

**PROJECT INFORMATION - 1**

**Project Title** The neurobiology of auditory sensory memory and relevance for schizophrenia

**Hypothesis:**

Blockade of glutamate receptors will impair the formation of auditory sensory memory in a similar way to what is seen in the psychiatric illness, schizophrenia

**PROJECT ATTRIBUTES AND BENEFIT TO THE STUDENT****Brief description of project:**

An electrophysiological index of auditory sensory memory will be measured by EEG in awake, freely moving rats given saline, and a range of doses of the glutamate NMDAR antagonist, MK-801. This drug also promotes behavioural changes related to schizophrenia and similar drugs, when given to humans, can elicit an entire syndrome that resembles the disorder. This project will help indicate how best we can assess 'schizophrenia-like' features in animal models for future drug development.

**Student's role in the project:**

Will be trained to assist in rodent surgeries to implant recording sensors, will be trained to perform welfare monitoring, injections and EEG recording in rats.

**Student's benefit from their involvement:**

The student will be trained on multiple techniques relevant to biomedical research and neuroscientific research. These data are being collected with the aim of supplementing data in a planned publication, so the student will have the opportunity to contribute to the publication as a co-author.

**Research Location Information** (where the project work will be conducted)

**Campus:** CAL

**Building & room number**

Behavioural Sciences W113-W114

**Supervisor's Information (primary supervisor should be in SBSP)****Supervisor Name:**

Lauren Harms

**Academic Appointment at UON:**

Lecturer, SBSP

**Preferred Phone:**

49215664

**E-mail Address:**

Lauren.Harms@newcastle.edu.au

Lauren.Harms@newcastle

## PROJECT INFORMATION - 2

<b>Project Title</b> <b>Gut bacteria and their products as regulators of lung disease</b>
<b>Hypothesis:</b> The abundance of specific bacteria in the gastrointestinal tract will regulate development and progression of respiratory diseases.

## PROJECT ATTRIBUTES AND BENEFIT TO THE STUDENT

<b>Brief description of project:</b> Previously, our research has demonstrated that the microbiome (gut bacteria) is altered in patients with lung diseases, including chronic obstructive pulmonary disease (COPD, often called emphysema). As part of this research, we have identified specific bacteria associated with health and disease.  However, this data is only observational and requires validation to demonstrate a causal role in disease development and progression. To test this, the project will use our group's highly representative animal model to assess the effects of these bacteria on disease. Bacteria will be administered to mice by oral gavage, and immune responses, histopathology and lung function will be assessed to determine the impacts on disease severity.
<b>Student's role in the project:</b> The student will be able to observe and experience how animal models are designed and can be used to answer questions about human health and, if they feel comfortable, students will be able to assist in tissue collections from animals. Students will also participate in lab work to assess changes in the model (staining, microscopy, RNA extraction, PCR, ELISA, etc) and data analysis, as well as interpretation and presentation of results.
<b>Student's benefit from their involvement:</b> Students will gain valuable experience in all stages of research – formation of a hypothesis, experiment design, data collection and interpretation, as well as presentation of the data. There will be extensive laboratory work to allow them to gain practical experience in a number of techniques, which will greatly assist their understanding of work in a lab. In addition, there will be time to talk with and assist researchers on other projects, allowing them to gain a more complete understanding of the research projects being undertaken and different experiences within the entire group.
<b>Research Location Information (where the project work will be conducted)</b>
<b>Campus:</b> Hunter Medical Research Institute <b>Building &amp; room number:</b> HMRI Building, Room 2408

<b>Supervisor's Information (primary supervisor should be in SBSP)</b>	
<b>Supervisor Name:</b> Dr Kurtis Budden	<b>Academic Appointment at UON:</b>
<b>Preferred Phone:</b>	(02) 4042 0818
<b>E-mail Address:</b>	kurtis.budden@newcastle.edu.au

**PROJECT INFORMATION - 3**

<b>Project Title</b> How do cancer cells adapt to evade therapy?
<b>Hypothesis:</b> Following treatment with epigenetic therapies, Acute myeloid leukaemia cells regain cancer-initiating capacity before DNA methylation is restored.

**PROJECT ATTRIBUTES AND BENEFIT TO THE STUDENT**

<p><b><u>Brief description of project:</u></b> Acute myeloid leukaemia (AML) is a devastating disease, with around 75% of patients dying within 5 years of their diagnosis. Epigenetic therapies are used to treat older patients that can't tolerate harsh chemotherapies; however, they only benefit some patients and relapse is common. Our preliminary data from suggests that HL-60 AML cells can adapt to epigenetic therapies, such that they regain their capacity to self-renew before the epigenetic changes induced by therapy are reset. This project will test whether this effect is observed in other AML cell lines including MOLM13 and MV411 cells.</p>
<p><b><u>Student's role in the project:</u></b> The student will perform experiments including cell culture, drug treatments and assessment of cancer-initiating capacity. The student will analyse results and summarise findings.</p> <p><b><u>Student's benefit from their involvement:</u></b> The student will learn experimental techniques applicable to most areas of biomedical research, as well as pathology laboratories. The student will be based at the Hunter Medical Research Institute and will be supported by a team including 3 PhD students, and 3 other scientists. We use advanced sequencing technologies to study genetic regulation in single cells, and the student will gain an appreciation of these methods through lab meetings.</p>
<b>Research Location Information (where the project work will be conducted)</b>
<p><b>Campus:</b> Hunter Medical Research Institute, John Hunter Hospital Campus</p> <p><b>Building &amp; room number</b> Medical Genetics, Level 3 West Hunter Medical Research Institute</p>

<b>Supervisor's Information (primary supervisor should be in SBSP)</b>	
<b>Supervisor Name:</b> Dr Heather Lee	<b>Academic Appointment at UON:</b> Cancer Institute NSW Fellow, School of Biomedical Sciences and Pharmacy
<b>Preferred Phone:</b> 40420680	
<b>E-mail Address:</b> Heather.lee@newcastle.edu.au	

## PROJECT INFORMATION -4

<b>Project Title</b> <b>Molecular mechanisms in colorectal cancer development in mice overexpressing mitochondrial catalase</b>
<b>Hypothesis:</b> Overexpression of mitochondrial catalase alters Wnt- $\beta$ -catenin signalling pathways in gastrointestinal tissues that delay colorectal cancer development in mice.

## PROJECT ATTRIBUTES AND BENEFIT TO THE STUDENT

<b>Brief description of project:</b> Colorectal cancer causes the second highest number of cancer related deaths in Australia. Reactive oxygen species (ROS) play a vital role in normal cellular function, however chronic inflammation in the gastrointestinal system increases ROS beyond normal levels and can contribute to development of colorectal cancer. In our colorectal cancer mouse model, catalase, a naturally occurring ROS scavenger has been overexpressed in the mitochondria, the primary source of ROS, with the intention of delaying colorectal cancer development. Moreover, previous studies have shown that the Wnt- $\beta$ -catenin signalling pathway can be activated by ROS. This project will therefore investigate whether overexpression of mitochondrial catalase in a colorectal cancer mouse model can alter the components of the Wnt- $\beta$ -catenin signalling pathway in colon tissues and whether these changes influence the development of colorectal cancer in this model.
<b>Student's role in the project:</b> This student will extract proteins from frozen mouse tissues and carry out protein quantitation and western blot assays for Wnt- $\beta$ -catenin signalling proteins. The student will also be involved in immunohistochemical staining of sectioned mouse tissues.
<b>Student's benefit from their involvement:</b> The student will benefit from receiving training in the laboratory techniques described above. Successful completion of this project may result in the publication of the data in journals/conferences which can be added to the student's CV. This project has the potential to lead into further study e.g Honours/PhD.
<b>Research Location Information (where the project work will be conducted)</b>
<b>Campus:</b> Hunter Medical Research Institute <b>Building &amp; room number</b> HMRI Level 3 East

Supervisor's Information (primary supervisor should be in SBSP)	
<b>Supervisor Name:</b> Doan Ngo	<b>Academic Appointment at UON:</b> A/Prof
<b>Preferred Phone:</b> 02 40339386	
<b>E-mail Address:</b> Doan.Ngo@newcastle.edu.au	

## PROJECT INFORMATION - 5

**Project Title: The role of mitochondria in oxygen sensing and fetal development**

**Hypothesis:** Mitochondrial mechanisms of oxygen sensing will be dysfunctional in gestational complications impairing fetal development.

## PROJECT ATTRIBUTES AND BENEFIT TO THE STUDENT

**Brief description of project:**

This project will examine the potential for mitochondria within the placenta to sense oxygen via molecular pathways and alter metabolism accordingly. While often referred to as the “powerhouse” of the cell, the mitochondria have a complex role in regulating homeostasis. This project will characterise the capacity of placental mitochondria to sense and respond to fluctuating oxygen concentrations, investigating if this mechanism is dysregulated in pregnancies which progress to pathologies such as fetal growth restriction.

**Student’s role in the project:**

The student will play a role in primary data generation, including PCR, western blotting and real time observations of mitochondrial functionality.

**Student’s benefit from their involvement:**

The student will gain first hand laboratory experience investigating mechanisms which may guide clinical practices. Further, as this project is based in mitochondrial mechanisms the student will gain knowledge into mitochondrial function and cellular metabolism.

**Research Location Information (where the project work will be conducted)**

**Campus:** HMRI

**Building & room number** Level 3 East

**Supervisor’s Information (primary supervisor should be in SBSP)**

**Supervisor Name:** Sarah Delforce

**Academic Appointment at UON:** Research Associate

**Preferred Phone:** 02 4043 0343

**E-mail Address:**

sarah.delforce@newcastle.edu.au

**Supervisor Name:** Joshua Fisher

**Academic Appointment at UON:** Research Associate

**Preferred Phone:** 02 4042 0763

**E-mail Address:**

Joshua.fisher@newcastle.edu.au