

# ASCIA

# 2011

**22nd Annual Scientific Meeting • Australasian Society of Clinical Immunology & Allergy (ASCIA)**

7-9 September 2011 • Sydney Convention & Exhibition Centre, Darling Harbour, NSW



Image courtesy of Tourism Australia

## Program & Abstract Book

**ASCIA 2011 is held in conjunction with:**

ASCIA 2011 Allergy & Immunology Update for Nurses, Dietitians and other health professionals • Tuesday 6 September 2011  
ASCIA 2011 Postgraduate Immunology Course • Saturday 10 September 2011

ASCIA is the peak professional body of clinical immunology and allergy specialists in Australia and New Zealand and is a member society of the Asia Pacific Association of Allergy, Asthma and Clinical Immunology and the World Allergy Organisation

**ascia**

australasian society of clinical immunology and allergy inc.

[www.allergy.org.au](http://www.allergy.org.au)

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# Welcome

# ASCIA2011

On behalf of the Australasian Society of Clinical Immunology and Allergy (ASCIA) it is a pleasure to welcome you to the 22nd ASCIA Annual Scientific Meeting, ASCIA 2011.

The ASCIA 2011 committee, chaired by Professor Connie Katelaris, has organised an exceptionally interesting scientific program, with an impressive range of speakers and topics in the areas of allergy and clinical immunology.

I look forward to seeing you at ASCIA 2011.

A/Professor Jo Douglass  
ASCIA President

On behalf of the Australasian Society of Clinical Immunology and Allergy (ASCIA) it is a pleasure to welcome you to ASCIA 2011.

The ASCIA 2011 Annual Scientific Meeting will run for 3 days, from Wednesday 7th to Friday 9th September and will be held in conjunction with:

- ASCIA 2011 Allergy & Immunology Update for health professionals on Tuesday 6th September
- ASCIA 2011 Postgraduate Immunology Course on Saturday 10th September.

We are honoured to have the following international keynote speakers presenting at ASCIA 2011:

- Professor Abul Abbas (USA)
- Professor Pascal Demoly (FRANCE)
- Dr George Du Toit (UK)
- Dr Montserrat Fernández-Rivas (SPAIN)
- Dr Steven Holland (USA)
- Professor Yehuda Shoenfeld (ISRAEL).

ASCIA 2011 also features more than 40 other speakers and chairs contributing to the meeting and a record number of poster and clinical grand rounds abstracts (84 in total) for an ASCIA Annual Scientific Meeting.

We are extremely grateful to our:

- Speakers and Chairs for taking time out of their busy schedules to contribute to this meeting
- Sponsors and Exhibitors for their generous support, which enables ASCIA to offer a high quality program whilst keeping the registration fees at a reasonable rate.

We trust you will enjoy the scientific program, social functions, the spectacular harbourside location and all the attractions that Sydney and its magnificent harbour has to offer.

Professor Connie Katelaris  
ASCIA 2011 Chair

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# Program at a Glance

## Secretariat



### Registration and Abstract submission

GPO Box 3270 Sydney  
NSW 2001  
Phone: 02 9254 5000  
Fax: 02 9251 3552  
Email: registration@ascia2011.com  
Website: www.ascia2011.com

in conjunction with:



## Sponsorship, Exhibition and Program Inquiries

ASCIA Executive Officer  
PO Box 450 Balgowlah NSW 2093  
Ph: 0425 216 402  
Fax: 02 9907 9773  
Email: education@allergy.org.au

## Organising Committee

Professor Connie Katelaris (Chair)  
Professor Brad Frankum  
Dr Preeti Joshi  
Dr Karuna Keat  
Dr Frederick Lee  
Dr Brynn Wainstein  
Ms Pamela Burton (ASCIA 2011 Allergy & Immunology Update)

Tues 6 Sept	Wed 7 Sept	Thurs 8 Sept	Fri 9 Sept	Sat 10 Sept
Allergy & Immunology Update	ASCIA ASM Day 1	ASCIA ASM Day 2	ASCIA ASM Day 3	Post Graduate Immunology Course
Registration	Registration Breakfast session: JSAC forum for trainees & supervisors	Breakfast symposium: Asthma therapy – what's new?	Breakfast symposium: HAE management	Introduction to the immune system
School based anaphylaxis education symposium	Climate change and allergy plenary	Autoimmunity plenary	Immunodeficiency plenary	Innate immunity, antigen presentation
Morning tea	Morning tea	Morning tea	Morning tea	Morning Tea
Immunotherapy symposium	Food allergy plenary	Immunotherapy symposium	GIT immune disorders symposium	T cell activation and co-stimulation  Effector T cell subsets, cytokines
Lunch	Lunch	Lunch	Lunch	Lunch
Concurrent sessions	Poster session	Immunity symposium	Poster session	B cells, antibodies and humoral immunity
Anaphylaxis training for primary care	Basten Oration		ASCIA AGM	Immune regulation and tolerance
Food allergy training for dietitians				
Afternoon Tea	Afternoon Tea	Afternoon Tea	Afternoon Tea	Afternoon Tea
Skin test workshop	Clinical grand rounds – oral presentations	Food allergy & anaphylaxis symposium	Drug allergy plenary	Autoimmunity and immune-mediated inflammatory disease
Food allergy training for dietitians (cont'd)				
Subcommittee meetings				
	Welcome Function	Gala Dinner	Closing Function	

## ATTENDANCE CERTIFICATES

Certificates of attendance will be provided to all delegates.

## ATTRACTIONS

Darling Harbour is a lively waterfront precinct and is one of Sydney's largest dining, shopping and entertainment areas. Attractions in Darling Harbour include Sydney Wildlife World, Sydney Aquarium, Chinese Garden of Friendship, Australian National Maritime Museum, Powerhouse Museum, Harbourside Shopping Centre and the many harbourside cafes and restaurants in Cockle Bay and King Street Wharf. From Darling Harbour you can take a ferry, yacht or speedboat for a tour of Sydney Harbour or to visit other areas of Sydney. For more information on attraction and tours visit [www.sydney.com](http://www.sydney.com)

## AWARDS AND TRAVEL SCHOLARSHIPS

Several prizes will be awarded, including the best Clinical Grand Rounds Prize (\$500), Paul Clarke Poster Prize (Allergy) \$500, National Asthma Council Poster Prize (\$500), Abacus ALS poster awards (\$500 each) and CSL PID Awards (\$500 each). Ten ASCIA 2011 Travel Scholarships of \$500 each, supported by MSD have been awarded to advanced trainees prior to the meeting.

## BREAKFAST SESSIONS

Breakfast sessions are included in the ASCIA 2011 ASM registration fee. For catering purposes delegates need to RSVP at least 48 hours prior to the session.

## CLIMATE

The average temperature for Sydney in September ranges from 11°C (minimum) to 20°C (maximum).

## DISCLAIMERS

In the event of industrial disruption the meeting organisers cannot be held responsible for any losses incurred by delegates. The program is correct at the time of printing; however the organisers reserve the right to alter the program, if and as is deemed necessary.

## DRESS CODE

The dress code is smart casual throughout the meeting.

## EXHIBITION

We encourage delegates to visit the ASCIA 2011 exhibition stands throughout the meeting.

## INTERNET CAFE

The ASCIA 2011 Internet Cafe is supported by VIROPHARMA and is located in the ASCIA 2011 exhibition area.

## PARKING

There are two car parks adjacent to the Sydney Convention & Exhibition Centre at Darling Harbour:

- Exhibition Carpark (enter via Darling Drive Pyrmont) – rate for 4 to 24 hours is \$32.
- Harbourside Carpark (enter via Murray St Pyrmont, beneath the Novotel Hotel) – Monday to Friday earlybird rate is \$14 (to obtain this rate you must enter between 06.00 and 09.30, exit between 15.00 and 19.00, prepay on level 3 in the morning and park on levels 1 or 2). On Saturday the rate for parking from 3 to 24 hours is \$28.

## SMOKE FREE POLICY

It is the policy of ASCIA 2011 that the meeting area of the venue and all related social functions are smoke-free.

## SOCIAL PROGRAM

### ASCIA 2011 Welcome Function

Wednesday 7th September, 17.30 to 19.30

Sydney Convention & Exhibition Centre – Parkside Foyer

### ASCIA 2011 Gala Dinner

Thursday 8th September, 18.30 to 23.00

Australian National Maritime Museum, Darling Harbour

Guest speaker presentation: Medicine and the Media - Dr John D'Arcy

Pre-dinner drinks and tours will be held from 18.30-19.30.

### ASCIA 2011 Closing Function

Friday 9th September, 17.30 to 18.30

Sydney Convention & Exhibition Centre – Parkside Foyer

## TRAVEL

- Sydney Airport is located approximately 30 minutes (by car, bus or train) from the Sydney Convention and Exhibition Centre at Darling Harbour, with regular flights from all capital cities.
- Transportation options to and from Sydney airport include taxi, train (to town hall station) or airport shuttle bus. If you are travelling from the airport during peak hour traffic additional time needs to be allowed and the train to and from the airport may be more efficient in this time period.
- If you catch a train from the airport, stop at Town Hall Station, then take the northern exit to Druiitt Street and it is a short walk downhill to Darling Harbour.
- If you catch a Darling Harbour/Balmain ferry service to Darling Harbour, alight at Pyrmont Bay Wharf.

# International Keynote Speakers



## Professor Abul Abbas MBBS

Professor & Chair, Department of Pathology, University of California, San Francisco (UCSF), USA

Professor Abbas completed his medical qualifications in New Delhi, India, before moving to the USA in 1970. As well as having an extensive commitment to training and fellowship programs, Professor Abbas:

- has received over 20 awards/honours
- has been a representative on over 40 committees
- has authored or co-authored more than 175 publications and 4 medical textbooks
- is the co-editor of Annual Reviews of Pathology
- is the current Vice President of the Federation of Clinical Immunology Societies (FOCIS) and will be President from July 2011

Professor Abbas's research is focused on immunological tolerance and autoimmunity. His laboratory is exploring the mechanisms that maintain tolerance to tissue and systemic self-antigens, and conditions that lead to the breakdown of self-tolerance and the development of autoimmunity. His laboratory is also analyzing the roles of different cytokines and T-cell subsets in autoimmune disease, and in the function of regulatory T-cells.



## Professor Pascal Demoly MD PhD

Chest Physician and Allergologist, University Hospital of Montpellier, FRANCE

Professor Demoly qualified from the Montpellier School of Medicine in 1992 and trained in allergy and chest medicine at the same university, where he has been Professor of Pulmonology since 2002. Head of the clinical department of Allergology and responsible for the allergy teaching programme, he is now chairman of the department of Pulmonology at the University of Montpellier, France.

In addition to his clinical and teaching commitments Professor Demoly is:

- actively involved in basic science related to the understanding of drug allergy and respiratory allergies
- ExCom member of the European Academy of Allergy and Clinical Immunology (EAACI)

- President of the French Society of Allergology
- a member of several medical societies and reviewer/evaluator for European grants and World Health Organization initiatives
- co-author of many publications and chapters of allergy, asthma and immunology
- Associate editor of *Allergy*, the *European Journal of Allergy and Clinical Immunology*
- Editorial board member of the *Journal of Investigational Allergology and Clinical Immunology*

*Professor Demoly's travel is supported by an educational grant from Stallergenes*



## Dr George Du Toit MBBCh FCP DCH MMed

Consultant Paediatric Allergist, Guy's and St Thomas' NHS Foundation Trust, King's College London UK

Dr Du Toit trained as a Paediatrician and then further specialised in paediatric allergy and asthma at the Red Cross Children's Hospital and University of Cape Town Lung Institute, in South Africa. In 2003, he took on the post of Consultant Paediatric Allergist at Imperial College London, St Mary's Hospital and in 2006 he joined the Children's Allergy Service at the Evelina Hospital.

Dr Du Toit's research interests include:

- prevention of peanut allergy - he is a co-investigator on the National Institute of Health (NIH) funded research study entitled "LEAP" (Learning Early about Peanut Allergies).
- food-dependent exercise-induced anaphylaxis
- drug allergy
- chronic urticaria

Dr Du Toit is a Fellow of the American Academy of Allergy & Immunology and the Royal College of Paediatrics & Child Health, a member of British Society of Allergy & Clinical Immunology (BSACI) and convener of the BSACI Paediatric Allergy Sub-Group.

*Dr Du Toit's travel is supported by an educational grant from Nestle Nutrition*



**Dr Montserrat Fernández-Rivas MD PhD**

Allergy Service, Hospital Clinico San Carlos, Madrid, SPAIN

Dr Fernández-Rivas is an allergy and clinical immunology specialist who is also involved in clinical research. She has been a consultant Allergist at the Hospital Clinico San Carlos since 2005 and was previously at the Hospital Virgen del Valle in Toledo from 1991 to 1994, Hospital Ntra.Sra. de Sonsoles in Avila from 1995 to 1999 and Fundacion Hospital Alcornon from 1999 to 2005.

Dr Fernández-Rivas is an author or co-author of more than 100 publications, is a board member of the Spanish Society of Allergy and Clinical Immunology (SEAIC) from 2006 to 2010, coordinator of the experts' committee on food allergy of the SEAIC from 2003-2010, EAACI member, has been an evaluator of proposals for the HEALTH topic of the European Commission and is a reviewer for several allergy and immunology publications.

Dr Fernández-Rivas's main research interests, funded by the European Commission and the Spanish Ministry of Science are:

- diagnosis, epidemiology and natural history of food allergy
- food and pollen allergens with a special focus on lipid transfer proteins and profilins
- Immunotherapy of pollen and food allergies

She has also participated as a principle investigator in several clinical trials of pollen and food allergens.

*Dr Fernández-Rivas's travel is supported by ALK-Abello*



**Dr Steven Holland MD**

Chief, Laboratory of Clinical Infectious Diseases (LCID) and LCID Immunopathogenesis Section, National Institute of Allergy and Infectious Diseases (NIAID), National Institute of Health (NIH), Bethesda, Maryland, USA

Dr Holland received his MD from the Johns Hopkins University School of Medicine in 1983 where he stayed as a resident in internal medicine, assistant chief of service in medicine, and fellow in infectious diseases. He moved to the National Institute of Health (NIH) in 1989 as a National Research Council fellow in the Laboratory of Molecular Microbiology, working on transcriptional regulation of HIV. In 1991, Dr Holland joined the

Laboratory of Host Defenses, shifting his research to the host side, with a focus on phagocyte defects and their associated infections. In 2004, he became chief of the LCID.

Dr Holland's major areas of research include:

- Immune defects of phagocytes: chronic granulomatous disease, hyper IgE (Job's) syndrome, leukocyte adhesion deficiency
- Cytokines in the pathogenesis and therapy of infections
- Susceptibility to disseminated mycobacterial infections, such as autoantibodies to interferon gamma and defects in the interferon gamma/IL-12 pathway
- Mechanisms of mycobacterial pathogenesis
- Mechanisms of bacterial pathogenesis (e.g., Burkholderia)
- Mechanisms of airway dysfunction

*Dr Holland's travel is supported by an educational grant from CSL Biotherapies*



**Professor Yehuda Shoenfeld MD FRCP**

Professor, Tel-Aviv University and Head, Department of Medicine 'B' and Center for Autoimmune Diseases, Sheba Medical Center, ISRAEL

Professor Shoenfeld is a physician and autoimmunity researcher and the current incumbent of the Laura Schwarz-Kipp Chair for Research of Autoimmune Diseases in the Sackler Faculty of Medicine at Tel-Aviv University. He completed his medical training at Hadassa Medical School, Hebrew University, Jerusalem in 1972 and his postgraduate studies in internal medicine at Tel Aviv University in 1978. Professor Shoenfeld's clinical and scientific work since the 1970's has largely focused on autoimmune disease.

As well as having an extensive commitment to training and fellowship programs, Professor Shoenfeld has:

- received more than 50 awards/grants
- been involved in organizing approximately 40 scientific meetings
- belonged to 24 professional associations
- been on more than 50 editorial boards
- had 9 patents
- authored or co-authored more than 1,600 publications, mostly in the area of autoimmune disease

# Other Speakers

## A/Professor Katie Allen

Paediatric Gastroenterologist and Allergist, Murdoch Children's Research Institute, Royal Children's Hospital, Melbourne, VIC, Australia

## Dr Bob Anderson

Laboratory Head, Autoimmunity and Transplantation Division, the Walter and Eliza Hall Institute, Victoria, Australia  
Chief Scientist and Chief Medical Officer, ImmusanT Inc.  
Cambridge, MA, USA

## Dr Domingo Barber

Senior Director of Research, ALK-Abello, Spain  
*Dr Domingo's travel is supported by ALK-ABELLO*

## Dr Paul Beggs

Deputy Head of Department and Senior Lecturer, Department of Environment and Geography, Faculty of Science, Macquarie University, NSW, Australia  
President, International Society of Biometeorology

## Pamela Burton

Clinical Research Coordinator  
Department of Medicine - Immunology and Allergy, Campbelltown Hospital, NSW, Australia

## Dr John D'Arcy

Medical practitioner and media spokesperson and writer on medical issues, based in Sydney NSW, Australia

## Geraldine Dunne

Clinical Nurse Consultant and Anaphylaxis Education Manager  
NSW Anaphylaxis Training Program  
Children's Hospital at Westmead, NSW, Australia

## Professor Paul Gatenby AM

Professor of Immunology and Director of Research, the Canberra Hospital, ACT, Australia

## Professor Gunnar Johansson

Professor of Clinical Immunology, Karolinska Institute, Sweden  
*Professor Johansson's travel is supported by the Australasian College of Anaesthetists (ACA)*

## Dr Preeti Joshi

Paediatric Clinical Immunologist/Allergist, Children's Hospital at Westmead, NSW, Australia

## A/Professor Bob Heddle

Clinical Immunologist/Allergist, Royal Adelaide Hospital and Chief Pathologist, South Australia

## Professor Connie Katelaris

Head of Unit and Professor of Immunology and Allergy, University of Western Sydney and Campbelltown Hospital, NSW, Australia  
President, Asia Pacific Association of Allergy, Asthma and Clinical Immunology (APAAACI)

## Hon Craig Knowles

President, Asthma Foundation NSW, St Leonards, NSW, Australia

## A/Professor Richard Loh

Paediatric Clinical Immunologist/Allergist, Princess Margaret Hospital for Children, Perth, WA, Australia

## Vicki McWilliam

Dietitian, Royal Children's Hospital, Melbourne, VIC, Australia

## Dr Raymond Mullins

Clinical Immunologist/Allergist, Canberra, ACT, Australia

## Merryn Netting

Dietitian, Adelaide, SA, Australia

## Dr Mark Riedl

Assistant Professor of Medicine and Section Chief, Clinical Immunology and Allergy at UCLA, California, USA  
*Dr Riedl's travel is supported by VIROPHARMA*



## Ingrid Roche

Dietitian, Princess Margaret Hospital for Children, Perth, Western Australia

## Dr Michael Rose

Specialist Anaesthetist, Royal North Shore Hospital, Sydney, NSW, Australia

Chairman of the Australian and New Zealand Anaesthetic Allergy Group

## Dr William Smith

Clinical Immunologist/Allergist, Royal Adelaide Hospital, South Australia

## A/Professor Frank Thien

Respiratory Physician and Allergist, Box Hill Hospital and Monash University, Victoria, Australia.

Secretary-General, Asia Pacific Association of Allergy, Asthma and Clinical Immunology (APAAACI)

## Sandra Vale

ASCIA Education Project Officer

## Professor Peter Van Asperen

Head, Department of Respiratory Medicine, Children's Hospital at Westmead, NSW, Australia

Macintosh Professor of Paediatric Respiratory Medicine, University of Sydney

## Dr Brynn Wainstein

Paediatric Clinical Immunologist/Allergist, Sydney Children's Hospital, Randwick, NSW, Australia

## Dr Melanie Wong

Paediatric Clinical Immunologist/Allergist, Children's Hospital at Westmead, NSW, Australia

## Chairs

Dr Stephen Adelstein (NSW)

Prof Tony Basten AO (NSW)

Pamela Burton (NSW)

Prof Dianne Campbell (NSW)

A/Prof Matthew Cook (ACT)

A/Prof Jo Douglass (VIC)

Rachael Dunn (WA)

Geraldine Dunne (NSW)

Dr David Elliott (NSW)

Dr Penny Fitzharris (NZ)

Prof Brad Frankum (NSW)

A/Prof Bob Heddle (SA)

Dr Preeti Joshi (NSW)

A/Prof Alyson Kakakios (NSW)

Prof Connie Katelaris (NSW)

Dr Karuna Keat (NSW)

Prof Ann Kupa (SA)

Dr Frederick Lee (NSW)

Dr Rob Loblay (NSW)

A/Prof Richard Loh (WA)

Prof Dominic Mallon (WA)

Dr Raymond Mullins (ACT)

Dr Richard Nolan (WA)

Prof Robyn O'Hehir (VIC)

Dr Jane Peake (QLD)

Dr Katrina Randall (ACT)

Dr Jan Sinclair (NZ)

Dr William Smith (SA)

Deryn Thompson (SA)

A/Prof Ron Walls AM (NSW)

Dr Melanie Wong (NSW)

A/Prof John Ziegler AM (NSW)

# Day 1 Program

## wednesday 7 september 2011

07.30-09.00	Registration	
07.00-08.30	<b>Breakfast Session</b> <b>JSAC Forum for Advanced Trainees and Supervisors</b> Chairs: A/Prof Matthew Cook, Dr Melanie Wong A light breakfast will be served from 07.00-07.30 and is supported by ASCIA <i>Please note that breakfast cannot be taken into the auditorium</i>	
09.00-10.30	<b>Plenary – Climate Change and Allergy</b> Chairs: Prof Connie Katelaris, A/Prof Jo Douglass	
09.00-09.10	<b>Introduction</b>	Hon Craig Knowles
09.10-09.50	<b>Climate change, aeroallergens and allergic disease</b>	Prof Connie Katelaris
09.50-10.30	<b>The science of climate change and aeroallergens</b>	Dr Paul Beggs
10.30-11.00	<b>Morning Tea – Exhibition Area</b>	
11.00-12.30	<b>Food Allergy Plenary</b> Chairs: A/Prof Alyson Kakakios, Dr Raymond Mullins	
11.00-11.45	<b>Dietary prevention of food allergy - an update</b>	Dr George DuToit
11.45-12.30	<b>Allergies to plant based foods (fruits and vegetables)</b>	Dr Montserrat Fernández-Rivas
12.30-14.00	<b>Lunch – Exhibition Area</b>	
14.00-14.45	<b>Poster Session – Exhibition Area</b> Chairs: Dr Rob Loblay, Prof Ann Kupa	
14.45-15.30	<b>Basten Oration</b> <b>Nature and nurture: the pathogenesis of systemic vasculitis</b> Chair: A/Prof Ron Walls AM	Prof Paul Gatenby AM
15.30-16.00	<b>Afternoon Tea – Exhibition Area</b>	
16.00-17.30	<b>Clinical Grand Rounds – oral presentations</b> Chairs: Prof Brad Frankum, Dr Preeti Joshi	
17.30-19.30	<b>ASCIA 2011 Welcome Function</b> Sydney Convention & Exhibition Centre – Parkside Foyer	

**Note:** ASCIA 2011 presentations will be held in the Sydney Convention & Exhibition Centre Parkside Auditorium, adjacent to the ASCIA 2011 exhibition area and ASCIA 2011 registration desk.

# Day 2 Program

thursday 8 september 2011

# ASCIA2011

07.00-08.50	<b>Breakfast Symposium</b> <b>Asthma Therapy – What’s New?</b> Chairs: Chairs: A/Prof Jo Douglass, Prof Brad Frankum	
07.30-08.10	<b>Difficult asthma (a review) and when to consider Xolair® in asthma?</b>	A/Prof Frank Thien
08.10-08.50	<b>Modern concepts of management of childhood asthma</b> Breakfast will be served from 07.00-07.30 and is supported by NOVARTIS <i>Please note that breakfast cannot be taken into the auditorium</i>	Prof Peter Van Asperen
09.00-10.30	<b>Autoimmunity Plenary</b> Chairs: Dr Karuna Keat, Dr Katrina Randall	
09.00-09.45	<b>Tolerance and autoimmunity: basic science and clinical implications</b>	Prof Abul Abbas
09.45-10.30	<b>ASIA - a new syndrome of autoimmune syndromes induced by adjuvants (vaccination, silicone, gulf war etc)</b>	Prof Yehuda Shoenfeld
10.30-11.00	<b>Morning Tea – Exhibition Area</b>	
11.00-12.30	<b>Immunotherapy Symposium</b> Chairs: A/Prof Bob Heddle, Prof Robyn O’Hehir	
11.00-11.45	<b>Long term effects of grass pollen sublingual immunotherapy in adults and children</b>	Prof Pascal Demoly
11.45-12.30	<b>New insights in sublingual immunotherapy</b>	Dr Montserrat Fernández-Rivas
12.30-14.00	<b>Lunch – Exhibition Area</b>	
14.00-15.30	<b>Immunity Symposium</b> Chairs: Prof Tony Basten AO, Dr Frederick Lee	
14.00-14.45	<b>Infection and autoimmunity</b>	Prof Yehuda Shoenfeld
14.45-15.30	<b>Cytokines for and against infection: disorders of innate immunity</b>	Dr Steven Holland
15.30-16.00	<b>Afternoon Tea – Exhibition Area</b>	
16.00-17.30	<b>Food Allergy &amp; Anaphylaxis Symposium</b> Chairs: A/Prof Richard Loh, A/Prof John Ziegler AM	
16.00-16.40	<b>Pathophysiology of exercise-induced anaphylaxis syndrome</b>	Dr George Du Toit
16.40-17.10	<b>Food allergen challenges</b>	Dr Brynn Wainstein
17.10-17.30	<b>New in vitro diagnostic tests for food allergy</b>	Dr Domingo Barber
18.30-23.00	<b>ASCIA 2011 Gala Dinner</b> Australian National Maritime Centre, Darling Harbour Guest speaker presentation: Medicine and the Media - Dr John D’Arcy <i>Pre-dinner drinks and tours will be held from 18.30-19.30</i>	

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# Day 3 Program

## friday 9 september 2011

07.00-08.50	<b>Breakfast Symposium</b> <b>Hereditary Angioedema (HAE) Management</b> Chairs: Dr Penny Fitzharris, Dr William Smith	
07.30-08.10	<b>HAE Management – a global perspective</b>	Dr Marc Riedl
08.10-08.50	<b>HAE Management – an Australasian perspective</b> Breakfast will be served from 07.00-07.30 and is supported by VIROPHARMA <i>Please note that breakfast cannot be taken into the auditorium</i>	Prof Connie Katelaris
09.00-10.30	<b>Immunodeficiency Plenary</b> Chairs: Dr Jane Peake, Dr Stephen Adelstein	
09.00-09.50	<b>Update on new immunodeficiency syndromes - clinical and laboratory correlations</b>	Dr Steven Holland
09.50-10.30	<b>Veno-occlusive disease and immunodeficiency (VODI)</b>	Dr Melanie Wong
10.30-11.00	<b>Morning Tea – Exhibition Area</b>	
11.00-12.30	<b>GIT Immune Disorders</b> Chairs: Prof Dianne Campbell, Prof Dominic Mallon	
11.00-11.45	<b>Coeliac Disease update</b>	Dr Bob Anderson
11.45-12.30	<b>Eosinophilic Eosophagitis and other eosinophilic Disorders of the GIT</b>	A/Prof Katie Allen
12.30-13.30	<b>Lunch – Exhibition Area</b>	
13.30-14.15	<b>Poster Session – Exhibition Area</b> Chairs: Dr Jan Sinclair, Dr Richard Nolan	
14.15-15.00	<b>ASCIA Annual General Meeting</b> Chair: A/Prof Jo Douglass	
15.00-15.30	<b>Afternoon Tea – Exhibition Area</b>	
15.30-17.30	<b>Drug Allergy Plenary</b> (in conjunction with the Australian Society of Anaesthetists (ASA)) Chairs: Prof Connie Katelaris, Dr David Elliott	
15.30-16.00	<b>Review of mechanisms of anaphylaxis</b>	Dr Michael Rose
16.00-16.40	<b>Epidemiology, risk factors and prevention of anaesthetic drug allergy</b>	Prof Pascal Demoly
16.40-17.10	<b>Anaphylaxis to neuromuscular blocking agents (NMBAs) - the role of environmental agents</b>	Prof Gunnar Johansson
17.10-17.30	<b>Allergic reactions in the theatre - anywhere, anytime</b>	Dr Michael Rose
17.30-18.30	<b>ASCIA 2011 Closing Function (including wine and cheese)</b> Sydney Convention & Exhibition Centre – Parkside Foyer	

**Note:** ASCIA 2011 presentations will be held in the Sydney Convention & Exhibition Centre Parkside Auditorium, adjacent to the ASCIA 2011 exhibition area and ASCIA 2011 registration desk.

# Postgraduate Immunology Course

saturday 10 september 2011

**Course Director:** Professor Abul Abbas

08.30-09.00 Introduction to the immune system

09.00-10.00 Innate immunity, antigen presentation

10.00-10.30 Morning Tea

10.30-11.30 T cell activation and co-stimulation

11.30-12.30 Effector T cell subsets, cytokines

12.30-13.30 Lunch

13.30-14.30 B cells, antibodies and humoral immunity

14.30-15.30 Immune regulation and tolerance

15.30-16.00 Afternoon Tea

16.00-17.00 Autoimmunity and immune-mediated inflammatory diseases

17.00-17.30 Open discussion

The course is based on the Federation on Immunological Societies (FOCIS) basic immunology courses directed by Professor Abul Abbas which have been extremely popular with FOCIS delegates.

The ASCIA 2011 Postgraduate Immunology Course presentations, exhibition area and registration desk will be located in the Parkside G04 room (one level down from the Parkside Auditorium, via the escalators).

# Allergy & Immunology Update

tuesday 6 september 2011

08.00-09.00	Registration	
09.00-09.10	Welcome and Introduction	Prof Connie Katelaris Pamela Burton
09.10-10.30	School Based Anaphylaxis Education Symposium Chairs: Pamela Burton, Geraldine Dunne	
09.10-09.45	Recent updates to the initial 2010 version of ASCIA anaphylaxis e-training	Sandra Vale
09.45-10.30	Challenges, barriers and where to from here?	Geraldine Dunne
10.30-11.00	Morning Tea – Exhibition Area	
11.00-11.45	Immunotherapy Symposium Chairs: Rachael Dunn, Deryn Thompson	
11.00-11.30	SLIT – rules for treatment	Dr Preeti Joshi
11.30-12.00	SCIT – precautions in practice	Pamela Burton
12.00-12.30	Venom IT – review of stinging insect allergy and its management	A/Prof Bob Heddele
12.30-13.30	Lunch – Exhibition Area	
13.30-16.30	Concurrent Sessions	
13.30-15.00	Food allergy training (for dietitians and other health professionals)	Dr Raymond Mullins Ingrid Roche
15.00-15.30	Afternoon Tea – Exhibition Area	
15.30-16.30	Food allergy training (continued)	
15.30-16.15	Case studies (3 x 15 minutes each)	Dr Raymond Mullins, Merryn Netting, Vicki McWilliam
16.15-16.30	Q & A	Dr Raymond Mullins, Merryn Netting, Ingrid Roche Vicki McWilliam
	Please note that the above afternoon sessions will be held concurrently with the following sessions:	
13.30-15.00	Anaphylaxis training for primary care health professionals (for medical practitioners and nurses)	A/Prof Richard Loh
15.00-15.30	Afternoon Tea – Exhibition Area	
15.30-16.30	Skin testing workshop	Dr William Smith
16.30-17.30	Concurrent meetings for ASCIA Nurses' and Dietitians' subcommittees	
17.30-18.30	Wine and Cheese - Sydney Convention & Exhibition Centre – Parkside Foyer	

**Note:**

ASCIA 2011 Allergy & Immunology Update presentations will be held in the Parkside G04 and G05 rooms, which are located downstairs from the Parkside Auditorium. The exhibition area and catering for the ASCIA 2011 Allergy & Immunology Update will be located in the area around the top of the escalators, adjacent to the Parkside Auditorium.

# Other Meetings

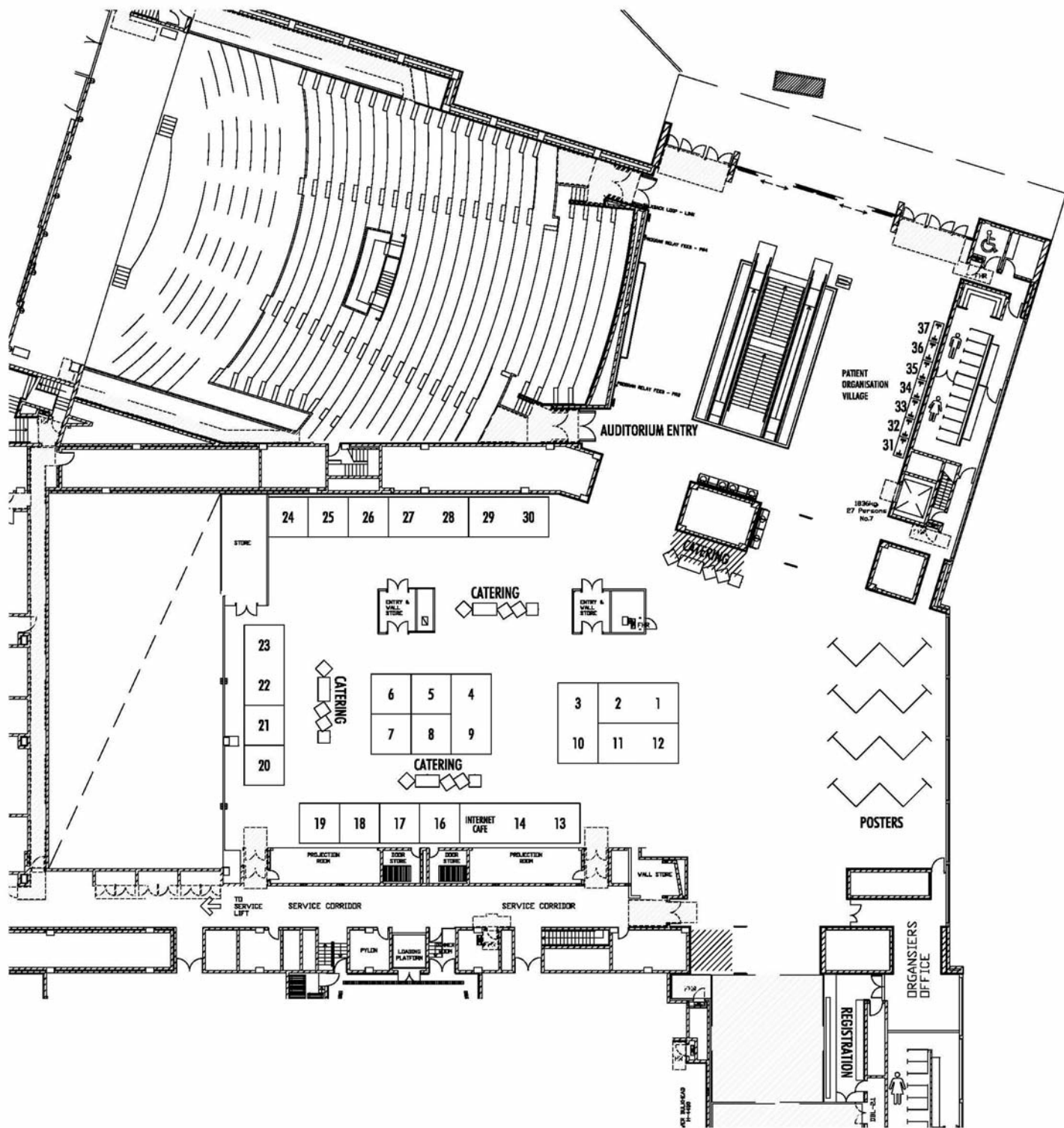
tuesday 6 september 2011

09.00-11.00	RACP PREP Advanced Training Supervisor Workshop Presenters: A/Prof Bob Heddle, Dr Frederick Lee	Parkside room G05
08.00-13.00	ASCIA Council Meeting	Parkside room G07
15.30-17.30	ASCIA Paediatric Committee	Parkside room G07
17.30-18.30	ASCIA Anaphylaxis Working Party	Parkside Foyer

# Sponsor & Exhibitor Directory

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# Sponsor & Exhibitor Profiles

## ABACUS-ALS

[www.abacus-als.com](http://www.abacus-als.com)

Abacus ALS represents Phadia, which provides world-leading diagnostic solutions for allergy, asthma and autoimmunity.

The latest developments in allergy include allergen components which improve patient management. Visit our stand to learn more about venom components and the ability to use these to resolve double positivity with CCD-free recombinant proteins (discriminating between ant and wasp venom sensitization). Other updates include peanut components and an update from recent material presented at the recent EAACI congress in June. The latest fully automated EliA autoimmune assays include EliA anti-IgA for assessing the risk of IgA deficient subjects for anaphalatic reactions upon blood transfusion as well as EliA PmScl aiding in clinical diagnosis of polymyositis / scleroderma overlap syndrome.

## ABBOTT NUTRITION

[www.abbottnutrition.com.au](http://www.abbottnutrition.com.au)

Abbott Nutrition, a division of Abbott Australasia is committed to advancing patient care by developing innovative solutions in nutritional products. Our leading-edge science is behind some of the world's most trusted names in medical nutrition, including EleCare®, which is a nutritionally complete amino acid based formula with 1/3 of fat as Medium Chain Triglycerides (MCTs), to aid fat absorption. This product has been specially formulated for infants and children who have multiple food allergies, including cow's milk protein allergy. In addition, EleCare Unflavoured and EleCare Vanilla are also indicated for the treatment of Eosinophilic Oesophagitis. EleCare® is PBS listed and is available in three variants, EleCare Unflavoured, EleCare Unflavoured with LCP and EleCare Vanilla.

## ALLERGEND

[www.allergend.com.au](http://www.allergend.com.au)

AllergEnd Plus is the Ultimate dust mite protective bedding. It is a breakthrough in dust mite protection being 100% Oeko-Tex Standard 100 certified chemical free cotton, super breathable and luxurious to sleep on. As well as offering these features, our zippered pillow, mattress and quilt encasings act as a complete barrier to dust mites and their associated allergens. Made in Melbourne, our AllergEnd Plus bedding range has established us as market leaders. With over 50 years of experience in specialist healthcare product manufacturing we pride ourselves on the quality of our products. AllergEnd Plus can be purchased online at [www.allergend.com.au](http://www.allergend.com.au), over the phone on Free Call 1800 656 484 or in person direct from our factory at 132a Cotham Road, Kew 3101.

## ALLERGY NEW ZEALAND

[www.allergy.org.nz](http://www.allergy.org.nz)

Allergy New Zealand Inc is the national patient support organisation in New Zealand. With the support of a Medical Panel (all members of ASCIA) we work to raise awareness and provide evidence-based information and education on all allergies, and support through a national volunteer network to families with children with food allergy. Our quarterly magazine, Allergy Today, is distributed to over 5,000 health professionals. We also represent the interests of the allergic population through submissions and participation in working groups; and are involved in research into the burden of food allergy in New Zealand. See our website for more information.

## ALPHAPHARM

[www.alphapharm.com.au](http://www.alphapharm.com.au)

Alphapharm is Australia's leading supplier of prescription medicines to the Government-subsidised Pharmaceutical Benefits Scheme (PBS). One in five prescriptions for PBS medicines is dispensed with an Alphapharm product. Our speciality is bringing generic medicines to market, which contributes to the sustainability of the PBS by providing affordable access to pharmaceuticals. Alphapharm medicines are made to the highest global quality standards and have the same effect on the body as initial brands.

Alphapharm pioneered generic medicines in Australia in 1982, setting up as a small pharmaceutical manufacturer in Queensland with 12 staff and four products. Today, we have 550 employees nationally, including 400 at our state-of-the-art manufacturing plant at Carole Park, Queensland. This year, the plant will produce 3.5 billion doses of which about 1.7 billion will be exported to some 50 countries around the world.

Alphapharm is part of US-based Mylan.

## ANAPHYLAXIS AUSTRALIA INC

[www.allergyfacts.org.au](http://www.allergyfacts.org.au)

Anaphylaxis Australia Inc (AAI) is a national not for profit organisation that was established in 1993 to assist and support those affected by allergy and the risk of anaphylaxis. AAI is an evidence based support organisation that assists individuals at risk of anaphylaxis and their families, school and childcare staff, health professionals, those in the food industry and all in the community needing to understand how to avoid an allergic reaction and what to do in an anaphylaxis emergency. AAI's aim is to enable individuals to manage everyday life whilst minimising risk to their health and wellbeing. The organisation advocates for people living with allergy when communicating with government, policy makers, food industry and the media. AAI develops resources and shares knowledge and expertise with the community at large whilst supporting research initiatives throughout

Australia. AAI is supported by a Medical Advisory Board consisting of ASCIA members. Visit [www.allergyfacts.org.au](http://www.allergyfacts.org.au) or call 1300 728 000 for more information.

## ASTHMA AUSTRALIA

[www.asthmaaustralia.org.au](http://www.asthmaaustralia.org.au)

Asthma Australia is the recognised national community voice of Australians with asthma and linked conditions and their carers. It comprises the Asthma Foundations from each Australian state and territory working together on national policy, advocacy and programs and promoting research. It is a national, non-government, incorporated body with no political affiliations. We provide asthma information, education, training and advocacy in the community and promote research. We do this in collaboration with a wide range of community, clinical, government and corporate partners. Information about our work can be obtained by calling local Foundations (1800 645 130) or via our website.

## AUSTRALIAN SUPPORT NETWORK FOR EOSINOPHILIC OESOPHAGITIS AND RELATED DISORDERS (AUSEE INC)

[www.ausee.org](http://www.ausee.org)

The Australian Support Network for Eosinophilic oEsophagitis and related disorders (ausEE inc) is a registered Australian charity and non-profit organisation dedicated to providing support and information to anyone diagnosed with or caring for someone with an Eosinophilic Gastrointestinal Disorder (EGID) including Eosinophilic oEsophagitis (EE or EoE). ausEE's mission is to improve the lives of those affected by providing support and information not only to those with an EGID, but to the greater medical community. ausEE Inc. currently campaigns and raises funds for further research in Australia. ausEE's website has valuable information and resources including a forum for members. For more information, please visit [www.ausee.org](http://www.ausee.org)

## AUSTRALASIAN MEDICAL & SCIENTIFIC (AMS) / ALK-ABELLO

[www.amsl.com.au](http://www.amsl.com.au)

AMS Allergy is proud to be the local Australian distributor for the world's leading and largest Immunotherapy Vaccine manufacturer; ALK-Abello of Denmark, Spain and USA. The ALK product range encompasses SPT Reagents, Lancet systems, Immunotherapy Vaccines of various types, Diluents, Sterile Empty Vials, and other Accessories. Of particular note is the ALK-Abello Sub-Lingual Drop Immunotherapy Vaccine supplied under Named Patient Basis. Also offered (through TGA channels) are various Injection Vaccines and the Tablet Immunotherapy Vaccine for European Grasses. Other products we offer are for the in vivo detection of Penicillin & Amoxycillin sensitivity, "TRUE Test" and "Allergeaze" Patch Tests for the identification of allergens causing Contact

Dermatitis, as well as the unique and very interesting Phototherapy system "RhinoLight" for the treatment of Allergic Rhinitis. To support Allergists and other medical practitioners with the use of these products, we at AMS pride ourselves on our unparalleled experience and expertise in the field of allergy diagnosis and immunotherapy, as well as our good contact with Allergists, our reliable and excellent service. See [www.amsl.com.au](http://www.amsl.com.au) for further information on AMS Allergy, or call 02-9882 3666.

## CARE PHARMACEUTICALS

[www.carepharmaceuticals.com.au](http://www.carepharmaceuticals.com.au)

Care Pharmaceuticals (formerly Paedpharm Pty Ltd) was established in Australia in 1986 to provide innovative over the counter products for all members of the family. Their trusted brands including the range of FESS® Nasal sprays that are synonymous with quality, safety and efficacy. Whether it's mild to moderate nasal congestion or severe congestion due to sinusitis, rhinitis and allergies, there's a FESS® product to help provide relief and help maintain nasal health. All FESS® products are non-medicated so they can be used as often as needed even if pregnant, breastfeeding or on medication. For further information and details on our new products please visit [www.carepharmaceuticals.com.au](http://www.carepharmaceuticals.com.au) or see us at Stand 5.

## CSL BIOTHERAPIES

[www.csl.com.au](http://www.csl.com.au)

CSL has been the chosen national plasma fractionator of Australia since 1952, and New Zealand since 1962. Today CSL Biotherapies works with Australian Governments, the Australian Red Cross Blood Service and the New Zealand Blood Service to deliver a world class and broad range of plasma-derived therapies specifically designed to meet the needs of healthcare professionals and patients in both these countries. CSL Biotherapies, through its research and development program, is committed to ensuring continued world class standards of quality, product yields, patient convenience and manufacturing efficiency in our broad portfolio of plasma-derived therapies. An example of this commitment is the use of modern manufacturing technologies for our products, such as large scale chromatography and the inclusion of at least two dedicated and complementary pathogen reduction steps in the manufacturing process of each product.

## EGO PHARMACEUTICALS

[www.egopharm.com](http://www.egopharm.com)

Ego Pharmaceuticals is an Australian family owned and operated company who for more than 50 years have specialised in the manufacture of quality skincare products such as market leaders 'QV', 'Sunsense', 'Pinetarsol', 'DermAid' and 'Moov'. The QV range of gentle moisturisers and cleansers has been scientifically

# Sponsor & Exhibitor Profiles

formulated for those who suffer from dry or sensitive skin conditions. The entire QV range is free from colour, fragrance, propylene glycol, lanolin and its derivatives making it ideal for the most sensitive skin types.

## GLAXOSMITHKLINE (GSK)

[www.gsk.com.au](http://www.gsk.com.au)

GlaxoSmithKline (GSK) is a leader in pharmaceutical research and development with a combination of skills and resources, providing a platform for delivering innovation in today's rapidly changing healthcare environment. GSK is a patient focused organisation with a mission to improve the quality of human life by enabling people to do more, feel better and live longer. We are committed to delivering the best quality pharmaceuticals, vaccines and over the counter products to the people of Australia. In 2010 GSK invested \$56 million in Australian research and development, and continues to be ranked as one of Australia's top 15 business investors in this area.

## GRIFOLS AUSTRALIA

Grifols Australia is proud to present Flebogamma 5% DIF human normal immunoglobulin (IVIG). Flebogamma 5% DIF is indicated for replacement therapy in Primary Immunodeficiency Syndromes, Myeloma or Chronic Lymphocytic Leukaemia, Immunomodulation, Allogenic Bone Marrow Transplantation and children with congenital AIDS. Flebogamma 5% DIF is liquid stable at room temperature for 2 years and is sorbitol stabilised.

Flebogamma 5% DIF is available in Australia in 4 ranges from 2.5g (50mL), 5g (100mL), 10g (200mL) and 20g (400mL). For more information on Grifols Australia, Clayton South, Victoria and our product range, please contact us on Freecall: 1800 339 479 or visit our website.

## HAEi

[www.haei.org](http://www.haei.org)

HAEi - International Patient Organization for C1-Inhibitor Deficiencies - is a global organization dedicated to raising awareness of C1 inhibitor deficiencies around the world. It is a non-profit international network of national HAE patient Associations. HAEi is established to promote co-operation, co-ordination and information sharing between HAE Specialists and National HAE Patient Associations in order to help facilitate the availability of effective diagnosis and management of C1 inhibitor deficiencies throughout the world.

## IMMUNE DEFICIENCIES FOUNDATION AUSTRALIA (IDFA)

[www.idfaustralia.org](http://www.idfaustralia.org)

The Immune Deficiencies Foundation Australia (IDFA) is a not for profit patient organisation which provides support services to

people with primary immune deficiencies (PID). Individual PID's are rare and many people who have these problems discover that their disorder is poorly understood by others. This can cause the patient and family to feel isolated and unable to access important information that can help them. IDFA helps keep the lines of communication open by matching people Australia wide with others who have the same or similar condition, by sharing information between members through newsletters, patient meetings, email and social media. The IDFA website is an excellent source of medical information for both the community and medical professionals.

## IMMUNE DEFICIENCIES FOUNDATION NEW ZEALAND (IDFNZ)

[www.idfnz.org.nz](http://www.idfnz.org.nz)

Immune Deficiencies Foundation of New Zealand (IDFNZ) is a non-profit organization, formed in 1987, which is dedicated to supporting children, teenagers and adults with diagnosed Primary Immune Deficiency (PID) disorders, ongoing medical and scientific research, lobbying and educating the public and health professionals about PID. IDFNZ is governed by an Executive Board including two leading medical professionals specialising in PID disorders, has medical and scientific subcommittees and is a member of the International Patient Organisation for Primary Immune Deficiencies (IPOP).

## iNOVA PHARMACEUTICALS

[www.inovapharma.com](http://www.inovapharma.com) [www.rinar.com.au](http://www.rinar.com.au)  
[www.zepallergyrelief.com.au](http://www.zepallergyrelief.com.au)

iNova Pharmaceuticals (Australia) Pty Limited is an Australian owned and operated pharmaceutical company, and is the headquarters for iNova operations across Australia, Asia-Pacific and Southern Africa. iNova has a diverse range of leading consumer healthcare and specialty prescription brands mainly in weight management, respiratory health, pain management, allergy, sexual health, dermatology and cardiology available in over 20 countries. Our allergy range includes: Rinar® Non-Allergic Rhinitis Nasal Spray, Azep® Nasal Spray - Hayfever Relief, Eyezep® Eye Drops - Allergic Conjunctivitis Relief, and ZepAllergy™ Hayfever and Allergy Tablets.

## IN VITRO TECHNOLOGIES

[www.invitro.com.au](http://www.invitro.com.au)

In Vitro Technologies is a privately owned, Australian company, specialising in distribution, sales, marketing and support of quality systems and products for the diagnostic, medical, life science and industrial segments of the market for Australia and New Zealand. The Diagnostics Division represents leading global companies with innovative niche products in biochemistry, immunology, haematology, molecular diagnostics, point of care and veterinary systems and test kits. To satisfy our discerning

customers the products are backed up by a combined sales, support and applications team across Australia and New Zealand with experience in the pathology and diagnostic industry to enable empathetic understanding of service to provide solutions to customer needs and constraints.

## LINK PHARMACEUTICALS

[www.linkgroup.com.au](http://www.linkgroup.com.au)

Link Pharmaceuticals has been committed to Australasian medicine for almost two decades. Link has extensive experience in providing specialist pharmaceuticals across a variety of therapeutic areas; their mission is not only to acquire but to ensure the supply of vital medicines. In recent years, Link alongside their partner, Stallergenes, has concentrated on the provision of allergy care covering all aspects, from diagnosis to the treatment of anaphylaxis. With the recent launch and PBS listing of Anapen®, Australia's new adrenaline auto-injector, Link has been implementing their education and training plans. The goal is to work alongside the profession and provide a smooth introduction of Anapen® to help those at risk of severe allergic reaction.

## MEDICALERT® FOUNDATION

[www.medicalert.org.au](http://www.medicalert.org.au)

Australia Medic Alert Foundation is a registered not-for-profit membership-based organisation that provides a trusted 24/7 personal medical emergency information system for people with medical conditions including allergies, special medications and/or advanced wishes. Members wear a bracelet or necklet with a MedicAlert emblem attached and engraved with their membership number, key medical information and 24/7 emergency hotline number for emergency services and healthcare personnel to access. For more information, please contact Membership Services on FREECALL 1800 88 22 22 or email [enquires@medicalert.org.au](mailto:enquires@medicalert.org.au)

## MSD

[www.msd-australia.com.au](http://www.msd-australia.com.au)

Today's MSD is a global healthcare leader working to help the world be well. MSD is a tradename of Merck & Co., Inc., with headquarters in Whitehouse Station, N.J., U.S.A. Through our prescription medicines, vaccines, biologic therapies, consumer care and animal health products, we work with customers and operate in more than 140 countries to deliver innovative health solutions. We also demonstrate our commitment to increasing access to healthcare through far-reaching policies, programs and partnerships. MSD. Be well. For more information, visit our website.

## MOLNLYCKE HEALTH CARE

[www.molnlycke.com.au](http://www.molnlycke.com.au)

Mölnlycke Health Care is a world leading manufacturer of single-use surgical and wound care solutions for the professional health care sector. Our Wound Care dressings offer gentle and effective wound healing and include a range of unique products based on a patented Safetac® soft silicone technology e.g. Mepilex® and Mepitel®, together with surgical, absorbent and fixation dressings, e.g. Mepore®, Mepore® IV and Mesorb®.

Mölnlycke Health Care also offers a range of products specifically designed to assist in the treatment of Eczema - from Epaderm™ Ointment (a complete emollient therapy) and Epaderm™ Cream to Tubifast™ Garments - a range of ready to wear garments for wet or dry wrapping. Mölnlycke Health Care, Inspiring Confidence. Phone: 1800 005 231

## NEILMED PHARMACEUTICALS

[www.neilmed.com](http://www.neilmed.com)

NeilMed Pharmaceuticals manufacture allergy & sinus relief products. NeilMed's Sinus Rinse Kit is a soothing saline nasal irrigation system that includes 60 packets of preservative free, isotonic, pH balanced mixture, irrigation bottle and custom designed cap. It has been proven to be highly effective for rhinosinusitis, allergies, and most beneficial for post FESS patients. Phone: 1800 158 900

## NESTLE NUTRITION

[www.nestlebaby.com.au](http://www.nestlebaby.com.au)

[www.nestlebaby.co.nz](http://www.nestlebaby.co.nz)

Nestlé Nutrition is the global leader in infant nutrition. Through integrating scientific and research expertise, Nestle Nutrition aims to launch the most innovative, cutting edge products and services to stay at the forefront of nutrition science and technology. As a long time partner of ASCIA please come and visit us during this year's meeting to find out more about our infant product range. Our product range includes Alfare & NAN HA Gold 1 - the only clinically proven HA infant formula for allergy prevention.

## NOVARTIS

[www.novartis.com.au](http://www.novartis.com.au)

Novartis is a world leader in the research, development and supply of medicines to prevent and cure disease, ease suffering and enhance quality of life. Headquartered in Basel, Switzerland, we operate in over 140 countries and employ approximately 98,000 people. Our medicines treat some of the most serious conditions confronting healthcare professionals and patients in the areas of Primary Care, Oncology, Immunology, Infectious Disease and Ophthalmics.

# Sponsor & Exhibitor Profiles

## NUTRICIA ADVANCED MEDICAL NUTRITION

[www.nutricia.com.au](http://www.nutricia.com.au)

As a recognised leader in specialised and infant nutrition, Nutricia has a mission to lead the use of Advanced Medical Nutrition in disease management. With a broad and unique portfolio of products, we seek to provide solutions and services wherever nutritional intervention can be shown to improve clinical outcomes. In partnership with healthcare professionals and caregivers, we work to make a difference by speeding recovery and encouraging independence. The specialised nutrition of today can help alleviate disease symptoms, or even delay the progression of disease in patients. Our ambition is to deliver only proven benefits through nutrition, as an integral part of disease treatment.

## SHIRE

[www.shireaustralia.com.au](http://www.shireaustralia.com.au)

As one of the world's leading specialty biopharmaceutical companies, Shire has emerged as a company fully focused on a single purpose: to enable people with life-altering conditions to lead better lives. Since opening its doors in May 2008, Shire Australia has registered products for use in rare genetic diseases such as Fabry Disease and Hereditary Angioedema and in specialty pharmaceuticals in the areas of Hyperphosphataemia in chronic Renal Disease, Essential Thrombocythaemia and mild to moderate active Ulcerative Colitis.

We are located at Level 3, 78 Waterloo Road, North Ryde, NSW 2113. Phone +61 2 8019 4400.

Customer Services: 1800 012 612 or email: [enquiriesaustralia@shire.com](mailto:enquiriesaustralia@shire.com)

## STALLERGENES

[www.stallergenes.com](http://www.stallergenes.com)

As a biopharmaceutical company, Stallergenes has been a leader and pioneer in allergen immunotherapy. Devoting more than 20% of its turnover to R&D, Stallergenes has significantly enhanced its commitment to developing innovative and evidence based treatment. The company has recently obtained marketing approval for Oralair, the grass pollen sublingual tablet. For almost 2 years, Stallergenes and Link Medical Products have combined their efforts to improve allergy care in Australia, strengthening their commitment to becoming a key partner to allergy specialists by providing them with diagnostics (Hollister-Stier), subcutaneous treatments (Alustal and Albey) and sublingual tablet (Oralair) together with sublingual drops as Named Patient Products.

## STAR ALLERGY ALERTS

[www.starallergyalerts.com.au](http://www.starallergyalerts.com.au)

Star Allergy Alerts provides a bright, fun range of food allergy and medical awareness products with designs that children love to wear, perfect for Child Care, Kindy, School, Camps, Parties, Picnics, Play-dates and Holidays! They offer a practical way to increase awareness to children's food allergies and medical conditions... to help protect them from well-meaning offerings that could put them in danger. Star Allergy Alerts hopes to make the lives of parents and carers a little easier, by providing a range of products to raise allergy awareness for the safety of those we love. Our products are meant to serve as a reminder to others when you are not around and should not replace the care of a qualified medical professional and open regular communication with your child and their carers. Our products are recommended by Allergy Specialists and General Practitioners, and are used in Schools, Kindergartens and Childcare Centres as an integral part of their Anaphylaxis Management Programs.

## VIOPHARMA

[www.viopharma.com](http://www.viopharma.com)

Please visit stand 13, 14, 15 to meet with the Medical Affairs staff of ViroPharma Incorporated to ask questions/hold discussions regarding Hereditary Angioedema (HAE).

- P1 Janet Anderson EVALUATION OF SPECIFIC IGE TESTING TO CHLORHEXIDINE IN CASES OF PERIOPERATIVE ANAPHYLAXIS
- P2 Bianca Angelica THE ASSOCIATION BETWEEN ONSET AND PERSISTENCE OF ECZEMA AND THE RISK OF OTHER ALLERGIC DISEASES
- P3 Sara Barnes MANAGEMENT OF ACUTE PULMONARY HAEMORRHAGE IN A PATIENT WITH ANCA POSITIVE VASCULITIS
- P4 Craig Barnett LOWER LIMB WEAKNESS AND ATAXIA IN A 59 YEAR OLD WOMAN POST PERTUSSIS VACCINATION WITH SEROLOGICAL FEATURES OF SLE
- P5 Melanie Batchelor CLINICAL LESSONS FROM AN ANAPHYLAXIS MANAGEMENT GUIDELINE AUDIT
- P6 Andrew Broadfoot WHEAT-DEPENDENT EXERCISE-INDUCED ANAPHYLAXIS: CLINICAL AND LABORATORY FINDINGS IN AUSTRALIAN SUBJECTS
- P7 Anna Brusch IGG BUT NOT IGM OR IGA ANTI-BETA 2 GLYCOPROTEIN ANTIBODY LEVELS ARE ASSOCIATED WITH HIGHEST RISK THROMBOTIC EVENTS AMONG PATIENTS AT A TERTIARY HOSPITAL
- P8 Anna Brusch SUCCESSFUL TREATMENT OF REFRACTORY ADULT ONSET STILL'S DISEASE WITH ANAKINRA
- P9 Christine Bundell ANTI-TISSUE TRANSGLUTAMINASE IGA TESTING - A COMPARISON OF FOUR AVAILABLE ASSAYS
- P10 Christine Bundell F-ACTIN ANTIBODY TESTING - WHICH METHOD TO IMPLEMENT?
- P11 Christine Bundell POST-MORTEM MAST CELL TRYPTASE MEASUREMENT AND HAEMOLYSIS LEVELS
- P12 Christine Bundell POST-MORTEM MAST CELL TRYPTASE IN NON-ANAPHYLACTIC DEATHS; DOES CAUSE OF DEATH HAVE A SIGNIFICANT EFFECT
- P13 Dianne Campbell ANAPHYLAXIS TO CITRUS FRUIT AND APPLE SEED
- P14 Dianne Campbell SAFETY OF FOOD CHALLENGES IN A TERTIARY REFERRAL CHILDREN'S HOSPITAL
- P15 Ahmed Daoud EXPERIMENTAL HETEROPLASIA: HISTOPATHOLOGICAL & IMMUNOLOGICAL STUDY
- P16 Janet Davies BUDESONIDE AND FORMOTEROL REDUCE EARLY INNATE ANTI-VIRAL IMMUNE RESPONSES IN VITRO
- P17 Janet Davies B CELL RESPONSES TO AEROALLERGENS IN PATIENT WITH ALLERGIC DISEASE
- P18 Georg Dewald A NEW TYPE OF HEREDITARY ANGIOEDEMA
- P19 Iman M Eftekharzadeh ALTERNARIA AND ALLERGIC DISEASES (ALLERGIC RHINITIS AND ASTHMA)
- P20 Marianne Empson EVOGAM, THE FIRST LOCALLY PRODUCED SCIG; A STUDY OF EFFICACY, SAFETY, AND ACCEPTABILITY IN PID
- P21 Lara Ford IDENTIFICATION OF BIOMARKERS TO DISTINGUISH VARIOUS LEVELS OF COWS MILK TOLERANCE
- P22 Rodney Ford WHICH CHILDREN WARRANT A GLUTEN-FREE DIET?
- P23 Lauren Frieze EVALUATION OF THE EUROIMMUNE EUROLINE SYSTEMIC SCLEROSIS (NUCLEOLI) PROFILE (IgG) KIT
- P24 Paul Gray CHILDHOOD ONSET AUTOSOMAL DOMINANT ORBITAL MYOSITIS IN AN AUSTRALIAN FAMILY
- P25 Anthony Griffiths A CASE OF RECURRENT FEVERS
- P26 Rory Hannah A REVIEW OF THE EXPANDED SPECIALIST TRAINING PROGRAM IN ITS FIRST YEAR OF IMPLEMENTATION IN ADELAIDE
- P27 Rory Hannah A CONTROLLED STUDY OF DELTA-INULIN ADJUVANT IN HONEY BEE VENOM IMMUNOTHERAPY
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## POSTER

### 1. EVALUATION OF SPECIFIC IGE TESTING TO CHLORHEXIDINE IN CASES OF PERIOPERATIVE ANAPHYLAXIS

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**Background:** A number of different agents have been implicated as causes of anaphylaxis during anaesthesia. Chlorhexidine is a widely used antiseptic found in creams, wound dressings, lubricant gels and surgical skin preparation solutions. As such it is a significant possible causal agent in cases of perioperative anaphylaxis.

**Aim:** To assess the usefulness of measuring specific IgE to chlorhexidine as part of a testing regimen in cases of anaesthetic related anaphylaxis.

**Method:** Thirty-three patients referred to the Anaesthesia Allergy and Adverse Events clinic at Royal North Shore Hospital were included in this study on the basis of clinical suspicion of allergy to chlorhexidine. Standardized skin tests were performed with all suspected agents to which patients had been exposed, including chlorhexidine. Testing for specific IgE to chlorhexidine in patient serum was performed via the Phadia ImmunoCAP system.

**Results:** Using the recommended cut-off value of 0.35 kUA/L, specific IgE to chlorhexidine showed a sensitivity of 86% and a specificity of 96% with reference to skin test results. Positive and negative predictive values were also 86% and 96%. An alternative positive cut-off value of 0.21 kUA/L was determined by ROC analysis resulting in a sensitivity of 100% and a specificity of 96%. Positive and negative predictive values were 88% and 100%.

**Conclusion:** Evaluation of specific IgE testing to chlorhexidine shows this to be a reliable adjunct to skin testing in determining diagnosis of allergy to chlorhexidine. Additionally, adjustment of the cut-off threshold for determination of positive results was able to increase test sensitivity without adversely affecting specificity in this patient group. Studies to date have all involved relatively small sample sizes. Further studies involving larger sample sizes would be helpful in elucidating accurate cut-off levels for determination of positive samples.

## POSTER

### 2. THE ASSOCIATION BETWEEN ONSET AND PERSISTENCE OF ECZEMA AND THE RISK OF OTHER ALLERGIC DISEASES

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**Background:** Few eczema studies have simultaneously addressed the importance of both age of onset and duration of eczema symptoms in early life for the subsequent persistence of eczema and development of asthma and allergic rhinitis.

**Objective:** To examine the importance of age of eczema onset combined with eczema duration as predictors for later childhood eczema, childhood asthma and allergic rhinitis.

**Methods:** A prospective birth cohort of 620 infants with a family history of allergic diseases was recruited. Telephone interviews were conducted 18 times in the first two years of life, and then annually from age 3 to 7 years to document any episodes of eczema. Current asthma and allergic rhinitis were assessed at age 6 and 7. Logistic regression models were fitted to adjust for potential confounders.

**Results:** Early-onset eczema, especially <6 months, was associated with greater risk of persistence at ages 6 and 7 (adjusted OR=11.06; 95%CI=5.91-20.69). Early-onset eczema which commenced <6 months and was persistent (still present between age 2 and 7), was also related to current asthma (aOR=3.85; 95%CI=1.90-7.79) and allergic rhinitis (aOR=2.22; 95%CI=1.09-4.53) at ages 6 and 7. There was no evidence that early-onset remitting eczema (only present < 2 years) or late-onset eczema (onset >2 years) were associated with current allergic diseases at ages 6 and 7.

**Conclusion:** Eczema which commences early in life and persists into toddler years is strongly associated with current asthma and allergic rhinitis at ages 6 and 7. However, remitting and late-onset eczema do not appear to be related to these outcomes. With effective early intervention, the risk of allergic diseases associated with early-onset eczema might be reduced.

## POSTER

### 3. MANAGEMENT OF ACUTE PULMONARY HAEMORRHAGE IN A PATIENT WITH ANCA POSITIVE VASCULITIS

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A 50 year lady with a long history of asthma presented with a two week history of worsening dyspnoea and cough with small volume haemoptysis on a background of chronic sinus congestion and joint pains. Her initial chest x-ray (CXR) and CT (computed tomography) scan showed right upper lobe consolidation with a repeat CT showing progression. Following admission to a rural hospital for intravenous antibiotics, she rapidly deteriorated necessitating intubation and retrieval to a tertiary hospital with venous-venous extra corpulmonary oxygenation (vv-ECMO). On arrival to a tertiary hospital on a public holiday, she was haemodynamically unstable requiring inotropic support and six unit blood transfusion. Bronchoscopy revealed haemoptysis. Urinary red cell morphology revealed glomerular red cells despite normal serum creatinine (65 umol/L) and urine output. Her renal function subsequently deteriorated and she was supported on continuous veno-veno haemodiafiltration via an ECMO circuit for a 36 hour period. A tentative diagnosis of vasculitis was made. Plasmapheresis was commenced with initiation of methyl prednisolone and cyclophosphamide. Due to the public holiday there were difficulties in obtaining immunological pathology results and co ordination of plasmapheresis and nerve conduction services.

In summary the use of v-v ECMO in acute respiratory failure with profound pulmonary haemorrhage requiring intensive multifaceted immunosuppression is rarely described particularly combined with plasmapheresis. V-v ECMO should be considered in patients with life threatening pulmonary haemorrhage due to vasculitis and plasmapheresis can be run concurrently whilst systemic immunosuppressants take effect. Finally access barriers to pathology services and technician dependant services are an issue for tertiary hospitals on public holidays and after hours.

## POSTER

### 4. LOWER LIMB WEAKNESS AND ATAXIA IN A 59 YEAR OLD WOMAN POST PERTUSSIS VACCINATION WITH SEROLOGICAL FEATURES OF SLE

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**Background:** Neurological symptoms inpatients with an

Autoimmune disease present a diagnostic challenge. This is particularly so when the choice of immunomodulatory treatments may be modified in light of specific autoantibody tests.

**Case report:** A 59 year old previously well health worker presented with lower limb weakness and ataxia 5 weeks after a booster immunisation with DTP vaccine. Further questioning revealed a longstanding photosensitive rash and a clinical history suggestive of Raynauds phenomenon. Eye examination was normal. Autoantibody screening revealed significant titres of ANA and DsDNA, and antibodies to aquaporin-4 (NMOIgG) were also present. MRI displayed changes consistent with a longitudinally extensive transverse myelitis extending from C2 to T10. The patients presentation raised an interesting differential diagnosis. She was treated with immunosuppressive therapy comprising prednisolone, azathioprine and hydroxychloroquine, with considerable clinical improvement.

**Conclusion:** This case highlights the complex nature of autoimmune diseases that fall under the neuro-immunology classification. The presence of specific antibodies can influence treatment of such disabling illnesses. The specificity of the aquaporin-4 antibodies, and their particular usefulness in this case supports the need for such assays to be performed in specialized centres.

## POSTER

### 5. CLINICAL LESSONS FROM AN ANAPHYLAXIS MANAGEMENT GUIDELINE AUDIT

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**Background:** Anaphylaxis Management Guidelines for Flinders Medical Centre (FMC) were endorsed in 2009 to improve acute clinical assessment, treatment and discharge management for patients presenting with Anaphylaxis. Allergy Specialists regularly promote the practical application of these guidelines to ED staff.

**Aim:** The purpose of the audit was to evaluate guideline adherence in clinical practice.

**Method:** The case notes of 30 adult patients who presented to ED with Grade2 or 3 anaphylaxis (Simon Brown criteria) in 2010 were audited retrospectively. Documentation of: adrenaline administration, monitoring in ED, adrenaline autoinjector prescription, ASCIA Anaphylaxis Action Plan completion, complementary education and Allergy Clinic referral, was audited.

**Results:** 22 of 30 patients received adrenaline as treatment for Anaphylaxis (14 in ED, 3 by self, 7 in other settings). Those 6 patients not given adrenaline had an unidentified trigger or a time delay before medical assessment. 3 patients were treated by the incorrect route.

2/30 patients met the guideline's monitoring criteria. 20 patients were observed for 4-6 hours after adrenaline injection, with no evidence of this observation occurring following symptom resolution.

21 patients were discharged with an adrenaline autoinjector. Documentation of Anaphylaxis Action Plan provision, autoinjector training or allergen avoidance education was not recorded for half of these patients. 5 patients received all components of this practice guideline.

18 patients were referred to Specialist Allergy services.

**Conclusion:** Evidence of compliance with the Anaphylaxis Management Guidelines for FMC is lacking. This audit could not identify cases where appropriate care was given but not documented. The core components of the guidelines (timely adrenaline administration, appropriate observation, patient education and specialist referral) are essential for patient safety. Guidelines need clarification to clearly state these expectations. Closer liaison with ED staff and innovative tools to promote adherence to the guidelines are suggested.

## POSTER

### 6. WHEAT-DEPENDENT EXERCISE-INDUCED ANAPHYLAXIS: CLINICAL AND LABORATORY FINDINGS IN AUSTRALIAN SUBJECTS

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**Introduction:** Food-dependent exercise-induced anaphylaxis (FDEIA) is a unique form of allergy in which ingestion of a certain food, followed by exercise several hours later, can precipitate anaphylaxis. Wheat is the most widely reported food associated with this condition. Wheat-dependent exercise-induced anaphylaxis (WDEIA) is mainly associated with gluten, particularly omega-5 gliadins and high-molecular-weight (HMWV) glutenin subunits. We reviewed patients with this syndrome and correlated their clinical and laboratory findings.

**Methods:** Patients with WDEIA were identified for review and the clinical details, results of their skin prick tests and serology specific IgE were correlated. Specific IgE was measured by Immucap (Phadia®).

**Results:** Seven patients, all with specific IgE to omega-5 gliadin were identified. Four of these individuals were male. The average age was 43 years (20-66). Four patients had suffered exercise-induced anaphylaxis (Mueller grade 4), the remainder had suffered urticaria after exercise, usually generalised. Length of time to diagnosis ranged between 3 months to 9 years. All but one of the patients had suffered urticaria following wheat ingestion, prior

to suffering an anaphylaxis. There was no correlation between the severity of the reaction and the levels of omega-5 gliadin specific IgE. Three of our seven patients were negative for wheat (as measured by Immucap) and positive to omega 5 gliadin alone (one patient tested negative to wheat-specific IgE in two different laboratories). 6/7 patients had positive wheat skin prick tests.

**Conclusions:** If WDEIA is suspected, then serology specific IgE to omega-5 gliadin should be performed on all patients, and not just for wheat alone as (43% (3/7) of our patients were negative for wheat and as generalised urticaria may occur prior to more severe allergic reactions to wheat, a positive omega-5 gliadin alone, in patients presenting with generalised urticaria after wheat, may prevent morbidity associated with delayed diagnosis.

## POSTER

### 7. IgG BUT NOT IgM OR IgA ANTI-BETA 2 GLYCOPROTEIN I ANTIBODY LEVELS ARE ASSOCIATED WITH HIGHEST RISK THROMBOTIC EVENTS AMONG PATIENTS AT A TERTIARY HOSPITAL

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The purpose of this study was to assess the significance of anti-beta 2 glycoprotein I (anti-B2GPI) isotypes among hospital patients by phenotyping patients with positive anti-B2GPI profiles.

**Methods:** A search of the WA tertiary hospital database for anti-B2GPI tests was performed. This identified 4647 patients who had undergone testing between August 2004 and August 2007 for whom a hospital number could be identified. 682 of these patients had International Classification of Diseases (ICD) data linked to our hospital site. Among this group, 46 patients had at least one positive anti-B2GPI isotype >99th percentile for our laboratory's reference control population. This cut-off was used as per the revised Sapporo guidelines relating to the laboratory criteria for the diagnosis of antiphospholipid syndrome (APS). Clinical records were reviewed and events categorized. Thrombotic events were further subdivided into provoked or unprovoked depending on the stated aetiology as a way of stratifying patients' risk of an underlying thrombophilic tendency such as APS.

**Results:** Of the 46 patients, 30 had a positive IgG anti-B2GPI result, 15 were positive for IgM and 23 for IgA. Unprovoked thrombotic events occurred with significantly higher frequency among patients with an anti-B2GPI profile containing IgG compared with IgM (20/30 vs 4/15, p=0.025). A positive IgA result was not seen in isolation in any patient with an unprovoked thrombosis. Of the 12 patients with a positive IgA result and

unprovoked thromboses, 11 also had a positive IgG result. The median IgG anti-B2GPI antibody level (but not IgM or IgA) was significantly higher for patients with unprovoked compared with provoked thromboses (100 SGU vs 8 SGU,  $p=0.004$ ).

**Conclusion:** These results show that the IgG anti-B2GPI isotype associates more strongly with unprovoked thromboses and is of greater value than IgM or IgA isotypes in the assessment of thrombotic events among hospital patients.

## POSTER

### 8. SUCCESSFUL TREATMENT OF REFRACTORY ADULT ONSET STILL'S DISEASE WITH ANAKINRA

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Adult onset Still's disease (AOSD) is a systemic inflammatory disorder of uncertain aetiology and pathogenesis. Approximately one third of cases develop chronic, persistent disease which often requires long-term treatment with glucocorticoids and immunosuppressants. We report the case of a man with refractory AOSD treated successfully with the IL-1 receptor antagonist, Anakinra.

The patient was diagnosed with AOSD in his early thirties after presenting with an acute febrile illness associated with pharyngitis, myalgias and skin rash. His disease followed a relapsing and remitting course over the next 20 years and required multiple courses of prednisolone and treatment with other immunosuppressants including methotrexate and leflunomide. He later developed continuous symptoms treated with increasing doses of prednisolone. His symptoms were associated with fevers and elevations in C-reactive protein and neutrophil count. Anakinra was initiated at a dose of 100mg per day by subcutaneous injection. This led to immediate resolution of his symptoms and normalization of his inflammatory markers and neutrophilia. His symptoms recurred transiently when the supply of Anakinra became temporarily unavailable and was mirrored by a rise in inflammatory markers. Otherwise he has remained symptom free for over six months. The patient experienced mild injection site reactions at the initiation of Anakinra therapy which resolved after the first few weeks of treatment. To date, no other side-effects have been encountered.

This case demonstrates that Anakinra is a well-tolerated, rapidly effective treatment for refractory AOSD and that IL-1 may be implicated in the pathogenesis and symptomatology of this rare, inflammatory condition.

## POSTER

### 9. ANTI TISSUE TRANSGLUTAMINASE IGA TESTING – A COMPARISON OF FOUR AVAILABLE ASSAYS

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**Background:** IgA tissue transglutaminase antibody (Ttg IgA) has become a first line test for coeliac disease (CD). In our laboratory, Ttg IgA requests have increased by more than 20%/year for the last 2 years.

**Aim:** To evaluate the Phadia 250 Celikey IgA automated FEIA as an alternative to Ttg IgA ELISA.

**Method:** Seventy samples were selected to represent high, medium and low values, established by Aesku or Genesis ELISA assays. Ten samples were selected because of a normal biopsy and a high Ttg IgA result by Aesku; and 10 because of an abnormal biopsy and a high Ttg IgA Aesku. These samples were tested on each of the Aesku and Genesis ELISA methods, the Phadia FEIA and the indirect immunofluorescence endomysial antibody assay (EMA).

**Results:** The Aesku ELISA was the least correlated (Pearson's) with the others, and the highest correlation was between Phadia and EMA.

Of the 10 samples with a normal biopsy and positive Ttg IgA Aesku, none was positive by any of the other assays. Of the 10 samples with an abnormal biopsy and positive Ttg IgA Aesku, 9 were positive or equivocal by each of the other methods.

**Discussion:** The high correlation between Phadia FEIA and the EMA results and between Phadia and the biopsy result support a change from a manual ELISA method to the automated Phadia FEIA method which allows random access and high sample throughput.

## POSTER

### 10. F-ACTIN ANTIBODY TESTING – WHICH METHOD TO IMPLEMENT?

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**Aim:** To evaluate the utility of various detection methods for antibodies against F-Actin.

**Background:** Anti smooth muscle antibodies are detected in patients with autoimmune hepatitis (>80%) however they are also detected in individuals with other liver diseases and in the normal population (~12%). Anti smooth muscle antibodies (SMA) directed against the filamentous actin (F-actin) are considered specific for AIH type 1. Various methods are available for diagnostic testing of F actin antibodies including: indirect immunofluorescence using rodent stomach and kidney tissue (LKS), F actin expressing vascular smooth muscle cells (VSM) and rat intestinal epithelial cells (RIE), and solid phase ELISA and immunoblot.

**Method:** Anti F actin antibody testing was performed on a total of eighty three serum samples comprising 42 consecutive samples testing positive for SMA (early 2010), 10 samples testing negative for SMA, 11 samples from known autoimmune hepatitis patients and 20 samples from the Australian Red Cross Blood Service and serums made available by the Royal College of Pathologists of Australia (used as normal controls). Samples were tested on each of the methods outlined above.

**Results:** The table compares F-actin testing for AIH disease patients compared to all other groups using measures of diagnostic test validity and predictive value.

**Conclusion:** IIF on rodent LKS tissue sections remains the first-level test for anti F-actin SMA detection as it is sensitive and the most specific available method. If a second test is required due to confounding factors, IIF on a VSM or RIE cell substrate provide a convenient and practical alternative approach.

Method	Detection Ig	Sensitivity	Specificity	PPV	NPV
VSM	IgG	76.47	84.61	56.52	93.22
RIE	IgG	75	81.25	57.14	90.69
IB	IgG	52.94	73.84	34.61	85.71
ELISA	IgG	100	61.81	44.73	100
LKS 1	IgGMA	88.23	89.23	68.18	96.66

## POSTER

### 11. POST MORTEM MAST CELL TRYPTASE MEASUREMENT AND HAEMOLYSIS LEVELS

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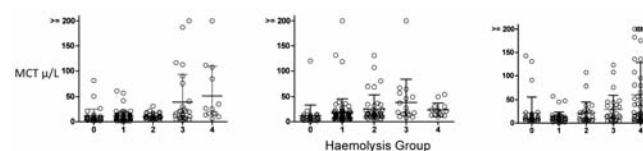
**Background:** Collection delay and method, and condition of the cadaver will affect the state of blood collected post mortem (PM). Phadia recommends PM samples should be collected within 48hrs of death. Published data suggests strong haemolysis may affect MCT levels.

**Aim:** The aim of this study was to investigate the effect of haemolysis on mast-cell tryptase (MCT) levels in samples from PM cases collected under the local condition.

**Method:** Post-mortem blood samples were collected with the approval of the Coronial Ethics committee from 187 cases received at the Western Australian State Mortuary. The delay between death and subclavian collection time was  $1.0 \pm 1.8$  days (mean  $\pm$  StDev). Blood was collected from the subclavian artery on arrival at the mortuary and from the aorta and femoral artery at necropsy. Separated serum was analysed for MCT (mg/L) using a Phadia 250. The haemolysis index (HI) was measured on an Architect (Abbott) and grouped into intervals (Group 0,  $0 < HI < 0.3$  AU; Group 1,  $0.3 < HI < 1.0$  AU; Group 2,  $1 < HI < 2$  AU; Group 3,  $2 < HI < 5$  AU; Group 4  $\geq 5$  AU). Data was analysed using IBM SPSS 19.

**Results:** Scatter plots below show HI and MCT levels in samples from each collection site. Comparison of MCT levels in HI groups does not show an association between HI group and MCT when HI is  $< 2$  AU. This represents 77%, 82% and 54% of subclavian, femoral and aortic samples respectively. However, there is a correlation between MCT and HI in samples with an HI  $\geq 2$  AU. In vitro interference testing using a fixed concentration of MCT mixed with artificially variably haemolysed serum did not interfere with MCT detection.

**Discussion:** MCT appears to be independent of haemolysis if the HI is  $< 2$ . However, MCT results in grossly haemolysed samples may need to be interpreted with caution.



## POSTER

### 12. POST-MORTEM MAST CELL TRYPTASE IN NON-ANAPHYLACTIC DEATHS; DOES CAUSE OF DEATH HAVE A SIGNIFICANT EFFECT?

Mark Goulding<sup>1</sup>, Christine Bundell<sup>1,2</sup>, Andrew McLean-Tooke<sup>1</sup>, Jodi White<sup>1,2</sup>, Clive Cooke<sup>1,2</sup> and Peter Hollingsworth<sup>1,2</sup>

<sup>1</sup> Clinical Immunology, PathWest Laboratory Medicine WA

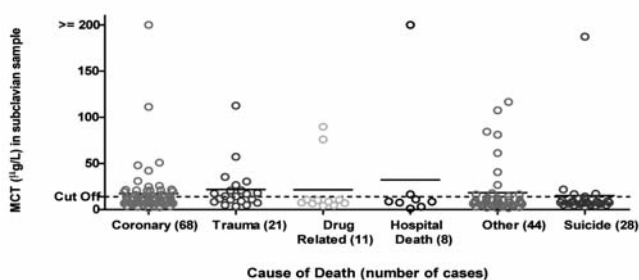
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**Aim:** To investigate the influence of cause of death on post mortem (PM) mast cell tryptase (MCT) levels.

**Background:** MCT is released following mast cell (MC) activation. MCT measurement in serum is used to infer MC activation in cases of suspect anaphylaxis. MCT measurement has also been used to investigate the possibility of death due to anaphylaxis. However, MCT levels in PM samples may be affected by the cause of death (COD).

**Method:** Blood samples were collected from the subclavian artery (on admission to mortuary) and from each of the aortic arch and femoral artery (at necropsy) in 89 and 98 consecutive permissible coronial PM cases in September 2009 and January 2010 respectively. COD category was determined by a forensic pathologist after necropsy. Anaphylaxis was not recorded as a COD in any of the cases received. Serum MCT levels were measured using the Phadia Immunocap 250. In samples with an MCT >200ug/L the value of 200 has been assigned.



**Results:** Figure 1 is a scatterplot showing MCT levels (including the mean) in the subclavian sample for each COD with a samples size of >2. The highest mean level of MCT (µg/L) is seen in the Hospital Death category (Subclavian = 32, Aortic = 54 and Femoral = 38). The MCT levels are higher in the aortic sample in all categories, however, the difference in mean MCT levels between groups at each site does not reach statistical significance at  $p < 0.05$ .

**Discussion:** The range of MCT results across the COD groups suggest that while the MCT assay is sensitive it will have a relatively poor specificity for anaphylaxis in post mortem samples.

## POSTER

### 13. ANAPHYLAXIS TO CITRUS FRUIT AND APPLE SEED

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**Summary:** Childhood fruit allergy is relatively common; however, there are very few reports of children reacting to fruit seeds in the absence of clinical reactivity to fruit pulp and no previous reports of apple seed allergy.

**Methods:** We present a case series of four children and evidence of serum specific IgE to apple seed in the child with anaphylaxis to apple seed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis and immunoblot.

**Results:** Three children presented with anaphylaxis (one at in-hospital challenge) after consuming orange (2 cases) or mandarin (1 case). Two of these children had known allergy to peanut and one to cashew. A fourth child, an 18 month old infant, had anaphylaxis following 2 teaspoons of baby apple food. All four children were skin test prick positive to the fruit seed in question and negative to the fruit pulp, and had previously tolerated the pulp of the fruits on many previous occasions. They were also sensitized to peanut, cashew and pistachio suggesting an association between nut and fruit seed allergy.

There are no previous reports of allergy to apple seed in the literature. We proceeded to demonstrate binding of IgE from the patient's serum to proteins from the apple seed extract, with particularly strong binding at 49kDa and ~70kDa on immunoblot. Binding was also observed to known allergens peanut and sesame, but not to the apple pulp, consistent with the SPT results.

**Conclusion:** We suggest that allergy to fruit seeds is far more common than currently appreciated but is probably under-recognized. Clinicians might not consider the role of seeds during the work-up for presumed fruit allergy, and confusion may arise if indeed symptoms only develop if the seed is cut or crushed. It is important to consider fruit seeds as a potential cause of fruit-associated allergic reactions.

## POSTER

### 14. SAFETY OF FOOD CHALLENGES IN A TERTIARY REFERRAL CHILDREN'S HOSPITAL

Karla Villafana-Soto, Sam Mehr, Preeti Joshi, Melanie Wong, Alyson Kakakios, **Dianne Campbell**

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**Background:** In-hospital observed food challenges (OFC) are increasingly used in the diagnosis and management of children with IgE and non IgE mediated food allergies. They play a particularly useful role in determining whether tolerance has been achieved in children with a past history of food allergy and in identifying clinical sensitivity in those children avoiding foods on the basis of IgE sensitisation alone. Safety of OFC has been an important consideration when centres consider setting up food challenge facilities.

**Methods:** All inpatient OFC performed during the period Jan 2010- May 2011 were reviewed for outcome of challenge, nature of reaction and whether adrenaline was administered. Symptoms of anaphylaxis were recorded as one or more of the following: tongue swelling, throat tightness, hoarse voice, stridor, wheeze, persistent cough, loss of consciousness or collapse.

**Results:** A total of 455 children underwent in hospital supervised OFC during the study period. 88 (19%) children reacted to the index food on challenge. The most common foods associated with a positive OFC were egg (34) and peanut (16) Of those positive challenges, 15 had signs and/or symptoms of anaphylaxis constituting 3% of the total challenge population. The foods associated with anaphylaxis on OFC were peanut (5), egg (including baked egg in muffin) (6), cow milk (1), tree nuts (2) and wheat (1). Only 7 children received adrenaline. Two children were given more than one dose of adrenaline, no cases required adrenaline infusion or admission to intensive care unit. There were no deaths or long term morbidity associated with any OFC during the study period.

**Conclusion:** OFC in the setting of a tertiary referral Children's Hospital appears safe. A low rate of children undergoing OFC required administration of adrenaline in our study. Provision of appropriate emergency equipment and ability to provide high dependency care should be ensured.

## POSTER

### 15. EXPERIMENTAL HETEROPHYIASIS: HISTOPATHOLOGICAL & IMMUNOLOGICAL STUDY

**Ahmed A. Daoud**<sup>1</sup>, Samy I. El-Kowranly<sup>1</sup>, Fetouh A. Deyab<sup>1</sup>, Kamal F. Eliwa<sup>1</sup>

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The present study was conducted to evaluate the effect of immunosuppression on course of infection, the extraintestinal pathological changes and immune complexes deposits in kidneys and brain tissues with *Heterophyes heterophyes* infection in mice. Seventy Swiss-albino mice were divided into four groups; G(I) 30 immunocompetent infected mice, G(II) 30 immunosuppressed by cyclophosphamide, infected mice and G(III) 5 non-infected immunocompetent control mice, G (IV) 5 immunosuppressed non-infected. Groups I & II were infected with 300 metacercariae/mouse orally. Two weeks post infection (p.i.) 5 animals from each group were sacrificed at 14, 16, 18, 21, 25 and 28 days p.i., and the kidneys and brain were processed for tissue digestion with KOH & histopathological and immunofluorescence examination. Adult worms were counted by mucosal scraping of the intestines. Result showed the adult worm count was higher in G(II) and G(I). Kidneys of G(I) mice showed mild congestion of the glomeruli with lymphoid aggregates. In G(II) mice, glomeruli showed size variation with mild thickening of their walls and the blood vessels showed moderate congestion with mild thickening of their walls. Brains in G (I) mice showed capillary haemorrhage with focal accumulation of endotheliocytes and histiocytes in a frame work of connective tissue. In G(II) mice, the brain showed congestion, oedema and hypercellularity. Gliosis accompanied with increased vascularity and endothelial hyperplasia was observed.

Mild immune complex deposits were detected from the 3rd week p.i. in G(I). The immunofluorescence reaction become moderate at the 4th week p.i. While in G(II) the immunofluorescence reaction was mild two weeks p.i. and became moderate at the 3rd week p.i. These results proved that the H. *Heterophyes* antigen or immune complex deposits were detected in the kidneys and brain of infected mice. These deposits play an important role in the histopathological changes in the kidneys and brain of infected animals.



## POSTER

### 16. BUDESONIDE AND FORMOTEROL REDUCE EARLY INNATE ANTI-VIRAL IMMUNE RESPONSES *IN VITRO*

Janet M. Davies<sup>1</sup>, Melanie L. Carroll<sup>1</sup>, Hongzhuo Li<sup>1</sup>, Alisa M. Poh<sup>1</sup>, Darren Kirkegard<sup>1</sup>, Michelle Towers<sup>1,2</sup>, John W. Upham<sup>1,2</sup>

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Asthma is a chronic inflammatory airways disease in which respiratory viral infections frequently trigger exacerbations. Current treatment of asthma with combinations of inhaled corticosteroids and long acting beta2 agonists improves asthma control and reduces exacerbations but what impact this might have on innate anti-viral immunity is unclear.

We investigated the *in vitro* effects of asthma drugs on innate anti-viral immunity. Peripheral blood mononuclear cells (PBMC) from healthy and asthmatic donors were cultured for 24 hours with the Toll-like receptor 7 agonist, imiquimod, or rhinovirus 16 (RV16) in the presence of budesonide and/or formoterol. Production of proinflammatory cytokines and expression of anti-viral intracellular signalling molecules were measured by ELISA and RT-PCR respectively.

In PBMC from healthy donors, budesonide alone inhibited IP-10 and IL-6 production induced by imiquimod in a concentration-dependent manner and the degree of inhibition was amplified when budesonide and formoterol were used in combination. Formoterol alone had little effect on these parameters, except at high concentrations (10<sup>-6</sup> M) when IL-6 production increased. In RV16 stimulated PBMC, the combination of budesonide and formoterol inhibited IFN $\alpha$  and IP-10 production in asthmatic as well as healthy donors. Combination of budesonide and formoterol also inhibited RV16-stimulated expression of the type I IFN induced genes myxovirus protein A and 2', 5' oligoadenylate synthetase.

These *in vitro* studies demonstrate that combinations of drugs commonly used in asthma therapy inhibit both early pro-inflammatory cytokines and key aspects of the type I IFN pathway. These findings suggest that budesonide and formoterol curtail excessive inflammation induced by rhinovirus infections in patients with asthma, but whether this inhibits viral clearance *in vivo* remains to be determined.

## POSTER

### 17. B CELL RESPONSES TO AREOALLERGENS IN PATIENT WITH ALLERGIC DISEASE

Julie Burel<sup>1</sup>, Hongzhuo Li<sup>1</sup>, Kelly Wong<sup>1</sup>, Michelle Towers<sup>1</sup>, John Upham<sup>1,2</sup>, Janet Davies<sup>1</sup>

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Grass pollen allergy is a major cause of allergic rhinitis and exacerbations of allergic asthma. IgE is the specific trigger for allergic reactions and clinical use of anti-IgE treatment ameliorates allergic diseases but surprisingly little is understood about the qualities of allergen-specific B cells. Our previous study of variable region repertoires of IgE transcripts from grass pollen allergic subjects indicated that IgE antibodies are typical of oligoclonally expanded, antigen-driven adaptive B cell responses but the subject number was small and allergen specificity was not determined. It remains controversial as to whether IgE+ B cells in allergy develop via the traditional pathway of activation of adaptive B cells in germinal centers.

We investigated B cell responses from blood of patients with grass pollen allergy and non-allergic healthy donors. Peripheral blood mononuclear cells were isolated and stained with a cytoplasmic dye carboxyfluorescein diacetate succinimidylester (CFSE) and cultured for 5 days with Bahia grass pollen allergen extract, FluVax 2009 (CSL), phytohaemagglutinin or without stimulus. Proliferation of cells was measured by dilution of CFSE and surface phenotype was monitored by flow cytometry.

B cells from grass pollen allergic donors showed higher proliferation to grass pollen allergen than healthy donors whereas responses to a viral antigen (FluVax) did not differ between patients and controls. Allergen-driven B cells that entered into division early (CD19+ CD3- CFSE<sub>lo</sub>) were CD27<sup>hi</sup> and CD20<sup>lo</sup>. Moreover, CD19+ CFSE<sub>lo</sub> CD27<sup>hi</sup> cells that rapidly divided in response to allergen showed higher staining for the plasmablast marker CD38 compared with undivided or CD19+ CFSE<sub>mid</sub> CD27<sup>lo</sup> cells.

These data are consistent with rapid *in vitro* expansion of circulating grass pollen allergen-specific memory B cells. Ongoing studies are focused on allergen specificity, B cell maturation and immunogenetic qualities of expressed immunoglobulin transcripts so as to investigate activation pathways of B cells in allergic disease.

## POSTER

### 18. A NEW TYPE OF HEREDITARY ANGIOEDEMA

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**Background:** The classic types of hereditary angioedema (HAE), HAE types I and II, are caused by a quantitative or qualitative deficiency of complement C1-inhibitor, due to mutations in the C1-inhibitor (SERPING1) gene. Mutations in the coagulation factor XII (F12) gene have been identified as one cause of so-called 'HAE type III' (HAE with normal C1 inhibitor), a condition that affects mainly women and shows normal C1-inhibitor measurements [Biochem. Biophys. Res. Comm. 343:1286-1289 (2006)]. Here we describe a large family with a dominantly inherited angioedema disease which segregates independently of the C1-inhibitor and the factor XII gene.

**Method:** Seventeen family members were personally interviewed; medical histories, pedigree information and venous blood samples were obtained. Polymorphic DNA markers representing the SERPING1 and the F12 locus were genotyped using PCR-based methods.

**Results:** Recurrent angioedema attacks occurred in four female and two male family members, from four subsequent generations. Tongue swellings, attacks involving the face, particularly the lips, and gastrointestinal symptoms dominated the clinical picture.

Segregation analysis of SERPING1 and F12 haplotypes indicated that the disease phenotype in this family segregates independently of the C1-inhibitor and the factor XII gene.

**Conclusion:** The dominantly inherited angioedema disease described here is neither caused by a complement C1-inhibitor mutation nor by a coagulation factor XII mutation. It, thus, represents a novel type of hereditary angioedema.

## POSTER

### 19. ALTERNARIA AND ALLERGIC DISEASES (ALLERGIC RHINITIS AND ASTHMA)

Iman Eftekharzadeh Mashhadi<sup>1</sup>, Monireh Mokhtari Amirmajidi<sup>1</sup>, Nemat Allah Mokhtari Amirmajidi<sup>2</sup>, Farahzad Jabari Azad<sup>3</sup>, Jalil Tavakol Afshari<sup>3</sup>, Mohammad Tohidi<sup>4</sup>.

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**Background:** Inhalation of *Alternaria* spores is shown to be important in the induction and severity of allergic rhinitis and asthma. Presence of *Alternaria* in the nasal mucous might be a trigger for the allergic process in these common diseases.

**Objectives:** To evaluate the relation between detection of *Alternaria* in the nasal mucous and allergic rhinitis and asthma.

**Methods:** In this case-control study, 58 patients with allergic rhinitis and 58 with allergic asthma were selected randomly in the allergy clinic according to the inclusion criteria - positive symptoms and signs, indicative pulmonary function test for asthma, and presence of atopy documented by skin prick test. They were compared with a randomly-selected well-matched control group (50 volunteers with none of the above criteria) for detection of *Alternaria* in their nasal mucous by culture. Within the case groups, severity was defined and its association with the detection of *Alternaria* was assessed.

**Results:** There was a significant association between both diseases and the presence of *Alternaria* in the nasal mucous. However, neither the severity of asthma nor rhinitis had a meaningful association with detection of *Alternaria* in the nasal mucous. Furthermore, relation between detection of *Alternaria* in the nasal mucous and some features of severity of asthma such as FEV1/FVC, number of admissions in the hospital was not significant.

**Conclusion:** This study verifies previous investigations showing relation between allergic diseases (asthma and rhinitis) and airborne concentration of *Alternaria*. Therefore, it seems advisable for the allergic patients to avoid exposure to *Alternaria*.

## POSTER

### 20. EVOGAM, THE FIRST LOCALLY PRODUCED SCIG; A STUDY OF EFFICACY, SAFETY, AND ACCEPTABILITY IN PID

Marianne Empson<sup>1</sup>, Mimi LK Tang<sup>2</sup>, Leon Rozen<sup>3</sup>, Lisa Pearce<sup>3</sup>

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**Introduction:** Subcutaneous Immunoglobulin (SCIG) therapy is an internationally accepted option for self-administered replacement therapy in PID. Australia and New Zealand utilise a self sufficiency model for plasma-derived products. Off-label IMiG administered SC is currently the only option under this model, with no locally produced SCIG. Evogam, a new chromatographically fractionated 16% SCIG, has been developed from domestically-produced plasma. The efficacy, safety, pharmacokinetics, and

quality of life impact of this product were investigated in a phase III, open-label, multi-centre study, using a 1:1 dose conversion from IVIg to SClg.

**Methods:** Thirty-five PID patients (30 adults, 5 children) previously treated with IVIg (n=34) or SClg (n=1) were recruited from nine major hospitals in Australia and New Zealand. Evogam was administered weekly for 36 weeks at a dose equivalent to their previous cumulative monthly dose. A syringe infusion pump was used; the first four infusions involved training and supervision at the hospital but subsequent infusions were self or guardian administered at home.

Primary endpoints were serious bacterial infections (SBIs) and steady-state serum immunoglobulin G (IgG) trough concentrations. Secondary endpoints included adverse events, infection episodes, antibiotic use, days off work/school, hospitalisation, pharmacokinetics and quality of life.

**Results:** No SBIs were reported during the study. The mean trough IgG concentration with SClg was significantly higher than with previous Ig treatment (8.94 versus 8.27 g/L,  $p = 0.0063$ ). Evogam was well tolerated with no withdrawals due to adverse events. Eighty three percent of subjects preferred SClg therapy.

**Conclusion:** Evogam was efficacious in prevention of infections and maintenance of trough levels using a 1:1 dose conversion. It was safe and well tolerated, and was preferred to IVIg by the majority of patients.

As an alternative to IVIg, Evogam offers a major advance in immunoglobulin replacement therapy for Australian and New Zealand patients.

## POSTER

### 21. IDENTIFICATION OF BIOMARKERS TO DISTINGUISH VARIOUS LEVELS OF COW'S MILK TOLERANCE

Lara Ford<sup>1</sup>, Katherine Bloom<sup>1</sup>, Anna Nowak-W grzyn<sup>1</sup>, Wayne Shreffler<sup>2</sup>, Madhan Masilamani<sup>1</sup>, Hugh Sampson<sup>1</sup>

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**Background:** About 80% of cow's milk allergic (CMA) children tolerate varying degrees of baked milk, improving prognosis and quality of life. We analysed basophil activation, milk- and casein-specific IgE and IgG4 and milk prick skin test (PST) to identify biomarkers that could distinguish varying degrees of clinical tolerance among a cohort of CMA children.

**Methods:** 132 subjects were classified as baked-milk-reactive, baked-milk-tolerant (muffin, pizza or rice pudding), or "outgrown milk allergy" based on oral food challenges. Serum samples were analysed for allergen-specific IgE and IgG4 levels, basophil

reactivity was assessed in whole blood stimulated with serial 10-fold dilutions of milk, and PST was performed to commercial milk extract. Activated basophils were defined using flow cytometry as CD63<sup>bright</sup> CD203c<sup>+</sup> CD123<sup>+</sup> HLA-DR<sup>dim</sup>/- CD41 $\alpha$ - lineage-. Differences between clinical groups in casein-specific IgE and IgG4 levels and IgE/IgG4 ratio; ratio of milk-specific to non-specific basophil activation, milk-specific basophil reactivity, and spontaneous basophil activation; and milk PST wheal diameter were analysed using the Jonckheere-Terpstra test (for ordered groups).

**Results:** Significant decreases across the five clinical groups, from baked-milk-reactive to "outgrown milk allergy", were seen for casein- and milk-specific IgE ( $p < 0.001$ ), casein-specific IgG4 ( $p < 0.05$ ) and casein IgE/IgG4 ( $p < 0.001$ ); milk-specific to non-specific basophil activation ratio ( $p < 0.005$ ), median basophil reactivity ( $p < 0.001$ ), and spontaneous basophil activation (CD203c expression following stimulation with RPMI;  $p < 0.05$ ); and milk PST wheal diameter ( $p < 0.001$ ). Casein- and milk-specific IgE, milk-specific basophil reactivity and PST wheal diameter are significantly greater among milk-allergic patients who react to baked milk than those who tolerate it.

**Conclusions:** Most milk-allergic patients can tolerate some forms of baked milk in their diets. Different phenotypes of CMA children can be distinguished by casein- and milk-specific IgE, milk-specific basophil reactivity, and milk PST mean wheal diameter. Spontaneous basophil activation is greater among patients with more severe clinical milk reactivity.

## POSTER

### 22. WHICH CHILDREN WARRANT A GLUTEN-FREE DIET?

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**Background:** The diagnosis of gluten-sensitivity is contentious. Clinical response to gluten-free diet in children with high anti-gliadin-antibodies, but without coeliac disease, was examined.

**Methods:** A clinical audit of sequential children referred to the Clinic, investigated for coeliac disease. Inclusion criteria: eating gluten prior to endoscopy; had elevated IgG-anti-gliadin antibody (Inova Diagnostics)  $> 14$  units; had measurements of tissue transglutaminase (tTG) or endomesial antibody (EMA). All children were offered a gluten-free diet, whatever the small bowel histology appearance, and reviewed after 3-6 months.

Results: 190 children were enrolled, (mean age 5.3 years, sd 3.8) and categorised into three groups:

31 (16%) with histology diagnosis of "definite coeliac".

31 (16%) assigned "possible coeliac" because of elevated tTG or EMA antibodies (but normal small bowel histology);

128 (67%) with no evidence of coeliac disease (normal small bowel histology and no elevation of tTG or EMA) – assigned “not-coeliac”.

Clinical and demographic features were similar across the three groups.

Intention to treat outcome: 81 of 128 (64%) “not-coeliac” reported substantial clinical improvement on the gluten-free diet. Of the remaining 47: 31 did not try gluten-free, and 8 reported no benefit. When gluten-free benefit was measured for only those who tried a gluten-free diet, then 81/97 (84%) of these “not-coeliacs” reported improvement.

**Conclusion:** Many children have symptoms consistent with coeliac disease, but have normal small bowel histology and normal tTG/EMA results. However, they frequently have high IgG-gliadin antibody levels. Such children can also respond to a gluten-free diet – they are gluten-sensitive. IgG-gliadin antibody is a test that can detect these children. Many more children, other than definite-coeliacs, warrant a gluten-free trial.

Patient group	Intention to treat: Clinical improvement on gluten-free diet
Definite coeliac	29/31 (94%)
Possible coeliac	21/31 (68%)
Not-coeliac (gluten-sensitive)	81/128 (64%)

## POSTER

### 23. EVALUATION OF THE EUROIMMUNE EUROLINE SYSTEMIC SCLEROSIS (NUCLEOLI) PROFILE (IgG) KIT

Richard Fulton<sup>1</sup>, Djemilla Hancox<sup>1</sup>, Lauren Frieze<sup>1</sup>, Rodger Laurent<sup>2</sup>, Stanley Seah<sup>2</sup>, Suran Fernando<sup>1,3</sup>

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**Background:** Systemic sclerosis is a disease of unknown aetiology that can affect multiple organ systems. It can be in a diffuse or limited form, the latter includes CREST Syndrome. Diagnosis is based on clinical findings but may be assisted by autoimmune serology.

The routine diagnostic antibodies include anti Scl70 and anti centromere antibodies however a wide range of antibodies may be detected with diagnostic and prognostic value. The Euroimmune Euroline Systemic Sclerosis blot detects 12 antibodies associated with systemic sclerosis. We assessed the utility of the assay in the clinical laboratory.

**Method:** Serum samples were collected from consecutive patients attending the RNSH scleroderma clinic. This group included patients with limited scleroderma and diffuse scleroderma. Disease control samples were obtained from patients with various autoimmune diseases including SLE, Sjogren’s syndrome, MCTD, Rheumatoid arthritis, and Hashimoto’s thyroiditis. Medical records were reviewed.

Samples were assayed using the Euroimmune systemic sclerosis (nucleoli) profile euroline (IgG) line blot as per the manufacturer’s instructions. Additionally samples were assayed for ANA (Immunoconcepts Hep2000) and ENA (in-house CIEP).

**Results:** The blot yielded a low specificity for scleroderma with antibodies to Ro 52, NOR90, PMScl75, PMScl100, Ku and CENP B being detected in the disease control population. Sensitivity for scleroderma was also low however antibodies to RP11 and RP155 were detected in otherwise seronegative samples.

**Conclusion:** The scleroderma blot is valuable both diagnostically and prognostically due to its ability to detect the less common autoantibodies such as RNA polymerase III, Th/To, NOR90 which are not reliably identified by routine laboratory tests. It is important however that clinicians be aware of the relative specificities of these antibodies.

## POSTER

### 24. CHILDHOOD ONSET AUTOSOMAL DOMINANT ORBITAL MYOSITIS IN AN AUSTRALIAN FAMILY

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**Abstract:** Idiopathic orbital myositis is a rare sporadic eye disease associated with extraocular eye muscle inflammation. We report 3 new Australian cases of familial orbital myositis (FOM). The children extend one of only two reported families with FOM, and confirm autosomal dominant inheritance.

**Cases:** A girl aged 14, presented with ptosis, proptosis and diplopia in all positions of gaze except primary position. CT scan of orbits demonstrated swelling of her right medial and superior rectus muscles, consistent with orbital myositis. She was initially treated with oral prednisolone but has followed a refractory course with multiple relapses and bilateral involvement, requiring methotrexate as a steroid sparing agent. Her brother presented aged 11 with diplopia and a lateral gaze palsy of his right eye, with CT confirming lateral rectus involvement. He responded to corticosteroids and has remained in remission for several years. A second cousin of the children, a boy, also presented with orbital myositis in childhood and has demonstrated a refractory course,

again requiring initiation of a steroid sparing agent. Four cases of FOM in this family were originally reported in 1999, including the children's mothers who both presented with orbital myositis in their teens. Both women have gone into remission in adult life.

**Conclusion:** The family presented here is informative regarding severity, age of onset and prognosis of FOM. The idiopathic form of orbital myositis rarely presents before adulthood, whereas all 7 cases of FOM in this family presented in childhood. This indicates an earlier onset of disease in FOM. The sporadic form of orbital myositis usually follows a benign course, particularly in children, whereas two of these patients have required a steroid sparing agent, suggesting that FOM may be more aggressive than the sporadic form. Finally, the history of remission for their mothers may indicate a relatively benign long-term prognosis.

## POSTER

### 25. A CASE OF RECURRENT FEVERS

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**Introduction:** We present the case of a 2 year 8 month old girl who presented to the paediatric clinical immunology clinic with multiple presentations of recurrent fever, pharyngitis, and lymphadenopathy.

**Case Presentation:** MR was 2 years old at first presentation. She was born full term, in an uncomplicated pregnancy and was breast fed for 6 months. From 6 months, she had multiple presentations to the Emergency Department and her GP with fevers up to 39°C, typically lasting 5 days, sometimes with accompanying tonsillitis, and bilateral axillary and cervical lymphadenopathy. The fevers usually responded to paracetamol or ibuprofen. She was well between episodes. She had normal developmental milestones. Both her parents were from Australia and of Caucasian ancestry. There was no symptoms of autoimmune disease.

Inflammatory markers were elevated during a few of these episodes. Blood, urine, and throat cultures and nasopharyngeal aspirates were repeatedly sterile. She was investigated for underlying autoinflammatory, autoimmune and immune deficiency.

**Conclusion:** This is a case of a young girl with recurrent fevers. Her results and differential diagnosis will be discussed.

## POSTER

### 26. A REVIEW OF THE EXPANDED SPECIALIST TRAINING PROGRAM IN ITS FIRST YEAR OF IMPLEMENTATION IN ADELAIDE

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In 2010 Adelaide put their first advanced trainee through the Expanded Specialist Training Program (ESTP). This is a federally funded program with the stated aim enabling medical specialist trainees to undertake training rotations in an expanded range of settings outside traditional public teaching hospitals. This involves funding a trainee to attend clinics in areas including private rooms.

The process of implementation was arduous but not insurmountable and the result was a federally funded advanced training position in addition to the traditional places available in Adelaide.

This position is suitable for a trainee in their last year of training and provided a different experience and different patient mix to the Royal Adelaide Hospital job while still providing adequate breadth of patients and supervision for training.

We illustrate our experience both in the implementation of the program and the experience of the supervisors and trainees with a view to aiding other centres whom may be considering the program.

## POSTER

### 27. A CONTROLLED STUDY OF DELTA-INULIN ADJUVANT IN HONEY BEE VENOM IMMUNOTHERAPY

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**Introduction:** Honey bee (HB) venom (HBV) immunotherapy

(HBVIT) reduces the frequency of immediate generalised allergic reactions (IGR) to subsequent sting by only about 80% and induces same in many subjects. Inulin has been used safely and extensively by intra-venous injection to study renal clearance and in studies involving influenza and hepatitis B vaccines has shown antigen sparing properties without evidence of activation of danger signal responses. In an ongoing, clinically double-blinded, study we have followed serological responses to HBVIT with and without inulin adjuvant.

**Methods:** 23 subjects with a history of IGR to HB sting and specific IgE to HB venom have been randomized 2:1 to receive HBVIT by semi-rush regime with (group A) or without (group B) GMP inulin mixed with venom. Assays for HBV specific IgE (sIgE) used CAP system and for IgG and IgG4 (sIgG and sIgG4), ELISA assay.

**Results:** Clinicians remain blinded. 2 subjects have withdrawn for personal reasons. There has been one IGR and one late systemic reaction to immunotherapy, both mild.

The striking difference has been in sIgG4 responses to HBV. Both groups showed peak responses at 14 weeks (early maintenance HBVIT), group A at mean 33 units (SEM 6.2), group B at 13 units (SEM 4.9) and these levels were well maintained in both. The response was far earlier in group A with mean 19 units (SEM 6.1) at 5 weeks following maximum dose 25 mcg HBV vs 3.5 (SEM 1.8) Group B. Similar but less pronounced trends were seen with sIgG responses. sIgE responses show a wide scatter but peak rise from baseline was less in group A (144% vs 231%) and return to baseline earlier (month 3 vs month 9).

**Conclusions:** With the caveat that only surrogate markers have yet been analysed, inulin appears to enhance wanted responses to HBVIT.

## POSTER

### 28. REVIEWING THE EXPERIENCE OF ULTRA-RUSH BEE VENOM DESENSITIZATION AT ROYAL ADELAIDE HOSPITAL. THREE YEARS RETROSPECTIVE AND ONE YEAR PROSPECTIVELY.

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**Introduction:** Honey bee (HB) venom (HBV) immunotherapy (HBVIT) reduces the frequency of immediate generalised allergic reactions (IGR) to subsequent sting by only about 80%. HBVIT is relatively safe compared to environmental stings but reactions during therapy can be severe and disruptive to the desensitization procedure. Royal Adelaide Hospital has been performing between twenty and thirty ultra-rush HBVIT's a year with the same protocol (last revised in 2006). This gives a wealth of data on

safety and reactions during immunotherapy. We reviewed, retrospectively for three years and prospectively for one year.

**Methods:** Retrospectively we looked at a number of factors including time since last sting, Sting reaction severity, IgE to HBV, the ratio of IgE to HBV vs total IgE, tryptase, medications and past medical history. These were then correlated with reactions during therapy.

Prospectively included rApi m1 and more details of effects of the immunotherapy including delayed symptoms and non-specific symptoms.

**Results:** Full analysis remains but there is a trend to increased frequency of reactions in the subsets with a high HBV IgE over Total IgE ratio. We also demonstrated safety of our protocol in the local population including patients with a high baseline tryptase.

## POSTER

### 29. TREAT THE SYNDROME. FORGET THE DIAGNOSIS.

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We present a case of a 58 year old man who presented with a sudden onset illness with many non-specific systemic symptoms including fever, arthralgias, myalgias and effusions. After an initial improvement he relapsed at 5 months with a similar but not identical clinical picture.

He now had a falling albumin and signs of capillary leak but intensive and exhaustive investigations yielded no diagnosis. Trial of methylprednisolone failed to alter his clinical state.

When he slipped into renal failure a renal biopsy yielded our first evidence of the pathological process allowing us to create a syndromic diagnosis that worked allowing targeted and effective empirical treatment.

## POSTER

### 30. LYMPHOPAENIA IN NON-HIV PATIENTS WITH PNEUMOCYSTIS JIROVECIJ DIAGNOSES

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**Introduction:** *Pneumocystis jirovecii* pneumonia (PCP) is a well described complication of an immunocompromised state, usually associated with HIV. Malignancy, malnutrition and immunosuppressant use are known risk factors for PCP in the HIV-negative population, presumably through affecting T helper

lymphocyte number and/or function. Indeed, low CD4 lymphocyte counts are associated with PCP in immunocompromised patients without HIV. Here we describe total lymphocyte and CD4 lymphocyte count in HIV-negative patients with *Pneumocystis jirovecii* positive respiratory specimens in one metropolitan Sydney hospital over the last 4 years.

**Method:** We retrospectively identified all *Pneumocystis jirovecii* diagnoses at Liverpool Hospital in Sydney between February 2007 and June 2011, and excluded those with known HIV infection. Total lymphocyte and absolute CD4 counts closest to the time of *Pneumocystis* diagnosis were recorded.

**Results:** 12 non-HIV infected patients with positive *Pneumocystis jirovecii* respiratory specimens were included in the final analysis. Diagnoses included chronic renal failure, transplant recipients, solid organ and haematological malignancies. Total lymphocyte count was available on all 12 patients and absolute CD4 count was available in 5 of those. There was an average of 4 days between *Pneumocystis jirovecii* diagnosis and total lymphocyte count, and an average of 8.7 days between diagnosis of *Pneumocystis jirovecii* and CD4 count. Mean total lymphocyte count was below normal at  $0.69 \times 10^9/L$  (range  $0.1-1.8 \times 10^9/L$ , normal range  $1.0-3.0 \times 10^9/L$ ). Mean CD4 count was low in 2 of 5 patients at  $50$  and  $210 \times 10^6/L$  respectively. Average CD4 count for all 5 patients was  $340 \times 10^6/L$  (range  $50-550 \times 10^6/L$ , normal range age dependent).

**Conclusions:** Serum CD4 count can be normal in PCP diagnosed immunocompromised hosts. Acquired impaired lymphocyte function, rather than number, may be the underlying risk factor for PCP acquisition in this group.

## POSTER

### 31. INTRAGAM 10 NF IN PRIMARY IMMUNE DEFICIENCY

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**Background:** Intragam® 10 NF is the next generation 10% intravenous immunoglobulin (IVIg) with three pathogen reduction steps and a non-carbohydrate stabiliser. This open label, crossover study in patients with primary immunodeficiency (PID) was designed to evaluate whether Intragam 10 NF differed in its pharmacokinetics (PK) compared to Intragam P, and to assess Intragam 10 NF safety and tolerability.

**Methods:** Nineteen PID patients were administered one cycle of their existing Intragam P dose (0.2-0.8 g/kg 3-4 weekly), followed by seven cycles of Intragam 10 NF, administered at the same dosing schedule as Intragam P. The primary objective was to compare serum immunoglobulin G (IgG) trough levels. Secondary endpoints were PK variables, safety and tolerability.

**Results:** There was no significant within patient difference in the average trough IgG concentration ( $C_{min}$ ) between Intragam P and Intragam 10 NF (8.76 g/L, 8.55 g/L respectively) (Geometric mean ratio 1.034; 95% CI: 0.996 to 1.073;  $p=0.079$ ). Mean PK parameters for both products were similar, with all 95% CI encompassing 1.0, except for time to maximum concentration ( $T_{max}$ ).  $T_{max}$  occurred earlier with Intragam 10 NF compared to Intragam P due principally to a shorter infusion time (mean 1.75 hrs versus 2.52 hrs respectively;  $p<0.05$ ). Headache was the most frequent treatment-related event following both products. There were no study withdrawals, deaths or notable changes in laboratory values or vital signs.

**Conclusion:** Intragam 10 NF was well tolerated and exhibited similar pharmacokinetics to Intragam P, with the advantage of a 45 minute shorter infusion time.

## POSTER

### 32. EVALUATION OF ANGIOGENIC AND FIBROGENIC CYTOKINES AS BIOMARKERS IN SUBGROUPS OF SYSTEMIC SCLEROSIS

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**Background:** The role of various angiogenic and fibrogenic cytokines in the pathogenesis of systemic sclerosis (SSc) is controversial.

**Aim:** To investigate the levels of various angiogenic and fibrogenic cytokines in patients with SSc with different clinical phenotypes.

**Methods:** Fifty six patients with SSc were included in this pilot study. They were further stratified into three main disease phenotypes: 1) SSc patients with PAH (n=14), 2) SSc patients with disproportionate fall in DLCO (<65%) with normal FEV1 and FVC but no evidence of PAH on 2D echocardiography and/or right heart catheter studies (n=17) and 3) SSc patients without known lung disease (n=25). Twenty two normal healthy control subjects with no evidence of connective tissue disease were

included. Fractalkine, FGF2, PDGF AB/BB, VEGF and G-CSF levels were measured on ALBIA and NTPro-BNP was measured by electrochemiluminescence (ECLIA) immunoassay. Statistical analysis used a non-parametric method, the Kruskal-Wallis test.

**Results:** Fractalkine, FGF-2 and NTPro-BNP levels were increased in SSc patients compared with healthy controls ( $p < 0.05$ ). PDGF AB/BB levels were significantly reduced in SSc patients compared with healthy subjects ( $p < 0.05$ ). There was no difference in VEGF and GM-CSF levels between SSc patients and healthy subjects. Among the SSc patients, NTPro-BNP levels were significantly different in all the three sub-groups (group 1 > group 2 > group 3) with the highest levels seen in patients with PAH ( $p < 0.001$ ). PDGF AB/BB levels showed the opposite trend with decreasing levels in the three subgroups (group 1 < group 2 < group 3) ( $p < 0.05$ ). The levels of fractalkine and FGF-2 did not differ among the SSc patient subgroups.

**Conclusion:** In addition to elevated levels of NTPro-BNP, reduced PDGF AB/BB identifies SSc patients at risk of developing PAH. Further studies are required to validate these initial observations and to evaluate the usefulness of serial measurements of these cytokines to predict patients at risk of developing PAH.

## POSTER

### 33. BETA-LACTAM ANTIBIOTIC USE BY PATIENTS AFTER NEGATIVE SKIN TESTING AND CHALLENGE

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**Introduction:** Patients with a history of possible IgE-mediated allergy to penicillins are typically investigated by skin testing and subsequent oral challenge in skin test negative individuals. There is little information on the willingness of these patients to subsequently use the challenged antibiotic. This study questioned whether such patients had taken or were willing to take the challenged drug, if prescribed.

**Methods:** Ethics approval was obtained for data collection and patient follow-up. We identified 83 patients who had negative incremental challenges with penicillins, typically 1/100th, 1/10th and full dose, at Auckland City Hospital, from April 2007 to November 2009. Previous skin testing, using major and minor penicillin determinants, benzylpenicillin, amoxicillin and/or Augmentin and/or flucloxacillin had been negative in all patients. These patients were phoned (RK or KL), and a standard questionnaire used to determine whether the challenged antibiotic had subsequently been prescribed, had actually been taken by the patient and if any adverse reaction had occurred.

**Results:** Of the 83 challenge-negative patients (61% female; mean age 47y) 75% (62/83, 58% female) were contactable and willing to participate. The interval after challenge was typically >12 months. Forty-five of the 62 (73%) had either already taken ( $n=14$ ) or were willing to take ( $n=31$ ) the relevant antibiotic while more than a quarter (27%, 17/62, 82% female) were unwilling to take the antibiotic. Two patients had experienced adverse effects after taking the antibiotic: skin rash ( $n=1$ ) and diarrhoea ( $n=1$ ).

**Conclusion:** More than a quarter of these patients were reluctant to use the relevant penicillin despite negative skin tests and challenge. The resource utilisation of skin testing and supervised challenge is considerable and has been only partly effective. We plan a follow up study to further examine the reasons for drug refusal, aiming to improve communication/education with patients and family practitioners.

## POSTER

### 34. CEREBROSPINAL FLUID FREE IMMUNOGLOBULIN LIGHT CHAIN ANALYSIS IS A MORE SENSITIVE MARKER OF INTRATHECAL IMMUNOGLOBULIN PRODUCTION THAN OLIGOCLONAL BANDS IN A PEDIATRIC POPULATION WITH INFLAMMATORY CENTRAL NERVOUS SYSTEM DISORDERS

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**Background:** Inflammatory and autoimmune disorders of the central nervous system (CNS) are a diverse group of disorders for which there are no definitive diagnostic tests. Detection of intrathecal immunoglobulin production can assist with the diagnosis in the appropriate clinical context. Current tests for this include detection of oligoclonal bands in cerebrospinal fluid (CSF) and demonstration of an elevated CSF index. Both are limited by poor sensitivity and/or specificity. Recent studies show an association with Multiple Sclerosis of detection of free immunoglobulin light chains, in an adult population. We present the results of an investigation of the utility of CSF free light chain analysis in a pediatric population with inflammatory CNS conditions.

**Materials and methods:** Consecutive paired samples of serum and CSF samples from children <18 years old collected at the Westmead Children's Hospital and sent to the Royal Prince Alfred Hospital Immunology Laboratory for analysis between March 2009 and February 2011 were included. Samples were tested for 1) CSF free light chains, 2) oligoclonal bands and 3) IgG and



free light chain index. The clinical diagnosis was determined by retrospective review of clinical notes by a paediatric neurologist.

**Results:** 55 patients with a diagnosis of CNS inflammation and 97 control patients with non-inflammatory disorders were included for analysis. Detectable free kappa and/or lambda light chains in CSF have a specificity of 40.0% and sensitivity of 87.60% for diagnosis of a CSF inflammatory disorder. Using a higher cut off level to achieve a specificity of 100%, the sensitivity was 29%. In comparison, CSF oligoclonal bands had a sensitivity of 10% and specificity 100%.

**Conclusion:** In a novel study investigating the utility of CSF free light chain analysis in a pediatric population, we demonstrate that their detection in CSF is a more sensitivity assay for demonstration of intrathecal immunoglobulin synthesis than oligoclonal band testing for diagnosis of CNS inflammatory disorders.

## POSTER

### 35. COMPARISON OF THREE METHODS FOR THE DETECTION OF IgG ANTIBODIES TO *ASPERGILLUS FUMIGATUS* IN THE DIAGNOSIS OF HYPERSENSITIVITY PNEUMONITIS

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The diagnosis of hypersensitivity pneumonitis is based on characteristic clinical features including a history of specific antigen exposure and demonstration of an antigen-specific IgG response. One of the most widely used methods to detect specific IgG is immunoprecipitation, although this method has many disadvantages including: poor reproducibility, subjective analysis, expense of commercial kits, long turnaround time and non quantitative results. Enzyme linked immunosorbent assays (ELISA) have been found to have increased sensitivity and better reproducibility than precipitin techniques but suboptimal specificity which may lead to false-positive results.

We compared results for the detection and quantitation of specific IgG antibodies to *Aspergillus Fumigatus* (M3) on the Immulite 2000 (Siemens Healthcare Diagnostics, Los Angeles, CA, USA) a liquid phase automated chemiluminescence assay, the ImmunoCAP 250 (Phadia, Uppsala, Sweden), an automated fluorescence enzyme immunoassay (FEIA) and immunoprecipitation by non-quantitative double diffusion (Microgen Bioproducts, Camberley, U.K). All assays were performed according to manufacturers' instructions.

Using an internally established cut-off of 80mg/L on both automated platforms the following correlations were achieved:

Immunoprecipitation / Immulite 2000: 84% concordance, kappa statistic: 0.69 (95% CI 0.36-1.01)

Immunoprecipitation / ImmunoCAP 250: 74% concordance, kappa statistic: 0.48 (95% CI 0.08 to 0.87) Immulite 2000 / ImmunoCAP 250: 74% concordance, kappa static: 0.44 (95% CI 0.22-0.67)

This study highlights the method specific variability in detection of antibodies to *Aspergillus Fumigatus*. This may be related to antigen specificity or differences in the avidity of antibody detected. Automated methods of testing reduced subjectivity, increased reproducibility and were more economical than the commercial immunoprecipitation method. Further study is required to ascertain optimal cut-off values for automated methods.

## POSTER

### 36. SYSTEMIC TACROLIMUS FOR THE TREATMENT OF SEVERE GENERALISED ATOPIC ECZEMA IN ADULTS: A CASE SERIES

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Severe atopic eczema (AE) is frequently refractory to conventional topical therapies. Whilst systemic corticosteroids, such as prednisolone can aid remission, side-effects limit their long-term use. Of the available immunosuppressant agents, the calcineurin inhibitor cyclosporine in particular has demonstrated effectiveness in severe eczema. Little data exists however, for the systemic use of another calcineurin inhibitor, tacrolimus, which is effective as a topical preparation. We report a small, novel case series of adults with severe, corticosteroid-dependent AE, not responsive to multiple other immunosuppressants, that were trialled on oral tacrolimus.

Patients with severe AE from the authors' clinical service were offered treatment with oral tacrolimus (5 mg twice-daily) from August 2008. The selected patients were reviewed clinically, underwent regular blood testing and changes in their prednisolone doses were monitored. All patients were previously assessed for sensitisation to aeroallergens.

Four adults with severe AE refractory to multiple medical treatments underwent treatment with oral tacrolimus. One patient failed to show any response. Three patients showed an initial clinical response, allowing reductions in their concomitant doses of prednisolone. Of these, one patient remains on tacrolimus as monotherapy, but the other two relapsed into severe disease, requiring an increase in prednisolone dose whilst still taking tacrolimus, which was ultimately

ceased. Oral tacrolimus was well-tolerated in this small group of adults, and there were no serious adverse events, although all patients reported a reversible mild tremor.

This is the first reported case series using systemic tacrolimus for atopic eczema. Despite its poor efficacy with only one responder, tacrolimus may yet have a potential role in eczema of lesser severity. However, larger prospective studies are required to qualify its place amongst existing immunosuppressive options in severe AE.

## POSTER

### 37. THE RELATIONSHIP BETWEEN PAEDIATRIC ATOPIC DERMATITIS AND PARENT QUALITY OF LIFE IS COMPLEX

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This study examines the impact of the severity of paediatric illness on the quality of life (QOL) of the patient's parent, in the setting of atopic dermatitis (AD).

**Methods:** Children between the ages of 2 and 17 years that present to the Paediatric Immunology Clinic at Campbelltown Hospital, Sydney, Australia, with AD have their disease severity assessed using the Scoring Atopic Dermatitis (SCORAD) tool. Their accompanying parent completes a Parents' Index of Quality of Life in Atopic Dermatitis (PIQoL-AD) questionnaire which is disease specific and scores the impact of the child's AD on the parent and family. Demographic information and atopic history is also collected. Children are reassessed at subsequent visits and tracked longitudinally. This study is an audit of this data.

**Results:** Preliminary results of 46 patients and their parents show a negative correlation between severity of illness and parent QOL,  $r=0.414$  ( $p=0.004$ ). Mothers ( $n=36$ ) had QOL scores with a stronger association with severity of illness ( $r=0.453$ ,  $p=0.006$ ) than fathers ( $n=4$ ) ( $r=0.168$ , not statistically significant). Stratification by severity suggests a much stronger relationship between the parent's QOL and the child's SCORAD in those with a SCORAD under 50 ( $n=36$ ),  $r=0.444$  ( $p=0.007$ ), as opposed to children with a higher disease severity ( $SCORAD \geq 50$ ,  $n=10$ ),  $r=0.060$  ( $p=0.868$ ). The strongest correlation between QOL and SCORAD exists in the parents of the 2-4 year old age group ( $n=12$ ),  $r=0.587$  ( $p=0.045$ ). This is much stronger than for children aged over 4 years old,  $r=0.358$  ( $p=0.038$ ).

**Conclusion:** This preliminary data indicates a complex relationship between disease severity and the effects it has on parental QOL, in the setting of atopic dermatitis. This relationship, and the factors which influence it, may become more evident as we examine a larger sample size.

## POSTER

### 38. THE ASSOCIATION BETWEEN MATERNAL OBESITY AND CHILDHOOD ASTHMA – AN ANALYSIS BASED ON SWEDISH SIB-PAIRS

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**Background:** The prevalence of both asthma and obesity have increased in westernised countries over recent decades. It has been proposed that maternal obesity during pregnancy may induce a pro-inflammatory intrauterine environment, which may increase the child's risk of asthma and allergic disease. Sib-pair analysis is a powerful technique for assessing the possibility that an exposure is causal for an outcome.

**Methods:** The study population comprised all children born between 1998 and 2005 in Stockholm ( $n=99,830$  born to 43,103 separate mothers) registered on the Swedish Medical Birth Registry. Maternal BMI was typically measured typically at 8-10 weeks post conception. Use of asthma medications (either inhaled corticosteroids or montelukast) was recorded in the Swedish Prescription Registry between July 2005 and February 2011. Conditional logistic regression models were used to assess the effect of changing maternal BMI on asthma medication use within sibling pairs matched for age. Adjustment was made for maternal smoking during pregnancy, pregnancy complications, the child's gender and other potential confounders.

**Results:** There were 4,311 children with siblings with discordant asthma medication use between 5 and 9 years of age. There was a trend for children born to obese mothers (30-34.9 kg/m<sup>2</sup>) to have an elevated risk ( $aOR=1.53$ ,  $95\%CI=0.88-2.65$ ) of asthma medication use when compared to their matched sibs. Children born to very obese mothers ( $BMI > 35$  kg/m<sup>2</sup>) had a much greater risk of asthma than their siblings ( $aOR=4.45$ ,  $95\%CI=1.79-11.05$ ).

**Conclusion:** Maternal early pregnancy obesity is associated with increased risk of asthma in the child. These associations are unlikely to be due to shared genetic or other familial risk factors for obesity and asthma, as the reported associations are based on a sib-pair analysis. Maternal obesity, or changes in lifestyle factors that lead to it, appear to cause an increase risk of childhood asthma.

## POSTER

### 39. NMDA RECEPTOR ANTIBODY POSITIVE NEUROLOGICAL SYNDROMES – THE WESTERN AUSTRALIAN EXPERIENCE

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**Introduction:** The N methyl D aspartate receptor (NMDAR) is a post synaptic neurotransmitter receptor expressed on muscle, postganglionic and central nervous system neurons. Antibodies against the NR1 subunit of NMDAR are associated with both paraneoplastic and non-paraneoplastic forms of limbic encephalitis.

**Method:** Since 2008, 11 patients in Western Australia have tested positive for NMDAR antibodies. NMDAR antibody testing was performed either at PathWest QEII and confirmed at the Oxford Radcliffe Laboratory (4 cases), at the Oxford Radcliffe Laboratory alone (6 cases) or at the University of Pennsylvania School of Medicine (1 case). IgG anti-NMDAR antibodies were detected by indirect immunofluorescence on Euroimmun mosaic slides comprising transfected Human Embryonic Kidney (HEK) 293 cells, rat hippocampus and cerebellum. Serial samples submitted for testing to monitor antibody levels were tested concurrently in dilution on the Euroimmun transfected cell slides.

**Results:** 8 female and 3 males patients were identified. The average age of NMDAR antibody positive patients was 38 years (male 53, female 32), with a range between 1 year and 71 years. We have been unable to find a younger patient described in the literature. 10 patients presented with clinical features suggestive of limbic encephalitis. 1 patient presented with weakness and bulbar palsy and was thought to have motor neurone disease. Teratomas were identified and removed in three of the females. Follow up testing was performed for the infant and two female adults. One patient had a clinical relapse with detectable antibodies at a 1:100 dilution. Apart from the patient who was thought to have motor neurone disease (who subsequently died), all patients clinically improved using various immunosuppressive treatments.

**Conclusion:** Detection and monitoring of anti NMDA R antibodies is essential for the prompt identification of NMDAR associated limbic encephalitis and may be useful evaluating the effectiveness of therapy.

## POSTER

### 40. THE PROCESS OF DEVELOPING A PATIENT INFORMATION RESOURCE FOR ALLERGIC RHINITIS

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Allergic Rhinitis (AR) is a chronic allergic condition affecting millions of people worldwide. The non life-threatening, chronic nature of AR has meant it has often been trivialised by both sufferers and health-care professionals. Consequently, AR is often under-treated, ineffectively treated or not treated at all (Hu et al. 2008). Self-management of AR requires effective evidence-based verbal and written information. There is a dearth of understandable, written information about diagnosis and management of AR for patients to source.

The aim of this project was to produce a written, evidence-based patient information resource about Allergic Rhinitis to address issues which lead to it being ineffectively treated.

A pre-production questionnaire was distributed to sufferers of AR to determine what information would satisfy their needs. An Ear Nose & Throat (ENT) specialist provided input of required clinical information. This information was collated and a pamphlet produced using design criteria which accommodated differing literacy levels and learning styles of patients.

The resource, together with a post-production questionnaire was distributed, to the same respondents as the previous questionnaire and to the ENT specialist to gain feedback on the pamphlet's contents and presentation. Changes were then made based on this feedback to produce the final draft of the pamphlet which will be presented at the ASCIA conference.

The pamphlet satisfied the information and learning needs of AR sufferers and the clinical requirements of the ENT specialist. While there are limitations to the pamphlet, such as its availability only in English, there is potential for it to be further developed to meet the needs of a wider audience.

Hu, W, Katelaris, C. H & Kemp, A.C 2008, 'Allergic Rhinitis; practical management strategies', *Australian Family Physician*, vol 37, no. 4, pp. 214-220.

## POSTER

### 41. PERFORMANCE OF QUANTIFERON-CMV® AND WHOLE-BLOOD CD4(+)/CD25(+)/CD134(+) ASSAYS IN IDENTIFICATION OF HUMAN CYTOMEGALOVIRUS-SPECIFIC T-CELLS IN CHILDREN POST-HAEMATOPOIETIC STEM CELL TRANSPLANTATION

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**Background:** Human Cytomegalovirus (HCMV) infection remains a significant cause of morbidity and mortality in patients following haematopoietic stem cell transplantation (HSCT). HCMV DNA polymerase chain reaction is the standard assay for guiding antiviral treatment. However, its poor specificity can lead to treatment with potentially toxic medications in many patients who would not progress to disease. Measurement of HCMV-specific T-cells can potentially be applied to help stratify risk of CMV disease and guide early treatment.

**Methods:** Our prospective, multicentre study evaluated the performance of the QuantiFERON-CMV assay (QFN-CMV) based on enzyme-linked immunosorbency and the novel Whole Blood CD4(+)/CD25(+)/CD134(+) assay (WB 4/25/134) based on 4 colour flow cytometry against the more labour-intensive Intracellular Cytokine Staining (ICS) assay for CD8 and CD4 responses respectively, in 25 children at defined time-points post-HSCT. ICS for Interferon-gamma and Interleukin-2 was performed in CD8 T-cells stimulated by the same HCMV peptides used in the QuantiFERON-CMV assay and in CD4 T-cells stimulated by HCMV lysate. All assays were compared against HCMV serostatus in a cohort of healthy adults.

**Results:** Table 1: A. Performance of assays using HCMV serostatus in healthy adults

B. Performance of QFN-CMV and WB 4/25/134 in patients post-HSCT using ICS as the "gold standard" for CD8 and CD4 responses respectively.

**Conclusion:** Performance of QuantiFERON-CMV and WB 4/25/134 assays appear promising compared to ICS in detection of CMV-specific CD8 and CD4 T-cell responses respectively, in children post-HSCT. Our study is the first to report the

performance of these assays exclusively in paediatric patients following HSCT. Further evaluation is required in this clinical setting.

A. Tests	Sensitivity	Specificity
ICS CD4	91% (10/11)	100% (3/3)
QFN-CMV	73% (8/11)	74% (26/35)
ICS CD8	91% (10/11)	100% (3/3)
WB 4/25/134	86% (18/21)	100% (32/32)
B. Tests	Sensitivity	Specificity
QFN-CMV	77% (10/13)	100% (4/4)
WB4/25/134	100% (11/11)	100% (3/3)

## POSTER

### 42. IMPAIRED IMMUNE RESPONSE TO INFLUENZA VACCINATION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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**Background:** Clinical guidelines recommend influenza vaccination in chronic obstructive pulmonary disease (COPD), though this is largely based on observational studies and a detailed examination of the immune response to vaccination in COPD has not been undertaken.

**Aims:** To assess the immunogenicity of influenza vaccination in COPD and the clinical and laboratory factors associated with vaccine responses.

**Methods:** Blood was collected prior to trivalent inactivated influenza vaccination and one month after vaccination from twenty one COPD patients, of whom 9 were current smokers and 14 used inhaled steroids. Fourteen healthy subjects of similar age served as controls. H1N1-specific antibody titres were measured by haemagglutination inhibition assay, while cellular responsiveness was assessed using blood mononuclear cells stimulated *in vitro* with vaccine and then analyzed by flow cytometry and ELISA.

**Results:** Seroconversion, defined as a  $\geq$  four-fold rise in Ab titre, occurred in 90% of healthy controls but in only 43% of COPD patients. Post vaccination Ab titres were significantly lower in COPD patients than in healthy controls ( $p=0.02$ ), and this was associated with lower serum IL-21, a cytokine that is important for B cell development and Ab synthesis (COPD  $50 \pm 130$  pg/ml; healthy controls  $75 \pm 84$  pg/ml;  $p<0.01$ ). *In vitro* functional

differences were also observed between COPD patients and healthy controls, with fewer proliferating B cells expressing CD27 (COPD  $38.1 \pm 18.5\%$ ; healthy controls  $51.2 \pm 17\%$ ;  $p = 0.04$ ) and reduced T-cell IFN $\gamma$  synthesis (COPD  $5.1 \pm 3.4$  ng/ml; healthy controls  $15.3 \pm 12$  ng/ml;  $p < 0.01$ ). Multivariate analysis showed that post vaccination Ab titres varied in association with having COPD and with previous influenza vaccination, but not with age.

**Conclusion:** COPD is associated with an impaired immune response to influenza vaccination.

## POSTER

### 43. A SPECIFIC MIXTURE OF NON-DIGESTIBLE OLIGOSACCHARIDES ENHANCES THE TOLERIZING CAPACITY OF A PARTIAL WHEY HYDROLYSATE IN A MOUSE MODEL FOR COW'S MILK ALLERGY

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Hypoallergenic infant formulas (HA) are considered a good alternative for infants at high risk for developing allergy if breastfeeding is not possible. Dietary intervention studies with HA combined with a specific mixture of non-digestible oligosaccharides, have been shown to reduce allergic symptoms in these children. However, the mechanisms by which these oligosaccharides exert their effect are yet to be explored. In this study, the contribution of this specific oligosaccharides mixture on the tolerizing capacity of a partial whey hydrolysate (WH) was investigated in a mice model of cow's milk allergy.

Mice were sensitized orally with whey using cholera toxin as adjuvant. Prior to sensitization mice were pre-treated orally with partial WH, PBS, with or without supplementation with the specific oligosaccharide mixture containing short chain-galacto-, long chain-fructo- and acidic-oligosaccharides (9:1:1). After challenge, the acute allergic skin response, the mast cell mediator mMCP-1 and whey-specific antibodies were measured. The presence of Foxp3<sup>+</sup> regulatory T-cells and CD103<sup>+</sup> DC were determined in mesenteric lymph nodes.

Oral pre-treatment of mice fed the partial WH induced tolerance as reflected by a reduced acute allergic skin response and a suppressed mMCP-1 release without affecting whey-specific IgE levels. This effect coincided with increased CD103<sup>+</sup> DC and Foxp3<sup>+</sup> regulatory T-cell numbers. Interestingly, a combination of

the partial WH and oligosaccharide diet completely abolished the acute allergic skin response and mMCP-1 release. In addition, a tendency towards decreased IgE levels and a further increase in intestinal CD103<sup>+</sup> DC numbers was observed.

A specific mixture of non-digestible oligosaccharides enhanced the capacity of a partial WH to induce oral tolerance. This effect was associated with increased numbers of CD103<sup>+</sup> DC in the mesenteric lymph nodes, suggesting a role of these cells in the observed tolerance inducing capacity of this specific oligosaccharide mixture combined with partial WH.

## POSTER

### 44. DIETARY INTERVENTION WITH SYNBIOTICS PROTECTS AGAINST ALLERGIC DISEASE VIA INDUCTION OF GALECTIN-9 BY INTESTINAL EPITHELIAL CELLS

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Intestinal epithelial cells (IEC) abundantly express galectins, which are known to modulate T cell responses. In this study, immune modulation and epithelial expression of galectin-9 (Gal9), induced by a specific oligosaccharide mixture of short chain galacto- and long chain fructooligosaccharide (scGOS/lcFOS) and TLR9 ligand and its relevance for the suppression of allergic disease were determined both in vitro and in vivo.

Human IEC were exposed to scGOS/lcFOS together with TLR ligands, co-cultured with PBMC and cytokines and immune cell phenotype were measured. In vivo, mice were sensitized orally to whey, fed a diet containing *Bifidobacterium breve* M-16V and scGOS/lcFOS (GF/Bb). Gal9 expression was determined by immunohistochemistry and by ELISA. In addition, in a double-blind, placebo-controlled multicentre trial, Gal9 levels were measured in sera of 90 infants with atopic dermatitis that received hydrolyzed formulae with or without GF/Bb for 12 weeks.

Apical exposure of IEC to scGOS/lcFOS and TLR9 ligand or genomic DNA from Bb M-16V enhanced IFN- secretion by co-

cultured PBMC and resulted in increased percentages of Th1 and Treg cells. The expression and secretion of IEC-derived Gal9 increased after combined addition of scGOS/lcFOS and TLR9 ligand. Furthermore, development of Th1 and Treg cells was enhanced in Gal9 treated PBMC, resulting in increased IL-10 and IFN- $\gamma$ , but suppressed IL-17. In vivo, the GF/Bb diet resulted in reduced acute ear swelling response upon dermal challenge with allergen and lower serum mMCP-1. Furthermore, the GF/Bb diet enhanced serum Gal9 levels, which correlated with decreased allergic symptoms and immunohistochemistry revealed specific basolateral Gal9 expression on IEC. In addition, infants suffering from atopic dermatitis receiving the GF/Bb diet also showed enhanced Gal-9 levels in serum, which coincided with less severe allergic symptoms.

These data indicate that dietary supplementation with scGOS/lcFOS has significant implications for the prevention of allergy through TLR9-induced Gal9 secretion by IEC.

## POSTER

### 45. NUTRITION ECONOMIC IMPACT OF A SPECIFIC MIXTURE OF PREBIOTICS IN PRIMARY PREVENTION OF ATOPIC DERMATITIS

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**Introduction:** Previous studies have demonstrated that an infant formula containing a specific mixture of prebiotics (8 g/L scGOS/lcFOS, 9:1, IMMUNOFORTIS®) is effective in the primary prevention of atopic dermatitis (AD). Our work aimed to evaluate the cost-effectiveness of this specific infant formula for the primary prevention of AD.

**Methods:** We developed a model to estimate the health economic impact of prebiotic preventive disease management of AD. Data sources used include published literature, clinical trials, official price/tariff lists and national population statistics. The model takes into account a cohort of children at risk for allergy, in line with the population of the clinical studies mentioned above.

Cost-effectiveness was calculated using a Markov model reflecting treatment patterns and outcomes in the management of AD. The analysis is conducted from the perspective of the health insurance in The Netherlands in 2009.

**Results:** The results show that the use of an infant formula with 8g/L scGOS/lcFOS (9:1) leads to an increase in Quality Adjusted Life Years (QALY) of 0.108, when compared with the same infant formula without this prebiotic mixture. Consequently, the use of infant formula with a specific mixture of prebiotics results in an Incremental Cost-Effectiveness Ratio (ICER) of € 472. Sensitivity analyses show that the ICER remains in all cases far below the Dutch threshold of € 20,000/QALY.

**Conclusion:** This assessment shows that, in addition to the health benefits and the improved quality of life during childhood, the use of an infant formula containing 8g/L scGOS/lcFOS (9:1) also results in positive nutrition economic outcomes in a cohort of children at risk for allergy. Furthermore the study demonstrates that the use of this infant formula is a highly cost-effective way of preventing AD in The Netherlands. On the long term this may lead to substantial cost savings for the health care budget.

## POSTER

### 46. URTICARIA DUE TO CLONORCHIS SINENSIS IN VIETNAM: IMPLICATIONS

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**Background:** *Clonorchis sinensis* (*Cl. sinensis*), the oriental liver fluke, belongs to the Opisthorchidae family. Clonorchiasis is endemic in many Asian countries, such as China (including Hong Kong and Taiwan), Japan, Korea and northern Vietnam (Rim, 2005). In 2004, the WHO estimated that over 600 million individuals are at risk of being infected with *Cl. sinensis*. Infestation with *Cl. sinensis* occurs mainly in the Red River delta in northern Vietnam and the increased prevalence in this region is attributed to the ingestion of raw or partly cooked fish (De et al., 2003). The major burden of disease from clonorchiasis is due to its exerting a WHO Group 1 carcinogen effect, causing cholangiocarcinoma. Rarely, *Cl. sinensis* has been reported to cause urticaria (Coskey, 1977). Recently, Choi et al, May 2011, have shown an association between Clonorchiasis and atopy, without this atopy being manifest as allergic disease. We report a case of urticaria, where the profile of the positive skin prick tests suggest the explanation for the findings of Choi et al., could well

be cross-reactivity, analogous to that seen due to tropomyosin, between house dust mite and shellfish (Fernandes et al. 2003).

**Case Report:** A 26-years-old male, who often ingested raw fish, and had no history of atopic diseases, presented to the Center of Allergology and Clinical Immunology<sup>1</sup> with chronic urticaria.

**Relevant Investigations:** Hepatic transaminases: markedly elevated. Total serum IgE=1004U/mL (NR< 100U/mL). Skin prick tests: strongly positive for house dust mites (*D pteryonyssinus* and *D farinae*), cockroach, *Blomia* spp., fish, prawn and crab. Liver ultrasound: focal hyperechogenicity. Stool culture: identified *Cl.sinensis* eggs with no other parasites present. *Cl. sinensis*-specific ELISA strongly positive at 1:3,200 dilution.

**Clinical Course:** Treatment with 25mg/Kg/day of praziquantel for three days resulted in remission of the urticaria, and over the ensuing two months, total serum IgE fell to 600 U/mL and transaminases returned to normal.

**Conclusion:** It is likely that in our patient, *Cl.sinensis* infestation both caused the urticaria and resulted in the atopy, which, given the skin prick test profile, suggests tropomyosin cross-reactivity as a mechanism. In addition, our patient demonstrates the importance of searching for *Cl.sinensis*, where urticaria and an elevated total serum IgE, occur in non-allergic individuals.

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## POSTER

### 47. ASPIRIN ALLERGY: THE FIRST SUCCESSFUL DESENSITIZATION TO ASPIRIN IN VIETNAM

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**Background:** The incidence of aspirin (ASA) hypersensitivity in the general Western population ranges from 0,6-2.5%, and in adults with asthma from 4.3-11% (Hedman 1999 and Szczeklik 2004). The prevalence of ASA hypersensitivity in the Asia-Pacific region is not precisely known. ASA hyper-sensitivity comprises five types, Types III (Cox-1 inhibitor mediated) and Types IV-V (IgE antibody mediated). Rapid ASA desensitization regimens have proven useful in patients with ASA/NSAID hypersensitivity who have co-existing coronary artery disease and require percutaneous coronary artery stenting (Page, 2007). We report a patient with probable IgE-mediated hypersensitivity to ASA, successfully desensitized to ASA.

**Case history:** A 43 years-old male, with asthma since childhood, had previously suffered episodes of angioedema following the ingestion of minor analgesics (precise constituents unknown). He was given clopidogrel 300mg and ASA 300mg prior to insertion of percutaneous cardiac stents and within 30 minutes had developed sneezing, periobital angioedema and pharyngeal discomfort, which settled after antihistamine was administered.

**Investigations:** His spirometry was normal. Skin prick testing with ASA 1mg/mL was positive. Total serum IgE was 106 U/L (NR < 100U/L). ENT examination and nasal/ sinus CT scans were normal.

**Clinical course:** ASA desensitization began with 0.0001mg of ASA. Initially, doses were doubled each 30 minutes, provided the peak expiratory flow (PEF) was not reduced. Breakthrough symptoms, which settled with fexofenadine 180mg, occurred 10 minutes after a single dose of 50mg and the protocol was modified, decreasing the rate of dosage increase. By day 10, the patient tolerated 100mg daily in a single dose and continues to do so.

**Conclusions:** Our report documents a safe and successful desensitization protocol in an individual with probable IgE-mediated hypersensitivity to ASA. This patient is the first patient desensitized to ASA in Vietnam. With thanks to the Hoc mai Foundation, University of Sydney, for making this possible.

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## POSTER

### 48. PRE CHALLENGE CLINIC– ARE WE PREPARING FAMILIES BETTER FOR FOOD CHALLENGE?

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A pre challenge clinic was established to assist in preparing families for the food challenge process. It is recognized that prior to a food challenge anxiety levels are high, especially in children with previous reactions to foods (Zijlstra et.al, 2010). Historically at the John Hunter Children's Hospital, the children were booked in for the food challenge in their outpatient consultation with their Paediatric Allergist. They then attended for the food challenge at the Paediatric Day Stay unit often over a year later. This delay appeared to have contributed to increased parental anxiety and a limited understanding of the food challenge process.

Once the pre challenge clinic process was established, a patient satisfaction survey was developed to ensure that the pre challenge clinic was advantageous. At the commencement of the food challenge a Patient Satisfaction Survey is provide to the parents to obtain feedback on the usefulness of the pre challenge clinic.

The pre challenge clinic consists of: a full medical review (by the Paediatric Registrar), a skin prick test, a review by the Paediatric allergy dietitian and a discussion regarding the process of challenge day and answer any questions that the child or family has about the challenge. Anxiety levels are generally high and the pre challenge clinic provides an opportunity for these concerns to be raised and addressed. It allows for families to make a more informed decision about proceeding with the food challenge.

Results indicated: 95% felt more prepared for the food challenge and had their concerns addressed. 97% indicated that they knew what to expect from the food challenge, 94% found the pre challenge clinic useful.

The pre challenge clinic has many benefits for all involved. Families are making a more informed decision and are more prepared. The team is able to play a more supportive role.

Zijlstra WT, Flinterman AE, Soeters L et al (2010), 'Parental anxiety before and after food challenges in children with suspected peanut and hazelnut allergy', *Paediatric Allergy Immunology*: 21; e439-e445

## POSTER

### 49. COMPARISON OF PEANUT COMPONENT AND WHOLE PEANUT SPECIFIC IGE WITH SKIN PRICK TEST FOR PREDICTING THE OUTCOME OF PEANUT CHALLENGE

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**Introduction:** Recent data suggests component-resolved diagnostic tests may be more accurate for the diagnosis of peanut allergy than SPT or sIgE to whole peanut extract. We sought to determine whether peanut component sIgE testing is superior to existing in vitro and in vivo diagnostic tests in predicting outcome of oral peanut challenge in a tertiary paediatric immunology service.

**Methods:** Children who underwent supervised peanut challenges at Princess Margaret Hospital and had serum stored within 12 months before the day of challenge were included in the study. Specific IgE to whole peanut extract and peanut components was measured using the ImmunoCAP assay. The diagnostic utility of each sIgE assay and SPT on day of challenge was assessed and compared using receiver operator characteristic (ROC) curve analysis.

**Results:** One hundred and ninety-three children were included in the study (69 who failed peanut challenge, and 124 who passed). Serum specific IgE to Ara h 1, Ara h 2 and whole peanut extract, and mean SPT wheal, were significantly higher in patients with peanut allergy than those who were peanut tolerant, however there was no significant difference between sIgE to Ara 3, 8 or 9. Area under ROC analysis identified sIgE to Ara h 2 as the best performing serological test assessed in this study, however mean SPT wheal on day of challenge had similar diagnostic accuracy to Ara h 2 sIgE. Sensitivity of Ara h 2 sIgE for diagnosis of peanut allergy at a cutoff of >0.35 kU/L was 69.6%, with specificity of 93.2%.

**Conclusion:** The additional benefit offered by peanut component sIgE, particularly Ara h 2, over existing diagnostic tests must be carefully evaluated before its introduction to routine practice. At present these assays are inadequate to consider replacing oral challenge as the gold standard diagnostic test for the majority of patients.



## POSTER

### 50. CO-SENSITISATION TO CASHEW AND ORANGE SEED

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**Introduction:** Co-sensitisation to botanically related foods has been widely described. Citrus fruit and cashew nut are related as members of the Sapindales order, but to our knowledge no data exists regarding co-sensitisation to these foods. We present the results of skin prick testing from 100 patients showing high rates of co-sensitisation to cashew and orange seed.

**Methods:** One hundred children with nut allergy or sensitisation were included in this study. Half of the children had positive SPT to cashew, and half had negative cashew SPT but were sensitised to another tree nut or peanut. All patients had SPT to a panel of food allergens, with histamine and saline controls. The Pearson correlation coefficient (r) was used to determine the strength of the relationship between mean SPT wheal diameters to each allergen.

**Results:** The strongest correlations found were between cashew and pistachio (r=0.85), pecan and walnut (r=0.77) and cashew and orange seed (r=0.73). There were statistically significant but weak correlations between orange seed and several other allergens, however there was no correlation between orange juice and orange seed SPT.

**Conclusion:** This study has demonstrated significant co-sensitisation to orange seed and cashew, however the prevalence of clinically relevant co-sensitisation has not been established and requires further research. Based on these findings, patients with cashew allergy may reasonably be counselled to avoid chewing citrus seeds and to be aware that seeds may be crushed during the preparation of freshly squeezed juice.

## POSTER

### 51. SKIN PRICK TEST PREDICTS CASHEW NUT CHALLENGE OUTCOME IN CHILDREN WITH LOW SPECIFIC IgE

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**Background:** Cashew nut allergy is common and carries a high risk of severe reactions including anaphylaxis. Although major advances have been made to guide clinical decision making regarding peanut allergy, to date limited data has been available to help clinicians predict the likely outcome of a cashew nut challenge.

**Methods:** A retrospective patient chart review of cashew nut challenges performed between 1 January 2005 and 31 December 2010 was carried out at Princess Margaret Hospital (PMH). Mean skin prick test (SPT) wheal diameter, cashew specific IgE (sIgE), age at challenge and previous clinical history were evaluated to determine whether any of these variables predicted the risk of a subsequent reaction during oral food challenge (OFC) to cashew. Statistical analysis was performed using the Mann-Whitney test and comparison of receiver operator characteristic (ROC) curves.

**Results:** Sixty-two OFC to cashew were performed. 36% (n=22) were positive challenges, 51% (n=32) were negative challenges, 6.4% (n=4) did not complete the challenge, 5% (n=3) of the challenges were cancelled due to large SPT size on the day of challenge and 1.6% (n=1) reported a delayed reaction. Only those children with an unequivocal challenge outcome (positive or negative) were included in the analysis.

Median Results	Positive challenges (n=22)	Negative challenge (n= 32)	Significance (p) (Mann-Whitney Test)
Age (years)	7.4	6.6	ns (p=0.23)
SPT (mm)	7.5	1	** p =<0.001
sIgE (kU/L)	<0.35	<0.35	ns (p=0.25)

\*\* Statistically significant.

**Conclusion:** In this study population there was no significant difference between median cashew sIgE for patients with positive and negative challenges, with most children from both groups having cashew sIgE <0.35 kU/L. Conversely median SPT was significantly higher in the positive challenge group and therefore found to be superior to sIgE in predicting outcome of challenge.

## POSTER

### 52. MANAGEMENT OF PERIPARTUM RISK OF EXERCISE-INDUCED ANAPHYLAXIS

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#### Case Report

**Background:** A 31 year old primiparous lady with a documented history of at least eight episodes of exercise-induced anaphylaxis (EIA) was referred by an Anaesthetist at 35 weeks gestation with concerns about the risk of anaphylaxis at or around labour.

**History:** The patient had a history of childhood allergic rhinitis and asthma which stabilized in adulthood. She described food allergies including oral allergy syndrome to apples, urticaria after

eating celery and abdominal cramps with mushrooms. There had been abdominal bloating and weight gain with ingestion of wheat for which she self-prescribed a gluten-free diet. Three presentations to the emergency department with anaphylaxis post meals have been recorded, each treated with adrenaline. All episodes were precipitated by minimal effort such as walking and usually began with gastrointestinal symptoms (nausea and vomiting or diarrhoea), progressing to periorbital and lip swelling, generalized pruritus with flushing, and followed by dyspnoea and dizziness. In addition, there have been 5 episodes of mild anaphylaxis after meals that were managed at home with antihistamines alone.

**Results:** Allergen skin testing showed positive results for bakers yeast, celery, walnut and apples. Apart from a mildly elevated total IgE level, blood tests were unremarkable with normal serum tryptase and specific-IgE to seafood, peanuts, omega-5-gliadin and mushroom.

**Discussion:** There is a single case report (Smith et al, 1985) of a patient who developed an episode of anaphylaxis from the effort of childbirth. Due to the unpredictability of the length of labour in a first pregnancy, there is considerable risk of an adverse outcome to the mother and/or foetus should anaphylaxis occur in this situation. Guidelines for managing such patients have not been established, but the empirical approach taken in this case, in light of only one reported previous case, is discussed.

**Reference:**

Smith HS, Hare MJ, Hoggarth CE, Assem ES. Delivery as a cause of exercise-induced anaphylactoid reaction: case report. *British Journal of Obstetrics and Gynaecology*. 1985 Nov;92(11):1196-8.

## POSTER

### 53. GUTS 'n' NUTS: THE ALLERGEN-EPITHELIAL RELATIONSHIP

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The food allergy epidemic has continued to rise over the past few decades, and has almost tripled in many westernized countries. Peanuts are the highest contributors of anaphylactic deaths after ingestion of a food allergen. The increasing occurrence of sensitivity to certain food remains to be identified, and, the allergen-epithelial relationship in particular remains elusive. Normally immune sampling of macromolecules and antigens in the intestine occurs via uptake by differentiated epithelial cells called M-cells, but little is known about the potential direct passage through normal epithelia. This ability to directly cross the

intestinal epithelium, unaided by M-cells, may increase immune cell exposure. This therefore, may possibly explain why peanut allergy is typically more severe and often continues through to adulthood. The aim of this project was to determine if peanut allergens are able to cross the gut epithelial barrier, using an in vitro model of the intestinal epithelium. We investigated the intestinal epithelial transport of peanut extract using the human Caco-2 cell culture model, exposed to varying concentrations of crude peanut extract. Western and immunofluorescence analysis were used to identify the cellular and molecular changes of peanut extract on the intestinal epithelium. The ability of peanut proteins to cross the intestinal epithelium, and their effect on trans-epithelial resistance, viability and tight junction modification will be presented. This work will provide novel insight into why peanut proteins are potent allergens. Consequently, permitting the development of innovative therapeutic and preventative measures for peanut allergy.

## POSTER

### 54. IMMUNOSUPPRESSION SCREEN: EVALUATION OF AN ONLINE IMMUNOSUPPRESSION RISK MANAGEMENT TOOL

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**Background/introduction:** We developed *Immunosuppression Screen* - a web-tool to rapidly assess common immunosuppression risks for individual patients, irrespective of diagnosis or the planned immunosuppressive regimen (Riminton et al 2011). We used the web-tool to complete a prospective audit of patient risk assessment within the Departments of Immunology and Neurology to help identify gaps in our practice.

**Methods:** The project was approved by the Human Research Ethics Committee of Concord Hospital. The web-tool was access at [www.immunosuppressionscreen.net.au](http://www.immunosuppressionscreen.net.au) Inpatients and outpatients qualified for inclusion in the audit by current use of an immunosuppression regimen.

**Results:** Immunosuppressed patients in Immunology and Neurology have diverse diagnosis and treatment regimens. *Immunosuppression Screen* filtered and organised information into six risk categories and two patient tailored lists of value in the ongoing care of the patient. As an audit tool, it identified unique problem lists and highlighted numerous tasks to be completed otherwise overlooked in routine care. Common tasks requiring

implementation included vaccination status and education, VZV, HIV and Hepatitis serologies, The tool was easy to use with minimal training. The study identified some data export issues with the web tool which has since been rectified.

**Conclusion:** *Immunosuppression Screen* was readily applied as an audit tool in busy clinics and has helped develop practice improvements within both departments.

**Reference:**

D. Sean Riminton, Hans-Peter Hartung, Stephen Reddell  
Managing the risks of immunosuppression — Current Opinion in Neurology 2011 24:217-223,

**POSTER**

**55. PREVALENCE OF TOXOPLASMOSIS IN DIALYSIS PATIENTS AND RELATIONSHIP WITH IMMUNE SYSTEM FUNCTION**

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**Introduction:** Chronic kidney disease (CKD) is a devastating disease with many systemic disturbances which is followed with progressive and irreversible reduction in kidney tissue. Currently about 1 million people suffering from kidney disease in the final ESRD stage and under dialysis condition exists in the world. Disorders related to immune system especially in uremia patients cause to high distribution of many infectious micro organisms in these patients. *Toxoplasma* is an intra cellular parasite which infects people with weakened immune system.

**Method:** This study was established in a cross-sectional pattern on 180 serum samples including 90 of hemodialysis people and 90 of healthy people. Serum samples were evaluated for IgG and IgM against *toxoplasma gondii* using ELISA method.

**Results:** IgG and IgM antibodies against *toxoplasma gondii* in hemodialysis patients were 55 (60/43%), 3(3.3%) and in control patients were 36(38%), 0(0%). Results showed that the difference between toxoplasmosis prevalence in hemodialysis patients was statistically significant in comparison to control group (P<0.05).

**Discussion:** Activation of immune system against the invasion of toxoplasma parasite lead to increase of IFN- and eventually the production of nitrite oxide followed with removal of toxoplasmosis tachyzoites. In hemodialysis patients, uremia induces the increase of Th<sub>2</sub> responses rather than Th<sub>1</sub> responses. Reduction in IFN- production weakens the cellular immune system in functioning against intra cellular infections like *toxoplasma gondii*. According to what discussed above, drug prophylaxis and interleukin therapy considering the high prevalence in patients, strongly recommend.

**POSTER**

**56. DOES VITAMIN D INTAKE DURING PREGNANCY INFLUENCE ALLERGIC OUTCOMES IN CHILDREN AT ONE YEAR OF AGE?**

**Kristina Rueter**<sup>1,2</sup>, Anderson P. Jones<sup>1</sup>, Debra Palmer<sup>1,2</sup>, Meri K. Tulic<sup>1</sup>, Susan L. Prescott<sup>1,2</sup>

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**Objectives:** Based on the immunological effects and epidemiological associations, rising rates of Vitamin D insufficiency have been implicated in the dramatic increase of allergic disease, raising questions over the need for routine vitamin D supplementation in pregnancy. Our study aim was to investigate maternal Vitamin D intake from supplements and diet in pregnancy in relation to infant allergic disease.

**Methods:** Vitamin D intake from multivitamins, supplements and dietary sources was determined in 670 women using validated semi-quantitative food frequency questionnaires in the third trimester of pregnancy. Infant allergic outcomes at one year of age (n= 484) were determined by allergy skin prick testing and diagnosis of eczema, food allergy or asthma.

**Results:** Total maternal Vitamin D intake in pregnancy was higher in infants who subsequently developed any allergic disease (p=.022) in particular those who developed eczema (p=.01). Vitamin D intake above the Recommended Daily Intake (RDI= 5 µg/day) was associated with a significant increased risk of eczema (p=.006), and any allergic disease (p=.003). This effect was largely due to the amount of vitamin D derived from supplements.

**Conclusion:** Interestingly these results do not support the hypothesis that boosting vitamin D intake during pregnancy with supplements protects against infant allergic disease. Rather, maternal vitamin D intake above the RDI appears to increase the risk of allergic disease in particular eczema at one year of age. Further studies are under way to assess the role of vitamin D on immunomodulation of early immune function.

**POSTER**

**57. THE PREVALENCE OF TRUE PENICILLIN ALLERGY FROM A STUDY OF CASES AT CAMPBELLTOWN HOSPITAL IMMUNOLOGY CLINIC**

**Pryanka S Sagar**<sup>1</sup>, Constance H Katelaris<sup>2</sup>

<sup>1</sup> Medical Student University of Western Sydney

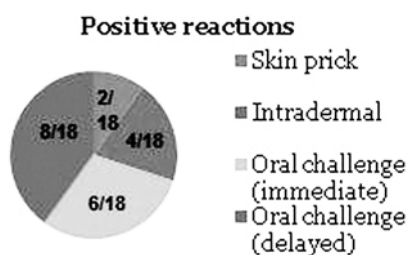
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**Background:** Current statistics show that 10% of patients claim to be allergic to penicillin yet only 10% of these have demonstrable allergy. The most appropriate, cost-effective antibiotics are sometimes withheld on the basis of patient history. This study aims to investigate the demonstration of IgE hypersensitivity in patients presenting with a history of penicillin allergy to a teaching hospital allergy clinic.

**Methods:** Patients underwent skin prick and intradermal tests with major and minor penicillin determinants. Those with negative skin tests were administered a 3-day oral challenge. Demographic and clinical details about the reactions were noted.

**Results:** 136 patients were tested and 18 had positive skin tests +/- oral challenge results. This corresponds to 13% of patients with self-reported allergy having true allergy. Analysis of clinical histories showed that patients with a well-defined history of allergy and a history of anaphylaxis were more likely to have a positive test compared to patients with vague histories. Skin testing proved to be less sensitive than oral challenge (Chart 1).



**Conclusions:** A minority of patients presenting with a history of penicillin allergy have evidence of IgE mediated hypersensitivity (18/136, 13%) patients in this study. Our study demonstrates the usefulness and discriminating power of objective testing which must include oral challenge.

Discriminating factors of true allergy within the patient history were the presence of a clear description of reaction and a history of anaphylaxis

Negative testing enables the use of penicillin as first-line treatment and this can significantly reduce the cost of antibiotics.

*(The authors acknowledge Prof B Frankum, Dr K Keat, Dr S Gala, Mrs P Burton, Mrs F Perram as contributors to this study)*

## POSTER

### 58. THE EXPERIENCE OF ASSESSING BETA-LACTAM ANTIBIOTIC ALLERGY WITH THE DIATER PENICILLIN AND AMOXYCILLIN REAGENTS IN A TERTIARY REFERRAL CENTRE

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**Introduction:** Allergic reactions to Beta-lactam antibiotics are the most common cause of adverse drug reactions however many patients who describe a history of penicillin allergy do not prove to have true Immunoglobulin E (Ig E) mediated allergy. Exclusion of penicillin allergy is challenging. Skin testing to major and minor determinants of penicillin has the potential to improve diagnosis. The Diater reagent kit is the only commercially available kit now utilized for the diagnosis of beta lactam allergy. We report our experience with the use of the Diater reagent kit with major and minor determinants of penicillin.

**Methods:** Patients referred to a tertiary hospital allergy clinic for assessment of reported allergy to beta-lactam antibiotics were included in the study. Drug hypersensitivity reactions were assessed by a standardised questionnaire completed by patients. Patients with a history or examination consistent with an immune mediated reaction to beta-lactam antibiotics with a recorded serum specific Ig E to penicillin proceeded to skin prick (SPT) and intradermal tests (IDT) based on the manufacturer's suggested protocol. Patients with negative SPT and IDT proceeded to oral challenge.

**Results:** 65 patients underwent testing for penicillin allergy. The median age was 55 years (range 3-84 years), 28 were men. Positive SPT and IDT were seen in 7 patients; 4 of which were to amoxicillin, 3 to cephalosporins. 1 patient had a positive oral challenge in the presence of negative skin tests. In patients with cephalosporin allergy none had cross-reactivity to penicillin on skin testing. No adverse reactions occurred during testing.

**Conclusion:** Our experience with the Diater reagent kit shows it to be a useful and safe mode for diagnosis of beta-lactam allergy. However, oral challenge should still be performed in those with negative skin test results. We did not find any cross- reactions between cephalosporins and penicillin in this group.

## POSTER

### 59. GIANT CELL ARTERITIS PRESENTING AS CARDIAC TAMPONADE

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**Introduction:** There has been increasing recognition of aortic involvement in giant cell arteritis. We provide the first report of a patient who presented with pericardial tamponade as a complication of giant cell arteritis.

**Case report:** A 67-year-old Caucasian female presented with a 3 day history of worsening of dyspnoea. This was on a background of lethargy and dyspnoea for 3 months. A myocardial perfusion scan performed 6 weeks before presentation was unremarkable. Examination revealed no fever, elevated JVP with Kussmaul sign, hypotension, decreased heart sounds and pulsus paradoxus. Computerised tomography showed Type A aortic dissection with evidence of pericardial effusion. Echocardiography confirmed pericardial tamponade with aortic regurgitation. The patient's dyspnoea improved rapidly after pericardiocentesis which yielded 770 mls of sterile, haemorrhagic pericardial fluid. CRP was markedly elevated. Autoimmune markers and syphilis screen were negative. Aortic root and valve replacement surgery was undertaken. Histology of the aorta showed inflammatory infiltrate with giant cell formation. The patient recovered well, after the surgery and an interval PET scan was normal.

**Discussion:** Giant cell arteritis is a systemic vasculitic syndrome affecting medium and large arteries. PET scans suggest aortic involvement in up to 50% of cases. Aortic dissection complicating giant cell arteritis is rare and usually fatal. This case reports the first known presentation of GCA with localized proximal aortic aneurysm and pericardial effusion, successfully treated with surgery.

**Conclusion:** Giant cell arteritis should be considered in the differential diagnosis of patients presenting with pericardial tamponade secondary to aortic dissection and a thorough histopathological examination, as conducted in this case, is essential.

## POSTER

### 60. AEROALLERGEN IMMUNOTHERAPY REDUCES DAYTIME SOMNOLENCE

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**Introduction:** Sleep disruption and daytime somnolence commonly accompany allergic rhinitis (AR)-associated nasal congestion. Intranasal corticosteroids and montelukast improve nasal and sleep related symptoms. Aeroallergen immunotherapy (IMT) improves AR symptoms, but any subsequent effect on sleepiness is unknown. This prospective study determined the effect of aeroallergen IMT on daytime somnolence.

**Methods:** 18 adult patients attending the hospital for aeroallergen IMT completed the Epworth Sleepiness Scale (ESS) before starting IMT and on achieving maintenance doses. Body-mass index (BMI) was determined from weight and height measurements.

**Results:** The mean BMI of the cohort was 28. The mean ESS pre-immunotherapy was 5.9 compared to an ESS of 4.8 post-immunotherapy. Obese patients (BMI>25) had mean pre-immunotherapy ESS of 6 compared to post-immunotherapy ESS of 4.5. In patients with moderate-severe obesity (BMI>30), the mean ESS pre-immunotherapy was 8.25 and 5.75 post-immunotherapy. In the subgroup of patients with severe obesity (BMI>35), the average ESS pre-immunotherapy was 10 compared to post-immunotherapy ESS of 7.5.

BMI	Average improvement in ESS post-immunotherapy
More than 25	1.5
More than 30	2.5
More than 35	2.5

**Conclusions:** Immunotherapy for allergic rhinitis in adult patients reduced daytime somnolence scores. These preliminary results showed a greater benefit for obese patients. Since immunotherapy is a 3 year treatment, this cohort requires ongoing assessment. The use of other ambulatory tools to assess sleep quality, somnolence and upper airway patency may add to our understanding of a persisting benefit. Longer term observation is required to determine whether immunotherapy improves nocturnal sleep, and can improve other unrelated patient outcomes such as cardiovascular events, similar to OSA.

## POSTER

### 61. GENETICS OF FOOD ALLERGY

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**Introduction:** There is an increasing body of evidence of several gene deletion syndromes and association of multiple genetic polymorphisms with food sensitisation, allergy and anaphylaxis.

**Methods:** Literature review of food sensitization, food allergy and/or anaphylaxis and the association with genetic syndromes, polymorphisms or specific genotypes. These associations have been arranged into a structure a plausible mechanistic inter-relationships

**Discussion:** The patterns of genetic dysfunction appear to be able to be subdivided into those involved with epithelial integrity, anti-proteases, innate and adaptive immunity as well as genes affecting immune regulatory pathways. There is clearly interplay between these components of dysfunction. The complexity of gene interactions and the contribution of multiple genes have made it difficult to interpret the pattern of dysfunction that might cause an allergic response to foods.

**Conclusions:** Food allergy susceptibility appears to be attributed to multiple genetic mechanisms. The genetic basis of food allergy does not explain the overwhelming increase in food allergy in the last 2 decades. Understanding the mechanisms that causes food allergy is critical to prevention and understanding the causes as well as environmental and immunological intervention strategies.

## POSTER

### 62. TREATING MOLECULAR PAIN IN ALLERGY

Danielle Litchfield<sup>1</sup>, Lisa Pinel<sup>1</sup>, Bianca Maiden<sup>1</sup>, Pete Smith<sup>1,2</sup>

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**Introduction:** Sensory and neural mechanisms contribute significantly to disease morbidity in all allergic diseases. Histamine (and other acute allergic mediators) exerts cellular effects on sensory nerves via activation of the TRPV1 receptor.

**Methods:** Healthy adult volunteers were pretreated with a photochemical mixture\* of TRPA1 and TRPV1 antagonists and agonists for the TRPM4, M8 receptors in s DBPRC manner, prior to application topical skin prick test to histamine dihydrochloride (Hollister-Stier, Spokane WA, USA) 10 mg/ml on the volar forearm. Patients were scored for itch, pain and wheal size

**Results:** Compared to placebo, the multi-TRP modification compound significantly reduced pain and itch scores immediately at 8 minutes and 15 minutes. N=9. Scores are a 10 point scale. Analysis via paired T Test. \* p<0.05,  $\Psi$  P<0.01

	Immediate		8 Minutes		15 minutes	
	Active	Placebo	Active	Placebo	Active	Placebo
PAIN	1.25*	2.125	1.25*	2.125 $\Psi$	0.625	1.375
ITCH	1.375*	2.25	1.875*	3.5 $\Psi$	0.625	2

**Conclusions:** Transient receptor potential modification represents an rapid and effective therapeutic target for treatment of sensory allergic mechanisms. TRP receptors are also present on mast cells, mucous glands and epithelial surfaces and modification could have impact in inflammatory processes.

*Declarations.* PS is a co-owner of a patent on this mixture and as such, the first 3 authors performed patient recruitment, consenting investigation and data recording independently.

## POSTER

### 63. PREDICTING EOSINOPHILIC CHRONIC RHINOSINUSITIS: RELATIONSHIP OF PHENOTYPE TO SERUM AND TISSUE BIOMARKERS

Kornkiat Snidvongs<sup>1</sup>, Mathew Lam<sup>2</sup>, Raymond Sacks<sup>1</sup>, George Marcellis<sup>3</sup>, Larry Kalish<sup>4</sup>, Seamus Phillips<sup>5</sup>, Richard Harvey<sup>1</sup>

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<sup>2</sup> University of New South Wales

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**Introduction:** Common features of eosinophilic chronic rhinosinusitis (ECRS) include asthma, polyps, aspirin sensitivity, high serum eosinophilia and IgE. However, when no traditional features of ECRS are present, the most significant finding may be the presence of tissue eosinophilia. The associations between histopathology and serology of ECRS have never been investigated.

**Methods:** A cross-sectional study was undertaken on patients with chronic rhinosinusitis with and without polyps undergoing surgery. Correlations between ECRS phenotype and serum and tissue biomarkers were analyzed.

**Results:** A total of 51 patients with histopathology and serum markers were assessed (47% female, age 46.6±4.1 yrs). Tissue eosinophilia (>10/HPF) significantly associated with the presence of polyps ( $\chi^2=25.76, p<0.01$ ), endoscopic scores ( $r=0.54, p<0.01$ ), computed tomography scores ( $r=0.57, p<0.01$ ) and serum eosinophilia ( $r=0.33, p=0.03$ ). Asthma status did not associate with eosinophilia ( $p=0.60$ ). Serum IgE was non-predictive ( $p=0.08$ ). The cut-off point of serum eosinophil percentage of >4.4% predicted ECRS (sensitivity 52%, specificity 87%, positive predictive value 79%, ROC  $p=0.001$ ).

**Conclusion:** Tissue eosinophilia is not associated with all of the common features of ECRS. It is associated with clinical severity and prognosis of ECRS.

## POSTER

### 64. PREDICTING SUCCESSFUL SUBCUTANEOUS IMMUNOTHERAPY IN CHILDREN: SERIAL EXHALED NITRIC OXIDE OR COMBINED SERIAL AEROALLERGEN SKIN PRICK TEST RESPONSES?

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**Background:** Subcutaneous immunotherapy (IT) is widely used in children with difficult asthma and rhinitis, and shown to reduce development of new allergic disease. Continuing or stopping a course of IT is judged on clinical response. A better predictor of which children might respond prior to the full 2-3 years of therapy would be valuable. None of the standard immunological measures (specific IgE, Skin Prick Tests (SPT), specific IgG4 etc.) reliably predicts successful IT. Serial exhaled nitric-oxide (eNO) levels have not been studied in IT. Sum specific IgE has been shown to reflect asthma severity, but serial sum SPT has not been reported in IT.

**Methods:** All children who had undergone IT for asthma or rhinitis in the Wellington (NZ) region since 2004 and completed or discontinued IT were reviewed. Those who had eNO (NIOX MINO) and SPT measured prior to starting and at the end of IT were selected. We collected demographics, IT history and outcomes. eNO and sum SPT diameters were measured at ~6 monthly intervals.

**Results:** Of 37 children, 20 met the entry criteria, 7-16 (median 10) yr., 14 males, 15 European, 3 European/Polynesian, 1 Filipino, 1 Chinese/European. 17 had rhinitis, 16 asthma and 12 eczema. IT vs. house dust mite 17, grass pollen 14, cat 8, tree pollen 3, dog 1 and horse 1. IT was continued for 1-5 years (median 3). At completion 14 derived clear benefit, 4 possible and 2 no benefit. eNO varied from 10-201 (median 54) ppb prior, and from 15-111 (median 45) ppb at the end of treatment.

There was no significant change in eNO.

Sum SPT diameters significantly dropped in responders (15-45 (median 31)) pre-therapy, 5-32 (median 15)) post-therapy but not in the other groups. (p<0.005)

**Conclusion:** Sum SPT but not eNO appears to be of value in assessing response to IT.

## POSTER

### 65. AN OPEN-LABEL STUDY OF SUBCUTANEOUS EVOGAM IN PATIENTS WITH PRIMARY IMMUNODEFICIENCY (PID): INTERIM ANALYSIS

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**Introduction:** This open-label study was a continuation of the registration study for a new subcutaneous immunoglobulin (SCIG), Evogam, with allowance for new patient recruitment. It was designed to evaluate the safety and tolerability of long term use in PID. We present here an interim analysis of this ongoing study.

**Methods:** Patients were enrolled either from the Phase III efficacy study or were newly recruited from routine care, IVIG or alternative SCIG therapy. Evogam was administered by the patient or guardian weekly or twice weekly. Primary end points were rate, severity and relatedness of reported adverse events (AEs), with IgG trough levels a secondary endpoint. Details of infusions and AEs were diarised, in addition to being elicited during 3 monthly reviews.

**Results:** Thirty adults and seven children were enrolled between April 2008 and April 2010. Twenty-seven patients (73%) were enrolled from the registration study and ten (27%) were transferred from IVIG (n=9) or other SCIG therapy (n=1). Six patients withdrew from the study with 31 patients still enrolled at the time of interim analysis. The majority of patients (26/32, 81%) experienced mild to moderate infusion site reactions. Adverse events were reported in 30/37 (81%) patients with six (16%) considered related to therapy. Serious adverse events occurred in eight adults and two children, seven of which were infection-related. Exacerbation of respiratory tract infection in one was felt to be related to the study drug, resulting in discontinuation. Median serum IgG trough levels remained in the therapeutic range, (>baseline 8.9g/L).

**Conclusion:** Evogam was well tolerated in adult and paediatric patients and maintained stable IgG trough levels. Local infusion site reactions were common but not severe. Most other AEs were consistent with the underlying diagnosis of PID. No new safety concerns were identified.

## POSTER

### 66. BRONCHIECTASIS: A NOVEL AUTOIMMUNE DISEASE?

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**Introduction:** Non cystic fibrosis (CF) is a suppurative lung disease characterised by chronic sputum production, airway colonisation and recurrent infective exacerbations. The aetiology of non-CF bronchiectasis is difficult to identify and the majority remain idiopathic, although humoral immunodeficiency is frequently recognised. Connective tissue disease is a less common disease association with bronchiectasis where the aetio-pathological mechanism remains unclear.

**Methods:** We sought to evaluate the expression of common autoantibodies in non-CF subjects presenting to the Alfred Hospital, Melbourne between 2007-2011. Subjects had CT confirmed bronchiectasis without evidence of CF. Autoantibody assays including ANA, ENA, RF and ANCA were measured in all bronchiectasis patients.

**Results:** n = 101, f = 66, age 59.4 (14.7) years, FEV1 78.1 (27.6), BMI 26(7.5), 38 ex smokers (mean SD 23.5 (19.2) pack years). 29 subjects were colonised with pseudomonas, 33 with haemophilus. ANA was positive in 30 subjects and ENA negative in all subjects. cANCA was identified in 16 subjects (8 weak positive, 8 positive) and pANCA positive in 1 subject. RF was found in a titre > 10 in 29 subjects. Pseudomonas colonisation was associated with cANCA expression (chi2 p<0.05).

**Conclusion:** Non-CF bronchiectasis is frequently described as idiopathic yet autoantibody expression is well recognized in this condition. The identification of these antibodies raises questions about autoantibodies as pathogenic contributors to airway damage in bronchiectasis. The presence of autoantibodies in the setting of conditions characterised by chronic colonisation and infection is of uncertain significance and potential concerns in interpretation of these results is discussed.

## POSTER

### 67. VALIDATION OF A MULTIPLEX ASSAY FOR QUANTITATION OF ANTIBODY RESPONSES TO PNEUMOCOCCAL VACCINES

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**Introduction:** The assessment of serotype-specific antibody responses to pneumococcal polysaccharide vaccine is used routinely in the evaluation of humoral immune function and diagnosis of specific antibody deficiency. Multiplex technologies have the potential to significantly improve the efficiency of measuring these antibodies by allowing simultaneous quantitation of serotype-specific IgG. However there has been limited validation of multiplex assays against the gold-standard WHO ELISA and more importantly, there has been no assessment of assay performance in the clinical setting where evaluation of humoral immune function involves an assessment of fold rise in titre of pre and post pneumococcal immunization.

**Methods:** We compared measurement of serotype-specific anti-pneumococcal IgG to 14 serotypes in Pneumovax® (23vPPV) using xMAP® Pneumo 14 multiplex (Luminex Inc, Austin, Texas, USA) with the 'gold standard' WHO ELISA method. Serum from children and adults taken pre- and post- PCV7 and post-23vPPV immunization were analyzed.

An adequate response to immunization was determined using the APIIEG (2009) and AAAAI expert guidelines: post titre > 1.3 ug/ml and/or post titre 4X baseline.

**Results:** The xMAP multiplex correlated poorly with the ELISA. The GMC for the majority of serotypes was higher when samples were assayed by xMAP as compared to ELISA. In post PCV7 immunization samples, high titres of serotype-specific IgG to serotypes not contained in PCV7 were detected by xMAP but not ELISA. Using the guidelines for an adequate response to pneumococcal immunization, seven of 25 (28%) infants were identified as having an inadequate response by ELISA and an adequate response by Luminex. There was agreement between the methods for 21 of 26 (81%) adult and 18 of 25 (72%) infant pairs.

**Conclusion:** Using APIIEG (2009) and AAAAI guidelines, the xMAP assay does not provide reliable evaluation of functional antibody responses to polysaccharide antigens for the assessment of immune competence.



## POSTER

### 68. PRENATAL MATERNAL STRESS AND INFANT ALLERGY: AN EXPLORATORY STUDY

Jody Tansy<sup>1,2</sup>, Meri Tulic<sup>1</sup>, Nina D'Vaz<sup>1</sup>, Brad Zhang<sup>1</sup>, Susan Prescott<sup>1,3</sup>

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**Background:** Maternal stress in pregnancy is associated with an elevated cord-blood IgE as well as a higher cord-blood lymphoproliferative and stimulated cytokine response to allergens. It has been hypothesised that prenatal stress affects fetal growth and development via activation of the physiological stress response or via effects on the normal Th2-predominant immune response in pregnancy.

**Method:** In our cohort of 230 maternal-infant pairs the Depression, Anxiety, Stress Scale (DASS) and Perceived Stress Scale (PSS) were collected at 37 weeks gestation. At 6 months of age T-cell responses to house dust mite antigen (HDM) and egg ovalbumin antigen (OVA) were assessed in a subset (n=42). At 12 months of age data on infant allergic disease was collected and skin prick testing (SPT) performed.

**Results:** Ten percent of our cohort was stressed and 5% were depressed. At 1 year the prevalence of allergic disease in the infants was 40.5%, and 22.5% had a positive SPT. At 6 months there was an inverse relationship between prenatal maternal stress and stimulated T-cell IL-13 production to HDM and OVA that approached statistical significance (DASS stress and HDM stimulated IL-13 production:  $R = -0.42$ ,  $p = 0.06$ ; DASS stress and OVA IL-13:  $R = -0.31$ ,  $p = 0.05$ ; PSS and OVA IL-13:  $R = -0.27$ ,  $p = 0.09$ ). There was no significant association between prenatal maternal DASS or PSS score and the prevalence of infant allergic disease or positive SPT at 1 year.

**Conclusion:** In our cohort we did not demonstrate a significant association between maternal DASS scores or PSS scores at 37 weeks gestation and infant allergic disease or positive SPT at 12 months of age. However, there was a trend towards significance between stress scores and stimulated 6 month T-cell Th2 cytokine production to allergens supporting the hypothesis that prenatal maternal stress alters the normal Th2 predominance in pregnancy and infancy.

## POSTER

### 69. TYPE I INTERFERONS AND PLASMACYTOID DENDRITIC CELLS SELECTIVELY CONSTRAIN TH2 CYTOKINE RESPONSES TO RHINOVIRUSES

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**Background:** Human rhinoviruses (RV) are frequent triggers for asthma exacerbations. An increasing body of evidence points to a dysregulated anti-viral immune response in asthma with reduced synthesis of innate interferons and relative Th2 skewing of the adaptive immune response. The mechanisms underlying these observations are not well understood.

**Aim:** Examine the regulation of adaptive immune responses to RV in healthy people.

**Methods:** Blood mononuclear cells from 27 healthy subjects were cultured with RV16. In some experiments, plasmacytoid DC (pDC) were depleted with immune-magnetic beads, while IFN alpha/beta was neutralized using B18R, a decoy receptor that blocks IFN signaling.

**Results:** RV stimulated PBMC secreted large amounts of IFN alpha/beta in the first 24 hours which was largely dependent on pDC. At later time points the adaptive immune response was dominated by IFN-gamma secretion by CD45RO+ antigen experienced memory T-cells, and only modest secretion of Th2 cytokines. Either depletion of pDC or neutralization of IFN alpha/beta led to a significant increase in expression and secretion of IL-5, IL-9 and IL-13, but had no effect on IFN-gamma secretion.

**Conclusion:** In healthy individuals, pDC and the type I IFNs they secrete selectively constrain Th2 cytokine synthesis following RV stimulation in vitro. These important regulatory mechanisms may be lost in allergic diseases such as asthma thereby worsening the inflammatory response to viral infection.

## POSTER

### 70. CLINICAL UTILITY OF ALLERGEN-SPECIFIC IgE MEASUREMENT IN THE DIAGNOSIS OF MAMMALIAN MEAT-INDUCED ANAPHYLAXIS ASSOCIATED WITH PRIOR TICK BITES

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**Background:** In 2007, van Nunen et al. first described the association between red meat-induced anaphylaxis and prior tick bites (van Nunen et al 2007). Given the distribution of *Ixodes holocyclus* (along the entire Eastern seaboard of Australia) and that ticks occur overall in a wide distribution within Australia, this complaint is likely to prove more common in the future, particularly considering predicted climate change effects on hosts' habitat. Accordingly, we have examined whether commercially available mammalian meat RAST results correlate with a clearcut clinical diagnosis.

**Methods:** The mammalian meat RAST results were reviewed in twenty patients whose clinical histories of mammalian meat anaphylaxis associated with tick bites satisfied a strict algorithm for diagnosis- prior large local reactions to tick bites, ingestion of mammalian meat 5-7 hours pre-anaphylaxis and total remission following strict mammalian meat exclusion.

**Results:** In this selected patient group, all individuals had positive mammalian meat RASTs (20/20).

**Conclusions:** A strong correlation appears likely then between positivity of the mammalian meat RASTs and typical mammalian meat anaphylaxis associated with prior tick bites, when the patient's history satisfies a strict clinical algorithm for the diagnosis of this condition. The clinical utility of this observation lies in the ability of mammalian meat RAST results to provide confirmatory evidence for the diagnosis in sufferers in a timely manner, with testing easily available to primary medical practitioners, which may thereby reduce the morbidity associated with delayed diagnoses of this cause of anaphylaxis, a diagnosis with unique clinical features lending itself to such an approach.

#### Reference:

van Nunen et al. The Association Between *Ixodes holocyclus* Tick Bite Reactions and Red Meat Allergy. Internal Medicine Journal; 39 (Suppl 5): A132, 2007.

## POSTER

### 71. INVESTIGATING PENICILLIN ALLERGY: THE AUCKLAND CITY HOSPITAL EXPERIENCE

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**Introduction:** Our previous audit (2007/8) confirmed PPL and MDM reagents as valuable in penicillin skin testing, but identified concerns of systemic reactions to skin testing and low completion rate of oral challenge after negative skin tests. New protocols were initiated in April 2009 and their use and effectiveness have been retrospectively audited here.

**Methods:** The records of all adult immunology patients undergoing penicillin skin tests at Auckland Hospital from April 2009 to February 2011 were examined and relevant details of history, test and challenge results recorded. Skin tests with benzylpenicillin, major(PPL) and minor(MDM) determinants, amoxicillin, Augmentin and/or flucloxacillin were undertaken using either a three or five-step protocol, dependant on physician-assessed risk of systemic reaction. If skin tests were negative an appointment was immediately made for subsequent oral challenge.

**Results:** The mean age of the 178 patients was 45y (15-86y), 72% female. Immediate positive skin tests (STpos) occurred in 21(12%); 3 to MDM and/or PPL only; 3 to amoxicillin/Augmentin only, with 8 positive only to the highest flucloxacillin concentration. Skin tests were positive in 36% (95% CI 17.2-54.8) of those with a history of anaphylaxis compared with 7.8% (3.6-12.1) of those without. Difference in ST positivity was not significant with respect to reaction history of <1y (17.0%; 7.4-26.5) or >1y (9.2%; 4.0-14.5). The 5-step protocol had been chosen for 38% of STpos and 23% of STneg patients. No patient experienced anaphylaxis related to skin testing; two had mild reactions. Ten of 100 (10%) skin test negative (STneg) patients gave positive responses to incremental oral challenge.

**Conclusion:** Immediate positive skin tests occurred in 12% of referred patients; with challenge further identifying 10% of the STneg patients as reactive. Challenge is an important part of the investigation process. It remains difficult to assess risk and allergy status based on clinical history alone.

## POSTER

### 72. PERCEPTIONS OF PAEDIATRIC ANAESTHETISTS ON THE USE OF PROPOFOL IN EGG/SOY/PEANUT ALLERGY

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**Introduction:** Propofol contains egg lethicin and soy oil. Hypersensitivity to egg, soy, and peanut are listed as contraindications for propofol use. However guidelines and recent evidence suggest propofol is generally safe in most sensitized patients. This study aimed to evaluate the practices of paediatric anaesthetists with respect to propofol use in egg/soy/peanut allergy children.

**Methods:** An online survey was sent to members of the Association of Paediatric Anaesthetists of Great Britain and Ireland and Society for Paediatric Anaesthesia in New Zealand and Australia. Membership to these societies is not restricted to the anaesthetist's country of practice.

**Results:** 349 responses were collated over 3 months. Most paediatric anaesthetists were consultants (88%), had > 10 years' experience (80%), and practiced in England (59%), Australia (17%), Scotland (5%), New Zealand (4%), Ireland (3%) and Wales (2%). The 9.5% anaesthetists working in "other" countries were practicing in 17 different countries.

55%, 69% and 72% of anaesthetists reported they would not use propofol in patients with a history of urticaria, wheeze or hypotension to egg/soy respectively. Most would administer propofol in children with only peanut allergy (91%).

Trainees and anaesthetists with < 10 years' experience were more likely to request a skin prick /intradermal test to propofol in patients with a history of urticaria to egg/soy prior to administration ( $p < 0.05$ ).

Australian/New Zealand anaesthetists were more likely to administer propofol in patients with a history of urticaria to egg/soy ( $p=0.0031$  vs other countries). Anaesthetists who stated they would use propofol in those with prior urticarial reactions to egg/soy, were also more likely to use propofol in patients with a history of wheeze or hypotension to egg/soy ( $p < 0.05$ ).

**Conclusion:** Contrary to published guidelines most paediatric anaesthetists would not administer propofol to patients with a history of mild-moderate egg/soy allergy. Approximately 1/10th of anaesthetists would not administer propofol to children with peanut allergy alone.

## POSTER

### 73. A COMPARISON OF SINGLE-HEADED SKIN TEST DEVICES: VARIABILITY OF RESULTS WHEN PERFORMED BY MULTIPLE OPERATORS

Rebecca L Werther<sup>1</sup>, Sharon Choo<sup>1</sup>, Katherine J Lee<sup>2,3</sup>, Katrina J Allen<sup>1,2,3</sup>, Debra Poole<sup>1</sup>, Mimi Tang<sup>1,2,3</sup>

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**Background:** Previous studies have compared the intra-user variability between skin prick test (SPT) devices used to aid in the diagnosis of IgE-mediated allergy, however no study to date has assessed the variability in performance of devices when used by different operators. We assessed the variability of four commonly used SPT devices when used by multiple operators, and when performed on arms compared to backs.

**Methods:** Eight adult volunteer 'testers' with no prior experience in performing SPT were trained equally in the use of four devices: Greer Pick, Quintip, Stallergenes Lancet and Feather Lancet. Each tester performed SPT with histamine (1 mg/ml) using each of the devices on the backs and forearms of 5 volunteer 'receivers'. Wheal sizes were measured at 10 minutes, with variability in the mean perpendicular diameters assessed using a multi-level (random effects) regression model.

**Results:** From the raw data, the SPT measurements for Greer Pick were the most variable (standard deviation [SD] = 2.1), followed by the Feather Lancet (SD = 1.3) and the Quintip (SD = 1.1), with the least variability observed with Stallergenes Lancet (SD = 0.9). Using multi-level modelling, the measurement error was the smallest with the Stallergenes Lancet, closely followed by the Quintip and the Feather Lancet, and then the Greer Pick. There was no evidence that the variability between testers or receivers was different across the devices. Comparison of SPT performance by location revealed greater variability in measurements from the arm compared to the back.

**Conclusion:** When multiple operators were equally trained in all four SPT devices, the devices using the 'puncture' method (i.e. Stallergenes Lancet and Quintip) provided less variability in SPT results than those using a 'prick' method (i.e. Greer Pick and Feather Lancet). There was overall less variability when SPT was performed on the back compared to the arm.

## POSTER

### 74. CHARACTERISTICS OF CHURG-STRAUSS SYNDROME IN SOUTH AUSTRALIA

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**Background:** Churg-Strauss syndrome (CSS) is a necrotising systemic vasculitis which affects small- to medium-sized blood vessels. To our knowledge, this is the first study specifically on CSS in Australia. We aimed to determine the clinical and laboratory features and prognosis of CSS in South Australia.

**Methods:** This was a retrospective review of records of all patients with CSS managed through the Immunology Department at the Royal Adelaide Hospital between 2002 and 2008. The clinical and laboratory features, management and outcome of these patients were analysed.

**Results:** Nineteen patients had a diagnosis of CSS, of whom 10 were male. Mean age of onset was 54 years. Eighteen patients had asthma and 16 had sinonasal disease. Fourteen patients had rash, 14 had neuritis, 11 had myositis, 7 had gastrointestinal involvement, 2 had renal disease, and 1 had coronary vasculitis. Two patients had received leukotriene antagonists previously. Mean eosinophil count at diagnosis was  $9.79 \times 10^3/\text{mL}$ , mean CRP 81 mg/L and mean ESR 42 mm/h. At induction, all patients received prednisolone. Five patients received additional treatment with methotrexate, cyclophosphamide, azathioprine or intravenous immunoglobulin. Patients experienced a mean 1.84 relapses, and 5 patients survived a mean 106 months and died at a mean age of 75.8 years. Complications included venous thromboembolism (VTE) (5 patients, associated with CSS activity in 4 patients), osteoporosis (5 patients), infection (3 patients), and persistent neuropathy (3 patients). Nine patients were p-ANCA/MPO+, 2 patients were c-ANCA/PR3+, and 8 patients were ANCA negative. There were no significant differences between ANCA-positive and ANCA-negative patients.

**Conclusion:** CSS is an eosinophil-dependent ANCA-associated vasculitis. Many cases can be managed with corticosteroids alone. In some cases cardiac, renal or neurological complications can be serious and immunosuppressive medications may be required for induction and maintenance. Thromboembolism is a major complication and should be addressed in the management of these patients.

## POSTER

### 75. RATES OF INCREASES FOR IVIG USE IN NSW AND AUSTRALIA

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**Background:** Intravenous immunoglobulin (IVIg) has been used as replacement therapy for antibody deficiency for several decades, and is also now an important immunomodulatory therapy for a growing list of autoimmune diseases, leading to burgeoning demand. Attempts to address this demand have included controlling supply (via clinical use criteria), increasing local production, and importing supplies. An understanding of the drivers of this growth and future trends is important to ensure appropriate plans are made for managing supply of an expensive and limited resource.

**Methods:** Time trends in monthly data on patient episodes and IVIg distribution from 07/2003 to 02/2011 in NSW were analysed for common indications, as well as annualised trends by prescribing discipline at a national level over the period 2004/5 to 2009/10. All analyses were conducted in R software.

**Results:** Nationally IVIg usage has been growing at an annual rate of 11.6%, while per 1000 population this is slightly lower at 9.9%, suggesting a small effect of population growth. Use in neurology and haematology have been growing above the overall trend, while the rate of increase in use in immunology has slowed in recent years. In NSW, IVIg use has been growing more rapidly (13.2% pa) than the national rate with patient episodes also increasing by 12.4% per year. Common indications which are growing above trend in NSW, include acquired hypogammaglobulinaemia, chronic inflammatory demyelinating polyneuropathy and myasthenia gravis, while usage for primary immunodeficiency and idiopathic thrombocytopenic purpura is growing below trend.

**Conclusion:** Despite recent efforts to control supply of IVIg, the rate of use in Australia continues to grow rapidly. Most of this increase is due to the rise in patient episodes. In NSW, this appears to be being driven by neurological and haematological indications. This analysis will now be extended to project future trends.

## CLINICAL GRAND ROUNDS – ORAL PRESENTATION

### 1. A CASE OF GRAVES' DERMOPATHY AFFECTING THE BREAST

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Infiltrative thyroid dermopathy is a known yet infrequent feature of Graves' disease. The most common location is the pretibial region or the dorsum of the foot (pretibial myxoedema, PTM). We describe a case of likely infiltrative dermopathy affecting the breast in a 53 year old woman with concurrent PTM and thyroid stimulating hormone receptor antibodies.

The patient was referred with a two year history of recurrent episodes of mastitis of increasing frequency. Each episode was marked by a prodrome of malaise with myalgia and arthralgia followed by the development of localised inflammation to the right breast with tender erythema and the appearance of peau d'orange. While initial episodes were treated as infection, the course of later episodes appeared independent of antibiotic therapy.

Her medical history is significant for autoimmune thyroid disease. She recalls a presentation ten years ago with fatigue and weight loss and was subsequently commenced on thyroxine. She also had an undiagnosed skin lesion on the pretibial region of her right leg that was characterised by erythema, pruritis and previous central ulceration. Interestingly, this lesion had improved with systemic corticosteroids.

The episodes of mastitis were associated with marked elevation of her inflammatory markers with C-reactive protein and erythrocyte sedimentation rate upwards of 120 mg/l and 75 mm/hour respectively. Ultrasound investigation revealed subcutaneous oedema with some poorly defined hypochoic areas. A core biopsy revealed fibrosis with patchy perilobular lymphocytic infiltrate. A later fine needle aspiration biopsy demonstrated a small lymphocytic population and some macrophages. Cultures were sterile. Further investigation revealed elevated TSH receptor and thyroperoxidase antibodies but normal thyroid function. Magnetic resonance imaging of her orbits revealed no evidence of Graves' ophthalmopathy.

The findings point to a unifying diagnosis of Graves' disease, with pretibial myxoedema and infiltrative dermopathy affecting the breast.

## CLINICAL GRAND ROUNDS – ORAL PRESENTATION

### 2. AN UNUSUAL PRESENTATION OF X LINKED VARIANT CHRONIC GRANULMATOUS DISEASE PREVIOUSLY DIAGNOSED WITH MUNCHAUSEN SYNDROME

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A 40 year old man presented with new right medial malleolus discharging ulcer with satellite superficial abscesses and left foot cellulitis. His recent past medical history had been complicated for the proceeding 3 years with bilateral recurrent *Staphylococcus Auerus* bacteraemia, osteomyelitis and abscesses following a traumatic left calcaneal fracture. In the year prior the patient had required sequential right toe and forefoot amputations. During his hospital admissions it was noted that whilst intravenous access was present he would develop recurrent septicaemia of varying organisms including *aspergillus*, *enterobacter*, *acinterobacter* and *staphylococcus auerus*. Treating clinicians at this point in time felt that the bacteraemia may in part be contributed by the patient tampering with his venous access.

His family medical history was remarkable for his mother suffering for previous miscarriages and systemic lupus. He had one daughter who was well and no siblings.

During his admission his abscesses grew *methicillin sensitive staphylococcus auerus*. These were treated with surgical drainage and intravenous antibiotics. Examination was remarkable for hepatosplenomegaly and superficial granulomas post cannulation. He was noted to have mild neutropenia, polyclonal hypergammaglobulinaemia. His T cells were within normal limits. There was no clinical evidence of vasculitis with negative autoantibody and vasculitis serology.

Neutrophil function tests were performed which showed reduction in a granulocyte deducing DHR response to Panasorbin. Western blot studies for gp91phox, p47phox and p67phox proteins showed a decreased in gp91phox with genetic studies pending. As a result a diagnosis of variant X-linked chronic granulomatous (CGD) disease was made. He was maintained on prophylactic bactrim with no recurrence of sepsis or abscesses for 5 months. This is an unusual case of X-CGD presenting in late age. A diagnosis of deliberate tampering or Munchausen's should only be made when other possibilities have been excluded.

## CLINICAL GRAND ROUNDS – ORAL PRESENTATION

### 3. EOSINOPHILIC MENINGITIS: A DIAGNOSTIC DILEMMA

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A 14 month old girl presented to emergency department with a two week prodrome of fever, irritability and vomiting. On examination she was noted to have a bulging anterior fontanelle.

MRI revealed multiple punctate lesions and a lumbar puncture demonstrated low CSF glucose and high protein. CSF microscopy showed a high percentage of eosinophils (45%) consistent with a diagnosis of eosinophilic meningitis. Empirical management included intravenous antibiotics (penicillin, cefotaxime and vancomycin) with acyclovir and dexamethasone. She was transferred to the Children's Hospital at Westmead for ongoing management.

Repeat lumbar punctures confirmed eosinophilic meningitis. Her antibiotics were stopped and she was commenced on an anti-helminthic with continued high dose steroids. Further investigations revealed a positive ANCA, mildly elevated LDH and a normal vitamin B12. A chest x-ray showed increased perihilar markings. Subsequent MRIs demonstrated disease progression with multiple new haemorrhagic foci.

Despite treatment her admission was complicated by raised intracranial pressure requiring an external ventricular drain, persistent fevers and cough with raised inflammatory markers and peripheral eosinophilia ( $1.2 \times 10^9/L$ ), and the onset of left leg weakness.

In our presentation we highlight the differential diagnoses and diagnostic challenges of this intriguing case.

## CLINICAL GRAND ROUNDS – ORAL PRESENTATION

### 4. IF THE DRESS FITS – A CASE OF DRUG INDUCED EOSINOPHILIA WITH SYSTEMIC SYMPTOMS SECONDARY TO PHENYTOIN WITH HHV-6 REACTIVATION AND CMV GASTRITIS

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Drug induced eosinophilia with systemic symptoms (DRESS) syndrome is a severe T-cell mediated hypersensitivity reaction. There is evidence that herpesviruses such as Human Herpes Virus 6 (HHV-6) may play a role in the pathogenesis of drug hypersensitivity.

Here, we present the case of a 33 year old Aboriginal male who presented to his GP with exfoliative dermatitis approximately 3 weeks after starting phenytoin. He later developed severe epigastric pain and was admitted to hospital. Eosinophils were raised ( $5.5 \times 10^9/L$ ) and liver function was deranged (GGT 1500 U/L, ALT 700 U/L). Phenytoin associated DRESS syndrome was suspected, therefore Phenytoin was ceased and the patient was started on steroids. Gastroscopy revealed severe circumferential ulceration of the distal stomach and biopsy confirmed Cytomegalovirus (CMV) infection. There was serological evidence of HHV-6 reactivation and HHV-6 DNA was detected by whole blood PCR. Peripheral blood flow cytometry revealed a CD8 lymphocytosis with increased activation markers on both CD4 and CD8 positive T-cells with severe CD19 B-cell lymphopaenia. His skin, epigastric pain, eosinophilia and liver function tests gradually improved after ceasing the phenytoin and he was discharged on a tapering regime of steroids.

This case highlights the link between drug hypersensitivity and human herpesviruses such as CMV and HHV-6. Further studies are needed to investigate this association and the role of these viruses in both the immunopathogenesis of DRESS and the immune dysregulation associated with this disease.

## CLINICAL GRAND ROUNDS – ORAL PRESENTATION

### 5. INFANT WITH SUSPECTED SEPSIS, HEPATOSPLENOMEGALY AND CYTOPENIAS - CONSIDER HAEMOPHAGOCYTOSIS

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**Background:** Haemophagocytic lymphohistiocytosis (HLH) is a severe multisystem inflammatory disease characterized by prolonged fevers, hepatosplenomegaly and cytopenias.

**Case Report:** A 5 month old boy of non-cosanguineous Caucasian parents was retrieved from a peripheral hospital to the ICU at RCH with presumed sepsis. He had a 4 day history of vomiting, lethargy and one episode of diarrhea. He was treated locally for probable gastroenteritis with nasogastric fluid rehydration. On day 3 of admission he deteriorated with lethargy, poor perfusion, fever and cytopenia. He was given a dose of iv ceftriaxone. On arrival of the RCH retrieval team he was periarrest with tachycardia, unrecordable blood pressure but palpable brachial pulses. Abdominal exam revealed hepatosplenomegaly. He was intubated and ventilated and transferred. Initial investigations at RCH showed anemia, thrombocytopenia, hypofibrinogenaemia and high ferritin. Management consisted of transfusions of red cells, platelets, FFP and cryoprecipitate, inotropic support, IV corticosteroid and antibiotics, and 2 doses of IVIG. On day 6 a bone marrow aspirate (BMA) was performed, which showed a normocellular bone marrow with toxic changes and megaloblastic features. Haemophagocytosis was demonstrated in some mature histiocytes. There was an increase in juvenile histiocytes. The patient was extubated and transferred to the ward on day 11 with a weaning dose of hydrocortisone. On Day 13 he developed lip smacking and jitteriness suggesting seizures. Repeat BMA on day 15 confirmed HLH. HLH04 protocol was commenced (dexamethasone, cyclosporin and etoposide), with intrathecal methotrexate and hydrocortisone.

**Results:** NK cell function was absent, intracellular NK cell perforin expression was normal, and NK cell degranulation (CD107a expression) was reduced. Genetic analysis showed compound heterozygous mutations in STXBP2 (Familial HLH type 5). The family were tissue typed with the view of proceeding to HSCT.

**Conclusion:** HLH is a severe disease that requires prompt diagnosis and management, and may present as sepsis.

## CLINICAL GRAND ROUNDS – ORAL PRESENTATION

### 6. CASE PRESENTATION: A LIFE-SAVING EPISODE OF ANAPHYLAXIS

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**Introduction:** Marasmickwashiorkor is the most severe of the childhood malnutrition syndromes resulting from marked protein and caloric insufficiency. It carries a high rate of morbidity and mortality. Although commonly associated with extreme poverty in developing countries, rare reports in the developed world have outlined protein-energy malnutrition in the context of chronic malabsorptive conditions, food faddism, nutritional ignorance and

food allergen avoidance without appropriate medical advice.

**Case:** An eleven-month old girl of Asian immigrant parents living in suburban Sydney presented to the Emergency Department with a history of anaphylaxis after her first known ingestion of soy, sesame and uncooked egg (in mayonnaise) for which she received appropriate acute treatment. Background history of generalised eczema had been treated briefly with topical mild corticosteroids. Following an internet search for eczema management, her diet was severely restricted and breastfeeding weaned. Examination revealed a lethargic infant with dependent oedema, erythroderma, sparse hair, conjunctival pallor, global developmental delay and poor stores of muscle bulk and fat. Significant laboratory abnormalities included severe hyponatraemia, hypoalbuminaemia with proteinuria, metabolic alkalosis, anaemia, and deficiencies of zinc, vitamin D, and iron. Workup for underlying causes including for metabolic and gastrointestinal disease revealed highly elevated anti-gliadin IgG antibody. She will undergo further evaluation to exclude coeliac disease, although she had inadvertently been on a gluten-free diet months before presentation. Following initial resuscitation, nutritional rehabilitation was commenced according to WHO guidelines with reversal of clinical and biochemical abnormalities including resolving proteinuria.

**Conclusion:** Presentation to hospital with anaphylaxis essentially saved this infant's life as it led to prompt diagnosis and treatment of the previously unrecognised severe protein and calorie malnutrition. This case also illustrates the perils of seeking health advice from non-reputable internet sites and implementing food elimination diets without appropriate medical supervision.

## CLINICAL GRAND ROUNDS – ORAL PRESENTATION

### 7. BLIND, DEAF AND DUMFOUNDNED; UNRAVELLING SOME OF THE MYSTERIES OF SUSAC'S SYNDROME

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**Introduction:** Susac's syndrome is a rare but devastating condition characterized by retinal branch artery occlusion, sensorineural deafness and encephalopathy mostly in young people. The pathogenesis and treatment are not well defined although the disease is attributed to a microangiopathy involving the arterioles of the brain, retina and cochlea. Prompt diagnosis is essential as early therapy may reduce sequelae and improve recovery.

**Case:** A 30 year old male was diagnosed with Susac's syndrome in February 2010, manifesting as right sided sensorineural hearing loss, encephalopathy and ataxia accompanied by typical MRI brain lesions (T2 hyperintensities in the corpus callosum and periventricular white matter) and right retinal branch artery occlusion on fundal photography. A brain biopsy of the meninges and cortex revealed a diffuse pial infiltrate predominated by CD8 T-lymphocytes and macrophages. Micro-infarctions were seen in the territory of small pial arteries. No classical vasculitic changes were seen although some vessels showed complement C3d in the wall and were swollen. The patient was commenced on immunosuppression comprising 3 days of pulsed methylprednisolone and subsequent reduction of oral prednisone, and 6 cycles of monthly cyclophosphamide (1 g) and IVlg 2g/kg. Remission was achieved although there was minimal reversibility of his right sided sensorineural deafness. His condition relapsed 6 months later following commencement of weekly methotrexate. Pulse methylprednisolone, plasma exchange, and rituximab (375mg/m<sup>2</sup> x4) and mycophenolate mofetil (MMF) 1.5g BD were commenced, achieving clinical remission and the patient was maintained on MMF 1.5g BD and tapering course of prednisone. However, he subsequently suffered another disease relapse (7 months later) for which pulsed methyl prednisolone and infliximab was commenced (5 mg/kg at weeks 0, 2, 6 and then every 8 weeks) and MMF continued. His disease has been in remission for 3 months although it is likely he will require a cochlear implant for deafness.

**Discussion:** Susac's syndrome is a rare condition that results in significant sequelae with varying functional outcomes and residual disability. The pathogenesis is not entirely clear but it is most likely to be an immune-mediated endotheliopathy that affects the microvasculature of the brain, retina, and inner ear. This is supported by recent pathological and serological studies. The best approach to treating refractory or relapsing disease remains to be established. This is the first report of infliximab used in the induction and maintenance of relapsing disease. Further multicentre trials may elucidate the role of biologic agents in inducing and maintaining remission. An early diagnosis and subsequent treatment may halt progression of disease and reduce the extent of permanent disability.

## CLINICAL GRAND ROUNDS – ORAL PRESENTATION

### 8. A CASE OF CIQ DEFICIENCY, LUPUS AND MOYA MOYA

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We describe a 4 year old girl with C1q deficiency, systemic lupus erythematosus and moya moya disease.

She presented at 19 months of age with pyrexia of unknown origin on a background of microcephaly, developmental delay, recurrent urinary tract infections and chronic diarrhoea. Clinically, a malar rash, microcephaly and a 6th nerve palsy was evident. Biochemically, elevated liver functions, inflammatory markers, triglycerides, and ferritin, were suggestive of haemophagocytic lymphohistocytosis. Pulse steroids resulted in elimination of her febrile episodes. CH50 was abnormal and further investigations reveal C1q deficiency.

Unfortunately, her symptoms persisted, with worsening of her rash, ongoing issues with UTIs persistent 6th nerve palsy, and the development of pancreatitis. She developed spasticity and regressed developmentally. ANA, dsDNA was elevated, C3 and C4 were low and a diagnosis of SLE was made..

She was commenced on steroids and azathioprine. Brain imaging showed changes consistent with cerebral lupus. With ongoing oral prednisolone and mycophenolate, her condition improved clinically and biochemically with decreased titers of ANA, dsDNA and normalization of C3 and C4.

In April 2011, she presented to ED with bloody diarrhoea, fevers, lethargy and seizures. Salmonella was isolated in stool. Cerebral imaging revealed infarcts and an angiogram showed severe cerebral vessel narrowing, suggestive of moya moya disease. Due to the severity of her cerebral disease, she underwent a bypass procedure (encephaloduromyomangiosis) in an attempt to increase blood flow to the brain.

The grand round presentation will focus on the unusual and severe complications of C1q deficiency and SLE. Management and treatment options such as BMT and immunomodulation will be discussed.



## CLINICAL GRAND ROUNDS – ORAL PRESENTATION

### 9. COMPOUND HETEROZYGOUS MUTATIONS OF PRF1 GENE CAUSING CHRONIC INTERSTITIAL PNEUMONITIS

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**Background:** Perforin defects have been implicated in cases of familial haemophagocytic lymphohistiocytosis manifesting in severe multisystemic inflammatory disease usually presenting in infancy. We present the case of an eight year old girl with perforin gene mutations presenting with chronic interstitial pneumonitis.

**Case Report:** A previously well six year old female presented with dyspnoea and cyanosis on a four week history of increasing lethargy. She was afebrile with an unremarkable chest examination. There were no specific extrapulmonary symptoms or

signs. A CT chest revealed bilateral lower lobe consolidation. Investigations revealed normal immunoglobulin levels, lymphocyte subsets analysis, lymphocyte function and neutrophil function. A lung biopsy revealed non-specific and extensive granulomatous proliferation of alveolar eosinophilic histiocytes. A causative organism (including *Mycobacterium tuberculosis*, *Epstein Barr Virus*, *Pneumocystis jiroveci*, *Aspergillus fumigatus*, *Mycoplasma pneumoniae*, *Legionella species* and *Coxiella burnetii*) was not detected. Bone marrow examination was normal. A diagnosis of severe "idiopathic" interstitial pneumonitis was made. There was an improvement with high dose corticosteroids but rapid clinical relapses followed attempts to wean steroid therapy. Another referral to immunology was made 14 months later with development of mild hypogammaglobulinaemia and mild NK and CD8+ T-cell lymphopenias.

**Results:** Further investigations revealed reduced intracellular perforin expression in NK cells, CD3+/CD56+ cells and CD8+ T-cells and poor NK cytolytic activity. Perforin gene analysis revealed compound heterozygous mutations of the PRF1 gene, K285del (amino acid deletion on exon 3) and A91V (amino acid substitution on exon 2). Sequencing confirmed that each of the patient's parents carries one of the mutations.

**Conclusion:** This case highlights an atypical phenotypic presentation of perforin gene mutation. Such defects should be considered in cases of steroid dependent inflammatory lung disease where tissue histology reveals a predominance of histiocytes. Further studies of perforin gene regulation are needed to better understand the clinical spectrum of perforin gene mutations.





# 22 years

## of ASCIA Annual Scientific Meetings 1990 – 2011



- 1990 1st ASCIA ASM – Melbourne VIC (Hilton on the Park, April 29 - May 1)
- 1991 2nd ASCIA ASM – Perth, WA (Burswood Hotel, December 1-3)
- 1992 3rd ASCIA ASM – Cairns, QLD (Hilton Hotel, September 13-15)
- 1993 4th ASCIA ASM – Sydney, NSW (Darling Harbour, April 29 – May 1)
- 1994 5th ASCIA ASM – Canberra, ACT (Lakeside Hotel, December 2-5)
- 1995 6th ASCIA ASM – Sydney, NSW (Regent Hotel, October 30-November 3) with TPAIS
- 1996 7th ASCIA ASM – Adelaide, SA (Hyatt Hotel, December 5-7)
- 1997 8th ASCIA ASM – Wellington, NZ (Convention Centre, April 5-8) held with TSANZ
- 1998 9th ASCIA ASM – Brisbane, QLD (Sheraton Hotel, August 28-30)
- 1999 10th ASCIA ASM – Uluru, NT (Ayers Rock Resort, September 24-27)
- 2000 11th ASCIA ASM – Sydney, NSW (Convention Centre, October 15-20)  
(held as part of the 17th World Allergy Congress)
- 2001 12th ASCIA ASM – Perth, WA (Sheraton Hotel, September 28-30)
- 2002 13th ASCIA ASM – Adelaide, SA (Hilton Hotel, September 27-29)
- 2003 14th ASCIA ASM – Melbourne, VIC (Sheraton Southgate Hotel, September 10-12)
- 2004 15th ASCIA ASM - Gold Coast, QLD (Marriott Hotel, September 8-10)
- 2005 16th ASCIA ASM – Queenstown, NZ (Millennium Hotel, August 31 - September 2)
- 2006 17th ASCIA ASM – Sydney, NSW (Manly Pacific Hotel, September 7-9)
- 2007 18th ASCIA ASM – Fremantle, WA (Esplanade Hotel, November 14-16)
- 2008 19th ASCIA ASM - Melbourne (Park Hyatt Hotel, November 12-14)
- 2009 20th ASCIA ASM – Adelaide, SA (Adelaide Convention Centre, September 16-18)
- 2010 21st ASCIA ASM – Broadbeach, QLD (Gold Coast Convention & Exhibition Centre, September 1-4)
- 2011 22nd ASCIA ASM – Sydney, NSW (Sydney Convention & Exhibition Centre, Darling Harbour, September 6-10)