Title: Evidence for anticipatory pacing strategies during supra-maximal exercise lasting longer than 30

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Running title: Pacing strategies in supramaximal exercise

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Abstract

The purpose of this study was to assess whether pacing strategies are adopted during supramaximal exercise bouts lasting longer than 30 s. Eight healthy males performed six Wingate Anaerobic Tests (WAnT). Subjects were informed that they were performing four 30 s WAnT and a 33 s and 36 s WAnT. However, they actually completed two trials of 30, 33 and 36 s each. Temporal feedback in the deception trials was manipulated so that subjects were unaware of the time discrepancy. Power output (PO) was determined from the angular displacement of the flywheel and averaged over 3 s. The peak power (PPI), mean power (MPI) and fatigue (FI) indices were calculated for each trial. Power output was similar for all trials up to 30 s. However, at 36 s the PO was significantly lower in the 36 s deception trial compared to the 36 s informed trial (392 ± 32 W vs 470 ± 88 W) ($p < 0.001$). The MPI was significantly lower in the 36 s trials (714 ± 76 W and 713 ± 78 W) compared to the 30 s trials (745 ± 65 W and 764 ± 82 W) although they were not different at 30 s (764 ± 83 W and 755 ± 79 W). The significant reduction in FI was greatest in the 36 s deception trial. In conclusion, the significant reduction in PO in the last six seconds of the 36 second deception trial, but not in the 36 second informed trial, indicates the presence of a pre-programmed 30 second "end point" based on the anticipated exercise duration from previous experience. Furthermore the similarity in pacing strategy in all informed trials suggests that the pacing strategy is centrally regulated.

Introduction

For the last 30 years researchers have used the Wingate Anaerobic Test (WAnT), or a variant thereof, as a means of quantifying anaerobic performance in absolute terms of power output which is then related to the maximum capacities of specific metabolic pathways. Traditionally the WAnT uses a 30-second cycle test protocol, performed against a constant force, in which subjects are instructed to cycle as hard as they can for the duration of the test. The three indices commonly derived from the WAnT are: i) peak power index (PPI), the highest power output obtained during the trial: ii) mean power index (MPI), the average power sustained over the duration of the trial; and iii) fatigue index (FI), the drop-off in power between the PPI and the final power output reading.

It was thought that during the initial 5 – 10 seconds of exercise energy is derived solely from alactic, phosphogenic pathways after which the metabolic fuel for the rest of the exercise bout is obtained from anaerobic glycolysis. Subsequent research has shown that, contrary to this belief, lactate accumulation begins within the first 10 seconds of supramaximal exercise (Jacobs et al, 1983; Bogdanis et al, 1996) and that mitochondrial oxidative ATP synthesis increases almost immediately at the onset of exercise (Nioka et al, 1998).

Despite this aerobic component of undetermined magnitude, the WAnT is considered to be largely dependant on anaerobic metabolism (Serresse et al, 1988) and the associated fatigue has been mainly attributed to the fall in pH (Cherry et al, 1997) that changes potassium concentration (Brody et al, 1991) and/or interferes with calcium release (Allen et al, 1992), impeding the ability of the muscle to contract (Mortimer et al, 1970; di Prampero et al, 1981; Arendt-Nielsen et al, 1989)

and resulting in selective fatiguing of the fast twitch fibres (Beelen and Sargeant, 1991; Hautier et al, 1998).

The WAnT may not have been designed to study muscle contractility or fatigue (Bar-Or, 1987) but it has been suggested (Green et al, 1995) that observing the effects of such all out efforts could provide insight into physiological and neuromuscular capabilities. While this peripherally mediated fatigue might account for some of the drop-off in power output observed during the WAnT, electromygraphic (EMG) signal recordings have revealed a decline in iEMG activity towards the end of the test, which suggests the occurrence of central fatigue (Vandewalle et al, 1991; Hunter et al, 2003). Indeed, the fact that the fall in power in the Wingate test is explained by the falling cadence (Katch and Katch, 1973) can really only be explained by central, neural regulation since it is not clear how peripheral fatigue alone can produce the necessary changes in motor recruitment necessary to cause the progressive and marked reduction in cadence. Although the 30 second duration of the WAnT is generally considered optimal (Margaria et al, 1969; Green, 1995), a number of authors have suggested that it may not be sufficiently long to measure the total anaerobic capacity (Vandewalle et al, 1987; Bacharach and von Duvillard, 1995; Calbet et al, 1997). The difficulty in extending the protocol beyond 30 seconds is the adoption of pacing strategies by subjects to ensure that they are able to complete the test (Bar-Or, 1987; Bacharach and von Duvillard, 1995). To prevent the adoption of such a conscious pacing strategy, it has been suggested that subjects should not be made aware of the elapsed time (Katch and Weltman, 1979). More recently the concept that subjects subconsciously pace

themselves during exercise on the basis of prior experience has been developed (Ulmer, 1996; St Clair Gibson et al, 2001).

There are a number of investigations into the competitive pacing strategies adopted during events lasting 1 - 6 minutes (Hagerman, 1984; Foster et al, 1993; Foster et al, 1994) and the optimal pacing strategies over these periods (Ingen Schenau et al, 1994). However, to the authors' knowledge, no study has yet evaluated the possible presence of pacing during bouts of supra-maximal exercise. The existence of a pacing strategy would suggest that substrate depletion or metabolic accumulation might not be the immediate cause of the power output profile during a WAnT.

Accordingly the aim of this study was to determine whether deception and temporal manipulation could identify whether a subconscious pacing strategy exists during a standard and modified WanT lasting between about 30 and 36 seconds.

Methods

Eight healthy males volunteered for this study. The age and body mass (mean ± SD) were 22 ± 2.5 years and 76.5 ± 6.9 kg, respectively. All subjects were physically active and each signed an informed consent before the study. The study was approved by the Research and Ethics Committee of the University of Cape Town Faculty of Health Sciences.

Subjects performed six Wingate Anaerobic Tests on a Monarck friction-braked cycle ergometer (814E). Trials were held one week apart at the same time of day (Hill and Smith, 1991; Reilly and Down, 1992) and the order of the trials was randomised. One week prior to the start of the trial subjects performed a habituation Wingate test.

Standardisation procedure

A load of 0.09 $kg.kg^{-1}$ body mass (Dotan and Bar-Or, 1983; Patton et al, 1985) was administered by placing calibrated weights on a weight pan, which exerted a resistive force against the flywheel, at the onset of exercise. Workloads were set to the nearest 0.1 kg. Saddle height was recorded on the first visit to the laboratory and the same height was used in all subsequent trials (Burke, 1986). Subjects were strapped to the saddle to avoid standing on the pedals (Vandewalle et al, 1987). Pedal crank length was constant (Inbar et al, 1983) and all subjects used cleated cycling shoes (LaVoie et al, 1984). The warm up was standardised to 2 minutes unloaded pedalling (Inbar and Bar-Or, 1975; Hawley et al, 1989). Before the start of each trial subjects were instructed to pedal as fast as possible and to attempt to maintain that cadence velocity for the duration of the test. All tests were conducted from a standing start (Kaczkowski et al, 1982). The same investigators offered

encouragement throughout the trial to minimise motivational differences (Bar-Or, 1987). Time feedback during the trial was provided on an LCD screen that displayed elapsed time.

Subjects were informed that they were completing four 30 second, one 33 second and one 36 second trial. However, they actually completed two trials of 30 (30_1 and 30_2), 33 (33_D and 33_I) and 36 (36_D and 36_I) seconds each. The suffixes D and I denote the deception trial and informed trial, respectively. A computer application altered the rate at which the LCD counter displayed second intervals. Therefore during the two deception trials the display time showed a 30 second count for the 33 and 36 second duration of the respective tests.

Data capture

The angular velocity of the flywheel was calculated every 36° from sensors attached to the flywheel. Pedal revolutions per minute were then derived from the formula:

$$Rpm = \frac{pedal\_revolutions}{flywheel\_angular\_displacement} \times \frac{flywheel\_angular\_displacement}{time}$$

Data capture occurred every 0.5 seconds and the power generated on the Monarch bicycle was calculated using the following formula:

$$Power = load\_mass \times rpm$$

Indices

The PPI was calculated as the highest average power over any 3 second period (Bar-Or, 1987), MPI as the average power over the entire trial and FI as the fractional decrease in power during the test (McCartney et al, 1983):

$$FI = \frac{PPI - FP}{PPI}$$

where FP is the power averaged over the final 3 seconds of the test.

Statistical analysis

Power output data for all calculations were averaged over 3 seconds. Calculations for MPI and FI during the modified trials were determined over the entire duration and over the first 30 s. An analysis of variance was used to assess differences between and within the trials. Once main effects were identified individual differences between the means were located using Tukey's HSD *post hoc* procedure. Significance was accepted at $P < 0.05$. All data were expressed as mean ± standard deviation (S.D.).

Results

On completion of the study the subjects were unable to identify on which occasions they performed the deception trials.

Power output

Power output was uncorrected for inertia. There were no differences in power output up to 30 s between any of the trials (Figure 1). However in the 36 s trials, the power output was significantly lower at 36 s in the deception trial compared with the informed trial (392 ± 32 W vs. 470 ± 88 W; $p < 0.001$).

Peak power index

There were no significant differences in the magnitude of PPI between any of the trials (30_1: 1055 ± 84 W; 30_2: 1076 ± 97 W; 33_D: 1061 ± 98 W; 33_I: 1047 ± 107 W; 36_D: 1056 ± 83 W; 36_I: 1055 ± 74 W) (Figure 2a) nor did the time at which peak power occurred differ (30_1: 5.8 ± 1.0 s; 30_2: 5.3 ± 1.5 s; 33_D: 5.4 ± 1.1 s; 33_I: 4.9 ± 2.6 s; 36_D: 4.6 ± 1.0 s; 36_I: 4.7 ± 1.0 s) (Figure 1).

Mean power index

The MPI for the 36 s trials (36_D: 714 ± 76 W; 36_I: 713 ± 78 W) were significantly lower than for either of the control 30 s trials (30_1: 745 ± 65 W; 30_2: 764 ± 82 W)($p < 0.05$), but not different from the 33 s trials (33_D: 748 ± 87 W; 33_I: 734 ± 100 W) (Figure 2b). The MPI for the 36 s trials also decline significantly from their value at 30 s (36_D: 764 ± 83 W; 36_I: 755 ± 79 W) where after there were no differences between any trials.

Fatigue index

The FI for the 36 s deception trial (63 ± 4.2%) was significantly greater than all other trials (30_1: 50 ± 7%; 30_2: 49 ± 3%; 33_I: 51 ± 3%)($p < 0.05$) except the 36 s informed trial (56 ± 7%) and the 33 s deception trial (54 ± 3%) (Figure 1c). There was no difference in the FI at 30 s between any of the trials. The FI in the 36 s deception trial declined significantly from 30 s to 36 s (50 ± 10% vs 63 ± 4%)($p < 0.001$).

Discussion

The first important finding of the study was that the power output during the first 30 seconds of all trials was similar, regardless of the final duration of the trial. This finding is surprising, if it is believed that muscle energy depletion alone acting peripherally, limits such exercise. Rather it would be predicted that the same amount of chemical energy would allow a faster rate of energy expenditure during the shorter exercise durations. Instead, this finding suggests that power output during the Wingate test is controlled by factors other than purely the magnitude of the skeletal muscle energy reserves and the maximum rate at which such reserves can be depleted.

Indeed the second novel finding of this study was that power output dropped significantly more over the last 3 s of the 36 s deception trial compared with the informed trial. The occurred even though subjects were not able to consciously differentiate between the duration of the longer deception trials and the standard 30 s Wingate trial.

The finding that subjects were able to perform more work when performing exercise, the duration of which they had been reliable informed about than when misinformed, indicates that central neural factors other than peripheral metabolite accumulation, determines the power output profile of the Wingate test.

Indeed, the fall-off in power during the Wingate test has been attributed to the early selective fatiguing of fast twitch skeletal muscle fibres (Beelen and Sargeant, 1991) once the optimum velocity for maximal contractions of slower muscle fibres has been exceeded. But, it has also been shown that the decline in iEMG activity,

accounts for nearly 30 percent of the drop-off in power at the end of supra-maximal exercise (Hunter et al, 2003; Vandewalle et al, 1991). Furthermore, the power output profile of the Wingate test reflects solely the change in the cadence (Katch and Katch, 1973), which must be centrally regulated in the brain. Changes in power output generated by the brain could, however, be in response to peripheral metabolic changes in the active muscles.

Indeed, the presence of a central neural component to pacing and fatigue has long been recognised as a factor influencing power output during supra-maximal exercise. Pilot studies at the Wingate Institute during the development of the Wingate Anaerobic Test showed a propensity for subjects to start at slower cadences in trials that lasted longer than 30 s (Bar-Or, 1987). This tendency was also observed by Katch and Weltman (Katch and Weltman, 1979) who remarked that the duration of the trial should not be revealed to the subjects lest they "pace themselves and not produce an initial all-out effort". In a study on elite Alpine skiers (Bacharach and von Duvillard, 1995), trial subjects were instructed to ease up after they had achieved peak power so that they would be able to maintain more constant effort for the remainder of the 90 s trial, implying that some form of pacing is necessary to complete extended supra-maximal tests.

An even pace has long been regarded as the most efficient strategy to employ during any exercise and this belief appears to be supported by evidence that during an even-paced Wingate trial, blood lactate concentrations are lower, blood pH is higher and recovery is faster, compared with an all-out trial, in which the same total amount of work is performed (Cherry et al, 1997). In contrast Ingen-Schenau et al (Ingen Schenau et al, 1994) argue that in events lasting less than 80 s, the optimal

pacing strategy is all out effort, even if this strategy produces a rapid drop-off in power at the end of the race. They suggested that an even-paced strategy should only be used in exercise of a longer duration. Our data do not address this question.

It has been speculated that muscle pH provides the sensory feedback linking central command of motor unit recruitment and intramuscular metabolism (Kent-Braun, 1999) so that athletes adjust their pace in order to ensure that a critically low pH is not reached even at the end of a maximum effort (Foster et al, 1994). This is supported by evidence of a continuous fall in intramuscular pH from 6.94 to 6.82 between 10 and 20 s in all-out exercise (Bogdanis et al, 1998), mirroring the decline in power output. The fall in power output ensures that the extent of the fall in pH is regulated.

Ulmer (Ulmer, 1996) extended this concept by proposing the existence of a programmer during exercise that regulates work based upon the remaining duration of an activity. In other words the effort required in performing an activity is anticipated prior to the commencement of the activity and the intensity of exertion is regulated according to calculations based on previous experience. This governor acts as a protective regulator of power output to prevent the development of a metabolic crisis that would damage the integrity of the muscle fibres (Sargeant, 1994).

Although there were significant differences in power output at 36 s when the subjects were deceived as compared to the informed trial, no differences were present at the end of the 33 s trials indicating that the modification in afferent control, causing a reduction in power in the last 3 seconds, must occur after 33 s but before 36 s. This is in agreement with the speculation of Hunter et al (Hunter et al,

2003) that 30 s was too short a duration for a change in recruitment strategies based upon feedback from intramuscular metabolism but predicted that the influence of this feedback loop on central motor control would have to come into effect if the exercise was sufficiently prolonged.

Evidence that the extent of muscle recruitment is controlled centrally during prolonged exercise has been presented by St Clair Gibson et al (St Clair Gibson et al, 2001) in a study that revealed that the fall in power output was mirrored by a reduction in the neural drive despite less than 20% of the muscle mass being recruited. They concluded that the early decline in neuromuscular activity during prolonged exercise explained the reduced power output experienced by their subjects.

During the informed trial the subjects prepared themselves for an effort that would last 36 s. During the 36 s deception trial, though, the subjects were only primed for a 30 s effort and despite confirmatory but deceptive evidence in the form of a clock, that was 6 seconds slow, there was still a disparity between their actual and possible power outputs (Figure 1). As the subjects were not consciously aware of the deception, this finding indicates that the deception was detected at a subconscious level but not a conscious level since the reduction in power output over the extra time was not consciously perceived. If the duration of the 36 s trials had been extended beyond 40 s the subjects may have employed a more obvious coping strategy. However, it is unlikely that subjects would have been deceived, that is they would not have realised that a deception was in place, by such a large (33%) discrepancy between the real and simulated time. Accordingly, this type of experiment may not be possible.

Rather we conclude that the subjects did detect the time discrepancies but ignored or filtered the conscious knowledge as incorrect based upon what they had been told and the visual cues from the slow-running clock. However, the findings of the present study suggest that the "lag" phase between afferent input and subconscious awareness of discrepancies between expected and actual time must be between 3 and 6 s during an exercise lasting up to 36 s, that is, approximately 12% of the total duration of the activity. More research is needed to determine the degree to which subjects can accurately assess temporal deceptions during exercise.

As peak power was similar and occurred at a similar time point, it seems that the subjects started out at a set work rate, although whether this is paced and submaximal itself cannot be determined from this study. The similar fatigue profiles over the initial 30 s for all the trials suggest that there was no difference in the pacing strategy employed for the different duration trials. However, the sudden decline in power output of the last 3 s of the 36 s deception trial may provide evidence for a pre-programmed "end point" that is different from the ultimate fatigue point. Once the anticipated end point is reached, the power output declines rapidly. It has been demonstrated that even in maximal sprint intervals, subjects maintain a reserve allowing an increase in power output over the last sprint consequent to an increased neural drive (Kay et al, 2001).

In summary, this study indicates the presence of a pre-programmed "end point" based on the anticipated exercise duration as a result of previous experience in a Wingate test. Furthermore the similarity in pacing strategy in all informed trials regardless of duration suggests that the pacing strategy is centrally regulated and is

independent of the total work to be performed, at least for up to 36 seconds of supramaximal exercise.

Legend to figures

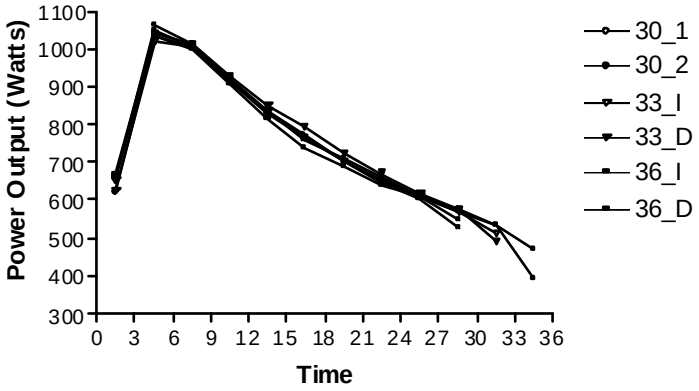
Figure 1 Comparison of power output profile for the two 30, 33 and 36 s Wingate Anaerobic Tests

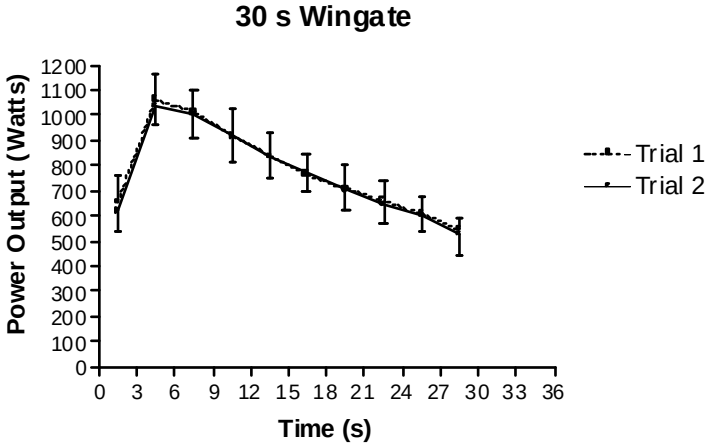
Figure 2 Peak power, mean power and fatigue indices at 30 s and at the completion of the Wingate Anaerobic Tests

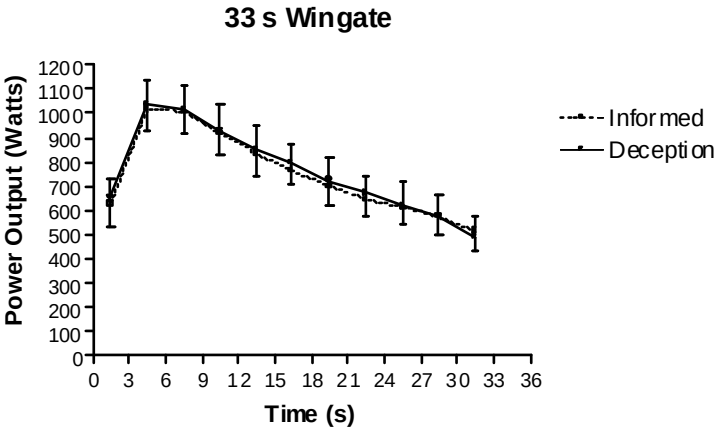
* significantly different from the 36s deception and 36 s informed trial ($p < 0.05$)

λ significantly different from the 36s deception ($p < 0.05$)

Figure 1


Power Output (Watts)
1100
1000
900
800
700
600
500
400
300
0 3 6 9 12 15 18 21 24 27 30 33 36
Time
30_1
30_2
33_I
33_D
36_I
36_D


30 s Wingate
Power Output (Watts)
1200
1100
1000
900
800
700
600
500
400
300
200
100
0
0 3 6 9 12 15 18 21 24 27 30 33 36
Time (s)
Trial 1
Trial 2


33 s Wingate
Power Output (Watts)
1200
1100
1000
900
800
700
600
500
400
300
200
100
0
0 3 6 9 12 15 18 21 24 27 30 33 36
Time (s)
Informed
Deception

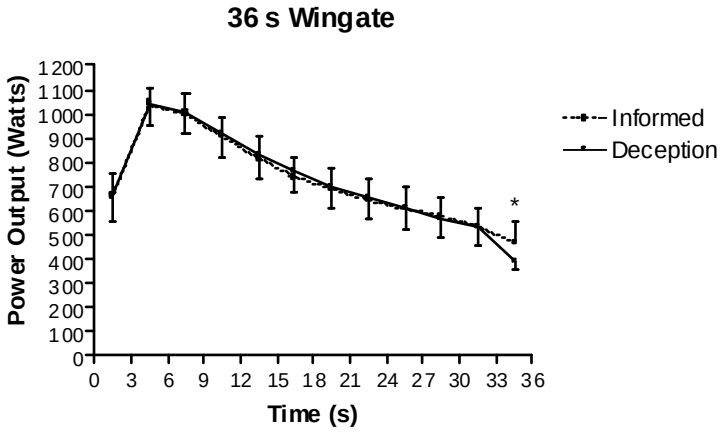
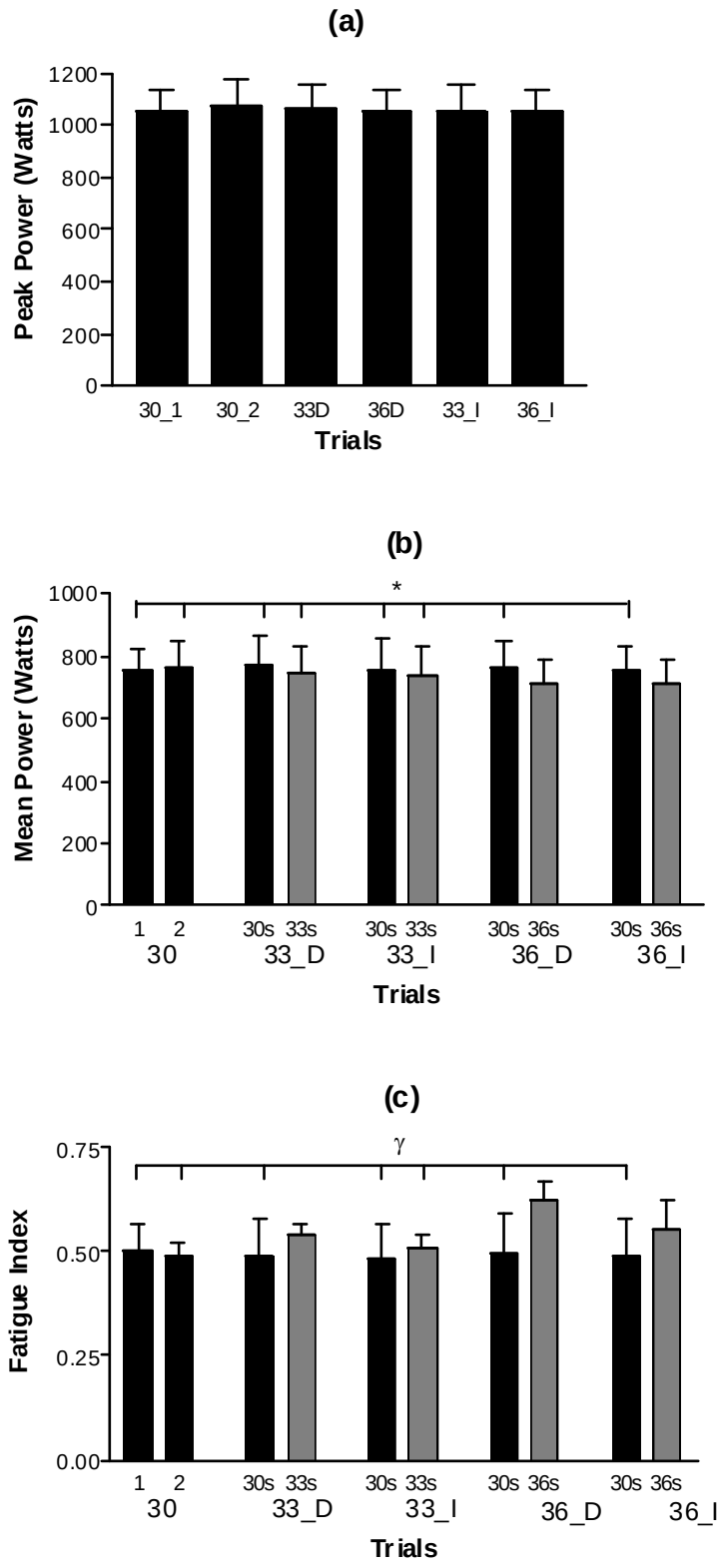

36 s Wingate
Power Output (Watts)
1200
1100
1000
900
800
700
600
500
400
300
200
100
0
0 3 6 9 12 15 18 21 24 27 30 33 36
Time (s)
*
Informed
Deception

Figure 2


(a)
Peak Power (Watts)
1200
1000
800
600
400
200
0
30_1
30_2
33D
36D
33_I
36_I
Trials
(b)
Mean Power (Watts)
*
1000
800
600
400
200
0
1 2
30s 33s
30s 33s
30s 36s
30s 36s
30
33_D
33_I
36_D
36_I
Trials
(c)
Fatigue Index
γ
0.75
0.50
0.25
0.00
1 2
30s 33s
30s 33s
30s 36s
30s 36s
30
33_D
33_I
36_D
36_I
Trials

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