PADUA DAYS ON MUSCLE AND MOBILITY MEDICINE 2026

International Conference

March 3rd to 6th Euganean Thermae and Padua, Italy

PADUA DAYS ON MUSCLE AND MOBILITY MEDICINE 2026 ABSTRACT N. 062

ELSEVIER SYMPOSIUM ON BIOLOGY OF AGING UNPACKED: IMPLICATIONS FOR GEROSCIENCE AND HEALTHSPAN

MITOCHONDRIAL SIGNALING IN AGE-RELATED CONDITIONS

Anna Picca[1|2]

[1]*Department of Medicine and Surgery, LUM University, Casamassima, Italy;* [2]*Fondazione Policlinico Universitario "A. Gemelli" IRCCS, Rome, Italy.*

Biological aging reflects the progressive decline of cellular adaptive and resilience mechanisms, resulting in damage accumulation and functional impairment. Mitochondria play a central role in this process not only through bioenergetic failure, but also via altered mitochondrial quality and signaling that influences inflammation, stress responses, and cell fate decisions (1). Mitochondrial dysfunction is tightly interconnected with other hallmarks of aging, including telomere attrition, genomic instability, impaired proteostasis, chronic inflammation, and cellular senescence, complicating the identification of primary molecular drivers of age-related conditions. Emerging evidence highlights mitochondrial-derived signals—such as reactive oxygen species, mitochondrial DNA, metabolic intermediates, and mitochondria-derived vesicles—as key mediators of inter-organelle and intercellular communication during aging. In particular, extracellular vesicles carrying mitochondrial components have gained attention as accessible biomarkers that reflect mitochondrial stress, immune activation, and inflammatory signaling (2). While mitochondria-derived vesicles contribute to mitochondrial quality control under physiological conditions, their dysregulated release can promote innate immune activation and amplify pro-inflammatory pathways associated with the senescence-associated secretory phenotype. A comprehensive characterization of mitochondrial signaling pathways and their extracellular mediators offers new insights into the mechanisms underlying chronic low-grade inflammation and functional decline in aging (3). Understanding how mitochondrial communication becomes maladaptive in age-related conditions may reveal novel targets for interventions aimed at preserving cellular homeostasis and mitigating aging-associated pathologies.

Keywords: *cell quality, extracellular vesicles, mitochondria.*