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Effect of aerobic exercise on glycemic control

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Abstract. In the case of pathologies with a slow onset, physical effort can correct certain molecular signaling pathways, but the primary factors that are often genetic, environmental or nutritional remain decisive. Blood glucose fluctuation is a primary indicator of a slow-onset pathology. The integration of the adaptive physiological response to physical effort in terms of carbohydrate metabolism is related to the functional humoral connection between skeletal muscle, adipose tissue, pancreas and liver. Physical effort is associated with a series of physiological changes, the intensity of which is influenced by the duration and intensity of physical effort. Physical effort, through the synthesis of specific molecular signals activates in the skeletal muscle the insulin-dependent and independent mechanisms that facilitate glucose absorption. In the case of our experimental model, the statistically significant increase in blood glucose levels during the training period is based on the activation of the central neuroendocrine axis with the release of hormones that remove glucose from stores or increase the degree of glucose conversion from glucoforming (glucose forming) molecules.
Key words: *exercise, aerobic, glycemic control.*

Introduction

Numerous experimental clinical and non-clinical studies have shown that physical effort underlies biological health and well-being regardless of the subject's age and is associated with a reduced risk of mortality. It has also been shown that physical effort, through the activated molecular and biochemical mechanisms, has a preventive effect on the risk of cancer, in other words the incidence of cancer decreases statistically significantly in physically active people (1). In the case of pathologies with a slow onset, physical effort can correct certain molecular signaling pathways, but the primary factors that are often genetic, environmental or nutritional remain decisive. Blood sugar fluctuation is a primary indicator of a slow-onset pathology. Physical effort is associated with a series of physiological changes, the intensity of which is influenced by the duration and intensity of physical effort. Thus, during physical effort, cardiac output increases to meet oxygen needs at the peripheral level, which induces a change in blood flow (2). Physical effort causes an increase in the metabolic rate which has a direct effect on glucose consumption and production, as does the level of lactate due to anaerobic metabolism in the muscle. Catecholamines are over-synthesized during exercise, and they are responsible for altering blood glucose levels (3). The integration of the adaptive physiological response to physical effort in terms of carbohydrate metabolism is related to the functional humoral connection between skeletal muscle, adipose tissue, pancreas and liver. In 1926 appears the first clarification related to the effect induced by physical effort on the synthesis and mode of action of insulin, being unknown the primary molecular mechanisms underlying the correlation physical effort - insulin - glycemic index (4). More and more experimental studies show that short-term physical effort modulates insulin-dependent and insulin-independent mechanisms, while long-term effects involve activating humoral mechanisms of communication between organs involved in the control and synthesis of protein and lipid carbohydrates (2,5). Physical effort, through the synthesis of specific molecular signals, activates in the skeletal muscle the insulin-dependent and independent mechanisms that facilitate glucose absorption and increase the speed of response of muscle receptors to insulin (6,7). Post-effort hypoglycemia is a controversial phenomenon whose mechanism is partially explained. The mechanism is based on the temporary increase in glucose uptake by skeletal muscle up to 5 times through increased insulin-independent but AMPK-dependent transport (8). Physical effort stimulates AMP-activated protein kinase activation, facilitates glucose uptake by insulin-independent mechanisms in striated muscle fibers, and muscle fiber responds adaptively by activating insulin-dependent glucose uptake mechanisms, mitochondrial biogenesis, and redox adaptability (9,2,10,13). The presented mechanism aims to increase the biosynthesis of proteins involved in the molecular signaling of the phenomenon of carbohydrate metabolic adaptability to physical effort. The liver is the most important organ in the storage of glucose in the form of glycogen or triglycerides

considered energy stores. The liver is the only structure that can export glucose during periods when the body needs energy, such as physical effort (14). During sustained exercise for at least 20 minutes, insulin secretion decreases and glucagon, catecholamines and cortisol secretion increase (15). The liver is the main field of action of pancreatic hormones during the transition from pre- to post-mental states (2). As shown above, physical effort stimulates the activation of AMP-activated protein kinase in skeletal muscle, but also in adipose tissue and liver, which facilitates glucose absorption in the hepatocyte. The liver is considered the organ involved in post-effort recovery, although more and more experimental studies place it on a leading place in the team of structures directly involved in supporting physical effort. Exercise-induced cellular redox changes can stimulate certain insulin-independent glucose transport pathways in the liver cell. The pancreas through hormonal syntheses contributes decisively to glucose management during exercise and post-exercise. Plasma glucose, insulin and glucagon are generally primary indicators of beta-pancreatic cell function (16). The pancreas is extremely sensitive to changes induced by physical effort on blood glucose levels. During physical effort, the synthesis of alpha-pancreatic cells increases, glucagon and insulin synthesis fall, without being able to explain this hormonal dynamics of the pancreas. Initially, the pancreatic hormonal dynamics during physical exertion were attributed to medullary-adrenal stimuli or to neural mechanisms, without elucidating possible mechanisms (17). The pancreas is extremely sensitive to changes induced by physical exertion on blood glucose levels. During physical exertion, the synthesis of alpha-pancreatic cells increases, glucagon and insulin synthesis fall, without being able to explain this hormonal dynamics of the pancreas. Initially, the pancreatic hormonal dynamics during physical effort were attributed to medullary-adrenal stimulus or to neural mechanisms, without elucidating possible mechanisms (17). An interesting aspect is that when the pancreas is denervated there are no changes in the dynamics of insulin or glucagon synthesis during exercise, the mechanism suggests the involvement of an endocrine or paracrine factor in the specific dynamic regulation of pancreatic hormones during exercise (7). During exercise certain organs such as skeletal striated muscle and liver release interleukin 6 (IL-6) which stimulates the release of pancreatic glucagon (16).

Material and method

The study group consisted of 7 student (female subjects) aged 21-25 years, with normal body weight. The study group are not amateur or performance sportsmen, and have not previously followed a physical effort program. The subjects expressed their agreement by signature to participate in the experimental study without remuneration or other type of conditioning. Subjects performed aerobic physical effort, such like running on the treadmill or pedaling, at medium intensity (65-70%VO2max), for 50 minutes/day, 5 days/week. The determination of the capillary blood glucose level (a jéun) was performed daily, during 5 days before training period (Preeffort), and 5 days during the post training period (Posteffort), with a portable device. The training period was of 5 consecutive days. We chose a portable device (glucometer) to increase the applicability of the study. The determination of the glycemia (a jéun) was performed every day, from the 5th day of training, at 24 hours after the physical training.

Statistical Analysis. Data are presented as means and standard deviations (±SD). Comparisons between matches for each variable were performed with one-way repeated-measures ANOVA. The level of significance for all statistical comparisons was set at $p < 0.05$ using GraphPad® (Prism 6.0, San Diego, CA, USA) software.

Results

The data interpretation was done only on study group, and not individually. The raw data collected in two experimental moments were statistically processed and interpreted according to the modification of the statistical significance threshold. Table I. and Table II. Blood glucose levels increased statistically significantly during the training cycle (Posteffort) (Fig.1).

Table I. Blood glucose level expressed in mmol/L before physical exertion and during the training cycle

Glycemia (mmol/L)	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
Preefort	35	4.9429	.22659	.03830	4.8650	5.0207	4.30	5.30
Posteffort	35	5.2400	.22387	.03784	5.1631	5.3169	4.60	5.50
Total	70	5.0914	.26905	.03216	5.0273	5.1556	4.30	5.50

Table II. Mean blood glucose levels measured before physical exertion and during the training cycle

Glycemia (mmol/L)	Sum of Squares	df	Mean Square	F	Sig.
Preefort	1.545	1	1.545	30.458	.000
Posteffort	3.450	68	.051		
Total	4.995	69			

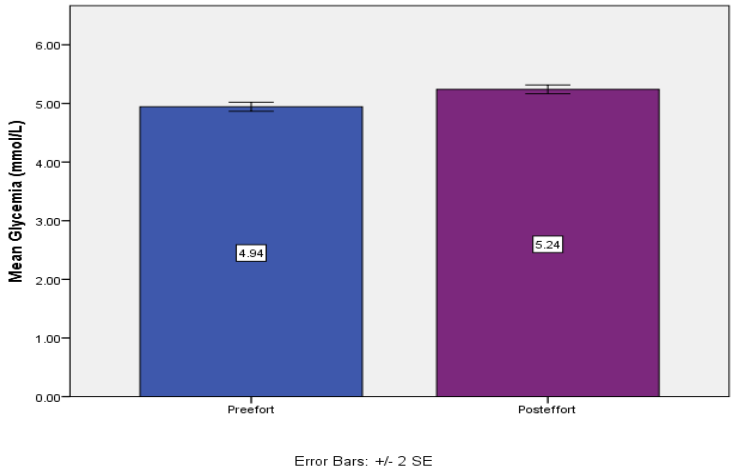

Figure 1. Blood glucose level (mean ± SE) before physical exertion and during the training cycle

Discussion

The combination of aerobic and resistance training, as recommended by current ADA guidelines, may be the most effective exercise modality to control glucose and lipids in type 2 diabetes (7). Physical effort is considered by practitioners the basis of the concept of healthy lifestyle, to which is added the nutrition factor and integrative concept called leisure nontoxicity. Numerous experimental studies suggest that regular aerobic exercise reduces the risk of cardiovascular disease, prevents acute and chronic stress, inflammation of soft connective tissue and even the tumor process aspect mentioned in the introduction to the article (18, 19). A noticeable and rarely mentioned aspect in the literature is that there are clear biochemical and physiological differences between the two sexes. Thus, hormonal baggage can influence the molecular basis of adaptability mechanisms or also the different response to the type, duration and intensity of physical exertion (20, 21).

The mechanisms by which exercise can adjust blood glucose levels are relatively well known, but all hypotheses have as a starting point the hormonal basis induced by physiological requirements during exercise. The new metabolic requirements induced by the physical effort of the skeletal muscle cannot be satisfied without an adequate metabolic response from the liver and pancreas. If the liver and pancreas did not respond specifically metabolically and hormonally, the striated muscle could not sustain physical effort, and adaptability to effort would be impossible. Through the two specific pathways, glucose export and import of glucoforming compounds, the liver actively participates in the metabolic adaptability to physical effort.

In the case of our experimental model, the statistically significant increase in blood glucose levels during the training period is based on the activation of the central neuroendocrine axis with the release of hormones that remove glucose from stores or increase the degree of glucose conversion from glucoforming molecules. Dietary control during training could explain the possible cause of high blood glucose in the experimental model presented. From the experimental data obtained it appears that the physical effort performed for 5 days induces a statistically significant increase in blood glucose levels. The experimental study requires a larger number of subjects as well as establishing the carbohydrate profile and the correlation with certain hormones of interest. The study is a starting point for more practical research on certain high-risk or potential-risk categories, such as patients with type 1 or type 2 diabetes.

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