Original Article

Exercise performance and neuromuscular activity at a fixed level of RPE following manipulation of peripheral physiological status

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ABSTRACT

Browne, S. & Renfree, A. (2013). Exercise performance and neuromuscular activity at a fixed level of RPE following manipulation of peripheral physiological status. *J. Hum. Sport Exerc., 8*(3), pp.820-828. PURPOSE: to analyse the effect of manipulation of peripheral physiological status on performance and neuromuscular activity during cycling at a fixed level of RPE. METHODS: following familiarisation trials, seven well trained individuals completed two exercise trials following ingestion of 0.2g.kg-1 NaHCO3 or a CaCO3 placebo which were performed in a randomised and blind manner. During exercise participants were required to cycle at exercise intensity equivalent to their perception of an RPE of 16 on the Category Ratio Scale. Blood pH was measured pre- and post-exercise, and power output, EMG activity in the active musculature, and heart rate were recorded continuously throughout exercise. Exercise was terminated when power output fell below 80% of the average recorded over the first 3 minutes of each trial. RESULTS: pre-exercise pH was higher following NaHCO3 ingestion, but post-exercise values did not differ between NaHCO3 and placebo trials. Exercise duration was 21% longer following NaHCO3 than the placebo, but no significant differences were found in power output or heart rate between trials at any point. EMG activity was higher throughout NaHCO3 trials. CONCLUSIONS: the findings of this study suggest that NaHCO3 ingestion enhanced exercise duration by allowing an increased volume of exercise to be performed prior to individuals reaching individual critical threshold values for pH. Lower neuromuscular activity despite a similar power output following NaHCO3 suggests that perceptions of effort may be based on absolute work rates rather than afferent physiological feedback, and that the work rate is achieved through regulation of efferent neural drive. **Key words**: PERCEIVED EXERTION, ELECTROMYOGRAPHY, SODIUM BICARBONATE.

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INTRODUCTION

The factors involved in the regulation of muscular work rate during self paced exercise have received considerable attention in academic literature in recent years. This is largely due to the realisation that variations in work rate, and in particular the 'endspurt' phenomenon that is frequently observed in the final stages of maximal endurance activity, cannot be explained through purely peripheral physiological mechanisms (Noakes, 2007). A peripheral model of exercise regulation would suggest that exercise capacity is limited by metabolic changes in the muscle that are ultimately determined by the availability of a sufficient oxygen supply (Noakes & St Clair Gibson, 2004). Taken to its logical conclusions, this model suggests that exercise must ultimately terminate as a result of some physiological catastrophe in the muscles, a prediction that is not congruent with the observation that exercise is not normally terminated as a result of absolute system failure (Noakes et al., 2004).

An alternative explanation for the regulation of work rate during exercise is provided by Noakes et al., (2005) who suggest that a central regulator in the brain continually alters exercise intensity in order to ensure there is no loss of homeostasis in the peripheral physiological systems. This regulation of exercise intensity is proposed to be achieved through variation in the number of individual motor units in the active skeletal muscle that are recruited. The number of motor units recruited is based on the brains interpretation of both afferent physiological feedback and knowledge of the endpoint of exercise (St Clair Gibson et al., 2006).

It has also been suggested that regulation of muscle recruitment is achieved through interpretation of the momentary Rating of Perceived Exertion (RPE). During exercise to volitional fatigue at a fixed workload, Noakes (2004) demonstrated that RPE increased in a linear manner during exercise, and that exercise termination coincided with reporting of the highest sustainable RPE. There also appeared to be a scalar quality to this observation, in that when participants completed the exercise task in both glycogen replete and depleted conditions, although there were differences in the rate of increase in RPE between conditions, RPE profiles were identical when plotted against percent of total exercise duration. This model has been further developed by Tucker (2009) who proposed that during self paced exercise of known duration, regulation of work rate is achieved through continual comparison of the 'conscious RPE', and the subconscious 'template RPE'. The template RPE is set based upon knowledge of the endpoint of exercise and provides a means by which the conscious RPE can be interpreted. It is suggested to be based on the athletes expectations of the rate of change in perceptions of effort during the exercise task, previous experience of similar exercise tasks, and is continually updated based on the duration of exercise still to be performed. The conscious RPE is calculated based on interpretation of afferent signals relating to peripheral physiological status. By ensuring the conscious RPE does not exceed the template RPE, it is ensured that maximal tolerable RPE is not achieved prior to task completion, thereby avoiding catastrophic loss of physiological function.

Through use of the RPE 'clamp' method, whereby an individual exercises at a fixed rating of perceived exertion whilst able to vary their power output freely, it has been demonstrated that work rate decreases as a linear function of exercise duration (Tucker et al., 2006). This finding was interpreted to suggest that RPE cannot simply be a measure of exercise intensity, because absolute exercise intensity dropped while RPE remained constant. When this protocol was utilised in differing environmental temperatures, it was found that in the heat the rate of decline was greater and resulted in significantly shorter exercise duration. This greater rate of decline in exercise intensity was accompanied by similar rates of heat storage in both hot

and cold environments, thereby supporting the assertion that exercise intensity is regulated in an anticipatory manner prior to the development of any physiological 'catastrophe'.

Swart et al. (2009) also used the RPE clamp method to examine the effects of ingestion of a centrally acting stimulant (methylphenidate) on CNS regulation of exercise intensity. Following methylphenidate ingestion, participants cycled for 32% longer before power output fell to 70% of starting values, despite displaying higher power outputs, VO2's, heart rates, and blood lactate concentrations at the equivalent time at which exercise was terminated in the placebo trials. These findings were suggested to provide evidence that work rate during prolonged exercise is highly regulated by the CNS to ensure a physiological reserve capacity is always maintained.

Although the study by Swart et al. (2009) does provide evidence that muscular work rate during this type of exercise bout is regulated by the CNS, it would seem plausible that manipulation of physiological status will also influence muscular performance during exercise at a fixed rating of RPE. If the conscious RPE is indeed generated as a result of afferent feedback from the peripheral physiology (Tucker, 2009), then ingestion of a peripherally acting substance would be expected to alter the nature of this feedback and therefore the work rate at the same conscious level of exertion. The aim of this study is therefore to investigate the effect of ingestion of a peripherally acting physiological substance (NaHCO3) on performance, muscular recruitment, and physiological disruption during exercise at a fixed level of RPE.

METHODS

Seven well-trained cyclists (6 male, 1 female, 39.1±12.4 years, 175±7.4 cm, 83.4±19.2 kg) completed the experimental procedures which had prior approval from an institutional ethics committee.

Experimental Procedures

Following an initial familiarisation trial, participants performed two experimental trials after ingestion of 0.2g.kg-1 sodium bicarbonate (NaHCO3) or a calcium carbonate (CaCO3) placebo. Participants were asked to prepare for the experimental trials as they would prior to a minor competition by avoiding high intensity training in the preceding 24 hours and following their usual pre-competition dietary practices.

Experimental treatments were administered in a blind, randomised manner. NaHCO3 and CaCO3 were both dissolved in 300ml of water over a 30 minute period ending 60 minutes prior to the commencement of exercise trials in order to maximise pre-exercise blood pH (Renfree, 2007). All experimental trials were performed on an electronically braked cycle ergometer (Lode Excalibur Sport; Lode Medical Technology, Groningen, The Netherlands). Participants preferred saddle and handlebar positions were recorded during the familiarisation trial and retained for subsequent experimental trials. The ergometer was set to isokinetic mode, and participants cycled at a fixed cadence of 80 r.p.m-1 and an intensity equating to their interpretation of level 16 on Borg's C20 Category Scale (Borg, 1985). Mean power output over the initial 3 minutes was calculated, and participants continued until power output fell below 80% of this value for two consecutive minutes. Participants were provided with no external feedback relating to either power output or duration of exercise at any point during the trials. Instantaneous power output and heart rate (Garmin Edge 305, Southampton, United Kingdom) were recorded at one minute intervals throughout all trials. Blood pH was measured using a Radiometer NPT7 blood gas analyzer (Radiometer Medical, Broshoj, Denmark) via fingertip capillary samples collected immediately prior to and post-exercise. Blood was collected in capillary tubes containing 6 IU of Na-heparin and 9 IU of Li-heparin per 100-µL tube volume,

and samples were taken following immersion of participant's hands in a water bath at ~50oC for 1 min. Blood lactate concentration was measured 3 min post exercise (Arkray Lactate Pro, Suffolk, UK).

Electromyographic Testing

Surface EMG was used to measure neuromuscular activity in the vastus lateralis through each experimental trial. A surface amplifier (SX230, Biometrics, Newport, United Kingdom) was placed on the muscle belly following preparation by shaving and cleaning with light abrasion and an alcohol swab. Immediately prior to commencement of the cycle trials participants performed three maximal 5s voluntary contractions (MVC) of the vastus lateralis on a CMSI Humac Norm dynamometer (Stoughton, Massachusetts, USA). EMG activity was continuously recorded during all cycle trials. All EMG data was subsequently processed using Datalog software version 8.0 (Biometrics, Newport, United Kingdom) and normalised by reporting EMG during experimental trials as a percentage of the highest value recorded during pre-exercise MVC's.

Statistical Analysis

Power output, EMG activity, and HR were averaged over epochs representing 25% of total exercise duration for each individual participant. Differences in each of these parameters between NaHCO3 and CaCO3 trials were assessed using one-way analysis of variance for repeated measures followed by the Tukey post hoc test where necessary. A paired samples t-test was used to identify differences in total exercise duration and blood parameters between trials. All statistical analysis was performed using Graphpad Prism version 5.0 software, and statistical significance was accepted at the $P<0.05$ level. All data is presented as means ± standard deviation (SD).

RESULTS

Mean pre-exercise pH was higher following ingestion of NaHCO3 than the placebo (NaHCO3: 7.49±0.03 vs. placebo: 7.46±0.03) ($P<0.05$), and six of the seven participants recorded higher pre-exercise pH values following NaCHO3.

Trial duration was longer during the NaHCO3 trial than during the placebo trial (1663 ± 483s vs. 2100±392s) ($P<0.05$) (Figure 1). However, power output was similar throughout both trials and no significant differences were found in any individual epoch (Figure 2).

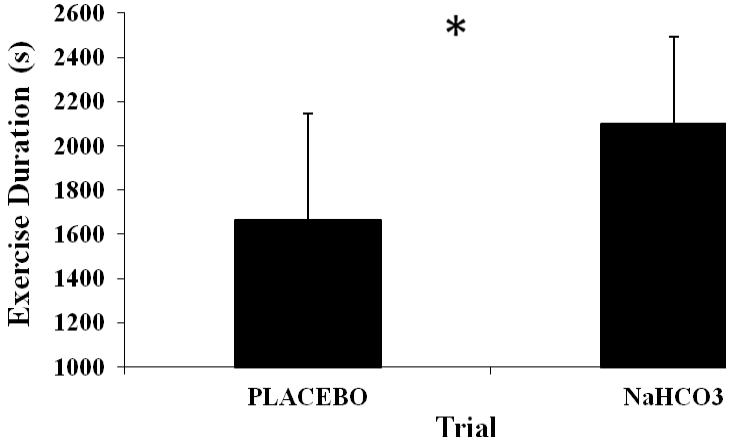

Figure 1. *Exercise duration in NaHCO3 and placebo trials *Significant difference between trials (P<0.05)*

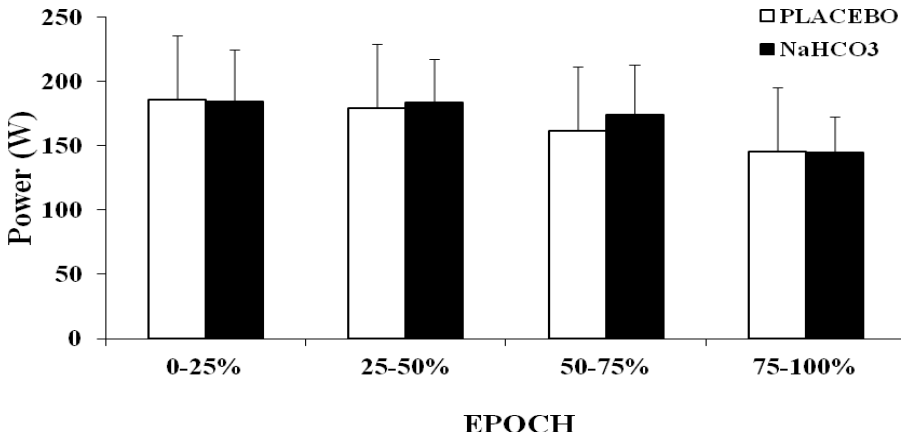

Figure 2. *Mean power output in each individual 25% epoch*

EMG amplitude was significantly lower in the 50-75% epoch in the NaHCO3 trial (NaHCO3: 26.6±10.5%MVC vs. placebo: 33.9±12.7%MVC) (P<0.05). Although the differences between trials did not achieve statistical significance, all individual participants displayed higher EMG activity during their placebo trials in all but the first 25% of exercise (Figure 3).

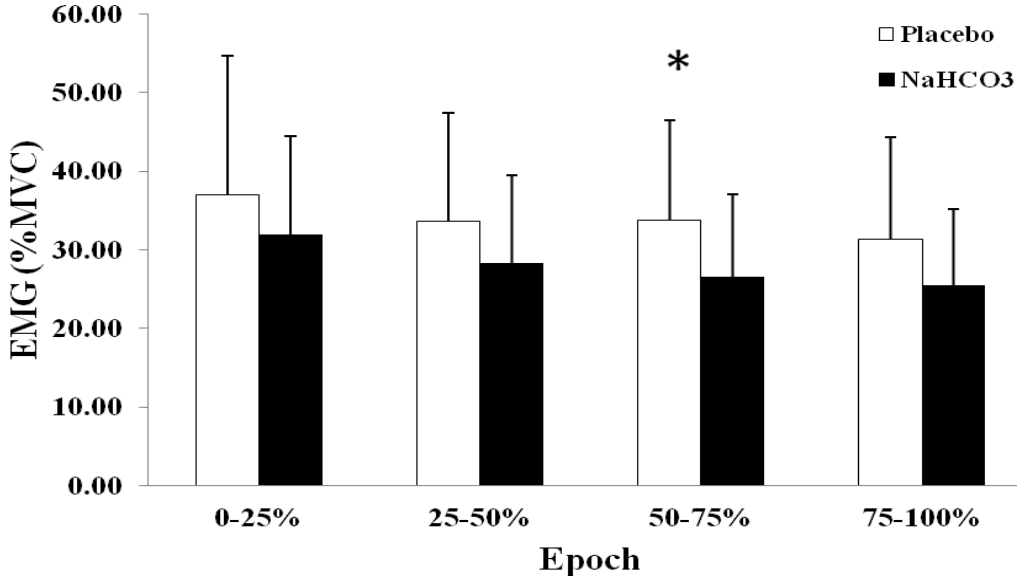

Figure 3. *Mean electromyographic recorded throughout NaHCO3 and placebo trials *Significant difference between trials (P<0.05)*

Heart rate increased progressively throughout both trials and there were no significant differences observed during any individual epoch. Recorded values increased from (NaHCO3: 143±12 vs. placebo: 138±8.4 bpm) in the first 25% to (NaHCO3: 154±6 vs. placebo: 151±6 bpm) in the final 25%.

Post-exercise blood parameters were similar following both NaHCO3 and placebo trials. Post exercise pH values were 7.5+0.04 and 7.5+0.03, whilst blood lactate concentrations were 7.8±4.5 and 5.3±2.2 mmol.l-1 after NaHCO3 and placebo trials respectively.

DISCUSSION

Pre-exercise pH was higher following NaHCO3 than following ingestion of the placebo, indicating that the dosage used in this study was successful in altering pre-exercise physiological status in a manner that should be beneficial to performance in the upcoming exercise task.

Exercise duration at the same level of perceived exertion was increased by 21% after NaHCO3 compared to the placebo. Although pre-exercise pH was higher following NaHCO3, post-exercise values were similar in both trials indicating that the greater total amount of work performed in the NaHCO3 trial was accompanied by a greater absolute change in blood pH. The similar post-exercise values lend support to the findings of Amman (2011) who suggested that the regulation of exercise induced peripheral muscle fatigue is achieved through reference to a feedback loop that uses afferent physiological feedback to the CNS which then varies central motor drive in order to prevent various physiological parameters exceeding "individual critical threshold" values. In the present study, it would seem possible that exercise duration may have been increased in the NaHCO3 trials because the higher pre-exercise pH values would mean that more high intensity work could be performed prior to H+ production associated with glycolytic metabolism resulting in the attainment of the individual critical threshold for pH. This explanation would seem plausible given that the RPE values at which participants exercised in this study was above that at which the lactate threshold has been found to occur during a graded exercise test in both trained and untrained individuals (Demello et al., 1987).

The finding that power output did not significantly differ between trials is particularly interesting. As RPE has been suggested to be partly based on interpretation of afferent physiological feedback and to be the primary regulator of muscular work rate during self paced exercise (Tucker, 2009), it may have been expected that power output would have been higher during NaHCO3 trials. Because NaHCO3 ingestion was successful in altering physiological status it would therefore have also altered the nature of the afferent feedback to the CNS. Based on the central governor model of exercise regulation (Noakes, 2012), it would therefore be predicted that the CNS would increase efferent neural drive (resulting in a higher muscular work rate) during NaHCO3 trials as the afferent feedback would indicate that pH values were further from individual critical threshold values than at the same stage of the placebo trials. However, in the present study this was not found to be the case. Indeed, not only was power output similar in both trials, but NaHCO3 also resulted in a reduction in the measured EMG activity, indicating that effectively the power / EMG ratio was higher in these trials. This finding is in line with the work of Renfree et al. (2012) who reported that EMG activity was higher during the slower of two self paced 20km cycle time trials. The authors proposed that the higher neuromuscular activity despite a lower power output in the slower trials was evidence for neuromuscular compensation for a sub-optimal peripheral physiological status. This concept of neuromuscular compensation had previously been described by Bundle et al. (2006) who suggested that compensatory neuromuscular activity occurs in response to impaired muscle contractile function. Therefore, in the present study the finding of a higher EMG activity during the placebo trials may be explained by suggesting that due to a relatively inferior peripheral physiological status with regards to the demands of the exercise task, a greater degree of neuromuscular activity was required in order to produce the same absolute muscular work rate. However, it is important to emphasise that in both trials, EMG activity tracked power output with a continual decline from the start to the termination of exercise, indicating that work rate was regulated by central rather than peripheral mechanisms. In both the present study and the study by Swart et al. (2009), measured EMG activity did not exceed 45% of the recorded during a pre-exercise MVC.

As muscular power output did not differ between trials despite differences in pre-exercise physiological status, it may be suggested that RPE was based on perception of absolute workload rather than the nature of afferent physiological feedback. These results suggest that manipulation of peripheral physiological status acts in a different manner to stimulation of the CNS. Roelands et al. (2008) have previously demonstrated that methylphenidate, a centrally acting stimulant, improved 20km cycle time trial performance in hot environmental conditions. The higher muscular work rates required to achieve faster times were achieved despite no increases in RPE values above those reported in a placebo trial, and also despite significantly higher core temperatures and heart rates. Similarly, Swart et al. (2009) used a mode of exercise similar to that used in the present study to demonstrate that methylphenidate resulted in a 19% increase in power output at the same RPE than during a placebo trial. As in the study by Roelands et al. (2008), this increased power output was accompanied by increases in heart rate, and also increases in VO2 and neuromuscular activity. Although it has been suggested that methylphenidate acts to improve performance by either increasing the fraction of metabolic and cardiorespiratory reserve capacity that can be accessed (Swart et al., 2009), an alternative explanation is that it acts through inhibition of CNS signals (Roelands et al., 2008). This could lead to dissociation between the conscious RPE and peripheral physiological factors. It has been previously demonstrated that cognitive factors influence RPE at the same absolute muscular work rate despite similar values for various physiological parameters (Baden et al., 2004). This may mean that the reason power output is increased at the same level of RPE in trials where the central nervous system is stimulated is because the participants conscious RPE is not an accurate reflection of their peripheral physiological status and they underestimate the true relative intensity of the muscular work rate. Alternatively, methylphenidate has been demonstrated to increase motivation (Kollins, 1998), and Hall et al. (2005) have demonstrated that individuals displaying high levels of motivation underestimate RPE at submaximal exercise intensities.

We suggest that differing levels of neuromuscular activity during trials in our study indicates that muscular recruitment was regulated in order to achieve the required sensation of effort. As peripheral physiological status relative to the up-coming exercise task was enhanced in the NaHCO3 trials, a smaller degree of activity was required in order to produce the same absolute work rate. Recently the term 'perceived exertion' has been questioned, as the term incorporates both physical sensations of effort and the psychological effort required to perform the task (Smirmaul, 2012). Similarly Hutchinson & Tenenbaum (2006) have argued that it is an oversimplification of the psychophysiological construct to base perceived exertion on a physiological index, and that a single-item measure of effort is insufficient to capture the wide range of sensations experienced during the course of an exercise bout. Both Hampton et al. (2001) and Swart et al. (2012) have also suggested that the sense of effort is a subjective sensation, and recently it has been proposed that the generation of the sense of effort is independent of afferent physiological feedback (Smirmaul, 2012). This suggestion is in congruence with the findings of this study whereby absolute work rates did not differ despite differences in peripheral physiological status.

CONCLUSIONS

This study has demonstrated that manipulation of peripheral physiological status in a manner that would be beneficial to exercise performance increases time to fatigue at a fixed level of RPE, but does not change absolute muscular work rates. Following manipulation of physiological status, similar work rates were achieved despite a reduction in neuromuscular activity. We suggest these findings are in accordance with the proposal by Amman (2011) that development of peripheral muscular fatigue is regulated by the CNS based on achievement of individual critical threshold values for various physiological parameters. We also propose the findings support suggestions that sensations of effort may be based on absolute work rates

rather than the nature of afferent physiological feedback, as neuromuscular activity appears to have been regulated to achieve a similar power output in both NaHCO3 and placebo trials. Finally, we acknowledge that there are limitations to the use of the RPE clamp method in fully elucidating the mechanisms underpinning the regulation of work rate during self paced exercise. Although this and previous studies have demonstrated the role of manipulation of both the CNS and peripheral physiological status during exercise at a fixed level of RPE, the results are difficult to fully interpret until the true determinants of the sensation of 'effort' are fully understood.

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