



Editorial

Muscle wasting: Current progress and future aims

Loss of muscle mass is encountered in patients with a wide variety of catabolic conditions, including cancer, severe injury, sepsis, kidney disease, diabetes, and heart failure. In addition, normal aging is associated with muscle atrophy. Muscle wasting has many significant clinical consequences. For example, muscle wasting results in weakness and fatigue that may delay or prevent ambulation in patients with severe injury and sepsis. When respiratory muscles are affected, patients may require prolonged ventilatory support and an extended length of stay in the intensive care unit after severe injury and during sepsis. Muscle wasting makes a significant contribution to the loss of quality of life in patients with cancer and contributes to mortality in many patients with malignant disease. It is obvious, then, that an increased understanding of mechanisms leading to muscle wasting has important clinical implications.

Muscle mass reflects the balance between anabolic and catabolic mechanisms and under steady state conditions, protein balance is maintained by equal rates of protein synthesis and degradation. In catabolic conditions, this balance is shifted towards loss of muscle tissue by increased protein breakdown, reduced protein synthesis or a combination of these changes. There is evidence that muscle wasting in various catabolic conditions is mainly caused by increased protein breakdown, in particular breakdown of myofibrillar proteins.

Recent years have seen a dramatic increase in our understanding of mechanisms involved in the regulation of muscle mass. Signaling pathways and proteolytic mechanisms that are activated in muscle wasting

conditions have attracted significant interest and in this directed issue of the International Journal of Biochemistry and Cell Biology, both these aspects of muscle atrophy are discussed. The issue contains review papers as well as original articles and describes various clinical conditions in which muscle wasting constitutes a significant problem.

Although we have not been able to cover all aspects of muscle wasting in this issue, the directed issue provides a summary of our present knowledge of many important aspects of muscle loss. In addition, several authors discuss directions for future studies that will enhance our understanding of mechanisms involved in muscle atrophy. We hope you will find the articles informative and stimulating and able to generate new ideas and continued advancement in the important field of muscle wasting.

Per-Olof Hasselgren*

*Beth Israel Deaconess Medical Center
Department of Surgery, Harvard Medical School
330 Brookline Avenue, ST8M10, Boston
MA 02215, USA*

Didier Attaix

*Human Nutrition Research Center of
Clermont-Ferrand, France*

* Corresponding author. Tel.: +1 617 667 5231

fax: +1 617 667 8172

E-mail address: phasselg@caregroup.harvard.edu
(P.-O. Hasselgren)

6 June 2005