Journal of Biodiversity and Environmental Sciences (JBES)
ISSN: 2220-6663 (Print) 2222-3045 (Online)
Vol. 4, No. 6, p. 422-427, 2014
http://www.innspub.net

RESEARCH PAPER OPEN ACCESS

Acute response of glucose homeostasis to one short exercise in asthma patients

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Article published on April 30, 2014

Key words: Short time exercise, Asthma, Insulin

Abstract

There is evidence that asthma is associated with insulin resistance or high glucose level, although the molecular mechanisms for this are less understood. In this study, we evaluate glucose or insulin resistance in response to one exercise in asthma patients. To archive this purpose, fourteen males (age 39 ± 8.8 year, height 175 ± 1.9 cm, weight 96 ± 11 kg) with mild to moderate asthma were completed a short time exercise included leg cycling. Blood samples were collected for measure glucose and insulin at before and at the end of exercise. The insulin resistance index was assessed by homoeostasis model assessment (HOMA-IR). Statistical analysis used by Independent paired T-test. Glucose level ($p = 0.000$) and insulin resistance decreased significantly ($p = 0.005$) by exercise test in studied patients. There was no significant change in serum insulin by exercise test ($p = 0.093$). Based on these data, we can say one short time exercise can be improving glucose homeostasis in asthma patients.

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Introduction

Asthma is a disease of airways of allergic origin which is manifested physiologically with narrowing of respiratory airways and clinically with sudden attacks of dyspnea, coughing, and wheezing (Armstrong *et al*., 2000). This disease exacerbates through increased recall and activation of inflammatory cells such as eosinophils and T cells in the mucosa of the respiratory airways. However, numerous clinical studies have frequently mentioned increased blood glucose levels or insulin resistance in obese and diabetic patients (Haber *et al*., 2006), they also pointed out that in diabetes, obesity, and obesity-related diseases, insulin level increases in response to its secretion from beta cells of the pancreas and the phenomenon of insulin resistance (Butler *et al*., 2003).

Some studies on asthmatic patients have also supported increased insulin resistance and impaired insulin secretion and glucose metabolism in these patients (Ma *et al*., 2010). These studies have also reported the increased levels of fasting glucose in asthmatic patients compared to healthy people (Hilda Segura *et al*., 2007). The main factors responsible for the increase in blood glucose levels in these patients are not yet fully known. The role of insulin resistance in this phenomenon should not be forgotten and similar to other diseases related to insulin resistance; it seems that insulin dysfunction is of special importance in higher levels of blood sugar in these patients.

However, several studies mentioned the role of peptide mediators or other hormonal factors in impaired glucose homeostasis in these patients (Popovic *et al*., 2005; Fleisch, 2007). On the other hand, the role of exercise as a medicinal treatment in improvement of blood glucose levels in obese populations or patients has been observed repeatedly by researchers (Nayak *et al*., 2010; Tang *et al*., 2005). Scientific resources have also supported the improvement of glucose or insulin resistance in asthmatic patients after long-term exercise programs (Khorshidi *et al*., 2014), but the effect of one-session exercise on these variables was less studied, especially in asthmatic patients. Therefore, the main objective of the present study was to investigate the effect of a relatively moderate intensity one-session exercise on glucose levels and insulin resistance in patients with asthma.

Patients and methods

Subjects were 14 males with mild to moderate intensity of asthma disorder. Asthma diagnosis and its severity were determined by FEV1/FVC. Written consent was obtained from each subject after the experimental procedures and possible risks and benefits were clearly explained.

Inclusion or Exclusion criteria

Obesity was first inclusion criteria. Participants were non-athletes and non-alcoholics. Persons with a known diagnosis of diabetes (defined as a physician's diagnosis or the regular use of diabetic medications) were excluded. The exclusion criteria were as follows: Patients with known history of acute or chronic respiratory infections which may interfere with lung function tests, neuromuscular disease, hypertension, cardiopulmonary disease and those who had undergone chest surgery or other major operations.

Measurement of related obesity factors

Anthropometric measurements of height, weight, percent body fat, and circumference measurements were taken. Weight was measured by an electronic balance and height by a stadiometer. Height of the barefoot subjects was measured to the nearest 0.1 cm. Hip circumference was measured at the level of the greater trochanter, all parameters being measured by well-trained dietitians. Waist to hip ratio (WHR) was calculated as waist circumference divided by hip circumference, anthropometric indices being measured by the same investigator. BMI was calculated as weight (kg)/height (m2).

Laboratory parameters

Blood samples were collected for measure insulin and glucose before and immediately after exercise test. All participants refrained from any severe physical activity 48 h before measurements. Exercise test lasted 15 min on for 5 stages without rest between stages including cycling or stationary cycle according to YMCA protocol. Plasma glucose was measured with the glucose oxidase method. Blood samples were centrifuged for 10 minutes by 3000 rpm speed for serum separation to analysis serum insulin. Insulin was determined by ELISA method (Demeditec, Germany) and the intra- assay and inter-assay coefficient of variation of the method were 2.6% and 2.88 respectively. The homoeostasis model assessment (HOMA) for estimating insulin resistance was calculated as serum glucose (mmol/L)×serum insulin (mU/L)/22·5 (Matthews *et al*., 1985)

Statistical Methods

Data were analyzed by computer using SPSS software version 15.0. Data were expressed as individual values or the mean ± SD for groups. Kolmogorov-Smirnov test was used to determine of normal status of the data. Student's paired 't' test was applied to compare the pre and post exercise values. A p-value of less than 0.05 was considered to be statistically significant.

Results

Body weight and blood chemistry parameters at baseline are shown in Table 1. Other anthropometrical of subjects were: abdominal circumference; 106 ± 10 cm, hip circumference: 106 ± 7 cm, AHO ratio: 0.99 ± 0.05. Exercise test

Exercise test resulted in improvements in some clinical markers such as significant decrease in glucose concentration (from 120 ± 41 to 110 ± 40, p = 0.000, Fig 1) and significant decrease in insulin resistance (from 2.79 ± 1.39 to 2.29 ± 1, p = 0.005, Fig 2), but no change in serum insulin (from 9.21 ± 2.46 to 8.58 ± 2.26, p = 0.093).

Table 1. Anthropometric and physiological characteristics of subjects (n = 14)

Variable	Age (years)	Height (cm)	Weight (kg)	Systolic BP (mmHG)	Diastolic BP (mmHG)	Body fat (%)	BMI (kg/m2)
Mean	38.5 ± 8.5	175 ± 1.9	96 ± 10.8	128 ± 8	88 ± 7	31.2 ± 2.9	31.3 ± 3
SD							

Abbreviations: BMI, body mass index; SD, standard deviation.

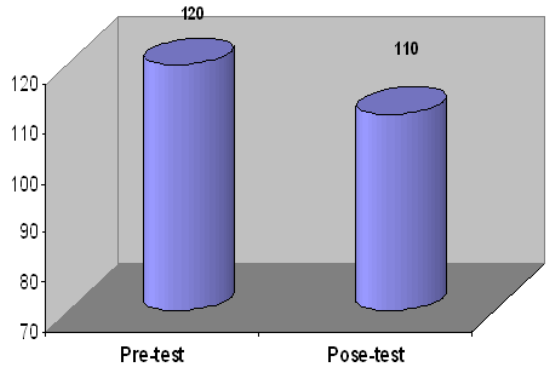

Fig 1. Acute response of glucose to exercise test. Exercise test result in significant decrease in glucose concentration

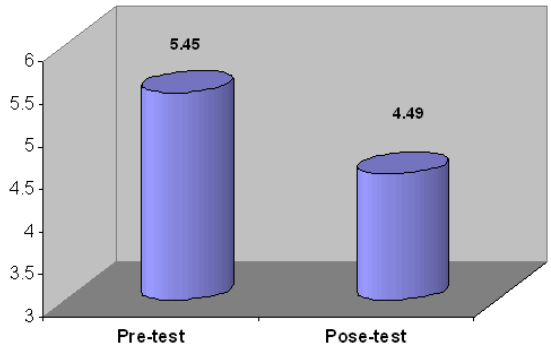

Fig 2. Acute response of insulin resistance to exercise test. Exercise test result in significant decrease in insulin resistance

Discussion

As mentioned earlier, the present study aimed to determine the immediate response of some key indicators of type 2 diabetes to a one-session moderate cycling in patients with chronic asthma. Although the findings of this study showed no change in serum levels in response to exercise in these patients, the concentration of glucose as well as resistance to insulin were significantly decreased. In fact, blood glucose levels or insulin resistance reduction following long-term exercise, which per se is associated with a significant reduction in body weight, has been previously reported by several studies (Tang *et al.*, 2005; Sheu *et al.*, 2008). However, the acute response of glucose or insulin resistance to a relatively short period of one-session exercise was less studied, especially in asthmatic patients. Scholarly resources have supported higher levels of blood glucose and insulin resistance in patients with asthma compared with healthy people (Ma *et al.*, 2010).

Several metabolic and hormonal factors are effective on association of obesity and asthma. Understanding of interactions between these factors and the pattern of their changes in response to exercise programs along with weight loss underlies many modern studies being performed to provide appropriate solutions for improvement of the devastating symptoms of this disease. In addition to medicinal therapy, exercise leads to beneficial effects of physiological parameters influencing insulin resistance in patients with asthma (Alioglu *et al.*, 2007). Apart from insulin resistance, it is possible that glucose levels be affected by other factors such as levels of inflammatory or anti-inflammatory cytokines.

Some previous studies have reported simultaneous reduction in blood glucose levels and improvement in inflammatory or anti-inflammatory cytokines profile in other patient or obese populations (Liu *et al.*, 2009; Karadag *et al.*, 2008). Indeed, lack of measurement of inflammatory cytokines in response to exercise test, which roles in blood glucose level were frequently reported, is a limitation of this study. For example, scientific studies have repeatedly pointed out the potential role of adiponectin as an anti-inflammatory cytokine (Tajima *et al.*, 2005; Reinehr *et al.*, 2005) and certain interleukins, including IL-6 and TNF-α, as inflammatory cytokines, in insulin resistance or glucose levels (Nayak *et al.*, 2010; Samaras *et al.*, 2010). Some other studies have supported the impairment of these cytokines in asthmatic patients, such as serum levels of adiponectin (Nagel *et al.*, 2008) or increased levels of leptin and other inflammatory cytokines (Deetz *et al.*, 1997; Popovic *et al.*, 2005). Given their role in blood glucose levels, it seems that their changes in response to exercise are associated with changes in blood glucose levels or insulin resistance. In support of the aforementioned issues, the findings of some previous studies with higher samples number have showed the close relationship of glucose or insulin resistance with inflammatory or anti-inflammatory cytokines in asthmatic patients (Fleisch, 2007).

Significant reduction in glucose and insulin resistance in patients with asthma in this study indicates that even a relatively short-term, moderate-intensity exercise led to a significant improvement in blood glucose levels in these patients; although it seems that the response of blood glucose to exercise test is short and transient, rather than a of long-term adaptation.

References

Alioglu B, Ertugrul T, Unal M. 2007. Cardiopulmonary responses of asthmatic children to exercise: analysis of systolic and diastolic cardiac function. Pediatric Pulmonology **42(3),** 283-9. http://dx.doi.org/10.1002/ppul.20575

Armstrong N, Van Mechelen W. 2000. Pediatric exercise science and medicine. London: Oxford University Press **3,** 323-328.

Butler AE, Janson J, Bonner-Weir S, Ritzel R, Rizza RA, Butler PC. 2003. Beta-cell deficit and

increased beta-cell apoptosis in humans with type 2 diabetes. Diabetes **52,** 102–110.
http://dx.doi.org/10.2337/diabetes.52.1.102

Deetz DC, Jagielo PJ, Quinn TJ, Thorne PS, Bleuer SA, Schwartz DA. 1997. The kinetics of grain dust–induced inflammation of the lower respiratory tract. American Journal of Respiratory and Critical Care Medicine **155,** 254-259. http://dx.doi.org/10.1164/ajrccm.155.1.9001321

Fleisch AF. 2007. Influence of serum leptin on weight and body fat growth in children at high risk for adult obesity. Journal of Clinical Endocrinology & Metabolism **92(3),** 948-54.
http://dx.doi.org/10.1210/jc.2006-1390

Haber EP, Procópio J, Carvalho CR, Carpinelli AR, Newsholme P, Curi R. 2006. New insights into fatty acid modulation of pancreatic beta-cell function. International Review of Cytology **248,** 1-41.
http://dx.doi.org/10.1016/S0074-7696(06)48001-3

Hilda Segura N, Hernández L, Velázquez C, Rodríguez J, Murillo E. 2007. Asthma and obesity: related inflammatory diseases. Revista Alergia México **54(1),** 24-8.

Karadag F, Karul AB, Cildag O, Yilmaz M, Ozcan H. 2008. Biomarkers of systemic inflammation in stable and exacerbation phases of COPD. Lung **186(6),** 403-9.
http://dx.doi.org/10.1007/s00408-008-9106-6

Khorshidi D, Ghanbari S, Samarikhalaj H, Kiani F. 2014. Aerobic exercise program affect insulin resistance in asthma patients. International Journal of Biosciences **4(2),** 322-326.

Liu X, Ji Y, Chen J, Li S, Luo F. 2009. Circulating visfatin in chronic obstructive pulmonary disease. Nutrition **25(4),** 373-8.
http://dx.doi.org/10.1016/j.nut.2008.09.008

Ma J, Xiao L, Knowles SB. 2010. Obesity, insulin resistance and the prevalence of atopy and asthma in US adults.Allergy **65(11),** 1455-63.
http://dx.doi.org/10.1111/j.1398-9995.2010.02402.x

Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. 1985. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia **28,** 412-419.
http://dx.doi.org/10.1007/BF00280883

Nagel G, Koenig W, Rapp K, Wabitsch M, Zoellner I, Weiland SK. 2008. Associations of adipokines with asthma, rhinoconjunctivitis, and eczema in German schoolchildren. Pediatric Allergy and Immunology [Epub ahead of print].

Nayak BS, Ramsingh D, Gooding S, Legall G, Bissram S, Mohammed A. 2010. Plasma adiponectin levels are related to obesity, inflammation, blood lipids and insulin in type 2 diabetic and non-diabetic Trinidadians. Primary Care Diabetes [Epub ahead of print]
http://dx.doi.org/10.1016/j.pcd.2010.05.006

Popovic V, Duntas LH. 2005. Brain somatic cross-talk: ghrelin, leptin and ultimate challengers of obesity. Nutr Neurosci **8(1),** 1-5.
http://dx.doi.org/10.1080/10284150400027107

Reinehr T, Roth CL, Alexy U, Kersting M, Kiess W, Andler W. 2005. Ghrelin levels before and after reduction of overweight due to a low-fat high-carbohydrate diet in obese children and adolescents. International Journal of Obesity (London) **29(4),** 362-8.
http://dx.doi.org/10.1038/sj.ijo.0802913

Samaras K, Botelho NK, Chisholm DJ, Lord RV. 2010. Subcutaneous and visceral adipose tissue gene expression of serum adipokines that predict type

2 diabetes. Obesity (Silver Spring) **18(5),** 884-9. http://dx.doi.org/10.1038/oby.2009.443

Sheu WH, Chang TM, Lee WJ, Ou HC, Wu CM, Tseng LN. 2008. Effect of weight loss on proinflammatory state of mononuclear cells in obese women. Obesity (Silver Spring) **16(5),** 1033-8. http://dx.doi.org/10.1038/oby.2008.37

Tajima D, Masaki T, Hidaka S, Kakuma T, Sakata T, Yoshimatsu H. 2005. Acute central infusion of leptin modulates fatty acid mobilization by affecting lipolysis and mRNA expression for uncoupling proteins. Experimental Biology and Medicine **230(3),** 200-6.

Tang Z, Yuan L, Gu C, Liu Y, Zhu L. 2005. Effect of exercise on the expression of adiponectin mRNA and GLUT4 mRNA in type 2 diabetic rats. Journal of Huazhong University of Science and Technology (Medical Sciences) **25(2),** 191-3, 201. http://dx.doi.org/10.1007/BF02873574