10TH INTERNATIONAL CONGRESS ON AUTOIMMUNITY

APRIL 6–10, 2014, LEIPZIG, GERMANY

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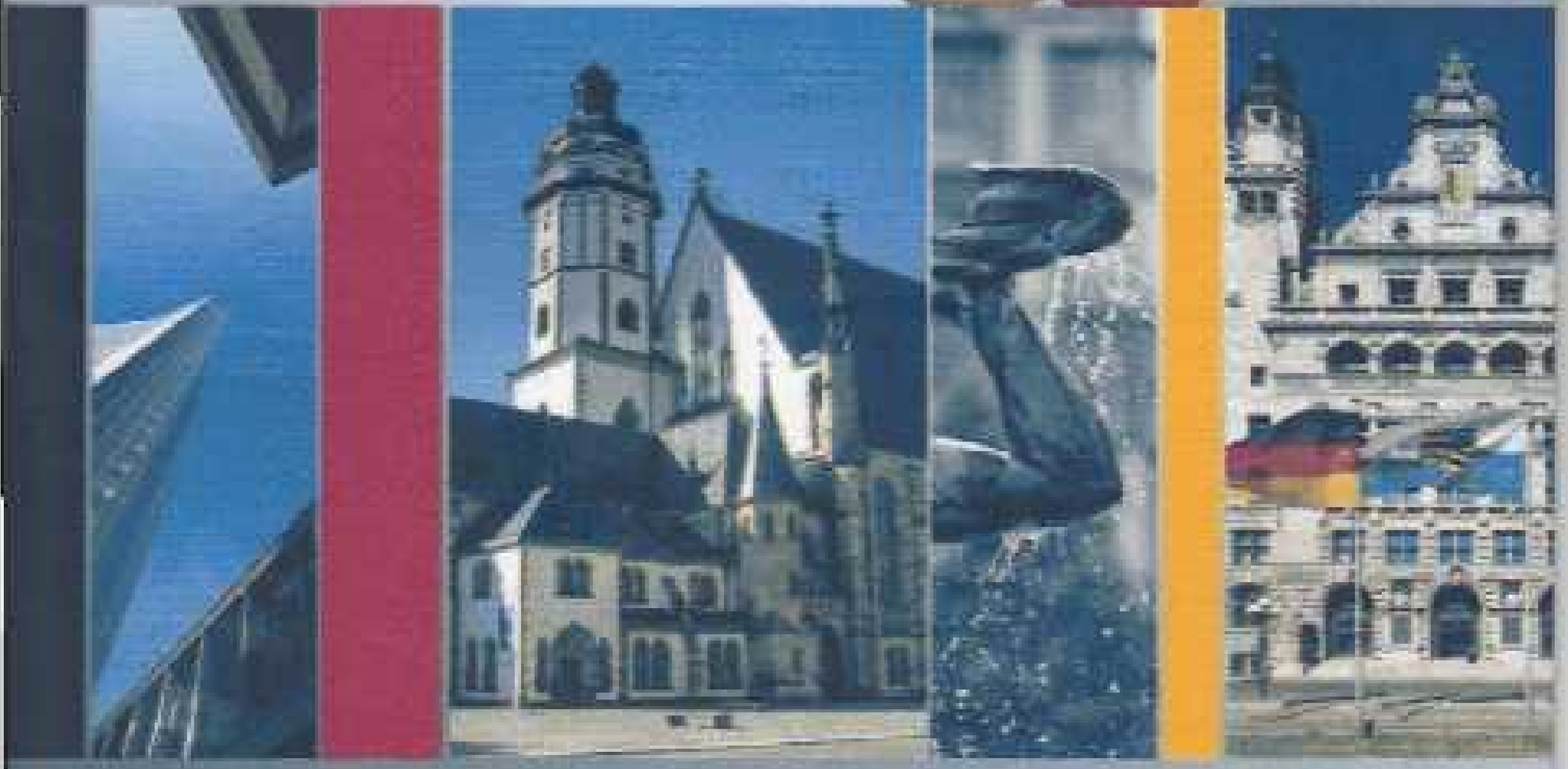

WELCOME MESSAGE

Dear Friends,

The International Congress on Autoimmunity has reached a historical moment: in April 2016 more than 2000 of the world's autoimmunologists have gathered for the 10th time to exchange knowledge about the more than 80 autoimmune diseases.

The meeting point this time is the artistic city of Leipzig, Germany, known for its long tradition in trade fairs and its compelling selection of museums, musical events and other cultural offerings.

Our loyal participants are already familiar with the high level of medical science that awaits them at the International Congresses on Autoimmunity and our newcomers will be impressed by the diversity of excellent sessions offered on a variety of topics, ranging from basic research to novel diagnostic and treatment methods of autoimmune diseases. This year's Congress introduces a variety of hot subjects for the first time, from spicy food and cannabis to obesity, smoking, the microbiome, novel peptides and revolutionary therapies.

The International Congress on Autoimmunity is the biggest multidisciplinary congress that discusses all aspects of the related diseases under one roof, offering courses and lectures by some of the world's most distinguished experts. At the same time, the Congress prides itself on providing a stage for young, upcoming talents to present their research to a first-rate audience.

Join us and enjoy the inspiring atmosphere of medical science among old and new colleagues; share, learn and network to build the future of autoimmunology at the 10th International Congress on Autoimmunity!

Sincerely,

Yehuda Shoenfeld, MD, FRCP, MaACR
Congress President

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TIMETABLE

PLENARY SESSIONS	NETWORKING EVENTS	PARALLEL SESSIONS	INDUSTRY SPONSORED SESSIONS	COURSES

WEDNESDAY, APRIL 6

	Hall 1	Hall MP3&4	Hall 2	Hall 3	Hall 4
12:00 – 12:30			4th INT'L SYMPOSIUM ON VACCINES in collaboration with CMSRI		
12:30 – 13:00				BASIC IMMUNOLOGY COURSE Abul K. Abbas, USA	
13:00 – 13:30					
13:30 – 14:00		Industry session (not included in main event CME/CPD credit)			
14:00 – 14:30					
14:30 – 14:50			Coffee Break	Coffee Break	
14:50 – 15:00			4th INT'L SYMPOSIUM ON VACCINES in collaboration with CMSRI		
15:00 – 15:30				BASIC IMMUNOLOGY COURSE Abul K. Abbas, USA	
15:30 – 16:10					Industry session (not included in main event CME/CPD credit)
16:10 – 16:30			Coffee Break		
16:30 – 17:00			4th INT'L SYMPOSIUM ON VACCINES in collaboration with CMSRI		
17:00 – 17:30					
17:30 – 18:00					
18:00 – 19:45	Opening Ceremony followed by Welcome Reception (in the Exhibition Area)				

THURSDAY, APRIL 7

	Hall 1	Hall MP3&4	Hall 2	Hall 3	Hall 4	Hall 5
08:00 – 10:00	**PL01** PLENARY SESSION					
10:00 – 10:30	Coffee break, Exhibition and E-Poster sessions					
10:30 – 12:30	**PS01** Etiology and pathogenesis of autoimmunity	**PS02** Celiac disease: novelties in 2016	**PS03** IVIg: the best biologic?	**PS04** Standardization and harmonization in autoantibody testing	**PS05** The m[illegible] and oral mucosa	**PS06** Citrullination (CCP)
12:30 – 14:00		Industry Session (not included in main event CME/CPD credit)	Lunch break, Exhibition and E-Poster viewing			
14:00 – 16:00	**PS07** Hygiene theory, microbiome, probiotics	**PS08** Novel biologics and biosimilars	**PS09** B cells: pathogenesis and suppression	**PS10** Immunomodulation by vitamin D and IVIg	**PS11** Systemic sclerosis: new approaches	**PS12** Novel autoantigens: DFS70, 14-3-3η
16:00 – 16:30	Coffee break, Exhibition and E-Poster sessions					
16:30 – 18:30	**PS13** Vasculitides	**PS14** Vaccines and autoimmunity	**PS15** Autoimmune pathways in multiple sclerosis, myasthenia gravis and the central nervous system	**PS16** Induction, determination and the pathogenic role of autoantibodies	**PS17** The autoimmune basis of reproductive failure	**PS18** Autoimmune dermatological diseases

FRIDAY, APRIL 8

	Hall 1	Hall MP3&4	Hall 2	Hall 3	Hall 4	Hall 5	Hall MP1
08:00 – 10:00	**PL02** PLENARY SESSION						
10:00 – 10:30	Coffee break, Exhibition and E-Poster sessions						
10:30 – 12:30	**PS19** T cells in autoimmunity	**PS20** Diagnostics: pathogenesis and autoantibody standardization	**PS21** SLE: Is it the classical autoimmune disease?	**PS22** Kidney involvement in autoimmunity	**PS23** Autoimmunity in dermatology	**PS24** Experimental animal models of autoimmune diseases	
12:30 – 14:00		Industry session not included in main event CME/CPD credit	Lunch break, Exhibition and E-Poster viewing				
14:00 – 16:00	**PS25** Prediction, monitoring and personalized medicine	**PS26** EASI session: ANCA and ANCA associated vasculitis	**PS27** Autoimmune aspects of neurological diseases	**PS28** Peptides and new molecules in therapeutics	**PS29** Autoimmune liver diseases	**PS30** Type 1 diabetes mellitus	**PS30B** New horizons in autoimmune therapy
16:00 – 16:30	Coffee break, Exhibition and E-Poster sessions						
16:30 – 18:30	**PS31** Environmental factors and sex hormones in autoimmunity	**PS32** Rheumatoid arthritis, challenges in the new era	**PS33** Genetics and epigenetics of autoimmune diseases	**PS34** European forum on antiphospholipid antibodies	**PS35** Big data analysis, registries and epidemiological studies in autoimmunity	**PS36** Systemic onset juvenile idiopathic arthritis and hereditary periodic fever syndromes	

SATURDAY, APRIL 9

	Hall 1	Hall MP3&4	Hall 2	Hall 3	Hall 4	Hall 5
08:00 – 10:00	**PL03** PLENARY SESSION					
10:00 – 10:30	Coffee break, Exhibition and E-Poster sessions					
10:30 – 12:30	**PS37** Systemic lupus erythematosus, the challenge	**PS38** Infection and autoimmunity: two edges of the sword	**PS39** Highlights in diagnostics	**PS40** Innate immunity and natural autoantibodies IVIg	**PS41** UCTD, MCTD and other connective tissue diseases	**PS42** Pregnancy and autoimmunity
12:30 – 14:00		Industry Session not included in main event CME/CPD credit	Lunch break, Exhibition and E-Poster viewing			
14:00 – 16:00	**PS43** ANA diagnostics and immunofluorescence	**PS44** T-reg, B-reg, tolerance and autoimmunity	**PS45** Autoimmunity and endocrinology	**PS46** Novel antibodies in autoimmune diseases	**PS47** Epstein-Barr virus driven inflammatory diseases: from primary immunodeficiencies to autoimmune diseases (NEDAI session)	**PS48** Pearls in autoimmunity: top candidates for the MA Award 2016
16:00 – 16:30	Coffee break, Exhibition and E-Poster sessions					
16:30 – 18:30	**PS 49** New therapeutic avenues in autoimmune diseases	**PS50** APS: Diagnostics and challenges for the future	**PS51** Autoimmune syndrome induced by adjuvants (ASIA syndrome)	**PS52** The autoimmune aspects of psoriasis, myositis and atherosclerosis	**PS53** The autoimmune side of inflammatory arthritis and autoinflammation	**PS54** Cytokines and autoimmunity

SUNDAY, APRIL 10

	Hall MP3&4	Hall 2	Hall 3	Hall 4	Hall 5
08:00 – 10:00	**PL04** PLENARY SESSION				
10:00 – 10:30	Coffee Break				
10:30 – 12:30	**PS55** Sjogren's syndrome	**PS56** Immunomodulation	**PS57** The autoimmune origin of celiac and gastrointestinal diseases	**PS58** New autoimmune diseases: cancer and autoimmunity	**PS59** IL-6 and JAK inhibition: new achievements

CME/CPD Certificate

This is to certify that

Dr. Nila Kasuma

(first, last name, degree)

participated in the

10th International Congress on Autoimmunity (Autoimmunity 2016)

Leipzig, Germany
April 6-10, 2016

As speaker in Short Oral Presentation

Y. Shoenfeld

Yehuda Shoenfeld, MD, FRCP, MaACR
Congress President

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The 10th International Congress on Autoimmunity (Autoimmunity 2016) is accredited by the European Accreditation Council for Continuing Medical Education (EACCME) to provide the following CME activity for medical specialists. The EACCME is an institution of the European Union of Medical Specialists (UEMS): www.uems.net

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EPD16-THE BUGS AND US: INTERRELATIONSHIP OF AUTOIMMUNITY

Abstract: 516

COMPARASION OF INTERLEUKIN-1 BETA (IL-1 B) IN SALIVA ON PERIODONTAL DISEASE

N. Kasuma[1], *A. Aida Fitria*[1]
[1]*Andalas University, Oral Biology, Padang, Indonesia*

Several studies have demonstrated the involvement of autoimmune responses in periodontal disease. Evidences of involvement of immunopathology have been reported in periodontal disease. Etiology and pathogenesis of periodontal disease multifactorial. increased of interleukin-1 beta (IL-1 β) levels is pro-inflammatory cytokines that may affect the destruction and tissue damage periodontal. Interleukin-1 beta (IL-1 β) was increased in the blood and saliva of patients peiodontitis. Periodontal disease affecting the gingival tissues is gingivitis, and when it is not properly treated, will be a destructive periodontitis of periodontal tissue structure where the periodontal tissues and loss of the dental arch and facial abnormalities. This study aims to prove the relationship of interleukin-1 beta (IL-1 β) levels in saliva on periodontal disease. In this study involving 30 people with a sample of 15 healthy samples and 15 periodontitis samples. Saliva was used as specimen in this study because saliva colecction process was easy and painless for the patient. Interleukin-1 beta (IL-1 β) was tested using ELISA technique. In a cross-sectional study comparing levels of interleukin-1 beta (IL-1 β) to sample healthy and periodontitis in each group. The data obtained were analyzed using SPSS 17 for parametric test by unpaired T test, whereas for non-parametric test with Kolmogorov Smirnov with a confidence level of 5%. This study concluded that there is a significant correlation between the levels of interleukin-1 beta (IL-1 β) in periodontal disease in saliva . The results of this study can describe comparasion IL-1β levels in healthy and periodontal diseases patients.

Comparison of interleukin-1 beta (IL-1 β) in saliva on periodontal disease

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1. Oral Biology Departement, Faculty of Denstistry, Andalas University, Padang, West Sumatra-Indonesia

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Introduction

- Several studies have demonstrated the involvement of autoimmune responses in periodontal disease.
- The innate immune system is one of the earliest mechanisms that provide immediate protection against infection or inflammation . The innate immune system in action through the recruitment of immune cells , activation of the complement system , the identification and removal of foreign substances , and the activation of the adaptive immune system .
- Phagocytic cells , such as polymorphonuclear neutrophils , monocytes , and macrophages, which are cells of the innate immune cells , triggering the release of chemicals such mediator- medoator cytokine tumor necrosis factor (TNF) and interleukin (IL) that activates various systems such as the complement system and phase response acute (Yucel et al, 2013)
- Interleukin (IL-1) is a multifactorial cytokine with potent inflammatory features. IL-1 is divided in two forms as IL-1α and IL-1β, of which IL-1β appears to be the most potent agent having a catabolic effect on bone approximately tenfold compared to IL-1α.
- IL-1β causes redness in periodontal disease , due in part to the capillary wall permeability that occurs in response to this cytokine. IL-1β stimulates collagenase production , which in turn breaks down periodontal connective tissues and leads to the formation of osteoclast and alveolar bone loss. (Javed and Ahmed, 2013).

Aims

This study aims to prove the comparison of interleukin-1 beta (IL-1 β) levels in saliva on periodontal disease.

Methods

- This study was cross sectional study. Samples were taken in Dental Faculty of Andalas University Dental Hospital. A total 30 people (15 healthy and 15 periodontitis samples; aged 17 - 35 years) . Periodontitis patients were diagnosed with chronic periodontitis with at least four sites with clinical attachment level (CAL) ≥ 4 mm and probing depth (PD) ≥ 5 mm . The teeth which were diagnosed is one of the teeth in anterior region.
- Unstimulated whole saliva (5 mL) was collected from all participants were asked to avoid oral hygiene activities.
- After collection it is important to keep samples cold in order to minimize bacterial growth and loss of IL-1 β in the specimen. Specimens were freeze at -20 C within 4 hours of collection .
- In this study reagent is Salimetrics Salivary IL-1 β wich contained IL-1 β standard and IL-1 β controls. Saliva samples then diluted 15 times in IL-1 β sample diluent.
- The data obtained were analyzed using parametric test by unpaired T test, whereas for non-parametric test with Kolmogorov Smirnov with a confidence level of 5%.

Result

- The concentration of IL-1β in saliva from periodontitis patients is significantly higher than in healthy samples (p< 0,0001) .with mean 235,61 ± 25, 06, higher than in healthy condition with mean 20,87 ± 18,68.
- Increasing of CAL and PD will increase the concentration of IL-1β in periodontitis patient.

Group	CAL	PD	IL-1β
Healthy			
Mean	0.32	0.31	20,87
n	15	15	15
Standard deviation	0.11	0.23	18,68
Minimum	0.29	0.27	4,35
Maximum	0.42	0.40	99,23
Median	0.41	0.44	45,56
Periodontitis			
Mean	6.00	6.00	235,61
n	15	15	15
Standard deviation	0.34	0.15	25,06
Minimum	5	6	124,75
Maximum	7	8	298,33
Median	6.00	7.00	118,40

Discussion and Conclusion

- The level of IL-1β increased in GCF in accordance with the increase of the periodontal inflammation
- It has been established that the severity of periodontal disease is dependent on a dynamic equilibrium of interactions between the mi-crobial challenge and host immune inflammatory responses.
- All the periodontopathogenic bacteria as well as extracted lipopolysaccharides (LPS) have primarily been shown to stimulate mo-nocytes to produce cytokines such as IL-1, IL-1β.

Yucel OO, Berker E, Mescil L, Eratalay K, Tepe E, Teczan I. Association of interleukin-1 beta (+3954) gene polymorphism and gingival crevicular fluid levels in patients with aggressive and chronic periodontitis. Genet Couns.2013;24(1):21-35.

Fawad Javed and Asma Ahmed (2013), “Proinflammatory Cytokines in the Saliva, Gingival Crevicular Fluid and Serum of Diabetic Patients with Periodontal Disease,” Journal of Research and Practice in Dentistry, Vol. 2013 (2013), Article ID 956990

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