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Comments and Controversies

Author's response to commentary 'Depressive symptomatology should be systematically controlled for in neuroticism research'

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ABSTRACT

In the commentary by Bianchi and Laurent (2015), the authors suggest that depressive symptoms should be controlled for when examining the neurobiology associated with trait neuroticism. We fully agree that the relation between neuroticism and symptoms of stress-related psychiatric disorders, such as major depressive disorder and anxiety disorders, should not be overlooked when studying its neural correlates. However, instead of treating this relation as a potential confound, we consider it to be of particular importance to include depressive symptoms when studying the influence of acute psychological stress on neural mechanisms related to trait neuroticism. Regardless of this principal disagreement, we also confirmed empirically that depression scores did not affect our voxel-wise results. In sum, our results were not confounded by depression scores and more importantly, our study question and design do not warrant including depression scores in our analysis.

We thank Bianchi and Laurent (2015) for their attentive remarks that we have read with great interest. In their commentary, the authors suggest that depressive symptoms should be controlled for when examining the neurobiology associated with trait neuroticism. The topic of the discussion that has been raised is important and we fully agree that the relation between neuroticism and symptoms of stress-related psychiatric disorders, such as major depressive disorder and anxiety disorders, should not be overlooked when studying its neural correlates. However, instead of treating this relation as a potential confound, we consider it to be of particular importance to include depressive symptoms when studying the influence of acute psychological stress on neural mechanisms related to trait neuroticism.

Neuroticism is a major risk factor for both mental and somatic disorders, although the risk for stress-related psychiatric disorders is particularly strong, with a prevalence of more than 40% of mood and anxiety disorders in the 5% of the population with the highest neuroticism scores (Cuijpers et al., 2010; Kotov et al., 2010). The aim of our study (Everaerd et al., 2015) was to examine the neurobiology of trait neuroticism as a risk factor for stress-related psychiatric disorders, such as depression. To exclude possible confounds of past or current disease, we performed our study in a young, healthy population, free of any somatic or psychiatric history (such as depression) or current disease. Beck Depression Inventory scores (BDI (Beck et al., 1961)) within this population were thus significantly left skewed (Kolmogorov–Smirnov test for normality: $p < 0.001$, as shown in Fig. 1a). Neuroticism as a trait, however, was normally distributed ($p > 0.2$, Fig. 1b) and therefore provided a much more sensitive measure of vulnerability for stress-related disorders than the BDI scores.

The literature that the authors refer to (Lahey, 2009) indeed suggests that depressive symptoms be controlled for in cross sectional studies investigating trait neuroticism, but in the context of adverse public health outcomes including physical health problems. This is a different research question, for which depressive symptoms could indeed confound the investigated relation between neuroticism and somatic symptoms or mental disorders other than depression. In contrast, with our stress induction paradigm, we were especially interested in the risk that neuroticism holds for stress-related psychiatric disorders, such as depression. Importantly, the occurrence of depressive symptoms is part of the neuroticism subscale

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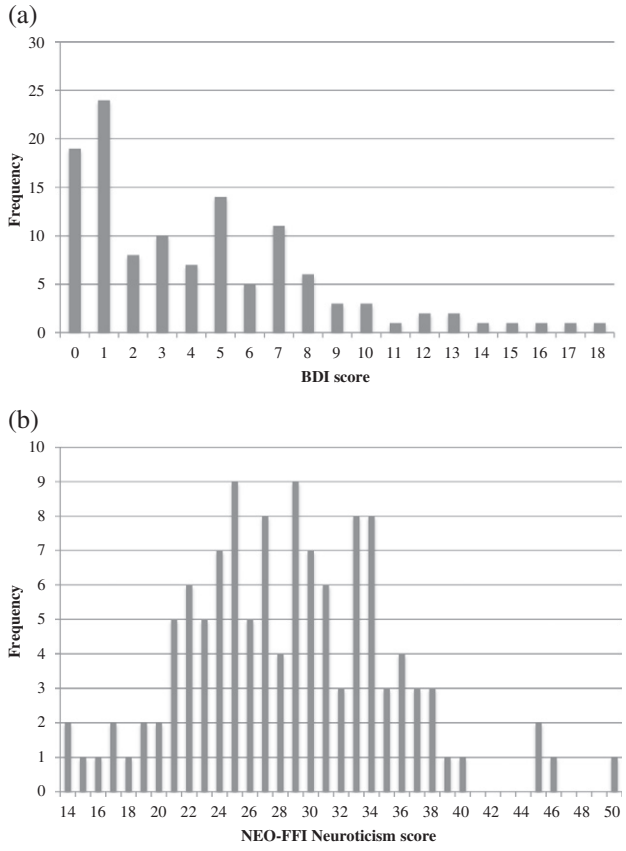

Fig. 1. Distributions of BDI scores (a) and NEO-FFI Neuroticism scores (b) in the study population. The single subject with a BDI score of 18 and a NEO-FFI neuroticism score of 50 was excluded from all analyses, as previously reported in (Everaerd et al., 2015).

questionnaire of the NEO-FFI questionnaire (McCrae and Costa, 1999) and thus constitutes an important part of this personality trait that should not be ignored.

Regardless of this principal disagreement, we confirmed empirically that depression scores did not affect our voxel-wise results. When controlling for BDI scores, we found the same interaction between condition and facial expression in the right amygdala (peak voxel 30 2 −22, T-value = 4.12, $p_{SVC} < 0.01$). In addition, we would like to mention that depressive symptoms were not included as a covariate in our analyses of blood pressure in our manuscript, as mentioned in the commentary. In sum, our results were not confounded by depression scores and more importantly, our study question and design do not warrant including depression scores in our analysis.

Therefore, while we agree that the issue deserves consideration, we respectfully disagree with the authors' suggestion to control for depressive symptoms when examining the neural correlates of neuroticism as a risk factor for stress-related psychiatric disorders. The construct of trait neuroticism is only relevant when it conveys a risk for malfunctioning or disease, and it is exactly this risk that we investigated with our study.

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