Last Word on Viewpoint: Reappraisal of the acute, moderate intensity exercise-catecholamines interaction effect on speed of cognition: role of the vagal/NTS afferent pathway

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TO THE EDITOR: Leischik et al. (see Ref. 1) provide an overview of the problems facing those researching the effects of exercise, both acute and chronic, on cognitive function. I agree with their comments, but the viewpoint (2) was only concerned with a specific anomaly in the ability of the catecholamine hypothesis to explain the effects of acute exercise on cognitive performance. This is only part of the process by which acute exercise affects immediate cognitive functioning and is not meant to be a comprehensive explanation of even the effects of acute exercise, let alone the comprehensive exercise-cognition interaction.

Jones (see Ref. 1) points to the fact that even moderate intensity exercise is a stressor and, as such, may initiate activation of the hypothalamic-pituitary-adrenal (HPA) axis hormones, resulting in release of norepinephrine by the locus ceruleus. This is in line with the argument put forward by my colleagues and me (3) that perceptions of stress lead to feed-forward from the higher centers of the brain to the hypothalamus, which projects to the locus ceruleus and induces the release of norepinephrine. However, this will only occur if the individual perceives the exercise as being stressful. Judging by the fact that moderate intensity exercise is generally thought to need to be of >45 min duration before plasma cortisol concentrations show an increase, one might claim that subcatecholamines threshold exercise is not perceived as being stressful enough to initiate HPA axis activation. However, this does not exclude the possibility of release of corticotrophin releasing hormone in the brain and this can initiate the synthesis and release of norepinephrine from the locus ceruleus (see Ref. 3 for a discussion). Although perceptions of stress vary from individual to individual, in all exercisers heart rate, tidal volume, and blood pressure begin to increase immediately after that exercise begins. Afferent feedback will activate mechanoreceptors, or more accurately stretch receptors, and baroreceptors on the vagus nerve and, via the nucleus tractus solitarii, stimulate the release of norepinephrine by the locus ceruleus.

Budde et al. (see Ref. 1) question how increased concentrations of norepinephrine affect cognition. This was not covered in the Viewpoint (2) but we have commented elsewhere. In McMorris et al. (3), we reviewed animal research showing that, in the prefrontal cortex, moderate concentrations of norepinephrine activate high-affinity $\alpha_{2A}$-adrenoceptors and dopamine D1 receptors, which work together to improve the signal-to-noise ratio. Thus central executive-type tasks are facilitated. Norepinephrine also activates the reticular formation resulting in increased arousal, which aids all types of task. Thus moderate intensity exercise directly aids cognition. Further increases in norepinephrine concentrations, because of undertaking heavy exercise or long-duration moderate intensity exercise, can have different effects as the lower affinity $\alpha_{1}$- and $\beta$-adrenoceptors are activated. These have a negative effect on prefrontal cortex-dependent tasks, by weakening the signal-to-noise ratio, but can aid performance of tasks requiring activation of the sensory cortices, as signal identification in these regions is facilitated. $\beta$-Adrenoceptors also aid long-term potentiation in the hippocampus and hence learning.

DISCLOSURES

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AUTHOR CONTRIBUTIONS

Author contributions: T.M. conception and design of research; T.M. analyzed data; T.M. interpreted results of experiments; T.M. drafted manuscript; T.M. edited and revised manuscript; T.M. approved final version of manuscript.

REFERENCES


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