Factors Affecting the Risk of Childhood Obesity in the Bağcılar region of İstanbul

İstanbul Bağcılar Bölgesinde Çocukluk Çağında Obeziteyi Etkileyen Risk Faktörleri ve Obezitenin Sonuçları

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ABSTRACT

Objective: Childhood obesity has recently become a common health problem worldwide. In the struggle against obesity, studies have focused on the risk factors playing a role in the development of obesity. In this study, we assessed the risk factors playing a role in childhood obesity and the resulting obesity in the İstanbul Bağcılar Region.

Methods: In total, 250 obese children, aged 4-15 years, and 98 non-obese children of the same age were included in this study. A standardized questionnaire aimed at determining the sociodemographic characteristics, television-watching schedule, nutritional habits, physical activity, presence of obesity in the family, and duration of breastfeeding was provided to the study and control groups. Glucose, insulin, cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglyceride levels were measured in fasting serum samples. Homeostasis model assessment-insulin resistance (HOMA-IR) values were calculated.

Results: The mean age of the study group was 10.71±2.69 years; there were 112 (44.80%) males and 138 (55.20%) females. The pubertal period (p=0.0001), the presence of obese individuals in the family (p=0.021), and watching television for more than 3 h per day (p=0.0001) were found to be risk factors for childhood obesity. Increased HOMA-IR (p=0.0001), increased fasting insulin (p=0.003), and decreased HDL (p=0.037) levels were the most influential parameters in obesity.

Conclusion: Childhood obesity can lead to serious health problems by affecting obesity in adulthood. To initially prevent obesity requires a full understanding of the risk factors and biological and social pathways leading to obesity in early life.

Keywords: Childhood, obesity, risk factors, HOMA-IR

ÖZ

Amaç: Çocukluk çağı obezitesi son yıllarda tüm dünyada yaygın bir sağlık sorunu haline gelmiştir. Son zamanlarda obezite ile mücadelede obezitede rol oynayan risk faktörleri üzerinde durulmaktadır. Bu çalışmada İstanbul Bağcılar Bölgesinde obezite gelişiminde rol oynayan risk faktörleri ve obezitenin sonuçları değerlendirilmiştir.

Yöntemler: Yaş aralığı 4-15 olan 250 obez hasta çalışmaya dahil edilmiştir. Çalışmaya alınan obez hastaların vücut kitle indeksi hesaplandı. Hastalara sosyodemografik verilerini değerlendirmek üzere televizyon izleme süresini, beslenme alışkanlıklarını, fiziksel aktivite durumunu, ailede obez birey varlığını ve tamamlayıcı beslenmeye geçiş zamanını sorgulayan anket formları uygulandı. Açlık glukoz, insülin, kolesterol, yüksek dansiteli lipoprotein, düşük dansiteli lipoprotein ve trigliserit düzeyleri ölçüldü. Homeostasis model assessment-insulin resistance (HOMA-IR) değerleri hesaplandı.

Bulgular: Çalışma grubunun yaş ortalaması 10,71±2,69 yıldı. Hastaların 112'si (%44,80) erkek, 138'i (%55,20) kızdı. Pubertal dönemin (p=0,0001), ailede obez birey varlığının (p=0,021), günlük 3 saatten fazla televizyon izleme süresinin (p=0,0001) çocukluk çağı obezitesinde risk faktörü olduğu görüldü. HOMA-IR yüksekliğinin (p=0,0001), açlık insulin düzeyi yüksekliğinin (0,003) ve yüksek dansiteli lipoprotein düşüklüğünün (p=0,037) obeziteden en fazla etkilenen parametreler olduğu gözlendi.

Sonuç: Çocukluk dönemi ve adelosan dönemde obez olmak yetişkin dönemde de devam edip ciddi sağlık sorunlarına neden olmaktadır. Obezitede risk faktörlerinin saptanıp beslenme konusunda ailelerin ve çocukların bilinçlendirilmesi obezite ile mücadelede önemlidir.

Anahtar kelimeler: Çocukluk çağı, obezite, risk faktörleri, HOMA-IR

INTRODUCTION

Obesity is a metabolic disease characterized by an abnormal or excessive level of fat, decreasing the health status of the body (1, 2). Recently, the prevalence of obesity has increased rapidly, and it has become a public health issue not only in developed countries but also in developing countries (3). Obesity causes both health and social problems, and it also results in approximately 30,000 early deaths in Turkey and worldwide (4). If obesity begins before the age of 5 years or after the age of 15 years, it can be

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more dangerous. It can cause depression, fatty liver syndrome, asthma, sleep apnea, hypertension, orthopedic problems, and type 2 diabetes (5). There are different methods to decrease the frequency of obesity, such as pharmacological treatments, diet, education, and behavioral methods, but their success has been very limited to date. Thus, studies to determine environmental factors to decrease calories and enable active lifestyles have continued, but the relative effects of environmental factors on obesity have not been clearly identified. These factors have generally been called "obesogenic" factors (6). According to Swinburn (7), who suggested the concept of an obesogenic environment, obesity in society or individuals can be prevented by decreasing obesogenic factors. Swinburn (7) stated that more than one factor may cause obesity, and the living environment has a significant effect on nutrition and physical activity. Risk factors for childhood obesity in developing countries have been suggested to include rapidly changing nutritional habits, sedentary lifestyles, increased socioeconomic level, increased urbanization, being female, misconceptions about nutrition, and limitations on physical activity (8). Childhood obesity is rapidly increasing in our country, as it is worldwide. Dealing with childhood obesity is very difficult, and instances of weight loss have been observed to be very low during childhood. Recent studies have concentrated on "obesogenic reasons" behind this problem. Attempts have been made to determine risk factors in obesity development (9). In this study, we evaluated the risk factors playing roles in childhood obesity.

METHODS

The study was conducted between April 2014 and May 2015, approved by the local ethics committee (protocol no. 2014/231), and performed according to the ethical standards of the Declaration of Helsinki. Written informed consent was obtained from all the patients.

We studied 250 obese children aged 4-15 years. No child had a chronic disease or was under medical treatment. Obesity was considered present when a patient's body mass index (BMI) was above the 95th percentile. Patients who needed to use drugs due to chronic disease and who had genetic problems were excluded from the study. In addition, patients who were considered to have developed obesity due to causes such as hypothyroidism (Cushing's syndrome) were excluded from the study. The control group comprised 98 children in the same age range with BMI percentiles <95%. The body weight and height of the children were measured by the same person. Weight was measured using a portable electronic scale with the subject wearing light clothes and no shoes. Height was measured using a stadiometer with the subject's shoes off, feet together, and head in the horizontal plane. All the children were examined by a pediatrician and a pediatric endocrine specialist; pubertal stage was scored using the Tanner scale. A testicle volume of ≥4 mL in males and the presence of breast development Tanner stage ≥2 in females were accepted as signs indicating the initiation of puberty. Blood samples taken from patients after 8-10 h of fasting were evaluated with standard methods using a Roche Modular P 800 device.

A standardized questionnaire was completed by these patients and their parents. Gender, age, physical activity, computer use, watching television (TV) time, weight status of parents, family history of obesity, nutritional habits, duration of breastfeeding, and birth weight were included in the questionnaire. Skipping breakfast and snacking instead, as well as snacking while studying, watching TV, or using a computer were considered atypical nutrition habits. To gather information on eating habits, we requested a 7 - day eating record from the children and their families. In these forms, families were questioned about how many meals their children eat a day, what they eat while snacking, and about whether they have breakfast. The level of physical activity was considered normal if the duration of such activity was >7 h/week. Breastfeeding duration was categorized as <6 months or ≥6 months. Daily screen time was categorized as < 3 h/day, 3-6 h/day, and >6 h/day. Glucose, insulin, cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglyceride levels were measured in fasting serum samples. Homeostasis model assessment-insulin resistance (HOMA-IR) values were calculated using the following equation: fasting insulin concentration (μU/mL) × fasting glucose concentration (mmol/L)/22.5 (10). A HOMAIR level > 2.5 in pre-pubertal patients was considered pathological; for pubertal patients, the value was > 3.16 (11). A fasting insulin level >15 μU/mL was considered pathological in pre-pubertal patients; for pubertal subjects, this value was > 20 μU/mL. Triglyceride levels >50 μg/dL and HDL-C levels <40 μg/dL were considered pathological (12).

Statistical Analysis

We used the Number Cruncher Statistical System 2007 (NCSS, Utah, USA) to conduct all the analyses. Continuous variables are reported as means with standard deviations. The independent *t*-test was used to compare data between groups, and the χ^2 test was used to compare qualitative data. Logistic regression analysis was used to define risk factors for obesity. Here, $p<0.05$ was considered to indicate statistical significance, and 95% confidence intervals were calculated.

RESULTS

The mean age of the obese group was 10.71±2.69 years and that of the control group was 10.13±3.25 years; this difference was not significant (p=0.09). In the obese group, 112 (44.80%) children were males and 138 (55.20%) children were females, and in the control group, 50% were females and 50% were males; this difference in gender between the groups distribution was not significant (p=0.38). The BMI percentile of the control group was 18.17±2.4, where as that of the obese group was 28.63±5.32; the difference was statistically significant (p=0.0001). In the study group, patients were above the 95th percentile, and therefore, considered obese. In the control group, children were between the 25th and 75th percentiles. Demographic data for the study and control groups are presented in Table 1. The adolescent age in the study group was significantly larger than that in the control group (p=0.0001). Irregular dietary habits (p=0.0001) were significantly more common in obese individuals in the study group (p=0.003) than the control group. Watching TV more than 6 h/day was significantly more common in the obese group than the control group (p=0.0001).

The mean fasting glucose, insulin level, HOMA-IR, cholesterol, triglyceride, and LDL levels in the obese group were significantly higher than those in the control group (p<0.005), whereas the

Table 1. Demographic features of the control group and study group

		Control group n=98		Obese Group n=250		p
Puberty	Prepubertal	53	54.08%	79	31.60%	0.0001
	Pubertal	45	45.92%	171	68.40%	
Birth weight		3368.37±554.71		3377.88±626.7		0.895
Obesity in family	Not present	63	64.29%	116	46.40%	0.003
	Present	35	35.71%	134	53.60%	
Physical activity	Irregular	31	31.63%	105	42.00%	0.097
	Regular	67	68.37%	145	58.00%	
Nutrition habits	Regular	98	100.00%	81	32.40%	0.0001
	Irregular	0	0.00%	169	67.60%	
Breastfeeding duration	<6 months	25	25.51%	75	30.00%	0.483
	>6 months	73	74.49%	175	70.00%	
TV watching duration	<3 h/day	50	51.02%	56	22.40%	0.0001
	3 - 6 h/day	37	37.76%	76	30.40%	
	>6 h/day	11	11.22%	118	47.20%	

Table 2. Laboratory findings of the study group and control group

	Control group n=98	Obese Group n=250	p
Fasting glucose level (mg/dL)	91.01±9.12	93.77±7.2	0.008
Fasting insulin level (IU/mL)	10±4.73	20.74±12.2	0.0001
HOMA-IR	2.33±1.12	4.71±2.93	0.0001
Total cholesterol (mg/dL)	147.36±23.96	164.46±29.85	0.0001
Triglyceride (mg/dL)	86.56±50	123.53±67.81	0.0001
HDL-cholesterol (mg/dL)	53.1±12.95	48.68±11.83	0.003
LDL-cholesterol (mg/dL)	82.39±16.73	91.91±27.25	0.001

HDL:high-density lipoprotein; LDL:low-density lipoprotein; HOMA-IR: homeostasis model assessment–insulin resistance

mean HDL level of the obese group was significantly lower than the control group (p=0.003, Table 2).

Univariate regression analysis was used to define the risk factors for obesity, and the results showed that irregular eating habits [49.7 (5.11-87.2)], daily screen time >6 h/day [9.58 (4.63-19.8)], obesity in the family [2.8 (1.28-3.36)], and puberty [2.54 (1.58-4.11)] were risk factors for childhood obesity. Furthermore, 3-6 h/day of screen time was also a risk factor for childhood obesity. HOMA-IR [2.16 (1.75-2.66)], increased fasting insulin levels [1.23 (1.16-1.29)], increased triglyceride levels [1.01 (1.00-1.02)], total cholesterol level [1.02 (1.01-1.03)], LDL-C level [1.02 (1.00-1.03)], fasting glucose level [0.96 (0.94-0.99)], and decreased HDL-C level [0.977 (0.95-0.99)] influenced obesity (Table 3). In the multivariate logistic regression analysis with the same variables, increased HOMA-IR and cholesterol levels and decreased HDL-C levels were associated with childhood obesity.

DISCUSSION

Childhood obesity is caused by excess energy intake resulting from unhealthy dietary habits and insufficient physical activity. Pediatric obesity develops due to genetic and non-genetic factors and/or their interaction. Genetics and the social environment (socioeconomic status, race, physical environment, media, and shopping culture) affect the energy consumption and energy expenditure (13). Children and adolescents with BMI above the 95th percentile are considered obese (14). The worldwide prevalence of childhood obesity has increased greatly over the last three decades (15, 16).

The frequency of childhood obesity varies by country and by regions within the same country (8). One in four children is obese or overweight among children in the 6-14-year-old age group in developed and developing countries (17), and the obesity and overweight rate is 11-39% (18). In some studies, obesity prevalence differed between males and females; some studies reported that it was more common in females (17), more common in males (18, 19), or equally occurring between males and females (17). In our study, obesity was observed equally in males and females.

We also found that pubertal status was a risk factor for obesity. The 2011-2012 National Health and Nutrition Examination Survey (NHANES) found that 31.8% children and 16.9% adolescents were overweight or obese. In terms of age, those aged 12-19 years were more likely to be overweight or obese than those aged 2-5 and 6-11 years (20). In one study, the prevalence of obesity in those aged 11-18 years was reported as 7.7% (8.4%

Table 3. Univariate and multiple logistic regression analyses results

		Univariate Risk		Multivariate Risk Family Feature		Multivariate Risk Laboratory	
		OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
Puberty		2.54 (1.58-4.11)	0.0001	0.48 (0.25-0.93)	0.031		
Obesity in family		2.8 (1.28-3.36)	0.003	0.67 (0.38-1.25)	0.021		
Feeding habits irregular		49.7 (5.11-87.2)	0.0001	0.01 (0.00-0.14)	0.992		
TV watching duration	3-6 h/d	1.83 (1.06-3.71)	0.0001	0.21 (0.11-0.59)	0.0001		
	>6 h/d	9.58 (4.63 -19.8)	0.0001	0.46 (0.17-0.87)	0.006		
Fasting glucose level (mg/dL)		0.96 (0.94-0.99)	0.008				
Fasting insulin level (IU/mL)		1.23 (1.16-1.29)	0.0001				
HOMA-IR		2.16 (1.75-2.66)	0.0001			2.02 (1.67-2.52)	0.0001
Total cholesterol (mg/dL)		1.02 (1.01-1.03)	0.0001			1.36 (1.09-1.56)	0.003
Triglyceride (mg/dL)		1.01 (1.00-1.02)	0.0001			0.99 (0.95-1.11)	0.764
HDL-cholesterol (mg/dL)		0.97 (0.95-0.99)	0.003			0.96 (0.94-1.10)	0.037
LDL-cholesterol (mg/dL)		1.02 (1.00-1.03)	0.001			0.99 (0.96-1.01)	0.238

HDL:high-density lipoprotein; LDL:low-density lipoprotein; HOMA-IR: homeostasis model assessment–insulin resistance; CI: confidence interval

for females and 7% for males) (21). In that study, time watching TV or using computers>2h per day and the presence of obese persons in the family were found to be the risk factors responsible for obesity development. Among early adolescents (10-14 years), poor diet quality along with physical inactivity may contribute toward an increased risk of obesity (22). Puberty is a transition period, when physical and hormonal changes are observed. Physical inactivity, using a computer for fun or study, sitting with friends or hanging out, and reading for fun may be excessive. Because it restricts movement, intensive urbanization is one factor increasing obesity. Adolescence is the transitional period from childhood to adulthood, and it is associated with rapid change in behaviors. Adult habits may be initiated in adolescence, but social hierarchies that influence adolescent behavior may differ from those that influence adult behavior (14). Recent studies have shown that obesity is less prominently associated with morbidity in adolescence, but it is a strong precursor of obesity and related morbidity in adulthood, with 50%-80% of obese adolescents becoming obese adults (23).

We found, as have others, that an obese family member was a risk factor for obesity. Having obese parents has been shown to be a strong determinant of childhood obesity (24). Obesity in one or both parents is an important predictor for whether a child's obesity will persist into adulthood (25). A study by Burke et al. (26) demonstrated the association between child BMI and parental BMI. According to this study, if the father was obese, the obesity risk in both male and female children was increased by four times, and if the mother was obese, the obesity risk for female children was increased by eight times. Genetic factors also play an important role in obesity. In various studies, 25%-40% variation in BMI was explained by genetic transmission (27). However, genetic factors alone do not determine childhood obesity, although they constitute a risk factor, together with environmental factors.

In this present study, irregular eating habits were found to be a risk factor for the development of obesity. The results showed that obese children (especially adolescents) skip breakfast more frequently or eat less at breakfast when compared with non-obese children. In fact, children who skipped breakfast tended to eat food that contained more energy and to increase the amount eaten at other meals. Although skipping breakfast is considered to be a facilitating risk factor for body adiposity, studies performed on this subject have given contradictory results. However, in studies, weight loss was also demonstrated to be better when obese children begin to have breakfast. Therefore, breakfast should certainly be included in the treatment of obesity (27). It was observed, in our study, that those who skipped breakfast ate high-calorie foods from school canteens. Not the frequency of snacks during the day, but the total energy obtained from them was associated with BMI (28). Fat- and energy-containing snacks are associated with obesity. The snacks that were particularly preferred by adolescents included potato chips, ice cream, candies, cereals, muffins, and carbonated beverages. Such snacks constituted a quarter or more of the daily total energy requirement, contributing toward being overweight. The consumption of junk food easily bought from supermarkets, the density of grocery stores in the neighborhood, ordering food for home delivery, an excessive number of fast food buffets, and the presence of restaurants were important factors associated with obesity (29). Among children and adolescents, the consumption of sodas and sugary beverages is very common. In one study, 11% of the calories needed by adolescents were contributed by these drinks, and they were consumed two times more often than beneficial drinks such as water, mineral water, and milk. A study published in the Lancet reported a relationship between sugar-intensive beverage consumption and the BMI in children, concluding that this is one of the most important reasons for the increase in childhood obesity (30).

The duration of screen time is a risk factor for developing obesity. A study in Germany demonstrated that watching TV or playing video games on a computer for more than 1 h per day increased weight gain. Movement during computer gaming is lower than that during other activities practiced outside. For each hour spent in watching TV, the obesity development risk increased (31). Sitting still while watching TV and watching advertisements about high-calorie products, as well as consuming these products, accelerate weight gain among children.

Worldwide, children spend an average of nearly 5.5 h in front of media tools and encounter commercial food advertisements nearly every 5 min (32). Advertisements aimed at children are found on the Internet, in educational materials, video games, toys, movies, and films, particularly in cartoons. Children younger than 8 years are more susceptible to these advertisements (33). In the present study, physical activity was not found to be a risk factor for obesity. Recent studies suggest that low levels of physical activity tend to increase the risk of obesity in children. The lack of sufficient areas suitable for walking and cycling in urban planning has been reported to be related to obesity as it results in children's preferring to stay indoors and watching TV (34).

The duration of breastfeeding in infancy and complementary food types are known to affect long-term food choices. Generally, it has been thought that rapid weight gain in infancy plays an important role in the development of obesity. For this reason, breastfeeding should continue longer to prevent obesity (10). In our study, we observed no significant relationship between obesity and the duration of breastfeeding time.

HOMA-IR is among the most important indicators in determining insulin resistance in obese children and adolescents (35). Insulin resistance and type 2 diabetes mellitus are among the most significant factors causing adverse health consequences related to obesity.

Approximately one-third of obese children and adolescents are insulin-resistant (36). Type 2 diabetes, obesity, hypertension, high LDL-C, low HDL-C, and elevated fasting insulin levels are the most common outcomes of obesity in childhood and adolescence (37). In the present study, HOMAIR was the parameter most affected by obesity. Dyslipidemia is more common in obese than non-obese patients. In obese individuals, serum-free fatty acid (FFA) levels are high as a result of lipolysis. FFA levels trigger hypertriglyceridemia by inhibiting the lipoprotein lipase of adipose and muscle tissues and increasing the production of very low density lipoprotein (VLDL) and triglyceride by the liver. The degradation of triglyceride-rich LDL-C and HDL-C by hepatic lipases increases LDL-C levels and reduces HDL-C levels (38). In the present study, it was found that dyslipidemia is common in obese children. This study was conducted only in one region of Istanbul, which is a limitation of this study.

CONCLUSION

Childhood obesity is an important problem for both Turkey and the world. The risk factors identified by us regarding childhood obesity in our country include puberty, family's eating habits, presence of obese individuals in the family, unhealthy and irregular eating habits, and watching TV for more than 3 h/day. During rapid urbanization, because there are few secure places where children can exercise and move, they tend to stay at home and indoors. This also causes an increase in the time spent by children in front of TV and computer screens, which decreases their energy expenditure. Furthermore, obesity in infancy and adolescence causes the development of insulin resistance and dyslipidemia, which adversely affects their health in adulthood.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Ministry of Health University of Health Sciences Bağcılar Training and Research Hospital.

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REFERENCES

1. Woods SC, Seeley RJ. Understanding the physiology of obesity: review of recent developments in obesity research. Int J Obes Relat Metab Disord 2002; 26: 8-10. [CrossRef]
2. Mc Carthy HD. Measuring growth and obesity across childhood and adolescence. Proc Nutr Soc 2014; 73: 210-7. [CrossRef]
3. Candido AP, Freitas SN, Machado-Coelho GL. Anthropometric measurements and obesity diagnosis in school children. Acta Paediatr 2011; 100: 120-4. [CrossRef]
4. Atabek ME, Pirgon O, Kurtoglu S. Prevalence of metabolic syndrome in obese Turkish children and adolescents. Diabetes Res Clin Pract 2006; 7: 315-21. [CrossRef]
5. Pirgon O, Sandal G, Gokcen C, Bilgin H, Dündar B. Social anxiety, depression and self-esteem in obese adolescent girls with acanthosis nigricans. J Clin Res Pediatr Endocrinol 2015; 7: 63-8. [CrossRef]
6. Swinburn B, Egger G. Preventive strategies against weight gain and obesity. Obes Rev 2002; 3: 289-301. [CrossRef]
7. Swinburn B, Egger G, Raza F. Dissecting obesogenic environments: the development and application of a frame work for identifying and prioritizing environmental interventions for obesity. Prev Med 1999; 29: 563-70. [CrossRef]

8. Goel K, Shah P, Misra A. Childhood obesity in developing countries: epidemiology, determinants, and prevention. Endocr Rev 2012; 33: 48-70. [CrossRef]
9. M. Karen Campbell. Biological, enviromental, and social influences on childhood obesity. Pediatr Res 2016; 79: 205-11. [CrossRef]
10. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, et al. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985; 28: 412-9. [CrossRef]
11. Singh Y, Garg MK, Tandon N, Marwaha RK. A study of insulin resistance by HOMA-IR and its cut-off value to identify metabolic syndrome in urban Indian adolescents. J Clin Res Pediatr Endocrinol 2013; 5: 245-51. [CrossRef]
12. Daniels SR, Greer FR; Committee on Nutrition. Lipid screening and cardiovascular health in childhood. Pediatrics 2008; 122: 198-208. [CrossRef]
13. Gungor NK. Overweight and obesity in children and adolescents. J Clin Res Pediatr Endocrinol 2014; 6: 129-43. [CrossRef]
14. Hubbard VS. Defining overweight and obesity: what are the issues? Am J Clin Nutr 2000; 72: 1067-68.
15. Han JC, Lawlor DA, Kimm SY. Childhood obesity 2010: progress and challenges. Lancet 2010; 375: 1737-48. [CrossRef]
16. Ara I, Moreno LA, Leiva MT, Gutin B, Casajús JA. Adiposity, physical activity, and physical fitness among children from Aragón, Spain. Obesity 2007; 15:1918-24. [CrossRef]
17. Brunet M, Chaput JP, Tremblay A. The association between low physical fitness and high body mass index or waist circumference is increasing with age in children: the 'Québec en Forme' Project. Int J Obes 2007; 31: 637-43.
18. Sur H, Kolotourou M, Dimitriou M, Kocaoglu B, Keskin Y, Hayran O, et al. Biochemical and behavioral indices related to BMI in school children in urban Turkey. Prev Med 2005; 41: 614-21. [CrossRef]
19. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. JAMA 2014; 311: 806-14. [CrossRef]
20. Ercan S, Dallar YB, Onen S, Engiz O. Prevalence of obesity and associated risk factors among adolescents in Ankara, Turkey. J Clin Res Pediatr Endocrinol 2012; 4: 204-7.
21. Reicks M, Banna J, Cluskey M, Gunther C, Hongu N, Richards R, Topham G, Wong SS. Influence of parenting practices on eating behaviors of early adolescents during independent eating occasions: implications for obesity prevention. Nutrients 2015; 7: 8783-801. [CrossRef]
22. Berenson GS, Srinivasan SR, Bao W, Newman WP3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. N Engl J Med 1998; 338: 1650-6. [CrossRef]
23. Lake JK, Power C, Cole TJ. Child to adult body mass index in the 1958 British birth cohort: associations with parental obesity. Arch Dis Child 1997; 77: 376-81. [CrossRef]
24. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med 1997; 337: 869-73. [CrossRef]
25. Burke V, Beilin LJ, Dunbar D. Family lifestyle and parental body mass index as predictors of body mass index in Australian children: a longitudinal study. Int J Obes Relat Metab Disord 2001; 25: 147-57. [CrossRef]
26. Anderson PM, Butcher KE. Childhood obesity: trends and potential causes. Future Child 2006; 16: 19-45. [CrossRef]
27. Spear BA, Barlow SE, Ervin C, Ludwig DS, Saelens BE, Schetzina KE, et al. Recommendations for treatment of child and adolescent overweight and obesity. Pediatrics 2007; 120: 254-88. [CrossRef]
28. Kerr MA, Rennie KL, McCaffrey TA, Wallace JM, Hannon-Fletcher MP, Livingstone MB. Snacking patterns among adolescents: a comparison of type, frequency and portion size between Britain in 1997 and Northern Ireland in 2005. Br J Nutr 2009; 101: 122-31. [CrossRef]
29. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. Lancet 2001; 357: 505-8. [CrossRef]
30. Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. JAMA 1999; 282: 1561-7. [CrossRef]
31. Kunkel D, Gantz W. Children's television advertising in the multichannel environment. J Comm 1992; 42: 134-52. [CrossRef]
32. Kotz K, Story M. Food advertisements during children's Saturday morning television programming: are they consistent with dietary recommendations? J Am Diet Assoc 1994; 94: 1296-300. [CrossRef]
33. Sobal J. Commentary: globalization and the epidemiology of obesity. Int J Epidemiol 2001; 30: 1136-7. [CrossRef]
34. Kurtoğlu S, Hatipoğlu N, Mazıcıoğlu M, Kendirici M, Keskin M, Kondolot M. Insulin resistance in obese children and adolescents: HOMA-IR cut-off levels in the prepubertal and pubertal periods. J Clin Res Pediatr Endocrinol 2010; 2: 100-6. [CrossRef]
35. Bereket A, Atay Z. Current status of childhood obesity and its associated morbidities in Turkey. J Clin Res Pediatr Endocrinol 2012; 4: 1-7. [CrossRef]
36. Clarson CL, Mahmud FH, Baker JE, Clark HE, McKay WM, Schauteet VD, et al. Metformin in combination with structured lifestyle intervention improved body mass index in obese adolescents, but did not improve insulin resistance. Endocrine 2009; 36:141-6. [CrossRef]
37. Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. Pediatrics 1999; 103: 1175-82. [CrossRef]
38. Klop B, Elte JW, Cabezas MC. Dyslipidemia in obesity: mechanisms and potential targets. Nutrients 2013; 5: 1218-40.[CrossRef]