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We were pleased to read the constructive commentary (Micoulaud-Franchi & Fovet, 2018) on our original piece (Thibault & Raz, 2017). In this response, we build on the theoretical framework for studying neurofeedback that the commentators sketch out while pointing out potential caveats to adopting a neuroreductionist approach.

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Micoulaud-Franchi and Fovet (2018) suggested that researchers should interpret the effects of neurofeedback through three distinct mechanisms: (a) psychosocial—including the elements involved in the motivation for and expectation associated with participating in a clinical procedure, interacting with a practitioner, and interfacing with neurotechnology; (b) cognitive—including the process of actively engaging in a form of mental or behavioral training, regardless of the type or contingency of the feedback provided; and (c) neurophysiological—including the effects of regulating a specific brain signal. In our previous publications, we largely conflated psychosocial and cognitive descriptors into the terms placebo and nonspecific effects, interchangeably.

To increase the usefulness of this proposed framework, we recommend that researchers further discuss the effects of electroencephalography neurofeedback (EEG-nf) in two distinct categories—(a) changes to the brain signal trained, including related neurophysiology, and (b) effects on behavior, mental state, or well-being—and test whether these variables correlate (see Figure 1). In the EEG-nf literature, however, researchers have often conflated these two outcome measures and assumed that one implies the other. In other words, they have speciously assumed that the “EEGCopia” that Micoulaud-Franchi and Fovet (2018) propose to develop, already exists.

Discussions of this type of EEGCopia hark back to the wishful idea that DNA sequences would eventually explain most medical conditions. Although scientists successfully reduced a few diseases to genes (e.g., sickle cell anemia and Huntington’s disease), the etiology of most medical conditions remains largely polygenetic, multifaceted, and diffi-

Figure 1. A framework for discussing neurofeedback. The red arrow (bottom arrow with the question mark) depicts the fundamental interaction on which the practice of electroencephalography neurofeedback (EEG-nf) rests, but which remains tenuous (Thibault & Raz, 2017). In terms of altering brain waves, EEG-nf seems to function through psychosocial and cognitive (e.g., Ninaus et al., 2013), as well as specific neurophysiological (e.g., Schabus et al., 2017), mechanisms. The dashed, bidirectional arrow indicates that brain and behavioral outcome measures sometimes, but not always, correlate. See the online article for the color version of this figure.
cult to explain in genetic terms alone, let alone by single genes (Ahn, Tewari, Poon, & Phillips, 2006). Similarly, brain imaging is unlikely to single-handedly identify the causal mechanisms responsible for mental disorders (Borsboom, Cramer, & Kalis, 2018). Examining brain activity alone and neglecting to consider nonbrain factors misses the critical insight that psychiatric conditions manifest through “significant distress or disability in social, occupational, or other important activities” (American Psychiatric Association, 2013, p. 20).

Genes play a role in arguably all medical conditions, just as brain activity plays a role in mental disorders. Neither of these statements, however, suggests that scientists best describe conditions in the “bottom-up” terms of genetics or neurobiology (Kirmayer & Gold, 2011). Because the neurofeedback literature suggests that psychosocial and cognitive mechanisms, rather than specific neurophysiological targets, seem to drive behavioral change (italicized in reference to the framework proposed earlier in the text and outlined in Figure 1), in our research we tend to discuss the mechanisms behind the behavioral benefits of neurofeedback as classifiable “top-down” psychological phenomena (e.g., motivation, expectation, implicit learning, effortful training, and time spent with practitioner). A mind–body dualist can speak of biology and psychology as independent processes; a cognitive neuroscientist cannot. Thus, we distinguish between bottom-up and top-down processes to discern quantifiable variables, facilitate discussion, and identify mechanisms of action in the hopes of fostering a better scientific understanding of neurofeedback and a more informed way of practicing it (Raz, 2011)—not to propose a dichotomy between the brain and psychological sciences.

One of us (Robert T. Thibault) recently met with Micoulaud-Franchi and Fovet and found a large overlap in terms of how we (Robert T. Thibault and Amir Raz) and they interpret the literature surrounding the application of EEG-nf as well as how researchers can best advance the field. Amid this consensus, we mainly diverge on one nonempirical issue: Whereas they maintain a steadfast opposition of the hyperbolic truth of neurofeedback: Comment on Thibault & Raz (2017). American Psychologist, 73, 933–935. 
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References


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