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Acute incremental exercise, performance of a central executive task, and sympathoadrenal system and hypothalamic-pituitary-adrenal axis activity

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ABSTRACT

The purposes of this study were to examine the effect of acute incremental exercise on the performance of a central executive task; the responses of the sympathoadrenal system (SAS) and hypothalamic-pituitary-adrenal axis (HPAA) during exercise, while simultaneously carrying out the central executive task; and the ability of Δ plasma concentrations of epinephrine, norepinephrine, adrenocorticotropin hormone (ACTH) and cortisol to predict Δ performance on the central executive task. Subjects undertook a flanker task at rest and during exercise at 50% and 80% maximum aerobic power (MAP). SAS and HPAA activity were measured pre- and post-treatment by plasma concentrations of catecholamines, and cortisol and ACTH, respectively. Reaction time (RT) and number of errors for congruent and incongruent trials on the flanker task showed significant main effects with performance at 80% MAP higher than in the other conditions. RT post-correct responses were significantly faster than RT post-error at rest and 50% MAP but not at 80%. Pre- and post-treatment catecholamines showed a main effect of exercise with a linear increase. Post-treatment ACTH concentrations at 80% MAP were significantly greater than in the other conditions. Δ epinephrine and ACTH combined were significant predictors of Δ RT and Δ norepinephrine was a significant predictor of Δ number of errors. It was concluded that exercise must be at a high intensity to affect performance on the flanker task. Both the SAS and HPAA appear to play a role in the exercise–cognition interaction.

1. Introduction

The primary purposes of this study were to examine the effect of acute incremental exercise on the performance of a central executive task; the responses of the sympathoadrenal system (SAS) and hypothalamic-pituitary-adrenal axis (HPAA) during exercise, while simultaneously carrying out the central executive task; and the ability of changes from baseline (Δ) plasma concentrations of epinephrine, norepinephrine, adrenocorticotropin hormone (ACTH) and cortisol to predict Δ performance on the central executive task. The need of sports performers and the military to make quick and accurate decisions, while simultaneously undertaking exercise of various intensities, has led to a large amount of research in this area (see Tomporowski, 2003; McMorris, 2006, for reviews). Moreover, as exercise is a stressor, there may be implications for the effect of other physiological stressors on cognition.

The majority of the research examining the effect of exercise on cognition has tended to use simple cognitive tasks, e.g. choice reaction time, visual search, which do not activate the same areas of the brain that are used during decision making in sports and military situations (Tomporowski, 2003; McMorris, 2006). Recently a number of researchers have examined the effects of exercise on central executive tasks, however in the majority of studies, cognitive testing has taken place post-exercise (Hillman et al., 2003; Themanson and Hillman, 2006; Kamijo et al., 2007; Coles and Tomporowski, 2008). The problems with testing post-exercise have long been documented (Tomporowski and Ellis, 1986). Individuals, especially fit ones as most subjects were in these experiments, recover very quickly from exercise of the types used by the researchers (Kjaer, 1989). Thus the psychophysiological status of the subjects during cognitive testing that is undertaken post-exercise is not the same as it was during the exercise. Also Tomporowski (2003) highlighted the fact that exercise intensity may be a key factor affecting results. Therefore, we decided to examine the effect of exercising at 50% and 80% maximum aerobic power (MAP) on performance of a central executive task, Eriksen's flanker task (Eriksen and Eriksen, 1974). Thus the problem of recovery would be controlled by testing during exercise, and the protocol would also allow comparison of the effects of exercising at differing intensities.

No other study has attempted to examine the effect of incremental exercise on performance of the flanker task. However, Pontifex and Hillman (2007) examined the effect of undertaking the task while

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exercising at 60% maximum heart rate (HR_{MAX}), a moderate intensity. They found no effect on response time (RT) and showed a decrease in accuracy of response but only on incongruent trials, i.e. where the target and noise stimuli elicit conflicting responses. In the present experiment, we also tested during moderate intensity exercise, albeit higher than that used by Pontifex and Hillman, and during heavy exercise. Pontifex and Hillman found that N2 amplitude, which is indicative of response inhibition (Ridderinkhof, 2002), was greater in incongruent trials compared to congruent, i.e. when the target and noise stimuli elicit the same response. They also found that N2 amplitude decreased during exercise.

Given that response inhibition is of greater importance during incongruent compared to congruent trials and that Pontifex and Hillman (2007) found a decrease in N2 amplitude during exercise, we hypothesized that incongruent and congruent trials would be affected differently by exercise. For incongruent trials, we hypothesized an increase in errors and RT at 80% MAP. Exercise at this intensity is far more stressful than that used by Pontifex and Hillman (Kjaer, 1989; Deuster et al., 1989) and several authors have claimed that central executive tasks will be disrupted at high exercise intensities only (e.g., Dietrich, 2003, 2009; Tomporowski, 2003). These claims are based on cognitive-energetic/arousal–performance interaction theories, which state that performance at high levels of stress will be disrupted (e.g., Sanders, 1983), and cognitive neuroscientific claims that, during high levels of stress, there is competition for resources between different centers of the brain resulting in weaker cognitive functioning (Miller and Cohen, 2001). Given that Pontifex and Hillman (2007) found a significant increase in errors at 60% HR_{MAX}, we hypothesized such an increase at 50% MAP. Although Pontifex and Hillman showed no significant effect on RT at 60% HR_{MAX}, we hypothesized an increase in RT at 50% MAP on incongruent trials as exercise at this intensity constitutes a significant increase in perceived stress from rest (Borg, 1998). We also hypothesized a significant increase in errors and RT from 50% to 80% MAP, as the latter is considerably more stressful than the former (Borg, 1998).

Pontifex and Hillman's (2007) results would suggest no effect of exercise on performance on the congruent trials. However, as there is little response inhibition involved in these trials they are very similar to choice reaction time tasks. Research examining the effect of exercise on choice reaction time tests has tended to show a linear improvement in performance during exercise at the same intensities as those used in this study (see Tomporowski, 2003; McMorris, 2006, for reviews). McMorris (2006) claimed that the exercise-induced increases in arousal, even at an intensity as high as 80% MAP, have a beneficial effect on these comparatively simple tasks. Therefore, we hypothesized a linear improvement in performance from rest to 80% MAP for congruent trials. We also examined the effect of exercise on responses following an error. Gehring et al. (1993) claimed that, when an individual perceives an error, they compensate by allocating more resources to higher centers of the brain, particularly the prefrontal cortex (PFC), thus slowing RT on the next response. Based on Dietrich's (2003) and Miller and Cohen's (2001) claims concerning competition for resources between different centers of the brain during stress, we expected that post-error responses would show a linear increase from rest to 80% MAP. However, we expected that post-correct responses would not be significantly effected as there would be no increase in PFC activity.

The second major aim of this study was to examine the interaction between exercise, cognitive performance, and SAS and HPAA activity, as measured by plasma concentrations of epinephrine and norepinephrine, and cortisol and ACTH, respectively. Animal studies have shown increased turnover of the neurotransmitters norepinephrine and dopamine in the brain during exercise (Gerin and Privat, 1998). As a result, researchers have argued that there is an interaction between SAS activity, exercise and cognitive performance (Cooper, 1973; Chmura et al., 1994; McMorris et al., 2008). Evidence for such an interaction is, however, somewhat equivocal (Winter et al., 2007; McMorris et al., 2008). This may be because, during exercise, the HPAA is also active, resulting in moderate concentrations of plasma cortisol during moderate intensity exercise and very high concentrations during heavy exercise (Deuster et al., 1989). Vedhara et al. (2000) found that high concentrations of cortisol had a negative effect on cognition. We hypothesized that plasma concentrations of norepinephrine, epinephrine, ACTH and cortisol would be greater during exercise than at rest and concentrations at 80% MAP would be higher than at 50%.

The third purpose of this study was to examine the ability of Δ plasma concentrations of epinephrine, norepinephrine, ACTH and cortisol to predict Δ performance on the central executive task. If plasma concentrations are indicative of the effects of SAS and HPAA activity in the brain, one would expect that Δ plasma concentrations would predict Δ performance on the central executive task.

2. Materials and methods

2.1. Subjects

Subjects ($N = 24$) were paid male students, mean (SD) age 24.32 (7.10) years. All participated regularly in recreational sports. HR_{MAX} (184 (9) bpm) was determined during a MAP test. MAP was 305.63 (50.4) W, which is classified as low to moderate (Shvartz and Reibold, 1990). They signed written consent forms, were fully informed about the protocol and completed medical questionnaires prior to testing. They were told that they could withdraw from the study at any time and would still be paid. The experiment was approved by the local ethics committee.

2.2. Flanker task

This was a variation of Eriksen and Eriksen's (1974) flanker test, with colors replacing letters. The test was displayed on a 43.18 cm CRT computer screen with a 1024 × 768 pixel resolution and 50 Hz frame. The stimulus corresponded to three colored circles (radius = 0.6 cm), horizontally arranged, which were presented in a known location immediately after a 1 s fixation point, on a grey rectangular background (7.2 cm × 1.6 cm). Participants had to respond according to the color of the central circle (the target) and to ignore the color of the flanker circles (the distractors) which were presented simultaneously on both sides of the target. The visual angle between the target and flanker was 0.06° and the distance, measured from center to center, was 3.0 cm. Participants sat on a cycle ergometer, with a response button on each of the handle grips, facing a computer screen. They were instructed to press the right-hand side button when the colors blue or yellow were presented centrally and the left button when red or green were presented centrally. As soon as a response key was pressed, the stimulus disappeared. When participants failed to respond within 1 s, the stimulus disappeared and the next trial began. The interval between the disappearance of the display and the onset of the next was 500 ms. The flanker circles could be the same as the target color or different to the color but representing the pressing of the same button (e.g. target stimulus—red and flanker—green). These were the congruent trials. Flanker color could be different to the stimulus color and representing the pressing of the opposite button (e.g. relevant stimulus—red and flanker—blue). These were the incongruent trials. The dependent variables were congruent and incongruent RT, and number of errors.

2.3. Procedure

Participants undertook a habituation session on the flanker task followed by a MAP test. There was a 48 h gap between the MAP test and the first experimental session. The MAP test and each of the

experimental tests took place at the same time of the day with 48 h between tests. Order of testing was counterbalanced.

The habituation session on the flanker task consisted of eight blocks of 64 trials, which a pilot study had shown was necessary in order to reach a stable level of performance and minimize potential learning effects. This was followed by a MAP test (Myles et al., 1980) from which 50% and 80% of the participant's MAP were calculated.

All testing took place in an environmental chamber with a constant temperature of 19 °C and a relative humidity of 60%. A venous blood sample (7.5 ml) was taken from an antecubital vein by a trained phlebotomist when the participant entered the chamber. This was to familiarize the participant with the process as previous research (Grayson et al., 1997) has shown that the initial needle puncture can cause a temporary raising of catecholamines concentrations. The data from this sample were not included in the statistical analyses. The participant then sat for 15 min, mounted the cycle ergometer and a second blood sample was taken, termed the pre-treatment sample. The sample was immediately dispensed into two 5 ml tubes each containing 50 µl of potassium ethylene diamine tetracetic acid and placed on ice. The samples were then centrifuged at 4 °C and 1900 *g* in a refrigerated centrifuge (8000 series, Centurion Scientific, Oxford, UK) after which plasma was removed and stored at −85 °C prior to analysis. Samples were assayed using CATCOMBI, ACTH and cortisol kits (IBL Hamburg, Germany) prior to analysis using solid phase enzyme-linked immunosorbent assay.

In the two exercise sessions the participant warmed-up with a resistance of 75 W for 5 min and then cycled, at the required intensity, for 15 min or until voluntary exhaustion. At the third, eighth and thirteenth minutes the participant undertook one of three blocks of 96 trials on the flanker test, while continuing to cycle. Heart rate was continuously recorded during all sessions and pedaling rate was freely chosen (the ergometer automatically adjusted the resistance of the electronic break as a function of the pedal rate in order to maintain a constant power output). Blood was taken immediately following cessation of exercise. Eight participants failed to complete the 13th minute of exercise in the 80% MAP condition. Their final blood sample was taken immediately following cessation of exercise. In the rest condition, the protocol was identical except that the participant sat on the ergometer without exercising.

2.4. Statistical analysis

Mean RT for congruent and incongruent trials at each exercise intensity were examined by a congruency × exercise intensity analysis of variance (ANOVA). Post hoc treatment was by Tukey LSD. Mean RT following correct and error responses were examined in the same way. Sphericity was examined using Mauchly's test. Number of errors at each exercise intensity was compared using the non-parametric Friedman's Test with follow-up Wilcoxon Signed Rank Tests, if appropriate. As eight subjects did not complete the full number of trials at 80% MAP a further analysis with these subjects removed was undertaken. Plasma concentrations of epinephrine and norepinephrine, pre- and post-treatment, at each condition were compared using a pre-/post-treatment × exercise intensity multivariate ANOVA (MANOVA) with follow-up separate univariate ANOVAs. Post hoc comparisons were by Tukey LSD tests. ACTH and cortisol concentrations were examined in the same way. Effect sizes were measured by the η^2_p method.

Multiple regression analyses, using the backward remove method, were carried out with post-treatment (50% and 80% MAP separately) Δ concentrations of epinephrine, norepinephrine, ACTH and cortisol as the independent variables and Δ RT, with congruent and incongruent data combined, as one dependent variable and Δ error as another. Delta concentrations and Δ performance variables were used rather than actual values as individuals differ at baseline in endocrine concentrations, and RT and number of errors. The key issue in the study was how exercise affects changes rather than the actual values. Actual values at a given intensity may be high but represent only a small increase or no increase at all.

3. Results

Mean (*SD*) percentage of HR_{MAX} was 35.83% (5.64) at rest, and 77.08% (5.00) and 89.83% (3.59) at 50% and 80% MAP, respectively. Mean (*SD*) power output at 50% MAP was 141.58 W (19.21) and 226.33 W (30.47) at 80% MAP.

Fig. 1 shows the mean (*SD*) RT for congruent and incongruent trials on the flanker task. A congruency × exercise intensity ANOVA showed that RT in the congruent trials was significantly faster than in the incongruent trials ($F(1,23) = 158.68$, $p<0.001$, $\eta^2_p = 0.87$). There was also a main effect for exercise intensity ($F(2,46) = 5.14$, $p<0.01$, $\eta^2_p = 0.18$). Post hoc Tukey LSD tests showed that RT at 80% MAP was significantly slower than at 50% MAP. RT at 80% MAP was also slower than at rest and the difference was approaching significance ($p = 0.08$). There was no significant difference between RT at rest and during exercise at 50% MAP. There were no violations of sphericity.

Mean (*SD*) number of errors at rest, and during exercise at 50% and 80% MAP were 1.29 (0.55), 1.62 (0.82) and 3.67 (2.41), respectively. Friedman's Test showed a significant effect of exercise ($\chi^2(N = 24) = 29.55$, $p<0.001$). There was no significant effect of removing the eight subjects who did not complete the 80% MAP condition ($\chi^2(N = 16) = 18.54$, $p<0.001$). Post hoc Wilcoxon Signed Rank Tests found significant differences between performance at 80% MAP and at rest ($Z = 3.89$, $p<0.001$) and 50% MAP ($Z = 3.71$, $p<0.001$). There were no significant effects of removing the eight subjects who did not complete the 80% MAP condition, $Z = 3.07$, $p<0.005$ between rest and 80% MAP and $Z = 2.97$, $p<0.005$ between 50% and 80% MAP.

Fig. 2 shows the mean (*SD*) RT for post-error and post-correct responses. A response type × exercise intensity ANOVA found main effects for response type ($F(1,23) = 36.66$, $p<0.001$, $\eta^2_p = 0.62$) and exercise intensity ($F2,46) = 4.99$, $p<0.01$, $\eta^2_p = 0.18$) but these were superseded by a two-way interaction ($F(2,46) = 3.86$, $p = 0.05$, $\eta^2_p = 0.14$). Post hoc tests showed that RT post-correct responses at 80% MAP was significantly slower than in the other two conditions,

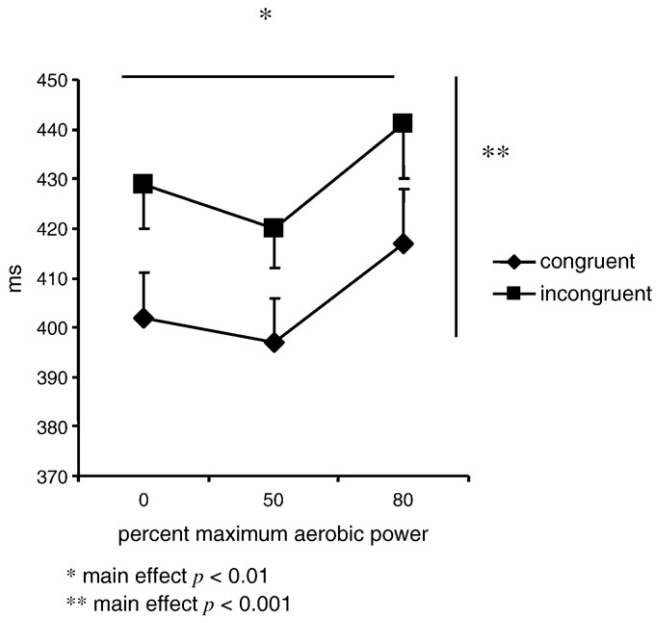

Fig. 1. Mean (SD) response times at rest and during exercise at 50% and 80% maximum aerobic power, for congruent and incongruent trials, on the flanker task.

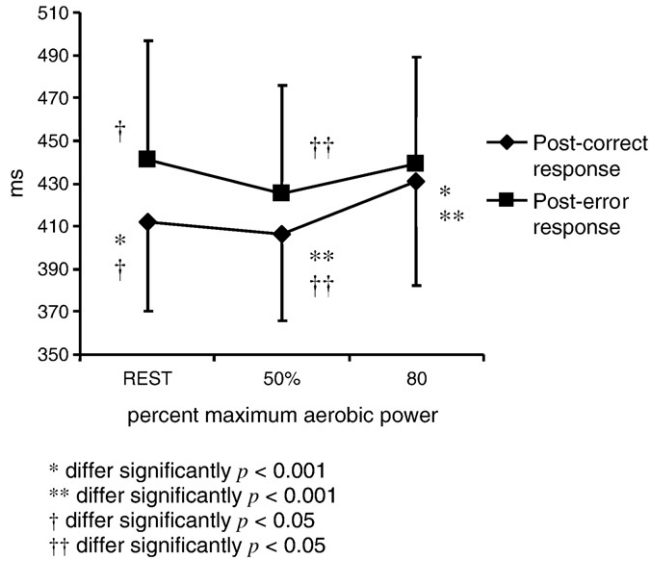

Fig. 2. Mean (SD) response times at rest and during exercise at 50% and 80% maximum aerobic power, post-correct and post-error responses, on the flanker task.

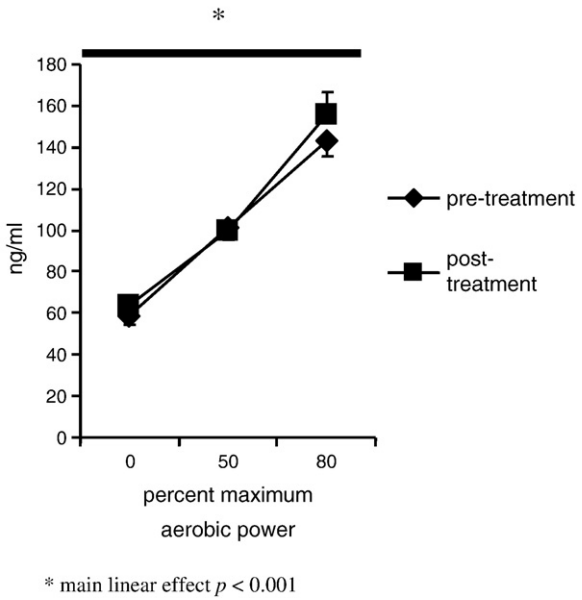

Fig. 4. Mean (SD) plasma concentrations of norepinephrine pre- and post-treatment at 50% and 80% maximum aerobic power.

which did not differ significantly from one another. Post-error responses showed no significant effect of exercise. Post-correct RTs at rest and 50% MAP were significantly lower than post-error at the same intensity but at 80% MAP there was no significant difference. There were no violations of sphericity.

Figs. 3 and 4 show the mean (*SD*) plasma concentrations of epinephrine and norepinephrine, respectively, at each exercise intensity. A pre-/post-treatment × exercise intensity MANOVA demonstrated a main effect of exercise for epinephrine and norepinephrine plasma concentrations ($\Lambda = 0.15$, $F(4,90) = 35.91$, $p < 0.001$, $\eta_p^2 = 0.62$). Examination of the separate univariate ANOVAs showed that data for both epinephrine ($F(2,46) = 74.58$, $p < 0.001$, $\eta_p^2 = 0.76$) and norepinephrine ($F(2,46) = 72.58$, $p < 0.001$, $\eta_p^2 = 0.76$) contributed significantly to the MANOVA data. Tukey LSD tests found that concentrations at rest were significantly lower than during exercise at either intensity and concentrations at 80% MAP were significantly higher than at 50%. There were no violations of the assumptions for the tests.

Figs. 5 and 6 show the mean (*SD*) plasma concentrations of ACTH and cortisol, respectively, at each exercise intensity. The failure of one participant to supply sufficient blood for assay pre-treatment at rest and 50% MAP meant that his data could not be included in the statistical treatment. Pre-/post-treatment x exercise intensity MANOVA demonstrated significant main effects for pre-/post-test ($\Lambda = 0.36$, $F(2,21) = 18.47$, $p < 0.001$, $\eta_p^2 = 0.64$) and exercise intensity ($\Lambda = 0.49$, $F(4,88) = 9.38$, $p < 0.001$, $\eta_p^2 = 0.30$) but these were superseded by a pre-/post-test × exercise intensity interaction effect

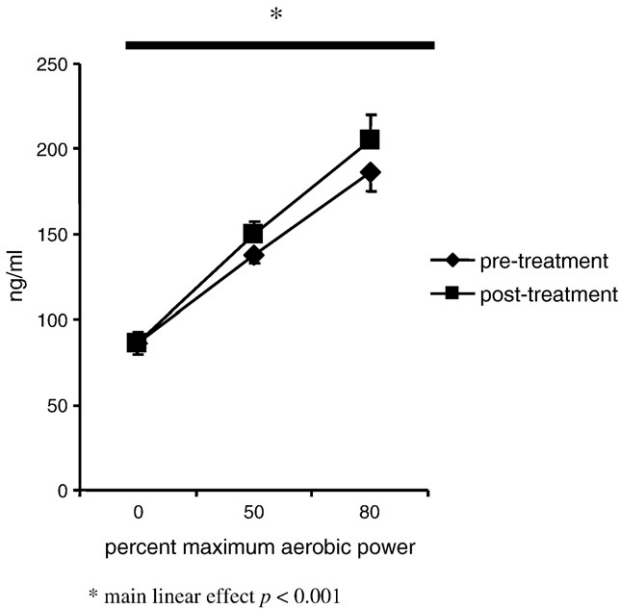

Fig. 3. Mean (SD) plasma concentrations of epinephrine pre- and post-treatment at 50% and 80% maximum aerobic power.

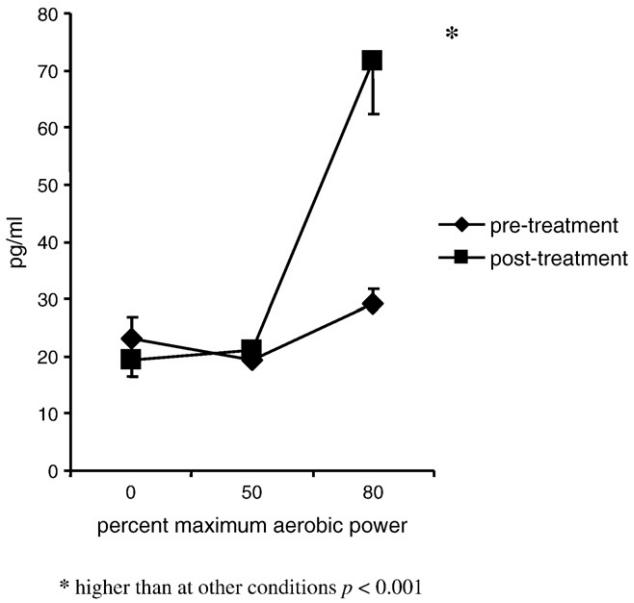

Fig. 5. Mean (SD) plasma concentrations of adrenocorticotropin hormone pre- and post-treatment at 50% and 80% maximum aerobic power.

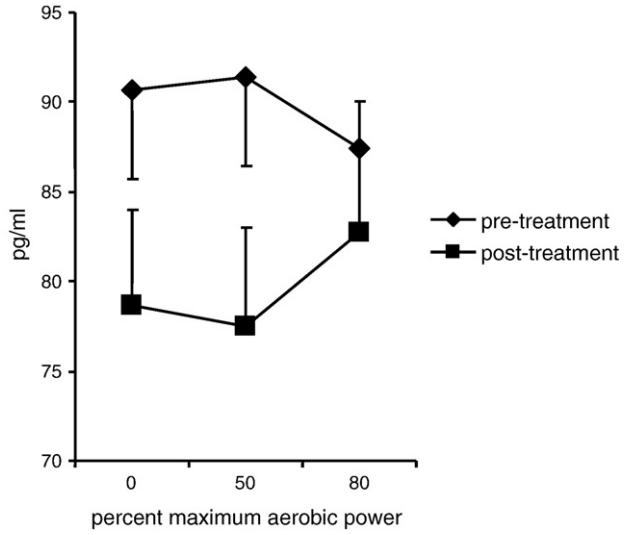

Fig. 6. Mean (SD) plasma concentrations of cortisol pre- and post-treatment at 50% and 80% maximum aerobic power.

($\Lambda = 0.46$, $F(4,88) = 10.12$, $p < 0.001$, $\eta_p^2 = 0.32$). Follow up separate univariate ANOVAs showed that cortisol data only contributed significantly to the pre-/post-treatment effect MANOVA data ($F(1,22) = 20.42$, $p < 0.001$, $\eta_p^2 = 0.48$), with pre-treatment concentrations being the greater. However, ACTH data contributed to the pre-/post-treatment effect MANOVA data ($F(1,22) = 27.43$, $p < 0.001$, $\eta_p^2 = 0.56$), the exercise effect MANOVA data ($F(2,44) = 22.80$, $p < 0.001$, $\eta_p^2 = 0.51$) and the interaction effect MANOVA data ($F(2,44) = 25.09$, $p < 0.001$, $\eta_p^2 = 0.53$). Post hoc Tukey tests found that ACTH plasma concentrations at 80% MAP post-treatment were significantly different to those in all other conditions, none of which differed significantly from one another. There were no violations of the assumptions for the tests.

Multiple regression analyses with post-treatment Δ epinephrine, norepinephrine, ACTH and cortisol as the independent variables and Δ RT (with congruent and incongruent data combined) as the dependent variable found that, at 80% MAP, Δ epinephrine and ACTH combined significantly predicted Δ RT ($R^2 = 0.29$, $p < 0.05$). Observation of the standardized β-coefficients showed that, for both variables, the relationship was inverse. Δ norepinephrine was a significant predictor of Δ error ($R^2 = 0.40$, $p < 0.01$) at 80% MAP. β-coefficients demonstrated a positive relationship. At 50% MAP there were no significant predictors for either variable.

4. Discussion

The results of this experiment provide partial support for our hypotheses. For the RT variable, results of the congruency × exercise intensity ANOVA showed main effects for congruency and exercise intensity but no interaction effect. Post hoc Tukey LSD tests showed that RT at 80% MAP was significantly slower than at 50% MAP. RT at 80% MAP was also slower than at rest and the difference was approaching significance ($p = 0.08$). Observation of Fig. 1 suggests a difference between RT at rest and during exercise at 50% MAP, however this is not reach significance ($p = 0.22$), probably due to the high SDs. These results are similar to those of Pontifex and Hillman (2007) who found no effect of exercise at 60% HR_{MAX} on RT. Although the exercise intensity in our experiment was higher (mean % HR_{MAX} at 50% MAP was 77.08 (5.00)) than that in Pontifex and Hillman's study, it would appear that 50% MAP induces insufficient arousal to affect any changes in RT for congruent and incongruent trials. To some extent this may be explained by the fact that, at 50% MAP, there were no significant increases in ACTH and cortisol plasma concentrations, while catecholamines showed only small, albeit significant, increases. These results differ from those of Kamijo et al. (2007), who found a decrease in RT for incongruent trials. However, as stated above, Kamijo et al. tested following cessation of exercise, therefore it is difficult to compare the results with those of the present study.

The most surprising aspect of our findings was that there was no congruency × exercise interaction effect. We had expected that congruent and incongruent trials would be affected differently. The results for the main effect of exercise, outlined above, were similar to those we expected for incongruent trials only, with an increase in RT at 80% MAP. For the congruent trials, however, we had expected either no effect or possibly a linear improvement in performance. The fact that there was a significant main effect for congruency, supports the notion that congruency affects performance. Moreover, the literature on the flanker task strongly demonstrates that incongruent trials show greater activation of the anterior cingulate cortex (Fan et al., 2005), which plays the major role in response inhibition, a vital component on the flanker task (Ridderinkhof, 2002). As Pontifex and Hillman (2007) demonstrated a significant exercise-induced decrease in N2 amplitude, which is indicative of decreased anterior cingulate cortex activity, one would have expected that the incongruent trials, which require high levels of response inhibition, would have been more detrimentally affected than the congruent trials.

It should be noted that even congruent trials involve response inhibition (Eriksen and Eriksen, 1974), albeit to a lesser extent. The possibility that the heavy exercise was such that it resulted in negative effects for both conditions cannot be discounted. That this occurred at 80% MAP but not 50% MAP, and not at 60% HR_{MAX} in Pontifex and Hillman's (2007) experiment, raises the possibility that there is a threshold level before which no effects are shown. We should note, however, that Dietrich (2003) presents another possible explanation. According to Dietrich, the key issue is the fact that both congruent and incongruent trials activate the PFC. Based on the ideas of Miller and Cohen (2001), he claims that, during exercise, there is competition for resources between the PFC and motor cortex. When the motor demands are high, resources are allocated to the motor cortex at the expense of the PFC, hence a detriment in performance. Dietrich (2009) argued that this will only happen when exercise demands are high.

Results for number of errors in the present study also show an increase at 80% MAP regardless of congruency. This is different to the findings of Pontifex and Hillman (2007) who showed an effect on incongruent trials only. As with RT it is possible that the higher exercise intensities used in the present study were the reason for this finding. The possibility of a threshold effect cannot be ignored. As exercise intensity increases it is possible that the stress on the ability of the anterior cingulate cortex to inhibit incorrect responses becomes such that both congruent and incongruent trials are affected in the same way.

Results for post-error responses failed to support our hypothesis, showing no significant effect of exercise on RT. Interestingly, post-correct response RT showed a decrement in performance at 80% MAP. The reasons for this are probably the same as those outlined above concerning RT for congruent and incongruent trials. We had expected post-error responses to be negatively affected by exercise. As stated earlier, Gehring et al. (1993) claimed that, when an individual perceives an error, they compensate by allocating more resources to higher centers of the brain, particularly the PFC, thus slowing RT on the next response. Based on Dietrich's (2003) and Miller and Cohen's (2001) claims concerning competition for resources between different centers of the brain, we expected that post-error responses would be negatively affected by exercise because of the increased resources allocated to the PFC. Not only did we not find this but, at 80% MAP, there was no significant post-correct/post-error difference in RT. It is possible that the post-error responses are so difficult that a floor effect

exists, therefore the participant cannot get any slower during exercise at 80% MAP. Another possible explanation for this is that, at 80% MAP, the stress is so high that the individual fails to monitor errors and so when responding in the post-error condition, in fact, responds in the same way as in the post-correct response condition.

Results for the effect of exercise at 50% and 80% MAP, while simultaneously undertaking a central executive task, on SAS and HPAA activity as measured by plasma concentrations of epinephrine and norepinephrine, and cortisol and ACTH, respectively, also provide only partial support for our hypotheses. MANOVA results for the catecholamines demonstrated a significant main effect for exercise intensity. This was as expected, however the lack of a main effect for pre-/post-treatment or an interaction effect is surprising. A significant linear increase in post-treatment catecholamines concentrations from rest to 80% MAP is inevitable due to the role played by these neurohormones in glycolysis, lipolysis and the regulation of cardiorespiratory responses during exercise (Borer, 2003). One would also be likely to see a significant pre-treatment linear increase as catecholamines concentrations are known to increase due to SAS activity resulting from increased arousal in anticipation of the upcoming task (Åstrand et al., 2003). Nevertheless, one would expect to see quite large pre-/post-treatment differences. Observation of Fig. 4 shows a difference between mean pre- and post-treatment concentrations, however the difference is not significant ($p = 0.34$) probably due to high inter-individual differences, which are indicated by the SDs. From these data it appears that pre-treatment arousal levels in some participants were high. It is possible that the participants' preconceptions of task difficulty were higher than the task turned out to be, resulting in a lack of significance between pre-and post-treatment data. Results for HPAA activity provide some support for this explanation.

MANOVA for cortisol and ACTH demonstrated significant main effects for pre-/post-treatment, exercise intensity and an interaction effect. Although cortisol did not contribute significantly to the interaction effect it did contribute to the pre-/post effect, with pre-treatment concentrations being significantly higher than post-treatment. Given that cortisol is indicative of arousal, as are epinephrine and norepinephrine, this finding supports the claim that participants were aroused in anticipation of task demands. Data from the interaction effect, to which only ACTH contributed significantly, suggest that the exercise-induced changes in HPAA activity only occurred at 80% MAP. For ACTH, plasma concentrations at 80% MAP post-treatment were significantly different to those in all other conditions, none of which differed significantly from one another. This finding is important because of the very limited peripheral role of ACTH during exercise of this duration (Deuster et al., 1989). With catecholamines it is difficult, in the post-treatment conditions, to state with certainty whether changes were due to increased central arousal, increased need for the neurohormones peripherally or, as is most likely, a combination of both. With ACTH, however, it is probable that this increase is due to central factors, possibly an emotional response to the physical and/or mental stress. From these results it would appear that HPAA activity is greatly increased at 80% MAP. These data suggest a possible threshold level at which HPAA activity occurs.

That cortisol and ACTH have produced different results may be due to the protocol used. ACTH is a precursor of cortisol and changes in plasma concentrations of ACTH normally precede changes in cortisol concentrations by about 15 min (Genuth, 2004). We considered taking a further blood sample 15 min after the post-treatment sample. However, we decided to limit the amount of samples due to the already invasive nature of the experiment and also because taking too many samples increases the possibility of results being contaminated by stress responses to the blood sampling rather than the activity. Given the ACTH-cortisol interaction one would assume that had we taken a blood sample 15 min later an increase in cortisol concentrations would have been demonstrated. Hence the ACTH concentrations are likely to be the better indicators of HPAA activity using our protocol.

That there were no significant regression correlations at 50% MAP between Δ plasma concentrations of cortisol, ACTH, epinephrine and norepinephrine, and Δ RT, with congruent and incongruent data combined, and Δ error is not surprising given the few significant effects in RT and number of errors, and small, significant only for catecholamines, increases in endocrine concentrations shown at 50% MAP. It is likely that, at this intensity, there is only a small interaction and with a sample size of only 24 there is insufficient power to demonstrate an effect. At 80% MAP there was a significant regression correlation between Δ epinephrine and ACTH combined, and Δ RT. This is not unexpected but observation of the standardized β-coefficients shows that the direction is inverse, i.e. smaller increases in ACTH and epinephrine concentrations indicate larger increases in RT, while larger increases indicate smaller increases in RT. ACTH and epinephrine concentrations are indicative of increases in stress and arousal. Therefore, according to arousal–performance interaction theories, we would have expected greater increases in arousal to be related to poorer performance. Similarly, one would expect increased concentrations of epinephrine and ACTH to be related to increased limbic system activity, which would negatively affect PFC activity and hence RT would increase (Miller and Cohen, 2001; Dietrich, 2003). In making sense of these data, we must keep in mind the fact that even the smaller Δ values still represent increases in ACTH and epinephrine concentrations. Similarly, the smaller Δ RT also represent slower performance compared to at rest.

A possible explanation for these data lies in the nature of the protocol for the flanker test. The task is a central executive one, therefore one would expect greater increases in epinephrine and ACTH, which are indicative of stress, to be related to slower information processing and hence indicate higher Δ RTs. However, the response, pressing a button, is motoric in nature and previous research has shown decreases in motor time in such situations (Davranche et al., 2005, 2006). Therefore, one might expect large increases in epinephrine and ACTH concentrations to predict decreases in the motor time aspect of the task. As a result, even if central processing is slower during exercise at 80% MAP, this will be off-set by faster motor time, resulting in a smaller increase in RT. Participants who had a smaller increase in epinephrine and ACTH combined would have a weaker or no positive effect on motor time. Hence, smaller Δ epinephrine and ACTH relates to larger Δ RT. Obviously one must proceed with caution when examining this explanation as no separate measures of central activity or motor time were taken.

The correlation between Δ norepinephrine and Δ error at 80% MAP, is not surprising. Increases in norepinephrine may be indicative of increased arousal, with very high increases representing over-arousal, thus performance would deteriorate. Indeed, this result adds support to the claim that the regression data for Δ RT is more related to motor time than to central information processing.

The strength of the correlations for both RT and number of errors are a little higher than we anticipated, as catecholamines do not cross the blood brain barrier and, therefore, only provide a rough indication of central changes. Also the role of cortisol peripherally during exercise will affect the ability of plasma concentrations of the hormone to predict central activity. Due to this problem we felt that the use of hierarchical regression analyses was inappropriate at this time, as it requires a model suggesting a direct interaction. Moreover, the fact that HPAA activity has not been measured previously in exercise–cognition interaction research made the presentation of such a model somewhat problematic. The results of this study provide useful information for those wishing to develop a testable model.

Before summarizing the findings of this study, we should point out a number of limitations. Blood sampling was taken by venepuncture rather than cannulation. The decision to do this was because, in pilot

work, several participants had felt that the cannula inserted in the lower arm inhibited their motor response. While the decision to use venepuncture may have had a good effect on this aspect of the task, venepuncture can be stressful to participants. Future research should examine the possibility of using other cannulation sites. Also no subjective measures of perceptions of fatigue, pain or stress were used during exercise. While we accept that this could be construed as a limitation, we felt that plasma concentrations of epinephrine, norepinephrine, ACTH and cortisol are better indicators of the stress induced by responses to heavy exercise than are subjective measures. Indeed, our hypotheses that exercise will affect cognition are based on the SAS-HPAA interaction that occurs during exercise, or due to any peripheral stressor. During exercise, feedback from the Autonomic Nervous System to the hypothalamus, concerning stress on the cardiorespiratory system, pain and glycogen depletion, triggers a response by the hypothalamus. This results in increased release of epinephrine and norepinephrine peripherally, and norepinephrine and dopamine centrally (Genuth, 2004). Moreover, as the physiological stress increases, feedback to the hypothalamus, concerning the individual's perceptions of both the physiological and psychological stress, sets in motion HPAA activity (Brandenberger et al., 1980; Genuth, 2004). Our argument is that SAS and HPAA activity are the organism's response to all of the stressors involved in exercise whether they be physiological or perceptual. The use of subjective, as well as objective measures, in future research, may allow us to differentiate between the comparative effects of these stressors, although it is likely that the relative importance varies greatly between subjects.

To summarize, the results of this study suggest that, when undertaking a central executive task, the physiological stress needs to be high before a detrimental effect is shown on RT. Congruent and incongruent RT combined, and post-correct RT demonstrated deteriorations in speed at 80% MAP. Number of errors also only showed an effect at 80% MAP. These results suggest the possibility of there being an exercise threshold level at which performance begins to deteriorate. Increases in SAS activity appear to begin pre-treatment with definite signs of an anticipation effect. As expected, plasma concentrations of the catecholamines increased during exercise. Given that these changes occurred pre- and post-treatment it would appear likely that changes occurred both peripherally and centrally. HPAA activity is more complex, with cortisol providing evidence of an anticipation effect but with no exercise intensity effect. However, ACTH concentrations were negatively affected by exercise at 80% MAP. As stated earlier, ACTH plays very little part in peripheral activity during exercise of this kind (Deuster et al., 1989), therefore changes are almost certainly due to central effects. However, the relationship between SAS and HPAA activity, and RT is counter-intuitive, with large increases in activity being related to less disruption in RT at 80% MAP rather than more. More research in which pre-motor and motor times are differentiated is required to help explain this surprising result. Given the changes in HPAA activity and the evidence of the regression analyses, we believe that we have presented a case for the claim that SAS activity alone does not explain the exercise–cognition interaction. It would appear that the HPAA plays a part, particularly during heavy exercise.

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