Psychoneuroendocrinology (2014) 41, 132—141

Available online at www.sciencedirect.com

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Associations of socioeconomic and psychosocial factors with urinary measures of cortisol and catecholamines in the Multi-Ethnic Study of Atherosclerosis (MESA)

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Received 8 May 2013; received in revised form 25 November 2013; accepted 19 December 2013

KEYWORDS
Urinary cortisol;
Urinary catecholamines;
Social factors;
Psychological factors;
Race/ethnicity;
Multi-Ethnic of Atherosclerosis

Summary
Background: Stress hormones have been hypothesized to contribute to the social patterning of cardiovascular disease but evidence of differences in hormone levels across social groups is scant.
Purpose: To examine the associations of socioeconomic and psychosocial factors with urinary levels of cortisol and catecholamines and determine whether these associations are modified by race/ethnicity.
Methods: Measures of cortisol, epinephrine, norepinephrine and dopamine were obtained on 12-h overnight urine specimens from 942 White, African American and Hispanic participants in the Multi-Ethnic Study of Atherosclerosis (MESA). Linear regression was used to examine associations of income-wealth index, education, depression, anger, anxiety and chronic stress with the four hormones after adjustment for covariates.

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0306-4530/$ — see front matter
http://dx.doi.org/10.1016/j.psyneuen.2013.12.013

Results: Higher income-wealth index was associated with lower levels of urinary cortisol, epinephrine, norepinephrine and dopamine, after adjustment for age, sex, race/ethnicity, medication use, body mass index, smoking, and alcohol use. Education and psychosocial factors were not associated with urinary stress hormone levels in the full sample. However, there was some evidence of effect modification by race: SES factors were more strongly inversely associated with cortisol in African Americans than in other groups and anger was inversely associated with catecholamines in African Americans but not in the other groups.
Conclusions: Lower SES as measured by income-wealth index in a multi-ethnic sample is associated with higher levels of urinary cortisol and catecholamines. Heterogeneity in these associations by race/ethnicity warrants further exploration.

1. Introduction

It has long been known that socioeconomically disadvantaged persons are at higher risk of developing cardiovascular disease (CVD), and tend to have worse outcomes once they develop it, compared to those who are less disadvantaged (Marmot et al., 1991; Kaplan and Keil, 1993; Diez Roux et al., 2001; Steenland et al., 2004; Williams and Jackson, 2005). Many studies have shown that behavioral and biomedical cardiovascular risk factors (Ramsay et al., 2011), such as smoking (Novotny et al., 1988), hypertension (Brummett et al., 2011), physical activity (Cauley et al., 1991), diet (Galobardes et al., 2001), obesity (Sobal and Stunkard, 1989), and diabetes and blood lipids (Manuck et al., 2010; Wild et al., 2010) are associated with measures of socioeconomic status (SES). However, behavioral and biomedical cardiovascular risk factors do not fully explain the association of CVD with SES (Kaplan and Keil, 1993; Diez Roux et al., 2001; Marmot, 2001). Some researchers have therefore hypothesized that psychosocial stress and its biological consequences may also contribute to the social patterning of CVD (Adler et al., 1994; Steptoe and Marmot, 2002; Gebreab et al., 2012).

Among the most widely recognized biomarkers of stress are cortisol and catecholamines, which are the effectors of the two major systems mediating most components of the stress response. Cortisol levels in plasma and urine reflect the functioning of the hypothalamic-pituitary axis (HPA) (Tsigos and Chrousos, 2002), while catecholamine concentrations in plasma and urine reflect activation of the sympathetic nervous system (SNS) (Axelrod and Reisine, 1984). A small but relatively constant proportion of the circulating levels of epinephrine and norepinephrine in the blood is excreted into the urine (Frankenhaeuser, 1971). Urinary measurements of both cortisol and epinephrine and norepinephrine have previously been used to assess the activity of the HPA axis and the SNS in laboratory and population studies (Lundberg and Frankenhaeuser, 1980; Seeman et al., 1994, 2001; Janicki-Deverts et al., 2007).

Dopamine is both a neurotransmitter and a precursor in the synthesis of norepinephrine, which is both a hormone and a neurotransmitter. To our knowledge, only two prior studies of stress and socioeconomic status and psychosocial factors have included dopamine; both studies combined dopamine measures into a multi-measure index of the allostatic load (Seplaki et al., 2004, 2006; Glei et al., 2007).

Dysregulation of the HPA or SNS or both has been hypothesized to contribute to the development of atherosclerosis or to cardiovascular events through a number of mechanisms (Manuck et al., 1995). Excessive cortisol production has been associated with development of the metabolic syndrome and visceral obesity, insulin resistance, dyslipidemia, and hypertension (Chrousos and Gold, 1998; Chrousos, 2000). An attenuated decline in norepinephrine and epinephrine excretion during nighttime sleep has been observed among ''nondippers'' (persons whose systolic blood pressure drop less than 10% during nighttime sleep) (Sherwood et al., 2002). Catecholamines may influence the development of atherosclerosis through their metabolic effects on glucose and insulin or through hemodynamic effects on the arterial wall (Pickering, 1999). Cortisol and catecholamines also initiate a cytokine response characterized by expression of adhesion molecules on the endothelium and a local inflammatory reaction in the vascular wall (Black and Garbutt, 2002). Stress hormones may also activate nuclear factor kappa (NF-κB) in macrophages, with consequences for inflammation and apoptosis (Black, 2006). In addition, epinephrine and to a lesser degree norepinephrine, are implicated in the activation and/or aggregation of platelets and may, therefore, promote thrombus formation (Markovitz and Matthews, 1991).

Although it is often hypothesized that stress hormones and related mechanisms could contribute to the social and psychosocial patterning of cardiovascular disease, few studies have directly examined the relationship between SES or psychosocial factors and stress hormones. A handful of studies have investigated associations of salivary cortisol measures obtained at varying times over the day with socioeconomic and psychosocial factors. For example, persons of low SES had lower salivary cortisol at wake up and less pronounce decline of salivary cortisol over the day than persons of high SES (Hajat et al., 2010). Higher levels of cynical hostility have also been linked to less pronounced daily declines in cortisol (Ranjit et al., 2009). Other studies have reported a relationship between elevated awakening salivary cortisol levels and depressive symptomatology (Pruessner et al., 2003).

Although it is often hypothesized that stress may also contribute to race and ethnic differences in cardiovascular disease (Williams and Jackson, 2005), very few studies have investigated differences in stress hormone levels by race/ethnicity. Persons of Black or Hispanic race/ethnicity have been found to have lower levels of wake up salivary cortisol and less pronounced daily cortisol decline over the day (Hajat et al., 2010) or higher values of bedtime salivary cortisol (Cohen et al., 2006b) than non-Hispanic whites. No studies of which we are aware have investigated whether associations