

Honours in Medical Research Handbook

2026

School of Biomedical Sciences

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Welcome to the School of Biomedical Sciences' Honours Program



This 2025 Handbook introduces the Honours in Medical Research program in the School of Biomedical Sciences at the University of Western Australia.

An Honours in Medical Research is an invaluable postgraduate qualification that expands your employment opportunities and competitiveness and provides the training and launching pad for a wide variety of professional and postgraduate careers, including those in science, health-related disciplines, and/or postgraduate research.

For many of you, undertaking an Honours project will be your first real taste of scientific research, and you will be faced with exciting - and perhaps daunting - new challenges. You will have to confront and master the rigours of scientific writing, experimental design, time management, data analysis and oral presentations. You will learn to undertake and master cutting-edge scientific techniques and methodologies, and become familiar with a range of experimental tools, models and equipment. You will need to be both diligent and resilient, for in science (as in life) things often do not go as planned and there are hurdles and disappointments to be overcome. Your experienced supervisors will be there to guide you and help you to achieve your goals and do the very best you can.

For some of you this will be a transformative year in your life and will set you on a career path of lifelong research and discovery. For others it will be a stepping-stone to other ventures. For all of you it will be an invaluable learning experience that will teach you a range of technical, analytical, intellectual and communication skills that will prove invaluable wherever life takes you.

I encourage you all to embrace the challenges ahead, keep your minds open to new experiences and knowledge, make friends and become part of the school community, and make the most of being in a stimulating environment at the cutting edge of biomedical research.

Good luck!



Professor Jeffrey Keelan, BSc (Hons) Liv., MSc PhD Auck., FSRB Head of School of

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Introduction

The purpose of the Honours program in UWA's School of Biomedical Sciences is to introduce students to contemporary scientific medical research practices and develop their practical research and communication skills and competencies.

The Honours course comprises an academic year of full-time research and training, centered on an individual research project, resulting in the preparation and submission of a compact medical research thesis under the supervision of an experienced researcher and/or co-supervisors. Students will develop enhanced skills in critical and lateral thinking, experimental design, problem solving, time-management and scientific literacy and communication, as well as mastery of a variety of laboratory and/or analytical skills and an understanding of laboratory safety, professional responsibility and ethical conduct in research.

The Honours program is structured as a 48-point course (24 points per semester) composed of six units that encompass different components of the program. Some of the units are split into two parts that span both semesters (colour coded below). The course structure is summarized in the diagram below:

SEM 1	SEM2
BMED4001. 6 pts – Literature Review and Research Proposal in Biomedical Sciences	BMED4005. 6 pts – Research Communication in Biomedical Sciences - part 2
BMED4002. 6 pts – Research Communication in Biomedical Sciences - part 1 (<i>assessment continuing</i>)	BMED4006. 18 pts – Medical Research Thesis part 2: completion, assembly, submission and examination
BMED4003. 6 pts – Medical Research Thesis part 1: Preparation, induction and training (<i>assessment continuing</i>)	
BMED4004. 6 pts – Research Ethics, Rationale & Design (<i>ungraded pass/fail</i>)	

Final marks breakdown across the units:

- | | |
|---|------------------|
| • Literature review and research proposal: | 15% |
| • Research communication parts 1 ... plus... plus part 2 | 25% |
| • Research Ethics, Rationale & Design: | Pass/fail |
| • Thesis part 1 plus... Thesis part 2: | 60% |

At the beginning of the year, students receive general training in biostatistics and chemistry, laboratory safety induction, and the appropriate use of research infrastructure. Additional specific training may also be required for certain types of research. For example, research using non-human animals will require completion of the PAWES training course offered by Animal Services; projects that involve human participants or their tissues/samples will need human ethical approval, while research using genetically modified organisms will require OGTR approval. General laboratory training, student-specific training, and instruction in research design, results presentation and analysis will contribute to BMED4003. Training in research ethics, rationale and design will be covered in BMED4004.

Scientific communication skills are taught and developed throughout the Honours year. Research contributing towards the research thesis commences at the start of the year and continues throughout the

program, culminating in the submission of the thesis and presentation of the project at a conference-style event. Honours graduates who achieve a 2A or higher degree are eligible to enroll in a PhD programme.

Learning outcomes

Students who complete Honours in Medical Research should be able to:

- 1) Critically evaluate literature relevant to the area of research and compile references in an appropriate style;
- 2) Demonstrate advanced oral and written scientific communication skills;
- 3) Develop a research plan to address the aims of the project;
- 4) Execute a range of statistical analyses relevant to biomedical research;
- 5) Discuss considerations relevant to laboratory safety;
- 6) Demonstrate an advanced understanding of the responsible conduct of research in the biomedical sciences;
- 7) Demonstrate a thorough understanding of good clinical practice as it pertains to medical research;
- 8) Evaluate and design a research project based on a biomedical question;
- 9) Perform experiments, interpret data, solve scientific problems, identify limitations and future directions
- 10) Apply chemistry fundamentals in a laboratory setting

Entry Requirements for Honours in Medical Research

Students require a minimum weighted average mark (WAM) of 65 per cent in the Level 3 units of a Biomedical Science-related discipline, such as Genetics, Neuroscience, Pathology, Pharmacology, Microbiology, Immunology, Anatomy, Human Biology, Physiology, Biochemistry, Molecular Biology, Psychology, Public health. The student's undergraduate program should be pertinent to the topic of the project. Students must be accepted by at least one Academic supervisor from the School of Biomedical Sciences, UWA.

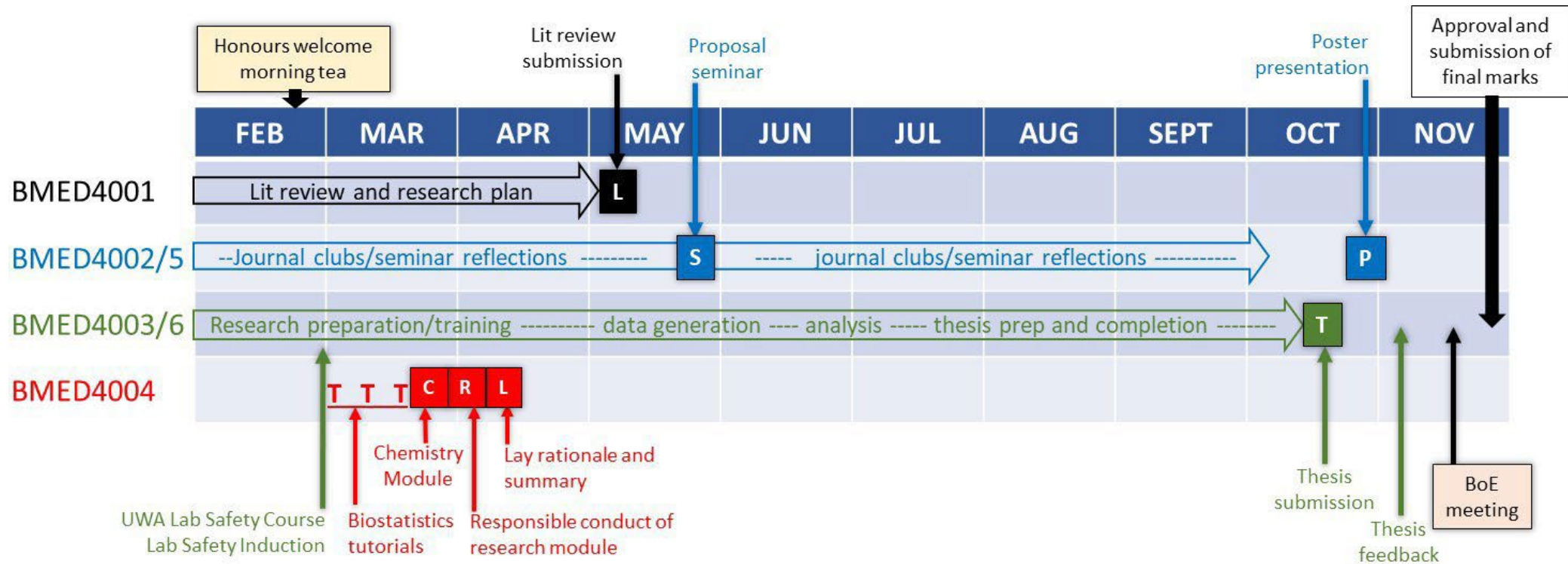
Details of the enrolment process can be found [here](#)

Outline and Structure of the Honours in Medical Research Program

The Honours program consists of a combination of research-based training modules and activities, attendance/presentation at research seminars and journal clubs, development and completion of an original research project with analysis and interpretation of the data generated, and submission of a written condensed literature review, research proposal and research thesis. These activities are organised and assessed via the six BMED units as outlined in the Table above: BMED4001, 4002, 4003, 4004, 4005, 4006. **All units must be passed to complete the course.** Failing any one unit will fail the course.

The course is a 1 year program of fulltime study, with research training commencing in February and the final assessment completed in November. There is no mid-year entry into the course. A typical timeline for the activities and assessments of the course is depicted in the diagram below:

Honours 2026 Schedule and Timeline



Unit content, learning outcomes and assessment structure

BMED4001 (6 pts)

Literature Review and Research Proposal in Biomedical Sciences

UNIT Coordinators: *Professor Jeff Keelan and Dr Mitali Sarkar-Tyson*

Students will write a literature review summarising the knowledge base and rationale underpinning the project, and present a research plan outlining the principle aims of the study and an overview of the design and methodology used to achieve the study objectives.

Learning outcomes: students will be able to:

- a) Compile, read and critically evaluate literature relevant to the area of research;
- b) Format bibliography in the [Vancouver style](#) of citation and bibliographic referencing;
- c) Identify gaps in current knowledge;
- d) Formulate aims to address the gaps in research;
- e) Develop a research plan to address the aims of the project
- f) Demonstrate high level written communication skills.

The literature review and research plan document is assessed by the two independent examiners nominated by the project supervisor(s); they will also assess the thesis. The literature review and research plan will comprise the first chapter of the thesis. It can be edited and improved following feedback after assessment prior to inclusion in the final thesis. The document can be up to 5000 words (not including references); a word count must be included at the end of the document. The mark for the Literature Review and Research Plan represents 100% of the mark for this unit.

Further details can be found in the **Assessment Details and Timelines** section below.

BMED4002 and BMED4005 (6 pts each)

Research Communication in Biomedical Sciences Parts 1 and 2

UNIT Coordinators: *Professor Jeff Keelan and Dr Mitali Sarkar-Tyson*

BMED4002 Part 1 and BMED4005 Part 2 are paired communications units taken over semester 1 and 2. These units have similar outcomes and assessments, with BMED4002 Part 1 being “assessment continuing”.

These two units are comprised of four components:

- 1) Research proposal seminar (50% of mark, received in the 1st semester)
- 2) Specific engagement with/presentation at weekly lab journal clubs (both semesters; ungraded pass/fail)
- 3) Attendance at School seminars, with submission of six structured personal reflections (both semesters; ungraded pass/fail)
- 4) Conference poster presentation and oral defence (50% of mark; 2nd semester)

These units deliver and assess key advanced biomedical research communications skills, including:

- a) Oral presentation to staff and students in the School of Biomedical Sciences of the project proposal (an in-depth overview of the literature pertaining to the field of study; an outline of the principal aims of the study and an overview of the approaches used to achieve the aims)
- b) Review and analysis of scientific papers in a discipline-related journal club;

- c) Attendance at the School Seminar program (or equivalent) and completion of Seminar Reflection Worksheets (6 minimum)
- d) Final conference poster presentation and oral Q&A/defence

BMED4003 (6 pts)

Medical Research Thesis part 1

UNIT Coordinators: *Professor Jeff Keelan and Dr Mitali Sarkar-Tyson*

This unit represents the preparative portion of the student's research project and training, which culminates in the submission of a medical research thesis at the end of semester 2 (BMED4006). It covers all aspects of laboratory safety, specialist training requirements, development of wet-/dry-lab bench skills, equipment operation/credentialing, ethical compliance issues, laboratory management awareness, and preliminary work on project establishment, rationale and design.

This unit is designated as 'assessment continuing', with thesis submission and examination occurring in BMED4006 at the end of semester 2. There are no specific marks allocated to this unit.

In addition to the preparative work undertaken on the research thesis, the unit encompasses a number of modules and courses that students may need to take to comply with safety and training regulations, in addition to project-specific training and upskilling. These include:

- 1) Basic biomedical statistics: Between-subjects and within-subjects designs, time-series and formal qualitative design; concepts of Bayesian and frequentist approaches to statistical analyses; standard group comparison methods (e.g., t-tests, ANOVAs, ANCOVAs, regression including correlations, methods of curve fitting, and nonparametric statistics); the concepts of power analysis and effect size;
- 2) General laboratory safety training (online);
- 3) Reading and sign-off on the School Laboratory Safety manual;
- 4) Project-specific training on individual equipment and infrastructure;
- 5) PAWES course for those working with animals;
- 6) Radiation Safety Course for those working with isotopes;
- 7) Gene Technology Awareness Session for those working with genetically modified organisms;
- 8) First-aid and aggressive incident management for those working with clinical samples;

Not all students will need to take all courses/modules. Completion of modules is pass/fail, but they are not graded and do not contribute to the marks for the unit.

BMED4004 (6 pts)

Research Ethics, Rationale and Design

Unit Coordinator: *Dr Lynette Fernandes*

Knowledge and understanding of medical research ethics are integral to research. In this unit, students participate in a face-to-face discussion of an interactive movie at the start of semester. Students are required to subscribe to the BMED4004 Discussion Forum on LMS and check their UWA emails every day for updates.

Students will also complete two online courses:

1. Global Health Training Centre Research Ethics Online Training Modular course

2. Global Health Training Centre Good Clinical Practice short course
3. Chemistry refresher module (via LMS)

Students will also be required to submit a clinical OR scientific rationale for their thesis project written in lay language in the format of a grant or ethics application summary.

All components of this unit are required to be completed satisfactorily to pass the unit. The unit is pass/fail and is not graded.

More details will be provided by the beginning of the first semester of 2026.

BMED4006 (18 pts)

Biomedical Research Thesis Part 2

UNIT Coordinators: *Professor Jeff Keelan and Dr Mitali Sarkar-Tyson*

This 2nd semester unit (18 pts) follows on from BMED4003 (6 pts; Thesis part 1); collectively these two units comprise the medical research thesis component of the course and are worth 24 credit points in total.

In brief, the thesis is a body of original research consisting of a title page, statement of contributions and acknowledgment, abstract, introduction and literature review, aims and objectives, methods, results, discussion, references/bibliography and appendices. The first section of the thesis (literature review, aims and objectives) is submitted in the first semester and examined independently (BMED4001). This allows first semester feedback to the student on progress and the opportunity to edit the literature review and make changes to the project if suggested by the examiners. The final completed thesis is submitted (BMED4006) for examination at the end of the program.

For this Honours course, the structure and length of the Medical Research Thesis is carefully prescribed and all students must follow the guidelines carefully (*see page 15*). Students must adhere to strict word limits for the various sections; this is to ensure the thesis is concise and readable. Adhering to word limits and writing succinctly is an important skill in the world of scientific writing; word limits are frequently imposed on research publications, so learning how to write within space constraints is a valuable “real world” skill. There are also strict instructions regarding referencing and attribution that must be followed, similar to those that apply to submission of research manuscripts for publication. Full details are provided below in the Assessment Instructions and Deadlines section.

The marks for the Medical Research Thesis (encompassed in BMED4003 and 4006) represent 60% of the total marks for the course. Completion of the thesis teaches students how to:

- a) Present, cite and critique biomedical literature supporting their research;
- b) Describe appropriate methodologies and statistical techniques used in their studies;
- c) Generate, analyse and present research findings clearly, accurately and professionally;
- d) Critically appraise and discuss their research in the context of the existing knowledge in the field and identify any strengths and weaknesses in their findings.

Detailed assessment instructions and deadlines

BMED4001 Literature Review and Research Proposal

Students will write a literature review introducing the background and rationale behind the project, and research plan outlining the principal aims and objectives of the study and an overview of the approaches and methodologies used to achieve the objectives. The submitted document is examined by two independent external examiners, who will provide comments and feedback via the examination process. The work undertaken to prepare this document will form the basis of your proposal seminar presented at the end of semester 1.

Layout: The structure, scope and breadth of the literature review should be decided in consultation with the supervisor, who will also edit drafts and give guidance on content, quality and style. Students will need to demonstrate an understanding of the literature supporting the project and its significance, identify areas where there are gaps or inconsistencies in knowledge, and demonstrate they are able to synthesise and interpret the findings of others in their own words. Mastery of written scientific English is a key aspect of this unit. Grammar, punctuation, layout and readability will all be taken into account in the marking process.

Students should work with their supervisor to establish the topic and order of the headings and sub-headings; structure and flow is very important in a review. A logical hierarchical numbering system should be employed, which should be used consistently and adhered to.

The title of the review should be stated on the first page in bold text (NB: this does not contribute to the page count or word count). It does not need to be the same as the title for the research thesis, but should accurately and succinctly describe the topic of the review.

The body of the text must be in Calibri 12 point font, single spacing throughout. A4 page size should be used. Figure/table legends should be single space, 11 point font. Page margins should be 20 mm for left and right, and 25 mm for top and bottom.

Following the literature review, the hypothesis, aims and objectives of the project should be stated. The research proposal should contain an outline of the experimental design, the primary and secondary outcomes of the study (where appropriate), a justification of the numbers of samples/animals/repeats, the main methodological / analytical approaches to be used, and a description of the planned statistical analyses.

Finally, a bibliography should be included, listing all the publications and sources of the literature cited in the review and research plan. The [Vancouver style](#) of citation and bibliographic referencing must be used. Students must comply with the University's [referencing requirements](#), and are strongly encouraged to use Endnote or a similar referencing software package to insert citations and manage/format their bibliography. The UWA library has several helpful online referencing resources to help students navigate the demands of referencing:

<https://guides.library.uwa.edu.au/referencinguwa>**Diagrams and Tables:** The use of figures and diagrams is strongly recommended to improve the clarity and readability of the review. Each figure, diagram and table should be accompanied with an explanatory legend (not included in the word limit); avoid splitting the figure/diagram and legend across different pages. Figures and diagrams should be embedded within the pages of text; they do not need to be on a separate page.

The inclusion of original figures is strongly encouraged; however, images or figures may be reproduced from published sources. They must, however, be appropriately referenced and cited in the bibliography to comply with the University's [plagiarism policy](#). Poor quality, pixelated figures with small illegible text should be avoided.

Word length and formatting: The literature review and research proposal should be no more than 5000 words (not including title and bibliography); a word count must be included at the end of the document. If the word count is exceeded, the document will be returned to the student for editing. Page numbers should be positioned in the footer at the bottom right of each page, commencing with the first page of the literature review and ending with the last page of the bibliography.

Submission and assessment: On or before the due date (which will be posted on LMS), one copy of the literature review and research proposal must be submitted in electronic format as a PDF document via LMS. The document will be assessed by two examiners against the following criteria:

- (a) How well does the literature review demonstrate an understanding of the central concepts in the field of study?
- (b) To what extent does the review summarise the current state of knowledge and identify gaps in that knowledge?
- (c) Does the review contain an appropriate number of figures, diagrams and tables and are they of high quality?
- (d) Does the review have a well-designed, logical structure and appropriate use of subheadings?
- (e) Does the review comply with the wording and formatting guidelines?
- (f) Does the document demonstrate an appropriate use of grammar and punctuation? Has care been demonstrated to avoid spelling mistakes and typographical errors?
- (g) Have the source materials been properly cited and formatted in a bibliography according to the style guide?
- (h) Are the *hypotheses* and *aims* clearly stated?
- (i) Is the proposed methodology and statistical analysis clear, appropriate and accurate?
- (j) Does the research proposal clearly establish how the hypotheses will be tested and the aims accomplished?

Marks and feedback: Marks will be deducted for late submission – *5% for every day late*, unless an extension has been granted. Remember, supervisors are busy and need time to read and edit their students' work. **Students should not demand a response within 24 hours to meet a deadline!**

Students will receive their marks at the end of semester 1. The marks for the literature review and research plan constitute 100% of the marks for this unit, which represents 15% of the entire grade for the course.

Following completion of the assessment process, students will receive written comments from the examiners, including a list of any typographical or stylistic changes recommended before final inclusion as part of your final research thesis submitted at the end of the year. Any changes made as a result of feedback will not be re-assessed and will not contribute to your final mark. However, the feedback gives students a final chance to do minor editing before a permanent thesis is submitted. The examiners may also comment on the content of the research plan and suggest changes if they have significant concerns regarding the proposed approach, power, methodology or feasibility.

BMED4002 and BMED4005 Research Communication Parts 1 and 2

The two research communication units contain four assessable items: *a research proposal seminar; participation in a journal club; attendance at the School Seminar program; and a conference-style poster presentation with oral defense*. The marks for these two units comprises 25% of the total marks for the Honours course.

1. Proposal seminar

Students are required to give an in-depth PowerPoint presentation of the literature pertaining to their field of study, an outline of the principal aims of the study and an overview of the approaches used to achieve the aims; this will be scheduled in mid-April. Preliminary data may be included if available. Students can and should receive guidance from their supervisors with respect to preparing the PPT presentation for this seminar and rehearsing the delivery of the seminar.

The seminar will consist of a 20-minute presentation followed by a 5-10 minute question and answer session. The seminar will count towards 50% of the Unit mark and will be assessed via a rubric given to all attending academics (with the exception of the supervisor(s)) in the audience against the following criteria:

(a) Clarity and quality of the overall presentation: oral and visual	33%
(b) Quality of the scientific content of the seminar	33%
(c) Ability to answer questions in a clear and logical manner	33%

Seminar marks will be released to students at the end of semester 1 of their seminar, with feedback where available.

2. Journal club

Students must regularly attend and participate in a lab journal club (or equivalent), presenting at least once to the group for discussion during the year. The supervisor will provide the Unit Coordinator a confirmation of attendance and participation. This activity is not directly assessed and is a pass/fail component.

3. School seminars

Students are required to attend **at least six** School Seminars and fill out a one-page Seminar Reflection Sheet for each, which must be forwarded to the Unit Coordinator within 24 h of the seminar. The Seminar Reflection Sheet template will be located on LMS. This is a pass/fail component and is not graded. Attendance at an alternative, equivalent seminar program is allowable, but must be approved by the Unit Coordinator.

4. Poster presentation and oral defense

At the end of the year, after submission of the thesis, students will prepare and present a 'conference-style' poster. This provides a concise overview of the entire research project completed by the student during the year. Examples of high-scoring posters from previous years will be made available via LMS. Formatting guidelines are listed below:

- Dimensions: up to 1 m wide and 1.41 m high (B0 page size)
- Material: uncoated paper (160 gsm) or fabric; gloss or lamination not necessary
- Title: located at the top of the poster in lettering of 4 cm or greater
- Content: Introduction; Methods: Results: Conclusions; References; Acknowledgements

- **Style:** the visual style, colours, font and layout are determined by the student.
- **Pointers:** Keep the poster simple, with logical flow/layout, and as concise as possible; rely on your presentation to expand, add clarity and explanation. Ensure text is large enough to be legible from 2 metres away. Avoid jargon/acronyms. Ensure figures are clearly labelled. Employ consistent formatting/colour options. Employ use of boxes and emphasis techniques to make key aspects stand out.

Posters can be printed by Uniprint or commercial suppliers such as Officeworks or Clockwork Print; note, printing may take up to 3 days.

Posters will be presented at a conference-style format, at a time and venue chosen by the discipline coordinator approximately 2 weeks after the thesis submission deadline. All students in the discipline will have their posters on display in the same session and are encouraged to view others' posters and actively engage with their colleagues to learn more about their project. Students are advised to bring a copy of their thesis along in case it needs to be referred to.

Students will individually present their poster to their examiners, supervisor and discipline coordinator, where they will be expected to explain the basic rationale and description of the study, present the findings, and discuss the significance and implications of the work. The examiners will then question the student on various aspects of the project over 10 minutes. Students may be asked to defend the use of specific methodology or approaches, their interpretation of the findings, or an aspect underpinning the rationale for the study.

During the presentation and Q&A session, it is important to speak loudly, fluently and clearly, avoid long rambling statements, and demonstrate a solid depth of knowledge and understanding of the topic. Students are advised to rehearse their presentations in front of a supportive audience and get feedback. The poster and presentation will be marked against the following criteria:

- a) Visual clarity, impact and effectiveness of the poster (50% of poster grade)
- b) Quality of the oral interpretation of the poster (20%)
- c) Student's ability to answer questions and demonstrate in-depth knowledge of the topic and area of research (30%)

Collectively, the poster presentation is worth 50% of the marks for the two communication units BMED4002 and 4005 (which equates to 25% of the marks for the Honours course).

BMED4003 Medical Research Thesis part 1

This unit encompasses the commencement of research that will contribute to the medical research thesis (completed in BMED4006), in particular the initial training, credentialing and induction required. The unit is "assessment continuing" and contributes (with BMED4006) to the 60% of marks allocated to the thesis.

A variety of various modules and credentialing/training activities are undertaken as part of the preparative work are required to safely undertake a research project; they are classed as pass/fail, but do not contribute to the grade. A copy or screenshot of the badge/certificate of completion must be uploaded into LMS as confirmation of completion.

Specific training modules

General lab health & safety training typically involves Building Safety Inductions, reading and certifying the lab safety manual and the UWA Biosafety 1 (Biohazards) unit, an online quiz that is completed via LMS (look under the *Community* tab, find *Biosafety* under "organizations" and enrol under '*UWA-Biosafety- Induction*'). A range of information on health services can be accessed here:

<http://www.student.uwa.edu.au/experience/health>

Students who are using laboratory animals during their Honours Project must complete the PAWES (Program in Animal Welfare, Ethics and Science) course, taught by the UWA Office of Research. Please note that this course fills up quickly and students should reserve a space as early as possible. The course is time- tabled on the Research UWA website and usually becomes available in January of the year of your Honours:

<http://www.Research.uwa.edu.au/staff/animals/pawes>

The online Gene Technology Awareness Session is essential for students who work with Genetically Modified Organisms (GMOs), and for anyone who works within (or administers) a facility certified by the Office of the Gene Technology Regulator (OGTR). Details can be found at:

<https://www.class2go.uwa.edu.au/enroll/3MHEFE>

Students carrying out clinical research involving recruitment of patients may need to do additional training (e.g. CPR; Defibrillation; Aggressive Incident Management; Manual Handling; How to obtain Informed Consent; etc); supervisors will need to be aware of these requirements and organise the appropriate modules for their students. Projects involving human participation will require approval from a Human Research Ethics Committee before commencement; applying for ethical approval may be part of the Honours project.

BMED4004 Research Ethics, Rationale and Design

1. **Basic Chemistry for Lab Researchers module:** Students will be required to successfully complete this refresher module available via the LMS. Students must obtain a minimum of 80% in the associated quiz to pass this module.
2. **Research Ethics module:** Students will watch and actively participate in interactive videos, provided via the LMS, at the start of semester. Students will successfully complete the following online courses: i) Research Question (Global Health Training Centre), ii) Essential Elements of Ethics (Global Health Training Centre) and iii) Good Clinical Practice (Western Australian Health Translation Network). Students must obtain a minimum of 80% in quizzes associated with each course to obtain a certificate and pass this module.
3. **Project rationale:** Students will also be required to prepare and submit a project summary and rationale by late March (this allows timely feedback for preparing the literature review and research plan). The objective of this task is for the student to clearly and succinctly describe the study rationale, objectives and design in lay-friendly language similar to that required for a grant or ethics application. The project rationale must be no more than two A4 pages in length (Submission must be in English, typewritten using 12 point, Times New Roman Font, 1.5-spacing throughout. Each page should consist of a single column of text with the following margins: 15 mm for left and right, and 25 mm for top and bottom) including references; figures and tables are not allowed. Feedback will be provided by the unit coordinator. Students must obtain at least 50% to pass this module.
4. **Statistics in Medical Research module:** An understanding of basic statistical approaches and techniques is necessary to design a robust study and properly analyze and interpret research data. The statistics module will be run as three weekly workshops, using real-world data to teach practical biomedical statistical analytical principles. Attendance at all workshops is required and completion of short assessment pieces are required in order to pass this module.

Note: where specialised statistical approaches and facilities are required (e.g., genetic analysis, multivariate statistics, 'omic data analysis), these will be taught by your supervisor using the own tools and platforms employed within their labs.

The Statistics in Medical Research module will cover the following:

- *Descriptive statistics:* Mean, median, mode; standard deviation and standard error; confidence intervals; normalcy of distribution; statistical outliers; data visualization, graphing and plotting.

- *Parametric statistics*: Probability testing; t-test, ANOVA, ANCOVA, analysis of repeated measures.
- *Nonparametric statistics*: Chi-Square, Fisher's exact test, Wilcoxon (paired or unpaired), Kruskal-Wallis ANOVA, Survival Analysis, etc.
- *Curve fitting*: Correlations, smoothing, linear regression, multiple linear regression, non-linear regression (e.g., dose-response curves), logistic regressions (where the dependent variable is categorical), factor analysis, principle component analysis.
- *Power analysis*: hypothesis testing, assumptions, effect size, sample size.

The modules will include training on the use of the free software package **Jamovi** to conduct statistical analyses and presentation of data (<https://www.jamovi.org/download.html>).

Students are required to pass each module in order to pass BMED4004. Furthermore, failure in any or all of these components will result in failure in the entire Medical Research Honours course. Attendance at all workshops is required and completion of short assessment pieces are required in order to pass this module. More details on teaching delivery and deadlines for completing each module will be provided by the beginning of the first semester.

BMED4006 Biomedical Research Thesis Part 2

At the completion of the Honours program, students will be required to submit a compact medical research thesis for marking, consisting of a brief introduction, followed by description of the research methods, findings and conclusions; the thesis ends with the bibliography and appendices. The introduction can be based on the original literature review and research proposal (submitted in BMED4001), significantly reduced in size, taking into account feedback suggested by the examiners and reflecting any changes in project scope that occurred during the Honours year. No figures or tables should be included. The bibliography should be trimmed and updated to include new references and papers relating to the Materials & Methods, Results and Discussion.

The thesis must be submitted as a PDF file for examination. Supervisors can and should provide feedback to the student before submission with respect to writing style, content, formatting and referencing. Feedback can be provided on the Introduction, Methods and Results, but **NOT** the discussion, which should entirely reflect the student's own work. The size and content of the thesis is outlined below; students must note the structure, layout and word limits and ensure these are followed. **Word limits are NOT optional.**

Structure and layout:

The thesis should be laid out according to the following guidelines:

1. Title page: stating the title of the thesis, the name and number of the submitting student and the name(s) and affiliations of the supervisor(s).
2. Contribution statement page: A signed statement indicating the contribution of the student to the work contained in the thesis submitted as part of the requirement for the Honours degree (a pro forma page will be provided). Contributions by others should be specifically stated, quantified if necessary.
3. Acknowledgement page: formal acknowledgement and thanks to those who helped with the project or who provided materials, data or other support.
4. Table of contents: neatly formatted and accurately paginated, listing major and minor headings.
5. Structured abstract: A single page summary of the project with the following sub-headings: introduction/rationale and aims, methods, results and conclusions.
6. Introduction, hypothesis and Aims: An updated and condensed version of the literature review providing a concise summary of the background, rationale and aims of the project. Bibliography should be included and augmented to encompass references cited in the remainder sections. Sources should be numbered according to the order in which they are cited (Vancouver referencing style).
7. Materials and Methods: relatively succinct description of the methodological and analytical aspects of

the study, written so that a suitably trained reader will be able to understand the approaches and procedures undertaken and replicate them. Reagents, platforms and equipment should be briefly defined in text as per a research manuscript. All methods and approaches should be properly referenced.

8. **Results:** A concise listing and description of the data and findings generated by the project; the use of properly formatted and labelled diagrams, figures and tables is strongly recommended (note: figure legends and tables are not included in the word limit). Statistical significance of findings should be clearly annotated. Discussion and interpretation should be avoided in this section.
9. **Discussion:** A structured, logical, informed and balanced discussion of the findings and significance of the project, noting any strengths, flaws or gaps in the study, and identifying opportunities or requirements for further study. Importantly, the Discussion must not be read or edited by the supervisor – it should reflect the student’s work alone.
10. **Bibliography:** A full, complete and accurate listing of all of the papers and sources of information cited in the thesis, numbered according to the order in which they are cited. Students are encouraged to use recent, high-quality reviews to support general statements and the state of knowledge around broad topics, while specific studies of particular relevance (old and/or new) should be cited individually.
11. **Appendices:** supplementary information, figures or data generated during the research, supplied for information but not formally assessed.

Word limits:

It is intended that the Honours research thesis should be a compact, readable document that provides a vehicle for students to adequately present their research work in a concise, lucid and easily examinable format. Word limits are in place to prevent the thesis becoming too large and unwieldy, and to reflect the reality of research practice where size constraints are commonplace when publishing or reporting research.

The word limits for the various sections of the thesis are as follows:

Section	Word limit
Title	25 words
Abstract	500 words
Acknowledgments	No limit
Table of Contents	No limit
List of Figures/List of Tables/Abbreviations	No limit
Introduction, Hypothesis and Aims	1000 words
Materials & Methods*	*3000 words combined
Results	
Discussion	2500 words
Bibliography	No limit
Appendices**	No limit

*Combining the Materials & Methods and Results in a single word limit is intended to allow projects that have a major method development/optimisation component (with few results) to be properly and adequately presented without being penalised.

** Appendices will not be formally assessed by the examiners (no word limit).

Printing and formatting:

The Thesis should be formatted in Word, in colour, and saved as a PDF file for submission and printing if desired. File size should be less than 10 MB if possible. The page size should be A4 with margins of 2 cm (width) and 2.5 cm (height). All pages from the Introduction onward should be numbered, with the page number located on the bottom right of the footer. The header should be left blank.

Electronic submission (as a PDF file) is required for the Honours thesis submission; these will be emailed to the examiners. Hard copies can be printed at the student's expense if needed.

Submission

Thesis is to be submitted electronically for examination via LMS.

Assessment grade guidelines

The marking bands for assessment are as follows:

Class	When apportioning marks please take into account the grades used by UWA
First class	90% - 100% H1: HD+ 80% - 89% H1: HD- Outstanding ability in research and communication.
2A Honours	75% - 79% H2A: D+ 70% - 74% H2A: D- Very competent. Candidate still very worthy of consideration for a postgraduate research award.
2B Honours	60% - 69% H2B: CR Competent but some inadequacies in content, scope understanding and/or presentation such that the person would be unlikely to make a good independent research worker.
Third class honours	50% - 59% H3: Pass Evidence of effort but inadequacies in research competence, understanding and/or presentation.
Fail	Fail: N+ Less than 50 % Unsatisfactory. Very serious inadequacies in all or most areas.

Similar general expectations to these will apply to the other items of assessment, that is, the Literature Review & Research Plan, the Preliminary Seminar, and the Poster Presentation & Interview.

ALL UNITS MUST BE PASSED TO PASS HONOURS.

LATE PENALTIES: All late submissions will incur a 5% mark penalty per day. Please see LMS unit outlines and [Exceptional Variation of Assessment \(EVA\) Policy \[UP23/11\]](#).

Thesis assessment policy

Thesis documents are emailed to two independent examiners for assessment. Where there is a difference in mark greater than 10%, or the examiners' score the thesis in a different grade band (e.g. H1 vs. H2A), then the examiners are requested to undertake a review of their assessment; a discussion chaired by the UC may be undertaken to resolve any differences. If, after the review, the discrepancy still remains significant, a third examiner will be asked to assess the thesis. The marks of the closest two examiners will be averaged to generate the final mark.

Responsibilities of the Honours Student

One of the exciting challenges of the Honours year is that you will encounter many challenges and learning curves. The inevitable downside of this is that each task will take longer than anticipated, so it is important to be highly organised. As an Honours student you should:

- Ensure you are aware of all the important dates and deadlines, as penalties for late submissions do apply.
- Ensure you manage your time carefully so that the requirements of the Honours course are completed within the stipulated time limits. Although it is understood that many students need to take on part-time work for financial reasons, ensure that this is kept to a reasonable level (e.g. less than 8 hours per week).
- Obtain a Medical Certificate to receive an [Exceptional Variation of Assessment \(EVA\) Policy \[UP23/11\]](#) if you are ill during the year. Consultation with the unit coordinator(s) is compulsory for requesting extensions longer than a week.
- Ensure you are aware of Unit requirements, particularly with respect to security and the safe and responsible usage of facilities such as the Internet and core equipment. If in doubt, consult your Supervisor, the Honours coordinators or the Senior Technical Officer (Sarah Power).
- Document all your experimental work in a laboratory book and show it to the Supervisor on a regular basis. It is a requirement that the laboratory notebook is accurately completed and remains the property of the Supervisor for up to five years post-Honours. Make sure that you protect electronic data by backing it up regularly (where your supervisor can get access) and having copies saved on several different sites. Don't store data where it can be lost or stolen.
- Be aware of the Guidelines on Research Ethics and Research Conduct, as outlined in http://www.Research.uwa.edu.au/policies3/guidelines_on_research_ethics_and_research_conduct
- Arrange regular meetings with your Supervisor to discuss all aspects of your work.
- Be open to suggestions and advice from your Supervisor, particularly during the early stages; as the year progresses you should grow in confidence and show signs of independence and initiative.
- Ensure that any conflicts that might develop with Supervisors or others are brought to the attention of the Honours Coordinators so that problems can be resolved quickly and amicably.
- Uphold the academic standards and good reputation of the School of Biomedical Sciences.

Responsibilities of the Honours Supervisor

The Supervisor is responsible for all matters directly related to the Research project. Specifically, the Supervisor should:

- Provide academic guidance with respect to the overall direction, day-to-day running of the research project, and editing and feedback on written and oral tasks.
- Meet frequently with the student, and establish open and good communication
- Ensure the appropriate level of support and training is provided to the student, including resourcing and regulatory approvals
- Be a good listener, and offer encouragement for good ideas and well-developed thoughts, with constructive criticism where appropriate.
- Keep the student informed about relevant regulations and administrative processes in the Unit, School and University
- Guide, advise, help, constructively criticise, but not push – it is the student's responsibility to be motivated to succeed and to assume ownership of the research project.

- Make arrangements for continuing supervision during periods of absence.
- Provide advice and guidance on the preparation of the:
 - Research proposal seminar,
 - Literature Review & Research Plan,
 - Research thesis, and
 - Poster presentation & defense
- Provide relevant feedback to the Honours Coordinator and Examiners by completing the 'Supervisor's Assessment' form (Appendix B).
- Participate as an observer during the poster presentation and defense.
- Attend an Examiners Meeting at end of year, if required, to enable major differences in thesis marking between examiners to be resolved.

Responsibilities of the Honours Examiner

Each student will have two examiners with expertise in the area of the project. The Examiners take part in multiple aspects of assessment of students. Specifically, the Examiners should:

- Attend and assess the research proposal seminar (late April)
- Read and assess the literature review & research plan and provide feedback on the work.
- Read and mark the research thesis (late October)
- Attend and participate in the Poster Presentation & defense (late October/early November)
- Contribute to achieving a fair and equitable grade for the student through discussions and negotiation with the Course Coordinator(s) via email, phone or online communication (early November)

Responsibilities of the Honours Coordinator

The Honours coordinator is responsible for organising and overseeing the entire Honours course. Specifically, the Honours Coordinator will:

- Call for Honours projects (July) and prepare the Honours booklet (September).
- Coordinate conditional enrolment of Honours students (from September onwards).
- Ensure all students are correctly enrolled and installed in the supervisors' labs (January/February).
- Organise Honours Orientation program and safety/training modules (for late-February).
- Organise welcome function for Honours students (late-February) and introduce them to the structure, assessment expectations and timelines of the course.
- Assign examiners to each student (late February).
- Organise Project proposal seminars (early May)
- Collect Literature Review/Research Plan from Students and distribute to examiners (late May); provide feedback to students from the examiners.
- Informally check on students' progress (May to August).
- Distribute the Research Thesis to examiners, together with marking guidelines (mid-October) and coordinate with examiners regarding the final grade.

- Organise and run the conference Poster Presentation sessions (late October) and collate the examination marks.
- Present the final marks and grades to the Board of Examiners for approval and submission.

Plagiarism

Plagiarism is defined as appropriating someone else's words or ideas without acknowledgment. New ideas and findings are crucial to the advancement of knowledge and are typically published in international journals under particular authors' names. It is extremely important that this credit be properly assigned for personal, ethical, financial and historical reasons. As scholars, we have to rigorously acknowledge previous contributions if we are to expect that in turn, we will be acknowledged in the future.

Copying material from a published source without properly citing the source, or copying from another Honours student or other thesis constitutes plagiarism. The University has strict rules about [academic integrity](#), and views plagiarism within a thesis as major misconduct.

If you are in doubt as to what constitutes plagiarism, make sure you consult your Supervisor or Honours Coordinator, and/or consult the University policy on Academic Misconduct:

<https://www.uwa.edu.au/students/-/media/Project/UWA/UWA/Students/Docs/UWA-Academic-Conduct-Policy.pdf>

Appendix A: Marking Guides

Literature Review and Research Plan Assessment Sheet

Page 1 of 2, please see below

Examiners: please complete the *assessment sheet (page 2)* and email it back to the Honours Coordinator *within* 2 weeks of receipt.

Some criteria to aid in your assessment have been listed below:

The review:

- Provides an appropriate and informed review of the relevant literature to introduce and support their hypothesis and does not include irrelevant Literature?
- Demonstrates critical thinking.
- Follows a logical flow and progression.
- Identifies gaps in knowledge leading into research question.
- Contains high quality and appropriately labelled diagrams and tables.
- Discusses the potential significance and impact of the project?

The proposal:

- Describes a well-designed program of research.
- Includes an overall hypothesis that is subdivided into well-defined objectives or specific aims.
- Provides an overview of the proposed experiments that are linked to the proposed aims. This should include experimental materials and methods, data management, analysis and statistics, ethics and recruitment of participants as required.
- Demonstrates that the student understands their project.

Grammar and formatting:

- Is there use of clear and concise scientific English with accurate spelling, punctuation & grammar?
- Is the document formatted correctly (page size, numbering etc) with appropriate use of the correct font (12 point Calibri) and headings?
- Are all figures/tables labelled and referred to in text?

Referencing:

- Are all scientific/factual statements and descriptions correctly referenced?
- Is the formatting of in-text citations uniform?
- Does the bibliography include an appropriate mix of reviews, original research, and recent plus older publications?
- Is the bibliography content and format consistent and accurate?

Student Name: _____ Assessor Name _____

Assessment Criteria	Mark
Literature review (50 marks) a) Does it provide a review of the relevant literature to support the hypothesis and does not include irrelevant literature? b) Demonstrate critical thinking? c) Follow a logical progression? d) Identify gaps in knowledge leading into research question. e) Appropriate use, quality and labelling of diagrams and tables. f) Include an overall hypothesis that is subdivided into well-defined objectives or specific aims? g) Discuss the potential significance of the project?	
Research proposal (35 marks): a) Provide a well-designed program of research? b) Provide an overview of the proposed experiments that are linked to the proposed aims? This should include experimental materials and methods, data management, analysis and statistics, ethics and recruitment of participants as required. c) Demonstrate that the student understands their project?	
Grammar, formatting and referencing (15 marks): a) Is it clear and concise scientific English? b) Is the document formatted appropriately and the spelling, punctuation & grammar correct? c) Is the document in 12pt Calibri font? d) Is the page size A4 with margins of 2 cm (width) and 2.5 cm (height) used consistently throughout the text? e) Are subheadings, page numbers, headers/footers consistent? f) Are all figures/tables labelled and referred to in text? g) Is the document fully and accurately referenced: <ul style="list-style-type: none"> • Statements correctly referenced. • Formatting of in-text citations is uniform. • Inclusion of reviews, original research, and recent plus older publications in the bibliography. • Consistency and accuracy of the bibliography content and format. 	

Assessor Signature _____

Date _____

Scales: *H1-D+* (90 – 100%); *H1-D* (80 – 89%); *H2A-D+* (75 – 79%) *H2A-D* (70 – 74%); *H2B* (60 – 69%); *H3* (50 – 59%); *F* (<50%)

Thesis Assessment Sheet

Page 1 of 2, please see below

Thesis Assessment Criteria	Mark
Abstract, Introduction, Aims and hypothesis (15 marks) a) Is the Abstract structured correctly and did it summarise succinctly and accurately the rationale, aims, findings and outcomes of the study, and could it be understood without reading the rest of the Thesis? b) Did the Introduction provide a summary of the study's background and rationale, including identification of any pertinent gaps or controversies? c) Did the Introduction cite appropriate references from the scientific literature? Were the Aims and Hypotheses clear and valid?	
Materials and Methods (15 marks) a) Were the Materials and Methods clearly described and fully referenced? b) Were the Methods used appropriate and valid for the stated aims?	
Results (30 marks): a) Does the Results section represent an adequate body of work? b) Are the results presented clearly and accurately? c) Were appropriate choices of experimental conditions, such as doses, concentrations, time-points, etc. used? Were sufficient controls and replicates performed? d) Were appropriate numbers of observations performed? e) Was there sound and appropriate use of statistical analyses and tests? f) Was the presentation of results (Figures, Tables, etc.) clear and logical?	
Discussion (20 marks): NB Please take into account that the discussion is written by the student with NO input from the supervisor. a) Is the Discussion relevant to the Introduction, Methods and Results? b) Is it logical in presentation and content? c) Is there evidence of critical and creative analysis? d) Does it place the findings in the context of past studies? e) Are there suggestions for future studies? Is there evidence of over-interpretation of data? f) What is frequency and extent of bias in interpreting the data? g) Have unexpected or inconsistent results been fairly and skillfully discussed?	
References (10 marks): a) Is the in-text citation style appropriate and consistent? b) Is the reference list free from careless errors? c) Is the content of the Thesis supported with appropriate in-text primary research citations, or is there over-reliance on reviews?	
Style and Presentation (10 marks): a) Is the Thesis well-organised (e.g. appropriate use of subheadings), succinct and clear? b) Is it of an appropriate length? c) Does the Thesis demonstrate an appropriate use of grammar & punctuation? d) Has care been demonstrated to avoid spelling mistakes and typographical errors? Has the nominated bibliographic style been followed consistently?	

Assessor Signature _____

Date _____

Scales: **H1-D⁺** (90 – 100%); **H1-D⁻** (80 – 89%); **H2A-D⁺** (75 – 79%) **H2A-D⁻** (70 – 74%); **H2B** (60 – 69%); **H3** (50 – 59%); **F** (<50%)

Student Name: _____ Assessor Name _____

Thesis Assessment Criteria	Comments
<i>Abstract, Introduction, Aims and hypothesis (15 marks)</i>	
<i>Materials and Methods (15 marks)</i>	
<i>Results (30 marks):</i>	
<i>Discussion (20 marks):</i> NB Please take into account that the discussion is written by the student with NO input from the supervisor.	
<i>References (10 marks):</i>	
<i>Style and Presentation (10 marks):</i>	

Comments:

Assessor Signature _____

Date _____

Appendix B: Supervisor's Assessment Form

Student Name:

Supervisor Name:

The aim of this form is to allow supervisors the opportunity to provide confidential feedback on the overall level of commitment, engagement, communication, effort and achievement on the part of their students. You are encouraged to consult with any co-supervisors or team members. This form will not be shared with the students.

Please assess your student's performance against the five key attributes listed below, using the four qualitative descriptors provided. Two examples are provided at the end of the form to help with calibration.

You may also provide written feedback if desired, but this is not a requirement.

Attribute	Assessment*
1. Motivation & commitment	
2. Participation, collegiality & engagement	
3. Communication	
4. Written submissions and deadlines	
5. Effort and accomplishment	

*Poor, satisfactory, good, excellent

What is your overall assessment of the student's Honours performance, taking into account project challenges, complexity, effort and accomplishments?	/100
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NB: this feedback evaluation is worth 10 of the 60 total marks awarded for this unit.

For your information the marking bands for assessment are as follows:

Class	Mark breakdown
HD1	90% - 100% H1: HD+ 80% - 89% H1: HD-
HD2A	75% - 79% H2A: D+ 70% - 74% H2A: D-
HD2B	60% - 69% H2B: CR
Third Class	50% - 59% H3: Pass
Fail	Fail: N+ Less than 50 %

Any other feedback/comments (optional):

Example 1 (poor):

1. Lacked enthusiasm and engagement for the project; unmotivated and needing frequent prompts/reminding to get things done; no initiative; poor work ethic.
2. Frequently absent from lab meetings/journal clubs; rarely contributed to discussions or asked questions; no attempts made to integrate with the rest of the team.
3. Poor oral and/or written communication with supervisor or team members.
4. Late to submit work for feedback; not meeting submission deadlines; poor quality drafts with little evidence of improvement after feedback.
5. Minimal effort in mastering new skills; large amounts of unproductive time; required constant supervision, with poor technical competency; slow to generate data or outputs; poor project awareness and problem-solving abilities.

Example 2 (Excellent):

1. Extremely enthusiastic and engaged in the project; self-motivated, independent, showed high levels of initiative; excellent work ethic.
2. Always present at lab meetings/journal clubs; frequently contributed to discussions and asked questions; highly collegial and active, valued team member..
3. Excellent oral and written communication with supervisor and/or team members.
4. Submitted work with plenty of time for feedback; consistently met submission deadlines; provided well-written and edited drafts, demonstrating significant improvement after feedback
5. Worked very hard to master new skills; worked productively and independently, with negligible down-time time; highly competent technically; generated large amounts of data or outputs; highly developed and mature project awareness and excellent problem-solving abilities.

School of Biomedical Sciences

Honours in Medical Research

Seminar Reflection Worksheet

Student name and date:

Seminar presenter (name):

Seminar title or topic:

Background and rationale:

Design & methodology:

Findings and take-home message:

Presentation lessons:

Enrolment in Honours in Medical Research for 2026

DECIDING ON AN HONOURS PROJECT

A list of Honours projects for 2026 is provided in the final section of this Booklet. Once you have identified a project of interest you should promptly contact the project Supervisor(s) to discuss the project. If you and a Supervisor agree to you undertaking a particular project you should then you can formally apply for entry to the Honours program.

All students must identify a project and supervisor before attempting to enrol.

Note: supervisors often have other projects that may not be listed in this booklet; if you are interested in undertaking an Honours project with a specific supervisor, arrange to meet with them and discuss options.

Identify the appropriate Entry Requirements

Students with a Biomedical Sciences or Biomedical Major:

If you have completed a Biomedical Science Major with an average of 65% or better in third year Units contributing to the major, you are eligible to apply directly online:

<https://handbooks.uwa.edu.au/undergraduate/honoursdetails?code=HON-BIOMS>

Students without a Biomedical Sciences or Biomedical Major:

The key requirement is that both UWA- and non-UWA applicants can demonstrate the equivalent of a 65% average in third year major units in disciplines that are relevant to their proposed project. Supporting documentation uploaded must include a brief research proposal with confirmation from the relevant supervisor, School or Research Institute that general facilities are available to support the project.

Submit an application

Step 1: Register your agreed project with the UWA Biomedical Sciences office.

Once you have met with your prospective supervisor, the project has been confirmed, and the necessary induction programmes have been advised, please complete the yellow 'Application for Medical Research Honours form' included in this Handbook. This Form must be submitted to the Administrative Office, School of Biomedical Sciences, UWA, prior to enrolling so that the School knows that a project and Supervisor have been assigned to you, when making a decision regarding approval of your on-line application.

Step 2: Apply to the Honours Programme.

Visit the UWA Honours page for links to the UWA application portals

<http://www.studyat.uwa.edu.au/courses-and-careers/honours#aust>

Use the following codes when applying:

BMED (Honours), Course Code: BH006, Major/Program Code: HON-BIOMS,

Step 3: Enrol in the appropriate units.

You will need to enrol in the Bachelor of Biomedical Science (Honours) six units shown below: BMED4001, BMED4002, BMED4003, BMED4004, BMED4005, BMED4006.

Please refer to Student Central for advice on enrolment dates and fees.

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Application for Honours in Medical Research 2026

To be filled in by STUDENT:

Name:

Student No:

Primary Supervisor:

E-mail address:

Contact Number:

Mailing address:

Biomedical Sciences Supervisor (if different from above):

E-mail address:

Contact Number:

Mailing address:

Project Title:

Honours Booklet Page No: (if applicable)

To be filled in by SUPERVISOR:

Names of **two** suitably qualified examiners who have agreed to examine the student:

Name:

Position & Institution:

Email address:

Contact number:

Name:

Position & Institution:

Email address:

Contact number:

Honours Induction Checklist – Supervisors, please indicate whether the above student will need to take any of the following induction programmes or ethics approvals:

☐ PAWES (Program in Animal Welfare, Ethics and Science)

☐ Gene Technology

☐ Radioisotope handling course

Other programmes required:

☐

☐

Ethics Requirements:

Non-human animal Research ethics approval - required ☐ already approved ☐

Human Research ethics approval - required ☐ already approved ☐

I agree to supervise this student in honours for 2026.

Supervisor's signature:

Date:

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Honours Projects Available in 2026

	Title	Primary Supervisor(s)	Project Title	Page
1	Prof	Alexey Terskikh	Is the aging process coordinated between different cell types within individual organs and tissues and between different organs and tissues?	34
2	Prof	Alexey Terskikh	How does cell heterogeneity change with age? Does the 2nd law of thermodynamics (entropy increase) apply to ageing of tissues and organs?	36
3	Prof	Alexey Terskikh	Could aging be reversed by reestablishing the “young” epigenetic state in vivo using transient expression of Yamanaka factors?	38
4	Dr	Alicia Brunet	Early development characterisation of neuronal cone migration in a mouse model of vision loss	40
5	Dr	Alicia Brunet	Investigating the molecular changes in the retinal environment of mouse models of inherited vision loss	42
6	Dr	Alicia Brunet	Identifying pyroptosis as the main culprit for cone degeneration in inherited retinal diseases	44
7	Dr	Alistair Cook	Exploring the effects of radiation on the tumour immune microenvironment	46
8	Dr	August Mikucki	Determining the role of a novel RNA chaperone in the virulence of Group A <i>Streptococcus</i>	47
9	Dr	Belinda Guo	Leukaemia monitoring using circulating DNA fragments	49
10	Dr	Belinda Guo	Using platelet genetics to monitor blood cancers	50
11	Dr	Bree Foley	From Donor to Design: Empowering NK Cells for Enhanced Cancer Killing	51
12	Dr	Bree Foley	Impact of Early-Life Cancer Therapy on Long-Term Immune Competence	52
13	A/Prof	Debbie Palmer	Determinants of infant feeding outcomes in allergic families	53
14	Adj. A/Prof	Elaine Wong	Development of Usher syndrome 1D patient-derived inner ear organoid model	55
15	Dr	Erika Bosio	Modelling a human microvessel network	57
16	Dr	Filippo Valente	Non-invasive Treatment of Acute Otitis Media Using Thermosensitive Silk Hydrogels	59
17	Dr	Hannah Newnes	Characterising tumour-infiltrating lymphocytes to inform personalised cancer therapy	61
18	Dr	Henry Hui	Precision diagnosis for the care of patients with blood cancers	62
19	Dr	Jessica Mountford	Characterising genetic variance associated with early-onset high myopia in zebrafish.	64
20	Dr	Jonathan Chee	Disrupting Copper Homeostasis to Enhance Immunotherapy and Chemotherapy in Mesothelioma	67
21	Dr	Jonathan Chee	Targeting Lipid Metabolism to Improve Immunotherapy in Lung Cancer and Mesothelioma	69
22	Dr	Jun Yuan	Identifying eBMD-related proteins and the association with incidence dementia’s risk	71

23	Dr	Kai Chen	Spatially measuring single-cell metabolism in the bone marrow	73
24	Dr	Kai Chen	Visualising bone marrow cell proliferation at nanoscale using thymidine-based imaging	74
25	A/Prof	Kate Hammer	Artificial skin models for evaluating the activity of topical antiseptics and honey	75
26	A/Prof	Kefyalew Alene	Applications of artificial intelligence for improving tuberculosis control	77
27	Dr	Liang Wang	Dynamic changes of gastric microbiota at different stages of Correa's cascade in the development of intestinal type gastric cancer	79
28	Dr	Liz Johnstone	Investigation of G Protein-Coupled Receptor Molecular Pharmacology	82
29	Dr	Lucy Furfaro	The art of induction: Uncovering Group B Streptococcus prophage functionality	83
30	Dr	Lucy Barrett	Assessing the local immune response to injury in burn tissue.	85
31	A/Prof	Lynette Fernandes	Identifying the processing demands of summative assessments in pharmacology	87
32	A/Prof	Matthew Payne	Swimming Upstream – Assessing the impact of chlorinated pool exposure on the vaginal microbiome	88
33	Prof	Mark Nicol	Understanding the role of <i>Moraxella</i> species in the airways: exploring interactions with human epithelial cells	90
34	Dr	Raelene Endersby, Annabel Short, Brittany Dewdney, Alison McDonnell	Developing innovative treatments for paediatric brain cancers	93
35	Dr	Minda Sarna	Effect of maternal pertussis vaccines on childhood pneumococcal vaccine effectiveness.	95
36	Dr	Mitali Sarkar-Tyson	Investigating the Effects of Inhibiting Mip and EptA in Chlamydia-Gonorrhoeae Co-infections	97
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Project title:	Is the aging process coordinated between different cell types within individual organs and tissues and between different organs and tissues?
Project location:	Perkins QEII 6 th floor
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Background</p> <p>The Terskikh laboratory discovered that patterns of epigenetic marks in the nucleus inform cellular identities and developed a novel technique: microscopic imaging of epigenetic landscapes (MIEL). Application of this technique to aging, termed image-based chromatin and epigenetic age (ImAge), captures intrinsic age-related progressions (trajectories) of the spatial organization of chromatin and epigenetic marks in single nuclei.</p> <p>The Terskikh laboratory employs both in vitro (cell culture) and in vivo (transgenic and reporter mice) models to understand the ageing process at the single cell level including the global age-related changes of chromatin and epigenetic landscape.</p> <p>The laboratory thrives on the crossroads of several interrelated fields and techniques, embracing a wide range of collaborations with leading researchers in the outstanding scientific environment of the Harry Perkins Institute, the University of Western Australia and worldwide (in particular, The Salk Research Institute and the University of California San Diego).</p> <p>Question</p> <p>Is the aging process coordinated between different cell types within individual organs and tissues and between different organs and tissues?</p> <p>Aims</p> <p>To quantify the pace of ageing in several mouse organs and tissues at single cell level. Whenever possible, correlate such pace of ageing with functional readouts.</p>

	<p>Design</p> <p>To address this question, we will develop focused pipelines for quantifying ageing trajectories in isolated cells/nuclei and in situ (spatial aging) in different organs and tissues. Whenever possible we will connect that knowledge with the changes in organs and tissues function.</p>
	<p>Techniques</p> <p>Immunolabeling, fluorescent microscopy, mouse behaviour analyses, advance computing. Possible integration with molecular and cellular biology approaches, including RNA-seq, ATAC-seq, Cut&Tag, and spatial OMICS at the single cell level.</p>
	<p>Outcomes</p> <p>This knowledge will advance our understanding of the cellular mechanism of aging and will help develop and test interventions that promote organ and tissue function.</p>
	<p>References</p> <p>Alvarez-Kuglen M, Ninomiya K, Qin H, Rodriguez D, Fiengo L, Farhy C, Hsu WM, Kirk B, Havas A, Feng GS, Roberts AJ, Anderson RM, Serrano M, Adams PD, Sharpee TO and Terskikh AV, Sept 2024, ImAge quantitates aging and rejuvenation. In: Nature Aging. 4, 9, p. 1308-1327 20 p.</p> <p>Ninomiya K and Terskikh AV, Sept 2024, Imaging the epigenetic landscape in single cells to study aging trajectories. In:Nature Aging. 4, 9, p. 1184-1185 2 p.</p> <p>Farhy C, Hariharan S, Ylanko J, Orozco L, Zeng F, Pass I, Ugarte F, Forsberg EC, Huang CT, Andrews DW, and Terskikh AV, Improving drug discovery using image-based multiparametric analysis of the epigenetic landscape. In: eLife. 8 (2019).</p>

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBC Email:
Project title:	How does cell heterogeneity change with age? Does the 2nd law of thermodynamics (entropy increase) apply to ageing of tissues and organs?
Project location:	Perkins QEII 6 th floor
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Background</p> <p>The Terskikh laboratory discovered that patterns of epigenetic marks in the nucleus inform cellular identities and developed a novel technique: microscopic imaging of epigenetic landscapes (MIEL). Application of this technique to aging, termed image-based chromatin and epigenetic age (ImAge), captures intrinsic age-related progressions (trajectories) of the spatial organization of chromatin and epigenetic marks in single nuclei.</p> <p>The Terskikh laboratory employs both in vitro (cell culture) and in vivo (transgenic and reporter mice) models to understand the ageing process at the single cell level including the global age-related changes of chromatin and epigenetic landscape.</p> <p>The laboratory thrives on the crossroads of several interrelated fields and techniques, embracing a wide range of collaborations with leading researchers in the outstanding scientific environment of the Harry Perkins Institute, the University of Western Australia and worldwide (in particular, The Salk Research Institute and the University of California San Diego).</p> <p>Question</p> <p>How does cell heterogeneity change with age? Does the 2nd law of thermodynamics (entropy increase) apply to ageing of tissues and organs?</p> <p>Aims</p>

	<p>To quantify the heterogeneity (i.e. Shannon and Rao's quadratic entropies) of ageing in different organs and tissues at single cell level.</p>
	<p>Design</p> <p>To address this question, we will develop focused pipelines for quantifying heterogeneity in isolated cells/nuclei and in situ (spatial aging) in different organs and tissues. Whenever possible we will connect that knowledge with the changes in organs and tissues function.</p>
	<p>Techniques</p> <p>Immunolabeling, fluorescent microscopy, mouse behaviour analyses, advance computing. Possible integration with molecular and cellular biology approaches, including RNA-seq, ATAC-seq, Cut&Tag, and spatial OMICS at the single cell level.</p>
	<p>Outcomes</p> <p>These experiments will elucidate fundamental principles of mammalian organs and tissues aging and may help develop and test novel type of biomarkers based on heterogeneity changes in different organs and tissues.</p>
	<p>References</p> <p>Alvarez-Kuglen M, Ninomiya K, Qin H, Rodriguez D, Fiengo L, Farhy C, Hsu WM, Kirk B, Havas A, Feng GS, Roberts AJ, Anderson RM, Serrano M, Adams PD, Sharpee TO and Terskikh AV, Sept 2024, ImAge quantitates aging and rejuvenation. In: Nature Aging. 4, 9, p. 1308-1327 20 p.</p> <p>Ninomiya K and Terskikh AV, Sept 2024, Imaging the epigenetic landscape in single cells to study aging trajectories. In:Nature Aging. 4, 9, p. 1184-1185 2 p.</p> <p>Farhy C, Hariharan S, Ylanko J, Orozco L, Zeng F, Pass I, Ugarte F, Forsberg EC, Huang CT, Andrews DW, and Terskikh AV, Improving drug discovery using image-based multiparametric analysis of the epigenetic landscape. In: eLife. 8 (2019).</p>

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Project title:	Could aging be reversed by reestablishing the “young” epigenetic state in vivo using transient expression of Yamanaka factors?
Project location:	Perkins QEII 6 th floor
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Background</p> <p>The Terskikh laboratory discovered that patterns of epigenetic marks in the nucleus inform cellular identities and developed a novel technique: microscopic imaging of epigenetic landscapes (MIEL). Application of this technique to aging, termed image-based chromatin and epigenetic age (ImAge), captures intrinsic age-related progressions (trajectories) of the spatial organization of chromatin and epigenetic marks in single nuclei.</p> <p>The Terskikh laboratory employs both in vitro (cell culture) and in vivo (transgenic and reporter mice) models to understand the ageing process at the single cell level including the global age-related changes of chromatin and epigenetic landscape.</p> <p>The laboratory thrives on the crossroads of several interrelated fields and techniques, embracing a wide range of collaborations with leading researchers in the outstanding scientific environment of the Harry Perkins Institute, the University of Western Australia and worldwide (in particular, The Salk Research Institute and the University of California San Diego).</p> <p>Question</p> <p>Could aging be reversed by reestablishing the “young” epigenetic state in vivo using transient expression of Yamanaka factors?</p> <p>Aims</p> <p>To induce rejuvenation by partial reprogramming in mouse model and to investigate the changes of chromatin and epigenetic age as well as function in different organs and tissues.</p>

	<p>Design</p> <p>We will employ transgenic mice engineered with inducible cassette of Yamanaka factors in multiple organs and tissues. Aged mice will be administered with doxycycline to induce Yamanaka factors followed by behavioral and functional analyses. At the end of the experiment, several organs and tissues will be collected and the changes or chromatin and epigenetic age will be examined in isolated single cell or nuclei. Whenever possible, the observed changes will be correlated with the functional changes in corresponding organs and tissues,</p>
	<p>Techniques</p> <p>Immunolabeling, fluorescent microscopy, mouse organs and tissue isolation and analysis, mouse behaviour analyses, advance computing. Possible integration with molecular and cellular biology approaches, including RNA-seq, ATAC-seq, Cut&Tag, and spatial OMICS at the single cell level.</p>
	<p>Outcomes</p> <p>These experiments will elucidate fundamental principles of mammalian organs and tissues rejuvenation by partial reprogramming and may help develop and test novel type of therapies to improve the function and healthspan of the ageing organism.</p>
	<p>References</p> <p>Ocampo A, Reddy P, Martinez-Redondo P, Platero-Luengo A, Hatanaka F, Hishida T, Li M, Lam D, Kurita M, Beyret E, Araoka T, Vazquez-Ferrer E, Donoso D, Roman JL, Xu J, Rodriguez Esteban C, Nuñez G, Nuñez Delicado E, Campistol JM, Guillen I, Guillen P, Izpisua Belmonte JC. In Vivo Amelioration of Age-Associated Hallmarks by Partial Reprogramming [Internet]. <i>Cell</i>. 2016. p. 1719-1733.e12. Available from: http://dx.doi.org/10.1016/j.cell.2016.11.052 PMID: PMC5679279</p> <p>Alvarez-Kuglen M, Ninomiya K, Qin H, Rodriguez D, Fiengo L, Farhy C, Hsu WM, Kirk B, Havas A, Feng GS, Roberts AJ, Anderson RM, Serrano M, Adams PD, Sharpee TO and Terskikh AV, Sept 2024, ImAge quantitates aging and rejuvenation. In: <i>Nature Aging</i>. 4, 9, p. 1308-1327 20 p.</p> <p>Ninomiya K and Terskikh AV, Sept 2024, Imaging the epigenetic landscape in single cells to study aging trajectories. In: <i>Nature Aging</i>. 4, 9, p. 1184-1185 2 p.</p> <p>Farhy C, Hariharan S, Ylanko J, Orozco L, Zeng F, Pass I, Ugarte F, Forsberg EC, Huang CT, Andrews DW, and Terskikh AV, Improving drug discovery using image-based multiparametric analysis of the epigenetic landscape. In: <i>eLife</i>. 8 (2019).</p>

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SBMS Coordinating supervisor Contact details (for non-SBMSsupervisors)	Name: Prof Jeffrey Keelan Email: jeff.keelan@uwa.edu.au
Project title:	Early development characterisation of neuronal cone migration in a mouse model of vision loss
Project location:	Lions Eye Institute, Level 4 Harry Perkins Building
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims:</p> <p>As with other laminated neural structures of the central nervous system (CNS), the spatiotemporal cues underlying neuronal migration in the retina are essential for normal development and function¹. One class of retinal neurons, the cone photoreceptors, undergoes a phase of postmitotic, postnatal migration during retinal development^{2,3}. However, cone migration is aberrant in instances of retinal disease^{4,5}.</p> <p>Retinitis pigmentosa is a debilitating inherited vision disorder that affects 1:4000 people worldwide⁶. Mutations lead to the primary degeneration of rod photoreceptors, followed by secondary degeneration of cone photoreceptors⁷. The P23H mutation in the rhodopsin gene accounts for 10% of autosomal dominant retinitis pigmentosa cases⁸. The <i>Rho</i> P23H mouse is a model that replicates the pathophysiology of the disease exhibited in humans⁹. Whether cone migration is affected during retinal developmental stages of <i>Rho</i> P23H mice is unexplored and could be an indicator that this pathophysiology is also present in humans.</p> <p>Using <i>Rho</i> P23H mice, this project aims to:</p> <ol style="list-style-type: none"> 1. Characterise the cone migration pattern during early retinal development 2. Measure cone photoreceptor somata length and width 3. Quantify the number of cone photoreceptors at different ages
	<p>Design:</p> <p>Retinal tissue will be collected and processed for immunohistochemistry from <i>Rho</i> P23H and wildtype mice at early developmental ages: post-natal day 8 (P8), P12 and P16. Cone photoreceptors will be labelled with a fluorescent tag for their visualisation and quantification. Retinal tissue will be imaged using confocal microscopy and analysed using the image quantification software ImageJ for differences in cone migration position, somata length and width, and number of cone photoreceptors within <i>Rho</i> P23H mice compared to wildtype controls.</p>

	<p>Techniques</p> <ul style="list-style-type: none"> • Animal handling (mouse) • Eye and retinal dissections • Tissue processing and immunohistochemistry • Microscopy • ImageJ analysis • Extensive record keeping via excel • Statistical analysis <p>Outcomes:</p> <ul style="list-style-type: none"> • Differences in cone migration will be identified between <i>Rho</i> P23H disease mice and healthy controls to evaluate whether cone migration is disrupted in disease mice • Differences between cone somata length and width will be evaluated between <i>Rho</i> P23H disease mice and healthy controls • Cone numbers will be quantified to assess whether <i>Rho</i> P23H mice begin losing cone photoreceptors at early developmental ages compared to healthy controls
	<p>References:</p> <ol style="list-style-type: none"> 1 Baye, L. M. & Link, B. A. Nuclear migration during retinal development. <i>Brain research</i> 1192, 29-36 (2008). 2 Rich, K. A., Zhan, Y. & Blanks, J. C. Migration and synaptogenesis of cone photoreceptors in the developing mouse retina. <i>Journal of Comparative Neurology</i> 388, 47-63 (1997). 3 Hussey, K. A., Hadyniak, S. E. & Johnston Jr, R. J. Patterning and development of photoreceptors in the human retina. <i>Frontiers in cell and developmental biology</i> 10, 878350 (2022). 4 Michalakakis, S., Geiger, H., Haverkamp, S., Hofmann, F., Gerstner, A. & Biel, M. Impaired opsin targeting and cone photoreceptor migration in the retina of mice lacking the cyclic nucleotide-gated channel CNGA3. <i>Investigative Ophthalmology & Visual Science</i> 46, 1516-1524 (2005). 5 Trifunović, D., Dengler, K., Michalakakis, S., Zrenner, E., Wissinger, B. & Paquet-Durand, F. cGMP-dependent cone photoreceptor degeneration in the cpfl1 mouse retina. <i>Journal of Comparative Neurology</i> 518, 3604-3617 (2010). 6 O'Neal, T. B. & Luther, E. E. Retinitis pigmentosa. <i>StatPearls [Internet]</i> (2020). 7 Brunet, A. A., Harvey, A. R. & Carvalho, L. S. Primary and secondary cone cell death mechanisms in inherited retinal diseases and potential treatment options. <i>International Journal of Molecular Sciences</i> 23, 726 (2022). 8 Leenders, M., Gaastra, M., Jayagopal, A. S. H. & Malone, K. E. Prevalence Estimates and Genetic Diversity for Autosomal Dominant Retinitis Pigmentosa Due to RHO, c.68C>A (p.P23H) Variant. <i>American Journal of Ophthalmology</i> 268, 340-347, doi:https://doi.org/10.1016/j.ajo.2024.08.038 (2024). 9 Sakami, S., Maeda, T., Bereta, G., Okano, K., Golczak, M., Sumaroka, A., Roman, A. J., Cideciyan, A. V., Jacobson, S. G. & Palczewski, K. Probing Mechanisms of Photoreceptor Degeneration in a New Mouse Model of the Common Form of Autosomal Dominant Retinitis Pigmentosa due to P23H Opsin Mutations. <i>Journal of Biological Chemistry</i> 286, 10551-10567 (2011).

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Prof Jeffrey Keelan Email: jeff.keelan@uwa.edu.au
Project title:	Investigating the molecular changes in the retinal environment of mouse models of inherited vision loss
Project location:	Lions Eye Institute, Level 4 Harry Perkins Building
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims:</p> <p>Congenital achromatopsia, or total colour blindness, is a rare autosomal recessive inherited retinal disease affecting 1:30,000 people worldwide¹. It is caused by the primary degeneration of cone photoreceptors which results in the loss of colour discrimination, loss of fine visual acuity, photophobia (light aversion), and nystagmus (involuntary eye movements)¹. Though some patients present with more severe symptoms due to degeneration of other retinal cells²⁻⁴, the mechanisms behind this is currently unclear.</p> <p>In mouse models of achromatopsia, previous research has suggested that only cone photoreceptors are affected⁵⁻⁸, much like the majority of achromatopsia patients. However, using single-cell RNA sequencing, our lab has identified transcriptional changes occurring in other retinal cells, suggesting the rest of the retinal environment is impacted from degenerating cone photoreceptors.</p> <p>The aims of this project include:</p> <ol style="list-style-type: none"> 1. investigating the molecular changes occurring in rod photoreceptors 2. evaluate the changes occurring in second order neurons due to loss of synaptic input from cone photoreceptors, and 3. elucidate how degeneration of cone photoreceptors leads to retinal inflammation. <p>Design:</p> <p>Using three mouse models of achromatopsia, the molecular changes occurring in the retinal environment will be assessed and compared against healthy controls. Retinal samples will be collected from both healthy and disease mice and processed for immunohistochemistry to evaluate whether protein changes corroborate with transcriptional changes identified previously within our lab.</p>

Techniques:

- Animal handling (mouse)
- Eye and retinal dissections
- Tissue processing and immunohistochemistry
- Microscopy
- Extensive record keeping via excel
- Statistical analysis

Outcomes:

- How rod photoreceptors are impacted by cone degeneration will be characterised on a protein level
- How second order neurons are impacted by cone degeneration will be characterised on protein level
- The extent of retinal inflammation will be characterised

References:

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- 2 Thiadens, A. A., Slingerland, N. W., Roosing, S., van Schooneveld, M. J., van Lith-Verhoeven, J. J., van Moll-Ramirez, N., van den Born, L. I., Hoyng, C. B., Cremers, F. P. & Klaver, C. C. Genetic etiology and clinical consequences of complete and incomplete achromatopsia. *Ophthalmology* **116**, 1984-1989.e1981, doi:10.1016/j.ophtha.2009.03.053 (2009).
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- 5 Trifunović, D., Dengler, K., Michalakis, S., Zrenner, E., Wissinger, B. & Paquet-Durand, F. cGMP-dependent cone photoreceptor degeneration in the cpfl1 mouse retina. *Journal of Comparative Neurology* **518**, 3604-3617 (2010).
- 6 Michalakis, S., Geiger, H., Haverkamp, S., Hofmann, F., Gerstner, A. & Biel, M. Impaired opsin targeting and cone photoreceptor migration in the retina of mice lacking the cyclic nucleotide-gated channel CNGA3. *Investigative Ophthalmology & Visual Science* **46**, 1516-1524 (2005).
- 7 Michalakis, S., Mühlfriedel, R., Tanimoto, N., Krishnamoorthy, V., Koch, S., Fischer, M. D., Becirovic, E., Bai, L., Huber, G. & Beck, S. C. Restoration of cone vision in the CNGA3-/- mouse model of congenital complete lack of cone photoreceptor function. *Molecular therapy* **18**, 2057-2063 (2010).
- 8 Biel, M., Seeliger, M., Pfeifer, A., Kohler, K., Gerstner, A., Ludwig, A., Jaissle, G., Fauser, S., Zrenner, E. & Hofmann, F. Selective loss of cone function in mice lacking the cyclic nucleotide-gated channel CNG3. *Proceedings of the National Academy of Sciences* **96**, 7553-7557 (1999).

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Prof Jeffrey Keelan Email: jeff.keelan@uwa.edu.au
Project title:	Identifying pyroptosis as the main culprit for cone degeneration in inherited retinal diseases
Project location:	Lions Eye Institute, Level 4 Harry Perkins Building
Project Description: Aims, Design, Techniques, Outcomes, References	Aims: Inherited retinal disease (IRDs) are a diverse group of visual disorders that affect approximately 1 in 2,000 people worldwide and are the leading cause of blindness in working-age adults ¹ . In Australia alone, it is estimated the economic burden of IRDs is \$781 million to \$1.56 billion ² . Over 280 disease genes have been identified, with majority of mutations affecting rod and cone photoreceptors ³ . Cones are essential for daylight vision, visual acuity, and colour visions, and their degeneration has the greatest impact on patients' quality of life ⁴ . However, the cellular mechanisms underlying cone degeneration are poorly understood, severely impacting the advancement of developing therapies to save cones from dying.
	Pyroptosis is a relatively newer form of cell death that has been identified, with currently limited research linking pyroptosis to photoreceptor degeneration ⁵ . Using mouse models of IRDs, our lab has identified upregulation of pyroptosis markers through transcriptomic sequencing. The proposed project aims to validate that pyroptosis is a key contributor to cone photoreceptor death in IRDs.
	This project aims to: <ol style="list-style-type: none"> 1. Confirm pyroptosis activation during cone degeneration 2. Define the upstream triggers of pyroptosis activation in degenerating cones 3. Determine whether inhibition of pyroptosis attenuates cone degeneration
	Design: Retinal samples will be collected from both healthy and two different IRD mouse lines at specific ages to assess both RNA and protein levels of pyroptosis markers to confirm pyroptosis activation in degenerating cone photoreceptors. After pyroptosis is confirmed, a literature search will be conducted to identify potential links between the IRD mutations within the mice and pyroptosis, and validate this link in the lab through RNA and protein levels to assess whether these differ between healthy and diseased retina.

	<p>The effects of blocking pyroptosis in IRD mice on preserving cone photoreceptors will be evaluated using one of two pyroptosis blocking drugs, either disulfiram or dimethyl fumarate, that will be administered via intravitreal injection into the mice by another lab member. Visual response testing via electroretinograms will then be conducted to evaluate whether pyroptosis inhibition preserves cone-mediated vision. Histological evaluation will also be conducted to assess overall retinal health after drug administration.</p> <p>Techniques:</p> <ul style="list-style-type: none"> • Animal handling (mouse) and assisting with animal surgeries • Visual response testing • Eye and retinal dissections • Tissue processing and immunohistochemistry • Microscopy • RNA extraction and quantification • Extensive record keeping via excel • Statistical analysis <p>Outcomes:</p> <p>The expected outcomes of this project are to:</p> <ul style="list-style-type: none"> • Verify that pyroptosis is a major contributor to cone photoreceptor degeneration in IRD mouse models • Confirm the upstream triggers of pyroptosis activation in cone photoreceptors • Evaluate the efficacy of different pharmaceutical pyroptosis inhibitors on halting cone degeneration in IRD mouse models • The future prospects of this project will be to translate pyroptosis inhibitors that prevent cone photoreceptor degeneration in patients with IRDs
	<p>References:</p> <ol style="list-style-type: none"> 1 Crewe, J. M., Morlet, N., Morgan, W. H., Spilsbury, K., Mukhtar, A. S., Clark, A. & Semmens, J. B. Mortality and hospital morbidity of working-age blind. <i>British Journal of Ophthalmology</i> 97, 1579-1585, doi:10.1136/bjophthalmol-2013-303993 (2013). 2 Schofield, D., Kraindler, J., Tan, O., Shrestha, R. N., West, S., Hart, N., Tan, L., Ma, A., Grigg, J. R. & Jamieson, R. V. The health care and societal costs of inherited retinal diseases in Australia: a microsimulation modelling study. <i>Medical Journal of Australia</i> (2023). 3 The University of Texas Health Science Center. (2024). 4 Crewe, J. M., Morlet, N., Morgan, W. H., Spilsbury, K., Mukhtar, A., Clark, A., Ng, J. Q., Crowley, M. & Semmens, J. B. Quality of life of the most severely vision-impaired. <i>Clinical & experimental ophthalmology</i> 39, 336-343 (2011). 5 Sun, Y., Li, F., Liu, Y., Qiao, D., Yao, X., Liu, G.-S., Li, D., Xiao, C., Wang, T. & Chi, W. Targeting inflammasomes and pyroptosis in retinal diseases—molecular mechanisms and future perspectives. <i>Progress in Retinal and Eye Research</i>, 101263 (2024).

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Project title:	Exploring the effects of radiation on the tumour immune microenvironment
Project location:	Harry Perkins Institute, QEII Medical Centre
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims Cancer immunotherapy using immune checkpoint inhibitors (ICI) has revolutionised the field of oncology in the last 5-10 years. However, whilst ICI drugs can produce remarkable responses, they are still only effective for a minority of people. Recent preclinical work in our lab, focussing on mesothelioma, has shown that certain types of radiotherapy can increase likelihood of successful ICI therapy through functional remodelling of tumour blood vessels – boosting the cure rate from around 20% to almost 100%. We are looking for an honours student to join our team with the aim of broadening our understanding of the mechanisms responsible for these improvements.</p> <p>Design We wish to examine the presence or absence of a range of immune cell types and their activational status, to identify characteristics of tumours that have the best chance of response to this type of therapy – with the aim of carrying out a clinical trial in the near future.</p> <p>Techniques This project will use techniques such as flow cytometry, immunofluorescent tissue staining and RNA sequencing.</p> <p>Outcomes</p>
	<p>References</p> <ol style="list-style-type: none"> 1. Hartley F, Ebert MA, Cook AM. Leveraging radiotherapy to improve immunotherapy outcomes: rationale, progress and research priorities. <i>Clin Transl Immunology</i>. 2025 DOI:10.1002/cti2.70030 2. D'Alonzo RA, Keam S, Gill S, Rowshanfarzad P, Nowak AK, Ebert MA, Cook AM. Fractionated low-dose radiotherapy primes the tumor microenvironment for immunotherapy in a murine mesothelioma model. <i>Cancer Immunol Immunother</i>. 2025 Jan 3;74(2):44. doi: 10.1007/s00262-024-03889-x.

Research Project Proposal 2026

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	Name: Dr Jua Iwasaki Email: jua.iwasaki@thekids.org.au	The Kids Research Institute Australia, 15 Hospital Ave, Nedlands WA 6009
Project title:	Determining the role of a novel RNA chaperone in the virulence of Group A <i>Streptococcus</i>	
Project location:	The Kids Research Institute Australia, 15 Hospital Ave, Nedlands WA 6009	
Project Description: Aims, Design, Techniques, Outcomes, References	<p><i>Streptococcus pyogenes</i> (Group A <i>Streptococcus</i>, GAS) is a global priority pathogen causing a spectrum of illnesses from mild pharyngitis and impetigo to life-threatening invasive disease and lifelong immune complications including acute glomerulonephritis and rheumatic heart disease (1). Although regulation of bacterial gene expression is best understood to occur through modulation of gene transcription, an entire additional layer of gene regulation exists which operates post-transcriptionally. Post-transcriptional regulation of RNA often acts on the expression hundreds of genes simultaneously and is a critical tool used by bacteria to respond to their environment or host. Such regulation commonly involves multiple RNA-binding proteins which modulate mRNA translation or stability through direct protein-RNA interactions or indirectly by facilitating interactions with small regulatory RNAs (sRNAs) (2, 3). Although GAS is known to encode multiple sRNAs with important roles in virulence (4), this organism does not encode homologs for common RNA binding proteins and the role of RNA binding proteins in GAS is unknown. A recent study by our collaborators has identified that a homolog of conserved virulence factor D (CvfD) from <i>S. pneumoniae</i> is present in GAS genomes and is essential for survival under in vivo conditions (5). This project will characterise the CvfD homolog in GAS and determine its potential role in GAS virulence.</p> <p>Aims:</p> <ol style="list-style-type: none"> 1. Construct and verify an inducible <i>cvfD</i> knock-down mutant. 2. Assess the impact of <i>cvfD</i> knock-down on a broad range of clinically-relevant GAS phenotypes including growth, stress response, antimicrobial resistance, and virulence during infection of host cells. 3. Identify transcripts under the regulation of <i>cvfD</i>. 	
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Design:</p> <p>In this study you will generate and characterise a <i>cvfD</i> knock-down strain of GAS using a recently published doxycycline-inducible CRISPR interference system. To test the role of CvfD in clinically relevant GAS phenotypes, you will compare wild-</p>	

	<p>type GAS, vector-only controls, and the uninduced mutant strain to the knock-down in variety of phenotypic assays including:</p> <ol style="list-style-type: none"> 1. Growth curves 2. Antimicrobial susceptibility tests 3. Acid stress, oxidative stress, or metal stress assays. 4. Human tonsil cell infection assays 4. Based on the observed phenotypes, you will then test the impact of CvdF knockdown on expression of virulence/antimicrobial resistance/stress response genes by designing RT-qPCR assays and testing the change in transcript levels for each target gene.
	<p>Techniques: bacterial culture; molecular cloning; bacterial mutagenesis; gene knock-down using CRISPR interference; maintenance of human cell-lines; primer design; RT-qPCR; antimicrobial susceptibility testing.</p>
	<p>Outcomes: In this project you conduct the first investigation into the role of RNA chaperone proteins in GAS virulence. By characterising a mutant GAS strain lacking CvdF, you will determine the role of this protein in regulating colonisation of human cells, antimicrobial resistance, stress response, and bacterial growth. You will also determine the role of this regulator in virulence by measuring its impact on the expression of important virulence factors known to play a role in GAS disease. This project will lay the foundation for larger RNAseq, RNA-RNA interaction, and RNA-protein interaction studies to help determine the role of CvdF and other proteins in coordinating gene expression in GAS to better understand and control this important pathogen.</p>
	<p>References/further reading:</p> <ol style="list-style-type: none"> 1. Brouwer S, Rivera-Hernandez T, Curren BF, Harbison-Price N, De Oliveira DMP, Jespersen MG, et al. Pathogenesis, epidemiology and control of Group A <i>Streptococcus</i> infection. <i>Nature Reviews Microbiology</i>. 2023;21(7):431-47. 2. Amemiya HM, Schroeder J, Freddolino PL. Nucleoid-associated proteins shape chromatin structure and transcriptional regulation across the bacterial kingdom. <i>Transcription</i>. 2021;12(4):182-218. 3. Hołowka J, Zakrzewska-Czerwińska J. Nucleoid Associated Proteins: The Small Organizers That Help to Cope With Stress. <i>Frontiers in Microbiology</i>. 2020;11. 4. Xiong Z-Q, Lv Z-X, Song X, Liu X-X, Xia Y-J, Ai L-Z. Recent Research Advances in Small Regulatory RNAs in <i>Streptococcus</i>. <i>Current Microbiology</i>. 2021;78(6):2231-41. 5. Jespersen MG, Hayes AJ, Tong SYC, Davies MR. Pangenome evaluation of gene essentiality in <i>Streptococcus pyogenes</i>. <i>Microbiology Spectrum</i>. 2024;0(0):e03240-23.

Research Project Proposal 2026

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Project title:	Leukaemia monitoring using circulating DNA fragments
Project location:	Translational Cancer Pathology Laboratory, UWA, M block, QEII Medical Centre
Project Description: Aims, Design, Techniques, Outcomes, References	Aims: Leukaemia is a blood cancer characterised by the production of abnormal white blood cells in the bone marrow. It can arise spontaneously without any obvious risk factors or occur secondary to a pre-existing chronic bone marrow disorder. More than 50,000 Australians are in the latter group, living in the knowledge that they are at risk of developing leukaemia. An example of this is patients with myeloproliferative neoplasms, who have up to 20% risk of developing leukaemia. Current tests are unable to predict when this may occur. This project aims to address this problem with a highly sensitive next sequencing approach to detect leukaemic mutations in circulating DNA fragments (cell-free DNA) isolated from blood.
	Design: In this project you will perform genetic sequencing of circulating fragments of DNA ("cell- free DNA") in the blood of patients with pre-leukaemic conditions. This will be used to determine whether changes occur in the cell-free DNA that may provide an indication of the genetic switch to leukaemia.
	Techniques: You will learn techniques such as ethical considerations of working with human samples, handling patient samples, blood cell isolation, cell-free DNA and genomic (cellular) DNA extraction, next generation sequencing, and bioinformatics.
	Outcomes: Patients want to know if and when the pre-leukaemic condition with which they live is progressing to leukaemia. The approach to be used in this study will generate data that may assist in informing patients about their risk, and doctors about whether to commence leukaemia treatment.
	<p>References: Chuah H. Genomic Profiling and Monitoring in Acute Myeloid Leukaemia using Plasma cell-free DNA. 2022. doi: 10.26182/j36b-f552.</p> <p>Hupe HC, Wienecke CP, Bartels S et al. Cell-free DNA for detection and monitoring of extramedullary AML relapse. Hemasphere. 2025;9(3):e70097.</p> <p>Thakral D, Gupta R, Sahoo RK et al. Real-Time Molecular Monitoring in Acute Myeloid Leukemia With Circulating Tumor DNA. Front Cell Dev Biol. 2020;8:604391.</p>

Research Project Proposal 2026

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	Name: Prof Wendy Erber Email: wendy.erber@uwa.edu.au
Project title:	Using platelet genetics to monitor blood cancers
Project location:	Translational Cancer Pathology Laboratory, UWA, M block, QEII Medical Centre
Project Description: Aims, Design, Techniques, Outcomes, References	Aims: Myeloproliferative neoplasms are a group of blood cancers where patients have abnormal blood cell production. Up to 20% of these patients are at increased risk of developing acute myeloid leukaemia, which is associated with poor prognosis and a reduced life expectancy. Current tests are unable to predict when this may occur. Leading work from our group have found that platelets in patients with myeloproliferative neoplasms have altered gene expression and we have developed a novel blood sequencing method to detect this. The aim of this project is to determine whether there are additional changes that can be detected when, or before, patients progress to leukaemia.
	Design: In this project, you will learn to handle patient samples and perform genetic sequencing of platelets in patients with myeloproliferative neoplasms. This will be used to determine whether there are additional changes that occur in platelets that may provide an indication of the genetic switch to leukaemia.
	Techniques You will learn techniques such as ethical considerations of working with human samples, handling patient samples, blood cell isolation, RNA extraction, next generation sequencing, and bioinformatics.
	Outcomes: Through this project, you will generate data that will improve our understanding of genetic changes that occur in platelets and how this relates to changes in disease status. This could assist patients and their doctors by providing information about their risk, and whether to commence treatment for leukaemia.
	References: Guo BB et al. Platelets in myeloproliferative neoplasms have a distinct transcript signature in the presence of marrow fibrosis. Br J Haematol. 2020;188(2):272-82. Collinson RJ et al. PlateletSeq: A novel method for discovery of blood-based biomarkers. Methods. 2023;219:139-49. Collinson RJ et al. Transcription factor 3 is dysregulated in megakaryocytes in myelofibrosis. Platelets. 2024;35(1):2304173.

Research Project Proposal 2026

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	Name: Dr Hannah Newnes Email: hannah.newnes@uwa.edu.au
Project title:	From Donor to Design: Empowering NK Cells for Enhanced Cancer Killing
Project location:	M Block
Project Description: Aims, Design, Techniques, Outcomes, References	Background: <p>Natural killer (NK) cells are a type of immune cell that has naturally evolved to eliminate cancer. However not all NK cells are equal in their capacity to eliminate cancer and cancer cells have developed various mechanisms to evade NK cell elimination. Our group is interested in finding the optimal ways we can boost NK cells responses against cancer and use this information to develop novel therapies to improve treatment responses.</p> <p>We utilise a number of different approaches to optimally boost NK cell activity against cancer. This includes the use of novel cytokine combinations to prime NK cells, antibody molecules to enhance NK cell recognition of cancer cells, and modulation of NK cell effector function through genetic engineering and small molecular inhibitors.</p>
	Design: <p>This project will involve optimising different strategies to improve NK cell responses against a panel of patient derived leukaemia and neuroblastoma cell lines. The specific approach that we will utilise in this honours project will be determine following discussion with interested students.</p>
	Techniques: <p>This project will use a number of techniques such as flow cytometry, cell culture, ELISA, and genetic engineering</p>
	Outcomes: <p>Our goal is to design and develop the optimal NK cell-based immunotherapy that provides patients with not only a highly effective treatment but a also a treatment that is less toxic than standard-of-care treatments ensuring patients have significantly improved quality of life outcomes including reducing future admissions to hospital during their lifetime.</p>

Research Project Proposal 2026

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	Name: Dr Hannah Newnes Email: hannah.newnes@uwa.edu.au	Name: Samantha Barnes Email: samantha.barnes@uwa.edu.au
Project title:	Impact of Early-Life Cancer Therapy on Long-Term Immune Competence	
Project location:	M Block and The Kids Research Institute Australia	
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Background: Over the past fifty years, tremendous progress has been made in curing childhood cancers, with over 80% of young patients now expected to survive into adulthood. However, the therapies responsible for this survival, such as chemotherapy and radiotherapy, can also lead to adverse, long-term health issues, known as late effects, which appear months to years after completion of treatment. Childhood cancer survivors are more likely to be hospitalised for infection >5 years following completion of treatment and have an increased risk of chronic health conditions such as osteoporosis, infertility, cardiovascular disease and obesity well into adulthood.</p> <p>Recent evidence suggests that childhood cancer survivors display characteristics of an aged immune system referred to as “inflamm-aging”. This chronic low-grade inflammation is thought to be linked with a higher risk of secondary diseases in children and teens for years following treatment. However, the mechanisms responsible for long-term chronic immune disturbances in cancer survivors and their potential consequences on survivors’ health remain unknown.</p>	
	<p>Design: Using age-appropriate preclinical models we will directly assess the impact of commonly used chemotherapy protocols on the immune system. Following treatment mice will be left to recover and following this we will perform comprehensive immunophenotyping of the major immune cell subsets across different tissues and determine the ability of the immune system to respond to various insults.</p>	
	<p>Techniques: This project will use a number of different techniques involving flow cytometry, cell culture, ELISA, and animal handling.</p>	
	<p>Outcomes: This project will develop for the first time an animal model of cancer survivorship where we can measure the impact of cancer treatment on the immune system. This will provide us with a blueprint for reversing these responses and significantly improving the quality of life for childhood cancer survivors.</p>	

Research Project Proposal 2026

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	Name: Email:
SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Associate Professor Demelza Ireland Email: demelza.ireland@uwa.edu.au
Project title:	Determinants of infant feeding outcomes in allergic families.
Project location:	The Kids Research Institute Australia (located within the Perth Children's Hospital)
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims: To determine maternal and family characteristics associated with infant feeding outcomes within an Australian cohort of families with allergic diseases.</p> <p>Design: Prospective infant feeding data has been collected on more than 2000 infants from birth to 12-months of age. The infants were participating in the PrEggNut Study¹ and located in Western Australia, South Australia, Victoria and New South Wales. All infants were at-risk of developing allergies due to at least two family members with a history of allergic disease. Maternal and family characteristics data such as maternal age, body mass index and education, ethnicity, parity, mode of birth, gestational age at birth, history of allergic diseases and level of socio-economic advantage/disadvantage were collected.</p> <p>Techniques Literature searching Large-scale data collation from a REDCap database Statistical analysis Scientific writing</p> <p>Outcomes</p> <ul style="list-style-type: none"> • Maternal and family characteristics associated with infant feeding outcomes: • Duration of exclusive breastfeeding • Duration of any breastfeeding • Age of introduction to infant formula • Age of introduction to solid foods • Age of introduction to allergenic foods, such as egg and peanut

	<p>References</p> <ol style="list-style-type: none">1) DJ Palmer, TR Sullivan, DE Campbell, et al. PrEggNut Study: protocol for a randomised controlled trial investigating the effect of a maternal diet rich in eggs and peanuts from <23 weeks' gestation during pregnancy to four months' lactation on infant IgE-mediated egg and peanut allergy outcomes. BMJ Open 2022 Jun 13;12(6):e056925. doi: 10.1136/bmjopen-2021-056925
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Research Project Proposal 2026

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	Name: Email:
SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: To be confirmed Email:
Project title:	Development of Usher syndrome 1D patient-derived inner ear organoid model
Project location:	Ear Science Institute Australia, Floor 3, Ralph and Patricia Sarich Neuroscience Research Institute, 8 Verdun Street, Nedlands, WA 6009
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims Hearing loss is a major public health problem affecting one in six people. In children, problems with hearing lead to delayed language development or speech impairment. In adults, loss of hearing and communication can lead to feelings of isolation and depression. Mainstay treatments include the use of hearing aids or cochlear implants, but these may be inadequate if hearing loss is profound or caused by extensive cochlear hair cell or neuron loss. These specialized cells, once lost or damaged, cannot regenerate and no treatment is available to cure hair cells or neuron loss in the inner ears.</p> <p>Design Our approach is to generate new sensory hair cells from human induced pluripotent stem cells (iPSC). Replacement of cochlear hair cells is a promising approach to reverse sensorineural deafness. This project will hold great potential for treatment of hearing loss to improve the quality of life for the hearing-impaired population.</p> <p>Techniques Demand for ototoxicity testing may be better satisfied with appropriate human organoid models, such as those recently generated from iPSC. Robust clinically-relevant protocols for human hair cell differentiation are still in development but there have been several advances in recent years towards safe and effective xeno-free methods. These largely recapitulate the regulation of signalling pathways (TGF, BMP, FGF, and Wnt) during development. In this project, we will identify the approaches to generate cochlear organoids with functional hair cells in implementing these models from normal and patient-specific iPSC and test for ototoxicity.</p>

	<p>Outcomes</p> <p>The differentiation of induced pluripotent stem cells to cell types of the inner ear is a new area, promising for regenerative studies, but still not developed enough for scalable, reliable and safe treatments. Another valuable contribution of pluripotent cells is in the generation of testing platforms for delivery, efficacy and safety of treatments. Many devices, materials and drug treatments have been compromised at late stages of development because of their poor targeting or ototoxic effects. Materials and drug testing are now performed mostly in animal models but are increasingly limited by ethical constraints and may be prone to error due to species differences.</p>
	<p>References</p> <p>2025 Leith, K.F., Jye, J., Delaney, D.S., McLenachan, S., Chen, F.K., Atlas, M.D., Wong, E.Y.* Current progress of stem-cell based modelling for Usher syndrome. <i>Frontiers in Cell and Developmental Biology</i> under review. Research topic: Studying Rare Diseases using induced pluripotent stem cell (iPSC)-based model systems. (Impact factor: 4.6)</p> <p>2024 Wong, E.Y., Khoh,, X..E., Lye, J., Leith, F., Zhang, D., Chen, S., Lamey, T., Thompson, J.A., McLaren, T., De Roach, J. N., Carvalho, Atlas, M.D., Chen, F.K., McLenachan, S. Generation of an induced pluripotent stem cell line from a patient with Usher syndrome type 1B caused by compound homozygous mutations in <i>MYO7A</i>. <i>Stem Cell Research</i>, 79, 103492. https://doi: 10.1016/j.scr.2024.103492</p>

Research Project Proposal 2026

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Project title:	Modelling a human microvessel network
Project location:	CCREM laboratories, level 6 RPH Research Foundation Building, Rear 50 Murray St, Perth.
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Sepsis is defined as life-threatening organ dysfunction due to a dysregulated host response to infection(1). While sepsis impacts all age groups, it disproportionately affects the elderly, very young and indigenous populations, and frequently results in life-changing long-term disability, including the potential loss of limbs, or death(2, 3). Septic shock is a severe form of sepsis, causing alterations in blood flow and oxygen delivery to tissues. It particularly affects microvessels in the body(4). The resulting organ failure is fatal in 20% of people with septic shock. Microvascular dysfunction is a feature of many critical illnesses(5), but is central in the progression of infection in sepsis(4).</p> <p>Our lab is developing a model of human microvascular dysfunction to be used to assess the efficacy of current sepsis treatments, and to test novel therapies designed to mitigate microvascular dysfunction and organ failure.</p> <p>Aims:</p> <ol style="list-style-type: none"> 1. To assess cultured human microvascular networks to determine the effect of perfusion on physiological representation and maturation status. 2. To optimise culture stability by evaluating approaches to inhibit type I collagen contraction.
	<p>Design:</p> <p>Human adipose-derived microvascular fragments will be cultured in type I collagen hydrogels in static and perfusion cultures. Immunofluorescence will be performed on fixed cultures to determine the cellular composition of the microvessels following different culture times/conditions. Samples will also be collected for RNA extraction and gene expression analysis to fully define vascular maturation status, and to determine the optimal culture conditions for evaluating sepsis-relevant endpoints in the model.</p> <p>Strategies to prevent collagen contraction and extend microvessel culture duration will also be defined and tested by applying these approaches to human microvessel cultures and assessing cellular composition and culture duration.</p>
	<p>Techniques:</p> <p>Immunofluorescence and confocal microscopy Image analysis 3D tissue culture RNA extraction and comparative q-PCR analysis.</p>

	<p>Outcomes:</p> <p>Comparative image and gene-expression analysis of cultured human microvessel cultures to determine the influence of perfusion (different flow rates), hydrogel composition and stability, and culture time on cellular composition and vascular maturation status. This data will identify the necessary culture conditions to generate a physiologically relevant and mature network to facilitate future investigations of microvascular dysfunction.</p>
	<p>References</p> <ol style="list-style-type: none"> 1. Cavaillon JM, <i>et al.</i> EMBO Mol Med. 2020;12(4):e10128. 2. Arise, Committee AAM. Crit Care Resusc. 2007;9(1):8-18. 3. Yende S, <i>et al.</i> Crit Care Med. 2016;44(8):1461-7. 4. Boisrame-Helms J, <i>et al.</i> Curr Vasc Pharmacol. 2013;11(2):150-60. 5. Austin SA, <i>et al.</i> Circ Res. 2010;107(12):1498-502.

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBC Email:	
Project title:	Non-invasive Treatment of Acute Otitis Media Using Thermosensitive Silk Hydrogels	
Project location:	Ear Science Institute Australia, Level 3, 8 Verdun Street, Nedlands 6009	
Project Description: Aims, Design, Techniques, Outcomes, References	<p>This project explores a novel, non-invasive approach to treating acute otitis media (AOM) by using thermosensitive silk hydrogels as a transtympanic drug delivery system. The hydrogel is designed to be applied directly to the tympanic membrane as a liquid, where it undergoes in situ gelation at body temperature, forming a biocompatible depot for sustained, localized drug release into the middle ear. This strategy avoids the need for systemic antibiotics or surgical intervention (e.g., tympanostomy tubes), potentially reducing antimicrobial resistance, minimizing side effects, and improving patient compliance, especially in pediatric populations. By combining the unique mechanical, tunable, and biodegradable properties of silk with the responsiveness of thermogels, this platform could provide a safe, effective, and child-friendly alternative for managing one of the most common pediatric infections worldwide.</p> <p>Aims</p> <ul style="list-style-type: none"> • Understand how silk hydrogels can be tuned to transition from liquid to a gel in physiological temperature (What parameters can we optimise?) • Determine the effect of incorporating drugs on the phase transition rate of thermosensitive silk hydrogels 	
	<p>Design</p> <p>Milestones include fast transition of silk hydrogels from liquid to solid upon exposure to physiological temperature with or without the drugs (typically this process can last for a month without optimisation)</p>	

	<p>Techniques: Silk materials manufacturing Freeze Drying Additive manufacturing FTIR Mechanical testing Accelerated degradation assay Cell biology and molecular assay SDS page</p> <p>The honours' student will:</p> <ul style="list-style-type: none"> • Optimise various parameters such as silk concentration, pH, addition of salt and evaluate the effects on the transition rate of silk hydrogels from liquid to solid upon exposure to physiological temperature • Evaluate the effects of adding one or two drugs and optimise the drug concentration to maintain fast transition rate from liquid to gel upon exposure to physiological temperature <p>Outcomes The honours' student will be able to understand what are the factors that influence the transition rate of silk hydrogel from liquid to solid and would be important in evaluating treatment options for noninvasive, transtympanic drug delivery towards the middle ear.</p>
	<p>References Yang, R., Sabharwal, V., Shlykova, N., Kim, S., Juhn, S. K., & Li, J. D. (2016). Treatment of otitis media by transtympanic delivery of antibiotics. <i>Science Translational Medicine</i>, 8(356), 356ra120. https://doi.org/10.1126/scitranslmed.aaf4363</p> <p>Zhou, C., Chen, J., Yang, J., Fu, Y., Yin, D., Fang, T., ... & Cai, L. (2024). Conformal immunomodulatory hydrogels for the treatment of otitis media. <i>Journal of Nanobiotechnology</i>, 22(1), 295. https://doi.org/10.1186/s12951-024-02908-4</p> <p>Wise, A. K., Gillespie, L. N., Shepherd, R. K., & Richardson, R. T. (2016). Cell and drug delivery for inner ear disease using injectable silk–PEG hydrogel. <i>Biomaterials</i>, 94, 9–18. https://doi.org/10.1016/j.biomaterials.2016.03.043</p>

Research Project Proposal 2026

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Project title:	Characterising tumour-infiltrating lymphocytes to inform personalised cancer therapy
Project location:	M-block, SBMS
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims: Tumour-infiltrating lymphocytes (TILs) are critical mediators of anti-tumour immunity and form the basis of adoptive cell therapies for melanoma and other cancers. However, their functional heterogeneity and limited persistence after expansion remain major barriers to effective personalised therapies. This project aims to investigate the biology of TILs using a multi-dimensional approach to identify features that correlate with anti-tumour function.</p> <p>Design:</p> <ul style="list-style-type: none"> • Characterise the metabolic profile of TILs to determine how energy utilisation patterns relate to cytotoxic function. • Analyse expression of NK-like markers and other immune phenotypes to identify signatures associated with reduced function or exhaustion. • Correlate metabolic and phenotypic profiles with functional readouts, including cytokine production and cytotoxicity assays. • Generate insights to inform strategies for optimising personalised TIL therapies. <p>Techniques:</p> <ul style="list-style-type: none"> • Cell culture of primary human melanoma samples. • Flow cytometry for surface and intracellular marker profiling. • Metabolite staining to assess glycolytic and oxidative activity. • Functional assays including target cell killing and ELISAs for IFN-γ production <p>Outcomes:: This project will provide a detailed understanding of TIL heterogeneity, linking metabolic and phenotypic traits to functional capacity. The findings will support the development of next-generation, personalised TIL therapies, improving patient outcomes in adoptive cell immunotherapy.</p>
	<p>References</p> <p>Rohaan et al. (2022). <i>Tumor-infiltrating lymphocyte therapy or Ipilimumab in Advanced Melanoma</i>. https://www.nejm.org/doi/full/10.1056/NEJMoa2210233</p> <p>Tarazona et al. (2001). <i>Increased expression of NK cell markers on T lymphocytes in aging and chronic activation of the immune system reflects the accumulation of effector/senescent T cells</i>. https://doi.org/10.1016/S0047-6374(00)00199-8.</p> <p>Good et al. (2021). <i>An NK-like CAR T cell transition in CAR T cell dysfunction</i>. https://doi.org/10.1016/j.cell.2021.11.016</p>

Research Project Proposal 2026

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	Name: Prof Wendy Erber Email: wendy.erber@uwa.edu.au ;	6457 3165 M Block, Queen Elizabeth II Medical Centre
Project title:	Precision diagnosis for the care of patients with blood cancers	
Project location:	Translational Cancer Pathology Laboratory, UWA, M block, Queen Elizabeth II Medical Centre. Nedlands 6009	
Project Description: Aims, Design, Techniques, Outcomes, References	Aims <p>Blood cancers are the third most common cancer and the second highest cause of cancer-related death in Australia. The incidence is increasing, and Australia is concerned about the number of cases and deaths. There are many types of blood cancer and these are characterised by different clinical behaviour, genetics and response to treatments. Detection of genetic abnormalities is critical to ensure the diagnosis is accurate, the risk to the patient accurately predicted and optimal treatment offered. Current genetic methods used in pathology focus on chromosomes with up to 200 cells being analysed. This may be insufficient to identify subsets of cells with prognostically-important genetic changes.</p> <p>Our team has invented a new type of flow cytometry test that is fast and automated and can be used to analyse thousands of blood cancer cells. This method can focus on the cells of interest (the cancer cells) by quantifying proteins to determine the cell identity (immunophenotype) and, simultaneously, detect chromosomes within the cells. This method, called “immuno-flowFISH”, is exquisitely sensitive in detecting critical genetic abnormalities in blood cancers. We have studied acute leukaemia, chronic leukaemias and multiple myeloma, and in each, been able to identify the cancers cells in blood and bone marrow with key genetic changes that will impact patient care.</p>	
	Design <p>In this project you will apply immuno-flowFISH technology to identify high risk cytogenetic chromosomal changes, such as del(17p) or loss of the TP53 gene by immuno-flowFISH in leukaemia. In this project, you will use research methods pioneered in our Translational Cancer Pathology Laboratory at UWA. This includes handling patient samples, immuno-flowFISH processing, imaging flow cytometry and data analysis.</p>	
	Techniques <ul style="list-style-type: none"> • Mononuclear cell gradient separation • Cytogentrifugation • Cell staining • Morphological assessment of leukaemia by light microscopy • Immunophenotyping • Fluorescence in situ hybridisation • Imaging flow cytometry • Immuno-flowFISH • High-end data analysis using a range of software tools 	

	Outcomes <ul style="list-style-type: none"> • You will have learned the skills of immuno-flowFISH, a UWA-invented technology. • You will have learned about del(17p) in blood cancers and its importance in risk stratification and prognosis • You will determine the value of this technique for rare event detection
	References <ol style="list-style-type: none"> 1. Hui H et al. Methods. 2018;1(134-135):32-40. 2. Hui H et al. Methods in Molecular Biology. 2023. 2635; 149-171. 3. Erber WN et al Journal of Human Genetics. 2023;68(7):515-516 4. Mincherton et al. IJLH 2024; 2024;46:495-502

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Prof. Jeffery Keelan Jeff.keelan@uwa.edu.au
Project title:	Characterising genetic variance associated with early-onset high myopia in zebrafish.
Project location:	Lions Eye Institute
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Background: Myopia, or near-sightedness has rapidly become one of the world's leading causes of distant visual impairment, where prevalence rates are as high as 97% in some countries. Misalignments between axial length and focusing power (refractive power) of the lens and corneal steepness leads to a shift in the focal plane and convergence in front of the neural retina, rather than on. In untreated, high myopia (>-5.00 diopters) can lead to severe visual disorders such as retinal detachment, retinal atrophy, myopic maculopathy, glaucoma, and cataracts. A complex interaction between genetic variance (>400 genetic loci) and environmental risk factors such as near-work (reading, screen time and schoolwork) and lack of time spent outdoors contribute to the progression to myopia, yet the fundamental causal mechanisms remain unknown. Early-onset high myopia, developing before the age of 10 yo, has a causation linked almost entirely to genetic variance and as such carries potential for novel treatment strategies targeting specific genes or conserved signaling pathways.</p> <p>Aims: This project aims to characterise the function and conserved roles of genes selected from large human genome-wide association studies (GWAS) through experimentally modelling early-onset high myopia (eoHM) in zebrafish using reverse genetics.</p>

	<p>Design: Morpholino anti-sense oligonucleotide have been designed to target and sterically bind mRNA of genes of interest for the transient knockdown of gene function by either translation blocking or splice modification in developing zebrafish embryos. This approach is well-characterised in our lab and provides a robust, and rapid myopia-associated gene screening platform. By utilising the fast, <i>ex vivo</i> development of the zebrafish embryo and larvae, genes of interest were chosen from large human GWAS and identified in our group as contributing to increased axial length or dysfunctional refractive power. A range of live, non-invasive techniques, such as optical coherence tomography, confocal time-lapse imaging and optokinetic response testing will be employed to study both the structure and function in response to lack of function. Molecular analyses will determine differential gene expression of associated genes.</p>
	<p>Techniques: Single cell microinjection of morpholino oligomers (MO) in zebrafish embryos; live imaging microscopy (fluorescence microscopy, optical coherence tomography (OCT) and confocal imaging); zebrafish handling and husbandry; live optokinetic response and colour vision testing; RNA extraction; cDNA generation; RT-PCR; qPCR; gel electrophoresis; and gene sequencing will be used in this study.</p> <div data-bbox="510 918 1420 1411"> <p style="text-align: center;">Experimental myopia platform</p> <p>The diagram illustrates the experimental myopia platform through five sequential steps, each with a corresponding icon and description:</p> <ol style="list-style-type: none"> 1. Transient knockdown of candidate gene in zebrafish embryos (Day 1): Represented by an icon of a zebrafish embryo and a pipette. 2. RT-PCR positive selection (Day 4): Represented by an icon of a microplate reader. 3. Live optical imaging (Day 5-6): Represented by an icon of an OCT machine. 4. Live electrophysiology (Day 6-7): Represented by an icon of an electrophysiology setup. 5. Gene expression (Day 8-9): Represented by an icon of a computer monitor showing a gel image. </div>
	<p>Outcomes: The potential significance emerging from the outcomes of this project are anticipated to contribute considerably to the framework of understanding the biological pathways and links between genetic and environmental factors in the development of myopia and in particular early-onset high myopia. Identifying which GWAS selected genes are responsible for a myopic phenotype forms an initial screening for further testing, including generation of stable knockout lines of zebrafish using CRISPR and pharmaceutical testing.</p>

	<p>References</p> <p>Holden, Brien A et al. "Global Prevalence of Myopia and High Myopia and Temporal Trends from 2000 through 2050." <i>Ophthalmology</i> vol. 123,5 (2016): 1036-42. doi:10.1016/j.ophtha.2016.01.006</p> <p>Šenk, Urh et al. "GeneBc background of high myopia in children." <i>PloS one</i> vol. 19,11e0313121. 4 Nov. 2024, doi:10.1371/journal.pone.0313121</p> <p>Hong, Y., & Luo, Y. (2021). Zebrafish Model in Ophthalmology to Study Disease Mechanism and Drug Discovery. <i>Pharmaceu6cals (Basel, Switzerland)</i>, 14(8), 716. https://doi.org/10.3390/ph14080716</p>
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Research Project Proposal 2026

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	Name: Email:
Project title:	Disrupting Copper Homeostasis to Enhance Immunotherapy and Chemotherapy in Mesothelioma
Project location:	Harry Perkins Institute of Medical Research (North) Level 5, 6 Verdun St, Nedlands WA 6009
Project Description: Aims, Design, Techniques, Outcomes, References	Aims <ul style="list-style-type: none"> • Mesothelioma is a rare, aggressive cancer with few effective treatments and poor survival. • Most patients do not respond to current immunotherapies. • Copper builds up in mesothelioma tumours, promoting growth, immune evasion, and chemotherapy resistance. • Copper-binding drugs (chelators) are already approved for other diseases and could be repurposed for cancer. • This project will test whether reducing copper levels: <ul style="list-style-type: none"> ○ Changes tumour behaviour and gene expression ○ Improves immune cell activity and tumour killing ○ Enhances response to chemo- and immunotherapy <p>Goal: Improve mesothelioma treatment by targeting copper.</p>
	Design Quantitative
	Techniques <ul style="list-style-type: none"> • Investigate copper levels alters tumour cell behaviour and gene expression, including chemotherapy transporters, MHC-I, and PD-L1. • Examine how copper affects tumour cell sensitivity to chemotherapy. • Assess the impact of copper on immune cell function and tumour visibility using in vitro T cell–tumour cell killing assays. • Characterise how copper chelation therapy modulates immune cell composition and activity within the tumour microenvironment. • Evaluate the in vivo effectiveness of copper chelation therapy, alone or in combination with chemotherapy and/or immunotherapy, and its impact on the tumour microenvironment.

	<ul style="list-style-type: none"> • Use CRISPR-Cas9 to knock out key copper transporter genes in tumour and immune cells to dissect their role in treatment response. • Analyse publicly available datasets to investigate expression of copper-regulatory genes in cancer patients and correlate these with clinical outcomes and treatment responses.
	<p>Outcomes</p> <p>Chance to travel to Sydney to work with UNSW collaborators.</p>

Research Project Proposal 2026

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	Name: Email:
Project title:	Targeting Lipid Metabolism to Improve Immunotherapy in Lung Cancer and Mesothelioma
Project location:	Harry Perkins Institute of Medical Research (North) Level 5, 6 Verdun St, Nedlands WA 6009
Project Description: Aims, Design, Techniques, Outcomes, References	Aims <ul style="list-style-type: none"> Immunotherapy, especially immune checkpoint blockade (ICB), has improved outcomes for advanced lung cancer and mesothelioma. However, up to 70% of patients do not respond, highlighting the need for more effective approaches. A key barrier is the presence of regulatory T cells (Tregs) in the tumour, which suppress anti-cancer immune responses. Tregs depend on cholesterol and lipid metabolism to function within tumours. Clinical data suggest that cholesterol-lowering drugs may improve ICB response rates. This project will investigate whether targeting lipid metabolism can reduce Treg activity and boost anti-tumour immunity.
	Design Quantitative
	Techniques <ul style="list-style-type: none"> Investigate how cholesterol-lowering drugs affect Treg survival, function, and gene expression. Assess how changes in lipid metabolism alter Treg suppression of anti-tumour T cells. Use co-culture assays to test how modifying Treg metabolism impacts immune cell killing of cancer cells. Apply CRISPR-Cas9 to disrupt key metabolic genes in Tregs and explore effects on immune suppression. Evaluate how cholesterol-lowering treatments such as statins influence Treg populations in the tumour microenvironment.

	<ul style="list-style-type: none"> • Test whether combining metabolic therapies with ICB improves tumour control in mouse models of lung cancer and mesothelioma. • Analyse patient data to explore links between cholesterol metabolism genes, Treg signatures, and immunotherapy outcomes.
	<p>Outcomes</p> <ul style="list-style-type: none"> • Work at the intersection of immunology, metabolism, and cancer therapy. • Gain experience in cutting-edge techniques including CRISPR, flow cytometry, and in vivo tumour models, lipidomics and genomics. • Contribute to translational research with real clinical relevance. • Opportunity to collaborate with national experts in tumour immunology and lipid metabolism.

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Email:
Project title:	Identifying eBMD-related proteins and the association with incidence dementia's risk
Project location:	Perron Institute (8 Verdun St, Nedlands WA 6009)
Project Description: Aims, Design, Techniques, Outcomes, References	Aims <ul style="list-style-type: none"> • To identify proteins associated with eBMD using proteomics data from UK Biobank. • To investigate the association between eBMD-proteins and the risk of incident dementia. • To develop a proteomic risk score based on eBMD-proteins for predicting incident dementia.
	Design <p>Osteoporosis and dementia are two common disorders that predominantly affect ageing adults¹. Recent clinical studies report that individuals with osteoporosis have a higher risk of developing dementia later in life^{1,2}. Estimated bone mineral density (eBMD) is a measure derived from quantitative ultrasound (QUS) and serves as a surrogate marker for bone strength. Interestingly, our recent study involving 131,000 elders demonstrated that lower estimated bone mineral density (eBMD) was associated with increased risk of dementia³.</p> <p>Recent advances in proteomics allow for large-scale profiling of plasma proteins⁴, providing a unique opportunity to identify biomarkers and biological pathways associated with bone health. Investigating the relationship between plasma proteins and eBMD can provide insights into the association between eBMD and incident dementia, paving the way for novel therapeutic targets and risk prediction models for fracture prevention.</p> <p>In the study, we will employ the proteomics data from a cohort of UK Biobank to identify the eBMD-related proteins and determine the association between these proteins with incident dementia's risk.</p>

	<p>Techniques</p> <p>R studio will be used to conduct proteomic analysis (association, enrichment, protein-protein interaction), survival analysis (Cox hazard ratio regression) and discrimination analysis (ROC curve).</p>
	<p>Outcomes</p> <ul style="list-style-type: none"> • Proteins associated with eBMD will be identified and profiled. • Association between eBMD-proteins with risk of incident dementia will be evaluated. • Potential of a proteomic risk score for predicting incident dementia will be assessed. • The student is expected to develop skills in conducting proteomic analysis and survival analysis using R.
	<p>References</p> <ol style="list-style-type: none"> 1. Xiao, T., et al. Association of Bone Mineral Density and Dementia: The Rotterdam Study. <i>Neurology</i> 100, e2125-e2133 (2023). 2. Zhang, X., et al. Low Bone Mineral Density With Risk of Dementia: A Prospective Cohort Study. <i>J Am Med Dir Assoc</i> 23, 1719 e1719-1719 e1719 (2022). 3. Lu J, Mastaglia F, Tai ACP, et al. Association of heel bone mineral density with incident dementia among ageing adults: a population-based study from the UK Biobank. <i>Aging Clin Exp Res</i>. 2025;37(1):217. 4. Oh HS, Rutledge J, Nachun D, et al. Organ aging signatures in the plasma proteome track health and disease. <i>Nature</i>. 2023;624(7990):164-172. doi:10.1038/s41586-023-06802-1

Research Project Proposal 2026

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Project title:	Spatially measuring single-cell metabolism in the bone marrow
Project location:	M Block, QEIIIMC
Project Description: Aims, Design, Techniques, Outcomes, References	Aims <p>The bone marrow is a vital organ that serves as the primary site of blood cell production and a central hub for immune regulation, skeletal remodelling, and systemic metabolism. This project aims to: i) Map the spatial distribution of key metabolic activities in the bone marrow at single-cell resolution; ii) Compare metabolic profiles of different marrow-resident cell populations (e.g., osteoblasts, adipocytes, and macrophages); iii) Establish a workflow integrating advanced imaging and isotope tracing for in situ metabolic analysis.</p>
	Design <p>The project involves a combination of <i>in vivo</i> metabolic labelling, tissue processing, and advanced correlative imaging. The student will:</p> <ul style="list-style-type: none"> • Use stable isotope-labelled nutrients (e.g., ¹³C-glucose, ¹⁵N-amino acids) to directly • visualise and quantify the metabolic incorporation in each mouse bone marrow cell. • Harvest and prepare mouse femurs/tibias at defined timepoints after isotope administration. • Section, stain, and prepare samples for both light and electron microscopy. • Select regions of interest for high-resolution imaging analysis. • Perform cell segmentation and spatial data analysis to compare metabolic profiles between cell types and bone marrow compartments.
	Techniques <ul style="list-style-type: none"> • Stable isotope tracing in vivo (e.g., ¹³C-glucose, ¹⁵N-amino acids) • Bone marrow sample preparation (staining, resin embedding) • Light and electron microscopy • Nanoscale secondary ion mass spectrometry (NanoSIMS) • Image registration and correlative analysis
	Outcomes <ul style="list-style-type: none"> • A spatial map of nutrient incorporation in distinct bone marrow cell types. • Quantitative comparison of metabolic activity in osteoblasts, adipocytes, and immune cells. • A mini-atlas of metabolism in the bone marrow microenvironment. • A potential publication-quality figure set contributing to ongoing projects in the lab.
	References <p>Kai Chen UWA profile: https://research-repository.uwa.edu.au/en/persons/kai-chen</p>

Research Project Proposal 2026

Primary supervisor (name):	Dr Kai Chen
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Project title:	Visualising bone marrow cell proliferation at nanoscale using thymidine-based imaging
Project location:	M Block, QEII MC
Project Description: Aims, Design, Techniques, Outcomes, References	Aims <p>The bone marrow is a dynamic tissue responsible for the continuous production of blood and immune cells through tightly regulated proliferation and differentiation. Tracking cell proliferation within the bone marrow is essential for understanding skeletal homeostasis and disease processes such as leukemia, osteoporosis, and cancer metastasis. This project aims: i) To investigate proliferative activity of bone marrow cells under physiological or experimental conditions; ii) To map spatial patterns of cell division within the complex bone marrow microenvironment; iii) To evaluate the utility of thymidine analog incorporation combined with advanced imaging for high-resolution proliferation analysis.</p>
	Design <ul style="list-style-type: none"> Mice will be injected with stable isotope-labeled thymidine (e.g. ^{15}N-thymidine) to label actively dividing cells in vivo. Bone marrow tissue will be collected and processed for resin embedding and sectioning. Proliferative zones will be mapped and correlated with tissue architecture and cell identity using imaging and, where needed, immunolabelling.
	Techniques <ul style="list-style-type: none"> Stable Isotope Labelling: Administration of isotopically labelled thymidine to mark DNA synthesis. Sample Preparation: Bone marrow fixation, staining, resin embedding, and sectioning for imaging. Correlative Imaging: NanoSIMS for high-resolution isotopic imaging of thymidine incorporation (visualising ^{15}N), and electron microscopy (EM) for structural context.
	Outcomes <ul style="list-style-type: none"> Spatial maps of proliferative activity in bone marrow at subcellular resolution. Identification of specific cell types and niches with high turnover or quiescence. A novel workflow for imaging DNA synthesis in situ using thymidine and correlative imaging. Foundational data for future studies on regeneration, hematological disorders, or drug responses.
	References Kai Chen UWA profile: https://research-repository.uwa.edu.au/en/persons/kai-chen

Research Project Proposal 2026

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Project title:	Artificial skin models for evaluating the activity of topical antiseptics and honey
Project location:	
Project Description: Aims, Design, Techniques, Outcomes, References	Aims <ul style="list-style-type: none"> To evaluate the usefulness of different artificial skin-like surfaces or models for evaluating and comparing the efficacy of topical antiseptic products, including honey. To investigate whether differences in the antibacterial activity of antiseptics/honeys seen in standard minimum inhibitory concentration tests are reflected in results obtained using artificial skin models To compare the susceptibilities of different wound pathogens to antiseptics/honeys using artificial skin or wound models
	Design <p>Many in vitro models for testing the efficacy of topical antimicrobials rely on animal or human tissue, or complex tissue culture systems that simulate real human skin. Whilst these systems are invaluable for generating pre-clinical data, testing on other surfaces that are potentially skin-like (and simpler) could also generate useful data. Simpler, animal-free testing systems could also be more widely used as they are inherently less complex. This project will evaluate several different artificial skin models, and will also evaluate skin-like surfaces for evaluating the activity of topically applied antiseptics including honey.</p>
	Techniques <p>Acellular skin models (those not using animal skin or cell lines) will be sourced from published literature. Also, skin substitutes, such as silicone, agar or gelatin will be investigated as possible testing surfaces. After preparation of model surfaces, organisms will be applied, then an antiseptic/honey will be applied, and after one or more specific time periods organisms will be recovered using standard viable counting techniques.</p> <p>The activity of all antiseptics/honeys will also be determined in standard minimum inhibitory concentration assays to allow comparison to results obtained using the various models. Data will be statistically analysed to determine whether there are significant differences in the activity of antiseptics/honeys.</p>
	Outcomes <p>This project will generate data on the usability of several artificial skin or wound models. The valuation of different honeys/antiseptics will enable generalisations to be drawn about the relative activity of each product. Lastly, data will be generated against different Gram positive and negative wound pathogens, providing information on which species are relatively easy, or hard to eradicate.</p>

References

- Kong C, Chee CF, Richter K, Thomas N, Abd Rahman N, Nathan S. Suppression of *Staphylococcus aureus* biofilm formation and virulence by a benzimidazole derivative, UM-C162. Sci Rep. 2018 Feb 9;8(1):2758. doi: 10.1038/s41598-018-21141-2.
- Kwakman PH, Van den Akker JP, Güçlü A et al. Medical-grade honey kills antibiotic-resistant bacteria in vitro and eradicates skin colonization. Clin Infect Dis. 2008 Jun 1;46(11):1677-82. doi: 10.1086/587892.
- Price BL, Lovering AM, Bowling FL, Dobson CB. 2016. Development of a novel collagen wound model to simulate the activity and distribution of antimicrobials in soft tissue during diabetic foot infection. Antimicrob Agents Chemother 60:6880 –6889. doi:10.1128/AAC.01064-16.

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBC Email:
Project title:	Applications of artificial intelligence for improving tuberculosis control
Project location:	The Kids Research Institute, Australia
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims This study aims to undertake a systematic review of the current literature to explore the application of artificial technology (AI) in tackling tuberculosis (TB). By examining the role of AI in prevention, diagnosis, treatment, and follow-up care, this study seeks to provide a comprehensive understanding of how AI technologies can address key challenges in TB control and improve patient outcomes.</p> <p>Design Systematic review and meta-analysis following the Preferred Reporting Items for Systematic Reviews and Metanalysis (PRISMA) guidelines.</p> <p>The following aspects of the TB-AI interface will be evaluated:</p> <ul style="list-style-type: none"> • Prevention: e.g., migration screening, education, and vaccine development. • Transmission: e.g., AI driven contact tracing, prediction of transmission patterns, and outbreak modeling. • Detection: e.g., AI based diagnostics, such as, computer assisted detection (CAD) on chest X-rays and AI in detecting drug resistance. • Treatment: e.g., AI models predicting treatment outcomes, assisting in case management, or guiding drug development and resistance profiling. • Post treatment sequelae: e.g., AI in surveillance of long-term health outcomes, predicting complications, and managing post-treatment conditions.

	<p><u>Techniques</u></p> <p>A comprehensive systematic search of electronic databases will be undertaken to identify qualitative and quantitative studies that evaluate the application of AI to human pulmonary and extrapulmonary TB infection. The project will scope the search criteria, inclusion and exclusion criteria and data extraction tool. Ryann, Endnote and Excel will be used to select suitable studies and extract data. The quality and risk of bias of the included studies will be assessed using Newcastle Ottawa Scale Quality assessment. In addition to a narrative synthesis, where sufficient data are available, a random effects meta-analysis will be conducted using STATA.</p>
	<p><u>Outcomes</u></p> <p>AI has transformed many aspects of healthcare and has the potential to improve the efficacy of TB control programs. While preliminary studies exploring the role of AI in TB management show promise, there is no comprehensive review to synthesize these findings. This study aims to fill this knowledge gap and present a comprehensive overview of the potential applications of AI to improve TB control programs.</p>

Research Project Proposal 2026

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	Name: Email:
SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBC Email:
Project title:	Dynamic changes of gastric microbiota at different stages of Correa's cascade in the development of intestinal type gastric cancer
Project location:	L Block, QE2 Medical Centre, Nedlands
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims</p> <p>In 2020, more than 1 million new gastric cancer cases were recorded and over three fourth million patients died of gastric cancer. Among these cases, intestinal type gastric cancer is the most common form of stomach adenocarcinoma, arising from a progression of chronic non-atrophic gastritis initiated by <i>Helicobacter pylori</i> infection, to atrophic gastritis and intestinal metaplasia and finally into gastric cancer (also known as the Correa's Cascade).</p> <p>It is well known that <i>H. pylori</i> infection is a carcinogen to the development of gastric cancer and it is estimated that <i>H. pylori</i> infects around half of the world's population. Recently, <i>Streptococcus anginosus</i> was reported to promote gastric inflammation, atrophy, and tumorigenesis. Although not as abundant as gut microbiota, gastric microbiota has been reported to contain five major bacterial phyla, that is, <i>Firmicutes</i>, <i>Bacteroidites</i>, <i>Actinobacteria</i>, <i>Fusobacteria</i> and <i>Proteobacteria</i>, the composition of which are affected by multiple factors such as diet, drug, and <i>H. pylori</i> infection, etc. Therefore, the disorder of bacterial homeostasis in the stomach, rather than just <i>H. pylori</i> and <i>S. anginosus</i> infections, may play essential roles in the development of intestinal type gastric cancer. Currently, few studies focus on the dynamic changes of gastric microbiota during the development of intestinal type gastric cancer by following the Correa's Cascade. Therefore, the aim of this project is to dissect the bacterial composition at each stage of the Correa's Cascade, identify the core bacterial genera and species during the development of intestinal type gastric cancer, and link their dynamic changes with gastric cancer development through the amplicon sequencing data sourced and integrated from public database.</p>

	<p>Design</p> <p>This study proposes to collect and integrate publicly available high-quality 16S rRNA sequencing datasets of gastric microbiome from the representative stages of the Correa's Cascade, including healthy controls, non-atrophic gastritis, atrophic gastritis, intestinal metaplasia, and intestinal-type gastric cancer, and systematically and comparatively analyze these data for characteristic bacterial genera and species in each stage. Following data acquisition, standardized preprocessing and batch effect removal will be performed. The study will compare bacterial composition, diversity, and ecological network alterations across gastric disease stages, and further evaluate the impact of <i>H. pylori</i> infection status on microbial community differences. Cluster-based analyses will be applied to define distinct gastric microbial community types, aiming to identify the dynamic abundance changes of core bacteria during the progression of intestinal type gastric cancer and to elucidate their potential roles in carcinogenesis.</p> <p>Techniques</p> <p>Sequencing data will be processed using the QIIME2 pipeline for quality filtering, feature table construction, and taxonomic assignment against the SILVA database. Community structure will be analysed by alpha- and beta-diversity metrics (Shannon, Simpson, Chao1, Bray-Curtis). Differential abundance analysis will be performed with DESeq2, while network-based methods (e.g., SparCC and NetMoss2) will be used to assess microbial interactions and robustness across datasets. Cluster analysis of microbial profiles will be carried out with Jensen-Shannon divergence and Partitioning Around Medoids algorithms. Machine learning algorithms, e.g., random forest classifiers, will be trained to identify key taxa that discriminate between different disease stages, and feature selection will be implemented with the Boruta algorithm. Statistical significance will be assessed by ANOVA or PERMANOVA with multiple-testing corrections.</p> <p>Outcomes</p> <p>The results of the project will facilitate the understanding of the dynamic changes of gastric microbiota during the progression of gastric cancer initiated by <i>H. pylori</i> infection and provide potential biomarkers for the therapeutic interventions of intestinal type gastric cancer via targeting key bacteria in gastric microbiota.</p>
	<p>References</p> <ul style="list-style-type: none"> • Lin JL, Lin JX, Lin GT, et al. Global incidence and mortality trends of gastric cancer and predicted mortality of gastric cancer by 2035. BMC Public Health. 2024 Jul 2;24(1):1763. • Correa P, Piazuelo MB. The gastric precancerous cascade. J Dig Dis. 2012 Jan;13(1):2-9. • Liou JM, Malfertheiner P, Smith SI, et al. 40 years after the discovery of Helicobacter pylori: towards elimination of H pylori for gastric cancer prevention. Lancet. 2024 Jun 15;403(10444):2570-2572. • Fu K, Cheung AHK, Wong CC, et al. Streptococcus anginosus promotes gastric inflammation, atrophy, and tumorigenesis in mice. Cell. 2024 Feb 15;187(4):882-896.e17. • Nardone G, Compare D. The human gastric microbiota: Is it time to rethink the pathogenesis of stomach diseases? United European Gastroenterol J. 2015 Jun;3(3):255-60.

	<ul style="list-style-type: none">• Liu, D., Zhang, R., Chen, S. et al. Analysis of gastric microbiome reveals three distinctive microbial communities associated with the occurrence of gastric cancer. BMC Microbiol 22, 184 (2022). <p>7. Erawijantari PP, Mizutani S, Shiroma H, et al. Influence of gastrectomy for gastric cancer treatment on faecal microbiome and metabolome profiles. Gut 2020;69:1404-1415.</p> <p>8. Coker OO, Dai Z, Nie Y, et al. Mucosal microbiome dysbiosis in gastric carcinogenesisGut 2018;67:1024-1032.</p> <p>9. Png CW, Lee WJJ, Chua SJ, et al. Mucosal microbiome associates with progression to gastric cancer. Theranostics. 2022 Jan 1;12(1):48-58.</p> <p>10. Wang, G., Wang, H., Ji, X., et al. Intratumoral microbiome is associated with gastric cancer prognosis and therapy efficacy. Gut Microbes, 2024; 16(1).</p>
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Research Project Proposal 2026

Primary supervisor (name):	Dr Liz Johnstone
Contact details (phone; email; location)	6151 0747, liz.johnstone@uwa.edu.au, SBMS and Harry Perkins Institute of Medical Research
Project title:	Investigation of G Protein-Coupled Receptor Molecular Pharmacology
Project location:	Harry Perkins Institute of Medical Research
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims</p> <p>G protein-coupled receptors (GPCRs) are critically important targets for pharmaceuticals due to their crucial role in responding to hormonal, neurotransmitter and environmental stimuli. We are looking to develop the next generation of medicines targeting these receptors that are not only more effective, but also have fewer side effects. This requires improved understanding of how GPCRs function at the molecular and cellular level, in terms of ligand binding, signalling, regulation, and cellular trafficking.</p> <p>Aim: to investigate various novel aspects of GPCR molecular pharmacology</p>
	<p>Design</p> <p>Receptor pharmacology will be monitored using a variety of cell-based assays, which enable pharmacological characterisation of receptor ligand binding, signalling, regulation, and cellular trafficking.</p>
	<p>Techniques</p> <p>Bioluminescence resonance energy transfer (BRET) and other cell-based assays will be used to monitor receptor pharmacology. Receptors and interacting biomolecules (protein and ligands) will be labelled with BRET tags and transfected or added to cells, with resulting interactions monitored using BRET.</p>
	<p>Outcomes</p> <p>The results will aid in future drug discovery efforts for the GPCRs under investigation.</p>

Research Project Proposal 2026

Primary supervisor (name):	Dr Lucy Furfaro	
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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Dr Lucy Furfaro Email: Lucy.Furfaro@uwa.edu.au Division of Obstetrics and Gynaecology, King Edward Memorial Hospital, Subiaco	
Project title:	The art of induction: Uncovering Group B Streptococcus prophage functionality	
Project location:	King Edward Memorial Hospital, Subiaco	
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Over a century since their discovery, bacterial viruses known as bacteriophages (phages) could provide an answer to the rapidly developing antibiotic resistance in bacterial pathogens. These viruses are ubiquitous and estimates of their abundance equate to a trillion phages per grain of sand on this Earth¹. The lytic lifecycle of phages is an attractive option for therapy, due to the ability to infect specific ranges of host bacteria, replicate within them, kill the host bacteria and continue the process with the now exponential number of phages released. This is essentially a self-dosing antimicrobial agent².</p> <p><i>Streptococcus agalactiae</i>, also known as Group B <i>Streptococcus</i> (GBS), is a leading neonatal pathogen that is screened for during pregnancy to prevent vertical transmission³. This results in widespread antibiotic administration to colonised women during pregnancy and may be an ideal clinical scenario for phage therapy⁴. To date, no obligately lytic GBS-specific phages have been isolated, rather only temperate phages have been characterised.</p> <p>Our group is interested in understanding the role these temperate phages play in GBS. Prophages are widespread in many microorganisms; however, some are present as remnants and are inactive. This project will assess the phenotypically active prophages of GBS by inducing their entry to the lytic cycle. There are a number of different mechanisms of induction and we aim to compare a variety of agents (e.g. common medications⁵ and antibiotics) and their impact on phage induction.</p> <p>Aims</p> <ul style="list-style-type: none"> • Compare the ability of different agents to induce prophages from clinical GBS isolates. • Isolate, purify and characterise induced phages to assess host range activity. • Assess the cross-infection of the induced phages in other phage-free GBS isolates. 	

	<p>Design</p> <p>This project involves several different techniques listed below to enable the assessment of a collection of diverse clinical GBS isolates from maternal vaginorectal carriage and neonatal invasive disease. Using these isolates, the functional nature of prophages will be assessed through induction assays using different bacterial stress inducing agents.</p>
	<p>Techniques</p> <ul style="list-style-type: none"> • Bacterial culture • Bacterial growth and induction curves • Antimicrobial susceptibility testing (optional) • Viral (bacteriophage) plaque assays • Bacteriophage isolation, purification and characterisation • Nucleic acid extraction • Whole genome sequencing • Bacterial genomic analyses (bioinformatics)
	<p>Outcomes</p> <p>This project pairs the genomic insights with biological function to determine which prophages of GBS are dormant genomic remnants or active viral hitchhikers. Understanding how readily induction occurs and under what conditions will provide insight into the role phages play in gene transfer among these neonatal pathogens and equip us with future candidates to develop phage-based therapeutics.</p>
	<p>References</p> <ul style="list-style-type: none"> • Keen EC. A century of phage research: bacteriophages and the shaping of modern biology. <i>Bioessays</i>. 2015 Jan;37(1):6-9. • Kutter EM, Kuhl SJ, Abedon ST. Re-establishing a place for phage therapy in western medicine. <i>Future Microbiol</i>. 2015;10(5):685-8. • Verani JR, McGee L, Schrag SJ. Prevention of perinatal group B streptococcal disease: revised guidelines from CDC, 2010. <i>Recommendations and Reports</i>. 2010;59(10):1-32. • Furfaro LL, Chang BJ, Payne MS. Applications for bacteriophage therapy during pregnancy and the perinatal period. <i>Frontiers in Microbiology</i> [Review]. 2018 2018- January-11;8(2660). • Sutcliffe SG et al. Common oral medications lead to prophage induction in bacterial isolates from the human gut. <i>Viruses</i>. 2022 Dec 21;15(1):25.

Research Project Proposal 2026

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	Name: Blair Johnson Email: blair.johnson@uwa.edu.au
SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Lucy Barrett Email: Lucy.Barrett@uwa.edu.au
Project title:	Assessing the local immune response to injury in burn tissue.
Project location:	Burn Injury Research Unit (UWA), Harry Perkins Research Institute (North)
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims</p> <ul style="list-style-type: none"> • Understand the impact of burn injury on the phenotype and function of skin-infiltrating immune cells, with a focus on neutrophils extracted from burn tissue. • Investigate if there are functional differences in immune cells in burn tissue from paediatric patients compared to adult patients. • Investigate correlations between the local immune response and burn patient outcome measures, including time to healing and length of stay in hospital. <p>Design</p> <p>Burn tissue will be obtained from adult and paediatric burn patients treated at the Burns Units at Fiona Stanley Hospital or Perth Children's Hospital. Immune cells will be isolated from the tissue using a protocol based on Mulder et al. 2022 (https://doi.org/10.3389/fimmu.2022.1034420). Part of the tissue will also be used for histology. Isolated immune cells will be used to study phenotype and functionality, including phagocytosis, migration activity and ROS production.</p> <p>Techniques</p> <p>For this project the following techniques will be used:</p> <ul style="list-style-type: none"> • Skin digestion to isolate immune cells from tissue samples • Flow cytometry to quantify and assess immune cell phenotypes, phagocytosis activity and ROS production • <i>In vitro</i> cell assays including cellular uptake of fluorescent bacteria to study phagocytosis, scratch assays to study migration, and other cell culture assays to study immune cell function. • Microscopy to study tissue histology (immunohistochemistry)

	<p>Outcomes</p> <p>This project will provide valuable insights into the response of immune cells to burn injury at the local level, complementing ongoing research being conducted by our collaborators in the Netherlands. Through replication of their techniques in an independent adult cohort, this research will strengthen our understanding of how burn injury alters neutrophil activity, phenotype and function.</p> <p>Additionally, this project aims to also utilise paediatric patient samples with the same assays, which has not been studied before.</p> <p>Finally, with our established flow cytometry panels, we will be able to analyse other immune cell types in the tissue, including macrophages, T cells, and B cells, which will complement our existing research investigating the systemic response to burn injury.</p> <p>This project is part of our larger goal to understand how burn injury can affect the immune system, patient outcomes, and long-term patient health.</p> <p>Ethics are already in place to collect samples from both adult and paediatric burn patients.</p>
	<p>References</p> <p>Mulder 2022 Burn-injured skin is marked by a prolonged local acute inflammatory response of innate immune cells and pro-inflammatory cytokines (https://doi.org/10.3389/fimmu.2022.1034420)</p> <p>Barrett 2022 Non-severe burn injury increases cancer incidence in mice and has long-term impacts on the activation and function of T cells (10.1093/burnst/tkac016)</p> <p>Mulder 2021 Persistent Systemic Inflammation in Patients With Severe Burn Injury Is Accompanied by Influx of Immature Neutrophils and Shifts in T Cell Subsets and Cytokine Profiles (https://doi.org/10.3389/fimmu.2020.621222)</p>

Research Project Proposal 2026

Primary supervisor (name):	A/Prof Lynette Fernandes
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Other supervisor/s if any (name):	Dr Orla Barry
Contact details (phone; email; location)	O.Barry@ucc.ie University College Cork, Ireland
Project title:	Identifying the processing demands of summative assessments in pharmacology
Project location:	M Block QEII Medical Centre
Project Description: Aims; Design; Techniques; Outcomes:	<p>Assessment is probably the most important event to drive student learning. However, unless students understand what is being asked of them in exams, learning may be steered in the wrong direction. While students may readily address the knowledge demands of exam questions, they often address processing demands poorly or not at all. This may be due to a misinterpretation of the action words / phrases in exam questions. While many action words have universal meanings, others have nuanced meanings within certain disciplines. This project aims to research, document and address the processing demands associated with assessment literacy in Pharmacology.</p> <p>This project would appeal to students who are interested in working on a project that involves education research.</p>

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: A/Prof Demelza Ireland Email: demelza.ireland@uwa.edu.au
Project title:	Swimming Upstream – Assessing the impact of chlorinated pool exposure on the vaginal microbiome
Project location:	Clinical Perinatal Research Laboratories, King Edward Memorial Hospital
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Swimming is widely recommended for women across all life stages – from pregnancy, to postpartum to menopause – as a safe and beneficial form of exercise. Yet, despite its popularity, no research has explored how the chlorinated pool environment might affect the vaginal microbiome.</p> <p>This gap is especially concerning given that 58% of the aquatic workforce is made up of women, according to the 2023 National Aquatic Workforce Report. These women are not just recreational swimmers, they are lifeguards, instructors and professionals who spend extended hours in chlorinated water. Understanding how this exposure may influence vaginal health is not only a matter of public health, but also important for workplace safety and gender equity.</p> <p>While studies have examined how chlorination affects the skin, gut, and oral microbiomes, there is a clear evidence gap regarding its impact on vaginal health. Interestingly, studies have found chlorine washing as an effective method of reducing <i>Lactobacillus brevis</i> spoilage during the brewing process. Although, this is not a vaginal microenvironment, it could give insight into potential clinical implications.</p> <p>This study was partly motivated by anecdotal observations from community interactions, including a conversation with a swimming instructor who reported experiencing recurrent episodes of thrush, a common experience amongst her female colleagues. Although informal, this account gave insight into a lack of understanding and support in regard to the vaginal health in the aquatic professional setting. By addressing this evidence gap, we can inform safer health guidelines, support wellbeing of women in aquatic professions, and empower all women with knowledge about how the surrounding environments may influence their bodies.</p> <p>Aims: To assess the differences in the composition and diversity of the vaginal microbiome between women who regularly swim in chlorinated pools and those who are active but not in a pool setting.</p>

	<ul style="list-style-type: none"> • Examine if frequency and length of pool exposure is correlated with vaginal microbiome diversity or relative abundance of specific microbes • Explore associations between the microbiome profiles and self-reported adverse vaginal symptoms <p>Design: Cross-sectional observational study.</p> <p>Sample size: 60-80 women aged 18-45 years divided into:</p> <ul style="list-style-type: none"> • Regular swimmers (>1 swim/week in a pool for more than 3 months) • Aquatic professionals (>1 long exposure/week for more than 3 months) • Non-swimmers (no pool exposure in the past 3 months) <p>Exclusion criteria:</p> <ul style="list-style-type: none"> • Recent antibiotic use • Pregnancy <p>Data collection:</p> <ul style="list-style-type: none"> • Questionnaire: Age, BMI, smoking status, sexual activity, contraceptive use, menstrual phase, urogenital symptoms, swimming frequency and duration • Sample collection: self-collected mid-vaginal swab <p>Techniques:</p> <ul style="list-style-type: none"> • Laboratory: DNA extraction and full length 16S rRNA gene sequencing will be used to characterise the vaginal microbiome. • Data analysis: Learn basic bioinformatic tools to analyse sequences and identify any associations with participant chlorination exposure and demographic characteristics. <p>Outcomes:</p> <p>These findings could inform public health recommendations for women who swim recreationally or professionally. By clarifying the impact of chlorination exposure on vaginal health, this research may inform safer swimming practices, raise awareness of environmental influences on the microbiome, and support the development of targeted interventions in women's health.</p>
	<p>References:</p> <ul style="list-style-type: none"> • Puce L, Hampton-Marcell J, Trabelsi K, Ammar A, Chtourou H, Boulares A, et al. Swimming and the human microbiome at the intersection of sports, clinical, and environmental sciences: A scoping review of the literature. <i>Front Microbiol.</i> 2022;13:984867. • Eken BF, Akkoç O, Yücesir İ, Akmansoy ŞC, Kadir T, Ulucan K. The alteration of oral microbiota before and after training in swimmers. <i>Cellular and molecular biology.</i> 2023;69 11:92-102. • Munford ARG, Chaves RD, Granato D, Sant'Ana AS. Modeling the inactivation of <i>Lactobacillus brevis</i> DSM 6235 and retaining the viability of brewing pitching yeast submitted to acid and chlorine washing. <i>Applied Microbiology and Biotechnology.</i> 2020;104(9):4071-80.

Research Project Proposal 2026

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Other supervisor/s if any (name and email address):	Name: Dr. Ritika Kar Bahal Email: ritika.karabahal@uwa.edu.au
	Name: Email:
Project title:	Understanding the role of <i>Moraxella</i> species in the airways: exploring interactions with human epithelial cells
Project location:	QEII, L block Level 2
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Background: The genus <i>Moraxella</i> is a key component of the human upper airway microbiome, with <i>Moraxella catarrhalis</i> established as both a common commensal and a significant opportunistic pathogen. It is implicated in otitis media, pneumonia, and acute exacerbations of chronic respiratory diseases, where it interacts dynamically with both host epithelial cells and co-colonising bacteria. In contrast, non-<i>catarrhalis</i> species such as <i>M. nonliquefaciens</i> and <i>M. lincolnii</i> are frequently detected in the nasopharynx, particularly in early life, yet their roles in airway health and disease remain poorly understood. Historically, these species have been overlooked as “benign commensals,” but emerging evidence suggests they may influence epithelial responses, shape microbial community dynamics, and potentially prime the airway environment for pathogen colonisation.</p> <p>Despite their prevalence, there is a striking lack of mechanistic studies on how non-<i>catarrhalis</i> <i>Moraxella</i> interact with human epithelial cells, whether they contribute to immune modulation, and how they compete or cooperate with pathogenic <i>M. catarrhalis</i>. This knowledge gap limits our understanding of airway colonisation ecology and the potential for these species to act as either protective commensals or cofactors in respiratory disease.</p> <p>This project will address these gaps by investigating epithelial responses to <i>M. nonliquefaciens</i> and <i>M. lincolnii</i> compared with <i>M. catarrhalis</i>, focusing on adhesion, invasion, cytotoxicity, and barrier integrity. In addition, inter-bacterial interaction models will assess how non-<i>catarrhalis</i> species influence <i>M. catarrhalis</i> colonisation dynamics, providing new insights into the ecological and functional roles of the broader <i>Moraxella</i> genus in the human airways.</p> <p>Aims: To describe the effect of infection with <i>Moraxella</i> species on human airway epithelial cells</p>

	<ul style="list-style-type: none"> • Culture respiratory epithelial cells at air-liquid interface (representative of the nasopharynx/upper airway) and in submerged monolayer cultures. • Investigate adhesion, invasion, cytotoxicity, and barrier impact in response to infection with <i>M. nonliquefaciens</i> and <i>M. lincolnii</i>, and compared with <i>M. catarrhalis</i>. • Investigate inter-bacterial interactions in epithelial cell infection models by sequential and simultaneous exposures) to the different <i>Moraxella</i> species <p>Design</p> <p>Aim 1 — Model establishment & validation</p> <ul style="list-style-type: none"> • Cell systems: A549 (human alveolar basal cells) and/or Detroit 562 (human pharyngeal epithelial; nasopharynx-like), and primary human nasal epithelial cells (optional, if available). • Bacteria & culture: <i>M. nonliquefaciens</i>, <i>M. lincolnii</i>, <i>M. catarrhalis</i> clinical strains. Growth on chocolate/5% sheep blood agar at 30 or 37 °C; prepare mid-log suspensions for infections. • Model optimization: Standardize multiplicity of infection, time of infection and time of readouts. • Baseline readouts: cell viability (LDH assay), barrier integrity (TEER), CFU for bacterial growth and adhesion <p>Aim 2 — Single-species infection kinetics</p> <ul style="list-style-type: none"> • MOIs: 1, 10, 50 for 1–6 h (adhesion/invasion) and 24 h (cytotoxicity/signaling). • Endpoints: <ul style="list-style-type: none"> • Adhesion & invasion (CFU counts; gentamicin protection). Assays/targets modeled on <i>M. catarrhalis</i> epithelial studies. • Barrier impact: TEER drop (%). • Cytotoxicity: LDH release. • Innate signaling: qPCR/ELISA for IL-6, IL-8/CXCL8, CXCL1, CCL20; pathway probes for EGFR/ERK/NF-κB where relevant. • Biofilm on epithelial monolayers (optional): crystal violet biomass + viable counts (compare species). <p>Aim 3 — Two-species interaction tests with <i>M. catarrhalis</i></p> <ul style="list-style-type: none"> • Co-infection (simultaneous): <i>M. nonliquefaciens</i>/<i>M. lincolnii</i> + <i>M. catarrhalis</i> at 1:1 and 1:10 ratios; quantify species-specific CFU (selective plates/qPCR), adhesion/invasion, and biofilm biomass. • Sequential exposure: pre-expose cells 2 h with <i>M. nonliquefaciens</i> (or <i>M. lincolnii</i>), then infect with <i>M. catarrhalis</i> (and vice versa) to test facilitation/competition. <p>Controls & quality</p> <ul style="list-style-type: none"> • Heat-killed bacteria controls; LPS control (low dose) to benchmark cytokines.
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	<ul style="list-style-type: none"> • Multiplicity controls for host cell viability; mycoplasma-free cell lines. • Biosafety: BSL-2 practices for all <i>Moraxella</i> spp.; institutional approvals for primary cells.
	<p>Techniques</p> <ul style="list-style-type: none"> • Human epithelial culture (Detroit 562/A549), MOI-controlled infections, CFU plating, gentamicin protection assay, TEER, LDH cytotoxicity, ELISAs/qPCR, crystal-violet biofilm assay, species-specific qPCR, basic biostatistics (ANOVA/ANCOVA with MOI, time, species factors).
	<p>Expected Outcomes</p> <ul style="list-style-type: none"> • Develop skills in epithelial cell culture and bacterial-host interaction model • First side-by-side phenotyping of <i>M. nonliquefaciens</i> and <i>M. lincolnii</i> on human airway epithelia: adhesion/invasion capacity, cytokine profiles, and barrier effects relative to <i>M. catarrhalis</i>. • Evidence for interaction logic: co-/sequential-infection data indicating whether <i>M. nonliquefaciens</i> (or <i>M. lincolnii</i>) enhances <i>M. catarrhalis</i> epithelial binding, biofilm stability, or inflammatory tone—mechanistic context for their positive association in nasal microbiota.
	<p>References</p> <ul style="list-style-type: none"> • Yu K, Tenaglia V, Chua EG, Haines R, Bahal G, Nicol MP, Bahal RK. Interactions between bacteria in the human nasopharynx: a scoping review. <i>Lancet Microbe</i>. 2025 Jul;6(7). • Claassen-Weitz S, Xia Y, Workman L, Hannan L, Gardner-Lubbe S, Mwaikono KS et al. The nasopharyngeal microbiome in South African children with lower respiratory tract infection: a nested case-control study of the Drakenstein Child Health Study. 2025 <i>Clinical Infectious Diseases</i> 2025 Apr 17. • Claassen-Weitz, S., Gardner-Lubbe, S., Xia, Y., Mwaikono, K. S., Mounaud, S. H., Nierman, W. C., Workman, L., Zar, H. J., & Nicol, M. P. (2023). Succession and determinants of the early life nasopharyngeal microbiota in a South African birth cohort. <i>Microbiome</i>, 11(1), [127].

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Dr Sonia Fernandez Email: Sonia.fernandez@uwa.edu.au
Project title:	Developing innovative treatments for paediatric brain cancers
Project location:	The Kids Institute Australia
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims</p> <p>The Brain Tumour Research team at Telethon Kids is co-directed by Professor Nick Gottardo and A/Prof Raelene Endersby. The overarching goals of our group are to define the poorly understood basic biology of several types of childhood brain tumours and improve therapies. We achieve this in the following ways:</p> <ul style="list-style-type: none"> • Elucidate the molecular basis of different brain tumour types, including medulloblastoma and ependymoma among others, through the analysis of primary patient specimens. • Improve understanding of the molecular events contributing to these diseases, by analysing the impact of altered signalling pathways on survival, proliferation, invasiveness and tumorigenicity of brain tumour cells. • Develop comprehensive preclinical models of paediatric brain tumours in which to test new treatments. We utilise transplantable xenograft, patient derived xenograft, and genetically engineered tumour models representative of paediatric brain cancer in our translational research. • Obtain and test new therapies within our preclinical pipeline that considers all aspects of standard of care treatment, including brain tumour resection surgery, MRI imaging, clinical chemotherapy, and radiation protocols in appropriate brain tumour models. We acquired Australia's first X-RAD SmART platform to model clinical radiation treatment and are currently investigating new therapies that can enhance its efficacy to hopefully reduce the harmful radiation dose.

	<p>Techniques</p> <p>We currently have a project opportunity for a self-motivated and enthusiastic individual. We invite you to meet with us to discuss specific projects. The student will develop expertise in a wide range of technologies including:</p> <ul style="list-style-type: none">• Animal techniques• Histology such as paraffin sectioning and immunohistochemistry• Cell/tissue culture from mouse and human specimens• Molecular techniques including DNA/RNA analysis, PCR and cloning• Biochemical techniques such as protein extraction, western blotting and IP <p>Students are expected to have or develop excellent writing and oral presentation skills.</p>
	<p>Outcomes</p> <p>Translate our findings into improved therapies through clinical collaborations.</p>

Research Project Proposal 2026

Primary supervisor (name):	Dr Minda Sarna
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	Name: Dr Sonia McAlister Email: Sonia.McAlister@thekids.org.au
SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBC Email:
Project title:	Effect of maternal pertussis vaccines on childhood pneumococcal vaccine effectiveness.
Project location:	The Kids Research Institute Australia
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims: Pertussis (whooping cough) is a highly contagious, potentially severe respiratory illness, with the highest burden of disease in infants <6months.¹ As infants are not fully protected until the age of 6 months when they receive their third pertussis childhood vaccine dose, pertussis vaccination in pregnancy (maternal immunisation) was introduced as a way of protecting young infants through passive transfer of maternal antibodies until infants generated their own antibody response.^{2,3}</p> <p>While maternal pertussis vaccines are effective in preventing infant pertussis, recent evidence has shown that maternal antibodies may interfere with the infant's response to childhood pneumococcal vaccines. Childhood pneumococcal vaccines prevent invasive pneumococcal disease (IPD) from <i>Streptococcus pneumoniae</i>.⁴ The aim of this project is to investigate possible interference of maternal pertussis vaccines on childhood pneumococcal vaccines using linked population data to conduct epidemiological analyses.</p> <p>Design: This is an exploratory proof-of-principle observational study using linked, population data from 2010-2018 of a cohort of West Australian children.⁵ Cohort data will be linked with maternal and childhood immunisation and hospitalisation data.</p> <p>Techniques: Skills learned will include management of large datasets, epidemiological analyses, including descriptive statistics, multivariable and Cox regression. Students will use statistical software (Stata) to query the data. Stata proficiency is desirable but not essential, training will be offered, and students will be supported to learn new data analytic skills.</p> <p>Outcomes: Vaccine effectiveness of childhood pneumococcal vaccine in infants whose mothers were vaccinated with pertussis vaccine during pregnancy, estimated by calculating the childhood incidence rate of IPD and other associated outcomes.</p>

	<p>References</p> <ol style="list-style-type: none">1. Skoff TH, Hadler S, Hariri S. The epidemiology of nationally reported pertussis in the United States, 2000-2016. Clin Infect Dis. 2019;68(10):1634–16402. Amirthalingam G. Strategies to control pertussis in infants. Arch Dis Child. 2013;98(7):552–555.3. Marshall KS, Quinn HE, Pillsbury AJ, et al. Australian vaccine preventable disease epidemiological review series: Pertussis, 2013–2018. Commun Dis Intell. 2022:464. Zimmermann P, Perrett KP, Messina NL, Donath S, Ritz N et al. The Effect of Maternal Immunisation During Pregnancy on Infant Vaccine Responses. EClinicalMedicine. 2019 Jul 26;13:21-30.5. Sarna M, Taye BW, Le H, Giannini F, Glass K et al. Cohort profile: A population-based record linkage platform to address critical epidemiological evidence gaps in respiratory syncytial virus and other respiratory infections. https://doi.org/10.23889/ijpds.v6i1.2376
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Research Project Proposal 2026

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	Name: Prof Charlene Kahler Email: Charlene.kahler@uwa.edu.au
Project title:	Investigating the Effects of Inhibiting Mip and EptA in Chlamydia-Gonorrhoeae Co-infections
Project location:	Marshall Centre for Infectious Disease Research and Training, Room 2.04, School of Biomedical Sciences, L Block, QEII Medical Centre
Project Description: Aims, Design, Techniques, Outcomes, References	<p><i>Chlamydia trachomatis</i> and <i>Neisseria gonorrhoeae</i> are both Gram-negative bacteria that cause sexually transmitted infections (STIs). Globally, <i>C. trachomatis</i> is estimated to cause 125 million infections and <i>N. gonorrhoeae</i> is estimated to cause 87 million infections each year. Both infections are often asymptomatic, but when left untreated cause severe health complications, such as pelvic inflammatory disease and infertility. Many of these cases are believed to be co-infections, where one person carries both infections. These co-infections can be particularly challenging to treat, as drugs effective against <i>C. trachomatis</i> are not effective against <i>N. gonorrhoeae</i> and vice versa. The rise of multi-drug resistance (MDR) in <i>N. gonorrhoeae</i> complicates this situation even further, as the bacteria becomes resistant to last-line antibiotics. Interestingly, a recent study has shown that <i>C. trachomatis</i> and <i>N. gonorrhoeae</i> may facilitate each other during co-infections, leading to persistent STIs.</p> <p>The macrophage infectivity potentiator (Mip) is peptidyl-prolyl <i>cis-trans</i> isomerase enzyme involved in correct protein folding and has been shown to play an important role in the virulence of several pathogenic bacteria. EptA is a phosphoethanolamine transferase enzyme that is important for modifying the lipooligosaccharide (LOS) in <i>N. gonorrhoeae</i> and contributes to resistance against certain antibiotics. Our lab has previously observed that Mip inhibitors can successfully reduce the virulence of <i>C. trachomatis</i> and <i>N. gonorrhoeae</i>, and that EptA inhibitors can successfully increase the sensitivity of <i>N. gonorrhoeae</i> to resistant antibiotics in <i>in vitro</i> mono-infections. However, the efficacy of these inhibitors in co-infections has not yet been investigated.</p> <p>Aims The aims of this project are to:</p> <ul style="list-style-type: none"> • Establish an <i>in-vitro</i> Chlamydia-Gonorrhoeae co-infection model • Screen Mip inhibitors in the <i>in-vitro</i> Chlamydia-Gonorrhoeae co-infection model • Screen EptA inhibitors in the <i>in-vitro</i> Chlamydia-Gonorrhoeae co-infection model

	<p>Design</p> <p>This project will involve the use of <i>in vitro</i> tissue culture models in cell culture plates to screen Mip and EptA inhibitors. Additionally, transwell inserts will be used to observe the effect of non-contact cultures through the culture medium.</p>
	<p>Techniques</p> <p>Bacterial and cell culture, aseptic technique, infection assays, immunofluorescence, SDS-PAGE and western blot analysis</p>
	<p>Outcomes</p> <p>This project will further our understanding of the interactions between <i>C. trachomatis</i> and <i>N. gonorrhoeae</i> during co-infections and help to gain a better understanding of the roles of that virulence factors, such as Mip and EptA, have during co-infections.</p>
	<p>References</p> <ol style="list-style-type: none"> 1. Ball LM, Bronstein E, Liechti GW, Maurelli AT. <i>Neisseria gonorrhoeae</i> drives <i>Chlamydia trachomatis</i> into a persistence-like state during <i>in vitro</i> co-infection. Infect Immun. 2024;92(1):e0017923. 2. Onorini D, Borel N, Schoborg RV, Leonard CA. <i>Neisseria gonorrhoeae</i> limits <i>Chlamydia trachomatis</i> inclusion development and infectivity in a novel <i>in vitro</i> co-infection model. Frontiers in Cellular and Infection Microbiology. 2022 Jul 7;12:911818. 3. Thai VC, Stubbs KA, Sarkar-Tyson M, Kahler CM. Phosphoethanolamine transferases as drug discovery targets for therapeutic treatment of multi-drug resistant pathogenic Gram-negative bacteria. Antibiotics. 2023 Aug 29;12(9):1382.

Research Project Proposal 2026

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Project title:	Development of a human cell model to study rare bone diseases
Project location:	Bone Biology & Disease Laboratory, UWA School of Biomedical Sciences and (2) Translation Genetics, Precision Health, Genetic and Rare Diseases, The Kids Institute
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Background:</p> <p>Paradoxically, rare diseases are common. Hundreds of millions of lives are globally affected by ~10,000 unique rare genetic diseases. In Australia alone, two million individuals suffer with a rare disease, a figure that is similar to the proportion of people living with diabetes or asthma. Despite this, people with rare diseases face disproportionate and longstanding inequity in care, including gruelling diagnostic delays and lack of treatment. Development of new diagnostics and treatments for patients with rare diseases ultimately requires a better understanding of the mechanisms underlying disease pathobiology. Autosomal Recessive Osteopetrosis (ARO) is a rare (1:250,000 births) but devastating high bone mass disease that occurs in children and is fatal unless a suitable bone marrow transplant is performed. Children suffering ARO commonly exhibit bone fractures, poor growth, absence of a bone marrow cavity and vision loss due to optic nerve compression. Currently, very little is known about the pathogenesis of ARO, partly due to the rarity of ARO and limited patient samples available to study ARO pathobiology in depth. ARO is typically associated with dysfunction of osteoclasts, giant bone-digesting cells that function to regulate bone turnover and homeostasis. Osteoclasts are multinucleated cells derived from the fusion of mononuclear macrophage precursors.</p> <p>Presently, we lack suitable cellular models of human osteoclasts that can be manipulated genetically to introduce ARO disease causing gene mutations and thus be used as a testbed to study the mechanisms underpinning osteoclast dysfunction in ARO patients.</p>

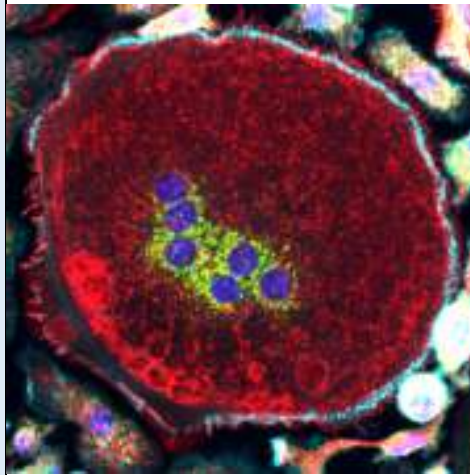


Figure 1: Morphology of an osteoclast

Aims:

This project aims to develop a new cellular model of ARO using osteoclasts derived from human inducible Pluripotent Stem (iPS) cells carrying ARO-disease causing gene mutations.

Techniques:

Students will become familiar with the following key methods: stem cell and osteoclast culture, immunofluorescence confocal microscopy, CRISPR gene editing, sequencing, bone resorption assays etc.

Outcomes

- (i) Optimise and characterise the differentiation of human iPS cell into functional osteoclasts (hiPSdOCs).
- (ii) Introduce an ARO causing genetic mutation in hiPSdOCs.

References

Xu et al., 2020, The molecular structure and function of sorting nexin 10 in skeletal disorders, cancers, and other pathological conditions, J. Cell Physiology. <https://doi.org/10.1002/jcp.30173>

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBC Email:
Project title:	Targeting Lipid Metabolism to Improve Immunotherapy in Lung Cancer and Mesothelioma
Project location:	Targeting Lipid Metabolism to Improve Immunotherapy in Lung Cancer and Mesothelioma
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Immunotherapy, particularly immune checkpoint blockade (ICB), has transformed the treatment of advanced lung cancer and mesothelioma. However, up to 70% of patients do not respond to these therapies, highlighting an urgent need to improve their effectiveness (1,2).</p> <p>One of the major barriers to successful immunotherapy is the presence of regulatory T cells (Tregs) in the tumour. These cells suppress the immune system and prevent it from attacking the cancer. Recent research suggests that Tregs inside tumours rely heavily on cholesterol and lipid metabolism to maintain their function (3,4). Interestingly, clinical studies have shown that patients taking cholesterol-lowering drugs may respond better to ICB (5), suggesting a potential way to target Tregs and improve outcomes.</p> <p>Project Overview</p> <p>This project aims to understand how altering lipid metabolism, particularly cholesterol, can reduce Treg-mediated immune suppression and enhance the body's immune response to cancer.</p> <p>Aims</p> <ul style="list-style-type: none"> • Investigate how cholesterol-lowering drugs affect Treg survival, function, and gene expression. • Assess how changes in lipid metabolism alter Treg suppression of anti-tumour T cells. • Test whether combining metabolic therapies with ICB improves tumour control in mouse models of lung cancer and mesothelioma. • Analyse patient data to explore links between cholesterol metabolism genes, Treg signatures, and immunotherapy outcomes.

	Techniques <ul style="list-style-type: none"> • Cell culture • In vivo tumour models • T cell tumour co-culture assays • Flow cytometry • Immunohistochemistry and immunofluorescence • Lipidomics • Genomics (RNA sequencing)
	Outcomes <ul style="list-style-type: none"> • Identify effective cholesterol lowering drugs to combine with immunotherapy to move into early phase clinical trials for mesothelioma and lung cancer patients. • Reveal immune metabolic programs that drive immunotherapy resistance • Identify T cell and lipid gene signatures that can be used as biomarkers to predict immunotherapy responses
	References <ol style="list-style-type: none"> 1. Baas P, Scherpereel A, Nowak AK, Fujimoto N, Peters S, Tsao AS, et al. First-line nivolumab plus ipilimumab in unresectable malignant pleural mesothelioma (CheckMate 743): a multicentre, randomised, open-label, phase 3 trial. <i>The Lancet</i>. 2021 Jan;397(10272):375–86. doi:10.1016/S0140-6736(20)32714-8 2. Hellmann MD, Paz-Ares L, Caro RB, Zurawski B, Kim SW, Costa EC, et al. Nivolumab plus Ipilimumab in Advanced Non–Small-Cell Lung Cancer. <i>New England Journal of Medicine</i>. 2019 Nov 21;381(21):2020–31. doi: 10.1056/NEJMoa1910231 3. Kempkes RWM, Joosten I, Koenen HJPM, He X. Metabolic Pathways Involved in Regulatory T Cell Functionality. <i>Front Immunol</i>. 2019 Dec 3;10:2839. Doi: 10.3389/fimmu.2019.02839 4. Principe N, Kidman J, Goh S, Tilsed CM, Fisher SA, et. al.,. Tumor Infiltrating Effector Memory Antigen-Specific CD8+ T Cells Predict Response to Immune Checkpoint Therapy. <i>Front Immunol</i>. 2020 Nov 12;11:584423. doi: 10.3389/fimmu.2020.584423 5. Cantini L, Pecci F, Hurkmans DP, Belderbos RA, Lanese A, Copparoni C, et al. High-intensity statins are associated with improved clinical activity of PD-1 inhibitors in malignant pleural mesothelioma and advanced non-small cell lung cancer patients. <i>European Journal of Cancer</i>. 2021 Feb 1;144:41–8. doi: 10.1016/j.ejca.2020.10.031

Research Project Proposal 2026

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Project title:	Precision immunotherapy for childhood cancers	
Project location:	The Kids Research Institute Australia, Perth Children Hospital	
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Immunotherapy—treatments that empower the immune system to fight cancer—has revolutionised cancer care for adults. But in children, these therapies often fall short. Why? Because they were developed based on adult immune systems, which function very differently from those of young patients.</p> <p>To address this, our team has developed the world’s first age-specific cancer models that closely mimic how a child’s immune system responds to cancer¹. These cutting-edge models have revealed that while children's T cells (a key type of cancer-fighting immune cell) launch an explosive early attack, they quickly burn out—losing the ability to control the tumour over time.</p> <p>This project will test new combination immunotherapies specifically designed to work with the developing immune systems of children. The goal is to reduce T cell exhaustion and improve long-term tumour control in paediatric cancers.</p>	
	<p>Design</p> <p>You will conduct preclinical testing of combination immunotherapy in mouse models of paediatric cancer. This will involve:</p> <ul style="list-style-type: none"> • Administering immunotherapy drugs to mice • Monitoring tumour growth and treatment response • Collecting and analysing tissue samples • Profiling immune cell activity and function 	

	<p>As part of a collaborative research team of cancer biologists, immunologists, oncologists, and bioinformaticians, you will gain hands-on experience in:</p> <ul style="list-style-type: none"> • Full Spectral Flow Cytometry & high-dimensional immune data analysis • RNA extraction and sample preparation for sequencing • Animal handling and live animal imaging • Tissue culture and basic molecular biology assays <p>You will work closely with an experienced postdoctoral researcher and skilled research assistants, who will provide direct training and supervision throughout the project.</p> <p>Outcomes</p> <p>This project will determine whether age-tailored combination immunotherapy can improve tumour control and reduce T cell exhaustion in paediatric cancer models. Your findings could directly contribute to the development of safer, more effective treatments for children with cancer.</p>
	<p>References</p> <p>Omar Elaskalani, Zahra Abbas., Sébastien Malinge, et al., . <i>Age-dependent tumor immune interactions underlie immunotherapy response in pediatric cancer</i>. <i>BioRxiv</i> (2025). https://doi.org:https://doi.org/10.1101/2025.07.16.663652</p>

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBC Email:
Project title:	Blood biomarkers and novel treatment strategies for Alzheimer's and rare childhood dementia
Project location:	Level 2, Sarich and Patricia Neuroscience Research Institute, QEII Campus, RR Block 8 Verdun street, Nedlands, Western Australia
Project Description: Aims, Design, Techniques, Outcomes, References	<div> Aims <ul style="list-style-type: none"> Understanding pathomechanisms in Alzheimer's and childhood dementia using cell and animal models Identify protein and gene biomarkers in blood to predict cognitive decline in Alzheimer's and childhood dementia Develop and characterize antibodies and assays for measuring biomarkers in Alzheimer's and childhood dementia Investigate novel treatment strategies including small molecules, peptides and gene therapies for Alzheimer's and childhood dementia </div> <div> Design <ul style="list-style-type: none"> Wet Lab based experimental design Data analysis experiment design </div> <div> Techniques <ul style="list-style-type: none"> Cell models for Alzheimer's and childhood dementia Animal models (worm) for Alzheimer's disease Biomarker assays Western blotting and immuno-techniques Standard biochemical assays </div>

	<p>Outcomes</p> <ul style="list-style-type: none"> • Manuscripts • Conference presentations • Conference abstracts and posters
	<p>References</p> <ul style="list-style-type: none"> • Bharadwaj P, Solomon T, Sahoo BR, Ignasiak K, Gaskin S, Rowles J, et al. Amylin and beta amyloid proteins interact to form amorphous heterocomplexes with enhanced toxicity in neuronal cells. <i>Sci Rep</i>. 2020;10(1):10356. • Dharmaraj GL, Arigo FD, Young KA, Martins R, Mancera RL, Bharadwaj P. Novel Amylin Analogues Reduce Amyloid-beta Cross-Seeding Aggregation and Neurotoxicity. <i>J Alzheimers Dis</i>. 2022;87(1):373-90. • Porter T, Bharadwaj P, Groth D, Paxman A, Laws SM, Martins RN, et al. The Effects of Latrepirdine on Amyloid-beta Aggregation and Toxicity. <i>Journal of Alzheimer's disease : JAD</i>. 2016;50(3):895-905. • Taddei K, Laws SM, Verdile G, Munns S, D'Costa K, Harvey AR, et al. Novel phage peptides attenuate beta amyloid-42 catalysed hydrogen peroxide production and associated neurotoxicity. <i>Neurobiol Aging</i>. 2010;31(2):203-14. • Bharadwaj PR, Verdile G, Barr RK, Gupta V, Steele JW, Lachenmayer ML, et al. Latrepirdine (dimebon) enhances autophagy and reduces intracellular GFP-Abeta42 levels in yeast. <i>Journal of Alzheimer's disease : JAD</i>. 2012;32(4):949-67. • Mputhia Z, Hone E, Tripathi T, Sargeant T, Martins R, Bharadwaj P. Autophagy Modulation as a Treatment of Amyloid Diseases. <i>Molecules</i>. 2019;24(18). • Belyaev ND, Kellett KA, Beckett C, Makova NZ, Revett TJ, Nalivaeva NN, et al. The transcriptionally active amyloid precursor protein (APP) intracellular domain is preferentially produced from the 695 isoform of APP in a beta-secretase-dependent pathway. <i>J Biol Chem</i>. 2010;285(53):41443-54. • Griffin EF, Scopel SE, Stephen CA, Holzhauer AC, Vaji MA, Tuckey RA, et al. ApoE-associated modulation of neuroprotection from Abeta-mediated neurodegeneration in transgenic <i>Caenorhabditis elegans</i>. <i>Dis Model Mech</i>. 2019;12(2).

Research Project Proposal 2026

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Project title:	Understanding commensal-pathogen interactions using <u>an</u> upper airway epithelial cell model
Project location:	QEII, L block Level 2
Project Description: Aims, Design, Techniques, Outcomes, References	<p>The <i>microbiome</i> has emerged as a critical frontier in the treatment and prevention of human disease. Current strategies include the use of probiotics such as <i>Lactobacillus</i> and <i>Bifidobacterium</i> to support gut health, as well as fecal microbiota transplants to treat severe, antibiotic-resistant <i>Clostridium difficile</i> infections. While these approaches have shown success, the development of microbiome-based therapies for extra-intestinal sites remains at an early stage. In this project, we will apply a physiologically relevant in vitro system that mimics the respiratory environment to investigate how commensal bacteria inhibit the growth of pneumonia-causing pathogens. Specifically, we will use a primary nasal epithelial model, where cells differentiated at an air–liquid interface for four weeks develop cilia and secrete mucus, thereby recapitulating the natural airway barrier. Using this system, we will explore how protective commensal strains interact with both host respiratory cells and pathogenic bacteria, including analysis of transcriptional responses in both the host and microbes. This work will provide mechanistic insight into commensal–pathogen–host interactions and inform novel strategies to prevent respiratory infections.</p> <p>Aims & Research Questions</p> <ul style="list-style-type: none"> • What biomechanisms govern the interaction between protective commensals, pathogens, and human respiratory epithelial cells? • How do host epithelial cells and commensal bacteria respond to colonization, as revealed by dual RNA sequencing? <p>Study Design</p> <ul style="list-style-type: none"> • Model development: Differentiate primary nasal epithelial cells at the air–liquid interface to form a physiologically relevant airway barrier with cilia and mucus production. • Experimental groups: <ul style="list-style-type: none"> ○ Nasal epithelial cells alone (control). ○ Epithelial cells colonized with commensal bacteria. ○ Epithelial cells infected with pneumonia-causing pathogens (<i>e.g.</i>, <i>Streptococcus pneumoniae</i>, <i>Haemophilus influenzae</i>). ○ Co-colonization model: commensal + pathogen.

	<ul style="list-style-type: none"> • Endpoints: <ul style="list-style-type: none"> ○ Pathogen colonization and growth inhibition (colony counts, microscopy). ○ Host epithelial integrity (TEER, cytokine assays). ○ Dual RNA-seq to assess transcriptional changes in both host and bacteria. • Data analysis: Comparative transcriptomic profiling to identify commensal-driven protective mechanisms and host immune pathways engaged during colonization. <p>Techniques</p> <ul style="list-style-type: none"> • Microbial culture and co-culture assays • Primary nasal cell culture at air–liquid interface • Quantitative real-time PCR • RNA isolation and RNA sequencing • Bioinformatics and transcriptional data analysis <p>Expected Outcomes</p> <ul style="list-style-type: none"> • Characterization of host epithelial responses (e.g., barrier reinforcement, cytokine/chemokine signaling) induced by commensal colonization. • Insights into bacterial gene expression programs that underpin commensal–pathogen competition in the airway niche. • Discovery of molecular mechanisms that could be leveraged for microbiome-based prophylactic or therapeutic strategies in respiratory disease. • Establishment of a robust <i>in vitro</i> nasal epithelial model as a platform for future translational studies.
	<p>References</p> <ul style="list-style-type: none"> • Yu K, Tenaglia V, Chua EG, Haines R, Bahal G, Nicol MP, Bahal RK. Interactions between bacteria in the human nasopharynx: a scoping review. <i>Lancet Microbe</i>. 2025 Jul;6(7). • Claassen-Weitz S, Xia Y, Workman L, Hannan L, Gardner-Lubbe S, Mwaikono KS et al. The nasopharyngeal microbiome in South African children with lower respiratory tract infection: a nested case-control study of the Drakenstein Child Health Study. 2025 <i>Clinical Infectious Diseases</i> 2025 Apr 17. • Claassen-Weitz, S., Gardner-Lubbe, S., Xia, Y., Mwaikono, K. S., Mounaud, S. H., Nierman, W. C., Workman, L., Zar, H. J., & Nicol, M. P. (2023). Succession and determinants of the early life nasopharyngeal microbiota in a South African birth cohort. <i>Microbiome</i>, 11(1), [127].

Research Project Proposal 2026

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Project title:	Defining the Metabolic Requirements of <i>Dolosigranulum pigrum</i> to Enable Prebiotic Development
Project location:	QEII, L block Level 2
Project Description: Aims, Design, Techniques, Outcomes, References	<p>The primary aim of this project is to define the metabolic requirements of <i>Dolosigranulum pigrum</i> (Dp), a commensal bacterium associated with respiratory health, in order to inform the rational development of a prebiotic that promotes its growth. Specifically, the project will:</p> <ol style="list-style-type: none"> 1. Integrate multiple existing datasets to generate a comprehensive prediction of Dp metabolic needs. 2. Experimentally validate these predictions using chemically defined media. 3. Identify specific metabolites or metabolic pathways that could be leveraged in a prebiotic formulation
	<p>Design</p> <p>The project is structured into two main phases:</p> <ul style="list-style-type: none"> • Data integration and in silico prediction: Existing datasets including genome-based auxotrophy predictions, metabolomic profiles from Dp culture supernatants, and Biolog phenotypic microarray data will be integrated to develop a predictive model of Dp metabolic requirements. Cross-referencing of these datasets will identify candidate metabolites that Dp is likely unable to synthesise but can utilise for growth. • Experimental validation: Predicted requirements will be tested in vitro using a base chemically defined medium (CDM) supplemented with individual or combinations of predicted essential metabolites. Growth of Dp will be assessed under these conditions using spectrophotometric and/or CFU-based methods.

	<p>Techniques</p> <ul style="list-style-type: none"> • Genomic data mining and pathway analysis for auxotrophy prediction (e.g., KEGG, BioCyc) • Integration of metabolomics data using software tools such as MetaboAnalyst • Analysis of Biolog plate data to identify utilisable carbon/nitrogen sources • Preparation and manipulation of CDM formulations • Bacterial culture under aerobic/microaerophilic conditions • Growth quantification using OD600 measurements and viable counts • Data visualisation and statistical analysis <p>Outcomes</p> <p>The project is expected to:</p> <ul style="list-style-type: none"> • Define a reproducible, experimentally validated profile of essential metabolic requirements for Dp. • Identify candidate metabolite(s) for prebiotic inclusion to selectively promote Dp growth in vitro. • Contribute foundational knowledge for future development of synbiotic therapies targeting respiratory health. <p>This project will also provide the student with experience in microbiology, metabolomics, data integration, experimental design, and translational research relevant to host-microbiome interactions.</p>
	<p>References</p> <ul style="list-style-type: none"> • Yu K, Tenaglia V, Chua EG, Haines R, Bahal G, Nicol MP, Bahal RK. Interactions between bacteria in the human nasopharynx: a scoping review. <i>Lancet Microbe</i>. 2025 Jul;6(7). • Claassen-Weitz S, Xia Y, Workman L, Hannan L, Gardner-Lubbe S, Mwaikono KS et al. The nasopharyngeal microbiome in South African children with lower respiratory tract infection: a nested case-control study of the Drakenstein Child Health Study. <i>2025 Clinical Infectious Diseases</i> 2025 Apr 17. • Claassen-Weitz, S., Gardner-Lubbe, S., Xia, Y., Mwaikono, K. S., Mounaud, S. H., Nierman, W. C., Workman, L., Zar, H. J., & Nicol, M. P. (2023). Succession and determinants of the early life nasopharyngeal microbiota in a South African birth cohort. <i>Microbiome</i>, 11(1), [127].

Research Project Proposal 2026

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Project title:	An investigation of the effects <i>Medulla Tetrapanacis</i> on milk production in cultured mammary epithelial cells
Project location:	M Block, QEII MC
Project Description: Aims, Design, Techniques, Outcomes, References	Aims: Breast milk is considered as an ideal diet for infants. World Health Organization (WHO) recommend children can be exclusively breastfed for the first 6 months of life then gradually introduce to appropriate foods and continue to breastfeed for up to 2 years or beyond. There are numerous reasons for early cessation of breastfeeding, such as insufficient milk supply, lack of sleep, latch problems and mastitis. Research undertaken in many countries including Australia have demonstrated substantial increase with the use of herbal medicine amongst lactating mothers with aim to increase milk production and manage mastitis. Our group recently demonstrated that <i>Medulla Tetrapanacis</i> (MT), an herb that was commonly use among breastfeeding mother in Asia, could suppress mastitis via its anti-inflammatory, anti-oxidative and immunomodulatory properties. Our work also identified the bioactive components in the MT responsible for these pharmacological actions [1, 2]. However, the effects of MT on milk production have not been studied in detail. In this study, we aim to investigate the effect of MT on milk production using an in vitro model.
	Design/ Techniques: The effects of MT on milk production pathway will be studied using an in vitro model of blood-milk barrier with cultured mammary epithelial cells. (HMECs). MECs will be cultured on a cell culture insert with a collagen gel in the presence of pituitary extract and dexamethasone to induce milk production and tight junction (TJ) formation. The secretion of the major milk components, such as β -casein, lactose and triglyceride will be measured using ELISA and Western blotting. Tight junction formation and the integrity of the blood-milk barrier will be examined.
	Outcomes: The results of this work will explain the pharmacological actions MT on milk production and provide scientific evidence to support its use for breastfeeding.
	References 1. Kwok CTK, Y Hu, Tsoi B, Wong F, Hau PT, Tam WT, Mok KW, Kwan YW, Leung GPH, Lee SMY, Li JJ, Chow FWN, Seto SW* (2025) Medulla Tetrapanacis water extract ameliorates mastitis by suppressing bacterial internalization and inflammation via MAPKs signaling in vitro and in vivo. Food Frontiers DOI: 10.1002/fft2.476

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| | <p>2. Kwok CTK, Chow FWN, Cheung KYC, Zhang XY, Mok DKW, Kwan YW, Chan GHH, Leung GPH, Cheung KW, Lee SMY, Wang N, Li J, Seto SW* (2023) Medulla Tetrapanax Water Extract Alleviates Inflammation and Infection by Regulating Macrophage Polarization Through MAPK Signalling Pathway. <i>Inflammopharmacology</i> DOI:10.1007/s10787-023-01266-1.</p> |
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Research Project Proposal 2026

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Project title:	An investigation of the effects of sclerostin (SOST) on macrophage migration and infiltration
Project location:	M Block, QEII MC
Project Description: Aims, Design, Techniques, Outcomes, References	Aims Sclerostin (SOST) is a glycoprotein secreted by osteocytes that acts as a negative regulator of bone formation by reducing osteoblast differentiation. While SOST is commonly referred to as a bone-specific protein, our previous works demonstrated the role of SOST in the development and progression of vascular diseases [1]. Interestingly, our recent work identified macrophages as a novel source of SOST, highlighting its potential role in inflammatory diseases and other non-skeletal disorders [2]. The aim of this study is (1) to investigate the effects of SOST on macrophage migration and infiltration and (2) to study the underlying molecular mechanisms involved.
	Design/ Techniques The effects of SOST will be studied using both RAW264.7 cells and primary peritoneal macrophage from C56BL/6 mice. Wound healing assay and Transwell assay will be used to evaluate migration and infiltration of the SOST-stimulated macrophages. The signalling mechanisms and molecular changes in the SOST-stimulated macrophage will be examined using Western blotting and ELISA.
	Outcomes This study explores an unreported role of SOST, emphasizing its potential as a therapeutic target beyond bone health.
	References <ol style="list-style-type: none"> 1. Krishna SM, Seto SW, Jose RJ, Li J, Morton SK, Biros E, Wang Y, Nsengiyumva V, Lindeman JH, Loots GG, Rush CM, Craig JM, Golledge J (2017) Wnt Signaling Pathway Inhibitor Sclerostin Inhibits Angiotensin II-Induced Aortic Aneurysm and Atherosclerosis. <i>Arterioscler Thromb Vasc Biol.</i> 37(3): 553-566 2. Kwok CTK, Wong CC, Li JJ, Kwan YW, Leung GPH, Tsoi B, Chow FWN, Seto SW (2025) Lipopolysaccharide (LPS) induces sclerostin secretion by extracellular vesicle via TLR4/miR-92a-3p/PTEN/NF-κB signalling pathway in murine macrophage. <i>Inflammation Research</i> 74(1):27

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Dr Mitali Sarkar-Tyson Email: mitali.sarkar-tyson@uwa.edu.au	
Project title:	Characterisation of Induced Pluripotent Stem Cell Lines Generated from Patients with Congenital Aniridia	
Project location:	Lions Eye Institute, Centre for Ophthalmology and Visual Science, UWA Medical School	
Project Description: Aims, Design, Techniques, Outcomes, References	Background: Heterozygous mutations in the <i>PAX6</i> gene cause congenital aniridia (the absence of the coloured part of the eye) and other eye conditions including corneal cloudiness that can lead to blindness [1]. Our team at the Lions Eye Institute has established the first biobank of patients with <i>PAX6</i> mutations in Western Australia and generated induced pluripotent stem cell (iPSC) lines from their skin fibroblasts.	
	Aims: This study aims to characterise the iPSC lines for future use of these stem cells as a disease model for congenital aniridia and therapeutic development.	
	Design: This is a basic science laboratory study using tissue donated by patients with congenital aniridia, in accordance with approved ethical protocols.	
	Techniques: Techniques that will be used in this project include cell culture, mycoplasma screening, direct sequencing, digital karyotyping, immunohistochemistry, and quantitative real-time PCR [2].	
	Outcomes: The results will be published in the Stem Cell Research journal and presented at ophthalmology, genetics, and stem cell conferences.	
	References [1] Landsend ECS, et al. Surv Ophthalmol. 2021;66:1031-1050. [2] Zhang D, et al. Stem Cell Res. 2025;82:103621.	

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBC Email:
Project title:	Find new cures for childhood leukaemia
Project location:	The Kids Research Institute Australia – Cancer Centre
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Leukaemia is the most common type of cancer in children. Despite remarkable advances in treatment over the decades, leukaemia remains the second cause of death by Cancer for Australian children, mostly due to treatment-related toxicity and relapse. Current treatments have reached their maximum potential highlighting the need for safer and better therapies.</p> <p>Our group is focused on finding new key vulnerabilities in the leukaemia cells to develop novel and less toxic targeted therapies. We are also exploring the impact of the microenvironment surrounding the leukaemia cells in order to design new immune-based therapies. To achieve this, we are using primary patient samples from which we developed leukaemia cell lines uniquely available in our lab, and sophisticated and clinically relevant models named Patient-derived Xenografts (PDX); We also recently generated new immune competent models of childhood leukaemia.</p> <p>Aims: We recently screen >7000 drugs and are now planning to validate our main candidates through the following aims:</p> <ul style="list-style-type: none"> - Test the efficacy of a new targeted therapy as single agent - Validate specific and efficacy of inhibitor - Assess potential synergy with standard of care (SOC) treatments <p>Design:</p> <ul style="list-style-type: none"> - Dose/response curves in up to human and murine leukaemia cell lines (AlamarBlue assay after 72h incubation) - Assess impact of the drug on cell survival and cell cycle (or other cellular function) - Synergistic treatments with 9 standard-of-care agents and 4 targeted therapies (AlamarBlue assay) <p>Techniques:</p> <ul style="list-style-type: none"> - Tissue culture, drug screen - Molecular biology (CRISPR/Cas9, transduction...), - Flow cytometry (survival, cell cycle) - Animal work (tissue preparation and drug testing),

	<p>Outcomes:</p> <p>Ultimately, our goal is to test novel therapies that target key weaknesses of the leukaemia cells, alone or in combination with immunotherapy to develop new synergistic approaches. If successful, this will provide key information to improve prevention, diagnosis, long-term survival and quality of care for all children with leukaemia.</p>
	<p>References:</p> <ul style="list-style-type: none"> - Carey-Smith SK*, Simad MH*, Panchal K, Aya-Bonilla C, Smolders H, Lin S, Armitage JD, Nguyen VT, Bentley KT, Ford J, Singh S, Oommen J, Laurent AP, Mercher T, Crispino JD, Montgomery AP, Kassiou M, Besson T, Deau E, Meijer E, Cheung LCC, Kotecha RS and Malinge S. Efficacy of DYRK1A inhibitors in novel models of Down syndrome acute lymphoblastic leukemia. <i>Haematologica</i>. 2024 Jul 1;109(7):2309-2315. - Laurent AP, Siret A, Ignacimouttou C, Panchal K, Diop MB, Jenni S, Tsai YC, Ross-Weil D, Aid Z, Prade N, Plassard D, Pierron G, Daudigeos-Dubus E, Lecluse Y, Droin N, Bornhauser B, Cheung L, Crispino JD, Gaudry M, Bernard OA, Macintyre E, Barin Bonnigal C, Kotecha R, Goeorger B, Ballerini P, Bourquin JP, Delabesse E, Mercher T and Malinge S. Constitutive activation of RAS/MAPK pathway cooperates with trisomy 21 and is therapeutically exploitable in Down syndrome B-cell Leukemia. <i>Clinical Cancer Research</i>. 2020 Jul 1;26(13):3307-3318.

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Dr Charlene Kahler Email: charlene.kahler@uwa.edu.au	
Project title:	Toxic tactics: Investigating colibactin's impact on <i>S. aureus</i> survival in blood culture models	
Project location:	King Edward Memorial Hospital	
Project Description: Aims, Design, Techniques, Outcomes, References	Aims Recent work on polymicrobial sepsis (infections involving more than one pathogen) have shown that co-culture of <i>E. coli</i> and <i>S. aureus</i> can lead to suppression of <i>S. aureus</i> growth. This suppression has been linked to colibactin, a genotoxin produced by some strains of <i>E. coli</i> which has been previously shown to inhibit <i>S. aureus</i> growth in wound infections. However, the role of colibactin in bloodstream infections, particularly in the context of diagnostic blood cultures remains unclear. The project aims to investigate the role of colibactin in the suppression of <i>Staphylococcus aureus</i> growth under blood culture conditions, with a focus on its relevance to diagnostic accuracy in bloodstream infections. Specific objectives are: <ol style="list-style-type: none"> 1. To screen a library of clinical <i>Escherichia coli</i> isolates for colibactin gene expression. 2. To assess whether co-culture with colibactin-positive <i>E. coli</i> suppresses <i>S. aureus</i> growth in healthy human blood samples. 3. To determine if the addition of a colibactin inhibitor can prevent <i>S. aureus</i> suppression in co-culture. 	
	Design This is a laboratory-based experimental project conducted under PC2 conditions in the Clinical Perinatal Research Laboratories at King Edward Memorial Hospital. The study will use an in vitro co-culture model of <i>E. coli</i> and <i>S. aureus</i> in freshly collected human blood samples, sourced from healthy adult volunteers. The project will proceed in three phases: <ol style="list-style-type: none"> 1. Isolate Screening <ul style="list-style-type: none"> o An existing library of clinical <i>E. coli</i> isolates will be cultured. 	

	<ul style="list-style-type: none"> ○ Isolates will be screened for the presence and expression of colibactin genes using RT-qPCR. ○ Colibactin-positive and colibactin-negative isolates will be selected for further experiments. <p>2. Co-culture Experiments</p> <ul style="list-style-type: none"> ○ Blood samples will be inoculated with <i>S. aureus</i> alone, <i>E. coli</i> alone, and in co-culture (colibactin-positive and negative <i>E. coli</i> with <i>S. aureus</i>). ○ Bacterial growth will be quantified over time using traditional plating and colony-forming unit (CFU) enumeration. ○ Comparative growth curves will be generated to assess suppression of <i>S. aureus</i> in the presence of colibactin-positive isolates. <p>3. Inhibitor Studies</p> <ul style="list-style-type: none"> ○ Co-cultures of <i>S. aureus</i> with colibactin-positive <i>E. coli</i> will be repeated in the presence of a colibactin inhibitor. ○ The extent of <i>S. aureus</i> recovery will be measured to evaluate the effectiveness of colibactin inhibition in preventing growth suppression. <p>Controls will include:</p> <ul style="list-style-type: none"> • Monocultures of <i>S. aureus</i> and <i>E. coli</i>. • Heat-killed bacterial controls to exclude nonspecific effects. • Technical replicates to ensure reproducibility. <p>Data will be statistically analysed to compare growth dynamics between conditions and to correlate colibactin gene expression with <i>S. aureus</i> suppression.</p>
	<p>Techniques</p> <p>This project will involve:</p> <ul style="list-style-type: none"> • Assay development and optimisation for co-culture conditions. • Microbiological methods including bacterial culture and quantification. • Gene expression analysis of colibactin using RT-qPCR. • Data analysis including statistical evaluation of growth and gene expression results.
	<p>Outcomes</p> <p>This project will:</p> <ul style="list-style-type: none"> • Establish whether colibactin production by <i>E. coli</i> contributes to <i>S. aureus</i> suppression in blood cultures. • Provide insight into the impact of colibactin on the accuracy of pathogen detection in polymicrobial bloodstream infections. • Evaluate whether colibactin inhibition can restore <i>S. aureus</i> growth, with implications for improving diagnostic microbiology practices.

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: Valerie Verhasselt Email: Valerie.verhasselt@uwa.edu.au .
Project title:	From First Feed to First Barrier: The Impact of Colostrum on Skin Development
Project location:	The KIDS Research Institute Australia
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Project Outline:</p> <p>This master project is part of a PhD research project led by Nivedithaa, investigating the role of colostrum in promoting healthy skin development and preventing early-life skin diseases such as allergies and infections. The overarching aim of the project is to provide foundational evidence that colostrum intake supports skin maturation through its rich composition of bioactive compounds. Within this broader context, the student will focus on one key objective: the analysis of skin histology in a mouse model. This will involve comparing skin architecture, inflammation, permeability and HF morphogenesis between colostrum-deprived and control mice. The findings from this objective will contribute to our understanding of the biological mechanisms through which colostrum supports immune and barrier function in early life.</p> <p>Background:</p> <p><u>Diet at birth may be critical for skin development:</u> Colostrum is the very first milk produced by the mammary gland and is the physiological food for the first 3 days of a newborn. Colostrum is often called “Liquid gold” due to its yellow colour and its composition that is rich in bioactive compounds. Compared to milk from more advanced stages of lactation (mature milk) and formula milk, colostrum is extremely rich in growth factors (Epidermal Growth Factor (EGF), Insulin Growth Factor (IGF), transforming growth factor (TGF), Fibroblast Growth Factor (FGF), platelet derived-growth factor (PDGF)), vitamin A, human milk oligosaccharides, antibodies, and microbiota-shaping molecules such as lactoferrin, SCFA and lipids (Ballard and Morrow 2013, Szyller, Antosz et al. 2024), suggesting it may be crucial for newborn health and skin development at early stages of life. Statistics from the World Health Organization (WHO) reveal that globally, one in three infants do not receive the full dose of colostrum. There is strong evidence that non-optimal colostrum feeding is associated with increased neonatal mortality due to infectious disease in low-middle-income countries (Debes, Kohli et al. 2013, Group 2016). Some data exist on colostrum impact</p>

	<p>on skin by topical application or in in vitro experiments. Due to its use throughout history, It is speculated that the physiological role of topical application of colostrum is for the prevention and treatment of nipple wounds(Witkowska-Zimny, Kamińska-El-Hassan et al. 2019). The topical use of colostrum showed benefits for the skin by hydrating, reducing inflammation, enhancing healing, helping in treating skin condition such acne, psoriasis, dermatitis, or skin ageing. In-vitro studies show that colostrum promotes cell differentiation (Kovacs, Maresca et al. 2020), increases fibroblast and keratinocyte levels(Amiot, Germain et al. 2004, Torre, Jeusette et al. 2006, Zava, Barello et al. 2009) (Hewitt, Mros et al. 2019), skin ageing and damage(Han, Kim et al. 2022), and reduces inflammation (Buescher and McIlheran 1988). Additionally, colostrum reduces the inflammatory response of cells to certain inflammatory stimuli (Rodríguez-Camejo, Puyol et al. 2023).</p> <p>There are also studies in adults suggesting that oral supplementation of bovine colostrum and its compounds such as glycine and lactoferrin reduce skin inflammation(Hartog, Leenders et al. 2007), heal wounds, help tissue growth, enhance immune regulation and encourage differentiation and proliferation of epidermal cells (Vollmer, West et al. 2018).</p> <p>Currently, studies on the impact of lack of optimal colostrum feeding at birth in the long term, including skin development is very limited. Nishimura et al, developed a mouse model that studied the impact of colostrum deprivation on healthy development(Nishimura 1953). Our Centre (LRF-CIBF) used this mouse model of colostrum deprivation to address the importance of colostrum and found it played a key role in gut immune ontogeny(Rekima, van den Elsen et al. 2024). We also observed major macroscopic skin alteration in young mice lacking colostrum at birth including hair growth retardation and scaly skin. Using the same mouse model, H Nishimura et al (Nishimura 1953), also observed abnormal skin development in colostrum-deprived mice, including an increased number of mast cells and macrophages. These preclinical data suggest that colostrum intake at birth may be critical for healthy skin development. Although it is highly unlikely that one or a specific colostrum compound may influence skin development, we hypothesise that a cocktail of the right bioactive factors may promote the release of beneficial skin metabolites which are able to circulate to the skin and promote skin health. Hence Nivedithaa's PhD project will involve looking at the impact and studying the underlying mechanisms in which intake of colostrum and its bioactive compounds during early life influences skin development.</p> <p>Aim: To investigate the role of colostrum in skin development</p>
	<p>Design</p> <p>Skin development will be compared in colostrum-deprived mice (nursed from birth by dams at an advanced stage of lactation) with control mice. Skin histology will be analysed at different time points in both groups to define the role of colostrum in skin development.</p>

	<p>Techniques</p> <p><u>Mouse model of colostrum deprivation:</u></p> <p>The student will use a mouse model of colostrum deprivation that allows us to determine the causal role of early life diet in the guidance of healthy skin development. In this model, one group of pups will be removed from dams immediately after birth and cross-fostered to mothers that delivered 9 days previously and do not produce colostrum anymore (no colostrum/mature milk group). Another group will be cross-fostered to mothers that delivered less than 1 hour before and will receive colostrum followed by mature milk (control group). Analysis will be performed at days 1-3, days 7- 14 (pre-weaning stage) and weeks 5-7 (post-weaned, adults). The time of analysis was selected to address the role of colostrum in neonatal, infant, and adult stage.</p> <p><u>Skin histology (Cell structure and integrity):</u> Hematoxylin eosin staining, Mason Trichrome stain, and toluidine blue staining on paraformaldehyde-fixed skin tissue will be performed to analyse the development of the stratum corneum, collagen density and monitor mast cells infiltration, respectively. Molecules that are important for skin integrity including filaggrin and tight junction protein expression will be determined by immunofluorescence and proteomics of snap frozen tissue.</p> <p><u>Skin Permeability:</u> Permeability of the skin will be tested using fluorochrome such as (FITC-dextran) using a Franz diffusion cell.</p> <p>Outcomes</p> <p>This project will be the first to provide evidence for a causal role of colostrum in healthy skin development, and provide the knowledge required to promote colostrum feeding through investments in breastfeeding support. This may be critically important since, globally, one in three newborns is not receiving the full dose of colostrum. It may also lead to the discovery of new compounds that are developmentally adapted for the prevention and treatment of diseases in children such allergies and infection.</p>
	<p>References</p> <ol style="list-style-type: none"> 1. Ballard O, Morrow AL. Human Milk Composition: Nutrients and Bioactive Factors. <i>Pediatric Clinics of North America</i>. 2013;60(1):49-74. doi:https://doi.org/10.1016/j.pcl.2012.10.002 2. Szyller H, Antosz K, Batko J, et al. Bioactive Components of Human Milk and Their Impact on Child's Health and Development, Literature Review. <i>Nutrients</i>. 2024;16(10):1487. Debes, Kohli et al. 2013 3. Group NS. Timing of initiation, patterns of breastfeeding, and infant survival: prospective analysis of pooled data from three randomised trials. <i>Lancet Glob Health</i>. Apr 2016;4(4):e266-75. doi:10.1016/S2214-109X(16)00040-1 4. Witkowska-Zimny M, Kamińska-El-Hassan E, Wróbel E. Milk Therapy: Unexpected Uses for Human Breast Milk. <i>Nutrients</i>. Apr 26

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Research Project Proposal 2026

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Project title:	Precision medicine and disease modelling for genetic diagnosis of children in WA	
Project location:		
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims: Today clinical genetic testing provides a diagnosis for only 30-50% of children with a genetic disease. This means for the majority of children, a genetic variant is identified, but the clinical significance of the variant remains undetermined. Currently, diagnosis takes on average 5-30 years, if achieved at all and in the interim the opportunity for early, effective, and appropriate intervention is often missed.</p> <p>The aim of this study is to provide a diagnosis for children in WA with a suspected genetic condition.</p>	
	<p>Design: We use CRISPR gene editing to modify induced pluripotent stem cells creating isogenic cell pairs. Next, patient-specific disease is modelled in a dish in the laboratory. This is possible as stem cells can be stimulated to form many different cell types that are relevant to the patient's disease including heart, nerve, lung, kidney, or other. Healthy and genetic variant cells are compared to determine changes in cell function and inform the molecular and cellular pathways affected.</p>	
	<p>Techniques: Techniques to be applied in the project include: human stem cell culture; CRISPR homology directed repair gene editing; genomic DNA targeted amplicon sequencing; PCR; RT-PCR; qPCR; karyotyping; gDNA preparation; RNA preparation; protein techniques (western blot, flow cytometry, immunofluorescent staining) and other techniques specific to the patient genetic variant.</p>	
	<p>Outcomes: The project will deliver an informative and accurate analysis for genetic variants identified in WA children. The data generated will be utilised toward patient diagnosis and selection of best clinical treatments. A diagnosis is powerful as it enables future planning and access to early interventions or treatments that will improve life trajectory.</p>	
	<p>References</p> <p>Shaw NC, Chen K, <i>et al.</i> (2024) <i>Molecular Autism</i>, 15(1):42.</p> <p>Farley KO, <i>et al.</i> (2024) <i>HGG Advances</i>, 5(1):100257.</p> <p>Fear VS, <i>et al.</i> (2023) <i>Stem Cell Research & Therapy</i>, 14(1):345.</p> <p>Fear VS, <i>et al.</i> (2022) <i>Stem Cell Research & Therapy</i>, 13(1): 69.</p>	

Research Project Proposal 2026

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Project title:	Can feeding hearts help achieve ultra long organ preservation?
Project location:	HLRI WA Labs (Harry Perkins South Campus)
Project Description: Aims, Design, Techniques, Outcomes, References	Aims A method of extended heart preservation has the potential to make many more organs available by affording time for matching and long distance transport. Our group is working on the concept of using oxygen gas instead of fluid to nourish hearts. This is known as gas persufflation. Adding food (energy substrates) to persufflation may prolong storage duration by allowing replenishment of ATP stores. This project will compares the effect of adding glucose or amino acids to the gas persufflation of rodent hearts with the aim of determining the optimal supplements for gas stored hearts.
	Design The project will be conducted in our lab within Harry Perkins South Campus. The project will use 24 sprague dawley rats. We will secure ethics approval before the end of 2025 and hands on experiments are expected to take 8 weeks.
	Techniques Students will be taught to remove rodent hearts, run our existing models of gas persufflation and isolated heart functional assessment and some simple biochemical assays of heart tissue. The remaining time will be used for training, data analysis and thesis writing. Students will be fully supervised by our on-site team.
	Outcomes Previous honours students with our group have had their work presented at the International Society of Heart & Lung Transplant meeting in the USA as well as the Transplant Society of Australia and New Zealand Annual Meeting.

Research Project Proposal 2026

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Other supervisor/s if any (name and email address):	Name: Dr Kevin Trentino Email: kevin.trentino@health.wa.gov.au
Project title:	Vital Sign Trends as Predictors of Transfusion-Related Complications
Project location:	Royal Perth Hospital
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Aims</p> <p>Continuous monitoring of vital signs holds significant potential for advancing red cell transfusion research. While previous studies have explored the impact of red cell transfusions on vital signs at specific intervals, little research has focused on the effects of transfusion on high frequency, continuously monitored vital signs or their potential to predict transfusion events.</p> <p>Aims: To investigate whether changes in continuous vital signs (e.g., HR, RR, SpO2) can predict adverse events during transfusion.</p> <p>Design</p> <p>This will involve a retrospective observational analysis of all adult patients transfused while connected to the Health in a Virtual Environment (HIVE) system. The student will include data from transfusion records to flag patients experiencing a transfusion-related complication. Following this statistical modelling techniques will be used to analyse the potential of vital sign trends to detect transfusion-related complications.</p> <p>These data will be compared to the manual vital signs recorded before, during, and after transfusion by the ward staff.</p>
	<p>Techniques</p> <p>Real-time HIVE data, as well as clinical pathology data and transfusion outcomes will be collected. The time of transfusion and effects on clinical and pathology parameters measured. Statistical analyses will be undertaken. Prediction models for adverse transfusion-related events will be developed.</p>

	<p>Outcomes</p> <p>The main outcome studied will be transfusion-related complication as recorded by the hospital. These include:</p> <ol style="list-style-type: none"> 1. Febrile Non-Haemolytic Transfusion Reaction (FNHTR) 2. Allergic Reactions 3. Acute Haemolytic Transfusion Reaction (AHTR) 4. Transfusion-Transmitted Bacterial Infection (TTBI) 5. Transfusion-Related Acute Lung Injury (TRALI) <p>Transfusion-Associated Circulatory Overload (TACO)</p>
	<p>References</p> <p>Trentino KM, Sanfilippo FM, Leahy MF, Farmer SL, Mace H, Lloyd A, et al. Multivariable statistical models to predict red cell transfusion in elective surgery. Blood Transfus. 2022.</p> <p>Bowles T, Trentino KM, Lloyd A, Trentino L, Murray K, Thompson A, et al. Health in a virtual environment (hive): A novel continuous remote monitoring service for inpatient management. Healthcare2024.</p> <p>Bowles T, Trentino KM, Lloyd A, Trentino L, Jones G, Murray K, et al. Outcomes in patients receiving continuous monitoring of vital signs on general wards: A systematic review and meta-analysis of randomised controlled trials. Digit Health. 2024;10:20552076241288826.</p>

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBC Email:
Project title:	Investigating organelle dysfunction in idiopathic pulmonary fibrosis
Project location:	Level 5, Harry Perkins Institute of Medical Research (North)
Project Description: Aims, Design, Techniques, Outcomes, References	Background Idiopathic pulmonary fibrosis (IPF) is a progressive and debilitating lung disease characterized by the irreversible scarring of lung tissue, leading to significant respiratory impairment and a poor prognosis. Current treatments are limited to slowing disease progression rather than providing a cure, making it imperative to uncover the underlying mechanisms driving IPF. Recent studies have highlighted the roles of endoplasmic reticulum (ER) stress and mitochondrial dysfunction in the pathogenesis of IPF, particularly in the apoptosis of alveolar epithelial cells (AECs). The subsequent damage to these cells stimulates fibroblast activation and excessive extracellular matrix production, contributing to fibrosis and loss of lung function. Despite advancements in our understanding of IPF, the interplay between ER stress and mitochondrial dysfunction remains poorly defined.
	Aim This study aims to elucidate the interplay between ER stress and mitochondrial dysfunction in an in vitro model of IPF. Specifically, we will investigate the effects of cigarette smoke exposure—a significant risk factor for IPF—on ER and mitochondrial function.
	Objectives: <ol style="list-style-type: none"> 1. To evaluate the impact of cigarette smoke on ER and mitochondrial function. 2. To assess changes in gene expression and protein levels associated with ER stress, mitochondrial dysfunction and apoptosis. 3. To characterize cellular morphological changes following exposure to cigarette smoke.

	<p>Techniques</p> <p>Cell Culture; RNA Isolation; Quantitative PCR (qPCR); Western Blotting; Confocal and/or Live Cell Imaging; Image Analysis using ImageJ</p>
	<p>Outcomes</p> <p>Understanding the interplay between ER stress and mitochondrial dysfunction in the context of IPF may provide new insights into the mechanisms driving disease progression. This knowledge could lead to the identification of novel therapeutic targets aimed at mitigating the effects of ER and mitochondrial dysfunction, ultimately improving outcomes for patients with IPF.</p>
	<p>References</p> <ol style="list-style-type: none"> 1. Alysandratos KD <i>et al.</i> Patient-specific iPSCs carrying an SFTPC mutation reveal the intrinsic alveolar epithelial dysfunction at the inception of interstitial lung disease. <i>Cell Rep.</i> 2021 Aug 31;36(9):109636. doi: 10.1016/j.celrep.2021.109636. PMID: 34469722; PMCID: PMC8432578. 2. Jiang D <i>et al.</i> ATF4 Mediates Mitochondrial Unfolded Protein Response in Alveolar Epithelial Cells. <i>Am J Respir Cell Mol Biol.</i> 2020 Oct;63(4):478-489. doi: 10.1165/rcmb.2020-0107OC. PMID: 32551949; PMCID: PMC7528926.

Research Project Proposal 2026

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SBMS Coordinating supervisor Contact details (for non-SBMS supervisors)	Name: TBA Email: TBA
Project title:	Optimising a cryopreservation matrix for recovering core <i>Lactobacillus</i> spp. from frozen clinical samples
Project location:	Clinical Perinatal Research Laboratories, King Edward Memorial Hospital
Project Description: Aims, Design, Techniques, Outcomes, References	<p>Cold storage of clinical samples and microbiological cultures is a necessary part of research and is critical for effective sample biobanking. To try and maximise cell viability after freezing, a cryoprotectant (commonly glycerol or DMSO) is typically added to the sample before the initial freeze at a specific final concentration. However, the efficacy of commonly used cryoprotectants can vary widely depending on the bacterial species within a sample, a factor which becomes even more important when trying to preserve microbiologically complex samples for culturomic analyses or microbial transplant therapies.</p> <p>In the context of the vaginal microbiome, <i>Lactobacillus</i> spp. are without a doubt the most important species related to promotion of vaginal health¹. Although numerous studies have focused on preservation of cell viability of common <i>Lactobacillus</i> spp. used as oral probiotics and food supplements², relatively little information is available for the key vaginal species, <i>L. crispatus</i>, <i>L. gasseri</i>, <i>L. iners</i> and <i>L. jensenii</i>. Most recently, Yockey et al.³ reported high cell viability after almost one year of -80°C storage for <i>L. crispatus</i> and <i>L. jensenii</i> without use of any cryoprotectant. However, the impact of long term freezing on the single sample that was dominated by <i>L. iners</i> is unknown as the culture protocols employed are unlikely to have supported the growth of this highly fastidious microbe.</p> <p><i>L. iners</i> is an atypical <i>Lactobacillus</i> sp. in many aspects, but of high relevance to cryopreservation is the thin layer of peptidoglycan in its bacterial cell wall relative to other Gram positive organisms⁴. Our own attempts at culturing <i>L. iners</i> from even recently frozen clinical samples suggests that cells may be highly sensitive to -80°C storage, with 0% recovery rates despite qPCR indicating large titres present. Surprisingly, however, we have been able to recover the organism after multiple freeze-thaws without cryopreservatives from high titre microbiological broth cultures, suggesting the cells are most vulnerable within clinical samples.</p> <p>Considering that <i>L. iners</i>-dominated vaginal microbiomes are the most common community state type seen among women⁵ and that <i>L. iners</i> is associated with risk of transition to bacterial vaginosis⁶ and preterm birth⁷, being able to culture this organism to conduct thorough genomic analyses is</p>

	<p>of critical importance. It is only once we have generated these data from a sufficiently large isolate biobank with matched clinical information that we will be able to begin to uncover what specific traits within certain strains of <i>L. iners</i> are responsible for negative health outcomes. Once identified, these will facilitate the development of new diagnostics and interventions to reduce associated negative health outcomes.</p> <p>Design: Laboratory-based with short initial prospective recruitment of volunteers for sample acquisition.</p> <p>Sample size: 20 women, no inclusion or exclusion criteria.</p> <p>Sample collection: three self-collected mid-vaginal swabs for culture and molecular analyses.</p> <p><i>Lactobacillus</i> spp. culture with neat sample and following -80 frozen storage for 1 week in the presence of either Trehalose, Sucrose, Glycerol, or Skim Milk Powder at concentrations of 50, 100 and 150g/L.</p> <p>DNA extraction and qPCR identification of dominant <i>Lactobacillus</i> spp. phenotypes.</p> <p>Techniques:</p> <ul style="list-style-type: none"> - Laboratory: Microbiological culture, DNA extraction, and qPCR. - Data analysis: Basic statistical analysis of CFU counts across treatments. <p>Outcomes:</p> <p>Findings from this study will directly inform how future vaginal swab samples are stored for research sample biobanking to maximise recovery of all relevant <i>Lactobacillus</i> spp., including <i>L. iners</i>. Findings will also be of high relevance to biobanking of vaginal swab eluates for use in vaginal microbial transplantation protocols.</p>
	<p>References:</p> <ol style="list-style-type: none"> 1. Holdcroft AM, Ireland DJ, Payne MS. The Vaginal Microbiome in Health and Disease-What Role Do Common Intimate Hygiene Practices Play? <i>Microorganisms</i>. 2023;11(2). 2. Wang G, Yu X, Lu Z, Yang Y, Xia Y, Lai PFH, et al. Optimal combination of multiple cryoprotectants and freezing-thawing conditions for high lactobacilli survival rate during freezing and frozen storage. <i>LWT</i>. 2019;99:217-23. 3. Yockey LJ, Hussain FA, Bergerat A, Reissis A, Worrall D, Xu J, et al. Screening and characterization of vaginal fluid donations for vaginal microbiota transplantation. <i>Sci Rep</i>. 2022;12(1):17948. 4. Vaneechoutte M. <i>Lactobacillus iners</i>, the unusual suspect. <i>Res Microbiol</i>. 2017;168(9-10):826-36. 5. Ravel J, Gajer P, Abdo Z, Schneider GM, Koenig SS, McCulle SL, et al. Vaginal microbiome of reproductive-age women. <i>Proc Natl Acad Sci U S A</i>. 2011;108 Suppl 1:4680-7. 6. Gajer P, Brotman RM, Bai G, Sakamoto J, Schutte UM, Zhong X, et al. Temporal Dynamics of the Human Vaginal Microbiota. <i>Sci Transl Med</i>. 2012;4(132):132ra52. 7. Kindinger LM, Bennett PR, Lee YS, Marchesi JR, Smith A, Cacciatore S, et al. The interaction between vaginal microbiota, cervical length, and vaginal progesterone treatment for preterm birth risk. <i>Microbiome</i>. 2017;5(1):6.