

The 6th Asia Pacific Anti-Aging Conference



Lecture

Hands-on Sessions

2023

APAC

DENTISTRY

ABSTRACT BOOK

1 - 3 December

Daegu, South Korea

December 1 (Fri) – 2 (Sat) EXCO (Daegu Exhibition & Convention Center)

December 3 (Sun) Kyungpook National University School of Dentistry

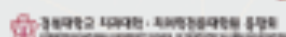
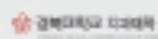
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Overview

Date	December 1(Fri) ~ 2(Sat)	December 3(Sun)
Venue	Room 325, EXCO, Daegu, Korea	Kyungpook National University School of Dentistry, Daegu, Korea
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Committee

Position	Name	Affiliation
Chair	Duhyeong Lee	Professor, Kyungpook National University School of Dentistry/ Dental Hospital
Supporting organization Director	Jaemok Lee	Dean, Kyungpook National University School of Dentistry
	Daegeun Kwon	President, Kyungpook National University Dental Hospital
	Chihong Ahn	President, Kyungpook National University School of Dentistry Alumni Association
	Seho Park	President, Taegu Dental Association
Member	Myunguk Jin	Professor, Kyungpook National University School of Dentistry/ Dental Hospital
	Wonhyuk Lee	Vice President, Taegu Dental Association
	Jinwook Kim	Professor, Kyungpook National University School of Dentistry/ Dental Hospital
	Yonggun Kim	Professor, Kyungpook National University School of Dentistry/ Dental Hospital
	Hojin Kim	Professor, Kyungpook National University School of Dentistry/ Dental Hospital
	Youngjoon Cho	Director, Mac Dental Clinic
	Joongbal Kwon	President, International Council for Medical Cultural Exchange
	Shinmo Kang	Director, Modern Dentist Shanghai, China

Targeting IL-1 β and IL-17 in Periodontitis-Associated Oral Senescence Pathways

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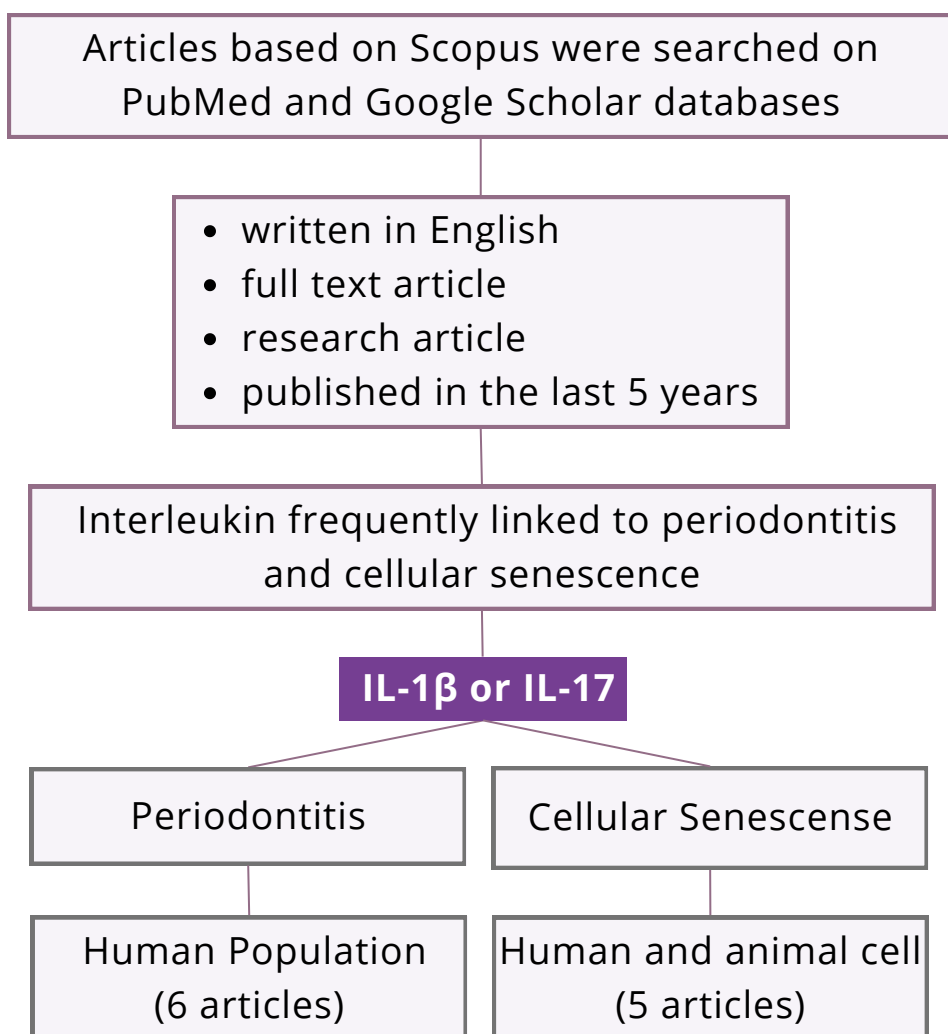
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BACKGROUND

Periodontitis, an advanced stage of periodontal disease characterized by chronic and irreversible inflammation, is associated with an extensive network of cytokines. By inducing osteoclast differentiation and activating collagenase and the receptor activator of nuclear factor-kappa B ligand (RANKL), the pro-inflammatory cytokines cause tissue damage and bone resorption (Aleksandrowicz P et al., 2021). Interleukin-1 β (IL-1 β) and interleukin-17 (IL-17) are known as immunological system and inflammation-related molecules that are thought to be the primary mediators of inflammation in periodontitis (Al-Taweel et al., 2021). The persistent inflammatory process in periodontitis results in significant genomic damage and the development of senescent phenotypes in oral tissues.

METHOD



RESULT

Table 1. The secretion of IL-1 β and IL-17 in healthy individual and periodontitis patient

STUDY	SUBJECT (n)	CONCENTRATION (pg/mL)	
		HEALTHY	PERIODONTITIS
IL-1β secretion			
Aleksandrowicz et al., 2021	189	16,90 \pm 18,65	61,04 \pm 41,41
Al-Taweel et al., 2021	80	585,11 \pm 53,19	1356,38 \pm 132,98
Kim et al., 2021	33	94,55 \pm 96,93	216,98 \pm 180,81
IL-17 secretion			
Kaczynski et al., 2019	106	12,64 \pm 28,28	49,43 \pm 75,40
Wankhede et al., 2022	45	0,64 \pm 0,23	1,96 \pm 1,71
Kalate et al., 2018	69	38,18 \pm 11,23	53,46 \pm 45

Table 2. Secretion of Senescence-Associated Secretory Phenotype Induced by IL-1 β and IL-17

STUDY	CELL LINE	TARGET	CONTROL	INTERVENTION
administration of IL-1β (10 ng/mL)				
Huang et al., 2021	C28/12	SA- β -Gal	0,99 \pm 0,12 cell	3,19 \pm 0,33 cell
		PAI-1	0,99 \pm 0,09	2,79 \pm 0,20
		p21	1,01 \pm 0,09	2,49 \pm 0,27
Huang et al., 2022	HNPC	SA- β -Gal	7,18 \pm 0,21 %	27,46 \pm 1,06 %
		p16	0,97 \pm 0,05	2,51 \pm 0,39
		p53	0,97 \pm 0,06	3,54 \pm 0,39
Zhao et al., 2021	hVSMCs	SA- β -Gal	0,99 \pm 0,11	3,10 \pm 0,33
		p16	1,01 \pm 0,10	2,59 \pm 0,30
		p21	1,00 \pm 0,10	2,87 \pm 0,35
administration of IL-17 (10 ng/mL)				
Wang et al., 2021	ATDC5	SA- β -Gal	1,01 \pm 0,10 cell	3,69 \pm 0,39 cell
		p21	24,81 \pm 7,52	77,44 \pm 8,27
administration of IL-17A (5 ng/mL)				
Zhang et al., 2021	MAECs	SA- β -Gal	5,23 \pm 4,58 %	71,90 \pm 13,73 %
		p53	17,01 \pm 1,84	30,80 \pm 3,22
		p21	17,77 \pm 1,65	36,78 \pm 2,48

CONCLUSION

- IL-1 β and IL-17 act as crucial mediators in the development of oral senescence linked to periodontitis. Their excess production induces SASP, contributing to cellular senescence in periodontal tissues.
- The elevated levels of inflammatory cytokines associated with periodontitis create a mild state of chronic inflammation in aging bodies. This connection underscores the systemic impact of periodontal disease on inflammatory processes.
- Understanding the mechanisms behind oral senescence in periodontitis is vital for developing targeted therapies. These interventions aim to counteract the detrimental effects of chronic inflammation, emphasizing the significance of such research for overall oral health.

REFERENCE

- Al-Taweel, F. B. H., Saliem, S. S., Abd, O. H., & Whawell, S. A. (2021). Assessment of Serum Interleukin-1 β and Interleukin-6 Levels in Patients with Chronic Periodontitis and Coronary Heart Disease. *European Journal of General Dentistry*, 10, 78-83.
- Aleksandrowicz, P., Brzezińska-Błaszczak, E., Kozłowska, E., Żelechowska, P., Borgonovo, A., & Justyna, A. (2021). Analysis of IL-1 β , CXCL8, and TNF- α levels in the crevicular fluid of patients with periodontitis or healthy implants. *BMC Oral Health*, 21, 120.
- Huang, X., Chen, C., Chen, Y., Xu, J., & Liu, L. (2022). Omentin-1 alleviate interleukin-1 β (IL-1 β)-induced nucleus pulposus cells senescence. *Bioengineered*, 13(5), 13849-13859.
- Kaczyński, T., Wroński, J., Glusko, P., Kryczka, T., Miskiewicz, A., Górski, B., Radkowski, M., Strzemecki, D., Grieb, P., & Górka, R. (2019). Salivary interleukin 6, interleukin 8, interleukin 17A, and tumour necrosis factor levels in patients with periodontitis and rheumatoid arthritis. *Central European Journal of Immunology*, 44(3), 269-276.
- Kalate, F. A., Gholami, L., Alijani, E., Hedayatpanah, M., & Kosari, S. (2018). Level of interleukin-17 in gingival crevicular fluid of patients with chronic periodontitis. *World Journal of Dentistry*, 9(6), 495-499.
- Kim, J. Y., Kim, K. R., & Kim, H. N. (2021). The potential impact of salivary IL-1 on the diagnosis of periodontal disease: A pilot study. *Healthcare (Switzerland)*, 13(9), 729.
- Wang, B., Sun, W., Bi, K., Li, Y., & Li, F. (2021). Apremilast prevents IL-17-induced cellular senescence in ATDC5 chondrocytes mediated by SIRT1. *International Journal of Molecular Medicine*, 47(3), 12.
- Wankhede, A. N., & Dhadse, P. V. (2022). Interleukin-17 levels in gingival crevicular fluid of aggressive periodontitis and chronic periodontitis patients. *Journal of Indian Society of Periodontology*, 26(6), 552-556.
- Zhang, L., Liu, M., Liu, W., Hu, C., Li, H., Deng, J., Cao, Q., Wang, Y., Hu, W., & Li, Q. (2021). Th17/IL-17 induces endothelial cell senescence via activation of NF- κ B/p53/Rb signaling pathway. *Laboratory Investigation*, 101, 1418-1426.
- Zhao, J., He, X., Zuo, M., Li, X., & Sun, Z. (2021). Anagliptin prevented interleukin 1 β (IL-1 β)-induced cellular senescence in vascular smooth muscle cells through increasing the expression of sirtuin1 (SIRT1). *Bioengineered*, 12(1), 3968-3977.
- Zhusong, H., Lan, J., & Xi, G. (2021). Feprazone Mitigates IL-1 β -Induced Cellular Senescence in Chondrocytes. *ACS Omega*, 6, 9442-9448.



Targeting IL-1 β and IL-17 in Periodontitis-Associated Oral Senescence Pathways

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Abstract

Background: Periodontitis, an advanced stage of periodontal disease characterized by chronic and irreversible inflammation, is associated with an extensive network of cytokines. The persistent inflammatory process in periodontitis results in significant genomic damage and the development of senescent phenotypes in oral tissues.

Methods: In this study, a comprehensive literature search was conducted using the MEDLINE database via PubMed, encompassing records up to 2018. The experimental group consisted of studies involving periodontitis patients, while the control group included studies on individuals with healthy periodontal conditions. Additionally, in vitro studies investigating Senescence Associated Secretory Phenotype (SASP) were incorporated.

Results: The findings revealed a substantial increase in the secretion of interleukin-1 β (IL-1 β) and interleukin-17 (IL-17) in periodontitis patients in comparison to individuals with healthy periodontal conditions. In vitro investigations demonstrated that IL-1 β and IL-17 played a direct role in inducing the secretion of Senescence Associated Secretory Phenotype (SASP) components, including SA- β -gal, p21, p53, plasminogen activator inhibitor-1 (PAI-1), and p16.

Conclusion: This study provides evidence that IL-1 β and IL-17 are key mediators in the pathogenesis of oral senescence in the context of periodontitis. The excessive production of these cytokines contributes to the induction of SASP, leading to cellular senescence in the periodontal tissues. Understanding the mechanisms underlying oral senescence in periodontitis is crucial for developing targeted therapeutic interventions to mitigate the detrimental effects of this chronic inflammatory disease on oral health.