International Journal of Sports Science 2015, 5(2): 59-64
DOI: 10.5923/j.sports.20150502.03

Glycemic and Blood Lactate Response to Maximal Incremental Treadmill Test

Filipe Dinato de Lima[1,*], Ricardo Jacó de Oliveira[1], Ana Luiza Matias Correia[1], Eduardo Silva Trindade[2], Renata Aparecida Elias Dantas[2], Márcio Rabelo Mota[2]

[1]University of Brasília - UnB, Brasília, Brazil
[2]Universitary Center of Brasília - UniCEUB, Brasília, Brazil

Abstract During the incremental test, after reaching the anaerobic threshold, the lactate concentration increases exponentially. Additionally, the blood glycemia changes with the intensity and duration of the exercise. The present study aimed to verify the lactate and glycemic responses to a maximal incremental non-incline treadmill test. For that, 17 individuals, volunteers, were submitted to an incremental treadmill test, with an initial speed of 5 km/h and 1 km/h increments every minute, with no inclination. Before the tests, the volunteers consumed a breakfast meal containing 60.61% carbohydrate, 12.91% protein and 26.48% lipids. For the analysis of the blood lactate, were collected two blood samples: at rest and from 3 to 5 minutes after the test. For the analysis of the blood glycemia, were collected three blood samples: fasting, at rest 15 minutes after the provided meal, and from 3 to 5 minutes after the test. The Shapiro Wilk test was used to verify the normality of the data. For the lactate analysis, was applied the student's t-test. For the glycemia analysis, was applied the ANOVA for repeated measures, with post hoc Bonferroni. The level of significance of the study was $p < 0.05$. The results indicated a significant increase in the lactate after the exercise ($p = 0.002$). The glycemia increased significantly after the exercise in relation to the fast ($p = 0.015$) and the rest ($p = 0.042$). Therefore, the maximal incremental in a non-incline treadmill test was capable of increasing significantly the lactate concentration and the blood glycemia.

Keywords Exercise test, Lactic acid, Blood glucose

1. Introduction

The glucose anaerobic reaction has as its final product lactic acid that, as it dissociates, produces lactate and hydrogen ions. In excess, these subproducts of the glycolytic metabolism reduce the PH of the cells, causing the metabolic acidosis [1, 2]. The lactate shows little effect over the muscular concentration, and it is not responsible for every negative effect on the organism [1, 3]. In this sense, the elevation on the lactate concentration seems not to be related to the diminution of the sports performance. [4] However, the presence and accumulation of H+ is a classic cause of muscle fatigue, causing the inhibition of the Glycogen Phosphorylase and Phosphofructokinase metabolic enzymes, and, consequently, of the glucose process [2, 3].

After the exercise, the concentration of lactate and H+ are similar, in a proportion of 1:1 [5]. For this reason, although having little influence on the muscle fatigue, the lactate shows a representative estimate of the acidosis, in addition to being a crucial mediator of the energy resynthesis metabolism on the skeletal muscle, brain and heart [1, 6, 7]. It is still not clear on the literature the real influence of the acidosis on the muscle fatigue [7]. Its effects seem to be relevant in an indirect way, through the activation of nerves of the groups III and IV of the afferent pathways, increasing the feeling of discomfort [3, 8].

According to Beneke [9], intensities of exercise in which the lactate concentration is maintained below 4 mmol represent the moment that the glycolytic production rate of lactate is equalized with the pyruvate conversion rate in the presence of oxygen. However, after exceeded the threshold value (4mmol), the lactate concentration increases exponentially [10]. In this sense, the capacity of the Lactate Dehydrogenase enzyme on the formation of pyruvate seems to be unlimited, considering the high oxidation capacity of the lactate [6].

Furthermore, exercises performed in hypoxia result in an increase of the blood lactate, suggesting a positive relation between the absence of oxygen for the muscle work and the production of lactate [11]. Such relation can be proved by the fact that there is a higher velocity of post exercise removal of lactate in individuals that present a higher maximum muscle oxidative capacity [12] and an apparent positive relation between the increasing of the aerobic capacity and the peak of production of the this metabolite [13].

* Corresponding author:
fdinatolima@gmail.com (Filipe Dinato de Lima)
Published online at http://journal.sapub.org/sports

In intense exercises, the glucose is the main fuel of the muscles in activity [14]. Its behavior is related to the increase of the catecholamines level, producing an increase in the glucose production between 7 and 8 times superior in relation to rest, while its utilization increases just between 3 and 4 times [14]. Thopson et al. [15], on the other hand, affirm that the blood glycemia suffers little alteration, even in vigorous exercises, for the degradation of the muscle glycogen and the reduction of the insulin resistance favor the maintenance of the blood glucose levels. Such uncertainties about the mechanisms and exercises that affect the blood glycemia stimulate studies such as this one.

The maximal test is the most indicated and precise on the evaluation of the cardiorespiratory capacity and maximal aerobic power, in addition of being more sensitive on the detection of cardiovascular diseases in asymptomatic individuals [16]. However, the protocols usually utilized involve increments on the inclination. In this sense, the aim of this study was to verify the glycemic and blood lactate response to a maximal incremental non-incline treadmill test.

2. Methods

The present study was approved by the Committee for Ethics in Research of Universitary Center of Brasília. All the experimental procedures were performed on the Human Physiology Laboratory and all volunteers agree with the terms of this study.

2.1. Participants

The sample was composed by 17 individuals, physically active, with minimum practice of exercises of 3.5 hours per week for at least 6 months. Were excluded alcoholic and/or smoker individuals who presented any hormonal or cardiac dysfunction, endocrine diseases and hypertension. These information were collected through a questionnaire during the initial anamnesis Were also excluded individuals that made use of stimulating supplements both natural and synthetic 48 hours before the test, thus avoiding, the values of lactate to be overrated, beside volunteers with restrictions to realization of maximum strain, such as osteoarticular problems and myopathies, avoiding the underestimation of the lactate peak by virtue of a precocious interruption of the test.

2.2. Procedures

The volunteers attended to the place of the test in two different days. The first day was designated to the anamnesis and the measurement of variables of sample characterization. The researchers explained all the procedures. Height and body mass were collected.

At the second day, the volunteers attended to the laboratory after fasting for 12 hours. It was provided balanced breakfast meal to all the individuals on the research. The meal consisted of 50 grams of integral bread, 10 grams of cheese, 5 grams of butter, one unity of banana and 200 milliliters of integral grape juice. The nutritional values of the meal are exposed in table 1.

After the meal, the volunteers remained at rest in the sitting position for 15 minutes. After the end of the rest, the individuals were submitted to a maximal incremental test in a Centurion 300 treadmill Micromed. The protocol consisted in an initial speed of 5 km/h with increments of 1 km/h every 1 minute. It is understood that, with this protocol, the muscle fatigue caused by inclinations on the treadmill was avoided, favoring the execution of the maximal test, with no underestimation of it. In this sense, stages with duration of 1 minute promoted quick increment and a higher VO2 reached at the test, as proposed by Machado et al. [17]. The exercise was interrupted when the perceived exertion reached 17 on the Borg scale, or was signalized voluntary exhaustion. The analysis of the cardiorespiratory fitness was made through a ventilometer of CEFISE biotechnology.

Table 1. Energy Value and Amount of Each Macronutrient Ingested on the Meal Before the Test

Nutrient	Energy Value	Quantity
Lipids	121	13.45 (26.48%)
Carbohydrates	277	69.23 (60.61%)
Proteins	59	14.88 (12.91%)
Fibers	-	6
Total Energy Value	457	-

2.3. Blood Collection

The blood lactate was measured after the volunteer remained at rest for 15 minutes and between 3 and 5 minutes after the end of the test. The glycemia was measured after fasting, after the rest, and 3 to 5 minutes after the end of the test.

The blood collection was made through a puncture on the tip of the index finger, on the dominant hand, after local asepsis with alcohol 70%. The first drop of blood was discarded, and on the sequence, a sample of approximately 3 µl of blood was analyzed in a monitor of lactate Accutrend Plus (Roche Brasil, São Paulo, Brazil), properly validated [18].

2.4. Statistical Analysis

The normality of the data was verified through the Shapiro-Wilk test. Descriptive statistics with mean and standard deviation was used on the variables of sample characterization. The comparison between the concentration of lactate at rest and after the maximal test was analyzed with the student's t-test. The comparison between the glycemia after fasting, at rest and after the maximal test was analyzed through an ANOVA analysis of variance for repeated measures, with Bonferroni post hoc. All the statistic procedures were performed on the software SPSS version 21.0. Was adopted as level of significance in all the tests $p < 0.05$.

3. Results

The maximal incremental test lasted 8.46 ± 2.36 minutes.

The variables of sample characterization are exposed in table 2. The value of $VO_{2máx}$ was estimated by the incremental test with use of ventilometer of CEFISE biotechnology.

Table 2. Variables of Sample Characterization Expressed in Mean ± Standart Deviation

Age	22.50 ± 3.26
Body Mass	73.41 ± 12.99
Stature	1.71 ± 0.11
BMI	24.02 ± 4.12
VO_2 Máx	40.64 ± 7.97

The behavior of the lactate concentration at rest and after the maximal incremental test is presented in table 3. It is noted a significant elevation on the post exercise in relation to the rest (p = 0.002).

Table 3. Values of Lactate Concentration Before and After Incremental Test Expressed in Mean ± Standart Deviation, and Significance

Pre	Post	p
2.53 ± 0.77	7.36 ± 2.92	0.002

The behavior of the glycemia after fasting, at rest and after the maximal incremental treadmill test is represented in table 4. It is noted a significant increase in the glycemia post exercise in relation to the fast (p = 0.015) and on the glycemia post exercise in relation to the rest (p = 0.042). The comparison between glycemia after fasting and glycemia at rest did not present a significant difference; however, it presented a difference leaning to significance (p = 0.058).

Table 4. Values of Blood Glycemia after Fasting, Before and After the Incremental Test Expressed in Mean ± Standart Deviation

Fast	Pre	Post
85.82 ± 9.43	96.82 ± 12.19	109.00 ± 15.32*#

* Significant difference in relation to the fast (0.015).
# Significant difference in relation to the pre exercise (0.042).

4. Discussion

The present study analyzed the lactate and glycemic responses induced by a maximal incremental non-incline treadmill test. Its results point to a significant increase in the lactate concentration after the exercise, corroborating with previous studies [6, 12, 17, 19]. This increase represents the utilization of the glycolytic system, for the lactate presents itself as an important mediator of the anaerobic metabolism of energy resynthesis [6, 7].

The lactate concentration increases in an incremental test from the time when the anaerobic threshold is reached. This term represents a point on which, during an incremental exercise, the increase in the lactate concentration is followed by a proportional reduction on the bicarbonate concentration, responsible for the tamponade [20]. Other metabolic alterations occur after the anaerobic threshold is reached, including the enhancement of the use of glucose as substrate process, the glucose resulting, besides an increase in the lactate concentration, in a metabolic acidosis and an increase in the VCO_2 [6, 7, 20]. Another factor that seems to influence on the lactate concentrate in intensities superior to the anaerobic threshold is the reduction of the plasma volume. However, such influence is not confirmed yet [21].

During exercise, the blood lactate concentration seems to be positively associated to the stimulation of the sympathetic nervous system and epinephrine release, provoking activation of the beta adrenergic receptors and promoting the glycogenolysis [22]. This substance inhibits the insulin and activates the glucagon, with stimulation of glycogen degradation through the activation of the enzyme glycogen phosphorylase and increase of the lactate production [23-25]. However, the influence of the epinephrine release on the lactate production was denied by Chudalla et al. [26]. For these authors, the increase in the concentration of this metabolite is result of the alteration on the ratio of muscle mass involved on the performance of the exercise and the volume of distribution of oxygen.

Variations on the lactate concentration induced by the exercise seem to be influenced by several factors. Young individuals present higher final lactate concentrations, demonstrating a low capacity of production in elderly individuals [27]. On the other side, children present a reduced lactate production in relation to adults, with lower values of the anaerobic threshold, due to a lower muscle production capacity of this metabolite, induced by a reduced presence of glycolytic enzymes [28]. In this sense, the low value of the standard deviation on the age variable seems to be fundamental on the validity of the present study.

As for the behavior of the glucose, the results of the present study point a significant increase in the post-exercise glycemia in relation to the pre-exercise and after fasting. This finding is opposite to proposed by Colberg et al. [29], that affirms that the blood glycemia tends to be reduced after the exercise, although being dependent of the type, duration and intensity of the activity.

In this sense, the blood glycemia seems to decrease after the exercise due to the increase in the insulin sensitivity of the muscle in exercise [30]. Indeed, the behavior of the glycemia is mediated during the exercise by the response of the insulin and glucagon hormones, in addition to the stimulation of catecholamines and lipase activity [31].

Unlike the previous studies, the results here presented indicated an increase in the blood glucose concentration, corroborating with performed study, with intensities superior to the glycemic and anaerobic threshold [32], however this study of Simões et al. [32] was performed without feed, so this similar results can not be applied in a general manner. This increase in the glycemia is due to the stimulation of the sympathetic nervous system, with epinephrine release and glycogenolysis stimulation [22], however it could be related to the meal provide before the test. Levels of blood glucose superior to the rest are justified by the intensification on its

production, from 7 to 8 times higher than before the exercise, in contrast to its utilization, just 3 to 4 times superior in relation to the rest [14].

The release of glucose into the blood is related to depletion of liver glycogen stimulated by hormones glucagon and catecholamines [22, 31]. In this sense, the exercise performed at a low liver glycogen supply promotes a lower glycemic response and a greater increase in lactate concentration when compared to exercises performed in a normal condition, showing the influence of glycogenolysis in the elevation of blood glucose during exercise [33]. Fanelli et al. [34] affirms that irregularities in the release of glucagon also affects the insulin response, since these hormones have antagonistic actions. Thus, the response to exercise seems to be crucial in the presence of hypoglycemia or hyperglycemia generated by the effort.

In fact, Withe et al. [35] had already shown a decrease in insulin concentration in response to elevated hormones cortisol, epinephrine and norepinephrine. Their results showed a maintenance of glucose levels in sedentary individuals and an increase in trained, confirmed by this study. However, other factors also seem to influence the response of blood glucose induced by exercise. According Howlett et al. [36] α- and β-adrenergic blockade decrease the elevation of plasma glucose concentration in response to intense exercise, due to the increased peripheral glucose uptake by skeletal muscles without modification of its production.

The maximum oxygen uptake of the participants, according to the average age and the classification proposed by American College of Sports Medicine [16] is classified as very poor, providing information that, despite being active and practice regular physical activity, had a low aerobic capacity.

The meal ingested by participants prior to performing the test may also have affected the glycemic response to exercise. The combined ingestion of glucose and protein promotes a smaller rise in blood glucose compared to glucose ingestion only [37]. The carbohydrates present in foods exert great influence on blood glucose levels [38]. The meal offered to volunteers was rich in simple carbohydrates and this tends to raise blood glucose levels acutely by raising the level of insulin secreted [39]. This is due to the glycemic index of foods, explained by Jenkins et al. [40]. However, the response of blood glucose, depends on several factors including the overall composition of the meal, whereas proteins and lipids [41]. In this context, must be taken into account the glycemic load concept [42]. The glycemic load of meals has been widely used and is related to postprandial blood glucose and insulin [41]. When blood glucose levels are high, glucose transporters (GLUTs) move increasing the amount of glucose in pancreatic β-cells. Thus, there is a greater release of insulin. In this situation, hepatic glucose output is completely suppressed by insulin infusion [43].

The meal provided is in accordance with current recommendations proposed by Trumbo et al [44]. The meal was chosen in order to standardize the food intake before the test. It provides sufficient amount of carbohydrates for breakfast and not change the performance in the test.

This study presented a few limitations, such as low number of individuals. Furthermore, due to the high variation of the blood lactate response depending on the age and the quantity of muscle mass, the findings presented here should not be generalized to populations with different features. Another limitation of this research was the blood collection performed in few moments. In addition, research has not been done in conditions without food or without exercise. Therefore, the present study shows the results for only the experimental condition studied here. Therefore, it is suggested for future studies that the lactate concentration and the blood glycemia are analyzed at other moments, as in the stages of the maximal test.

5. Conclusions

In sum, the maximal incremental test is capable of increasing significantly the blood lactate concentration and the glycemia, due to hormonal mediators, such as catecholamines, insulin, and glucagon. Furthermore, the low offer of oxygen during vigorous exercises such as the final stages of maximal tests seems to influence and promote intense production of lactate without any possibility of its proper removal.

REFERENCES

[1] Gladden, L. B., 2004, Lactate metabolism: a new paradigm for the third millennium. The Journal of Physiology, 558(1), 5-30.

[2] Phypers, B., and Pierce, J. M. T. Lactate physiology in health and disease. Continuing Education in Anesthesia, Critical Care & Pain, 6(3), 128-132.

[3] Westerblad, H., and Allen, D. G., Lannergren, J., 2002, Muscle fatigue: lactic acid or inorganic phosphate the major cause? News in Physiological sciences: an international journal of physiology produced jointly by the International Union of Physiological Sciences and the American Physiological Society. 17, 17-21.

[4] Macedo, D. V., Lazarim, F. L., Catanho da Silva, F. O., Tessuti, L. S., and Hohl, R., 2009, Is lactate production related to muscular fatigue? A pedagogical proposition using empiricl facts. Advances in Physiology Education, 33(4), 302-307.

[5] Boning, D., Klarholz, C., Himmelsbach, B., Hutler, M., and Maassen, N., 2007, Causes of differences in exercise-induced changes of base excess and blood lactate. European Journal of Applied Physiology, 99(2), 163-171.

[6] Van Hall, G., 2010, Lactate kinetics in human tissues at rest and during exercise. Acta Physiologica (Oxford, England), 199(4), 499-508.

[7] Robergs, R. A., Ghiasvand, F., and Parker, D., 2004, Biochemistry of exercise-induced metabolic acidosis.

American Journal of Physiology Regulatory, Integrative and Comparative Physiology, 287(3), R502-R516.

[8] Ishii, H., and Nishida, Y., 2013, Effect of lactate accumulation during exercise-induced muscle fatigue on sensoriomotor cortex. Journal of Physical Therapy Science, 25(12), 1637-1642.

[9] Beneke, R., 2012, Training at lactate threshold: science based concept or irrational myth? Journal of Sports Medicine & Doping Studies, 2(3), 1-2.

[10] Goodwin, M. L., Harris, J. E., Hernández, A., and Gladden, L. B., 2007, Blood lactate measurements and analysis during exercise: a guide for clinicians. Journal os Diabetes Science and Technology, 1(4), 558-569.

[11] Woorons, X., Bourdillon, N., Vandewalle, H., Lamberto, C., Mollard, P., Richalet, J. P., and Pichon, A., 2010, Exercise with hypoventilation induces lower muscle oxygenation and higher blood lactate concentration: role of hypoxia and hypercapnia. European Journal of Applied Physiology, 110(2), 367-377.

[12] Thomas, C., Sirvent, P., Perrey, S., Raynaud, E., and Mercier, J., 2004, Relationships between maximal muscle oxidative capacity and blood lactate removal after supramaximal exercise and fatigue indexes in humans. Journal of Applied Physiology, 97(6), 2132-2138.

[13] Gharbi, A., Chamari, K., Kallel, A., Ahmaidi, S., Tabka, Z., and Abdelkarim, Z., 2008, Lactate kinetics after intermittent and continuous exercise training. Journal of Sports Science & Medicine, 7(2), 279-285.

[14] Adams, O. P., 2013, The impact of brief high-intensity exercise on blood glucose levels. Diabetes, metabolic syndrome and obesity: targets and therapy, 6, 113-122.

[15] Thompson, P. D., Crouse, S. F., Goodpaster, B., Kelley, D., Moyna, N., and Pescatello, L., 2001, The acute versus the cronic response to exercise. Medicine and Science in Sports and Exercise, 33(6), S438-S445.

[16] American College of Sports Medicine, ACSM`s Guidelines for Exercise Testing and Prescription. 9th ed. Senio editor: L. S. Pescatello. Lippincott Williams & Wilkins, 2013.

[17] Machado, F. A., Kravchychyn, A. C., Peserico, C. S., da Silva, D. F., and Mezzaroba, P. V., 2013, Effect of stage duration on maximal heart rate and post-exercise blood lactate concentration during incrementl teradmill tests. Journal of Science and Medicine in Sports, 16(3), 276-280.

[18] Perez, E. H., Dawood, H., Chetty, U., Esterhuizen, T. M., and Bizaare, M., 2008, Validation of the Accutrend lactate meter for hyperlactatemia screening during antiretroviral therapy in a resource-poor setting. International Journal of Infectious Disease, 16(3), 276-280.

[19] Beneke, R., Hutler, M., Jung, M., and Leithauser, R. M., 2005, Modeling the blood lactate kinetics at maximal short-term exercise conditions in children, adolescents, and adults. Journal of Applied Physiology, 99(2), 499-504.

[20] Older, P., 2013, Anaerobic threshold, is it a magic number to determine fitness for surgery? Perioperative Medicine, 2(2), 1-13.

[21] Davis, J. A., Rozennek, R., Decicco, D. M., Crizzi, M. T., and Pham, P. H., 2007, Effect of plasma volume loss during graded exercise testing on blood lactate concentration. The Journal of Physiological Science, 57(2), 95-99.

[22] Brooks, G. A., Wolfel, E. E., Butterfield, G. E., Cymerman, A., Roberts, A. C., Mazzeo, R. S., and Reeves, J. T., 1998, Poor relationship between arterial [lactate] and leg net release during exercise at 4,300m altitude. The American Journal of Physiology, 275(4), R1192-R1201.

[23] Lehmann, M., and Schmid, P., 1985, Plasma catecholamine and blood lactate cumulation during encremental exhaustive exercise. International Journal of Sports Medicine, 6(2), 78-81.

[24] Mazzeo, R. S., and Marshall, P., 1989, Influence of plasma catecholamines on the lactate threshold during graded exercise. Journal of Applied Physiology, 67(4), 1319-1322.

[25] Stainsby, W. N., Sumners, C., and Andrew., G. M., 1984, Plasma catecholamines and their effect on blood lactate and muscle lactate output. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 57(2), 321-325.

[26] Chudalla, R., Baerwalde, S., Schneider, G., and Maassen, N., 2006, Local and systemic effects on blood lactate concentration during exercise with small and large muscle groups. European Journal of Physiology, 452(6), 690-697.

[27] Edvardsen, E., Scient, C., Hansen, B. H., Holme, I. M., Dyrstad, S. M., and Anderssen, S. A., 2013, Reference values for cardiorespiratory response and fitness on the treadmill in a 20- to 83-years-old population. Chest, 144(1), 241-248.

[28] Tolfrey, K., and Armstrong, N., 1995, Child-adult differences in whole blood lactate responses to incremental treadmill exercise. British Journal of Sports Medicine, 29(3), 196-199.

[29] Colberg, S. R., Hernandez, M. J., and Shahzad, F., 2013, Blood glucose responses to type, intensity, duration and timing of exercise. Diabetes Care, 36(10), e177.

[30] Awobajo, F. O., Olawale, O. A., and Bassey, S., 2013, Changes in blood glucose, lipid profile and antioxidant activities in trained and untrained adult male subjects during programmed exercise on the treadmill. Nigerian Quarterly Journal os Hospital Medicine, 23(2), 117-124.

[31] Goodwin, M. L., 2010, Blood glucose regulation during prolonged, submaximal, continuous exercise: A guide for clinicians. Journal of Diabetes Science and Technology, 4(3), 694-705.

[32] Simões, H. G., Campbell, C. S., Kushnick, M. R., Nakamura, A., Katsanos, C. S., Baldissera, V., and Moffatt, R. J., 2003, Blood glucose threshold and metabolic response to incremental exercise tests with and without prior lactic acidosis induction. European Journal of Applied Physiology, 89(6), 603-611.

[33] Gavin, J. P., Myers, S. D., and Willems, M. E., 2015, The effect ok glycogen reduction on cardiorespiratory and metabolic responses during downhill running. European Journal of Applied Physiology, 1.

[34] Fanelli, C. G., Porcellati, F., Rossetti, P., and Bolli, G. B., 2006, Glucagon: the effects of its excess and deficiency on insuline action. Nutrition, Metabolism and Cardiovascular Disease, 16, S28-S34.

[35] Withe, J. A., Ismail, A. H., and Bradley, C. A., 1978, Serum

insulin and glucose response to graded exercise in adults: Part II, the effect of exercise conditioning. British Journal of Sports Medicine, 12(3),137-141.

[36] Howlett, K. F., Watt, M. J., Hargreaves, M., and Febbraio, A, 2003, Regulation of glucose kinetics during intense exercise in humans: effects of alpha- and beta-adrenergic blockade. Metabolism: clinical and experimental, 52(12), 1615-1620.

[37] Roberts, S., Desbrow, B., Grant, G., Anoopkumar-Dukie, S., Leveritt, M., 2013, Glycemic response to carbohydrate and the effects of exercise and protein. Nutrition, 29(6), 881-885.

[38] Wolever, T. M., 2003, Carbohydrate and regulation of blood glucose and metabolism. Nutrition Reviwes, 61(5 Pt 2), S40-S48.

[39] Crapo, P. A., Reaven, G., and Olefsky, J., 1977, Postprandial plasma-glucose and –insulin responses to different complex carbohydrates. Diabetes, 26(12), 1178-1183.

[40] Jenkins, D. J., Wolever, T. M., Taylor, R. H., Barker, H., Fielden, H., Baldwin, J. M., Newman, H. C., Jenkins, A. L., and Goff, D. V., 1981, Glycemic index of foods: a physiological basis for carbohydrate exchange. The American Journal of Clinical Nutrition, 34(3), 362-366.

[41] Gannon, M. C., and Nuttall, F. Q., 2004, Effect of a high-protein, low-carbohydrate diet on blood glucose control in people with type 2 diabetes. Diabetes, 53(9), 2375-2382.

[42] Brand-Miller, J. C., Thomas, M., Swan, V., Ahmad, Z. I., Petocz, P., and Colagiuri, S., 2003, Physiological validation of the concept of glycemic load in lean young adults. The Journal of Nutrition, 133(9), 2728-2732.

[43] Tsintzas, K., Simpson, E. J., Seevaratnam, N., and Jones, S., 2003, Effect of exercise mode on blood glucose disposal during physilogical hyperinsulinaemia in humans. European Journal of Applied Physiology, 89(2), 217-220.

[44] Trumbo, P., Schlicker, S., Yates, A. A., Poos, M., Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. Journal of the American Dietetic Association, 102(11), 1621-1630.