ORIGINAL ARTICLE

CONTENTS

Correlation between interleukin-17 levels and oral manifestations in patients with oral squamous cell carcinoma

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ABSTRACT

Aim: To assess the level of salivary IL-17 in Oral Squamous Cell Carcinoma patients compared to healthy individuals and their association with molecular etiology and oral manifestations.

Materials and Methods: A case-control study was carried out in 56 patients with OSCC and 46 healthy matched subjects. The demographics, tumor molecular profiles (TP53 mutations and EGFR overexpression), and oral examination findings were systematically recorded. ELISA was used to detect the concentration of IL-17 in saliva.

Results: Salivary IL-17 levels were significantly elevated in patients with Oral Squamous Cell Carcinoma (21.34 ± 4.6 pg/mL) compared to the control group (19.72 ± 4.2 pg/mL, $p = 0.040$). IL-17 was higher in TILs of those patients; tumors with overexpression of EGFR exhibited higher levels than those with TP53 mutations. The most common oral manifestations observed in this study included mucositis and xerostomia. The increase in salivary IL-17 was also found to be independently associated with mucosal alterations and changes in pigmentation.

Conclusions: Salivary IL-17 levels were elevated in patients with Oral Squamous Cell Carcinoma, with this biomarker being correlated in such cases with tumors driven by EGFR and with specific oral manifestations. Thus, this biomarker has the potential to be a non-invasive tool for disease detection and follow-up.

KEYWORDS: oral squamous cell carcinoma, salivary IL-17, biomarker, TP53 mutation, EGFR overexpression, oral manifestations

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INTRODUCTION

Oral squamous cell carcinoma is the most common among oral cancers accounting for more than 90% of global cases [1]. It is an important public health issue, particularly in some areas where specific risk factors of tobacco intake, alcohol consumption and betel nut chewing are highly prevalent [2]. Although improvements have been achieved regarding improved therapeutic regimens and refined diagnostic tools, OSCC (Oral Squamous Cell Carcinoma) still has a poor prognosis [3]. The survival rate for 5-year-olds ranges mostly from 50 to 60%. Most of the deaths related to this disease can be ascribed to late clinical presentation and its complex biology [4]. In the past years, immune responses in OSCC have been increasingly observed. Of all cytokines, interleukin-17 (IL-17) has emerged as one of the most important pro-inflammatory cytokines that modulates tumor growth, angiogenesis and metastasis [5]. Th17 cells secrete interleukin-17 and have dichotomized roles in both tumor promotion and suppression depending on the specific tumor microenvironment [6]. Elevated expression of IL-17 is seen in a range of tumors but can be particularly pronounced in head and neck cancers. This makes hypotheses about its potential role in disease progression and prognosis [7]. Ulceration, induration, pain and dysphagia are extremely important presenting symptoms in clinical examination of a patient diagnosed with OSCC by the patient themselves [8]. Although they are of diagnostic value, they likely represent patient's immunological and background inflammatory status [9]. Analysis of the association between IL-17 levels and oral clinical features might shed light on cancer biology and lead to new targeted therapies [10]. The present scientific environment highlights precision medicine and the incorporation of immunological indicators for early detection, surveillance, and customized therapy in OSCC [11]. Since IL-17 plays two roles in cancer biology, relating its serum concentrations to particular oral symptoms might uncover clinically significant relationships that would be useful for risk assessment and intervention planning [12]. Anyhow, the association of circulating

levels of IL-17 with oral clinical features is yet to be adequately investigated, and the available evidence remains scarce and inconsistent. To our knowledge, only a few studies have directly addressed whether high circulating levels of IL-17 can be a clinically relevant biomarker connecting tumor biology with the clinical manifestation of oral symptoms in OSCC.

AIM

The researchers seek to systematically examine the correlation between serum levels of interleukin-17 (IL-17) and clinical symptoms of the oral cavity in patients with OSCC and to obtain new information on immunopathological mechanisms that shape the natural history of disease and symptom development. The current study, therefore, aims at integrating systemic cytokine profiling with comprehensive oral clinical assessment to determine if the use of the signature IL-17 to predict OSCC risk, prognosis, and treatment response can be utilized as a non-invasive biomarker of precision medicine in oral oncology.

MATERIALS AND METHODS

PATIENTS AND DATA COLLECTION

This descriptive cross-sectional study took place at the Middle Euphrates Oncology Center in Al-Najaf, Iraq, from December 2024 through June 2025. The study included 56 patients with OSCC and 46 healthy matched subjects. Inclusion criteria for patients required a histopathologically confirmed diagnosis of OSCC and an age of 18 years or older. Participants were enrolled either during routine oncology outpatient visits or while hospitalized for the management of OSCC. Exclusion criteria encompassed patients with chronic systemic conditions such as uncontrolled diabetes mellitus, autoimmune disorders, or cardiovascular diseases. Additionally, individuals with a history of other malignancies, those experiencing ongoing acute infections, or inflammatory conditions not related to OSCC were also excluded. Data regarding demographics and clinical characteristics, including age, gender, tumor location, and disease stage, were collected from hospital medical records.

SALIVA COLLECTION AND PROCESSING

Unstimulated whole saliva was collected from each patient in the morning between 9 and 11 AM to minimize diurnal variation. Participants were advised not to eat, drink (excluding water) or perform oral hygiene activities for at least 1 h before sampling. Each subject was in a comfortable seated position and asked to slightly lean the head downward allowing saliva to pool within their mouth for expectoration into a sterile 15 mL polypropylene collection tube during a period of 5 min. The samples were immediately put on ice and centrifuged at 3000 rpm for 10 min to remove debris. The resulting supernatants were divided into aliquoted and stored frozen at –80 °C up to analysis.

ORAL MANIFESTATION ASSESSMENT

All patients underwent the same oral examination, performed by the same experienced specialist in oral medicine. These assessments were performed with adequate illumination and disposable mouth mirrors and tongue blades. Three major categories of oral findings were reviewed: mucosal changes, salivary gland disease, and mucosal pigmentation alterations. Mucosal changes included differences in mucositis, lichenoid reactions, ulceration and other discernable epithelial lesions. Salivary gland dysfunction was evaluated using patient-reported symptoms (e.g., dry mouth and swallowing discomfort) in conjunction with findings during clinical examination such as decreased salivary pooling or signs of inflammation of the salivary glands. Alterations in mucosal pigmentation were recorded for pigmented or depigmented areas in terms of their location and extent. All findings were documented on standardized clinical data sheets and wherever possible corroborated with photographic documentation for visual aid.

DETECTION OF TP53 MUTATIONS AND EGFR OVEREXPRESSION

Saliva samples were collected from each patient in the morning between 9:00 and 11:00 AM to avoid diurnal fluctuation. Participants were asked not to eat, drink (other than water), or brush their teeth for at least one hour before saliva collection. Depressurized whole saliva was collected in sterile tubes and centrifuged at 3000 × g for 10 minutes, and the supernatants were stored at −80°C until analysis. Genomic DNA was extracted from the pellet of salivary gland cells for analysis of TP53 mutations using a commercial kit. PCR was performed to generate exons 5-8 of the TP53 gene, followed by sequence analysis using the Sanger sequencing technique. The generated sequence data were aligned with the reference TP53 sequence (GenBank accession no. NM_000546) for mutation detection, using point-substitution mutations, insertions or deletions in accordance with IARC guidelines. For EGFR

(Epidermal Growth Factor Receptor) over-expression, the levels of protein in saliva were quantified with the use a commercially available ELISA kit (Diagnostic Systems Laboratories), specific for human epidermal growth factor receptor determined by ELISA. All samples were performed in duplicate and the OD was measured at 450 nm using a microplate reader. Overexpression was assessed based on cut-off values provided by the manufacturer of the ELISA kit, derived from reference ranges established using samples from healthy controls.

MEASUREMENT OF SALIVARY IL-17 LEVELS

Salivary IL-17 concentrations were determined using a high-sensitivity sandwich ELISA kit (specific for human IL-17A) according to the manufacturer's instructions. Prior to analysis, frozen saliva supernatants were thawed at room temperature and centrifuged at 10,000 rpm for 5 minutes to remove residual particulates. Standards and samples were added in duplicate to 96-well microplates pre-coated with monoclonal anti-IL-17 antibodies. After incubation and washing steps, a biotin-conjugated detection antibody was added, followed by streptavidin–HRP solution. The colorimetric reaction was developed using TMB substrate and stopped with 2 N sulfuric acid. OD values were measured at 450 nm using a microplate reader, and IL-17 concentrations were calculated from a standard curve generated using known concentrations of recombinant IL-17A. The detection limit of the assay was 0.5 pg/mL, and intra-assay and inter-assay coefficients of variation were both below 10%.

ETHICAL CONSIDERATION

Approval for conduct of this study was obtained from the Institutional Review Board of the Middle Euphrates Oncology Center and the College of Medicine, University of Kufa (No. 143# in 2025). Written informed consent was obtained from all participants in accordance with the Declaration of Helsinki. Before performing collection of samples, it was mandatory for all patients participating in the study to furnish written consent for their involvement

STATISTICAL ANALYSIS

The SPSS software version 25.0 (SPSS, Chicago) was used for all the appropriate statistical analyses. The normality test which was used to test who want to continuous data for Shapiro Wilk test. All normally distributed data are acted as mean ± standard deviation. Independent t test was used to find out the statistical difference in the respiratory function markers between patients' and control. ANOVA test was used to find out the statistical difference in the respiratory function markers among patients' subgroups classified according to IBs subtypes. A p-value less than 0.05 was considered to indicate a statistically significant difference.

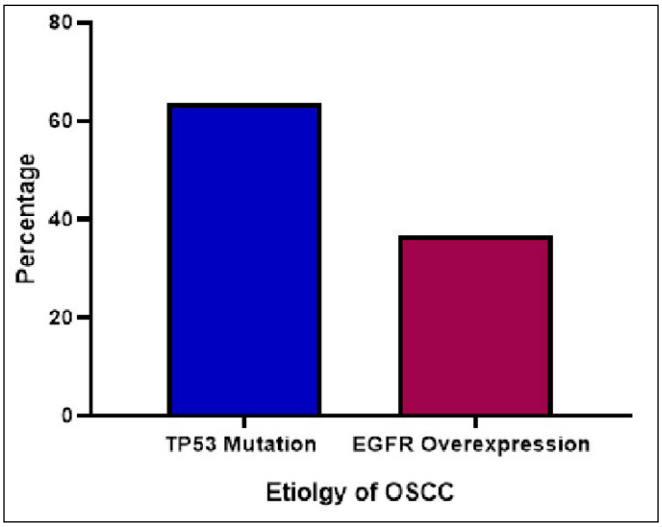

Fig. 1. The proportion of OSCC patients showing TP53 mutations and EGFR overexpression based on salivary molecular analysis. TP53 = tumor protein p53; EGFR = epidermal growth factor receptor. Values are expressed as percentages of the total study population
Source: Own materials

RESULTS

The finding of the demographic data indicated that there was no statistically significant difference in the distribution of age, gender, and residence between oral squamous cell carcinoma patients and the control group ($p > 0.05$ for all). This proves that both groups were comparable, hence minimal demographic confounding effects on further analyses (Table 1).

Regarding molecular etiology of OSCC, TP53 mutations were seen in 64.3% of cases and EGFR overexpressed in 35.7%, making the TP53 mutation the predominant molecular subtype figure 1.

Comparison of salivary IL-17 levels in OSCC patients and healthy controls showed a statistically significant elevation (p value 0.040) in the patient group. The mean IL-17 concentration in OSCC patients was 21.34 ± 4.6 pg/mL, which more than that in the control group which was 19.72 ± 4.2 pg/mL (Table 2).

Differences in salivary IL-17 levels between OSCC patients and control participants

On the other hand, EGFR overexpression presents with much higher salivary IL-17 levels in comparison to those with TP53 mutation p=0.036. This denotes a

Table 1. Demographic characteristics for patients and control groups

Demographic characteristics		Patients (No. = 56)		Control (No. = 46)		Chi square	P value (sig.)
		Freq.	%	Freq.	%		
Age/Years	23–32	18	32.1	15	32.6	0.002	0.998
	33–42	20	35.7	16	34.8		
	≥ 43	18	32.1	15	32.6		
Gender	Male	28	50	23	50	0	1
	Female	28	50	23	50		
Residence	Urban	30	53.6	25	54.3	0.005	0.943
	Rural	26	46.4	21	45.7		

Source: Compiled by the authors of this study

Table 2. Differences in salivary IL-17 levels between OSCC patients and control participants

Groups	No.	Salivary IL-17 (pg/ml) Mean ± SD	T Test (P Value)
Patient	56	21.34 ± 4.56	0.040
Control	46	19.72 ± 4.18	

Source: Compiled by the authors of this study

Table 3. Differences in salivary IL-17 levels between patients' subgroups classified according to molecular etiology

Groups	No.	Salivary IL-17 (pg/ml) Mean ± SD	T test (P Value)
TP53 Mutations	36	58.4 ± 12.6	2.15 (p = 0.036)
EGFR Overexpression	20	66.8 ± 14.1	

Source: Compiled by the authors of this study

Table 4. Oral manifestation for patients participated in the study

Indicators		Patients (No. = 56)	
		Freq.	[%]
Mucosal Change	Oral mucositis	24	42.9
	Lichenoid reactions	10	17.9
	Aphthous-like ulcers	6	10.7
	No Change	16	28.5
Salivary Gland Dysfunction	Xerostomia	22	39.3
	Sialadenitis	8	14.3
	Normal	26	46.4
Mucosal Pigmentation Changes	Hyperpigmentation	12	21.4
	Depigmentation	7	12.5
	No Change	37	66.1

Source: Compiled by the authors of this study

Table 5. Logistic regression analysis for the association between salivary IL-17 levels and oral manifestations in OSCC patients

Oral manifestation	β (coefficient)	SE	Wald χ^2	OR (95% CI)	p-value
Mucosal Change	0.215	0.085	6.38	1.24 (1.05–1.47)	0.012
Salivary Gland Dysfunction	0.178	0.091	3.85	1.19 (0.99–1.42)	0.050
Mucosal Pigmentation Changes	0.241	0.088	7.50	1.27 (1.07–1.51)	0.006

Source: Compiled by the authors of this study

possible connection between the biology of tumors driven by EGFR and an increased inflammatory response (Table 3).

The findings of the oral manifestation indicated that mucositis turned out to be the most frequent mucosal alteration among OSCC patients-42.9%, followed by

lichenoid reaction at 17.9% and aphthous-like ulcers at 10.7%. 28.5% show no any kind of mucosal alterations, describing xerostomia as their primary salivary gland dysfunction accounting for 39.3% with fewer cases of sialadenitis -14.3%. Almost half the patients, or rather 46.4%, have normal salivary function. Hyperpigmentation describes the outcome in 21.4% of patients; depigmentation results in 12.5%. About two-thirds or rather a full 66.1% have no pigmentation changes (Table 4).

Logistic regression analysis revealed that higher levels of salivary IL-17 were significantly related to mucosal change (OR = 1.24, 95% CI: 1.05-1.47, p = 0.012) and mucosal pigmentation change (OR = 1.27, 95% CI: 1.07-1.51, p=0.006) in patients with OSCC. Salivary gland dysfunction showed a borderline association with IL-17 levels (p=0.050). These results could indicate that increased level of this cytokine may be involved in the pathogenesis of some oral lesions in OSCC (Table 5).

DISCUSSION

Our study shows significantly increased salivary IL-17 in OSCC patients (21.34 ± 4.6 pg/mL) when compared to healthy controls (19.72 ± 4.2 pg/mL; p=0.022). This further strengthens the role of IL-17 in inflammation related to OSCC, and hence, potentially as a non-invasive biomarker of the disease for its detection and monitoring [13-14]. Meta-analyses and systematic reviews conducted on salivary cytokines in OSCC verified raised expression levels of IL-6, IL8, TNF-, and recently available data also add IL17 to emphasize salivary cytokine panels as promising tools in diagnostics [15-19]. On the molecular level, IL-17 signals various interacting pathways regarding the pathogenesis of OSCC. It does so by inducing the expression of IL-6, vascular endothelial growth factor (VEGF), and matrix metalloproteinases (MMPs) through JAK–STAT3 and NF-κB signaling pathways that would result in the proliferation, angiogenesis, invasion, and survival of malignant keratinocytes [7]. In addition to this, IL-17 facilitates the recruitment of myeloid-derived suppressor cells (MDSCs) and cancer-associated fibroblasts which support epithelial–mesenchymal transition (EMT) immune escape as well as a tumor-promoting microenvironment. The immunohistochemical study gives evidence of more IL-17+ cells both within tumor nests and stroma in OSCC tissues when compared to those in nonmalignant controls [20]. IL-17 levels were considerably greater in EGFR-overexpressing tumors than in TP53-mutant ones p=0.035, indicating a possible cross-talk between oncogenic pathways and inflammatory pathways. This fulfills the first leg of the hypothesis that EGFR on activation increases pro-inflammatory cytokines making more available mediators through which IL-17 can promote tumor progression while TP53 mutation channels carcinogenesis through different, less inflammatory pathways [21]. Clinically, the predominance of mucositis 42.9%, lichenoid reaction 17.9%, aphthous-like ulcers 10.7% and xerostomia 39.3% among dysfunctions underscore inflammatory and immunological pathways in the pathogenesis of OSCC. Higher IL-17 levels with mucosal changes (OR = 1.24; p=0.012) and pigmentation alterations (OR = 1.27; p=0.006) from a logistic regression analysis support that IL-17 is directly involved in epithelial integrity and vascular permeability as well as melanocyte activation most probably through upregulation of INFs and melanogenic mediators by IL-17 [22]. The borderline association with salivary gland dysfunction p=0.050 indicates possible involvement of IL-17 in glandular inflammation which needs further studies for clarification. Inhibitors of the IL-17 pathway are already in therapeutic use for autoimmune conditions; therefore, they may have adjunctive therapeutic potential in the management of tumor-promoting inflammation and mucosal symptoms in OSCC [23]. Preclinical data support that inhibition of IL-17 can further sensitize PD-1 inhibition and reduce progression of oral tumors [24-26]. However, preclinical considerations must include potential risks based on the highly complex and context-dependent functions of IL-17 in mucosal immunity related to tissue repair and host defense [27]. Limitations involved the cross-sectional design, which limited causal inference and potential confounders such as oral hygiene, microbiome shifts, and other concurrent medications. Future longitudinal and multi-marker studies-in which salivary cytokine panels are integrated with molecular tumor profiles [e.g., EGFR, TP53]-are mandated in the interest of further algorithmic refinements in diagnostics and IL-17–targeted interventions in OSCC.

CONCLUSIONS

Salivary levels of IL-17 are increased in patients with OSCC. Salivary IL-17 can be considered a feasible, non-invasive biomarker for disease presence and follow-up. It comes out higher in tumors with overexpression of EGFR; thereby, it denotes possible relationships between tumor biology and inflammatory activity. Mucosal and pigmentation changes relate it to a possible role in some oral lesion developments.

REFERENCES

1. Zielińska K, Fiedor P, Rabska-Pietrzak R, et al. Salivary IL-17A, IL-17F, and TNF-α are associated with disease advancement in patients with oral and oropharyngeal cancer. J Immunol Res. 2020;2020:Article 3928504. doi: 10.1155/2020/3928504. DOI
2. Almahmoudi R, Mian B, Cima I, et al. Extracellular interleukin-17F has a protective effect in oral tongue squamous cell carcinoma. Head Neck. 2018;40(9):2155-2165. doi: 10.1002/hed.25207 DOI
3. Ferrara SD, de la Rosa A. Salivary cytokines as biomarkers for oral squamous cell carcinoma: A systematic review. Int J Mol Sci. 2021;22(13):6795. doi: 10.3390/ijms22136795. DOI
4. Ladjevac N, Milovanovic M, Jevtovic A, et al. The Role of IL-17 in the Pathogenesis of Oral Squamous Cell Carcinoma. Int J Mol Sci. 2023;24(12):9874. doi:10.3390/ijms24129874. DOI
5. Jiang L, Ma Z, Song L, et al. Expression of interleukin-17 in oral tongue squamous cell carcinoma and its effect on biological behavior. Sci Rep. 2025;15(1):3195. doi:10.1038/s41598-025-87637-w DOI
6. Avadhani AV, Parachuru VPB, Milne T, Seymour GJ, Rich AM. Multiple cells express interleukin 17 in oral squamous cell carcinoma. J Oral Pathol Med. 2017;46(1):39-45. doi:10.1111/jop.12465. DOI
7. Wongpattaraworakul W, Gibson-Corley KN, Choi A, et al. Prognostic Role of Combined EGFR and Tumor-Infiltrating Lymphocytes in Oral Squamous Cell Carcinoma. Front Oncol. 2022;12:885236. doi:10.3389/fonc.2022.885236. DOI
8. Anwar N, Pervez S, Moatter T, et al. Analysis of EGFR signaling pathway; miRNAs and inflammatory biomarkers in a high-risk oral cancer population in Pakistan - An exploratory study. PLoS One. 2026;21(2):e0340264. doi:10.1371/journal.pone.0340264. DOI
9. Chen Y. The role of IL17 in cancer progression. AJST. 2025;18(1):183-190. doi:10.54097/bw9x3h56. DOI
10. Warnakulasuriya S. Global epidemiology of oral and oropharyngeal cancer. Oral Oncol. 2009; 45(4–5):309-316. doi: 10.1016/j.oraloncology.2008.06.002. DOI
11. Warnakulasuriya S, Kerr AR. Oral Cancer Screening: Past, Present, and Future. J Dent Res. 2021;100(12):1313-1320. doi: 10.1177/00220345211014795 DOI
12. Rivera C. Essentials of oral cancer. Int J Clin Exp Pathol. 2015;8(9):11884-11894. Published 2015 Sep 1.
13. Petersen PE. Oral cancer prevention and control - the approach of the World Health Organization. Oral Oncol. 2009;45(4-5):454-460. doi:10.1016/j.oraloncology.2008.05.023 DOI
14. Li C, Dong X, Li B. Tumor microenvironment in oral squamous cell carcinoma. Front Immunol. 2024;15:1485174. doi:10.3389/fimmu.2024.1485174 DOI
15. Kuen DS, Kim BS, Chung Y. IL-17-Producing Cells in Tumor Immunity: Friends or Foes?. Immune Netw. 2020;20(1):e6. doi:10.4110/in.2020.20.e6. DOI
16. Zhang JP, Yan J, Xu J, et al. Increased intratumoral IL-17-producing cells correlate with poor survival in hepatocellular carcinoma patients. J Hepatol. 2009;50(5):980-989. doi:10.1016/j.jhep.2008.12.033. DOI
17. Lingen MW, Kalmar JR, Karrison T, Speight PM. Critical evaluation of diagnostic aids for the detection of oral cancer. Oral Oncol. 2008;44(1):10-22. doi:10.1016/j.oraloncology.2007.06.011. DOI
18. Markopoulos AK. Current aspects on oral squamous cell carcinoma. Open Dent J. 2012;6:126-130. doi: 10.2174/1874210601206010126. DOI
19. He D, Li H, Yusuf N, et al. IL-17 promotes tumor development through the induction of tumor promoting microenvironments at tumor sites and myeloid-derived suppressor cells. J Immunol. 2010;184(5):2281-2288. doi: 10.4049/jimmunol.0902574. DOI
20. Zhao J, Chen X, Herjan T, et al. The role of interleukin-17 in tumor development and progression. J Experiment Med. 2020;217(1):e20190297. doi: 10.1084/jem.20190297. DOI
21. Tampa M, Mitran MI, Mitran CI, et al. Mediators of Inflammation - A Potential Source of Biomarkers in Oral Squamous Cell Carcinoma. J Immunol Res. 2018;2018:1061780. doi:10.1155/2018/1061780. DOI
22. Hwang SH, Woo JS, Moon J, et al. IL-17 and CCR9+α4β7- Th17 Cells Promote Salivary Gland Inflammation, Dysfunction, and Cell Death in Sjögren›s Syndrome. Front Immunol. 2021;12:721453. doi: 10.3389/fimmu.2021.721453. DOI
23. Zhang X, Li B, Lan T, et al. The role of interleukin-17 in inflammation-related cancers. Front Immunol. 2025;15:1479505. doi: 10.3389/fimmu.2024.1479505. DOI
24. Brailo V, Vucićević-Boras V, Cekić-Arambasin A, Alajbeg IZ, Milenović A, Lukac J. The significance of salivary interleukin 6 and tumor necrosis factor alpha in patients with oral leukoplakia. Oral Oncol. 2006;42(4):370-373. doi:10.1016/j.oraloncology.2005.09.001 DOI
25. Ferris RL, Blumenschein G Jr, Fayette J, et al. Nivolumab for Recurrent Squamous-Cell Carcinoma of the Head and Neck. N Engl J Med. 2016;375(19):1856-1867. doi:10.1056/NEJMoa1602252 DOI
26. Koh C H, Kim B S, Kang C Y, et al. IL-17 and IL-21: Their Immunobiology and Therapeutic Potentials. Immune Network. 2024; 24(1): e2. doi: 10.4110/in.2024.24.e2. DOI
27. Hadi WS, Salman RS, Al-Fahham AA, et al. Evaluation of IL-17 and IL-35 in patients with giardiasis in Thi-Qar province, Iraq. J Med Life. 2022;15(9):1096-1099. doi:10.25122/jml-2021-0328. DOI

CONFLICT OF INTEREST
The Authors declare no conflict of interest

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