Body Mass Index, Adipose Tissue, and Resting Metabolic Rate Could Be Affected by Age at Onset of Obesity in Overweight and Obese Adult Women

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Abstract

Background: Obesity is introduced as one of the chronic diseases in most of the countries. In fact, 75 to 80% of obese youngsters will remain obese in adulthood. This study as a novel one was conducted to evaluate the possible association between the age at onset of obesity and body composition as well as Resting Metabolic Rate (RMR) in overweight and obese adult women.

Methods: A total of 295 overweight and obese women aged 18-50 years were enrolled in this cross-sectional study. Over the past year, dietary intake and nutritional status were evaluated using a semi-quantitative Food Frequency Questionnaire (FFQ) including a list of 147 food items. An impedance fat analyzer (InBody 720, Korea) was used to obtain the body composition and an indirect calorimeter was applied to assess the RMR. T-test was used to compare the means of two groups at onset of obesity for quantitative variables, while multiple linear regression was used to assess the relationship between the age at onset of obesity with body composition and RMR.

Results: The results of our study showed that subjects in the upper age group at onset of obesity had lower RMR ($p=0.02$) and age ($p \leq 0.001$) than those in the lower age group.

The outcomes showed that an earlier onset of obesity was significantly associated with higher body weight (Beta: -0.11, CI: -0.30 to -0.00, $p=0.005$) and fat mass (Beta: -0.13, CI: -0.23 to -0.01, $p=0.02$). After adjustment for confounders including age, physical activities, and total energy intake, those differences still remained significant. Furthermore, RMR per body weight became significant too (Beta: 0.16, CI: 0.00 to 0.11, $p=0.03$).

Conclusion: The results of the present study demonstrated a direct association between the age at onset of obesity and RMR per body weight, but it was inversely related to body weight and fat mass. Prospective studies are needed to confirm these findings.

Keywords: Body composition, Energy Metabolism, Obesity, Overweight

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Introduction

World Health Organization has declared that obesity is one of the major problems in many developed and developing countries in the last two decades (1). Obesity is a multifactorial chronic disease and its growing prevalence attracted public attention (2,3). Behavioral and lifestyle changes can result in obesity, and various studies have shown that this significantly contributes to cardiovascular disease and the higher prevalence of hypertension, lipid profile disorders, diabetes, cancer, gall bladder, and hormone abnormalities (4). In addition, the excess amount of fat in the abdomen and upper abdomen, known as abdominal obesity, is an independent prognostic factor for cardiovascular diseases and the consequences. The people with increased visceral fat are faced with the risk of premature death and chronic diseases in comparison with individuals who developed fat accumulation elsewhere in their bodies. Moreover, it was shown that women have a higher prevalence of abdominal obesity (4,5).

Obese adults are divided into two main groups by Mullins. Subjects with early onset of obesity (begins in childhood or adolescence) and those with late onset of obesity (after adolescence) (6). The onset of obesity during adolescence was well observed in one-third of obese adults. Obesity in adolescence will logically remain as adolescent obesity. In general, some consequences accompany adolescent obesity. In fact, adolescent obesity would remain in adulthood (75-80%) and young women are at higher risk of such problem (6-8). Studies in adults, on the other hand, have shown that the increase in adipose tissue that results in obesity may be due to the enlargement of existing adipocytes or an increase in the total number of adipocytes or the combination of both processes (9). Knittle and Hirsch have shown that a higher number of adipocytes in obese adults may have started in childhood (10). An animal study has also shown that overeating in rats for the first three weeks of life (before weaning) has sustained effects on both body weight and adipose tissue cells. Therefore, animals that received more food in the first weeks of life than those who received less food had higher body weight and fat cells (11). Moreover, it has been declared that overweight in the first six months of life is associated with an increased incidence of obesity and overweight in childhood and adolescence (9). So the age at onset of obesity can be considered a good predictor of adult obesity. The aim of the present study was to investigate the association between the age at onset of obesity and the Resting Metabolic Rate (RMR) and, the body composition of Iranian overweight and obese women.

Materials and Methods

Study Population

This cross-sectional study was performed on 295 women aged 18-56 years who were referred to health centers in Tehran. Participants were apparently in good and appropriate general health based on general self-questionnaire, which was filled by trained nutritionists. The protocol was approved by ethics committee of Tehran University of Medical Sciences (IR.TUMS.VCR.REC.1395.1597) and a consent form was signed by all participants. Moreover, participants were excluded if they had a history of chronic diseases including hypertension, cardiovascular diseases, diabetes, thyroid disease, cancer, liver or renal dysfunction, or diet related diseases, and weight fluctuations over the past year. Pregnancy, lactation, smoking, use of medications and the oral contraceptive pill or alcohol were also considered as exclusion criteria.

The Age at Onset of Obesity Assessment

The age data at onset of obesity was collected by a questionnaire.

Dietary Assessment

Usual dietary intake was evaluated by using a 147-item semi-quantitative food frequency questionnaire (FFQ) and its reliability and validity were approved in Iran (12). In face-to-face interviews, participants were asked by expert dieticians to report their consumption frequency for each food item over the past year on a daily, weekly, or monthly basis. Household measures were used to change portion sizes to weight in grams. Due to limited data on the nutrient content of some foods and beverages of Iranian Food Composition Tables (FCT), US Department of Agriculture (USDA) FCT was used to compute nutrient and energy content of dietary intake. Nutrients intakes in micro and macro types were calculated using Nutritionist IV software (First Databank Division, the Hearst Corporation, San Bruno, CA, USA, modified for Iranian foods).

Weight was measured to the nearest 0.1*kg* while wearing one layer of clothing and not wearing shoes by a digital scale (SECA, Hamburg, Germany). Height was recorded to the nearest 0.5 *cm* by a wall-mounted stadiometer whereas the shoulders were relaxed and shoes were removed. Body Mass Index (BMI) was calculated based on the following equation "weight/height^2 (kg/m^2)". To acquire demographic characteristics, a questionnaire was given to all participants which contained questions including age, family history of migraine, marital condition, particular diets, education level, chronic disease background, occupation status, and medicine consumption.

International Physical Activity Questionnaire (IPAC) was used to assess Physical Activity (PA) and was shown as metabolic equivalent hours per week (METs h/week) (13). Activity levels were classified into low, moderate, and high levels according to the IPAQ scoring protocol.

Anthropometric Measurements (Body composition analysis)

An impedance fat analyzer (InBody 720, Korea) was utilized to acquire weight, BMI, Total Body Water (TBW), Body Fat Mass (BFM), Fat Mass (FM), Fat-Free Mass (FFM), Body Cell Mass (BCM), body fat percentage (%), Waist to Hip Ratio (WHR), and Visceral Fat Area (VFA) of the subjects following a standardized procedure according to guidelines. In detail, low-level electrical current was sent through the body into the electrodes of hands and feet. Then, the impedance of currents was measured with the aim of evaluating body composition.

There are various methods for the diagnosis of overweight and obesity; one of the most common methods used in epidemiological studies to determine overweight and obesity is Body Mass Index (BMI). The percentage of healthy weight (BMI 18.5-24.9), overweight (BMI 25-29.9) and obesity (BMI≥30) were 47.8, 29.3, and 22.9, respectively.

Resting Metabolic Rate (RMR) Measurement

All samples were measured by a trained and experienced nutritionist using the standard protocol. The RMR was determined using indirect calorimetry based on the device protocol. Indirect calorimetry is a method of measuring RMR based on oxygen and the amount of CO_2 excreted by the body. The amount of inhaling and exhaled breath was transmitted to the device via a filter attached to the mask that completely covers a person's nose and mouth, and sensor. The device was fitted with a system that measured the volume and oxygen concentration of the respiratory and excretory gases of the individual understudy and analyzed the amount of RMR. It directly measured the concentration of oxygen and carbon dioxide that a person breathes through a mask placed on the mouth and nose. Concentrations of CO_2 and O_2 were measured using the ventilated hood and these gas concentrations were then used to determine 24 h RMR using the equation of Weir. Subjects were instructed to 1) fast and drink only water for 12 h before testing, 2) wear comfortable clothing, and 3) report to the lab for testing at 7 a.m. Subjects were also instructed to keep physical activity to a minimum the morning of the test by dressing slowly and not taking a shower before testing. Once subjects reported for testing, the test was explained to them in detail. They were instructed to lie on the bed as still as possible for 60 min. The first 15 min was used as an acclimatization period to the testing environment. After the first 15 min, the ventilated hood was placed on the subject, and another 15 min acclimatization period started. During this second 15 min period, the flow rate in the ventilated hood was adjusted to maintain the fraction of expired CO_2 between 0.5 and 1.0%. The last 30 min of the test comprised the collection period. Twenty-four-hour RMR was calculated from the average of the O_2 and CO_2 concentrations collected during the last 30 min.

Statistical Analysis

Statistical analysis was performed using SPSS v21 software. Subjects whose total daily energy intake was less than or more than 3 standard deviations from the mean energy intake were excluded. General characteristics of the study population in two groups included the age below 18 at onset of obesity and the age above 18 at onset of obesity. Next, independent sample t-test was used for quantitative variables. Linear regression was also used to determine the relationship between the age at onset of obesity and the quantitative variables. A P-value less than 0.05 was defined as the significance level.

Results

The mean age of the subjects was 36.49±8.38 (Mean±standard deviation) years and mean BMI and weight were 31.04±4.31 kg/m^2 and 80.89±12.45 *kg*, respectively, and the mean waist circumference was 5.24±1.23 *cm* (Table 1).

Table 2 shows the quantitative variables measured in this study along with the mean and standard deviation of each group (the age below 18 at onset of obesity and the age above 18 at onset of obesity). Our findings showed that there was a significant difference in age ($p \leq 0.001$) and RMR ($p=0.02$) between age at onset of obesity over and under 18 years. There was also a marginal correlation between the amount of adipose tissue ($p=0.06$).

The association of age at onset of obesity with the

Table 1. Characteristics of the investigating subjects

Variables	Mean±SD	Maximum	Minimum
Age (years)	36.49±8.38	56	18
Weight (*kg*)	80.89±12.45	136.60	59.50
BMI (kg/m^2)	31.04±4.31	49	24
FM (*kg*)	34.04±8.69	74.20	19.40
FFM (*kg*)	46.8±5.64	67.70	35.30
Body fat(٪)	41.53±5.48	54.30	15.00
WC (*cm*)	99.01±10.05	136.00	80.10
WHR(٪)	0.93±0.05	1.08	0.81
Visceral fat (cm^2)	168.30±103.11	1817.00	20.00
RMR (*kcal*)	1574.96±259.1	2480.00	952.00
RMR per weight (*kcal/kg*)	19.59±3.09	32.50	9.30

Mean± SD: mean ± standard deviation; BMI: Body Mass Index; FM: Fat Mass; FFM: Fat Free Mass; WC: Waist circumference; WHR: Waist Hip Ratio: RMR, Resting Metabolic Rate

Table 2. Association of age at onset of obesity with body composition and RMR

Variables	Mean (SD) Less than 18 years	Mean (SD) more than 18 years	p-value
Age (years)	43.13±8.53	39.46±7.20	≤0.001
Weight (*kg*)	82.78±13.29	79.54±11.30	0.37
BMI (kg/m^2)	31.47±4.61	30.79±4.12	0.22
FM (*kg*)	35.45±9.79	33.25±7.87	0.06
FFM (*kg*)	47.39±5.48	46.22±5.44	0.09
Body fat(٪)	42.14±5.88	41.31±5.35	0.24
WC (*cm*)	100.16±10.65	98.21±9.30	0.12
WHR(٪)	0.93±0.05	0.93±0.05	0.58
Visceral fat (cm^2)	168.82±42.30	168.92±129.56	0.99
RMR (*kcal*)	1619.67±274.45	1544.38±248.98	0.02
RMR per weight (*kcal/kg*)	19.59±3.08	19.54±3.03	0.90

Mean± SD: mean ± standard deviation; p-value: p-value result from independent sample t test; BMI: Body Mass Index; FM: Fat Mass; FFM: Fat Free Mass; WC: Waist Circumference; WHR: Waist Hip Ratio: RMR, Resting Metabolic Rate dependent variables is shown in table 3. The findings of this study indicate that without adjusting for variables, there was a significant inverse relationship between age at onset of obesity and weight (Beta: -0.11, CI: -0.30 to 0.00, p:0.005) as well as adipose tissue (Beta: -0.13, CI: -0.23 to -0.01, p:0.02). After adjusting for confounders (Age, physical activity, and energy intake), body weight (Beta: -0.017, CI: -0.41 to -0.02, p:0.02) and adipose tissue (Beta: -0.20, CI: -0.31 to -0.04, p:0.01), this association remained significant and RMR/*kg* body weight was also significant (Beta: 0.16, CI: 0.00 to 0.11, p:0.03). After adjusting for confounders, waist circumference (Beta: -0.14, CI: -0.30 to 0.01, p:0.07) was also close to significance.

Discussion

The present study shows that there is a significant inverse relationship between body composition such as weight, waist circumference, and adipose tissue with age at onset of obesity. The subjects with younger age at onset of obesity experience a higher incidence of obesity and fat mass in adulthood. In addition, our findings suggest that age at onset of obesity has a direct relationship with RMR/*kg* body weight. It means the younger age at onset of obesity is associated with lower RMR/*kg* body weight.

Bauer *et al* in 1929 stated that the birth weight of obese people was often higher than that of non-obese people. Since then, contradictory evidence that obese people are at a high risk for reproductive health has been emerged by Wolf (14). Mossberg studied the birth weight of obese children and presented their data to support these observations (15). Since then, Wolff, Bruch and Heald showed that there is no difference in birth weight among obese and non-obese individuals (14,16,17). In addition, when Wolff divided birth

Table 3. Association of Age at onset of obesity with body composition and RMR among obese and overweight female subjects

Variable	Model	Beta	CI (95%)	p-value
Weight (*kg*)	Model 1	-0.11	-0.30 to -0.00	0.005
	Model 2	-0.17	-0.41 to -0.02	0.02
BMI (*kg/m*^{2})	Model 1	-0.06	-0.08 to 0.02	0.25
	Model 2	-0.20	-0.15 to -0.01	0.01
FM (*kg*)	Model 1	-0.13	-0.23 to -0.01	0.02
	Model 2	-0.20	-0.31 to -0.04	0.01
FFM (*kg*)	Model 1	-0.04	-0.09 to 0.04	0.42
	Model 2	-0.10	-0.15 to 0.03	0.20
Body fat (%)	Model 1	-0.10	-0.13 to 0.00	0.08
	Model 2	-0.11	-0.16 to 0.02	0.13
WC (*cm*)	Model 1	-0.09	-0.22 to 0.02	0.12
	Model 2	-0.14	-0.30 to 0.01	0.07
WHR (%)	Model 1	-0.04	-0.00 to 0.00	0.50
	Model 2	-0.03	-0.00 to 0.00	0.65
Visceral fat (*cm*^{2})	Model 1	0.09	-0.24 to 2.48	0.09
	Model 2	0.11	-0.51 to 3.52	0.14
RMR (*kcal*)	Model 1	-0.08	-5.54 to 1.11	0.19
	Model 2	-0.02	-5.15 to 3.80	0.76
RMR per weight (*kcal/kg*)	Model 1	0.05	-0.02 to 0.05	0.38
	Model 2	0.16	0.00 to 0.11	0.03

p-value result from linear regression; CI: Confidence Interval; Model 1: (crud); Model 2: (adjusted for Total Energy intake, physical activity, age): BMI: Body Mass Index; FM: Fat Mass; FFM: Fat Fee Mass; WC: Waist Circumference; WHR: Waist Hip Ratio: RMR, Resting Metabolic Rate;
Total energy intake is collinear for RMR

weight to childhood obesity, the mean birth weight in the obese children group did not significantly differ from normal children (14,18).

Our findings suggest that weight gain in adulthood is related to the age at onset of obesity rather than birth weight. An unknown part of obesity in childhood and adolescence may represent an inherent error in metabolism. The genetics of obesity is not clearly defined and cannot be assessed until a metabolic marker that has an etiological role in obesity is accurately identified and traced. Evidence suggested that people with a familial inherent predisposition to obesity tend to be obese at a younger age. Therefore, if hereditary metabolic factors are involved in obesity, their expression should be apparent early in life (6,8,15). Mossberg HO noted the onset of obesity may occur until the first year of life, but these early observations were not confirmed (15). Mossberg clarified that there are two important periods of childhood for obesity in adulthood, ages 0 to 4 and 7 to 11 years (15). Similarly, Heald FP in his study has found that birth weight was not associated with adolescent obesity, but the weight in the first year of life was associated with obesity in adulthood (17). The research has suggested that all obese people developed the same number and size of fat cells, but those who were obese at a younger age had more fat cells.

Our findings showed that the subjects in the upper age group at onset of obesity had lower RMR than those in the lower age group. The reason for this was the difference in body weight between the groups.

In fact, the study found that people who became obese as a child had more fat cells than adults. Obesity is also linked to an increase in adipocytes (9). Moreover, the research has shown that the early obesity pattern was significantly associated with overweight and obesity in adulthood (19). In this regard, an animal study has shown that the early growth of fat organs in mice was associated with an increase in the number and size of fat cells (20). However, after the 15th week of ectopic pregnancy, no further increase in the number of adipocytes occurs and further growth is only due to the growth of cells. At this age, the skeletal maturation of a rat is 99% complete and the corresponding age for human (Based on bone age) will be about 16. In adult life, weight changes in mice and humans have been found to be associated with changes in adipocyte size, but there was no relation in total cell number (21-23). The studies have shown that adolescent obesity is associated with an increase in adipose cell numbers in childhood. Our results show that people who were obese at a younger age have lower metabolic energy per kilogram and thus higher incidence of obesity in adulthood. It is not only related to the increase in cell number but also to other factors such as RMR. Prospective studies in this field are needed to confirm these results.

Conclusion

The results of this study showed that the age at onset of obesity was associated with obesity and overweight and RMR in adulthood. In fact, people with young age at obesity have higher body weight and fat mass and less RMR/*kg* body weight.

Limitations

There are certain limitations in our study which are worth considering. The main one might be the low number of participants. The cross-sectional design of the study is another important limitation as it prevented us from inferring causality. The absence of men is another major limitation. It should be noted that due to the cross-sectional design of the study, no exact cause-effect documentation can be associated with the current study. One of the inevitable limitations of this type of study is that questionnaire responses are subjectively based on participants' memory and their perception of pain.

References

1. Shah SM, Nanan D, Rahbar MH, Rahim M, Nowshad G. Assessing obesity and overweight in a high mountain Pakistani population. Trop Med Int Health 2004;9(4):526-32.

2. Yologlu S, Sezgin AT, Ozdemir R, Sezgin N, Colak C, Topal E, et al. Identifying risk factors in a mostly overweight patient population with coronary artery disease. Angiology 2003;54(2):181-6.

3. Rao SV, Donahue M, Pi-Sunyer FX, Fuster V. Obesity as a risk factor in coronary artery disease. The Am Heart J 2001;142(6):1102-7.

4. Cikim A, Ozbey N, Orhan Y. Relationship between cardiovascular risk indicators and types of obesity in overweight and obese women. J Int Med Res 2004;32(3):268-73.

5. Mohan V, Deepa R. Obesity and abdominal obesity in Asian Indians. Indian J Med Res 2006;123(5):593-6.

6. Mullins AG. The prognosis in juvenile obesity. Arch Dis Child 1958;33(170):307-14.

7. Haase KE, Hosenfeld H. [Obesity in childhood]. Zeitschrift Kinderheilkunde 1956;78(1):1-27. German.

8. Lloyd JK, Wolff OH, Whelen WS. Childhood obesity. A long-term study of height and weight. Brit Med J 1961;2:145-8.

9. Brook CGD, Lloyd JK, Wolf OH. Relation between age of onset of obesity and size and number of adipose cells. Br Med J 1972;2(5804):25-7.

10. Knittle JL, Hirsch J. Effect of early nutrition on the development of rat epididymal fat pads: cellularity and metabolism. J Clin Invest 1968;47(9):2091-8.

11. Asher P. Fat babies and fat children. The prognosis of obesity in the very young. Arch Dis Child 1966;41(220):672-3.

12. Mirmiran P, Esfahani FH, Mehrabi Y, Hedayati M, Azizi F. Reliability and relative validity of an FFQ for nutrients in the Tehran lipid and glucose study. Public Health Nutr 2010;13(5):654-62.

13. Hagströmer M, Oja P, Sjöström M. The International Physical Activity Questionnaire (IPAQ): a study of concurrent and construct validity. Public Health Nutr 2006;9(6):755-62.

14. Wolff OH. Obesity in childhood: a study of the birth weight, the height, and the onset of puberty. Q J Med 1955;24(94):109-23.

15. Mossberg HO. Obesity in children: A clinical-prognostical investigation. Acta Pediatr 1948;35(Suppl 2).

16. Bruch H. Obesity in childhood: I. Physical growth and development of obese children. Am J Dis Child 1939;58(3):457-84.

17. Heald FP, Hollander RJ. The relationship between obesity in adolescence and early growth. The J Pediatr 1965;67(1):35-8.

18. Bancroft T, Anderson R. Statistical theory in research. New York, USA: McGraw Hill Book Company, Inc. 399 p.

19. Mills JK, Andrianopoulos GD. The relationship between childhood onset obesity and psychopathology in adulthood. J Psychol 1993;127(5):547-51.

20. Hirsch J, Gallian E. Methods for the determination of adipose cell size in man and animals. J Lipid Res 1968;9(1):110-9.

21. Hughes PC, Tanner JM. The assessment of skeletal maturity in the growing rat. J Anat 1970;106(Pt 2):371-402.

22. Hollenberg C, Vost A. Regulation of DNA synthesis in fat cells and stromal elements from rat adipose tissue. J Clin Invest 1968;47(11):2485-98.

23. Sims EA, Goldman RF, Gluck CM, Horton ES, Kelleher PC, Rowe DW. Experimental obesity in man. Trans Assoc Am Physicians 1968;81:153-70.