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Social strain and cortisol regulation in midlife in the US

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ABSTRACT

Chronic stress has been implicated in a variety of adverse health outcomes, from compromised immunity to cardiovascular disease to cognitive decline. The hypothalamic pituitary adrenal (HPA) axis has been postulated to play the primary biological role in translating chronic stress into ill health. Stressful stimuli activate the HPA-axis and cause an increase in circulating levels of cortisol. Frequent and long-lasting activation of the HPA-axis, as occurs in recurrently stressful environments, can in the long run compromise HPA-axis functioning and ultimately affect health. Negative social interactions with family and friends may be a significant source of stress in daily life, constituting the type of recurrently stressful environment that could lead to compromised HPA functioning and altered diurnal cortisol rhythms. We use data from two waves (1995 and 2004–2005) of the Midlife in the U.S. (MIDUS) study and from the National Study of Daily Experiences (NSDE) and piecewise growth curve models to investigate relationships between histories of social strain and patterns of diurnal cortisol rhythms. We find that reported levels of social strain were significantly associated with their diurnal cortisol rhythm. These effects were more pronounced for individuals with a history of greater reported strain across a ten-year period.

Introduction

The role of social support on a variety of health outcomes is well-documented in the sociological, psychological, and epidemiological literatures (see, for example, Anderson & Armstead, 1995; House et al., 1994; Seeman, Seeman, & Sayles, 1985; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Although attention has focused more heavily on the protective influences of social contact and support, a smaller literature has also documented the negative health consequences of adverse social interactions. Stressful relationships with family and friends, for example, are related to a variety of health outcomes, including functional limitations (Newsom, Mahan, Rook, & Krause, 2008), cardiovascular disease (Coyne et al., 2001; Ewart, Taylor, Kraemer, & Agras, 1991; Orth-Gomer et al., 2000), decreased immunity (Seeman, 1996), and even mortality (Patterson & Veenstra, 2010; Seeman, Kaplan, Knudsen, Cohen, & Guralnik, 1987).

The biological mechanisms through which these health effects of social relationships are thought to operate include influences on the brain and resulting changes in physiological activity in major biological regulatory systems (DeVries, Glasper, & Detillion, 2003; Hofer, 1987, 1995; McEwen, 2007). Functional magnetic imaging (fMRI) studies have begun to illuminate the ways in which social relationships are processed by the brain, showing effects on brain processes likely to influence biological systems, including HPA function (Eisenberger et al., 2007; Taylor et al., 2008). One study found that greater reported social support was associated with diminished neuroendocrine reactivity to social stressors (Eisenberger et al., 2007).

Indeed, a growing body of evidence indicates that both positive and negative social relationships influence biology (see Seeman & McEwen, 1996; Uchino, 2006 for reviews). Community-based studies, for example, have linked social support to lower heart rate, systolic blood pressure, serum cholesterol, smaller waist–hip ratios, lower risk of metabolic syndrome, lower urinary cortisol and catecholamines, and sharper, more pronounced diurnal cortisol rhythms (Ryff, Singer, & Love, 2004; Seeman & McEwen, 1996; Sjögren, Leanderson, & Kristenson, 2006; Vogelzangs et al., 2007). Greater reported social conflict, on the other hand, has been linked to higher blood pressure, cholesterol, inflammation, poorer metabolic profiles, and higher urinary catecholamines and cortisol (Seeman & McEwen, 1996). Experimental evidence similarly shows that positive social relationships decrease cardiovascular and neuroendocrine responses to challenging tasks (Floyd et al., 2007;

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Grewen, Anderson, Girdler, & Light, 2003) while interpersonal conflict or hostility leads to increased cardiovascular and neuroendocrine reactivity (Gerin, Pieper, Levy, & Pickering, 1992; Seeman & McEwen, 1996).

Why salivary cortisol?

Large-scale surveys have increasingly sought to include salivary cortisol assessments to index HPA-axis reactivity. This is because of the centrality of the HPA-axis in regulating multiple aspects of human physiology that are critical to health and well-being, and the hypothesized links between such HPA-axis activity and cognitive-emotional responses to the world around us, including importantly our social worlds. Stimuli that activate the HPA function cause an increase in cortisol which triggers downstream physiological responses that help provide the energy and physiological resources needed to adapt to that stimulus. Activation of cortisol also helps to contain other components of the physiological stress response such as increases in inflammatory processes which, if unchecked, can themselves have negative health consequences. Thus, short-term activation of the HPA-axis is necessary for optimal everyday physiological functioning. However, recurrent or chronic activation of this system has been linked to increased risks for a variety of adverse health outcomes, including cardiovascular disease, diabetes, cancer, cognitive decline, and reduced immune function (for a review, see McEwen & Seeman, 1999). In addition, the diurnal rhythm is sensitive to and altered by a variety of stressful situations (Adam & Gunnar, 2001; Steptoe et al., 2003; Vedhara, Hyde, Gilchrist, Tytherleigh, & Plummer, 2000).

Earlier work on the relationships between cortisol and health has focused on average cortisol measures with an interest in cortisol levels over the entire day. This is the approach used, for example, when collecting urinary cortisol, which involves one cortisol sample that is an aggregate measure of, typically, 12–24 h of cortisol (Seeman et al., 2002). With the advent of salivary cortisol protocols, research has examined patterns of cortisol activity at multiple times of the day for one or more days. Such data capture what is typically referred to as the "cortisol diurnal rhythm." The diurnal rhythm is characterized by a rapid increase in cortisol over the first 30–45 min after waking, followed by a rapid decline over approximately the next 2 h and then a slower decline through the late afternoon and evening. Younger, healthier individuals show a more pronounced diurnal rhythm with a higher morning peak and a lower night-time nadir and less healthy and older individuals have a flatter curve (Adam & Kumari, 2009). Examining salivary cortisol over the course of the day provides a more complete picture of cortisol regulation (or dysregulation). In fact, one of the primary advantages of salivary cortisol samples over urinary or blood samples is that they allow for repeated and unobtrusive measurement of cortisol over multiple times of the day (Almeida, McGonagle, & King, 2009).

Prior research on social relationships and cortisol

The influence of social relationships on cortisol response has been a topic of interest in both human and animal research. Animal research has long suggested that contact with others of the same species plays a critical role in successful development, and animals demonstrate the potential for both positive and negative health effects of the social environment (Cassel, 1976; Henry, Meehan, & Stephens, 1967; Levine, 1993). To date, research examining social support and cortisol in human populations has largely taken experimental approaches. Experimental manipulations provide strong evidence that social contact or support from a friend or partner during challenge tests (such as math or public speaking tasks) *decreases* neuroendocrine responses, including cortisol (Grewen et al., 2003; Seeman & McEwen, 1996; Uchino et al., 1996). In contrast, reported inadequate support has been linked to *greater* physiological reactivity, again including cortisol responses to laboratory-based challenge tests (Nausheen, Gidron, Gregg, Tissarchondou, & Peveler, 2007; Seeman & McEwen, 1996; Uchino et al., 1996).

Community-based and, more recently, population-level studies have also begun to focus on associations between aspects of the social environment and cortisol regulation. For instance, one community-based study of social strain and urinary cortisol found that increased frequency of demands and criticism was positively related to overnight urinary cortisol levels for men but not women (Seeman, Berkman, Blazer, & Rowe, 1994). Greater reports of hostility and cynicism are related to higher levels of cortisol in the daytime (Pope & Smith, 1991; Ranjit et al., 2009). In addition, social relationships with parents in childhood may have lasting effects on cortisol levels well into middle and later life (Repetti, Taylor, & Seeman, 2002; Taylor, Karlamangla, Friedman, & Seeman, 2011).

One of the difficulties in investigating the relationship between social stressors and salivary cortisol is that salivary cortisol must be measured at multiple time points over the day, and there are therefore many different measures of cortisol that may be used to capture a dysregulated rhythm. One might think about dysfunction as a blunted morning peak, a slower decline in cortisol levels in the evening, or as a measure of accumulated daily cortisol, such as the area under the curve. For the most part, the influence of social factors on cortisol appears to be strongest for measures capturing cortisol decline over the course of the day, particularly for the evening and resting cortisol levels (Adam & Kumari, 2009; Seltzer et al., 2009). Blunting of the diurnal rhythm over the day (lower peak and higher nadir) occurs systematically with age, and this same blunted rhythm is also evident for individuals exposed to frequent life stresses (Birditt, Cichy, & Almeida, 2011; Varadhan et al., 2008). We therefore hypothesize that individuals with greater levels of social strain will show a flatter cortisol rhythm overall, and show particularly pronounced dysregulation in the latter part of the day.

Conceptualization of social strain

Social strain may be conceived of in a variety of ways. It can be thought of as network stress, problematic social exchanges, or interactions with network members that induce psychological distress. In keeping with Goffman's analysis of "face work," we treat social strain as a characteristic of interactions that "is neither inherent nor a permanent aspect of the person" (Trevino, 2003, p. 37). The index of social strain that we use makes no assumption regarding the respondent's inherent qualities; it simply reflects the person's varied self-reported perceived experiences of critical, irritating, or other negative interactions with significant others in his or her social milieu. These experiences, we hypothesize, have adverse consequences that linger in the individual's biological repertoire.

Data and methods

The National Survey of Midlife Development in the United States (MIDUS) study was initiated in 1995 to determine how social, psychological, and behavioral factors interrelate to influence mental and physical health. The first wave (1995) collected sociodemographic and psychosocial data on 7108 Americans, ages 25–74 years, from a representative sample of English-speaking, non-institutionalized adults residing in the contiguous 48 states,