Single Cell Biology
ISSN: 2168-9431

Baker, et al., Single Cell Biol 2016, 5:3
DOI: 10.4172/2168-9431.1000148

Short Commentary Open Access

High Intensity Exercise and Glycogen Depletion

Julien S Baker[1], **Duncan Buchan**[1] **and Mike Graham**[2*]

[1]*Institute of Clinical Exercise and Health Science, Applied Physiology Research Laboratory, School of Science and Sport, University of the West of Scotland, Hamilton, Lanark shire, Scotland*

[2]*Visiting Professor, Ningbo University, PR China*

[*]**Corresponding author:** Michael R Graham, Visiting Professor, Ningbo University, PR China, Tel: 01698 283100 Ext: 8271; Fax: 01698 894404; E-mail: jsbaker@uws.ac.uk

Rec date: Jul 25, 2016; **Acc date:** Aug 27, 2016; **Pub date:** Aug 29, 2016

Short Communication

Research pertaining to the relevance of skeletal muscle glycogen availability during high intensity exercise was first suggested in the work of Saltin [1,2]. Intense physical exercise is accompanied by rapid glycogen depletion and lactate accumulation in the working muscle, indicating a high rate of glycolysis (Figure 1). Glycogen depletion in human skeletal muscle following heavy exercise was investigated by Gollnick et al. [3,4]. Six 1 min sprints with 10 mins recovery between each exercise were performed at 150% of each individual subjects maximal aerobic power (VO_2 max). Biopsy samples were taken from the vastus lateralis muscle at rest and after the first, third and final work bouts. Total muscle glycogen declined as a function of the number of work bouts. The relative concentration of glycogen was determined from histochemical staining. The first fibres to become depleted of their glycogen stores were the low oxidative, high glycolytic, fast twitch fibres. This suggests an early recruitment of these fibres during heavy exercise.

This pattern is in contrast to that seen during prolonged, moderately intense exercise when the high oxidative slow twitch fibres are the first to become depleted of their glycogen stores. Hargreaves and Richter [5] examined the regulation of skeletal muscle glycogenolysis during exercise.

They observed that muscle glycogen breakdown during exercise is influenced by both systemic and local factors. Contractions per se were found to increase glycogenolysis via a calcium induced, transient increase in the activity of phosphorylase a, and probably also via increased concentrations of Pi. In fast twitch muscle, increases in the AMP and IMP levels may increase phosphorylase activity. The authors also observed that the rate of muscle glycogen breakdown during exercise depends on the pre exercise glycogen concentration and hormonal influences.

They also suggest that insulin may inhibit glycogen breakdown, whereas adrenaline enhances the rate of glycogen use in contracting muscle by increasing the phosphorylase an activity via increased cyclic AMP production. The authors concluded that the availability of blood borne substrates may also influence muscle glycogenolysis and therefore exercise performance. Regulation of glycolysis during intermittent exercise was studied by Essen and Kaijser [6]. Seven healthy male volunteers performed intermittent exercise (15 sec work 15 sec rest) at a high work load for 60 minutes, while six subjects performed continuous exercise at an equally high intensity to exhaustion, which occurred after 6 minutes. Exercise was performed on a Siemens-Elma cycle ergometer at a pedalling rate of 60 rpm. The subjects performing the intermittent exercise protocol averaged work loads of 284W, while the remaining six subjects maintained average work loads of 280W. It was concluded that ATP and CP concentrations were important in the regulation of glycolysis, and influenced carbohydrate and lipid contribution to energy metabolism.

During the first few seconds of muscular contraction glycolysis accounts for approximately half of the ATP used [7]. Continued contraction may result in the total depletion of the phosphocreatine store, and this coincides with a loss of the subjects ability to sustain a voluntary contraction at near maximal force.

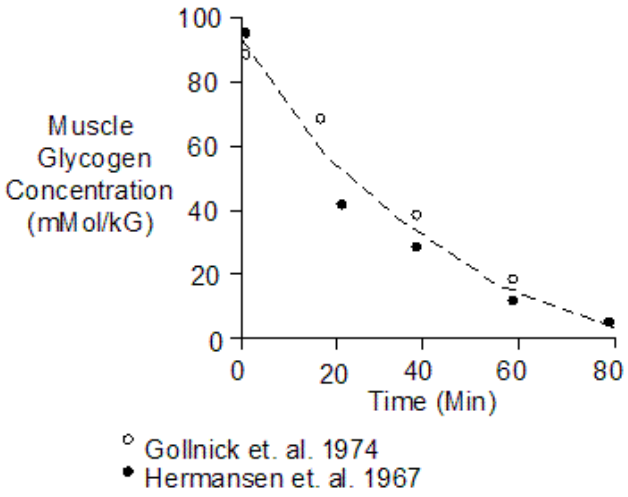

Figure 1: Decrease in muscle glycogen concentration over time.

Eventually the force may decrease to 20% or less of the original value produced. About 50% of the ATP is transformed to IMP and the pH decreases to 6.4. The ATP hydrolysis rate decreases during the same contraction period by 70% to 80% [7]. It is suggested that the reason for the decrease in force and ATP hydrolysis is an inhibition of cross bridge cycling primarily by increases in Pi and H^+ produced by phosphocreatine, ATP splitting and glycolysis [8]. During the last part of the contraction increase in free Mg^{2+} may also add to the inhibiting effect.

Decreased repolarization of the sarcolemma and T-tubular membranes could additionally decrease activation of the contractile system. Harris et al. [9] Jacobs et al. [8] measured changes in muscle metabolites in females after 30 sec exhaustive exercise. Nine female physical education students volunteered as subjects. They were instructed to pedal at maximum frequency against a resistive force of 45 g/kg^{-1} total body mass. Samples obtained by needle biopsy were

taken before and after exercise from the vastus lateralis. Peak power output values of 561W were attained, with mean values of 453W, and end power output values of 228W. The results showed that pronounced intramuscular lactate accumulation occurred after 10-sec maximal cycle exercise.

This suggests that glycogenolysis commences with a very short time delay after the onset of muscular contraction, in such high-intensity performance. Human muscle metabolism during brief maximal cycle ergometer exercise has been examined by Boobis et al. [10]. Subjects performed a 30-sec cycle ergometer test, against a pre-set load of 75 g/kg^{-1} total body mass. The rotation of the fly wheel was continuously recorded so that power output during each second of the test could be calculated. Peak power output was achieved during the initial 3 to 6 sec period. Thereafter, power values declined rapidly which resulted in a "fatigue profile". The findings of the study suggested that subjects who recorded high power output values in the early stages of a high intensity cycle ergometer test exhibited large fatigue profiles indicating an inability to maintain performance of high power and quality for long time periods.

Lactate in human skeletal muscle after 10 and 30 sec of maximal cycle ergometer performance has been measured by Jacobs et al. [8]. Twenty-two physical education students participated in the study, (15 males and 7 females). All subjects performed two bouts of exercise. On the first occasion the subjects exercised for the full 30 sec period, whereas on the second occasion the exercise time was terminated after 10 sec.

Muscle biopsies were obtained pre and post exercise on both occasions. Results showed that skeletal muscle lactate concentrations increased in males and females after 10 and 30 sec of maximal exercise. McCartney et al. [11] investigated muscle power and metabolism in intermittent exercise. Eight male subjects performed four 30 sec bouts of maximal isokinetic cycling at 100 rpm, with four minutes recovery interval. Exercise was performed on an isokinetic cycle ergometer. The final exercise period was followed by 20 minutes recovery. External work and power decreased by 20% in the second and third periods of exercise, but no further changes were observed in the fourth period. It was concluded that despite minimal flux in the third and fourth exercise periods, subjects generated 1000W peak power, and sustained 400W for 30 sec, 60% of the values recorded in the first exercise period.

Medbo and Tabata [12] examined high intensity energy release in working muscle. Sixteen males cycled as long as possible at constant powers chosen to exhaust the subjects in approximately 30 sec, 1 min or 2-3 mins. Muscle biopsies were taken before and approximately 10s after exercise and analyzed for lactate, phosphocreatine, and other metabolites. O_2 uptake was measured for determination of the accumulated O_2 deficit a whole body measure of high intensity energy release. The value recorded was then compared to the direct measures obtained from the muscle metabolites. Muscle lactate concentration increased by 30 ± 1.2 $mmol/kg^{-1}$ and muscle PCr concentration fell by 12.4 ± 0.9 $mmol/kg^{-1}$ during the 2-3 min of exhausting exercise ($P<0.05$).

The ATP production was 58 ± 2 $mmol/kg^{-1}$ wet muscles mass, which may be the maximum energy release for human muscle during cycle ergometry. The ATP production was 6 and 32% less for 1 min and 30s of exercise respectively, than for 2 min of exercise, suggesting that 2 min of exhausting exercise may be required for maximal use of high intensity energy sources.

Glycolysis provided three times more ATP than PCR breakdown for all exercise duration's. There were also close linear relationships between the rates of ATP production in muscle and the value estimated for the whole body by O_2 deficit ($r=0.94$, $P<0.05$). This suggests that the accumulated O_2 deficit may be quantifiable as a measure of energy release during high intensity cycle ergometry.

Sahlin et al. [13] stated that during high intensity exercise there is a pronounced accumulation of lactic acid in the body fluids and acidosis is often implicated as a cause of fatigue. *In vitro* studies of skinned muscle fibres have shown that Ca sensitivity [14], maximal tension [14,15] and shortening velocity [15] are reduced during acidotic conditions. However, it has been recently shown that these effects of acidosis were absent at a more physiological temperature [16].

Although acidosis correlates with impaired performance during exercise, improvements in performance have been recorded during the recovery period [7,17,18]. Thus during the first two minutes after sustained contraction to fatigue there is a near complete recovery of force whereas muscle pH remains depressed [17]. These findings indicate that the direct inhibitory effect of acidosis on the contractile machinery is negligible. An alternative hypothesis may be that the effect of acidosis on performance is indirect and is mediated through impairment of the ATP generating processes. Several enzymes in glycolysis exhibit pronounced pH sensitivity and hydrogen ions will also influence the creatine kinase equilibrium [13]. It may be possible that acidosis interferes with the contraction process indirectly through its effect on energy metabolism.

The observed relationships between muscle lactate and IMP and between blood lactate and plasma NH_3 support this contention [19].

References

1. Saltin B (1971) Guidelines for physical training. Scand J Rehabil Med 3: 39-46.
2. Hermansen L, Hultman E, Saltin B (1967) Muscle glycogen during prolonged severe exercise. Acta Physiol Scand 71: 129-139.
3. Gollnick PD, Piehl K, Saltin B (1974) Selective glycogen depletion pattern in human muscle fibres after exercise of varying intensity and at varying pedalling rates. J Physiol 241: 45-57.
4. Gollnick PD, Armstrong RB, Sembrowich WL, Shepherd RE, Saltin B (1973) Glycogen depletion pattern in human skeletal muscle fibres after heavy exercise. J Appl Physiol 34: 615-618.
5. Hargreaves M, Richter EA (1988) Regulation of skeletal muscle glycogenolysis during exercise. Can J Sport Sci 13: 197-203.
6. Essen B, Kaijser L (1978) Regulation of glycolysis in intermittent exercise in man. J Physiol 281: 499-511.
7. Nevill ME, Bogdanis GC, Boobis LH, Lakomy HK, Williams C (1996) Muscle metabolism and performance during sprinting. In: Maughan, Shereffs (Eds) 243-260.
8. Jacobs I, Tesch P, Bar-Or O, Karlsson J, Dotan R (1983) Lactate in human skeletal muscle after 10 and 30 secs of supramaximal exercise. J Appl Physiol Respir Environ Exerc Physiol 55: 365-367.
9. Harris RC, Soderlund K, Hultman E (1992) Elevation of creatine in resting and exercised muscle of normal subjects by creatine supplementation. Clin Sci 83: 367-374.
10. Boobis L, Williams C, Wootton SA (1982) Human muscle metabolism during brief maximal exercise. J Appl Physiol 75: 712-719.
11. McCartney N, Spriet LL, Heigenhauser GJ, Kowalchuk JM, Sutton JR, et al. (1986) Muscle power and metabolism in maximal intermittent exercise. J Appl Physiol 60: 1164-1169.

12. Medbø JI, Tabata I (1993) Anaerobic energy release in working muscle during 30 s to 3 mins of exhausting bicycling. J Appl Physiol 75: 1654-1660.
13. Sahlin K, Tonkonogi M, Soderlund K (1998) Energy supply and muscle fatigue in humans. Acta Physiol Scand 162: 261-266.
14. Godt RE, Nosek TM (1989) Changes of intracellular milieu with fatigue or hypoxia depress contraction of skinned rabbit skeletal and cardiac muscle. J Physiol 412: 155-180.
15. Cooke R, Franks K, Luciani GB, Pate E (1988) The inhibition of rabbit skeletal muscle contraction by hydrogen ions and phosphate. J Physiol 395: 77-97.
16. Pate E, Bhimani M, Franks-Skiba K, Cooke R (1995) Reduced effect of pH on skinned rabbit psoas muscle mechanics at high temperatures: implications for fatigue. J Physiol 486: 689-694.
17. Sahlin K, Ren JM (1989) Relationship of contraction capacity to metabolic changes during recovery from a fatiguing contraction. J Appl Physiol 67: 648-654.
18. Fitts RH (1994) Cellular mechanisms of muscle fatigue. Physiol Rev 74: 49-94.
19. Sahlin K, Katz A, Broberg S (1990) Tricarboxylic acid cycle intermediates in human muscle during prolonged exercise. Am J Physiol 259: 834-841.