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Regular physical activity protects skeletal muscle fibers from age-related dysfunctional remodeling of sarcotubular system and mitochondria

Proper skeletal muscle function is controlled by intracellular Ca^{2+} concentration and by efficient production of energy (ATP), which in turn depend on: (a) release and re-uptake of Ca^{2+} from sarcoplasmic-reticulum during excitation-contraction (EC) coupling, which controls the contraction and relaxation of sarcomeres; (b) uptake of Ca^{2+} into the mitochondrial matrix, which stimulates aerobic ATP production; and finally (c) entry of Ca^{2+} from the extracellular space via store-operated Ca^{2+} entry (SOCE), a mechanism that is important to limit/delay muscle fatigue. Abnormalities in Ca^{2+} handling and inefficient ATP production underlie many physio-pathological conditions, including dysfunction in ageing. In different, but complementary, projects carried out in our laboratory in the past 15-20 years studying: i) structural and functional changes caused by ageing in muscle of humans and mice; and ii)the positive-rescuing effect of exercise, we collected compelling evidence that: a) sedentary ageing causes partial disarray/damage of membrane systems involved in EC coupling and SOCE (collectively the Sarcotubular System), and loss/misplacement of Mitochondria; b) regular exercise promote the rescue/maintenance of both Sarcotubular System and Mitochondria. All these structural changes were accompanied by related functional changes, i.e., loss/decay of function caused by sedentary ageing, and improved function associated to regular exercise. These findings demonstrate that the integrity and proper disposition of intracellular organelles deputed to Ca^{2+} handling and aerobic production of ATP is challenged by reduced activity, but can be maintained-restored by regular physical activity.

Keywords: Excitation–Contraction (EC) coupling; Mitochondria; Store-Operated Calcium Entry (SOCE); Skeletal Muscle

Biography

Feliciano Protasi directs a multi-disciplinary research program 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 (Italy), Dr. Protasi moved to the USA to join the lab. of Prof. Clara Franzini-Armstrong (1993-1997) at the Univ. of Pennsylvania (Philadelphia, PA), before moving to Harvard Medical School (Boston, MA), joining the lab. of Prof. Paul D. Allen. Dr. Protasi returned to Italy in 2002 as Associate Professor and 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.

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