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Title: Does acute exercise affect the performance of whole-body, psychomotor skills in an inverted-U fashion? A meta-analytic investigation.

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Keywords: arousal; fatigue; perception; action; catecholamines; prefrontal cortex

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Abstract: The primary purpose of this study was to examine, using meta-analytical measures, whether research into the performance of whole-body, psychomotor tasks following moderate and heavy exercise demonstrates an inverted-U effect. A secondary purpose was to compare the effects of acute exercise on tasks requiring static maintenance of posture versus dynamic, ballistic skills. Moderate intensity exercise was determined as being between 40% and 79% maximum power output (WMAX) or equivalent, while  $\geq$ 80% WMAX was considered to be heavy. There was a significant difference (Zdiff = 4.29, p = 0.001, R2 = 0.42) between the mean effect size for moderate intensity exercise (g = 0.15) and that for heavy exercise size (g = -0.86). These data suggest a catastrophe effect during heavy exercises. Mean effect size for static tasks (g = -1.24) was significantly different (Zdiff = 3.24, p = 0.001, R2 = 0.90) to those for dynamic/ballistic tasks (g = -0.30). The result for the static versus dynamic tasks moderating variables point to perception being more of an issue than peripheral fatigue for maintenance of static posture. The difference between this result and those found in meta-analyses examining the effects of acute exercise on cognition show that, when perception and action are combined, the complexity of the interaction induces different effects to when cognition is detached from motor performance.

Dear Professor,

I respectfully submit this manuscript for publication in Physiology & Behavior. It has not been published elsewhere, nor is it under consideration for publication elsewhere. There are no conflicts of interest. The main text consists of 4793 words, the Abstract has 218 words. There are four figures and one table. All co-authors are in agreement with the decision to submit to Physiology & Behavior. Thank you for taking the time to process this submission. Yours sincerely,

Terry McMorris

- There was an overall negative effect of exercise on skilled performance
- Moderate intensity exercise had no significant effect on performance
- Heavy exercise disrupted performance
- Static balance tasks were disrupted more than ballistic skills

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6 7	2	inverted-U fashion? A meta-analytic investigation.
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The primary purpose of this study was to examine, using meta-analytical measures, whether research into the performance of whole-body, psychomotor tasks following moderate and heavy exercise demonstrates an inverted-U effect. A secondary purpose was to compare the effects of acute exercise on tasks requiring static maintenance of posture versus dynamic, ballistic skills. Moderate intensity exercise was determined as being between 40% and 79% maximum power output ( $W_{MAX}$ ) or equivalent, while  $\geq 80\% W_{MAX}$  was considered to be heavy. There was a significant difference ( $Z_{diff} = 4.29$ , p = 0.001, R<sup>2</sup> = 0.42) between the mean effect size for moderate intensity exercise (g = 0.15) and that for heavy exercise size (g = -0.86). These data suggest a catastrophe effect during heavy exercise. Mean effect size for static tasks (g = -1.24) was significantly different ( $Z_{diff}$  = 3.24, p = 0.001,  $R^2$  = 0.90) to those for dynamic/ballistic tasks (g = -0.30). The result for the static versus dynamic tasks moderating variables point to perception being more of an issue than peripheral fatigue for maintenance of static posture. The difference between this result and those found in meta-analyses examining the effects of acute exercise on cognition show that, when perception and action are combined, the complexity of the interaction induces different effects to when cognition is detached from motor performance.

- 40 Keywords: arousal; fatigue; perception; action; catecholamines; prefrontal cortex

#### **1. Introduction**

Yerkes and Dodson's [1] inverted-U theory, concerning the effect of arousal on performance, and theories developed from this early work [2-4] have generally been used as the rationales for studies examining the effect of acute exercise on cognition (e. g., [5-7]). It has normally been hypothesized that moderate intensity exercise equates to moderate levels of arousal and so optimal performance is expected, while heavy exercise equates to over-arousal and so performance returns to the same level as that at rest. A recent meta-analysis [8] supported this hypothesis. The primary purpose of the present study was to examine, using meta-analytical measures, whether research into the performance of whole-body, psychomotor tasks following moderate and heavy exercise also demonstrates an inverted-U effect. Meta-analytic methods were undertaken as they facilitate the use of a larger sample size than one normally finds in research on this topic. Moreover, the emphasis on effect sizes rather than probability allows for a better evaluation of those studies where failure to show a significant effect was due to sample size possibly resulting in Type II errors. Whole-body, psychomotor skills require integrated control by the Central and Peripheral Nervous Systems (PNS). The decision to act is made by the higher centers of the brain, particularly the prefrontal cortex, and action is initiated by the premotor cortex and/or supplementary motor area, with the former being primarily concerned with movement in response to external events while the latter mainly controls voluntary movement, although both are active during any type of movement. The information is passed downwards to the PNS via several Central Nervous System (CNS) regions including the basal ganglia, brainstem, cerebellum and spinal cord. Information from the CNS is transmitted by efferent nerves to motor units in the musculature. These neurons activate the musculature. Once the action begins information from the PNS, about the movement, is fedback to the brain by

afferent neurons, situated in the muscles, joints and spinal cord. The spinal cord itself can make very fast (~ 30 ms), but very limited alterations to the movement, using the process of  $\alpha$ - $\gamma$ coactivation [9]. Feedback to the cerebellum, the so-called long loop feedback, is greater in scope than the  $\alpha$ - $\gamma$ coactivation process but takes ~ 80 ms to be activated [10]. The most important feedback is to the sensory regions of the brain, particularly the visual and somatosensory cortices, and, in some skills, the auditory cortex. The prefrontal cortex and the sensory association areas receive information from the sensory cortices and organize and interpret this information. These higher centers of the brain can initiate large alterations to the movement but take time (> 400 ms) [11]. This CNS-PNS interaction ensures that the

movements are coordinated and smooth, and that motor unit recruitment allows for theproduction of the required power.

The processes outlined above can be affected by a number of stressors, including acute exercise. The most obvious effect of acute exercise on the performance of such skills is physiological, although the precise nature of these physiological effects will vary primarily as a function of the intensity (e.g. moderate vs. heavy) but may also be influenced by the duration; the environment under which the exercise is conducted; the fitness level of the exerciser and the elapsed time between the exercise and performance of the criterion task (see [12] for a review). Broadly speaking, relevant physiological effects could incorporate central processes related to alterations in the intrinsic motoneuron properties, sensory feedback, or descending drive [13] and peripheral processes occurring distal to the neuromuscular junction, including those related to sarcoplasmic reticulum calcium release and decreased myofibrilar force production [14]. More precisely, authors have previously emphasized the role of physiological factors related to the level of nervous system activation [6, 15]; the efficiency of the peripheral motor processes (i.e. better synchronisation of the motor units discharge); peripheral sensorial processes [16-17]; the involvement of different metabolic

92 systems [18] and associated effects related to metabolic acidosis, or the accumulation of93 metabolic waste products and humoral changes [19].

The physiological changes induced by acute exercise are accompanied by biochemical changes peripherally and centrally. 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. If exercise increases in intensity to a moderate level, there is also release of epinephrine and, to a lesser extent norepinephrine, into the blood from the adrenal medulla. As exercise intensity increases further to a level which we could describe as heavy, there are larger increases in plasma norepinephrine and epinephrine concentrations. Peripherally, norepinephrine and epinephrine aid lipolysis, stimulate receptors in muscle and activate receptors in the pancreas to suppress insulin release. Epinephrine plays a major role in glycogenolysis and control of the cardiovascular system by activating receptors responsible for increasing heart rate and contractile force (see [20] for a review). Although catecholamines do not readily cross the blood brain barrier, rodent studies (see [21] for a review) have demonstrated significant increases in brain concentrations of dopamine and norepinephrine following acute exercise. This is most likely due to the fact that peripherally circulating epinephrine and norepinephrine activate  $\beta$ -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. Noradrenergic cells in the NTS, which project into the locus coeruleus, stimulate norepinephrine synthesis and release to other parts of the brain [22-23]. This may also affect brain dopamine concentrations, as Devoto et al. [24] showed that electrical stimulation of the rat locus coeruleus resulted in increased brain concentrations of dopamine and one of its metabolites, 3,4-dihydroxyphenylacetic acid. During moderate intensity exercise, there are moderate

increases in concentrations of brain catecholamines, which activate the prefrontal cortex, sensory cortices and their association areas. These increases lead to improved sensation and

perception by increasing the signal to 'noise' ratio within the brain. Heavy exercise, however,
leads to even greater increases in brain concentrations of catecholamines, which disrupts the
signal to 'noise' ratio, hence inhibiting sensation and perception [25-26].

Catecholamines are not the only neurochemicals, activated during exercise, which may affect sensation and perception. There are several but the hypothalamic-pituitary-adrenal cortex (HPA) axis hormones are probably the most important. Peripherally, during exercise, the HPA axis hormone cortisol plays major roles in glucose production from proteins, the facilitation of fat metabolism and muscle function, and the maintenance of blood pressure [27]. However, this appears to only occur when exercise is heavy [28]. Cortisol readily crosses the blood brain barrier, so peripheral increases in concentrations will lead to central increases. Moreover, the synthesis and release of cortisol by the HPA axis is initiated by the synthesis and release of the protein corticotrophin releasing factor (CRF) and the hormone adrenocorticotrophin hormone (ACTH) [29]. Given that CRF is released in the brain and ACTH in the anterior pituitary, which lies within the CNS, it is not surprising to find that rodent studies have demonstrated acute exercise-induced increases in brain concentrations of the HPA axis neurochemicals [30-32]. These neurochemicals interact with catecholamines in the brain, resulting in increased synthesis and release of dopamine and norepinephrine, which should inhibit sensation and perception during heavy exercise [33-35].

137 The theory outlined above suggests that moderate, acute exercise will facilitate the 138 performance of whole-body, psychomotor skills, due to moderate increases in concentrations 139 of the brain neurotransmitters dopamine and norepinephrine inducing improved perceptual 140 performance. Moreover, physiological mechanisms that could positively influence motor 141 processes through mechanisms, including effects on nervous system activation, motor unit coordination [13] or improved contractile function [14], will also be facilitated. During heavy exercise, changes related to metabolic acidosis and fatigue may elicit different physiological changes which have the potential to impair the motor process at a variety of central and peripheral sites, although the influence of acidosis on contractile function is controversial [36]. Also excessive brain concentrations of catecholamines should inhibit sensation and perception, meaning that we can hypothesize that there will be a significant difference between effect sizes during moderate and heavy exercise. In humans, when the stressor is psychological and the task cognitive, one tends to find that these changes in brain concentrations of catecholamines result in an inverted-U effect. However, we assert that it is possible that, when the task is physical, a combination of the central and peripheral changes might result in heavy exercise inducing poorer performance than at rest, thus demonstrating an inverted-J effect. A secondary purpose of this study was to compare the effects of acute exercise on

tasks requiring static maintenance of posture (e. g. static balance and shooting) and dynamic, ballistic skills (e. g. most sports skills). While both require the integration of the CNS and PNS, the nature of the movements and the integration of perceptual information differ. Maintenance of posture has been shown to be heavily dependent on central perception of balance, which appears to be negatively affected by exercise [37-39], while the perceptual and decision making aspects of many dynamic skills have been shown not to be affected even by heavy exercise and indeed in some cases are facilitated [7, 40]. However, the physiological demands of dynamic skills may result in deterioration in performance of such skills. 

2. Materials and methods

A literature search using the computer data bases PsycArticles, PsycINFO, Pubmed, SPORTSDiscus and Web of Knowledge was undertaken. Key words used in the searches were combinations of "acute", "exercise", psychomotor performance", "psychomotor skills", "physical activity", "fatigue" and the actual names of a large variety of whole-body, psychomotor skills. In addition, reference lists from empirical reports and reviews were examined. Studies were included if they were performed on healthy individuals and repeated measures, within-subject designs were used. In studies using pharmacological or nutritional treatments, the control or placebo groups' data were included in the meta-analyses but not the experimental groups.

### 177 2.1. Definitions of moderate and heavy exercise.

When exercise was aerobic, Borer's [20] classifications of moderate and heavy exercise formed the basis of our definitions, the same as those used by McMorris and colleagues [8, 41]. Moderate intensity exercise was determined as being between 40% and 79% maximum power output ( $W_{MAX}$ ) or equivalent, while  $\geq 80\% W_{MAX}$  was considered to be heavy. If  $W_{MAX}$  values were not presented but percent volume of maximum oxygen uptake ( $V = O_{2MAX}$ ) or percent maximum heart rate were given, the conversion formulae of Arts and Kuipers [42] were applied. For other indicators of intensity, e.g. percent heart rate reserve, percent maximum aerobic power, percent ventilatory threshold and percent lactate threshold power, the exercise physiology and exercise endocrinology literatures were examined to ascertain whether or not the intensity would be below, within or above the 40-79%  $W_{\text{MAX}}$  limits. In those studies using isotonic or isometric exercise, contractions  $\geq 80\%$ of the participants' maximal number of contractions were considered to be heavy, while contractions < 80% but > 40% maximum were deemed moderate. Where exercise was intermittent anaerobic and aerobic, duration and time working at each of the intensities were

used to determine whether or not the overall intensity could be classed as moderate or heavy.
Where exercise was to voluntary exhaustion or until the individual could not maintain the
required intensity, it was deemed heavy.

*2.3. Data analyses* 

Where means and SDs were available, effect sizes were calculated using the 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 and especially the 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  $\tau^2$ , which is a measure of absolute variance whereas Q is a measure of relative variance [43]. Orwin's [44] Fail-safe N was calculated when the mean effect size g was  $\geq 0.20$ . Where sub-group analyses were undertaken, effect sizes for each group were compared using a Z-test on the differences with a random-effects model, with separate estimates of  $\tau^2$  for each sub-group. The proportion of variance explained by the moderator variable,  $R^2$ , was calculated [43]. Most 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 intensity per study was calculated, with one exception ([45] see section 3). 3. Results The literature reviewed showed that there were 89 articles which examined the effect of acute exercise on the performance of whole-body, psychomotor skills but only 28 which

217	met the criteria for inclusion and provided sufficient statistical information. However, one
218	study [45] provided data for novice and expert performers separately and so was treated as
219	two separate studies. This meant that there were 23 studies in which effect sizes were
220	calculated for one exercise intensity only and six where two intensities were included. In
221	total, there were 35 effect sizes and 570 participants. The types of tasks and exercise
222	intensities used in each study can be seen in Table 1.
223	Insert Table 1 about here
224	An initial overall analysis, including both the moderate and heavy exercise dependent
225	variables, is necessary before comparisons can be undertaken [43]. This showed that effect
226	sizes were heterogeneous Q(34) = 155.68 (p < 0.001), $\tau^2$ = 0.46. The mean effect size was
227	significant, $g = -0.55$ (Z = 4.14, $p < 0.001$ ), variance 0.018, SE = 0.13, and 95% confidence
228	interval (CI) -0.81 to -0.29. The fail-safe N was 61. Twenty-six effect sizes were negative and
229	nine positive. Sub-group analyses for moderate and heavy exercise showed a significant
230	difference between the two variables (Mean <sub>diff</sub> = 1.03, SE = 0.24, $Z_{diff}$ = 4.29, p = 0.001, R <sup>2</sup> =
231	0.42). Mean effect size for moderate intensity exercise was non-significant ( $g = 0.15$ , SE =
232	0.12), while heavy exercise demonstrated a significant mean effect size ( $g = -0.86$ , $Z = 5.85$ ,
233	p < 0.001, variance = 0.02, SE = 0.15, CI = -1.14 to -0.57).
234	Sub-group analyses for static and dynamic skills demonstrated a significant difference
235	between the two variables (Mean <sub>diff</sub> = 0.94, SE = 0.29, $Z_{diff}$ = 3.24, p = 0.001, R <sup>2</sup> = 0.90).
236	Mean effect size for static tasks was significant (g = -1.24, Z = 4.84, p < 0.001, variance =
237	0.07, SE = 0.26, CI = -1.75 to -0.74), as was the mean effect size for dynamic skills (g = -
238	0.30, Z = 2.33, p < 0.02, variance = 0.02, SE = 0.13, CI = -0.55 to -0.05). It was decided, a
239	posteriori, to examine the effect of using counterbalanced/randomized designs compared to a
240	pre-exercise/post-exercise design. Sub-group analyses showed no significant differences.

244 4. Discussion

The overall analysis shows a moderate to high effect size. That it was negative is not too surprising, given that there were more studies measuring the effects of heavy exercise than moderate. Nevertheless, a regression towards zero was expected, as we thought that moderate exercise would induce positive effect sizes while heavy would result in negative effect sizes. This was not demonstrated as 45.45% of the of the moderate intensity results showed negative effects. This result is very different to those found in meta-analyses examining the effect of acute exercise on cognitive skills, including perception, when the skill is carried out either during exercise or immediately following cessation of the exercise. In those studies, the overall analyses with both moderate and heavy exercise included, have tended to show small to moderate, but significant effects, mostly positive [8, 46-48] but one negative [49].

258 4.1. Moderate intensity exercise effects

The effect size for moderate exercise was positive but small and non-significant. We expected a high effect size ( $g \ge 0.70$ ) because of the strong theoretical rationale. One would expect the increase in body temperature during moderate intensity exercise to induce increased speed of nerve transmission [50], which would aid coordination and power production. Moreover, when brain catecholamines concentrations are increased to a moderate level, there is increased firing of  $\alpha_{2A}$ -adrenoreceptors by norepinephrine [51], which increases the strength of the neural signal, and D1 dopaminergic receptors by dopamine [52], which

dampens the 'noise' by inhibiting firing to non-preferred stimuli, thus improving the signal to
'noise' ratio [25-27]. This should strongly improve perceptual performance by optimizing
activity in the reticular formation, which controls attention, alertness and vigilance, and the
prefrontal cortex, which is responsible for integration and interpretation of information from
the sensory cortices and their association areas.

Before attempting to look at possible physiological and neuroscientific reasons for these results, we must examine some possible methodological issues. As the sample size (k =11) was small from the point of view of number of studies, the possibility of a lack of power resulting in a Type II error has to be taken into account. According to Clarke-Carter [53], to attain a power of 0.80, with k = 11, we would need to elicit an effect size of g = 0.75, very close to our expected g = 0.70. This may account for the failure of g = 0.15 to reach significance but does not explain why we failed to show the effect size we expected or at least one near to it. The possibility that study designs failed to properly control exercise intensity also needs to be addressed. All exercise intensities classified as moderate met the criteria set out in 2.1. The only questionable issue might be the time spent exercising in the McRae et al. [54] study (2 hours), which could have resulted in increased brain cortisol concentrations as well as increased dopamine and norepinephrine concentrations. Moreover, three studies [54-56) failed to take into account individual differences in fitness. Given the mean effect sizes for these studies, it does not appear that this has been a contributor, certainly not a major contributor, to our results.

However, the failure of all but two studies [57-58] to take into account individuals'
lactate and catecholamines thresholds may have affected results. When we plot plasma
concentrations of epinephrine and norepinephrine against exercise intensity, concentrations
rise exponentially [59-60]. Green et al. termed the points at which there is a significant rise in
concentrations, the epinephrine threshold and the norepinephrine threshold. Although the two

291	thresholds generally show moderate to high correlations, the correlations differ between
292	individuals [61]. It would appear that exercise intensity needs to be moderate before the
293	thresholds are reached but there are large inter-individual variations [62]. It is generally
294	thought that, for aerobic exercise, intensity needs to be ~ 75% V $O_{2MAX}$ [61], which
295	according to Arts and Kuipers [42] equates to ~ 65% $\dot{W}_{MAX}$ . Moreover, blood lactate
296	concentrations follow a similar exponential profile and the lactate threshold shows moderate
297	to high correlations with the catecholamines thresholds [6, 61, 63]. Chmura, Nazar and
298	Kaciuba-Uścilko [6] argued that it is at or immediately following the catecholamines
299	thresholds that a significant improvement in cognitive function will be induced due to
300	increased brain catecholamines concentrations. This makes sense, as increased concentrations
301	of circulating epinephrine and norepinephrine will activate the $\beta$ -adrenoreceptors on the
302	vagus nerve, thus initiating the action of the vagus/NTS pathway and increased synthesis and
303	release of norepinephrine in the locus coeruleus. Improved cognitive performance at or
304	following the catecholamines thresholds [6-7] and the lactate threshold [64-65] has been
305	demonstrated. Also, improved cognition has been shown at the ventilatory threshold [66-70],
306	the point at which ventilatory carbon dioxide shows a greater increase than ventilatory
307	oxygen and which occurs about the same time as the lactate threshold [71].
308	Participants exercising below their catecholamines thresholds would probably not
309	induce increased brain catecholamines concentrations and hence not show improved
310	performance. However, individuals exercising above their thresholds might synthesize and
311	release too much, thus negatively affecting performance. This might account for the
312	equivocal nature of the results and also points to the need for those wishing to use warm-up
313	exercise to aid whole-body, psychomotor performance to individualize the exercise intensity.
314	Another possible reason for the failure to demonstrate positive and significant results could
315	be that, if exercise is above the individual's threshold, CNS and PNS integration are

compromised due to increased blood and muscle lactate concentrations, and changes in the balance between ventilatory carbon dioxide and oxygen having detrimental effects on the motor aspects of the psychomotor task.

These results suggest that the CNS-PNS interaction during the performance of psychomotor skills, following moderate intensity exercise, may be very complex. While the catecholamines thresholds may be ideal for brain activation, especially in the prefrontal cortex, this intensity may have negative effects on the physiological aspects. Indeed it could be that different skills require different intensities to induce optimal performance. A great deal more research is necessary. Moreover, research should include more physiological and biochemical measurements than the research at present in the literature.

# 327 4.2. Heavy intensity exercise effects

Results for the effects of heavy exercise were as expected with a high effect size being demonstrated. Neurophysiologically, research has shown that reduced excitations of motoneurons resulting from afferent feedback from muscle spindles [72], reduced impulse frequency to muscle fibers at the neuromuscular junction [73] and failures in the calcium release process [74] significantly affect coordination and power. Metabolically, decreased adenosine triphosphate supply [75], decreased glycogen concentrations [76], decreased pH and increased concentrations of inorganic phosphate [77] all have inhibitory affects on motor control. From a neurochemical perspective, heavy exercise induces very large increases in brain concentrations of catecholamines. This, in turn, leads to the excess norepinephrine activating the lower affinity  $\alpha 1$ - and  $\beta$ -adrenoreceptors [51].  $\alpha 1$ -adrenoreceptors can result in reduced neuronal firing in the prefrontal cortex by phosphatidylinositol-protein kinase C intracellular signaling pathway activation. Excessive stimulation of D1 receptors and  $\beta$ -adrenoreceptors can induce 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 (see [25-26]). During high levels of stress, this is probably exacerbated by stimulation of D2 receptors [25]. Although, stimulation of  $\alpha$ 1- and  $\beta$ -adrenoreceptors can improve the signal to 'noise' ratio in the sensory cortices [78-80] and can aid some prefrontal cortex activities, overall it has a negative effect on prefrontal cortex activity [25-26]. This is important because the prefrontal cortex is responsible for the integration and coordination of perceptual information from a variety of sensory regions of the brain [81]. Moreover, as well as integrating and coordinating sensory and perceptual feedback, the prefrontal cortex, particularly the right inferior prefrontal cortex, plays a major role in inhibition of inappropriate motor responses [82], which have a negative effect on the performance of psychomotor skills. As we saw in section 1, heavy exercise also initiates the release of the HPA axis hormones, which exacerbate the negative effects of the catecholamines. These neurochemicals interact with catecholamines in the brain to affect perception and cognition. In the locus coeruleus, CRF neurons innervate noradrenergic neurons and norepinephrine is released [83-84]. Similarly there is strong evidence for an interaction between corticosteroid concentrations and dopamine release [85-88]. Thus increased brain concentrations of CRF, ACTH and cortisol during heavy exercise add to the negative effects of catecholamines. 4.3 Effects on static versus dynamic/ballistic psychomotor skills The results of the sub-groups analyses show that we were correct to expect differences but the proportion of variance was far greater than we had anticipated. We expected only a relatively low coefficient, e. g.  $R^2 \simeq 0.40$ , as both sets of skills require high levels of perpetual-action coupling. That the effect for the static skills was negative was as

365 expected but that it was so high was surprising. These skills recruit muscles in the vicinity of

the knees, ankles, calves, toes and hips but the range of movement and power required are comparatively small [89-91]. Centrally they require a large input from the dorsolateral prefrontal cortex to integrate information from the visual and somatosensory cortices, cerebellum and vestibular apparatus. Given that the dorsolateral prefrontal cortex is especially susceptible to disruption by excess catecholamines [25-26], inhibition of performance is not surprising. Furthermore, several researchers have argued that exercise affects proprioception more than the motor aspects of balance [37, 39] and it has been shown that the attentional demands of balance actually increase following heavy exercise [37]. The dynamic, ballistic skills also require central integration of perceptual information but even heavy exercise has been shown to have only a limited negative effect on the perceptual and decision-making aspects of such skills [8, 40, 92]. Moreover, many of the skills utilized in the research covered by this analysis may well have been autonomic to the participants. This is especially so given that many of the skills were sports skills and the participants were often physical education and/or sports science majors. Autonomic skills may well be less negatively affected by stress, even physiological stress. This has been shown for well-learned cognitive skills and implicitly-learned motor skills [92-93]. However, the peripheral physiological adaptations to exercise probably have a negative effect on coordination and power, which results in a small but significant negative effect. If automaticity is a key moderator, it would appear that overlearning could help to lessen the problem of performing following heavy exercise. 4.4. Use of counterbalancing/randomization of testing

388 Observation of the raw data led us, a posteriori, to examine the use of 389 counterbalancing/randomization of testing as opposed to the use of pre-exercise testing 390 followed by post-exercise testing. Research methods texts recommend counterbalancing or

randomization but many of the studies included in the analyses utilized a pre-exercise followed by post-exercise protocol. The possibility of a learning or habituation effect is obvious. Therefore, we decided to compare mean effect sizes for studies using counterbalanced/randomized designs with those using pre-exercise followed by post-exercise testing. That there was no significant difference between studies using counterbalance/randomization and those using a pre-exercise testing followed by post-exercise testing protocol was a little surprising. McMorris and Hale [8], examining the effect of acute exercise on cognition, showed that randomized/counterbalanced designs elicited higher effect sizes than pre-exercise followed by post-exercise testing. They claimed that testing pre-exercise meant that the individual's dopamine and norepinephrine, and possibly cortisol, concentrations would show an increase pre-exercise due to anticipation of the exercise to come, a phenomenon which has been known for some time [94] and demonstrated recently [95]. This could lead to pre-exercise cognitive performance being better than that at a real baseline. With psychomotor skills one might expect the same pre-exercise increase due to anticipation but there is likely to be a fall in brain catecholamines concentrations once the participants begin the pre-test. Falls in peripheral catecholamines concentrations have been shown when individuals begin to perform a skill compared to pre-performance concentrations [96]. This is probably due to the perception of the stress being greater than the actual stress [94]. This would negate the pre-exercise levels affecting the following rest performance. 5. Conclusion The results of this study failed to fully support either an inverted-U or an inverted-J effect of acute exercise on the performance of whole-body, psychomotor skills. Moderate

415 intensity exercise demonstrated no significant effect, while heavy exercise showed a negative

effect. That moderate intensity exercise failed to induce a significant improvement from rest questions the use of a moderate intensity warm-up for improving performance, a practice that is common particularly in sport. Moreover, the difference between this result and those found in meta-analyses examining the effects of acute exercise on cognition [8, 41, 46-49] show that, when perception and action are combined, the complexity of the interaction induces different effects to when cognition is detached from motor performance. The same appears to be the case with heavy exercise, following which the neurochemical and physiological stress appear to combine to induce a detrimental effect compared to not only moderate intensity

dynamic tasks moderating variables possibly point to perception being more of an issue than

exercise but also compared to at rest, baseline measures. The result for the static versus

peripheral muscular fatigue for maintenance of static posture.

There are several issues that future research needs to examine. Firstly, a lot more research is required and studies should include physiological and neurochemical measures. Such measures would allow for a better knowledge of the amount of physiological stress placed on the participants. Moreover, individual differences in participants' fitness levels need to be taken onto account in the experimental designs. Comparison of tasks involving greater and less prefrontal cortex activation would allow for the investigation of whether the problems were mainly central or peripheral or equally both.

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729 Figure 1. Forest plot for all studies.

Figure captions

- 730 Note: H heavy exercise; M moderate intensity exercise; D dynamic/ballistic skills; S static
- 731 balance; CR counterbalanced or random design; SO (same order) pre-exercise test followed
- 732 by post-exercise test.

- 733 Figure 2. Forest plot for heavy exercise studies.
- 734 Note: H heavy exercise; D dynamic/ballistic skills; S static balance; CR counterbalanced or
- random design; SO (same order) pre-exercise test followed by post-exercise test.
- 736 Figure 3. Forest plot for dynamic/ballistic skills studies.
- 737 Note: H heavy exercise; M moderate intensity exercise; D dynamic/ballistic skills; CR
- 738 counterbalanced or random design; SO (same order) pre-exercise test followed by post-

739 exercise test.

- 740 Figure 4. Forest plot for static balance studies.
- 741 Note: H heavy exercise; M moderate intensity exercise; S static balance; CR counterbalanced
- 742 or random design; SO (same order) pre-exercise test followed by post-exercise test.

Authors	N	M/F	Mean Age (SD) years	Exercise type and intensity	RD	Test	dv	Effect size (g)
Ali et al. [97]	16	М	21.3 (3)	LIST [98]	P-P	LSPT, LSST [99]	Time, Acc	-0.30
Anshel & Novak [101]	44	М	20.10 (1.10)	cycling @ 45, 60, 75% V□O <sub>2MAX</sub> , fatigue	P-P	shot putt	distance	0.70
Bottoms et al. [102]	9	М	24.8 (6.76)	b'ton circuit to fatigue	P-P	b'ton serve [100], WBCRT	Acc	-0.96
Bullock et al. [55]	42	М	18.5 (3.5)	LIST [98]	P-P	soccer passing, agility	Acc, time	0.33
Davey et al. [103]	18	9 M 9 F	21.2 (0.75)	tennis simulation to fatigue	P-P	LTST [103]	Acc	-0.27
Dickin & Doan [104]	16	M 9 F 7	22.35 (1.70)	isometric contractions to fatigue	P-P	posture	amplitude displacement	-0.42
Evans et al. [105]	12	M 9 F 3	21.45 (3.40)	hand-crank to fatigue, obstacle course to fatigue	P-P	shooting	Acc	-2.10
Gabbett [106]	8	М	23.00 (1.20)	Rugby simulation (HR > 180 bpm)	P-P	Rugby tackle	Time, Acc	-0.54
Groslambert et al. [107]	10	М	18.50 (1.00)	ski 85%, 100% HR <sub>MAX</sub>	P-P	shooting	Acc	-2.06

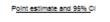
Table 1. Participants' demographics, exercise intensities, psychomotor tests and type of research design (RD).

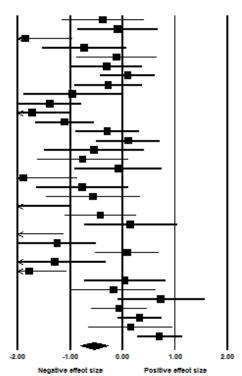
Impellizzieri et al. [108]	26	Μ	17.8 (0.6)	HI soccer simulation (HR > 180 bpm) [109]	P-P	LSPT [99]	Time	-1.39
Johnston et al. [110]	20	M 12 F 8	29 (N/R)	isometric contractions to fatigue	P-P	balance	Time in balance	-1.79
Lidor et al. [56]	26	М	16.40 (0.82)	v'ball simulation (HR ~ 150 bpm)	P-P	v'ball serve	Acc	-0.07
Lyons et al. [111]	20	Μ	22.90 (5.30)	70%, 90% max squats	С	LSPT [99]	Time	0.11 (M) -0.29 (H)
Lyons et al. [45] experts	10	М	22.50 (0.41)	70%, 90% max squats	С	b'ball pass test	Acc + speed	-0.09 (M) -0.76 (H)
Lyons et al. [45] novices	10	М	23.30 (1.05)	70%, 90% max squats	С	b'ball pass test	Acc + speed	-0.77 (M) -1.89
McGregor et al. [112]	9	Μ	20.20 (0.40)	LIST [98]	P-P	soccer dribble	Time	0.15
McMorris et al. [113]	11	Μ	N/R	cycling @ 70%, 100% W□ <sub>MAX</sub>	R	soccer passing	CE, VE, No. passes, total score	0.73 (M) -0.17 (H)
McMorris et al. [57]	12	Μ	20 (2)	cycling @ $T_E, W \square_{MAX}$	С	soccer passing	AE, CE, VE	-0.11 (M) -0.74 (H)
McMorris et al. [114]	12	М	24.24 (3.10)	cycling @ 70%, 100% W□ <sub>MAX</sub>	С	slalom run	Time	-0.10 (M) -0.38 (H)
McMorris et al. [58]	12	М	21.04 (2.12)	cycling @ T <sub>LA</sub>	R	soccer passing	CE, VE, MT	0.05
McMorris & Rayment [115]	13	М	20.5 (2.0)	1 x yo-yo shuttle (HR ~ 150 bpm) 3 x shuttle (30 s rest)	R	soccer passing	AE, VE	-1.85

				(HR > 180 bpm)				
McRae & Galloway [54]	11	M 15 F 7	22.00 (7.30)	matchplay (HR ~ 130 bpm)	P-P	tennis serve, ground strokes	Acc	0.15
Sarshin et al. [116]	20	N/R	21.4 (1.63)	balance task to fatigue [110]	P-P	balance	Acc	-1.73
Sunderland & Nevill [117]	9	F	21.70 (0.40)	LIST [98]	P-P	field hockey skills drill	Time	-0.56
Tharion et al. [118]	27	М	29 (4)	14.7 km run with backpack @ 6.5 km/hr*	P-P	shooting	Acc	-1.10
Vuillerme et al. [37]	9	М	22.00 (3.10)	isometric contractions to fatigue	P-P	balance	Acc	-1.29
Wickstrom et al. [119]	20	M 8 F 12	22.00 (1.75)	isometric contractions to fatigue, agility drill to fatigue	P-P	balance	Acc	0.09
Yaggie & Armstrong [91]	16	М	24 (3)	Wingate Test [120] x 2*	P-P	balance	Acc	-1.25
Young et al. [121]	27	М	N/R	2 x 3 mins sprint (3 mins rest) HR 174 (9)	P-P	kicking accuracy ARF	Acc	0.10

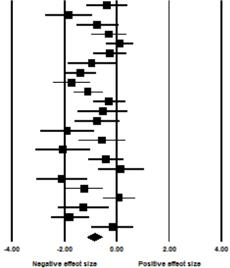
Note: \* no physiological measures taken but the nature of the tasks are such that they constitute heavy exercise: RD = research design: M = male: F = female: dv = dependent variable: LIST = Loughborough intermittent shuttle test: P-P = pre-exercise followed by postexercise: LSPT = Loughborough soccer passing test: LSST = Louhgboirough soccer shooting test: Acc = accuracy:  $V \square O_{2MAX}$  = maximum volume of oxygen uptake: b'ton = badminton: WBCRT = whole-body choice reaction time: LTST = : HR = heart rate: bpm = beats per minute:  $HR_{MAX} =$  maximum heart rate: HI = high intensity: N/R = not recorded: v'ball = volleyball: C = counterbalanced: b'ball = basketball: CE = constant error:, VE = variable error: No. = number of:  $T_E$  = epinephrine threshold:  $W\Box_{MAX}$  = maximum power output:  $T_{LA}$  = lactate threshold: R = randomized: MT = movement time: ARF = Australian Rules football

Study name	Subgroup within st								
			Point estimate	Standard error	Variance	Lower limit		Z-Value	p-Value
McMorris et al. (114)	H D CR	Heavy	-0.377	0.399	0.159	-1.159	0.404	-0.946	0.344
McMorris et al. [114]	MDCR	Moderate	-0.097	0.395	0.156	-0.871	0.678	-0.245	0.807
McMorris & Rayment [ 115	H D CR	Heavy	-1.853	0.459	0.211	-2.752	-0.953	-4.036	0.000
McMorris et al. (57)	HDCR	Heavy	-0.735	0.409	0.167	-1.537	0.066	-1.798	0.072
McMorris et al. [57]	MDCR	Moderate	-0.116	0.395	0.156	-0.891	0.659	-0.294	0.769
All et al. 1971	H D SO	Heavy	-0.303	0.347	0.120	-0.983	0.378	-0.872	0.383
Young et al. [121]	H D SO	Heavy	0.099	0.268	0.072	-0.428	0.625	0.367	0.713
Davey et al. [103]	H D SO	Heavy	-0.274	0.328	0.107	-0.916	0.368	-0.836	0.403
Bottoms et al. [102]	H D SO	Heavy	-0.956	0.478	0.228	-1.893	-0.019	-2.000	0.046
Impellizzieri et al. (108)	H D SO	Heavy	-1.389	0.305	0.093	-1.988	-0.791	-4.550	0.000
Sarshin et al. [116]	H S SO	Heavy	-1.726	0.365	0.133	-2.442	-1.010	-4.725	0.000
Tharion et al. [118]	H S SO	Heavy	-1.104	0.289	0.083	-1.670	-0.539	-3.826	0.000
Lyons et al. [111]	H D SO	Heavy	-0.294	0.312	0.097	-0.906	0.317	-0.943	0.345
Lyons et al. [111]	MDCR	Moderate	0.108	0.310	0.096	-0.500	0.716	0.348	0.728
Gabbett [106]	H D SO	Heavy	-0.542	0.485	0.235	-1.491	0.408	-1.118	0.264
Lyons et al. [45]	Expert H D CR	Heavy	-0.759	0.446	0.199	-1.633	0.115	-1.701	0.089
Lyons et al. [45]	Expert M D CR	Moderate	-0.086	0.430	0.185	-0.929	0.756	-0.201	0.841
Lyons et al. [45]	Novice H D CR	Heavy	-1.892	0.523	0.274	-2.918	-0.866	-3.615	0.000
Lyons et al. [45]	Novice M D CR	Moderate	-0.768	0.446	0.199	-1.643	0.107	-1.721	0.085
Sunderland & Nevill [117]	H D SO	Heavy	-0.564	0.460	0.212	-1.466	0.338	-1.225	0.220
Groslambert et al. [107]	H S SO	Heavy	-2.056	0.539	0.290	-3.111	-1.000	-3.816	0.000
Dickin & Doan (104)	H S SO	Heavy	-0.420	0.349	0.122	-1.104	0.264	-1.202	0.225
McGregor et al. [112]	H D SO	Heavy	0.153	0.451	0.204	-0.732	1.038	0.339	0.735
Evans et al. [105]	H S SO	Heavy	-2.099	0.498	0.248	-3.075	-1.124	-4.217	0.000
Yaggle & Armstrong (91)	H S SO	Heavy	-1.249	0.375	0.140	-1.983	-0.515	-3.333	0.001
Wikstrom et al. [119]	H S SO	Heavy	0.088	0.310	0.096	-0.520	0.696	0.284	0.776
Vullierme et al. [37]	H S SO	Heavy	-1.290	0.499	0.249	-2.269	-0.312	-2.584	0.010
Johnston et al. [110]	H S SO	Heavy	-1.785	0.369	0.136	-2.508	-1.062	-4.840	0.000
McMorris et al. (58)	MDCR	Moderate	0.048	0.395	0.156	-0.726	0.823	0.122	0.903
McMorris et al. [113]	H D CR	Heavy	-0.174	0.412	0.170	-0.981	0.634	-0.421	0.674
McMorris et al. (113)	MDCR	Moderate	0.733	0.426	0.181	-0.102	1.567	1.721	0.085
Lidor et al. (56)	M D SO	Moderate	-0.069	0.273	0.075	-0.605	0.467	-0.252	0.801
Bullock et al (55)	M D SO	Moderate	0.327	0.218	0.047	-0.100	0.754	1.502	0.133
McRae et al. [54]	M D SO	Moderate	0.154	0.412	0.170	-0.653	0.961	0.375	0.708
Anshel & Novak [101]	M D SO	Moderate	0.704	0.218	0.047	0.277	1.131	3.230	0.001
			-0.548	0.132	0.018	-0.808	-0.289	-4,139	0.000



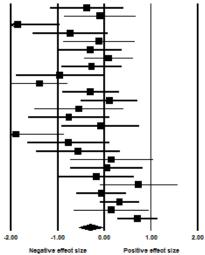


Study name	Subgroup within s	studycompariso	<u>n</u>		Statistics	tor each	study		
			Point estimate	Standard error	Variance		Upper limit	Z-Value	p-Value
McMorris et al. [114]	H D CR	Heavy	-0.377	0.399	0.159	-1.159	0.404	-0.946	0.344
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Tharion et al. [118]	H S SO	Heavy	-1.104	0.289	0.083	-1.670	-0.539	-3.826	0.000
Lyons et al. [111]	H D SO	Heavy	-0.294	0.312	0.097	-0.906	0.317	-0.943	0.345
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Lyons et al. [45]	Expert H D CR	Heavy	-0.759	0.446	0.199	-1.633	0.115	-1.701	0.089
Lyons et al. [45]	Novice H D CR	Heavy	-1.892	0.523	0.274	-2.918	-0.866	-3.615	0.000
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Groslambert et al. [107]	H S SO	Heavy	-2.056	0.539	0.290	-3.111	-1.000	-3.816	0.000
Dickin & Doan (104)	H S SO	Heavy	-0.420	0.349	0.122	-1.104	0.264	-1.202	0.229
McGregor et al. [112]	H D SO	Heavy	0.153	0.451	0.204	-0.732	1.038	0.339	0.735
Evans et al. [105]	H S SO	Heavy	-2.099	0.498	0.248	-3.075	-1.124	-4.217	0.000
Yaggle & Armstrong [91]	H S SO	Heavy	-1.249	0.375	0.140	-1.983	-0.515	-3.333	0.001
Wikstrom et al. [119]	H S SO	Heavy	0.088	0.310	0.096	-0.520	0.696	0.284	0.776
Vullierme et al. [37]	H S SO	Heavy	-1.290	0.499	0.249	-2.269	-0.312	-2.584	0.010
Johnston et al. [110]	H S SO	Heavy	-1.785	0.369	0.136	-2.508	-1.062	-4.840	0.000
McMorris et al. (113)	H D CR	Heavy	-0.174	0.412	0.170	-0.981	0.634	-0.421	0.674
		-	-0.857	0.146	0.021	-1,144	-0.570	-5.851	0.000



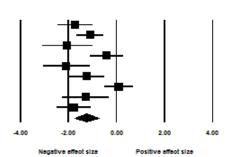
Point estimate and 95% CI

Study name	Subgroup within stu	d <u>©ompariso</u> n	1		Statistics	for each	study			
			Point estimate	Standard error	Variance		Upper limit	Z-Value	p-Value	
McMorris et al. [114]	H D CR	Heavy	-0.377	0.399	0.159	-1.159	0.404	-0.946	0.344	I
McMorris et al. [114]	MDCR	Moderate	-0.097	0.395	0.156	-0.871	0.678	-0.245	0.807	I
McMorris & Rayment [ 115	H D CR	Heavy	-1.853	0.459	0.211	-2.752	-0.953	-4.036	0.000	-
McMorris et al. (57)	H D CR	Heavy	-0.735	0.409	0.167	-1.537	0.066	-1.798	0.072	I
McMorris et al. [57]	MDCR	Moderate	-0.116	0.395	0.156	-0.891	0.659	-0.294	0.769	I
All et al. [97]	H D SO	Heavy	-0.303	0.347	0.120	-0.983	0.378	-0.872	0.383	I
Young et al. [121]	H D SO	Heavy	0.099	0.268	0.072	-0.428	0.625	0.367	0.713	I
Davey et al. [103]	H D SO	Heavy	-0.274	0.328	0.107	-0.916	0.368	-0.836	0.403	I
Bottoms et al. [102]	H D SO	Heavy	-0.956	0.478	0.228	-1.893	-0.019	-2.000	0.046	I
Impellizzieri et al. [108]	H D SO	Heavy	-1.389	0.305	0.093	-1.988	-0.791	-4.550	0.000	ŀ
Lyons et al. [111]	H D SO	Heavy	-0.294	0.312	0.097	-0.906	0.317	-0.943	0.345	I
Lyons et al. [111]	MDCR	Moderate	0.108	0.310	0.096	-0.500	0.716	0.348	0.728	I
Gabbett [106]	H D SO	Heavy	-0.542	0.485	0.235	-1.491	0.408	-1.118	0.264	I
Lyons et al. [45]	Expert H D CR	Heavy	-0.759	0.446	0.199	-1.633	0.115	-1.701	0.089	I
Lyons et al. [45]	Expert M D CR	Moderate	-0.086	0.430	0.185	-0.929	0.756	-0.201	0.841	I
Lyons et al. [45]	Novice H D CR	Heavy	-1.892	0.523	0.274	-2.918	-0.866	-3.615	0.000	
Lyons et al. [45]	Novice M D CR	Moderate	-0.768	0.446	0.199	-1.643	0.107	-1.721	0.085	I
Sunderland & Nevill [117]	H D SO	Heavy	-0.564	0.460	0.212	-1.466	0.338	-1.225	0.220	I
McGregor et al. [112]	H D SO	Heavy	0.153	0.451	0.204	-0.732	1.038	0.339	0.735	I
McMorris et al. (58)	MDCR	Moderate	0.048	0.395	0.156	-0.726	0.823	0.122	0.903	I
McMorris et al. [113]	H D CR	Heavy	-0.174	0.412	0.170	-0.981	0.634	-0.421	0.674	I
McMorris et al. [113]	MDCR	Moderate	0.733	0.426	0.181	-0.102	1.567	1.721	0.085	I
Lidor et al. [56]	M D SO	Moderate	-0.069	0.273	0.075	-0.605	0.467	-0.252	0.801	I
Bullock et al (55)	M D SO	Moderate	0.327	0.218	0.047	-0.100	0.754	1.502	0.133	I
McRae et al. [54]	M D SO	Moderate	0.154	0.412	0.170	-0.653	0.961	0.375	0.708	I
Anshel & Novak [101]	M D SO	Moderate	0.704	0.218	0.047	0.277	1.131	3.230	0.001	I
			-0.296	0.127	0.016	-0.546	-0.047	-2.327	0.020	



Point estimate and 95% CI

Point estimate and 95% CI



Study name	Subgroup within	Subgroup within stud Comparison				Statistics for each study					
			Point estimate	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value		
Sarshin et al. (116)	H S SO	Heavy	-1.726	0.365	0.133	-2.442	-1.010	-4.725	0.000		
Tharion et al. [118]	H S SO	Heavy	-1.104	0.289	0.083	-1.670	-0.539	-3.826	0.000		
Groslambert et al. [107]	H S SO	Heavy	-2.056	0.539	0.290	-3.111	-1.000	-3.816	0.000		
Dickin & Doan (104)	H S SO	Heavy	-0.420	0.349	0.122	-1.104	0.264	-1.202	0.229		
Evans et al. [105]	H S SO	Heavy	-2.099	0.498	0.248	-3.075	-1.124	-4.217	0.000		
Yaggle & Armstrong (91	1)H S SO	Heavy	-1.249	0.375	0.140	-1.983	-0.515	-3.333	0.001		
Wikstrom et al. [119]	H S SO	Heavy	0.088	0.310	0.096	-0.520	0.696	0.284	0.776		
Vullierme et al. [37]	H S SO	Heavy	-1.290	0.499	0.249	-2.269	-0.312	-2.584	0.010		
Johnston et al. [110]	H S SO	Heavy	-1.785	0.369	0.136	-2.508	-1.062	-4.840	0.000		
			-1.243	0.257	0.066	-1.747	-0.740	-4.839	0.000		