Long-term physical exercise induces changes in sirtuin 1 pathway and oxidative parameters in adult rat tissues

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The protein deacetylase, sirtuin 1, is suggested as a master regulator of exercise-induced beneficial effects. Sirtuin 1 modulates mitochondrial biogenesis, primarily via its ability to deacetylate and activate proliferator-activated receptor-γ coactivator-1α (PGC-1α), interacting with AMPK kinase. Redox cell status can also influence this regulatory axis and together they form an important convergence point in hormesis during the aging process. Here, we tested whether treadmill training (36 weeks), as a paradigm of long-term moderate exercise, modifies the AMPK–sirtuin 1–PGC-1α axis and redox balance in rat gastrocnemius muscle, liver and heart. Physical activity induced increases in sirtuin 1 protein levels in all the aged rat tissues studied, as well as total PGC-1α levels. However, no changes in AMPK activation or significant differences in mitochondrial biogenesis (by measuring electron transport chain protein content) were found after exercise training. Parallel to these changes, we observed an improvement of oxidative stress defenses, mainly in muscle, with modification of the antioxidant enzyme machinery resulting in a reduction in lipid peroxidation and protein carbonylation. Thus, we demonstrate that moderate long-term exercise promotes tissue adaptations, increasing muscle, liver and heart sirtuin 1 protein content and activity and increasing PGC-1α protein expression. However, AMPK activation or mitochondrial biogenesis is not modified, but it cannot be discarded that its participation in the adaptive mechanism which prevents the development of the deleterious effects of age.

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