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Review

Interoceptive modulation of neuroendocrine, emotional, and hypophagic responses to stress☆

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HIGHLIGHTS

- Hindbrain neurons help control neuroendocrine and behavioral stress responses.
- Brief periods of fasting profoundly suppress acute stress responsiveness.
- Fasting suppresses central drive to the HPA axis, and reduces anxiety behavior.
- Fasting suppresses stress-induced activation of hindbrain neurons.
- The effect of fasting on stress responsiveness is likely due to suppression of hindbrain signaling.

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ABSTRACT

Periods of caloric deficit substantially attenuate many centrally mediated responses to acute stress, including neural drive to the hypothalamic-pituitary-adrenal (HPA) axis, anxiety-like behavior, and stress-induced suppression of food intake (i.e., stress hypophagia). It is posited that this stress response plasticity supports food foraging and promotes intake during periods of negative energy balance, even in the face of other internal or external threats, thereby increasing the likelihood that energy stores are repleted. The mechanisms by which caloric deficit alters central stress responses, however, remain unclear. The caudal brainstem contains two distinct populations of stress-recruited neurons [i.e., noradrenergic neurons of the A2 cell group that co-express prolactin-releasing peptide (PrRP+ A2 neurons), and glucagon-like peptide 1 (GLP-1) neurons] that also are responsive to interoceptive feedback about feeding and metabolic status. A2/PrRP and GLP-1 neurons have been implicated anatomically and functionally in the central control of the HPA axis, anxiety-like behavior, and stress hypophagia. The current review summarizes a growing body of evidence that caloric deficits attenuate physiological and behavioral responses to acute stress as a consequence of reduced recruitment of PrRP+ A2 and hindbrain GLP-1 neurons, accompanied by reduced signaling to their brainstem, hypothalamic, and limbic forebrain targets.

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Despite marked variation in environmental conditions, the internal milieu of mammals is maintained in a state of dynamic equilibrium known as homeostasis. Homeostatic regulation of critical physiological processes such as body temperature, blood pressure, fluid balance, and blood glucose levels is necessary for survival and well-being. However, this physiological balance is frequently challenged by internal and external forces, referred to as stressors, which can be as severe as an attack by a predator or as mild as a slight drop in blood pressure when moving from a seated to a standing position [124]. In response to acute (i.e., occasional, short-duration) stressors, the central nervous system (CNS) elicits a constellation of neural, neuroendocrine, and behavioral responses that facilitate immediate survival and eventual restoration of homeostatic balance. These responses include activation of the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system, modulation of emotional state to redirect cognitive and attentional resources, and suppression of competing behaviors such as eating and sexual reproduction. These and other responses to acute stressors are complex and multifaceted, and provide a critical means by which to adapt widespread and diverse organismic functions to the frequent homeostatic challenges and threats that pervade daily life. However, stressful challenges also can be chronic and unremitting [e.g., social stress; [131,145–147]], eliciting persistent changes in physiology and behavior that can become maladaptive and compromise health and well-being [158]. Centrally-mediated stress responses of prolonged duration and/or magnitude have been linked to numerous neuropsychiatric, neurological, and physiological diseases and disorders, emphasizing the need to better understand how the brain coordinates responses to both acute and chronic stress exposure [23].

Importantly, centrally-mediated responses to acute and chronic stressors are malleable, and can be modified and reorganized based on current physical state and/or prior experience [9,92,105,172]. As a case in point, caloric deficit (i.e., periods of food restriction or deprivation) substantially attenuates many centrally-mediated responses to acute stress, including stress-induced activation of the neuroendocrine HPA axis and stress-induced suppression of food intake (a.k.a. stress-induced hypophagia). Caloric deficit appears to suppress these stress responses in a coordinated manner, suggesting a process through which their underlying neural substrates are "metabolically tuned" by interoceptive feedback signals reflecting energy balance. These alterations in stress responsiveness can be viewed as adaptive and beneficial during periods of negative energy balance [9,96]; however, the neural mechanisms by which caloric deprivation attenuates stress responsiveness remain unclear. We posit that the apparent "metabolic tuning" of stress responses occurs via modulation of neural activity within a common central circuit node comprising two phenotypically distinct neural populations within the caudal nucleus of the solitary tract (cNTS): glucagon-like peptide 1 (GLP-1) neurons, and prolactin releasing peptide (PrRP)-positive(+) noradrenergic (NA) neurons of the caudal A2 cell group (PrRP+ A2 neurons). As detailed further, below, both neural populations are activated by a wide variety of acute stressors, and both receive robust direct and relayed synaptic input from visceral sensory systems that convey ingestive/metabolic feedback signals from body to brain. Moreover, both neural populations project to multiple stress-related regions of the brainstem, hypothalamus, and limbic forebrain, and both populations contribute to neuroendocrine, emotional, and behavioral stress responses. Considering this, we propose that the ability of caloric deficit to attenuate physiological and behavioral responsiveness to acute stress depends on reduced signaling from hindbrain GLP-1 and PrRP+ A2 neurons. This article will summarize the results of several experiments that we have conducted to test this hypothesis. First, however, we will review the general features of some key neuroendocrine and behavioral stress responses, and the modulation of these responses during states of negative energy balance.

1. Neuroendocrine stress responses

1.1. Hypothalamic-pituitary-adrenal axis

The HPA axis is a metabolic neuroendocrine system that functions continuously to meet hour-by-hour energetic demands of organisms with predictable daily rhythms of behavioral state (i.e., arousal, somnolence, feeding, etc.). However, the typical circadian ebb and flow of HPA axis activity can be "co-opted" by marked activation in response to real or perceived threats, which facilitates meeting the energetic demands of catabolic stress responses. The apex of the HPA axis comprises neurons within the medial parvocellular paraventricular nucleus of the hypothalamus (mpPVN) that synthesize corticotropin-releasing hormone (CRH). Activation of these neurons causes CRH to be released into the hypophyseal portal system, a small system of blood vessels that carry CRH into the anterior lobe of the pituitary gland, where it binds to CRH receptors on corticotropes to induce release of adrenocorticotropic hormone (ACTH) into the systemic circulation. Circulating ACTH then binds to ACTH receptors in the adrenal cortex to increase synthesis of the steroid hormone corticosterone (CORT) [22]. In conjunction with the stress-induced increase in sympathetic outflow, CORT facilitates muscular and hepatic glycogenolysis to mobilize stored energy, and stimulates gluconeogenesis to maintain/elevate circulating glucose. Glucose availability is critical to fuel energy-demanding skeletal muscle contractions occurring as part of the "fight or flight" response to stress. Concurrently, CORT and sympathetic outflow function to suppress energy utilization by physiological maintenance processes such as immune system function, growth, and digestion, instead allocating resources to processes adaptive for immediate survival [124]. Thus, the recruitment of the HPA axis is necessary for producing a massive physiological shift from a state in which priority is given to processes that are adaptive in the long term (i.e., days to weeks) to a state in which priority is given to functions with more immediate value (i.e., seconds to hours). Activation of the HPA axis is regulated by a complex network of central circuits, all of which converge on hypophysiotropic mpPVN neurons [159], and major changes in the neuroendocrine response can be elicited by influencing individual or multiple components of these circuits.

1.2. HPA axis modulation during caloric deficit

In rodents, chronic caloric restriction, complete fasting for 2–4 days, or even a single overnight fast are each sufficient to attenuate central

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