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Resumo	The characterization of the hypothalamic neuronal network, that controls food intake and energy expenditure, has provided great advances in the understanding of the pathophysiology of obesity. Most of the advances in this field were obtained thanks to the development of a number of genetic and nongenetic animal models that, at least in part, overtook the anatomical constraints that impair the study of the human hypothalamus. Despite the undisputed differences between human and rodent physiology, most seminal studies undertaken in rodents that have unveiled details of the neural regulation of energy homeostasis were eventually confirmed in humans; thus, placing experimental studies in the forefront of obesity research. During the last 15 years, researchers have provided extensive experimental proof that supports the existence of hypothalamic dysfunction, which leads to a progressive whole-body positive energy balance, and thus, to obesity. Here, we review the experimental work that unveiled the mechanisms behind hypothalamic dysfunction in obesity.
Fomento	