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TITLE

EFFECT OF SIMULATED AIR COMBAT MANEUVERING ON MUSCLE GLYCOGEN AND LACTATE

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Effect of Simulated Air Combat Maneuvering on Muscle Glycogen and Lactate

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Muscle glycogen and muscle and blood lactate were evaluated before and after a +4.0/7.0 G_z simulated air combat maneuvering (SACM) protocol in the human centrifuge. The subjects were eight healthy males, ages 25–43 years. Muscle glycogen and lactate were determined from biopsies of m. vastus lateralis in six subjects and whole blood lactate was analyzed in finger-tip blood samples from eight subjects. G-tolerance time was 256 ± 33 s (Mean ± S.E.M.). The decrease in glycogen concentration averaged 81 ± 36 mmol · kg^{-1} dry wt ($p = 0.07$). The rate of glycogen utilization was low, averaging 0.4 ± 0.1 mmol · kg^{-1} · s^{-1}. Muscle lactate increased significantly from 28 ± 2 mmol · kg^{-1} dry wt pre-SACM to 51 ± 4 mmol · kg^{-1} post-SACM. Post-SACM blood lactate was 4.2 ± 0.3 mmol · L^{-1}. Neither final blood nor muscle lactate values nor the difference between pre- and post-SACM muscle lactate concentrations were related to G-tolerance time. It was concluded that glycogen availability in m. vastus lateralis is not a limiting factor during exposure to headward acceleration of this type and duration. The lactate values, while high, cannot fully explain the muscular fatigue occurring during centrifuge exposures of the type used here. Therefore, the suggestion by others that anaerobic energy metabolism in skeletal muscles is the crucial factor limiting the ability to resist fatigue during exposure SACM is not supported and is likely an oversimplification of a much more complex problem.

EXPOSURE TO HEADWARD ($+G_z$) acceleration causes a decrease in head-level blood pressure. Physiological mechanisms to combat this reduction of arterial pressure include arterial baroreceptor reflexes (6), venous "siphoning" through the jugular vein (which maintains the pressure differential between the arterial and venous sides of the cerebral circulation) (10), maintenance of venous return through the abdomen by proportional increases in intra-abdominal pressure due to hydrostatic effects (16), and peripheral venoconstriction (24). However, these are only effective up to around +4–5 G_z for most people, and their efficacy is closely related to the rate of onset of the acceleration because of the time required for most of these mechanisms to become fully activated. Higher levels of acceleration can be tolerated only with the aid of mechanical intervention (i.e., G-suits, positive-pressure breathing, tilt-back seats), physical intervention such as the anti-G straining maneuver (AGSM), or both.

The AGSM involves voluntary isometric tensing of skeletal muscles in addition to an intermittent Valsalva maneuver, which is held for 3 s followed by a rapid exhalation and inspiration and then repeated. This maneuver causes an increase in head-level blood pressure by transmitting the increase in intrathoracic pressure, generated by the Valsalva maneuver, directly to the heart and great vessels and by improving and maintaining venous return from the abdomen and legs. The skeletal muscle tensing may also act to increase peripheral vascular resistance, as isometric muscular contraction has been shown to have this effect (22).

The AGSM increases $+G_z$ tolerance, but it is also very fatiguing. Muscular fatigue could impair the ability of the subject to generate high levels of arterial blood pressure. It likely also increases the possibility of venous pooling in the lower extremities, due to the reduced intensity of muscular contraction, resulting in gray-out, blackout, and eventual loss of consciousness. Therefore, muscular fatigue can adversely affect $+G_z$ tolerance.

It has been implied that muscle glycogen is the primary fuel used by skeletal muscle during the AGSM and that the reduction of glycogen stores could limit $+G_z$ tolerance in situations where the AGSM is used for relatively prolonged periods, such as during a dogfight or

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aerial combat maneuvering (ACM) (8). However, exercise of the duration associated with such situations (~2–8 min), regardless of the intensity, has not previously been associated with any exhaustion of muscle glycogen stores (15). Accumulation of lactate in the muscle, due to the probable ischemic conditions of the muscle during the AGSM, however, could be an important factor in the development of muscle fatigue. Therefore, it has been suggested (3,4) that anaerobic metabolism plays an important role in energy transduction during ACM or its centrifuge analog, simulated air combat maneuvering (SACM). It has also been suggested that anaerobic capacity (the ability of the contracting musculature to transduce energy from anaerobic glycolysis and endogenous ATP/CP stores) is the primary factor in determining the ability to sustain the AGSM during simulated air combat maneuvering (4). This conclusion was based primarily on blood lactate data (3,28) and observations that muscle strength training increases G-tolerance time during SACM (7,28). Previous studies have not, however, measured concentrations of muscle glycogen and muscle lactate before and after a centrifuge exposure. The purpose of this present study, therefore, was to determine the changes in glycogen and lactate concentrations in muscle caused by exhaustive SACM requiring the AGSM. The muscle examined was vastus lateralis. This muscle was chosen because of its ease of access, the availability of well-documented physiological characteristics in the scientific literature, and the general opinion of centrifuge subjects that this muscle is a primary area of perceived fatigue.

It should be noted that intense muscular contractions are only performed intermittently during SACM because substantial relaxation is usually possible during the +4.0 G_z portion of this profile.

METHODS

The subjects were eight male volunteers, ages 25–43 years. They wore shorts, a t-shirt, running shoes and socks, and an anti-G suit (CSU-12/P). To be able to participate in the study, subjects were required to pass a stringent medical examination. They were fully briefed on the discomforts and risks associated with the experimental protocol and signed an informed consent form indicating that they had been briefed to their satisfaction.

The centrifuge protocol was as follows: acceleration from baseline +1.4 G_z to +4.0 G_z; 10-s plateau at +4.0 G_z, accelerate to +7.0 G_z (rate of onset 1.0 $G \cdot s^{-1}$), hold for 10 s; return to +4.0 G_z; alternate +4.0/7.0 G_z plateau until endpoint (fatigue). Endpoint was defined as tunnelling of vision bilaterally to within 25° of the central fixation point, or gray-out resulting in loss of more than half of the central vision. The visual effects associated with fatigue during exposure to +G_z are caused by decreased blood flow to the retina when the AGSM fails to raise head-level arterial pressure to the required level (20). Additionally, subjects were able to terminate the run at any time by releasing the braking switch if unable to continue for any reason. Other termination points included the following cardiac indices: heart rate reaching age-predicted maximum; more than 5 ectopic beats/60 s or; more than 2 strings of ventricular premature beats.

As this was part of a larger study, prior to each run the electromyographic activity and force production of several muscle groups were evaluated. These data will be reported in a subsequent paper.

Muscle biopsies were taken from m. vastus lateralis using a standard percutaneous needle biopsy technique (1). Only six of the eight subjects volunteered for the biopsy procedure. Approximately 5 min elapsed between the end of the evaluation of force production and EMG activity and the first biopsy which was taken immediately prior to the centrifuge run. The post-SACM biopsy was taken with the subject still seated in the centrifuge within 1 min after the endpoint. The biopsy could not be taken sooner because of the time required to brake the centrifuge. The biopsy incision site was chosen so that the thigh bladder of the anti-G suit covered the site, thus acting as a pressure bandage. The muscle sample was divided into two parts; one was immediately frozen in liquid nitrogen and stored at −70°C for later analysis of glycogen and lactate. The second portion, for histochemical analysis, was first mounted in an embedding medium, then frozen in isopentane cooled in liquid nitrogen. Later, these were cut into sections 10 μm thick, stained for myofibrillar ATPase, and incubated at alkaline pH for fiber-type determination (type I and II) (5). A photodensitometric method, using a periodic acid Schiff (PAS) reagent histochemical stain, was employed to evaluate glycogen content within type I and type II fibers (2).

Fibers from the freeze-dried portion were dissected free of fat, blood, and connective tissue using a dissecting microscope. Samples for glycogen and lactate were assayed in triplicate. Glycogen was quantified as the concentration of glucose units after hydrochloric acid hydrolysis according to the method of Lowry *et al.* (17) as modified by Karlsson (14). Muscle lactate was determined using a fluorometric enzymatic method (14,17).

Blood lactate was determined in fingertip capillary samples from eight subjects using a YSI Model 23A glucose analyzer (Yellow Springs Instruments, Yellow Springs, OH) modified to determine lactate concentrations. Blood was collected in microcapillary tubes immediately prior to and 3 min after the centrifuge runs.

Statistical analysis was performed using two-tailed paired *t*-tests and linear regression. Statistical significance was accepted at the 0.05 level but trends are reported as well. Values are reported as mean ± S.E.M.

RESULTS

G-tolerance time averaged 256 ± 33 s (range 125–373 s).

Muscle glycogen content in the vastus lateralis decreased by 81 ± 36 mmol $\cdot$ kg^{-1} dry weight (wt) ($p = 0.07$). Although this indicates a trend, the average change is not very large. Pre- vs. post-SACM differences ranged from an increase of 49 to a decrease of 210 mmol $\cdot$ kg^{-1} dry wt among the individual subjects (Fig. 1). The rate of glycogen utilization was 0.4 ± 0.1 mmol $\cdot$ $kg^{-1} \cdot s^{-1}$. Photodensitometric analysis re-

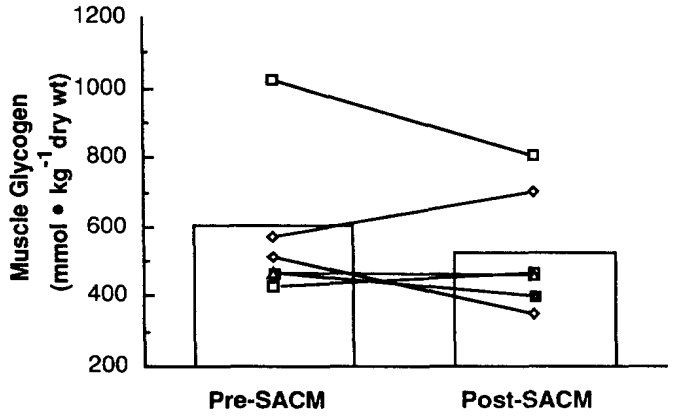

Fig. 1. Glycogen content of m. vastus lateralis before and after muscular contraction using the anti-G straining maneuver during SACM. Individual responses superimposed on mean values.

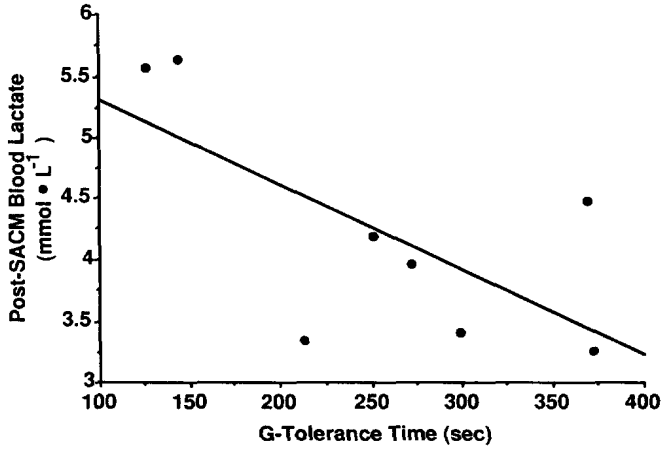

Fig. 3. Relationship between G-tolerance time and post-SACM blood lactate concentrations. r = −0.68, p = 0.06.

vealed no differences in glycogen depletion patterns between fiber types and confirmed the statistically nonsignificant difference in muscle glycogen content pre- and post-SACM.

Muscle lactate concentration increased significantly from 28 ± 2 mmol · kg^{-1} dry wt pre-SACM to 51 ± 4 mmol · kg^{-1} post-SACM (Fig. 2). Pre-SACM muscle lactate concentrations were higher than normal resting conditions, probably due to some accumulation from the contractions performed in the static chair prior to the centrifuge runs.

Blood lactate levels increased significantly from 1.1 ± 0.1 to 4.2 ± 0.3 mmol · L^{-1} after SACM.

Linear regression analyses showed no significant linear relationship between G-tolerance and final blood lactate or muscle lactate values (Fig. 3–4) or the difference between pre- and post-SACM muscle lactate (Fig. 5).

DISCUSSION

Our results for G-tolerance time compare favorably to the results of Tesch *et al.* (28), but our average G-tolerance time is longer than that found by Burton *et al.* (3) using a similar centrifuge profile. Our blood lactate values are also similar to those found in both these studies.

The main finding of the present investigation is that G-tolerance time is not related to skeletal muscle glycogen depletion nor lactate accumulation. This is not surprising considering the length of the exposure. Glycogen depletion has been shown to be a factor associated with fatigue only in prolonged exercise; i.e., 30 min to 2–3 h (11). Karlsson and Ollander (15) reported reductions in intramuscular glycogen of up to 143 mmol · kg^{-1} dry wt during static leg exercise at 25% of maximal voluntary contraction (MVC) performed to exhaustion (average 5.8 min). If one assumes a normal intramuscular glycogen concentration of about 350–500 mmol · kg^{-1} dry wt, however, this represents only a 30–40% decrease in glycogen stores. This is a much greater percentage decrease in intramuscular glycogen than in the present study but it was not associated with termination of the exercise (15). Recently, it has also been shown that force generation is not impaired when muscle glycogen levels are markedly reduced during high-intensity, short duration (~60 s) isometric exercise (26).

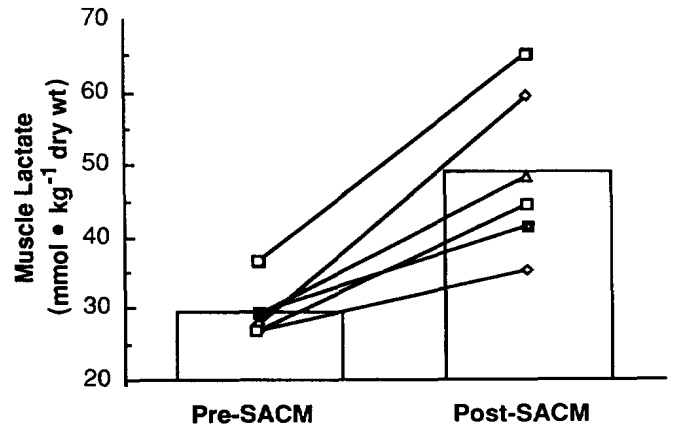

Fig. 2. Muscle lactate in m. vastus lateralis before and after muscular contraction using the anti-G straining maneuver during SACM. Individual responses superimposed on mean values.

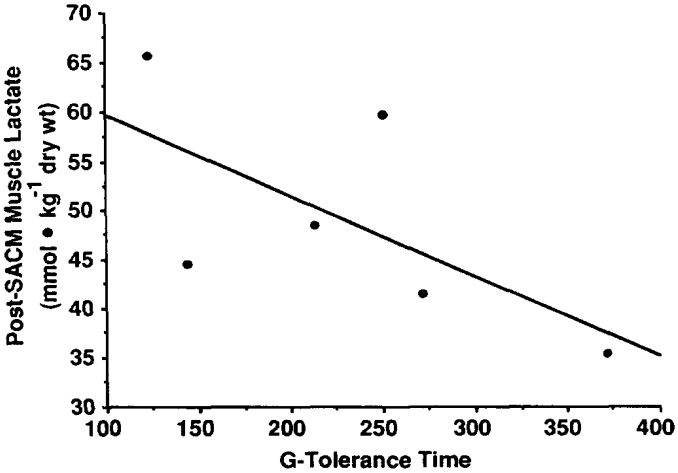

Fig. 4. Relationship between G-tolerance time and post-SACM muscle lactate concentrations. r = −0.65, p = 0.15.

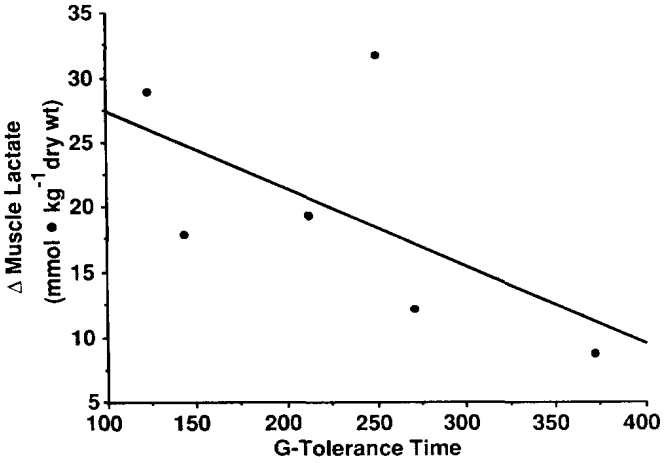

Fig. 5. Relationship between G-tolerance time and the difference between pre- and post-SACM muscle lactate concentrations. r = −0.59, p = 0.21.

Our calculated rate of glycogen utilization of 0.4 mmol · kg^{-1} · s^{-1} dry wt suggests a relatively mild intensity of muscular contraction. For example, Robergs *et al.* (21) reported a rate of glycogenolysis of 0.91 mmol · kg^{-1} · s^{-1} dry wt during isotonic leg contractions at only 35% of maximal force. Interestingly glycogen utilization during isometric contraction at 25% MVC, in the study by Karlsson and Ollander (15), is estimated to have been approximately 0.4 mmol · kg^{-1} · s^{-1} dry wt, which is identical to our results.

It has also been implied in previous work that the ability to sustain an anti-G straining maneuver during SACM is directly related to anaerobic capacity (4) [anaerobic capacity in this case being defined as the cumulated work produced in a muscle fatigue test using one leg on an isokinetic dynamometer (28)]. This explanation has been based, largely, on a study by Tesch *et al.* (28), which showed a positive relationship between blood lactate concentration after an SACM and G-tolerance time. The calculation of that regression equation was based on repeated samples taken on the same subjects before, in the middle, and after an 11-week strength-training program. Thus, they do not represent independent observations. This relationship has not been confirmed by our data nor those of Burton *et al.* (3). Although the regression of blood lactate and G-tolerance time in the present study seems to have a moderately strong regression coefficient of −0.68 and is almost statistically significant at the 0.05 level ($p = 0.06$), it is apparent from Fig. 3 that this is caused by the two points at the upper left hand corner of the graph. Without these points, the regression fit is very weak and extremely non-significant.

Similarly, we did not find a significant relationship between G-tolerance time and either post-SACM muscle lactate values or the difference between pre- and post-SACM muscle lactate values. The muscle lactate values at the end of the centrifuge runs, while elevated, are probably not high enough to be the limiting factor in the performance of the AGSM. Tesch and Karlsson (27), found that mean muscle lactate concentration was approximately 90 mmol · kg^{-1} dry wt after an exhaustive isometric contraction with the quadriceps femoris at 50% MVC. Muscle lactate values at 75 and 25% MVC were not as high, indicating that intramuscular acidosis secondary to lactate accumulation could not have been the limiting factor at these intensities of muscle contraction. The highest individual muscle lactate concentration in the present study was only 65 mmol · kg^{-1} dry wt. Our finding of an average post-SACM muscle lactate concentration of about 51 mmol · kg^{-1} dry wt is similar to that found by Tesch and Karlsson (27) at 25% MVC (i.e., 59 mmol · kg^{-1} dry wt). Therefore, it is likely that anaerobic capacity *per se* has little bearing on G-tolerance time. Thus, we agree with those who suggest (29) that to try to attribute the ability to sustain an AGSM during air combat maneuvering to this one factor would be an oversimplification of the problem.

The fact that weight-training has been shown to improve G-tolerance times during SACM, and also improves anaerobic power (7,28) does not, in and of itself, imply a causal relationship between anaerobic power and G-tolerance times during SACM. Weight-training also produces increases in maximal strength (23), neuromuscular adaptation (19) and, in certain cases, depending on the type of training, an increase in muscular endurance (18,23). All these factors will have an effect on the ability to sustain an AGSM and, therefore, G-tolerance time. As maximal strength is increased, it is possible that a given level of $+G_z$ requires an AGSM that represents a lower percentage of maximal effort. This would likely improve endurance (12,18). It may also be that, since the contraction represents a smaller percentage of MVC, intramuscular pressure is decreased and blood flow is improved relative to the metabolic demand (25). This would also likely improve endurance.

It must be stressed that the interpretation of these data is limited to the one muscle that was studied. It is possible that greater reductions in glycogen concentration occur in other muscles perhaps more crucial to the AGSM, such as rectus abdominis or the external obliques, or that lactate accumulation is greater in these other muscles. There is evidence that some muscle fibers in a muscle are actually able to synthesize glycogen and use some of the lactate produced by other fibers or other muscles during contraction (9,13). Our results from pre- and post-exercise biopsy samples simply reflect the net result of catabolic and anabolic processes between the times the samples are taken. Thus, it is possible that the vastus lateralis muscle became a net user of lactate and that there was some resynthesis of glycogen in some muscle fibers during the course of the AGSM and during the 1-min period between the end of the centrifuge run and the post-SACM biopsy. It may be that in other muscles involved in the AGSM, catabolic processes far outweighed anabolic ones, and, therefore, we might see a greater loss of glycogen and accumulation of lactate in those muscles.

In conclusion, our results suggest that G-tolerance time during simulated air combat maneuvering on the human centrifuge is not related to muscle glycogen availability in m. vastus lateralis and cannot be explained solely on the basis of the ability of the anaerobic energy systems to produce ATP. Clearly, there must be

involvement of these systems, however, their relative contributions to overall energy metabolism and fatigue during SACM remain to be elucidated.

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