

Seasonal Mood Change and Neuroticism: The Same Construct?

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The personality trait of neuroticism has been found to be associated with a polymorphism in the regulatory region of the serotonin (5-HT) transporter gene (5-HTTLPR). This same genetic polymorphism has also been associated with seasonal changes in mood and behavior, or seasonality. The purpose of the current study was to determine whether seasonality and neuroticism are actually the same construct given that they are both associated with the same genetic polymorphism. We administered the Seasonal Pattern Assessment Questionnaire (SPAQ), which measures the severity of seasonality, and the Revised NEO

Personality Inventory (NEO-PI-R), which measures the severity of neuroticism, to 45 subjects diagnosed with seasonal affective disorder (SAD). SAD is a clinical expression of seasonality in which patients develop a major depressive disorder in the winter that remits in the summer and can be treated with light therapy. No significant correlation was found between neuroticism and seasonality. We conclude that seasonality and neuroticism are not the same construct, even though the 5-HTTLPR polymorphism is a genetic risk factor for each.

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THE TENDENCY to experience seasonal changes in mood and behavior, known as seasonality, is manifested to different degrees in a large segment of the population. For example, Kasper et al.¹ reported that 27% of the respondents in a survey of 416 people experienced some problematic changes during the winter in their mood, energy, sleep, appetite, weight, or social activity. At the extreme end of the spectrum of these seasonal changes is the clinical diagnosis of seasonal affective disorder (SAD), which has been estimated to affect 1.4% to 9.7% of the United States population depending on latitude.² SAD is characterized by depression, low energy, increased carbohydrate craving, weight gain, and increased sleep occurring annually in winter and remitting in summer.³

Although the effective treatment of SAD with light therapy has been well documented,⁴ the etiology of the disease is not as clear. Seasonal fluctuations in brain serotonin (5-HT) have been hypothesized to be involved in SAD.⁵ This hypothesis has led researchers to investigate genes involved in serotonergic functioning. Recently, Rosenthal et al.⁶ found an association between a polymorphism in the regulatory region of the 5-HT transporter gene (5-HTTLPR) and seasonality: the short (*s*) allele, as opposed to the long (*l*) allele, of the 5-HTTLPR gene is more prevalent in people with SAD. This same genetic polymorphism has been connected to the personality trait of neuroticism.⁷ Given this dual connection of seasonality and neuroticism with the 5-HTTLPR polymorphism, the question arises as to whether seasonality and neuroticism might be the same construct.

In a 1995 twin study ($N = 258$), Murray et al.⁸ found a correlation ($r = .38$) between seasonality

as measured by the Seasonal Pattern Assessment Questionnaire (SPAQ)⁹ and neuroticism as measured by the Eysenck Personality Questionnaire.¹⁰ Similarly, Jang et al.¹¹ found a correlation ($r = .35$) between seasonality and neuroticism in a normal population sample of twins ($N = 297$) when using the short version of the NEO Personality Inventory (NEO-PI-R)¹² and the SPAQ.⁹ In a follow-up study, Jang et al.¹³ statistically estimated the genetic and environmental relationships between seasonality and neuroticism in normal monozygotic and dizygotic twin pairs. They found a significant genetic correlation of .52 and an environmental correlation of .02 between neuroticism and seasonality. Notwithstanding the results of these two studies, we hypothesize that seasonality and neuroticism are not the same construct and will not be significantly correlated in a nondepressed, nontwin, highly seasonal population. This hypothesis is based on the clinical observation that patients with SAD are not apparently neurotic when not depressed.

Seasonality is characterized by observable changes in mood and behavior that occur with the seasons. Neuroticism, on the other hand, is a general tendency to experience negative affect and is manifest throughout the year, unrelated to environmental change. In a highly seasonal group, the differences between these two constructs should be extreme enough for observation.

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METHOD

Subjects

Forty-five of 101 potential subjects completed the study. All subjects provided informed consent. Their mean age was 44 ± 10 years (Table 1). All subjects were diagnosed with SAD within the last 6 years and participated in previous studies of SAD. They were Caucasian with the exception of one Hispanic and one Asian subject. There were no significant differences in age, race, or gender between 45 subjects who completed the study and 56 nonparticipants, of whom 38 were lost to follow-up study, 13 declined to be studied, and five had unusable data. Genetic information about the 5-HTTLPR polymorphism was available for 29 of the subjects. The mean age of patients for whom genetic information was available was 45 ± 10 years.

Measures

The NEO-PI-R¹² is a measure of normal personality traits consisting of 240 self-report statements that subjects respond to using a five-point Likert format, from "strongly agree" to "strongly disagree." It was designed according to the five-factor model of personality and assesses the five personality traits of neuroticism, extroversion, openness, agreeableness, and conscientiousness (N, E, O, A, and C). Each domain consists of six correlated facets. Neuroticism, the domain of greatest interest for this study, is the general tendency to experience negative affects such as fear, sadness, embarrassment, anger, guilt, and disgust. It is a measure of normal personality. Thus, while men and women high in neuroticism are prone to irrational ideas, and to difficulty coping with stress, it is possible to have a high score for neuroticism without having any diagnosable psychopathology. Scores on the NEO-PI-R were normalized using *T* scores (mean \pm SD, 50 ± 10) to permit comparison of our sample to the general population that the NEO-PI-R was tested on. *T* scores were derived from the standard *z*-score statistic and personal communication with Dr. Costa's research group.

The SPAQ⁹ was used to assess seasonality. It is an 18-part self-administered questionnaire that measures the extent to which an individual's mood and behavior change across the seasons. Seasonality was defined by the Global Seasonality Score (GSS), a scale derived from the SPAQ. The GSS segment of the SPAQ is a series of six questions about the severity of change between seasons in the subject's sleep, socializing, mood, weight, energy, and appetite. Responses range from "no change" to "extremely marked change" and are scored as 0 to 4, for a total GSS range of 0 to 24. GSS scores were normalized using the standard *z*-score statistic.

The subjects' mood was assessed using the Structured Interview Guide for the Hamilton Depression Rating Scale, Seasonal

Affective Disorder Version (SIGH-SAD).¹⁴ Although, by definition, patients with SAD historically remit during the summer, it is nevertheless our experience that depressive symptoms sometimes persist. The SIGH-SAD was used to quantify any such potential depressive symptoms. Due to the evidence that depression can influence neuroticism scores,¹⁵ it was important to have a measure of mood to determine whether the mood state was affecting personality trait measurements.

Procedure

Patients were contacted by telephone during the summer and were asked if they would be willing to participate in a study of personality and SAD. Those who agreed to participate were mailed a packet containing an introductory letter with instructions, the NEO-PI-R, the SPAQ, and consent forms. Subjects were instructed to complete the questionnaires without consulting others and to return them in the stamped envelope provided. Within 2 weeks of receiving the completed questionnaires, clinicians contacted the subjects by telephone for a mood rating using the SIGH-SAD.

Analysis

A correlation was performed between normalized neuroticism scores and normalized GSS scores. To compare our SAD patients with the general population, we performed one-sample *t* tests on mean neuroticism and seasonality scores. For neuroticism, this comparison was based on the normal data presented by Costa and McCrae in the NEO-PI-R Manual.¹² For seasonality, we compared our group with a 1989 epidemiological study group of seasonality and SAD that was drawn from the same metropolitan Washington, DC, locale.¹ The variance of our sample was consistent with the variance of the epidemiological study, permitting the use of a one-sample *t* test. We performed a correlation between SIGH-SAD scores and neuroticism scores to ensure that mood state did not relate to personality trait. Finally, we performed a *t* test on the neuroticism scores of *ll* versus non-*ll* genotypes, followed by a power analysis to assist with interpretation of the *t* test results.

RESULTS

There was no correlation between neuroticism and the GSS ($r = .057$, nonsignificant). The mean neuroticism score was higher in the SAD group ($T = 56.1 \pm 10$) relative to the normal population ($T = 50.0 \pm 10$, $t = 3.2$, $P < .005$). However, it was still within 1 SD of the norm.

As expected, our sample was significantly more seasonal than the normal population. The mean GSS was 16.1 ± 3.3 versus 5.4 ± 3.9 , respectively ($t = 21.5$, $P < .001$). Thus, our sample was approximately 3 SD greater than the norm for seasonality.

Although 19 of the subjects had slightly elevated SIGH-SAD scores (>7 but <25), there was no correlation between SIGH-SAD scores and neuroticism scores ($r = .05$).

There was no significant difference in neuroticism scores between the *ll* group and the non-*ll*

Table 1. Subject Characteristics

Characteristic	Value
Age (yr)*	44 ± 10
Age of onset (yr)*	23 ± 11
Gender ratio (female:male)	2:1 (31:14)
Psychiatric diagnosis (DSM-III-R criteria)	
Bipolar II	10
Unipolar	35

*Mean \pm SD.

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