Research article

# The Effects of Non-Contingent Feedback on the Incidence of Plateau at $\dot{V}O_{2max}$

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#### **Abstract**

The purpose of this study was to examine the effects of noncontingent feedback in the form of heart rate (HR) on the incidence of plateau at  $\dot{VO}_{2max}$ . Ten physically active males (age 24.8 yrs  $\pm$  4.2; mass 81.4  $\pm$  9.0 kg; stature 1.80  $\pm$  0.11 m,  $\dot{V}O$  $_{2\text{max}}$  53.2 ± 5.8 ml·kg<sup>-1</sup>·min<sup>-1</sup>) who were  $\dot{v}_{O\,2\text{max}}$  testing naïve but were cognisant as to the heart rate responses to exercise completed four incremental tests to volitional exhaustion, separated by ~72 h for the determination of  $\dot{V}O_{2max}$  and gas exchange threshold. The first trial served as a familiarisation with the remaining three being experimental conditions where HR was presented in a screen projection as either the actual response (HR-A) or 10 b min<sup>-1</sup> higher than recorded (HR-H) or 10 b min<sup>-1</sup> lower (HR-L). Throughout all trials  $\dot{v}_{O\,2}$  was recorded on a breath-by-breath basis with plateau criteria of  $\leq 50$  ml min<sup>-1</sup>. RESULTS: A significant difference was observed for Δ VO 2 over the final two consecutive 30s sampling periods between HR-A, both HR-L and HR-H (p = 0.049) and for the incidence of plateau response between condition (p = 0.021). An additional significant difference was observed for sub-maximal  $\Delta \dot{V}O_2$ responses between HR-A and HR-H (p = 0.049) and HR-A and HR-L (p = 0.006). Non-significant differences were observed for all other criteria. These data indicate that when presented with non-contingent feedback in the form of HR, that the perceptually orientated pacing schema becomes disrupted promoting a sparing of the finite anaerobic capacity to compensate for the imbalance between the afferent signal and perception of

**Key words:** Pacing, incremental exercise,  $\dot{VO}_{2max}$ ,  $\dot{VO}_{2}$ -plateau

# Introduction

The traditionally accepted primary criterion for determination of  $\dot{VO}_{2max}$  is the 'levelling out' in  $\dot{VO}_2$  prior to the point of volitional exhaustion in spite of a continued increase in exercise intensity, the  $\dot{v}_{O_2}$ -plateau. The physiological rationale for the manifestation of the VO<sub>2</sub>-plateau is that as exercise intensity increases an imbalance ensues between the delivery of oxygen to the muscle as expressed by the cardiac output  $(\dot{Q})$  and the ability to extract oxygen at the muscle as represented by the arterio-venous oxygen difference (a-vO<sub>2dif</sub>) (Wagner, 2000). The role of HR in the manifestation of both  $\dot{Q}_{\,max}$  and  $\,\dot{V}O_{\,2max}$  is well defined (Calbert et al., 2007) displaying a linear-like response as a function of exercise intensity, as a consequence of an ensuing imbalance between parasympathetic and sympathetic nervous activity (Persson, 1996), with the former exhibiting a down regulation and the

latter being up-regulated.

Recent works (Gordon, 2011; Hawkins, 2007) have established that the expression of the  $\dot{V}O_2$ -plateau is a function of the finite anaerobic capacity and the ability to regulate anaerobic substrate metabolism and the concomitant recruitment of less metabolically efficient type II muscle fibres. When the anaerobic capacity was expressed through the surrogate measure of the maximally accumulated oxygen deficit an inverse relationship was witnessed with  $\Delta \dot{V}O_2$  during the final 60 s of a  $\dot{V}O_{2max}$ trial. These data were later confirmed through both the use of prior-priming in the heavy domain (Gordon 2012) and acute reductions in blood volume (~450 ml) (Gordon 2013) suggesting that when the availability of the finite anaerobic capacity was promoted there was an increased incidence of plateaus at VO 2max. Thus the significance and implications of identifying a plateau in VO 2 is apparent, manifestation of such a response represents the primary criteria in establishing a VO <sub>2max</sub> has been achieved.

During exercise which is classed as 'closed-loop' in design it is recognised that the individual will adopt a pacing strategy in order to optimise performance as a means of maximising substrate metabolism and compensating for the artefacts of fatigue (Stone et al 2012, Scruton et al., 2015). A proposed model (St Clair Gibson et al., 2006; Stone et al., 2012; Tucker, 2009) reflecting pace (exercise intensity) is implemented through efferent homeostatic-orientated responses is modulated through afferent feedback systems which are both physiological and psychological in nature. The modulation in pace is thus a product of a perceptually mediated algorithm which is continually compared to a sub-conscious template derived from exposure to previous exercise challenges. Thus the 'template' is based upon the associated sensations of pain, fatigue and the expectations for the duration of the exercise challenge. While, the modulations of pace that occur during exercise account for the need to preserve the finite anaerobic capacity and thereby prevent a catastrophic depletion in associated substrates and accumulation of associated metabolites (Foster et al., 2004). In contrast, open-looped exercise is defined by the lack of an anchor against which perception of effort is regulated, such as known duration or time (Jones et al., 2013). Thus under these conditions the perception of effort remains stable, based on the experience of the participant to judge an exercise intensity that can be tolerated (Tucker, 2009)

 $\dot{VO}_{2max}$  testing conducted using motorized treadmills or cycle ergometers pose a potential contradiction to the pacing schema. Firstly during such an exercise chal-

lenge aside from the participant altering stride rate and length when running or cadence when cycling there is little means of modulating pace, given that exercise intensity is a function of the imposed externally applied resistance. Secondly, unlike time-trial (TT) conditions a VO <sub>2max</sub> test is defined as being open-looped in nature as the end time is not known and the test is only terminated when the participant reaches volitional exhaustion. However recent work (Gordon, 2015) has established that a pacing affect ensues across a series of four repeat VO 2max trials over a 2-week period. In a group of VO 2max testing naïve participants it was suggested that a metabolically orientated pacing strategy was present as highlighted by significant increases in incidence of plateau at VO 2max from 20% in trial 1 to 70% in trial 4, together with an increase in RPE from 17.7  $\pm$  1.3 in trial 1 to 19.0  $\pm$  1.4 in trial 4 despite no change in exercise time to volitional exhaustion, VO 2max or onset of gas exchange threshold (GET) representing ventilatory threshold 2 (VT2). It was concluded that a closed loop condition was developed, firstly by informing the participants of the total number of trials to be completed and secondly by completing the initial trial and thereby establishing the perceptual pacing template for trials 2-4. Consequently it was postulated that plateau incidence was highest in the final trial as the anaerobic capacity, which represents the augmentation of anaerobic substrate metabolism had been preserved across trials 1-3 and the fact that there was to be no fifth trial meant that there was little need to preserve the limited anaerobic capacity.

The pacing paradigm has been challenged through the application of non-contingent feedback in the form of auditory, visual and extrinsic cues as a means of disturbing the pacing algorithm. It is argued that the provision of contingent visual feedback can foster an enhanced relationship between perceived and actual performance whilst under non-contingent (deception) conditions serves to magnify a discrepancy (Morton et al., 2009; Micklewright et al., 2010; Mauger et al., 2011, Ness and Patton, 1979). Indeed it has been show that during simulated TT's, performance was enhanced by the provision of noncontingent feedback (visual representation of past performance) through increased mean power output and heightened RPE which were associated with an increased energy yield from anaerobic energy sources. Alternatively during a series of repeat 4000m cycling time-trials recent work (Mauger et al., 2011) showed that performance was optimized when exposed to accurate feedback in the form of an animated avatar as opposed to the non-contingent feedback. However to date there is no data addressing the effects of non-contingent feedback on the outcome of a VO <sub>2max</sub> test. Thus given the notion that pacing and effort regulation aim to prevent a catastrophic depletion of the finite anaerobic capacity and that the plateau at VO 2max has been attributed to the size of the finite anaerobic capacity the a-priori hypothesis was formed. We contend that in the presence of non-contingent feedback there would be an absence of VO<sub>2</sub>-plateau formation in order to prevent a depletion of the anaerobic capacity and the onset of premature volitional exhaustion. Therefore the purpose of this study was to assess if the application of non-contingent feedback in the form of a visual representation of exercise heart rate was associated with an altered response for the  $\dot{V}O$ <sub>2</sub>-plateau at  $\dot{V}O$ <sub>2max</sub> and associated responses during a series of incremental tests to exhaustion.

## **Methods**

# **Participants**

Following local institutional ethical approval (Faculty Research and Ethics Panel, Anglia Ruskin University University) and having provided written informed consent, n = 10 physically active males (mean  $\pm$  SD: age 24.8  $\pm$  4.2 yrs; mass 81.4  $\pm$  9.0 kg; stature 1.80  $\pm$  0.11 m) volunteered and agreed to participate. The criterion for inclusion in the study was all participants had to be VO <sub>2max</sub> testing naïve and not indicate any contra-indications to exercise such as asthma, hypertension or a recent viral infection. Additionally as the feedback throughout the course of each exercise challenge was to be presented in the form of cardiovascular responses the participants had to show an appreciation of heart rate (HR) responses to exercise. For the duration of the study the participants were instructed to maintain their normal balanced diet, to refrain from strenuous exercise in the 24-h period preceding any test and to report to the laboratory for testing in a hydrated state and having consumed a balanced meal at least 3-h prior to the test.

# Study design

Participants reported for testing on four separate occasions at the same time of day so as to minimise diurnal variation with all trials completed between the hours of 14:00 and 16:00. During all trials at intervals of 30 s the participants were presented with a large visual display using a projection screen mounted in front of the treadmill-ergometer at eye-level showing in numerals the associated HR. The first trial served as a familiarisation (FAM) where the participants were exposed to the actual HR response while the remaining three trials were completed using a single-blind randomised design where HR was presented as either the actual response (HR-A) or through non-contingent feedback of -10 b·min<sup>-1</sup> of the actual response (HR-L) or +10 b·min<sup>-1</sup> of the actual HR response (HR-H). The magnitude of manipulation was selected through pilot testing where participants were asked to identify during exercise if the HR they were presented with was accurate. A meaningful effect was ascertained at 10 b·min<sup>-1</sup> with smaller increments being un-detectable and larger manipulations highlighted as being too obvious. Each trial was separated by a minimum duration of 48-h and a maximum interval of 72-h. Throughout all VO 2 max trials the participants exercised to volitional exhaustion on a pre-calibrated motorised treadmill ergometer (Quasar, HP Cosmos, Nussdorf-Traunstein, Germany).

## Treadmill protocol

For determination of  $\dot{VO}_{2max}$  and GET (VT2) the participants undertook an incremental step test to volitional

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exhaustion with the resistance (gradient) increasing at a rate of 0.5%.30 s<sup>-1</sup> from an initial starting gradient of 0%, with speed remaining constant at 2.7 m·s<sup>-1</sup>. The test was terminated either when the participant reached volitional exhaustion or showed an inability to run at the front of the treadmill which is indicative of an inability to maintain the running cycle. Pulmonary gas exchange responses (VO<sub>2</sub>, VCO<sub>2</sub>, VE and RER) were recorded on a breathby-breath basis throughout all trials using a pre-calibrated metabolic cart (Zan680, nSpire Health, Germany). Heart rate was monitored continuously throughout all trials using a short range telemetric system (Polar 810s, Kempele, Finland) with data averaged on a 30 s epoch. In order to minimise variability within repeat trials the intensity and duration of the warm-up undertaken during FAM was monitored for each participant and then replicated for all subsequent trials.

For all trials the plateau at VO 2 max was established according to previously established methods (Gordon et al., 2011; 2012) of  $\Delta \dot{V}O_2 \leq 50 \text{ ml} \cdot \text{min}^{-1}$  over the final two consecutive 30 s sampling periods, with the isolated final 60 s of breath-by-breath data averaged using a rolling 15 breath average. In order to address the magnitude of the of the VO<sub>2</sub> slope during the final 60 s of the test a least squares regression was applied to the data, with additional quantification of the plateau magnitude determined by calculating the  $\Delta \dot{V}O_2 dif$  (plateau criteria 50 ml min<sup>-1</sup> -  $\Delta \dot{V}O_2$ ). In the absence of a plateau established 'secondary' criteria were applied: RER  $\geq$ 1.15,  $\Delta$ RER  $\geq$ 0.4, a RPE >19 (Gordon et al 2011 ) and a maximal heart rate (HRmax) 205.8-0.685(age) ± 3 b·min<sup>-1</sup> (Gordon et al., 2015). While a  $\Delta \dot{V}O_2$  ( $\leq 50 \text{ ml} \cdot \text{min}^{-1}$ ) and hence a plateau was deemed  $\dot{VO}_2$ max,  $\Delta \dot{VO}_2 > 0$  ml·min <sup>1</sup> but in the presence of the secondary criteria was defined as a maximal effort (VO 2peak). In the absence of both the primary (plateau) and secondary criteria the response was considered to be non-maximal and discarded. GET (VT2) was determined using a mixed methods approach (Gaskill et al., 2001) using the V-slope (Beaver et al., 1986) excess CO<sub>2</sub> method (Volkov et al., 1975) and ventilatory equivalents (Shimizu et al., 1991).

Pulmonary gas exchange variables: Throughout all trials participants breathed through low resistance variableimpedance pneumotachometer for the determination of respiratory flow rates and volumes. Expired gases ( $O_2$ ,  $CO_2$  and  $N_2$ ) were continuously drawn from the mouthpiece assembly unit at a rate of 80 ml·min<sup>-1</sup> through a small bore capillary line of 0.5 mm diameter and 2 m in length. Using custom metabolic cart software the respiratory and gas concentrations were integrated and time aligned. In accordance with other groups and studies the coefficient of variation within our laboratory for  $\dot{V}O_2$ max of athletes of similar age and athletic status is 3.4%.

## Statistical analysis

The incidence of plateau at VO  $_2$ max ( $\Delta$ VO $_2 \leq 50$  ml·min 1) and associated secondary criteria were quantified along with mean and standard deviations for all numerically derived data. As the data were shown to be normally distributed a parametric one-way ANOVA was used to test the hypothesis that plateau incidence and associated indices of a VO 2max test would be affected by noncontingent feedback. Confirmation of plateau manifestation was assessed using a non-parametric binomial test where  $\Delta \dot{V}O_2 \leq 50 \text{ ml} \cdot \text{min}^{-1} = 1 \text{ (plateau) and } \Delta \dot{V}O_2 > 50$ ml<sup>-</sup>min<sup>-1</sup> =0 (no-plateau) across the final 60 s of the incremental test. RPE scores at volitional exhaustion were assessed using a non-parametric Kruskal-Wallis test. For all statistical analyses the alpha level was set at p < 0.05and were completed using SPSS version 19 (SPSS, Chicago, IL, USA).

#### Results

The cardio-respiratory and performance-based responses recorded across the three experimental conditions are presented in Table 1. Individual responses for  $\Delta\,\dot{V}O_2$  across three experimental conditions are presented in Figure 1. Mean gas exchange threshold responses across the three experimental conditions are presented in Figure 2.

Mean VO  $_{2max}$  for all participants across conditions were (FAM)  $53.4 \pm 8.2 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ; (HR-H)  $53.8 \pm 5.9$ 

Table 1. Mean ( $\pm$  SD) for parameters derived during the three  $\dot{V}O_{2max}$  trials.

	HR-A	HR-L	HR-H
VO <sub>2max</sub> (ml <sup>-</sup> kg <sup>-1</sup> -min <sup>-1</sup> )	53.2 (5.8	52.7 (5.4)	53.8 (5.9
VO <sub>2max</sub> (L'min <sup>-1</sup> )	4.31 (.50)	4.28 (.51)	4.4 (.3
$\Delta VO_2$ (ml <sup>-</sup> min <sup>-1</sup> )	37.0 (20.6) <sup>a b</sup>	75.4 (45.7)	80.2 (52.3)
VCO <sub>2max 2max</sub> (ml'kg <sup>-1</sup> ·min <sup>-1</sup> )	60.4 (9.9)	62.1 (7.6)	64.0 (9.7)
HR <sub>max</sub> (b·min <sup>-1</sup> )	186 (8)	185 (5)	186 (5)
$RER_{max}$	1.14 (.15)	1.18 (.06)	1.18 (.10)
RPE	18.7 (1.6)	18.4 (1.4)	18.7 (1.2)
GET (%VO <sub>2max</sub> )	83.1 (3.4)	85.3 (13.7)	83.5 (7.4
GET (W)	225.5 (71.0)	218.5 (59.9)	223.5 (66.6
GET- VO <sub>2</sub> (ml·kg <sup>-1</sup> ·min <sup>-1</sup> )	44.2 (1.8)	45.0 (6.4)	44.9 (3.5
TET (s)	615.0 (95.6)	610.0 (106.0)	624.8 (98.8
$VO_{2max}$ -ET (s)	42.0 (29.8)	42.0 (38.6)	51.0 (41.9
Onset- $VO_{2max}$ (s)	573.0 (95.4)	568.0 (81.3)	574.0 (106.0)
GET (s)	311.0 (142.0)	297.0 (120.0)	307.0 (133.0)
GET- $VO_{2max}(s)$	262.0 (72.4)	271.0 (76.2)	267.0 (81.8)

HR-A= actual heart rate displayed, HR-L= heart rate presented 10 b min<sup>-1</sup> lower than recorded and HR-H= heart rate presented 10 b min<sup>-1</sup> higher than recorded. Where a= significant difference between HR-A and HR-L and b= significant difference between HR-A and HR-H (p < 0.05).

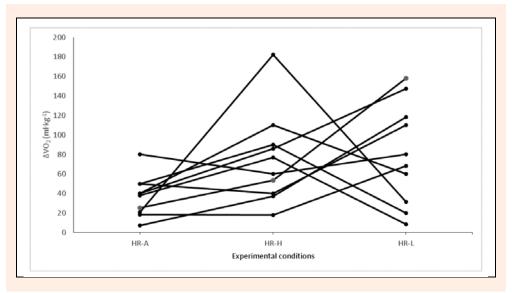


Figure 1. Individual response for  $\Delta \dot{V}O_2$  across three experimental conditions. HR-A = actual representation of the recorded heart rate, HR-L = visual representation of the heart rate – 10 b min<sup>-1</sup> and HR-H = visual representation heart rate + 10 b min<sup>-1</sup>.

 $ml \cdot kg^{-1} \cdot min^{-1}$ ; (HR-L) 52.7 ± 5.4  $ml \cdot kg^{-1} \cdot min^{-1}$ ; (HR-A)  $53.2 \pm 5.8 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ . There was a non-significant difference for  $\dot{V}O$  <sub>2max</sub> across condition (p > 0.05). Nonsignificant differences were also observed for RPE, HR<sub>max</sub>, GET, GET (%  $\dot{V}O_{2max}$ ), RER<sub>max</sub> and  $\Delta$ RER between all conditions (p > 0.05). Using the plateau criteria of  $\Delta \dot{V}O_2 < 50 \text{ ml} \cdot \text{min}^{-1}$  across the final two 30 s sampling periods incidence rates were (FAM) (70%), (HR-A) (90%), (HR-H) (30%) and (HR-L) (30%). Using the nonparametric binomial test a significant difference was observed for plateau incidence between and HR-A, HR-H and HR-L (p = 0.021). Additionally the  $\Delta \dot{V}O_2$  response during the final 60 s of the incremental tests was shown to be significantly different between both HR-L and HR-H and HR-A (p = 0.050). There was however a nonsignificant difference for Δ VO 2 between FAM and HR-A (p = 0.484). The  $\Delta \dot{V}O_2$  responses were then compared directly to the actual criterion value (50 ml·min<sup>-1</sup>) to get a reflection on the actual slope size, which equated to -13.0  $\pm$  20.6 ml min  $^{-1}$  (HR-A), 30.2  $\pm$  52.3 (HR-L) and 25.4  $\pm$ 26.7 ml·min<sup>-1</sup> (HR-H), with significant differences observed between HR-A and HR-H (p = 0.014) and HR-A and HR-L (p = 0.013). When comparing the  $\Delta \dot{V}CO_2$ during the last 60 s of the tests, response rates of 95.5  $\pm$ 83.8,  $116.1 \pm 134.0$  and  $128.3 \pm 81.7$  ml min<sup>-1</sup> were observed for HR-A, HR-H and HR-L respectively (p > 0.05). The O<sub>2</sub> pulse was determined across quartiles of the exercise challenge to reflect changes in O2 extraction at the muscle. Where during the  $1^{st}$  1/4 responses were 21.2  $\pm$ 78.7 (HR-A),  $21.3 \pm 80.0$  HR-L) and  $21.1 \pm 78.8$  ml·beat<sup>-1</sup> (p > 0.05). During the  $2^{nd}$  1/4 the HR-A response was 23.7  $\pm$  49.3, compared to 20.9  $\pm$  41.5 (HR-L) and 20.6  $\pm$  34.4 ml'beat<sup>-1</sup> (HR-H) (p > 0.05). Similarly a non-significant difference (p > 0.05) was observed during the  $3^{r\bar{d}}$  and  $4^{th}$ quartiles where responses were 23.3  $\pm$  106.1, 22.1  $\pm$  71.9 ml'beat<sup>-1</sup> (HR-A),  $20.3 \pm 41.4$ ,  $20.1 \pm 37.5$  ml'beat<sup>-1</sup> (HR-L) and  $19.9 \pm 30.3$ ,  $19.5 \pm 29.2$  ml·beat<sup>-1</sup> (HR-A). Additionally when the response within condition was expressed non-significant differences were observed throughout (p > 0.05). When considering the time orientated responses non-significant differences were observed for total exercise time (TET),  $\dot{V}O_{2max}$ -ET, onset- $\dot{V}O_{2max}$  and GET (p > 0.05). Additionally an analysis was made of the magnitude of the  $\dot{V}O_2$  response across the incremental tests, with the outcomes based on data collected every 30s from 90s post-test onset to the onset of the GET. Significant differences were observed between HR-A (1.80  $\pm$  0.26 ml·kg<sup>-1</sup>·min<sup>-1</sup>) and HR-H (1.36  $\pm$  0.57 ml·kg<sup>-1</sup>·min<sup>-1</sup>) (p = 0.049), HR-A and HR-L (1.25  $\pm$  0.36 ml·kg<sup>-1</sup>·min<sup>-1</sup>) (p = 0.006), with a non-significant difference observed between HR-H and HR-L. A trial order effect was assessed for all parameters and was shown to be non-significant (p > 0.05).

# **Discussion**

The purpose of this study was to ascertain if the provision of non-contingent feedback in the form of manipulated HR could influence the outcome of a graded exercise test to exhaustion with particular reference to the manifestation of the VO<sub>2</sub>-plateau. Ana-priori hypothesis was established based on the notion that during an incremental stress test to exhaustion there is a need to regulate substrate metabolism in order to prevent an early onset of fatigue associated with a depletion of the finite anaerobic capacity. It was contended that in the presence of noncontingent feedback there would be a decrease in the manifestation of a VO<sub>2</sub>-plateau suggesting the anaerobic capacity was not fully taxed due to the pacing schema being out of alignment with the previously established effort regulation template (Gordon et al., 2015, Foster et al., 2004). The reported findings lend support to this contention showing an increased plateau incidence and smaller  $\Delta \dot{V}O_2$  during the final 60s of the test for HR-A compared to HR-L and HR-H. These findings were supported by the significant difference of  $\Delta VO_{2dif}$  between

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HR-A and both HR-L and HR-H, despite no difference in any other parameter including  $VO_{2max}$ , TET and GET. These findings are though in contrast to previously reported outcomes (Albertus et al., 2005; Faulkner et al., 2010) which highlighted that when the external cue was manipulated, in these instances through inaccurate presentation of distance, no discernible difference was observed in exercise time to exhaustion during TT efforts.

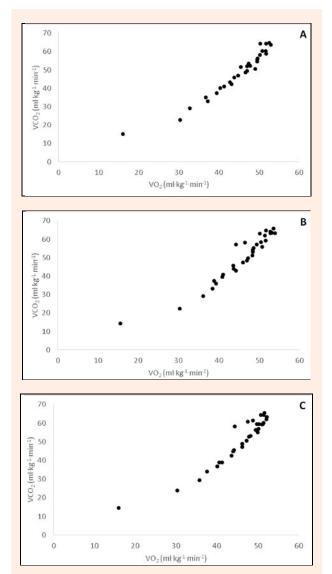


Figure 2. Mean gas exchange threshold responses across the three experimental conditions, Panel A = HR-A, Panel B = HR-L, Panel C = HR-H. GET calculated as 83.1% (HR-A), 85.4% (HR-L) and 83.5% (HR-H).

The crucial component and difference in the current study in the misalignment of the pacing schema is the presentation of HR as an external cue, with previous works highlighting that perceived exertion is a function of afferent signals such as ventilation, oxygen uptake, heart rate and more peripherally orientated factors such as lactic acid concentration, skin temperature and substrate depletion (Albertus et al., 2005, Mihevic, 1983). Heart rate has been proposed as a key mediator in the perception of effort due to its associative effect with the individuals'

rating of perceived exertion (Borg, 1967) and hence has a significant role in the development of the schema. The notion of pacing during exercise is based on the integration of feedback which develops the subconscious RPE and anticipatory forecasting which itself is a function of prior experience and afferent inputs (Tucker, 2009). At the onset of exercise and throughout the course of an exercise challenge the afferent inputs are matched against the RPE yielding an effort outcome. In the case of the current work this matching of input to output is disrupted causing a break in the interpretation of the physiological response, in this instance HR. Under conditions of a VO <sub>2max</sub> test there would be an increased level of sympathetic activity driving up o through an increased HR and SV. However when exposed to the non-contingent feedback the RPE dissociates from the HR creating a feedback loop, heightening a perceived awareness of cardiovascular response at the expense of the peripherally mediated sensations of pain and fatigue. Thus although the subconscious perception is manifest the bio-feedback derived from the visual representation of HR would appear to supersede this and circumvent the process. This implies that unlike in previous work where attempts have been made to influence the pacing schema through the visual representation of distance or time (Albertus et al., 2005; Faulkner et al., 2010; Morton, 2009) that a representation of a physiological component potentially evokes a potent stimulus for feedback than more conventional analogue variables.

The concept of effort regulation and the pacing schema are based on the notion of exercise being closedloop in nature, with a known end-point. A criticism of conventional VO 2max tests such as that adopted in this study is that they do not reflect everyday scenarios, with the test being to volitional exhaustion the time-domain component of the effort regulation schema is rendered redundant potentially yielding conflicting outcomes. However, in a recent study (Gordon et al., 2015) during which VO 2max naive participants completed a series of 4 repeat VO 2max trials it was demonstrated that the trials became closed-loop, firstly because the participants were informed of the total number of trials, thus establishing a total endpoint for effort regulation and also, once trial-1 was completed the perceptual template for effort regulation under these unique conditions had been established. The outcome of this study was that plateau incidence rose from just 20% for trial-1 with an associated  $\Delta \dot{V}O_2$  1.16  $\pm$ 1.94 ml kg  $^{-1}$  min  $^{-1}$  to 70% in trial-4 and a  $\Delta\dot{V}O_2$  0.86  $\pm$  1.33 ml kg  $^{-1}$  min  $^{-1}$  . In the current study the participants were VO 2max testing naive, but undertook a familiarisation trial during which they were exposed to full experimental conditions, including the visualisation of heart rate. The role of the familiarisation trial in the context of the effort-regulation paradigm is paramount for this would expose the participants to the sensations of pain and fatigue thus establishing a perceptually orientated template for these exercise conditions (Micklewright et al., 2010). This assertion is supported in the current study where the VO<sub>2</sub> plateau incidence was 70% for FAM and

90% for HR-A associated with respective  $\Delta \dot{\text{VO}}_2$  responses of 44.1  $\pm$  19.1 and 37.0  $\pm$  20.6 ml min<sup>-1</sup>. Additionally the external cues from the pacing schema would be established in the presentation of the heart rate, providing a bio-feedback mechanism for subsequent trials. Indeed as stated in previous works (Billaut et al., 2011; St Clair Gibson et al., 2006) the sub-conscious regulation of effort which reflects the interplay of the biological and cognitive components would be established at the onset of exercise, based on the previous experience and perceptions to the exercise challenge.

The plateau at VO 2max has previously been shown to be a function of the finite anaerobic capacity and the ability to recruit less efficient type-II muscle fibres (Hawkins, 2007, Gordon et al., 2011), with size of the capacity dependent upon the availability of both the high energy phosphates (ATP-PCr) and substrate glycolysis. Recent support for this model was provided, showing that following donation of ~450mm<sup>3</sup> of blood there was no change in the incidence of plateau at  $\dot{V}O_{2max}$  despite a 5.6% decrease in  $\dot{V}O_{2max}$  consequent to a 9.4% decrease in haemoglobin concentration, highlighting the disparity between the  $\dot{V}O_2$  plateau and  $O_2$  supply (Gordon et al., 2013). Coincident to this, the pacing paradigm is also dependent on the exposure of the cell to the artefacts of substrate metabolism (H<sup>+</sup>, free Pi and ADP) which show a proportional increase to the declining PCr concentration (Green and Patla 1992) for exercise in the heavy and severe domains beyond the GET. Indeed Jones et al. (2008) demonstrated that for exercise accomplished in excess of the 'Critical power' the tolerance of the workload is a function of both the rate of declining substrates and accompanying accumulation of H<sup>+</sup> and free Pi. Thus it is proposed for the current study, that when the feedback loop is disrupted in the presence of the noncontingent feedback, a 'threat' is presented to the schema which invokes a preservation of the finite anaerobic capacity, as reflected by both the increased  $\Delta \dot{V}O_2$  during the final 60s of the incremental test when compared to a baseline criterion value and the decreased sub-maximal  $\Delta$ VO<sub>2</sub> response from exercise onset to GET. Under these conditions the provision of the non-contingent feedback renders the exercise challenge 'open-looped' as the clarification of the end-point is now less clear in the context of the pacing schema (Foster et al., 2004, Stone et al., 2012).

# **Conclusion**

This study has demonstrated that when presented with non-contingent feedback in the form of heart rate that there is a sparing of the finite anaerobic capacity as evidenced by decreased plateau responses at  $\dot{VO}_{2max}$ . It is proposed that under these non-contingent conditions the sensitivity of the afferent feedback is disrupted resulting in a mismatching between the physiological and perceptually orientated cues. Under these conditions the exercise condition would appear to resemble an open-looped condition where the pacing schema is less robust as reflected by the ameliorated sub-maximal  $\Delta \dot{VO}_2$  response coupled with the rising  $\dot{VO}_2$  to the point of volitional exhaustion.

Future work should address the acute physiological response to this biological non-contingent feedback through inspection of the components of the Fick equation during exercise and whether such a response is manifest in individuals who are both  $\dot{V}O_{2max}$  and heart rate response to exercise naïve.

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# **Key points**

- The manifestation of the plateau at VO<sub>2max</sub> is disrupted (lower incidence rates) when participants are exposed to non-contingent feedback in the form of heart rate.
- Non-contingent feedback in the form of heart rate does not affect the VO<sub>2max</sub> score or associated cardio-respiratory parameters.
- Given the association between the VO<sub>2</sub>-plateau and the finite anaerobic capacity it is proposed that non-contingent feedback creates a sparing of this resource due to an imbalance between the perception of effort and the bio-feedback.

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