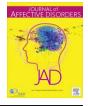
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# Journal of Affective Disorders



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## Has the existence of seasonal affective disorder been disproven?

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Traffanstedt et al. (2016) recently published a study designed to determine if a seasonally related pattern of major depressive disorder could be demonstrated in a cross-sectional, population-wide approach. The eight item days version of the Patient Health Questionnaire (PHQ-8 days; Dhingra et al., 2011), an adaptation of the PHQ-8, was used to detect DSM criteria of depression over 14 days prior to assessment. Data from 34,294 adult respondents, who were interviewed by telephone throughout the year, were used in the final analysis. The authors claim that PHQ-8 scores were unrelated to season, to sunlight exposure on the day of the interview or to latitude of residence. They conclude that their results undermine the validity of the concept of seasonal affective disorder (SAD).

In our opinion, the main problem with the conclusions drawn from these results is overgeneralization. Traffanstedt et al. might have indeed been able to show that PHQ-8 scores do not differ significantly between the seasons in their sample. However, the data from their study is not at all sufficient to disprove the validity of SAD. The authors have investigated apples and try to make inferences on the properties of oranges, so to say.

The report by Traffanstedt et al. demonstrates several methodological and rational shortcomings, which discredit the assumptions the authors make. We would like to discuss these issues in a point-by-point manner.

- 1. A diagnosis of SAD according to the Kasper-Rosenthal criteria (Kasper et al., 1989; Rosenthal et al., 1984) as well as the criteria of the ICD-10 (World Health Organization, 1991) and the DSM-5 (American Psychiatric Association, 2013) is always based on patient history. Traffanstedt et al. did not collect longitudinal data, especially on the medical history and prior affective episodes of their study subjects. Their assumptions are merely built upon a cross-sectional analysis of the present symptomatology. This would be akin to diagnosing a developmental disorder by a cross-sectional snapshot.
- 2. The treatment status of the depressed patients was not taken into consideration. This is a serious flaw as it might well be, and cannot be excluded based on the published data, that the SAD patients assessed were effectively treated and thereby not recognized by the methods employed in this study. Likewise, many SAD patients with a long-standing history of the disorder possess adequate coping strategies to deal with the symptoms and would also have been missed.
- 3. Although the study included several thousand participants, it may still have been under-powered to detect a seasonal variation in mood because the interviews were performed randomly throughout the year, so the chance of catching a symptomatic SAD patient was low. Therefore, patients with SAD assessed during non-winter months were not included as such in the analysis, but as healthy individuals, hereby greatly distorting the prevalence of SAD in their sample and diluting a potential effect that SAD patients might have on PHQ-8 data. The factor for dilution can be approximated by 365 days ÷ (length of symptomatic seasonal depression in days). The length of untreated SAD according to clinical experience is typically 3–4 months and is even less in treated patients. This means that only 1/3 to 1/4 of untreated SAD patients and a much lower proportion of treated SAD patients were correctly identified by the cross-sectional approach used here.
- 4. The authors have investigated a possible link between sunlight exposure on the day of the interview and depressive symptomatology. However, treatment studies strongly suggest that the effect of light on mood states is delayed (Winkler et al., 2005). For suicidal behavior light seems to have a bimodal effect with a facilitating effect of light on suicides for short periods of exposure and a decrease after longer periods (Vyssoki et al., 2014). Therefore, rather than using the sunshine exposure of only a single day, it would have been methodologically more sound to correlate sunlight exposure during the weeks before the assessment with subjects' symptomatology.
- 5. Traffanstedt et al. criticize the use of the Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal et al., 1987) as a diagnostic instrument for SAD. It is true that the SPAQ yields higher prevalence rates than seasonal depression according to the DSM-5 or the ICD-10. However, the authors have not recognized that a number of newer epidemiological studies (e.g. Levitt and Boyle, 2002; Thompson et al., 2004; Steinhausen et al., 2009), including our recent study on the epidemiology of SAD in Austria (Pjrek et al., 2016) have employed DSM-based diagnostic tests for SAD, such as the Seasonal Health Questionnaire (SHQ; Thompson and Cowan, 2001) and still found prevalence rates for SAD in the general population of around 2% (5% in a sample of primary care patients by Thomsson et al., 2004).
- 6. SAD is a cyclic disorder and symptoms, especially vegetative symptoms such as appetite and sleep, are increased during winter (hyperphagia and hypersomnia) while they are reduced during summer in a number of patients (Rosenthal et al., 1984). If, for example, a change in these symptoms occurs in one direction in winter and in the other in summer, this is both measured by the PHQ-8 and seasonal effects might level out. This thought again emphasizes that it is impossible to falsify the concept of SAD by the given cross-sectional methodology.
- 7. A further problem might be that the authors have included data from all over the United States of America. The authors might have simply missed a seasonal effect because southern regions of the USA were over-proportionately investigated. Concerning this matter, Rosen et al. (1990) suggested that summer problems are more common than winter ones in the southern United States. In addition, states demonstrating drastically different weather conditions, for example New Mexico and Oklahoma were grouped. Incorporation of latitude as a covariate into analyses would require a more differentiated approach that does not lump meteorologically unique regions into one group.

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8. One remarkable aspect of the paper of Traffanstedt et al. is the dismissal of dozens of studies from multiple research centers documenting the existence of thousands of patients who meet criteria for SAD. In contrast, their paper exhibits a supreme confidence in their own data and conclusions and an uncritical consideration of the possibility of error, especially when contradicting an established literature going back thirty years. In fact, the authors have not reviewed the many studies showing a relationship between mood and season nor did they review the studies that showed that light deprivation decreases mood while light treatment improves it. This makes their literature citation highly selective and inadequate.

To sum up, the greatest problem of the study of Traffanstedt et al. is their cross-sectional approach. Future studies working on the validity of the concept of SAD will have to take treatment variables into account for more accurate results. Selecting a sufficiently northernly sample is another important aspect. Furthermore, sunshine should not be quantified merely by the difference between sunset and sunrise as calculated by Traffanstedt et al., but as the mean daily number of sunshine hours or other more refined meteorological measures. Finally, timing diagnostic interviews to match the period of symptoms associated with light deprivation is crucial in SAD research.

### Statement of interest

Without any relevance to this work, Dr. Dorffner is shareholder and employee of the Siesta Group GmbH. Dr. Kasper received grants/research support, consulting fees and/or honoraria within the last three years from Angelini, AOP Orphan, AstraZeneca, Eli Lilly, Janssen, Krka, Lundbeck, Neuraxpharm, Pfizer, Pierre Fabre, Schwabe and Servier. Dr. Lanzenberger has received travel grants and/or speaker honoraria from AstraZeneca, Lundbeck, Dr. Willmar Schwabe GmbH, AOP Orphan, Janssen-Cilag and Roche. Dr. Pjrek has received travel grants from AOP Orphan. Dr. Spies has received travel grants from AOP Orphan and Eli Lilly and workshop compensation from Eli Lilly. Matthäus Willeit has served as consultant for GlaxoSmithKline, Lundbeck and Janssen-Cilag. Dr. Winkler has received travel grants and/or speaker honoraria from Angelini, AOP Orphan, Bristol-Myers Squibb, Lundbeck, Pfizer and Servier.

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