Chapter 1

Introductory Chapter: Current Status of Research Field in Muscle Tissue

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Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/intechopen.79771

1. Introduction

Skeletal muscle tissue accounts for almost half of the human body mass. Muscle contractions of the skeletal muscle enable to move body and maintain homeostasis. Human health is markedly affected by any deterioration in the material, metabolic, and contractile properties of skeletal muscle. Skeletal muscle is a highly plastic organ that is modulated by various pathways controlling cell and protein turnover. Nowadays, the autophagy-dependent system and ubiquitin-proteasome signaling are well known as a major intracellular degradation system, and its appropriate function is crucial to health and muscle homeostasis. Indeed, muscle wasting and weakness such as cachexia, atrophy, and sarcopenia are characterized by marked decreases in the protein content, myonuclear number, muscle fiber size, and muscle strength [1]. Muscle wasting elicits a poor functional status and reduces the quality of life. Thirty-five percent of all cancer patients directly die because of cachexia and not from cancer. Different types of molecular triggers/catabolic factors such as pro-inflammatory cytokines and myostatin also seem to involve muscle wasting [2]. In contrast, mTOR- or serum response factor (SRF)-dependent signaling are positive regulators to promote protein synthesis and skeletal muscle-specific mRNA transcription. Interestingly, a functional defect in autophagy-dependent signaling in sarcopenic mice and humans are recently suggested [3, 4]. Such a condition accumulates the denaturing protein and nonfunctional mitochondria eventually result in the atrophy of sarcopenic muscle fibers because of the deterioration of homeostasis.

2. Various therapeutic approaches for muscle wasting

To attenuate various forms of muscle wasting, many researchers have investigated exercise-based, supplemental, and pharmacological approaches. For example, the combination of resistance training and amino acid-containing supplements is thought to effectively prevent sarcopenia. In addition, myostatin inhibition for sarcopenic patients was successful in phase II trials, but the effect on muscular dystrophy is unclear. The administrations of ghrelin and megestrol acetate have shown good results against cancer cachexia [5]. Furthermore, recent studies [6, 7] indicated the possible application of novel supplements such as soy isoflavone and ursolic acid to prevent muscle atrophy in rodents. More recently, pharmacological treatment with fibroblast growth factor 19 markedly ameliorated two different type of muscle atrophy after aging and glucocorticoid treatment, probably via an obligate co-receptor for fibroblast growth factor 15/19, β-Klotho.

3. The function of smooth muscle cells

Our circulatory system is modulated of the heart, lungs, and vasculature. These components serve crucial roles in controlling blood and lymph flow and in the delivery of gases, hormone, and essential nutrients (i.e., glucose, fat, or amino acids). Vascular smooth muscle cells (VSMCs) are the most numerous cell types in blood vessels. They are located in the medial layer of the vascular wall, i.e., in the tunica media. The media also contains sparse fibroblasts and macrophages along with an interstitial matrix consisting collagens; chondroitin sulfate proteoglycans including versican; glycoproteins such as tenascin, vitronectin, and fibronectin; and elastic laminae. VSMCs serve critical regulatory roles of blood vessels, particularly for vasoconstriction, vasodilatation, and synthesis of vascular extracellular matrix. Adult blood vessels are normally contractile, static, and quiescent. However, under cardiovascular disease including atherosclerosis, hypertension, and diabetic angiopathy, VSMCs undergo phenotypic alterations and revert to a growth-promoting, synthetic nature. Indeed, after biochemical or mechanical damage to blood vessels, VSMCs undergo phenotypic modulation, characterized by increased proteosynthesis and by activation of the migration and growth of VSMCs [8, 9]. These changes often lead to severe damage to blood vessels, including stenosis and occlusion. Ischemia of the tissues supplied by the damaged vessels is then manifested by serious disorders, e.g., heart failure, brain stroke, or necrosis of leg tissues, which can result in amputation of the leg.

Vascular remodeling is an adaptive alternating process of vascular wall architecture and is caused by various stimuli such as vascular injury, oxidative stress, and hemodynamic stress [10]. VSMCs and endothelial cells compose the arteries and have essential roles in vascular remodeling in conjunction with inflammatory cells (macrophages, monocytes, leucocytes, and lymphocytes) [11]. During vascular remodeling, the infiltration of macrophages and monocytes, synthetic or contractile phenotypic changes of VSMCs, and the EC dysfunction promote vascular diseases such as atherosclerosis. Therefore, modulation of VSMC phenotype, maintenance of ECs, and regulation of inflammation in the vessel wall are important in arterial function and homeostasis.

This book deals with current progress and perspectives in a variety topic of skeletal and smooth muscle, stem cells, growth, regeneration, disease, biomaterials, or therapeutics. Novel applications for cell and tissue engineering including cell therapy, tissue models, and disease pathology modeling are welcomed. The molecular mechanism of hypertrophy and atrophy in muscle cell would be also discussed by linking with the signal pathway of protein synthesis and degradation.

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