Commentary

Biological Psychiatry: CNNI

Peak Alpha Frequency, Visual Perception, and Cognition in Schizophrenia

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One of the first features observed in the earliest electroencephalograms was a prominent periodic waveform that cycled roughly 10 times per second. Hans Berger named this waveform the alpha wave, presumably because it was the most noticeable feature of the electroencephalogram. In the years after this discovery, the biological provenance and functional significance of the alpha wave has been the topic of much study and controversy.

One area of particular interest concerns the substantial individual differences in the speed of alpha. That is, although the alpha wave tends to cycle at around 10 Hz on average, there is tremendous subject-to-subject variability in this cycling speed. Converging evidence suggests that variability in the speed of alpha cycling (i.e., peak alpha frequency [PAF]) reflects a mixture of trait and state factors (1). Evidence for a trait component includes the high test-retest reliability of PAF (2). Furthermore, PAF is highly heritable and strongly correlated between monozygotic twins (3). However, state-dependent modulations of PAF are also well documented. For example, PAF has been shown to change as a function of age, wakefulness, exercise, cognitive demand, medication, and pain stimulation (1). Thus, a given individual's PAF likely reflects a combination of both trait- and state-dependent influences.

One promising theory of the functional significance of PAF suggests that the rhythmic fluctuations of alpha waves, which tend to be most prominent over the occipital cortex, are linked to rhythmic fluctuations in visual perception. Evidence for this view is largely drawn from studies in which participants are asked to report the number of stimuli (e.g., flashes of light, letters, figures) perceived within a short period of time. When the period of time between successive stimuli is $<$100 ms, the 2 stimuli often fuse into a single percept and the subject will report observing only 1 stimulus. As early as 1954, researchers began to speculate that this 10-Hz limit may reflect a fundamental "psychological unit of duration" that roughly coincides with the speed of alpha wave cycling (4). Indeed, work within the past decade has provided tantalizing evidence that individual differences in the speed of alpha cycling are associated with the speed of temporal sampling of visual information (2,5).

In addition to associations with visual processing, faster alpha cycling has been linked to cognitive functioning. This relationship can be pithily summarized by the maxim "smarter brains run faster" (6). While some researchers have indeed observed associations between PAF and a general cognitive performance factor (7), others have found that PAF is related to specific cognitive domains. For example, Posthuma *et al.* (6) reported associations only with working memory in older adults, while Ociepka *et al.* (8) reported associations only with processing speed. Still others have failed to observe any strong correlations between PAF and cognitive performance (9). Therefore, the degree to which smarter brains run faster remains an open question.

Perhaps most relevant to the audience of the *Journal*, atypical PAF has been linked to a variety of mental health disorders. Such links, in conjunction with the relative stability of PAF over time, have led to conjecture that PAF may be clinically useful as a biomarker for psychiatric disorders. However, PAF's utility as a biomarker is limited by a lack of diagnostic specificity, as demonstrated by the lengthy list of disorders linked to altered PAF (e.g., schizophrenia, bipolar disorder, major depressive disorder, posttraumatic stress disorder, and autism). Furthermore, group differences in PAF may be confounded by group differences in other domains such as cognition and perception. Indeed, it is possible that altered PAF is a neural index of cognitive and/or perceptual differences rather than a biomarker of a mental health disorder. Given these limitations, there is a need for work that 1) examines the diagnostic specificity of altered PAF and 2) clarifies the degree to which altered PAF is associated with cognitive and perceptual differences within psychiatric conditions.

In the current issue of *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, Catalano *et al.* (10) examined relationships between PAF, cognition, and visual processing in 90 individuals with schizophrenia, 62 individuals with bipolar disorder, and 69 healthy control participants. The authors replicated previous findings of reduced PAF in schizophrenia compared with healthy control participants. Interestingly, PAF was not reduced in the group with bipolar disorder relative to healthy control participants—in fact, the average PAF of the group with bipolar disorder was slightly higher than the average PAF of the healthy control participants. This suggests that PAF may demonstrate some diagnostic specificity as a biomarker of schizophrenia.

Catalano *et al.* (10) also observed associations between PAF and visual backwards masking performance in both the schizophrenia and healthy control groups. In the backwards masking task, subjects were presented with a target stimulus followed by a masking stimulus of overlapping black and white curved lines. As the period of time between the target and masking stimulus decreased, perception of the target became more difficult. The authors found that subjects with lower PAF required longer periods of time between target and mask to successfully perceive the target. Such a finding provides further evidence that PAF may reflect the speed of temporal sampling of visual information. Surprisingly, the group with schizophrenia did not significantly differ from the control group in terms of backwards masking performance. However, the trend was in the expected direction: on average, individuals with schizophrenia required more time between the target and

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mask for successful target detection relative to healthy control participants.

Catalano *et al.* (10) also observed that PAF correlated with general cognitive performance in the schizophrenia group. Associations for bipolar disorder and healthy control groups were trending in the expected direction but did not survive correction for multiple comparisons. One reason for this lack of significance may be that the correlations were attenuated by range restriction. Unfortunately, the authors did not report correlations across groups to test this hypothesis. Nevertheless, these findings were generally consistent with the idea that higher PAF was related to improved cognitive functioning.

Characterizing the causal nature of the associations reported by Catalano *et al.* (10) will be a crucial next step of this line of research. Bivariate correlations, except in some rare cases, are unable to clarify causality. Indeed, Figure 1 shows 4 possible causal chains that are all generally consistent with the pattern of results reported by Catalano *et al.* Such causal chains are useful for generating falsifiable hypotheses to be tested by future studies. For example, the causal chain shown in Figure 1A supposes that reductions in PAF cause downstream disruptions in visual sampling and cognition that in turn lead to the symptoms of schizophrenia. One way to test this hypothesis would be to directly manipulate PAF (perhaps via pharmacological, behavioral, or neuromodulatory interventions) and assess downstream changes in visual information processing, cognition, and psychotic symptoms. In contrast, the causal chain in Figure 1B supposes that the symptoms of schizophrenia cause disruptions in visual and cognitive processes that in turn lead to reduced PAF. This causal hypothesis can be tested by examining whether intervening on symptoms leads to changes in visual information processing and cognition, which are then reflected by changes in PAF. Other possibilities are shown in Figure 1C and D. Importantly, the causal chains shown in Figure 1 represent only a subset of the possible chains that might give rise to the observed pattern of results and do not account for unmeasured confounding variables. However, through careful and systematic study, it is possible to iteratively rule out causal chains that are inconsistent with empirical evidence and converge on the chain that is most likely to give rise to the observed evidence.

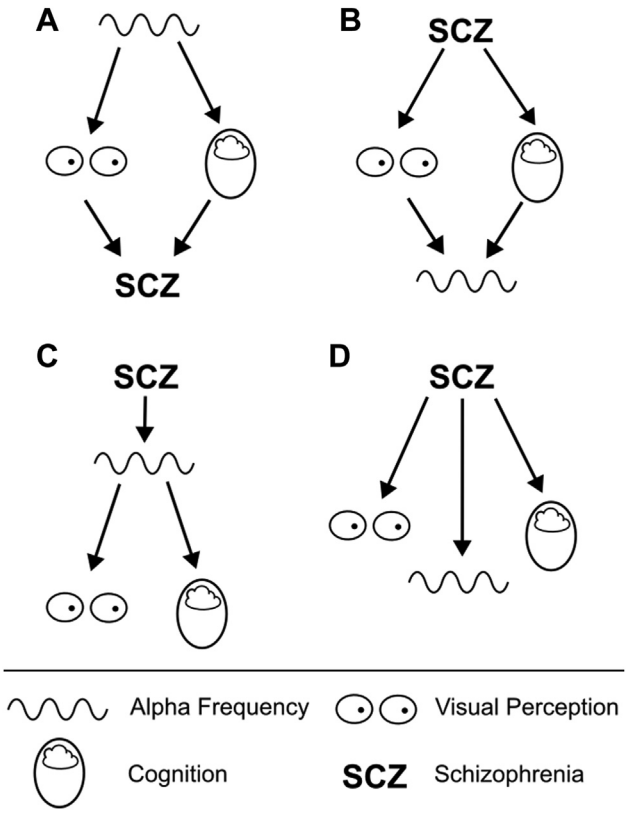

Figure 1. Examples of possible causal chains between peak alpha frequency, cognition, visual perception, and schizophrenia. The chains represent possible scenarios that might give rise to the results observed by Catalano *et al.* (10). These are only a subset of possible causal chains. The influence of unmeasured confounding variables is not depicted.

This work by Catalano *et al.* (10) represents an important step toward understanding the relationships between PAF, cognition, perception, and schizophrenia. Future studies may seek to elucidate the causality of such relationships. Doing so will inform the degree to which PAF is clinically useful for understanding, diagnosing, and treating schizophrenia.

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Article Information

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