Honours in Pharmacology

2013

Projects and Program Information
Message from the Head of Discipline and Honours Coordinator

The Discipline of Pharmacology invites you to apply to undertake a research year in the fourth year of your studies (Honours in Pharmacology). This program is designed to give students a greater depth to their studies in biomedical science and to promote research-led inquiry and intellectual endeavour. Students who complete Honours in Pharmacology will be equipped with a skill set that improves their employment prospects in industry or government and is a requirement for pursuing postgraduate studies in Pharmacology or related areas.

In the Discipline of Pharmacology at The University of Sydney, we have a group of dedicated academic staff who are conducting cutting-edge research across a variety of fields, including asthma pharmacology, cancer therapeutics, chemical biology, chronic inflammation and pain, clinical pharmacology, drug design and development, drug delivery, neuropharmacology, pain management, pharmacoinformatics, pharmacology of cannabis, and transporter biology. We have a changing staff profile, with several recent appointments having been made in frontier areas. This booklet is designed to provide further details about the Honours program and describes in some detail the projects on offer to students in 2013. We hope you’ll join us.

Please contact the Honours Coordinator (Dr Rachel Codd: rachel.codd@sydney.edu.au) with any further enquiries you may have.

I’m interested in Honours in Pharmacology - what do I do next?

Please join us for our Honours Information session, which is to be held on:

Monday 17 September at 1 pm in Bosch Lecture Theatre 4.

At this session, the Honours Coordinator will provide further details on the structure of the program and staff will give a snapshot of their research areas. After formal proceedings, you are warmly invited to a lunch in the Bosch precinct courtyard from 2:00 pm, where you will be able to talk with individual members of staff in whose projects you have an interest. You have about 2 months to reach a decision about which project/research group interests you the most, before submitting to the Honours Coordinator your Honours Preference Form (end of this booklet), which is due on Friday 23 November 2012. Students can elect to start their Honours year in Semester 1 or Semester 2. To enable academics to plan their group composition, students who wish to begin in Semester 2 are advised to make their supervisor selection at the start of the year.

In addition to lodging your Honours Preference Form with Pharmacology, you must lodge an application for Honours through the Faculty of Science. Further information is available on the Faculty of Science URL: http://sydney.edu.au/science/fstudent/undergrad/course/honours/index.shtml

Am I eligible for Honours in Pharmacology?

All students with a sound record in Pharmacology are strongly encouraged to apply to the Honours Program. Students are required to have completed a major in the area relevant to Honours and have a Science Weighted Average Mark (SCIWAM) of at least 65. If you are uncertain about your eligibility, you should arrange to meet with the Honours Coordinator and have your academic transcript available for review. Further information is available from the Honours Coordinator and on the Faculty of Science URL: http://sydney.edu.au/science/fstudent/undergrad/course/honours/index.shtml

What will I do during my Honours year?

You will undertake a research project under the direct supervision of a member of staff, and as part of their research group. You will deliver two oral presentations to the Discipline (one in May/June (10%) and another in Oct/Nov (10%)), write a 16-page combined literature review and research proposal (May/June (15%)) and write a 50-page thesis detailing the aims, methods, results and discussion of your project (55%). Your supervisor will award you a mark (10%) that reflects your research dedication, competency and aptitude.
<table>
<thead>
<tr>
<th>Name</th>
<th>Location</th>
<th>Research Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dr Jonathon Arnold</td>
<td>BKB: 307</td>
<td>Endogenous cannabinoid system, behavioural neuropharmacology</td>
</tr>
<tr>
<td>Dr Elena Bagley</td>
<td>BKB: W326</td>
<td>Synaptic physiology/plasticity, synaptic function/dysfunction, brain disorders</td>
</tr>
<tr>
<td>Prof. Judy Black</td>
<td>WIMR</td>
<td>Asthma pharmacology, smooth muscle culture, inflammation</td>
</tr>
<tr>
<td>A/Prof Janette Burgess</td>
<td>WIMR</td>
<td>Asthma, angiogenesis, molecular biology, gene discovery</td>
</tr>
<tr>
<td>Dr Kellie Charles</td>
<td>BKB: 306</td>
<td>Cancer pharmacology, tumour-immune cell interactions</td>
</tr>
<tr>
<td>Prof MacDonald Christie</td>
<td>BKB: W326</td>
<td>Cellular/molecular neuropharmacology, pain pathways and pain therapeutics</td>
</tr>
<tr>
<td>Dr Rachel Codd</td>
<td>BKB: 274</td>
<td>Chemical biology and medicinal chemistry, metals in biology</td>
</tr>
<tr>
<td>A/Prof Sarah Hilmer</td>
<td>RNSH: W11c</td>
<td>Geriatric medicine and clinical pharmacology</td>
</tr>
<tr>
<td>Dr Tina Hinton</td>
<td>BKB: 294</td>
<td>GABAergic neurotransmission in the CNS, schizophrenia</td>
</tr>
<tr>
<td>Prof. Michael Kassiou</td>
<td>BMRI</td>
<td>Drug design and medicinal chemistry, CNS active compounds</td>
</tr>
<tr>
<td>Dr Hilary Lloyd</td>
<td>BKB: 219</td>
<td>Neurotransmitter release mechanisms, neuroprotection</td>
</tr>
<tr>
<td>Dr Slade Matthews</td>
<td>BKB: 214</td>
<td>Machine learning in biomedicine</td>
</tr>
<tr>
<td>Dr Brent McParland</td>
<td>BKB: 304</td>
<td>Asthma pharmacology, human bronchus, smooth muscle</td>
</tr>
<tr>
<td>Dr Ann Mitrovic</td>
<td>BKB: 294</td>
<td>Lymes disease, molecular biology</td>
</tr>
<tr>
<td>Dr Lenka Munoz</td>
<td>BKB: TBA</td>
<td>Glioblastoma, p38 MAPK inhibitors, medicinal chemistry</td>
</tr>
<tr>
<td>Dr Brian Oliver</td>
<td>WIMR</td>
<td>Respiratory pharmacology</td>
</tr>
<tr>
<td>A/Prof. Renae Ryan</td>
<td>BKB: 212</td>
<td>Biophysics of membrane transport, glycine transport</td>
</tr>
<tr>
<td>Prof. Paul Seale</td>
<td>BKB: 301B</td>
<td>Clinical pharmacology, asthma, corticosteroids</td>
</tr>
<tr>
<td>Dr Margaret Sunde</td>
<td>BKB: TBA</td>
<td>Protein biophysics, protein misfolding, amyloid fibril formation and structure</td>
</tr>
<tr>
<td>A/Prof Daniela Traini</td>
<td>WIMR</td>
<td>Respiratory drug delivery science, asthma, COPD, bronchiectasis</td>
</tr>
<tr>
<td>Prof. Robert Vandenberg</td>
<td>BKB: 223</td>
<td>Molecular biology, glutamate transport, electrophysiology</td>
</tr>
<tr>
<td>Dr Chris Vaughn</td>
<td>KOL (RNSH)</td>
<td>Chronic pains and endocannabinoids, models of neuropathic pain</td>
</tr>
<tr>
<td>A/Prof. Paul Young</td>
<td>WIMR</td>
<td>Respiratory diseases, medical devices, lung-specific advanced formulations</td>
</tr>
</tbody>
</table>

To e-mail staff, the generic format is: firstname.familyname@sydney.edu.au. For example: rachel.codd@sydney.edu.au.

BKB = Blackburn building (D06), BSH = Bosch building (D05). BMRI = Brain & Mind Research Institute; KOL, Kolling Building; WIMR = Woolcock Institute of Medical Research. RNSH = Royal North Shore Hospital. RPAH = Royal Prince Alfred Hospital.
PROJECT 1  AN ANIMAL MODEL OF GENE-ENVIRONMENT INTERACTION IN SCHIZOPHRENIA

Schizophrenia (SCZ) arises due to a complex interaction between genetic and environmental factors during early neurodevelopment, culminating with disease onset in late adolescence/early adulthood. This project aims to model in mice how genetic vulnerability interacts with environmental insults to disturb brain maturation subserving the development of SCZ symptoms. Our unique model focuses on a SCZ susceptibility gene, neuregulin 1 (NRG1), and two environmental insults linked to SCZ, early life stress and adolescent cannabis use. In rodents such insults promote loss of dendritic spines and long-lasting behavioural deficits. This is significant as dendritic spines support excitatory synaptic connections and are less abundant in SCZ brain. The brains of SCZ patients show reduced N-methyl-D-aspartate receptor (NMDAr) levels, a key regulator of dendritic spine growth and maturation. Mice heterozygous for the Nrg1 gene (Nrg1 Het mice) provide a powerful model of SCZ as they have dysfunctional NMDAr and display a time-dependent expression of SCZ-related behaviour. We have data showing repeated adolescent stress exposure in these mice unmasks attention deficits earlier than in the absence of stress. Here we aim to examine whether this is subserved by a genetic vulnerability to stress-induced NMDAr dysfunction and loss of dendritic spines in key cognitive areas of the brain. Further, we will observe whether repeated environmental insults (e.g. prenatal stress and adolescent cannabinoid exposure) amplifies neurobehavioural deficits. Once our model has been developed, we will test whether we can restore NMDAr function and dendritic spine growth. Recombinant Nrg1 (rNrg1) and the atypical antipsychotic clozapine are beneficial or harmful effects of cannabis use on antipsychotic drug therapy. This project aims to investigate whether cannabis use might alter the effectiveness of antipsychotic treatment in schizophrenia patients. Many antipsychotic drugs are substrates for ABC transporters. These transporters are localized at the blood brain barrier where they bind substrate drugs and transport them out of the brain back into the peripheral blood supply. Our work has shown acute cannabinoid exposure inhibits the transport function of the ABC transporters P-gp and BCRP. Therefore, cannabis-using schizophrenia patients may have increased CNS retention of antipsychotic drugs that would either assist in reducing schizophrenia symptoms and/or increase the incidence of side effects. An alternate mechanism whereby cannabis might affect the brain retention of antipsychotic drugs is by altering the expression of ABC transporters. Our preliminary data suggests that longer-term cannabinoid exposure increases P-gp expression at the blood brain barrier. Thus, chronic cannabinoid exposure may reduce brain levels of antipsychotic drugs. Taken together, this project will help illuminate a novel mechanism for antipsychotic drug interactions.

TECHNIQUES knockout mice, behavioural analysis, western blotting, visualisation/quantification of dendritic morphology

PROJECT 2  ROLE OF ABC TRANSPORTERS IN CANNABIS-ANTIPSYCHOTIC DRUG INTERACTIONS

A quarter of schizophrenia patients use cannabis and there is little research examining the beneficial or harmful effects of cannabis use on antipsychotic drug therapy. This project aims to investigate whether cannabis use might alter the effectiveness of antipsychotic treatment in schizophrenia patients. Many antipsychotic drugs are substrates for ABC transporters. These transporters are localized at the blood brain barrier where they bind substrate drugs and transport them out of the brain back into the peripheral blood supply. Our work has shown acute cannabinoid exposure inhibits the transport function of the ABC transporters P-gp and BCRP. Therefore, cannabis-using schizophrenia patients may have increased CNS retention of antipsychotic drugs that would either assist in reducing schizophrenia symptoms and/or increase the incidence of side effects. An alternate mechanism whereby cannabis might affect the brain retention of antipsychotic drugs is by altering the expression of ABC transporters. Our preliminary data suggests that longer-term cannabinoid exposure increases P-gp expression at the blood brain barrier. Thus, chronic cannabinoid exposure may reduce brain levels of antipsychotic drugs. Taken together, this project will help illuminate a novel mechanism for antipsychotic drug interactions.

TECHNIQUES knockout mice, behavioural analysis, laser capture microdissection (LCM), qPCR, western blotting, analytical techniques (HPLC and GCMS)

PROJECT 3  DOES DIETING CAUSE CANNABINOID RE-INTOXICATION IN HUMANS?

The main psychoactive constituent of cannabis, THC, is stored in fat for significant periods of time which explains its long elimination half-life. We have recently demonstrated in THC-treated rats that dieting or stress, by promoting fat breakdown, cause THC to be released back into the blood. Accordingly, it is possible that individuals who have kicked their cannabis habit for some time, who decide to go on a diet, may experience a sufficient increase in THC blood levels causing them to be “spontaneously” intoxicated. This phenomenon we have termed “re-intoxication” and it has significant implications for cannabis-related medicolegal cases. This project aims to demonstrate cannabis re-intoxication in human users. Cannabis withdrawing patients will undergo 24 hours of dieting and we will measure whether this increases THC blood levels that correlates with neuropsychological impairment.

TECHNIQUES human study, neuropsychological tests, analytical techniques (HPLC and GCMS)

Our research group is interested in normal synaptic function and synapse dysfunction. Synaptic dysfunction is emerging as a key player in many brain disorders. We use patch-clamp electrophysiology in brain slices, immunohistochemistry and biochemical assays to study synaptic properties and synaptic plasticity that may participate in physiological or pathophysiological processes. These honours projects focus on how endogenously released opioid peptides alter synaptic function and plasticity in the amygdala. Fear and anxiety are adaptive responses that allow animals to defend themselves against harm. Neural circuits in the amygdala are key for fear memory acquisition and storage but also for reducing the fear response (extinction). Extinction of the fear response relies on a special group of GABAergic interneurons in the amygdala, the intercalated cells.

**PROJECT 1  Do opioids inhibit cortical glutamate release onto amygdala neurons?**

The intercalated cells can be activated by glutamatergic inputs/synapses from the cortex and from the basolateral amygdala. When activated these cells inhibit the amygdala output neurons and reduce the expression of the fear response. Intercalated neurons express very high levels of the μ-opiate receptor (MOR) and the endogenous opioid ligand enkephalin. We are interested in whether endogenous opioids modulate intercalated cell activity. This project will study whether the glutamatergic inputs from the cortex are inhibited by opioids.

**TECHNIQUES**  In vitro patch-clamp electrophysiology

**PROJECT 2  Does stress change endogenous opioid expression in the amygdala?**

Enkephalins are endogenous opioids that are strongly expressed in the amygdala and are thought to be involved in several aspects of fear. Mice deficient in the enkephalin precursor, preproenkephalin, are highly anxious and aggressive. Intercalated neurons (IA in figure) express very high levels of the μ-opiate receptor (MOR) and the endogenous opioid ligand enkephalin. Stress or anxiety may change enkephalin expression in the amygdala. This project will determine whether a stressful experience alters enkephalin expression in the intercalated cells.

**TECHNIQUES**  Immunohistochemistry, biochemistry

**PUBLICATIONS.**


PROJECT 1 What makes an asthmatic lung asthmatic?

The structure of an asthmatic airway is different to a nonasthmatic airway, including changes in the amount and type of extracellular matrix proteins (fibrotic changes, blue and pink fibres in picture) and an increase in the bulk of the airway smooth muscle cells (yellow band in picture). We know that the airway smooth muscle cell is a key player in regulating the fibrotic changes. We want to learn whether cells from asthmatic and nonasthmatic donors have differences in their ability to synthesize matrix proteins and the content of the resulting matrices. In addition, we will assess the intracellular signalling events leading to the fibrotic changes, with a focus on whether there are differences between the asthmatic and nonasthmatic cells.

TECHNIQUES

- cell culture, real time live cell imaging and immunofluorescence and confocal microscopy, real time PCR, ELISAs, cell behavioural assays including proliferation and migration

PUBLICATIONS.

1. Johnson PRA, Roth M, Tamm M, Hughes JM, Ge, Q, King, G, Burgess, JK and Black JL. 2001 Airway smooth muscle cell proliferation is increased in asthma. American Journal of Respiratory and Critical Care Medicine, 164, 474-477.


Our research group is primarily focused on how chemotherapy drugs (currently used and new agents) alter the local and systemic inflammatory response. Our group has shown that inflammation impacts the pharmacological response to chemotherapy in terms of response and toxicity.

We conduct both clinical and preclinical investigations of the response and toxicity induced by chemotherapy drugs to further understand how to improve the treatment of patients with cancer. New drugs are also tested in our research group to limit the toxicity of the current anti-cancer treatments.

**PROJECT 1  Response of MDSC to chemotherapy**

This challenging project is designed for an honours student with an interest and background knowledge in pharmacology, pathology and immunology. The project aims to analyse the changes in myeloid derived suppressor cells (MDSCs) in response to chemotherapy. MDSC are key immunosuppressive cells that are found in higher numbers in patients with cancer. Understanding the behaviour of MDSCs during therapy remains unknown and is key to determining pharmacological interventions for novel cancer therapy.

We have developed a multi-colour flow cytometry assay to measure MDSC in blood, which will be applied to blood samples collected from patients with cancer before and during chemotherapy. Dynamic changes in MDSC phenotype and number will then be correlated with markers of response and toxicity.

**TECHNIQUES**  Clinical sample processing, Blood cell isolation, Flow Cytometry and Biostatistics
Projects in the Chemical Biology in Drug Discovery group sit at the interface of chemistry, biochemistry and microbiology - often with a dash of biotechnology. We isolate/prepare compounds used to treat iron overload, infection and cancer; and study function. Some projects use traditional chemical synthesis, and other projects use bacteria to produce compounds, which we purify from culture using a specialist technique we designed in our group. New platforms for drug design are also explored.

**PROJECT 1  EXPLOITING BACTERIAL BIOSYNTHETIC MACHINERY FOR MOLECULAR DIVERSITY**

*Will suit a student with interests in microbiology/biochemistry (no chemistry required).*

Siderophores are compounds produced by bacteria that bind iron(III) with high affinity. These complexes form as the first step in the cascade that directs the supply of iron to the bacterial cytoplasm, which is essential for growth. The ability to bind metal ions confers value upon siderophores as inhibitors of metalloproteins; and as anticancer and anti-infective agents, through iron starvation. The macrocyclic siderophore putrebactin (at right) native to *Shewanella putrefaciens* is assembled via a four-component enzyme system, beginning with the Odc-catalysed decarboxylation of ornithine to give precursor 1,4-diaminobutane (blue). Our group has demonstrated that inhibition of Odc directed the biosynthesis in *S. putrefaciens* of a non-native siderophore, assembled from 1,5-diaminopentane. In this project, you will use *S. putrefaciens* as a biosynthetic machine to produce novel siderophores by augmenting media with combinations of Odc inhibitors and non-native diamine precursors.

**TECHNIQUES**
- Precursor directed biosynthesis
- Bioinformatics
- Purification
- LC-MS
- Isotopic labelling

**PROJECT 2  PROBING DESIGN PRINCIPLES OF HISTONE DEACETYLASE INHIBITORS**

*Will suit a student with an interest in medicinal chemistry (chemistry required).*

We recently prepared a library of hydroxamic acid-based inhibitors of Zn(II)-containing histone deacetylases (HDACs), which are frontline oncology targets. The properties of the compounds, which were structurally related to trichostatin A (high potency HDAC inhibitor, IC₅₀ 12 nM), were explored through HDAC inhibition profiles, anti-proliferative activity against BE(2)-C neuroblastoma cells, and computer modelling of interactions at the HDAC active site, which provided unexpected insights into the drug design principles for these agents. In this project, you will prepare and characterise a second-generation library of HDAC inhibitors, which explore the effects of contributing resonance structures; and amide bond position, on potency.

**TECHNIQUES**
- Synthesis/characterisation
- Fluorometric HDAC inhibition assay
- Computer modelling

**PROJECT 3  EFFICIENT CAPTURE OF DOXORUBICIN VIA METAL AFFINITY CHROMATOGRAPHY**

In February 2012, Australia faced a shortage of the anticancer agent doxorubicin, used to treat children with leukaemia and adults with Hodgkin’s lymphoma. In our laboratory, we have discovered a new metal affinity capture technique, which is effective in selecting high value bacterial metabolites from culture. In this project we will optimise the technique towards doxorubicin and isolate native doxorubicin from cultures of *Streptomyces peucetius* (ATCC27952).

**TECHNIQUES**
- Immobilised metal affinity chromatography
- Biodiscovery
- Characterisation

Sarah Hilmer leads a geriatric pharmacology research group based at Royal North Shore Hospital. We study pharmacology in ageing, aiming to improve the safety and efficacy of medicines for older people. Using basic experimental pharmacology, we study the hepatic disposition and hepatotoxicity of drugs in our Laboratory of Ageing and Pharmacology in the Kolling Institute. Our clinical pharmacology research measures risk and benefit of drugs in fit and frail older people. Pharmacology honours students are co-supervised by Dr Slade Matthews (slade.matthews@sydney.edu.au) and Professor Peter Carroll (peter.carroll@sydney.edu.au), with associate supervisor Dr Danijela Gnjidic (danijela.gnjidic@sydney.edu.au).

**PROJECT 1  High risk medication exposures and clinical outcomes in older adults**

Several patterns of medication exposure increase the risk of adverse outcomes in older patients. These include exposure to multiple medicines (polypharmacy), anticholinergic and sedative drugs (high risk medicines) and drug-drug interactions (DDIs). The combined effect of these exposures on clinical outcomes is unknown. The student will collect data from a cohort of older patients in hospital and describe (1) prevalence of high risk medication exposures; (2) clinical outcomes of individual and concurrent exposures over three months.

**TECHNIQUES**  Clinical research (ethics, data collection from patients and records, data analysis)

**PROJECT 2  The effect of old age and frailty on fentanyl pharmacokinetics and pharmacodynamics**

In old age, there is an increase in inter-individual variability in clinical pharmacology. Older people may be described as robust or frail. Frail older people have many features that may impact on pharmacokinetics and pharmacodynamics, such as loss of muscle mass, chronic inflammation and increased risk of disease. This project will compare the pharmacokinetics and pharmacodynamics of fentanyl, a hepatically metabolised drug, in young, old robust and old frail patients. The student will recruit a cohort of patients, assess frailty using a clinical scale and biomarkers, assess pharmacokinetics from blood samples, and pharmacodynamics from clinical measures. The results will determine whether frailty is a useful guide of drug distribution, clearance and response.

**TECHNIQUES**  Clinical research (ethics, data collection from patients, records), ELISA, PKPD modelling

**PUBLICATIONS.**
These projects form part of a wider collaboration that seeks to understand the link between the role of GABA and stress-induced modulation of disease states including anxiety, depression, schizophrenia and cardiovascular disease.

PROJECT 1 NATURAL AGENTS AFFECTING GABAA RECEPTOR FUNCTION

This project is to be carried out in collaboration with Dr Ann Mitrovic (ann.mitrovic@sydney.edu.au) and eProf Graham Johnston (grahamj@mail.usyd.edu.au).

There is a great deal of popular and scientific interest in natural chemicals such as flavonoids, alkaloids and amino acids found in the foods we consume which have been linked to numerous actions, including a reduction, or increase, in stress and anxiety. Many of these chemicals are also able to modulate receptors in the brain and periphery for the inhibitory neurotransmitter, GABA. Some of these chemicals may actually have useful actions on GABA receptor function in the brain, for example by acting as anti-stress and anti-anxiety agents, and thus represent important foods for the brain. This project is directed at studying the effects of chemicals found in foods and beverages on cloned GABA receptors.

TECHNIQUES molecular biology to synthesise GABA receptor RNA, expression of recombinant GABA receptors in *Xenopus* oocytes, and *in vitro* testing, through electrophysiology, of extracts from teas as well as other natural active constituents.

PROJECT 2 STRESS, TEA AND GABA

This project is to be carried out in collaboration with Dr Slade Matthews (see Dr Matthews’ project descriptions (slade.matthews@sydney.edu.au)) and eProf Graham Johnston (grahamj@mail.usyd.edu.au).

The anxiolytic and sedative properties of GABAergic compounds are well described and their use is prevalent in our community. There exist a plethora of teas available which claim to have calmative effects or to assist with stress. Some of these teas are known to contain GABA and GABAergic compounds, and therefore have some reasonable scientific basis to their claim, but questions regarding content levels and bioavailability remain. Several teas containing GABA or GABAergic compounds have been identified and will be assayed in both chemical and physiological ways in this project.

TECHNIQUES high performance liquid chromatography of commercially available tea preparations; human testing of the effects of teas on heart rate variability as a biomarker of stress.

PUBLICATIONS.

Professor Michael KASSIOU  
Drug Discovery Research Unit  
Brain & Mind Research Institute  
michael.kassiou@sydney.edu.au

Drug discovery research within my group is multidisciplinary and at the interface between chemistry and biology. The research is primarily concerned with the understanding of drug-protein and drug-binding site interactions in order to obtain structure-activity relationships of bioactive CNS molecules. This allows the rational design of more efficacious treatments for diseases of the brain.

PROJECT 1  P2X7 RECEPTOR LIGANDS IN THE TREATMENT OF DEPRESSION

Activation of P2X7 receptors (P2X7R) by ATP stimulates the release of interleukin-1β (IL-1β) (1), inducing behavioural changes that resemble depression. It is hypothesised that blockade of P2X7Rs might result in antidepressant-like properties. This project will determine the ability of P2X7R molecules, newly developed by our group, to reduce IL-1β levels and the evaluation of lead molecules in rodent antidepressant behavioural studies.

TECHNIQUES  Cell culture, in vitro functional assays, animal behaviour

PROJECT 2  CHARACTERISING THE INTERACTION OF THERAPEUTIC DRUGS AT THE TRANSLOCATOR PROTEIN

The translocator protein (TSPO) is a potential drug target for the treatment of CNS diseases, with TSPO ligands being able to modulate steroidogenesis, apoptosis, and cell proliferation (2). While there exist multiple TSPO binding sites, the nature of these sites - either overlapping or allosterically linked - remains largely uncharacterized. Furthermore, while evidence suggests that microglial activation and polymerization result in changes to TSPO binding sites, these changes are poorly understood. This project will involve characterising the nature of binding and functional consequence of a variety of novel TSPO ligands generated in our laboratory.

TECHNIQUES  In vitro binding, SAR, molecular modelling

PROJECT 3  SIGMA LIGANDS AS NEUROPROTECTIVE AGENTS IN BRAIN DISEASE

During brain injury, microglia become activated and migrate to areas of degenerating neurons. These microglia release proinflammatory cytokines and reactive oxygen species causing additional neuronal death. Microglia express high levels of sigma receptors, however, the function of these receptors in microglia and how they may affect the activation of these cells remain poorly understood. This project will evaluate the ability of a library of sigma ligands attenuate the release of nitric oxide and cytokines.

TECHNIQUES  Cell culture, cell based assays, ELISA

REFERENCES.  
The PharmacoInformatics Laboratory uses computer technologies to uncover previously unknown relationships in biomedical data. PharmacoInformatics incorporates the principles of computerised data management, machine learning techniques and complexity analysis in a pharmacology context. These techniques as well as applied statistics are used on a range of problems in this lab including clinical observational studies and laboratory based data driven studies.

**PROJECT 1 STRESS, TEA AND GABA**

This project is to be carried out in collaboration with Dr Tina Hinton (see Dr Hinton’s project descriptions (tina.hinton@sydney.edu.au)) and eProf Graham Johnston (grahamj@mail.usyd.edu.au). The anxiolytic and sedative properties of GABAergic compounds are well described and their use is prevalent in our community. There exist a plethora of teas available which claim to have calming effects or to assist with stress. Some of these teas are known to contain GABA and GABAergic compounds, and therefore have some reasonable scientific basis to their claim, but questions regarding content levels and bioavailability remain. Several teas containing GABA or GABAergic compounds have been identified and will be assayed in both chemical and physiological ways in this project.

**TECHNIQUES**

- high performance liquid chromatography of commercially available tea preparations;
- human testing of the effects of teas on heart rate variability as a biomarker of stress.

**PROJECT 2 GAMMA GLOBULIN BIOMARKER-DOSE EFFICACY STUDY**

It is possible to measure gamma globulin in the serum in various clinical states? We aim to measure pre and post gamma globulin administration levels. Dosages of Gamma Globulin are suspected to be elevated for 3-4 weeks following a specific single dose, in some conditions the elevated response is less than 5 days. Are elevated levels of gamma globulin are dosage dependent? Gamma Globulin Drives Th2-inducing T cells and stimulates IL-4. Measurement of IL-4 as a biomarker for gamma globulin dose-dependent changes in IL-4. This work aims to improve the efficient use of precious/rare blood-derived resources in the Australian Community.

**TECHNIQUES**

- Data Analysis and Laboratory Based Techniques at Australian Red Cross Laboratories.
- External supv.: Dr Wayne Dwyer, Dr Hugh Capper

**RECENT PUBLICATIONS:**

4. Craig S. McLachlan, PhD, MPH, Ryan Ocsan, MSc, Ian Spence, PhD, Brett Hambly, MD, PhD, Slade Matthews, PhD, Lexin Wang, MD, PhD, and Herbert F. Jelinek, PhD. (2010) Increased total heart rate variability and enhanced cardiac vagal autonomic activity in healthy humans with sinus bradycardia. Proc (Bayl Univ Med Cent) 23(3):1-3.
1) Asthma and chronic obstructive lung disorders

**PROJECT 1** Inhibiting airway contraction

The epithelium provides a barrier between the outside and the inside of the body. People with asthma have airways that are too sensitive and narrow too much. Perhaps in asthma a relaxant factor is not released to the same extent in healthy airways. This project will be investigating the effect of an epithelial derived relaxing factor on contraction using a coaxial bioassay system.

**TECHNIQUES** Surgical skills, organ bath experiments, histology

**PROJECT 2** Does mucosal folding in the airways decrease airway narrowing?

In asthma the airways narrow too easily and too much. When the airways narrow the tissue internal to the airway smooth muscle layer buckles and forms folds (mucosal folding). To date it is unknown whether the buckling of the layer serves as a breaking mechanism to decrease the degree of narrowing. Understanding factors which alter the degree of narrowing help us to understand how changes in structure affect function. For this project narrowing will be assessed using porcine airways obtained from pigs killed at abattoir.

**TECHNIQUES** Dissecting skills, organ bath experiments, histology;

Tick borne diseases (TBD) group includes Dr Brent McParland, Dr Ann Mitrovic and Ann Cincotta.

Our group investigates:
- vectors that transmit TBD
- animal reservoirs that propagate the micro-organisms that cause TBD
- clinical presentation of TBD and impact on health
- role of immune system dysregulation in TBD

PROJECT 1  Tick borne diseases: Are they a threat to the health of Australians?

Human tick borne diseases (TBD) are not well characterised in Australia and the potential health impacts have not been carefully assessed. TBDs such as Borreliosis, or Lyme disease (LD), are controversial in Australia, with a high percentage of clinicians believing LD cannot be locally acquired. Babesiosis, a serious TBD in livestock in Australia, was also thought not to be a threat to Australians, until March 2012 when the first report of a locally acquired case was published. Other TBD that will be investigated include Anaplasmosis, Ehrlichiosis, Bartonellosis, Tullaremia and Rickettsiosis. Detection of pathogen DNA in ticks, reservoir animals and clinical samples by PCR will be analysed using multi-locus sequence analysis.

TECHNIQUES
Genomic DNA purification from ticks, animal tissue and clinical specimens. PCR - Polymerase Chain Reaction, electrophoresis, light and dark field microscopy. Analysis of phylogenetic relationships using multi-locus sequence analysis (MLSA).

PROJECT 2  Lyme Disease: The great imitator

Lyme disease is said to be the great imitator of the 21st century. The causative agent is a spirochete, Borrelia burgdorferi sensu lato, and can cause a broad spectrum of clinical symptoms, similar to the great imitator of the 20th century, syphilis, caused by another spirochete Treponema pallidum. In Europe and USA, Lyme disease has been found to “imitate” the clinical symptoms of such conditions such as; chronic fatigue syndrome, motor neurone disease, multiple sclerosis, Parkinson’s disease, Alzheimer’s disease, and arthritis amongst others. We will conduct pilot studies of patients diagnosed with these syndromes that have a history of tick bite to look for evidence of exposure to Borrelia by using serology and PCR.

TECHNIQUES
Genomic DNA purification from clinical specimens. PCR, electrophoresis, light and dark field microscopy. Analysis of phylogenetic relationships using multi-locus sequence analysis (MLSA). Western blot to detect IgG, IgM, antibodies against B. burgdorferi sensu stricto.

PUBLICATIONS.
Glioblastoma (GBM) is a fatal brain tumour of glial origin and my goal is to contribute to the development of better GBM treatments by targeting the inflammatory tumour microenvironment. Our current research is directed towards better understanding of the inflammation-cancer axis in GBM pathophysiology and development of novel kinase inhibitors that may find utility as anti-cancer agents. We aim to achieve this through integration of molecular pharmacology, cell biology and medicinal chemistry.

**PROJECT 1  Testing novel kinase inhibitors in brain tumour stem cells**

Brain Tumour Stem Cells (BTSCs) are a subpopulation of GBM cells that display much greater tumourigenic potential than non-stem GBM cells and thereby more closely resemble the genotypic and phenotypic characteristics of the original GBM and recapitulate many of its properties in vitro. Furthermore, the stem-like cells are most likely the cells of origin for tumour relapse as they exhibit a poor response to therapy. This project will examine anti-inflammatory and anti-invasion efficacy of novel kinase inhibitors in BTSCs in order to minimise the development of a therapeutic for which GBM could be resistant.

**TECHNIQUES**
- cell culture
- immunoblotting
- ELISA
- migration and invasion assays

**PROJECT 2  Novel anti-metastatic agents: mode of action in tumour cells**

Metastasis is the major life-threatening consequence of malignant tumours. At present there are no effective drugs to prevent metastasis. In our current work we have designed a new class on anti-cancer agents that inhibit the growth and migration capacity of highly aggressive breast and prostate tumour cells. Understanding how these agents prevent tumour cell migration will identify new drug targets for the rational design of next generation anti-cancer drugs.

**TECHNIQUES**
- cell culture
- immunoblotting
- real-time PCR
- medicinal chemistry
- cell-based assays

**PROJECT 3  Development of novel MK2 inhibitors**

Mitogen activate protein kinase-activated protein kinase 2 (MK2) is a Ser/Thr protein kinase that controls cytoskeletal architecture, cell-cycle progression and is implicated in inflammation and cancer. MK2 is a direct substrate of p38 MAPK and is important for cytokine production. MK2 knock-out mice have a normal phenotype, but are resistant to develop inflammatory diseases; suggesting that MK2 may be a safe target for therapeutic intervention. This project involves design and synthesis of novel MK2 inhibitors using microwave-assisted organic synthesis. Pharmacological evaluation of novel agents will include in vitro kinase assays and inflammatory assays using microglia and glioblastoma cells.

**TECHNIQUES**
- medicinal chemistry
- SAR studies
- cell-based inflammation assays

My research group investigates the pathophysiology of respiratory diseases, with a particular emphasis on understanding basic mechanisms leading to disease exacerbations and progression.

**PROJECT 1**  **Is rhinovirus-induced inflammation different in COPD?**

The prevalence of COPD continues to rise, currently affecting 3.5% of the Australian population, causing significant mortality (4th leading cause of death worldwide) and morbidity. Exacerbations of COPD result in approximately 50,000 hospitalisations annually in Australia, and are associated with a more rapid disease progression. Until recently exacerbations were thought to be caused by only bacteria; however viruses are now emerging as important precipitants, with up to 50% of exacerbations being caused by viruses. It is therefore vital to better understand the role of viral infections in exacerbations of COPD. This project will establish if the innate immune response to rhinovirus is: 1) intrinsically different in lung cells from people with COPD, and 2) impaired by current therapeutic regimes.

**TECHNIQUES**  Cell culture, virus assays, qPCR, Western blotting, ELISA

**PROJECT 2**  **Do epigenetic changes occur in COPD?**

Chronic obstructive pulmonary disease (COPD) is a major global cause of ill-health and mortality which is increasing in prevalence worldwide and constitutes a huge socioeconomic burden. Whilst the aetiology of COPD is multifactorial, the main risk factor in western societies is cigarette smoking. In COPD the majority of the disease burden is caused by fibrosis of the small airway. In ground-breaking experiments we have discovered that cigarette smoke extract triggers the release of two key ECM proteins, fibronectin and perlecan, in airway cells from people with COPD, whilst in cells from people without COPD no such release occur. In this project you will investigate if epigenetic and/or cell signalling abnormalities result in fibroblasts from patients with COPD producing ECM in response to cigarette smoke extract.

**TECHNIQUES**  Cell Culture, ELISA, Molecular Biology

**PROJECT 3**  **How does rhinovirus reduce the efficacy of asthma medications?**

During virus-induced asthma exacerbations bronchodilators such as β2-agonists do not work as well as they do at other times. In our previous studies we have found that the infected bronchial epithelium drives this phenomenon, for example it promotes desensitisation of the β2-adrenocceptor on airway smooth muscle cells. In our recent studies, in collaboration with A/Profs Traini and Young we have begun to investigate how the transport of bronchodilators across the epithelial is affected by rhinovirus infection. In this study you would assess how the transport of β2-agonists across epithelial cell layers is affected by rhinovirus infection, toll like receptor agonists (to begin to understand which aspect of virus replication are involved), and virus-induced cytokines (to model what happens to cells not infected by rhinovirus)

**TECHNIQUES**  Cell culture, qPCR, western blotting, ELISA, HPLC

**SELECTED PUBLICATIONS.**  
Glutamate is the predominant excitatory neurotransmitter in the mammalian central nervous system and activates a wide range of receptors to mediate a complex array of functions. Extracellular glutamate concentrations are tightly controlled by a family of glutamate transporters expressed in both neurons and glia. The aim of our research is to develop a structural model for how glutamate transporters work, and in this way lay the foundations for a more rational approach to the development of drugs that are transporter-specific and subtype selective. Such compounds will help to delineate the roles of different transporter subtypes in normal brain functions and also in various neuropathological conditions, such as ischemia following a stroke, Alzheimer’s disease, motor neurone disease and obsessive compulsive disorder.

PROJECT 1 Understanding the mechanism of transport by the glutamate transporter family using chimeras and site-directed mutagenesis

The glutamate transporter family is made up of proteins from several species and includes the human glutamate transporters (EAATs) and neutral amino acid transporters (ASCTs), and a prokaryotic aspartate transporter GltPh. All of these transporters share significant amino acid homology and share some functional properties but also exhibit some differences. For example, the EAATs are secondary active transporters of acidic amino acids while the ASCTs are electroneutral exchangers of neutral amino acids (see figure below).

The aim of this project is to exploit the similarities and differences between these 3 transporters by making chimeric transporters and performing site-directed mutagenesis to identify the molecular determinants for substrate selectivity and ion-coupling in the glutamate transporter family. Glutamate transporter dysfunction has been implicated in disease states such as ischemia following a stroke, Alzheimer’s disease and obsessive compulsive disorder. The expression of the neutral amino acid transporter ASCT2 is known to be upregulated in some prostate, breast and skin cancers. Through a better understanding of the mechanism of these transporters we can develop compounds that may have therapeutic benefits in these disease states.
Protein misfolding and the formation of stable, fibrillar protein deposits known as amyloid fibrils are associated with many diseases. The process of misfolding and aggregation that leads to fibril formation can disrupt proteostasis in the cell. Amyloid protein fibrils that have similar structural features but which have essential biological functions have recently been identified in mammals and many different microorganisms. In these cases the amyloid self-assembly is advantageous to the organism. The Sunde lab uses a range of molecular biology, protein chemistry and structural techniques to study the formation and structure of amyloid fibrils, both from disease states and from Nature.

PROJECT 1  Amyloid fibril formation by hydrophobin proteins involved in fungal infections

Fungal spores are coated with amyloid fibrils made from hydrophobin proteins. These amyloid fibrils form a protective coating on the spores. In infection of humans by *Aspergillus fumigatus*, the hydrophobin coating prevents activation of the immune system, allowing prolonged infection of the host and, in severe cases, development of invasive aspergillosis. In rice blast, a disease that causes loss of 25% of the world-wide rice crop annually, spores from the fungus *Magnaporthe grisea* are coated with hydrophobin fibrils that facilitate attachment to the rice leaf and infection of the plant. You will study the structure and formation of these functional amyloid fibrils in order to understand the mechanism of their interaction with the host.

**TECHNIQUES**  Molecular biology, protein expression and purification, fibril formation assays and fluorescence studies

PROJECT 2  Engineering amyloid fibrils as drug delivery vehicles

The hydrophobin protein RodA from the fungus *Aspergillus fumigatus* forms monolayers composed of amyloid fibrils that do not stimulate the immune system. The protein can be engineered to incorporate amino acid sequences that are recognised by particular receptors and can also be covalently modified to carry fluorescent reporter molecules. You will carry out protein engineering on RodA to develop forms of the protein that carry specific targeting and reporter sequences. You will coat silicon nanoparticles with modified RodA fibrils in order to prepare drug-loaded, targeted nanoparticles, determine the stability and biocompatibility of these constructs and determine whether they are taken up by the targeted cells.

**TECHNIQUES**  Protein expression and purification, amyloid assembly assays, cell culture, fluorescence and electron microscopy

**PUBLICATIONS.**


A/Prof Daniela Traini - ARC Future Fellow
Respiratory Technology
The Woolcock Institute of Medical Research-Glebe
daniela.traini@sydney.edu.au

Dr Traini’s research explores respiratory drug delivery science. It focuses on understanding the physical properties of materials used in pharmaceutical sciences and then in relating those to in-vitro and subsequent in-vivo performance. She has expertise in projects related to asthma, chronic obstructive pulmonary diseases and bronchiectasis. High-end imaging is also one of her interests. She is currently working toward understanding the co-formulation and co-deposition of inhalation active pharmaceutical ingredients to enhance their synergistic therapeutic effect.

PROJECT 1  Investigation into macrophages migration on the lung surface

Cell line lung models representative of the lung epithelium (Calu-3) and alveolar region (A549), will be used to study macrophages mobility and phagocytosis post inhalation drug deposition, in a simulated lung fluid media, on a ‘thin film’. In addition drug dissolution in this media will be measured to ascertain the effects of lung surfactant on micro-nano sized-particle dissolution. (Left) Fluorescence microscope image of uptake fluorescent polymer microspheres by alveolar macrophages (blue staining is nucleolus and red cytoplasm). (Right) Uptake of 4.16 μm particles by macrophages as a function of particle concentration.

TECHNIQUES  Live Cell Imaging; Dissolution testing; HPLC

Co-supervisors: A/Prof Young and Dr Brian Oliver

PROJECT 2  Nebulization of nanoparticle using per-fluorinated liquids

Targeting lung alveolar macrophages is an attractive approach for Tuberculosis therapy since is where the infesting mycobacteria are. By delivering nano-particles to these sites, the mortality of the parasite could be enhanced. The incorporation of nano-particles into per-fluorinated liquids by nebulization overcomes the drawbacks of delivering particles to this region. This project will study nano-particle suspension with respect to aerosol performance as nebulizers. Drug deposition on macrophage uptake and cell viability could also be studied. (Left) Beclomethasone dipropionate spray-dried; (Right) After exposure to HPFP

TECHNIQUES  Particle size, Aerosol deposition, Spray-drying, DSC, DVS, XRPD, HPLC, SEM

Co-supervisors: A/Prof Young and Dr Brian Oliver

PROJECT 3  Repurposing: the use of old drugs for new therapeutic outcomes

Many drugs like Simvastatin (SV-hypolipidemic) and Erythromycin (ER-antibiotic) have been found to have ‘other’ therapeutic effects. For example the SV has an inhibitory effect on mucus production while ER is an anti-inflammatory. In this study, these drugs will be physico-chemically characterised, formulated for inhalation delivery and their effect on in vitro lung cell line studied. RGB ratio of Alcian blue intensity following SV exposure. Data represents mean ± SD (n=3) (D)

Co-supervisors: A/Prof Young and Dr Brian Oliver

TECHNIQUES  Particle size, Aerosol deposition, TEER, Mucus inhibition studies

Research in the Transporter Biology Group is focused on understanding the molecular basis for neurotransmitter transporter functions and how this can be manipulated by endogenous regulators and pharmacological agents. Glycine is an unusual neurotransmitter in that it acts on inhibitory glycine receptors and excitatory NMDA receptors. The Glycine Transporter GLYT1 regulates the concentrations of glycine at excitatory synapses, whilst a combination of GLYT1 and GLYT2 are required for regulation of glycine at inhibitory synapses. GLYT1 inhibitors are currently being developed for the treatment of schizophrenia, whilst GLYT2 inhibitors may have potential as analgesics in the treatment of chronic pain.

PROJECT 1  Lipid Inhibitors of GlyT2

The Transporter Biology Group has identified a class of novel lipids that inhibit GlyT2 which have potential for use as analgesics. This project will investigate how these lipids inhibit the transporter and also how endogenous cell membrane lipids influence the function of GlyT2. Figure to the right shows the exterior surface of GlyT2 with potential lipid binding sites highlighted in blue, red and yellow. A number of research directions and techniques are possible with this project. Students are encouraged to discuss these projects with Professor Vandenberg so that the style of the project can be tailored to the student’s interests. Collaborative projects with medicinal chemists and neurophysiologists may also be possible.

TECHNIQUES  Molecular biology, site-directed mutagenesis, electrophysiology, molecular modelling

PUBLICATIONS.


Our group is focused on the actions of novel analgesics, particularly those based on cannabis-related agents, in models of chronic pain (inflammatory and neuropathic pain). We also examine the cellular actions of these agents on pain pathways within the central nervous system in order to understand how they work and to improve their efficacy. This work is carried out using behavioural techniques in chronic pain models to determine drug efficacy, plus in vitro techniques to examine how these drugs alter communication between neurons in identified pain pathways.

**PROJECT 1  Chronic Pain & Endocannabinoids**

Chronic pain syndromes particularly those caused by injury to the nervous system are resistant to traditional analgesics such as opioids. The psychoactive ingredient in marijuana, THC, has been shown to be effective in alleviating these pain syndromes by acting on an endogenous cannabinoid neurotransmitter system. Recently, a number of agents that modulate the body’s own endocannabinoid neurotransmitter system have been identified. The project would examine whether these novel endocannabinoid modulators have pain relieving activity in animal models of chronic pain (as in Jayamanne et al, 2006).

**TECHNIQUES**
In vivo chronic pain model - surgery, pain behavioural testing, drug administration (systemic & intrathecal)

**PROJECT 2  Cannabinoid/opioid interactions in a neuropathic pain model**

Chronic pain syndromes particularly those caused by injury to the nervous system are resistant to traditional analgesics such as opioids. The psychoactive ingredient in marijuana, THC, has been shown to be effective in alleviating these pain syndromes by acting on an endogenous cannabinoid neurotransmitter system. Recently, it has been suggested that together opioid and cannabinoid agonists might act synergistically to relieve chronic pain. The project would examine whether opioid/cannabinoid combinations have enhanced pain relieving activity in an animal model of chronic neuropathic pain (as in Jayamanne et al, 2006).

**TECHNIQUES**
In vivo chronic pain model - surgery, pain behavioural testing, drug administration (systemic)

My research group develops new approaches and tools to study and treat respiratory diseases. We focus on developing new medical devices and advanced formulations that can target specific regions of the lung.

**PROJECT 1 Developing new medical devices to treat respiratory disease**

While there are a multitude of inhalers available on the market to treat asthma, the delivery of the active ingredient is inherently inefficient and there is no control of regional deposition (in many cases <30% of the drug will reach the respiratory tract). This project will study how device design affects lung deposition with a view to design a new inhaler capable of targeted and efficient delivery. You will gain experience in the areas of device design, rapid prototyping (3D-printing), *in vitro* aerosol analysis and formulation. This project will result in advances in drug delivery to the respiratory tract. (Co-supervisor D. Traini)

**TECHNIQUES** Rapid prototyping, computational methods, aerosol characterisation, in vitro, HPLC

**PROJECT 2 Developing new particulate systems to treat respiratory disease**

Respiratory tract infection is the number 1 cause of communicable disease worldwide. Currently treatment regimes involve ether oral delivery of antibiotics or, when in intensive care, antibiotic intravenous injection. A logical approach would be to deliver antibiotics by inhalation since this would reduce the required dose and the potential for antibacterial resistance. However, in order to achieve this the particles must have a diameter < 5µm and have enhanced residency time at the epithelia. In this project, you will gain experience in the area of particle engineering, state-of-the-art physico-chemical characterisation and drug delivery. We will design a novel inhaled antibiotic particle that has enhanced residence in the lung through its interaction with the epithelia/surface lung fluid. (Co-supervisors D. Traini, B. Oliver)

**TECHNIQUES** Particle engineering, in vitro testing, HPLC, microscopy, colloid science

**PROJECT 3 Understanding what happens at the respiratory epithelia**

Current cell culture protocols are designed to study cellular processes and mechanisms in liquid covered culture when a cell is exposed to µM drug concentrations. In the lung, however, respiratory cells are exposed to the ‘environment’ and thus establish a different morphology to that which would be observed in conventional cultures. This project will culture a series of respiratory cells under air-interface conditions and investigate the processes of drug uptake and transport when real drug particulate systems (i.e. see above) are deposited on their surface. You will gain experience in cell culture, drug delivery, protein analysis and advanced microscopy. (Co-supervisors B Oliver, D Traini)

**TECHNIQUES** Cell culture, western blotting, HPLC, microscopy, drug delivery

Where are they now?

Honours is a fantastic year in itself, but is also a springboard to postgraduate studies and careers in industry and government. Shown in the Table below are the current positions of a selection of students who have completed Honours or a Graduate Diploma in Pharmacology.

<table>
<thead>
<tr>
<th>Name</th>
<th>Completed</th>
<th>Current Position</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phuoc Huynh</td>
<td>2010</td>
<td>PhD Candidate (Pharmacology, University of Sydney)</td>
</tr>
<tr>
<td>Carleen Fernandez</td>
<td>2010</td>
<td>PhD Candidate (Centenary Institute)</td>
</tr>
<tr>
<td>Vivian Liao</td>
<td>2010</td>
<td>Research Assistant (Chemical Biology Group)</td>
</tr>
<tr>
<td>Dmitry Goloskokov</td>
<td>2010</td>
<td>Laboratory Aide (Douglass Hanly Moir Pathology)</td>
</tr>
<tr>
<td>Lauren Brites</td>
<td>2009</td>
<td>Research Assistant (EnGeneIC)</td>
</tr>
<tr>
<td>Sai Krishnan</td>
<td>2009</td>
<td>PhD Candidate (Children's Medical Research Institute)</td>
</tr>
<tr>
<td>Marietta Salim</td>
<td>2009</td>
<td>Research Assistant (Transporter Biology Group)</td>
</tr>
<tr>
<td>Areeg Hamdi</td>
<td>2009</td>
<td>Masters Candidate (Pharmacy, University of Sydney)</td>
</tr>
<tr>
<td>Steven Devenish</td>
<td>2008</td>
<td>PhD Candidate (Pharmacy, University of Sydney)</td>
</tr>
<tr>
<td>Nicholas Kortt</td>
<td>2008</td>
<td>Medicine (University of Notre Dame)</td>
</tr>
<tr>
<td>Phoebe Hone</td>
<td>2008</td>
<td>Research Assistant (Veterinary Science)</td>
</tr>
<tr>
<td>Cho Zin Soe</td>
<td>2007</td>
<td>PhD Candidate (Pharmacology, University of Sydney)</td>
</tr>
<tr>
<td>Jonathon Tobin</td>
<td>2007</td>
<td>Medicine (University of Wollongong)</td>
</tr>
<tr>
<td>Jessica Kermale</td>
<td>2007</td>
<td>PhD Candidate (Woolcock Institute of Medical Research)</td>
</tr>
<tr>
<td>Amelia Eddington</td>
<td>2007</td>
<td>PhD Candidate (Pharmacology, University of Sydney)</td>
</tr>
<tr>
<td>Alana Scarf</td>
<td>2007</td>
<td>PhD Candidate (Brain &amp; Mind Research Institute)</td>
</tr>
<tr>
<td>Chiu Chin Ng</td>
<td>2006</td>
<td>PhD Candidate (Pharmacology, University of Sydney)</td>
</tr>
<tr>
<td>Tim Bakas</td>
<td>2006</td>
<td>MPhil Candidate (Pharmacology, University of Sydney)</td>
</tr>
<tr>
<td>Brina Sheriff</td>
<td>2005</td>
<td>Poisons Information Centre</td>
</tr>
<tr>
<td>Nathan Gunasekaran</td>
<td>2005</td>
<td>PhD (University of Sydney), Medicine (University of Notre Dame)</td>
</tr>
</tbody>
</table>
Discipline of Pharmacology: Honours Preference Form (2013)

This form must be submitted to the Honours Coordinator by: Friday 23 November 2012.
An application for Honours must be lodged on line through the Faculty of Science.

I wish to apply for the following course in 2013 (circle choice):

- BSc (Hons)
- BSc Adv (Hons)
- BMedSc (Hons)
- Graduate Diploma

I intend starting my studies in (circle choice): Semester 1 or Semester 2.

STUDENT DETAILS:

First Name

Family Name

SID

E-mail (University of Sydney Account)

Postal address

Phone (home)

Phone (mobile)

STUDENT PREFERENCES:

Please list your preferences for an Honours supervisor (from 1st to 4th preference). You must provide 4 names.

1

2

3

4

STUDENT TRANSCRIPT:

Please attach your academic transcript (photocopy or original) to this application.

Return to: Dr Rachel Codd, Room 274 Blackburn Building (D06)