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Oxidative Stress and Skeletal Muscle Function

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Deadline for manuscript submissions:
closed (31 December 2022)

Message from the Guest Editor

Excessive stretching and intensive exercise causing rupture of myofibril filaments lead to skeletal muscle loss of function through the failure in the excitation–contraction coupling system. These events generate inflammatory response and an higher reactive oxygen species (ROS) production. ROS are continuously generated in the body and are usually promptly inactivated by the cellular antioxidant defenses. In skeletal muscle, low concentrations of ROS modulate cell signaling processes and are required for normal force production, while higher ROS concentrations can lead to DNA, lipid, protein, and carbohydrate modifications, which can cause cellular function impairment and a reduced force production thereby contributing to muscle fatigue.

This Special Issue Oxidative Stress and Skeletal Muscle Function invites submissions of manuscripts, either original research or reviews with an emphasis on describing new biomarkers or novel exercise-regulated signaling pathways as well as new techniques and research approaches involved in the interplay between oxidative stress, physical activity, nutritional strategies and skeletal muscle damage.

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Message from the Editor-in-Chief

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