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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.

1. Introduction

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

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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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Table 1 Demographic characteristics of self-assessed SAD and control participants.

	Control ($n = 26$)	SAD ($n = 26$)
Female participants (%)	15 (58%)	19 (73%)
Age in years (mean ±SD)	48.6 (±11.7)	50.9 (±12.3)
Health rating (mean ±SD)	4.31 (±0.7)	4.04 (±0.8)
Non-smokers (%)	23 (88%)	23 (88%)
Married/cohabiting (%)	19 (73%)	20 (77%)
In full time employment (%)	19 (73%)	16 (61%)
Retired (%)	5 (19%)	7 (26%)
Seasonality score (mean ±SD)	4.3 (±3.1)	15.7 (±3.3)*

* $p < 0.0005$.

light therapy in the winter months. A community sample of age- and sex-matched healthy control participants was additionally screened for SAD using the SPAQ.

Interested participants attended a briefing session at the university or were visited in their own homes. Participants were given a practice session in the self-collection of saliva and were informed about the importance of adhering to the study protocol. Sixty participants were recruited and each signed a consent form. Out of these, a total of 52 participants completed both summer and winter components of the study, with 26 participants in each group. Overall there were 34 females and 18 males, age ranging between 26 and 75 years with a mean (±SD) age of 50 (±12) years. Relative age and sex distributions for each group are given in Table 1. All participants were white European.

2.2. Materials and measures

Participants were provided with a study pack containing full standardised written instructions, questionnaires and saliva sampling kits containing labelled Salivettes (Sarstedt Ltd.). A number of measures were included as cortisol and particularly the CAR has been found to be associated with multiple variables such as awakening time and obligations in the day ahead. Daily record sheets required participants to record awakening time, approximate sleeping time and perceived sleep quality (1–5 likert scale) at the 45 min sample. Participants were also asked to record their expectations of the day ahead using a 100 mm visual analogue scale ("How much of today do you expect to spend fulfilling obligations"). The Stress Arousal Checklist (SACL, Mackay et al., 1978) was also completed to assess momentary state stress and arousal 45 min after awakening. High scores represent greater stress and greater arousal (more alert, active, energetic, less drowsy and sluggish) respectively. Seasonality was measured using the validated Seasonal Pattern Assessment Questionnaire (SPAQ, Rosenthal et al., 1987). The central aspect of the SPAQ is the six item Seasonality Scale Index (SSI) which has possible scores ranging from 0 to 24, with high scores indicating greater changes in mood and behaviour. According to the authors a score from 8 to 11 is indicative of subsyndromal Seasonal Affective Disorder (SAD) and a score of 11 or more indicates that the individual may well suffer from SAD. In addition, in both winter and summer, the Hospital Anxiety and Depression Scale (HADS, Zigmond and Snaith, 1983) provided a state measure of both anxiety and depression. Scores from 8 to 10 on each scale have been taken to indicate possible clinical disorder and scores above 10 indicate probable clinical anxiety. Perceived stress over the previous month was measured using the 14-item Perceived Stress Scale (PSS, Cohen et al., 1983). Demographic variables measured included perceived health, employment and smoking status.

2.3. Procedure

The ethics committee of the University of Westminster approved the protocol for this study. The study was a naturalistic design with participants collecting multiple saliva samples in their home environment in the UK (51°30'N latitude). The study took place during the months of November/December and June/July, with order of season being counterbalanced between participants. As awakening stimulates cortisol secretion, samples synchronised to clock time have been reported to have poor reliability. Therefore, in line with best practice, cortisol was measured in relation to awakening time rather than clock time across two consecutive weekdays (Pruessner et al., 1997; Edwards et al., 2001). Participants were asked to choose two consecutive, normal weekdays during the summer and winter months in which to carry out the study. An identical protocol was applied to each sampling day. Participants were asked to collect saliva samples immediately on awakening, then at 15, 30, 45 min and 3, 6, 9 and 12 h post awakening. During the saliva collection period following awakening, participants were instructed to take nil by mouth bar water and not to smoke or brush their teeth to avoid abrasion and vascular leakage. They were also instructed to take nil by mouth for at least 30 min prior to each of the other samples. SAD participants were asked to refrain from using light therapy on the winter study days. Other than these instructions participants were asked to follow their normal routine. Samples were stored in the participants' home freezer as soon as possible after collection of saliva before being transferred or posted to the laboratory in insulated packs, where they were stored at −20 °C until assay. Cortisol analyses were carried out using a standard enzyme-linked immunosorbent assay protocol (Salimetrics, USA).

2.4. Statistical analysis

Cortisol data were moderately skewed and therefore a square root transformation was applied which normalised distributions. Differences in demographic characteristics and between SAD and control participants were analysed using independent t-tests and Chi-square analyses. Two-way mixed ANOVAs were used to inspect differences in psychosocial data between groups and seasons with the factors season (winter, summer) × group (SAD, control). Four-way mixed ANOVAs were used to examine differences in cortisol secretion between groups across seasons for both the CAR (0, 15, 30 and 45 min samples) and the day samples (3, 6, 9, 12 h samples) separately, with factors season (winter, summer) × day (day1, day2) × sample (4) × group (SAD, control). Composite

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Table 2 Mean (±SD) values for psychosocial variables measured in winter and summer, for self-assessed SAD and control participant.

	Summer		Winter	
	Control	SAD	Control	SAD
PSS	18.9 (±8.8)	23.1 (±6.6)	20.2 (±9.2)	30.0 (±6.9)*
HADS anxiety	6.1 (±3.1)	8.5 (±4.4)	6.4 (±3.5)	11.0 (±3.6)*
HADS depression	3.1 (±2.8)	5.3 (±3.9)	3.4 (±3.3)	8.5 (±4.0)**
SACL stress	3.9 (±4.7)	5.8 (±6.2)	3.8 (±5.0)	10.8 (±4.8)**
SACL arousal	5.5 (±3.2)	5.5 (±3.9)	5.8 (±3.6)	2.8 (±2.9)**

* $p < 0.05$.
** $p < 0.005$.

measures of cortisol were computed for each participant to give the area under the cortisol curve (AUCg) with reference to zero (s2 + s3 + (s1 + s4)/2). The mean increase (MnInc) was also calculated from the awakening sample ((s2 + s3 + s4)/3 − s1) to examine the dynamic aspect of the cortisol awakening response. A mean of samples taken at 3, 6, 9 and 12 h following awakening (the day mean), and the diurnal decline (3 h sample minus 12 h sample) were also calculated. Stability of these composite measures across the two study days was assessed by correlational analysis, as were relationships between composites and psychosocial measures. To clarify relationships between cortisol composites and psychosocial data principal component analyses, with varimax rotation were performed for summer and winter on the psychological variables, which were highly intercorrelated. Relationships between factors and cortisol composites were assessed by Pearson's tests of correlation. Mean cortisol concentrations shown in figures and tables represent original units. In ANOVA analyses reduced degrees of freedom reflect Greenhouse-Geisser correction where the assumption of sphericity was violated. All given values of p are two-tailed.

3. Results

There were no differences in demographic characteristics between self-assessed SAD and control participants (see Table 1). As expected, SAD participants had a higher mean seasonality score compared with controls. The mean scores for psychosocial variables are shown in Table 2. Mixed ANOVAs revealed significant interactions between group and season for each variable. SAD participants reported greater perceived stress, anxiety, depression and state stress in winter, compared to control participants. For the SAD participants in winter their mean scores indicated possible clinical depression and probable clinical anxiety according to Zigmond and Snaith (1983). State arousal, measured at 45 min on each study day exhibited the opposite pattern; lower levels of arousal in SAD participants in winter.

The CAR (samples taken at 0, 15, 30 and 45 min post awakening) and the day samples (taken at 3, 6, 9 and 12 h post awakening) were analysed separately, as they are reported to be distinct aspects of the diurnal cortisol pattern. ANOVA revealed, as expected, a significant main effect of sample for the CAR ($F_{(1.9,97.3)} = 75.915$, $p < 0.0005$, partial $\eta^2 = 0.603$). Importantly, there was a significant three-way interaction between season, sample and group ($F_{(2,100)} = 4.499$, $p = 0.013$, partial $\eta^2 = 0.063$). In winter the CAR was significantly attenuated in SAD participants in comparison to the healthy control participants (see Fig. 1). This three-way interaction accounted for a significant main effect of season ($F_{(1,50)} = 5.662$, $p = 0.021$, partial $\eta^2 = 0.102$) and a significant two-way interaction between sample and group ($F_{(1.9,94.1)} = 3.159$, $p = 0.048$, partial $\eta^2 = 0.059$). There were no other significant main effects or interaction including any effects for day of sampling. Effects involving group, season, or both were not observed in the day samples. Here ANOVA revealed only a significant main effect of sampling time, indicating the

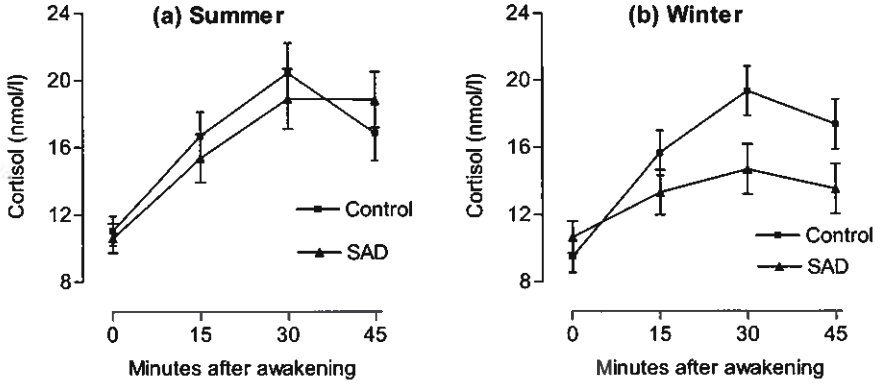

Figure 1 Mean (±S.E.M.) salivary free cortisol concentrations (nmol/l) following awakening in control ($n = 26$) and self-assessed SAD ($n = 26$) participants in (a) summer (b) winter, illustrating the attenuated CAR for self-assessed SAD participants in winter, but not in summer.

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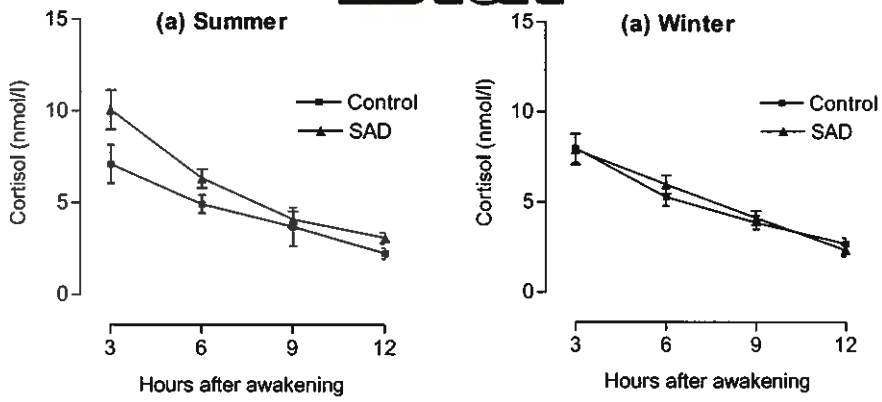

Figure 2 Mean (±S.E.M.) salivary free cortisol concentrations (nmol/l) following awakening in control ($n = 26$) and self-assessed SAD ($n = 26$) participants in (a) summer (b) winter, illustrating no difference in summer or winter for either group.

expected decline in cortisol levels over the course of the day (see Fig. 2).

The data were further explored to investigate whether other variables, previously reported to be associated with the CAR, could explain our reported effects. There were no group, season, or interactive effects for awakening time, sleep quality or score for expectations of daily obligations. Indeed, when these variables as well as order of season and gender were added as covariates to the original analysis, the previously reported three-way interaction remained significant. To check the possibility of confounding effects of smoking status, occupational status, or possible non-adherence to protocol, we also repeated the analysis excluding the small number of participants who were smokers ($n = 6$), not employed (either unemployed or retired) ($n = 14$) and who did not exhibit a CAR on at least one day in winter ($n = 6$). Again, the three-way interaction remained statistically significant.

Correlational analysis of relevant composite CAR measures revealed highly significant correlations between first and second day data for rise in cortisol following awakening (MnInc) for both summer data ($r = .468$) and winter data ($r = .595$). Significant correlations between data for the two days were also evident for the cortisol AUCg measure both in summer ($r = .478$) and winter ($r = .731$). Cortisol composites from data gathered at 3, 6, 9, and 12 h post-awakening (mean and decline) were also consistent over days one and two, (day mean: $r = .667$ in summer and $r = .601$ in winter; day decline: $r = .461$ in summer and $r = .308$ in winter). These associations indicated stability across days in both winter and summer, legitimising the use of average composite values across days in order to check for associations with psychosocial variables.

In line with the above ANOVA findings there was a negative relationship between the increase in cortisol following awakening in winter and a measure of seasonality for the whole group in winter ($r = -.349$, $p = .011$). Participants who reported greater propensity for seasonal changes in mood and behaviour (as assessed by the SPAQ) exhibited an attenuated CAR in winter. There was no equivalent relationship in summer ($r = -.015$, $p = .914$).

In both summer and winter bivariate correlations between the psychosocial variables (state stress and arousal, PSS, HADS anxiety and depression) were high (average magnitude of Pearson's correlation coefficient was .67 in winter and .61 in summer). This suggested the possibility that parsimony could be achieved if we extracted from the data a single factor which might plausibly be called 'dysphoria'. Principal component analyses with varimax rotation were performed for both summer and winter data on the psychological variables to reduce considerably the number of correlational analyses carried out between psychological data and cortisol data. In both analyses the indicators of factorability were good. Each generated a single psychological construct (one for winter and one for summer) with eigenvalues greater than one. All variables had a strong loading (all greater than 0.7), with state arousal exhibiting a significant negative loading. In winter, but not in summer, the general dysphoria construct correlated inversely with cortisol MnInc ($r = -.307$, $p = .032$). No associations between dysphoria and other cortisol composites were evident in any of the data.

4. Discussion

The results of this study demonstrated that participants with self-assessed SAD have different cortisol and psychological profiles in winter but not in summer in comparison to non-seasonal participants. In summer, participants who reported having seasonal affective disorder exhibited a diurnal cortisol profile that was comparable to healthy controls. However, in winter SAD participants evidenced an attenuated CAR despite retaining normal decline in cortisol levels across the remainder of the day. There was no seasonal change in the diurnal pattern of cortisol secretion observed in healthy non-seasonal participants.

Although the evidence for seasonal variations in glucocorticoid release in non-human species including amphibians, reptiles, birds and mammals appears robust (Romero, 2002), the evidence for seasonal cortisol differences in healthy populations is limited and inconsistent. However, in line with the findings for adult male populations (Wehr et al., 1995; Lac and Chamoux, 2006) the current study did not observe any seasonal changes in the post-awakening diurnal cortisol profile. Seasonal cortisol differences have been reported in small populations in blood and urine samples (Weitzman et al., 1975; Hansen et al., 2001), and in saliva samples either not synchronised to awakening (King et al., 2000) or not counter-

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balanced for season (Persson et al., 2008). These methodological differences may explain the incongruence with the findings of the current study. Further, the healthy control population in this study did not report seasonal changes; the mean seasonality score for this group was comfortably below subsyndromal level according to their scores on the SPAQ.

Although the diurnal decline in cortisol levels across the day was similar for SAD and control participants in summer and in winter, in winter SAD participants exhibited an attenuated cortisol awakening response. This seasonal effect on the CAR could not be accounted for by any other variable measured in this study, previously shown to relate to the CAR (e.g. awakening time, sampling day, anticipated obligations in the day ahead and suspected participant non-adherence to protocol). The relationship between seasonality and the CAR observed in the current study complements the findings of a previous study published by our group (Thorn et al., 2009), which demonstrated that the average CAR across two days in winter was negatively correlated with seasonality score, indicating that participants who were more seasonal had on average across both winter days an attenuated rise in cortisol levels following awakening in comparison to those who were less seasonal. Taken together, these results suggest that season is important in studies measuring awakening cortisol secretion (the CAR) incorporating populations reporting seasonal changes in behaviour and mood. The null finding for seasonal differences in the diurnal decline in cortisol for both the control and SAD participants is also noteworthy as it suggests that season of testing can be largely ignored in psychophysiological studies that include this aspect of the cortisol cycle as a variable.

The significant finding for the CAR samples but not the day samples for SAD participants in winter is not unexpected, since previous studies have suggested that the cortisol awakening response is under a different regulatory influence from the remaining diurnal profile (see Clow et al., 2010). In healthy participants the CAR is enhanced by light exposure both pre and post awakening (Scheer and Buijs, 1999; Thorn et al., 2004), whereas cortisol levels later in the day do not appear to be influenced by light (Scheer and Buijs, 1999; Leproult et al., 2001). This may explain why the shortened photoperiod in the winter phase of our study had a greater effect on the awakening samples than on the day samples for the SAD participants. Indeed it is thought that the light sensitive suprachiasmatic nucleus (SCN), which receives light information from the retina, plays a role in the regulation of the CAR in healthy humans, and may enhance cortisol levels following awakening through extra-pituitary neural pathways (Clow et al., 2004, 2010). Interestingly, circadian clock-related polymorphisms are implicated in susceptibility to SAD (Johansson et al., 2003) and a significant genetic influence has been observed for the CAR but not on cortisol secretion over the remainder of day (Wuest et al., 2000). It would be interesting to explore this relationship in future studies.

Given the role of the SCN in the CAR, the attenuated CAR in self-assessed SAD participants in this study could also be explained by retinal subsensitivity to light (Rohan et al., 2009), which has been observed in SAD. In terms of understanding the pathophysiology of SAD, the findings of this study also implicate a circadian rhythm mechanism, in line with previous reports (e.g. Sher, 2004). Circadian rhythms, including cortisol secretion have sometimes been found to be phase-delayed in SAD (Lewy et al., 1987; Avery et al., 1997). The attenuated CAR could be conceptualised as a phase-delay. However, a phase delay would also suggest higher cortisol levels later in the day, not observed in this study. Whatever the underlying mechanism, reduced photoperiod and light availability appear to be involved in the aetiology of SAD and morning light therapy is the established treatment (Rohan et al., 2009). In a recent study, Martiny et al. (2009) demonstrated in patients with non-seasonal major depression that those who had a lower CAR, had a substantial effect of morning bright light therapy compared with dim light therapy, whereas patients with a high CAR evidenced no effect of bright light therapy. It is interesting to speculate that individual differences in light sensitivity, and associated differences in the CAR, may not be limited to those participants reporting SAD (as in this study) but may be more widely implicated in mood disorder.

As well as seasonal differences in the CAR, self-assessed SAD participants reported greater perceived stress, anxiety, depression and state stress in winter, and lower levels of state arousal, in comparison to control participants. These differences reflect the changes in mood experienced by people with SAD. In the whole participant group there were highly significant relationships between psychosocial data, which were expressed in a single dysphoria construct, for both summer and winter. In winter, but not in summer, the general dysphoria construct correlated inversely with the increase in cortisol following awakening, indicating that participants reporting greater, depression, stress and anxiety and lower arousal, exhibited lower CARs. The literature regarding associations between the CAR and psychosocial data is characterised by inconsistencies (Clow et al., 2004). To date, no consensus has been reached about whether a 'healthy' CAR pattern consists of a blunted or elevated awakening cortisol response. A tentative conclusion arising from the findings of this study was that, in terms of both state and trait-like psychosocial variables, an attenuated CAR was associated with poorer psychological health.

A potential limitation of this study is that a structured clinical assessment was not employed. Thirty-five percent of the SAD participants reported that they had received a psychiatric diagnosis; the majority had self-diagnosed. Although the Seasonal Pattern Assessment Questionnaire is not sensitive enough to be considered a diagnostic instrument, all SAD participants scored in the range for possible SAD and importantly, reported significant seasonal changes in psychosocial variables. In winter their mean scores indicated possible clinical depression and probable clinical anxiety. A further limitation is that there was no objective measure of participant adherence to protocol. However, the engagement with and instructions to participants were such that suspected non-adherence to protocol (the absence of a CAR, see Thorn et al., 2006) represented only 7% of study days. Further, the main findings remained significant following exclusion of the small number of participants who were suspected non-adherent in winter. Obviously the current findings do not indicate any direction of causality. It is impossible to determine from the present data whether the attenuated CAR in SAD participants during the winter in any way contributes to the seasonal changes in mood

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observed. However, evidence that light exposure in the morning has been shown to be especially effective in the treatment of SAD (Lewy et al., 1998; Terman et al., 2001) and that light exposure pre and post awakening also enhances the CAR awakening (Scheer and Buijs, 1999; Thorn et al., 2004) may implicate a causal relationship. It is also possible that the withdrawal of light therapy (requested as part of the protocol) caused the observed attenuation in the CAR. Further research is needed to address these issues.

In conclusion we have demonstrated that individual differences in seasonal changes in mood and behaviour are associated with seasonal variation in the CAR but not in cortisol secretion across the remainder of the day. These data underscore the separate nature of the CAR relative to cortisol secretion across the rest of the day and indicate that seasonality is an important factor in research involving measurement of the CAR. Furthermore, these data shed light upon physiological correlates of SAD and possibly the role of the CAR. Further work is indicated to investigate causal linkages between these variables.

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Conflicts of interest

There are no conflicts of interest.

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