

Dietary Habits and Early Pubertal Development in Girls: A Nutritional Study from the Kurdistan Region of Iraq

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ABSTRACT

Background: Precocious puberty, characterised by the onset of secondary sexual characteristics before the age of 8 years in girls, is becoming increasingly prevalent globally. To explore this issue, the present study investigated the link between dietary patterns, body composition, and puberty among 6–9-year-old girls in Erbil, Iraq.

Aim: This study aimed to investigate the association between nutritional status and precocious puberty.

Study design: The study included 100 girls aged 6–9 years living in Erbil, who were recruited from various sources. Of these, 50 were diagnosed with precocious puberty and 50 were age-matched controls. A structured questionnaire was used to assess the participants' health status and dietary habits.

Results: No significant age differences were observed between the case and control groups. Factors such as family history and mode of childbirth were also significant. Several food groups, particularly snacks, desserts, fried foods, and eggs, significantly influenced precocious puberty.

Conclusion: Dietary habits and health status, including mode of childbirth, family history, medication condition, and consumption of snacks, desserts, and eggs, are crucial factors influencing pubertal development.

Keywords: Dietary Habits, Precocious Pubertal Development, Nutritional Status, Childhood Obesity, Kurdistan Region.

I. INTRODUCTION

Puberty is complex with physical and hormonal changes occurring at a rapid pace, bringing about sexual maturation and the potential for reproduction (1). While the average pubertal age has fallen, increasing concerns about health implications, especially in the case of girls, have arisen associated with early puberty and subsequent cancer, cardiovascular disease, obesity and psychosocial issues (2, 3). Eating habits have changed toward the consumption of Westernised diets, which are composed of convenience foods, sugar-rich refined products and unhealthy fats, which have affected pubertal timing as a key point for public health strategies (4, 5). Evidence suggests that nutrition plays an important role in pubertal timing (6). High intake of energy-dense foods, sugar-sweetened beverages and animal protein sources promotes earlier pubertal timing, whereas a high-quality diet characterised by a variety of fruit, vegetables and whole grains as well as healthy fats delays sexual maturation (7). There is evidence associating early puberty onset with increased intake of milk and dairy foods or red meat, perhaps through hormonal stimulation (8). Childhood obesity and the endocrine-disrupting chemicals in food packaging and in pesticides can have implications for puberty timing (9). However, little is known about the neuroendocrinological role of these receptors in the HPG axis and the onset of puberty (10).

While there is increasing evidence about the diet and pubertal timing, comprehensive data are still lacking in the Kurdistan region of Iraq. The region's unique dietary practices, cultural influences, and environmental conditions may limit the applicability of existing findings, making it challenging to design targeted public health interventions and nutritional guidelines.

The present study seeks to investigate the association between dietary factors and early puberty in the Kurdistan region of Iraq among children and adolescents. It will measure present dietary intake, age at mean pubertal

onset, and trends toward a more precocious development. The study also explores associations between the intake of different groups of food and pubertal onset, determining particular dietary factors that contribute to earlier or later onset of puberty among this population.

II. MATERIALS AND METHODS

This was a case-control study conducted in Erbil, Iraq, from December 2024 to June 2025. The participants were recruited from diverse places such as primary schools, a pediatric outpatient clinic, and a local teaching hospital. Four techniques, purposive sampling and convenience sampling from each group, were applied to a study among 100 girls aged between 6-9 years in Erbil. A structured questionnaire for data collection was completed through individual interviews with parents of participants. The survey contained questions concerning the child's demographic data and health status, as well as dietary aspects such as favorite food and eating behaviors. The analysis provided an opportunity to examine whether precocious puberty was related to dietary factors by seeking comprehensive data.

III. ETHICAL CONSIDERATIONS

The Ethics Committee of the College of Science at Salahaddin University in Erbil, Iraq, granted approval for this study (Approval No. 45/433, dated 24/12/2024). All procedures were conducted in accordance with the ethical standards and relevant guidelines established by the committee. Prior to participation, written informed consent was obtained from the parents of the young participants, indicating their understanding of the study's purpose and their voluntary endorsement of their children's involvement in the research.

IV. DATA ANALYSIS

Data were analyzed using GraphPad Prism version 10, using both descriptive and inferential statistical techniques. Associations

between variables were evaluated using the chi-square test and the unpaired t-test, with a

p-value of <0.05 considered statistically significant

V. RESULT

TABLE I

FAMILY AND MEDICAL HISTORY OF THE 2 GROUPS

Clinical Characteristics		Case Group (N = 50)	Control Group (N = 50)	P Value
Age of the child		7.920 ± 0.098	7.680 ± 0.096	0.084
Childbirth By	Normal vaginal delivery	21(42.00%)	10 (20.00%)	0.02
	Cesarean section	29 (58.00%)	40 (80.00%)	
Family History	Yes	22 (44.00%)	10 (20.00%)	0.01
	No	28 (54.00%)	40 80.00%)	
Medical Condition	Yes	24 (48.00%)	12 (24.00%)	0.01
	No	26 (52.00%)	38 (76.00%)	
Disease	Yes	3 (6.00%)	6 (12.00%)	0.294
	No	47 (94.00%)	44 (88.00%)	
Father's Smoke	Yes	25 (50.00%)	23 (46.00%)	0.841
	No	25 (50.00%)	27 (54.00%)	
Mother's Smoke	Yes	1 (2.00%)	1 (2.00%)	>0.999
	No	49 (98.00%)	49 (98.00%)	
Values are presented as mean ± standard error (SE) for continuous variables and as frequency (percentage) for categorical variables. P > 0.05: Non-significant; P < 0.05: Significant.				

Table I The study is based on a total of 100 participants aged 6 to 9 years, the mean age was 7.9 ± 0.098 years according to SEM (Standard Error of the Mean). This indicates a homogenous sample in terms of age distribution. The participants were divided into two groups i.e. caseload and control and both groups were compared at the baseline, and the statistical results does not show any significant difference in age between both of the groups ($p = 0.084$).

The results illustrated in table 1 highlight the factors that were significantly associated with the family and medical history in which precocious puberty was based on family history and medical condition ($p=0.01$), and mode of delivery, or whether the mode of delivery was cesarean or not (normal vaginal delivery) ($p=0.02$). The presence of child diseases ($p=0.294$), father's smoking ($p=0.841$), and mother's smoking ($p>0.999$). All these results did not demonstrate a significant association.

Table II

Nutritional Childhood

Characteristics	Case Group (N = 50)	Control Group (N = 50)	P Values
Number Of Meals	2.540 ± 0.099	2.840 ± 0.125	0.064
Number Of Snacks	2.500 ± 0.091	2.100 ± 0.107	0.005
Dairy product	None	7 (14.00%)	0.783
	2--3 times per month	6 (12.00%)	
	1 time per week	3 (6.00%)	
	2-3 times per week	14 (28.00%)	
	once per day	17 (36.00%)	
	Twice per day	3 (6.00%)	
Nuts	None	6 (12.00%)	0.060
	2--3 Times Per Month	8 (16.00%)	
	1 Time Per Week	15 (30.00%)	
	2-3 Times Per Week	14 (28.00%)	
	Once Per Day	7 (14.00%)	
Dessert	2--3 Times Per Month	2 (4.00%)	0.02
	1 Time Per Week	1 (2.00%)	
	2-3 Times Per Week	9 (18.00%)	
	Once Per Day	28 (56.00%)	
	Twice Per Day	10 (16.00%)	
Egg	None	6 (12.00%)	0.04
	2--3 Times Per Month	1 (2.00%)	
	1 Time Per Week	7 (14.00%)	
	2-3 Times Per Week	24 (48.00%)	
	Once Per Day	12 (24.00%)	
Fried Food	None	1 (2.00%)	0.01
	2--3 Times Per Month	13 (26.00%)	
	One Per Week	18 (36.00%)	
	2-3 Times Per Week	10 (20.00%)	
	Once Per Day	7 (14.00%)	
	Twice Per Day	1(2.00%)	

Values are presented as mean ± standard error (SE) for continuous variables and as frequency (percentage) for categorical variables.
P > 0.05: Non-significant; P < 0.05: Significant.

Table II. The statistically significant correlations found between the consumption of some food groups with precocious puberty are presented in Table II. The strongest correlations were found with the consumption of desserts ($p = 0.02$), fried food ($p = 0.01$), and eggs ($p = 0.04$). Overall, the findings robustly support the conclusion that the

dietary patterns are important in the timing of puberty.

Furthermore, there were no dietary factors analyzed that were associated with the results obtained. The p-values (for dairy products, $p = 0.783$; for nuts, $p = 0.060$) indicated no statistically significant results. They also barely reached the customary 0.05 threshold of

significance for number of meals ($p=0.064$) and were therefore non-significant.

VI. DISCUSSION

The study was a comparison of normal cesarean deliveries and vaginal delivery between girls aged 6-9 years, with increased risks of precocious puberty in children born via normal delivery as opposed to children born via cesarean section. One possible explanation is that vaginally delivered infants are exposed to maternal vaginal microbiota, which influences early immune and metabolic programming. Differences in gut microbiota colonisation between vaginal and cesarean births may affect hormone metabolism, which can lead to variations in pubertal onset. Egyptian research The natal history revealed non-significant difference, in the type of delivery between the studied group (11). In a sweeping Danish study involving over 15,000 mother-daughter pairs, it was discovered that neither the urgency of emergency nor the calm precision of elective caesarean sections cast any shadow on the early bloom of puberty in young girls (12). The results indicated that cesarean birth decreased the risk of hormonal development because of lifestyle, maternal characteristics, and postnatal care (13). The study discovered that there is a strong association between family history and precocious puberty in girls (14). This can be explained by genetic factors regulating the hypothalamic-pituitary-gonadal axis. Mutations in genes such as MKRN3 or KISS1 have been seen to affect familial central precocious puberty, showing a strong hereditary component to pubertal timing. Phytoestrogen-containing supplements. Plant derived oestrogens (phytoestrogens) like genistein and daidzein from soy and other plants have weak oestrogenic activity by modulating the oestrogen receptor. High serum isoflavone levels are associated with CPP and Korean girls whose levels fall in the highest quartile of serum concentrations are fourfold more likely to experience CPP than

those in the lower quartile (15). rhGH therapy. rhGH is commonly prescribed for idiopathic short stature (ISS) and growth hormone deficiency. Through a systematic review and meta-analysis, pubertal timing was earlier, 0.46 year, and slightly increased the risk of pubertal onset (risk ratio 1.26) in rhGH-treated vs. untreated ISS children (16). The conclusion was that no significant association was found between parental smoking and precocious puberty, which could be an indication of other environmental or genetic factors. Exposure to secondhand smoke at home was linked to early age at menarche in girls in South Korea (17). Cigarette use by mothers during pregnancy has also been found to be associated with both sons' and daughters' development of precocious puberty, with earlier development by 1-4.5 months per 10 cigarettes/day (18).

The study found no significant link between main meals and precocious puberty; however, a higher number of daily snacks increased the risk of early pubertal onset. Frequent snacking usually involves energy-dense, high-sugar foods, which help increase insulin and IGF-1 levels. These hormones can quicken activation of the hypothalamic-pituitary-gonadal axis, leading to earlier pubertal development. A Turkish pilot study reported that 84% of girls with precocious puberty consumed three main meals and three snacks daily, a pattern that may contribute to obesity (19). Similarly, a study in China identified frequent snack consumption as a significant risk factor for increased rates of female advanced puberty (20). Evidence on dairy intake, however, remains inconsistent. Dairy foods may contribute to earlier puberty because they contain bioactive hormones (such as estrogens and insulin-like growth factor) and growth-promoting nutrients. However, variations in

processing, fat content, and portion size may explain inconsistent associations across studies. While one study found no association between dairy consumption and precocious puberty, a review and meta-analysis show that bioactive hormones and growth factors in dairy could accelerate pubertal onset (21). Findings from large cohort studies also vary: in the U.S., milk consumption after age nine did not meaningfully influence early menarche, though low-fat milk was modestly linked to later pubertal onset (22). Another study reported a weak association between childhood milk intake and early menarche, with moderate milk consumption appearing protective and higher calcium intake associated with earlier onset (23). Additionally, regular consumption of desserts and sugary beverages has been linked to hormonal changes that may cause early puberty in females (24).

A study in China concluded that high consumption of sugar and desserts increases the risk of precocious puberty in children by increasing insulin resistance and the imbalance of other hormones (25). High egg consumption reduced the risk of precocious puberty in the control group, while nut consumption had no significant association

VII. CONCLUSION

This study revealed family history, health conditions, and diet in early life as predictive factors for the occurrence of precocious puberty in children. Therefore, the results of this study may have opened up a possibly important window of opportunity for lifestyle and dietary interventions in early life which could potentially prevent this.

VIII. CONFLICT OF INTEREST

The authors have nothing to disclose.

with early pubertal onset. In the Iranian study, results imply that dietary patterns with high intake of protein sources such as eggs and adherence to a healthy body size might influence the onset time of menarche among girls (26). Greater overall diet quality, which was associated with higher nut intake, was linked to delayed puberty onset in both sexes, after controlling for body fat (27). The intake of nuts was closely connected with gut microbiota and metabolites, which may impact hormonal activity, thus may play a role in the relationship between dietary factors and the risk of precocious puberty(28). Fried food-eating preteen girls are more likely to start puberty early. This could be due to trans fats and advanced glycation end products generated during frying that disturb endocrine balance Furthermore, fried food often harbors obesogenic contaminants, which drive over accumulation of adiposity, a compelling accelerating factor of pubertal timing. Studies have shown that a high level of fried foods can increase the risk of precocious puberty in children due to fat and endocrine disruptors in them(25). In a preliminary investigation, consumption of fried foods was related to precocious puberty in girls. Therefore, there is a potential association between a high-fat, high-energy-dense diet and early puberty (19).

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REFERENCES

1. Susman EJ, Dorn LD, Schiefelbein VL. Puberty, sexuality, and health. Handbook of psychology: Developmental psychology. 2003;6:295-324.
2. Finne E, Bucksch J, Lampert T, Kolip P. Age, puberty, body dissatisfaction, and physical activity decline in adolescents. Results of the German Health Interview and Examination Survey (KiGGS). International Journal of Behavioral Nutrition and Physical Activity. 2011;8(1):119.
3. Muñoz-Calvo MT, Argente J. Nutritional and Pubertal Disorders. Puberty from Bench to Clinic. 2015;29:153-73.
4. Dhir B, Singla N. Consumption pattern and health implications of convenience foods: A practical review. Curr J Appl Sci Technol. 2019;38:1-9.
5. Nier A, Brandt A, Conzelmann IB, Özel Y, Bergheim I. Non-alcoholic fatty liver disease in overweight children: role of fructose intake and dietary pattern. Nutrients. 2018;10(9):1329.
6. Duittoz A, Kenny D. Early and late determinants of puberty in ruminants and the role of nutrition. animal. 2023;17:100812.
7. Hart TL, Petersen KS, Kris-Etherton PM. Early nutrition and development of cardiovascular disease. Early Nutrition and Long-Term Health: Elsevier; 2022. p. 309-25.
8. Gu Q, Wu Y, Feng Z, Chai Y, Hou S, Yu Z, et al. Dietary pattern and precocious puberty risk in Chinese girls: a case-control study. Nutrition Journal. 2024;23(1):14.
9. Demir A, Aydin A, Büyükgebiz A. Thematic Review of Endocrine Disruptors and Their Role in Shaping Pubertal Timing. Children. 2025;12(1):93.
10. Smith K. Sex-Specific Mechanisms of Pubertal Stress-Induced Inhibition of the HPG Axis: Université d'Ottawa/University of Ottawa; 2024.
11. Mohsen R, El-Zohairy E, Hassan M, Fathy M, Magdi M, Atef S, et al. The Possible Association between Phthalates and Bisphenol A Exposure and Idiopathic Precocious Puberty in Egyptian Girls. Open-Access Maced J Med Sci. 2022 May 12; 10 (B): 1411-1418. 2022.
12. Huang K, Gaml-Sørensen A, Lunddorf LLH, Ernst A, Brix N, Olsen J, et al. Caesarean delivery and pubertal timing in boys and girls: A Danish population-based cohort study. Paediatric and Perinatal Epidemiology. 2022;36(1):104-12.
13. Bigambo FM, Wang D, Niu Q, Zhang M, Mzava SM, Wang Y, et al. The effect of environmental factors on precocious puberty in children: a case-control study. BMC pediatrics. 2023;23(1):207.
14. Durand A, Bashamboo A, McElreavey K, Brauner R. Familial early puberty: presentation and inheritance pattern in 139 families. BMC endocrine disorders. 2016;16:1-8.
15. Kim JiHye KJ, Kim ShinHye KS, Huh KyoUng HK, Kim YunJe KY, Joung HyoJee JH, Park MiJung PM. High serum isoflavone concentrations are associated with the risk of precocious puberty in Korean girls. 2011.
16. Olwi D, Day F, Ong K. Effect of growth hormone therapy on pubertal timing: systematic review and meta-analysis. Hormone Research in Paediatrics. 2024;97(1):1-10.
17. Kang S, Joo J, Jang S, Park E. Association of exposure to secondhand smoke at home with early age at menarche in South Korea. Public health. 2020;185:144-9.
18. Suutela M, Hero M, Kosola S, Miettinen PJ, Raivio T. Prenatal, newborn and childhood factors and the timing of puberty in boys and girls. Pediatric Research. 2024;96(3):799-804.
19. Koc N, YARDIMCI H, Arslan N, Ucakturk S. Nutritional habits and precocious puberty in girls: a pilot study. JOURNAL OF PEDIATRIC RESEARCH. 2018;5(4).
20. Qi H, Pu S, Zhai H. Clinical characteristics and risk factors of female precocious puberty. Medicine. 2025;104(6):e41483.

21. Rastad H, Shahrestanaki E, Heydarian HR, Maarefvand M. Dairy consumption and its association with anthropometric measurements, blood glucose status, insulin levels, and testosterone levels in women with polycystic ovary syndrome: a comprehensive systematic review and meta-analysis. *Frontiers in endocrinology*. 2024;15:1334496.
22. Carwile JL, Willett WC, Wang M, Rich-Edwards J, Frazier AL, Michels KB. Milk consumption after age 9 years does not predict age at menarche. *The journal of nutrition*. 2015;145(8):1900-8.
23. Wiley AS. Milk intake and total dairy consumption: associations with early menarche in NHANES 1999-2004. *PloS one*. 2011;6(2):e14685.
24. Carwile J, Willett W, Spiegelman D, Hertzmark E, Rich-Edwards J, Frazier A, et al. Sugar-sweetened beverage consumption and age at menarche in a prospective study of US girls. *Human Reproduction*. 2015;30(3):675-83.
25. Chen C, Chen Y, Zhang Y, Sun W, Jiang Y, Song Y, et al. Association between Dietary Patterns and Precocious Puberty in Children: A Population- Based Study. *International journal of endocrinology*. 2018;2018(1):4528704.
26. Dashtabi A, Kohansal A, Mirzaee A, Akhlaghi M. Age at menarche and its nutrition-related factors among school girls in Shiraz, Southern Iran. *International Journal of Nutrition Sciences*. 2018;3(3):133-8.
27. Duan R, Qiao T, Chen Y, Chen M, Xue H, Zhou X, et al. The overall diet quality in childhood is prospectively associated with the timing of puberty. *European Journal of Nutrition*. 2021;60(5):2423-34.
28. Wang Y, Jin C, Li H, Liang X, Zhao C, Wu N, et al. Gut microbiota-metabolite interactions mediate the effect of dietary patterns on precocious puberty. *Iscience*. 2024;27(6).