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Habitual sleep quality and diurnal rhythms of salivary cortisol and dehydroepiandrosterone in postmenopausal women

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ABSTRACT

Dysregulation of the hypothalamus-pituitary-adrenal (HPA) axis has been suggested as a potential mechanism linking sleep and cardiometabolic disorders. However, the associations of two primary outputs of the HPA axis, cortisol and its antagonist dehydroepiandrosterone (DHEA), with sleep are less well studied. In the Nurses' Health Study II, 233 postmenopausal women provided five timed saliva samples over one day (immediately upon waking, 45 min, 4 h, and 10 h after waking, and prior to going to sleep) to measure cortisol and DHEA. Of these, 209 completed assessment of their habitual sleep patterns using the Pittsburgh Sleep Quality Index (PSQI). We used piecewise linear mixed models to compare cross-sectional associations of slopes reflecting diurnal cortisol and DHEA rhythms with overall sleep quality and with seven sub-components. Overall, we observed no differences in the diurnal patterns of cortisol or DHEA between good versus poor sleepers as assessed by the global PSQI score. However, longer sleep latency was associated with significantly reduced cortisol awakening rise ($p = 0.02$). Poorer subjective sleep quality ($p = 0.02$), shorter sleep duration ($p = 0.02$), and lower sleep efficiency ($p = 0.03$) were associated with slower rate of cortisol decline later in the day. Women reporting daytime dysfunction had a sharper cortisol decline early in the day ($p = 0.03$) but a flattened decline later in the day ($p = 0.01$). The differences in diurnal patterns of DHEA between good versus poor sleepers, though less pronounced, were similar in direction to those of cortisol. Self-reported sleep duration, efficiency, latency and daytime dysfunction were associated with altered diurnal rhythms of cortisol and, to a lesser extent, DHEA. These findings provide support for the interplay between sleep and the HPA axis that may contribute to cardiometabolic disease.

1. Introduction

Epidemiologic data provide strong evidence linking sleep quantity and quality with cardiometabolic outcomes, such as cardiovascular disease and diabetes (Cappuccio et al., 2011; Cappuccio et al., 2010). However, the biological alterations by which disrupted sleep leads to these adverse health outcomes are poorly understood. Sleep plays an essential role in modulating diurnal cycles of various human physiological and behavioral processes, including functioning of the hypothalamus-pituitary-adrenal (HPA) axis (Kalsbeek et al., 2012). It has been suggested that dysregulation of the HPA axis and its basal diurnal rhythms may be an important pathway through which sleep disturbances influence cardiometabolic risk.

The diurnal rhythmicity of the HPA axis is apparent in variations of cortisol secretion across the day, which are thought to optimize glucose utilization and energy metabolism. There is a marked, continued increase in the cortisol level prior to and immediately after awakening, with the peak reached 30–45 min after awakening (Edwards et al.,

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2001; Pruessner et al., 1997; Wust et al., 2000). After the awakening rise, cortisol secretion starts to decrease. Despite fluctuations in the release of cortisol due to varying environmental stimuli (e.g., exercise), basal cortisol secretion continues to decline over the day and such decline tends to get slower later in the day (Edwards et al., 2001; Wust et al., 2000). Prior work has identified specific alterations in cortisol rhythms (e.g., reduced awakening rise, flattened decline) as being unhealthy, given their associations with obesity, diabetes, coronary heart disease, and mortality (Champaneri et al., 2013; Hackett et al., 2014; Kumari et al., 2011; Smith et al., 2005). Dehydroepiandrosterone (DHEA), a precursor steroid for testosterone, provides resilience to the stress response by antagonizing the effect of cortisol (Charney, 2004). DHEA exhibits diurnal rhythms that parallel cortisol (Hucklebridge et al., 2005), and has previously been inversely associated with risk of cardiovascular disease and mortality in some, though not all, studies (Barrett-Connor et al., 1986; Tchernof and Labrie, 2004). The ratio of cortisol to DHEA has been used to indicate the biologic activity of the HPA axis, with higher ratios suggesting greater dysregulation of HPA axis (Phillips et al., 2010; Young et al., 2002).

Existing evidence is mixed regarding the associations between sleep and diurnal cortisol rhythms. Some studies have reported that poor sleep quality is associated with blunted cortisol awakening rise and slower cortisol decline over the day, yielding increased total diurnal cortisol output (Castro-Diehl et al., 2015; Kumari et al., 2009b; Leproult et al., 1997); others have reported no association (Rao et al., 2013; Zhang et al., 2011). However, most studies have focused on a limited characterization of sleep quality, considering either sleep duration or sleep efficiency. No study has evaluated DHEA or cortisol-DHEA ratio. Further, few studies considered these associations specific to women, who may be more vulnerable to sleep disturbances and associated consequences (Cappuccio et al., 2007; Santhi et al., 2016). Therefore, we investigated the associations of self-reported sleep quality, overall and by its sub-components (e.g., sleep latency, duration, disturbances, etc.), with diurnal rhythms of saliva cortisol, DHEA and their ratio in the Nurses' Health Study II (NHSII).

2. Methods

2.1. Study population

NHSII is an ongoing, longitudinal study of US female registered nurses established in 1989. At baseline, 116,429 women, ages 25–42, returned a questionnaire regarding their health and lifestyle. Biennial follow-up questionnaires were mailed to all participants to update their information on disease diagnoses and exposures. We conducted a substudy with additional data collection to address research questions not covered by the main questionnaire. The study protocols for NHSII and nested substudy were approved by the institutional review board of the Brigham and Women's Hospital.

In April 2013, 688 postmenopausal NHSII participants who had previously given blood were invited to participate in the Mind-Body Study (MBS), which aimed to characterize psychosocial stress and explore associated biological alterations using biomarkers. Briefly, women who reported childhood abuse were oversampled to increase the likelihood of experiencing chronic stress. Participants who expressed interest were mailed a consent form, discussing the extensive data collection at multiple time points over a 1-year period through questionnaires and biospecimens (including blood, urine, timed saliva, toenails, hair, stool, and oral microbiome sample). All collections were repeated once 6–12 months apart to assess within-person reproducibility. We used data from the first collection wave, in which 233 women returned the sample kits (including timed saliva collection over 1 day) and completed an online questionnaire on psychosocial stress (including habitual sleep quality). Here, we analyzed data from 209 women who had no missing responses to sleep quality assessment and provided ≥ 4 timed saliva samples.

2.2. Collection and assay of timed saliva samples

MBS participants were mailed a kit containing equipment and instructions for collecting five timed saliva samples during one day: immediately upon waking (before getting out of bed), 45 min after waking, 4 h after waking, 10 h after waking, and just prior to bed. Participants filled out a log to record collection times and indicate whether they ate, drank, brushed the teeth or exercised and whether they felt excited or anxious near the time of each saliva collection (Supplemental Table 1) (Stalder et al., 2016). All samples were refrigerated overnight until shipping by overnight mail the next day with a cold pack to the laboratory. Assays were conducted in the Laboratory for Biological Health Psychology (Dr. Nicolas Rohleder) at Brandeis University using a competitive chemiluminescence immunoassay (CLIA-approved) with high sensitivity. Samples were measured in duplicate, and were repeated if CVs were > 10%. The CVs of blinded QC pools were < 6%. Cortisol is stable in samples with delayed freezing of up to 4 days (Inder et al., 2012).

2.3. Sleep assessment

Within one month after saliva collection, MBS women were sent an link for an online, comprehensive assessment of their sleep patterns using the Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989). PSQI is a 19-item self-rated scale summarizing 7 components of sleep quality and patterns during the past month: subjective sleep quality; sleep latency; sleep duration; habitual sleep efficiency; sleep disturbances; use of sleep medication; and daytime dysfunction. Each component is equally weighted on a 0–3 scale, and the global PSQI score is scaled from 0 to 21, with higher scores indicating worse sleep quality. A global PSQI score > 5 has been shown to have excellent sensitivity (0.896) and specificity (0.865) to indicate clinically relevant sleep disturbances as assessed using a combination of structured interviews, sleep logs and polysomnographic data; a sleep component score > 1 suggests a moderate to severe difficulty in that area (Buysse et al., 1989). The psychometric properties of PSQI have been extensively validated in different populations (Mollayeva et al., 2016). Among MBS participants who completed 1-year follow-up psychosocial assessment, the intraclass correlation coefficient for PSQI was 0.64, consistent with prior evidence that sleep patterns are stable over time (Knutson et al., 2006).

2.4. Assessment of covariates

Birth date and height were assessed on 1989 baseline questionnaire. Information on weight, night shiftwork, smoking and diseases (hypertension, diabetes, sleep apnea) was used from the questionnaire in 2013. Alcohol and caffeine were assessed using validated semi-quantitative food frequency questionnaire (Willett et al., 1985); we used information collected in 2011. At the time of saliva collection, women completed a questionnaire regarding medication use in the past month, including antidepressants, beta-blockers, minor tranquilizers, oral steroids, and DHEA medication. At the time of sleep assessment, women were also evaluated for depressive symptoms using the short-form Center for Epidemiological Study–Depression (CES-D) scale (Irwin et al., 1999) and for anxiety symptoms using the Generalized Anxiety Disorder–7 items (GAD-7) scale (Spitzer et al., 2006).

2.5. Statistical analyses

Saliva collection time was centered at awakening; all subsequent measures were considered with respect to time since awakening. Both cortisol and DHEA were log-transformed due to their skewed distributions. To determine the optimal time points at which inflections in diurnal rhythms occurred, we fit mixed models with piecewise cubic spline functions with 3 knots chosen at the designated collection times

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