Commentary

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Training alpha to treat pain: dissociable pathways to analgesia

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Can individuals learn to change their pain experience by modulating their own brain rhythms? In this issue of PAIN,[25] Zheng et al suggest that this is possible, particularly for the affective dimension of pain. As pharmacological pain treatments remain widely used despite modest relief and adverse effects,[23] the interest in nonpharmacological strategies is increasing, with neuromodulation targeting neural oscillations emerging as a compelling avenue.[11,23] This interest is grounded in the growing recognition that oscillatory activity shapes perception, cognition, and behavior,[6,20] contributing to the processing and experience of pain.[13,15,24]

Within this framework, alpha-band activity has emerged as a promising target.[14,15] Peak alpha frequency was shown to predict individual sensitivity to sustained pain,[7,8] and pain states are frequently accompanied by alpha suppression or slowing.[3,5,12,19] The crucial question, however, is whether alpha rhythms are simply markers of pain processing, or whether they can be modulated to reduce pain. To address this, considerable effort has focused on exogenous approaches—particularly transcranial alternating current stimulation.[4] Neurofeedback offers a complementary endogenous route: rather than imposing rhythms externally, it trains individuals to recruit them internally.[22] This process is considered to rely on reinforcement-based learning mechanisms engaging corticostriatal circuits, thought to support the gradual acquisition of control over specific neural dynamics.[22] Whether this form of self-regulation can induce reliable and specific effects on pain perception remains debated, given substantial interindividual variability and an incomplete understanding of the underlying neurophysiological mechanisms.[2,10,21]

Zheng et al[25] directly tested whether alpha oscillations could be volitionally engaged to reduce pain, using a double-blind, sham-controlled neurofeedback paradigm targeting somatosensory alpha during capsaicin-induced tonic pain. Participants receiving contingent neurofeedback exhibited greater alpha power over targeted somatosensory regions compared with those receiving sham feedback, indicating successful modulation of the trained rhythm. Importantly, this neural change was accompanied by reductions in pain ratings, with a notable dissociation: Pain unpleasantness decreased early and consistently, whereas pain intensity showed a later and more modest reduction.

The early reduction in unpleasantness is informative. Successfully influencing the feedback signal may enhance self-efficacy, positive affect, and engagement, all of which can meaningfully alter the affective burden of pain. The authors' mediation analyses are consistent with this interpretation, linking training efficacy and positive affect to reductions of unpleasantness. While such analyses cannot establish causality, they highlight an often underappreciated point: the effects of neurofeedback are embedded in the subjective experience of training, including engagement and perceived control. By contrast, the effect on pain intensity seems weaker and less consistent. While a late reduction is reported, the overall pattern suggests that neurofeedback more reliably modulates the affective than the sensory dimension of pain. Neural correlates of these effects—including alpha modulation and associated changes in large-scale EEG dynamics—suggest possible pathways through which local oscillatory regulation may relate to broader network processes.

The reported dissociation between pain unpleasantness and pain intensity deserves particular attention. While these components are closely related, they can be dissociated behaviorally and neurally, with partially distinct contributions from sensory-discriminative systems (including the somatosensory cortex and the posterior insula) and affective-motivational systems (comprising the anterior insula and the anterior cingulate cortex).[16–18] By showing distinct temporal trajectories for unpleasantness and intensity, Zheng et al. suggest that analgesic effects may emerge through partially independent mechanisms operating on different timescales, rather than reflecting a unitary reduction in a single pain dimension. Early changes in pain unpleasantness may reflect rapid engagement of affective-motivational systems, whereas later changes in pain intensity may depend on slower modulation of sensory-discriminative processing. This is not a trivial nuance; it fundamentally shapes how treatment effects should be interpreted. Importantly, an intervention that reduces unpleasantness may already be clinically meaningful, even in the absence of immediate changes in perceived intensity. These findings emphasize the need to consider how different dimensions of pain are assessed, and to align outcome measures and timescales with their potentially distinct temporal dynamics.

The study resonates with ongoing developments in pain modulation, as neurofeedback and brain stimulation techniques may represent converging strategies for engaging oscillatory mechanisms of analgesia. In fact, neurofeedback and brain stimulation may be viewed as distinct entry points into the same dynamic system, differing in whether they rely on endogenous control or externally imposed perturbation. Neurofeedback may help characterize individual capacity to regulate relevant rhythms,

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whereas brain stimulation approaches offer a complementary means of modulating these dynamics when endogenous control is limited. A key question is whether these approaches could interact in a systematic manner: for example, does successful alpha upregulation through neurofeedback predict responsiveness to stimulation, or can exogenous stimulation prime oscillatory circuits to enhance subsequent neurofeedback learning? Both approaches reflect a shared constraint—interindividual variability in oscillatory states. Understanding how they relate may enable combined or sequential interventions, in which stimulation and neurofeedback are used to inform and reinforce each other.

At the same time, several translational questions remain open. Because, in this study, sustained pain was experimentally induced in young, healthy volunteers, the model cannot capture the full complexity and psychosocial embedding of clinical pain. Moreover, as chronic pain conditions are often associated with altered alpha activity and disrupted large-scale dynamics,[3,5,12,19] it cannot be assumed that protocols effective in healthy participants will generalize straightforwardly to patient populations. A second challenge concerns heterogeneity. Alpha is not a single phenomenon: Its peak frequency, spatial generators, functional role, and responsiveness vary substantially across individuals.[1,9] Although neurofeedback is inherently individualized in its implementation, it often relies on standardized targets, such as fixed frequency bands or recording sites, which may obscure meaningful responder differences. Future work should test whether baseline alpha phenotype, individual peak alpha frequency, pain profile, or cognitive–affective traits predict benefit. Ultimately, the future of oscillation-based analgesia will depend not only on whether alpha can be trained, but on clarifying which dimensions of pain it influences, through which mechanisms, and over what timescales.

Conflict of interest statement

The author has no conflict of interest to declare.

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