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The central role of hypothalamic inflammation in the acute illness response and cachexia

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Abstract

When challenged with a variety of inflammatory threats, multiple systems across the body undergo physiological responses to promote defense and survival. The constellation of fever, anorexia, and fatigue is known as the acute illness response, and represents an adaptive behavioral and physiological reaction to stimuli such as infection. On the other end of the spectrum, cachexia is a deadly and clinically challenging syndrome involving anorexia, fatigue, and muscle wasting. Both of these processes are governed by inflammatory mediators including cytokines, chemokines, and immune cells. Though the effects of cachexia can be partially explained by direct effects of disease processes on wasting tissues, a growing body of evidence shows the central nervous system (CNS) also plays an essential mechanistic role in cachexia. In the context of inflammatory stress, the hypothalamus integrates signals from peripheral systems, which it translates into neuroendocrine perturbations, altered neuronal signaling, and global metabolic derangements. Therefore, this review will discuss how hypothalamic

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