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Original research

LACK OF ASSOCIATION BETWEEN ENAMEL FORMATION GENE VARIANTS AND DENTAL CARIES IN ADULTS

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ABSTRACT

Objectives: Studies report that gene polymorphisms associated with mineralization may change the structure of enamel and create a predisposition for developing dental caries. The aim of the study was to evaluate the *VDR* and *TFIP11* gene variants in adults with caries experience and to investigate their interactions with the environmental factors.

Materials and Methods: A total of 160 individuals at the age of 24-40 years were included in the study and they were assigned to two groups according to decayed-missing-filled teeth index (DMFT); namely the low caries experience (LCE, DMFT≤4) and high caries experience (HCE, DMFT> 9.13). DNA was isolated from buccal swab samples to genotype the *VDR* (TaqI; rs731236) and *TFIP11* (rs5997096) gene variants. The real-time PCR was used for genotyping. The frequency of tooth brushing, carbohydrate intake, smoking, and the dental plaques were evaluated as environmental risk factors.

Results: Between the caries groups and the distribution of the genotypes and alleles of the *VDR* rs731236 and *TFIP11* rs5997096 gene variants were not statistically different. There was also no significant difference when homozygous, heterozygous, dominant, and recessive models were evaluated for the two variants. The frequency of tooth brushing was significantly higher in the LCE group. According to the regression analysis; the amount of plaque explained the high caries experience at a rate of 51.4%.

Conclusions: The study findings indicated that common variants in the VDR and TFIP11 genes were not associated with high caries experiences in Turkish adults.

Key words: Dental caries, enamel, genes, vitamin D receptor.

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INTRODUCTION

Dental caries, one of the most common chronic diseases in the world, occurs as a consequence of a series of pathological events starting from the fermentation of carbohydrates by cariogenic bacteria in the dental plaque leading to formation of acid, which gradually converts the organic-inorganic molecules of the dental hard tissues to soluble forms, breaking down their chemical bonds.[1]

Researchers have so far evaluated the caries risk by addressing environmental factors including diet, bacteria, oral hygiene habits, dental plaque, and saliva alone or in combination.[2,3] Additionally; innate defense mechanisms are other important factors in the formation and progression of dental caries. Especially, soluble mediators contained in saliva include many antimicrobial molecules such as statherin, proline-rich proteins, cystatins, and histatins.[4] Saliva has also antioxidant system which prevents dental caries by influencing oral bacteria when inflammation beginning.[5] Previous studies have shown significantly higher levels of total antioxidant capacity in dental caries.[6,7] Furthermore, it has been shown that good oral hygiene and toothbrushing decrease salivary oxidative stresses.[8]

The studies in the literature have underlined that the assessment of individual environmental factors alone does not explain the formation of dental caries.[9,10] Because the caries risk is not the same for individuals exposed to the same environmental risk factors, it has been suggested that genetic factors are other players in the etiology of caries.[11]

It is reported that there may be a relationship between the susceptibility to caries and the genes encoding the proteins involved in enamel and dentin formation.[12-15] The following proteins have been investigated so far; including amelogenin, enamelin, tuftelin, tuftelin interactive protein, ameloblastin, and kallikrein involved in enamel mineralization; sialophosphoprotein involved in dentin formation, and the vitamin D receptor (VDR) involved in both enamel and dentin mineralization.[9-11,16]

Vitamin D regulates the balance between the calcium and phosphate ions, playing a vital role in making the teeth stronger.[17,18] The deficiency of vitamin D compromises the immune system defenses against oral pathogens in periodontitis and untreated dental caries.[17] The biologically active form of vitamin D; 1,25 $(OH)_2D_3$, is activated only after binding to the VDR encoded by the VDR gene.[18] Although numerous polymorphisms of the VDR gene have been reported on the chromosome 12q13.11 of the human genome, only the nucleotide polymorphisms ApaI, FokI, Cdx2, and TaqI have been investigated in regards to tooth decay. TaqI (A>G, rs731236) single nucleotide polymorphism (SNP) is located in the region of intron 8/exon 9 of the vitamin D receptor gene (VDR), close to the 3' terminus of the gene, and does not determine structural modification in the receptor. Nevertheless, most researchers suggest that it is related to mRNA stability. Previous studies on Chinese adults, Chinese young adults, Turkish children, and Czech children investigated the relationship between the TaqI polymorphism and dental caries, reporting contradicting findings.[17-20] In the literature, there are no studies investigating the relationship between the TaqI polymorphism and dental caries in Turkish adults.

Genetic variations influencing the development of enamel, which is the most mineralized tissue in the human body, have been investigated in genetic studies in association with dental caries. These studies have reached a consensus on a common hypothesis saying that the mineralization-related gene polymorphisms change the enamel structure, creating a predisposition to dental caries.[14,15,21-25]

The tuftelin-interacting protein 11 (TFIP11) is localized in the 22q12.1 region, playing an important role in the formation and mineralization of the enamel by interacting with the tuftelin protein.[20] The TFIP11 rs5997096 (C>T) single nucleotide polymorphism is the most common intron variant in populations. The studies in the literature investigating the relationship between TFIP11 and dental caries in different age groups and populations report variable findings.[12,13,24,26,27]

In this study, the null hypothesis is that the variants in the mineralization-related VDR and TFIP11 genes in adults elevate the risk of caries in combination with the effects of the environmental factors including gender, tooth brushing frequency, carbohydrate intake, and smoking. Therefore, the aim of the study is to evaluate the interactions between the caries-experience-related environmental factors and the variants of the VDR and TFIP11 genes involved in enamel formation in adults.

MATERIALS AND METHODS

Study population and oral examination

The study was registered at clinicaltrials.gov with registration No. NCT04124718. A total of 160 adults (86 women, 74 men) at the age range from 24 to 40 years and living in the Northeast of Turkey were included in the study. Individuals with neurological, mental, systemic, and genetic diseases and individuals with regular medicine intake were excluded from the study. The study was approved by the the Clinical Research Ethics Committee of Recep Tayyip Erdogan University School of Medicine (ID: 2019/204).

In the clinical evaluation, the patients were examined for the presence of caries and dental plaque by an investigator (GYT) using dental unit light, mouth mirror, and probe. According to WHO criteria; clinically visible cavitated lesions, softened enamel surfaces caught by the dental probe, radiolucent areas invading the dentine starting from the enamel-dentin border were documented as 'caries lesions'. Whitish-brown discolorations not caught by the exploring probe were not considered a caries lesion.[28]

To determine the risk groups based on past caries experiences; DMFT indices were calculated based on the WHO criteria after summing up the teeth count with following features, including decay (D), extracted teeth due to caries (M), and filled (F) teeth in the mouth of each study participant. According to these calculations; 80 individuals with DMFT indices of>13.9 and previous HCE were included in the experimental group and 80 individuals with DMFT indices of ≤4 and previous LCE were included in the control group (Table 1).

Table 1. Distributions of gender and age among adult in the HCE and LCE groups.

Characteristic	Total N = 160	High caries experience HCE N=80	Low caries experience LCE N=80	p value
Gender n (%)[a]				
Female	86 (53.8)	47 (54.7)	39 (45.3)	0.267
Male	74 (46.2)	33 (44.6)	41 (55.4)	
Age in years, median (IQR)[b]	33 (17.0)	35 (14.0)	29.5 (15.0)	0.005

[a]$\chi 2$ test — P < 0.05, statistically significant difference between HCE and LCE.
[b]Mann-Whitney test. — P > 0.05, no statistically significant difference between HCE and LCE.
HCE, Adult with dental caries experience, DMFT>13.9. — LCE, Adult with dental caries experience, DMFT≤4.

A standard questionnaire form, comprising items about the frequency of tooth brushing, carbohydrate intake, and smoking was administered to all study participants to evaluate the environmental risk factors for caries. The Silness & Löe plaque index was used for determining the amount of dental plaque.[29]

Collection of Samples and DNA Isolation

Samples were obtained from the buccal mucosa using swaps which are the material of the Swab Collection and DNA Preservation System (Norgen Biotek Corp., Ontario, Canada). The components of this system allow DNA samples to be stored at room temperature for over 2 years. The samples were stored at room temperature in the preservative solution until the DNA isolation step. Genomic DNA was isolated with Genomic DNA Isolation Kit (Norgen Biotek Corp.Ontario, Canada). The purity and concentration of the isolated DNA samples were analyzed using a fluorescent dye on a fluorometer (Denovix QFX Fluorometer, Denovix Inc., DE, USA). The

purified DNA samples were then stored at -20 until further use.

Genotyping

Genotyping of VDR rs731236 and TFIP 11 rs5997096 gene variants were performed using the TaqMan® SNP Genotyping Assays (C___2404008_10 for VDR rs731236, C__29903745_10 for TFIP11). The reactions were carried out in LightCycler® 480 Instrument II (Roche Diagnostics, Basel, Switzerland), a real-time PCR system. A standard PCR reaction mixture was prepared, and the same mixture was used for both candidate genes. The cycling conditions were as follows: an initial denaturation at 95°C for 10 min followed by 40 cycles of denaturation at 92°C for 15 s and annealing/extension at 60°C for 1 min.

Statistical analyses

The statistical analysis was processed with SPSS Statistics 23.0 (IBM, Chicago, IL, USA). For data that were not normally distributed, as shown by a Kolmogorov-Smirnov test, Mann-Whitney U test was used to compare age differences. The determination of the deviations from the Hardy–Weinberg Equilibrium and the differences between genetic models and the calculation of odds ratios in genetic models were carried out in the FINNETI program (http://ihg.gsf.de/cgi-bin/hw/hwa1.pl.21). Binary logistic regression analysis was used to evaluate the effects of genetic and enviromental factors on caries experience. Power analysis was performed with respect to the cross-sectional study design. In all tests, the level of significance was set at $P<0.05$.

RESULTS

As the age and DMFT scores were not normally distributed, median with interquartile range (IQR) was used to determine between group differences (Table 1). The median age was 33.0 (IQR = 17.0, minimum = 25.0, maximum = 44.0). Significant differences in age were observed between the low and high caries experience groups (Mann-Whitney U test, P = 0.005). Median DMFT scores of 15.0 (IQR = 5.0) and 2.0 (IQR= 3.0) were observed in the high and low caries experience groups, respectively.

The graph indicating the genotype results of the VDR TaqI; rs731236 gene variant obtained from the LightCycler® 480 Instrument II are representatively shown in Figure 1(A-B).

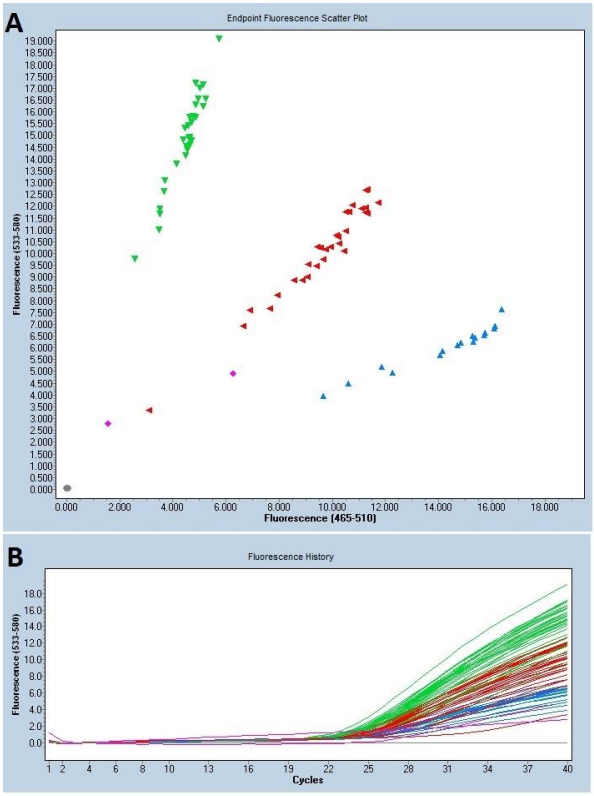

Figure 1. Typical results for the VDR rs731236 gene variant, using the LightCycler® 480 II Instrument. (A) A fragment of the human VDR gene was amplified using the 2X Taqman Universal PCR Master mix and subjected to endpoint analysis. Scatter plots consistently revealed the wild-type (AA), mutant (GG) and heterozygote (AG) variants. The green small triangles show the AA genotypes, the blue small triangles show the GG genotypes and the red small triangles show the AG genotypes. The pink small squares show samples without distinctive fluorescent light that need to be analyzed again, while the gray round shows the negative control. (B) Amplification curves of the VDR gene fragments amplified from each sample were shown.

The instrument also graphed for the TFIP11 rs5997096, giving us the results of genotyping. The Pearson Chi-Square test was used between the genotype frequencies of the VDR rs731236 and TFIP11 rs5997096 gene variants of the groups to test for deviations from the Hardy-Weinberg Equilibrium (HWE). The frequencies of both polymorphisms did not deviate from this equilibrium ($p>0.05$, Table 2 and Table 3).

Table 2. Frequencies and odds values of alleles and genotypes for the VDR rs731236 in HCE and LCE groups.

Group	Allele		P	OR (95 % CI)	Genotype			HWE	
	A	**G**			**AA**	**AG**	**GG**		
	n (%)	**n (%)**			**n (%)**	**n (%)**	**n (%)**		
LCE (n=80)	97 (60.6)	63 (39.4)	0.30	1.2 (0.8-1.96)	30 (37.5)	37 (46.25)	13 (16.25)	0.77	0.32
HCE (n=80)	88 (55)	72 (45)			27 (33.75)	34 (42.5)	19 (23.75)	0.20	

Test for association OR (95% CI) (Risk allele G)

Heterozygous	**Homozygous**	**Dominant**	**Recessive**
AA vs. AG	**AA vs. GG**	**AA vs. AG+GG**	**AA+AG vs. GG**
1.02 (0.5-2.05)	1.6 (0.6-3.9)	1.17 (0.6-2.25)	0.6 (0.2-1.36)
P=0.95	P=0.27	P=0.62	P=0.23

OR: Odds Ratio
CI: Confidence Interval
HWE: Hardy-Weinberg Equilibrium

Table 3. Frequencies and odds value of alleles and genotypes for the TFIP11 rs5997096 in HCE and LCE groups

Group	Allele		P	OR (95 % CI)	Genotype			HWE	P
	C	**T**			**CC**	**CT**	**TT**		
	n (%)	**n (%)**			**n (%)**	**n (%)**	**n (%)**		
LCE (n=80)	79 (49.3)	81 (50.7)	0.91	1.02 (0.6-1.58)	18 (22.5)	43 (53.75)	19 (23.75)	0.50	0.91
HCE (n=80)	78 (48.75)	82 (51.25)			20 (25)	38 (47.5)	22 (27.5)	0.65	

Test for association OR (95% CI) (Risk allele T)

Heterozygous	**Homozygous**	**Dominant**	**Recessive**
CC vs. CT	**CC vs. TT**	**CC vs. CT+TT**	**CC+CT vs. TT**
0.79 (0.36-1.72)	1.04 (0.4-2.5)	0.87 (0.42-1.8)	0.82 (0.4-1.67)
P=0.56	P=0.92	P=0.71	P=0.58

OR: Odds Ratio
CI: Confidence Interval
HWE: Hardy-Weinberg Equilibrium

The comparisons of the genotype and allele frequencies of the VDR rs731236 gene variant between the HCE and LCE groups were shown in Table 2. Analysis of different genetic models including dominant (AA vs. AG+GG), recessive (AA+AG vs. GG), and co-dominant (AA vs. AG, AA vs. GG) was done using Chi-square test. The genotype frequencies of the AG and GG genotypes versus the ancestral genotype (AA) were not statistically significantly different between the HCE and LCE groups (P=0.95 and P=0.27, respectively). Similarly, the allele frequencies were not statistically significantly different between these two groups (P=0.30). As shown in Table 2, no evidence of significant association was found in any genetic model.

Table 3 shows the comparisons of the genotype and allele frequencies of the TFIP11 rs5997096 gene variant between the high and low caries experience groups.

TT homozygous polymorphic genotype of the TFIP11 gene was found at frequencies of 23.75% and 27.5% in the LCE and HCE groups, respectively. There were no statistically significant differences in the TFIP11 genotype and allele distributions between the LCE and HCE groups (P=0.91, P=0.91). Analysis of different genetic models including dominant (CC vs. CT + TT), recessive (CC + CT vs. TT), and co-dominant (CC vs. CT, CC vs. TT) was done using Chi-square test. As shown in Table 3, no

statistical difference was found in any of the genetic models.

Based on the scores received by the study participants, the statistical comparisons of the frequency distributions of the environmental caries risk factors in the LCE and HCE groups are shown in Table 4. The amount of dental plaque was significantly larger in the HCE group compared to the LCE group (2 test; p=0.000). The tooth brushing frequency was significantly higher in the LCE group compared to the HCE group (2 test; p=0.042).

Table 4: Comparison of enviromental factors between Low caries experience (LCE) and High caries experience (HCE) groups

	LCE	HCE	Total	p value
Dental plaque				
PI<1.0	54 (68)	6 (7)	60 (38)	
PI 1.1-2.0	25 (31)	59 (74)	84 (52)	**0.000**
PI>2	1 (1)	15 (19)	16 (10)	
Toothbrushing frequency				
>twice a day	12 (15)	8 (10)	20 (12)	
Twice a day	35 (43)	21 (26)	56 (35)	**0.042**
Once a day	22 (28)	35 (44)	57 (36)	
<once a day	11 (14)	16 (20)	27 (17)	
Carbohydrate intake				
Less than 1/day	15 (19)	11 (13)	26 (16)	
1 or 2/day	46 (58)	37 (45)	83 (52)	0.151
3 or 4/day	13 (16)	19 (22)	32 (20)	
5 or more/day	6 (7)	13 (20)	19 (12)	
Smoking				
no	59 (74)	64 (80)	123 (77)	0.454
yes	21 (26)	16 (20)	37 (23)	

Values are presented as n (%) of subjects. P values based on χ2 test, P < 0.05.

The binary logistic regression analysis was used for evaluating the effects of genetic and environmental factors on caries experience (Table 5). The participants with larger plaque accumulation were significantly at higher risk of experiencing dental caries as compared to their counterparts.

Table 5. Binary logistic regression analyse showing odds ratio (OR) and 95% confidence interval for caries experience

Independent variables	**OR (%95 CI)**	**p**
Dental plaque	21.345 (7.722- 58.996)	**0.000**
Carbohydrate intake	1.198 (0.734 – 1.956)	0.470
Toothbrushig frequency	0.915 (0.554 – 1.511)	0.728
Age	1.004 (0.945 – 1.067)	0.902
Gender	0.428 (0.175 – 1.042)	0.062
Smoking	0.889 (0.324 – 2.440)	0.820
VDR	1.159 (0.654 – 2.054)	0.614
TFIP11	0.780 (0.431- 1.410)	0.411

Coding: Caries experience (low = 0, high = 1), age (25-35 = 1, 35-44 = 2); gender (male=1, female = 2); VDR (AA=1, AG=2, GG=3); TFIP11 (CC=1, CT=2, TT=3)
R^2=0.514

DISCUSSION

It has been shown that environmental factors including oral hygiene, diet, and bacteria, as well as the individual genetic differences, are associated with the caries risk.[11,22,30] Therefore, the influences of enamel formation-related VDR and TFIP11 gene polymorphisms and the gene-environment interactions on the caries experience were investigated in this study.

In many epidemiological studies, past caries experiences have been reported as important risk indicators for developing new caries in the future.[31,32] Several studies[18,19,22,26,33-35] evaluating the caries risk have described the caries risk groups based on past caries experiences. According to the World Health Organization data, the DMFT index should be less than 5 to be considered the low caries risk; whereas the DMFT index should be higher than 13.9 to be considered the high caries risk.[36] Therefore, the risk groups of the individuals included in the study were determined based on their past caries experiences.

It has been reported that VDR gene polymorphisms are important factors for the normal enamel formation[20] and that the variations in this gene lead to inherited phenotypes of enamel malformations.[37] Hypothesis of the present study says that the TaqI polymorphism of the VDR gene affects enamel formation, leading to a higher caries experience in adults. The study findings did not indicate differences in the allele and genotype frequency distributions of the TaqI polymorphism of the VDR gene between the individuals with low and high caries experiences. This result was similar to those previously reported by the Kong *et al*.[17] study on 0-4-year-old Chinese children, Yu *et al*.[20] study on 12-year-old Chinese children, and Holla *et al*.[18] study on 13-15-year-old Czech children. On the contrary, two other studies on Chinese adults[16] and Turkish children[19] have demonstrated that the polymorphic allele in the TaqI variation may constitute a risk for developing dental caries. These differences across the studies may be explained with the use of the PCR-RFLP method to determine the VDR gene polymorphisms in the latter two studies in contrast to the use of the TaqMan technique in the present study, allowing to obtain more precise results. Secondly, another explanation could be that Turkish adults were evalulated in this study.

Current study has found out no differences in the polymorphic TT genotype and T allele frequencies in the TFIP11 rs5997096 variation between the low and high caries experience groups. Several studies are available in the literature, reporting the same results as ours. Of them; Abbasoglu *et al*.[38] study examined 23 gene markers in Turkish children, including the TFIP11 rs5997096 polymorphism and found out no relationships between the TFIP11 gene and dental caries. In another study on Turkish children, Patir *et al*.[12] did not find out any relationship between TFIP11 rs134136 and tooth decay. Other studies that did not find any associations between this polymorphism and dental caries were performed on children from Poland[39], on young adults from Guatemala[24], and on children from Western Norway.[40] In another study[21] on 1831 children and young adults from Philippines, Turkey, Argentina, and Brazil; rs134136 and rs5997096 polymorphisms of the TFIP11 gene was not associated with past caries experiences, however, it was found out that the TFIP11 rs134136 polymorphism affected the enamel microhardness of 48 extracted teeth with artificially induced caries lesions. In another *in vitro* study[25], the variations of the TFIP11 (rs2097470, rs134143) gene have been shown to affect enamel demineralization in a *Streptococcus mutans* biofilm model. The *in vitro* design of those studies may have led to differences in the results compared to those of the present study.

There are also studies in the literature showing that a single nucleotide polymorphism in TFIP11 is associated with dental caries.[26,27] The differences in the results of that latter study compared to the former studies have been explained by arguing that; firstly, TFIP11 is responsible for early demineralization and fluoride-mediated remineralization of the enamel and secondly, the environmental heterogeneity might be involved, such as fluoride, which affects the risk of caries.

Epigenetic change of the genetic code are caused by environmental stimuli and hence are responsible for our ability to adapt to different environments.[41] No single host gene that directly regulates dental caries progression has been identified. Epigenetics however, may provide the missing link to these unanswered questions.[42]

In the literature, there are studies available, using regression analyses and risk models investigating the effect of gene polymorphisms on caries development in combination with the effects of environmental factors.[12,13,22,35] Among these studies, Slayton *et al.*[13] have reported a 26.8% accuracy of the caries risk model; which included the polymorphism of the TUFT1 gene and the S. mutans count. Another study[12] found out that the best explanation for the past caries experience was provided at an accuracy rate of 40.2% by the model comprising the polymorphisms of the TUFT1 (rs3790506) and AMELX (rs17878486) genes in female children with caries lesions on both anterior and posterior teeth. Similarly; Tennure *et al*.[35] reported that in black children with MMP20 (rs1784418) polymorphism and sugar consumption between the meals elevated the caries risk by 74.61%. In this study, the effects of gene polymorphisms and environmental factors on the high caries experience were examined using a risk model and regression analysis. The study findings showed that the presence of dental plaques provided an explanation at a rate of 51.4%. This result is similar to that of a previous study reporting that the dental plaque ranks the first with a rate of 77.6% in the model, which explained the gene-environment interaction on developing risk for caries with an accuracy of 87.8%.[22]

Recently, the development of tissue engineering raises regenerative methods in dentistry. Its major component is the mesenchymal stem cells that are seeded on the surface of scaffolds, in order to create a biocomplex.[43] Animal studies have reported that mesenchymal stem cells provide alveolar bone regeneration, dentine formation and repair damaged tooth tissues.[44,45] Gene therapy presents an attractive concept of restoring the oral tissues lost due to caries by managing the differentiation of stem cells.[46] Recently, osteogenic genes are presented to promote the bone formation and cellular differentiation using tissue engineering approaches.[47] However, it has been reported that more genetic research is needed to better understand odontogenesis.[43]

The limitation of the present study can be the selection and investigation of the genes among the previously reported caries-associated polymorphisms. Instead of selecting these polymorphisms, conducting studies on different populations, as well as selecting new genes that have not been investigated previously and may potentially be associated with enamel formation, can yield different results.

CONCLUSIONS

In conclusion; findings from this study indicated that the VDR TaqI; rs731236 and TFIP11 rs5997096 gene polymorphisms were not associated with high caries experiences in Turkish adults. The presence of dental plaques provided an explanation for the past caries experience at an accuracy rate of 51.4%. Studies with larger sample size in different populations will contribute to better understanding the role of these variants. There is also a need for further studies investigating the effects of variants in different genes to better explain the genetic basis of the dental caries etiology. If future studies can recognize risk or protective genetic factors, researchers will potentially be able to design more effective treatments aimed at preventing dental caries.

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CONFLICTS OF INTEREST STATEMENT

The authors declare that they have no conflict of interest.

Erişkinlerde Mine Formasyon Gen Varyantları ve Diş Çürüğü Arasındaki İlişki Yokluğu

ÖZ

Amaç: *Çalışmalarda mineralizasyonla ilişkili gen polimorfizmlerinin minenin yapısını değiştirerek, diş çürüğüne yatkınlık oluşturabileceği bildirilmektedir.*

Bu çalışmada erişkinlerde mine oluşumunda yer alan VDR ve TFIP11 gen polimorfizmlerini ve çürük deneyimi üzerindeki çevresel faktörlerle etkileşimini değerlendirmeyi amaçladık. ***Gereç ve Yöntemler:*** *Çalışmaya 24-40 yaş aralığındaki toplam 160 birey katıldı ve DMFT indeksine göre geçmiş çürük deneyimi düşük (DMFT≤4) ve yüksek (DMFT>13,9) olan iki gruba ayrıldı. VDR (TaqI; rs731236) ve TFIP11 (rs5997096) polimorfizmlerini değerlendirmek için yanak içi sürüntü örneklerinden DNA izolasyonu yapıldı. Genotipleme işlemi için real-time PCR ile gerçekleştirildi. Çevresel çürük risk faktörleri olarak diş fırçalama sıklığı, karbonhidrat tüketimi, sigara kullanımı ve dental plak miktarı değerlendirildi.* ***Bulgular:*** *Yüksek çürük deneyimi ve düşük çürük deneyimi grupları arasında VDR ve TFIP polimorfizmlerinin genotip ve alel frekanslarının dağılımları açısından istatistiksel açıdan fark gözlenmedi. İki varyant için homozigot, heterozigot, baskın ve resesif modeller değerlendirildiğinde anlamlı farklılık saptanmadı. Diş fırçalama sıklığı LCE grubundaki bireylerde HCE grubundaki bireylere göre anlamlı derecede daha fazlaydı. Binary lojistik regresyon analizine göre; plak miktarı yüksek çürük deneyimini %51,4 oranında açıkladı.* ***Sonuçlar:*** *Bulgularımız VDR ve TFIP11 genlerindeki yaygın görülen varyantların Türk erişkinlerindeki yüksek çürük deneyimleriyle ilişkili olmadığını göstermiştir.*
Anahtar Kelimeler: *Diş çürüğü, mine, genler, vitamin D reseptörü.*

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