Skeletal muscle represents over 40% of the body and is important for metabolism, exercise, and movement. The acute nature of muscle energy failure is manifested in a metabolic crisis with muscle weakness, sometimes associated with acute failure, while exercise is vital for muscle maintenance and regeneration.

In muscle, several organelles (sarcoplasmic reticulum, triads, T-tubules, mitochondria, etc.) contribute to the regulation of calcium homeostasis, oxidative stress, mitochondrial function, and diminished activity of the potent anabolic regulator mTOR (mammalian target of rapamycin), including its failure to shuttle to and from the lysosomes in response to cellular stress. All these organelles may contribute to tissue damage in aging and muscle diseases. Autophagy is controlled by FoxO transcription factors that control protein expression [1].

Why do we need a muscle journal? An easy answer would simply be to say that this journal will be indeed needed for the recent progress in the field, particularly sections that will be devoted to muscle biochemistry, biology, and pharmacology; however, the response is simply that there is currently an explosion of new techniques and approaches in the field of muscle biology and pathology, where new techniques such as next generation sequencing and muscle imaging are needed for its full interpretation and discussion. This was the impetus for starting the journal in which you are now collecting both human and animal models of muscle adaptation and plasticity, as well as the pathophysiology of muscle disease.

I am pleased to announce a new open access journal—Muscles (ISSN: 2813-0413) [2]. Muscles will provide a platform for scientists and academics all over the world to promote, share, and discuss various new advances and developments in the field of muscles.

Muscles will publish papers covering a broad range of areas, including but not limited to: muscle microanatomy; muscle biochemistry; muscle cell biology; muscle epidemiology; muscle immunology; muscle pathology; muscle pharmacology; muscle physiology; muscle toxicology, muscle diseases, etc.

We welcome you to Muscles and invite you to contribute your papers or submit Special Issue proposals. We look forward to receiving your manuscripts for publication in this journal.

Conflicts of Interest: The author declares no conflict of interest.

References

1. Nascimbeni, A.C.; Fanin, M.; Masiero, E.; Angelini, C.; Sandri, M. Impaired Autophagy Contributes to Muscle Atrophy in Glycogen Storage Disease Type II Patients. Autophagy 2012, 8, 1697–1700. [CrossRef] [PubMed]
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