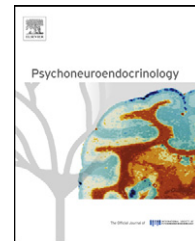




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Seasonal differences in the diurnal pattern of cortisol secretion in healthy participants and those with self-assessed seasonal affective disorder

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Summary This study compared the daily pattern of free salivary cortisol secretion in winter and in summer between two groups; participants with self-assessed seasonal affective disorder (SAD) and age- and sex-matched healthy controls. Fifty-two participants completed the study with an equal number in each group. The diurnal pattern of cortisol secretion was assessed across two consecutive weekdays in summer, and two in winter, with conditions being counterbalanced. On each study day participants collected multiple saliva samples in the domestic setting to capture the cortisol awakening response (CAR) and declining levels across the day. In addition, perceived stress, anxiety, depression, state stress and state arousal were assessed using validated questionnaires. There was no evidence for any seasonal changes in psychological data or cortisol pattern for the healthy control population. In summer, self-assessed SAD and control participants had similar psychological and cortisol profiles. In winter however, SAD participants reported greater depression, stress and anxiety, and lower levels of arousal. Furthermore, the CAR was significantly attenuated in SAD participants during winter months. There was no difference in cortisol levels during the rest of the day between controls and SAD participants in winter. In line with the above findings and previous research, there was an inverse relationship between the increase in cortisol following awakening and a measure of seasonality in winter. Furthermore in winter, a general dysphoria construct correlated inversely with the CAR, indicating that participants reporting greater depression, stress and anxiety and lower arousal, exhibited lower CARs. In conclusion, during the shortened photoperiod in winter, the cortisol response to awakening is attenuated in participants with self-assessed SAD in comparison to controls. These findings contribute to the understanding of the physiology of SAD.

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1. Introduction

Cortisol has a well-established circadian rhythm which is synchronised with the light–dark and sleep–wake cycles.

This rhythm is characterised by an early sleep nadir, gradually increasing concentrations during late sleep, a burst of secretion following awakening peaking at 30–45 min post awakening (the cortisol awakening response or CAR) and a declining pattern thereafter (Weitzman et al., 1971; Edwards et al., 2001). Given this marked rhythm, any study measuring basal cortisol should be synchronised to awakening time. The importance of this rhythm is illustrated by accumulating evidence that aberrant circadian rhythms are associated with physical and psychological disorders, such as depression, post traumatic stress disorder and cancer (e.g. Yehuda, 2002; Linkowski, 2003; Abercrombie et al., 2004). Thus the circadian pattern of cortisol is believed to be a mediator linking mind and body and provides a biomarker of healthy functioning (for use in between-subject and longitudinal within-subjects analyses).

This pattern of cortisol secretion is regulated by the suprachiasmatic nucleus (SCN), located in the hypothalamus (Buijs et al., 2003). The intrinsic rhythms of the SCN are synchronised with the external day-night cycle by light, via the retinohypothalamic tract. The SCN is able to detect seasonal variations in day length and to respond to these differences by making corresponding adjustments in the organism's diurnal and nocturnal physiological states. Numerous studies have described seasonal variations in glucocorticoid release in free-living species such as amphibians, reptiles, birds and mammals (for a review see Romero, 2002). However, research examining seasonal differences in cortisol secretion in humans is limited and presents conflicting results. For example, greater 24 h plasma cortisol (Weitzman et al., 1975) and 24 h urinary cortisol concentrations (Hansen et al., 2001) have been found in winter in studies that have small samples sizes. King et al. (2000) also found higher morning and evening salivary cortisol in autumn and winter, and Persson et al. (2008) in salivary sampling synchronised to awakening, found higher diurnal levels in February, March and April. However, Wehr et al. (1995) failed to observe any seasonal changes in circadian plasma cortisol in summer and winter in men in a laboratory setting, and in a cross-sectional study Lac and Chamoux (2006) found no evidence of a seasonal salivary cortisol rhythm in men. In contrast to all the above studies, higher diurnal cortisol levels have been observed in summer in populations of children (Rosmalen et al., 2005; Matchock et al., 2007). Methodological differences may account for these discrepancies. Clearly more evidence is needed to ascertain whether there are seasonal variations in the pattern of cortisol secretion in healthy populations to inform best practice for the ever increasing number of psychophysiological studies that include cortisol cycles as a variable.

Additionally, although the relationship between elevated cortisol secretion and major depression is well-documented (e.g. Linkowski, 2003), the link between cortisol and seasonal depression is less clearly understood. We recently demonstrated in a non-clinical population that a greater propensity for seasonal changes in mood was associated with a smaller average CAR in winter (Thorn et al., 2009). Individuals suffering with seasonal affective disorder (SAD) experience extreme changes in mood across seasons, characterised by depression in autumn/winter alternating with non-depressed periods in spring/summer (Rosenthal et al., 1984). Most SAD patients also report the 'atypical' depressive symptoms of

hypersomnia, extreme lethargy, overeating, and carbohydrate craving (Sher et al., 1999). SAD may be attributable to decreased daylight hours during winter (Bunney and Bunney, 2000), and indeed the first line of treatment for SAD is light therapy based on the evidence of numerous studies demonstrating efficacy (reviewed by Golden et al., 2005). There is also evidence to suggest that light therapy is more effective when administered in the morning rather than later in the day (Lewy et al., 1998; Terman et al., 2001). Although there is no consensus regarding the underlying pathophysiological mechanisms of SAD, proposed mechanisms include circadian phase shift and retinal subsensitivity to light (see Rohan et al., 2009, for a review). Cortisol secretion has sometimes been found to be phase-delayed in SAD meaning that the acrophase and the nadir occur later than would be expected (Lewy et al., 1987; Avery et al., 1997). The majority of studies examining cortisol rhythms in SAD have relied upon blood sampling. Salivary measurement of cortisol is preferable because it enables repeated collection (allowing for close scrutiny of the dynamics of cortisol secretory activity) without the need for medical personnel, within the domestic setting. Further, the assessment of cortisol in saliva represents the biologically active, 'free' component of the respective unbound hormone in blood (Kirschbaum and Hellhammer, 1994).

As far as we are aware no studies have examined adult seasonal differences in salivary cortisol secretion using a counterbalanced, repeated measures design. To date, seasonal changes in the diurnal salivary cortisol profile and in particular, the CAR have not been measured in a SAD population. The present study aimed to measure the salivary cortisol profile of individuals with self-assessed SAD and healthy controls in both summer and winter. Due to the inconsistency in previous findings we made no specific directional hypotheses regarding seasonal differences in the healthy, non-SAD sample. However with the weight of existing evidence regarding seasonality and SAD we hypothesised that in winter SAD sufferers would exhibit an attenuated rise in cortisol secretion (the CAR) characteristically observed first thing in the morning, relative to a healthy control population.

2. Methods

2.1. Participants

Both SAD and control participants were recruited on the basis that they were healthy, i.e. no medication, no chronic illness, no history of psychiatric illness (other than SAD), no eating or sleep disorder. SAD participants were recruited from a sample of patients with seasonal affective disorder, accessed from the Seasonal Affective Disorder Association (SADA) in the UK. Interest was generated through advertisements on the newsletter and the SADA website (<http://www.sada.org.uk>). All SAD participants reached the criteria for SAD as assessed by Seasonal Pattern Assessment Questionnaire (SPAQ, Rosenthal et al., 1987). Twenty-five out of the 26 participants reported that their seasonal changes were a problem for them and most reported that it was a marked problem. Participants were not clinically assessed for SAD, although 35% reported that they had received a psychiatric diagnosis. All self-assessed SAD participants reported using

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