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# Differences in the body composition and biochemistry in women grouped as normal-weight, overweight and obese according to body mass index and their relation with cardiometabolic risk

**Research Article**

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**Abstract:** Morbidity of obesity-related diseases tends to increase due to a rise in the body mass index (BMI). We aimed to investigate how the body composition and biochemical parameters change while BMI increases in adult women were categorized as so: as normal weight, overweight and obese. Our objectives are to study the effects of those changes in the development of metabolic disturbances and to find out which parameters are the most sensitive to predict cardiometabolic risks. Three hundred and twenty two records of adult women (mean age: 38.62±12.71 year) who admitted to our unit concerning about losing or preserving their weights, were analyzed in the study. All patients had undergone anthropometric measurements and body composition analyses as well as some biochemical tests. Body composition analyses were performed by means of the Bioelectrical Impedance Analyzer (BIA). Increase in BMI significantly increased the body fat, blood sugar, insulin, triglyceride and uric acid levels. BMI and circumference of the waist were significantly and negatively correlated with the ratio of body water and lean mass/fat mass. However they were positively correlated with the ratio of fat mass and basal metabolism. Furthermore, it was also found that BMI and circumference of the waist were significantly and positively correlated with level of fasting blood sugar, insulin, triglyceride, homeostasis model assessment insulin resistance (HOMA-IR), uric acid and fibrinogen levels, and negatively correlated with high density lipoprotein (HDL) cholesterol level. In multiple regression analyses, circumference of waist measurements was significantly correlated with insulin, triglyseride and HDL, whereas the correlation between BMI and these parameters was not found significant. Total body fat mass (as %) showed significant correlation only with HDL-C level. It could be said that obesity which is a disorder that causes many health complications and affects the quality of life in the short and long term could be prevented or cured by keeping negative environmental conditions under control. According to our results, visceral adipose tissue (VAT) measurement was thought to be more related for metabolic and cardiovascular disorders rather than BMI. We also propose to test fasting blood glucose, insulin, triglyceride, HDL, fibrinogen, homocystein (HOM) levels along with VAT measurements to predict more truly about not only global cardiometabolic risk but also dementia in later life.

**Keywords:** *Body composition • Obesity • Insulin • Triglyceride • Cardiometabolic risk*

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## 1. Introduction

Obesity is a chronic, multifactorial disorder which is also an important public health issue attaining global epidemic (globosity) rates in many countries and negatively affecting the expectancy and quality of life. Obesity does not only affect an individual's psychological condition but also increases the risk of contracting many chronic diseases. Morbidity of obesity-related diseases tends to increase with the rise in the body mass index (BMI). Obese patients are under greater risk in terms of disorders such as the coronary artery disease, hypertension, hyperlipidemia, diabetes, some types of cancer, cerebrovascular incidents, osteoarthritis, pulmonary diseases and sleep apnea etc [1]. Moreover, it is reported that there is a positive correlation between obesity and depression by the increase in the specific nutritional psychopathologies of obese people such as a decrease in their self-esteem [2]. For this reason, reducing weight plays a key role in preventing those who are obese from many chronic diseases and improve the prognosis for any existing disease.

According to the clasification of Body Mass Index (BMI=Weight (kg) / Height ($m^2$)), people with a BMI of 18.5-24.9 kg/$m^2$ are considered to be within normal range while those with a BMI of 25.0-29.9 kg/$m^2$ are considered overweight and of 30.0-39.9 kg/$m^2$ are obese. Individuals with a BMI of over 40 kg/$m^2$ are classified as being morbidly obese [1]. Independent from underlying reasons, overweight and obesity do not only cause aesthetic problems but also significantly increase mortality and morbidity. In addition, it is also proven that insulin resistance and metabolic syndrome arising out of obesity accelerate cognitive degeneration in later years, and makes individuals more susceptible to the Alzheimer disease and the formation of vascular dementia [3-10].

Although the body mass index is a simple tool and widely used in clinical practice to determine obesity, an elevated BMI alone is not sufficient to diagnose visceral adiposity and related cardiometabolic risks. So, additional anthropometric and biochemical measurements seem to be needed. In this study we wanted to investigate how the body composition and biochemistry changes, as BMI is increasing from normal weight to overweight and obese. Since, the body fat content and its distribution differ according to age, sex, ethnicity, physical activity level, disease status etc. only adult women who were not in menopause and didn't have any known disease or drug usage were included in this study. We also aimed that which parameters were the best predictors to estimate obesity related metabolic risks.

## 2. Material and Methods

In our obesity clinic, we made anthropometric and biochemical tests as a primary investigation for our patients who came to lose weight or to keep their weight under control. Our routine anthropometric tests include measurements of height, weight, waist and hip circumferences, and analysis of body composition. In the course of our study, we reviewed the history of adult women who came to our obesity clinic with weight problems; 322 adult women were selected (mean age: 38.62±12.71 year), our candidates had no history of endocrine-metabolic, autoimmune, psychiatric problems in their first visit to our clinic and had no history of using drugs and did not reach menopause yet. Patients who had showed any medical problems in the primary tests and were got medical therapy and followed up were discarded from the study. All data about the patients' body composition analysis, anthropometric and biochemical tests was thoroughly studied.

Body composition analyses are performed by means of the Bioelectrical Impedance Analyzer (Bodystat 1500MDD, England). Blood samples are taken in the morning from the antecubital vein after an overnight fasting for some biochemical parameters (serum fasting blood glucose (FBG), insulin, thyroid stimulating hormone (TSH), free T3 (FT3), free T4 (FT4), high sensitive C reactive protein (hsCRP), uric acid, triglyceride, high density lipoprotein (HDL), low density lipoprotein (LDL) and total cholesterol, homocystein (HOM), vitamin B12, folic acid, glycosylated hemoglobin A1C (HBA1C), fibrinogen). Body mass index and homeostasis model assessment (HOMA) index were calculated based on related formula [11].

Biochemical parameters (serum FBG, uric acid, triglyceride, HDL, LDL and total cholesterol) were measured using an Olympus AU 800 autoanalyzer (Olympus, Japan). FBG was analyzed using the Hexokinase method. The method for uric acid was Uricase. HDL, total cholesterol and triglycerides were measured by enzymatic methods in all samples. Serum hsCRP concentrations were determined with immunonephelometry using BN II Systems Analyzer (Dade Behring, Malburg, Germany). FT3, FT4 and TSH were measured by Immulite 2000 (DPC; LosAngeles, USA). FT3 and FT4 were measured by competitive analog based immunoassay. TSH levels were determined by two side chemiluminescent immunometric assay. Insulin was measured with Immulite 2000 analyzer (DPC, USA) by Chemiluminescent immunometric assay. Serum B12, folic acid levels were measured by radioimmunoassay (RIA) (DPC,USA). Plasma level of



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**Table 1.** Comparison of anthropometric measurements, body composition and biochemical parameters by BMI groups.

| BMI (kg/m²) | 18.5-24.9 n=57 | | 25-29.9 n=83 | | 30+ n=182 | | |
|---|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | Mean | SD | p |
| Weight (kg) | 57.50 | 6.60 | 69.24 | 6.14 | 91.14 | 16.41 | <0.001 |
| BMI (kg/m²) | 22.37 | 1.85 | 27.35 | 1.71 | 37.00 | 6.36 | <0.001 |
| Waist Circumference (cm) | 72.20 | 5.48 | 82.32 | 4.83 | 101.32 | 12.11 | <0.001 |
| Body water % | 54.92 | 6.95 | 48.70 | 4.02 | 44.91 | 4.34 | <0.001 |
| Fat mass % | 26.04 | 4.98 | 33.35 | 5.85 | 37.28 | 5.61 | <0.001 |
| Lean Mass % | 73.97 | 4.99 | 66.15 | 6.69 | 62.52 | 5.65 | <0.001 |
| Basal Met.(kcal) | 1417.19 | 102.68 | 1516.33 | 113.27 | 1732.86 | 221.08 | <0.001 |
| Glucose (mg/dl) | 89,05 | 15.31 | 92.14 | 10.04 | 96.08 | 12.89 | <0.01 |
| Insulin (mIU/ml) | 8,96 | 5,01 | 11,05 | 5,67 | 17,69 | 11,04 | <0.001 |
| Triglyceride (mg/dl) | 74.44 | 30.94 | 103.45 | 54.58 | 126.24 | 66.94 | <0.001 |
| Cholesterol (mg/dl) | 194.65 | 34.75 | 205.17 | 44.22 | 203.80 | 40.38 | 0.405 |
| HDL-C (mg/dl) | 55.54 | 10.10 | 55.77 | 16.25 | 48.61 | 11.40 | <0.001 |
| LDL-C(mg/dl) | 125.79 | 32.51 | 130.20 | 37.95 | 133.34 | 37.28 | 0.503 |
| HOMA-IR | 1.96 | 1.51 | 2.70 | 1.50 | 4.55 | 2.42 | <0.01 |
| Uric Acid (mg/dl) | 3.73 | .58 | 4.01 | 1.02 | 4.85 | 1.35 | <0.01 |

*SD: Standard deviation, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, HOMA-IR: Homeostasis model assessment-insulin resistance.*

homocysteine was determined by high performance liquid chromatography (HPLC) (by Agilent 1100 Series), coupled with fluorescence detector. Plasma fibrinogen levels were measured by Dade Behring BCT analyzer (Dade Behring, Malburg, Germany).

Data analyses were assessed by using the SPSS for windows 10.0 software. The Anova, Kruskal Walls, Multiple-Regression analysis and Pearson methods were used. Multiple Regresyon analyse was applied to identify the best predictor for cardiometabolic risk factors whether only BMI or adipose tissue measurements together with several biochemical parameters are the best.

## 3. Results

Changes in anthropometric measurements, body composition and biochemical parameters by BMI groups were as presented in Table 1. As BMI increases, circumference of waist and body fat ratio increase and the ratio of body water and ratio of lean body mass were significantly decreased. In addition, it was also found that increase in the BMI causes significant increases in the levels of blood sugar, insulin, triglyceride and uric acid, but significant decrease in HDL-C level. CRP, THS, HBA1C, ALT, AST, Fibrinogen and Homocystein levels didn't show any significant differences between BMI groups.

The correlation of BMI and waist circumference with the body composition and biochemical parameters are as presented in Table 2.

BMI and waist circumference were significantly and negatively correlated with ratio of body water and lean mass/fat mass. They were positively correlated with ratio of fat mass and basal metabolism. Furthermore, it was also found out that BMI and circumference of waist were significantly and positively correlated with level of fasting blood sugar, insulin, triglyceride, HOMA-IR, uric acid and fibrinogen levels. However, BMI and waist circumference were significantly and negatively correlated with HDL cholesterol.

In multiple-regression analyses, circumference of waist and body fat percentage for HDL; circumference of waist for insulin; circumference of waist for triglyceride were found significantly correlated (Table 3).

## 4. Discussion

The obesity epidemic is a major public health problem worldwide. Adult obesity is associated with increased morbidity and mortality. Measurement of abdominal obesity is strongly associated with increased cardiometabolic risk, cardiovascular events, and mortality. Although waist circumference is a crude measurement, it correlates with obesity and visceral fat amount, and is a surrogate marker for insulin



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**Table 2.** Correlation of the body mass index and waist circumference with the body composition and biochemical parameters.

| | BMI (kg/m²) n=322 | | Waist Circumference (cm) n=278 | |
|---|---|---|---|---|
| | r | p | r | p |
| Waist Circumference (cm) | .915 | <0.001 | | |
| BMI (kg/m²) | .023 | .799 | .696 | <0.001 |
| Body water (%) | -.659 | <0.001 | -.617 | <0.001 |
| Body fat mass (%) | .584 | <0.001 | .598 | <0.001 |
| Lean body mass (%) | -.115 | 0.063 | -.112 | 0.091 |
| LEAN/FAT | -.740 | <0.001 | -.692 | <0.001 |
| Basal Metab. (kcal) | .772 | <0.001 | .738 | <0.001 |
| Glucose (mg/dl) | .314 | <0.001 | .351 | <0.001 |
| Insulin (mIU/ml) | .484 | <0.001 | .512 | <0.001 |
| Triglyceride (mg/dl) | .271 | <0.001 | .358 | <0.001 |
| Cholesterol (mg/dl) | .035 | .556 | .055 | .399 |
| HDL-C (mg/dl) | -.248 | <0.001 | -.240 | <0.001 |
| LDL-C (mg/dl) | .069 | .266 | .054 | .418 |
| HOMA-IR | .524 | <0.001 | .567 | <0.001 |
| HBA1C (%) | .067 | .653 | .030 | .852 |
| TSH (mIU/ml) | -.066 | .329 | -.072 | .322 |
| Uric Acid (mg/dl) | .409 | <0.001 | .411 | <0.001 |
| ALT (U/L) | .133 | .389 | .176 | .284 |
| AST (U/L) | .127 | .422 | .114 | .503 |
| Fibrinogen (mg/dl) | .312 | <0.001 | .317 | <0.05 |
| Homocystein (umol/L) | .620 | <0.01 | .447 | .072 |

*BMI: Body mass index, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, HOMA-IR: Homeostasis model assessment-insulin resistance, HBA1C: Glycosylated hemoglobin, THS: Tyroid stimulating hormone, ALT: Alanine aminotransferase, AST: Aspartate transaminase*

resistance. A normal waist circumference differs for specific ethnic groups due to different cardiometabolic risk. One criterion for the diagnosis of the metabolic syndrome, according to different study groups, includes measurement of abdominal obesity (waist circumference or waist-to-hip ratio) because visceral adipose tissue is a key component of the syndrome [11-14]. The waist circumference measurement is a simple tool that should be widely implemented in clinical practice to improve cardiometabolic risk stratification.

The metabolic syndrome is a cluster of cardiovascular risk factors associated with obesity. The defining components of the metabolic syndrome are elevated waist circumference, elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), elevated blood pressure, and elevated fasting glucose [15]. The risk of type 2 diabetes mellitus (DM) and cardiovascular diseases (CVD) increases dramatically with the accumulation of these risk factors. The pathophysiologic basis of metabolic syndrom is complex and reflects several interrelated disturbances of glucose and lipid homeostasis.

The expansion of adipose tissue mass, especially of the visceral adipose tissue depot, is observed in the vast majority of individuals carrying the clinical features of the metabolic syndrom. As waist circumference can be used as a crude estimate of visceral fat accumulation, its measurement provides furter information on cardiovascular and type 2 diabetes risks, at any given body mass index value. However, an elevated waist circumference might also be the result of an increased "cardioprotective" subcutaneous adipose tissue

**Table 3.** Multiple-regression analyses results for anthropometric measurements of biochemical parameters related with cardiovascular risk factors.

| | Unstandardized Coefficients | | Standardized Coefficients | t | P |
|---|---|---|---|---|---|
| HDL | Beta | Std. Error | Beta | | |
| (Constant) | 73.436 | 8.237 | | 8.916 | .000 |
| BMI | .113 | .284 | .048 | .397 | .692 |
| Waist Circumference | -.452 | .141 | -.384 | -3.197 | .002 |
| Body fat mass % | .452 | .174 | .193 | 2.597 | .010 |
| Triglyceride | | | | | |
| (Constant) | -58.798 | 35.968 | | -1.635 | .104 |
| BMI | -1.333 | 1.246 | -.124 | -1.070 | .286 |
| Waist Circumference | 2.558 | .612 | .480 | 4.182 | <0.001 |
| Body fat mass % | -.638 | .769 | -.059 | -.830 | .408 |
| Insulin | | | | | |
| (Constant) | -10.665 | 5.413 | | -1.970 | .050 |
| BMI | .133 | .185 | .088 | .717 | .474 |
| Waist Circumference | .280 | .091 | .374 | 3.058 | .003 |
| Body fat mass % | -.175 | .117 | -.118 | -1.493 | .137 |



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mass. So, additional biochemical parameters seem to be needed along with BMI and waist circumference measurements to predict cardiometabolic risks in adult obese people. Some authors proposed the measurement of plasma triglycerides along with waist circumference, the so-called "hypertriglyceridemic waist" might better quantify visceral obesity and its health hazards than waist circumference alone [16,17]. Hypertriglyceridemic waist is thought to represent an altered, dysfunctional and highly lipolytic adipose tissue that is major culprit abnormality behind the metabolic syndrom and associated cardiometabolic risk, independently from classical cardiovascular disease risk factors such as age, sex and plasma low density lipoprotein (LDL) cholesterol levels.

In a study conducted among US adults, there is a high prevalance of clustering of cardiometabolic abnormalities among normal-weight individuals and a high prevalence of overweight and obese individuals who are metabolically healthy [18].

Since there might be several metabolic abnormalities in also normal weight ranges, we wanted to investigate the changes in biochemical and anthropometric measurements among adult female grouped as normal weight, overweight and obese according to known formula as BMI.

In our study, we found a parallel increase in the anthropometric measurements (waist circumference and percentage of body fat) and biochemical parameters (i.e. a significant increase in fasting blood glucose, insulin, triglyceride and levels of uric acid and a significant decrease in HDL levels) with obesity level (Table 1). These findings show that the increase in the obesity level of the individual will increase the risk of metabolic syndrome.

However CRP, TSH, HBA1C, ALT, AST, Fibrinogen and Homocystein levels didn't show any significant differences between BMI groups.

The clustering of hypertension, hyperglycaemia and gout was first recognised in 1920 [19]. In 2004, The National Cholesterol Education Program's Adult Treatment Panel III (ATPIII) defined the metabolic syndrom, which were furter modified by the International Diabetes Federation (IDF) in 2005. According to ATP III, patients having at least 3 of 5 characteristics – abdominal obesity, elevated triglycerides, decreased HDL-cholesterol, increased blood pressure or increased fasting plasma glucose – can be diagnosed as having the metabolic syndrom [20]. IDF declares similar criteria fairly consistent with ATP III [21]. Slight differences include central obesity as the major feature and the fulfillment of two of the other four manifestations. The borderline glucose concentration is lower in IDF criteria (100 mg/dl), with a strong recommendation for oral glucose tolerance test (OGTT) when exceed. The waist circumference criteria are more strict by IDF (≥80 cm for Europid women) and different for different ethnic groups.

In the course of our study, the average waist circumference in overweight and obese groups; and the HDL-C values of obese group counterbalances the diagnostic criteria of the IDF metabolic syndrome. Fasting glucose and triglyceride levels of any individual in the groups didn't counterbalance neither ATP III nor IDF diagnostic criteria. However, fasting insulin levels and HOMA-IR indexes showed significant differences in the groups, and the highest level found in obese group (Table 1).

Insulin resistance (IR) is a common pathologic state in which target cells fail to respond to ordinary levels of circulating insulin. It results in inability of insulin to provide normal glucose and lipid homeostasis [11]. Hence, higher than normal concentrations of insulin are needed in order to maintain normoglycaemia.

Fasting plasma insulin concentrations and HOMA index are usually applied to evaluate IR. Degree of insulin resistance (IR) is determined according to the HOMA-IR formula [11,22].

In cases when HOMA-IR >4.0, then the patient is diagnosed as having insulin resistance. The addition of pharmacological agents (metformine, roziglitazone) to diet and exercise in treatment is considered to be fairly efficient method in overcoming insulin resistance, obtaining weight loss and reducing metabolic risks [22-25].

The presence of insulin resistance in obese group is considered since their mean HOMA index is 4.55±2.42. in our study.

Visceral obesity is thought to be the main factor causing IR. Visceral obesity predisposes to the development of hypertension, type 2 DM, cardiovascular disease and certain types of cancer [11,12]. Increased risk exist already in the overweight group. Besides total body fat mass, distribution of fat predisposes to eventual metabolic complications. Visceral and subcutan adipose tissue differ in their endocrine activities. Visceral adipose tissue (VAT) secretes its product into the portal circulation which brings released free faty acid (FFA) directly to the liver where they promote gluconeogenesis, VLDL synthesis, decrease glucose uptake and couse overall IR.

In our study, the percentage of body fat in overweight group (33.35%±5.85) was appropriate to obese group according to the reference values that we got from another study [26]. In addition, according to IDF diagnostic criteria, central obesity is considered since



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the average value of waist circumference is ≥80 cm. On the other hand, total levelsof total and LDL cholesterol were slightly high in this group (Table 1). This shows that although a person is in normal or overweight ranges according to BMI, he metabolically might have a tendency to the risk factors related to obesity. Therefore, we think that it is useful to make a scan of global cardio-metabolic risks not only for obese individuals according to BMI, but also for normal or overweight individuals.

Substantial evidence exists that obesity, as a clinical condition, is very heterogeneous and that the regional distribution of body fat, especially the abdominal visceral adipose tissue (VAT) accumulation, is a key feature of the form of overweight/obesity frequently associated with insulin resistance and type 2 diabetes [27-31].

Although waist circumference is a better marker of abdominal fat accumulation than the body mass index, an elevated waistline alone is not sufficient to diagnose visceral obesity and related risk factors.

In order to predict cardio-metabolic risks associated with obesity, it is beneficial to know the ratio of body fat as well as some biochemical parameters (primarily glucose, insulin, triglyceride, HDL and hsCRP values) in addition to BMI and waist circumference values.

In our study BMI, as well as waist circumference, showed significant positive relationship with the percentage of body fat, fasting blood glucose, insulin, triglyceride, uric acid, fibrinogen and HOMA-IR levels, on the contrary showed significant negative relationship with HDL-C (Table 2). So, we thought that to predict the cardiometabolic risks related to obesity it would be useful to measure body fat ratio and some biochemical parameters (mainly fasting blood glucose, insulin, triglyceride, HDL-C and CRP levels) along with BMI and waist circumference measurements.

In order to predict the increase of cardio-metabolic risk, we made multiple regression analysis to find out whether the BMI or the waist circumferences were more sensitive to this risk. In the analysis we proved that the waist circumference is the best indicator for the levels of HDL, triglyceride and insulin (Table 3). In the multiple regression analysis, we found that the percentage of body fat has significance on determining the HDL, while it has no significance in determining the level of triglyceride and insulin. By these findings, it is proved that the estimation of accumulated visceral fat is more helpful in determining cardiovascular disease and type II diabetes risks than estimating the total body fat.

Unlike previous studies [13,16,17] which depended on measuring waist circumference and levels of plasma triglyceride in predicting the cardio-metabolic risks that associated with visceral adiposity, in our study we highlighted the importance of measuring insulin, fibrinogen and homocystein levels in addition to measurements of waist circumference and levels of plasma triglyceride for the first time.

Epidemiologic studies demonstrated that obesity is associated with the increase in the blood levels of prothrombotic proinflammatory factors and endothelial dysfunction indicators such as fibrinogen, C-reactive protein (CRP) and homocystein etc. In such a study [24], body fat and fibrinogen were found to be positively correlated whereas plasma leptin concentration was shown to be correlated with CRP. It was concluded that the increase in body fat ratio which is also affected by leptin, and the impairment in insulin sensitivity is associated with the increase in cardiovascular risk factors.

In the course of our study, no significant differences were found between groups with respect to CRP, fibrinogen and HOM levels; but there were significant positive correlations between BMI and fibrinogen and HOM levels (Table 2). In addition, there were increases in the levels of insulin, HOMA-IR, triglyceride and uric acid in line with the increase in BMI and waist circumference. These findings were compatible with the findings of previous studies concerning the increase of the metabolic risk factors by increasing of VAT. It was concluded that the increase in visceral adipose depot may easily trigger the global risk being defined as cardiometabolic risk.

Recently, particular attention has focused on the presence of a longitudinal link between middle-age adiposity [32,33] and later-life obesity [34,35] and the decline in cognitive function. Thus, obesity seems responsible for further health implications beside cardiovascular disorders (CVD), and the presence of a vascular burden is recently recognised as a possible common cofactor in the development of dementia [33].

Insulin sensitivity plays an important role on the effect of glucose on memory [8]. Although increasing plasma insulin levels by keeping the blood glucose levels within normal limits in healthy adults and AD patients can improve the memory, increasing plasma glucose by repressing endogenous insulin secretion fails to have the same effect. This serves to show that a sufficient amount of glucose and insulin is required to improve memory [8,23]. Although insulin may be necessary for normal cognitive functions, impairments in insulin secretion may accelerate cognitive degeneration just like in AD. For this reason, treating insulin resistance could reduce or postpone the risk of AD. In our study, the significant increase in the fasting blood glucose and insulin levels with the increase in BMI and the positive significant correlation of the BMI and circumference of waist with the blood sugar and insulin levels lead



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us to believe that obesity could contribute to cognitive degeneration by means of insulin resistance in older age.

Although BMI is widely accepted as a simple marker of adiposity in population based studies, and recognized as an instrument to diagnose obesity for all age groups (BMI $\geq$ 30 kg/m$^2$). It should be more properly seen as an index of weight excess, rather than body fatness. Regardless of BMI value, patients with increased intraabdominal fat usually have an atherogenic lipid profile, high fasting serum glucose, insulin levels and high blood pressure, all metabolic factors participating in atherosclerotic process. These factors subsequently contribute to the occurrence of CHD, stroke, as well as peripheral vascular diseases [36,37].

Recent studies showed that intra-abdominal fat accumulation, rather than BMI, is a significant independent predictor of the IR, and dyslipidemia seen in both metabolic syndrome and diabetes [14,36,38].

According to the findings of our study and the results of previous studies, we suggested that measuring the waist circumference only or measuring waist circumference plus the level of triglyceride are not enough to predict the short term and long term risks factors associated with obesity.

So, we propose a "global cardiometabolic risk assessment package" consisting of the measurements of BMI, waist circumference, total body fat percent, fasting blood glucose, insulin, triglyceride, HDL-C, CRP, fibrinogen and HOM levels.

Today, the gradual increase in long life expectancy causes a rapid increase in the ratio of older population. The quality of life in old ages is closely related with the level of health in earlier periods of life. Health issues, such as uncontrollable obesity, impaired glucose tolerance, insulin resistance, dislipidemia, high plasma homocystein levels, hypertension and estrogen deficiency are considered to be risk factors, could have negative effects not only from a physiological viewpoint but also on the cognitive functions and in the mental conditions as getting older [2,7]. It is reported that Mediterranean type nutrition, regular exercise and other healthy life recommendations decrease not only the risk of dementia but also the coronary artery and cancer risks. It is also indicated, activities keep the conscious mind alert, are more protective against dementia rather than physical activity. This could be attained by means of training at a younger age [7].

## 5. Conclusions

Obesity has emerged as a major health problem because of our largely sedentary lifestyle and unlimited access to nutrient-rich, high-calorie foods. In turn, obesity, particularly abdominal obesity associated with an excess deposition of VAT, is strongly linked to the development of the metabolic syndrome, type 2 diabetes, and related cardiovascular disease. However, waist circumference provides a crude assessment of excess VAT particularly when the elevated waistline is accompanied by hypertriglyceridemia and low HDL cholesterol levels.

Increase in the body weight above normal levels causes a significant increase in not only body fat, but also blood sugar, insulin, TG, uric acid and prothrombotic proinflammatory factors levels. Obesity and the increase in body fat tissue increases susceptibility against chronic diseases including the Alzheimer disease and dementia. As a remarkable point, many of the vascular (hypertension, atherosclerosis), inflammatory (adipocytokine-cytokine) and metabolic (insulin resistance, fasting hyperglycemia) factors contributing to the development of cognitive decline seem strongly related to body fat distribution, particularly the VAT, rather than BMI. For this reason, it is very important to make optimal biochemical and anthropometric parameters in predicting the global risks associated with obesity. As many normal or overweight individuals may have metabolic disorders, we strongly recommend measuring body fat, VAT measures and blood levels of glucose, insulin, triglyceride, HDL-C, fibrinogen, CRP and HOM in patients who have complaints about their weight. Studying the parameters and especially insulin resistance can give us insight not only into the cardio-metabolic risk but also into the demans risk in the future.

Consequently, obesity cause many health threatening disorders and effects the quality of life in the short and long term. It could be prevented or cured while getting older by keeping negative environmental conditions under control.



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## References


[1] Wadden TA, Stunkard AJ (eds). Translators: Saçıkara A, Yılmaz A.M: Obesity treatment handbook. And Publishing Ltd. Sti. Vol. I. 2003. (Book in Turkish)

[2] Werrij MQ, Mulkens S, Hospers HJ, Jansen A. Overweight and obesity: The significance of a depressed mood. Patient Education and Counselling 2006 Jul; 62(1):126-31

[3] Nicolls MR. The clinical and biological relationship between Type II diabetes mellitus and Alzheimer's disease. Curr Alzheimer Res. 2004; 1(1):47-54

[4] Monte SM, wands JR. Review of insulid and insulin like growth factor expression, signaling and malfunction in the central nervous system:relevance to alzheimer's disease. J Alzheimers Dis. 2005; 7(1):45-61

[5] Yaffe K, Kanaya A, Lindquist K, Simonsick EM, et al. The metabolic syndrom, inflammation and the risk of cognitive decline. JAMA 2004; 292 (18):2237-42

[6] Luchsinger JA, Mayeux R. Cardiovascular risk factors and Alzheirmer's disease. Curr Atheroscler Rep. 2004;6 (4):261-6

[7] Kornhuber HH. Prevention of dementia. Gesundheitswesen 2004;66 (5):346-51

[8] Watson GS, Craft S. Modulation of memory by insulin and glucose neuropsychological observation in Alzheimer's disease. Eur J Pharmacol. 2004, 19;490 (1-3):97-113

[9] Rasgon N, Jarvik L. Insulin resistance, affective disorders, and Alzheimer's disease: review and hypothesis. J Gerontol A Biol Sci Med Sci 2004;59 (2):178-83

[10] Grossman H. Does diabetes protect or provoke Alzheimer's disease? Insight into the pathobiology and future treatment of Alzheimer's disease. CNS Spectr. 2003; 8 (11):815-23

[11] Mlinar B, Marc J, Janez A, Pfeifer M. Molecular mechanisms of insulin resistance and associated diseases. Clin Chim Acta 2007; 375: 20-35

[12] Fonseca VA. The metabolic syndrome, hyperlipidemia, and insulin resistance. Clinical Cornerstone 2005; 7(2/3): 61-72

[13] Despres JP, Lemieux I, Bergeron J, Pibarot P, Mathieu P, Larose E, et al. Abdominal obesity and the metabolic syndrome: contribution to global cardiometabolic risk. Arterioscler Thromb Vasc Biol 2008 Jun; 28(6): 1039-49

[14] Ness-Abramof R, Apovian CM. Waist circumference measurements in clinical practice. Nutr Clin Pract 2008 Aug-Sept; 23(4): 397-404

[15] Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA 2001; 285: 2486-97

[16] Despres JP, Cartier A, Cote M, Arsenault BJ. The cocept of cardiometabolic risk: Bridging the fields of diabetology and cardiology. Ann Med 2008 Apr; 10: 1-10

[17] Depres JP. Cardiovascular disease under the influence of excess visceral fat. Crit Pathw Cardiol 2007 Jun; 6(2): 51-9

[18] Wildman RP, Muntner P, Reynolds K, McGinn AP, Rajpathak S. Et al: The obese without cardiometabolic risk factor clustering and the normal weight with cardiometabolic risk factor clustering. Arch Intern Med 2008; 168(15): 1617-24

[19] Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet 2005; 365: 1415-28

[20] Grundy SM, Brewer Jr HB, Cleeman JI, Smith Jr SC, Lenfant C. Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related definition. Circulation 2004; 109; 433-8

[21] Alberti KGMM, Zimmet P, Shaw J. Metabolic syndrome-a new worldwide definition: A Consensus Statement from the International Diabetes Federation. Diabet Med 2006; 23: 469-80

[22] Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacker DF, Tumer RC. Homeostasis model assesment: insulin resistance and B-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologica 1985; 28:412-9

[23] Watson GS, Craft S. The role of insulin resistance in the pathogenesis of Alzheimer's disease: implications for treatment. CNS Drugs. 2003; 17 (1):27-45

[24] Gomez-Ambrosi J, Salvador J, Paramo JA, et al. Involvement of leptin in the association between percentage of body fat and cardiovascular risk factors. Clin Biochem 2002; 35(4):315-20

[25] Groop L, Orho-Melander M. The dysmetabolic syndrom. Journal of Internal Medicine 2001; 250:105-20.

[26] Ozenoglu A, Can G, Hatemi H. Body composition referance values of adult women grouped as normal-weight, overweight, obese and morbid obese according to body mass index. Endocrinologic Invest 2001; 10 (2): 58-63 (Journal in Turkish)




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[27] Després JP. Is visceral obesity the cause of the metabolic syndrome? Ann Med 2006;38:52–63
[28] Björntorp P. Metabolic implications of body fat distribution. Diabetes Care. 1991;14:1132–1143
[29] Després JP, Moorjani S, Lupien PJ, et al. Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. Arteriosclerosis. 1990;10:497–511
[30] Kissebah AH, Krakower GR. Regional adiposity and morbidity. Physiol Rev. 1994;74:761–811
[31] Lebovitz HE, Banerji MA. Point: visceral adiposity is causally related to insulin resistance. Diabetes Care. 2005;28:2322–2325
[32] Grospe EC, Dave JK. The risk of dementia with increased body mass index: a systematic review. Age Aging 2007; 36: 23-9
[33] Gustafson D. Adiposity indices and dementia. Lancet Neurol 2006; 5: 713-20
[34] Razay G, Vreugdenhil A, Wilcock G. Obesity, abdominal obesity and Alzheimer disease. Dement Geriatr Cogn Disord 2006; 22: 173-6
[35] Luchsinger JA, Patel B, Tang MX, Schupf N, Mayeux R. Measures of adiposity and dementia risk in elderly persons. Arch Neurol 2007; 64:392-8
[36] Despres JP, Lemieux I. Abdominal obesity and metabolic syndrome. Nature 2006; 444: 881-7
[37] Scheneider HJ, Glaesmer H, Klotsche J, et al. Accuracy of anthropometric indicators of obesity to predict cardiovascular risk. J Clin Endocrinol Metab 2007; 92: 589-94
[38] Cereda E, Sansone V, Meola G, Malavazos AE. Increased visceral adipose tissue rather than BMI as a risk factor for dementia. Age and Aging 2007; 36: 488-91



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