INNSPUB

International Journal of Biosciences | IJB |
ISSN: 2220-6655 (Print) 2222-5234 (Online)
http://www.innspub.net
Vol. 4, No. 8, p. 256-261, 2014

RESEARCH PAPER OPEN ACCESS

Serum interleukin 1 beta in sedentary healthy obese and non-obese men

Seyyed Rafi Shafabakhsh Kolor, Mona Hafezi, Reza Naseri Rad

Department of Physical Education and Sport Sciences, Islamshahr Branch, Islamic Azad University, Islamshahr, Iran

Key words: Obesity, systemic inflammation, chronic diseases.

http://dx.doi.org/10.12692/ijb/4.11.256-261 Article published on April 22, 2014

Abstract

Obesity is associated with low-grade systemic inflammation and plays a key role in the pathogenic mechanism of chronic diseases. This study was aimed to compare serum IL-1ß between obese and non-obese men. For this purpose, fasting blood samples were obtained of fifteen sedentary obese and fifteen non-obese men matched to age and height (34 – 44 years, 170 – 180 cm) in order to measuring serum IL-1ß and compare between two groups. Data of independent T test showed that serum IL-1ß was significant higher in obese when compared to non-obese group (2.42 ± 0.87 obese vs 1.76 ± 0.64 non-obese, pg/ml, p = 0.026). There was no significant correlation between IL-1ß and body mass index in obese subjects (p = 0.75, r = 0.09). Based on these data, we can say obesity has an inflammation property in even in healthy subjects and increased serum IL-1ß may be due to disturbance of other cytokines in this population.

* **Corresponding Author:** Seyyed Rafi Shafabakhsh Kolor ✉ shafabakhsh_s@yahoo.com

Introduction

Similar to tumor necrosis factor alpha, interleukins are also involved in creating innate and inflammatory immune responses. Scientific evidences have revealed that the levels of IL-1ß is not only of special importance in inflammatory and respiratory diseases, but disturbance in its systemic levels affects metabolic disorders effective on obesity and body fat levels (Urboniene *et al*., 2008; Guler *et al*., 2004). In addition, the findings indicate a significant relationship between increased levels of IL-1ß and change in BMI (Saltevo *et al*., 2007). But in Cosette's study (1991), no significant correlation was observed between plasma levels of IL-1ß and BMI as a predictor of obesity (Gosset *et al*., 1991). Scientific studies have frequently pointed out that increased serum or plasma level of this inflammatory cytokine is associated with increased insulin resistance (Urboniene *et al*., 2008; Guler *et al*., 2004). It is known that IL-1ß levels rise in obesity and lead to a decreased insulin secretion from beta cells of the pancreas (Osborn *et al*., 2008). Its role in lipid metabolism through regulating insulin levels and lipase activity has also been reported (Matsuki *et al*., 2003). Scientific resources have revealed that increased secretion of IL-1ß results in lung inflammation, destruction of pulmonary alveoli elastic fibers, fibrosis or obstruction of respiratory tract wall, and accumulation of lymphocytes in airways of respiratory patients (Urboniene *et al*., 2008). Some literature has reported its higher levels in smokers than non-smokers (Frohlich *et al*., 2003). Despite these findings, no association was observed between serum levels of IL-1ß and body mass index in another study (Segura *et al*., 2007). According to some studies which stated the lack of correlation between IL-1ß and indicators of obesity, the present study is carried out aiming to compare this inflammatory cytokine between obese and normal weight men.

Method and Subjects

Study population, inclusion or exclusion criteria

Subjects included fifteen sedentary healthy obese and same numbers non-obese men matched to age and sex (aged 36.8 ± 2.99 years) participated in this study by accessible samples. After the nature of the study was explained in detail, informed consent was obtained from all participants. The purpose of this study was to compare serum IL-1ß between obese and none-obese adult men. A medical history to retrieve information about health status, current medications, alcohol consumption; a physical examination including height, weight, waist circumference were performed of all participants. Participants were non-athletes and non-alcoholics. The exclusion criteria were history of acute or chronic respiratory infections, neuromuscular disease, cardiopulmonary disease and type II diabetes and other chronic diseases.

Anthropometric measurements

Anthropometric measurements of height, weight, percent body fat, and circumference measurements of all subjects were taken at first. Weight and height were measured in the morning, in fasting condition, standing, wearing light clothing and no shoes. Body mass index was measured for each individual by division of body weight (kg) by height (m2). Waist-to-hip ratio was calculated as abdominal circumference divided by hip circumference as measured to the nearest 0.5 cm with a standard measuring tape. All of these measurements were conducted by the same researcher.

Biochemical Analysis

Participants were instructed not to heavy physical activity for at least 48 h before blood collection. Blood was collected between 8:00 a.m. and 9:00 a.m. after 12-h water-only fast with participants in the seated position. After sampling in ETDA- or serum-tubes, blood was immediately chilled on ice and centrifuged in order to separate serum. Serum wwew analyzed for measuring IL-1ß in two groups. Serum IL-1ß was determined by ELISA method Enzyme-linked Immunosorbent Assay for quantitative detection of human IL-1β), using a Biovendor- Laboratorial kit made by Biovendor Company, Austria. The sensitivity of the IL-6 assay was 0.3 Pg/mL.

Statistical analysis

All values are reported as mean and standard deviation. Statistic analysis was done with SPSS 15.0 for Windows. Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. Comparisons between the means of each group were done using the independent t-test. Partial Spearman correlations were calculated to determine the relations of IL-1ß with body mass index and other anthropometrical markers. The results were considered statistically significant for $p<0.05$.

Results

As mentioned, in this study we compared serum resistin in sedentary obese and non-obese subjects. IL-1ß is an inflammatory cytokine and increased in chronic diseases as cardiovascular disease. Anthropometric characteristics of the study participants are described in Table 1. Data of table 1 shows that all anthropometrical markers are significant higher in obese subjects ($p < 0.05$). Based on independent analysis, significant higher serum IL-1ß was found in obese subjects when compared to non-obese group (2.42 ± 0.87 obese vs 1.76 ± 0.64 non-obese, pg/ml, $p = 0.026$, Fig 1). There were no correlations between serum IL-1ß concentrations and body mass index in obese men ($p = 0.75$, $r = 0.09$).

Table 1. Mean and standard deviation of anthropometrical characteristics of studied subjects

Variables	obese group	None-obese group
Age (year)	36.8 ± 2.99	38.5 ± 3.20
Weight (kg)	101.6 ± 12.25	71.7 ± 6.62
Height (cm)	176.2 ± 3.65	173.5 ± .96
Body Fat (%)	32.93 ± 2.55	22.71 ± 1.70
Body mass index (kg/m^2)	32.67 ± 3.09	23.77 ± 1.41
Abdominal circumference (cm)	108 ± 8.9	85.9 ± 4.07

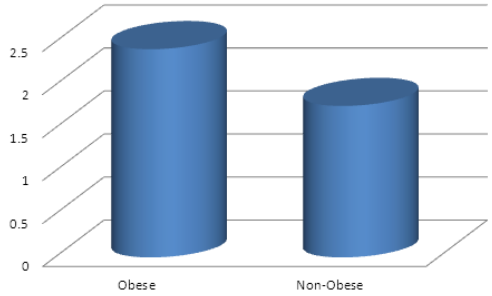

Fig. 1. Serum IL-1ß concentration in obese and normal groups. Data shows higher serum IL-1ß in obese subjects when compared to non-obese group.

Discussion

In addition to genetics and environmental factors such as diet, lifestyle, or levels of daily physical activity, researchers have recently reported the role of metabolic disorders in development of obesity and disorders related to overweight. Peptide hormones secreted by adipose tissue and other endocrine tissues such as gastrointestinal tract have been frequently suggested to be involved in regulation of appetite and energy balance which are considered as the main causes of obesity. Besides a great source of fat, adipose tissue as an endocrine organ secretes some peptide hormones including cytokines or adipokines which have a particular role in energy homeostasis and glucose or fat balance (Kahn *et al.*, 2000; Spiegelman *et al.*, 2001).

Several studies compared the effects of inflammatory cytokine levels in different populations, such as obese and lean, athletic and non-athletic, and healthy individuals and patients. The majority of these studies showed higher levels of inflammatory cytokines in those people who were overweight or had higher body fat percent (Pickup *et al.*, 1997; Grau *et al.*, 1996; Bruun *et al.*, 2003). The present study also supports the increment of IL-1ß levels in obese compared with normal weight men.

Direct and indirect effects of peptide hormones, such as ghrelin, leptin, adiponectin, resistin, visfatin, pre-inflammatory and anti-inflammatory cytokines, and other peptides secreted by adipose tissue and other endocrine tissues, on the prevalence of obesity and its

related disorders have been frequently studied by biochemists and health sciences professionals (Tschop *et al.*, 2001; Yukihiro *et al.*, 2002; Snehalatha *et al.*, 2008). Interleukins 1 beta, including IL-1α and IL-1ß, which act as mediators of inflammation in the body have as much as about thirty percent similar structural features, however, they have common receptors and exert relatively similar biological effects. IL-1ß plays an important role in inflammation (Matsuki *et al.*, 2003).

It is known that production and secretion of IL-1ß is associated not only with inflammatory and immune system diseases but also with metabolic abnormalities (Urboniene *et al.*, 2008; Guler *et al.*, 2004). The main source of this inflammatory cytokine is circulating monocytes, tissue macrophages, and dendritic cells; however, it can also be secreted by B lymphocytes and NK cells (Dinarello *et al.*, 2009). Apart from its physiological functions in protection or preservation of host cells, IL-1ß has also an important role in certain severe inflammatory diseases, such as hereditary diseases or polygenic inflammatory diseases which can often be controlled by anti-IL-1ß medicines (Kontzias *et al.*, 2012; Quartier *et al.*, 2011). It is shown that similar to IL-1ß, the secretion rate of macrophages has a potential association with obesity in mice and humans (Taniguchi *et al.*, 2006; Rui *et al.*, 2002). Its higher levels in obese people have been also reported by some other studies (Taniguchi *et al.*, 2006; Rui *et al.*, 2002). Increased levels of this inflammatory cytokine in respiratory or allergic diseases have been already observed (Urboniene *et al.*, 2008). On the other hand, some studies have introduced obesity as a background for increased respiratory disease (Nystad *et al.*, 2004; Kyle *et al.*, 2009); so that increased and decreased body weight has respectively resulted in decreased and increased severity of asthma as a respiratory disease (Shore *et al.*, 2007). In a general summary, the present study confirms most of the previous findings and stresses on higher levels of IL-1ß as an inflammatory cytokine in obese compared to normal weight individuals.

References

Bruun JM, Lihn AS, Verdich C, Pedersen SB, Toubro S, Astrup A, Richelsen B. 2003. Regulation of adiponectin by adipose tissue-derived cytokines: in vivo and in vitro investigations in humans. American Journal of Physiology - Endocrinology **285,** 527–33.

Dinarello CA. 2009. Immunological and infl ammatory functions of the interleukin-1 family. Annual Review of Immunology **27,** 519 -50. http://dx.doi.org/10.1146/annurev.immunol.021908.132612

Frohlich M, Sund M, Frolich M. 2003. Independent association of various smoking characteristics with markers of systemic inflammation in men: results from a representative sample of the general population (MONICA Augsburg Survey 1994/95). European Heart **24,** 1365–1372. http://dx.doi.org/10.1016/S0195-668X(03)00260-4

Gosset A, Tsicopoulos A, Wallaert B, Vannimenus C. 1991. Increased secretion of tumor necrosis factor and interleukin-6 by alveolar macrophages consecutive to the development of the late asthmatic reaction. Journal of Allergy and Clinical Immunology **88,** 561–571. http://dx.doi.org/10.1016/0091-6749(91)90149-I

Grau AJ, Buggle F, Becher H, Werle E, HackeW. 1996. The association of leukocyte count, fibrinogen and C- reactive protein with vascular risk factors and ischemic vascular diseases. Thrombosis Research **82,** 245-55. http://dx.doi.org/10.1016/0049-3848(96)00071-0

Guler N, Kirerleri E, Ones U, Tamay Z, Salmayenli N, Darendeliler F.2004. Leptin: does it have any role in childhood asthma? Journal of Allergy and Clinical Immunology **114(2),** 254-9. http://dx.doi.org/10.1016/j.jaci.2004.03.053

Kahn CR. 2000. Triglycerides and toggling the tummy. Nature Genetics **25**, 6–7. http://dx.doi.org/10.1038/75610

Kontzias A, Efthimiou P. 2012. The use of canakinumab, a novel IL-1beta long-acting inhibitor, in refractory adult-onset still's disease. Seminars in Arthritis and Rheumatism
http://dx.doi.org/10.1016/j.semarthrit.2012.03.004

Kyle E, Michael S, Gulshan S. 2009. Asthma, obesity and type 2 diabetes: mechanisms, management and prevention. Diabetes Voice **54(2),** 30-33.

Matsuki T, Horai R, Sudo K, Iwakura Y. 2003. IL-1 plays an important role in lipid metabolism by regulating insulin levels under physiological conditions. Journal of Experimental Medicine **198(6),** 877-88.
http://dx.doi.org/10.1084/jem.20030299

Nystad W, Meyer HE, Nafstad P, Tverdal A, Engeland A. 2004. Body mass index in relation to adult asthma among 135,000 Norwegian men and women. American Journal of Epidemiology **160(10),** 969-76.
http://dx.doi.org/10.1093/aje/kwh303

Osborn O, Brownell SE, Sanchez-Alavez M, Salomon D, Gram H, Bartfai T. 2008. Treatment with an Interleukin 1 beta antibody improves glycemic control in diet-induced obesity. Cytokine **44(1),** 141-8.
http://dx.doi.org/10.1016/j.cyto.2008.07.004

Pickup JC, Mattock MB, Chusney GD, Burt D. 1997. NIDDMas a disease of the innate immune system: association of acute-phase reactants and interleukin-6 with metabolic syndrome X. Diabetologia **40,** 1286-92.
http://dx.doi.org/10.1007/s001250050822

Quartier P, Allantaz F, Cimaz R. 2011. A multicentre, randomised, double-blind, placebo-controlled trial with the interleukin-1 receptor antagonist anakinra in patients with systemic-onset juvenile idiopathic arthritis (ANAJIS trial). Annals of the Rheumatic Diseases **70,** 747–754.
http://dx.doi.org/10.1136/ard.2010.134254

Rui L, Yuan M, Frantz D. 2002. SOCS-1 and SOCS-3 block insulin signaling by ubiquitin-mediated degradation of IRS1 and IRS2. Journal of Biological Chemistry **277,** 42394 -8.
http://dx.doi.org/10.1074/jbc.C200444200

Rui L, Yuan M, Frantz D. 2002. SOCS-1 and SOCS-3 block insulin signaling by ubiquitin-mediated degradation of IRS1 and IRS2. Journal of Biological Chemistry **277,** 42394 -8.
http://dx.doi.org/10.1074/jbc.C200444200

Saltevo J, Vanhala M, Kautiainen H, Laakso M. 2007. Levels of adiponectin, C-reactive protein and interleukin-1 receptor antagonist are associated with the relative change in body mass index between childhood and adulthood. Diabetes & vascular disease research **4(4),** 328-31.
http://dx.doi.org/10.3132/dvdr.2007.060

Segura MN, Murillo GE, Rojas-Dotor S, Rico G, Martínez HL, Sandoval SC. 2007. Inflammatory markers associated with asthma and body mass index. Revista Alergia México **54(6),** 196-200.

Shore SA. 2007. Obesity and asthma: implications for treatment. Current Opinion in Pulmonary Medicine's **13(1),** 56-62.
http://dx.doi.org/10.1097/MCP.0b013e3280110196

Snehalatha C, Yamuna A, Ramachandran A. 2008. Plasma Adiponectin Does Not Correlate With Insulin Resistance and Cardiometabolic Variables in Nondiabetic Asian Indian Teenagers. Diabetes Care **31(12),** 2374-9.
http://dx.doi.org/10.2337/dc08-1083

Spiegelman BM, Flier JS. 2001. Obesity and regulation of energy balance. Cell **104,** 531–543.
http://dx.doi.org/10.1016/S0092-8674(01)00240-9

Taniguchi CM, Emanuelli B, Kahn CR. 2006. Critical nodes in signalling pathways: insights into insulin action. Nature Reviews Molecular Cell Biology **7,** 85 -96.
http://dx.doi.org/10.1038/nrm1837

Taniguchi CM, Emanuelli B, Kahn CR. 2006. Critical nodes in signalling pathways: insights into insulin action. Nature Reviews Molecular Cell Biology **7,** 85 -96.
http://dx.doi.org/10.1038/nrm1837

Tschop M, Wawarta R, Riepl RL, Friedrich S, Bidlingmaier M, Landgraf R, Folwaczny C. 2001. Post-prandial decrease of circulating human ghrelin levels. Journal of Endocrinological Investigation **24,** 19–21.

Urboniene D, Sakalauskas R, Sitkauskiene B. 2008. C-reactive protein levels in patients with chronic obstructive pulmonary disease and asthma. Medicina (Kaunas) **44(11),** 833-40.

Yukihiro Y, Hiroshi H, Ikuo S, Motowo T, Matsuo T, Koichi M. 2002. Correlation of the adipocyte-derived protein adiponectin with insulin resistance index and serum high-density lipoprotein-cholesterol, independent of body mass index, in the Japanese population. Clinical Science **103,** 137–142.
http://dx.doi.org/10.1042/CS20010336