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The Transplantation Society of Australia and New Zealand

Thirty-sixth Annual Scientific Meeting

PROGRAM AT A GLANCE

Sunday, 29 April 2018		
09:00–10:00	Donor Surgeons and Donor Coordinators (DSDC)	Room 101, Level 1, Melbourne Convention and Exhibition Centre (MCEC)
12:00–13:00	Renal Transplant Surgeons Sub-Committee (RTSS)	Room 102, Level 1, MCEC
13:00–14:00	Pancreas & Islet Advisory Committee Meeting (PITAC)	Room 101, Level 1, MCEC
13:00–14:30	Paediatric Donor Working Group (PDWG)	Room 102, Level 1, MCEC
14:00–15:00	Registration	Foyer, Level 1 (MCEC)
15:00–15:10	Official Opening: TSANZ President	Main Plenary – Rooms 106-105 Level 1, MCEC
15:10–15:40	PLENARY 1: Astellas Symposium Desensitisation of the Highly Sensitised HLA patient	Main Plenary – Rooms 106-105 Level 1, MCEC
15:45–16:45	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 1: Ischaemia Reperfusion and New Techniques Free Communications 2: Transplant Complications Free Communications 3: Regulatory T cells	Rooms 106-105, Level 1, MCEC Room 104, Level 1, MCEC Room 103, Level 1, MCEC
16:45–17:00	Afternoon tea	Foyer, Level 1, MCEC
17:00–18:00	Novartis Josette Eris Lecture The Importance of Unconscious Bias in Decision Making	Main Plenary – Rooms 106-105 Level 1, MCEC
18:00–19:00	Welcome Reception and Poster Viewing	Foyer, Level 1, MCEC

Monday, 30 April 2018		
06:15–07:15	Fit For Life Fun Run/Walk (5km) Sponsor: Transplant Australia	Convention Centre to MCG return
07:00–08:00	Vascular Composite Allograft Advisory Committee (VCAAC)	St Vincent's Private Hospital Ground floor Café 59 Victoria Parade, Fitzroy
07:30–08:00	Coffee with sponsors	Foyer, Level 1, MCEC
08:00–09:40	PLENARY 2: Joint TSANZ /OTA/ATCA Session	Main Plenary – Rooms 106-105 Level 1, MCEC
09:40–10:40	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 4: Organ Donation and Ethics#1 Free Communications 5: Immunobiology Free Communications 6: Outcome Measures	Main Plenary – Rooms 106-105 Level 1, MCEC Room 104, Level 1, MCEC Room 103, Level 1, MCEC
10:40–11:10	Morning Tea and Poster viewing	Foyer, Level 1, MCEC
11:10–12:50	PLENARY 3: Astellas Symposium New Approaches to Donor-Recipient Matching	Main Plenary – Rooms 106-105 Level 1, MCEC
12:50–13:35	Lunch and Poster viewing Immunology and Tolerance Advisory Committee (ITAC) Xenotransplantation Working Group Meeting (XTWG)	Foyer, Level 1, MCEC Room 102, Level 1, MCEC Room 103, Level 1, MCEC
12:50–14:20	Lung Transplant Advisory Committee (LTAC)	Room 101, Level 1, MCEC
13:00–13:30	Paediatric Transplant Advisory Committee (PTAC)	Room 104, Level 1, MCEC
13:35–15:35	President's Prize Symposium	Main Plenary – Rooms 106-105 Level 1, MCEC
15:35–16:00	Afternoon Tea	Foyer, Level 1, MCEC
16:00–17:00	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 7: Surgical Free Communications 8: Xenotransplantation Free Communications 9: Organ Donation and Ethics #2	Main Plenary – Rooms 106-105 Level 1, MCEC Room 104, Level 1, MCEC Room 103, Level 1, MCEC
17:00–18:00	TSANZ Annual General Meeting	Main Plenary – Rooms 106-105 Level 1, MCEC
19:00–23:00	TSANZ Annual Dinner	Showtime Event Centre South Wharf, Melbourne

Tuesday, 01 May 2018		
07:30–08:00	Coffee with sponsors	Foyer, Level 1, MCEC
08:00–09:30	Plenary 4: Novartis Symposium Immune Monitoring in Transplantation	Main Plenary – Rooms 106-105
09:30–10:30	CONCURRENT STATE OF THE ART SESSIONS	
	STATE OF THE ART 1: Novartis Symposium Women's Health Post Transplantation	Main Plenary – Rooms 106-105
	STATE OF THE ART 2: Astellas Symposium Management of Post-Transplant Complications	Room 104
10:30–11:00	Morning Tea	Foyer, Level 1, MCEC
11:00–12:30	CONCURRENT STATE OF THE ART SESSIONS	
	STATE OF THE ART 3: Novartis Symposium Transplantation Surgical Session	Main Plenary – Rooms 106-105
	STATE OF THE ART 4: Astellas Symposium Paired Kidney Exchange Program	Room 104
12:30–13:30	Lunch	Foyer, Level 1, MCEC
	Cardiac Transplant AC (CTAC)	Room 103, Level 1, MCEC
13:00–16:00	Liver and Intestinal Transplant (LITAC)	Room 104, Level 1, MCEC
13:30–15:00	Plenary 5: Novartis Symposium What's new in Transplantation	Main Plenary – Rooms 106-105
15:00–15:25	Afternoon Tea	Foyer, Level 1, MCEC
15:25–16:00	The Great Debate: 'Australia Should Allow Public Solicitation of Organ Donors'	Main Plenary – Rooms 106-105
16:00	ASM Concludes	



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OFFICE BEARERS OF THE TRANSPLANTATION SOCIETY OF AUSTRALIA AND NEW ZEALAND

President

Professor Stephen Alexander

President Elect & Chair, Advisory Committees/Working Groups

Professor Patrick Coates

Honorary Secretary

Dr Natasha Rogers

Treasurer

Dr Robert Carroll

Councillors

Dr Nick Cross - New Zealand Representative

A/Professor Kelli MacDonald - Liaison with Scientific Societies

Dr Christine Russell - Surgical Representative

Professor Nick Shackel - RACP AMDC Liaison Rep

A/Professor Bronwyn Levvey

Nigel Palk - ATCA Representative

Scientific Program & Education Committee (SPEC)

Prof Daniel Chambers (Co-Chair)	A/Prof Rosemary Masterson
A/Prof. Kelli MacDonald (Co-Chair)	Dr William Mulley
Dr Ross Francis	Prof. Henry Pleass
Prof Wayne Hawthorne	Dr Veena Roberts
Ms Rhonda Holdsworth	Dr Lucy Sullivan
A/Prof Andrew Jabbour	A/Prof Allison Tong
Dr Darren Lee	Dr John Whitlam

TSANZ Administrative Staff

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Program and Abstract Book

Ms Marina Katerehos

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SPONSORS

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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 – Basic Science & Clinical
(supported by TSANZ)

TSANZ Lafferty Award

TSANZ Aviva Rosenfeld Award for Excellence in Patient Care in Transplantation

Ian McKenzie Prize for Outstanding Contribution in Transplantation

Mark Cocks Transplantation Research Scholarship
(supported by Transplant Australia)

Novartis/TSANZ Early Career Researcher Awards
(previously Young Investigator Awards)

Kidney Health Australia Awards

FINANCIAL STATEMENTS

The Transplantation Society of Australia and New Zealand (TSANZ) Financials for the Year Ended December 2017 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

Dr Sandy Feng, MD, PhD

Professor of Surgery in Residence
Director, Abdominal Transplant Surgery Fellowship
University of California San Francisco
San Francisco, USA

Astellas Lecturer

Dr Stanley Jordan MD, FASN, FAST

Director, Nephrology & Transplant Immunology
Medical Director of Kidney Transplant Program
Cedars-Sinai Medical Center

Novartis Lecturer

Professor Vijay K Kuchroo D.V.M., PhD

Samuel L Wasserstrom Professor of Neurology, Harvard Medical School
Associate Member, Broad Institute
Director, Evergrande Centre for Immunologic Diseases
Harvard Medical School and Brigham and Women's Hospital
Boston, USA

Astellas Lecturer

Kathryn Tinckam MD MSc FRCPC FAST

Physician, Division of Nephrology
Director, Quality Improvement and Innovation,
UHN Transplant Program
Medical Director, HLA Laboratory,
Laboratory Medicine Program
Associate Professor, University of Toronto



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INVITED SPEAKERS

Professor Steve Alexander

Nephrology Department, Children's Hospital at Westmead, NSW

Dr Katherine Barraclough

Nephrology Department, The Alfred and Royal Melbourne Hospitals, Melbourne, VIC

Mrs Lucinda Barry

CEO & Board Members, Organ and Tissue Authority, Canberra, ACT

Professor Jeremy Chapman AC

Renal Unit, Westmead Hospital, Westmead, NSW

Professor Patrick (Toby) Coates

Renal Unit, Royal Adelaide Hospital, Adelaide, SA

Dr Rohit d'Costa

Royal Melbourne Hospital, Melbourne, VIC

Dr Paul J. Champion de Crespigny

Department of Nephrology, Royal Melbourne Hospital, Melbourne, VIC

Professor Mariapia Degli-Esposti

Lions Eye Institute, Perth, WA

Dr Emily Granger

Cardiothoracic Department, St Vincent's Hospital, Darlinghurst, NSW

A/Professor Carmel Hawley

Department of Nephrology, Princess Alexandra Hospital, Woolloongabba, QLD

Professor Martha Hickey

Gynaecology Research Centre, The Royal Women's Hospital, Melbourne, VIC

Ms Alison Hodak

Donation Specialist Coordinator, Donatelife SA

Ms Rhonda Holdsworth

Australian Red Cross Blood Service, South Melbourne, VIC

A/Professor Alex Holmes

Psychiatry Department, Royal Melbourne Hospital, Melbourne, VIC



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INVITED SPEAKERS

A/Professor Peter Hughes

Nephrology Department, Royal Melbourne Hospital, Melbourne, VIC

Dr Misty Jenkins

Immunology Department, Walter & Eliza Hall Institute for Medical Research, Parkville, VIC

Dr Shilpa Jesudason

Central Northern Adelaide Renal and Transplant Service, Adelaide, SA

Professor Axel Kallies

Molecular Immunology, Walter & Eliza Hall Institute for Medical Research, Parkville, VIC

Dr Joshua Kausman

Department of Nephrology, Royal Children's Hospital, Melbourne, VIC

Dr Lawrence Lau

Abdominal Organ Transplantation Program, Hepato-Pancreato-Biliary Program
University of Toronto

Dr Paul Lawton

Menzies School of Health Research, Darwin, NT

A/Professor Kelli MacDonald

Division of Immunobiology & Infectious Disease, QIMR Berghofer, Brisbane, QLD

Dr Amanda Robertson

Renal Unit, Royal Melbourne Hospital, Melbourne, VIC

Dr Karen Sheppard

The Sir Peter MacCallum Department of Oncology, The University of Melbourne, VIC

Dr Adam Testro

VIC Liver Transplant Unit, Austin Hospital, Melbourne, VIC

Dr Glen Westall

Dept of Allergy, Immunology & Respiratory Medicine, Alfred Hospital, Melbourne, VIC

Dr Chris Yates

Royal Melbourne Hospital, Melbourne, VIC



ABSTRACT REVIEW PROCESS AND PRESENTATION FORMATS

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A total of 115 abstracts were submitted this year. Abstracts were blinded for authors and institutions and were reviewed by four 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 2018 ASM. Each Free Communications session will have up to 4 oral presentations (15 minutes each; 12 minutes presentation and 3 minutes questions). Abstracts will also be displayed as posters and the poster session will be held during the Welcome Reception on Sunday April 29 and morning and afternoon tea on Monday April 30. Presenters should be at their posters during the poster sessions to answer any questions from delegates.

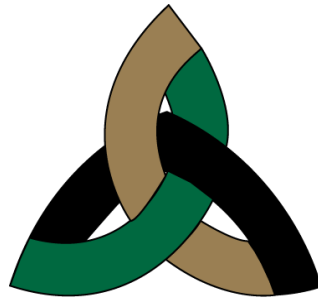
The President's Prize (PP) will be awarded in two categories: Basic Science and Clinical. The highest-ranked abstracts from eligible applicants in both categories will be presented in a single PP session. The award in each category will be based on the quality of the abstract and the presentation on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

The reviewers of the abstracts for the TSANZ 2018 meeting were:

Stephen Alexander	Bruce Hall	Ian McKenzie
Richard Allen	Wayne Hancock	Solomon Menahen
Adam Bartlett	Wayne Hawthorne	Bill Mulley
Michael Burke	Bulang He	Brian Nankivell
Scott Campbell	Geoff Hill	Kathy Nicholls
Robert Carroll	Peter Hopkins	Kathy Paizis
Steven Chadban	Min Hu	Henry Pleass
Daniel Chambers	Peter Hughes	Amanda Robertson
Philip Clayton	Frank Ierino	Paul Robertson
Toby Coates	Ashley Irish	Natasha Rogers
Shlomo Cohn	Nikky Isbel	Christine Russell
Michael Collins	Andrew Jabbour	Mauro Sandrin
Peter Cowan	Shilpanjali Jesudason	Shaundee Sen
Nick Cross	John Kanellis	Alexandra Sharland
Ian Dittmer	Sean Kennedy	Lucy Sullivan
Randall Faull	Gayathri Kumarasinghe	Suda Swaminathan
Jonathan Fawcett	Darren Lee	Paul Trevillian
Michael Fink	Wai Lim	Angela Webster
Ross Francis	Tom Loudovaris	Glen Westall
Allan Glanville	Grant Luxton	Germaine Wong
Hilton Gock	Peter Macdonald	Walker Rowan
David Goodman	Rosemary Masterson	Kate Wyburn
David Gracey	Geoffrey McCaughan	

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-sixth Annual Scientific Meeting

PROGRAM

Sunday, 29 April 2018

09:00–10:00	Donor Surgeons and Donor Coordinators (DCDS)	Room 101, Level 1, MCEC
12:00–13:00	Renal Transplant Surgeons Sub-Committee (RTSS)	Room 102, Level 1, MCEC
13:00–14:00	Pancreas & Islet Advisory Committee Meeting (PITAC)	Room 101, Level 1, MCEC
13:00–14:30	Paediatric Donor Working Group (PDWG)	Room 102, Level 1, MCEC

Sunday, April 29 2018

14:00–15:00	Registration	Foyer, Level 1 Melbourne Convention and Exhibition Centre (MCEC)
15:00–15:10	Official Opening: TSANZ President Prof Stephen Alexander	Main Plenary – Rooms 106-105 Level 1, MCEC
15:10–15:40	PLENARY 1: Astellas Symposium <i>Chairs: Dr Bill Mulley and Dr Kate Wyburn</i> Desensitisation of the Highly HLA Sensitised Patient Dr Stanley Jordan	Main Plenary – Rooms 106-105 Level 1, MCEC
15:45–16:45	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 1: Ischaemia Reperfusion Injury and New Techniques <i>Chairs: A/Prof Helen Thomas and Dr Wai Lim</i>	Rooms 106-105, Level 1, MCEC
Abstract	— Oral presentations —	
1	15:45 ISOLATING ISLETS FOR TRANSPLANTATION USING AN ISOLATOR SYSTEM, A VIABLE ALTERNATIVE TO CONVENTIONAL CLEAN ROOM FACILITIES. <u>KOS C</u> , LOUDOVARIS T, _MARIANA L, MCCORMICK K, BLEASDALE N, IRVIN A, WAIBEL M, THOMAS H, KAY T	
2	16:00 ANALYTICAL AND CLINICAL VALIDATION OF A NEW, HIGHLY INFORMATIVE, SENSITIVE AND PRECISE METHOD FOR CELLULAR CHIMERISM ANALYSIS <u>WHITLAM John</u> , LING Ling, SWAIN Michael, HARRINGTON Tom, MIROCHNIK Oksana, BROOKS Ian, CRONIN Sara, CHALLIS Jackie, PETROVIC Vida, BRUNO Damien, MECHINAUD Francoise, CONYERS Rachel, SLATER Howard	
3	16:15 CYCLOPHILIN BLOCKADE PROTECTS FROM RENAL ISCHAEMIA/REPERFUSION INJURY <u>LEONG Khai Gene</u> , OZOLS Elyce, KANELIS John, LILES John, NIKOLIC-PATERSON David, MA Frank	
4	16:30 ACTIVATED CD47 PROMOTES ACUTE KIDNEY INJURY BY LIMITING AUTOPHAGY <u>EL RASHID Mary</u> , SANGANERIA Barkha, ROGERS Natasha M	

Sunday, April 29 2018

15:45–16:45	Free Communications 2: Transplant Complications		Room 104, Level 1, MCEC
	<i>Chairs: Prof Matthew Jose and Dr Katherine Barraclough</i>		
Abstract	— <i>Oral presentations</i> —		
5	15:45	THE EFFECT OF POST-TRANSPLANT LYMPHOPROLIFERATIVE DISEASE (PTLD) ON GRAFT AND PATIENT SURVIVAL IN KIDNEY TRANSPLANT RECIPIENTS <u>FRANCIS Anna</u> , CRAIG Jonathan, JOHNSON David, WONG Germaine	
6	16:00	DE NOVO OR EARLY CONVERSION TO EVEROLIMUS AND LONG-TERM CANCER OUTCOMES IN KIDNEY TRANSPLANT RECIPIENTS: A TRIAL-BASED ANZDATA LINKAGE STUDY <u>YING Tracey</u> , WONG G, LIM W, RUSS G, PILMORE H, KANELIS J, GOODMAN D, TREVILLIAN P, CAMPBELL S, SURANYI M, MATHEW M, FAULL R, MASTERSON R, WALKER R, O'CONNELL P, CHADBAN S	
7	16:15	CANCER MORTALITY IN KIDNEY TRANSPLANT RECIPIENTS IN AUSTRALIA AND NEW ZEALAND: A COHORT STUDY FROM 1980 TO 2013. <u>ROSALES Brenda</u> , DE LA MATA Nicole, KELLY Patrick, WEBSTER Angela	
8	16:30	MORTALITY RATES IN LIVING KIDNEY DONORS: AN AUSTRALIAN AND NEW ZEALAND COHORT STUDY USING DATA LINKAGE DE LA MATA Nicole, CLAYTON Philip, MCDONALD Stephen, CHADBAN Steven, POLKINGHORN Kevan, <u>WEBSTER Angela</u>	
15:45–16:45	Free Communications 3: Regulatory T Cells		Room 103, Level 1, MCEC
	<i>Chairs: Prof Bruce Hall and A/Prof Alexandra Sharland</i>		
Abstract	— <i>Oral presentations</i> —		
9	15:45	ADMINISTRATING IL2 COMPLEX IN THE PRESENCE OF ALLOANTIGEN COMBINED WITH DONOR CELL TRANSFUSION EXPANDS ALLO-SPECIFIC TREGS FACILITATING TOLERANCE INDUCTION ACROSS A RESTRICTED MAJOR MISMATCH IN A SKIN GRAFT MODEL <u>ZHANG Geoff Y</u> , WANG Yuan Min , HU Min, KARUNIA Jevin, GREY Shane, ALEXANDER Stephen I	

Sunday, April 29 2018

10	16:00	HIGH FIBRE DIET INDUCES DONOR SPECIFIC TOLERANCE OF KIDNEY ALLOGRAFT THROUGH SHORT CHAIN FATTY ACID INDUCTION OF TREGS <u>WU Huiling</u> , KWAN Tony, LOH Yik Wen, WANG Chuanmin, MACIA Lanrence, ALEXANDER Stephen, CHADBAN Steven
11	16:15	IN-VIVO COSTIMULATION-BLOCKADE INDUCED LONG-TERM FOXP3+ REGULATORY T CELLS WITH MARKERS OF CD4+GFP+CD44+CD127HICD62L- DEMONSTRATE THE ANTIGEN-SPECIFIC POTENCY FROM IMMUNODEFICIENT MICE WITH NEONATAL ISLET CELL CLUSTERS TOLERANT XENOGRAFTS <u>ZHAO Yuanfei</u> , HAWTHORNE Wayne, BURNS Heather, QIAN YIWEN, YI SHOUNAN, ZHANG GEOFF, ALEXANDER Stephen, HU Min, O'CONNELL Philip
12	16:30	IN VITRO EVALUATION OF HUMAN REGULATORY T-CELLS IN A 3D-PRINTED STRUCTURE <u>KIM Juewan</u> , YUE Zhillian, LIU Xiao, HOPE Christopher, ROJAS-CANALES Darling, DROGEMULLER Christopher, CARROLL Robert, BARRY Simon C, WALLACE Gordon G, COATES P. Toby
16:45–17:00 Afternoon tea Foyer, Level 1, MCEC		
17:00–18:00	Novartis Josette Eris Lecture <i>Chairs: Prof Stephen Alexander and Dr Natasha Rogers</i> The Importance of Unconscious Bias in Decision Making Dr Paul Lawton and Dr Misty Jenkins	
18:00–19:00 Welcome Reception and Poster Viewing		Foyer, Level 1, MCEC

Monday, April 30 2018

06:15–07:15	Fit For Life Fun Run/Walk (5km) Sponsor: Transplant Australia	Convention Centre to MCG return
07:00–8:00	Vascular Composite Allograft Advisory Committee (VCAAC)	St Vincent's Private Hospital, Ground floor Café, 59 Victoria Parade, Fitzroy, Melb
07:30–08:00	Coffee with sponsors	Foyer, Level 1, MCEC
08:00–09:40	PLENARY 2: Organ and Tissue Authority Symposium Joint TSANZ / OTA/ATCA Session <i>Chairs: A/Prof Shlomo Cohnen and Dr Lucy Sullivan</i> 08:00 Regulatory Network That Controls Development of Treg and Th17 Cells Prof Vijay Kuchroo 08:30 Differentiation and Function of Tissue-Specific Treg Cells Prof Axel Kallies 08:50 Donor Disease Transmission in Australia Prof Jeremy Chapman 09:10 2017 ATCA Organ Allocation Rotations Audit & Quality Control Report Ms Alison Hodak 09:25 OTA Session Mrs Lucinda Barry	Main Plenary – Rooms 106-105 Level 1, MCEC
09:40–10:40	CONCURRENT FREE COMMUNICATIONS SESSIONS Free Communications 4: Organ Donation and Ethics #1 <i>Chairs: Dr Stella McGinn and Dr Peter Hughes</i>	Main Plenary – Rooms 106-105 Level 1, MCEC
Abstract	— Oral presentations —	
13	09:40 CHARACTERISING FAMILY REFUSALS IN SOLID ORGAN DONATION (CREDO) STUDY <u>SKLIROS Christopher</u> , DE LA MATA Nicole, HEDLEY James, WYBURN Kate, O'LEARY Michael, WEBSTER Angela	

Monday, April 30 2018

14	09:55	INTRODUCTION OF SHARE 35 INTERREGIONAL ALLOCATION FOR HIGH MELD LIVER TRANSPLANT WAITING LIST PATIENTS IN AUSTRALIA AND NEW ZEALAND <u>FINK Michael</u> , GOW Paul, BALDERSON Glenda, JONES Robert
15	10:10	WHAT HAPPENED WHEN THE ‘SOFT OPT-OUT’ TO ORGAN DONATION WAS IMPLEMENTED IN WALES? FAMILY AND PROFESSIONAL VIEWS AND EXPERIENCES, AND CONSENT RATES FOR THE FIRST 18 MONTHS. NOYES Jane, <u>MC LAUGHLIN Leah</u> , MORGAN Karen, WALTON Phil, ROBERTS Abigail, STEPHENS Michael
16	10:25	THE INCREASED RATE OF NON-UTILISATION OF KIDNEYS RETRIEVED FOR TRANSPLANTATION IN AUSTRALIA IS INDEPENDENT OF DONOR CHARACTERISTICS. <u>SYPEK MATTHEW</u> , ULLAH Shahid, CLAYTON Phil, MCDONALD Stephen
09:40–10:40		Free Communications 5: Immunobiology <i>Chairs: Dr Kelli MacDonald and Prof Wayne Hawthorn</i>
Abstract		— <i>Oral presentations</i> —
17	09:40	THE CHANGES OF DYNAMIC IMMUNE PROFILE AND OUTCOME DIFFERED BETWEEN INDIVIDUAL WITH TYPE 1 DIABETES ISLET TRANSPLANTATION UNDER ATG/TACROLIMUS/MMF/ETANERCEPT SUPPRESSION REGIMEN HU Min, VERA Elvira Jimenez, CHEW Yi Vee, BURNS Heather, ANDERSON Patricia, WILLIAMS Lindy, KEUNG Karen, DERVISH Suat, WANG Xin Maggie, ROGERS Natasha, YI Shounan, HAWTHORNE Wayne, ALEXANDER Stephen, <u>O’CONNELL Philip</u>
18	09:55	THE ROLE OF THE CD73/A2A SIGNALLING AXIS IN A HUMANISED MOUSE MODEL OF GRAFT-VERSUS-HOST DISEASE <u>GERAGHTY Nicholas</u> , ADHIKARY Sam, SLUYTER Ronald, WATSON Debbie

Room 104, Level 1,
MCEC

Monday, April 30 2018

19	10:10	CHANGES IN THE EXTRACELLULAR MATRIX - SIGNS OF REMODELING LEADING TO CHRONIC REJECTION AFTER LUNG TRANSPLANTATION MULLER Catharina, HEINKE Paula, ANDERSSON-SJÖLAND Annika, SCHULTZ Hans Henrik, ANDERSEN Claus, IVERSEN Martin, WESTERGREN-THORSSON Gunilla, <u>ERIKSSON Leif</u>	
20	10:25	MTORC2 DEFICIENCY IN DENDRITIC CELLS PROMOTES ACUTE KIDNEY INJURY <u>ROGERS Natasha</u> , DAI Helong, WATSON Alicia, THOMSON Angus	
09:40–10:40	Free Communications 6: Outcome Measures <i>Chairs: A/Prof Daniel Chambers and Dr Nikki Isbel</i>		Room 103, Level 1, MCEC
Abstract	— Oral presentations —		
21	09:40	OUTCOMES OF WESTERN AUSTRALIAN LUNG TRANSPLANT RECIPIENTS – THE FIRST DECADE <u>DHILLON Sarbroop</u> , MCKINNON Elizabeth, MUSK Michael, WROBEL Jeremy, LAVENDER Melanie, GABBAY Eli	
22	09:55	ALLOGRAFT OUTCOME FOLLOWING RETRANSPLANTATION OF PATIENTS WITH FAILED FIRST KIDNEY ALLOGRAFT ATTRIBUTED TO NON-ADHERENCE <u>MANICKAVASAGAR Revathy</u> , WONG Germaine, LIM Wai H	
23	10:10	EVEROLIMUS AND LONG-TERM CLINICAL OUTCOMES IN KIDNEY TRANSPLANT RECIPIENTS: A TRIAL-BASED ANZDATA LINKAGE STUDY <u>YING Tracey</u> , WONG G, LIM W, RUSS G, PILMORE H, KANELIS J, GOODMAN D, TREVILLIAN P, CAMPBELL S, SURANYI M, MATHEW M, FAULL R, MASTERSON R, WALKER R, O'CONNELL P, CHADBAN S	
24	10:25	POST-TRANSPLANT SURVIVAL IN TYPE 1 DIABETICS IN AUSTRALIA AND NEW ZEALAND <u>WEBSTER Angela</u> , HEDLEY James, KELLY Patrick	
10:40–11:10	Morning tea and Poster viewing		Foyer, Level 1, MCEC

Monday, April 30 2018

Abstract

— *Poster presentations* —

- | | |
|----|---|
| 25 | DECEASED-DONOR KIDNEY TRANSPLANTATION PROGRAM IN NEW CALEDONIA
<u>DELEZIRE Arnaud</u> , QUIRIN Nicolas, HAIDAR Fadi, CARCELES Odette, LAMY Thomas, BUTTE Yann |
| 26 | DELAYED KIDNEY TRANSPLANTATION WAIT-LISTING- CLIENT AND CLINICIAN PERCEPTIONS OF THE IMPACT OF EXTENSIVE CHRONIC CUTANEOUS DERMATOPHYTE INFECTION
<u>MAJONI Sandawana William</u> , HUGHES Jaquelyne T , AYE Min Oka, WHITE Evonne, HALL Heather, CURRIE Bart J, KIRKHARM Ranae |
| 27 | SURVIVAL AND FUNCTION OF HUMAN ADRENAL CELL IN IMMUNOISOLATION DEVICE IN ADRENALECTOMIZED IMMUNODEFICIENT MICE
<u>CATTERALL T</u> , KRISHNA MURTHY B, MARIANA L, KOS C, SACHITHANANDAN N, THOMAS H, LOUDOVARIS T, KAY T |
| 28 | DEVELOPING PHOSPHOLIPASE A2 RECEPTOR ScFv FOR CAR TREGS FOR THE TREATMENT OF AUTOIMMUNE RENAL DISEASE
<u>KARUNIA J</u> , Wang YM, ZHANG GY, WILARAS A, BAKHTIAR M, McCarthy H, ALEXANDER SI |
| 29 | DONATION AFTER CIRCULATORY DEATH COMPARED WITH DONATION AFTER BRAIN DEATH: OUTCOMES FOR ISLET TRANSPLANTATION IN AUSTRALIA
<u>HAWTHORNE Wayne</u> , CHEW YiVee, WILLIAMS Lindy, HARON Christian, HITOS Kerry, MARIANA Lina, KAY Tom, O'CONNELL Philip, LOUDOVARIS Tom |
| 30 | COMPARISON OF PANCREATA AND ISLET PREPARATIONS FROM HUMAN ORGAN DONORS
<u>MARIANA Lina</u> , LOUDOVARIS Thomas, KOS Cameron, PAPAS Evan, SELCK Claudia, CATTERALL Tara, THOMAS Helen, KAY Thomas WH |
| 31 | THE PREVALENCE OF ACQUIRED CYSTIC KIDNEY DISEASE (ACKD) IS NOT INCREASED IN RENAL TRANSPLANT RECIPIENTS WITH RENAL TUMOURS.
<u>RHEE Handoo</u> , TAN Ai Lin, GRIFFIN Anthony, PRESTON John, LAWSON Malcom, WOOD Simon |

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| 32 | <p>COMPARISON OF TUMOUR CHARACTERISTICS IDENTIFIED IN THE ALLOGRAFT AND THE NATIVE KIDNEYS OF RENAL TRANSPLANT RECIPIENTS
 <u>TAN Ai Lin</u>, WOOD Simon, PRESTON John, LAWSON Malcolm, GRIFFIN Anthony, Rhee Handoo</p> |
| 33 | <p>INFECTIOUS COMPLICATIONS IN THE SOUTHERN TASMANIAN KIDNEY TRANSPLANT POPULATION
 <u>ABEYSEKERA N</u>, GRAVER A, COOLEY L, KIRKLAND G, JOSE MD</p> |
| 34 | <p>CLEARANCE OF BK VIRUS NEPHROPATHY BY COMBINATION ANTIVIRAL THERAPY WITH INTRAVENOUS IMMUNOGLOBULIN
 <u>KABLE Kathy</u>, DAVIES Carmen, O'CONNELL Philip, CHAPMAN Jeremy, NANKIVELL Brian</p> |
| 35 | <p>FIRST REPORTED CASE OF GANCICLOVIR-RESISTANT POST-TRANSPLANT CYTOMEGALOVIRUS INFECTION DUE TO COMBINED DELETION MUTATION IN CODONS 595-596 OF THE UL97 GENE
 <u>LEUNG PO YEE Mia</u>, TRAN Thomas, TESTRO Adam, PAIZIS Kathy, KWONG Jason, WHITLAM John</p> |
| 36 | <p>EMPHYSEMATOUS PYELONEPHRITIS IN A DUAL KIDNEY TRANSPLANT RECIPIENT
 <u>TANGIRALA Nishanta</u>, SINGER Julian, ANDERSON Lyndal, LAURENCE Jerome, GRACEY David</p> |
| 37 | <p>GLP1RA SUCCESSFULLY TREATS HYPERGLYCAEMIA IN RENAL TRANSPLANT RECIPIENTS AND ENABLES SUBSTANTIAL REDUCTION IN INSULIN REQUIREMENTS AND WEIGHT
 <u>KAMESHWAR Kamya</u>, FOURLANOS Spiros, HIDAYATI Leny, CHEONG Jamie, LEVIDIOTIS Vicki, COHNEY Solomon</p> |

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| 38 | <p>ONE YEAR INCIDENCE & PREVALENCE OF NEWLY DETECTED ABNORMAL GLUCOSE METABOLISM IN RENAL TRANSPLANT PATIENTS ON MAINTENANCE PREDNISOLONE AND CNI (2004-2009)</p> <p><u>PIMENTEL AL</u>, MASTERSON R, YATES C, HUGHES P, CAMARGO JL, COHNEY S</p> |
| 39 | <p>METFORMIN, GLICLAZIDE AND INSULIN REMAIN THE MOST COMMONLY USED AGENTS FOR POST-TRANSPLANT DIABETES (PTDM) IN A COHORT OF RENAL TRANSPLANT RECIPIENTS</p> <p><u>PIMENTEL AL</u>, MASTERSON R, YATES C, HUGHES P, COHNEY S</p> |
| 40 | <p>ABSENT SMOOTH MUSCLE ACTIN IMMUNOREACTIVITY OF THE SMALL BOWEL MUSCULARIS PROPRIA CIRCULAR LAYER – A NOVEL FINDING IN A DYSMOTILE INTESTINAL ALLOGRAFT</p> <p><u>HARDIKAR Winita</u>, BOLIA Rishi, STARKEY Graham, TESTRO ADAM, Holmes Kathe, MURPHY Samantha, JONES Robert</p> |
| 41 | <p>INTRACTABLE ASCITES FOLLOWING RENAL TRANSPLANT IN AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE PATIENTS WITH MASSIVE POLYCYSTIC LIVER</p> <p><u>MARUI Yuhji</u>, FUJIMOTO Eisuke, AIDA Koichiro, SASAKI Hideo, KOIZUMI Satoshi, OTSUBO Takehito, CHIKARAISHI Tatsuya</p> |
| 42 | <p>RENAL TRANSPLANTATION IN THE ELDERLY POPULATION: SURGICAL OUTCOMES IN THE QUEENSLAND NETWORK OVER A 10 YEAR PERIOD.</p> <p><u>FADAE NEESA</u>, ROBERTSON Ian, RHEE Handoo, GRIFFIN Anthony</p> |
| 43 | <p>EARLY REMOVAL OF JJ STENTS IN RENAL TRANSPLANT RECIPIENTS : A PILOT STUDY OF FEASIBILITY AND SAFETY.</p> <p><u>JAMBOTI Jagadish</u>, BHANDARI Mayank, GODDARD Kim, NAVADGI Suresh, SWAMINATHAN Ramyasuda, ABRAHAM Abu, IRISH Ashley, TAN Andrew, PUTTAGUNTA Harish, O'BRIEN Orla, WARGER Anne, STINNETTE Megan, PRADABHAN Salivahane</p> |

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| 44 | OUTCOMES OF EARLY URETERIC STENT REMOVAL IN PAEDIATRIC KIDNEY TRANSPLANTATION
<u>NG Zi Qin</u> , HE Bulang |
| 45 | PARTIAL VERSUS COMPLETE THROMBOSIS MODERATED BY INTRA-OPERATIVE VASOPRESSOR USE IN SPK PATIENTS
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| 46 | MACHINE LEARNING PREDICTION FOR DE NOVO DONOR SPECIFIC ANTIBODIES (DNDSA) AND GRAFT LOSS IN SIMULTANEOUS KIDNEY PANCREAS TRANSPLANT (SPK) RECIPIENTS
<u>COOREY Craig</u> , SHARMA Ankit, CHAPMAN Jeremy, CRAIG Jonathan, O'CONNELL Philip, LIM Wai, NANKIVELL Brian, TAVERNITI Anne, WONG Germaine, YANG Jean |
| 47 | HLA EPLET MISMATCH AND DONOR SPECIFIC ANTIBODIES IN KIDNEY TRANSPLANTATION
<u>WAN Susan</u> , ANGEL DE WILDE Sian, ROSALES Brenda, CHADBAN Steven, WYBURN Kate |
| 48 | PROPHYLACTIC PLASMA EXCHANGE IS ASSOCIATED WITH A HIGH INCIDENCE OF AMR IN SENSITISED RECIPIENTS
<u>CHAMBERLAIN AJ</u> , SNIDER J, POWER DA, WHITLAM JB |
| 49 | TISSUE-RESIDENT LYMPHOCYTES IN SOLID ORGAN TRANSPLANTATION
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| 50 | CYTOMEGALOVIRUS INFECTION FOLLOWING LUNG TRANSPLANTATION INCREASES NATURAL KILLER CELLS EXPRESSING ACTIVATING RECEPTORS
<u>SULLIVAN Lucy</u> , HARPUR Christopher, STANKOVIC Sanda, BROOKS Andrew, WESTALL Glen |

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| 51 | TREG RECONSTITUTION PREVENTS GVHD
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FAZEKAS DE ST GROTH Barbara |
| 52 | IDENTIFICATION OF CHANGES IN PHENOTYPE OF ALLOANTIGEN ACTIVATED CD4+CD25+TREG COMPARED TO NAÏVE TTREG
<u>HALL Bruce M</u> , ROBINSON Catherine M, VERMA Nirupama D, NOMURA Masaru, WILCOX Paul, TRAN Giang T, CARTER Nicole, BOYD Rochelle, HODGKINSON Suzanne J |
| 53 | MONITORING OF HUMAN CD4+CD25HICD127LOCD45RA-FOX3HI TREG POPULATION AS POSSIBLE ALLOANTIGEN-SPECIFIC TREG
<u>VERMA Nirupama D</u> , LAM Andrew, CHIU Chris, ROBINSON Catherine M, TRAN Ginag T, HODGKINSON Suzanne J, HALL Bruce M |
| 54 | ALLOACTIVATION OF HUMAN CD4+CD25+CD127LOFOX3+TREG TO INDUCE ACTIVATED TREG
<u>VERMA Nirupama D</u> , LAM Andrew, CHIU Chris, ROBINSON Catherine M, TRAN Giang T, HODGKINSON Suzanne J, HALL Bruce M |
| 55 | RISK STRATIFICATION FOR REJECTION BY EPLET MISMATCH AFTER EARLY MYCOPHENOLATE DOSE REDUCTION IN KIDNEY TRANSPLANT RECIPIENTS
<u>COUGHLAN Timothy</u> , CANTWELL Linda, LEE Darren |
| 56 | EFFICACY OF EVEROLIMUS FOR IMPROVING RENAL IMPAIRMENT IN LIVER TRANSPLANT PATIENTS: A SINGLE CENTRE, AUSTRALIAN EXPERIENCE
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| 57 | DONOR HEART PRESERVATION: APPLYING THE ACID TEST
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| 58 | OVERCOMING BARRIERS FOR INDIGENOUS AUSTRALIANS GAINING ACCESS TO THE KIDNEY TRANSPLANT WAITING LIST
<u>ATKINSON Amy</u> , FORD Sharon, GOCK Hilton, IERINO Frank, GOODMAN David |
| 59 | EXPLORING THE IMPACT OF RECIPIENT AGE WITH KIDNEY DONOR RISK INDEX AND ESTIMATED GLOMERULAR FILTRATION RATE AT ONE YEAR FOLLOWING KIDNEY TRANSPLANT
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| 60 | NORMOTHERMIC MACHINE PERFUSION OF NON-UTILIZED HUMAN KIDNEYS – OUR FIRST TWO CASES
<u>HAMEED Ahmer</u> , ROGERS Natasha, DE ROO Ronald, LU Bo, ROBERTSON Paul, ZHANG Chris, GASPI Renan, MIRAZIZ Ray, NGUYEN Hien, YUEN Lawrence, ALLEN Richard, HAWTHORNE Wayne, PLEASS Henry |
| 61 | WITHDRAWN |
| 62 | WITHDRAWN |
| 63 | CHALLENGES TO PROCEEDING TO ORGAN DONATION IN THE NORTHERN TERRITORY
<u>MCAULIFFE Kathryn</u> , WOOD Lee, JONES Sarah |
| 64 | IMPACT OF A DEDICATED LIVING DONOR CLINIC AND ASSESSMENT TEAM: A SINGLE CENTRE EXPERIENCE
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| 65 | CLINICIAN'S ATTITUDES AND PERSPECTIVES ON THE ACCEPTABILITY OF ANTE-MORTEM INTERVENTIONS: AN INTERNATIONAL SEMI-STRUCTURED INTERVIEW STUDY
<u>SHAHRESTANI Sara</u> , HAWTHORNE Wayne, PLEASS Henry, WONG Germaine, TONG Allison |

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| 66 | WHERE ARE THE DONORS? A SIX-MONTH AUDIT OF DEATHS IN A LARGE SYDNEY TEACHING HOSPITAL
<u>SPOSARI Venessa</u> , LEIJTEN Monique |
| 67 | ALLOCATION OF DECEASED DONOR KIDNEYS IN CLINICAL PRACTICE: MATCHING GRAFT LIFE-YEARS AND RECIPIENT LIFE EXPECTANCY
<u>YONG Bryan</u> , IERINO Frank, PAIZIS Kathy, POWER David |
| 68 | IN VIVO DEPLETION OF REACTIVE DONOR HUMAN CELLS REDUCES THE DEVELOPMENT OF GRAFT-VERSUS-HOST DISEASE IN A HUMANISED MOUSE MODEL
<u>ADHIKARY Sam</u> , GERAGHTY Nicholas, SLUYTER Ronald, WATSON Debbie |
| 69 | VALIDATION OF THE ONE LAMBDA FLOWDSA™ ASSAY FOR LIVING DONOR TRANSPLANT WORKUP
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| 70 | IMMUNOSUPPRESSANT PRESCRIBING PRACTICES IN YOUNGER ADULTS COMPARED TO ELDERLY RENAL TRANSPLANT RECIPIENTS ACROSS AUSTRALIA AND NEW ZEALAND
<u>COSSART Amelia</u> , COTTRELL Neil, MCSTEAN Megan, ISBEL Nicole, CAMPBELL Scott, STAATZ Christine |
| 71 | ANZDATA INDIVIDUAL HOSPITAL REPORTING METHODOLOGY CHANGES
DAVIES Christopher, <u>SYPEK Matthew</u> , CLAYTON Philip, MCDONALD, Stephen |
| 72 | PROTOCOL RENAL TRANSPLANT BIOPSIES IN A NON-TRANSPLANTING HOSPITAL – DO THEY CHANGE MANAGEMENT?
<u>HEPBURN Kirsten</u> , BROWN Mark |
| 73 | IMMUNE PHENOTYPE BY FLOW CYTOMETRY OF PEDIATRIC KIDNEY TRANSPLANT RECIPIENTS AND HEALTHY ADULT CONTROLS.
JIMENEZ-VERA Elvira, <u>ZHAO Yuanfei</u> , HU Min, CHEW Yi Vee, BURNS Heather, ANDERSON Patricia, WILLIAMS Lindy, DERVISH Suat, WANG Xin Maggie, YI Shounan, HAWTHORNE Wayne, ALEXANDER Stephen, O'CONNELL Philip |

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| 74 | <p>RISK INDICES IN DECEASED DONOR ORGAN ALLOCATION FOR TRANSPLANTATION: REVIEW FROM AN AUSTRALIAN PERSPECTIVE</p> <p><u>LING Jonathan</u>, FINK Michael, WESTALL Glen, MACDONALD Peter, CLAYTON Philip, OPDAM Helen, HOLDSWORTH Rhonda, POLKINGHORNE Kevan, KANELIS John</p> |
| 75 | <p>IN VITRO SCREENING OF GENES ASSOCIATED WITH KIDNEY FIBROSIS</p> <p><u>MA Xiaoqian</u>, SUN Lei, LU CAO, YI Shounan, O'CONNELL Philip</p> |
| 76 | <p>FACTORS ASSOCIATED WITH SUCCESSFUL RENAL TRANSPLANTATION IN INDIGENOUS RECIPIENTS FROM THE TOP END OF NORTHERN AUSTRALIA WHERE GRAFT AND PATIENT OUTCOMES ARE GENERALLY POOR</p> <p><u>MAJONI Sandawana William</u>, TINSLEY Nadine, CASILLI Alyce, DOLE Kerry</p> |
| 77 | <p>WITHDRAWN</p> |
| 78 | <p>POST-TRANSPLANT ACUTE KIDNEY INJURY AFFECTS LONG-TERM GRAFT FUNCTION</p> <p><u>PRAKASH MP</u>, ZHUO Tally, HEDLEY James, WEBSTER Angela, ROGERS Natasha</p> |
| 79 | <p>LONG-TERM GRAFT SURVIVAL AND FUNCTION IN RECIPIENTS OF DCD COMPARED TO DBD RENAL ALLOGRAFTS: A SINGLE CENTRE REVIEW.</p> <p><u>SALTER Sherry</u>, TAN Sarah, MULLEY William, CHAMBERLAIN Stacey, POLKINGHORNE Kevan, SAUNDER Alan, KANELIS John</p> |
| 80 | <p>COMPARISON OF 3 LYMPHOCYTE SEPARATION METHODS FOR FLOW CROSSMATCH ASSAY</p> <p><u>TASSONE Gabriella</u>, BAZLEY Scott, D'ORSOGNA Lloyd, MARTINEZ Patricia, DE SANTIS Dianne</p> |
| 81 | <p>EXTENDED CRITERIA DONATION UNDER EXTENDED CRITERIA CIRCUMSTANCES</p> <p><u>THOMPSON Sophie</u>, PILCHER David, IHLE Joshua</p> |

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| 82 | <p>EPIDEMIOLOGY AND ESTIMATED COST OF COMPLICATED SIMULTANEOUS PANCREAS KIDNEY TRANSPLANTATION</p> <p><u>XU Joshua</u>, HITOS Kerry, HORT Amy, SHAHRESTANI Sara, ROBERTSON Paul, YUEN Lawrence , RYAN Brendan, DE ROO Ronald , HAWTHORNE Wayne, PLEASS Henry</p> |
| 83 | <p>TUMOUR RESECTED KIDNEY GRAFTS FOR TRANSPLANTATION IN WESTERN AUSTRALIA: OUCTOMES OF THE TRK PROGRAM, 10 YEARS ON.</p> <p><u>APIKOTOA Sharie</u>, HE Bulang</p> |
| 84 | <p>“TRUST TRIAL: TIMING OF REMOVAL FOR URETERIC STENTS POST-RENAL TRANSPLANTATION – EARLY VS STANDARD REMOVAL”</p> <p><u>CHA Ryan</u>, DARE A, HECKER W, YASITOMO M, PHILLIPS A, MUNN S, BARTLETT A</p> |
| 85 | <p>STROKE MORTALITY IN KIDNEEY TRANSPLANT RECIPIENTS: A POPULATION-BASED COHORT STUDY USING DATA LINKAGE</p> <p>DE LA MATA Nicole, MASSON Philip, AL-SHAHI SALMAN Rustam, KELLY Patrick, <u>WEBSTER Angela</u></p> |
| 86 | <p>THE ASSOCIATION BETWEEN ETHNICITY, ALLOGRAFT FAILURE AND MORTALITY AFTER KIDNEY TRANSPLANTATION IN INDIGENOUS AND NON-INDIGENOUS AUSTRALIANS: IS THIS EXPLAINED BY ACUTE REJECTION?</p> <p><u>HOWSON Prue</u>, IRISH Ashley, D'ORSOGNA Lloyd, SWAMINATHAN Ramyasuda, PERRY Gregory, De Santis Dianne, Wong Germaine, Lim Wai H</p> |
| 87 | <p>LONG-TERM OUTCOME OF KIDNEY TRANSPLANTATION IN PATIENTS WITH CONGENITAL ANOMALIES OF THE KIDNEY & URINARY TRACT</p> <p><u>MCKAY Ashlene</u>, KIM Siah, KENNEDY Sean</p> |

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| 88 | <p>COMPARISON OF KIDNEY ALLOGRAFT SURVIVAL IN THE EUROTRANSPLANT REGION AFTER CHANGING THE ALLOCATION CRITERIA IN 2010 – A SINGLE CENTER EXPERIENCE</p> <p><u>MEHDORN Anne-Sophie</u>, BECKER Felix, REUTER Stefan, SUWELACK Barbara, SENNINGER Norbert, VOGEL Thomas, PALMES Daniel, BAHDE Ralf</p> |
| 89 | <p>RENAL TRANSPLANT PATIENT AND GRAFT SURVIVAL UNAFFECTED BY POST-TRANSPLANT DIABETES IN THE ERA OF LOW MAINTENANCE IMMUNOSUPPRESSION</p> <p><u>PIMENTEL AL</u>, MASTERSON R, YATES C, HUGHES P, COHNEY S</p> |
| 90 | <p>REVIEW OF THE NEW ZEALAND (NZ) EXPERIENCE WITH DONATION AFTER CIRCULATORY DEATH (DCD) KIDNEY TRANSPLANTATIONS 2008-2016</p> <p><u>SUN Tina</u>, DITTMER Ian, MATHESON Philip</p> |
| 91 | <p>EVALUATING ALLOGRAFT RENAL FUNCTION BY CYSTATIN C ESTIMATING GLOMERULAR FILTRATION RATE FORMULAS AFTER KIDNEY TRANSPLANTATION</p> <p><u>TAM Tran Thai Thanh</u>, HOANG Khac Chuan, DU Thi Ngoc Thu, THAI Minh Sam, LE NGUYEN Thi , TRAN Ngoc Sinh</p> |
| 92 | <p>RANGE AND CONSISTENCY OF CARDIOVASCULAR OUTCOMES REPORTED IN CONTEMPORARY RANDOMISED TRIALS IN KIDNEY TRANSPLANT PATIENTS: A SYSTEMATIC REVIEW</p> <p><u>VAN Kim Linh</u>, O'LONE Emma, TONG Allison, VIECELLI Andrea, HOWELL Martin, SAUTENET Benedicte, MANERA Karine, CRAIG Jonathan</p> |
| 93 | <p>ARTERIAL RECONSTRUCTION IN LIVER TRANSPLANT WITH BOTH DONOR AND RECIPIENT HEPATIC ARTERY ANATOMICAL VARIATIONS</p> <p><u>MOU Lingjun</u>, KNAUSENBERGER Hannah, HE Bulang, HUANG Yangyang, PRADHAN Sharin, DELRIVIERE Luc, JAUQUES Bryon</p> |
| 94 | <p>PROPHYLACTIC DRAIN INSERTION IN RENAL TRANSPLANTATION: SURGEON PREFERENCE ACROSS AUSTRALIA AND NEW ZEALAND</p> <p><u>MUGINO Miho</u>, LAM Susanna, LAURENCE Yuen, VERRAN Deborah, ALLEN Richard, PLEASS Henry, LAURENCE Jerome</p> |

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95	<p>USE OF AN ICE BAG TO MINIMIZE THE PERIOD OF SECOND WARM ISCHAEMIC TIME DURING KIDNEY & PANCREAS TRANSPLANTATION – OUR INITIAL EXPERIENCE</p> <p><u>YOON Peter</u>, HAMEED Ahmer, NGUYEN Hien, GASPI Renan, HAWTHORNE Wayne, PLEASS Henry, YUEN Lawrence</p>	
11:10–12:50	<p>PLENARY 3: Astellas Symposium</p> <p>New Approaches to Donor-Recipient Matching</p> <p><i>Chairs: A/Prof John Kanellis and Ms Linda Cantwell</i></p> <p>11:10 Epitope and Virtual Cross-Matching A/Prof Kathryn Tinckam</p> <p>11:40 Epitopes - The Genomics of HLA Antibodies Ms Rhonda Holdsworth</p> <p>12:00 Eplet Matching and Outcomes Following Lung Transplantation Dr Glen Westall</p> <p>12:20 Eplets in Paediatric Renal Allograft Allocation Dr Joshua Kausman</p> <p>12:40 Questions</p>	<p>Main Plenary – Rooms 106-105 Level 1, MCEC</p>
12:50–13:35	<p>Lunch and Poster viewing</p> <p>Immunology and Tolerance Advisory Committee (ITAC) Xenotransplantation Working Group Meeting (XTWG)</p>	<p>Foyer, Level 1, MCEC</p> <p>Room 102, MCEC Room 103, MCEC</p>
12:50–14:20	Lung Transplant Advisory Committee (LTAC)	Room 101, MCEC
13:00–13:30	Paediatric Transplant Advisory Committee (PTAC)	Room 104, MCEC

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13:35–15:35	President's Prize Symposium		Main Plenary–Rooms
	<i>Chairs: Prof Stephen Alexander and Prof Toby Coates</i>		106-105 Level 1,
			MCEC
Abstract	— Oral presentations —		
96	13:35	THE GP130 RECEPTOR IS LOST DURING GRANULOPOIESIS, RENDERING NEUTROPHILS INERT TO IL-6 AND RELATED CYTOKINES. <u>WILKINSON AN</u> , GARTLAN KH, KELLY G, SAMSON LD, OLVER SD, AVERY Judy, ZOMERDIJK N, TEY SK, LEE JS, VUCKOVIC S, HILL GR	
97	13:50	DO INDIGENOUS PATIENTS HAVE BETTER SURVIVAL WITH A KIDNEY TRANSPLANT COMPARED TO STAYING ON DIALYSIS? A PROPENSITY MATCHED STUDY <u>LAWTON Paul</u> , CUNNINGHAM Joan, ZHAO Yuejen, Jose MATTHEW, CASS Alan	
98	14:05	IN VIVO REGULATORY T-CELL GENERATION WITH DENDRITIC CELL TARGETING NANOPARTICLES <u>STEAD SO</u> , KIRETA S, McINNES SJP, KETTE FD, SIVANATHAN K, KIM J, DROGEMULLER CJ, CARROLL RP, VOELCKER N, COATES PT	
99	14:20	RESIDUAL RISK OF BLOOD BORNE VIRUS INFECTION WHEN AUSTRALIAN ORGAN DONOR REFERRALS TEST NEGATIVE: A SYSTEMATIC REVIEW AND META-ANALYSIS. <u>WALLER Karen</u> , DE LA MATA Nicole, WYBURN Kate, KELLY Patrick, VIDIYA Ramachandran, RAWLINSON William, WEBSTER Angela	
100	14:35	UBIQUITIN LIGASE MARCH8 ATTENUATES GRAFT VERSUS HOST DISEASE VIA REGULATION OF GUT EPITHELIAL CELL SURFACE MHC II EXPRESSION <u>LINEBURG Katie E</u> , Le TEXIER Laetitia, MELINO Michelle, WANG Ran, BLAZAR Bruce R, MCGUCKIN Michael A, VILLADANGOS Jose A, MINTER Justine D, MACDONALD Kelli PA	
101	14:50	DONOR SPECIFIC ANTIBODIES AND CLINICAL OUTCOMES IN KIDNEY TRANSPLANT RECIPIENTS <u>WAN Susan</u> , CHADBAN Steven, WYBURN Kate	

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102	15:05	INTRA-RENAL DELIVERY OF DRUGS TAGETING ISCHEMIA-REPERFUSION INJURY OF THE KIDNEY IN A RODENT MODEL & PORCINE MODEL OF NORMOTHERMIC MACHINE PERFUSION <u>HAMEED Ahmer</u> , LU Bo, MIRAZIZ Ray, BURNS Heather, ROGERS Natasha, PLEASS Henry, HAWTHORNE Wayne	
103	15:20	FAVOURABLE CARDIAC REMODELING AND FUNCTIONAL CARDIAC BENEFITS ASSESSED WITH CARDIAC MAGNETIC RESONANCE IMAGING FOLLOWING LIGATION OF ARTERIOVENOUS FISTULA IN STABLE RENAL TRANSPLANT RECIPIENTS: A RANDOMIZED, CONTROLLED, OPEN LABEL STUDY <u>RAO Nitesh</u> , MCDONALD Stephen, WORTHLEY Matthew, COATES Patrick Toby	
15:35–16:00 Afternoon tea Foyer, Level 1, MCEC			
16:00–17:00	CONCURRENT FREE COMMUNICATIONS SESSIONS		
	Free Communications 7: Surgical <i>Chairs: Dr Amanda Robertson and Prof Henry Pleass</i>		Main Plenary – Rooms 106-105, Level 1, MCEC
Abstract	— <i>Oral presentations</i> —		
104	16:00	ENHANCED RECOVERY AFTER SURGERY AND THE RENAL TRANSPLANT RECIPIENT – USEFUL OR A WASTE OF TIME? <u>LAMBERT Virginia</u> , CHANDRA Abhilash, RUSSELL Christine, OLAKKENGIL Santosh, BHATTACHARJYA Shantanu	
105	16:15	TRANSITION FROM LAPAROSCOPY TO RETROPERITONEOSCOPY FOR LIVE DONOR NEPHRECTOMY - A CASE CONTROL STUDY <u>NG Zi Qin</u> , REA Alethea, HE Bulang	
106	16:30	EVALUATION OF RISK FACTORS FOR ENTERIC LEAKS FOLLOWING SIMULTANEOUS PANCREAS AND KIDNEY TRANSPLANTATION <u>HORT Amy</u> , SHAHRESTANI Sara, HITOS Kerry, ROBERTSON Paul, LAM Vincent, YUEN Lawrence, RYAN Brendan, DE ROO Ronald, HAWTHORNE Wayne J, PLEASS Henry	

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107	16:45	AORTIC VERSUS DUAL PERFUSION FOR RETRIEVAL OF THE DBD LIVER – AN ANALYSIS OF RECIPIENT OUTCOMES USING THE ANZ LIVER TRANSPLANT REGISTRY <u>HAMEED Ahmer</u> , PANG Tony, YOON Peter, BALDERSON Glenda, DE ROO Ronald, YUEN Lawrence, LAURENCE Jerome, LAM Vincent, CRAWFORD Michael, HAWTHORNE Wayne, PLEASS Henry	
16:00–17:00	Free Communications 8: Xenotransplantation <i>Chairs: Ms Evelyn Salvaris and Prof Peter Cowan</i>		Room 104, Level 1, MCEC
Abstract	— <i>Oral presentations</i> —		
108	16:00	GENETICALLY MODIFIED PORCINE NEONATAL ISLET XENOGRAFTS PROVIDE LONG-TERM FUNCTION IN BABOONS <u>HAWTHORNE Wayne</u> , CHEW YiVee, BURNS Heather, SALVARIS Evelyn, HAWKES Joanne, BRADY Jamie, BARLOW Helen, YI Shounan, HU Min, LEW Andrew, O'CONNELL Philip, NOTTLE Mark, COWAN Peter	
109	16:15	HUMAN HLA-DR+CD27+ MEMORY-TYPE REGULATORY T CELLS SHOW POTENT XENOANTIGEN-SPECIFIC SUPPRESSION IN VITRO <u>LU CAO</u> , HU Min, HUANG Dandan, MA Xiaoqian, SUN Lei, JIMENEZ-VERA Elvira, BURNS Heather, ZHAO Yuanfei, HAWTHORNE Wayne, YI Shounan, O'CONNELL Philip	
110	16:30	GENETIC MODIFICATION TO CONTROL THE LOCAL T CELL RESPONSE TO PIG ISLET XENOGRAFTS <u>SALVARIS Evelyn</u> , FISICARO Nella, VASSILIEV Ivan, McILFATRICK Stephen, BRADY Jamie, LEW Andrew, HAWTHORNE Wayne, NOTTLE Mark, COWAN Peter	
111	16:45	ENCAPSULATED PIG CELLS SECRETING ANTI-HUCD2 ANTIBODY REDUCES THE NUMBER OF HUMAN CD2 CELLS LOCALLY BUT NOT SYSTEMICALLY IN HUMANIZED MIC <u>LOUDOVARIS T</u> , COWAN P, HAWTHORNE W, SALVARIS E, FISICARO N, CATTERALL T, KOS C, MARIANA L, LEW A, KAY T	

Monday, April 30 2018

16:00–17:00	Free Communications 9: Organ Donation and Ethics #2 <i>Chairs: A/Prof David Goodman and Dr Vicki Levidiotis</i>		Room 103, Level 1, MCEC
Abstract	— Oral presentations —		
112	16:00	PROMOTING DECEASED DONOR ORGAN TRANSPLANTATION IN VIETNAM: WHERE TO START? <u>ALLEN Richard</u> , PLEASS Henry, KABLE Kathy, ROBERTSON Paul, MACKIE Fiona, THOMAS Gordon, NGOC Sinh Tran, KHANH Pham Gia, NGUYEN Truong Son	
113	16:15	THE WEEKEND EFFECT: AN AUSTRALIAN COHORT STUDY ANALYSING TEMPORAL TRENDS IN SOLID ORGAN DONATION <u>WEBSTER Angela</u> , HEDLEY James, CHANG Nicholas, ROSALES Brenda, WYBURN Kate, KELLY Patrick, OLEARY Michael, CAVAZZONI Elena	
114	16:30	FACTORS ASSOCIATED WITH TIME TO DECEASED DONOR RENAL TRANSPLANT WAITLISTING IN AUSTRALIA <u>SYPEK Matthew</u> , CLAYTON Phil, LIM Wai, HUGHES Peter, KANELIS John, WRIGHT Jenni, CHAPMAN Jeremy, MCDONALD Stephen	
115	16:45	ALLOCATION OF LOW-RISK KIDNEYS: CAN WE OPTIMISE UTILISATION? CLAYTON Phil, GULYANI Aarti, <u>SYPEK Matthew</u> , KANELIS JOHN, MCDONALD Stephen	
17:00–18:00	TSANZ Annual General Meeting		Main Plenary – Rooms 106-105 Level 1, MCEC
19:00–23:00	TSANZ Annual Dinner		Showtime Event Centre South Wharf, Melbourne

Tuesday, May 1 2018

07:30–08:00	Coffee with sponsors	Foyer, Level 1, MCEC
08:00–09:30	PLENARY 4: Novartis Symposium Immune Monitoring in Transplantation <i>Chairs: Prof Steve Chadban and Dr Kathy Paizis</i> 08:00 Tolerance in Kidney/Liver Transplantation Dr Sandy Feng 08:30 Qiagen QF Monitoring in Liver Transplantation Dr Adam Testro 08:50 Defining Optimal Immunosuppression Regimens Dr Katherine Barraclough 09:10 Machine-Learning Algorithms to Predict Graft Failure Dr Lawrence Lau	Main Plenary – Rooms 106-105 Level 1, MCEC
09:30–10:30	CONCURRENT STATE OF THE ART SESSIONS STATE OF THE ART 1: Novartis Symposium Women's Health Post Transplantation <i>Chairs: Dr Bronwyn Levvey and Professor Greg Snell</i> 09:30 Contraception and Pre-Pregnancy Management Basic Dr Paul de Crespigny 09:50 Pregnancy Outcomes and Impact of Pregnancy on Graft Function in Renal Allograft Recipients Dr Shilpa Jesudason 10:10 Menopause Management in Transplant Recipients Prof Martha Hickey	Main Plenary – Rooms 106-105 Level 1, MCEC

Tuesday, May 1 2018

09:30–10:30	STATE OF THE ART 2: Astellas Symposium Management of Post-Transplant Complications <i>Chairs: Dr Angela Webster and A/Prof Peter Hughes</i> 09:30 New Therapies for Management of Post-Transplant Diabetes Dr Chris Yates 09:50 Targeted Therapy for Post-Transplant Melanoma Dr Karen Sheppard 10:10 The Immune–Mechanisms Preventing CMV Disease; New Insights for Therapy Prof Maripia Degli-Esposti	Room 104 , Level 1, MCEC
10:30–11:00	Morning tea	Foyer, Level 1, MCEC

Tuesday, May 1 2018

11:00–12:30	CONCURRENT STATE OF THE ART SESSIONS STATE OF THE ART 3: Novartis Symposium Transplantation Surgical Session <i>Chairs: Dr Nancy Suh and Dr Graham Starkey</i> 11:00 Frailty Index to Predict Recipient Outcomes and Outcomes Following Combined Liver/Kidney Transplant Prof Sandy Feng 11:30 Pros and Cons of Using Neonatal and Paediatric Donor Organs Dr Amanda Robertson 11:50 Expansion of the DCD Programme –Manpower Issues Dr Rohit d’Costa 12:10 Aortic Pathology Post Heart Transplant-Redo Surgery Challenges Dr Emily Granger	Main Plenary – Rooms 106-105 Level 1, MCEC
11:00–12:30	STATE OF THE ART 4: Astellas Symposium Paired Kidney Exchange Program <i>Chairs: A/Prof Rosemary Masterson and Dr Darren Lee</i> 11:00 Non-Directed Living Kidney Donation – What Do We Know? A/Professor Alex Holmes 11:20 Paired Kidney Exchange-the Canadian Experience A/Professor Kathryn Tinckam 11:40 New Directions in PKE A/Professor Peter Hughes 12:00 An Update on Organ Trafficking and Declaration of Istanbul Professor Toby Coates 12:20 Questions	Room 104 , Level 1, MCEC
12:30–13:30	Lunch Cardiac Transplant Advisory Committee (CTAC)	Foyer, Level 1, MCEC Room 103, MCEC

Tuesday, May 1 2018

13:00–16:00	Liver and Intestinal Transplant Advisory Committee (LITAC)	Room 104, MCEC
13:30–15:00	PLENARY 5: Novartis Symposium What's new in Transplantation <i>Chairs: Prof Toby Coates and Prof Karen Dwyer</i> 13:30 Complement Inhibitors for Treatment of Antibody Rejection and Delayed Graft Function Dr Stanley Jordan 13:50 Treg Therapy for cGVHD A/Professor Kelli MacDonald 14:10 TIM Family Genes and Regulation of auto-and allo-Immunity Professor Vijay K Kuchroo 14:30 Clinical Trials in Kidney Transplantation: Resetting the Compass A/Professor Carmel Hawley 14:50 Questions	Main Plenary – Rooms 106-105 Level 1, MCEC
15:00–15:25	Afternoon tea	Foyer, Level 1, MCEC
15:25–16:00	The Great Debate: Australia Should Allow Public Solicitation of Organ Donors <i>Moderator: Prof Robyn Langham</i> Pro team: Prof Kathryn Tinckam/Prof Greg Snell Con team: Prof Sandy Feng/Dr Graham Starkey 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	Main Plenary – Rooms 106-105
16:00	ASM Concludes	

TSANZ ASM ABSTRACTS

IN SESSION ORDER

IRI and New Techniques

Abstract No. 1

ISOLATING ISLETS FOR TRANSPLANTATION USING AN ISOLATOR SYSTEM, A VIABLE ALTERNATIVE TO CONVENTIONAL CLEAN ROOM FACILITIES.

KOS C¹, LOUDOVARIS T, MARIANA L¹, MCCORMICK K², BLEASDALE N², IRVIN A¹, WAIBEL M¹, THOMAS H¹, KAY T¹

¹Immunology & Diabetes, St Vincent's Institute, Melbourne, ²Australian Red Cross Blood Service

Aims: Islet transplantation is now used as therapy for type 1 diabetic patients with severe hypoglycaemic unawareness. In Victoria, we previously isolated islets in a conventional cleanroom facility. An equivalent facility was established on the St Vincent's campus, in which processing occurs within an enclosed custom-built BioSpherix Xvivo biological system ('isolator') instead of open biosafety cabinets. By transferring this process to an isolator, we aimed to replicate our processes and procedures for equivalent and possibly improved islet yield outcomes.

Methods: The isolator contains three processing chambers, cell culture incubators, centrifuge and microscope modules. In contrast to conventional clean room facilities, islets processed in the isolator are maintained at optimal conditions in chambers capable of operating between 4°C and 45°C to maximize yields. Islets were isolated using a modified Ricordi method at both facilities.

Results: Isolations from 43 pancreata have been infused into patients in Melbourne, Adelaide and Sydney, with 22 of these processed using the isolator facility. Isolator Islet yields (280,103±173,108 islet equivalents, n=117) were significantly higher (p<0.0014*) than conventional cleanroom yields (207,870±139,962 islet equivalents, n=88) resulting in an 8% increase in the proportion transplanted.

Conclusions: We have replicated our cleanroom islet isolations using an isolator. Islet yields have increased overall, likely due to tight control of temperature and oxygen, as compared to the regulation of temperature with ice packs and water baths in the cleanroom process. The isolator facility provides a fully contained environment suitable for processing not only islets but also other human cells and tissue.

* Unpaired t-test

Abstract No. 2

ANALYTICAL AND CLINICAL VALIDATION OF A NEW, HIGHLY INFORMATIVE, SENSITIVE AND PRECISE METHOD FOR CELLULAR CHIMERISM ANALYSIS

WHITLAM John^{1,2,3,4}, **LING Ling**^{5,3}, **SWAIN Michael**⁶, **HARRINGTON Tom**^{2,3}, **MIROCHNIK Oksana**⁷, **BROOKS Ian**^{5,3}, **CRONIN Sara**^{5,3}, **CHALLIS Jackie**^{5,3}, **PETROVIC Vida**^{5,3}, **BRUNO Damien**^{5,3}, **MECHINAUD Francoise**⁸, **CONYERS Rachel**⁸, **SLATER Howard**^{5,3,9}

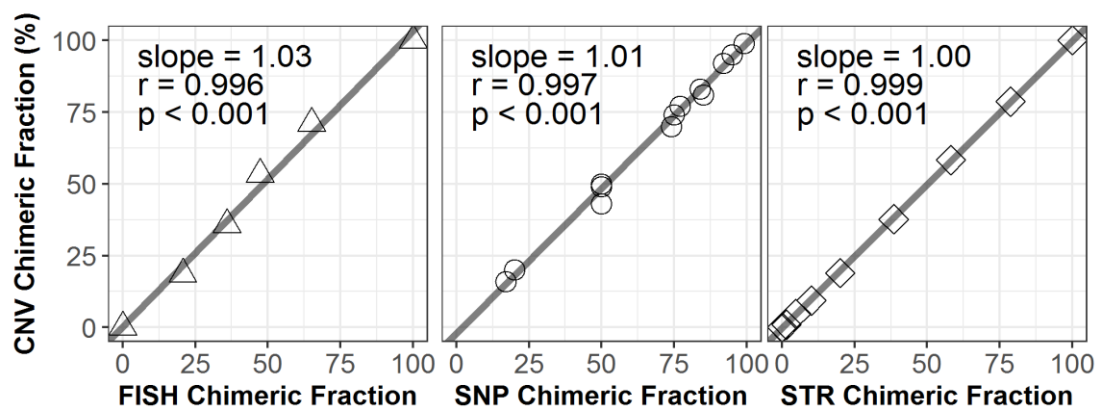
¹Department of Nephrology, Austin Hospital, Melbourne, ²Victorian Clinical Genetics Services, ³Murdoch Childrens Research Institute, Royal Children's Hospital, Melbourne, ⁴Department of Medicine, University of Melbourne, ⁵Victorian Clinical Genetics Services, Royal Children's Hospital, Melbourne, ⁶Laboratory Services, Royal Children's Hospital, Melbourne, ⁷Pathology West Institute of Clinical Pathology and Medical Research, Westmead Hospital, Sydney, ⁸Children's Cancer Centre, Royal Children's Hospital, Melbourne, ⁹Department of Pediatrics, University of Melbourne

Aims: To analytically and clinically validate a new droplet digital PCR-based method of cellular chimerism analysis exploiting ubiquitous, highly heterozygous copy number variants (CNV).

Methods: A panel of 38 CNV assays were developed targeting CNV loci with homozygous deletion frequencies of 0.4-0.6. Donor-recipient informativity was assessed in 39 recipients and 43 donors of allogeneic bone marrow transplants at the Royal Children's Hospital, Melbourne. Sensitivity, precision, reproducibility and informativity were tested. CNV chimerism analysis was compared against established fluorescence in situ hybridization, single nucleotide polymorphism, and short tandem repeat-based methods, including presently established clinical methods at laboratories across Australia.

Results: High informativity was seen with a median of four informative markers detectable per individual in a transplant arrangement. Using 30ng input DNA per well, limit of detection was 0.05% and limit of quantification was 0.5% chimerism. Chimerism result coefficient of variation in reproducibility studies was $\leq 2\%$. CNV chimerism analysis exceeded or matched performance of all current methods studied (Figure) and all Australian laboratories participating in the RCPA quality assurance program (N=21).

Conclusions: The precision, sensitivity and informativity of CNV-based cellular chimerism analysis recommend it for use in clinical practice after allogeneic bone marrow transplantation. Additional applications include monitoring maternal engraftment in infants with severe combined immunodeficiency, and monitoring homing and survival in emerging stem cell and cytotoxic T cell-based therapies. Platform performance at extreme low level chimerism can be improved with addition of greater quantities of DNA to the reaction.



Abstract No. 3**CYCLOPHILIN BLOCKADE PROTECTS FROM RENAL ISCHAEMIA/REPERFUSION INJURY****LEONG Khai Gene^{1,2}, OZOLS Elyce^{1,2}, KANELIS John^{1,2}, LILES John³, NIKOLIC-PATERSON David^{1,4}, MA Frank^{1,2}**¹*Department of Nephrology, Monash Medical Centre, Melbourne,* ²*Centre for Inflammatory Diseases, Monash University, Melbourne,* ³*Other, Gilead Sciences,* ⁴*Other, Monash University, Melbourne*

Cyclophilins are proteins that regulate protein folding. During pathological conditions, cyclophilin A (CypA) is an important pro-inflammatory molecule, while CypD facilitates mitochondrial-dependent cell death.

Aims: (1) Investigate whether a novel pan-cyclophilin inhibitor (CYPi), which does not block calcineurin function, can prevent anticipated renal ischaemia/reperfusion injury (IRI); (2) Assess the contribution of CypA in renal IRI.

Methods: Groups of 10 mice underwent bilateral renal ischaemia and were killed 24hr after reperfusion. Controls were sham operated. Study 1: C57BL/6J mice were treated with CYPi (30mg/kg/BID) or vehicle by oral gavage. Study 2: CypA^{-/-} versus wild type (WT) mice on the 129 background. Effects of renal IRI were compared.

Results: Study 1: Renal IRI caused acute kidney injury (AKI) in C57BL/6J mice (179.0±19.1 vs 12.2±1.4µmol/L serum creatinine (sCr) in sham; P<0.001). CYPi protected against AKI (sCr 36.4±6.7µmol/L; P<0.001) and reduced the histologic tubular damage score (P<0.001). CYPi reduced apoptotic tubular cells, TNF-α mRNA levels and neutrophil and macrophage infiltration (all P<0.01 vs vehicle). Study 2: Renal IRI induced AKI in 129 mice (sCr 41±13.76 vs 6.25±1.66µmol/L in sham; P<0.0001). CypA^{-/-} mice were protected from renal dysfunction (sCr 20.7±3.53µmol/L; P<0.001). CypA^{-/-} mice had less histologic tubular damage (P<0.01) and lower KIM-1 mRNA levels (P<0.01). CypA^{-/-} mice also had less tubular cell death (TUNEL+ cells), inflammatory cytokines (TNF-α and IL-36-α PCR; P<0.05), and reduced neutrophil infiltration (P<0.001).

Conclusions: Pharmaceutical pan-cyclophilin inhibition prevents anticipated IRI-induced AKI by suppressing tubular cell death and inflammation. Based on knockout studies, CypA specifically contributes to inflammation in renal IRI.

Abstract No. 4**ACTIVATED CD47 PROMOTES ACUTE KIDNEY INJURY BY LIMITING AUTOPHAGY****EL RASHID Mary, SANGANERIA Barkha, ROGERS Natasha M***Westmead Institute for Medical Research*

Background: Acute kidney injury (AKI) initiates a complex pathophysiological cascade leading to epithelial cell death. Recent studies identify autophagy, the mechanism of intracellular degradation of cytoplasmic constituents, as important in protection against injury. We have reported that the protein thrombospondin-1 (TSP1), and its receptor CD47, are induced in AKI, however their role in regulating renal injury is unknown.

Methods: Age and gender-matched wild-type (WT) and CD47^{-/-} mice were challenged with renal ischemia reperfusion injury. All animals underwent analysis of renal function and biomolecular phenotyping. Human and murine WT and CD47^{-/-} renal tubular epithelial cells (rTEC) were studied *in vitro*.

Results: CD47^{-/-} mice were resistant to AKI, with decreased serum creatinine, and ameliorated histological changes compared to WT animals. CD47^{-/-} mice demonstrated concurrent upregulation of key autophagy genes, including Atg5, Atg7, Beclin-1, and LC3 at baseline and post-AKI. WT mice demonstrated negligible autophagy expression at all time points. rTEC from CD47^{-/-} mice displayed basal upregulation of autophagy that was preserved under hypoxic stress, and correlated with enhanced viability when compared to WT cells. Treatment of WT rTEC with a CD47 antagonist antibody or oligonucleotide to block TSP1-CD47 signalling increased autophagy. Finally, in a syngeneic mouse kidney transplantation model, treatment with a CD47 blocking antibody improved renal function and decreased histologic damage compared to control mice, and this was associated with increased autophagy.

Conclusions: These data suggest activated CD47 is a proximate promoter of AKI through inhibition of autophagy, and point to CD47 as a target to restore renal function following injury.

Transplant Complications

Abstract No. 5

THE EFFECT OF POST-TRANSPLANT LYMPHOPROLIFERATIVE DISEASE (PTLD) ON GRAFT AND PATIENT SURVIVAL IN KIDNEY TRANSPLANT RECIPIENTS

FRANCIS Anna¹, CRAIG Jonathan¹, JOHNSON David², WONG Germaine¹

¹Centre for Kidney Research, University of Sydney, ²Renal & Transplantation Unit, University of Queensland at the Princess Alexandra Hospital

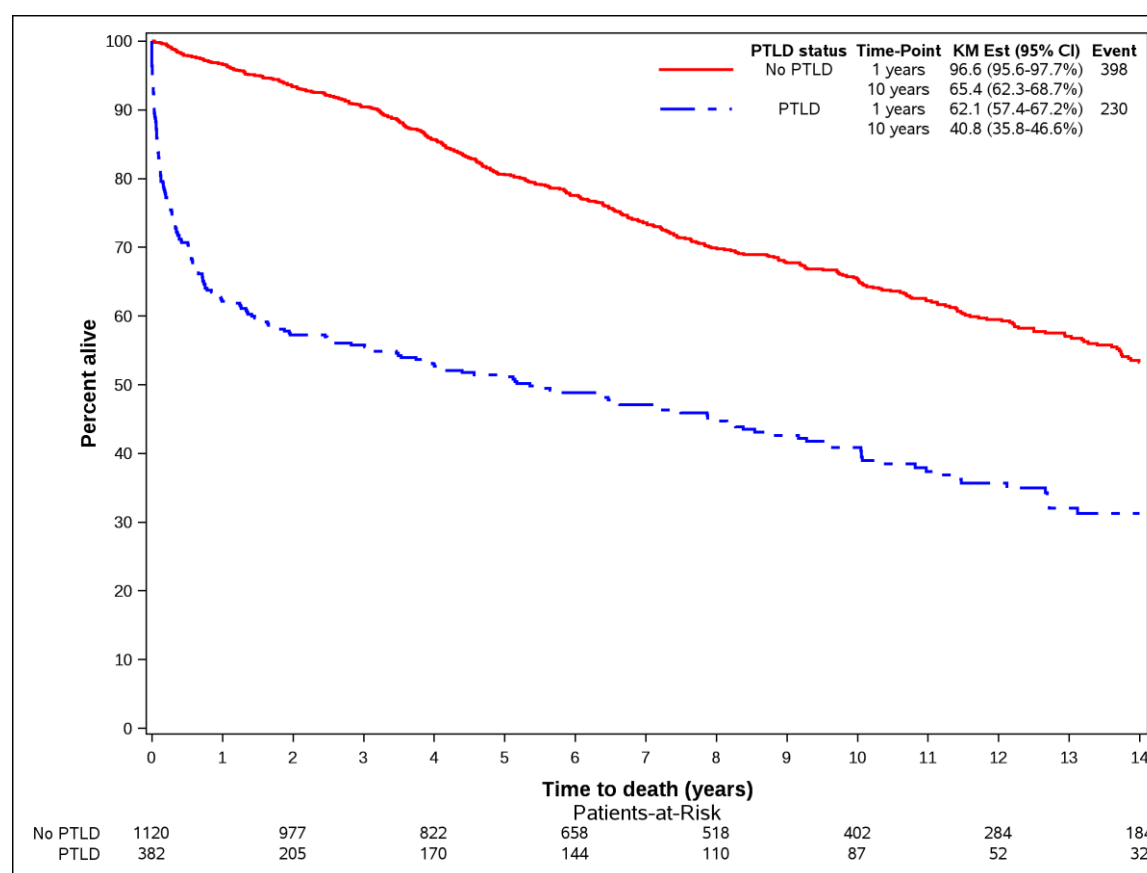
Aim The aim was to estimate the excess risk of death and graft loss in kidney transplant recipients due to PTLD, and to determine risk factors for death.

Methods Patients with PTLD in their first transplant (1990-2015) were identified from ANZDATA and matched to three controls. The risks of mortality and graft loss (with competing risk of death) were estimated using survival analysis and Cox models explored risk factors for death after PTLD.

Results There were 395 patients with PTLD, of which 382 cases (68% male, 88% Caucasian, mean age 43 years) were matched to 1120 controls (58% male, 87% Caucasian, mean age 43 years). Mean follow up was 7.6 years (SD 5.8 years). 10-year survival (95%CI) was 40.8% (35.8%-46.6%) among recipients with PTLD compared to those without (65.4%, 62.3%-68.7%). (Figure 1) The excess mortality was all in the first year (HR 14.4, 95%CI 10.0-20.6), with no difference in mortality after 1 year (HR 1.17, 95%CI 0.92-1.47). The 10-year graft loss (95%CI) was similar for those with and without PTLD [16.4% (12.8%-21.1%) vs. 20.5% (18.0%-23.4%)]. Increasing age at diagnosis (per 10 years increased) [(adjusted HR:95%CI) 1.46:1.32-1.63], site of disease (brain compared to nodal) [1.98:1.28-3.06] and diagnosis before the year 2000 [2.55:1.59-4.09] were associated with an increased risk of mortality after PTLD.

Conclusions PTLD increased the risk of mortality 14-fold in the first year after diagnosis only, with no effect on graft loss unrelated to death. PTLD site, increased age and diagnosis in earlier era were associated with increased mortality risk.

Figure 1. Patient survival, stratified by PTLD status



Abstract No. 6

DE NOVO OR EARLY CONVERSION TO EVEROLIMUS AND LONG-TERM CANCER OUTCOMES IN KIDNEY TRANSPLANT RECIPIENTS: A TRIAL-BASED ANZDATA LINKAGE STUDY

YING Tracey^{1,2}, **WONG G**³, **LIM W**⁴, **RUSS G**⁵, **PILMORE H**⁶, **KANELIS J**⁷, **GOODMAN D**⁸, **TREVILLIAN P**⁹, **CAMPBELL S**¹⁰, **SURANYI M**¹¹, **MATHEW M**, **FAULL R**¹², **MASTERSON R**¹³, **WALKER R**¹⁴, **O'CONNELL P**¹⁵, **CHADBAN S**^{1,16}

¹Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney, ²Charles Perkins Centre, University of Sydney, ³Centre for Kidney Research, University of Sydney, ⁴Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth ⁵Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, ⁶Nephrology Department, Auckland City Hospital, Auckland, New Zealand, ⁷Department of Nephrology, Monash Medical Centre, Melbourne, ⁸Department of Nephrology, St Vincent's Hospital Melbourne, ⁹Newcastle Transplant Unit, John Hunter Hospital, New Lambton Heights, ¹⁰Nephrology Department, Princess Alexandra Hospital, ¹¹Liverpool Renal Clinical Research Centre, ¹²Renal Unit, Royal Adelaide Hospital, ¹³Department of Nephrology, Royal Melbourne Hospital, ¹⁴Department of Renal Medicine, Alfred Health, ¹⁵Department of Renal Medicine, Westmead Hospital, ¹⁶Sydney Medical School, University of Sydney

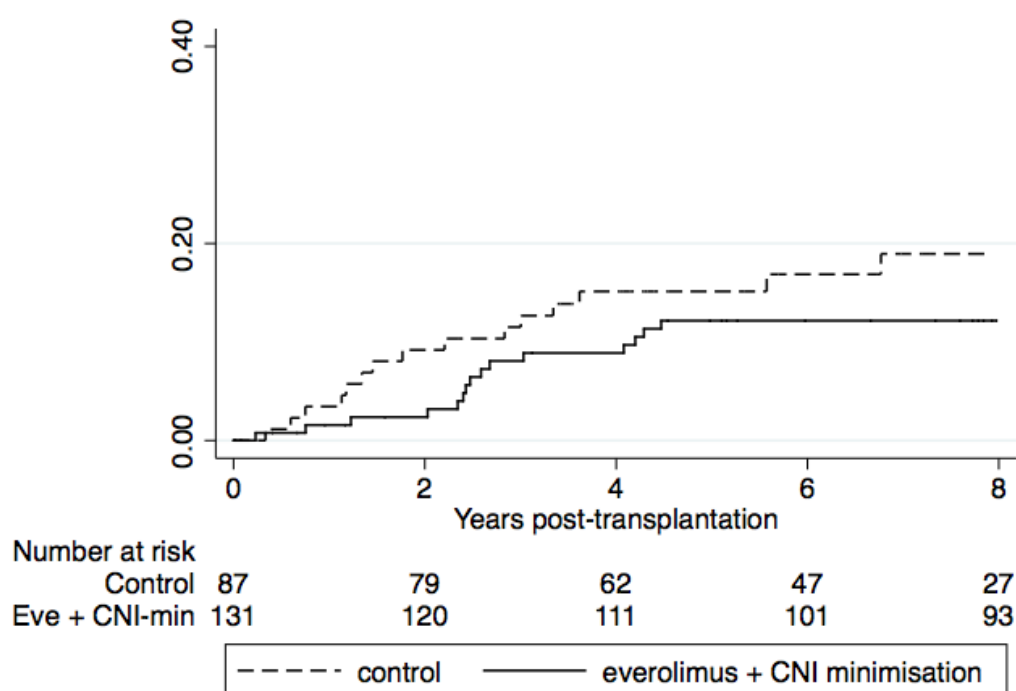
Aim: To determine the long-term risks of incident cancer, non-melanoma skin cancer (NMSC) and cancer-related death in kidney transplant recipients randomised to everolimus (EVL), *de novo* or early-switch, compared with controls on calcineurin inhibitor-based triple therapy.

Methods: Australian and NZ participants from *de novo* or early-switch trials were linked to ANZDATA for the outcomes of incident cancer (including NMSC), first NMSC and cancer-death. Adjusted random effect Cox regression models were constructed to examine the association between treatment type and cancer outcomes with sub-analysis stratified into EVL+CNI minimisation (CNI-min) and EVL+CNI-withdrawal (CNI-WD).

Results: A total of 279 patients were followed for a median of 9 years (IQR 6.8, 9.7). Of 192 EVL patients, 131 were randomised to EVL+CNI-min and 61 to EVL+CNI-WD. The cumulative incidence of any cancer was similar between EVL and control (log-rank $p=0.8$). Thirty (15.6%) EVL patients developed NMSC (16 SCC/14 BCC) vs. 17 (19.5%) in the controls (8 SCC/9 BCC) ($p=0.4$). The risk of NMSC was reduced by 55% in the EVL+CNI-min arm (adjHR 0.45, 95% CI 0.21–0.94) vs. controls (Figure 1), however, no difference was detected in the CNI-WD-subgroup (adjHR 0.98, 95% CI 0.42–2.27). Of 39 deaths (EVL=30, control=9), 12 were attributed to cancer. The risk of cancer-death for EVL patients was similar to controls (adjHR 1.74, 95% CI 0.44–6.83).

Conclusion: *De novo* or switch to EVL did not alter long-term cancer outcomes including any cancer or cancer-related deaths. However, the EVL+CNI-min strategy may reduce the risk of incident NMSC.

Figure 1 – Unadjusted Kaplan-Meier curve of non-melanoma skin cancer by treatment subgroup



Abstract No. 7

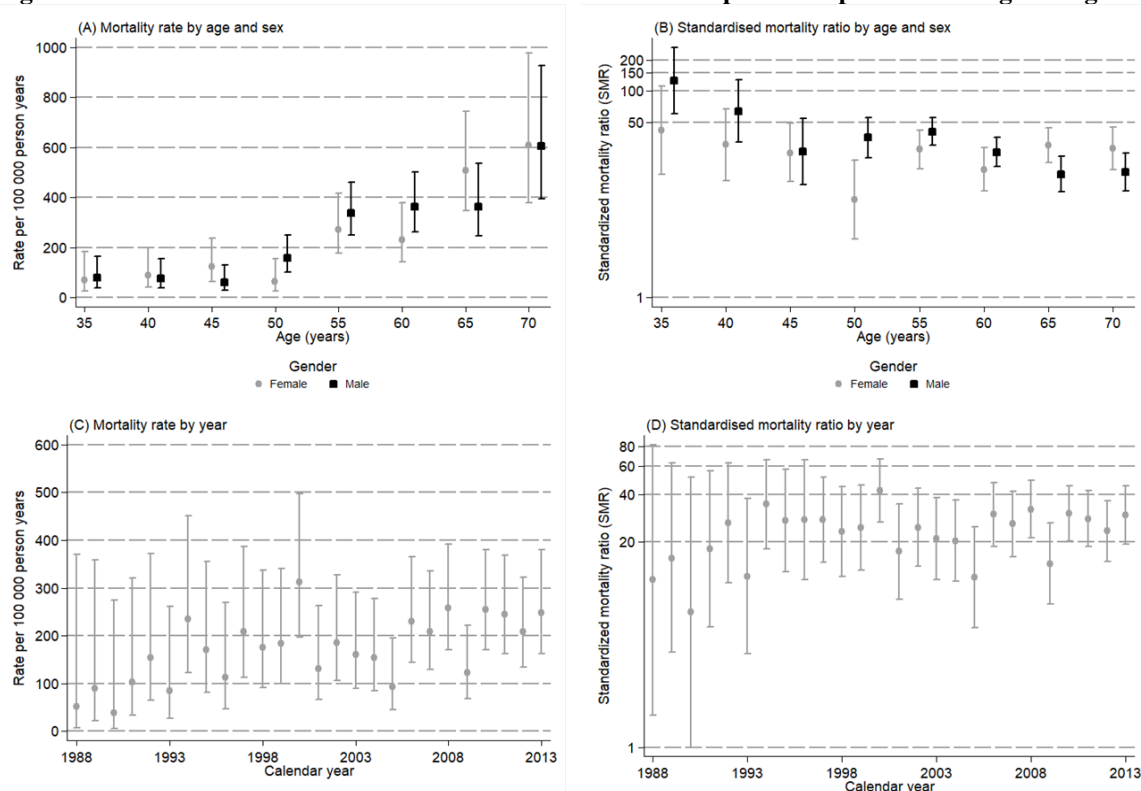
CANCER MORTALITY IN KIDNEY TRANSPLANT RECIPIENTS IN AUSTRALIA AND NEW ZEALAND: A COHORT STUDY FROM 1980 TO 2013.**ROSALES Brenda¹, DE LA MATA Nicole¹, KELLY Patrick¹, WEBSTER Angela^{1,2,3}**¹Sydney School of Public Health, University of Sydney, ²Centre for Kidney Research, The Children's Hospital at Westmead, Sydney, ³Nephrology and Renal Transplant, Westmead Hospital, Sydney

Aims: International guidelines from 2009 suggest post-transplant screening for specific cancers, but any impact on recipient mortality for screened-cancers remains unclear. We compared death from all cancer and screened cancers for kidney transplant recipients versus the general population in Australia and New Zealand.

Methods: We conducted a population-based cohort study, using ANZDATA linked with Australian and New Zealand death registries, in incident kidney transplant recipients from 1980-2013. Cancers were categorised using ICD-10-AM codes. Standardised mortality ratios (SMR) were estimated using indirect standardisation.

Results: We included 17,621 recipients with 160,332 person-years (pys) of follow-up. Of 5,284 deaths, 1,063 (20.1%) were from cancer. Of cancer deaths, 293 (27.6%) were from screened-cancers, including: 75 colorectal, 72 renal, 69 melanoma, 26 breast, 26 liver, 19 prostate and 6 cervical. Cancer-related mortality rate was 663 per 100,000 pys (95%CI 624-704), and higher in men (727 per 100,000 pys; 95%CI 675-783). Transplant recipients were >3 times more likely to die cancer deaths (SMR 3.2; 95%CI 3.03-3.4) compared to the general population. Screened-cancer mortality rates increased with age (Figure 1A). Relative mortality (SMR) decreased in men, as age increased ($p < 0.001$), however was unchanged for women ($p = 0.4$) (Figure 1B). Overall, screened-cancer deaths increased over time since 1980 (Figure 1C), however SMR remained steady ($p = 0.2$) (Figure 1D).

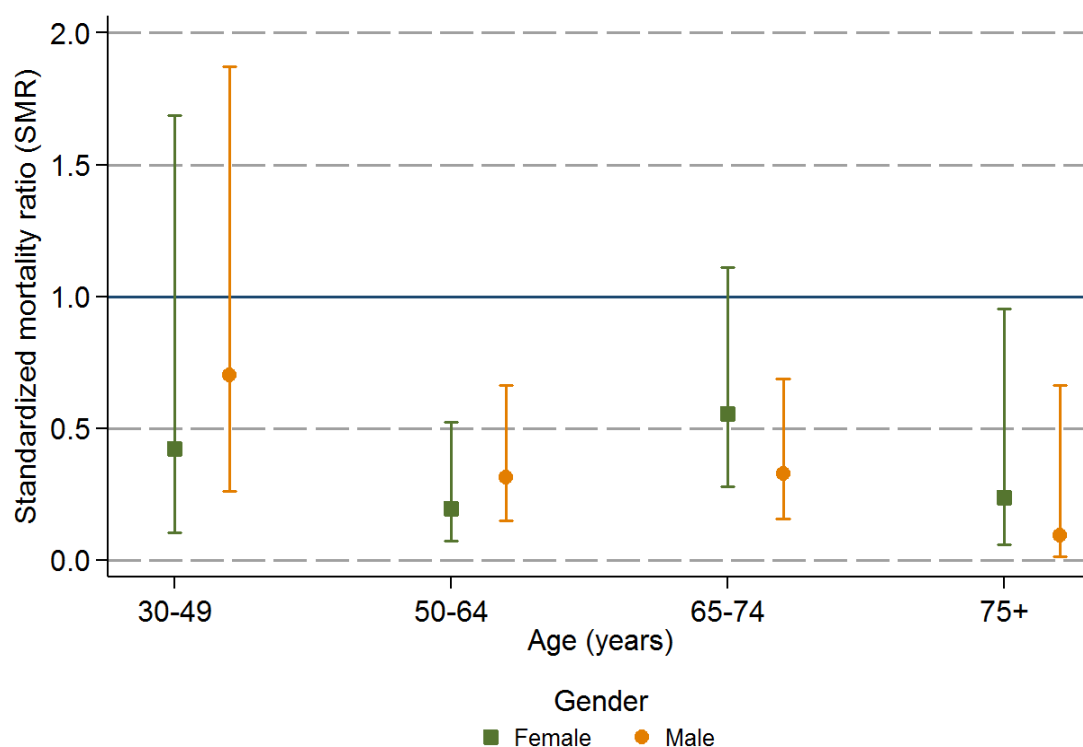
Conclusions: Kidney transplant recipients are at increased risk of cancer death. Cancer death rates are increased over time but SMR remained stable suggesting similar changes in the general population. There was no evidence of impact of screening on mortality for kidney recipients.

Figure 1: Cancer related deaths for cancers with recommended post-transplant screening strategies

Abstract No. 8

MORTALITY RATES IN LIVING KIDNEY DONORS: AN AUSTRALIAN AND NEW ZEALAND COHORT STUDY USING DATA LINKAGE**DE LA MATA Nicole¹, CLAYTON Philip^{2,3}, MCDONALD Stephen^{4,5,2}, CHADBAN Steven^{2,6,7}, POLKINGHORN Kevan^{8,9}, WEBSTER Angela^{1,10}**¹*Sydney School of Public Health, University of Sydney*, ²*ANZDATA*, ³*Faculty of Health Sciences, University of Adelaide*, ⁴*Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital*, ⁵*Department of Medicine, University of Adelaide*, ⁶*Transplantation Services, Royal Prince Alfred Hospital, Sydney*, ⁷*Sydney Medical School, University of Sydney*, ⁸*Department of Epidemiology and Preventative Medicine, Monash University, Melbourne*, ⁹*Department of Nephrology, Monash Medical Centre, Melbourne*, ¹⁰*Centre for Transplant and Renal Research, Westmead Hospital, Sydney*

Aims: Living kidney donors are a highly selected group whom could be expected to have better than average life expectancy. We aimed to compare deaths in living kidney donors with the general population. **Methods:** We included all living donors in Australia and New Zealand from 1996 to 2013. Where dead, we established the primary cause of death using data linkage between the Australian and New Zealand Living Kidney Donor Registry and national death registries: Australia, 1996-2013 and New Zealand, 2003-2013. Standardized mortality ratios (SMR) were estimated using indirect standardization. **Results:** Among 3,374 living kidney donors, there were 35 deaths in 22,551 person-years (pys) of follow-up. The most common cause of death was cancer (n=19), followed by coronary heart disease (n=3) and accidental deaths (n=3). Donors who had died were generally older than those still alive, having a median age of 61 years [IQR: 57-64]. The crude mortality rate during the first year from donation was 148 (95% CI: 62-357) per 100,000 pys and increased to 187 (95% CI: 70-498) per 100,000 pys at 5 years since donation. The overall SMR was 0.32 (95% CI: 0.23-0.45), where living kidney donors had 68% fewer deaths than expected in the general population of the same age and sex. There were few differences in SMR by sex or age (Fig. 1). **Conclusion:** All-cause mortality was significantly lower among living kidney donors compared to the general population, with no evidence of increased deaths from any cause

Figure 1. The estimated standardized mortality ratios (SMR) for all-cause deaths, by age and sex.

Regulatory T cells

Abstract No. 9

ADMINISTRATING IL2 COMPLEX IN THE PRESENCE OF ALLOANTIGEN COMBINED WITH DONOR CELL TRANSFUSION EXPANDS ALLO-SPECIFIC TREGS FACILITATING TOLERANCE INDUCTION ACROSS A RESTRICTED MAJOR MISMATCH IN A SKIN GRAFT MODEL

ZHANG Geoff Y¹, WANG Yuan Min¹, HU Min², KARUNIA Jevin¹, GREY Shane³, ALEXANDER Stephen I¹

¹Centre for Kidney Research, Children's Hospital at Westmead, ²Centre for Transplant and Renal Research, Westmead Hospital, Sydney, ³Transplant Immunobiology Laboratory, Garvan Institute of Medical Research, Sydney

Background: *In vivo* Treg expansion using IL2 complexed with anti-IL2 antibody (IL2 complex) has been demonstrated to be effective in the induction of long-term acceptance of islet allografts. In this study, we investigated the effects of administration of IL2 complex in donor specific transfusion (DST) skin allograft model with restricted MHC class I disparity.

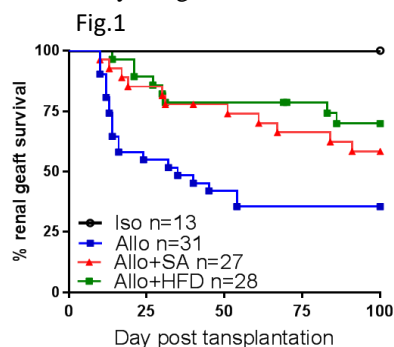
Method: B6 received IL2 complex in combination with transfusions from Bm1 splenocytes followed by Bm1 skin grafting. The effect of IL2 complex plus DST were compared with IL-2 complex only, DST only and non-treatment groups. Graft survival, histology, mixed lymphocyte reaction and IFN- γ ELISPOT, *in vitro* Treg suppression and Foxp3 Treg TCR Vbeta CDR3 spectrotypes were assessed.

Results: Injection of IL2 complex induced a 7.5 fold increase of Foxp3 Tregs in peripheral blood at Day4 after injection. Despite the substantial expansion in numbers of Tregs, B6 acutely rejected Bm1 skin grafts at a similar rate as the no IL2 complex control group (MST=22 vs MST=14). DST using Bm1 splenocytes prolonged survival of subsequent skin grafting but failed to induce long-term graft acceptance (MST=50). In contrast, administration of IL2 complex before DST rendered long-term graft acceptance (MST=100 days).

Cells from mice received IL2 complex plus DST group showed significantly reduced responses to Bm1 stimulators in MLRs while maintaining equivalent responses to allogeneic third party stimulators. IFN- γ ELISPOT showed a similar pattern of response as in the MLR. In *in vitro* suppression assays, Foxp3 Tregs expanded with IL2 complex in combination of DST were more potent in suppressing antigen-specific reactions than Foxp3 Tregs from IL2 complex only group. To investigate whether Treg expansion in the presence of alloantigen has a restricted Treg TCR repertoire, proliferation dye stained gfp-Foxp3 Tregs were FACS sorted into populations of dividing and non-dividing and more restricted TCR Vbeta CDR3 usage were found in dividing Tregs in comparison with non-dividing Foxp3 Tregs, as well as Foxp3 Tregs expanded by IL2 complex alone.

Conclusion: IL-2 complex in combination with DST, when given prior to DST, leads long term tolerance. The IL-2 complex and the DST are essential for the development of tolerance as treatment of either IL2 complex or DST alone resulted in rejection. Administering IL2 complex in the presence of alloantigen provided by donor cell transfusion leads to expanded allo-specific Tregs, facilitating tolerance induction.

Abstract No. 10

HIGH FIBRE DIET INDUCES DONOR SPECIFIC TOLERANCE OF KIDNEY ALLOGRAFT THROUGH SHORT CHAIN FATTY ACID INDUCTION OF TREGS**WU Huiling^{1,2}, KWAN Tony², LOH Yik Wen², WANG Chuanmin^{1,2}, MACIA Lanrence³, ALEXANDER Stephen⁴, CHADBAN Steven^{1,2}**¹*Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney,* ²*Kidney Node Lab, The Charles Perkins Centre, University of Sydney,* ³*Nutritional Immunometabolism Lab, The Charles Perkins Centre, University of Sydney,* ⁴*Department of Nephrology, The Children's Hospital at Westmead, Sydney***Aim:** To investigate the impact of high fibre diet(HFD) or dietary supplementation with sodium acetate(SA) on kidney allograft rejection and survival in mice.**Methods:** Life sustaining kidney transplants were performed: B6 to B6 isografts and BALB/c to B6 WT or B6 GPR43^{-/-} mice as allografts. Mice were fed HFD for two weeks prior and throughout experiments(Allo+HFD), or received SA 200mg/kg ip for 14 days post-transplantation then SA 150mM solution orally(Allo+SA; GPR43^{-/-}+SA). Allograft controls received normal chow only(Allo). To deplete CD4+CD25+ cells, selected groups received anti-CD25mAb(PC61).**Results:** HFD preserved renal allograft function and prolonged allograft survival compared to control-allografts(Figure 1 p<0.01). HFD increased the release of SCFAs, particularly acetate. Similarly, Allo+SA allografts were protected from both acute and chronic allograft rejection with better renal function(p<0.05), less tubulitis(p<0.001) and increased CD4+Foxp3+ Treg accumulation at day14 post-transplant, and improved renal function(p<0.05) and less proteinuria(p<0.001) at day 100 post-transplant versus control-allografts. Allo+SA allografts exhibited superior survival to control-allografts(Fig.1, p<0.05) due to the development of donor antigen specific tolerance, confirmed by acceptance of donor strain but rejection of 3rd party skin grafts. The survival benefit conferred by SA was broken by depletion of CD25⁺ Tregs(p<0.05). SA treatment was ineffective in GPR43^{-/-} allograft recipients(GPR43^{-/-}+SA, p<0.05). **Conclusions:** HFD or supplementation with SA induced donor specific kidney allograft tolerance in a fully MHC mismatched murine model of kidney allograft rejection. Tolerance was dependent on a CD4+CD25+FoxP3+ regulatory mechanism. GPR43 is required for the molecular action of SA induced donor specific tolerance of kidney allografts.

Abstract No. 11

IN-VIVO COSTIMULATION-BLOCKADE INDUCED LONG-TERM FOXP3+ REGULATORY T CELLS WITH MARKERS OF CD4+GFP+CD44+CD127^{hi}CD62L- DEMONSTRATE THE ANTIGEN-SPECIFIC POTENCY FROM IMMUNODEFICIENT MICE WITH NEONATAL ISLET CELL CLUSTERS TOLERANT XENOGRAFTS**ZHAO YUANFEI^{1,2}, J'HAWTHORNE WAYNE^{1,3}, BURNS HEATHER¹, QIAN YIWEN^{1,2}, ALEXANDER STEPHEN⁴, ZHANG GEOFF⁴, YI SHOUNAN¹, HU MIN¹, O'CONNELL PHILIP^{1,2}**¹Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney,²Department of Medicine, University of Sydney, ³Department of Surgery, University of Sydney, ⁴Centre for Kidney Research, The Children's Hospital at Westmead, Sydney

Aims: 1) To define xeno-antigen-specific Tregs and memory-Tregs phenotypically in the porcine-neonatal-islet-cell-cluster(NICCs) xenograft-tolerant model. 2) To assess their function in vivo.

Methods: Tolerance to NICC-xenografts in C57BL/6-GFP⁺Foxp3⁺ mice(CD45.2) was induced using CTLA-4Fc/MR-1. The phenotype of Foxp3⁺Tregs were analyzed by FACS. CD4⁺GFP⁺Foxp3⁺Tregs were sorted from spleens of mice with tolerant-xenografts(day100) (tolerant-Tregs), rejecting-xenografts(day21) (rejected-Tregs), and non-transplanted(naïve-Tregs). CD44^{hi}CD127^{hi}CD62L⁺Foxp3⁺Tregs were sorted from tolerant-Tregs. Rag^{-/-} mice were transplanted with NICCs, then adoptive-transferred with these Tregs at day22-Post-transplantation, further challenged with effector-cells(CD4⁺GFP⁺CD45.1⁺) day22 after adoptive-transfer(day44-Post-transplantation) at ratio of 1:3.

Results: Phenotypically, upregulations of CD127, CD44, MHC-II, CD39 and downregulation of CD25, CD27 and CD62L occurred in tolerant-Tregs of C57BL/6-GFP⁺Foxp3⁺ mice with tolerant NICC-xenografts at day100-post-transplantation, compared to rejected-Tregs(day100-post-transplantation) and naïve-Tregs. Specifically, the proportion of CD127 expression in tolerant-Tregs increased significantly to 80.9±17.0%, compared with 6.6±1.4% in rejected-Tregs and 4.2% in naïve-Tregs. In Rag^{-/-} mice at day 22-25 after adoptive-transfer, all types of Foxp3⁺Tregs(CD45.2⁺) including CD44^{hi}CD127^{hi}CD62L⁺Foxp3⁺Tregs, tolerant-Tregs, rejected-Tregs and naïve-Tregs groups were detected. Moreover, both Foxp3⁺Tregs(CD45.2) and effector CD4⁺GFP⁺(CD45.1) cells existed in Rag^{-/-} mice (1:3 ratio) at day 66 after challenge. Furthermore, porcine-C-peptide was detected in serum of Rag^{-/-} with CD44^{hi}CD127^{hi}CD62L⁺Tregs (355.0±182.3pmol/L) and tolerant-Tregs (64.5 pmol/L) at day 75 after challenge, but not in Rag^{-/-} with naïve-Tregs, rejected-Tregs, and control effector cell only. This demonstrated CD44^{hi}CD127^{hi}CD62L⁺Tregs had more potent and specific suppression function compared to tolerant-Tregs, rejected-Tregs and naïve-Tregs. It also suggested CD44^{hi}CD127^{hi}CD62L⁺Tregs may be the memory-Tregs.

Conclusions: Highly-selected CD44^{hi}CD127^{hi}CD62L⁺Foxp3⁺ tolerant-Tregs were capable of transferring dominant and specific tolerance, and may be the memory-Tregs.

Abstract No. 12

IN VITRO EVALUATION OF HUMAN REGULATORY T-CELLS IN A 3D-PRINTED STRUCTURE

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Introduction: 3D bioprinting allows for the fabrication of complex 3D architectures. 3D bioprinting of regulatory T-cells (Tregs) with islets may overcome inherent immunosuppressive failings in islet transplantation. This project aims to investigate the viability and functionality of bioprinted Tregs and to evaluate the effect of hydrogel modification with IL-2. **Method:** Natural Tregs (nTregs) were isolated from human blood by FACS. Induced Tregs (iTregs) were induced from naïve CD4⁺ T-cells. These cells were either suspended in media ('non-printed'), or printed in a disc structure with an alginate-gelMA hydrogel then photo- (400nm) and chemically crosslinked with CaCl₂. These 'printed' cells were recovered by enzymatically dissolving the discs. Viability and Treg functional markers were quantified by flow-cytometry using propidium iodide and anti-LAP, CD69, CD39 and CTLA-4 antibodies. **Results:** At day 1, the viability of nTregs decreased by 7% (p<0.0001) and 9% (p<0.0001) while iTreg viability decreased by 4% (p=0.0042) and 6% (p<0.0001), with and without IL-2 respectively, compared to non-printed controls. At day 3, modification with IL-2 significantly improved viability of printed Tregs by 15% (nTreg, p=0.003) and 29% (iTreg, p<0.0001). Furthermore, no decrease in LAP, CD69, CD39 or CTLA-4 expression was observed upon printing. **Conclusion:** Firstly, our data suggests Tregs can be safely bioprinted with minimal impact on viability or functional marker expression. Secondly, we demonstrate that hydrogel modification with IL-2 has a positive impact on the survival of bio-printed Tregs. Finally, this study serves as proof of principle for the capacity of immune cells to survive within printed hydrogel constructs.

Organ Donation and Ethics#1

Abstract No. 13

CHARACTERISING FAMILY REFUSALS IN SOLID ORGAN DONATION (CREDO) STUDY
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Background: Understanding the reasons for and characteristics that differentiate between families that consent and refuse organ donation may provide insights into strategies to reduce family refusals.

Aim: To evaluate the characteristics of organ donor referrals whose family refused consent to donation.

Methods: We included all solid organ donor referrals captured by the NSW Organ and Tissue Donation Service (OTDS), 2010-2016. We used descriptive statistics to summarise and compare characteristics of organ donors according to family consent status. Characteristics included donor age, sex, religion, socioeconomic background, ethnicity, reason for family refusal and referring hospital.

Results: There were 3,824 organ donor referrals, consent was sought for 1,927 referrals (Table 1). Nearly half of those where family consent was not sought were aged 65 years or older. Family consent was refused for 831 referrals, the most common reason was the family believing the patient did not want to donate (n=178), followed by family not being prepared to wait (n=77) and family unaware of patient's wishes (n=57). The majority of referrals whose family consented were of Caucasian background (83%) compared to 56% of referrals whose family refused. Of the families that provided consent, 34% of referrals were listed as having no religion compared to 21% of families that refused consent. However, religion and ethnicity were only routinely collected from 2014.

Conclusions: There is potential to increase organ donors in NSW by reducing family refusals. Discussing organ donation preferences with family members and better understanding cultural or religious barriers may assist in reducing family refusals.

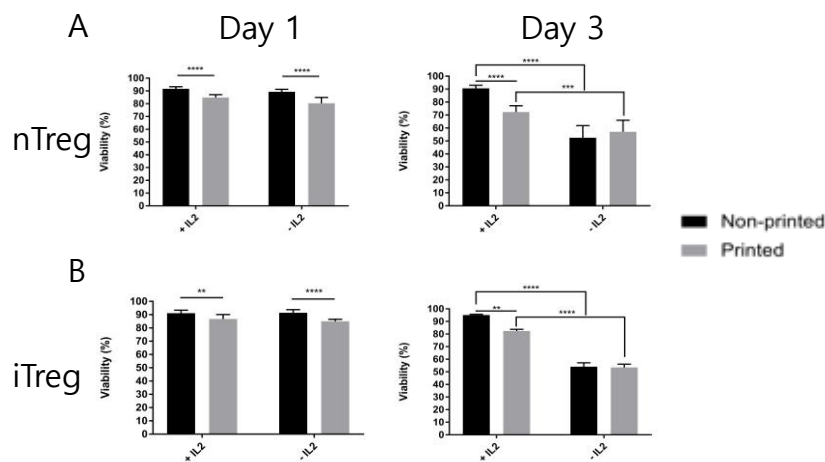


Figure 1. Viability of Tregs in alginate-gelMA hydrogel at day 1 and 3. (A) Assessment of nTreg viability at day 1 and 3. (B) Assessment of iTreg viability at day 1 and 3 (n=3, mean±SD; mean±SEM for iTreg day 3; One-way ANOVA with Tukey's multiple comparison test; **P<0.01, ***0.001, ****0.0001).

Abstract No. 13 (cont)

Table 1. Characteristics of NSW organ donor referrals in 2010-2016, by family consent status.

	Family Consent			Total n (%)
	Refused n (%)	Given n (%)	Not Sought n (%)	
Total	831	1096	1856	3783
Age Category				
<1	2 (<1)	4 (<1)	6 (<1)	12 (<1)
1-17	43 (5)	62 (6)	49 (3)	154 (4)
18-44	164 (20)	261 (24)	267 (14)	692 (18)
45-54	145 (18)	196 (18)	277 (15)	618 (16)
55-64	207 (25)	261 (24)	351 (19)	819 (22)
65-74	160 (19)	204 (19)	438 (24)	802 (21)
75+	101 (12)	106 (10)	450 (24)	657 (17)
Unknown	9 (1)	2 (<1)	18 (1)	29 (<1)
Sex				
Female	339 (41)	470 (43)	721 (39)	1,530 (40)
Male	489 (59)	622 (57)	1,119 (60)	2,230 (59)
Unknown	3 (<1)	4 (<1)	16 (<1)	23 (<1)
Religion				
Christian	226 (61)	444 (61)	497 (66)	1,167 (63)
Muslim	19 (5)	4 (1)	24 (3)	47 (3)
Buddhist	31 (8)	11 (2)	19 (3)	61 (3)
Hindu	6 (2)	7 (1)	16 (2)	29 (2)
Jewish	2 (1)	2 (<1)	1 (<1)	5 (<1)
Other	9 (2)	12 (2)	23 (3)	44 (2)
No religion	80 (21)	246 (34)	173 (23)	499 (27)
Unknown	458 -	370 -	1,103 -	1,931 -
Ethnicity				
Indigenous Australian	30 (6)	26 (3)	42 (4)	98 (4)
White	270 (57)	785 (83)	720 (68)	1,775 (72)
Maori	9 (2)	4 (<1)	4 (<1)	17 (1)
Pacific Islander	8 (2)	3 (<1)	17 (2)	28 (1)
North-East Asian	43 (9)	15 (2)	70 (7)	128 (5)
South East Asian	42 (9)	33 (4)	60 (6)	135 (5)
Southern Asian	12 (3)	15 (2)	24 (2)	51 (2)
South-East European	22 (5)	15 (2)	40 (4)	77 (3)
North African and Mid	10 (2)	3 (<1)	17 (2)	30 (1)
Sub-Saharan African	0 (0)	1 (<1)	2 (<1)	3 (<1)
Other	30 (6)	41 (4)	57 (5)	128 (5)
South American	1 (<1)	1 (<1)	0 (0)	2 (<1)
Unknown	354 -	154 -	803 -	1311 -

Abstract No. 14**INTRODUCTION OF SHARE 35 INTERREGIONAL ALLOCATION FOR HIGH MELD LIVER TRANSPLANT WAITING LIST PATIENTS IN AUSTRALIA AND NEW ZEALAND****FINK Michael^{1,2}, GOW Paul², BALDERSON Glenda³, JONES Robert^{2,1}**¹*Department of Surgery, University of Melbourne,* ²*Liver Transplant Unit Victoria, Austin Hospital, Melbourne,*³*Australia and New Zealand Liver Transplant Registry*

Aims Patients with high MELD scores awaiting liver transplantation have a high risk of waiting list mortality and a short window of opportunity for rescue. A voluntary trial of sharing of livers between units in Australia and New Zealand for patients with a MELD score ≥ 35 (Share 35) was undertaken. The aim of this study is to assess the impact of the trial on waiting list mortality.

Methods The waiting list mortality rate of patients whose MELD score reached 35 prior to commencement of the Share 35 trial, "Share 35 candidates", was compared with that of patients listed as Share 35 patients during the period of the trial, "Share 35 listed", using Chi square. Post-transplant survival of the two groups was compared using Kaplan-Meier graphs with log-rank.

Results During the 21-month period of the trial, 24 patients were Share 35 listed, of whom 13 were transplanted with a shipped liver, eight were transplanted with a local donor liver and three died waiting. The waiting list mortality rate of Share 35 listed patients (3 of 24, 13%) was significantly less than that of Share 35 candidates (13 of 27, 48%, $P = 0.006$). Post-transplant survival was not significantly different between the groups ($P = 0.420$).

Conclusions Introduction of Share 35 to Australia and New Zealand has resulted in improved access to liver transplantation for a group of patients that previously were at high risk of waiting list death without adversely affecting utility.

Abstract No. 15**WHAT HAPPENED WHEN THE 'SOFT OPT-OUT' TO ORGAN DONATION WAS IMPLEMENTED IN WALES? FAMILY AND PROFESSIONAL VIEWS AND EXPERIENCES, AND CONSENT RATES FOR THE FIRST 18 MONTHS.**

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¹School of Social Sciences, Bangor University, ²Wales Kidney Research Unit, Bangor University, ³National Centre for Population Health and Well being Research,, ⁴Major Health Conditions Policy Team, Welsh Government, ⁵Department of Organ Donation, NHS Blood and Transplant, ⁶North West Regional Office, Liverpool, UK, NHS Blood and Transplant, ⁷Department of Nephrology and Transplantation, Cardiff and Vale University Health Board,, University Hospital of Wales, Cardiff, UK.

Introduction: On 01.12.15 Wales introduced a 'soft opt-out' system of organ donation.

Methods: Co-productive, mixed-methods study partnered with National Health Service Blood and Transplant and patient and public representatives. Data were collected on all 211 approaches between 01.01.15-31.05.17: 182/211 deceased patients came under the Act. Depth data (62 interviews with 85 family members, and questionnaires) on 60 patients who were potential/actual organ donors; and 2 focus group/individual interviews with 19 NHS BT professionals [figure1]. Organ Donor Register (ODR) activity was monitored.

Results: Welsh consent rates increased by around 10% to 61%; 64% when family consent was removed. This was higher than England and has reversed an unexplained drop to 48.5% before implementation. However, family member(s) still overrode the patient's organ donation decision 31/205 times. 46/205 cases had their consent deemed with a consent rate of 61%. The Act provided a useful framework but family members did not fully understand deemed consent. Negative personal organ donation views and health systems issues affected support for organ donation. The media campaign missed the changed role of the family; that they were no longer the decision maker about organ donation. ODR 'opt-outs' were 6%, less than anticipated. **Discussion:** The media campaign mostly worked but was not memorable and had gaps. More work is needed to inform the family about their changed role. Scotland and England are now in the consultation process to move to an 'opt-out' system. As a result of this study Welsh Government commissioned a new campaign launched 01.11.17.

Abstract No. 15 (cont)

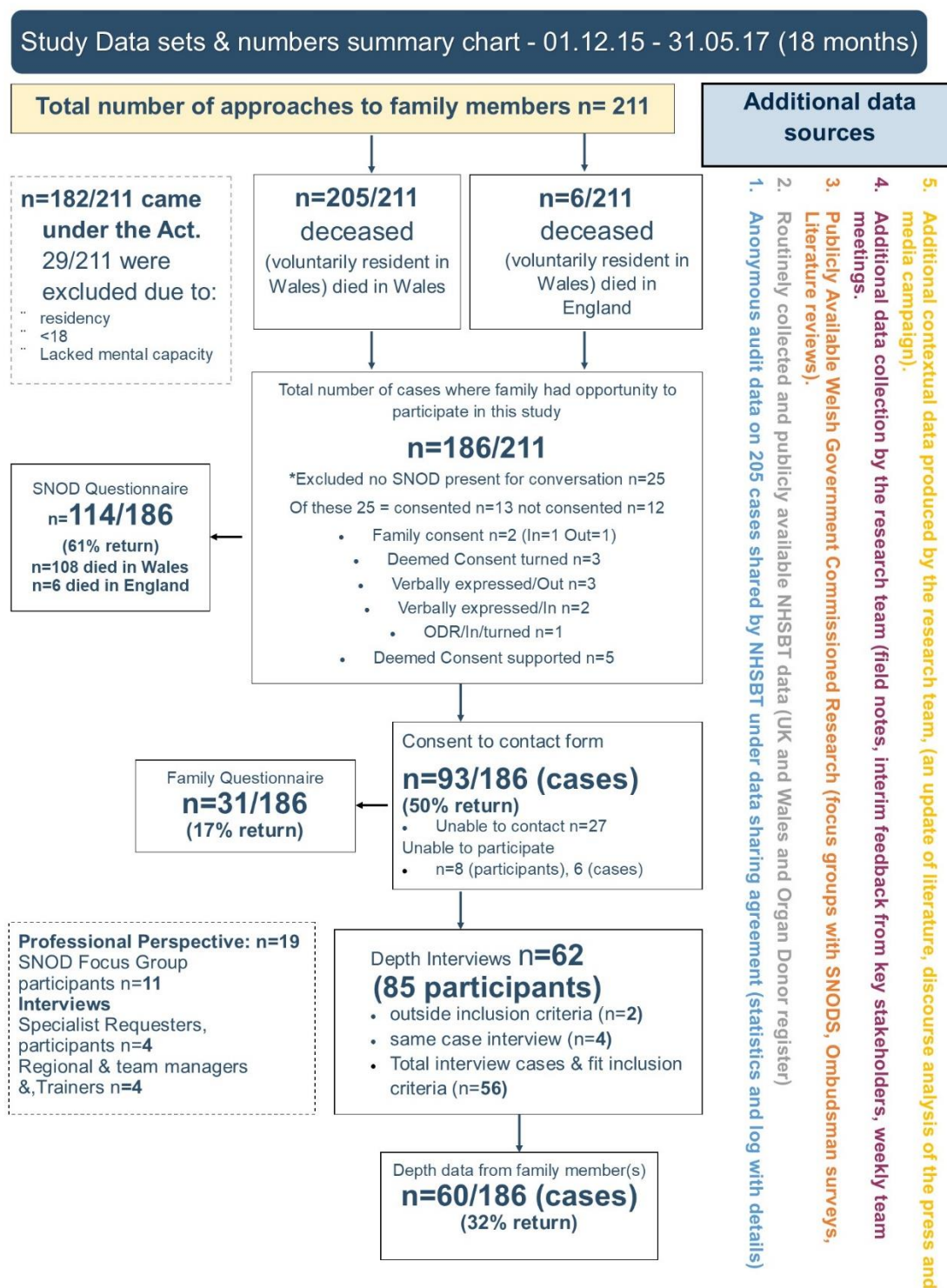


Figure 1.

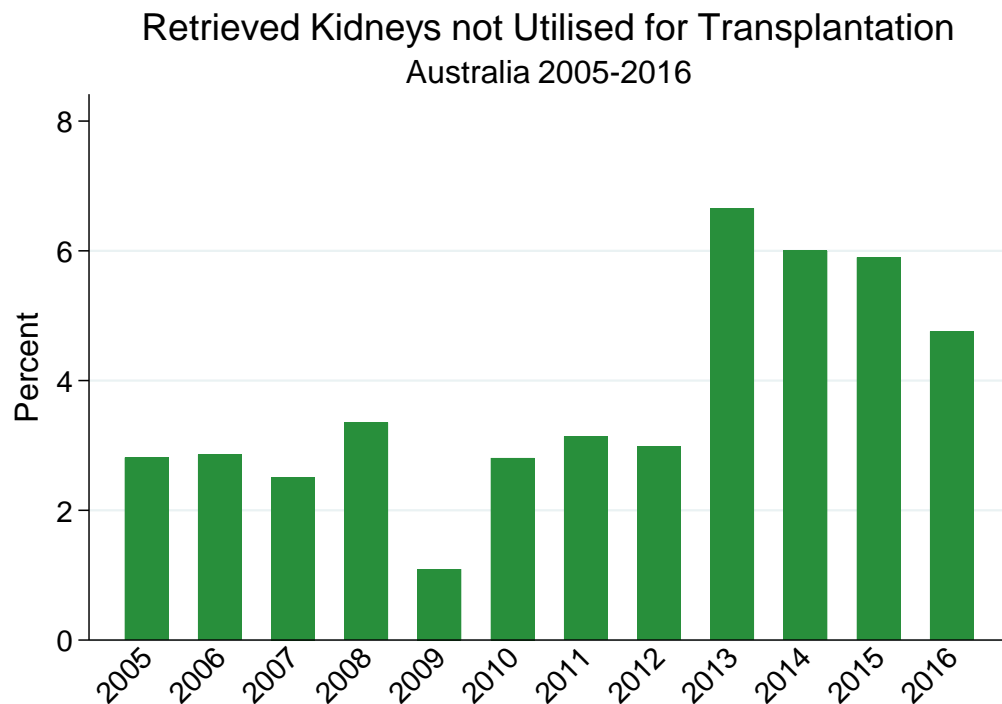
Abstract No. 16

THE INCREASED RATE OF NON-UTILISATION OF KIDNEYS RETRIEVED FOR TRANSPLANTATION IN AUSTRALIA IS INDEPENDENT OF DONOR CHARACTERISTICS.**SYPEK Matthew^{1,2,3}, ULLAH Shahid^{1,4}, CLAYTON Phil^{1,4,5}, MSDONALD Stephen^{1,4,5}**¹ANZDATA, ²Department of Nephrology, Royal Melbourne Hospital, ³Department of Medicine, University of Melbourne, ⁴Adelaide Medical School, University of Adelaide, ⁵Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital

Aims: In 2013, Australia experienced an increase in the percentage of deceased donor kidneys that were retrieved but not utilised for transplantation. We aimed to determine if the increase could be explained by changes in donor characteristics over this time period.

Methods: Data from ANZOD were used to examine donor characteristics over the period 2005-2016. A multi-level logistic regression model was used to determine if era of donation (2013-2016 vs 2005-2012) was an independent predictor of organ non-utilisation after adjusting for donor characteristics.

Results: During the study period, 6,916 kidneys were retrieved for transplantation in Australia with 281 (4.0%) not utilised; 2.7% in 2005-2012 compared to 5.8% in 2013-2016 (figure 1). Donors from 2013-2016 were older (median age 47 vs 50, $p < 0.001$), more likely to have donated after circulatory death (25.7% vs 15.6%, $p < 0.001$), and had a higher median KDRI, but were less likely to have oliguria or be on inotropes.



Era of donation (2013-2016 vs 2005-2012) was a significant predictor of kidney non-utilisation on univariate analysis (OR 2.17, 95% CI 1.70-2.77) and after adjustment for donor factors (age, diabetes, hypertension, HCV, cause of death, pathway, inotropes, oliguria, and admission and terminal creatinine) and state (adjusted OR 2.08, 95% CI 1.58-2.72).

Conclusion: Kidneys retrieved in 2013-2016 were more likely to be not-utilised for transplantation even after adjusting for changes in donor characteristics over time. Further investigation is required to determine if increased organ non-utilisation is due to changes in reporting or clinical practice, and help maximise utilisation rates.

Immunobiology

Abstract No. 17

THE CHANGES OF DYNAMIC IMMUNE PROFILE AND OUTCOME DIFFERED BETWEEN INDIVIDUAL WITH TYPE 1 DIABETES ISLET TRANSPLANTATION UNDER ATG/TACROLIMUS/MMF/ETANERCEPT SUPPRESSION REGIMEN

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Aim: To evaluate the dynamic-whole-blood-immune-profile(WBIP) of T1D-islet-transplant-recipients(R) pre&post-transplant (Tx), and correlate with Tx outcomes.

Method&Material: WBIP of 4 islet-Tx-recipients(R1-4) was performed pre-&post-Tx [(at 2weeks(w), and 1,3,6,&2 months(m)]. R1 received 3-islet-Tx, R2 received 2-Tx, and R3&R4 had 1-Tx. The fold change in cell numbers following Tx was compared to levels prior to 1st-Tx. The induction immunosuppression-regimen was ATG/Tacrolimus/MMF/Etanercept, except R1 did not receive Etanercept at his 1st-Tx.

Results: R1 had a significant reduction in insulin requirement after the-2nd-Tx, and was insulin free after the-3rd-Tx. R2 remains on insulin after 2-Txs, but is c-peptide positive with a partial reduction in insulin use. In R3 remains on insulin after 1-Tx, but dosage has halved. In R4 after 1-Tx has a partial reduction in insulin dosage. Prior to their 1st-Tx all recipients had immunesubset numbers within normal range; except R2 who had an elevated CD45+cells (1272cells/uL) due to high granulocyte count (9383cells/ul). We observed that the changes of dynamic WBIP for absolute cell numbers after immunosuppression differed between individual Tx-recipients (Table 1). There were small changes of the subset proportion of CD14+ monocytes(classical/intermediate/non-classical) in R1-4 and changes in the proportion CD56+HiCD16-/CD56+HiCD16+NK cells in R1,2,&4 were observed. The ratio of CD4/CD8 T-cells were reversed at most post-Tx time points for all recipients. The degree of reduction in T cell number did not correlate with successful outcomes.

Conclusion: Dynamic WBIP after Tx can be performed and used to evaluate an individual's response to Tx and immunosuppression. Monitoring WBIP may be important after Tx to guide the usage of immunosuppression.

Table 1: The change of absolute immune cell numbers after islet transplantation in 4 recipients

*Fold: 1 indicate no change, >1 indicate decrease and <1 (in red) indicate increase compared to Pre-TX.

Time Point	CD45+ Fold*	Granulocyte fold	Monocyte fold	B Cell fold	T Cell fold	NK fold	NKT fold
PRE /R1	1	1	1	1	1	1	1
2 W	1.15	0.99	1.49	1.31	3.7	1.64	1.41
1 M	1.72	1.55	2.72	1.07	4.1	1.42	1.32
3 M	1.3	1.18	1.08	1.35	4.63	1.22	1.54
5M (2W 2nd Tx)	1.71	1.48	1.33	1.61	43.52	3.85	14.82
5.5M (1M 2nd Tx)	2.37	2.02	3.48	1.81	21.15	2.33	6.9
6M (2M 2nd Tx)	3.48	3.27	2.64	1.98	21.24	2.61	11.42
8M (1M 3rd Tx)	6.95	13.53	1.62	2.47	17.78	1.54	8.99

10M (3M 3rd TX)	1.5	1.41	1.4	1.142	3.93	1.02	4.08
12M (6M 3rd Tx)	1.87	1.85	1.41	1.43	5.1	0.85	5.066
PRE/R2	1	1	1	1	1	1	1
2 W	2.22	2.28	2.21	1.19	2.5	0.93	0.77
1 M	2.17	2.03	2.6	2.35	3.29	1	0.77
3 M	2.19	2.03	2.678	2.8	4.09	0.64	1.11
4.5M (2W 2nd Tx)	2.77	2.5	3.23	2.27	6.52	1.38	2.18
5 (1M 2nd Tx)	2.65	2.42	2.7	2.993	6.475	1	1.67
6.5M (3M 2nd Tx)	2.22	1.93	2.8	2.54	6.75	1.23	2.098
PRE/R3	1	1	1	1	1	1	1
2 W	1.52	1.22	0.87	2.4	8.83	2.87	5.93
1 M	1.33	1.05	1.67	1.4	4.57	1.49	3.48
3 M	1.53	1.25	1.48	1.94	4.47	1.47	3.42
6 M	1.73	1.4	1.27	2.16	7.32	2.25	5.92
Pre	1	1	1	1	1	1	1
2w	1.46	0.8	2.9	2.153	77.82	11	15.17
1m	0.68	0.36	1.45	3.27	38.21	6.03	9.33
6m	1.88	1.089	2.75	2.85	27.3	5.53	11.79

Abstract No. 18**THE ROLE OF THE CD73/A2A SIGNALLING AXIS IN A HUMANISED MOUSE MODEL OF GRAFT-VERSUS-HOST DISEASE****GERAGHTY Nicholas^{1,2,3}, ADHIKARY Sam^{1,2,4}, SLUYTER Ronald^{1,2,3}, WATSON Debbie^{1,2,3}**¹*School of Biological Sciences, University of Wollongong*, ²*Centre for Medical and Molecular Biosciences, University of Wollongong*, ³*Illawarra Health and Medical Research Institute, University of Wollongong*, ⁴*Illawarra Health and Medical Research Institute*

Graft-versus-host disease (GVHD) is a complication that occurs in approximately 50% of bone marrow transplantations, due to donor leukocytes (predominantly T cells) in the graft mounting an immune response against the patient (host). Extracellular adenosine, generated by the ecto-enzyme CD73, activates A2A to limit T cell responses. CD73 or A2A blockade worsens disease, while A2A activation reduces disease in allogeneic mouse models of GVHD.

Aim: The current study aimed to investigate the role of the CD73/A2A signalling axis in a humanised mouse model of GVHD.

Methods: NOD-SCID-IL2 γ^{null} (NSG) mice injected with 10 x 10⁶ human (h) peripheral blood mononuclear cells (PBMC), were subsequently injected with $\alpha\beta$ -methyleneADP (APCP) (CD73 antagonist) or CGS21680 (A2A agonist) or control diluent for 14 days. GVHD development was assessed by weight loss, clinical parameters, and survival. The impact of APCP and CGS21680 on immune cells and cytokines were investigated.

Results: CD73 blockade enhanced weight loss but did not alter clinical score or survival. CD73 blockade increased serum human interleukin (IL)-2 concentrations. A2A activation increased weight loss, but did not impact clinical score or survival. CGS21680 led to a decrease in immunosuppressive regulatory T cells, however serum tumor necrosis factor (TNF)- α and IL-2 were reduced, and IL-6 was increased.

Conclusion: A2A activation represents a potential therapeutic target for GVHD due to reduced inflammation, but should be carefully considered due to the negative effects on weight loss and regulatory T cells. Therefore, further investigation into A2A activation is warranted before it can be used as a therapeutic strategy for GVHD in humans.

Abstract No. 19**CHANGES IN THE EXTRACELLULAR MATRIX - SIGNS OF REMODELING LEADING TO CHRONIC REJECTION AFTER LUNG TRANSPLANTATION****MULLER Catharina¹, HEINKE Paula¹, ANDERSSON-SJÖLAND Annika¹, SCHULTZ Hans Henrik², ANDERSEN Claus³, IVERSEN Martin², WESTERGREN-THORSSON Gunilla¹, LEIF Eriksson¹**¹*Experimental Medical Science, Lund University, Sweden*, ²*Section for lung transplantation, Copenhagen University Hospital, Denmark*, ³*Department of pathology, Copenhagen University Hospital, Denmark*

Background: About 50% of lung transplanted patients develop chronic rejection in the form of bronchiolitis obliterans syndrome (BOS) within 5 years after transplantation. BOS is characterized by a decrease in lung function, caused by progressive fibrosis. However, little is known about its initiation. We hypothesize that changes in the distribution of extracellular matrix proteins might be a marker for the disease process.

Methods/Material: Our study aimed to map total collagen, collagen type IV, biglycan and periostin in transbronchial biopsies taken at 3 and 12 months after transplantation using Masson's Trichrome staining and immunohistochemistry. Staining patterns were quantified and related to patient data (n=58) in a 5-years follow-up.

Results: Compartment specific patterns could be revealed between 3 and 12 months post-transplantation. Alveolar total collagen (p=0.019) and small airway biglycan (p=0.02) increased in BOS-developing patients. Alveolar collagen type IV increased in BOS-free patients (p=0.01) (3 vs. 12 months). Individual calculation of the change in protein content (12 minus 3 months for the respective patient) confirmed the increase in biglycan (p=0.012) and showed a trend for increased periostin (p=0.057) in the small airways of BOS patients compared to BOS-free patients. Already at 3 months, before onset of BOS, increased total alveolar collagen (p=0.036) and small airway collagen type IV (p=0.034) could discriminate between patients developing less severe and severe forms of BOS (BOS grade 1+2 vs. 3).

Conclusion: The results show distinct alterations of the extracellular matrix which might be part of the complex remodeling processes that eventually lead to BOS.

Abstract No. 20

MTORC2 DEFICIENCY IN DENDRITIC CELLS PROMOTES ACUTE KIDNEY INJURY

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Introduction: The role of the mammalian/mechanistic target of rapamycin (mTOR) in the pathophysiology of acute kidney injury (AKI) is poorly characterized. Furthermore, the influence of dendritic cell (DC)-based alterations in mTOR signalling in AKI has not been investigated.

Methods: Bone marrow-derived mTORC2-deficient (Rictor^{-/-}) or wild-type (WT) DC underwent hypoxia-reoxygenation and were analysed by flow cytometry. Age- and gender-matched DC-specific Rictor^{-/-} mice or littermate controls underwent bilateral renal ischemia-reperfusion injury followed by assessment of renal function, histopathology, renal DC metabolism, bio-molecular and cell infiltration analysis. Adoptive transfer of WT or Rictor^{-/-} DC to C57BL/6 mice was used to assess migratory capacity.

Results: AKI upregulates expression of phospho-S6K (downstream of mTORC1), but downregulates phosphorylated Akt S473 (downstream of mTORC2) in whole kidney tissue. Rictor^{-/-} DC expressed more CD80/CD86 but less programmed death ligand-1 (PDL1) that was enhanced by hypoxia-reoxygenation, and demonstrated enhanced migration to the injured kidney. Following AKI, Rictor^{-/-}DC mice developed higher serum creatinine, more severe histologic damage, and greater pro-inflammatory mRNA transcript profiles of IL-1 β , IL-6 and TNF- α compared to littermate controls. A greater influx of neutrophils and T cells was seen in Rictor^{-/-} DC mice, in addition to CD11c⁺MHCII⁺CD11b^{hi}F4/80⁺ renal DC, that expressed more CD86 but less PDL1. Rictor^{-/-} DC showed increased TNF- α but significantly reduced IL-10 production, and were glycolytically biased compared to WT DC under both basal and AKI conditions.

Conclusions: These data suggest that mTORC2 signaling in DC negatively regulates AKI, highlighting the regulatory roles of both DC and Rictor in the pathophysiology renal injury.

Outcome Measures

Abstract No. 21

OUTCOMES OF WESTERN AUSTRALIAN LUNG TRANSPLANT RECIPIENTS – THE FIRST DECADE

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¹Fiona Stanley Hospital, ²Institute for Immunology and Infectious Diseases, Murdoch University, ³Lung Transplant Service, Fiona Stanley Hospital, ⁴School of Medicine, University of Notre Dame

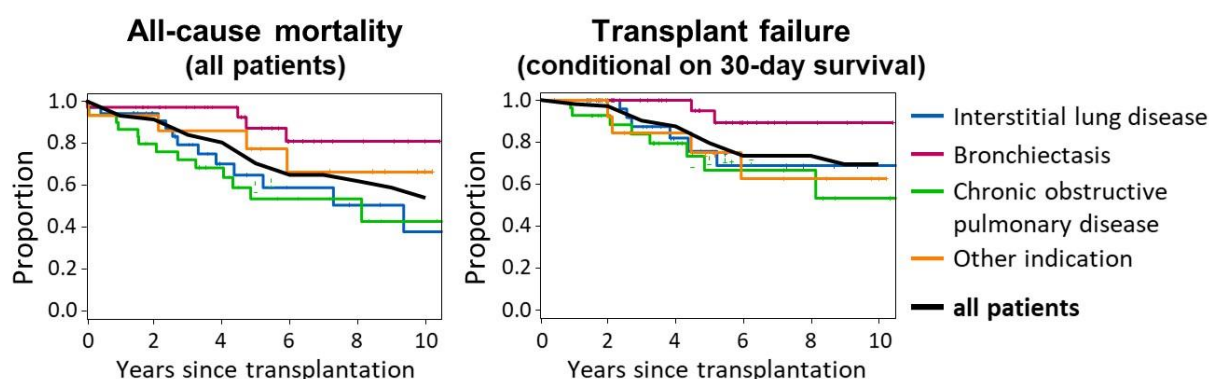
Background: Lung transplantation has evolved into an effective treatment option for end-stage lung disease. Growing local demand prompted the establishment of the Advanced Lung Disease Unit at Royal Perth Hospital in Western Australia in 2004. Operating now for just over a decade and recently relocated to Fiona Stanley Hospital, we sought to assess our recipient characteristics and outcomes, and compare ourselves to the international standard.

Method: Basic characteristics of all transplant recipients between 2004 and 2015 were collected at the time of transplant. This data was retrospectively augmented from our electronic hospital medical records system. Survival analysis was performed using the Kaplan-Meier method.

Results: A total of 115 lung transplants were performed. Transplant rates have trended upwards over the years, with 20 lung transplants performed in 2015. Half the recipients were over the age of 50. The most common indications for transplant, each accounting for a quarter of total transplants, were Cystic Fibrosis, Interstitial Pulmonary Fibrosis and Chronic Obstructive Pulmonary Disease. Overall survival rates were 96% at 3 months, 93% at 1 year, 84% at 3 years, and 70% at 5 years (**Figure 1**). This compares well to international survival rates, published by the International Society of Heart and Lung Transplantation, of 89% at 3 months, 80% at 1 year, 65% at 3 years, and 54% at 5 years.

Conclusion: Lung transplants rates continue to rise and our patients enjoy international standard outcomes.

Figure 1. Kaplan-Meier survival estimates of time to death (all-cause) and time to transplant failure conditional on survival beyond hospital discharge.



Abstract No. 22

ALLOGRAFT OUTCOME FOLLOWING RETRANSPLANTATION OF PATIENTS WITH FAILED FIRST KIDNEY ALLOGRAFT ATTRIBUTED TO NON-ADHERENCE

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Background: It remains unknown whether first allograft failure secondary to non-adherence leads to increased risk of allograft failure following retransplantation.

Aim: To determine the association between causes of first allograft failure and outcomes following retransplantation.

Materials and Methods: Using the ANZDATA Registry, patients who had received a second kidney transplant between 1960-2014 were included. The association between causes of first allograft failure, death censored graft failure (DCGF) and non-adherence-related DCGF following retransplantation were examined using Cox regression and competing risk analyses.

Results and Discussion: Of 2822 patients who have received second kidney allografts, 59 (2%) lost their first allografts from non-adherence. Patients who had non-adherence-related first graft failure were younger at the time of first allograft failure (median 25 vs 38 years, $p < 0.001$) and had significantly longer waiting times for retransplantation (waiting time > 5 years: 57% vs. 20%, $p < 0.001$) compared with those who lost their first graft from other causes. The adjusted HR for DCGF was 0.76 (95%CI 0.44, 1.32; $p = 0.342$) for those who had lost their first allograft from non-adherence. Following retransplantation, the adjusted subdistribution HR of second allograft failure attributed to non-adherence for patients who had experienced non-adherence-related first allograft failure was 2.84 (95%CI 0.83, 17.79; $p = 0.082$).

Conclusion: In patients who had experienced non-adherence-related first allograft failure, the long-term risk of DCGF in the second allograft was similar to those who had lost their first allografts from other causes. Non-adherence-related allograft failure should not be considered a contraindication to successful retransplantation.

Abstract No. 23

EVEROLIMUS AND LONG-TERM CLINICAL OUTCOMES IN KIDNEY TRANSPLANT RECIPIENTS: A TRIAL-BASED ANZDATA LINKAGE STUDY

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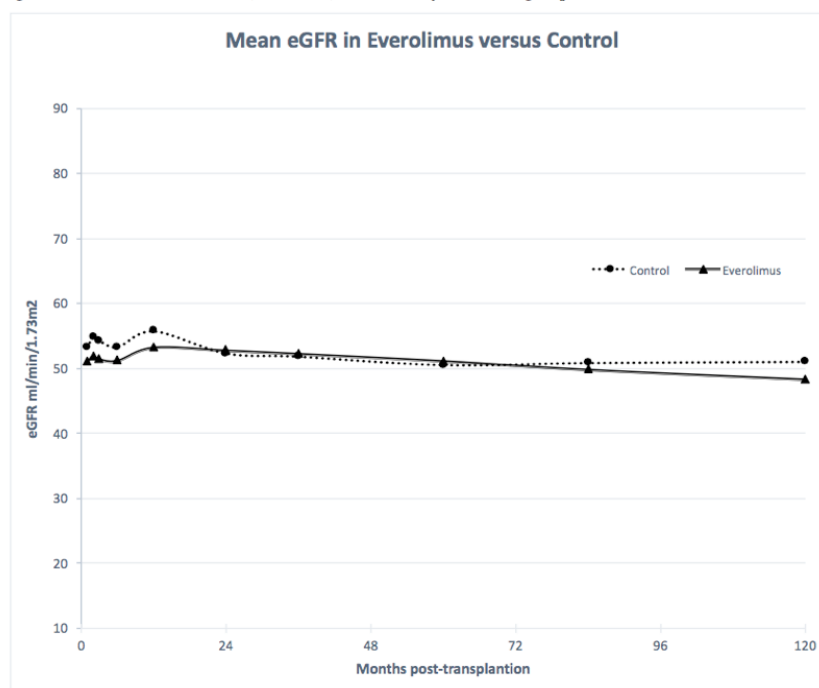
Aim: To compare long-term graft function and the risk of graft loss and death in patients randomised to everolimus (EVL) compared with controls on calcineurin-inhibitor-based triple therapy.

Method: We included all Australian and NZ kidney transplant recipients who were enrolled in RCTs which compared an EVL-containing regimen with control. Participants were linked to ANZDATA for the outcomes of eGFR, graft loss and death. Multilevel mixed-effects modelling was used for repeated measures of eGFR (for *de-novo*/early-conversion trials) and adjusted random-effects Cox models were constructed to examine the association between treatment types and the risk of graft loss and death.

Results: Five RCTs with 349 participants (EVL=242, Control=107) were included. Decline in eGFR did not differ significantly between EVL vs. control over a median follow-up of 7-years (mean diff: 0.01ml/min/1.73m², 95%CI -0.06 to 0.09)(Fig-1) There were 39(16%) graft-losses and 41(17%) deaths in EVL vs. 12(11%) graft-losses and 13 (12%) deaths in controls after a median follow-up of 9.3-years (IQR 7.2, 13.5). The proportion who died from cardiovascular disease was higher in EVL (5.4%) vs. control (0.9%)(p=0.05). Death from other causes such as cancer and infection were similar. There was no association between EVL and death-censored graft-loss (adjHR=1.17 95% CI 0.59–2.31) or all-cause death (adjHR=1.50, 95% CI 0.79–2.85).

Conclusion: Renal function, graft and patient survival were similar amongst EVL patients vs. standard-of-care after 7 to 9 years of follow-up. Although overall numbers were small, there was a higher proportion of death attributed to cardiovascular disease in the EVL arm.

Figure 1: Mean estimated GFR (CKD-EPI) over time by treatment group



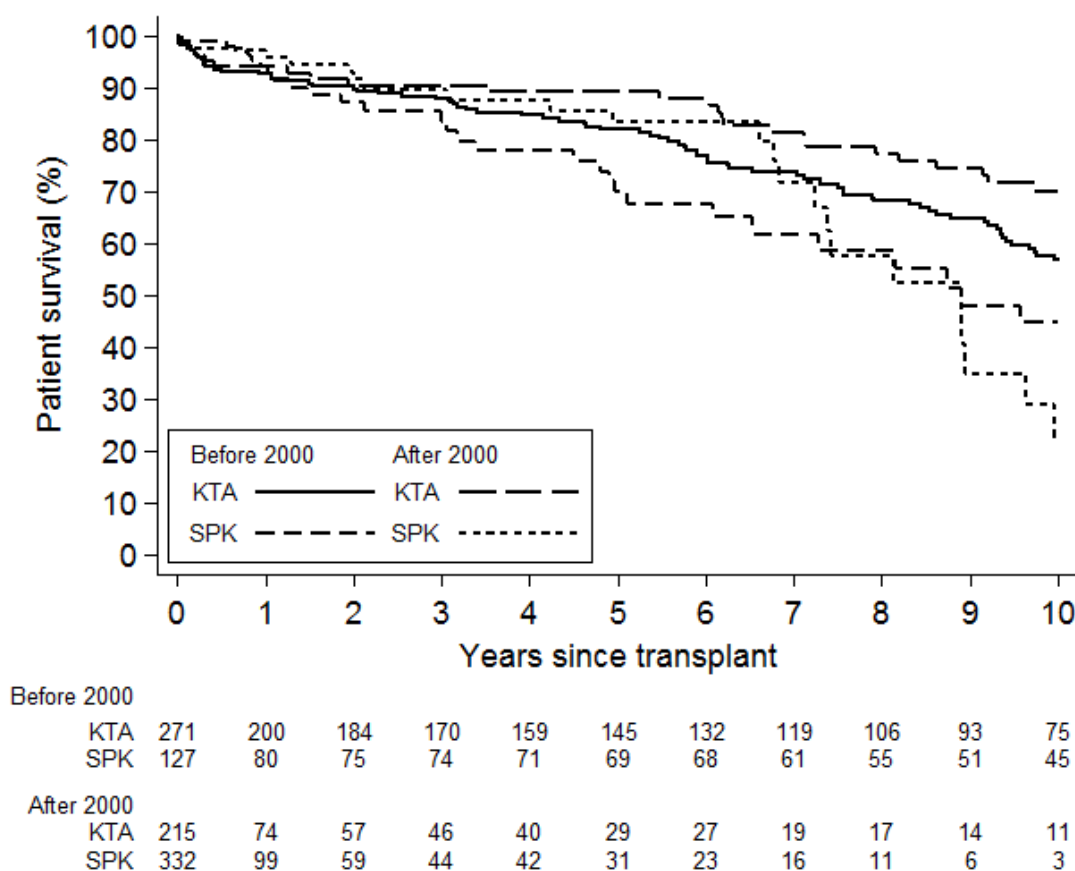
Abstract No. 24

POST-TRANSPLANT SURVIVAL IN TYPE 1 DIABETICS IN AUSTRALIA AND NEW ZEALAND**WEBSTER Angela^{1,2}, HEDLEY James¹, KELLY Patrick¹**¹*School of Public Health, University of Sydney*, ²*Centre for Transplant and Renal Research, Westmead Hospital, Sydney*

Introduction. We analysed data from the Australian and New Zealand Pancreas and Islet Transplant Registry (ANZIPTR) as well as the Australian and New Zealand Dialysis and Transplant Registry (ANZDATA) to estimate differences in transplant and patient survival by transplant type among recipients with type 1 diabetes. **Materials and Methods.** We conducted an inception cohort study from 1984-2012, using data linkage of ANZIPTR and ANZDATA. We compared kidney graft and patient survival from the date of transplant for SPK and deceased-donor KTA recipients using Cox regression, and censored patients at last known follow-up. We adjusted for age, sex, state/country, previous transplants, age difference between recipient and donor, and immunosuppression used. To meet the proportional hazards assumption we stratified by era (1984-1999, 2000-2012).

Results. We included 1,090 transplant recipients (462 SPK, 493 deceased donor kidney, 135 living donor kidney). SPK had improved kidney survival compared to deceased donor KTA; including death with function (graft loss HR 0.35; 95% CI 0.21-0.57; $p < 0.001$) and censored for death (graft loss HR 0.45; 95% CI 0.22-0.90; $p = 0.02$). Patient survival was also better among SPK recipients compared to deceased donor KTA (death HR 0.48; 95% CI 0.24-0.95; $p = 0.03$).

Conclusion. Overall, patient and kidney transplant survival has improved over time for SPK and KTA recipients. At 5 years, patient survival is $>90\%$, and kidney transplant survival $>80\%$. The diminishing advantage of SPK over KTA may reflect selection bias compared to earlier years when SPK donors were scarcer and PAK was more common.

Figure 1: Survival of type I diabetics receiving transplants in Australia and New Zealand

Abstract No. 25

DECEASED-DONOR KIDNEY TRANSPLANTATION PROGRAM IN NEW CALEDONIA

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Aims. New-Caledonia is a French territory located in South-Pacific, counting 268 767 inhabitants in 2014. Distance from Sydney by flight is 3 hours, and 24 hours from Paris. Its ESRD prevalence is one of the highest in the world, more than 3000 pmh. Before 2012, kidney transplantation access was poor, with living donor kidney transplantation possible either in France or Australia, but cadaveric donor transplantation only possible in France. In 2012, a deceased donor program opened in New Caledonia, through a workshop with Australia.

Methods. Extension of French bioethic laws was needed to establish a brain death legislation. International collaboration was conventionned between Australia and New Caledonia. The French “Agence de la Biomedecine” helped to dictate the standards of organ retrieval and organ allocation. A tissue typing laboratory was created in 2009, in agreement with the RPAH, Sydney. Noumea’s physicians started inscribing patients on transplantation waitlist in 2011, reviewed by Sydney’s physicians on a yearly basis.

Results. From April 2013 to January 2018, 78 brain deaths have been evaluated in Noumea, leading to 14 procedures retrievals (8 by Sydney physicians, and 6 by Noumea’s team). 28 patients underwent kidney transplantation in RPAH, Sydney, with mean cold ischemia of 14.8 hours, and delay graft function in 22 %. Mean follow up was 28 months, with patients global survival 96 %, and graft survival of 96 %.

Conclusions. This program delivers a safe and successful transplantation access for patients without living donors, through a unique international medical collaboration.

Abstract No. 26

DELAYED KIDNEY TRANSPLANTATION WAIT-LISTING- CLIENT AND CLINICIAN PERCEPTIONS OF THE IMPACT OF EXTENSIVE CHRONIC CUTANEOUS DERMATOPHYTE INFECTION**MAJONI Sandawana William^{1,2,3}, HUGHES Jaquelyne T^{2,1}, AYE MIN Oka¹, WHITE Evonne⁴, CURRIE Bart J^{5,7}, HALL Heather⁶, KIRKHARM Ranae²***¹Department of Nephrology, Royal Darwin Hospital, ²Wellbeing and Preventable Chronic Disease Division, Menzies School of Health Research, Charles Darwin University, ³Northern Territory Medical Programme, Flinders University School of Medicine, ⁴Top-End Renal Patient Advisory and Advocacy Committee,, Top End Health Services, ⁵Department of Infectious Diseases, Royal Darwin Hospital, ⁶Panuku, Western Desert Nganampa Walytja Palyantjaku Tjutaku,, ⁷Global and Tropical Health Division, Menzies School of Health Research, Charles Darwin University***Aim:** To examine factors associated with delayed kidney transplant wait-listing in a patient with end stage kidney disease and extensive cutaneous dermatophyte infection.**Background:** Access to kidney transplant wait-listing is partly determined by efficient transplantation assessments, including cure of existing infections. Skin infections are common in Northern Australia, including in patients with end stage kidney disease.**Methods:** We documented the treatment response for extensive chronic tinea corporis (TC) for one client during the time between haemodialysis initiation and transplant wait-listing. Follow-up interviews with the client and supporting clinicians were completed to learn individual perspectives of care.**Results:** A 41 year old Aboriginal female with diabetic nephropathy achieved wait-listing 16 months after dialysis initiation. Assessments with visiting transplant physician recommended wait-listing pending transplant surgeon review, and cure of TC. The *Trichophyton rubrum* infection, present since dialysis initiation, achieved partial but not complete cure with topical clotrimazole and oral terbinafine 250mg oral post dialysis three times a week. Complete resolution was documented at 13.2 months following one month oral fluconazole (100mg weekly at dialysis). Timeliness of skin infection management did not meet the expectations of clinical guidelines (recommending 2 weeks treatment) or clinicians or client. Systemic and individual factors contributed to the management of this condition, including disjointed communication, competing priorities of staff and patients and medication delivery systems.**Conclusions:** Optimization of standard treatment guidelines for use in haemodialysis is required, and may, through timely cure, improve access to kidney transplant wait-listing for clients in Northern Australia with chronic extensive tinea corporis.

Transplant Workup Milestones	months
Transplant Physician Review	9.5
Achieved infection clearance	13
Transplant Surgeon review	15.5
Achieved wait-listing	16.5

Abstract No. 27**SURVIVAL AND FUNCTION OF HUMAN ADRENAL CELL IN IMMUNOISOLATION DEVICE IN ADRENALECTOMIZED IMMUNODEFICIENT MICE****CATTERALL T¹, KRISHNA MURTHY B¹, MARIANA L¹, KOS C¹, SACHITHANANDAN N², THOMAS H¹, LOUDOVARIS T¹, KAY T¹**¹*Immunology & Diabetes, St Vincent's Institute, Melbourne,* ²*Department of Endocrine and Metabolism, St Vincent's Hospital, Melbourne*

Background: Primary adrenal insufficiency (PAI) is caused by failure of the adrenal gland to produce steroid hormones - glucocorticoids and mineralocorticoids - and is a potentially lethal disease. Synthetic steroid hormones have transformed PAI from a lethal condition to a chronic one. However, management of PAI is still challenging for patients and clinicians as the current regimens do not restore or replicate normal cortisol secretion in normal conditions and during illness and stress. Hence with current treatment mortality is not normalized and quality of life is poor.

Aim: To treat PAI patients with adrenocortical cells in immunoisolation devices to restore physiological steroid hormone secretion without needing immunosuppression.

Method: We studied the survival and function of isolated human adrenal cells in vitro and in vivo in NRG - SCID mutated NOD mice. About 300x10⁶ adrenocortical cells/ human adrenal gland are routinely obtained with > 80% cells viable, with survival and function in vitro for more than 14 days. A cohort of 10 immunodeficient mice were implanted with an immunoisolation devices into epididymal or ovarian fat pad to allow vascularisation to establish. After 4 weeks, mice underwent bilateral adrenalectomy and were transplanted with 5 million human adrenocortical cells into the device. One mouse died almost 4 weeks after adrenalectomy and the remaining mice are healthy and are being followed up for >10 weeks, secreting cortisol and responding to stimulation with synthetic ACTH 1-24 (synacthen).

Conclusion: Results indicate survival and function of human adrenal cells in the vascularised encapsulation device.

Abstract No. 28**DEVELOPING PHOSPHOLIPASE A2 RECEPTOR ScFv FOR CAR TREGS FOR THE TREATMENT OF AUTOIMMUNE RENAL DISEASE****KARUNIA J¹, WANG YM², ZHANG GY², WILARAS A², BAKHTIAR M², MCCARTHEY H², ALEXANDER SI¹**¹*Centre for Kidney Research, Children's Hospital at Westmead,* ²*Centre for Transplant and Renal Research , Westmead Institute for Medical Research* ³*Children's Hospital at Westmead*

Background: Idiopathic membranous nephropathy (IMN) is a leading cause of autoimmune renal disease driven in many cases by the recently described cognate antigen M-type phospholipase A2 receptor (PLA2R) expressed on glomerular epithelium. Chimeric antigen receptors (CAR) T cells use antibody fragments to direct T cells to specific antigens, and have achieved clinical success in cancer. The strategy can be translated to treat idiopathic membranous nephropathy (IMN), an autoimmune condition that involves PLA2R, a target antigen that is exclusively expressed on the podocyte lining of the kidneys.

Aims: In this project, we aim to use PLA2R as a target antigen for treating IMN and design a single chain fragment of variable region (ScFv) to use in PLA2R-CAR-Tregs directed towards this antigen by generating a PLA2R-specific monoclonal antibody against this antigen on human, mouse and rat podocytes.

Method: By using genetic sequence search tools, the PLA2R amino acid sequence across three (3) species of human, mouse and rat, were aligned and compared to generate three common peptide immunogens. Using a conditionally-immortalized podocyte cell line (ciPod) we examined immunohistochemically for M-Type PLA2R expression on human podocytes *in vitro* as an assay for antibody testing. Mice were immunized with the PLA2R peptides to produce monoclonal antibodies against PLA2R. Hybridomas were established and screened and the hybridoma antibody sequenced for use in making the ScFv for the CAR construct.

Results: We have confirmed human expression of the M-type PLA2R in human podocytes on their cell membrane *in vitro* by immunohistochemical staining. The anti-PLA2R monoclonal antibody (mAb) has been detected in the mouse sera of immunized mice by Western Blot and ELISA. The mAb hybridoma is being sequenced. The anti-PLA2R mAb from these hybridomas is reactive for the human M-Type PLA2R.

Conclusion: We have developed hybridomas against a podocyte target antigen that is also a disease antigen in membranous nephritis and are developing this as a kidney targeting strategy.

Abstract No. 29

DONATION AFTER CIRCULATORY DEATH COMPARED WITH DONATION AFTER BRAIN DEATH: OUTCOMES FOR ISLET TRANSPLANTATION IN AUSTRALIA**HAWTHORNE Wayne^{1,2}, CHEW YiVee², WILLIAMS Lindy², HARON Christian², HITOS Kerry¹, MARIANA Lina³, KAY Tom³, O'CONNELL Philip^{2,4}, LOUDOVARIS Tom³**¹*Sydney Medical School, University of Sydney, Westmead Hospital, Sydney*, ²*Centre for Transplant and Renal Research, The Westmead Institute of Medical Research*, ³*Tom Mandel Islet Transplant Program, St Vincent's Institute, Melbourne*, ⁴*Western Clinical School, University of Sydney*

INTRODUCTION: Islet cell transplantation provides long-term insulin independence treating T1D patient's severe hypoglycaemic unawareness. Significant lack of organ donors results in patients remaining on the waitlist for years. Donation after Circulatory Death (DCD) donors may be a potential resource that could help solve this shortage.

MATERIALS AND METHODS: Islet donor pancreata were compared from the Australian National Islet Transplant program with multiple donor and isolation variable outcomes analysed.

RESULTS: A total of 27 DCD and 73 DBD islet donor pancreata were compared with no significant differences seen in donor characteristics between DCD and DBD. Isolation outcomes showed post-purification yield (IEQ) was significantly lower in DCD group (146,518±28,971) compared to DBD (256,986±17,652; P=0.001). Post-purification yield gm/pancreas was significantly lower in DCD (2,154±504 vs. 2,681±372 IEQ/g, P<0.0001). The quality and functionality of DCD and DBD islets were also significantly different in terms of the viability (%) – P=0.017 (higher in DBD than DCD), purity (%) – P=0.001 (higher in DBD than DCD). The proportion of DCD islets transplanted (1/27) was significantly lower than DBD (29/73) going to transplant (OR, 0.1093; 95% CI; P=0.001).

CONCLUSION: In the Australian setting with vast distances to ship pancreata we have had poorer outcomes from DCD pancreata for islet isolation and have thus far not yielded outcomes comparable to those from our DBD donors. Earlier intervention, the use of ante mortem heparin and faster logistics in transport may not only improve the DCD organs for transplantation but also help alleviate donor shortages allowing treatment of those with T1DM and severe hypoglycaemic unawareness.

Abstract No. 30

COMPARISON OF PANCREATA AND ISLET PREPARATIONS FROM HUMAN ORGAN DONORS

MARIANA Lina, LOUDOVARIS Thomas, KOS Cameron, PAPAS Evan, SELCK Claudia, CATTERALL Tara, THOMAS Helen, KAY Thomas WH

Immunology & Diabetes, St Vincent's Institute, Melbourne

Background In the past ten years we have received over 300 pancreata and most were processed into islets for either transplant and/or research. Forty-nine of the donors resulted in transplants (into 26 diabetic recipients, 5 as autotransplants), 49 were diabetic (T1D and T2D), 24 were from non-heart beating donors (DCD) and the remaining were heart beating brain dead (BD) donors. Many factors influence the outcome of isolating islets. Here we compare the characteristics of the donor, pancreas and islet preparations of transplantable isolations with isolations that failed to meet transplant criteria, including diabetic and DCD.

Methods Islets were isolated based on the Ricordi Method. The Edmonton Score used indicates donor quality and incorporates age, CIT, BMI, cause of death, hospital stay, amylase/lipase, procuring team, medical history pancreas fat content, quality of flush and damage.

Results

As shown in the table, the quantity of islets/g pancreas in diabetic donors was significantl			TX	BD	DCD	T1D	T2D
	Edmonton Score		72.8±10.3	60.3±10.2*	57.3±10	54.4±9.4	53.1±9.0
	Islet Yield (IEQ/g)	Pre-Purification	6836±3498	4502±2463	5055±5659	459±346	2547±1991
		Post-Purification	4357±3108	1809±1401	2601±5220	136±123	1170±948
	Insulin Secretion (Stimulation index)		3.28	1.69*	1.82*	n/a	1.18*
	Pancreas Weight (g)		104.6±29.1	100.9±28.7	98.2±31.8	29.1±7.4*	96.9±36.9

than non-diabetic donors both pre-purification and post-purification. Similar results were found with glucose stimulated insulin secretion among the group. T1D pancreata were not only deficient in islet numbers but their pancreas size was significantly smaller compared to the other groups.

While there was no difference in body weight between the two groups, T2D: 86.02±16 kg vs non-diabetic: 85.07±21 kg, the IEQ/kg body weight was significantly different, T2D: 1907±1410 vs non-diabetic: 4344±2577 (t-test p<0.0001).

Conclusion The quality of donors in the transplant group was significantly higher than the other groups as measured by the Edmonton Score. T2D donors are insulin resistant and have islet function deficiencies. Our data show that fewer islets can be isolated from T2D than non-diabetic donors, further validating their exclusion as an islet transplant donor.

Abstract No. 31

THE PREVALENCE OF ACQUIRED CYSTIC KIDNEY DISEASE (ACKD) IS NOT INCREASED IN RENAL TRANSPLANT RECIPIENTS WITH RENAL TUMOURS.

RHEE Handoo, TAN Ai Lin, GRIFFIN Anthony, PRESTON John, LAWSON Malcom, WOOD Simon
Renal Transplant Unit, Princess Alexandra Hospital, Brisbane

Aim: To determine the association between ACKD and renal malignancies in the renal transplant population.

Methods: The study included literature review using PubMed and CINAHL according to the PRISMA guideline. Using the key words ((*transplant*[Title] and renal*[title]) AND (cancer*[Title] or mass*[Title] or malignan*[Title] or carcinoma*[Title] or lesion*[title])).

Results: In 13 studies that assessed specifically at the presence of ACKD in the context of renal malignancies, 59.95% (n=241/402) of patients with renal tumours had concurrent bilateral, and multiple cysts in the native kidneys. There was only 1 case report of ACKD associated clear cell RCC in the allograft. In renal transplant-population studies, 1% (n=97/9740) developed renal malignancies over 3 years. Of 97 patients, 67 (69%) had ACKD. Most studies identified ACKD with either routine ultrasound or computed tomography and only 4 patients presented with a symptom. 4 cancer specific deaths were reported during the follow up. Papillary renal cell carcinoma (RCC) was the most common (43%) subtype.

Discussion: Historically, ACKD has been reported in 40-60% of patients undergoing renal replacement therapy (a figure similar to ACKD found in patients with renal tumours). Although the presence of ACKD has been thought to be a strong predictor/harbinger of diagnosis of renal tumours in the future, this study raises the question: is ACKD a function of chronic insult to the kidneys or a cause of future malignancies. Understanding the aetiology of ACKD and associated RCC may provide insight into papillary RCC which currently has poor prognosis with metastasis.

Abstract No. 32

COMPARISON OF TUMOUR CHARACTERISTICS IDENTIFIED IN THE ALLOGRAFT AND THE NATIVE KIDNEYS OF RENAL TRANSPLANT RECIPIENTS

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Renal Transplant Unit, Princess Alexandra Hospital, Brisbane

Aims. The aim of this study is to determine the difference in the characteristics of renal tumours in the allograft or native kidneys of renal transplant recipients. Elucidating the subtleties may aid in the understanding of the pathophysiology behind cancer development in renal transplant recipients.

Methods. The study included literature review using PubMed and CINAHL according to the PRISMA guideline. Using the key words ((*transplant*[Title] and renal*[title]) AND (cancer*[Title] or mass*[Title] or malignan*[Title] or carcinoma*[Title] or lesion*[title])).

Results. Allograft tumours had a trend for papillary renal cell carcinoma (RCC) (37% vs 31%, p=0.00956) and less clear cell RCC (35 vs 41%, p=0.138). The size of the lesions in the allograft was smaller but not statistically significant (3.23 vs 3.97cm, p=0.433). The time to cancer development from renal transplant was however, longer with the allograft (10.63 vs 8.63 yrs, p=0.00244). Overall, the risk of cancer recurrence and cancer specific survival was also similar (88.4% (native) vs 90.8% (allograft)).

Conclusions. Given the similarities between the tumours identified in the allograft and the native kidneys in renal transplant recipients, similar pathophysiology maybe behind the development of renal tumours. This hypothesis is supported by previous reports where papillary RCC is slightly more dominant in ESRF population without transplant. Other cancer-associated aetiologies such as chronic inflammation may be a consideration. For example, papillary RCC is significantly more common in this population (15% in the general population). Papillary RCC has been associated with chronic inflammation markers such as IL-8.

Abstract No. 33**INFECTIOUS COMPLICATIONS IN THE SOUTHERN TASMANIAN KIDNEY TRANSPLANT POPULATION****ABEYSEKERA N¹, GRAVER A², COOLEY L³, KIRKLAND G², JOSE MD^{1,2}**¹*School of Medicine, University of Tasmania*, ²*Renal Unit, Royal Hobart Hospital*, ³*Infectious Diseases Unit, Royal Hobart Hospital*

Aim To examine the infectious complications of kidney transplant recipients (KTRs), including the number of episodes, use of antimicrobials and episodes of hospitalisation.

Methods All KTRs managed by the Royal Hobart Hospital between 1st of January 2015 and 31st of December 2016 were included. During this period, data regarding all infectious episodes, antibiotic use, hospital admissions and patient survival was obtained from medical records.

Results Of the 163 KTRs (mean age 44 years, 40% female), 98 developed 365 infectious episodes. Females were more likely to have an infectious episode ($p=0.033$) due to the high rate of urinary tract infections. Overall, 50 infectious episodes required hospital admission, for a total of 227 admitted bed days (range 1-18 days). Causative organisms were bacterial ($n=217$), viral ($n=74$) and fungal ($n=13$); 61 episodes were culture-negative. The most commonly isolated organism was *Escherichia coli* ($n=61$), followed by *Enterococcus faecalis* ($n=46$). There were 19 cases of multi-resistant organisms in 10 patients. Overall, these 98 KTRs received a total of 250 courses of antimicrobials.

Conclusion Episodes of infection, use of antimicrobials, hospitalisation and development of multi-resistant organisms are common following kidney transplantation in this southern Tasmanian cohort. This study emphasises the need to evaluate prophylaxis and management guidelines for infectious episodes in KTRs, due to the high frequency and potential for adverse outcomes, including development of antibiotic resistance and hospital admission.

Abstract No. 34**CLEARANCE OF BK VIRUS NEPHROPATHY BY COMBINATION ANTIVIRAL THERAPY WITH INTRAVENOUS IMMUNOGLOBULIN****KABLE Kathy, DAVIES Carmen, O'CONNELL Philip, CHAPMAN Jeremy, NANKIVELL Brian***Department of Renal Medicine, Westmead Hospital, Sydney*

Background: Reactivation of BK polyoma virus causes a destructive virus allograft nephropathy (BKVAN) with graft loss in 46%. Treatment options are limited to reduced immunosuppression and largely ineffective antiviral agents. Some studies suggest benefit from intravenous immunoglobulin (IVIG).

Methods: We evaluated effectiveness of adjuvant IVIG to eliminate virus from blood and tissue, in a retrospective, single-centre cohort study, against standard-of-care controls. Both groups underwent reduced immunosuppression, conversion of tacrolimus to cyclosporine, and mycophenolate to leflunomide, oral ciprofloxacin, and intravenous cidofovir.

Results: Biopsy-proven BKVAN occurred in 50 kidneys at 7 (median, IQR 3-12) months after transplantation, predominantly as histological stage B (92%), diagnosed following by dysfunction in 46%, screening viremia in 20%, and protocol biopsy in 34%. Following treatment, mean viral loads fell from $1,581 \pm 4,220 \times 10^3$ copies at diagnosis to $1,434 \pm 7,063$ mid-treatment, and 0.138 ± 0.331 after 3 months ($P < 0.001$). IVIG at 1.01 ± 0.18 g/kg was given to 22 patients (44%). The IVIG group more effectively cleared viremia (HR 3.68; 95% CI, 1.56-8.68, $P=0.003$) and BK immunohistochemistry from repeated tissue sampling (HR 2.24; 95% CI, 1.09-4.58, $P=0.028$), and resulted in faster (11.3 ± 10.4 vs. 29.1 ± 31.8 months, $P=0.015$) and more complete resolution of viremia (33.3% vs. 77.3%, $P=0.044$). Numerically fewer graft losses occurred with IVIG (27.3% vs. 53.6% for control, $P=0.06$), although graft and patient survivals were not statistically different. Acute renal dysfunction requiring pulse corticosteroid was common (59.1% vs. 78.6%, $P=0.09$), respectively, following immunosuppression reduction.

Conclusions: Combination treatment incorporating adjuvant IVIG was more effective eliminating virus from BKVAN, compared with conventional therapy. Validation by multi-centre randomised trial is needed.

Abstract No. 35**FIRST REPORTED CASE OF GANCICLOVIR-RESISTANT POST-TRANSPLANT CYTOMEGALOVIRUS INFECTION DUE TO COMBINED DELETION MUTATION IN CODONS 595-596 OF THE UL97 GENE****LEUNG Po Yee Mia¹, TRAN Thomas², TESTRO Adam³, PAIZIS Kathy¹, KWONG Jason⁴, WHITLAM John^{1,5,6}*****¹Department of Nephrology, Austin Hospital, Melbourne, ²Virus Identification Laboratory, Victorian Infectious Diseases Reference Laboratory, ³Liver Transplant Unit Victoria, Austin Hospital, Melbourne, ⁴Department of Infectious Diseases, Austin Hospital, Melbourne, ⁵Department of Medicine, University of Melbourne, ⁶Murdoch Childrens Research Institute, Royal Children's Hospital, Melbourne***

The development of antiviral resistant cytomegalovirus (CMV) infection significantly complicates management of transplant patients.

Case: We describe a case of a 65-year-old male who developed breakthrough CMV disease (donor CMV IgG positive, recipient CMV IgG indeterminate) 30-days after a combined liver-kidney transplantation for alcoholic cirrhosis and hepato-renal syndrome. After an initial complete response to treatment dose of oral valganciclovir, he developed recurrent CMV viraemia. Resistance testing revealed a UL97 mutation with in-frame deletions of codons 595-596. He was treated successfully with foscarnet and reduction in immunosuppression. This mutation has not been previously described and was suspected to confer ganciclovir resistance, which was supported by his clinical course.

Discussion: Ganciclovir resistance occurs most commonly due to mutations in the UL97 gene or the UL54 gene, which encode a protein kinase and a DNA polymerase, respectively. The UL97-encoded protein kinase phosphorylates ganciclovir to ganciclovir-triphosphate, which competitively inhibits viral replication. Mutations in the UL97 gene are typically point mutations or deletions that prevent phosphorylation of ganciclovir to its active form.

Conclusion: We describe the case of a mutation, del595-596, in the CMV UL97 gene in the context of clinical treatment failure with standard and double-dose ganciclovir and successful virological control was achieved with foscarnet. Given the location of the mutation in the UL97 gene, it is likely to result in ganciclovir resistance, though recombinant phenotyping is required for confirmation.

Abstract No. 36

EMPHYSEMATOUS PYELONEPHRITIS IN A DUAL KIDNEY TRANSPLANT RECIPIENT**TANGIRALA Nishanta¹, SINGER Julian¹, ANDERSON Lyndal², LAURENCE Jerome³, GRACEY David¹**¹*Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney,* ²*Department of Pathology, Royal Prince Alfred Hospital, Sydney,* ³*Department of Surgery, Royal Prince Alfred Hospital, Sydney*

Case: A 54 year old diabetic man presented with one week history of fevers and abdominal pain. He received a dual-kidney deceased donor transplant thirteen months prior, achieving excellent graft function at one year (serum creatinine 97 μ mol/L). At presentation the patient was anuric with a serum creatinine of 518 μ mol/L and a white cell count of 25.32×10^9 /L. A non-contrast computed tomography scan revealed an enlarged right iliac fossa kidney with gas throughout the renal parenchyma and transplant renal vein, extending into the great saphenous veins (Figure 1). There was no air within the collecting system and the second allograft was grossly normal. The patient received antibiotics and underwent an urgent allograft nephrectomy of the emphysematous kidney. The second renal allograft was left in situ. *Escherichia coli* was grown in blood and urine cultures. Antibiotics and circulatory support were administered. Haemodialysis was required for fourteen days post nephrectomy prior to recovery of the remaining allograft. Eight weeks post nephrectomy the serum creatinine was 180 μ mol/L.

Discussion: Emphysematous pyelonephritis (EPN) is a fulminant, necrotizing infection of the renal parenchyma, from gas producing organisms. There have been 26 reported cases of EPN in renal allografts, however, to our knowledge this is the only reported case in a patient with dual allografts. It remains a rare but devastating complication following transplant, and is associated with high risk of mortality. Early recognition and prompt nephrectomy can be life saving, and in this case also enabled retention of the remaining allograft leading to dialysis free survival.



Figure 1

Abstract No. 37

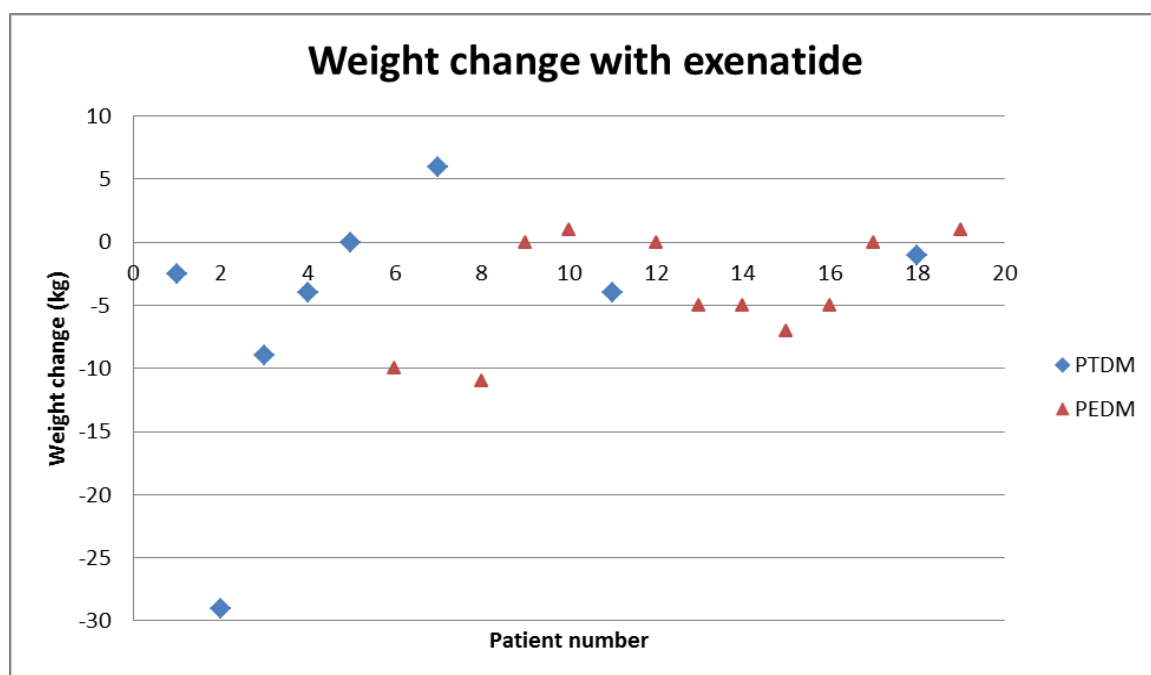
GLP1RA SUCCESSFULLY TREATS HYPERGLYCAEMIA IN RENAL TRANSPLANT RECIPIENTS AND ENABLES SUBSTANTIAL REDUCTION IN INSULIN REQUIREMENTS AND WEIGHT**KAMESHWAR Kamya¹, FOURLANOS Spiros^{2,3}, HIDAYATI Leny¹, CHEONG Jamie¹, LEVIDIOTIS Vicki¹, COHNEY Solomon^{1,3}**¹*Department of Nephrology, Western Health, Victoria, Australia,* ²*Department of Endocrine and Metabolism, Royal Melbourne Hospital,* ³*Faculty of Health Sciences, University of Melbourne*

Background: While the range of glucose lowering therapies for diabetes mellitus (DM) has recently increased, CKD, ESKD and renal transplant recipients continue to be largely restricted to sulphonylureas and insulin, with potential for weight gain and hypoglycaemia. Of additional concern, obesity now commonly complicates ESKD and transplant management. GLP1 receptor agonists (GLP1RA) facilitate weight loss, carrying minimal risk of hypoglycaemia when used without insulin or sulphonylureas. We studied 19 renal transplant recipients receiving GLP1RA for DM predating transplantation (PEDM) or post-transplant diabetes mellitus (PTDM).

Methods: Nineteen patients taking B.D. exenatide (Byetta) or weekly exenatide (Bydureon) were studied prospectively following renal transplantation (11 PEDM, 8 PTDM).

Results: Patients were on average 60 months post-transplant (range 7 to 120); 11 on insulin (10 PEDM, 1 PTDM), mean weight 87kg (range 64 to 108), mean HbA1c 7.8% (range 6.1 to 9.7), mean creatinine 125µmol/L (range 65 to 187). After a median 10.5 months (range 3-36), 3 PEDM patients were insulin free, while the remainder had a 60% reduction in insulin requirements (IR). Weight fell by a mean 4kg (median -4, range -29 to +6) with weight loss greatest in those with highest baseline IR. HbA1c fell on average by 0.1% (range -2.6 to 1.8). Significant gastrointestinal side-effects occurred in 8 patients receiving Byetta, 2 tolerated half-maximal dose, and a third successfully converted to Bydureon.

Conclusion: GLP1RA in renal transplant recipients with diabetes enabled reduction in IR and weight, with improved glycaemic control in some patients. Further evaluation in larger randomised trials is warranted.



Abstract No. 38

ONE YEAR INCIDENCE & PREVALENCE OF NEWLY DETECTED ABNORMAL GLUCOSE METABOLISM IN RENAL TRANSPLANT PATIENTS ON MAINTENANCE PREDNISOLONE AND CNI (2004-2009)**PIMENTEL AL^{1,2}, MASTERTSON R², YATES C^{3,4}, HUGHES P², CAMARGO JL^{1,5}, COHNEY S^{6,7,8}**¹*Graduate Program in Endocrinology, Universidade Federal do Rio Grande do Sul (UFRGS),* ²*Department of Nephrology, Melbourne Health,* ³*Department of Diabetes and Endocrinology, Melbourne Health,* ⁴*Department of Endocrinology, Western Health,* ⁵*Department of Endocrinology, Hospital de Clinicas de Porto Alegre (HCPA),* ⁶*Department of Nephrology, Western Health,* ⁷*Department of Medicine, University of Melbourne,* ⁸*Department of Epidemiology, Monash University, Melbourne*

Aims: The reported incidence of newly diagnosed diabetes after renal transplantation (PTDM) varies widely according to definitions and immunosuppressive regimen. Data on PTDM incidence amongst patients receiving modest corticosteroid and lower maintenance CNI is sparse. This study analysed PTDM incidence within a single centre cohort of 534 consecutive patients transplanted from 2004 to 2009, and followed to 2017.

Methods: Patients received a single methylprednisolone pulse ≤ 500 mg at transplant, 20 to 25 mg prednisolone for the first month tapered to 5mg by 8 to 12 weeks, tacrolimus ≤ 7 ng/ml after week 8, and ≤ 4 ng/ml beyond 12 months with analysis based on a combination of prospectively recorded data from an electronic database, medical records and ANZDATA. Pre-existing diabetes mellitus (PEDM) and PTDM diagnosed during the first year post-transplant were based on glucose levels, HbA1c and/or use of glucose lowering therapy (GluLT).

Results: Of 534 patients, 63 had PEDM and 64 (13.6%) developed PTDM during the first year, 83% of the cases occurring in the first 3 months (Table 1). Amongst those with PTDM, 48 started GluLT - predominantly metformin, insulin and/or gliclazide. Glucose levels returned to normal in 6 patients who discontinued GluLT, leaving a PTDM prevalence of 12.3% at one year. Twelve patients had transient hyperglycaemia that normalized within days/weeks without treatment and were not considered PTDM.

Conclusions: In this single centre cohort of renal transplant recipients maintained on low dose tacrolimus, mycophenolate and prednisolone, the 1-year incidence and prevalence of PTDM were 13.6% and 12.3% respectively, with 75% on any GluLT.

Table 1. Characteristics of patients in the study.

Characteristics	N = 534
Age (years)	45.2 \pm 14.0
Gender (% men)	64.6
Deceased donor (%)	43.3
First transplant (%)	83.6
Weight at transplant (kg)	76.6 \pm 15.6

Data are expressed as mean \pm SD or frequencies (%).

Abstract No. 39**METFORMIN, GLICLAZIDE AND INSULIN REMAIN THE MOST COMMONLY USED AGENTS FOR POST-TRANSPLANT DIABETES (PTDM) IN A COHORT OF RENAL TRANSPLANT RECIPIENTS**
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Aims: Given the paucity of literature on treatment of PTDM, this study was undertaken to analyse glucose lowering therapy (GluLT) usage in a sizeable cohort of PTDM patients.

Methods: Review of all renal transplant recipients transplanted between December 2004-2009 followed to December 2017 using data collected prospectively from an electronic database and medical records. HbA1c, glucose levels and/or use of hypoglycemic therapy identified patients with PTDM.

Results: Amongst 534 patients, 86 developed PTDM with 59 commencing GluLT: 3 insulin monotherapy, 11 metformin, 12 gliclazide. An additional 29 received metformin in combination with other therapies (including 14 taking insulin), 3 on insulin in combination with other drugs and 1 patient on gliclazide and linagliptin. Glucose metabolism normalised by 12 months in 6 patients, and GluLT was discontinued. 23/40 patients commencing metformin remained on it at end of follow-up; cessation of metformin was due to resolution of PTDM in 4 patients, 1 when renal function deteriorated, 2 because of gastrointestinal symptoms, and uncertain in 9 patients (1 lost to follow-up. No cases of lactic acidosis were reported. Eleven patients commenced on newer GluLT (6 GLP1 receptor agonist, 3 SGLT2i, 7 DPP4i). There was no indication of any difference in outcome according to GluLT.

Conclusions: Insulin, metformin and gliclazide were the commonest glucose lowering therapies prescribed with intolerance to Metformin uncommon. Prospective studies on GLuLT are needed preferably with outcome data and randomized if possible.

Abstract No. 40**ABSENT SMOOTH MUSCLE ACTIN IMMUNOREACTIVITY OF THE SMALL BOWEL MUSCULARIS PROPRIA CIRCULAR LAYER – A NOVEL FINDING IN A DYSMOTILE INTESTINAL ALLOGRAFT**
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Background: Intestinal dysmotility leading to poor graft function is a recognized complication of intestinal transplantation however the causes are poorly understood.

Aim To describe a novel potential cause of dysmotility in an intestinal graft

Case report: A 14 year old male, underwent combined liver/intestinal transplant for Hirschsprungs disease in March, 2012. 3 years post-transplant he developed recurrent episodes of increased stoma losses, nausea and abdominal pain requiring repeated hospital admissions. Rejection, viral infection, bacterial overgrowth and PTLTD were excluded. Exploratory laparotomy revealed no stricture or obstruction. Around 5cm of proximal end of donor jejunum was resected and a Roux loop jejunojejunostomy was created. The roux loop was pulled up to create a gastrojejunostomy. This procedure relieved the symptoms. Histopathology of the resected allograft specimen showed an abrupt segmental disappearance of the inner, circular layer of muscularis propria in the donor jejunum. Immunohistochemical staining of multiple blocks showed almost complete loss of staining for smooth muscle actin in the inner, circular layer of muscularis propria with preservation in the outer layer. This finding has been described in chronic intestinal pseudo-obstruction and in megacystis-microcolon. Occurrence of these changes in the jejunum, as seen in our patient, is pathological. **Conclusion:** We present a novel finding of acquired loss of alpha-smooth muscle actin immunostaining in circular muscle myocytes in an intestinal allograft which may be a cause of dysmotility. The cause of this acquired change is uncertain but immune mediated destruction of enteric myocytes by host immune cells is worthy of further exploration.

Abstract No. 41

INTRACTABLE ASCITES FOLLOWING RENAL TRANSPLANT IN AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE PATIENTS WITH MASSIVE POLYCYSTIC LIVER**MARUI Yuhji¹, FUJIMOTO Eisuke¹, AIDA Koichiro¹, SASAKI Hideo¹, KOIZUMI Satoshi², OTSUBO Takehito², CHIKARAISHI Tatsuya¹****¹Department of Urology, St Marianna University School of Medicine, ²Division of Gastro-enterological and General Surgery, St Marianna University School of Medicine**

Aim: To consider the mechanism of the intractable ascites developed in renal transplant (RTx) recipients with autosomal dominant polycystic kidney disease (ADPKD) with massive polycystic liver. **Case 1:** A 60s year-old man developed massive ascites 3 years after RTx despite bilateral diminishment of native kidneys. MRI revealed multiple liver cysts distributing throughout all hepatic segments, and extrinsic compression of inferior vena cava (IVC). (Figure) The ascites became symptomatic and resistant to medical treatment with diuretics and paracentesis. As any surgical revisions were unsuitable, he underwent peritoneovenous shunt placement. Then the ascites was managed by least diuretics with durable relief of symptoms, and renal function improved. **case 2:** A 50s year-old man with ADPKD who had undergone pre-emptive RTx and simultaneous left nephrectomy 4 years before, developed massive ascites following acute colitis. MRI revealed massively enlarged polycystic liver and severe stenosis of the intrahepatic part of IVC. As the ascites became refractory to medical treatment, he underwent hepatic resection and cyst fenestration. After these procedure the ascites resolved followed by improvement of renal graft function. **Discussion:** In these recipients, in addition to enlarging multiple hepatic cysts, the lost of supporting effect by bilateral renal enlargement buttressing up the liver might have caused that liver shifted inferiorly, which resulted in severe stenosis of the intrahepatic part of IVC. In case 2 due to colitis induced dehydration the consequent reduction of intracaval blood flow might have led critical stenosis of IVC. The improvement of graft function following intervention was noteworthy.

Figure



Abstract No. 42**RENAL TRANSPLANTATION IN THE ELDERLY POPULATION: SURGICAL OUTCOMES IN THE QUEENSLAND NETWORK OVER A 10 YEAR PERIOD.****FADAE Neesa, ROBERTSON Ian, RHEE Handoo, GRIFFIN Anthony***Renal Transplant Unit, Princess Alexandra Hospital, Brisbane*

Aims To analyse the surgical outcomes of kidney transplantation in patients aged over 70 including postoperative complications and graft function 3 months post-transplantation.

Method A retrospective analysis of a prospectively maintained database was completed. Patients aged 70 years and older who received a kidney transplant at the Princess Alexandra Hospital over a 10 year period (January 2007-December 2017) were included in the study. Data was collected by completing patient chart reviews and results were analysed to determine surgical outcomes including graft function, dialysis requirements and creatinine levels three months post-transplantation.

Results There were 46 patients included in this study with the mean age at the time of transplant being 71.8 years. 38 patients received a cadaveric graft. Majority of patients (n=28) had delayed graft function with only 43.5% (n=20) of patients reaching a normal creatinine (Cr 80-110) by 3 months. 15% (n=7) required dialysis within 72 hours post-op. The most common surgical complications included postoperative anaemia (n=5) and perinephric collection/haemorrhage (n=4). Only 2 patients required return to theatre with 1 patient requiring a transplant nephrectomy. The two most common post-transplant medical diagnoses were osteoporosis (n=14) and type 2 diabetes (n=12).

Conclusion An ageing population with end stage renal disease combined with advances in transplantation have led to an increasing number of transplants in the over 70s population. It is crucial to identify complications of transplantation and assess their impact on graft function. Further research through a longitudinal or prospective comparative study is required.

Abstract No. 43

EARLY REMOVAL OF JJ STENTS IN RENAL TRANSPLANT RECIPIENTS : A PILOT STUDY OF FEASIBILITY AND SAFETY.

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Background: JJ stents are routinely inserted intra-operatively in renal transplant recipients (RTR) to minimize early post-surgical complications including urinary leak and ureteric obstruction. Usual clinical practice is to remove the JJ stents cystoscopically at 4-6 weeks. However, the optimum duration of the stents has not been established. Stent placement can cause dysuria, urinary tract infections, ureteric epithelial ulceration and inflammation with longer dwells. Use of JJ stents was reported to be a risk factor for developing BK Virus infection. Studies have looked at reducing the complications of JJ stents by early removal.

Methods: After extensive literature review and discussions, we have initiated early JJ stent removal protocol in RTR. Intra operatively, JJ stent is attached to the Indwelling Catheter (IDC) by tying the distal end of the JJ stent string to the IDC. The IDC and JJ stent are together removed by the bedside on post transplant Day 5.

RESULTS: In the 4 RTR trialed so far (3 males; age 17-61 years), early stent removal has been achieved satisfactorily, with excellent patient acceptance and no clinical or imaging evidence of any complications. 2 RTR received kidneys from live donors, and 2 from deceased donors.

CONCLUSIONS: In this ongoing safety and feasibility study, we aim is to document the safety and efficacy of early removal of JJ stents in a larger cohort. We envisage a prospective randomized study for documenting the efficacy of this approach in preventing major urologic complications and in reduction of BKV infection in RTR.



Photograph of removed JJ stent with IDC on Post Transplant Day 5 by the bedside

Abstract No. 44**OUTCOMES OF EARLY URETERIC STENT REMOVAL IN PAEDIATRIC KIDNEY TRANSPLANTATION****NG Zi Qin¹, HE Bulang^{1,2}**¹*WA Liver & Kidney Transplant Service, Sir Charles Gairdner Hospital, Perth,* ²*Department of Surgery, University of Western Australia, Perth*

Introduction In kidney transplantation, the timing for removal of ureteric stent is debatable and usually it is removed at 4-6 weeks post-surgery by cystoscopy. The aim of this study is to review the outcomes of early removal of ureteric stent simultaneously with removal of the indwelling urethral catheter in paediatric transplant recipients.

Methods & Materials A retrospective review was performed for all paediatric transplant recipients from 2009 to 2017. The refinement to kidney transplantation was that the end of ureteric stent was connected to the tip of indwelling urethral catheter (IDC) by a suture-string. As such the stent can be removed concurrently at the time of IDC removal 5-7 days after transplantation. The data of demographics, episode of infection, urological complications and kidney function was collected for analysis.

Results Overall, there were 28 cases of paediatric kidney transplantation, age from 2 to 18 (median 10.5 years). There were 23 males and 5 female patients. Twenty-six patients had early stent removal. There were no cases of urine leakage. One recipient developed distal ureteric stenosis which resolved after interventional balloon dilatation. Two cases developed BK nephropathy 4-6 months post transplantation. One case had urinary tract infection (< 3 months).

Conclusion Early removal of ureteric stent is safe and feasible without increasing the risk of urological complications. It helps significantly in cost-saving and improves patient quality of life.

Abstract No. 45**PARTIAL VERSUS COMPLETE THROMBOSIS MODERATED BY INTRA-OPERATIVE VASOPRESSOR USE IN SPK PATIENTS****SHAHRESTANI Sara^{1,2}, HORT Amy³, SPIKE Erin³, GIBBONS Thomas¹, HITOS Kerry³, ROBINSON Paul⁴, KABLE Kathy⁴, LAM Vincent³, DE ROO Ronald³, YUEN Lawrence³, PLEASS Henry³, HAWTHORNE Wayne²**¹*Western Clinical School, University of Sydney,* ²*Centre for Transplant and Renal Research, Westmead Hospital, Sydney,* ³*Department of Surgery, Westmead Hospital, Sydney,* ⁴*Department of Renal Medicine, Westmead Hospital, Sydney*

Aims: Simultaneous pancreas-kidney (SPK) transplantation is the gold standard treatment for patients with type 1 diabetes and end stage renal failure. Thrombosis is a devastating complication of SPK that can result in graft loss and return to theatre for pancreatectomy.

Methods: We reviewed 235 SPKs performed at Westmead hospital over the past decade (2008-2017). We examined risk factors (donor and recipient) and characteristics of thrombosis in order to ascertain the clinical course for patients.

Results: 41 (17.4%) of patients experienced a thrombosis. In 85% (35/41) of cases, this thrombosis occurred early, in the first 6 weeks following transplantation. The majority of thromboses (68%, n=28/41) were venous. Importantly thrombosis associated with graft loss and pancreatectomy only occurred in less than half of the patients with a thrombosis (n=17, 7.2%). Graft loss was strongly associated with the use of intraoperative vasopressors with 71% (n=12/17) of the patients that lost their graft requiring intraoperative vasopressors, while only 46% (n=11/24) of those with partial thrombosis required this intervention.

Conclusion: While graft thrombosis is a devastating complication of SPK transplantation that leads to graft loss, it is reassuring to know that less than half of the grafts that thrombose are lost and require return to theatre for pancreatectomy. A strong risk factor for thrombosis leading to graft loss is the use of intra-operative vasopressors, leading us to believe careful management of blood pressure may be key to reducing the devastating outcomes of this not uncommon complication.

Abstract No. 46

MACHINE LEARNING PREDICTION FOR DE NOVO DONOR SPECIFIC ANTIBODIES (dnDSA) AND GRAFT LOSS IN SIMULTANEOUS KIDNEY PANCREAS TRANSPLANT (SPK) RECIPIENTS**COOREY Craig^{1,2}, SHARMA Ankit^{1,2}, CHAPMAN Jeremy³, CRAIG Jonathan^{1,2}, O'CONNELL Philip³, LIM Wai⁴, NANKIVELL Brian³, TAVERNITI Anne², WONG Germaine^{1,2,3}, YANG Jean^{5,6}**¹*School of Public Health, University of Sydney*, ²*Centre for Kidney Research, The Children's Hospital at Westmead, Sydney*, ³*Centre for Transplant and Renal Research, Westmead Institute for Medical Research, Westmead Hospital, Sydney*, ⁴*Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth*, ⁵*School of Mathematics and Statistics, University of Sydney*, ⁶*Charles Perkins Centre, University of Sydney***Aim:** To develop a prediction model for *dnDSA* and allograft loss based on the location of eplet mismatches in SPK transplant recipients.**Methods:** A total of 198 SPK transplant recipients (1990-2017) were assessed using data from ANZDATA registry and National Organ Matching System. Machine learning models (random forests) were used to predict *dnDSA* and allograft loss based on the location of eplet mismatches. The sites of the three most important eplet mismatches were determined using 'mean decrease in accuracy'.**Results:** The cohort included 111 (56%) males, mean age: 38.5 years (SD 6.9) and median follow up time of 6.6 years (IQR: 3.9,11.0). The most common Class I and II eplet mismatches were at 156RA (35%), 82LR (35%) and 76EN (33%); and 70D (55%), 56PD (54%) and 67I (52%), respectively. A total of 38 (20%) and 56 (32%) recipients developed Class I and II *dnDSA* and 14 (7%) and 29 (15%) patients experienced kidney and pancreas graft loss. Random forest model with the location of eplet mismatches as features achieved a mean cross-validation error of 47.6% and 49.8% for Class I and II *dnDSA*, 52.3% and 49.1% for kidney and pancreas allograft losses (Table 1). For *dnDSA* prediction, the three most important Class I eplet mismatches are present in the HLA A antigens, while for Class II eplet mismatches, only DQB1*03 is implicated.**Conclusions:** The location of the most important eplet mismatches for prediction differed between *dnDSA* and allograft loss, but random forest model performance was largely indistinguishable.**Table 1: Performance of a 100 times 5-fold cross-validated random forest model in predicting *dnDSA* and allograft loss**

Models	Sites of the three most important eplet mismatches	50% discrimination threshold			Area under the curve (AUC) (SD)
		Mean balanced error rate (SD)	Sensitivity (SD)	Specificity (SD)	
Class I <i>dnDSA</i> from Class I eplets	102HV, 44RM, 71HS	47.6 (1.8)	6.2 (3.3)	98.7 (0.8)	57.6 (4.7)
Class II <i>dnDSA</i> from Class II eplets	55PP, 52PL, 140T	49.8 (2.2)	18.7 (3.5)	81.8 (2.6)	52.9 (2.8)
Kidney allograft loss from Class I and II eplets	13FE, 156QA, 13SE	52.3 (4.9)	54.4 (10.7)	40.9 (4.2)	54.2 (4.8)
Pancreas allograft loss from Class I and II eplets	57V, 60S, 150AAH	49.1 (4.3)	45.0 (8.2)	56.8 (3.6)	53.8 (3.6)

Abstract No. 47

HLA EPLET MISMATCH AND DONOR SPECIFIC ANTIBODIES IN KIDNEY TRANSPLANTATION**WAN Susan^{1,2}**, ANGEL DE WILDE Sian², ROSALES Brenda³, CHADBAN Steven^{1,4}, WYBURN Kate^{1,2}¹*Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney*, ²*Sydney Medical School, University of Sydney*, ³*School of Public Health, University of Sydney*, ⁴*Other, University of Sydney*

BACKGROUND: Eplet mismatch provides higher resolution information than HLA and has potential to better predict alloimmune events, including donor specific antibodies (DSA). However, limited prospective data exists on the association between eplet mismatch and DSA.

AIM: To determine the relationship between eplet mismatch and DSA.

METHODS: We characterised the number of HLA-A, B, C, DR and DQ eplet mismatches in kidney transplant recipients from 2010-2017 using HLA Matchmaker. Molecular HLA typing was converted from low-resolution (2-digit) to high-resolution (4-digit) using HLA Matchmaker Converter where necessary. All patients were prospectively screened for DSA at 0, 3 and 12-months post-transplant. Associations between eplet mismatches, pretransplant DSA (preDSA), *denovo* DSA (dnDSA) and clinical outcomes were assessed using multivariable analysis.

RESULTS: Of 313 recipients, high-resolution HLA conversion was not possible for 147 (47%) due to the absence of ethnicity (n=83) or haplotype (n=64) data in the Converter database. Eplet mismatch determination was therefore possible for 166 donor-recipient pairs, of whom DSA screening was complete for 150. The mean number of Class I and II eplet mismatches was 14(±7.7) and 17(±11.8) respectively (Table 1). DSA was detected in 111 recipients (74%); 64 (43%) had preDSA, 30 (20%) had dnDSA, and 17 (11%) had both. The number of eplet mismatches was associated with preDSA (OR 1.04; 95% CI 1.01-1.07; P=0.007), but not with dnDSA or acute rejection.

CONCLUSION: Calculated eplet mismatches were not predictive of dnDSA development or acute rejection, raising doubt about the utility of HLA Matchmaker based eplet matching to predict post-transplant alloimmune events.

Table 1: Mean Number of Eplet Mismatches by Donor Specific Antibodies (DSA)

<i>Eplet Mismatches</i>	<i>All Recipients</i>	<i>No DSA</i>	<i>Pretransplant DSA</i>	<i>dnDSA</i>
	<i>N=166</i>	<i>n=89</i>	<i>n=64</i>	<i>n=30</i>
Class I	14 (7.7)	13 (7.8)	16 (7.4)	15 (6.8)
Class II	17 (11.8)	16 (13.0)	17 (10.0)	16 (10.3)
Total	31 (16.7)	29 (17.6)	34 (15.4)	31 (14.9)

Number of eplet mismatches presented as mean (SD).

Abstract No. 48**PROPHYLACTIC PLASMA EXCHANGE IS ASSOCIATED WITH A HIGH INCIDENCE OF AMR IN SENSITISED RECIPIENTS****CHAMBERLAIN AJ¹, SNIDER J², POWER DA², WHITLAM JB²**¹*Austin Hospital, Melbourne,* ²*Department of Nephrology, Austin Hospital, Melbourne*

Aims: Thresholds for peri-operative plasma exchange (PPEX) in recipients with donor specific antibody (DSA) and negative crossmatch are not clear. We sought to review indications for and outcomes following PPEX at our centre. **Methods:** All adult kidney transplant recipients who received PPEX between 2012 and 2016 were identified. Demographic, immunologic, clinical and plasma exchange treatment data were collected from the clinical record. **Results:** 63/251 (24%) recipients received PPEX. Indications were DSA (78%), ABO incompatibility (14%), DSA+ABO incompatibility (3%), and other (5%). Of 51 recipients with DSA who received PPEX, number of DSAs was 1 (57%), 2 (31%), 3 (10%) and 4 (2%). DSA target was class I (50%), class II (36%) and class I+II (14%). The median maximum recipient DSA mean fluorescence intensity (MFI) was 1659 (IQR 1090-2789). 57% of maximum DSA MFI were < 2000. The median number of PPEX treatments was 6 (IQR 4-8). 43% developed antibody mediated rejection (AMR) at median of 40 (IQR 9-269) days post-operatively. Development of AMR was not predicted by DSA number, class or MFI. Time to AMR was predicted by DSA class (class I+II = 6 days, IQR 5-9; class II 15, IQR 9-123; class I 40, IQR 19-207; p=0.03), but not DSA number or MFI. **Conclusions:** In this cohort of kidney transplant recipients who received PPEX for relatively low risk HLA sensitisation, development of AMR was common and not predicted by traditional indicators for PPEX. The optimal use of PPEX in this setting is yet to be defined.

Abstract No. 49**TISSUE-RESIDENT LYMPHOCYTES IN SOLID ORGAN TRANSPLANTATION****PROSSER Amy^{1,2}, HUANG Wen Hua³, LIU Liu¹, LARMA-CORNWALL Irma⁴, JEFFREY Gary¹, GAUDIERI Silvana², DELRIVIERE Luc^{5,3}, KALLIES Axel⁶, LUCAS Michaela^{1,7}**¹*Medical School, University of Western Australia, Perth,* ²*School of Anatomy, Physiology and Human Biology, University of Western Australia, Perth,* ³*School of Surgery, University of Western Australia, Perth,* ⁴*Centre for Microscopy, Characterisation and Analysis, University of Western Australia, Perth,* ⁵*WA Liver & Kidney Transplant Service, Sir Charles Gairdner Hospital, Perth,* ⁶*Department of Microbiology and Immunology, The Peter Doherty Institute for Infection and Immunity,* ⁷*Department of Immunology, Sir Charles Gairdner Hospital, Perth*

Introduction: Solid organ transplantation is the standard treatment option for many patients with end-stage diseases. Despite improvements in short-term outcomes, long-term organ graft survival has remained poor for the past two decades. Newly characterised tissue-resident lymphocytes are suspected to play a significant role in graft survival and rejection, although their function in transplantation has not yet been tested. Similarly, the contribution of donor- and recipient-derived lymphocytes to allograft survival and rejection has not been investigated.

Methods: We have performed orthotopic liver transplants in either congenic or MHC mismatched mice. At various timepoints up to one month post-surgery, rejection of the organ was scored histologically and donor- and recipient-derived lymphocytes were analysed in the graft and peripheral organs by flow cytometry. The maintenance and differentiation to a tissue-resident phenotype of various cellular subsets was also assessed.

Results: Tissue-resident lymphocytes were successfully transplanted with the liver, with long-term survival of these cells observed only in a congenic transplantation context. MHC mismatch of donor and recipient mice, however, led to severe rejection and rapid depletion of most donor cells. Vast numbers of recipient lymphocytes also quickly infiltrate the allograft and upregulate markers associated with tissue-residency.

Conclusions: Donor-derived tissue-resident lymphocytes in the murine liver are readily transferrable with whole liver transplantation. Depletion of these cells in MHC mismatched transplants and infiltration of recipient lymphocytes differentiating to a tissue-resident phenotype coincided with severe rejection of the allograft. This suggests tissue-residency of lymphocytes, whether donor- or recipient-derived, is important in the context of solid organ rejection.

Abstract No. 50**CYTOMEGALOVIRUS INFECTION FOLLOWING LUNG TRANSPLANTATION INCREASES NATURAL KILLER CELLS EXPRESSING ACTIVATING RECEPTORS****SULLIVAN Lucy^{1,2}, HARPUR Christopher¹, STANKOVIC Sanda³, BROOKS Andrew¹, WESTALL Glen²**¹*University of Melbourne*, ²*Lung Transplant Unit, Alfred Hospital, Melbourne*, ³*University of Adelaide*

Aims: The control of cytomegalovirus (CMV) is a significant hurdle to successful lung transplantation. In healthy individuals, natural killer (NK) cells play a key role in immunity to CMV. NK cell function is controlled by a suite of activating and inhibitory receptors, including the CD94-NKG2 family. These receptors consist of CD94 paired with an inhibitory (NKG2A) or activating (NKG2C) member of the NKG2 family. Following CMV infection in healthy donors, there is an expansion of NK cells expressing activating NKG2C receptors, which are typically characterised by increased killing activity, indicating an important mechanism for the control of the virus. We aimed to determine whether a similar subset of NK cells is expanded following CMV infection in lung transplant recipients and whether they can contribute to viral control.

Methods: We longitudinally assessed the changes in NK cells in the blood and bronchoalveolar lavage (BAL) of lung transplant recipients, with a particular focus on individuals who were CMV naïve at the time of transplant and received a CMV+ allograft.

Results: We observed the expansion of NKG2C+ NK cells in the blood of lung transplant recipients, coinciding with the detection of CMV reactivation in the lung allograft. Furthermore, NKG2C+ NK cells were enriched in the BAL of these recipients and expressed an activated phenotype. Additionally, in this small cohort the presence of NKG2C+ NK cells appeared to protect against a high titre CMV reactivation.

Conclusions: Monitoring the expansion of NKG2C+ NK cells following lung transplantation may be a novel biomarker for assessing recipient levels of CMV immunity.

Abstract No. 51**TREG RECONSTITUTION PREVENTS GVHD****BOLTON Holly^{1,2}, PIJNING Aster^{3,4}, ROMANO Adelina^{2,4}, FAZEKAS DE ST GROTH Barbara¹**¹*Department of Pathology, University of Sydney*, ²*Dendritic Cell Research, ANZAC Research Institute*, ³*Centenary Institute of Cancer Medicine and Cell Biology, Sydney*, ⁴*University of Sydney*

Background: Regulatory T cell (Treg) based therapies have been proposed as a therapy for graft-versus-host disease (GVHD) in recipients of allogeneic haematopoietic cell transplants (alloHCT). However, the precise mechanisms by which Tregs control GVHD are not well understood.

Aims: To investigate the effect of Tregs on the early GVH response of allogeneic T cells.

Methods: We utilised a mouse model of alloHCT in which the Treg compartment was selectively reconstituted prior to transfer of allogeneic T cells, which we have previously shown protects against GVHD up to 120 days post-transplant. AlloHCT recipients received purified Treg cells at the time of transplant, and were expanded with IL-2/anti-IL-2 complexes. Allogeneic T cells were transferred at day 7 post-transplant, and their proliferation, phenotype and cytokine expression analysed at day 4 and 7 post-transfer.

Results: Allogeneic T cells adoptively transferred into Treg-reconstituted alloHCT recipients underwent slow homeostatic division and retained their naïve phenotype. In contrast, allogeneic T cells transferred into hosts without Tregs underwent rapid proliferation and acquired an effector/memory phenotype. Inhibition of rapid proliferation of donor T cells in Treg-reconstituted hosts was also associated with a reduction in inflammatory cytokine expression in lymph nodes, and a reduction in the number of infiltrating T cells in target tissues.

Conclusion: These results demonstrate the importance of reconstitution of the Treg compartment prior to transfer of allogeneic T cells both for protection against GVHD and for promoting optimal T cell reconstitution after alloHCT.

Abstract No. 52

IDENTIFICATION OF CHANGES IN PHENOTYPE OF ALLOANTIGEN ACTIVATED CD4⁺CD25⁺TREG COMPARED TO NAÏVE TTREG.**HALL Bruce M^{1,2,3}, ROBINSON Catherine M^{1,2,3}, VERMA Nirupama D^{1,2,3}, NOMURA Masaru^{1,4}, WILCOX Paul^{1,2,3}, TRAN Giang T^{5,2,3}, CARTER Nicole^{1,2,3}, BOYD Rochelle^{1,2,3}, HODGKINSON Suzanne J^{1,2,3}**¹Department of Medicine, University of New South Wales, Sydney, ²Immune Tolerance Laboratory, Ingham Research Institute, ³Liverpool Hospital, ⁴Department of Surgery, Nakashibetu Hospital Shibetugun, ⁵Discipline of Medicine, University of New South Wales, Sydney

In vitro induction of alloantigen-specific Treg has potential to induce transplant tolerance. Naïve tTreg cultured with alloantigen and IL-2 become Ts1 expressing IFN-gamma receptor (IFNGR) and IL-12 receptor beta2 and have increased capacity to mediate alloantigen-specific suppression. This study examined changes in phenotype that may identify the activated antigen-specific Treg.

Methods: CD4⁺CD8⁻CD25⁺Treg (tTreg) from naïve DA rats were cultured with PVG stimulators and IL-2 for 3-4 days to induce Ts1 cells. We studied known markers of activated Treg including, higher CD25 and FOXP3 expression, class II MHC expression and loss of CD62L. We have also identified co-expression of CD8 on Ts1 cells.

Results: Compared to naïve tTreg, Ts1 populations had higher CD25 expression. Increased expression of Foxp3 and class II MHC were not identified. CD62L expression increased.

tTreg preparations had 0.94±0.35% (mean ± SD) CD8⁺ cells that increased to 14.11±4.75% (p=0.0004) in Ts1 cells. tTreg had 1.24±0.75% CD8⁺ and after culture 4.18±1.79% (p=0.009) suggesting most were CD8⁺ homodimers. CD8⁺ cells had higher expression of CD25 and FOXP3 than the CD8⁻. RT-PCR showed the increase in *Ifngr* and *Il12rβ2* that characterizes Ts1 cells was mainly on the CD8⁺ cells, as was *Irf4*, a transcription factor induced by TCR-activation.

Ts1 cells activated by specific alloantigen and IL-12 are induced to Th1-like Treg. Proliferation induced in Ts1 cells by IL-12 was mainly in CD4⁺CD8⁺CD25⁺Treg.

Conclusions: We have previously shown that the CD8⁺Treg are required for the enhanced antigen-specific suppression of Ts1 suggesting CD8 expression is a marker of activated Treg, and may be used to separate these cells.

Abstract No. 53

MONITORING OF HUMAN CD4⁺CD25^{hi}CD127^{lo}CD45RA⁻FOX3^{hi} TREG POPULATION AS POSSIBLE ALLOANTIGEN-SPECIFIC TREG.**VERMA Nirupama D^{1,2,3}, LAM Andrew^{1,2,3}, CHIU Chris^{1,2,3}, ROBINSON Catherine M^{1,2,3}, TRAN Ginag T^{1,2,3}, HODGKINSON Suzanne J^{1,2,3}, HALL Bruce M^{1,2,3}**¹Department of Medicine, University of New South Wales, Sydney, ²Immune Tolerance Laboratory, Ingham Research Institute, ³Liverpool Hospital

Background: CD4⁺CD25⁺FOXP3⁺Treg (tTreg) are main mediators of transplant tolerance. Monitoring this population has generally been unrewarding. Human Treg populations are heterogeneous, and comprising naïve Treg expressing CD45RA (Pop I), and CD45RA^{lo/-} activated and memory Treg. Within the CD45RA⁻ are CD25^{hi}FOXP3^{hi} cells, which are activated (Pop II) and FOXP3^{lo}CD25^{lo} cells (Pop III). Activated Treg express chemokine receptors of activated T cells, including CXCR3 (Th1) and CCR6 (Th17).

Methods: Healthy human blood (HD) was examined by FACS in normal healthy controls and patients with MS. CD4⁺CD25⁺CD127^{lo}Treg isolated from HD by FACS were cultured with human rIL-2 or rIL-4 (200Units/ml) and irradiated allogeneic stimulator cells for 4 days then examined for induction of activated Treg.

Results: Studies of blood samples from MS patients showed they had greater numbers of activated Treg (Pop II). Further, the CXCR3 population was similar, but the CCR6⁺ population was less in MS patients. Sequential changes were induced by immunomodulating therapy.

Healthy donor CD4⁺CD25⁺CD127^{lo}Treg cultured with rIL-2 and alloantigen had an increased proportion of CD25^{hi}FOXP3^{hi} Pop II cells that had higher expression of CXCR3, consistent with activation of Th1 like Treg.

Conclusions: CD4⁺CD25^{hi}CD127^{lo}FOXP3^{hi}Treg (Pop II) can be monitored in peripheral blood and detect changes associated with immune activation and the effects of immunotherapy. These activated Treg can be induced in vitro by culture of Treg with alloantigen and IL-2, suggesting that activated Treg can be induced from tTreg and can be monitored.

Abstract No. 54

ALLOACTIVATION OF HUMAN CD4⁺CD25⁺CD127^{lo}FOXP3⁺TREG TO INDUCE ACTIVATED TREG
VERMA Nirupama D^{1,2,3}, LAM Andrew^{1,2,3}, CHIU Chris^{1,2,3}, ROBINSON Catherine M^{1,2,3}, TRAN Giang T^{1,2,3}, HODGKINSON Suzanne J^{1,2,3}, HALL Bruce M^{1,2,3}¹*Department of Medicine, University of New South Wales, Sydney,* ²*Immune Tolerance Laboratory, Ingham Research Institute,* ³*Liverpool Hospital*

Background: Human T regulatory cell (Treg) population is heterogenous, and comprise of naive Treg expressing CD45RA (PopI), and CD45RA^{lo/-} activated and memory Treg. Within the CD45RA⁺ population, there are cells with high expression of CD25 and FOXP3, which are activated Treg (PopII) and cells with low expression of FOXP3 and CD25 (PopIII). We examined phenotypes of human Treg after culture with either rIL-2 or rIL-4 alone or with allogeneic stimulator cells.

Methods: CD4⁺CD127^{lo}CD25⁺Treg were either isolated from PBMC or from blood directly using kits. Enriched tTreg were cultured with human rIL-2 or rIL-4 (200Units/ml) and irradiated allogeneic stimulators. Changes in the three populations were assayed by FACS.

Results: The first method had enrichment to 90% FOXP3⁺ and the second method yielded 70% FOXP3⁺. The highly enriched population had greater activation of Treg with alloantigen and cytokine, whereas the less enriched populations had marked activation with alloantigen alone and no cytokine, suggesting the FOXP3⁺T cells may have been activated by alloantigen to produce IL-2 and/or IL-4.

FACS profile of Treg within three populations based on CD45RA and Foxp3 expression showed an increase in Foxp3⁺CD45RA⁺ PopI, with a marked increase in Foxp3^{hi}CD45RA⁺ population (Pop II, 22% vs 10%). Cells continued to express CD62L.

Conclusions: CD4⁺CD25⁺CD127^{lo}FOXP3⁺Treg can be activated by alloantigen and either rIL-2 or rIL-4 to increase the PopII, which is FOXP3^{hi} and CD25^{hi}, and represents the activated Treg. The increase in PopI may represent polyclonal expansion of naive tTreg. These tTreg activation pathways may produce potent antigen-specific Treg for therapy.

Abstract No. 55

RISK STRATIFICATION FOR REJECTION BY EPLET MISMATCH AFTER EARLY MYCOPHENOLATE DOSE REDUCTION IN KIDNEY TRANSPLANT RECIPIENTS
COUGHLAN Timothy¹, CANTWELL Linda², LEE Darren¹¹*Department of Renal Medicine, Eastern Health,* ²*Victorian Transplantation and Immunogenetics Service, Australian Red Cross Blood Service*

Aims: Eplet mismatch (EpMM) is associated with de novo donor-specific antibodies and long-term graft loss in kidney transplant recipients (KTR) especially with poor adherence and low tacrolimus levels. We investigated whether EpMM predicts rejection post-mycophenolate dose reduction within the first year.

Methods: Data on KTR receiving tacrolimus, mycophenolate and prednisolone in a single centre (February 2011 – January 2017) was retrospectively analysed, excluding those with rejection within the first month. We explored the association of conventional HLA mismatches, EpMM (HLAMatchmaker, antibody verified and unverified) and mycophenolate dosing with acute rejection in those with early mycophenolate reduction (<1.5g/d within the first year).

Results: Of the 63 eligible patients (median follow-up: 3.1 years, 12 months minimum), 44 had early mycophenolate reduction, mostly due to cytopenia (68%). There was no difference in rejection rates with or without early dose reduction (27% vs 28%). Within the dose-reduced cohort, there was no significant difference in conventional HLA mismatches (4.3±1.8 vs 3.3±1.9, p=0.12), or total (51±27 vs 55±35, p=0.73), class I (17±8 vs 15±8, p=0.35), class II (34±22 vs 30±29, p=0.51) or HLA-DQ (16±15 vs 21±18, p=0.74) EpMM loads between rejectors and non-rejectors. No difference in rejection rates was observed between those with >17 (n=22) vs ≤17 (n=22) HLA-DQ EpMM (32% vs 23%, p=0.73). There was also no significant difference in the duration of mycophenolate dosing <1.5g/d and <1.0g/d, or the nadir dose between rejectors and non-rejectors.

Conclusions: EpMM was not associated with acute rejection in KTR with early mycophenolate dose reduction within the first year.

Abstract No. 56

EFFICACY OF EVEROLIMUS FOR IMPROVING RENAL IMPAIRMENT IN LIVER TRANSPLANT PATIENTS: A SINGLE CENTRE, AUSTRALIAN EXPERIENCE**EASTMENT Jacques^{1,2}, JARRETT Maree¹, HODGKINSON Peter¹, TALLIS Caroline¹, FAWCETT Jonathon¹**¹*Department of Liver Transplantation, Princess Alexandra Hospital, Brisbane, ²School of Medicine, University of Queensland, Brisbane*

Aims: Calcineurin inhibitor (CNI) immunosuppression is a major cause of nephrotoxicity in liver transplantation. Evidence suggests that use of everolimus, in combination with a reduced dose CNI, can improve renal function in post-transplant patients. This study aimed to assess the efficacy of the introduction of everolimus + reduced dose CNI immunosuppression in improving renal function in liver transplant patients with renal impairment at a major tertiary centre in Australia.

Methods: Using a database of liver transplant recipients, all patients on everolimus + reduced CNI dosing were identified. Over a period of 18 months, primary outcomes analysed were: serum creatinine, incidence of biopsy-proven allograft rejection (BPAR), and deaths. The secondary outcomes analysed were: systolic blood pressure, total cholesterol, and wound healing.

Results: Eighteen patients were started on an everolimus + reduced dose CNI due to poor renal function. This represented 22% of all liver transplant patients over an 18-month period. Using a non-interaction mixed effects linear regression model, it was found that the effect of time taking everolimus on reducing mean serum creatinine was significant ($p < 0.01$). No patients died and no patients developed BPAR whilst on everolimus. The introduction of everolimus did not significantly affect systolic blood pressure but it did elevate total serum cholesterol levels. There was a high premature cessation rate due to everolimus-related side effects (55%).

Conclusion: Everolimus + reduced dose CNI regimes significantly improve renal function in post transplant patients with CNI-related kidney impairment. However, the drug remains poorly tolerated in approximately half the liver transplant population.

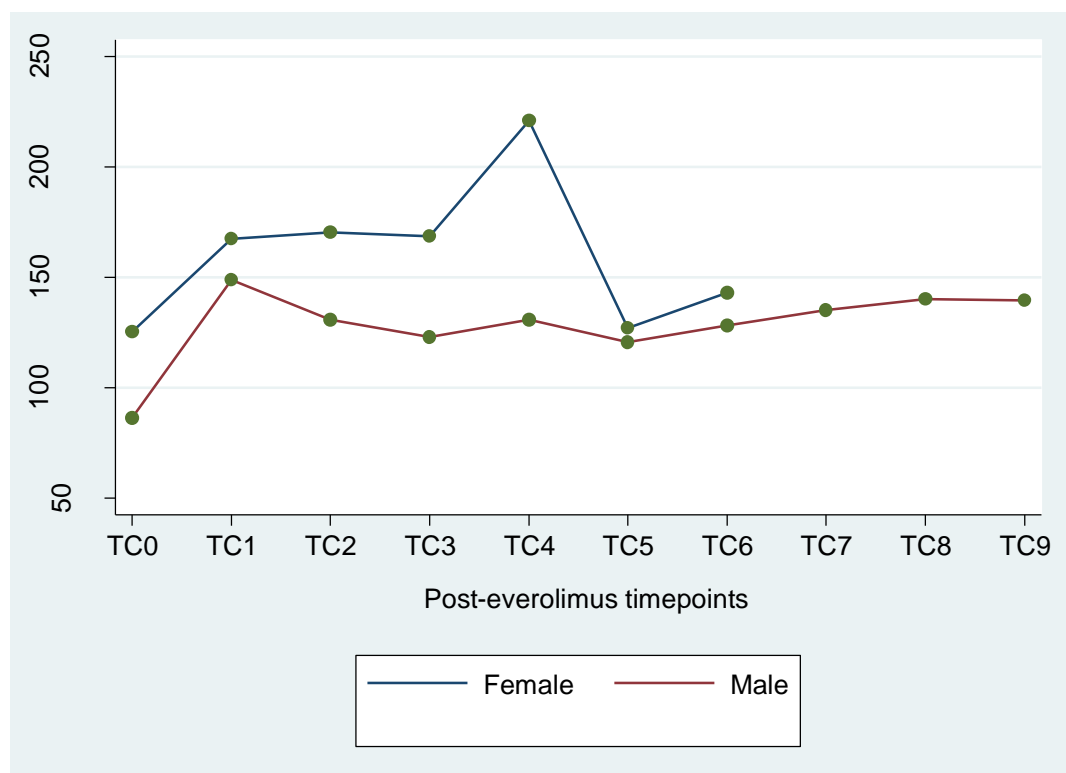


Figure 1: Mean serum creatinine in post-liver transplant patients identified to have renal impairment who were subsequently started on an immunosuppression regime of everolimus and reduced dose calcineurin inhibitor (n=18). Mean serum creatinine was recorded over an 18 month period from commencement of everolimus. It was found that time taking everolimus significantly reduced serum creatinine. While the overall effect of Time was significant ($p < 0.01$), it should be noted this is conditional on one of the time points being TC0 (i.e. day of transplantation) or TC1 (i.e. day of everolimus introduction). TC0 = day of transplant; TC1 = day of everolimus introduction; TC2 = 1 week post everolimus introduction; TC3 = 4-6 weeks post everolimus introduction; TC4 = 3 months post everolimus introduction; TC5 = 6 months post everolimus introduction; TC6 = 9 months post everolimus introduction; TC7 = 12 months post everolimus introduction; TC8 = 15 months post everolimus introduction; TC9 = 18 months post everolimus introduction.

Abstract No. 57

DONOR HEART PRESERVATION: APPLYING THE ACID TEST

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Aims: This project sought to investigate the cardioprotective effects of Hi1a, an acid-sensing ion channel 1a (ASIC1a) inhibitor derived from funnel web spider venom, in the context of donor heart preservation.

Methods: Studies were conducted utilizing an isolated working rat heart model of donor heart preservation. Hearts were retrieved from male Wistar rats (350-450g) prior to obtaining baseline measurements of key haemodynamic parameters in both ex-vivo Langendorff and working modes. Hearts were then arrested with, and stored in, either Celsior alone, Celsior + GTN and Zoniporide, or Celsior + Hi1a (n=6, each group). Following an 8-hour cold storage, hearts were reperfused ex-vivo, and post-storage cardiac function (AF, CF, HR, CO) expressed as percentage recovery of pre-storage values.

Results: Following prolonged hypothermic ischaemia, hearts preserved and stored in Celsior solution supplemented with Hi1a [10 nM] demonstrated a superior recovery to those stored in un-supplemented Celsior; recovering, on average, an aortic flow of $49.73 \pm 23.71\%$ and $7.84 \pm 12.94\%$ of baseline measurements, respectively ($p=0.01$).

Conclusions: Supplementation of Celsior with the ASIC1a inhibitor, Hi1a, significantly improves the function of cardiac allografts following prolonged hypothermic ischaemia. Further studies will investigate the mechanisms responsible for its cardioprotective effects, as well as potential synergistic performance with the current clinically used supplements of GTN and EPO, in both DBD and DCD models of donor heart preservation. By reducing the incidence of ischaemic reperfusion injury, Hi1a may afford an opportunity to extend the current tolerable warm ischaemic time during DCD cardiac allograft retrieval, leading to a significant increase in transplant volume.

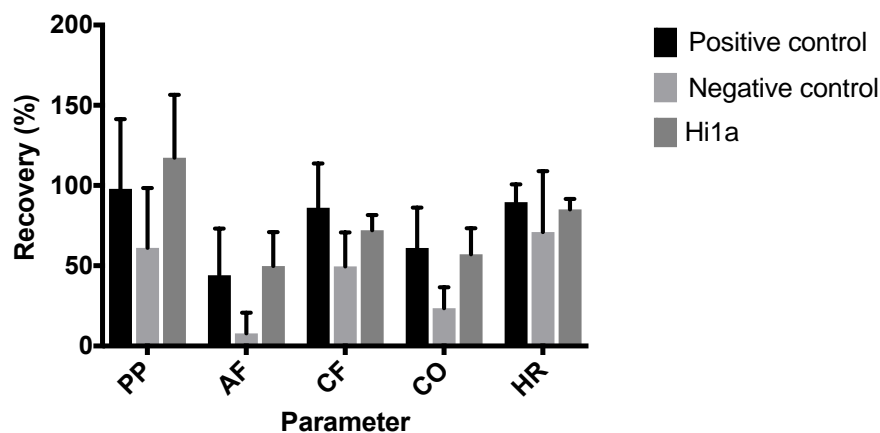
8 hr protocol - Hi1a

Figure 1: Results of Hi1a pilot study. Statistical significance was achieved between the Hi1a group and the negative control group for the parameters of pulse pressure (PP), aortic flow (AF), coronary flow (CF) and cardiac output (CO).

Abstract No. 58**OVERCOMING BARRIERS FOR INDIGENOUS AUSTRALIANS GAINING ACCESS TO THE KIDNEY TRANSPLANT WAITING LIST****ATKINSON Amy, FORD Sharon, GOCK Hilton, IERINO Frank, GOODMAN David***Department of Nephrology, St Vincent's Hospital, Melbourne*

Aims: Aboriginals constitute 10% of the Australian dialysis population but few ever receive a kidney transplant. We studied our dialysis and transplant population to identify the main barriers to Victorian patients being listed for transplantation.

Methods: All Aboriginal patients on dialysis (n=12) or with kidney transplant (n=7) were included in the study. Information was derived from the hospital records and interview by study nurse.

Results: Twelve of 304 current dialysis patients (3.9%), mean age 59 (range 39-80), 6 male & 6 females, living in Melbourne 6 & Country Victoria 6, had mean dialysis 4.8 years (range 1-11 years). Only 1 had previously been on active list. Ten of 12 have diabetes mellitus, 5 ischaemic heart disease, 4 ex-IVDU, 2 mental illness, 2 BMI>35, 1 foot ulceration, 1 osteomyelitis, 1 bacterial endocarditis, 1 recurrent pneumonia and 1 recent colon cancer. Only 1 patient, a smoker/drug user regularly missed dialysis. One patient has declined transplant work up and another had previously done so. Seven of 265 receiving a kidney transplant over the past 10 years (2.6%) waited on average 5 years from dialysis commencement to transplantation.

Conclusion: Medical co-morbidities including heart disease, infection and psycho-social issues are the main barriers to transplant listing. Concerted efforts to manage medical issues involving a multidisciplinary team of transplant physicians and nurses, GP's, Aboriginal liaison officers and social workers may allow more Aboriginals to be listed for transplantation. Once listed the current organ match system appears to provide equal access to kidneys for all Australians.

Abstract No. 59**EXPLORING THE IMPACT OF RECIPIENT AGE WITH KIDNEY DONOR RISK INDEX AND ESTIMATED GLOMERULAR FILTRATION RATE AT ONE YEAR FOLLOWING KIDNEY TRANSPLANT****CHAN Samuel^{1,2,3}, CHATFIELD Mark⁴, BABOOLAL Keshwar^{1,2}***¹Department of Nephrology, Royal Brisbane Hospital, ²Department of Medicine, University of Queensland, Brisbane, ³ANZDATA, ⁴Statistics Unit, QIMR Berghofer Medical Research Institute*

Background: Various kidney donor risk indexes (KDRI) have been developed to predict graft survival with various combinations of donor and recipient characteristics. The aim of this study was to;

1. Explore relationships between Rao KDRI and recipient estimated glomerular filtration rate (eGFR) at 1yr
2. Examine the impact of recipient age on eGFR at 1yr

Methods: A retrospective analysis of deceased donor and recipient data from Australian, New Zealand, UK and USA Organ Donor Registries was conducted from 2000-2015. KDRI and recipient age was categorised into four and six groups, respectively. Median eGFR was calculated for the 24 combinations of age and KDRI.

Results: Overall, there were 6,512 Australian, 851 New Zealand, 21,077 UK and 157,664 USA recipients (median age 52yrs, [IQR 41-61]). Recipients aged <30yrs receiving a good-quality kidney (KDRI<1.0) achieved a higher median eGFR at 1yr (87.4ml/min/1.73m²) compared with other age groups (median eGFR range: 56.0-63.3ml/min/1.73m²). Recipients aged <30yrs receiving an average-quality kidney (KDRI 1.0-1.5) yielded a better median eGFR of 67.1ml/min/1.73m² compared with other age groups (median range: 47.8-52.6 ml/min/1.73m²). Recipients aged <30yrs receiving a marginal-quality kidney (KDRI 1.5-2.0) achieved a median eGFR of 53.7ml/min/1.73m² compared with other age groups (median range: 39.2-44.4ml/min/1.73m²). Recipients aged <30yrs receiving a poor-quality kidney (KDRI>2.0) yielded a better median eGFR of 45.3ml/min/1.73m² compared with other ages (range: 35.1-36.2 ml/min/1.73m²).

Conclusions: As KDRI increases, eGFR decreases. Recipients aged <30yrs achieved a substantially higher eGFR at 1yr, independent of donor quality. Only small differences in median eGFR were seen between other age groups.

Abstract No. 60**NORMOTHERMIC MACHINE PERFUSION OF NON-UTILIZED HUMAN KIDNEYS – OUR FIRST TWO CASES**

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Aims: Normothermic machine perfusion (NMP) is an emerging modality that may improve graft function of higher KDPI kidneys and/or reduce kidney discard rates. We aimed to test this modality in a series of human kidneys deemed unsuitable for transplantation.

Methods: The first kidney was from a 74 year-old male proceeding down the DBD pathway (KDPI 96%). Both kidneys were not utilized due to suspected intra-abdominal malignancy noted during the donation procedure. The second kidney was from a 71 year-old female proceeding down the DCD pathway (KDPI 89%). The right kidney was deemed unsuitable for transplantation due to very poor/patchy perfusion. Both kidneys were transported to our centre in standard cold preservation solution for subsequent NMP. Perfusion parameters, renal function, and histology (haematoxylin & eosin [H&E] section) were compared over this time period.

Results: NMP was performed for 3 hours using the left kidney from the first donor; the second donor's kidney underwent NMP for 2 hours. Both kidneys displayed a significant improvement in perfusion parameters over time, with a drop in intra-renal resistance and increase renal blood flow. The grossly non-perfused regions in the DCD kidney were no longer evident. Both kidneys produced urine (2 ml/hr, donor 1; 22 ml/hr, donor 2). Creatinine clearance and tubular function (FeNa) improved over time, especially in donor 2. Sequential histology revealed no significant deterioration in renal tubular and glomerular cyto-architecture after 2-3 hrs of NMP.

Conclusions: NMP is a promising modality that has the potential to resuscitate grafts and thereby maximize kidney utilization.

Abstract No. 61 WITHDRAWNAbstract No. 62 WITHDRAWNAbstract No. 63**CHALLENGES TO PROCEEDING TO ORGAN DONATION IN THE NORTHERN TERRITORY**

MCAULIFFE Kathryn, WOOD Lee, JONES Sarah

DonateLife NT, DonateLife

Aims: Organ donation in the Northern Territory (NT) remains infrequent despite extensive efforts to improve community education and awareness. We aimed to examine the challenges to it occurring.

Methods: All referrals to the DonateLife NT agency between January 2014 and December 2017 were reviewed. Referral numbers increased year on year. We looked at consent rates, ethnic group, registration on the Australian Organ Donor Register (AODR) and reasons for referrals not proceeding.

Results: There were 142 referrals over the four year period. The mean age was 48.6 years (range 2 months to 82 years). Sixty-three (44.3%) of referrals were Indigenous, 54 were Caucasian (38%) and 25 (17.6%) were from a different culturally and linguistically diverse (CALD) background. Of the 142 referrals, only 55 (38.7%) proceeded to the Family Donation Conversation (FDC). Consent for organ donation was obtained from 25 (45%), 20 of whom became organ donors. There were 5 intended donors. Only 14.5% (8) of all referrals that proceeded to FDC were registered on the Australian Organ Donor Register (AODR). Of the 87 referrals that did not proceed to FDC, 45 (51.7%) were deemed either medically unsuitable or medically unsupportable. Diabetes, hypertension and hazardous alcohol use were common comorbidities amongst medically unsuitable patients.

Conclusions: Organ donation poses many challenges within the NT which require ongoing attention. Although patients are young, medical suitability issues often prevent conversations about organ donation from taking place. Registration rates on the AODR are also low.

Abstract No. 64

IMPACT OF A DEDICATED LIVING DONOR CLINIC AND ASSESSMENT TEAM: A SINGLE CENTRE EXPERIENCE**SANDIFORD Megan¹, COOK Natasha¹, WHITLAM John¹, CHAN Yee², KAUSMAN Joshua³, IERINO Frank⁴, LEE Darren^{1,5}**¹*Department of Nephrology, Austin Hospital, Melbourne,* ²*Department of Urology, Austin Hospital, Melbourne,*³*Department of Nephrology, Royal Children's Hospital, Melbourne,* ⁴*Department of Nephrology, St Vincent's Hospital, Melbourne,* ⁵*Eastern Health, Melbourne*

Aims: International guidelines recommend independent assessment of living kidney donor candidates (LKDC) by nephrologists not involved in the care or evaluation of the intended recipients. Whether this approach might have a negative impact on the determination of suitability and timely living donor (LD) transplant is unclear. We examined the efficiency of LKDC assessment before and after the establishment of a dedicated LD clinic staffed by a LD coordinator and two nephrologists.

Methods: We retrospectively compared the number of renal clinic appointments attended by LKDC to determine medical suitability and proportion of pre-emptive LD transplants pre-LD clinic (January 2006 to October 2009) and post-LD clinic (November 2009 to Oct 2017) establishment at Austin Health. LKDC with part of their assessment performed elsewhere were excluded.

Results: In the post-LD clinic, fewer clinic appointments were required to determine suitability for both accepted and declined LKDC (Table). For accepted LKDC, a further reduction in clinic appointments was observed in the second versus first half of the post-LD clinic era (median 2 (IQR 2-3) vs 4 (3-4.75); $P<0.001$), suggesting a learning curve for improvement. An increase in pre-emptive LD transplant rate also occurred (46.7% vs 14.3%). The likelihood of LKDC being accepted and LD transplant rate did not significantly change over the two eras.

Conclusions: Our experience suggests that LD clinic staffed by a dedicated assessment team improves the efficiency of LKDC assessment and facilitates pre-emptive LD transplantation. This allows the development of expertise and quality improvement without altering the acceptance threshold for medical suitability.

	Pre-LD clinic Jan 2006 - Oct 2009		Post-LD clinic* Nov 2009 – Oct 2017		<i>P</i>
	Accepted	Declined	Accepted	Declined	
Proportion of LKDC accepted	n=28 (43.8%)	n=36 (56.2%)	n=85 (57.8%)	n=62 (42.2%)	0.07
Number of renal clinic appointments to determine medical suitability	6.5 (4.25-9)	3 (2-4)	3 (2-3)	1 (1-2)	<0.001 <0.001
Proportion of pre-emptive LD transplants - Excluding paediatric transplants	14.3% (3/21) 14.3% (3/21)		46.7% (33/75) 51.0% (26/51)		<0.05 <0.01
Proportion of LD transplants - Excluding paediatric transplants	27.3% (21/77) 27.3% (21/77)		26.8% (75/280) 21.7% (51/235)		>0.99 0.35

*Assessment of LD for paediatric transplants commenced in April 2013

Abstract No. 65**CLINICIAN'S ATTITUDES AND PERSPECTIVES ON THE ACCEPTABILITY OF ANTE-MORTEM INTERVENTIONS: AN INTERNATIONAL SEMI-STRUCTURED INTERVIEW STUDY****SHAHRESTANI Sara^{1,2}, HAWTHORNE Wayne^{1,3}, PLEASS Henry³, WONG Germaine⁴, TONG Allison⁵**¹*Centre for Transplant and Renal Research, Westmead Hospital, Sydney*, ²*School of Medicine, University of Sydney*, ³*Department of Surgery, Westmead Hospital, Sydney*, ⁴*Department of Renal Medicine, Westmead Hospital, Sydney*, ⁵*Centre for Kidney Research, The Children's Hospital at Westmead, Sydney*

Background: The use of ante-mortem interventions in transplantation remains contentious due to ethical concerns and potential for harm to donors. There is variability in the acceptance and use of ante-mortem interventions across individual centers.

Methods: We conducted semi-structured interviews with 42 clinicians (transplant physicians, surgeons, ICU physicians, and donation specialist nurses), purposively sampled from eight countries including Australia, Italy, Japan, Korea, United Kingdom, United States, New Zealand, and Vietnam. We used thematic analysis to analyse the data.

Results: Four themes were identified: respecting the donor family's experience of grief; optimising 'the gift' as a duty to the donor; ambiguity in operationalising 'informed' consent, and fears of harming the donor. Participants feared burdening the grieving family with organ donation, in often-traumatic circumstances and the donation specialist role was necessary for sensitive discussion of wishes. Clinicians felt a tension between their duty to enact donor wishes, protect donors as 'patients in their own rights,' and prevent 'unsuccessful' transplantation. The complete dissemination of information for consent in a time-pressured and emotionally-charged context was described as unrealistic. Instead, the legal concept of 'authorisation,' with less onus of information, was raised. The principle of 'first do no harm' applied to the potential harms of interventions and adhering to the donor's wishes.

Conclusions: Respect for the rights and wishes of donors, minimisation of harm and optimisation of 'the gift' were paramount to clinicians. Clarity around what constitutes 'benefit' and 'harm,' along with informed discussion with families, will help clinicians resolve tensions regarding the acceptability of interventions in the donation process.

Abstract No. 66**WHERE ARE THE DONORS? A SIX-MONTH AUDIT OF DEATHS IN A LARGE SYDNEY TEACHING HOSPITAL****SPOSARI Venessa, LEIJTEN Monique***Organ and Tissue Donation Service, Liverpool Hospital*

Aim The DonateLife Audit (DLA) is undertaken at Liverpool Hospital to measure and report on actual and potential organ donation activity. This includes all patient deaths in the ICU and ED; and, patients with a neurological condition who have died within 24 hours of discharge from the ICU or ED. A review committee was established mid-2017, to review all in-hospital deaths to determine whether our hospital is missing potential organ donors beyond the DLA parameters.

Method The committee reviewed all deaths from July to December 2017. Of 655 deaths, we excluded neonatal deaths, patients who were dead on arrival to ED and ward deaths. Although these patients could be potential tissue donors, our review is targeted at potential solid organ donors.

Results The remaining 178 deaths were reviewed by a Donation Specialist Medical, 2 Donation Specialist Nurses, Intensivist, ED Physician and Transplant Physicians from the Renal and Hepatology Services. Of the 178 deaths, 95 patients were ventilated: 39 had an irreversible neurological condition and 56 were non-neurological deaths. Upon review of the non-neurological deaths, all were deemed as not medically suitable to donate due to having no suitable organs to donate (N= 15), recent/active cancer (N= 5) and 36 patients were deemed unsupportable.

Conclusion The findings of this review showed that our hospital did not miss any potential organ donors however it highlighted a significant number of unsupportable patients who presented in extremis. It can be inferred that this limits the potential donor pool and therefore further exploration of this phenomena is warranted.

Abstract No. 67

ALLOCATION OF DECEASED DONOR KIDNEYS IN CLINICAL PRACTICE: MATCHING GRAFT LIFE-YEARS AND RECIPIENT LIFE EXPECTANCY**YONG Bryan^{1,2,3}, IERINO Frank², PAIZIS Kathy¹, POWER David¹***¹Renal & Transplantation Unit, Austin Hospital, Melbourne, ²Renal & Transplantation Unit, St Vincent's Hospital, Melbourne, ³School of Medicine, University of Melbourne*

Introduction: The current allocation of deceased donor kidney organs in the Australia National Organ Matching System (NOMS) does not match expected graft life-years to patient life-expectancy. Such longevity mismatches between donor grafts and recipients may result in loss of potential graft life-years, which is known to confer improved recipient life-expectancy. Evaluating the matching of graft and recipient longevity with the current NOMS allocation algorithm is an essential step towards the formal introduction of donor-recipient matching.

Aims: To determine the correlation between deceased donor kidney graft longevity and recipient life expectancy.

Methods: Adult deceased donor kidney transplants (n = 125) from a single centre from 2011 to 2015 were examined retrospectively. Two validated clinical calculators, the Australian Kidney Donor Profile Index (KDPI) and Expected Post-Transplant Survival (EPTS) were used to predict graft and patient survival respectively. Data for the KDPI and EPTS parameters were collected from the clinical notes and transplant registry databases. The statistical relationship between KDPI and EPTS scores was then correlated using the Spearman rank correlation. **Results:** A statistical analysis between KDPI and EPTS demonstrated a poor correlation with a Spearman rank correlation of 0.179 (CI 95% 0.006-0.361, p-value = 0.046).

Conclusion: Current practices do not optimise the matching of organ expected graft life-years to recipient life expectancy, leading to loss of potential graft life-years. This supports the need to improve longevity matching of the donated kidney to the estimated life expectancy of the potential recipient.

Abstract No. 68

IN VIVO DEPLETION OF REACTIVE DONOR HUMAN CELLS REDUCES THE DEVELOPMENT OF GRAFT-VERSUS-HOST DISEASE IN A HUMANISED MOUSE MODEL**ADHIKARY Sam, GERAGHTY Nicholas, SLUYTER Ronald, WATSON Debbie***Illawarra Health and Medical Research Institute, University of Wollongong*

Graft-versus-host disease (GVHD) is a common life threatening consequence following allogeneic donor bone marrow transplantation. Reactive donor cells are the main effectors of GVHD, and depletion of these cells reduces GVHD severity in allogeneic mouse models, but there is limited data in the humanised mouse models.

Aim: This study aimed to investigate the effect of depleting reactive donor human cells on the development of GVHD in a humanised mouse model.

Methods: NOD-SCID-IL2R γ^{null} (NSG) mice were injected (i.p.) with 20×10^6 human (h) peripheral blood mononuclear cells (PBMCs) to induce GVHD, and subsequently injected with post-transplant cyclophosphamide (PTCy) (33mg/kg), or saline on days 3 and 4 post-hPBMC injection. Mice were monitored for GVHD development for 10 weeks, with human cell engraftment examined at 3 weeks post-hPBMC injection, and at end-point by flow cytometry.

Results: PTCy did not affect the engraftment of human cells in NSG mice at 3 weeks post-hPBMC injection. PTCy lowered the development of GVHD in humanised mice, significantly reducing weight loss ($P=0.0447$) and GVHD clinical score ($P=0.0478$), and increasing survival (MST=52 days) compared to saline-injected mice (MST=28 days) ($P=0.0004$). Additionally, PTCy significantly increased the proportion of hCD4⁺ T cells, and significantly lowered the proportions of hCD8⁺ T cells and hCD4⁺hCD25⁺hCD127^{lo} regulatory T cells. Finally, PTCy did not effect relative expression of pro-inflammatory cytokines, including hIFN- γ and hIL-17, in the liver, spleen or small-intestine of humanised mice.

Conclusion: Depletion of reactive human cells reduces GVHD development in this humanised mouse model, supporting its use in future studies investigating depletion strategies against GVHD.

Abstract No. 69

VALIDATION OF THE ONE LAMBDA FLOWDSA™ ASSAY FOR LIVING DONOR TRANSPLANT WORKUP

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Clinical Immunology, PathWest, Fiona Stanley Hospital, Perth

Introduction: Routine flow cytometric crossmatches (FCXM) detect donor specific HLA IgG-alloantibodies in transplant recipients. One constraint of FCXMs is false positives caused by non-HLA-mediated cytotoxicity caused by autoantibodies, non-HLA antibodies and immune complexes, and other interfering factors including treatment regimens in desensitisation protocols (e.g. rituximab), that do not reflect patient transplant outcomes. The new One Lambda FlowDSA™ assay specifically labels recipient IgG-alloantibodies bound on the donor cell surface thereby distinguishing alloantibodies from autoantibodies. In this kit, HLA molecules are separated into three groups: Class I, Class IIa (DQ), and Class IIb (DR, DP). The aim of this validation was to determine whether this assay could overcome the limitations of the current flow crossmatch assays. The validation of FlowDSA™ assay required compensation to correct for spectral overlap in fluorochromes, and the establishment of cut-offs to define the crossmatch interpretation.

Methods: FlowDSA™ compensation occurred using a bead-only control and an HLA positive control to correct PE and PerCP spectral overlap and separate the Class I, Class IIa and Class IIb populations. Positive serum with known donor specific antibodies (DSA) and negative serum was evaluated using the FlowDSA™ assay and compared results obtained with current methods.

Results: All three HLA molecule groups were distinguished as separate populations. The FlowDSA™ assay reported a positive crossmatch in the presence of strong DSA and a negative crossmatch in the absence of DSA.

Conclusions: The FlowDSA™ assay appears to be an alternative to current flow crossmatch methods. Suitable positive and negative crossmatch cut-offs, the ability to detect weak DSA and the rate of false positives due to interference from non-HLA factors including Rituximab will be important to determine its suitability for routine clinical use.

Abstract No. 70**IMMUNOSUPPRESSANT PRESCRIBING PRACTICES IN YOUNGER ADULTS COMPARED TO ELDERLY RENAL TRANSPLANT RECIPIENTS ACROSS AUSTRALIA AND NEW ZEALAND****COSSART Amelia¹, COTTRELL Neil¹, MCSTEA Megan², ISBEL Nicole³, CAMPBELL Scott³, STAATZ Christine³****¹School of Pharmacy, University of Queensland, Brisbane, ²University of Queensland, Brisbane, ³Department of Nephrology, University of Queensland at the Princess Alexandra Hospital**

Background: Kidney transplantation is first-line treatment for patients with end-stage renal failure. Optimising immunosuppressant regimens is crucial; current guidelines make no specific recommendations for elderly patients.

Aim: To evaluate the immunosuppressant medicine prescribing differences of elderly and younger adult renal transplant recipients across Australia and New Zealand.

Methods: A descriptive study of data obtained from the ANZDATA (Australia and New Zealand dialysis and transplant) registry including all patients transplanted from 2000-2015 was conducted. Patients were categorised according to age: younger adults (<70 years) and elderly (>70 years). The types and doses of immunosuppressant medicines prescribed were compared between groups using descriptive statistics (Mann-Whitney test or chi-statistic, as appropriate).

Results: A total of 6,930 patients were included in the analysis; 39% of younger adults and 41% of elderly patients were female, with an average age of 48 and 72 years respectively. The three most commonly prescribed immunosuppressant drugs were prednisolone, mycophenolate and tacrolimus; with 87% of younger adults and 89% of elderly patients taking three immunosuppressant medicines. Initial doses of mycophenolate and tacrolimus were significantly lower in elderly patients ($p<0.05$), and this trend continued at one-year, with doses of mycophenolate, tacrolimus, cyclosporin A and azathioprine significantly lower in elderly recipients ($p<0.05$; Figure 1). Elderly patients also had greater median reductions from initial to one-year post transplant in their doses of mycophenolate and azathioprine ($p<0.05$).

Conclusions: In our sample, immunosuppressant medicine doses were reduced more in elderly patients. Further investigation of drug levels and patient outcomes in the elderly is warranted.

Abstract No. 70 (cont)

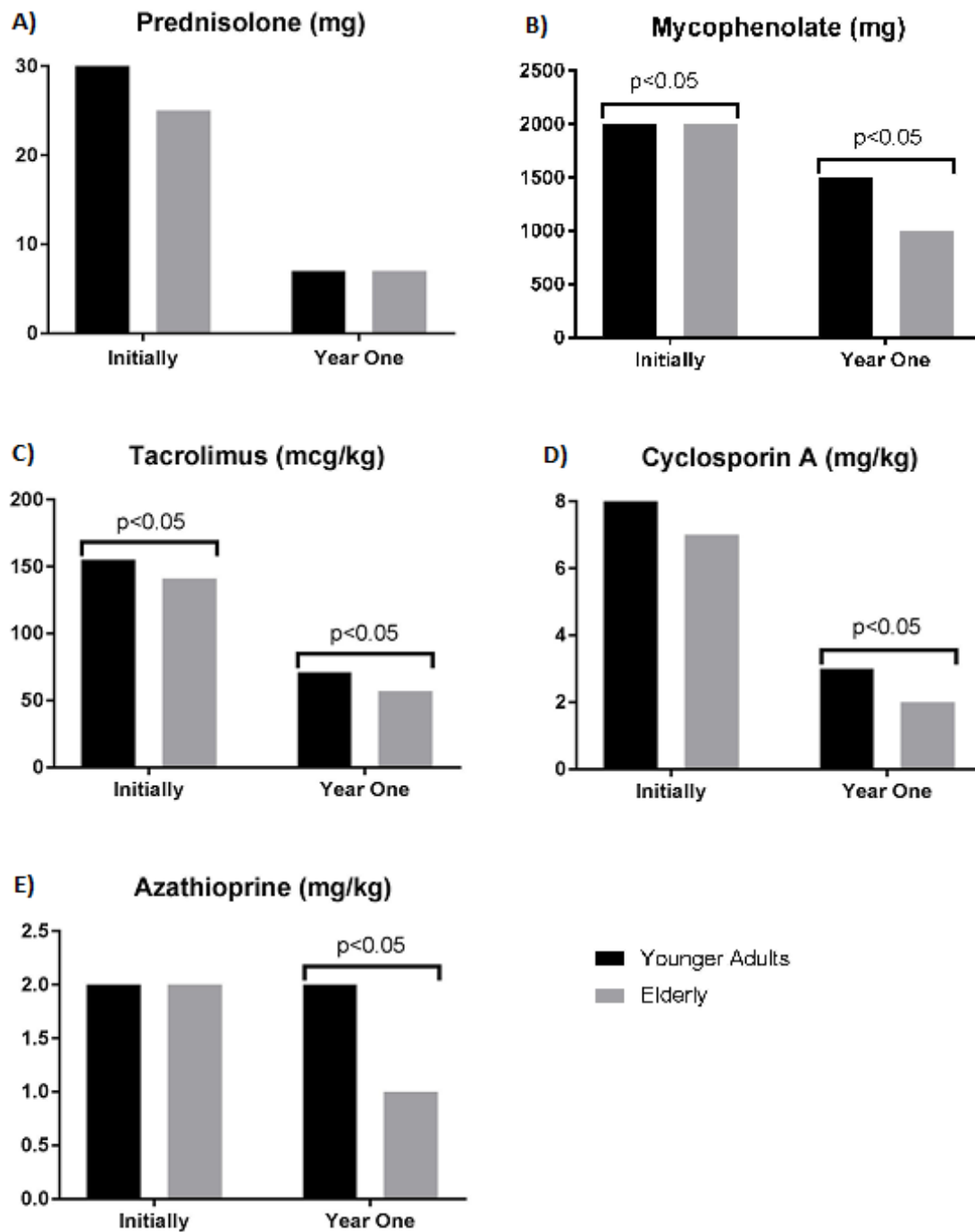


Figure 1: Median immunosuppressant medicine doses of a) prednisolone, b) mycophenolate, c) tacrolimus, d) cyclosporin A and e) azathioprine prescribed initially and at one-year post transplant in elderly and younger adult recipients

Abstract No. 71

ANZDATA INDIVIDUAL HOSPITAL REPORTING METHODOLOGY CHANGES

DAVIES Christopher^{1,2}, SYPEK Matthew^{1,3,4}, CLAYTON Philip^{1,2,3}, MCDONALD Stephen^{1,2,3}¹ANZDATA, ²Adelaide Medical School, University of Adelaide, ³Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, ⁴Faculty of Medicine, Dentistry and Health Sciences, University of Melbourne,

Aim: The Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) produces individual hospital reports annually for kidney transplantation centres. We compared alternative statistical methodologies for producing these reports to more accurately describe performance relative to the overall average.

Methods: Four alternative methods were identified to define the expected number of events (graft failures or deaths at 12 months post-transplant) at each centre and to determine centres with performance outside of plausible random variation. The methods differed in how they determined rates for each centre depending on the theoretical assignment of patients to hospitals within the model.

Results: Three of the four methods identified no units with higher than expected rates of either outcome, with one method identifying one unit with higher than expected rates of graft failure, and two units with a higher than expected rate of mortality. The chosen methodology defines the expected number of events as the number of events expected if the patients transplanted at that hospital had instead been assigned at random to any unit, weighted by hospital size. Clinical consultation supported this option; it reflects expectations for patients in the overall population, and accounts for unit size. The methodology has also been modified to control false discovery rates, accounting for the multiple comparisons made between centres.

Conclusion: Statistically, performance of almost all units is at the same level. Further developments in methods are anticipated, but generally the small size of the transplanting units precludes statistically confident observation of a clinically meaningful change in performance.

Risk-adjusted Mortality Ratio at 1 Year

Adult kidney-only transplants performed 2011-2015

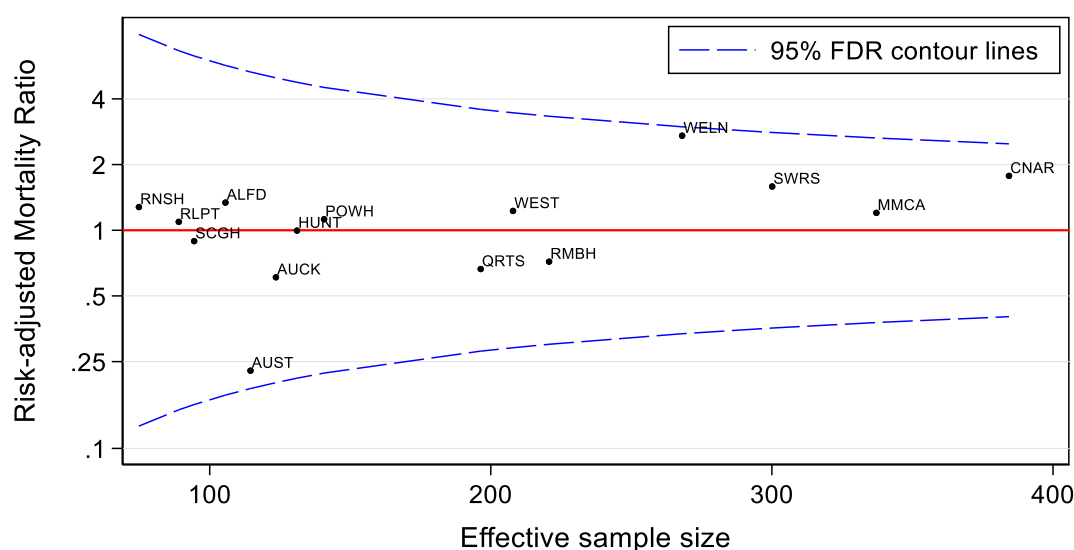


Figure 1: Funnel plot of risk adjusted mortality ratios (log scale) against the effective sample size of transplanting units in Australia and New Zealand, using the chosen methodology.

Abstract No. 72

PROTOCOL RENAL TRANSPLANT BIOPSIES IN A NON-TRANSPLANTING HOSPITAL – DO THEY CHANGE MANAGEMENT?**HEPBURN Kirsten, BROWN Mark***Department of Renal Medicine, St George Hospital*

Introduction: There is limited evidence for the utility of protocol (surveillance) biopsies in renal transplantation; however KHA-CARI guidelines suggest surveillance biopsies at 3-months for patients on Azathioprine or cyclosporine and within a year for all recipients.

Aim: To examine whether surveillance biopsies at 3 and 12-months post-transplant changed management in one non-transplanting centre.

Methods: Retrospective analysis of renal transplant recipients who underwent 3 and 12-month renal transplant surveillance biopsies from July 2010 to April 2015. Data accessed via the electronic medical record and clinic letters/documentation.

Preliminary Results: 62 (78%) transplant patients had a total of 94 biopsies. 32 patients had both 3 and 12-month biopsies, 16 had only a 3-month biopsy, and 14 had only a 12-month biopsy. The remainder had indication biopsies, no biopsy for medical reasons or unavailable records. Average age at transplantation was 52years, average creatinine and eGFR at 3-month biopsy 49ml/min/1.73m² and 126umol/L respectively and at 12-month biopsy 50ml/min/1.73m² and 131umol/L respectively. The most common primary renal disease was glomerulonephritis (40%). 69% of biopsies led to no change in management. Of the 29 biopsies (31%) that resulted in management change, 86% involved adjusting immunosuppression. Biopsy findings shown in Table 1. Analysis is ongoing.

Conclusions: The majority of surveillance biopsies did not result in changed management, though immunosuppression was changed on the basis of the biopsy in around 1 in 4 cases. This can be viewed as surveillance biopsies being of limited utility or as providing important reassurance that current management is appropriate at these times post-transplant.

Table 1: Biopsy result by change in management

Management Change	Biopsy Result
No Change	Normal (14) Minimal abnormality (15) Non-diagnostic (1) Mild IFTA (16) Possible Chronic Antibody mediated rejection (4) 'Calcineurin inhibitor toxicity' (7) Other Patchy IFTA and possible FSGS (1) Focal mild tubulitis (1) Diabetic changes (2) Hypertensive glomerular changes and IFTA (1) Focal segmental proliferative and sclerosing GN and mild IFTA (1) Mild arterial intimal fibrosis with mild small vessel hyalinosis (1) Mild interstitial fibrosis with patchy glomerulosclerosis (1)
Change to management	Minimal Abnormality (2) Mild IFTA (6) BK nephropathy (3) T cell mediated rejection (3) Acute on chronic antibody mediated rejection (1) Possible chronic antibody mediated rejection (1) 'Calcineurin inhibitor toxicity' (8) Other Moderate to severe IFTA and moderate hypertensive vascular changes (1) De-novo IgA GN and moderate hypertensive vascular changes (1) Mesangial proliferation consistent with mild glomerulitis- ? de Novo GN (1) Patchy arterial hyalinosis (1) Mild endothelitis (1)

Abstract No. 73

IMMUNE PHENOTYPE BY FLOW CYTOMETRY OF PEDIATRIC KIDNEY TRANSPLANT RECIPIENTS AND HEALTHY ADULT CONTROLS.

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Aim: To determine the differences in the immunophenotype of pediatric kidney transplant recipients and healthy adult controls.

Methods: Seven leukocyte-profiling panels containing 8-10 marker for each- were used to monitor the immune profiles of 9 paediatric kidney transplant and 8 adult control samples. Whole-blood (1.5 ml) samples were stained and acquired on BD-LSRFortessa and Flowjo was used for data analysis.

Results: Differences in subpopulations between pediatric and healthy adult control can be seen in Table 1. This showed a significant increase in absolute numbers of Granulocytes, CD14+Monocytes, double negative NKT cells and CD56+HICD16+ Intermediate NK cells. B cell panel showed a significant increase in naïve B cells, IgD+IgM+ B cells, and IgM+CD27- B cells. The naïve CD4+ and naïve CD8+ T cells, and naïve Tregs which were higher than in adult controls. Moreover, naïve Foxp3 Tregs in pediatric transplanted patients had higher CD25. Memory CD4+ T cells in pediatric patients has a similar HLA-DR expression. Further we found a significant decrease in the following cell populations in our pediatric kidney transplant patients: Non-classical monocytes, CD8+NKT, memory B cells, IgD-IgM- B cells, CD27+CD38low Class-switched memory, CD27-CD28+, CD25+ of CD4+, CD57+CD27-CD28+ and CD25+ of CD8, CCR7-/CD62L-CD45RA- of CD4 and CD8, CXCR3+CD45RO+ of CD4 and of CD8, and CD127+CD45RO+ and CD25+CD45RO+ on CD4 T cells (effector Foxp3 Tregs).

Conclusion: Immune profiling of pediatric transplant recipients demonstrated more naïve T cells, B cells and Tregs and less memory and effector memory T cells compared to healthy adult controls.

Table 1.

Pediatric cell type compare to healthy adult control	
Increase Cell Type	Decrease Cell Type
Granulocytes *	Non-classical monocytes **
CD14+Monocytes *	CD8+NKT **
Double negative NKT **	Memory B cells (IgD-CD27+) **
CD56+HICD16+ Intermediate *	IgD-IgM- B cells *
Naïve B cells *	CD27+CD38low Class-switched memory **
IgD+IgM+ B cells *	HLADR-CD45RA- of CD4 *** and of CD8 **
IgM+CD27- B cells *	CD27-CD28+ of CD4 * and of CD8 **
HLADR-CD45RA+ of CD4 ***	CD57+ **
CCR7+CD45RA on CD8 *, CD4** and Tregs*	CD4+CD25+ of CD4 and CD8 **
CD62+CD45RA+ on CD8*, CD4** and Tregs***	CD8+CD25+ of CD4 and CD8 **
CXCR3-CD45RO- on CD4 ***	CCR7-CD45RA- of CD4 and CD8 *
CD88-CD45RO- on CD4 ***	CD62L-CD45RA- of CD8 *
CXCR3+CD45RO- on CD8 ***	CD62L+CD45RA- of CD4** and of Tregs
CD25+CD45RO- on FoxP3 **	CXCR3+CD45RO+ on CD4 *** and on CD8 ***
	CXCR3-CD45RO+ on CD4 ** and CD8 ***
* <0.05	CD88-CD45RO+ of CD4*** and of CD8**
<0.01	CD127+CD45RO+ and CD25+CD45RO+ on non FoxP3 CD4 *
***<0.001	

Abstract No. 74**RISK INDICES IN DECEASED DONOR ORGAN ALLOCATION FOR TRANSPLANTATION: REVIEW FROM AN AUSTRALIAN PERSPECTIVE****LING Jonathan^{1,2}, FINK Michael³, WESTALL Glen⁴, MACDONALD Peter⁵, CLAYTON Philip⁶, OPDAM Helen⁷, HOLDSWORTH Rhonda⁸, POLKINGHORNE Kevan¹, KANELIS John¹***¹Department of Nephrology, Monash Medical Centre, Melbourne, ²School of Medicine, Faculty of Health Sciences, Monash University, Melbourne, ³General and Hepato-Pancreato-Biliary Surgery, Austin Hospital, Melbourne, ⁴Alfred Hospital, Melbourne, ⁵St Vincent's Hospital, Sydney, ⁶Department of Nephrology, Royal Adelaide Hospital, ⁷Organ and Tissue Authority, ⁸National Laboratory Manager, Australian Red Cross Blood Service, ⁹Department of Nephrology, Monash Medical Centre, Melbourne*

Recently, organ donation and transplantation rates have increased both worldwide and in Australia. Concurrently, the Australian software used for donor and recipient data management (NOMS) is being rebuilt (called OrganMatch). As an added consequence, organ allocation processes are being reviewed. Worthwhile capabilities of the new software would include the ability to use risk indices to guide organ allocation and help streamline transplantation decisions. Risk indices comprising donor, recipient and transplant factors play an important role in organ allocation policies worldwide by assimilating pertinent data to help guide transplant clinicians.

Aims To identify risk indices in use worldwide and contrast their use abroad with current Australian organ allocation policies.

Methods and Results We reviewed risk indices used in organ allocation policies worldwide for kidney, liver, heart, lung and pancreas organs and their predictive capacity for post-transplant outcomes. We collated the Australian organ allocation policies for these organs and have noted the use of similar risk indices where available. Significant donor, recipient and transplant factors used in the scores were summarised.

Conclusions Risk indices, when used together with other clinical information can assist the organ allocation process. They should not be used in isolation to make decisions regarding transplantation. Very few risk indices are currently part of the current Australian organ allocation process. Any risk index derived abroad needs to be validated in an Australian cohort before use. Modifying or adding variables in a risk index might provide an easier way to update organ allocation policies in the future.

Abstract No. 75**IN VITRO SCREENING OF GENES ASSOCIATED WITH KIDNEY FIBROSIS****MA Xiaoqian^{1,2}, SUN Lei¹, LU CAO^{1,2}, YI Shounan¹, O'CONNELL Philip¹***¹Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney, ²Institute for cell transplantation and gene therapy, The Third Xiangya Hospital of Central South University*

Aims: Chronic injury in kidney transplants remains a major cause of allograft loss. Our GoCAR multicenter study has identified a set of 13 genes were independently predictive for the development of fibrosis at 1 year after kidney transplantation. The high predictive capacity of the gene set was superior to clinical indicators. The aim of this study was to identify one or few of these genes which are associated with pathogenesis of kidney fibrosis.

Methods: The murine C1.1 tubular epithelial cell line, FOXO^{-/-} C1.1 and TCF^{-/-} C1.1 cell line were treated with or without TGF-β for 48h. Then the cells were harvest for real-time PCR to detect the expression of the 13 genes.

Results: We found there were big changes for four genes' expression. FJX1 and KLHL13 were low expressed in C1.1 and FOXO^{-/-} but upregulated when cells treated with TGF-β. Especially in FOXO^{-/-}, TGF-β induced more than 10 times expression of them which suggested FJX1 and KLHL13 may play important role in profibrotic effect. The expression of CHCHD10 was opposite with FJX1 and KLHL13 in FOXO^{-/-} and TCF^{-/-}. It was downregulated in FOXO^{-/-} while upregulated in TCF^{-/-} when treated with TGF-β. The expression of ASB15 was almost undetectable in C1.1 and FOXO^{-/-} no matter with or without TGF-β but more than 100 folds in TCF^{-/-}.

Conclusions: The results suggested the four genes may be involved with the signaling pathway of fibrosis and we will further confirm their function by CRISP/CAS9 technique.

Abstract No. 76**FACTORS ASSOCIATED WITH SUCCESSFUL RENAL TRANSPLANTATION IN INDIGENOUS RECIPIENTS FROM THE TOP END OF NORTHERN AUSTRALIA WHERE GRAFT AND PATIENT OUTCOMES ARE GENERALLY POOR****MAJONI Sandawana William^{1,2,3}, TINSLEY Nadine¹, CASILLI Alyce¹, DOLE Kerry¹***¹Department of Nephrology, Royal Darwin Hospital, ²Wellbeing and Preventable Chronic Disease Division, Menzies School of Health Research, ³Northern Territory Medical Programme, Flinders University School of Medicine*

Background Renal transplantation would provide the best treatment option for Indigenous people of Northern Australia living in remote communities. However, the number of Indigenous Australians receiving a renal transplant is low. Poor graft and patient survival rates are common. We have observed that a significant number of patients do well. This study assessed factors contributing to the difference between those with favourable and unfavourable outcomes

Methods Study design We performed a descriptive case review study of 14 Indigenous Australians who received a renal transplant between 1998- 2013. Patients were divided into favourable (graft survival ≥ 5 years) and unfavourable outcomes groups (graft survival < 5 years). We extracted data for potential influential factors; age, gender, renal diagnosis, co-morbidities, donor, cold ischemia time, delayed graft function, post-operative complications, rejection episodes and treatment, cancers, socioeconomic status, dialysis modality and vintage, human leucocyte antigen matching, induction and maintenance immunosuppression, renal biopsies, infections and outcomes.

Results There were 7 patients in each group. Those with unfavourable outcomes tended to be younger at the time of transplantation, have shorter dialysis vintage, higher numbers of human leucocyte antigen mismatches, stronger induction immunosuppression, more episodes of rejections, more episodes of infections, delayed graft function, shorter cold ischaemia time and from urban environment.

Conclusion Northern Territory Indigenous Australians can do well with renal transplants. There are potential predictors for both favourable and unfavourable outcomes which can potentially be identified before listing and at the time of transplantation. Performing prospective studies with appropriate sample sizes can identify these factors.

Abstract No. 77 **WITHDRAWN**

Abstract No. 78

POST-TRANSPLANT ACUTE KIDNEY INJURY AFFECTS LONG-TERM GRAFT FUNCTION

PRAKASH MP¹, ZHUO Tally¹, HEDLEY James², WEBSTER Angela¹, ROGERS Natasha^{1,3}

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Background: Acute kidney injury (AKI) is a common clinical condition affecting the hospitalized population, and is associated with significant morbidity and mortality. Kidney transplant recipients are predisposed to AKI due to surgical complications, medication toxicity and susceptibility to infection. However, existing literature on the frequency of AKI and its impact on graft survival is limited.

Aims: The aims of this study were to look at the incidence, aetiology and outcome of AKI in a kidney transplant population.

Methods: A retrospective cohort analysis was undertaken on recipients of a kidney or simultaneous pancreas-kidney (SPK) transplant. Patients were transplanted at Westmead Hospital from 2000-2017, and had graft survival >6 months. Baseline demographic data and information regarding post-transplant AKI were collected using recipient medical records. Episodes of AKI were defined as >25% elevation in creatinine not caused by rejection or BK virus. Primary outcome was graft dysfunction by measured glomerular filtration rate (GFR).

Results: We identified 1411 transplant recipients matching the initial inclusion criteria, of which 392 had follow-up. Two hundred and six recipients had at least one episode of AKI (mean 2.1 ± 1.5), with the first episode occurring after a mean of 28 ± 50 months. Compared to those with no AKI, AKI in the first year was associated with a 9.7ml/min decrease in GFR within 1 year (95% CI 5.1 - 14.3ml/min, $p < 0.001$). Furthermore, 11 patients (5%) demonstrated no deleterious effect of AKI on GFR.

Conclusion: AKI following kidney transplantation is common, frequent and is associated deleterious changes in GFR.

Abstract No. 78 (ctd)

	AKI (N=206)	No AKI (N=186)	p-value
Age at transplant, mean (SD)	45.50 (13.65)	46.20 (13.81)	0.6
Female, n (%)	86 (42)	73 (39)	0.6
Donor type, n (%)			0.3
DCD	19 (9)	18 (10)	
DBD	114 (55)	94 (51)	
Living	70 (34)	70 (38)	
Unknown	3 (1)	4 (2)	
Ethnicity, n (%)			0.2
Caucasian	93 (45)	70 (38)	
Non-Caucasian	67 (33)	60 (32)	
Unknown	46 (22)	56 (30)	
Transplant type, n (% of total transplant recipients)			0.001
Kidney alone	184 (50)	182 (50)	
SPK	22 (85)	4 (15)	
Months to AKI, mean (SD)	27.52 (49.97)	-	-
Average AKI episodes (SD)	2.09 (1.45)	-	-
Cause of AKI, total n (%)	Total frequency (N=507)		
Infection	136 (27)		
Sepsis	57 (11)		
Gastrointestinal illness	38 (7)		
Dehydration	9 (2)		
Obstruction	25 (5)		
Renal artery stenosis	2 (<1)		
Transplant site complication	5 (1)		
Post surgical procedure	14 (3)		
Drug induced	20 (4)		
Other (e.g. pancreatitis, hypercalcaemia, hyperglycaemia, etc.)	47 (9)		
Unknown	154 (30)		
Change in GFR after AKI (mL/min), mean (SD)	-9.55 (17.16)	-	-

Abstract No. 79

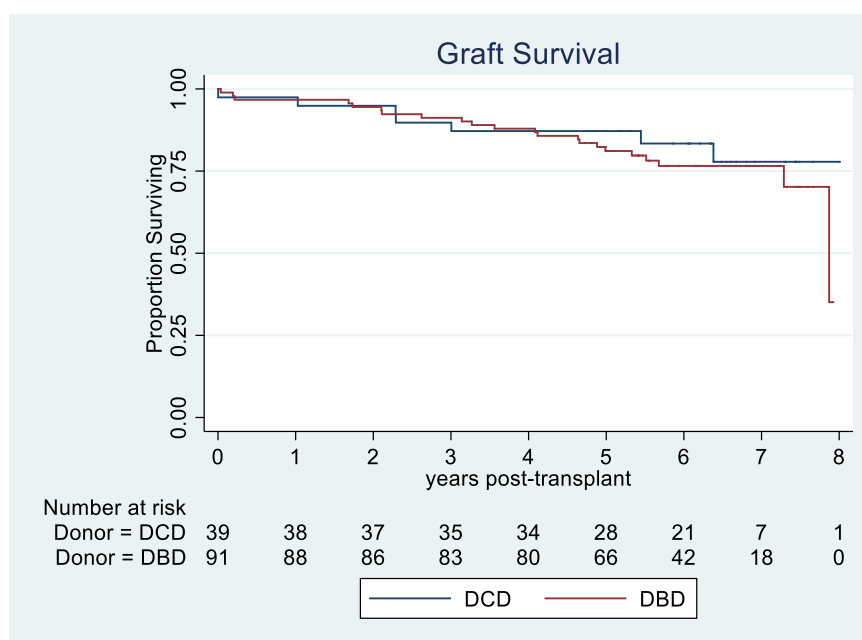
LONG-TERM GRAFT SURVIVAL AND FUNCTION IN RECIPIENTS OF DCD COMPARED TO DBD RENAL ALLOGRAFTS: A SINGLE CENTRE REVIEW.**SALTER Sherry¹, TAN Sarah¹, MULLEY William^{2,3}, CHAMBERLAIN Stacey¹, POLKINGHORNE Kevan^{2,3}, SAUNDER Alan¹, KANELIS John^{2,3}**¹*Department of Surgery, Monash Medical Centre, Melbourne,* ²*Department of Nephrology, Monash Medical Centre, Melbourne,* ³*Centre for Inflammatory Diseases, Department of Medicine, Monash University, Melbourne*

Aim: We previously described our short-term outcomes for DCD compared with DBD renal allograft recipients and now sought to extend those comparisons for long-term patient survival, graft survival and graft function between these groups.

Methods: Retrospective cohort study. All patients receiving a renal transplant from a deceased donor at our centre between 1 January 2010 to 30 April 2013 were included. Multi-organ transplant recipients were excluded. Baseline patient characteristics were compared. Graft and patient survival and mean eGFRs were compared between DCD and DBD recipients using the log-rank test and the student t-test respectively.

Results: The group comprised 91 DBD and 39 DCD recipients. The median follow-up was 6.2 years (range 4.7 to 8.0 years). There were no differences in donor or recipient age between groups however there was more delayed graft function in the DCD group. DCD kidney recipients had a longer length of admission (9.5 ± 8.3 days vs 11.6 ± 5.1 days (Rank Sum $P < 0.01$). Mean eGFR was significantly lower in the DCD group until 2 months post-transplant where after there were no differences to 8 years. There were 16 recipient deaths and 5 graft losses during the study period. Patient and graft survival (Figure) were not different between groups.

Conclusion: Equivalent outcomes in the longer term can be achieved with DCD and DBD renal allografts with similar donor characteristics. Early differences in the rate of DGF and renal function did not result in differences in graft survival or renal function after the first 2 post-transplant months.



Abstract No. 80**COMPARISON OF 3 LYMPHOCYTE SEPARATION METHODS FOR FLOW CROSSMATCH ASSAY****TASSONE Gabriella, BAZLEY Scott, D'ORSOGNA Lloyd, MARTINEZ Patricia, DE SANTIS Dianne***Clinical Immunology Fiona Stanley Hospital, Pathwest*

Introduction: The Stem Cell EasySep Direct Total Human Lymphocyte Isolation kit™ (DTHLI) uses immunomagnetic beads technology to bind non-lymphocytes within the sample. The beads are then removed by a magnet, while the supernatant contains the lymphocytes. The Stem Cell SepMate gradient centrifugation tubes™ (SepMate) use a plastic insert to keep the Ficoll at the base of the tube for spinning and pouring off the lymphocyte layer.

Method: The SepMate, DTHLI, and the current routine Ficoll gradient isolation methods were compared. The isolation time, cell yield and suitability for the routine flow crossmatch assay (FCXM) were assessed.

Results: The SepMate and DTHLI methods were more rapid, 30 and 45 minutes respectively, compared to 3 hours for the current method. The cell yield obtained by the SepMate and the current method were sufficient to perform the FCXM on untreated and pronase treated serum, while the DTHLI provided sufficient cells to perform the FCXM only on the pronase treated serum. Despite the lower cell yield the purity of CD3 and CD19 was superior in the DTHLI isolation method compared to the other methods. The lymphocyte preparations were then evaluated using the FCXM using a serum known to have donor specific antibody (DSA) to donor mismatches and a negative serum.

Conclusion: The results indicated that both SepMate and DTHLI were more rapid than the current method. The cell yield of SepMate was comparable to the current method however the cell yield obtained from the DTHLI was lower than both the current method and SepMate. However, the DTHLI isolated a greater proportion of CD3 and CD19 positive cells and therefore the total number of lymphocytes required to perform FCXM may be less than currently required. All three methods produced comparable flow crossmatch results.

Abstract No. 81**EXTENDED CRITERIA DONATION UNDER EXTENDED CRITERIA CIRCUMSTANCES****THOMPSON Sophie¹, PILCHER David², IHLE Joshua³***¹DonateLife Victoria, Alfred Hospital, Melbourne, ²DonateLife Victoria, Alfred Hospital, Melbourne, ³Intensive Care Unit, Alfred Hospital, Melbourne*

Introduction In Donation after Circulatory Death (DCD), withdrawal of cardiorespiratory support (WCRS) usually begins with extubation. Death is most commonly determined after a period of 5 minutes' observation of a non-pulsatile invasive arterial pressure trace. We present a case of a patient who wished to be a donor but had neither invasive arterial monitoring, nor mechanical ventilation.

Case presentation A 61-year-old woman was admitted to the Intensive Care Unit for management of respiratory failure following a lung transplant. After a prolonged hospital stay, the patient's care transitioned to end of life treatment. Consent was obtained from the senior available next of kin for organ donation and a rapid retrieval DCD protocol was activated. The patient was receiving oxygen via High Flow Nasal Prongs (HFNP) and was monitored only via a pulse oximeter. 23 minutes after removal of HFNP, oxygen saturation was no longer recordable. 31 minutes later the treating Intensivist examined the patient to confirm death. Time from removal of HFNP to cold perfusion of the organs was 54 minutes. Both kidneys were subsequently transplanted into recipients who are recovering well.

Discussion This was a unique situation where an extended criteria donor was able to donate organs due to a combination of being registered, family being aware and supportive of her wishes in a situation where donation would not usually be considered. Dependence on HFNP enabled death to occur in a timely fashion after WCRS, resulting in kidney donation via a rapid retrieval DCD pathway.

Abstract No. 82**EPIDEMIOLOGY AND ESTIMATED COST OF COMPLICATED SIMULTANEOUS PANCREAS KIDNEY TRANSPLANTATION****XU Joshua¹, HITOS Kerry^{2,1}, HORT Amy², SHAHRESTANI Sara², ROBERTSON Paul^{2,1}, YUEN Lawrence^{2,1}, RYAN Brendan^{2,1}, DE ROO Ronald^{2,1}, HAWTHORNE Wayne^{3,4}, PLEASS Henry^{2,1}**¹*Westmead Clinical School, University of Sydney*, ²*Department of Surgery, Westmead Hospital, Sydney*, ³*Discipline of Surgery, Sydney Medical School, University of Sydney*, ⁴*Centre for Transplant and Renal Research, Westmead Institute of Medical Research***Background:** Simultaneous pancreas-kidney transplantation (SPK) is a well-established treatment for type 1 diabetes mellitus and end-stage renal disease. Limited detail on cost-effectiveness exists for transplantation and treatment complexity specifically relating to surgery and complications.**Aims:** To examine the cost associated with SPK transplantation for in-hospital admissions based on co-morbidities, complications and procedure complexity.**Methods:** 234 SPK transplantations were reviewed at Westmead Hospital (2008-2017). Donor and recipient demographic details, co-morbidities, operative characteristics, hospital and ICU length of stay (LOS), enteric leaks and graft thrombosis were collected. Estimated DRG price weights and national weight activity units (NWAU) were used to calculate in-patient costs (Australian dollars (AUD)).**Results:** Median donor and recipient age was 26 years (IQR:19-34) and 39 years (IQR:34-44) respectively. Median donor BMI was 24.2 kg/m² (IQR:21.9-25.6) and 24.2 kg/m² (IQR:21.7-27.7) for recipients. Median in-hospital LOS was 9 days (IQR:8-12). Overall, 17% of recipients experienced graft thrombosis and 5.2% enteric leaks. Complications such as sepsis, haemodialysis, enteric leaks and ICU admission increased the in-hospital and surgery costs per patient by more than \$49,237 AUD. Greater complexity such as an increase in ICU LOS and factors like volume depletion, infection, thrombosis, hypertension, hyperkalemia, osteoporosis, re-operation and asthma increased the additional in-hospital per patient cost by more \$153,841 AUD compared to uncomplicated cases.**Conclusions:** Treatment complexity, co-morbidities, ICU LOS, enteric leaks, graft thrombosis and re-operation influences in-hospital only costs greatly. This economic impact is further amplified when wages, patient assessments, organ retrieval, pharmaceutical needs, monitoring and long term follow-up costs are added.Abstract No. 83**TUMOUR RESECTED KIDNEY GRAFTS FOR TRANSPLANTATION IN WESTERN AUSTRALIA: OUTCOMES OF THE TRK PROGRAM, 10 YEARS ON.****APIKOTOA Sharie¹, HE Bulang^{1,2}**¹*WA Liver & Kidney Transplant Service, Sir Charles Gairdner Hospital, Perth*, ²*School of Medicine, University of Western Australia, Perth***Introduction:** Kidney transplantation is the definitive treatment for end stage renal failure, and chronic organ shortage is a global issue. The Tumour Resected Kidney (TRK) program was implemented in Western Australia, 2007. The aim of this study is to review the outcomes of the TRK transplantation in patients enrolled in the program by the Western Australian Kidney Transplant Service (WAKTS).**Materials and Methods:** Data was prospectively collected using a registry of all selected patients receiving TRK transplantation between Feb 2007 and February 2017. Twenty-seven patients received a TRK transplant. Follow up was from 2-10 years with a median of 7 years. Data was analysed regarding patient and graft survival, surgical complications, kidney graft function and tumour recurrence.**Results:** There were 27 TRK transplanted in patients with an age range between 32-76 years (average 63 years). The tumour size ranged from 1-4cm (mean 2.7cm) with histopathology confirming renal cell carcinoma (RCC) in 20 kidneys, 1 chromophobe tumour, 3 papillary RCC and 4 benign tumours. Complications included urine leakage in 3 patients, requiring prolonged drainage, 1 non-functional graft, 1 graft loss and 1 pseudoaneurysm formation. The Graft function was satisfactory with the average creatinine at 135 µmol/L. There has been no tumour recurrence during follow-up.**Conclusion:** The outcome of transplants by using TRK has shown to be satisfactory. The process of cold perfusion and preservation may help prevent the tumour recurrence. It is an option in the selected recipients under strict criteria.

Abstract No. 84

TRUST TRIAL: TIMING OF REMOVAL FOR URETERIC STENTS POST-RENAL TRANSPLANTATION – EARLY VS STANDARD REMOVAL

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¹Auckland Renal Transplant Group, Auckland City Hospital, ²Department of Surgery, University of Auckland, ³School of Biological Sciences and Department of Surgery, University of Auckland, ⁴New Zealand Liver Transplant Unit, Auckland City Hospital

Background: Routine placement of a ureteric stent across the anastomosis at the time of renal transplantation (RT) reduces the rate of early urological complications. The timing of stent removal involves a balance between prevention of early urological complications and development of stent-related complications.

Aim: Perform a prospective randomised controlled non-inferiority trial to determine whether early removal (day 4) of ureteric stent is as equally effective and safe as standard removal (4-6 weeks with cystoscopy) in patients following RT.

Method: 100 RT recipients at a single centre were randomised.

Results: 100 patients were randomised to either early (n=50) or late (n=50) removal. The primary outcome of graft survival at 12-months was not different ($p = 0.49$). Urine output and serum creatinine on days 0 to 10 post RT were higher in the control group but did not reach statistical significance. There was no difference in secondary outcomes including patient survival at 12 months, urological complications (leak or obstruction), graft rejection or rates of infection.

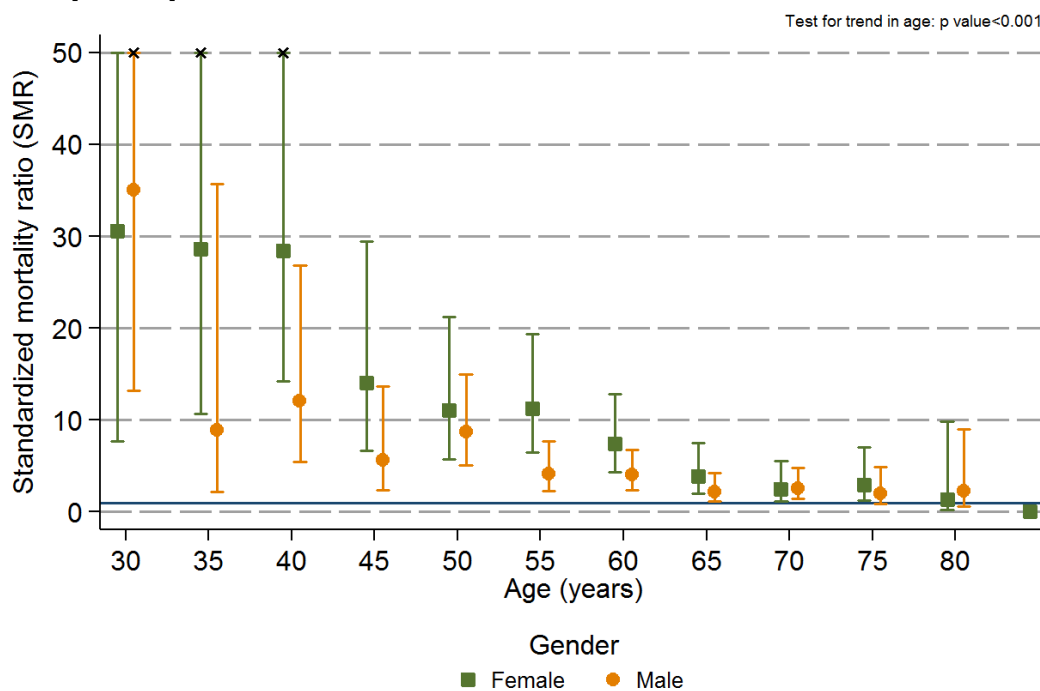
Conclusion: Non-inferiority in graft survival was demonstrated between early ureteric stent removal at the time of removal of bladder catheter and standard removal of the ureteric stent at 4-6 weeks post RT by cystoscopy.

Abstract No. 85

STROKE MORTALITY IN KIDNEY TRANSPLANT RECIPIENTS: A POPULATION-BASED COHORT STUDY USING DATA LINKAGE**DE LA MATA Nicole¹, MASSON Philip², AL-SHAHI SALMAN Rustam³, KELLY Patrick¹, WEBSTER Angela^{1,4}**¹*Sydney School of Public Health, University of Sydney*, ²*Department of Renal Medicine, Royal Free London NHS Foundation Trust*, ³*Centre for Clinical Brain Sciences, University of Edinburgh*, ⁴*Centre for Transplant and Renal Research, Westmead Hospital, Sydney*

Aims: We aimed to compare stroke deaths in kidney transplant recipients with the general population. **Methods:** We established the primary cause of death for incident kidney transplant recipients using data linkage between the Australian and New Zealand Dialysis and Transplant Registry (ANZDATA) and national death registries: Australia, 1980-2013 and New Zealand, 1988-2012. We used indirect standardisation to estimate standardised mortality ratios (SMR) with 95% confidence intervals (CI) and a competing risks regression model to identify risk factors for stroke and non-stroke mortality. **Results:** Among 17,621 kidney transplant recipients, there were 158 stroke deaths and 5,126 non-stroke deaths in 160,332 person-years of follow-up. Stroke death rates steadily increased from transplantation. All-cause stroke SMR were higher in people who were younger and particularly in females (Fig. 1). Kidney transplant recipients aged 30-49 had much greater stroke deaths than expected in the general population (Females: SMR 21.3, 95% CI: 13.9-32.7; Males SMR 9.9, 95% CI: 6.2-15.9). A higher risk of stroke death was associated with older age at transplant, earlier year of transplant and prior known cerebrovascular disease. **Conclusion:** Stroke mortality is significantly higher among kidney transplant recipients than in the general population, particularly for young people and females. Cardiovascular risk factor control and acute stroke interventions have reduced stroke mortality in the general population, but their effectiveness and the extent to which they are used in kidney recipients is less clear.

Figure 1. Standardized mortality ratios (SMR) by gender and age for all-cause stroke in kidney transplant recipients.



*95% confidence interval has been cut.

Abstract No. 86

THE ASSOCIATION BETWEEN ETHNICITY, ALLOGRAFT FAILURE AND MORTALITY AFTER KIDNEY TRANSPLANTATION IN INDIGENOUS AND NON-INDIGENOUS AUSTRALIANS: IS THIS EXPLAINED BY ACUTE REJECTION?**HOWSON Prue¹, IRISH Ashley², D'ORSOGNA Lloyd^{3,4}, SWAMINATHAN Ramyasuda², PERRY Gregory⁵, DE SANTIS Dianne³, WONG Germaine^{6,7,8}, LIM Wai H^{1,4}**¹*Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth,* ²*Department of Renal Medicine, Fiona Stanley Hospital, Perth* ³*Department of Immunology, Fiona Stanley Hospital, Perth,* ⁴*School of Medicine, University of Western Australia, Perth,* ⁵*Department of Renal Medicine, Royal Perth Hospital,* ⁶*University of Sydney,* ⁷*Centre for Transplant and Renal Research, Westmead Hospital, Sydney,* ⁸*Centre for Kidney Research, The Children's Hospital at Westmead, Sydney*

Aim: We aimed to determine whether acute rejection (AR) was a mediator between ethnicity (Indigenous/Non-Indigenous), allograft failure and mortality after kidney transplantation and whether ethnicity was a risk factor for allograft failure and mortality in those who had experienced AR.

Materials and Methods: End-stage kidney disease patients who have received a kidney-only transplant between 2000-2010 in Western Australia were included. Cox proportional modelling was used to determine the association between ethnicity, AR, allograft failure and all-cause mortality. Mediation analysis was conducted to determine whether AR was a causal intermediate between ethnicity and outcomes, and propensity-scored analysis was used to examine the association between ethnicity and outcomes in recipients who had experienced AR.

Results and Discussion: Of 618 patients who received a kidney transplant, 59(9.5%) were indigenous. During a median(IQR) patient follow-up of time of 7.9(5.7) years, indigenous recipients were more likely to experience AR (73%vs.42%, $p<0.001$), allograft failure (66%vs.37%, $p<0.001$) or death (44%vs.25%, $p=0.002$) compared to non-indigenous recipients, with adjusted hazard ratios (HR) of 1.86(95%CI 1.28-2.70, $p<0.001$), 2.17(1.97-4.00, $p<0.001$) and 2.35(1.49-3.71, $p<0.001$), respectively. Approximately 29% and 2% of the effects between ethnicity and allograft failure and death, respectively were explained by AR. In the propensity-scored analysis in recipients who had experienced AR (1:1 ratio of indigenous/non-indigenous recipients matched for recipient age, donor type, HLA-mismatches and diabetes), indigenous recipients remained at a higher risk of allograft failure and death with respective adjusted HR of 1.88(1.09-3.25, $p=0.023$) and 2.18(1.03-4.60, $p=0.041$).

Conclusions: Acute rejection explained almost 30% of the association between ethnicity and allograft failure. Following rejection, the risk of mortality was 2-fold greater in indigenous compared to non-indigenous recipients. A greater understanding of factors contributing to these adverse outcomes is urgently required.

Abstract No. 87

LONG-TERM OUTCOME OF KIDNEY TRANSPLANTATION IN PATIENTS WITH CONGENITAL ANOMALIES OF THE KIDNEY & URINARY TRACT**MCKAY Ashlene¹, KIM Siah^{1,2}, KENNEDY Sean^{1,2}**¹*Department of Nephrology, Sydney Children's Hospital, ²School of Women's & Children's Health, University of New South Wales, Sydney*

Aim: Congenital anomalies of the kidney and urinary tract (CAKUT) are a leading cause of end stage kidney failure in the young. However, there is limited information on long term outcomes after kidney transplantation in this group. We explored the outcomes of kidney transplant in patients with the 3 most common severe forms of CAKUT; posterior urethral valves (PUV), reflux nephropathy and renal hypoplasia/dysplasia.

Methods: Data were extracted from ANZDATA on all first kidney transplants performed between 1976 and 2015 in recipients with a primary diagnosis of PUV, reflux nephropathy or renal dysplasia, who were younger than 30 years when they received their transplant. Using multivariate Cox regression, we compared death censored graft survival between the three groups.

Results: 142 patients with PUV, 272 with renal dysplasia and 938 with reflux nephropathy were included. 10-year graft survival in PUV, renal dysplasia and reflux nephropathy was 67%, 72% and 64% respectively and 20-year graft survival was 32%, 51% and 43%.

After adjusting for age at transplant, era of transplantation, graft source and HLA matching, there was no significant difference in graft survival, although there was a trend to poorer outcome in PUV (HR 1.31, 95% CI 0.93 to 1.84).

Conclusions: Graft survival of first transplant in CAKUT is favourable at 10 years. We report a trend towards poorer graft survival for patients with PUV. Larger studies are required to determine whether the risk of graft failure is increased in patients with PUV.

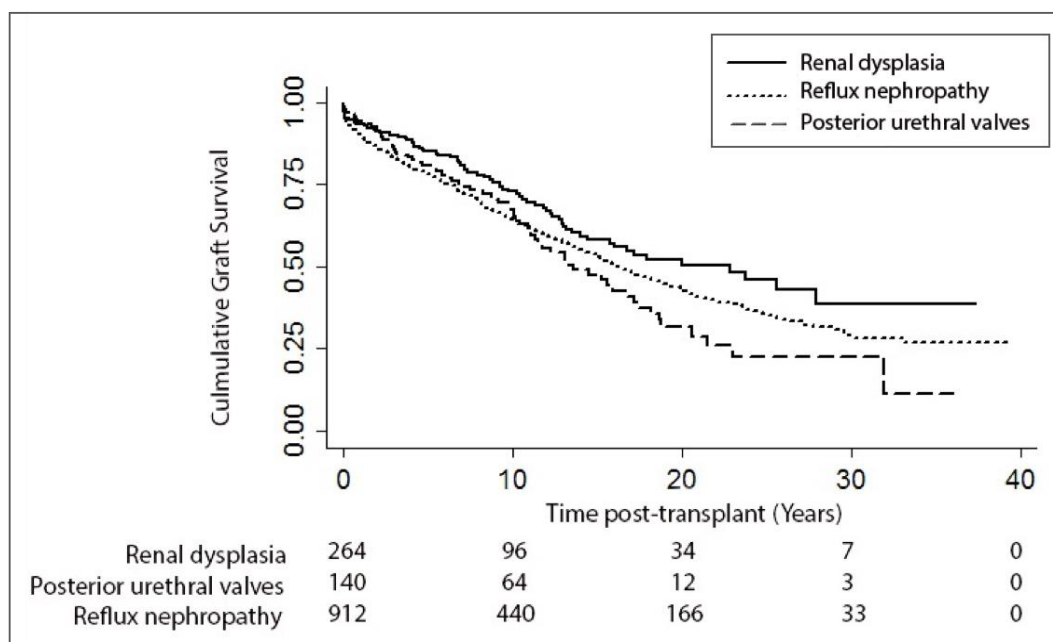


Figure 1. Poorer graft survival in patients with posterior urethral valves and reflux nephropathy. Graft survival stratified by primary renal disease.

Abstract No. 88**COMPARISON OF KIDNEY ALLOGRAFT SURVIVAL IN THE EUROTRANSPLANT REGION AFTER CHANGING THE ALLOCATION CRITERIA IN 2010 – A SINGLE CENTER EXPERIENCE****MEHDORN Anne-Sophie¹, BECKER Felix¹, REUTER Stefan², SUWELACK Barbara³, SENNINGER Norbert¹, VOGEL Thomas¹, PALMES Daniel³, BAHDE Ralf³****¹General, Visceral and Transplant Surgery, Universityhospital Muenster, Germany, ²Department of Nephrology, Universityhospital Muenster, Germany, ³Universityhospital Muenster, Germany, Universityhospital Muenster, Germany**

In 2010 Eurotransplant introduced the European Senior Program (ESP) aiming to avoid waiting list competition between young and elderly patients suffering from end stage renal disease and thus shorten waiting times for both groups. ESP-donors have to be older than 65 years and grafts are preferably allocated regional in order to shorten cold ischemia time not primarily taking HLA matching into account. This study aims to compare a historic cohort with a collectiv receiving grafts according to new guidelines.

We stratified 159 eligible patients > 65 years (ESP (n=69), former allocation criteria (n=89)) from the transplant center of Muenster, Germany and analyzed patient and graft survival as well as surrogate markers of short- and long term graft function (acute rejection, primary function (PF), delayed graft function (DGF), glomerula filtration rate (GFR).

While donors were comparable in both groups, recipients in the ESP-group were significantly older ($69.51 \text{ y} \pm 3.42$ vs. $67.06 \text{ y} \pm 2.59$, $p < 0.05$), had significantly shorter time of dialysis ($13.64 \text{ m} \pm 20.06$ vs. $60.17 \text{ m} \pm 28.06$, $p < 0.05$) and suffered from more comorbidities. Cold and warm ischemia time were significantly reduced in the ESP-group and the latter had more grafts with PF. Longterm graft function was similar. Yet, graft survival was significantly better in the ESP-group. Overall patient survival was comparable after five years.

Patients receiving grafts from older donors according to the new ESP-criteria did not have disadvantages compared to patients receiving grafts according to the former allocation criteria.

Abstract No. 89

RENAL TRANSPLANT PATIENT AND GRAFT SURVIVAL UNAFFECTED BY POST-TRANSPLANT DIABETES IN THE ERA OF LOW MAINTENANCE IMMUNOSUPPRESSION**PIMENTEL AL^{1,2}, MASTERSON R², YATES C^{3,4}, HUGHES P², COHNEY S^{5,6,7}**¹*Graduate Program in Endocrinology, Universidade Federal do Rio Grande do Sul (UFRGS),* ²*Department of Nephrology, Melbourne Health,* ³*Department of Diabetes and Endocrinology, Melbourne Health,* ⁴*Department of Endocrinology, Western Health,* ⁵*Department of Nephrology, Western Health,* ⁶*Department of Medicine, University of Melbourne,* ⁷*Department of Epidemiology, Monash University, Melbourne*

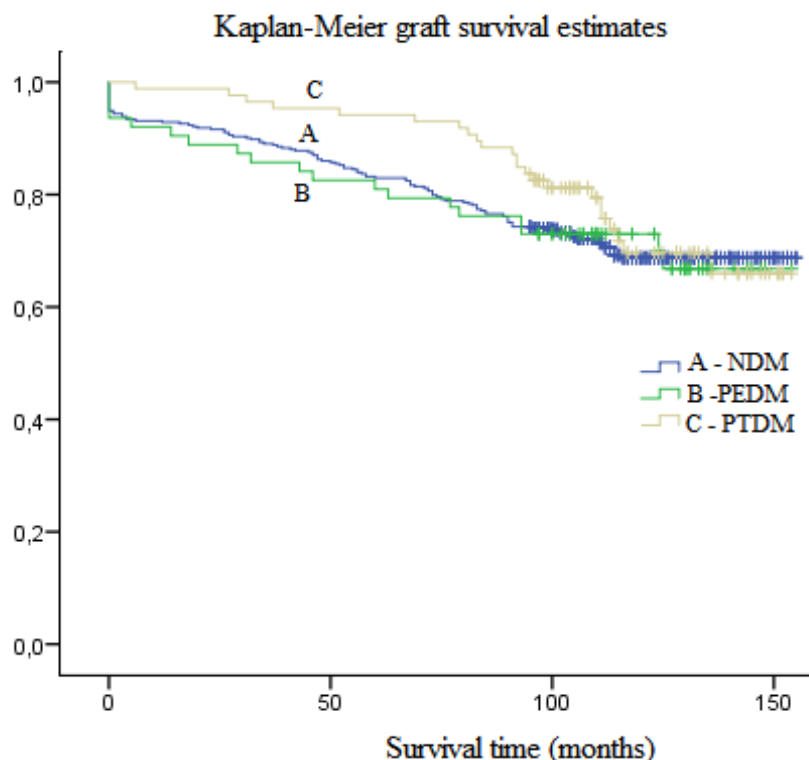
Aims: Preexisting diabetes (PEDM) and newly detected diabetes after transplant (PTDM) have been associated with reduced patient and graft survival. However, outcome data since adoption of lower maintenance immunosuppression is sparse. This study examined outcomes in patients undergoing renal transplantation between December 2004 and 2009 according to diabetes status, with patients receiving prednisolone ≤ 5 mg, MMF ≤ 500 mg b.d, and tacrolimus ≤ 4 ng/ml beyond 12 months.

Methods: All patients transplanted between December 2004-2009 were analyzed using prospectively collected data from an electronic database, patient records and ANZDATA. Diabetes status was determined using HbA1c, blood glucose levels and/or use of glucose lowering therapy.

Results: 534 patients were assessed, 7 receiving more than 1 KT. Mean age 45.2 ± 14.1 years, 64.6% male, 63 PEDM, 86 PTDM (64 diagnosed within 12 months, 22 subsequently). After mean follow-up of 9.2 ± 2.2 years patient survival was 89.9%, 81% and 90.6%, respectively, in NDM, PEDM & PTDM diagnosed within the first year. When considering PTDM diagnosed anytime, patient survival was 87.2%. Mean Tac level at 1 year was 3.7 ± 2.3 ng/mL and <4 ng/mL at 4 years. Graft survival was 70.5%, 69.8% and 73.3%, respectively, in non-DM, PEDM & those with PTDM diagnosed within 12 months, and 76.6 % when considering patients diagnosed with PTDM at any time (Figure 1).

Conclusions: In this large single centre analysis of renal transplant recipients receiving more contemporary immunosuppression, PTDM had no impact on patient or graft survival, though there was a statistically significant reduction on patient survival in patients with PEDM.

Figure 1: Graft Survival in Renal Transplant Recipients According to Diabetes Status



Abstract No. 90**REVIEW OF THE NEW ZEALAND (NZ) EXPERIENCE WITH DONATION AFTER CIRCULATORY DEATH (DCD) KIDNEY TRANSPLANTATIONS 2008-2016****SUN Tina¹, DITTMER Ian², MATHESON Philip³**¹*Department of Renal Medicine, Middlemore Hospital,* ²*Auckland Renal Transplant Group, Auckland City Hospital,* ³*Department of Renal Medicine, Wellington Hospital***Aim** Review of the NZ national experience and outcomes with DCD kidney transplantations since its introduction in 2008.**Background** Deceased donor kidney donation in NZ has been exclusively from donation after brain death donors for many years. This changed in 2008 with the introduction of DCD transplantations to increase the availability of deceased donors.**Method** A retrospective review of DCD kidney transplantations performed in NZ between January 2008 and December 2016, with follow-up until March 2017. Patients were identified from ANZDATA registry and Organ Donation New Zealand database. Data collected were: age, gender, ethnicity, mortality, immediate and long-term graft function, cold ischaemic time, graft number and co-morbidities.**Results** A total of 42 DCD transplantations were conducted in NZ during the study period from 22 donors. The majority of the recipients were male (71%) with a mean age of 50.1 (+/-14.4). 57% of the recipients developed delayed graft function (DGF) requiring renal replacement therapy for a mean duration of 7.25 (+/- 5.7) days after transplantation. There was no primary graft non-function. All-cause graft survival was 90% at 1 year, 86% at 2 years, and 86% at 5 years. Death-censored graft survival was 100% at 1 year, 95% at 2 years, and 95% at 5 years. Mean creatinine were 181umol/L, 140umol/L, 139umol/L, 132umol/L, and 150umol/L at 1 month, 3 months, 6 months, 1 year, and 5 years after transplantation respectively.**Conclusion** DCD kidney transplantations in NZ had favourable long-term graft survival with good renal function despite high DGF in the initial post-transplantation period.Abstract No. 91**EVALUATING ALLOGRAFT RENAL FUNCTION BY CYSTATIN C ESTIMATING GLOMERULAR FILTRATION RATE FORMULAS AFTER KIDNEY TRANSPLANTATION****TAM Tran Thai Thanh¹, HOANG Khac Chuan², DU Thi Ngoc Thu², THAI Minh Sam², LE Nguyen Thi³, TRAN Ngoc Sinh³**¹*Cho ray Hospital, HCMC, Vietnam,* ²*Renal Transplant Unit, Cho Ray Hospital, HCMC, Vietnam,* ³*School of Medicine, HCMC University of Medicine and Pharmacy, Vietnam***Background:** Assessing accurate estimation of glomerular filtration rate (GFR) in kidney transplant recipients is very important.**Objectives:** We compared equations based on serum cystatin C (ScysC) alone or combined with serum creatinine (Scr), with formulas based on Scr-based alone to estimate GFR as precisely and simply as possible in kidney transplant recipients.**Subjects and methods:** 186 kidney transplant recipients with stable kidney function were included in our study. The patients' GFRs were estimated by 2 creatinine-based equations (the modification of diet in renal disease (MDRD), Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI creatinine 2009), 1 creatinine-cystatin C based equation (CKD-EPI creatinine-cystatin C 2012) and 7 cystatin C-based equations (Arnall Dade, Filler-LePage, Hoek, Grubb, Le Bricon, Rule, and CKD-EPI cystatin C 2012).**Results:** The mean age of the subjects was 42.95±11.2 years. CKD-EPI creatinine-cystatin C 2012 and Hoek equations appeared the least biased (Δ mGFR: 0.64±13.2; 0.13±14.3 ml/min/1.73m², respectively) and had the best correlation with mGFR ($r_1 = 0.734$, $r_2 = 0.736$, ($p < 0.001$)). CKD-EPI creatinine-cystatin C 2012 had the highest sensitivity and specificity 84,8% and 67,4% to diagnose mGFR <60mL/min/1.73m².**Conclusions:** We found that equations based on ScysC alone or combined with Scr were better than formulas based on Scr-based alone to estimate GFR in kidney transplant recipients.

Abstract No. 92

RANGE AND CONSISTENCY OF CARDIOVASCULAR OUTCOMES REPORTED IN CONTEMPORARY RANDOMISED TRIALS IN KIDNEY TRANSPLANT PATIENTS: A SYSTEMATIC REVIEW

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Background: Cardiovascular disease (CVD) is the primary cause of death and a major contributor to graft loss in kidney transplant recipients. However, inconsistent reporting of cardiovascular outcomes may limit assessment of the comparative effect of interventions across trials and the use of trial evidence in decision-making.

Aims: To determine the scope and consistency of cardiovascular outcomes reported in contemporary trials in kidney transplant recipients.

Methods: MEDLINE, Embase, the Cochrane Kidney and Transplant Specialized Register, and ClinicalTrials.gov were searched from 2013 and 2017 to identify randomised trials and trial protocols reporting any cardiovascular outcome. Definitions, measures and timepoints for all CVD outcomes were extracted and analysed.

Results: From 81 trials, 1097 CVD different measures were extracted and categorised into 37 CVD outcomes. The three most frequently reported outcomes were: cardiovascular composites (35 [43%] trials), all-cause mortality (29 [36%] trials), and acute coronary syndrome (28 [35%] trials). Cardiovascular composites were reported in 33 different combinations of components, with 29 being unique to a single trial.

Conclusions: There is extreme heterogeneity in the reporting of cardiovascular outcomes in trials in kidney transplant patients. CVD composite outcomes vary widely. Establishing a standardized CVD outcome, that is critically important to patients and clinicians, may improve the relevance and use of trials to inform decision-making.

Abstract No. 93

ARTERIAL RECONSTRUCTION IN LIVER TRANSPLANT WITH BOTH DONOR AND RECIPIENT HEPATIC ARTERY ANATOMICAL VARIATIONS

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Background Hepatic artery anatomical variation is not uncommon. Any variation in donor or recipient hepatic arterial supply potentially increases the arterial complication rate following liver transplantation.

Methods Here we present 2 cases of liver transplant where both the donor and recipient had unusual variation of the hepatic arterial supply. In the first case, the donor had stenotic coeliac trunk whereby all hepatic arterial inflow occurred via a dilated tortuous gastroduodenal artery (GDA) which back filled via SMA. The recipient had an aberrant right hepatic artery (RHA) from SMA, plus conventional common hepatic artery (CHA) from coeliac trunk. During the transplant two arterial anastomoses were performed using donor CHA to recipient CHA/GDA patch, and using donor GDA to recipient aberrant RHA. In the second case, the donor had an aberrant LHA from LGA which independently arose from the supra-coeliac aorta, and accessory RHA from SMA, plus conventional CHA. The recipient had conventional CHA and both accessory LHA from LGA and RHA from SMA. Considering each artery's size, orientation and flow, the donor LGA/aortic patch was anastomosed to donor splenic artery on back table, then we anastomosed the donor coeliac trunk to recipient CHA/GDA patch, plus donor aberrant RHA/SMA patch to recipient aberrant RHA during implantation.

Results The total arterial anastomosis time in each case was 39min and 76min respectively. Neither case required blood transfusion, and both post-transplant recoveries were uneventful.

Conclusions Techniques of hepatic arterial reconstruction in liver transplant with both donor and recipient hepatic artery anatomical variation should be individualised. Factors to be considered include the types of variation, size, orientation, flow and quality of the arteries on both sides.

Abstract No. 94

PROPHYLACTIC DRAIN INSERTION IN RENAL TRANSPLANTATION: SURGEON PREFERENCE ACROSS AUSTRALIA AND NEW ZEALAND

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Aims: There are no guidelines available concerning the use of prophylactic drain at the conclusion of renal transplantation (RT) in order to prevent post-operative complications such as lymphoceles. We aim to provide a summary of practice amongst renal transplant surgeons across Australia and New Zealand (ANZ).

Methods: An online survey for surgeons who routinely conduct RT across ANZ transplant centres was conducted to study respondents' demographic information, surgical experience, preference regarding prophylactic drain insertion and their post-operative practice.

Results: 43 out of 66 identified surgeons completed the survey. 41.9% were general surgeons with subspecialisation in transplantation (18.6%) and hepatobiliary surgery (18.6%); 37.2% were vascular surgeons; 13.9% were urologists and 7% were transplantation and dialysis access surgeons.

60.5% of surgeons reported that they insert perigraft drain routinely whereas 20.9% seldom insert drains. The most common reason (58.1%) for drain insertion was "routine practice". 30.2% of respondents were "uncertain about benefit of drain use" whereas 48.8% felt that this reduced symptomatic peritransplant fluid.

44.2% of respondents consider both volume and time as important factors for drain removal with less emphasis on the fluid composition. Mean post-operative day for drain removal was at 4.56 days. Some surgeons test drain creatinine to exclude urine leak (16.3%). 74.4% of surgeons would consider enrolling their patients for RCT to determine benefit of drain insertion.

Conclusion: There is a wide range of practices amongst RT surgeons. Individual surgeons' experience appears to be the greatest factor in decision making.

Abstract No. 95

USE OF AN ICE BAG TO MINIMIZE THE PERIOD OF SECOND WARM ISCHAEMIC TIME DURING KIDNEY & PANCREAS TRANSPLANTATION – OUR INITIAL EXPERIENCE**YOON Peter¹, HAMEED Ahmer^{1,2}, NGUYEN Hien^{1,3}, GASPI Renan⁴, HAWTHORNE Wayne⁵, PLEASS Henry¹, YUEN Lawrence¹****¹Department of Surgery, Westmead Hospital, Sydney, ²Centre for Transplant and Renal Research, The Westmead Institute for Medical Research, ³Department of Urology and Kidney Transplant, Cho Ray Hospital, Vietnam, ⁴Department of Renal Medicine, Westmead Hospital, Sydney, ⁵Centre for Transplant and Renal Research, Other****Aims:** To assess the safety and feasibility of performing anastomoses for kidney and/or pancreas transplantation after organ immersion in a bag of ice slush.**Methods:** Kidneys alone (n = 4) and/or the kidney & pancreas (n = 1) were retrieved from deceased donors and transported to our center using standard cold static storage. After back-table preparation of the graft, each organ was immersed in ice slush within a sterile bowel bag. The bag was sealed superiorly, and a small perforation was made to allow vessel extrusion. During anastomoses, ice slush was replenished as required; anastomoses were performed in a standard manner to the iliac vessels. The bag was removed prior to reperfusion. (Representative video will be shown during presentation).**Results:** All transplants were completed safely, without any visual obstruction during anastomoses. Whilst mean anastomotic time for kidneys and pancreas was 49 ± 8 mins and 32 mins, respectively, the second warm ischaemic time for all organs was < 1 minute. There were two cases of delayed graft function, both in DCD kidneys (KDPI 98 & 38). One-month creatinine in these recipients was 214 and 134 $\mu\text{mol/L}$, respectively. Both DBD kidney recipients (KDPI 69 & 74) had immediate graft function. The kidney/pancreas recipient also had immediate graft function, a one-month creatinine of 60 $\mu\text{mol/L}$, and was off all insulin.**Conclusions:** Kidney/pancreas placement in an ice bag is a convenient, simple, and non-obstructive means of minimizing the secondary warm ischaemic insult. A planned RCT will formally test its efficacy.

President's Prize Symposium

Abstract No. 96

THE GP130 RECEPTOR IS LOST DURING GRANULOPOIESIS, RENDERING NEUTROPHILS INERT TO IL-6 AND RELATED CYTOKINES.

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Introduction: Interleukin-6 (IL-6) mediates broad physiological and pathological effects on numerous cell types through a number of signalling cascades, complicating our understanding of the mechanisms of action of IL-6R inhibition in the clinic. IL-6 is thought to signal ubiquitously due to wide cellular expression of its signal transducing protein, gp130. IL-6 forms a complex with gp130 and either the membrane-bound IL-6 receptor (mIL-6R α) or the soluble IL-6 receptor (sIL-6R α), resulting in classical or trans-signalling that induces phosphorylation of STAT3 (pSTAT3). IL-6R inhibition shows efficacy in preventing GVHD and neutrophils are increasingly recognized to mediate pathology after stem cell transplantation (SCT). Given that granulopoiesis is IL-6 sensitive and neutropenia is the principal side effect of IL-6R inhibition, we sort to clarify the signalling cascades in immune cell subsets after SCT.

Results: In contrast to dogma, we show that unlike monocytes or T cells, circulating human and mouse granulocytes are unresponsive to IL-6 since stimulation with either IL-6 or IL-6/sIL-6R α fails to induce pSTAT3 expression. Signalling to other gp130 family cytokines (e.g. IL-27) was also absent. We demonstrate that this is due to the absence of gp130 expression on granulocytes, despite high expression of the IL-6R α . Importantly, the absence of gp130 is not only a feature of mature granulocytes in healthy individuals, but is also observed after count recovery from clinical SCT. Analysis of stem cell populations in mouse bone marrow and human G-CSF mobilized grafts demonstrates that hematopoietic stem cells and granulocyte-monocyte progenitors (GMPs) express gp130 and respond normally to IL-6, indicating that gp130 expression is lost during myeloid maturation. These data confirm that the effects of IL-6 inhibition are independent of neutrophils and the transient neutropenia observed in patients after IL-6 inhibition is thus likely a result of indirect effects on neutrophils or direct modulation of immature progenitors.

Conclusion: Given that granulocytes constitute 50-70% of circulating leukocytes, this indicates a significantly smaller scope of IL-6 signalling than previously anticipated. This data has important implications for therapeutic IL-6 inhibition and challenges the currently espoused mechanisms of action.

Abstract No. 97

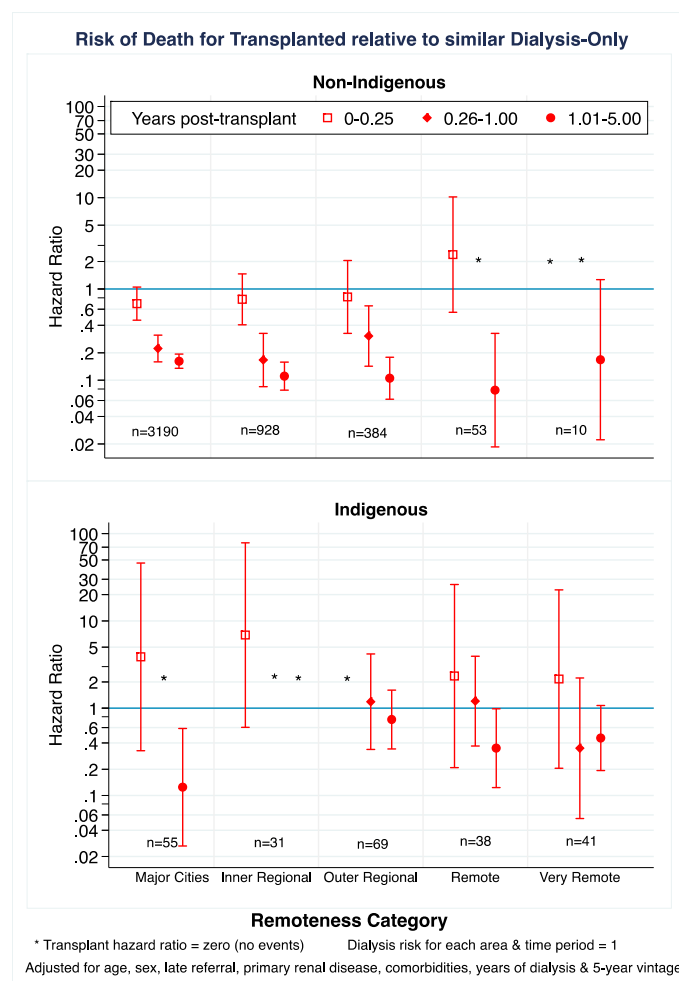
DO INDIGENOUS PATIENTS HAVE BETTER SURVIVAL WITH A KIDNEY TRANSPLANT COMPARED TO STAYING ON DIALYSIS? A PROPENSITY MATCHED STUDY**LAWTON Paul¹, CUNNINGHAM Joan¹, ZHAO Yuejen², JOSE Matthew³, CASS Alan¹**¹*Wellbeing & Preventable Chronic Diseases Division, Menzies School of Health Research, Charles Darwin University,* ²*Innovation & Research Branch, Department of Health, Northern Territory Government,* ³*Department of Medicine, University of Tasmania*

Aim: Indigenous patients are unlikely to be wait-listed for or receive a kidney transplant. Many clinicians are concerned about Indigenous transplant outcomes. We compared survival for Indigenous transplant patients with similar Indigenous dialysis-only patients, contrasting with non-Indigenous patients.

Methods: Using ANZDATA, all Australians commencing renal replacement therapy from 1st April 1995 were followed until 31st December 2015. Transplant recipients were paired by propensity score with similar dialysis-only patients of the same ethnicity within four time cohorts: time at risk for each pair was taken from the transplant date. All-cause survival was compared using unadjusted and stratified Cox proportional hazards models for three time periods post-transplant (accounting for non-proportional hazards), adjusted for demographic and clinical differences and a transplanted-remoteness interaction term.

Results: Indigenous dialysis-only patients were similar to their transplanted pair at baseline, but paired non-Indigenous patients were less similar. Unadjusted five year survival was better for transplanted patients than their dialysis-only pair for non-Indigenous ($p<0.0001$) and Indigenous patients ($p=0.0005$). Adjusted Cox models comparing transplanted with dialysis-only patients showed early (0-0.25 years post-transplant) survival equivalence for both Indigenous and non-Indigenous patients, with improvements in subsequent transplanted survival clearest for all non-Indigenous patients except from very remote areas, and for Indigenous patients in major cities (MC) and inner regional (IR) areas.

Conclusions: Indigenous transplanted patients have similar or better survival to similar dialysis-only patients, with long-term benefit in MC/IR. Relatively fewer apparently suitable Indigenous patients received transplants. These data provide direction for future targeted clinical and health services research.



Abstract No. 98

IN VIVO REGULATORY T-CELL GENERATION WITH DENDRITIC CELL TARGETING NANOPARTICLES

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Porous silicon nanoparticles (pSiNP), modified to target DC, provide a novel strategy for the delivery of immunosuppressive drugs.

Aim: Conjugation of DC-SIGN and CD11c antibodies to rapamycin-loaded-pSiNP inducing immune regulation.

Method: pSiNP functionalised with mAb were co-cultured with human monocyte-derived DC to assessed uptake after 24h. Rapamycin-pSiNP treated DC were allogeneic stimulators in MLRs to assess their stimulatory capacity. In vivo pSiNP targeting was determined by i.v. injection of B57BL/6 mice and common marmosets (*Callithrix jacchus*) with fluorescent CD11c or DC-SIGN-pSiNP (20mg/kg). Mice and marmosets (n=5) were imaged to assess particle distribution and flow cytometry was used to determine blood and splenocytes DC uptake. Ovalbumin (OVA) sensitized mice received i.v. injections of rapamycin-CD11c-pSiNP ± OVA₃₂₃₋₃₃₉. Splenic CD4⁺CD25⁺FoxP3⁺ T-regs numbers were assessed via flow cytometry.

Results: pSiNP showed time- and dose-dependent DC targeting *in vitro*. Rapamycin-pSiNP decreased DC markers CD40, CD80, CD86 and CD83 expression. Treated DC significantly inhibited allogeneic T-cell proliferation by 50% compared to mature DC (n=3, p<0.01). *In vivo*, pSiNP tracked to kidneys, lungs, liver and spleen in mice and marmosets. Flow cytometry indicated splenic DC uptake of pSiNP. OVA sensitized mice, receiving CD11c-pSiNP (rapamycin + OVA₃₂₃₋₃₃₉), had a 5-fold higher concentration of splenic T-regs compared to control mice (n=5, p<0.01).

Conclusion: Rapamycin loaded nanoparticles functionalised with DC-SIGN mAb provide targeted delivery to DC *in vitro*. *In vivo* we are the first to show the tracking of DC-SIGN-pSiNP in a non-human primate model. CD11c-pSiNP loaded with immunosuppressant rapamycin and antigen demonstrate an ability to enhance regulatory T-cell generation *in vivo*.

Abstract No. 99

RESIDUAL RISK OF BLOOD BORNE VIRUS INFECTION WHEN AUSTRALIAN ORGAN DONOR REFERRALS TEST NEGATIVE: A SYSTEMATIC REVIEW AND META-ANALYSIS.

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Introduction: Donor referrals with increased risk behaviours who test negative for blood borne viruses are currently submitted to recipient teams as suitable organ donors. However window period infections (the period between infection and tests becoming positive) may pose risk to recipients unless suitable screening is undertaken. **Aim:** To estimate the prevalence and incidence of hepatitis B (HBV), hepatitis C (HCV) and HIV (human immunodeficiency virus) among increased risk groups in Australia, and hence infer the residual risk of window period infection for organ donors with negative testing.

Methods: We performed a systematic review and meta-analysis including studies 2000-2017 reporting original estimates of Australian HIV, HCV or HBV prevalence or incidence in increased risk groups. Pooled prevalence and incidence rates were estimated using random effects. The probability of window period infection was estimated by assuming days since infection followed an exponential distribution.

Results: We included 55 studies (353,846 participants), with most data for MSM (men who have sex with men), IVDU (intravenous drug users) and prisoners; HIV and HCV. The absolute residual risk of HIV infection remained low in all cases; the highest was MSM with up to 9 window period infections per 10,000 not detected with negative enzyme immunoassay (EIA) testing alone (Table 1). HCV and HBV incidence was highest in IVDU, with up to 158 window period HCV cases per 10,000 people not detected by EIA testing alone.

Conclusions: BBV risk estimates inform decisions about increased risk donor referrals. Negative NAT substantially reduces window period risks.

Abstract No. 99 (ctd)

Table 1: Pooled incidence and estimate of residual infection risk given negative EIA (enzyme immunoassay), or both negative NAT (nucleic acid testing) and EIA, for each blood borne virus and each increased risk group.

Risk group	Patients	Pooled incidence (95% CI) per 100 person-years	Residual risk per 10,000 with negative testing (95% CI)	
			EIA	EIA + NAT
Human immunodeficiency virus				
MSM	7167	0.98 (0.61-1.43)	5.9 (3.7-8.6)	1.9 (1.2-2.7)
IVDU	38836*	0.15 (0.13-0.17)	0.9 (0.8-1.1)	0.3 (0.3-0.3)
Prisoners	9409*	0.03 (0.00-0.08)	0.2 (0.0-0.5)	0.1 (0.0-0.1)
CSW	2516*	0.05 (0.04-0.07)	0.3 (0.3-0.4)	0.1 (0.1-0.1)
Increased risk partner	522*	0.03 (0.00-0.12)	0.2 (0.0-0.7)	0.1 (0.0-0.2)
Hepatitis C				
MSM	1917*	1.9 (0.2-5.3)	11.4 (0.9-31.9)	3.6 (0.3-10.2)
IVDU	1859	17.0 (9.3-26.3)	101.8 (56.0-157.2)	32.5 (17.9-50.3)
Prisoners	565	15.6 (13.4-19.6)	93.5 (80.6-117.1)	29.9 (25.7-37.4)
CSW	390*	6.7 (0.3-18.2)	40.2 (1.5-108.9)	12.8 (0.5-34.8)
Increased risk partner	50*	8.3 (5.9-13.4)	50.0 (35.3-80.2)	15.9 (11.3-25.6)
Hepatitis B core antibody				
MSM	11035*	1.9 (0.3-5.7)	11.3 (2.1-34.3)	3.6 (0.7-10.9)
IVDU	1401*	8.1 (3.3-15.8)	48.8 (20.0-94.9)	15.6 (6.4-30.3)
Prisoners	1599*	3.5 (1.7-6.0)	21.1 (10.3-36.2)	6.7 (3.3-11.5)
CSW	1089*	0.4 (0.2-1.0)	2.7 (1.0-6.2)	0.9 (0.3-2.0)
Increased risk partner	471*	0.2 (0.0-0.7)	0.9 (0.1-4.0)	0.3 (0.0-1.3)
Hepatitis B surface antigen				
MSM	3974*	0.4 (0.2-0.8)	2.6 (1.2-4.9)	0.8 (0.4-1.6)
IVDU	30970*	0.7 (0.3-1.3)	4.1 (1.7-8.0)	1.3 (0.6-2.5)
Prisoners	1270*	0.4 (0.2-1.0)	2.7 (1.0-5.8)	0.9 (0.3-1.9)

CSW - commercial sex workers

* denotes patient numbers where pooled incidence was calculated from pooled prevalence data. CI – confidence interval.

Abstract No. 100

UBIQUITIN LIGASE MARCH8 ATTENUATES GRAFT VERSUS HOST DISEASE VIA REGULATION OF GUT EPITHELIAL CELL SURFACE MHC II EXPRESSION

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Following Allogeneic stem cell transplantation (SCT), MHCII dependent priming of donor CD4+ T cells by host non-hematopoietic cells is sufficient to elicit GVHD. The membrane-associated RING-CH (MARCH) family of E3 ubiquitin ligases function as immune regulators by limiting the expression of key immune receptors. Notably, March8 limits the expression of MHCII on non-hematopoietic cells by ubiquitinating MHCII, leading to it being trafficked from the plasma membrane for lysosomal degradation.

Results: In this study we demonstrate constitutive, non-hematopoietic-restricted expression of March8 mRNA throughout the intestinal tract, and elevated cell surface MHCII expression on intestinal epithelial cells (IEC) from MARCH8 deficient (MARCH8^{-/-}) mice compared to wild type (WT). Histological comparison of the gut between MARCH8^{-/-} and WT mice appeared normal. Further to this, colonic crypts were cultured from both MARCH8^{-/-} and WT mice suggesting no difference in their capacity for growth. Using the BALB/c into B6 model of GVHD, MARCH8^{-/-} recipients exhibited significantly increased clinical scores and reduced survival with 100% of MARCH8^{-/-} recipients succumbing to disease by D9 post-transplant while >60% WT recipient mice survive beyond D40 (p= 0.0062). At D7 post-SCT MARCH8^{-/-} recipients demonstrated significantly increased gut pathology associated with a marked increase in donor T cell infiltration. This was donor T cell dependent, as MARCH8^{-/-} recipients of T cell-depleted grafts survive long term.

Conclusions: Taken together our data identifies a critical role for MHCII antigen presentation by IEC in the initiation of GVHD, and an immunomodulatory role for MARCH8 for the control of IEC MHCII expression and GVHD severity.

Abstract No. 101

DONOR SPECIFIC ANTIBODIES AND CLINICAL OUTCOMES IN KIDNEY TRANSPLANT RECIPIENTS**WAN Susan^{1,2}, CHADBAN Steven^{1,2}, WYBURN Kate^{1,3}**¹Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney, ²Sydney Medical School, University of Sydney, ³Other, University of Sydney

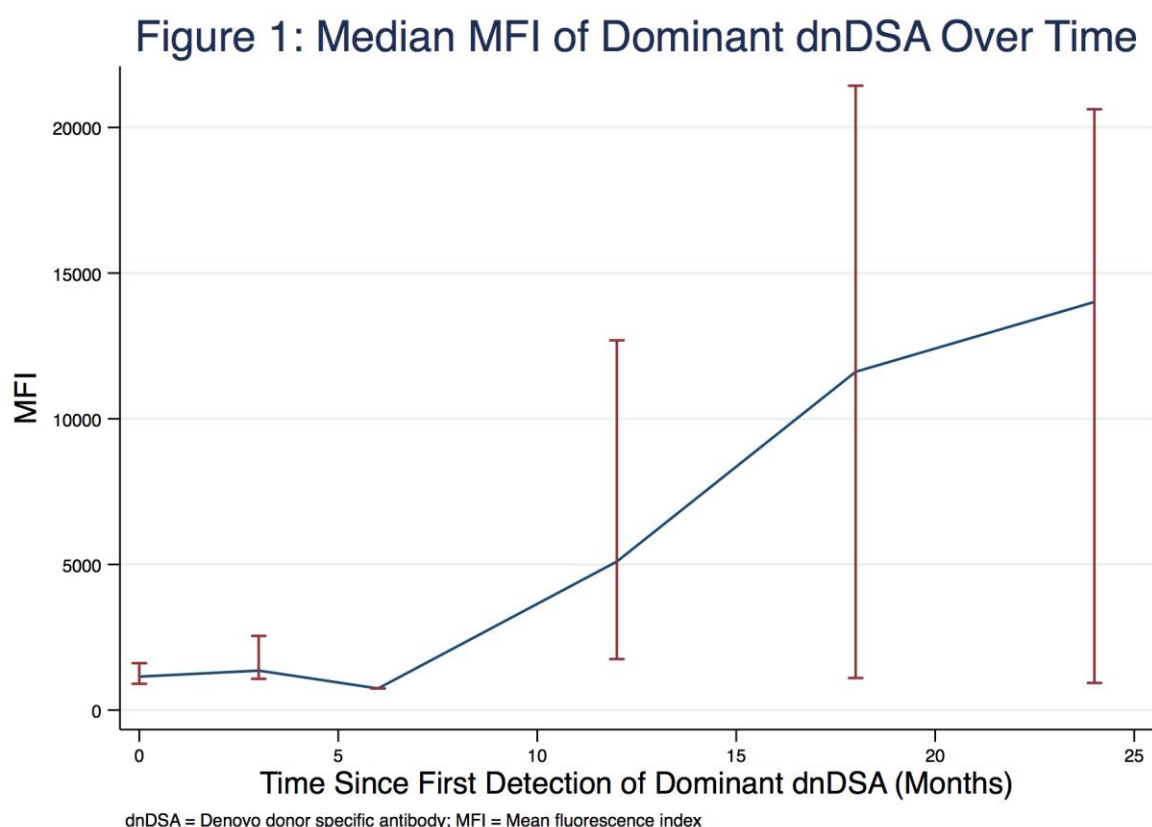
BACKGROUND: Donor specific antibodies (DSA) are implicated in acute rejection (AR) and graft dysfunction in kidney transplant recipients (KTx). However, limited data exists on their natural history post-transplantation.

AIMS: To describe the natural history of pre-transplant (preDSA) and *denovo* DSA (dnDSA) in KTx.

METHODS: We performed a prospective single-centre cohort study in KTx. Patients were screened for DSA at 0, 3 and 12-months post-transplant, and associations between DSA and outcomes were assessed.

RESULTS: 363 KTx between 2010-2017 underwent pre- and post-transplant DSA screening. 136(37%) had preDSA at transplantation; 86(63%) had ClassI and 89(65%) had ClassII. The median MFI of the dominant preDSA was 1230 (IQR 746-2528) at transplantation and declined rapidly, becoming undetectable by 1-month post-transplant. DnDSA were detected in 62(17%) recipients; 28(45%) had ClassI and 45(73%) had ClassII. The median time to first detection of dnDSA was 58 days (IQR 15-267) and the median MFI of the dominant dnDSA was 1150 (IQR 707-2694) at first detection. The MFI of the dominant dnDSA increased over time reaching a median of 14,011 (IQR 931-20,626) at 2 years (Figure 1). 58(26%) of 220 patients with ≥ 2 -years follow-up developed AR; 42(19%) had cell-mediated rejection, 20(9%) had antibody-mediated rejection. The development of dnDSA was strongly associated with AR (OR 4.48; 95% CI 2.14-9.36; $P < 0.001$) but not with eGFR or graft-survival.

CONCLUSION: PreDSA were present in 37% of KTx and were significantly reduced by 1-month post-transplant. dnDSA were detected in 17% and increased in intensity over time. The development of dnDSA was strongly associated with AR.



Abstract No. 102

INTRA-RENAL DELIVERY OF DRUGS TARGETING ISCHEMIA-REPERFUSION INJURY OF THE KIDNEY IN A RODENT MODEL & PORCINE MODEL OF NORMOTHERMIC MACHINE PERFUSION

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Aims: To investigate the utility of direct renal delivery of drugs targeting ischaemia-reperfusion injury (IRI) using machine perfusion (MP).

Methods: (i) Three different IRI drugs targeting the IRI process (CD47 blocking antibody, soluble complement receptor 1 [sCR1], and recombinant thrombomodulin [rTM]) were compared in a rodent model of renal IRI. A single drug or combination was delivered via the inferior vena cava after a right nephrectomy, and preceding induction of left kidney ischaemia. (ii) A normothermic MP (NMP) system was developed and optimized using a porcine donation after circulatory death (DCD) model. The impact of drug/s identified from (i) on NMP perfusion parameters and histology was subsequently investigated.

Results: (i) Preliminary evidence in the rodent renal IRI model was indicative of significant amelioration of IRI when CD47 and/or sCR1 were given prior to the ischaemic hit. Ongoing work is investigating whether the combined use of these agents will produce synergistic effects. (ii) A clinically translatable system of NMP was developed and optimized. Ideal perfusion conditions were produced when pressure-controlled perfusion, utilizing a leukocyte-depleted and colloid-containing perfusion solution were employed. Addition of CD47 to the perfusion circuit impacts on perfusion and biochemical parameters, including resistance, urine output and creatinine clearance. Ongoing work will test the utility of combined agents in this system.

Conclusions: Renal IRI can be ameliorated by the direct delivery of CD47 and/or sCR1. These drugs can be directly delivered to the kidney using NMP, thereby avoiding systemic treatment. The impact on transplantation outcomes remains to be investigated.

Abstract No. 103

FAVOURABLE CARDIAC REMODELING AND FUNCTIONAL CARDIAC BENEFITS ASSESSED WITH CARDIAC MAGNETIC RESONANCE IMAGING FOLLOWING LIGATION OF ARTERIOVENOUS FISTULA IN STABLE RENAL TRANSPLANT RECIPIENTS: A RANDOMIZED, CONTROLLED, OPEN LABEL STUDY

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Aim: To study the change in left ventricular mass(LVM) following ligation of arteriovenous fistula(AVF) in stable renal transplant recipients(RTR), utilizing cardiac MRI(CMR).

Methods: In this randomized controlled trial, we recruited participants aged 18 years or older with stable kidney function twelve months post kidney transplantation, and a functioning AVF, from a tertiary network of renal transplantation service in Australia. Participants were randomly assigned (1:1) to have AVF ligated or not, with all participants undergoing a baseline CMR followed by repeat scan six months later. The primary outcome was change in LVM at 6 months, analyzed according to intention-to-treat principles.

Results: We enrolled 93 participants. 63 eligible participants underwent randomization. 54 out of 63 participants completed assessments after second CMR. The mean LVM decreased by 22 gms (14.7%) [151.2 ± 36.5 gm vs. 129.1 ± 32.4 gm, $p < 0.001$ in the intervention group vs 153.4 ± 47.8 gm vs. 154.6 ± 43.0 gm, $p = 0.69$ in the non-intervention group]. Significant improvements were also noted in end-diastolic volumes, end-systolic volumes, and stroke volumes of both left and right ventricles. There was also an improvement in the atrial volumes. No significant complications were noted after AVF ligation.

Conclusion: In this randomized controlled trial for adults with stable kidney transplantation and functioning AVF, elective ligation of AVF is associated with a 14.7% decrease in LVM as assessed by CMR. This was also associated with improvements in other cardiac parameters.

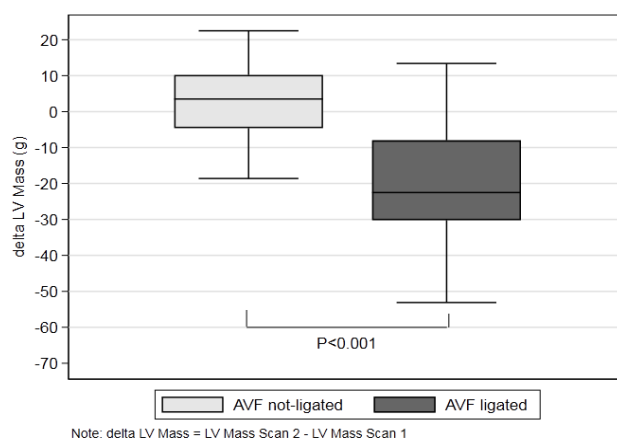


Figure 1: Difference (delta) in the LV mass between the two scans in each group

Figure 7. (a) Difference (delta) in the LV mass between the two scans in each group, (b) Difference in the LV mass index(BSA) between the two scans in each group

Surgical

Abstract No. 104

ENHANCED RECOVERY AFTER SURGERY AND THE RENAL TRANSPLANT RECIPIENT – USEFUL OR A WASTE OF TIME?

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Enhanced Recovery After Surgery (ERAS) pathways are an accepted part of modern surgical practice. In renal transplant recipients, however, there is no clear consensus regarding their utility.

The renal transplant unit at the Royal Adelaide Hospital introduced an ERAS protocol for the perioperative management of transplant recipients from June 2017. We present the outcomes of 39 consecutive cases.

Patient and Methods All patients who had a renal transplant from introduction of the protocol until the time of writing were enrolled in the ERAS protocol. The protocol included pre-operative weight optimization on dialysis; perioperative carbohydrate loading; goal-directed fluid management prior to reperfusion; fluid balance, aiming to achieve a net weight gain $\leq 3\text{kg}$ in the first 24 hours; opiate avoidance and use of regional wound infusers.

There were nine recipients from a live donor; 23 from donation after brainstem death (DBD) and 7 from donation after circulatory death (DCD).

Results The mean weight gain on post-operative day 1 was 3.06Kg.

None of the live donor recipients had delayed graft function (DGF) requiring dialysis. DGF was observed in 28.6% of the DCD graft recipients and 39.1% of the DBD graft recipients.

Mean length of stay was 4.6 days.

Conclusion Our experience challenges the widespread practice of fluid loading post renal transplant. Our readmission rate has not increased and early results suggest that there are significant gains to be made via reduced length of hospital stay.

Abstract No. 105

TRANSITION FROM LAPAROSCOPY TO RETROPERITONEOSCOPY FOR LIVE DONOR NEPHRECTOMY - A CASE CONTROL STUDY

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Aims Transperitoneal (TLDN) approach for laparoscopic donor nephrectomy (LDN) is widely adopted in most centres. However, a systematic review has shown that retroperitoneoscopic (RLDN) approach is associated with fewer complications due to the anatomical advantage by avoidance of manipulation of the intraperitoneal organs. The aims of this study were to compare the outcomes of RLDN and TLDN by a case control study. The learning curve for transition from TLDN to RLDN was analyzed.

Methods A retrospective analysis of all LDNs from 2010 to Oct 2017 were performed. Data on demographics, peri-operative parameters, analgesia consumption, pain scores and kidney graft function were collected and analyzed. A CUSUM analysis was performed to explore the learning curve of RLDN by setting the mean operative goal time of TLDN as a target.

Results All these 122 donor nephrectomies (60 TLDN and 62 RLDN) were successful with no conversion to open surgery. There was no blood transfusion, readmission or mortality. There were no post-operative complications which were graded over Clavien II. The kidney graft function was comparable in both groups. The follow-up period ranged from 4 to 90 months. The CUSUM analysis demonstrated that approximately 30 cases are required for the surgeon to be proficient in the transition from TLDN to RLDN.

Conclusions RLDN is a safe approach with comparable results to TLDN. It avoids manipulating the intraperitoneal organs and retains a virgin abdomen and hence reduces peri-operative complication risk. The learning curve of transitioning from TLDN to RLDN is acceptable.

Abstract No. 106

EVALUATION OF RISK FACTORS FOR ENTERIC LEAKS FOLLOWING SIMULTANEOUS PANCREAS AND KIDNEY TRANSPLANTATION**HORT Amy¹, SHAHRESTANI Sara¹, HITOS Kerry¹, ROBERTSON Paul¹, LAM Vincent¹, YUEN Lawrence¹, RYAN Brendan¹, DE ROO Ronald², HAWTHORNE Wayne J^{2,3,4}, PLEASS Henry¹**¹*Westmead Hospital, Sydney*, ²*Department of Surgery, Westmead Hospital, Sydney*, ³*Discipline of Surgery, Sydney Medical School, University of Sydney, Sydney, Australia*, ⁴*University of Sydney, Centre for Transplant and Renal Research, Westmead Institute of Medical Research, Westmead Hospital, Westmead, Australia, Westmead Hospital, Sydney***Introduction** Simultaneous pancreas-kidney transplantation (SPK) is the gold standard treatment for patients suffering Type I Diabetes Mellitus and End-Stage Renal Failure. Enteric drainage is utilised to handle the exocrine drainage, however, enteric leaks (ELs) are one of its more specific and challenging complications. There remains a lack of published research regarding risk factors for ELs, particularly associated with vascular disease.**Methods** SPK transplants performed at Westmead Hospital over ten years (between 2008-2017, n = 234) were analysed to identify ELs. Donor, patient and transplantation procedure risk factors for ELs were collected and analysed. Adjusting for possible confounders, a multivariate logistic regression model was used to assess the risk and predictors of ELs.**Results and Discussion** Of the 234 patients, 12 (5%) experienced an EL. Of these recipients, 9 (75%) had vascular disease, 6 (50%) were ex-smokers, 1 (8%) a current smoker and 3 (25%) were obese with a BMI >30kg/m². The risk of EL increased by as much as 4.4 fold in recipients with vascular disease (OR: 4.4; 95% CI: 0.80-24.21; P=0.088). Other factors such as recipient BMI >24.2kg/m² increased the risk of EL by as much as 1.8 fold (OR: 1.8; 95% CI: 0.4-9.3; P=0.46).**Conclusions** We have a possible trend between vascular disease and ELs. These findings also identify other possible risk factors for ELs and the need for further research in this area including careful screening of recipients for vascular disease.

Abstract No. 107

AORTIC VERSUS DUAL PERFUSION FOR RETRIEVAL OF THE DBD LIVER – AN ANALYSIS OF RECIPIENT OUTCOMES USING THE ANZ LIVER TRANSPLANT REGISTRY**HAMEED Ahmer^{1,2}, PANG Tony^{2,3}, YOON Peter², BALDERSON Glenda⁴, RONALD De Roo², YUEN Lawrence^{2,3}, LAURENCE Jerome^{5,3}, LAM Vincent^{2,3}, CRAWFORD Michael⁶, HAWTHORNE Wayne^{1,2}, PLEASS Henry^{2,3}**¹*Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney*, ²*Department of Surgery, Westmead Hospital, Sydney*, ³*School of Medicine, University of Sydney*, ⁴*Australia and New Zealand Liver Transplant Registry, Princess Alexandra Hospital, Brisbane*, ⁵*Institute for Academic Surgery, Royal Prince Alfred Hospital, Sydney*, ⁶*Department of Surgery, Royal Prince Alfred Hospital, Sydney***Aims:** To compare the impact of aortic-only and dual (aorta and portal vein) perfusion on donation after brain death (DBD) liver transplantation outcomes.**Methods:** DBD liver transplants performed in Australia (2007-16) were included in analyses, and stratified by aortic or dual perfusion routes. The ANZLTR, ANZOD, and a national survey of senior donor surgeons were used to obtain all data-points. Only livers preserved in University of Wisconsin solution were included; patients receiving a subsequent liver transplant, or a reduced size graft were excluded. Graft and patient survival were compared using Kaplan-Meier curves and Cox proportional hazards. Causes of graft loss, including primary non-function, hepatic artery and portal vein thrombosis, biliary complications, and acute rejection, were compared using logistic regression.**Results:** Aortic-only perfusion was utilized in 957 cases, compared to 425 dual-perfused livers. The dual-perfused group had a lower mean cold ischaemia time, secondary warm ischaemic time, and MELD score (p < 0.001). Actuarial 5-year graft and patient survivals in the aortic-only and dual-perfused cohorts were 80.1% vs 84.6% (p = 0.066), and 82.6% vs 87.8% (p = 0.026), respectively. After accounting for all confounders, graft (HR 0.81, 95% CI 0.60-1.11, p = 0.188) and patient (HR 0.74, 95% CI 0.52-1.05, p = 0.087) survival were not significantly different between both cohorts. There were no differences between both groups with respect to causes of graft loss. Subgroup analyses are being conducted to compare high-risk donors.**Conclusions:** The retrieval technique employed does not impact outcomes when all DBD donors are considered together.

Xenotransplantation

Abstract No. 108

GENETICALLY MODIFIED PORCINE NEONATAL ISLET XENOGRAFTS PROVIDE LONG-TERM FUNCTION IN BABOONS

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INTRODUCTION: Alternative strategies such as Xenotransplantation show great promise to be able to provide the organs required to treat diseases such as Type 1 Diabetes.

AIMS: To achieve long-term normoglycemia in diabetic baboons transplanted with neonatal pig islets, and to investigate the effect of ceasing immunosuppression.

MATERIALS AND METHODS: Five diabetic baboons received transplantation of NICC (10,000-50,000 IEQ/kg) from GTKO/CD55-CD59-HT piglets. From day -3 recipients were treated with anti-CD2 induction and maintenance with oral tacrolimus, anti-CD154 and belatacept, which were progressively ceased. Graft survival and function followed by daily blood sugar levels (BSL), IVGTT, OGTT and immunohistochemical analysis of liver biopsies taken at various time points.

RESULTS: No baboon exhibited signs of thrombosis associated with IBMIR. Recipients developed normal fasting BSL and normal IVGTT and OGTT, with porcine insulin and C-peptide secreted in response to glucose stimulus. All animals have become normoglycaemic off all exogenous insulin. Liver biopsies reveal strong positive staining for insulin, glucagon and somatostatin in xenografts. One recipient receiving 50,000 IEQ/kg was insulin-independent for >7 months, including 7 weeks after the last drug (belatacept) was ceased. A second recipient receiving 10,000 IEQ/kg remained insulin independent >18 months, including 6-months off all immunosuppression. The fourth and fifth animals continue to be followed past 6-months and 4-months post transplant.

CONCLUSION: We have demonstrated for the first time long-term survival and function of porcine islets in baboons. The costimulation blockade-based immunosuppression permitted maturation of the islets such that the dose required to achieve normoglycemia is equivalent to the clinical setting 10,000 IEQ/kg.

Abstract No. 109

HUMAN HLA-DR+CD27+ MEMORY-TYPE REGULATORY T CELLS SHOW POTENT XENOANTIGEN-SPECIFIC SUPPRESSION IN VITRO

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Introduction: Strategies for immunomodulation of xenograft rejection response whilst minimizing long-term immunosuppression need be developed. We have previously shown that xenoantigen-stimulation enhanced human Treg capacity to suppress the xenogeneic response. However, whether xenoantigen-expanded Treg express specific cell surface markers which can be used for separating a particular xenoantigen-specific Treg subset for an effective Treg therapy needs be identified.

Materials and Methods: Human CD4⁺CD25⁺CD127⁻ Treg isolated from healthy donor PBMC were expanded for 3 weeks with anti-CD3/CD28 beads alone or combined with irradiated porcine PBMC as polyclonally (PITreg) or xenoantigen-stimulated Treg (XnTreg), respectively. FACS was performed to determine candidate cell surface markers and consequent xenoantigen-specific Treg subset was isolated XnTreg by cell sorting. After sorting, the resulting Treg subset was assessed for their suppressive capacity by mixed lymphocyte reaction (MLR) using irradiated porcine PBMC as xenogeneic-stimulating cells, human PBMC as responder cells and autologous XnTreg as suppressing cells.

Results: After 3 weeks of expansion, XnTreg exhibited substantially upregulated expression of HLA-DR and CD27 with a larger proportion of them being HLA-DR⁺CD27⁺. The HLA-DR⁺CD27⁺ Treg subset from XnTreg demonstrated significantly enhanced potency in suppression of proliferating xenoreactive responder cells at ratios of 1:4 through to 1:64, or 1:32 and 1:64 of Treg:responder cells when compared to HLA-DR+CD27+ cell-depleted or unsorted XnTreg, respectively.

Conclusion: Our data suggest that human HLA-DR+CD27+ memory-type Treg are xenoantigen-specific and have potential as an effective immunotherapy in xenotransplantation.

Abstract No. 110

GENETIC MODIFICATION TO CONTROL THE LOCAL T CELL RESPONSE TO PIG ISLET XENOGRAFTS

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Background: T cell infiltration is a feature of rejecting pig-to-primate islet xenografts. To address this, we developed diliximab, a chimeric anti-CD2 monoclonal antibody (mAb) that depletes and inhibits human and primate T cells. Using a high-fidelity CRISPR system we generated pigs containing a diliximab transgene knocked into *GGTA1* (the gene for major xenoantigen, α Gal). The initial construct employed an MHC class I promoter for widespread transgene expression.

Aim: To generate *GGTA1* knock-in pigs with strong islet-specific expression of diliximab.

Methods: Constructs were transfected into cultured cells by nucleofection. Expression of α Gal was determined by FACS using IB4-FITC. Secretion of diliximab was detected by FACS of human T cells incubated with culture supernatant followed by anti-human IgG.

Results: The MHC-I promoter in the knock-in construct was replaced by the pig insulin promoter. The resulting construct was validated by demonstrating diliximab secretion by transfected NIT-1 insulinoma cells. It was then co-transfected with CRISPR components into wild type pig fibroblasts. 8 of 22 neomycin-resistant stable clones did not express α Gal, and showed correct transgene integration into one allele of *GGTA1*. Three clones analysed further were shown to contain a 26-bp deletion in the second allele. Eight rounds of somatic cell nuclear transfer have been performed using one of these clones, with two pregnancies to date.

Conclusion: We have commenced cloning of pigs containing an islet-specific diliximab transgene knocked into *GGTA1*. Progeny will be analysed for the distribution and level of expression of diliximab, before testing in our pig-to-primate islet xenotransplantation model.

Abstract No. 111

ENCAPSULATED PIG CELLS SECRETING ANTI-HUCD2 ANTIBODY REDUCES THE NUMBER OF HUMAN CD2 CELLS LOCALLY BUT NOT SYSTEMICALLY IN HUMANIZED MIC

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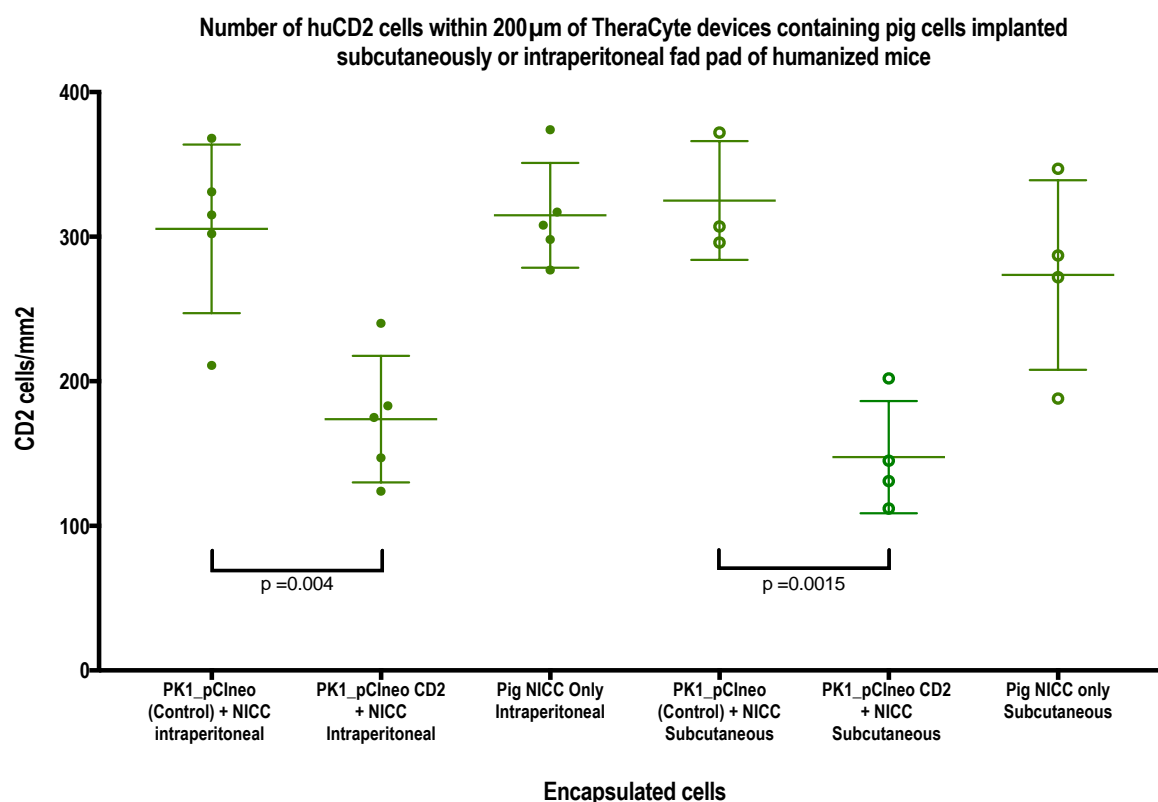
⁴Immunology & Diabetes, St Vincent's Hospital, Melbourne, ⁵Department of Immunology, Walter and Eliza Hall Institute of Medical Research, Melbourne

Background: The TheraCyte™ Implantable System, with outer membranes that induce the development of vasculature, was developed to encapsulate and protect cells that secrete insulin or other proteins the patient is deficient in. This implant system has been shown to be biocompatible and protective of allogeneic tissues in animal and human trials. However, the immune protection of xenogeneic tissues (as a potentially unlimited source of therapeutic tissue) has been so far unremarkable, as the intensity of the surrounding inflammatory response suffocates the encapsulated cells.

Method: To mollify or eliminate this local response, pig kidney cell line PK1 genetically engineered (pCIneo_CD2_GFP+) to secrete a monoclonal antibody to human CD2, which inhibits and depletes T cells. PK1 cells transfected with vector alone (pCIneo_GFP+) were used as controls. Encapsulated PK1_pCIneo_CD2_GFP+ or PK1_pCIneo_GFP+, co-encapsulated with porcine neonatal islet cell clusters (NICC's), were implanted at two sites into immunodeficient NSG mice that were then reconstituted with human PBMCs to generate a human anti-pig response.

Results: There was a statistically significant decrease in the numbers of human T cells around the devices containing anti-CD2 secreting cells, compared to those with control pig cells. This occurred at both sites, while the number of huCD2 cells in the spleen were similar in all mice. We could not determine whether protection was improved or not as all encapsulated Xeno-cells including the NICC's survived.

Conclusion: Although the xeno-protective properties of anti-CD2 could not be demonstrated, the local impact of secreted factors is a promising result, which cogently advocates further investigation.



Organ Donation and Ethics #2

Abstract No. 112

PROMOTING DECEASED DONOR ORGAN TRANSPLANTATION IN VIETNAM: WHERE TO START?

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Aim Vietnam is a developing country of 93,000,000 people with central government and wide disparities in wealth, education and healthcare. We review organ transplant activity and describe challenges for deceased organ donation (DD).

Materials and Methods National registry relies on self-reporting from 17 kidney, 3 liver and 3 heart units.

Results 2,249 living donor (LD) transplants, 174 DBD transplants and 3 DCD transplants have been reported. No child has received a deceased adult organ. Only 3 kidney centers presented data at Vietnamese Society of Transplantation (VSOT) 2017 meeting, reporting unrelated kidney LD activity of 6.3% (Figure), 71.4% and 85.7% respectively with latter two relying on police determination that unrelated donors were not rewarded. Barriers to DD exist despite DD legislation and >12,000 annual head injury deaths. Brain death diagnosis is complex. Family consent for DD is impeded by immense clinical pressures and limited resources. Requests are cursory without consensus for organ allocation. Results are not published. Wait-listing with stored sera and environment of trust between ICU and transplant surgeons do not exist. Transplant training from Europe and SE Asia is based on surgical skills for elective procedures. Careers for transplant physicians and nurses for recipient preparation and long-term care are limited.

Conclusions Potential exists to improve DD activity with simultaneous 1. Cost-effective local resourcing of ICUs; 2. Transparent allocation guidelines and waiting-list criteria led by VSOT (\pm TSANZ); and, 3. Co-ordinated international hospital partnerships. Subsequent growth of heart, liver and paediatric transplantation will enhance community and donation sector appreciation of DD.

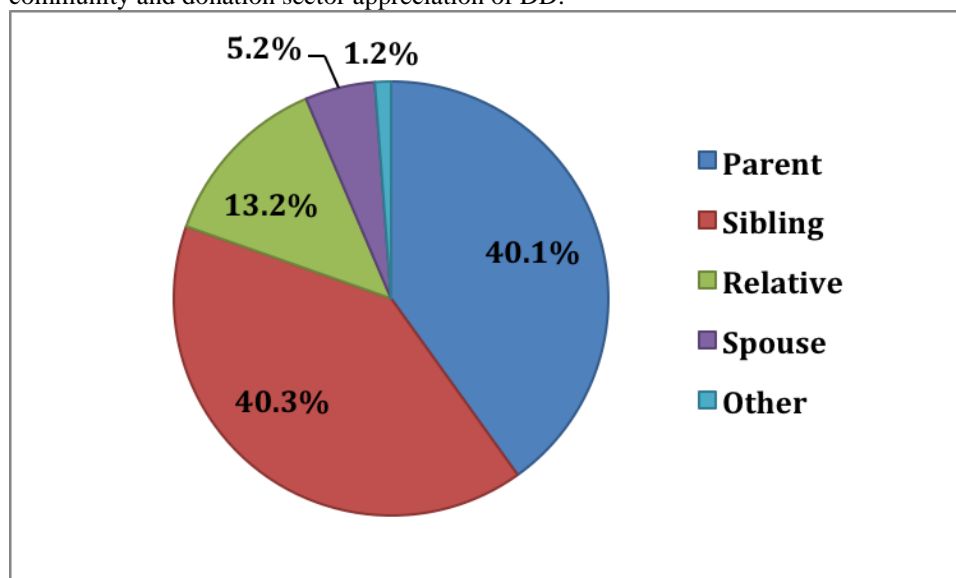


Figure: Source of living kidney donors at Cho Ray Hospital, HCMC, VN since 1992 (n=598)

Abstract No. 113

THE WEEKEND EFFECT: AN AUSTRALIAN COHORT STUDY ANALYSING TEMPORAL TRENDS IN SOLID ORGAN DONATION

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Introduction A US study suggested donation rates were poorer on weekends. We investigated the effect of day-of-week of donor referral on organ donation in Australia.

Methods In Australia, potential donor referrals are made to a state-based Donation Service, who then simultaneously seek family consent for donation and assess the medical suitability of the referral for donation. Organ retrieval occurs when utilisation is almost certain, hence discard rates are extremely low. We retrospectively reviewed all New South Wales referral logs from 2010 to 2016. Our outcomes were actual donation (retrieval), family consent, and medical suitability. We used logistic regression with random effects adjusting for clustering of referral hospitals. We used mortality data from the Australian Institute of Health and Welfare to compare donation referrals to background mortality rates by day of the week for all-cause mortality and from motor vehicle accident deaths (MVA).

Results Of 3,383 referrals (potential donors), 692 (20%) became actual donors. We found no evidence of reduced donation (adjusted OR: 1.15; 95% CI 0.93 – 1.42; p=0.2), consent (adjusted OR 1.07; 95% CI 0.85-1.35; p=0.6), or medical suitability (adjusted OR 1.15; 95% CI 0.96-1.39; p=0.1) among weekend referrals. The rate of donor-referral was lower on weekends compared to weekdays for all-cause mortality (p<0.001) and MVA mortality (p=0.03).

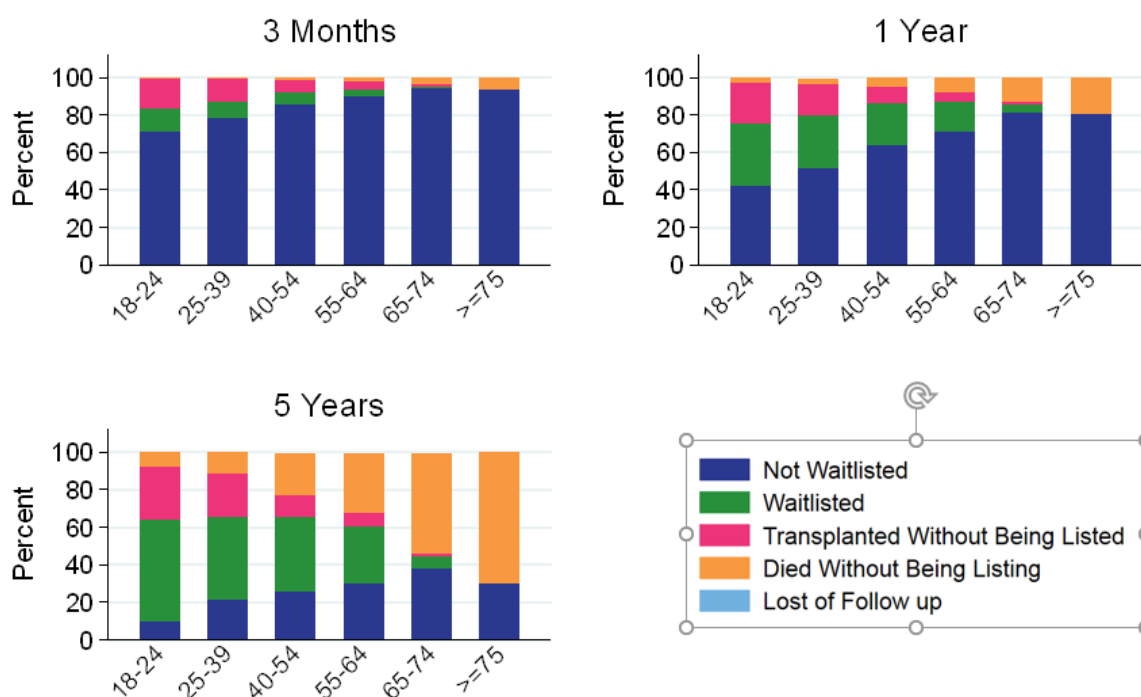
Conclusion There was no association between day-of-the-week or weekend referrals and actual donation, family consent, or medical suitability. There was some indirect evidence that donor referrals may be more selective at weekends. These results contrast findings from the USA.

Abstract No. 113 (ctd)

Table 1: Characteristics of donor referrals in New South Wales, 2010-2016

Referrals, n (%)	Population	
	Potential donors (%)	Consent sought (%)
Total	3,383 (100)	1,764 (100)
Day of the week		
Monday	520 (15)	280 (16)
Tuesday	530 (16)	275 (16)
Wednesday	507 (15)	250 (14)
Thursday	555 (16)	270 (15)
Friday	543 (16)	269 (15)
Saturday	354 (10)	200 (11)
Sunday	374 (11)	220 (12)
Period of the week		
Weekday	2,655 (78)	1,344 (76)
Weekend	728 (22)	420 (24)
Age		
0-17	158 (5)	112 (6)
18-44	618 (18)	395 (22)
45-54	546 (16)	317 (18)
55-64	703 (21)	409 (23)
65-74	740 (22)	335 (19)
75+	618 (18)	196 (11)
Sex		
Female	1,400 (41)	758 (43)
Male	1,983 (59)	1,006 (57)
Cause of death		
Cerebral Hypoxia/Ischaemia	374 (11)	220 (12)
Intracranial Haemorrhage	1,114 (33)	682 (39)
Non-Neurological Condition	1,428 (42)	555 (31)
Other neurological condition	19 (1)	6 (<1)
Trauma	448 (13)	301 (17)
Remoteness		
Regional	345 (10)	184 (10)
Major city	3,038 (90)	1,580 (90)

Abstract No. 114

FACTORS ASSOCIATED WITH TIME TO DECEASED DONOR RENAL TRANSPLANT WAITLISTING IN AUSTRALIA**SYPEK Matthew^{1,2,3}, CLAYTON Phil^{1,4,5}, LIM Wai^{6,7}, HUGHES Peter^{3,2}, KANELIS John⁸, WRIGHT Jenni⁹, CHAPMAN Jeremy¹⁰, MCDONALD Stephen^{1,11,5}**¹ANZDATA, ²Department of Nephrology, Royal Melbourne Hospital, ³Department of Medicine, University of Melbourne, ⁴Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, ⁵Adelaide Medical School, University of Adelaide, ⁶Renal Unit, Sir Charles Gairdner Hospital, Perth, ⁷School of Medicine & Pharmacology, University of Western Australia, Perth, ⁸Department of Nephrology, Monash Health and Centre for Inflammatory Diseases, Department of Medicine, Monash University, Melbourne, ⁹National Organ Matching Service, Australian Red Cross Blood Service, ¹⁰Department of Renal Medicine, Westmead Hospital, Sydney, ¹¹Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital**Background:** A detailed description of access to waitlisting for deceased donor (DD) renal transplantation in Australia has not previously been reported.**Methods:** Data from NOMS and ANZDATA were used to analyse access to DD waitlisting for adult incident renal replacement therapy patients in Australia between July 2006-July 2015. Competing risk regression time-to-event models, with transplantation before waitlisting and death as competing events, were used to determine predictors of waitlisting.**Results:** The cohort consisted of 21,290 patients with a median age of 63 years. At the completion of the follow up period 4,360 (20.5%) had been waitlisted, 1,315 (6.2%) were transplanted without being waitlisted and 7,800 (36.6%) had died without being waitlisted or transplanted. Outcomes varied widely across age groups (figure 1).**Outcome at Specified Timepoints After Commencing RRT
By Age Group**

On multivariate regression all comorbidities included were associated with a lower likelihood of waitlisting, as was older age. There were differences in access to waitlisting across states. Indigenous patients were less likely to be waitlisted (SHR 0.47 [95% CI 0.39-0.56]), as were female patients (SHR 0.88 [95% CI 0.80-0.91]), both underweight and obese patients (SHRs 0.80 [95% CI 0.67-0.96] and 0.83 [95% CI 0.77-0.90], respectively) and regional patients (SHR 0.84 [95% CI 0.78-0.91]).

Conclusion: In addition to factors related to poor post-transplant outcomes, a number of clinical, demographic and geographic factors are associated with reduced access to waitlisting for patients in Australia. Further analysis is required to determine if this reflects other, unmeasured factors or highlights a need to address differences based on age, ethnicity, location of residence or gender

Abstract No. 115

ALLOCATION OF LOW-RISK KIDNEYS: CAN WE OPTIMISE UTILISATION?**CLAYTON Phil^{1,2,3}, GULYANI Aarti^{4,2}, SYPEK Matthew^{1,5}, KANELIS John⁶, MCDONALD Stephen^{1,2,3}**¹ANZDATA, ²Adelaide Medical School, University of Adelaide, ³Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, ⁴Empty, ANZDATA, ⁵Department of Medicine, University of Melbourne, ⁶Department of Nephrology, Monash Health and Centre for Inflammatory Diseases, Department of Medicine, Monash Medical Centre, Melbourne

Aims. The Australian deceased donor kidney allocation system does not attempt to match the prognosis of kidneys and recipients, leading in some instances to low-risk kidneys (ie those with a high projected graft survival) being allocated to high-risk recipients (ie those with a limited life expectancy). We simulated restricting the allocation of such kidneys to recipients with favourable prognoses.

Methods. We constructed an event-based simulation model by adapting the US Kidney-Pancreas Simulated Allocation Model software to Australian data and allocation rules. We simulated the current allocation system over 2010-2014, and two alternative models: (1) low-risk kidneys (lowest 20% kidney donor risk index) restricted to non-high-risk recipients (lowest 80% estimated post-transplant survival score [EPTS]), (2) low-risk kidneys restricted to low-risk recipients (lowest 20% EPTS). In each model national sharing for immunological advantage was preserved. Outcomes were the distribution of low-risk kidneys and the overall projected life-years.

Results. The characteristics of recipients of low-risk kidneys were similar when comparing model 1 and the current allocation system, with the exception of waiting time (table 1). In model 2, these kidneys were allocated to younger patients with less waiting time and fewer comorbidities. Overall projected life-years increased from 80,432 life-years in the current system to 80,455 in model 1 and 80,508 in model 2.

Conclusions. Avoiding allocating low-risk kidneys to high-risk recipients is likely to make only a negligible difference; restricting their allocation to low-risk recipients would make more of difference to organ distribution but overall life-years gained would remain modest.

Table 1: Characteristics of recipients who received kidneys with KDRI in lowest 20%

	Base Model	Model 1		Model 2	
	Current Allocation Rules	KDRI ≤20%/ EPTS(AU) ≤80%	p-value	KDRI ≤20%/ EPTS(AU) ≤20%	p-value
Organs Transplanted (KDRI in lowest 20%), n	556	556		545	
Waiting time (months), median (IQR)	39.0 (19.3, 61.7)	34.2 (17.2, 57.2)	0.017	22.0 (11.8, 40.3)	<0.001
Age (years), median (IQR)	51.3 (40.4, 59.9)	50.1 (40.2, 58.0)	0.053	37.9 (29.9, 46.4)	<0.001
Gender (Female)	238 (42.8%)	224 (40.3%)	0.39	205 (37.6%)	0.079
Ethnicity			0.62		0.35
Caucasian	425 (76.4%)	407 (73.2%)		395 (72.5%)	
Indigenous	26 (4.7%)	33 (5.9%)		32 (5.9%)	
Asian	74 (13.3%)	82 (14.7%)		76 (13.9%)	
Other	31 (5.6%)	34 (6.1%)		42 (7.7%)	
Diabetes	86 (15.5%)	83 (14.9%)	0.80	61 (11.2%)	0.037
Coronary disease	87 (15.6%)	84 (15.1%)	0.80	41 (7.5%)	<0.001
Cerebrovascular disease	26 (4.7%)	28 (5.0%)	0.78	19 (3.5%)	0.32
Peripheral vascular disease	35 (6.3%)	29 (5.2%)	0.44	17 (3.1%)	0.013
Chronic lung disease	30 (5.4%)	22 (4.0%)	0.26	24 (4.4%)	0.45
Graft number			0.04		<0.001
1	463 (83.3%)	487 (87.6%)		495 (90.8%)	
2 +	93 (16.7%)	69 (12.4%)		50 (9.2%)	

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NOTICE OF ANNUAL GENERAL MEETING

The Annual General Meeting of the Transplantation Society of Australia and New Zealand held on Monday 8th May 2017 in the Boulevard Auditorium, Boulevard Level of the Brisbane Convention Centre (Grey Street entrance) commencing at 4.45pm.

Present: 50 members of the Society and 3 WA proxies (Ashley Irish) and 1 proxy (Dan Chambers) were present at the meeting which was chaired by the President, Professor Steve Chadban, who welcomed everyone to the meeting.

Also in attendance: Mrs Nieves Piaggio, Administrative Officer and Mrs Sonia Hill (temp).

1. APOLOGIES

Apologies were received from Richard Allen, Napier Thomson and Jeremy Chapman.

2. CONFIRMATION OF THE MINUTES

The minutes of the Annual General Meeting held on 10th April 2016 were passed as a true record.

Moved: David Goodman

Seconded: Ashley Irish

3. BUSINESS ARISING FROM THE MINUTES

Nil

4. PRESIDENT'S REPORT

The President, Professor Steve Chadban, said that after feedback from members, moving to Sydney had been a strategic move. Data showed that numbers were down in 2015. The plan was to stay in Sydney for two years and then Queensland for two years, then Victoria for two years and thereafter alternate along the eastern seaboard as this would be equal flying time for most.

He then went on to give a special thanks to A/Prof. Kelli MacDonald and Dr Ross Francis for organising the ASM and to A/ Prof. Dan Chambers of SPEC for his guidance. He also thanked Dr Veena Roberts, Dr Siok Tey and Dr Darren Lee for organising the weekend educational programs, all of which had received positive feedback.

He told members that the financial standing of the the Society was a good news story and was going well and improving.

Professor Steve Chadban then went on to tell members how the current Council had continued on with Prof. Geoff McCaughan's initiative for 'Ideas and Issues to be addressed' as thinking time on the council agenda to enable focus and discussion of specific ideas to be progressed or issue to be addressed. He said that this had allowed them to articulate such things as the current Mission Statement, which was available on the website, as well as other important documents such as the Ethical Guidelines, which were also posted on the website. He said that the website was due for an update and that was going to be taken up by the new Council.

He went on to state how great the current Council had functioned and how its cohesiveness had allowed, together with the funding support of OTA, such strategic projects such as: the machine perfusion workshop; a forthcoming second focus group on transplantation of indigenous people; a focus report which was underway on donor transmission risks; a planned group meeting on Paediatric challenges especially with regard to disability and access.

Next Prof. Chadban told members that the felt that Council had pushed forward with one of Josette Eris' legacies; that of gender equity. The drive behind that being Professor Karen Dwyer and Professor Shane Grey and all of the current Council's support. He informed members that he was delighted to announce that the new Council, for first time, would have 50/50 (male to female) split which would be a good move forward.

Professor Steve Chadban thanked all the Advisory Committees and RTAC, A/Prof. John Kanellis in particular. He said that he was grateful to all chairs for the hard work they do.

Members were told that TSANZ's relationship with the government was smooth and productive. He reiterated that OTA gave significant funding for project officers which in turn supported TSANZ projects and initiatives, it being a good two way relationship. He said that the key contact between the two was the Transplant Liaison and Reference Group (TLRG) where the chairs of the Advisory Committees together with the President of the Society and a couple of others are sent as representatives in discussions at OTA funded face to face meetings which were held three/four times per year. He said that this initiative had been thanks to Yael Cass and had continued with Felicity McNeil and was continuing now with Ann Smith and it was thanks to these meetings that donation and transplant rates had doubled. It was good for patients and good for TSANZ

Professor Steve Chadban then went on to thank the outgoing Council members: Professor Shane Grey, Professor Henry Pleass, Professor Greg Snell and Professor Karen Dwyer. He welcomed the new Council: Professor Steve Alexander (incoming President), Professor Patrick (Toby) Coates (President Elect), A/Prof. Kelli MacDonald, Dr Christine Russell (surgeon), Dr Natasha Rogers (Honorary Secretary) and Associate Professor Bronwyn Levvey. This left Dr Rob Carroll (Treasurer), Dr Nick Cross (NZ), A/Prof. Nick Shackel (Liaison officer with the College of Physicians) and Nigel Palk (ATCA representative) as well as the Administration staff of Sommer Twycross and Nieves Piaggio (since Aviva Rosenfeld's death).

He also paid a tribute to Prof. Paolo Ferrari for the work that he had done in heading up the paired Kidney Exchange program. He thanked Paolo on behalf of TSANZ in putting the Australian paired kidney exchange program on the world stage.

5. TREASURER'S REPORT

The Treasurer, Dr Rob Carroll, presented his Treasurer's Report on behalf of the Society. He told members that in the last audit, the Auditor had said to "be sensible" in that we had falling revenue, falling investments and falling attendance at the ASM. Therefore the budget had been tightened up by capping awards at approx. \$50,000 and the ASM at approx. \$200,000. He said that members in arrears had been chased and those who did not pay had their membership cancelled. This had brought in approx. \$40,000 in revenue and had resolved the number of active members which allowed for future budgeting.

He informed members that contrary to when Dr Hilton Gock was Treasurer, our investments were not making much money so they had been moved into 4 separate term deposits which were only paying 2 to 3% but were safe. He said that investment people had advised that the only way to maintain capital, in the current climate, were term deposits. Sponsorship was better this year at approx. \$200,000.

He referred the membership to the Financial Report of 31st December 2016 where the society was in a sound financial position with retained funds of approximately \$1.2m (not 1.5m as the OTA accounts had been separated). He said that we had more revenue due to the chasing of arrears and that in 2017 the Society was in an overall better position as numbers were up on all the meetings. He confirmed that this year the ASM had cost approx. \$203,000 (within budget of +/- 10%) and if attendance continued to grow, as it had done in Brisbane, then it might get to the point where the cost to attend the meetings would get cheaper.

Rob Carroll proposed that the 2016 Financial Report be accepted: Moved:

Steve Alexander

Seconded: Philip O'Connell Passed
unanimously.

6. SECRETARY'S REPORT

Dr Karen Dwyer, the Honorary Secretary, confirmed that the number of members was stable at 598. It was slightly down from last year due to some members being cancelled for non payment of subscription but it was picking up with 29 new members being approved prior to this meeting.

Karen Dwyer next discussed improving gender equality. She told members that TSANZ had undertaken a Workshop facilitated by Novartis in November. An EOI for this workshop had been sent by email to all members and 8 had attended. She confirmed that a summary of the processes and outcomes had been sent to members and that TSANZ were currently in the process of forming working groups to facilitate the vision of the workshop namely *prioritising equity and diversity inclusiveness in Transplantation*. She said that gender equality was also the push behind the Josette Eris memorial award which would be presented to a female member tonight. She confirmed that this was a perpetual award with a lecture to be given each year by an aspiring or established women in transplantation. This year's lecture had been given by Lori West with Lori underlining the importance of gender equity as it helps to improve science output. Karen felt that

both the award and the lecture recognised Josette Eris, the TSANZ and transplantation in general. The Josette Eris Award was funded by Astellas and it aimed at encouraging early to mid-career female members to remain competitive in their field.

She advised members that for the 1st time there had been subsidised childcare at this meeting which had been utilised by a small number of members. She felt that in the coming years, it would gain some traction as it was something important which allowed some members to attend. Also, starting in 2017, SPEC were to report to council the gender makeup of speakers and chairs in order to get a gender balance.

Karen Dwyer then directed members to the proposed constitution change (for 2018) with regard to members of Council. She said that Section 2 under Article 4 already required a certain makeup of council with regard to States and organs or tissue groups and the proposal was for this to be extended to include the wording “at least 3 men and 3 women” in Section 2(b)(vi).

The final initiative was for TSANZ to embrace technology and therefore the idea of forming a digital/technology working group. EOI’s would be sent out to membership. Council as a whole felt that this was a very important step moving forward as too was improving our website and enhancing TSANZ’s social medial presence. She informed members that an informal social media task force had been set up for this meeting and it had had quite a significant reach.

7. REPORT ON ADVISORY COMMITTEES/WORKING GROUPS

Steve Alexander, the chair of the Advisory Committees & Working Groups gave a brief report to the membership. He advised that the Advisory Committees and Working Groups were working well and that the heads had done an amazing job. He felt that there was ample consultation and this had allowed for initiative and resolutions to be proposed across the country. He thanked Iman Ali for the great work he had done. He also reflected that TLRG was working very well. It allowed the bringing together of the chairs of the working groups so that they could throw ideas and initiatives around that overall improved patient care. He especially thanked Professor Steve Chadban for the huge amount of work he had undertaken as President of TSANZ.

8. SCIENTIFIC PROGRAM & EDUCATION COMMITTEE REPORT (SPEC)

A/Professor Daniel Chambers, Co-Chair of SPEC, told the membership that the change of venue from Canberra and the structure of the ASM were bearing fruit. He thanked A/Prof. Kelli MacDonald and Dr Ross Francis and all of the members for their support. Next Daniel said that the 2017 ASM had run reasonably smooth considering all the changes. He confirmed that the 2018 ASM would be held in Melbourne with Dr Lucy Sullivan and Dr Rosemary Masterson working on the program. He went on to thank Dr Siok Tey, Dr Darren Lee and Dr Veena Roberts for the great educational program held over the weekend which was very well attended. The ASM would then move back to Sydney in 2019 and Adelaide in 2020 as the change of venues was proving successful with an increase in numbers. Moving forward, the one thing that SPEC would change

was the criteria for the awards with a draft review to be sent to Council shortly. He encouraged members to provide formal feedback

9. LIAISON WITH SCIENTIFIC SOCIETIES

No discussion.

10. GENERAL BUSINESS

Professor Steve Chadban started the general business by speaking about Aviva Rosenfeld. He said that Professor Peter Macdonald had given a brilliant tribute talk for Aviva at the start of ASM but asked whether the membership felt that something more permanent and ongoing was needed to celebrate her memory. Professor Geoff McCaughan suggested an award which would include service to the Society as the determinate. Professor Phil O'Connell suggested that a small committee within Council take suggestions rather than giving out countless awards. Professor Frank Ierino suggested a portrait or painting with Professor Bruce Hall requesting that the family have some input as to what type of award. He also felt that a copy of Peter Macdonald's tribute be sent to the husband and documented in these minutes. Steve Chadban said that he would channel these thoughts to the new Council for proper consideration.

Noted in budget was the travel awards. Members in general felt that it was a great initiative to support young people especially in helping them to get to overseas meetings. It was suggested that the amount going to young people be minuted. Rob Carroll advised that travel awards were a big factor and they would continue.

Peter Macdonald requested that since Council were looking at making a change to the constitution, he would like the new council to consider separate representation on Council for heart and lung. Steve Chadban said he would pass that thought over to the next Council and wished the new council the very best.

There being no further business the meeting closed at 5.40pm.

Steve Chadban

Karen Dwyer

President

Honorary Secretary



T · S · A · N · Z

The Transplantation
Society of Australia and
New Zealand Inc.

ABN 90 796 930 798

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(President)

Patrick Coates
(Vice President)

Natasha Rogers
(Secretary)

Rob Carroll
(Treasurer)

COUNCILLORS

Nick Cross
Kelli MacDonald
Christine Russell
Nicholas Shackel
Bronwyn Levvey
Nigel Palk
(ATCA Representative)

PAST PRESIDENTS

AG Ross Sheil
James Biggs
Anthony JF d'Apice
Kerry Atkinson
Ian FC McKenzie
Tom Mandel
Bruce Hall
Jeremy Chapman
Mauro Sandrin
Stephen Lynch
Randall Faull
Philip O'Connell
Josette Eris
Frank Ierino
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Geoff McCaughan
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THE PRESIDENT'S REPORT

It is a great pleasure to write about the achievements of many of the members of the society in working to improve outcomes for patients and their families with organ transplants. Across many areas of clinical delivery, allocation, equity, training, research and education many of our members through Council, SPEC, the ASM, the Masterclass and PGC course have worked to improve care for patients throughout Australia and New Zealand.

This year marks the third year since we began taking the ASM on the road to Sydney then Brisbane and now to Melbourne. The ASM organised by Lucy Sullivan and Rosemary Masterson looks to be fantastic. It has an impressive range of international speakers covering crucial areas of tolerance and antibody mediated rejection and fundamental immunology with speakers from Toronto, California and Boston. It also includes Carmel Hawley, chair of the AKTN, bringing a strong translational focus to our efforts.

SPEC has been exceptionally ably led by Kelli MacDonald and Dan Chambers along with the work on the Masterclass organised by Darren Lee with assistance from Veena Roberts and the PGC organised by John Whitlam and Rhonda Holdsworth, providing tremendous education for clinicians, surgeons, scientists, trainees and allied professionals.

The Council contains a diverse range of talents, and has followed on many of the initiatives of Steve Chadban and the previous council in areas of infectious risk, equity and allocation. Toby Coates has brought a range of skill, enthusiasm and advice to the Council as President-elect. Rob Carroll has excelled in controlling the society's purse strings, Natasha Rogers as Secretary has kept the Council on track. We have also been exceptionally lucky to have Nick Shackel who has done great work on ethics and the new ethics subcommittee, Chris Russell has added a great surgical perspective, and Bronwyn Levvey has increased our discussions of nurse practitioners in transplantation and areas of collaboration with allied health as well as the need for separate cardiac and thoracic representation. Nick Cross keeps the Australian side of TSANZ honest as the NZ representative and has been leading efforts to promote donation in NZ, Kelli MacDonald as the scientific representative and co-chair of SPEC has enhanced our scientific content and improved communication with SPEC and with associated scientific organisations such as ASI. Nigel Palk representing ATCA has been a major lead in issues of state based allocations and organisation. As a group the issue of equity in areas of gender, profession and access to transplantation to all groups have been major pieces of work.

Given the strong SA representation on Council, Council was able to meet in Adelaide this year and had the chance to visit the new hospital and research precinct which were extremely impressive and where there is a strong transplant presence and where we were able to meet with Graeme Russ, Randall Faull, Steve McDonald heading ANZDATA, and Shilpa Jesudason the clinical director of KHA.

All of council's work has been supported by Sommer Twycross and Nieves Piaggio who have done a tremendous job; and also Kim Rawson who has recently joined as the TSANZ Project Officer replacing Iman Ali who has recently retired and who we are extremely grateful to for his support of the Advisory Committees.

There have been many initiatives that have occurred through collaboration with the Organ and Tissue Authority where we are extremely sad to see the upcoming retirement of Eva Mehakovic and wish her well and thank her for her many contributions to transplantation. These include Sara White's work on infectious risk in transplantation, Josh Kausman's review of paediatric donors, support for the work of RTAC and the co-ordination through TLRG of many initiatives to enhance transplantation as well as support for the paired kidney exchange. TLRG is headed by our ex-President Steve Chadban and has provided a venue for many of the stakeholders in transplantation to meet and work on improvements in many aspects of organ transplantation in co-operation with the OTA.

RTAC lead by John Kanellis has seen the introduction of better allocation software and the introduction of KDPI to our lexicon. The improvements in paediatric allocation have already been implemented with great success.

We continue to have a close partnership with TTS and with Jeremy Chapman's editorship of their journal *Transplantation* have seen the involvement of many TSANZ members, as with Toby Coates' transplant editorship at *Kidney International*.

In the area of research there have been many exciting advances in both clinical and basic research with a range of projects across Australia and New Zealand developing many basic findings into translational clinical studies and work in areas such as outcomes led by Allison Tong improving our understanding of what patient's concerns and priorities are. This particular area has led to the implementation of the Aviva award to recognise transplant professional's nominated by patients for going the extra mile. This recognises Aviva Rosenfeld who exemplified this area of caring in transplantation and will be awarded at the annual dinner. Of note are our many young researchers across an enormous breadth of topics many of whom will be presenting in the President's prize at the ASM. Of note many of our researchers are heading to Madrid for the TTS meeting and we wish them well.

We continue to have strong support from our platinum sponsors with several new initiatives. We are very grateful to Novartis for their help with putting lectures on-line and with developing a mentoring and networking workshop, and Astellas for their support of the ANZ Liver meeting, chaired by Geoff McCaughan and Richard Allen's efforts in leading the VSOT congress in Vietnam and his great achievements there in 2017.

Toby Coates and Kim Rawson have worked closely with the Advisory Committees to update guidelines and deal with issues of specific interest to the groups. Paediatrics is moving from a working group to an advisory group.

In indigenous transplantation, Steve Chadban and Steve McDonald have assembled a group of stakeholders to work with OTA and the Health Minister to advance priorities in

indigenous transplantation and are looking at developing an indigenous advisory group to drive this.

We continue work with a variety of groups including ANZSN, Beat CKD, ANZDATA, KHA and patient groups including Transplant Australia to represent patients and the transplant community and improve outcomes for our patients.

We note with sadness the passing of colleagues and friends Derek Hart and Terry Strom but remember their tremendous achievements in the area of transplantation.

We also look forward to 2019 when Natasha Rogers and Bill Mulley present the next ASM in Sydney end of July.

Best wishes to everyone for the 2018 ASM and good luck to all those receiving organ transplants and those caring for them!

Prof. Steve Alexander
President, TSANZ

TSANZ Membership List

AUSTRALIAN CAPITAL TERRITORY

CARNEY, G
CHOONG, F J
*CUNNINGHAM, J
FALK, M
KWAN, T
*NORTHAM, H L
*RUSSELL, T
SIMEONOVIC, C
*WELLS (NEE BROWN), E

NEW SOUTH WALES

ADHIKARY, S R
AFSHAR, S
ALEXANDER, S I
ALLEN, R D M
ALLEN, P
ANDERSON, P F
AOUAD, L
ARAVINDAN, A N
AU, E H K
#BORIC, R
BURNS, H
#BURTON, M
CALISA, V
CAO, J
CAO, L
*CELCER, J
CHADBAN, S J
CHAN, E
CHEN, T
CHEW, Y V
CHEW, H C
CHONG, C H Y
COLLETT, J P
CONWAY, J
COOPER, B A
COULSHED, S J
#CRAIG, E
CRAWFORD, M D
*DATSON, L
*DAVIDSON, D
DE LA MATA, N L
DHITAL, K
DIDSbury, M

DIEP, J
DURKAN, A
EL-RASHID
M ERLICH, J H
EVANGELIDIS, N M
FAZEKAS DE ST GROTH, B
FERNANDO, M R
*FRITIS-LAMORA, R
GALLAGHER, M P
GALLAGHER, A
GAO, L
GEORGE, C R P
GERAGHTY, N
GHORAISHI, T
GILLIES, A H B
GLANVILLE, A R
GRACEY, D
GRAHAM, A R
GRANGER, E K
GREY, S T
#HABIJANEC (NEE BELMAR), B
HAGHIGHI, K
HAHN, D
HALOOB, I
HAMEED, A M
HANCOCK, R
HANSON, C
HARKESS, M
HAVRYK, A P
HAWTHORNE, W J
HEER, M K
HERON, J E
HIBBERD, A D
HICKS, M
HODGKINSON, S
HOWELL, M
HU, M
HUANG, D
IMRAN, M
IYER, A
JABBOUR, A
JAMES (NEE RYDING), L J
JAMESON, C
JAMIESON, N
JARDINE, M
JAYASINGHE, K
JHA, S
JIMENEZ-VERA, E

JU, X
KABLE, K
KELLY, J J
KELLY, P
KENNEDY, S E
KEOGH, A M
KEUNG, K KIM, S
KUBITSKIY, A
KWAN, J C
*LACEY, J
LAI, C
LAM, V
LAN, P
LE PAGE, A K
LEE, A
LEONG, M
LEWIS, D
LIN, R
LIUWANTARA, D
LU, D B
LUXTON, G
MA, S W H
MACDONALD, P S
MACKIE, J D
MACKIE, F E
MAHONY, J F
MAJUMDAR, A
MALOUF, M
MANERA, K E
#MAWSON, J
MAY, S J
MAZID, S
MCCAUGHAN, G W
MCGINN, S
MCKENZIE, J M
MEARS, D C
MELICK, G K
MITCHELL, A B
MOAWADH, M
*MONTGOMERY, E
#MUNRO, C
MURUGASU, R
MUTHIAH, K
NANKIVELL, B J
NARESH, C N
NATFAJI, A
O'CONNELL, P J
PAUL, M
#PAUL, P

PHOON, R K S
 PLEASS, H C C
 POLLOCK, C A
 PRAKASH, M P
 PULITANO, C
 PUSSELL, B A
 QIAN, Y W
 RALPH, A
 RAMAN, A
 RAWLINSON, W D
 REIMANN, F
 RICHARDS (NEE
 MACKAY), K
 RITCHIE, A
 ROBERTSON, M R
 *ROBERTSON, P R
 ROBINSON, C
 ROGERS, N
 ROSALES, B M
 *ROSS, C M
 ROXBURGH, S
 RUTHERFORD, D
 SANDERY, B J
 SCHEUER, S
 *SEIFERT, N
 SEN, S
 #SGORBINI, M
 SHACKEL, N A
 SHAHRESTANI, S
 SHARLAND, A F
 SHARMA, A
 SHEN, Y Y H
 SHINGDE, R
 SHUN, A
 SILVEIRA, P A
 SIMPSON, A M
 SINGER, J
 SKALICKY, D
 *SMITH, A
 SPICER, T
 SPROTT, P
 *STEIN, A M
 STORMON, M O
 STRASSER, S I
 SUD, K
 #TAGUE, G
 TALAEI ZANJANI, N
 TAN, Y
 TANG, J
 TANGIRALA, N
 THOMSON, I K
 TONG, A
 TREVILLIAN, P R
 TUCH, B E

#UTSIWEGOTA, M
 VAJDIC, C M
 VAUGHAN, E
 VERMA, N
 VERRAN, D J
 VILLANUEVA, J
 VOLOVETS, A
 WALLER, K
 WALTERS, S N
 WAN, S
 WANG, C
 #WARE, J
 WATSON, D
 WEBSTER, A C

 WILCOX, P L
 WILLIAMS, L J
 WONG, G
 WONG, M G
 WONG, N L
 WOODHOUSE, E
 WRIGHT, J
 WU, H
 WYBURN, K
 WYLD, M
 YI, S
 YIN, J L
 YING, T
 YONG, K
 ZAHOROWSKA, B
 ZAMMIT, N
 ZHANG, G Y
 ZHAO, Y

NORTHERN TERRITORY

CASS, A HUGHES, J
 LAWTON, P D

QUEENSLAND

ABBOTT, W J
 ALEXANDER, K
 BALDERSON, G A
 #BETTENS, P
 BROOKS, E
 #BRUCE, L A
 BURKE, M
 BYRNE, S J
 CAMPBELL, S B
 CAREY, J M
 CATERSON, R J

CHAMBERS, D
 CHAN, S
 CLARK, C
 *COCO, T
 DIVITHOTAWELA, C
 #EGAN, F
 #ELDRIDGE, A N
 FAWCETT, J
 FIENE, A
 FRANCIS, A
 FRANCIS, R
 GARTLAN, K H
 GRIFFIN, A D
 HARDIE, I R
 HAWLEY, C M
 HENDEN, A
 HICKLING, D
 HILL, G R

#HILTON, L
 HOLLETT, P
 HOPKINS, P
 ISBEL, N M
 #JARRETT, M
 JAVORSKY, G
 KANAGARAJAH, V
 KOYAMA, M
 LAURENCE, J
 LAWSON, M
 LE TEXIER, L
 #LEISFIELD, P M
 LINEBURG, K
 #LIPKA, G
 LOCKWOOD, D
 LYNCH, S V
 MACDONALD, K P A
 MACDONALD, G A
 MALLETT, A
 MARKEY, K A
 MARTINS, P
 MCTAGGART, S J
 MINNIE, S A
 MON, S Y
 MUDGE, D
 #NATAKUAPA, J A
 #PORRA, M
 PRESTON, J M
 REILING, J
 RHEE, H
 #RIXON, S E
 *ROURKE, F
 SENEVIRATNE, I S

SINCLAIR, K A
SINNYA, S
SLADDEN, T M
STAATZ, C E
STEPTOE, R J
STUART, K
TALLIS, C
TEY, S
VAN EPS, C L
VARELIAS, A
WILKINSON, A N
WONG, J S C

SOUTH AUSTRALIA

BARBARA, J A J
BROOKE-SMITH,
M E CARROLL, R P
CLAYTON, P A
COATES, P T H
DOLAN, P M
DROGEMULLER, C J
FAULL, R J
#HODAK, A
HOLMES, M
HOPE, C
IRISH, G L
JESSUP, C F
JESUDASON, S
*JOHN, E V
*JONES, P
JUNEJA, R
KANG, K (DANNY)
KAPOJOS, J
KETTE, F
KIM, J
KRIGE, A J
LADHANI, M
LETT, B
MCDONALD, S P
MILLS, R A
MOHAN RAO, M MULLER,
K
NEO, F L
PALK, N
PASSARIS, G
PAVATHUPARAMBIL
ABDUL MANAPH, N
PERKINS, G B
*PUMPA, E
RADFORD, T M
RAO, N
ROJAS-CANALES, D

ROSE, P
RUSS, G R
RUSSELL, C H
SIVANATHAN, K
STEAD, S
SYPEK, M P
TAN, B Q
WEIGHTMAN, A
*WILLIAMS, N R

TASMANIA

GAN, J S
GRAVER, A S
JOSE, M D
KIRKLAND, G
KUO, S

VICTORIA

ANGUS, P W
ATKINSON, A R
BARRACLOUGH, N
BARRACLOUGH, K
BATEMAN, S
BERGIN, P
BLAIR, S L
BONGONI, A K
BOURNE, B J
BROWN, M
BROWN, F
*BRYEN, E L
CANTWELL, L S
CARRINGTON, E
CHIA, J S J
CHONG, J
CHOW, F Y F
CHOW, K
CHOY, S
#CHRISTIANO, Y
COHNEY, S
COUGHLAN, T
COWAN, P J
CROSTHWAITE, A
DAVE, V
#DEVINE, C M
*DUNCAN, C
DWYER, K M
*DWYER, B M
#ELLIS, C
FANG, D
FINK, M

FISICARO, N
FURLONG, T
#GARDNER-DIXON, P
GOCK, H
GOH, S K
GOODMAN, D J
GOPAL, B
GOW, P J
GRYNBERG, K
HARDIKAR, W
*HENRIKSEN, A J
*HERD, L
*HOBSON, J
HOLDSWORTH, R F
HOLMES (NEE BEYERLE),
K M
HUGHES, P
*HUGHES, T
IDEL, T
IERINO, F L
#IRELAND, K
JAW, J
JONES, R M
KAMESHWAR, K
KANELLIS, J
KATSOULIS, J
KAUSMAN, J
KAY, T W H
#KENNEDY, E
KERR, P G
KOS, C
KOTECHA, S
KWONG, M
LANTERI, M B
LEE, D
LEONG, K G
LEUNG, P Y M
LEVVEY, B
LEW, A
LIAN, M
LOUDOVARIS, T
#LUSCOMBE, B
MANZOOR, M
MARIANA, L
MARTIN, D
MASTERSON, R
MCGIFFIN, D C
MCRAE, J
MENAHEM, S
METZ, D
MIACH, P J
MICHELL, I D
*MINIO, G
MULLEY, W R

MURPHY, S S
 NGU, K
 NICHOLLS, K M
 PAIZIS, K
 PANNU, K
 PARASKEVA,
 M PAVLOVIC, J
 PERINI, M
 PIMENTEL, A L
 RAMESSUR, S
 *REA, A
 ROBERTS, V
 #ROBERTS, C M
 ROBERTSON, A J
 ROZENKOV, V
 RYAN, J
 SALVARIS, E
 SANDRIN, M S
 SAUNDER, A
 SEIBT, B
 *SHIPP, A
 SKEAT, L
 SNELL, G I
 SOMERVILLE, C A
 STARKEY, G D
 STEINBERG, A G
 STEVEN, M
 SUH, N
 SULLIVAN, L C
 TA, J
 TAIT, B D
 TAN, S J
 TESTRO, A
 THOMAS, H
 THOMSON, N M
 TRAPANI, J A
 #VAGO, A
 WALKER, A M
 WALKER, R G
 WANG, B Z
 WESTALL, G
 WHITLAM, J
 WILSON, S
 #YIP, D
 YONG, B
WESTERN AUSTRALIA

ADAMS, L
 ASSUNE, G
 DELRIVIERE, L D D
 DEMBO, L
 DO NGUYEN, H
 #FAZACKERLEY, C A
 FIDLER, S

#GALLIZZI, S D
 GARAS, G
 GODDARD, K A
 HE, B
 HOWSON, P E
 IRISH, A B
 JAMBOTI, J S
 #JARY, C
 JAYARAMAN, V K
 LARBALESTIER, R
 LARKINS, N G
 LAVENDER, M
 #LAWRENCE, S
 LIM, W H
 LUCAS, M
 MANICKAVASAGAR, R
 MOU, L
 MUSK, M
 NG, Z Q
 PERRY, G
 PROSSER, A
 PUTRINO (NEE CHUA), S
 PUTTAGUNTA, H
 *SCHUAMANN, M A
 *SMITH, M S
 SORARU, J
 SWAMINATHAN, R
 VIECELLI, A
 #WARGER, A
 YEO, R

NEW ZEALAND

ALLAWATI,
 H BARTLETT, A
 CHATTERJEE, A
 CLARK, C
 COLLINS, M
 CROSS, N
 DITTMER, I D
 DONNELLAN, S
 DOWNING, J
 DUNCKLEY, H
 EVANS, H
 HECKER, W
 IRVINE, J H
 JOHNSTON, M J
 KARA, T
 *LANGLANDS,
 J LEIKIS, M J
 #LOWE, K E
 MANLEY, P
 MCNALLY, A

MCWILLIAMS, T
 MUNN, S R
 MUTHU, C
 PILMORE, H L
 PRESTIDGE, C
 REYNOLDS, A
 ROAKE, J A
 RUYGROK, P N
 HETTIGAR, R P
 *SPRENGER, L
 WALKER, R J
 WASYWICH, C A
 YASUTOMI, M

OVERSEAS MEMBERS

ALTAMIMI, A
 BOND, G J
 ERICZON, B G
 FERRARI, P
 HANCOCK, W W
 ISON, M
 KLEEMANN, F
 KOULMANDA, M
 MAINRA, R
 MARUI, Y
 MATSUNAMI, H
 NEIL, D A H
 TAYLOR, C

HONORARY LIFE MEMBERS

BIGGS, J C
 BISHOP, G A
 CHAPMAN OAM, J R
 D'APICE, A
 HALL, B M
 MARSHALL, V C
 MATHEW, T H
 MCKENZIE, I F C
 MILLER, J F A P
 MONACO, A
 MORRIS AC, P J
 NOSSAL, G J V
 SHEIL, A G R
 WILLIAMS, K

**AFFILIATE
ORGANISATIONS**

Australian Transplant
Coordinators Association
(ATCA)

Kidney Health Australia
(KHA)

Royal Australasian College of
Physicians (RACP)

Transplant Australia

Transplant Nurses Association
(TNA)

The Transplantation Society
(TTS)

