AJDR
Avicenna Journal of Dental Research
Avicenna J Dent Res, 2022; 14(1):10-13. doi:10.34172/ajdr.2022.02
http://ajdr.umsha.ac.ir

Original Article

Prevalence of Developmental Defects of Enamel Among Children With Down Syndrome in Damascus, Syria

Ramah Eimad Makieh[1*], Chaza Kouchaji[2], Saleh Al Kurdi[3]

[1]Damascus University, Damascus, Syria
[2]Head of Syrian Pedodontic Association, Professor Dr. Pedodontic Department, Damascus University, Damascus, Syria
[3]Ph.D. Resident in Pediatric Dentistry, Pediatric Dentistry Department, Damascus University, Damascus, Syria

Article history:
Received: 23 March 2021
Accepted: 27 July 2021
ePublished: 29 March 2022

***Corresponding author:**
Ramah Eimad Makieh, BDS., Preparing for Master's Degree in Pediatric Dentistry; Damascus University, Damascus, Syria.
Tel: +963991692667,
Email: rmakkieh@yahoo.com

Abstract

Background: Down syndrome (DS) is a disorder which has conditions that may contribute to the developmental defects of enamel (DDE) etiologies. The aim of this study was to assess the prevalence of DDE among children with DS.

Methods: This study cross-sectional observational study examined a total of 88 children with DS and 87 healthy children. A modified DDE index for screening surveys was employed in this regard. Demarcated opacities, diffuse opacities, dysplasia, and combinations between types were recorded, and finally, data were analyzed by chi-square and Mann-Whitney U tests using SPSS software.

Results: The prevalence of enamel defects in DS and control groups was obtained as 45% and 34%, respectively, with no statistically significant differences between the groups ($P=0.139$). The mean number of teeth with DDE was 2.48 ± 3.79 and 1.09 ± 2.11 in the DS and control groups, respectively. Based on the results, statistically significant differences were found between DDE means ($P=0.009$). Demarcated opacities were the most frequent type of enamel defects in both groups. Eventually, a statistically significant relationship was observed between diffuse opacities and DS ($P=0.000$).

Conclusions: In general, DS had no influence on DDE prevalence; however, it increases affected dental units. Diffuse opacities are more frequent among children with DS compared to healthy children. Further studies are required on the DDE prevalence in Syria.

Keywords: Down syndrome, Enamel defects, Children, Opacities

Please cite this article as follows: Makieh RE, Kouchaji C, Al Kurdi S.Prevalence of developmental defects of enamel among children with down syndrome in Damascus, Syria. Avicenna J Dent Res. 2022; 14(1):10-13. doi:10.34172/ajdr.2022.02

Background

Down syndrome (DS) is the most common genetic disorder with an incidence of 1 per 374 live births (1).

Children with DS have shown a high prevalence of dental anomalies containing hypodontia, shape anomalies, supernumerary teeth, and taurodontism (2). Further, they suffer from digestive anomalies, metabolic disorders, nutrient deficiencies, and intellectual disabilities (3,4).

The inadequate diet and digestive system dysfunctions cause vitamin and mineral deficiencies (vitamin A, C, and B groups, calcium, zinc, and the like) in these children (5,6).

Studies have reported that the above-mentioned conditions contribute to the occurrence of enamel defects (7-9).

Moreover, some studies found an association between mental retardation and enamel defects as a result of the correlation between the development of enamel and the development of the brain (10,11).

Dental enamel is a unique non-regenerative tissue and, changes during its formation are permanent (12). Enamel formation is classified into three extensive stages, namely, matrix formation, calcification, and maturation (13).

Generally, enamel hypoplasia is the quantitative reduction of the enamel resulting from alternations in the matrix formation stage, while opacities result from alternations in calcification or maturation stages (14,15). Previous processes are affected by genetic and environmental factors.

Accordingly, DS and developmental defects of enamel (DDE) have multiple factors in common. Unfortunately, there is little information about the oral health of people with DS in Syria.

To our knowledge, no study has so far assessed the prevalence of DDE among children with DS in particular.

In addition, DDE are responsible for many clinical problems, including tooth sensitivity, aesthetic impairment, and increased risk of dental caries (16). Investigating DDE in those children can improve our understanding of potential etiologies and aid in the early implementation of adequate management.

Therefore, this study sought to assess the prevalence of

DDE in children with DS and compare the findings with matched healthy children.

Materials and Methods

This cross-sectional study (April-August 2019) included 88 individuals with DS aged 8-15 years from 5 special needs centers around Damascus and 87 matched healthy children.

Children in the DS group had confirmed diagnoses by specialists. Healthy children were selected from schools in the same district, were matched with DS children in terms of age and gender, and suffered from no chronic disease. Children with active infectious diseases or other health conditions needing extra precautions for their health were excluded from the study. Extremely uncooperative children and children undergoing orthodontic treatment were excluded as well.

A modified DDE index for screening surveys was employed to assess DDE (17). The World Health Organization's guidelines were taken into consideration in the methods used in this study (18).

To calibrate the examiner, photographs with all possible conditions for assessment were displayed twice on ten different days. Then, 15 patients with DDE were evaluated by another experienced examiner. After the study initiation, all examinations were performed by a single examiner.

Examinations were conducted under an artificial headlight using a plane mirror with the patient's head against the wall. The Tell-show-do technique was employed to facilitate the examination. Defects on primary and permanent teeth were recorded in this phase.

Sterile gauze was used to dry teeth and clean debris when necessary. Defects under 1 mm were ignored, and demarcated and diffused opacities, dysplasia, and combinations between types were recorded on a pre-designed form.

Demarcated opacities can be defined as an enamel defect that has clear borders with adjacent normal enamel. However, diffuse opacities have no clear borders with adjacent normal enamel and can appear in a linear, patchy, or continuous formation. In addition, hypoplasia is defined as a deficiency in the thickness or quantity of enamel and can appear as grooves, pits, or larger areas of missing enamel (19).

Data were analyzed using IBM SPSS Statistics, version 26. Mann-Whitney U and chi-square tests were implemented to discover the relationships between variables.

Results

A total of 88 children with DS and 87 healthy children were included in this study. The prevalence of enamel defects in the study group was 45%, while it was 34% in the control group with no statistically significant relationship between the groups ($P=0.139$, Table 1).

The DDE prevalence was higher in mixed dentition in the study and control groups (31% and 33%, respectively) compared to the permanent dentition stage in both groups (24% and 32%, respectively). Moreover, there was no significant relationship between the groups ($P=0.171$) in mixed and permanent dentitions ($P=0.692$, Table 2).

The mean number of teeth with DDE in the study group was 2.48 ± 3.79, which was higher than that of the control group (1.09 ± 2.11). There was a statistically significant relationship between the groups ($P=0.009$, Table 3).

The demarcated opacities were the most frequent type of DDE in the DS and control groups, and the percentage rate was 42% and 34%, respectively. However, the prevalence of diffuse opacities in the DS group was 22%, while that of the control group was only 2% ($P=0.000$, Table 4).

Discussion

The prevalence of DDE varies between countries due to environmental influences such as adverse events during early childhood (20,21). Malnutrition has been reported to have an effect on tooth formation, especially during

Table 1. Demographic Characteristics and Prevalence of DDE in the Sample

Variable	Study Group		Control Group		*P* Value
	No.	%	No.	%	
Gender					
Male	47	53	41	47	0.406[a]
Female	41	47	46	53	
Age (y)					
8-11	49	56	48	55	0.946[a]
12-15	39	44	39	45	
DDE					
None	48	55	57	66	0.139[a]
One defect or more	40	45	30	34	
Total	88	100.0	87	100.0	

Note. DDE: Developmental defects of enamel.
[a] Pearson chi-square test.

Table 2. Prevalence of DDE in Mixed and Permanent Dentition

Variable	Study Group		Control Group		*P* Value
	No.	%	No.	%	
Mixed dentition					
None	27	31	29	33	0.171[a]
One defect or more	29	33	18	21	
Permanent dentition					
None	21	24	28	32	0.692[a]
One defect or more	11	13	12	14	
Total	88	100.0	87	100.0	

Note. DDE: Developmental defects of enamel.
[a] Pearson chi-square test.

Table 3. Differences in the Mean Number of Teeth With DDE Between the Sample Groups

Variable	Study Group		Control Group		*P* Value
	Mean	SD	Mean	SD	
Number of teeth with DDE	2.48	3.79	1.09	2.11	0.009[a]

Note. SD: Standard deviation; DDE: Developmental defects of enamel.
[a] Mann-Whitney U test.

Table 4. Frequencies of the Developmental Defect of Enamel's Types in the Sample

DDE Type	Study Group		Control Group		*P* Value
	No.	%	No.	%	
Demarcated opacities					
No	51	58	57	66	0.303[a]
Yes	37	42	30	34	
Total	88	100.0	87	100.0	
Diffuse opacities					
No	69	78	85	98	0.000[a]
Yes	19	22	2	2	
Dysplasia					
No	84	96	85	98	0.414[a]
Yes	4	5	2	2	
Other defects					
No	87	99	87	100	0.319[a]
Yes	1	1	0	0	
Demarcated opacities and dysplasia					
No	86	98	87	100	0.157[a]
Yes	2	2	0	0	
Diffuse opacities and dysplasia					
No	86	98	87	100	0.157[a]
Yes	2	2	0	0	
Total	88	100.0	87	100.0	

Note. [a]Pearson Chi-square test.

early childhood (7,22). Socioeconomic status, pre- and peri-natal events, as well as maternal nutrition during pregnancy have also an effect in this regard (23,24).

Discovering relationships between DDE and possible etiologies supports understanding predispositions and allows taking into consideration early appropriate approaches. Previous studies reported that DDE can cause tooth sensitivity, early childhood caries, malocclusion, and aesthetic complications (20,25,26). Therefore, the current study aimed to evaluate DS correlation with DDE.

The DDE prevalence was 45% and 34% in the DS and control groups, respectively, with no statistically significant relationship between the groups. The previously mentioned findings are in line with those of Nogueira et al (27). They assessed DDE among cerebral palsy (CP) individuals and found no statistically significant relationship between CP and controls. Furthermore, they concluded that etiological factors in CP occurrence had no effect on DDE. In another study, Erika et al reported that intellectually disabled children had twice more DDE than the control group (11). Martínez et al also found statistically significant differences in the DDE prevalence among mentally retarded children with a history of bacterial diseases (28).

Al Habashneh et al indicated that the prevalence rate of hypoplasia was 8.73% (29). The higher prevalence rate in our study was due to the investigation of all DDE types. However, the higher prevalence rate of hypoplasia in the above-mentioned study can be due to the difference between study societies. In general, Robles et al found that the prevalence rate of DDE was 40.2% and 52% in primary and permanent dentitions, respectively (30).

Although DS as a disorder has conditions that contribute to the etiological factors of DDE, there is no evidence confirming that DDE occurred more often among DS. However, the number of DDE teeth was significantly higher in DS compared to matched healthy subjects. This leads us to believe that DS has no effect on the prevalence rate of DDE, but it increases affected dental units. Likewise, Moraes et al reported that individuals with DS had a variety of dental anomalies and often more than one anomaly in the same patient (2).

Our findings represented that the most frequent type of enamel defect was demarcated opacities in both groups. However, in the study of Lin et al, enamel hypoplasia was the most frequent type of enamel defect (31). On the other hand, our results conform to those of the study by Erika et al, indicating that the isolated demarcated opacities were the most recurrent type of DDE in intellectually disabled children (11).

Diffuse opacities are considered to be the characteristic of the DS group, although the tap water of most zones of Damascus has low fluoride concentration rates. The control group had a low prevalence of diffuse opacities, which were located in the same residential area of DS institutes. Familial habits and nutritional supplement intake may have a role in this respect. Nogueira et al concluded that the most prevalent DDE type among CP patients was diffuse opacities (27). Additionally, Gerreth et al suggested that numerous detected developmental defects in children with intellectual disability were caries-resistant which can be due to excess fluoride consumption during childhood as a result of poor control over the swallowing reflex (10).

Limitations of our study are being less interested in differences between white and yellow opacities than demarcated and diffuse opacities. However, differentiation between opacities' color is less important than differentiation between demarcated and diffuse opacities (32). The included DS children were selected only from special needs institutes because it is difficult to obtain DS subjects unless they are integrated into society.

We suggest promoting oral hygiene through educational programs aiming at educating parents or caregivers on oral hygiene practices and dietary counseling and encouraging them to regularly visit dental specialists.

Conclusions

The findings of this study revealed no relationship between children with DS and healthy children in terms of the prevalence of enamel defects, even though DS children had an increase in the mean number of teeth with enamel defects. Further studies are necessary on the DDE prevalence in Syria.

Acknowledgments

The authors would like to thank all the children who cooperated in the study and their families. The authors would also like to thank Dr. Muhammad Oweis Makieh for the careful revision of the manuscript.

Conflict of Interest Disclosures

The authors declare that they have no conflict of interests.

Ethical Statement

Ethical approval was obtained from Damascus University and the Ministry of Social Affairs and Labor. Written free and informed consent forms were obtained from children's parents or caregivers.

References

1. Aburawi EH, Nagelkerke N, Deeb A, Abdulla S, Abdulrazzaq YM. National growth charts for United Arab Emirates children with Down syndrome from birth to 15 years of age. J Epidemiol. 2015;25(1):20-9. doi: 10.2188/jea.JE20130081.
2. de Moraes ME, de Moraes LC, Dotto GN, Dotto PP, dos Santos LR. Dental anomalies in patients with Down syndrome. Braz Dent J. 2007;18(4):346-50. doi: 10.1590/s0103-64402007000400014.
3. Mircher C, Toulas J, Cieuta-Walti C, Marey I, Conte M, González Briceño L, et al. Anthropometric charts and congenital anomalies in newborns with Down syndrome. Am J Med Genet A. 2017;173(8):2166-75. doi: 10.1002/ajmg.a.38305.
4. Mazurek D, Wyka J. Down syndrome--genetic and nutritional aspects of accompanying disorders. Rocz Panstw Zakl Hig. 2015;66(3):189-94.
5. Samarkandy MM, Mohamed BA, Al-Hamdan AA. Nutritional assessment and obesity in Down syndrome children and their siblings in Saudi Arabia. Saudi Med J. 2012;33(11):1216-21.
6. Grammatikopoulou MG, Manai A, Tsigga M, Tsiligiroglou-Fachantidou A, Galli-Tsinopoulou A, Zakas A. Nutrient intake and anthropometry in children and adolescents with Down syndrome--a preliminary study. Dev Neurorehabil. 2008;11(4):260-7. doi: 10.1080/17518420802525526.
7. Pindborg JJ. Aetiology of developmental enamel defects not related to fluorosis. Int Dent J. 1982;32(2):123-34.
8. Sweeney EA, Saffir AJ, De Leon R. Linear hypoplasia of deciduous incisor teeth in malnourished children. Am J Clin Nutr. 1971;24(1):29-31. doi: 10.1093/ajcn/24.1.29.
9. Jontell M, Linde A. Nutritional aspects on tooth formation. World Rev Nutr Diet. 1986;48:114-36. doi: 10.1159/000413028.
10. Gerreth K, Opydo-Szymaczek J, Borysewicz-Lewicka M. A study of enamel defects and dental caries of permanent dentition in school children with intellectual disability. J Clin Med. 2020;9(4):1031. doi: 10.3390/jcm9041031.
11. Erika V, Modrić, Verzak Ž, Karlović Z. Developmental defects of enamel in children with intellectual disability. Acta Stomatol Croat. 2016;50(1):65-71. doi: 10.15644/asc50/1/9.
12. Sunderland EP, Smith CJ, Sunderland R. A histological study of the chronology of initial mineralization in the human deciduous dentition. Arch Oral Biol. 1987;32(3):167-74. doi: 10.1016/0003-9969(87)90130-0.
13. Steward RE. Pediatric Dentistry: Scientific Foundations and Clinical Practice. St. Louis: Mosby; 1982.
14. Suckling GW. Developmental defects of enamel--historical and present-day perspectives of their pathogenesis. Adv Dent Res. 1989;3(2):87-94. doi: 10.1177/08959374890030022901.
15. Seow WK. Clinical diagnosis of enamel defects: pitfalls and practical guidelines. Int Dent J. 1997;47(3):173-82. doi: 10.1002/j.1875-595x.1997.tb00783.x.
16. Seow WK. Developmental defects of enamel and dentine: challenges for basic science research and clinical management. Aust Dent J. 2014;59 Suppl 1:143-54. doi: 10.1111/adj.12104.
17. Clarkson J, O'Mullane D. A modified DDE Index for use in epidemiological studies of enamel defects. J Dent Res. 1989;68(3):445-50. doi: 10.1177/00220345890680030201.
18. World Health Organization (WHO). Oral Health Surveys: Basic Methods. 5th ed. Geneva, Switzerland: WHO; 2013.
19. Seow WK. Clinical diagnosis of enamel defects: pitfalls and practical guidelines. Int Dent J. 1997;47(3):173-82. doi: 10.1002/j.1875-595x.1997.tb00783.x.
20. Ibiyemi O, Zohoori FV, Valentine RA, Kometa S, Maguire A. Prevalence and extent of enamel defects in the permanent teeth of 8-year-old Nigerian children. Community Dent Oral Epidemiol. 2018;46(1):54-62. doi: 10.1111/cdoe.12328.
21. Li Y, Navia JM, Bian JY. Prevalence and distribution of developmental enamel defects in primary dentition of Chinese children 3-5 years old. Community Dent Oral Epidemiol. 1995;23(2):72-9. doi: 10.1111/j.1600-0528.1995.tb00204.x.
22. Infante PF, Gillespie GM. An epidemiologic study of linear enamel hypoplasia of deciduous anterior teeth in Guatemalan children. Arch Oral Biol. 1974;19(11):1055-61. doi: 10.1016/0003-9969(74)90095-8.
23. Costa FS, Silveira ER, Pinto GS, Nascimento GG, Thomson WM, Demarco FF. Developmental defects of enamel and dental caries in the primary dentition: a systematic review and meta-analysis. J Dent. 2017;60:1-7. doi: 10.1016/j.jdent.2017.03.006.
24. Enwonwu CO. Influence of socio-economic conditions on dental development in Nigerian children. Arch Oral Biol. 1973;18(1):95-107. doi: 10.1016/0003-9969(73)90024-1.
25. Pascoe L, Seow WK. Enamel hypoplasia and dental caries in Australian aboriginal children: prevalence and correlation between the two diseases. Pediatr Dent. 1994;16(3):193-9.
26. Folayan MO, Chukwumah NM, Popoola BO, Temilola DO, Onyejaka NK, Oyedele TA, et al. Developmental defects of the enamel and its impact on the oral health quality of life of children resident in Southwest Nigeria. BMC Oral Health. 2018;18(1):160. doi: 10.1186/s12903-018-0622-3.
27. Nogueira BR, Silva AM, de Castelo Branco Araújo T, Ferreira MC, Mendes RF, Prado Júnior RR. Exploring the association of predisposing factors of Cerebral Palsy and developmental defects of enamel: a case-control study. Eur Arch Paediatr Dent. 2021;22(3):367-74. doi: 10.1007/s40368-020-00558-2.
28. Martínez A, Cubillos P, Jiménez M, Brethauer U, Catalán P, González U. Prevalence of developmental enamel defects in mentally retarded children. ASDC J Dent Child. 2002;69(2):151-5.
29. Al Habashneh R, Al-Jundi S, Khader Y, Nofel N. Oral health status and reasons for not attending dental care among 12- to 16-year-old children with Down syndrome in special needs centres in Jordan. Int J Dent Hyg. 2012;10(4):259-64. doi: 10.1111/j.1601-5037.2012.00545.x.
30. Robles MJ, Ruiz M, Bravo-Perez M, González E, Peñalver MA. Prevalence of enamel defects in primary and permanent teeth in a group of schoolchildren from Granada (Spain). Med Oral Patol Oral Cir Bucal. 2013;18(2):e187-93. doi: 10.4317/medoral.18580.
31. Lin X, Wu W, Zhang C, Lo EC, Chu CH, Dissanayaka WL. Prevalence and distribution of developmental enamel defects in children with cerebral palsy in Beijing, China. Int J Paediatr Dent. 2011;21(1):23-8. doi: 10.1111/j.1365-263X.2010.01075.x.
32. Clarkson J. An Epidemiological Study of Dental Enamel Defects in Fluoridated and Non-Fluoridated Areas of Ireland [dissertation]. University College Cork; 1987.