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

COMMENTARY: REAPPRAISAL OF THE ACUTE, MODERATE INTENSITY EXERCISE-CATECHOLAMINES INTERACTION EFFECT ON SPEED OF COGNITION: ROLE OF THE VAGAL/NTS AFFERENT PATHWAY

TO THE EDITOR: The authors present an interesting analysis of the effect of moderate-intensity exercise-induced catecholamines on cognition speed (4). They summarize that moderate and catecholamine threshold (CT) exercise facilitates the speed of cognition and attribute it to activation of adrenergic neurons in the vagal/NTS/LC pathway (4). Catecholamine release is also regulated by stress (1), in addition to other factors. Stress, and subsequent stress hormone release, may affect this pathway and the resulting speed of cognition. Even moderate-intensity exercise is a stressor, which requires adaptive responses of many body systems (3), including the vagal/NTS afferent pathway. Corticotropin-releasing hormone (CRH) neurons release CRH to the pituitary gland for subsequent release of ACTH, but they also possess axons that terminate in the LC/NE-sympathetic system neurons in the NTS (3). Physiological stressors have been shown to activate neurons in the NTS and induce ACTH release (5). Cortisol released by the adrenal cortex in response to ACTH can result in negative feedback effects on ACTH secretion, the hypothalamic CRH neurons, and the LC/NE-sympathetic system (5). In this way, cortisol may cause disruptions in dopamine neurochemistry or alter regulation of dopamine release. Alterations in dopamine neurotransmission (excessive or insufficient) by cortisol can affect cognitive function (2), and individual responses to the stress of exercise may explain the lack of mean effect size differences for moderate vs. CT exercise. Conversely, moderate-intensity exercise-induced cortisol release may parallel the CT and positively affect dopamine-containing neurons excitability and speed of cognition.

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COGNITIVE ENRICHMENT AS MULTIDISCIPLINARY APPROACH AND VIEWPOINT

TO THE EDITOR: Increasing knowledge of the interactions in the brain suggests that exercise catecholamines are not only one of the interacting components on cognition (2). Microvasculature (endothelium/capillaries), cell function (neurons/glia cells), synapses, and social communication are a complex system that regulates cognitive function and plasticity/neurochemistry of the brain (4). Physical activity (PA) is a polypill for metabolism and atherosclerosis (3). There also exists a great desire to improve cognitive performance or to protect the decline of cognition in the elderly or even more of Alzheimer disease using PA (1). Many different factors may influence neurotransmitters involved in brain function and cognition (2, 4). Clinically based research in neuroscience and other related fields argues that as people age, brain plasticity processes with negative consequences begin to dominate brain functioning (4). Four core factors, reduced schedules of brain activity, noisy processing, weakened neuromodulatory control, and negative learning, interact to create downward regulation spiral (4). Driving brain plasticity with positive outcomes requires complex training paradigm in demanding sensory, cognitive, and motor activities on an intensive basis in physical/physiological rehabilitation (4). In behavioral context, all daily living/social activities have to be included in pathophysiological models. Physical activity alone with enhancement of catecholamines and vagal pathways (5) are very simplified mechanisms that may influence the cognitive function of the brain. The concept of cognitive enrichment (2) is a clinical challenge of the 21st century in medical, neurological, psychological, and social sciences (multidisciplinary approach). Positive effects of PA on maintaining cognitive function and their complex neurochemical processes are open for multimodal intervention research.

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HOW DOES ACUTE EXERCISE INFLUENCE COGNITION?

TO THE EDITOR: McMorris (4) composed a well-written and interesting Viewpoint. The main hypothesis is that acute exercise changes the concentration of catecholamines in the brain, which leads to a higher speed of cognition. This adds

to the topic because there is no monocausal explanation for this relationship. However, propositions about catecholamine changes due to exercise are very complex, there are region-specific interactions between the catecholamines and various receptors, transporters, and autoreceptors that can have both stimulating and inhibiting effects (3). There is a possible link but no cause and effect, suggesting that the relationship is much more complex than that asserted by proponents of the catecholamine hypothesis. It further remains unclear how an elevation in catecholamines would neurobiologically enhance cognition. McMorris (4) further describes the problem of permeability of the blood-brain barrier (BBB) for catecholamines, indicating that a possible positive cognitive effect of hormones, crossing the BBB needs to be tested specifically. Previous research shows that steroid hormones are possibly involved in the acute exercise-cognition link (1). In addition to the issue of intensity outlined by McMorris (4), the type of exercise is an important moderator of moderate acute exercise effects on cognition. Budde et al. (2) observed significantly higher attention after 10 min of coordinatively demanding exercise compared with endurance exercise. With the heart rate being the same in both groups, it was assumed that the coordinative character was responsible for the differences. This is interesting because previous findings suggest that the type of exercise affects the catecholamine responses strongly (5).

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