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Chapter

Prologue: Energy Metabolism and Weight Control

Po-Shiuan Hsieh

1. Introduction

The prevalence of overweight and obesity has increased remarkably over the past decades and become a global epidemic and health threat. Obesity not only has strong genetic determinants but also results from an imbalance between energy storage and energy expenditure. Control of energy homeostasis involved in multiple complicated processes is essential for the maintenance of body weight and life. It becomes extremely important to understand the underlying mechanisms since obesity due to energy excess represents a major threat to health and quality of life.

2. Main subjects

For instance, the progress regarding neuronal circuits that control food intake could extend our understanding of energy homeostasis. In particular, the brain has been considered to play a crucial role in the central regulation of energy intake and also energy expenditure. There are many candidate genes in the central nervous system associated with obesity. In traditional view of homeostatic regulation of the body, weight is mainly by the hypothalamus. However, the recent report showed that the hedonic control of appetite by cortical and subcortical brain areas interacts with homeostatic controls to regulate body weight in a flexible manner to respond to the environmental changes [1]. This new concept has several important implications for the therapeutic strategies of obesity.

On the other hand, the role of adipocytes including brown and white adipocytes has been considered to substantially contribute to the integration of the endocrine and metabolic signaling in energy metabolism regulation. Brown adipose tissue (BAT) thermogenesis is one of the key homeostatic mechanisms for energy expenditure. It is around 60% of "non-shivering" thermogenesis in small mammals attributing to the BAT [2, 3] to sustain their body temperature and survival in the cold [4, 5]. In addition, BAT is currently considered a promising target for the treatment of obesity and T2D [6–10]. Accordingly, there were a number of studies focusing on the related drug development and the underlying mechanism and the several factors implicated in BAT and WAT "browning" such as immune cell-mediated modulation of adipose tissue sympathetic innervation [11]. Nevertheless, although the functional role of BAT in the regulation of energy expenditure, especially thermogenesis and substrate utilization, is dominant in rodent models, the contribution of BAT to energy metabolism and homeostasis in humans is more controversial.

The regulation of mitochondrial metabolism and their consequence also crucially participate in the maintenance of energy homeostasis at the cellular and physiological level. Mitochondria play a central role in the regulation of cellular metabolic homeostasis, which is under the control of the balance between nutrient

supply and energy demand [12]. Metabolic oversupply is followed by fragmentation of mitochondrial network, which leads to a decrease of mitochondrial bioenergetic efficiency that, in association with an increase in nutrient storage, will avoid energy waste. Conversely, under metabolic undersupply, mitochondria elongate in order to increase mitochondrial bioenergetic efficiency and sustain the energy need. Thereby, the mitochondrial function is crucial for the regulation of energy metabolism and weight control.

Even at rest, we need energy for all of the vital functions known as basal metabolic rate (BMR). The determinant factors such as thyroid hormones T3, T4 [13, 14], and sarcolipin [15] and their impact on energy homeostasis will be the other important issue. Thyroid hormones have been well documented as the key regulator of energy metabolism (calorigenic effect), especially the basal metabolic rate for decades. However, the cellular and molecular mechanisms underlying the regulatory role of thyroid hormones are still not fully understood. For instance, recent investigation showed that T4 has been speculated to have more rapid effect on the regulation of basal metabolic rate than T3 in animals [14]. Sarcolipin (SLN) is a novel regulator of sarcoplasmic reticulum Ca^{2+} ATPase (SERCA) in muscle and has been speculated as an important determinant of the BMR in animal with diet-induced obesity [15].

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