Commentary

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Motivational Significance of Control Failures as a Window on Risk for Problematic Alcohol Involvement

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In a recent study published in *Biological Psychiatry: Global Open Science*, Boer *et al.* (1) present data from the largest study to date examining associations between electrophysiological responses to performance errors and risk for alcohol use disorder (AUD) in young people. They reported that smaller error-related negativity (ERN) amplitude was associated with several AUD risk behaviors, including earlier initiation of alcohol use and higher frequency of binge drinking. Unexpectedly, larger error positivity (Pe) values were also associated with higher binge drinking frequency. The authors interpreted their findings as consistent with the notion that altered cognitive control increases risk for early and potentially problematic alcohol use in adolescence.

The study of Boer *et al.* (1) represents an important contribution to the literature on neurocognitive AUD risk factors in adolescents and emerging adults. The large birth cohort sample allowed the researchers to account for numerous person-level confounders (e.g., IQ, prenatal alcohol/tobacco exposure) that otherwise could offer alternative explanations for associations that they observed. The study also adds large-scale validation of previously identified links between blunted ERN amplitude and alcohol/drug involvement. However, several conceptual and methodological issues in the work of Boer *et al.* (1) warrant some caution when considering their findings.

How Should Individual Differences in Error Reactivity Be Interpreted?

Boer *et al.* (1) framed their findings in terms of aberrant cognitive control as an AUD risk factor. This framing makes sense in the context of the large literature linking control deficits with risky alcohol involvement, as well as the literature associating the ERN with cognitive control. However, there are reasons to question whether a cognitive control framework provides the best understanding of the findings of Boer *et al.* (1). First, as others have highlighted (2), there is little evidence linking ERN amplitude (or Pe or frontal midline theta power) with between-person differences in cognitive control. Rather, the ERN often is larger within persons in situations requiring greater control compared to situations in which control is de-emphasized, and it should not be assumed that phenomena varying within individuals translate directly to between-individual differences.

Second, there is a logical inconsistency in the idea that neurophysiological activity elicited by failures of control (i.e., errors) is somehow indicative of control itself. To the extent that they relate to control at all, it seems more likely that ERN, Pe, and frontal midline theta power reflect functions adjacent and potentially antecedent to control adjustments. Considerable evidence links ERN amplitude with a cascade of affective/motivational processes that covary with the importance or significance of the error (3). Given the known neural source of the ERN in the medial frontal cortex (especially dorsal anterior cingulate cortex), this signal likely reflects engagement of the salience network that functions to switch between default mode processing and the focused attention and motor control instigated by engagement of the fronto-parietal executive control network (4). Pe amplitude seems to index a distinct function that tracks the degree of postdecision evidence accumulation concerning the correctness of the decision/response, with larger amplitude indicating stronger evidence for an error (5). Together, the ERN and Pe reflect processes integral to performance monitoring that, while clearly important for initiating control adjustments, should not be considered core elements of control.

A second reason why the findings of Boer *et al.* (1) may not best be interpreted through the lens of cognitive control is that their report presented no behavioral data pertinent to control. Fundamentally, cognitive control is about controlling behavior, regulating attention and action to ensure the consistency of behavior with one's goals (6). As a trait-level construct, cognitive control (i.e., executive function) encapsulates a set of related yet distinct higher-order cognitive operations the purpose of which is to orchestrate the actions of lower-level cognitive and motor operations, whose purpose in turn is the regulation of behavior. The electrophysiological response data that Boer *et al.* (1) presented were acquired as participants completed a task, the go/no-go, for which cognitive control is required. In such tasks, engagement of control typically is operationalized in terms of adjustments to behavior necessitated by task demands or by failures of control. Boer *et al.* (1) did not include any such measures or any measures of task performance in their report, leaving open questions concerning a potential role for control in understanding alcohol involvement in their sample.

If not aberrant control, then what might explain the associations that Boer *et al.* (1) observed between blunted ERN, exaggerated Pe, and AUD risk behaviors? I contend that their findings likely reflect the involvement of underlying trait dimensions rooted in affective-motivational dysregulation that also generally covary with substance involvement. In particular, the internalizing-externalizing spectrum has been associated with both a blunted ERN and early and risky alcohol

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and drug involvement (7). Although internalizing symptoms (i.e., anxiety) have been linked to an exaggerated ERN (7), that association seems to be limited to certain facets of internalizing (e.g., shyness), while other facets (e.g., fear) may be related to a blunted ERN (8). Alternatively, it could be that the reported link between smaller ERN and AUD risk behaviors, especially earlier onset of drinking, reflects an effect of chronic drinking across adolescence. The cross-sectional nature of the data of Boer *et al.* (1) cloud interpretation of the causal direction of the effect, and at least some prior work suggests that the association between blunted ERN and risky drinking reflects current alcohol use more than a liability for AUD (9).

Far less is known about individual-difference factors that can modulate the Pe, but available evidence also points to motivational processes. The current understanding of Pe as indexing postdecision accumulation of evidence concerning the appropriateness of the response suggests that a blunted Pe could reflect a facet of the externalizing spectrum related to disregard of consequences, whereas an enlarged Pe may be associated with concern or worry over substandard performance, a hallmark of some forms of anxiety. In the context of findings of Boer *et al.* (1) and given the functional dissociability of the ERN and Pe, it could be that a blunted ERN reflects AUD risk associated with some facet(s) of the externalizing spectrum, whereas an exaggerated Pe reflects risk associated with anxious worry or concern over evaluation, both of which relate to increased substance use in adolescents.

Methodological Considerations

Leaving aside issues of interpretation of the ERN/Pe and their associations with alcohol involvement, the study of Boer *et al.* (1) suffered from some methodological limitations that also warrant consideration. First and foremost is the fact that the analyses of Boer *et al.* (1) were based on participants with as few as 5 analyzable error trials (the sample average was 9–10 error trials). This matters because the internal consistency of any measure directly determines its suitability as an index of individual differences, and internal consistency generally increases with increasing numbers of items (i.e., trials). Boer *et al.* (1) argued—understandably—that the lower-bound threshold for numbers of trials per participant in any electroencephalography (EEG) study must be balanced against the desire to retain as much data as possible. Even so, setting the threshold at 5 error trials is problematic given that the ERN's reliability is known to be poor ($\alpha \approx 0.40$) when derived from so few trials (10). Indeed, a preprint authored by Boer and colleagues (cited in their article) describing the psychometric properties of the event-related potentials in this sample reports that reliability for ERN amplitude was low (intraclass correlation coefficient [ICC] = 0.41; $\alpha = 0.45$) when only 5 errors were included and became only moderate (ICC = 0.60; $\alpha = 0.62$) when 10 errors were included. Based on these findings, the authors suggested a minimum of 9 to 10 errors for ERN analyses examining brain-behavior relationship in large-scale EEG studies. This is sound advice, and it would be interesting to know whether the findings of Boer *et al.* (1) would change if they applied this threshold for inclusion in their analyses.

A second and related methodological concern is that the go/no-go task that Boer *et al.* (1) used comprised just 160 trials, of which only 32 were no-go trials. Use of truncated tasks is often necessary in large-scale studies due to the sheer number of assessments administered. Nevertheless, it still holds that a measure of interindividual differences is only as good as the task (or questionnaire, or interview, etc.) used to assess it, and a task in which only 32 errors of commission are possible—and that delivered an average of 9 to 10 errors—simply is not very well suited to indexing individual differences in error-related neurophysiological responses.

In summary, although large birth cohort studies such as the one reported by Boer *et al.* (1) offer rare opportunities for discovery that simply cannot be realized in typical, smaller-scale studies, consideration of basic conceptual and methodological issues is no less important in large-scale studies than it is in smaller ones. Regardless of study size, future work should aim to go beyond individual measures derived from single tasks to characterize associations between atypical action monitoring and alcohol involvement. Understanding such associations is more likely to be advanced using approaches that embed error reactivity and other performance-monitoring measures into multidomain constructs representing broad domains of functioning.

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Article Information

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