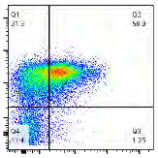
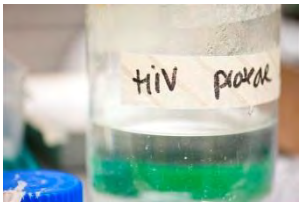
Department of Infectious Diseases

Central Clinical School, AMREP Campus

Monash University and Alfred Health



The Department of Infectious Diseases, Central Clinical School, and Alfred Health, is a premier centre for clinical and biomedical research and education, offering undergraduate and postgraduate study programs.

The Department of Infectious Diseases has clinical expertise in general infectious diseases (eg. pneumonia, meningitis, urogenital infection, cellulitis), tuberculosis, respiratory infections, HIV/AIDS, sexually transmissible infections, and travel related infections. The unit also specialises in antibiotic usage, infection control, and HIV palliative and continuing care, and has expanded its research to include international health, evidence mapping and health information technology.

The Department integrates clinical services with clinical and basic science research. The clinical services work closely with research staff and laboratories are based within the Burnet Institute, with a presence within the Central Clinical School.

Head of Department:

Prof Sharon Lewin

Student inquiries:

Jasminka Sterjovski (jasminka.sterjovski@monash.edu)

Research Teams:

The Lewin/Cameron Research Group

Head Sharon Lewin (left) and Deputy Head Dr Paul Cameron (right)

Headed by scientist physicians, Sharon Lewin (left) and Dr Paul Cameron (right), the lab is a melting pot of health related skill sets and backgrounds, combining clinical expertise with knowledge in pharmacology, immunology, molecular virology. The research focus is mainly on HIV and Hepatitis B infection but extends into all related co-morbidities and inflammation.

PROJECT: Study of Fibrosis and Immune Activation (SOFIA)

Contact person:

Dr Megan Crane

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Research group: Lewin/Cameron Laboratory

Overview:

Morbidity and mortality secondary to liver disease is greatly increased in people infected with both HIV and HBV compared to those infected with HBV alone. Mortality remains elevated even after treating both the HIV and HBV virus. The HBV Immunology Lab investigates the mechanism of how HIV can accelerate liver disease in patients co-infected with HBV. We hypothesize that this occurs by combined effects of HIV and HBV on inflammation in the liver. These studies could potentially lead to new treatments for liver disease.

PROJECT: Towards an HIV Cure - the role of histone deacetylase inhibitors (HDACi)

Contact person:

Dr Hao Lu

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Research group: Lewin/Cameron Laboratory

Overview:

Current combination antiretroviral therapy (cART) cannot cure HIV is due to the persistence of HIV in CD4+ T-cells in a latent form, where the virus is integrated into the host genome, but there is limited virus transcription or protein expression.

One strategy to eliminate latently infected cells would be to "drive" a cell out of latency by promoting transcription from the "silent" integrated latent virus. Although this might sound counter-intuitive i.e., promoting viral replication in an infected person, this treatment would be given while a patient is receiving cART which would prevent further rounds of replication.

The overall goal is therefore that the latently infected cell would start to produce virus and die, by either virus induced cell-death or recognition by the immune system. Ultimately this could lead to eradication of latently infected cells and a potential cure for HIV.

Recently the histone deacetylases inhibitor (HDACi), vorinostat, was shown to activate transcription of HIV in CD4+ T-cells in two separate clinical trials in HIV-infected patients on cART.

The main aim of this project will be to develop a library of stably transfected cell lines that mimic HIV latent infection. We will then determine the effect of treatment with HDACi alone or in combination with other cell activation agents on the activity of HIV transcription. We hope to find a combination of drugs that have a synergistic effect on HIV activation that could be used in future clinical trials.

PROJECT: The effects of disulfiram *in vivo* on latency

Contact person:

Prof Sharon Lewin

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Research group: Lewin/Cameron Laboratory

Overview:

The biggest hurdle in curing HIV infection in an individual is that the virus remains dormant in some populations of CD4+ T cells, hiding from the immune system and the cocktail of antiviral drugs used to treat HIV+ positive patients, or highly active antiretroviral therapy (HAART). These cells act as a viral reservoir of HIV and are a barrier to HIV eradication in the host.

While the process of establishing latency is complex, control of histone acetylation by cellular histone deacetylases (HDAC) has been shown to be critical in maintaining latency of HIV.

However, a new class of histone deacetylase inhibitors (HDACi) recently approved for the treatment of various lymphomas has also been demonstrated to reverse HIV latency by "waking up" HIV transcription and protein production in latently infected cells.

This project investigates whether HDACi will promote HIV reactivation in latently infected primary T-cells and whether mutation of the regulatory region of HIV confers resistance to HDACi activity.

In collaboration with investigators from the Alfred (Julian Elliott) and the University of California, San Francisco (UCSF, Steve Deeks) we are also investigating the effects of activating latency using the anti-alcohol drug disulfiram.

PROJECT: The effects of HDACi on HIV-infected cells in the brain

Contact person:

Prof Sharon Lewin

Phone: +61 3 9076 8491

Email: Sharon.lewin@monash.edu

Research group: Lewin/Cameron Laboratory

Overview:

HIV can remain dormant in some populations of cells, hiding from the immune system and the cocktail of antiviral drugs used to treat HIV+ positive patients.

This project investigates whether histone deacetylase inhibitors (HDACi) will promote HIV reactivation in astrocytes, a major reservoir for HIV in the central nervous system.

PROJECT: The effects of chemokines on the establishment of latency

Contact person:

Prof Sharon Lewin

Phone: +61 3 9076 8491

Email: Sharon.lewin@monash.edu

Research group: Lewin/Cameron Laboratory

Overview:

HIV can hide from current anti-HIV drugs in resting CD4+ T cells by establishing latent infection. Infection of resting CD4+ T-cells is difficult to establish in the laboratory. However, in patients, resting T-cells are clearly infected.

We have shown that latency can be established in resting CD4+ T cells following incubation with a family of proteins called chemokines. Chemokine receptor ligation allowed for enhanced efficiency of viral nuclear localization and integration in resting CD4+ T-cells.

We are now looking at which signalling pathways are involved in both nuclear localisation and integration. We are also seeking to identify ways to block or inhibit chemokine mediated latency using either chemokine receptor antagonists or chemokine antagonists.

PROJECT: Imaging early events of HIV entry into resting CD4+ T-cells

Contact person:

Prof Sharon Lewin

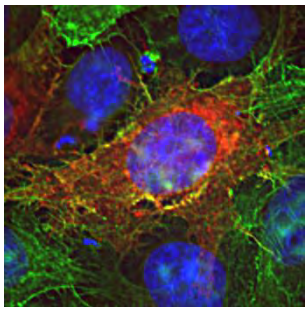
Phone: +61 3 9076 8491

Email: Sharon.lewin@monash.edu

Research group: Lewin/Cameron Laboratory

Overview:

HIV can hide from current anti-HIV drugs in resting CD4+ T cells by establishing latent infection. Although it is difficult to establish infection of resting CD4+ T-cells in the laboratory, we have shown that latency can be established in these cells following incubation with a family of proteins called chemokines. Chemokine receptor ligation allowed for enhanced efficiency of viral nuclear localization and integration in resting CD4+ T-cells.



This project will utilise state-of-the-art imaging facilities to look at early events during HIV entry into resting CD4+ T cells.

PROJECT: Dendritic cell control of latency in central memory and memory stem T cells.

Contact person:

Dr Paul U Cameron

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Research group: Lewin/Cameron Laboratory

Overview:

HIV targeting and latency in long lived T cells is critical in determining outcome and limiting strategies to cure HIV infection. The development of different T cell subsets and antigen specific activation depends on interactions between dendritic cells and T cells. The overall aim of this project is to study in human dendritic cell-T cell models the important role played by different sub-populations of dendritic cells in controlling HIV latency in T cells.

PROJECT: Cytoskeletal control of viral nuclear entry and integration in resting and migrating CD4 T cells.

Contact person:

Dr Paul U Cameron

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Email: paul.u.cameron@monash.edu

Research group: Lewin/Cameron Laboratory

Overview:

HIV targeting and latency in long lived T cells is critical in determining outcome and limiting strategies to cure HIV infection. The development of different T cell subsets and antigen specific activation depends on interactions between dendritic cells and T cells. The overall aim of this project is to study in human dendritic cell-T cell models the important role played by different sub-populations of dendritic cells in controlling HIV latency in T cells.

PROJECT: Role of innate immunity in HIV pathogenesis

Contact person: **Dr J. Judy Chang**

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Research group: Lewin/Cameron Laboratory

Overview:

During chronic HIV-1 infection, the inflammatory responses normally associated with an acute viral infection persist. The resulting chronic immune activation has been shown to be a strong predictor of HIV-1 disease progression.

HIV-1 can directly induce production of IFNα by plasmacytoid dendritic cells (pDCs) through the recognition of HIV-1 ssRNA via the TLR7 receptor. Stimulation of TLR7 by HIV-1 is associated with increased immune activation, and unlike synthetic TLR7 ligands, HIV-1 has been shown to persistently stimulate IFNα production by pDCs, and may therefore contribute to chronic immune activation.

The aim of this project is to characterize the role toll-like receptors (TLRs) and other innate immune receptors in sensing and responding to HIV-1 during different phases of disease and identify possible venues of manipulation for better disease control.

PROJECT: Sex-based difference in human innate immunity

Contact person: **Dr J. Judy Chang**

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Research group: Lewin/Cameron Laboratory

Overview:

Differences in the clinical manifestations of HIV-1 infection between the males and females have been described previously, including a lower average viral replication set-point but faster disease progression and higher levels of immune in females as compared to males

The precise mechanisms underlying these sex differences in the manifestations of HIV-1 disease are not understood, but previous in vitro studies have shown that plasmacytoid dendritic cells (pDCs) from females produced significantly more IFNα in response to HIV-1 and HIV-1-derived TLR7 ligands than pDCs from males.

The aim of this project is to elucidate the differences in innate immune responses between males and females and its implications on contributing to control or pathogenesis in viral infections.

PROJECT: Intersection of autophagy and TLR signaling in HIV

Contact person: **Dr J. Judy Chang**

Phone: +61 (03) 8506 2499

Email: ju-hui.chang@monash.edu

Research group: Lewin/Cameron Laboratory

Overview:
HIV-1 can directly induce production of IFNα by plasmacytoid dendritic cells (pDCs) through the recognition of HIV-1 ssRNA via the TLR7 receptor. Stimulation of TLR7 by HIV-1 is associated with increased immune activation, and unlike synthetic TLR7 ligands, HIV-1 has been shown to persistently stimulate IFNα production by pDCs, and may therefore contribute to chronic immune activation.

The aim of this project is to investigate how autophagy is utilized in both the TLR signaling pathway as well as viral protein degradation which are both important in the shaping the adaptive immune response.

Research Teams:

HIV Complications

Prof Jennifer Hoy
Director of HIV Medicine
Professor of Medicine Department of Infectious Diseases, Monash University
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Email: Jennifer.Hoy@monash.edu or J.Hoy@alfred.org.au

Alfred Staff web site:
http://www.alfredhealth.org.au/id/staff#3

Overview:

Professor Hoy is the Director of the Victorian HIV Service, Infectious Diseases Unit, The Alfred Hospital/Monash University. Her research interests include:

- HIV and its complications including cardiovascular disease and bone disease,
- Effects of HIV and antiretroviral treatment on bone disease, and co-chairs the Bone Mineral Density sub-study of the International START study.

PROJECT: HIV and Bone Disease

Contact person:

Prof Jennifer Hoy

Phone: +61 3 9076 6900

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Overview:

HIV-infected persons have lower bone mineral density (BMD) and more fractures than the general population of similar age. Both HIV-infection and antiretroviral treatment (ART) for HIV contribute to BMD loss, along with traditional risk factors; the relative contributions of HIV and ART are unknown.

Current collaborative studies underway include:

The effect of HIV alone and introduction of ART is being evaluated in 400 patients in the START study (START BMD substudy). Recruitment is complete worldwide, and follow-up continues.

It is known that all ART drugs can increase BMD loss in HIV patients, but tenofovir appears to have a significantly greater effect. In patients with low BMD, it is unknown whether switching the tenofovir for another ART drug or treatment with a bisphosphonate is the more effective and safe option. A randomised trial is underway to determine this.

PROJECT: HIV and Cardiovascular Diseases

Contact person:

Prof Jennifer Hoy

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Overview:

Cardiovascular disease has now become one of the major causes of morbidity and mortality in people living with HIV, who have a 2-fold increased risk when compared with the general population.

This elevated risk is likely due to a complex interplay between the increased incidence of traditional cardiac risk factors, the effects of ARV medications and the pro-inflammatory actions of HIV itself.

A number of projects are being undertaken to better understand the contributions of lipid abnormalities, chronic inflammation and platelet function in the observed increased risk of atherosclerosis and clinical CVD events.

Research Teams:

HIV Clinical Research, Health Services and Evidence Synthesis

Dr Julian Elliott

Head of Clinical Research at the Alfred Hospital and Monash Department of Infectious Diseases

HIV Clinical Advisor, Centre for Population Health, Burnet Institute.

Contact number: +61 3 9076 6077

Email: julian.elliott@alfred.org.au

Dr Julian Elliott is Head of Clinical Research at the Alfred Hospital Infectious Diseases Unit, Senior Lecturer in the Department of Infectious Diseases, Monash University and HIV Clinical Advisor, Burnet Institute.

Dr Elliott's research interests include:

- Understanding and preventing the high burden of chronic illness in people with HIV
- Evidence synthesis for decision making in healthcare
- Antiretroviral therapy in low- and middle-income countries, including treatment monitoring, treatment failure and immune restoration disease

PROJECT: HealthMap - a cluster randomised trial of interactive self-care plans to prevent and manage chronic conditions by people living with HIV.

Contact person:

Dr Julian Elliott

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Research group: Clinical Research

Overview:

Australians living with the human immunodeficiency virus (HIV) are exposed to an increased risk of several chronic conditions normally associated with ageing, including cardiovascular disease. The overall goal of this project to improve the health status of people living with HIV in Australia.

The aim of the project is to develop a self-management tool that can be accessed via smartphone, tablet or desktop that will help patients manage their medications, health goals and connect them with health care providers.

The impact of the HealthMap self-management tool will be rigorously evaluated in a cluster-randomized trial at 24 clinics throughout Australia over 2014/2015.

An opportunity exists for a student to join the HealthMap team to evaluate the outcomes of the trial, including effects on cardiovascular risk, sociobehavioural outcomes and quality of care.

PROJECT: Health knowledge technology

Contact person:

Dr Julian Elliott

Phone: +61 3 9076 6077

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Research group: Clinical Research

Overview:

Biomedical research is growing exponentially, and existing systems are inadequate for making sense of this large volume of unstructured biomedical research.

In collaboration with the Australasian Cochrane Centre and the Cochrane Collaboration, we are developing a new approach to healthcare decision-making called “Living Systematic Review”.

This involves the establishment of networks of contributors that jointly maintain high quality online systematic reviews that are updated whenever new research becomes available.

Opportunities exist within our group to pursue BMedSc, Honours and PhD projects that investigate how large linked datasets can be utilized by academic networks to co-curate high quality evidence services.

Projects include:

- Creating an online network for systematic review task exchange.
- Machine learning and semantic tagging for triaging reports of controlled trials.
- Optimising participation and quality for mass participation in systematic review.

Please contact Julian Elliott for more information.

Research Teams:

Wright Group

Assoc/Prof Edwina Wright

Head, Wright Group

Phone: +61 3 9076 6078

Email: e.wright@alfred.org.au

Overview

Assoc/Prod Wright's main research focus includes neurological disorders and opportunistic infections of the central nervous system (CNS) in HIV infected individuals.

The main objectives are to:

- To determine the impact of different antiretroviral treatment strategies upon HIV-associated neurocognitive performance
- To determine the prevalence of HIV-related neurological disorders, nationally and internationally
- To assess, diagnose and treat HIV related neurological complications.

See also: http://www.burnet.edu.au/staff_members/108_edwina_wright

PROJECT: The Victorian HIV Pre-Exposure Prophylaxis Demonstration Project

Contact person:

Assoc/Prof Edwina Wright

Phone: +61 3 9076 6078

Email: e.wright@alfred.org.au

Overview :

New HIV prevention strategies are necessary in Australia because HIV infection rates continue to rise. Victoria has the highest rate of HIV diagnoses across all Australian jurisdictions and saw a rise in the rate of HIV diagnosis in its population from 4.1 to 5.7 per 100,000 between 2003-2011. HIV pre-exposure prophylaxis (PrEP) when given as daily oral antiretroviral therapy and coupled with traditional HIV prevention measures, is efficacious in preventing HIV infection. Several studies have shown that daily tenofovir, or daily tenofovir plus emtricitabine reduce HIV transmission by ≥ 44% in Men who have Sex with Men (MSM) and by > 70% in heterosexuals at risk of HIV acquisition.There is now a critical need to translate the results of these PrEP efficacy studies into the 'real world' as noted in recent editorials in The Lancet and The New England Journal of Medicine. We are proposing to undertake a PrEP demonstration project in VIctoria to show that PrEP can be implemented feasibly, safely and effectively in the Victorian context through an accessible program targeted at the populations at highest risk of HIV acquisition. In 2014 we plan to enrol 200 people at risk of HIV infection who wish to commence PrEP and 200 people at risk who do not wish to commence PrEP and follow them for 12 months.

Key Research aims: To determine (1) the uptake and pattern of use of PrEP by those at highest risk of HIV, (2) the adherence to study medication and identify factors that facilitate or impede adherence, (3) the feasibility of and the factors that are key to providing a successful Victorian PrEP program, (4) the acceptability including side effects and behavioural change associated with PrEP use and (5) the risk of HIV infection in populations using PrEP.

This is an exciting project that will involve clinical, social and epidemiological research in the area of HIV prevention.

PROJECT TITLE: Do ACE inhibitors and Angiotensin II Receptor Antagonists increase CD4+ cell counts in virologically suppressed HIV+ patients with hypertension?

Contact person:

Assoc/Prof Edwina Wright

Phone: +61 3 9076 6078

Email: e.wright@alfred.org.au

PROJECT DESCRIPTION: Background: Hypertension is associated with increased risk for cardiac and cerebrovascular disease and occurs in up to 20% of HIV+ patients. Furthermore in virologically suppressed HIV+ patients, hypertension is associated with poorer neurocognitive function. The Australian National Heart Foundation Guidelines recommends that hypertension should be treated with ACE inhibitors or Angiotensin II Receptor Antagonists or calcium channel blockers or low dose thiazide diuretics.

Interestingly, ACE inhibitors and Angiotensin II Receptor Antagonists (AIIRA) have been shown to reduce fibrosis in pancreatic and hepatic tissue in animal models and reduce myocardial fibrosis in humans. Angiotensin –converting enzyme (ACE) is present in lymph nodes and other tissues. ACE converts Angiotensin I (AT1) to Angiotensin II (AT2). AT2 is pro-inflammatory and increases levels of TGF-β1, which is associated with fibrosis. Hence ACE inhibitors and Angiotensin II receptor antagonists-- through reduction of AT2 levels-- may reduce tissue fibrosis. During HIV infection, lymph node fibrosis destroys the architecture of lymph nodes and hence reduces the capacity for antigen presentation and development and proliferation of naive CD4 cells.

A recent feasibility study of the use of lisinopril (an ACE inhibitor) in normotensive, virologically suppressed HIV+ patients showed that the lisinopril was well tolerated, afforded a small but significant fall in diastolic blood pressure and was associated with a reduction in inflammatory cytokines.

Hence, given that hypertension is prevalent in HIV+ populations and that ACE inhibitors and Angiotensin II Receptor antagonists are used to treat hypertension, it is reasonable and plausible to explore the hypothesis that these medications may reduce lymphoid tissue

fibrosis and may be associated with an increase in CD4+ cells and other measures of immune function (CD4:CD8 ratios), which are routinely performed at clinical visits.

Study plan: We propose to undertake a review of treated, virologically suppressed HIV+ patients who have commenced anti-hypertensive treatment in the past 5 years to determine treatment with ACE inhibitors/AIIRA versus other anti-hypertensive agents is associated with a significant increase in CD4+ cells counts and other measures of immune function.

This is would be a novel and exciting study to undertake to determine whether or not some antihypertensive therapies that are widely used in HIV+ populations may confer improved immunity.

Research Teams:

Infectious in the immune-compromised

Dr Orla Morissey

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Email: O.Morrissey@alfred.org.au

Dr. Orla Morrissey's research has focused on optimising the prevention, diagnosis and treatment of invasive fungal infections. She has recently completed a randomised controlled trial examining the clinical utility of new diagnostic tests for invasive aspergillosis. This study demonstrated that Aspergillus galactomannan detection and PCR improve our ability to diagnose and treat invasive fungal infections. These findings have recently been accepted for publication in Lancet Infectious Diseases (IF: 17.4) and will significantly change the future management of invasive aspergillosis in the clinical setting.

PROJECT: Do Aspergillus fumigatus strains have different levels of virulence or ability to cause invasive infection?

Contact person:
Dr Orla Morissey

Phone: +61 3 9076 2631

Email: O.Morrissey@alfred.org.au

PROJECT DESCRIPTION:

Invasive or deep tissue infection with Aspergillus fumigatus is a major cause of death in the immunocompromised. Whilst there are considerable data available on the host factors that contribute to invasive aspergillosis (IA) there are very little data published on the virulence level of different strains of A. fumigatus.

Using internationally accepted and validated genotyping techniques we found in our laboratory that certain clinical strains of A. fumigatus appear to be associated with an increased risk of developing IA. An invertebrate larvae model (the greater wax moth or Galleria mellonella model) has used to detect virulence factors of A. fumigatus.1,2Preliminary wax moth larvae (WML) and reverse transcriptase (RT)-PCR experiments performed with collaborators at University of Manchester U.K. reported increased levels of total transcribed A. fumigatus RNA and expression of the toxin producing gene Asp F1 from clinically more virulent isolates. However, these data need to be confirmed in larger studies, examining a wider range of genes.

This project will use a large collection A. fumigatus isolates for genotyping, WML model and RT-PCR experiments to detect different strains, analyse their survival patterns and determine A. fumigatus RNA levels and the expression of specific genes. This work will be done in collaboration with Dr. Sarah Kidd, Head of Mycology Unit, SA Pathology in Adelaide; who has extensive experience in genotyping.

This research will allow us to identify virulence genes of A. fumigatus which will enable the detection of new drug targets. This is of enormous importance as there are very few new antifungal agents in development to combat this frequently fatal infection.

A wide range of skills will be used including fungal culture techniques, genotyping, invertebrate models and molecular biology.

- Reeves EP et al. Mycopathologia 2004;158:73.
- Jackson JC et al. PLoS One. 2009;4:e4224.

LINKS:

http://www.alfred.org.au/Department.aspx?ID=383

PROJECT: How should we manage Mycobacterium abscessus infection in lung transplant recipients?

Contact person:
Dr Orla Morissey

Phone: +61 3 9076 2631

Email: O.Morrissey@alfred.org.au

PROJECT DESCRIPTION:

Mycobacterium abscessus, whilst an uncommon organism complicating lung transplantation, is associated with significant morbidity and mortality. M. abscessus is resistant to many antimicrobial agents and has a high propensity to disseminate to the skin and brain. It appears to be on the increase.

A number of small studies have been performed looking at the incidence and outcomes and have attempted to provide guidelines for the management of M. abscessus infections. However, there were a number of problems with these studies including:

- Failure to examine the relative benefits of different drug regimens
- Inclusion of only the most severe cases
- Failure to examine the relationship between M. abscessus and the timing of other infections
- Lack of data on chronic allograft rejection
- The reporting of single centre experiences only

Given the current increase in the infection and the dearth of good quality data on which to base the management of M. abscessus infection; it is timely to perform an international survey of M. abscessus infection in lung transplant recipients, world-wide looking at outcomes, therapies and relationship to other infections and chronic allograft rejection and methods of transmission. A web-based cross-sectional survey will be developed and sent to lung transplant centres world-wide. Relevant clinical and microbiological data will be collected. The data will be analysed using Stata statistical package and used to develop improved guidelines for the management of M. abscessus infection and thus, has the potential to improve patient outcomes.

A wide range of clinical research and epidemiological skills will be used in this project. The project is ideal for a medical student or a B Med Sci (Hons) student.

LINKS:

http://www.med.monash.edu.au/cecs/infectious-diseases/

http://www.alfred.org.au/Department.aspx?ID=383

Research Teams:

Hospital Epidemiology

Assoc/Prof Anton Peleg

Head, Hospital Epidemiology

Phone: +61 3 99029159

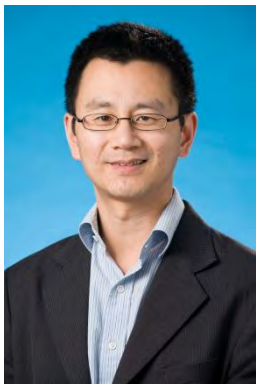
Overview:

Our research program focuses on the mechanisms of pathogenesis of important hospital-acquired pathogens. More specifically, ***Acinetobacter baumannii***, an emerging gram-negative bacterium, ***Staphylococcus aureus***, a gram-positive bacterium, and ***Candida albicans***, the most common human fungal pathogen. We combine bacterial and fungal genetic techniques with exciting *in vivo* infection model systems (mammalian and non-mammalian [*Caenorhabditis elegans* and Zebrafish]) to characterise the role of novel genes in virulence and antimicrobial resistance. Our over-arching goal is to identify new targets that may be amenable for future drug development, with a focus on microbial virulence, persistence and adaptation.

For information on project opportunities, please contact Assoc/Prof Peleg.

Research Teams:

Infection Prevention and Healthcare Epidemiology Unit



A/Prof Allen Cheng

Head, Infection Prevention Unit

Phone: +61 3 9076 2000

Email: allen.cheng@monash.edu

Overview:

The Infection Prevention and Healthcare Epidemiology Unit aims to prevent infections associated with health care in hospitals. Areas of particular interest include infections with resistant organisms, surgical site infection, hand hygiene and infections due to intravenous access devices. The unit also works closely with the Antimicrobial Stewardship Team, which aims to optimize antibiotic use to reduce antimicrobial resistance. Research in this unit aims to evaluate the effectiveness of initiatives in improving process outcomes (appropriate antibiotic use, hand hygiene, antibiotic prophylaxis, knowledge and attitudes) and patient outcomes (infection rates, infections with resistant organisms, mortality).

For information on project opportunities, please contact Assoc/Prof Cheng.