Farmer et al (2002) examined whether self-reports of neuroticism and extraversion represent enduring vulnerabilities to depression. While their findings make a valuable contribution, their conclusions regarding neuroticism warrant additional consideration. They found variables reflecting past and current depression were significant predictors of neuroticism and interpreted this as inconsistent with their hypothesis that neuroticism would ‘exhibit trait-like qualities’ and would not be ‘substantially influenced by alteration in mood-state’ (p. 118). Farmer et al’s findings did not directly address this hypothesis because they did not use a longitudinal design needed to observe fluctuations in mood-state. None the less, several longitudinal studies indicate that neuroticism is affective-state dependent. For example, Hirschfeld et al. (1983) found that patients in remission from depression reported lower levels of neuroticism than they originally reported when depressed. Findings of this nature have been used to argue that neuroticism and related traits are not contaminants of depression. More recently, investigators (e.g. Santor et al., 1997) studying samples of patients have noted that absolute changes in depression-related traits are associated with changes in mood (i.e. affective-state dependent), but that there is also a consistency in the rank of patients with regard to their scores on these measures (i.e. relative stability). Findings such as these have been interpreted as indicating that depression-related personality traits have both state-like and trait-like properties.

Given the large association between neuroticism and depression, Farmer et al suggested that neuroticism may be largely ‘a proxy measure for present or past depression’ (p. 121) and questioned whether neuroticism reflects a vulnerability for depression. Neuroticism refers to a tendency to experience negative affect, so this high degree of overlap is not surprising. More importantly, longitudinal studies (Hirschfeld et al., 1989; Krueger et al., 1996) have found that high premorbid neuroticism is positively associated with the development of depression.

In summary, Farmer et al’s conclusion that neuroticism does not measure a vulnerability to depression and primarily reflects symptoms of depression is not warranted. Self-reports of neuroticism prospectively predict depression. Longitudinal studies support Farmer et al’s conclusion that neuroticism is strongly associated with a person’s current affective state. However, such studies also suggest that neuroticism is likely to have trait-like properties in addition to the state-like properties noted by Farmer et al.


Authors’ reply: We are pleased to respond to the comments of Drs Hodgins & Ellenbogen and Dr McWilliams and are grateful for their interest in our work.

Hodgins and Ellenbogen suggest that an absence of a difference in mean scores for neuroticism (N) for never-depressed siblings of probands with depression and never-depressed siblings of healthy controls can be interpreted as showing that the siblings of probands with depression have not inherited the vulnerability for the disorders. However, this is missing the point. We hypothesised that scores represent a genetically influenced trait that underpins the risk of developing depression in the presence of precipitating factors such as adverse life events. If N were such a trait, then it would be expected that all first-degree relatives of probands with depression who share an average 50% of their genes with their relative with depression, would have higher mean scores than subjects without such a genetic relationship to a proband with depression, irrespective of affective status. Our failure to detect a difference for N scores is not due to lack of power since we have shown differences between the relatives of probands with depression and controls for other personality measures (Farmer et al., 2003) such as the Harm Avoidance Scale of the Temperament and Character Inventory (Cloninger et al., 1993).

Both Hodgins & Ellenbogen and McWilliams rightly point out that longitudinal studies may help to disentangle the relationship between N and depression. However, even longitudinal studies can fail to answer the issue of what came first, the chicken of neuroticism or the egg of depression. For example, it is now well recognised that depressive symptoms occur in children and adolescents as well as in adults. Consequently, in order to demonstrate that N scores represent an underlying vulnerability to depression and are not merely a proxy measure of depressive symptoms, it is necessary to show that elevated N scores occur in the absence of significant depressive symptoms at the first point of measurement. To our knowledge, no longitudinal study to date has shown this.

Our study is in keeping with a growing literature showing that N has considerable state-dependent as well as trait-like properties. Despite this, there remains a cherished belief that the measure does indeed represent a trait underlying the vulnerability to depression. We have not demonstrated such properties for the scale in our Cardiff study.

Declaration of interest

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