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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

activated by dopamine, which dampens the "noise" by inhibiting firing to non-preferred stimuli,[24] thus improving the signal to "noise" ratio. This should lead to optimal performance. However, when stress levels are high, NE and dopamine concentrations become excessive. The excess NE activates the lower affinity α1- and β-adrenoreceptors.[22] Activation of α1-adrenoreceptors results in reduced neuronal firing in the prefrontal cortex by phosphatidylinositol-protein kinase C intracellular signalling pathway activation, while excessive stimulation of D1 receptors and β-adrenoreceptors induces excess activity of the secondary messenger cyclic adenosine monophosphate which dampens all neuronal activity, thus weakening the signal to "noise" ratio in the prefrontal cortex.[22–24]

Since Tomporowski and Ellis'[6] review, most authors have assumed that exercise is a stressor and that moderate intensity exercise is equivalent to moderate levels of stress. However, as we described in Section 1, the nature of moderate intensity exercise needs to be more rigorously scrutinized as does the assumption that moderate intensity exercise induces increased brain concentrations of NE and dopamine. In order to do that we begin, in the next sub-section, to examine the relationship between plasma and central concentrations of catecholamines relative to acute bouts of exercise.

2.3. Acute exercise-induced increases in brain catecholamines concentrations

Centrally, dopamine and NE play major roles in the activation of areas of the brain critical for movement including the premotor cortex, supplementary motor area, basal ganglia, and somatosensory and parietal cortices; thus there will be increased brain turnover of these neurotransmitters during exercise. Peripherally, during low intensity exercise, NE and Epi aid lipolysis, stimulate receptors in muscle, activate receptors in the pancreas to suppress insulin release, and stimulate secretion of the hormones glucagon, growth hormone and cortisol. As exercise intensity increases hypoglycemia occurs, which results in large increases in plasma NE concentrations. Epi concentrations rapidly increase when there is a decline in hepatic glucose concentrations. At this stage, Epi stimulates glycogenolysis and hepatic glucose release in the liver, and glycogenolysis in muscle. Epi and, to a lesser extent, NE also act on the cardiovascular system by activating receptors responsible for increasing heart rate and contractile force, while stimulating arteriolar constriction in renal, splanchnic and cutaneous vascular beds.[25,26] Thus exercise will not only induce increased peripheral and central catecholamine concentrations because it is stressful but also because we could not maintain work rate without such an increase.

In order to meet the demand for catecholamines centrally and peripherally, during and even immediately before exercise, the hypothalamus and brainstem initiate action of the sympathoadrenal system. This results in the release of catecholamines at the postganglionic cells of those neurons that require activating or inhibiting. When exercise reaches a moderate level, there is a large increase in plasma catecholamines which are important in the regulation of the cardiovascular system. It is at this stage that T_{CATS} is reached. According to Chmura et al.,[10] this is when optimal cognitive functioning is induced. However, catecholamines do not cross the blood–brain barrier. Nevertheless, peripherally circulating Epi and NE activate β-adrenoreceptors on the afferent vagus nerve, which runs from the abdomen through the chest, neck, and head, and terminates in the nucleus tractus solitarii (NTS) within the blood–brain barrier. Glutamate mediates synaptic communication between the vagal afferents and the NTS, allowing noradrenergic cells in the NTS, which project into the locus coeruleus, to stimulate NE synthesis and release to other parts of the brain.[27] This may also affect brain dopamine concentrations, as Devoto et al.[28] showed that electrical stimulation of the rat locus coeruleus resulted in increased concentrations of dopamine and one of its metabolites, 3,4-dihydroxyphenylacetic acid (DOPAC).

2.4. Empirical evidence for acute exercise-induced increases in brain catecholamine concentrations

Research into the effect of acute exercise on brain concentrations of catecholamines has been largely done in animal studies, mostly using microdialysis. Increased dopamine concentrations have been shown particularly in the brainstem and hypothalamus during and immediately following acute exercise.[17] However, Hattori et al.[29] found that this only occurred when intensity increased to a moderate level. Kitaoka et al.[30] supported previous findings showing increased dopamine concentrations in the hypothalamus. Although the results for studies measuring catecholamine brain concentrations show support for increased dopamine concentrations, they provide limited support for increased NE concentrations. The effect of acute exercise on whole brain concentrations of NE in animals has shown either a decrease in concentrations or no significant effect.[17] More recently, Kitaoka et al.[30] demonstrated increased NE concentrations in the hypothalamus. Moreover, animal studies have shown increases in brain concentrations of the NE metabolite 3-methoxy 4-hydroxyphenylglycol (MHPG), and the dopamine metabolites DOPAC and 4-hydroxy 3-methoxyphenylacetic acid, also known as homovanillic acid (HVA). Increased concentrations of MHPG have been found in most brain regions,[17] while increased concentrations of DOPAC and HVA have been shown, particularly in the brainstem and hypothalamus.[31] These results indicate increased turnover of dopamine and NE by the brain during exercise. Animal studies have also shown increased TH activity during exercise.[29] This would facilitate the synthesis of catecholamines during exercise, as TH is the rate-limiter for catecholamine synthesis.

As far as we know, only two studies exploring the effects of exercise on brain catecholamines have, so far, been attempted with humans. Wang et al.[32] examined the effect of treadmill running on striatal dopamine release in the human brain, using intravenous injection of the radiotracer [^{11}C]raclopride and

positron emission tomography (PET) scans of the putamen and cerebellum. Exercising on a treadmill at an intensity >85% estimated maximum heart rate (HR_{max}) showed no significant effect. The authors argued that this was probably due to the PET protocol not being robust enough to highlight small changes in [^{11}C]raclopride binding. Dalsgaard et al.[33] examined NE and Epi concentrations in cerebrospinal fluid (CSF), by lumbar puncture and the arterial to internal jugular venous difference (*a-v* diff) following exercise to exhaustion. Arterial concentrations of NE and Epi showed large increases but the *a-v* diff was not affected significantly indicating that there was no uptake by the brain. Exercise increased the CSF concentrations of NE only. The authors argued that the CSF increase in NE concentrations was probably due to activity in the locus coeruleus. This study provides some support for acute exercise inducing increased brain concentrations of NE in humans.

2.5. Summary

Theoretically, exercise at T_{CATS} and related thresholds, T_{LA} and VT, will result in peripheral NE and Epi initiating vagal/NTS pathway stimulation which in turn will result in synthesis and release of NE from the locus coeruleus to the rest of the brain. Animal studies would also suggest that this induces increased brain concentrations of dopamine. Thus one would expect improved cognitive functioning. Moreover, acute exercise-induced expression of brain TH messenger ribonucleic acid should facilitate synthesis and release of catecholamines in the brain.

3. Materials and methods

A literature search using the computer databases PsycArticles, PsycINFO, PubMed, SportsDISCUS and Web of Knowledge was undertaken. Key words used in the searches were combinations of ''acute'', ''exercise'', ''cognition'', ''cognitive function'', ''cognitive performance'', ''reaction time'', ''response time'', ''working memory'', ''short-term memory'', ''long-term memory'', ''memory'', ''recall'', ''executive function'', ''central executive'', ''oddball'', ''visual search'', ''attention'', ''anticipation'', ''coincidence anticipation'', ''decision making'', ''Flanker Task'', ''Simon Task'', ''Sternberg Test'', ''Wisconsin Card Sorting Task'', ''Tower of London'', and ''Tower of Hanoi''. In addition, reference lists from empirical reports and reviews were examined. Studies were included if they were performed on healthy individuals; the exercise intervention required the activation of large muscle groups; repeated measures and within-subject design were used; data were provided showing the intensity of the exercise with reference to a maximum or threshold; exercise intensity was based on objective measures; the dependent variables were measures of time; the range for a specific intensity was not $\geq$10% W_{max} or equivalent; and the dependent variables were behavioral and objective. Studies including pharmacological treatments were not included.

3.1. Definition of moderate exercise

As the second hypothesis required comparison of speed of cognitive performance at or immediately following acute exercise at T_{CATS}, T_{LA}, and VT with that when the individuals were exercising at a moderate intensity but one in which the thresholds had not been measured, we needed to define ''moderate'' intensity exercise. Based on Borer's[34] classification, moderate intensity exercise was determined as being between 40% and 79% W_{max} or equivalent. If W_{max} values were not presented but percent VO_{2max} or percent HR_{max} were given, the conversion formulae of Arts and Kuipers[35] were applied. For other indicators of intensity, e.g., percent heart rate reserve (HR_{RES}), percent maximum aerobic power (MAP), percent VT, and percent T_{LA}, the exercise physiology and exercise endocrinology literatures were examined to ascertain whether or not the intensity would be considered to be below, within, or above the 40%−79% W_{max} limits. This is in line with the definitions used by McMorris and colleagues[4,5] in previous studies. T_{CATS} is thought to occur at ~75% VO_{2max},[12] which equates to ~65% W_{max}, but there are large inter-individual differences, with some participants demonstrating T_{CATS} at as low as 40% W_{max}.[13]

3.2. Data analyses

When means and SDs were available, effect sizes for reaction time measures were calculated using Cohen's *d* formula (mean at rest−mean during or following exercise/SD at rest, where rest acts as the control). Studies in which means and SDs were only provided graphically were not included as it was not possible to accurately determine the means or SDs. Each individual *d* was then transformed to the bias-corrected standardized mean difference, Hedges' *g*, by applying the correction factor *J* ($J = 1-(3/4df-1)$) and this was used to calculate a mean effect size using the random-effects model. Results of the *Q* test for homogeneity were calculated and reported as was τ^2, which is a measure of absolute variance whereas *Q* is a measure of relative variance.[36] Orwin's[37] Fail-safe *N* was calculated. In the sub-group analyses, effect sizes for each group were compared using a *Z*-test on the differences with a random-effects model, with separate estimates of τ^2 for each sub-group. The proportion of variance explained by the moderator variable, R^2, was calculated.[36] Where studies provided more than one effect size, in order to control for one or more studies having an undue bias on the results, one effect size per study was calculated, except for one study,[38] which provided data for two independent treatment groups and two others[39,40] which reported two separate experiments. These were treated as separate studies.

4. Results

The literature reviewed showed that there were 10 articles which examined the effect of acute exercise at T_{CATS}, T_{LA}, or VT on speed of cognition and which met the criteria for

inclusion and provided sufficient statistical information. This meant that there were 10 effect sizes and 125 participants. The types of tasks and exercise intensities used in each study can be seen in Table 1.

The literature reviewed also showed that there were 33 articles which examined the effect of moderate, acute exercise on speed of cognition and which met the criteria for inclusion and provided sufficient statistical information. As previously described, one study[38] provided data for two independent treatment groups and two others[39,40] reported two separate experiments, therefore these were treated as separate studies. In total, there were 36 effect sizes and 800 participants. The types of tasks and exercise intensities used in each study can be seen in Table 2.

In order to compare mean effect size at the thresholds and mean effect size from studies at moderate intensity, it was necessary to measure the overall effect size.[36] This showed that effect sizes were heterogeneous, $Q(45) = 171.73$ ($p < 0.001$), $\tau^2 = 0.29$. The mean effect size was significant, $g = 0.55$ ($Z = 5.79$, $p < 0.001$), variance 0.01, SE = 0.10, and 95% confidence interval (CI): 0.36−0.74. The fail-safe N was 81 indicating that this number of non-significant studies would be required to lower the effect size to below $g = 0.00$. Forty-one effect sizes were positive and five were negative.

Effect sizes for studies at threshold were heterogeneous $Q(9) = 22.25$ ($p < 0.008$), $\tau^2 = 0.23$. The mean effect size was significant, $g = 0.58$ ($Z = 2.98$, $p < 0.003$), variance 0.04, SE = 0.20, and 95%CI: 0.20−0.97. The fail-safe N was 19 indicating that this number of non-significant studies that would be required to lower the effect size to below $g = 0.00$. Eight effect sizes were positive and two were negative (Fig. 1).

Effect sizes for moderate intensity exercise studies were heterogeneous, $Q(35) = 149.46$ ($p < 0.001$), $\tau^2 = 0.30$. The mean effect size was significant, $g = 0.54$ ($Z = 5.01$, $p < 0.001$), variance 0.01, SE = 0.11, and 95%CI: 0.33−0.75. The fail-safe N was 61 showing that this many non-significant studies would be necessary to decrease the observed effect size to below $g = 0.00$. Thirty-three effect sizes were positive and three were negative.

Importantly, there was no significant difference ($p > 0.05$) between mean effect sizes for the two groups (i.e., studies of exercise at threshold and studies of exercise at moderate intensity) (Fig. 2).

Observation of Table 2 showed that in six studies, two of which involved two experiments,[39–41] participants were exercising at intensities close to 75% VO_{2max} (~65% Wmax), which Podolin et al.[12] described as being approximately the intensity at which T_{CATS} occurs. Therefore, we decided to remove these studies from the data for moderate intensity exercise studies. This made very little difference to the results. Effect sizes for studies if exercise at moderate intensity remained heterogeneous, $Q(28) = 137.79$ ($p < 0.001$), $\tau^2 = 0.35$. The mean effect size was significant, $g = 0.63$ ($Z = 4.88$, $p < 0.001$), variance 0.02, SE = 0.13, and 95%CI: 0.38−0.88. Again there was no significant difference between groups.

5. Discussion

5.1. Acute exercise-cognition interaction at the thresholds

Observation of the positive and significant medium, mean effect size for studies at the thresholds may at first appear to support the hypothesis that acute exercise at the thresholds is the point at which peripheral physiological and biochemical changes trigger increased speed of cognitive functioning. The data undoubtedly do support those researchers who have chosen acute exercise at one or another of the thresholds to induce increased speed of cognition. However, the fact that moderate intensity exercise also showed a significant medium, mean effect size raises doubts as to the extent to which the thresholds are a trigger-point. More importantly, the two mean effect sizes do not differ significantly. Thus, it would appear from these data that moderate intensity exercise, which we classed as being 40%−79% VO_{2max} or equivalent, is sufficient to induce improved cogntion. Moreover, when we removed those studies[38,42] that were most likely to be above the thresholds from the studies in which thresholds were not measured, it did not significantly alter the results. Given that exercise sub-threshold does not induce increased plasma catecholamines concentrations, the question remains as to how exercise of this intensity affects cognition.

Table 1
Types of threshold, exercise duration, and cognitive tasks used by authors in threshold studies.

Study	n	Threshold type	Exercise duration	Cognitive test	Effect size (g)
Davranche and McMorris[60]	12	Ventilatory	21 min	Simon Task	0.39
Kashihara and Nakahara[61]	6	Lactate	10 min	Choice reaction time	0.87
Córdova et al.[38] —Test 3	12	~OBLA 4 mmol/L	20 min	Simple reaction time, Tower of Hanoi	−0.01
McMorris et al.[62]	12	Epinephrine	~12 min[a]	Soccer decision making	−0.14
McMorris et al.[63]	9	Epinephrine	12 min	Soccer decision making	0.46
Collardeau et al.[64]	11	Ventilatory	90 min	Simple reaction time	0.47
McMorris et al.[65]	12	Lactate	~12 min[a]	Choice reaction time	0.21
Hyodo et al.[66]	16	Ventilatory	10 min	Stroop Task	0.79
Chmura et al.[10]	22	Epinephrine, norepinephrine	~15 min[a]	Choice reaction time	1.89
Chmura and Nazar[67]	13	OBLA 4 mmol/L	~18 min[a]	Choice reaction time	0.87

Abbreviation: OBLA = onset of blood lactate.
[a] Incremental increases in workload until threshold reached, exercise at threshold ~2−3 min.

Table 2
Exercise intensities and durations, and cognitive tasks used in moderate intensity exercise studies.

Study	*n*	Exercise intensity	Exercise duration	Cognitive task	Effect size (*g*)
Joyce et al.[68]	10	40% MAP	30 min	Stop signal	0.35
Brisswalter et al.[69]	10	50% VO_{2max}	6 min	Simple reaction time	−1.03
Pesce et al.[70]	25	60% HR_{RES}	See Note[a]	Visual attention switching	0.15
Pesce et al.[71]	16	60% VO_{2max}	See Note[a]	Visual attention switching	1.17
Hogervorst et al.[41]	15	75% W_{max}	60 min	Simple reaction time Choice reaction time Stroop Task	0.28
Cereatti et al.[72]	24	60% HR_{RES}	See Note[a]	Visual attention switching	2.35
Heckler and Croce[73]	18	70% HR_{max}	20 min	Arithmetic	0.60
Pesce et al.[74]	42	60% HR_{RES}	See Note[a]	Visual attention switching	0.89
Pesce et al.[75]	48	60% HR_{RES}	See Note[a]	Visual attention switching	1.15
Pesce et al.[76]	16	60% VO_{2max}	See Note[a]	Visual attention switching	1.30
Pontifex and Hillman[77]	41	60% VO_{2max}	6.5 min	Flanker Task	0.01
Davranche et al.[78]	12	50% MAP	See Note[b]	Choice reaction time	0.56
Davranche et al.[79]	14	50% MAP	20 min	Flanker Task	0.40
Kamijo et al.[80]	24	30%, 50% VO_{2max}	20 min	Flanker Task	0.76
Davranche et al.[42]	11	90% VT	17 min	Choice reaction time	1.05
Ozymesci-Taskiran et al.[81]	11	70% HR_{max}	20 min	Electromyographic reaction time	0.53
McMorris et al.[82]	9	70% W_{max}	See Note[c]	Non-compatible choice reaction time	0.16
Yanagisawa et al.[83]	20	50% VO_{2max}	10 min	Stroop Task	2.13
McMorris and Keen[84]	12	70% W_{max}	See Note[c]	Simple reaction time	−0.20
Pesce and Audiffren[85]	100	60% HR_{RES}	20−24 min	Choice reaction time	1.13
McMorris et al.[55]	24	50% W_{max}	15 min	Flanker Task	0.78
Arcelin et al.[86]	22	60% MAP	10 min	Choice reaction time	0.59
McMorris and Graydon[87]	20	70% W_{max}	See Note[c]	Soccer decision making	0.57
McMorris and Graydon[40]−Exp 1	12	70% W_{max}	See Note[c]	Visual search	0.05
McMorris and Graydon[40]−Exp 2	12	70% W_{max}	See Note[c]	Soccer decision making	0.28
McMorris and Graydon[39]−Exp 1	10	70% W_{max}	See Note[c]	Soccer decision making	0.38
McMorris and Graydon[39]−Exp 2	20	70% W_{max}	See Note[c]	Soccer decision making	0.18
Brisswalter et al.[88]	20	40%, 60% MAP	10 min	Simple reaction time	−1.53
Delignières et al.[89]	20	40%, 60% VO_{2max}	4 min	Choice reaction time	0.21
Guizani et al.[90]	12	40%, 60% VO_{2max}	6 min	Simple reaction time Choice reaction time	0.65
Fontana et al.[91]	32	40%, 60% VO_{2max}	Not reported	Soccer decision making	0.44
Endo et al.[92]	13	40%, 60% W_{max}	15 min	Stroop Task	1.24
Nanda et al.[93]	10	70% HR_{RES}	30 min	Memory Reasoning Concentration Planning	0.83
Hogan et al.[94]	71	50% HR_{RES}	15 min	*n*-back Task	0.29
Córdova et al.[38]−Test 1	12	60% T_{LA}	20 min	Simple reaction time Tower of Hanoi	0.11
Córdova et al.[38]−Test 2	12	90% T_{LA}	20 min	Simple reaction time Tower of Hanoi	0.48

Abbreviations: MAP = maximum aerobic power; VO_{2max} = maximum volume of oxygen uptake; HR_{RES} = heart rate reserve; W_{max} = maximum power output; HR_{max} = maximum heart rate; VT = ventilatory threshold; T_{LA} = lactate threshold.
[a] Incremental warm-up to target HR followed by 3−4 min at target (total time: ∼14 min).
[b] 3 × 14−15 min sets with 10-min rest between sets.
[c] Incremental warm-up to target resistance followed by ∼2 min at target (total time: ∼12 min).

In attempting to answer the question of how does exercise of this intensity affect cognition, we should first acknowledge that some authors claim that the key issue is increased cerebral blood flow.[43] However, most authors subscribe to the theory that the key point is increased arousal,[6−9] which is largely reliant on increased central concentrations of the neurotransmitters dopamine and NE. As we saw in Section 2.2, moderate stress leads to increased firing of the high affinity α_{2A}-adrenoreceptors by NE,[22] which increases the strength of the neural signal. Similarly the high affinity D1 dopaminergic receptors are activated by dopamine,[24] which dampens the "noise" by inhibiting firing to non-preferred stimuli, thus improving the signal to "noise" ratio.[22−24] The literature on the effect of sub-threshold level exercise on plasma concentrations of catecholamines presents two possible answers as to how sub-threshold level exercise may affect central concentrations of dopamine and NE.

In several of the studies, exercise duration is ≥30 min (Table 2). During exercise at sub-threshold intensities, plasma catecholamine concentrations begin to rise after ∼30 min.[44] In fact, Chmura et al.[45] actually showed a significant increase in plasma catecholamines at 20 min for a group who

Study	Subgroup within study	Statistics for each study						
		Point estimate	SD	Variance	Lower limit	Upper limit	Z-value	p-value
Davranche and McMorris[60]	Threshold	0.391	0.385	0.148	-0.362	1.145	1.018	0.309
Kashihara and Nakahara[61]	Threshold	0.867	0.522	0.272	-0.155	1.889	1.662	0.097
Córdova et al.[38] –Test 3	Threshold	-0.009	0.380	0.145	-0.775	0.736	-0.024	0.980
McMorris et al.[62]	Threshold	-0.140	0.381	0.145	-0.886	0.607	-0.367	0.714
McMorris et al.[63]	Threshold	0.482	0.434	0.188	-0.389	1.313	1.065	0.287
Collardeau et al.[64]	Threshold	0.472	0.401	0.181	-0.314	1.257	1.177	0.239
McMorris et al.[65]	Threshold	0.205	0.382	0.146	-0.543	0.953	0.537	0.591
Hyodo et al.[66]	Threshold	0.789	0.350	0.123	0.102	1.475	2.253	0.024
Chmura et al.[10]	Threshold	1.890	0.354	0.125	1.197	2.583	5.343	0.000
Chmura and Nazar[67]	Threshold	0.872	0.387	0.150	0.113	1.531	2.252	0.024
		0.595	0.124	0.015	0.353	0.837	4.815	0.000

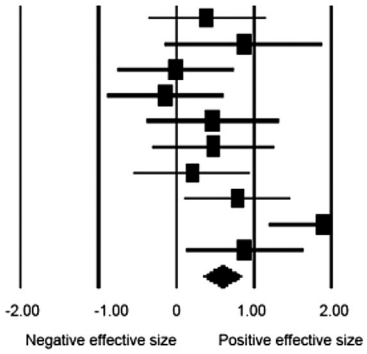

Fig. 1. Effect sizes for studies at threshold.

exercised at 75% T_{LA}. It is a distinct possibility that this moderate-to-long duration, moderate intensity exercise may well result in a sufficient increase in plasma catecholamines for the vagal/NTS pathway to be stimulated. It should be noted, however, that, if exercise continues for ≥45 min, the hypothalamic-pituitary-adrenal (HPA) axis is activated. This stimulates the synthesis and release of corticotrophin releasing factor (CRF) and adrenocorticotrophin hormone (ACTH) centrally, and cortisol centrally and peripherally.[46] HPA axis hormones affect cognitive functioning mostly by interacting with dopamine and NE. In the locus coeruleus, CRF neurons innervate noradrenergic neurons and NE is released.[47,48]

Study	Subgroup within study	Statistics for each study						
		Point estimate	SD	Variance	Lower limit	Upper limit	Z-value	p-value
Joyce et al.[68]	Non-threshold	0.348	0.414	0.171	-0.462	1.159	0.842	0.400
Brisswalter et al.[69]	Non-threshold	-1.027	0.441	0.194	-1.891	-0.162	-2.328	0.020
Pesce et al.[70]	Non-threshold	0.145	0.274	0.075	-0.392	0.683	0.530	0.598
Pesce et al.[71]	Non-threshold	1.169	0.366	0.134	0.451	1.886	3.190	0.001
Hogervorst et al.[41]	Non-threshold	0.284	0.348	0.121	-0.397	0.965	0.817	0.414
Cereatti et al.[72]	Non-threshold	2.351	0.368	0.136	1.629	3.072	6.385	0.000
Heckler and Croce[73]	Non-threshold	0.602	0.326	0.107	-0.038	1.242	1.845	0.065
Pesce et al[74]	Non-threshold	0.893	0.225	0.051	0.452	1.334	3.970	0.000
Pesce et al.[75]	Non-threshold	1.151	0.217	0.047	0.725	1.577	5.297	0.000
Pesce et al.[76]	Non-threshold	1.302	0.373	0.139	0.570	2.033	3.487	0.000
Pontifex and Hillman[77]	Non-threshold	0.010	0.217	0.047	-0.415	0.435	0.045	0.964
Davranche et al.[78]	Non-threshold	0.559	0.389	0.151	-0.203	1.321	1.438	0.151
Davranche et al.[79]	Non-threshold	0.396	0.360	0.130	-0.310	1.102	1.100	0.271
Kamijo et al.[80]	Non-threshold	0.764	0.290	0.084	0.196	1.333	2.636	0.008
Davranche et al.[42]	Non-threshold	1.045	0.425	0.180	0.213	1.878	2.461	0.014
Ozymesci-Taskiran et al.[81]	Non-threshold	0.527	0.402	0.162	-0.261	1.316	1.310	0.190
McMorris et al.[82]	Non-threshold	0.163	0.428	0.183	-0.676	1.002	0.381	0.703
Yanagisawa et al.[83]	Non-threshold	2.132	0.386	0.149	1.376	2.889	5.522	0.000
McMorris and Keen[84]	Non-threshold	-0.196	0.381	0.146	-0.943	0.552	-0.513	0.608
Pesce and Audiffren[85]	Non-threshold	1.131	0.151	0.023	0.835	1.428	7.477	0.000
McMorris et al.[55]	Non-threshold	0.784	0.290	0.084	0.214	1.353	2.697	0.007
Arcelin et al.[86]	Non-threshold	0.588	0.297	0.088	0.005	1.171	1.978	0.048
McMorris and Graydon[87]	Non-threshold	0.567	0.310	0.096	-0.041	1.175	1.826	0.068
McMorris and Graydon[40]-Exp 1	Non-threshold	0.047	0.380	0.145	0.699	0.792	0.122	0.903
McMorris and Graydon[40]-Exp 2	Non-threshold	0.280	0.383	0.145	-0.470	1.029	0.731	0.465
McMorris and Graydon[39]-Exp 1	Non-threshold	0.385	0.414	0.172	-0.427	1.197	0.929	0.353
McMorris and Graydon[39]-Exp 2	Non-threshold	0.183	0.304	0.093	-0.414	0.779	0.599	0.549
Brisswalter et al.[88]	Non-threshold	-1.527	0.348	0.121	-2.210	-0.844	-4.383	0.000
Delignières et al.[89]	Non-threshold	0.211	0.305	0.093	-0.386	0.808	0.694	0.488
Guizani et al.[90]	Non-threshold	0.652	0.392	0.154	-0.116	1.420	1.664	0.096
Fontana et al.[91]	Non-threshold	0.439	0.247	0.061	-0.045	0.923	1.778	0.075
Endo et al.[92]	Non-threshold	1.238	0.406	0.165	0.442	2.033	3.050	0.002
Nanda et al.[93]	Non-threshold	0.825	0.430	0.185	-0.018	1.668	1.918	0.055
Hogan et al.[94]	Non-threshold	0.287	0.167	0.028	-0.040	0.614	1.719	0.086
Córdova et al.[38]–Test 1	Non-threshold	0.112	0.381	0.145	-0.634	0.858	0.294	0.769
Córdova et al.[38]–Test 2	Non-threshold	0.485	0.387	0.150	-0.274	1.243	1.253	0.210
		0.541	0.108	0.012	0.330	0.753	5.013	0.000

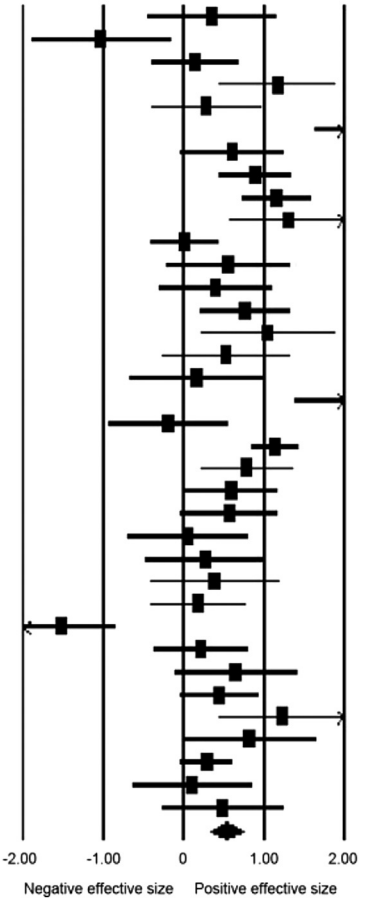

Fig. 2. Effect sizes for studies at non-threshold.

Moreover, several authors have shown relationships between brain release of NE and increased plasma ACTH and corticosterone concentrations in animals.[49,50] Similarly there is strong evidence for an interaction between HPA axis hormones and dopamine release.[51,52] Like central catecholamines, concentrations of the HPA hormones demonstrate an inverted-U effect on cognition with moderate increases inducing optimal performance.[53] So as long as the sub-threshold, long duration exercise does not result in high concentrations of HPA axis hormones, cognition should show increased speed. The point at which high concentrations are induced will be dependent on the relationship between duration and intensity, with the latter being individualized, i.e., measured as a percentage of VO_{2max} or W_{max}.

While the above may account for moderate-to-long duration, sub-threshold exercise inducing improved speed of cognition, it does not explain how speed was increased in those studies in which the duration was only 10−20 min (Table 2). A possible explanation for this comes from evidence for increased synthesis and release of catecholamines in anticipation of the upcoming exercise. Pre-exercise one often sees increases in plasma catecholamines.[54,55] McMorris et al.[55] examined cognition at rest and during exercise at 50% and 80% MAP. Results showed that pre-exercise, plasma concentrations of catecholamines significantly rose linearly from before cognitive testing immediately prior to exercise to before cognitive testing during exercise at 80% MAP despite the fact that, in all cases, the participants were simply seated on a cycle ergometer. This pre-exercise increase in plasma concentrations may well be due to the hypothalamus and brainstem initiating action of the sympathoadrenal system in readiness for undertaking the exercise. However, within the brain there is also likely to be increased catecholamine synthesis and release due to increased limbic system activity as a result of anticipation of the exercise inducing stress. Once exercise begins this centrally-induced increased synthesis and release of dopamine and NE is likely to increase. This is in line with Mason's[56,57] argument that stress is induced when the individual perceives the situation as being unpredictable and/or one in which he/she is not in control. Psychological stress of this kind has been shown to induce the release of catecholamines in plasma in humans[58] and in the brain in animals.[59] If there are increases in catecholamine concentrations due to central release as a result of perception of stress, this could result in improved speed of cognitive functioning.

5.2. Limitations of study

Due to the relatively small number of studies that met the criteria for inclusion in this review, we were unable to examine moderating variables such as whether cognitive testing took place during or after exercise, whether the order of conditions were randomized or counterbalanced, and whether testing took place on the same or different days. The inability to examine the differing effects of exercise on working memory, particularly central executive tasks, versus attention/perception tasks is a major limitation as these have been show to be affected differently by exercise at different intensities.[4,5]

5.3. Future directions

From a theoretical perspective it would appear that T_{CATS} is the logical intensity to ensure optimal performance. In order to examine this empirically, we need to compare participants' performance at sub-threshold, threshold, and supra-threshold intensities. The question of the interaction between the threshold and task type could also be examined in this way. More studies in which plasma catecholamines concentrations are measured regardless of intensity, would also help us to build up a better understanding of the interaction between acute exercise, catecholamines, and cognition. Studies that allow for mediation analyses to examine how acute intensity predicts catecholamine responses which in turn explain changes in behavioral measures of cognition would undoubtedly be useful in examining the interaction between acute exercise, catecholamines, and cognition, but these require much larger sample sizes than most previous researchers have utilized.

6. Conclusion

The results of this study failed to fully support the hypothesis that there is a physiological/biochemical trigger point which induces increased speed of cognition. It would appear that exercise of moderate intensity (40%−79% VO_{2max} or equivalent) can induce increased speed of cognition but that this is more likely to be due to physiological and biochemical changes peripherally plus increased central neurochemical activity, resulting from exercise-induced perceptions of stress, rather than simply being due to peripheral catecholamine concentrations. However, more research is required, particularly research which will allow for the measurement of the effects of possible moderators such as fitness, task complexity, exercise duration, and perception of stress.

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