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Implication of the central nucleus of the amygdala in cardiovascular regulation and limiting maximum exercise performance during high-intensity exercise in rats

（高強度運動時の循環調節と最大運動パフォーマンスにおける扁桃体中心核の役割）

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Abstract

We elucidate the central mechanisms of cardiovascular regulation during high-intensity exercise with a focus on the hypothesis that amygdala activation acts to limit maximum exercise performance. In the first of three experiments using Wistar rats, we probed the involvement of the central nucleus of the amygdala (CeA) in such regulation. Rats were subjected to a maximum exercise test and their performance measures (total running time and cardiovascular responses) were compared before and after bilateral CeA lesions. In the second experiment, probing the role of central pathways, we tested whether high-intensity exercise activated neurons in CeA and/or the hypothalamic paraventricular nucleus (PVN) that project to the nucleus of the solitary tract (NTS). Finally, to understand the potential autonomic mechanisms affecting maximum exercise performance, we measured the cardiovascular responses in anesthetized rats to electrical microstimulation of the CeA, PVN, or both. We have found that (1) CeA lesions resulted in an increase in the total exercise time ($p < 0.05$) and the time at which an abrupt increase in arterial pressure appeared ($p < 0.05$), indicating an apparent suppression of fatigue. (2) We confirmed that high-intensity exercise activated both the PVN-NTS ($p < 0.001$) and CeA-NTS ($p < 0.001$) pathways. Moreover, we discovered that (3) while stimulation of the CeA or PVN alone both induced pressor (CeA: $p < 0.001$, PVN: $p < 0.01$), tachycardiac responses (CeA: $p < 0.01$, PVN: $p < 0.05$), and muscle blood flow (CeA: $p < 0.01$, PVN: $p < 0.01$), their simultaneous stimulation also increased muscle vascular resistance ($p < 0.05$). These results are evidence that cardiovascular responses during high-intensity exercise (a) are affected by CeA activation, which acts to limit maximum exercise performance, and (b) may implicate autonomic control modulating the PVN-NTS pathway via the CeA.