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Metabolic pathways link childhood adversity to elevated blood pressure in midlife adults

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KEYWORDS

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Summary Childhood adversity is a risk factor for adult health outcomes, including obesity and hypertension. This study examines whether childhood adversity predicted mean arterial pressure through mechanisms of central obesity and leptin, adiponectin, and/or insulin resistance, and including dietary quality.

210 Black/African Americans and White/European Americans, mean age = 45.8; ± 3.3 years, were studied cross-sectionally. Path analyses were used to specify a chain of predictive variables in which childhood adversity predicted waist–hip ratio and dietary quality, circulating levels of hormones, and in turn, mean arterial pressure, adjusting for race, gender, and antihypertensive medications.

Direct paths were found between childhood adversity, waist–hip ratio, and leptin levels and between leptin and dietary quality to mean arterial pressure. Systolic and diastolic blood pressures were similarly predicted.

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Early adversity appears to developmentally overload and dysregulate endocrine systems through increased risk for obesity, and through a direct impact on leptin that in turn, impacts blood pressure.

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Introduction

Childhood adversity has been identified as a key risk factor in the development of adult obesity and a number of disease states, such as Type 2 diabetes, ischaemic heart disease, cancer, and emphysema [1–3]. Childhood adversity has also been identified as a risk factor in the development of hypertension [4–8]. However, despite the well-described association between childhood adversity and obesity and between obesity and hypertension, there is little evidence regarding mechanisms linking childhood adversity to hypertension in adulthood [8].

A large body of research has examined the stress–hypertension link, yielding mixed results. Although acute stress leads to increased heart rate and raised blood pressure, it is not yet understood how this translates into sustained hypertension [9,10]. There is speculation that repeated activation of the hypothalamic-pituitary-adrenal (HPA) axis with failure to recover or habituate is a factor in the development of hypertension [10]. Similarly, it is hypothesised that repeated adverse experiences with little recovery time and/or chronic exposure to stress, e.g., allostatic overload, is a mechanism by which adverse events early in life lead to physical and mental health disorders in adult life [11,12].

There is mounting evidence that childhood stress and adversities directly alter metabolic functioning [12–16]. Furthermore, a chain of dysregulation from obesity to adipocytokine activity to sympathetic nervous system (SNS) function and hypertension has been hypothesised. However, we found no studies that examine how early adversities are linked to a metabolic path to hypertension [14,16–18].

Adipocytokines, especially leptin, appear to regulate metabolic links between stress, the HPA axis, neuropeptide Y and appetite, energy expenditure, and weight [13,16,19]. Perceived stress has been linked to higher leptin levels [20], and the specific stress of sleep deprivation has been shown to impact leptin, hunger, diet, and hypertension. Childhood adversity is associated with elevated circulating leptin, but not adiponectin, controlling for demographic variables, physical activity, smoking, diet, current mental health, and body mass index

(BMI) [15]. This suggests that leptin dysregulation has a role in the pathogenesis of adult obesity in the context of childhood adversity [13].

Obesity-related hypertension is hypothesised to be due not only to mechanical load, but the effect of energy homeostatic mediator molecules, e.g., leptin, on increased sympathetic nervous system activity, arteriole constriction, and renal sodium reabsorption [14,18,21–27]. A number of cross-sectional and prospective studies have identified links between obesity, leptin and adiponectin, insulin resistance, dietary quality, and hypertension [17,18,22–24,27–31].

Current study

In this study, we examine childhood adversity as a predictor of mean arterial pressure (MAP), that is, the steady flow of blood through arteries combining childhood cardiac output and vascular resistance [32,33], as well as systolic and diastolic blood pressures. In a sample of mixed-risk midlife Black/African Americans and White/European American men and women, we use path analyses to specify a chain of predictive variables in which childhood adversity predicts central obesity (waist–hip ratio, WHR) that in turn impact leptin levels. We hypothesise that childhood adversities will be directly associated with leptin and through WHR, and that circulating leptin, dietary quality, and WHR are associated with MAP. We also examine path models for the potential roles of insulin resistance using homeostatic model assessment (HOMA) and adiponectin (see Fig. 1).

This diverse sample allows us to control for race and gender-related differences in obesity, leptin, and hypertension [10,31,34–36]. We also control for anti-hypertensive medications. We include dietary quality both because of its association with blood pressure, but because childhood adversity may impact dietary quality for psychosocial reasons, e.g., poverty [2,37,38].

Methods

Sample

Participants were 210 White/European American and Black/African American adults (mean

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