Enamel defect of primary and permanent teeth in relation to nutrients daily intake among Down's syndrome children in comparison to normal children

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ABSTRACT

Background: Any child with Down's syndrome does not develop in the same manner as normal child. Therefore, the child should not be viewed as being like everyone else. Developmental enamel defects in primary teeth have been found at least twice as frequently in disabled children as in control children. Down's syndrome consumed protein more than the recommended daily allowance compared to other disabled groups. Therefore, the aim of this study was to investigate developmental defects of enamel and their relations to nutrient intake among Down's syndrome children in comparison to normal children.

Materials and Methods: A sample consisted of fifty institutionalized Down's syndrome children (study group) and 50 normal children (control group) aged 7-10 years old. Enamel anomalies were assessed according to criteria of WHO (1997). The dietary history was assessed through the use of food frequency questionnaire. Nutrients analysis was achieved by using a special software program designed by Diab (2003). All data were analyzed using SPSS version 13.

Results: A higher percentage of children with enamel anomalies were recorded among study compared to control group. Demarcated opacities were the most distributed type in permanent teeth of the study group while diffuse opacities were the most distributed type among the control group. Most of nutrients showed weak negative non significant correlations with enamel defects (demarcated opacities, hypoplasia) of primary teeth in study and control groups (P> 0.05). Results revealed lower mean values of most of daily nutrients intake among the study group than the control group.

Conclusion: This study reports a higher percentage of Down's syndrome children with enamel anomalies compared to normal children which may explain a negative correlation with most of daily nutrients intake, this may indicates that those population in need of preventive dietary program.

Key Words: Down's syndrome, enamel defects, protein, vitamin C, vitamin A, phosphorus, calcium. (J Bagh Coll Dentistry 2015; 27(3):152-158).

INTRODUCTION

Down's syndrome was the first chromosomal abnormality discovered in humans and results from the presence of an extra copy of chromosome 21 [1]. The incidence of trisomy 21 correlates strongly with increasing age, that is, young mothers have a low probability of having trisomy 21 children, but the risk increases rapidly after the age of 35 years [2]. Concerning the enamel defect among Down's syndrome, Almost 50% of persons with Down's syndrome exhibit three or more dental anomalies. Enamel hypocalcification occurs in about 20% of persons [3].

Iraqi studies regarding normals reported that the mean number of primary and permanent teeth with demarcated opacities was higher among well-nourished children than among underweighted and stunted children [4,-6]. It was reported that the deficiencies of nutrients intake during teeth development increased the developmental defects of these dentition [4,7].

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No previous Iraqi study has been conducted regarding the enamel defects and nutrients intake of Down's syndrome children. The aim of this study was to measure enamel defects of both primary and permanent teeth and their relations to daily nutrients intake among Down's syndrome children in comparison to normal children.

MATERIALS AND METHODS

The sample consists of 50 Down's syndrome children (study group) in mentally retarded institutions in Baghdad city with age range (7-10) years and a matching comparative sample of the control group was chosen randomly from primary schools [8] in the same geographical area of institutions. Enamel defects were recorded following the criteria of WHO; Ten index teeth were examined on the buccal surface only, if any index tooth is missing, the area was excluded. These teeth are for permanent: upper left and right central and lateral incisors, canine, first premolar and the lower left and right first molar while for primary teeth: upper left and right central and lateral incisors, canine, first molar, and lower left and right second molar [9].

Clinical examination was conducted using plane mouth mirror and dental probe. A food

frequency questionnaire was used to assess the dietary history of the sample [10]. It consists of food items commonly consumed in Iraq, this was achieved from the Nutrition Research Institution in Baghdad province, for each food items, child's parent was asked to indicate the average frequency of consumption over the past year by selection one of frequency categories ranging from never to four items per day. The selected frequency categories for each food items were converted to daily intake. In addition, other food components were added which are prepared by other countries and now consumed in Iraq. Nutrient analysis was measured by a special software program [4].

Statistical Package for Social Sciences (SPSS) version 13 was used for statistical analysis. The normally distributed variables were described by mean and SD and the parametric statistical tests were used (t-test), while non-normally distributed variables were statistically analysis by the non-parametric tests (Mann-Whitney test). Multiple regression models were used to assess the net and independent effect of each of a set of explanatory variables on a response (dependent) quantitative variable. P value less than the 0.05 level of significance was considered statistically significant.

RESULTS

Table (1) illustrates the distribution of children with enamel anomalies among study and control groups according to age groups and gender. A higher percentage of children with anomalies were recorded among study compared to control group.

Table (2) demonstrates the mean number of primary and permanent teeth with enamel anomalies among study and control groups. The mean numbers of primary teeth with enamel anomalies were higher among the study group compared to the control group with no significant difference (P> 0.05). At both age groups no statistical significant differences were noticed in mean number of primary teeth with enamel anomalies for both genders among study and control groups. Concerning the permanent teeth with enamel anomalies a higher mean value was recorded among the study group compared to the control group, difference was statistically significant (Mann-Whitney= 1083, Z= -1.971, P< 0.05). Total males and females demonstrated a higher mean number of teeth with anomalies among the study group than the control group, this was statistically not significant (P> 0.05). At 9-10 years of age a higher mean number of permanent teeth with enamel anomalies was noticed among the study group compared to the control group, difference was statistically significant (Mann-Whitney= 347.5, Z= -2.44, P< 0.05).

Table (3) shows the distribution of children concerning enamel anomalies types in primary and permanent teeth among study and control groups. Demarcated opacities were the most distributed type in permanent teeth of the study group while diffuse opacities were the most distributed type among the control group. Hypoplasia was recorded in primary teeth of the study group compared to the control group. The percentage of children with demarcated and diffuse opacities in their permanent teeth was higher among the study group compared to the control group.

Tables (4) and (5) demonstrate the mean percentage of primary and permanent teeth with different types of enamel anomalies. Results showed a lower mean percentage of primary teeth with demarcated opacities among the study group compared to the control group. A higher mean percentage of permanent teeth were recorded with demarcated and diffuse opacities among the study group compared to the control group.

Table 1: Distribution of children with enamel anomalies among study and control groups by age groups and gender

Age Group (Years)	Gender	Study Group			Control Group		
		Total No.	No.	%	Total No.	No.	%
7-8	M	14	2	14.3	14	3	21.4
	F	6	0	0	6	0	0
	T	20	2	10	20	3	15
9-10	M	22	7	31.8	22	1	4.5
	F	8	1	12.5	8	0	0
	T	30	8	26.7	30	1	3.3
All	M	36	9	25	36	4	11.1
	F	14	1	7.1	14	0	0
	T	50	10	20	50	4	8

Table 2: Mean number of primary and permanent teeth with enamel anomalies among study and control groups by age groups and gender.

Age Group (Years)	Gender	Study Group		Control Group	
		Primary Teeth	Permanent Teeth	Primary Teeth	Permanent Teeth
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
7-8	M	0.29 ± 1.07	0.14 ± 0.36	0.14 ± 0.53	0.43 ± 1.16
	F	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	T	0.2 ± 0.89	0.1 ± 0.31	0.1 ± 0.45	0.3 ± 0.98
9-10	M	0.00 ± 0.00	0.95 ± 2.57	0.00 ± 0.00	0.36 ± 1.71
	F	0.00 ± 0.00	1.00 ± 2.83	0.00 ± 0.00	0.00 ± 0.00
	T	0.00 ± 0.00	0.97 ± 2.59	0.00 ± 0.00	0.27 ±1.46
All	M	0.11 ± 0.67	0.64± 2.04	0.06 ± 0.33	0.39 ± 1.5
	F	0.00 ± 0.00	0.57 ± 2.14	0.00 ± 0.00	0.00 ± 0.00
	T	0.08 ± 0.57	0.62 ± 2.05	0.04 ± 0.28	0.28 ± 1.28

Table 3: Distribution of children concerning enamel anomalies types among study and control groups.

Types of Defect	Study Group (Total No.= 50)				Control Group (Total No.= 50)			
	Primary Teeth		Permanent Teeth		Primary Teeth		Permanent Teeth	
	No.	%	No.	%	No.	%	No.	%
Demarcated Opacities	1	2	7	14	1	2	1	2
Diffuse Opacities	0	0	3	6	0	0	2	4
Hypoplasia	1	2	0	0	0	0	0	0

Table 4: Mean percentage of primary teeth with enamel anomalies among study and control groups by age groups and gender.

Age Group (Years)	Gender	Study Group			Control Group		
		Demarcated Opacities	DiffuseOpacities	Hypoplasia	Demarcated Opacities	DiffuseOpacities	Hypoplasia
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
7-8	M	0.7 ± 2.67	0.00 ± 0.00	0.7 ± 2.67	1.4 ± 3.35	0.00 ± 0.00	0.00 ± 0.00
	F	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	T	0.5 ± 2.24	0.00 ± 0.00	0.5 ± 2.24	1.0 ± 4.47	0.00 ± 0.00	0.00 ± 0.00
9-10	M	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	F	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	T	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
All	M	0.3 ± 1.67	0.00 ± 0.00	0.3 ± 1.67	0.6 ± 3.33	0.00 ± 0.00	0.00 ± 0.00
	F	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	T	0.2 ± 1.41	0.00 ± 0.00	0.2 ± 1.41	0.4 ± 2.83	0.00 ± 0.00	0.00 ± 0.00

Table 5: Mean percentage of permanent teeth with enamel anomalies among study and control groups by age groups and gender.

Age Group (Years)	Gender	Study Group			Control Group		
		Demarcated Opacities	DiffuseOpacities	Hypoplasia	Demarcated Opacities	DiffuseOpacities	Hypoplasia
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
7-8	M	1.4 ± 3.63	0.00 ± 0.00	0.00 ± 0.00	1.4 ± 5.35	1.4 ± 5.35	0.00 ± 0.00
	F	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	T	1.0 ± 3.08	0.00 ± 0.00	0.00 ± 0.00	1.0 ± 4.47	1.0 ± 4.47	0.00 ± 0.00
9-10	M	3.2 ± 6.46	3.2 ± 12.87	0.00 ± 0.00	0.00 ± 0.00	1.8 ± 8.53	0.00 ± 0.00
	F	0.00 ± 0.00	5.00 ± 14.14	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	T	2.3 ± 5.68	3.9 ± 12.99	0.00 ± 0.00	0.00 ± 0.00	1.3 ± 7.3	0.00 ± 0.00
All	M	2.5 ± 5.54	1.9 ± 10.09	0.00 ± 0.00	0.6 ± 3.33	1.7 ± 7.37	0.00 ± 0.00
	F	0.00 ± 0.00	2.9 ± 10.69	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	T	1.8 ± 4.82	2.2 ± 10.16	0.00 ± 0.00	0.4 ± 0.83	1.2 ± 6.27	0.00 ± 0.00

The present study revealed a higher amount of calcium (mg) was consumed by the study group (789.94± 135.39) compared to the control group (778.30 ± 77.29) with no statistically significant difference **(**P> 0.05). Concerning vitamin C (mg), higher amount was consumed among study (108.02 ± 15.53) compared to control group (96.81 ± 11.79) with highly significant difference (t= 4.518, df= 98, P< 0.01). While lower amounts of protein (gm), phosphorus (mg), iron (mg) and vitamin A (I.U) were consumed by the study group (60.60 ± 3.85; 964.34 ± 210.68; 9.89 ± 1.97; 3863.74 ± 548.26 respectively) compared to the control group (60.75 ± 2.60; 964.94 ± 159.70; 12.23 ± 2.42; 4402.43 ± 635.25 respectively). Highly significant differences were recorded regarding iron (t= 5.305, df= 98, P< 0.001) and vitamin A (t= 4.497, df= 98, P< 0.001).

Table (6) demonstrates the correlation coefficient between mean percentage of primary and permanent teeth with enamel defect and nutrient daily intake in study and control groups. Most of nutrients showed weak negative non-significant correlations with enamel defects (demarcated opacities, hypoplasia) of primary teeth in study and control groups. Positive correlations were found for vitamin C in the study group and vitamin A in the control group.

Concerning defects (demarcated opacities, diffuse opacities) in permanent teeth the direction of correlation was found positive for all nutrients except Ca/P ratio in the study group. Significant correlation was achieved concerning phosphorus only with demarcated opacities. In the control group the direction of correlation with enamel defect (demarcated opacities, diffuse opacities) was observed negative with most nutrients except a positive correlation between vitamin C and demarcated opacities.

Table 6: Correlation coefficient between nutrient daily intake and enamel defect of primary and permanent teeth among study and control groups

Groups	Teeth		Protein (gm)		Calcium (mg)		Phosphorus (mg)		Ca/P Ratio		Iron (mg)		Vitamin A (I.U)		Vitamin C (mg)	
			r	P	r	P	r	P	r	P	r	P	r	P	r	P
Study	Primary	1	-0.124	0.392	-0.144	0.32	-0.025	0.865	-0.163	0.257	-0.213	0.138	-0.124	0.392	0.025	0.864
		2	------	------	------	------	------	------	------	------	------	------	------	------	------	------
		3	-0.124	0.392	-0.144	0.32	-0.025	0.865	-0.163	0.257	-0.213	0.138	-0.124	0.392	0.025	0.864
	Permanent	1	0.222	0.122	0.139	0.335	0.312	0.027*	-0.225	0.116	0.074	0.609	0.245	0.087	0.12	0.405
		2	0.231	0.106	0.182	0.205	0.122	0.399	-0.062	0.668	0.001	0.997	0.198	0.167	0.072	0.617
		3	------	------	------	------	------	------	------	------	------	------	------	------	------	------
Control	Primary	1	-0.134	0.355	-0.223	0.119	-0.054	0.707	-0.144	0.32	-0.153	0.287	0.069	0.632	-0.144	0.32
		2	------	------	------	------	------	------	------	------	------	------	------	------	------	------
		3	------	------	------	------	------	------	------	------	------	------	------	------	------	------
	Permanent	1	-0.104	0.473	-0.096	0.508	-0.104	0.473	-0.074	0.608	-0.134	0.355	-0.129	0.373	0.094	0.516
		2	-0.096	0.508	-0.223	0.12	-0.089	0.537	-0.091	0.529	-0.197	0.17	-0.096	0.505	-0.071	0.625
		3	------	------	------	------	------	------	------	------	------	------	------	------	------	------

1= Demarcated Opacities, 2= Diffuse Opacities, 3= Hypoplasia * Significant

Table (7) demonstrates the multiple linear regression of enamel defect of primary teeth explained by nutrient intake. Only negative association with iron was noticed while positive non-significant associations were recorded with other nutrients. The model was statistically not significant and explains 6% of variation.

The multiple linear regression of enamel defect of permanent teeth explained by nutrient intake is showed in Table (8). Iron and vitamin C were negatively associated with enamel defect of permanent teeth, highly significant correlation was recorded with iron. Other nutrients were positively associated, only significant correlation was observed with protein. The model was statistically not significant and was able to explain 12% of variation.

Table 7: Multiple linear regression of enamel defect of primary teeth explained by nutrient daily intake

	Partial Regression Coefficient	S.E	Standardized Coefficient	t	P-Value
***Study Group	-0.066	0.151	0.074	0.434	0.67
Protein	0.023	0.029	0.17	0.795	0.43
Calcium	0.000	0.001	-0.066	-0.343	0.73
Ca/P Ratio	3.012	0.315	0.137	1.051	0.3
Iron	-0.052	0.037	-0.291	-1.39	0.17
Vitamin A	0.00000338	0.000	0.006	0.041	0.97
Vitamin C	0.000	0.005	-0.013	-0.086	0.93

P (model) = 0.6 (Not Significant) R2= 0.06 ***Being study compared to control group

Table 8: Multiple linear regression of enamel defect of permanent teeth explained by nutrient daily intake

	Partial Regression Coefficient	S.E	Standardized Coefficient	t	P-Value
***Study Group	-0.57	0.561	0.168	1.017	0.31
Protein	0.227	0.108	0.434	2.094	0.039*
Calcium	0.003	0.003	0.18	0.963	0.34
Ca/P Ratio	0.789	1.171	0.136	1.082	0.28
Iron	-0.418	0.139	-0.609	-3.003	0.003**
Vitamin A	0.000	0.000	-0.051	-0.384	0.70
Vitamin C	-0.011	0.017	-0.09	-0.626	0.53

P (model) = 0.11 (Not Significant) R2= 0.12 *Significant, **highly significant ***Being study compared to control group

DISCUSSION

The present study showed that enamel anomalies in both primary and permanent dentitions were higher in Down's syndrome compared to the control group. This observation was also recorded by previous studies [11-13], this may be attributed to genetic disorders. No previous Iraqi study has been conducted among Down's syndrome children to allow comparison the result of current study with. In the present study, results revealed a lower mean value of most daily nutrients intake among Down's syndrome compared to control group, this may explain the inverse relations between nutrients and the enamel defects of primary teeth. Malnutrition in early childhood is associated with enamel hypoplasia of the primary dentition both of the classic, structural hypoplasia and with more limited evidence, for enamel opacities; there is weaker support for an enamel hypoplasia association with the permanent dentition due to a limited number of studies, potential malnutrition misclassification and confounding. The few studies of enamel hypoplasia properties not associated with known nutritional deficiencies in humans, suggest a decreased mineralization surface and subsurface of enamel affected by protein energy malnutrition [14]. There has been a considerable interest in role of calcium and protein in tooth formation. The effect of long term deficiency upon cellular activity of the ameloblast seen by the effect of plasma level on structural abnormalities and consider as a risk factor [15,16]. Daily nutrients intake of current study were found to be higher regarding calcium and vitamin C among study group compared to controls. It was reported that vitamin and mineral intakes were lower overall in individual with Down's syndrome than in normal, except for vitamin C [17]. Other studies also reported that children with Down's syndrome tended to consume more calcium and vitamin C than the recommended dietary allowance [17,18]. Results showed a less iron consumption by study compared to control group. This was also found in other studies [17,18]. This finding may be related to feeding difficulties and inappropriate nutrient and energy intakes which are common in children with Down's syndrome [17], some children rejected to eat from specific food groups, such as milk, meat and/or fruits and vegetables, or these foods may be offered only in limited amounts as explained by parents in current study. The current study revealed a lower amount of vitamin A consumption among study compared to control group. It was reported that vitamin A supplements have been proposed for children with Down's syndrome with claims of improving cognitive abilities, or immune function [19]. Vitamin A deficiency can slow down and even completely stop the grow of the incisor teeth of rat, accompanying this growth retardation was disturbance in differentiation and function of ameloblast, therefore enamel formation is interfered, this interference produces hypoplastic and chalky white incisor [20]. However, this study recorded a negative correlation of vitamin A with

enamel defects of primary teeth among study group.

There is a limitation regarding the possible etiological factors of enamel defects among Down's syndrome populations. Therefore, this study was conducted to explore the possible etiological factors of this defect related to nutrients intake.

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REFERENCES

1. Behrman RE, Kliegman RM. Nelson essentials of pediatrics. 4th ed. St. Louis: W.B. Saunders; 2002.
2. Cummings MR. Human heredity, principles and issues. 6th ed. Canada: Thomson; 2003. p.155-7.
3. Regezi J, Sciubba J, Jordan R. Oral pathology. 4th ed. St. Louis: Saunders; 2003. p.358-60.
4. Diab BS. Nutritional status in relation to oral health condition among 6-10 years primary school Iraqi children in the middle region of Iraq. Ph.D. Thesis, College of Dentistry, University of Baghdad, 2003.
5. Gatta EA. Primary teeth emergence and enamel anomalies in relation to nutritional status among 4-48 months old children in Baghdad city. A master thesis, College of Dentistry, University of Baghdad, 2005.
6. Droosh MK. Protein-energy malnutrition in relation to oral health condition among 6 and 9 year old primary school children in Sulaimania city in Iraq. A master thesis, College of Dentistry, University of Baghdad, 2007.
7. Koch MJ, Spranger S and Bettendorf M. Distinctive pitted enamel hypoplasia and short stature. J Craniofac-Genet Develo Biol 2000; 20:155-6.
8. Yammane T. Statistic and introductory analysis. 3rd ed. Tokyo: Harper International Edition; 1973. p. 677-759.
9. WHO. Oral health surveys basic methods 4th ed. World Health Organization. Geneva, Switzerland, 1997.
10. Thompson FE, Byers T. Dietary assessment resource manual. J Nutr 1994; 124: 2245-2317.
11. Bhat M, Nelson K. Developmental enamel defects in primary teeth in children with cerebral palsy, mental retardation, or hearing deficits: A review. Adv Dent Res 1989; 3(2):132-42.
12. Regezi J, Sciubba J, Jordan R. Oral pathology. 4th ed. St. Louis: Saunders; 2003. p. 358-60.
13. Scully C and Cawson R. Medical problems in dentistry. 5th ed. Italy: Elsevier Churhill Livingstone; 2005. p. 423-5.
14. Psoter WJ, Reid BC, Katz RV. Malnutrition and dental caries: A review of literature. Caries Res 2005; 39(6):441-7.
15. Rugg-Gunn A, Nunn J. Nutrition diet and oral health. 1st ed. Oxford: Oxford University Press, 1999.
16. Koch MJ, Spranger S and Bettendorf M. Distinctive pitted enamel hypoplasia and short stature. J Craniofac-Genet Develo Biol 2000; 20:155-6.
17. Luke A, Sutton M, Schoeller DA, Roizen NJ. Nutrient intake and obesity in prepubescent children with Down syndrome. J Am Diet Assoc 1996; 96(12):1262-7.
18. Chad K, Jobling A, Frail H. Metabolic rate: a factor of developing obesity in children with Down syndrome? Am J Ment Retard 1990; 95(2): 228-35.
19. Blair K, Roesler M, Xie Y, Gamis A, Olshan A, Heerema N, Robison L and Ross J. Vitamin supplement use among children with Down syndrome and risk of leukemia: A Children's Oncology Group (COG) Study. Paediatr Perinat Epidemiol 2008; 22(3): 288-95.
20. Johansson I, Ericson T, Lumikari M. The effect of vitamin A deficiency on the secretion of saliva and two salivary glycoprotein's in adult rat. J Vit Nutr 1989; 59: 234-5.

الخلاصة

يوجد اختلاف في نمو طفل متلازمة داون قياسا الى الطفل الطبيعي. وجد ان عيوب الميناء لديهم اعلى مرتين من الاطفال الطبيعيين ويستهلك اطفال متلازمة داون البروتين بنسبة اعلى من الاعاقات الاخرى. لذلك كان الهدف من هذه الدراسة هو لبحث عيوب الميناء وعلاقته بالعناصر الغذائية المتناولة لدى اطفال متلازمة داون مقارنة الى الاطفال الطبيعيين.

المواد والطرق :تكونت العينه من خمسين طفل متلازمة داون في المعاهد (مجموعة الدراسة) وخمسين طفل طبيعي (مجموعة الدراسه) بعمر 7-10 سنوات. تم قياس عيوب الميناء تبعا لتصنيف (WHO, 1997). تم التحليل الغذائي من خلال احتساب العناصر الغذائية بواسطة برنامج حاسوب صمم من قبل دراسة (Diab, 2003). حللت جميع البيانات احصائيا باستخدام SPSS 13.

النتائج :سجلت النسبة العالية من الاطفال المصابين بعيوب الميناء لدى مجموعة الدراسة مقارنة الى المجموعة الضابطة. العتمة البيضاء هي النوع الاكثر انتشارا في الاسنان الدائمية لدى اطفال متلازمة داون بينما لوحظ انتشار العتمة لدى المجموعة الضابطة. اشارت الدراسة الى علاقة عكسية ضعيفة وغير واضحة معنويا بين العناصر الغذائية وعيوب الميناء(العتمة، نقص التنسج) في الاسنان اللبنية لدى مجموعة الدراسة و المجموعة الضابطة. كشفت النتائج انخفاض في قيم العناصر الغذائية المأخوذة يوميا لدى اطفال متلازمة داون مقارنة الى المجموعة الضابطة.

الخلاصة :اقرت الدراسة ارتفاع في نسب الاطفال المصابين بعيوب الميناء مقارنة الى الاطفال الطبيعيين والذي يمكن ان يفسر العلاقة العكسية مع معظم العناصر الغذائية المتناولة يوميا، وهذا يشير الى ان اطفال متلازمة داون بحاجة الى برنامج غذائي وقائي.