

The effect of body composition on blood lipids, leptin, bone mineral density, and nutrition in females

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ABSTRACT

الأهداف: دراسة أثر تركيبة الجسم على دهون الدم، اللبتين، كثافة العظام، والتغذية لدى الإناث.

الطريقة: تم تحديد وأخذ قياسات 131 طالبة في السنة الرابعة بجامعة غازي - كلية التعليم المهني - أنقرة - تركيا، خلال عام 2006م، من أجل تركيبة أجسامهن، قياساتهن البشرية، دهون الدم، مستويات اللبتين، وكثافة العظام. تم استخدام طريقة البقاء 24 ساعة لتحديد حالة التغذية لديهن.

النتائج: كانت مستويات الكوليسترول الكامل ($p=0.002$)، الدهون الثلاثية ($p=0.00$)، الكوليسترول منخفض الكثافة ($p=0.004$)، الكوليسترول منخفض الكثافة للغاية ($p=0.000$)، الكوليسترول الكامل / الكوليسترول عالي الكثافة ($p=0.000$)، الكوليسترول منخفض الكثافة / الكوليسترول عالي الكثافة ($p=0.000$)، اللبتين ($p=0.000$) كانت أكثر انخفاضا بين النحيفات من المعتدلات والبدينات، بينما وجد أن مستوى الكوليسترول عالي الكثافة أعلى ($p=0.277$). كانت كثافة العظم لدى البدينات أعلى من النحيفات ($p<0.001$). تم تحديد صلة ملحوظة بين تركيبة الجسم، دهون الدم، اللبتين، وكثافة العظام ($p<0.001$).

خاتمة: وفقاً للنتائج، البقاء على الوزن المطلوب لديه أثر إيجابي على دهون الدم، اللبتين، وكتلة العظام. المزيد من الدراسات مطلوبة من أجل فهم أكثر لهذه العلاقة.

Objective: To study the effect of body composition on blood lipids, leptin, bone mineral density, and nutrition in females.

Methods: One hundred and thirty-one fourth-year females students studying at the Faculty of Vocational Education, Gazi University, Ankara, Turkey, during the year 2006, were determined for their body composition. Their anthropometric measurements, blood lipids, leptin levels, and bone mineral densities were measured. Moreover, 24-hour reminder method was used to determine their state of nutrition.

Results: The total cholesterol ($p=0.002$), triglyceride ($p=0.00$), low-density lipoprotein (LDL) cholesterol ($p=0.004$), very LDL cholesterol ($p=0.000$), total cholesterol/high-density lipoprotein (HDL) cholesterol ($p=0.000$), LDL cholesterol/HDL cholesterol ($p=0.000$) and leptin ($p=0.000$) levels were found to be lower among the thin than the normal-weight and the overweight, whereas the HDL cholesterol ($p=0.277$) level was found higher. The bone mineral density of the overweight participants were higher than the thin ($p<0.001$). Significant correlations were determined between the body composition and blood lipids, leptin, and bone mineral density ($p<0.001$).

Conclusion: According to the results, being at the required weight has a positive effect on blood lipids, leptin, and bone mass. To better understand this relationship, further studies are needed.

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Obesity is an increasingly important public health problem not only in developed countries but also in developing countries. If not treated, obesity may cause diabetes mellitus, insulin resistance, hyperlipidemia, hypertension, arteriosclerosis, and other diseases involving vein complications.^{1,2} Leptin is a body weight organizing protein that is responsible for controlling and organizing the body fat amount, and controls the homeostasis of energy consumption in humans.³⁻⁵ Leptin provides the brain with information on the increase in body fat percentage and influences the behavior, metabolism, endocrine physiology, and

nutritional state of the organism. An increase in body fat leads to an increase in leptin concentration, and thus, a decrease in appetite, which results in increased energy consumption. The opposite happens when the fat tissue decreases. In the case of a lack of leptin, food consumption is not hindered so obesity occurs due to decreased energy consumption.^{5,6} In short, it is suggested that leptin regulates body weight by decreasing food intake and increasing energy consumption. Studies indicate that there is a high correlation between body composition and serum leptin concentrations, and leptin concentration in females is twice as high as that in males. This is because the leptin blood level of females is high as their fat rate and fat distribution are different.^{7,8} It was determined that the level of serum leptin increases with increased level of daily energy consumption and fat amount⁹ and one day of hunger decreases the leptin level by 30%, while excessive food intake increases the leptin level by 50% in 12 hours.¹⁰ Several studies establish that leptin has a positive effect on body fat mass, bone density, and bone formation.¹¹ Yet, other studies conclude that leptin affects bone formation adversely,¹² or it does not have any function on the tissue.¹³ This study was conducted to study the relation between body composition and serum leptin, blood lipids, bone mineral density (BMD), and nutrition.

Methods. The study was carried out among 131 fourth-year female students studying at Vocational Education Faculty, Gazi University, Ankara, Turkey, during the year 2006. None of the participants used any medicine, vitamins, mineral supplements, or oral contraceptives. None of the females were pregnant or breast-feeding, nor had they a metabolic disease. The participants were informed on the subject, purpose, and rules of the research. Each participant signed a voluntary participation form before approval from the University Ethics Committee.

Anthropometric measurement. Height was measured to the nearest 0.1 cm, and weight to the nearest 0.5 kg in light clothing and without shoes. Body mass index (BMI) was calculated as weight (kg)/height (m²). All anthropometric measurements were taken by trained dietitians. Since reference data on BMI for the Turkish population are not available therefore, World Health Organization reference data were used for estimating obesity.^{14,15} Females were classified according to their BMI into groups as under weight (BMI<18.5 kg/m²), normal weight (BMI:18.5-24.9 kg/m²), and over weight (BMI>25.0 kg/m²).¹⁴ The amount and distribution of body fat were assessed by measuring the thickness of subcutaneous adipose tissue with

Lange skinfold caliper. The skinfold thickness (SFT) was measured on the left side of the body at 4 sites: biceps, triceps, subscapular and suprailiac thickness. The sum of the 4 SFT measurements was considered an indicator of total subcutaneous fat and the sum of trunk SFT as an index of central obesity. The waist hip ratio (WHR) was used to assess body fat distribution and specifically as indicators of intra abdominal or visceral fat deposition.¹⁵ Measurements were taken of the mid upper arm circumference (MUAC) on the bare arms of the females, who stood in a straight position with left arm bent at a 90° angle. The distance between acromion and olecranon was measured with a tape measure and marked at the middle point. Later a measurement was made with a non-stretch measuring tape around the circumference at the mark with arms at sides and inner palms to the femur. Waist and hip measurements of the participants were made with the arms at sides, feet next to each other and legs balanced equally.^{14,15}

Biochemical analysis. Early morning venous blood samples were obtained from each participant for biochemical screening tests, following a 12-hour overnight fast. Professional staff performed venipuncture, using Vacutainers to obtain 15 mL of whole blood. Blood was centrifuged for plasma separation at the local Ankara Government Hospital where the actual biochemical analyses were made. Roche Diagnostic Kits were used for triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), very low density lipoprotein cholesterol (VLDL-C), and total cholesterol (TC) analysis, and Modular D+P (Roche Diagnostics, GmbH Monnherin, Germany) was used for analysis. The LDL-C was calculated by the Friedewald et al's¹⁶ formula:

$$LDL = \text{Total cholesterol} - (\text{HDL cholesterol} + (\text{TG})/5)$$

The level of serum leptin was analyzed by means of a Biosource Leptin Easia Kit, BioSource Europe S. A., rue de l'Industrie 8 B-1400 Nivelles Belgium (Catalogue no: KAP2281) and the BMD was measured by dual energy x-ray.

Nutritional assessment. We determined each subject's food intake with 24-hour records. We estimated the volume and portion size picture booklet, which included 120 photographs of food, each with 3-5 different portion sizes. Two dietitians assisted with the dietary recall and reviewed all questionnaires with the subjects, probing for inaccurate and omitted responses. The average energy, protein, carbohydrate, lipid, thiamine, riboflavine, and niacin content for each individual's diet were analyzed using food composition tables for preparing Turkish foods.

Statistical analysis. The data were analyzed using the Statistical Package for Social Sciences software for Windows 10.0 (SPSS Inc, Chicago, IL, USA). When evaluating the demographic properties of the participants, numbers, and percentages were taken. When determining the blood lipids according to BMI, BMD, anthropometric measures, and the daily energy consumption of nutrition elements, arithmetical averages, and standard deviation values were taken. To evaluate the relation between them, one way analysis of variance (ANOVA) test and Pearson coefficient correlations were used. In all analyses, 5% significance level was used.

Results. The research was carried out among 131 females between the ages of 19 and 23, and the average age was 20.64 ± 1.17 years. Forty (30.5%) of the participants were underweight, 56 of them (42.8%) were normal-weight and 35 of them (26.7%) were overweight. The BMI average was 22.95 ± 2.81 kg/m² (15.6-31.8 kg/m²). All of the anthropometric measure values were found to be significant for thin, normal-weight and overweight participants as expected according to the BMI (Table 1). The TC, TG, LDL-C, VLDL-C, TC/HDL-C, LDL-C/HDL-C, and leptin levels were found to be lower among the underweight than the normal and the overweight, whereas the HDL-C level was found higher (Table 2). The BMD of the overweight participants were higher than the underweight, and the difference between the groups was significant ($p=0.000$) (Table 3). Although the daily energy and nutrients intake of the overweight females were higher than the underweight and the normal weight participants, the difference was not statistically significant ($p>0.05$) (Table 4). A positive correlation was found between the BMI and biochemical findings ($p<0.001$); namely, between TG ($r=0.350$, $p<0.001$), TC ($r=0.232$, $p<0.05$), VLDL-C ($r=0.351$, $p<0.001$), TC/HDL-C ($r=0.390$,

$p<0.001$), LDL-C/HDL C ($r=0.346$, $p<0.001$), leptin ($r=0.416$, $p<0.001$) and BMD (Table 5). A positive correlation was also found between WHR, which is an indication of obesity, and all blood lipids, except for total cholesterol and leptin, BMD, and the daily intake of energy, protein, fats and fibre ($p<0.001$). The similar correlation between MUAC and HDL-C is in the negative direction ($r=-0.380$, $p<0.001$) and with other parameters, it is in the positive direction ($p<0.001$). The body fat mass and HDL-C are in the positive direction in all parameters except for VLDL-C and energy and nutrients intake ($p<0.001$) (Table 5).

Discussion. Obesity is an important health problem, which is considered a disease in itself and leptin is a hormone that controls body weight by arranging the energy balance. Leptin informs the brain on body fat deposits.¹⁷ It decreases food intake via hypothalamic receptors and increases the metabolic

Table 1 - Anthropometric measurements of participants.

Anthropometric measurements	Underweight	Normal weight	Overweight
	Mean±SD		
MUAC (cm)	22.83±8.84	26.59±2.40	30.86±2.01
Triceps SFT (mm)	9.85±4.07	14.00±6.64	16.97±6.14
FM (%)	22.83±4.76	26.00±7.19	31.42±3.89
FFM (%)	77.17±4.76	73.99±7.19	68.57±3.89
Waist circumference (cm)	66.20±4.84	75.70±4.90	88.46±7.01
Hip circumference (cm)	89.60±2.67	97.46±4.82	107.20±6.57
WHR	0.73±0.04	0.77±0.03	0.82±0.04
Height (cm)	165.00±6.83	170.32±8.73	171.86±8.86
Weight (kg)	50.26±6.42	65.30±8.51	82.10±13.33
BMI (kg/m ²)	18.34±1.26	22.44±1.75	27.67±2.80

MUAC - mid upper arm circumference, SFT - skinfold thickness, BMI - body mass index, WHR - waist hip ratio, FM - fat mass, FFM - fat free mass

Table 2 - The results of main biochemical tests in groups.

Biochemical tests	Underweight	Normal weight	Overweight	F	P-value
	Mean±SD				
TG (mg/dL)	70.06±26.17	93.18±42.20	144.80±79.21	11.241	0.000†
TC (mg/dL)	151.13±23.43	161.52±30.11	185.46±42.61	6.375	0.002*
HDL-C (mg/dL)	58.74±11.56	55.79±13.86	52.34±8.92	1.299	0.277
LDL-C (mg/dL)	76.80±13.91	87.11±26.51	104.13±36.22	5.780	0.004*
VLDL-C (mg/dL)	15.60±5.13	18.59±8.49	29.00±15.88	11.287	0.000†
TC/HDL-C	2.61±0.37	3.02±0.74	3.71±1.23	10.846	0.000†
LDL-C/HDL-C	1.33±0.29	1.65±0.60	2.10±0.92	8.651	0.000†
Leptin (ng/mL)	2.76±4.27	7.50±7.20	10.85±6.99	8.922	0.000†

* $p<0.05$ and † $p<0.001$. TC - total cholesterol, HDL-C - high density lipoprotein cholesterol, LDL-C - low density lipoprotein cholesterol, VLDL-C - very low density lipoprotein cholesterol, TG - triglycerides

Table 3 - The bone mineral density according to BMI in the participants.

BMD (g/cm ²)	Underweight	Normal weight	Overweight	F	P value
		Mean±SD			
L1	0.91±0.12	1.17±0.11	1.18±0.07	31.428	0.000**
L2	0.98±0.13	1.25±0.14	1.26±0.06	21.923	0.000†
L3	1.05±0.12	1.30±0.14	1.34±0.03	22.072	0.000†
L4	1.05±0.13	1.26±0.14	1.27±0.03	14.300	0.000†

**p*<0.05 and †*p*<0.001. L - lumbar, BMD - bone mineral density, BMI - body mass index

Table 4 - The daily energy and nutrients intake according to body mass index in the participants.

Daily energy and Nutrients intake	Underweight	Normal weight	Overweight	F	P value
		Mean±SD			
Energy (kcal)	1562±616.8	1922±971.6	2027±749.6	2.208	0.114
Protein (g)	61.7±26.0	71.8±40.0	80.0±28.3	1.451	0.239
Fat (g)	54.9±16.2	64.6±38.3	74.4±48.7	1.594	0.207
Thiamine (mg)	0.68±0.30	0.88±0.43	0.80±0.13	2.795	0.065
Riboflavine (mg)	1.00±0.43	1.15±0.62	1.22±0.44	0.987	0.376
Niacin (mg)	9.13±4.54	1.15±0.62	1.22±0.44	1.212	0.301
Saturated fat (g)	18.82±5.93	21.83±15.26	24.37±13.98	0.960	0.386
Monounsaturated fat (g)	18.09±5.61	20.63±11.70	25.31±14.55	2.176	0.118
Polyunsaturated fat (g)	14.11±4.76	17.50±11.18	19.69±18.05	1.530	0.221

Table 5 - The relationship between anthropometric measurement of blood lipid, serum leptin, bone mineral density, and daily energy-nutrients intakes (r).

Anthropometric measurement	Pearson correlation coefficient (r)				
	BMI	WHR	MUAC	FM	FFM
<i>Biochemical tests</i>					
TG	0.350†	0.447**	0.319†	0.274†	0.296†
TC	0.232†	0.147	0.165	0.249†	-0.039
HDL-C	-0.167	-0.412†	-0.380†	0.061	0.457†
LDL-C	0.230	0.212†	0.266†	0.169	0.065
VLDL-C	0.351†	0.452†	0.319†	0.277†	0.294†
TC/HDL-C	0.390†	0.497†	0.401†	0.236†	0.420†
LDL-C/HDL-C	0.346†	0.429†	0.397†	0.185†	0.346†
Leptin	0.416†	0.165	0.305†	0.263†	0.236†
<i>Bone mineral density</i>					
L1	0.746†	0.557†	0.420†	0.543†	0.588†
L2	0.711†	0.475†	0.402†	0.543†	0.480†
L3	0.689†	0.451†	0.395†	0.486†	0.507†
L4	0.623†	0.357†	0.389†	0.415†	0.469†
<i>Daily energy and nutrients intakes</i>					
Energy	0.163	0.357†	0.337†	-0.002	0.366†
Protein	0.165	0.350†	0.292†	0.033	0.313†
Fat	0.164	0.268†	0.343	0.042	0.267
Saturated fat	0.131	0.250†	0.164	0.129	0.148
Monounsaturated fat	0.171	0.294†	0.259†	0.134	0.228†
Polyunsaturated fat	0.167	0.207†	0.219†	0.093	0.77
Cholesterol	0.042	0.207†	0.164	0.062	0.078
Fiber	0.154	0.251†	0.187†	0.045	0.164

**p*<0.05 and †*p*<0.001. BMI - body mass index, WHR - waist hip ratio, MUAC - mid upper arm circumference, FM - fat mass, FFM - fat free mass, TG - triglycerides, TC - total cholesterol, HDL-C - high density lipoprotein cholesterol, LDL-C - low density lipoprotein cholesterol, VLDL-C - very low density lipoprotein cholesterol, L - lumbar

rate.¹⁸ However, the serum leptin levels are evidently higher in the obese compared to the normal-weight people. Leptin is expressed exclusively in adipose tissue, and the serum leptin levels correlate well, and to a close degree, with the percentage of body fat and BMI. It is stated that the best determinant of the leptin level is the BMI and the waist circumference.¹⁹ A very strong correlation between the leptin level and body fat was determined in the studies conducted. Studies have shown that obese individuals have higher levels of circulating leptin compared to lean subjects. This elevation is mainly caused by a receptor resistance to the effects of leptin in obesity.^{20,21,22} This result is an important finding in that it shows that fat mass has a determining role on leptin secretion, the BMI and the percentage of body fat. The leptin level was found to be higher in the obese than the normal-weight (Table 2). The results support our findings in this research. In theory, it is expected that the level of the leptin hormone, which decreases the appetite, and increases energy consumption, is low in the obese. This may result from an insensitivity developed against leptin in hypothalamic receptors in the obese.¹⁹ Clement²³ stated that a mutation occurring in the leptin and its receptors rarely caused obesity and that this was not a cause of obesity for all the obese population. Hekimoglu²⁴ established that obesity is only caused by the lack of leptin and that leptin was not effective in the obese due to the resistance developed against it. It was further determined that more leptin was required to defeat the resistance against leptin, and so more leptin was secreted from the fat tissues and the secretion of more leptin caused the fat tissue that produces it to increase. The insulin level was found to be higher in overweight females with large waist circumferences. It was established that hyperleptinemia may be caused by hyperinsulinemia. Ruhl and Everhart²⁵ determined a positive correlation between waist/hip proportions, BMI, SFT, sum of the 4 areas' SFT, and triceps SFT, and the serum leptin levels. In another study, however, it was found that there was not a correlation between leptin and the waist/hip proportion, while the hip circumference, and the serum leptin level were related.²⁶ Fernandez-Real et al,²⁷ however, found a very weak positive correlation between the muscle mass and leptin, whereas there was a strong correlation between the upper arm circumference and leptin in individuals with a high amount of fatless mass. This finding has recently brought on the view that leptin is secreted from the muscle mass, even if in small amounts. In other study, an increase in the leptin level is expected with the increase in the body fat mass.²⁸ The results support the findings of this research (Tables 2 & 5).

It is known that there is a positive relation between the BMI and the levels of TG and cholesterol. Sanlier and Yabanci²⁹ determined a meaningful correlation between obesity and blood lipids. In general, obesity is seen together with high TG, TC, LDL cholesterol, VLDL cholesterol, and low HD cholesterol.³⁰ Every 1% decrease in the total and LDL cholesterol reduces the risk of developing coronary heart diseases by 2% in the society. An accumulation of fat in the abdomen, waist, and hip areas increases the levels of blood lipids, LDL/HDL cholesterol and TG.³¹ In a study, a positive relation between the BMI and TG, total lipid, and LDL-C and a negative relation between the BMI and HDL cholesterol were found.³² The results have parallels with our findings (Tables 2 & 3). A close relation was found between leptin, BMI, and HDL cholesterol in the research of Saka,³³ and in others, a relation between total cholesterol, TG, VLDL cholesterol, and leptin were determined.^{34,35} While Gulturk et al³⁶ determined a positive relation between VLDL cholesterol and serum leptin levels, they did not find any relation between HDL cholesterol, LDL cholesterol, total cholesterol, and leptin. They stated that the correlation between leptin and TG was due to the excessive fat mass in the abdomen; on one hand, leptin increased fat mobilization, and fat acid oxidation, and however, it reduced lipid synthesis, increasing the TG level.

Females with low BMI face the risk of developing osteoporosis. Yanik et al³⁷ determined a positive correlation between BMD and BMI. In another study, it was established that the risk of developing osteoporosis was 33% lower for individuals with a BMI of 30 and above compared to those with a normal BMI.³⁸ Other studies have similarities with the results of this study (Tables 3 & 5).^{39,40} Although body composition is important for protection from osteoporosis, keeping the BMI at normal levels is also important for increasing the quality of life. While leptin informs the hypothalamus on the energy deposit in the fat tissue, acting as the adipostat, it causes changes in the appetite, metabolism, and nutrition through the leptin receptors. When the daily consumed energy, proteins, fats, and carbohydrates are excessive, the body composition is affected. In particular, when the excessive energy intake is converted to fat and accumulated especially in the waist and hips, grounds for many diseases such as cardiovascular diseases and diabetes are set forth. In this study, no meaningful relation was found in the daily intake of energy and nutrition element values according to BMI ($p>0.05$). Yet, excessive consumption of fats, protein saturated fat acids, and mono- and polyunsaturated fatty acids is a critical issue for the overweight.

As the research was conducted on a limited number of participants, it will not be right to generalize its results for a large human population. Therefore, it is advisable that the research is further conducted on people in different age groups.

In conclusion, BMI, FM, WHR, blood lipids, leptin, BMD, and nutrition should be evaluated together. Anthropometric measurements, BMD, blood lipids, and serum leptin levels should be routinely controlled in order to reduce the risk of cardiovascular diseases, diabetes mellitus, osteoporosis and so forth in obesity. The positive effects of increasing the bone mass to the highest level, adequate, and balanced nutrition, and regular physical activities on maintaining the body weight should be considered well to increase the quality of life from childhood to old age.

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References

- Ren J. Leptin and hyperleptinemia from friend to foe for cardiovascular function. *J Endocrinol* 2004; 181: 1-10.
- Sanlier N. Biochemical findings among the youth, anthropometrical measurements, body composition, evaluation of nutritional and physical activity status. *Journal of Gazi Educational Faculty* 2005; 25: 47-73.
- Dursun N. Cardiovascular effects of leptin. *Erciyes Medical Journal* 2005; 27: 167-176.
- Hirsch J. The search for new ways to treat obesity. *Proc Natl Acad Sci USA* 2002; 99: 9096-9097.
- O'Rahilly S. Leptin: defining its role in humans by the clinical study of genetic disorders. *Nutr Rev* 2002; 60: S30-S34.
- Barsh GS, Farooqi IS, O'Rahilly S. Genetics of body weight regulation. *Nature* 2004; 404: 644-651.
- Correia ML, Hayne WG. Leptin, obesity, and cardiovascular disease. *Curr Opin Nephrol Hypertens* 2004; 13: 215-223.
- Ruhl CE, Everhart JE, Ding J, Goodpaster BH, Kanaya AM, Simonsick EM, et al. Serum leptin concentrations and body adipose measures in older black and white adults. *Am J Clin Nutr* 2004; 80: 576-583.
- Miyawaki T, Masuzaki H, Ogawa Y, Hosoda K, Nishimura H, Azuma N, et al. Clinical implications of leptin and its potential humoral regulators in long-term low-calorie diet therapy for obese humans. *Eur J Clin Nutr* 2002; 56: 593-600.
- Goumenou AG, Matalliotakis IM, Koumantakis GE, Panidis DK. The role of leptin in fertility. *Eur J Obstet Gynecol Reprod Biol* 2003; 106: 118-124.
- Steppan CM, Crawford DT, Chidsey-Frink KL, Ke H, Swick AG. Leptin is a potent stimulator of bone growth in ob/ob mice. *Regul Pept* 2000; 92: 73-78.
- Sato M, Takeda N, Sarui H, Takami R, Takami K, Hayashi M, et al. Association between serum leptin concentrations and bone mineral density, and biochemical markers of bone turnover in adult men. *J Clin Endocrinol Metab* 2001; 18: 5273-5276.
- Martini G, Valenti R, Giovani S, Franci B, Campagna S, Nuti R. Influence of insulin-like growth factor-1 and leptin on bone mass in healthy postmenopausal women. *Bone* 2001; 28: 113-117.
- WHO Food and Health in Europe a new basis for action. Regional Publications Europe Series No. 96. Copenhagen, Denmark, 2004.
- Heymsfield SB, Lohman TG, Wang ZM, Going SB. Human Body Composition. 2nd ed. Champaign (IL): Human Kinetics Publisher; 2005. p. 536.
- Friedewald WT, Levy RI, Fedickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972; 18: 499-502.
- Frühbeck G, Gómez-Ambrosi J, Salvador J. Leptin-induced lipolysis opposes the tonic inhibition of endogenous adenosine in white adipocytes. *FASEBJ* 2001; 15: 333-340.
- Seufert J, Kieffer TJ, Leech CA, Holz GG, Moritz W, Ricordi C, et al. Leptin suppression of insulin secretion and gene expression in human pancreatic islets: implications for the development of adipogenic diabetes mellitus. *J Clin Endocrinol Metab* 1999; 84: 670-676.
- Ruige JB, Dekker JM, Blum WF, Stehouwer CD, Nijpels G, Mooy J, et al. Leptin and variables of body adiposity, energy balance and insulin resistance in a population based study. *Diabetes Care* 1999; 22: 1097-1104.
- Gultürk S, Imir G. Leptin and neuroendocrine regulation. *Journal of Adnan Menderes University Medical Faculty* 2006; 7: 49-54.
- Crepaldi G, Romanato G, Tonin P, Maggi S. Osteoporosis and body composition. *J Endocrinol Invest* 2007; 30: 42-47.
- El Maghraoui A, Guerbouh AA, Mounach A, Ghozlani I, Nouijai A, Ghazi M, et al. Body mass index and gynecological factors as determinants of bone mass in healthy Moroccan women. *Maturitas* 2007; 56: 375-382.
- Clement K. Leptin and the genetics of obesity. *Acta Paediatr* 1999; 428: 51-57.
- Hekimoglu A. Role of leptin on physiopathologic actions. *Journal of Dicle University Medical Faculty* 2006; 33: 259-267.
- Ruhl EC, Everhart JE. Leptin concentrations in the United States: relations with demographic and anthropometric measures. *Am J Clin Nutr* 2001; 74: 295-301.
- Wauthers M, Mertens I, Considine R. Are leptin levels dependent on body fat distribution in obese men and women. *Eat Weight Disord* 1998; 3: 124-130.
- Fernandez-Real JM, Vayreda M, Casamitjan R, Gonzalez-Huix F, Ricart W. The fat-free mass compartment influences serum leptin in men. *Eur J Endocrinol* 2000; 142: 25-29.
- Akaliin O. The difference between leptin levels according to body mass index in postmenopausal women and the effects of leptin and endothelin on endometrial thickness. Ege University Ph.D. Dissertation, Kayseri, 2006.
- Sanlier N, Yabanci N. Relationship between body mass index, lipids and homocysteine levels in university students. *J Pak Med Assoc* 2007; 57: 491-496.
- Herbert V, Filer LJ Jr. Vitamin B-12. In: Ziegler EE, editor. Present Knowledge in Nutrition. 8th ed. Washington (DC): ILSI Press; 1996.
- Maffei C, Pietrobelli A, Grezzani A, Provera S, Tato L. Waist circumference and cardiovascular risk factors in prepubertal children. *Obes Res J* 2001; 9: 893-899.
- Sarrafzade N, Rafiei M, Boshtam M, Sarraf Zadegan N. Lipids profiles in the Isfahan population an Isfahan cardiovascular disease risk factor survey. *Nutr J Isfahan University* 1999; 36: 766-777.

33. Saka M. Leptin levels in women and influence of factors. Hacettepe University Ph.D. Dissertation, Ankara, 2001.
34. Boden G. Role fatty acids in pathogenesis of insulin resistance and NIDDM. *Diabetes* 1997; 46: 3-10.
35. Clement K, Lohlou N, Ruiz J, Hager J, Bougneres P, Basdevant A, et al. Association of poorly controlled diabetes with low serum leptin in morbid obesity. *Int J Obes Relat Metab Disord* 1997; 21: 556-561.
36. Gültürk S, Özdemir E, Erdal S, Demir T. Correlation between serum leptin levels and blood lipids and body adiposity in the patients with type 2 diabetes mellitus. *Cumhuriyet University Medical Faculty* 2006; 27: 105-112.
37. Yanik B, Atalar H, Kulcu DG, Gokmen D. The effect of body mass index on bone mineral density in postmenopausal women. *From the World of Osteoporosis* 2007; 13: 56-59.
38. Barrera G, Bunout D, Gattas V, De La Maza MP, Leival L, Hirsch S. A high body mass index protects against femoral neck osteoporosis in healthy elderly subjects. *Nutrition* 2004; 20: 769-771.
39. Ostrowska B. The shape of anterior- posterior spinal curvature in post- menopause women with osteoporosis. *Ortop Traumatol Rehabil* 2006; 8: 537-542.
40. Almehed K, Forsblad d'Elia H, Kvist G, Ohlsson C, Carlsten H. Prevalence and risk factors of osteoporosis in female SLE patients- extended report. *Rheumatology (Oxford)* 2007; 46: 1185-1190.

Related topics

Mosavi Jazayeri SM. Loss of body weight and changes of lipid profile. *Saudi Med J* 2007; 28: 156.

Yilmaz A, Arıkan O, Dokmetas S. Effects of statins on bone mineral density. *Saudi Med J* 2006; 27: 1433-1435.

Gulturk S, Cetin A, Erdal S. Association of leptin with insulin resistance, body composition, and lipid parameters in postmenopausal women and men in type 2 diabetes mellitus. *Saudi Med J* 2008; 29: 813-820.

Al-Dabbagh TQ. Dietary intake and nutritional status of Turkish pregnant women during Ramadan. *Saudi Med J* 2006; 27: 1614.