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CIRCULATING ADIPONECTIN INCREASES IN HEALTHY INDIVIDUALS AFTER A CYCLING PROGRAM WITH NEUROMUSCULAR ELECTRICAL STIMULATION

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Physical activity is known to provide broad health benefits, including reducing the risk of metabolic syndrome, diabetes and obesity, and it is therefore recommended as a pillar of cardiovascular disease prevention (1). Exercise also modulates the secretion of metabolic hormones and adipokines, such as adiponectin. This hormone is mainly secreted from adipose tissue and contributes to energy homeostasis by stimulating fatty acid oxidation in skeletal muscle and by inhibiting hepatic glucose production (2). Although many studies have investigated adiponectin release following aerobic training (3,4,5), less is known about its modulation when neuromuscular electrical stimulation (NMES) is applied during exercise. In this study, we examined both acute and chronic responses of plasmatic adiponectin concentration following 6-week cycling program (14 sessions) with or without percutaneous NMES. Electrical stimulation was delivered by a novel technology of Adaptive Functional Electrical Stimulation Kinesitherapy (AFESK™) through the VIK8 device (AFESK™ technology, VIK8, VIKTOR S.r.l., Italy). Sixteen healthy, physically active males matched for V?O2peak and age were assigned to either a cycling group or an AFESK group. Both groups completed the same interval training: 4x5m intervals at 60% peak power output (PPO) (achieved during an incremental test to exhaustion) interspersed with 3m recovery at 40% PPO. In the AFESK group, the VIK8 device provided electrical stimulation synchronized with voluntary contraction of the targeted skeletal muscles on lower limbs. Blood samples were collected at baseline and 15 min, 24h and 48h after the first (S1) and the last (S14) training session. Plasma adiponectin levels were detected by ELISA assay. After S1, adiponectin significantly increased at 15 min in both groups (+8,6% in the cycling group; +9,6% in the AFESK group). In the AFESK group only, adiponectin levels remained elevated at 24h (+8,5% compared to baseline), returning to basal levels after 48h in both groups. After S14, adiponectin increased after 15 min and 24 h post-exercise in both groups; however, the AFESK group displayed a greater rise (up to 15%, $p<0.01$) compared to the cycling group (8,8%, $p<0.001$). These results suggest that adding electrical stimulation to cycling is associated with enhanced adiponectin release. No significant between-group differences in baseline adiponectin were observed before S1. However, after 13 training sessions, adiponectin at baseline was significantly higher in the AFESK group compared to the cycling group, probably indicating a training-induced adaptation in adipose tissue. Within the AFESK group, significant differences were also detected among the three time-points (baseline, 15 min, 24 h) when comparing S1 to S14, further supporting a chronic effect of repeated NMES-assisted training on adiponectin secretion. Overall, these data contribute to a deeper understanding of how integrating endurance exercise with NMES affects adipose tissue endocrine activity by increasing adiponectin secretion. Our findings also highlight a potential role for AFESK technology in optimizing metabolic health.

Keywords: *exercise, neuromuscular electrical stimulation, adiponectin release, endurance training.*