Clinical and Experimental Health Sciences

Analysis of Saliva and Gingival Crevicular Fluid Immunoglobulin a in Adults Having Different Caries Status

Pinar Gul[1], Neslihan Celik[1], Hayrunisa Hanci[2], Tugba Aydin[3], Nilgun Akgul[4], Nilgun Seven[1]

[1] Ataturk University, Faculty of Dentistry, Department of Restorative Dentistry, Erzurum, Türkiye.
[2] Ataturk University, Faculty of Pharmacy, Department of Pharmaceutical Microbiology, Erzurum, Türkiye.
[3] Ataturk University, Faculty of Dentistry, Department of Periodontology, Erzurum, Türkiye.
[4] Pamukkale University, Faculty of Dentistry, Department of Restorative Dentistry, Denizli, Türkiye.

Correspondence Author: Pinar Gul
E-mail: opinargul@gmail.com
Received: 15.12.2020 **Accepted:** 15.11.2021

ABSTRACT

Objective: The aim of the study was to compare the salivary IgA and gingival crevicular fluid IgA levels in caries active and caries free subjects.

Methods: The study was carried out on 40 subjects divided into two groups, Group I caries free, (DMFT =0) and Group II caries active, (DMFT >10). Saliva and gingival crevicular fluid samples were obtained and plaque index, gingival index, gingival crevicular fluid volume and salivary flow rate were determined. The levels of S. mutans and lactobacillus were determined by culture. IgA concentrations of saliva and gingival crevicular fluid samples were determined using the ELISA. Data were analyzed using the Mann-Whitney U and $\chi 2$ tests ($\alpha = 0.05$).

Results: Gingival crevicular fluid volume, plaque index and gingival index values of group II were significantly higher than group I ($p< 0.05$). Although lactobacillus counts of group II were higher than group I, no statistically significant differences were observed for microorganism counts ($p>0.05$). Salivary flow rate, gingival crevicular fluid-IgA and saliva-IgA levels were no statistically significant between the groups ($p>0.05$).

Conclusion: The present study showed that microorganism counts in the saliva did not by themselves influence the DMFT index. There is no dependence between secretory immunity and dental caries in subjects.

Keywords: Dental caries, gingival creviculer fluid, immunoglobulin, saliva.

1. INTRODUCTION

Dental caries is a multifactorial disease and microorganisms play the most important role in the formation and development of caries. Streptococcus mutans are often isolated from cavitated carious lesions and Streptococcus mutans to be considered a primary pathogen in caries due to the fact that it produces glucan for bacteria to adhere to the tooth surface, is acidic and aciduric (1). Lactobacilli are colonized later, so they do not play an active role at the initiation of caries, but they are effective in the development of caries after the lesions progress. Since the level of lactobacilli in saliva is associated with a high consumption of carbohydrates, it can be a useful indicator for a cariogenic diet (2).

Human saliva is a clear, slightly acidic (pH 6.0 to 7.0) liquid consisting of 98% water and 2% electrolytes, mucus, antibacterial compounds, and various enzymes (2). Saliva also contains many proteins involved in maintaining oral tissue, such as lysozyme, lactoferrins, lactoperoxidase, albumin, mucin, histatins, defensins, and immunoglobulins (3). In addition, saliva is also a mixture of salivary gland secretions, gingival crevicular fluid (GCF), bronchial and nasal secretions, bacteria and bacterial products, desquamation epithelial cells, and other cellular oral fluids (2). Gingival crevicular immune mechanism involves both cellular and humoral immunity (4).

Saliva immunoglobulins are the first line of defense against pathogenic bacteria and their secretions (5). Salivary IgA (S-IgA) prevents adhesion of bacteria to the tooth surface. S-IgA does this by neutralizing bacterial toxins and enzymes by blocking the binding of bacteria to cell receptors (5).

Numerous studies have reported the protective role of S-IgA against dental caries in both children and adults (6, 7). These studies investigated the correlation between total salivary S-IgA concentrations and caries susceptibility as recorded by an index of decayed, missing, or filled teeth (DMFT) or surfaces (DMFS). However, the results differ; while some studies showed positive or negative correlation between S-IgA and dental caries, in some studies, no correlation was reported. Therefore, the protective mechanism of S-IgA against dental caries is still unclear.

For this reason, the aim of the present study was to quantify the microbiological and immunological profiles of caries active and caries free subjects and to identify the active

DOI: 10.33808/clinexphealthsci.841002

cariogenic factors that can be used to predict dental caries in susceptible populations.

The null hypotheses of the study, there is no significant difference between immunological and microbiological profiles of in caries active and caries free subjects.

2. METHODS

2.1. Subjects and Study Design

This study was carried out with the ethical approval of Ataturk University Faculty of Dentistry Ethics Committee (16.10.2012/012) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all individual participants included in the study. The study was carried out on 40 subjects (20 females and 20 males) aged 15–40 years, who attended the Department of Restorative Dentistry, Faculty of Dentistry, Ataturk University. Inclusion criteria are systemically healthy, anti-inflammatory drugs have not been used within the previous 2 months, and no antibiotic therapy has been taken in the past 6 months. The exclusion criteria were tobacco consumption, diabetes, pregnancy, breastfeeding, medication intake that caused a decrease in saliva flow, use of antibiotics within 6 months or dental or periodontal treatment that continued 12 months before the beginning of the study (8). Dental caries examination was performed according to the World Health Organization criteria using traditional dental chairs, artificial light, flat mirror and explorer (9). Subjects were divided into 2 groups as Group I DMFT = 0 caries-free group and DMFT> 10 caries active group.

Saliva and GCF samples were collected from participants in the study. Saliva flow rate, Streptococcus mutans and lactobacillus levels and Salivary IgA were analyzed in saliva samples. Plaque index (PI), gingival index (GI) GCF volume and GCF-IgA analysis were performed on GCF samples.

2.2. Saliva Sampling

Subjects were asked to brush their teeth and not eat or drink for 2 hours before sampling. All procedures were performed in the same order to reduce the effect of circadian rhythm on saliva flow and content (8). Subjects were seated in dental chairs and 2 cc of unstimulated saliva was collected in especial tubes according to Scully method (10), (In this method, subjects were asked to spit in the tubes once a minute for ten minutes). All samples were collected between 09:00 and 11:00, and the time spent for each procedure was set to a maximum of 30 minutes (11). The average salivary flow rate (SFR) was measured from the total volume according to Krasse (12). After collection of 0.5mL of salivary sample, it was transferred immediately to the laboratory at a temperature of –80ºC.

2.3. Periodontal Examination and GCF Sampling

Plaque index (PI), gingival index (GI) and GCF volume were determined. The same trained examiner recorded PI and GI using a periodontal probe according to Löe and Silness (13). Four different sampling areas were created in the mouths of the subjects in the premolar molar region. Plaque and gingival index were evaluated on the facial, lingual / palatinal, mesial and distal surface of the teeth to be examined. According to these results, a separate score was given for the four surfaces of the tooth and the total score was divided into four and the score of the tooth was found.

GCF was collected from the mesial or distal surface of the respective teeth. Following the evaluation of the PI, the supragingival plaque was removed and the areas to be sampled were isolated using cotton rolls. Saliva absorber was used to prevent saliva contamination. A paper strip (Periopaper, ProFlow Inc., Amityville, USA) was inserted intracreviculer 1 mm below the gingival margin and left in place for 30 s. The procedure was repeated three times. A total of 3 strips of paper were sampled and the strips of paper were transferred to a chairside electronic gingival fluid meter (Periotron 8000, Oraflow Inc., Plainview, USA) for volume determination. The paper strips were then immediately placed into three labeled Eppendorf tubes (Microcentrifuge tubes, ISOLAB, Wertheim, Germany) containing 100 µl of 0.9% of physiological saline solution, isolated with ParafilmH M, (SPI Supplies Inc., West Chester, USA) to avoid evaporation and sent to the laboratory. The samples were stored at – 80°C for subsequent assays (14).

2.4. Microbiological Tests

S. mutans and Lactobacilli analysis in the saliva of the subjects were carried out according to the Koga-Ito(15) method in the Microbiology Laboratory of Ataturk University Medical Faculty. Within 3 h after sampling, saliva samples were diluted to 10^{-1} and the following tests were performed:

2.4.1. Mutans streptococci Counts

Diluted saliva samples from subjects were planted in Mitis salivarius Bacitracin sucrose agar (Difco, Detroit, Mich., USA) and incubated at 37 ° C in candle jars for 72 hours. S. mutans colonies formed at the end of this period were counted, and amounts over 100,000 CFU / mL were considered the high risk group and those below were considered the low risk group.

2.4.2. Lactobacilli Counts

Diluted saliva samples from subjects were planted in Rogosa agar (Difco, Detroit, Mich., USA) under aerobic conditions for 72 hours at 37 ° C. Lactobacilli colonies formed at the end of this period were counted and amounts above 10,000 CFU / mL were considered as the high-risk group and those below were considered the low-risk group.

2.5. S-IgA and GCF-IgA Analysis

S-IgA and GCF-IgA analysis were performed using commercial ELISA kits (Human Secretory Immunglobulin A ELISA Kit, East Biopharm, LOT NO: 201408) according to manufacturer's recommendations at the Ataturk University, Faculty of Medicine Microbiology Laboratory. Firstly, solutions were prepared for the test. The samples were homogenized by shaking and centrifuge at 2000 rpm for 20 minutes and supernatants were removed. For test procedure 40 µL sample,10 µL S-IgA-antibody and 50 µL Streptavidin-HRP were added to test wells and incubated 60 minutes at 37°C. At the end of incubation plates were washed five times. After washing procedure, 50 µL chromogen A and 50 µL chromogen B solutions were added to each well and mixed gently. Plates were incubated 10 minutes at 37°C away from light. After this period, 50 µL stop solution was added into each well to stop the reaction. Finally measurements were made optical densit (OD) under 450 nm wavelenght within 15 minutes after adding stop solution. A standard curve was obtained and salivary SIgA levels were calculated and expressed in µg/mL.

2.6. Statistical Analysis

Statistical analysis was performed using SPSS 20 (IBM, Chicago, IL, USA). Kolmogorov-Smirnov test was used to determine the distribution of the data. The Mann-Whitney *U* test was used to compare the parameters between caries free and caries active group. In addition, χ2 test was used to compare the mutans and lactobacillus levels of groups. The value of $p<0.05$ was considered as statistically significant.

3. RESULTS

The clinical and laboratory data obtained from caries free and caries active subjects and the statistical comparison results are given in Table 1. SFR is within normal limits in both groups and there is no statistically significant difference between them (p=0,056). GCF volume, GI and PI values of caries active group were significantly higher than caries free group (p< 0.05). But, GCF-IgA and S-IgA levels were no statistically significant between the groups (p>0.05).

In addition, lactobacillus counts of caries active group were higher than caries free group but no statistically significant differences were observed for microorganism counts between the groups (p>0.05) (Table 2).

Table 1. *Clinical and laboratory values and Mann-Whitney U test results between the groups*

GROUPS			DMFT	PI	GI	GCF (µL)	SFR (mL/min)	GCF-IgA (µg/mL)	S-IgA (µg/mL)
CF (n=20)	Median		0.00	0.64	0.29	0.03	0.40	5.56	6.40
	Minimum		0	0.00	0.00	0.02	0.13	4.16	3.00
	Maximum		0	1.71	1.71	0.08	5.00	9.04	12.71
	Percentiles	25	0.00	0.33	0.073	0.03	0.28	4.82	4.38
		50	0.00	0.64	0.29	0.03	0.40	5.56	6.40
		75	0.00	1.00	0.68	0.048	0.62	6.13	8.11
CA (n=20)	Median		14.00	1.50	0.71	0.05	0.88	5.21	5.77
	Minimum		9	0.29	0.12	0.03	0.05	4.12	3.72
	Maximum		25	3.43	2.43	0.10	2.50	6.82	20.70
	Percentiles	25	11.00	0.71	0.43	0.04	0.46	4.82	4.41
		50	14.00	1.50	0.71	0.05	0.88	5.21	5.77
		75	15.00	2.50	1.07	0.07	1.36	5.87	8.18
p			0.001*	0.002*	0.011*	0.002*	0.056	0.579	0.978

**p<0.05, CF: Caries free, CA: Caries active, SFR: Saliva flow rate, PI: Plaque index, GI: Gingival index, GCF: Gingival crevicular fluid, GCF-IgA: Gingival crevicular fluid IgA, S-IgA: Salivary IgA*

Table 2. *Distribution of Streptococcus mutans and lactobacillus levels in the subjects' saliva and their statistical comparisons*

	CF n(%)	CA n(%)	χ2	p
S. mutans				
≤ 10^5 CFU/mL	19 (95)	19 (95)	0.00	1.00
> 10^5 CFU/mL	1 (5)	1 (5)		
Lactobacilli				
≤ 10^4 CFU/mL	14 (70)	8 (40)	3.64	0.06
> 10^4 CFU/mL	6 (30)	12 (60)		

**Data are presented as n (%). CF: Caries Free; CA: Caries Active; CFU = colony-forming units.*

4. DISCUSSION

Studies in recent years have shown that antibodies are valuable components in preventing dental caries. Research on immunology has indicated that bacterial products modulate the immune response (16). Saliva components that can affect bacterial proliferation and plaque ecology have been described and have been reported to form caries resistance mechanisms (16). Three effective mechanisms have been described in the pathogenesis of caries. These are S-IgA and other antibodies, serum antibodies and cellular immunity. Especially S-IgA is known to play an important role in preventing caries (16).

One of the antibacterial properties of saliva is that it has secretory immune system components. Saliva has an important role in maintaining oral health. It has been proved to be a credible diagnostic aid in detecting different biomarkers (3). Gingival crevicular fluid is a biological fluid originating from blood plasma, which has different compositions in the gingival sulcus and has the properties to determine the ecology of the gingival sulcus. Cellular components, electrolytes, bacterial-metabolic products, cytokines, host and bacterial enzyme and enzyme products-inhibitors and immunoglobulins in the GCF content are characteristic of the liquid (14). Therefore, in the present study, salivary and GCF immunoglobulins and S. mutans and lactobacilli status in saliva were investigated in caries free and caries active subjects. According to our research, although IgA studies in adults' saliva are few, no study investigating IgA in GCF has been found. There are a few and inadequate studies on adult's salivary IgA and only the studies by Koga-ito (17) and Gornowicz (18) involved adults. Therefore, it is necessary to conduct research on this subject. In the present study, GCF and S-IgA levels were no statistically significant between the groups ($p>0.05$). In addition, lactobacillus counts of caries active group were higher than caries free group but no statistically significant differences were observed for microorganism counts between the groups ($p>0.05$). Therefore, the hypothesis was accepted.

In the present study, dental carries index was used to assess the relationship with S-IgA. Many previously conducted studies used decay-missing-filled (DMF) index (19). In this study, clinical parameters such as PI, GI, GCF volume and saliva flow rate were also determined. The salivary flow determination was performed according to Krasse (12) and the results were classified as follows: normal (up to 1 ml/min), reduced salivary flow (lower than 0.7 ml/min) and xerostomia (values below 0.1 ml/min). In this study, it was found that the saliva flow rates of caries free and caries active subjects were in the range of 0.98 and 1.00 ml / min and these values were within normal limits in both groups. Saliva flow rate was not statistically significant between the groups ($p>0.05$), because all subjects were selected among clinically healthy individuals who did not take any medication that could affect saliva flow rate.

GCF is an exudate from the blood plasma in the gingival sulcus between the tooth and the gingival edge or in the periodontal pocket. In fact, there is very little GCF in the healthy sulcus, and when the gum is healthy, this liquid is in the form of a transudate or serum exudate in the sulcus. With the increase of inflammation in the gingiva, transudate turns into inflammatory exudate, which contains molecules derived from high amounts of gingival tissues, vascular cellular components of inflammation and serum-derived molecules. Increased GCF volume is positively associated with the degree of gingival inflammation (20). In the present study, GCF volume, GI and PI values of caries active group were significantly higher than caries free group ($p< 0.05$). As a result, in the present study, a moderate inflammation can be mentioned in the caries active group according to the system of Löe and Silness. This may be due to exclusion of individuals with periodontal disease from the study, since only the effect of caries on IgA level was examined.

There is no consensus on the S-IgA concentration in caries free and caries active subjects in different studies, despite the fact that immunological factors seem to play an important role in microorganism colonization and dental caries. Although some studies demonstrated high concentrations of S-IgA in a lower caries activity (3, 17, 21, 22), other studies showed high levels of S-IgA with an increase in caries activity (4, 11, 18, 23-25) . However, there are studies that have not observed any correlation between S-IgA and dental caries (17, 26-29).

Shetty et al.,(3) found that IgA decreased with increase in caries prevalence. According to the authors, this may be due to the high specific binding ability of the immunoglobulins to the microbial species and their neutralizing effect. Thus, it protects against dental caries by preventing bacterial adhesion and colonization on the tooth surface. Gregory et al., (21) also reported similar results and explained this difference in IgA levels may be due to increased production of IgA antibodies against Streptococcus mutans in caries free than caries active childrens. Koga-ito (17) and Kuriakose (22) indicated in their studies that the amount of S-IgA reduced following the increase in the number of decayed teeth and according to their conclusion, this reduction could be as a result of body defence mechanism.

Nawaz et al.,(4) found that S-IgA was raised in patients with dental caries compared to healthy controls and the authors stated that this may be due to the protective mechanism of immunoglobulins against dental caries to reduce and control caries severity. Ranadheer et al. (25) and Amoundi et al. (23) have also detected an increased level of IgA in patients of dental caries**.** In similar to the results of the study, Gornowicz and Fidalgo indicated higher levels of S-IgA in people with more decayed teeth (18, 24) As the results of the study showed, there was a positive correlation between high levels of dental caries and salivary levels of IgA. The result is confirmed by Bagherian (11) that revealed a weak inverse correlation between the variables.

Interestingly, some studies showed no correlation between dental caries and IgA levels. A study by Camling and Kohler (26) demonstrated no clear evidence for the protective role

of salivary IgA antibodies against Streptococcus mutans colonization. Zengo et al., (29) and Fukuda et al., (27) stated that the difference between caries free and caries active groups did not reach statistical significance in terms of immunoglobulins in parotid saliva. The results of these studies are similar to the findings of our study. Studies conducted by Koga et al. and Shifa et al. supported this hypothesis (17, 28).

These differences as a result of studies may be due to variations in sampling size, different conditions in collecting saliva samples, different criteria applied in subject selection, differences among individuals in terms of oral hygiene and diet. In addition, immunoglobulin concentrations may vary depending on saliva flow rate, hormonal factors, physical activity and emotional state (30).

The limitation of this study is that it was conducted with a small number of participants.

5. CONCLUSION

Within the limits of this study, it may be considered that caries status may not affect S-IgA and GCF-IgA levels. However, further studies with larger sample sizes will provide a better understanding of the subject.

ACKNOWLEDGMENTS

This work was supported by the Research Fund of Ataturk University (Project number: 2012/372). This study was presented at the 47th Meeting of the Continental European Division of the International Association for Dental Research (CED-IADR), Antalya, Turkey, 15-17 October 2015.

REFERENCES

[1] Yang Y, Li Y, Lin Y, Du M, Zhang P, Fan M. Comparison of immunological and microbiological characteristics in children and the elderly with or without dental caries. Eur J Oral Sci 2015;123(2):80-87.

[2] Bansode PV, Pathak SD, Wavdhane M, Birage PP. Salivary biomarkers of dental caries–A Review Article. J Dent Med Sci 2018;17:12-18.

[3] Shetty N, Hegde M, Kumari S, Ullal H, Kedilaya V. Salivary proteins as biomarkers in dental caries: In vivo study. Dent Oral Craniofac Res 2017;3:1-7.

[4] Nawaz A, Batool H, Kashif M, Abbas A, Shahzad F, Tahir R, Afzal N. Immune profiling of saliva in patients with and without dental caries. Bangladesh J Dent Med Sci 2019;18(3):536-539.

[5] Badabaan GM, Arafa AA. The Relationship between salivary IgA level and dental caries in healthy school-aged children in makkah al-mukarramah. IJHSR 2017;7(6):125-134.

[6] Bratthall D, Serinirach R, Hamberg K, Widerström L. Immunoglobulin A reaction to oral streptococci in saliva of subjects with different combinations of caries and levels of mutans streptococci. Oral Microbiol Immunol 1997;12(4):212-218.

[7] Golpasand Hagh L, Zakavi F, Ansarifar S, Ghasemzadeh O, Solgi G. Association of dental caries and salivary sIgA with tobacco smoking. Aust Dent J 2013;58(2):219-223.

[8] Karaoğlanoğlu S, Akgül N, Akgül HM. The association between the DMFS index and levels of salivary Streptococcus mutans and lactobacilli of subjects living in Erzurum, Turkey. J Dent Sci 2010;5(2):70-74.

[9] WHO, Oral Health Surveys, Basic Method. 4th ed ed. Geneva 1997.

[10] Scully C. Comparative opsonic activity for Steptococcus mutans in oral fluids, and phagocytic activity of blood, crevicular, and salivary polymorphonuclear leucocytes in rhesus monkeys. Immunology 1980;39(1):101.

[11] Bagherian A, Jafarzadeh AE, Rezaeian M, Ahmadi S, Rezayati M. Comparison of the salivary immunoglobulin concentration levels between children with early childhood caries and caries-free children. Iran J Immunol 2008;5(4):217-221.

[12] Krasse B. Interpretation and use of microbiologic findings in dental caries. Oral Microbiol Immunol 1986;1(1):85-86.

[13] Silness J, Löe H. Periodontal disease in pregnancy II. Correlation between oral hygiene and periodontal condition. Acta Odontol Scand 1964;22(1):121-135.

[14] Celik N, Askın S, Gul MA, Seven N. The effect of restorative materials on cytokines in gingival crevicular fluid. Arch Oral Biol 2017;84:139-144.

[15] Koga-Ito C, Unterkircher C, Watanabe H, Martins C, Vidotto V, Jorge AOC. Caries risk tests and salivary levels of immunoglobulins to Streptococcus mutans and Candida albicans in mouthbreathing syndrome patients. Caries Res 2003;37(1):38-43.

[16] Alaçam T, Misirligil Z, Misirligil A. The relationship between the caries activity test (snyder) and salivary IgA level. J Nihon Univ Sch Dent 1986;28(3):195-198.

[17] Koga-Ito CY, Martins CAdP, Balducci I, Jorge AOC. Correlation among mutans streptococci counts, dental caries, and IgA to Streptococcus mutans in saliva. Braz Oral Res 2004;18(4):350-355.

[18] Gornowicz A, Tokajuk G, Bielawska A, Maciorkowska E, Jabłoński R, Wójcicka A, Bielawski K. The assessment of sIgA, histatin-5, and lactoperoxidase levels in saliva of adolescents with dental caries. Med Sci Mon Int Med J Exp Clin Res 2014;20:1095.

[19] Haeri-Araghi H, Zarabadipour M, Safarzadeh-Khosroshahi S, Mirzadeh M. Evaluating the relationship between dental caries number and salivary level of IgA in adults. J Clin Exp Dent 2018;10(1):e66.

[20] Emingil G, Çinarcik S, Baylas H, Çoker I, Hüseyinov A. Levels of leukotriene B4 in gingival crevicular fluid and gingival tissue in specific periodontal diseases. J Periodontol 2001;72(8):1025-1031.

[21] Gregory RL, Kindle JC, Hobbs LC, Filler SJ, Malmstrom HS. Function of anti-Strepfococcus mutans antibodies: inhibition of virulence factors and enzyme neutralization. Oral Microbiol Immunol 1990;5(4):181-188.

[22] Kuriakose S, Sundaresan C, Mathai V, Khosla E, Gaffoor F. A comparative study of salivary buffering capacity, flow rate, resting pH, and salivary Immunoglobulin A in children with

rampant caries and caries-resistant children. J Indian Soc Pedod Prev Dent 2013;31(2):69.

[23] Al Amoudi N, Al Shukairy H, Hanno A. A comparative study of the secretory IgA immunoglobulins (s. IgA) in mothers and children with SECC versus a caries free group children and their mothers. J Clin Pediatr Dent 2007;32(1):53-56.

[24] da Silva Fidalgo TK, Freitas-Fernandes LB, Ammari M, Mattos CT, de Souza IPR, Maia LC. The relationship between unspecific s-IgA and dental caries: a systematic review and meta-analysis. J Dent 2014;42(11):1372-1381.

[25] Ranadheer E, Nayak UA, Reddy NV, Rao VAP. The relationship between salivary IgA levels and dental caries in children. J Indian Soc Pedod Prev Dent 2011;29(2):106.

[26] Camling E, Köhler B. Infection with the bacterium Streptococcus mutans and salivary IgA antibodies in mothers and their children. Arch Oral Biol 1987;32(11):817-823.

[27] Fukuda T, Aono M, Onoue K. A study on the relationship between the IgA content of parotid saliva and dental caries (author's transl). Nihon Shishubyo Gakkai Kaishi 1975;17(1):55-58.

[28] Shifa S, Muthu M, Amarlal D, Prabhu VR. Quantitative assessment of IgA levels in the unstimulated whole saliva of caries-free and caries-active children. J Indian Soc Pedod Prev Dent 2008;26(4):158.

[29] Zengo A, Mandel I, Goldman R, Khurana H. Salivary studies in human caries resistance. Arch Oral Biol 1971;16(5):557-560.

[30] Marcotte H, Lavoie MC. Oral microbial ecology and the role of salivary immunoglobulin A. Microbiol Mol Biol Rev 1998;62(1):71-109.

How to cite this article: Gul P, Celik N, Hanci H, Aydin T, Akgul N, Seven N. Analysis of Saliva and Gingival Crevicular Fluid Immunoglobulin a in Adults Having Different Caries Status. Clin Exp Health Sci 2022; 12: 128-133. DOI: 10.33808/clinexphealthsci.841002