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Lecture

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ABSTRACT BOOK

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

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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-1β (IL-1β) 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

Articles based on Scopus were searched on PubMed and Google Scholar databases

- written in English
- full text article
- research article
- published in the last 5 years

Interleukin frequently linked to periodontitis and cellular senescence

IL-1β or IL-17

Periodontitis	Cellular Senescense
Human Population (6 articles)	Human and animal cell (5 articles)

RESULT

Table 1. The secretion of IL-1β 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 ± 18,65	61,04 ± 41,41
Al-Taweel et al., 2021	80	585,11 ± 53,19	1356,38 ± 132,98
Kim et al., 2021	33	94,55 ± 96,93	216,98 ± 180,81
IL-17 secretion			
Kaczynski et al., 2019	106	12,64 ± 28,28	49,43 ± 75,40
Wankhede et al., 2022	45	0,64 ± 0,23	1,96 ± 1,71
Kalate et al., 2018	69	38,18 ± 11,23	53,46 ± 45

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

CONCLUSION

- IL-1β 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.

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Targeting IL-1β 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-1β (IL- 1β) and interleukin-17 (IL-17) in periodontitis patients in comparison to individuals with healthy periodontal conditions. In vitro investigations demonstrated that IL-1β 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-1β 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.