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10.20960/nh.05933

10/13/2025

OR 5933

# Obesity in youth and its relatıonship with the diagnosis of vıtamin D deficiency and insulin resistance according to pubertal status – A retrospective study from Turkey

*Obesidad en jóvenes y su relación con la deficiencia de vitamina D y la resistencia a la insulina según el estado puberal: estudio retrospectivo de Turquía*

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Received: 12/05/2025
Accepted: 02/09/2025
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*Acknowledgements: We thank all Baskent University pediatricians for their valuable time.*

*This study was presented in ESPGHAN 55th Annual Meeting in Obesity, growth and metabolism section.*

*Conflict of interest: The authors declare no conflict of interest.*

*Artificial intelligence: The authors declare not to have used artificial intelligence (AI) or any AI-assisted technologies in the elaboration of the article.*


**ABSTRACT**

**Introduction:** as children progressed into puberty, a notable escalation in the prevelance of obesity and insulin resistance was observed. Specifically during this developmental stage, both obesity and insulin resistance exhibited a pronounced surge.

**Objective:** this study aimed to determine the relationships between serum 25(OH) vitamin D deficiency and insulin resistance in overweight and obese children and adolescents.

**Methods**: a total of 418 individuals aged 6-18 years (11.9 ± 3.23) diagnosed with obesity or overweight retrospectively were included in the study. Data including gender, age, body weight, height, anthropometric measurements, and biochemical parameters were recorded from files between 2020-2022. Body mass index (BMI) for age values were evaluated using WHO 5-19 years Growth Reference.

**Results:** among participants, 37.5 % of them were prepubertal and the mean age was 11.94 ± 3.32 years. Vitamin D insufficiency was found 43.9 % in prepuberty and 51.3 % in pubertal stage. While the presence of insulin resistance in the prepubertal stage was 36.8 %, it was 25.7 % in the pubertal stage. In the pubertal group, HOMA-IR values significantly increased as serum 25-OH vitamin D levels decreased ($p$ = 0.002). Mean 25-OH Vitamin D was negatively associated with HDL-C and positively associated with BMI for age and HOMA-IR.

**Conclusion:** a noteworthy corrrelation was established between vitamin D deficiency and the incidence of insulin resistance, alongside obesity. As a result, this study, which included a limited number of cases, showed significantly lower levels of vitamin D especially in

obese patients with insulin resistance. There is a need for prospective, large-scale and long-term studies to verify our results.

**Keywords:** Childhood obesity. Vitamin D. Insulin resistance. Body mass index.

**RESUMEN**

**Introducción:** a medida que los niños avanzan hacia la pubertad, se observa una notable escalada en la prevalencia de la obesidad y la resistencia a la insulina. Específicamente durante esta etapa del desarrollo, tanto la obesidad como la resistencia a la insulina muestran un aumento pronunciado.

**Objetivo**: este estudio pretendía determinar las relaciones entre la deficiencia sérica de 25(OH)-vitamina D y la resistencia a la insulina en niños y adolescentes con sobrepeso y obesidad.

**Métodos:** se incluyó en el estudio a un total de 418 individuos de edades comprendidas entre los 6 y los 18 años (11,9 ± 3,23), diagnosticados retrospectivamente de obesidad o sobrepeso. Los datos, que incluían sexo, edad, peso corporal, altura, medidas antropométricas y parámetros bioquímicos, se registraron a partir de archivos de 2020-2022. Los valores del índice de masa corporal (IMC) para la edad se evaluaron utilizando la referencia de crecimiento de 5-19 años de la OMS.

**Resultados:** el 37,5 % de los participantes eran prepúberes y la edad media era de 11,94 ± 3,32 años. Se encontró insuficiencia de vitamina D en el 43,9 % de los prepúberes y en el 51,3 % de los púberes. Si bien la presencia de resistencia a la insulina en la etapa prepuberal fue del 36,8 %, en la puberal fue del 25,7 %. En el grupo puberal, los valores HOMA-IR aumentaron significativamente a medida que disminuían los niveles séricos de 25-OH-vitamina D ($p$ =

0,002). La media de 25-OH-vitamina D se asoció negativamente con el HDL-C y positivamente con el IMC para la edad y el HOMA-IR.
**Conclusiones:** se estableció una notable correlación entre la deficiencia de vitamina D y la incidencia de resistencia a la insulina, junto con la obesidad. En consecuencia, en este estudio, que incluyó un número limitado de casos, se observaron niveles significativamente más bajos de vitamina D, especialmente en los pacientes obesos con resistencia a la insulina. Se necesitan estudios prospectivos, amplios y a largo plazo para verificar nuestros resultados.

**Palabras clave:** Obesidad infantil. Vitamina D. Resistencia a la insulina. Índice de masa corporal.

## INTRODUCTION

Obesity is a global epidemic affecting all age groups, characterized by the accumulation of excess fat tissue in the body (1). Childhood obesity, which often results from unhealthy and unbalanced nutrition, plays a significant role in the future development of various health problems such as diabetes, hypertension, respiratory issues, and cardiovascular diseases (2-4).

There is a strong relationship between increased prevalence of insulin resistance (IR), type 2 diabetes, metabolic syndrome, and higher cardiovascular risk in the obese child and adolescents (5). Although treatment of obesity is possible, focus should be on prevention. Therefore, growth monitoring is heplful in preventing childhood obesity at early stages. Moreover identifying the epigenetic and environmental factors, establishing healthy eating habits, and lifestyle changes should be provided (6).

Obesity and the contributing factors have been the subject of numerous studies for years (7-9). In addition to endocrine,

environmental, and genetic factors affecting obesity, various physiological mechanisms, psychological and cultural factors, excessive consumption of high-energy fatty and sugary foods, easy access to unhealthy packaged foods, incorrect dietary habits such as overconsumption, sedentary lifestyle, more than 2-3 hours daily screen time, and factors such as low income, which interact with each other, are among the causes of obesity (7). Endogenous obesity results from hormonal conditions where medications, endocrine, and genetic factors are involved in the etiopathogenesis. Exogenous obesity, on the other hand, is linked to environmental factors such as dietary habits (8). Exogenous obesity is more common in childhood since most obese children do not have an underlying medical condition (6).

Insulin resistance can be seen widely in childhood, parallel with the increase in childhood obesity. Vitamin D has known anti-proliferative, pro-differential, pro-apoptotic and immuno-modulation effects and functions in insulin-sensitive organs such as the liver and skeletal muscle, can increase insulin sensitivity (9). In recent years, the role of low serum 25-OH vitamin D levels has been extensively investigated in relation to obesity (10,11). A meta-analysis has identified that the prevalence of vitamin D deficiency was 35 % and 24 % higher in obese subjects and in overweight subjects, compared ton on-obese individuals (12). The increase in renin angiotensin activity with increasing fat tissue in obesity reduces adiponectin levels, which may be exacerbated by vitamin D deficiency. Moreover, adipocytokines found to be elevated in circulation in obese individuals, such as leptin and interleukin-6, may also have an inhibitory effect on vitamin D synthesis through receptor mediation (13). A study conducted in the United States found vitamin D deficiency in 29-34 % of obese children and in 49 % of morbid obese children (14). For this reason, the current research has been conducted to investigate the association between serum 25-OH vitamin and insulin resistance in exogenous obese children and adolescents.

## MATERIALS AND METHODS

### Subjects and study design

The data for this retrospective cross-sectional study was recorded from the files of children and adolescents aged 6-18 years diagnosed with obesity or overweight who were admitted to the Pediatric Endocrinology Clinic of Baskent University Hospital between January 2020 and December 2022. Our study approved by the XXX University Faculty of Medicine Ethical Committee (KA21/484). A total of 1348 patient records were reviewed. Among these, 789 records were excluded for not meeting the inclusion criteria, and 141 records were excluded due to duplications. Therefore, data from 418 patients were included in the final analysis.

The files included in the study were those of individuals aged 6-18 years who were either overweight or obese (between ≥ 85th to 95th percentile for overweight, ≥ 95th percentile for obese, ≥ 99th percentile for morbid obese) according to the World Health Organization (15) criteria. Individuals with celiac disease, growth retardation, severe and chronic infections, any metabolic or endocrine diseases, who had received vitamin D supplementation in the past year, and were pregnant at the time of their visit were excluded. Pubertal status was determined in the patient folder by the pediatric endocrinologist according to testicular volume equal to or greater than 4 ml in boys and onset of breast development in girls, according to that children were divided into two groups as pubertal and prepubertal.

### Data form

Data was collected using a form developed by the researcher. The research form included demographic characteristics (gender, age), anthropometric measurements (body weight, height, BMI), and blood

biochemical parameters [fasting blood glucose (mg/dL), fasting insulin (mg/dL), HOMA-IR, total cholesterol (mg/dL), LDL-C (mg/dL), HDL-C (mg/dL), triglycerides (mg/dL), AST (IU/L), ALT (IU/L), TSH (mU/mL), iron (ng/mL), ferritin (ng/mL), 25-OH vitamin D (ng/mL), vitamin B12 (pg/mL)].

The insulin resistance status in patients was determined using homeostatic model assessment of insulin resistance (HOMA-IR) calculated by the formula: fasting insulin (uIU/mL) × fasting blood glucose (mg/dL) / 405. HOMA-IR reference values for Turkish children and adolescents (5-18 years); prepubertal period, ≥ 2.67 in males and ≥ 2.22 in females; and in the pubertal period, ≥ 5.22 in males and ≥ 3.82 in females were used for insulin resistance definition (16). In order to determine Vitamin D status of patients, global consensus recommendations was used. According to that, Vitamin D levels were classified into three groups as follows: < 12 ng/mL indicating deficiency, 12-20 ng/mL indicating insufficiency, and 20 ng/mL indicating normal (17).

Height and weight measurements of the children were obtained from the patient folders by the researcher and recorded in the study form. The body mass index (BMI) was calculated using the formula "body weight (kg) / [height (m)]$^2$". BMI, weight and height values were evaluated according to WHO Anthro plus programme developed by Multicentre Growth Reference Study (MGRS) Child Growth Standards for 5-19 years old (18).

**Statistical analysis**

The data was analyzed using the IBM SPSS Statistics 25.0 program. Continuous variables were expressed as mean ($\bar{x}$) and standard deviation (SD), while categorical variables were presented as frequency (*n*) and percentage ( %). The relationship between groups was analyzed using the chi-square test. For data with a normal distribution, Student's t-test was used to analyze the significance between two groups, while ANOVA test was employed for analyzing

significance among more than two groups. A statistical significance level of $p < 0.05$ was used (19).

## RESULTS

The demographic, biochemical and anthropometric distributions according to puberty are presented in table I.

Table II presents a comparison of HOMA-IR and BMI for age across different serum 25-OH Vitamin D status groups. In prepubertal children, there was a significant difference in HOMA-IR values among the groups with progressive levels of serum 25-OH vitamin D ($p = 0.04$). Specially, children with vitamin D deficiency exhibited higher HOMA-IR values compared to those with insufficient or normal vitamin D levels. Moreover, among obese children, there was a significant assosiation between serum 25-OH vitamin D levels and BMI for age ($p = 0.015$).

Table III presents comparison of some biochemical results according to puberty. There was notable differences between prepubertal and pubertal children in terms of fasting insulin levels, HOMA-IR, total cholesterol, HDL-C levels. Insulin levels during fasting were significantly higher in pubertal children ($p = 0.000$). HOMA-IR values were significantly higher in pubertal children compared to prepubertal ones ($p = 0.000$). There was a significant difference in total cholesterol levels between prepubertal and pubertal groups ($p < 0.05$). HDL cholesterol levels were significantly lower in pubertal children ($p < 0.05$). Except for these values, no significant differences were found in any blood parameters between prepubertal and pubertal groups ($p > 0.05$).

The correlations between serum 25-OH Vitamin D levels and various parameters are presented in table IV. There was weak but significant negative correlation between serum 25-OH vitamin D levels and HOMA-IR ($r = -0.219$, $p = 0.022$). There was a weak negative correlation between serum 25-OH vitamin D levels and BMI for age ($r = -0.237$, $p = 0.050$). There was a moderate and significant positive

correlation between serum 25-OH vitamin D levels and HDL cholesterol (r = -0.106, $p$ = 0.040). Except for these values, no significant differences were found in any blood parameters between 25-OH D vitamin levels ($p > 0.05$).

## DISCUSSION

Obesity has become a global health concern affecting individuals across all groups, with significant implications for public health. Childhood obesity, in particular, has increased attention due to its rising prevalence and associated health risks (20).

The findings of our study highligt the significant burden of obesity among children and adolescents. Consistent with other research, our results indicate that childhood obesity is associated with a higher prevalence of insulin resistance, which is key precursor to type 2 diabetes and metabolic syndrome (21-24). Furthermore, our study revealed a notable difference in insulin resistance status between prepubertal and pubertal individuals, with a higher proportion of pubertal participants exhibiting insulin resistance. This underscores the importance of addressing obesity-relates health concerns early in life to prevent the onset of metabolic complications (25).

In the present study, as vitamin D levels decrease, BMI for age tends to increase, indicating a significant relationship between vitamin D deficiency and overweight. Vitamin D insufficiency, which is common in overweight and obese children, was found to be 67.4 % in our study conducted in Turkey. The prevalence of vitamin D insufficiency among children and adolescents with obesity is extremely high: 96.0 % in Germany, 78.4 % in the United States, and up to 92.0 % in the Russian Federation (26). Regarding vitamin D status, the data indicate a concerning prevalence of deficiency and insufficiency among both prepubertal and pubertal individuals. This underscores

the importance of addressing vitamin D status in children and adolescents, especially during critical periods of growth and development. Our findings are consistent with a meta-analysis involving 24,600 children and adolescents aged 0-18 years, the relative risk of vitamin D deficiency in pediatric obesity was found to be 1.41 (14). Moreover, our study revealed a significant association between serum 25-OH vitamin D levels and BMI for age, particularly among obese children.

As serum vitamin D levels decreased, BMI for age tended to increase, suggesting a potential link between Vitamin D deficiency and excess weight. This finding aligns with existing literature highlighting the relationship between vitamin D deficiency and obesity. B Greene-Finestone et al. (27) reported an association between low serum 25 (OH) D vitamin levels and adiposity in obese children and adolescents in their study conducted in Canada. The predominant physiological mechanism underlying the association between childhood obesity and hypovitaminosis D is the increased absorption of vitamin D by adipose tissue due to its fat-soluble nature. Additionally, the correlation between excessive screen time and lower vitamin D levels suggests a multifactorial relationship, which may also be influenced by reduced outdoor activity and sunlight exposure (28). The retrospective design of the study resulted in certain limitations. Factors such as exposure to sunlight, dietary habits, and clothing style, which may potentially influence the vitamin D levels of the included children and adolescents, were not evaluated. This is one of the limitations of the study. Understanding whether vitamin D deficiency is a result or a cause of obesity is difficult. Published findings suggest that periodic monitoring of vitamin D concentration in overweight and obese children and adolescents, as well as considering vitamin D supplementation in those with deficiency or insufficiency of vitamin D, may be effective. Larger-scale prospective randomized trials are needed to obtain clearer results (29).

Pires et al. (30) reported prepubertal children with overweight/obesity that became IR during puberty showed a significant decrease in 25(OH)D over time. An association between inadequate vitamin D levels and insulin sensitivity and β-cell function has been observed in a sample of overweight/obese children and adolescents (31). Our study also investigated the association between serum 25-OH vitamin D levels and insulin resistance in children and adolescents with obesity. We found a significant negative correlation between serum vitamin D levels and HOMA-IR, indicating that higher vitamin D levels are associated with lower insulin resistance. In a study conducted on obese children aged 10-16 years in Iran to investigate the effect of oral vitamin D supplementation on insulin resistance (IR), 21 individuals receiving 300,000 IU of oral vitamin D were compared to 22 individuals receiving placebo. Significant reductions in serum insulin levels and HOMA-IR index were observed after 12 weeks in the group receiving oral vitamin D compared to the placebo group (32). This finding is consistent with previous research suggesting a potential role for Vitamin D in modulating insulin sensitivity (28,32). Correlations between serum vitamin D levels and BMI for age among children ($p$ = 0.015), highlighting a notable link between vitamin D deficiency and excess weight in our study. Nevertheless, obese children and adolescents with both vitamin D deficiency and impaired glucose metabolism could respond differently to vitamin D supplementation remains unclear and warrants further studies (31). Although the relationship between obesity, insulin resistance, and vitamin D has been a common topic in many studies, there is still a lack of sufficient research on this subject. To gain clarity, there is a need for prospective, long-term studies (30).

In conclusion, our study provides further evidence of the complex interplay between obesity, vitamin D status, and metabolic health in children and adolescents. Future research should focus on elucidating the underlying mechanisms linking vitamin D deficiency to insulin resistance and obesity, as well as evaluating the effectiveness of

vitamin D supplementation as a potential intervention strategy in this population.

## REFERENCES


1. Weiss R, Caprio S. The metabolic consequences of childhood obesity. Best Pract Res Clin Endocrinol Metab 2005;19(3):405-19. DOI: 10.1016/j.beem.2005.04.009
2. Chiarelli F, Marcovecchio ML. Insulin resistance and obesity in childhood. Eur J Endocrinol 2008;159(Suppl 1):S67-74. DOI: 10.1530/EJE-08-0245
3. Lister NB, Baur LA, Felix JF, Hill AJ, Marcus C, Reinehr T, et al. Child and adolescent obesity. Nat Rev Dis Primers 2023;9(1):24. DOI: 10.1038/s41572-023-00435-4
4. Valerio G, Licenziati MR, Iannuzzi A, Franzese A, Siani P, Riccardi G, et al. Insulin resistance and impaired glucose tolerance in obese children and adolescents from Southern Italy. Nutr Metab Cardiovasc Dis 2006;16(4):279-84. DOI: 10.1016/j.numecd.2005.12.007
5. Tagi VM, Giannini C, Chiarelli F. Insulin resistance in children. Frontiers in endocrinology 2019;10:342.
6. Keskin M, Kurtoglu S, Kendirci M, Atabek ME, Yazici C. Homeostasis model assessment is more reliable than the fasting glucose/insulin ratio and quantitative insulin sensitivity check index for assessing insulin resistance among obese children and adolescents. Pediatrics 2005;115(4):e500-3. DOI: 10.1542/peds.2004-1921
7. Saglam D, Samur G, Turan S. Assessment of vitamin D status in Turkish adolescents: its relation to obesity, cardiometabolic risk factors and nutritional status. Prog Nutr 2019;21(4):762-8.
8. Szymczak-Pajor I, Śliwińska A. Analysis of association between vitamin D deficiency and insulin resistance. Nutrients 2019;11(4):794. DOI: 10.3390/nu11040794

9. Chiarelli F, Marcovecchio ML. Insulin resistance and obesity in childhood. Eur Jendocrinol 2008;159(1):S67-74. DOI: 10.1530/EJE-08-0245
10. Maffeis C, Morandi A. Body composition and insulin resistance in children. Eur J ClinNutr 2018;72(9):1239-45. DOI: 10.1038/s41430-018-0239-2
11. Savastano S, Barrea L, Savanelli MC, Nappi F, Di Somma C, Orio F, et al. Low vitamin D status and obesity: Role of nutritionist. Rev Endocr Metab Disord 2017;18(2):215-25. DOI: 10.1007/s11154-017-9410-7
12. Pereira-Santos CP, Santos C, Santos D. Obesity and vitamin D deficiency: Asystematic review and meta-analysis. Obes Rev 2015;16(4):341-9. DOI: 10.1111/obr.12239
13. Cândido FG, Bressan J. Vitamin D: link between osteoporosis, obesity, and diabetes? Int J Mol Sci 2014;15(4):6569-91. DOI: 10.3390/ijms15046569
14. Fiamenghi VI, Mello ED. Vitamin D deficiency in children and adolescents with obesity: a meta-analysis. Jornal de Pediatria 2021;97(3):273-9. DOI: 10.1016/j.jped.2020.08.006
15. World Health Organisation. Growth Reference Data for 5-19 years BMI for age (5-19 years) [Access date: February 10, 2024]. Available from https://www.who.int/tools/growth-reference-data-for-5to19-years/indicators/bmi-for-age
16. 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(3):100-6. DOI: 10.4274/jcrpe.v2i3.100
17. Munns CF, Shaw N, Kiely M, Specker BL, Thacher TD, Ozono K, et al. Global Consensus Recommendations on Prevention and Management of Nutritional Rickets. Horm Res Paediatr 2016;85(2):83-106. DOI: 10.1159/000443136

18. WHO AnthroPlus for personal computers Manual: Software for assessing growth of the world's children and adolescents. Geneva: WHO, 2009.
19. Tao S, Yuan Q, Mao L, Chen FL, Ji F, Cui ZH. Vitamin D deficiency causes insulin resistance by provoking oxidative stress in hepatocytes. Oncotarget 2017;8(40):67605-13. DOI: 10.18632/oncotarget.18754
20. Kansra AR, Lakkunarajah S, Jay MS. Childhood and Adolescent Obesity: A Review. Front Pediatr 2021;8:581461. DOI: 10.3389/fped.2020.581461
21. Yuca SA, Yılmaz C, Cesur Y, Doğan M, Kaya A, Başaranoğlu M. Prevalence of overweight and obesity in children and adolescents in eastern Turkey. J Clin Res Pediatr Endocrinol 2010;2(4):159-63. DOI: 10.4274/jcrpe.v2i4.159
22. Whiting S, Buoncristiano M, Gelius P, Abu-Omar K, Pattison M, Hyska J, et al. Physical Activity, Screen Time, and Sleep Duration of Children Aged 6-9 Years in 25 Countries: An Analysis within the WHO European Childhood Obesity Surveillance Initiative (COSI) 2015-2017. Obes Facts 2021;14(1):32-44. DOI: 10.1159/000511263
23. Gupta N, Goel K, Shah P, Misra A. Childhood obesity in developing countries: epidemiology, determinants, and prevention. Endocr Rev 2012;33(1):48-70. DOI: 10.1210/er.2010-0028
24. Niu Y, Tang Q, Zhao X, Zhao X, Mao X, Sheng J, et al. Obesity-Induced Insulin Resistance Is Mediated by High Uric Acid in Obese Children and Adolescents. Front Endocrinol (Lausanne) 2021;12:773820. DOI: 10.3389/fendo.2021.773820
25. Drozdz D, Alvarez-Pitti J, Wójcik M, Borghi C, Gabbianelli R, Mazur A, et al. Obesity and Cardiometabolic Risk Factors: From Childhood to Adulthood. Nutrients 2021;13(11):4176. DOI: 10.3390/nu13114176

26. Zakharova I, Klimov L, Kuryaninova V, Nikitina I, Malyavskaya S, Dolbnya S, et al. Vitamin D Insufficiency in Overweight and Obese Children and Adolescents. Front Endocrinol (Lausanne) 2019;10:103. DOI: 10.3389/fendo.2019.00103
27. Greene-Finestone LS, Garriguet D, Brooks S, Langlois K, Whiting SJ. Overweight and obesity are associated with lower vitamin D status in Canadian children and adolescents. Paediatr Child Health 2017;22(8):438-44. DOI: 10.1093/pch/pxx116
28. Fiamenghi VI, Mello ED. Vitamin D deficiency in children and adolescents with obesity: a meta-analysis. J Pediatr (Rio J) 2021;97(3):273-9. DOI: 10.1016/j.jped.2020.08.006
29. Calcaterra V, Verduci E, Vandoni M, Rossi V, Fiore G, Massini G, et al. The Effect of Healthy Lifestyle Strategies on the Management of Insulin Resistance in Children and Adolescents with Obesity: A Narrative Review. Nutrients 2022;14(21):4692. DOI: 10.3390/nu14214692
30. Pires LV, González-Gil EM, Anguita-Ruiz A, Bueno G, Gil-Campos M, Vázquez-Cobela R, et al. The Vitamin D Decrease in Children with Obesity Is Associated with the Development of Insulin Resistance during Puberty: The PUBMEP Study. Nutrients 2021;13(12):4488. DOI: 10.3390/nu13124488
31. Javed A, Vella A, Balagopal PB, Fischer PR, Weaver AL, Piccinini F, et al. Cholecalciferol supplementation does not influence β-cell function and insulin action in obese adolescents: a prospective double-blind randomized trial. J Nutr 2015;145(2):284-90. DOI: 10.3945/jn.114.202010
32. Kelishadi R, Salek S, Salek M, Hashemipour M, Movahedian M. Effects of vitamin D supplementation on insulin resistance and cardiometabolic risk factors in children with metabolic syndrome: a triple-masked controlled trial. J Pediatr (Rio J) 2014;90(1):28-34. DOI: 10.1016/j.jped.2013.06.006

Table I. Demographic, biochemical and anthropometric distributions according to pubertal status

| | **Prepubertal (*n* = 114)** | | **Pubertal (*n* = 304)** | |
|---|---|---|---|---|
| | ***n*** | **%** | ***n*** | **%** |
| *Gender* | | | | |
| Boys | 70 | 61.4 | 136 | 44.7 |
| Girls | 44 | 38.6 | 168 | 55.3 |
| *Age (years)* | | | | |
| 6-9 | 84 | 73.6 | 52 | 17.1 |
| 10-17 | 30 | 26.4 | 252 | 82.9 |
| (X ± SS) | 11.94 ± 3.32 | | | |
| *Vitamin D status* | | | | |
| Deficient | 30 | 26.3 | 80 | 26.3 |
| Insufficiency | 50 | 43.9 | 156 | 51.3 |
| Normal | 34 | 29.8 | 68 | 22.4 |
| *IR status* | | | | |
| İR + | 42 | 36.8 | 78 | 25.7 |
| İR - | 72 | 63.2 | 226 | 74.3 |
| *BMI for age* | | | | |
| Overweight | 22 | 19.3 | 72 | 23.7 |
| Obese | 92 | 80.7 | 232 | 76.2 |

IR: insulin resistance; BMI: body mass index.

Table II. Comparison of HOMA-IR and BMI for age according to serum 25-OH-vitamin D status and correlations of 25-OH-vitamin D with some parameters

| | **25-OH Vitamin D status** | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | **Deficiency < 12 ng/mL (*n* = 110)** | | **Insufficiency 12-20 ng/mL (*n* = 206)** | | **Normal > 20 ng/mL (*n* = 102)** | | | |
| | **$\bar{X}$** | **SD** | **$\bar{X}$** | **SD** | **$\bar{X}$** | **SD** | ***p*** | |
| *HOMA-IR* | | | | | | | | |
| Prepubertty | 1.92[a] | 0.97 | 1.75[b] | 0.82 | 1.70[b] | 0.47 | *0.041* | |
| Puberty | 3.60[a] | 2.07 | 2.70[b] | 1.49 | 2.16[c] | 1.11 | *0.002* | |
| *BMI for age* | | | | | | | | |
| Overweight | 1.79[a] | 0.35 | 1.64[a] | 0.62 | 1.26[b] | 0.86 | *0.030* | |
| Obese | 2.55[b] | 1.24 | 2.41[b] | 1.13 | 2.28[c] | 1.14 | *0.015* | |
| | | | *25-OH Vitamin D* | | | | | |
| | | | *r* | *p* | | | | |
| HOMA-IR | | | -0.219 | *0.022* | | | | |
| Age | | | 0.043 | 0.537 | | | | |
| BMI for age | | | -0.237 | *0.039* | | | | |
| Fasting glucose | | | -0.08 | *0.028* | | | | |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | 6 | | | | | |
| Fasting insulin | | -0.115 | *0.043* | | | | |
| Total cholesterol | | -0.090 | 0.256 | | | | |
| LDL-cholesterol | | -0.106 | 0.163 | | | | |
| HDL-cholesterol | | 0.563 | *0.040* | | | | |
| ALT | | -0.019 | 0.797 | | | | |
| TSH | | 0.107 | 0.174 | | | | |
| Vitamin $B_{12}$ | | 0.022 | 0.811 | | | | |
| Ferritin | | 0.143 | 0.259 | | | | |

Different letters indicate differences according to post-hoc statistical analysis; $p < 0.05$.

Table III. Comparison of some biochemical results according to pubertal status

| | **Prepubertal (*n* = 114)** | | **Pubertal (*n* = 304)** | | ***p*** |
|---|---|---|---|---|---|
| | **$\bar{X}$** | **SD** | **$\bar{X}$** | **SD** | |
| Fasting glucose (mg/dL) | 88.47 | 6.19 | 87.0 | 6.46 | 0.153 |
| Fasting insulin (uIU/mL) | 8.44 | 3.53 | 13.42 | 7.94 | *0.000** |
| HOMA-IR | 1.83 | 0.82 | 2.92 | 1.81 | *0.000** |
| Total cholesterol (mg/dL) | 182.25 | 44.93 | 163.82 | 38.48 | *0.049** |
| LDL-cholesterol (mg/dL) | 112.87 | 39.88 | 104.89 | 32.10 | 0.171 |
| HDL-cholesterol (mg/dL) | 57.15 | 25.53 | 49.87 | 11.38 | *0.020** |
| ALT (U/L) | 19.24 | 6.27 | 22.14 | 13.85 | 0.056 |
| TSH (mU/ml) | 2.21 | 0.71 | 2.25 | 1.19 | 0.806 |
| 25-OH Vitamin D (ng/mL) | 17.69 | 6.70 | 17.08 | 7.24 | 0.586 |
| Vitamin $B_{12}$ (pg/mL) | 488.44 | 173.89 | 434.83 | 150.77 | 0.130 |
| Ferritin (ng/mL) | 40.50 | 18.26 | 35.93 | 19.47 | 0.361 |

*$p < 0.05$.