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Review

Hypothalamic-pituitary-adrenal axis dysregulation and cortisol activity in obesity: A systematic review

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ABSTRACT

Background: Although there is substantial evidence of differential hypothalamic-pituitary-adrenal (HPA) axis activity in both generalized and abdominal obesity, consistent trends in obesity-related HPA axis perturbations have yet to be identified.
Objectives: To systematically review the existing literature on HPA activity in obesity, identify possible explanations for inconsistencies in the literature, and suggest methodological improvements for future study.
Data sources: Included papers used Pubmed, Google Scholar, and the University of California Library search engines with search terms body mass index (BMI), waist-to-hip ratio (WHR), waist circumference, sagittal diameter, abdominal versus peripheral body fat distribution, body fat percentage, DEXA, abdominal obesity, and cortisol with terms awakening response, slope, total daily output, reactivity, feedback sensitivity, long-term output, and 11β-HSD expression.
Study eligibility criteria: Empirical research papers were eligible provided that they included at least one type of obesity (general or abdominal), measured at least one relevant cortisol parameter, and a priori tested for a relationship between obesity and cortisol.
Results: A general pattern of findings emerged where greater abdominal fat is associated with greater responsivity of the HPA axis, reflected in morning awakening and acute stress reactivity, but some studies did show underresponsiveness. When examined in adipocytes, there is a clear upregulation of cortisol output (due to greater expression of 11β-HSD1), but in hepatic tissue this cortisol is downregulated. Overall obesity (BMI) appears to also be related to a hyperresponsive HPA axis in many but not all studies, such as when acute reactivity is examined.
Limitations: The reviewed literature contains numerous inconsistencies and contradictions in research methodologies, sample characteristics, and results, which partially precluded the development of clear and reliable patterns of dysregulation in each investigated cortisol parameter.
Conclusions and implications: The literature to date is inconclusive, which may well arise from differential effects of generalized obesity vs. abdominal obesity or from modulators such as sex, sex hormones, and chronic stress. While the relationship between obesity and adipocyte cortisol seems to be clear, further research is warranted to understand how adipocyte cortisol metabolism influences circulating cortisol levels and to establish consistent patterns of perturbations in adrenal cortisol activity in both generalized and abdominal obesity.

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1. Introduction

1.1. Rationale

In an age where obesity has reached epidemic levels and is implicated in several of the leading causes of death in the United States (Ogden et al., 2014), it is essential to understand the physiological correlates, predictors, and consequences of obesity. This systematic review examines the relationship between obesity and perturbations of the hypothalamic-pituitary-adrenal (HPA) axis. Understanding these perturbations in obesity is particularly important given that dysregulation of the HPA axis is a risk factor for physical health conditions such as cardiovascular disease, insulin resistance and type 2 diabetes, stroke, and Cushing's syndrome (Pasquali et al., 2006; Rosmond and Bjorntorp, 2001), as well as mental health conditions such as depression and cognitive impairment (Hinkelmann et al., 2009; Reppermund et al., 2007). Additionally, in both human and animal models, cortisol has been causally demonstrated to promote the accumulation of fat cells and weight gain (Björntorp and Rosmond, 2000; Bjorntorp, 2001), implicating HPA axis functioning in the etiology of obesity. The literature, however, is inconsistent at best in terms of how the HPA axis is dysregulated in obesity. This gap precludes a comprehensive understanding of the pathophysiology of obesity. Furthermore, because it is unclear whether obesity contributes to HPA dysregulation or vice versa, it is difficult to identify appropriate intervention targets, thus hindering the development of effective treatments.

1.2. Cortisol background: measurement, stress-related weight gain, and adipocyte biology

To aid in the reading of this review, we first briefly summarize the broader relationships among stress, cortisol, adipocyte biology, and weight gain (see Fig. 1). Fluctuations in cortisol concentrations occur according to a natural diurnal pattern as well as in response to both physiological and psychological stressors. Stress-related

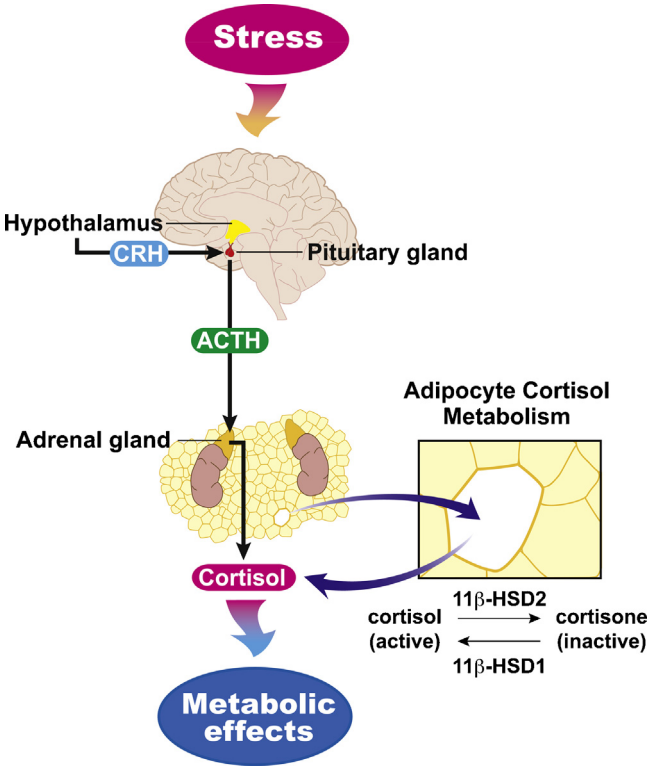

Fig. 1. Adipocyte cortisol metabolism in the context of HPA axis activity.

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