Increased Vagal Tone during Winter in Subsyndromal Seasonal Affective Disorder

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Background: *Seasonal affective disorder (SAD) is characterized by recurrent winter depression with summer remissions and/or hypomania. Further symptoms include hypersomnia, increased appetite, weight gain, fatigue, and social withdrawal, which may indicate autonomic changes during winter.*

Methods: *Measurements of respiratory sinus arrhythmia, heart rate (HR), and skin conductance level (SCL) were taken from 32 participants in subsyndromal SAD and control groups (eight male and eight female subjects in each group) in autumn and winter to determine any change in autonomic function. Measures were taken at baseline and during two stressor tasks. Single determinations of blood pressure, sublingual temperature, depression, aerobic fitness, and body mass index were also measured at each session. Replication in a second data collection period over subsequent winter and spring periods was conducted with an additional 32 participants to extend the findings and to counterbalance order effects in testing. Data were combined to produce "winter" and "nonwinter" test periods and statistically corrected for testing order.*

Results: *Respiratory sinus arrhythmia differences indicated that SAD subjects have increased vagal tone in winter. Both groups show a decrease for HR and increases for SCL and diastolic blood pressure in winter.*

Conclusions: *Seasonal affective disorder may show similarities with hibernation, and the results may indicate mechanisms different from those of nonseasonal depression.* Biol Psychiatry 2001;50:28–34 © *2001 Society of Biological Psychiatry*

Key Words: Seasonal affective disorder, seasonality, autonomic nervous system, respiratory sinus arrhythmia, heart rate, vagal tone

Introduction

Seasonal affective disorder (SAD) was first described as a clinical entity by Rosenthal et al (1984), although seasonal changes in affective disorder have been recorded since Hippocrates' time in 400 BC (Wehr 1989). Seasonal affective disorder is characterized by recurrent autumn–winter depressions with spring and summer remissions and/or hypomania. Further symptoms include hypersomnia, fatigue, increased appetite with carbohydrate craving, weight gain, and social withdrawal (Rosenthal et al 1984). Seasonal affective disorder may also be experienced at a subsyndromal level with depressive episodes to a lesser degree. Rosenthal and colleagues' (Rosenthal et al 1984) criteria for SAD include that the depression should not relate to any obvious effect of season-related psychosocial stressors.

Although the etiology of SAD is unclear, the reduced daily photoperiod in the autumn and winter months triggers this disorder in susceptible individuals. The incidence is higher at latitudes of greater than 40° (Rosenthal et al 1984). Neurotransmitters are involved in SAD, with noradrenaline, melatonin, and serotonin levels being reduced in SAD patients relative to normal subjects (Hill 1992; Shafi and Shafi 1990; Skwerer et al 1989). Several physiologic and psychologic variables that are involved in SAD have also been reported to show seasonal variation in the normal population (Lacoste and Wirz-Justice 1989). For example, blood pressure peaks in winter, though resting heart rate (HR) does not vary (Lacoste and Wirz-Justice 1989). Increases in metabolism (Lacoste and Wirz-Justice 1989) and skin conductance level (SCL) (Neumann 1968) have also been shown in winter.

The atypical vegetative symptoms of SAD differ from those typical of nonseasonal depression (e.g., insomnia, decreased appetite, weight loss) (American Psychiatric Association 1994). The symptoms of SAD (e.g., hypersomnia, increased appetite, weight gain) appear consistent with the effect of increased parasympathetic or decreased sympathetic tone. Thus it is possible that SAD patients have an inappropriate autonomic nervous system response to winter onset. Respiratory sinus arrhythmia (RSA) is recognized as a pure measure of parasympathetic nervous

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system activity that is sensitive to behavioral and cognitive changes (Bernston et al 1993; Porges and Byrne 1992). For example, RSA is enhanced with aerobic fitness (de Geus et al 1990), high during sleep, and reduced under conditions of stressful task performance (Porges 1995). On the other hand, HR (e.g., Porges and Byrne 1992), innervated by both the sympathetic and parasympathetic nervous systems, and SCL (e.g., Boucsein 1992), innervated solely by the sympathetic nervous system, typically increase under stressful task performance.

It remains unclear as to why some individuals show an increased vulnerability to the effects of changing seasons. Hobart, Tasmania, with a latitude of 43° south, is ideally situated to conduct studies with the purpose of investigating mood and physiologic arousal across two seasons (autumn and winter, or winter and spring) in subsyndromal SAD participants and in control subjects who show no seasonal disturbance.

The measurement of RSA, HR, and SCL in SAD and control participants in both winter and the other seasons should indicate any differences in autonomic activity. Additional constitutional and autonomic response measures were taken to support the proposition as well as to confirm previous findings. These were body mass index (BMI), systolic and diastolic blood pressure, and sublingual temperature. Aerobic fitness was measured using a bicycle ergometer test to control for any differential effect of fitness on RSA and HR between groups (de Geus et al 1990).

Mood and seasonal symptom ratings were predicted to be greater in SAD for winter and to indicate remission from symptoms during nonwinter periods. Respiratory sinus arrhythmia was predicted to be higher in winter and HR lower in winter for SAD subjects, indicating greater vagal tone. Respiratory sinus arrhythmia was predicted to decrease, and HR and SCL to increase, with cognitive stressor conditions for all participants irrespective of season or group. A higher BMI in SAD for winter was predicted, reflecting the weight gain symptom. A lower blood pressure may be expected for SAD subjects in winter, indicative of increased parasympathetic tone. A winter maximum in skin conductance, blood pressure, and temperature was expected for control subjects to confirm previous findings.

Methods and Materials

Participants

PARTICIPANT SELECTION. Psychology 1 students completed the Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal et al 1984), which screens seasonal change in mood and behavior. The sum of six (sleep length, social activity, mood, weight, appetite, and energy level) five-point scales ranging from 0 (no change) to 4 (extremely marked change) gave a global seasonality score (GSS). Subsyndromal SAD participants were selected on the basis of a moderate to high GSS together with their reported peak of symptoms in the winter months. Participants also satisfied Rosenthal et al (1984) criteria for the SAD symptoms with depressions that did not meet criteria for a major depressive episode (DSM-IV; American Psychiatric Association 1994). Control participants were selected on the basis of a low GSS and not responding with changes in mood and behavior across the seasons.

There were 64 participants, 32 (eight male and eight female in each group) who completed the autumn/winter data collection period and 32 (eight male and eight female in each group) who completed the second winter/spring data collection period. Course credit was gained for participation. All participants were Tasmanian residents for the past 3 years, to control for climatic adjustment at 43° south latitude. Ethical approval was obtained from the university's Human Research Ethics Committee and written informed consent was obtained from participants.

AUTUMN AND WINTER DATA COLLECTION PERIOD. Mean GSSs were 13.94 (SD 2.78, range 9–17) for subsyndromal SAD participants and 4.34 (SD 1.30, range 2–6) for control subjects. Mean ages for SAD and control groups were 19.3 and 20.4 years, respectively. Two control subjects (one male and one female) who recorded no seasonal complaint on the SPAQ were excluded from statistical analyses after scoring high seasonal depression during a laboratory testing session.

WINTER AND SPRING DATA COLLECTION PERIOD. Mean GSSs were 11.88 (SD 2.18, range 8–15) for subsyndromal SAD participants and 3.34 (SD 1.92, range 0–6) for control groups. Mean ages for SAD and control groups were 20.5 and 19.8 years, respectively. Winter results were excluded from statistical analyses for five participants (two control and three SAD) who did not complete the spring testing.

Materials

BECK DEPRESSION INVENTORY WITH ADDENDUM (BDI-ADD). The Beck Depression Inventory (Beck and Steer 1987) is a self-rating scale for depression. An addendum for assessing seasonal depression (Meesters and Jansen 1993; Y. Meesters and J.H.C. Jansen, personal communication, March 29, 1995) included items 12 (social withdrawal) and 17 (fatigability) from the BDI as well as items 16 (hypersomnia), 18 (appetite), and 19 (weight gain), which were inversely formulated.

WEEKLY MOOD INVENTORY (WMI). The Weekly Mood Inventory (Rosenthal et al 1989) is a self-rated seven-point scale developed for the assessment of SAD patients and requesting information about major life events, illness, and mood during the previous week. Factors of Depressed Mood and Ideation (DMI), Atypical Vegetative Symptoms (AVS), Typical Vegetative Symptoms (TVS), and Hypomania Mood and Ideation (HMI) were based on the clinical findings of others. The internal cohesiveness of the factors is high, ranging from .76 to .89 (Rosenthal et al 1989).

PSYCHOPHYSIOLOGICAL MEASURES. Psychophysiology recordings used a MacLab Data Acquisition System configured to record electrocardiograms (ECGs), cardiotachometer, respiration, and SCL. Electrocardiogram standard Ag/AgCl electrodes were filled with electrode gel and attached to the L-rib, R-rib placements and mastoid earth. A BioAmplifier coupler (AOInstruments, Sydney, Australia) with a bandpass filter of 0.3–50 Hz was used for ECG. Beat-to-beat cardiotachometer readings were computed directly from the ECG channel. Ag/AgCl electrodes for skin conductance were filled with electrode gel and attached to the first phalanx of first and third fingers on the subject's nondominant hand. A 0.5-V constant voltage bridge circuit was used to measure SCL, with output of the bridge applied to the MacLab (Lykken and Venables 1971) with a bandpass filter of DC to 10 Hz. A Pneumotrace (AOInstruments, Sydney, Australia) Respiratory Chest Transducer was fitted around the participant's upper chest during full inspiration and then connected to a general purpose amplifier for respiration recording with a bandpass filter of DC to 50 Hz. Sampling rates were 200/sec for the ECG and cardiotachometer channels, 10/sec for SCL, and 100/sec for respiration. Respiratory pacing (see Procedure) used a four-channel interval generator delivering two tones for inspiration and expiration (1 kHz and 1.5 kHz) using Sonalerts. Each complete cycle was 6 sec (10 breaths/min).

CONSTITUTIONAL MEASURES. Height (m) and weight (kg) were measured using a KAWE (Salzkotten, Germany) wall-mounted height measure and Tanita (Tokyo) electronic digital scales, respectively. Blood pressure was measured using a Sein (Koyang, South Korea) portable Digital Blood Pressure Meter (Model SE-2000) and sublingual temperature taken using a Sharp (Osaka, Japan) Digital thermometer (Model MT 20). Submaximal fitness level was assessed by a 6-min period of exercise on a Repco (Melbourne, Australia) bicycle ergometer.

STRESSOR TASKS. The psychophysiologic stressors in the experiment were the Digit Span subtest from the Wechsler Adult Intelligence Scale Revised (WAIS-R; Wechsler 1981) and Stroop Color Word Test (SCWT) (Stroop 1935). After practice with 40 stimuli, 100 stimuli were given in four blocks. Of these, 40 were the same (e.g., the word *red* and red ink) and 60 were different (e.g., the word *red* and blue ink). Difficulty level of the SCWT was varied according to response time allowed for each trial: the easy condition allowed 2500 m/sec and the hard condition 1500 m/sec.

Procedure

Participants were tested individually. The laboratory was sound attenuated and temperature controlled (24°C). Half of the participants were tested in the morning (8:30 AM to noon) and half in the afternoon (2:00 PM–5:30 PM) to control for any circadian differences in physiologic and performance measures. Counterbalancing was across group and gender. Participants were requested to refrain from eating and caffeine or nicotine ingestion for 1 hour before testing. Compliance was checked when each subject presented at the laboratory. Each participant in the first data collection period was tested twice, in autumn and then again in winter. The second data collection period was conducted the following year, with each participant tested first in winter and then again in spring. Each testing session was conducted within a 14-day period as close as possible after the autumn equinox, winter solstice, or spring equinox according to the testing session. All participants were tested at the same time of day for their second session.

The experimenter recorded psychophysiologic responses from an adjoining room. Height and weight measurements were taken. Heart rate and SC electrodes were attached, and the respiration band fitted around participant's upper chest. The participant then relaxed in an armchair and electrode leads were connected into MacLab. Blood pressure and sublingual temperature were taken and the participant completed BDIadd and WMI. Breathing was paced to 10 breaths/min throughout the session. A visual check of the respiratory record ensured compliance with respiratory pacing.

Baseline physiologic measurements were recorded for a period of approximately 2 min, and then tasks (SCWT and Digit Span) were given in counterbalanced order. Standard instructions from the WAIS-R manual were given for the Digit Span subtest. At completion of stressor tasks, the respiration band and finger electrodes were removed and fitness determined. The workload on the bicycle ergometer was set according to a participant's usual level of physical activity. Heart rate was monitored during the 6-min submaximal fitness test.

Data Scoring

Body mass index was calculated using the formula mass (kg)/height^2 (m). Respiratory sinus arrhythmia was calculated using the peak-trough method (Porges and Byrne 1992): the difference between maximum and minimum HRs associated with each respiratory cycle and averaged over five consecutive breaths was used as an index of mean RSA. Mean HR and SCL were taken from the same five-breath period. A measure of O_2 uptake was calculated from the last 30 sec HR in the submaximal fitness test using a nomogram according to Åstrand (1960).

Results

Analyses

The winter data from both collection periods were combined, and autumn and spring data were also combined, to provide a comparison between a winter and nonwinter assessment period. For depression measures (BDIadd and WMI), 2 × 2 (Group × Season) mixed analyses of variance (ANOVAs) were performed. For RSA, HR, and SCL, preliminary analyses showed no significant differences between the four cognitive stressors (easy and hard conditions of SCWT, Forward and Backward Digit Span). The physiologic responses were averaged for the four stressors to give a baseline measure and the average physiologic physiological response for the stressors, and analyzed by 2 × 2 × 2 (Group × Season × Task Level) ANOVAs. For constitutional measures (systolic and dia-

Table 1. Means and SDs for Self-Rated Depression (Beck Depression Inventory with Addendum) and Weekly Mood Inventory Subscales for Subsyndromal Seasonal Affective Disorder (SAD) and Control Groups in Winter and Nonwinter

	Winter				Nonwinter			
	Control		SAD		Control		SAD	
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Beck Depression Inventory with Addendum	4.46	(4.29)	14.03	(9.39)	5.00	(5.16)	7.90	(5.81)
Weekly Mood Inventory								
Depressed Mood and Ideation (6 items)	6.64	(5.36)	14.14	(8.28)	8.50	(7.29)	11.21	(7.04)
Typical Vegetative Symptoms (5 items)	6.64	(6.14)	11.45	(7.90)	6.79	(6.62)	12.00	(5.79)
Atypical Vegetative Symptoms (5 items)	7.93	(4.34)	15.79	(6.13)	8.14	(4.19)	13.10	(5.65)
Hypomania Mood and Ideation (10 items)	36.61	(7.55)	27.41	(13.00)	37.32	(8.75)	34.31	(9.19)

Weekly Mood Inventory ratings were made on a seven-point scale and totaled for each factor. $n = 28$ control and 29 subsyndromal SAD participants.

stolic blood pressure, sublingual temperature, fitness, and BMI) 2 × 2 (Group × Season) ANOVAs were performed. The α level was set at .05 for all analyses. Where analyses revealed a significant interaction, post hoc two-tailed *t* tests were performed to identify the source of interaction with Bonferroni adjustment (.05/*n*) used (Maxwell and Delaney 1990). Between-group differences and season differences were tested with independent sample and paired sample *t* tests, respectively. Order effects for season were further removed statistically to ensure there were no systematic effects from winter or nonwinter testing by including a factor for order in the statistical model (Maxwell and Delaney 1990). Significant effects for order were detected in the AVS scale of the WMI, HR, RSA, and systolic and diastolic blood pressure. Reported *F* values indicate values after statistical adjustment of any order effects were made. All other dependent variables were nonsignificant for order.

Psychologic Analyses

Self-rated depression scores for BDIadd and WMI are shown in Table 1. For BDIadd, ANOVA revealed a main effect for Group [$F(1,55) = 19.16$, $p < .001$]. Seasonal affective disorder participants rated depression significantly higher than control subjects irrespective of season. There was a main effect for Season [$F(1,53) = 8.89$, $p = .004$], indicating that significantly greater depression ratings are recorded for winter than for nonwinter. A significant Group × Season interaction [$F(1,55) = 12.50$, $p = .001$] showed SAD and control groups to vary differentially across season. Post hoc *t* test analyses show that SAD subjects rate depression significantly greater than control subjects in winter [$t(55) = 4.98$, $p < .001$] and are significantly more depressed in winter than in nonwinter [$t(28) = 3.56$, $p = .001$], as shown in Table 1. Control subjects do not differ significantly across seasons. These differences remain significant after applying the Bonferroni adjustment ($\alpha = .05/4 = .0125$).

With ANOVA, significant differences were shown between SAD and control groups for DMI [$F(1,55) = 13.41$, $p = .001$], TVS [$F(1,55) = 11.51$, $p = .001$], AVS [$F(1,55) = 32.23$, $p < .001$], and HMI [$F(1,55) = 8.93$, $p = .004$]. From Table 1, the SAD group rated DMI, TVS, and AVS significantly higher than control subjects, whereas HMI was rated significantly lower by the SAD group. For AVS and HMI, ANOVA also shows main effects for Season [$F(1,53) = 4.08$, $p = .049$, and $F(1,53) = 5.26$, $p = .023$, respectively]. Atypical Vegetative Symptoms are rated significantly higher and HMI significantly lower in winter. For AVS there was a significant two-way interaction between Group and Season [$F(1,53) = 5.40$, $p = .024$], indicating the groups vary differentially across seasons. Post hoc *t* test analyses show significant group differences. Seasonal affective disorder subjects rate symptoms significantly higher than control subjects for both winter [$t(55) = 5.57$, $p < .01$] and nonwinter [$t(55) = 3.76$, $p < .01$] (Table 1). These seasonal differences remain significant after the Bonferroni adjustment is applied ($\alpha = .05/4 = .0125$). There is also a strong seasonal tendency for SAD subjects to rate symptoms higher in winter than in nonwinter [$t(28) = 1.939$, $p = .06$].

Psychophysiologic Analyses

RSA. There was a significant decrease in RSA between base and stressor tasks [$F(1,161) = 14.15$, $p < .001$], and RSA is significantly lower for stressor tasks (mean = 13.76, SD = 5.23) than baseline (mean = 15.67, SD = 7.15). Analysis of variance also showed a significant two-way interaction between Group and Season [$F(1,161) = 6.37$, $p = .01$]. Seasonal affective disorder and control groups varied differentially in RSA. This interaction is shown in Figure 1.

Post hoc *t* test analyses were performed between groups and seasons. Figure 1 shows SAD subjects to have higher RSA for winter (mean = 15.95, SD = 4.44) than for nonwinter (mean = 13.52, SD = 5.36) [$t(28) = 2.34$, $p = .026$]. This difference for control subjects is nonsignifi-

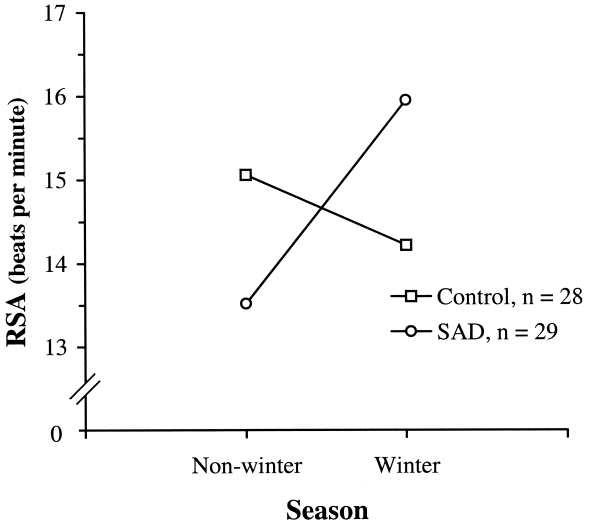

Figure 1. Group mean respiratory sinus arrhythmia during nonwinter and winter for subsyndromal seasonal affective disorder (SAD) and control participants. RSA, respiratory sinus arrhythmia.

cant. The differences between groups in winter and in nonwinter were nonsignificant. When the conservative Bonferroni adjustment is applied ($\alpha = .05/4 = .0125$), the difference in RSA for SAD subjects from winter to nonwinter is no longer significant.

HR. The main effect for Task Level [$F(1,161) = 35.35$, $p < .001$] indicates a highly significant increase in HR from baseline (mean = 73.90, SD = 10.79) to stressor tasks (mean = 79.13, SD = 9.99) (Figure 2).

The main effect for Season [$F(1,158) = 3.89$, $p = .05$] indicates that HR is significantly lower for winter

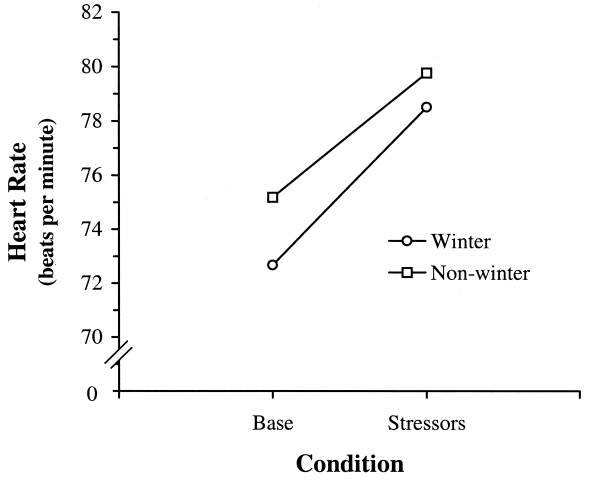

Figure 2. Group mean heart rate (beats/min) during winter and nonwinter for subsyndromal seasonal affective disorder and control participants combined.

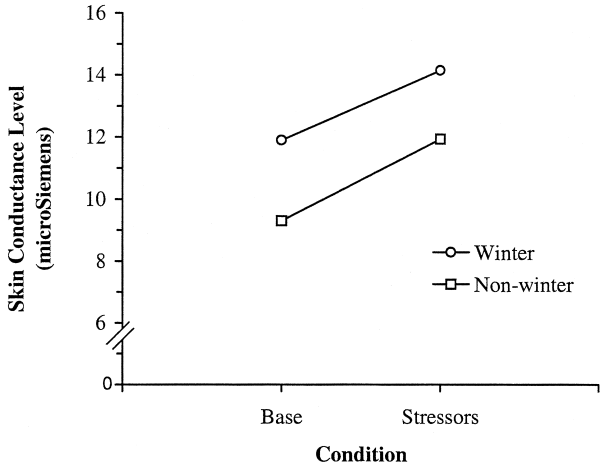

Figure 3. Group mean skin conductance level during winter and nonwinter for subsyndromal seasonal affective disorder and control participants combined.

(mean = 75.59, SD = 10.17) than for nonwinter (mean = 77.47, SD = 10.60).

SCL. The main effect for Task Level (Figure 3) [$F(1,158) = 22.64$, $p < .001$] indicates that SCL increases significantly from base (mean = 10.62, SD = 6.26) to stressors (mean = 13.04, SD = 6.58) (Figure 3).

The main effect for Season [$F(1,158) = 21.82$, $p = .001$] indicates SCL is higher for winter (mean = 13.03, SD = 6.94) than for nonwinter (mean = 10.63, SD = 5.89).

Constitutional Variable Analyses

For diastolic blood pressure, ANOVA showed a significant main effect for Season [$F(1,53) = 5.45$, $p = .023$]. Diastolic blood pressure is significantly higher in winter (mean = 74.96, SD = 8.15) than in nonwinter (mean = 73.23, SD = 6.97). Analyses of systolic blood pressure, sublingual temperature, BMI, and submaximal fitness showed no significant main effects or interactions.

Discussion

Seasonal affective disorder participants reported significantly higher depression and seasonal symptom ratings than control subjects for winter than for nonwinter. Significant interactions for BDIadd and the WMI factor for SAD symptoms, AVS, show higher ratings for depression and associated symptoms for SADs in winter. Atypical Vegetative Symptoms showed a nonsignificant seasonal tendency for SAD subjects to rate symptoms higher in winter than in nonwinter. Typical Vegetative Symptoms, the scale for nonseasonal symptoms of depression, did not differ between seasons.

The significant interaction between group and season for RSA, together with the significant main effect for season for HR, offer support for the hypothesis that SAD subjects have higher RSA, or greater vagal tone, in winter. The significant interaction for RSA is due to the significantly higher winter scores for SAD subjects. However, after Bonferroni correction this effect became nonsignificant and should be replicated as the type 2 error rate is greatly increased by Bonferroni. Main effects for Task Level show RSA to decrease, whereas HR and SCL increase significantly from baseline to stressors for both groups as expected. Combined groups show a decrease in RSA and increases in HR and SCL in winter.

Seasonality is evident for control subjects showing a lower HR in winter. Respiratory sinus arrhythmia does not vary between seasons for control subjects. These differences in RSA and HR cannot be accounted for by fitness level. Submaximal fitness tests found no significant differences between SAD and control groups or between seasons. Seasonality is also evident in SCL and blood pressure. A winter peak has been shown previously in SCL (Neumann 1968; Venables and Christie 1973) and is supported by the present study. Blood pressure has also been shown to peak in winter for normal subjects (Lacoste and Wirz-Justice 1989). The present study shows diastolic blood pressure to be higher in winter. No differences were shown for systolic blood pressure.

Several implications can be made from the study. First, it indicates that SAD subjects have increased RSA in winter. This increase in vagal tone may reflect the sleep symptom in SAD, thus showing a similarity with the hibernation process. Seasonal affective disorder has been compared to animal models of hibernation (Rosenthal et al 1984), with similarities including hypersomnia, overeating, change in food preference, and weight gain. Although this does not imply similar underlying mechanisms, the comparison may assist in identifying similarities and differences in SAD and hibernating mechanisms. Burlington and Milsom (1989) have identified the parasympathetic nervous system as having an involvement in the hibernation process. Further, a suppression of sympathetic activity during hibernation has been reported in Syrian hamsters (Wade 1989) and is possibly implicated for SAD participants in the present study. On the other hand, nonseasonal major depressive disorder has been associated with *decreased* vagal tone (Balogh et al 1993), which may indicate different mechanisms between the two disorders.

Second, the reduction in HR during winter suggests that, for SAD participants, this is consistent with increased vagal tone but may also be due to reduced sympathetic nervous system arousal. Previous research indicates that disruption of noradrenergic pathways (Shafi and Shafi 1990) may implicate noradrenaline in the pathogenesis of SAD. The present finding supports several other research findings. These are 1) low levels of noradrenaline in SAD subjects relative to normal subjects during winter (Hill 1992; Shafi and Shafi 1990; Skwerer et al 1989), 2) significant positive correlations reported between increases in noradrenaline after light therapy and decreases in seasonal depression (Skwerer et al 1989), and 3) no improvement (Hill 1992) or a worsening (Schlager 1994) in winter depression found after treatment with β-blockers.

A third implication from the study relates to the seasonality shown in variables for all participants regardless of group. Lower HR was shown for winter, whereas SCL and diastolic blood pressure were shown to peak for winter, suggesting caution is required when comparing these variables across seasons. Research across four seasons is necessary to provide data showing the extent of seasonal changes in physiologic responses for control subjects, thus extending our knowledge about circannual changes.

In conclusion, SADs have increased vagal tone in winter. Comparison with the hibernation process in animals may help explain SAD and show that SAD is distinct from nonseasonal depression. Further research across a year-long period is being conducted with additional measures to confirm and extend the differential physiologic response changes across groups and find out which would support the view of the syndrome as being a disorder distinct from nonseasonal depression.

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