Melbourne Dental School

2021 Honours & Master of Biomedical Science Research Project Handbook

bio21 institute

THE UNIVERSITY OF MELBOURNE

Faculty of Medicine, Dentistry & Health Sciences
Melbourne Dental School

Welcome to the Melbourne Dental School

The Melbourne Dental School is proud of its longstanding record of excellence in health research. Research at the School covers a broad range of scientific endeavour from basic science to clinical studies involving various disciplines including **microbiology**, **immunology**, **cancer cell biology**, **biochemistry & molecular biology**, **chemistry**, **anatomy**, and **materials engineering**.

We are incredibly passionate about the mentoring and the training of future researchers. Indeed, our mission is to continue to be a world-class, research-based school, offering education of the highest quality. To achieve this goal, we provide excellence in research training and support for all laboratory and clinician research students as they develop research knowledge and expertise and help drive new discoveries that lead to better outcomes for patients. So, if you are passionate about improving patient health, we encourage you to join us in the pursuit of knowledge by applying to do Honours or a Masters degree at the Melbourne Dental School. Working closely with researchers, students undertake their project in state-of-the-art research laboratories at the Melbourne Dental School and Bio21 Institute. Students will be eligible to apply for the Noel Arthur Twiss Scholarship ($5,000).

There are a number of factors you might want to consider when making the decision about undertaking an Honours year or Masters degree, such as the amount of time spent on your research project, opportunities to undertake professional skills-based subjects, and which pathway would be most advantageous for possible entry into a PhD program in the future. Regardless of your choice, the School provides a stimulating and challenging intellectual environment that allows you to experience research firsthand and put your scientific knowledge into practice. The diverse range of Australian and international students from many social and ethnic backgrounds at the School greatly enhances the learning experience.

This booklet provides information that will help you decide on potential research projects. Please take your time to identify projects that are of interest and contact potential supervisors for more information. I am very confident they will be eager to discuss your research interests and talk about their own research, show you around their laboratories, and introduce you to other students and researchers.

I look forward to seeing you at the School next year and hearing about your exciting research project.

Professor Alastair J Sloan
Head of School

Honours & Master of Biomedical Science Projects

Host–Microbe Interactions in Health & Disease

How epithelial cells tailor different immune responses to commensal bacteria and pathogens

The epithelial cells that line the oral cavity express immune receptors which enable them to detect the presence of bacteria. However, how epithelial cells tolerate the presence of commensal bacteria and yet stimulate host inflammation to prevent infection by bacterial pathogens is poorly understood. The spatial context in which bacteria are detected by epithelial cells likely plays a critical role in enabling them to discriminate between commensal bacteria and pathogens. In this project you will investigate how the spatial regulation of inflammatory pathways enables the tailoring of the host immune response to bacteria. The new knowledge you create will help us to develop better ways to prevent bacteria-induced diseases.

Areas/techniques in which expertise will be developed

Mammalian and bacterial cell culture, manipulating gene expression, analysis of cell signalling and gene expression, immunofluorescence confocal microscopy, critical thinking, time management, scientific writing, and oral communication.

Supervisors

A/Prof Glen Scholz – glenms@unimelb.edu.au
Dr William Stanley – william.stanley@unimelb.edu.au

Degree availability

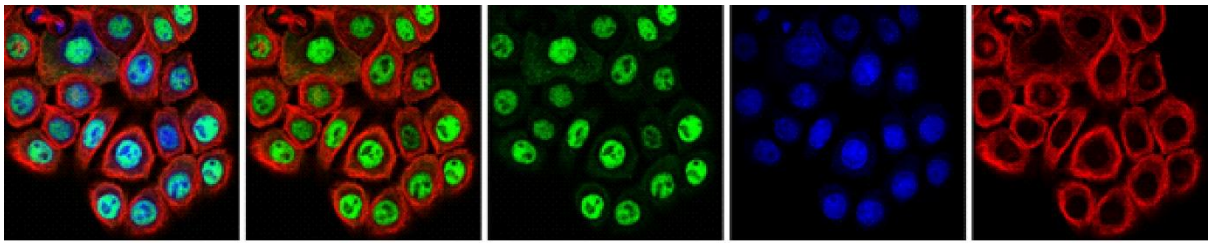
Bachelor of Science (Honours)
Bachelor of Biomedicine (Honours)
Master of Biomedical Science

Location

Melbourne Dental School & Bio21 Institute

Number of vacancies

Two students



Immunofluorescence confocal microscopic images showing the subcellular localisation of the transcription factor IRF6 (green staining) in human epithelial cells. Blue staining = Nuclei; Red staining = Structural protein.

Recent publications

- Kwa MQ*^, Nguyen T^, Huynh J*^, Ramnath D^, De Nardo D*^, Lam PY*, Reynolds EC, Hamilton JA, Sweet MJ, Scholz GM. Interferon regulatory factor 6 differentially regulates Toll-like receptor 2-dependent chemokine gene expression in epithelial cells. Journal of Biological Chemistry (2014) 289:19758-66.
- Huynh J*^, Scholz GM, Aw J*^, Kwa MQ*^, Achuthan A*^, Hamilton JA, Reynolds EC. IRF6 regulates the expression of IL-36γ by human oral epithelial cells in response to *Porphyromonas gingivalis*. Journal of Immunology (2016) 196:2230-38.
- Aw J*^, Scholz GM, Huq NL, Huynh J, O'Brien-Simpson NM, Reynold EC. Interplay between *Porphyromonas gingivalis* and EGF signaling in the regulation of CXCL14. Cellular Microbiology (2018) 20:e12837.
- Scholz GM, Heath JE, Aw J*^, Reynolds EC. Regulation of the peptidoglycan amidase PGLYRP2 in epithelial cells by IL-36γ. Infection and Immunity (2018) 86:e00384-18.
- Scholz GM, Heath JE, Walsh KA, Reynolds EC. MEK-ERK signaling diametrically controls the stimulation of IL-23p19 and EBI3 expression in epithelial cells by IL-36γ. Immunology & Cell Biology (2018) 96:646-55.
- Heath JE, Scholz GM, Veith PD, Reynolds EC. IL-36γ regulates mediators of tissue homeostasis in epithelial cells. Cytokine (2019) 19: 24-31.

**Former Honours student; ^former PhD student*

Host defence functions of the novel inflammatory cytokine IL-36G

The oral cavity is a major entry portal for important bacterial pathogens. We recently demonstrated that the epithelial cells which line the oral cavity produce the inflammatory cytokine IL-36G when they detect the presence of bacteria. Importantly, we have shown that IL-36G not only stimulates the production of proteins which possess direct antimicrobial activities, but also cytokines that activate and recruit immune cells (e.g. neutrophils and lymphocytes). In this project you will use gene knockout mice to investigate how IL-36G prevents infection by bacterial pathogens. The new knowledge you create will help us to develop better ways to treat infections caused by antibiotic-resistant bacteria.

Areas/techniques in which expertise will be developed

Bacterial cell culture, mouse models of bacterial infection with gene knockout mice, cytokine assays, gene expression analysis, immunofluorescence confocal microscopy, critical thinking, time management, scientific writing, and oral communication.

Supervisors

A/Prof Glen Scholz – glenms@unimelb.edu.au
Dr William Stanley – william.stanley@unimelb.edu.au

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- Heath JE, Scholz GM, Veith PD, Reynolds EC. IL-36γ regulates mediators of tissue homeostasis in epithelial cells. Cytokine (2019) 19: 24-31.

**Former Honours student; ^former PhD student*

How bacterial metabolites can disrupt mucosal immunity

The mucosal surfaces of the body are colonised by large numbers of bacteria. Some of the metabolites produced by the bacteria (e.g. short-chain fatty acids) can affect the functions of immune cells. We recently demonstrated that these metabolites can also affect the host defence functions of oral mucosal epithelial cells. Oral epithelial cells play critical roles in preventing infection because they are the first cells to encounter pathogens and subsequently activate the host immune response. In this project you will investigate how bacterial metabolites can affect specific host defence functions of oral mucosal epithelial cells. The new knowledge you create will help us to understand how changes in the bacterial composition of biofilms can disrupt host-bacteria homeostasis and thereby cause disease.

Areas/techniques in which expertise will be developed

Mammalian and bacterial cell culture, manipulating gene expression, analysis of cell signalling and gene expression, immunofluorescence confocal microscopy, critical thinking, time management, scientific writing, and oral communication.

Supervisors

A/Prof Glen Scholz – glenms@unimelb.edu.au
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- Aw J*^, Scholz GM, Huq NL, Huynh J, O'Brien-Simpson NM, Reynold EC. Interplay between *Porphyromonas gingivalis* and EGF signaling in the regulation of CXCL14. Cellular Microbiology (2018) 20:e12837.
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- Scholz GM, Heath JE, Walsh KA, Reynolds EC. MEK-ERK signaling diametrically controls the stimulation of IL-23p19 and EBI3 expression in epithelial cells by IL-36γ. Immunology & Cell Biology (2018) 96:646-55.

**Former Honours student; ^former PhD student*

Antiviral functions of the novel inflammatory cytokine IL-36G

The oral cavity is a major entry portal for important microbial pathogens, including viruses. We have demonstrated that the epithelial cells which line the oral cavity strongly express the novel inflammatory cytokine IL-36G when they detect the presence of microbes. Importantly, we have recently found that IL-36G stimulates epithelial cells to express genes that mediate antiviral responses. This suggests that IL-36G plays a role in preventing viral infection. In this project you will use cell culture systems and gene knockout mice to investigate how IL-36G prevents infection by viruses. The new knowledge you create will help us to develop better ways to treat diseases caused by viruses.

Areas/techniques in which expertise will be developed

Mammalian cell culture, manipulating gene expression, cytokine assays, analysis of cell signalling and gene expression, immunofluorescence confocal microscopy, critical thinking, time management, scientific writing, and oral communication.

Supervisors

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Dr William Stanley – william.stanley@unimelb.edu.au

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- Scholz GM, Heath JE, Aw J*^, Reynolds EC. Regulation of the peptidoglycan amidase PGLYRP2 in epithelial cells by IL-36γ. Infection and Immunity (2018) 86:e00384-18.
- Scholz GM, Heath JE, Walsh KA, Reynolds EC. MEK-ERK signaling diametrically controls the stimulation of IL-23p19 and EBI3 expression in epithelial cells by IL-36γ. Immunology & Cell Biology (2018) 96:646-55.
- Heath JE, Scholz GM, Veith PD, Reynolds EC. IL-36γ regulates mediators of tissue homeostasis in epithelial cells. Cytokine (2019) 19: 24-31.

**Former Honours student; ^former PhD student*

Microbiomes in Health & Disease

Oral microbiome in health and disease

The human oral cavity is home to over 700 species of bacteria, many of these species are beneficial to our health whilst others are associated with the development of chronic diseases. During disease initiation there is a shift in the composition of the microbiome that leads to the development of a dysbiotic biofilm community that sustains disease progression. In this project you will determine the oral microbiome in health compared with that in disease states and identify those bacteria associated with disease. Ultimately, we will identify microbial biomarkers that can be used as predictors of health and disease.

Areas/techniques in which expertise will be developed

Genomic DNA extraction from different sample types, PCR amplification, next generation sequencing using Ion Torrent technology, bioinformatics, biostatistics, microbial ecology, research management, oral presentation skills, scientific writing skills.

Supervisors

Prof Stuart Dashper – stuartgd@unimelb.edu.au
Dr Catherine Butler – cbutler@unimelb.edu.au
Dr Samantha Byrne – sbyrne@unimelb.edu.au
Mr Geoff Adams – g.adams@unimelb.edu.au

Degree availability

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Master of Biomedical Science
Master of Bioinformatics

Location

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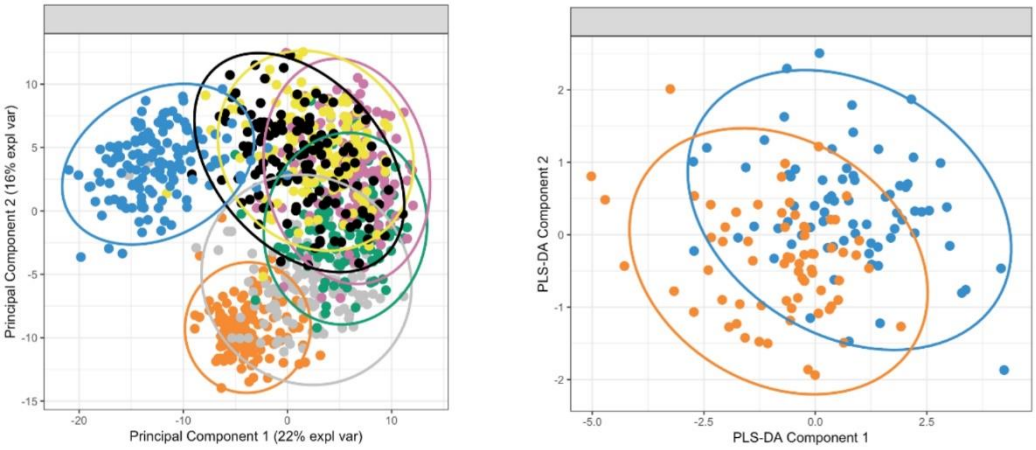
Three students



Temporal development of the human oral microbiome (left) and discrimination of children with early childhood caries from heathy children based on their oral microbiome (right).

Recent publications

- Dashper SG *et al*. Temporal development of the oral microbiome and prediction of early childhood caries. *Scientific Reports* **9,** 19732 doi:10.1038/s41598-019-56233-0, (2019).
- Fernando JR, Butler CA, Adams GG, Mitchell HL, Dashper SG et al. The Prebiotic Effect of CPP-ACP Sugar-Free Chewing Gum. *Journal of Dentistry*, 91:103225 doi.org/10.1016/j.jdent.2019.103225 (2019).
- Gussy M, Mnatzaganian G, Dashper S*, et al*. Identifying predictors of early childhood caries among Australian children using sequential modelling: Findings from the VicGen birth cohort study. Journal of Dentistry 103276, doi.org/10.1016/j.jdent.2020.103276 (2020).
- Byrne S, Dashper SG, Darby I, Adams G, *et al*. Progression of chronic periodontitis can be predicted by the proportions of *Porphyromonas gingivalis* and *Treponema denticola* in subgingival plaque samples. *Oral Microbiology and Immunology*. 24:469-477 (2009). (205 citations).

Alzheimer's disease and oral bacteria

A growing number of studies are now linking bacterial infection and chronic periodontitis with sporadic Alzheimer's disease. Two recent studies have provided strong evidence for a potential causal link between the pathogenic oral bacterium *Porphyromonas gingivalis* and Alzheimer's disease. There are also reports of oral bacterial proteolytic enzymes and genomic DNA, particularly those of *P. gingivalis* and *Treponema denticola*, in the brain tissue of AD sufferers. We have demonstrated that *P. gingivalis* and *T. denticola* are intimately associated in the human oral cavity and work cooperatively to cause damage to the host, resulting in a number of oral and systemic diseases. *P. gingivalis* and *T. denticola* likely use complementary characteristics to cooperate to escape the oral cavity. In this set of related studies you will be able to characterise a range of synergistic interactions from how these two oral pathobionts cooperate to escape the oral cavity, focussing on the roles of *T. denticola* chemotaxis and motility, to how they work to invade the brain in animal models of disease.

Areas/techniques in which expertise will be developed

Bioinformatics, microbial ecology, biofilm formation, chemotaxis and motility assays, molecular biology, small animal models of disease, continuous coculture of bacteria, confocal microscopy, protein expression, research management, oral presentation skills, scientific writing skills.

Supervisors

Prof Stuart Dashper – stuartgd@unimelb.edu.au
Dr Catherine Butler – cbutler@unimelb.edu.au
Dr Nada Slakeski –nslak@unimelb.edu.au

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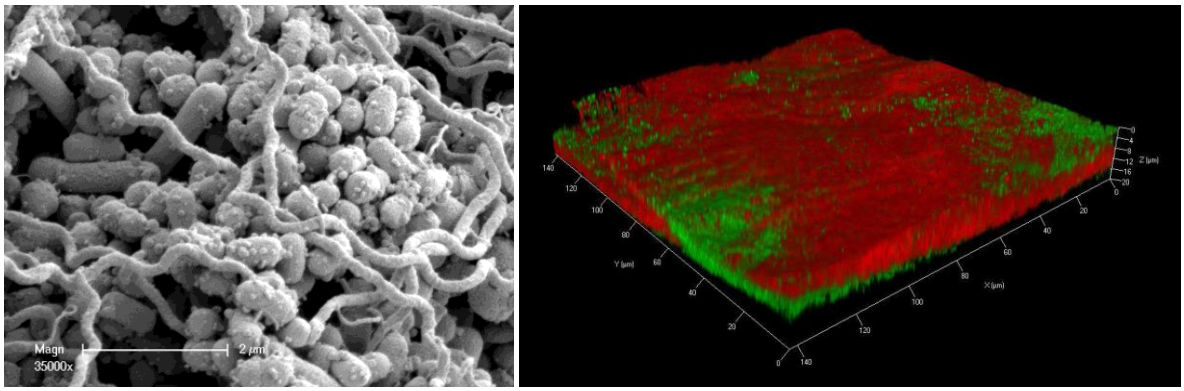
Three students



Electron micrograph and confocal micrograph of synergistic biofilm formation by *P. gingivalis* and *T. denticola*.

Recent publications

- Ng H*^, Slakeski N, Butler CA, Veith PD, Chen YY, Liu SW, Hoffmann B, Dashper SG† and Reynolds EC†. The role of *Treponema denticola* motility in synergistic biofilm formation with *Porphyromonas gingivalis*. *Frontiers in Cellular and Infection Microbiology,* 9:432, (2019).
- Tan K[1]^, Seers CA[1], Dashper SG[1], Mitchell H*, et al*. *Treponema denticola* and *Porphyromonas gingivalis* exhibit metabolic symbioses. *PLOS Pathogens*, DOI: 10.1371/journal.ppat.1003955 (2014). (82 citations).
- Dashper SG, et al. *Porphyromonas gingivalis* uses specific domain rearrangements and allelic exchange to generate diversity in surface proteins associated with virulence. *Frontiers in Microbiology*, 8:48 doi: 10.3389/fmicb.2017.00048 (2017).
- Xin LK^, Butler C, Slakeski N, Hoffmann B, Dashper SG and Reynolds EC. Synergistic Interactions between *Treponema denticola* and *Porphyromonas gingivalis*. *Journal of Oral Microbiology,* 2020.

**Former Honours student; ^former PhD student*

Bacterial Virulence

Characterisation of potential virulence factors secreted by the type 9 secretion system of *Porphyromonas gingivalis*

Chronic periodontitis is an inflammatory disease causing the destruction of the supporting gum and bone of teeth. *Porphyromonas gingivalis* (*Pg*) is the major agent of this disease and is also associated with other diseases/conditions such as cardiovascular diseases, adverse pregnancy outcomes (preterm birth, low birth weight and pre-eclampsia), rheumatoid arthritis, diabetes, non-alcoholic fatty liver disease and Alzheimer's disease. This emphasizes the importance of *Pg* host dissemination, immune evasion, immune modulatory tactics and *Pg*-induced inflammation. *Pg* possesses a type 9 secretion system (T9SS) which secretes abundant gingipain proteases that are involved in this disease process. Many other proteins are secreted by this T9SS but their functions are unknown. Recent structural analyses have predicted virulence functions for several of these T9SS substrates. The honours projects on offer will characterize these candidate virulence factors to further our understanding of the arsenal of virulence factors that *Pg* deploys and to identify potential vaccine candidates.

Areas/techniques in which expertise will be developed

Microbiological, molecular biology, immunological and biochemical techniques, including anaerobic bacterial growth, cell growth inhibition assays, *Pg* gene deletion, PCR, DNA purification, DNA gel electrophoresis, recombinant protein expression, SDS-PAGE, Western blot, 2D Blue-Native PAGE and proteomics, super-resolution fluorescence microscopy and electron microscopy.

Supervisors

Dr Michelle Glew – mglew@unimelb.edu.au
A/Prof Paul Veith – pdv@unimelb.edu.au

Degree availability

Bachelor of Science (Honours)
Master of Biomedical Science

Location

Bio21 Institute

Number of vacancies

Two students

Recent publications

- Glew MD, Veith PD, Chen D, Gorasia DG, Peng B, Reynolds EC. PorV is an outer membrane shuttle protein for the type IX secretion system. Scientific Reports (2017) 7(1):8790.
- Veith PD, Glew MD, Gorasia DG, Reynolds EC. Type IX secretion: the generation of bacterial cell surface coatings involved in virulence, gliding motility and the degradation of complex biopolymers. Molecular Microbiology (2017) 106(1):35-53.

Identification of the interacting regions between an essential component of the type 9 secretion system, PorV, and secreted virulence factors of the oral pathogen, *Porphyromonas gingivalis*

The type 9 secretion system (T9SS) of the oral Gram-negative pathogen, *Porphyromonas gingivalis,* is responsible for secreting many CTD-proteins including abundant gingipain proteases that are major virulence factors and contribute to chronic periodontitis in humans. PorV is an outer membrane beta-barrel protein and we have shown it to be an essential component involved in binding to CTD-proteins and interacting with the attachment complex (PorU, PorQ and PorZ) that is responsible for the covalent linkage of CTD-proteins to anionic lipopolysaccharide (A-LPS) which ultimately anchors them to the cell surface. Recently, the structure of an outer membrane component of the T9SS called Sov was determined by cryo-electron microscopy and found to exist as two separate complexes: Sov-Plug and Sov-PorV. We propose that Sov aids PorV in recruiting and translocating the CTD-proteins across the outer membrane in preparation for linkage to A-LPS. To better understand how PorV interacts with T9SS substrates and Sov, this project will involve mutagenesis of the inner and outer loop amino acids of PorV followed by characterization of the resulting *P. gingivalis* mutants to identify specific secretion defects. Any interaction defects of the mutated PorV protein with either the CTD-proteins and/or Sov will be identified. The student will join a team that are leaders in the field and publishing in high ranking journals.

Areas/techniques in which expertise will be developed

Microbiological, molecular biology, immunological and biochemical techniques, including anaerobic bacterial growth, gene mutagenesis in *P. gingivalis*, PCR, DNA purification, DNA gel electrophoresis, SDS-PAGE, Western blot, 2D Blue-Native PAGE, proteomics, protein complex purification and electron microscopy.

Supervisors

Dr Michelle Glew – mglew@unimelb.edu.au
A/Prof Paul Veith – pdv@unimelb.edu.au

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Master of Biomedical Science

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One student

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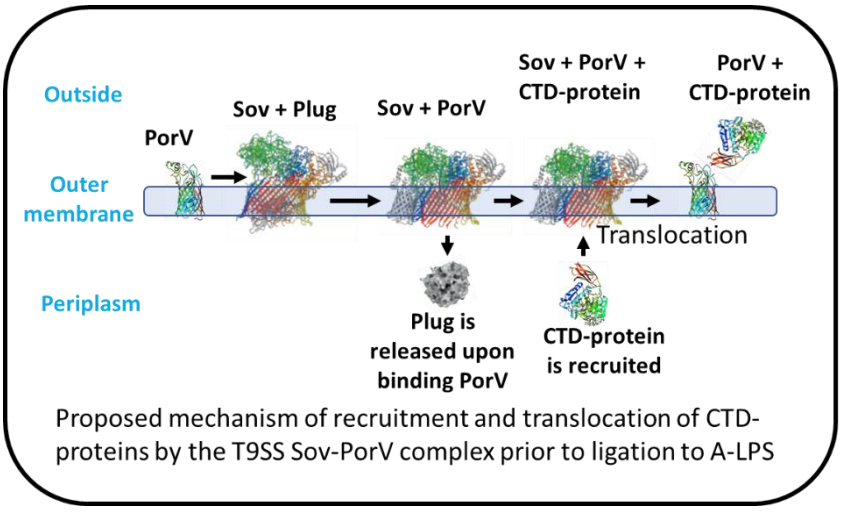

Proposed mechanism of recruitment and translocation of CTD-proteins by the T9SS Sov-PorV complex prior to ligation to A-LPS

Project Theme: Oral Cancer

The effect of anticoagulants on OSCC treatment

Oral squamous cell carcinoma (OSCC) is one of the most common forms of epithelial cancer in the head and neck area with over 300,000 new cases per year worldwide and its prognosis still remains poor. Current treatment strategies of OSCC include surgery, radiation therapy, and adjuvant therapy such as chemotherapy with agents such as cisplatin, carboplatin, 5-fluorouracil (5-FU), paclitaxel, and docetaxel. Among these, 5-FU and cisplatin are most commonly used for OSCC.

Accumulating evidence has shown the role of several components of the coagulatory system in different phases of carcinogenesis including precancerous and initial stages, tumour growth, angiogenesis, stroma generation, and metastasis of malignant cells. In fact, during carcinogenesis, angiogenesis is favoured by local conditions of hypoxia, cell-to-cell interactions, and by expression of paracrine growth factors and inflammatory cytokines. Anticoagulants are widely prescribed medications, routinely administered to help prevent blood clots. They are administered to people at a high risk of blood clots, to reduce their chances of developing serious conditions such as stroke and heart attack. This may include people with atrial fibrillation or an irregular heartbeat. Atrial fibrillation is a significant risk factor for stroke and around 300,000 Australians have atrial fibrillation. Anticoagulants may also be prescribed to people who have had major surgery, such as aortic valve replacement, or those with certain blood disorders. Because of the links between coagulation, cancer biology and prognosis, interest has grown in the potential benefit of anticoagulants such as warfarin and heparin for the prevention or treatment of several types of cancer. However, to date there is a complete lack of studies investigating the effect of anticoagulants on OSCC.

The aims of this project are to assess the effect of the wide spectrum of anticoagulant medications on:
OSCC cell viability, OSCC cancer cell invasive phenotype, and on the efficacy of chemotherapeutic agents in the treatment of OSCC.

Areas/techniques in which expertise will be developed

Mammalian cell culture, including proliferation, migration and invasion assays; immunohistochemistry and immunofluorescence; ELISA; Western blot.

Supervisors

Dr Antonio Celentano
antonio.celentano@unimelb.edu.au

Degree availability

Bachelor of Science (Honours)
Bachelor of Biomedicine (Honours)
Master of Biomedical Science

Location

Melbourne Dental School

Number of vacancies

One student

Becoming an Honours or Master of Biomedical Science Student at the Melbourne Dental School

Entry to the Honours and Master of Biomedical Science programs is based on: (1) project availability, (2) academic background, and (3) suitability.

HOW TO APPLY FOR HONOURS

1. Identify projects in this handbook that are of interest to you.
2. Contact the relevant project supervisor to discuss your interest in their research. It is a good idea to email them a copy of your CV and your academic transcripts to help them understand your background, interests and academic strengths.
3. Make a time to meet with potential supervisors to discuss your project interests and discuss your academic record.
4. Visit the laboratory and meet other students and researchers.
5. In some cases, supervisors may be willing to offer you a provisional place in their laboratory (a provisional offer indicates that you have a guaranteed place in the Honours course, providing you satisfy all other entry requirements).
6. Apply online through the Faculty of Medicine, Dentistry and Health Sciences website: http://mdhs-study.unimelb.edu.au/degrees/honours/apply-now#apply-now.

HOW TO APPLY FOR MASTER OF BIOMEDICAL SCIENCE

1. Identify projects in this handbook that are of interest to you.
2. Contact the relevant project supervisor to discuss your interest in their research. It is a good idea to email them a copy of your CV and your academic transcripts to help them understand your background, interests and academic strengths.
3. Make a time to meet with potential supervisors to discuss your project interests and discuss your academic record.
4. Visit the laboratory and meet other students and researchers.
5. In some cases, supervisors may be willing to offer you a provisional place in their laboratory (a provisional offer indicates that you have a guaranteed place in the Honours course, providing you satisfy all other entry requirements).
6. Apply online through the Faculty of Medicine, Dentistry and Health Sciences website: http://mdhs-study.unimelb.edu.au/degrees/master-of-biomedical-science/overview.

SCHOOL CONTACTS

Honours Coordinator

A/Prof Glen M. Scholz
Email: mds-honours@unimelb.edu.au
Phone: 9341 1545

Academic Programs Officer

Ms Janelle Christie
Email: dental-office@unimelb.edu.au