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EVIDENCE FOR MUSCLE DAMAGE FOLLOWING VARIATION IN RESISTIVE FORCE DURING CONCENTRIC HIGH INTENSITY CYCLE ERGOMETRY EXERCISE. BODY MASS OR COMPOSITION?

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ABSTRACT

Baker JS, Hullin D, Davies B. Evidence For Muscle Damage Following Variation In Resistive Force During Concentric High Intensity Cycle Ergometry Exercise. Body Mass Or Composition? *JEPonline* 2005;8(5):43-51. The purpose of this study was to compare power outputs, and concentrations of creatine kinase (S-CK), myoglobin (S-Mb) and blood lactate ($[La^-]_b$) following 30 s of maximal cycle ergometry exercise when resistive forces were dependent on total-body mass (TBM) and fat-free mass (FFM). Cardiac troponin concentrations (S-cTnl) were also determined to exclude protein leakage from the myocardium. Significant differences ($P<0.05$) in peak power output (PPO) were found between the TBM and FFM protocols (1020 ± 134 vs. 953 ± 114 Watts respectively). Differences were also found between pedal velocities and resistive forces (134 ± 8 vs. 141 ± 7 rev/min; 6 ± 1 vs. 5 ± 1 kg respectively). Significant differences were also noted for S-CK from rest to immediately post exercise during both the TBM and FFM protocols. Data for $La^-]_b$ were also significantly different recorded from rest, to immediately post and 24 h post exercise for both the TBM and FFM protocols. Differences were observed immediately post exercise between the TBM and FFM protocols for S-Mb concentrations ($P<0.05$). The results of the study suggest that greater power outputs are obtainable with significantly less muscle damage when resistive forces reflect FFM mass during loading procedures.

Key Words: Creatine Kinase, Anaerobic Ability

INTRODUCTION

High intensity cycle ergometry has been widely employed to assess indices of muscle performance during maximal exercise. Traditionally, the resistive force selected for such a test is determined from individual total body mass for a friction loaded Monark cycle ergometer (i.e. 75 g/kg) (1). More recent studies (2-5) have shown that traditional forces may be too light to elicit maximal performances and that optimization protocols can produce higher PPO.

Conceptually, selecting the optimal resistive force according to total body mass may not be the best approach. Fat-free mass or active muscle mass may be better alternatives (4). Van mil et al. (6) suggested that optimal performance during high intensity cycle ergometry is highly related to an individual's lean body mass, or to the mass of the skeletal muscles involved in the exercise task. Clearly, individuals of equal body mass may have very different body compositions (7). Because body mass and not composition is the most commonly used index to determine resistive force, over- or under- estimation of the "correct" load may occur. In support of these findings recent research conducted in our laboratory has shown a clear increase in PPO when subjects were optimized for fat-free mass (8). The increases in power observed however, may compromise the integrity of skeletal muscle. Exercise induced muscle damage has been widely reported following different types of exercise in humans (9-11). Increases of myocellular protein levels in serum occurring simultaneously or following exercise can be used as indicators of muscle damage (10,12). This damage leads to a temporary loss of the exercising muscles capacity for force production, has implications for increases in muscle soreness and can be related to histological signs of muscle damage. Muscle damage may be detected by a transient rise in serum concentration of muscle proteins such as S-CK and S-Mb.

The purpose of this study was to compare changes in the levels of S-CK, S-Mb and $[La^-]_b$ following a bout of 30 s high intensity cycle ergometry performance when resistive forces were derived from TBM or FFM.

METHODS

Subjects and Experimental Design

Eighteen apparently healthy male university students volunteered as subjects. Prior to testing, all subjects read, completed and signed an informed consent form. Approval for the study was granted by the local ethics committee.

All subjects were fully habituated to the experimental procedures prior to testing. Habituation periods were performed on three occasions duplicating the experimental data collection conditions. The study was designed using a randomised double blind crossover design. Two rest days (no physical activity) preceded each test and subjects attended the laboratory following an overnight fast in an attempt to attenuate any influence of diet on performance. Throughout, and three weeks prior to data collection, subjects refrained from additional dietary supplementation and no appreciable deviations from their normal eating habits were recorded (Nutri - check, UK).

Anthropometric Measures

Stature, body mass and body composition were determined using a calibrated weighing scales (Seca, UK), stadiometer (Seca, UK) and underwater weighing procedures, respectively. Body mass was measured to the nearest 0.1 kg and stature to 0.1 cm. Body density was assessed using underwater weighing techniques described previously by Behnke et al. (13). Relative body fat was estimated from body density using the equations of Siri (14). Residual lung volume was measured using the modified oxygen rebreathing method (15). FFM was determined by subtracting fat mass from TBM.

Force Velocity Test

A force velocity test was performed one week prior to the 30 s cycle ergometer test to determine optimal resistive forces for TBM and FFM using procedures outlined by Jaskolska et al. (5). Briefly, the test consisted of six short maximal sprints (6-8 s) against randomly assigned resistive forces (70, 75, 80, 85, 90 and 95 g/kg). Successive exercise bouts were separated by a 5 min rest period. The load that produced the highest PPO value for TBM and FFM was considered optimal and was used in the 30 s test.

30 S Cycle Ergometer Test

A cycle ergometer (Monark 864) was calibrated prior to data collection, following guidelines outlined by Coleman (16). The same calibration procedure was used for the force velocity test and the 30 s test. Subjects were assigned randomly to a previously determined TBM or FFM resistive force. Each subject returned to perform the remaining test with 1 week intervening between experimental conditions. This time period was observed to facilitate full recovery. All subjects were very physically active, utilised concentric contractions during testing conditions and were fully familiarised to experimental conditions, therefore any observable repeated bout effect would be minimal.

Saddle heights were adjusted to accommodate partial knee flexion of between 170° to 175° (with 180° denoting a straight leg position) during the down stroke. Throughout test duration the feet were supported by toe clips and straps. All subjects remained seated during the test and were verbally encouraged to perform maximally. All performed a standardised 5 min warm up according to procedures outlined previously (5).

Indices of performance were determined from flywheel revolutions using an inertia corrected computer program (16). Data transfer was made possible using a suitably mounted sensor unit and power supply attached to the fork of the ergometer. The sampling frequency of the sensor was 18.2 Hz. Validity and reliability of the cycle ergometer as a test of muscle power has been reported as $r = 0.93$ (17). Heart rate recordings for each subject were measured continuously using a short-range telemetry system (Sport Tester 3000, Polar Electro Finland).

Blood Sampling

Duplicate blood samples were collected at the same time of day and by the same investigator in an attempt to control for biological and between subject variations (18). In an attempt to control for plasma volume changes, all resting samples were taken following 30 min of supine rest. The immediate post exercise samples were taken with subjects placed in a supine position on a clinical couch to minimise the risk of fainting. This procedure was followed for both protocols. In addition, both capillary and venous samples were corrected for plasma volume changes using the equations of Dill and Costill (19).

Venous Blood

10 ml of blood was collected from an antecubital forearm vein using the Vacutainer system (Becton Dickinson, Rutherford, NJ, USA). Following collection the samples were placed immediately on ice, allowed to clot and centrifuged at 3,500 rpm for 10 mins. Serum was then extracted and placed into eppendorfs (plastic containers) and stored at -80°C prior to biochemical analysis.

Capillary blood

Duplicate blood samples from the right ear lobe were collected using a capillary tube. Samples were analysed immediately for $[La^-]_b$ concentrations using a calibrated automated electrochemical analyser (Analox PGM7 Champion, London UK).

Creatine Kinase (S-CK)

S-CK concentration was measured using the Kodac Ektachem Clinical Chemistry Slide (CK). Reflection densities were monitored during incubation. The rate of change in reflection density was

then converted to the measurement of enzyme activity. Coefficient of variation during measurement was 4.4%.

Determination of Myoglobin (S-Mb)

S-Mb concentration was analysed using the Chiron Diagnostics ACS: 180[®] Automated Chemiluminescence System. Myoglobin concentration was determined by the direct relationship of the amount present in the patient sample and the amount of relative light units (RLUs) detected by the system. Coefficient of variation was established at 3.5%.

Determination of Cardiac Troponin I (S-cTnI)

Cardiac Troponin was measured using the Chiron Diagnostics ACS: 180[®] Automated Chemiluminescence Systems. Cardiac Troponin concentration was determined by the direct relationship between the amount of troponin I present in the patient sample and the amount of relative light units (RLUs) detected by the system. Coefficient of variation was established for the measurement at 3.5%.

Statistical Analyses

Differences in power components between TBM and FFM were analysed using Students *t*-test. A two factor repeated measures ANOVA with Bonferroni corrections was used to investigate differences in S-CK, S-Mb, S-cTnI and $[La^-]_b$ concentrations within and between protocols over the three blood sampling stages. Significance was accepted at $P < 0.05$. All statistical computations were performed using the SPSS for Windows package (SPSS, Surrey UK.), and all data reported as mean \pm SD.

RESULTS

Age and physiological characteristics of the subjects are given in Table 1. Values for power outputs generated during the study for the TBM and FFM protocols are presented in Table 2.

Significant differences ($P < 0.05$) in PPO were found between the TBM and FFM protocol (953 \pm 114 vs. 1020 \pm 134 Watts, respectively). Differences were also found ($P < 0.01$) between

pedal velocities and resistive forces (134 \pm 8 vs. 141 \pm 7 rev/min; 6 \pm 1 vs. 5 \pm 1 kg, respectively). No

differences ($P > 0.05$) were found between MPO, FI %, WD or heart

rate. S-CK, S-Mb, S-cTnI and $[La^-]_b$ concentrations recorded during the study are given in Table 3.

Significant differences were found for S-CK from rest to immediately post exercise during the TBM and FFM protocols ($P < 0.01$; $P < 0.05$

respectively). $[La^-]_b$ values were

also significantly different ($P < 0.01$) from rest to immediately post and 24 h post exercise for both the TBM and FFM protocols. No significant differences were recorded during any of the blood sampling stages for S-Mb or S-cTnI within groups ($P > 0.05$). However, significant differences were observed immediately post exercise between the TBM and FFM protocols for S-Mb concentrations ($P < 0.05$) with the TBM protocol recording the higher values.

Table 1. Age and anthropometric characteristics of subjects (n=18).

Variable	Mean \pm SD
Age (yrs)	23.0 \pm 2.0
Stature (cm)	175.8 \pm 5.7
Mass (kg)	75.3 \pm 11.0
Fat (%)	11.6 \pm 2.7
Fat Mass (kg)	8.9 \pm 3.3

Table 2. Cycle ergometry power profiles for both the TBM and FFM protocols. Time to PPO, pedal revolutions, fatigue index, work done, resistive forces and maximal heart rates are also given.

Variable	TBM	FFM	Sig
PPO (Watts)	953 \pm 114	1020 \pm 134	$P < 0.05$
MPO (Watts)	535 \pm 63	512 \pm 68	NS
T to PPO (s)	4 \pm 3	3 \pm 2	NS
Prevs (rev/min)	134 \pm 8	141 \pm 7	$P < 0.05$
FI (%)	42 \pm 8	38 \pm 10	NS
WD (J)	16050 \pm 1828	15369 \pm 1975	NS
Rf (kg)	6 \pm 1	5 \pm 1	$P < 0.05$
HR (beats/min) post	177 \pm 8	175 \pm 8	NS
HR (beats/min) pre	68 \pm 10	66 \pm 11	NS

Table 3. Creatine Kinase (S-CK), Myoglobin (S-Mb), Cardiac Troponin (S-cTnl) and Blood lactate ([La⁻]b) concentrations for TBM and FFM measured at rest, immediately post and 24 hr post exercise.

Variable	Condition	Pre	Post	24 hr Post
S-CK (units/L)	TBM	202 ± 162	2236 ± 213†	175 ± 110
	FFM	157 ± 81	1172 ± 93 †	136 ± 67
S-Mb (ng/mL)	TBM	53 ± 22.1	54.5 ± 25.4†	49.7 ± 12.4
	FFM	46 ± 13.9	446.3 ± 13†	42 ± 7.5
S-cTnl	TBM	0.06 ± 0.04	00.05 ± 0.04	0.03 ± 0.02
Blood Lactate (mmol/L)	TBM	0.5 ± 0.7	9.0 ± 1.2	00.6 ± 0.6
	FFM	0.5 ± 0.7	9.3 ± 1.4	00.7 ± 0.8

†,p<0.05

DISCUSSION

S-cTnl was not detected in any participant over any of the three experimental conditions for either the TBM or FFM protocols, which provides evidence that S-CK leakage, occurred predominantly from skeletal muscle and that myocardial damage did not occur. Increases in serum concentrations of intracellular proteins are generally accepted as good indicators of muscle fiber disruption, damage or permeability of muscle cell membranes (20-22). The mechanisms responsible for the escape of intramuscular proteins into the blood are poorly understood. However, two mechanisms have been proposed by Pyne (22), and include mechanical damage to skeletal muscle and the cell membrane. Further mechanisms have been outlined by other researchers (12,23-25). These mechanisms include metabolic damage, insufficient rate of ATP production, muscle ischemia or hypoxia, alterations in ion concentration and free radical production resulting in lipid peroxidation of cell membranes.

The greater concentrations of S-CK and S-Mb observed for the TBM protocol, when compared to FFM may be related to the heavier resistive forces used. These observations were consistent when the TBM protocol was assigned randomly as the first or second experimental condition. The results from this study indicate that the significantly lighter resistive forces used for FFM and the higher pedal velocities obtained may be reflecting effectiveness and efficiency in force velocity relationships for FFM when compared to TBM. These observations agree with the suggestions of Wilkie (26), who stated that force should be matched to the capacity of muscle in order to exploit the full force velocity relationship. The findings also indicate that the FFM protocol may be causing less mechanical damage to the muscle fibers and cell membranes in spite of the PPO recorded being greater when compared to TBM.

Kyrolainen et al. (11) measured the concentration of carbonic anhydrase III (S-CAIII) and found that the protein response to jumping performance was curvilinear. These authors suggested that a certain intensity or threshold of exercise was required before leakage of proteins occurred. No relationships were found in this study between power outputs recorded for the TBM or FFM protocols and serum concentrations of S-CK or S-Mb. This may be explained by the fact that the power measures and protein concentrations observed by Kyrolainen et al. (11) were obtained using power athletes. The subjects in this study were university students and by comparison were probably less powerful. Also, the duration of testing was shorter and S-CAIII, a cytosolic enzyme, may be more concentrated in type I fibers (27). S-Mb has been observed in high concentrations in type I and type II fibers (28). Because fiber types were not established in this study comparisons of concentrations between tests is difficult.

A major problem in attempting to assess maximal short-term power output is the dependence of power output on contraction velocity (29-31). Because the relationship between force and velocity is

of an inverse curvilinear form, power, the product of force and velocity, has a parabolic relationship, reaching a maximum value at some intermediate optimal velocity. Therefore, contraction at slower or faster velocities may reduce power output. During the present study the resistive forces were fixed, and muscular contraction was concentric in nature, and any increases in pedal velocity for a given resistance over a specified time would result in a power increase. There are however several factors that need consideration regarding protein efflux. These include protein transportation from the muscle interstitium to the intravascular space through lymphatic vessels, and their clearance rate. It has been demonstrated that lymph and blood flow from the muscles is greatly affected by muscular activity (32, 33). The higher concentrations of S-CK observed following the TBM protocol were probably the result of a greater disruption of the muscle cell membrane and possible accelerated transport to serum when the TBM and FFM protocols were compared.

Mair et al. (34) suggested that if the mechanical strain is greater than the ability of the muscle to resist, disruption of the contractile apparatus, the myofibrils and mechanical damage to the plasma membrane and the sarcoplasmic reticulum will occur. This may then lead to a disturbed intracellular calcium homeostasis and contractile function. These observations appear to be consistent with smaller concentrations of S-CK, and S-Mb observed following the FFM protocol. The early efflux of muscle proteins can also be attributed to the early increase in the permeability of muscle fiber plasma membranes. The resistive forces used during the TBM protocol may be causing more mechanical trauma in the early stages of the test thereby accelerating membrane permeability.

This suggestion may be substantiated by the higher pedal velocities recorded for the FFM protocol, indicating ease of motion and therefore less mechanical trauma at the onset of exercise. Van der Meulen et al. (35) demonstrated that the amount of structural muscle damage induced by exercise was significantly lower than the estimated amount of damage based on intracellular enzyme release into the blood. These researchers also concluded that the increase in plasma activity of muscle enzymes may reflect changes in membrane permeability. McNeil et al. (36) demonstrated that membrane disruptions caused by the imposition of mechanical force on this fragile structure are common in exercised muscle.

McNeil et al. (36) further commented that in some cases resealing of these localized membrane disruptions often occurs. In this study significant increases in S-CK were recorded for both the TBM and FFM protocols, the higher values recorded for the TBM protocol may indicate a slower resealing process reflected by greater muscular and/or membrane damage. Muscle phosphagen content plays a primary role in maximal exercise as observed by several researchers (37, 38). The greater work and resistive force used for the TBM protocol may increase energy depletion as measured by ATP. Cerny et al. (39) suggested that the energy demanded by working muscles reduced the amount of ATP available for membrane integrity, leading to a loss of cellular enzymes and damage. This is in contrast to the FFM protocol. The higher pedal velocities and lower resistive forces may have resulted in a more economic use of energy derived from phosphagenic sources. The increase in PPO observed for FFM may also be indicating a more efficient mechanism of ATP resynthesis, which may contribute to less muscle damage by the mechanisms outlined by Cerny et al. (39). This suggestion may explain in part the lack of significant difference in $[La^-]$ production between the TBM and FFM protocols. The $[La^-]$ values indicate that anaerobic glycolysis was active during both protocols and suggests that the magnitude of activation was similar for both groups in spite of the increases in PPO recorded for FFM.

CONCLUSIONS

In conclusion, the FFM protocol produced significantly higher PPO when compared to the TBM protocol. The differences in PPO were reflected by significant increases in S-CK in both the TBM and FFM protocols from rest to immediately post exercise, with the TBM protocol recording the highest concentrations. No differences in S-Mb concentrations were recorded within protocols for any of the experimental conditions. However, the S-Mb concentration during the TBM protocol was significantly higher, immediately post exercise, when the TBM and FFM protocols were compared. The increase in PPO observed during the FFM protocol, and lack of significance between $[La^-]_b$ concentrations suggest that this method of resistive force selection represents a more valid index of ATP-PC activity and/or muscular efficiency. The concentrations of S-CK and S-Mb recorded indicate significantly less muscle damage when the FFM protocol was compared to protocols that include the fat component of body mass.

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