Regular physical exercise has many health benefits (1). Paradoxically, it is also clear that contracting skeletal muscles generate reactive oxygen species (ROS) and that prolonged and intense exercise can result in oxidative damage to cellular constituents (2-4). Reactive oxygen species production is dependent on the intensity of the exercise with higher amount of ROS generated by strenuous exercise (5, 6). Antioxidants may reduce the adverse effects of exercise-induced ROS (2-4). However, ROS are not only toxic but rather play an important role in cell signalling and in the regulation of gene expression (7, 8) and force production in skeletal muscle (9). Thus, we have recently raised questions about the validity of using oral antioxidant supplementation during training by athletes (10, 11).

We have found that redox sensitive signaling pathways are involved in skeletal muscle atrophy due to disuse and aging (12). Loss of skeletal muscle mass and function is an important limiting factor in the maintenance of health and well-being both in the young and in the old population (13, 14). I will report experiments showing that both hormonal (15) and non-hormonal interventions (13, 16) are effective in the maintenance of redox homeostasis and in the prevention of skeletal muscle atrophy.

References:

12. F. Derbre et al., Age (Dordr) 34, 669 (Jun, 2011).