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SEX-SPECIFIC LONG-TERM BEHAVIORAL AND NEUROENDOCRINE CONSEQUENCES OF TRANSIENT PERIPUBERTAL METABOLIC CHALLENGES

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Interaction between neuroendocrine stress and metabolic signals has become increasingly linked to the pathogenesis of affective and cognitive disorders. Independent evidence shows that nuclear receptors represent an essential interface of communication between these signals and the central nervous system.

On the premise that lifelong brain functions are subject to programming by stressful and metabolic signals during early life, we induced voluntary hyperphagia in prepubertal male and female rats by providing them with a cafeteria-like choice between normal chow and foods with high motivational value (palatable, high fat/high energy). The cafeteria diet was withdrawn from one subset of animals during early adulthood (postnatal day, PND 80), and several endpoints of interest were monitored until PND 200. While both sexes displayed similar metabolic outcomes during exposure to the cafeteria diet, there were substantial sex differences in the metabolic, behavioral and endocrine consequences of withdrawal of palatable food. Consistently increased food consumption and body mass gain were recorded in males, whereas females that were pre-exposed to the cafeteria diet showed clear signs of adrenocortical hyperactivity. Thus, besides verification of sex-specific mal-programming of appetitive behavior and metabolic set-points by early life events, we also demonstrate evidence for differential translation of metabolic challenges during peripubertal development into aberrations suggestive of altered neuroendocrine responsiveness to stress.