



RESEARCH PAPER

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Insulin resistance and anthropometric parameters: A cross-sectional study in adult men

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Key words: Obesity, insulin resistance, anthropometrical markers.

doi: <http://dx.doi.org/10.12692/ijb/3.4.228-233>

Article published on April 22, 2013

Abstract

To determine relationship between insulin resistance and anthropometrical indexes, twenty four healthy sedentary men aged 33 ± 3.8 years were participated in this study by accessible sampling. Fasting serum of all participants were collected in order to measuring insulin and glucose for calculation insulin resistance after an overnight fast. Body weight, body fat percentage, body mass index (BMI) and other parameters of anthropometrical indexes were also measured in all subjects. The correlations between variables were determined using the bivariate correlation test. High positive associations was observed between insulin resistance with all anthropometrical indexes as body weight ($p = 0.005$, $r = 0.533$), body fat percentage ($p = 0.002$, $r = 0.600$), visceral fat ($p = 0.000$, $r = 0.750$), BMI ($p = 0.000$, $r = 0.711$) and other anthropometrical markers in studied subjects. Based on these data, we can suggest anthropometrical markers as a precise predictor of insulin resistance in obese men.

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Introduction

Obesity is associated with increased adipose tissue mass and is known to be a major risk factor for metabolic disorders such as diabetes, hypertension, and atherogenic diseases (Spiegelman *et al.*, 1996; Reaven, 1995). However, little information on the influence of obesity on these disorders is available and additional studies are needed in this area.

On the other hand, the previous studies on metabolic disorders have shown insulin resistance to be an independent predictor for the development of hypertension, coronary heart disease or type 2 diabetes (Facchini *et al.*, 2001). Therefore, this raises the question of whether insulin resistance is associated with obesity determinative in these populations. In addition, review of research evidence shows that high-fat diets increase insulin resistance, which results in the development and/or exacerbation of Type 2 diabetes (Kraegen *et al.*, 1991; Storlien *et al.*, 2000).

Several articles were published about promoting factors of insulin resistance in obese subjects or about diseases associated with metabolic syndrome, and each one supported somehow the impact of the factors involved in the body fat levels in promotion of insulin resistance in healthy populations or patients (Park *et al.*, 2007; Morioka *et al.*, 2007). But so far, the specific molecular mechanisms that clearly justify the interaction between insulin function and obesity level cannot be found in literature and in most of new studies the mentioned subjects has been quoted from previous studies. Some of these studies have reported that hormonal factors secreted by adipose tissue are involved in prevalence of insulin resistance (Koebsnick *et al.*, 2008). But little attention was paid to whether fat determining factors such as body weight, body mass index, or body fat percentage directly affect insulin resistance. In line with some previous studies, this research has tried to reinvestigate the relationship between these anthropometric

parameters and insulin resistance in a group of obese adult males.

Materials and methods

The objective of present study was to evaluate relationship between insulin resistance with all anthropometrical indexes as body weight, body fat percentage, body mass index (BMI) and other parameters in twenty four healthy sedentary men aged 33 ± 3.8 years were participated in this study by accessible sampling. The nature and purpose of the study were carefully explained to all participants and written informed consent was obtained. The study protocol was approved by the Research Ethics Committee of University of Social Welfare and Rehabilitation Sciences, Iran.

Subjects were reported to be non-smokers, not currently taking supplements of any kind, and having no major health problems. Those with diabetes, cardiovascular disease were excluded from the study. Participants were included if they had not been involved in regular physical activity/diet in the previous 6 months.

Anthropometric measurements (body height and weight, waist and hip circumference ...) were performed with the subjects wearing light underwear and without shoes. Body weight and height were measured with a standard physician's scale and a stadiometer, respectively when subjects were in a fasting state when the participant had thin clothes on and was wearing no shoes. Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter. Depending on the height and weight, body mass index was calculated using the software of BMI percentile. Visceral fat and body fat percentage was determined using body composition monitor (OMRON, Finland). After anthropometrical measuring, all participants were asked to attend hematology laboratory and a venous blood sample was collected after a 12-h overnight fast for measuring fasting insulin and

glucose concentration in order to calculation of insulin resistance by special formula (Matthews *et al.*, 1985).

Statistical analysis

Statistical analysis was done for all the parameters. The Kolmogorov-Smirnov test was applied to determine the variables with normal distribution. The correlations between variables were determined using the bivariate correlation test. A p-value of less than 0.05 was considered to be statistically significant.

Results

In this study, the relationship between insulin resistances with anthropometrical markers was evaluated in sedentary adult men. The analysis data showed that the subjects have a BMI of 32.4 ± 3.7 kg/m². They also have fasting glucose 105 ± 16.5 mg/dL and fasting insulin 7.53 ± 2.14 μ IU/ml. The other anthropometric and physiological characteristics of the study participants are described in Table 1. All values are given as mean and standard deviation.

Table 1. Anthropometric and biochemical characteristics of the study participants.

	N	Minimum	Maximum	Mean	Std. Deviation
Insulin Resistance	24	.82	3.01	1.9571	.64136
Age	24	26	40	33.33	3.763
Body weight (kg)	24	84	140	102.17	15.018
Height (m)	24	168.50	185.00	177.4167	4.56277
Abdominal Circumference (cm)	24	99	138	109.00	10.675
Hip Circumference (cm)	24	98	135	109.05	10.000
Body Mass Index (kg/m ²)	23	29	41	32.43	3.742
Body Fat (%)	24	24	41	32.30	4.108
Visceral Fat	24	10	20	14.04	2.437
Valid N (listwise)	23				

The finding of Pearson method showed that insulin resistance in studied subjects was positively associated with body weight ($p = 0.005$, $r = 0.533$, Fig 1). In addition, a significant positive correlation was found between insulin resistance with body mass index ($p = 0.000$, $r = 0.711$). Pearson correlation analyses revealed that insulin resistance were also positively associated with abdominal circumference ($p = 0.001$, $r = 0.642$), visceral fat ($p = 0.000$, $r = 0.750$, Fig 2), hip circumference ($p = 0.019$, $r = 0.475$) and body fat percentage ($p = 0.002$, $r = 0.600$, Fig. 3).

Discussion and conclusion

Our study data showed that a high strong significant correlation between insulin resistance and all anthropometrical markers in studied subjects. Despite far less number of subjects in the present study compared to previous studies in this field, a

highly significant relationship was observed between insulin resistance and the mentioned indices.

Insulin resistance was known to be one of the main risk factors for cardiovascular diseases, presenting high morbimortality and socioeconomic expenses. Previous studies simultaneously showing the presence of hyperinsulinemia and insulin resistance in the forearm of obese subjects (Rabinowitz *et al.*, 1962).

Recent evidence has shown that is associated with visceral obesity; arterial hypertension; intolerance to glucose and to type 2 diabetes; dyslipidaemia; hyperuricemia, and other metabolic changes (Silva *et al.*, 2007; Ribeiro Filho *et al.*, 2006). Several studies have suggested that even in adolescents, overweight is associated with increased dyslipidemia, hypertension, and insulin resistance (Duncan *et al.*, 2004). A growing body of literature suggests that Insulin resistance is increased with increasing obesity,

especially central obesity, and there is associated impairment of glucose tolerance, dyslipidaemia and systemic hypertension (Dixon *et al.*, 2003).

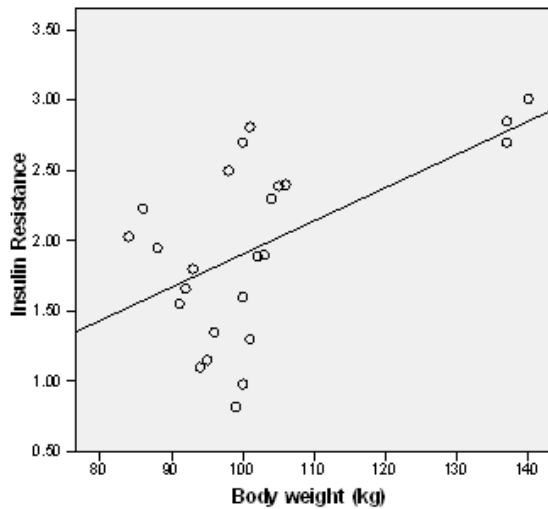


Fig. 1. The correlation pattern between insulin resistance and body weight in studied subjects.

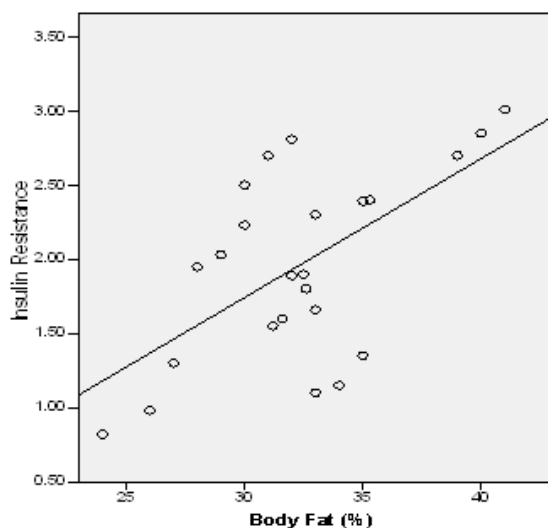


Fig. 2. The correlation pattern between insulin resistance and body fat (%) in studied subjects.

Measuring of the abdominal circumference for the diagnosis of the insulin resistance is in the proposals of the European Group for the Study of Insulin resistance (Balkau *et al.*, 1999), the International Diabetes Federation (Alberti *et al.*, 2006) and of the National Cholesterol Education Program (NCEP-ATP III). To support these data, our study findings showed that abdominal circumference have a high positive significant with insulin resistance in studied subjects. We also observed a strong positive correlation

between visceral fat and insulin resistance. Close relationship between body weight and insulin resistance is another finding of present study. On the other hand, it has been demonstrated that Weight reduction increases insulin sensitivity and improves diseases associated with the metabolic syndrome (Sjostrom *et al.*, 1999; Dengel *et al.*, 1998). Beneficial effects of weight loss or reduced body fat levels on insulin resistance or beta cell function was reported by other previous studies in obese or its related diseases (Polonsky *et al.*, 1994).

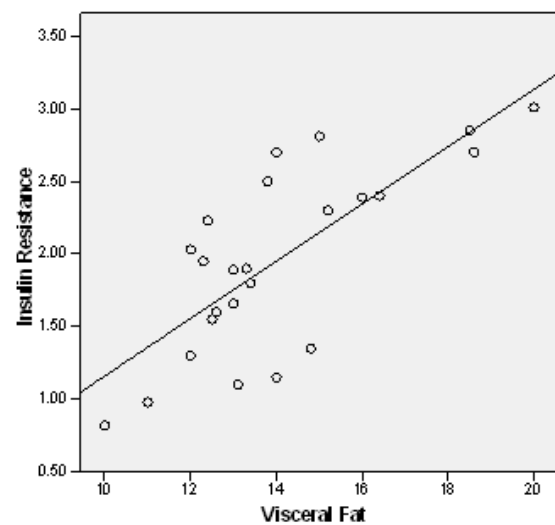


Fig. 3. The correlation pattern between insulin resistance and visceral fat in studied subjects.

Accumulating evidence indicates that with increasing adiposity, adipose tissue undergoes marked morphologic and physiologic changes, including the infiltration of macrophages and the release of proinflammatory cytokines (Weisberg *et al.*, 2003). The body mass index is weight in kilograms divided by height in meters squared (Monteiro *et al.*, 1998). It has been demonstrated that body mass index represents the most known and used indicator of nutritional status to evaluate adults and old-aged, due to its easiness of application and low cost (Ana Carolina *et al.*, 2010). Although some previous has reported that due to the BMI's incapacity of distinguishing thin or fat body mass, its use for the prediction of IR can overestimate the risk in individuals with increased quantity of muscular mass (Ana Carolina *et al.*, 2010), our study findings also

showed a significant positive correlation in insulin resistance with body mass index in studied subjects.

In an aggregation, the present study supports the close relationship between any of the anthropometric indices such as weight, waist and hip circumferences, waist to hip ratio, and body mass index with insulin resistance in obese subjects. Moreover, a very close relationship was found between insulin resistance index and the two indices of body fat percentage and visceral fat which are considered as the major determining factors of obesity. Reminding again, a very close relationship was observed between these components and insulin resistance while the number of obese people in the study was lower than samples in previous studies. The significant close correlation in this study with a very small sample size strongly supports the potential role of obesity and anthropometric indices in promotion and prevalence of insulin resistance in healthy subjects or patients. These findings support also other results of certain studies which emphasized on the role of inflammatory cytokines secreted by adipose tissue in development of insulin resistance.

References

- Alberti KG, Zimmet P, Shaw J.** 2006. Metabolic syndrome--a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabetic Medicine* **23(5)**, 469-80.
- Ana Carolina V, Lina R, Gilberto R, Rita de Cassia R, Sylvia F, Bruno G.** 2010. Anthropometric indicators of insulin resistance. *Arquivos Brasileiros de Cardiologia* **95(1)**, 14-22.
<http://dx.doi.org/10.1590/S0066-782X2010001100025>
- Balkau B, Charles MA.** 1999. Comment on the provisional report from the WHO consultation. European Group for the Study of Insulin Resistance (EGIR). *Diabetic Medicine* **16(5)**, 442-3.
- Dengel DR, Hagberg JM, Pratley RE, Rogus EM, Goldberg AP.** 1998. Improvements in blood pressure, glucose metabolism, and lipoprotein lipids after aerobic exercise plus weight loss in obese, hypertensive middle-aged men. *Metabolism* **47**, 1075-1082.
[http://dx.doi.org/10.1016/S0026-0495\(98\)90281-5](http://dx.doi.org/10.1016/S0026-0495(98)90281-5)
- Dixon JB, Dixon AF, O'Brien PE.** 2003. Improvements in insulin sensitivity and β -cell function (HOMA) with weight loss in the severely obese. *Diabetic Medicine* **20(2)**, 127-34.
- Duncan GE, Li SM, Zhou XH.** 2004. Prevalence and trends of a metabolic syndrome phenotype among U.S. adolescents, 1999-2000. *Diabetes Care* **27**, 2438-2443.
- Facchini FS, Hua N, Abbasi F, Reaven GM.** 2001. Insulin resistance as a predictor of age-related diseases. *Journal of Clinical Endocrinology & Metabolism* **86**, 3574-8.
<http://dx.doi.org/10.1210/jc.86.8.3574>
- Koebnick C, Roberts CK, Shaibi GQ, Kelly LA, Lane CJ, Toledo-Corral CM.** 2008. Adiponectin and Leptin are Independently Associated with Insulin Sensitivity, but not with Insulin Secretion or Beta-cell Function in Overweight Hispanic Adolescents. *Hormone Metabolic Research Journal* **40(10)**, 708-12.
<http://dx.doi.org/10.1055/s-2008-1077097>
- Kraegen EW, Clark PW, Jenkins AB, Daley EA, Chisholm DJ, Storlien LH.** 1991. Development of muscle insulin resistance after liver insulin resistance in high fat fed rats. *Diabetes* **40**, 1397-403.
- 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(7)**, 412-419.
- Monteiro JC. Obesidade.** 1998. diagnóstico, métodos e fundamentos. In: Halpern A, Matos AFG,

Suplicy HL, Mancini MC, Zanella MT. Obesidade. São Paulo: Lemos p. 31.

Morioka T, Asilmaz E, Hu J, Dishinger JF, Kurpad AJ, Elias CF. 2007. Disruption of leptin receptor expression in the pancreas directly affects β cell growth and function in mice. *Journal of Clinical Investigation* **117(10)**, 2860-8.
<http://dx.doi.org/10.1172/JCI30910>

Park S, Hong SM, Lee JE, Sung SR. 2007. Exercise improves glucose homeostasis that has been impaired by a high-fat diet by potentiating pancreatic B- cell function and mass through IRS2 in diabetic rats. *Journal of Applied Physiology* **103(5)**, 1764-71.
<http://dx.doi.org/10.1152/jappphysiol.00434.2007>

Polonsky KS, Gumbiner B, Ostrega D, Griver K, Tager H, Henry RR. 1994. Alterations in immunoreactive proinsulin and insulin clearance induced by weight loss in NIDDM. *Diabetes* **43**, 871-877.

Rabinowitz D, Zierler KL. 1962. Forearm metabolism in obesity and its response to intra-arterial insulin: characterization of insulin resistance and evidence for adaptive hyperinsulinism. *Journal of Clinical Investigation* **41**, 2173-2181.
<http://dx.doi.org/10.1172/JCI104676>

Reaven GM. 1995. The fourth Musketeer from Alexandre Dumas to Claude Bernard. *Diabetologia* **38**, 3-13.

Ribeiro Filho FF, Mariosa LS, Ferreira SR, Zanella MT. 2006. Gordura visceral e síndrome

metabólica: mais que uma simple's associação. *Arquivos Brasileiros de Endocrinologia & Metabologia* **50(2)**, 230-8.

<http://dx.doi.org/10.1590/S0004-27302006000200009>

Silva EA, Flexa F, Zanella MT. 2007. Obesidade abdominal, resistência à insulina e hipertensão: impacto sobre a massa e a função do ventrículo esquerdo em mulheres. *Arquivos Brasileiros de Cardiologia* **89(2)**, 86-92.

<http://dx.doi.org/10.1590/S0066-782X2007001400003>

Sjostrom CD, Lissner L, Wedel H, Sjostrom L. 1999. Reduction in incidence of diabetes, hypertension and lipid disturbances after intentional weight loss induced by bariatric surgery: the SOS Intervention Study. *Obesity Research* **7**, 477-484.

<http://dx.doi.org/10.1002/j.1550-8528.1999.tb00436.x>

Spiegelman BM, Flier JS. 1996. Adipogenesis and obesity: rounding out the big picture. *Cell* **87(3)**, 377-89.

Storlien LH, Higgins JA, Thomas TC. 2000. Diet composition and insulin action in animal models. *British Journal of Nutrition* **83**, 85-90.

<http://dx.doi.org/10.1017/S0007114500001008>

Weisberg SP, McCann D, Desai M, Rosenbaum M, Leibe IRL. 2003. Obesity is associated with macrophage accumulation in adipose tissue. *Journal of Clinical Investigation* **112**, 1796-1808.

<http://dx.doi.org/10.1172/JCI200319246>