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Title: Brain and cardiorespiratory responses to exercise in hot and thermoneutral conditions

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ABSTRACT

The aim of this study was to test whether or not concurrent evaluations of brain (electroencephalography [EEG]) and cardiorespiratory responses to exercise are influenced by environmental conditions. Ten adult male participants performed a standardised incremental exercise test to exhaustion on a cycle ergometer in an environment controlled laboratory on two separate occasions, in a randomised order; one in a hot condition (34.5°C) and one in a thermoneutral condition (20°C). EEG, heart rate and expired air were collected throughout. EEG data were decontaminated for artefacts, log-transformed and expressed as aggregated alpha and beta power responses across electrodes reflecting the frontal cortex of the brain. Performance outcomes showed there was no difference in $\dot{V}O_2$ peak across Hot (42.5 ml/kg/min) and Neutral (42.8 ml/kg/min) conditions, although ventilatory threshold (V_T) occurred at a lower threshold (68%) in Hot, compared with Neutral condition (74%) ($p < 0.05$). EEG alpha and beta wave responses both demonstrated significant increases from baseline to V_T ($p < 0.01$). EEG beta-band activity was significantly elevated in the heat compared to the Neutral condition. In conclusion, elevated EEG beta-band activity in response to incremental exercise in the heat suggests that beta-band activation and cortical awareness increases as exercise becomes increasingly intense.

Key words: EEG; heat; ventilatory threshold; incremental exercise

Introduction

Few studies have compared simultaneous brain, cardiorespiratory and performance outcomes, either in thermoneutral [21] or hot conditions. This is surprising as increasing evidence suggests exercise performance is continuously manipulated in response to the interaction of numerous physiological systems monitored by the brain via constant feedforward and feedback loops between peripheral systems and the brain [7, 18, 23]. In this sense, the brain controls (and oscillates) motor unit recruitment so that the individual can complete the known duration of a task prior to experiencing conscious sensations of intolerable physical discomfort [7], which might otherwise necessitate the immediate cessation of exercise.

Early investigations have shown hot environmental conditions are associated with altered brain activity of the frontal cortex with progressive hyperthermia [19]; however, this effect has not yet been demonstrated in studies comprising an exercise-matched thermoneutral control condition, nor in the context of whether or not alterations to brain activity coincide with cardiorespiratory and performance factors in challenging exercise situations. If the brain regulates performance then it would be expected that altered brain activity may be identifiable across 1) progressively intense, incremental exercise and 2) across matched-exercise trials performed in environmental conditions imposing differential thermal challenge.

It is well-known that in response to high-intensity exercise, many individuals reach a maximal level of tolerable discomfort, at which point they voluntarily decide to cease exercise without exhibiting a plateau in $\dot{V}O_2$ [19, 23]. This raises the question as to whether maximal performance is truly experienced, or simply whether a maximal level of tolerable discomfort (or effort) is reached in advance of physiological system failure [22]. It seems plausible that an exercise challenge evoking greater sensations of discomfort such as in hot

conditions might also result in differential neural activity in the frontal cortex of the brain where planning, motivation and the projection of future consequences resulting from current actions occur [3]. However, simultaneous evaluations of cortical brain activity, physical performance and cardiorespiratory evaluations have rarely been considered [7] and to our knowledge no studies have yet examined responses to the same exercise challenge across different environmental conditions, nor included matched control conditions.

Electroencephalography (EEG) provides important indices of change in cortical potentials, rhythmic activity and frequency of synaptic processes [26]. Therefore, if brain regulation is influential and/or limiting to physical activity, EEG outputs from the frontal cortex may demonstrate increased neural processing in response to incremental exercise. Although data are currently scarce, EEG outputs to incremental exercise in thermoneutral conditions have shown EEG activity from the frontal cortex to increase up to ~150 W (approx. 50-60% of an exercise test to exhaustion) and thereafter either plateau or decline, although current data are derived from limited sampling frequencies [4, 7]. EEG derived data may reveal systematic differences in electro cortical activity between experimental exercise conditions such as in hot and thermoneutral conditions where the exercise challenge is matched across trials so that motor unit recruitment patterns are comparable, yet the different environmental conditions create an additional stimulatory challenge.

It seems likely that a study involving time aligned and frequent sampling of both EEG and cardiorespiratory signals prior to exhaustion in different environmental conditions may yield important data relating to the role of the brain in the fatiguing process [3, 7]. Therefore the hypothesis of this study is that completing a standardised incremental exercise test to exhaustion in hot conditions will result in greater EEG activity from the frontal cortex of the brain reflecting elevated neural processing and conscious awareness of undesirable

environmental heat compared to exercise in a thermoneutral environment. Alterations in EEG outcomes may also coincide with compromised physical performance and/or physiological system deficits in the heat compared to a thermoneutral condition.

Materials & Methods

Participants

Ten moderately fit men (age = 27 ± 4 years; mass = 88.0 ± 10.6 kg; height = 180 ± 5.6 cm) were given written instructions describing all procedures related to the study and each provided informed consent prior to participation. The study was approved by the Research and Ethics Committee of James Cook University. The research project was further conducted in accordance with the ethical standards of the Int J Sports Med journal and meets those ethical standards [10].

All participants were recreational exercisers, free of medications (e.g. antidepressants, antipsychotics) and free of known disease (e.g. epilepsy) that may influence exercise tolerance and EEG responses.

Experimental design

The study required the participants to attend the laboratory on two occasions, seven days apart at the same time of day. On their first visit basic anthropometric measurements were recorded. On both visits the participants underwent an incremental exercise test to volitional exhaustion on a cycle ergometer (Velotron, Racermate, United States) in either ambient, thermoneutral conditions (Neutral: 20°C , $\sim 60\%$ Relative Humidity [RH]) or hot conditions (Hot: 34.5°C , $\sim 65\%$ RH), performed in a random order. Tests were performed in the tropical,

humid environment of Cairns, Australia and the introduction of heat to the environmental chamber generally raised the physical challenge of that condition and hence clear differentiation between neutral and hot tests with additional humidity. Following EEG cap fitting and cardiorespiratory equipment in a thermoneutral environment, participants were transferred via a connecting door into an enclosed environmental chamber, which both regulated the environment and minimised the influence of external electrical interference on EEG signals. On entry to the chamber, participants completed a standardised 3-min warm-up, before commencing the incremental exercise test protocol, during which pulmonary gas exchange, heart rate and EEG parameters was recorded at 1-min intervals. For accurate between condition comparisons of EEG, data were further aligned into epochs at 10% increments of exercise bout duration for standardisation due to inevitable differences in the duration of time taken to complete maximal exercise bouts between individuals [7]. The exercise protocol required participants to pedal at 85-90 revolutions per minute, commencing at 100W for the first 3-min. At the end of the third minute power (W) was increased by $20\text{W}\cdot\text{min}^{-1}$ and the test continued until volitional exhaustion or if revolutions per min fell to below 70. Participants were required to remain seated and in a fixed posture controlled position via mouthpiece alignment and hand placements on handlebars throughout the protocol.

The experimental protocol was designed to investigate brief, acute exposure responses to the heat and thus total exercise and exposure time was approx. 20min for each participant. Core body temperature was not measured as preliminary testing and interrogation of the literature identified that the brief duration of heat and exercise exposure would be insufficient to differentially raise core temperature as this typically requires exposures of approx. 60min [6]. Core temperature assessment was therefore not included. The utilisation of a brief duration exercise protocol was further motivated by the desire to minimise the influence of sweat

artefacts. Throughout preliminary trials and the main experiment there was no evidence of differential sweat production from the scalp, while the selected EEG measurement sites (F3, F4, Fz) are also well known to display 70% less sweat production than the forehead [15], facilitating meaningful comparisons between conditions. In addition, opportunities for sweat induced cross-talk between electrode sites were minimised by utilising only a short protocol and investigating only EEG sites of interest in the frontal cortex that were well spaced on the cranium rather than a broader spectrum of electrodes.

Expired gases were collected via a one-way breathing system (Hans-Rudolph, United States). Oxygen uptake corresponding to a ventilatory threshold was determined for each participant in response to both incremental exercise tests using the validated V Slope method [2] by independent analysis. Ratings of perceived exertion (RPE) using the 6-20 Borg Scale were collected immediately following the exercise protocol rather than during exercise so to not disturb concentration or evoke changes in eye, mouth or limb movement which could induce data artefacts to the EEG signals.

Electroencephalogram recordings and analysis

Prior to each condition, an EEG-cap (Electro-cap International, Inc., USA) with 16 measurement and EEG electrodes arranged in the international 10-20 system was fitted to the participants head. In accordance with similar exercise-based experiments examining exercise and fatigue [3], EEG electrode sites (F3, F4, Fz) were analysed from the 16 measurement sites as the area of focus and aggregated to reflect frontal cortex activation. EEG data were log transformed and auto decontaminated for artefacts such as blinking and heart rhythms using the auto-elimination tool of the BESA software (version 5.2 MEGIS software GmbH, Germany) for consistent quantitative practice across all participants and all tests [3, 5, 9] expressed as alpha (8-13Hz) and beta (13-30Hz) power responses for the frontal cortex. The

EEG cap was permeable to air in order to prevent an increase in heat during cycling. Electrodes were filled with Signa-gel™ (Parker Laboratories Inc., USA) for optimal signal transduction. A Biosemi Active two amplifier (Biosemi Inc., Amsterdam, Netherlands) was used with high and low pass filters across a frequency range from 0.16 to 100Hz for analysis. The Biosemi amplifier utilises a system of active electrodes which detects small uV EEG signals and then outputs the high impedance (80K) signal via the active electrode to the amplifier in low impedance. The active electrodes (output impedance of less than 1 Ohm) are relatively robust to interference pickup of the cable and this suppresses interference by impedance transformation directly on the electrode. The active electrode system is therefore considerably more robust to movement artefacts in response to exercise than earlier passive electrode systems. A sample rate of 2 KHZ was used for the Biosemi Active Two EEG amplifier on advice from the system manufacturer. All EEG recordings were sampled for a period of 1-min to minimise the effects of non-physiological noise on the signal. EEG data were processed according to log transformation, decomposed into frequency components and clustered into broad bands. EEG in each band was then quantified according to absolute power. Data were analysed using BESA software (version 5.2 MEGIS software GmbH, Germany) [4]. A power spectrum was calculated from the EEG signal using Fast Fourier Transformation and expressed as log transformed outputs for alpha, beta and alpha/beta ratio results for comparison.

Statistical analysis

Data for Hot and Neutral were checked for normal distribution, analysed using repeated measures ANOVA (time x condition) with Greenhouse-Geisser correction when the assumption of sphericity was violated (SPSS version 19.0). Post-hoc Tukey tests of honest significant difference were used to examine differences where indicated by significant

interaction effects from ANOVA evaluation. Statistical significance was accepted at $p < 0.05$.

All data are presented as means \pm SD unless otherwise stated.

Results

Cardiorespiratory responses

Measurements of cardiorespiratory responses are shown in Table 1. There was no difference in $\dot{V}O_2$ peak between Hot and Neutral conditions, nor was there a difference in peak power output, peak heart rate or post-test blood lactate concentration. However, ventilatory threshold was experienced at a significantly lower threshold of peak performance in the Hot condition ($p < 0.05$).

Average heart rate responses did not differ between the Hot and Neutral condition at V_T (Neutral: 150.4 ± 13.5 b/min; Hot: 154.9 ± 13.9 b/min; $P = 0.09$) or at $\dot{V}O_2$ peak (Neutral: 190.2 ± 15.5 b/min; Hot: 187.1 ± 16.8 b/min; $P = 0.1$). There was no change to body mass from pre to post-test in either Hot or Neutral condition.

The EEG responses to exercise demonstrated significant main time effects for increased activity in Hot and Neutral conditions for both α - and β -bands for the frontal cortex from baseline (eyes open) to V_T ($p < 0.01$) in both Hot and Neutral conditions (Fig. 1 and 2 respectively). Thereafter, α - and β -band activity maintained elevation compared to baseline but did not increase further, instead plateauing in both α - and β -bands until the cessation of exercise.

Although there was a main time effect for increased EEG α -band activity in response to the exercise protocol, repeated measures ANOVA (time \times condition) did not indicate between-condition difference across the exercise protocol for α -band activity (Fig. 1). However,

analysis of β -band activity demonstrated both main effect (time) and interaction (time x condition), such that β -band activity was significantly elevated in the Hot condition (Fig. 2) from the 40% measurement interval of the protocol until the cessation of exercise (100%). This effect tended to coincide with the range over which V_T occurred in the test population (47 – 93% of peak of $\dot{V}O_2$ peak).

Analysis of alpha/beta band ratio identified clearly different trajectories across the exercise protocol for Hot and Neutral conditions, primarily reflecting the impact of elevated β -band activity in the Hot condition. Repeated measures ANOVA indicated significant interaction (time x condition) and pairwise comparisons revealed significant effects at measurement intervals 10-60% of the protocol duration (Fig. 3).

Ratings of perceived exertion (RPE) were collected immediately post-test rather than during the exercise protocols so as not to interfere with the participants by introducing movement, changes to visual direction and encouraging speech, all of which could have influenced EEG signals. Post-test evaluations identified that both incremental tests were rated as RPE 20 ± 1 as equally exhausting. Practical retrospective evaluation of more detailed perceived responses such as RPE did not yield robust data.

Discussion

The main finding of this study was that the EEG β -band responses from the frontal cortex displayed significantly elevated activity when participants completed short duration (~20min), incremental exercise to volitional fatigue in the heat compared with a thermoneutral condition. Evidence from this study indicates that such exercise performed in the heat appears to trigger moderate suppression of α -band and elevation of β -band activity of

the frontal cortex, resulting in significant differences in alpha/beta ratio scores between conditions which most likely reflect elevated neural processing of environmental factors, greater arousal and conscious awareness of the likely demands of the impending exercise challenge [16, 25]. This study therefore provides new evidence supporting the contention that elevations to β -band activity may be of importance for the execution of exercise performed when exposed to challenging circumstances where greater intrinsic motivation, planning, and projection of anticipated outcome are required.

EEG alpha brain waves have traditionally been described as evidence of an inactive cortex and reduced arousal [17, 20]. In this study, α -band activity increased similarly in both conditions at the onset of exercise (Fig. 1). It is well-known that at the start of exercise, the anticipatory role of catecholamines (specifically adrenaline) is particularly to up-regulate cardiovascular preparedness for physical challenges [8, 28]. An abrupt up-regulation of both α - and β -band activity is therefore normal at the onset of exercise [24] and is supported by similar changes to heart rate, vasodilatory responses and mobilization of metabolic fuel which would all be expected to also increase in anticipation of exercise [28].

Although few studies have yet examined simultaneous EEG and exercise responses, evidence indicates that alpha activity shows no sign of an effect specific to the α -band. Studies that have found significant change in this band have tended to analyse raw absolute power values [14]. However, due to the lack of normality in EEG data, outputs ought to be previously treated with a log power transformation [5, 9] as has been conducted in this experiment. Consequently, particular focus on alpha activity may lead to premature conclusions about the meaning of brain electro cortical responses to exercise.

The observation of significant elevations to β -band activity in the heat trial (Fig. 2) suggests a specific effect may be evident at this frequency in challenging and/or hot conditions. This

may be explained by greater hypothalamic modulation of increased temperature during exercise which is thought to influence electro cortical activity in the β -band range [16-17, 21]. Alterations in brain blood flow particularly cause changes in the EEG pattern in the beta range [12]; however, underlying causes for the influence of exercise on brain blood flow require further investigation.

In accordance with earlier research, elevated β -band activity in response to exercise may be related to an attentional demand and a higher arousal level [16], such as processing undesirable conscious sensations. Literature generally agrees that exercise enhances cortical activation, which is typically represented by increased beta activity [13, 29]. Therefore, our observations of an increase in beta activity at the frontal cortex as a significant main time effect for both conditions could be directly related to an excited state of mind and increased cortical activation [12], such as would be expected when processing awareness of the implications of exercise to exhaustion, such as at the period over which V_T occurs when exercise starts to become more intense, blood lactate and H^+ accumulate and sensations such as nausea begin to accrue [7]. Neural processes are exacerbated in the heat where a more challenging physical environment could add to the conscious awareness of the impending demands of the task, which may explain the elevation of β -band in the heat [21].

Currently, no studies have reported simultaneous EEG responses and ventilatory parameters in hot conditions, nor included comparative matched-exercise thermoneutral (control) observations. Exercise based comparisons of EEG responses are complicated by the possible inclusion of muscle artefacts and this cannot be ruled out in this study. However, movement was severely restricted (participants remained seated at all times, with hands in a fixed position on handle bars and 2 research observers present for all trials) in our cycling protocol in accordance with earlier experiments from this group and with standard procedures from other

groups [1, 3-4, 13], there was no observable change in posture or position while cycling in thermoneutral or hot conditions during the protocol and standardised artefact elimination tools were utilised for the purpose of eliminating bias. Therefore, any systematic effect observed is likely to be a meaningful physiological effect that is of statistically significant magnitude in excess of any random influence of muscle noise interference.

In this study, $\dot{V}O_2$ peak and peak power output were similar across environmental conditions; however, V_T occurred at a lower percentage of peak performance in the Hot condition (Hot: 68.3%, Neutral: 74.4%) (Table 1) ($p < 0.05$), indicating that maximal performance was preserved by factors such as intrinsic motivation across conditions [7, 17]. V_T is independent of motivation and so significant difference in β -band activation in the Hot condition, particularly over the period where V_T occurred when the brain would have been processing regulatory feedback from peripheral physiological systems. It is possible this might explain the observations of this study, as considerable motivation and effort is needed to sustain exercise and this represents a conscious desire to override overwhelming sensations of fatigue [17]. This would be manifested in greater neural activation in a more challenging condition such as in the heat. Examples of neural regulation of performance can easily be discerned in hot conditions, where athletes are well known to self-regulate (pace) performance from the initial stages of the bout [26], well in advance of the failure of heat dissipation mechanisms such as sweating [23]. Therefore, conscious attention may be focused on achieving the best possible outcome, tempered by the realisation of the physical consequences and projected sensations of system failure in the heat [7]. In response to exercise in this experiment, the incremental exercise test was matched across conditions, perhaps due to increased β -band activation. This might not be the case in longer duration, challenging protocols designed to elicit a change in core temperature rather than the short duration heat exposure from this experiment. These observations require further investigation in other studies.

A recent study has also observed a rapid rise in EEG activation from the onset of exercise up to approx. 50% of peak power, thereafter they reported a decline in all EEG activity, indicating this might demonstrate evidence of brain regulatory control in maximal exercise via inhibitory effects [20] although this could also reflect changes to CO₂ tension in the cerebral tissue as this is known to reduce the amplitude of EEG signal [11]. It was also not possible to measure PaCO₂ in our study and this is therefore a potential limitation for exercise and EEG observations as CO₂ tension can produce changes in the EEG power spectrum; however, these tend to be particularly evident in the alpha band rather than beta which was the area of change in this experiment [10].

Our observations were of maintained, rather than reduced, EEG activity at 50% of peak power and were thus consistent with current evidence from other studies [1, 4]. Nevertheless, EEG power certainly plateaued from the period over which V_T occurred and did not increase in line with ventilatory parameters. Therefore, whether a decline or a plateau is observed in EEG from 50% of the bout is largely irrelevant as it does not keep pace with increases to power output during incremental exercise, nor ventilation, heart rate or oxygen uptake. Beta activity therefore appears to reach a maximal level much earlier in the exercise bout, coinciding loosely with the period at which V_T occurs. It is therefore possible that this supports the contention that a form of neural inhibitory control may be plausible and worthy of further investigation [7, 17, 21, 26].

In summary, while there may be no argument that limits exist to which humans can perform physical tasks, the causes of such limitations remain disputed. The findings of this study indicate that brain processes detected by EEG β -band activation from the frontal cortex differentiate exercise performance in the heat vs. thermoneutral conditions. This may support the contention that increased awareness during exercise influences neural drive and in turn motor unit recruitment.

Conflict of Interests Statement:

The authors of this manuscript have no conflicts of interest.

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Figures

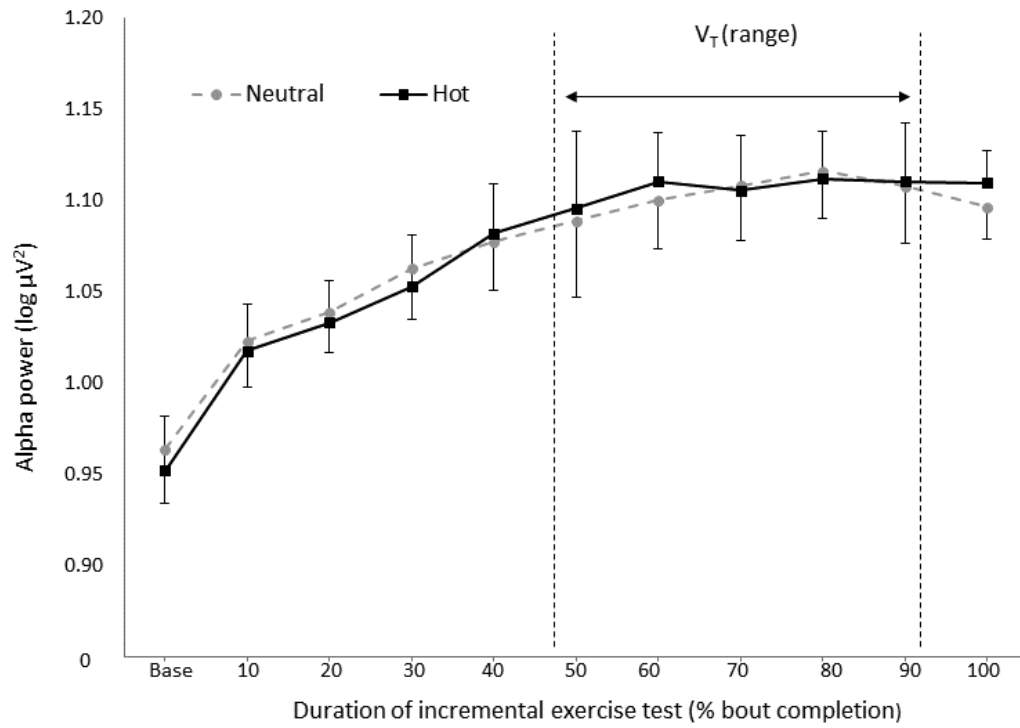


Fig. 1 Mean EEG alpha band activity aggregated across F3, F4, Fz electrodes at 10% intervals of exercise bout duration in thermoneutral and hot conditions.

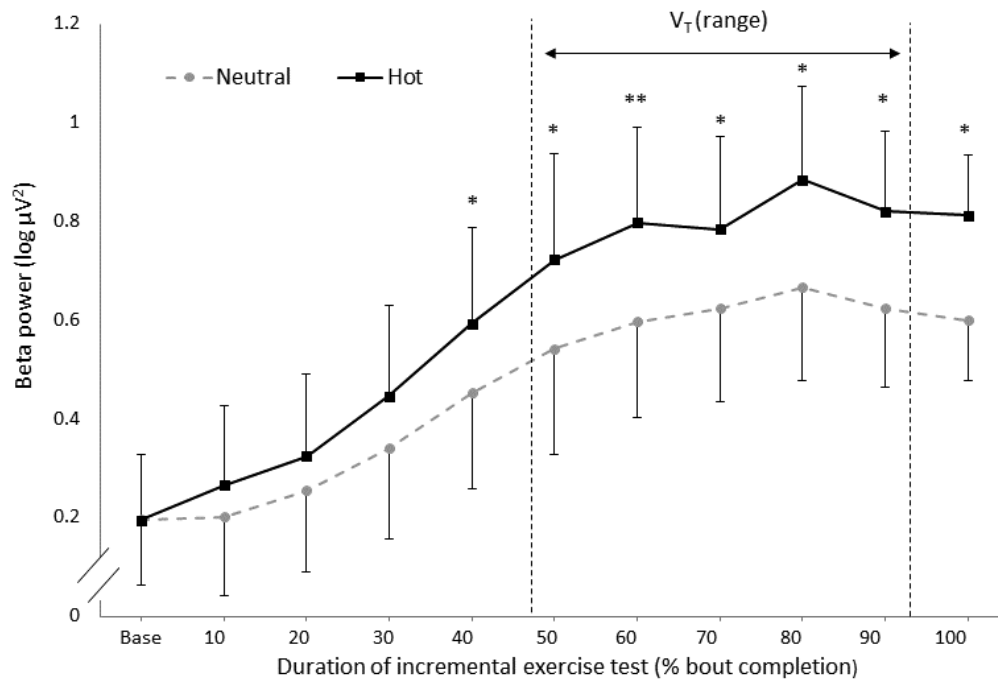


Fig. 2 Mean EEG beta band activity aggregated across F3, F4, Fz electrodes at 10% intervals of exercise bout duration in thermoneutral and hot conditions.* between condition difference = $p < 0.05$, ** between condition difference = $p < 0.01$.

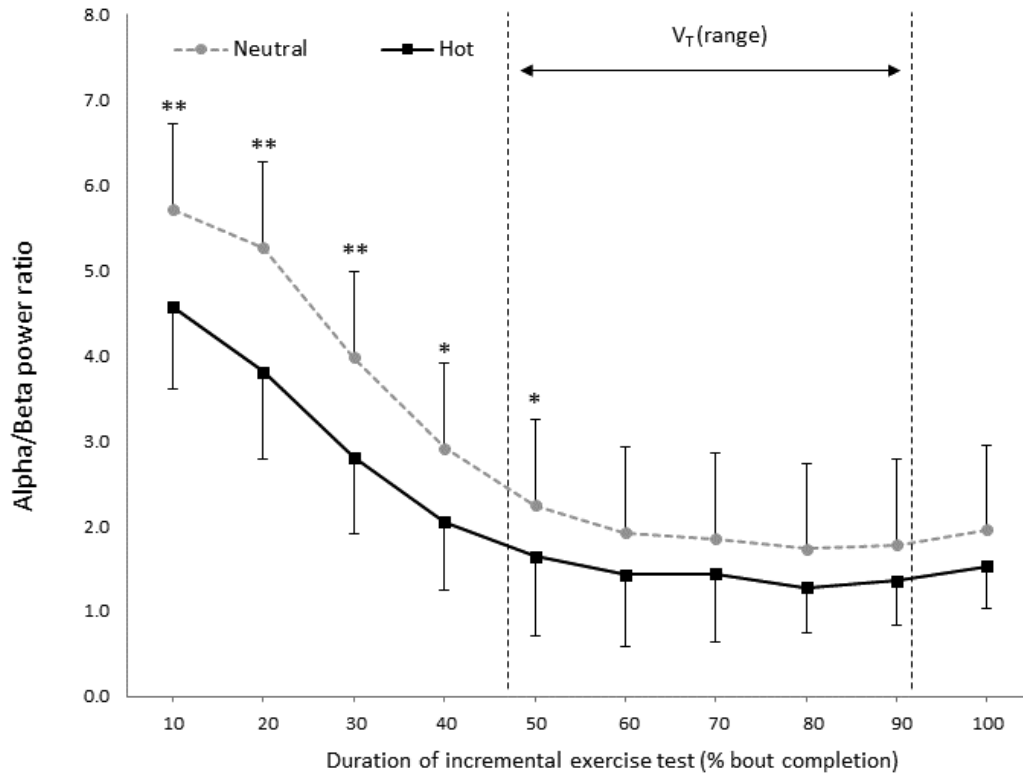


Fig. 3 Mean EEG alpha/beta ratio band activity aggregated across F3, F4, Fz electrodes at 10% intervals of exercise bout duration in thermoneutral and hot conditions.* between condition difference = $p < 0.05$, ** between condition difference = $p < 0.01$.

Table 1. Cardiorespiratory responses to the Hot and Neutral incremental exercise tests to exhaustion.

	V_T	V_T	V_T	$\dot{V}O_2$ peak	$\dot{V}O_2$ peak
Condition	(ml/kg/min)	(% $\dot{V}O_2$ peak)	(W)	(ml/kg/min)	(W)
Neutral (n=10)	31.8 ±2.5*	74.4 ±6.3*	204.2 ±16.6*	42.8 ±3.4	320 ±25.6
Hot (n=10)	28.9 ±2.3	68.3 ±5.5	181.2 ±14.5	42.5 ±3.2	306 ±24.2

V_T = ventilatory threshold; $\dot{V}O_2$ peak = peak oxygen uptake. * p<0.05. ±SD