



# IADR

INTERNATIONAL ASSOCIATION  
FOR DENTAL, ORAL, AND  
CRANIOFACIAL RESEARCH  
ASIA/PACIFIC REGION

## Second Workshop of IADR APR Mentor Mentee Programme

**MARCH 27, 2025**

2 p.m. – 5 p.m. Brisbane (UTC +10)

1 p.m. – 4 p.m. Seoul/Tokyo (UTC +9)

12 p.m. – 3 p.m. China (UTC +8)

9:30 a.m. 12:30 p.m. New Delhi (UTC +5:30)

9 a.m. – 12 p.m. Karachi (UTC +5)

**Join Virtually on Zoom at:**

**<https://uqz.zoom.us/j/8364649448>**



**PROGRAM**

March 27, 2 p.m. – 5 p.m. Brisbane Time (GMT+10)

**2 - 2.05 p.m.**

*Brief speech by IADR APR President*

**2:05 - 2.10 p.m.**

*Brief speech by IADR President*

**2:10 - 2:25 p.m.**

*Mentee from Japanese Division, **Jun Ohshima**, Osaka University | Handai · Graduate School of Dentistry, Japan*

*Title: Elucidation of the mechanism of inflammasome activation by the periodontal pathogen *Fusobacterium nucleatum**



**2:25 - 2:40 p.m.**

*Mentee from ANZ Division: **Nadeeka Udawatte**, The University of Queensland, Australia*

*Title: Temporal Dynamics of Probiotic Colonization on Composition and Functionality of Oral Microbiome using 3D Salivary Polymicrobial Biofilm Model*



**2:40 - 2:55 p.m.**

*Mentee from Indian Division: **Dr. Arpit Gupta**, Oral Health Sciences Centre, PGIMER Chandigarh, India*

*Title: Can colonization of the fetoplacental unit by oral bacteria, high levels of pro-inflammatory biomarkers and poor periodontal health lead to adverse pregnancy outcomes?*



**2:55 - 3:10 p.m.**

*Mentee from Korean Division: **Dong Hyun Park**, Seoul National University School of Dentistry, South Korea*

**Limosilactobacillus reuteri* lipoteichoic acid inhibits *Candida albicans* biofilm formation through interaction with Hsp70*





## PROGRAM (CONT)

March 27, 2 p.m. – 5 p.m. Brisbane Time (GMT+10)



**3:10 - 3:25 p.m.**

Mentee from Chinese Division: **Chuyi Han**, West China Hospital of Stomatology, Sichuan University

Title: Bio-inspired Reactive Oxygen Catalysts Potently Amplify Immune Checkpoint Blockade Therapy

**3:25 - 2:30 p.m.**

Short Break



**3:30 - 3:45 p.m.**

Mentee from SEA Division: **Kanokwan (Home) Sriwattanapong**, Faculty of Dentistry, Chulalongkorn University, Bangkok, Thailand

Title: Influence of Genetic Variants on Oral Inflammation and Microbial Shifts in Autoimmune Patients



**3:45 - 4 p.m.**

Mentee from Pakistan Section: **Muhammad Kashif**, Pakistan

Title: Human Papillomavirus in Saliva and Its Role in Oral and Oropharyngeal Cancers

**4 - 4:30 p.m.**

Hot Topics in Oral Microbiology & Immunology Research: Insights from Leading Experts (Interactive session)

### *Guiding stars Panel:*

- Prof. **Lakshman Samaranayake**, The University of Hong Kong, Hong SAR, China
- Prof. **Gordon Ramage**, Glasgow Caledonian University, United Kingdom
- Associate Prof. **Catherine Butler**, The University of Melbourne, Australia

**4:30 - 4:55 p.m.**

Strategies for Basic Science Researchers to Write Competitive Grants in a Clinical Funding Landscape (Interactive session)

**4:55 - 5 p.m.**

Future directions of the IADR APR Mentor Mentee Programme A/**Prof. Jaya Seneviratne**





## ABSTRACTS OF THE PRESENTATIONS

(In order of presentation)

### **I. Jun Ohshima, Osaka University, Handai Graduate School of Dentistry, Japan. Mentee from Japanese Division**

#### ***Elucidation of the mechanism of inflammasome activation by the periodontal pathogen *Fusobacterium nucleatum****

*Fusobacterium nucleatum* (*F. nucleatum*), an oral commensal bacterium, induces the production of inflammatory cytokines at the site of infection. Some reports indicate that *F. nucleatum* is associated with various systemic diseases and has been observed to colonize ectopically in the intestinal tract. However, the precise mechanism by which *F. nucleatum* induces inflammation remains unclear. In this study, we focused on the relationship between *F. nucleatum* and GBPs, one of the interferon-inducing genes to elucidate how *F. nucleatum* induces IL-1 $\beta$ . The human monocytic leukemia cell line THP-1 was infected with *F. nucleatum* at a multiplicity of infection 100, and post-infection cells and supernatants were collected and subjected to qPCR and ELISA. To clarify the relationship with GBPs, GBPs knockout macrophages and overexpression cells were generated and subjected to the same experiments. During *F. nucleatum* infection, IL-1 $\beta$  production was increased, which was enhanced by interferon gamma (IFN $\gamma$ ) stimulation. A low level of IL-1 $\beta$  production was observed in mouse GBPs knockout macrophages during infection compared to the wild type. In addition, immunoprecipitation with GBP antibodies revealed a dimerization band only with *F. nucleatum* infection in the presence of IFN $\gamma$  stimulation. Inflammation was also enhanced using drugs that promote GBP1 dimerization. These results suggest that the production of IL-1 $\beta$  in *F. nucleatum* is controlled by GBPs. In particular, dimerization of GBPs has been suggested to play an important role in inflammasome activation by *F. nucleatum* infection.

### **II. Nadeeka Udawatte, The University of Queensland, Australia, Mentee from ANZ Division**

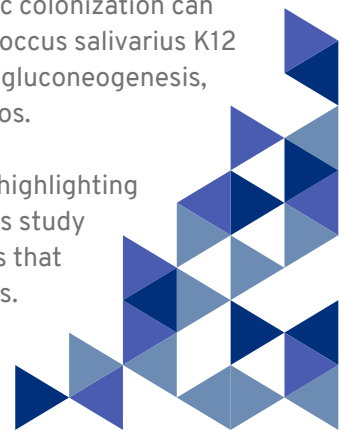
#### ***Temporal Dynamics of Probiotic Colonization on Composition and Functionality of Oral Microbiome using 3D Salivary Polymicrobial Biofilm Model***

**Background:** The advancement of multi-layered 3D scaffold models has improved understanding of polymicrobial biofilms, offering insights into probiotics' role in modulating biofilm dynamics and mitigating dysbiosis. While probiotics are widely studied for restoring microbial balance, few studies assess whether their benefits stem from colonization, microbial interactions, or transient effects. Our pilot study aimed to evaluate how the temporal dynamics of oral probiotic colonization influence the composition and functionality of the oral microbiome.

**Methods:** The colonization dynamics of the oral probiotic *Streptococcus salivarius* K12 (Ssk12) were evaluated within a saliva-derived oral biofilm model cultured on a 3D melt-electrowritten poly( $\epsilon$ -caprolactone) (MEW-PCL) scaffold. Strain-specific probes, confocal microscopy, and RT-qPCR were employed to assess colonization, while 16S rRNA and mRNA sequencing were used to analyze oral microbial composition and gene expression changes.

**Results:** The results revealed that a single administration of Ssk12 achieved colonization within the oral microbiome for up to one week. While overall microbial diversity remained stable, distinct shifts in community composition were observed during colonization and decolonization, favoring the growth of beneficial bacteria such as *Lactobacillus* and *Bifidobacterium*. Functional analysis of the oral microbiota revealed that even short-term probiotic colonization can significantly influence the functional composition of the oral microbiome. The success of *Streptococcus salivarius* K12 (Ssk12) colonization appears to rely on the regulation of specific metabolic adaptations, including gluconeogenesis, hexose metabolism, and pathways associated with redox balance, such as altered NADP/NAD ratios.

**Conclusion:** These findings underscore the transient impact of probiotics on the oral microbiome, highlighting the need for strategies to sustain colonization for long-term therapeutic benefits. Additionally, this study is the first to demonstrate the effectiveness of 3D MEW PCL scaffolds in culturing salivary biofilms that closely mimic both functional and compositional characteristics of naturally occurring oral biofilms.





## ABSTRACTS OF THE PRESENTATIONS

(In order of presentation)

### III. Arpit Gupta, Oral Health Sciences Centre, PGIMER Chandigarh, India, Mentee from Indian Division

#### *Can colonization of fetoplacental unit by oral bacteria, high levels of pro-inflammatory biomarkers and poor periodontal health lead to adverse pregnancy outcomes?*

Background: Periodontal disease, caused by bacteria like *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola*, triggers chronic gum inflammation and a systemic immune response, elevating pro-inflammatory markers such as IL2, IL4, IL6, IL10, IL17A, IFN- Gamma and TNF- $\alpha$ . These inflammatory markers can cross the placental barrier, potentially disrupting foetal development and increasing the risk of preterm labour. They may also impair nutrient and oxygen flow to the foetus, contributing to restricted growth and low birth weight.

Objectives: To assess the effectiveness of non-surgical periodontal therapy (NSPT) on the prevention of adverse pregnancy outcomes (like preterm births and LBW), change in oral & fetoplacental microbiome, pro-inflammatory biomarkers and the periodontal health status.

Methods: Total of 140 subjects fulfilling the eligibility criteria will be recruited under two treatment regimens: OHI + NSPT, and OHI alone. The saliva, dental plaque and foeto-placenta tissue samples will be collected and processed for identification of the oral microflora by using Matrix-assisted laser desorption ionization-time-of-flight mass spectrometry (MALDI-TOF MS) at four time-points (saliva- 18-22 weeks of gestation; 28-32 weeks of gestation; at the first post-partum visit; foeto-placental tissue-at the time of delivery). Serial dilutions of the identified individual isolates/strains will be done to assess the number of colonies. Inflammatory biomarkers (IL2, IL4, IL6, IL10, IL17A, IFN-Gamma and TNF) will be assessed from saliva and blood samples by using ELISA kits at three time-points (saliva- 18-22 weeks; 28-32 weeks; at first post-partum visit; blood -18-22 weeks; 28-32 weeks). Assessment of preterm birth, low birth weight of infant and periodontal health status by using OHI-S index, GR, PD and BOP will be done.

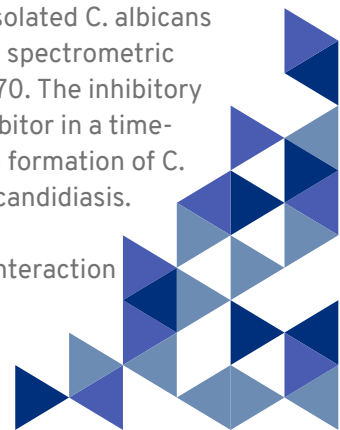
Expected outcome: This study could help in understanding the crucial relationship between periodontal health and pregnancy outcomes.

### IV. Dong Hyun Park, Seoul National University School of Dentistry, South Korea, Mentee from Korean Division

#### *Limosilactobacillus reuteri lipoteichoic acid inhibits Candida albicans biofilm through interaction with Hsp70*

*Candida albicans* is an opportunistic pathogen responsible for fungal infectious diseases, such as oral candidiasis. Its biofilm can exacerbate the severity of disease by developing physical and chemical barriers against medical treatments. Lipoteichoic acid (LTA) is a major cell wall component of Gram-positive bacteria, and LTAs are known to possess an anti-biofilm ability against various bacterial pathogens. However, the effects of LTAs on pathogenic fungal biofilm remain undetermined. In this study, we investigated the effect of LTA purified from *Limosilactobacillus reuteri* (Lre.LTA) on *C. albicans* biofilm formation. Lre.LTA inhibited *C. albicans* biofilm formation in a dose-dependent manner without affecting its growth. In addition, Lre.LTA also inhibited the biofilm formation of clinically isolated *C. albicans* strains and even effectively disrupted pre-formed biofilm of *C. albicans* as well. LTQ orbitrap mass spectrometric analysis showed that Lre.LTA mainly binds to the heat shock protein (Hsp) families, including Hsp70. The inhibitory effect of Lre.LTA on *C. albicans* biofilm formation was diminished by treatment with an Hsp70 inhibitor in a time- and dose-dependent manner. Furthermore, Lre.LTA potently suppressed the adhesion and biofilm formation of *C. albicans* and showed anti-biofilm effects in murine ex vivo and in vivo models for oral and vaginal candidiasis.

In conclusion, our results suggest that Lre.LTA inhibits *C. albicans* biofilm formation through the interaction with Hsp70 and could be an effective anti-biofilm agent against the *C. albicans* biofilm-related infectious diseases.





## ABSTRACTS OF THE PRESENTATIONS

(In order of presentation)

### **V. Chuyi Han, West China Hospital of Stomatology, Sichuan University, Mentee from Chinese Division**

#### ***Bio-inspired Reactive Oxygen Catalysts Potently Amplify Immune Checkpoint Blockade Therapy***

Clinical immune checkpoint blockade (ICB)-based immunotherapy of malignant tumors only elicits durable responses in a minority of patients, primarily due to the highly immunosuppressive tumor microenvironment. Although inducing immunogenic cell death (ICD) through reactive oxygen biocatalyst represents an attractive therapeutic strategy to amplify ICB, currently reported biocatalysts encounter insurmountable challenges in achieving high ROS-generating activity to induce potent ICD. Here, inspired by the natural catalytic characteristics of NADPH oxidases, the design of efficient, robust, and electron-rich Pt-based redox centers on the non-stoichiometric W18O49 substrates (Pt-WOx) to serve as bioinspired reactive oxygen biocatalysts to potently activate the ICD, which eventually enhance cancer immune responses and amplifies the ICB-based immunotherapy is reported. These studies demonstrate that the Pt-WOx exhibits rapid electron transfer capability and can promote the formation of electron-rich and low oxophilic Pt redox centers for superior reactive oxygen biocatalysis, which enables the Pt-WOx-based inducers to trigger endoplasmic reticulum stress directly and stimulate immune responses potently for amplifying the anti-PD-L1-based ICB therapy. This bioinspired design provides a straightforward strategy to engineer efficient, robust, and electron-rich reactive oxygen biocatalysts and also opens up a new avenue to create efficient ICD inducers for primary/metastatic tumor treatments.

### **VI. Kanokwan (Home) Sriwattanapong, Faculty of Dentistry, Chulalongkorn University, Bangkok, Thailand, Mentee from SEA Division**

#### ***Influence of Genetic Variants on Oral Inflammation and Microbial Shifts in Autoimmune Patients***

Autoimmune disorders such as Hyper-IgE syndrome, neutropenia, and systemic lupus erythematosus (SLE) often present with heightened susceptibility to infections, particularly in the oral cavity. These conditions disrupt immune regulation, potentially influencing both oral cellular behaviors and the microbial ecosystem, leading to chronic inflammation and impaired tissue repair. This research proposes a comprehensive investigation into how genetic variants associated with autoimmune diseases impact oral health by exploring their effects on oral cell behavior and microbial dysbiosis. Using next-generation sequencing, we will identify specific genetic variants in autoimmune patients and investigate their effects on oral cells through RNA sequencing (RNA-Seq). This will allow us to uncover transcriptional changes linked to inflammation, wound healing, cell proliferation, attachment, and spreading—key processes essential for maintaining oral tissue integrity. Functional assays such as wound healing, MTT assays, real-time PCR, western blotting, and scanning electron microscopy will further characterize these cellular responses at the molecular and structural levels. In addition, we will analyze microbial dysbiosis in saliva and dental plaque using 16S rRNA sequencing to identify shifts in the microbial community associated with autoimmune conditions. By integrating these datasets, we aim to elucidate the intricate cross-talk between genetic variants, oral cellular dysfunction, and microbial imbalances. Our findings will shed light on how these interactions contribute to increased susceptibility to oral infections and chronic inflammation in autoimmune patients.





## ABSTRACTS OF THE PRESENTATIONS

(In order of presentation)

### **VII. Mentee from Pakistan Section: Muhammad Kashif, Bakhtawar Amin Medical & Dental College, Multan, Pakistan, Mentee from Pakistan Section**

#### ***Human Papillomavirus in Saliva and Its Role in Oral and Oropharyngeal Cancers***

Human papillomavirus (HPV) is a significant risk factor for certain head and neck squamous cell carcinomas, regardless of traditional causes like smoking or alcohol use. HPV is a small, double-stranded DNA virus that infects epithelial cells in areas such as the skin, mouth, throat, and anogenital region. There are over 200 known HPV types, classified as low-risk or high-risk based on their potential to cause cancer. High-risk types, especially HPV16, can trigger cancer development by producing E6 and E7 oncoproteins, which inactivate tumor suppressor genes (TP53 and Rb). HPV16 is strongly linked to oropharyngeal cancer, but its role in other head and neck cancer subsites remains unclear.

Several methods are used to detect and identify HPV, including polymerase chain reaction (PCR), real-time PCR, in situ hybridization, immunohistochemistry, and next-generation sequencing (NGS). However, collecting tumor tissue is invasive, and techniques like oral swabs or cytobrushes only sample accessible areas, making it hard to detect hidden or early-stage tumors.

Saliva testing offers a simple, non-invasive alternative for HPV detection in oral and oropharyngeal cancers. Collecting oral exfoliated cells from saliva or oral rinses is easy, quick, and suitable for screening high-risk populations. This presentation will provide an overview of HPV detection in saliva and its role in oropharyngeal cancer development.

