

## Special Issue

# Exercise Training and Supplements for Improving Function in Muscular Dystrophy

### Message from the Guest Editors

Muscular dystrophy is a generic term describing heritable conditions resulting in the progressive weakness and atrophy of muscle. The most severe is Duchenne Muscular Dystrophy (DMD), which is caused by a lack of dystrophin, encoded by one of the largest genes in the human body. Genetic cures of diseases, such as DMD, are the obvious approach. Exercise has become a mainstream treatment option for many diseases, such as cancer, heart failure and depression. However, since exercise uses the very muscles that are damaged and wasting in muscular dystrophy, there are still question marks as to whether exercise is beneficial in muscular dystrophy. Similarly, a number of supplements have proven to be of benefit to skeletal muscles, either augmenting the effects of exercise training, or directly improving the size and strength of muscles. The aim of this Special Issue is to highlight the effects of exercise and supplements on muscular dystrophy, with a particular focus on DMD.

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### Guest Editors

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### Deadline for manuscript submissions

closed (30 November 2020)



## Sports

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## About the Journal

### Message from the Editor-in-Chief

*Sports* (ISSN 2075-4663) is a peer-reviewed scientific journal that publishes original articles, critical reviews, research notes and short communications in the interdisciplinary area of sport sciences and public health. It links several scientific disciplines in an integrated fashion, to address critical issues related to sport science and public health. The journal presents diverse original articles, including systematic and narrative reviews, cohort and case control studies, innovative randomized trials, and formative research using qualitative and quantitative methods with the aim to provide information for researchers to plan intervention programs. It addresses diverse public health, physical activity and exercise science topics.

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### Editor-in-Chief

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