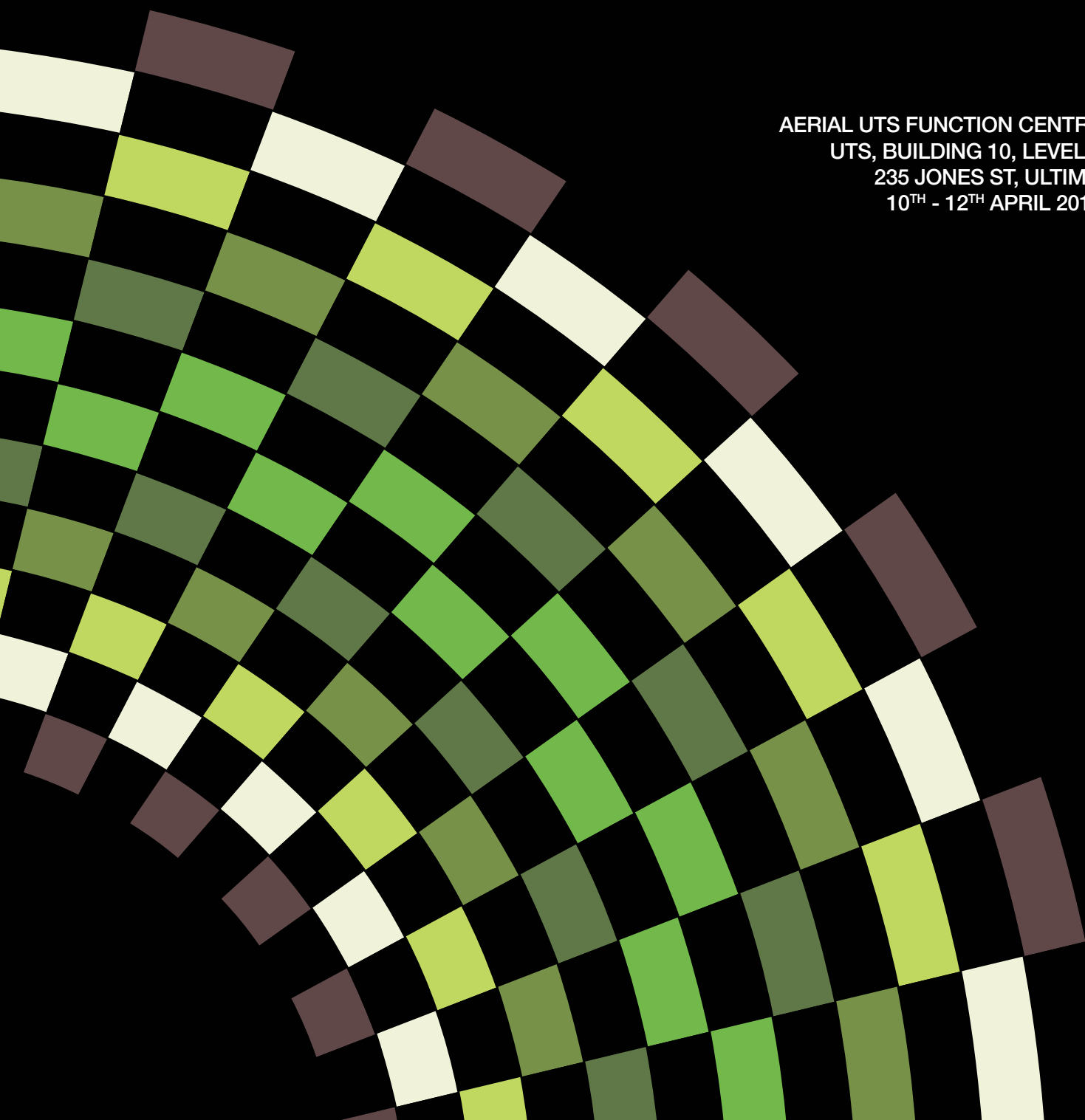
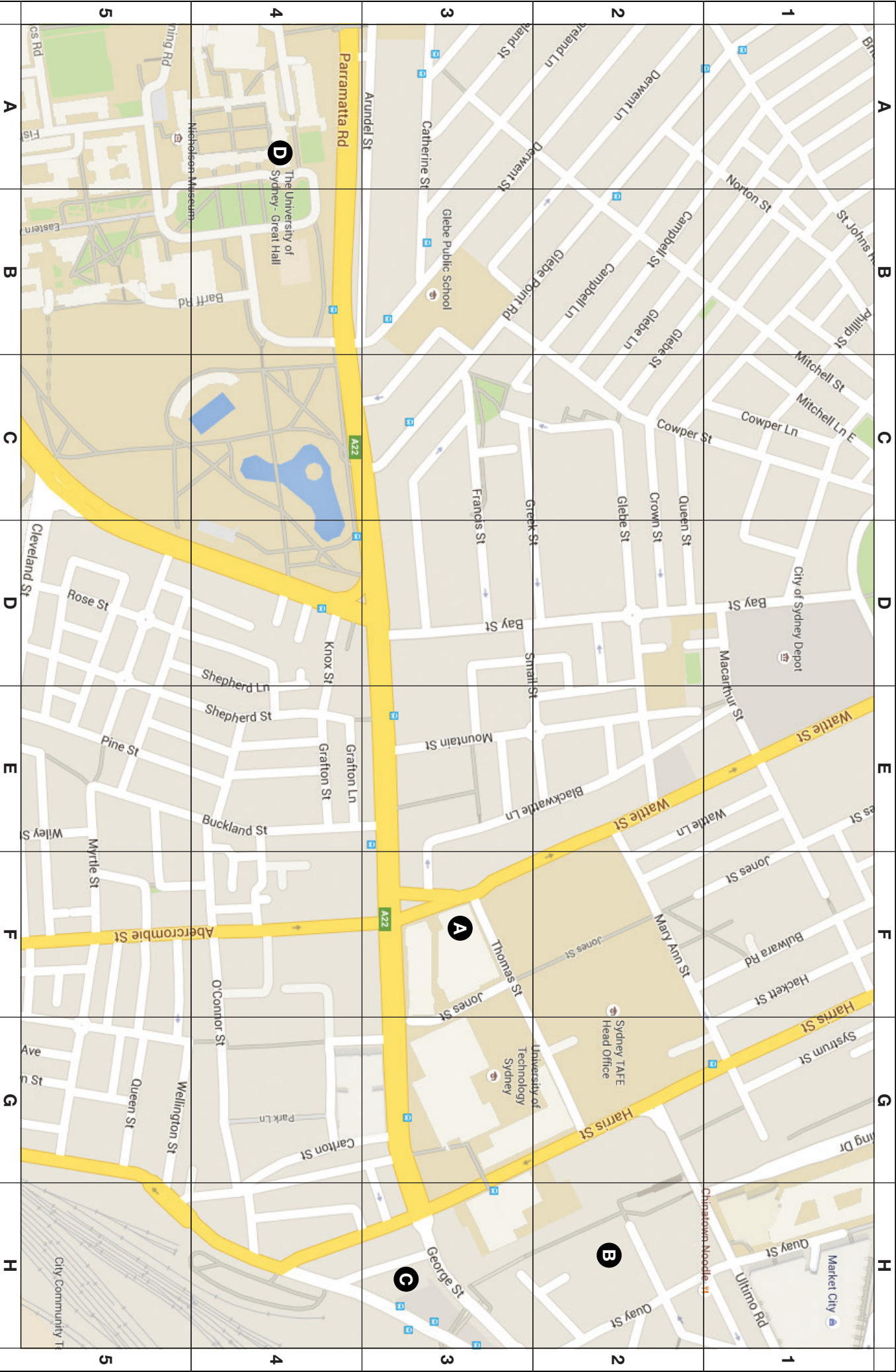


THE
TRANSPLANTATION SOCIETY
OF AUSTRALIAN AND NEW ZEALAND

34TH ANNUAL SCIENTIFIC MEETING
PROGRAM AND ABSTRACT BOOK

AERIAL UTS FUNCTION CENTRE
UTS, BUILDING 10, LEVEL 7
235 JONES ST, ULTIMO
10TH - 12TH APRIL 2016





A AERIAL UTS FUNCTION CENTRE (F3) **B** NOVOTEL SYDNEY (H2) **C** MECURE HOTEL (H3) **D** GREAT HALL, SYDNEY UNIVERSITY (A4)



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The Transplantation Society of Australia and New Zealand

Thirty-fourth Annual Scientific Meeting

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PROGRAM AT A GLANCE

Sunday, 10 April 2016		
10:00–12:00	Renal Transplant Advisory Committee Meeting	Aerial UTS Function Centre, Wattle Theatre
11:30–12:30	Pancreas & Islet Advisory Committee Meeting	Aerial UTS Function Centre, Thomas Theatre
12:00–13:00	Registration and Lunch with sponsors	Aerial UTS Function Centre, Foyer
13:00–13:15	Official Opening: <i>TSANZ President</i>	Aerial UTS Function Centre, Harris Theatre
13:15–14:15	PLENARY 1: Novartis Symposium Issues in Donors and Recipients That can Alter Outcomes: Biomarkers and the Metabolic Syndrome	Aerial UTS Function Centre, Harris Theatre
14:15–14:30	Afternoon tea	Aerial UTS Function Centre, Foyer
14:30–15:15	Ian McKenzie Award Lecture Generating Clinical Research Evidence for Transplantation	Aerial UTS Function Centre, Harris Theatre
15:15–16:15	Improving Gender Equality in Science and Medicine	Aerial UTS Function Centre, Harris Theatre
16:15–17:45	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 1: Outcome Measures #1 Free Communications 2: Immunobiology: Tolerance and Tregs Free Communications 3: Sensitisation, Antibodies, ABO Incompatible Transplantation & Organ Donation and Ethics	Aerial UTS Function Centre, Harris Theatre Thomas Theatre Wattle Theatre
17:45–18:45	TSANZ Annual General Meeting	Aerial UTS Function Centre, Harris Theatre
18:30–19:30	Donor Surgeons & Donor Co-ordinators Advisory Committee Meeting	Aerial UTS Function Centre, Wattle Theatre
18:45–19:45	Welcome Reception	Aerial UTS Function Centre, Foyer
19:30–21:00	Live Donor Surgeons Workshop	Aerial UTS Function Centre, Thomas Theatre

Monday, 11 April 2016		
06:30–07:30	TSANZ Fun Run/Walk (5km) Start: Aerial UTS Function Centre Sponsor: Organ & Tissue Authority	
07:30–08:00	Breakfast with sponsors	Aerial UTS Function Centre, Foyer
08:00–09:15	PLENARY 2: Organ and Tissue Authority Symposium Joint TSANZ /OTA/ATCA Session	Aerial UTS Function Centre, Harris Theatre
09:15–10:45	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 4: Transplant Complications #1 Free Communications 5: Xenotransplantation & Cell/tissue - Experimental Free Communications 6: Outcome Measures #2 Free Communications 7: Ischaemia Reperfusion Injury, Metabolism and Islet Transplantation	Aerial UTS Function Centre, Harris Theatre Broadway Theatre Thomas Theatre Wattle Theatre
10:45–11:15	Morning tea	Aerial UTS Function Centre, Foyer
11:15–12:45	PLENARY 3: Astellas Symposium Improvement of Transplant Outcome From Donor to Patient	Aerial UTS Function Centre, Harris Theatre
12:45–13:30	Lunch Lung Advisory Committee Meeting Tolerance Advisory Committee Meeting Vascularised Composite Allotransplantation Working Group Meeting Xenotransplantation Working Group Meeting	Aerial UTS Function Centre, Foyer Wattle Theatre Thomas Theatre Seminar Room 2 Seminar Room 1
13:30–15:30	President's Prize Symposium	Aerial UTS Function Centre, Harris Theatre
15:30–15:45	Afternoon tea	Aerial UTS Function Centre, Foyer
15:45–17:15	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 8: Transplant Complications #2 Free Communications 9: From disease to donation to the final destination – where is your patient on the transplant journey? Free Communications 10: Immunosuppression and Trials & Surgical techniques	Aerial UTS Function Centre, Harris Theatre Thomas Theatre Wattle Theatre
19:00–23:00	TSANZ Annual Dinner	The Great Hall, Sydney University

Tuesday, 12 April 2016		
07:30–08:00	Breakfast with sponsors	Aerial UTS Function Centre, Foyer
08:00–09:30	PLENARY 4: Novartis Symposium What's new in Transplant Immunology and Stem Cell Transplantation?	Aerial UTS Function Centre, Harris Theatre
09:30–10:00	Morning tea	Aerial UTS Function Centre, Foyer
10:00–15:00	Liver and Intestinal Advisory Committee Meeting	Aerial UTS Function Centre, Seminar Room 2
10:00–11:30	CONCURRENT STATE OF THE ART SESSIONS STATE OF THE ART 1: Astellas Symposium The Cell Therapies in Transplantation	Aerial UTS Function Centre, Harris Theatre
	STATE OF THE ART 2: Organ & Tissue Authority Symposium New Treatments in Transplantation	Broadway Theatre
11:30–13:00	CONCURRENT STATE OF THE ART SESSIONS STATE OF THE ART 3: Novartis Symposium Transplantation From Bench to Beside	Aerial UTS Function Centre, Harris Theatre
	STATE OF THE ART 4: Organ & Tissue Authority Symposium What is new in Organ Transplantation?	Broadway Theatre
13:00–14:00	Lunch	Aerial UTS Function Centre, Foyer
	Cardiac Advisory Committee Meeting	Wattle Theatre
	Paediatric Working Group Meeting	Thomas Theatre
14:00–15:30	PLENARY 5: Astellas Symposium Solutions and Challenges	Aerial UTS Function Centre, Harris Theatre
15:30–15:45	Afternoon tea	Aerial UTS Function Centre, Foyer
15:45–16:30	The Great Debate: 'The Future of Transplantation Research - Big Data Beats Small Animals'	Aerial UTS Function Centre, Harris Theatre
17:00	ASM Concludes	



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OFFICE BEARERS OF THE TRANSPLANTATION SOCIETY OF AUSTRALIA AND NEW ZEALAND

President

Professor Steve Chadban

President Elect & Chair, Advisory Committees/Working Groups

Professor Stephen Alexander

Honorary Secretary

Dr Karen Dwyer

Treasurer

Dr Robert Carroll

Councillors

Dr Nick Cross

New Zealand Representative

Dr Shane Grey

Liaison with Scientific Societies

Professor Henry Pleass

Surgical Representative

A/Professor Nick Shackel

RACP AMDC Liaison Rep

Professor Greg Snell

Nigel Palk

ATCA Representative

Scientific Program & Education Committee (SPEC)

A/Prof Daniel Chambers (Co-Chair)

Dr Kelli MacDonald (Co-Chair)

Dr Carolyn Clark

Dr Michael Fink

A/Prof Wayne Hawthorne

Dr Min Hu*

*TSANZ ASM Convenor

A/Prof Andrew Jabbour*

Dr Darren Lee

Dr Lucy Sullivan

A/Prof Allison Tong

TSANZ Administrative Staff

Ms Sommer Twycross

Executive Officer

Email: tsanz@tsanz.com.au

Mrs Nieves Piaggio

Administrative Officer

Email: admin@tsanz.com.au

Program and Abstract Book

Ms Marina Katerelos

Email: abstracts.tsanz.asm@gmail.com



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SPONSORS

The Transplantation Society of Australia and New Zealand gratefully acknowledges the support of the following companies in providing sponsorship for the Annual Scientific Meeting.

Platinum Sponsors

Novartis Pharmaceuticals Australia Pty Ltd

Astellas Pharma Australia Pty Ltd



Silver Sponsors

Organ and Tissue Authority



Australian Government
Organ and Tissue Authority





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AWARDS

The Transplantation Society of Australia and New Zealand gratefully acknowledges the support of the following companies for sponsoring awards presented at the Annual Scientific Meeting.

AWARDS

The President's Prizes
(supported by TSANZ)

Novartis Young Investigator Awards

Astellas Clinical Presentation Awards

Kidney Health Australia Awards

Ian McKenzie Prize for Outstanding Contribution in Transplantation
(supported by TSANZ)

Mark Cocks Transplantation Research Scholarship
(supported by Transplant Australia)

FINANCIAL STATEMENTS

The Transplantation Society of Australia and New Zealand (TSANZ) Financials for the Year Ended December 2015 are available on the easily accessible member password protected section of the TSANZ website www.tsanz.com.au.



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INVITED SPEAKERS

Novartis Lecturer

Professor John Lake
Director, Division of Gastroenterology,
Hepatology and Nutrition, University of Minnesota;
Director, Liver Transplant Program

Novartis Lecturer

Mr Gabriel C Oniscu MD, FRCS
Consultant Transplant Surgeon
Honorary Clinical Senior Lecturer
NRS Career Research Fellow
Chair of Research, Innovation and Novel Technology Advisory Group NHSBT
Transplant Unit
Royal Infirmary of Edinburgh

Astellas Lecturer

Professor Alberto Sanchez Fueyo
Head of Renal Sciences,
Professor in Hepatology, Liver, Renal, Urology, Transplant,
Gastro/Gastro Intestinal Surgery Clinical Academic Group
Kings College London, UK

TSANZ Lecturer

A/Professor Dorry Segev, MD, PhD
Associate Vice Chair, Department of Surgery,
Director, Epidemiology Research Group in Organ Transplantation
Johns Hopkins University
Baltimore, MD, USA

Astellas Lecturer

Professor Abraham Shaked
Hospital of the University of Pennsylvania
Department of Surgery
Philadelphia



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INVITED SPEAKERS

A/Professor Simon Barry

Robinson Research Institute, The University of Adelaide, SA

Professor Steve Chadban

Department of Renal Medicine, Royal Prince Alfred Hospital, NSW

Dr James Chong

Westmead Institute for Medical Research, NSW

Dr Philip Clayton

Royal Adelaide Hospital, SA

Dr Kumud Dhital

Department of Cardiothoracic Surgery, St Vincent's Hospital, NSW

Professor Karen Dwyer

Deakin University, VIC

Professor David Gottlieb

Professor of Haematology, University of Sydney, NSW

A/Prof Shane Grey

Garvan Institute of Medical Research, NSW

A/Professor Wayne Hawthorne

National Pancreas Transplant Unit, Westmead Hospital, NSW

Dr Geoff Hill

Queensland Institute of Medical Research, QLD

A/Professor Nicole Isbel

Department of Nephrology, Princess Alexandra Hospital, NSW

Dr Shilpa Jesudason

Royal Adelaide Hospital, SA

A/Professor Jerome Laurence

University of Sydney, NSW



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INVITED SPEAKERS

Professor Peter Macdonald
Cardiac Transplant Clinic, St Vincent's Hospital, NSW

Professor Jenny Martin
Institute for Molecular Bioscience, University of Queensland, QLD

Professor David McGiffin
The Alfred Hospital, VIC

Dr Scott McKenzie
Heart Failure & Cardiac Transplantation Unit, Prince Charles Hospital, QLD

Ms Felicity McNeill
CEO, The Organ and Tissue Authority, ACT

Dr Kenneth Micklethwaite
The Westmead Institute for Medical Research, NSW

Mr Nigel Palk
DonateLife, SA

Professor Henry Pleass
Department of Surgery, Westmead Hospital, NSW

Professor Greg Snell
Lung Transplant Service, Alfred Hospital, VIC

Dr Debbie Watson
School of Biological Sciences, University of Wollongong, NSW

Dr Angela Webster
University of Sydney, NSW

A/Professor Germaine Wong
Centre for Transplant and Renal Research, Westmead Hospital, NSW



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ABSTRACT REVIEW PROCESS AND PRESENTATION FORMATS

A total of 110 abstracts were submitted this year. Abstracts were blinded for authors and institutions and were reviewed by three reviewers (see below) assigned by the Scientific Program and Education Committee (SPEC). Reviewers did not review abstracts if a conflict of interest was identified. Reviewers scored between 6 to 12 abstracts and in general there was a close agreement between scores.

Two presentation formats will be used at the 2016 ASM. Each Free Communications session will commence with oral presentations (12 minutes each; 10 minutes presentation and 2 minutes questions) and conclude with mini-oral presentations (6 minutes each; 4 minutes presentation and 2 minutes questions; limited to 3 slides excluding title and acknowledgements).

The President's Prize (PP) will be awarded in two categories: Basic Science and Clinical. The highest-ranked abstracts from eligible applicants in both categories will be presented in a single PP session. The award in each category will be based on the quality of the abstract and the presentation on the day.

The reviewers of the abstracts for the TSANZ 2016 meeting were:

Stephen Alexander	Derek Hart	Bill Mulley
Richard Allen	Wayne Hawthorne	Brian Nankivell
Scott Campbell	Geoff Hill	Philip O'Connell
John Chen	Min Hu	Kathy Paizis
Carolyn Clark	Frank Ierino	Helen Pilmore
Philip Clayton	Nikky Isbel	Henry Pleass
Nick Cross	Andrew Jabbour	Sid Rajakumar
Ian Dittmer	Shilpanjali Jesudason	Paul Robertson
Lloyd D'Orsogna	Sean Kennedy	Natasha Rogers
Karen Dwyer	Gayathri Kumarasinghe	Christine Russell
Randall Faull	Jair Kwan	Mauro Sandrin
Jonathan Fawcett	Darren Lee	Shaundee Sen
Paolo Ferrari	Wai Lim	Nick Shackel
Allan Glanville	Grant Luxton	Alexandra Sharland
Hilton Gock	Peter Macdonald	Angela Webster
David Goodman	Kelli MacDonald	Keryn Williams
David Gracey	Fiona Mackie	Germaine Wong
Bruce Hall	Geoffrey McCaughan	
Wayne Hancock	Ian McKenzie	

The committee members thank these reviewers for their reviews and effort in supporting the meeting.

Daniel Chambers and Kelli MacDonald
Chairs of TSANZ Scientific Program & Education Committee (SPEC)



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The Transplantation Society of Australia and New Zealand

Thirty-fourth Annual Scientific Meeting

PROGRAM

Sunday, 10 April 2016

10:00–12:00	Renal Transplant Advisory Committee Meeting	Aerial UTS Function Centre, Wattle Theatre
11:30–12:30	Pancreas & Islet Advisory Committee Meeting	Aerial UTS Function Centre, Thomas Theatre

Sunday, 10 April 2016

12:00–13:00	Registration and lunch with sponsors	Aerial UTS Function Centre, Foyer
13:00–13:15	Official Opening: <i>TSANZ President</i> Prof Steve Chadban	Aerial UTS Function Centre, Harris Theatre
13:15–14:15	PLENARY 1: Novartis Symposium Issues in Donors and Recipients That can Alter Outcomes: Biomarkers and the Metabolic Syndrome <i>Chairs: Dr Robert Carroll and Dr Carolyn Clark</i> 13:15 Utilising Geonome Biomarkers in the Management of Immunosuppression Prof Abraham Shaked 13:45 Metabolic Syndrome and Liver Transplantation Prof John Lake	Aerial UTS Function Centre, Harris Theatre
14:15–14:30	Afternoon tea	Aerial UTS Function Centre, Foyer
14:30–15:15	IAN McKENZIE AWARD LECTURE <i>Chair: TSANZ President, Prof Steve Chadban</i> Generating Clinical Research Evidence for Transplantation A/Prof Angela Webster	Aerial UTS Function Centre, Harris Theatre
15:15–16:15	Improving Gender Equality in Science and Medicine <i>Chairs: Prof Karen Dwyer and A/Prof Shane Grey</i> 15:15 Gender Equity and the Athena SWAN Pilot Prof Jenny Martin 15:45 Allodepletion Strategies in Transplantation Dr Debbie Watson	Aerial UTS Function Centre, Harris Theatre

Sunday, 10 April 2016

16:15–17:45	CONCURRENT FREE COMMUNICATIONS SESSIONS		
	Free Communications 1: Outcome Measures #1		Aerial UTS Function
	<i>Chairs: Prof Peter Macdonald and Dr Ashley Irish</i>		Centre, Harris Theatre
Abstract	<i>— Oral presentations —</i>		
1	16:15	RECIPIENT AND PANCREAS GRAFT SURVIVAL AFTER KIDNEY-PANCREAS TRANSPLANTATION IN AUSTRALIA AND NEW ZEALAND: A COHORT STUDY 1984-2014 <u>WEBSTER Angela</u> , PENG Xi (Alex), KELLY Patrick, ANZIPTR on behalf of	
2	16:27	RETROSPECTIVE SINGLE CENTRE COMPARISON OF OUTCOMES BETWEEN NORMAL CRITERIA AND MARGINAL CRITERIA BRAIN DEAD HEART TRANSPLANTATION <u>CHEW Hong Chee</u> , LO Phillip, CAO Jacob, SUGIANTO Nara, DHITAL Kumud, GRANGER Emily, HAYWARD Christopher, JABBOUR Andrew, JANSZ Paul, KEOGH Anne, KOTLYAR Eugene, SPRATT Phillip, MACDONALD Peter	
3	16:39	OUTCOMES FOLLOWING TRANSFER OF PAEDIATRIC LIVER TRANSPLANT RECIPIENTS TO ADULT HEALTHCARE IN VICTORIA <u>SRINIVASAN Ashish</u> , APOSTOLOV Ross, LEONG Amanda, TESTRO Adam, JONES RobertT, HARDIKAR Winita	
4	16:51	ASSOCIATION BETWEEN DELAYED GRAFT FUNCTION (DGF) AND GRAFT LOSS IN DONATION AFTER CARDIAC DEATH (DCD) DONOR KIDNEY TRANSPLANTS – A PAIRED KIDNEY ANALYSIS <u>LIM WAI</u> , McDONALD Stephen, CHAPMAN Jeremy, PLEASS Henry, JAUQUES Bryon, WONG Germaine	
5	17:03	LONG-TERM GRAFT OUTCOMES OF KIDNEY TRANSPLANT RECIPIENTS WITH MEMBRANO-PROLIFERATIVE GLOMERULONEPHRITIS (MPGN) <u>LIM Wai</u> , WONG Germaine, PILMORE Helen, MULLEY William, WALKER Rowan, MENAHEM Solomon	
	<i>— Mini-oral presentations —</i>		
6	17:15	SEASONAL VARIATION IN KIDNEY TRANSPLANT OUTCOMES <u>LIM Wai</u> , WONG Germaine	

Sunday, 10 April 2016

7	17:21	REHABILITATION OUTCOMES FOLLOWING CARDIOPULMONARY TRANSPLANTATION AND INPATIENT REHABILITATION <u>SKALICKY David</u> , BOWMAN Malcolm, WU Jane, WOODBRIDGE Genevieve, THOMPSON-BUTEL Angelica, FAUX Steven
8	17:27	LIVING KIDNEY DONOR PRIORITIES FOR OUTCOMES: A NOMINAL GROUP TECHNIQUE STUDY <u>HANSON CS</u> , KANELIS J, CHADBAN SJ, CHAPMAN JR, CRAIG JC, WONG G, PINTER J, GARG AX, GILL JS, LEWIS JR, TONG A
9	17:33	CIRCULATING SOLUBLE-KLOTHO LEVELS MODESTLY INCREASE AFTER RENAL TRANSPLANTATION <u>TAN Sven-Jean</u> , CROSTHWAITE Amy, LANGSFORD David, OBEYSEKERE Varuni, IERINO Frank L, ROBERTS Matthew A, HOLT Stephen G, HEWITSON Timothy D, DWYER Karen M, TOUSSAINT Nigel D
10	17:39	SCOPE AND HETEROGENEITY OF OUTCOMES REPORTED IN COCHRANE SYSTEMATIC REVIEWS OF KIDNEY TRANSPLANTATION <u>SAUTENET Benedicte</u> , CRAIG Jonathan C, EVANGELIDIS Nicole , CHAPMAN Jeremy R, GILL John , WONG Germaine, TONG Allison
<hr/>		
16:15–17:51	Free Communications 2: Immunobiology: Tolerance and Tregs <i>Chairs: Dr Kelli MacDonald and Prof Geoff Hill</i>	
Abstract	— Oral presentations —	
11	16:15	MICROBIAL CONCORDANCE LEADS TO SKEWING OF THE LUNG ALLOGRAFT MACROPHAGE PHENOTYPE FROM PRO-FIBROTIC M2 TOWARDS TOLEROGENIC M1 <u>NELLES Ricky</u> , YERKOVICH Stephanie, HOPKINS Peter, CHAMBERS Daniel

Sunday, 10 April 2016

- | | | |
|----|-------|--|
| 12 | 16:27 | ROLE OF MHC CLASS I MOLECULES AND CO-STIMULATORY MOLECULES IN RODENT MHC MISMATCH SKIN TRANSPLANT MODELS

<u>LEONG Mario</u> , PAUL Moumita, MOUAWADH Mamdoh, CUNNINGHAM Eithne, TAY Szun Szun, WANG Chuanmin, BERTOLINO Patrick, BOWEN David, BISHOP Alex, ALEXANDER Ian, SHARLAND Alexandra |
| 13 | 16:39 | STIMULATION WITH ANTIGEN AND CYTOKINES INDUCE EXPRESSION IRF4 IN NAÏVE CD4+CD25+ T REGULATORY CELLS

<u>WILCOX Paul, L.</u> , ROBINSON Catherine, M., TRAN Giang, T., VERMA Nirupama, D., HALL Bruce M., HODGKINSON Suzanne, J. |
| 14 | 16:51 | TARGETED MODIFICATION OF DC PHENOTYPE AND FUNCTION WITH POROUS SILICON NANOPARTICLES.

<u>STEAD Sebastian</u> , McINNES Steven, Kireta Svjetlana, ROSE Peter, ROJAS-CANALES Darling, JESUDASON Shilpa, GREY Shane, CARROLL Robert, VOELCKER Nico, COATES Toby |
| 15 | 17:03 | TH17 PLASTICITY AND TRANSITION TOWARDS A PATHOGENIC CYTOKINE SIGNATURE IS REGULATED BY CYCLOSPORIN AFTER ALLOGENEIC-SCT

<u>GARTLAN Kate</u> , VARELIAS Antiopi, KOYAMA Motoko, MARKEY Kate, KUNS Rachel, RAFFELT Neil, OLVER Stuart, LINEBURG Katie, TEAL Bianca, CHEONG Melody, TEY Siok-Keen, MACDONALD Kelli, HILL Geoff |
| 16 | 17:15 | EXPRESSION OF THREE ALLOGENEIC MHC CLASS I IN RECIPIENT LIVER SIGNIFICANTLY PROLONGS SURVIVAL OF FULLY-ALLOGENEIC VASCULARISED CARDIAC ALLOGRAFTS

<u>MOAWADH Mamdoh</u> , PAUL Moumita, CUNNINGHAM Eithne, WANG Chuanmin, CUNNINGHAM Sharon, TAY Szun Szun, GRANT Logan, ALEXANDER Ian, BERTOLINO Patrick, BOWEN David, BISHOP Alexander, SHARLAND Alexandra |

Sunday, 10 April 2016

— <i>Mini-oral presentations</i> —		
17	17:27	CAN EXPRESSION OF ALLOGENEIC MHC CLASS II IN RECIPIENT LIVER INDUCE REGULATORY TRANSPLANTATION TOLERANCE? <u>MOAWADH Mamdoh</u> , PAUL Moumita, SON Taeuoung, CUNNINGHAM Eithne, WANG Chuanmin, TAY Szun Szun, HU Min, ALEXANDER Stephen, LOGAN Grant, ALEXANDER Ian, BERTOLINO Patrick, BOWEN David, DUDEK Nadine, PURCELL Anthony, BISHOP Alexander, SHARLAND Alexandra
18	17:33	ROLE OF IL-7 AND IL-7/ANTI-IL-7 ANTIBODY COMPLEXES IN TREG EXPANSION AND A MURINE SKIN ALLOGRAFT TOLERANCE MODEL <u>HU Min</u> , BURNS Heather, LIUWANTARA David, QIAN Yi Wen, WANG Yuan Min, ZHANG Geoff, HAWTHORNE Wayne, YI Shounan, ALEXANDER Stephen, O'CONNELL Philip
19	17:39	IN VITRO REACTIVITY OF CD4+CD25+ AND CD4+CD25- T CELL SUBSETS FROM RATS WITH TOLERANCE TO AN ALLOGRAFT <u>HALL Bruce M</u> , ROBINSON Catherine M, PLAIN Karren M, VERMA Nirupama D, TRAN Giang T, NOMURA Masaru, CARTER Nicole, BOYD Rochelle, HODGKINSON Suzanne J
20	17:45	ADMINISTRATION OF IL-2/IL-2 AB COMPLEX IN COMBINATION WITH ALLO-PEPTIDE PULSED HOST SPLENOCYTES PROLONGS SKIN ALLOGRAFT SURVIVAL <u>ZHANG Geoff</u> , WANG Yuan Min, HU Min, SAWYER Andrew, ZHOU Jimmy, GREY Shane, ALEXANDER Stephen
16:15–17:45	Free Communications 3: Sensitisation, Antibodies, ABO Incompatible Transplantation & Organ Donation and Ethics <i>Chairs: Dr Darren Lee and Prof Allan Glanville</i>	
Abstract	— <i>Oral presentations</i> —	
21	16:15	PROVIDING BETTER MATCHED DONORS FOR HLA MISMATCHED COMPATIBLE PAIRS THROUGH KIDNEY PAIRED DONATION <u>CANTWELL Linda</u> , WOODROFFE Claudia, D'ORSOGNA Lloyd, HOLDSWORTH Rhonda, FERRARI Paolo

Aerial UTS Function
Centre, Wattle Theatre

Sunday, 10 April 2016

- | | | |
|------------------------------------|-------|---|
| 22 | 16:27 | SYK INHIBITION REDUCES RENAL ALLOGRAFT INJURY IN A RAT MODEL OF ACUTE ANTIBODY-MEDIATED REJECTION IN HIGHLY SENSITIZED RECIPIENTS.
<u>RAMESSUR CHANDRAN</u> Sharmila, MA Frank, Y, TESCH Greg, H, HAN Yingjie, OZOLS Elyce, DI PAOLO Julie, MULLEY William, KANELIS John, NIKOLIC-PATERSON David, J |
| 23 | 16:39 | THE NATURAL HISTORY OF DONOR SPECIFIC ANTIBODIES (DSA) IN KIDNEY TRANSPLANT RECIPIENTS (KTX) AND ASSOCIATED CLINICAL OUTCOMES
<u>WAN Susan</u> , WYBURN Kate, YIN Jianlin, WATSON Narelle, SAUNDERS John, ERIS Josette |
| 24 | 16:51 | ADDITIONAL OPPORTUNITIES FOR TRANSPLANTING ORGANS FROM DONORS WITH BRAIN MALIGNANCIES? AN AUDIT OF THE NSW ORGAN AND TISSUE DONATION SERVICE (OTDS) ORGAN DONOR REGISTER
HANCOCK Rebecca, WYBURN Kate, O'LEARY Michael, <u>WEBSTER Angela</u> |
| — <i>Mini-oral presentations</i> — | | |
| 25 | 17:03 | UTILISING ORGANS FROM DONORS WITH BLOOD-BORNE VIRUSES (BBVS) IN NSW, 2010-2015
HANCOCK Rebecca, <u>WYBURN Kate</u> , O'LEARY Michael, WEBSTER Angela |
| 26 | 17:09 | DE NOVO DONOR SPECIFIC ANTI-HUMAN LEUKOCYTE ANTIGEN (HLA) ANTIBODIES IN KIDNEY TRANSPLANTATION - A SYSTEMATIC REVIEW AND META-ANALYSIS
<u>SHARMA Ankit</u> , LEWIS Joshua, WAI Lim, PALMER Suetonia, STRIPPOLI Giovanni, CHAPMAN Jeremy, CRAIG Jonathan, WONG Germaine |
| 27 | 17:15 | SUCCESSFUL ABO INCOMPATIBLE NON- HEART BEATING DECEASED DONOR KIDNEY TRANSPLANTATION
<u>KRISHNAN Anoushka</u> , PUTTAGUNTA Harish, HE Bulang, THOMPSON Ivan, BHANDARI Myank, SWAMINATHAN Ramyasuda, IRISH Ashley |
| 28 | 17:21 | TREATMENT OF ACTIVE ANTIBODY MEDIATED REJECTION IN RENAL TRANSPLANT RECIPIENTS
<u>MARUI Yuhji</u> , YAMAGUCHI Haruna, TANAKA Kiho, ISHII Yasuo |

Sunday, 10 April 2016

29	17:27	ALLOGRAFT DYSFUNCTION COMPLICATING PARATHYROIDECTOMY IN RENAL TRANSPLANT RECIPIENTS: A TYPICAL CASE AND REVIEW OF THE LITERATURE ON UNDERLYING MECHANISM <u>SEE Emily</u> , DWYER Karen	
30	17:33	ANTI BLOOD GROUP ANTIBODY TITRES IN BLOOD GROUP A AND B TRANSPLANT WAIT LISTED PATIENTS <u>RUDEMAN Irene</u> , VANHARDEVELDT Emma, HUGHES Peter, MASTERSON Rosemary	
31	17:39	BENEFITS OF MODERATING LOCATION OF DONOR LIFE SUPPORT WITHDRAWAL ON LIVER TRANSPLANTATION USING DONATION AFTER CIRCULATORY DEATH: A META-ANALYSIS <u>CAO Yiming</u> , SHAHRESTANI Sara, CHEW HC, CRAWFORD Michael, MACDONALD Peter, LAURENCE Jerome, HAWTHORNE WJ, DHITAL Kumud, PLEASS Henry	
17:55–18:45	TSANZ Annual General Meeting		Aerial UTS Function Centre, Harris Theatre
18:30–19:30	Donor Surgeons & Donor Co-ordinators Advisory Committee Meeting		Aerial UTS Function Centre, Wattle Theatre
18:45–19:45	Welcome Reception		Aerial UTS Function Centre, Foyer
19:30–21:00	Live Donor Surgeons Workshop		Aerial UTS Function Centre, Thomas Theatre

Monday, 11 April 2016

06:30–07:30	TSANZ Fun Run/Walk (5km) Commencing at Aerial UTS Function Centre	Sponsor: Organ & Tissue Authority
07:30–08:00	Breakfast with sponsors	Aerial UTS Function Centre, Foyer
08:00–09:15	PLENARY 2: Organ and Tissue Authority Symposium Joint TSANZ / ATCA Session <i>Chairs: Prof Frank Ierino and A/Prof Germaine Wong</i> 08:00 Organ Donors-Working Together to Achieve Successful Organ Transplantation (OTA) Ms Felicity McNeill 08:25 A new Model for Organ Offering in Australia (ATCA) Mr Nigel Palk 08:50 ANZDATA Registry Parenthood Data Update Dr Shilpanjali Jesudason	Aerial UTS Function Centre, Harris Theatre
09:15–10:45	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 4:Transplant Complications #1 <i>Chairs: A/Prof Wai Lim and A/Prof Kumud Dhittal</i>	Aerial UTS Function Centre, Harris Theatre
Abstract	— Oral presentations —	
32	09:15 RISK OF HEPATITIS B REACTIVATION IN CORE ANTIBODY POSITIVE PATIENTS AFTER RENAL TRANSPLANTATION <u>CHOU Eric</u> , STUART Katherine, CAMPBELL Scott, HAWLEY Carmel, FRANCIS Ross, ISBEL Nicole	
33	09:27 SUBCLINICAL CYTOMEGALOVIRUS VIRAEMIA IN RENAL TRANSPLANT RECIPIENTS <u>Barker Kristeen</u> , COOK Natasha, POLKINGHORNE Kevan, IERINO Frank	

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| 34 | 09:39 | GRAFT-VERSUS-HOST DISEASE PRECIPITATES CYTOMEGALOVIRUS REACTIVATION AFTER ALLOGENEIC BONE MARROW TRANSPLANTATION: LESSONS FROM THE FIRST PRECLINICAL MODEL.

<u>MARTINS Paulo</u> , TEY Siok-Keen, FLEMING Peter, KUNS Rachel D, ULLAH Md Ashik, VARELIAS Antiopi, KOYAMA Motoko, ANDONIOU Christopher E, DEGLI-ESPOSTI Mariapia A, HILL Geoffrey R

— <i>Mini-oral presentations</i> — |
| 35 | 09:51 | AZATHIOPRINE AND THE RISK OF SKIN CANCER IN SOLID ORGAN TRANSPLANT RECIPIENTS: A SYSTEMATIC REVIEW AND META-ANALYSIS
<u>ISBEL Nicole</u> , JIYAD Zainab, OLSEN Catherine, BURKE Michael, GREEN Adele |
| 36 | 09:57 | CRYPTOCOCCAL INFECTIONS IN SOLID ORGAN TRANSPLANT RECIPIENTS OVER A 15 YEAR PERIOD IN QUEENSLAND
<u>GASSIEP Ian</u> , McDOUGALL David, PLAYFORD Geoffrey, FRANCIS Ross S |
| 37 | 10:03 | INTRODUCTION OF AN IMMUNISATION SCHEDULE FOR ADULT RENAL TRANSPLANT PATIENTS IN NEW ZEALAND
<u>KARA Tonya</u> , MANLEY Paul R, CROSS Nicholas B |
| 38 | 10:09 | EXTENDED PROPHYLAXIS WITH VALGANCICLOVIR REDUCES THE INCIDENCE OF CYTOMEGALOVIRUS DISEASE IN RENAL TRANSPLANT RECIPIENTS
<u>STRAW S</u> , HUGHES P, MASTERSON R, COHNEY SJ |
| 39 | 10:15 | OBESITY IS A RISK FACTOR FOR CMV DISEASE IN RENAL TRANSPLANT RECIPIENTS RECEIVING EXTENDED PROPHYLAXIS WITH VALGANCICLOVIR
<u>STRAW SC</u> , HUGHES P, MASTERSON R, COHNEY SJ |
| 40 | 10:21 | CMV DISEASE IS NOT ASSOCIATED WITH ANY INCREASE IN GRAFT LOSS OR MORTALITY IN RENAL TRANSPLANT RECIPIENTS RECEIVING EXTENDED PROPHYLAXIS WITH VALGANCICLOVIR
<u>STRAW SC</u> , HUGHES P, MASTERSON R, COHNEY SJ |

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41	10:27	CYTOMEGALOVIRUS (CMV) SEROSTATUS, PATIENT AND ALLOGRAFT SURVIVAL AND PATTERNS OF CMV PROPHYLAXIS IN AUSTRALIAN AND NEW ZEALAND KIDNEY TRANSPLANT RECIPIENTS. <u>WONG Germaine</u> , PILMORE Helen, CHADBAN Steve, LIM Wai	
42	10:33	RENAL TRANSPLANTATION AND THE INCIDENCE OF BK AT 3 MONTHS POST TRANSPLANT <u>GARRY Lorraine</u> , CATAGUE Rachel, WYBURN Kate, GRACEY David, CHADBAN Steve, ERIS Josette	
43	10:39	RESULTS OF A BIOPSY BASED, EARLY INTERVENTION TREATMENT PROTOCOLS FOR BK VIRAEMIA AND BK VIRUS ASSOCIATED NEPHROPATHY. <u>JONES Scott</u> , TREVILLIAN Paul, LAI Katy, WALLER Sophie, HEER Munish, HIBBARD Adrian	
09:15–10:45	Free Communications 5: Xenotransplantation & Cell/Tissue - Experimental <i>Chairs: A/Prof Wayne Hawthorne and Dr David Liuwantara</i>		Aerial UTS Function Centre, Broadway Theatre
Abstract	— Oral presentations —		
44	09:15	GENERATION OF KNOCK-IN PIGS FOR XENOTRANSPLANTATION USING A HIGH FIDELITY CRISPR/CAS9 SYSTEM <u>SALVARIS Evelyn</u> , FISICARO Nella, VASSILIEV Ivan, McILFATRICK Stephen, BRADY Jamie, HAWTHORNE Wayne, LEW Andrew, NOTTLE Mark, COWAN Peter	
45	09:27	GTKO/CD55-CD59-HT PORCINE NEONATAL ISLET CELL CLUSTER (NICC) XENOGRAFTS PROVIDE LONG-TERM REVERSAL OF DIABETES <u>HAWTHORNE Wayne John</u> , HAWKES Joanne, CHEW YiVee, SALVARIS Evelyn, BURNS Heather, DAVIES Sussan, LIUWANTARA David, BARLOW Helen, BRADY Jamie, LEW Andrew, NOTTLE Mark, O'CONNELL Philip, COWAN Peter	
46	09:39	PROTECTION FROM INSTANT BLOOD MEDIATED INFLAMMATORY REACTION IN GAL-KO PORCINE NEONATAL ISLET CELLS EXPRESSING COMPLEMENT REGULATORS CD55/CD59 <u>LIUWANTARA David</u> , CHEW Yi Vee, FAVALORO Emmanuel, HAWKES Joanne, BURNS Heather, NOTTLE Mark, COWAN Peter, O'CONNELL Philip, HAWTHORNE Wayne	

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47	09:51	IL-28 IS A CRITICAL CYTOPROTECTANT IN TRANSPLANTATION <u>HENDEN Andrea</u> , GARTLAN Kate, LANE Steven, ROBB Renee, KUNS Rachel, CLOUSTON Andrew, HILL Geoff
48	10:03	HEMATOPOIETIC STEM CELLS AND THEIR PROGENITORS CRITICALLY REQUIRE AUTOPHAGY TO PROMOTE EARLY ENGRAFTMENT FOLLOWING ALLOGENEIC STEM CELL TRANSPLANTATION <u>LINEBURG Katie E</u> , LEVEQUE-EL MOUTTIE Lucie, LE TEXIER Laetitia, TEAL Bianca, KUNS Rachel, LANE Steven W, HILL Geoffrey R, MACDONALD Kelli PA
49	10:15	TRANSGENIC EXPRESSION OF HUMAN THROMBOMODULIN INHIBITS HMGB1-INDUCED PIG AORTIC ENDOTHELIAL CELL ACTIVATION <u>BONGONI Anjan Kumar</u> , WOLF Eckhard, AYARES David, RIEBEN Robert, COWAN Peter
50	10:27	CONJUGATION OF APYRASE TO PORCINE AORTIC ENDOTHELIAL CELLS PROLONGS CLOTTING OF WHOLE HUMAN BLOOD <u>BONGONI Anjan Kumar</u> , SALVARIS Evelyn, TERAMURA Yuji, ASIF Sana, NILSSON Bo, COWAN Peter
<i>— Mini-oral presentations —</i>		
51	10:33	A NEW PORCINE MODEL OF NORMOTHERMIC MACHINE PERFUSION OF LIVER DONATION AFTER CIRCUTORY DEATH: A PRELIMINARY STUDY <u>CAO Yiming</u> , CHEW HC, FERNANDEZ Karen, VILLANUEVA Jeanette, GAO Ling, HICKS Mark, MACDONALD Peter, DHITAL Kumud, PLEASS Henry
52	10:39	MICROARRAY GENE PROFILING OF IMMUNOSUPPRESSIVE INTERLEUKIN-17A PREACTIVATED HUMAN BONE MARROW-DERIVED MESENCHYMAL STEM CELLS (MSC-17) <u>SIVANATHAN Kisha Nandini</u> , ROJAS-CANALES Darling, GRONTHOS Stan, GREY Shane T, COATES Patrick T

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09:15–10:45	Free Communications 6: Outcome measures #2 <i>Chairs: Prof Richard Allen and Dr Peter Bergin</i>		Aerial UTS Function Centre, Thomas Theatre
Absract	— <i>Oral presentations</i> —		
53	09:15	EFFECT OF PROLONGED ISCHEMIC TIME ON GRAFT AND PATIENT OUTCOMES IN LIVE-DONOR KIDNEY TRANSPLANT RECIPIENTS <u>KRISHNAN Anoushka</u> , WONG Germaine, CHAPMAN Jeremy, COATES Patrick T, RUSS Graeme, RUSSELL Christine, HE Bulang, LIM Wai	
54	09:27	LONG-TERM GRAFT AND PATIENT OUTCOMES OF KIDNEY TRANSPLANT RECIPIENTS WITH AND WITHOUT TYPE II DIABETES MELLITUS (T2DM) <u>LIM Wai</u> , WONG Germaine, PILMORE Helen, McDONALD Stephen, CHADBAN Steve	
55	09:39	RELATIONSHIP BETWEEN 12-MONTH ESTIMATED GLOMERULAR FILTRATION RATE AND LONG-TERM GRAFT LOSS AFTER KIDNEY TRANSPLANTATION <u>LIM Wai</u> , WONG Germaine, CLAYTON Phil, PILMORE Helen, CHADBAN Steven	
56	09:51	REJECTION, GRAFT LOSS AND DEATH IN PAEDIATRIC AND ADOLESCENT KIDNEY TRANSPLANT RECIPIENTS <u>LIM Wai</u> , KENNEDY Sean, ALEXANDER Steve, WILLIS Francis, WONG Germaine	
57	10:03	LONG-TERM GRAFT OUTCOMES OF KIDNEY TRANSPLANT RECIPIENTS WITH PRESUMED GLOMERULONEPHRITIS (GN) <u>LIM Wai</u> , CHADBAN Steve, LUXTON Grant, PILMORE Helen, WONG Germaine	
	— <i>Mini-oral presentations</i> —		
58	10:15	RELATIONSHIP BETWEEN CHANGE IN ESTIMATED GLOMERULAR FILTRATION RATE AND LONG-TERM GRAFT LOSS AFTER KIDNEY TRANSPLANTATION <u>LIM Wai</u> , WONG Germaine, CLAYTON Phil, PILMORE Helen, CHADBAN Steven	

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59	10:21	OUTCOMES OF LATE ANTIBODY MEDIATED REJECTION: SINGLE CENTRE RETROSPECTIVE STUDY <u>RUDERMAN Irene</u> , MASTERSON Rosemary, HUGHES Peter	
60	10:27	REVERSIBILITY OF FRAILTY IN HEART TRANSPLANT LISTED PATIENTS <u>JHA Sunita</u> , HANNU Malin, NEWTON Phillip, GORE KAREN, WILHELM Kay, HAYWARD Chris, JABBOUR Andrew, KOTLYAR Eugene, KEOGH Anne, DHITAL Kumud, GRANGER Emily, JANSZ Paul, SPRATT Phillip, MONTGOMERY Elyn, HARKESS Michelle, TUNNICLIFF Peta, MACDONALD Peter	
61	10:33	THE VALUE OF SURVEILLANCE BIOPSIES AFTER PAEDIATRIC KIDNEY TRANSPLANTATION <u>ROSE Edward</u> , MACKIE Fiona, KENNEDY Sean	
62	10:39	DETERMINANTS OF TUBULAR MICROCALCIFICATION IN PROTOCOL BIOPSIES POST RENAL TRANSPLANTATION <u>JAW Juli</u> , LECAMWASAM Ashani, RICHARDS Avisha, COCHRANE-DAVIS Alex, SUNDARARAJAN Vijaya, HILL Prue, LANGHAM Robyn	
09:15–10:45	Free Communications 7: Ischaemia Reperfusion Injury, Metabolism and Islet Transplantation <i>Chairs: Prof Karen Dwyer and Dr Debbie Watson</i>		Aerial UTS Function Centre, Wattle Theatre
Abstract	— <i>Oral presentations</i> —		
63	09:15	MATRICELLULAR ACTIVATION OF CD47 LIMITS SELF-RENEWAL TO PROMOTE RENAL ISCHEMIA REPERFUSION INJURY <u>ROGERS Natasha</u> , ZHANG Zheng J, THOMSON Angus, ISENBERG Jeffrey	
64	09:27	SIGNAL INHIBITORY REGULATORY PROTEIN-γ; REGULATES GENERATION OF PATHOLOGIC REACTIVE OXYGEN SPECIES IN ACUTE KIDNEY INJURY <u>ROGERS Natasha</u> , AL-GHOULEH Imad, PAGANO Patrick, ISENBERG Jeffrey	

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65	09:39	ASSESSMENT OF THE EFFECT OF FOLLISTATIN TREATMENT AND KINETICS OF ACTIVIN A AND B IN RENAL ISCHEMIA-REPERFUSION INJURY IN MICE <u>FANG Doreen</u> , LU Bo, HAYWARD Susan, de Kretser David, COWAN Peter, DWYER Karen
66	09:51	WITHDRAWN
67	10:03	FACTORS EFFECTING ISLET ISOLATION OUTCOMES OVER THE PAST 15 YEARS FOR THE WESTMEAD ISLET TRANSPLANT PROGRAM <u>CHEW Yi Vee</u> , WILLIAMS Lindy, DAVIES Sussan, LIUWANTARA David, BURNS Heather, HAWKES Joanne, O'CONNELL Philip, HAWTHORNE Wayne <i>— Mini-oral presentations —</i>
68	10:15	INVESTIGATING THE POTENTIAL OF DANTROLENE SODIUM SALT AS A CARDIOPROTECTIVE AGENT DURING ISCHAEMIA-REPERFUSION INJURY. <u>VILLANUEVA Jeanette</u> , GAO Ling, CHEW Hong, HICKS Mark, MACDONALD Peter, JABBOUR Andrew
69	10:21	POST-CONDITIONING WITH CYCLOSPORINE: IMPACT ON ISCHEMIA REPERFUSION INJURY IN A RODENT MODEL OF DONOR HEART PRESERVATION <u>GAO Ling</u> , VILLANUEVA Jeanette, CHEW Hong, HICKS Mark, JABBOUR Andrew, CAO Jacob, MACDONALD Peter
70	10:27	METABOLIC PROFILE OF DCD HEARTS DURING RECONDITIONING <u>CHEW Hong Chee</u> , CAO Jacob, FERNANDEZ Karen, VILLANEUVA Jeanette, GAO Ling, HICKS Mark, JABBOUR Andrew, DHITAL Kumud, MACDONALD Peter

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10:45–11:15	Morning tea	Aerial UTS Function Centre, Foyer
11:15–12:45	PLENARY 3: Astellas Symposium Improvement of Transplant Outcome From Donor to Patient <i>Chairs: A/Prof Daniel Chambers and Dr Monique Malouf</i> 11:15 Long-Term Survival in Living and Kidney Donors A/Prof Dorry Segev 11:45 Organ Perfusion in Preservation in the DCD Setting Mr Gabriel C Oniscu 12:15 Ex-vivo Lung and Heart Perfusion – an Update A/Prof Kumud Dhittal 12:30 Ex-vivo Kidney and Liver Perfusion – an Update Prof Henry Pleass	Aerial UTS Function Centre, Harris Theatre
12:45–13:30	Lunch	Aerial UTS Function Centre, Foyer
12:45–13:30	Lung Advisory Committee Meeting Tolerance Advisory Committee Meeting Vascularised Composite Allotransplantation Working Group Meeting Xenotransplantation Working Group Meeting	Wattle Theatre Thomas Theatre Seminar Room 2 Seminar Room 1

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13:30–15:30	President's Prize Symposium		Aerial UTS Function
	<i>Chairs: Prof Steven Chadban and Prof Stephen Alexander</i>		Centre, Harris Theatre
Abstract	— Oral presentations —		
71	13:30	DELETION OF RECIPIENT CD8+ DENDRITIC CELLS FACILITATES THE PERSISTENCE OF HIGHLY CYTOLYTIC DONOR CTL THAT ELIMINATE LEUKAEMIA AFTER BMT <u>MARKEY Kate</u> , KUNS Rachel, ROBB Renee, KOYAMA Motoko, GARTLAN Kate, HENDEN Andrea, MACDONALD Kelli, BROCKER Thomas, BELZ Gabrielle, LANE Steven, HILL Geoff	
72	13:42	TIME-DEPENDENT CHANGES IN CARDIAC BIOMARKERS AND CARDIAC MAGNETIC RESONANCE IMAGING (CMRI) DETERMINED CARDIAC STRUCTURE AND FUNCTION IN END-STAGE KIDNEY DISEASE (ESKD) AND FOLLOWING RENAL TRANSPLANTATION <u>CROSTHWAITE AA</u> , LIM R, MASTERSON R, HEDLEY A, ROBERTS M, FAROUQUE O, IERINO F	
73	13:54	INNATE ALLO-RECOGNITION RESULTS IN RAPID ACCUMULATION OF MONOCYTE DERIVED DENDRITIC CELLS <u>CHOW Kevin</u> , ZHAN Yifan, SUTHERLAND Robyn, DELCONTE Rebecca, HUNTINGTON Nicholas, LEW Andrew	
74	14:06	RECURRENT GLOMERULONEPHRITIS AND LONG-TERM GRAFT OUTCOMES AFTER KIDNEY TRANSPLANTATION <u>ALLEN Penelope</u> , CRAIG Jonathan, LIM WAI, CHADBAN Steve, ALLEN Richard, WONG Germaine	
75	14:18	THE IMPACT OF DONOR-RECIPIENT AGE MISMATCH ON GRAFT AND PATIENT OUTCOMES AFTER DECEASED DONOR KIDNEY TRANSPLANTATION <u>CALISA Vaishnavi</u> , CRAIG Jonathan, CHADBAN Steve, LIM Wai, HOWARD Kirsten, CHAPMAN Jeremy, MCDONALD Stephen, WONG Germaine	

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76	14:30	CARDIOVASCULAR MAGNETIC RESONANCE NON-INVASIVELY DETECTS CARDIAC TRANSPLANT REJECTION: A PROSPECTIVE, HISTOLOGICALLY-VALIDATED STUDY <u>IMRAN Muhammad</u> , WANG Louis, MCCROHON Jane, YU Chung, HOLLOWAY Cameron, OTTON James, HUANG Justyn, MOFFAT Kirsten, ROSS Joanne, KOTLYAR Eugene, KEOGH Anne, HAYWARD Christopher, MACDONALD Peter, JABBOUR Andrew
77	14:42	COMBINED HEART AND LIVER RETRIEVAL AFTER CIRCULATORY DEATH WITH NORMOTHERMIC MACHINE REPERFUSION IN A PORCINE MODEL <u>CAO Yiming</u> , CHEW HC, FERNANDEZ Karen, VILLANUEVA Jeanette, GAO Ling, HICKS Mark, JABBOUR Andrew, PLEASS Henry, DHITAL Kumud, MACDONALD Peter
78	14:54	THE ADDITION OF COGNITIVE IMPAIRMENT TO PHYSICAL FRAILTY IMPROVES SURVIVAL PREDICTION IN HEART-TRANSPLANT REFERRED PATIENTS <u>JHA Sunita</u> , HANNU Malin, GORE Karen, NEWTON Phillip, HAYWARD Chris, WILHELM Kay, JABBOUR Andrew, KOTLYAR Eugene, KEOGH Anne, DHITAL Kumud, GRANGER Emily, JANSZ Paul, SPRATT Phillip, MONTGOMERY Elyn, HARKESS Michelle, TUNNICLIFF Peta, MACDONALD Peter
79	15:06	AUTOPHAGY-DEPENDENT TIGIT+ REGULATORY T CELLS ARE CRITICAL FOR THE MAINTENANCE OF TOLERANCE AND THE CONTROL OF GRAFT VERSUS HOST DISEASE <u>LE TEXIER LAETITIA</u> , Lineburg Katie E, Leveque-ElMouttie Lucie , Alexander Kylie, Teal Bianca , Melino Michelle , Kuns Rachel D, Lane Steven W, Stephen Blake, Teng Michele , Blazar Bruce R , Clouston Andrew D, Hill Geoffrey R , MacDonald Kelli P.A.
80	15:18	AGREEMENT BETWEEN NUMBER OF DONOR/RECIPIENT EPLET MISMATCHES CALCULATED USING TWO-DIGIT SEROLOGICAL VERSUS FOUR-DIGIT MOLECULAR HUMAN LEUKOCYTE ANTIGEN (HLA)-TYPING <u>FIDLER Samantha</u> , WONG Germaine, LEWIS Joshua, LIM Wai

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15:30–15:45	Afternoon tea	Aerial UTS Function Centre, Foyer
15:45–17:15	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 8: Transplant Complications #2 <i>Chairs: Dr Nick Cross and Dr Monique Malouf</i>	
Abstract	— Oral presentations —	
81	15:45	A PROSPECTIVE STUDY OF RENAL TRANSPLANT RECIPIENTS: THE INTER-RELATIONSHIP BETWEEN INSULIN SECRETION AND SENSITIVITY UNDERPINS DYSGLYCEMIA POST FOLLOWING RENAL TRANSPLANTATION <u>LANGSFORD David</u> , OBEYESEKERE Varuni, TENG Jessie, WARD Glenn, MACISAAC Richard, ALFORD Frank, DWYER Karen
82	15:57	IL-17 REGULATES INTESTINAL DYSBIOSIS AND IS CRITICAL FOR THE PREVENTION OF INTESTINAL GRAFT-VERSUS-HOST DISEASE <u>VARELIAS Antiopi</u> , ORMEROD Kate L, BUNTING Mark D, KOYAMA Motoko, GARTLAN Kate H, ROBB Renee J, ZHANG Ping, KUNS Rachel D, LOCKE Kelly, CLOUSTON Andrew D, HASAIN Sumaira, MCGUCKIN Michael, MacDONALD Kelli PA, HUGENHOLTZ Philip, HILL Geoff R
83	16:09	ASSOCIATION BETWEEN DELAYED GRAFT FUNCTION AND LONG-TERM OUTCOMES AFTER KIDNEY TRANSPLANTATION FROM DONORS AFTER CIRCULATORY DEATH <u>CLAYTON Philip</u> , RUSS Graeme, McDONALD Stephen, CHADBAN Steve
		— Mini-oral presentations —
84	16:21	SHORT TERM SURGICAL COMPLICATION RATES IN TRANSPLANT SURGERY: CONSULTANT VS TRAINEE <u>SALTER Sherry</u> , CHOU Angela, PLEASS Henry, HAWTHORNE Wayne
85	16:27	NEPHROGENIC ADENOMA - A CASE REPORT AND UPDATE <u>NORTH Daniel</u> , JAW Juli, HILL Prue, BATEMAN Samantha, BARRACLOUGH Nick, LANGHAM Robyn

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86	16:33	SCLEROSING PERITONITIS FOLLOWING LIVER TRANSPLANTATION: A CASE SERIES KONG Y, TAN AL, <u>VERRAN DJ</u>	
87	16:39	CIRCULATING DNA: AN APPROACH TO MONITOR ORGAN REJECTION AFTER LIVER TRANSPLANTATION. <u>Goh Su Kah</u> , Do Hongdo, Muralidharan Vijayaragan, Dobrovic Alexander, Christophi Chris	
88	16:45	CELL-FREE DNA CAN IDENTIFY MILD CELL MEDIATED REJECTION IN PAEDIATRIC HEART TRANSPLANT RECIPIENTS <u>WHITLAM JB</u> , LING L, HARRINGTON T, PRAPORSKI S, BRUNO D, POWER D, KONSTANTINOV I, SLATER H	
89	16:51	PYELOURETERIC JUNCTION OBSTRUCTION IN RENAL ALLOGRAFTS <u>GRAJN Andrej</u> , GRIFFIN Anthony D, PRESTON John, WOOD Simon, LAWSON Malcolm	
15:45–17:15	Free Communications 9: From Disease to Donation to the Final Destination – Where is Your Patient on the Transplant Journey? <i>Chairs: Dr Bill Mulley and A/Prof Daniel Chambers</i>		Aerial UTS Function Centre, Thomas Theatre
Abstract	— Oral presentations —		
90	15:45	MARGINAL DONOR HEARTS ASSESSED WITH EX-VIVO PERFUSION TO FACILITATE ORTHOTOPIC HEART TRANSPLANTATION <u>CHAN Samuel</u> , MUDGE David, JOHNSON David, CAMPBELL Scott, FRANCIS Ross	
91	15:57	CHANGE IN DECEASED KIDNEY DONOR CHARACTERISTICS IN AUSTRALIA AND NEW ZEALAND OVER TWENTY YEARS <u>CHAN Samuel</u> , CAMPBELL Scott B, CLAYTON Philip A, MUDGE David W, JOHNSON David W, FRANCIS Ross S	
92	16:09	A NOVEL CARDIAC ALLOCATION SCORE FOR PREDICTING WAIT-LIST AND POST-TRANSPLANT SURVIVAL <u>MARGELIS Stamati</u> , KARAS Pamela, GRANGER Emily, JANSZ Paul, SPRATT Phillip, HAYWARD Christopher, JABBOUR Andrew, KEOGH Anne, KOTLYAR Eugene, MACDONALD Peter, DHITAL Kumud	

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— Mini-oral presentations —

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| 93 | 16:21 | EVALUATION OF A TRANSITION PROGRAM FOR ADOLESCENTS WITH SEVERE LIVER DISEASE/LIVER TRANSPLANT
<u>HARDIKAR Winita</u> , MCCARTHY Jamie, BEYERLE Kathe, CULNANE Evelyn |
| 94 | 16:27 | KIDNEY TRANSPLANT PATIENT PREFERENCES AND TRADE-OFFS FOR OUTCOMES AFTER TRANSPLANTATION.
<u>HOWELL Martin</u> , WONG Germaine, ROSE John, TONG Allison, CRAIG Jonathan, HOWARD Kirsten |
| 95 | 16:33 | TRANSPLANTATION AND DIABETES (TRANSDIAB): A PILOT RANDOMISED CONTROLLED TRIAL OF METFORMIN IN PRE-DIABETES AFTER KIDNEY TRANSPLANTATION
<u>ALNASRALLAH Basil</u> , PILMORE Helen, MANLEY Paul |
| 96 | 16:39 | RESEARCH PRIORITY SETTING IN ORGAN TRANSPLANTATION: A SYSTEMATIC REVIEW
<u>HARPER Claudia</u> , CRAIG Jonathan, CHAPMAN Jeremy, TONG Allison |
| 97 | 16:45 | NEUROCOGNITIVE DEFICITS IN CHILDREN TRANSPLANTED IN EARLY CHILDHOOD
<u>ROBINSON Lucy</u> , KARA Tonya |
| 98 | 16:51 | COGNITIVE AND ACADEMIC OUTCOMES IN CHILDREN WITH CHRONIC KIDNEY DISEASE AND KIDNEY TRANSPLANTS
<u>VAN ZWIETEN Anita</u> , CHEN Kerry, DIDSBURY Madeleine, LORENZO Jennifer, BARTON Belinda, LAH Suncica, CRAIG Jonathan, TONG Allison, HOWARD Kirsten, WONG Germaine |
| 99 | 16:57 | INPATIENT REHABILITATION OF HEART AND LUNG TRANSPLANT PATIENTS A PHYSIOTHERAPY PERSPECTIVE- RETROSPECTIVE ANALYSIS 2011-2015 ST VINCENT'S HOSPITAL, SYDNEY
<u>WOODBIDGE Genevieve</u> |
| 100 | 17:03 | USE OF VISUAL AIDS IN PROMOTING PATIENT UNDERSTANDING OF SURVIVAL WITH TREATMENTS FOR END STAGE KIDNEY DISEASE
<u>DOWEN Frances</u> , PILMORE Helen |

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101	17:09	THE INCIDENCE OF GRAFT PANCREATECTOMY SECONDARY TO THROMBOSIS IN SIMULTANEOUS PANCREAS AND KIDNEY TRANSPLANTATION: A SINGLE-CENTER EXPERIENCE <u>LENDZION Rebecca</u> , SPIKE Erin, YUEN Lawrence, LAM Vincent, RYAN Brendan, PLEASS Henry, HAWTHORNE Wayne	
15:45–17:15		Free Communications 10: Immunosuppression and Trials & Surgical Techniques <i>Chairs: Dr Peter Hughes and A/Prof Andrew Jabbour</i> — <i>Oral presentations</i> —	Aerial UTS Function Centre, Wattle Theatre
102	15:45	ESTIMATION OF MYCOPHENOLIC ACID EXPOSURE POST RENAL TRANSPLANTATION: COMPARISON BETWEEN THE TRAPEZOIDAL METHOD AND MULTIPLE REGRESSION DERIVED LIMITED SAMPLING STRATEGIES. <u>EMMETT CJ</u> , HUGHES Peter, BARRACLOUGH KA	
103	15:57	EVALUATION OF PREVIOUSLY PUBLISHED LIMITED SAMPLING STRATEGIES FOR ENTERIC-COATED MYCOPHENOLATE SODIUM IN ADULT KIDNEY TRANSPLANT RECIPIENTS <u>BROOKS Emily</u> , TETT Susan, ISBEL Nicole, STAATZ Christine	
104	16:09	RETROPERITONEOSCOPIC LIVE DONOR NEPHRECTOMY <u>NG Zi Qin</u> , HE Bulang	
105	16:21	RECOVERY OF KIDNEY GRAFT FUNCTION AFTER TRANSPLANT BY LAPAROSCOPIC TECHNIQUE VS OPEN: A PILOT STUDY <u>He Bulang</u> , MUSK Gabrielle, MOU Lingjun, NG Zi Qin, De ROO Ronald, TAN Daren, DARIAN-SMITH Erica, LUCAS Michaela, DELRIVIERE Luc	
106	16:33	BARRIERS TO EARLY TRANSPLANTATION LISTING FOR PATIENTS UNDER 65 <u>BROWN Megan</u> , POLKINGHORNE Kevan, MANEY Orla, KANELLIS John — <i>Mini-oral presentations</i> —	
107	16:45	A QUALITATIVE REVIEW OF MEDICATION ERRORS MADE BY NEW KIDNEY TRANSPLANT RECIPIENTS <u>FISHER Susan</u> , NALDER Michelle, HUGHES Peter, HOLT Steve	

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108	16:51	SIROLIMUS PRECIPITATES INCISIONAL HERNIA IN TRANSPLANT PATIENTS <u>SHAHRESTANI Sara</u> , TRAN Hanh, PLEASS Henry, HAWTHORNE Wayne
109	16:57	RESTORED KIDNEY TRANSPLANTATION - EXTENDED CRITERIA DONORS <u>SPROTT Philip</u> , TREVILLIAN Paul, HIBBERD Adrian, HEER Munish, STEIN Ann, DAVIDSON Deidre
110	17:03	COMBINATION OF LEFLUNOMIDE AND EVEROLIMUS FOR TREATMENT OF BK VIRUS NEPHROPATHY <u>JAW Juli</u> , GOODMAN David, HILL Prue
19:00–23:00		TSANZ Annual Dinner
		The Great Hall, Sydney University

Tuesday, 12 April 2016

07:30–08:00	Breakfast with sponsors	Aerial UTS Function Centre, Foyer
08:00–09:30	PLENARY 4: Novartis Symposium What's new in Transplant Immunology and Stem Cell Transplantation? <i>Chairs: A/Prof. Alexandra Sharland and Prof. Stephen Alexander</i> 08:00 Liver Transplantation Tolerance Prof Alberto Sanchez-Fueyo 08:30 Stem Cell Therapies for Heart Regeneration Dr James Chong 09:00 Seeking Tolerance by Modifying the Microbiome Prof Steve Chadban	Aerial UTS Function Centre, Harris Theatre
09:30–10:00	Morning tea	Aerial UTS Function Centre, Foyer
10:00–15:00	Liver and Intestinal Advisory Committee Meeting	Aerial UTS Function Centre, Seminar Room 2

Tuesday, 12 April 2016

10:00–11:30	CONCURRENT STATE OF THE ART SESSIONS	
	STATE OF THE ART 1: Astellas Symposium	Aerial UTS Function Centre, Harris Theatre
	Cell Therapies in Transplantation <i>Chairs: Prof Bruce Hall and Prof Karen Dwyer</i>	
	10:00 Prospects for Regulatory T Cell Therapy in Transplant Patients in Australia Prof Geoff Hill	
	10:30 Using Pathogen Specific T Cells From Normal Donors to Treat Opportunistic Infection After Stem Cell and Solid Organ Transplant Prof David Gottlieb	
	11:00 Novel Approaches for T Cell Expansion Using Functionalised Surfaces for Cell Therapy A/Prof Simon Barry	
10:00–11:30	STATE OF THE ART 2: Organ & Tissue Authority Symposium	Aerial UTS Function Centre, Broadway Theatre
	New Treatments in Transplantation <i>Chairs: Prof Steve Chadban and Prof Toby Coates</i>	
	10:00 Shared Decision Making in Cancer Screening and Prevention for Kidney Transplant Recipients: Finding the Sweet Spot A/Prof Germaine Wong	
	10:30 Complement Blockade in Transplantation A/Prof Nikky Isbel	
	11:00 Telehealth: Tools for Increasing Access to Care for Transplant Recipients Dr Scott McKenzie	

Tuesday, 12 April 2016

11:30–13:00	CONCURRENT STATE OF THE ART SESSIONS	Aerial UTS Function Centre, Harris Theatre
	STATE OF THE ART 3: Novartis Symposium	
	Transplantation From Bench to Beside <i>Chairs: Prof Peter Cowan and Dr Kelli MacDonald</i>	
	11:30 Making the Graft Stronger A/Prof Shane Grey	
	11:55 Gene Modified T Cells Expressing a Chimeric Antigen Receptor for Treatment of Multiple Myeloma Dr Ken Micklethwaite	
	12:20 The Three Remaining Steps to Make Xenotransplantation Work A/Prof Wayne Hawthorne	
	12:40 Donor Organ Preservation at sub-Zero Temperatures Prof David McGiffin	
11:30–13:00	STATE OF THE ART 4: Organ & Tissue Authority Symposium	Aerial UTS Function Centre, Broadway Theatre
	What's new in Organ Transplantation? <i>Chairs: A/Prof Nick Shackel and A/Prof Kate Wyburn</i>	
	11:30 Kidney and Liver Transplantation A/Prof Jerome Laurence	
	11:55 Heart Transplantation Prof Peter Macdonald	
	12:20 Kidney and Pancreas Transplantation Prof Henry Pleass	
	12:40 Lung Transplantation Prof Greg Snell	

Tuesday, 12 April 2016

13:00–14:00	Lunch	Aerial UTS Function Centre, Foyer
	Cardiac Advisory Committee Meeting	Wattle Theatre
	Paediatric Working Group Meeting	Thomas Theatre
14:00–15:30	PLENARY 5: Astellas Symposium Solutions and Challenges <i>Chairs: A/Prof Andrew Jabbour and A/Prof Angela Webster</i> 14:00 Donor-Recipient Matching Solution A/Prof Dorry Segev 14:30 Treg Immunotherapy in Transplantation Prof Alberto Sanchez-Fueyo 15:00 Obesity and Transplantation Mr Gabriel Oniscu	Aerial UTS Function Centre, Harris Theatre
15:30–15:45	Afternoon tea	Aerial UTS Function Centre, Foyer
15:45–16:30	The Great Debate: The Future of Transplantation Research - Big Data Beats Small Animals <i>Moderator: Prof Steve Chadban</i> Pro team: A/Prof Dorry Segev / Dr Philip Clayton Con team: Mr Gabriel Oniscu / Prof Karen Dwyer Order decided by coin-toss Team A, speaker 1 Team B, speaker 1 Team A, speaker 2 Team B, speaker 2 Team A rebuttal Team B rebuttal	Aerial UTS Function Centre, Harris Theatre
17:00	ASM Concludes	

SUNDAY ABSTRACTS

IN SESSION ORDER

Outcome measures #1

Abstract No. 1

RECIPIENT AND PANCREAS GRAFT SURVIVAL AFTER KIDNEY-PANCREAS TRANSPLANTATION IN AUSTRALIA AND NEW ZEALAND: A COHORT STUDY 1984-2014

WEBSTER Angela^{1,2}, PENG Xi (Alex)², KELLY Patrick², ANZIPTR On behalf of³

¹Centre for Transplant and Renal Research, Westmead Hospital, Sydney, ²School of Public Health, University of Sydney, ³contributors, Australia and New Zealand Islet and Pancreas Transplant Registry

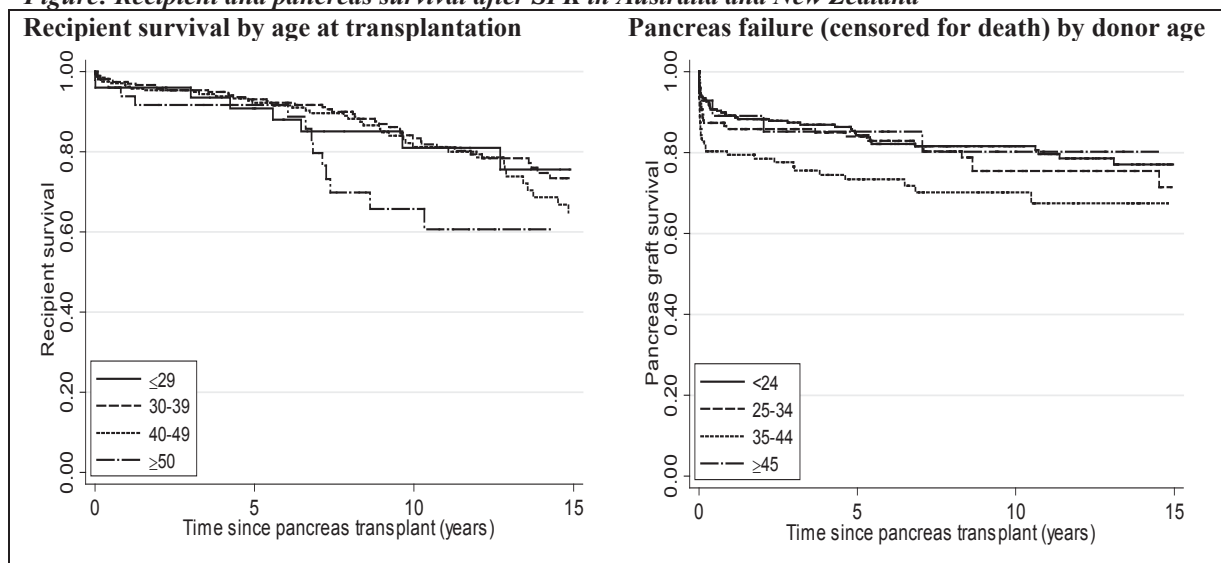
Introduction: We evaluated survival for kidney-pancreas recipients (SPK) in Australia and New Zealand.

Methods: Data 1984-2014 from the Australia and New Zealand Islet and Pancreas Transplant Registry were used to analyse time to pancreas failure (first of; pancreatectomy, insulin-dependence, with and without death) or death (all-cause), using Kaplan-Meier survival curves, censoring at last follow-up. We used Cox models (Hazard ratios HR, with 95%CI) to identify prognostic factors.

Results: We included 627 recipients, with 5,370 years of observation, 119 (19%) deaths and 214 (34%) pancreas failures. Patient survival was 97% at 1 year, 93% 5 years, 81% 10 years, 69% 15 years and 64% 20 years (figure). After adjusting for other differences, risk of dying decreased by 48% for people receiving SPK in 2010-2014 compared to 1989-1994 (HR0.52; $p<0.01$). Recipient age increased risk of death 4% for every year older at transplantation (HR1.04; $p=0.04$). There was no evidence of increased risk with any other factors ($p>0.05$). Pancreas survival was 84% at 1 year, 76% 5 years, 64% 10 years, 56% 15 years and 50% 20 years. Pancreas failure decreased 40% between 1989-1994 and 2010-2014 (HR0.60; $p<0.02$). After adjusting for other differences, risk of pancreas failure increased by 2% for every year of donor age (HR1.02; $p=0.03$). There was some suggestion that longer time on RRT associated with higher risk of pancreas failure ($p=0.08$).

Conclusion: There has been substantial improvement in patient survival and a substantial reduction in the risk of pancreas failure since SPK first began in ANZ.

Figure: Recipient and pancreas survival after SPK in Australia and New Zealand



Abstract No. 2

RETROSPECTIVE SINGLE CENTRE COMPARISON OF OUTCOMES BETWEEN NORMAL CRITERIA AND MARGINAL CRITERIA BRAIN DEAD HEART TRANSPLANTATION

CHEW Hong Chee¹, LO Phillip², CAO Jacob³, SUGIANTO Nara², DHITAL Kumud¹, GRANGER Emily¹, HAYWARD Christopher⁴, JABBOUR Andrew⁴, JANSZ Paul¹, KEOGH Anne⁴, KOTLYAR Eugene⁴, SPRATT Phillip¹, MACDONALD Peter^{4,3}

¹Department of Cardiothoracic Surgery, St Vincent's Hospital, Sydney, ²School of Medicine, University of New South Wales, Sydney, ³Transplantation Laboratory, Victor Chang Cardiac Research Institute, Sydney,

⁴Department of Cardiology, St Vincent's Hospital, Sydney

Aims: Donor left ventricular dysfunction, high inotropic requirement and old age have previously precluded heart donations from being used for transplantation. However, shortage of donor organs, as well as improvements in donor medical management, organ preservation and surgical techniques and interventions, has led to the re-consideration of these marginal organs. We compare the post transplantation outcomes of standard criteria (SD) and marginal brain dead (MBD) heart transplantation in our unit. **Methods:** All heart transplant recipients from January 2000 until December 2013 were included. MBD donors (n=108) were defined by LV dysfunction (EF<50%), high inotrope (norepinephrine) requirement >0.2mcg/kg and age >50years versus SD (n=195). Donor data and recipient medical records were used to identify key outcomes and survival data.

Results: Key demographics (height, weight and medical history) were comparable between both groups apart from significantly older donor age in the MBD group. Other key donor characteristics including LV dysfunction and ischaemic time were significantly higher in the MBD group. Overall patient survival was comparable between BD and MBD at 1, 3, 5 and 10 years (90%, 87%, 80%, 69% vs. 84%, 80%, 72%, 60%; p=0.148 Kaplan-Meier analysis). Other outcomes are presented in the table.

Conclusion: Current experience with MBD heart transplantation shows a higher risk of early post transplantation graft dysfunction in the MBD group requiring short term mechanical support and longer ICU stay. However, short to long term survival trends remain favourable when comparing MBD to SD heart transplants.

	<i>SD</i>	<i>MBD</i>	<i>P</i>
<i>Age</i>	33	48	n/a
<i>Ischaemic Time</i>	213 ± 42 min	266 ± 67 min	0.0005
<i>LV Dysfunction</i>	0% (0/195)	11% (12/108)	0.000002
<i>BPT (bypass time)</i>	168 ± 48 min	182 ± 41 min	0.02
<i>Mechanical Support</i>	21%	31.5%	0.04
<i>ECMO/ IABP</i>	7.2%/ 14.4%	18.5%/19.4%	0.002
<i>Return to OT</i>	23.6%	25%	0.8
<i>ICU LOS</i>	7 ± 5days	11 ± 9days	0.0005
<i>Hospital LOS</i>	27 ± 16days	29 ± 17days	0.02

Abstract No. 3

OUTCOMES FOLLOWING TRANSFER OF PAEDIATRIC LIVER TRANSPLANT RECIPIENTS TO ADULT HEALTHCARE IN VICTORIA**SRINIVASAN Ashish¹, APOSTOLOV Ross¹, LEONG Amanda¹, TESTRO Adam¹, JONES Robert¹, HARDIKAR Winita²**¹*Liver Transplant Unit Victoria, Austin Hospital, Melbourne,* ²*Liver Transplant Unit Victoria, Royal Children's Hospital, Melbourne***Introduction:** Transition of children with chronic health issues to adult medical care is often associated with poor outcomes, stemming from non-compliance and psychosocial stressors.**Aims:** This study aims to document health related outcomes within the Victorian paediatric liver transplant population, in the period following transition to adult care at Austin Health.**Methods:** A retrospective search of the Victorian Liver Transplant Unit database and patient clinical records was performed to identify paediatric liver transplant recipients under the age of 18 as at 1st January 2000, who had been subsequently transitioned to Austin Health, and followed-up for a period of at least 12 months. Key outcome measures included medical non-adherence, chronic rejection, healthcare disengagement, evidence of 'at risk' behaviour, and active mental health issues. Non-adherence was defined as undetectable immunosuppressive serum drug levels or self-reported medication non-compliance.**Results:** Of the 47 patients who met the inclusion criteria, 25 (53%) were adherent to medical therapy (Table 1). Rates of chronic rejection were 12% and 50% across the adherent and non-adherent groups respectively ($p=0.01$), with a greater proportion of mental health issues noted amongst non-adherent patients ($p=0.02$). Adherent patients were more likely to have higher rates of ongoing healthcare engagement, and miss fewer clinic appointments.**Conclusions:** A significant portion of paediatric liver transplant recipients transitioned to adult follow up were non-adherent with medical therapy, predisposing them to poorer health and transplant related outcomes. This suggests that our current model of transition needs to be improved.**Table 1** Demographic data and health outcomes

	<u>Adherent</u>		<u>Non Adherent</u>		<u>p-value</u>
Patients	25	53%	22	47%	
Australian Born	24	96%	21	95%	1.00
Male	12	48%	12	55%	0.77
Median Age at Transplant	9.27	(1-19)	8.58	(0-19)	1.00
Median Age at Transfer	17.73	(7-20)	17.68	(9-21)	1.00
Chronic Rejection	3	12%	11	50%	0.01
Graft Loss	1	4%	1	5%	1.00
Death	1	4%	1	5%	1.00
Healthcare Disengagement*	0	0%	6	27%	0.01
Missed >3 Clinic Appointments	8	32%	16	73%	0.01
Smoking	5	20%	9	41%	0.20
Alcohol	2	8%	4	18%	0.39
Mental Health Issues	6	24%	13	59%	0.02

*Consistent non-attendance at clinic for at least 12 months

Abstract No. 4

ASSOCIATION BETWEEN DELAYED GRAFT FUNCTION (DGF) AND GRAFT LOSS IN DONATION AFTER CARDIAC DEATH (DCD) DONOR KIDNEY TRANSPLANTS – A PAIRED KIDNEY ANALYSIS

LIM Wai¹, MCDONALD Stephen², CHAPMAN Jeremy³, PLEASS Henry³, JAQUES Bryon¹, WONG Germaine³

¹Sir Charles Gairdner Hospital, Perth, ²Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, ³Westmead Millennium Institute, Westmead Hospital, Sydney

Background: Previous epidemiological studies have suggested that DGF is a risk factor for long-term graft loss in brain-dead donor kidney transplants but not DCD kidney transplants.

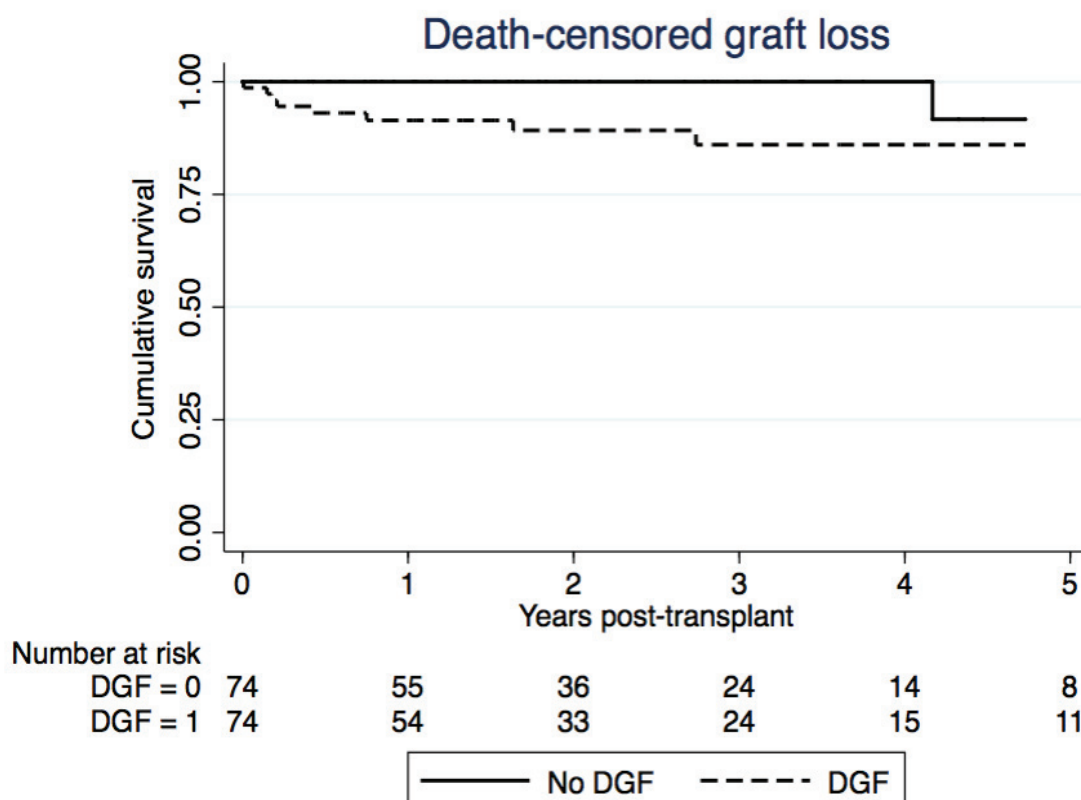
Aim: We aimed to determine the association between DGF and long-term overall and death-censored graft loss (DCGL) in DCD kidney transplants using the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry.

Methods: Of the 201 paired DCD kidney transplant recipients identified between 1994-2012, 74 pairs were included because of differences in the presence of DGF (i.e. only one of the two recipients from the same donor experienced DGF, defined as requiring dialysis after transplantation). Associations between DGF and overall and DCGL were examined using adjusted Cox regression models.

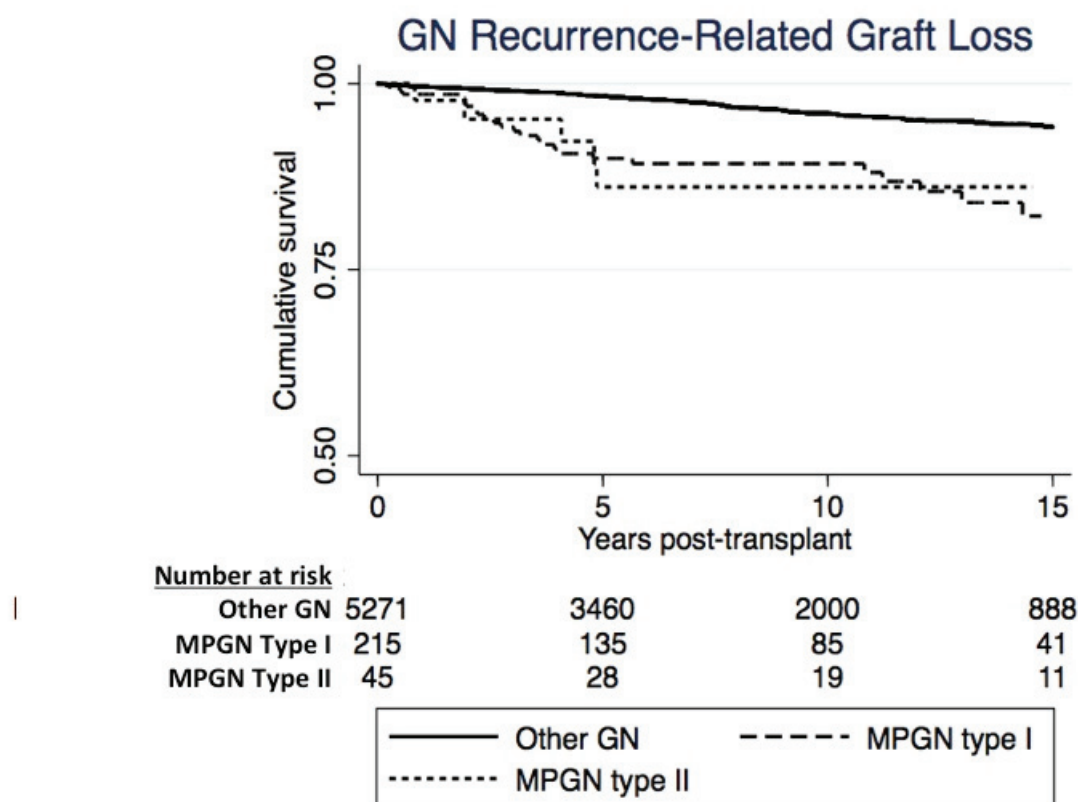
Results: Of the 74 DCD donors, 50 (68%) were males with mean (SD) age of 45.2 (16.3) years. Recipients who had experienced DGF were of similar age and sensitization status compared to those who did not experienced DGF. A greater proportion with DGF experienced DCGL (14% vs. 3%, $p=0.016$). After adjusting for model covariates, DGF was associated with an increased risk of overall and DCGL at 5-years with adjusted hazard ratios of 2.82 (95%CI 0.91, 8.76) and 18.62 (95%CI 2.06, 168.29) respectively. Adjusted cumulative incidence curves for DCGL are shown in Figure 1.

Conclusions: DGF in DCD kidneys transplants is an independent risk factor for early graft loss after kidney transplantation.

Figure 1.



Abstract No. 5

LONG-TERM GRAFT OUTCOMES OF KIDNEY TRANSPLANT RECIPIENTS WITH MEMBRANO-PROLIFERATIVE GLOMERULONEPHRITIS (MPGN)**LIM Wai¹, WONG Germaine², PILMORE Helen³, MULLEY William⁴, WALKER Rowan⁵, MENAHEM Solomon⁵**¹*Sir Charles Gairdner Hospital, Perth*, ²*Westmead Millennium Institute, Westmead Hospital, Sydney*, ³*Auckland Renal Transplant Group, Auckland City Hospital*, ⁴*Monash Medical Centre, Melbourne*, ⁵*Alfred Hospital, Melbourne***Aim:** We aimed to determine the association between types of MPGN and long-term graft and patient outcomes using the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry.**Methods:** Primary live and deceased-donor kidney transplant recipients between 1990-2012 whose end-stage renal disease was attributed to glomerulonephritis were included. Associations between types of MPGN, acute rejection, all-cause mortality, overall and death-censored graft loss (DCGL) were examined using adjusted logistic and Cox regression models.**Results:** Of 5532 kidney transplant recipients followed for a median (IQR) of 8.0 (3.7-13.1) years resulting in 48,692 person-years, 215 (3.9%) and 45 (0.8%) have type I and II MPGN respectively. Recipients with type I and II MPGN were older with mean age of 39.8 and 35.8 years compared to those with non-MPGN (43.7 years, $p < 0.001$). The incidence of overall (50%, 44% and 34% respectively, $p < 0.001$) and DCGL (42%, 38% and 22% respectively, $p < 0.001$) were higher in recipients with type I and II MPGN compared to non-MPGN. Compared to recipients with non-MPGN, the adjusted hazard ratios for overall graft loss for recipients with type I and II MPGN were 1.59 (95%CI 1.22, 2.08) and 1.63 (95%CI 0.99, 2.68) respectively; and were 2.10 (95%CI 1.57, 2.81) and 1.86 (95%CI 1.09, 3.17) for DCGL respectively. Graft loss attributed to recurrent MPGN in recipients with type I and II MPGN was 19% and 25%, with adjusted cumulative survival curves shown in Figure 1. There were no associations between MPGN, acute rejection and mortality.**Conclusions:** Recipients with MPGN have poorer graft survivals with nearly one-quarter of recipients experiencing graft loss secondary to recurrent disease after a median of between 3-4 years.**Figure 1.**

Abstract No. 6

SEASONAL VARIATION IN KIDNEY TRANSPLANT OUTCOMES

LIM Wai¹, WONG Germaine²¹Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth, ²Westmead Millennium Institute, Westmead Hospital, Sydney

Background: Viral infection has been shown to be a risk factor for acute rejection after kidney transplantation. On epidemiological grounds, these results may support the hypothesis that winter months, which are associated with a higher risk of viral infections, are implicated in acute rejection. The aim of this study is to examine the relationship between seasonal changes and risk of acute rejection after kidney transplantation using ANZDATA registry.

Methods: Primary live and deceased donor kidney transplant recipients between 2004-2012 were included in this study. Association between seasons and acute rejection was examined using adjusted logistic and Cox regression analyses.

Results: Of 6108 kidney transplants performed, 1672 (27.4%), 1599 (26.2%), 1563 (25.6%) and 1274 (20.9%) were transplanted in the spring (September to November), autumn (March to May), winter (June to August) and summer (December to February) months respectively. A greater proportion of deceased donor transplants occurred in the summer compared to winter months ($p=0.037$). For early rejection that occurred in the first 6 months after transplant and multiple rejections, transplants that occurred in the winter months were associated with higher risks compared to those that occurred in spring with adjusted odds ratios of 1.30 (95%CI 1.09, 1.56, $p=0.004$) and 1.38 (95%CI 1.05, 1.82, $p=0.023$) respectively. Compared to spring, the adjusted hazard ratio for any rejection of transplants that occurred during winter months were 1.20 (95%CI 1.04, 1.38, $p=0.011$). There were no associations between seasons and risk of late rejection or types of rejection.

Conclusion: There appears to be a seasonal variation in the risk of acute rejection after kidney transplantation, independent of age, initial immunosuppression and sensitization status. It remains unknown whether this observation is an epiphenomenon or reflects the higher incidence of viral and other infections and future studies exploring the biological rationale of this association is required.

Abstract No. 7

REHABILITATION OUTCOMES FOLLOWING CARDIOPULMONARY TRANSPLANTATION AND INPATIENT REHABILITATION

SKALICKY David¹, BOWMAN Malcolm², WU Jane³, WOODBRIDGE Genevieve¹, THOMPSON-BUTEL Angelica¹, FAUX Steven^{4,5}¹Rehabilitation Medicine, St Vincent's Hospital, Sydney, ²Dept Rehabilitation Medicine, St Joseph's Hospital, Auburn, ³Other, St Vincent's Hospital, Sydney, ⁴Dept of Rehabilitation Medicine, St Vincent's Hospital, Sydney, ⁵Other, University of New South Wales, Sydney

Background and Aim: For today's transplant recipients, the median survival for heart transplant is 15 years, and for lung transplant 8 years (ANZCOTR data). This study aims to outline rehabilitation outcome data for this unique cohort of inpatients from 2004-2009, and to provide updated rehabilitation outcome data for the last 5 years (2009-2014).

Methods: From 2009-2014, there were 86 recipients of cardiopulmonary transplantation (32 heart and 54 lung) who completed inpatient rehabilitation programs at Sacred Heart Rehabilitation Services, St Vincent's Hospital, Sydney. This represents 20% of the total number of transplant recipients treated under the Heart/Lung program at St Vincent's Hospital. This retrospective audit examines rehabilitation outcome data for these patients. Trends regarding length of stay (LOS), complication rates and Functional Independence Measure (FIM) change will be presented showing variation in outcomes achieved.

Results: Results from the first cohort are discussed in Bowman 2013 (1). From 2009-2014, there were 32 patients with heart transplant, 37 rehabilitation admissions, an average LOS of 26 ± 26 days (mean \pm standard deviation), admission FIM of 83 ± 18 and discharge FIM of 109 ± 20 . There were 54 patients with lung transplant, 70 rehabilitation admissions, an average LOS of 22 ± 15 days, admission FIM of 81 ± 16 and discharge FIM of 98 ± 26 .

Discussion: Our inpatient program is uniquely designed to provide multidisciplinary rehabilitation to a population who require intense medical and surgical monitoring for rejection and side effects of anti-rejection drugs. Transplant recipients had similar LOS to the average LOS (23.4 days) at St Vincent's Rehabilitation services, and represent 5-10% of the total inpatient rehabilitation admissions. Complexities in managing these patients include monitoring for complications of high levels of pharmacological immunosuppression, the management of chronotropic incompetence of the denervated heart and the psychological sequelae of transplantation.

Reference

1. Bowman M, Odelli R, Woodbridge G and Faux SG, Outcomes of an Inpatient Rehabilitation Program Following Complicated Cardio-Pulmonary Transplantation, Int J Phys Med Rehabil 2013, 1:6

Abstract No. 8

LIVING KIDNEY DONOR PRIORITIES FOR OUTCOMES: A NOMINAL GROUP TECHNIQUE STUDY

HANSON CS^{1,2}, KANELIS J^{3,4}, CHADBAN SJ^{5,6}, CHAPMAN JR⁷, CRAIG JC^{6,2}, WONG G^{1,2}, PINTER J², GARG AX⁸, GILL JS⁹, LEWIS JR^{1,2}, TONG A^{1,2}

¹*School of Public Health, University of Sydney*, ²*Centre for Kidney Research, The Children's Hospital at Westmead, Sydney*, ³*Department of Medicine, Monash University, Melbourne*, ⁴*Department of Nephrology, Monash Medical Centre, Melbourne*, ⁵*Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney*, ⁶*School of Medicine, University of Sydney*, ⁷*Centre for Transplant and Renal Research, Westmead Hospital, Sydney*, ⁸*Division of Nephrology, Western University*, ⁹*Division of Nephrology, University of British Columbia*

Background: Risks posed to living kidney donors are ethically justified with informed consent, screening and follow-up care, but the outcomes that are most important to donors are yet to be established. We aimed to identify living kidney donor's priorities for outcomes and describe the reasons for their choices.

Methods: Living kidney donors were purposively sampled from two Australian transplant centres. Participants identified important outcomes of kidney donation, ranked their importance, and discussed reasons for their priorities. For each outcome, we calculated a mean rank score from zero (least important) to 10 (most important) and analysed the transcripts thematically.

Results: Across 8 nominal groups, 67 participants aged 27-78 years identified 32 outcomes. The highest ranked outcomes were: time to recovery (mean rank score 5.39, SD = 3.77), family life (5.24, SD = 4.02), donor-recipient relationship (4.25, SD = 4.07), diet and lifestyle restrictions (3.90, SD = 3.55), and kidney function (3.76, SD = 3.50). Kidney failure and mortality ranked 10th and 13th respectively. Women ranked the donor-recipient relationship, life satisfaction, and family life higher than men; whilst men ranked kidney failure, physical function, mortality and kidney function higher. The themes underpinning participants' priorities included: overriding concern for recipient wellbeing, undeterred by low risks, heightened susceptibility and unfulfilled expectations.

Conclusions: Donors prioritised outcomes that could potentially disrupt their lifestyle and relationships, were unexpected, or threatened their health. Assessment and follow-up should address expectations regarding recovery time, relationship challenges and lifestyle restrictions; and donors may feel empowered with advice to prevent long-term health problems.

Abstract No. 9

CIRCULATING SOLUBLE-KLOTHO LEVELS MODESTLY INCREASE AFTER RENAL TRANSPLANTATION

TAN Sven-Jean^{1,2}, **CROSTHWAITE Amy**^{2,3}, **LANGSFORD David**⁴, **OBEYSEKERE Varuni**⁵, **IERINO Frank L**^{2,3,6}, **ROBERTS Matthew A**^{6,7}, **HOLT Stephen G**^{1,2}, **HEWITSON Timothy D**^{1,2}, **DWYER Karen M**^{6,8,9}, **TOUSSAINT Nigel D**^{1,2}

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³Department of Nephrology, Austin Hospital, Melbourne, ⁴Department of Nephrology, The Northern Hospital,

Melbourne, VIC, ⁵Department of Endocrinology, St Vincent's Hospital, Melbourne, ⁶Victorian Kidney

Transplantation Collaborative, ⁷Department of Renal Medicine, Eastern Health, Box Hill Hospital, VIC,

⁸Department of Nephrology, St Vincent's Hospital, Melbourne, ⁹School of Medicine, Deakin University

Background: Klotho, a co-receptor for FGF23, is predominantly expressed in kidney and reported to have anti-oxidant and anti-fibrotic properties. Soluble-Klotho (sKl), the circulating protein cleaved from membrane-bound Klotho, declines dramatically with kidney disease and is inversely associated with mortality. sKl has not been thoroughly evaluated prospectively after kidney transplantation.

Aim: To evaluate change in sKl over 12 months following kidney transplantation.

Methods: Incident kidney transplant recipients (KTRs) were recruited with blood sampled at 4 time-points; pre-transplantation (baseline), 1-week (1w), 12-weeks (12w) and 52-weeks (52w) post transplantation. Samples were assayed for basic biochemistry and sKl (IBL, Japan). Within-subject comparisons were evaluated using repeat-measure ANOVA or Friedman's analysis.

Results: Samples from 29 KTRs were available for final analysis. Median KTR age was 49 (35-55) years. Seventeen (59%) were male, 26 (90%) were living kidney allografts and 10 (34%) were pre-emptive. Table 1 summarises the change across the measured parameters. Compared with baseline, sKl exhibited an increase at 52w following initial decline at 1w ($p < 0.005$ and $p < 0.01$ respectively).

Conclusions: This prospective study demonstrated modest sKl increase post kidney transplantation despite excellent graft function achieved, suggesting factors beyond renal capacity influencing circulating sKl. Longer-term evaluation and investigation specifically addressing effects of immunosuppression on sKl are required to understand and modify the potential protective properties of sKl.

Table 1. Change in mineral metabolism parameters subsequent to kidney transplantation (n=29).

Parameter	Baseline	1-week	12-weeks	52-weeks
sKlotho, pg/mL	307 (279-460)	273 (246-343) ^b	352 (286-417) ^c	460 (311-525) ^{a,c}
Serum phosphate (sPi), mmol/L	1.78 ± 0.5	0.81 ± 0.29 ^a	0.88 ± 0.19 ^a	0.92 ± 0.17 ^a
Serum calcium (sCa), mmol/L	2.40 ± 0.18	2.37 ± 0.20	2.46 ± 0.13	2.44 ± 0.13
Serum albumin, g/L	36 ± 4	31 ± 3 ^a	38 ± 3 ^c	38 ± 4 ^{b,c}
Serum creatinine (sCr), umol/L	638 (537-722)	113 (92-142) ^a	112 (99-130) ^a	111 (97-131) ^a
eGFR, mL/min/1.73m ²	7.4 (6.5-8.7)	63.2 (46.5-87.4) ^a	61.2 (51.7-71.9) ^a	60.4 (50.5-71.6) ^a

Data presented as Mean ± SD or Median (IQR). Friedman test with Dunn's multiple comparisons performed for non-parametric values and repeat measures ANOVA performed for parametric values. ^a $p < 0.005$ compared with baseline. ^b $p \leq 0.01$ compared with baseline. ^c $p < 0.005$ compared with 7-day.

Abstract No. 10**SCOPE AND HETEROGENEITY OF OUTCOMES REPORTED IN COCHRANE SYSTEMATIC REVIEWS OF KIDNEY TRANSPLANTATION****SAUTENET Benedicte^{1,2,3}, CRAIG Jonathan C^{1,2}, EVANGELIDIS Nicole^{1,2}, CHAPMAN Jeremy R⁴, GILL John⁵, WONG Germaine^{1,2,4}, TONG Allison^{1,2}**¹*School of Public Health, University of Sydney*, ²*Centre for Kidney Research, Westmead Hospital, Sydney*,³*University Francois Rabelais, Tours, France*, ⁴*Centre for Transplant and Renal Research, Westmead Hospital, Sydney*, ⁵*Department of Nephrology, University of British Columbia, Vancouver, Canada*

Aims: The heterogeneity and bias in selecting and reporting outcomes can limit the relevance and utility of systematic reviews (SRs) in informing shared decision-making. We aimed to assess the scope and consistency of outcomes reported in SRs for kidney transplant recipients.

Methods: The Cochrane Database of SRs was searched to November 2015 for published SRs of all interventions for kidney transplant recipients. All outcomes were extracted and clustered into domains, and the frequency of outcomes reported across all SRs was assessed.

Results: 30 SRs with 422 trials reported 1115 outcomes that clustered in 35 outcome domains. Only five outcome domains were reported in at least half of the SRs: mortality (29 SRs [97%]), graft function (25 [83%]), graft loss (24[80%]), graft rejection (17[57%]) and infection (15[50%]). The next three most frequently reported outcomes were cancer (14[47%]), cardiovascular diseases (13[43%]) and lipids (10[33%]). Patient-reported outcomes including mental health, health status, sleep, pain, physical function, were seldom reported (<20% of SRs). There was substantial variability in the tests, timing, and thresholds used to define and measure the outcomes.

Conclusions: Mortality and graft outcomes are frequently reported in Cochrane SRs of kidney transplantation, whereas other patient-centred outcomes including psychosocial status, mental and physical function are uncommon. These findings presumably reflect the outcomes reported in the corresponding trials. A standardised set of core outcomes based on the shared priorities of patients and health professionals in kidney transplantation may help maximise the value of SRs to inform clinical decision-making.

Immunobiology: Tolerance and TregsAbstract No. 11**MICROBIAL CONCORDANCE LEADS TO SKEWING OF THE LUNG ALLOGRAFT MACROPHAGE PHENOTYPE FROM PRO-FIBROTIC M2 TOWARDS TOLEROGENIC M1****NELLES Ricky^{1,2}, YERKOVICH Stephanie^{2,1}, HOPKINS Peter^{2,1}, CHAMBERS Daniel^{2,1}**¹*School of Medicine, University of Queensland, Brisbane, ²Lung Transplant Service, Prince Charles Hospital, Brisbane*

Aims: Chronic lung allograft dysfunction (CLAD) remains one of the major barriers for long-term survival following lung transplantation. We previously found in cystic fibrosis lung transplant recipients that re-colonization of the allograft (concordance) with *Pseudomonas* was not associated with CLAD, while *de novo* acquisition (discordance) was associated with CLAD, however the mechanism(s) remain unknown. In this study we investigated the possibility that a concordant microbiome might induce tolerogenic innate immune machinery, favouring graft survival.

Methods: This retrospective, cross-sectional study was performed on stored bronchoalveolar lavage (BAL) samples. Pre- and post-transplant bacterial cultures were used to classify patients into concordant or discordant microbiome groups. BAL cellularity was determined. Markers of innate immune activation (mannose-binding lectin (MBL), IL-1 β , IL-6, IL-8, IL-10, TNF α , TGF- β), M1 (TNF α , IL-1 β , IL-6, NOS, ICAM-1) and M2 (IL-10, arginase, TGF- β , CD36, macrophage scavenger receptor 1 (MSR1)) macrophage subtypes were assayed.

Results: There was no association between any patient demographic (time post-transplant, age, pre-transplant diagnosis) and any outcome measure. There was no association between any innate immune marker or BAL cellularity and microbial concordance ($p > 0.05$). A concordant microbiome was associated with increased levels of ICAM-1 (Odds ratio (95% CI), 1.81 (1.12 – 2.93), $p = 0.016$), TNF α (2.41 (1.33 – 4.35), $p = 0.004$), and NOS2 (1.25 (1.02 – 1.53), $p = 0.036$), all markers associated with an M1 macrophage phenotype, and IL-10 (1.81 (1.16 – 2.83), $p = 0.009$), an M2 macrophage associated marker.

Conclusions: Microbial concordance was associated with a skew towards an M1 macrophage pattern. These observations are consistent with the idea that macrophage polarisation, induced by the microbiome, may lead to skewing away from the pro-fibrotic M2 phenotype, hence limiting immune injury, airway fibrosis and CLAD development.

Abstract No. 12**ROLE OF MHC CLASS I MOLECULES AND CO-STIMULATORY MOLECULES IN RODENT MHC MISMATCH SKIN TRANSPLANT MODELS****LEONG Mario¹, PAUL Moumita², MOUAWADH Mamdoh², CUNNINGHAM Eithne², TAY Szun Szun², WANG Chuanmin², BERTOLINO Patrick², BOWEN David², BISHOP Alex², ALEXANDER Ian², SHARLAND Alexandra²**¹*School of Medicine, University of Sydney, ²Department of Surgery, University of Sydney*

Background/Aims: Expression of allogeneic MHC class I by recipient hepatocytes following inoculation with a liver specific AAV vector results in tolerance to subsequent skin grafts expressing the same mismatched MHC allele. Tolerance may result from direct recognition of intact allogeneic MHC on hepatocyte surface or from indirect recognition of processed peptides. D227K mutant class I molecules cannot be directly recognized by majority of alloreactive T-cells. Hepatocytes are non-professional antigen presenting cells and lack expression of co-stimulatory molecules. It is postulated that CD86 expression may negate their ability to induce tolerance in alloreactive CD8+ T-cells.

Methods: C57BL/6 mice were inoculated with AAV-Kd doses ranging from 5×10^9 to 5×10^{11} vgc or AAV-D227K-Kd at 5×10^{11} vgc. Some mice also received AAV-CD86. All mice then received Kd skin grafts. Skin graft survival, liver inflammation and Kd expression were monitored.

Results: There was dose-dependent prolongation of survival of Kd skin grafts, culminating in indefinite survival for all grafts at 5×10^{11} vgc. Inoculation with AAV-D227Kd improved median graft survival from 15 to 29 days. Co-expressing CD86 abrogates tolerance induction (MST 20 days) and was accompanied by significant hepatic inflammation and CD8+ infiltration. By the time of graft rejection, Kd expression had been lost in most hepatocytes, in contrast to tolerance where it persists long term.

Conclusions: Dose-dependent prolongation of Kd-mismatched skin graft survival was achieved by administration of AAV-Kd in C57BL/6 mice. Tolerance was not induced in the absence of direct MHC class I recognition and was abrogated when CD86 was co-expressed on hepatocytes.

Abstract No. 13

STIMULATION WITH ANTIGEN AND CYTOKINES INDUCE EXPRESSION IRF4 IN NAÏVE CD4⁺CD25⁺ T REGULATORY CELLS**WILCOX Paul, L.¹, ROBINSON Catherine, M.¹, TRAN Giang, T.¹, VERMA Nirupama, D.¹, HALL Bruce M.^{2,4}, HODGKINSON Suzanne, J.^{2,3}**¹*Immune Tolerance Laboratory, University of New South Wales, Sydney*, ²*Department of Medicine, University of New South Wales, Sydney*, ³*Department of Neurology, Liverpool Hospital*, ⁴*Department of Renal Medicine, Liverpool Hospital*

We have previously shown that enriched naïve CD4⁺CD8⁻CD25⁺FOXP3⁺ Treg cultured for 3-4 day with alloantigen and IL-2 generate antigen-specific Treg. 10-30% are induced to express CD8 as well as CD4, and these are thought to be the cells stimulated by their TCR recognizing alloantigen.

Aim: We examined if the transcription factor IRF4, which is induced by antigen binding to TCR, was increased in these antigen specific Treg.

Method: Naïve CD4⁺CD8⁻CD25⁺Foxp3⁺ Treg were prepared from normal DA rats, and cultured for 4 days with PVG alloantigen and IL-2. Cultured cells separated to CD8⁺ and CD8⁻ were analyzed by FACS, RT-PCR, and for capacity to suppress *in vivo* and in MLC.

Results: These cultures induce expression of IFN- γ R, and the cells have enhanced capacity to suppress both proliferation in MLC at 1:32-1:64 and graft rejection *in vivo* to induce tolerance. The CD4⁺CD8⁺CD25⁺FOXP3⁺ subpopulation suppress MLC in an Ag-specific manner at ratios of 1:1056, while CD4⁺CD8⁻CD25⁺ cells showed no increase in potency. These CD8⁺ cells are essential to their capacity to suppress rejection and induce tolerance. This suggests CD8⁺ cells recognized alloantigen.

IRF4 was not detected in naïve CD4⁺CD8⁻CD25⁺Treg, yet was induced by culture; IRF4 expression was much greater in the CD4⁺CD8⁺ cells than the CD4⁺CD8⁻. Similar induction of IRF4 has been observed in naïve Treg cultured with antigen and IL-4.

Conclusion: Our data are consistent with the hypothesis that the CD4⁺CD8⁺ cells are the activated Ag-specific T_H1 cells, and induction of IRF4 may be a marker of antigen activated Treg.

Abstract No. 14

TARGETED MODIFICATION OF DC PHENOTYPE AND FUNCTION WITH POROUS SILICON NANOPARTICLES.**STEAD Sebastian^{1,2}, MCINNES Steven³, KIRETA Svjetlana², ROSE Peter¹, ROJAS-CANALES Darling², JESUDASON Shilpa², GREY Shane⁴, CARROLL Robert², VOELCKER Nico³, COATES Toby^{2,1}**¹*School of Medicine, University of Adelaide*, ²*Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital*, ³*Future Industries Institute, University of South Australia*, ⁴*Transplantation Immunology Laboratory, Garvan Institute of Medical Research, Sydney*

Aims: Dendritic cells (DC) are the most potent antigen-presenting cell and are fundamental in the establishment of transplant tolerance. Targeting DC via the DC-SIGN receptor is a potential target for cell specific therapy. Porous silicon nanoparticles (pSiNP) loaded with immunosuppressant rapamycin (RAPA-pSiNP) provides a unique platform to target and modify DC *in vivo*. The aim of this study was to conjugate monoclonal antibody anti-DC-SIGN on to the surface of RAPA-pSiNP and determine the effects on targeted DC phenotype and stimulatory capacity *in vitro*.

Methods: Fluorescein isothiocyanate (FITC)-labelled pSiNP conjugated to either anti-DC-SIGN or isotype control were cultured with whole blood samples *in vitro* to assess specific targeting of DC. Uptake was determined via flow cytometry and transmission electron microscopy. Rapamycin loading of pSiNP was confirmed with ultraviolet visualisation and inferred spectrometry. DC were co-cultured with rapamycin loaded pSiNP for 2 days (\pm LPS), irradiated and co-cultured with CFSE stained allogeneic T-cells.

Results: Anti-DC-SIGN pSiNP favourably targeted and were phagocytised by myeloid DC in whole blood samples in a time and dose dependent manner. Myeloid DC were 42% positive for Anti-DC-SIGN functionalised NP compared to only 10% for Isotype control and 5% for unfunctionalised NP. DC preconditioning with RAPA-pSiNP results in a maturation resistant phenotype and significantly suppresses allogeneic T-cell proliferation by $28.6 \pm 1.9\%$ ($p < 0.0001$).

Conclusions: RAPA-pSiNP conjugated to anti-DC-SIGN actively targets and modifies DC function and may serve as a novel therapy to target DC *in vivo*.

Abstract No. 15**TH17 PLASTICITY AND TRANSITION TOWARDS A PATHOGENIC CYTOKINE SIGNATURE IS REGULATED BY CYCLOSPORIN AFTER ALLOGENEIC-SCT**

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Th17 have been widely implicated as drivers of autoimmune disease. In particular, Th17 cytokine plasticity and acquisition of an IL-17A⁺IFN γ ⁺ cytokine profile is associated with increased pathogenic capacity. Donor Th17 polarisation is known to exacerbate GVHD after allogeneic stem cell transplant (allo-SCT), however donor Th17 cytokine co-expression and plasticity have not been fully examined. Using IL-17 'fate-mapping' reporter mice, we identified IL-6-dependent Th17 early after allo-SCT, characterised by significantly elevated expression of pro-inflammatory cytokines, IL-17A, IL-22, GM-CSF and TNF. This population did not maintain lineage fidelity, with a marked loss of IL-17A and IL-22 expression late post-transplant. Th17 could be further segregated based on IFN γ co-expression and IL-17A⁺IFN γ ⁺ Th17 displayed a dramatic pro-inflammatory phenotype. This poly-cytokine phenotype and IFN γ production was critically dependent upon donor derived IL-12/IL-23 and cyclosporin treatment prevented this differentiation pathway. This observation was highly concordant with clinical samples from recipients receiving cyclosporin-based immune suppression where although both populations were present, the IL-17A⁺IFN γ ⁺ Th17 subset predominated. In sum, Th17 polarization and ensuing differentiation are mediated by sequential inflammatory signals which are modulated by immunosuppressive therapy, leading to distinct phenotypes within this lineage.

Abstract No. 16

EXPRESSION OF THREE ALLOGENEIC MHC CLASS I IN RECIPIENT LIVER SIGNIFICANTLY PROLONGS SURVIVAL OF FULLY-ALLOGENEIC VASCULARISED CARDIAC ALLOGRAFTS

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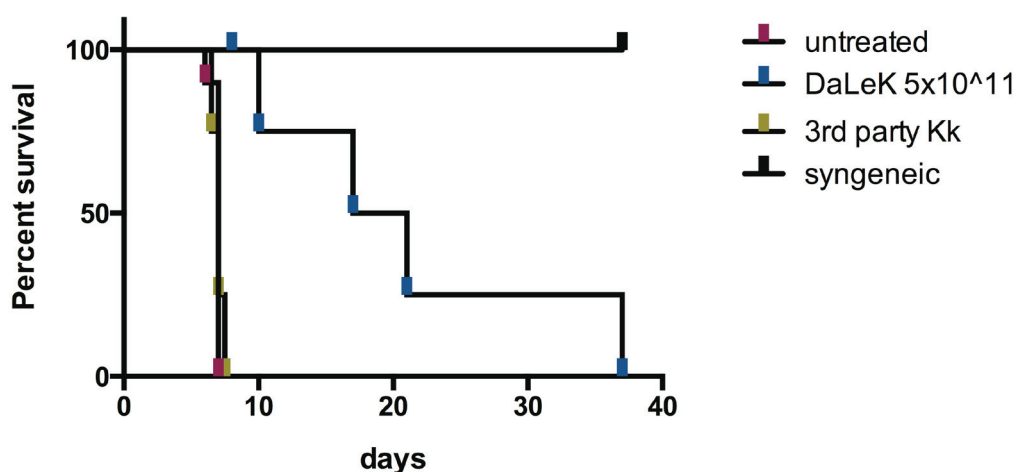
Background and Aim: In previous studies, AAV-mediated gene transfer of a single mismatched donor MHC class I molecule (Kd or Kb) to C57BL/6 or B10.BR recipient liver respectively induced tolerance to skin grafts expressing the same mismatched MHCI molecule. However, such survival is not extended to fully mismatched skin or heart grafts. Tolerance induction may require expression of all mismatched MHC molecules (3 class I and 2 class II). To facilitate expression of multiple MHCI in recipient liver, we created a construct in which the three d-haplotype heavy chains D^d, L^d and K^d were separated by an F2A linker (DaLeK), and then determined the effect of administration of this vector upon heart graft survival.

Methods: DaLeK was packaged into a liver-specific rAAV2/8 vector. Fully-allogeneic hearts from DBA/2 (H-2^d) were transplanted into C57BL/6 (H-2^b) at either d7 or d14 post-inoculation.

Results: Administration of 5×10^{11} vector genome copies AAV-DaLeK to C57BL/6 mice yielded strong expression of D^d, L^d and K^d on hepatocytes. Expression was enhanced by co-transduction with a vector encoding $\beta 2$ microglobulin, ALT levels remained normal and no inflammatory infiltrates were detected. Survival of DBA/2 hearts transplanted into AAV-DaLeK treated mice was prolonged from a MST of 7 days to 23 days. Administration of a control vector did not alter survival (figure).

Conclusion: AAV-DaLeK permits expression of multiple MHCI from a single vector, and its administration significantly prolongs survival of fully-allogeneic heart transplants. A combination of AAV-DaLeK with vectors expressing CIITA and/or allogeneic MHC class II may produce tolerance to fully-allogeneic grafts.

Survival of DbA/2 to C57BL/6 transplants - DaLeK



Abstract No. 17

CAN EXPRESSION OF ALLOGENEIC MHC CLASS II IN RECIPIENT LIVER INDUCE REGULATORY TRANSPLANTATION TOLERANCE?

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Background: Regulatory tolerance to allogeneic cardiac grafts after donor MHC II gene transfer to recipient bone marrow is reported. Allogeneic MHC II is strongly expressed in recipient liver following AAV-mediated gene transfer, accompanied by a dose-dependent increase in liver T regs. However, survival of allogeneic heart grafts in transduced recipients is unchanged. Hepatocytes are not professional APC, lacking significant expression of co-stimulatory molecules, and chaperones required for antigen processing and presentation.

Aim: To determine whether augmenting expression of molecular chaperones and/or co-stimulatory molecules by hepatocytes would facilitate induction of allograft tolerance.

Method: C57BL/6 mice received 1×10^{11} vgc AAV2/8 encoding Class II transactivator (CIITA) and 5×10^{11} vgc IA^d or IA^d alone. MHC II, co-stimulatory molecules, chaperones and inflammatory infiltrate were assessed. IA^d-binding peptides eluted from livers expressing IA^d alone or IA^d / CIITA, were identified by mass spectrometry. DBA/2 hearts were transplanted at d7 or d30 post-inoculation.

Results: CIITA transduction upregulated expression of native IA^b. Expression of H-2M α and β and of Invariant chain were increased 70 to 500-fold by CIITA, attaining levels comparable to those in spleen. Peptides eluted in the presence of CIITA conformed to the IA^d-binding motif (Figure). Expression of co-stimulatory molecules on hepatocytes was not increased by CIITA. Survival of DBA/2 grafts was not altered by the addition of CIITA to IA^d.

Conclusion: Expression of CIITA and a single mismatched MHC II in hepatocytes was not sufficient to confer tolerance to fully-allogeneic heart grafts. Ongoing experiments are evaluating the combination of two mismatched MHC II and/or CD86 with CIITA.

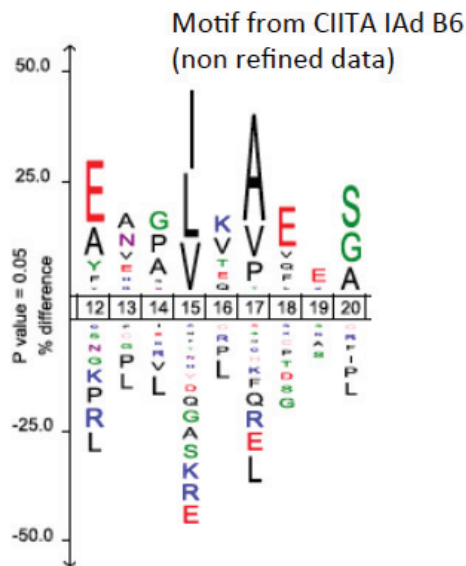


Figure: peptides eluted from livers expressing both IA^d and CIITA conform to the IA^d-binding motif at anchor residues P1, P4, P6 and P9.

Abstract No. 18

ROLE OF IL-7 AND IL-7/ANTI-IL-7 ANTIBODY COMPLEXES IN TREG EXPANSION AND A MURINE SKIN ALLOGRAFT TOLERANCE MODEL

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Aim: To determine whether 1) IL-7 supports CD4⁺Foxp3⁺-Treg survival and 2) IL-7/anti-IL-7mAb complexes induce Treg expansion in the alloresponse.

Methods: CD4⁺Foxp3⁺-Tregs were sorted from Foxp3-GFP (C57BL/6)-mice. Two MLRs were performed. Responders from 1) splenocytes of Foxp3GFP-mice, or 2) sorted- CD4⁺Foxp3⁺-Tregs were both stained with CellTrace™Violet, and stimulated with irradiated splenocytes of NOD/LtSz-scidIL2Rγ^{null}-mice as APCs. IL-7, anti-IL-7mAb, IL-7/anti-IL-7mAb, IL-2 were added to the MLR. At day-5, cells were analysed using flow cytometry. CD4⁺Foxp3⁺-Tregs were adoptively transferred into Rag-/- mice. Skin transplantation with C57BL/6, DBA, B10.Br/CBA grafts were performed at day 2 after adoptive transfer. At >100 days post-transplant, Rag-/- mice were treated with IL-7/anti-IL-7mAb complexes once a week for 3 weeks. These mice were challenged with CD4⁺ effector-T cells at week 2.

Results: At day-5 of MLR (Tregs), the proportion of Foxp3⁺-Tregs were similar under IL-7 (63%), IL-2 (68%), IL-7/anti-IL-7mAb (67%) conditions but not anti-IL-7mAb (21%) or control (19%). Expression of CD127 was present on Foxp3⁺-Tregs with IL-2 (22%) and anti-IL-7mAb (17%), but not IL-7 (2%) and IL7/anti-IL-7mAbs (3%) conditions. There were no differences in T cell proliferation or the proportion of memory (CD4⁺CD44^{high}) T cells using these conditions at day-5 of MLR (splenocytes). The proportion of Foxp3⁺-Tregs was higher in spleens of Rag-/- mice reconstituted with Tregs and IL-7/anti-IL-7mAb treatment group (7.0±2.6%) when compared Tregs from control mice (2.6±1.9%) at week 3 of treatment. However, CD4 effectors did not reject skin grafts in either group.

Conclusions: IL-7 supported Foxp3 Treg survival and IL-7/anti-IL-7mAb complexes can expand Tregs in a model of skin allograft tolerance.

Abstract No. 19

IN VITRO REACTIVITY OF CD4⁺CD25⁺ AND CD4⁺CD25⁻ T CELL SUBSETS FROM RATS WITH TOLERANCE TO AN ALLOGRAFT

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Aims: Transplant tolerance induced in adult animals is mediated by alloantigen-specific CD4⁺CD25⁺T cells but proliferation of tolerant CD4⁺T cells to specific alloantigen in MLR is not impaired. We aim to identify changes that may diagnose alloantigen-specific tolerance.

Methods: The proliferation of CD4⁺, CD4⁺CD25⁺ and CD4⁺CD25⁻T cells from rats with tolerance to a cardiac allograft and from naïve rats were compared in MLR. Their cytokine and cytokine receptor expression was also examined.

Results: Paradoxically tolerant CD4⁺CD25⁺T cells did not proliferate to specific donor or self, but did to third-party, whereas naïve CD4⁺CD25⁺T cells responded to both alloantigens but not self. Proliferation of naïve and tolerant CD4⁺T cells was similar, to both specific and third-party. Compared to CD4⁺T cells, tolerant CD4⁺CD25⁻T cells had greater responses to third-party but not to specific donor, whereas naïve CD4⁺CD25⁻T cells' proliferation to both alloantigens was greater than CD4⁺T cells. Tolerant but not naïve CD4⁺CD25⁺T cells expressed receptors for IFN-γ and IL-5 and these cytokines promoted their proliferation to specific donor but not third-party. IFN-γ and IL-5 suppressed tolerant CD4⁺T cells proliferation to specific donor. These findings demonstrate that in vitro reactivity of CD4⁺T cell subsets from animal with alloantigen specific tolerance is different to that of naïve animals and could be used to detect transplant tolerance.

Conclusions: Our findings suggested that CD4⁺CD25⁺T cells that mediate transplant tolerance depend upon IFN- γ or IL-5 from alloactivated Th1 and Th2 cells. One or more of these differences between naïve and tolerant CD4⁺ T cell subsets may provide a test for transplant tolerance.

Abstract No. 20

ADMINISTRATION OF IL-2/IL-2 AB COMPLEX IN COMBINATION WITH ALLO-PEPTIDE PULSED HOST SPLENOCYTES PROLONGS SKIN ALLOGRAFT SURVIVAL

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Background: *In vivo* Treg expansion using IL2 complexed with anti-IL2 antibody (JES6-1) has been demonstrated to be effective in the induction of long-term tolerance of islet allografts but was unable to induce skin graft acceptance. In this study, we investigated whether Tregs expanded using IL2/IL2 Ab complex in combination with donor allo-peptides can improve skin graft acceptance.

Method: B6 hosts were given IL2/IL2 Ab complex ip for 3 consecutive days before bm1 skin grafting. bm1 peptides pulsed B6 splenocytes were transfused at various time points: Group1, at Day1 and skin-grafting at Day4; Group 2, at Day1 and Day3, skin grafting at Day4; Group 3, at Day4 and skin grafting at Day7. B6 peptides were used as control.

Results: Injection of IL2 complex induced a 7.5 fold increase of Foxp3 Tregs in peripheral blood at Day4 after injection. B6 peptide control group rejected bm1 skin grafts acutely (MST=14). Bm1peptides at Day1 showed prolonged graft survival (MST=22), while additional administration of bm1 peptides at Day3 did not further improve graft survival; bm1 peptides administered at a later time point at Day4 had less effect on prolonging graft acceptance (MST=18).

Conclusion: IL-2 complex in combination with bm1 peptide pulsed host splenocytes can expand antigen specific Tregs promoting subsequent skin graft acceptance.

Sensitisation, Antibodies and ABO Incompatible Transplantation & Organ Donation and Ethics

Abstract No. 21

PROVIDING BETTER MATCHED DONORS FOR HLA MISMATCHED COMPATIBLE PAIRS THOUGH KIDNEY PAIRED DONATION

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Aim: To define allocation metrics that enable compatible pairs (CP) receiving a better-matched kidney in kidney paired donation (KPD) program, without disadvantage to incompatible pairs (ICP).

Background: Participation of CP in KPD could be attractive to CP who have a high degree of HLA-mismatch, if the KPD allocation algorithm provides a better HLA match for the CP recipient. Because KPD programs were not designed to help CP, it is important to define allocation metrics that enable CP receiving a better-matched kidney, without disadvantage to ICP.

Methods: Virtual crossmatch is used for ICP allocation in the Australian KPD program. The algorithm ignores HLA matching rules and therefore is unlikely to provide better HLA matching to CP. Simulations using 46 ICP and 11 randomly selected CP with 6/6 ABDR mismatch were undertaken. Allocations were preformed adding one CP at a time or all 11 CP at once, without and with exclusion of unacceptable antigens selected to give a virtual cPRA in the range of 70-80% to improve HLA matching in CP recipients.

Results: Inclusion of one CP at a time increased matching in ICP by up to 33% and inclusion of all 11 CP at once increased ICP matching by 50%. The difference in the average eplet mismatch (EpMM) with the own donor (78 ± 19) was significantly lower (57 ± 15 , $P < 0.02$) only when individual CP recipients had unacceptable antigens assigned for exclusion. When the 11 CP were added at once the EpMM with the matched donor was significantly better than with the own donor when they were added without (58 ± 10 , $P < 0.03$) and with (60 ± 11 , $P < 0.02$) exclusion of unacceptable antigens. Only recipients whose EpMM to own donor was > 65 significantly reduced the EpMM with the matched donor.

Conclusions: CP participation in KPD can increase match rates in ICP and can provide a better immunological profile in CP recipients who have a high EpMM to their own donor when using allocation based on virtual crossmatch.

Abstract No. 22**SYK INHIBITION REDUCES RENAL ALLOGRAFT INJURY IN A RAT MODEL OF ACUTE ANTIBODY-MEDIATED REJECTION IN HIGHLY SENSITIZED RECIPIENTS.****RAMESSUR CHANDRAN Sharmila^{1,2}, MA Frank, Y^{1,2}, TESCH Greg, H^{1,2}, HAN Yingjie^{1,2}, OZOLS Elyce¹, DI PAOLO Julie³, MULLEY William^{1,2}, KANELIS John^{1,2}, NIKOLIC-PATERSON David, J^{1,2}**¹*Department of Nephrology, Monash Medical Centre, Melbourne,* ²*Department of Medicine, Monash University, Melbourne,* ³*Empty, Gilead, Foster City, CA, USA***AIM:** To determine the therapeutic potential of Syk inhibition in a rat model of antibody-mediated renal allograft rejection in sensitized recipients.**METHODS:** Recipient Lewis rats were immunized with donor(Dark Agouti) spleen on day-5. Recipients underwent bilateral nephrectomies and orthotopic renal transplantation(day 0). Groups received Syk inhibitor(SYK-A, 30mg/kg/bid)(n=11) or vehicle(n=12) from -1hr until being euthanized on day 3. Cellular rejection was minimized by administration of the IL-2 inhibitor, Tacrolimus, given from day -1.**RESULTS.** Vehicle treated recipients exhibited delayed graft function on day 1(serum creatinine 230±84µmol/L vs 45±5µmol/Lnormal) which worsened by day 3(363±192 µmol/L). Histology showed severe damage(thrombosis, acute tubular injury, capillaritis). High serum levels of donor-specific antibodies were detected by flow cytometry and rat IgG and C3 were deposited in allografts. A modest T cell infiltrate was evident, but little up-regulation of IL-2 mRNA, indicating effective Tacrolimus inhibition of cellular rejection. SYK-A did not prevent delayed graft function on day 1, but significantly improved graft function on day 3(199±131µmol/L; P<0.05 vs vehicle) with reductions in capillaritis, tubular injury and thrombosis. Immunostaining showed a 45% reduction in the macrophage infiltrate(P<0.05), and an 80% reduction in IFN-γ mRNA levels in the Syk inhibitor treated animals suggesting reduced macrophage activation. T cell infiltration, IL-2 mRNA levels and serum DSA levels were equivalent to levels observed in the vehicle treated group.**CONCLUSION:** Syk inhibition significantly attenuated allograft injury in a model of severe antibody-mediated damage in highly sensitized recipients. These findings suggest Syk inhibition as a potential adjunctive treatment in clinical AMR.Abstract No. 23**THE NATURAL HISTORY OF DONOR SPECIFIC ANTIBODIES (DSA) IN KIDNEY TRANSPLANT RECIPIENTS (KTX) AND ASSOCIATED CLINICAL OUTCOMES****WAN Susan^{1,2}, WYBURN Kate^{1,2}, YIN Jianlin^{1,2}, WATSON Narelle³, SAUNDERS John¹, ERIS Josette^{1,2}**¹*Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney,* ²*Faculty of Medicine, Central Clinical School, University of Sydney,* ³*NSW Transplantation and Immunogenetics Laboratory, Clinical Services and Research Division, Australian Red Cross Blood Service***Background:** DSA are implicated in the development of rejection and graft dysfunction. However, the natural history remains poorly characterized.**Aim:** To characterize DSA development and associations with clinical outcomes in KTx.**Methods:** Serum samples from adult KTx between July2010-May2015 were prospectively collected pre-transplant and at Days7,28 and Months3,6,9,12 post-transplant and tested for DSA on a luminex platform. Acute rejection (AR) episodes were biopsy proven.**Results:** 122 recipients had pre- and post-transplant DSA testing. Mean follow-up was 29±15 months. Seventy (57%) patients had DSA; 48 (39%) had pre-existing DSA only, 10 (8%) had denovo (dn)DSA only, and 12 (10%) had pre-existing and dnDSA. Of patients with dnDSA; 7(32%) had ClassI antibodies only, 12(55%) had ClassII antibodies only and 3(14%) had Class I&II. Twenty-four (40%) patients with pre-existing DSA and 14 (64%) patients with dnDSA had AR. Patients with dnDSA vs those without were more likely to develop AR (OR4.75; 95%CI 1.64-13.75; P=0.004), however there was no increased risk in patients with pre-existing DSA only (OR1.36; 95%CI 0.58-3.20; P=0.485). Median time to rejection was 2.3 months (IQR0.2-7.4). dnDSA appeared prior to the rejection episode in all patients with dnDSA and AR. Mean creatinine at 6 and 12 months post-transplant was higher in patients with dnDSA vs without dnDSA (Mean 12-month creatinine: 171 vs 128µmol/L; t₈₃=-3.33; P=0.001).**Conclusions:** 18% of patients developed dnDSA. This was associated with a higher risk of AR and worse graft function. Further evaluation of the risk factors for dnDSA and the association with long-term outcomes is required.

Abstract No. 24

ADDITIONAL OPPORTUNITIES FOR TRANSPLANTING ORGANS FROM DONORS WITH BRAIN MALIGNANCIES? AN AUDIT OF THE NSW ORGAN AND TISSUE DONATION SERVICE (OTDS) ORGAN DONOR REGISTER**HANCOCK Rebecca¹, WYBURN Kate^{2,3}, O'LEARY Michael^{4,2,5}, WEBSTER Angela^{2,6}**¹*School of Public Health, University of Sydney*, ²*Sydney Medical School, University of Sydney*, ³*Renal Unit, Royal Prince Alfred Hospital, Sydney*, ⁴*NSW Organ and Tissue Donation Service*, ⁵*Royal Prince Alfred Hospital, Sydney*, ⁶*Centre for Kidney Research, Westmead Hospital*

Aims: Donors with primary brain malignancies (PBMs) present an on-going challenge due to uncertainty around transmission risk, classification complexity and variability in guidelines. Data for donors who don't proceed is not captured by ANZOD. We sought to retrospectively identify any untapped potential opportunities for organ donation among people with PBMs referred to the NSW OTDS.

Methods: We reviewed all NSW OTDS referral logs for 2010-2015. We compared people with past/current PBM who donated (actual/intended) and did not donate (potential donors), including those deemed not medically suitable due to cancer, in light of current evidence. Reasons for outcome variability were evaluated.

Results: Of 2,611 total donation referrals (2,032 potential, 579 intended/actual), 49 patients had PBMs, 10 of whom donated (7 actual, 3 intended) and 39 who did not (21 excluded due to PBM) (Table). Those who donated had lower grade tumours, while patients excluded due to PBM were more likely to have higher grade tumours or unclear grading. Medical suitability decisions were variable for astrocytoma and meningioma. We identified 19 additional potential donor opportunities, including 3 people with PBM of 'low' transmission risk (<2%) (2 astrocytoma, meningioma). A further 16 people had 'intermediate' transmission risk (2.2% with an upper 95% CI of 6.4%) malignancies (glioblastoma, germinoma, ependymoblastoma).

Conclusions: Realisation of an additional potential 19 donors would increase intended/actual donor pool by 3.3%. Limitations in administrative data mean all considerations informing past decisions may not be clear. Further consideration of the potential for people with PBMs, including higher grade tumours, to be donors may be warranted.

Table: Type of primary brain malignancy by donation outcome

Type of primary brain malignancy	Tumour grading ¹	Estimated transmission risk (upper 95% CI) ²	Potential (NMS due to cancer)	Actual and Intended
Acoustic neuroma	Low	<2%	3	2
Astrocytoma	Variable	<2%	3 (2)	3
Craniopharyngioma	Low	<2%	1	1
Ependymoblastoma	High	2.2% (6.4%)	1(1)	
Ganglioma	Low	<2%	1	
Glioma/Glioblastoma/ Glioblastoma multiforme	High	2.2% (6.4%)	18 (14)	
Medulloblastoma (past cancer, deemed cured)	High	N/A		1
Meningioma	Variable	<2%	9 (2)	3
Thalamic germinoma	High	2.2% (6.4%)	1 (1)	
Unspecified Cerebral	N/A	N/A	2 (1)	
Total			39 (21)	10

1. Tumour grades obtained from OTDS registry records, with WHO Grading of Tumours of the Central Nervous System guideline used where grades were not recorded. 2. Estimated transmission risks were obtained from SaBTO Advisory Committee on the Safety of Blood, Tissues and Organs, *Transplantation of Organs from Deceased Donors with Cancer or a History of Cancer*, UK, 2014.

Abstract No. 25

UTILISING ORGANS FROM DONORS WITH BLOOD-BORNE VIRUSES (BBVS) IN NSW, 2010-2015
HANCOCK Rebecca¹, WYBURN Kate^{2,3}, O'LEARY Michael^{4,2,5}, WEBSTER Angela^{2,6}¹*School of Public Health, University of Sydney*, ²*Sydney Medical School, University of Sydney*, ³*Renal Unit, Royal Prince Alfred Hospital, Sydney*, ⁴*NSW Organ and Tissue Donation Service*, ⁵*Intensive Care Service, Royal Prince Alfred Hospital, Sydney*, ⁶*Centre for Kidney Research, Westmead Hospital, Sydney*

Aims: Data for people referred for organ donation who do not proceed is not captured by ANZOD. We sought to describe NSW donor referrals from 2010-2015 with hepatitis C (HCV), hepatitis B (HBV) and human immunodeficiency virus (HIV) who did and did not proceed to donation.

Methods: We reviewed NSW Organ and Tissue Donation Service referral logs for 2010-2015. We reviewed referrals with BBVs (hepatitis B, C and HIV) who donated (actual/intended donors) and did not donate (potential donors), including those deemed not medically suitable (NMS) due to behaviours that are high risk for BBVs. Reasons for outcome variability were evaluated.

Results: Of 2,611 total donation referrals (2,032 potential, 579 intended/actual), 151 (5.8%) were recorded as having BBVs, 124 of these with HCV, 25 with HBV (table). There were 135 potential BBV donors and, 16 actual/intended donors (10.6% of referrals with BBVs). For 37 (24.5%) of referrals with BBVs no suitable recipient was found. None of the 5 patients with HIV became actual donors. Among the 135 potential BBV donors, consent was not granted (family or coronial refusal) in 32 cases, 23 were deemed NMS due to high risk behaviour, and 66 were NMS for reasons unrelated to BBVs.

Conclusions: In NSW, referrals with BBVs are considered for and in certain circumstances proceed to organ donation. With potentially curative treatments for HCV imminent, new risk paradigms may evolve. Consideration of opportunities to increase donation rates, particularly among those identified as high risk for BBV transmission, may be warranted.

Table: Donors with blood borne viruses by donation outcome in NSW, January 2015 to June 2015

Blood borne virus	Total donors	Potential donors (NMS high risk*)	Actual & intended donors
Hepatitis B virus (HBV)	13	12 (3)	1
Hepatitis C virus (HCV)	112	99 (15)	13
HBV + HCV	12	12 (3)	0
Unspecified hepatitis	3	2	1
Possible hepatitis	6	6	0
HIV	5	4 (2)	1
Total	151	135 (23)	16

* In total, 56 of 2,611 total referrals were deemed NMS due to 'high risk' behaviour, including the 23 with identified BBVs noted above.

Abstract No. 26

DE NOVO DONOR SPECIFIC ANTI-HUMAN LEUKOCYTE ANTIGEN (HLA) ANTIBODIES IN KIDNEY TRANSPLANTATION - A SYSTEMATIC REVIEW AND META-ANALYSIS**SHARMA Ankit¹, LEWIS Joshua^{1,2}, WAI Lim^{2,3}, PALMER Suetonia⁴, STRIPPOLI Giovanni¹, CHAPMAN Jeremy⁵, CRAIG Jonathan¹, WONG Germaine¹**¹Centre for Kidney Research, The Children's Hospital at Westmead, Sydney, ²School of Medicine & Pharmacology, University of Western Australia, Perth, ³Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth, ⁴Department of Medicine, University of Otago Christchurch, ⁵Centre for Transplant and Renal Research, Westmead Hospital, Sydney

Pre-transplant donor specific anti-human leukocyte antigen antibodies (DSA) are associated with poorer graft outcomes after kidney transplantation. De novo DSA (dnDSA) are common but the implications of these antibodies for patient-relevant outcomes are uncertain. We undertook a systematic review and meta-analysis of prospective and retrospective cohort studies of adult and paediatric kidney or adult simultaneous kidney-pancreas transplant recipients to determine the association between dnDSA and transplant and patient outcomes. We searched MEDLINE and Embase for studies published by November, 2015. Thirty-seven studies were eligible, involving 9844 transplant recipients. Associations between dnDSA and outcomes were estimated by random effects meta-analysis with univariate meta-regression used to explore sources of between-study heterogeneity. Pre-specified outcomes were graft loss (23 studies, 636/5177), any acute rejection (13 studies, 352/2103), antibody mediated rejection (14 studies, 149/2395) and death (6 studies, 64/1517). The prevalence of recipients with dnDSA was 16.3%. Transplant recipients with dnDSA experienced an increased relative risk (RR) for any acute rejection (RR 3.12; 95% CI 2.04-4.79, antibody-mediated rejection (RR 12.98; 7.09-23.76), graft loss (RR 4.17; 3.31-5.24) and death from any cause (RR 2.32, 1.00-5.37). There was moderate to substantial heterogeneity in estimates between studies (I^2 41-80%). Study-level factors explaining some heterogeneity included proportion of living donors, recipient age, and diagnostic assay for dnDSA. These findings suggest the presence of dnDSA is associated with an increased risk of acute rejection, particularly antibody mediated rejection and subsequent graft loss. Therefore studies evaluating the effects of interventions to lower dnDSA with graft and patient outcomes are needed.

Abstract No. 27

SUCCESSFUL ABO INCOMPATIBLE NON- HEART BEATING DECEASED DONOR KIDNEY TRANSPLANTATION**KRISHNAN Anoushka¹, PUTTAGUNTA Harish¹, HE Bulang², THOMPSON Ivan², BHANDARI Myank², SWAMINATHAN Ramyasuda¹, IRISH Ashley¹**¹Department of Nephrology, Fiona Stanley Hospital, WA, ²Department of Surgery, Fiona Stanley Hospital, WA

Background: Deceased donor kidney allocation is conventionally made by ABO acceptable criteria. Western Australia (WA) does not ship donation after circulatory death (DCD) kidneys due to concerns with prolonged cold ischaemic time. To maximize allocation and overcome the difficulty in allocating DCD kidneys within blood groups A, B and AB due to low numbers of potential recipients, WA devised a strategy to ensure DCD allocation could occur regardless of ABO blood group by measuring Anti-A and B titres on waitlisted patients.

Case Reports: We report two cases of DCD transplantation where non-availability of appropriate blood group matched recipient in WA required implementing ABO-incompatible (ABOi) transplantation.

Patient	Age/Gen der	Renal Disease	Donor age/gender	Recipient blood group	Donor blood group	Anti-A titre pre	Anti-A titre post	Total ischaemic time
1	54/F	ADPCKD	63/M	B	A2	1:16	1:2*	7:25
2	66/M	ADPCKD	65/F	B	A1	1:8	1:4#	7:36

ADPCKD= Autosomal dominant polycystic kidney disease *Anti-A column # conventional PEX

Both patients received pre-operative plasmapheresis and induction with rabbit anti-thymocyte globulin or basiliximab, with reduction in pre-transplant anti-A titres to 1:4 or less. Though their courses were complicated by delayed graft function, creatinine improved to nadir of 130µmol/L and 165µmol/L respectively, with early protocol biopsies showing no cellular or humoral rejection. To our knowledge, patients 1 and 2 are the first deliberate ABOi DCD and ABOi ECD DCD performed in Australia.

Conclusion: Local allocation of ABOi DCD donors is a viable option to allow renal transplantation, avoiding prolonged ischaemic time from shipping or organ discard due to no ABO acceptable recipient.

Abstract No. 28**TREATMENT OF ACTIVE ANTIBODY MEDIATED REJECTION IN RENAL TRANSPLANT RECIPIENTS****MARUI Yuhji, YAMAGUCHI Haruna, TANAKA Kiho, ISHII Yasuo***Department of renal transplantation surgery, Toranomon Hospital Kajigaya*

Aims: To evaluate the adequacy of our treatments for active antibody mediated rejection (aAMR) in renal transplant recipients based on the outcome.

Methods: Out of 120 recipients from 2007 to 2015 at our hospital, biopsy proven aAMR cases were identified and examined about patient background, immunological risk, pathological finding, treatment and outcome.

Results: Four early onset aAMR (E-group) and 6 late onset aAMR (L-group) were diagnosed with biopsy, of which 4 cases (2 E-group and 2 L-group) were not fulfilled Banff criteria but considered as aAMR. All cases had deterioration of graft function. Treatment for aAMR included bolus steroid (all), IVIG (all), Deoxyspergualin (4/9), and plasma exchange (PE) (6/9). None of them was applied Rituximab after diagnosis of aAMR, but 2 cases had this prior to the operation due to ABO incompatible transplantation. Mean follow-up period after diagnosis were 2.0 years (range 0.3-7 years), and one graft was lost in E-group. Mean creatinine level was 2.2mg/dl (range 1.2-5.0mg/dl). Three cases of E-group were complicated at diagnosis and one of them did not receive PE resulting in graft loss, whilst others with severe renal dysfunction had PE immediately. On a case of L-group due to difficult pathological findings PE was hesitated for 2 years after biopsy, then his graft function worsen in the meantime.

Conclusions: With short term follow-up most of aAMR could have been managed with these treatments. Our experience suggested that just-in-time treatment including PE might improve the aAMR patients' outcome especially in complicated pathological finding cases.

Abstract No. 29**ALLOGRAFT DYSFUNCTION COMPLICATING PARATHYROIDECTOMY IN RENAL TRANSPLANT RECIPIENTS: A TYPICAL CASE AND REVIEW OF THE LITERATURE ON UNDERLYING MECHANISM****SEE Emily¹, DWYER Karen²***¹Department of Nephrology, Barwon Health, ²School of Medicine, Deakin University*

Introduction: Persistent hyperparathyroidism after successful renal transplantation is a common clinical problem. Deterioration in renal allograft function following parathyroidectomy is recognised, however the mechanism underpinning this decline is not well described. We present a typical case and a review of the literature surrounding the pathogenesis.

Case: A 60 year-old male underwent living unrelated renal transplantation for ESKD secondary to IgA nephropathy. He underwent subtotal parathyroidectomy 6-months later due to persistent tertiary hyperparathyroidism. His creatinine immediately rose from 170 to 270umol/L. Renal biopsy demonstrated medullary calcium deposition and mild CNI toxicity. His creatinine improved to 200umol/L after several months where it has remained for 5 years.

Discussion: Evidence from pre-clinical studies suggests that PTH has a regulatory effect on renal perfusion, glomerular filtration and mesangial cell function. By mimicking the vascular action of PTHrP, elevated levels of circulating PTH result in vasodilatation of the renal artery, pre-glomerular arterioles and glomeruli leading to augmented renal perfusion and glomerular filtration. In human studies, infusions of PTH/PTHrP result in a dose-dependent increase in renal blood flow as well as enhanced cardiac output. Therefore, a rapid decline in circulating PTH unsurprisingly results in an acute reduction in graft perfusion. This reduction may be further augmented by the vasoconstrictive effect of calcineurin inhibitors.

Conclusion: Hyperparathyroidism following renal transplantation poses a complex clinical problem. Alterations in renal blood flow and microcirculation have deleterious effects on graft function although fortunately do not affect graft survival. Ultimately, this reinforces the necessity for optimisation of biochemical parameters prior to transplantation.

Abstract No. 30

ANTI BLOOD GROUP ANTIBODY TITRES IN BLOOD GROUP A AND B TRANSPLANT WAIT LISTED PATIENTS**RUDERMAN IRENE¹, VANHARDEVELDT Emma¹, HUGHES PETER^{2,3}, MASTERSON ROSEMARY^{2,4}**¹*Nephrology and Renal Transplant, Royal Melbourne Hospital,* ²*Renal & Transplantation Unit, Royal Melbourne Hospital,* ³*School of Medicine, Monash University, Melbourne,* ⁴*Department of Medicine, University of Melbourne*

Background: Historically, deceased donor ABO-incompatible renal transplantation (DD-ABOi) has been limited to kidneys from A2 or A2B blood group donors transplanted into group B recipients with low anti-A titres. Recent successful living donor ABOi from all blood groups into recipients with low titre anti-blood group antibody (ABGAb) using standard immunosuppression alone suggests DD-ABOi may be possible into selected recipients with limited or no antibody removal pre-transplantation. We sought to systematically measure ABGAb titres in our deceased donor wait listed group A and B patients.

Methods: ABGAb titres of wait listed patients were compared with our institutional threshold for transplantation of $\leq 1:8$ (Ortho) to determine how many might accept a DD-ABOi kidney with one or no antibody removal treatments prior to transplantation.

Results: To date 36/106 (34%) group A and 16/55 (29%) group B patients have had titres measured. Of the group A patients, 66% had an anti B titre $\leq 1:8$ with 69% of group B patients having an anti A titre $\leq 1:8$. Of the 52 patients who have had titres measured, 25 (48%) have titres $<1:4$

Conclusion: A significant number patients have ABGAb sufficiently low to enable DD-ABOi with limited or no antibody removal.

Abstract No. 31

BENEFITS OF MODERATING LOCATION OF DONOR LIFE SUPPORT WITHDRAWAL ON LIVER TRANSPLANTATION USING DONATION AFTER CIRCULATORY DEATH: A META-ANALYSIS**CAO Yiming¹, SHAHRESTANI Sara², CHEW HC¹, CRAWFORD Michael³, MACDONALD Peter¹, LAURENCE Jerome^{3,4}, HAWTHORNE WJ⁵, DHITAL Kumud¹, PLEASS Henry⁵**¹*Transplantation Laboratory, Victor Chang Cardiac Research Institute, Sydney,* ²*Western Clinical School, Westmead Hospital, Sydney,* ³*Australian National Liver Transplantation Unit, Royal Prince Alfred Hospital, Sydney,* ⁴*Institute of Academic Surgery, Royal Prince Alfred Hospital, Sydney,* ⁵*Department of Surgery, Westmead Hospital, Sydney*

Aims: Prolonged warm ischaemic time, and the associated micro-thrombi formation, have been identified as the key factors responsible for poor outcomes of liver transplantation using donation after circulatory death (DCD) compared to using donation after brain death (DBD). Therefore, we sought to investigate the effects of ante-mortem heparin administration and location of donor life support withdrawal in intensive care unit (ICU) vs. operating theatre (OT) on DCD outcomes.

Methods: Medline, EMBASE and Cochrane libraries were systematically searched and 23 relevant studies identified for analysis.

Results: Donor life support withdrawal in OT, compared to ICU, was associated with reduced DCD patient mortality (OT: OR = 1.2, 95% CI 0.85-1.68; ICU: OR = 2.15, 95% CI 1.15-4.02), graft loss (OT: OR = 1.65, 95% CI 1.16-2.36; ICU: OR = 1.98, 95% CI 1.13-3.47) and incidence of ischaemic cholangiopathy (OT: OR = 13.73, 95% CI = 5.18-36.44; ICU: OR = 19.68, 95% CI 7.48-51.75) relative to DBD recipients. Ante-mortem administration of heparin attenuated the rate of allograft primary non-function (heparin: OR = 3.48, 95% CI 1.79-6.76; no heparin: OR = 11.24, 95% CI 1.99-63.37).

Conclusions: Our evidence suggests that these changes in DCD donor life support withdrawal could confer significant benefits upon their recipients. Specifically the practice of using ante-mortem heparin and withdrawal of treatment in the OT could reduce rate of graft and patient loss, and ischaemic cholangiopathy, thereby maximising benefits derived from these valuable organs.

MONDAY ABSTRACTS

IN SESSION ORDER

Transplant Complications #1

Abstract No. 32

RISK OF HEPATITIS B REACTIVATION IN CORE ANTIBODY POSITIVE PATIENTS AFTER RENAL TRANSPLANTATION

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Background and Aim: Reactivation of Hepatitis B virus (HBV) post transplantation may cause severe acute hepatitis. The risk of HBV reactivation in HBV core antibody positive (HepBcAb+) and HBV surface antigen negative (HepBSAg-) renal transplant recipients (RTR) is unknown. Following an index case of HBV reactivation, practice at our centre was evaluated.

Methods: Retrospective cohort study of RTR transplanted between 1970 – 2014. HBV status was recorded and HepBcAb+, HepBSAg- patients had HBV serology and quantitative DNA measured.

Results: Of 740 patients studied, 692 had HBV status evaluated, 11 patients were HepBSAg positive at transplant and treated with antiviral agents. 57 (8.2%) patients were HepBcAb+, HepBSAg - at transplant. Twenty nine (50%) were Asian, thirteen (23%) were Pacific Islander. Median time from transplant was 7.6y. Two patients were pre-emptively treated with anti-viral therapy. No systematic screening for HBV seroreversion was undertaken. On rescreening, four had detectable HBV DNA (including index case) ranging from 1.9×10^2 to $>1.1 \times 10^8$ IU/ml. Two patients had mild derangements of liver function tests (ALT < x2 ULN). Liver ultrasound was mildly abnormal in three. At transplant, HepBSAb was < 100 in 3 of 4 patients.

Conclusions: We found a low rate of HBV reactivation in cAb positive RTR which was not associated with clinically significant disease. Patients undergoing renal transplantation should be screened prior to transplant for HBV and standardised protocols for monitoring HBV status should be implemented (such as annual HepBSAg testing) however pre-emptive antiviral therapy does not appear to be warranted.

Abstract No. 33

SUBCLINICAL CYTOMEGALOVIRUS VIRAEMIA IN RENAL TRANSPLANT RECIPIENTS

BARKER Kristeen¹, COOK Natasha¹, POLKINGHORNE Kevan², IERINO Frank¹¹Renal Transplant Unit, Austin Hospital, Melbourne, ²Renal Transplant Unit, Monash Medical Centre, Melbourne

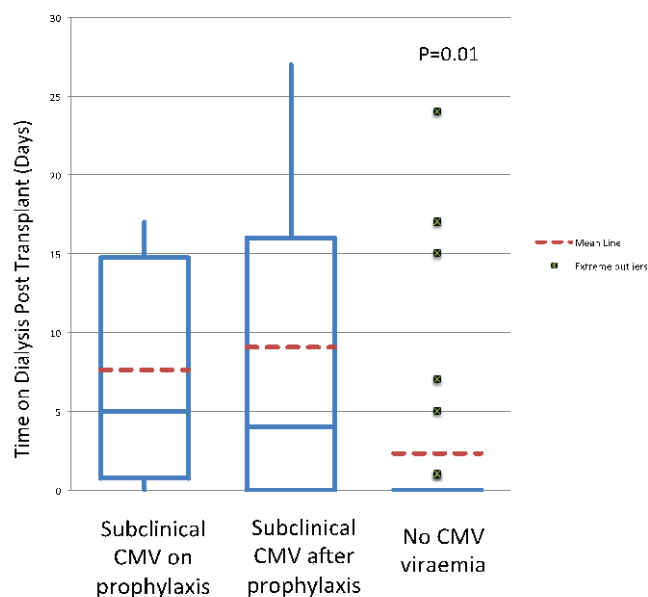
Background: Studies suggest low-level cytomegalovirus (CMV) viraemia, detected with sensitive molecular assays, may lead to poorer graft outcomes following renal transplantation.

Aims: Investigate the incidence, outcomes, and clinical associations of subclinical CMV viraemia in adult renal transplant recipients.

Methods: A retrospective cohort study from January 2010 to March 2012 with 3 years follow up.

Results: 22/59 patients were diagnosed with subclinical viraemia. 8/22 cases occurred whilst receiving anti-viral prophylaxis. Renal function was similar at 12, 24 and 36 months in both CMV viraemic and non-viraemic patients. Total rejection was not different ($p=0.13$) between groups. Anti-viral prophylaxis was under-dosed when corrected for GFR at day 7 in 83.3% of patients who developed viraemia on prophylaxis, compared to 23.1% of patients with viraemia after prophylaxis stopped and 22.7% of patients who were never viraemic ($p=0.03$). By 21 days there was no significant difference in prophylaxis dosing between groups. In those who developed CMV during prophylaxis the percentage improvement in GFR at 14 to 28 days was 161.7% compared to 49.7% in the CMV after prophylaxis and 36.3% in the never viraemic group $p=0.001$. Median time on dialysis post transplant was higher in those who later developed subclinical viraemia $p=0.01$ (Figure).

Conclusion: Subclinical CMV viraemia is common and associated with early dialysis requirements following transplantation. Early under-dosing of antiviral prophylaxis was significantly associated with rapid improvement in GFR and a risk factor for subclinical CMV during the prophylaxis period. Accurate early prophylaxis dosing and CMV surveillance may benefit patients with delayed graft function.



Abstract No. 34**GRAFT-VERSUS-HOST DISEASE PRECIPITATES CYTOMEGALOVIRUS REACTIVATION AFTER ALLOGENEIC BONE MARROW TRANSPLANTATION: LESSONS FROM THE FIRST PRECLINICAL MODEL.****MARTINS Paulo¹**, TEY Siok-Keen¹, FLEMING Peter², KUNS Rachel D¹, ULLAH Md Ashik¹, VARELIAS Antiopi¹, KOYAMA Motoko¹, ANDONIOU Christopher E², DEGLI-ESPOSTI Mariapia A², HILL Geoffrey R¹¹*Bone Marrow Transplantation Laboratory, Queensland Institute of Medical Research, Brisbane,* ²*Centre for Experimental Immunology, Lions Eye Institute*

Cytomegalovirus (CMV) infection remains a significant complication after allogeneic bone marrow transplantation (BMT) and for reasons that are poorly understood, CMV seropositivity remains a major determinant of clinical transplant outcome.

Aim: To elucidate the mechanisms and immunological consequences of CMV reactivation in a newly established model.

Methods: Mice were infected with murine CMV (MCMV) and functional latency defined as the resolution of viraemia in target organs and plasma. Latently infected mice were transplanted with Bone Marrow (BM) and T cells or T cell-depleted (TCD) BM alone from MHC-disparate or MHC-matched uninfected donors to generate GVHD and non-GVHD conditions respectively. Reactivation was determined by qPCR on plasma and plaque assays in target organs after BMT.

Results: MCMV enters latency two months after primary infection with the resolution of viremia correlating with absence of replicating virus in organs by plaque assays. The reactivation of MCMV after BMT is GVHD-dependent: BALB/c→B6: 63% vs 17% (p<0.05) and B6→BALB/c: 100% vs 15% (p<0.0001) in GVHD vs non-GVHD recipients. This is corroborated by plaque assays, with virus replication detected in 54% vs 0% (p<0.01) of livers and 46% vs 8% of lungs in the former model. GVHD is characterized in mice by an interferon-based cytokine storm early after BMT and recipients of IFN γ ^{-/-} grafts are more susceptible to CMV reactivation: wild type vs. IFN γ ^{-/-}: 20% vs 100% (p<0.01) and 18% vs 100% (p<0.001) in MHC-mismatched and MHC-matched systems, respectively.

Conclusion: CMV reactivation after BMT is GVHD-dependent (i.e. immunosuppressant independent) and is inhibited by IFN γ signaling.

Abstract No. 35**AZATHIOPRINE AND THE RISK OF SKIN CANCER IN SOLID ORGAN TRANSPLANT RECIPIENTS: A SYSTEMATIC REVIEW AND META-ANALYSIS****ISBEL Nicole¹**, JIYAD Zainab², OLSEN Catherine², BURKE Michael³, GREEN Adele²¹*Renal Transplant Unit, Princess Alexandra Hospital, Brisbane,* ²*Cancer and Population Studies, Queensland Institute of Medical Research, Brisbane,* ³*Renal & Transplantation Unit, Princess Alexandra Hospital, Brisbane*

Aim: To examine the risk of squamous cell carcinoma (SCC) and other skin cancers in solid organ transplant recipients treated with azathioprine.

Methods: A systematic literature search of Medline (PubMed and Ovid), EMBASE, CINAHL and The Cochrane library was conducted, in addition to reference hand searching and citation searching using the Web of Science. Data from eligible studies was extracted according to pre-defined criteria and recorded in a data extraction table. Quality assessment of included studies was performed and a random-effects meta-analysis model was used to calculate the pooled estimate for the three skin cancer outcomes: SCC, basal cell carcinoma (BCC) and keratinocyte cancer (KC = SCC and BCC and SCC in-situ).

Results: 568 articles were identified from the systematic search and 27 eligible studies were included in the final analysis. Risk estimates from 13 studies were pooled together for the quantitative analysis. The overall summary estimate showed a significantly increased risk of SCC in relation to azathioprine exposure (1.56, 95% CI 1.11-2.18). No significant association between azathioprine treatment and BCC (0.96, 95% CI 0.66-1.40) or KC (0.84, 95% CI 0.59-1.21) risk was identified. There was significant heterogeneity between studies for azathioprine risk estimates and the outcomes of SCC, BCC and KC.

Conclusion: The findings suggest that treatment with azathioprine increases the risk of SCC by 56%. No association was found between azathioprine and BCC or KC risk. There was considerable variation in azathioprine exposure definitions, regimens and skin cancer definitions between studies.

Abstract No. 36

CRYPTOCOCCAL INFECTIONS IN SOLID ORGAN TRANSPLANT RECIPIENTS OVER A 15 YEAR PERIOD IN QUEENSLAND

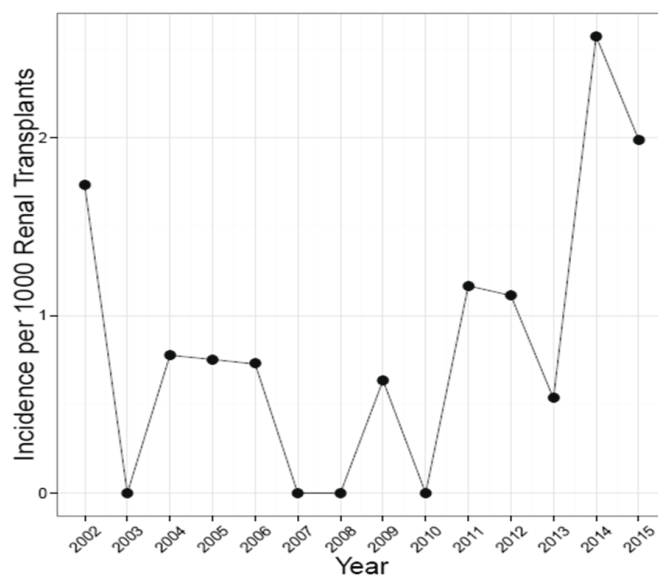
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Aim: To determine the incidence, risk factors and clinical outcome of solid organ transplant recipients diagnosed and treated for cryptococcosis at Princess Alexandra Hospital in Brisbane.

Methods: Retrospective analysis of all patients with solid organ transplant (SOT) diagnosed and treated for cryptococcal infection occurring between January 2001 and December 2015.

Results: Of 102 patients diagnosed with cryptococcal infection, 21 were SOT recipients. Renal transplant accounted for 20/21 cases. The annual incidence of infection has risen significantly, and is now greater than 2/1000 prevalent renal transplant recipients. There was no statistically significant difference between meningitis vs. non-meningitis cohorts with regards to patient demographic, clinical presentation or treatment outcomes.



Conclusion: Cryptococcal infection in solid organ transplant recipients remains rare, however there has been a marked increase in incidence since 2014. This study reveals a need for increased vigilance for a potential emerging infectious disease. It further highlights the need for ongoing research in order to further aid diagnosis, management and prognostication.

Abstract No. 37

INTRODUCTION OF AN IMMUNISATION SCHEDULE FOR ADULT RENAL TRANSPLANT PATIENTS IN NEW ZEALAND**KARA Tonya¹, MANLEY Paul R², CROSS Nicholas B^{3,4}**¹*Department of Nephrology, Starship Children's Hospital,* ²*Auckland Renal Transplant Group, Auckland City Hospital,* ³*National Renal Transplant Service,* ⁴*Department of Nephrology, Canterbury District Health Board*

Aims Death from infection remains a significant cause of mortality for patients in ANZDATA. Improving access to vaccination both pre and post transplant could potentially reduce this. In 2014 members of the National Renal Transplant leadership group with other nephrologists undertook a review of vaccine provision worldwide for this patient group and a comparison with those funded for transplant patients in New Zealand

Methods All published vaccine schedules for patients pre and post transplant were reviewed to determine best practice. The disparities between this and the vaccines funded were discussed with representative of the immunization advisory group for Pharmac, the national funding body for medications, along with evidence regarding the timing of vaccination, and ring fencing.

Results There were differences in the provision of pneumococcal, varicella, HPV, Hepatitis A and meningococcal vaccinations, particularly in those awaiting transplant where vaccination may be more effective. Pharmac approved funding and additional vaccinations were made available to renal transplant patients from July 2015. We designed a schedule with immunization experts specifically for adult renal patients to provide information at a glance for patients and all health care professionals involved in their care. This has been distributed via the national nephrology group, by the Kidney Health New Zealand website and to GPs via NZ Doctor

Conclusion We have widened the funded vaccines available for renal transplant patients and have introduced the first immunization schedule for a specific disease group in New Zealand.

Abstract No. 38

EXTENDED PROPHYLAXIS WITH VALGANCICLOVIR REDUCES THE INCIDENCE OF CYTOMEGALOVIRUS DISEASE IN RENAL TRANSPLANT RECIPIENTS**STRAW S¹, HUGHES P², MASTERSON R², COHNEY SJ^{2,3}**¹*Department of Medicine, Royal Melbourne Hospital,* ²*Department of Nephrology, Royal Melbourne Hospital,* ³*Department of Nephrology, Western Hospital*

Cytomegalovirus (CMV) disease is the commonest life-threatening opportunistic infection following renal transplantation, remaining a major source of morbidity, despite regimens aimed at preventing disease. Here we report CMV outcomes in 469 consecutive patients undergoing renal transplantation from 2004 to 2009 following adoption of a protocol in which valganciclovir prophylaxis was extended to 6 months for seronegative recipients receiving kidneys from seropositive donors (D+/R-), and 3 months for D+/R+ and D-/R+. Over 90% received tacrolimus/mycophenolate/prednisolone based immunosuppression with CMV disease occurring in 14 patients (3%) at a median 248 days post-transplant: 8 of 63 D+/R- (12.7%), 4 of 229 D+/R+ (1.7%), 1 of 95 D-/R+ (1.1%) and 1 of 82 D-/R- (1.2%). Of these 14 patients, 6 had received additional immunosuppression for rejection, and one had received additional high dose steroid for recurrent FSGS. By comparison, in 203 patients transplanted from 2000 to 2004 when prophylaxis was restricted to D+/R- & consisted of 3 months of valganciclovir and 2 months CMV immunoglobulin (CMVlg) 21 patients developed CMV disease (10.3%) at a median of 125 days post-transplant: 10 of 46 D+/R- (21.7%), 9 of 103 D+/R+ (8.7%), 1 of 32 D-/R+ (3.1%), 1 of 22 D-/R- (4.5%), $p < 0.001$ (Table 1). Extending valganciclovir prophylaxis to 6 months in D+/R- and 3 months for D+/R+ and D-/R+ reduced the incidence of CMV disease compared to the previous valganciclovir based regimen. These results also compare favourably to recent published series and may additionally reflect exposure to lower levels of immunosuppression.

Table 1. CMV disease within D/R subgroups, comparing current and previous prophylaxis regimens

Group	2000-2004 Study		2004-2009 Study		p-value
	n	CMV (%)	n	CMV (%)	
Total	203	21 (10.3)	469	14 (3.0)	<0.001
D+/R-	46	10 (21.7)	63	8 (12.7)	
D+/R+	103	9 (8.7)	229	4 (1.7)	
D-/R+	32	1 (3.1)	95	1 (1.1)	
D-/R-	22	1 (4.5)	82	1 (1.2)	

Abstract No. 39

OBESITY IS A RISK FACTOR FOR CMV DISEASE IN RENAL TRANSPLANT RECIPIENTS RECEIVING EXTENDED PROPHYLAXIS WITH VALGANCICLOVIR**STRAW SC¹, HUGHES P², MASTERSON R², COHNEY SJ^{2,3}**¹*Department of Medicine, Royal Melbourne Hospital,* ²*Department of Nephrology, Royal Melbourne Hospital,*³*Department of Nephrology, Western Hospital*

Cytomegalovirus (CMV) disease remains a major source of morbidity with potentially fatal consequences despite a number of regimens aimed at prevention. Historically, a variety of risk factors have been identified including donor age, rejection episodes and impaired transplant function, though the major risk factors remain the extent of immunosuppression and the CMV serostatus of donor (D) and recipient (R). In this study, we examined risk factors for CMV disease amongst 469 consecutive ESKD patients undergoing renal transplantation from 2004 to 2009 managed according to a protocol in which CMV prophylaxis with valganciclovir was 6 months for D+/R-, and 3 months for D+/R+ & D-/R+. More than 90% of patients received a regimen based on tacrolimus, mycophenolate and prednisolone. CMV disease occurred in only 3% of patients (14 of 469), with a rate of 12.7% amongst D+/R-. As expected CMV disease was more common in D+/R-, following treatment for rejection, and occurred in 2 out of 6 patients receiving the janus kinase 3 inhibitor (Jak3i) tofacitinib. None of the 25 patients receiving mTOR inhibitors developed CMV disease. Additional risk factors were number of HLA mismatches, and weight prior to transplant (Table 1). Despite low rates of CMV disease with this prophylaxis protocol, HLA mismatches, rejection and pre-transplant weight remain as significant risk factors for CMV disease. This provides an additional reason to counsel patients to lose weight prior to transplantation.

Table 1. Baseline characteristics

Characteristic	No CMV disease post-transplant, n=455 (%)	CMV disease post-transplant, n=14 (%)	p-value
Gender			0.779
Female	165 (36.3)	4 (28.6)	
Male	290 (63.7)	10 (71.4)	
Age, Mean \pm SD	47.4 \pm 12.6	52.9 \pm 12.1	0.110
Weight at Transplant (kg), mean \pm SD	74.1 \pm 14.9	83.1 \pm 16.9	0.027
Aetiology of renal disease			0.118
GN	88 (19.3)	3 (21.4)	
DM	35 (7.7)	0	
Hypertensive	14 (3.0)	0	
Polycystic	78 (17.1)	5 (35.7)	
Reflux	56 (12.3)	1 (7.1)	
Interstitial nephritis	4 (0.9)	0	
Vasculitis	7 (1.5)	2 (14.3)	
Obstructive	5 (1.0)	0	
Other	168 (36.9)	3 (21.4)	
Number of grafts			0.789
1 graft	377 (82.9)	13 (92.9)	
> 1 graft	78 (17.1)	1 (7.1)	
Donor source			0.093
Cadaveric	189 (41.5)	9 (64.3)	
Living donor	266 (58.5)	5 (35.7)	
CMV D/R status			0.001
D+/R-	55 (12.1)	8 (57.1)	
D+/R+	225 (49.4)	4 (28.6)	
D-/R+	94 (20.7)	1 (7.1)	
D-/R-	81 (17.8)	1 (7.1)	
HLA mismatches			
Ave total mismatches \pm SD	3.45 \pm 1.67	4.25 \pm 1.54	0.044
Donor Age, median (IQR)	53 (43 – 61)	57 (48 – 63)	0.382

Abstract No. 40**CMV DISEASE IS NOT ASSOCIATED WITH ANY INCREASE IN GRAFT LOSS OR MORTALITY IN RENAL TRANSPLANT RECIPIENTS RECEIVING EXTENDED PROPHYLAXIS WITH VALGANCICLOVIR****STRAW SC¹, HUGHES P², MASTERSON R², COHNEY SJ^{2,3}**¹*Department of Medicine, Royal Melbourne Hospital,* ²*Department of Nephrology, Royal Melbourne Hospital,*³*Department of Nephrology, Western Hospital*

Cytomegalovirus (CMV) disease is the commonest life-threatening opportunistic infection following renal transplantation. It has remained a major source of morbidity and mortality despite various regimens to prevent disease. In 2004, a protocol was adopted in which CMV prophylaxis was extended to 6 months valganciclovir for seropositive donor (D+) to seronegative recipient (R-), and 3 months for D+/R+ & D-/R+. Amongst 469 consecutive renal transplant recipients managed according to this regimen from 2004 to 2009, CMV disease occurred in 14 patients (3%) at a median 248 days post-transplant, with 7 developing organ specific disease and the remainder vague systemic symptoms. Three were managed as outpatients, while amongst those hospitalised, median length of stay was 5 days (average 8.6 days). One patient required ICU. At a median 1556 days following CMV disease (average 1455), graft loss was 4/14 (28.6%) in patients who experienced CMV disease and 78/455 (17.1%) in those remaining CMV free ($p=0.281$). Subsequent mortality was 2/14 (14.3%) in those with CMV disease and 17/455 (3.7%) in those without ($p=0.106$). In one of the two deaths in the CMV group, CMV viraemia was an intercurrent event in a patient dying from alcoholic liver disease and pancreatitis. Thus, in patients receiving “extended” valganciclovir prophylaxis (6 months in D+/R-, 3 months for D+/R+ and D-/R+) CMV disease was associated with only short hospital stays and was not associated with an increased rate of graft loss or mortality.

Abstract No. 41

CYTOMEGALOVIRUS (CMV) SEROSTATUS, PATIENT AND ALLOGRAFT SURVIVAL AND PATTERNS OF CMV PROPHYLAXIS IN AUSTRALIAN AND NEW ZEALAND KIDNEY TRANSPLANT RECIPIENTS.**WONG Germaine¹, PILMORE Helen², CHADBAN Steve³, LIM Wai⁴**¹*Centre for Kidney Research, The Children's Hospital at Westmead, Sydney,* ²*Auckland Renal Transplant Group, Auckland City Hospital,* ³*Royal Prince Alfred Hospital, Sydney,* ⁴*Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth*

CMV syndrome and disease is the commonest opportunistic infection after kidney transplantation although whether CMV serostatus is associated with survival is controversial.

Aims: We aimed to determine the association between donor and recipient CMV serostatus and patient survival and to investigate the use of CMV prophylaxis within the ANZ kidney transplant population.

Methods: All adult (age > 18 years) transplant recipients who received their first deceased donor kidney transplants between 1990 and 2012 were included. We examined cause specific and all-cause mortality according to CMV donor and recipient serostatus using data from the ANDZATA registry. Additionally we surveyed the ANZSN and TSANZ membership to determine current clinical practice for CMV prophylaxis.

Results: Over a follow-up of 48,742 person-years, 2028 /7513 recipients died. Overall, there was no significant association between CMV serostatus, all-cause and cause-specific mortality (Table 1). However, the association between CMV serostatus and cancer death was modified by human leukocyte antigen (HLA) matching (p-value for interaction < 0.001). Compared to the reference (CMV D+/R+), the adjusted hazard estimates for cancer death in those with CMV D-/R-, D-/R+ and D+/R- were 0.49 (95%CI: 0.25 – 0.95), 1.44 (95%CI: 0.98 – 2.11) and 1.04 (95%CI: 0.68 – 1.61), respectively in recipients with HLA ABDR 0-1 mismatches. Over 80% of transplanted patients who are D+ are given prophylaxis for CMV with 95% using valganciclovir. Most units use prophylaxis for 3 months, however 6 months is prescribed in D+/R- in over half of responses.

Conclusions: The association between CMV serostatus and cancer mortality appears to be modified by the number of HLA broad antigen mismatches. Use of Valganciclovir as prophylaxis is high in ANZ.

Table 1. Adjusted hazard ratios for cause-specific mortality

CMV status	HR	95%CI	P-values
Cardiovascular disease related death*			
D+/R+	ref	ref	
D+/R-	0.85	0.65 – 1.11	0.24
D-/R+	0.99	0.79 – 1.26	0.96
D-/R-	0.94	0.68 – 1.29	0.69
Infection related death #			
D+/R+	ref	ref	
D+/R-	0.88	0.65 – 1.21	0.25
D-/R+	0.96	0.72 – 1.27	0.92
D-/R-	0.78	0.52 – 1.18	0.79
Cancer related death**			
D+/R+	ref	ref	
D+/R-	1.10	0.91 – 1.33	0.35
D-/R+	1.11	0.92 – 1.32	0.27
D-/R-	1.07	0.85 – 1.35	0.59

adjusted for age of the recipient, age of the donor, smoking status, duration on dialysis, HLA ABDR matching, year of transplantation

*adjusted for age of the recipient, age of donor, smoking status, duration on dialysis, HLA ABDR matching, year of transplantation, prior history of cancer, cardiovascular disease, diabetes and BMI

**adjusted for age of the recipient, age of donor, smoking status, duration on dialysis

Abstract No. 42

RENAL TRANSPLANTATION AND THE INCIDENCE OF BK AT 3 MONTHS POST TRANSPLANT
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Background: Polyoma BK virus infection results in latency in the urothelium and can lead to viraemia and subsequent nephropathy following kidney transplantation. BK nephropathy is a significant cause of long term graft dysfunction and graft loss

Aim: To assess the utility of testing for BK infection as part of a 3 month screening protocol.

Method: A retrospective review of BK screening by PCR and biopsy was undertaken in 363 patients transplanted at a single centre (January 2010 - September 2015). Patients had a minimum of 3 month followup. Graft and patient outcomes were examined.

Results: 363 patients were screened. 28 (7.7%) patients had BK viremia. Of these 10 (35.7%) were low positive (< 1000 copies) and a further 18 (64.3%) had intermediate or high levels. 6 patients in the latter group (33%) had BK nephropathy on biopsy being 1.7% of screened cohort.

Patients who did not have nephropathy were monitored fortnightly and where possible immunosuppression was reduced. All returned to low positive or negative in time. Patients with BK nephropathy were treated with cidofovir +/- leflunamide.

4 patients cleared the BK virus on PCR., 1 patient had ongoing low positive viraemia but no nephropathy, whilst 1 patient died due to cardiovascular complications during the treatment period with cidofovir and leflunamide.

Conclusion: Screening for BK infection routinely at 3 months resulted in detection of viraemia and nephropathy which was responsive to therapeutic intervention. Protocolised screening at this timepoint is likely to have prevented subsequent development of clinically significant BK infection.

Abstract No. 43

RESULTS OF A BIOPSY BASED, EARLY INTERVENTION TREATMENT PROTOCOLS FOR BK VIRAEMIA AND BK VIRUS ASSOCIATED NEPHROPATHY.**JONES Scott¹, TREVILLIAN Paul¹, LAI Katy², WALLER Sophie³, HEER Munish¹, HIBBARD Adrian¹**¹Renal Transplant Unit, John Hunter Hospital, Newcastle, ²Department of Microbiology, John Hunter Hospital, Newcastle, ³Department of Medicine, John Hunter Hospital, Newcastle

Background: This study reports our experience (January 2006 – 30 June 2015) with protocols for early detection and intervention with BK Viremia and BKVAN.

Methods: Patients detected to have BKviraemia or decoy cells had transplant biopsies. Biopsies were also performed on all patients at 3 months. Those treated for BKVAN were re-biopsied after each course of treatment. Management of BKViremia involved substitution of Mycophenolate with Leflunomide, halving Tacrolimus dose and corticosteroid reduction. The BKVAN protocol required addition of Cidofovir (0.25-0.50 mg/kg IVI fortnightly x 4 doses). A second course of Cidofovir with IVIG was given for persistent BKVAN. If the index biopsy was deemed to show likely acute rejection, pulsed Methylprednisolone or Thymoglobulin/PLEX/IVIG was given prior to the BK protocol.

Results: BKViremia was detected in 57/264(21.7%), of which 37/57(64.9%) had decoy celluria. 22/264(38.6%) had biopsy proven BKVAN. 11/22 patients required a single course of Cidofovir; 7/22 patients completed a second course; 4 patients had an aborted course because of SAE's (neutropenia=4, AKI=1). BKViremia clearance rate is shown in table 1. Creatinine/eGFR at index biopsy and 6 months later improved in 14/2 (636%), unchanged in 4/22 (18.2%) and deteriorated in 4/22(18.2%) of patients. No grafts were deemed to be lost from BKVAN. Two grafts were subsequently lost from chronic rejection however both having cleared the virus prior. There was 1 death unrelated to BKV.

Conclusion: Biopsy based, early intervention for BKViremia and BKVAN results in viral clearance and improved graft function in the majority of affected patients.

Table 1: BK Viremia clearance rate after protocol treatment

	BK Virus not detected	BK Virus detected below cut off	BK virus detected above cut off
BK Viremia: 57 Patients	38(66.7%)	11(19.3%)	8(14%)
BKVAN: 22 patients	15(68.2%)	4(18.2%)	3(13.6%)

Xenotransplantation & Cells/Tissues-Experimental

Abstract No. 44

GENERATION OF KNOCK-IN PIGS FOR XENOTRANSPLANTATION USING A HIGH FIDELITY CRISPR/CAS9 SYSTEM

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Background: Successful xenotransplantation will likely depend on genetically modifying the donor pig to attenuate the antibody, coagulation and T cell responses that result in xenograft rejection. CRISPR/Cas9 genome editing can be used to target *GGTA1*, the gene for the predominant porcine xenoantigen α Gal, and simultaneously introduce xeno-protective transgenes.

Aim: To generate *GGTA1* knock-in pigs using the high fidelity *FokI*-dCas9 CRISPR system.

Methods: Fetal pig fibroblasts were co-transfected with *FokI*-dCas9, 2 guide RNAs targeting *GGTA1* and either human thrombomodulin (hTBM) or anti-CD2 monoclonal antibody (CD2hb11) knock-in constructs. Gene editing in stable transfectants was determined at the DNA level using Surveyor nuclease assay and confirmed by PCR and sequencing. Expression of α Gal and hTBM was determined by FACS using IB4 lectin and anti-hTBM, respectively. Anti-CD2 secretion was detected by incubation of culture supernatants with human T cells, using labeled anti-human IgG as the secondary antibody. Knock-in clones were used for somatic cell nuclear transfer (SCNT).

Results: 12 and 240 stable transfectants were isolated using the anti-CD2 and hTBM knock-in constructs, respectively. 1/12 and 11/240 clones expressed anti-CD2 or hTBM and lacked expression of α Gal. PCR and DNA sequencing confirmed correct insertion of the knock-in constructs. To date, 3 rounds of SCNT have been performed using the anti-CD2 knock-in clone.

Conclusion: We have generated knock-in pig cell clones that lack α Gal expression and express either anti-CD2 or hTBM. These clones are being used to generate genetically modified pigs which will be tested in preclinical xenotransplantation models.

Abstract No. 45

GTKO/CD55-CD59-HT PORCINE NEONATAL ISLET CELL CLUSTER (NICC) XENOGRAFTS PROVIDE LONG-TERM REVERSAL OF DIABETES

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Aim: Transgenic expression of human complement regulatory proteins or deletion of α Gal (GTKO) has been shown to protect porcine islet xenografts in monkeys treated with costimulation blockade-based immunosuppression. However, this has not been examined in the immunologically more challenging baboon model. We investigated the combined effects of these modifications on the outcome of intraportal NICC transplantation in immunosuppressed baboons.

Method: 1-5 day old GTKO piglets transgenic for human CD55, CD59 and H-transferase were used as donors. Recipient baboons received GTKO/CD55-CD59-HT NICC under standard (ATG, tacrolimus, mycophenolate mofetil; n=5) or costimulation blockade-based immunosuppression (anti-CD2, anti-CD154, belatacept, tacrolimus; n=4). Graft survival and function was followed by daily blood sugar levels, IVGTT and graft immunohistochemical analysis for up to 12 months post-transplant.

Results: GTKO/CD55-CD59-HT xenografts exhibited no signs of early thrombosis or infiltrate, nor changes to recipient platelet counts, fibrinogen and D-dimer levels from baseline. However, liver biopsies from recipients under standard immunosuppression revealed rejection of NICC within one month. Changing to costimulation blockade-based immunosuppression reduced cellular infiltration, and cells staining positive for insulin, glucagon and somatostatin were present in all xenografts at three months. On the extended protocol the two animals performed thus far have normal glucose handling out to as far as one-year post transplant.

Conclusions: Deletion of α Gal and expression of human CD55 and CD59 prevent early thrombotic destruction of porcine NICCs in the baboon model. Costimulation blockade-based immunosuppression appears to be more effective than standard immunosuppression in prolonging the survival of genetically modified porcine NICC xenografts.

Abstract No. 46

PROTECTION FROM INSTANT BLOOD MEDIATED INFLAMMATORY REACTION IN GAL-KO PORCINE NEONATAL ISLET CELLS EXPRESSING COMPLEMENT REGULATORS CD55/CD59**LIUWANTARA David¹, CHEW Yi Vee², FAVALORO Emmanuel³, HAWKES Joanne¹, BURNS Heather¹, NOTTLE Mark⁴, COWAN Peter⁵, O'CONNELL Philip¹, HAWTHORNE Wayne¹**¹Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney,²Centre for Transplant and Renal Research, Westmead Hospital, Sydney, ³Institute of Clinical Pathology and Medical Research (ICPMR), Westmead Hospital, Sydney, ⁴Department of Obstetrics and Gynaecology, University of Adelaide, ⁵Immunology Research Centre, St Vincent's Hospital, Melbourne

Clinical islet cell transplantation is a viable treatment option for type-1 diabetes. Unfortunately the lack of donor pancreata remains a major hurdle to offering this treatment to larger groups of patients. Porcine Neonatal Islet Cell Clusters (NICC) are a suitable alternative to human islets. However, hyperacute rejection (HAR) and the Instant Blood Mediated Inflammatory Reaction (IBMIR) pose major problems following NICC transplantation.

Aim: To explore the benefits of using galactosyltransferase knockout (gal-KO) or gal-KO NICC expressing the complement regulators CD55/CD59 transgene (gal-KO CD55/59-tg), on xenogeneic IBMIR *in vitro*.

Methods: IBMIR was compartmentalised *in vitro*, to measure independently, the effect of thrombosis, complement and neutrophil activation on wild-type vs. transgenic NICC.

Results: The peak thrombin concentration and thrombin time were similarly elevated between gal-KO (198.3±83.1 nM; 15.0±4.4 minutes) and wild-type NICC (188.6±29.3 nM; 16.2±3.9 minutes), but the combination of gal-KO CD55/59-tg NICC provided significantly delayed thrombin generation (23.9±6.2 minutes, $p<0.05$) and reduced peak thrombin levels (69.9±27.7 nM, $p<0.05$). Formation of complement C3a was significantly reduced when gal-KO (76.9±3.6 ng/mL) or gal-KO CD55/59-tg (67.1±9.5 ng/mL) NICC was exposed to human blood compared to WT NICC (542.7±334.5 ng/mL; $p<0.05$). Furthermore, significant reduction in neutrophil activation was observed in both gal-KO and gal-KO CD55/59-tg NICC compared to WT NICC (WT: 1.7±0.2-fold, gal-KO: 0.89 ±0.1-fold or gal-KO CD55/59: 1.1±0.02-fold, $p<0.05$).

Conclusions: Our data demonstrated that whilst removing the galactosyltransferase gene partially protected NICC against IBMIR, additional regulators such as the complement inhibitors CD55 and/or CD59 is critical to ensure successful NICC transplantation.

Abstract No. 47

IL-28 IS A CRITICAL CYTOPROTECTANT IN TRANSPLANTATION**HENDEN Andrea^{1,2}, GARTLAN Kate¹, LANE Steven^{3,2}, ROBB Renee¹, KUNS Rachel¹, CLOUSTON Andrew⁴, HILL Geoff^{1,2}**¹Transplantation Laboratory, Queensland Institute of Medical Research, Brisbane, ²Department of Haematology and Bone Marrow Transplantation, Royal Brisbane Hospital, ³Gordon and Jessie Gilmour Leukaemia Research Laboratory, Queensland Institute of Medical Research, Brisbane, ⁴Histopathology, Envoi Pathology, Brisbane

Aims: We have demonstrated a protective role for type I Interferons (IFN) through inhibition of Th1 differentiation invoked by recipient CD8^{neg} dendritic cells after experimental bone marrow transplantation. Recently described Type III IFNs (IFNλ/IL-28) signal through the unique IL-28R primarily expressed in epithelial tissues and implicated in mucosal pathogen defence. Clinical use of type I IFN is associated with adverse neurological, haematological and constitutional symptoms where IL-28 is better tolerated, yet still demonstrates potent anti-viral effects.

Methods: We used IL-28R^{-/-} and IFNαR1^{-/-} donors and recipients in murine models of GVHD and GVL to develop logical therapeutic strategies to improve transplant outcomes.

Results: IL-28R^{-/-} donors invoked similar GVHD and GVL to WT. However IL-28R^{-/-} recipients had accelerated acute GVHD (aGVHD) mortality and disease relative to WT with a phenotype intermediate to that and the hyperacute aGVHD seen in IFNαR1^{-/-} recipients (median survival WT 42 vs. IL28R^{-/-} 26 vs. IFNαR^{-/-} 6.5 days, $p<0.0001$). IL28R^{-/-} recipients have augmented colonic GVHD histopathology early (*d* 7) after BMT (WT 7.111±0.6550 vs. IL28R^{-/-} 12.33±0.7993, $p=0.0004$) and exaggerated inflammatory cytokine generation (*d*4 IFNγ WT 331±41.47pg/mL vs. IL28R^{-/-} 667±48.79 pg/mL, $p<0.0001$ and IL-6 WT 61.25±10.91pg/mL vs. IL28R^{-/-} 91.99±11.23pg/mL, $p=0.024$). Re-transplantation of chimeras with WT or IL28R^{-/-} haematopoietic, non-haematopoietic tissue or combinations thereof demonstrated that IL-28 mediated protection required signalling through both compartments, putatively recipient antigen presenting cells and colonic epithelia.

Conclusion: IL-28 represents an attractive therapeutic to mediate cytoprotection within the GI tract and attenuate to GVHD in the peri-transplant period.

Abstract No. 48

HEMATOPOIETIC STEM CELLS AND THEIR PROGENITORS CRITICALLY REQUIRE AUTOPHAGY TO PROMOTE EARLY ENGRAFTMENT FOLLOWING ALLOGENEIC STEM CELL TRANSPLANTATION**LINEBURG Katie E¹, LEVEQUE-EL MOUTTIE Lucie¹, LE TEXIER Laetitia¹, TEAL Bianca¹, KUNS Rachel¹, LANE Steven W^{1,2}, HILL Geoffrey R^{1,3}, MACDONALD Kelli PA¹**¹*Department of Immunology, Queensland Institute of Medical Research, Brisbane*, ²*Department of Haematology, Royal Brisbane Hospital*, ³*Department of Bone Marrow Transplantation, Royal Brisbane Hospital*

Hematopoietic Stem Cells (HSC) are critical for the success of stem cell transplantation. Autophagy is an intracellular process that has an established role in the long-term survival and function of HSCs. We investigated the contribution of autophagy to HSC in the setting of allogeneic transplantation, in which GVHD results in a T cell derived cytokine storm early post-transplant. We demonstrate increased autophagy in donor HSC and progenitor cells in the setting of GVHD compared to non-GVHD. Competitive transplant experiments of 1:1 Atg5^{-/-} fetal liver (FL) with WT FL demonstrated that autophagy deficient cells display reduced capacity to reconstitute. In an MHC mismatch model of GVHD we demonstrated that while Atg5^{-/-} cells are capable of engraftment they are overcome in the presence of alloreactive T cells and undergo primary graft failure by day 10 post-transplant while WT cells survive and engraft. We confirmed this early graft failure in a second model, using donor VAV^{cre}xAtg7^{fl/fl} mice. The essential requirement for autophagy, specifically in early progenitors and HSC, was confirmed using LysM^{cre}xAtg7^{fl/fl} mice. We demonstrate that autophagy is increased in the GVHD setting and that without autophagy early myeloid precursors fail to provide short term reconstitution leading to primary graft failure and mortality. This primary graft failure can be rescued by the administration of cyclosporine, which works to dampen the T cell induced cytokine storm post-transplant. Thus intervention to increase autophagy in these cells post-transplant may improve engraftment in the clinic.

Abstract No. 49

TRANSGENIC EXPRESSION OF HUMAN THROMBOMODULIN INHIBITS HMGB1-INDUCED PIG AORTIC ENDOTHELIAL CELL ACTIVATION**BONGONI Anjan Kumar¹, WOLF Eckhard², AYARES David³, RIEBEN Robert⁴, COWAN Peter^{1,5}**¹*Immunology Research Centre, St Vincent's Hospital, Melbourne*, ²*Institute of Molecular Animal Breeding and Biotechnology, Ludwig-Maximilian University, Munich, Germany*, ³*Revivicor, Inc., Blacksburg, Virginia, USA*, ⁴*Department of Clinical Research, University of Bern, Bern, Switzerland*, ⁵*Department of Medicine, St Vincent's Hospital, Melbourne*

Background: Transgenic expression of human thrombomodulin (hTBM), a species-specific vascular anti-coagulant, has the potential to solve the problem of coagulation dysregulation in pig-to-primate xenotransplantation. However, the further benefits of hTBM via its anti-inflammatory properties, notably against the pro-inflammatory cytokine high-mobility group box 1 (HMGB1), have not been examined.

Aim: To test HMGB1-mediated effects on wild-type (WT) porcine aortic endothelial cells (PAEC), and to assess the capacity of hTBM on PAEC to neutralize HMGB1.

Methods: WT and hTBM-transgenic PAEC were treated with HMGB1, hTNF α or lipopolysaccharide (LPS) and analysed for expression of cell surface markers, secretion of porcine cytokines and chemokines, and formation of tPA/PAI-1 complexes. Thrombin-induced HMGB1 cleavage in the presence of PAEC was examined by western blot and functional assays.

Results: HMGB1 potently activated WT PAEC, increasing the surface expression of E-selectin, VCAM-1, ICAM-1, FGL2 and PAI-1, and the secretion of TNF α , IL-8, and MCP-1. hTNF α - or LPS-induced activation of WT PAEC was inhibited by treatment with rabbit anti-HMGB1 antibody. Transgenic hTBM significantly attenuated HMGB1- or hTNF α -induced PAEC activation, and significantly enhanced thrombin-mediated HMGB1 cleavage. Removal of the lectin-like domain of TBM resulted in significantly increased HMGB1- or hTNF α -induced PAEC activation.

Conclusion: HMGB1 stimulated powerful pro-inflammatory and pro-coagulant effects on WT PAEC. Transgenic hTBM, via its lectin-like domain, significantly inhibited HMGB1-mediated actions on PAEC and increased thrombin-induced degradation of HMGB1 to a less proinflammatory form. These results indicate that transgenic hTBM has anti-coagulant and anti-inflammatory effects that are likely to be beneficial in pig-to-primate xenotransplantation.

Abstract No. 50

CONJUGATION OF APYRASE TO PORCINE AORTIC ENDOTHELIAL CELLS PROLONGS CLOTING OF WHOLE HUMAN BLOOD

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Background: Recipient platelets play an important role in the dysregulated coagulation that is frequently observed in pig-to-primate xenotransplantation. ADP is essential for activation of platelets and coagulation, and is an obvious target for platelet inhibition.

Aim: To investigate the impact of surface immobilization of the ATP/ADP-degrading enzyme apyrase on porcine aortic endothelial cells (PAEC) in xenotransplantation-induced thrombosis.

Methods: PEG-conjugated phospholipid was used to immobilize apyrase on the PAEC surface. The enzymatic activity of apyrase-PAEC was evaluated as ATPDase activity. The anti-coagulant properties of apyrase-PAEC were tested using a microcarrier bead-based coagulation assay with freshly drawn non-anticoagulated whole human blood in a proportion that closely mimics the in vivo small vessel endothelial surface-to-blood volume ratio.

Results: Immobilization of Alexa 488-labeled apyrase on PAEC was confirmed by immunofluorescence/confocal microscopy. Beads coated with apyrase-PAEC degraded exogenously added ATP in a dose-dependent manner (ATPDase activity: 9.9 ± 1.1 nmol/20min at $10 \mu\text{g/ml}$ apyrase; 17.7 ± 0.6 at $25 \mu\text{g/ml}$). Immobilized apyrase retained approximately 81% of the activity of the native form. Apyrase-PAEC significantly prolonged clotting of human blood (70.4 ± 17.7 min, $p < 0.001$ vs. untreated PAEC: 32.5 ± 6.9 min). The concentration of markers of complement activation (sC5b-9) ($p = 0.015$), coagulation activation (thrombin-antithrombin complex and D-dimer) (both, $p < 0.0001$) and inhibition of fibrinolysis (tPA/PAI-1) ($p < 0.0001$) was lower in EDTA-plasma of coagulation assays with apyrase-PAEC than untreated PAEC.

Conclusion: Apyrase-immobilized PAEC inhibit xenogenic platelet activation and suppress coagulation in a whole blood assay. This assay can be used to evaluate the potential of novel therapeutic substances not only in xenotransplantation but also in allotransplantation and ischemia/reperfusion injury.

Abstract No. 51**A NEW PORCINE MODEL OF NORMOTHERMIC MACHINE PERFUSION OF LIVER DONATION AFTER CIRCULATORY DEATH: A PRELIMINARY STUDY****CAO Yiming, CHEW HC, FERNANDEZ Karen, VILLANUEVA Jeanette, GAO Ling, HICKS Mark, MACDONALD Peter, DHITAL Kumud, PLEASS Henry*****Transplantation Laboratory, Victor Chang Cardiac Research Institute, Sydney***

Aims: Interventions aimed at improving liver donations after circulatory death (DCD) are under active investigation on account of poor outcomes of human DCD liver recipients. However, current large animal models induce circulatory death by either exsanguination or potassium chloride injection, neither of which are practised clinically. We sought to develop a clinically relevant model of DCD liver retrieval followed by normothermic machine perfusion (NMP).

Methods: Landrace pigs (60-70 kg) were anaesthetised and intubated. To mimic clinical practice, cessation of mechanical ventilation was used to induce circulatory death, which was confirmed by ECG electrical silence or disappearance of pulse pressure. A 5 min stand-off period was instituted prior to cold preservation flush of the liver and explantation. The liver was subsequently prepared on the backtable for machine perfusion.

Results: 12 donor animals underwent this protocol. Average time to asystole was 9.27 min. Average warm ischaemic time, as defined from ventilation cessation to cold preservation flush, was 20.25 min. Livers were maintained on machine perfusion for 4 hours, exhibiting favourable aminotransferase release, lactate and pH profiles and bile production.

Conclusions: our preliminary results indicate that porcine livers can be viably explanted and machine perfused under a clinically relevant model. Further research is required to identify optimal time of NMP and potentially optimise liver preservation with improved perfusion parameters and pharmacological reconditioning agents in perfusate.

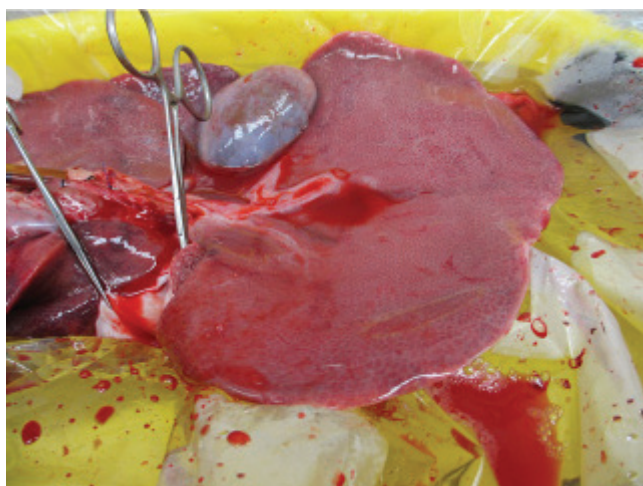


Figure 1. Successfully machine perfused porcine liver

Abstract No. 52

**MICROARRAY GENE PROFILING OF IMMUNOSUPPRESSIVE INTERLEUKIN-17A
PREACTIVATED HUMAN BONE MARROW-DERIVED MESENCHYMAL STEM CELLS (MSC-17)
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MSC-17 are superior T cell immunomodulators for clinical allotransplantation.

Methods: Untreated-MSC (UT-MSC) or 5-days IFN- γ (MSC- γ) or IL-17A (MSC-17) treated MSC were assessed for T cell immunosuppression and ability to induce Tregs. Immunophenotype (flow cytometry) and gene expression profile (microarray) of 3 MSC donors were also analyzed. Significantly regulated genes ($p < 0.05$, fold change (FC) < -2 or > 2) were identified for their biological functions (Database for Annotation, Visualisation and Integrated Discovery, DAVID).

Results: MSC-17 potently suppressed PHA-induced T cell proliferation (^3H -thymidine) and T cell activation (downregulated CD25, IFN- γ , TNF- α , IL-2) by inducing Tregs expressing functional Treg markers (CD39, CD73, CD69, OX40, CTLA-4, GITR). Different to MSC- γ , MSC-17 showed no upregulation of MHC or CD40 molecules. Microarray analyses identified 1278 differentially regulated genes (902 upregulated; 376 downregulated) between MSC- γ and UT-MSC; and 67 genes (39 upregulated; 28 downregulated) between MSC-17 and UT-MSC. MSC- γ were enriched for genes involved in immune response, antigen processing and presentation, humoral response and complement activation; consistent with increased MSC- γ immunogenicity. MSC-17 genes were associated with chemotaxis response, which may be involved in T cell recruitment for MSC-17 immunosuppression. MSC are known to express MMP with chemotaxis and immunosuppressive properties. MMP13 was highly expressed specifically in MSC-17 (FC 15.6) and was validated by RT-PCR. Hence, MMP13 may mediate the superior immunomodulatory function of MSC-17.

Conclusion: MSC-17 represent a potential cellular therapy to suppress immunological T cell responses in allotransplantation, with minimal immunogenicity. Studies on the functional role of the key candidate molecule MMP13 in MSC-17 immunomodulation are currently underway.

Outcome Measures #2

Abstract No. 53

EFFECT OF PROLONGED ISCHEMIC TIME ON GRAFT AND PATIENT OUTCOMES IN LIVE-DONOR KIDNEY TRANSPLANT RECIPIENTS

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Background: The association between prolonged cold ischaemic time (CIT) and graft and patient outcomes in live-donor kidney transplant recipients remains unclear.

Aim: The aims of this study are to examine the association between CIT, delayed graft function and graft loss in live-donor kidney transplant recipients and those who had participated in the Australian Paired Kidney Exchange program (AKX) using data from the Australia and New Zealand Dialysis and Transplant (ANZDATA) registry.

Methods: 3717 live-donor transplant recipients between 1997-2012 were followed up for a median of 6.6 years (25,977 person-years). The exposure factor was cold ischaemic time. Linearity between CIT and clinical outcomes was examined using restricted cubic spline models, with $p < 0.05$ indicating a deviation from linearity.

Results: Of 3717 live-donor transplant recipients, 224 patients had experienced CIT > 4 hours. Donor age was an effect modifier between CIT and graft outcomes. In recipients who have received kidneys from older donors, every hour increase in CIT was associated with an adjusted odds of 1.28 (95%CI 1.07, 1.53, $p = 0.007$); whereas CIT of > 4 hours was associated with adjusted hazards of 1.93 (95%CI 1.21, 3.09, $p = 0.006$) and 1.91 (95%CI 1.05, 3.49, $p = 0.035$) for overall and death-censored graft loss respectively compared to CIT of 1-2 hours. Recipients in the AKX program ($n = 33$), despite having longer CIT, had favourable short-term outcome with no reported delayed graft function.

Conclusion: Attempts to reduce CIT in live-donor kidney transplants involving older donor kidneys may lead to improvement in graft outcomes.

Abstract No. 54

LONG-TERM GRAFT AND PATIENT OUTCOMES OF KIDNEY TRANSPLANT RECIPIENTS WITH AND WITHOUT TYPE II DIABETES MELLITUS (T2DM)

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Aim: We aimed to determine the association between pre-transplant diabetes status and long-term outcomes in kidney transplant recipients using the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry.

Methods: Primary live and deceased-donor kidney transplant recipients between 1994-2012 were included and associations between pre-transplant diabetes status, all-cause mortality, overall and death-censored graft loss (DCGL) were examined using adjusted Cox regression models. Recipients with new-onset diabetes after transplantation are not identified by ANZDATA registry.

Results: Of 11,056 kidney transplant recipients followed for a median of 6.6 years resulting in 80,855 person-years, 985 (8.9%) had T2DM, of whom 655 (66.5%) had diabetic nephropathy recorded as cause of end-stage renal disease. Compared to recipients without diabetes, recipients with T2DM were associated with adjusted hazard ratios of 1.66 (95%CI 1.43, 1.93) for all-cause mortality; 1.38 (95%CI 1.15, 1.65) and 1.55 (95%CI 1.37, 1.76) for DCGL and overall graft loss respectively, with similar associations being observed across different eras of 1994-99, 2000-05 and 2006-12. Recipients with T2DM but without diabetic nephropathy had a lower risk of all-cause mortality with adjusted hazard ratio of 0.67 (95%CI 0.51, 0.88), particularly CVD mortality with adjusted hazard ratio of 0.41 (95%CI 0.25, 0.67) compared to T2DM recipients with established diabetic nephropathy.

Conclusions: Our findings suggest a continuing survival disadvantage in kidney transplant recipients with T2DM, particularly those with end-stage kidney disease attributed to diabetic nephropathy. This temporal trend is in contrast to improved survival for people with T2DM in the general population.

Abstract No. 55

RELATIONSHIP BETWEEN 12-MONTH ESTIMATED GLOMERULAR FILTRATION RATE AND LONG-TERM GRAFT LOSS AFTER KIDNEY TRANSPLANTATION**LIM WAI¹, WONG GERMAINE², CLAYTON PHIL³, PILMORE HELEN⁴, CHADBAN STEVEN⁵**¹*Sir Charles Gairdner Hospital, Perth*, ²*Westmead Millennium Institute, Westmead Hospital, Sydney*,³*ANZDATA*, ⁴*Auckland City Hospital*, ⁵*Royal Prince Alfred Hospital, Sydney*

Background: Estimated glomerular filtration rate (eGFR) at 12-months after kidney transplantation has been shown to be an independent predictor of graft loss, but it remains unclear whether this relationship is modified by donor types.

Aim: We aimed to determine whether donor types modified the association between 12-month eGFR and overall graft loss.

Methods: Primary live (LD) and deceased donor kidney transplant recipients between 1994-2012 were included in this study. Restricted cubic spline was constructed to establish the linearity of the association between eGFR and overall graft loss. Association between categories of eGFR (<30, 30-44.9, 45-59.9, 60-74.9 and ≥ 75 ml/min/1.73m²) and overall graft loss was examined using adjusted Cox regression analysis.

Results: We categorized donor age/types as live-donor age <55 years (LD<55, n=2297), LD ≥ 55 (n=713), standard criteria deceased donors (SCD, n=5979) and expanded criteria deceased donors (ECD, n=1279). Donor age and type were effect modifiers between 12-month eGFR and graft loss (p-value for three-way interactions 0.003). A greater proportion of LD<55 (34%) and SCD (28%) recipients had eGFR ≥ 60 ml/min/1.73m² at 12 months compared to LD ≥ 55 (13%) and ECD recipients (7%, p<0.001). There was a parabolic association between 12-month eGFR and overall graft loss (deviation from linearity p<0.001). Controlling for model covariates, there was an inverse association between eGFR and graft loss across all donor categories, particularly for LD<55 (Table 1).

Conclusion: The association between 12-month eGFR and overall graft loss is modified by donor age and type. We have shown an inverse relationship between 12-month eGFR and graft loss, which was more pronounced in LD<55. Clinical trials utilizing 12-month eGFR as a surrogate for graft loss must consider the differential impact of donor age/type on eGFR.

Table 1:

12m eGFR in ml/min/1.73m²	LD<55	LD≥ 55	SCD	ECD
eGFR<30	4.24 (2.80, 6.42)	2.29 (0.96, 5.44)	2.89 (2.22, 3.68)	3.95 (1.25, 12.42)
eGFR 30-44.9	2.00 (1.37, 2.88)	0.83 (0.36, 1.92)	1.12 (0.89, 1.42)	1.46 (0.46, 4.62)
eGFR 45-59.9	1.34 (0.94, 1.92)	0.52 (0.22, 1.26)	0.84 (0.66, 1.06)	1.52 (0.47, 4.89)
eGFR 60-74.9	1.02 (0.69, 1.53)	0.34 (0.10, 1.11)	0.86 (0.66, 1.11)	1.98 (0.57, 6.84)
eGFR ≥ 75	1.00	1.00	1.00	1.00

Data expressed as adjusted hazard ratios and 95% confidence intervals. Model adjusted for recipient age, gender, body mass index and era. eGFR – estimated glomerular filtration rate by MDRD equation, LD – live donor, SCD – standard criteria deceased donors, ECD – expanded criteria deceased donors.

Abstract No. 56

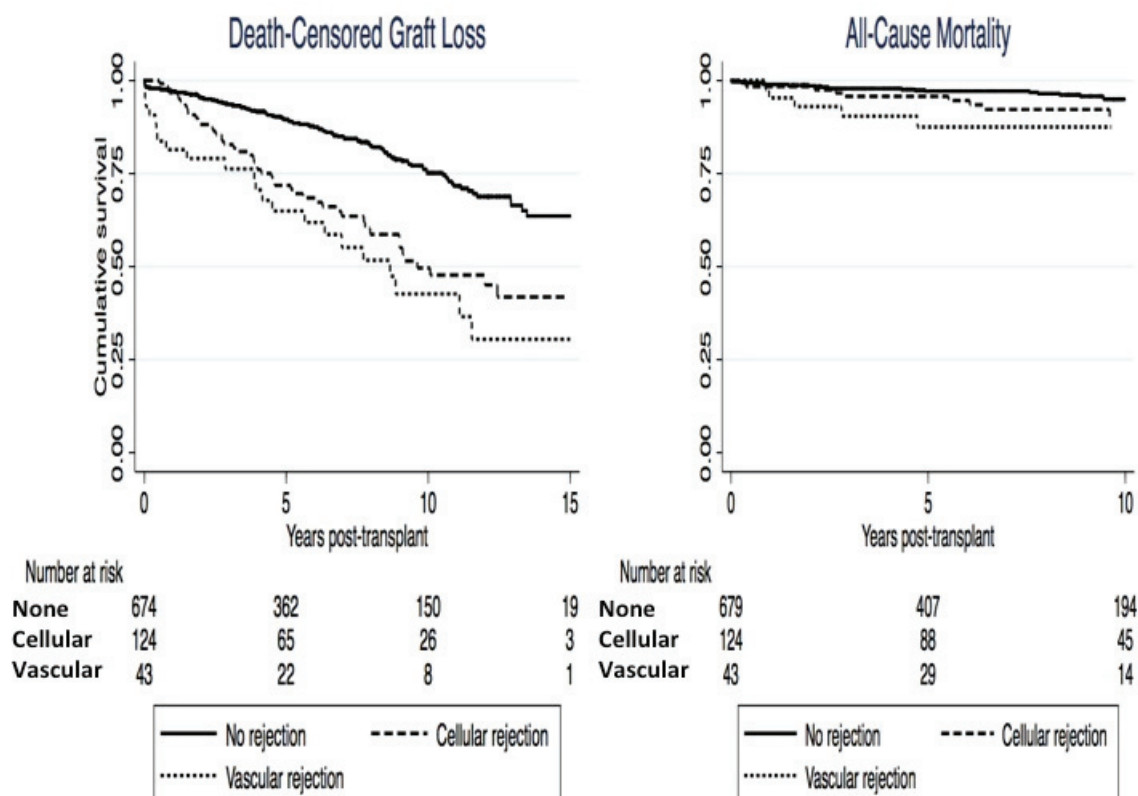
REJECTION, GRAFT LOSS AND DEATH IN PAEDIATRIC AND ADOLESCENT KIDNEY TRANSPLANT RECIPIENTS**LIM WAI¹, KENNEDY SEAN², ALEXANDER STEVE³, WILLIS FRANCIS⁴, WONG GERMAINE⁵**¹*Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth,* ²*Sydney Children's Hospital,* ³*Children's Hospital Westmead,* ⁴*PRINCESS MARGARET HOSPITAL,* ⁵*Westmead Millennium Institute, Westmead Hospital, Sydney*

Aim: We aimed to determine the association between acute rejection (AR) and long-term graft and patient outcomes of paediatric and adolescent kidney transplant recipients using the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry.

Methods: Primary live and deceased-donor paediatric and adolescent kidney transplant recipients aged ≤ 21 years between 1997-2012 were included. Associations between AR, death-censored graft loss (DCGL) and all-cause mortality were examined using adjusted Cox regression models.

Results: Of 846 kidney transplant recipients followed for a median (IQR) of 7.2 (3.1-10.9) years resulting in 6104 person-years, 262 (31.0%) experienced AR. Recipients who had experienced rejection were significantly older compared to those who did not experience rejection (mean [SD] age 14.4 [5.8] vs. 12.6 [6.4], $p < 0.001$). Controlling for model covariates, any rejection was associated with DCGL with adjusted hazard ratio (HR) of 2.17 (95%CI 1.63, 2.89), with similar HRs for early rejection (occurring within first 90 days post-transplant; 1.49 [95%CI 1.06, 2.09]) and late rejection (occurring after 90 days; 2.10 [95%CI 1.52, 2.90]). Increasing number of episodes of AR was associated with an incremental risk of DCGL (referent: no rejection; 1 episode: adjusted HR 1.82 [95%CI 1.28, 2.58]; 2 episodes: adjusted HR 2.48 [95%CI 1.60, 3.84]; and ≥ 3 episodes: adjusted HR 2.97 [1.86, 4.75]). Types of AR were associated with DCGL and all-cause mortality; with those experiencing vascular rejection have the poorest outcomes (adjusted cumulative survival curves below).

Conclusions: There is a strong association between rejection, particularly multiple rejections and vascular rejections and risk of DCGL and/or mortality in paediatric/adolescent kidney transplant recipients.



Abstract No. 57

LONG-TERM GRAFT OUTCOMES OF KIDNEY TRANSPLANT RECIPIENTS WITH PRESUMED GLOMERULONEPHRITIS (GN)**LIM WAI¹, CHADBAN STEVE², LUXTON GRANT³, PILMORE HELEN⁴, WONG GERMAINE⁵**¹*Sir Charles Gairdner Hospital, Perth*, ²*Royal Prince Alfred Hospital, Sydney*, ³*Prince of Wales Hospital, Sydney*, ⁴*Auckland City Hospital*, ⁵*Westmead Millennium Institute, Westmead Hospital, Sydney***Background:** The transplant outcomes of end-stage renal disease (ESRD) patients with clinical diagnosis of presumed GN or uncertain diagnosis are unknown.**Aim:** We aimed to determine the association between known and presumed diagnosis of GN and long-term graft outcomes using the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry.**Methods:** Primary live and deceased-donor kidney transplant recipients between 1994-2012 whose end-stage renal disease was attributed to GN or “uncertain diagnosis” were included. GN was categorized into GN deemed at high risk of recurrence post-transplant (i.e. IgA nephropathy, membranoproliferative GN or primary focal segmental glomerulosclerosis – high-risk GN), other GN and presumed/advanced GN. Associations between GN types and death-censored graft loss (DCGL) were examined using adjusted Cox regression models.**Results:** Of 6056 kidney transplant recipients followed for a median (IQR) of 7.9 (3.6-13.0) years resulting in 52,641 person-years, 2360 (39.0%), 1977 (32.6%), 1195 (19.7%) and 524 (8.7%) have high-risk GN, other GN, presumed/advanced GN and “uncertain diagnosis” respectively. A greater proportion of those with presumed/advanced GN and uncertain diagnosis cause of ESRD were indigenous patients and were older compared to the other GN groups ($p<0.001$). Controlling for model covariates, the associations between GN categories and risk of DCGL and recurrent/de novo GN-related DCGL are shown in Table 1. The proportion of recipients who had experienced graft loss attributed to de novo/recurrent GN, including the types of recorded GN resulting in graft loss are shown in Table 1.**Conclusions:** Recipients with presumed GN or uncertain cause of ESRD have a low risk of DCGL attributed to de novo/recurrent GN.**Table 1.**

Adjusted hazard ratios	DCGL	De novo/recurrent GN-related DCGL
GN categories:		
Other GN	1.00	1.00
High-risk GN	0.86 (0.73, 1.00)*	0.92 (0.64, 1.31)
Presumed/advanced GN	0.93 (0.77, 1.11)	0.34 (0.18, 0.65)*
Uncertain diagnosis	0.67 (0.50, 0.89)*	0.22 (0.07, 0.70)*
Incidence	GN-related graft loss	
GN categories:		
Other GN	n=90 (11.9%): 49 focal sclerosing GN, 18 membranous, 4 IgAN, 5 MPGN	
High-risk GN	n=94 (13.6%): 66 IgAN, 25 MPGN, 2 focal sclerosing GN	
Presumed/advanced GN	n=15 (3.2%): 7 focal sclerosing GN, 3 membranous, 3 IgAN	
Uncertain diagnosis	n=3 (1.5%): 1 IgAN, 1 membranous	

*Data expressed as adjusted hazard ratios and 95% confidence intervals, * $p<0.05$. IgAN – IgA nephropathy, MPGN – membranoproliferative glomerulonephritis.*

Abstract No. 58

RELATIONSHIP BETWEEN CHANGE IN ESTIMATED GLOMERULAR FILTRATION RATE AND LONG-TERM GRAFT LOSS AFTER KIDNEY TRANSPLANTATION**LIM Wai¹, WONG Germaine², CLAYTON Phil³, PILMORE Helen⁴, CHADBAN Steven⁵**¹*Sir Charles Gairdner Hospital, Perth*, ²*Westmead Millennium Institute, Westmead Hospital, Sydney*,³*ANZDATA*, ⁴*Auckland City Hospital*, ⁵*Royal Prince Alfred Hospital, Sydney*

Background: Change in estimated glomerular filtration rate (eGFR) between two time-points after kidney transplantation has been shown to be a strong predictor of graft loss, but it remains unclear whether this association is independent of single time-point eGFR.

Aim: We aimed to determine the association between delta eGFR and single time-point eGFR and overall graft loss.

Methods: Primary live and deceased donor kidney transplant recipients between 1994-2012 were included in this study. Restricted cubic spline was constructed to establish the linearity of the association between delta eGFR and graft loss. Association of delta eGFR between 6-12 months after transplant (\geq -15ml/min decline, -5 to <15ml/min decline, <-5ml/min decline to +5ml/min increase, +>5 to <15ml/min increase and \geq 15ml/min increase), 12-month eGFR and overall graft loss was examined using adjusted Cox regression analysis.

Results: Twelve-month eGFR was an effect modifier of delta eGFR between 6-12 months and overall graft loss (p-value for interaction 0.007). There was a parabolic association between delta eGFR and graft loss (deviation from linearity p<0.001). Controlling for model covariates, there was an inverse association between delta eGFR and overall graft loss, which was more apparent in those with 12-month eGFR of at least 60ml/min/1.73m² (Table 1).

Conclusion: Decline in eGFR between 6-12 months after transplantation, particularly those who have experienced at least a 15% decline, improved the risk stratification of graft loss independent of a single time-point eGFR. In recipients with 12-month eGFR of at least 60ml/min/1.73m², only delta eGFR and not single measurement of eGFR was associated with graft loss.

Table 1	Adjusted hazard ratio (95%CI)	p-value
<u>eGFR <60ml/min/1.73m² (n=7915)</u>		
Delta eGFR		
\geq -15ml/min/1.73m ² decline	1.36 (1.19, 1.56)	<0.001
-5 to <15ml/min/1.73m ² decline	1.12 (0.98, 1.28)	0.104
<-5ml/min/1.73m ² decline to +5ml/min/1.73m ² increase	1.00	
+>5 to <15ml/min/1.73m ² increase	1.05 (0.91, 1.21)	0.523
\geq 15ml/min increase	1.03 (0.89, 1.19)	0.680
12-month eGFR		
<30ml/min/1.73m ²	2.99 (2.62, 3.41)	<0.001
30-<45ml/min/1.73m ²	1.24 (1.11, 1.38)	<0.001
45-<60ml/min/1.73m ²	1.00	
<u>eGFR \geq60ml/min/1.73m² (n=2670)</u>		
Delta eGFR		
\geq -15ml/min/1.73m ² decline	1.49 (1.01, 2.17)	0.043
-5 to <15ml/min/1.73m ² decline	1.51 (1.06, 2.13)	0.022
<-5ml/min/1.73m ² decline to +5ml/min/1.73m ² increase	1.00	
+>5 to <15ml/min increase	1.15 (0.84, 1.56)	0.384
\geq 15ml/min/1.73m ² increase	1.22 (0.91, 1.62)	0.179
12-month eGFR		
60-<75ml/min/1.73m ²	0.90 (0.71, 1.13)	0.362
\geq 75ml/min/1.73m ²	1.00	

Data expressed as adjusted hazard ratios and 95% confidence intervals. Model adjusted for recipient age, gender, body mass index and donor age.
eGFR – estimated glomerular filtration rate by MDRD equation.

Abstract No. 59

OUTCOMES OF LATE ANTIBODY MEDIATED REJECTION: SINGLE CENTRE RETROSPECTIVE STUDY

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Aims: Late antibody mediated rejection (AMR) is recognised as a major contributing cause to late allograft failure, with current therapies having little impact on graft outcomes

Our aim was to identify predictors of outcome in a single centre transplant population with a diagnosis of late AMR and previously normal three-month protocol biopsy.

Methods: We conducted a retrospective review of all renal transplant patients between January 2005 and December 2014. 106 patients were identified with late AMR using our local pathology database. 968 patients transplanted during the same period were used as control group.

Results: Median time to diagnosis of rejection was 58 months (range, 26-97) post-transplant. 33% of the cohort was ABO incompatible (ABOi). Preceding acute cellular rejection was found in 31% of patients. Class 2 de-novo donor specific antibodies (DSA) were present in 32%. When compared with the control group, the late AMR group had higher rates of ABOi and were younger.

Late AMR was associated with a two-fold increased risk of graft loss compared to non-AMR controls. However history of ABOi, C4d positivity or de-novo DSA was not associated with worse graft outcomes. Predictably high chronicity scores on diagnostic biopsy were associated with worse prognosis.

Overall graft survival was poor in late AMR group, 58% of patients losing their graft during the study period with 50% graft survival at 40 months post late AMR diagnosis.

Conclusion: Late AMR continues to be a major post-transplant therapeutic challenge, with high rates of graft loss post diagnosis.

Abstract No. 60

REVERSIBILITY OF FRAILTY IN HEART TRANSPLANT LISTED PATIENTS

JHA Sunita^{1,2}, HANNU Malin¹, NEWTON Phillip³, GORE KAREN¹, WILHELM Kay⁴, HAYWARD Chris¹, JABBOUR Andrew¹, KOTLYAR Eugene¹, KEOGH Anne¹, DHITAL Kumud¹, GRANGER Emily¹, JANSZ Paul¹, SPRATT Phillip¹, MONTGOMERY Elyn¹, HARKESS Michelle¹, TUNNICLIFF Peta¹, MACDONALD Peter¹

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Aim: The aim of this study was to evaluate the reversibility of frailty in AHF patients undergoing bridge-to-transplant ventricular-assist-device (BTT-VAD) implantation and heart transplantation (HTx).

Methods: Since 2013, all AHF patients referred to our center were assessed for physical frailty (Fried phenotype, FP > 3/5 = frail) and a single-item measure of frailty (hand grip strength (HGS)) pre and post BTT-VAD/HTx.

Results: 156 patients (109M:47F; age 53±13 years, range 16-73; LVEF 27±14%) were assessed for frailty. Prevalence was: frail 51 (33%) and not-frail 105 (67%). During listing, 31 patients underwent BTT-VAD implantation and 46 patients were transplanted. 8 frail pre-VAD and 5 frail pre-HTx patients were assessed at follow-up (avg. 17 days post-VAD and avg. 50 post-HTx). Frailty was significantly reversed in all patients who were classified as frail pre-intervention (Figure 1).

Conclusion: While frailty is predictive of increased mortality in patients referred for heart transplantation, surgical interventions have the potential to ameliorate cardiac-induced frailty.

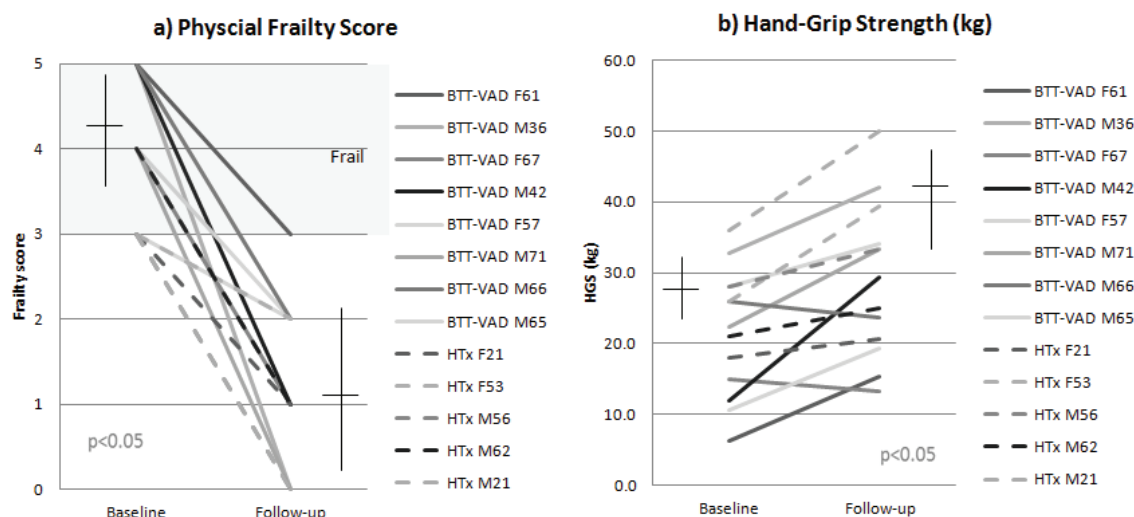


Figure 1: Changes in frailty and hand-grip strength pre-post VAD/HTx

Abstract No. 61**THE VALUE OF SURVEILLANCE BIOPSIES AFTER PAEDIATRIC KIDNEY TRANSPLANTATION****ROSE Edward^{1,2}, MACKIE Fiona^{2,1}, KENNEDY Sean^{1,2}**¹*School of Women's & Children's Health, University of New South Wales, Sydney,* ²*Department of Nephrology, Sydney Children's Hospital***Aims:** To examine the benefit of surveillance biopsies performed 6 months after kidney transplantation in children.**Methods:** A retrospective study of children transplanted at a single centre since 2005. Our protocol was to perform surveillance biopsies at 6 months. Excluded were highly sensitised recipients, ABO incompatible transplants and grafts with less than 12 months of follow-up.**Results:** 35 kidney transplant recipients had a surveillance biopsy at a mean time of 187.9 ± 64.4 days after transplant. Recipients were followed for 1344 person-months (mean 38.4 months) with 97% graft survival. All recipients received induction therapy followed by triple immunosuppression including tacrolimus (n=33) or cyclosporine (n=2) prior to biopsy. Pathology was diagnosed on 24 (69%) of biopsies. Eight (23%) showed subclinical rejection (SCR, n=1) or borderline SCR (n=7); 6 of these were treated with pulse corticosteroids. Calcineurin inhibitor (CNI) toxicity &/or IF/TA was evident on 20 biopsies (57%), including 5 that had borderline SCR. BK nephropathy was diagnosed on 3 biopsies. 9 children had a reduction in CNI dose after biopsy and 7 were switched to sirolimus. The change in eGFR from 1 month after transplantation until the time of surveillance biopsy was not different between patients with biopsy-proven pathology and those with no pathology. Neither was there a difference in change eGFR from time of biopsy through to 3 months afterwards.**Conclusions:** Pathology was evident on more than two thirds of 6 month surveillance biopsies. This information allowed adjustment of immunosuppression in the majority of patients without any evidence of harm.Abstract No. 62**DETERMINANTS OF TUBULAR MICROCALCIFICATION IN PROTOCOL BIOPSIES POST RENAL TRANSPLANTATION****JAW Juli¹, LECAMWASAM Ashani², COCHRANE-DAVIS Alex³, RICHARDS Avisha⁴, SUNDARARAJAN Vijaya⁵, HILL Prue⁶, LANGHAM Robyn^{1,5}**¹*Nephrology and Renal Transplant, St Vincent's Hospital, Melbourne,* ²*Department of Nephrology, Northern Health,* ³*Department of Medicine, Monash University, Melbourne,* ⁴*Department of Medicine, University of Dublin, Ireland,* ⁵*Department of Medicine, University of Melbourne,* ⁶*Department of Anatomical Pathology, St Vincent's Hospital, Melbourne***Introduction:** Tubular microcalcification is a common histopathological feature, often recognised in early post transplant protocol biopsy. The presence of microcalcification is commonly thought to be a result of calcineuric inhibitor toxicity, though hyperparathyroidism and hypercalcaemia may also be predisposing risk factors. The clinical consequence of such finding is unclear, though it is assumed to be associated with inferior allograft function and survival. This study sought to identify clinical features associated with finding of tubular microcalcification in 3 month protocol biopsies.**Methods:** A retrospective study was undertaken of routine three-month protocol biopsies from 2006-2014, where microcalcification was described. Clinical history and serial biochemical data were collected (Table 1). Biochemical parameters were analysed at pre-transplant and at 3, 6 and 12 months post-transplant, along with immunosuppressant use and dose, as well as calcimimetic and calcium/phosphate supplementation**Results:** Tubular microcalcification was identified in 15 patients with mean creatinine of 121.3 ± 25.5 $\mu\text{mol/l}$. CNI toxicity was detected in 1 patient, while metabolic acidosis was evidenced in 2 patients. PTH level was observed to be less than twice upper limit of normal. In 3 patients, tubular microcalcification was associated with allograft rejection. Of note, poorer graft outcome at 6 and 12 months was associated with pre transplant metabolic acidosis and use of calcium based phosphate binders ($p < 0.05$).**Conclusion:** In this cohort, we observed no association between microcalcification and CNI toxicity or hyperparathyroidism. More studies are required to understand the genesis of tubular microcalcification in the acute transplant setting.

Abstract No. 62 (continued)

Table 1. Baseline characteristic of patients (n=15)

	Mean \pm SD (range)/ n (%)
Age (years)	49.5 \pm 12.2 (24-69)
Gender	
Male	13 (87)
Female	2 (13)
Etiology of ESRD	
Glomerulonephritis	6 (40)
Diabetes	3 (20)
Reflux nephropathy	1 (7)
Renal cell sarcoma	1 (7)
Primary oxalosis	1 (7)
Medullary sponge kidney	1 (7)
Unknown	2 (13)
Renal replacement therapy	
Haemodialysis	6 (40)
Peritoneal dialysis	6 (40)
No dialysis	3 (20)
Donor age (years)	41.9 \pm 14.7 (20-71)
Donor gender	
Male	7 (47)
Female	8 (53)
Type of transplantation	
Cadaveric	11 (73)
Living donation	3 (20)
Kidney pancreas	1 (7)
Graft function	
Delayed graft function	1 (7)
Primary graft non-functioning (require dialysis)	3 (20)
Immunosuppression	
Cyclosporine	4 (27)
Tacrolimus	9 (60)
Sirolimus	2 (13)
Rejection	
T-cell mediated	2 (13)
Antibody mediated	1 (7)
Calcineurin toxicity	1 (7)
Graft survival at 12 years	14 (93)

Ischaemia Reperfusion Injury, Metabolism and Islet Transplantation

Abstract No. 63

MATRICELLULAR ACTIVATION OF CD47 LIMITS SELF-RENEWAL TO PROMOTE RENAL ISCHEMIA REPERFUSION INJURY

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Introduction: Ischemia reperfusion injury (IRI) is a consequence of transplantation and initiates kidney repair. The basis for maladaptive repair following IRI remains unclear, although pre-clinical studies have verified a defect in renal tubular epithelial cell (rTEC) proliferation. We reported that the matricellular protein thrombospondin-1 (TSP1), and its receptor CD47 are induced in renal IRI, although their role in recovery is unknown.

Methods: Age-matched wild-type (WT) and CD47^{-/-} mice were challenged with bilateral renal IRI. WT mice underwent syngeneic renal transplant with some recipients treated with CD47 blocking antibody. All animals underwent assessment of renal function and biomolecular analysis. Human and murine rTEC were studied *in vitro*.

Results: Mice lacking CD47 were resistant to renal IRI with decreased urea and creatinine, and demonstrated return of renal function after 7 days, compared to ongoing renal impairment in WT controls. CD47^{-/-} animals displayed constitutive upregulation of self-renewal genes cMyc, Klf4, Oct4, and Sox2. WT animals demonstrated negligible self-renewal gene expression at all time points. Following kidney transplantation, administration of a CD47 blocking antibody to the recipient improved serum creatinine and upregulated self-renewal targets. Indicative of a cell-dependent process, CD47^{-/-} rTEC displayed basal upregulation of self-renewal genes that correlated with enhanced proliferative capacity. Addition of TSP1 to WT rTEC downregulated self-renewal gene expression, which was not seen in CD47^{-/-} cells. Conversely, treatment with a CD47 antagonist antibody increased self-renewal and promoted proliferation.

Conclusions: CD47 impairs rTEC recovery through inhibition of self-renewal, and thus may be a possible clinical target to facilitate organ repair following transplantation.

Abstract No. 64

SIGNAL INHIBITORY REGULATORY PROTEIN- α ; REGULATES GENERATION OF PATHOLOGIC REACTIVE OXYGEN SPECIES IN ACUTE KIDNEY INJURY

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Background: Ischemia reperfusion injury (IRI) is mediated by reactive oxygen species (ROS). We have recently reported that signal regulatory inhibitory protein (SIRP)- α is expressed by renal tubular epithelial cells (rTEC). We have also shown that the protein thrombospondin-1 (TSP1) is increased following renal IRI and binds to SIRP α . However, it is unclear how the TSP1-SIRP α signaling contributes to the pathophysiology of IRI.

Methods: Age-matched male wild-type (WT) mice, and SIRP α mutant (SIRP α^{mut}) mice, which lack the cytoplasmic recruitment domains, underwent bilateral renal IRI. Animals underwent biomolecular analysis at 24h reperfusion. WT and SIRP α^{mut} rTEC were studied *in vitro*. Mice were also lethally irradiated and rescued with WT or SIRP α^{mut} bone marrow, to interrogate the contribution of the parenchymal cell compartment to IRI.

Results: IRI significantly elevated serum creatinine in WT mice, which was mitigated in SIRP α^{mut} animals (2.3 ± 0.46 versus 0.98 ± 0.41 mg/dL, $p < 0.01$). TSP1 was expressed to a similar degree post-IRI. Measurement of ROS in whole kidney demonstrated a 2-fold increase in WT mice post-IRI, but no increase in SIRP α^{mut} or sham-operated animals. Expression of oxidative protein modification was reduced in SIRP α^{mut} kidneys compared to WT, although expression of NADPH oxidases was unchanged. WT rTEC displayed upregulation of ROS in response to TSP1, which was not demonstrated in SIRP α^{mut} cells. In chimeric animals, SIRP α^{mut} mice, regardless of hematopoietic reconstitution, were protected against renal dysfunction and ROS generation following IRI.

Conclusion: These data provide genetic evidence for a role for SIRP α promoting renal IRI through generation of pathologic ROS. Blockade of SIRP α may provide a novel therapeutic target to modify IR-mediated damage.

Abstract No. 65

ASSESSMENT OF THE EFFECT OF FOLLISTATIN TREATMENT AND KINETICS OF ACTIVIN A AND B IN RENAL ISCHEMIA-REPERFUSION INJURY IN MICE**FANG Doreen^{1,2}, LU Bo¹, HAYWARD Susan³, DE KRETZER David^{3,4}, COWAN Peter^{1,2}, DWYER Karen^{2,5}**¹*Immunology Research Centre, St Vincent's Hospital, Melbourne,* ²*Department of Medicine, University of Melbourne,* ³*Centre for Reproductive Health, Hudson Institute of Medical Research,* ⁴*Department of Anatomy & Developmental Biology, Monash University, Melbourne,* ⁵*School of Medicine, Deakin University*

Aims: Ischemia-reperfusion injury (IRI) is an inherent process of renal transplantation and has a profound influence on early and late allograft function. Activins, members of the TGF- β superfamily, are key drivers of inflammation and their action is blocked by follistatin (FS). We previously presented data showing protective effect of FS treatment against renal IRI in mice. This study aimed to delineate the kinetics of activins and FS post-renal IRI.

Methods: We measured levels of activin A, activin B and FS in the serum and kidney at 3, 6 and 24h post-renal IRI in mice, and tested the effect of FS treatment.

Results: (Table 1) Serum and kidney activin A and B were elevated post-IRI albeit with different kinetics. Early renal injury, characterized by elevated serum creatinine, was evident at 3h post-IRI and may be activin-independent: reduction in activin B with FS treatment had no apparent impact on creatinine. Further renal injury was potentiated mainly by activin A as mitigation of the elevated serum activin A 24h post-IRI reduced the extent of renal injury, defined by a reduction in serum creatinine, renal tubular injury score, kidney injury molecule-1 (KIM-1) expression, and renal cell apoptosis. Serum levels of the pro-inflammatory molecules IL-6 and MCP-1 were also reduced by FS treatment.

Conclusions: Activin A rather than activin B is the principal mediator in renal IRI. FS treatment attenuates renal IRI through binding of activin A, suggesting a potential clinical application of FS treatment in renal transplantation to improve graft function and survival.

Table 1: Summary of serum activins, FS and renal injury parameters at 3, 6 and 24h post-renal IRI

Reperfusion	Groups	Activin A pg/ml	Activin B pg/ml	FS ng/ml	Cr μ mol/L	Renal tubular injury	KIM-1	Renal cell apoptosis
3h	Sham n=6	20.60 \pm 3.09	764.30 \pm 152.80	7.58 \pm 0.55	21.17 \pm 0.70	-	-	-
	Vehicle n=7	18.34 \pm 1.64	746.50 \pm 55.12	7.41 \pm 0.54	63.14 \pm 3.58	-	-	-
	FS n=7	17.48 \pm 2.45	72.33 \pm 0.0	16.83 \pm 0.62	64.29 \pm 3.38	-	-	-
p-value	Vehicle vs FS	ns	$p < 0.0001$	$p < 0.001$	ns	-	-	-
6h	Sham n=8	17.30 \pm 1.67	1361.0 \pm 258.40	5.98 \pm 0.17	22.50 \pm 1.48	-	-	-
	Vehicle n=8	19.0 \pm 2.49	2481.0 \pm 308.40	7.31 \pm 0.85	70.25 \pm 5.32	-	-	-
	FS n=7	15.86 \pm 0.79	134.50 \pm 42.75	11.27 \pm 0.58	74.0 \pm 9.30	-	-	-
p-value	Vehicle vs FS	ns	$p < 0.0001$	$p < 0.05$	ns	-	-	-
24h	Sham n=7	54.01 \pm 11.74	1080.0 \pm 85.83	4.55 \pm 0.34	22.71 \pm 1.44	0.14 \pm 0.14	3.80 \pm 0.56	8.0 \pm 1.4
	Vehicle n=9	205.50 \pm 23.94	3003.0 \pm 335.60	5.69 \pm 0.68	126.1 \pm 13.93	3.56 \pm 0.17	4693.0 \pm 879.3	64.20 \pm 2.78
	FS n=6	112.0 \pm 18.49	2342.0 \pm 369.30	3.96 \pm 0.14	51.33 \pm 10.53	2.83 \pm 0.17	2537.0 \pm 410.6	44.67 \pm 3.42
p-value	Vehicle vs FS	$p < 0.001$	ns	ns	$p < 0.01$	$p < 0.05$	$p < 0.05$	$p < 0.001$

Abstract No. 66

WITHDRAWN

Abstract No. 67**FACTORS EFFECTING ISLET ISOLATION OUTCOMES OVER THE PAST 15 YEARS FOR THE WESTMEAD ISLET TRANSPLANT PROGRAM****CHEW Yi Vee¹, WILLIAMS Lindy¹, DAVIES Sussan¹, LIUWANTARA David², BURNS Heather², HAWKES Joanne¹, O'CONNELL Philip¹, HAWTHORNE Wayne¹****¹Centre for Transplant and Renal Research, Westmead Hospital, Sydney, ²Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney**

Aims: Islet cell transplantation for type 1 diabetes relies heavily on successful isolation outcomes to ensure successful transplantation. As such we aimed to evaluate our islet isolation outcomes and identify factors during donor selection, organ procurement and islet isolation influencing the preparation leading to a transplant.

Methods: Islets were isolated from pancreata of heart beating deceased donors using collagenase and NP (SERVA). Donor characteristics, pancreas procurement data, isolation yield and outcomes were collected and compared to determine correlation between each variable. Isolations were also divided into Transplanted (Tx) VS Non-transplanted preparations (Non-Tx) to identify variables significantly influencing isolation outcomes.

Results: Data from 207 islet isolations collected between July 2000 and December 2015 were evaluated. On average, 24.9% of islet preparations were transplanted, with 45.8% of isolations in 2014-2015 reaching release criteria. Transplantable yields (defined as 300,000 IEQ; 4,000 IEQ/kg for a 75kg recipient) were obtained from donors aged between 20-60 years, with BMI >20kg/m², and weight >55kg. Digestion times >25 mins were found to negatively affect cell viability and yield.

Compared to non-tx (n=158), Tx (n=49) had significantly higher total IEQ (611,212±329,905 VS 329,905±202,249 IEQ) and IEQ/g pancreas (8,389±6,525 VS 5,229±3,792 IEQ/g). Higher donor BMI, donor weight, pancreas weight, and lower CIT, were significantly correlated (p<0.05) to Tx. Tx also exhibited significantly higher viability, purity and beta cell viability indices compared to Non-tx.

Conclusions: We found that increased donor BMI/weight and lower CIT all have significant effects on outcomes. In particular these influence which islet isolations resulted in transplantable yields/outcomes.

Abstract No. 68

INVESTIGATING THE POTENTIAL OF DANTROLENE SODIUM SALT AS A CARDIOPROTECTIVE AGENT DURING ISCHAEMIA-REPERFUSION INJURY.

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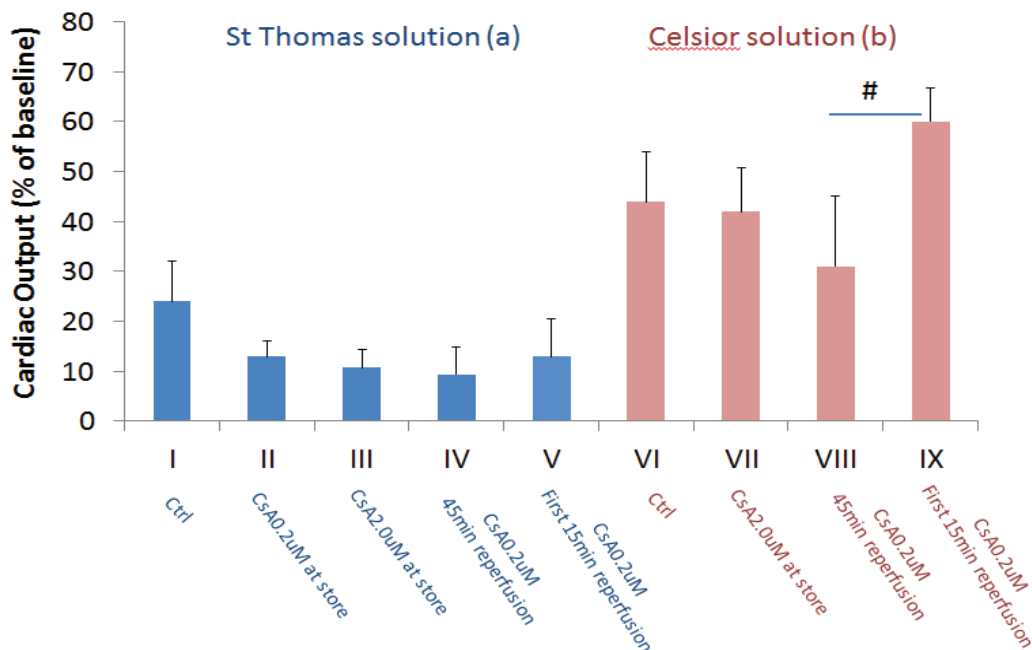
Aim: To determine whether the ryanodine receptor antagonist Dantrolene, traditionally used to treat malignant hyperthermia, can be repurposed as a cardioprotective agent for minimizing ischaemia-reperfusion injury in the donor heart.

Methods: Rat hearts (Wistar; 320-410g) were placed on a Langendorff ex-vivo perfusion circuit for baseline coronary flow (CF), aortic flow (AF), cardiac output (CO) and heart rate (HR) measurements. Hearts were stored for 6 hours at 4°C. Dantrolene (4uM) was added to either pre-storage perfusate, cardioplegia (Celsior), or post-storage reperfusate. Dantrolene (0.2 – 40uM) during storage or Dantrolene (4uM) in entire protocol (pre-storage, cardioplegia and reperfusion) was tested. CF, AF, CO, and HR recovery following reperfusion was calculated as a percentage of pre-storage baseline. Coronary effluent was collected during baseline and reperfusion for lactate dehydrogenase (LDH) release assessment.

Results: Hearts treated with 4uM Dantrolene during either pre-storage, cardioplegia or reperfusion recovered similar to Celsior controls. Hearts stored in 0.4uM Dantrolene exhibited a trend towards increased AF recovery compared to all other storage groups whereas cardiac recovery was abolished following 40uM Dantrolene storage. Further, combined Dantrolene treatment in pre-storage, cardioplegia and reperfusion was detrimental to cardiac recovery and showed no AF recovery– correlating with a significant increase in LDH release compared to hearts with a single Dantrolene exposure ($p < 0.0001$ after 45 min reperfusion).

Conclusions: The presence of 4uM Dantrolene either before, during, or after cold storage of donor hearts does not improve cardiac recovery. Excessive Dantrolene exposure reduces AF, CF and CO recovery and significantly increases cellular injury indicative of dose-dependent cardiomyocyte toxicity.

Abstract No. 69

POST-CONDITIONING WITH CYCLOSPORINE: IMPACT ON ISCHEMIA REPERFUSION INJURY IN A RODENT MODEL OF DONOR HEART PRESERVATION**GAO Ling¹, VILLANUEVA Jeanette¹, CHEW Hong¹, HICKS Mark^{2,3}, JABBOUR Andrew^{4,1,5}, CAO Jacob^{1,6}, MACDONALD Peter^{4,1,5}**¹*Transplantation Laboratory, Victor Chang Cardiac Research Institute, Sydney,* ²*Department of Clinical Pharmacology, St Vincent's Hospital, Sydney,* ³*School of Medicine & Pharmacology, University of New South Wales, Sydney,* ⁴*Department of Cardiology, St Vincent's Hospital, Sydney,* ⁵*School of Medicine, University of New South Wales, Sydney,* ⁶*School of Medicine, University of Sydney***Aims:** To investigate the potential benefits of the mitochondrial permeability transition pore inhibitor cyclosporine (CsA) in donor heart preservation after 6 hours of hypothermic storage.**Methods:** Isolated working rat hearts were stored for 6 hours in Celsior or St Thomas solution (STH). CsA was added to the preservation solution (0.2 or 2.0 μ M) during cardioplegia and storage (2-3°C), or to KH buffer (0.2 μ M) during the first 15 min or the whole 45 min of normothermic reperfusion after storage. Preservation of cardiac function was calculated by recovery of cardiac output (CO) as percentage of pre-storage baseline. Lactate dehydrogenase (LDH) release during perfusion of the heart was assessed prior to and after storage.**Results:** Addition of CsA to STH or Celsior during storage did not improve post-storage functional recovery. After storage in Celsior, hearts exposed to 0.2 μ M CsA only for the first 15 min of reperfusion displayed significantly better CO recovery compared to those exposed to CsA for 45 min of reperfusion ($60 \pm 7\%$ vs $31 \pm 14\%$ $p = 0.02$). Also the most significant increase in LDH release occurred in hearts perfused with 0.2 μ M CsA for 45 min of reperfusion after storage ($p < 0.05$ compared to all other groups).**Conclusions:** The addition of CsA to preservation solutions during cold storage provided no additive protection. CsA improved post-storage cardiac functional recovery only when delivered for the first 15 minutes of reperfusion. Prolonged exposure to CsA during reperfusion resulted in significantly increased LDH release, suggesting direct toxicity to cardiomyocytes.

Abstract No. 70

METABOLIC PROFILE OF DCD HEARTS DURING RECONDITIONING**CHEW Hong Chee^{1,2}, CAO Jacob¹, FERNANDEZ Karen¹, VILLANEUVA Jeanette¹, GAO Ling¹, HICKS Mark¹, JABBOUR Andrew^{1,2}, DHITAL Kumud^{1,3}, MACDONALD Peter^{1,2}**¹*Transplantation Laboratory, Victor Chang Cardiac Research Institute, Sydney*, ²*Department of Cardiology, St Vincent's Hospital, Sydney*, ³*Department of Cardiothoracic Surgery, St Vincent's Hospital, Sydney***Aim:** To understand cardiac metabolic demands during early ex-vivo perfusion in order to limit ischaemic reperfusion injury (IRI) and improve functional recovery in donation after circulatory death (DCD) hearts.**Method:** Landrace pigs (n=12) were anaesthetised and asphyxiated until circulatory arrest, followed by a stand-off period of 5mins. Blood collection and preservation flush occurred prior to explantation and installation on the ex-vivo device. Blood samples at baseline, following circuit priming, and every hour thereafter were taken. Arterial pH, haematocrit (Hct) and electrolyte profile (K^+ , Ca^{2+} , and HCO_3^-) were measured using an I-Stat analyser.**Results:** All results are presented in table below. There is considerable alkalosis when introducing blood to the priming solution (500mls Krebs solution + Gelofusine) but this is overcompensated with acidosis when the heart is initially installed on the ex-vivo circuit. However, this acidosis is corrected overtime. A significant drop to the Hct, [Ca] and [HCO] is seen when priming solution is added to blood volume. This worsens overtime due to consumption, unless externally replaced. In contrast, a rise in [K] with circuit priming is partially corrected following heart installation.**Conclusion:** In porcine model, significant biochemical derangement occurs from the time of withdrawal. In addition to the dilutional effect of the priming solution, there is consumptive trend overtime unless externally replaced. Correction by the organ is limited and requires stringent replacement to ensure adequate recovery.

	<i>Baseline</i>	<i>Blood + Priming Solution</i>	<i>Installation of Heart</i>	<i>180 mins post reconditioning</i>
<i>pH</i>	7.36 ± 0.08	7.53 ± 0.06	7.27 ± 0.05	7.31 ± 0.06
<i>Hct</i>	23 ± 5.5%	15 ± 6.0%	13 ± 3.1%	
<i>Ca²⁺ (mM)</i>	1.3 ± 0.15	0.64 ± 0.32	0.53 ± 0.36	
<i>K⁺ (mM)</i>	3.89 ± 0.19	5.59 ± 1.78	4.66 ± 1.98	
<i>HCO₃⁻ (mM)</i>	33.78 ± 2.93	23.92 ± 3.15	20.42 ± 2.67	
<i>Lactate</i>	2.14 ± 2.33		2.64 ± 0.56	1.75 ± 0.63

President's Prize Symposium

Abstract No. 71

DELETION OF RECIPIENT CD8⁺ DENDRITIC CELLS FACILITATES THE PERSISTENCE OF HIGHLY CYTOLYTIC DONOR CTL THAT ELIMINATE LEUKAEMIA AFTER BMT**MARKEY Kate^{1,2,3}, KUNS Rachel¹, ROBB Renee¹, KOYAMA Motoko¹, GARTLAN Kate^{1,3}, HENDEN Andrea^{1,2}, MACDONALD Kelli⁴, BROCKER Thomas⁵, BELZ Gabrielle⁶, LANE Steven⁷, HILL Geoff^{1,2}****¹BMT Laboratory, Queensland Institute of Medical Research, Brisbane, ²Department of Haematology, Royal Brisbane and Women's Hospital, ³School of Medicine, University of Queensland, Brisbane, ⁴Antigen Presentation and Immunology Laboratory, Queensland Institute of Medical Research, Brisbane, ⁵Institute for Immunology, Ludwig Maximilians University Munich, Germany, ⁶Walter and Eliza Hall Institute of Medical Research, Melbourne, ⁷Translational Leukaemia Laboratory, Queensland Institute of Medical Research, Brisbane**

Introduction: Allogeneic bone marrow transplantation remains the therapy of choice for many haematological malignancies, but despite the immunologically mediated graft-versus-leukaemia (GVL) effect, relapse remains a key cause of death. Here we investigated the role of dendritic cell (DC) subsets in the presentation of antigen to donor CD8 cytotoxic T cells (CTL) in pre-clinical models of BMT.

Methods and Results: Donor C3H.Sw bone marrow and purified CD8 T cell grafts were transplanted with recipient-derived MLL-AF9 induced primary acute myeloid leukaemia into lethally irradiated recipient B6.CD11c.DOG recipients (diphtheria toxin receptor (DTR), ovalbumin and GFP expression driven off the CD11c promoter) such that recipient DC can be deleted by DT administration. Surprisingly, depletion of recipient DC resulted in improved leukaemic control (median survival 43 vs 31 days, $P < 0.001$). The use of IRF8^{-/-} BMT recipients (in which the CD8⁺ DC subset is absent) confirmed that recipient CD8⁺ DC were critical for this regulation of GVL (median survival 43 vs 34 days, $P = 0.0005$). Conversely, when recipient CD8⁺ DC were expanded using Flt3L treatment for 10 days prior to BMT, GVL effects were completely eliminated, rendering relapse rates equivalent to that seen in the recipients of T cell depleted (TCD) grafts (median survival 11 days in Flt3L treated BM+T and TCD grafts). Mechanistically, recipient CD8⁺ DC induced high levels of activation induced apoptotic cell death (AICD) in both polyclonal and antigen-specific donor T cells, resulting in a marked CTL contraction, permissive of leukemic relapse. Consistent with this, recipient CD8 DC-mediated AICD in donor T cells also mediated protection from acute GVHD. In contrast, donor DC exacerbated acute GVHD but were not required for GVL effects.

Conclusion: DC-targeted therapeutics should only be applied late after BMT when donor DC, which can drive acute GVHD, and recipient DC that conversely protect from GVHD and leukaemia relapse have been eliminated.

Abstract No. 72

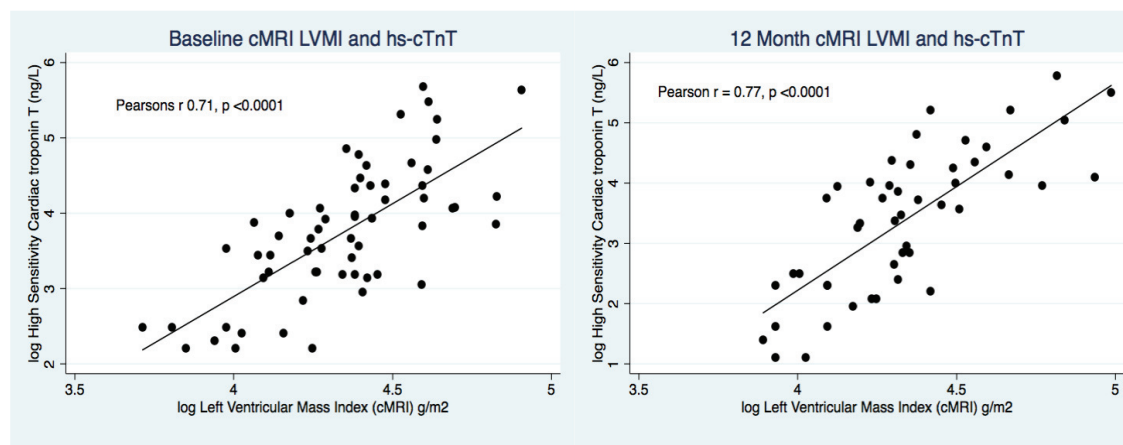
TIME-DEPENDENT CHANGES IN CARDIAC BIOMARKERS AND CARDIAC MAGNETIC RESONANCE IMAGING (CMRI) DETERMINED CARDIAC STRUCTURE AND FUNCTION IN END-STAGE KIDNEY DISEASE (ESKD) AND FOLLOWING RENAL TRANSPLANTATION**CROSTHWAITE AA^{1,2}, LIM R³, MASTERSON R⁴, HEDLEY A¹, ROBERTS M^{5,6}, FAROUQUE O^{7,2}, IERINO F^{1,2}**¹Department of Nephrology, Austin Hospital, Melbourne, ²Department of Medicine, University of Melbourne,³Diagnostic Imaging, Austin Hospital, Melbourne, ⁴Department of Nephrology, Royal Melbourne Hospital,⁵Department of Nephrology, Eastern Health, Victoria, ⁶Eastern Health Clinical School, Monash University,⁷Department of Cardiology, Austin Hospital, Melbourne**Background:** Defining reversible and irreversible cardiac pathophysiology using cMRI and cardiac biomarkers may provide mechanistic and therapeutic insights for CKD associated cardiovascular disease (CVD).**Aim:** Characterise changes in cardiac pathology by cMRI/TTE, high-sensitivity cardiac troponin T (hs-cTnT) and NT-proBNP in ESKD and following renal transplantation**Methods:** Ongoing prospective, study in incident renal transplant recipients (RTR) (n=25, 44% pre-emptive, follow-up complete n=19), prevalent haemodialysis (HD) patients (Satellite-HD, n=26; Home-HD, n=14). cMRI/TTE were performed at enrolment and after 12 months follow up. Serial cardiac biomarkers and routine serum biochemistry (18 timepoints) were analysed.**Results:** At baseline (n=65) no difference in any cardiac imaging parameter was observed between subgroups of ESKD. After 12 months, a significant reduction in LVMI (cMRI but not TTE) was measured for RTR (n=19, mean (95%CI) 69g/m² (60-77 g/m²) vs 62 g/m² (55-69 g/m²) p=0.03) but not for HD patients (n=22 Satellite-HD; 13 Home-HD mean (95%CI) 82g/m² (74-90g/m²) vs 83g/m² (75-91g/m²) p=0.63). Following GFR stabilization, RTR demonstrated continued significant decline in hs-cTnT (p<0.0013) and NT-proBNP (p<0.0003). Furthermore, hs-cTnT but not NTproBNP correlated strongly with LVMI at baseline (r=0.71, p<0.0001) and 12 months (r=0.77, p<0.0001) Fig 1. Significant associations with LVMI were: mean-arterial-pressure (p=0.025), any CVD (p=0.011) and loghs-cTnT (p<0.0001) at baseline, and any CVD (p=0.046), loghsc-TnT(p=0.001) and logNTproBNP (p=0.01).**Conclusions:** This is one of the largest cohort studies utilising cMRI demonstrating that LVMI reduces following kidney transplantation and hs-cTnT levels reflect this change. Ongoing analysis of cardiac fibrosis, trophic changes and global-longitudinal-strain should provide further mechanistic insights into CVD in CKD.

Fig. 1. Correlation between high-sensitivity cardiac troponin T and LVMI measured by cMRI at baseline and 12 months.

Abstract No. 73

INNATE ALLO-RECOGNITION RESULTS IN RAPID ACCUMULATION OF MONOCYTE DERIVED DENDRITIC CELLS

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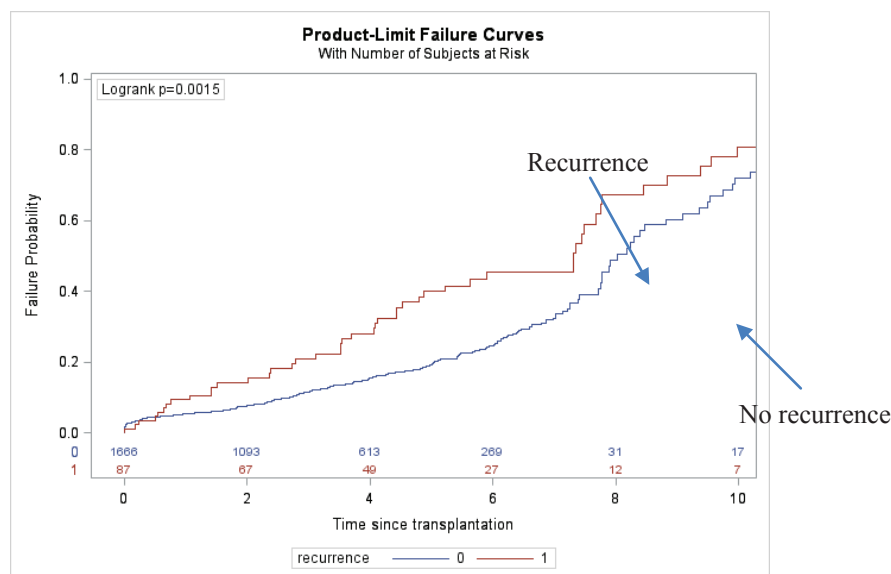
Aims: Monocyte derived dendritic cells (moDCs) are rare in the steady state but accumulate during infection and inflammation. They are involved in innate and adaptive immune responses in various conditions; however how they are evoked in response to allogeneic stimuli is poorly understood.

Methods: In mice on the C57BL/6, BALB/c, DBA/2 and NOD genetic backgrounds, we enumerated splenic moDCs and conventional dendritic cells (cDCs), following adoptive transfer of syngeneic or allogeneic splenocytes, and compared them to untreated mice.

Results: moDCs became abundant 1 day after IV injection of allogeneic splenocytes but not syngeneic splenocytes, while cDC numbers were unaffected by either allogeneic or syngeneic stimulation. This occurred in various donor-host strain combinations. Using cells from MHC-matched DBA/2 and BALB/c mice, we found that allogeneic moDC induction did not require MHC mismatch. The potency of allogeneic moDC induction was dependent on the number of donor cells transferred and could be induced by various donor cell types including B cells, T cells or natural killer (NK) cells. Using cells from lymphoid cell-deficient RAG2^{-/-}γ^{-/-} and NOD-scid-IL2Rγ^{-/-}; and selectively NK-deficient Mcl1^{fl/fl}Ncr1-Cre mice, we found that allogeneic moDC induction only occurred in the presence of either host or donor lymphoid cells, particularly NK cells.

Conclusion: moDCs accumulate rapidly following exposure to allogeneic antigen. This process requires the presence of either host or donor lymphoid cells, and occurs independently of MHC mismatch. This innate allo-recognition raises potential new insights into how the immune system responds to allogeneic encounters such as that which occurs during organ transplantation.

Abstract No. 74

RECURRENT GLOMERULONEPHRITIS AND LONG-TERM GRAFT OUTCOMES AFTER KIDNEY TRANSPLANTATION**ALLEN Penelope¹, CRAIG Jonathan^{1,2}, LIM Wai^{3,4}, CHADBAN Steve^{2,5}, ALLEN Richard^{2,5}, WONG Germaine^{1,6}**¹*Centre for Kidney Research, The Children's Hospital at Westmead, Sydney,* ²*School of Medicine, University of Sydney,* ³*School of Medicine & Pharmacology, University of Western Australia, Perth,* ⁴*Renal Unit, Sir Charles Gairdner Hospital, Perth,* ⁵*Royal Prince Alfred Hospital, Sydney,* ⁶*Renal Unit, Westmead Hospital, Sydney***Aims:** To determine the prevalence of recurrent glomerulonephritis and the risk of death-censored and overall graft loss in recipients with recurrent glomerulonephritis after kidney transplantation.**Methods:** Primary live and deceased-donor kidney transplant recipients between 1997 and 2013 whose end-stage kidney disease (ESKD) was attributed to glomerulonephritis were included. We determined the association between the risk of overall and death-censored graft loss and recurrent disease using adjusted Cox proportional regression models.**Results:** A total of 4,053 recipients were followed for a median follow-up time of 3.34 (IQR: 4.03) years. Glomerulonephritis was the commonest cause of ESKD (n=1753, 43.3%), followed by congenital/genetic disease (n=769, 19.0%) and diabetes (n=431, 10.6%). The most common forms of glomerulonephritis were IgA disease (n=650, 16.0%), focal segmental glomerulonephritis (n=91, 2.25%) and mesangiocapillary glomerulonephritis (n=60, 1.48%). A total of 87 recipients (out of 1753, 4.90%) experienced disease recurrence, with the median time to recurrence of 1.63 (IQR: 2.75) years. There were no significant differences in recipient characteristics with and without recurrence. Recipients with recurrent glomerulonephritis were more likely to lose their allograft than those without. The median time to graft loss for those with and without disease recurrence were 5.9 (IQR: 4.3) and 8.4 (IQR: 4.6) years, respectively. The adjusted HR for overall graft loss and death-censored graft loss were 1.41 [(95%CI: 1.12 – 1.75)] and 1.42 [(95%CI: 1.13 – 1.79)] (Figure 1).**Conclusions:** Whilst there was a small risk of recurrent glomerulonephritis in the transplanted kidney, disease recurrence appeared to have significant impact on long-term graft survival even in the current era.**Figure 1. Cumulative incidence of overall graft loss in recipients with and without recurrence**

Abstract No. 75

THE IMPACT OF DONOR-RECIPIENT AGE MISMATCH ON GRAFT AND PATIENT OUTCOMES AFTER DECEASED DONOR KIDNEY TRANSPLANTATION**CALISA Vaishnavi, CRAIG Jonathan, CHADBAN Steve, LIM Wai, HOWARD Kirsten, CHAPMAN****Jeremy, MCDONALD Stephen, WONG Germaine****Centre for Kidney Research, The Children's Hospital at Westmead, Sydney**

Aim: To determine the distribution of donor-recipient age differences and its influence on patient and graft outcomes after deceased donor kidney transplantation.

Methods: Using data from the ANZDATA registry (1997-2013), we examined the distribution of donor-recipient age differences and their association with overall graft loss and mortality, using Cox proportional hazards modelling.

Results: A total of 3,025 recipients were followed for a median follow-up time of 2.9 years. The mean (SD: 13.7 years) age of the cohort was 49.5 years and 1,161 (38.4%) were female. The donor-recipient age differences were normally distributed [mean (SD) 3.8 +/- 20.0 years], confirming that it is not presently a criterion in assigning donor kidneys to recipients. A total of 212 (7.0%) recipients experienced allograft loss and 172 (5.7%) died with functioning grafts. The excess risk of mortality increased exponentially to 1.7 times (95%CI: 1.18, 2.35) when the donor was 40 years older than the recipient, but decreased asymptotically as the donor-recipient age differences fell below 0 (Figure 1). For every year increase in the donor-recipient age mismatch, the adjusted hazard ratios for overall graft loss and mortality were 1.016 (95%CI: 1.016, 1.023) and 1.013 (95%CI: 1.004, 1.022), respectively. Compared to those without donor-recipient age mismatches, older recipients who received kidneys from younger donors incur a significant survival advantage, with mortality risk decreased by up to 40% (95%CI: 15.2%, 57.5%), corresponding to a donor-recipient age difference of -40 years (Figure 1).

Conclusions: Reducing donor-recipient age mismatches may yield improved equity and utility in organ allocation.

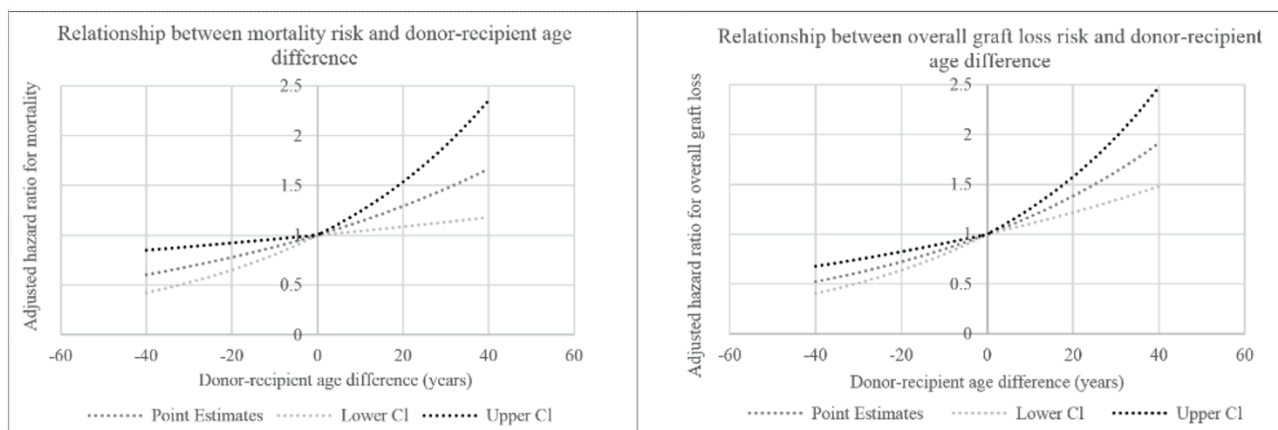


Figure 1: (on left) Relationship between adjusted hazard ratio for overall graft loss and donor-recipient age difference (on right) Relationship between adjusted hazard ratio for mortality and donor-recipient age difference

Abstract No. 76

CARDIOVASCULAR MAGNETIC RESONANCE NON-INVASIVELY DETECTS CARDIAC TRANSPLANT REJECTION: A PROSPECTIVE, HISTOLOGICALLY-VALIDATED STUDY

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Aims: Endomyocardial biopsies (EMBx) are required for transplant rejection surveillance but are associated with potentially serious complications. Cardiovascular Magnetic Resonance (CMR)-based tissue characterisation using T1 and T2 mapping sequences detects interstitial oedema in myocarditis. This study aimed to determine the role of CMR in the detection of cardiac transplant rejection.

Methods: Patients underwent CMR within 24 hours of their EMBx which was also stained with Masson's trichrome to assess interstitial fibrosis. Serum troponin T and pro-BNP levels were also measured.

Results: Of 84 scans, 42 were in group 0 (ISHLT grade 0), 27 in group 1 (ISHLT grade 1R), and 15 in group 2 (ISHLT grades 2R, 3R, antibody mediated or clinically-diagnosed rejections). T1 values were significantly higher in group 2 (1033 (10.42); mean (SEM)) vs. group 0 (983 (7.18); $p=0.0001$) and group 1 (995.2 (17.06); $p=0.01$). T2 values were also significantly higher (in group 2 (67.59 (1.95)) vs. group 1 (56.07 (1.266)) and group 0 (53.60 (0.687); all $p=0.0001$). Left ventricular ejection fraction, troponin T and pro-BNP did not correlate significantly with rejection. The combination of T2- and T1-mapping data further improved transplant rejection detection (100% sensitivity, 85% specificity, and a 100% negative predictive value). Patients with more histologically-determined interstitial fibrosis also had higher T1 values ($p<0.05$).

Conclusion: Novel CMR-based tissue characterisation displays excellent negative predictive capacity and holds promise to improve the non-invasive detection of cardiac allograft rejection.

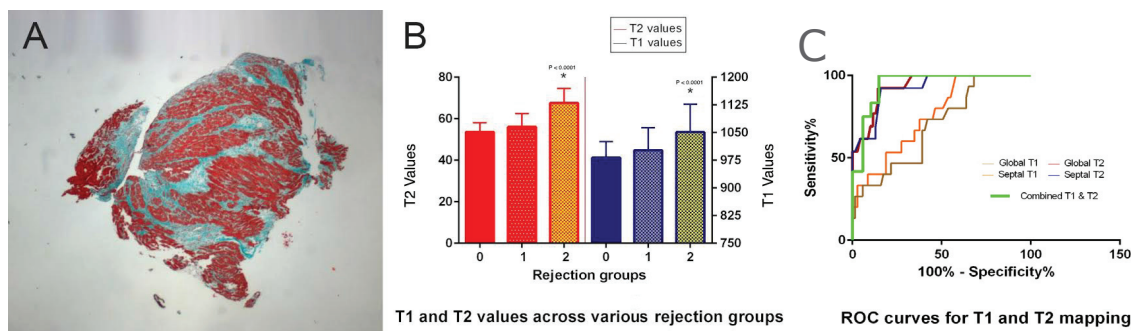


Figure 1.A. EMBx tissue with Masson's Trichrome stain demonstrating interstitial myocardial fibrosis. 1. B. Comparison of T1 and T2 values between various rejection groups. Group 0: ISHLT grade 0, Group 1: ISHLT grade 1R, Group 2: ISHLT grade 2R, 3R, antibody mediated rejection and clinically-diagnosed rejection. 1. C. ROC curves for T1 and T2 values measured along the interventricular septum (septal T1 & T2) as well as circumferentially at mid-ventricular short axis slice (Global T1 and T2) to diagnose rejection in heart transplant recipients. The area under the curve for combined T1 and T2 values was 0.946 with sensitivity of 100% and specificity of 85%.

Abstract No. 77**COMBINED HEART AND LIVER RETRIEVAL AFTER CIRCULATORY DEATH WITH NORMOTHERMIC MACHINE REPERFUSION IN A PORCINE MODEL****CAO Yiming, CHEW HC, FERNANDEZ Karen, VILLANUEVA Jeanette, GAO Ling, HICKS Mark, JABBOUR Andrew, PLEASS Henry, DHITAL Kumud, MACDONALD Peter*****Transplantation Laboratory, Victor Chang Cardiac Research Institute, Sydney***

Aims: When donation after circulatory death(DCD) heart and liver retrieval are combined, there are concerns regarding delays to institution of liver cold preservation. We aim to develop a combined DCD heart and liver retrieval protocol in a pig model with subsequent normothermic machine perfusion(NMP).

Methods: Pigs(n=12; 60-70kg) were anaesthetised. Baseline observations recorded. After sternotomy, and laparotomy, ventilatory support

was withdrawn to mimic DCD conditions. Death was defined as equalisation of central venous and mean arterial pressures. After a 5-minute stand-off period, blood was collected from a right atrial cannula then cardiac preservation flush(4°C) commenced via the proximal ascending aorta. The inferior vena cava was vented. The thoracic cavity was kept cold with saline ice slush. The heart was explanted and prepared on back-table for NMP. Liver preservation begun immediately after blood collection by cold preservation solution flush via the infra-renal aorta. The hepatic artery, portal vein and common bile duct were transected, and the liver explanted for back-table NMP preparation.

Results: Warm ischaemic time(WIT) and back-table time(BTT) are presented in table below. The average blood volume collected was 1.6L; 1:1 dilution with Krebs resulted in significant hemodilution and hypocalcemia. This resulted in sub-optimal cardiac contractile recovery despite a favourable lactate profile. Liver enzyme release, bile production, and lactate and pH profiles were favourable during 4-6 hours of NMP.

Conclusions: Heart and liver retrieval under standard DCD protocol is possible without excessively extending the WIT for either organ. However, there is insufficient donor blood to support NMP of both organs.

	Heart	Liver
WIT (Warm Ischaemic Time)	21 ± 5min	20 ± 6min (commencement of flush)
BTT (Back Table Time)	29 ± 6min	33 ± 19 min
Blood Volume Collected	1621 ± 279ml	
	Baseline (Heart)	On Rig (Heart)
Hct	23 ± 5%	15 ± 6%
Ca ²⁺	1.30 ± 0.15mM	0.64 ± 0.32mM

Abstract No. 78**THE ADDITION OF COGNITIVE IMPAIRMENT TO PHYSICAL FRAILTY IMPROVES SURVIVAL PREDICTION IN HEART-TRANSPLANT REFERRED PATIENTS**

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Aim: The aim of this study was to identify whether the addition of cognitive impairment (CI) to the assessment of physical frailty (PF) better enhanced mortality prediction in heart-transplant referred patients.

Methods: Since 2013, all patients referred to our transplant center were consecutively assessed for physical frailty using an adapted version of Fried's Phenotype. A patient was classified as frail if $\geq 3/5$ domains were present. Assessment of cognitive impairment (Montreal Cognitive Assessment - MoCA) was also conducted at the time of frailty assessment. A patient was classified as 'cognitively frail' if $\geq 3/6$ domains were present.

Results: 156 patients (109M:47F; age 53 ± 13 years, range 16-73; LVEF $27 \pm 14\%$) underwent frailty assessment. Prevalence by physical frailty was: Frail 51 (33%) and 105 (67%) non-frail. Prevalence by cognitive frailty was: Frail 62 (40%) and 94 (60%) non-frail. Frailty, either physical or cognitive, was associated with lower BMI, NYHA class IV, hypoalbuminaemia and anaemia ($p < 0.01$). Cognitive frailty was additionally associated with increased right arterial pressure and lower cardiac index ($p < 0.05$). Actuarial survival curves are shown in figure 1 for physical (a) and cognitive (b) frailty. Survival adjusted for bridge-to-transplant ventricular assist device (BTT-VAD) and heart transplant (HTx) are also shown in figure 1 (c)/(d).

Conclusion: Cognitive frailty was highly prevalent and the addition of CI to PF, provided a better predictor of early mortality in transplant referred patients.

Abstract No. 78 (continued)

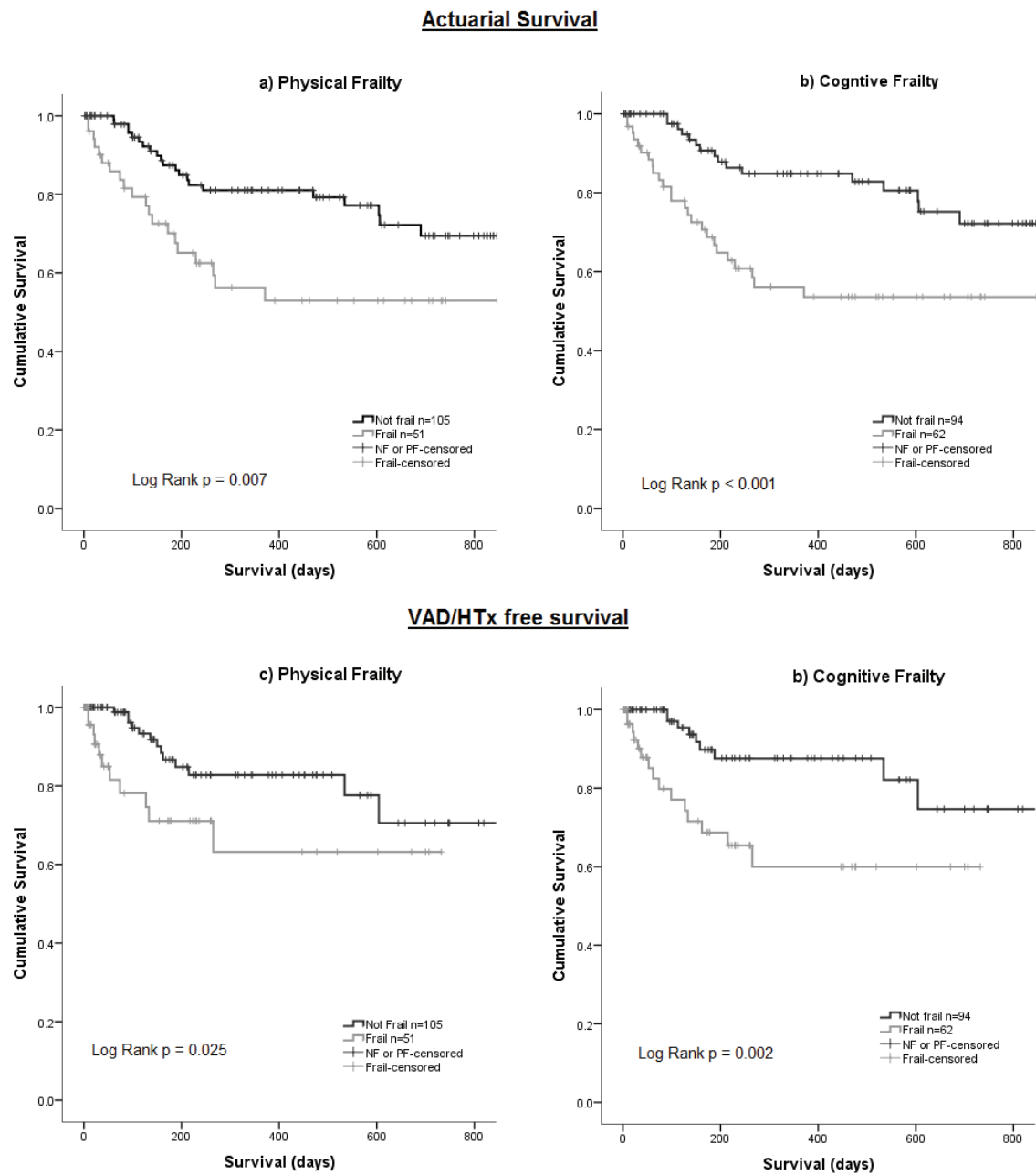


Figure 2: Actuarial and VAD/HTx-free kaplain-meier survival curves for physical and cognitive frailty.

Abstract No. 79

AUTOPHAGY-DEPENDENT TIGIT⁺ REGULATORY T CELLS ARE CRITICAL FOR THE MAINTENANCE OF TOLERANCE AND THE CONTROL OF GRAFT VERSUS HOST DISEASE
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Regulatory T cells (Treg) play a crucial role in the maintenance of peripheral tolerance. Quantitative and/or qualitative defects in Treg result in diseases such as autoimmunity and graft-versus-host disease (GVHD), a serious complication of bone marrow transplantation (BMT). We recently reported that G-CSF mobilisation of stem cells in donor mice increases autophagy related genes in Treg and this is associated with an improved GVHD outcome after BMT. Autophagy is a self-degradative process for cytosolic components which promotes cell homeostasis and survival. Here, we demonstrate that the lineage-specific disruption of autophagy within FoxP3⁺ Treg (Atg7^{fl/fl}.FoxP3cre⁺ mice) resulted in a profound loss of Treg within the bone marrow (BM). This resulted in dysregulated effector T cell activation and expansion, and in aged mice, the development of enterocolitis and severe scleroderma. We show that the BM compartment is highly enriched in TIGIT⁺ Treg and that this subset is differentially lost in the absence of autophagy. Moreover, G-CSF administration to naive mice results in the mobilization of BM TIGIT⁺ Treg explaining the autophagy signature of peripheral Treg in G-CSF treated mice. Importantly, lethally irradiated B6D2F1 recipients of splenic grafts from G-CSF mobilized B6.Atg7^{fl/fl}.FoxP3cre⁺ donors failed to reconstitute the Treg compartment within the BM. This resulted in GVHD exacerbation and reduced survival compared to recipients of B6.WT.FoxP3cre⁺ grafts. Collectively, these data indicate that autophagy-dependent TIGIT⁺ Treg in the BM are critical for the maintenance of tolerance and to control GVHD; thus the induction of autophagy may represent an attractive immune-restorative therapeutic strategy.

Abstract No. 80

AGREEMENT BETWEEN NUMBER OF DONOR/RECIPIENT EPLET MISMATCHES CALCULATED USING TWO-DIGIT SEROLOGICAL VERSUS FOUR-DIGIT MOLECULAR HUMAN LEUKOCYTE ANTIGEN (HLA)-TYPING**FIDLER SAMANTHA¹, WONG GERMAINE², LEWIS JOSHUA³, LIM WAI⁴**¹*Department of Clinical Immunology, Fiona Stanley Hospital,* ²*Westmead Millennium Institute, Westmead Hospital, Sydney,* ³*University of Sydney,* ⁴*Sir Charles Gairdner Hospital, Perth*

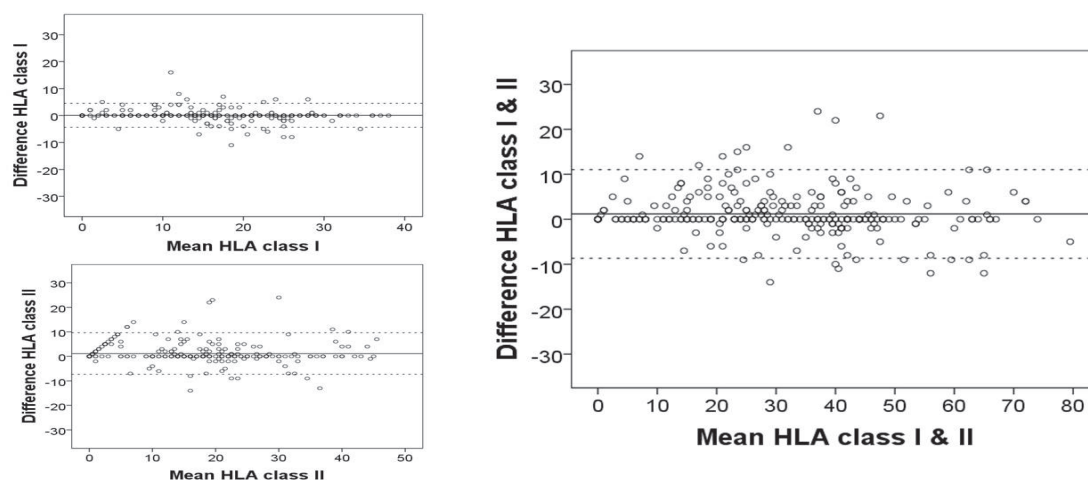
Aim: We aimed to assess the agreement of the number of eplet mismatches at the HLA-A, -B, -C, -DQ and -DR loci determined by 2-digit serological and 4-digit molecular typing.

Methods: We included patients who received live or deceased-donor kidney transplants between 2003 and 2007. Donor and recipient serological typing was determined using complement-dependent cytotoxicity, and molecular 4-digit typing determined using sequence based typing (Sanger). The number of eplet mismatches was calculated by converting the 2 and 4-digit HLA-typing using HLA-Matchmaker. Correlation and agreement of HLA-A, -B, -C, -DP and -DR mismatches between the two methods was analysed using Spearman rank correlation and Bland Altman plots respectively. HLA-DP can only be determined using 4-digit typing and therefore not included in this study.

Results: Of 264 kidney transplant recipients, 86 (33%) were females. The correlation between class I (HLA-A, -B, -C) and class II (HLA-DQ, -DR) between 2 and 4-digit converted eplet mismatches were 0.966 and 0.931 respectively. Bland-Altman's limits of agreement between class I, class II and combined class I and II eplet mismatches using 2 and 4-digit typing is shown below (figure 1). The number of class I and II eplet mismatches determined by 4-digit conversion exceeded that of 2-digit conversion in 37% of recipients, with 10% exceeding 7 eplet mismatches. One hundred and sixteen (44%) recipients had identical number of class I and II eplet mismatches as determined by 2 and 4-digit typing. Of the 21 "outliers" for both class I and II eplet mismatches, 5 (24%) of patients were females and 8 (38%) of either patients or donors were non-Caucasians. A further 4 (19%) had unusual (non-Caucasian) HLA alleles.

Conclusions: It appears that there is good correlation and agreement between 2 and 4 digit typing for total eplet mismatches at the HLA-A, -B, -C, -DQ and -DR loci. Future research should focus on exploring the clinical significance of total eplet mismatches determined by the two different methods.

Figure 1.



Intraclass correlation coefficients* for consistency and absolute agreement (n=264)

	Consistency 4 Digit (95% CI)	Absolute agreement 4 Digit (95% CI)
2 Digit		
HLA-A	0.995 (0.994-0.996)	0.995 (0.994-0.996)
HLA-B	0.983 (0.978-0.987)	0.982 (0.977-0.986)
HLA-C	0.875 (0.843-0.900)	0.875 (0.843-0.900)
Class I	0.969 (0.960-0.975)	0.969 (0.960-0.975)
HLA-DR	0.987 (0.984-0.990)	0.987 (0.983-0.992)
HLA-DQ	0.810 (0.764-0.848)	0.802 (0.748-0.845)
Class II†	0.935 (0.918-0.949)	0.931 (0.908-0.948)

* Two-way fixed intraclass correlation coefficients. † Excluding HLA-DP.

Transplant Complications #2

Abstract No. 81

A PROSPECTIVE STUDY OF RENAL TRANSPLANT RECIPIENTS: THE INTER-RELATIONSHIP BETWEEN INSULIN SECRETION AND SENSITIVITY UNDERPINS DYSGLYCEMIA POST FOLLOWING RENAL TRANSPLANTATION

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Background: Dysglycemia (encompassing impaired glucose tolerance and diabetes) arising after renal transplantation is common and confers a significant cardiovascular mortality risk. The aim of this study was to prospectively and comprehensively assess glucose handling in renal transplant recipients from before to 12 months after transplantation in order to determine the underpinning pathophysiology.

Methods: Complete 12 month data was obtained on 14 renal transplant recipients. Intravenous and oral glucose tolerance testing was conducted prior to and at 3 and 12 months following transplantation. An additional intravenous test was performed on day 7. Insulin secretion, resistance and calculation of the disposition index (DI), a measure of beta cell responsiveness in the context of prevailing insulin resistance were determined.

Results: At 12 months 50% of renal transplant recipients had dysglycemia. This cohort were older with features of the metabolic syndrome. Dysglycemia was associated with a dramatic fall in DI and this loss in beta cell function was evident as early as 3 months post transplantation. Differences in the beta cell response to oral glucose challenge were evident pre-transplant in those destined to develop dysglycemia post-transplant.

Conclusion: Dysglycemia following renal transplantation occurs in older patients with features of the metabolic syndrome. The predominant mechanism is loss of insulin secretion. Subclinical differences in glucose handling are evident pre-transplant in those destined to develop dysglycemia potentially heralding a susceptible beta cell which under the stressors associated with transplantation fails.

Abstract No. 82

IL-17 REGULATES INTESTINAL DYSBIOSIS AND IS CRITICAL FOR THE PREVENTION OF INTESTINAL GRAFT-VERSUS-HOST DISEASE

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Donor T cell-derived IL-17A can mediate immunopathology in graft-versus-host disease (GVHD). However protective roles for IL-17A are also widely reported in autoimmune disease settings but the nature of any such effect remains unclear in allogeneic stem cell transplantation (allo-SCT). Using systems that utilize multiple cytokine and cytokine receptor subunit knockout mice we demonstrate that allo-SCT recipients lacking the ability to generate or signal IL-17A develop hyper-acute GVHD within the gastrointestinal tract. This protective effect is restricted to the IL-17A and/or IL-17F molecular interaction with the IL-17 receptor as the IL-17E and IL-17B molecules did not invoke a similar effect upon ligation to their cognate receptor (IL-17RB). Conditional IL-17 deletion peri-transplant also enhanced GVHD. The protection required IL-17A/F secretion from and signaling in both hematopoietic and non-hematopoietic host tissue. Critically, hyper-acute GVHD occurred in IL-17A deficient recipients in the absence of all recipient and donor IL-17A signaling, suggesting a developmental contribution. Microbiome analysis of gut flora using 16S ribosomal RNA gene sequencing confirmed dramatic perturbation in the gut microbiota of these mice whilst additional defensin defects were seen within 48 hrs of conditional IL-17 deletion after BMT. Co-housing experiments transferred the IL-17 phenotype to wild type mice in association with a clear convergence of microbiota composition and the identification of multiple putative pathogenic bacteria. Thus IL-17 is a critical mediator of microbiome homeostasis and gastrointestinal tract immunity that determine transplant outcome.

Abstract No. 83

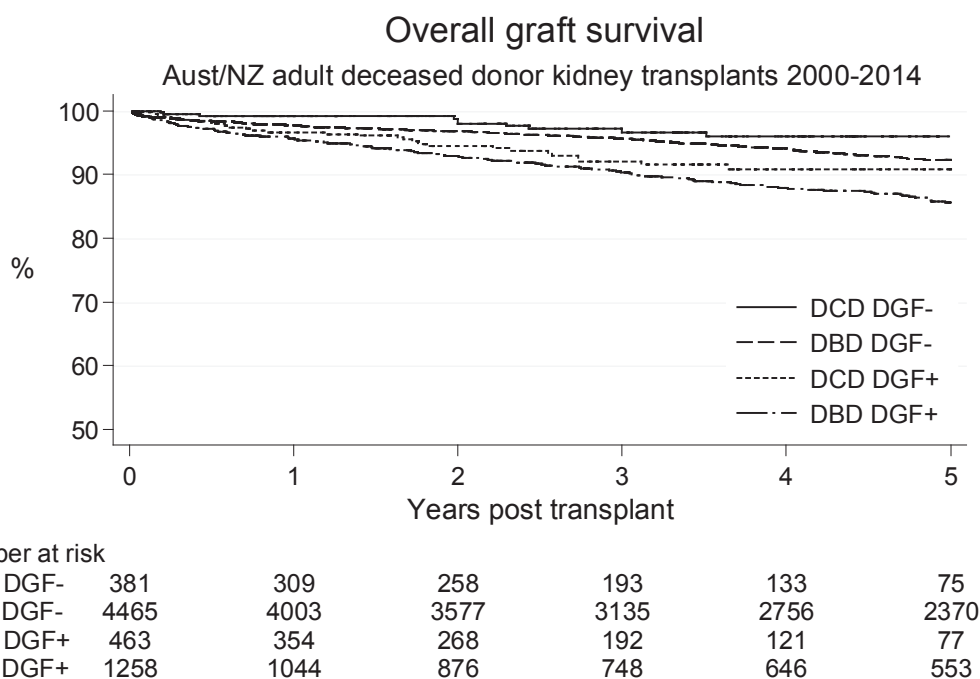
ASSOCIATION BETWEEN DELAYED GRAFT FUNCTION AND LONG-TERM OUTCOMES AFTER KIDNEY TRANSPLANTATION FROM DONORS AFTER CIRCULATORY DEATH**CLAYTON Philip^{1,2,3}, RUSS Graeme^{1,2,3}, MCDONALD Stephen^{1,2,3}, CHADBAN Steve^{1,4,5}**¹*Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry, Royal Adelaide Hospital,* ²*Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital,* ³*School of Medicine, University of Adelaide,* ⁴*Transplantation Services, Royal Prince Alfred Hospital, Sydney,* ⁵*Sydney Medical School, University of Sydney*

Aims: Delayed graft function (DGF) is associated with worse long-term outcomes after kidney transplantation from donation after brain death (DBD) donors. We aimed to determine whether this association held for transplantation from donation after circulatory death (DCD) donors.

Methods: Using data from the Australia and New Zealand Organ Donor (ANZOD) and Australia and New Zealand Dialysis and Transplant (ANZDATA) Registries, we included adult recipients of deceased donor kidney-only transplants in Australia and New Zealand over 2000-2014 (n=6748), excluding grafts with <7 days function (n=181). DGF was defined as the need for dialysis within 72h of transplantation. The relationship between DGF and death-censored graft survival was examined in a Cox model adjusted for donor, transplant and recipient factors.

Results: DGF occurred in 22% of DBD and 55% of DCD transplants (p<0.001). On unadjusted analysis DGF was associated with worse death-censored graft survival in both DBD and DCD donors (figure). After adjusting for confounders, the hazard ratio (HR) (95% CI) for DGF was 1.32 (1.11, 1.58) in DBD transplants and 1.77 (0.87, 3.58) in DCD transplants (interaction HR 1.34 (0.65, 2.77), p=0.11).

Conclusions: DGF was associated with worse death-censored graft survival in both DBD and DCD kidney transplants, with no statistically significant difference in the magnitude of this association between DBD and DCD transplants.



Abstract No. 84**SHORT TERM SURGICAL COMPLICATION RATES IN TRANSPLANT SURGERY: CONSULTANT VS TRAINEE****SALTER Sherry¹, CHOU Angela¹, PLEASS Henry^{1,2}, HAWTHORNE Wayne^{1,3}**¹*Department of Surgery, Westmead Hospital, Sydney*, ²*Discipline of Surgery, Sydney Medical School, University of Sydney*, ³*Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney*

Aim: To investigate whether there is a significant difference between the short term surgical complication rates when either the Consultant or the Trainee is the primary surgeon for transplant operations.

Method: All patients who had a cadaveric, live renal or simultaneous pancreas kidney (SPK) transplant at Westmead Hospital from August 2014 – August 2015 were included. Data was collected on patient demographics, primary surgeon and surgical complications. Short term surgical complication was defined as a complication that occurred within 30 days of surgery. Complications were graded using Clavien-Dindo classification and also grouped into types of complication.

Results: There were 101 patients in this study (21 live, 53 cadaveric renal transplants and 27 SPK). There were 55 complications in 46 patients with bleeding as the predominant complication. The majority of complications were in the Clavien-Dindo Grade III group (requiring surgical or radiological intervention). The SPK complication rate for the Consultant was 75% compared with 66.7% ($p=0.64$) for the Trainee. The live renal transplant complication rate for the Consultant was 41.2% compared with 25% ($p=0.55$) for the Trainee. The cadaveric renal transplant complication rate for the Consultant was 64% compared with 39.3% ($p=0.07$) for the Trainee.

Conclusion: This study found that there is no significant difference between the short term surgical complication rates when either the Consultant or Trainee was the primary surgeon for all three transplant operations. However, the Consultant compared to the Trainee had the higher complication rate for all transplant operations. This result may reflect that Consultants typically operated on the more complex cases.

Abstract No. 85**NEPHROGENIC ADENOMA - A CASE REPORT AND UPDATE****NORTH Daniel¹, JAW Juli¹, HILL Prue², BATEMAN Samantha¹, BARRACLOUGH Nick^{3,4}, LANGHAM Robyn^{1,5}**¹*Department of Nephrology, St Vincent's Hospital, Melbourne*, ²*Department of Anatomical Pathology, St Vincent's Hospital, Melbourne*, ³*Department of Medicine, South West Healthcare*, ⁴*Faculty of Health, Deakin University*, ⁵*School of Medicine, University of Melbourne*

Nephrogenic adenoma (NA) is a benign adenomatous lesion of the urinary tract. Long considered a rare phenomenon, case series from the renal transplant population suggest that it may be much less uncommon within this group. The pathogenesis of NA remains to be clearly elucidated, however recent studies support the hypothesis that NA represents the proliferation of renal tubule cells that have been shed, and re-implanted within the lower urinary tract. While NA is considered a lesion with low pre-malignant potential; haematuria, lower urinary tract symptoms and recurrent urinary tract infections are frequently observed in the context of NA. Furthermore, following resection of NA, lesion recurrence and persistent symptoms frequently remain problematic. Here we present the case of a 69-year-old male renal transplant recipient with NA, and a review of the literature. Our patient's clinical course was characterised by recurrent urinary tract infection with associated graft dysfunction, despite cystoscopic resection of the primary lesion. This case is illustrative of the clinical impact of NA, and the need for ongoing work into the development of strategies to manage this problematic phenomenon.

Abstract No. 86**SCLEROSING PERITONITIS FOLLOWING LIVER TRANSPLANTATION: A CASE SERIES****KONG Y, TAN AL, VERRAN DJ***Australian National Liver Transplantation Unit, Royal Prince Alfred Hospital, Sydney*

Background: Sclerosing peritonitis (SP) is a rare but potentially fatal complication following liver transplantation. It has been linked to refractory ascites, biliary obstruction, IVC and porta hepatis obstruction. The definitive surgical management is via laparotomy and PEEL procedure, but carries risks particularly in the immunosuppressed transplant patient population. The natural history of SP is known from a handful of case reports and series, which report de novo cases arising early in the weeks to months following transplantation.

Aims: To identify the incidence of SP following orthotopic liver transplantation (OLT) and the outcomes post management.

Methods: 2 cases of late development of SP post OLT were identified from the Australian National Liver Transplantation Unit database.

Results: 2 cases of SP were identified, one diagnosed at 2 years and the other at almost 10 years post transplantation. Both patients presented with symptoms suggestive of small bowel obstruction. CT scan of the abdomen of patient 1 was suggestive of an internal hernia and patient 2 showed a transition point in the distal jejunum. After failing conservative measures, both patients proceeded to laparotomy and a PEEL procedure was undertaken of the cocooned bowel.

Conclusions: SP should be considered as a differential diagnosis in patients post OLT presenting with symptoms of bowel obstruction, even years after the transplantation.

Abstract No. 87**CIRCULATING DNA: AN APPROACH TO MONITOR ORGAN REJECTION AFTER LIVER TRANSPLANTATION.****GOH Su Kah^{1,2}, DO Hongdo^{2,4,4}, MURALIDHARAN Vijayaragan¹, DOBROVIC Alexander^{2,3,4}, CHRISTOPHI Chris^{1,5}***¹Department of Surgery, Austin Hospital, Melbourne, ²Translational Genomics and Epigenomics Laboratory, Olivia-Newton John Cancer Research Institute, ³Department of Pathology, University of Melbourne, ⁴School of Cancer Medicine, La Trobe University, ⁵Liver Transplant Unit Victoria, Austin Hospital, Melbourne*

Aims: Up to twenty percent of patients will develop an episode of rejection in the first twelve months after liver transplantation. Liver biopsy is the gold standard for the diagnosis of organ rejection. However, this procedure is invasive and carries a risk of bleeding and sepsis. Recent studies have proposed the use of donor-specific circulating cell-free DNA (dscfDNA) as a blood-based biomarker for organ rejection. Unlike current methodologies used to quantify dscfDNA, we aimed to develop a rapid and cost-effective approach for serial monitoring of graft health after liver transplantation.

Methods: Five patients undergoing liver transplantation were prospectively recruited. Droplet digital PCR was used to analyze recipient blood samples collected at various timepoints. This PCR platform allow precise quantification of dscfDNA molecules in the circulation of the recipient. The levels of dscfDNA were compared with serum liver biochemistry and clinicopathological factors.

Results: Levels of dscfDNA were reflective of graft health. Marked increase in dscfDNA levels were observed in one patient who developed an episode of acute cellular rejection. Cholestasis did not increase the levels of dscfDNA after liver transplantation. Turnaround time for quantification of dscfDNA is attainable under 6 hours.

Conclusion: Our methodology to accurately quantify dscfDNA was feasible and clinically applicable. Furthermore, our preliminary results suggest that this non-invasive biomarker can facilitate timely and serial monitoring of graft health for organ rejection.

Abstract No. 88**CELL-FREE DNA CAN IDENTIFY MILD CELL MEDIATED REJECTION IN PAEDIATRIC HEART TRANSPLANT RECIPIENTS****WHITLAM JB^{1,2}, LING L², HARRINGTON T², PRAPORSKI S³, BRUNO D², POWER D¹, KONSTANTINOV I³, SLATER H²****¹Department of Nephrology, Austin Hospital, Melbourne, ²Victorian Clinical Genetics Services, Murdoch Childrens Research Institute, Melbourne, ³Department of Cardiothoracic Surgery, Royal Children's Hospital, Melbourne**

Aims: To measure graft-derived cell-free DNA (gdcfDNA) and total cfDNA (tcfDNA) in paediatric heart transplant recipients (PHTR) as proof of principle application of our digital droplet PCR (ddPCR) methodology based upon ubiquitous copy number variation (CNV) differences between donor and recipient.

Methods: Cf-DNA was extracted from plasma collected longitudinally from 13 PHTR. A panel of ddPCR assays directed at polymorphic CNV loci was used to absolutely quantify gdcfDNA and tcfDNA. Variation in cfDNA levels over time was correlated with protocol biopsy results.

Results: In all recipients, gdcfDNA and tcfDNA was detected and quantified. There were a total of 17 rejection episodes (16xgrade 1 cell mediated rejection (CMR) and 1xgrade 2 CMR). The combined interpretation of gdcfDNA, tcfDNA and graft fraction was able to identify CMR (by rising above baseline levels before or on the day of biopsy) in 9/17 occasions. In 5/8 false negatives, there was insufficient sampling, no preceding normal biopsy or excessive variation that prevented establishment of a baseline level. Establishment of a diagnostic threshold was difficult given variation in observed baseline levels. In a further 2/8 false negatives, the failed identification of rejection occurred after successful identification of an earlier episode.

Conclusion: GdcfDNA can be quantified in PHTR using our methodology. Given other genomic diagnostic methods have performed poorly in paediatric recipients and detection of mild CMR, our method warrants further investigation. More frequent sampling and correction for body size/weight may improve diagnostic performance.

Abstract No. 89**PYELOURETERIC JUNCTION OBSTRUCTION OF RENAL ALLOGRAFTS****GRAJN Andrej, GRIFFIN Anthony D, PRESTON John, WOOD Simon, LAWSON Malcolm**
Transplantation Services, Princess Alexandra Hospital, Brisbane

Congenital Pyeloureteric Junction (PUJ) obstruction is present in 1/500 live births. Baggy extra renal pelvises are more frequently encountered. PUJ obstruction in kidney transplantation an uncommon problem and literature is sparse. Following transplantation the obstruction may become urodynamically significant due to diuresis, autonomic denervation, minor ureteric torsion, kink, external scarring or reduced blood supply.

We report two cases of PUJ obstruction in renal allograft recipients. In the first case, following deceased donor transplant, PUJ obstruction was diagnosed following stent removal. In the second case a 5 mm stone was noted in baggy extra renal pelvis during live donor assessment. Back table flexible ureteroscopy was planned with standard CH6 instrument. During the procedure the ureteroscope could not be advanced though the PUJ.

Options for reconstruction of transplant PUJ obstruction include native ureteropyelostomy, Boari flap vesicopyelostomy, non-dismembered flap reconstruction, long term stenting or nephrostomy. Both of our cases were reconstructed with native ureteropyelostomy with good intermediate term outcomes.

From Disease to Donation to the Final Destination – Where is Your Patient on the Transplant Journey?

Abstract No. 90

MARGINAL DONOR HEARTS ASSESSED WITH EX-VIVO PERFUSION TO FACILITATE ORTHOTOPIC HEART TRANSPLANTATION

CHAN Samuel, MUDGE David, JOHNSON David, CAMPBELL Scott, FRANCIS Ross

Department of Nephrology, Princess Alexandra Hospital, Brisbane

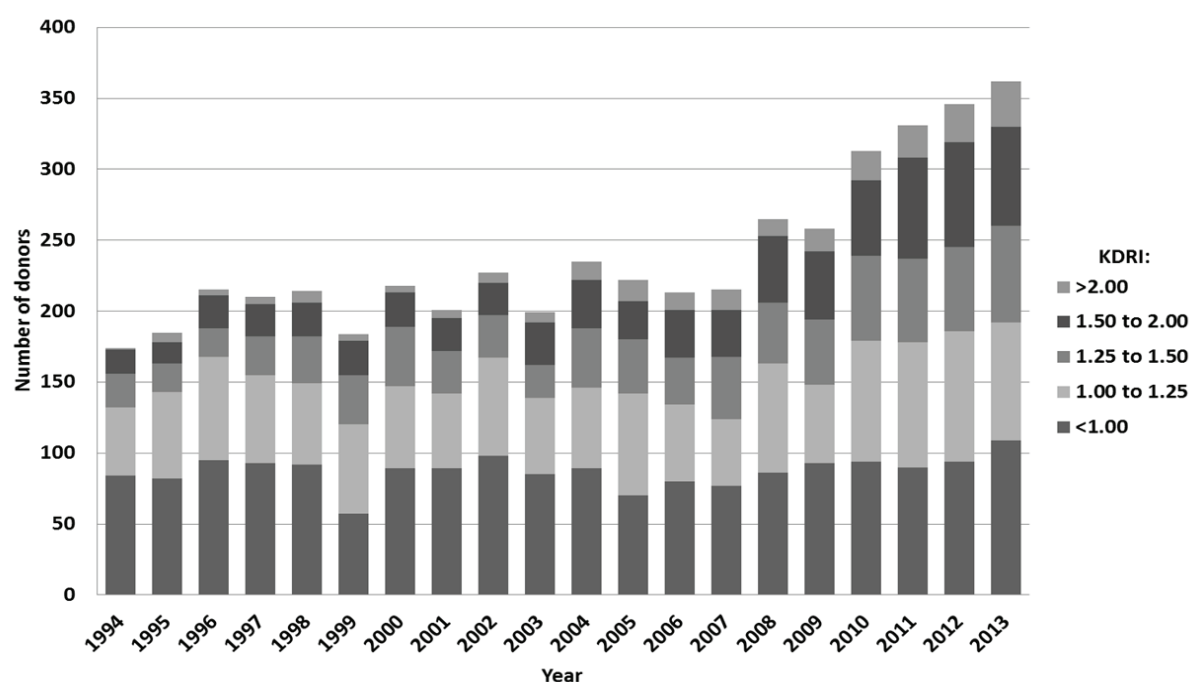
A key challenge in donor heart utilization lies in differentiating marginal hearts that have the capacity to function normally after transplantation as opposed to those which have been irrevocably damaged. The aim of this study was to review our experience with the use of the TransMedics™ Organ Care System (OCS) in the evaluation and transplantation of marginal donor hearts.

Marginal brain dead (BD) donor hearts were retrieved from donors with: left ventricular ejection fraction (EF) <50%, interventricular septum > 12mm, requirement for noradrenaline >0.2ug/kg/min or donation after circulatory death (DCD) donors. All donor hearts were rejected for standard cold storage retrieval. Seven marginal hearts from BD and five from DCD donors were retrieved on the OCS from October 2012 to December 2014, resulting in 9 successful transplants (75%). Three hearts (2 BD and 1 DCD) did not demonstrate satisfactory function on the OCS and were not utilized.

Postoperatively, all transplant recipients had a normal EF ($64 \pm 3\%$) at 9 ± 5 days. Echocardiography at 122 ± 76 days was preserved in eight of nine recipients (EF $61 \pm 6\%$). One patient was implanted with a Total Artificial Heart 8 months post transplant, having experienced intractable rejection and remains on support. There was no early mortality, however one infection related late death occurred over a follow-up period of 227 ± 80 days.

Donor heart perfusion on the OCS has allowed for an increase in our transplant numbers by utilizing previously rejected marginal hearts in a reproducible manner with satisfactory outcomes.

Abstract No. 91

CHANGE IN DECEASED KIDNEY DONOR CHARACTERISTICS IN AUSTRALIA AND NEW ZEALAND OVER TWENTY YEARS**CHAN Samuel^{1,2,3}, CAMPBELL Scott B^{1,2,3}, CLAYTON Philip A^{4,5}, MUDGE David W^{1,2,3}, JOHNSON David W^{1,2,3}, FRANCIS Ross S^{1,6,3}**¹*Nephrology and Renal Transplant, Princess Alexandra Hospital, Brisbane,* ²*School of Medicine, Faculty of Health Sciences, University of Queensland, Brisbane,* ³*Queensland Renal Transplant Service, Princess Alexandra Hospital, Brisbane,* ⁴*ANZDATA,* ⁵*Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital,* ⁶*School of Medicine, Faculty of Health Sciences, Princess Alexandra Hospital, Brisbane***Aim:** To review changes in deceased kidney donor characteristics in Australia and New Zealand between 1994 and 2013.**Methods:** Retrospective analysis of deceased kidney donor data from the Australia and New Zealand Organ Donor Registry using the Kidney Donor Risk Index (KDRI) as a surrogate measure of donor quality.**Results:** Of 4689 deceased donors, 57% were male and 89% were from Australia. Mean donor age increased from 35.7 ± 16 years to 46.1 ± 17.6 years over time. There was an increase in donor numbers from 158 in 1994 to 360 in 2013. For primary cause of death, motor vehicle accidents reduced from 16.5% to 3.5% and cerebral pathology increased from 52% to 64%. There was an increase in donors with hypertension (11.4% to 24.2%), diabetes (1.3% to 7%) and a rise in mean body mass index (24.4 ± 4.4 kg/m² to 27.4 ± 6.2 kg/m²) between 1994 and 2013. These changes were reflected by a rise in the median KDRI from 1.01 ± 0.29 to 1.22 ± 0.4 (slope for linear regression of log-transformed KDRI = 0.009 (95% CI 0.007 to 0.010, $p < 0.001$). When divided into 5 categories of KDRI, the proportion of higher risk donors has increased over time (Figure 1).**Figure 1: Total number of donors by KDRI****Conclusions:** As deceased kidney donor numbers have risen, the range of donor quality has increased, and average donor quality has decreased. These data highlight the need for kidney allocation algorithms to evolve to ensure appropriate allocation of both better quality and more marginal kidneys.

Abstract No. 92**A NOVEL CARDIAC ALLOCATION SCORE FOR PREDICTING WAIT-LIST AND POST-TRANSPLANT SURVIVAL**

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¹*School of Medicine, Faculty of Health Sciences, University of New South Wales, Sydney*, ²*School of Medicine, University of New South Wales, Sydney*, ³*Department of Cardiology, St Vincent's Hospital, Sydney*

Aims: We propose that a survival-benefit allocation scheme similar to the lung allocation score should be considered to increase the utility of each heart donated. In this study, a novel cardiac allocation score (CAS) algorithm was developed that identifies the patient on the waiting list with the greatest expected survival benefit from transplantation.

Methods: All adult patients from the cardiac transplant waiting list at our institution, between 2008 and 2015 were included in the study (n = 260). Univariate survival analysis of pre-listing variables was undertaken to ascertain factors that significantly predicted 1-year mortality on the waiting list and post-transplant. Significant variables were placed in a multivariate Cox Regression to develop models predicting pre- and post-transplant survival of wait-list patients. The difference in estimated pre- and post-transplant survival time gave the predicted survival benefit of transplantation; this measure was normalised, and a score between 0 and 100 was given to each patient, a higher score correlating with a higher expected post-transplant survival benefit.

Results: Two statistically significant models were developed to predict waiting list mortality (P<0.0001) and post-transplant mortality (P=0.0106) respectively. A higher CAS was shown to significantly predict higher risk for wait list mortality (HR: 1.110738, P<0.001) and lower risk for post-transplant mortality (HR: 0.8715659, P<0.001).

Conclusion: We present a novel allocation system for donor hearts that considers the expected waiting list and post-transplant survival. The clinical adoption of such a model could allow better transplant prioritisation and achieve improved outcomes for recipients post-orthotopic heart transplantation.

Abstract No. 93**EVALUATION OF A TRANSITION PROGRAM FOR ADOLESCENTS WITH SEVERE LIVER DISEASE/LIVER TRANSPLANT**

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Background: Failure of adequate transition from paediatric to adult care results in morbidity, graft loss and mortality in liver transplant recipients.

Aims We aimed to assess the effect of a formalised transition program using a standardised healthcare skills checklist (HSC)

Methods: Adolescents >15 years with severe liver disease/liver transplant completed a standardised HSC. They and their parents underwent a formal transition clinic program involving a transition manager, transition coordinator and youth mentor. Competencies and gaps were identified and individual goals developed. After 2 or more transition clinic visits and prior to transfer, the HSC was administered again.

Results: Of 32 adolescents who completed the program with a median of 2.3 visits, a significant improvement was seen across the following domains: Knowledge of condition (43% to 80%), knowledge of medications (50% to 80%), adherence to treatment (52% to 74%), confidence in speaking about their liver disease (30 to 71%) and independence with healthcare (16% to 55%). Anxiety regarding transfer was significantly reduced (82 to 29%).

Conclusions: A formalised transition program appears to significantly improve patient readiness for transition. We are currently assessing whether this improved knowledge is retained after the transfer process, and whether it translates to improved patient outcomes.

Abstract No. 94

KIDNEY TRANSPLANT PATIENT PREFERENCES AND TRADE-OFFS FOR OUTCOMES AFTER TRANSPLANTATION.**HOWELL Martin^{1,2}, WONG Germaine^{1,3,2}, ROSE John⁴, TONG Allison^{1,2}, CRAIG Jonathan^{1,2}, HOWARD Kirsten²**¹Centre for Kidney Research, The Children's Hospital at Westmead, Sydney, ²School of Public Health, University of Sydney, ³Centre for Transplant and Renal Research, Westmead Hospital, Sydney, ⁴Institute of Choice, University of South Australia


Aims: Patient preferences in clinical decisions are increasingly relevant, yet transplant patient perspectives through structured, quantified preference elicitation methods are unknown. We aimed to evaluate preferences and trade-offs patients may accept to avoid adverse outcomes of long-term immunosuppression.


Method: Preferences and trade-offs between graft duration and the risk of dying, cancer, cardiovascular disease (CVD), diabetes, infection, anxiety/depression, diarrhoea/nausea, and weight gain, was assessed using a best-worst scaling survey. Participants, recruited from clinics, were shown 10 scenarios with varying outcome probabilities and years of graft duration and asked to choose best, next-best, worst and next-worst outcomes. Responses were analysed with multinomial-logit models.

Results: Ninety-three patients (age 18-69 years, transplant duration 0.9-31 years) completed the survey. The most important outcome was death followed by (in order), duration of graft survival, cancer, CVD, anxiety/depression, infection, diarrhoea/nausea, diabetes and weight gain. A 0% risk of dying ($\beta=1.0$ 95%CI:0.92,1.08) was more important than 25 years of graft survival ($\beta=0.8$ 95%CI:0.64,0.78). One year graft survival ($\beta=0.0$ 95%CI:-0.05,0.05) was worse than any other outcome including 100% risk of dying ($\beta=0.19$ 95%CI:0.13,0.24). Preferences varied with age, gender, comorbidities, dialysis and transplant duration, and number of transplants (Table 1). Respondents were willing to trade 2.8 (95%CI:1.8,4.1), 1.5 (95%CI:1.0,2.2), 1.1 (95%CI:0.8,1.7) and 0.7 (95%CI:0.5,1.1) years graft survival to achieve a 0% probability of cancer, dying, CVD and anxiety/depression respectively.

Conclusion: Preferences and trade-offs suggest that 5 years graft survival is worse than dying and serious outcomes including cancer. Long-term clinical studies are needed to support patient preferences and shared-decisions.

Patient Characteristic	Adverse events							
	Graft survival	Dying before graft fails	Cancer	CVD	Diabetes	Serious infection	Severe diarrhoea or nausea	Severe anxiety or depression
Older age	*							
Women								
More comorbidities	**							
More years on dialysis								
Longer years since transplant								
More than 1 transplant								

Less important 

More important 

* Short graft duration

** Long graft duration

Table 1. Transplant recipient characteristics associated with outcome preference (P<0.05).

Abstract No. 95**TRANSPLANTATION AND DIABETES (TRANSDIAB): A PILOT RANDOMISED CONTROLLED TRIAL OF METFORMIN IN PRE-DIABETES AFTER KIDNEY TRANSPLANTATION****ALNASRALLAH Basil¹, PILMORE Helen^{2,1}, MANLEY Paul^{2,1}**¹*Department of Renal Medicine, Auckland City Hospital,* ²*Auckland Renal Transplant Group, Auckland City Hospital***Aim:** To evaluate the feasibility, safety and tolerability of Metformin in prediabetic kidney transplant patients.**Background:** Diabetes occurs in up to 50% of patients after kidney transplantation and is associated with increased mortality and transplant failure. Impaired glucose tolerance (IGT) is a known risk factor for developing diabetes. We are undertaking a pilot RCT to evaluate the feasibility, safety and tolerability of Metformin in prediabetic kidney transplant patients.**Methods:** All patients without pre-existing diabetes who receive renal transplants at Auckland City Hospital from November 2014 are considered for the trial. Consenting patients have an oral glucose tolerance test (OGTT) performed 4-12 weeks post transplantation. Patients with IGT are randomized to standard care versus standard care plus Metformin 500mg bd and followed up for 12 months. Feasibility is examined by consent rate, tolerability by using Gastrointestinal Symptom Rating Scale and safety by recording adverse events.**Results:** 73 patients received renal transplantation within 12 months. 56 patients (76.7%) had no pre-existing diabetes. 5 patients were excluded. 37 out of 51 (72.5%) eligible patients consented. Of these, 8 had NODAT, 16 had normal OGTT and 13 had IGT. 2 patients with IGT (15.4%) were excluded prior to randomization (1 withdrew consent, 1 clinical decision). 11 patients were randomized, 6 to the Metformin arm and 5 to the standard care arm.**Conclusion:** A high proportion of patients had either NODAT or IGT after kidney transplantation. Tolerability and safety of Metformin will be analysed as markers of feasibility to examine this drug in a larger RCT.Abstract No. 96**RESEARCH PRIORITY SETTING IN ORGAN TRANSPLANTATION: A SYSTEMATIC REVIEW****HARPER Claudia^{1,2}, CRAIG Jonathan^{1,2}, CHAPMAN Jeremy³, TONG Allison^{1,2}**¹*School of Public Health, University of Sydney,* ²*Centre for Kidney Research, The Children's Hospital at Westmead, Sydney,* ³*Centre for Kidney Research, Westmead Hospital, Sydney***Aims:** We aimed to evaluate approaches to research priority setting in solid organ transplantation and to describe the research priorities of patients, their caregivers, healthcare providers, and policy makers.**Methods:** Electronic databases were searched to December 2015. Studies that elicited patient, caregiver, healthcare provider, or policy maker priorities for research in solid organ transplantation were included.**Results:** We identified 16 studies (n=1250 participants) conducted in the United States, the Netherlands, Australia and Canada. The studies focused on kidney (8 [50%] studies), heart (2 [13%] studies), lung (1 [6%] study), and non-specified solid organ transplantation (5 [31%] studies). Various priority setting methods were used including the Delphi technique, expert panels, consensus conference, ranking or voting surveys, focus groups and interviews, of which the process was described in detail by 10 (63%) studies. Only six (38%) studies reported patient involvement. The priority areas for research were: improving immunosuppression (12 [75%] studies), organ donation and allocation (9 [56%] studies), psychosocial support including adherence (8 [50%] studies), patient communication and education (7 [44%] studies), organ preservation (2 [13%] studies), wait-listing (1 [6%] study), and prevention of diabetes (1 [6%] study).**Conclusion:** The research priorities identified in solid organ transplantation are broad in scope. However, few priority setting initiatives engage patients and just over half have a well-described process. Setting research priorities in an explicit manner with equitable involvement of patients can help to ensure that resources are directed towards research that is important and relevant to patients and health professionals in solid organ transplantation.

Abstract No. 97**NEUROCOGNITIVE DEFICITS IN CHILDREN TRANSPLANTED IN EARLY CHILDHOOD****ROBINSON Lucy, KARA Tonya***Auckland Renal Transplant Group, Starship Children's Hospital*

Aims: The impact of early end stage kidney disease on neurological development is unclear. We hypothesised that children who have received dialysis followed by transplantation in early life may show normal physical growth and development whilst subtle cognitive deficits may not be recognised. This may have an impact on education, work, and ability to manage issues such as adherence and transition as they grow older. We conducted an initial study on the neuropsychological functioning of a cohort of children who received both dialysis and transplantation under the age of 5 years.

Methods: 16 children were identified as potential study candidates. 5 children have completed cognitive testing so far using a well-validated neuropsychological measure of intellectual functioning (WISC-IV). The prevalence of neurocognitive deficits was determined through comparison of participant results with normative data.

Results: The mean Full Scale IQ (FSIQ) of this cohort falls two standard deviations below population norms (FSIQ 67, range 59-77), with a high prevalence of additional comorbidities in expressive and/or receptive language, and attention deficits. One child was unable to complete the formal assessment due to these comorbidities.

Conclusions: Initial findings suggest that children who have undergone dialysis and transplantation in early life are at increased risk of lower intellectual functioning, and comorbid language and attention deficits. This highlights the importance of close follow-up of this population and referral to early intervention services as indicated. Assessment of the remainder of this cohort is warranted.

Abstract No. 98**COGNITIVE AND ACADEMIC OUTCOMES IN CHILDREN WITH CHRONIC KIDNEY DISEASE AND KIDNEY TRANSPLANTS**

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Aims: Whilst chronic kidney disease (CKD) is known to reduce children's life expectancy and physical health, disease-related biological and psychosocial factors may also impair cognitive and academic development. We aimed to examine cognitive and academic functioning in children with pre-dialysis CKD and kidney transplants, and determine whether specific domains of cognition are differentially affected by CKD.

Methods: Tests of cognitive (IQ, attention, memory) and academic (literacy and mathematical) skills were administered to 16 children with CKD (pre-dialysis n = 9, transplant n = 7). Their scores were classified as average (≥ 90 scaled score, ≥ 8 subtest score) or below average, and the groups were compared using independent samples t-tests.

Results: The mean age was 11.4 (SD=1.9) years, and 11 (69%) were male. On average children missed 1.7 (SD=2.5) days of school per month. The mean full-scale IQ (95.2, SD=16.96), reading (99.8; SD=15.75), spelling (94.7; SD=15.14) and mathematical skills (90.3; SD=22.37) fell in the average range. Mean scores on verbal learning/memory (6.3, SD=3.21), sustained auditory attention (7.3, SD=3.62) and divided attention (6.9, SD=2.75) were below average. Across tests no differences were found between pre-dialysis and transplanted children (all $p > 0.1$).

Conclusions: Children with CKD appear to have average overall intelligence and academic skills, but may experience difficulties with attention and verbal learning. Such difficulties can negatively impact daily functioning, and in turn affect overall well-being. Further research should elucidate the nature of specific cognitive deficits in children with CKD to enable the development of targeted interventions that facilitate healthy cognitive development.

Abstract No. 99

INPATIENT REHABILITATION OF HEART AND LUNG TRANSPLANT PATIENTS A PHYSIOTHERAPY PERSPECTIVE- RETROSPECTIVE ANALYSIS 2011-2015 ST VINCENT'S HOSPITAL, SYDNEY

WOODBRIIDGE Genevieve

Rehabilitation Medicine- Physiotherapy, St Vincent's Hospital, Sydney

Aims: To do a retrospective analysis of physiotherapy outcomes of patients admitted for inpatient rehabilitation at Sacred Heart Rehabilitation, St Vincent's Hospital Sydney following heart, lung or heart and lung transplantation from 2011- 2015 and analysis of results and physiotherapy intervention.

Methods: A sample size of 67 patients were assessed by physiotherapy on admission and discharge to inpatient rehabilitation including functional assessments of transfers and mobility, manual muscle testing, respiratory assessment (RPE and BORG scales, oxygen saturation), Timed up and Go, 10m walk, 6 minute walk test and Berg Balance Scale.

Results: Typically they have significant deconditioning related to often long standing illness and significant activity limitation prior to surgery, multiple and complex admissions and post-operative recovery and potential complications. On admission many patients are very debilitated and dependent requiring assistance to stand, transfer and mobilise, some requiring a hoist to transfer. Steroid induced and critical care myopathy are further complications. Physiotherapy treatment focusses on increasing independence with functional tasks and mobility, improving muscle strength with graduated weight training, postural adaptations, altered patterns of breathing, improving static and dynamic balance and increasing cardiovascular fitness. This specialised patient population have extra problems to be considered including increased monitoring of vital signs during therapy, denervation of heart, immune-suppression and risk of organ rejection.

Outcome	Average on admission to inpatient rehab	Average on discharge from inpatient rehab	% improvement on initial score	% of patients unable to do the test on initial assessment as too disabled
6 minute walk test	60.3m	146m	142% or 85.7m further	30%
10m walk	29.9 secs (0.33m/sec)	17.4 secs (0.57 m/sec)	42% or 12.5 secs faster	28%
Timed Up and Go	28.4 secs	19.3 secs	32% or 9.1 secs faster	39%
Berg Balance Scale	22.2/56	42.1/56	90% or 19.9 point increase	N/A

Conclusion: Patients responded well to physiotherapy intervention with improving independence, mobility, strength, balance and endurance. While the improvements are significant they are still below normal ranges.

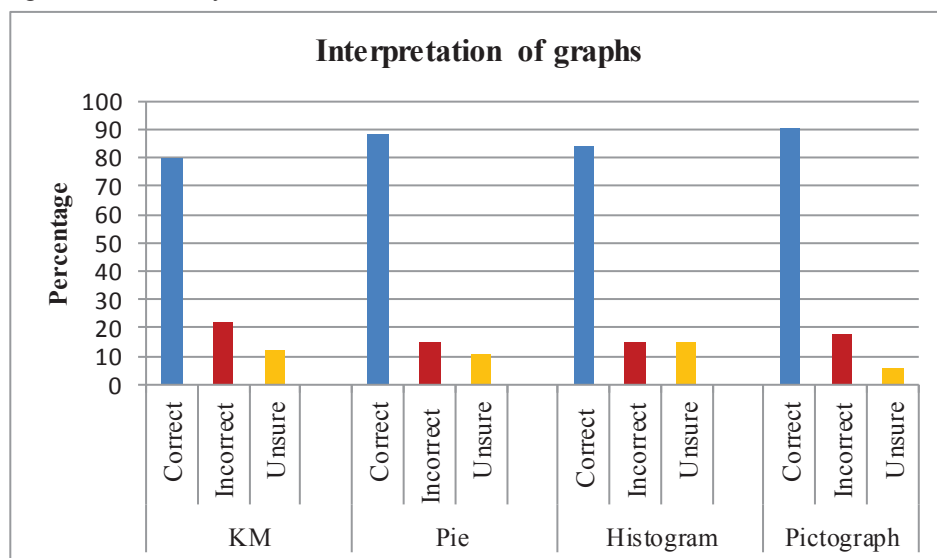
Abstract No. 100

USE OF VISUAL AIDS IN PROMOTING PATIENT UNDERSTANDING OF SURVIVAL WITH TREATMENTS FOR END STAGE KIDNEY DISEASE**DOWEN Frances¹, PILMORE Helen^{1,2}**¹*Department of Nephrology, Auckland City Hospital,* ²*Department of Medicine, University of Auckland*

Aims: Mortality in end stage kidney disease (ESKD) is higher than in many malignancies. Data shows that 97% patients want information about life expectancy and that 93% of nephrologists in Australia and New Zealand would find it helpful to have tools showing projected survival during assessment for dialysis and transplantation. There is no data about the optimal way to present this information to patients with ESKD. In other specialties, graphs have been shown to be more easily understood than narrative alone. We aimed to determine which visual method of communicating projected survival is preferable in patients with ESKD.

Methods: We asked 114 patients with chronic kidney disease to look at four graphs showing post transplantation survival data. Each patient was required to interpret a Kaplan Meier curve, pie chart, histogram and pictograph and answer a multi-choice question to determine understanding of the graphs.

Results: 86% patients found the graphs useful and the majority had no preference in the type of graph used. The pictograph yielded the most correct answers and pie chart the least incorrect interpretations. The Kaplan Meier curve received the fewest correct answers and greatest number of incorrect readings. The histogram met the greatest degree of uncertainty.



Conclusion: Visual representation of data is a useful tool in communicating projected survival to patients with ESKD. We plan to facilitate use of graphs in counselling patients with ESKD on treatment choices. This has the potential to shape our interactions, empower our patients, and further develop patient centred and directed care.

Abstract No. 101

THE INCIDENCE OF GRAFT PANCREATECTOMY SECONDARY TO THROMBOSIS IN SIMULTANEOUS PANCREAS AND KIDNEY TRANSPLANTATION: A SINGLE-CENTER EXPERIENCE**LENDZION Rebecca¹, SPIKE Erin¹, YUEN Lawrence¹, LAM Vincent¹, RYAN Brendan¹, PLEASS Henry^{1,2}, HAWTHORNE Wayne^{1,2,3}**¹*Department of Surgery, Westmead Hospital, Sydney,* ²*Discipline of Surgery, Sydney Medical School, University of Sydney,* ³*Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney*

Despite simultaneous pancreas and kidney (SPK) transplantation survival rates improving over recent years, pancreas transplant is still associated with significant morbidity. In particular, graft thrombosis rates are as high as 29% in the first six months.

Aims: Our aim was to evaluate rates of pancreas graft loss secondary to thrombosis and identify contributing factors.

Methods: A retrospective study of graft thrombosis was conducted on 141 SPK transplants at Westmead Hospital from January 2010 to September 2015. Patients experiencing graft loss secondary to thrombosis; confirmed on histopathology were evaluated. Early graft loss was defined as ≤ 14 days and delayed as ≥ 14 days. Prolonged cold ischemia time (CIT) and donor BMI >30 kg/m² have been reported as risk factors for graft failure.

Results: Overall thrombosis rate was 19.8% (28 of 141 patients), consistent with previous reports with thrombosis rates of 10–20%. The rate of allograft pancreatectomy secondary to thrombosis was 7%. Organs were retrieved from around Australia. The longest pancreas CIT was 15.6 hours; interstate retrieval was associated with longer CIT. Early graft loss secondary to thrombosis was associated with longer CIT, 12.3 hours compared to 9 hours in delayed graft loss. In general donor and recipient BMI were well matched, however in 40% of patient's donor BMI was significantly greater compared to the recipient warranting further investigation.

Conclusions: SPK provides a quality of life improvement providing normal glucose homeostasis and renal function. However, it remains an extremely technical procedure associated with high technical failure rates for the pancreas.

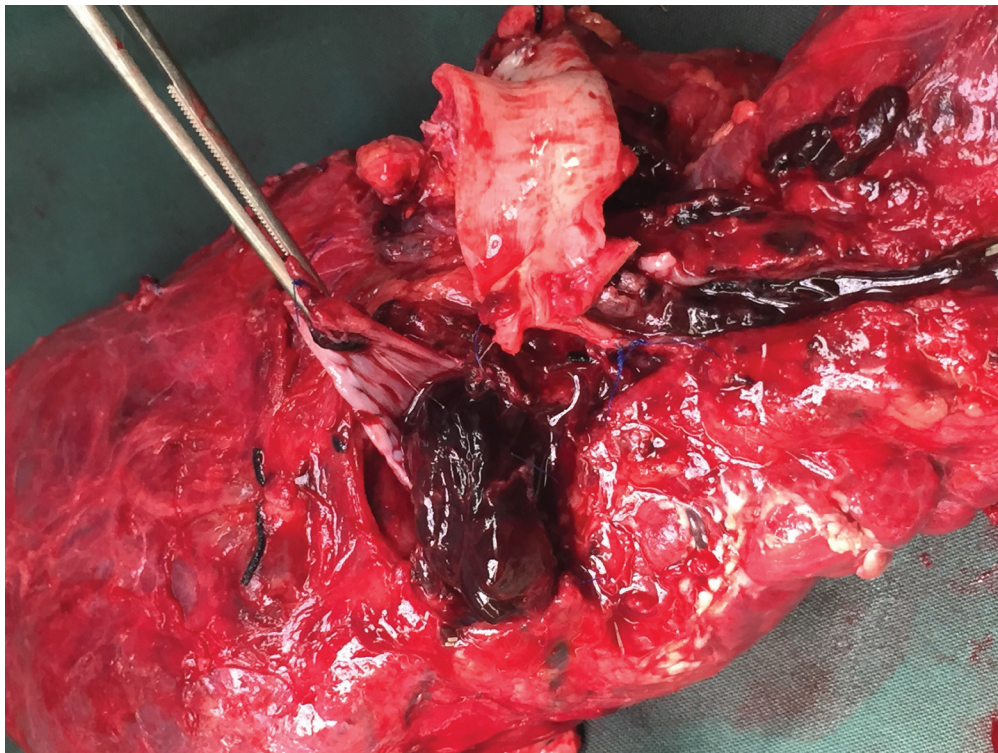


Image 1: Allograft pancreatectomy day one post-SPK transplant demonstrating thrombosis within the portal vein and splenic artery.

Immunosuppression and Trials & Surgical Techniques

Abstract No. 102

ESTIMATION OF MYCOPHENOLIC ACID EXPOSURE POST RENAL TRANSPLANTATION: COMPARISON BETWEEN THE TRAPEZOIDAL METHOD AND MULTIPLE REGRESSION DERIVED LIMITED SAMPLING STRATEGIES.

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¹School of Medicine, Faculty of Health Sciences, University of Melbourne, ²Department of Nephrology, Royal Melbourne Hospital

Aims: The trapezoidal method is typically used in clinical practice for estimating mycophenolic acid (MPA) area under the concentration-time curve from 0-12 hours post-dose (AUC₀₋₁₂). However, this method can be inaccurate when the AUC₀₋₄ profile is atypical. This study examined the performance of the trapezoidal method in estimating MPA AUC₀₋₁₂. Concordance between trapezoidal and multiple regression derived limited sampling strategy (LSS) AUC estimations was assessed.

Methods: MPA samples were collected pre-dose and at 1, 2 and 4 hours post-dose in 157 kidney transplant recipients receiving tacrolimus and prednisolone co-therapy. MPA AUC₀₋₁₂ was estimated using the linear trapezoidal rule and 7 previously published LSSs. AUC estimates were categorized into groups (<20, 20-40, 40-60, or >60 mg.h/L) and discordance in categorization between the two methods was assessed.

Results: The trapezoidal method was unable to estimate AUC₀₋₁₂ in 26/157 (18%) due to a late or secondary C_{max} (n=19) or high pre-dose concentrations (n=7). Table 1 shows the absolute differences between trapezoidal and LSS estimates and the proportion of cases where the two methods resulted in different categorisation. AUC₀₋₁₂ varied up to 4.8-fold depending on the LSS equation.

Conclusions: The trapezoidal method was unable to estimate AUC in a substantial number. Because wide differences exist between trapezoidal and LSS estimates in some patients, the two methods cannot be considered equivalent and shouldn't be routinely substituted for each other in clinical practice. AUC estimates also vary widely depending on the LSS. To ensure accuracy, LSSs should be validated in the population of interest prior to use.

Table 1: Absolute difference and proportion of cases in which categorisation differed between trapezoidal and LSS-derived AUC estimates.

	LSS Equation 1	LSS Equation 2	LSS Equation 3	LSS Equation 4	LSS Equation 5	LSS Equation 6	LSS Equation 7
Minimum	0.10 mg.h/L	0.007 mg.h/L	0.02 mg.h/L	0.08 mg.h/L	0.05 mg.h/L	0.004 mg.h/L	0.08 mg.h/L
Q1	2.2 mg.h/L	3.1 mg.h/L	2.6 mg.h/L	3.2 mg.h/L	2.9 mg.h/L	2.9 mg.h/L	1.5 mg.h/L
Median	3.9 mg.h/L	5.9 mg.h/L	5.4 mg.h/L	6.5 mg.h/L	6.0 mg.h/L	6.8 mg.h/L	4.2 mg.h/L
Q3	8.8 mg.h/L	10.4 mg.h/L	7.3 mg.h/L	10.0 mg.h/L	11.3 mg.h/L	10.5 mg.h/L	8.5 mg.h/L
Maximum	40.2 mg.h/L	37.8 mg.h/L	20.3 mg.h/L	43.8 mg.h/L	37.8 mg.h/L	43.0 mg.h/L	35.5 mg.h/L
Discrepant Categorisation	25%	37%	29%	37%	37%	37%	22%

Abstract No. 103**EVALUATION OF PREVIOUSLY PUBLISHED LIMITED SAMPLING STRATEGIES FOR ENTERIC-COATED MYCOPHENOLATE SODIUM IN ADULT KIDNEY TRANSPLANT RECIPIENTS****BROOKS Emily¹, TETT SUSAN², ISBEL Nicole³, STAATZ Christine⁴**¹*School of Medicine, University of Queensland, Brisbane,* ²*School of Pharmacy, University of Queensland, Brisbane,* ³*Department of Nephrology, Princess Alexandra Hospital, Brisbane,* ⁴*School of Pharmacy, University of Queensland at the Princess Alexandra Hospital*

Aims: The aim was to evaluate the predictive performance of published limited sampling strategies (LSS) for estimation of mycophenolic acid (MPA) exposure (area under the concentration time curve, AUC) following enteric-coated mycophenolate sodium (EC-MS) in adult renal transplant recipients.

Methods: MPA concentrations were measured in 20 recipients (one-month post transplant, receiving EC-MS twice daily). Samples were taken at 0, 0.33, 0.5, 1, 1.5, 2.0, 2.5, 3.0, 3.5, 4, 8 hours and 0, 0.25, 0.5, 0.75, 1.0, 1.25, 1.5, 2.0, 3.0, 4.0, 6.0, 9.0 and 12.0 hours post-dose on two sampling occasions a week apart. MPA plasma concentrations were determined using HPLC-MS-MS. Predicted MPA AUC were calculated using fourteen different LSS (5 reported studies) with data from the first sampling occasion for each patient, and compared to the second occasion full MPA AUC calculated from all measured concentration-time points using the linear trapezoidal rule. Bias (median prediction error [MPE]) and precision (root mean squared error [RMSE]) were calculated.

Results: Bias and precision (as % of median) for prediction of a future MPA AUC were <15% for seven equations. Two equations using concentrations at 1, 3 and 9 hours and 1.5, 2.5, 3.5 and 6 hours post dose were superior to other models tested. Two equations using concentrations at 0.5, 1 and 3 hours and 1, 2, 2.5 and 4 hours post dose showed acceptable bias and precision.

Conclusions: Several LSS, including two used to develop equations with concentrations taken within four hours post-dose, predicted future MPA AUC after EC-MS with reasonable bias and precision.

Abstract No. 104**RETROPERITONEOSCOPIC LIVE DONOR NEPHRECTOMY****NG Zi Qin, HE Bulang***WA Liver & Kidney Transplant Service, Sir Charles Gairdner Hospital, Perth*

Aim: The aim of this study is to evaluate the outcomes of retroperitoneoscopic donor nephrectomy (RDN) in comparison with laparoscopic intraperitoneal approach (LIA).

Materials and Methods: From January 2010 to November 2015, 90 live donor nephrectomies were performed in our institute. Mean age was 50.98 years (26 – 75 years). Of 90, 34 were done by RDN whereas 56 were done by LIA. Operative time, graft warm ischaemic time, intraoperative and postoperative complications were recorded and analyzed. All forms of postoperative analgesia were converted to oral morphine equivalent for comparison. Kidney graft function was followed-up at 1 week, 3 months and 1 year post-surgery. Mean recipient age was 41.20 years.

Results: All donor nephrectomies were successfully performed with no conversions. There was no difference in operative time in both groups. No intraoperative complications were observed in both groups. No patients received blood transfusions. Mean graft warm ischaemic time was 4.34 minutes. There were 3 cases of chyle leak in RDN group and were managed conservatively. There was no significant difference in analgesia use in both groups. All kidneys were transplanted successfully and followed-up from 1 to 59 months. There was no delayed graft function and urological complications. Mean creatinine level at 3 months was 123.15.

Conclusion: RDN is an attractive alternative approach with comparable kidney graft function to LIA. This approach maintains the virgin abdomen for the donor and lowers the risk of intraperitoneal organ injury. The analgesic consumption in both groups was equivalent.

Abstract No. 105**RECOVERY OF KIDNEY GRAFT FUNCTION AFTER TRANSPLANT BY LAPAROSCOPIC TECHNIQUE VS OPEN: A PILOT STUDY****HE Bulang¹, MUSK Gabrielle², MOU Lingjun¹, NG Ziqing³, DE ROO Ronald¹, TAN Daren¹, DARIAN-SMITH Erica¹, LUCAS Michaela⁴, DELRIVIERE Luc¹****¹WA Liver & Kidney Transplant Service, Sir Charles Gairdner Hospital, Perth, ²Animal care service, University of Western Australia, Perth, ³Royal Perth Hospital, ⁴Sir Charles Gairdner Hospital, Perth**

Aim: The aim of this study is to investigate the ischemic reperfusion injury and its effect on the recovery of kidney graft function in the context of laparoscopic kidney transplant.

Materials and Methods: Five domestic pigs were subject to this pilot study with 3 pigs in laparoscopic group (LG) and 2 pigs in open group (OG). The orthotopic left auto transplantation was performed by purely laparoscopic technique in LG whereas it was done by open surgery in OG. The vessel anastomotic time and the temperature change in the kidney graft were recorded respectively. The kidney biopsy was taken at the time of pre implant, post implant and 4 weeks after transplantation. Doppler US was performed immediately after completion of surgery. The blood sample was also taken at various time points for serum creatinine (Cr) level test.

Results: All 5 pigs recovered from surgery and were observed for 4 weeks. The arterial anastomotic time is longer in LG than in OG while the venous anastomotic time is similar in both groups. The kidney graft perfusion was satisfactory on immediate post-op Doppler ultrasound. The peak Cr level was on day 1 and return to nadir on day 7. The Cr level was higher in OG at day 1 and day 3 than those in LG. The tissue biopsy is still under review at the time of submission.

Conclusion: It was recognized that the kidney graft function is better after laparoscopic kidney transplant in comparison with open kidney transplant in this pilot study.

Abstract No. 106**BARRIERS TO EARLY TRANSPLANTATION LISTING FOR PATIENTS UNDER 65****BROWN Megan, POLKINGHORNE Kevan, MANEY Orla, KANELIS John*****Department of Nephrology, Monash Medical Centre, Melbourne***

Aims: To identify the patient and systemic barriers to transplantation and transplantation waitlisting within six months of starting renal replacement therapy (RRT); this is a key performance indicator (KPI) in Victoria.

Methods: Patients under 65 years old starting RRT for the first time through Monash Health over a three year period (January 2012 to December 2014) were retrospectively analysed. Patients were analysed in two groups according to whether they met the KPI or not. Data was collected on demographics, dialysis factors, comorbidities, social and systemic factors. For those who did not meet the KPI the main barrier to early listing was categorised.

Results: 183 patients were included; 62 met the KPI and 121 did not. 27 (43.5%) of those meeting the KPI received a transplant within 6 months, with 21 occurring pre-emptively. The groups differed significantly with regard to cause of renal failure and comorbidities (see table 1). Diabetes, peripheral vascular disease, cardiovascular disease, obesity and nonadherence were more common in those not meeting the KPI ($p \leq 0.001$). In contrast polycystic kidney disease and glomerulonephritis were more common in those meeting the KPI ($p < 0.001$). Most delays in listing were multifactorial, however 27 (22%) of the patients not meeting the KPI were limited by systemic factors alone.

Conclusion: Multiple factors make early transplantation and listing difficult. Correcting systemic factors alone could increase the percentage of patients listed or transplanted within 6 months of commencing RRT to 48.6%. This will require targeted improvement in both referral practices and workup processes.

Abstract No. 106 (continued)

Table 1: Comparison of major factors between those who met the KPI and those who did not

	Listed within 6 months (N=63)		Not listed within 6 months (N=121)		P value
	Total no.	%	Total no.	%	P=
Patient Demographics					
Age at RRT	47	NA	42.8	NA	0.018
Gender (female)	22	35.4%	38	31.4%	0.578
Rural residence	11	17.7%	22	18.0%	0.769
Cause of ESRF					
Glomerulonephritis	23	37.1%	25	20.7%	<0.001
Diabetes	6	9.6%	56	46.2%	
Hypertension/Renovascular	3	4.8%	9	7.4%	
Polycystic kidney disease	12	19.4%	6	5.0%	
Other	19	29.0%	25	20.7%	
Dialysis factors					
Nephrologist review at least 3 months pre RRT	59	95.2%	97	80.2%	0.007
Education pre RRT	60	96.8%	85	70.2%	<0.001
Initial Access					
Fistula	23	37.1%	41	22.8%	<0.001
Tenckhoff	17	27.4%	19	15.7%	
Central Vein Access (Permcath or Vascath)	2	3.2%	61	50.4%	
Transplant (Pre-emptive)	20	32.3%	0	0%	
Type of RRT at 6 months					
Satellite Haemodialysis	23	37.1%	89	73.6%	<0.001
Home Haemodialysis	6	9.6%	6	5.0%	
Peritoneal Dialysis	16	25.8%	24	19.8%	
Transplant	27	43.5%	0	0%	
Other	0	0%	0	0%	
Comorbidities					
Diabetes	12	19.4%	64	52.5%	<0.001
Cardiovascular	11	17.7%	54	44.3%	<0.001
Obesity	10	16.1%	55	45.5%	<0.001
Airways disease	12	19.4%	20	16.4%	0.634
Hypertension	56	90.3%	111	91.0%	0.749
PVD	2	3.2%	26	21.3%	0.001
Infection-related risk	6	9.7%	24	19.7%	0.079
Cancer Risk	9	14.5%	17	13.9%	0.932
Psychiatric illness	10	16.1%	33	27.0%	0.092
Social Factors					
Current smoker	4	6.5%	15	12.4%	0.220
Past smoker	17	27.4%	41	33.9%	
Never Smoked	41	66.1%	65	53.7%	
Non-English Speaking Background	4	6.5%	16	13.2%	0.165
Non-Adherence	3	4.8%	47	38.8%	<0.001
Systemic issues					
Referred for transplant assessment before RRT	60	96.8%	50	40.9%	<0.001

Abstract No. 107**A QUALITATIVE REVIEW OF MEDICATION ERRORS MADE BY NEW KIDNEY TRANSPLANT RECIPIENTS****FISHER Susan¹, NALDER Michelle¹, HUGHES Peter², HOLT Steve²**¹*Department of Pharmacy, Royal Melbourne Hospital,* ²*Department of Nephrology, Royal Melbourne Hospital*

Background: After kidney transplantation, patients are required to follow a complex and frequently changing medication regimen. Non-adherence to the prescribed schedule is associated with increased rates of rejection and graft loss, but detecting patients who make unintentional errors or who are intentionally non-adherent can be difficult. To assist with improving medication management in this group, the Renal Transplant Outpatient Pharmacist (RTOP) role was established.

Aims: To identify the rate and types of medication administration errors made by patients after renal transplantation; to explore reasons for these errors and to develop strategies to prevent further deviation from the prescribed regimen.

Methods: The RTOP reviews all new renal transplant patients in clinic after hospital discharge, providing medication education and early identification of medication errors. Medication administration errors were recorded in the nephrology patient database (Nephworks). Records were reviewed retrospectively for the first 50 transplant patients since the RTOP role was established to identify and characterise those errors.

Results: The RTOP identified numerous dangerous mistakes including: confusion over medication strengths leading to under and overdosing; tablets halved inappropriately; incorrectly packed dose administration aids; incorrect administration times and failure to make prescribed dosage changes.

Conclusions: Medication errors were common in the early post-transplant period. The majority of errors identified at this early stage were unintentional and related to poor medication knowledge, the complexity of the medication regimen and misunderstandings about changes.

Abstract No. 108**SIROLIMUS PRECIPITATES INCISIONAL HERNIA IN TRANSPLANT PATIENTS****SHAHRESTANI Sara¹, TRAN Hanh¹, PLEASS Henry^{2,1}, HAWTHORNE Wayne^{2,1,3}**¹*Discipline of Surgery, Sydney Medical School, University of Sydney,* ²*Department of Surgery, Westmead Hospital, Sydney,* ³*Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney*

Aims: To examine the rate of hernia in abdominal transplant patients, and investigate immunosuppression effect.

Methods: Systematic review was utilized to investigate the rate of hernia in abdominal organ transplantation and compare rates between different organ transplants. We moderated by immunosuppression to investigate whether this played a role in moderating risk for hernia.

Results: 72 relevant articles were identified for systematic review and 51 individual studies contained statistics necessary for meta-analysis. This included 31,203 abdominal transplant recipients consisting of 18,686 kidney transplant recipients, 11,957 liver transplant recipients, 171 pancreas transplant recipients and 208 intestinal transplant recipients. In the remaining 181 patients, the organ transplanted was unclear or multi-organ transplantation occurred. Hernia in abdominal transplants occurred at an overall event rate of 0.06 (n=56, CI 95% 0.05-0.09, p<0.001). These effects were moderated by use of sirolimus with sirolimus associated with around a three times higher rate of hernia (n=10, ER= 0.18, CI 95% 0.10-0.29, p<0.001) compared with no sirolimus (n=24, ER= 0.06, CI 95% 0.04-0.10, p<0.001). The rate of hernia was highest in liver transplants (n=29, ER= 0.10, CI 95% 0.07-0.14, p<0.001) followed by intestinal transplant (n=3, ER= 0.07, CI 95% 0.02-0.27, p=0.001), kidney transplants (n=22, ER= 0.03, CI 95% 0.02-0.06, p<0.001) and pancreas transplants (n=3, ER= 0.02, CI 95% 0.01-0.06, p<0.001).

Conclusions: The rate of hernia is low in transplant patients and varies by the type of organ transplanted, the rate being highest in liver recipients and lowest in pancreas transplants. Sirolimus greatly increases the rate of hernia formation.

Abstract No. 109

RESTORED KIDNEY TRANSPLANTATION - EXTENDED CRITERIA DONORS

SPROTT PHILIP¹, TREVILLIAN PAUL¹, HIBBERD ADRIAN¹, HEER MUNISH¹, STEIN ANN², DAVIDSON DEIDRE¹

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Objective: Many patients have a total nephrectomy to treat localised cancer, discarding a significant volume of normal kidney. Restored renal donation involves excising focal pathology after nephrectomy and transplanting the residual kidney. Existing reports describe excising small renal lesions, and the increasing popularity of partial nephrectomy has almost eliminated this donor source. We questioned the validity of only using kidneys with small cancers, and report our experience transplanting kidneys with larger renal cancers.

Patients and Methods: We used outcomes from partial nephrectomy to estimate cancer transplantation and complication rates for renal cancers of all sizes. We reviewed the cancers we were treating with total nephrectomy, and estimated the functional tissue in these kidneys. We used this information to obtain Clinical Governance and NSW State Transplant Advisory Committee approval to transplant restored kidneys after excising cancers up to 5 cm. Only older high risk recipients were eligible. All data and outcomes were recorded prospectively.

Results: 26 patients were referred for evaluation, and 20 proceeded to donation. Data is being collected continuously, including patient and graft survival, and operative complications. Urinary fistulae and urinary tract infection are the commonest complications. There is no 90 day mortality in recipients or donors. There has been one cancer recurrence attributable to transplantation with the kidney.

Conclusion: Renal cell carcinomas up to 5cm in size can be safely excised ex vivo, leaving adequate nephron mass to provide a viable transplant. Low complication rates and high graft function rates justify persisting with this technique.

Abstract No. 110

COMBINATION OF LEFLUNOMIDE AND EVEROLIMUS FOR TREATMENT OF BK VIRUS NEPHROPATHY**JAW Juli¹, GOODMAN David¹, HILL Prue²**¹*Nephrology and Renal Transplant, St Vincent's Hospital, Melbourne,* ²*Department of Anatomical Pathology, St Vincent's Hospital, Melbourne*

BK nephropathy (BKN) is one of leading causes of graft loss following renal transplantation, possibly due to newer immunosuppressive therapy. Currently minimization of immunosuppressive regimen is the first line of therapy but this needs to be balanced with the increased risk of rejection. In some cases, despite lowering immunosuppression, BK infection can persist and leads to graft loss. Recent *in-vitro* experiments demonstrate reduction in BK viral replication when infected cells are treated with the combination of Leflunomide and Everolimus.

Aim: This study aims to explore the effect of this drugs combination on viral clearance and graft function in patients with persistent disease despite reduction in immunosuppression.

Methods: We treated 3 patients with combination of Leflunomide and Everolimus. Data on medical history, biochemical parameters and viral loads were collected (Table 1).

Results: Significant improvement in viral loads was observed in 2 cases with resolution of viraemia in another (Table 1). Two recipients had preservation of allograft function. The remaining graft was lost due to combination of obstruction and BKN. No adverse reaction such as bone marrow toxicity was observed. One patient has elevated ALP level which correlates to moderate renal impairment.

Conclusion: Combination of Leflunomide and Everolimus is safe and should be considered as a rescue therapy in treatment of BKN, especially in those who fail to clear this infection despite reduction of immunosuppressive therapy.

Patient	1	2	3
Gender	F	M	M
Age (years)	56	55	67
Type of transplantation	Deceased donor	Kidney-Pancreas	Deceased donor
No of transplantation	2nd graft	1st graft	1st graft
Initial immunosuppression	Pred, MMF, Tac	Pred, MMF, Tac	Pred, MMF, Tac
Time to diagnosis (months post transplant)	4	48	5
Peak BK titre (copies/mL)	40 million	6 million	9.3 million
Histological classification on biopsy	Stage B3	Stage B3	Stage B3
Baseline Creatinine (µmol/L)	110 -120	80-100	170
Peak Creatinine (µmol/L)	300	150	ESRF
Current Creatinine (µmol/L)	180	130-140	ESRF on dialysis
Years since transplantation	2	5	2
Current BK titre (copies/mL)	undetectable	271,000	<25,000

Table 1 - Baseline characteristic and result of patients on combination of Leflunomide and Everolimus

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The
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ABN 90 796 930 798

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Karen Dwyer
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NOTICE OF ANNUAL GENERAL MEETING

The Annual General Meeting of the Transplantation Society of Australia and New Zealand will be held on Sunday 10th April 2016 in the Harris room of Aerial UTS Function Centre, Sydney at 5.45pm.

AGENDA

1. Apologies
2. Confirmation of the minutes of the Annual General Meeting held on 21st June 2015
3. Business arising from the minutes
4. President's Report - Professor Steve Chadban
5. Treasurer's Report – Dr Robert Carroll
6. Secretary's Report – Dr Karen Dwyer
7. Report on Advisory Committees/Working Groups
- Professor Steve Alexander
8. Scientific Program & Education Committee Report (SPEC)
- A/Professor Daniel Chambers
9. Liaison with Scientific Societies
- Dr Shane Grey
10. General Business

Professor Karen Dwyer
Honorary Secretary
March 2016



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The Annual General Meeting of the Transplantation Society of Australia and New Zealand held on Sunday 21st June 2015 in the Manning Clark Centre, ANU Campus, Canberra, ACT at 5.45pm.

Present: 52 members of the Society were present at the meeting which was chaired by the President, Professor Geoff McCaughan, who welcomed everyone to the meeting.

Also in attendance: Ms Sommer Twycross, TSANZ Administrative Officer.

1. APOLOGIES

Apologies were received from Ian McKenzie, Napier Thomas & Keryn Williams.

2. CONFIRMATION OF THE MINUTES

The minutes of the Annual General Meeting held on 11th June 2014 were passed as a true record.

Moved: Helen Pilmore
Seconded: John Kanellis

3. BUSINESS ARISING FROM THE MINUTES

Any business arising from the minutes would be covered elsewhere.

4. PRESIDENT'S REPORT

The President, Professor Geoff McCaughan, referred to his written report on pages 136-137 of the Program and Abstract Book which reviewed activities and events over the past 12 months. Professor McCaughan started off by thanking all members of council for their hard work over the past 12 months.

Prof. McCaughan provided the membership with a brief update on the health of Ms Aviva Rosenfeld who has been on sick leave from the Society since February 2015. Currently Aviva is remaining significantly unwell and is sadly missed. The membership will be updated on the situation as soon as the TSANZ council has been given further details.

Next Prof. McCaughan mentioned an item that has arisen recently with regards to the government review of the Organ & Tissue Authority by an independent review body – Ernst & Young. A number of TSANZ Council members (Steve Chadban, Steve

Alexander, Greg Snell & Henry Pleass) will be attending a meeting with the team from Ernest & Young during this ASM to discuss the review and to provide whatever information is required of TSANZ. So that TSANZ council can express the thoughts of the Society membership during the upcoming meeting with Ernst & Young Prof. McCaughan asked for the membership to provide ideas on what they feel the TSANZ council should discuss? A number of comments and ideas were given by various attending members and Prof. McCaughan said the comments would be taken into account.

Prof. McCaughan thanked the membership for their attendance at this year's ASM.

5. TREASURER'S REPORT

The Treasurer, Hilton Gock, presented his last Treasurer's Report on behalf of the Society and thanked all fellow council members for the opportunity to work together as council members over the past 4 years.

Hilton Gock referred the membership to the Financial Report of 31st December 2014 which appears on the TSANZ website. Currently the Society is in a sound financial position with retained profits of approximately \$1.3m.

Hilton Gock proposed the following motion:-

Hilton Gock proposed that the 2014 Financial Report be accepted:

Moved: David Goodman

Seconded: Josette Eris

Passed unanimously.

Next Hilton Gock spoke about the gradual & sustained reduction in sponsorship of the Society and the increase in running costs including quite a significant increase in joint membership fees received from TTS as of 2016. Therefore the subscriptions and ASM registration pricing should be based corresponding to the expenses for long term viability of the Society. The TSANZ council discussed increases in some ASM registration fees and subscriptions, details of these increases will be presented by John Kanellis in the Secretary's Report.

Next Hilton Gock discussed with the membership the financial viability of holding the ASM at a different location and or state other than Canberra, ACT. Having an alternative ASM venues has been raised by SPEC and some members since the 2014 ASM however the views of the broader TSANZ membership is unknown. Some reasons for and against moving out of Canberra have been debated for years so Hilton Gock sought to test the financial viability by asking for a quote at a suitable Gold Coast Venue. After considering the financials it does seem that the cost to hold the ASM in an alternative venue is financially viable. The following comments by members were noted:- some sponsors may not be able to support the meeting if held at a venue that is considered to be a 'resort' due to change in policies, the number of delegates has been dropping over the years and perhaps a change in venue may revive the Society and more attend, if change in location of the meeting would a conference organizer be required and can the Society afford this? Could the Society consider linking up with other meeting such as ANZSN to increase numbers? All comments were

noted by TSANZ council. Geoff McCaughan said that council have not made a definite decision and this will be left up to the incoming new TSANZ council members to discuss and decide upon in the coming years. In summary the Society is in a relatively secure financial position for now despite gradual & sustained reduction in sponsorship. The TSANZ subscriptions and ASM registration pricing should be based corresponding expenses for long term viability. The Society should continue to look at preserving pricing scaling to encourage & support younger members. And finally aside from the arguments 'for' and 'against' an ASM venue change, it is financially viable.

6. SECRETARY'S REPORT

John Kanellis, the Honorary Secretary, welcomed the new members of TSANZ Council: Dr Nick Cross, Dr Rob Carroll, A/Prof Nick Shackel & Dr Steve Alexander. On behalf of the TSANZ Council John Kanellis thanked the out-going TSANZ President Prof. Geoff McCaughan for his focus and innovation during his term on council over the past 4 years.

John Kanellis confirmed the number of delegates attending the 2015 ASM was approx. 230, 101 abstracts to be presented and approx. 80 delegates attended a very successful Post Graduate Course. The awards to be presented during the ASM will be: 2 x President's Prizes, Ian McKenzie Prize, Mark Cocks Scholarship, 20 x Young Investigator Awards & 2 x KHA awards. There are currently 466 voting members of the Society with sub total of 601 members of the Society.

John Kanellis next discussed the membership fee structure of the Society. The TSANZ Council discussed during the 20th June council meeting the need to increase the subscription fees due to the recent increase in TTS joint membership fees, the fee increase will not benefit the Society financially but will just cover the fee increase from TTS. Therefore council reviewed the current membership fees taking into account the TTS increases and decided upon the below membership fees to take effect as of 1st January 2016 (fees incl. GST where applicable):-

Full Member = \$385
Full Member NZ = \$350
International Member = \$125
Student (with TTS) = \$137.50
Retired (with TTS) = \$137.50
Retired (without TTS) = \$55
Student (without TTS) = \$55
Joint ATCA/TSANZ Member = \$55
Joint TNA/TSANZ Member = \$55

Various members expressed their support of the suggested fee increases.

John Kanellis ended his report by thanking all for attending.

7. REPORT ON ADVISORY COMMITTEES/WORKING GROUPS

Steve Chadban, chair of the Advisory Committees & Working Groups gave a brief report to the membership. The Advisory Committees and Working Groups have been working extremely hard to complete the Clinical Guidelines by the final edits deadline of 30th July 2015. Sarah White has also been working along with the AC & WG's towards a final collated draft of the guidelines which she will edit and send as a final draft to TSANZ & OTA for consultation by 31st July 2015.

8. SCIENTIFIC PROGRAM & EDUCATION COMMITTEE REPORT (SPEC)

Karen Dwyer, Chair of SPEC, said that members of SPEC have been working really well together and have produced a really great PGC & ASM program for 2015.

9. LIAISON WITH SCIENTIFIC SOCIETIES

Shane Grey have a brief update on the upcoming 2015 TSS/BSS Lorne meeting. The program is finalised with 18 international speakers, TSANZ have provided \$30K USD sponsorship plus \$20K AUD for members to attend by way of receiving a mentee/mentor award or a TSANZ travel grant. The Society has a strong interest in education which is reflected in the travel grants provided to members to attend this meeting.

10. GENERAL BUSINESS

The President thanked all for attending the AGM and concluded the meeting

There being no further business the meeting closed at 6.30pm.

Steve Chadban
President

Karen Dwyer
Honorary Secretary



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THE PRESIDENT'S REPORT

Dear Membership,

Welcome to Sydney! For the first time in the memories of most TSANZ members, our ASM and AGM will be in Sydney for 2016. The move has been made in response to numerous requests to move from Canberra, and only after robust discussion with members and within Council. We will miss the familiarity of Canberra, but are confident the move will add interest, bring greater numbers of registrants and rejuvenate our meeting. We hope you find the venue at UTS, the scientific quality of our Post-Grad Course and Annual Scientific Meeting, and the standard of our social functions to be as outstanding as they look to be on paper. Council and I would like to sincerely thank our organisers Min Hu and Andrew Jabbour (ASM), Lucy Sullivan and Darren Lee (PGC), and Sommer Twycross (all things administrative and logistical) for the hard work, vision and commitment that has gone into organising our meeting. We sincerely hope you enjoy the proceeds of their work.

As a new initiative, we are also conducting two parallel meetings the day after our ASM concludes: (1) TSANZ Transplant Update for Clinicians – aimed to up-skill practicing clinicians in maintenance phase management of transplant recipients; and (2) TSANZ/OTA Donation and Transplantation Forum – designed as a melting pot for discussion of key issues in organ donation for transplantation between transplanters and donation sector experts, co-sponsored by TSANZ and OTA. We aim to promote a spirit of collaboration, advance the science and practice of transplant medicine, and share the perspectives of transplanters, donation sector experts and funders in striving to improve both rates of organ donation and outcomes of transplantation for our patients.

What do we stand for as a Society? Members see us as the peak representative body for transplant professionals and have, I think, a good grasp of our beliefs, values and aspirations. However, this may be less apparent to those outside our Society membership. Council has drafted a Mission Statement that has been circulated to the membership. We hope to receive feedback and ultimately endorsement of this important statement with the aim of placing it upon our website for public access.

Donation of organs for transplantation and how to maximise the transplant outcomes resulting from such donations has remained a key priority for TSANZ. Our interactions with the Organ and Tissue Authority, including regular meetings between the Transplant Liaison Reference Group and OTA, co-operation in the preparation of various Guideline Documents, and collaboration in the planning and conduct of the TSANZ/OTA Donation and Transplantation Forum, have been positive and productive. The performance of OTA in promoting and managing organ donation from deceased donors for transplantation was formally audited by Ernst & Young, commissioned by the responsible Federal Minister, in 2015. TSANZ, via a delegation from Council, provided the auditors with our opinions and perspectives on the performance of OTA, our assessment of

of the current status of organ donation in Australia, and our suggestions for areas to improve. Many of our recommendations and opinions are reflected in the formal report of this Audit, released by the Minister in early 2016. Whilst the audit acknowledged many of the gains made in

this sector and strongly endorsed the current strategies and plans of the OTA, key additional recommendations were also made and these will likely form a blueprint for ongoing efforts to improve performance. As the TSANZ membership are likely aware, the CEO of the OTA, Ms Yael Cass, was replaced by Ms Felicity O'Neill as Acting CEO in early 2016. Council would like to pay tribute to the commitment, skill and vision displayed by Ms Cass in her leadership of the OTA, and compliment her not only on the profound gains made in organ donation rates under her stewardship, but also on her leadership and professionalism. Although Ms O'Neill has very large shoes to fill, her impressive track record coupled with rapid acquisition of knowledge of our sector bode very well for our future collaborations.

TSANZ has been active in re-writing our Consensus Documents, now titled the TSANZ Clinical Guidelines, and in collaborating with the NHMRC and OTA in producing the NHMRC Ethical Guidelines on Transplantation of Organs from Deceased Donors. Both documents are to be released at this meeting. Their importance should not be underestimated, as both articulate the ethical and practical framework within which we conduct transplantation in Australia and New Zealand. I would personally like to thank the many TSANZ members who contributed by writing or reviewing this work, and in particular the Chairs of the Advisory Committees who organised and edited their respective sections. For the expertise, dedication, management and production skills of our Project Manager Sarah White, ably supported by Project Officer Iman Ali and by Eva Mehakovic and Shari Jensen from OTA, we are truly grateful. We hope you find these documents to be useful and insightful. Given the rate at which the field of transplantation evolves, we aim to provide regular updates to the Clinical Guidelines, which already contain a list of "areas in evolution to be addressed in subsequent editions".

TTS/TSANZ Lorne Basic Science meeting. TSANZ were delighted to join with The Transplantation Society to part sponsor this basic science meeting which was truly at the cutting edge of transplant immunology. Stephen Alexander and Shane Grey from TSANZ were instrumental in securing this meeting on Australian soil and, together with Stefan Tullius, Anita Chong and Phil O'Connell from TTS, they put up a feast of basic science. It is rare to be able to immerse in such consistently high quality presentations. Participant interaction and satisfaction levels were high. Many TSANZ young investigators were able to attend, mingle, learn and be inspired by the 40 invited speakers. The meeting attracted 150 registrants from Asia, Europe, North America and the Middle East in addition to ANZ, and sponsorship from TSANZ, TTS, OTA, societies and pharma, thereby making a small profit. This was, all in all, a fine example of what we seek to achieve in the promotion of research and fellowship - congratulations to Stephen, Shane, Phil and all concerned!

Finally, I would like to acknowledge the Council and our administrative team. It is always a pleasure to work with energetic, positive, clear thinkers and doers and our Council is a fine example of that. Cohesive, constructive and resourceful into the bargain! In particular I would like to thank Stephen Alexander as President-Elect for his sound advice, happily provided at zero notice; Karen Dwyer for her insights, initiative and energy as Secretary; and Rob Carroll as Treasurer for steering the finances into safe

waters. In the current climate of financial contractions and low interest rates, it has been challenging to maintain our revenue:expenditure balance. Rob will present our status, including some savings made through various initiatives, and also some new proposals for research prizes. No society can function without administration. Whilst we continue to lament Aviva Rosenfeld's retirement in 2015 through ill health, we are grateful to Sommer Twycross for rising to the challenge and facilitating all that we do with great skill and efficiency, recently and ably assisted by Nieves Piaggio.

A handwritten signature in blue ink, appearing to read 'Steve Chadban', on a light blue background.

Steve Chadban
President TSANZ

TSANZ Membership List

AUSTRALIAN CAPITAL TERRITORY

*BROWN, E
CARNEY, G M
CHOONG, F J
*CUNNINGHAM, J
FALK, M C
HURST, H S
*NORTHAM, H L
PENG, A
*RUSSELL, T
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