Endocrine control of energy homeostasis

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Editorial

Endocrine control of energy homeostasis

Energy homeostasis depends on the balance between the amount of food consumed (energy intake) and the amount of calories used (energy expenditure). The precise coordination between these two processes is essential for the correct maintenance of body composition. Indeed, the physiological control of energy homeostasis involves multiple and compensatory mechanisms located in a large variety of organs. The brain integrates peripheral and central signals to generate satiety or hunger as well as to regulate nutrient partitioning, energy expenditure and the insulin response of glucose metabolism. Numerous neuronal populations in the hypothalamus, brainstem, hindbrain and forebrain have the ability to control multiple aspects of metabolism. Recently, several new sophisticated technologies have been developed to unravel the role of specific neural circuits in the control of body energy balance and glucose homeostasis.

In this Special issue, four reviews are dedicated to give a general overview on how the brain can modulate energy balance and glucose metabolism from different perspectives. *Magnan* et al. review how fatty acid sensitive neurons control energy balance (Magnan et al., 2015). Along this line, *Stark* et al. focus on the functions of hypothalamic fatty acid oxidation, and in particular on the role of hypothalamic carnitine metabolism to facilitate and control both nutrient and hormonal feedback (Stark et al., 2015). Moving from fatty acids to neuropeptides, *FernØ* et al. review the classical and novel pathways by which one of the most relevant neuropeptides, namely orexin, modulates not only feeding but also thermogenesis (Fernø et al., 2015). Finally, the actions of the brain on insulin sensitivity are reviewed by *Sandoval* et al., with particular attention to the central GLP-1 system (Sandoval and Sisley, 2015).

It is also evident that a wide variety of peripheral organs are crucial to control body weight and energy expenditure. One of the most relevant organs is white adipose tissue, which not only responds to signals from traditional hormone systems and the central nervous system, but also expresses and secretes factors with important endocrine functions. As a matter of fact, the discovery of leptin in 1994, a hormone secreted by and in proportion to the amount of fat, led to an explosion of studies in the field of obesity research.

The gastrointestinal tract is also a major physiological system involved in the regulation of energy homeostasis. It secretes several peptides that influence energy balance through regulation of food intake and energy expenditure, besides their local effects controlling gastric-empting, gut motility and nutrient utilization. The gut–brain axis represents a bidirectional exchange of information, and gastrointestinal-derived peptides act mainly as "satiety signals" with the exception of ghrelin, which is the only peripheral hormone with orexigenic and adipogenic properties. In addition to these gastrointestinal peptides, recent evidence has demonstrated that the intestinal microbiota is also linked to obesity and metabolic disorders. In this regard, *Khandekar* et al. review the potentials of the anorexigenic factor, pancreatic polypeptide, as a target for the treatment of obesity, as well as the mechanisms involved in its effects (Khandekar et al., 2015). During the last years, several reports have demonstrated that glucagon-like peptide-1 based multiagonists are promising tools to treat obesity and type 2 diabetes, and the roles of these newly designed molecules is summarized by *Finan* et al. (Finan et al., 2015).

The liver maintains a constant supply of oxidizable substrates, which provide energy to the body. During feeding, the liver generates energy stores in the form of glycogen and triglyceride, the latter being exported and stored in white adipose tissue. During fasting, it releases glucose and ketone bodies. The major signals regulating the transition between the fed and the fasted state are glucose, insulin and glucagon. These changes will affect the enzymes regulating liver carbohydrate and fatty acid metabolism, thereby orienting metabolic fluxes towards either energy storage or substrate release. The liver also plays a major role in the control of insulin signaling, and the ectopic storage of fat in the liver (non-alcoholic fatty liver disease) is one of the most common alterations encountered in obesity and type 2 diabetes. The role of insulin resistance as a frequent link between the metabolic syndrome and non-alcoholic fatty liver disease is precisely the topic of the review by *Asrih* et al. (Asrih and Jornayvaz, 2015). One of the liver-derived factors that is attracting more attention during the last years is fibroblast growth factor-21, which reduces glycemia and lipidemia in rodent models of obesity and type 2 diabetes, as well as induces weight loss by increasing energy expenditure. These aspects are reviewed by *Giralt* et al. (Giralt et al., 2015).

Finally, besides hormonal and nutritional control of energy balance, circadian oscillators play an essential role in the coordination of physiological processes with the cyclic changes in the physical environment. The observations that most cells and tissues express autonomous clocks, and that disruption of clock genes results in metabolic dysregulation have revealed interactions between metabolism and circadian rhythms at neural, molecular, and cellular levels. The studies aimed at understanding the interplay between brain and peripheral clocks and at determining how these interactions regulate glucose and lipid metabolism in nocturnal and diurnal mammals are reviewed by *Jha* et al. (Jha et al., 2015).

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