A Consensus Framework for Neurofeedback Research

(and the perils of unfounded neuroreductionism)

A reply to Micoulaud-Franchi and Fovet

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ABSTRACT

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.

MAIN TEXT

Micoulaud-Franchi and Fovet suggest that researchers should interpret the effects of neurofeedback through three distinct mechanisms: (1) *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; (2) *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 (3) *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 EEG-nf in two distinct categories and test whether these variables correlate: (i) changes to the brain signal trained, including related neurophysiology, and (ii) effects on behavior, mental state, or well-being (see Figure 1). In the EEG-nf literature, however, researchers often conflate these two outcome measures and assume that one implies the other. In other words, they speciously assume that the "EEGCopia" that Micoulaud-Franchi and Fovet propose to develop, already exists.

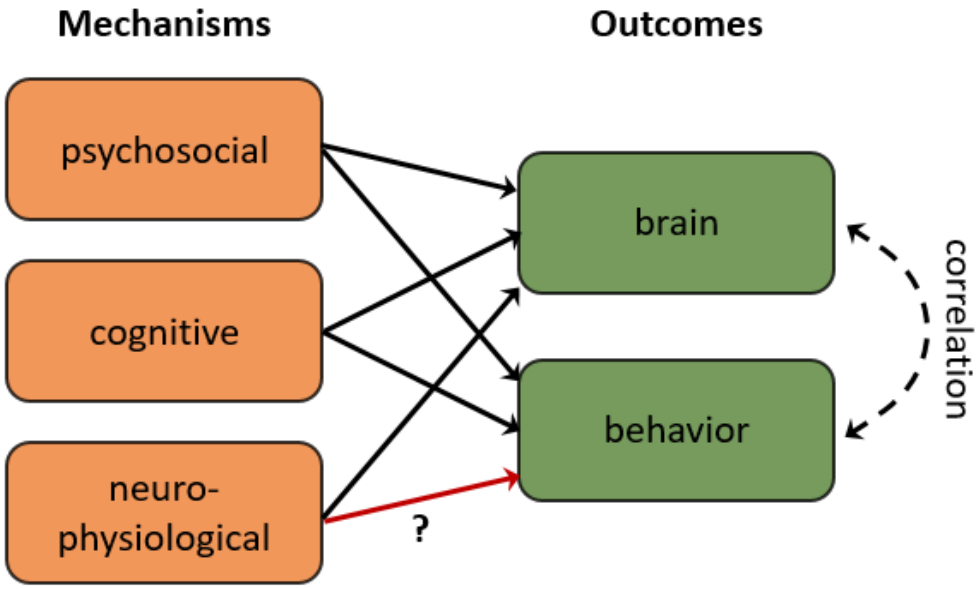

Figure 1. A framework for discussing neurofeedback. The red arrow depicts the fundamental interaction on which the practice of EEG-nf rests, but which remains tenuous (Thibault & Raz, 2017). In terms of altering brain waves, EEG-nf seems to function through psychosocial, cognitive (e.g., Ninaus et al., 2013), and specific neurophysiological mechanisms (e.g., Schabus et al., 2017).

Discussions of this type of EEGCopia harks 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 difficult 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 non-brain 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).

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 above), 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 (RTT) recently met with Micoulaud-Franchi and Fovet and found a large overlap in terms of how we (RTT and AR) and they interpret the literature surrounding the application of EEG-nf as well as how researchers can best advance the field. Amidst this consensus, we mainly diverge on one non-empirical issue: whereas they maintain a steadfast optimism that an EEGCopia will soon emerge, we remain skeptical that science will soon find causal and engineerable EEG biomarkers for most mental disorders. Whether resolutely hopeful or principally proceeding by inquiry, the EEG-nf community would do well to hope for the best and prepare for the worst.

Footnote

We shared a draft of this reply with Micoulaud-Franchi and Fovet. They confirmed that the opinions ascribed to them herein accurately depict their viewpoints.

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Micoulaud-Franchi & Fovet 2018 -- please add full reference once available.

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