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Review

Is there an acute exercise-induced physiological/biochemical threshold which triggers increased speed of cognitive functioning? A meta-analytic investigation

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Abstract

Purpose: The purpose of this study was to examine, using meta-analytic measures, the evidence regarding the optimal exercise intensity at which improvements in speed of cognitive function are triggered. Specifically, it was hypothesized that the catecholamine, lactate, and ventilatory thresholds is the point at which significant improvements in speed of cognitive function are observed.
Methods: We compared mean effect sizes for threshold studies and for those studies where exercise intensity was classed as moderate (40%–79% VO_{2max} or equivalent) but in which the thresholds were not measured.
Results: Random effects meta-analysis showed significant, moderate, mean effect sizes for studies at the threshold ($g = 0.58$, $Z = 2.98$, $p < 0.003$) and for those during moderate intensity exercise but in which the threshold was not measured ($g = 0.54$, $Z = 5.01$, $p < 0.001$). There was no significant difference between mean effect sizes, which suggests that the thresholds are unlikely to represent a trigger point.
Conclusion: Moderate intensity exercise, even below the thresholds, can induce improved speed of cognition, possibly due to a combination of increased peripheral catecholamine concentrations inducing vagal/nucleus tractus solitarii pathway activation and central increases due to perceptions of stress.

Keywords: Catecholamine threshold; Lactate threshold; Stress; Vagus nerve; Ventilatory threshold

1. Introduction

Meta-analyses[1–5] and qualitative reviews[6–9] have provided support, albeit limited in some cases, for the hypothesis that acute, moderate intensity exercise has a positive effect on speed of cognitive functioning. However, an issue, first highlighted by Tomporowski and Ellis[6] in their seminal review that has yet to be settled, is the exercise intensity at which significant improvements in speed of cognitive functioning are triggered. The purpose of this study was to examine, using meta-analytic measures, the evidence for the existence of a specific trigger point or exercise threshold at which benefits to cognitive performance are optimized. We have limited the review to examination of speed of cognitive function rather than including both speed and accuracy due to the fact that McMorris and colleagues[4,5] found that research in which accuracy was the dependent variable did not demonstrate a significant improvement in performance. They explained that this lack of a significant effect for accuracy measures was probably due to the inability of the tests of accuracy used in those studies to accurately differentiate performance as a result of factors such as ceiling and floor effects.

As Tomporowski and Ellis[6] stated, early attempts to determine an exercise intensity which would induce optimal performance were somewhat arbitrary in nature. The first to suggest a theoretically based intensity were Chmura et al.,[10] who examined the possibility that the norepinephrine (NE) and epinephrine (Epi) thresholds, the points at which plasma

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concentrations of NE and Epi demonstrate the beginning of an exponential rise[11] in response to exercise, would induce increased speed of cognition. These thresholds are highly correlated[12] and, given that NE and Epi are catecholamines, we will use the catecholamine threshold (T_{CATS}) as a single descriptor for both. As measurement of T_{CATS} requires the taking of venous blood samples, other authors have preferred to measure lactate or ventilatory thresholds, which require less invasive methods. Blood lactate concentrations follow a similar exponential profile to catecholamines in response to exercise, and the lactate threshold (T_{LA}) shows moderate to high correlations with T_{CATS}.[12,13] Moreover, the ventilatory threshold (VT), the point at which ventilatory carbon dioxide shows a greater increase than ventilatory oxygen[14] with increasing exercise intensity, occurs about the same time as T_{LA}[15] and is also moderately to highly correlated with T_{LA} and T_{CATS}.[16] It is important to note that, with reference to the acute exercise-cognition interaction, VT and T_{LA} are only important in that they correlate with T_{CATS}. It is the effect of exercise at T_{CATS} on brain concentrations of dopamine and NE which is the key factor in the acute exercise-cognition interaction, at least from a neurochemical perspective.

The interesting point about these thresholds is that they are biomarkers of increases in exercise intensity from a level which requires no significant increase in physiological or biochemical responses to a level at which there are significant peripheral changes. As we will see in Section 3, there is strong theoretical evidence and some empirical evidence, albeit mainly from animal studies, to support the argument that these peripheral changes induce increases in brain concentrations of the catecholamine neurotransmitters, dopamine and NE.[17] More importantly, with regard to the acute exercise-cognition interaction in the brain, these neurotransmitters play major roles in cognition, arousal, and motor control.[18] Therefore, we hypothesized that speed of cognition at or immediately following acute exercise at T_{CATS}, T_{LA}, and VT would demonstrate a moderate to high mean effect size. Moreover, mean effect sizes for speed of cognitive performance at the thresholds would be significantly higher than that of individuals exercising at a moderate intensity. It is important to point out that in most/all of the studies in which exercise was at moderate intensity, thresholds were not assessed; hence a limitation of this review is that it is possible that some of the studies in the moderate intensity group actually included some individuals who were exercising at threshold. A literature search using computer databases was undertaken to identify studies claiming to use moderate intensity exercise. Only studies where exercise intensity fell between 40% and 79% maximum power output (W_{max}) or equivalents were included in the analyses.

2. Mechanistic explanation of the importance of T_{CATS}

2.1. Catecholamine synthesis and release

The synthesis of catecholamines takes place both centrally, within the brain, and peripherally. The precursor for catecholamine synthesis is the aromatic amino acid tyrosine, which is either taken directly from food or is formed in the liver by the hydroxylation of phenylalanine. Thus tyrosine is readily available peripherally and is transported across the blood–brain barrier by the facilitative transporter L1.[19] In both the brain and peripherally, tyrosine is broken down into the metabolite 3,4 dihydroxy-L-phenylalanine (L-DOPA), under the influence of tyrosine hydroxylase (TH). L-DOPA is then catalysed by aromatic amino acid decarboxylase (AADC) and dopamine is formed. In neurons that use dopamine as a neurotransmitter, no further action occurs and the dopamine is stored in vesicles. In neurons that use NE as the neurotransmitter, dopamine is further synthesized into NE. This takes place with the aid of dopamine-β-hydroxylase (DBH). The majority of NE is stored in vesicles in these neurons and there is no further processing. In the periphery, dopamine is stored in some neurons in the pulmonary artery and kidney. The majority of peripheral dopamine, however, is further synthesized into NE. This takes place in the granules of cells in the adrenal medulla. In about 15% of the granules the process terminates and NE is stored. The rest of the NE diffuses back into the cytoplasm where it is *N*-methylated by phenylethanolamine-*N*-methyltransferase (PNMT) and Epi is synthesized. Epi is then transported back into chromaffin granules for storage in the medulla of the adrenal glands. In the brain, as in the periphery, the further synthesis of NE into Epi requires the presence of PNMT. This is present only in a few neurons in the pons and medulla. Some NE is *N*-methylated by PNMT in these neurons, thus a small amount of Epi is synthesized and stored in the brain. It should be noted that TH is the rate-limiting enzyme in the whole process.[20]

2.2. Catecholamines and brain functions

NE, dopamine, and, to a much lesser extent, Epi act as neurotransmitters in the brain. Once synthesized they are held in vesicles and, when released, innervate the noradrenergic and dopaminergic pathways. The neurons serving the noradrenergic pathway are mainly found in the locus coeruleus. They rely on NE as the neurotransmitter and innervate most areas of the brain including those regions involved in working memory (e.g., prefrontal cortex, anterior cingulate cortex), perception (e.g., somatosensory cortex, parietal cortex), attention (e.g., reticular activation system, amygdala) and long-term memory (e.g., hippocampus). Neurons serving the dopaminergic pathway are found mainly in the substantia nigra and ventral tegmental regions, and innervate the hippocampus, amygdala, dorsolateral prefrontal cortex, basal ganglia—all areas involved in cognition and memory.[21]

NE and dopamine work together to control cognition and their efficiency is affected by stress levels. When stress levels are low, performance is comparatively poor as receptor activation is limited. When stress rises to a moderate level, brain catecholamine concentrations rise and there is increased firing of the high affinity α_{2A}-adrenoreceptors by NE,[22] which increases the strength of the neural signal.[23] Similarly the high affinity D1 dopaminergic receptors are