

Light Treatment and Biological Rhythms

Bulletin of the Society for Light Treatment and Biological Rhythms



Volume 11, Number 1

November 2000

President's Message

Sonia Ancoli-Israel, PhD

I feel honored to take over as President of SLTBR, honored to follow in the footsteps of Dan Oren, Ray Lam, Chris Gillin, Anna Wirz-Justice, Michael Terman, Norm Rosenthal and Al Lewy. It is nice to know that I have such a wise team of advisers to turn to as I begin my new term.

For those of you who missed the 12th Annual Meeting in Evanston, Illinois, you missed a great meeting. Anthony Levitt did a superb job organizing a terrific scientific meeting. Highlights included a keynote address by Anna Wirz-Justice on "Light, Melatonin, Thermoregulation and Sleep," the ALPCO/Buhmann Distinguished Lecturer Presentation by Ben Rusak on, "Molecular, Cellular and Systemic Effects of Light on Rodent Circadian Systems," and the SLTBR Young Investigator Award and Research Presentation, sponsored by Apollo Light Systems. This year's winner was Katherine Sharkey who presented her study, "Phase-Advancing Human Circadian Rhythms with Melatonin."

The status of our organization is stable. Our finances remain on a steady course, with an overall yearly profit of approximately \$8,000. In keeping with the timing of our meetings and membership drives, we are changing our fiscal year to October-September.

Our offices have moved from New Haven to San Francisco. Please note our new address, phone number and email address (PO Box 591687, San Francisco, CA 94159-1687; 415-876-0716). We welcome Kathy Matikonis as our new executive director and look forward to many years of working together. Kathy comes to us with 15 years' experience in medical association management. She has served as executive director of the California Society of Internal Medicine and holds a Bachelors Degree in English and a Masters Degree in special education from the University of Colorado.

Our board continues to be active. Juan Terman has completed her tenure and we want to thank her for her contributions to the society. Barbara Parry has joined the board. At its annual meeting, the board voted to add the positions of membership chair and of Webmaster as voting members of the board. Ray Lam continues on as our Webmaster. Paul Arbisi's term as U.S. Membership Committee chair is complete and we are still looking for a new U.S. membership chair, so if any of you is interested in this very important position, please let me know.

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President's Message (continued from page 1)

We are beginning a membership drive, as one of my goals as president is to increase membership. Please talk to your colleagues and encourage them to join SLTBR. We may be a small organization, but our science is excellent and our interactions stimulating. Talk to your students and have them join, for they are our future. You received the new directory in your mailboxes. Look through it and let us know who is missing so we can send them membership information.

One of the benefits of membership is our quarterly bulletin, *LTBR*. The newsletter is returning to our hands and will no longer be co-published with the Society for Research in Biological Rhythms. Alex Neumeister and Robert Levitan will now edit *LTBR*. Please send in articles, commentaries, etc. We want to hear from you.

For next year's meeting, we are planning to return to Europe. Bengt Kjellman is working on our meeting to be held in Stockholm, beginning on June 25, 2001 just prior to the World Biological Psychiatry conference in Berlin. We hope that our European colleagues who find it difficult to come to the States each year will join us in Sweden, and we hope our American colleagues will take this opportunity to meet in the Nobel Prize Hall (we may or may not ever get an invitation to the Nobel Hall again!).

Let me close by repeating what Dan Oren said at the business meeting in Evanston. Who are we? We are a volunteer organization and like all volunteer organizations we have more work than people. If any member would like to volunteer or to be more involved, the board welcomes the help. Please let us hear from you.

Looking forward to seeing you in Stockholm in 2001.

Sonia Ancoli-Israel, Ph.D.
Professor of Psychiatry
University of California, San Diego

SLTBR 13th Annual Meeting

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Nobel Forum, Stockholm

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LIGHT TREATMENT FOR DEPRESSION IN SUMMER AND WINTER

Daniel F. Kripke, MD

There are patients who may be depressed to the extent of a major depressive disorder in winter who do not recover completely in summer. Such patients may remain dysthymic or in a summer state of residual or minor depression. Patients with summer symptoms do not meet formal criteria for Seasonal Affective Disorder or DSM-IV seasonal trend, since they do not fully recover in summer, yet the probability is high that they are light-responsive.

Since by definition, we are dealing with a rather chronic depressive illness, we should concede at the outset that there are no long-term controlled trials of any treatment for this particular group of patients: no long-term trials of antidepressant drugs, no long-term trials of psychotherapy, and no long-term trials of light treatment. Thus, the clinician must judge the wisest course based on evidence that is not fully conclusive.

Our available information is that antidepressant drugs are useful for most depressed patients, and antidepressants are effective for chronic depressions. Bright light treatment is useful for seasonal and non-seasonal depression (Kripke, 1998). Specific brief psychotherapies, e.g., cognitive therapy, are also useful for depression. Fortunately, it appears that bright light treatment and antidepressant drugs work well when they are combined, which is often the ultimate clinical choice in patients initially identified as classic cases of SAD (Schwartz, et al., 1996).

With these considerations, for the patient with persistent year-round depression, I usually recommend a combination of long-term bright light treatment and a maintenance antidepressant drug. I offer short-term psychotherapy in addition, if the patient has not already received a likely maximum benefit. My experience is that persistent combined therapy often produces effective control of chronic and difficult depressions.

Kripke, D.F. Light treatment for nonseasonal depression: speed, efficacy, and combined treatment. *J. Affect. Dis.* 49:109-117, 1998.

Schwartz, P.J., Brown, C., Wehr, T.A., and Rosenthal, N.E. Winter seasonal affective disorder: A follow-up study of the first 59 patients of the National Institute of Mental Health Seasonal Studies Program. *Am. J. Psychiatry.* 153:1028-1036, 1996.

LIGHT THERAPY AND BIOLOGICAL RHYTHMS IN THE NEW MILLENNIUM: An Assessment of Critical Issues in the Field

Robert D. Levitan, MD

This year's annual meeting identified three major areas of interest for the new millennium, including Melatonin, alternatives to light therapy, and general biological research. The following is a summary of recent highlights and future directions in these areas:

Melatonin

As outlined by Wehr et al, the human retino-hypothalamic/SCN tract is able to detect and respond to photoperiod, with several potential implications for SAD. In animals, the pars tuberalis has a high density of Melatonin receptors and one potential area of human work is to identify analogous receptors in the human system. Another important goal is to delineate the particular signaling mechanism at the level of the SCN, and how it might operate in SAD.

Regarding the therapeutic use of Melatonin, controlled studies to fully assess toxicity is of utmost importance. There needs to be further work on possible interactions with medical illnesses such as liver disease. There is also the issue of possible photosensitizing effects. Across Melatonin studies there is a need for uniform reporting of side effects so that a toxicity profile can be accurately delineated.

Going forward in time, there is the possibility of creating analogues of Melatonin, which might act at different receptor subtypes in various brain areas. One area of work for which there is preliminary evidence relates to the use of Melatonin as a sedative and as a phase shifter; however, more work on both needs to be done in both normal and clinical populations.

Alternatives to Light Treatment

A number of positive developments in the past few years were mentioned including improvements in dawn simulation, which is now more convenient, well accepted and has improved compliance overall versus light. Similarly, there is an improved understanding of the use of visors and light masks that enables higher levels of activity in patients undergoing treatment. Finding particular wavelengths

Continued next page

Levitan (continued from page 3)

of light, which might have less ocular hazards, for example green light or low intensity red light, is an important area of past and future work. Given the important findings relating to the circadian system delineated in this annual meeting, the ability of various types of light to affect the SCN would be a natural focus for future work.

Going forward in time, further verification and optimization of dawn simulation is needed. A key issue in this regard is the cost of clinical trials for new devices and the need for large sample sizes to validly assess them. For all light therapy devices, we need to assess the way people actually use them at home as opposed to the ideal delineated in our grant proposals. The effects of various types of light treatment on quality of life will also be an important area paralleling similar work in other mood disorders.

Establishing standards for various types of light boxes and devices will be important for both clinical and research purposes, and the development of more pragmatic light therapy trials is needed. One example is the possible use of special room lighting as opposed to a light box the individual sits close to.

General Psychobiology

Paralleling the psychiatric genetics field in general, there is a need to establish the optimal phenotype for genetic studies of SAD and seasonality and where possible, to develop endo-phenotypes based on biological markers such as depletion and/or agonist studies. Using light sensitivity and hypothalamic responses to light using functional MRI were also proposed as highly novel phenotypic strategies for genetics work. In performing genetic studies it will be important to implement family based controls to minimize the effect of population stratification. Other genetic strategies will include identifying new polymorphisms related to Melatonin and the biological clock, and assessing whether particular genotypes have actual functional significance. Finally, looking at gene-environmental interactions will be an important component of future genetics work.

Regarding biochemical studies, much progress has been made in the area of serotonin, and more recently norepinephrine. A more complete and systematic study of dopaminergic contribution to SAD and possible stimulant effects of light would complete our understanding of the monoamine transmitters in SAD.

Other brain systems that were mentioned include CRH, which is an activating neuropeptide, and opiate systems that play a role in hedonic responses and may contribute to the carbohydrate cravings seen in SAD.

Regarding the biology of light, an important priority is to establish an action spectrum for Melatonin suppression based on different wavelengths of light, matching this with treatment response. Regarding other mechanisms contributing to seasonality and SAD, work on cognitive factors in SAD and possible use of CBT strategies was alluded to, paralleling work in other forms of depression.

In summary, we have come a long way in the past sixteen years in understanding the vulnerability factors and optimal treatment programs for Seasonal Affective Disorder and seasonality. As outlined above, there is much work to be done going forward. Once you have finished reading this bulletin (every last word!), please proceed directly to your lab.

*Robert D. Levitan, MD
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CONTEMPORARY ISSUES IN THE EPIDEMIOLOGY OF SAD AND SEASONALITY

Gregory W. Murray¹, Raymond W. Lam, Andres Magnusson, Peter Paul A. Mersch, Anthony J. Levitt

This paper presents the major arguments from a recent symposium "Controversies in the Epidemiology of SAD and Seasonality" organized by SLTBR for the International Congress on Chronobiology, Washington, DC, 1999. Emerging and contentious issues in the field were the primary focus of the symposium.

Epidemiology of SAD and the importance of seasonality

Although Seasonal Affective Disorder (SAD) (Rosenthal et al., 1984) is described in DSM as a variant of categorical mood disorder, it is typically understood as the extreme end of a continuum of normative seasonal variation in mood and behavior (Hardin et al., 1991, p.75). It has in fact been suggested that the tendency towards seasonal variation in mood ("seasonality") may have more empirical support than categorical SAD (Bauer & Dunner, 1993). The operationalization and measurement of seasonality is therefore an important element of the epidemiology of SAD.

Retrospective measurement

The majority of epidemiological research into SAD has measured SAD/seasonality on the Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal, Genhart, Sack, Skwerer, & Wehr, 1987). The SPAQ is a retrospective self-report instrument, originally designed to screen for SAD. A major strength of the instrument is that it is a brief questionnaire with good face validity. It has therefore been widely used (e.g. Blacker, Thomas, & Thompson, 1997; Booker & Hellekson, 1992; Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989; Magnusson & Axelsson, 1993; Rosen et al., 1990; Terman, 1988; Thompson, Stinson, Fernandez, Fine, & Isaacs, 1988) and provides an invaluable reference point for seasonality research in different locales. SPAQ-based studies have estimated SAD prevalence in North America to range between 0.8% and 9.7% across various regions (Lam & Levitt, 1998). Significant levels of seasonality have been reported by 10-20% of SPAQ respondents (Bauer & Dunner, 1993). Across a wide range of temperate latitudes,

SPAQ responses overwhelmingly suggest that the most-common *pattern* of seasonality is one of lowered mood in winter and relatively elevated mood in the warmer months (particularly summer).

The limitations of retrospective estimation of seasonality are commonly noted, especially by authors of prospective studies (e.g. Harmatz, Well, Overtree, & Kawamura, 1999; Harris & Dawson-Hughes, 1993; Murray, Allen, & Trinder, submitted-a; Nayyar & Cochrane, 1996; Schlager, Schwartz, & Bromet, 1993; Suhail & Cochrane, 1997). Possible confounds include preconceptions (Eastwood & Peter, 1988; Stiles, Barkham, & Shapiro, 1993) and restrictions on long-term recall (Wicki, Angst, & Merikangas, 1992). Studies based on the SPAQ may also exaggerate seasonality in another way: people who believe themselves to be non-seasonal may be systematically less likely to volunteer to complete the SPAQ (Kasper et al., 1989; Magnusson, in press; Sakamoto, Kamo, Nakadaira, Tamura, & Takahashi, 1993). Consistent with this assertion, Mersch has highlighted that in two regions, telephone interviews have generated substantially higher response rates, and substantially lower prevalence estimates (Mersch, 1999; Mersch, Middendorp, Bouhuys, Beersma, & van den Hoofdakker, 1999b). A growing number of studies conclude that the SPAQ substantially overestimates the prevalence of SAD and the extent of seasonality (Blazer, Kessler, & Swartz, 1998; Levitt & Boyle, 1997; Nayyar & Cochrane, 1996; Raheja, King, & Thompson, 1996)².

Prospective measurement

In prospective designs, season-related effects must compete with other potential sources of mood variation, potentially offering a more valid estimate of seasonality (Terman et al., 1989). Some sixteen published projects have generated prospective data on seasonal mood variation in the general population. These sixteen studies form a heterogeneous set, with a variety of sampling procedures, mood measures and definitions of

² It has been argued that the SPAQ's apparent tendency to overestimate SAD prevalence is attenuated when subsyndromal SAD cases are included in calculations (Magnusson, 1996). Magnusson presents data suggesting that, against the criterion of diagnostic interview, the SPAQ has weak case finding ability largely because of its poor distinction of SAD and subsyndromal SAD categories. Magnusson proposes that the capacity to identify the exact individuals with a characteristic is a more stringent test of an instrument, than is its capacity to identify what proportion of a group are likely to have the characteristic. The SPAQ, he suggests, may be a relatively accurate gauge of SAD at the group level, but relatively inaccurate at the individual level.

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seasonal pattern. Only three published studies have tracked the same individuals across the seasonal year (true longitudinal designs). Of the reviewed studies, only three failed to find significant seasonal trends in mood (Hansen, Jacobsen, & Husby, 1991; Oskarsson, Magnusson, & Axelsson, 1997; Stiles et al., 1993). Among those that identified significant seasonal variation in mood, two identified spring peaks in depression (Lacoste & Wirz-Justice, 1989; Nayha, Vaisanen, & Hassi, 1994) and one identified an autumn peak (Harris & Dawson-Hughes, 1993). The remaining ten papers found the expected seasonal pattern of mood, with winter and summer as its extreme poles (Eagles, McLeod, & Douglas, 1997; Haggag, Eklund, Linaker, & Gotestam, 1990; Harmatz et al., 1999; Mersch, Middendorp, Bouhuys, Beersma, & van den Hoofdakker, 1999a; Murase, Murase, Kitabatake, Yamauchi, & Mathe, 1995; Palinkas, Cravalho, & Browner, 1995; Schlager et al., 1993, (among females and not males); Smith, 1979; Suhail & Cochrane, 1997; Terman et al., 1989). The published prospective data are therefore broadly consistent with retrospective studies, in suggesting that winter pattern seasonality of mood is a widespread phenomenon (Mersch et al., 1999a).

Prospective studies also permit quantification of the seasonal effect. We were able to calculate effect sizes (Cohen, 1988) from four published studies that have identified winter worsening of mood. The maximum estimate of the size of the seasonal effect in each study was 4% (Mersch et al., 1999a), 6% (among females only, Schlager et al., 1993), 3% (Terman et al., 1989), and 1% (in an elderly sample, Eagles et al., 1997). The estimated effect sizes can be described as small to medium in magnitude (Cohen, 1988)³. This effect size analysis of prospective data is consistent with retrospective analyses, which reliably suggest that seasonality is more pronounced among females and decreases in later middle age (Lam & Levitt, 1998).

Repeated cross-sectional versus true longitudinal designs

A peak in cross-sectional incidence does not mean that this pattern exists longitudinally in any individual (Eastwood & Peter, 1988). Only a handful of true longitudinal studies have been conducted in non-clinical samples (viz. Harmatz et al., 1999; Harris & Dawson-Hughes, 1993; Suhail & Cochrane, 1997), none of which collected data across more than one annual cycle. A recent Australian study by Murray and colleagues (Murray et al., submitted-a)

addressed some of the limitations in previous research by investigating seasonality of mood longitudinally across winter and summer for three years in a random community sample. Personality was measured at every time point as a covariate, and the SPAQ was included (at T4 and T5) as a retrospective measure of seasonality. On the basis of contemporary mood theory, Positive Affect (PA) and Behavioural Engagement (BE) were selected as the mood variables most likely to demonstrate seasonality in the normal population (Depue, Arbisi, Spont, Leon, & Ainsworth, 1989; Depue, Krauss, & Spont, 1987; Watson & Clark, 1997; Watson, Wiese, Vaidya, & Tellegen, 1999)⁴. Mood measurements were taken twice a year: winter (July-August) and summer (January-February) over three years. Statistically significant winter lowering of mood was found among the 380 respondents who completed all waves (59.6% of subjects responding to wave 1), but the effect was small in magnitude (2.5% of the total variance in the BE mood scale). Based on SPAQ response (self-report as winter pattern), a subgroup with more robust seasonality was identified. This winter pattern subgroup (36.1% of the sample) exhibited a stronger seasonal effect (up to 7.7% of variance in BE scores).

Not surprisingly, given the size of the seasonal effect, there was little evidence of cross-year stability in seasonality. This finding is consistent with long-term SAD follow-up studies, which suggest that, even among individuals in whom the trait is presumably most pronounced, the course of depressions remains strictly seasonal in less than half (Leonhardt et al., 1994; Sakamoto, Nakadaira, Kamo, Kamo, & Takahashi, 1995; Schwartz, Brown, Wehr, & Rosenthal, 1996; Thompson, Raheja, & King, 1995). The strongest evidence for a stable trait of seasonality in the longitudinal study of Murray and colleagues was therefore not prospective data alone, but the *concordance* between SPAQ report as winter-pattern and elevated prospective seasonality of mood⁵.

Correlates of SAD prevalence/seasonality

If not consensus, there is perhaps a majority opinion that winter pattern seasonal mood variation

³ As a point of comparison, in a recent longitudinal study (Murray et al., submitted-a), the personality trait of Neuroticism measured in summer explained 16.8% of variance in the Beck Depression Inventory Scores in the subsequent winter.

⁴ BE and PA are closely related constructs, both understood as the affective component of a fundamental appetitive motivational system. They stand in contrast to Negative Affect (NA or anxiety) which would not be expected to vary with predictable cycles in the availability of resources. In this study, BE data were collected across three years, PA and NA data across two.

⁵ The validity of this item on the SPAQ ("Month(s) of feeling worst") has also been prospectively affirmed by Mersch and colleagues (Mersch et al., 1999a)

can be discerned in the general population. Having identified the pattern, it is useful to briefly investigate some proposed correlates.

Latitude and environmental factors

Seasonal changes in day length have always been considered important in the etiology of SAD. Two early uncontrolled studies suggested that the photoperiodic effect on mood was so great that a correlation between SAD prevalence/ seasonality and latitude existed (Lingjaerde, Bratlid, Hansen, & Gotestam, 1986; Potkin, Zetin, Stamenkovic, Kripke, & Bunney, 1986). This "latitude hypothesis" has since received substantial research attention.

Levitt and colleagues (Levitt & Boyle, 1997) have reported preliminary findings of a rigorous epidemiological study in Canada, which included a test of the latitude hypothesis. This study was unique in that latitude was sampled rather than haphazardly selected (c/f, e.g. Murray & Hay, 1997; Rosen et al., 1990). Eight strata of 1° of latitude were selected between 41.5°N and 49.5°N in the Province of Ontario. The design was also strengthened by the use of a validated, telephone-based diagnostic interview (the Depression and Seasonality Interview, DSI). The interview determined the presence of major depression, the relationship between episodes and time of year and the seasonality of all depressive symptoms. The SPAQ was also administered. Interviews were distributed across the months of the year. Representation of rural and urban dwellers was proportional, with the restriction that fewer than 20% of a stratum was either rural or urban. Complete interviews were obtained from 1605 respondents, from an initial 4475 telephone contacts.

When strict DSM-IV criteria were applied, the estimated prevalence of SAD was 1.7%. The diagnosis was more prevalent in females than males, and most common in the 30-39 year age range (there was a curvilinear decline in prevalence with age). There was no relationship between latitude and prevalence as estimated by DSM-IV diagnosis (Linear by Linear association Chi square = .411, n.s.), and GSS score showed a small but significant *negative* correlation with latitude ($r = -.05$, $p < 0.05$). This study also provided useful validating data, by way of comparison of SPAQ and DSI diagnoses of SAD. Consistent with other data suggesting that the SPAQ may overestimate SAD prevalence, its positive predictive power was only 13% (of 107 people diagnosed as SAD on the SPAQ, only 14 were diagnosed on the DSI).

Mersch, Middendorp, Bouhuys, Beersma and Hoofdakker (1999b) used prevalence findings (SPAQ-based) from single-latitude and multi-latitude studies to derive a correlation co-efficient for the effect of latitude on SAD prevalence. Studies that used unusual subgroups of the population were excluded from the analysis, leaving seven locales in the USA (Booker & Hellekson, 1992; Kasper et al.,

1989; Levitt & Boyle, 1997; Rosen et al., 1990; Terman, 1988) and six from Europe (Hagfors, Koskela, & Tikkanen, 1992; Hagfors, Thorell, & Arned, 1995; Magnusson & Stefansson, 1993; Mersch, Middendorp, Bouhuys, Beersma, & Van den Hoofdakker, 1995; Wirz-Justice, Krauchi, Graw, Schulman, & Wirz, 1992). When all thirteen regions were included in the calculation, a small and non-significant correlation was found between latitude and prevalence (Spearman's $r_{n=13} = .07$, n.s.). However, when the two continents were investigated separately, a highly significant correlation was found in the USA ($r_{n=7} = .90$, one-tailed $P = 0.003$), while the correlation across the six European locales did not reach one-tailed significance ($r_{n=6} = 0.70$, $P = .061$). On the basis of the small overall correlation, and the marked differences between the European and USA correlations, Mersch and colleagues conclude that the effect of latitude on prevalence (if it exists) must be small, and other factors (e.g. sunlight and cloudiness) may play a role.

There is not complete consensus on the latitude hypothesis, however. As noted by Magnusson, given that seasonal variation appears to be insignificant at the equator, and is discernible at higher latitudes, a latitude gradient must exist (Magnusson, 1999). This gradient may not, however, manifest as a linear relationship between latitude and SAD prevalence/seasonality. Drawing on the seminal research of Aschoff (1981), Magnusson (Magnusson, 1997; Magnusson, 1999) proposes that the latitude gradient may level off at 40°N. This proposition is consistent with the relatively low prevalence estimates for SAD found in Europe, and especially far northern Europe (e.g. Iceland, Magnusson & Stefansson, 1993).

A number of studies suggest that weather and climate may be stronger predictors of SAD prevalence/seasonality than is latitude (see, for reviews, Magnusson, in press; Mersch et al., 1999b). Factors such as weather and climate may mediate the relationship between latitude and perceived photoperiod, and/or have direct effects on mood (Murray & Hay, 1997). There is also some evidence that, at least among SAD patients, levels of depression in winter correlate with daylength and associated meteorological variables (Molin, Mellerup, Bolwig, Scheike, & Dam, 1996; Oren et al., 1994; Young, Meaden, Fogg, Cherin, & Eastman, 1997).

Neuroticism

SAD/seasonality appears to be associated with the general vulnerability trait of neuroticism (N) (Eysenck, 1981; Eysenck, 1990; McCrae & John, 1992). Significant and specific correlations between the SPAQ's GSS and measures of N have been

Vernon, 1997b; Murray, Hay, & Armstrong, 1995), and this covariation has been shown to be largely genetic in origin (Jang, Lam, Harris, Vernon, & Livesley, 1998). Similarly, in the Australian longitudinal study of Murray and colleagues (above), membership of the robustly seasonal subgroup was specifically predicted by levels of N (Odds ratio = 1.05 (1.02-1.08)).

The etiological implications of the relationship between N and seasonality are unclear. For example, the two phenomena might share a diathesis, as suggested by the finding that SPAQ-identified SAD patients are characterized by high levels of symptomatology *across the year* (Broman & Hetta, 1998; Eagles, Naji, Gray, Christie, & Beattie, 1998; Harmatz et al., 1999; Mersch et al., 1999a). Indeed, there is some evidence that serotonergic dysregulation may be a common element in conditions characterised by seasonality (Lam & Goldner, 1998; Tam, Lam, Yatham, & Zis, 1998) and in the physiology of N (Knutson et al., 1998; Lesch et al., 1996). There is also some evidence that N might measure a predisposition to circadian lability (Murray, Allen, Trinder, & Burgess, submitted-b), which might in turn be characteristic of seasonality (Blehar & Lewy, 1990; Bouhuys, Meesters, Jansen, & Bloem, 1998). On the other hand, seasonality and depression-proneness could be phenomena that co-occur in SAD but which do not share a physiological locus (Sher, Goodman, Ozaki, & Rosenthal, 1999; Young, 1999). Further integration of SAD/seasonality with personality theory will help to elaborate hypotheses about the physiological basis of both vulnerability and seasonality (O'Rourke, Wurtman, Brzezinski, Nader, & Chew, 1987; Thompson, Childs, Martin, Rodin, & Smythe, 1997).

Acclimatization and adaptation

It might be expected that individuals who have lived at lower latitudes would be relatively sensitive to seasonal environmental changes in far northern climes -- for example, migrants may take time to adapt to more extreme variations in daylength (Magnusson, 1999; Magnusson, in press). A number of studies have found that new migrants to far northern latitudes do indeed exhibit greater seasonality than natives or long-term residents of these locales (Booker & Hellekson, 1992; Eagles, Mercer, Boshier, & Jamieson, 1996; Low & Fleissner, 1998; Saarijarvi, Lauerma, Helenius, & Saarilehto, 1999; Suhail & Cochrane, 1997; Williams & Schmidt, 1993). While there have also been negative findings (Murase et al., 1995; Nayha et al., 1994), Magnusson has proposed that natives of northern locations may tolerate the winter better than those who migrate there.

Two studies have concluded that (SPAQ-measured) seasonality is a substantially heritable trait (Jang, Lam, Livesley, & Vernon, 1997a; Madden, Heath, Rosenthal, & Martin, 1996). It can therefore

be postulated that ethnic groups may differ in terms of seasonal adaptations (Magnusson, in press). Magnusson has hypothesised that the finding of relatively little seasonality in Iceland (Magnusson & Stefansson, 1993; Oskarsson et al., 1997) may be attributed to genetic adaptation of the population, which has lived in virtual isolation in the north Atlantic ocean for 1000 years. To test this hypothesis, Magnusson and Axelsson (1993) investigated seasonality among a community in Manitoba, Canada, which is of completely Icelandic descent (Axelsson et al., 1990). Consistent with the hypothesis of genetic adaptation among Icelanders, it was found that the SAD prevalence rate was significantly lower among this group (1.2%) than had been found in previous USA samples using similar methodologies (e.g. 9.7% in Nashua, NH, Rosen et al., 1990). The rate was nonetheless lower than the 3.8% prevalence estimated among Icelanders in Iceland itself, which might be consistent with the latitude hypothesis (Magnusson & Stefansson, 1993). These findings have recently been replicated in a study comparing Icelandic descendants in the city of Winnipeg, Manitoba, with other Winnipegian citizens (Axelsson, Stefansson, Magnusson, & Karlson, 1998).

Conclusions

Much remains unknown about the extent, the quality, the pattern and the potential correlates of season-linked mood changes. However, research over the past decade has substantially advanced our understanding of this important process. The majority of relevant studies (both retrospective and prospective) suggest that a seasonal rhythm in human mood can be discerned among the general population living in the temperate zones. The rhythm has summer and winter as its extreme poles, with winter being associated with greater depressed mood. The seasonal effect is small to medium in size, which may explain negative findings in some studies. Equally, stability across time is probably difficult to demonstrate in such a subtle tendency. There is not strong evidence that latitude is a predictor of SAD prevalence/seasonality, with major analyses showing little or no effect. The significance of the apparent relationship between SAD/seasonality and broader vulnerability remains controversial, as does the hypothesis that seasonality might be moderated by acclimatization of individuals and/or adaptation within ethnic groups.

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