, San Francisco, California, November 2005

1996-2000

- EMBO Workshop on Myogenesis and Molecular Genetics of Neuromuscular Diseases, Ein-Gedi, Israel, February - March 1996
- Biomed Inc., Seattle, Washington, November 1996
- Proctor & Gamble Pharmaceutical Research Division, Biology Department, December, 1996
- First International Conference on Postnatal Myogenesis: Satellite Cells in Action! Boston, MA, August, 1998
- Gordon Research Conference on Myogenesis. (invited participant),Tilton, NH, August 1998
- Procter & Gamble Pharmaceutical Research Division, International Program for Alternatives to Animal Use, Mason, Ohio, November 1999
- Visiting Lecturer, 14th Annual Visiting Professor Lectureship Series ('99-'00), The Miami Project to Cure Paralysis, International Center for Spinal Cord Injury Research, University of Miami, School of Medicine, January 2000
- International Meeting on Molecular Biology of Muscle Development and Disease, Society of Muscle Biology (formerly Keystone Symposium), Asilomar Conference Center, Pacific Grove, California, May, 2000
- Symposium on the Control of Muscle Growth, 1st International Conference on Muscle-Movement-Contractility Biomechanics & the 21st European Society for Comparative Physiology and Biochemistry, Liege, Belgium, July, 2000

1991-1995

- Keystone Symposia, Gene Expression in Neuromuscular Development, Keystone, Colorado, January 1991

• European Developmental Biology Congress, Jerusalem, Israel, August 1991
• Dept. of Veterinary and Comparative Anatomy, Pharmacology and Physiology, College of Veterinary
Medicine, Washington State University, Pullman, Washington, February 1992
• Faculty of Medicine Seminars in the Basic Sciences, University of Ottawa (Co-hosted by Depts. of Anatomy and Physiology), Ottawa, Ontario, Canada, February 1992
• Symposium on Myogenesis and Somites, Program in Developmental Biology, University of California, San Francisco, California, July 1992
• The fourth International Conference on Limb Development and Regeneration, Asilomar Conference Center, Pacific Grove, California, July 1992
• XXI European Muscle Congress, European Society for Muscle Research, University of Bielefeld, Bielefeld, Fed. Rep. Germany, September 1992
• EMBO Workshop on Molecular Biology and Pathology of Skeletal and Cardiac Myogenesis,Capo Caccia (Alghero, Sardinia) Italy, September 1992
• Strong Children Research Center and the Department of Pediatric Cardiology, School of Medicine, University of Rochester, November 1992
• Keystone Symposia, Molecular Biology of Muscle Development, Snowbird, Utah, April 1994
• Dept. of Membrane Research & Biophysics and Dept. of Cell Biology, Weizmann Institute of Science, Rehovot, Israel, July 1994
• Symposium on Cellular and Molecular Embryology of the Cardiovascular System, Program of Excellence in Molecular Biology and the Cardiovascular Research Institute, at the University of California, San Francisco, California, June 1994
• Faculty of Medicine Visiting Scientist Program and the Dept. of Anatomy, University of Manitoba, Winnipeg, Manitoba, Canada, May 1995
• M.R.C. Clinical Research Centre, Royal Postgraduate Medical School, Hammersmith Hospital,
London, U.K., July 1995
• Musculoskeletal Institute, Children's Hospital of Pittsburgh and Dept. of Orthopaedic Surgery, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, September 1995
• Conference on Molecular Mechanisms Regulating Skeletal Muscle Plasticity, Airlie Conference Center, Virginia, September 1995

1986-1990

• Depts. of Ophthalmology and Cell Biology, School of Medicine, New York University, New York,

New York, January 1986

• The Upjohn Company, Kalamazoo, Michigan, January 1986
• American Cyanamid, Agricultural Research Division, Princeton, New Jersey, June 1986
• Dept. of Cell Biology, Weizmann Institute of Science, Rehovot, Israel, August 1987
• Centre for Biomaterials, University of Toronto, Toronto, Ontario, Canada, May 1988
• Dept. of Animal Sciences, Cook Campus, Rutgers, State University of New Jersey, New Brunswick, New Jersey, March 1989
• 1st International Conference on Myoblasts Transfer Therapy, (invited participant), New York, New York, June 1989

- Faculty of Biology, University of Konstanz, Konstanz, Fed. Rep. Germany, September 1989
- Dept. of Developmental Biology, Univ. of Bielefeld, Bielefeld, Fed. Rep. Germany, September 1989
- Institut d'Embryology, Centre National de la Recherche Scientifique et du College de France, Nogents-sur-Marne, France, September 1989
- International Symposium on the Dynamic State of Muscle Fibers, University of Konstanz, Konstanz, Fed. Rep. Germany, October 1989

1981-1985

- Annual Meeting of the American Association of Anatomists, Seattle, Washington, 1984
- Dept. of Life Sciences, Bar Illan University, Ramat Gan, Israel, June 1985
- Dept. of Biology, Brock University, St. Catherine's, Ontario, Canada, October 1985
- Dept. of Animal and Poultry Science, Univ. of Guelph, Canada, November 1985

1975-1980

- EMBO Workshop on Muscle Cell Cultures in the Study of Gene Expression During Differentiation, Shoresh, Israel, 1975
- Annual Meeting of the Israel Biochemistry Society, Beersheva, Israel, 1976

CHAPTER 13. Inspirers & Supporters

13.7. Terence Partridge

Ugo Carraro ***Terry Partridge***
Padua Muscle Days around 2000, partying in Montagnana. (Photo credit: Ugo Carraro)

I don't remember when and where I met Terry for the first time, perhaps during one of the European Muscle Club Meetings organized by Marcus Schaub. But I know that I was in Tokyo with him for a Muscle Conference organized by Ryoichi Matsuda and that He went to Padua several times for the Muscle Days. Last time was for the PDM3 Virtual presentations, May 26-29, 2021. The COVID-19 pandemic was still very active and was interfering with travel and large gatherings of people. Although he had agreed to schedule his two presentations well in advance, he asked me to move both interviews due to last minute family commitments in London. I did so, of course, being confident that his Zoom Talks would have good acceptance. In fact, his catchy title: "Regeneration of Aged Human Muscle" attracted the maximum number of ZOOM participants and was followed by the longest discussion. Additionally, the full-day “Satellite Cells & Muscle Regeneration: Biology & Pathology”, a Session co-chaired by Zipora Yablonka-Reuveni, University of Washington, Seattle, USA, Terence Partridge, Institute of Child Health, London, UK, and David Israeli, Genethon, France, has probably been the most successful event in 30 years of Padova Muscle Days. I attach below his abstract on xenografts of aged muscle fragments (biopsed from cadavers …) into immunodeficient mice.
Just the experimental plan excited the young audience!

Regeneration of Aged Human Muscle
It is widely speculated that age related muscle atrophy is a result of failure to maintain muscle in the face of loss by damage during day-to-day interaction with the environment. This failure is, in turn, associated with a loss of regenerative capacity by the satellite cell. To examine this idea, we used a previously established model in which fragments of human

muscle regenerate when grafted into immunodeficient mouse hosts [1]. Grafts were obtained during autopsy of cadavers of individuals of a range of ages at death. We found strong regeneration of muscle fragments across the entire range of ages up to 91, suggesting that there was little evidence of a decline of regenerative vigour with age.

1. Zhang Y, et al. Human skeletal muscle xenograft as a new preclinical model for muscle disorders. Hum Mol Genet. 2014. 23(12): 3180-8.

CURRICULUM of Terence Partidge

DATE OF BIRTH 5th. August 1940.

STATUS Married, 2 Children

QUALIFICATIONS

BSc (Special) in Zoology, London University, 1962.
PhD Zoology, London University, 1970.

1959-62, Department of Zoology, University College London.
Undergraduate.
1962-65, Department of Zoology, University College London.
PhD research student- Supervisor, Michael Abercrombie.
1965-66. Laboratoire de Zoologie (Vers), Museum National D'Histoire Naturelle, Paris.
Research Assistant: working on the isolation and characterization of strains of rodent malaria. Director, Professor Alain Chabaud.
1967-70, Department of Cell Biology, University of Glasgow. Assistant Lecturer.
1970-75, Department of Experimental Pathology, Charing Cross Hospital Medical School,London.
Research Fellow supported by the Muscular Dystrophy Group of Great Britain.
Working on the regeneration, transplantation, and pathology of skeletal muscle
1975-78, Lecturer in above department.
1978-89, Senior Lecturer in above department.
1989-92 Reader in Experimental Pathology, Charing Cross & Westminster Medical School
1993-94 Professor of Experimental Pathology, Charing Cross & Westminster Medical School
1994- 2005 Professor of Experimental Pathology,
Head of Muscle Cell Biology Group, MRC Clinical Sciences Centre,Royal Postgraduate Medical School,
December 2005-present
Principal Investigator Center for Genetic Medicine, Children's National Medical Center,
Professor of Integrative Systemic Biology,George Washington University Washington DC

Memberships

Cell Transplantation Society.
American Society for Gene Therapy

Honours

Award of ‘Chaire International de Rechearche Blaise Pascal’ 2004-5

Research Interests

The main thrust of my research over the past 40 years has been the investigation of the cell-biological, and more recently the molecular biological basis of diseases of skeletal muscle. Particular interests are the mechanisms that regulate the repair of muscles and of the control of the activity of muscle precursor cells that underlies this control. Investigation of these matters has largely been conducted by a combination of controlled damage of muscle and of grafting of muscle and its precursors, in alliance with tissue culture techniques that mimic as closely as possible in vivo conditions. Study of this interface between in vivo and in vitro models of myogenic mechanisms has provided extremely detailed information on the behaviour of muscle and its precursors which seems to parallel closely what we can observe at lower spatial and temporal resolution in regenerating muscle in vivo. This strategy has benefited increasingly over the past few years from the availability of a number of molecular biological tools in the form of mice in which particular genes have been targeted or which carry transgenes that act as markers or that alter the behaviour of the system in a discrete manner. My aim over the next few years is to more fully explore these evolving experimental systems.

An additional interest in recent times is that of the mechanisms of exon-skipping in the Duchenne gene. This occurs spontaneously in DMD boys and in the animal models of this disease, and results in the local production of functional dystrophin from the mutated gene. My research group has been in the vanguard of research into this topic, which we suspect is a particularly accessible example of an otherwise under-explored manifestation of highly specific epigenetic control of gene transcription. Over the past couple of years, we have also shown that targeted exon-skipping by use of specific oligonucleotides can achieve the same effect of restoring open reading frame in the dystrophin gene in all of the main body muscles and thus can be applied to partly rescue these muscles from the disease. This rationale is now subject of several trials in Duchenne patients.

At the same time, we have continued with background research to test strategies for regulating dose and evaluating outcome in the mouse and dog models of this disease as well as investigating new chemistries for antisense reagents. In Washington, we are currently undertaking preclinical experiments with new antisense backbone chemistries in collaboration with Professor Luis Garcia’s team at Versailles.

We have also begun to use stable isotope labeling to investigate the turnover dynamics of the various dystrophin proteins that can be achieved by exon-skipping, or that can be delivered by AAV viral vectors, with the idea that such information will be needed to make rationale choices as to which exons to skip in mult-skipping agents and which mini/micro-dystrophins to choose for AAV delivery.

Publications - * Refereed research articles

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2*) Davies, P.S. & Partridge,T.A. (1972). Limpet haemocytes.I. Studies on aggregation and spike formation. J.Cell Sci. 11, 757-769.

3*) Partridge, T.A., Manghani, D. & Sloper, J.C. (1973). Antimuscle antibodies in polymyositis. Lancet, (1973),i,676.

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Letters

- Emphysema in the blotchy mouse: a morphometric study. Reply to letter from M. Wilkinson. J. Path. 157, 156.
- Letter commenting on article by Karpati et al. J. Neuropathol. Exp. Neurol.50, 278-279.
- Letters to Editor on conduct of myoblast transplantation in Cell Therapy Research Foundation. Cell Transplantation 6: 195-196 and 198
- Partridge, T.A., Beauchamp, J., Morgan, J.E., Tremblay, J.P., Huard, J., Watt, D., Wernig, A., Irintchev, A., Grounds, M., Springer, M.L., Bartlett, R.J., Mendell, J., Vilquin, J-T., Bower, J.J. (1997) Letter to Editor. Cell Transplantation , 6, 195-196.
- Partridge, T.A. (1997) Letter to Editor. . Cell Transplantation , 6, 198.Partridge T (2008) Denominator problems in a muscle stem cell study? Cell 135: 997-998; author reply 998-999

Contributor to Gray’s Anatomy, 38th edition, 1995 & 39th edition, 2007.

Consultant on skeletal muscle section of ‘Molecular Biology of the Cell’ 4th Edn. 2002.

Contributor to Encyclopaedia of the Human Genome 2003, 2007, 2012

Book Editorship

“Molecular & Cell Biology of Muscular Dystrophy” commissioned by Publishers Chapman & Hall. Multi-author volume, edited by T. A. Partridge, 1993.

“Multimedia Methods in Molecular Biology”, Editors, T. Partridge & D. Rickwood CD-ROM Published by Chapman & Hall. 1997

“Muscle Regeneration” Co –editor with Stefano Schiaffino, published by Springer 2007.

SCIENTIC ADVISORY BOARDS

Italian Telethon

Boston Biomedical Research Institute

Scientific Advisory Board for AFM (Association Français Contre les Myopathies) on fundamental myology.

CHAPTER 13 Inspirers & Supporters

13.8. Ines Bersch-Porada

Ines Bersch-Porada 2022

Ines Dora Angela Bersch-Porada

Date of birth	12.01.1967
Place of birth	Cologne
Marital status	married
Profession	gratuated Physiotherapist

School education

1973 – 1977	4 years elementary school
1977 – 1986	9 years grammar school with general qualification for university entrance

Professional education

April 1987 – March 1990

Education for physiotherapist (German system) with certification of graduated Physiotherapist

Professional life

May 1990 – October 1991

Physiotherapist General Hospital Glarus/Switzerland

Since December 1991

Physiotherapist at the Swiss Paraplegic Centre Nottwil / Switzerland

1993 – 2014

Substitute chef of the department of Physiotherapy

2014-2018

Therapy Instructor of the department Physio- and Occupational Therapy

Since 2018

Head of the International FES Centre® , Swiss Paraplegic Centre Nottwil, Switzerland

Knowledge of languages

English C1
Italian C1
French B1
Swedish A2

Teaching experience

- since 1992 instructor at the university of applied science for Physiotherapy of Bern for „Peripheral Nerve Lesions", "Functional Electrical Stimulation" and "Spinal Cord Injury"
- since 1998 instructor at the School of Physiotherapy of Zürich for „ Evidenced based therapy and the "Treatment of Neurologic Patients", „Peripheral Nerve Lesions" and "Spinal Cord Injury"
- since 2007 instructor at the university of applied science for Physiotherapy of Basel for "Functional Electrical Stimulation" and "Spinal Cord Injury"
- since 2016 lecturer at the University of Zurich (ETH), "Artificial Breathing"
- since 2018 instructor for Functional electrical stimulation in neurological diseases, university Basel (master students)

Extra-occupational education

Bachelor of Education Science 2005 -2008

Academic Study

May 2012 Master of Science Neurorehabilitation Research (Danube University Krems, Austria) April 2021 PhD in Medical Science (Sahlgrenska Academy, University of Gothenburg)

Thesis: Upper and Lower Motoneuron Lesions in Tetraplegia Diagnostic and Therapeutic Implications of Electrical Stimulation

Further education

- „Funktionelle Bewegungslehre" (Dr.hc. S.Klein-Vogelbach)
- Manual Therapy with diploma
- Vojta Therapy
- Hallwick Methode
- PNF
- Robotics in Rehabilitation
- Ultrasound Imaging, University of Twente
- Survival Statistics: Secrets for Demystifing Numbers, Royal Holloway, University of London
- Discovering Science: Science Writing, University of Leeds
- Good Clinical Practice (GCP) Basiskurs und Aufbaukurs
- Swissmedic-Level: Sub-Investigator und Investigator Swissmedic-Level: Sponsor-Investigator

Personal remarks

- Clinical expertise since 1993 in Functional Electrical Stimulation in upper and lower motor neuron lesions

- Clinical expertise since 1991 in the rehabilitation of patients with spinal cord injury
- Focus on clinical research in spinal cord rehabilitation and application of Functional Electrical Stimulation and direct muscle stimulation
- Board member of the "International Functional Electrical Stimulation Society" (IFESS) since 2014
- Member of the scientific board of the DMGP (German-Speaking Society for Paraplegia) since 2019

Guest lectures

- IMTEK – Department of Microsystems, University Freiburg, Germany, "An interaction of research, treatment and education"
- St. Maartjes Klinik , Netherlands, "Lower motoneuron lesion in spinal cord injury"
- Center för Avancerad Rekonstruktion av Extremiteter (C.A.R.E.), Sahlgrenska Universitetssjukhuset/Mölndal, Sweden, "Lower motoneuron lesion-plexus brachialis"
- Department for Hand Surgery, Lucerne, Switzerland, «Elektrostimulation bei Schädigung des peripheren Nervensytems, Von der wissenschaftlichen Evidenz zur Umsetzung in die klinische Praxis»
- Universitätsklinik Balgrist, Zürich, Switzerland «Elektrostimulation in Diagnostik und Behandlung»

Supervision Master Students

- Sabrina Koch-Borner, «Ist die Kraft der Muskulatur der horizontalen Schulteradduktoren ein Prädiktor für die Ellbogenstreckung nach Trizepsersatzrekonstruktion bei Patienten mit einer Tetraplegie?"
- Marie-Sophie Alberty, „Effects of a high intensity interval training on the skeletal musculature and metabolic markers-A case series"
- Maartje Vletter, "Muscle Fatigue during two FES-cycling Training Modalities in Spinal Cord Injury"

Publication

1. Bersch I, Alberty M, Fridén J. (2022). Robot-assisted training with functional electrical stimulation enhances lower extremity function after spinal cord injury. Artif Organs. Aug 17. doi: 10.1111/aor.14386. Epub ahead of print. PMID: 35976046.
2. Bersch, I., Krebs, J. & Fridén, J. (2022). A Prediction Model for Various Treatment Pathways of Upper Extremity in Tetraplegia. Frontiers Rehabilitation Sciene 3, 889577, doi: 10.3389/fresc.2022.889577
3. Bersch, I. & Fridén, J. (2021) Electrical stimulation alters muscle morphological properties in denervated upper limb muscles. Ebiomedicine published by The Lancet 74, 103737, doi.org/10.1016/j.ebiom.2021.103737
4. Bersch, I. & Fridén, J. (2021). Long-term effect of task-oriented functional electrical stimulation in chronic Guillain Barré syndrome– a single-subject study. Spinal Cord Series and Cases 7, 53.
5. Bersch, I. (2021) Einsatz der Funktionellen Elektrostimulation (FES) in der Neurorehabilitation-Ein Überblick, Orthopädie Technik 10, 2-9
6. Bersch, I. & Fridén, J. (2020). Upper and lower motor neuron lesions in tetraplegia: implications for surgical nerve transfer to restore hand function. Journal of Applied

Physiology 129, 1214–1219.
7. Chandrasekaran, S., Davis, J., Bersch, I., Goldberg, G. & Gorgey, A. S. (2020). Electrical stimulation and denervated muscles after spinal cord injury. Neural regeneration research 15, 1397–1407.
8. Bersch, I., Koch-Borner, S. & Fridén, J. (2019). Motor point topography of fundamental grip actuators in tetraplegia - implications in nerve transfer surgery. Journal of Neurotrauma doi:10.1089/neu.2019.6444.
9. Bersch I, Koch-Borner S, Fridén J. (2018). Electrical stimulation-a mapping system for hand dysfunction in tetraplegia, Spinal Cord 56(5):516- 522.
10. Lampart P, Gemperli A, Baumberger M, Bersch I, Prodinger B, Schmitt K, et al. (2018) Administration of assessment instruments during the first rehabilitation of patients with spinal cord injury: a retrospective chart analysis. Spinal Cord. 56(4):322–31.
11. Laubacher M, Aksöz EA, Bersch I, Hunt KJ. (2017). The road to Cybathlon 2016-Functional electrical stimulation cycling Team IRPT/SPZ European Journal of Translational Myology. 6;27(4)
12. Bersch I, Fridén J. (2016). Role of Functional Electrical Stimulation in Tetraplegia Hand Surgery, Archives of Physical Medicine and Rehabilitation. 97(6 Suppl) :S 154-9.
13. Bersch I, Tesini S, Bersch U, Frotzler A. (2015). Functional Electrical Stimulation in Spinal Cord Injury: Clinical Evidence versus Daily Practice, Artificial Organs 39(10):849-54.
14. Mueller G, Bersch-Porada I, Koch-Borner S, Raab AM, Jonker M, Baumberger M, Michel F. (2014). Laboratory evaluation of four different devices for secretion mobilization: Acapella choice, green and blue versus water bottle, Respiration and Care. 59(5):673-7
15. Tesini S, Frotzler A, Bersch I, Tobón A. (2013). Prevention of Orthostatic Hypotension with Electric Stimulation in Persons with Acute Spinal Cord Injury, Biomedical engineering (Berl).58

Book Chapter Elsevier 2020
Telerehabilitation Principles and Practice (Editor Marcalee Alexander)
Surgical Rehabilitation Across Countries: A Model for Planning in Telerehabilitation
Jan Fridén, Ines Bersch, Fabrizio Fiumedinisi, Sivia Schibli, Sabrina Koch-Borner, Page 363-376
© 2021 Springer-Verlag Berlin Heidelberg
Funktionelle Elektrostimulation in der Neurorehabilitation (Herausgeber Thomas Schick)
Strukturelle und Funktionelle Elektrostimulation bei Schädigung des unteren motorischen Neurons, Seiten 117- 148, Bersch-Porada, Ines
© 2021 Springer-Verlag Berlin Heidelberg
Neuroprosthetics and Brain-Computer Interfaces in Spinal Cord Injury
A Guide for Clinicians and End Users (Herausgeber: Müller-Putz, Gernot, Rupp, Rüdiger (Eds.) Therapeutic Applications of Electrical Stimulation in Spinal Cord Injury
Seiten 253-279, Bersch, Ines Functional Electrical Stimulation in Neurorehabilitation (Herausgeber Thomas Schick) Synergy Effects of Technology and Therapy
Electrical Stimulation for Improvement of Function and Muscle Architecture in Lower Motoneuron Lesions, Page 107- 136

Presented workshops/webinars

- November 2011, Workshop, 2 days “Basics of Functional Electrical Stimulation and Clinical Practice in the Rehabilitation of People with Spinal Cord Injury”, Spinal Cord Unit Murnau (Germany)
- January 2012, „Functional Electrical Simulation from Theory into Practice”, Swiss Paraplegic Centre Nottwil (Switzerland)
- July 2012, „Peripheral Lesions of the Upper Extremities and Functional Electrical Stimulation”, Inselspital Bern (Switzerland)
- September/October 2012, „Biomechanics of the Spine in Seated Position in People with Spinal Cord Injury”, Swiss Paraplegic Centre Nottwil (Switzerland)
- March 2017, “Basics of Functional Electrical Stimulation and Clinical Practice in Rehabilitation of People with an Upper and Lower Motor Neuron Lesion “, Swiss Paraplegic Centre Nottwil (Switzerland)
- April 2017, Workshop, 2 days “Basics of Functional Electrical Stimulation and Clinical Practice in Rehabilitation of People with Spinal Cord Injury “, Spinal Cord Unit, Bad Häring (Austria)
- June 2017, Workshop, 1 day “Basics of Functional Electrical Stimulation and Clinical Practice in Rehabilitation of People with an Upper and Lower Motor Neuron Lesion”, Bale (Switzerland)
- October 2017, Functional electrical stimulation - transfer from theory into clinical practice (part I and part II), Nottwil Switzerland
- January 2018, Functional electrical stimulation in pediatrics, Affoltern am Albis (Switzerland)
- February 2018, Functional electrical stimulation and neuromodulation in the acute phase after spinal cord injury, Innsbruck (Austria)
- November 2019, Electrical stimulation in denervated muscles- Theory and practice (International Course), Nottwil Switzerland
- November 2020, Electrical stimulation for functional improvement of the upper extremity-from clinical reasoning to evidence-based practice (International Course, due to Covid online), Nottwil Switzerland
- November 2020, Electrical Stimulation in Neurological Disease-Electrical Stimulation in Neurological Disease (Webinar organized by IFESS)
- November 2021, Treatment of spasticity with neuromuscular electrical stimulation, (International Course, hybrid format)
- March 2021, Functional Electrical Stimulation of the Upper Limb-Lessons learned from clinical practice (Webinar organized by IFESS)
- March 2022, Pressure Ulcer Prevention, Wound Healing and Electrical Stimulation for People with Spinal Cord Injury (Webinar organized by IFESS)

Oral and Poster Presentation relating to Functional Electrical Stimulation
- IMSOP „International Medical Society of Paraplegia“Nottwil 2001, Switzerland
- ISCOS “International Spinal Cord Society” Reykjavik 2007, Island
- ISCOS “International Spinal Cord Society” Washington 2011, USA„ The importance of the intensity and the number of sessions per week of functional electrical stimulation in patients with post traumatic Spinal Cord Injury: A case report“
- ISCOS “International Spinal Cord Society” London 2012, United Kingdom “Does functional electrical stimulation (FES) of the lower limbs influence the development of bladder dysfunction and autonomic dysreflexia (AD) in traumatic spinal cord

injured patients? “

- „ International Meeting on Surgical Rehabilitation of the Tetraplegic Upper Limb, Hong Kong 2013, Keynote Lecture „Functional Electrical Stimulation in Tetraplegia Hand Surgery “
- 11th Vienna International Workshop on FES, Graz September 2013, “Functional Electrical Stimulation in Spinal Cord Injury: Clinical Evidence versus Daily Practice”
- IFESS “International Functional Electrical Stimulation Society” 2014, Kuala Lumpur, Malaysia,” Functional Electrical Stimulation Transferring the theoretical background into clinical practice”, Proposals for practitioners, 2 hours’ workshop
- ISCOS “International Spinal Cord Society”, Maastricht 2014, Netherlands, “FES of the Upper Limbs, Theoretical Background and Transfer in Daily Practice” Preconference Workshop
- ISCOS “International Spinal Cord Society”, Maastricht 2014, Netherlands, “Prevention of Orthostatic Hypotension with Electric Stimulation in Persons with acute Spinal Cord Injury”
- ISCOS “International Spinal Cord Society “, Montreal 2015, Canada, “Impact of Botulinum toxin A on the function of the upper limb: an outcome analysis”
- IFESS “International Functional Electrical Stimulation Society” 2015, Chicago, USA, “Functional Electrical Stimulation Options - Limits – Barriers, Patients’ and Clinicians’ Perspective”
- IFESS “International Functional Electrical Stimulation Society” 2016, La Grande Motte, France, “Functional Electrical Stimulation (FES): How to best utilize it in clinical practice”, preconference workshop
- IFESS “International Functional Electrical Stimulation Society” 2016, La Grande Motte, France, “FES and Fun” Patients’ Motivation and Goal Attainment, A realistic task?”
- 12th Vienna International Workshop on FES, Vienna 2016, Austria, “Robotic assisted training in combination with functional electrical stimulation to improve lower limb function after spinal cord injury”
- ISCOS “International Spinal Cord Society”, Vienna 2016, Austria, “Robotic assisted training in combination with functional electrical stimulation to improve lower limb function after spinal cord injury”
- ISCOS “International Spinal Cord Society”, Dublin 2017, Ireland, “Robotic assisted training in combination with functional electrical stimulation to improve lower limb function after spinal cord injury”
- IFESS “International Functional Electrical Stimulation Society” 2017, London, United Kingdom, “Bone and Skin Quality after FES Exercise”
- IFESS “International Functional Electrical Stimulation Society” 2018, Nottwil Switzerland, “Stimulation of denervated muscles in patients with a lower motor neuron lesion (LMN) - Implementation in clinical setting” – Conference Host and Organizer
- International Conference for seating and mobility, Nottwil 2018, Switzerland “Functional electrical stimulation, a method to prevent pressure sore in spinal cord injured patients”
- DMGP (German-Speaking Society for Paraplegia), Vienna 2018, Austria, «Was macht der EDC? – Die Bedeutung des M. extensor digitorum communis bei der Entwicklung einer Funktionshand“
- DMGP (German-Speaking Society for Paraplegia), Koblenz 2019, Germany,

«Innerviert – teilinnerviert – Die unterschätzte Innervationsstörung der Fingerflexoren nach Tetraplegie»

- IFESS "International Functional Electrical Stimulation Society"/ Rehabweek 2019, Toronto, Canada, "Different Technologies-how to use them and when in a rehabilitation process"
- 13th Vienna International Workshop on Functional Electrical Stimulation, Vienna 2019, Keynote Lecture "Applications of electrical stimulation in movement rehabilitation in spinal cord injury- From a simple understanding to a complex implementation
- 13th Vienna International Workshop on Functional Electrical Stimulation, Vienna 2019," FES in Spinal Cord Injury-Prevention of pressure sores – skin injury, Prevention of osteoporosis, Amelioration of spasticity, Support of restoration of movement, Maintenance of cardiovascular fitness (Post Conference Workshop)
- ISCOS "International Spinal Cord Society", Nice 2019, France, "Finger flexor innervation-denervation patternin tetraplegia"
- Annual Conference of the German Society for Neurorehabilitation and the

German Society for Neuro-Traumatology and Clinical Neurorehabilitation, December 2020,

Neurorehabilitation im demografischen Wandel

"Digital, Electrical Stimulation in Lower Motoneuron Lesions, Clinical Practice and Scientific Evidence"

- DMGP (German-Speaking Society for Paraplegia), Bochum 2021, Germany, «Roboterunterstütztes Training mit Funktioneller Elektrostimulation in Echtzeit zur Funktionsverbesserung der unteren Extremitäten»
- DMGP (German-Speaking Society for Paraplegia), Bochum 2021, Germany, «Vor- und Nachbehandlung bei Neurotisationstechniken unter Berücksichtigung einer Schädigung des unteren Motoneurons der intrinsischen Muskulatur und Konsequenzen auf die therapeutische Behandlung der Hand bei Tetraplegie»
- IFESS "International Functional Electrical Stimulation Society", Rehabweek 2021 online conference, «Robot-assisted training with functional electrical stimulation enhances lower extremity function after spinal cord injury»
- OT World, Leipzig 2022, Germany, «Elektrostimulation und neue Technologien in der Rehabilitation von Menschen mit Querschnittlähmung»
- IFSSH, London 2022, United Kingdom, «Motor point Mapping – a Diagnostic Assessment for Nerve Transfers»
- DMGP (German-Speaking Society for Paraplegia), Bad Wildungen 2022, Germany, «Veränderung von morphologischen Eigenschaften denervierter Muskulatur der oberen Extremität durch Elektrostimulation
- IFESS "International Functional Electrical Stimulation Society", Rehabweek 2022 Rotterdam, Netherlands, "An interaction of research, treatment and education"
- IFESS "International Functional Electrical Stimulation Society", Rehabweek 2022 Rotterdam, Netherlands, "Damage to the lower motor neuron of the intrinsic musculature and the occurrence of intrinsic tightness in tetraplegic hands"

CHAPTER 13. Inspirers & Supporters

13.9. Christiaan Leeuwenburgh

1990 Chrstiaan Leewenburgh 2022

Christiaan Leewenburgh reached out to me via email at a time when muscle apoptosis was a hot topic nd apoptosis is still central to the biology of aging. But we became pen pals until he decided to become a strong supporter, both scientifically and financially, of Padova Muscle Days in 2010 or thereabouts. He was instrumental to invite speakers from the US and Canada from many Universities including the University of Florida.Then he accepted invitation to be the EJTM Section Editor of “Aging: Biology and Physiology” submitting and soliciting excellent papers.

He is a good friend who always supported my dreams and tried to guide them to great a ccomplishments! To him Ugo he said “Ugo could do nothing wrong because he does not know anyone in this world who moves the field forward as Ugo does and Ugo confronts new challenges and visions”!

CURRICULUM VITAE of Christiaan Leeuwenburgh, PhD

Name:	**Christiaan Leeuwenburgh, PhD**
Rank:	**Chief and Professor**
Department / Center:	**MD-AGING/ GERIATRIC RES-OTHER (PHYSIOLOGY AND AGING)**
College / School:	**MD-AGING / GERIATRIC RESEARCH**

University of Florida
College of Medicine, Department of Physiology and Aging Tel: (352) 273-5735
Institute on Aging and Clinical Translational Science Institute Fax: (352) 294-5836
Clinical and Translational Science Building 3125 Email: cleeuwen@ufl.edu
2004 Mowry Rd, Gainesville, FL 32610 PO Box 100107

EMPLOYMENT

Institution	Position	Dates	Tenure
University of Florida	Professor, College of Medicine, Department of Physiology and Aging (New Department Created at UF)	2022–Present	Tenured
University of Florida	Co-Director KL2 Program, Clinical Translational Science Institute (CTSI)	2021–Present	Non-Tenure-Accruing
University of Florida	Co-Director Professional and Career Development; Training Workforce Development (CTSI)	2015–Present	Non-Tenure-Accruing
University of Florida	Vice-Chair of Research, Department of Aging and Geriatric Research and Institute on Aging	2015–2022	Non-Tenure-Accruing
University of Florida	Professor, College of Medicine, Department of Aging and Geriatric Research	2007–2022	Tenured
University of Florida	Chief, Division of Biology of Aging, Department of Aging and Geriatric Research, Institute on Aging	2006–2022	Non-Tenure-Accruing
University of Florida	Affiliate Faculty and Graduate Faculty Status, Departments of Anatomy and Cell Biology and Biochemistry and Molecular Biology	2005–Present	Non-Tenure-Accruing
University of Florida	Associate Professor, Department of Aging and Geriatric Research, College of Medicine (New Department Created at UF)	2005–2007	Tenured
University of Florida	Associate Professor, College of Health and Human Performance, Department of Applied Physiology	2002–2005	Tenured
University of Florida	Faculty Associate Center for Gerontological Studies and the Institute on Aging	1998–2005	Non-Tenure-Accruing
University of Florida	Director, Biochemistry of Aging Laboratory, College of Health and Human Performance	1999–2005	Non-Tenure-Accruing
University of Florida	Assistant Professor, Department of Applied Physiology, College of Health and Human Performance	1998–2005	Tenure-Accruing

Institution	Position	Dates	Tenure
Washington University School of Medicine	Adjunct Instructor, Department of Internal Medicine, Washington University School of Medicine.	1997–1998	Non-Tenure-Accruing
Washington University School of Medicine	Ruth L. Kirschstein National Research Service Award (NRSA) Individual Fellowship. Washington University School of Medicine	1997–1998	Non-Tenure-Accruing
Washington University School of Medicine	Post-Doctoral Research Associate in Medicine, Department of Internal Medicine, Divisions of Geriatrics and Gerontology and Division of Atherosclerosis, Nutrition and **Lipid** Research	1995–1998	Non-Tenure-Accruing

EDUCATION

1995–1998 Washington University School of Medicine, St. Louis, MO. Department of Internal Medicine, Divisions of Geriatrics and Gerontology, and the Division of Atherosclerosis, Nutrition and **Lipid** Research
Postdoctoral Fellow in Internal Medicine and Geriatrics and Gerontology; Research Associate in Medicine; Adjunct Instructor.

1993–1995 Honorary Fellow and Predoctoral Fellow, American Heart Association, University of Wisconsin, Madison, WI and University of Illinois, Urbana-Champaign, IL (Primary Mentor moved to UW)

1990–1995 PhD (1995), University of Illinois, Urbana-Champaign, IL

1986–1990 BS (1988) and MS (1990), University of Florida, Gainesville, FL

HONORS/AWARDS

2019 University of Florida, Professorship Award

2018 Dr. G. Lombard Kelly Lecturer, Medical College of Georgia, Augusta University.

2017 University of Florida, Professorship Award

2011-2013 University of Florida, Research Foundation Professor

2010 Exemplary Teacher Award, College of Medicine

2004 NIA Nathan W. Shock Lecture Award Winner from the National Institute on Aging
(Nathan W. Shock was a former scientific director of the NIA and an NIH Scientist Emeritus)

2004–2006 University of Florida, Research Foundation Professor

2000-2002 American Heart Association, Young Investigator Award, FL

1999–2000 Merck Geriatric Cardiology Research Award, Society of Geriatric Cardiology

1997–1998 National Research Service Award, NRSA-NIH, National Institute of Aging

1996 Young Investigator Award, Oxygen Society, Intern. Soc. Free Rad. Res., Miami, FL

1994-1995 Honorary Fellow, University of Wisconsin, Madison, WI

1993–1995 American Heart Association, Pre-doctoral Fellowship, Illinois Affiliate

1993 The Avery Brundage Scholarship Award, University of Illinois, Urbana-Champaign, IL

GRANT REVIEW

Date	Location	Work Performed	Organization/ Employer
2022	Mail	Review Scientific Proposal Competitive Research Programme, Prime Minister Office Singapore	National Research Foundation of Singapore, Singapore
2022	Video Conference	UF Jacksonville Scholars Program	UF College of Medicine, Jacksonville
2022	Mail	Austrian Science Fund (FWF); patient oriented clinical research (KLIF) review.	Austrian Science Fund (FWF)
2022	Mail	UTMB Claude D. Pepper Older Americans Independence Center Scientific Review Committee (Pepper-SRC).	UTMB Claude D. Pepper Older Americans Independence Center
2022	Mail	Interdisciplinary Research Programme "The Gerontopole Brussels - Centre of expertise for Gerontology at the Vrije Universiteit Brussel" - Central theme: Active & Healthy Ageing.	The Vrije Universiteit Brussel (VUB), Belgium
2021	Mail	Johns Hopkins University Research Education Core, Pilot, Development Project Claude D. Pepper Older Americans Independence Center	Johns Hopkins University
2021	UF	UF Opportunity Funds; UF Division of Sponsored Research	UF Division of Sponsored Research
2021	Mail	Claude D. Pepper Older Americans Independence Center, Collaborative Pilots	Wake Forest, National Pepper Coordinator Center
2021	Mail	American Federation for Aging Research	AFAR
2021	Video Conference	UF Jacksonville Scholars Program	UF College of Medicine, Jacksonville
2021	Mail Review	Grant proposal review Division of Research & Graduate Studies	The United Arab Emirates University, United Arab Emirates
2020	Video Conference	NIH Study Section ZRG1 MOSS K (02). NIH applications on topics related to skeletal/cardiac muscle biology and diseases	NIH
2020	Video Conference	Advisory Board, To discuss nutritional solutions to modulate age-associated cellular decline for Nestlé Health Science Global Medical Affairs	Nestlé Health Science
2020	Video Conference	Grant reviews for the UF Jacksonville Scholar Program	UF College of Medicine, Jacksonville

Date	Location	Work Performed	Organization/ Employer
2020	Mail	Grant reviews for the American Federation for Aging Research	AFAR
2020	Video Conference	Grant reviews for the UF Opportunity Funds; UF-Division of Sponsored Research	UF Division of Sponsored Research
2020	Mail	Grant reviews for Johns Hopkins University Research Education Core, Pilot, Development Project OAIC	Johns Hopkins University
2020	Mail	Application for promotion to the status of Professor in the Department of Clinical Laboratories, College of Applied Medical Sciences.	King Saud University, Riyadh, KSA
2020	Mail	Grant proposal review Division of Research & Graduate Studies	The United Arab Emirates University, United Arab Emirates
2019	Advisory Board, New York	To discuss nutritional solutions to modulate age-associated cellular decline for Nestlé Health Science Global Medical Affairs	Nestlé Health Science
2019	Mail	Grant reviews for the UF CTSI pilot program	UF CTSI
2019	Video Conference	Grant reviews for the UF College of Medicine Opportunity Funds	UF College of Medicine
2018	Mail	Grant reviews for American Federation for Aging Research	AFAR
2018	Mail	Application for promotion to the status of Professor in the Department of Clinical Laboratories, College of Applied Medical Sciences.	King Saud University, Riyadh, KSA
2017	Mail	Review for College of Medicine; Mauren Post-doctoral Awards	College of Medicine
2016	Mail	Grant reviews for the from the Human Frontier Science Program Organization	Human Frontier Science Program Organization, Strasbourg – FRANCE.
2016	UF	Grant reviews for the University of Florida Southeast Center for Integrated Metabolomics Pilot and Feasibility Projects (SEICM)	SEICM
2016	Mail	Grant reviews for the Michigan Pepper Center Research Education Awards and Pilots	U. Michigan Pepper Center
2016	Mail	Grant Reviews Biotechnology and *Biological Sciences* Research Council (*BBSRC*).	BBSRC, Bioscience for the Future, United Kingdom.

Date	Location	Work Performed	Organization/ Employer
2016	Mail	Grant Review, Scientific Research, Art and Culture, Environment and Social Welfare.	Fondazione Cariplo, an Italian, private philanthropic organization, Milan, Italy
2015	Mail	Grant reviews for Johns Hopkins NIH Pepper Pilot funds review committee	Johns Hopkins University
2015	Mail	Grant reviews for UT Galveston NIH Pepper Pilot funds review committee	UT Galveston
2015	UF in Person	Grant reviews UF Cancer and Aging Pilot Funds review Committee	UF Cancer center
	Mail	Review of Proposal for the NIA Intramural Program (Biology of Aging).	NIH
2015	Mail	Grant review University of Florida Southeast Center for Integrated Metabolomics Pilot and Feasibility Projects (SEICM)	SEICM
2014	GSK symposium on Mitochondria Science, Baltimore	Scientific symposium, to discuss latest on mitochondrial science	GSK
2014	Mail	Review Sir Henry Dale Fellowship,	Wellcome Trust, The Royal Society, England and Wales
2014–2018	External Advisory Board, Los Angeles, Miami and by Video Conference.	*Ad hoc* member of the external advisory board Program Project Grant (PO1) “Mitochondrial Quality Control in Cardioprotection: Overcoming Comorbidities.”	Roberta A. Gottlieb, MD Cedars-Sinai Heart Institute, Barbra Streisand Women’s Heart Center
2014	Video Conference	NIH Study Section Aging Systems and Geriatrics [ASG] study section reviews.	NIH
2014	UF	Grant reviews for the CTSI Clinical Research Pilot Proposals	CTSI
	Video Conference	Grant reviews for the NIH Clinical Trials Review Committee (CLTR)	Clinical Trials Review Committee Office of Scientific Review
2013	UF	Grant reviews for the Opportunity Review Grant Panel; UF Division of Sponsored Research and College of Medicine	Division of Sponsored Research and College of Medicine

Date	Location	Work Performed	Organization/ Employer
2013	External Advisory Board, Winston-Salem	*Ad hoc* member of the external advisory board Wake Forest Pepper Center	Wake Forest University
2012	Mail	Mail Review, Chemical Sciences of the Netherlands Organization for Scientific Research (NWO)	*Dutch* Research Council (*NWO*), Netherlands
2012	Mail	Review for Wittgenstein Award: The Wittgenstein Award is aimed at scientists of any discipline, who are working at Austrian research institutions and who are doing recognized pioneering research	Executive Board of the Austrian Science Fund, Austria
2012	UF	Grant reviews for College Liberal Arts and Sciences (CLAS)	UF Gerontological Grant Reviews CLAS(
2012	UF	CTSI Pilot Projects Grant Reviews	CTSI
2012	Washington DC	NIH SMEP Study section	NIH
2011	Video Conference	NIH MOSS-C03 Review Special Emphasis Panel	NIH
2011	Mail	Grant Reviews for American Federation for Aging Research (AFAR)	AFAR

Grant review prior to 2011. 2010 Netherlands Princes Beatrix Funds, Muscle Diseases, Medical Research Council, United Kingdom, Program Grant Application, The Dunhill Medical Trust, United Kingdom, Grant Application, NIH CMAD Study section, San Francisco, American Federation for Aging Research; 2009;NIH Challenge Grants (Stage I reviewer), Canada Foundation for Innovation (CFI), Netherlands Princess Beatrix Fonds, Muscle Diseases, 2008; ZRG1 CVS-P (02) Center for Scientific Review SEP, Cardiac Metabolism, 2007; ZAT1 SM-07, "National Centers of Excellence for Research on Complementary and Alternative Medicine (CERC)"; 2006, ZAG1 ZIJ-6 NIA Special Emphasis Panel/Scientific Review Group 2006/10, ZAG1 ZIJ-5 NIA Program Project Grants, Special Emphasis Panel/Scientific Review, ZAT1 SM National center for complementary & alternative medicine, American Heart Association, Peer Review Committee (Florida), ZAG1 ZIJ-2 NIA Program Project Grants, Special Emphasis Panel/Scientific Review Group; 2005; ZAG1 ZIJ-5 NIA Program Project Grants, Special Emphasis Panel/Scientific Review Group, ZAG1 ZIJ-2 NIA Special Emphasis Panel/Scientific Review Group, 2004, NIA Special Emphasis Panel/Scientific Review Group, 2003, ZAG1 ZIJ-5 NIA Program Project Grants, Special Emphasis Panel/Scientific Review Group, 2000-2003, American Heart Association, Peer Review Committee (Florida). 2002-2003, Research Committee Society of Geriatric Cardiology

TEACHING

I have taught more than 15 different classes at UF since 1998, including very large required classes (Physiology, Applied Physiology, etc) attended by undergraduates as an assistant/associate professor. Below are my most recent classes **since 2011**, mainly attended by graduate students (in Doctor of Philosophy (PhD) and Master of Science (MS) programs) and professional students (Physician Assistant Program).

Recent Classroom Teaching

GMS 6893-06ED CTSI

GMS 6421 Advanced Cell Biology
GMS 6063 Mechanisms of Aging
GMS 6622 Mitochondrial Biology in Aging and Disease
GMS 6417 Integrative Physiology of Aging
GMS 7593 Neurobiology of Aging
PAS 5020 Introduction to Medicine II Physician Assistant

National and International Collaborations

- ✓ Mary M. McDermott, MD, Jeremiah Stamler Professor, Northwestern University, Feinberg School of Medicine, Deputy Editor, JAMA
- ✓ Stanley Hazen, MD, PhD, Cleveland Clinic, Director, Center for Cardiovascular Diagnostics and Prevention
- ✓ Ian Holt, PhD, Medical Research Council, Mitochondrial Biology Unit, Cambridge, UK
- ✓ Hae Young Chung, Ph.D., Dean College of Pharmacy, Pusan National University, Pusan South Korea
- ✓ Thomas Prolla, PhD, University of Wisconsin-Madison, Departments of Genetics & Medical Genetics
- ✓ Charlotte Peterson, PhD, University of Kentucky, Center on Aging
- ✓ Esther Dupont-Versteegden, PhD, University of Kentucky, Center on Aging
- ✓ Gustavo Barja, PhD, Universidad Complutense, Department of Biology, Madrid, Spain
- ✓ Angela Lezza, PhD and Nicola Maria Gadaleta, PhD, University of Bari, Bari, Italy
- ✓ Colin Selman, PhD, Integrative Physiology, School of Biological Sciences, University of Aberdeen

Patent

Patent No: US 6,541,265 B2; Date of Patent Apr. 1, 2003. "Method and system to test a substance for inflammatory or oxidant properties", Inventor: Christiaan Leeuwenburgh. Assignee: University of Florida, Gainesville, FL (US); Application No. 09/852,194; Filed May 9, 2001.
http://apps.rgp.ufl.edu/otl/pdf/marketing/10523.pdf

FUNDING

ACTIVE

NIH 1 R01 AG075136-01A1 (Leeuwenburgh/Anton) 8/01/2022-7/30/2027
Title: Functional Decline in Low Functioning Older Adults; Role of iron dysregulation

NIH 1RO1AG068458-01A1 (PI: M.M McDermott)
07/01/21-06/30/26 Co-I Cocoa flavanols to improve walking performance in PAD: the COCOA-PAD II Trial. $708,737

NIH/NCATs KL2 TR001429 (MPI Guirgis/Leeuwenburgh), (07/01/21-06/30/24) (MPI)
Institutional Career Development Award $2,487,525

NIH/NIA 2P30AG028740-16 (Contact PI/Project Leader Leeuwenburgh) 07/01/07-03/31/27 PI/PLeader)
The Metabolism and Translational Science Core (RC2)
$185,714

NIH/NIA 2P30AG028740-16 (Contact PI/Project Leader Leeuwenburgh) 07/01/07-03/31/27 PI/PLeader)
The Research Education Core (REC)
$185,714

NIH 1RM1GM139690 (PD/PI: Moldawer, L.L.; Efron, P.A.; Kladde, M; Morel, L) 05/01/21-04/30/26 Co-I

Dysfunctional Myelopoiesis and Myeloid-Derived Suppressor Cells in Sepsis Pathobiology $8,282,350

AHA SFRN 18SFRN33900136 (Leeuwenburgh Project 1) 4/1/2018-3/31/2023 PI

Calf Muscle Mitochondrial Dysfunction and Impaired Autophagy in Peripheral Artery Disease (PAD).

(SFRN Total $3,709,200 through 2023; Project 1 $385,412).

NIH/NIA 1T32AG062728-01A1 (Manini-Leeuwenburgh) 05/01/20-04/30/25 co-PI

Translational research training on aging and mobility (TRAM) program $148,766

NIH/NIHR R01NR016986 (Stechmiller/Lyon) 4/1/2018-6/30/2023 Co-I

Biobehavioral mechanisms underlying symptoms and healing outcomes in older individuals with CVLU $585,881

AHA SFRN 18SFRN33900136 Pilot (Leeuwenburgh) 7/1/2020-6/30/2022 PI

Discovery and validation of miRNAs concurrently in plasma and skeletal muscle in subjects PAD $40,615

6/30/2026 Co-I

COCOA flavanols to improve walking performance in PAD: the COCOA-PAD II Trial $4,068,135

NIH GM RO1133815 (Guirgis) 4/1/2020-3/31/2025 Co-I

The Role and Mechanisms of Lipid and Lipoprotein Dysregulation $495,400

NIH R01AG057693 (McDermott) 8/1/2018-4/30/2023 Co-I

INTERmittent pneumaticomprEssion for Disablility rEversal in PAD: the INTERCEDE Study $3,083,190

NIH RO1AR072328 (Martin/Smith/Beaver) 7/1/2017-5/31/2022 Co-I $1,652,592

In this study, we will determine if intraoperative electrical diaphragm stimulation attenuate early VIDD manifestations in humans.

U24 AR071113 NIH/NIA (Pahor) 12/06/2016-11/30/2022 Co-I

Molecular Transducers of Physical Activity Consortium (MoTrPAC) Consortium Coordinating Center. $20,283,331

NIH 1U01AG055137 (Esser) 12/15/2016-11/30/2022 Co-I PASS (Physical Activity Preclinical Study Sites): Regulation of exercise transducers. $2,279,187

NIH 1R33AG056540 (Pahor, Anton) 9/15/17-5/31/22Co-I

The University of Florida Jacksonville Aging Studies Center (JAXASCENT) $2,958,699

P30 AG028740 (Pahor) 4/01/2007 – 3/31/2022 Co-I

National Institutes of Health/National Institute on Aging $15,018,744

Claude D. Pepper Older Americans Independence Center (OAIC)

PI Metabolism and Translational Science Core and the Research Education Core
NIH R01 (McDermott) 12/1/2016-11/30/2021 Co-I
Improve PAD Performance with Metformin: The PERMET Trial
$3,624,780
KL2 TR001429 CTSI KL-2 (Pearson) 8/1/2015-7/31/2021
Program Mentor
National Institutes of Health Clinical and Translational Science Awards Program
$3,298,627
The CTSI KL2 Multidisciplinary Scholars Program is a research training and funding opportunity for junior faculty at UF to foster a career in clinical/translational research.
NIH 1UG3 HL141729-01A1 (McDermott) 4/1/2019-3/31/2025
Co-I
PROmote weight loss in obese PAD patients to preVEnt mobility loss: The PROVE Trial.
$1,242,184
NIH/NIA R21 AG064282 (Mankowski) 9/01/2019-5/31/2022
Co-I
Nicotinamide riboside as an Enhancer of Exercise Therapy in hypertensive older adults: The NEET Trial $429,957
University of Florida Health Cancer Center Pilot Fund (Zhang) 10/20/2021-10/20/2023
Co-I
"Nicotinamide riboside and walking exercise intervention to reduce fatigue in older breast cancer survivors-A pilot trial" $100,000

PENDING

NIH/NIA (Leeuwenburgh, Anton; score 13; 1%) 12/01/2021-11/30/2026 PI Functional Decline in Low Functioning Older Adults; Role of iron dysregulation the proposed study, we will examine cross-sectional and longitudinal associations of dysfunctional iron regulation with levels of Mt and physical function.
NIH/NIA (McDermott score 23, 6.0%) R21 AG080426-01, entitled Far Red Light to Improve Functioning in PAD: the LIGHT PAD Trial.
NIH (Yoon) 1R01CA230448-01 4/1/2021-3/31/2025 Co-I
SAFEWIC- Supportive Acupuncture for Enhancing Weight in Gastrointestinal Cancer Cachexia
NIH R21AG077096 (Zhang) 04/01/2022-03/31/2024
"Nicotinamide riboside and walking exercise intervention to reduce fatigue in older breast cancer survivors" Total budget: $275,000 Score: 44/Percentile: 47

TRAINING GRANTS/Current Past
NIH KL2 TR001429 CTSI KL-2 (Leeuwenburgh/Guirgis) 8/1/2015-7/31/2021 MPI
National Institutes of Health Clinical and Translational Science Awards Program
$3,298,627
NIH/NIA T32 AG062728 (Manini-Leeuwenburgh) 05/01/20-04/30/25 Co-PI
Translational research on aging and mobility (TRAM) program $148,766/y
NIH 1R33AG056540 (Pahor, Anton) 9/15/17-5/31/22 Co-I
The University of Florida Jacksonville Aging Studies Center (JAXASCENT) $2,958,699
1K01AG048259-01A1 (Cruz-Almeida) (Mentor) 5/15/2015-4/30/2020 Co-Primary Mentor
Title: Neuroimaging age-related changes in pain modulation $831,442
T32 HD043730 NIH (Vandenborne/Fuller) 6/11/2003-4/30/2021
Mentor/Advisory Board

Training in Rehabilitation and Neuromuscular Plasticity $3,624,422
NIH K23GM115690 (Guirgis) 9/23/2016-8/31/2020 Co-Primary Mentor
The Role of Dysfunctional HDL in Sepsis
$699,289
NIH NIAMS K23AR061146 K Vincent (PI) 07/01/2012-06/30/2017
(Primary Mentor)
Comparative Resistance Exercise Effects on Knee Osteoarthritis Pain, Functional Impairment and Cartilage Turnover. $374,933
NIH K23AR062099, (Sibille) 07/01/2012-06/30/2017 (Co-Primary Mentor)
Biological Markers of System Burden in Symptomatic Knee OA: A Prospective Study $472,510
NIH T32HL083810 (Wood/Baylis) 9/1/2007-8/31/2017 Mentor
Multidisciplinary training program in hypertension $2,522,919
NIH K01HD052713 (Chmielewski) (Leeuwenburgh 9/30/07-8/31/13 Co-Primary Mentor)
National Institutes of Health $526,835
Muscle Weakness and Post-Traumatic Knee OA
NIH K23AT004251 (Anton) 12/1/09 – 11/30/14 Co-Primary Mentor
Investigations of Botanicals on Food Intake, Satiety, and Weight Loss $574,814
10PRE4310091, AHA Fellowship to Priya Dutta (Leeuwenburgh, Primary Mentor), 07/01/2010-06/30/2012
Mitochondrial Dysfunction and the Role of Autophagy in Cardiomyocytes $43,400
AHA 2060112 AHA Fellowship to Jinze Xu (Leeuwenburgh Primary Mentor)
07/01/2009-06/30/2011
Cardiac mitochondrial iron transport and accumulation and the effects on bioenergetics with age. $100,040
T32 AG000196-20 (P Scarpace) 2007-2012 Mentor
Training in the Neurobiology of Aging $2,799,650
AHA Fellowship to Arnold Seo 0615256B (Leeuwenburgh)
07/01/2006-06/30/2008
Cardiac mitochondrial biogenesis and macro-autophagy $21,770/yearly
AHA: Fellowship to Tim Hofer 0525346B (Leeuwenburgh) 7/01/05-6/30/2007
Oxidative RNA and DNA damage to heart mitochondrial sub-populations with age and life-long calorie restriction. $96,476.
0415166B (Leeuwenburgh)
AHA Fellowship to Asimina Hiona $80,000
The use of p66Shc knockout mice to investigate the mechanisms responsible for cardiomyocyte apoptosis with age. P66Sch is a protein which regulates mitochondrial oxidative stress and it's role in aging is investigated.
0415187B; American Heart Association (Leeuwenburgh Fellowship for Young Mok Ja
2004-2006
Sarcoplasmic Reticulum Mediated Apoptosis in Cardiotoxicity induced by Doxorubicin in vitro and in vivo. $80,000
0225194B, American Heart Association (Fellowship for Barry Drew) 2002-2004
Doxorubicin-induced damage to cardiac mitochondrial enzymes
$72,000
Goal: Attenuating doxorubicin-induced damage to cardiac mitochondrial enzymes.
0215053B, American Heart Association (Fellowship for Sharon Phaneuf) 2002-2004
Lifelong, voluntary exercise as a strategy to prevent mitochondrial-mediated cardiomyocyte apoptosis with age
Goal: Attenuating apoptosis in the heart with life-long moderate exercise training. $76,000
National Institute of Aging; National Research Service Award (NRSA) (Leeuwenburgh) 1997-1998
NIA/NRSA; 1F32AG05780-01, Molecular Mechanism of Oxidative Stress in Aging $51,000

University of Illinois
Pre-Doctoral Fellowship AHA SS-08, American Heart Association, Illinois Affiliate 1993-1995
Myocardial Ischemia-Reperfusion Injury in vivo (Leeuwenburgh) $24,000

Prior to 2020.

NIH R01 DC014437 (Someya) 4/1/2015-3/31/2020 Co-I
Cochlear detoxification system $2,239,816

NIH R01 GM113945 (Efron) 4/1/2015-1/31/2020 Co-I
Hematopoietic stem cell dysfunction in the elderly after severe injury $1,576,905

NIH 1R01 HL126117-01 (McDermott) 7/1/2015-6/30/2020 Co-I
TELmisartan plus EXercise to improve function in PAD: The TELEX Study $3,738,470

NIH U01AG050499 (Pahor) 7/1/2015-6/30/2018 Co-I
ENRGISE- Enabling reduction of low-grade inflammation in seniors
$5,515,881

NIH R21AG050897 (McDermott) 6/15/2016-2/28/2018 Co-I
COCOA to improve walking performance in Peripheral ARter Disease: The COCOA-PAD Study $615,378

NIH R01 HL122846 (McDermott) 4/1/2015-1/31/2018 Co-I
Low intensity exercise intervention in peripheral artery disease - The LITE Trial
$2,990,679

P50 GM111152 NIH (Moore) 9/1/2014-5/31/2019 Co-I
PICS: A New Horizon for Surgical Critical Care
$10,747,256
Role: PI Core C; Co-I Project 2; Co-PI Project 4

1R01DK099334 NIH (Cohen) 6/25/2014-5/31/2019 Co-I
Obesity and type-2 diabetes: Bariatric surgery effects of brain function
$2,663,490

U01-AG022376 NIH/NIA (Pahor) 9/1/2009-11/30/2018 Co-I
Physical Exercise to Prevent Disability – LIFE Study
$83,338,482

NIH 1R01DC012552 (Someya) 7/1/2013-6/30/2019 Co-I
Mitochondrial thioredoxin, caloric restriction, and age-related hearing loss $1,859,650

1 R01 AT007564 (Anton) 4/30/2014-8/31/2018 Co-I
REVIVE - Resveratrol to Enhance Vitality and Vigor in Elders (REVIVE) $1,411,746

R01 AG042525 (Tranah/Manini) 7/15/2013-6/30/2018 Co-I
MtDNA variant modifiers of cardiopulmonary responsiveness to physical activity
$1,615,097
This project will identify mtDNA variants that predispose older individuals to a high or low cardiopulmonary response to chronic exercise.

R01 DK079879-10 (Kim) 9/22/2014-8/31/2019 Co-I
Autophagy in Liver injury $3,230,225

RO1 DK090115 (Kim-Leeuwenburgh) 4/1/2012-3/31/2017 Co-PI
Mitophagy: A novel target to improve liver function after ischemia/reperfusion injury
$1,263,400

R21 AG047510 NIH (McDermott) 5/15/2014-4/30/2017 Co-I
Resveratrol to improve outcomes in older people with PAD (The RESTORE Trial) $532,732

Osato Research Institute (Anton-Leeuwenburgh) 07/1/2013 – 6/30/2016 Co-I
Efficacy of fermented papaya preparation (FPP) in improving health and physical

function in older adults with mild functional limitations. $187,778
UFRF (Sunny) 5/1/2013-4/30/2015 Co-I
Mitochondrial dysfunction in nonalcoholic fatty liver disease (NAFLD): Mechanisms and role of insulin resistance and oxidative stress $90,386
1 P30 AG028740-01Pepper Pilot (Efron) 7/1/12-3/31/14 Co-I
Project Title: Emergency myelopoiesis in sepsis and polytrauma and its potential impact on the elderly
1 P30 AG028740-01 Pepper Pilot (Joseph) 7/1/12-3/31/14 Co-I
Project Title: Aging induced pluripotent stem cell (iPSC) study
1 P30 AG028740-01Pepper Pilot (Tornaletti) 7/1/12-3/31/14 Co-I
Project Title: DNA Repair in the Aging Heart
Eli Lilly and Company (Martin/Leeuwenburgh) 12/17/2012-6/30/2014 Co-I
Identification of biomarkers of diaphragmatic dysfunction in mechanically ventilated patients
RO1 AG17994-10 NIH (Leeuwenburgh) 8/1/2005-6/30/2012
National Institutes of Health/National Institute on Aging $2,892,619
Project Title: Molecular Mechanisms of Oxidative Stress in Aging Muscle
CTSI (Leeuwenburgh/Martin) 12/1/2009-11/1 2012 Co-PI
Diaphragm Mitochondrial Dysfunction During Prolonged Mechanical Ventilation. $91,738
NIA R01AG14979 (Foster) 6/1/2007-5/31/2012 Co-I
National Institutes of Health/National Institute on Aging $2,689,723
Mechanism for Altered Synaptic Function During Aging
NIH/NIA (Cummings) 9/30/2009-9/29/2011 Co-I
Study of Energy and Aging (SEA) $855,594
American Heart Association (Anton) 7/1/09 – 6/30/11 Co-I
Dose Response Effects of Weight Loss on Systemic and Vascular Inflammation $110,000
1R01-AG024526 NIH/NIA (Carter) 8/1/2005-7/31/2011 Co-I
ACE Inhibition and Physical Performance in Aged Rats $1,250,000
1P30-AG028740-S2 (Manini/Wohlgemuth) 10/1/2009-9/31/2011 Co-I
Mitochondrial function and fatigue in the elderly $150,000
UF CTSI Pilot Grant (Fillingim) 11/16/2009-11/15/2011 Co-I
Effects of OA-Related Pain on Telomere Length and Telomerase Activity $77,876
Merck & Co, Inc (Buford) 2010-2011 Co-I
Role of skeletal muscle blood flow and regeneration in sarcopenia $60,000
Investigate the role of skeletal muscle angiogenesis and perfusion on inflammation, extracellular matrix remodeling and satellite cell number in young and old persons.
Nestle Purina Pet Care Global Resources, Inc. (Leeuwenburgh) 3/15/2009-3/15/2011
DNA/RNA oxidation analysis in blood, urine and tissue $7,375
The Evelyn F. and William L. McKnight Brain Institute (Manini & Anton) 02/01/2008-01/31/2009 Co-I
Resveratrol supplementation to improve memory dysfunction in older adults $100,000
Institute on Aging (Anton) 7/1/2008-6/30/2010 Co-I
Dose-response effects of weight loss on oxidative stress and inflammation
NIH/NIA (Pahor/Manini) 7/1/2008-6/30/2010 Co-I
Pepper Center Supplement - Molecular mechanisms of skeletal muscle loss in HIV-infected older persons
Brooks Rehabilitation Research Endowment (Chmielewski) 4/01/07-2/28/2010 Co-I
Brooks Healthcare System $40,000
Urinary Levels of a Knee Osteoarthritis Biomarker in the Early Period after ACL

Reconstruction
NFL Charities (Chmielewski) 7/1/2007-6/30/2010 Co-I
The Effect of Plyometric Exercise Intensity on Function & Articular Cartilage Metabolism after ACL Reconstruction $125,000
Joint Cancer Centers Opportunity Fund (Manini) 05/01/09 – 04/31/10 Co-I
UF/Moffitt cancer centers $93,744
Chemotherapy-induced muscle weakness, fatigue & functional limitation in older breast cancer survivors
Role: Dual-Principal Investigator (Co-PI: Martine Extermann, MD).
Sharper Contract - Eufortyn Study (Leeuwenburgh) 11/15/2007-11/14/2009 PI
USDA/NRICGP (Kristinsson) 09/01/06-08/31/09 Co-I
Function, characterization and stability of antioxidative hydrolysates and peptides made from proteins isolated from fish processing byproducts.
RO1 AG 21042 (Leeuwenburgh) 8/01/2003-7/31/2009
National Institutes of Health/National Institute on Aging $1,675,560
Apoptosis and life-long caloric restriction
The National Institute on Aging: (Aspirin proposal, Leeuwenburgh) 2005-2008
Intervention testing program for compounds to test their ability to extend mean and/or maximum life-span
LifeGen Technologies (Leeuwenburgh) 12/01/06-115/4/2009 PI $44,394
This research project is designed to measure oxidative stress with 8-OH-Dg (DNA) and 8-OH-G (RNA) levels in canines and mice.
RGP Opportunity Fund (M. Perri) 5/1/06-4/30/07 Co-I $81,876
Biological Effects of Weight Loss Plus Exercise in Obese Older African-American Women
James and Esther King Program (Segal/Johnson) 7/1/05 – 6/30/07 PI (project 4)
FL Department of Health $73,179
James and Esther King Program
Smoking as a novel risk factor for progression of renal disease
This study will elucidate the mechanisms of renal disease due to smoking.
2RO1 AG17994 NIH (Leeuwenburgh) 08/01/00-7/31/06
Molecular Mechanisms of Oxidative Stress in Aging Muscle $2,892,619
The major goals for this project are to study mitochondrial function, energy production and oxidative stress with age in cardiac and skeletal muscle.
American Heart Association (Florida). 6/1/2000-5/31/2003 PI
Scientist Development Grant AHA 0030334B (Leeuwenburgh) $225,000
Doxorubicin-induced oxidative stress and apoptosis in cardiac myocytes: The role of the mitochondria.
Galileo-IRB 658-2000 (Leeuwenburgh) 2001-2002 $123,750
A Single Center Double Blind Placebo Controlled Study of Nutritional Ingredient Systems in Post-Exercise Muscle Injury to Assess Symptomatic Response and Surrogate Markers of Oxidative Stress and Inflammation
NIA, AG 10485 (Meyer) 8/01/1999-7/31/2004 Co-I
Program Project Grant National Institute of Health $5,217,615
Discovery of novel drugs for Alzheimer's disease
Co-Investigator Neurochemistry Core (Leeuwenburgh)
Florida Department of Health: Biomedical Research Program (Powers) 2001-2003 Co-I
Exercise and myocardial protection against I-R injury $355,525
Goal: To determine the mechanisms of exercise-induced cardiac protection.
RO1 HL62361 (Powers) 2/1/2001-1/31/2005 Co-I
NIH-National Heart, Lung and Blood Institute $1,268,750
Mechanical ventilation and respiratory muscles
RO1 HL607855 (Powers) 2003-2006 Co-I
NIH - National Heart, Lung and Blood Institute $1,310,990
Exercise, Antioxidants, and I-R Injury
Society of Geriatric Cardiology, Merck Geriatric Cardiology Research Award (Leeuwenburgh)

1999-2000
Myocardial Aging: Mitochondrial Control of Apoptosis? $10,000
Cause for Ventricular Dysfunction and Failure in the Old Heart?
American Heart Association-Florida affiliate (Powers) 1998-2001
Protective strategies against myocardial ischemia-reperfusion injury $109,388
Washington University School of Medicine
NIH-NIA, 1 P60 AG 1362901 Claude Pepper OAIC (Holloszy) 1995-2000
Pilot Project Principal Investigator (Leeuwenburgh)

SERVICES

Administrative Duties: University, College and Departmental

2005- Chief Division of Biology of Aging
Age-Related Memory Loss Program/MBI-ARML program (bi-Annual Meeting)
Dean's Executive Meeting (select monthly meetings only)
VP HSC Executive Meeting (select monthly meeting only)
2005- Seminar co-Director (UF-VA Aging Rehabilitation Seminar Series and Department of Aging and Geriatrics Seminar series) (Weekly Fall and Spring)
2013 Conflict of Interest Management Plan Okunieff (Chair, Stephen Sugrue) (Company vs UF staff conflict solution monitoring plan development)
2014 Investigative Clinical Trials Misconduct Committee, DSR.
2014-2015 Organize yearly Institute on Aging Research Day
2010- K-College Round Table CTSI-Pepper Center Scholar's Meeting (monthly meeting)
2005- Institute on Aging Executive Board meetings (monthly meeting)
2006- Core Leader of the Pepper Center Metabolism and Translational Science Core 2006-current (Monthly Meeting)
2015- HSC and CTSI Director of the Professional Development Programs which includes CTSA-sponsored seminars, roundtable workshops with all KL-2 trainees and research day activities
2015- Vice-Chair of Research Department of Aging and Geriatric Research, Institute on Aging.

Department Search Committee's
Search Committee Muscle Biology Preeminence (Full/Associate/Assistant Professor).
Search Committee Cancer Biology.
Search Committee and Chair Translational Science Position II (Full/Associate/Assistant Professor).
Search Committee Department of Aging and Geriatric Research (Assistant Scientist).

Board Member
American Aging Association (AGE) 2005-2010
American Aging Association (AGE) 2013-2018
American Federation for Aging Research (AFAR Florida 2010-2012)
Methuselah Prize Scientific Advisory Board (MPSAB)
"The McKnight Brain Institute CAM Basic Science Funds Board"2005
Wake Forest Pepper Center External Board 2012
American College of Sports Medicine and The American Physiological Society (APS) 1997
Society for Free Radical Biology and Medicine (SFRBM) 1995-2008
International Society for Free Radical Research (ISFRBM) 1995-2008

Editor
2019-current Editor-in-Chief Experimental Gerontology
2008-current Executive Deputy Editor Journal of Experimental Gerontology (Editor

Musculoskeletal Systems and Exercise, Section Editor) Promoted to Executive Deputy Editor 2013).

2021-2022 Cells, Special Issue Editor “Autophagy Meets Aging”

2012- Associate Editor Journal of Frailty and Aging Section "Biology of Frailty and Aging"

2010-2011 Guest Editor Journal of Aging Research, Mitochondria and Ageing

2005-2006, Guest Editor Antioxidant and Redox Signaling

Editorial board

Journal of the American Aging Association (AGE) 2004-

Basic Applied Myology - Advisory Board Editor 2010

Editorial Board: Aging Reviews 2011-

PUBLICATIONS

Refereed Publications (295) – Selected PubMed 2022 and EJTM papers

1. Fielding RA, Atkinson EJ, Aversa Z, White TA, Heeren AA, Achenbach SJ, Mielke MM, Cummings SR, Pahor M, Leeuwenburgh C, LeBrasseur NK. Associations between biomarkers of cellular senescence and physical function in humans: observations from the lifestyle interventions for elders (LIFE) study. Geroscience. 2022 Nov 11. doi: 10.1007/s11357-022-00685-2. Epub ahead of print. PMID: 36367600.
2. Saini SK, Pérez-Cremades D, Cheng HS, Kosmac K, Peterson CA, Li L, Tian L, Dong G, Wu KK, Bouverat B, Wohlgemuth SE, Ryan T, Sufit RL, Ferrucci L, McDermott MM, Leeuwenburgh C, Feinberg MW. Dysregulated Genes, MicroRNAs, Biological Pathways, and Gastrocnemius Muscle Fiber Types Associated With Progression of Peripheral Artery Disease: A Preliminary Analysis. J Am Heart Assoc. 2022 Nov;11(21):e023085. doi: 10.1161/JAHA.121.023085. Epub 2022 Oct 27. PMID: 36300658.
3. McDermott MM, Bazzano L, Peterson CA, Sufit R, Ferrucci L, Domanchuk K, Zhao L, Polonsky TS, Zhang D, Lloyd-Jones D, Leeuwenburgh C, Guralnik JM, Kibbe MR, Kosmac K, Criqui MH, Tian L. Effect of Telmisartan on Walking Performance in Patients With Lower Extremity Peripheral Artery Disease: The TELEX Randomized Clinical Trial. JAMA. 2022 Oct 4;328(13):1315-1325. doi: 10.1001/jama.2022.16797. PMID: 36194220; PMCID: PMC9533188.
4. Hammond MM, Spring B, Rejeski WJ, Sufit R, Criqui MH, Tian L, Zhao L, Xu S, Kibbe MR, Leeuwenburgh C, Manini T, Forman DE, Treat-Jacobson D, Polonsky TS, Bazzano L, Ferrucci L, Guralnik J, Lloyd-Jones DM, McDermott MM. Effects of Walking Exercise at a Pace With Versus Without Ischemic Leg Symptoms on Functional Performance Measures in People With Lower Extremity Peripheral Artery Disease: The LITE Randomized Clinical Trial. J Am Heart Assoc. 2022 Aug 2;11(15):e025063. doi: 10.1161/JAHA.121.025063. Epub 2022 Jul 27. PMID: 35894088; PMCID: PMC9375509.
5. Zhang D, Leeuwenburgh C, Zhou D, Gong Y, Pahor M, Licht JD, Braithwaite D. Analysis of Biological Aging and Risks of All-Cause and Cardiovascular Disease-Specific Death in Cancer Survivors. JAMA Netw Open. 2022 Jun 1;5(6):e2218183. doi: 10.1001/jamanetworkopen.2022.18183. PMID: 35731518; PMCID: PMC9218849.
6. Zhang D, Mobley EM, Manini TM, Leeuwenburgh C, Anton SD, Washington CJ,

Zhou D, Parker AS, Okunieff PG, Bian J, Guo Y, Pahor M, Hiatt RA, Braithwaite D. Frailty and risk of mortality in older cancer survivors and adults without a cancer history: Evidence from the National Health and Nutrition Examination Survey, 1999-2014. Cancer. 2022 Aug 1;128(15):2978-2987. doi: 10.1002/cncr.34258. Epub 2022 May 24. PMID: 35608563; PMCID: PMC9671088.

7. Saini SK, Singh A, Saini M, Gonzalez-Freire M, Leeuwenburgh C, Anton SD. Time-Restricted Eating Regimen Differentially Affects Circulatory miRNA Expression in Older Overweight Adults. Nutrients. 2022 Apr 28;14(9):1843. doi: 10.3390/nu14091843. PMID: 35565812; PMCID: PMC9100641.
8. Carraro U, Bittmann F, Ivanova E, Jónsson H Jr, Kern H, Leeuwenburgh C, Mayr W, Scalabrin M, Schaefer L, Smeriglio P, Zampieri S. Post-meeting report of the 2022 On-site Padua Days on Muscle and Mobility Medicine, March 30 - April 3, 2022, Padua, Italy. Eur J Transl Myol. 2022 Apr 13;32(2):10521. doi: 10.4081/ejtm.2022.10521. PMID: 35421919; PMCID: PMC9295170.
9. Picca A, Guerra F, Calvani R, Romano R, Coelho-Junior HJ, Bucci C, Leeuwenburgh C, Marzetti E. Mitochondrial-derived vesicles in skeletal muscle remodeling and adaptation. Semin Cell Dev Biol. 2022 Mar 30:S1084-9521(22)00095-7. doi: 10.1016/j.semcdb.2022.03.023. Epub ahead of print. PMID: 35367122.
10. Efron PA, Darden DB, Li EC, Munley J, Kelly L, Fenner B, Nacionales DC, Ungaro RF, Dirain ML, Rincon J, Mankowski RT, Leeuwenburgh C, Moore FA, Brakenridge SC, Foster TC, Laitano O, Casadesus G, Moldawer LL, Mohr AM, Thomas RM. Sex differences associate with late microbiome alterations after murine surgical sepsis. J Trauma Acute Care Surg. 2022 Aug 1;93(2):137-146. doi: 10.1097/TA.0000000000003599. Epub 2022 Mar 24. PMID: 35324554; PMCID: PMC9323556.
11. Mankowski RT, Anton SD, Ghita GL, Brumback B, Darden DB, Bihorac A, Leeuwenburgh C, Moldawer LL, Efron PA, Brakenridge SC, Moore FA. Older Adults Demonstrate Biomarker Evidence of the Persistent Inflammation, Immunosuppression, and Catabolism Syndrome (PICS) After Sepsis. J Gerontol A Biol Sci Med Sci. 2022 Jan 7;77(1):188-196. doi: 10.1093/gerona/glab080. PMID: 33721883; PMCID: PMC8751807.
12. Przkora R, Sibille K, Victor S, Meroney M, Leeuwenburgh C, Gardner A, Vasilopoulos T, Parvataneni HK. Blood flow restriction exercise to attenuate postoperative loss of function after total knee replacement: a randomized pilot study. Eur J Transl Myol. 2021 Aug 26. doi: 10.4081/ejtm.2021.9932. Epub ahead of print.
13. Przkora R, Sibille K, Victor S, Meroney M, Leeuwenburgh C, Gardner A, Vasilopoulos T, Parvataneni HK. Assessing the feasibility of using the short physical performance battery to measure function in the immediate postoperative period after total knee replacement. Eur J Transl Myol. 2021 Apr 7;31(2):9673. doi: 10.4081/ejtm.2021.9673. PMID: 33840178; PMCID: PMC8274223.

Books and Book Chapters:

1. "Molecular and Cellular Biology of Aging". Calorie Restriction, Xu, Kapahi, Leeuwenburgh. Jones & Bartlett Learning, 2013
2. Redox Signaling and Regulation in Biology and Medicine (2009) Free Radicals in Mammalian Aging. Editor, Claus Jacob. Publisher, Wiley-VCH Verlag, p473-519.
3. Free Radicals in Biology and Medicine (2008). Editors Carlos Gutierrez Merino and Christiaan Leeuwenburgh, Publisher Research Signpost, ISBN 978-81-308-0267-1, 263 pages
4. Dirks and Leeuwenburgh. Pharmacotherapy of Cachexia"; Apoptosis in skeletal muscle cachexia and aging. 2005, p49-69.
5. Pollack, M and C. Leeuwenburgh. Molecular Mechanisms of Oxidative Stress and Aging: Free radicals, aging, antioxidants, and disease. Handbook of Oxidants and Antioxidants in Exercise. p 881-926, C.K. Sen, L. Packer and O. Hanninen, editors. Chapter 30: Elsevier Science, 1999.
6. Ji, L. L. and C. Leeuwenburgh. Glutathione and Exercise. In Pharmacology in Exercise and Sports. p 97-124, (Ed. S. Somani) CRC Press, Boca Raton. Florida, 1996.
7. Leeuwenburgh C., and L. L. Ji. The role of glutathione in preventing oxidative stress during exercise and training. In: Skeletal Muscle Research, pp 69-84. (eds. C.K. Sen & and M. Ataley). University Kuopio Proceedings, 1994.

PRESENTATIONS AT PROFESSIONAL CONFERENCES

A. International

2022 (March) Padua Days on Muscle and Mobility Medicine, Euganean Hills, Padova, Italy. (Co-Organizers, Invited Speaker, and Session Chair)
2022 (July) Costa Rican Association of Dietitians and Nutritionists; Nestle, San Jose, Costa Rica. (Invited).
2022 (July) Association of Nutritionists and Dietitians of Guatemala; Nestle, Guatemala City, Guatemala. (Invited)
2021 (November) APPTO Annual Congress: Panama Association for the Prevention and Treatment of Obesity, Panama City. (Invited)
2021 (June) Padua Days on Muscle and Mobility Medicine, Euganean Hills, Padova, Italy. (Co-Organizers, Invited speaker, and session Chair; Virtual)
2020–2019 (COVID limited travel in 2020)
2018 (March) Translational Myology in Health and Disease Monte Grotto, Padova, Italy. (Co-Organizers, Invited speaker, and Session Chair)
2017 (March) Target Audience Scientist, 2nd Interventions in Aging Conference, Cancun, Mexico. (Invited)
2017 (March) Translational Myology in Aging and Neuromuscular Disorders Monte Grotto, Italy. (Co-Organizers, Invited Speaker, and Session Chair)
2017 (Oct) European Molecular Biology Organization/EMBO|FEBS Lecture Course on Mitochondria in Life, Death, and Disease, Brindisi, Italy. (Invited)
2016 (April) Muscle Decline in Aging and Neuromuscular Disorders: Mechanisms and Countermeasures, Padova, Italy. (Co-Organizers, Invited Speaker, and Session Chair).
2015 (Sept) Erasmus University, Research Seminar. Rotterdam, Netherlands. (Invited)
2015 (March) Translational Myology in Aging and Neuromuscular Disorders Terme Euganee, Padova, Italy. (Co-Organizers, Invited Speaker, and Session Chair)
2014 (Oct) International Society of Geriatric Oncology, SIOG Annual Conference, Lisbon, Portugal. (Invited)
2014 (March) International Conference on Frailty and Sarcopenia Research (ICFSR). The International Association of Gerontology and Geriatrics—Global Aging Research Network (IAGG-GARN). Barcelona, Spain. (invited)
2014 (March) 83rd Nestlé Nutrition Institute Workshop, "Frailty: Pathophysiology, Phenotype and Patient Care," Barcelona, Spain. (Invited)

2013 (Nov) 2nd World Congress on Controversies, Debates and Consensus in Bone, Muscle, and Joint Diseases (BMJD), Brussels, Belgium. (Invited)

2013 (Sept) Society for Free Radical Research—Europe (SFRR-E) Conference, “The New Era of –omics in Free Radicals in Biology and Medicine,” Athens, Greece. (Invited)

2013 (Sept) 7th International Conference, “Tear Film and Ocular Surface: Basic Science and Clinical Relevance,” Taormina, Sicily, Italy. (Invited)

2013 (June) 5th International Symposium: “Nutrition, Oxygen Biology, and Medicine; Development and Aging; Nutrition Epigenetics; and Lifestyle and Health Span,” Paris, France. (Invited)

2012 (Nov) Milan, REGENERA Society, Dissemination on Predictive Medicine and Prevention Regenerative and Healthy-Aging, Milan, Italy. (Invited)

2012 (Oct) German Federation of Sports Medicine (DGSP) 100 Years of German Sports Medicine, Berlin, Germany. (Invited)

2012 (July) 5th Tokyo Anti-Aging Academy, Tokyo, Japan. (Invited)

2012 (July) Keio University, Tokyo, Japan. (Invited)

2012 (July) 12th Japanese Anti-Aging Medicine Conference, Tokyo, Japan. (Invited)

2012 (July) Osato Research Institute¸ Gifu, Japan. (Invited)

2011 (Sept) Catholic University of the Sacred Heart, Rome, Italy. (Invited)

2011 (Sept) **8th International Conference of** Mitochondrial Physiology and Pathology, Bordeaux, France. (Invited)

2011 (July) The 21st International Conference of Korean Society for Gerontology: Interventions of Aging and Age-Related Diseases, Busan, South Korea. (Invited)

2010 (June) The 1st International Congress on Controversies in Longevity, Health and Aging (CoLONGY), Barcelona, Spain, June 24-27, 2010.

2010 (March) First International Congress on Translational Research in Human Nutrition, Clermont-Ferrand (France) on March 19-20, 2010. ‘Protein-energy metabolism in aging and chronic diseases: Role of nutrition and physical activity.

2009 (Dec) Italian Society of Gerontology and Geriatrics (SIGG) National Congress, 2-5 December 2009 (Oct) Congress "Genes, Drugs and Gender" organized by the Foundation Menarini, Sassari, Italy.

2009 (June) FEDERA conference, Leiden, Netherlands (Invited Speaker; Seminar and Public Lecture)

2009 (June) Mini-Symposium Exercise Therapy in Cancer Patients, Erasmus Medical Center Rotterdam, ‘Muscle weakness in Cancer patients, fact or fiction?’

2009 (Apr) Nutrition, Oxygen Biology and Medicine symposium, Paris, France (Invited Speaker)

2008 (Dec) Bispebjerg Symposium on Sports Medicine, Skeletal Muscle Atrophy, Copenhagen, Denmark (Invited Speaker)

2008 (Nov) Italian Society of Gerontology and Geriatrics (SIGG), Florence,

2008 (Nov) The International Society of Chinese Scholars for Exercise Physiology, Tianjin, China (Invited Speaker)

2008 (Nov) Pusan University, College of Pharmacy, Invited Talk, Pusan, S. Korea

2008 (June) 6th Northern Light Summer Conference, Canadian Federation of Biological Societies 52nd Scientific conference, Winnipeg, Manitoba, Canada (Invited Speaker)

2006 (Oct) Aging and Exercise in the 13th International Conference of Biochemistry of Exercise, Korean Society of Exercise Biochemistry and Exercise Physiology, "Effects of exercise on ageing muscle and other tissue functions and metabolism", Seoul, S. Korea (Invited Speaker)

2006 (Oct) The International Society of Chinese Scholars for Exercise Physiology, Tianjin, China (Invited Speaker)

2006 (July) European Sports Congress 2006 in Lausanne, Switzerland, “Exercise and Oxidative Stress” (invited Speaker)

2006 DANONE ageing workshop, Paris, 4–5 May, 2006 (Invited Speaker and Consultant)

2005 (Dec) Mitochondria: from Molecular Insight to Physiology and Pathology. University

of Bari, Bari, Italy, (Invited speaker)
2004 (July) 14th Qualitative and Quantitative Perspectives of Longevity, Kyungjoo, South Korea, Invited Speaker
2004 (July) European Cell Death Organization; Death on the Sea, Crete, Greece (Poster)
2004 (Sept) Gordon Conference, Biology of Aging, Aussois, France (Invited Speaker)
2004 (May) XII Meeting of the International Society for Free Radical Research, Buenos Aires, Argentina (Invited Speaker)
2003 (Nov) Free Radicals and Aging, McMaster University, Hamilton, Canada, (Invited Speaker)
2003 (Nov) Invited External Reviewer for PhD dissertation defense, Gianni Parise, McMaster University, Hamilton, Canada
2003 (Sept) Queens' College, Cambridge University, England, Association of Biomedical Gerontology 10th Congress (Invited Lecture)
2003 (Oct) Symposium of the German Society for Sports Medicine, Potsdam, Germany (Invited Lecture)
2003 (June) Oxidants and Antioxidants in Biology, Cadiz, Spain, (Invited Lecture)
2002 (July) 4th International Congress of Pathophysiology, Budapest, Hungary, (Invited Lecture)
2002 (Sept) 9th Biennial Meeting of the Society for Free Radical Research International, Paris, France
2002 (June) Erasmus University, Rotterdam, Netherlands (Invited Lecture)
2002 (May) University of Catania, Department of Pharmacology, Sicily, Italy (Invited Lectures)
2001 (June) University of Bologna, Department of Biochemistry, Italy (Invited Lecture)
2001 (June) University of Bari, Department of Biochemistry, Italy (Invited Seminar)
2001 (Oct) International Association of Biomedical Gerontology (9th), Vancouver, Canada (Invited Lecture-1)
2001 (Oct) International Association of Biomedical Gerontology (9th), Vancouver, Canada (Invited Lecture-2)
2001 (May) 2nd International Conference on Oxidative Stress and Aging, Maui, Hawaii, USA (Poster)
2000 (June) Universidad Complutense, Department of Biology, Madrid, Spain, (Invited Seminar)
1998 (Sept) International Society for Free Radical Research Sao Paulo, Brazil, 1998 (Poster)
1998 (Jan) The University of Stellenbosch Medical School, Stellenbosch, South Africa,
(Workshop and Invited External Reviewer for Medical Students)

B. National

2021 (April) American Heart Association, Strategically Focused Research Networks, Chicago, IL. (Chair of Session and Poster)
2020 (April) American Heart Association, Strategically Focused Research Networks, Vanderbilt, TN. (Network Presentation)
2019 (December) NIA, Rodent Care and Use for Aging Research, Baltimore, MD. (Invited)
2019 (February) International Conference on Frailty & Sarcopenia Research. Miami, FL. (Poster)
2019 (July) Florida Geriatric Society, Orlando, FL. (Invited)
2019 (April) American Heart Association, Strategically Focused Research Networks, Lexington, KY. (Network Presentation)
2018 (March) International Conference on Frailty and Sarcopenia Research. Miami, FL. (Poster)
2018 (April) Dr. G. Lombard Kelly Lecturer, Medical College of Georgia, Augusta University, August, GA. (Invited Award Lecture)

2017 (April) RISE Program. School of Medicine, University of Porto Rico, PR. (Invited)
2017 (June) American Aging Association National Meeting, New York, NY. (Poster)
2016 (June) American Aging Association National Meeting, Seattle, WA. (Scientific Board Meeting and Invited Speaker)
2016 (October) Medical University of South Carolina, MUSC, Research Seminar, Charleston, SC.
2016 Nemours Children' Health Jacksonville, FL. (Invited)
2015 (April) Dept. Environmental & Occupational Health, Robert Stempel College of Public Health and Social Work, Florida International University, Miami, FL. (Invited)
2015 (July) Florida Academy of Nutrition and Dietetics annual symposium, Orlando, FL. (Invited)
2014 (May) Annual American College of Sports Medicine, Orlando, FL. (Poster)
2014 (July) IANA (International Academy on Nutrition and Aging) Albuquerque, NM. (Invited)
2014 (May) ARVO Annual Meeting, Leading Eye and Vision Research, Orlando, FL. (Invited)
2014 (September) ACSM's Integrative Physiology of Exercise conference, Miami, FL (Invited)
2013 (May) Robert M. Berne Cardiovascular Research Center at the University of Virginia, Cardiovascular Seminar Series, Charlottesville, VA. (Invited)
2013 (May) American Aging Association (AGE) 2013 Meeting "Aging: Prevention, Reversal, Slowing," Baltimore, MD. (Invited)
2012 (May) 41st Annual Meeting of the American Aging Association, Fort Worth, TX. (Invited)
2012 (March) Life Ancillary Study Symposium, Biomarkers Symposium, Washington, DC. (Invited)
2012 (February) Department of Physiology, The Brody School of Medicine at East Carolina University (ECU), Greenville, NC. (Invited)
2011 (October) Johns Hopkins University Seminars on Aging Series, Baltimore, MD. (Invited)
2011(November) The Gerontological Society of America's 64th Annual Scientific Meeting, Boston, MA. (Invited)
2011 (October) University of Southern California (USC) School of Pharmacy and the American Association of Pharmaceutical Scientists (AAPS) Symposium, "Moving Targets," Los Angeles, CA. (Invited)
2011 (November) Gordon Research Conference on Bioenergetics, Andover, NH. (Invited)
2011 (April) Washington University School of Medicine, Department of Obstetrics and Gynecology, St. Louis, MO. (Invited)
2010 Tulane University, Center for Aging, New Orleans
2010 Department of Pharmacology and Neuroscience, UNT HSC, Fort Worth
2009 Aging Muscle Symposium, San Francisco, CA (Invited Speaker)
2009 American Aging Association Conference, Scottsdale, AZ (Invited Speaker)
2009 ACSM Annual Conference, Seattle, WA (Invited Speaker)
2008 Understanding Aging Conference, Los Angeles, CA (Invited Speaker)
2008 10th Longevity Consortium Symposium, Boulder, CO (Invited Seminar)
2008 Linus Pauling Institute, Oregon, Oregon State University, Corvallis (Invited Speaker)
2007 Cachexia Conference, Tampa FL Presentation title: Mitochondria and Muscle (Invited Speaker)
2007 Intl. College of Geriatric Psychopharmacology, San Diego, CA (Invited Speaker)
2007 GSA National Meeting, San Francisco, CA (Invited Speaker)
2007 ACSM, New Orleans, LA (Invited Speaker)
2007 WORKSHOP NIA unexplained fatigue in the elderly, Bethesda, MO
2007 Nathan Shock Center Conference, Mayan Ranch, San Antonio, TX (Invited Speaker)
2007 Cachexia Conference, Tampa FL (Invited Speaker)
2007 University of Colorado (Invited Seminar)

2007 Longevity Consortium, Santa Fe, New Mexico (Invited Speaker)
2006 6th Annual S. Mouchly Small Muscle Symposium, Amherst, MA (Invited Speaker)
2006 American Aging Association, Boston, Massachusetts (Invited Speaker)
2005 USC, Los Angeles, Distinguished Professor Lecture
2005 Free Radical Biology and Medicine, (Invited Speaker) (USA)
2005 Workshop NIA, Calorie Restriction (Invited Lecture), Baltimore, USA
2004 Gerontological Society of America (Invited Lecture)
2004 Baltimore; Nathan Shock Center Award Lecture at NIA; USA
2004 The Calorie Restriction Society, Charleston, SC (Invited Speaker)
2004 Gerontological Society of America, Washington DC, USA, (Invited Speaker)
2004 American Aging Association (AGE), Public Lecture, St. Petersburg, FL (Invited Speaker)
2004 Organizer and Lecturer, Pre-Symposium American Aging Association (AGE), St. Petersburg, FL
2004 University of Texas at San Antonio, TX (Invited Seminar)
2004 University of Colorado, Boulder, CO (Invited Lecture)
2003 Texas A & M, College Station, TX, (Invited Lecture)
2003 Grand Rounds, Why do we age? Vermont Medical School, Burlington, VT, (Invited Lecture)
2003 Vermont Medical School, Burlington, Vermont, (Invited Seminar Lecture)
2003 The Gerontological Research Center and San Antonio Nathan Shock Aging Center, San Antonio, TX
2003 Gerontological Society of America, San Diego, CA, (Invited Lecture)
2003 Diet and Optimum Health, Linus Pauling Institute, Oxygen Club California Portland (Invited Lecture)
2003 American College of Sports Medicine, San Francisco, (Mini-Symposium)
2002 Kronos, Sarcopenia and Aging, San Diego (Invited lecture)
2002 American College of Sports Medicine, Indianapolis (Mini-Symposium)
2001 American College of Sports Medicine, Baltimore, (Invited Lecture)
2000 Oxygen Society, San Diego, CA
2000 Society of Geriatric Cardiology, Anaheim, CA, (Invited Lecture)
1999 American College of Sports Medicine, Seattle, (Invited Lecture)
1998 American College of Sports Medicine, Orlando, (Poster)
1997 American Aging Association, Philadelphia, (Invited Lecture)
1997 Oxygen Society, San Francisco, (Selected Lecture Presentation)
1996 Oxygen Society, Miami, (Selected Lecture Presentation and Poster)
1996 American Heart Association 69th Scientific Session, New Orleans, (Selected Lecture Presentation 1)
1996 American Heart Association 69th Scientific Session, New Orleans, (Selected Lecture Presentation 2)
1996 Federation of the American Society for Experimental Biological, Washington D.C., (Selected Lecture Presentation)
1995 Federation of the American Society for Experimental Biological, Atlanta (Poster)

Local/Regional:

2010 UF Running Medicine Conference, UF Orthopedic and Sports Medicine Institute
2009 Whitney Laboratory for Marine Bioscience, Marineland, FL (Invited Speaker)
2009 University of Florida, Gainesville, FL, Animal Sciences, Invited Seminar speaker.
2006 From Frail to Fit After Fifty, Dept. of Veterans Affairs, GRECC St. Petersburg, Florida (Invited Lecture)
2006 University of Florida, Gainesville, FL, Alumni Association Grand Guard Reunion presentation
2006 University of Florida, Gainesville, FL, “IDH3931 Science for Life Seminar Series – Fall 2006 Schedule Howard Hughes MI Science For Life seminar course
2006 University of Florida, Gainesville, FL, “Center for Neurobiology of Aging” seminar.
2006 University of Florida, Gainesville, FL, “Medical Residents; house staff noon conference lecture
2006 University of Florida, Gainesville, FL, “Biology of Aging” IDP course, guest lecture.
2006 University of Florida, Gainesville, FL, Guest lecture, Dietician Association

2005 University of Florida, Gainesville, FL, Institute on Aging
2005 University of Florida, Gainesville, FL, IDP Graduate Program Seminar Series
2005 University of Florida, Gainesville, FL, College of Public Health and Health Professions
2004 Tallahassee, Florida State University, Dept. of Nutrition, Food and Exercise Science and Program in Neuroscience, USA.
2004 University of Florida, Gainesville, FL, College of Medicine, Hypertension Center
2004 University of Florida, Gainesville, FL, College of Nursing
2004 University of Florida, Gainesville, FL, Center for Gerontological Studies
2003 University of Florida, Gainesville, FL, College of Health Professions
2003 University of Florida, Gainesville, FL, College of Nursing
2003 University of Florida, Gainesville, FL, College of Veterinary Medicine
2003 University of Florida, Gainesville, FL, Department of Food Science and Human Nutrition
2003 University of Florida, Gainesville, FL, Grant Writing Workshop
2003 University of Florida, Gainesville, FL, Free Radical Biology Meeting
2003 University of Florida, Gainesville, FL, Anesthesiology Residents
2003 University of Florida, Gainesville, FL, Gerontology Students
2002 Washington University, JOH Meeting, Dept. of Internal Medicine, St. Louis, MO, (Invited Seminar)
2002 University of Florida, Gainesville, FL, Alumni Association (Graduation Series)
2002 University of Florida, Gainesville, FL, Alumni Association (Back to School)
2002 University of Florida, Gainesville, FL, Institute on Aging
2001 University of Florida, Gainesville, FL, Center for Exercise Science
2001 University of Florida, Gainesville, FL, Center for Gerontology and Institute on Aging
2001 University of Florida, Gainesville, FL, Free Radical Meeting (Invited Lecture 1)
2001 University of Florida, Gainesville, FL, Free Radical Meeting (Invited Lecture 2)
2001 University of Florida, Gainesville, FL, VA Medical School
2001 University of Florida, Gainesville, FL, Veterinarian Medicine
2001 University of Florida, Gainesville, FL, Department of Nutrition
2000 Cardiopulmonary Rehabilitation Symposium: Status 2000. Orlando, FL, (Invited Lecture)
1999 Southeastern ACSM Regional Conference Meeting, Norfolk, VI. (Invited Lecture)
1999 Cardiopulmonary Rehabilitation Symposium: Status”99”, Orlando, FL, (Invited Lecture)
2000 University of Florida, Gainesville, FL, Geriatric Research Educational Clinical Center
1999 University of Florida, Gainesville, FL, Free Radical Meeting (Invited Lecture)
1998 University of Florida, Gainesville, FL, Department of Pharmaceutics and Pharmacodynamics
1996 Washington University School of Medicine, St. Louis, MO, Mass Spectrometer Resource Center
1995 Washington University, Department of Internal Medicine, St. Louis, MO, (Invited Seminar)

CHAPTER 13. Inspirers & Supporters

13.10. Marco Narici

Marco Narici 2022

CURRICULUM of Marco Narici

Professional Address
University of Padova
Department of Biomedical Sciences
Institute of Physiology
Via Marzolo, 3
35131 Padova
Italy

Nanionality Italian

Position Title
Full Professor of Physiology
E-mail: marco.narici@unipd.it

Education/Training

Institution	DEGREE	YEAR	FIELD OF STUDY
Bedford College, University of London,	BSc	1982	Physiology
King's College, University of London,	MSc	1984	Human Physiology
University of Pavia, Italy	BSc	1991	Biology
University of Pavia, Italy	PhD	1995	Physiology

Professional and Honours
1983-1999: Research Fellow at the National Research Centre, Milan, Italy
1994-1996: Maître d'Enseignement et de Richerche, Centre Medical Universitaire, University of Geneva, Switzerland.
1993-1997: Project manager of NASA experiment "The effects of Microgravity on Skeletal Muscle Contractile Properties", Spacelab mission STS-78.
1998-2000: Chairman European Space Agency (ESA) Skeletal Muscle Topical Team

1999: Full Professor in Physiology of Ageing, Institute for Biophysical and Clinical Research into Human Movement, Manchester Metropolitan University
2005: Member of ESA "Artificial Gravity Topical Team"
2007-2012: Member of ESA Life Sciences Peer Review Board
2008: Member of European Science Foundation microgravity review panel
2011-2012: Director of the Institute for Biomedical Research into Human Movement and Health (IRM), Manchester Metropolitan University
2012-2017: Professor and Head of Division of Clinical Physiology, School of Graduate Entry to Medicine and Health, University of Nottingham
2013- 2015: President of the European College of Sport Science (ECSS)
2017 to date Professor of Physiology, appointed through the 'Brain Gain Project' of the Italian Ministry of Education, University and Research (MIUR), University of Padua, Italy
2018 to date: Director of Myology Centre MyoCIR of the University of Padova
2020 to date: Coordinator of Italian Space Agency Integrated Physiology Board for the definition of the National Space Research Roadmap

Research summary and interests:
Marco Narici has published 265 peer reviewed journal articles (Scopus H-index 70) and book chapters. His present work and interests are focused on the mechanisms of remodeling of human neuromuscular system with exercise, inactivity (including spaceflight) and ageing. He is presently coordinating the NeuAge PRIN Project funded by the Italian Ministry of Education, University and Research (MIUR) focusing on the Mechanisms of Neuromuscular Ageing and its Functional Implications, and is coordinator of Italian Space Agency (ASI) project, MARS-PRE, focusing on the identification of early biomarkers of pathophysiological alterations of different organs and systems to simulated microgravity.

SELECTED PUBLICATIONS
From 265 ISI publications and 9 book chapters, H-index: 70, 16992 citations, Scopus

1. Sarto F, Stashuk DW, Franchi MV, Monti E, Zampieri S, Valli G, Sirago G, Candia J, Hartnell LM, Paganini M, McPhee JS, De Vito G, Ferrucci L, Reggiani C, Narici MV. Effects of short-term unloading and active recovery on human motor unit properties, neuromuscular junction transmission and transcriptomic profile. J Physiol. 2022 Nov;600:4731-4751
2. Sarto F, Pizzichemi M, Chiossi F, Bisiacchi PS, Franchi MV, Narici MV, Monti E, Paoli A, Marcolin G. Physical active lifestyle promotes static and dynamic balance performance in young and older adults. Front Physiol. 2022 Aug 17;13:986881.
3. Pratt J, De Vito G, Segurado R, Pessanha L, Dolan J, Narici M, Boreham C. Plasma neurofilament light levels associate with muscle mass and strength in middle-aged and older adults: findings from GenoFit. J Cachexia Sarcopenia Muscle. 2022;13:1811-1820.
4. Zuccarelli L, Baldassarre G, Magnesa B, Degano C, Comelli M, Gasparini M, Manferdelli G, Marzorati M, Mavelli I, Pilotto A, Porcelli S, Rasica L, Šimunič B, Pišot R, Narici M, Grassi B. Peripheral impairments of oxidative metabolism after a 10-day bed rest are upstream of mitochondrial respiration. J Physiol. 2021 Nov;599:4813-4829.
5. Monti E, Waldvogel J, Ritzmann R, Freyler K, Albracht K, Helm M, De Cesare N, Pavan P, Reggiani C, Gollhofer A, Narici MV. Muscle in Variable Gravity: "I Do Not Know Where I Am, But I Know What to Do". Front Physiol. 2021 Aug 4;12:714655.
6. Manganotti P, Buoite Stella A, Ajcevic M, di Girolamo FG, Biolo G, Franchi MV, Monti E, Sirago G, Marusic U, Simunic B, Narici MV, Pisot R. Peripheral nerve adaptations to 10 days of horizontal bed rest in healthy young adult males. Am

J Physiol Regul Integr Comp Physiol. 2021 Sep 1;321(3):R495-R503.
7. Narici M, McPhee J, Conte M, Franchi MV, Mitchell K, Tagliaferri S, Monti E, Marcolin G, Atherton PJ, Smith K, Phillips B, Lund J, Franceschi C, Maggio M, Butler-Browne GS. Age-related alterations in muscle architecture are a signature of sarcopenia: the ultrasound sarcopenia index. J Cachexia Sarcopenia Muscle. 2021 Aug;12(4):973-982.
8. Pratt J, De Vito G, Narici M, Segurado R, Pessanha L, Dolan J, Conroy J, Boreham C. Plasma C-Terminal Agrin Fragment as an Early Biomarker for Sarcopenia: Results From the GenoFit Study. J Gerontol A Biol Sci Med Sci. 2021 Nov 15;76(12):2090-2096.
9. Monti E, Reggiani C, Franchi MV, Toniolo L, Sandri M, Armani A, Zampieri S, Giacomello E, Sarto F, Sirago G, Murgia M, Nogara L, Marcucci L, Ciciliot S, Šimunic B, Pišot R, Narici MV. Neuromuscular junction instability and altered intracellular calcium handling as early determinants of force loss during unloading in humans. J Physiol. 2021 Jun;599(12):3037-3061.
10. Marusic U, Narici M, Simunic B, Pisot R, Ritzmann R. Nonuniform loss of muscle strength and atrophy during bed rest: a systematic review. J Appl Physiol (1985). 2021 Jul 1;131(1):194-206.
11. Monti E, Toniolo L, Marcucci L, Bondì M, Martellato I, Šimunič B, Toninello P, Franchi MV, Narici MV, Reggiani C. Are muscle fibres of body builders intrinsically weaker? A comparison with single fibres of aged-matched controls. Acta Physiol (Oxf). 2021 Feb;231(2):e13557.
12. Capri M, Morsiani C, Santoro A, Moriggi M, Conte M, Martucci M, Bellavista E, Fabbri C, Giampieri E, Albracht K, Flück M, Ruoss S, Brocca L, Canepari M, Longa E, Di Giulio I, Bottinelli R, Cerretelli P, Salvioli S, Gelfi C, Franceschi C, Narici M, Rittweger J. Recovery from 6-month spaceflight at the International Space Station: muscle-related stress into a proinflammatory setting. FASEB J. 2019 Apr;33(4):5168-5180.
13. Rittweger J, Albracht K, Flück M, Ruoss S, Brocca L, Longa E, Moriggi M, Seynnes O, Di Giulio I, Tenori L, Vignoli A, Capri M, Gelfi C, Luchinat C,Francheschi C, Bottinelli R, Cerretelli P, Narici M. Sarcolab pilot study into skeletal muscle's adaptation to long-term spaceflight. NPJ Microgravity. 2018 Sep 17;4:18.
14. Floreani M, Rejc E, Taboga P, Ganzini A, Pišot R, Šimunič B, Biolo G, Reggiani C, Passaro A, Narici M, Rittweger J, di Prampero PE, Lazzer S. Effects of 14 days of bed rest and following physical training on metabolic cost, mechanical work, and efficiency during walking in older and young healthy males. PLoS One. 2018 Mar 12;13(3):e0194291.
15. Rejc E, Floreani M, Taboga P, Botter A, Toniolo L, Cancellara L, Narici M, Šimunič B, Pišot R, Biolo G, Passaro A, Rittweger J, Reggiani C, Lazzer S. Loss of maximal explosive power of lower limbs after 2 weeks of disuse and incomplete recovery after retraining in older adults. J Physiol. 2018 Feb 15;596(4):647-665.
16. Quinlan JI, Maganaris CN, Franchi MV, Smith K, Atherton PJ, Szewczyk NJ, Greenhaff PL, Phillips BE, Blackwell JI, Boereboom C, Williams JP, Lund J, Narici MV. Muscle and Tendon Contributions to Reduced Rate of Torque Development in Healthy Older Males. J Gerontol A Biol Sci Med Sci. 2018 Mar 14;73(4):539-545.
17. Sarcopenia, dynapenia, and the impact of advancing age on human skeletal muscle size and strength; a quantitative review.
18. Mitchell WK, Williams J, Atherton P, Larvin M, Lund J, Narici M. Front Physiol. 2012 Jul 11;3:260. doi: 10.3389/fphys.2012.00260. eCollection 2012. PMID: 22934016 Free PMC article.

CHAPTER 13. Inspirers & Supporters

13.10. Gabriele Siciliano

Gabriele Siciliano 2022

Gabriele Siciliano was a postgraduate in Neurology at the University of Pisa and then a PhD student when he moved to Padua in the 1980s under the guidance of Corrado Angelini of the Neurological Clinic of the University of Padua, with a UILDM scholarship for the study of muscle pathologies. In that period he met several great representatives of the great Paduan muscle tradition, from Prof. Massimiliano Aloisi to GianAntonio Danieli, Marisa Mostacciuolo, Daniela Pozzobon, Stefano Schiaffino and, among others, myself. For his interest in muscle fatigue in neuromuscular diseases, after spending a research period at the University of Liverpool in the group of Richard HT Edwards, in the 90s participated and contributed to several "Padua Muscle Days", publishing and serving as reviewer of the journal BAM since 1990 and then EJTM since 2010. While we have no co-authorship in our publication lists, we have collaborated and exchanged ideas on new avenues in muscle science, as was the case with surgical transposition of the adductor magnum muscle to treat anal striatum sphincter insufficiency. Already a founding member in 2000 of AIM (Italian Myological Association), Gabriele was appointed President of that scientific association for a three-year period from 2015 to 2018, during which time BAM published the annual contributions of the AIM Congress. Gabriele is now Full Professor of Neurology at the University of Pisa and Director of the Neurological Clinics of Pisa. I hope he will accept invitation to serve as new 2023 Editor of the EJTM Section: Myology Reviews.

In any case, I am very grateful that Gabriele has accepted to include the following Curriculum Vitae in this book among my Inspirers and Supporters.

Curriculum

PERSONAL INFORMATION

Gabriele Siciliano
University of Pisa
Department of Clinical and Experimental Medicine
Via Savi, 10 - 56126 Pisa, Italy
+39 050993604 +39 3387221010
Email: g.siciliano@med.unipi.it
https://unimap.unipi.it/cercapersone/dettaglio.php?ri=258

Sex Male | Date of birth 08/08/1955 | Nationality Italian

Enterprise	University	EPR
☐ Management Level	☒ Full professor	☐ Research Director and 1st level Technologist / First Researcher and 2nd level Technologist
☐ Mid-Management Level	☐ Associate Professor	☐ Level III Researcher and Technologist
☐ Employee / worker level	☐ Researcher and Technologist of IV, V, VI and VII level / Technical collaborator	☐ Researcher and Technologist of IV, V, VI and VII level / Technical collaborator

WORK EXPERIENCE

2016-present Full Professor, SSD MED/26, Neurology, Dep. Clinical and Experimental Medicine, University of Pisa
2001-2016 Associate Professor, SSD MED/26, Neurology, University of Pisa
1997 - 2001 Researcher, Department of Neuroscience, University of Pisa

1993 - 1997 Neurologist Assistant Register, SSD MED/26, Neurology, University of Pisa
1991 - 1993 Post doc fellow, University of Ancona

EDUCATION AND TRAINING

1988-1992 Specialization in Physical and Rehabilitation Medicine, University of Pisa
1986-1989 PhD in Neurological And Neurosensorial Sciences, University of Pisa
1980 – 1984 Specialization in Neurology, University of Pisa
1974-1980 Degree in Medicine and surgery, University of Pisa

WORK ACTIVITIES

Award and positions

2021 and 1998: Honor for recognition of dedicated professional activity, Associazione Italiana CIDP
Since 2020: Chair of Neurology, SSD MED/26, Department of Clinical and Experimental Medicine, University of Pisa
Since 2018: Director of the Residency Program in Neurology, University of Pisa, Medical

	School
Since 2018:	Chair of Muscle and NMJ disorders Scientific Panel at European Academy of Neurology
Since 2016:	Chairman of the National Reference Centre for European Rare Disease Network Euro-NMD, Azienda Ospedaliero Universitaria Pisana
Since 1993:	Responsible for the Unit of Neuromuscular Diseases and for the Laboratory of Molecular Diagnosis, Department of Clinical and Experimental Medicine, Azienda Ospedaliero Universitaria Pisana
1998:	Honor for recognition of dedicated professional actvity - Unione Italiana Lotta alla Distrofia Muscolare.
1991:	"Antonio Arrigo Price"- Award for the best PhD thesis from the Italian Society of Clinical Neurophysiology
Since 1984:	Member, Italian Society of Neurology (Executive Board 2007-2011 and 2019)
Since 1989:	Member, Italian Association of Neuropathology (Executive Board 2001-2004 and 2012-2015)
since 1989:	Member, Italian Society of Clinical Neurophysiology
since 1990:	Member, International Society of Neuropathology
since 1990:	Member, World Society of Neurology
1991-2000:	Member, Royal Society of Medicine, Section of Neurology
since 1996:	Member, World Society of Myology
since 2005:	Member, Mediterranean Society of Myology, (Executive Board since 2010)
since 1998-2007	Member, American Association for the Advancement of Science
since 2000	Italian Association of Myology (Executive Board 2009-2015; Chairman 2015-2018);
since 2001	Motorneuron Study Group of Italian Society of Neurology (Coordinator 2003-2005)
since 2001	Member, European Amyotrophic Lateral Sclerosis Consortium
since 2016	Member, European Federation of Neurological Societies, since 2012 and European Academy of Neurology (Chair of Muscle Panel since the current year)

Publications

515 total publications, 243 publications in peer-reviewed journals in the last 10 year
Total Impact Factor (IF): 2076,3 (average IF/paper): 4,8; total number of citations: 14164; H index: 57
Impact Factor (IF) in the last 10 year: 1191,5 (average IF/paper) in the last 10 year: 5,11

Editorial activity

EDITORIAL BOARD OF INTERNATIONAL JOURNALS IN NEUROLOGIC AREA
Since 2019- Frontiers Neurology
Since 2017: Acta Myologica, Pacini Ed, ISSN 1124-8874
Since 2017: Neurological Sciences, Springer-Verlag Italia, ISSN:1590-1874 (Print)
Since 2015: Basic and Applied Myology, Unipress, ISSN: 1120-9992
Since 2011: Journal of Alzheimer's Disease (JAD), IOS Press, ISSN:1387-2877
Since 2010: Nutrition, Tarrytown, NY : Elsevier Science, 0899-9007 (Print)

Since 2006: Bioscience Reports, Kluwer Academic/Plenum Publishers, ISSN 0144-8463 (Print)
EDITORIAL BOARD FOR SPECIAL ISSUES OF INTERNAL JOURNALS IN NEUROLOGIC AREA
- Neurological Sciences Supplement- Perspectives In Molecular Therapy On Muscle Diseases, Springer-Verlag Italia, ISSN:1590-1874 (Print)
- Bioscience Reports SUPPLEMENT- Mitochondrial Diseases: Advances In Understanding and Treating Pathologies, Kluwer Academic/Plenum Publishers, ISSN 0144-8463 (Print).
- CNS Neurol Disord Drug Targets SUPPLEMENT- Neuroprotection In ALS: From Pathology To Treatment, Bentham Science Publishers, 1871-5273 (Print)
- Neuromuscular Disorders SUPPLEMENT- Muscle Fatigue in Neuromuscular Disorders: Pathogenic Mechanisms and Treatment, Pergamon Press, ISSN:0960-8966 (Print)

Invited speaker, seminars, lectures and/or chairmanships
in International/National Congresses: 555.

More recent
Telethon 2021: North Star Assessment (NSAD) and longitudinal outcome measures for pediatric and adult Limb Girdle Muscular Dystrophies - PI of Partner Research Unit
EJPRD 2020: Safety and efficacy of a possible epigenetic therapy for FSHD muscular dystrophy (EpiThe4FSHD)- PI of Partner Research Unit
Progetto PRA Università di Pisa 2020 "Improving daily leaving for severely disabled people through enhanced automatic speech recognition technology (DESIRE) - PI of Partner Research Unit
2020- Biogen Pharm- Study of muscle fatigue in adult patients with spinal muscular atrophy- Coordinating PI
Parent Project 2019: Phenotypic variability in Becker Muscular Dystrophy: proposal of a Clinical and Molecular characterization protocol to stratify patients towards trial readiness- Coordinating PI
Bando Ministeriale Ricerca Salute 2018: Development and application of an integrated, multiparametric system for early genetic diagnosis and personalized treatments in children and adults with neuromuscular diseases (InGene 2.0)- PI of Partner Research Unit
AIFA-TRS-2018 - 00001525- "Deflazcort TREATment in LMNA related congenital muscular dystrophy: study of clinical effectlveness and search for reliable biomarkers"- Coordinating PI
2017- Aliveda Farmaceutici- Oxidative stress and therapeutic antioxidant therapy in Amyotrophic Lateral Sclerosis- Coordinating PI
2016- AmareOltre ONLUS- Exercise training as therapeutic strategy in Amyotrophic Lateral Sclerosis- Coordinating PI
2014- Lucca Cassa di Risparmio FOUNDATION- Mitochondrial involvement and receptor pharmacogenomics for immune response in multiple sclerosis- Coordinating PI
2012- Pisa Cassa di Risparmio FOUNDATION- TRAIN THE BRAIN: an interventional trial for mild cognitive impairment- PI of Partner Research Unit

Selected Publications

1. Maggi L, Bello L, Bonanno S, … Siciliano G, … Comi G, Pegoraro E. Nusinersen safety and effects on motor function in adult spinal muscular atrophy type 2 and 3. J Neurol

Neurosurg Psychiatry. 2020 Nov;91(11):1166-1174. doi: 10.1136/jnnp-2020-323822. Epub 2020 Sep 11. PMID: 32917822.

2. Palermo G, Mazzucchi S, Della Vecchia A, Siciliano G, …, Hampel H, Baldacci F. Different Clinical Contexts of Use of Blood Neurofilament Light Chain Protein in the Spectrum of Neurodegenerative Diseases. Mol Neurobiol. 2020 Nov;57(11):4667-4691. doi: 10.1007/s12035-020-02035-9. PMID: 32772223.
3. Doneddu PE, Cocito D, Manganelli F, …, Siciliano G, …Sabatelli M, Nobile-Orazio E; Italian CIDP Database study group. Atypical CIDP: diagnostic criteria, progression and treatment response. Data from the Italian CIDP Database. J Neurol Neurosurg Psychiatry. 2019 Feb;90(2):125-132. doi: 10.1136/jnnp-2018-318714. PMID: 30297520.
4. Nicolas A, Kenna KP, SLAGEN Consortium,Traynor BJ, Landers JE. Genome-wide Analyses Identify KIF5A as a Novel ALS Gene. Neuron. 2018 Mar 21;97(6):1268-1283.e6. doi: 10.1016/j.neuron.2018.02.027. PMID: 29566793.
5. Chico L, Ienco EC, Bisordi C, Lo Gerfo A, Mancuso M, Siciliano G. Amyotrophic Lateral Sclerosis and Oxidative Stress: A Double-Blind Therapeutic Trial After Curcumin Supplementation. CNS Neurol Disord Drug Targets. 2018;17(10):767-779. doi: 10.2174/1871527317666180720162029. PMID: 30033879
6. Train the Brain Consortium. Randomized trial on the effects of a combined physical/cognitive training in aged MCI subjects: the Train the Brain study. Sci Rep. 2017 Jan 3;7:39471. doi: 10.1038/srep39471. PMID: 28045051.
7. Stoccoro A, Siciliano G, Migliore L, Coppedè F. Decreased Methylation of the Mitochondrial D-Loop Region in Late-Onset Alzheimer's Disease. J Alzheimers Dis. 2017;59(2):559-564. doi: 10.3233/JAD-170139. PMID: 28655136
8. Magri F, Nigro V, Angelini C, … Siciliano G, … Bresolin N, Comi GP. The italian limb girdle muscular dystrophy registry: Relative frequency, clinical features, and differential diagnosis. Muscle Nerve. 2017 Jan;55(1):55-68. doi: 10.1002/mus.25192. Epub 2016 Oct 28. PMID: 27184587.
9. Costagli M, Donatelli G, Biagi L, Caldarazzo Ienco E, Siciliano G, Tosetti M, Cosottini M. Magnetic susceptibility in the deep layers of the primary motor cortex in Amyotrophic Lateral Sclerosis. Neuroimage Clin. 2016 May 2;12:965-969. doi: 10.1016/j.nicl.2016.04.011. PMID: 27995062.

Prof. Gabriele Siciliano

Pisa, October 31, 2022

CHAPTER 13. Inspirers & Supporters

13.11. Guglielmo Sorci

2018 Guglielmo Sorci 2022

Rigth panel. Advanced Course of Myology, in Assisi (23.10.2022), together with the course participants, people under 35. Front row, from left, Guglielmo Sorci, Davide Gabellini, Libero Vitiello, and Rosanna Piccirillo; back row, from left, Doug Millay, Sestina Falcone, and Scott Harper.

I met Guglielmo at one of the first Meetings of IIM (the Interuniversity Institute of Myology, founded by Giorgio Fano-Illic) and then every time I went to the IIM annual Meetings. He is not an aggressive person, so I only noticed him a few years later, when he became one of Antonio Musarò's supporters in organizing the IIM Meetings. He is now the Director of IIM and the key local organizer of the Meetings in Assisi, not far from Perugia, where he is now full professor of Human Anatomy in the Department of Medicine and Surgery of the University of Perugia, Italy. Author of EJTM articles, as soon as I asked him, he warmly agreed to act as the Editor of the EJTM Section: Mobility Medicine, Diagnostics and Managements, to write editorials and articles presenting the collection of abstracts of the recent IIM annual meetings. By my standard he is a perfect companion, sharing my same dreams: being a relevant basic scientist in the applications of new concepts, methods and protocols for the diagnosis and management of human muscle diseases.
He was also lucky enough to see his dreams come true.

CURRICULUM VITAE ET STUDIORUM

Guglielmo SORCI

Personal data: born in Spello (Perugia, Italy) on 30 March, 1965
Nationality: Italian
ORCID iD: 0000-0002-1973-9679
Web site: www.myolab-unipg.com

Full professor of Human Anatomy at Dept. Medicine and Surgery, University of Perugia (UniPG), Perugia, Italy
Scientific Coordinator of Sect. Human Anatomy, Dept. Medicine and Surgery, UniPG
Director of the Interuniversity Institute of Myology (IIM), an international association of researchers involved in the study of muscle physiology and pathologies (www.coram-iim.it)

EDUCATION
Graduated in Biology Summa cum laude with the honorable mention, "Press dignity" at UniPG, Perugia, Italy (17.07.1989)
Enabled as a professional Biologist at UniPG (November 1990)

MAIN SCIENTIFIC INTERESTS
The biology of the calcium-binding protein S100B and its receptor, RAGE (receptor for advanced glycation end-products) in muscle precursor cells, myogenesis, muscle regeneration, and skeletal muscle pathologies (sarcopenia, cachexia, muscle atrophy, rhabdomyosarcoma) with special emphasis in Duchenne muscular dystrophy (DMD).
Modifications induced in muscle precursor cells by the exposure to microgravity during space flights.
Use of microencapsulated Sertoli cells to counteract muscle degeneration in animal models of muscular dystrophy and inflammatory myopathies.

AFFILIATIONS AND MEMBERSHIP IN RESEARCH CENTERS
2019- : CIRTEMER (International Biotechnology Center for Translational Research in Endocrinology, Metabolism and Embryo-reproduction), UniPG (Scientific Committee);
2019- : Anatomy and Surgery Academy of Perugia, Italy (Executive board);
2018- : CIB (Consorzio Interuniversitario Biotecnologie), Italy (Responsible of Operative Unit);
2018- : CURGeF (University Research Center on Functional Genomics), UniPG (Scientific Committee);
2010- : IIM (Interuniversity Institute of Myology; www.coram-iim.it), Italy (Member of the Scientific Committee since 2010; Deputy Director 2020-2022; current Director);
2002- : Member of the Italian Society of Anatomy and Histology (SIAI)

EDITORIAL BOARDS
Frontiers in Cell and Developmental Biology - Stem Cell Research (Review Editor) (2022-)
European Journal of Translational Myology (2021-)
Frontiers in Physiology - Striated Muscle Physiology (Review Editor) (2021-)
Cell Death & Disease (Receiving editor; 2020-)
Journal of Functional Morphology and Kinesiology (2015-)
2021-2022: Co-Guest Editor Special Issue "State-of-the-Art of Myology in Italy", *Biomolecules* (MDPI)
2017: Guest Editor of Special Issue "Muscular Dystrophy", *J. Functional Morphology*

and Kinesiology
2019: Co-author of the book “Treatise of Topographical Anatomy”, I. Barajon et al., EdiErmes
2015: Co-curator of Netter Tables Selection for Atlas of Human Anatomy - Nurses, 5th ed. EDRA (ISBN 9788821440175)

EMPLOYMENT
2020- : Full Professor of Human Anatomy, Dept. Medicine and Surgery, UniPG
2018- : Scientific Coordinator of the Section of Human Anatomy - Dept. Medicine and Surgery, UniPG
2019- : Coordinator of the High Training Course in “Advanced Myology”, UniPG
2018–2020: Director of the First Level University Master in “Professional in Physical Activity for Prevention, Rescue and Optimization of Health” (P.A.M.P.R.O.S.), UniPG
2010-2020: Associate Professor of Human Anatomy at Dept. Experimental Medicine, UniPG
2002-2010: Assistant Professor of Human Anatomy, Faculty of Medicine, UniPG
1991-2002: Graduated Technician at the Section of Anatomy, Dept. Exp. Med. Biochem. Sci., UniPG

RESEARCH EXPERIENCE
1991-present: He has been involved in research projects granted by MURST/MiUR Italy, European Community (EU), CNR, Telethon-Italia, AIRC (*Associazione Italiana per la Ricerca sul Cancro*), AFM-Téléthon (*Association Française contre les Myopathies*), Duchenne Parent Project –Italy, Duchenne Parent Project -The Netherlands, CIB (*Consorzio Interuniversitario per le Biotecnologie*), Italfarmaco (Italy), ASI (Italian Space Agency), Fondazione Perugia, and University of Perugia.

TEACHING ACTIVITIES
2019 –“Muscle regeneration, muscle atrophy, and muscular dystrophy (DMD)” in the university high training course in “Advanced Myology Update”, UniPG, Italy
2002 –“Human Anatomy” in several degree Courses and Specialization Schools of the Dept Medicine and Surgery, UniPG, Italy

SUPERVISOR ACTIVITY
2003- : No. 5 Postdoctoral Fellows / No. 8 PhD Students / No. 7 Master Students / No. 2 foreign students of the International Federation of Medical Students' Association (IFMSA) / No. 108 First or Second Degree Course Students, UniPG, Italy

INSTITUTIONAL RESPONSIBILITIES
2019-2022: Member of the Quality Committee, UniPG
2018- : Member of the Scientific Committee of the University Center on Functional Genomics, UniPG
2018-2020: Member of the Council of the University Center for Scientific Museums (CAMS), UniPG
2017-2019: Member of the Education Committee, School of Medicine, UniPG
2014- : Member of the Scientific Committee of the Regional School of Sport C.O.N.I.

Umbria, Italy
2013- : Member of Teachers Board of the PhD Program in Systems Biology in Immunity and Infectious Pathologies, UniPG
2008–2013: Member of Teachers Board of the PhD Program in Biology and Experimental Medicine, UniPG

MEETINGS ORGANIZATION
2012- : he organized 22 meetings/seminars, and co-organized No. 11 IIM international meetings focused on muscle anatomy, physiology and pathologies.

REVIEWING ACTIVITIES
2016- : Member of the Referees Board of the Italian journal Scienze e Ricerche (ISSN 2283-5873)
2011: Review panel member in the ANVUR Evaluation of Research Quality VQR 2004-2010
2011- : Reviewer of research projects from MIUR-Italy, Université Pierre et Marie Curie (Paris, France), Research Foundation Flanders (FWO; Belgium), AFM-Téléthon (France), Swiss Foundation for Research on Muscle Diseases, The Netherlands Organisation for Scientific Research (NWO/ZonMw), Ministry of Health of Singapore, Italian Foundation Multiple Sclerosis (FISM), and Italian Universities.
2011- : *Ad hoc* reviewer for PLoS ONE, Am J Physiol-Regul Integr Comp Physiol, J Cell Mol Med, Curr Pharm Design, Int J Immunopathol Pharmacol, Cell Death Disease, Acta Physiol, J Spine, J Appl Physiol, Onco Targets Ther, Cell Cycle, Nat Commun, Cancer Med, Oncotarget, Immunopharmacol Immunotoxicol, Frontiers Physiol, Transl Cancer Res, Cancer Lett, Genes, FASEB J, Int J Mol Sci, Cells, Sci Rep, Life, Biomed Pharmacother, Eur J Transl Myol.

SCIENTIFIC DISSEMINATION
2021: "Youth is (even) a question of muscles", article by Andrea Barchiesi in the Italian journal, La Repubblica - Salute. Includes interview to G. Sorci on muscle atrophy, 31.07.2021
2021: Testimonial in the fundraising event "Dolomiti for Duchenne" by Parent Project aps, 18.06.2021
2021: Guest in "Houston, we have a problem", ApeRicerca - People meet UniPG Researchers; Perugia, 17.06.2021
2018- : Co-organizer of "Sharper, the European Night of Researchers" – Perugia, Italy
2018: Guest in "The MyoGravity project. Thinking a journey to Mars" XI Cycle of conferences at Liceo Jacopone da Todi; Todi, Italy, 09.11.2018
2017: Guest in "Intraperitoneal injection of microencapsulated Sertoli cells restores muscle morphology and performance in dystrophic mice" X Cycle of conferences at Liceo Jacopone da Todi; Todi, Italy, 31.03.2017 Todi, Italy
2012: Article "RAGE, the receptor that regenerates muscles" by F. Riuzzi, G. Sorci, R. Donato, in the journal "L'Università" (UniPG) n. 2 2012, pag. 7

PARTICIPATION TO TV AND RADIO PROGRAMS
2020: Interview by Parent Project aps "Final report of the project: Use of

microencapsulated Sertoli cells in Duchenne muscular dystrophy. Towards an application to patients” (http://parentproject.it/2020/08/06/report-finale-del-progetto-di-ricerca-utilizzo-di-cellule-di-sertoli-microincapsulate-nella-distrofia-muscolare-di-duchenne-verso-lapplicazione-sui-pazienti/)
2020: Guest in the radio program “L’Uovo di Colombo” UmbriaRadio, “Myolab team identified a potential biomarker and therapeutic target in cancer cachexia. G. Sorci & F. Riuzzi; 02.04.2020
2018: Guest in the national TV program “Medicina33” Rai2 - “The MyoGravity project”; 09.05.2018
2017-2020: Guest in several editions of the TV program “Speciale Università” TeF Channel, about Brain Awareness Week, the MyoGravity project, the Use of Sertoli cells to treat DMD, and Targeting the receptor RAGE to counteract cancer cachexia.

AWARDS

2017: The Parent Project association devolves the national Easter fundraising to the project “Use of microencapsulated Sertoli cells in Duchenne muscular dystrophy. Towards an application to patients”, PI Prof. Guglielmo Sorci.
2000: Honorable Mention in the International photocontest "Sigma 2000 Life Science Photo Contest", Cell Biology category.

SCIENTIFIC PRODUCTION

Co-author of 65 papers *in extenso* published in international scientific journals (total Impact Factor, 298.8; average Impact Factor, 5.34; H index, 31; Scopus citations, more than 4,000), and 184 congress communications (No. 31 on invitation).

1. Salvadori L., Belladonna M.L., Castiglioni B., Paiella M., Panfili E., Manenti T., Ercolani C., Cornioli L., Chiappalupi S., Gentili G., Leigheb M., Sorci G.[1], Bosetti M.[1], Filigheddu N.[1], Riuzzi F.[1] (2022) KYMASIN UP natural product inhibits osteoclastogenesis and improves osteoblast activity by modulating Src and p38 MAPK. Nutrients 14(15), 3053; doi: 10.3390/nu14153053. Cover figure assigned. [1] Shared senior authorship
2. Di Filippo E.S., Chiappalupi S., Balsamo M., Vukich M., Sorci G.[1], Fulle S.[1] (2022) Preparation of Human Muscle Precursor Cells for the MyoGravity Project - Cell Culture in Experiment Units for Space Flight Purpose. Appl. Sci. 12, 7013; doi: 10.3390/app12147013. [1] Shared senior authorship
3. Chiappalupi S., Salvadori L., Mancuso F., Arato I., Calvitti M., Riuzzi F., Calafiore R., Luca G., Sorci G. (2021) Microencapsulated Sertoli cells sustain myoblast proliferation without affecting the myogenic potential. In vitro data. Data Brief 40(107744); doi: 10.1016/j.dib.2021.107744
4. Salvadori L., Chiappalupi S., Arato I., Mancuso F., Calvitti M., Marchetti M.C., Riuzzi F., Calafiore R., Luca G., Sorci G. (2021) Sertoli Cells Improve Myogenic Differentiation, Reduce Fibrogenic Markers, and Induce Utrophin Expression in Human DMD Myoblasts. Biomolecules 11(10):1504; doi: 10.3390/biom11101504
5. Chiappalupi S., Salvadori L., Donato R., Riuzzi F., Sorci G. (2021) Hyperactivated RAGE in comorbidities as a risk factor for severe COVID-19–The role of RAGE-RAS crosstalk. Biomolecules 11:876; doi: 10.3390/biom11060876
6. Chiappalupi S., Salvadori L., Vukasinovic A., Donato R., Sorci G.[1], Riuzzi F.[1] (2021) Targeting RAGE to prevent SARS-CoV-2-mediated multiple organ failure: Hypotheses and perspectives. Life Sci. 272:119251; doi: 10.1016/j.lfs.2021.119251. [1] Shared senior authorship

7. Salvadori L., Mandrone, M., Manenti, T., Ercolani, C., Cornioli, L., Lianza, M., Tomasi, P., Chiappalupi, S., Di Filippo, E.S., Fulle, S., Poli F., Sorci G.[1], Riuzzi F.[1] (2021) Identification of Withania somnifera-Silybum marianum-Trigonella foenum-graecum formulation as a nutritional supplement to contrast muscle atrophy and sarcopenia. Nutrients 13:49; dx.doi.org/10.3390/nu13010049. [1] Shared senior authorship
8. Sorci G.[1], Gabellini D.[1] (2020) Report and Abstracts of the 17th Meeting of IIM, the Interuniversity Institute of Myology: Virtual meeting, October 16-18, 2020. Europ. J. Transl. Myol. 30(4):9485; doi: 10.4081/ejtm.2020.9485. [1] Contributed equally
9. Chiappalupi S.[1], Sorci G.[1], Vukasinovic A., Salvadori L., Sagheddu R., Coletti D., Renga G., Romani L., Donato R., Riuzzi F. (2020) Targeting RAGE prevents muscle wasting and prolongs survival in cancer cachexia. J. Cachexia Sarcopenia Muscle 11(4):929-946; doi: 10.1002/jcsm.12561. [1] Contributed equally. (Wiley Top Cited Article 2020-2021)
10. Bellezza I., Riuzzi F. Chiappalupi S., Arcuri C., Giambanco I., Sorci G., Donato R. (2020) Reductive stress in striated muscle cells. Cell. Mol. Life Sci. 77(18):3547-3565; doi: 10.1007/s00018-020-03476-0
11. Riuzzi F., Chiappalupi S., Arcuri C., Giambanco I., Sorci G.[1] and Donato R.[1] (2020) S100 proteins in obesity: liaisons dangereuses. Cell. Mol. Life Sci. 77:129-47. doi: 10.1007/s00018-019-03257-4. [1] Shared senior authorship
12. Chiappalupi S., Salvadori L., Luca G., Riuzzi F., Calafiore R., Donato R. and Sorci G. (2019) Do porcine Sertoli cells represent an opportunity for Duchenne muscular dystrophy? Cell Proliferation 26:e12599. doi: 10.1111/cpr.12599
13. Riuzzi F.[1], Sorci G.[1], Arcuri C., Giambanco I., Bellezza I., Minelli A. and Donato R. (2018) Cellular and molecular mechanisms of sarcopenia: the S100B perspective. J. Cachexia Sarcopenia Muscle 9:1255-68; doi: 10.1002/jcsm.12363. [1] Contributed equally
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18. Chiappalupi S., Salvadori L., Luca G., Riuzzi F., Calafiore R., Donato R. and Sorci G. (2017) Employment of microencapsulated Sertoli cells as a new tool to treat Duchenne muscular dystrophy. J. Funct. Morphol. Kinesiol. 2(4):47
19. Riuzzi F., Beccafico S., Sagheddu R., Chiappalupi S., Giambanco I., Bereshchenko O., Riccardi C., Sorci G.[1] and Donato R.[1] (2017) Levels of S100B protein drive the reparative process in acute muscle injury and muscular dystrophy. Sci. Rep. 7(1):1253; doi: 10.1038/s41598-017-12880-9. [1] Shared Senior authorship
20. Donato R., Sorci G. and Giambanco I. (2017) S100A6 protein: functional roles. Cell. Mol. Life Sci. 74(15):2749-60; doi: 10.1007/s00018-017-2526-9
21. Donato R., Sorci G. and Giambanco I. (2017) Le proteine S100. Ligand Assay 22(1):11-44. Cover figure assigned

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23. Chiappalupi S., Luca G., Mancuso F., Madaro L., Fallarino F., Nicoletti C., Calvitti M., Arato I., Falabella F., Salvadori L., Di Meo A., Bufalari A., Giovagnoli S., Calafiore R., Donato R. and Sorci G. (2015) Effects of intraperitoneal injection of microencapsulated Sertoli cells on chronic and presymptomatic dystrophic mice. Data Brief 5:1015-21; dx.doi.org/10.1016/j.dib.2015.11.016
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Chapter 14
Collaborators

Helmut Kern, Paolo Gargiulo, Amber Pond, Simona Boncompagni, Feliciano Protasi, Antonio Musarò, Nejc Sarabon, Marcello Cantini, Stefano Masiero, Dario Coletti, Corrado Angelini, Giovanna Albertin, Gianluca Rigatelli, Shelia Schills, Mauro Salvatore Alessandro Alaibac, and more than 150 other coauthors of my PubMed articles.

14.1. Helmut Kern

1998 2020 2022

Helmut KERN

I met Helmut Kern at one of the Vienna Workshop of Functional Electrical Stimulation (FES), perhaps in 1998, and then in 1999 when he came to visit me at the Department of Biomedical Sciences of the University of Padua during one of his stays at the Continental Hotel in Montegrotto Terme (Padua), where, as a lover of the Euganean Hills and warm swigging pools, he was used to ride up and down the hills avoiding the bad weather of Vienna.

Helmut had brought with him his postgraduate thesis in Rehabilitation that described his experiences as a young Rehabilitation Specialists, who had treated the first Wien cases of implantation of FES stimulators in paraplegic patients. He asked for my opinions and offered me the opportunity to collaborate to collect more solid evidence of muscle improvements even in the worst cases, that is, those of patients with permanent denervation of the legs due to severe injuries of the Conus Cauda.

My immediate response was: Dear Helmut, could you collect biopsy samples from the treated leg muscles? As he was very positive about this approach, a longstanding collaboration began almost immediately and continues today. The first paper was published in 2004, but the previous chapters of this book described in details what happened during 20 years of collaborations!

Helmut Kern and I are indebted with all Partners of the European Project RISE: Use of electrical stimulation to restore standing in paraplegics with long-term denervated degenerated muscles (Contract no. QLG5-CT-2001-02191): M. Bijak and E. Unger, Biomedical Technology Center, Vienna, Austria; H. A. Cerrel Bazo, Neuromotor Rehabilitation, Cernusco, Milan, Italy; M. R. Dimitrijevic, Physical Medicine and Rehabilitation, Baylor College of Medicine, Houston, TX, USA; G. Exner, Spinal Cord Injury Center, Hamburg, Germany; E. Gallasch, Physiology, Graz, Austria; H. J. Gerner and R. Rupp, Orthopedics, Heidelberg, Germany; W. Girsch, Orthopedics, Speising, Vienna, Austria; T. Helgason, P. Ingvarsson, and S. Yngvason, Landspitali-University Hospital, Reykjavik, Iceland; J. Hufgard and M. Obrovsky, Rehabilitation, Klosterneuburg, Austria; H. P. Jonas, Rehabilitation, Bad Häring, Tirol, Austria; S. Lotta, Villanova sull'Arda (PC), Italy; D. Maier and M. Potulski, Murnau, Spinal Cord Injury, Murnau, Germany; D. Rafolt, Institut für Biomedizinische Technik und Physik, Vienna, Austria.
We are also indebted with all the coauthors of the papers reporting RISE and Mobility in Aging results published from 2002 to date: Abruzzo PM, Adami N, Barberi L, Bassetto F, Biral D, Boato N, Boncompagni S, Bosco G, Burggraf S, Coletto L, Corbianco S, Cvecka J, Danieli-Betto D, De Rossi M, di Tullio S, Doria A, Fanò G, Ferrero M, Forstner C , Francini F, Franz C, Fruhmann H, Fulle S, Gargiulo P, Germinario E, Grim-Stieger M, Hamar D, Helgason B, Helgason T, Hoellwarth U, Hofer C, Ingvarsson P, Kovarik J, Krenn M, La Rovere R, Lapalombella R, Löfler S, Mancinelli R, Marcante A, Marini M, Masiero S, Mayr W, Merigliano S, Mödlin M, Mosole S, Musarò A, Nori A, Pond A, Paolini C, Paternostro- Sluga T, Pelosi L, Pietrangelo L, Pietrangelo T, Podhorska-Okolow M, Pond A, Protasi F, Rampudda ME, Reynisson PJ, Romanello V, Rossini K, Rupp R, Salmons S, Sandri M, Sarabon N, Sarzo G, Scordari A, Sedliak M, Squecco R, Stramare R, Tirpáková V, Trimmel L, Valente M, Vecchiato M, Vindigni V, Vogelauer M, Zampieri S, Zanato R, Zanin ME.

Curriculum of Helmut Kern

Personal Information:

Name:	Prim. Univ.-Prof. DDr. Helmut Kern
Born:	February 25, 1951, Pöchlarn, Austria
Residence:	Vienna, Austria
Pers. status:	married, 3 children

Professional Experience

2019-to date	Partner Board Member of the "Ludwig Boltzmann Institute of Rehabilitation Research", St. Pölten & Vienna, Austria
1990-to date	Medical and Managing Director of 7 Institutes for Physical Medicine and Rehabilitation in Lower Austria and Vienna
1987-2019	Founder and Head of the Research Institute "Ludwig Boltzmann Institute of Electrical Stimulation and Rehabilitation", Vienna, Austria
1984-2016	Head of the "Department of Physical Medicine and Rehabilitation" Wilhelminenspital, Vienna, Austria
1988-2009	Medical director of "Academy of Physiotherapy", Wilhelminenspital, Vienna, Austria

Founder and 1st President of the Austrian Society for Sports-Physiotherapy

Education

2012 "University Professor" for Physical medicine & Rehabilitation
1996 Venia docendi; „Functional Electrical Stimulation on paraplegic patients" for Physical medicine & Rehabilitation
1995 Dr. rer. nat. in Sports Science; „Electrical Stimulation in Sports and Rehabilitation"; Faculty of Natural Sciences, University of Vienna
1983- 1984 Residency; University Clinic of Orthopedics, Heidelberg, Germany
1979-1984 Residency; Dept. of Physical Medicine and Rehabilitation, University Clinic, Vienna
1978-1983 Studies of sports science, University of Vienna
1977-1979 Residency at Hospital Lainz, KAV, Vienna
1977 MD Graduation at the University of Vienna

Projects, Publications, Presentations and Metrics

6 international EU projects, 5 of these in cooperation with Commenius University in Bratislava
2001-2006 "RISE" - Use of electrical stimulation to restore standing in paraplegics with longterm denervated degenerated muscles DDM
2003-2006 Interreg IIIa: "Medical and sports science cooperation"
2005-2008 Interreg IIIa: "Grenzenlos Bewegen" after K-TEP implantation
2008-2014 Interreg IVa: "Mobility in Aging"
2019-2022 Interreg Va: "Centre of Active Ageing"
2021-2022 Interreg Va: Ambulant Remobilisation after Knie-TEP and Hip-TEP implantation

9 national research projects in Austria

third-party funding of more than € 4 million

276 scientific publications; >7000 citations; 44 h-index.

Editorial Boards

2014 to date Physikalische Medizin, Rehabilitationsmedizin, Kurortmedizin ISSN 0940-6689
2010 to date Annales Kinesiologiae ISSN 2232-2620
2004 to date Basic and Applied Myology – European Journal of Translational Myology ISSN 1120 -9992
2000 to date Sportverletzung Sportschaden ISSN 0932-0555

Selected Publications

1. Linear Motor Driven Leg-Press Dynamometer for Testing, Training, and Rehabilitation: A Scoping Review with a Focus on the Concept of Serial Stretch Loading. Cvečka J, Krčmár M, Hamar D, Kern H, Hofer C, Löfler S, Vajda M. Int J Environ Res Public Health. 2022 Apr 7;19(8):4445. doi: 10.3390/ijerph19084445.
2. Trauma of Peripheral Innervation Impairs Content of Epidermal Langerhans Cells. Albertin G, Ravara B, Kern H, Zampieri S, Loefler S, Hofer C, Guidolin D, Messina F, De Caro R, Alaibac M, Carraro U. Diagnostics (Basel). 2022 Feb 23;12(3):567. doi: 10.3390/diagnostics12030567.
3. Post-meeting report of the 2022 On-site Padua Days on Muscle and Mobility Medicine, March 30 - April 3, 2022, Padua, Italy. Carraro U, Bittmann F, Ivanova E, Jónsson H Jr, Kern H, Leeuwenburgh C, Mayr W, Scalabrin M, Schaefer L, Smeriglio P, Zampieri S. Eur J Transl Myol. 2022 Apr 13;32(2):10521. doi: 10.4081/ejtm.2022.10521.
4. Skeletal muscle weakness in older adults home-restricted due to COVID-19 pandemic: a role for full-body in-bed gym and functional electrical stimulation. Carraro U, Marcante A, Ravara B, Albertin G, Maccarone MC, Piccione F, Kern H, Masiero S. Aging Clin Exp Res. 2021 Jul;33(7):2053-2059. doi: 10.1007/s40520-021-01885-0.
5. Physical Abilities in Low Back Pain Patients: A Cross-Sectional Study with Exploratory Comparison of Patient Subgroups. Šarabon, N., Vreček, N., Hofer, C., Löfler, S. Kozinc, Ž., Kern,H. Life. 2021;11:226. https://doi.org/10.3390/life11030226
6. To contrast and reverse skeletal muscle weakness by Full-Body In-Bed Gym in chronic COVID-19 pandemic syndrome. Carraro U, Albertin G, Martini A, Giuriati W, Guidolin D, Masiero S, Kern H, Hofer C, Marcante A, Ravara B. Eur J Transl Myol. 2021 Mar 26;31(1):9641. doi: 10.4081/ejtm.2021.9641.
7. Paolo Gava, a professional engineer, who has become a Master athlete, an amateur scientist and a lifelong friend. Carraro U, Kern H, Albertin G. Eur J Transl Myol. 2021 Nov 5. doi: 10.4081/ejtm.2021.10260. Online ahead of print.
8. Home-Based Functional Electrical Stimulation of Human Permanent Denervated Muscles: A Narrative Review on Diagnostics, Managements, Results and Byproducts Revisited 2020. Kern H, Carraro U. Diagnostics (Basel). 2020 Jul 29;10(8):529. doi: 10.3390/diagnostics10080529.
9. Speed-power based training in the elderly and its potential for daily movement function enhancement. Šarabon N, Smajla D, Kozinc Ž, Kern H. Eur J Transl Myol. 2020 Apr 1;30(1):8898. doi: 10.4081/ejtm.2019.8898. eCollection 2020 Apr 7
10. Influence of electrical stimulation therapy on permanent pacemaker function. Egger F, Hofer C, Hammerle FP, Löfler S, Nürnberg M, Fiedler L, Kriz R, Kern H, Huber K. Wien Klin Wochenschr. 2019 Jul;131(13-14):313-320. doi: 10.1007/s00508-019-1494-5.
11. Cayenne Pepper Cataplasm "Munari" reduces pain and improves mobility in patients with non-specific chronic low back pain. Zampieri, S., Sarabon, N., Löfler, S., Hofer, C., Sajer, S., Kabas, F., Cvecka, J., Sedliak, M., Krenn, M., Hüebl, W., & Kern, H. Physical Medicine and Rehabilitation Research. 2019 4(2). https://doi.org/10.15761/PMRR.1000202
12. Blood contamination, a problem or a lucky chance to analyze non-invasively Myokines in mouth fluids? Ravara B, Zampieri S, Kern H, Carraro U. Eur J Transl Myol. 2019 Dec 10;29(4):8713. doi: 10.4081/ejtm.2019.8713. eCollection 2019 Oct 29.

13. Dermal papillae flattening of thigh skin in Conus Cauda Syndrome. Ravara B, Hofer C, Kern H, Guidolin D, Porzionato A, De Caro R, Albertin G. Eur J Transl Myol. 2018 Dec 13;28(4):7914. doi: 10.4081/ejtm.2018.7914. eCollection 2018 Nov 2.
14. Use it or Lose It: Tonic Activity of Slow Motoneurons Promotes Their Survival and Preferentially Increases Slow Fiber-Type Groupings in Muscles of Old Lifelong Recreational Sportsmen. Mosole S, Carraro U, Kern H, Loefler S, Zampieri S. Eur J Transl Myol. 2016 Nov 25;26(4):5972. doi: 10.4081/ejtm.2016.5972. eCollection 2016 Sep 15.
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17. Physical exercise in aging human skeletal muscle increases mitochondrial calcium uniporter expression levels and affects mitochondria dynamics. Zampieri S, Mammucari C, Romanello V, Barberi L, Pietrangelo L, Fusella A, Mosole S, Gherardi G, Höfer C, Löfler S, Sarabon N, Cvecka J, Krenn M, Carraro U, Kern H, Protasi F, Musarò A, Sandri M, Rizzuto R. Physiol Rep. 2016 Dec;4(24):e13005. doi: 10.14814/phy2.13005.
18. Editorial: The EJTM Special "Mobility in Elderly". Kern H, Jakubiec-Puka A, Carraro U. Eur J Transl Myol. 2015 Aug 25;25(4):208-13. doi: 10.4081/ejtm.2015.5412. eCollection 2015 Aug 24.
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CHAPTER 14. Collaborators
14.2. Paolo Gargiulo

2008 Paolo Gargiulo 2022

I meet Paolo Gargiulo in Vienna, Austria, where he was a PhD student in Medical Engeneering with Prof. Winfried Mayr of the University of Vienna, after his graduation in Medical Enegeneering at the University of Reykjavik, Icaland. It was easy to spend a few time with him and start a collaboration speaking in Italian!
I visited Reykjavik and the friends I had there, in particular Thordur Helgason and every time with Paolo we exchanged ideas for collaborating, but the key discussion we had was during the drive to the airport. Talking about his methods to quantify density by CT scan of bone, cartilage and MUSCLE I suggested to use false colors to distinguish normal from denervation-degenerating muscle tissue, something Cardiologists were initiating to publish to indentify infarcted cardiac tissue: the 3D Color Skeletal Muscle Imaging was impleneted in few days by Paolo and a very successful and clinical relevant method was established.
Paolo was able to develop 3D Color Skeletal Muscle Imaging to bone and cartilage, further extending it by Artificial Inteligence approaches obtaining clinical significant powerful methods.

One of our dreams had come reality!

Curriculum of Paolo Gargiulo

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Paolo Gargiulo is full Professor and works at center of Medical Technology Center - Reykjavik University /University Hospital Landspitali. He studied at TU Wien and finished his PhD in 2008. Paolo interests and expertise are mostly in: Medical Image processing, Neuroengineering, 3-D printing and Medical technologies. He developed at Landspitali a 3D-Printing service to support surgical planning with over 200 operation planned with a significant impact on the Icelandic health care system and he currently cooperate with institutions in Italy and UK to establish similar infrastructures. He has been a consultant for MedEl (from 2010 to 2016) for the development of larynx pacemaker.

Since December 2013 Paolo Gargiulo is the director of the Institute of Biomedical and Neural Engineering and the Icelandic center of Neurophysiology and manages the center of Medical Technology at the University Hospital Landspitali/ Reykjavik University. Thanks to domestic cooperation's with Össur, University of Iceland, Decode and the Icelandic Heart association and the support of infrastructure grants from RANNIS, Paolos lab currently include the following facilities: high density Electroencephalographic system (256-EEG), Postural control platform and Virtual reality system, polyjet 3D printer and multimetric Biosignal platform.

He has published 80 papers in peer reviewed international journals, several chapters in academic books and presented his work in many international conferences and workshops. He has been collaborating as associated editor for EJTM since 2015 and he is currentely member of the journal scientific board.

In January 2019 he received an EU grant, H2020-NMBP-TR-IND-2018-2020, with a project entitled: RESTORE User-centred smart nanobiomaterial-based 3D matrices for chondral repair. The Icelandic team will be responsible for the 1st European Database of patient-specific anatomical models for condyle lesions. In January 2022 he received a EU grant called SINPAIN with aim of Developing a Next-Generation Advanced Therapy for Knee Osteoarthritis

GRANT RECEIVED IN 2018-2022

2022: EU Project, Developing a Next-Generation Advanced Therapy for Knee Osteoarthritis: Eurice is Partner of Game-Changing Horizon Europe Project SINPAIN. EUR 5,3 million

2022: Center of Additive manufacturing, Icelandic technology road map: 0.5 M euro funding from Rannis infrastructure

2021: Innovation fund (nyskopunarsjodur): metal 3d printer (co-proposer)

2019: EU project, H2020-NMBP-TR-IND-2018-2020, User-centred smart nanobiomaterial-based 3D matrices for chondral repair, PI for the Icelandic group. 5.500.000 EURO

2018: Innovation fund (nyskopunarsjodur): upgrade of X-RAY micro CT system , project number 181572-0031, 75.000 EURO (CO –PROPOSER)

Research

Development and assessment of numerical profiles based on radiodensitometric distributions characteristics from computed tomography images. Thanks to this approach we have been demonstrating correlations between soft tissues and several biometrics parameters and comorbidity using the AGES database from the Icelandic heart association. The main potential impact of this work is possibility to predict a number of conditions such as cholesterol, diabetes and cardiovascular risks from a single CT image. I would like also to remark that this methodology and some applications were published in a chapter book on the: Encyclopedia of Biomedical Engineering, 2019; Vol. 2: 119-34. Elsevier. P Gargiulo, MK Gislason, KJ Edmunds . CT-Based Bone and Muscle Assessment in Normal and Pathological Conditions.

Neural engineering and postural control assessment. In 2013 I have established a facility based on high density EEG system where we have been study cortical changes and brain connectivity during postural control adaptation and habituation. Currently we are also assessing with our technology (HD-EEG) the effect of transcranial magnetic stimulation effect on schizophrenic patients in the frame of a scientific project in collaboration with the department of neurology at Landspitali, Iceland. The educational impact of this infrastructure and of its work is considerable since this lab provide teaching modules for different courses and a scientific facility for several scientists in Iceland and abroad.

Rehabilitation engineering. For several years we have been working on the optimisation of prosthetic implant decision making in patients undergoing total hip replacement, collecting multi metric data from these patients at different points of time, before and after implant. This work is still ongoing and have as ultimate goal to provide surgeons with an application tool that elaborate biometrics data and computational data to support their decision. However thanks to the international resonance of this work we have been participating and received an EU grant H2020-NMBP-TR-IND-2018-2020, with a project entitled: RESTORE User-centred smart nanobiomaterial-based 3D matrices for chondral repair. In the frame of this project my team will be responsible for the 1stEuropean Database of patient-specific anatomical models for condyle lesions.

3-D printing and Surgical planning. In 2005 I developed and established at Landspitali a 3D-Printing service to support surgical planning with over 200 operation planned with a significant impact on the Icelandic health care system. Thanks to this experience and know how I currently cooperate with institutions in Italy and UK to establish similar infrastructures.

Publications

Ciliberti FK, Cesarelli G, Guerrini L, Gunnarsson AE, Forni R, Aubonnet R, Recenti M, Jacob D, Jónsson H Jr, Cangiano V, Islind AS, Gambacorta M, Gargiulo P. The role of bone mineral density and cartilage volume to predict knee cartilage degeneration. Eur J Transl Myol. 2022 Jun 28;32(2):10678. doi: 10.4081/ejtm.2022.10678.

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CHAPTER 14. Collaborators
14.3. Amber L. Pond

April 16, 2016 **Amber L. Pond** **2022**

2016 Spring Padua Muscle Days : From left: Ugo Carraro, Paolo Gargiulo, Amber L. Pond, Magnús Gislason and Halla Kristín Guðfinnsdóttir (Photo credit: Amber L. Pond)

About 1995, I went to the Purdue University for a Conference on Dynamic Cardiomyoplasty. There, I met and shared my sympathies with one of the organizers, a biomedical engineer, who, after Medtronic ended its efforts to sell pacemakers to heart failure patients, was among the patent owners of a most clinically successful cardiac pacemaker, namely the implantable defibrillator. Over the next few years we have kept in touch by emailing and meeting at international conferences. Then, in 2008, he wrote me that he had decided to visit Italy, including Venice, with his wife and another couple. He was asking me for advice on the destinations of their trip. It was easy to convince them to extend their visit to Venice by spending a few days in Padua. Since his friend worked in Veterinary Medicine, next to the historic Galileo Galilei Chathedra and the Anatomical Theater at Palazzo Bo of the University of Padua, I took them to visit the new Veterinary Faculty just outside the city. Upon returning home they wrote me their gratitude for having convinced them to cancel their visit to Lake of Como to spend three days in Padua and to spend the rest of their time in Italy visiting see-side "Cinque Terre" in Liguria. I don't know who was between them and a young Purdue University post.doc, but I do know that after a couple of months I got the first email from Amber. But the full story follows below. I am grateful to Amber because, not only has she made my Paduan-English readable, but because she has done much more from the beginning, underlining in Introduction and Discussion of our over-focused typescripts implications of our results and further developments of our researches, we weren't aware of. From this to a stricter collaboration on new research topics the way was short. Amber accepted invitation and is a member of the Editorial Board of BAM / EJTM. She also was a frequent speaker at the Padua Muscle Days. For the last two 2021 and 2022 Padua Days on Muscle and Mobility Medicine she was a log-distance Speakers by Zoom, something that fully agreed with our usual collaboration style.

Thank you so much Amber, for what you have done over the past twelve years and for what you will contribute over the next twelve!

Although I am listed under the "collaborators" section, I am indeed an unofficial "pupil" of Ugo Carraro. From across many miles and a huge ocean, Ugo has influenced my science, my career, and my outlook on life. We were introduced through email by a mutual colleague when I was working at Purdue University as a Research Associate, having just completed an NIH funded postdoctoral fellowship in the Biochemistry department there. Ugo wanted to acquire a different perspective on a paper upon which he and some colleagues were working. He asked me to collaborate and I was elated to do so! And, so it began!

Ugo and I have worked together since 2009. Initially, my role was mainly reviewer and editor of some work Ugo and his group were doing. However, we discovered that we worked extremely well together and we began exchanging research samples and ideas – with most of the really good ideas flowing westward to me! Ugo has acted as a collaborator and mentor, giving me excellent advice on science and my career. He has introduced me to collaborators and broadened my work and my visions. Most importantly, Ugo has influenced my outlook on my career and my life.

His positivity and forward thinking have truly given me many smiles and helped me to dream.

Curriculum of Amber Pond

Amber Pond, PhD is an Associate Professor with the Southern Illinois University School of Medicine in Carbondale, IL. She is with the anatomy department where she currently teaches physiology and pharmacology to medical students and researches the role of the ERG1 potassium channel in skeletal muscle pathology.

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Wang, Z., J. Feng, J. Shi, **A.L.Pond**, J.M. Nerbonne and S. Nattel. 1999. The potential molecular basis of different physiological properties of transient outward current in rabbit and human atrial myocytes. *Circulation Research* 84:551-561.

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Peebles, E.D., **A.L. Pond**, J.R. Thompson, C. R. McDaniel, N.M. Cox and M.A. Latour. 1997. Naloxone attenuates serum corticosterone and augments serum glucose concentrations in ACTH-stimulated broilers. *Poultry Science* 76:511-515.

Pond, A.L., C.P. Coyne, H.W. Chambers and J.E. Chambers. 1996. Identification and isolation of two rat serum proteins with A-esterase activity. *Biochem. Pharmacol.* 52:363-369.

Latour, M. A., S.A. Laiche, J.R. Thompson, **A.L. Pond** and E.D. Peebles. 1996. Continuous infusion of adrenocorticotropin elevates circulating lipoprotein cholesterol and corticosterone concentrations in chickens. *Poultry Science* 75:1428-1432.

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Carraro U, Kern H, Gava P, Hofer C, Loefler S, Gargiulo P, Edmunds K, Dröfn Árnadóttir I, Zampieri S, Ravara B, Gava F, Nori A, Gobbo V, Masiero S, Marcante A, Baba A, Piccione F, Schils S, **Pond A**, Mosole S. Recovery from muscle weakness by exercise and FES: Lessons from Masters, active or sedentary seniors and SCI patients. September, 2016. *Aging Clin Exp Res.* DOI: 10.1007/s40520-016-0619-1. PubMed PMID: 27592133.

Invited Review Papers

Carraro U, Kern H, Albertin G, Masiero S, **Pond AL**, Gargiulo P. Functional Electrical Stimulation of Permanently denervated muscles, updated 2020. 2020. Russian Bulletin of Rehabilitation Medicine 3(XX). Just Accepted, May 2020.

Pond AL, Marcante A, Zanato R, Stramare R, Vindigni V, Zampieri S, Kern H, Masiero S, Piccione F. History, mechanisms, clinical value and future of fibrillation analyses in muscle denervation and reineervation by single fiber EMG and dynamic echomyography. 2014. *European J of Translational Myology* 24(1):124-133.

Pond AL, Nerbonne JM. 2001. ERG proteins and functional cardiac I_{Kr} channels inrat, mouse and human heart. *Trends in Cardiovascular Medicine* 11(7):286-294.

Book Chapters

Ugo Carraro, Helmut Kern, Sandra Zampieri, Paolo Gargiulo, **Amber Pond**, Francesco Piccione, Stefano Masiero, Franco Bassetto, Vincenzo Vindigni. Muscle Fiber Regeneration in Long-Term Denervated Muscles: Basics and Clinical Perspectives. 2019. In: Duscher D., Shiffman M. (eds) *Regenerative Medicine and Plastic Surgery*. pp. 301-309. Springer, Cham. https://doi.org/10.1007/978-3-030-19962-3_21. Online ISBN 978-3-030-19962-3.

Helmut Kern, Paolo Gargiulo, **Amber Pond**, Giovani Albertin, Andrea Marcante, Ugo Carraro. To Reverse Atrophy of Human Muscles in Complete SCI Lower Motor Neuron Denervation by Home-Based Functional Electrical Stimulation. 2018. In: Xiao J. (eds) *Muscle Atrophy*. *Advances in Experimental Medicine and Biology*, Vol 1088, pp. 585-591. Springer, Singapore. https://www.springer.com/us/book/9789811314346#aboutAuthors.
https://doi.org/10.1007/978-981-13-1435-3. ISBN 978-981-13-1435-3 (eBook).

GRANT AWARDS

July 1, 2018-December 31, 2020. $200,000 from U.S. Department of Defense for a project titled: "The Modulation of Intracellular Calcium by the ERG1A K^+ Channel in Skeletal Muscle."

January 2016-present: SIU School of Medicine "Start Up" funds to Assistant Professor.

July 2014 – June 2015. Research Seed Grant Award for an application titled: "Investigation into the Role of the ERG1 Potassium Channel in Denervation Atrophy." This award ($15,000) is funded by the Southern Illinois University School of Medicine.

June 2009 - May 2013. NIH R03 for a project titled: "Role of Merg1a Potassium Channel in the Onset of Skeletal Muscle Atrophy." This award ($150,000 total direct) was funded by the NIH National Institute of Arthritis and Musculoskeletal and Skin Diseases.

February 2010. SVM Major Equipment Grant titled: "AKTA Prime Plus fast performance liquid chromatography (fplc) system." This one time award ($24,000 total) was awarded by the Purdue University School of Veterinary Medicine.

July 1, 2002 - June 31, 2006. Scientist Development Grant for an application titled "Composition and Regulation of the Cardiac I_{Kr} Channel." This award ($260,000 total) was funded through the American Heart Association.

September 1997 – June 1999. NIH Department of Health and Human Services. Individual National Research Service Award Postdoctoral Fellowship (1F32HLO9653-01) for a grant application titled "Distribution and Structure of HERG Cardiac Potassium Channel."

CHAPTER 14. Collaborators
14.4. Simona Boncompagni


2008 Simona Boncompagni 2022

Left Panel: From left to right: Feliciano Protasi, **Simona Boncompagni** and Ugo Carraro in Vienna to collect the first muscle biopsis and to teach friends there how to prepare and fix muscle samples for electron microscopy. Right panel: Simona today.
(Photo credit: Simona Boncompagni)

One of the major byproducts of supporting Giorgio Fanò-Illic proposal to establish the Interuniversity Institute of Myology (IIM) was an invitation of Giorgio to Chieti University to present some potential topics for collaboration. Of course 99% of my slides were on preliminary results of the European Project RISE, led by Helmut Kern. I included also some preliminary data of electron microscopy of long-term denervated muscles before and after hbFES made in Padova by Valerio Gobbo (see Chapter 12). I was very lucky that in the audience were present Feliciano Protasi, just moved to Chieti University after his long stay in the States in particular in the electro microscopy lab of Clara Franzini-Armstrong, and a pupil of him Simona Boncompagni. Immediately after my seminar the possibility of a collaboration was explored, fixed and implemented in a few months. After a first session of electron microscopy observation of samples harvested in Vienna that Simona and I made in Chieti providing strong evidence of the effectiveness of hbFES for denervated degenerating human muscles, we went to Vienna to establish a long-running collaboration that is producing valuable results up date.

It is an honor for me that Simona and Feliciano decided to be present in this book sending their CV and a few pictures of those exciting times.

Curriculum of Simona Boncompagni

Researcher unique identifier: ORCID iD: 0000-0001-5308-5069

Date of Birth: 12 January 1974 - Place of Birth: Sansepolcro (AR), Italy –
Status: Married
Office Address: CAST, Center for Advanced Technological Studies, room 458
G. D’Annunzio University, Chieti I-66100 Italy
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Current Position:
Associate Professor of Physiology
DNICS - Dept. of Neuroscience, Imaging and Clinical Sciences
G. d’Annunzio University, Chieti I-66100 Italy

Bibliometric Parameters (up to 2022)

Peer Reviewed Original Articles:	75
H index :	34
Total number of Citations:	3776

Education:
2000 Laurea in Chimica - Magna cum laude Perugia University - Italy
2006 Ph.D. - Degree in Physiopathology of Muscle, Department of Basic and Applied Medical Sciences, Ce.S.I. Center of Research on Aging; G. D’Annunzio University, Chieti I-66100 Italy
Supervisor: Prof. Feliciano Protasi

Previous Position:
From March 2020
Ph.D. Student
Department of Basic and Applied Medical Sciences,
Ce.S.I. Center of Research on Aging;
G. D’Annunzio University, Chieti I-66100 Italy.
Supervisor: Prof. Feliciano Protasi
Post-Doctoral Fellow
Department of Basic and Applied Medical Sciences,
Ce.S.I. Center of Research on Aging;
G. D’Annunzio University, Chieti I-66100 Italy
Researcher TD (Moratti)
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G. D’Annunzio University, Chieti I-66100 Italy
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SSD BIO/09 - Physiology
DNICS - Dept. of Neuroscience, Imaging and Clinical Sciences
G. D’Annunzio University, Chieti I-66100 Italy
Researcher TD type B (Senior, Assistant Professor Equivalent)
SSD BIO/09 - Physiology

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G. d'Annunzio University, Chieti I-66100 Italy
Associate Professor of Physiology
DNICS - Dept. of Neuroscience, Imaging and Clinical Sciences
G. d'Annunzio University, Chieti I-66100 Italy

Fellowships:
2006 - 2011
Visiting Researcher
University of Pennsylvania School of Medicine
Dept. of Cell and Developmental Biology
Philadelphia, PA - USA (laboratory of Prof. Clara Franzini-Armstrong)

Teaching Activities and other appointments:
from 2019 Lecturer of Human Physiology and Neurophysiology
Faculty of Medicine and Surgery, Degree in Physiotherapy
School of Medicine and Health Sciences. G. d'Annunzio Univ., Chieti –Italy
Lecturer of Human Physiology
Faculty of Medicine and Surgery, Degree in Radioterapy
School of Medicine and Health Sciences. G. d'Annunzio Univ., Chieti –Italy
Lecturer of Human Physiology and Neurophysiology
Faculty of Medicine and Surgery, Degree in Orthoptic and Ophtalmologic Assistance
School of Medicine and Health Sciences. G. d'Annunzio Univ., Chieti –Italy
Committee member of PhD scholarship
Course: Medical Biotechnology. School of Medicine and Health Sciences
G. d'Annunzio Univ., Chieti –Italy
Committee member of UdA University Research (www.ricerca.unich.it)
Università degli Studi G. D'Annunzio di Chieti-Pescara.

Funding history:
FIRB-Futuro in Ricerca 2013 – MIUR (Project: RBFR13A20K)
Multi-center project
Title of the project: Structural and functional alterations in Central Core Disease (CCD): understand molecular genetic background to develop therapeutic interventions. Role: Principal Investigator and Coordinator (587.561,31 euro/3 years)

Ricerca Finalizzata - Italian Ministry of Health (Project: GR-2011-2352681)
Title of the projec: Central core Disease: understand the molecular mechanisms leading to mitochondrial damage in cores to develop effective pharmacological treatments. Role: Principal Investigator (212.087,76 euro/3 years)

NIH-NIAMS
Project RO1 AR059646-06 (Lan-Way Lapierre)
Title: Using mitochondrial Ca2+ uptake as a therapeutic target for ALS.
Co-investigator ($ 162.000/5 years)

Awards and Recognitions:
EMC - European Muscle Conference 2011 (Berlin, Germany)
Young Investigators Award
Title of the presentation: Gradual formation and accumulation of tubular aggregates in fast-twitch muscle fibers: SERCA and calsequestrin involvement.
2018 National Scientific Qualification (ASN): Associate Professor
MIUR (Italian Ministry of University and Research) 05/D1 - Physiology
National Scientific Qualification to be able to apply for an associate professor position at any Italian University.
National Scientific Qualification(ASN): Full Professor
MIUR (Italian Ministry of University and Research) 05/D1 - Physiology
National Scientific Qualification to be able to apply for a full professor position at any Italian University.

Professional Society Involvement:
Member of the scientific committee of Young Investigators Award. EMC - European Muscle Conference 2019 – Canterbury, United Kingdom

Invited Speaker at International Meetings (selection)
Nov. 2008 Pennsylvania Muscle Institute Retreat and Symposium (Philadelphia-PA, USA)
Title: Clues to the formation of cores in a mouse model of Malignant Hyperthermia.
June 2009 Gordon Research Conference on EC coupling (New London, NH).
Title: Clues to the formation of cores in mouse models of malignant hyperthermia and central core disease.
Sept. 2011
Italian Physiological Society (Sorrento, NA)
Symposium: Signaling between Mitochondria and Ca2+ stores in skeletal muscle function and disease.
Title: Mitochondria coupling to calcium stores in skeletal fibers.
June 2012
Gordon Research Conference sull'Accoppiamento EC (Les Diablerets , Switzerland).
Title: Reciprocal positioning of CRU and mitochondria in vertebrate skeletal muscle: evolution and age dependence.
March 2015 Spring Padua Muscle Days (Padova, Italy)
Title: Ageing causes severe ultratrcutural modification of calcium release units and mitochondria in cardiomyocites.

June 2017 Spring Padua Muscle Days (Padova, Italy)
Title: Dysfunctional accumulation of STIM1 and Orai1 in Tubular Aggregates
results in impaired Ca2+ entry in ageing muscle.
Gordon Research Conference sull'Accoppiamento EC (Les Diablerets , Switzerland). Title: Exercise-dependant formation of new SR-TT junctions which promotes increased STIM1-Orai1 colocalization.
August 2017 II International Symposium of experimental Pathology (ISEP 2017) and VII Symposium of Experimental Pathology of the Cell (Brazil, Londrina)

Title: Store operated calcium Entry (SOCE) in skeletal muscle: where?
April 2018 Spring Padua Muscle Days (Padova, Italy)
Title: Lesson from ultrastructure: what images tell if you look closely
May 2019 Gordon Research Conference on Muscle: Excitation-Contraction Coupling (Lucca, Italy).Title: Dysfunctional accumulation of STIM1 and Orai1 in Tubular Aggregates results in impaired Ca2+ entry in aging muscle
March 2020 Padua Days on Myology & Mobilty Medicine (PDM3)
Title: Why EM structure is still important in muscle research
November 2021 General Physiolgy Simposium - November 17th-18th, 2021
Title. Discovery of new intracellular junctions: the Calcium Entry Units (CEUs)

Editorial Board
from 2020
Advisory Board Member of European Journal of Translational Myology (editor in Chief: Prof. Ugo Carraro, Università di Padova)
Review Editor in Frontiers in Physiology Journal - Striated Muscle Physiology

Bibliography Author of 74 Publications (68 original papers and 6 reviews)

Peer reviewed Publications

1 - Fulle, S., F. Protasi, G. Di Tano, T. Pietrangelo, A. Beltramin, S. Boncompagni, L. Vecchiet, and G. Fanò. 2004. The contribution of reactive oxygen species to sarcopenia and muscle ageing. Exp. Gerontol. 39:17-24. REVIEW. I.F. = 2.880
2 - Kern, H., S. Boncompagni, K. Rossini, W. Mayr, G. Fano', M. E. Zanin, M. Podhorska-Okolow, F. Protasi, and Ugo Carraro. 2004. Long-term denervation in humans causes degeneration of both contractile and excitation-contraction coupling apparatus that can be reversed by functional electrical stimulation (FES). A role for myofiber regeneration? J. Neuropath. Exp. Neurol. 63: 919-931. I.F. = 5.037
3 - Boncompagni, S., L. d'Amelio, S. Fulle, G. Fanò, and F. Protasi. 2006. Progressive disorganization of the excitation-contraction coupling apparatus in ageing human skeletal muscle as revealed by electron microscopy: a possible role in the decline of muscle performance. J. Gerontol. Biol. Sci. 61:995-1008. I.F. = 2.861
4 - Ashley, Z., H. Sutherland, H. Lanmuller, M. F. Russold, E. Unger, M. Bijak, W. Mayr, S. Boncompagni, F. Protasi, S. Salmons, J. C. Jarvis. 2007. Atrophy, but not necrosis, in rabbit skeletal muscle denervated for periods up to one year. Am. J. Physiol. Cell Physiol. 292:C440-451. I.F. = 4.230
5 - Paolini, C., M. Quarta, A. Nori, S. Boncompagni, M. Canato, P. Volpe, C. Reggiani, P. D. Allen, and F. Protasi. 2007. Re-organized stores and impaired calcium handling in skeletal muscle of mice lacking calsequestrin-1. J. Physiol. 583:767-784. I.F. = 4.580
6 - Ashley, Z., S. Salmons, S. Boncompagni, F. Protasi, M.F. Russold, H. Lanmuller, W. Mayr, H. Sutherland, and J. C. Jarvis. 2007. Effects of chronic electrical stimulation on long-term denervated muscles of the rabbit hind limb. J. Mus. Res. Cell Motil. 28:203-217. I.F. = 1.731
7 - Angelini, G., S. Boncompagni, P. De Maria, M. De Nardi, A. Fontana, C. Gasbarri, and E.

Menna. Layer-by-Layer deposition of shortened nanotubes or polyethylene glycol-derivatized nanotubes on liposomes: a tool for increasing liposome stability. Carbon. 2007; 45 (13): 2479-2485. *non compare in PUBMED I.F. = 4.260

8 - Zvaritch, E., F. Depreux, N. Kraeva N, R. E. Loy, S. A. Goonasekera, S. Boncompagni, A. Kraev, A. O. Gramolini, R. T. Dirksen, C. Franzini-Armstrong, S. E. Seidman, J. G. Seidman, and D. H. Maclennan. 2007. An Ryr1I4895T mutation abolishes Ca2+ release channel function and delays development in homozygous offspring of a mutant mouse line. Proc. Natl. Acad. Sci. USA. 2007 104:18537-18542. I.F. = 9.598

9 - Boncompagni, S., H. Kern, K. Rossini, W. Mayr, U. Carraro, and F. Protasi. 2007. Structural differentiation of skeletal muscle fibers in absence of innervation in humans. Proc. Natl. Acad. Sci. USA. 104:19339-19344. I.F. = 9.598

10 - Bolaños, P., A. Guillen, H. Rojas, S. Boncompagni, and C. Caputo. 2008. The use of CalciumOrange-5N as a specific marker of mitochondrial Ca2+ in mouse skeletal muscle fibers. Pflugers Archiv. (Eur. J. Physiol). 455:721-731. I.F. = 3.526

11 - Kern, H., C. Hofer, M. Mödlin, W. Mayr, V. Vindigni, S. Zampieri, S. Boncompagni, F. Protasi, and U. Carraro. 2008. Stable muscle atrophy in long-term paraplegics with complete upper motor neuron lesion. Spinal Cord. 46:293-304.
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14.5. Feliciano Protasi

 

2008 **Feliciano Protasi** **2022**

Left Panel: From left to right: Feliciano Protasi, **Simona Boncompagni** and Ugo Carraro in Vienna to collect the first muscle biopsis and to teach friends there how to prepare and fix muscle samples for electron microscopy. Right panel: Simona today.
(Photo credit: Simona Boncompagniand Feliciano Protasi)

Curriculum of FELICIANO PROTASI

Research Unique Identifier:

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Brief Biographical Sketch

Dr. Feliciano Protasi is Full Professor of Physiology and directs a multi- disciplinary research program focused on human diseases of proven genetic origin, mainly supported by Telethon ONLUS (Italy) and by the National Institute of Health (USA).

After graduating in 1991 in Biological Sciences at the University of Perugia, Dr. Protasi moved to the USA to join the laboratory of Prof. Clara Franzini-Armstrong (1993-1997) at the Univ.of Pennsylvania (Philadelphia, PA), where he was involved in projects aiming to understand the differences between skeletal and cardiac excitation-contraction (EC) coupling, the mechanism that activates release of Ca2+ (and hence contraction) in muscle. In the second part of his experience abroad (1997-2002), Dr. Protasi joined the lab. of Prof. Paul D. Allen at the Harvard Medical School (Boston, MA) where he received training in molecular biology and Ca2+ imaging while continuing his studies in the interaction between proteins involved in EC coupling.

Dr. Protasi returned to Italy in 2002 as Associate Professor to join the newly opened institute CeSI (Center for Research of Ageing at Ud'A). He soon established his own lines of research, mainly focused in unraveling the patho-physiological mechanisms underlying ageing and myopathies caused by alterations in Ca^{2+} handling in striated muscles.

Current Position

Since March 2011 Università Degli Studi G. d'Annunzio, Chieti, Italy

School of Sport Medicine - Full Professor of Physiology

Previous Academic Appointments

July 2000 – July 2002	Harvard Medical School, Boston, MA: Instructor
Dic. 2002 – Feb. 2011	University G. d'Annunzio of Chieti Pescara Chieti, Italy -Associate Professor of Physiology

Bibliometric Parameters

n. publications: 107; H index: 44; n. of citations: ~5900 (from ~4100 documents)

Funding History

Sep 2002 – Aug 2005	MIUR Funds (Project Rientro dei Cervelli).Title: The role of Calsequestrin in skeletal EC coupling. Role in the project: Principal Investigator.
Jan 2004 – Jan 2006	TELETHON ONLUS Funds (Project ID: GGP030289) Title: The role of Calsequestrin in excitation-contraction coupling and its possible contribution to skeletal muscle diseases. Role in the project: Principal Investigator.
Feb 2007 – Feb 2009	PRIN – MIUR Funds (Multicentre Project 2006052901_003 coordinated by P. Volpe - Università di Padova) Title: Structural and functional importance of the major Ca2+ binding protein of the sarcoplasmic reticulum (calsequestrin) in the development and full maturation of skeletal muscle fibers. Role in the project: Co-Investigator.
Nov 2008 – Nov 2011	TELETHON ONLUS Funds (Multicentre Project GGP08153 coordinated by F. Protasi) Title: Calsequestrins in calcium homeostasis and potential role in inherited human skeletal muscle diseases. Role in the project: Principal Investigator and Coordinator.
Aug 2010 – June2015	NIH-NIAMS Funds (Multicentre Project RO1 AR059646-01 coordinated by R.T. Dirksen - Univ. of Roch., NY) Title: Molecular Mechanism and functional role of SOCE in skeletal muscle. Role in the project: Co-Investigator.
Feb 2011 – Jan 2016	NIH-NIAMS Funds (Multicentre Project R01 AR053349-06 coordinated by S. H. Hamilton - Baylor College, TX) Title: Basis of muscle dysfunction in Malignant Hyperthermia and Central Core Disease. Role in the project: Co-Investigator
Oct 2011 – Sept 2014	Finanziamento Fondazione TELETHON ONLUS (Multicentre Project GGP11141 coordinated by S. Priori – Univ. of Pavia. Title:

	Mutations of cardiac calsequestrin and cardiac arrhythmias: novel insights on pathogenesis and therapy. Role in the project: Co-Investigator.
Aug 2013 - July 2016	MDA - Muscular Dystrophy Association USA (Multicentre Project 275574 coordinated by R.T. Dirksen - Univ. of Roch., NY) Title: Orai1 as a Therapeutic Target for Central Core Disease. Role in the project: Co-Investigator
Jan 2014 – July. 2016	TELETHON ONLUS Funds (Multicentre Project GGP13213 coordinated by F. Protasi) Project Title: Altered calcium handling in Central Core Disease (CCD) and Malignant Hyperthermia (MH): understand molecular mechanisms and genetic background to develop innovative therapeutic interventions. Role in the project: Principal Investigator and Coordinator
Apr 2016 – Mar 2021	NIH-NIAMS Funds (Multicentre Project RO1 AR059646-06 coordinated by R.T. Dirksen - Univ. of Roch., NY) Title: Molecular Mechanism and Functional Role of SOCE in Skeletal Muscle. Role in the project: Co-Investigator
Feb 2017 – Jan 2020	PRIN – MIUR Funds (Multicentre Project 2015ZZR4W3 coordinated by V. Sorrentino - University of Siena). Title: Novel developments in studies of Ca2+ entry mechanisms: relevance to skeletal muscle function and disease. Role in the project: Co-Investigator
Jan 2020 – Dec 2022	TELETHON ONLUS Funds (Multicentre Project GGP19231 coordinated by F. Protasi) Title: Store-Operated Calcium Entry (SOCE): role in skeletal muscle function and disease. Role in the project: Principal Investigator and Coordinator.

Reviewer:

Scientific Journals	Ageing Cell; American Journal of Physiology; Biophysical Journal; Human Mutation; Journal of Cell Biology; Journal of Histochemistry and Cytochemistry; Pflugers Archives-European Journal of Physiology; Proc. Natl.Acad. Sci. USA; Biochemical Journal; Faseb Journal; Plos ONE; Human Molecular Genetics; Skeletal Muscle; Cell Calcium; Oxid Med Cell Long; Nature Communications; J Muscle Research Cell Motility.
Funding Agencies:	Biotechnology and Biological Sciences Research Council (UK Universities); Science Foundation of Ireland (Ireland); Myotubular Trust Foundation (UK); Agence Nationale de la Recherche (France).
Professional Society Involvement:	Member of Biophysical Society (since1998) Member of Italian Society of Physiology (since 2003) Member of Interuniversitary Institute of Myology (since 2004)

Education

July 1985	Liceo Scientifico Guglielmo Marconi Foligno (PG), Italy - Diploma di

	Maturità Scientifica
July 1991	Università degli Studi di Perugia Perugia, Italy Laurea (Doctorate) in Scienze Biologiche (Magna cum Laude). Thesis: Effects of S-100ab on the binding of Ryanodine to its receptor in the Sarcoplasmic Reticulum (Supervisor: Prof. Giorgio Fanò).
Postdoctoral Training	
July 1991 – May 1993	Università degli Studi di Perugia Perugia, Italy, Institute of Cellular Biology: Tirocinando (Supervisor: Prof. Giorgio Fano’)
June 1993 – Aug. 1997	University of Pennsylvania School of Medicine, Philadelphia, PA Dept. of Cell and Developmental Biology: Post-Doctoral Fellow (Supervisor: Dr. Clara Franzini-Armstrong)
Sep. 1997 – June 2000	Brigham and Women’s Hospital (Harvard Medical School) Boston, MA, Dept. of Anesthesia Research, Post-Doctoral Fellow (Supervisor: Dr. P. D. Allen)
July 2000 – July 2002	Brigham and Women’s Hospital (Harvard Medical School) Boston, MA, Dept. of Anesthesia Research: Research Associate (Supervisor: Dr. P. D. Allen)

Invited Presentations at International Meetings (last 10 years)

September 2010	39th European Muscle Conference (Padova, Italy) - Title: Calcium release units / mitochondria coupling in developing, ageing and diseased skeletal muscle.
September 2011	40th European Muscle Conference (Berlin, Germany) -Title: Calsequestrin-1, a new candidate gene for human muscle disorders.
November 2012	Société Française de Myologie (Grenoble, France) - Title: Core formation in Mouse Models of Malignant Hyperthermia and Central Core Disease.
August 2014	International Biophysics Congress (Brisbane, Australia) - Title: The puzzling phenotype of calsequestrin-1 knockout mice: what have we learned?
October 2014	XI Meeting of the Italian Institute of Myology (Monteriggioni, SI) - Title: Link between malignant hyperthermia (MH) and environmental heat stroke (EHS): just a medical hypothesis?
November 2014	3rd Wiener Muskeltag (Vienna, Austria) - Title: Degeneration of chronically denervated human muscle is reversible.
June 2015	Gordon Research Conference on Muscle EC coupling (Newry, ME). Title: Store-operated Calcium Entry (SOCE) in skeletal muscle: where?
December 2015	AuPS, Australian Physiological Society (Hobart, Tasmania).- Title: Exercise-dependent formation of new SR-TT junctions containing STIM1 and Orai1.
February 2016	Medical School of T. Jefferson University (Philadelphia, PA) - Title: Calcium Entry Units: discovery of new intracellular junctions containing STIM1 and Orai1 in skeletal muscle.

March 2019	Advances in Skeletal Muscle Biology in Health and Disease (Gainsville, FL) Title: Store-Operated Ca2+ Entry (SOCE) in skeletal muscle: where?
October 2019	telethon Scientific Convention (Riva del Garda, TR) - Title: Store-Operated Ca2+ Entry (SOCE): role in Skeletal Muscle function and disease.
April 2022	Medical Academy of Rome (Roma, Italia) New Frontiers in Regenerative Medicine. - Title: Muscle remodeling in response to ageing, inactivity and exercise.
Bibliography:	Author of 120 Publications (12 reviews and 108 original papers)

PubMed 2021-2022

Potenza F, Cufaro MC, Di Biase L, Panella V, Di Campli A, Ruggieri AG, Dufrusine B, Restelli E, Pietrangelo L, Protasi F, Pieragostino D, De Laurenzi V, Federici L, Chiesa R, Sallese M. Proteomic Analysis of Marinesco-Sjogren Syndrome Fibroblasts Indicates Pro-Survival Metabolic Adaptation to SIL1 Loss. Int J Mol Sci. 2021 Nov 18;22(22):12449. doi: 10.3390/ijms222212449. PMID: 34830330; PMCID: PMC8620507.

Di Fonso A, Pietrangelo L, D'Onofrio L, Michelucci A, Boncompagni S, Protasi F. Ageing Causes Ultrastructural Modification to Calcium Release Units and Mitochondria in Cardiomyocytes. Int J Mol Sci. 2021 Aug 4;22(16):8364. doi: 10.3390/ijms22168364. PMID: 34445071; PMCID: PMC8395047.

Michelucci A, Liang C, Protasi F, Dirksen RT. Altered Ca2+ Handling and Oxidative Stress Underlie Mitochondrial Damage and Skeletal Muscle Dysfunction in Aging and Disease. Metabolites. 2021 Jun 28;11(7):424. doi: 10.3390/metabo11070424. PMID: 34203260; PMCID: PMC8304741.

Protasi F, Pietrangelo L, Boncompagni S. Improper Remodeling of Organelles Deputed to Ca2+ Handling and Aerobic ATP Production Underlies Muscle Dysfunction in Ageing. Int J Mol Sci. 2021 Jun 8;22(12):6195. doi: 10.3390/ijms22126195. PMID: 34201319; PMCID: PMC8228829.

Yin L, Zahradnikova A Jr, Rizzetto R, Boncompagni S, Rabesahala de Meritens C, Zhang Y, Joanne P, Marqués-Sulé E, Aguilar-Sánchez Y, Fernández-Tenorio M, Villejoubert O, Li L, Wang YY, Mateo P, Nicolas V, Gerbaud P, Lai FA, Perrier R, Álvarez JL, Niggli E, Valdivia HH, Valdivia CR, Ramos-Franco J, Zorio E, Zissimopoulos S, Protasi F, Benitah JP, Gómez AM. Impaired Binding to Junctophilin-2 and Nanostructural Alteration in CPVT Mutation. Circ Res. 2021 Jul 23;129(3):e35-e52. doi: 10.1161/CIRCRESAHA.121.319094. Epub 2021 Jun 11. PMID: 34111951; PMCID: PMC8320243.

Di Tomo P, Alessio N, Falone S, Pietrangelo L, Lanuti P, Cordone V, Santini SJ, Di Pietrantonio N, Marchisio M, Protasi F, Di Pietro N, Formoso G, Amicarelli F, Galderisi U, Pandolfi A. Endothelial cells from umbilical cord of women affected by gestational diabetes: A suitable in vitro model to study mechanisms of early vascular senescence in diabetes. FASEB J. 2021 Jun;35(6):e21662. doi: 10.1096/fj.202002072RR. PMID: 34046935.

Butera G, Vecellio Reane D, Canato M, Pietrangelo L, Boncompagni S, Protasi F, Rizzuto R, Reggiani C, Raffaello A. Parvalbumin affects skeletal muscle trophism through modulation of mitochondrial calcium uptake. Cell Rep. 2021 May 4;35(5):109087. doi: 10.1016/j.celrep.2021.109087. PMID: 33951435; PMCID: PMC8113653.

Tinari N, Protasi F, Stassi G, Visone R, Di Franco S, Veronese A. A perspective analysis: microRNAs, glucose metabolism, and drug resistance in colon cancer stem cells. Cancer Gene Ther. 2022 Jan;29(1):4-9. doi: 10.1038/s41417-021-00298-5. Epub 2021 Feb 1. PMID: 33526845.

Protasi F, Pietrangelo L, Boncompagni S. Calcium entry units (CEUs): perspectives in skeletal muscle function and disease. J Muscle Res Cell Motil. 2021 Jun;42(2):233-249. doi: 10.1007/s10974-020-09586-3. Epub 2020 Aug 18. PMID: 32812118; PMCID: PMC8332569.

Boncompagni S, Pecorai C, Michelucci A, Pietrangelo L, Protasi F. Long-Term Exercise Reduces Formation of Tubular Aggregates and Promotes Maintenance of Ca2+ Entry Units in Aged Muscle. Front Physiol. 2021 Jan 5;11:601057. doi: 10.3389/fphys.2020.601057. PMID: 33469430; PMCID: PMC7813885.

Protasi F, Pietrangelo L, Boncompagni S. Calcium entry units (CEUs): perspectives in skeletal muscle function and disease. J Muscle Res Cell Motil. 2021 Jun;42(2):233-249. doi: 10.1007/s10974-020-09586-3. Epub 2020 Aug 18. PMID: 32812118; PMCID: PMC8332569

Protasi F, Girolami B, Serano M, Pietrangelo L, Paolini C. Ablation of Calsequestrin-1, Ca2+ unbalance, and susceptibility to heat stroke. Front Physiol. 2022 Oct 12;13:1033300. doi: 10.3389/fphys.2022.1033300. PMID: 36311237; PMCID: PMC9598425.

Vajda M, Oreská Ľ, Černáčková A, Čupka M, Tirpáková V, Cvečka J, Hamar D, Protasi F, Šarabon N, Zampieri S, Löfler S, Kern H, Sedliak M. Aging and Possible Benefits or Negatives of Lifelong Endurance Running: How Master Male Athletes Differ from Young Athletes and Elderly Sedentary? Int J Environ Res Public Health. 2022 Oct 13;19(20):13184. doi: 10.3390/ijerph192013184. PMID: 36293774; PMCID: PMC9602696.

Michelucci A, Pietrangelo L, Rastelli G, Protasi F, Dirksen RT, Boncompagni S. Constitutive assembly of Ca2+ entry units in soleus muscle from calsequestrin knockout mice. J Gen Physiol. 2022 Dec 5;154(12):e202213114. doi: 10.1085/jgp.202213114. Epub 2022 Oct 12. PMID: 36222861; PMCID: PMC9565155.

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Pagotto S, Colorito ML, Nicotra A, Apuzzo T, Tinari N, Protasi F, Stassi G, Visone R, Di Franco S, Veronese A. A perspective analysis: microRNAs, glucose metabolism, and drug

resistance in colon cancer stem cells. Cancer Gene Ther. 2022 Jan;29(1):4-9. doi: 10.1038/s41417-021-00298-5. Epub 2021 Feb 1. PMID: 33526845.

For the complete list of Publications: https://pubmed.ncbi.nlm.nih.gov/?term=protasi+f

CHAPTER 14.Collaborators
14.6. Antonio Musarò

2017 Antonio Musarò 2022
Left Panel: From left: Antonio Musarò, Maurilio Sanpaolesi, Feliciano Protasi. Right panel: Antonio today. (Photo credit: Antonio Musarò)

I went in contact as a junior researcher in the 80' with the Histology Groups of Mario Molinaro, Sergio Adamo and of their students at the Sapienza University of Rome through a series of meetings organize to discuss muscle development and regeneration, among other embryology topics. Thus, when Helmut Kern asked me to extend the expertises of his growing team with other specialists of muscle basic biology it was quite obvious to contact one of those experts who contributed in the characterization of muscle growth factors, now renamed Myokines, that can exert paracrine, autocrine, or endocrine effects. I wrote to Antonio Musarò and he enthusiastically accepted invitation to collaborate with Marco Sandri, Katia Rossini and Sandra Zampieri to extend analyses of the muscle biopsies harvested in Vienna from the patients enrolled in the European Project RISE. Combining molecular, structural, ultrastructural, and functional biomechanical approaches opened the door of top journals that, without or with revision of our submitted typescripts, allowed the Helmut's International Team to reach international recognitions of the effectiveness of home-based Functional Electrical Stimulation of permanent denervated human muscles.
This is why several CV of the collaborators of Helmut Kern in this book list a long series of common publications, including those below of the Curriculum Vitae of Antonio Musarò. Year after year the aims of the Helmut Kern's team changed, but the core-group of experts remained friendly collaborating, not a very usual event in scientific research, where competition is a fundamental value, but also a risk factor for interpersonal conflicts.

Curriculum of Antonio Musarò

POSITION TITLE

Full Professor
Professor of Histology and Embryology
Professor of Biotechnology

Lab web site: http://musarolab-uniroma1.jimdo.com/
Antonio Musarò web site: https://sites.google.com/a/uniroma1.it/antoniomusaro/
Orcid: 0000-0002-2944-9739
Scopus ID: 6602410173

EDUCATION/TRAINING

Sapienza University of Rome, Biological Science: Biology 1991 Muscle Biology

Sapienza University of Rome, Medical School: Ph.D. 1996 Biotechnological Sciences

Harvard University, Boston, USA: Research Fellow in Medicine 1996-2000 Molecular biology of aging

A. Positions and Honours

Positions and Employment

1996–2000 Postdoctoral training– Research fellow, Cardiovascular Research Center; Harvard University.
1999–2007 Assistant professor, Sapienza University of Rome –Medical School.
1999-present Professor of Histology and Embryology, Medical School, Sapienza University of Rome.
Member of the academic committee of PhD program in Morphogenesis & Tissue Engineering
2002 Visiting Professor, Edith Cowan University, Australia.
2003– present Professor of Biotechnology; Sapienza University of Rome.
2003–2014 Adjunct Associate Professor (honorary position), School of Biomedical & Sports Science; Faculty of Computing, Health and Science. Edith Cowan University; Western Australia.
2015-2020 Professor of Histology, nursing school- Cassino-Sapienza University.
2016-2020 Junior Research Fellow, Scuola Superiore di Studi Avanzati Sapienza.
2007- Jan 2017 Associate professor, Medical and Biotechnology School, Sapienza University of Rome.
Feb 2017-present Full professor, Medical and Biotechnology School, Sapienza University of Rome
2018-present Coordinator of the Ph.D. program in Morphogenesis and Tissue Engineering
2018-2022 Director of Master in Stem cells and genome editing
2020-present Senior Research Fellow and Coordinator of Life Science accademic class, Sapienza School for Advanced Studies (SSAS)

Other Experience and Professional Memberships

2001-present Expert reviewer for international scientific journals
2004-present Member of the Society of Cell Biology

2005 Lecturer and Instructor of EMBO Practical Course: From Mice to Cells
2010-present Section Editor for Molecular Myology of European Journal of Translational Myology
2010-2017 Member of the editorial board of World Journal of Biological Chemistry
2010-present Scientific director of “Festa della Scienza”
2010-present Member of the editorial board of Skeletal Muscle
2011-present Member of the editorial board of PlosOne
2015-present Member of the editorial board of Current Genomics
2019-present Academic editor of Cells
2014-present Academic Member of Accademia Medica di Roma
2018-present Board member of Accademia Medica di Roma
2011-2016 Chief of Interuniversity Institute of Myology (IIM)

Honours
2001 Honour for advance in Biological Research
2003 Award for Scientific Communication (Rotary Club)
2006 Award for Scientific Communication, Foglia di Tabacco
2009 Award Sapienza Ricerca for best research 2009 (Sapienza University of Rome)
2014 La Plejade ANCIS International Award 2014 for Scientific Research
2018 Unitel-Puglia (Pergamena D’onore)
2021 Award “Union Invictus” for scientific career (PassioneSport.tv)

B. Major Research Interests and Contributions to Science:
Aging and neuromuscular diseases (ALS, muscular dystrophies); role of stem cells and tissue niche on muscle regeneration.

1. Characterization of the roles of IGF-1 in skeletal muscle homeostasis, regeneration, and diseases
Focusing on specific pathways controlling muscle growth and regeneration, I carefully constructed a program of basic research to characterize the role of specific isoforms of insulin-like growth factor (IGF-1) in the physiopathology of skeletal muscle and in muscle pathologies. We made significant contributions in the field of muscle hypertrophy and muscle aging and contributed to identify signalling pathways involved in skeletal muscle regeneration and diseases. We demonstrated an essential role of inflammatory response in muscle regeneration and repair and characterized the specific role of IGF-1 in the modulation of the tissue niche and on the recruitment of stem cells into the injured muscle.

2. Characterization of the physiopathologic interplay between muscle and nerve
A crucial system severely affected in several neuromuscular diseases, including ALS, is the loss of effective connection between muscle and nerve, leading to a pathological non-communication between the two tissues. In the last 10 years we have made a breakthrough in research into ALS, demonstrating a key role of skeletal muscle in the pathogenesis of ALS. Our research supported the

redefinition of ALS as a “multi-systemic” disease in which alterations in structural, physiological, and metabolic parameters in different cell types (muscle, motorneuron, glia) may act synergistically to exacerbate the disease.

3. Define the signature of hostile microenvironment in muscular dystrophy and sarcopenia.
We study muscle homeostasis and regeneration under normal and pathologic conditions. The main goal of our project is to define the tissue signals and to characterize the molecular mechanisms of muscle wasting. Although considerable information has accumulated regarding the physiopathology of muscle diseases, the associated molecular mechanisms are still poorly understood. We recently provided evidence about specific molecules that modulate the hostile microenvironment and propose alternative pharmacological strategy for treatment muscle diseases.

4. Muscle engineered in vitro model to study muscle homeostasis and differentiation
In our laboratory it has been recently developed a 3-dimensional skeletal muscle construct, called eX-vivo Muscle engineered Tissue, X-MET. X-MET was obtained from murine skeletal muscle primary culture. The isolation from skeletal muscle of heterogeneous cell populations such as satellite cells, fibroblasts and endothelial cells, is a prerequisite of X-MET formation. Since the X-MET mimics the complex morphological properties of skeletal muscle tissue, it may be considered an ideal in vitro model of skeletal muscle, simplifying the study of complex processes such as muscle homeostasis, differentiation and muscle-nerve interplay under physiologic and pathologic conditions such as, muscular dystrophy and ALS.

C. Publications (h index = 44 by Scopus and WoS; 51 by Google Scholar)

1. Germani A., Fusco C., Martinotti S., Musarò A., Molinaro M., Zani BM. TPA-induced differentiation of human rhabdomyosarcoma cells involves dephosphorylation and nuclear accumulation of mutant p53. Biochem Biophys Res Commun. 1994, 202:17-24.
2. Musarò A., Cusella De Angelis MG, Germani A., Ciccarelli C., Molinaro M., Zani BM; Enhanced expression of myogenic regulatory genes in aging skeletal muscle Exp Cell Res. 1995; 221:241-8.
3. Barton-Davis ER, Shoturma DI, Musarò A, Rosenthal N, Sweeney HL. Viral mediated expression of insulin-like growth factor I blocks the aging-related loss of skeletal muscle function. Proc Natl Acad Sci U S A. 1998 95(26):15603-7.
4. Musarò A., Rosenthal N. Maturation of the myogenic program is induced by postmitotic expression of insulin-like growth factor I. Mol Cell Biol. 1999 19:3115-24.
5. Musarò A., Rosenthal N. Transgenic mouse models of muscle aging. Exp Gerontol. 1999; 34(2):147-56. Review.

6. Musarò A, McCullagh KJ, Naya FJ, Olson EN, Rosenthal N. IGF-1 induces skeletal myocyte hypertrophy through calcineurin in association with GATA-2 and NF-ATc1. Nature. 1999; 400: 581-5.
7. Tsao L, Neville G, Musarò A, McCullagh KJ, Rosenthal N. Revisiting calcineurin and human heart failure. Nature Medicine 2000; 6: 2-3.
8. Musarò A, McCullagh K, Paul A, Houghton L, Dobrowolny G, Molinaro M, Barton ER, Sweeney HL, Rosenthal N. Localized Igf-1 transgene expression sustains hypertrophy and regeneration in senescent skeletal muscle. Nature Genetics 2001; 27: 195-200.
9. Barton ER, Morris L., Musaro A., Rosenthal N., and Sweeney H.L. Muscle specific expression of Insulin-like Growth Factor I counters muscle decline in mdx mice. J.Cell Biol. 2002; 157: 137-147.
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11. Musarò A. and Rosenthal N. The role of local Insulin-like Growth Factor-1 isoforms in the pathophysiology of skeletal muscle. Current Genomics 2002; 3: 149-162.
12. Rosenthal, N, Musarò A. Gene therapy for cardiac cachexia? International Journal of Cardiology 2002 85: 185-191
13. Winn N., Paul A., Musarò A., Rosenthal N. Insulin-like Growth Factor isoforms in skeletal muscle aging, regeneration and disease. Cold Spring Harbor Symposia on Quantitative Biology. 2002; LXVII: 507-518.
14. Musarò A, Rosenthal N. Attenuating muscle wasting: cell and gene therapy approaches. Current Genomics 2003; 4:575-585.
15. Bertini E, Biancalana V, Bolino A, Buj Bello A, Clague M, Guicheney P, Jungbluth H, Kress W, Musarò A, Nandurkar H, Pirola L, Romero N, Senderek J, Suter U, Sewry C, Tronchere H, Wallgren-Pettersson C, Wishart MJ, Laporte J. 118th ENMC International Workshop on Advances in Myotubular Myopathy. 26-28 September 2003, Naarden, The Netherlands. (5th Workshop of the International Consortium on Myotubular Myopathy). Neuromuscul Disord. 2004 14:387-96.
16. Musarò A, Giacinti C, Borsellino G, Dobrowolny G, Pelosi L, Cairns L, Ottolenghi S, Bernardi G, Cossu G, Battistini L, Molinaro M, Rosenthal N. Muscle restricted expression of mIGF-1 enhances the recruitment of stem cells during muscle regeneration. Proc Natl Acad Sci U S A 2004; 101: 1206-1210.
17. Dobrowolny G, Giacinti C, Pelosi L, Nicoletti C, Winn N, Barberi L, Molinaro M, Rosenthal N, Musarò A. Muscle expression of a local Igf-1 isoform protects motor neurons in an ALS mouse model. J Cell Biol. 2005; 168:193-9.
18. Scicchitano BM, Spath L, Musarò A, Molinaro M, Rosenthal N, Nervi C, Adamo S. Vasopressin-dependent Myogenic Cell Differentiation Is Mediated by Both Ca2+/Calmodulin-dependent Kinase and Calcineurin Pathways. Mol Biol Cell. 2005; 16:3632-41.
19. Musarò A. Growth factor enhancement of muscle regeneration: a central role of IGF-1. Arch Ital Biol. 2005; 143:243-8
20. Denti MA, Rosa A, D'Antona G, Sthandier O, De Angelis FG, Nicoletti C, Allocca M, Pansarasa O, Parente V, Musarò A, Auricchio A, Bottinelli R, Bozzoni I. Body-

wide gene therapy of Duchenne muscular dystrophy in the mdx mouse model. Proc Natl Acad Sci U S A. 2006; 103:3758-63.

21. Denti MA, Rosa A, D'Antona G, Sthandier O, Angelis FG, Nicoletti C, Allocca M, Pansarasa O, Parente V, Musaro A, Auricchio A, Bottinelli R, Bozzoni I. Chimeric Adeno-Associated Virus/Antisense U1 Small Nuclear RNA Effectively Rescues Dystrophin Synthesis and Muscle Function by Local Treatment of mdx Mice. Human Gene Ther. 2006 17: 1-10.
22. Musarò A., Dobrowolny G., Rosenthal N. The neuroprotective effects of a locally acting IGF-1 isoform. Experimental Gerontology 2007; 42:76-80.
23. Rosenthal N, Santini MP, Musaro A. Growth factor enhancement of cardiac regeneration. Cell Transplant. 2006;15 Suppl 1:S41-5.
24. Musarò A., Rosenthal N. The critical role of Insulin-like Growth Factor-1 isoforms in the physiopathology of skeletal muscle. Current Genomics 2006; 3: 19-32.
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27. Fanzani A, Musarò A, Stoppani E, Giuliani R, Colombo F, Preti A, Marchesini S. Hypertrophy and atrophy inversely regulate Caveolin-3 expression in myoblasts. Biochem Biophys Res Commun. 2007; 357:314-8.
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30. Del Prete Z, Musarò A, Rizzuto E. Measuring Mechanical Properties, Including Isotonic Fatigue, of Fast and Slow MLC/mIgf-1 Transgenic Skeletal Muscle. Ann Biomed Eng. 2008; 36:1281-90.
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CHAPTER 14. Collaborators,
14.7. Nejc Sarabon

2014 Nejc Sarabon 2022

I first met Prof. Nejc Sarabon around 2005 in Vienna, where he was a young collaborator of Helmut Kern to support Christian Hofer and Stefan Lofler in the biomechanical analyses of enrolled patients or young sports science students, usually enrolled in control groups. We also met in Ljubljana for some Meetings that were held there and once or twice a year in the Euganean Hills, when Helmut was spending a few relaxing days at the Continental Hotel, mixing hot pool, pedaling up and down (or around) the Euganean Hills and discussions of research activities. Nejc was often an Invited Speaker at the PADUA MUSCLE DAYS and he is a current Section Editor of EJTM, contributing and attracting interesting typescripts to the JEuropean Journal of Translational Myology.
It has been and continues to be encouraging to follow Nejc's brilliant career as a scientist, an entrepreneur, a teacher and academic administrator.

Curriculum of Nejc Sarabon

Prof. Dr. Nejc Šarabon, born in Ljubljana (Slovenia) is the dean of the Faculty of Health Sciences at the University of Primorska, the head of research at S2P, Science to Practice, and the research group leader in Human Health in the Built Environment at the InnoRenew CoE. He graduated with two undergraduate degrees (physiotherapy and sport pedagogy) and obtained a PhD in sport science.
His scientific focus is motor abilities and neuromuscular control in the context of sport performance and health. He is closely involved in bringing scientific advances to society and has authored over ten patents. He is regularly involved with the Slovenian Olympic committee to translate scientific knowledge into practice. Dr Šarabon prides himself on incorporating theory and practice, research and development, prevention and training, academics, and enterprise to create real health impacts for society. Until 2022 (in the first 20 years of his career) he has published over 800 scientific and professional publications and registered 15 patents.

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CHAPTER 14. Collaborators
14.8. Marcello Cantini

MARCELLO CANTINI UGO CARRARO
Padua, Italy October 30, 2022

Myoblast-Mediated Gene Therapy
Telethon Meeting, Naples, Italy 1993

Marcello Cantini, born in in Capraia e Limite (Florence, Italy) April 8, 1945, graduated in 1971 in "Biological Sciences" of the Padua University Faculty of Sciences, but after an internship in the Institute of General Pathology (belonging to the Faculty of Medicine) were he developedthe beginning of a laboratory of muscle cultures in collaboration with Silvia D'Ancona. His thesis: *In vitro cultures of rhabdomyoblasts of epithelial origin* [In vitro cultures of rhabdomyoblasts of epithelial origin] was signed by Prof. Massimiliano Aloisi. At that time the competition between the Faculties was very strong in Padua so that after Marcello's graduation, Prof. Aloisi not seeing possibilities of inserting another Graduate in Biology in the medical faculty personnel, offered him a position using a scholarship from the Italian CNR at the Center for Biology and Physiopathology of Striated Muscle. Later on, he obtained a permanent position as a CNR Researcher. This ambiguous condition, despite Marcello have always been required to teach to students of the Medical Faculty and he did it with interest and creativity [see also Chapter 2 for the Book: ATLANTE per le "ESERCITAZIONI DI PATOLOGIA GENERALE (Atlas for the practical examinations of pathologic microscopy slides) by Ugo Carraro, Marcello Cantini, Armando Fantinato, Lint, Trieste, Italy], was for him a handicap. On the other hand, his expertises as head of the first laboratory of skeletal muscle cultures developed in Padua, was appreciated by many senior and junior colleagues. His curriculum testifies to this, in particular the wealth of collaborations with senior professors of the Faculty of Medicine of the University of Padua. After Aloisi's retirement as Director of the Institute, the poor availability of lab spaces of the Institute of General Pathology led to a clash between Prof Aloisi's senior students. The crisis found solution with the avalaibility of the younger groups to obtain scientific and organizational independence. It was an opportunity for me to collaborate more closely with Marcello Cantini, obtaining personal fundings from the MIUR (Ministry for Education, University and Research of the Italian Government) and from the newborn Italian TELETHON on common proposals. I am also indebted to the initiatives of Marcello for the apoptosis research line in the musculo skeletal field. Indeed, Marcello collaborated also for three years with Prof. Claudio Franceschi, who had moved to Padua as Professor of Immunology. It was Claudio Franceschi who proposed to evaluate the relevance of apoptotic phenomena in the myocardium and skeletal muscle (see Chapter 8). The analyses of muscles of "nocturnal runner" mice revealed indisputable signs of previously ignored or denied apoptosis in skeletal muscles. I owe even more to Marcello in the field

of muscle regeneration studies, because his observations in cocultures of myoblasts and macrophages are the result once again of a discussion, not in the laboratory in front of tables of results, but of a chat on the train at the return from a Telethon Congress in Naple. I have no difficulty in admitting that mine was more an encouragement to explore the unknown: the role of macrophages not as scavangers of muscle fiber necrosis, but as secretors of activation and differentiation factors of muscle satellite cells, rather than a planned collaboration on my lone proposal. Luckely, a hope did not ended with a bitter disappointment. Some of our works on machrophage-skeletal muscle interactions from 1995-1996 were internationally recognized and are still widely cited in international publications after 25 years.
Suddenly, Marcello decided to retire early both for the conflicts in the Department of Biomedical Sciences, but above all for his further interests. However, I have lost a valuable collaborator, not a friend. We continue to meet from time to time. In particular I would like to remind you that we attended a conference of the Academy of Sciences and Arts of Padua to honor Silvia D'Ancona for her legacy at the Academy after her untimely death. Our talks has been opportunities to rejuvenate of 20 and more years!

Curriculum of Marcello Cantini

Marcello Cantini is born in Capraia e Limite (Florence, Italy) April 8, 1945.

Education/Training
Graduated in Biological Sciences at the University of Padua, Italy: 1971,
Relator: Prof Massimiliano Aloisi
Colture in vitro di rabdomioblastidi origine epiteliale [In vitro cultures of rhabdomyoblasts of epithelial origin]

Positions and Employment

Actual position: Retirement from 2004

1971-1972: Italian CNR Borsa di Studio
1973-1974: Contract with the Microbiology Institute of the University of Trieste, Italy directed by the Prof. Carlo Monti-Bragadin
1974-1984: Senior researcher of the Italian CNR at the Center for Biology and Physiopatholgy of the Skeletal Muscle, Institute of General Pathology of the University of Padua, Italy directed by Prof. Massimiliano Aloisi
1984-2004 University Senior Researcher at the Faculty of Medicine of the University of Padua, Italy

Didactic Activities
1971-2004: Practical exercises of Histopathology for the students of the Faculty of Medicine of the University of Padua, Italy
1994-2004 Docent of General Pathology for the students of the Laurea Breve of Obstrics and Nursing, Faculty of Medicine of the University of Padua, Italy
1996-2002 Docent of General Pathology and Immunology for the School of Specialization in Human Medical Pharmacy, Faculty of Medicine of the

University of Padua, Italy

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Dalla Libera L, Podhorska-Okolow M, Martin B, Massimino ML, Brugnolo R, Cantini M. Smooth muscle myosin light chain kinase is transiently expressed in skeletal muscle during embryogenesis and muscle regeneration both in vivo and in vitro. J Muscle Res Cell Motil. 1997 Jun;18(3):295-303. doi: 10.1023/a:1018618008483. PMID: 9172072.

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Sandri M, Massimino ML, Cantini M, Giurisato E, Sandri C, Arslan P, Carraro U. Dystrophin deficient myotubes undergo apoptosis in mouse primary muscle cell culture after DNA damage. Neurosci Lett. 1998 Aug 14;252(2):123-6. doi: 10.1016/s0304-3940(98)00563-1. PMID: 9756337.

Robert V, De Giorgi F, Massimino ML, Cantini M, Pozzan T. Direct monitoring of the calcium concentration in the sarcoplasmic and endoplasmic reticulum of skeletal muscle myotubes. J Biol Chem. 1998 Nov 13;273(46):30372-8. doi: 10.1074/jbc.273.46.30372. PMID: 9804801.

Nori A, Gola E, Tosato S, Cantini M, Volpe P. Targeting of calsequestrin to sarcoplasmic reticulum after deletions of its acidic carboxy terminus. Am J Physiol. 1999 Nov;277(5):C974-81. doi: 10.1152/ajpcell.1999.277.5.C974. PMID: 10564090.

Nori A, Furlan S, Patiri F, Cantini M, Volpe P. Site-directed mutagenesis and deletion of three phosphorylation sites of calsequestrin of skeletal muscle sarcoplasmic reticulum. Effects on intracellular targeting. Exp Cell Res. 2000 Oct 10;260(1):40-9. doi: 10.1006/excr.2000.4989. PMID: 11010809.

Robert V, Massimino ML, Tosello V, Marsault R, Cantini M, Sorrentino V, Pozzan T. Alteration in calcium handling at the subcellular level in mdx myotubes. J Biol Chem. 2001 Feb 16;276(7):4647-51. doi: 10.1074/jbc.M006337200. Epub 2000 Oct 11. PMID: 11029464.

Sandri M, Sandri C, Brun B, Giurisato E, Cantini M, Rossini K, Destro C, Arslan P, Carraro U. Inhibition of fasL sustains phagocytic cells and delays myogenesis in regenerating muscle fibers. J Leukoc Biol. 2001 Mar;69(3):482-9. PMID: 11261797.

Pampinella F, Pozzobon M, Zanetti E, Gamba PG, McLachlan I, Cantini M, Vitiello L. Gene transfer in skeletal muscle by systemic injection of DODAC lipopolyplexes. Neurol Sci. 2000;21(5 Suppl):S967-9. doi: 10.1007/s100720070011. PMID: 11382197.

Galvagni F, Cantini M, Oliviero S. The utrophin gene is transcriptionally up-regulated in regenerating muscle. J Biol Chem. 2002 May 24;277(21):19106-13. doi: 10.1074/jbc.M109642200. Epub 2002 Mar 1. PMID: 11875058

Cantini M, Giurisato E, Radu C, Tiozzo S, Pampinella F, Senigaglia D, Zaniolo G, Mazzoleni F, Vitiello L. Macrophage-secreted myogenic factors: a promising tool for greatly enhancing the proliferative capacity of myoblasts in vitro and in vivo. Neurol Sci. 2002 Oct;23(4):189-94. doi: 10.1007/s100720200060. PMID: 12536288.

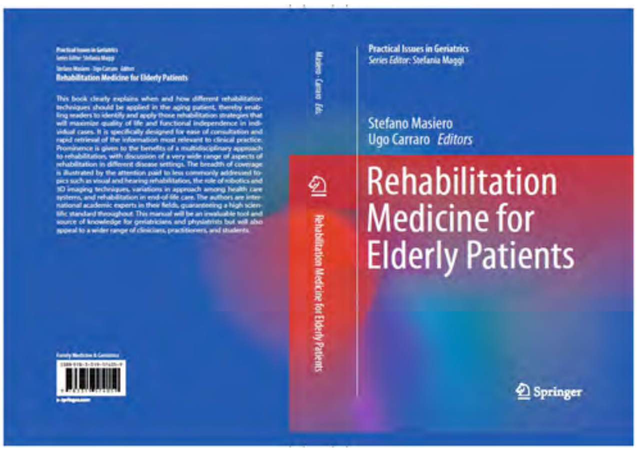
Book

“ATLANTE per le “ESERCITAZIONI DI PATOLOGIA GENERALE (Atlas for the practical examinations of pathologic microscopic slides) by Ugo Carraro, Marcello Cantini, Armando Fantinato, Lint, Trieste, Italy.

CHAPTER 14. Collaborators

14.9. Stefano Masiero

Stefano Masiero 2021



Stefano Masiero, Ugo Carraro (Eds) 2018

I meet for the first time Stefano Masiero during the first Meeting to organize the Italian-RISE 2 Project, a project to find additional candidates for the hb-FES RISE European Project. Several colleagues of the University of Padova, Verona, Udine and Piacenza were present and the program started, founded by the Italian University System.
Stefano was very active and together whe attended Italian Meetings of Rehabilitations, one of them organized in Rome to present evidence of the training protocol for permanently denervated muscles.
Our collaboration strengetened after he become Full Professor in Physical Medicine and Rehabilitation at the University of Padova, Director of the Physical and Rehabilitation Medicine School at the University of Padua and Chair of Rehabilitation Unit at the Padua University-General Hospital.
He accepted also the duties to be a Section Editor of EJTM, submitting and soliciting good typescripts to the journal, recently also with Russian Colleagues.
He asked me to collaborate in the editing of the Nature / Springer Book: Rehabilitation Medicine for Elderly Patients, Stefano Masiero and Ugo Carraro, Eds, 2018, a very successful book with more than 100,000 e-readers and thousands of e-books downloads.
I continue to teach General Pathology, as a voluntary expert, in the Physical and Rehabilitation Medicine School at the University of Padua. It is a pleasure to be in contact with young doctors, female and male and try to attract them to research activities with some success. Indeed, some of them are among the authors of recent publications in EJTM and in more qualified medical journals, as is well documented below in the selected papers listed ta the end of the short CV of Prof. Stefano Masiero.

Curriculum of Stefano Masiero

Present position
Full Professor in Physical Medicine and Rehabilitation at the University of Padova.
Director of the Physical and Rehabilitation Medicine School at the University of Padua.
Chair of Rehabilitation Unit at the Padua University-General Hospital.
Director of Laboratory of Robotic and Bioengineering and Clinical of Movement of Padua University-General Hospital.
Postgraduate Diploma in "Epidemiology and Medical Statistics" at the University of Verona. During his career, he received several academic awards and funding and published over 200 peer reviewed manuscripts including some books of Physical Medicine and Rehabilitation.

Selected Publication - 2021-2022

Carraro U, Piccirillo R, Masiero S, Papathanasiou J, Coplin M. Will there be large or small gifts to PDM3 attendees and EJTM authors in March and June 2023? Eur J Transl Myol. 2022 Sep 16;32(3):10860. doi: 10.4081/ejtm.2022.10860. PMID: 36112069; PMCID: PMC9580539.

Maccarone MC, Magro G, Albertin C, Barbetta G, Barone S, Castaldelli C, Manica P, Marcoli S, Mediati M, Minuto D, Poli P, Sigurtà C, Raffaetà G, Masiero S. Short-time effects of spa rehabilitation on pain, mood and quality of life among patients with degenerative or post-surgery musculoskeletal disorders. Int J Biometeorol. 2022 Oct 8:1–8. doi: 10.1007/s00484-022-02381-4. Epub ahead of print. PMID: 36207541; PMCID: PMC9546417.

Pellegrino G, Pinardi M, Schuler AL, Kobayashi E, Masiero S, Marioni G, di Lazzaro V, Keller F, Arcara G, Piccione F, Di Pino G. Stimulation with acoustic white noise enhances motor excitability and sensorimotor integration. Sci Rep. 2022 Jul 30;12(1):13108. doi: 10.1038/s41598-022-17055-9. PMID: 35907889; PMCID: PMC9338990.

Rubega M, Del Felice A, Masiero S, Carli R, Petrone N, Menegatti E, Tonin L. Neural correlates of user learning during long-term BCI training for the Cybathlon competition. J Neuroeng Rehabil. 2022 Jul 5;19(1):69. doi: 10.1186/s12984-022-01047-x. PMID: 35790978; PMCID: PMC9254548.

Rubega M, Ciringione L, Bertuccelli M, Paramento M, Sparacino G, Vianello A, Masiero S, Vallesi A, Formaggio E, Del Felice A. High-density EEG sleep correlates of cognitive and affective impairment at 12-month follow-up after COVID-19. Clin Neurophysiol. 2022 Aug;140:126-135. doi: 10.1016/j.clinph.2022.05.017. Epub 2022 Jun 15. PMID: 35763985; PMCID: PMC9292469.

Coraci D, Maccarone MC, Ragazzo L, Ronconi G, Masiero S. "Catch me if you can". The contribution of ultrasound to rapidly unveil a nerve lesion. J Clin Neurosci. 2022 May 29:S0967-5868(22)00238-7. doi: 10.1016/j.jocn.2022.05.024. Epub ahead of print. PMID: 35641397.Formaggio E, Bertuccelli M, Rubega M, Di Marco R, Cantele F, Gottardello F, De Giuseppe M, Masiero S. Brain oscillatory activity in adolescent idiopathic scoliosis. Sci Rep. 2022 Oct 14;12(1):17266. doi: 10.1038/s41598-022-19449-1. PMID: 36241666; PMCID: PMC9568615.

Piccione F, Maccarone MC, Cortese AM, Rocca G, Sansubrino U, Piran G, Masiero S.

Rehabilitative management of pelvic fractures: a literature-based update. Eur J Transl Myol. 2021 Sep 17;31(3):9933. doi: 10.4081/ejtm.2021.9933. PMID: 34533018; PMCID: PMC8495369.

Siviero P, Limongi F, Noale M, Della Dora F, Martini A, Castiglione A, Masiero S, Sergi G, Maggi S; Alvise Cornaro Center Study Group. The prevalence of frailty and its associated factors in an Italian institutionalized older population: findings from the cross-sectional Alvise Cornaro Center Study. Aging Clin Exp Res. 2022 May;34(5):1103-1112. doi: 10.1007/s40520-021-02020-9. Epub 2021 Nov 11. PMID: 34762253.

Lebedeva OD, Achilov AA, Mavlyanova ZF, Baranov AV, Achilova SA, Sanina NP, Fesyun AD, Rachin AP, Yakovlev MY, Terentev KV, Reverchuk IV, Velilyaeva AS, Maccarone MC, Masiero S. Is relaxation exercise therapy effective in the management of patients with severe arterial hypertension? Eur J Transl Myol. 2021 Dec 15;31(4):10327. doi: 10.4081/ejtm.2021.10327. PMID: 34911289; PMCID: PMC8758959.

Papathanasiou J, Kashilska Y, Bozov H, Petrov I, Masiero S. The outbreak of the SARS-CoV-2 Omicron variant make imperative the adoption of telerehabilitation in the Bulgarian health care system. Eur J Transl Myol. 2022 Feb 2;32(1):10355. doi: 10.4081/ejtm.2022.10355. PMID: 35107088; PMCID: PMC8992671.

Sweeney HL, Masiero S, Carraro U. The 2022 On-site Padua Days on Muscle and Mobility Medicine hosts the University of Florida Institute of Myology and the Wellstone Center, March 30 - April 3, 2022 at the University of Padua and Thermae of Euganean Hills, Padua, Italy: The collection of abstracts. Eur J Transl Myol. 2022 Mar 10;32(1):10440. doi: 10.4081/ejtm.2022.10440. PMID: 35272451; PMCID: PMC8992680.

Brambullo T, Kohlscheen E, Faccio D, Messana F, Vezzaro R, Pranovi G, Masiero S, Zampieri S, Ravara B, Bassetto F, Vindigni V. A New CT Analysis of Abdominal Wall after DIEP Flap Harvesting. Diagnostics (Basel). 2022 Mar 11;12(3):683. doi: 10.3390/diagnostics12030683. PMID: 35328236; PMCID: PMC8947670.

CHAPTER 14.Collaborators
14.10. Dario Coletti

Dario Coletti 2022

Dario was kind enough to refresh my memory of the first time we met by sending me the following too kind words.

We met for the first time in the year 2001, when I came to Padua to learn the technique of gene delivery by electroporation – an advanced technique to create snap-transgenic organs, of which Ugo Carraro was a pioneer. I was the only one person in the world to come from the USA, where I was doing a postdoc training at that time, to Italy to learn something. That is the other way around as compared to the usual path! I was hosted in Stefano Schiaffino's lab and trained by Marco Sandri. During my stay I met several colleagues of the Department, including Prof. Carraro and his collaborators. It was a typical, raining and cold winter – with those drizzly days so common in Padova and I had not much to do after work. That is likely the reason why the conception of my daughter occurred there in Padua. In Padua not only I learned a lot but also had the opportunity to build a network of collaboration that are still lasting up to date. Our scientific exchanges were strengthened at the first IIM Meeting, that was held in Montegrotto in 2004. Since then, we have started working together on different forms of muscle atrophy, including neurogenic atrophy and cancer cachexia.

Dario being an excellent specialist in cachexia, it was easy to find comon interests and potential collaborations, as it will be clear from his following Curriculum Vitae.

Dario accepted also to be part of the Editorial Board of BAM (it was much earlier than 2010) as Editor of the Section Cellular Myologyand actively supported the journal, not only by submitting interesting papers, but also editing an excellent BAM Special on **Translational Myology: Focus on Cachexia**.

Curriculum of Dario Coletti

First name:	Dario
Last name:	Coletti
Date and place of birth:	1/1/1971, Latina, Italy
Citizenship:	Italian
e-mail address:	dario.coletti@uniroma1.it
web sites:	Sapienza http://dariocoletti.site.uniroma1.it/
blog	http://dariocolettithescientist.blogspot.com/
Foreign languages:	English, French

Current positions:
Associate professor; tenured, from 2013
University of Rome Sapienza
Dept. Biomedical Sciences, Section of Histology & Medical Embryology
Va Scarpa, 16 00161 Rome, Italy
telephone: +39 06 49 76 65 77
fax: +39 06 44 62 854
skype: dario.coletti

Maître de Conférences (Assistant professor); tenured, from 2010 to 2020 (on leave)
Sorbonne University (formerly Pierre et Marie Curie University Paris 06)
Institute of Biology Paris-Seine
B2A Biological Adaptation & Ageing
7, quai Saint Bernard - case 256
Bat A, 6me étage
75252 Paris Cedex 5
France
telephone: +33 (0) 1 44 27 34 75
fax: +33 (0) 1 44 27 21 35

National (Italian) Scientific Habilitation for Full Professor in:
Physical exercise and Sport Sciences
Histology

Biography and Current scientific interests:
Following my education in muscle cell biology and differentiation, I have been working on cachexia and muscle homeostasis since my postdoctoral training in the US. I contributed to highlight the molecular mechanisms underlying impaired muscle regeneration and stem cell function in cachexia, shifting the attention from events intrinsic to the myofiber to potential contributory factors outside the fiber within the muscle microenvironment (stem cell niche) affecting muscle wasting.
The molecular and cellular bases of the pathophysiology and aging of striated muscle tissues, as well as the effects of physical activity on cachexia, are my current major interests. Stem cell biology and tissue engineering aimed to regenerative medicine of the musculo-skeletal system (in particular volumetric muscle loss and the exploitation of biomaterials from decellularized muscles) represent additional research activities.

1. Education:

2000 Doctoral degree in Morphogenetic and Cytological Sciences, Sapienza University of Rome, Italy;
School Director: Prof. Mario Molinaro; Thesis Committee: Prof. Gregorio Siracusa (Univ. of Rome 2 Tor Vergata); Prof. Elio Ziparo (Sapienza Univ. of Rome 1); Prof. Massimo De Felici (Univ. of Rome 2 Tor Vergata)
Thesis title: "Vesicle mediated transport leads to membranes homeostasis, restoring the phospholipid pool consumed in signal transduction"
1995 Degree in Biological Sciences summa cum laude
Thesis title: "Effect of vasopressin on myogenic cells in culture: morphological modifications and membrane traffic", tutor: Prof. Sergio Adamo

2. Professional experience and in-service training:

2010 - to date Research and teaching in Cell biology, Histology, Embryology and Regenerative medicine at Sapienza University, Faculty of Pharmacy and Medicine
2010 - 2020 Maître de Conférences Universitaire (MCU, Assistant Professor) Sorbonne Université; B2A Biological Adaptation & Ageng. Double affiliation on the basis art. 6 Legge Gelmini L240/2010, *nulla osta* Faculty of Medicine on 10/7/13.
2022 Animal experimentation and welfare: the 3 Rs (3d training)
2021 Innovative pedagogies, Tubingen University
2021 Animal experimentation: updates on 2010/63/UE, IZSAM
2018 Animal surgery; UPMC, now Sorbonne, authorized forming agency n° R-75UPMC-F1-08
2016 Managing your group, Dicom for CNRS
2016 Health and safety at work. E-learning Sapienza – 1 credit
2015 Authorization to perform animal research. Ethics, well-being and project design; UPMC, authorized forming agency n° R-75UPMC-F1-08
2014 Evaluating students; D.R.H - Bureau de la Formation des Personnels UPMC
2012 Learn to teach; D.R.H - Bureau de la Formation des Personnels UPMC
2007 - 2010 Responsible for the laboratory of Transmission Electron Microscopy and Calcium Imaging, DAHFOS–Section of Histology & Medical Embryology, University of Rome
2005 - 2010 Research associate at the Department Histology and Medical Embryology, Sapienza University of Rome
2010 Course of Confocal Microscopy with Leica TCS-SP2
2007 Invited researcher at the Myology Group, UMR S 787 Inserm Université Paris 6 PMC, Paris
2004 Contract Professor of Histology and Embryology, School of Dentistry, Sapienza University of Rome
2000 - 2003 Postdoctoral fellow at the Department of Molecular Cell and Developmental Biology, Mount Sinai School of Medicine, New York, NY (laboratory of Prof. David Sassoon)
1999 Visiting scholar at the Division of Reproductive Biology, Department of Gynecology and Obstetrics, Stanford University, Stanford, CA (laboratory of Prof. Marco Conti)

1996-2000 Graduate student in Cell Science and Morhpogenesis, directed by Prof. Mario Molinaro, at the Department of Histology and Medical Embryology, Sapienza University of Rome, Italy

1992 - 1995 Undergraduate student in Biological Sciences at the Department of Histology and Medical Embryology, Sapienza University of Rome, Italy

3. Short/Long invitations for scientific visits to international Institutions or meetings:

2021 Visiting professor at the Faculty of Medicine, USP- University of Sao Paolo (BR)

2021 Invited speaker to the Symposium on Intensive Care Oncology " (Simpósio de medicina intensiva oncológica), Sao Paulo University, BRA 27/11/2021

2021 Invited speaker to the Cancer Cachexia Conference, virtual, 26-29/8/2021

2021 Invited speaker to the 2021 Padua Days on Myology & Mobilty Medicine (PDM3) Thermae of Euganean Hills Padova, (IT) 26-29/5/2021

2018 Invited speaker to the 2018 Spring PaduaMuscleDays, Euganei Hills, Padova (IT), March 15 - 17, 2018

2017 Invited speaker to the 10th Cachexia Conference, Rome, ITA 5-8/12/2017

2017 Invited speaker to the Spring Padua Muscle Days, Montegrotto (Padova), ITA 23-25/3/2017

2016 Invited speaker to the 7th ICNO – CBNC, June 15-18 2016, Sao Paulo, (BR)

2015, 2016 Visiting professor at the Biomedical Sciences Institute, USP-University of Sao Paolo (BR)

2015 Invited speaker to the International Research Group on Biochemistry of Exercise, Sept 7-9 2015, Sao Paulo (BR)

2014 Invited speaker to the 2nd Cancer Cachexia Conference, Montreal, CAN, Sept 28

2014 Seminar at Ohio State University entitled 'Mechanisms underlying cancer cachexia and countermeasures: role of physical activity and the exercise pill' , Columbus (OH), Sept 5

2014 Invited speaker to Spring Padua Muscle Days Montegrotto (Padova), ITA 3-5/4/2014

2013 Invited speaker to the workshop «The tissue factory: from bench to bedside », Fondazione San Raffaele di Ceglie Messapica, Lecce, ITA 16/9/2013

2012 Seminar at the Katholieke Universiteit Leuven entitled 'Myogenic cell deregulation in cachexia', Leuven, BE, 27 Feb

2012 Invited speaker to the congress Healthcare India New Delhi, Delhi Institute of Technology and Jamia Hamdard University India , 20-23/2/2012

4. Teaching activity:

2013 - to date Sapienza Univ. of Rome Faculty of Medicine. Courses for the School of Medicine and the School of Dentistry: Histology and Embryology

2010 - to date UPMC Faculty of Biology - UFR 927. Courses for the School of Life Science: Cell and Developmental Biology, Histology, Methodology in Life Sciences (Licence, LV101, LXM10; Master BMC UE504, MP032); Courses for the School of Medicine: Cell and Developmental Biology, Histology (PAES, UE2)
2003 - 2010 Teaching Assistant for the course of Histology and Embryology, School of Dentistry, Sapienza University of Rome: member of the examination commission, responsible for the laboratory of histology, seminars on specific topics; 10-20 hrs /year
Organization and teaching of the course Biotechnological and Clinical Applications of Histology, School of Dentistry, Sapienza University of Rome; 2-6 hrs /year
2010 Qualification to apply for University positions as maître de conferences in Cell Biology in France
2006 Qualification to teach Sciences in Italy; offer of a tenured position as professor of Sciences in public high school (declined)
2004 Contract Professor of Histology and Embryology, School of Dentistry, Sapienza University of Rome
1997 - 2000 Teaching in the program “Terza Area Disciplinare”, at the Istituto Professionale per l’Industria e l’Artigianato “E. Mattei” in Latina, Italy. Topics: soil, water and air pollution, 75 hrs/year

5. PhD Schools and committees, tutoring and direction of research

2013-to date PhD Committee of the Sapienza University PhD School in Morphogenesis and Tissue Engineering
2010-to date PhD Committee of the Sorbonne University PhD School # 394 in Physiology, Physiopathology and Therapeutics

2022 PhD Jury, Università degli Studi di Torino (various candidates)
2021 Member of the selection committee for the admission to the Ph School in Morphogenesis and Tissue Engineering, at Sapienza University of Rome
2021 PhD external reviewer, University of Victoria, Australia (thesis Dr. Dean Campelj)
2020 PhD Jury, Université de Paris (thesis Dr. Chiara Noviello)
2018 PhD Jury, Université Sorbonne Paris (thesis Dr. Lei Tian)
2017 Member of the selection committee for the admission to the Ph School in Morphogenesis and Tissue Engineering, at Sapienza University of Rome
2017 PhD Jury, University of Calabria (Dr. Chiara Gramaccioni and Dr. Floriana Magaro’)
2016 PhD Jury, Université Paris Descartes (thesis Dr. Aikaterini Papaefthymiou)
2013 PhD Jury, Université Paris Descartes (thesis Dr. Laura Collard)

Juries, tutoring activity and direction of research:

2021 Jury member, Master degree in Medical Biotechnologies

Post-doc
2021 PI, Dr Alessandra Renzini, Sapienza University of Rome

2020-2021 PI di Dr. Medhi Hassani, postdoc a Sorbonne Universrsité
2013-2016 PI, Dr. Nissrine Daou, UPMC Paris 6
2010-2013 PI, Dr. Barbara Perniconi, UPMC Paris 6

PhD
(Graduate students in the doctoral program in PhD School in « Molecula Biology and Medicine", program Morhpogenesis and Tissue Engineering, and undergraduate students in Biology and Biotechnologies)

2018 - 2021 Co- Tutoring of Alexandra Benoni-Sermiovic, PhD student University of Rome
2016 - 2019 Co- Tutoring of Medhi Hassani, PhD st. Univ. of Rome
2013 - 2016 Co- Tutoring of Alexandra Baccam, PhD st. Univ. of Rome
(thereafter, at Aupay, Paris)
2011 - 2012 Co-tutoring of Claudia Serradifalco, PhD st. Univ. of Palermo

Master
2022 Tutoring of Lucas Maughan, Erasmus trainee, MS Pharmacy, Trinity College, Dublin
2020, 2021 Tutoring of Mattia Cossarini, Erasmus trainee, MS Biolgy, UniMORE, Modena
2019 Tutoring of Caterina Gargano, Erasmus trainee, MS Biolgy, UniMORE, Modena
2017 Tutoring of Thomas Costa, MS Biology UPMC Paris 6
2017 Tutoring ofAlexandra-Benoni Sviercovich, Paris Sud, Univ.
2015 - 2016 Tutoring of Medhi Hassani, MS Biology UPMC Paris 6
2012 - 2013 Tutoring of Alexandra Baccam, MS Biology UPMC Paris 6
2011 - 2012 Co-tutoring of Eleonora Rossi, Erasmus trainee, MS Biotech Univ. of Rome

Direction of scientific research
2017 - Scientific director at Sorbonne University, cooperation agreement (Univ. of Palermo, Univ. eCampus, Technischen Universitat Munchen, UPMC, Sapienza Univ. of Rome, and Nanovector srl) for the patent "physiactisome – a new treatment against cachexia".

2011-2013 Scientific director for the project entitled "PRO.ME.T.E.O.: Development of an innovative technological platform and processes optimization for applications in oral and maxillofacial regenerative medicine, hematology, neurology and cardiology", in collaboration with CALABRODENTAL S.r.1., Crotone, Via Enrico Fermi - Loc. Passovecchio, Italy

6. Awards and grants:
2021 SIRIC Groupe Hospitalier AP-PH Sorbonne Université Curamus research grant (€ 30,000), PI
2021 Erasmus+ (€ 2500 travel grant)
2019 Sapienza research +fellowship (€ 10.740 + € 23.787), PI
2017 AFM, Association Francaise contre le Myopathies research grant (€ 15,000), PI
2016 EFEM, European Federation for Experimental Morphology, (€ 1000, travel grant)

2014 IBPS, Institut Biologie Paris Seine (€ 15,000)
2014 NIH, National Institute of Health, co-PI at 5%
2013 ANR, National Agency for the Research research grant (€ 204,000)
2012 AFM, French Association against Myopathies research grant (€ 38,000)
2012 UFI, Italian French University VINCI postdoctoral fellowship, host lab (€ 22,000)
2011 UPMC EMERGENCE 2011 fellowship and research grant# EME1115 (€ 108,000)
2011 Support from the clinic Calabrodental, PROMETEO project (€ 10000 + 55000), PI
2008 Prometeo Network Travel award for the EMBO conference on myogenesis
2007 Grant Ville de Paris – Guest Researchers Office
2006 International Society for Analytical Cytology-ISAC Scholarship
2004 MIUR Rientro Cervelli 2003 fellowship and research grant # 1081, 28/7/'04 (€ 106,000)
2003 NATO Advanced fellowship – no.215.34 (16/1/'02)
1999 Fulbright Commission scholarship
1994 Department of Animal and Human Biology scholarship, Sapienza University of Rome

7. Committees, Scientific societies and Peer reviews:

2014 - to date	Member of the Italian Society of Anatomy and Histology
2012 - to date	Board member of the Indo-Italian Forum on Biomaterials & Tissue Engineering
2020 - to date	Member of the Cancer Cachexia Society
2006 – 2015	Member of the International Society for Advancement of Cytometry (ISAC)
2017	Guest co-Editor for: Frontiers in Muscle Physiology – Striated Muscle Physiology («Myokines, Adipokines, Cytokines in Muscle Pathophysiology»)
2016- to date	Member of the editorial board of Current Updates in Stem Cell Research and Therapy, and of Frontiers in Nutrition – Clinical Nutrition
2014	Guest co-Editor for: Frontiers in Muscle Physiology ("Biomaterials and bioactive molecules to drive differentiation in striated muscle tissue engineering"), BioMed Research International ("Inflammation in Muscle Repair, Aging and Myopathies") and European Journal of Translational Myology (" BAM Specials: The long-term denervated muscle")
2012- to date	**Member of the Editorial Board of Applied Cell Biology,** http://www.tradescienceinc.com/index.php?
2011- to date	**Member of the Editorial Board of World Journal of Stem** Cells (WJSC), http://www.wjgnet.com/

2008- to date **Section editor of** [European Journal of Translational Myology;](#)

European Journal of Translational Myology

Basic Applied Myology - *BAM On-Line*

Editor-in-Chief: Ugo Carraro, Department of Biomedical Sciences, Padua University, Padua, Italy
Phone: +39 338 1575745; E-mail: ugo.carraro@unipd.it

BAM 18 (5) 2008

Table of Content

Translational Myology: Focus on Cachexia

Dario Coletti, Editor

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Editor-in-Chief: Ugo Carraro, University of Padova, Italy - Phone: +39 338 1575745; E-mail: ugo.carraro@unipd.it

Guest-Editor for a focused issue on Cachexia [18(5), 2008](#)
2003- to date Reviewer for: Association Française contre les Myopathies (AFM), BMC Cell Biology, Cancers, FEBS Letters, Frontiers in Muscle Physiology, FWO Belgium, Human

Molecular genetics, Iran National Science Foundation, ISAC, Journal of Cachexia Sarcopenia and Muscle, Journal of Cell Science, Journal of Cellular Physiology, Ligue Contre le Cancer, Mediators of Inflammation, Medical Research Council UK, Mol Biol Cell, Oncotarget, PloS ONE, Science Translational Medicine, Stem Cells, US-Italy Fulbright Commission, World Journal of Stem Cells

Bibliometrics of the scientific production:

D.C. is the (co-)author of a total of about 78 full papers on high quality peer-reviewed journals, including: EMBO J, Genes and Dev, J Cachexia Sarcopenia Muscle, J Clin Invest, Stem Cells, Circ research, Mol Biol Cell etc. In the majority of his papers D.C. holds a relevant position among authors (first, last or corresponding author).

Dario Coletti's Author ID: Scopus 6701742611; ORCID 0000-0001-7373-1953; WOS Researcher ID U-2219-2018. Impact as an author and as a reviewer. Reviews = paper peer-reviewed by DCBibliometric records for the indicated period of time:

Item / Source	PubMed (2007-2022)	ISI Web of Science (1996-2019)	Scopus (2007-2022)	Google Scholar (1996-2022)
Indexed articles	76	64	75	110
Total citations		2052	2121	3241
Total impact factor	326.293			
Average if	5.495			
Hirsch index		25	25	31

Individual Impact Factor 2021: 48/8=6*

* Last Individual IF is calculated as follows: Impact Factor = Cites in 2021 to authored articles published in 2020 and 2019 /Total number of authored articles published in 2020 and 2019

Link to Dario Coletti's appers in PubMed:
https://pubmed.ncbi.nlm.nih.gov/?term=coletti+d+NOT+coletti+dj+NOT+coletti+dab+NOT+coletti+dp+NOT+%22Oral+Surg+Oral+Med%22+NOT+%22Leuk+Res%22+NOT+%22J+Oral+Maxillofac+Surg%22&filter=years.1992-2022&sort=date

PUBLICATIONS

Full Publications

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CHAPTER 14.Collaborators
14.11. Corrado Angelini

Corrado Angelini 2010 **Daniela Tavian and Corrado Angelini 2022**

Corrado Angelini was an Internal Student at the Institute of General Pathology from 1960 (tree years before me), directed by Prof.Massimiliani Aloisi. Despite this, we started our collaboration in 1985 because Corrado choosed to become a Clinical Neurologist specialized in diagnosis and managements of Muscle Genetic Disorders, while I was focused on muscle denevation. We have only one common paper in 2001, but from 1985 we coorganized international conferences, specifically, in 1985 in Abano Terme, Padua, Italy a satellite Meeting of the World Neurology Conference. Then after the *Associatione Italiana di Miologia* was founded in 2000, we coorganized the second Meeting in Thermae of Euganean Hills, Padua, Italy. Corrado from 1991, the year the Basic Applied Myology (BAM) journal foundation, is Editor of the Section: Muscle genetic disorders, recently in collaboration with Daniela Tavian of Milan, Italy. Corrado published only 6 paper after the journal was renamed EJTM in 2010 and was include in PubMed from 2014. Corrado supported me in many, if not all Padua Muscle Days.
I am gratefull that he accepted to be present in this book.

CURRICULUM VITAE

CORRADO ANGELINI, M.D.

Education: MD, University of Padua, Italy

- FLEX Examination, Minneapolis, 1973
- North Dakota State license Examination (No. 3339), 1973
- California State License Examination (No. A-33174), 1978; renewed 2020

Fellowships:

Research Fellowship of Muscular Dystrophy Association, 1972
Senior Fellowship of Muscular Dystrophy Association, 1978

Professional Appointments:

Postgraduate Research Assistant and Associate Mayo Clinic, Rochester, Minnesota, 1970-1972; Resident in Neurology, Mayo Clinic, Rochester, Minnesota, 1973
Assistant Professor of Neurology, University of Padova, Italy, 1973-1978
Associate Professor of Paediatric Neurology, University of Padova, Italy, 1976-1979
Visiting Assistant Professor of Neurology, Reed Neurological Research Center, UCLA Medical School, 1978-1979; University of Colorado Health Sciences Center, Denver 1984
Director Neuromuscular Center, University of Padova, 1980-2011
Associate Professor of Neurology, University of Padova, Italy, 1980-1993
Full Professor of Neurology, University of Padova, Italy, 1994- 2011
Council of PhD Program in Neurosciences, University of Padova, Italy, 2001-present
Director Neurology Residency Program, University of Padova, Italy, 2002- 2011
Consultant Neuromuscular Disorders,IRCCS S.Camillo Hospital.,Venice,Italy,2010-present
Senior Researcher University of Padova,2013-present

Honors:

Awarded MDA Senior Fellowship, 1978
Lion Club Milano Host award for Neurological Sciences,1981
Grands Prix Newropeans 2004
Gaetano Conte's Prize, Kusadasi 2005

Memberships in Scholarly Societies:

Associate member, American Academy of Neurology, 1976
European Academy of Neurology and ANA.
Founding Member of WMS
On Editorial Board of Neuromuscular Disorders, Neurological Sciences, Neurology, Acta Myologica, European Journal Translational Myology , Therapeutic Advances Neurological Disorders.
Editor-in Chief :Muscles.

Major Research Interests:

Primary biochemical defects in inherited neuromuscular diseases, clinical trials in muscular dystrophies and DMD, ALS, myasthenia gravis, congenital muscle diseases, carnitine and lipid metabolism. Glycogenoses

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MANUSCRIPTS PUBLISHED (942)
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CHAPTER 14.Collaborators
14.12. Giovanna Albertin


From left to right: Helmut Kern, Ugo Carraro, Feliciano Protasi, Sandra Zampieri and Giovanna Albertin, the key researcher for the hbFES skin project.

2018 Giovanna Albertin 2022

As described below in her CV, I meet Giovanna at the Human Anatomy Section of the Department of Neuroscience of the University of Padua, Italy. I don't remember why I was there, but it was the beginning of a research collaboration based on a research tool that I find essential in any muscle study: use results collected by light microscopy. I know people can't believe that, in time of MiRNA and LNC-RNA research, such an approch could be relevant, but it is and could be enough to publish in decent scientific journals reports based only on those results. In her below CV, Giovanna will explain much better than me why it is possible.
Thanks Giovanna for accepting invitation to join us and even more for the final words of your CV introduction!

CURRICULUM VITAE of Giovanna Albertin - Short

Born in Abano Terme, Padua, Itay. the April 25, 1965, I graduated in 1991 in Biological Sciences from the Faculty of Sciences MM.FF.NN. of the University of Padua. I did my internship at the Medical Clinic I of Prof. Pessina and through the collaboration with the microbiology institute of Prof. G. Palù, I did my PhD in Microbiological Sciences at the University of Genoa from 1995 to 1999. Since 2000 I have been admitted to the Section of Human Anatomy of the Department of Neuroscience of the University of Padua, first as a researcher and from 2017 as associate professor. I was interested in the pathophysiology of human peptides of the adrenal gland, prostate, thymus; analysis of angiogenic, proliferative and antiapoptotic activity of various factors such as endothelin, adrenomedullin, ghrelin and urotensin; toxicity and biocompatibility of gold nanoparticles.
In 2007, 2008, and 2019, I was a guest at Prof. Michael Underhill's Skeletogenesis and Organogenesis lab at the University of British Columbia (UBC) in Vancouver, Canada. During those periods I followed the activity of developing expression vectors including

mainly vectors related to the understanding of the functions of the human genes in the early stages of embryonic development. The research acitivity in these last years is dedicated to collaborate with the group of Professor Carla Stecco in the biomolecular and histological analysis of peptides in the fascial system (endocannabinoid receptors and hormonal factors) with the objectives of understand the structure of the fascial tissue and its role in anti-inflammatory and immunomodulatory processes.

With Ugo Carraro

I met prof. Ugo Carraro as a colleague in the Faculty of Medicine when he was still officially a professor at the University of Padua, but we became as research colleagues only a few years ago. It was one morning in January 2016 when I met prof. Ugo Carraro in the corridors of the Institute of Human Anatomy. I had had the opportunity to read his full-body in-bed gym gymnastics program and since my dad had to be stimulated to do a certain physical activity to get him back in shape, I immediately found the availability to give me a hand and the desire to give himself do and test his studies and projects. We did some gymnastics sessions with my dad that I then tried to carry on for a while, and I must say that they were very stimulating. At the same time Ugo wanted to involve me in a research that I had understood was in his mind for a long time. It concerned the skin biopsies included in formalin, they were parts of the muscle biopsies of the RISE, European project, of prof. Helmut Kern with whom Ugo was been collaborating for several years. The skin biopsies collected between 2002 and 2007 had been put aside because they were not involved in the research of that European project. With Ugo, and the collaboration of technicians of the Institute of Human Anatomy, we analyzed these biopsies in their epidermal thickness, dermal papillae and Langerhans cells and the results were published on some paper, highlighting that regular and continuous h-bFES led to an improvement in the thickening of the epidermis in subjects suffering from SCI for different time from neurological injury. The epidermis improved in the formations of the dermal papillae and the Langerhans cells did not differ significantly in number between before and after the h-bFES such as to allow us to say that the electrostimulation did not involve a statistically significant activation of the Langerhans cell, seen as "sentinel cell of the skin's immune system". With Ugo and his friend Paolo Gava we also started a collaboration to analyze data from marathon runners and put them in relation to physiological decay, and for this reason I hope to continue to collaborate with Ugo despite his angular and not very patient character, small defects that take second place if one observes his passion for research on different fronts.

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CHAPTER 14.Collaborators
14.13. Gianluca Rigatelli

Curriculum of Dr. Gianluca Rigatelli

Name, title and age: Dr Gianluca Rigatelli, MD, PhD, EBIR, born in Embu Kieny, Kenya, 10.12.1971, 51 year-old

Education:

- First class medical graduation 07.22.1996 c/o University of Padua School of Medicine and Surgery
- First class Board certification in Cardiology 12.11.2000 c/o University of Padua School of Medicine and Surgery
- Ph Degree, Verona University School of Medicine, 6.06.2010
- European Board certified in Interventional Radiology, EBIR 2010

Postgraduation:

- Residency, Division of Cardiology, Legnago General Hospital, Verona, Italy 1996-1998
- Fellowship, Catheterization Lab, Department of Cardiovascular Disease, Cittadella General Hospital, Padua, Italy, 1998-2000
- visiting physician c/o L'Unitè de Cardiologie Interventionelle , Polinique Les Nancy, Nancy, France, July 2000
- visiting physician, Interventional Lab, Cardiovascular Center, Sankt Katherinen Hospital, Frankfurth, Germany, November 2004
- visiting physician, Texas Heart Institute, Houston, Texas, USA November 2003, December 2004

Hospital appointments:

- Staff physician, Interventional Cardiology Unit , Department of Cardiovascular Disease, Cittadella General Hospital, Padua, Italy, 2000 to 2003

- Staff physician, Interventional Cardiology Unit, Division of Cardiology, Rovigo General Hospital, Rovigo Italy, 2003 to 2005
- Director, PFO-mediated syndrome management program 2004 to 2021
- Director, adult congenital heart disease diagnosis and treatment program, 2005 to 2021
- Director, Interventional Cardiology, Aulss6 Ospedali Riuniti Padova Sud, Monselice, Padova, Italy

Academic appointments:

- Professor on contract, Radiological Sciences, Padua University School of Medicine, Padua, Italy since 2012
- Professor on contract, Nursing Sciences, Padua University School of Medicine, Padua, Italy since 2012
- Visiting Professor of Cardiology, Thong Nhat Hospital, Ho Chi Minh City, Vietnam, since 2006
- Visiting Professor of Medicine, Tan Tao University, Long An, Ho Chi Minh City, September 2013
- Honorary Professor of Medicine, 10th People Hospital, Tonji University medical School, Shangai, China 2014
- ASN Cardiovascular Disease, Associate Professorship April 30th 2019

Research

- H-index 30, i-10 134, ranking in Italian Best Scientists List
- 500 articles, 460 indexed in Pub Med
- 150 abstracts in International Meetings
- More than 32 book chapters
- More than 150 lectures in national and international congresses
- Field of interest: Left main and bifurcation physiology and interventions, Coronary artery anomalies diagnosis and endovascular therapy Adult congenital heart disease catheter –based interventions, , Peripheral Vascular disease endovascular therapy
- **Scientific activity**
- Reviewer for: CHEST, International Journal of Cardiology, Cardiovascular Interventional Radiology, European Journal of CardioThoracic Surgery, Italian Heart Journal, Circulation, Heart and Vessel, American Journal of Hypertension, Journal of Cardiovascular Medicine, European Heart Journal, Expert Review of Cardiovascular Therapy, Lancet , Annals of Internal Medicine, American Journal of Cardiology, JACC interv, Cardiology in the Young, J Pediatric, Circulation, Catheterization and Cardiovascular Interventions
- Editorial board: Journal of Geriatric Cardiology, American Journal Cardiovascular Disease, Journal of Translational Internal Medicine

Awards:

- Who'sWho Marquis in Medicine and Health, Science and Engineering, in the World 2003-2004 and 2004-2005, 2006-2007, 2007-2008, 2009-2010, 2011-12
- National research awards

Professional:

- fellow of Associazione Nazionale Medici Cardiologi Ospedalieri-FANMCO
- fellow of American College of Cardiology-FACC
- fellow of European Society of Cardiology-FESC
- fellow of Society for Cardiovascular Angiography and Interventions-FSCAI
- fellow of Cardiovascular Interventional Radiological Society of Europe-FCIRSE
- fellow of American College of Physician-FACP
- fellow of European Society of Pediatric Cardiology
- fellow of GISE-Italian Society of Invasive Cardiology

Selected papers from PubMed

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dynamic demonstration. Rev Cardiovasc Med. 2022 Feb 12;23(2):58. doi: 10.31083/j.rcm2302058. PMID: 35229549

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Rigatelli G, Barbiero M, Rigatelli G, Cotocni A, Riccardi R, Cobelli F, Carraro U. Cardiocirculatory bio-assist: is it time to reconsider demand dynamic cardiomyoplasty? Review and future perspectives. ASAIO J. 2003 Jan-Feb;49(1):24-9. doi: 10.1097/00002480-200301000-00004. PMID: 12558303 Review.

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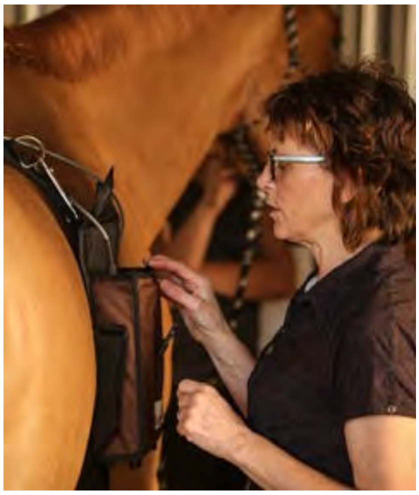
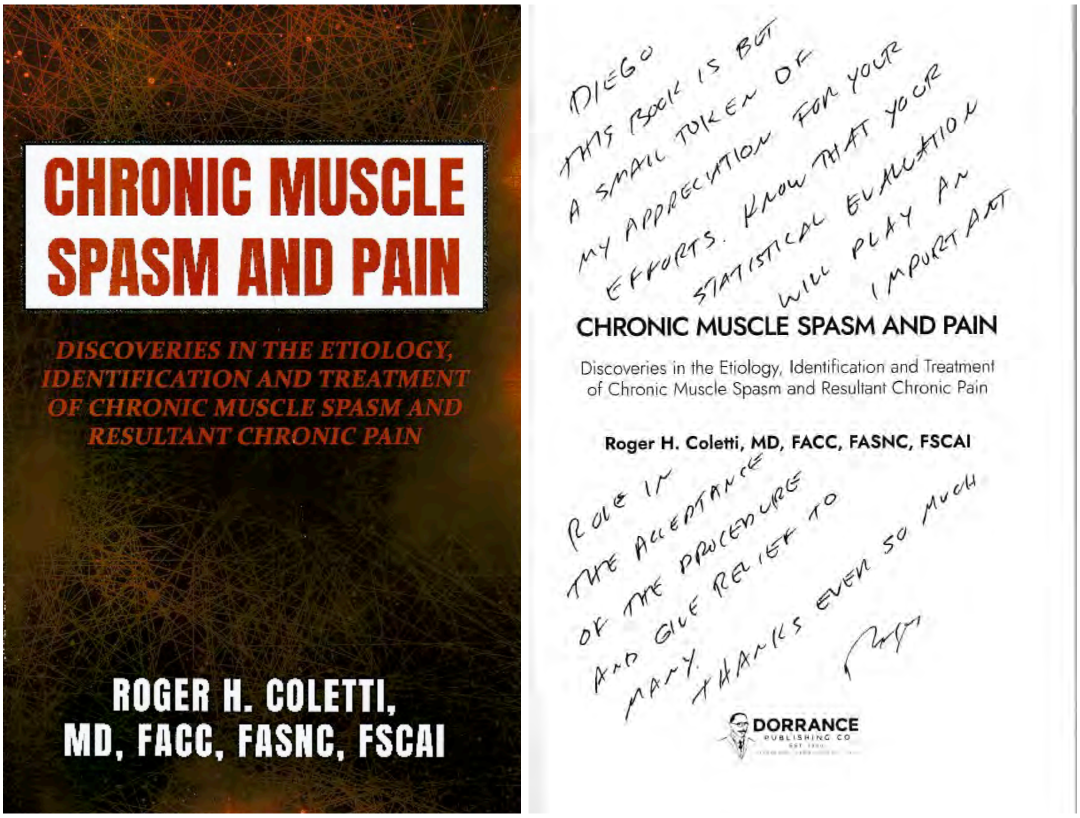
CHAPTER 14. Collaborators
14.14. Sheila Schills



Padua Muscle Days 2015 Sheila Schils 2021

Sheila Schills 2022

Sheila Schils is another contributor who found me through my PubMed publications. She wrote and then called me from the States to get help studying a series of histological muscle biopsies taken from horses that she had treated by electrical stimulation for muscle spasms. The conclusion of the veterinary pathologist who performed the muscle histology was that electrical stimulation was damaging the muscles on the basis of a single horse, which showed clear signs of diffuse atrophy of muscle fibers, despite being one case among the many analysed. By repeating the quantitative analyzes with Barbara Ravara in Padua, we demonstrated that not only did FES not damage all musculature in the treated horses, but that all the animals showed statistically clear increase in content of mitochondria in the treated muscle fibres, a proof of the positive effect of the induced contractions by electrical stimulation. Our publications on Sheila's horses recently attracted the interest of a colleague, Roger H. Coletti, who is treating human cases of low back pain by chemodenervation of spastic muscles. In his opinion, Schils's horses did not have denervated, but "hibernated" myofibers, such as those often present in ischemic myocardium. By the way, Roger H. Coletti is a retired interventional cardiologist who



Roger H Coletti. Chronic Muscle Spasm and Pain: Discoveries in the Etiology, Identification and Treatment of Chronic Muscle Spasm and Resultant Chronic Pain.

DORRANCE PUBLISHING COMPANY - Printed and eBook.
585 Alpha Drive Suite 103 Pittsburgh 15238 PA - Phone 800-788-7654.

began treating himself for the painful consequences of having "sleeping" muscles on his back. I recommend that Sheila and any other specialist treating animal or human cases of muscle spasms read Roger's recently published book. For preliminary information see: Coletti RH. The ischemic model of chronic muscle spasm and pain. Eur J Transl Myol. 2022 Jan 18;32(1):10323. doi: 10.4081/ejtm.2022.10323. PMID: 35044134; PMCID: PMC8992665.

But I must conclude this lengthy introduction by thanking Sheila for her overwhelming confidence in my experience and warm memories. Not sure I deserve them!

I am honored to be able to express my appreciation and gratitude to Dr. Ugo Carraro for his encouragement and support of my work over the years. In 2010 I was working on a research project evaluating the histology of muscle cells pre- and post-FES therapy on horses. I had reviewed the research onFunctional Electrical Stimulation (FES) and muscle cell histology and the researcher's name that continued to come up time and time again was Dr. Ugo Carraro. I took a chance and contacted Dr. Carraro and he immediately responded that he would be willing to offer any help necessary in the evaluation of our samples. I then boarded a plane and we met in Padua, Italy to discuss the project. From that point on Dr. Carraro has invited me to participate in research, present at conferences and encouraged me to write about my work with FES and horses. His ability to connect people together to perform collaborative work as well as to support and encourage novel *ideas is what brings him to the top of his profession.*

CURRICULUM VITAE

SHEILA SCHILS, Ph.D.
N8139 900th Street
River Falls, WI 54022
sbschils@EquiNew.com
715 222-8279

RELEVANT WORK EXPERIENCE

2002 – Present EquiNew, LLC - Owner

1997-2001 Professor, University of Wisconsin-River Falls

◊ 75% Teaching, Animal and Food Science Department

◊ 25% University Faculty Mentoring Program Coordinator

1993, Associate Professor, University of Wisconsin-River Falls

◊ 75% Teaching, Animal and Food Science Department

◊ 25% University Faculty Mentoring Program Coordinator

1990, Sabbatical, Agricultural University, Uppsala, Sweden

◊ 100% Research

1989, Assistant Professor, University of Wisconsin-River Falls

◊ 100% Teaching, Animal and Food Science Department

1979, Instructor, University of Wisconsin-River Falls

◊ 100% Teaching, Animal and Food Science Department

EDUCATION

1987-1990	University of Minnesota Minneapolis, Minnesota	Doctor of Philosophy in Kinesiology/Biomechanics
1982-1988	University of Minnesota St. Paul, Minnesota	Master of Science in Animal Sc./Equine Nutrition
1976-1979	William Woods College Fulton, Missouri	Bachelor of Science in Equine Studies

UNIVERSITY CURRICULUM DESIGN AND COURSES TAUGHT

I was responsible for the development and implementation of a new curriculum of courses during my tenure at UWRF. The equine program grew to the extent that it had the highest student enrollment of the three species emphases.

The equine-based course work I was responsible for covered the major areas of nutrition, feeds, ration balancing, diseases and vaccinations, hoof care, stabling, equitation theory and training, equine conformation and selection. In addition, I taught an interdisciplinary course in biomechanics and kinesiology as well as laboratory classes in equine handling techniques, equitation and training.

ADDITIONAL PROFESSIONAL CONTRIBUTIONS IN EQUINE SCIENCE

I have won several national and regional titles in dressage and jumping since 1982. The most recent competitions were the 2013 and 2016 United States Dressage Federation Finals for the Top 20 horses in the US at Intermediare 1 and the 2000 American Horse Shows Association National Dressage Championships at Prix St. George, Third Place.

SELECTED PROFESSIONAL PAPERS AND PRESENTATIONS

Schils S, Ober T. Functional Electrical Stimulation (FES) in the diagnosis and treatment of musculoskeletal and neuromuscular control abnormalities in horses – Selected case studies. J of Equine Vet Sc 2022, 117:1-30.

Taylor MJ, Schils S, Ruys AJ. Home FES: An exploratory review. Eur J Transl Myol 2019, 29(4):283-292.

Schils S, Ober TR, Butcher MT. Review of the biomechanics of injury in the equine athlete: From research to clinical practice. American Association of Equine Practitioners (AAEP) Proceedings, November, Orlando, Florida 65:273-280, 2019.

Schils S. Exercise and treatment strategies focusing on the stifle in the horse. 14th Annual Promoting Excellence Symposium, FAEP Proceedings October 18-21, Naples, Florida, 2018.

Schils S. Video gait analysis of the horse: common problems of the stifle. 14th Annual Promoting Excellence Symposium, FAEP Proceedings October 18-21, Naples, Florida, 2018.

Isbell D, Schils S, Oakley S. Functional Electrical Stimulation (FES) and the effect on Equine Multifidi Asymmetry. American Association of Equine Practitioners (AAEP) Proceedings, San Antonio, Texas,

November, 2017.

Carraro U, Kern, H, Gava, P, et al. Recovery from muscle weakness by exercise and FES: lessons from Masters, active or sedentary seniors and SCI patients. Aging Clin Exp Res 2017, 29L579-590.

Schils S. The development of training programs to reduce injury and specific rehabilitation protocols when injury occurs.13th Annual Promoting Excellence Symposium, FAEP Proceedings October 17-22, Naples, Florida, 2017.

Schils S. Biomechanical basis of rehabilitation protocol development and timelines. 12th Annual Promoting Excellence Symposium, FAEP Proceedings October 15-18, San Juan, Puerto Rico, 2016.

Schils S, Hofer C. Lofler S, et al. Functional Electrical Stimulation (FES) for mobility in human spinal cord injury and in muscle spasm and atrophy rehabilitation in horses 12th Vienna International Workshop on FES. September 7-9, Vienna, Austria, 2016.

Schils S. Preventing Injury through balanced movement- how can biomechanics help. What is whole horse rehabilitation? Saratoga Horse Symposium. Cornell University Extension. April 9, 2016.

Schils S, Butcher MT. Biomechanics of Injury and Healing. 11th Annual Promoting

Excellence Symposium, FAEP Proceedings, October 15-18, Naples, Florida, 2015.

Schils S, Carraro U, Turner T, Ravara B, Gobbo V, Kern H, Gelbmann L, Pribyl J. Functional Electrical Stimulation (FES) for equine muscle hypertonicity: histological changes in mitochondrial density and distribution. J of Equine Vet Sc 2015, 35:907-916.

Schils SJ, Isbell D.The whole horse approach to equine physical rehabilitation: The biomechanical view. Proceedings of the ENUTRACO, Bengen, Germany Wageningen Academic Publishers, Wageningen, Netherlands, 2015.

Ravara B, Gobbo V, Carraro U, Gelbmann L, Pribyl J, Schils S. Functional electrical stimulation as a safe and effective treatment for equine epaxial muscle spasms: clinical evaluations and histochemical morphometry of mitochondria in muscle biopsies. Eur J Transl Myol/ Basic Appl Myol 2015, 25:109-20.

Ravara B, Gobbo V, Carraro U, Gelbmann L, Pribyl J, Schils S. Mitochondrial density and distribution by histochemical approaches distinguish muscle fiber types and support clinical improvements due to FES as a treatment of equine epaxial muscle spasms. Eur J Transl Myol/Basic Appl Myol 2015; 25 (3): 145-182 (149) CIR-Myo News: Abstracts of the 2015 Spring Padua Muscle Days, Terme Euganee Padua (Italy), March 12-14, 2015.

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Schils SJ, Turner TA. Functional electrical stimulation for equine epaxial muscle spasms: retrospective study of 241 clinical cases. Comp Ex Phys 2014, 10(2):89-97.

Schils S, Lacher E, Thaler R, Oakley S. Novel applications of functional electrical stimulation in equine rehabilitation. 8th International Symposium on Veterinary Rehabilitation/Physical Therapy and Sports Medicine. Oregon State University, Corvallis, Oregon, August 4-8, 2014.

Schils S. The Science Behind the Development of Rehabilitation Protocols. Proceedings of the Florida Association of Equine Practitioners (FAEP) 10th Annual Promoting Excellence Symposium in the Southeast, Hilton Head, South Carolina, October 9-12, 2014.

Schils, S. Applying functional electrical stimulation (FES) in the rehabilitation of muscle and tendons in horses. Proceedings of the ENUTRACO, Bonn, Germany Wageningen Academic Publishers, Wageningen, Netherlands, 2013.

Schils S. Rehabilitation Science. Proceeding of the 9th Annual Promoting Excellence Symposium in the Southeast, Boca Raton, Florida, October 17-20, 2013.

Schils SJ. Functional electrical stimulation for muscle wasting in equine rehabilitation. 7th International Symposium on Veterinary Rehabilitation and Physical Therapy (ISVRPT) Proceedings. Vienna, Austria, August 15-18, p 97, 2012.

Schils S. Functional electrical stimulation (FES) in equine rehabilitation: Initial observations. Proceedings Conference of the International Functional Electrical Stimulation Society, Smart Machines- Neural Evolution, Banff, AB, Canada, September, 2012.

Schils SJ. Boots on the ground: Rehabilitation protocols utilized by rehabilitation facilities. Proceeding of the 8th Annual Promoting Excellence Symposium in the Southeast, Naples, Florida, October 11-14, 2012.

Schils SJ. Functional electrical stimulation (FES) for treatment of muscle spasticity and atrophy in horses. Proceedings 2nd Annual Conference of the International Functional Electrical Stimulation Society (UK and Ireland Chapter), University College Dublin, March, 2011.

Schils SJ. Early mobilization for acute and chronic injuries. Proceedings 7th Annual Promoting Excellence Symposium in the Southeast, Amelia Island, Florida September 29-October 2, 2011.

Schils SJ. Functional electrical stimulation (FES) for use in equine medicine. Proceedings Conference on Equine Sports Medicine and Science, Sigtuna, Sweden, June 28-30, Wageningen Academic Publishers, Wageningen, Netherlands, 103-108, 2010.

Schils SJ. Turner TA. Review of early mobilization of muscle, tendon, and ligament after injury in equine rehabilitation. Proceedings 56th Annual AAEP Convention, Baltimore, December 4-8, p 374-380, 2010

Schils SJ. How to use functional electrical stimulation (FES) for rehabilitation after stem cell therapy . Proceedings 6th Annual Promoting Excellence Symposium in the Southeast, Orlando, FL, November 4-6, 2010.

Schils SJ. Review of electrotherapy devices for use in veterinary medicine. Proceedings 55th Annual AAEP Convention, Las Vegas, December 5-9, p 68-73, 2009.

Schils SJ, Greer, NL,. Stoner, LJ, Kobluk, CN. Kinematic analysis of the equestrian-Walk, posting trot and sitting trot. Human Movement Science 1993, 12:693-712.

Schils SJ. "Biomechanics of the Equine and Equestrian". Proceedings of the National Association of Animal Scientists Annual Meeting. Minneapolis, Minnesota, 1994.

Schils SJ. Kinematics of the equestrian: How the rider influences movement of the horse. Proceedings of the 13th Annual Kansas State University Horse Extension Conference. March 4, Manhattan, Kansas, 1995.

Schils SJ., Greer, NL. Stoner, LJ, Kobluk, CN. "Relative and Absolute Angles of the Equestrian: Walk, Posting Trot and Sitting Trot. Proceedings from the 16th Meeting of the Association for Equine Sports Medicine, March 15-18, San Antonio, Texas, 1997.

Schils SJ. Kinematic analysis of the equestrian. Published doctoral dissertation, University of Minnesota, Minneapolis. 1990.

Schils SJ, Jordan RM. Nutritional practices and philosophies of racehorse trainers. In Proceedings of the Eleventh Equine Nutrition and Physiology Society. Oklahoma State University, Oklahoma City, 1989.

Schils SJ. Nutritional practices of racehorse trainers. Master's thesis, University of Minnesota. 1988.

CHAPTER 14. Collaborators
14.15.
Mauro Salvatore Alessandro Alaibac

Mauro Salvatore Alessandro Alaibac 2022

To plan common research projects, I met Prof. Alaibac of the Dermatology Section of the Medicine Department of the University of Padua twice, the first time perhaps in 2010 because as a clinical dermatologist at the head of a skin immunohistology laboratory, he was a potential collaborator to analyze skin biopsies that Dr. Kern had collected in Vienna to biopsy thigh muscles. After some discussions, the project did not move from planning to implementation. Only after Giovanna Albertin, from the Human Anatomy Section of the Department of Neuroscience at the University of Padua agreed in 2018 to study those biopsies, did the research team include Mauro. He has provided some antibodies for immunohistochemistry, particularly those that can trace immunocompetent cells, but even more his expertise in skin diseases. Fortunately, the hypothesis that long-term denervation of human legs affects not only muscles but also local skin was confirmed by quantifying skin thickness, which decreased almost linearly with years of denervation, but was fully recovered after two years. of hbFES (see details in Chapter 7). Furthermore, Mauro and Giovanna have provided statistically proven evidence that long-term denervation due to lesion of the lower motor neuron, but not of the central motor neuron, induces an early decrease in Langerhns cells and therefore in skin immunoprotection lasting at least 10 years. All together, these are original clinically relevant findings suggesting possible immune repression in the epidermis of permanently denervated patients. Mauro was the key author to convince the referees of the clinical journals of relevance of our observations to explain on a cellular basis the well-known fragility of the skin of paraplegic patients, adding a new mechanism to the tissue ischemia due to the body weight of seated patients many times of the day in their wheelchairs. It should be noted that the skin biopsies were taken from the anterior part of the thigh which is not subject to pressure. In conclusion, without the expert help of Prof. Mauro Alaibac, Director of the Dermatology Section of the Medicine Department of the University of Padua, nothing would have been published.

Curriculum Mauro Salvatore Alessandro Alaibac

Personal Information

Mauro Salvatore Alessandro Alaibac
Date of birth 2-4-1961

Work experience
1986-1989 Resident at the School of Dermatology University of Florence.
1989-1995 Research fellow-PhD student Unit of Dermatology, Royal Postgraduate Medical School, University of London.
1995-1998 Research Fellow Unit of Experimental Oncology, National Institute for Cancer Research, Bari 1998-2005 Research Fellow Unit of Dermatology, University of Padua
2005 to date Associate Professor, Unit of Dermatology, University of Padua

230 international publications in the field of immunodermatology and cutaneous oncology (please see PubMed)

Education and training
High School at the Liceo Scientifico "De Giorgi", Lecce
Degree in Medicine and surgery at University of Florence marks 110 cum laude date 15 July 1986
Specialitation in Dermatology and Venereology at the University of Florence marks 70/70 date 19 July 1989
Ph.D. in Cutaneous Immunology at the Royal Postgraduate Medical School, University of London date 21 February 1996

Mother tongue Italiano
Other languages

Comprensione	Parlato	Scritto			
	Ascolto	Lettura	Interazione orale	Produzione orale	
English	C1	C1	C1	C1	C1

Padova, 05 Agoust 2022

Selected PubMed Publications 2022.

1. Russano F, Russo I, Del Fiore P, Di Prata C, Mocellin S, Alaibac M. Bleomycin-based electrochemotherapy for the treatment of a Buschke-Löwenstein tumor (perianal giant condyloma) in an HIV-positive kidney transplant recipient: A case report. Oncol Lett. 2022 Nov 7;24(6):466. doi: 10.3892/ol.2022.13586. eCollection 2022 Dec. PMID: 36406182 Free PMC article.

2. Hernandez Navarro S, Segura Tejedor J, Bajona Roig M, Luisetto R, Fedrigo M, Castellani C, Angelini A, Alaibac M, Bordignon M. Medicine (Baltimore). Efficacy of a topical formulation containing MIA (Melanoma Inhibitory Activity) - Inhibitory peptides in a case of recalcitrant vitiligo in combination with UV exposure. 2022 Nov 18;101(46):e31833. doi: 10.1097/MD.0000000000031833. PMID: 36401489 Free PMC article.
3 Ciolfi C, Sernicola A, Alaibac M. Role of Rituximab in the Treatment of Pemphigus Vulgaris: Patient Selection and Acceptability. Patient Prefer Adherence. 2022 Nov 7;16:3035-3043. doi: 10.2147/PPA.S350756. eCollection 2022. PMID: 36387051 Free PMC article. Review.
4. Sernicola A, Colpo A, Leahu AI, Alaibac M. Granulocyte Apheresis: Can It Be Associated with Anti PD-1 Therapy for Melanoma? Medicina (Kaunas). 2022 Oct 6;58(10):1398. doi: 10.3390/medicina58101398. PMID: 36295558 Free PMC article.
5. Gnesotto L, Mioso G, Alaibac M. Use of granulocyte and monocyte adsorption apheresis in dermatology (Review). Exp Ther Med. 2022 Jun 24;24(2):536. doi: 10.3892/etm.2022.11463. eCollection 2022 Aug. PMID: 35837066 Free PMC article. Review.
6. Russo I, Sartor E, Fagotto L, Colombo A, Tiso N, Alaibac M. The Zebrafish model in dermatology: an update for clinicians. Discov Oncol. 2022 Jun 17;13(1):48. doi: 10.1007/s12672-022-00511-3. PMID: 35713744 Free PMC article. Review.
7. Orlando G, Molon B, Viola A, Alaibac M, Angioni R, Piaserico S. Psoriasis and Cardiovascular Diseases: An Immune-Mediated Cross Talk? Front Immunol. 2022 May 24;13:868277. doi: 10.3389/fimmu.2022.868277. eCollection 2022. PMID: 35686132 Free PMC article. Review.
8. Del Fiore P, Russo I, Dal Monico A, Tartaglia J, Ferrazzi B, Mazza M, Cavallin F, Tropea S, Buja A, Cappellesso R, Nicolè L, Chiarion-Sileni V, Menin C, Vecchiato A, Dei Tos AP, Alaibac M, Mocellin S. Altitude Effect on Cutaneous Melanoma Epidemiology in the Veneto Region (Northern Italy): A Pilot Study. Life (Basel). 2022 May 17;12(5):745. doi: 10.3390/life12050745. PMID: 35629411 Free PMC article.
9. Guarnieri G, Bertagna De Marchi L, Marcon A, Panunzi S, Batani V, Caminati M, Furci F, Senna G, Alaibac M, Vianello A. Relationship between hair shedding and systemic inflammation in COVID-19 pneumonia. Ann Med. 2022 Dec;54(1):869-874. doi: 10.1080/07853890.2022.2054026. PMID: 35341398 Free PMC article.
10. Albertin G, Ravara B, Kern H, Zampieri S, Loefler S, Hofer C, Guidolin D, Messina F, De Caro R, Alaibac M, Carraro U. Trauma of Peripheral Innervation Impairs Content of Epidermal Langerhans Cells. Diagnostics (Basel). 2022 Feb 23;12(3):567. doi: 10.3390/diagnostics12030567. PMID: 35328120 Free PMC article.
11. Sernicola A, Cama E, Pelizzo MG, Tessarolo E, Nicolli A, Viero G, Alaibac M. In vitro Assessment of Solar Filters for Erythropoietic Protoporphyria in the Action Spectrum of Protoporphyrin IX. Front Med (Lausanne). 2021 Dec 20;8:796884. doi: 10.3389/fmed.2021.796884. eCollection 2021. PMID: 34988101 Free PMC article.
12. Deotto ML, Spiller A, Sernicola A, Alaibac M. Bullous pemphigoid: An immune disorder related to aging (Review). Exp Ther Med. 2022 Jan;23(1):50. doi: 10.3892/etm.2021.10972. Epub 2021 Nov 15. PMID: 34934428 Free PMC article. Review.
13. Near-infrared photoimmunotherapy for the treatment of skin disorders. Russo I, Fagotto L, Colombo A, Sartor E, Luisetto R, Alaibac M. Expert Opin Biol Ther. 2022

Apr;22(4):509-517. doi: 10.1080/14712598.2022.2012147. Epub 2021 Dec 3. PMID: 34860146.

14. Alaibac M, Albertin G, Ravar B, Kern H, Hofer C, Loefler S, Guidolin D, Rambaldo A, Porzionato A, De Caro R, Zampieri S, Pond AL, Carraro U, Jurecka W. 2019. Two-years of home based functional electrical stimulation recovers epidermis from atrophy and flattening after years of complete Conus-Cauda Syndrome. Medicine (Baltimore). 98(52):e18509. doi.org/10.1097/MD.0000000000018509.

Chapter 15
Pupils and endless dreams

CHAPTER 15.1. Pupils

15.1. Pupils

Marco Sandri, Sandra Zampieri, Katia Rossini, Corrado Rizzi, Vincenzo Vindigni, Barbara Ravara, Alessandro Salviati, Nicoletta Adami, Anna Jakubiec-Puka, Donatella Biral, Marzena Podhorska-Okolow, Maria Chiara Maccarone with residents enrolled in the 2022 Course of the School of Physical Medicine and Reahbilitation of the Padua University, Italy and Roger H. Coletti

Marco Sandri and Sandra Zampieri decided more or less in the same years to select my lab group for their laboratory experiences and to respectively obtain degrees in Medicine and Biological Sciences. It was the early 90s. I never understood their decisions with so many brilliant mentors around the Vallisneri Building of the University of Padua, where 500 researchers work in both Biomedicine and Biological Sciences. In any case, they both graduated under my supervision and then moved on to other independent experiences, little influenced by me. After international experiences, both returned to Padua at the Department of Biomedical Sciences, where they still work.
Three particular cases are Katia Rossini Corrado Rizzi and Barbara Ravara.
Katia Rossini, after graduating in Biological Sciences at the University of Padua (Italy) in 1994, spent several years in my and other Padua labs. She then decided to leave the Doctorate in Biomedical Sciences and choosed the Degree Course in Neuro and Psychomotricity Therapy for the Developmental Age at the University of Padua, achieved in 2009. Now she is very happy to do her work for children with neurodevelopmental disorders. Graduated in Medicine and Specialist in Plastic Surgery, Corrado Rizzi passionate about laboratory work and electrophoretic analyses of protein, moved to Food Biotechnology at the University of Verona (Italy) where he is still active in meat science.
Barbara Ravara graduated in “Biochemical Sciences, biophysic and biochemistry” at the Department of Biology, University of Padova with a Thesis on: “Fluorescence studies in the evaluation of ultraviolet radiation damage on plants”, but soon she moved at the Instute of General Pathology, where she started working with Luciano Dalla Libera and a group of medical doctors specialist in arterial hypertension. After Luciano retired, she remained at the Department of Biomedical Sciences working with various colleagues and finally with Dr. Helmut Kern and my self. She was and is able to work under the supervision of very different responsible persons, recently also with Colleagues of the Department of Neurosciences, Human Anatomy Section (Prof. Giovanna Albertin) always collaborating with Sandra Zampieri. Her dedication and capacities to bench-work, good moode and numerous experiences with different personalities make of her an excellent collaborator able to spend her expertises in biochemistry and microscopic anatomy in succesful international collaborations, as it is well documented below. But I have to add

a few more pupils, though I have not all the details, specifically Alessandro Salviati, Nicoletta Adami, Anna Jakubiec-Puka, Donatella Biral and Marzena Podhorska-Okolow.
To this Chapter 15 I must also add the curriculum vitae of Maria Chiara Maccarone. She is a PhD Student in Neuroscience, Department of Neuroscience, University of Padua and Resident doctor in Physical Medicine and Rehabilitation, University of Padua, Padua, Italy She last year agreed to invest part of her time to "sell" to her patients one of my latest dreams: making my proposal of home-based Full Body in-Bed Gym (hbFBiBG) acceptable to people with mobility difficulties or just elderly people with very sedentary lifestyle. It won't be easy to convince those people to change their lifestyle, but it's something Maria Chiara and the 2022 students of the School of Physical Medicine and Rehabilitation of the University of Padua might be worth planning and realizing. I am confident that readers of this book will find PubMed publications describing how my dream came true. The first goal is achieved: most of these new collaborators are actively recruiting patients. We'll see how many of them take the message home and how long they practice in bed at least three times a week for the next six months. The road will be long, but the first steps have been taken and the decision of this group of young doctors is a non-trivial result (something I've been looking for for five years). Curious readers of my book will find more about hbFBiBG in the references below, at the pages 166 and 167 of this book (Chapter 10. Muscle aging decay: Countermeasures by FES and Full-Body in-Bed Gym) and in the YouTube video dynamically presenting the series of exercises in bed at the 2018 link:
https://www.youtube.com/watch?v=N1RuG3371-Y&feature=youtu.be
and then 5 years later at:
https://www.youtube.com/watch?v=pcHKmxCLYFs&t=336s

Also added to Proofs is the CV of Roger H. Coletti, a retired interventional cardiologist turned "translational myologist", perhaps my last student.

I am proud to have worked for and with all my old and recent pupils.

Their following CVs explain why!

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Carraro U, Gava K, Baba A, Marcante A, Piccione F. To Contrast and Reverse Skeletal Muscle Atrophy by Full-Body In-Bed Gym, a Mandatory Lifestyle for Older Olds and Borderline Mobility-Impaired Persons. Adv Exp Med Biol. 2018;1088:549-560. doi: 10.1007/978-981-13-1435-3_25. PMID: 30390269 Review.

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Coletti RH. The ischemic model of chronic muscle spasm and pain. Eur J Transl Myol. 2022 Jan 18; 32(1):10323. doi: 10.4081/ejtm.2022.10323. PMID: 35044134; PMCID: PMC8992665. [Cited by Qi F, Huang H, Cai Y, Fu Z. Adjacent Fu's subcutaneous needling as an adjunctive healing strategy for diabetic foot ulcers: Two case reports. Medicine (Baltimore). 2022 Dec

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Chapter 15.1. Pupils

15.1.1. Marco Sandri

1996 MARCO SANDRI 2022

Curriculum of Marco Sandri, M.D.

Full Professor and Chair of Department of Biomedical Science,
Medical School, University of Padova;
Group Leader of the Venetian Institute of Molecular Medicine
via Orus 2, 35129 Padova, Italy
Tel: 0039 049 8276363; Tax code: SNDMRC67S05G224T
E-mail: marco.sandri@unipd.it

Personal information

Born: 5 November 1967, Padova, Italy.

Citizenship: Italian.

Marital Status: Married

Education

07/1996. Medical Doctor graduated with honours, University of Padova.

12/2001 Specialist (PhD equivalent) in Pathology, University of Padova, Italy.

Research experience

1989-2000. Internship, Department of Biomedical Science, University of Padova, Laboratory of Prof. Ugo Carraro. Experiences in biochemistry, cell cultures, histochemistry, molecular biology, and in vivo experiments on rats and mice.

1996-2000. Fellowship, Institute of Experimental and Laboratory Medicine, University of Padova. Experience in haematology, in particular on leukemic cells. Methods: cell culture, flow cytometry, RT-PCR, cell proliferation assay, cell death assay.

2001-2002. Postdoc, Department of Biomedical Science, University of Padova, laboratory of Prof. S. Schiaffino. Experience in molecular biology and in vivo gene delivery on skeletal muscles of rat and mouse.

2002-2005. Postdoc, Department of Cell Biology, Harvard Medical School, laboratory of Prof. AL Goldberg. Experience in molecular biology, biochemistry and cell culture.

2005-2009. Assistant Telethon Scientist, Dulbecco Telethon Institute at Venetian Institute of Molecular Medicine (VIMM), Padova.

2006- 2013. Assistant Professor, Department of Biomedical Science, Medical School, University of Padova, Padova.

2006-present. Group Leader at Venetian Institute of Molecular Medicine (VIMM), Padova.

2010-2015. Associate Telethon Scientist, Dulbecco Telethon Institute at Venetian Institute of Molecular Medicine (VIMM), Padova

2011-present. Adjunct Professor, Department of Medicine, Faculty of Medicine, McGill University, Montreal, Canada.

2013- 2014. Associate Professor, Department of Biomedical Science, Medical School, University of Padova, Padova.

2013-2015. Principal Investigator at TIGEM, Napoli.

2013-2019. Chair of the CIR-Myo, Interdepartmental Research Center of Myology. Univeristy of Padova, Padova

2014-present Full Professor Department of Biomedical Science, Medical School, University of Padova, Padova.

2015-2019. Vice Chair of the Department of Biomedical Science, Medical School, University of Padova, Padova.

2019-present. Head of the Department of Biomedical Science, Medical School, University of Padova, Padova.

Awards and Prizes

1997. "Luigi Casati" prize, conferred by National Academy of Lincei.

2003 "Terme Euganee Award" on Skeletal Muscle Regeneration, Reconstruction and Engineering

2004. Selected between the five finalist for the world-wide award "Young Cell Signaller 2004" on "Regulation and therapeutic potential of the PI3-kinase/PKB signalling pathway".

2005. Dulbecco Telethon Institute carrier award (Assistant Level)

2006. "Best Poster" prize at "4th International symposium on Autophagy" Mishima, Japan

2007. "Best Poster" prize at "4th Cachexia Conference", St. Petersburg, USA.

2008. "Best Poster" prize at "Autophagy in stress, development and disease" Gordon Conference, Ventura, USA

2009. "Best Poster" prize at "5th Cachexia Conference", Barcelona, ESP.

2010 Dulbecco Telethon Institute carrier award (Associate level)

2011. ERC Consolidator research grant award

2018 ERC panel member of LS3 Cellular and Developmental Biology.

2020 ERC panel member of LS3 Cellular and Developmental Biology

2021 Highly Cited Researcher, which recognizes the true pioneers in their fields over the last decade, demonstrated by the production of multiple highly-cited papers that rank in the top 1% by citations for field and year in the Web of Science™. Of the world's scientists and social scientists, Highly Cited Researchers truly are one in 1,000. See the link Recipients - Highly Cited | Researcher Recognition (webofscience.com)

Editorial board of:

Journal of Cachexia, Sarcopenia and Muscle Wasting
Skeletal Muscle
Cell Stress
Life Science Alliance
European Journal Translational Myology

Teaching experience

2004-2017 Lecturer in General Pathology at Nurse School, University of Medicine, Padova (IT)

2004-2005 Lecturer in Physiology at School of Laboratory Technician, University of Medicine, Padova (IT)

2007-2012 Lecturer in General Pathology at Nutritional School, University of Medicine, Padova (IT)

2009-2013 Lecturer in General Pathology at Medical School, University of Padova (IT)

2014-present Lecturer in Clinical Pathology at Medical School, University of Padova (IT)

2015-present Lecturer in Physiopathology at Medical School, University of Padova (IT)

Supervision of Graduate Students, Postdoctoral Fellows and their career development

Since 2005, I mentored 20 PhD, 20 postdocs at Medical School, University of Padova. All the postdocs trained in my lab found a position in local pharma/industry as well as at University [1 Assistant Professor at University of Padova, 2 Associate Professors at University of Padova, 1 Assistant Professor at Federal University of Minas Gerais (Brazil)].

Invited Speaker at International Meeting

I have been invited as speaker at the following international conferences:

1. "7th Terme Euganee Meeting on Rehabilitation" 2003 Padova, (Italy);
2. "Regulation and therapeutic potential of the PI3-kinase/PKB signalling pathway" 2004, Dundee (UK);
3. "FISV" 2004 Riva del Garda, Trento (Italy).
4. "3rd Cachexia Meeting"2005, Rome, Italy;
5. EMBO/FEBS workshop "The Molecular and Cellular Mechanisms underlying Skeletal Muscle Formation and Repair"; EMBO workshop 2005, Fontevraud, France;
6. "2nd Myores Congress" 2006, Prague, Czech Republic;
7. "Gutmann Memorial, 30-year after The Long Lasting Denervated Muscle" 2007, Padova, (Italy);
8. "2nd Italian meeting of Italian Society for Space Biomedicine and Biotechnology" 2007 Bari, Italy;
9. "XXVI European Muscle Conference" 2007, Stockholm, Sweden;

10. Marie Curie Symposium “The ubiquitin-proteasome system in cardiovascular disease” 2007, Hamburg, Germany;
11. Gordon Conference “Autophagy in stress, development and disease” 2008, Ventura, Los Angeles, USA;
12. Cold Spring Harbor Laboratory "Molecular Mechanisms Modulating Skeletal Muscle Mass and Function", 2008, Long Island, New York, USA;
13. “2008 Spring Padua Muscle Days Functional Recovery of Muscle Tissue” 2008, Padova, Italy;
14. “FISV” 2008 Riva del Garda, Trento (Italy);
15. “XXVII European Muscle Conference” 2008, Oxford, UK;
16. EMBO Conference “The molecular and cellular mechanisms regulating skeletal muscle development and regeneration” 2009 Barcelona, Spain;
17. Gordon Conference “Oxidative Stress & Disease.” 2009, Ciocco, Lucca, Italy;
18. 38th Annual Meeting of the American Aging Association" Workshop “Protein Quality and Aging” 2009, Phoenix, AZ, USA;
19. “XXVIII European Muscle Conference”. 2009 Lille, France.
20. EMBO conference “Autophagy. Cell Biology, Physiology and Pathology” 2009, Monte Verita, Ascona, Switzerland.
21. 7th Annual Scientific Sessions of the Society for Heart and Vascular Metabolism. “Cardiac metabolism in health and disease. Mitochondria and Oxidative Stress.” 2009, Padova, Italy.
22. Gordon Conference “Autophagy in Stress Development and Disease.” 2010, Ciocco, Lucca, Italy.;
23. 1st International Congress of Translational Research in Human Nutrition “Protein-energy metabolism in aging and chronic diseases: role of nutrition and physiscal activity” 2010, Clermont-Ferrand, France.
24. XX world congress of the International Society of Heart Research (ISHR) World Congress. 2010, Tokyo, Japan.
25. International conference IFR 83 2010 “Oxidative Metabolism in Health and Diseases”, 2010, Paris, France.
26. Heart Failure Association Winter Research Meeting 2011, Les Diablerets, Switzerland.
27. IRB Barcelona BioMed Conference on “Mitochondrial autophagy”. 2011, Barcelona, Spain
28. Experimental Biology 2011, American Physiological Society, Environmental and Exercise Physiology Section, “Autophagy in Skeletal Muscle” session, Washington, USA
29. Fourth International Congress of Myology “Myology2011”, Lille, France.
30. American Diabetes Association’s 71st Scientific Session “Autophagy, Ageing and Metabolic Control” session, 2011, San Diego, USA.
31. The 61st Annual Scientific Meeting of the British Society for Research on Ageing (BSRA). “The Science of Ageing – Global Progress” 2011, Brighton, UK.
32. EMBO Meeting 2011, Autophagy in disease & development, Vienna, Austria.
33. “XXX European Muscle Conference”. 2011 Berlin, Germany.
34. International Conference on Muscle Wasting 2011. “Molecular Mechanisms of Muscle Growth and Wasting in Health and Disease”. 2011, Monte Verita, Ascona, Switzerland.

35. Gordon Conference “Autophagy in Stress Development and Disease.” 2012, Ventura, USA
36. 7th Ascona International Workshop on Cardiomyocyte Biology, “Cardiac Pathway of Differentiation, Metabolism and Contraction”. 2012, Monte Verita, Ascona, Switzerland.
37. 15th International Biochemistry of Exercise Congress (IBEC). 2012, Stockholm, Sweden.
38. Cancer Cachexia Conference. 2012, Boston, USA.
39. 57th Annual Meeting of the German Society of Neuropathology and Neuroanatomy (DGNN). 2012, Erlangen, Germany.
40. “XXXI European Muscle Conference”. 2012 Rhodes, Greece
41. Symposium “Skeletal Muscle Dysfunction in the Critical ill”. 2012, Montreal, Canada.
42. Experimental Biology (EB) 2013, American Physiological Society, Environmental and Exercise Physiology Section, “Mitochondrial Dynamics and Turnover with exercise”. 2013 Boston, USA.
43. V Covian Symposium. 2013 Ribeirão Preto, Brazil.
44. AIM - Associazione Italiana di Miologia. 2013, Stresa, Italy.
45. EMBO Workshop - Molecular Mechanisms of muscle growth and wasting in health and disease. 2013, Monte Verita, Ascona, Switzerland.
46. ABCD 2013, Ravenna, Italy
47. EMBL conference, Myofibrillar Z-disk Structure and Dynamics. 2013, EMBL Hamburg, Germany.
48. ENMC European Neuro-Muscular Center . 201st ENMC International Workshop. Autophagy in Muscle Dystrophies, translational approach. 2013, Naarden, The Netherlands.
49. Keystone Symposia, Growth and wasting in Heart and Skeletal Muscle. 2014, Santa Fe, New Mexico, USA
50. Advances in Skeletal Muscle Biology in Health and Disease. 2014, Gainesville, Florida, USA
51. 93rd Annual Meeting of the German Physiological Society (DPG 2014). 2014, Mainz, Germany.
52. EMBO conference. Molecular biology of muscle development and regeneration. 2014, Acaya (Lecce) Italy
53. CIM Conference on Inflammation and Metabolism. 2014, Copenhagen, Denmark
54. Special Interest Meeting. Molecular Insight into Muscle Function and Protein Aggregate Myopathies. 2014, Potsdam, Germany.
55. 13th International Congress on Neuromuscular Diseases. 2014, Nice, France.
56. Society for Free Radical Research-Europe (SFRR-E) Meeting 2014, Paris, France
57. 65th SIF National Congress, (Italian Society of Physiology). 2014 Anacapri, Italy
58. Fall Meeting of the International Graduate School in Molecular Medicine Ulm. 2014, Ulm, Germany
59. 9th International Conference on Strength Training (ICST). 2014, Abano Terme (Padova), Italy
60. Australian Physiological Society Meeting 2014, University of Queensland, Brisbane, Australia
61. Experimental Biology 2015, American Physiological Society, Environmental and

Exercise Physiology Section, "Autophagy in Muscle", Boston, USA
62. Neurosciences in Critical Care International Symposium (NICIS). 2015, Paris, France
63. Physiology 2015, Cardiff, UK
64. EMBO Workshop -Molecular Mechanisms of muscle growth and wasting in health and disease. 2015, Monte Verita, Ascona, Switzerland.
65. 8th International Conference on Cachexia, Sarcopenia and Muscle Wasting. 2015, Paris, France
66. FRIAS Black Forest Winter Conference on "Autophgay Membrane Trafficking & Dynamics in Ageing and Disease. 2016. Friburg, Germany
67. 1st INEM International Symposium, Proteostasis in Growth & Disease. 2016, Paris, France
68. Myology 2016. 5th International Congress of Myology. 2016. Lyon, France
69. 7th Proteasome & Autophagy Workshop. 2016 Clermont-Ferrand, France
70. XXII world congress of the International Society of Heart Research (ISHR) World Congress. 2016, Buenos Aires, Argentina
71. SSIEM (Society for the Study of Inborn Errors of Metabolism) annual symposium. 2016, Roma, Italia
72. 3rd Cancer Cachexia Conference. 2016, Washington, USA
73. 1st International Conference on targeting Skeletal Muscle Oxidative Metabolism to Treat Human Disease. 2016, London, UK.
74. 16th Fondation IPSEN Meeting "Hormone, metabolism and the benefits of exercise". 2016, Paris, France
75. 9th International Conference on Cachexia, Sarcopenia & Muscle Wasting. 2016, Berlin, Germany
76. Padua-Innsbruck Joint Meeting 2016 "Mitochondria in Health & Disease". 2016, Innsbruck, Austria
77. Advance in Skeletal Muscle Biology in Health and Disease. 2017, Gainesville, Florida, USA
78. 53 Congresso Associazione Italiana Neuropatologia e Neurobiologia clinica. Workshop: I meccanismi dell'autofagia nelle patologie del sistema nervoso e del muscolo. 2017, Padova, Italy
79. Gordon Conference "Myogenesis" Advanced mechanisms of growth and repair in myogenesis" 2017, Ciocco, Lucca, Italy.
80. 4th Ottawa International Conference on Neuromuscular Disease & Biology. 2017, Ottawa, Canada
81. FEPS 2017, the joint meeting of the Federation of European Physiological Societies and the Austrian Physiological Society. 2017, Wien, Austria
82. 4th ShanghaiTech- SIAS BioForum. Advances and Perspectives in Integrative Biology of Cellular Processes. 2017, Shanghai, China.
83. SFEIM. La 6°edition du livre "Inborn Metabolic Diseases - Diagnosis and Treatment". 2017, Paris, France
84. 10th International Conference on Cachexia, Sarcopenia & Muscle Wasting. 2017, Rome, Italy.
85. EMBO Workshop. Lysosome and Metabolism. 2018, Pozzuoli, Napoli, Italy
86. Muscle Development Regeneration and Disease. 2018, Berlin, Germany
87. 8th Proteasome and Autophagy Congress. 2018, Clermont Ferrand, France
88. ACSM Conference on Integrative Physiology of Exercise. 2018, San Diego, USA

89. 4th Cancer Cachexia Conference, 2018 Philadelphia, USA.
90. 11th International Conference on Cachexia, Sarcopenia & Muscle Wasting. 2018, Maastricht, Netherland.
91. Molecular Mechanisms of Muscle Wasting during Ageing and Disease. 2018 Ascona, Switzerland.
92. 7th European Symposium: Steps Forward in Pompe Disease. 2018 Copenhagen, Denmark
93. Advances in Skeletal Muscle Biology in Health and Disease. 2019 Gainesville, USA
94. 21st Annual Meakins-Christe laboratories international workshop. 2019 Montreal, Canada
95. Gordon Conference “Myogenesis” Advanced mechanisms of growth and repair in myogenesis” 2019, Ciocco, Lucca, Italy.
96. International meeting on Metabolism Meet Function. 2019, Torino Italy
97. ECTS PhD training course, Bologna, Italy
98. ABCD congress. The biennial congress of the Italian Association of Cell Biology and Differentiation. 2019, Bologna, Italy
99. X International Congress on Glycogenosis, 2019 Malaga, Spain
100. ECTS 2020 Digital Congress. Plenary Symposium 4: Insights from Outside: Muscle & Bone
101. ECTS 2020 Digital Congress ECTS@Home weekly sessions. Working Group 5: Mammalian Models – Focus on Bone and Metabolism and Bone Marrow Adiposity
102. ISCaM2020, 7th Annual Meeting Webinar Series, Systemic Metabolism and Cancer, London, The Francis Crick Institute
103. AIM/ASNP – Digital Meeting 2020
104. SCWD Digital Cachexia Conference 2020:
105. 1st Brazilian Muscle Biology Symposium, Digital Meeting 2021
106. EMBO Workshop. Muscle formation, maintenance, regeneration and pathology. 2022, Gouvieux-Chantilly, France

Keynote Lectures/Plenary Lectures.

1. “XXXIII European Muscle Conference”. 2014 Salzburg, Austria
2. 19th International Congress of World Muscle Society. 2014, Berlin, Germany
3. 7th European Symposium. Steps Forward in Pompe Disease. 2014, Torino, Italy
4. New Pathophysiological Mechanisms in Obesity and Type2 Diabetes. 2015 Padova, Italy.
5. Multifaceted Muscle. 2016, Montreal, Canada
6. 21th International Congress of World Muscle Society. 2016, Granada, Spain
7. 2nd Annual Research Meeting Amsterdam Movement Science, 2018, Amsterdam, Netherlands.
8. 9th PROTEASOME and AUTOPHAGY CONGRESS, 2021 Clermont Ferrand, France

Organizer of International Conference and Workshop

I have organized the following international conferences:

1. XXIX European Muscle Conference. 2010, Padova, Italy.
2. Workshop MUSCLE MASS REGULATION, 2011, FP7 MYOAGE, Acaya, Lecce, Italy
3. EMBO conference, Molecular Biology of Muscle Development and Regeneration.

2014, Acaya, Lecce, Italy

4. 2014 Spring Padua Muscle Days 'Activity-dependent trophism of neurons and their target organs in aging, pathology and rehabilitation' 2014, Montegrotto, Padova, Italy.
5. 2015 Spring Padua Muscle Days 'Translational Myology in Aging and Neuromuscular Disorders' 2015, Montegrotto, Padova, Italy.
6. 2016 Spring Padua Muscle Days. Muscle Decline in Aging and Neuromuscular Disorders Mechanisms and Countermeasures. 2016, Montegrotto, Padova, Italy
7. 2018 Spring Padua Muscle Days. Giovanni Salviati Memorial. 2018, Montegrotto, Padova, Italy
8. 2019 Spring Padua Muscle Days. Translational myology and mobility medicine. 2019, Montegrotto, Padova, Italy
9. 2022 Padua Days of Muscle & Mobility Medicine. On site (150 attendees), Padova, Italy

Participation in Scientific Reviews

I have reviewed papers for the following Journals: Nature, Science, Nature Medicine, Nature Genetics, Nature Cell Biology, Nature Communication, Nature Metabolism, Cell Metabolism, Cell Reports, Blood, EMBO Journal, Journal of Clinical Investigation, Plos Biology, Plos ONE, Journal of Biological Chemistry, EMBO Molecular Medicine, Cardiovascular Research, American Journal of Physiology Cell Physiology, American Journal of Physiology Heart and Circulatory Physiology, Human Molecular Genetics, Autophagy, Cell Death and Differentiation, Cell Death and Disease, FEBS Letters, Developmental Biology, Molecular Medicine, Stem Cells, Trends in Endocrinology and Metabolism, Traffic, Metabolism, Chest, Neuromuscular Disorders, European Journal of Applied Physiology, Acta Physiologica, Journal of Physiology, Biochim Biophys Acta, Experimental Gerontology, J. Muscle Res. Cell Motility, Skeletal Muscle.

Publications in journals with IF. h-index: 76 (scopus). Total Citations: 31900 (scopus)

1) Carraro U, Rizzi C, Sandri M. SDS PAGE: Effective recovery by KCl precipitation of highly diluted muscle proteins solubilized with sodium dodecy sulfate. Electrophoresis 1991; 112: 1005-1010.
2) Sandri M, C. Rizzi, C Catani, Carraro U. Selective Removal of Free Dodecyl Sulfate from 2-Mercaptoethanol-SDS-Solubilized Proteins before KDS-protein Precipitation. Anal. Biochem. 1993; 213: 34-39.
3) Carraro U, Rizzi C, Sandri M, Doria D. A new two-step precipitation method removes free-SDS and Thiol reagents from diluted solutions, and then allows recovery and quantitation of proteins. Biochem. Bioph. Res. Com. 1994; 200: 916-924.
4) Rossini K, Rizzi C, Sandri M, Bruson A, Carrararo U. High-resolution sodium dodecyl sulfate-polyacrylamide gel elecrophoresis and immunochemical identification of the 2X and embryonic myosin heavy chains in complex mixtures of isomyosin. Electrophoresis 1995; 16: 101-104.
5) Carraro U, Bruson A, Catani C, Dalla Libera L, Massimino ML, Rizzi C, Rossini K, Sandri M, Cantini M. Effects of beta1-Integrin Antisense Phosphorothioate-Modified Oligonucleotide on Myoblast Behaviour In Vitro. Cell Biochem. Funct. 1995; 13: 99-104.
6) Sandri M, Carraro U, Podhorska-Okolov M, Rizzi C, Arlsan P, Monti D, Franceschi C.

Apoptosis, DNA damage and ubiquitin expression in normal and mdx muscle fibers after exercise. FEBS Lett. 1995 ; 373: 291-295.

7) Sandri M, Podhorska-Okolov M, Geromel V, Rizzi C, Arlsan P, Franceschi C, Carraro U. Exercise induces myonuclear ubiquitination and apoptosis in dystrophin deficient muscle of mice. J. Neuropath. Exp. Neur. 1997; 56: 45-57.
8) Sandri M, Massimino ML, Cantini M, Giurisato E, Sandri C, Arlsan P, Carraro U. Dystrophin deficient myotubes undergo apoptosis in mouse primary muscle cell culture after DNA damage. Neurosci Lett. 1998; 252: 123-126.
9) Sandri M, Minetti C, Pedemonte M, Carraro U. Apoptotic myonuclei in human Duchenne muscular dystrophy. Lab. Invest. 1998; 78: 1005-1016.
10) Podhorska-Okolov M, Sandri M, Zampieri S, Brun B, Carraro U. Apoptosis of myofiber and satellite cells: exercise induced damage in skeletal muscle of mouse. Neuropath. Appl. Neuro. 1998; 24: 518-531.
11) Vescovo G, Zennaro R, Sandri M, Carraro U, Leprotti C, Ceconi C, Ambrosio GB, Dalla Libera L. Apoptosis of skeletal muscle myofibers and interstitial cells in experimental heart failure. J. Mol. Cell Cardiol. 1998; 30: 2449-2459.
12) Sandri M, Carraro U. Apoptosis of skeletal muscles during development and disease. Int. J. Biochem. Cell. Biol. 1999; 31: 1373-1390.
13) Dalla Libera L, Zennaro R, Sandri M, Ambrosio GB, Vescovo G. Apoptosis and atrophy in rat slow skeletal muscle in chronic heart failure. Am. J. Physiol. 1999; 277: C982-C986.
14) Biral D, Jakubiec-Puka A, Ciechomska I, Sandri M, Rossini K, Carraro U, Betto R. Loss of dystrophin and some dystrophin-associated proteins with concomitant signs of apoptosis in rat leg muscle overworked in extension. Acta Neuropathol. 2000; 100: 618-626.
15) Vescovo G, Volterrani M, Zennaro R, Sandri M, Ceconi C, Lorusso R, Ferrari R, Ambrosio GB, Dalla Libera L. Apoptosis in the skeletal muscle of patients with heart failure: is it associated with clinical and biochemical changes? Heart 2000; 84: 431-437.
16) Sandri M, Sandri C, Brun B, Giurisato E, Cantini M, Rossini K, Destro C, Arslan P, Carraro U. Inhibition of FasL sustains phagocytic cells and delays myogenesis in regenerating muscles fibers. J. Leukocyte Biol. 2001; 69: 482-489.
17) Dalla Libera L, Ravara B, Angelini A, Rossini K, Sandri M, Tiene G, Ambrosio GB, Vescovo G. Beneficial effects on skeletal muscle of the ATII blocker irbesartan in experimental heart failure. Circulation 2001; 103: 2195-2200.
18) Sandri M, El Meslemani A, Sandri C, Schjerling P, Vissing K, Andersen JL, Rossini K, Carraro U, Angelini C. Caspase 3 Expression Correlates With Skeletal Muscle Apoptosis in Duchenne and Facioscapulo Human Muscular Dystrophy. A potential target for pharmacological treatment. J. Neuropath. Exp. Neur. 2001; 60: 302-312.
19) Dalla Libera L, Sabbadini R, Renken C, Ravara B, Sandri M, Betto R, Angelini A, Vescovo G. Apoptosis in the skeletal muscle of rats with heart failure is associated with increased serum levels of TNF□ and sphingosine. J. Mol. Cell Cardiol. 2001; 33: 1871-1878.
20) Vescovo G, Ravara B, Angelini A, Sandri M, Carraro U, Ceconi C, Dalla Libera L. Efect of thalidomide on the skeletal muscle in experimental heart failure. Eur. J. Heart Fail. 2002; 4(4):455-60.
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Tacchetti G, Bari M, Manconi R, D'Andrea MR, Silvestri B, Vinante O. Human fibroblasts from normal and malignant breast tissue grown in vitro show a distinct senescence profile and telomerase activity. Histochem J. 2002; 34: 403-10.

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23) Vescovo G, Ravara B, Gobbo V, Sandri M, Angelini A, Della Barbera M, Dona M, Peluso G, Calvani M, Mosconi L, Dalla Libera L. L-Carnitine: a potential treatment for blocking apoptosis and preventing skeletal muscle myopathy in heart failure. Am J Physiol Cell Physiol. 2002; 283: C802-10.

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Chapters of Books of Pathology

1. Capitolo 60. PATOLOGIA GENERALE DEL MUSCOLO SCHELETRICO. Carraro U, Sandri M, Zampieri S. Tomo II PATOLOGIA GENERALE. IV Edizione, Piccin.
2. Translation of the Chapter 27. Skeletal Muscle. RUBIN' S PATHOLOGY: CLINICOPATHOLOGICAL FOUNDATIONS OF MEDICINE. Sixth Edition. Sandri M, and Mammucari C. Edizione Piccin
3. PATHOBIOLOGY OF HUMAN DISEASE, A DYNAMIC ENCYCLOPEDIA OF DISEASE MECHANISMS, Sandri M. (2014) Atrophy and Hypertrophy: The Balance Between Removal and Synthesis of Proteins and Organelles. Editor: Elsevier; 2014. p. 64-71.
4. Capitolo 62. PATOLOGIA GENERALE DEL MUSCOLO SCHELETRICO. Sandri M. Tomo II PATOLOGIA GENERALE. V Edizione, Piccin.
5. Capitolo 49. STRUTTURA E FUNZIONE DEL MUSCOLO SCHELETRICO. Sandri M. Tomo II FISIOPATOLOGIA GENERALE. VI Edizione, Piccin
6. Translation of the Chapter on Skeletal Muscle. RUBIN' S PATHOLOGY: CLINICOPATHOLOGICAL FOUNDATIONS OF MEDICINE. Seventh Edition. Sandri M, and Mammucari C. Edizione Piccin

Patent

A patent, for which I am one of the inventors, for treating myopathies and dystrophies via autophagy modulators has been published in European Patent Office.

Past Grant Achieved

1. TCP04009 Sandri M (PI) 01/04/05-31/01/10 Founding Agency: Telethon Foundation, Total award 450000,00 euro; year award: 90000,00 euro - Cell signaling in muscle wasting. Identification of critical targets in FoxO, myostatin and ubiquitin-proteasome pathways to develop new therapeutic strategies for muscular dystrophy.
2. AFM, Sandri M (PI) 31/03/2005 31/03/2006 Founding Agency: Association Francaise Contre les Myopathies. Total award 15000,00 euro - Regulation of the ubiquitin−proteasome system by the FoxO and myostatin pathway in skeletal muscle
3. OSMA WP1B33-2 Sandri M (PI) 24/03/07-24/03/09, Founding Agency: ASI (Italian Space Agency), Total award: 155000,00 euro; year award: 51666,00 euro - Cell-based high throughput screen to identify inhibitors of muscle atrophy
4. PRIN 2007ABK385_005 Sandri M (PI) 22/10/08-22/10/10, Programmi di Ricerca Scientifica di Rilevante Interesse Nazionale (Research Program of Relevant National Interest), Founding agency: Italian Ministry of Science , Total award: 32.229 euro; year award: 16114,5 euro - Molecular Mechanisms of Muscle Wasting
5. AFM Sandri M (P.I.) 03/09/2009-03/09/2011, Founding Agency: Association Francaise Contre les Myopathies, Total award: 50000,00 Euro - Role of Autophagy in maintenance of muscle mass

6. CARIPARO project of excellence. Founding Angency: Fondazione Cassa di Risparmio, Sandri M. (P.I.) 10/01/2010-10/01/2013, Total award 420.000,00 Euro. Euro 210.000,00 available to Sandri M lab - In vivo analysis of mitochondrial remodelling system and its role in muscle function and signalling.
7. MYOAGE Schiaffino S. and Sandri M. (co-P.I.)1/01/09-1/06/13, Founding Agency: E.U. HEALTH-2007-2.4.5-10, ID: 223576, Total award: 500.000,00 Euro - Understanding and combating age-related muscle weakness.
8. TCR04003 Sandri M (P.I.) 01/02/10-01/02/15. Founding agency: Telethon Foundation. Total award 610.000,00 euro - Defining the molecular signature of muscle wasting. Identification of therapeutic targets to counteract muscle degeneration
9. PRIN 2010-11 Sandri M (PI) 1/02/13-1/02/16, Programmi di Ricerca Scientifica di Rilevante Interesse Nazionale (Research Program of Relevant National Interest). Founding agency: Italian Ministry of Science, total award: 99.120 euro. - Pathological and Physiological Mechanisms in Skeletal Muscle
10. European Research Council (ERC). Starting Grant: Consolidator Program. Founding agency: EU 7th Research Framework Programme, ID: 282310, Sandri M. (P.I). 01/11/2011-31/10/2016, Total award: 1.250.000,00 Euro - Defining The Mechanisms Of Age-Related Muscle Loss: Focus On Autophagy (MYOPHAGY)
11. Transatlantic Networks of Excellence in Cardiovascular Research Program. Founding agency: LEDUCQ Foundation. Sandri M. (P.I.) 01/10/2011-30/09/2017, Total award: 1.000.000,00 $ - Proteotoxicity: an unappreciated mechanism of heart disease and its potential for novel therapeutics
12. Founding agency: EU H2020-MSCA-RISE-2014, project no 645648 "Muscle Stress Relief". Sandri M. (P.I.) 2014-2019, Total award 1.420.000,00 Euro. Euro 200.000,00 available to Sandri M lab
13. Founding agency: Associazione Italiana Ricerca sul Cancro (AIRC) ID: 17388, Sandri M. (P.I.). 1/02/2016-1/01/2019, Total award: 334.000,00 Euro - Controlling BMP/MUSA1 axis to prevent cancer cachexia
14. Founding agency: Association Francaise Contre les Myopathies (AFM), ID: 19524, Sandri M. (P.I.). 1/06/2016-1/06/2018, Total award: 84.000,00 Euro - Dissecting the Retrograde Signal Controlling Neuomuscular Junction
15. Founding agency: CARIPARO (Starting Grant), Sandri M. (P.I.) 1/10/2016-30/09/2019 Total award: 255.388,41 Euro - Defining the Contribution of Calcium and Mitochondria to Age-Related Muscle Loss

Actual Grant

1. Founding agency: ASI (Italian Space Agency, Sandri M. (P.I.) 2019-2022, Total award: 110.000 Euro - Marcatori biologici e funzionali per la biomedicina astronautica di precisione – MARS-PRE"
2. Founding agency: AFM Telethon (#22982), Sandri M. (P.I.) 2021-2023, Total award: 74.000 Euro - Dissecting the role of an uncharacterized FoxO-dependent gene that controls autophagy and ageing"
3. Founding agency: AIRC (23257), Sandri M. (P.I.) 2019-2024, Total award: 452.000 Euro - Understanding Bmp Signaling In Cancer Cachexia
4. CARIPARO project of excellence.
5. Founding Angency: Fondazione Cassa di Risparmio, ID: 59566, Sandri M. (P.I.)

02/2022-31/01/2025 , Total award 393.000,00 Euro - Exploring the neglected genome to discover new longevity-related genes.

CHAPTER 15.1. Pupils
15.1.2. Sandra Zampieri

From left to right:
Barbara Ravara, Donatella Biral, Valerio Gobbo, Nicoletta Adami, **Sandra Zampieri**, Ugo Carraro
SANDRA ZAMPIERI 2005

SANDRA ZAMPIERI 2022

Curriculum of Sandra Zampieri

Assistant Professor
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E-mail: sanzamp@unipd.it

Research interests and career highlights
During the PhD, the research activity has addressed the role of apoptosis in the pathogenesis of autoimmune diseases, such as SLE and Inflammatory myopathies. During the post doctoral trainee, the research interest has focused on studies on muscle plasticity and their application to medical research, in particular on the role of physical exercise and electrical stimulation to restore muscle mass. The current research topic concerns the physiopathology of denervation-related skeletal muscle wasting conditions, such as aging and cancer cahexia by morphological characterizations and expression analyses of t he signalling pathways that regulate skeletal muscle trophism and innervation.

KEY PUBBLICATIONS
Studies on the role of apoptosis in the pathogenesis of the exercise-induced skeletal muscles damage and in the induction of autoimmune response in SLE.

1. Zampieri S, Degen W, Ghirardello A, Doria A, van Venrooij WJ. Dephosphorylation of autoantigenic ribosomal P proteins during Fas-L induced apoptosis: a possible trigger for the development of the autoimmune response in patients with systemic lupus erythematosus. Ann Rheum Dis 2001; 60: 72-76.

2. Podhorska-Okolow M, Sandri M, Zampieri S, Brun B, Rossini K, Carraro U. Apoptosis of myofibres and satellite cells: exercise-induced damage in skeletal muscle of the mouse. Neuropathol Appl Neurobiol 1998; 24: 518-31.

Characterization and serological detection of antibodies in autoimmune diseases.

1. Zampieri S, Ghirardello A, Rossini K, Iaccarino L, Bassi N, Atzeni F, Sarzi-Puttini P, Doria A. Antigen preparation for immunological studies in systemic autoimmune diseases. Ann NY Acad Sci 2007; 1109: 193-202.
2. Zampieri S, Ghirardello A, Iaccarino L, Tarricone E, Gambari PF, Doria A. Anti-Jo-1 antibodies. Autoimmunity 2005; 38: 73-78.
3. Zampieri S, Mahler M, Blüthner M, Qiu Z, Malmegrim K, Ghirardello A, Doria A, van Venrooij WJ, Raats JMH. Recombinant anti-P proteins autoantibodies isolated from a human autoimmune library: reactivity, specificity and epitope recognition. Cell Mol Life Sci 2003; 60:588-98.
4. Zampieri S, Ghirardello A, Doria A, Tonello M, Bendo R, Rossini K, Gambari PF. The use of Tween 20 in immunoblotting assays for the detection of autoantibodies in connective tissue diseases. J Immunol Methods 2000; 239: 1-11.

Clinical and histopathological characterization of the paraneoplastic forms of myopathies

1. Zampieri S, Valente M, Adami N, Biral D, Ghirardello A, Rampudda ME, Vecchiato M, Sarzo G, Corbianco S, Kern H, Carraro U, Bassetto F, Merigliano S, Doria A. Polymyositis, dermatomyositis and malignancy: A further intriguing link. Autoimmun Rev 2010; 9: 449-53.
2. Zampieri S, Doria A, Adami N, Biral D, Vecchiato M, Savastano S, Corbianco S, Carraro U, Merigliano S. Subclinical myopathy in patients affected with newly diagnosed colorectal cancer at clinical onset of disease: evidence from skeletal muscle biopsies. Neurol Res, 2010, 32: 20-5.

Characterization of the structure and function of long term denervated skeletal muscles in patients affected with *Conus Cauda* lesion and in the animal model of denervated adult rats.

1. Mancinelli R, Kern H, Fulle S, Carraro U, Zampieri S, La Rovere R, Fanò G, Pietrangelo T. Transcriptional profile of denervated vastus lateralis muscle derived from a patient 8 months after spinal cord injury: a case-report. Int J Immunopathol Pharmacol. 2011; 24: 749-59.
2. Kern H, Carraro U, Biral D, Adami N, Zampieri S. Severely atrophic muscle fibers with nuclear clumps survive many years in permanently denervated human muscle. The Open Pathology Journal 2009; 3:106-110.
3. Squecco R, Carraro U, Kern H, Pond A, Adami N, Biral B, Vindigni V, Boncompagni S, Pietrangelo T, Bosco G, Fanò G, Marini M, Abruzzo PM, Germinario E, Danieli-Betto D, Protasi F, Francini F, Zampieri S. A sub-population of rat muscle fibers maintains an assessable excitation-contraction coupling mechanism after long-standing denervation, despite lost contractility J Neuropathol Neurobiol 2009; 68: 1256-68.

Studies on the physiopathology of skeletal muscles during denervation, ageing and cancer cachexia: role of physical exercise in the recovery of muscle atrophy

1. Kern H, Carraro U, Adami N, Biral D, Hofer C, Forstner C, Modlin M, Vogelauer M, Pond A, Boncompagni S, Paolini C, Mayer W, Protasi F, Zampieri S. Home-based Functional Electrical Stimulation (h-bFES) rescues permanently denervated muscles

in paraplegic patients with complete lower motor neuron lesion Neurorehab Neural Re 2010; 24:709-21.
2. Zampieri S, Pietrangelo L, Loefler S, Fruhmann H, Vogelauer M, Burggraf S, Pond A, Grim-Stieger M, Cvecka J, Sedliak M, Tirpáková V, Mayr W, Sarabon N, Rossini K, Barberi L, De Rossi M, Romanello V, Boncompagni S, Musarò A, Sandri M, Protasi F, Carraro U, Kern H. Lifelong Physical Exercise Delays Age-Associated Skeletal Muscle Decline. J Gerontol A Biol Sci Med Sci. 2014 Feb 18.
3. Mosole S, Carraro U, Kern H, Loefler S, Fruhmann H, Vogelauer M, Burggraf S, Mayr W, Krenn M, Paternostro-Sluga T, Hamar D, Cvecka J, Sedliak M, Tirpakova V, Sarabon N, Musarò A, Sandri M, Protasi F, Nori A, Pond A, Zampieri S. Long-term high-level exercise promotes muscle reinnervation with age. J Neuropathol Exp Neurol. 2014; 73: 284-94.
4. Kern H, Barberi L, Löfler S, Sbardella S, Burggraf S, Fruhmann H, Carraro U,Mosole S, Sarabon N, Vogelauer M, Mayr W, Krenn M, Cvecka J, Romanello V, Pietrangelo L, Protasi F, Sandri M, Zampieri S, Musaro A. Electrical stimulation counteracts muscle decline in seniors. Front Aging Neurosci. 2014 Jul 24;6:189.
5. Pigna E, Berardi E, Aulino P, Rizzuto E, Zampieri S, Carraro U, Kern H, Merigliano S, Gruppo M, Mericskay M, Li Z, Rocchi M, Barone R, Macaluso F, Di Felice V, Adamo S, Coletti D, Moresi V. Aerobic Exercise and Pharmacological Treatments Counteract Cachexia by Modulating Autophagy in Colon Cancer. Sci Rep. 2016 May 31;6:26991.
6. Zampieri S, Mammucari C, Romanello V, Barberi L, Pietrangelo L, Fusella A, Mosole S, Gherardi G, Höfer C, Löfler S, Sarabon N; Cvecka J, Krenn M, Carraro U, Kern H, Protasi F, Musarò A,Sandri M, and Rizzuto R. Physical exercise in aging human skeletal muscle increases Mitochondrial Calcium Uniporter (MCU) expression levels and affects mitochondria dynamics. Physiol Rep 2016, Dec;4(24). pii: e13005.
7. Tezze C, Romanello V, Desbats MA, Fadini GP, Albiero M, Favaro G, Ciciliot S, Soriano ME, Morbidoni V, Cerqua C, Loefler S, Kern H, Franceschi C, Salvioli S, Conte M, Blaauw B, Zampieri S, Salviati L, Scorrano L, Sandri M. Age-Associated Loss of OPA1 in Muscle Impacts Muscle Mass, Metabolic Homeostasis, Systemic Inflammation, and Epithelial Senescence. Cell Metab. 2017 Jun 6;25(6):1374-1389.e6.
8. Bolotta A, Filardo G, Abruzzo PM, Astolfi A, De Sanctis P, Di Martino A, Hofer C, Indio V, Kern H, Löfler S, Marcacci M, Zampieri S, Marini M, Zucchini C. Skeletal Muscle Gene Expression in Long-Term Endurance and Resistance Trained Elderly. Int J Mol Sci. 2020 Jun 2;21(11): E3988.

Personal info

Date and place of birth 11th August 1972, Padova.
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Current work place

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Qualifications
1991 Classical studies at the Tito Livio high school in Padua
1996 Degree in Biological Sciences (five-year degree), pathophysiological address at the University of Padua
1998 Qualification to the profession of Biologist
2002 PhD in Experimental Rheumatology at the University of Padua

National Scientific Qualification
Qualified as 2nd level Professor, SSD MED / 04, valid from 31/05/2021 to 31/05/2030 (art.16, paragraph 1, Law 240/10)
Qualified as 2nd level Professor, SSD BIO / 09, valid from 23/07/2021 to 23/07/2030 (art.16, paragraph 1, Law 240/10)

Current job position
Type A researcher at the Department of Surgical, Oncological and Gastroenterological Sciences, University of Padua.

Current research topics
Study of the pathophysiology of skeletal muscle, with particular reference to the conditions of denervation atrophy in aging sarcopenia and neoplastic cachexia. Specifically, the research activity aims at analyzing muscle morphology and the expression of the signal pathways involved in the regulation of trophism and muscle function in relation to rehabilitation approaches through functional electrical stimulation and physical exercise.

Educational activities
9 years currently holder of the teaching of GENERAL PATHOLOGY at the Degree Course in *"Health Care"*, Università degli Studi di Padova.

11 years (continuous): Contract Professor for the teaching of GENERAL PATHOLOGY at the Degree Course in "Nursing", University of Padua.
3 years (continuous) as holder of supplementary teaching contracts at the School of Specialization in Rheumatology of the University of Padua and in the degree courses in "Dentistry and Dental Prosthetics" and in "Nursing" at the University of Padua.

Scientific projects
She has participated in the drafting and implementation of numerous projects and study protocols:
- 1998-2000 National study project: "Autoantibodies and organ damage in autoimmune diseases systemic ". Co-financing of the Ministry of University and Scientific Research (MURST).
- 2000-2004 National study project: "Anti-SSA / Ro antibodies and cardiac electrophysiological anomalies: prospective multicentre study on the appearance of a prolongation of the QT interval in children of women with anti-SSA / Ro autoantibodies and development of models experimental tests for the evaluation of the pathogenetic role of anti-SSA / Ro in the induction of electrocardiographic anomalies. "Co-funded by the Ministry of University and Scientific Research (MURST).

- 2004-2006 National study project: “Autoimmunity and anomalies of the sex chromosomes: instability of the sex chromosomes in patients with poly-dermatomyositis and other connective tissue diseases and in their relatives”. Co-financing of the Ministry of University and Scientific Research (MURST).
- 2006-2008 National study project: "Relationship between genetic polymorphisms and systemic and in situ phenotypic expression in the pathogenesis of rheumatoid arthritis and primary synovitis: role of cell death by apoptosis". Co-financing of the Ministry of University and Scientific Research (MURST).
- 2007-2013 EU Program INTERREG IVa (European Regional Development Fund - Cross Border Cooperation Program Slovakia - Austria (Interreg-IVa), project Mobility in Elderly, MOBIL, N_00033). - 2013-2016 EU Program INTERREG IVa Mobility in elderly MOBIL, N_00033-elongation
- 2015-2020 "CONTROLLING BMP / MUSA1 AXIS TO PREVENT CANCER CACHEXIA". Study protocol for the collection of muscle biopsies and peripheral blood from patients suffering from colic, esophagus-gastric and pancreatic neoplasia (Classified according to TNM classification -UICC 7th Ed. 2010) undergoing treatment at the Surgical Clinic 3 of the Padua Hospital. Reference code AOP0696 (Prot. N. 3674 / AO / 15).
- 2017-2020 R.I.C.A.V.O.Project (Intensive Rehabilitation of Brain Injuries and Honey Injuries Acquired in Eastern Veneto). Development of a regional model of "vertical" type rehabilitation network for people affected by acquired lesion of the central nervous system and creation of an IT platform for the collection of data for the identification of prognostic and prognostic factors of recovery identified through the analysis of specific circulating and tissue biomarkers.

Book chapters

1. Zampieri S, Ghirardello A, Iaccarino L, Gambari PF, Doria A. Infections in Polymyositis and Dermatomyositis. In: Infection and Autoimmunity. Shoenfeld Y, Rose NR. eds. Elsevier, Amsterdam 2004. Pg 583-90.
2. Doria A, Iaccarino L, Ghirardello A, Briani C, Zampieri S, Tincani A, Gambari PF. Pregnancy in rheumatoid arthritis, Sjögren syndrome and other rare autoimmune rheumatic diseases. In: Reproductive and hormonal aspects of systemic autoimmune diseases. M. Lockshin, DW Branch eds. Elsevier, Amsterdam 2006. Pg 77-93.
3. Doria A, Briani C, Ghirardello A, Zampieri S, Sarzi-Puttini P, Rondinone R. Dermatomyositis. In: Diagnostic criteria in Autoimmune diseases. Shoenfeld Y, Cervera R, Gershwin ME eds. Humana Press, USA. 2008. Pg 153-58.
4. Gargiulo P, Carraro U, Mandl T, Kern H, Zampieri S, Mayr W, Helgason T. Anthropometry of Human Muscle Using Segmentation Techniques and 3D Modelling: Applications to Lower Motor Neuron Denervated Muscle in Spinal Cord Injury. In: Handbook of Anthropometry. Physical Measures of Human Form in Health and Disease. Preedy VR eds. Springer US. 2012. Pg. 323-54.

Educational publications

1. Carraro U, Sandri S, Zampieri S. “*Patologia generale del tessuto muscolare scheletrico*”. In “Patologia Generale”. Pontieri GM, Russo MA, Frati L. Piccin, Padova. V edizione
2. Carraro U, Zampieri S. “*Risposte integrate e sistemiche. Immunologia ed Immunopatologia*” In “Principi di Patologia Generale” Carraro U, Unipress, Padova.

3. Zampieri S. *"La cellula ed il sistema immunitario: organizzazione e funzioni".* In "Il Lupus: la malattia dai mille volti". Doria A, Rondinone R. GPAnet, Milano.

Scientific publications

(PubMed indexed journals) IF Total (ISI 2016) 370.005 h- index (Scopus) 33 IF (ISI 2016) and citations (Scopus) are listed below individually for each article.

1. Arpesella G, Carraro U, Mikus P, Dozza F, Lombardi P, Marinelli G, Zampieri S. El Messlemani AH, Rossigni K, Pierangeli A. Activity-rest stimulation of Latissimus Dorsi for cardiomyoplasty: 1-year results in Sheep Ann Thorac Surg 1998; 66: 1983-90. IF 3.700, Citations 23
2. Podhorska-Okolow M, Sandri M, Zampieri S, Brun B, Rossini K, Carraro U. Apoptosis of myofibres and satellite cells: exercise-induced damage in skeletal muscle of the mouse. Neuropathol Appl Neurobiol 1998; 24: 518-31. IF 5.347, Citations 88
3. Zampieri S, Ghirardello A, Doria A, Tonello M, Bendo R, Rossini K, Gambari PF. The use of Tween 20 in immunoblotting assays for the detection of autoantibodies in connective tissue diseases J Immunol Methods 2000; 239: 1-11. IF 2.100, Citations 34
4. Ghirardello A, Doria A, Zampieri S, Gerli R, Rapizzi E, Gambari PF. Anti-ribosomal P protein antibodies detected by immunoblotting in patients with connective tissue diseases: their specificity for SLE and association with IgG anticardiolipin antibodies. Ann Rheum Dis 2000; 59: 975-81. IF 12.811, Citations 65
5. Zampieri S, Degen W, Ghirardello A, Doria A, van Venrooij WJ. Dephosphorylation of autoantigenic ribosomal P proteins during Fas-L induced apoptosis: a possible trigger for the development of the autoimmune response in patients with systemic lupus erythematosus. Ann Rheum Dis 2001; 60: 72-76. IF 12.811, Citations 44
6. Ghirardello A, Doria A, Zampieri S, Gambari PF, Todesco S. Autoantibodies to ribosomal P proteins in systemic lupus erythematosus. Isr Med Assoc J 2001; 3: 854-57. IF 1.036, Citations 9
7. Doria A, Cutolo M, Ghirardello A, Zampieri S, Vescovi F, Sulli A, Giusti M, Piccoli A, Grella P Gambari PF. Steroid hormones and disease activity during pregnancy in systemic lupus eythematosus. Arthritis Rheum 2002; 47: 202-09. (adsorbed by Arthritis Care Res in 2010) IF 3.319, Citations 94
8. Ghirardello A, Caponi L, Franceschini F, Zampieri S, Gambari PF, Doria A. Diagnostic tests for anti-ribosomal P protein antibodies: a comparative evaluation of immunoblotting and ELISA assays. J Autoimmunity 2002; 19: 71-77. IF 7.641, Citations 32
9. Zampieri S, Mahler M, Blüthner M, Qiu Z, Malmegrim K, Ghirardello A, Doria A, van Venrooij WJ, Raats JMH. Recombinant anti-P proteins autoantibodies isolated from a human autoimmune library: reactivity, specificity and epitope recognition. Cell Mol Life Sci 2003; 60:588-98. IF 5.788, Citations 21
10. Doria A, Shoenfeld Y, Wu R, Gambari PF, Puato M, Ghirardello A, Gilburd B, Corbanese S, Patnaik M, Zampieri S, Peter JB, Favaretto E, Iaccarino L, Sherer Y, Todesco S, Pauletto P. Risk factors for subclinical atherosclerosis in a prospective cohort of patients with systemic lupus erythematosus. Ann Rheum Dis. 2003; 62 :1071-77. IF 12.811, Citations 302
11. Ghirardello A, Doria A, Zampieri S, Tarricone E, Tozzoli R, Villalta D, Bizzaro N, Piccoli

A, Gambari PF. Antinucleosome antibodies in SLE: a two-year follow-up study of 101 patients. J Autoimmunity 2004; 22: 235-40. IF 7.641, Citations 73

12. Doria A, Ghirardello A, Iaccarino L, Zampieri S, Punzi L, Tarricone E, Ruffatti A, Sulli A, Sarzi-Puttini PC, Gambari PF, Cutolo M. Pregnancy, cytokines, and disease activity in systemic lupus erythematosus. Arthritis Rheum. 2004; 51:989-95. (adsorbed by Arthritis Care Res in 2010) IF 3.319, Citations 76
13. Briani C, Zara G, Rondinone R, Della Libera S, Ermani M, Ruggero S, Ghirardello A, Zampieri S, Doria A. Thalidomide neurotoxicity: prospective study in patients with lupus erythematosus. Neurology 2004; 62: 2288-90. IF 7.592, Citations 54
14. Rinaldi S, Doria A, Salaffi F, Ermani M, Iaccarino L, Ghirardello A, Zampieri S, Sarzi-Puttini P, Gambari PF, Perini G. Health-related quality of life in Italian patients with systemic lupus erythematosus. I. Relationship between physical and mental dimension and impact of age. Rheumatology (Oxford). 2004; 43: 1574-79. IF 4.818, Citations 67
15. Doria A, Rinaldi S, Ermani M, Salaffi F, Iaccarino L, Ghirardello A, Zampieri S, Della Libera S, Perini G, Todesco S. Health-related quality of life in Italian patients with systemic lupus erythematosus. II. Role of clinical, immunological and psychological determinants. Rheumatology (Oxford). 2004; 43: 1580-86. IF 4.818, Citations 95
16. Doria A, Iaccarino L, Ghirardello A, Briani C, Zampieri S, Tarricone E, Gambari PF. Pregnancy in rare autoimmune rheumatic diseases: UCTD, MCTD, myositis, systemic vasculitis and Bechet disease. Lupus. 2004; 13: 690-95. IF 2.454, Citations 27
17. Zampieri S, Ghirardello A, Iaccarino L, Tarricone E, Gambari PF, Doria A. Anti-Jo-1 antibodies. Autoimmunity. 2005; 38: 73-78. IF 2.629, Citations 55
18. Ghirardello A, Zampieri S, Iaccarino L, Tarricone E, Gambari PF, Doria A. Anti-Mi-2 antibodies. Autoimmunity
19. Zampieri S, Alaibac M, Iaccarino L, Rondinone R, Ghirardello A, Sarzi-Puttini P, Peserico A, Doria A. TNF-□ is expressed n refractory skin lesions from subacute cutaneous lupus erythematosus patients. Ann Rheum Dis 2006; 65: 545-548. IF 12.811, Citations 44
20. Zampieri S, Iaccarino L, Ghirardello A, Tarricone E, Arienti S, Sarzi-Puttini P, Gambari P, Doria A. Systemic lupus erythematosus, atherosclerosis, and autoantibodies. Ann NY Acad Sci. 2005; 1051:351-61. IF 4.706, Citations 44
21. Briani C, Zara G, Rondinone R, Iaccarino L, Ruggero S, Toffanin E, Ermani M, Ghirardello A, Zampieri S, Sarzi- puttini P, Doria A. Positive and negative effects of thalidomide on refractory cutaneous lupus erythematosus. Autoimmunity 2005; 38: 549-55. IF 2.629, Citations 16
22. Rinaldi S, Ghisi M, Iaccarino L, Zampieri S, Ghirardello A, Sarzi-Puttini P, Ronconi L, Perini G, Todesco S, Sanavio E, Doria A. Influence of coping skills on health-related quality of life in patients with systemic lupus erythematosus. Arthritis Rheum 2006; 55: 427-33. (adsorbed by Arthritis Care Res in 2010) IF 6.918, Citations 32
23. Drosera M, Facchetti F, Landolfo S, Mondini M, Nyberg F, Parodi A, Santoro A, Zampieri S, Doria A. Role of soluble and cell surface molecules in the pathogenesis of autoimmune skin diseases. Clin Exp Rheumatol 2006; 24: S7-S13. IF 2.643, Citations 8
24. Doria A, Iaccarino L, Arienti S, Ghirardello A, Zampieri S, Rampudda ME, Cutolo M, Tincani A, Todesco S. Th2 immune deviation induced by pregnancy: The two faces of autoimmune rheumatic diseases. Reprod Toxicol 2006; 22: 234:41. IF 2.341, Citations

97
25. Ghirardello A, Zampieri S, Tarricone E, Iaccarino L, Bendo R, Briani C, Rondinone R, Sarzi-Puttini P, Todesco S, Doria A. Clinical implications of autoantibody screening in patients with autoimmune myositis. Autoimmunity 2006; 39:217-21. IF 2.629, Citations 65
26. Zampieri S, Ghirardello A, Iaccarino L, Briani C, Sarzi-Puttini P, Atzeni F, Arienti S, Todesco S, Doria A. Polymyositis-dermatomyositis and infections. Autoimmunity 2006; 39: 191-96. IF 2.629, Citations 32
27. Doria A, Iaccarino L, Ghirardello A, Zampieri S, Arienti S, Sarzi-Puttini P, Atzeni F, Piccoli A, Todesco S. Long- term prognosis and causes of death in systemic lupus erythematosus. Am J Med 2006; 119:700-06. IF 5.550, Citations 182
28. Doria A, Iaccarino L, Sarzi-Puttini P, Ghirardello A, Zampieri S, Arienti S, Cutolo M, Todesco S. Estrogens in pregnancy and systemic lupus erythematosus. Ann NY Acad Sci 2006; 1069:247-56. IF 4.706, Citations 51
29. Tarricone E, Ghirardello A, Zampieri S, Elisa RM, Doria A, Gorza L. Cell stress response in skeletal muscle myofibers. Ann NY Acad Sci 2006; 1069:472-76. IF 4.706, Citations 8
30. Briani C, Doria A, Marcolongo R, Tognon S, Ruggero S, Toffanin E, Ermani M, Ghirardello A, Zampieri S, Semenzato G. Increased titers of IgM anti-heparan sulfate antibody in Behcet's disease. Clin Exp Rheumatol 2006; 24: S104-07. IF 2.643, Citations 8
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CHAPTER 15.1.Pupils
15.1.3. Katia Rossini

1994 Katia Rossini 2020

Dr. Katia Rossini, Neuro and Psychomotricity Therapist for the Developmental Age

Dedicated and experienced Neuro and Psychomotricity Therapist with over ten years of experience in neuropsychomotor assessment, in identifying the rehabilitation needs of the child. I carry out evaluation and rehabilitation of neurological and neuropsychological disorders of the developmental age; evaluation and rehabilitation of neuropsychomotor disorders of the developmental age; assessment and rehabilitation of autism spectrum disorders.

I earned:
Degree in Biological Sciences at the University of Padua in 1994 and I devoted myself to research on muscle pathologies at the Applied Myology laboratory of prof. U. Carraro of the Department of Biomedical Sciences of the University of Padua, from 1995 to 2012 dealing with the development of analytical methods for the study of the mechanisms of muscle plasticity, monitoring of trophism and muscle damage in physical exercise, in progression of muscular dystrophy, in functional rehabilitation by electrostimulation in patients with complete flaccid paraplegia.
The results of these studies have been communicated in conferences and / or published in international journals available at the site: http://www.ncbi.nlm.nih.gov/sites/entrez?db=pubmed.

Between 1998 and 2012 I also was Contract Professor for the teaching of General Pathology at the Degree Course in "Nursing", University of Padua.

My first professional experience led me to approach the world of rehabilitation and, since I have always been passionate about the world of childhood, I chose a new cycle of university studies that allowed me to reconcile the passion for rehabilitation and that for the development of the child and thus I achieved:

- Degree in Neuro and Psychomotricity Therapy for the Developmental Age at the University of Padua in 2009 with honors. Thesis entitled "Eating difficulties in early childhood: the role of the neuro and psychomotor therapist". Registered with no. 4 in the register of the health profession of Neuro and Psychomotricity Therapist of the Developmental Age at the TSRM PSTRP order of Venice-Padua.

  The interest in a global vision of the person and in the role of "contact" in the therapeutic relationship as well as the possibility of continuing the path of personal growth that passes through the awareness of one's physical, mental and emotional body leads me to undertake a three-year course of Shiatsu and to become:
- Shiatsu Professional Operator in 2016, since then I have been registered with n. 005910 to the Professional Association of Shiatsu Operators and Teachers (APOS) and I continued my training by participating in various professional refresher and specialization seminars.

  In continuous training, for over 10 years I have been dealing with neuropsychomotor evaluation and rehabilitation of children from the age of a few months to eight to nine years, in particular with:
- delay in psychomotor development
- cognitive deficits
- autism spectrum disorders
- Developmental Coordination Desorder and dyspraxia
- deficit of executive functions
- Attention-Deficit/Hyperactivity Disorder (ADHD)
- difficulty in recognizing the body pattern and lateralization
- difficulties in the affective-emotional, relational sphere.

I have been working as Neuro and Psychomotricity Therapist for the Developmental Age as freelancer since 2010 at Coop. Sociale CRESCENDO, Albignasego (PD) and from 2019 at the Piccoli Passi Studio in Vigonza (PD). From 2012 to 2014 I held it at the Medical Center for Phoniatrics in Padua.

For several years I have been involved in educational-preventive group psychomotor activity in kindergartens and preschools. From 2014 to 2018 I was in charge of the "Neuropsychomotricity in water" project for children with neuromotor disabilities, cognitive retardation and / or autism spectrum disorder at the Associazione Famiglie e Abilità Onlus, Bojon (VE). From 2016 to 2018 I was the contact person for the province of Padua of the AIDEE Veneto Region Group (Italian Association of Developmental Dyspraxia).

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CHAPTER 15.1. Pupils
15.1.4. Corrado Rizzi

Curriculum of Corrado Rizzi

Born in Vicenza on February 12, 1963
Doctor in Medicine, Specialist in Plastic Surgery, PhD in Agroindustrial Biotechnology.
Assistant Professor of Food Science and Technology, Department of Biotechnology, University of Verona

Has attended several national and international scientific meetings. He has taken part in national research programs. He is the author of about 40 scientific notes. His research, in the last years, concerned the biochemistry of agro-food products, with particular attention to the study of both the proteins and the enzymes of alcoholic beverages and cereal products. He has applied the study of some enzymes, allergens of wheat flour and of the lectins in the bakery foods. At present his main interest is the use of oenological industry waste as a source of antioxidant and other nutritional helpful molecules

Research interests

Topic	**Description**	**Research area**
Food Biochemistry	Extraction, purification and characterization of food proteins. Research on digestibility and allergenicity of cereal based foodstuffs. Identification of hidden allergens in foods.	Food sciences
Food Science & Technology	Food product development. Process optimization. Valorization of by-products of the agro-food industries, using "green solvents" (Supercritical fluids; NADES - Natural Deep Eutectic Solvents) for the extraction and recovery of compounds of interest for the food, cosmetic and pharmaceutical industries. Food fortification.	Food sciences

Research interests

Topic	**Description**	**Research area**

Projects

Title	**Starting date**
Recovery of winemaking by-products for innovative food applications: production of functional Italian salami	4/1/19
Valorizzazione delle vinacce quali ingredienti funzionali per la formulazione di salumi	8/1/18
Valorizzazione di germoplasma di frumento duro, di nuova e antica costituzione, per proprietà salutistiche e tecnologiche	7/20/11
Messa punto di nuove strategie per lo sviluppo di vaccini per il trattamento dell'allergia al frumento	6/1/11
Valutazione del potenziale antinutrizionale ed allergenico di sfarinati e derivati di nuove e vecchie varietà di frumento duro	1/27/10
SVILUPPO DI MODELLI DI STUDIO PER LA VALUTAZIONE DELLA SICUREZZA DEGLI ENZIMI UTILIZZATI NELLE FARINE DEI CEREALI	8/31/08
Studio comparativo in vitro dell'evoluzione degli allergeni durante le trasformazioni tecnologiche e i processi digestivi in frumenti convenzionali e transgenici	1/3/07
Studio comparativo in vitro dell'evoluzione degli allergeni durante le trasformazioni tecnologiche e i processi digestivi in frumenti convenzionali e transgenici. (2006)	1/1/06
Quantificazione degli inibitori delle alfa-amilasi di frumento nelle farine e nelle polveri ambientali	9/5/05
Caratterizzazione delle componenti molecolari delle farine di frumento e degli alimenti derivati dotate di attività biologica in vitro ed in vivo	11/15/04
STUDIO DEGLI EFFETTI DEI TRATTAMENTI DI STABILIZZAZIONE DEI VINI BIANCHI CON COADIUVANTI ALTERNATIVI	4/1/04
Studio delle proteine delle farine del frumento e degli alimenti derivati in relazione alla loro attività citotossica e alle interazioni con il sistema immunitario (2002)	1/1/02

Present Position: Assistant Professor
Academic sector: AGR/15 - FOOD SCIENCE AND TECHNOLOGY
Research sector (ERC): LS9_5 - Food sciences (including food technology, nutrition)
Office Ca' Vignal 1, Floor 2, Room 2.04 Telephone 045 802 7947
Email: corrado.rizzi@univr.it

Key Pubblications (From PubMed DataBase)

1. Olivieri M, Spiteri G, Brandi J, Cecconi D, Fusi M, Zanoni G, Rizzi C. Glucose/Ribitol Dehydrogenase and 16.9 kDa Class I Heat Shock Protein 1 as Novel Wheat Allergens in Baker's Respiratory Allergy. Molecules. 2022 Feb 11;27(4):1212. doi: 10.3390/molecules27041212. PMID: 35209002 Free PMC article.

2. Bianchi F, Lomuscio E, Rizzi C, Simonato B. Predicted Shelf-Life, Thermodynamic Study and Antioxidant Capacity of Breadsticks Fortified with Grape Pomace Powders.Foods. 2021 Nov 16;10(11):2815. doi: 10.3390/foods10112815. PMID: 34829095 Free PMC article.
3. Rainero G, Bianchi F, Rizzi C, Cervini M, Giuberti G, Simonato B. Breadstick fortification with red grape pomace: effect on nutritional, technological and sensory properties.J Sci Food Agric. 2022 Apr;102(6):2545-2552. doi: 10.1002/jsfa.11596. Epub 2021 Nov 3. PMID: 34676540 Free PMC article.
4. Rocchetti G, Rizzi C, Cervini M, Rainero G, Bianchi F, Giuberti G, Lucini L, Simonato B. Impact of Grape Pomace Powder on the Phenolic Bioaccessibility and on In Vitro Starch Digestibility of Wheat Based Bread.Foods. 2021 Feb 27;10(3):507. doi: 10.3390/foods10030507. PMID: 33673445 Free PMC article.
5. Tolve R, Simonato B, Rainero G, Bianchi F, Rizzi C, Cervini M, Giuberti G. Wheat Bread Fortification by Grape Pomace Powder: Nutritional, Technological, Antioxidant, and Sensory Properties.Foods. 2021 Jan 2;10(1):75. doi: 10.3390/foods10010075. PMID: 33401782 Free PMC article.
6. Simonato B, Tolve R, Rainero G, Rizzi C, Sega D, Rocchetti G, Lucini L, Giuberti G. Technological, nutritional, and sensory properties of durum wheat fresh pasta fortified with Moringa oleifera L. leaf powder. J Sci Food Agric. 2021 Mar 30;101(5):1920-1925. doi: 10.1002/jsfa.10807. Epub 2020 Sep 22. PMID: 32898294
7. Cisneros-Yupanqui M, Zagotto A, Alberton A, Lante A, Zagotto G, Ribaudo G, Rizzi C. Monitoring the antioxidant activity of an eco-friendly processed grape pomace along the storage. Nat Prod Res. 2021 Dec;35(24):6030-6033. doi: 10.1080/14786419.2020.1815741. Epub 2020 Sep 2. PMID: 32878452
8. Cisneros-Yupanqui M, Zagotto A, Alberton A, Lante A, Zagotto G, Ribaudo G, Rizzi C. Study of the phenolic profile of a grape pomace powder and its impact on delaying corn oil oxidation. Nat Prod Res. 2022 Jan;36(1):455-459. doi: 10.1080/14786419.2020.1777414. Epub 2020 Jun 19. PMID: 32552183
9. Rocchetti G, Rizzi C, Pasini G, Lucini L, Giuberti G, Simonato B. Effect of Moringa oleifera L. Leaf Powder Addition on the Phenolic Bioaccessibility and on In Vitro Starch Digestibility of Durum Wheat Fresh Pasta.Foods. 2020 May 14;9(5):628. doi: 10.3390/foods9050628. PMID: 32422925 Free PMC article.
10. Menin A, Zanoni F, Vakarelova M, Chignola R, Donà G, Rizzi C, Mainente F, Zoccatelli G. Effects of microencapsulation by ionic gelation on the oxidative stability of flaxseed oil. Food Chem. 2018 Dec 15;269:293-299. doi: 10.1016/j.foodchem.2018.06.144. Epub 2018 Jun 30. PMID: 30100437
11. Vakarelova M, Zanoni F, Lardo P, Rossin G, Mainente F, Chignola R, Menin A, Rizzi C, Zoccatelli G. Production of stable food-grade microencapsulated astaxanthin by vibrating nozzle technology. Food Chem. 2017 Apr 15;221:289-295. doi: 10.1016/j.foodchem.2016.10.085. Epub 2016 Oct 20. PMID: 27979204
12. Mainente F, Simonato B, Pasini G, Franchin C, Arrigoni G, Rizzi C. Hen egg white lysozyme is a hidden allergen in Italian commercial ciders. Food Addit Contam Part A Chem Anal Control Expo Risk Assess. 2017 Feb;34(2):145-151. doi: 10.1080/19440049.2016.1265673. Epub 2016 Dec 21. PMID: 27892783
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analysis. Food Chem. 2014 Dec 1;164:413-7. doi: 10.1016/j.foodchem.2014.05.051. Epub 2014 May 17. PMID: 24996352

14. Zoccatelli G, Sega M, Bolla M, Cecconi D, Vaccino P, Rizzi C, Chignola R, Brandolini A. Expression of α-amylase inhibitors in diploid Triticum species.Food Chem. 2012 Dec 15;135(4):2643-9. doi: 10.1016/j.foodchem.2012.06.123. Epub 2012 Jul 14. PMID: 22980853
15. Dalla Pellegrina C, Perbellini O, Scupoli MT, Tomelleri C, Zanetti C, Zoccatelli G, Fusi M, Peruffo A, Rizzi C, Chignola R. Effects of wheat germ agglutinin on human gastrointestinal epithelium: insights from an experimental model of immune/epithelial cell interaction. Toxicol Appl Pharmacol. 2009 Jun 1;237(2):146-53. doi: 10.1016/j.taap.2009.03.012. Epub 2009 Mar 28. PMID: 19332085
16. Zoccatelli G, Dalla Pellegrina C, Consolini M, Fusi M, Sforza S, Aquino G, Dossena A, Chignola R, Peruffo A, Olivieri M, Rizzi C. Isolation and identification of two lipid transfer proteins in pomegranate (Punica granatum). J Agric Food Chem. 2007 Dec 26;55(26):11057-62. doi: 10.1021/jf072644x. Epub 2007 Nov 27. PMID: 18038997
17. Zoccatelli G, Dalla Pellegrina C, Mosconi S, Consolini M, Veneri G, Chignola R, Peruffo A, Rizzi C. Full-fledged proteomic analysis of bioactive wheat amylase inhibitors by a 3-D analytical technique: Identification of new heterodimeric aggregation states. Electrophoresis. 2007 Feb;28(3):460-6. doi: 10.1002/elps.200600348. PMID: 17203506
18. Dalla Pellegrina C, Rizzi C, Mosconi S, Zoccatelli G, Peruffo A, Chignola R. Plant lectins as carriers for oral drugs: is wheat germ agglutinin a suitable candidate? Toxicol Appl Pharmacol. 2005 Sep 1;207(2):170-8. doi: 10.1016/j.taap.2005.01.001. PMID: 16102568
19. Pellegrina CD, Padovani G, Mainente F, Zoccatelli G, Bissoli G, Mosconi S, Veneri G, Peruffo A, Andrighetto G, Rizzi C, Chignola R. Anti-tumour potential of a gallic acid-containing phenolic fraction from Oenothera biennis. Cancer Lett. 2005 Aug 8;226(1):17-25. doi: 10.1016/j.canlet.2004.11.033. Epub 2005 Jan 8. PMID: 16004929
20. Dalla Pellegrina C, Matucci A, Zoccatelli G, Rizzi C, Vincenzi S, Veneri G, Andrighetto G, Peruffo AD, Chignola R. Studies on the joint cytotoxicity of Wheat Germ Agglutinin and monensin.Toxicol In Vitro. 2004 Dec;18(6):821-7. doi: 10.1016/j.tiv.2004.04.008. PMID: 15465648
21. Zoccatelli G, Dalla Pellegrina C, Vincenzi S, Rizzi C, Chignola R, Peruffo AD. Egg-matrix for large-scale single-step affinity purification of plant lectins with different carbohydrate specificities.
22. Vincenzi S, Zoccatelli G, Perbellini F, Rizzi C, Chignola R, Curioni A, Per Quantitative determination of dietary lectin activities by enzyme-linked immunosorbent assay using specific glycoproteins immobilized on microtiter plates.Protein Expr Purif. 2003 Jan;27(1):182-5. doi: 10.1016/s1046-5928(02)00590-9. PMID: 12510002
23. Rizzi C, Rossini K, Bruson A, Sandri M, Dal Belin Peruffo A, Carraro U. Fully reversible procedure for silver staining improves densitometry of complex mixtures of biopolymers resolved by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. Electrophoresis. 2002 Sep;23(19):3266-9. doi: 10.1002/1522-2683(200210)23:19<3266::AID-ELPS3266>3.0.CO;2-L.23(19):3266-9.

CHAPTER 15.1.Pupils
15.1.5. Vincenzo Vindigni

Vincenzo Vindigni 2020

Curriculum of Vincenzo Vindigni

Vincenzo Vindigni, M.D., Ph.D.
Professore Associato

Clinica di Chirurgia Plastica
Università di Padova
Direttore: Prof. F. Bassetto
Via Giustiniani n° 2, 35100 Padova

Data e luogo di nascita: 14 Febbraio 1971; Udine, Italia

e-mail: vincenzo.vindigni@unipd.it

POSITION TITLE: Associate Professor of Plastic Surgery

Degree 1997 Medicine
Diploma 2002 Plastic, Reconstructive and Aesthetic Surgery, University of Padova
Doctorate 2005 Tissue Engineering and Regenerative Medicine, University of Padova
Assistant Professor 2007 Plastic and Reconstructive Surgery, University of Padova, Italy
Associate Professor 2016 Plastic and Reconstructive Surgery, University of Padova, Italy

Chief of the Residency Program in Plastic, Reconstructive, and Aesthetic Surgery 2016
Plastic and Reconstructive Surgery, University of Padova, Italy

A. Personal Statement

AESTHETIC AND CLINICAL GENERAL

Prof. Vincenzo Vindigni is currently employed at the Clinic of Plastic and Reconstructive Surgery of Padova University Italy as Associate Professor. His main fields of clinical investigation are breast reconstruction, post-bariatric plastic surgery, and general plastic reconstructive surgery. He presented the results of his activities at EURAPS meeting 8 times (2002, 2007, 2009, 2012, 2015, 2018, 2019, 2020/21), and the last presentation was "The Posterior Arm Flap for Reshaping the Postbariatric Breast".

RESEARCH

Prof. Vincenzo Vindigni has 15 years' experience about tissue engineering and regenerative medicine, and his team has published numerous manuscripts in top journal regarding this field of investigation. A well-defined collaboration between University, Industrial Partner (Cutech) and Padua General Hospital has been drawn. Cutech is an Italian biotech company that offers screening services based on unique pre-clinical models for skin and related annexes (hair, sebaceous glands). It focus on ex-vivo human skin models based on a distinctive tissue culture, image analysis and bioinformation know-how developed during more than 15 years. A collaboration with National Research Council (CNR) Institute for the Dynamics of Environmental Processes (IDPA) (Chemical Analysis Group Venice) was also drawn allowing the obtaining of "FIRB – Innovation in Research" founds, to study the effects of silver on wounds.

B. Positions and Honors

- Best Presentation Award "Decellularized human skeletal muscle as biologic scaffold for reconstructive surgery" Porzionato A, Sfriso MM, Pontini A, Macchi V, Petrelli L, Bassetto F, Vindigni V, De Caro R. International Society for Matrix Biology – Inaugural Meeting of Matrix Biology Ireland, Galway 19-21 November 2014
- Best oral presentation 2011 (UPRAS AWARD): Preparation of a 3D scaffold derived from xonologous omentum for autologous recellularization G.Lago, L.Lancerotto, M. Sfriso, A. Porzionato, R. De Caro, F. Bassetto, V. Vindigni UPRAS 2011, Londra 26-27 Novembre 2011 (Best oral presentation)
- October 2011: Observer at Department of Plastic and Reconstructive Surgery of the University of California (field of interest microsurgery). Winner of a Fellowship by University of Padova.
- Winners of the best papers competition at the ECSAPS meeting in Pamplona, Spain, October 2011. Cairns WRL, Rigo C, Roman M, Munivrana I, Vindigni V, Azzena B, Barbante C. Characterization and the release kinetics of silver from four different dressings;
- "Young Investigator Award" al 5th Joint Meeting of the European Tissue Repair

Society and Wound Healing Society, Limoges - France; 25th - 29th August 2009. Vindigni V, Zavan B, Lepidi S, Cortivo R, Bassetto F, Abatangelo G. In vivo regeneration of microvascular pedicle;

- October 2009: Observer at Department of Plastic and Reconstructive Surgery of the University of California (field of interest microsurgery) – Winner of a Fellowship by University of Padova
- May 2008: Observer at Bernard O'Brien Institute of Microsurgery, Melbourne, Australia (field of interest microsurgery and regenerative medicine). Winner of a Fellowship by University of Padova.
- 01 November 2000 – 31 January 2001: winner of the university scholarship ERASMUS to attend the Institute of Plastic and Reconstructive Surgery, Innsbruck University School of Medicine. Field of investigation: Microsurgery.
- 10 August 1998 – 31 August 1998: Observer at Institute of Plastic and Reconstructive Surgery, Washington University School of Medicine, St. Louis, USA, Head of the Institute: Prof. S. Mackinnon. Field of investigation: Microsurgery of the Peripheral Nervous System.

C. Contributions to Science

The following publications were selected from among a total of 130 (H index 34):

AESTHETIC AND CLINICAL GENERAL

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2. Facchin F, Pagani A, Marchica P, Pandis L, Scarpa C, Brambullo T, Bassetto F, Vindigni V. The Role of Portable Incisional Negative Pressure Wound Therapy (piNPWT) in Reducing Local Complications of Post-bariatric Brachioplasty: A Case-Control Study. Aesthetic Plast Surg. 2021 Jan 22:1-7. doi: 10.1007/s00266-020-02122-1.
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D. Additional Information: Research Support and/or Scholastic Performance

Professional Societies & Organizations

Member, Italian Society of Plastic, Reconstructive and Aesthetic Surgery (SICPRE)
Member, Italian Society of Microsurgery (SIM)
Member, European Association of Plastic Surgeons (EURAPS)
Member, International Society of Aesthetic Plastic Surgery (ISAPS)
Member, World Society for Reconstructive Microsurgery (WSRM)
Member, International Society of Plastic Regenerative Surgeons (ISPRES)
International Member, American Society of Plastic Surgeons (ASPS)

CHAPTER 15.1.Pupils
15.1.6. Barbara Ravara

Barbara Ravara 2022

Department of Biomedical Sciences, Padua, Italy. From Left: Barbara Ravara, Alessandra Nori, Walter Giuriati, Sandra Furlan, Ugo Carraro, Dorianna Sandonà.

Curriculum of Barbara Ravara

Date and place of birth 18th October 1968, Padova

Current work place

University of Padova, Department of Biomedical Sciences
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Qualifications

1987 Scientific high school, at the S. PioX College in Treviso

1995 Degree in Biochemical Sciences, biophysic and biochemistry at the Department of Biology, University of Padova

Thesis Title: "Fluorescence studies in the evaluation of ultraviolet radiation damage on plants"

1996 Qualification to the profession of Biologist

2002 Specialist in Biochemistry and Clinical Chemisty at the University of Padova

Thesis Title: "Apoptosis of skeletal muscle fibers in rats with experimental heart failure: role of TNF-α and sphingosine"

Current job position

Research Collaborator at the Department of Biomedical Sciences, Interdipartimental Research Center of Myology (CIR- Myo), University of Padova

Scientific projects

1997 Scholarship funded by UILDM "Unione Italiana Lotta Distrofie Muscolari"

Foundation “Subcellular localization and distribution of protein Kinase C isoforms in skeletal and cardiac muscles” at the Department of Biomedical Sciences, University of Padova
Responsible of the project Prof. G. Salviati and Prof. S. Salvatori
1998-1999 Scholarship funded by Telethon Foundation “Myoblast- selective mitogen released by macrophage as a tool for muscleregeneration and gene teraphy” at the Interdipartimental Research Center of Myology (CIR- Myo) c/o Department of Biomediacal Sciences, University of Padova.
Responsible of project Dr. L. Dalla Libera and Dr. M. Cantini
1999-2002 Scholarship funded by by Sigma- Tau Foundation “Apoptosis and modifications in skeletal muscle in heart failure” at Interdipartimental Research Center of Myology (CIR- Myo) c/o Department of Biomedical Sciences, University of Padova.
Responsible of project Dr. L. Dalla Libera and Dr. G. Vescovo
2003- 2005 Collaboration agreement funded by Telethon Foundation “Inhibition of apoptosis of skeletal muscle fibers in heart failure as a therapeutical tool to antagonize muscle atrophy” at the Interdipartimental Research Center of Myology (CIR- Myo) c/o Department of Biomedical Sciences, University of Padova.
Responsible of project Dr. L. Dalla Libera and Dr. G. Vescovo
2006 Collaboration agreement funded by C. N. R National Research Council: “Study of the effects of carnitine on cardiac and skeletal muscle changes induced by mildronate” at the Interdipartimental Research Center of Myology (CIR- Myo) c/o Department of Biomedical Sciences, University of Padova.
Responsible of project Dr. L. Dalla Libera and Dr. G. Vescovo
2006- 2009 Collaboration agreement funded by A.S.I Italian Space Agency “Stress – response as a tool to monitor and to conteract progression of muscle atrophy” at the Interdipartimental Research Center of Myology (CIR- Myo) c/o Department of Biomedical Sciences, University of Padova.
Responsible of project Dr. L. Dalla libera and Prof. L. Gorza
2010- 2013 Collaboration agreement funded by A.S.I Italian Space Agency “Chaperon proteins in hypertrophy and heart failure” at the Interdipartimental Research Center of Myology (CIR- Myo) c/o Department of Biomedical Sciences, University of Padova.
Responsible of project Prof. L. Gorza
2014- 2022 Research Collaborator at the Translational Mobility Medicine Laboratory of Interdipartimental Research Center of Myology (CIR- Myo) c/o Department of Biomediacal Sciences, University of Padova under the supervision of Prof. U. Carraro and H. Kern research projects: Mobility in Aging; Centre of Active Aging; Ambulant Remobilisation after Knie- Tep and Hip- Tep Implantation.

Scientific Pubblications

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L. Improvment in walking distance after rehabilitation in patients with peripherical arterial disease is associated with changes in skeletal muscle myosin heavy chains. Basic Applied Myology 10 (5): 231-236, 2000

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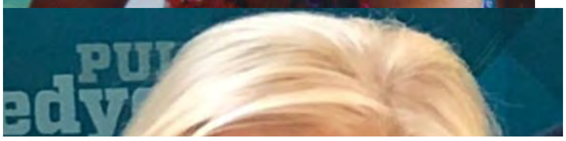
Chapter 15.1.7

More pupils … Alessandro Salviati, Nicoletta Adami, Anna Jakubiec-Puka, Donatella Biral, Marzena Podhorska-Okolow

2006
From left:
Alessandro Salviati, Nicoletta Adami, Anna Jakubiec-Puka, Ugo Carraro, Donatella Biral.

I cannot conclude my book without thanking other students and scientists who have helped my group achieve the long list of achievements described in chapters 2 through 10 of this book. The list should be longer, but unfortunately I have only a few pictures and papers (mostly their names in my PubMed publications list) of most of them. Luckily, I found the photo above taken in my lab in 2006. I'll start from the left, saying that Alessandro Salviati spent just two years in the lab trying to replicate previous results on muscle rebuilding after serious injuries. Alessandro was already an expert plastic surgeon, but not all dreams/projects come true and so it happened to him (as it happened to me a few years after my degree in Medicine and Surgery). The next young woman is Nicoletta Adami, a graduate in Biological Sciences who became Katia Rossini's expert substitute, [1-9] when Katia decided to work at the Institute of Physiology and then to become a Neuro and Psychomotrician Therapist (see Chapter 15.1.3. The elderly lady in the center of the photo is Anna Jakubiec-Puka, a researcher at the Nencki Institute of Biology in Warsaw, Poland, who took several sabbaticals every 3-5 years at the Institute of General Pathology and then at the Department of Biomedicine Sciences always in collaboration with Donatella Biral [10-16]. Donatella deserves a special mention because she was the first student of Biological Sciences, who prepared her thesis and published her first paper with my group. Subsequently she was hired as an Italian researcher of the CNR until her retirement. Looking at the list of references added to this sub-chapter, the reader will find that we have remained in contact, while officially collaborating with other Colleagues, in particular with the late Prof. Giovanni Salviati, who in his first fifty years left his family and all of us sad.



In those years we had another visiting scientist, who is present among authors of the papers related to muscle apoptosis. She arrived early in the morning at a service station along the Padua-Bologna highway, from where I drove her to Padua in my car. Marzena Podhorska-Okolow had immediately set to work, she was a talented electron microscopist. The first evidence of muscle apoptosis was patiently collected by her by spending long days at the microscope [17,18]. She had also become a friend of Katia Rossini and Nicoletta Adami returning several times for periods of work, but also for vacation with her family. As clearly shown in her photo (kindly provided by Katia Rossini), she was gracious and kind to everyone. She hosted me twice in the Department of Human Morphology and Embryology, Faculty of Medicine, Wroclaw Medical University, Wroclaw, Poland maintaining an active and successful collaboration for years.

Chapter 15.1. Pupils

15.1.8. Maria Chiara Maccarone

Maria Chiara Maccarone 2022

I met a brilliant PhD student enrolled in the School of Physical Medicine and Rehabilitation of the University of Padua during the seminars that I offer to second year postgraduates. Maria Chiara was immediately interested in implementing my proposals for a series of simple and safe physical exercises that even elderly people can do in the morning before getting out of bed. As I explained in the presentation of this Chapter 15, Maria Chiara Maccarone is making one of my last dreams come true. She is explaining in simple words, much better than me, to sedentary elderly people the advantages they can get by changing their lifestyle by simply investing 10-20 minutes early in the morning to perform the 12 exercises of home-based Full-Body in-Bed Gym. Maria Chiara is managing this project with the help of postgraduates from the School of Specialization in Physical Medicine and Rehabilitation of the University of Padua, who attended my seminars during the 2022 autumn.
Curious readers of my book will find more about hbFBiBG in the references below, at the pages 166 and 167 of this book (Chapter 10. Muscle aging decay: Countermeasures by FES and Full-Body in-Bed Gym) and in the YouTube video dynamically presenting the series of exercises in bed at the 2018 link:
https://www.youtube.com/watch?v=N1RuG3371-Y&feature=youtu.be
and then 5 years later at:
https://www.youtube.com/watch?v=pcHKmxCLYFs&t=336s

I had been pursuing this goal for 5 years with scarce enrolments of old people.

I am confident they will succeed!

Maria Chiara is managing the Project: home-based Full-Body in-Bed Gym with the help of postgraduates, who during the Autumn of 2022 attended my seminars at the School of Specialization in Physical Medicine and Rehabilitation of the University of Padua, Italy.

From left to right: Gianluca Regazzo (in seated position), Rossella Jirillo, Irene Seno, Giacomo Casellato, Allegra Caregnato, Maria Chiara Maccarone, Alessandra Carriero, Hillary Veronese, Chiara Venturin, Elena Marigo, Claudia Finamoni and Giuseppina Masitto (in seated position).

Dr. Maria Chiara Maccarone graduated in Medicine and Surgery at the University of Padua in 2018, and is currently enrolled as a Resident Doctor at the School of Physical Medicine and Rehabilitation of the University of Padua, directed by Prof. Stefano Masiero. She is an active member of the research group coordinated by prof. Masiero. She recently won a PhD grant in Neuroscience with a research project related to adolescent idiopathic scoliosis and body schema. For years engaged in numerous research projects, she has produced numerous scientific papers published in national and international peer-reviewed journals in the field of rehabilitation and is the author of numerous chapters of books on Physical Medicine and Rehabilitation (in Italy and in Europe) currently in course of publication. She also carries out scientific review activities for the journals Frontiers in Rehabilitation Medicine, INQUIRY, International Journal of Biometeorology, Sport Science for Health, PLOS One and the European Journal of Translational Myology. She is also a member of the Editorial Board of the journals Balneo and PRM Research Journals and International Journal of Balneology and Health Resort Medicine since 2022. Since 2022 she have been Secretary of section no. 12 - Thermal Rehabilitation of the Italian Society of Physical and Rehabilitative Medicine (SIMFER). Dr. Maccarone is in fact active in the dissemination of scientific culture in relation to the applications of traditional spa interventions in rehabilitation and possible synergistic strategies between rehabilitation and spa treatments.

Curriculum of Maria Chiara Maccarone

M.D. and SURGEON N°06571
ORDER OF SURGEONS AND DENTISTRY DOCTORS OF VICENZA
Email: mariachiara.maccarone@phd.unipd.it
Italian nationality
Date of birth 08/03/1993

Current Position

PhD Student in Neuroscience, Department of Neuroscience, University of Padua
Resident doctor in Physical Medicine and Rehabilitation, University of Padua, Via Giustiniani 2, 35128 Padua

Education

2012-2018
University of Padua, Faculty of Medicine and Surgery
2008 – 2012
"G.B.Brocchi" Classical High School, Bassano del Grappa (VI)

Other educational activities

April 14, 2021	University of Padua - Training for operators on rodents and lagomorphs in user establishments Authorization to operate in animal enclosures and to carry out in vivo studies on animal models.
January 20, 2021	Shock wave training course, Padua, Italy Use of focal and radial shock waves in rehabilitation, applications and contraindications, use of a vibrating handpiece, fundamentals of ultrasound investigation.
December 2019	Hilterapia Advanced Course, Padua, Italy. Use of the HILT pulse, indications and contraindications, treatment parameters and methods, treatment protocols.
2015-2016	Extra-curricular course in Gender Medicine at the University of Padua
2015-2016	Extra-curricular course in Clinical Bioethics at the University of Padua

Activities in scientific journals

Member of the Editorial Board of Balneo and PRM Research Journal and International Journal of Balneology and Health Resort Medicine since 2022.

Scientific review activity for journals: Frontiers in Rehabilitation Sciences; INQUIRY; European Journal of Translational Myology; International Journal of Biometeorology; Sport Science for Health; Applied Sciences; PLOS One

Invited lectures & conference presentations

- Il cervello-intestino, Rosà (VI), Italy, 23 October 2013
- Romanian Congress of Rehabilitation, Physical Medicine and Balneology, Bucarest, Romania, 02 September 2020
- I International Congress on Water and Health - I Congreso Internacional Sobre Agua y Salud – Termatalia, Ourense (Galician) – Spain, 16-17 September 2020
- VI International Congress Spa Treatment, Moscow – Russia, 28 September

2020

- PREVENZIONE, CURA, RIABILITAZIONE: Terapie termali tra attualità e prospettive future, Levico Terme, Italy, 22 May 2021
- Padua Days of Muscle & Mobility Medicine, Abano Terme, Italy, 29 May 2021
- 45° ISMH Congress, Dax, France, 10-11 June 2021
- Romanian National Congress of Rehabilitation, Physical Medicine and Balneology, Bucarest, Romania, 01 September 2021
- 49° National Congress SIMFER 2021 Le radici del futuro, Milano, Italy, 28- 31 October 2021
- On-site Padua Days on Muscle and Mobility Medicine, Abano Terme (PD), Italy, 03 April 2022
- 1st International Congress for Minimal Invasive Orthopaedic Surgery, Thessaloniki, Greece, 13- 15 May 2022
- Aquatea – AQUAtic Therapeutic ExercisARQUS Research Focus Forum on Healthy aging from a multidisciplinary perspective, Vilnius, Lithuania, 27-29 June 2022
- VIII International Sanatorium-Resort Congress, Russia, 07 July 2022
- Romanian Congress of Physical and Rehabilitation Medicine & Balneology, Slanic Moldova and Techirghiol, Romania, 04 September 2022
- 36° Congresso Nazionale Società Italiana di Flebologia, San Benedetto del Tronto (AP), Italy, 30 settembre - 1/2 October 2022
- 46th ISMH World Congress, Salsomaggiore Terme (PR), Italy, 19-21 October 2022
- 50° Congresso Nazionale SIMFER, Acireale (CT), Italy, 23 – 26 October 2022

Other contributions to national and international congresses

- Tognolo L., Maccarone M.C. La riabilitazione in ambiente termale, 48° SIMFER National Congress, 02 December 2020
- Masiero S., Maccarone M.C., Regazzo G., Forcato B. Spas and rehabilitation: current events and future prospects, INAIL Patologie professionali e cure termali, conoscenze attuali e nuovi orizzonti, Ischia, Italy 30 June 2022 – 01 July 2022
- Maccarone M.C., Masiero S. Can the spa environment be an effective setting for rehabilitation? 36th National Congress of the Italian Society of Phlebology, San Benedetto del Tronto (AP), Italy, 30 September - 1/2 October 2022
- Maccarone M.C., Venturini E., Masiero S. Rehabilitation in Health Resort Medicine: what are the future perspectives? 46th ISMH World Congress, Salsomaggiore Terme (PR), Italy, 19-21 October 2022
- Maccarone M.C., Masiero S. The thermal rehabilitation: reality and perspectives, 73rd International Congress of Femtec, Castel San Pietro Terme (BO), Italy, 3-6 November 2022

Honors and awards

- 2021 First place at the III International Student Olympiad on Medical

Rehabilitation and Spa Treatment, Moscow, Russia

- 2019 Honor for the Young Rosatesi Graduates of the Municipality of Rosà (VI), Italy
- 2009 First place at the "Certamen Senecanum" in Bassano del Grappa (VI).
- 2006-2007 Scholarship for deceased donor and Alpine members, Municipality of Rosà (VI)
- 2006-2007 Scholarship and Certificate of Merit - Municipality of Rosà (VI) for the academic results achieved

Publications

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2. Maccarone M.C., Magro G., Solimene U., Masiero S. Spa therapy can improve quality of life in chronic musculoskeletal disorder subjects: a narrative review. Bulletin of rehabilitation medicine. 2020; 96 (2): 3-6. https://doi.org/10.38025/ 2078-1962-2020-96-2-3-6
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8. Barberio B., Zingone F., Marinelli C., Maccarone M.C., Savarino E.V. T04.01.3 real life comparison of different anti-tnf biologic therapies for ulcerative colitis treatment: a retrospective cohort study October 2020 Digestive and Liver Disease https://doi.org/10.1016/S1590-8658(20)30757-X
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1. Barberio B., Zingone F., D’incà R., Marinelli C., Maccarone M.C., Gubbiotti A., Cingolani L., Lorenzon G., Ghisa M., Savarino E.V. p668 real-life comparison of different anti-tnf biologic therapies for ulcerative colitis treatment: a retrospective cohort study ECCO 2020 European crohn's and colitis organisation 15th annual congress, Vienna 12 - 15 febbraio 2020
2. Masiero S., Magro G., Maccarone M. C. Real-life effectiveness of rehabilitation treatments in the Italian thermal environment on pain perception and quality of life of musculoskeletal disorders patients VIRTUAL ISPRM 2021 Congress “Furthering Rehabilitation in a New World” 12-15 giugno 2021
3. Masiero S., Maccarone M.C., Magro G. Health resort medicine and human immunity in the covid-19 era: a narrative review VIRTUAL ISPRM 2021 Congress “Furthering Rehabilitation in a New World” 12-15 giugno 2021
4. Masiero S., Maccarone M.C., Magro G. et al. Spa setting for rehabilitation treatments: an italian multicentre study evaluating pain and quality of life of patients suffering from musculoskeletal disorders International Congress of Biometeorology ICB 2021 21-22 settembre 2021
5. Maccarone M.C., Coraci D., Piccione F., Masiero S. Preventing disabilities in neurodegenerative diseases at the early stage: the role of exercise in thermal water The Lancet Summit: Presymptomatic Prevention and Treatment of Neurodegenerative Diseases 14-16 dicembre 2021
6. Maccarone M.C., Masiero S. The role of balneotherapy in elderly rheumatic patients with frailty: a literature update Poster accepted for oral presentation 2022 ISPRM 2022 World Congress, Lisbona, Portugal 03 – 07 luglio 2022
7. Maccarone M.C., Masiero S. Can exercise in thermal water play a role in the rehabilitation of neurological disorders? A literature review Poster accepted for oral presentation 2022 ISPRM 2022 World Congress, Lisbona, Portugal 03 – 07 luglio 2022
8. Coraci D., Romano M., Tognolo L., Maccarone M.C., Piccione F., Ragazzo L., Masiero S. Peripheral neuropathies after common organ transplantations. Literature review and the use of electrophysiological tests and ultrasound Abstracts of the Twelfth Annual Meeting of the Italian Association for the study of the Peripheral Nervous System (ASNP) 9–11 giugno 2022 Genova, Italy
9. Maccarone M.C., Venturini E., Menegatti E., Gianesini S., Masiero S. Lymphedema, water-based exercise and gender: a scoping review of current literature10th Congress of the International Society of Gender Medicine, 16-17 settembre 2022, Padova, Italia
10. Maccarone M.C., Masiero S. Short-time effects of spa-rehabilitative interventions on pain, mood and quality of life in musculoskeletal disorders, ACRM 99th Annual Conference, 9-11 novembre 2022, Chicago, Illinois, Archives of Physical Medicine and Rehabilitation 103(12):e142
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Chicago, Illinois

12. Maccarone M.C., Masiero S. Impact of COVID-19 Pandemic on Reorganization of a Rehabilitation Unit in Italy, ACRM 99th Annual Conference, 9-11 novembre 2022, Chicago, Illinois, Archives of Physical Medicine and Rehabilitation 103(12):e142

Chapter 15.1. Pupils
15.1.9. Roger Coletti

Roger H. Coletti 2022

Finally, added in PROOFS, I have to mention my last (for now…) pupil: Roger H. Coletti. The reader will decide whether he is my student or I am his student.

I find it easier to present him in his own words.

I am an interventional cardiologist, now retired from clinical practice. However, I had a long-standing interest in chronic muscle spasm and sought to discover what kept the muscles in chronic spasm ... I followed the pioneering work of Janet Travel and David Simon, but the actual cause of chronic muscle spasm was never fully elucidated. What follow is a chronology of how I came develop a treatment, the outcome data for the use of that procedure and the insights into muscle pathophysiology with proposed theories of cellular and electrophysiological abnormalities … One obstacle for clinical use is tied to the very benefit of the procedure, that is the absence of recurrence. Muscles in chronic spasm fully treated to eliminate SEA do not need a second injection for long term benefit. Financially, this may not be beneficial for the practioner unless compensated on an outcome basis. It should be great in countries with government health services, but may not be a hit in the US ... One indication for widespread use is the potential to limit the opioid crisis. None of the patients treated received any pain medication and a significant number that had been taking opioid medications were able to discontinue them.

I hope that his proposals are recognized internationally. It's more than enough for me to be a minor actor in this show.

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Short Curriculum of Roger H. Coletti

Born in New York City, attended Georgetown University completing a BA degree in 1967, completed a MA degree from Hofstra University in 1973, did one year of bench research at New York University Medical School in a PhD pharmacology curriculum but transferred to State University of New York School of Medicine at Downstate and completed a MD in 1977, completed a 3 year internship and residency in medicine from Nassau County Medical Center in 1980, completed a 2 year cardiology fellowship at Columbia University Medical center in 1982 and completed a one year interventional cardiology fellowship at Westchester County Medical center in 1983. He continued to work as an interventional cardiologist until 2017, board certified in Internal Medicine, Cardiology, Nuclear Cardiology, and Interventional Cardiology and was granted Fellowship status in Cardiology, Nuclear Cardiology, and Interventional Cardiology.

Research lines of Roger H. Coletti

- CPR, Intra-aortic balloon counterpulsation, Coronary blood flow, Procedural advances in coronary angioplasty and rotoablation.
- Skeletal muscle physiopathology, chronic muscle spasm, chronic pain (from 2007 to now)

Personal memories and reflections on Ugo and the Myology in Padova
With Ugo I shared my preliminary book contents and he saw the need for me to publish articles on the theory and practice associated with my findings. He was extraordinary generous with his time and supervision, without which my work would have had little chance of actually being seen in light of day. This led to recognition internationally of my work which now stands a chance of being put into practice. I owe him a debt of gratitude that I cannot repay.

On EJTM I've published the following full paper
Coletti RH. The ischemic model of chronic muscle spasm and pain. Eur J Transl Myol. 2022 Jan 18;32(1):10323. doi: 10.4081/ejtm.2022.10323. PMID: 35044134; PMCID: PMC8992665.

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Roger H. Coletti. Chronic Muscle Spasm and Pain: Discoveries in the Etiology, Identification and Treatment of Chronic Muscle Spasm and Resultant Chronic Pain. Originally published: September 28, 2022. Publisher: Dorrance Publishing Co. (14 October 2022) ASIN: B0BNLVZ3H9
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CHAPTER 15.2. *Ugo's Endless dreams for the future*

15.2.1. Validate non-invasive blood analyses to monitor anti- and pro-inflammatory Cytokines and Myokines via saliva and sweat collection. Them will increase acceptability and frequency of sampling, critical factors in evaluating the many transient effects of training and rehabilitation in early aging and aging.

15.2.2. Validate Home-based Full-Body in-Bed Gym (hbFBiBG) as a clinical accepted rehabilitation program of self-administered physical exercise learned during hospitalization and performed regularly by seniors at home to improve and maintain muscle strength, self-confidence and a better mood related to the new lifestyle.

15.2.3 Help Roger H. Coletti to validate by international clinical trials his EMG guided chemodenervation procedure of acquired chronic muscle spasm designated as CMECD®.

15.2.4 Increase to a decent value the Impact Factor of European Journal of Translational Myology. Clarivates will release next June 2023 the 2022 Impact Factor of the European Journal of Translational Myology (EJTM).

15.2.5. Attract new young basic scientists and doctors to Translational Mobility Medicine.

15.2.6 See that the University of Padua scientific conferences " 2023 On-site PADUA DAYS ON MUSCLE AND MOBILITY MEDICINE (2023 On-site Pdm$_3$)" held in Thermae of Euganean Hills, Padua, Italy will continue after I will be unable to motivate EJTM Friends.

15.2.7. To publish this book before my birthday on February 23, 2023!

Dear Reader,
I hope you enjoyed reading the book. Since I'm hoping to rewrite this for my 90th birthday, I'd be very grateful to anyone who could send me suggestions for improvements and bug fixes.

Ugo Carraro

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- Local Organizer of the annual international meeting: "PADUA DAYS ON MUSCLE AND MOBILITY MEDICINE (Pdm$_3$)"
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I summarized in this book my experiences as a teacher and scientist, who spent more than 60 years at the University of Padua, Italy. The readers will find in the 15 Chapters, the beginning of my research activities and the serendipitous events of my dedication to denervation-reinnervation and death-regeneration of skeletal muscles and their electrical stimulation in animal models and patients. On the way, other scientists and clinicians with my own interests contacted me. Some collaborations provided exciting results, the majority frustrations. Nevertheless, this is the normal ratio in Translational Studies from Basic Science to Medicine: many preliminary exciting results end in failure, in particular those more original and promising. The majority of my dreams ended in disappointments, but I continue to think that it is more than enough to have dreams and the great fortune to test them by rigorous scientific approaches. This is why the book will end, though I will be 80-years-young the February 23, 2023, with a short series of my unending dreams.
Here I thank the many young and old persons who inspired, supported and collaborated with me, including the editorial assistants of PAGEPress, the Italian publisher of the European Journal of Translational Myology. Some of the supporters, collaborators and pupils have been kind enough to send me their CVs upon my invitation. But many others I have not been able to contact them or have had very little to share with them other than unpleasant memories. The list is very long, but I hope that CLEUP will accept a few more pages. I apologies for any missing names. They weren't deliberate omissions.
Finally, thanks to CLEUP. Indeed, I am very happy to print this book with the Publisher of the Proceedings of the first International Scientific Conference that I organized in 1985 in Abano Terme, my hometown, a long time ago.

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Padua (Italy), January 15, 2023.