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Comments and Controversies

Depressive symptomatology should be systematically controlled for in neuroticism research

Renzo Bianchi[a,*], Eric Laurent[b]

[a] Faculty of Science, University of Neuchâtel, Emile-Argand 11, 2000 Neuchâtel, Switzerland
[b] Laboratoire de Psychologie (EA 3188), University of Franche-Comté, 30-32 rue Mégevand, 25030 Besançon Cedex, France

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In a recent fMRI study, Everaerd et al. (2015) examined the relationship between (experimentally induced) stress, (personality trait) neuroticism, and amygdala responsivity to emotional information. The authors found that neuroticism selectively enhanced amygdalian response to potential threat in the stress induction condition. While the sophistication and ambition of Everaerd et al.'s (2015) study should be underlined, we are concerned with the fact that the authors did not statistically control for depressive symptoms when analyzing the effects of neuroticism.

The authors assessed depressive symptoms using the Beck Depression Inventory and found a strong correlation between depressive symptoms and neuroticism ($\rho = 0.729$, $p < 0.001$). Such an association suggests that depressive symptoms constitute a key potential confounding variable in the relationship between neuroticism and the other variables under study. However, the authors did not introduce depressive symptoms as a covariate in their analyses of neuroticism—while they did so with blood pressure, a variable whose correlation with neuroticism was much weaker ($\rho = 0.248$, $p < 0.01$). This state of affairs is problematic in that it prevents clear conclusions on the role of neuroticism from being drawn. Put differently, the effects imputed to neuroticism in this study may actually be due to depressive symptomatology, acting as a third variable.

The strength of the link between neuroticism and depressive symptomatology has often been underlined (e.g., Farmer et al., 2002; Jylha and Isometsa, 2006). As a corollary, the issue of the statistical control of depression is crucial in neuroticism research (Lahey, 2009). Moreover, it is worth noting that amygdala response to emotional information has been shown to be altered, not only in clinical depression but also in subclinical depression (Abler et al., 2010; Laeger et al., 2012). Structural alterations in fronto-limbic regions (including the amygdala) have been reported in subthreshold depression (Li et al., 2015), and abnormalities in emotional information processing have been associated with altered connectivity between the medial prefrontal cortex, the amygdala, and the ventral striatum in dysphoria (Sabatinelli et al., 2015). In this context, not taking into account depressive symptoms when examining the neurobiology of neuroticism constitutes, in our view, a serious limitation.

To conclude, we would like to emphasize that future research should be careful not only about the statistical significance of neuroticism-related effects but also about the size of these effects once depressive symptoms are controlled for. Indeed, an effect can be statistically significant without having an important explanatory role or being clinically or practically relevant (Cumming, 2014; Nuzzo, 2014; Simmons et al., 2011). All in all, the relationship between stress, neuroticism, and amygdala responsivity to emotional information needs to be re-examined before firm conclusions can be drawn about it.

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* Corresponding author. Tel.: + 41 32 718 1390; fax: + 41 32 718 1391.
E-mail addresses: renzo.bianchi@unine.ch (R. Bianchi), eric.laurent@univ-fcomte.fr (E. Laurent).

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