The Effect of Weight Reduction by Energy-Restrictive Diet, on Serum Fasting Insulin and Glucose Levels, in Non-diabetic Obese Men

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Weight loss reduces many of the health hazards associated with obesity including insulin resistance, diabetes mellitus, hypertension, dyslipidaemia, sleep apnoea, hypoxemia and hypercarbia, and osteoarthritis. This study is based on the hypothesis that weight-reduction through caloric restriction may normalize the obesity-induced hyperinsulinaemia or insulin resistance in middle-aged obese men who are at a great risk of development of glucose intolerance and diabetes mellitus. Twenty five non-diabetic males with mean age 35.16± 0.48 years (ranged 30 to 40 years) and mean body mass index (BMI) 32.71± 0.34 kg/m^2 were placed at a low caloric diet (1000-1400 kcal), for 60 days. Following 60-day dietary treatment BMI reduced from 32.71± 0.34 to 29.79± 0.36 kg/m^2 (P<0.001). This reduction in BMI was accompanied by decrease in fasting levels of insulin (from 217.48± 2.73 to 147.08± 3.83 pmol/l with P<0.001) and glucose (from 91.08±1.35 to 83.36± 1.41 mg/l with P<0.001). The findings of the present study are suggestive that weight reduction through caloric restriction is an effective measure to normalize the obesity-induced hyperinsulinaemia in middle-aged obese men, hence decreasing the risk of development of insulin resistance and glucose intolerance.

Keywords: Insulin resistance, obesity, glucose intolerance, body weight, body mass index

One of the most important health goals for middle-aged non-diabetic men is to develop strategies to delay or prevent the onset of major diseases, including cardiovascular disease and diabetes. An understanding of the variables related to insulin sensitivity in middle-aged men and of the changes in insulin level that are occurring during this time may be important in the development of such strategies. Weight loss, in addition to improving insulin sensitivity[1], leads to blood pressure reduction[2] even more effectively than does antihypertensive medication with beta-adrenergic antagonists[3]. From a physiologic viewpoint, insulin resistance can be defined as a state in which a normal amount of insulin produces a subnormal biologic response[4]. An elevated circulating level of endogenous insulin is a common feature of insulin resistance, and in the absence of a defect in insulin secretory capacity this level correlates well with more sophisticated measures of insulin sensitivity[5]. Hyperinsulinaemia results not only from increased secretion of insulin by beta cells of pancreas, but also from impairment of receptor-mediated clearance of insulin by resistant peripheral target cells[6]. Hyperinsulinaemia is a well-recognized if incompletely understood concomitant of obesity. Diminished sensitivity to the effects of insulin on glucose uptake and metabolism in skeletal muscle, commonly referred to as "insulin resistance," is known to contribute to the hyperinsulinaemia of obesity by evoking an increase in pancreatic insulin secretion in order to maintain euglycaemia. Decreased hepatic extraction of insulin in obese persons appears to be involved as well, as a consequence of increased levels of free fatty acids in the portal blood[7]. Interest in the hyperinsulinaemia of obesity has been rekindled by some clinical and epidemiologic studies that demonstrate an association between hypertension and hyperinsulinaemia in the obese[8,9,10]. This study was designed to investigate the association of weight loss by energy restrictive diet with improvement in insulin resistance in Pakistani non-diabetic obese men.

Material and Methods

Twenty five men aged 30 to 40 years, who presented with obesity (body mass index 30 or more kg/m^2), wished to lose weight by diet, and did not have diabetes mellitus when screened by a 75-gram oral glucose tolerance test[11]. All subjects were healthy and not taking any drug that could alter insulin level and effect. Before dietary treatment, baseline weight and height were recorded and fasting blood samples were drawn for estimation of serum insulin and glucose (as baseline values). All subjects were advised to continue their usual physical activity for the whole period of study that was 60 days. They were placed on low-caloric diet (1000-1400 kcal) based on the meal planning exchange list with total daily intake of approximately 134 g carbohydrates, 68 g protein, and 47 g fat. Weight was recorded at the end of 60-day dietary treatment for comparison with the baseline value. Fasting blood sample was also taken at the end of study for assay of insulin and glucose.

Sera were obtained following clotting of blood samples and were stored at –30°C until assay. Serum insulin was determined by enzyme-immunological test using commercially available reagent kit (Enymun-Test® Insulin, Cat. No. 1289101, Boehringer Mannheim, GmbH, Germany). Glucose in the serum samples was estimated by enzymatic colorimetric method using commercially available reagent kit (Cod.: 10001190, Spinreact, Spain).

Results

Following 60-day dietary treatment body mass index

(BMI) was significantly reduced by 8.97%. After dietary treatment the reduction in fasting serum insulin level was 32.54%. Fasting glucose level decreased from 91.08±1.35 to 83.36±1.41 (8.52% reduction).

TABLE 1: Baseline and final values of body mass index (BMI), serum insulin and serum glucose. Data expressed as mean ± SEM

	Baseline	Final	Change	% Change	P value
Age (years)	35.16± 0.48				
BMI (kg/m^2)	32.71± 0.34	29.79± 0.36	-2.92±0.07	-8.97±0.24	<0.001
Fasting glucose (mg/dl)	91.08±1.35	83.36± 1.41	-7.72±0 .43	-8.52±0.49	<0.001
Fasting insulin (pmol/l)	217.48± 2.73	147.08± 3.83	-70.40± 2.25	-32.54± 1.14	<0.001

Discussion

It has been suggested that insulin sensitivity decreases progressively as BMI increases[12]. In the present study, weight reduction by low caloric diet for 60 days resulted in marked improvement in insulin sensitivity. Fasting serum glucose levels decreased along with reduction in fasting insulin levels, following reduction in BMI by energy restrictive diet and this finding supports the findings of other studies[13,14,15]. In the present study, about 8.97% reduction in BMI in obese Pakistani men through energy-restrictive diet for 60 days, led to significant reductions in fasting serum glucose and insulin concentrations.

The findings of present study are suggestive that obesity is one of the major causes of glucose intolerance in middle-aged Pakistani males. It has been reported that obesity is associated with metabolic abnormalities that are considered to be risk factors for cardiovascular disease, including impaired glucose tolerance and non-insulin-dependent diabetes mellitus, hypertension, and an abnormal lipoprotein profile[16,17,18]. In another relatively large study[19], men and women who gained 10 pounds or more between ages 40 and 60 years had a similar associated risk for developing diabetes. The findings of the present study emphasise the continuum that exists between weight gain and the risk for developing glucose intolerance or non-insulin-dependent diabetes mellitus in middle-aged Pakistani men. Persons with a preponderance of abdominal fat tend to be insulin-resistant and are especially prone to develop non-insulin-dependent diabetes mellitus[20].

So weight reduction by caloric restriction may be an effective measure to normalise the obesity-induced hyperinsulinaemia and elevated fasting glucose level in non-diabetic obese Pakistani men.

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