Supervisor(s): Mr Malcolm Starkey and Prof Philip Hansbro

Contact Details: Malcolm.Starkey@newcastle.edu.au and Philip.Hansbro@newcastle.edu.au

Project Title: Elucidation of the role of Natural Killer T cells in COPD

Project Outline: Using our well established mouse model of COPD, this project aims to quantify alterations in the number and activation of Natural Killer T cells (NKT cells) using multicolour flow cytometry. We will also determine the functional role of NKT cells in COPD pathogenesis using two different strains of NKT cell deficient mice. Students would be involved in analysis of flow cytometry and lung function data, histological analysis of lung tissue sections, quantitative PCR for genes and microRNAs, and quantification of proteins using ELISA and western blot.

Supervisor(s): A/Prof Lisa Wood

Contact Details: Lisa.Wood@newcastle.edu.au

Project Title: Effects of a prebiotic/probiotic combination on GPR43 activity in asthma

Project Outline: The intestinal mucosal surface is a complex immunological system, being the interface between nutrient influx, the gut microbiota, the epithelium and immune cells of the lamina propria. Complex interactions occur at the mucosal surface that influence the development of the host immune system. This highlights the potential role of dietary intake in the development and progression of diseases involving aberrant immune responses, such as asthma. One example of a mechanism linking nutrient intake and immunity involves the G-protein-coupled receptor 43 (GPR43). GPR43 is activated by short chain fatty acids (SCFA) in the gut, which are produced by the fermentation of dietary fibre and non-digestible carbohydrates (eg inulin, which is classified as a prebiotic) by intestinal microbes. Recent work by Maslowski et al has demonstrated that stimulation of GPR43 by SCFAs is necessary for the normal resolution of inflammation. The absence of GPR43 leads to the production of inflammatory mediators and immune cell recruitment, both systemically and in the airways. We have conducted a pilot study, in which asthmatic adults (n=19) consumed a single dose of SCFA, delivered as a pre/probiotic supplement containing lactobacillus GG, acidophilus, bifidus and inulin. We now need to examine changes in systemic and airway inflammation following the supplement. Inflammatory markers will be analysed using ELISA and PCR techniques. These will be related to clinical asthma outcomes.

Supervisor(s): Miss Bernadette Jones and Prof Philip Hansbro

Contact Details: Bernadette.Jones@uon.edu.au and Philip.Hansbro@newcastle.edu.au

Project Title: Determining the role of epigenetic regulation in the development of chronic obstructive pulmonary disease

Project Outline: Chronic obstructive pulmonary disease (COPD) is one of the leading causes of morbidity and mortality worldwide and is expected to increase in the coming years, approaching epidemic proportions. Tobacco smoke is the major risk factor for the development of COPD where 25-30% of smokers will develop the disease. And while the risk of developing COPD is reduced, ex-smokers still can develop the disease several years after quitting. Currently high doses of inhaled steroids are used to suppress symptoms, but there are no effective treatments that halt the progression of disease.

One of the major problems faced with COPD is that little is known about the underlying genetic mechanisms that lead to the development and progression of the disease. Epigenetics represents modifications of the genetic material without alteration to the primary DNA sequence. Epigenetic variations act like switches to regulate gene expression in some individuals, and these variations may...
account for the reason why only some smokers get COPD, and why some smokers develop COPD long after smoking cessation. These types of variations cannot be identified by simply studying DNA sequences, and represent a novel mechanism in disease development and progression.

We have access to a unique experimental model of COPD, which we have used to identify alterations in gene expression associated with pathological status. Using this knowledge the student will be determining epigenetic variations, such as DNA methylation, that may contribute to these expression changes. Students will be working with this experimental model and, using a suite of cutting edge techniques available in our laboratory, will compare the alterations in disease state and healthy controls and identify novel epigenetic effects in the disease state. Findings from these experiments will determine if these epigenetic mechanisms play a role in the pathogenesis of COPD and, therefore, identify potential therapeutic targets. This is an extremely novel project, since studies involving epigenetics and COPD have been limited.

Supervisor(s): Laureate Professor Paul Foster

Contact Details: Paul.Foster@newcastle.edu.au

Project Title: Understanding the mechanisms that regulate respiratory and infectious disease of the lung.

Project Outline: A number of honours projects with scholarships will be available in the priority research Centre for Asthma and Respiratory Disease (CARD) under the supervision of Professor Paul Foster and colleagues.

The Foster laboratory focuses on understanding the pathogenesis of respiratory diseases such as asthma and the role of infections in pathogenesis. We particularly focus on the interplay between viral and bacterial infections and new ways to treat these respiratory disorders.

Current projects focus on:

1. How do microRNA and long-non coding RNA regulate immune cell development and inflammation linked to respiratory disease and infection: These molecules are now recognized as key regulator of gene expression by regulating translation and we have demonstrated there importance in the regulation of inflammation processes.

2. Characterisation of a new T helper (Th) subset known as Th22 cells: we have recently generated these cells and now need to clarify their immunological role and molecular description at a cellular level.

3. Development of new ways to treat viral and bacterial infections of the lung. We urgently need new ways to treat bacterial infections as antibiotic resistant increases and to treat pathogenic viral infections associated with chronic diseases and pandemics.

4. Cellular and molecular characterisation of the pathways that lead to the inability of steroid treatment to be effective: this is a major clinical problem

5. Role of T cells and innate immune cells in disease progression, and the role of the airway epithelium in providing the early signals for induction of inflammation

The group is well funded by the NHMRC and consists of postdoctoral fellows that will participate in supervision. The group collaborates widely and co-projects with leading investigators are often available (e.g. Professors Mattes, Hansbro and Knight).

Supervisor(s): Dr Katie Baines, A/Prof Jodie Simpson, A/Prof Lisa Wood

Contact Details: Katherine.Baines@newcastle.edu.au, Jodie.Simpson@newcastle.edu.au
Project Title: Effect of corticosteroid treatment of asthma on the inflammasome

Project Outline: Although the success of corticosteroid treatment of asthma lies in their anti-inflammatory actions, recent data suggests that corticosteroids may also have pro-inflammatory actions on innate immune responses, including sensitisation of the inflammasome. This project will characterise the effects of oral corticosteroid treatment of asthma on inflammasome expression and activation in human airway samples.

Supervisor(s): Prof Joerg Mattes and Laureate Prof Paul Foster

Contact Details: Joerg.Mattes@newcastle.edu.au

Project Title: Mechanisms of viral infection-induced asthma exacerbation

Project Outline: The Experimental and Translational Respiratory Medicine group based at the HMRI as a part of the Centre for Asthma and Respiratory Disease have an opportunity for a B.BiomedSci graduate to undertake an honours year program. The research focus will build upon the group’s recent publications (Weckman et al Nat Med 2007, Collison et al Nat Med 2013, Hatchwell et al JACI pending review) in which the importance of TRAIL signalling through MID1 modulation of PP2A activity in allergic asthma and viral exacerbations were first identified. The successful applicant will evaluate novel drugs specifically targeting PP2A in well characterised mouse models of house dust mite induced allergic airways disease and Rhinovirus induced exacerbation. Currently there is no clinical option for the prevention for viral induced exacerbations of asthma and these are responsible for 80% of emergency room presentations of asthma attacks, highlighting the urgent need for the development of novel therapeutic options.

Supervisor(s): A/Prof Jodie Simpson, A/Prof Lisa Wood, Dr Katie Baines and Prof Peter Gibson

Contact Details: Jodie.Simpson@newcastle.edu.au, Lisa.Wood@newcastle.edu.au, Katherine.Baines@newcastle.edu.au and Peter.Gibson@newcastle.edu.au

Project title: Characterising the NLRP3 inflammasome in asthma and obesity

Project Outline: Asthma and obesity are important chronic disorders that impose a significant burden at both an individual and a community level. The prevalence of both obesity and asthma has increased globally in recent decades (Wood LG et al. Am. J. Respir. Crit. Care Med. 2012) and there an association between obesity and asthma (Beckett WS, et al. Am J Respir Crit Care Med 2001, Guerra S, et al. Chest 2002, Camargo CA Jr, et al.Arch Intern Med1999), with asthma incidence increased by 50% in those who are overweight or obese (Beuther DA, et al.Am J Respir Crit Care Med 2007). We do not yet understand how asthma and obesity are associated.

We know that in asthma around half of all adults have a pattern of inflammation which does not respond to current asthma therapies (inhaled corticosteroids). In addition we have made a recent discovery (Scott HA, et al Eur Resp J 2011) that adults with asthma who are obese also have an inflammatory profile that results in poor response to asthma therapy. The pattern of inflammation is dominated by an inflammatory cell type called neutrophils, which is one of the white blood cells recruited to the airways in response to infection and stress.

In a separate study of inflammatory mechanisms in asthma we have shown that patients with asthma, who have high neutrophils, also have increased levels of a protein called IL-1 b which is a potent inducer of inflammation.

These discoveries in our research at HMRI have led us to question if IL-1b is important in obese asthma, and if the pathway involved in the production and release of IL-1 b in the airways (called the inflammasome) is one of the pieces in the asthma/obesity puzzle.
The aims of this study are to (1) investigate the levels of IL-1b in the sputum supernatant from adults with asthma and healthy controls who are obese and also those of a healthy weight, (2) examine the association of airway IL-1b with the gene expression of NALP3, ASC and caspase-1 and (3) determine the effect of weight loss on the level of airway IL-1b.

**Supervisor(s):** Dr Dean Sculley, A/Prof Jane Taylor and Dr Zoe Yates

**Contact Details:** Dean.Sculley@newcastle.edu.au, Jane.Taylor@newcastle.edu.au and Zoe.Yates@newcastle.edu.au

**Project Title:** Effects of green tea catechins on salivary inflammatory biomarkers and periodontal disease status

**Project Outline:** Epigallocatechin-3-gallate (EGCG) is a potent antioxidant and the main catechin in green tea. Periodontal disease is characterised by high levels of inflammation around the gingiva, which results in host tissue degradation including loss of gingiva and associated increase in periodontal pocket depth, alveolar bone resorption and destruction of the periodontal ligament. Consumption of green tea may help to reduce the inflammatory cascade and limit the progression and severity of periodontal disease. A RCT trial with intervention group (EGCG) and placebo group. Biomarkers to be assessed include IL-6, TNF-a and total antioxidant capacity. Periodontal pocket depth to be assessed by Oral Health staff.

**Supervisor(s):** Dr Dean Sculley, A/Prof Jane Taylor and Dr Zoe Yates

**Contact Details:** Dean.Sculley@newcastle.edu.au, Jane.Taylor@newcastle.edu.au and Zoe.Yates@newcastle.edu.au

**Project Title:** Effects of Resveratrol on salivary inflammatory biomarkers and periodontal disease status

**Project Outline:** Resveratrol is a naturally-occurring phenol with antioxidant potential. Periodontal disease is characterised by high levels of inflammation around the gingiva which results in host tissue degradation including loss of gingiva and associated increase in periodontal pocket depth, alveolar bone resorption and destruction of the periodontal ligament. Consumption of resveratrol, in tablet form, may attenuate inflammation via the COX-2 pathway and also reduce the production of prostaglandin E2, a known driver of bone resorption. RCT trial with intervention group (resveratrol) and placebo. Biomarkers to be assessed include IL-6, TNF-a, prostaglandin E2 and total antioxidant capacity. Periodontal pocket depth to be assessed by Oral Health staff.

**Supervisor(s):** Dr Jay Horvat and Prof Philip Hansbro

**Contact Details:** Jay.Horvat@newcastle.edu.au and Philip.Hansbro@newcastle.edu.au

**Project Title:** Investigation of novel immune molecules in the pathogenesis of female Chlamydia genital tract infections

**Project Outline:** Chlamydia genital tract infections are common and mostly asymptomatic. The asymptomatic nature of infections, means that most infections go undetected and, therefore, untreated. This is a major problem as asymptomatic infections in the upper reproductive tract induce inflammatory responses that cause tissue damage and result a variety of diseases, including pelvic inflammatory disease, endometritis and ectopic pregnancy. Significantly, Chlamydia-induced immunopathology is a leading cause of tubal factor infertility.

One of the major problems faced when combating Chlamydia-induced disease is little is known about the immune responses that are important in clearing infection versus those that drive pathology. Our research group has developed a research program to help elucidate these immune responses.
We have access to a number of mouse strains that have had genes that code for specific immune molecules deleted or added to their genome. Students will be infecting these mice with Chlamydia and, using a suite of cutting edge techniques available in our laboratory, will compare the effects of loss/gain of immune molecules on bacterial growth, immune function and pathology during infection. Findings from these experiments determine if these immune molecules play a role in the pathogenesis of infection and, therefore, may be novel therapeutic targets. This is an extremely novel project, since none of these genes have ever been investigated in terms of Chlamydia infection before.
Supervisor(s): Zhenya (Eugene) Nalivaiko

Contact Details: Eugene.Nalivaiko@newcastle.edu.au

Project Title: Where is nausea perceived? A new rodent model for advancing preclinical studies of nausea

Project Outline: Chemotherapy drugs used to kill breast cancer cells provoke potent aversive side effect – nausea and vomiting. This not only dramatically worsens patients’ quality of life, but may affect their willingness to continue anti-cancer treatment. Chemotherapy-induced vomiting could be relatively well controlled by the last generation anti-emetic drugs, but nausea is still the most feared symptom of chemotherapy. Mechanisms mediating nausea are unknown. There are no adequate animal models for nausea studies and for drug testing. We will first focus on validating our new model of nausea in rats (that cannot vomit) and in musk shrews (a mouse-like animal that commonly used in emesis studies as they have vomiting reflex). During subjecting our experimental animals to stimuli that cause nausea in humans, we will record several physiological variables that closely mirror human symptoms of nausea. Using this new model, we will determine which parts of the brain mediate nausea signs.

Supervisor(s): Dr Kathryn Skelding

Contact Details: Kathryn.Skelding@newcastle.edu.au

Project Title: Improving survival and reducing treatment side effects in childhood leukaemia

Project Outline: Cancer is the most common cause of childhood disease-related deaths, and acute lymphoblastic leukaemia (A.L.L) is the most common childhood cancer. Whilst remission is achievable in 95% of cases, approximately 1/3 of patients will relapse, and 20-30% of children with leukaemia will not be long-term survivors. The major cause of relapse in these children is the development of drug resistance. Therefore, new anti-cancer targets in these chemotherapeutic resistant cells need to be identified, in order to improve the outcomes for children with A.L.L. Our recent work has identified a new target that is present in A.L.L, and is associated with the development of resistance to chemotherapeutics. We have developed a new drug that can effectively target this protein, and our initial studies indicate that this drug is highly cancer-cell specific, raising the possibility that this drug may reduce treatment associated side-effects. This project will examine the role of this protein in A.L.L, and will also investigate the pre-clinical effectiveness of our new drug, as a new treatment for chemotherapeutic resistant A.L.L.

Supervisor(s): Dr Kirsty Pringle and Prof Eugenie Lumbers

Contact Details: Kirsty.Pringle@newcastle.edu.au and Eugenie.Lumbers@newcastle.edu.au

Project Title: Expression of the renin angiotensin system (RAS) in endometrial cancer and the therapeutic potential of RAS blocking drugs

Project Outline: There is evidence that individuals taking RAS blocking drugs for treatment of high blood pressure have a lower incidence of cancer. Using qPCR, immunohistochemistry and western blotting, you will first determine whether the expression of the endometrial renin angiotensin system is dysregulated in endometrial cancer. Second you will find out if drugs that affect the activity of the various renin angiotensin system pathways inhibit tumour growth by inhibiting angiogenesis and cell proliferation and can be used as cancer therapeutics using a variety of cellular and molecular biology techniques in an endometrial cancer cell line.
Supervisor(s): Dr. Jean-Marie Sontag and A/Prof Estelle Sontag,

Contact Details: Jean-Marie.Sontag@newcastle.edu.au and Estelle.Sontag@newcastle.edu.au.

Project Title: Elucidating the mechanisms of epithelial cell transformation

Project Outline: This project will use cultured cell models and mouse tissue to investigate the molecular mechanisms that allow small DNA viruses to transform normal epithelial cells into cancerous cells by more specifically affecting cell adhesion and signal transduction cascades.
Supervisor(s): A/Prof Estelle Sontag and Dr. Jean-Marie Sontag

Contact Details: Estelle.Sontag@newcastle.edu.au and Jean-Marie.Sontag@newcastle.edu.au

Project Title: Understanding the mechanisms underlying Alzheimer’s disease pathogenesis

Project Outline: Alzheimer’s disease is the most prevalent neurodegenerative disorder. The goal of this NHMRC-funded project is to further understand the molecular mechanisms underlying Alzheimer’s disease pathogenesis using molecular and biochemical analysis of cultured cells and brain tissue from mouse models. It is performed in collaboration with two major US laboratories (Baylor University in Dallas, Columbia University in New York) that are international leaders in the field of neuropharmacology and neurodegeneration.

Supervisor(s): A/Prof Estelle Sontag and Dr. Jean-Marie Sontag

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Project Title: Improving treatment of patients affected by Parkinson’s disease

Project Outline: Currently, L-Dopa is the standard therapy for treating motor symptoms in patients affected by Parkinson’s disease (PD), a major age-related neurodegenerative disease. However, it can result in prevalent debilitating side-effects, including cognitive dysfunction, neuropsychiatric symptoms and vascular disease. Recently, our lab has identified a novel mechanism that may underlie some of the side-effects associated with traditional L-Dopa therapy (J Neuroscience 2012; 32(27):9173). The proposed project, performed in collaboration with a leading US laboratory, will test in mouse models whether the side-effects of chronic L-Dopa can be abrogated by dietary supplementation with selected compounds.

Supervisor(s): Prof Peter Howe and Dr Rachel Wong

Contact Details: Peter.Howe@newcastle.edu.au and Rachel.Wong@newcastle.edu.au

Project Title: Effects of resveratrol on cerebral circulation and cognitive function

Project Outline: AIMS: To determine the effects of resveratrol supplementation on cerebrovascular responsiveness to hypercapnia and cognitive stimuli and cognitive performance and whether these improvements correlate with plasma resveratrol levels. As a secondary objective, we will also determine whether resveratrol can improve orthostatic cerebral blood flow and its relationship with cerebral vasodilator function.

BACKGROUND: Maintaining optimal blood flow in the brain protects neuronal tissues and is critical for brain function. Impaired blood flow is reflective of poor vasodilatory capacity of blood vessels to meet its demands, resulting in either under or over perfusion in the brain. This can manifests as dysregulation of orthostatic blood pressure, which has been linked to future risk of stroke, silent cerebrovascular disease and cognitive decline. However, the link between impaired vasodilatory function and orthostatic cerebral blood flow changes is unknown. There is increasing evidence that certain vasoactive nutrients such as polyphenols from a variety of plant sources including cocoa, soy, oats and resveratrol can act on endothelial cells to enhance vasodilator function to improve blood flow. We have previously demonstrated the acute and chronic benefits of resveratrol, a vasoactive ingredient in red wine, on systemic vasodilator function in mildly untreated hypertensive, obese adults. We now need to further examine whether this benefit can be extended to the cerebral circulatory function and whether it can impact cognitive performance.
STUDY DESIGN: The transCranial Doppler (TCD) ultrasound offers a direct, non-invasive measure of blood vessel function in the brain by assessing changes in blood flow velocity in the middle cerebral artery (MCA) resulting from downstream dilatation of cerebral arterioles, which can occur in response to physiological (acute hypercapnia) or cognitive stimuli. This technique, known as cerebral vasodilator responsiveness (CVR), can be used to quantify the vasodilator capacity of cerebral arteries.

The acute of effects resveratrol on CVR to hypercapnia and orthostatic cerebral blood flow will be determined an hour after a single dose of 75mg of resveratrol or placebo. The protocol will be repeated a week later with the alternate treatment. Participants will then be randomised in a crossover fashion to consume either 75mg of resveratrol daily or a placebo for 6 weeks. Outcome measures of CVR to hypercapnia and cognitive stimuli, orthostatic cerebral blood flow response, cognitive performance and plasma resveratrol levels will be assessed at week 6 and repeated in week 12 after crossing over to the alternate treatment. The outcomes of neuropsychological tests and CVR to cognitive stimuli of this project will be undertaken by a student with an interest in psychology.

Supervisor(s): Dr Murray Cairns and Dr Matt Dun

Contact Details: Murray.Cairns@newcastle.edu.au and Matt.Dun@newcastle.edu.au

Project Title: Non-coding RNA regulation of ribonucleoprotein complexes and their intracellular traffic of mRNA

Project Outline: RNA translation in highly differentiated cells is decentralized and occurs in discrete foci in response to specific intracellular landmarks and cues. This enables very precise intracellular regulation of new protein synthesis to support diverse and highly localised function within complex cellular architecture. While the post-transcriptional mechanism that support this refinement in gene expression are not well understood, they are critical for the development and physiology of complex mammalian systems. They are also prone to dysfunction and therefore have implications in a broad range of human health problems, including cancer and neurological disorders. We now have powerful new high-throughput sequencing methodology for tracking the interactions, traffic and fate of nucleic acids and protein molecules associated with these mechanisms, and will use these approaches in this project to determine their regulatory function in a model system. This has the potential to reveal significant new insight into the nexus between transcription and translation and highlight the function of non-coding RNA as guides and adapter molecules in these processes.

Supervisor(s): Dr Susan Hua

Contact Details: Susan.Hua@newcastle.edu.au

Project Title: Mimicking ‘pain-relieving’ immune cells in rheumatoid arthritis

Project Outline: The aim of this project is to demonstrate that exogenous opioids can be targeted specifically to peripheral injured tissue using targeted immunoliposomes to exert analgesic, anti-inflammatory, and disease ameliorating activity in the antigen-induced arthritis (AIA) rat model of rheumatoid arthritis. From this project, you will acquire essential techniques in the field of pharmaceutical design and formulation, in vitro cell studies, and in vivo animal studies.

Supervisor(s): Dr Doug Smith

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Project Title: The neurobiology of ageing

Project Outline: Ageing is inevitable and most populations around the world are getting increasingly older. Soon there will be more 65+ year olds than children in developed countries – the grey tsunami! The long-term goal of our research is to understand the processes of ageing. We can then develop
ways to increase health span, the length of time one remains in good health, functional and independent from care, and therefore having an acceptable quality of life. What most people do not realise is that ageing, while inevitable, is also modifiable and we are attempting to understand how this can be achieved. We are characterising the changes that occur in the nervous system during normal ageing, that is, without pathology. As the nervous system is involved in all aspects of life, its maintenance during ageing is critical to improved health span. We use state-of-the art molecular, proteomic and cellular techniques to probe changes in nuclear and mitochondrial genomes and proteomes in various regions and cells of both the central and peripheral nervous systems. There are a number of projects available for Honours students.
Supervisor(s): Dr Hannah Palliser and A/Prof Jon Hirst

Contact Details: Jon.Hirst@newcastle.edu.au and Hannah.Palliser@newcastle.edu.au

Project Title: Determining the link between maternal stress in pregnancy and behavioural disorders in childhood and adolescence

Project Outline: Increasing evidence demonstrates an important link between maternal psychosocial stress and serious behavioural problems (anxiety, hyperactivity as well as learning and memory deficits) in children and young adults. We have used a guinea pig model to show reproducible stress exposure in pregnancy causes increased anxiety in the adolescent offspring. This exposure also results in a marked disruption of steroid metabolism in the newborn brain suggesting a persistent reduction in steroid levels leads to increased anxiety.

The aim of this project is to examine the effect of stress in pregnancy on factors regulating of key enzymes that control neuroactive steroid production after birth. This project will delineate unique mechanisms that lead from maternal stress to behavioural disorders in childhood and adolescence. Furthermore, the work will indicate the potential effectiveness of neuroactive steroid supplementation after birth in reversing the behavioural abnormalities.

Supervisor(s): Dr Rebecca Vanders and Prof Philip Hansbro

Contact Details: Rebecca.Vanders@newcastle.edu.au and Philip.Hansbro@newcastle.edu.au

Project Title: Investigation of influenza induced alterations in antiviral immunity during pregnancy and identification of novel therapeutic strategies

Project Outline: To examine the changes that occur in the maternal and fetal antiviral immune system following influenza infections during pregnancy with and without asthma. Maternal and fetal immunity will also be examined following vaccination and application of anti-PDL1 therapy.

This project utilises a mouse model of influenza infection in pregnancy and asthma sensitisation. A range of techniques are employed including PCR, WB, flow cytometry, immunohistochemistry, histology, mouse handling and administration, tissue culture, virus propagation etc.

Supervisor(s): Dr Kirsty Pringle and Prof Eugenie Lumbers

Contact Details: Kirsty.Prinle@newcastle.edu.au and Eugenie.Lumbers@newcastle.edu.au

Project Title: Regulation of the placental renin-angiotensin system by microRNAs in pregnancy pathologies

Project Outline: Abnormal placentation is a major cause of preterm birth, preeclampsia and intrauterine growth restriction. The placental renin-angiotensin system (RAS) plays an important role in human placentation, but the mechanisms controlling expression of the placental RAS are unknown. This project will employ cell and tissue culture and a variety of molecular biology techniques to determine the role(s) of microRNAs in regulating the placental RAS.

Supervisor(s): Dr Kirsty Pringle and Dr Kym Rae

Contact Details: Kirsty.Prinle@newcastle.edu.au and Kym.Rae@hnehealth.nsw.gov.au
Project Title: Investigation of relationships between renal function and glucose metabolism in Indigenous pregnant women

Project Outline: Indigenous Australians are ten times more likely to develop chronic kidney disease than non-Indigenous Australians. There is also a high level of diabetes mellitus in Indigenous populations. We are studying the renal health of Indigenous pregnant women in the Gomeroi Gaaynggal ArtsHealth program. We want to assess relationships between glucose homeostasis, plasma glucose levels, HbA1c, and the circulating and intrarenal renin angiotensin systems using novel biomarkers that we have developed in the laboratory.