Adipocytic Contribution to Lactate Production in Male Athletes of West African Descent

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ABSTRACT

Lactate is of particular concern to athletes, especially in the running events, as its production and accumulation are associated with muscular fatigue. To optimise performance it is important to fully understand the biomechanical and physiological factors that influence lactate production. This study aimed to improve this understanding by examining the effects of body fat on basal lactate production. Thirty-five individuals (19 athletes and 16 non-athletes) of similar ethnic backgrounds had their body fat assessed and their blood lactate measured before and after an intense bout of running. Significance was observed for pre-exercise basal lactate in the athletes ($p=0.005$) but not in the non-athletes ($p=0.026$). A 17% increase in body fat in athletes accounted for an additional 2 mmol/L of basal lactate while a 15% increase in non-athletes resulted in 1.5 mmol/L of basal lactate. No significance was observed between adiposity and lactate concentration post-exercise. The authors concluded that body fat has a significant impact on pre-exercise basal lactate level, possibly affecting the athlete's cellular metabolic status prior to competition.

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Introduction

Exercise is dependent on muscular contraction, which is powered by the catabolism of adenosine triphosphate (ATP)[1]. It has been estimated that the stored cellular ATP in the human body is approximately 8 mmol/kg of wet muscle and that this can fuel intense exercise for a few seconds[1].

The body has several mechanisms to satisfy the muscles' energy requirement when the ATP supply is low. The phosphagen, glycolytic and mitochondrial respiration systems all work together contributing to the production of ATP, their contributions varying depending on the quantity and duration of the exercise[1]. Research has shown that the phosphagen system, which utilises creatine phosphate, supplies the bulk of energy during the initial 10

seconds of intense exercise[2]. The concentration of creatine phosphate, which accounts for approximately 26 mmol/kg wet weight of muscle, begins to decline 1.3 seconds following the start of intense exercise[1]. Glycolysis occurs in conjunction with the other energy systems during exercise but the maximal rate of regeneration of ATP takes place between 10 to 15 seconds of exercise, kicking in after energy from the phosphagen system has been depleted[1].

Unlike the phosphagen and glycolytic systems, mitochondrial respiration, including the Krebs cycle, relies on the presence of sufficient oxygen to produce fuel. During intense physical exercise, when the oxygen supply is inadequate to burn fuel fast enough to supply the energy needed, the body employs the anaerobic or glycolytic pathway. During hypoxia, the glycolytic pathway converts pyruvate to lactate through the action of lactate dehydrogenase, resulting in the regeneration of an oxidised form of nicotinamide adenine dinucleotide (NAD^+)[3]. This prevents the accumulation of pyruvate and allows glycolysis to continue using the regenerated NAD^+[3].

Conflicting views exist regarding the accumulation and fatigue inducing effect of lactate. Some researchers believe that increased lactate production occurs simultaneously with cellular acidosis and as a result can be used as a good indicator of the cells' metabolic status[3]. Some believe that if lactate production did not occur, the effects of cellular acidosis would be more pronounced and physical activity would be severely hampered[3].

A more recent review, however, indicates that lactate production actually does contribute to the muscular fatigue experienced during exercise[4]. Lactic acid, like all acids, dissociates in aqueous solution. The blood is aqueous and as such 99% of the lactic acid produced dissociates into lactate anions (La^-) and hydrogen ions (H^+). The hydrogen ions and lactate anions can increase to very high levels during exercise. Research suggests that the hydrogen ions released can inhibit sarcoplasmic ATPase and competitively inhibit Ca^{2+} binding to troponin C, which could depress muscle function[5].

In light of the known association of lactate production with the muscular fatigue experienced by sprinters and middle-distance athletes, it becomes important to understand fully the biochemical and physiological factors dictating lactate production. This knowledge can aid in controlling the level of production so that optimised performance can be achieved.

Some studies have reported that adipocytes contribute approximately 3% of the glucose metabolised, however after revision it was noted that they contribute as much as 30% of the glucose metabolised to lactate, pyruvate, triglycerides and carbon dioxide[6]. More obese rats were found to convert up to a half of the glucose ingested to lactate while smaller rats that were fed ad libitum converted up to 15% of glucose to lactate[7].

There seems to be a positive correlation between the size of the rat fat cell and the quantity of produced lactate with similar results found in humans[7, 8]. A study done on lean and obese men indicated that subcutaneous fat releases a significant amount of lactate and this was markedly increased in obese individuals[9].

Athletics is, of course, highly competitive and in the effort to optimise performance within the rules and gain advantage fairly, consideration must be given to all physiological and biochemical parameters contributing to performance. Such considerations must include the correlation of basal lactate production to adiposity, hypoxia and muscular fatigue, which the literature indicates retards performance. This study on the effects of body fat on basal lactate production adds to the data on adipocytic contribution to lactate metabolism.

Method

Subjects

Two groups of subjects were selected for this study. One group consisted of 19 athletes currently training with an IAAF certified coach while the second group consisted of 16 relatively sedentary individuals (non-athletes) who served as controls. The athletes were selected from the University of the West Indies (Mona), Jamaica, track and field team and an athletics club that trains on university campus. The non-athletes were volunteer male students from the same university. All the subjects were Jamaicans of West African descent aged between 18 and 30 years.

Each participant was given a consent form detailing the procedure of the study and asked to sign before participating. Ethical approval for the conduct of the research was granted by the University Hospital of the West Indies/University of the West Indies/Faculty of Medical Sciences Ethics Committee.

Questionnaires were used to determine if the participants were healthy as well as the level and quantity of exercise they did weekly. This tool enabled appropriate distinction between the two groups. From the data provided on their questionnaires, all the participants were deemed healthy with no history of serious illness. On average, the athletes trained approximately five times per week, two hours per session, while the non-athletes trained for less than 20 minutes per week.

Prior to the test, measurements for height, weight, hip, and waist circumference were taken, which allowed the matching of the two groups for body mass index (BMI).

Procedure

The participants were requested to fast and rest overnight. A pre-exercise fasting blood sample was collected and tested for lactate concentration using a hand-held Lactate Plus analyser. Prior to blood being taken, an area on the forefinger was cleaned using 70% isopropyl alcohol. The finger was then pricked with a lancet, and a drop of blood was placed on the analysis strip whereupon a read-out of lactate concentration was obtained.

Body fat percentages were measured using the BodyMetrix analyser. With this method the three site technique was utilised, employing a modified version of the Jackson-Pollock 3 site skinfold equation. The analyser measured the thickness of the fat using ultrasound technology[15]. Three sites on the right side of the body were identified. With the BodyMetrix placed at the chest mid-way between the nipple and shoulder joint, a read out was displayed on a computer, which recorded the thickness of each tissue in millimetres (mm). This was performed two to three times with the average score recorded. This procedure was repeated at two other areas, an inch right of the umbilicus and on the thigh mid-way between the knee and hip joints. From the readings obtained the percentage body fat was calculated for each participant.

For comparative purposes the Harpenden skinfold calliper was used to measure body fat using the Jackson-Pollock 3 site skinfold equation[16]. The percentage body fat was determined using the Siri equation[17]. In using the Harpenden skinfold calliper the same three sites were also used. Each site was pinched with the index finger and the thumb, with the calliper used to measure the thickness (mm) of the skinfold one centimetre away from the fingers. For each site the measurement was repeated within 1mm of each other and an average recorded. The equations used to calculate the body fat are summarised as follows:

Jackson-Pollock 3 Site Skinfold Equation[16]

Body Density of Males = 1.1093800 - 0.0008267 (X2) + 0.0000016 (X2)2 - 0.0002574 (X3)
X2 = Sum of Chest Abdomen and Thigh
X3 = Age

Siri Equation[17]

Fat (%) = [(4.95/body density) - 4.51] x 100

After the measurements the participants performed an intense bout of physical exercise, running on a treadmill set at a 10% incline for 90 seconds at a speed of six miles/h (approximately 9.8 km/h). Immediately following the run, a post exercise lactate test was done. The results were analysed using SPSS 17 with the level of significance set at $p \leq 0.05$.

Results

The anthropometric features of the subjects were not significantly different when the two groups were compared ($p>0.05$) and the two groups were distinguished by their level of regular physical activity.

The results from the BodyMetrix analyser correlated positively with the Harpenden skinfold calliper ($p=0.01$). The Harpenden calliper however underestimated the body fat of both the athletes and non-athletes by an average of 3%.

The basal lactate concentrations pre- and post-exercise were lower in the athletes when compared to the control subjects, however the differences were not significant ($p>0.05$). There was a significant positive correlation with adiposity and pre-exercise basal lactate concentration. In the athletes, an increase in body fat percentage from 3% to 20% resulted in approximately 2 mmol/L of additional pre-exercise basal lactate. A 15% change in body fat in the non-athletes corresponded with a 1.5 mmol/L increase. When the body fat percentage was 4% in the athlete group, the corresponding pre-exercise basal lactate concentration was 1 mmol/L, while at 19% the concentration was 3 mmol/L. The non-athletes registered 1 mmol/L and 2.5 mmol/L, respectively. Although lactate increased after exercise in both groups, the results indicated that there was no significant correlation between adiposity and basal lactate concentration post-exercise.

Discussion

The BodyMetrix analyser is very effective in measuring body fat percentage[18]. It uses ultrasound technology, releasing sound waves in order to record an image. The sound waves pass through each layer of tissue whereupon an echo is created and transmitted back to the machine. The strength of the echo is used to create an image that measures the thickness of each tissue[18]. The BodyMetrix analyser has been found to produce comparable results with the skinfold method of body fat assessment. The advantage of using the BodyMetrix analyser is that no reading was detected until the analyser was placed directly perpendicular to the surface of the skin ensuring increased accuracy and precision[19]. The study confirmed a significant positive correlation between the Harpenden skinfold calliper method and the BodyMetrix analyser. However, the calliper

Table 1: Comparisons of anthropometrical characteristics and lactate concentrations between male athletes and non-athletes of West African descent

	ATHLETES	NON-ATHLETES
Age (years)	21.47±1.95	22.25±2.52
Body Fat %	8.29±5.42	10.06±4.31
BMI (kg/m^2)	23.31±3.33	21.33±2.93
WHR	0.79±0.03	0.79±0.03
Mean Basal Lactate (mmol/L)	1.53±1.23	1.69±0.83
Post Exercise Lactate (mmol/L)	6.71±3.29	8.36±3.91

Data presented as mean ± SD. P≤0.05 is significant.

method underestimated the total body fat percentage by 3%, pointing to the subjectivity of the method as indicated by the literature[20].

The body's ability to perform at its maximum potential is dependent on a variety of conditions. Training, mental fitness and diet are essential factors to consider when trying to improve and increase physical performance. Genetics has also been suggested to play a significant role[10]. In order to ensure that accurate comparisons between the groups were made, the athletes and non-athletes selected for the study were all Jamaicans of West African descent[21]. The two groups were matched for age and BMI, with undetectable visual differences in adiposity, in order to assess the effects of the subtle differences in adiposity on basal lactate production with exercise.

Fitness is associated with lower lactate production. This study showed that increased fat elicited an elevation of basal lactate, even in fit individuals. The results (Figure 1 and Figure 2) indicated that an increase in body fat resulted in an increase in pre-exercise basal lactate. These associations expressed significance at $p=0.005$ and $p=0.026$ respectively in the two groups. In the athletes, an approximately 17% increase in body fat resulted in an additional 2mmol/L of pre-exercise basal lactate while a 15% increase in body fat resulted in an additional 1.5mmol/L in the non-athletes. Since the groups were anthropometrically similar, the results presented an accurate correlation between basal lactate production and adiposity of individuals at opposite ends of the fitness spectrum.

As athletes are expected to have more lean mass/muscles and to be more efficient at using a larger volume of oxygen during exercise due to increased VO_2 max[11], their post-exercise lactate level is expected to be lower than that of unfit individuals with less muscle mass. The treadmill in this study was set at an incline and the duration of the exercise was set at 90 seconds to ensure that the lactate threshold for each individual would be exceeded. In exceeding the lactate threshold there is an abrupt increase in lactate concentration[12]. Without exceeding the threshold it would not have been possible to make the association between post-exercise lactate and adiposity. The study confirmed that basal lactate concentration post-exercise in the athletes was less compared to the controls (6.71±3.29mmol/L versus 8.36±3.91mmol/L). There was however no significant difference between the two groups in basal lactate concentration post-exercise.

The fatigue associated with greater production and build-up of lactate is a major disadvantage for the athlete. A better understanding of how this occurs would aid in optimising athletic performance[13]. Obesity can significantly increase lactate production[9]. Decades ago, it was proposed that fat may contribute to lactate produced during exercise[14]. In the present study there was no significant association between adiposity and basal lactate concentration post-exercise in either group. Even though no significant association was found during exercise, it is not definitive as the lack of significance observed may be due to the short duration of the exercise.

It was observed that even though the non-athletes had a larger mean body fat percentage, their mean BMIs were lower than those of the athletes. The traditional method of determining BMI using the weight to height ratio (kg/m^2) may not be the best method of determining normal body profile as indicated by this study.

Several studies have shown that lactate accumulation retards performance and that the duration of physical activity is extended when there is limited increase in lactate concentration[22, 23]. To increase performance, athletes participate in vigorous training activities. Training is said to reduce the basal lactate concentration as well as the peak lactate concentration[24]. The present study confirmed that the well-trained athletes produced less basal lactate pre-exercise (1.53±1.23 mmol/L versus 1.69±0.83 mmol/L) compared to the untrained controls.

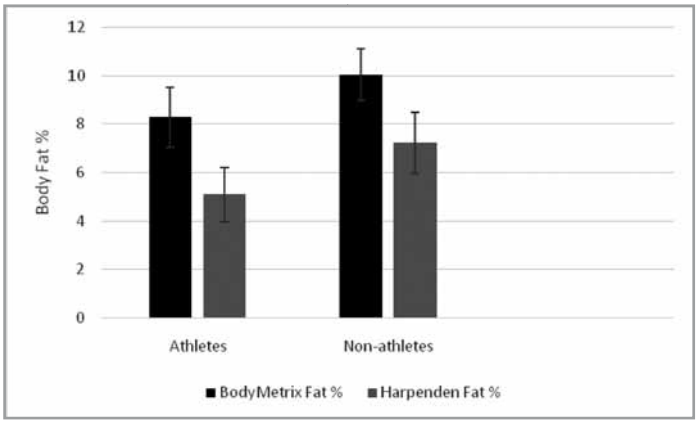

Figure 1: A comparison between the Harpenden skinfold caliper and the BodyMetrix analyser in measuring body fat percentage (Note: the body fat percentage was significantly underestimated by the Harpenden caliper (P<0.05) even though there was a positive correlation with the Bodymetrix analyser.)

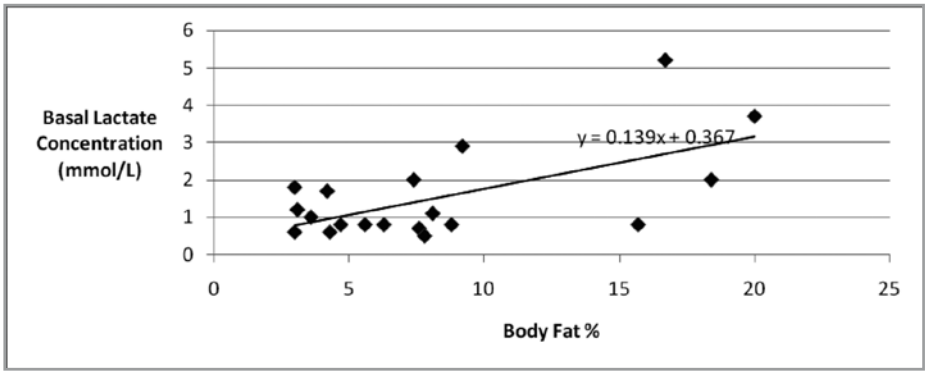

Figure 2: The association of body fat % with pre-exercise basal lactate concentration in athletes (Note: there was a significant positive association between body fat percentage and pre-exercise basal lactate concentration in the athletes. The association had a P value of 0.005.)

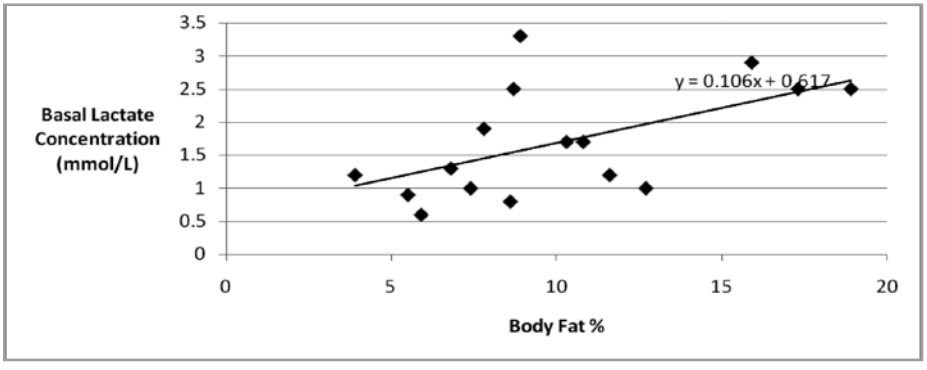

Figure 3: The association of body fat percentage with pre-exercise basal lactate concentration in non-athletes (Note: there was a significant positive association of body fat percentage to pre-exercise basal lactate in the controls (P=0.026).)

The greater significance of basal lactate with adiposity in the athletes demonstrated that fitness correlated well with the level of fat cells, lactate production and ultimately performance. This study therefore confirmed that adiposity is important in performance and lactate dynamics.

Conclusion

The BodyMetrix analyser is the more accurate method of assessing body fat percentage when compared to the Harpenden calliper, as it is less subjective. Lactate concentration is indicative of fitness levels but is significantly influenced by adiposity. Basal lactate can be used as a tool for athletes in various sporting disciplines as a means of monitoring their body profile and fitness status. Pre-exercise basal lactate can indicate an athlete's potential to develop fatigue during training/competition as a higher pre-exercise concentration indicates that the corresponding hydrogen ion concentration will likewise be high and the athlete may be hypoxic. Knowledge of the specific mechanisms via which adipocytes contribute to the production of lactate would be very significant and should be explored further in performance studies.

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REFERENCES

1. BAKER, J.S.; MCCORMICK, M.C. & ROBERGS, R.A. (2010). Interaction among Skeletal Muscle Metabolic Energy Systems during Intense Exercise. ***Journal of Nutrition and Metabolism*** Article ID 905612, 13 pages.

2. BOGDANIS, G.C.; NEVILL, M.E.; BOOBIS, L.H. & LAKOMY, H.K. (1996). Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. ***Journal of Applied Physiology;*** 80 (3): 876-884.

3. ROBERGS, R.A.; GHIASVAND, F. & PARKER, D. (2004). Biochemistry of exercise-induced metabolic acidosis. ***American Journal of Physiology, Regulatory, Integrative and Comparative Physiology***; 287 (3): 502-516.

4. LINDINGER, M.I. (2005). Intracellular [H+]: a determinant of cell volume in skeletal muscle. ***The Journal of Physiology***; 563 (3): 643.

5. GLADDEN, L.B. (2004). Lactate metabolism: a new paradigm for the third millennium. The Journal of Physiology; 558: 5-30.

6. DIGIROLAMO, M.; NEWBY, F.D. & LOVEJOY, J. (1992). Lactate production in adipose tissue: a regulated function with extra-adipose implications. ***FASEB***; 6 (7): 2405-2412.

7. CRANDALL, D.L.; FRIED, S.K.; FRANCENDESE, A.A.; NICKEL, M. & DIGIROLAMO, M. (1983). Lactate release from isolated rat adipocytes: influence of cell size, glucose concentration, insulin and epinephrine. ***Horm. Metab Res***; 15: 326-329.

8. SANDQVIST, M.M.; ERIKSSON, J.W. & JANSSON, P.E. (2001). Increased Lactate Release per Fat Cell in Normoglycemic First-Degree Relatives of Individuals With Type 2 Diabetes. ***Diabetes***; 50 (10): 2344-2348.

9. JANSSON, P.A.; LARSSON, A.; SMITH, U. & LONNROTH, P. (1994). Lactate release from the subcutaneous tissue in lean and obese men. ***The Journal of Clinical Investigation***; 93 (1): 240-246.

10. OSTRANDER, E.A.; HUSON, H.J. & OSTRANDER, G.K. (2009). Genetics of athletic performance. ***Annual Review of Genomics in Human Genetics***; 10: 407-429.

11. FLEG, J.L. & LAKATTA, E.G. (1988). Role of muscle loss in the age-associated reduction in VO2 max. ***Journal of Applied Physiology***; 65 (3): 1147-1151.

12. KRAVITZ, L. & DALLECK, L. (2005). Lactate threshold training. ***Network, the Official Magazine of Australian Fitness Network***; 27-30.

13. FACEY, A.; IRVING, R. & DILWORTH, L. (2013). Overview of Lactate Metabolism and the Implications for Athlete. ***American Journal of Sports Science and Medicine***; 1 (3): 42-46.

14. JONES, N.L.; HEINGENHAUSER, G.J.; KUKSIS, A.; MATSOS, C.G.; SUTTON, J.R. & TOEWS, C.J. (1980). Fat Metabolism in Heavy Exercise. ***Clinical Science***; 59: 469-478.

15. JOHNSON, K.E.; MILLER, B.; JUVANCIC-HELTZEL, J.A.; AGNOR, S.E.; KIGER, D.L.; KAPPER, R.M. & OTTERSTETTER, R. (2014). Agreement between ultrasound and dual-energy X-ray absorptiometry in assessing percentage body fat in college-aged adults. ***Clinical Physiology and Functional Imaging***; doi: 10.1111/cpf.12150

16. JACKSON, A. & POLLOCK, M. (1978). Generalized equations for predicting body density of men. ***British Journal of Nutrition***; 40: 497-504.

17. DURNIN, J.V. & RAHAMAN, M.M. (1967). The assessment of the amount of fat in the human body from measurements of skinfold thickness. ***British Journal of Nutrition***; 21 (3): 681-689.

18. WAGNER, D.R. (2013). Ultrasound as a Tool to Assess Body Fat. ***Journal of Obesity***; doi: 10.1155/2013/280713

19. ULBRICHT, L.; NEVES, E.B.; RIPKA, W.L. & ROMANELI, E.F. (2012). Comparison between body fat measurements obtained by portable ultrasound and calliper in young adults. ***Engineering in Medicine and Biology Society (EMBC), Annual International Conference of the IEEE***; 1952-1955.

20. WOMERSLEY, J. & DURNIN, J.V. (1973). An Experimental Study on Variability of Measurements of Skinfold Thickness on Young Adults. ***Human Biology***; 45 (2): 281-292.

21. DEASON, M.; SCOTT, R.; IRWIN, L.; FUKU, N.; TANAKA, M.; IRVING, R. & CHARLTON V. (2012). Importance of mitochondrial haplotypes and maternal lineage in sprint performance among individuals of West African ancestry. ***Scandinavian Journal of Medicine & Science in Sports***; 22 (2): 217-223.

22. SJODIN, B. & JACOBS, I. (1981). Onset of Blood Lactate Accumulation and Marathon Running Performance. ***International Journal of Sports Medicine***; 2 (1): 23-26.

23. FARRELL, P.A.; WILMORE, J.H.; COYLE, E.F.; BILLING, J.E. & COSTILL, D.L. (1979). Plasma lactate accumulation and distance running performance. ***Medicine and Science in Sports***; 11 (4): 338-344.

24. MANNA, I. & KHANNA, G.L. (2013). Effect of Training on Selected Biochemical Variables of Elite Male Swimmers. ***American Journal of Sports Science and Medicine***; 1 (2): 13-16.