Effects of long-lasting and intensive physical exercise regime on experimental pain response and sensitization, and their EEG GABAergic correlates: a case-control study

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Master thesis in Sport Science

Chronic pain (CP) is a major public health problem affecting millions worldwide. Current literature expresses the beta (β) oscillation, indicating GABA-dependent inhibition, as a potential objective pain biomarker and regular physical exercise as an attractive analgesic strategy. Thus, a better understanding of the underlying mechanisms of physical exercise analgesic effect could improve treatment decisions. This preliminary investigation comparatively assessed central sensitization (CS) clinical features, pain responses and the GABA-dependent inhibition dynamics during cold-induced pain in athletes and non-athletes.
This case-control study investigated 26 healthy right-handed, highly trained athletes (at least 7 h/week) and 24 age-and sex-matched non-athletes, all submitted to a cold pressor test (CPT) protocol consisting of hand immersion in ice-cold water at 4°C and concomitant high-density (64 electrodes) EEG recording (BIOSEMI). The numerical rating scale (NRS) for the sensory and affective (unpleasantness) dimensions measured pain perception at onset (threshold) and when it became unbearable (tolerance), reflecting pain sensitivity; the immersion time from pain threshold to tolerance (i.e. pain perception time, PPT) indicated resistance to pain; while the CS index (CSI) evaluated CS features.
Pain indicators tend to be lower in athletes than non-athletes but did not reach significance. When comparing the low (Lβ, 13-20Hz) and the high (Hβ, 21-30Hz) β global power spectrum (GPS) computed during cold and pain perceptions, athletes showed a decrease in both sub-bands. In contrast, non-athletes witnessed an increase (Lβ p=.014). During PPT, Hβ GPS further dropped in athletes (p=.008) and raised in non-athletes, while the Lβ GPS increased in both groups. This period also witnessed a negative correlation between Hβ GPS and PPT (R=-0.434, p=.028) in athletes and between Lβ GPS and CS scores (R=-0.438, p=.032), which correlated with PPT (R=-0.492, p=.015) in non-athletes. No EEG marker correlated to NRS scores.
As hypothesized, there is a trend of decreased pain sensitivity and CS but increased resistance to pain under the intensive physical exercise regime. Furthermore, compared to non-athletes, athletes display different GABAergic EEG markers modifications during pain and associations with pain indicators, affecting CS and resistance to pain. Indeed, it dissociates CS from resistance to pain and Lβ while reinforcing the link between Hβ and pain resistance.
This first insight into physical exercise-induced GABAergic modifications and their clinical correlates could mediate physical exercise's beneficial effect and participate in analgesia in CP patients, even if further investigations must confirm these preliminary results.

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