TABLE OF CONTENTS

PROGRAM AT A GLANCE 2
OFFICE BEARERS OF THE TRANSPLANTATION SOCIETY OF AUSTRALIA AND NEW ZEALAND 5
SPONSORS 6
AWARDS 8
INVITED INTERNATIONAL SPEAKERS 9
INVITED SPEAKERS 10
ABSTRACT REVIEW PROCESS AND PRESENTATION FORMATS 12
PROGRAM 13
2019 ASM ACCEPTED ABSTRACTS 38
AUTHOR INDEX 148
NOTICE OF ANNUAL GENERAL MEETING 153
PRESIDENT'S MESSAGE 156
TSANZ Membership List 158

The Transplantation Society of Australia and New Zealand

Thirty-seventh Annual Scientific Meeting

PROGRAM AT A GLANCE

Saturday, 27 July 2019		
14:00–15:00	Cardiac Transplant Advisory Committee (CTAC)	Sutherland Room (back partition)
Sunday, 28 July 2019		
10:00–12:00	National review of Paediatric kidney transplant recipients - Work Shop	Room 210, Social Sciences Building
13:00–14:00	Pancreas and Islet Transplant Advisory Committee (PITAC)	Chancellors Room
13:30–14:30	Paediatric Transplant Advisory Committee (PTAC)	Sutherland Room (back partition)
14:00–15:00	Registration	Bevery
15:00–15:10	**Official Opening:** *TSANZ President*	Refectory
15:10–15:40	**PLENARY 1: Astellas Symposium** Detecting and Managing the At-Risk Graft	Refectory
15:45–16:45	***CONCURRENT FREE COMMUNICATIONS SESSIONS*** **Free Communications 1:** Surgical Techniques **Free Communications 2:** Outcome Measures **Free Communications 3:** T-Regulatory cells	 Refectory Sutherland Room (front) Cullen Room
16:30–17:00	Vascular Composite Allograft Advisory Committee (VCAAC)	Sutherland Room (back partition)
16:45–17:00	**Afternoon tea**	Bevery and Drawing Room
17:00–18:00	**Novartis Josette Eris Lecture and Ian McKenzie Award** Navigating the Waters of Science With Dendritic Cells and Neuropeptides	Refectory
18:00–19:00	**Welcome Reception and Poster Viewing**	Drawing Room

Monday, 29 July 2019		
06:15–07:15	**Fun Run/Walk (5km) Sponsor: Transplant Australia**	Circular Quay to Hyde Park
07:30–08:00	**Breakfast** with sponsors	Bevery
08:00–09:40	**PLENARY 2: Joint TSANZ /OTA/ATCA Session**	Refectory
09:40–10:40	***CONCURRENT FREE COMMUNICATIONS SESSIONS***	
	Free Communications 4: Organ Donation and Preservation	Refectory
	Free Communications 5: Antibodies	Sutherland Room (front)
	Free Communications 6: Tolerance	Cullen Room
09:50–10:50	Lung Transplant Advisory Committee (LTAC)	Chancellors Room
10:40–11:10	**Morning tea and Poster viewing**	Bevery and Drawing Room
	Xeno-Transplantation Working Group (XTWG)	Sutherland Room (back partition)
11:10–12:50	**PLENARY 3: Astellas Symposium**	Refectory
	Complications and Rejection - Prevention and Prediction	
12:50–13:35	**Lunch and Poster Viewing**	Bevery and Drawing Room
	Donor Surgeons Donor Coordinators Advisory Committee (DSDC)	Sutherland Room (back partition)
13:35–15:35	**President's Prize Symposium**	Refectory
15:35–16:00	**Afternoon tea**	Bevery and Drawing Room
16:00–17:00	***CONCURRENT FREE COMMUNICATIONS SESSIONS***	
	Free Communications 7: Transplant Outcomes and Complications	Refectory
	Free Communications 8: Liver, Islet and Pancreas Transplantation	Sutherland Room (front)
	Free Communications 9: IRI, Immunobiology and GvHD	Cullen Room
17:00–18:00	**TSANZ Annual General Meeting**	Refectory
19:00–23:00	**TSANZ Annual Dinner**	Great Hall, Sydney University

Tuesday, 30 July 2019		
07:30–08:00	**Coffee** with sponsors	Bevery
08:00–09:30	**Plenary 4: Novartis Symposium** Manipulating the Immune Response	Refectory
09:30–10:30	***CONCURRENT STATE OF THE ART SESSIONS***	
	STATE OF THE ART 1: Astellas Symposium Infections	Refectory
	STATE OF THE ART 2: Novartis Symposium Surgical Session	Sutherland Room (front)
10:30–11:00	**Morning tea**	Bevery and Drawing Room
11:00–12:30	**CONCURRENT STATE OF THE ART SESSIONS**	
	STATE OF THE ART 3: Astellas Symposium Trials and Allocation	Refectory
	STATE OF THE ART 4: Novartis Symposium Equity in Transplantation	Sutherland Room (front)
12:30–13:30	**Lunch**	Bevery and Drawing Room
	Immune Tolerance Advisory Committee (ITAC)	Chancellors Room
	Paediatric Donor Working Group (PDWG)	Sutherland Room (back partition)
12:30–14:30	Renal Transplant Advisory Committee (RTAC)	Cullen Room
13:30–15:00	**Plenary 5: Novartis Symposium** Different Perspectives on Determining Immune Responses	Refectory
15:00–15:25	Afternoon tea	Bevery and Drawing Room
15:25–16:00	**The Great Debate: 'Transplant Clinicians Focus too Much on Rejection and not Enough on Infection'**	Refectory
16:00	**ASM Concludes**	
16:00–18:30	Liver and Intestinal Transplant Advisory Committee (LITAC)	Sutherland Room (back partition)

OFFICE BEARERS OF THE TRANSPLANTATION SOCIETY OF AUSTRALIA AND NEW ZEALAND

President
Professor Stephen Alexander

President Elect & Chair, Advisory Committees/Working Groups
Professor Patrick (Toby) Coates

Honorary Secretary
A/Professor 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 Kelli MacDonald (Co-Chair)
Prof Henry Pleass	Prof Wayne Hawthorne
A/Prof William Mulley (ASM)	A/Prof Andrew Jabbour
Dr Darren Lee	Prof Allison Tong
A/Prof Natasha Rogers (ASM)	Dr Karen Keung (PGC)
Dr Michael Collins (PGC)	Prof Karen Dwyer (Masterclass)
Dr Leyla Aouad (Masterclass)	

TSANZ Administrative Staff
Mrs Nieves Piaggio
Executive Officer
Email: tsanz@tsanz.com.au

Ms Katie Graham
Administrative Officer
Email: admin@tsanz.com.au

Program and Abstract Book
Ms Marina Katerelos
Email: abstracts.tsanz.asm@gmail.com

SPONSORS

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

Platinum Sponsors

astellas | TRANSPLANT
ADVANCING TRANSPLANTATION TOGETHER

NOVARTIS

Gold Sponsor

XVIVO PERFUSION

Silver Sponsor

Australian Government
Organ and Tissue Authority

donatelife

Bronze Sponsors/ Exhibitors

Award Sponsors

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)

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

Josette Eris Award
(supported by Astellas)

Kidney Health Australia Awards

Mark Cocks Transplantation Research Scholarship
(supported by Transplant Australia)

Ian McKenzie Award
(supported by TSANZ)

Aviva Rosenfeld Award for Excellence in Patient Care in Transplantation
(supported by TSANZ)

FINANCIAL STATEMENTS

The Transplantation Society of Australia and New Zealand (TSANZ) Financials for the Year Ended December 2018 are available on the easily accessible member password protected section of the TSANZ website www.tsanz.com.au.

INVITED INTERNATIONAL SPEAKERS

Astellas Lecturer
Professor Mark Stegall MD
James C. Masson Professor of Surgery Research
Mayo Clinic College of Medicine
Mayo Clinic
Rochester, USA

Novartis Lecturer
Professor Adriana Larregina, MD, PhD
Professor of Dermatology and Immunology
Faculty, The McGowan Institute for Regenerative Medicine
University of Pittsburgh

Astellas Lecturer
Professor Deepali Kumar, MD, MSc, FRCP(C)
Professor of Medicine, University of Toronto
Transplant Infectious Diseases & Multi Organ Transplant Program
University Health Network
Toronto, Canada

Novartis Lecturer
Professor Adrian Morelli, MD, PhD
Professor of Surgery and Immunology
Starzl Transplant Institute
University of Pittsburgh

INVITED SPEAKERS

Professor Stephen Alexander
Nephrology Department, Children's Hospital at Westmead, NSW

Ms Lucinda Barry
Organ and Tissue Authority, Canberra, ACT

A/Professor Patrick Bertolino
Liver Immunology Group, Centenary Institute, NSW

Dr Tatyana Chtanova
Garvan Institute of Medical Research, NSW

Dr Carolyn Clark
Nephrology Department, Wellington Hospital, NZ

Professor Jonathan Craig
College of Medicine and Public Health, SA

Mr Luke Datson
NSW Organ and Tissue Donation Service, NSW

Dr Claire Dendle
Monash Health and Monash University, VIC

Ms Mary Diviney
Victorian Transplantation & Immunogenetics Service, VIC

Dr Sharon Ford
Nephrology Department, St Vincent's Hospital, VIC

Professor David Gottlieb
University of Sydney and Westmead Hospital NSW

Dr Elizabeth Hinde
University of Melbourne, VIC

Ms Rhonda Holdsworth
Australian Red Cross Blood Service, VIC

Dr Paul Lawton
Menzies School of Health Research and Nephrology Department, Royal Darwin Hospital, NT

INVITED SPEAKERS

A/Professor Jerome Laurence
RPA and University of Sydney, NSW

Dr Darren Lee
Department of Nephrology, Eastern Health and Austin Health, VIC

Professor Peter MacDonald
St Vincent's Hospital and Victor Chang Cardiac Research Institute, NSW

Dr William Majoni
Nephrology Department, Royal Darwin Hospital, NT

Professor Pablo-Peñas
Department of Dermatology, Westmead Hospital, NSW

Professor Henry Pleass
Westmead Clinical School, NSW

Professor William Rawlinson
Serology, Virology and OTDS Laboratories (SAViD), NSW Health Pathology and University of NSW, NSW

Ms Nicki Scholes-Robertson
Centre for Kidney Research and School of Public Health, University of Sydney, NSW

Dr Nancy Suh
Monash Health and Royal Melbourne Hospital, VIC

Professor Ranjeny Thomas
Arthritis Queensland and The University of Queensland, Diamantina Institute, QLD

Professor Allison Tong
University of Sydney School of Public Health and Centre for Kidney Research, The Children's Hospital at Westmead, NSW

Dr John Whitlam
Department of Nephrology, Austin Health, VIC

A/Professor Germaine Wong
University of Sydney School of Public Health and Nephrology Department, Westmead Hospital, NSW

A/Professor Kate Wyburn
Nephrology Department, Royal Prince Alfred Hospital, NSW

ABSTRACT REVIEW PROCESS AND PRESENTATION FORMATS

A total of 122 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 2019 ASM. Free Communications session will have 5 oral presentations for Clinical abstracts (10 min presentation, 2 min questions) and 4 oral Presentations for Scientific abstracts (12 min presentation, 3 min questions). Abstracts will also be displayed as posters and the poster session will be held during the Welcome Reception on Sunday July 28 and morning tea and lunch on Monday July 29. Presenters should be at their posters during the poster sessions to answer any questions from delegates.

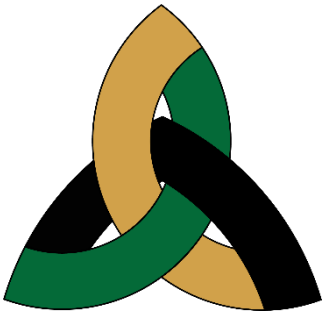
The President's Prize (PP) will be awarded in two categories: Basic Science and Clinical. The highest-ranked abstracts from eligible applicants in both categories will be presented in a single PP session. The award in each category will be based on the quality of the abstract and the presentation on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

The reviewers of the abstracts for the TSANZ 2019 meeting were:

Stephen Alexander
Richard Allen
Leyla Aouad
Adam Bartlett
Michael Burke
Steve Chadban
Daniel Chambers
Carolyn Clark
Philip Clayton
Toby Coates
Michael Collins
Peter Cowan
Nick Cross
Ian Dittmer
Randall Faull
Michael Fink
Ross Francis
David Goodman
David Gracey
Bruce Hall
Wayne Hancock
Wayne Hawthorne
Bulang He
Munish Heer
Peter Hopkins
Min Hu
Peter Hughes
Frank Ierino
Ashley Irish
Nikky Isbel
Andrew Jabbour
John Kanellis
Sean Kennedy
Jair Kwan
Darren Lee
Wai Lim
Tom Loudovaris
Grant Luxton
Fiona Mackie
Rosemary Masterson
Geoff McCaughan
Ian McKenzie
Solomon Menahem
Bill Mulley
Brian Nankivell
Kathy Nicholls
Philip O'Connell
Kathy Paizis
Helen Pilmore
Henry Pleass
Amanda Robertson
Paul Robertson
Natasha Rogers
Jessica Ryan
Shaundeep Sen
Alexandra Sharland
Lucy Sullivan
Suda Swaminathan
Alison Tong
Paul Trevillian
Jeanette Villanueva
Debbie Watson
Angela Webster
Germaine Wong
Kate Wyburn

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)



The Transplantation Society of Australia and New Zealand
Thirty-seventh Annual Scientific Meeting

PROGRAM

Saturday, 27 July 2019

14:00–15:00	Cardiac Transplant Advisory Committee (CTAC)	Sutherland Room (back partition)

Sunday, 28 July 2019

10:00–12:00	National Review of Paediatric Kidney Transplant Recipients - Work Shop	Room 210, Social Sciences Building
13:00–14:00	Pancreas & Islet Advisory Committee Meeting (PITAC)	Chancellors Room
13:30–14:30	Paediatric Transplant Advisory Committee (PTAC)	Sutherland Room (back partition)

Sunday, July 28, 2019

14:00–15:00	**Registration**	Bevery
15:00–15:10	**Official Opening:** *TSANZ President* Prof Stephen Alexander	Refectory
15:10–15:40	**PLENARY 1: Astellas Symposium** *Chair: Prof Helen Pilmore* **Detecting and Managing the At-Risk Graft** Prof Mark Stegall	Refectory
15:45–16:45	***CONCURRENT FREE COMMUNICATIONS SESSIONS*** **Free Communications 1: Surgical Techniques** *Chairs: Dr Amanda Robertson and Dr Nancy Suh*	Refectory

— Oral presentations —

Abstract		
1	15:45	**RETROPERITONEAL LAPAROSCOPIC LIVING DONOR NEPHRECTOMY: 13-YEAR EXPERIENCE AT A SINGLE CENTRE** NGUYEN Thanh-Tuan, CHAU Quy Thuan, QUACH Do La, THAI Kinh Luan, VU Duc Huy, NGUYEN Duy Dien, TRAN Trong Tri, NGUYEN Trong Hien, HOANG Khac Chuan, NGO Xuan Thai, THAI Minh Sam, TRAN Ngoc Sinh
2	15:57	**SINGLE VS DUAL (EN BLOC) KIDNEY TRANSPLANTS FROM SMALL PAEDIATRIC DONORS: AUSTRALIAN AND NEW ZEALAND (ANZ) DATA** EASTMENT Jacques, TAN Ai Lin, FRANCIS Ross, KANAGARAJAH Vijay, MUNN Stephen, GRIFFIN Anthony, JOHNSON David, PRESTON John, ISBEL Nikky
3	16:09	**CAN AN EARLY BILE DUCT FLUSH DURING DONOR PROCUREMENT REDUCE BILIARY STRICTURES AFTER LIVER TRANSPLANTATION A RANDOMISED CLINICAL STUDY** LY Mark, PULITANO Carlo, CRAWFORD Michael
4	16:21	**UPDATED SAFETY OUTCOMES AND COST-ANALYSIS OF EARLY URETERIC STENT REMOVAL IN PAEDIATRIC KIDNEY TRANSPLANTATION** TONG Marcus Chen Yee, LARKINS Nick, MINCHAM Christine, CROMPTON Charles, WILLIS Frank, HE Bulang
5	16:33	**DEVELOPMENT AND IMPLEMENTATION OF AN ENHANCED RECOVERY AFTER SURGERY (ERAS) PROTOCOL IN KIDNEY TRANSPLANTATION** RANA Abdul Ahad, DIAS Brendan, OLAKKENGIL Santosh, CLAYTON Philip, COATES Toby, BHATTACHARJYA Shanthanu

Sunday, July 28, 2019

15:45–16:45	**Free Communications 2: Outcome Measures** *Chairs: Dr Leyla Aouad and Dr Ashley Irish*		Sutherland Room (front)
Abstract	***— Oral presentations —***		
6	15:45	**WAITLISTING FOR KIDNEY TRANSPLANTATION IN AUSTRALIA: ESTIMATING THE DEMAND** CLAYTON Philip, GULYANI Aarti, SYPEK Matthew, JESUDASON Shilpa, MCDONALD Stephen	
7	15:57	**LIVING KIDNEY DONOR PROFILE INDEX: A POOR PREDICTOR OF OUTCOMES** IRISH Georgina L, CHADBAN Steve, BOUDVILLE Neil, CAMPBELL Scott, KANELLIS John, CLAYTON Philip A	
8	16:09	**OVERALL GRAFT AND PATIENT SURVIVAL AMONGST ELDERLY TRANSPLANT RECIPIENTS** SO Sarah, AU Eric HK, LEE VWS, WONG Germaine	
9	16:21	**LIVE KIDNEY DONATION FOR TYPE 2 DIABETIC (T2D) RECIPIENTS. IS THERE A BENEFIT COMPARED TO DECEASED DONATION?** GOODMAN David, ULLAH Shahid, MCDONALD Stephen P	
10	16:33	**EXTERNAL VALIDATION OF AUSTRALIA AND NEW ZEALAND TYPE 2 DIABETES (T2D) KIDNEY TRANSPLANT RISK CALCULATOR (KTRC) WITH FRENCH DATABASE AND COMPARISON TO ESTIMATED POST TRANSPLANT SURVIVAL SCORE (EPTS)** GOODMAN David, ULLAH Shahid, MCDONALD Stephen P	
15:45–16:45	**Free Communications 3: T-Regulatory Cells** *Chairs: Dr Sanda Stankovic and Dr Min Hu*		Cullen Room
Abstract	***— Oral presentations —***		
11	15:45	**A WINDOW TO TOLERANCE - HUMAN LUNG ALLOGRAFTS ARE ENRICHED FOR CD39+FOXP3+ REGULATORY T CELLS** DE SILVA Tharushi, O'SULLIVAN Brendan, APTE Simon, VOISEY Joanne, DIVITHOTAWELA Chandima, TAN Maxine, HOPKINS Peter, CHAMBERS Daniel	
12	16:00	**DEVELOPING A MODEL OF KIDNEY DIRECTED T CELL THERAPY USING CAR T CELLS** LU B, ZHANG GY, HU M, ROBINSON S, WILARUS A, WAN H, ALEXANDER SI, WANG YM	
13	16:15	**HUMAN CD27+HLA-DR+ MEMORY LIKE TREG SHOW XENOANTIGEN SPECIFIC SUPPRESSION OF PORCINE ISLET XENOGRAFT REJECTION IN HUMANIZED MICE** XIAOQIAN Ma, HU Min, CAO Lu, ZHAO Yuanfei, HUANG Dandan, BURNS Heather, HAWTHORNE Wayne, YI Shounan, O'CONNELL Phillip	

Sunday, July 28, 2019

14	16:30	**THE IMMUNOSUPPRESSIVE AGENT DILIXIMAB (ANTI-CD2) IS SUPERIOR TO ANTITHYMOCYTE GLOBULIN IN HAVING REDUCED PROTHROMBOTIC AND CELL-ACTIVATING EFFECTS** BONGONI Anjan K, SALVARIS Evelyn J, LEW Andrew M, COWAN Peter J	
16:30–17:00	Vascular Composite Allograft Advisory Committee (VCAAC)		Sutherland Room (Back Partition)
16:45–17:00	**Afternoon tea**		Bevery and Drawing Room
17:00–18:00	**Novartis Josette Eris Lecture and Ian McKenzie Award** *Chairs: A/Prof Natasha Rogers and Dr Chien-Li Liew* 17:00 **Ian McKenzie Award** A/Prof Germaine Wong 17:20 **Navigating the Waters of Science With Dendritic Cells and Neuropeptides** Prof Adriana Larregina		Refectory
18:00–19:00	**Welcome Reception and Poster Viewing**		Drawing Room

Monday, July 29, 2019

06:15–07:15	**TSANZ Fun Run/Walk** (5km)	Sponsor: Transplant Australia	Circular Quay to Hyde Park
07:30–08:00	**Breakfast** with sponsors		Bevery

08:00–09:40 — Refectory

PLENARY 2: Organ and Tissue Authority Symposium

Joint TSANZ /OTA/ATCA Session
Chairs: Prof John Kanellis and A/Prof Georgina Clark

08:00 **Dendritic Cell-Based Therapies in Transplantation: What do They do *in vivo***
Prof Adrian Morelli

08:30 **Targeting Dendritic Cells for Antigen-Specific Immunotherapy**
Prof Ranjeny Thomas

08:50 **Implementation of Halifaster Flow Crossmatching**
Ms Mary Diviney

09:10 **Organ Offers – a Year in Review**
Mr Luke Datson

09:25 **OTA report**
Ms Lucinda Barry

09:40–19:40 — Refectory

CONCURRENT FREE COMMUNICATIONS SESSIONS

Free Communications 4: Organ Donation and Preservation
Chairs: Prof Henry Pleass and Dr Jinna Yao

— Oral presentations —

Abstract

15 09:40 **DONOR SOURCE OF KIDNEY TRANSPLANTATION IN NEW ZEALAND BY ETHNICITY: A LONGITUDINAL COHORT STUDY**
CROSS Nicholas , DONNELLAN Sine, WILLIMAN Jonathon, PALMER Suetonia

16 09:52 **OFFER DECLINES HAVE INCREASED FOR HIGHER RISK KIDNEYS IN SINCE KIDNEY DONOR PERFORMANCE INDEX (KDPI) REPORTING WAS INTRODUCED IN AUSTRALIA.**
SYPEK MP, HUGHES P, MCDONALD S, CLAYTON P

17 10:04 **THE CHANGING PATTERN OF LIVING KIDNEY DONATION IN AUSTRALIA AND NEW ZEALAND**
ALLEN Richard, DOBRIJEVIC Ellen, CLAYTON Phil, WONG Germaine, CROSS Nicholas, ROAKE Justin, VASILARAS Arthur, PLEASS Henry

Monday, July 29, 2019

18	10:16	**COLD PERFUSION MACHINE ALLOWS PROLONGED STORAGE OF DONOR MATCHED KIDNEYS WITHOUT ADVERSE OUTCOME** RYAN Brendan, HORT Amy, SHAHRESTANI Sara, PLEASS Henry, YUEN Lawrence, LAM Vincent, HAWTHORNE Wayne J, ROBERTSON Paul, HITOS Kerry, DE ROO Ronald, BYRNE Sarah, PEREIRA Ryan, ROBERTSON Ian, TAN Ai Lin, LOCKWOOD David, KANAGARAJAH Vijay, RAY Mark, PRESTON John, WOOD Simon, LAWSON Malcolm, GRIFFIN Anthony, RHEE Handoo	
19	10:28	**COLD PERFUSION MACHINE DATASETS USED FOR KIDNEY TRANSPLANTS MAY PREDICT DELAYED GRAFT FUNCTION INCLUDING THE NEED FOR POST-TRANSPLANT DIALYSIS** Byrne Sarah, Pereira Ryan, Robertson Ian, Tan Ai Lin, Lockwood David, Kanagarajah Vijay, Ray Mark, Preston John, Wood Simon, Lawson Malcolm, Griffin Anthony, Rhee Handoo	
09:40–10:40		**Free Communications 5: Antibodies** *Chairs: A/Prof Kate Wyburn and Dr Stella McGinn*	Sutherland Room (front)
Abstract		***— Oral presentations —***	
20	09:40	**CORRELATION AND AGREEMENT BETWEEN HLA-DRB AND HLA-DQ EPLET MISMATCHES BY LINKAGE DISEQUILIBRIUM AND HIGH-RESOLUTION HLA TYPING** LARKINS NG, TAVERNITI A, SHARMA A, DE SANTIS D, IRISH A, CHAKERA A, D'ORSOGNA L, WONG G, LIM WH	
21	09:52	**ABO INCOMPATIBLE LIVING DONOR KIDNEY TRANSPLANTATION IN AUSTRALIA AND NEW ZEALAND: A REPORT FROM THE ANZDATA REGISTRY** CLAYTON Philip, DANSIE Kathryn, CAMPBELL Scott, COATES Toby, COHNEY Solomon, IERINO Frank, IRISH Ashley, ISBEL Nicole, HUGHES Peter, KANELLIS John, LIM Wai, MULLEY William, PILMORE Helen, RUSS Graeme, TREVILLIAN Paul, WONG Germaine, WYBURN Kate	
22	10:04	**DEVELOPMENT OF DE NOVO HLA DONOR SPECIFIC ANTIBODIES AND ALLOGRAFT REJECTION POST BLOOD TRANSFUSION IN KIDNEY TRANSPLANT RECIPIENTS** JALALONMUHALI Maisarah, CARROLL Robert, CLAYTON Philip, COATES Toby	
23	10:16	**RAPID REDUCTION OF DONOR-SPECIFIC ANTIBODIES IN SIMULTANEOUS LIVER-KIDNEY TRANSPLANTATION** NEWMAN Allyson , LAI Sum Wing Christina, ABOU-DAHER Frederika, WALTON Duncan, WATSON NARELLE, MAWSON JANE, WYBURN KATE, CHADBAN STEVE, GRACEY DAVID	

Monday, July 29, 2019

24	10:28	**COSTS IN CONTEMPORARY TRANSPLANT PRACTICE** WYLD Melanie, YING Tracey, WYBURN Kate, CHADBAN Steve	
09:40–10:40		**Free Communications 6: Tolerance** *Chairs: A/Prof Alexandra Sharland and A/Prof David Goodman*	Cullen Room
Abstract		***— Oral presentations —***	
25	09:40	**IN VITRO EXPANSION OF IL-10-COMPETENT HUMAN B CELLS** PERKINS Griffith, COATES Toby, HURTADO Plinio	
26	09:55	**EXPRESSION OF DONOR MHC CLASS I IN RECIPIENT HEPATOCYTES DOES NOT INDUCE LINKED SUPPRESSION TO H-Y** LEONG Mario, PAUL-HENG Moumita, WANG Chuanmin, SON Taeyoung, BERTOLINO Patrick, BISHOP Alex, BOWEN David, SHARLAND Alexandra	
27	10:10	**THE SELF-PEPTIDE REPERTOIRE PLAYS A CRITICAL ROLE IN TRANSPLANT TOLERANCE INDUCTION.** SON Eric, PAUL-HENG Moumita, LEONG Mario, FARIDI Pouya, MIFSUD Nicole, ALEXANDER Ian, BERTOLINO Patrick, PURCELL Anthony, BOWEN David, SHARLAND Alexandra	
28	10:25	**EFFECT ON CHRONIC ALLOGRAFT REJECTION OF ANTIGEN SPECIFIC TREG INDUCED BY ACTIVATION OF NAIVE TREG WITH ALLOANTIGEN AND IL-4** RAKESH PRATEEK, BEDI SUKHANDEEP K, VERMA NIRUPAMA D, TRAN GIANG, ROBINSON CATHERINE M., WANG CHUANMIN, SHARLAND ALEXANDRA, HALL RACHEL M, WILCOX PAUL L, HODGKINSON SUZANNE J, HALL BRUCE M	
09:50–10:50		Lung Transplant Advisory Committee (LTAC)	Chancellors Room
10:40–11:10		**Morning tea and Poster Viewing** Xeno-Transplantation Working Group (XTWG)	Bevery and Drawing Room Sutherland Room (Back Partition)

Monday, July 29, 2019

11:10–12:50	**PLENARY 3: Astellas Symposium** **Complications and Rejection - Prevention and Prediction** *Chairs: A/Prof Germaine Wong and A/Prof Scott Campbell* 11:10 **Update on Immunisation of the Transplant Recipient** Prof Deepali Kumar 11:40 **Predicting Infections in Transplant Recipients** Dr Claire Dendle 12:10 **Diagnosis of Kidney Transplant Rejection Using Cell-Free DNA** Dr John Whitlam 12:30 **Skin Cancer** Prof Pablo Penas	Refectory
12:50–13:35	**Lunch and Poster Viewing** Donor Surgeons Donor Coordinators Advisory Committee (DSDC)	Bevery and Drawing Room Sutherland Room (Back Partition)
13:33–15:35	**President's Prize Symposium** *Chair: TSANZ President, Prof Stephen Alexander* ***— Oral presentations —***	Refectory
29	13:35 **LONG-TERM P2X7 BLOCKADE REDUCES LIVER GRAFT-VERSUS-HOST DISEASE IN HUMANISED MICE** GERAGHTY Nicholas, WATSON Debbie, SLUYTER Ronald	
30	13:50 **RISK FACTORS FOR ADVANCED COLORECTAL NEOPLASIA IN KIDNEY TRANSPLANT RECIPIENTS** AU Eric HK, HOWARD Kirsten, CHAPMAN Jeremy R, CASTELLS Antoni, ROGER Simon, BURKE Michael J, MACASKILL Petra, TURNER Robin, WILLIAMS Gabrielle, LIM Wai H, LOK Charmaine E, DIEKMAN Fritz, CROSS Nicholas, SEN Shaundeep, ALLEN Richard DM, CHADBAN Steven J, POLLOCK Carol A, TONG Allison, TEIXEIRA-PINTO Armando, YANG Jean YH, WILLIAMS Narelle, KIEU Anh, JAMES Laura, CRAIG Jonathan, WONG Germaine	
31	14:05 **TARGETING INFLAMMATORY MONOCYTES BY IMMUNE-MODIFYING NANOPARTICLES PREVENTS KIDNEY ALLOGRAFT REJECTION** LAI Sum Wing Christina, WU Huiling, LOH Yik Wen, SINGER Julian, NIEWOLD Paula, GETTS Daniel, KING Nicholas, CHADBAN Steve	

Monday, July 29, 2019

32	14:20	**EFFECT OF PROTON PUMP INHIBITOR ON MYCOPHENOLIC ACID EXPOSURE IN KIDNEY AND LIVER TRANSPLANT RECIPIENTS: A DOUBLE-BLIND RANDOMISED CROSS-OVER TRIAL** SUNDERLAND Andrew, RUSS Graeme, SALLUSTIO Benedetta, CERVELLI Matthew, JOYCE David, JEFFREY Gary, BOUDVILLE Neil, CHAKERA Aron, DOGRA Sharan, CHAN Doris, WONG Germaine, LIM Wai	
33	14:35	**A20 IS PROTECTIVE AGAINST ACUTE KIDNEY INJURY REGARDLESS OF IMMUNOLOGICAL ENVIRONMENT** LI Jennifer, NGUYEN-NGO Danny, ZAMMIT Nathan, EL-RASHID Maryam, MINHAS Nikita, GREY Shane, ROGERS Natasha	
34	14:50	**EVEROLIMUS IN THE PREVENTION OF CALCINEURIN-INHIBITOR-INDUCED LEFT VENTRICULAR HYPERTROPHY IN HEART TRANSPLANTATION (RAD-TAC STUDY)** ANTHONY Chris, IMRAN Mohammad, EMMANUEL Sam, ILIFF James, KOTLYAR Eugene, MUTHIAH Kavitha, KEOGH Anne, HAYWARD Christopher, MACDONALD Peter, JABBOUR Andrew	
35	15:05	**HIGH FIBRE DIET PREVENTS KIDNEY ALLOGRAFT REJECTION AND PROTECTS AGAINST TRANSPLANT-ASSOCIATED DYSBIOSIS** SINGER Julian, WU Huiling, KWAN Tony, LOH Yik Wen, WANG Chuanmin, TAN Jian, Li Yan J, LAI Sum Wing Christina, MACIA Laurence, ALEXANDER Stephen I, CHADBAN Stephen J	
36	15:20	**EFFECT OF LANGUAGE AND COUNTRY OF BIRTH ON MEDICAL SUITABILITY AND CONSENT IN SOLID ORGAN DONOR REFERRALS IN NEW SOUTH WALES 2010-2015 – A LINKED-DATA COHORT STUDY** WALLER Karen, HEDLEY James, DE LA MATA Nicole, ROSALES Brenda, WYBURN Kate, KELLY Patrick, O'LEARY Michael, CAVAZZONI Elena, WEBSTER Angela	
15:35–16:00		**Afternoon tea**	Bevery and Drawing Room
16:00–17:00		***CONCURRENT FREE COMMUNICATIONS SESSIONS***	
		Free Communications 7: Transplant Outcomes and Complications *Chairs: Dr Michael Collins and Dr Tracey Ying*	Refectory
Abstract		***— Oral presentations —***	

Monday, July 29, 2019

37	16:00	**INCIDENCE AND PREDICTORS OF INFECTIOUS-RELATED MORTALITY IN RECIPIENTS OF A KIDNEY TRANSPLANT: A REGISTRY STUDY** CHAN Samuel, PASCOE Elaine Mary, CLAYTON Philip Andrew, MCDONALD Stephen, LIM Wai, SYPEK Matthew, PALMER Suetonia, ISBEL Nicole, FRANCIS Ross, CAMPBELL Scott, HAWLEY Carmel, JOHNSON David	
38	16:12	**A LONGITUDINAL STUDY OF GAMMA DELTA T CELLS POST LUNG TRANSPLANT: POTENTIAL PLAYERS IN IMMUNITY TO CYTOMEGALOVIRUS** SULLIVAN Lucy, SHAW Evangeline, STANKOVIC Sanda, SNELL Gregory, BROOKS Andrew, WESTALL Glen	
39	16:24	**DOES EARLY REMOVAL OF URETERIC STENT SIMULTANEOUSLY WITH INDWELLING URETHRAL CATHETER POST KIDNEY TRANSPLANTATION REDUCE INFECTION RATES AND HEALTHCARE COSTS?** TRAPNELL FRANK, HEIN Martin, MOU Lingjun, JAQUES Bryon, BOUDVILLE Neil, CHAKERA Aron, HE Bulang	
40	16:36	**CARDIAC MORTALITY IN TRANSPLANT PATIENTS; A POPULATION-BASED COHORT STUDY 1988-2013 IN AUSTRALIA AND NEW ZEALAND** WYLD Melanie, DE LA MATA Nicole, MASSON Philip, O'LONE Emily, WEBSTER Angela	
41	16:48	**ASSOCIATION BETWEEN THE SIDE OF LIVE DONOR KIDNEYS AND TRANSPLANT RECIPIENT OUTCOMES** DOBRIJEVIC Ellen, WONG Germaine, CLAYTON Phil, ALLEN Richard	
16:00–17:00		**Free Communications 8: Liver, Islet and Pancreas Transplantation** *Chairs: Ms Heather Burns and Dr Nicholas Cocco*	Sutherland Room (front)
Abstract		***— Oral presentations —***	
42	16:00	**LONG-TERM OUTCOMES OF UTILISING DONATION AFTER CIRCULATORY DEATH GRAFTS IN LIVER TRANSPLANTATION – AN AUSTRALIAN 12-YEAR COHORT STUDY** SASTRY Vinay, PANDYA Keval, PANLILIO Mara, WEST Claire, VIRTUE Susan, WELLS Mark, CRAWFORD Michael, PULITANO Carlo, STRASSER Simone, MCCAUGHAN Geoff, MAJUMDAR Avik, LIU Ken	

Monday, July 29, 2019

43	16:12	**LONG-TERM OUTCOMES OF UTILISING EXTENDED CRITERIA DECEASED DONORS IN LIVER TRANSPLANTATION – AN AUSTRALIAN 12-YEAR COHORT STUDY** PANDYA Keval, SASTRY Vinay, PANLILIO Mara, WEST Claire, VIRTUE Susan, WELLS Mark, CRAWFORD Michael, PULITANO Carlo, STRASSER Simone, MCCAUGHAN Geoff, MAJUMDAR Avik, LIU Ken	
44	16:24	**IGLS CRITERIA APPLIED TO THE AUSTRALIAN ISLET TRANSPLANT PROGRAM.** KAY Thomas, GOODMAN David, LOUDOVARIS Thomas, RADFORD Toni, ANDERSON Patricia, HOWE Kathy, COATES Toby, O'CONNELL Philip	
45	16:36	**FACTORS INFLUENCING ISLET ISOLATION OUTCOMES: 20-YEAR DATA FROM THE WESTMEAD ISLET TRANSPLANT PROGRAM** CHEW Yi Vee, WILLIAMS Lindy, BURNS Heather, JIMENEZ-VERA Elvira, HUANG Dandan, O'CONNELL Philip, HAWTHORNE Wayne	
46	16:48	**A SYSTEMATIC REVIEW AND META-ANALYSIS TO IDENTIFY THE RISK FACTORS FOR PANCREATIC ALLOGRAFT THROMBOSIS FOLLOWING SIMULTANEOUS PANCREAS-KIDNEY TRANSPLANTATION** BLUNDELL Jian, SHAHRESTANI Sara, LENDZION Rebecca, HAWTHORNE Wayne	
16:00–17:00		**Free Communications 9: IRI, Immunobiology and GvHD** *Chairs: Dr Debbie Watson and Dr Moumita Paul-Heng*	Cullen Room
Abstract		***— Oral presentations —***	
47	16:00	**RECOMBINANT SOLUBLE CR1 TREATMENT IS PROTECTIVE IN A MOUSE MODEL OF RENAL ISCHEMIA REPERFUSION INJURY** BONGONI Anjan K, MCRAE Jennifer L, SALVARIS Evelyn J, VIKSTROM Ingela, MORELLI Adriana Baz, WYMANN Sandra, HARDY Matthew P, PEARSE Martin J, COWAN Peter J	
48	16:15	**NECROPTOSIS IN RENAL ISCHAEMIA REPERFUSION INJURY** PEFANIS A, NACHBUR U, MCRAE JL, BONGONI AK, SALVARIS EJ, MURPHY JM, IERINO FL, COWAN PJ	
49	16:30	**SELECTIVE RETENTION OF DONOR MYELOID CELLS IN CONGENIC LIVER TRANSPLANTS** DART SJ, PROSSER A, HUANG WH, LIU L, DE BOER B, JEFFREY G, DELRIVIERE L, KALLIES A, LUCAS M	

Monday, July 29, 2019

50	16:45	**THE P2X7 ANTAGONIST BRILLIANT BLUE G PRESERVES REGULATORY T CELLS AND REDUCES SERUM HUMAN INTERFERON-GAMMA IN A HUMANISED MOUSE MODEL OF GRAFT-VERSUS-HOST DISEASE** CUTHBERTSON Peter, ADHIKARY Sam, SLUYTER Ronald, WATSON Debbie	
17:00–18:00		**TSANZ Annual General Meeting**	Refectory
19:00–23:00		**TSANZ Annual Dinner**	Great Hall, Sydney University

Tuesday, July 30, 2019

07:30–08:00	**Coffee** with sponsors	Bevery
08:00–09:30	**PLENARY 4: Novartis Symposium** **Manipulating the Immune Response** *Chairs: Prof Philip O'Connell and Dr Lucy Sullivan* 08:00 **Proinflammatory Neuropeptides as Regulators of T Cell Function** Prof Adriana Larregina 08:30 **Chromatin Dynamics and Genome Function** Dr Elizabeth Hinde 09:00 **Donor Derived and Banked Third Party T Cells to Prevent Relapse and Infection After Allogeneic Stem Cell Transplant** Prof David Gottlieb	Refectory
09:30–10:30	***CONCURRENT STATE OF THE ART SESSIONS*** **STATE OF THE ART 1: Astellas Symposium** **Infections** *Chairs: A/Prof Peter Hughes and Dr Karen Keung* 09:30 **Infection Risk Donors** Prof Deepali Kumar 09:55 **Development and Implementation of Vic IVRD Consent** Dr Darren Lee 10:10 **CMV resistance to Antivirals in Transplantation-Mechanisms, Consequences and Potential Responses** Prof William Rawlinson	Refectory

Tuesday, July 30, 2019

09:30–10:30	**STATE OF THE ART 2: Novartis Symposium** **Surgical Session** *Chairs: Mr Michael Fink and Dr Sherry Salter* 09:30 **Robotic Kidney Transplantation** A/Prof Jerome Laurence 09:50 **Machine Perfusion** Prof Henry Pleass 10:10 **Laparoscopic Donor Surgery – What Not to do: a Single Surgeon's Experience** Dr Nancy Suh	Sutherland Room (front)
10:30–11:00	**Morning tea**	Bevery and Drawing Room
11:00–12:30	***CONCURRENT STATE OF THE ART SESSIONS*** **STATE OF THE ART 3: Astellas Symposium** **Trials and Allocation** *Chairs: Prof Steve Chadban and Ms Rhonda Holdsworth* 11:00 **The Need for Novel Clinical Trial Designs in Transplantation** Prof Mark Stegall 11:30 **ANZ Trials in Transplantation** Prof Jonathan Craig 11:50 **Organ Match Update and Now How to Use it** Ms Rhonda Holdsworth 12:10 **Heart Allocation and Outcomes** Prof Peter MacDonald	Refectory

Tuesday, July 30, 2019

11:00–12:30	**STATE OF THE ART 4: Novartis Symposium** **Equity in Transplantation** *Chairs: Dr Darren Lee and A/Prof Bronwyn Levvey* 11:00 **Patient Outcomes and Perspectives** Prof Allison Tong and Ms Nicki Scholes-Robertson 11:30 **Women in Transplantation** A/Prof Kate Wyburn 11:50 **Indigenous Australians and Transplantation** Dr Paul Lawton 12:10 **Transplantation in NT** Dr William Majoni	Sutherland Room (front)
12:30–13:30	**Lunch**	Bevery and Drawing Room
	Immune Tolerance Advisory Committee (ITAC)	Chancellors Room
	Paediatric Donor Working Group (PDWG)	Sutherland Room (Back Partition)
12:30–14:30	Renal Transplant Advisory Committee (RTAC)	Cullen Room
13:30–15:00	**PLENARY 5: Novartis Symposium** **Different Perspectives on Determining Immune Responses** *Chairs: Prof Shane Grey and Dr Jeanette Villanueva* 13:30 **Neutrophils in the Innate Immune Response** Dr Tatyana Chtanova 14:00 **Extracellular Vesicles in Transplantation: From Concealed Mechanisms of Allorecognition to Biomarkers** Prof Adrian Morelli 14:30 **Liver Immunology** A/Prof Patrick Bertolino	Refectory
15:00–15:25	**Afternoon tea**	Bevery and Drawing Room

Tuesday, July 30, 2019

15:25–16:00	**The Great Debate: Transplant Clinicians Focus too Much on Rejection and Not Enough on Infection** *Moderator: Prof Toby Coates* Pro team: Prof Mark Stegall and Dr Carolyn Clark Con team: Prof Deepali Kumar and Dr Sharon Ford Pro Team, speaker 1 Con Team, speaker 1 Pro Team, speaker 2 Con Team, speaker 2 Pro Team rebuttal (if required) Con Team rebuttal (if required)	Refectory
16:00	**ASM Concludes**	
16:00–18:30	Liver and Intestinal Transplant Advisory Committee (LITAC)	Sutherland Room (Back Partition)

TSANZ ASM, Sydney July 28-30, 2019 Posters

Abstract	— *Poster* —	Drawing Room
51	**A SURVEY OF THE CONTEMPORARY USE OF JJ STENTS IN RENAL TRANSPLANTATION IN AUSTRALIA AND NEW ZEALAND AND THE FEASIBILITY OF A CLINICAL TRIAL OF OPTIMAL TIMING OF STENT REMOVAL** JAMBOTI Jagadish, IRISH Ashley, BHANDARI Mayank, HAWLEY Carmel	
52	**CLOSED INCISION NEGATIVE PRESSURE WOUND THERAPY IS SAFE AND FEASIBLE IN KIDNEY TRANSPLANT PATIENTS** HERLIHY David, TRAN Quoc, PUTTASWAMY Vikram, FISHER Charles	
53	**CONTEMPORARY MANAGEMENT OF PROSTATE CANCER IN RENAL TRANSPLANT PATIENTS** PEREIRA Ryan, ROBERTSON Ian, BYRNE Sarah, GRIFFIN Anthony, LAWSON Malcolm, PRESTON John, WOOD Simon, RHEE Handoo	
54	**LAPAROSCOPIC TRANSPLANT NEPHRECTOMY FOR A FAILED INTRA-PERITONEAL TRANSPLANT KIDNEY** HEER MK, BULL Nick, TREVILLIAN PR	
55	**KIDNEY TRANSPLANTATION USING DONORS WITH SINGLE AND MULTIPLE RENAL ARTERIES – IS THERE A DIFFERENCE?** TRAN Quoc, HERLIHY David, MORITZ Peter, PUTTASWAMY Vikram	
56	**RISK FACTORS AND OUTCOMES OF VERY EARLY ACUTE REJECTION COMPLICATING KIDNEY TRANSPLANTATION IN AUSTRALIA AND NEW ZEALAND – A REGISTRY ANALYSIS** PUTRINO Samantha, FRANCIS Ross, JOHNSON David, CHO Yeoungjee, HAWLEY Carmel, CAMPBELL Scott, ISBEL Nikky, FAHIM Magid, PASCOE Elaine, MUDGE David	
57	**EVALUATION OF KIDNEY FUNCTION IN LIVING DONORS** GUO Henry, MCGINN Stella, LI Yan	
58	**IMPACT OF DONOR HOSPITAL LOCATION ON LUNG ACCEPTANCE AND TRANSPLANT OUTCOMES** CAREW AM, YERKOVICH ST, HOPKINS PMA, CHAMBERS DC	

TSANZ ASM, Sydney July 28-30, 2019 Posters

Abstract	— *Poster* —	Drawing Room
59	**ALLOSEQ CFDNA ASSAY MEASURES DONOR-DERIVED CELL-FREE DNA IN SOLID ORGAN TRANSPLANT RECIPIENTS** BALTIC Svetlana, VIARD Thierry, AMOKRANE Kahina, DRABBELS Jos JM., FAÉ Ingrid, FORTIER Catherine, MATHERS Simon, WENDA Sabine, LEE Deanna, O'CONNOR Timothy, ROSS David, WANG Sarah, GRSKOVIC Marica, ROELEN Dave, BURLINSO1N Natalia Diaz, TAUPIN Jean-Luc, FISCHER Gottfried F	
60	**PATIENT AND GRAFT OUTCOMES FOLLOWING SIMULTANEOUS LIVER-KIDNEY TRANSPLANTATION: AN ANZDATA REGISTRY ANALYSIS** TANGIRALA NISHANTA, GRACEY David1, WONG Germaine, FINK Michael, WYBURN Kate1, CHADBAN Steven, MCCAUGHAN Geoff, ADAMS Leon, JEFFREY Gary, FAWCETT Jonathan, BYRNE Mandy, CATALAN Aimee, LIM Wai	
61	**RESPONSE PROCESS OF A PROPOSED CORE OUTCOME MEASURE FOR LIFE PARTICIPATION FOR TRIALS IN KIDNEY TRANSPLANT RECIPIENTS: A COGNITIVE PILOT STUDY** BAUMGART Amanda, TONG Allison, HOWELL Martin, CRAIG Jonathan C, JOSEPHSON Michelle, JU Angela	
62	**FACTORS INFLUENCING SOUTH WESTERN SYDNEY LOCAL HEALTH DISTRICT (SWSLHD) DECEASED DONOR RENAL TRANSPLANT WAITING LIST ACTIVATION** CHEUNG Jason, ZAHOROWSKA Beata, SHANMUGALINGAM Renuka, MUNRO Colleen E, WONG Jeffrey KW	
63	**CLINICAL CHARACTERISTICS OF SIMULTANEOUS LIVER KIDNEY TRANSPLANT RECIPIENTS AND AN ANALYSIS OF THEIR LONG-TERM RENAL ALLOGRAFT AND PATIENT OUTCOMES COMPARED TO KIDNEY TRANSPLANT ALONE RECIPIENTS: AN ANZDATA REGISTRY ANALYSIS** TANGIRALA Nishanta, LIM Wai, WONG Germaine, FINK Michael, CHADBAN Steven, WYBURN Kate, MCCAUGHAN Geoffrey, ADAMS Leon, FAWCETT Jonathan, JEFFREY Gary, CATALAN Aimee, BYRNE Mandy, GRACEY David	
64	**HUMAN LEUKOCYTE ANTIGEN MATCHING IN DECEASED DONOR ALLOCATION DOES NOT PREDICT ACUTE REJECTION EPISODES** GRAMLICK Madelyn E, YAUSIS Samuel, TREVILLIAN Paul R, HEER Munish K	

TSANZ ASM, Sydney July 28-30, 2019 Posters

Abstract	— *Poster* —	Drawing Room
65	**VACCINATION SERORESPONSE AS A PREDICTOR FOR SUBSEQUENT KIDNEY TRANSPLANT REJECTION AND SERIOUS INFECTION** TA'EED A, POLKINGHORNE KR, MULLEY WR	
66	**EVALUATION OF THE EQUATIONS TO ESTIMATE THE RENAL FUNCTION OF KIDNEY TRANSPLANT RECIPIENTS FOCUSING ON CREATININE CLEARANCE** MARUI Yuhji, YOZA Naoto, SATO Yoshitsugu, MATSUMURA Kaori, USUBA Wataru, AOKI Naoto, NISHI Tomohiro, KATSUOKA Yuichi, NAKAZAWA Ryuto, SASAKI Hideo, CHIKARAISHI Tatsuya	
67	**CANCER INCIDENCE IN DONOR REFERRALS - A NSW COHORT STUDY 2010-2015 USING DATA LINKAGE** HEDLEY James, DE LA MATA Nicole, ROSALES Brenda, O'LEARY Michael, CAVAZZONI Elena, KELLY Patrick1, WYBURN Kate, WEBSTER Angela	
68	**GENETIC TESTING SHOWS HIGH FREQUENCY OF MENDELIAN DISORDERS IN PAEDIATRIC KIDNEY TRANPLANT RECIPIENTS** MCCARTHY Hugh, HOLMAN K, HO G, BENNETTS B, ALEXANDER SI	
69	**EHEALTH INTERVENTIONS FOR SOLID ORGAN TRANSPLANT RECIPIENTS: A SYSTEMATIC REVIEW AND META-ANALYSIS OF RANDOMISED CONTROLLED TRIALS** TANG James, JAMES Laura, HOWELL Martin, TONG Allison, WONG Germaine	
70	**A CASE OF ABO INCOMPATIBLE RENAL TRANSPLANTATION FOLLOWING BLOOD GROUP SWITCHING IN THE RENAL TRANSPLANT PATIENT** LAM Susanna, HULTIN Sebastian, PRESTON John, CAMPBELL Scott	
71	**FREQUENCY AND OUTCOMES OF KIDNEY DONATION FROM INTENSIVE CARE PATIENTS WITH ACUTE RENAL FAILURE REQUIRING RENAL REPLACEMENT THERAPY** SANDERS Jo, OPDAM Helen, FURNISS Hayley, HUGHES Peter, KANELLIS John, JONES Daryl	

TSANZ ASM, Sydney July 28-30, 2019 Posters

Abstract	— *Poster* —	Drawing Room
72	**SATISFACTORY PERFORMANCE OF THE ABBOTT ARCHITECT I2000 TACROLIMUS IMMUNOASSAY AGAINST THE LC-MS/MS TACROLIMUS ASSAY DEMONSTRATED IN KIDNEY TRANSPLANT PATIENTS FROM THE NORTHERN TERRITORY, AUSTRALIA** KAREPALLI Vijay K, RATHNAYAKE Geetha, MOGULLA Manohar, ASHFORD Jenna, MAJONI Sandawana William, SALLUSTIO Benedetta C	
73	**TRAJECTORY OF DECLINE IN KIDNEY FUNCTION AND ASSOCIATION WITH ALL-CAUSE GRAFT LOSS IN AUSTRALIAN KIDNEY TRANSPLANT PATIENTS: JOINT LATENT CLASS MIXED MODELS** VON HUBEN Amy, TEIXEIRA-PINTO Armando, AU Eric, WONG Germaine	
74	**DONOR REFERRALS WITH PRIMARY BRAIN TUMOUR – PERCEIVED VS. VERIFIED RISK** THOMSON Imogen, HEDLEY James, DE LA MATA Nicole, ROSALES Brenda, O'LEARY Michael, CAVAZZONI Elena, KELLY Patrick, WYBURN Kate, WEBSTER Angela	
75	**ANALYSING THE EFFECTS OF CLINICAL PREDICTIVE VARIABLES ON KIDNEY TRANSPLANT OUTCOMES IN RANDOM FOREST MODELS** PAIZIS Kathy, SLOGGETT Clare, SYPEK Matthew, IERINO Francesco	
76	**HBA1C AS A PREDICTOR OF POSTOPERATIVE KIDNEY COMPENSATORY HYPERTROPHY IN MALE LIVING DONORS** KOSUKE Tanaka, KOHEI Kinoshita, YUJI Hidaka, MARIKO Toyoda, AKITO Inadome, ASAMI Takeda, JUN Shoji, SHIGEYOSHI Yamanaga	
77	**COMBATING LOSS TO FOLLOW UP OF LIVING KIDNEY DONORS** GUO Henry, MCGINN Stella, LI Yan	
78	**ENDOTHELIAL GLYCOCALYX DYSFUNCTION IN ORGAN DONORS IS ASSOCIATED WITH DELAYED GRAFT FUNCTION IN RENAL TRANSPLANT RECIPIENTS BUT NOT EARLY ALLOGRAFT DYSFUNCTION IN LIVER RECIPIENTS** SLADDEN TM, YERKOVICH S, JAFFREY L, REILING J, FAWCETT J, ISBEL N, CHAMBERS D	
79	**DIAGNOSTIC TESTS FOR DELAYED GRAFT FUNCTION: A SYSTEMATIC REVIEW** LAI, Christina, YEE, Seow Yeing, YING, Tracey, CHADBAN, Steve	

TSANZ ASM, Sydney July 28-30, 2019 Posters

Abstract	— *Poster* —	Drawing Room
80	**IDENTIFICATION OF BARRIERS FOR INDIGNEOUS AUSTRALIANS GAINING ACCESS TO THE KIDNEY TRNASPLANT WAITING LIST: A VICTORIAN PILOT STUDY** ATKINSON Amy, FORD Sharon, GOCK Hilton, IERINO Frank, GOODMAN David	
81	**HEALTHCARE PROFESSIONAL AND COMMUNITY PREFERENCES IN DECEASED DONOR KIDNEY ALLOCATION: DO THE PRIORITIES ALIGN?** SYPEK MP, HOWELL M, DUNCANSON E, HUGHES P, WONG G, CLAYTON P, HOWARD K, MCDONALD S	
82	**IATROGENIC LIVER INJURY SUSTAINED DURING DECEASED DONOR ORGAN PROCUREMENT: AN ANALYSIS OF THE RISK FACTORS AND CONSEQUENCES IN AN AUSTRALIAN TRANSPLANT CENTRE** WALCOTT James, FINK Michael, MURALIDHARAN Vijayaragavan, CHRISTOPHI Christopher	
83	**MISSED OPPORTUNITIES FOR ORGAN DONATION AMONG DONORS WITH PRIMARY BRAIN MALIGNANCIES (PBMS): NEW SOUTH WALES (NSW) COHORT STUDY 2010-2015** THOMSON Imogen, HEDLEY James, ROSALES Brenda, WYBURN Kate, WEBSTER Angela	
84	**IMPLEMENTATION OF INCREASED VIRAL RISK DONOR WAITING LIST IN VICTORIA – A USEFUL ADDITION TO THE DONOR POOL** LEE Darren, SENG Nina, GRAMNEA Indra, HUDSON Fiona, D'COSTA Rohit, MCEVOY Leanne, SASADEUSZ Joe, GOPAL Basu, KAUSMAN Joshua, MASTERSON Rosemary, PAIZIS Kathy, KANELLIS John, HUGHES Peter, GOODMAN David, WHITLAM John	
85	**THE IMPORTANCE OF DONOR CANCER HISTORY IN DETERMINING USE OF KIDNEYS FROM CONSENTED DONORS** AU Eric HK, PHILLIPS Jessica, OPDAM Helen, MCDONALD Mark, CHAPMAN Jeremy R, MCDONALD Stephen, JOHNSON David, KANELLIS John, WONG Germaine, LIM Wai H	
86	**MISSED OPPORTUNITIES FOR ORGAN DONATION? USE OF LINKED HEALTH DATA TO VERIFY INCREASED BLOODBORNE VIRUS (BBV) RISK AMONG NSW ORGAN DONOR REFERRALS, 2010-2015** DE LA MATA Nicole, WALLER Karen, HEDLEY James H, ROSALES Brenda, KELLY Patrick J, WYBURN Kate, O'LEARY Michael, CAVAZZONI Elena, WEBSTER Angela C	

TSANZ ASM, Sydney July 28-30, 2019 Posters

Abstract	— *Poster* —	Drawing Room
87	**ADDRESSING TRANSPLANT TOURISM INTO CHINA** MATAS David	
88	**BLOODBORNE VIRUS (BBV) INFECTIONS IN NSW ORGAN DONOR REFERRALS USING LINKED HEALTH DATA: THE SAFEBOD COHORT, 2010-2015** DE LA MATA Nicole, WALLER Karen, HEDLEY James H, ROSALES Brenda, KELLY Patrick J, WYBURN Kate, O'LEARY Michael, CAVAZZONI Elena, WEBSTER Angela C	
89	**EXTENDING LIVING KIDNEY DONOR SELECTION CRITERIA: A BIOETHICAL PERSPECTIVE** WEIGHTMAN AC	
90	**INCIDENCE AND OUTCOMES OF ANTIBODY MEDIATED REJECTION IN THE AUSTRALIAN AND NEW ZEALAND RENAL TRANSPLANT POPULATION** DANSIE Kathryn, CLAYTON Philip	
91	**SUCCESSFUL SIMULTANEOUS LIVER-KIDNEY TRANSPLANT WITH HIGH LEVEL DSA USING LOW-INTENSITY IMMUNOSUPPRESSIVE REGIMEN** HOWSON Prue, KULKARNI Hemant, CHAKERA Aron, LIM Wai H	
92	**IDENTIFICATION OF DONOR ANTI-HLA ANTIBODIES IN MULTIPLE TRANSPLANT RECIPIENTS** HIHO Steven, KUMMROW Megan, LEVVEY Bronwyn, SULLIVAN Lucy, WESTALL Glen, SNELL Greg	
93	**SURGICAL APPROACHES FOR MANAGING THE DIFFICULT ABDOMINAL WALL IN KIDNEY TRANSPLANT RECIPIENTS: CASE REPORTS** LAM Susanna, LAURENCE Jerome, VERRAN Deborah	
94	**USING DONOR BILE DUCT INJURY SCORES TO PREDICT BILIARY STRICTURES AFTER LIVER TRANSPLANTATION: RESULTS FROM THE AUSTRALIAN NATIONAL LIVER TRANSPLANTATION UNIT** LY Mark, PULITANO Carlo, MCKENZIE Catriona, KENCH James, CRAWFORD Michael	
95	**COMPLICATIONS RESULTING FROM THE USE OF A SURGICAL MESH PATCH IN PAEDIATRIC RENAL TRANSPLANT RECIPIENTS** DURKAN Anne, TAHER Amir, THOMAS Gordon, SHUN Albert	
96	**ATYPICAL HAEMOLYTIC URAEMIC SYNDROME POST-TRANSPLANTATION** SO Sarah, SPICER Timothy	

TSANZ ASM, Sydney July 28-30, 2019 Posters

Abstract	— *Poster* —	Drawing Room
97	**METABOLIC HEALTH POST LIVING KIDNEY DONATION** GUO Henry, MCGINN Stella, LI Yan	
98	**ASPERGILLUS PROSTATITIS FOLLOWING RENAL TRANSPLANTATION** HEPBURN Kirsten, ELIAS Anthony, KIM Dana, KOTSIOU George, MCGINN Stella	
99	**AN UNUSUAL CAUSE OF HEADACHE IN A RENAL TRANSPLANT PATIENT** HEPBURN Kirsten, KIM Dana, DARBAR Archie, POLLOCK Carol	
100	**CRYPTOCOCCUS INFECTION IN RENAL TRANSPLANT RECIPIENTS: A CASE CONTROL STUDY** ZAHOROWSKA Beata, CHEN Sharon CA, KABLE Kathryn, NANKIVELL Brian J	
101	**A CASE OF MALAKOPLAKIA MIMICKING MALIGNANCY IN A RENAL TRANSPLANT PATIENT** SRINIVASA Vinay, GOVINDARAJULU Sridevi	
102	**NOT JUST A SIMPLE LUMP** SCOTT Tahira, YAXLEY Julian	
103	**SUCCESSFUL KIDNEY TRANSPLANTATION OF A HEPATITIS B SURFACE ANTIGEN MUTANT POSITIVE DONOR TO A HEPATITIS B NEGATIVE RECIPIENT** WILSON Gregory, FRANCIS Ross	
104	**A RARE PRESENTATION OF CMV DISEASE IN A KIDNEY TRANSPLANT RECIPIENT** YAXLEY Julian, TITUS Thomas	
105	**SAFETY, TOLERABILITY AND EFFECT ON HIGH-SENSITIVITY CRP OF LOW-DOSE COLCHICINE IN KIDNEY TRANSPLANT RECIPIENTS** SORARU Jacqueline, WONG Germaine, THOMPSON Peter, JOYCE David, TEIXEIRA-PINTO Armando, LIM Wai H	
106	**CANCER TRANSMISSION FROM DECEASED ORGAN DONORS WITH PRIOR CANCERS** PHILLIPS Jessica, WONG Germaine, AU Eric, MCDONALD Stephen, CHAPMAN Jeremy, OPDAM Helen, MCDONALD Mark, PILMORE Helen, KANELLIS John, LIM Wai	
107	**POST TRANSPLANT LYMPHOPROLIFERATIVE DISEASE (PTLD) PRESENTING AS ENTEROCOLITIS IN A RENAL TRANSPLANT PATIENT** HEPBURN Kirsten, JAHAN Sadia, MARSH Bree1, FRANCIS Leo, JOHN George	

TSANZ ASM, Sydney July 28-30, 2019 Posters

Abstract	— *Poster* —	Drawing Room
108	**VALIDATION OF THE METROTICKET 2.0 MODEL FOR 5-YEAR HCC SPECIFIC SURVIVAL IN AN AUSTRALIAN LIVER TRANSPLANT COHORT** STOKLOSA Ted, SANTHAKUMAR Cositha, FERNANDES Brian, PERERA Nadia, HU Xinxin, TATIANA Tsoutsman, LIU Ken, MCCAUGHAN Geoffrey, STRASSER Simone, MAJUMDAR Avik	
109	**METASTATIC ROUND CELL SARCOMA IN A RENAL TRANSPLANT RECIPIENT** SHETTIGAR Reshma, PUTT Tracey, SCHOLLUM John, WALKER Robert	
110	**RAPIDLY PROGRESSIVE METASTATIC ADENOCARCINOMA OF UNKNOWN PRIMARY 2 MONTHS FOLLOWING DECEASED DONOR RENAL TRANSPLANTATION, RECIPIENT OR DONOR DERIVED–A CASE REPORT** MCMICHAEL Lachlan, CARROLL Robert	
111	**COLONIC MUCOSA-ASSOCIATED LYMPHOID TISSUE LYMPHOMA IN A KIDNEY TRANSPLANT RECIPIENT** MARTIN Kylie, TAYLOR Andrew, TAM Constantine, HILL Prue, MACHET David, GOODMAN David	
112	**YOUNG WOMAN WITH TYPE 1 DIABETES AND SEVERE SUB-CUTANEOUS SKIN REACTION TO INSULIN, BECOMES INSULIN INDEPENDENT AFTER ISLET TRANSPLANT** RADFORD Toni, DROGEMULLER Christopher, LOUDOVARIS Thomas, KAY Thomas, O'CONNELL Philip, HAWTHORNE Wayne, TORPY David, COATES TOBY	
113	**THE USE OF FREESTYLE LIBRE FLASH GLUCOSE MONITORING SYSTEM TO MONITOR BLOOD GLUCOSE LEVELS IN LARGE ANIMAL PORCINE MODEL** PENKO Daniella, NITSCHKE Jodie, JOHNSTON Julie, MATHEWS Loren, JOHNS Daniel, MUIRHEAD Robb, PULLAN Caitlin, DROGEMULLER Christopher, TORPY David, KUCHEL Timothy, COATES Patrick	
114	**REVISITING PANCREAS TRANSPLANTATION IN SOUTH AUSTRALIA** BHATTACHARJYA Shantanu, OLAKKENGIL Santosh, RUSSELL Christine, COATES Patrick Toby, RUSS Graeme	
115	**NORMOTHERMIC MACHINE PERFUSION OF DCD LIVERS – A TOOL THAT COULD HELP EXPAND THE DONOR POOL** REILING Janske, HODGKINSON Peter, COCO Tina, BUTLER Nicholas, FACWETT Jonathan	

TSANZ ASM, Sydney July 28-30, 2019 Posters

Abstract	— *Poster* —	Drawing Room
116	**EMPAGLIFLOZIN AS A SODIUM-HYDROGEN EXCHANGE INHIBITOR (NHEI) FOR DONOR HEART PRESERVATION** VILLANUEVA Jeanette, GAO Ling, SCHEUER Sarah, DOYLE Aoife, BOKKERS Kim, HICKS Mark, MACDONALD Peter	
117	**ASSESSMENT OF 3D-BIOPRINTED HUMAN REGULATORY T-CELLS FOR CO-PRINTING WITH HUMAN PANCREATIC ISLETS** KIM Juewan, HOPE Christopher M, PERKINS Griffith B, YUE Zhilian, LIU Xiao, DROGEMULLER Christopher J, CARROLL Robert P, BARRY Simon C, WALLACE Gordon G, COATES P Toby	
118	**IL-2 ALONE DOES NOT MAINTAIN ACTIVATED HUMAN CD4+CD25+CD127LOCD45RA FOXP3HI TREG POPULATION IN CULTURE** VERMA Niruapama D, LAM Andrew, CHIU Chris, ROBINSON Catherine M, TRAN Giang T, HODGKINSON Suzanne J, HALL Bruce M	
119	**INTERLEUKIN-6 RECEPTOR BLOCKADE IMPROVES SURVIVAL AND REDUCES GRAFT-VERSUS-HOST DISEASE IN HUMANISED MICE TREATED WITH POST-TRANSPLANT CYCLOPHOSPHAMIDE** MILES Nicole, ADHIKARY Sam, GERAGHTY Nicholas, SLUYTER Ronald, ALEXANDER Stephen, WATSON Debbie	
120	**LOW-DOSE INTERLEUKIN-2 INCREASES HUMAN REGULATORY T CELLS IN HUMANISED MICE WITH GRAFT-VERSUS-HOST DISEASE TREATED WITH POST-TRANSPLANT CYCLOPHOSPHAMIDE** ADHIKARY Sam, GERAGHTY Nicholas, SLUYTER Ronald, ALEXANDER Stephen, WATSON Debbie	
121	**CHARACTERISATION OF TISSUE-RESIDENT T CELLS BY TRANSPLANTATION** PROSSER Amy, HUANG Wen Hua, LIU Liu, DE BOER Bastiaan, WATSON Monalyssa, DART Sarah, LUCAS Andrew, JEFFREY Gary, DELRIVIERE Luc, KALLIES Axel, LUCAS Michaela	
122	**CXCR3 DEPLETION IN PROLONGING ISLET GRAFT SURVIVAL IN MOUSE XENOTRANSPLANTATION** NICHOLSON Leigh, HU Min, PILGRIM Suzanna, HAWTHORNE Wayne, GREY Shane, O'CONNELL Philip	

2019 ASM ACCEPTED ABSTRACTS

Abstract No. 1

RETROPERITONEAL LAPAROSCOPIC LIVING DONOR NEPHRECTOMY: 13-YEAR EXPERIENCE AT A SINGLE CENTRE

NGUYEN Thanh-Tuan[1,2], CHAU Quy Thuan[2], QUACH Do La[2], THAI Kinh Luan[1,2], VU Duc Huy[2], NGUYEN Duy Dien[2], TRAN Trong Tri[2], NGUYEN Trong Hien[2], HOANG Khac Chuan[2], NGO Xuan Thai[1,2], THAI Minh Sam[2,1], TRAN Ngoc Sinh[1]

[1]Urology, University of Medicine and Pharmacy at Ho Chi Minh City, [2]Urology, Cho Ray hospital

Aim: To assess safety and results of retroperitoneal laparoscopic living donor nephrectomy (RLLDN), we analyzed our single centre data over a 13-year period.

Methods: Data were collected prospectively from 505 donors undergoing RLLDN at our hospital from August 2005 to August 2018. All donors were approved by our Government hospital Renal Transplantation Council. Multiple surgeons used a standard and low cost surgical technique involving eventual donor kidney removal by hand through an 8cm skin crease incision made between two laparoscopic port sites. Patient demographics, radiology findings, surgery results and complications were recorded.

Results: Mean age was 47 ± 9 years and 53% were females. Mean BMI was 22.2 ± 3.1 (17.1- 35.4). There were 131 (26%) right kidneys and 374 (74%) left kidneys. Donor-recipient relationships were parent (43%), sibling (37%), aunt or uncle (7%), other relative (6%), spouse (4%) and unrelated friend (1%). Paired donation involved 1%. All donor nephrectomies were performed without conversion to open surgery. No intraoperative complications were recorded and no deaths occurred. Mean warm ischemic time was 4.5 ± 1.4 minutes. Postoperative complications were two cases (0.4%) of postoperative bleeding requiring surgical intervention. All the grafts functioned within the first hour after transplantation. Average donor hospitalization time was 4.8 ± 1.2 days. Mean serum creatinine of the donor was 1.2 ± 0.2 mg/dL at time of hospital discharge compared with 0.9 ± 0.5 mg/dL before nephrectomy.

Conclusion: Using standard instruments for laparoscopic surgery, RLLDN has proved to be safe, low-cost and reproducible technique in Vietnam.

Abstract No. 2

SINGLE VS DUAL (EN BLOC) KIDNEY TRANSPLANTS FROM SMALL PAEDIATRIC DONORS: AUSTRALIAN AND NEW ZEALAND (ANZ) DATA

EASTMENT Jacques[1], TAN Ai Lin[1], FRANCIS Ross[1], KANAGARAJAH Vijay[1], MUNN Stephen[2], GRIFFIN Anthony[1], JOHNSON David[1], PRESTON John[1], ISBEL Nikky[1]

[1]Renal Transplant Unit, Princess Alexandra Hospital, Brisbane, [2]New Zealand Liver Transplant Unit, Auckland City Hospital

Aims: The utilisation of kidneys from very young (≤5years) paediatric donors is complex. Concerns regarding provision of adequate nephron mass are balanced against the loss of utility in using two organs for a single patient. The aim of this study was to examine the patterns of practice in ANZ and to compare outcomes of adult recipients of single *vs* dual en-bloc paediatric grafts.

Methods: This was a retrospective cohort study using data from the ANZDATA registry. Data was included for all donors aged five years or younger and the corresponding adult recipients. The primary outcomes of interest were graft survival (death censored) and causes of early graft loss.

Results: Between 1963-2016, 208 patients received a kidney transplant from a deceased donor aged ≤ 5 years. Eight percent of donors were < 12m of age. The average weight of donors was 17kg (range: 7kg-33kg). The graft type is known for 154 patients - there were 91 single kidney transplants (SKTs) and 63 *en-bloc* kidney transplants (EBKTs). There were significantly more EBKTs performed after the year 2000 (p=0.0003) although donor weight and age remained constant. The mean ages of SKT donors and EBKT donors were 4.2 years (range: 1-5 years) and 2.0 years (range: 0-5 years), respectively (p<0.001). Donor weights were significantly lighter in the EBKT group (p = <0.001). SKT were more likely to be allocated to smaller recipients. Within the cohort of patients with a known graft type, there was an early (within 1 month) graft loss rate of 13%. Early graft loss was not more likely to occur in EBKT patients compared to SKT patients. There were six early thrombotic events causing graft loss (3 renal vein thromboses and 3 renal artery thromboses). Smaller donor weight was associated with an increased risk of graft failure secondary to vascular thrombosis that approached significance (p = 0.055). For grafts that survived the first month, long-term outcomes were excellent and not different between groups. Mean death censored graft half-life was not influenced by donor weight.

Conclusion: Small paediatric donors are associated with an increased risk of early graft loss from thrombosis which is influenced by weight of the donor, however the overall long term graft survival rates are excellent and independent of donor weight

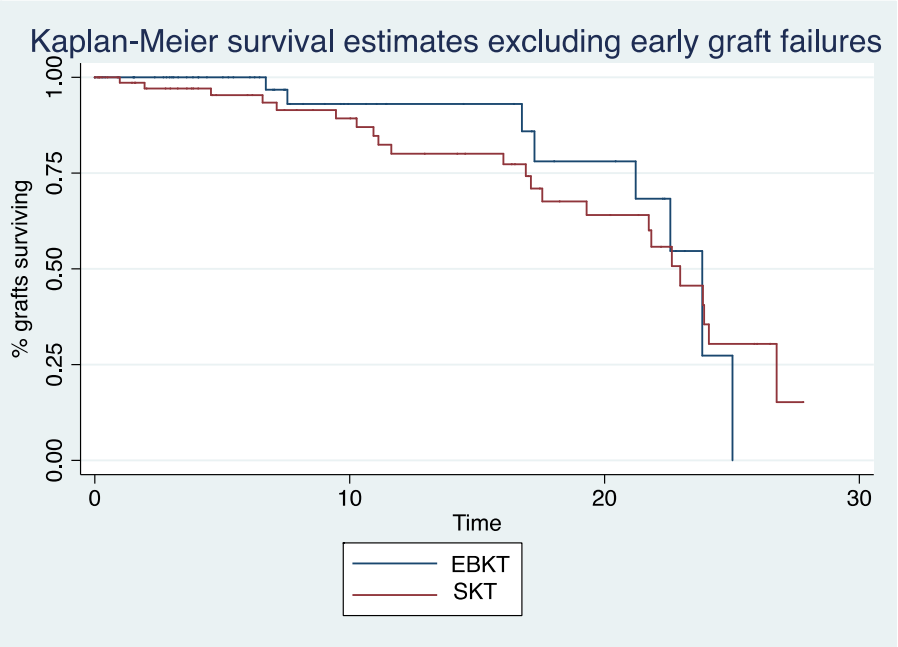


Figure 1: Graft survival rates for recipients of dual en bloc and single kidney grafts from small paediatric donors. Recipients who experienced graft failure within the first 28 days of transplant have been excluded.

Abstract No. 3

CAN AN EARLY BILE DUCT FLUSH DURING DONOR PROCUREMENT REDUCE BILIARY STRICTURES AFTER LIVER TRANSPLANTATION A RANDOMISED CLINICAL STUDY

LY Mark[1], PULITANO Carlo[2,3], CRAWFORD Michael[2]

[1]Royal Prince Alfred Hospital, Sydney, [2]Australian National Liver Transplantation Unit, Royal Prince Alfred Hospital, Sydney, [3]University of Sydney

Introduction: Experimental studies suggest bile salts play a role in bile duct injury and consequent biliary strictures (BS) in liver transplantation. Animal studies suggested that inadequate flush out of bile during cold ischemia is associated to bile duct injury. However, no clinical study has investigated the importance of early bile duct flush during organ procurement in determining BS. We performed a randomized clinical study to investigate whether an additional earlier bile duct flush reduces the incidence of BS after liver transplantation.

Methods: Brain Dead Donors retrieved and transplanted within NSW, Australia from March 2016 to June 2017 were randomised into two groups. The intervention group received bile duct flushes during cold perfusion and after donor hepatectomy. The control group received a bile duct flush after donor hepatectomy. The primary end point was 12 month incidence of BS in liver transplant recipients. Data was also extracted for donor and recipient characteristics and other post-operative complications.

Results: Sixty-four donors met inclusion criteria (Intervention (N=29), Control (N=35)). The overall incidence of BS was 23.4% (N=15) and there was no difference between groups for BS ($p>0.05$). There was also no difference between groups for Non Anastomotic Strictures (NAS) (6.9% vs 2.9%) or Anastomotic Strictures (20.7% vs 20%)($p>0.05$).

Conclusions: An additional earlier bile duct flush during donor cold perfusion did not reduce the incidence of biliary strictures at 1 year compared to a bile duct flush after hepatectomy alone.

Abstract No. 4

UPDATED SAFETY OUTCOMES AND COST-ANALYSIS OF EARLY URETERIC STENT REMOVAL IN PAEDIATRIC KIDNEY TRANSPLANTATION

TONG Marcus Chen Yee[1], LARKINS Nick[2], MINCHAM Christine[2], CROMPTON Charles[2], WILLIS Frank[2], HE Bulang[1]

[1]WA Liver & Kidney Transplant Service, Sir Charles Gairdner Hospital, Perth, [2]Department of Nephrology, Perth Children's Hospital

Introduction: The insertion of a double-J stent in renal transplantation reduces urological complications but is associated with the risk of urinary tract infection (UTI). Suturing stents to indwelling catheters (IDC) allows for early simultaneously removal (5 days) in contrast to late stent removal via cystoscopy 4-6 weeks post-operation. This study aims to audit the safety and cost of early stent removal amongst paediatric recipients.

Methods: A ten year retrospective review of all paediatric kidney transplantations in a single-centre performed between 2009 and 2019 was conducted. A total of 32 kidney transplants were performed in 31 recipients. The age ranges from 2 to 18 years (median 10 years). Data pertaining to patient demographics and urological complications was collected and analysed with independent samples T-test.

Results: In this cohort, 23 cases of early versus 9 cases of late stent removal were identified. There was one ureteric stenosis resolved by percutaneous balloon dilatation within the early stent removal group. There were 3 patients in each group who had UTI requiring antibiotic therapy – 5 hospital presentations and 0 admissions in the early group versus 10 presentations and 5 admissions in the late group. Early stent removal reduced the incidence of UTIs by 20% ($P=0.045$). The calculated cost saved by early removal of ureteric stent is $1752 AUD per case.

Conclusion: Early removal of ureteric stents with IDCs results in lower risk of UTIs in paediatric recipients. It has obvious cost-saving merits as it prevents a further day admission for cystoscopy under general anaesthesia.

Abstract No. 5

DEVELOPMENT AND IMPLEMENTATION OF AN ENHANCED RECOVERY AFTER SURGERY (ERAS) PROTOCOL IN KIDNEY TRANSPLANTATION

RANA Abdul Ahad[1], DIAS Brendan[1], OLAKKENGIL Santosh[2], PHILIP Clayton[2], COATES Toby[2], BHATTACHARJYA Shanthanu[2]

[1]Central Northern Adelaide Renal and Transplantation Service, Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [2]Central Northern Adelaide Renal and Transplantation Service,

Enhanced recovery after surgery (ERAS) protocols are multimodal perioperative care pathways designed to achieve early recovery after surgical procedures by maintaining pre-operative organ function and reducing the profound stress response following surgery. However, to date no ERAS protocol has been established in renal transplantation.

Methods: A standardized ERAS protocol was designed for the renal transplant recipients and implemented in July 2017. Data collected prospectively of recipients transplanted from July 2017 to December 2018 was compared to prospectively collected data of recipients who were transplanted prior to ERAS implementation from January 2016 to July 2017 from our renal database. Outcomes following renal transplantation in 100 consecutive recipients was compared to outcomes in 100 consecutive renal recipients in the pre-ERAS era.

Results: Table 1 demonstrates patient characteristics in two patient cohorts. Subgroup analysis revealed significantly higher delayed graft function rates in patients transplanted with DCD kidneys in the pre-ERAS group (80% vs 40%, p=0.009). The mean length of stay for patients on ERAS protocol was 5.35 ± 2.4. This was two days shorter than the mean length of stay for patients not on ERAS protocol (7.42 ± 1.7, $p<0.001$). More importantly 79% of the patients on ERAS protocol were discharged on post-operative day four.

Conclusions: An ERAS protocol for renal transplant patients is feasible. Data shows that implementation of an ERAS protocol in a renal transplant unit resulted in reduction in post-operative length of stay thereby reducing hospitalization costs.

Key Words

ERAS; Renal Transplantation; Clinical Pathways

Abstract No. 5 ctd

Table 1 – Baseline data of renal transplant patients included in the two groups

	Patients on ERAS N=100	Patients not on ERAS N=100	P value
Age (years), mean (SD)	51.4 (14.2)	53.1 (14.3)	0.409
Sex, no. (%)			
• Male	54 (54)	64 (64)	
• Female	46 (46)	36 (36)	
Co-morbidities, no. (%)			
• Diabetes			
• Hypertension			
• Coronary Artery Disease			
Previous Transplant, no. (%)	12 (12)	13 (13)	0.698
Previous intra-abdominal surgery, no. (%)	7 (7)	6 (6)	0.575
Length of Pre-transplant dialysis (months), mean (SD)	18.8 (7.2)	19.2 (6.5)	0.311
Donor details, no. (%)			
• Living donor	19 (19)	22 (22)	0.601
• Donor after cardiac death (DCD)	25 (25)	16 (16)	0.116
• Brain dead donor (BDD)	56 (56)	62 (62)	0.391
Delayed graft function, no. (%)	31 (31)	36 (36)	0.456
• Living Donor	2 (10.5)	1 (4.5)	0.476
• DCD	10 (40)	13 (81.3)	*0.009**
• BDD	19 (33.9)	22 (35.5)	0.861
Total Ischemic time (hours), mean (SD)	9.6 (4.5)	10.7 (6.4)	0.171
• Living Donor	4.7 (2.5)	3.7 (0.5)	0.092
• DCD	10.5 (3.9)	14.1 (5.5)	*0.022**
• BDD	10.9 (4.2)	12.4 (6.0)	0.147
Length of hospitalization (days), mean (SD)			
• Living Donor	5.42 (2.8)	7.5 (1.4)	*0.004*
• DCD	5.2 (1.8)	7.75 (1.1)	*<0.001*
• BDD	5.39 (2.4)	7.31 (1.4)	*<0.001*
Complications, no. (%)			
• Overall	12 (12)	11 (11)	0.769
• Clavien 1 & 2	4 (4)	5 (5)	0.542
• > Clavien 2	8 (8)	6 (6)	0.326

Abstract No. 6

WAITLISTING FOR KIDNEY TRANSPLANTATION IN AUSTRALIA: ESTIMATING THE DEMAND

CLAYTON Philip[1,2,3], GULYANI Aarti[1], SYPEK Matthew[1], JESUDASON Shilpa[1,2,3], MCDONALD Stephen[1,2,3]

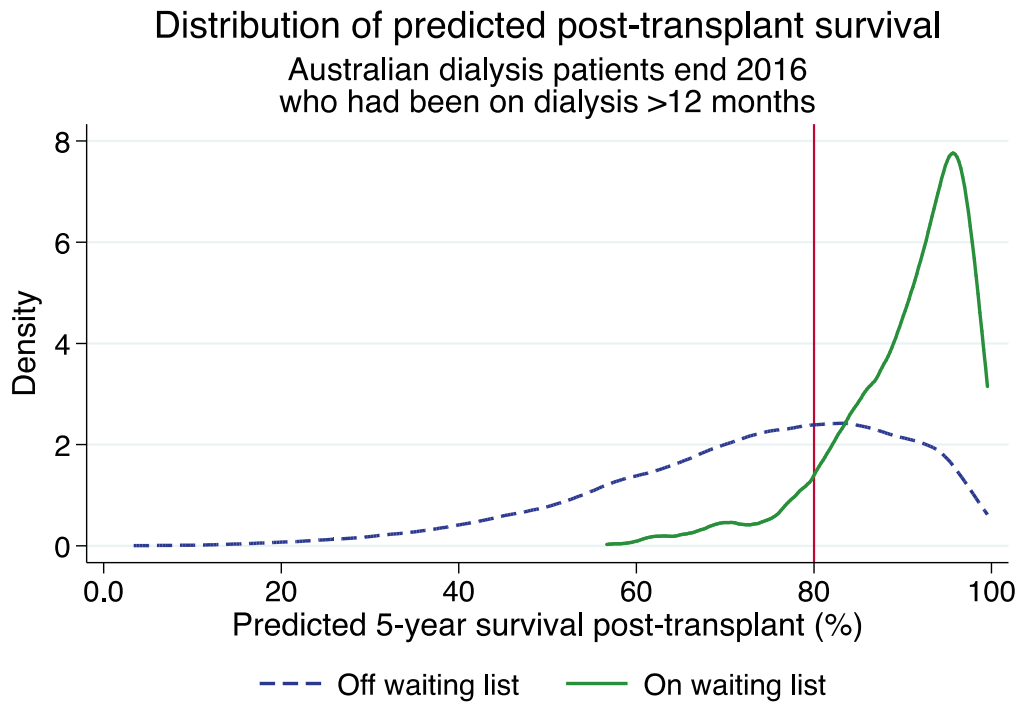
[1]ANZDATA, [2]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [3]School of Medicine, University of Adelaide

Aims. Fewer than 10% of Australian dialysis patients are wait-listed for transplantation. We examined the predicted 5-year post-transplant survival of prevalent Australian dialysis patients, and predictors of wait-listing in those with >=80% predicted survival.

Methods. Using data from the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry and National Organ Matching System, we included Australian patients who had been on dialysis for >=12 months at 31/12/2016. We used a Cox model developed in an earlier ANZDATA cohort to estimate 5-year survival post-transplant from an average deceased donor. In the subset of patients with predicted survival >=80%, we examined predictors of wait-listing using multilevel logistic regression.

Results. Of the 9,968 patients who met inclusion criteria, 323 (3%) were excluded due to missing data. 772 (8%) of the remaining 9,645 patients were wait-listed. 4,229 (44%) of patients had a predicted post-transplant survival of >=80%, of whom 696 (16%) were wait-listed. 76 patients with a predicted survival <80% were wait-listed, representing 11% of those on the waiting list (figure). Amongst patients with a predicted survival of >=80%, predictors of being listed included younger age, male sex, Asian ethnicity and prior transplantation. Predictors of not being listed included Indigenous status, obesity, all recorded comorbidities, previous cancer, and receiving dialysis in Western Australia or Queensland.

Conclusions. The majority of dialysis patients in Australia with predicted >=80% 5-year post-transplant survival are not wait-listed. Most but not all predictors of wait-listing are also predictors of post-transplant survival. Further study is needed to determine reasons for non-listing.



Abstract No. 7

LIVING KIDNEY DONOR PROFILE INDEX: A POOR PREDICTOR OF OUTCOMES

IRISH Georgina L[1,2,3], CHADBAN Steve[1,4,5], BOUDVILLE Neil[6,7], CAMPBELL Scott[8,9], KANELLIS John[10,11], CLAYTON Philip A[1,2,3]

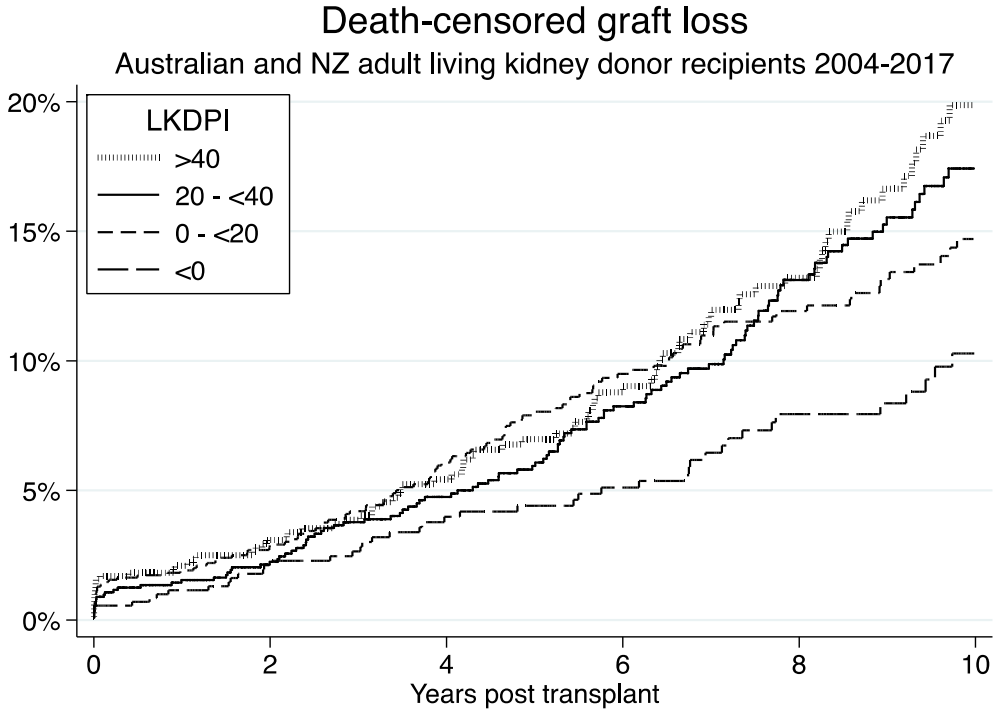
[1]ANZDATA registry, South Australian Health and Medical Research Institute (SAHMRI), [2]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [3]Department of Medicine, University of Adelaide, [4]Renal Medicine, Royal Prince Alfred Hospital, Sydney, [5]Kidney Node, Charles Perkins Centre, University of Sydney, [6]Medical School, University of Western Australia, Perth, [7]Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth, [8]Department of Nephrology, Princess Alexandra Hospital, Brisbane, [9]School of Medicine, University of Queensland, Brisbane, [10]Department of Nephrology, Monash Health, [11]Centre for Inflammatory Diseases, Monash University, Melbourne

Aim Risk scores can aid risk quantification and decision-making in kidney transplantation. The Living Kidney Donor Profile Index (LKDPI), developed in the US, has not been validated in Australia/NZ. We examined its performance in Australian/NZ kidney transplant recipients.

Methods Using data from the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry, we included adult recipients of kidney-only transplants over 2004-2017. Outcomes were overall and death-censored graft survival, and patient survival. For each outcome we constructed Cox models including: 1) LKDPI, 2) recipient factors, 3) LKDPI plus recipient factors. To allow comparison with deceased donor (DD) kidneys we rescaled the LKDPI to its DD KDPI equivalent, created prognosis-matched cohorts of living and DD kidney recipients, and examined the discrimination of the integrated KDPI in matched pairs. For each model Harrell's C-statistic (C) was used to determine discrimination.

Results 3826 live donor (LD) and 7618 DD recipients were included. The LKDPI's predictive ability was poor for all LD outcomes (C-statistics 0.54, 0.54 and 0.55 for graft survival, death-censored graft survival (figure) and patient survival respectively in LKDPI-only models). Adding the LKDPI to recipient-only models added minimal discrimination (changes in C +0.01, 0.00, 0.00). In the LD/DD matched analyses, C-statistics were similarly low (0.58, 0.58, 0.57).

Conclusion The LKDPI showed poor ability to discriminate the outcomes of different LD kidneys, and discrimination of outcomes between LD and DD based on the integrated KDPI was also poor. Choosing between kidney donors on the basis of the LKDPI should be done with caution.



Abstract No. 8

OVERALL GRAFT AND PATIENT SURVIVAL AMONGST ELDERLY TRANSPLANT RECIPIENTS

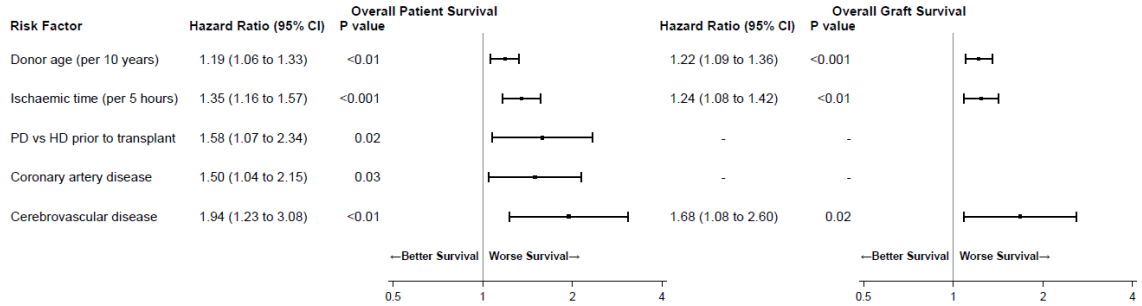
SO Sarah[1], AU Eric HK[2,3], LEE VWS[1,4], WONG Germaine[1,2,3]

[1]Renal & Transplantation Unit, Westmead Hospital, Sydney, [2]School of Public Health, University of Sydney, [3]Centre for Kidney Research, The Children's Hospital at Westmead, Sydney, [4]University of Sydney

Aims: To determine the association between recipient and donor factors and clinical outcomes including patient and graft survival in older kidney transplant recipients.

Method: We included all patients aged ≥65 years who received their first kidney transplants between June 2006 and December 2016. Multivariable Cox regression modelling was used to determine factors associated with all-cause death, death with functioning graft, and graft survival.

Results: Of 1324 candidates between June 2006 and December 2016, 802 older patients received their first kidney transplant. Of these, 531 (66.2%) were male, 705 (87.9%) received deceased donor grafts, and the median age at transplantation was 68 (Interquartile range: 66–69) years. Over a follow-up time of 2706.9 patient-years, 136 patients died (111 with functioning graft) and 51 lost their allografts. One and five-years (%)(95%) overall patient survivals, survivals with functioning grafts, overall graft and death-censored survivals were: [95.1 (93.5-96.7) and 79 (75.1-82.9)]; [95.7 (94.3-97.1) and 82.4 (78.7-86.1)]; [92.9 (91.1-94.7) and 75.4 (71.3-79.5)]; [96.8 (95.4-98.2) and 92 (89.8-94.2)], respectively. Factors associated with all-cause death included increasing donor age, total ischemic time, prior history of coronary and cerebrovascular disease and peritoneal dialysis as treatment modality prior to transplantation. Proportion of time off waitlisting was not associated with adverse patient and graft outcomes [adjusted HR (95%)] [1.51 (0.89–2.55)] and [1.01 (0.62–1.64)].



Conclusions: In this selected cohort of elderly transplant recipients, patient and graft survivals exceed 75% five years post-transplant. Recipient factors associated with all-cause death include coronary artery disease, cerebrovascular disease or peritoneal dialysis at transplantation.

Abstract No. 9

LIVE KIDNEY DONATION FOR TYPE 2 DIABETIC (T2D) RECIPIENTS. IS THERE A BENEFIT COMPARED TO DECEASED DONATION?

GOODMAN David[1], ULLAH Shahid[2,3], MCDONALD Stephen P[2,3]

[1]Department of Nephrology, St Vincent's Hospital, Melbourne, [2]Central Northern Adelaide Renal and Transplantation Service, University of Adelaide, [3]ANZDATA

Aims Evaluate the benefits of live versus deceased donor kidney transplantation in T2D and non-T2D recipients on patient survival

Methods Data for all adult first kidney transplant recipients from 2005-2014 was extracted from ANZDATA registry. Donor source, age, gender and dialysis duration were included. A cox proportional hazard model was used to predict 5-year graft survival.

Results Of 7010 kidney transplants 15% of recipients were T2D. Live donors comprised 41% of non-T2D and 25% T2D with pre-emptive transplantation in non-T2D 32% and T2D 29%. In non T2D recipient 5-year patient survival was greater for live compared to deceased donors (HR 1.46, 95%CI 1.07-2.00, p=0.02). This was not seen in T2D (HR 1.19, 95% CI 0.68-2.09, p= 0.54). There was no significant interaction between DM and donor type. When recipients were grouped according to dialysis duration pre-emptive transplantation was beneficial to T2D recipients, but the Hazard ratios climbs after the commencement of dialysis (Table). In contrast, non-T2D recipients receiving a live donor transplant within the 1st 12 months of dialysis had the same survival as pre-emptive transplantation.

Conclusion Pre-emptive live kidney transplantation for T2D recipients improves patient survival but the benefit is lost after commencement of dialysis. In non-T2D, live donor kidney transplantation is beneficial both before and after commencement of dialysis. This data highlights the importance of evaluating patients prior to commencement of dialysis and aiming for pre-emptive transplantation particularly in T2D recipients. In T2D with extended dialysis time, medical co-morbidities may negate the benefits of live compared to deceased donor kidney transplantation.

Table: Patient survival: Diabetic status and renal replacement therapy duration for live donor recipients

RRT duration	**T2D** n=262			**Non-T2D** n=2438		
	Hazard ratio	CI	p	Hazard ratio	CI	p
Pre-emptive	1			1		
<1 year	4.79	0.993-23.0	0.051	1.03	0.53-1.97	0.935
1-5 years	6.64	1.55-28.4	0.011	2.81	1.68-4.69	0.000
>5years	2.30	0.21-25.4	0.496	3.07	1.34-7.01	0.008

Abstract No. 10

EXTERNAL VALIDATION OF AUSTRALIA AND NEW ZEALAND TYPE 2 DIABETES (T2D) KIDNEY TRANSPLANT RISK CALCULATOR (KTRC) WITH FRENCH DATABASE AND COMPARISON TO ESTIMATED POST TRANSPLANT SURVIVAL SCORE (EPTS).

GOODMAN David[1], ULLAH Shahid[2,3], MCDONALD Stephen P[2,3]

[1]Department of Nephrology, St Vincent's Hospital, Melbourne, [2]Central Northern Adelaide Renal and Transplantation Service, University of Adelaide, [3]ANZDATA

Aims Externally validate T2D kidney transplant risk calculator (KTRC) using large French database and compare predictive ability of KTRC with EPTS.

Methods Data for all first adult kidney transplants from 2005-2015 was extracted from the French Renal Epidemiology and Information Network (REIN) registry. Age, gender, BMI, smoking, history of coronary artery disease, cerebrovascular disease, peripheral vascular disease, dialysis duration, donor source and HLA matching was included. The 5-year graft and patient survival (G/PS) was calculated using the previous described 13 variable T2D-KTRC. Cox proportional hazard model was used to predict 5-year G/PS. Goodness of fit was evaluated by receiver operator characteristics (ROC) and C-statistic. EPTS score was calculated from patient cohort utilized to derive KTRC.

Results Data from 2480 French renal transplant recipients with T2D was evaluated. using KTRC and C-statistic calculated (Table). We also removed indigenous patients from Australian cohort to allow a more direct comparison with French cohort. A similar C statistic was seen with French data models (0.66-v-0.64). When Australian and French cohort KTRC scores were compared to EPTS the c-statistic was lower for EPTS in each group, consistent with lower predictive ability (Table).

Conclusion We have validated KTRC using data from a large external cohort of T2D transplant recipients and demonstrated similar predictive ability. When compared to EPTS, KTRC had better predictive ability. A calculator with improved predictive ability is particularly important to ensure equitable allocation of kidneys in countries where organ allocation agencies utilize recipient risk calculators to allocate kidneys.

Table. C statistics – graft failure

Cohort	EPTS	KTRC
Australia	0.5728 (0.55122-0.59429)	0.6463 (0.62572-0.66686)
T2DM Australia	0.5406 (0.49423-0.58696)	0.6732 (0.62837-0.71808)
T2DM Australian Non-Indigenous	0.5844 (0.53341-0.63530)	0.6633 (0.61411-0.71257)
France	0.5525 (0.52091-0.58417)	0.6383 (0.60279-0.67383)

Abstract No. 11

A WINDOW TO TOLERANCE - HUMAN LUNG ALLOGRAFTS ARE ENRICHED FOR CD39+FOXP3+ REGULATORY T CELLS

DE SILVA Tharushi[1,2], O'SULLIVAN Brendan[1,2,3], APTE Simon[2,3], VOISEY Joanne[1], DIVITHOTAWELA Chandima[2], TAN Maxine[3,2], HOPKINS Peter[4,3], CHAMBERS Daniel[2,3]

[1]School of Biomedical Sciences, Queensland University of Technology, Brisbane, [2]Lung Transplant Service, Prince Charles Hospital, Brisbane, [3]School of Clinical Medicine, University of Queensland, Brisbane, [4]Prince Charles Hospital, Brisbane

Aims: Regulatory T cells (Tregs) play a vital role in the induction and maintenance of transplant tolerance. Though suppressive pathways of Tregs are well defined, identifying phenotypes with respect to local tissue environment is of importance to understand their role in graft tolerance. Adenosine triphosphate (ATP) is released from apoptotic, necrotic cells and from inflammatory cells and platelets during inflammation and infection. The ecto-enzyme CD39 hydrolyses ATP to immunosuppressive adenosine which functions to limit effector T cell proliferation and enhance suppressive function of Tregs. Since inflammation is associated with poor outcome in transplant recipients, we compared the proportion of blood and lung CD39+Treg in T cells with the aim of determining if immunosuppressive CD39+Tregs are present in the lung post-transplant.
Methods: Blood and bronchoalveolar lavage (BAL) cells isolated from 23 transplant patients (median 13.07 (1-140) months post-transplant; 11 female; 12 male; 13 CF; 4 IPF; 5 COPD; 1 Histiocytsis). These cells were analysed by a newly developed multicolour flow cytometry panel comprising 14 fluorescent antibody markers to identify Treg subsets.
Results: CD39+ FoxP3+ Treg were more prevalent in BAL than in blood (2.13 ± 0.58 (SEM) vs 0.69 ± 0.17 (SEM) of total CD4+ T cells, p=0.02(Figure 1A)). There was no correlation between BAL and blood CD39+ FoxP3+ Treg (r= -0.059, p= 0.788 (Figure 1B)).
Conclusions: The lung allograft is enriched with a specialized subpopulation of $FOXP3^{+}CD39^{+}$ Tregs with capability of metabolising pro-inflammatory ATP to enrich the lung microenvironment with immunosuppressive adenosine. Future work will examine the frequency and functional capacity of these cells.

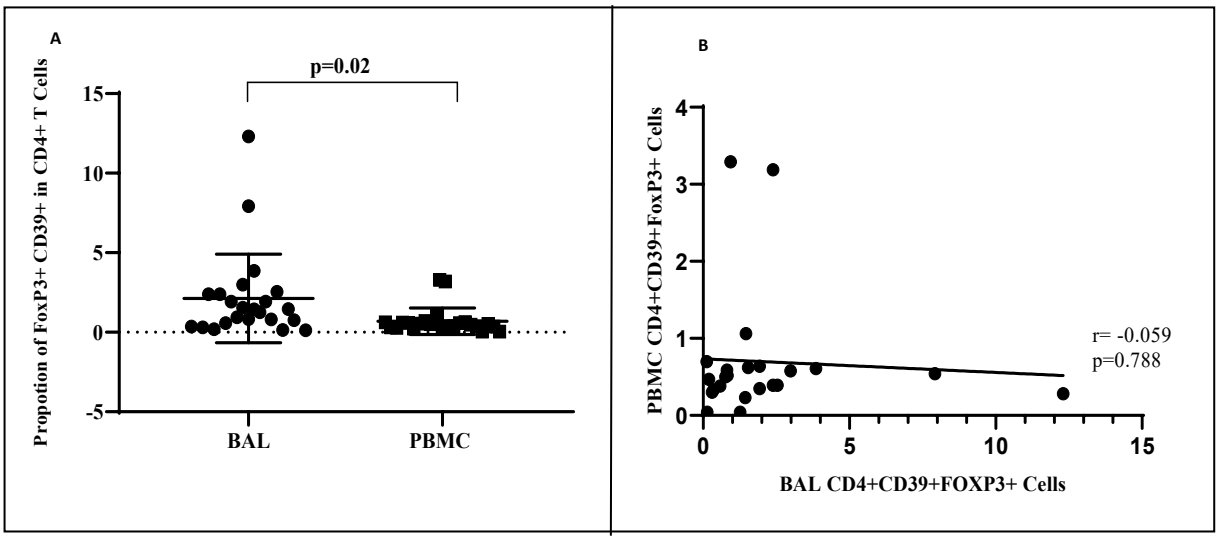


Figure 1 A: Proportion of CD39+FOXP3+ Tregs in BAL and PBMC. B: Correlation between CD4+CD39+FOXP3+ Cells in blood and BAL

Abstract No. 12

DEVELOPING A MODEL OF KIDNEY DIRECTED T CELL THERAPY USING CAR T CELLS

LU B[1], ZHANG GY[2], HU M[3], ROBINSON S[2], WILARUS A[2], WAN H[2], ALEXANDER SI[2], WANG YM[2]

[1]Centre for Transplant and Renal Research, Westmead Hospital, Sydney, [2]Centre for Kidney Research, The Children's Hospital at Westmead, Sydney, [3]Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney

Abstract: Modulation of T cell function has been a major component of solid organ transplantation; initially using immunosuppression, but more recently using regulatory cells including Tregs. This has now reached clinical use with the One Study in Europe. Increasing the specificity of Tregs to the target organ has major advantages in improving efficacy. This can be achieved by directing T cells to a transplant using T cell receptors constructed to target HLA.

Aim: To target CAR T cells to HLA-A2 expressed on donor tissues in a mouse model of transplantation.

Methods: An MIGR-1 retrovirus containing a scFv targeted at HLA-A2 and the signalling component of the TCR were transfected into CD4 lymphocytes purified from splenocytes of B6 mice. Skin and kidney transplants from B6-HLA2 tg mice were transplanted into immunodeficient RAG mice recipients. The kidney transplants were done as orthotopic transplants with one recipient kidney removed and the other left in place.

Results: CD4 T cells were successfully transfected with an HLA-A2 construct. Immunohistochemistry of D8 skin-grafts identified HLA-2 scFv transfected T cells in HLA-A2 transgenic skin-grafts but not in third party BALB/c skin grafts and control CD4 T cells were not found in either graft but both were present in spleen.

Conclusions: CD4 T cells expressing a CAR construct containing a scFV targeted at HLA-A2 preferentially target tissues expressing HLA-A2 suggesting that Class I can be used as a transplant organ and tissue targeting strategy.

Abstract No. 13

HUMAN CD27+HLA-DR+ MEMORY LIKE TREG SHOW XENOANTIGEN SPECIFIC SUPPRESSION OF PORCINE ISLET XENOGRAFT REJECTION IN HUMANIZED MICE

XIAOQIAN Ma[1,2], HU Min[1], CAO Lu[1,2], ZHAO Yuanfei[1], HUANG Dandan[1], BURNS Heather[1], HAWTHORNE Wayne[1], YI Shounan[1], O'CONNELL Phillip[1]

[1]Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney, [2]Institute for cell transplantation and gene therapy, The Third Xiangya Hospital of Central South University

Aims: To achieve effective suppression and avoid opportunistic infection and malignancy for clinical xenotransplantation, xenoantigen-specific Treg will be required. Previously, we have shown that a CD27+HLA-DR+subset of Treg separated from xenoantigen stimulated human Treg (XnTreg) were potent and xenoantigen-specific in vitro. In this study, we aim to determine their xenoantigen specificity and potency in protecting against neonatal porcine islet cell clusters (NICC) xenograft rejection in vivo.

Methods: Human XnTreg were separated by cell sorting, using the Treg cell surface markers CD27 and HLADR, into non-selected, CD27+HLADR+ and non-CD27+HLADR+ Treg subsets prior to cotransfer into NICC recipient NOD-SCID IL2rg-/- mice in association with autologous PBMC at a 1:25 ratio of Treg ($4x10^5$): PBMC ($1x10^7$). Serum, spleen and NICC xenografts were harvested from recipient mice at day 60 after human cell transfer for analysis of xenograft survival and Treg in vivo function.

Results: Recipient mice transferred with human PBMC alone rejected their xenografts completely within 35 days. Co-transfer with CD27+HLADR+ Treg prolonged NICC xenograft survival beyond 60 days with detectable serum porcine C-peptide and intact xenografts which stained positive for insulin and were surrounded but not infiltrated by a few human CD8+ effector cells. By contrast, non-selected and non-CD27+HLADR+ Treg co-transferred at the same ratio did not protect against rejection.

Conclusions: Human CD27+HLADR+ memory-like Treg were sufficient to suppress porcine islet xenograft rejection at a 5-fold decreased cell number compared to that previously reported for polyclonal Treg in same model, suggesting they are more potent at preventing rejection than polyclonal Treg in vivo.

Abstract No. 14

THE IMMUNOSUPPRESSIVE AGENT DILIXIMAB (ANTI-CD2) IS SUPERIOR TO ANTITHYMOCYTE GLOBULIN IN HAVING REDUCED PROTHROMBOTIC AND CELL-ACTIVATING EFFECTS

BONGONI Anjan K[1], SALVARIS Evelyn J[1], LEW Andrew M[2], COWAN Peter J[1,3]

[1]Immunology Research Centre, St Vincent's Hospital, Melbourne, [2]Walter and Eliza Hall Institute, Parkville, [3]Department of Medicine, University of Melbourne

Background: Antithymocyte globulin (ATG) is used to deplete T cells in transplantation. Although ATG is highly effective, its non-specific polyclonal composition can cause adverse side effects including systemic coagulation. We have previously developed a chimeric anti-CD2 monoclonal antibody (diliximab), which depletes and blocks costimulation of T cells, as a potential alternative to ATG.

Aims: To compare the prothrombotic and cell-activating effects of diliximab and ATG on human monocytes (THP-1 cell line) and peripheral blood mononuclear cells (PBMCs).

Methods: THP-1 cells ($2x10^6$) were treated with diliximab or ATG (200µg/ml) and incubated with 100% normal human plasma in the presence or absence of the complement inhibitor compstatin (10µg/ml). Surface procoagulant tissue factor (TF) activity was measured using a chromogenic factor (F)Xa generation assay. Whole blood and PBMCs were treated with diliximab or ATG (500µg/ml, 24 hrs) and cytokine secretion was analysed by ELISA.

Results: Diliximab had a small effect on monocyte TF procoagulant activity, measured by FXa generation (2.0±0.02-fold increase versus untreated). In contrast, ATG markedly increased FXa generation (20.0±0.7-fold increase). ATG's prothrombotic effect, which has been shown to be complement-dependent, was completely inhibited by compstatin. Diliximab treatment of whole blood or PBMCs showed minimal supernatant cytokine concentrations that were similar to isotype antibody treatment. However, ATG significantly increased cytokine levels (Table 1).

Conclusion: Our data show that diliximab lacks the prothrombotic and cell-activating side effects of ATG, and warrant its further development for the prevention and treatment of T cell-mediated rejection in transplantation.

	Whole blood			
	Rabbit IgG (ATG isotype control)	ATG	Human IgG3 (diliximab isotype control)	Diliximab
IFN-γ (pg/ml)	17.2 ± 0.2	85.8 ± 2.6	17.8 ± 1.3	22.9 ± 0.2
IL-2 (pg/ml)	1.0 ± 0.2	13.5 ± 4.5	1.2 ± 0.1	1.3 ± 0.5
TNF-α (pg/ml)	21.4 ± 1.0	53.4 ± 4.3	19.7 ± 1.2	25.9 ± 0.9
IL-1ra (pg/ml)	3034.0 ± 155.3	9349.0 ± 362.4	3219.0 ± 99.2	3655.0 ± 172.6
MCP-1 (pg/ml)	1237.0 ± 146.7	3104.0 ± 226.7	1064.0 ± 400.0	1464.0 ± 186.7
	PBMCs (2×10^6)			
	Rabbit IgG (ATG isotype control)	ATG	Human IgG3 (diliximab isotype control)	Diliximab
IFN-γ (pg/ml)	2.2 ± 0.5	50.4 ± 2.8	1.7 ± 0.3	3.3 ± 0.2
IL-2 (pg/ml)	1.1 ± 0.3	69.8 ± 4.0	1.8 ± 0.1	2.4 ± 1.1
TNF-α (pg/ml)	20.1 ± 3.6	77.5 ± 2.3	20.4 ± 2.5	29.6 ± 0.3
IL-1ra(pg/ml)	2331.0 ± 120.0	5517.0 ± 853.3	2091.0 ± 40.0	2451.0 ± 266.7
MCP-1 (pg/ml)	4393.0 ± 116.5	13443.0 ± 893.0	4522.0 ± 128.1	5044.0 ± 284.7

Table 1: Cytokine concentrations in whole blood or PBMCs stimulated with diliximab or ATG

Abstract No. 15

DONOR SOURCE OF KIDNEY TRANSPLANTATION IN NEW ZEALAND BY ETHNICITY: A LONGITUDINAL COHORT STUDY

CROSS Nicholas[1], DONNELLAN Sine[1], WILLIMAN Jonathon[2], PALMER Suetonia[1,3]

[1]Department of Nephrology, Christchurch Hospital, New Zealand, [2]Biostatistics and Computational Biology Unit, University of Otago, Christchurch, New Zealand, [3]Department of Medicine, University of Otago, Christchurch, New Zealand

Aims: To explore whether donor source for kidney transplantation in New Zealand was associated with recipient ethnicity adjusting for socioeconomic and clinical factors.

Methods: We performed a longitudinal cohort study in patients ≥18 years who commenced RRT in New Zealand between 2006-2015, using ANZDATA. Deprivation score was obtained by data linkage with the National Health Index. Poisson regression was performed for pre-emptive transplantation and competing risks regression for living and deceased donor transplantation with 95% CI. Estimates were adjusted for age, sex, smoking, deprivation, BMI, late referral, treating centre, diabetes, and coronary artery disease.

Results: Among the 5106 participants, 822 received a kidney only transplant. Compared to European patients, Māori and Pacific patients were younger, and more frequently had diabetes and referred late to specialist care, and lived in more socioeconomically deprived areas. In European patients, 65% received a live donor kidney transplant, while the proportion was smaller for Asian (44%), Māori (44%), and Pacific (39%) patients. Compared to European patients, patients who identified as Māori, Pacific and Asian were markedly less likely to receive a pre-emptive and living donor kidney transplant after adjustment for socioeconomic factors, comorbidity, and late referral(Table 1). The difference in transplantation rates between patient groups based on ethnicity was less marked for deceased donor kidney transplantation and was not evident for Māori and Asian patients after adjustment.

Conclusion: Transplantation rates for pre-emptive and live donor but not deceased donor kidneys varies with ethnicity, socioeconomic factors and late referral to specialist services within New Zealand.

Variable	Pre-emptive Adjusted IRR (95% CI)	Living donor Adjusted SHR (95% CI)	Deceased donor Adjusted SHR (95% CI)
Maori (ref: European)	0.34(0.18-0.64)	0.40(0.28-0.56)	0.72(0.50-1.03)
Pacific (ref: European)	0.09 (0.02-0.36)	0.24 (0.12-0.41)	0.60 (0.38-0.96)
Asian (ref: European)	0.33 (0.16-0.68)	0.50 (0.32-0.78)	1.13 (0.79-1.61)
NZdep13 deciles 9-10 (ref: deciles 1-5)	0.45 (0.28-0.74)	0.56 (0.41-0.78)	0.76 (0.54-1.06)
Current smoker(ref: nonsmoker)	0.30 (0.15-0.59)	0.37 (0.24-0.55)	0.57 (0.39-0.85)
Late referral (ref: not late)	n/a	0.66 (0.49-0.88)	0.76 (0.58-1.02)
Diabetes (ref: no diabetes)	0.26(0.14-0.47)	0.30 (0.22-0.42)	0.28 (0.20-0.38)
Coronary artery disease (ref: none)	0.35(0.19-0.65)	0.41 (0.28-0.62)	0.33 (0.22-0.49)

Abstract No. 16

OFFER DECLINES HAVE INCREASED FOR HIGHER RISK KIDNEYS IN SINCE KIDNEY DONOR PERFORMANCE INDEX (KDPI) REPORTING WAS INTRODUCED IN AUSTRALIA.

SYPEK MP[1,2,3,4], HUGHES P[2,3], MCDONALD S[5,6,7], CLAYTON P[5,6,7]

[1]Empty, ANZDATA, [2]Nephrology and Renal Transplant, Melbourne Health, [3]Faculty of Medicine, Denistry and Health Sciences, University of Melbourne, [4]Nephrology and Renal Transplant, Royal Children's Hospital, Melbourne, [5]ANZDATA, [6]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [7]Department of Medicine, University of Adelaide

Background: Concerns have been raised that reporting of KDPI with all deceased donor kidney offers since November 2016 may have resulted in a change of acceptance behaviour. We aimed to determine if the number of offer declines has increased for higher risk kidneys following KDPI reporting.

Methods: All kidney only transplants in Australia between 2015 and 2018 were included. Offer declines were defined as patients ranked higher than the actual recipient who were not transplanted, with positive CDC T-cell crossmatches excluded. Multi-level negative binomial regression was used to explore the association between KDPI reporting and number of declines in higher risk kidneys (KDPI≥80%) and non-high risk kidneys (KDPI 0-79%). Models were sequentially adjusted for donor factors and monthly region specific kidney transplant waiting list numbers.

Results: 2,987 deceased donor kidney only transplants were performed during the study period, of which 631(21%) had a KDPI ≥80%. Figure 1 shows the trends in offer declines over time. For higher risk kidneys the era of KDPI reporting was associated with an increased number of declines per kidney (IRR 1.40, 95%CI 1.09-1.80, p=0.009) which persisted after adjustment for KDRI, individual donor factors and monthly regional waiting lists. There was no increase in offer declines for non-high risk kidneys (p=0.211).

Conclusion: The era of KPDI reporting has been associated with an increase in kidney offer declines for higher risk but not for non-high risk kidneys in Australia. Further analysis of recipient characteristics and outcomes is required to see if this represents more effective use of these organs.

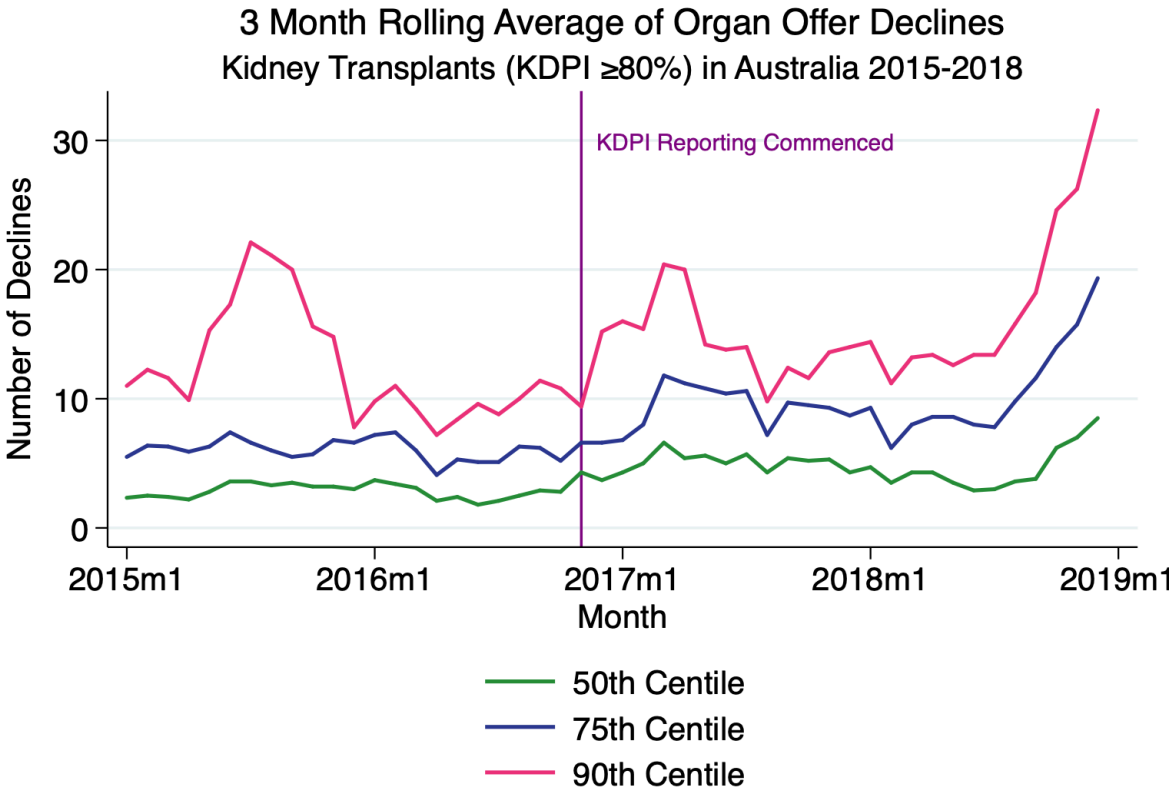


Figure 1 shows the 3 month rolling average for the 50th, 75th and 90th centiles of offer declines for all kidneys with KDPI ≥ 80% transplanted in Australia 2015-2018 (multi-organ transplants excluded).

Abstract No. 17

THE CHANGING PATTERN OF LIVING KIDNEY DONATION IN AUSTRALIA AND NEW ZEALAND

ALLEN Richard[1], DOBRIJEVIC Ellen[2], CLAYTON Phil[3,4], WONG Germaine[5], CROSS Nicholas[6], ROAKE Justin[7], VASILARAS Arthur[8], PLEASS Henry[9,10]

[1]Sydney Medical School, University of Sydney, [2]Clinical Westmead Hospital, University of Sydney, [3]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [4]ANZDATA Registry, Royal Adelaide Hospital, [5]Centre for Transplant and Renal Research, Westmead Hospital, Sydney, [6]Department of Renal Medicine, Christchurch Hospital, NZ, [7]Department of Surgery, Christchurch Hospital, NZ, [8]Transplantation Services, Royal Prince Alfred Hospital, Sydney, [9]Department of Surgery, Westmead Hospital, Sydney, [10]Department of Surgery, University of Sydney

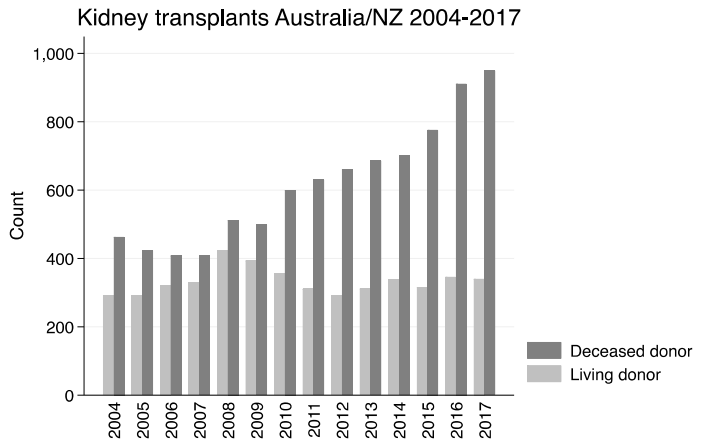
Living donor nephrectomy (LDN) surgery is a procedure of necessity when deceased donation (DD) of kidneys is insufficient to meet transplant demand.

Aim: Assess patterns of LDN surgery technique and activity and to correlate with DD activity.

Methods: Analysis of data contributed annually by 25 centres to the ANZDATA Living Kidney Donor Registry, from establishment in 2004, to 2017.

Results: 4,644 LD kidneys were transplanted into 4287 adults and 377 children. Two (0.044%) LDN related deaths occurred. Paired kidney exchange (PKE) countered for 281(6%) of LDK procedures. LDN side data was missing for 74 donors (1.6%). Left kidneys was donated in 3,777(82.6%) LDN procedures and 813 were right sided. Five centres have avoided right-sided LDN surgery. Open LDN has declined from 36% in 2004 to 4% by 2011. By 2017, there were 18 transplant centres and 61% of LDN were performed by pure laparoscopic surgery and 36% by hand-assisted surgery. LDN activity peaked in 2008(n=386). Despite introduction of PKE in 2009, LDN activity fell and plateaued from 2011. LDs provided 24.6% and 36.9% of kidneys transplanted in Australia and New Zealand in 2017 respectively.

Conclusions: Two LDN related donor deaths highlight risks undertaken by living kidney donors. Importantly, the necessity for living kidney donation has diminished. Despite introduction of PKE and minimally invasive LDN surgery, LDN activity continues to fall as a percentage of annual number of kidney transplant recipients. PKE may however have contributed to a reduction of transplant centres because of their need to undertake right-sided LDN surgery.



Abstract No. 18

COLD PERFUSION MACHINE ALLOWS PROLONGED STORAGE OF DONOR MATCHED KIDNEYS WITHOUT ADVERSE OUTCOME

BYRNE Sarah[1], PEREIRA Ryan[1], ROBERTSON Ian[2], TAN Ai Lin[1], LOCKWOOD David[1], KANAGARAJAH Vijay[1], RAY Mark[1], PRESTON John[1], WOOD Simon[1], LAWSON Malcolm[1], GRIFFIN Anthony[1], RHEE Handoo[1]

[1]Department of Surgery, Princess Alexandra Hospital, Brisbane, [2]Renal Transplant Unit, Princess Alexandra Hospital, Brisbane

Aims: To compare outcomes of hypothermic perfusion storage and static hypothermic storage of renal allografts at the Princess Alexandra Hospital (PAH) since the procurement of cold perfusion machines (CPM).

Methods: All deceased donor renal transplants occurring at the PAH from 2011 to 2017 were included. Data reports from the CPM parameters were analysed. Outcome data was obtained from medical records and statistical analyses were performed. Primary outcome was delayed graft function (DGF).

Results: During this period, 1136 renal transplants occurred and the CPM was used on 109 occasions. Of the 109 renal allografts, 66 were donated after brain death (DBD) and 43 were donated after cardiac death (DCD). The mean total storage time was 17.19 +/- 4.71 hours with a mean CPM time of 10.91 +/- 4.22 hours. Datasets from the CPM showed that an allograft with reduced flow rates was associated with a dialysis requirement. Cold ischaemic time (CIT) >18hrs was associated with reduced rates of DGF in DCD kidneys placed on the CPM compared with CIT <18 hrs but results did not power statistical significance. There was no difference in the DGF or 1 year serum creatinine for paired kidneys where one was placed on the CPM and one was transplanted immediately.

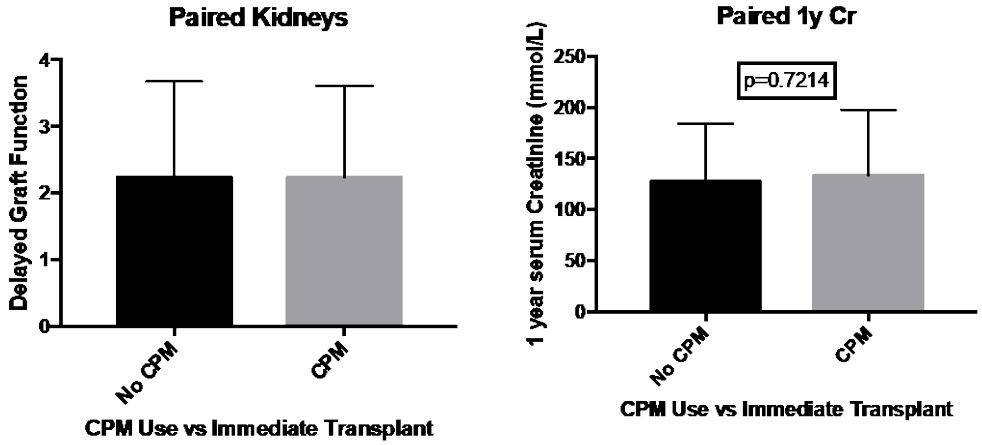


Figure. Comparison of paired kidneys.

Conclusions: Outcomes of renal transplants placed on the CPM are comparable to static hypothermic storage. There was no difference in DGF or 1 year serum creatinine. CPM datasets may be used to predict outcome such as need for dialysis after transplantation. Increased CIT is not associated with poor clinical outcome.

Abstract No. 19

COLD PERFUSION MACHINE DATASETS USED FOR KIDNEY TRANSPLANTS MAY PREDICT DELAYED GRAFT FUNCTION INCLUDING THE NEED FOR POST-TRANSPLANT DIALYSIS

BYRNE Sarah[1], PEREIRA Ryan[2], ROBERTSON Ian[2], TAN Ai Lin[2], LOCKWOOD David[3], KANAGARAJAH Vijay[2], RAY Mark[2], PRESTON John[2], WOOD Simon[2], LAWSON Malcolm[2], GRIFFIN Anthony[2], RHEE Handoo[2]

[1]Renal Transplant Unit, Princess Alexandra Hospital, Brisbane, [2]Department of Surgery, Princess Alexandra Hospital, Brisbane, [3]Princess Alexandra Hospital, Brisbane

Aims: To assess outcomes of renal transplant allografts that underwent a period of hypothermic perfusion storage at the Princess Alexandra Hospital (PAH) since the procurement of cold perfusion machines (CPM).

Method: All deceased donor renal transplants that were stored on the CPM at the PAH were analysed from 2011 to 2017. Datasets available from the CPM include systolic and diastolic pressure, flow rate, resistance and temperature. Data reports from the CPM parameters were analysed. Outcome data was obtained from electronic medical records. Primary outcome was delayed graft function (DGF).

Results: During this period 109 renal allografts were placed on the CPM. Of the 109 kidneys, 43 were donated after cardiac death (DCD) and 66 were donated after brain death (DBD). There was no difference in systolic/diastolic pressure, flow rate or resistance between DCD and DBD allografts on the CPM. Kidneys that required postoperative dialysis were associated with lower flow rates on the CPM compared to kidneys that did not require dialysis 123.8 ml/min vs 141.8 ml/min, p=0.0427. Subgroup analysis of the upper and lower 50% of CPM datasets showed statistical significance for flow rates and diastolic pressure. Increased CPM time was not associated with adverse outcome. There was a trend for improved DGF with longer CPM time.

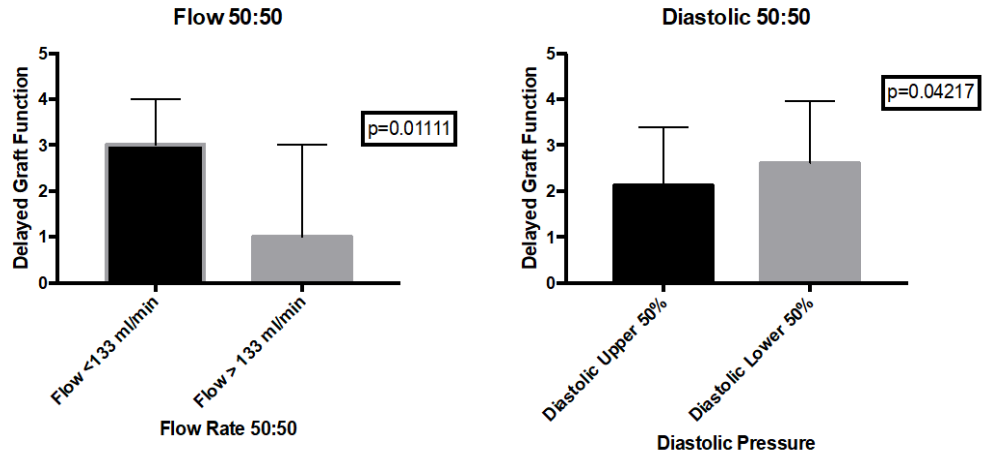


Figure. Upper and lower 50% of CPM datasets have demonstrated that poor flow may predict increased risk for dialysis post-transplant. Higher diastolic pressures were associated with lower rates of DGF.

Conclusion: Reduced flow rates and lower diastolic pressure in renal allografts on the CPM may be a predictor of DGF requiring postoperative dialysis. Prolonged CPM time is not associated with adverse outcome.

Abstract No. 20

CORRELATION AND AGREEMENT BETWEEN HLA-DRB AND HLA-DQ EPLET MISMATCHES BY LINKAGE DISEQUILIBRIUM AND HIGH-RESOLUTION HLA TYPING

LARKINS NG[1,2], TAVERNITI A[3], SHARMA A[4,3,5], DE SANTIS D[6], IRISH A[7], CHAKERA A[8,9,10], D'ORSOGNA L[11,10], WONG G[4,5,3], LIM WH[9,10]

[1]Department of Nephrology, Perth Children's Hospital, [2]School of Paediatrics and Child Health, University of Western Australia, Perth, [3]Centre for Kidney Research, The Children's Hospital at Westmead, Sydney, [4]Department of Nephrology, Westmead Hospital, Sydney, [5]School of Public Health, University of Sydney, [6]Transplant Immunobiology Laboratory, Fiona Stanley Hospital, [7]Department of Nephrology, Fiona Stanley Hospital, [8]Translational Renal Research Group, Harry Perkins Institute of Medical Research, [9]Department of Nephrology, Sir Charles Gairdner Hospital, Perth, [10]School of Medicine & Pharmacology, University of Western Australia, Perth, [11]Department of Immunology, Fiona Stanley Hospital

Aims: To determine the concordance and agreement of the HLA-DR and HLA-DQ eplet mismatches load calculated using common allelic haplotype association for HLA-DQA1 and DRB3/4/5 typing compared to high resolution typing for HLA-DRB1, DRB345, DQA1 and DQB1.

Methods: A retrospective cohort of 126 donor/kidney transplant recipient pairs in a single centre in Western Australia were included. Total number of eplet mismatches at HLA-DQ (HLA-DQA1+DQB1) and HLA-DR (HLA-DRB1+DRB3/4/5) were calculated using HLAMatchmaker from high-resolution typing (Next Generation Sequencer [NGS-typing]) across all alleles and from linkage disequilibrium (LD-typing) to assign HLA-DRB3/4/5 and HLA-DQA1 from HLA-DRB1 and HLA-DQB1 (LD-typing). Concordance and agreement between NGS-typing and LD-typing were determined.

Results: Of 126 donor/recipient pairs, 10 (8%) and 50 (40%) recipients were non-Caucasian and females, respectively. There were no Indigenous donors/recipients. The concordance coefficients for calculated HLA-DR+DQ eplet mismatches between the two methods was 0.997 (95%CI 0.996-0.998), with a tighter concordance for HLA-DQ>HLA-DR. The 95% limits of agreement contain 95% of the difference scores. The mean difference (bias) of the measurements between NGS-typing and LD-typing was 0.32 (95%CI 0.10 to 0.53). The SD of the difference was 1.22 and the width of the 95% limits of agreements was -2.08 (95%CI -2.46 to -1.71) to 2.72 (95%CI 2.34 to 3.09). There were 10 (8%) donor/recipient pairs with discordant results, of which 6 (5%) pairs had a difference of at least 5 eplet mismatches (see Table).

Conclusions: Allelic typing of HLA-DRB1 and DQB1 allows the assignment of HLA-DRB3/4/5 and DQA1 typing with a high degree of confidence in over 90% of donor/recipient pairs.

	NUMBER OF HLA-DR + DQ EPLET MISMATCHES DETERMINED BY NGS-TYPING	NUMBER OF HLA-DR + DQ EPLET MISMATCHES DETERMINED BY LD-TYPING	UNIQUE DR + DQ MM IDENTIFIED BY NGS -TYPING ONLY	UNIQUE HLA-DR + DQ EPLET MISMATCHES IDENTIFIED BY LD-TYPING ONLY
PAIR 1	21	21	1	1
PAIR 2#	45	43	6	4
PAIR 3#	29	28	2	1
PAIR 4#	39	39	3	3
PAIR 5#	40	40	3	3
PAIR 6#	35	38	2	5
PAIR 7	26	25	3	2
PAIR 8	28	30	1	3
PAIR 9	58	55	5	2
PAIR 10	8	7	2	1

#denotes recipients of Asian ethnicity.

Abstract No. 21

ABO INCOMPATIBLE LIVING DONOR KIDNEY TRANSPLANTATION IN AUSTRALIA AND NEW ZEALAND: A REPORT FROM THE ANZDATA REGISTRY

CLAYTON Philip[1], DANSIE Kathryn[2], CAMPBELL Scott[3], COATES Toby[1], COHNEY Solomon[4], IERINO Frank[5], IRISH Ashley[6], ISBEL Nicole[3], HUGHES Peter[7], KANELLIS John[8], LIM Wai[9], MULLEY William[8], PILMORE Helen[10], RUSS Graeme[1], TREVILLIAN Paul[11], WONG Germaine[12], WYBURN Kate[13]

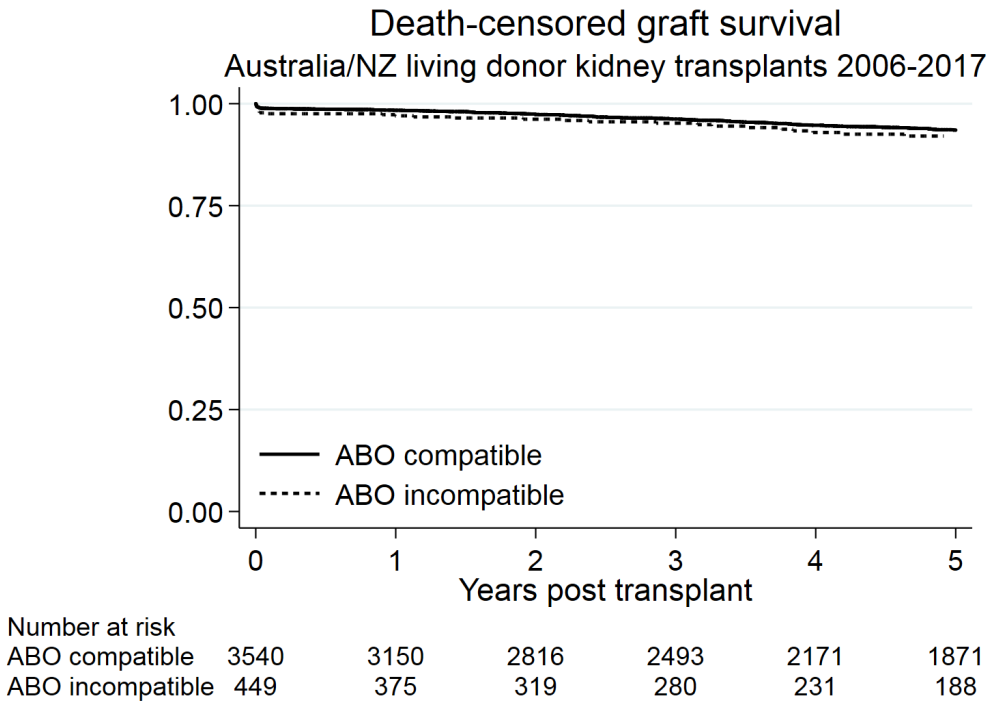
[1]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [2]Australia and New Zealand Dialysis and Transplant Registry (ANZDATA), South Australian Health and Medical Research Institute (SAHMRI), [3]Department of Nephrology, Princess Alexandra Hospital, Brisbane, [4]Department of Medicine, University of Melbourne, [5]Department of Nephrology, St Vincent's Hospital, Melbourne, [6]Renal Unit, Fiona Stanley Hospital, [7]Renal Unit, Royal Melbourne Hospital, [8]Department of Nephrology, Monash Medical Centre, Melbourne, [9]Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth, [10]Department of Renal Medicine, Auckland City Hospital, [11]Department of Nephrology, John Hunter Hospital, Newcastle, [12]Department of Renal Medicine, Westmead Hospital, Sydney, [13]Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney

Aim: To describe outcomes of ABO incompatible (ABOi) living kidney donor (LKD) transplantation in Australia and New Zealand.

Methods: We included all LKD recipients in Australia/NZ over 2006-17 from the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry. Outcomes were overall and death-censored graft survival, patient survival, delayed graft function (DGF), time to first rejection and first antibody-mediated rejection (AMR), and estimated GFR (eGFR). We used adjusted Cox models to examine time-to-event data, chi-squared tests to compare proportions and a linear mixed model to compare eGFR.

Results: 3989 transplants met inclusion criteria and contained ABO data, of which 449 (11%) were ABOi. ABOi recipients were older (49 vs 45 years), with older donors (52 vs 50 years) who were more commonly receiving antihypertensives (13 vs 10%) and had more HLA mismatches. ABOi recipients received more B cell depletion at induction (24% vs <1%) and were more likely to receive tacrolimus at baseline (94% vs 69%). Death-censored graft survival for ABOi recipients was worse in the first month (hazard ratio (HR) 2.2, 95% CI 1.1-4.3) but not thereafter (HR 1.1, 95% CI 0.8-1.7) (figure). There was more rejection in the ABOi group driven by excess AMR (HR 2.0, 95% CI 1.5-2.7). There were no differences in overall graft survival or eGFR, and patient survival and DGF were similar.

Conclusion: ABOi recipients experienced more AMR and slightly worse death-censored graft survival in the first month post-transplant, but overall results were similar to ABO compatible recipients.



Abstract No. 22

DEVELOPMENT OF DE NOVO HLA DONOR SPECIFIC ANTIBODIES AND ALLOGRAFT REJECTION POST BLOOD TRANSFUSION IN KIDNEY TRANSPLANT RECIPIENTS

JALALONMUHALI Maisarah[1,2], CARROLL Robert[1], CLAYTON Philip[1], COATES Toby[1]

[1]Central Northern Adelaide Renal and Transplantation Service, Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [2]Department of Medicine, University Malaya Medical Centre, Kuala Lumpur, Malaysia

Aim: To look at the incidence of *de novo* HLA donor specific antibody (DSA) formation and the immediate allograft rejection post blood transfusion in highly immunosuppressed kidney transplant recipients (KTR).

Methods: KTR who had blood transfusion within 1 week of surgery at Royal Adelaide Hospital (RAH) between 2010 and 2018 were recruited. They received either basiliximab or ATG as an induction therapy, followed by tacrolimus (trough level 8-12 ng/mL), mycophenolate mofetil 1500 mg bd and prednisolone 30 mg od as maintenance therapy. HLA DSA positivity was determined by MFI of ≥ 500 as measured by Luminex xMAP™ technology. The test was performed between 2 weeks to 3-month post-transplant.

Results: A total of 706 patients underwent kidney transplant at RAH between 2010 and 2018 (8 patients were excluded from analysis – 2 had graft biopsy prior to blood transfusion, 6 with non-functioning graft (0.8%)). 203 (29%) patients received blood transfusion during 1 week perioperative period. Out of this, 134 patients needed transfusion within 48 hours. Mean age was 52.61 ± 13.29 with 110 (54.2%) patients were male. The perioperative hemoglobin was 111.12 ± 11.75 g/L and the lowest hemoglobin was 74.74 ± 9.55 g/L in the transfusion group. Table 1 illustrated the main outcome of blood transfusion.

Conclusions: Blood transfusion with immunosuppression cover was not associated with any significance impact on development of *de novo* HLA DSA and acute rejection. This has been illustrated in pediatric pre-kidney transplant blood transfusion with cyclosporine cover[1].

Parameters	**Transfusion group**	**Non-transfusion group**	**p-value**
De novo DSA	15 (8.2%)	43 (9.6%)	0.567[a]
Class I	5 (2.7%)	26 (5.8%)	
Class II	10 (5.4%)	17 (3.8%)	
MFI (Immunodominant HLA DSA)	3701.50 ± 5564.41	2999.02 ± 4706.66	0.630[b]
Rejection (Histology)			
Borderline	5 (2.5%)	23 (4.7%)	0.122[c]
TCMR 1A/B	8 (4.0%)	24 (4.9%)	
TCMR + V	10 (5.0%)	40 (8.2%)	
MVI	7 (3.5%)	15 (3.1%)	
ABMR	1 (0.5%)	2 (0.4%)	

a – Chi-square, b – Independent samples t-test, c – Kruskal-Wallis

Table 1: De novo DSA and acute rejection episode post blood transfusion.

References

1. Niaudet P et al. Pretransplant blood transfusions with cyclosporine in pediatric renal transplantation. Padiatr Nephrol. 2000 Jun; 14(6): 451-6.

Acknowledgements

1. Central Northern Adelaide Renal and Transplantation Services (CNART)
2. International Society of Nephrology (ISN)

Abstract No. 23

RAPID REDUCTION OF DONOR-SPECIFIC ANTIBODIES IN SIMULTANEOUS LIVER-KIDNEY TRANSPLANTATION

NEWMAN Allyson[1], LAI Sum Wing Christina[1], ABOU-DAHER Frederika[2], WALTON Duncan[2], WATSON NARELLE[2], MAWSON JANE[1], WYBURN KATE[1], CHADBAN STEVE[1], GRACEY DAVID[1]

[1]Renal & Transplantation Unit, Royal Prince Alfred Hospital, Sydney, [2]Australian Red Cross Blood Service

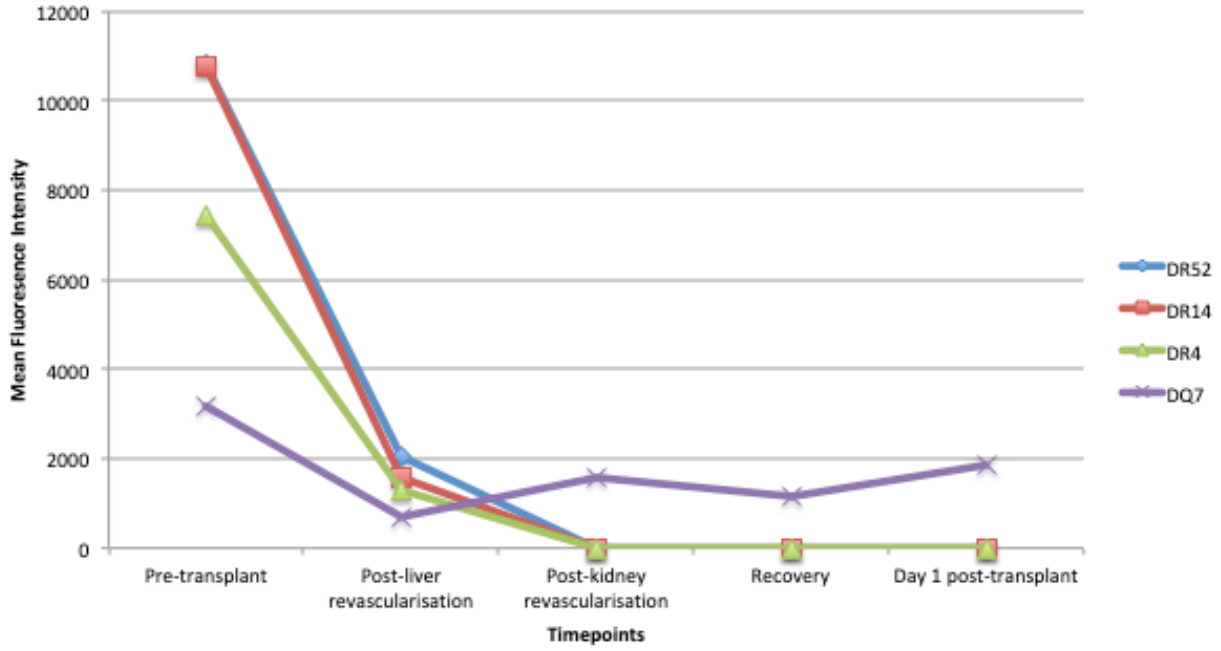
Background: Kidney transplantation performed in the presence of high-titre donor-specific antibodies (DSA) may result in hyper-acute or accelerated antibody-mediated rejection and graft loss. Previous studies have shown that this risk may be mitigated in cases of simultaneous liver-kidney transplantation (SLKT); however, the mechanisms are not well defined. Here we report the evolution of pre-formed, high level DSAs in two highly sensitised liver-kidney transplant recipients at various timepoints peri-operatively and describe a profound sustained depletion of all DSAs from the time of liver anastomosis.

Methods: HLA antibody samples were collected pre-operatively, and immediately post-liver and post-kidney revascularisation. HLA Matchmaker was used to assess HLA epitope and Complement Dependant Cytotoxicity (CDC) cross-matches were performed. Both patients received standard immunosuppression with Basiliximab and Methylprednisolone as induction therapy and Prednisolone, Mycophenolate and Tacrolimus as maintenance therapy.

Results: Both patients were highly sensitised with PRA up to 97%. One patient had positive B- and T-cell crossmatch pre-transplant. HLA antibodies rapidly reduced post-liver revascularisation and remained low. In particular, HLA antibodies associated with graft specific eplets showed greater reduction (Figure 1) and the observation was apparent with C1q result. Positive CDC crossmatches became negative within 3 hours post-liver revascularisation. Both patients maintained good graft function with no rejection on liver and kidney biopsies at 10 weeks post-transplant.

Conclusion: The reduction in DSAs occurred immediately post-liver revascularisation. These cases supports the hypothesis of the protective immunoregulatory mechanism of the liver in the setting of SLKT with no extra antibody removal treatment required peri-operatively for highly sensitised patients.

Figure 1: HLA IgG DSA Level of Patient 1



Abstract No. 24

COSTS IN CONTEMPORARY TRANSPLANT PRACTICE

WYLD Melanie[1,2], YING Tracey[1,2], WYBURN Kate[1,2], CHADBAN Steve[1,2]

[1]Renal Unit, Royal Prince Alfred Hospital, Sydney, [2]Sydney Medical School, University of Sydney

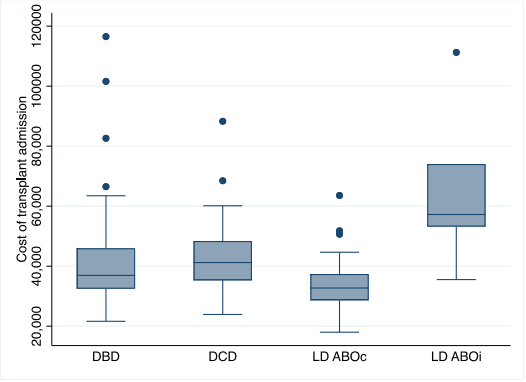
Background: The cost of kidney transplantation, in the context of increasing immunological and medical complexity and higher donor and recipient age, remains uncertain. There is scant published cost data, and none from Australia in the last decade. We sought to evaluate transplantation costs in contemporary practice.

Methods: Hospital level costs for all kidney transplant admissions July 2015-July 2016 in an Australian Transplant unit captured by accounting software were included. Costs were analysed using multivariate linear regression.

Results: 98 transplants were performed from 34 living and 64 deceased donors, 45 after brain death(DBD) and 19 after circulatory death(DCD). Most transplants were immunologically complex: 71(72%) had ≥3 HLA mismatches, 34(35%) had pre-transplant donor specific antibodies, 16(16%) were re-transplants and 7 were ABO incompatible (ABOi). Delayed graft function occurred in 36(37%). The age and sex adjusted transplant admission cost was $45,313AUD (95%CI:$28,849,$61,778) for DBD transplants, $46,541 (95%CI:$36,583,$56,498) for DCD transplants, and $42,880 (95%CI:$36,664,$51,096) for LD transplants(p=0.7). ABOi cost significantly more than ABO compatible transplants at $63,172 (95%CI:$49,854,$76,491) and $40,387 (95%CI:25,331,55,443) respectively (p<0.001)(Figure1). DGF increased transplant costs by $15,356 (95%CI:$7953,$22,760, p<0.001) after adjusting for age, sex, donor type, and ABO compatibility. These additional costs were primarily driven by increased length of stay. In our centre, 40% of complex transplants lead to financial losses for the hospital, compared to 27% for standard transplants.

Conclusions: Immunologically complex transplants have increased costs of transplantation. Current funding mechanisms may need to be revised to take this into account.

Figure 1: Costs of transplant admission by donor type ($AUD)



Abstract No. 25

IN VITRO EXPANSION OF IL-10-COMPETENT HUMAN B CELLS

PERKINS Griffith[1], COATES Toby[2,3], HURTADO Plinio[2,3]

[1]School of Biological Sciences, University of Adelaide, [2]School of Medicine, University of Adelaide, [3]Renal Unit, Royal Adelaide Hospital

Aim: To develop a system for expanding IL-10-competent B cells from human peripheral blood.

Methods: Transitional, naïve and memory B cell populations were isolated by fluorescence-activated cell sorting based on differential expression of CD24 and CD38. Cells were expanded for 7 days by co-culture with CD40-ligand-expressing NIH3T3 cells plus IL-4 and/or IL-2. Fold-expansion of $CD19^{+}$ cells was enumerated at Day 7, and IL-10 concentrations in supernatants were measured at 24-hour intervals by ELISA. Thirty immunomodulatory factors were investigated for their capacity to induce IL-10-competence and secretion in the expanded populations, where 'competence' is quantified by the amount of IL-10 secreted per cell after 5-hours of PMA/ionomycin-driven cytokine production.

Results: $CD24^{hi}CD38^{hi}$ (transitional), $CD24^{int}CD38^{int}$ (naïve), and $CD24^{hi}CD38^{neg}$ (memory) populations expanded on average 3-4-fold over 7-days and secreted detectable amounts of IL-10 during expansion, peaking around Day 7. In all expanded populations, the combination of CpG-B (2.7μg/mL) and IFN-α and/or IFN-β (2,000-50,000U/mL) induced the greatest IL-10 secretion into supernatants. Expanded transitional cells demonstrated the greatest IL-10 competence following CpG/IFN-α/β treatment, and secreted more IL-10 when expanded with IL-2 and IL-4, compared with IL-2 or IL-4 alone. Expanded transitional cells produced more IL-10 than non-expanded transitional cells similarly treated with CpG/IFN-α/β.

Conclusion: We compare, for the first time, the capacity of expanded transitional, naïve and memory B cells to produce IL-10, and report a reproducible cell culture system for expanding IL-10-competent B cells from human transitional B cells, which will facilitate the study of their potential therapeutic use in transplantation to promote immune tolerance.

Abstract No. 26

EXPRESSION OF DONOR MHC CLASS I IN RECIPIENT HEPATOCYTES DOES NOT INDUCE LINKED SUPPRESSION TO H-Y

LEONG Mario[1], PAUL-HENG Moumita[1], WANG Chuanmin[1], SON Taeyoung[1], BERTOLINO Patrick[2], BISHOP Alex[1], BOWEN David[1,3], SHARLAND Alexandra[1]

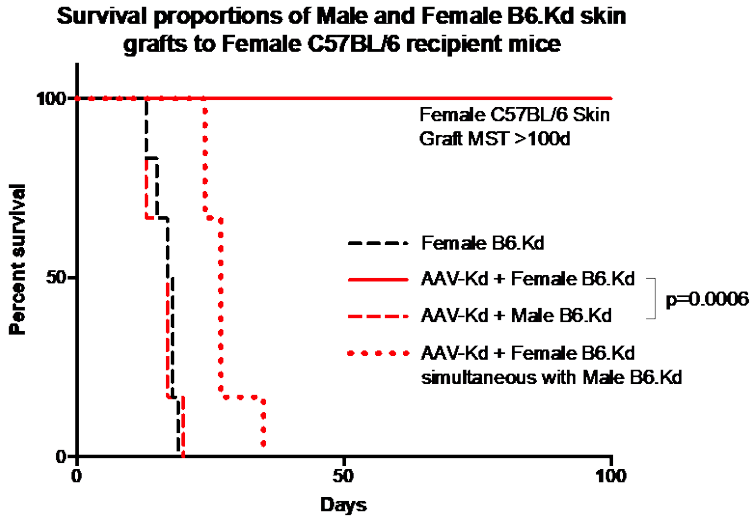
[1]Transplantation Immunobiology Group, Central Clinical School, Faculty of Medicine and Health, University of Sydney, [2]Royal Prince Alfred Hospital and University of Sydney, [3]AW Morrow Gastroenterology & Liver Centre, Royal Prince Alfred Hospital and University of Sydney

Background: Expression of allogeneic MHC I by recipient hepatocytes results in tolerance to subsequent skin grafts expressing the same mismatched MHC allele. To determine whether tolerance to allogeneic MHC I could induce linked suppression to minor antigen mismatches, we examined responses to the minor antigens H-Y in association with the allogeneic MHC I allele H-2Kd.

Methods: Female B6.Kd or C57BL/6 mice inoculated with AAV-Kd $5x10^{11}$ vgc received skin grafts from female C57BL/6, female B6.Kd or male B6.Kd donors. Graft survival and Kd expression were monitored.

Results: Tolerance to female B6.Kd skin was achieved in all AAV-Kd treated female C57BL/6. Conversely, Kd treated female C57BL/6 mice receiving simultaneous grafts of male and female skin rejected both grafts (MST 16.2 d and 27.3 d). Kd-tolerant female mice rejected secondary female and male B6.Kd grafts with similar tempo (MST 22 d and 24 d) and tolerance to the original female B6.Kd skin grafts was also broken. Kd expression in all recipient livers persisted despite skin graft rejection. Female B6.Kd mice receiving simultaneous male and female B6.Kd skin grafts also rejected 50% of the syngeneic female grafts (MST 32.5 d) suggesting that a bystander effect may be responsible for female graft destruction.

Conclusions: Expression of Kd in C57BL/6 hepatocytes does not induce linked suppression to H-Y. In C57BL/6 mice, two class I H-Y epitopes are known. Co-expression of these as single chain trimers (Db-HY) may synergise with Kd heavy chain to induce tolerance across the sex-mismatch in this stringent skin graft model.



Abstract No. 27

THE SELF-PEPTIDE REPERTOIRE PLAYS A CRITICAL ROLE IN TRANSPLANT TOLERANCE INDUCTION

SON Eric[1], PAUL-HENG Moumita[1], LEONG Mario[1], FARIDI Pouya[2], MIFSUD Nicole[2], ALEXANDER Ian[3,4], BERTOLINO Patrick[5], PURCELL Anthony[2], BOWEN David[1,5], SHARLAND Alexandra[1]

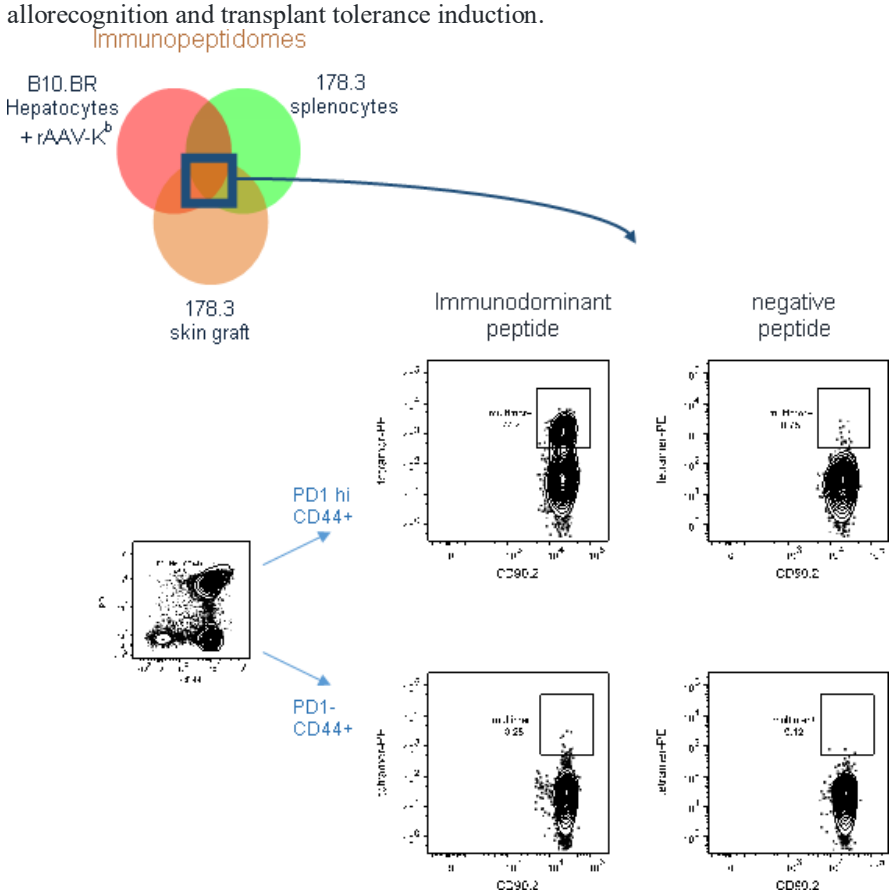
[1]Transplantation Immunobiology Group, Central Clinical School, Faculty of Medicine and Health, University of Sydney, [2]Department of Biochemistry and Molecular Biology, Monash University, Melbourne, [3]Gene Therapy Research Unit, Children's Medical Research Institute, University of Sydney, Faculty of Medicine and Health and Sydney Children's Hospitals Network, [4]Discipline of Child and Adolescent Health, the University of Sydney, Sydney Medical School, [5]AW Morrow Gastroenterology & Liver Centre, Royal Prince Alfred Hospital and University of Sydney

Background: Donor-specific tolerance can be induced by adeno-associated viral vector (AAV) mediated expression of donor MHC-I heavy chains (HC) in the recipient liver. Tolerance induction depends upon direct recognition of intact donor MHC-I. The role of the hepatocyte endogenous peptide repertoire in tolerance induction is unknown.

Methods: To express high levels of donor class I while excluding binding of naturally-processed endogenous peptides, we engineered AAV vectors expressing a single-chain trimer (SCT) of β2-microglobulin, MHC heavy chain and a defined peptide sequence (KIITYRNL or SIINFEKL). B10.BR or B10.BR-RAG mice reconstituted with Des-RAG cells (which recognise the Kb-KIITYRNL epitope), were inoculated with either Kb, SCT-Kb_KIITYRNL or SCT-Kb_SIINFEKL vectors, then challenged with Kb-bearing skin grafts. The Kb immunopeptidomes of liver, skin and spleen were determined using mass spectrometry and self-peptides were screened by tetramer binding (Figure).

Results: B10.BR-RAG mice reconstituted with Des-RAG cells accepted Kb-bearing skin grafts indefinitely when transduced with SCT-Kb_KIITYRNL but rejected grafts with a MST of 20 days after inoculation with SCT-Kb_SIINFEKL ($p<0.0005$). Conversely, while inoculation of B10.BR mice with AAV-Kb induced tolerance, treatment with either SCT-Kb vector only prolonged graft survival by a few days ($p<0.0005$). From a pilot pool of self-peptides, we identified one immunodominant peptide recognized by 27% of activated alloreactive CD8+ T cells, and two subdominant peptides. Screening is ongoing.

Conclusions: Self-peptides play a critical role in tolerance induction. Identification of individual pMHC epitopes is feasible using mass spectrometry and multimer staining. These findings have broad implications for allorecognition and transplant tolerance induction.



Abstract No. 28

EFFECT ON CHRONIC ALLOGRAFT REJECTION OF ANTIGEN SPECIFIC TREG INDUCED BY ACTIVATION OF NAIVE TREG WITH ALLOANTIGEN AND IL-4

RAKESH PRATEEK[1,2], BEDI SUKHANDEEP K.[1,2], VERMA NIRUPAMA D.[1,2], TRAN GIANG[1,2], ROBINSON CATHERINE M.[1,2], WANG CHUANMIN[3,4], SHARLAND ALEXANDRA[3,4], HALL RACHEL M.[5], WILCOX PAUL L.[5], HODGKINSON SUZANNE J.[1,2,6], HALL BRUCE M[1,5,7]

[1]Department of Medicine, University of New South Wales, Sydney, [2]Immune Tolerance Laboratory, Ingham Research Institute for Applied Medical Research, [3]Australian National Liver Transplantation Unit, Royal Prince Alfred Hospital, Sydney, [4]Faculty of Medicine and Health, University of Sydney, [5]Immune Tolerance Laboratory, Ingham Institute for Applied Medical Research, [6]Department of Neurology, Liverpool Hospital, [7]Department of Nephrology, Liverpool Hospital

Background: Naive $CD4^+CD25^+Foxp3^+$ T regulatory cells (Treg) activated by antigen and IL-4 express the specific receptor for IL-5 (IL-5R alpha). These cells (Ts2) can be further activated with IL-5 and specific antigen to generate more potent Th2-like Treg.

Methods and Results: A chronic cardiac allograft rejection model in F344 rats that rejected Lewis grafts in 18-28 days (n=8). Treatment with 5000U of rIL-5 (i.p.) daily for 10d from d7 post grafting prolonged survival of all grafts to >60d (n=8), and reduced severity of rejection. IL-5-treated rats had $CD4^+CD25^+$T cells that proliferated to Lewis but not self and this response was enhanced by rIL-5. Continued treatment with rIL-5 (n=5) showed better graft protection (n=5). The beneficial effect of rIL-5 was abolished by pre-treatment with neutralising anti IL-4 or blocking anti-CD25 mAb consistent with activation of host tTreg to Ts2 cells in presence of IL-4.

$CD4^+CD25^+$Treg from naïve F344 were activated with IL-4 and Lewis stimulator cells to generate Ts2 cells. $5x10^6$ Ts2 cells given to F344 bearing a Lewis heart graft at 9d post-grafting reduced the severity of rejection (n=4). $CD4^+CD25^+$Treg activated with IL-4 and PVG (third-party) antigen (n=4) did not reduce rejection. $CD4^+CD25^+$T cells either activated by alloantigen in vitro with rIL-4 or expanded by rIL-5 treatment in vivo, expressed IL-5Ralpha and their proliferation to specific donor, but not self or third party was enhanced by rIL-5.

Conclusion: These studies suggest activation of tTreg with IL-4 and antigen to Ts2 cells may have therapeutic potential for chronic allograft rejection.

Abstract No. 29

LONG-TERM P2X7 BLOCKADE REDUCES LIVER GRAFT-VERSUS-HOST DISEASE IN HUMANISED MICE

GERAGHTY Nicholas[1,2,3], WATSON Debbie[1,2,3], SLUYTER Ronald[1,2,3]

[1]School of Chemistry and Molecular Bioscience, University of Wollongong, [2]Molecular Horizons, University of Wollongong, [3]Illawarra Health and Medical Research Institute, University of Wollongong

INTRODUCTION: Graft-versus-host disease (GVHD) is a complication of bone marrow transplantation, where donor leukocytes mount an immune response against the recipient. Extracellular adenosine triphosphate (ATP) activates P2X7 to promote GVHD in allogeneic mouse models. Short-term P2X7 blockade with Brilliant Blue G (BBG) reduces serum human interferon gamma (hIFN-γ) and histological GVHD in a humanised mouse model.
AIM: To investigate long-term P2X7 blockade on GVHD development in a humanised mouse model.
METHODS: NOD-SCID-IL2γ^{null} (NSG) mice injected with 10 x 10^6 human (h) peripheral blood mononuclear cells (PBMC), were injected with BBG (50 mg/kg) or saline thrice weekly for up to 10 weeks. Weight loss and clinical parameters for GVHD were assessed. Flow cytometry for human cell engraftment, ELISA for serum cytokines, and histological and immunohistochemical analysis of liver damage were performed.
RESULTS: BBG did not impact human cell engraftment (hCD45$^+$, hCD3$^+$, hCD4$^+$, hCD8$^+$ and NK cells). Both groups showed similar weight loss, clinical score and survival (median survival time; BBG, 45.5 days, n = 10, Saline, 50 days, n = 7) (P = 0.3014). However, BBG-mice demonstrated reduced histological damage and leukocyte infiltration (52% reduction) in the liver (P = 0.0020). Further, BBG-mice had reduced apoptosis (57% reduction) in the liver (P = 0.0244). Contrary to a short-term regime, long-term P2X7 blockade with BBG did not impact serum hIFN-γ concentrations.
CONCLUSION: P2X7 plays a role in liver GVHD in this humanised mouse model, but further investigation into P2X7 blockade is warranted before it can be used as a therapeutic strategy for GVHD in humans.

Abstract No. 30

RISK FACTORS FOR ADVANCED COLORECTAL NEOPLASIA IN KIDNEY TRANSPLANT RECIPIENTS

AU Eric HK[1,2], HOWARD Kirsten[1], CHAPMAN Jeremy R[3], CASTELLS Antoni[4,5], ROGER Simon[6], BOURKE Michael J[7], MACASKILL Petra[1], TURNER Robin[1,8], WILLIAMS Gabrielle[1], LIM Wai H[9], LOK Charmaine E[10], DIEKMAN Fritz[11], CROSS Nicholas[12], SEN Shaundeep[13], ALLEN Richard DM[14], CHADBAN Steven J[14,15], POLLOCK Carol A[16], TONG Allison[1,2], TEIXEIRA-PINTO Armando[1,2], YANG Jean YH[17], WILLIAMS Narelle[1,2], KIEU Anh[1,2], JAMES Laura[1,2], CRAIG Jonathan[18], WONG Germaine[1,2,3]

[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 Hospital, Sydney, [4]Gastroenterology Department, Hospital Clinic, Hospital Clinic, University of Barcelona, [5]University of Barcelona,, [6]Department of Renal Medicine, Gosford Hospital, [7]Department of Gastroenterology, Westmead Hospital, Sydney, [8]Biostatistics Unit, Dunedin School of Medicine, University of Otago, [9]Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth, [10]Faculty of Medicine, University of Toronto, [11]Department of Nephrology and Kidney Transplantation, Hospital Clinic, Barcelona, Spain, [12]Department of Nephrology and Kidney Transplantation, Christchurch Hospital and University of Otago, [13]Department of Renal Medicine, Concord Repatriation General Hospital, [14]Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney, [15]Charles Perkins Centre, University of Sydney, [16]Department of Medicine, Northern Clinical School, Kolling Institute of Medical Research, [17]School of Mathematics and Statistics, University of Sydney, [18]College of Medicine and Public Health, Flinders University

Background: The incidence of colorectal cancer is over 1.5-times higher in kidney transplant recipients than the general population, but the risk factors are poorly understood.

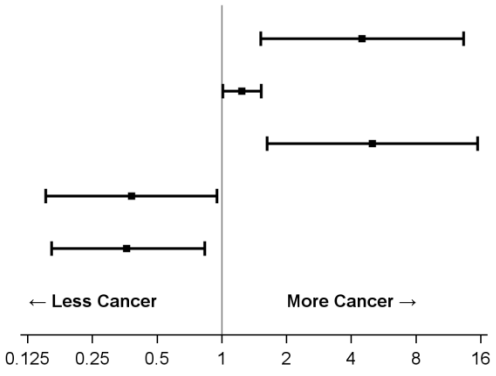
Aim: To identify risk factors for developing advanced colorectal neoplasia in kidney transplant recipients.

Methods: Kidney transplant recipients across eleven sites in Australia, New Zealand, Canada and Spain were screened for colorectal neoplasm using faecal immunochemical test (FIT) as part of the DETECT study (n=1706) on screening using FIT across all CKD stages. Advanced colorectal neoplasia were confirmed through a 2-step process with colonoscopies performed for positive FIT and 2-years follow-up for all patients. Potential risk factors for advanced colorectal neoplasia were assessed using multivariable logistic regression.

Results: A total of 497 transplant recipients (63% male, median age 54.3 years) received FIT screening. Of these, 96 (19.3%) had colonoscopy for positive FIT and 28 advanced colorectal neoplasia were identified (detection rate 5.6 %). One patient with negative FIT was diagnosed with advanced colorectal neoplasia during 2-year follow-up. Factors associated with advanced colorectal neoplasia included male sex [Odds ratio 4.5 (95% CI 1.5-13.4)], older age [1.2 per 5-year increase (1.0-1.5)] and anticoagulant use [5.0 (1.6-15.6)]. The number of acute rejection episodes [0.38 (0.15-0.96)] and mycophenolate mofetil (MMF) use [0.36 (0.16-0.84)] were associated with lower odds of advanced colorectal neoplasia.

Conclusion: Older age, male sex, and anticoagulant use were independent predictors for advanced colorectal neoplasia in transplant recipients. Prior acute rejection episodes and users of MMF may be associated with lower risk for colorectal neoplasia.

Risk Factor	Odds Ratio (95% CI)	P value
Male vs Female	4.5 (1.5 to 13.4)	0.002
Age (per 5 year increase)	1.2 (1.0 to 1.5)	0.05
Regular anticoagulant use	5.0 (1.6 to 15.6)	0.01
Acute rejection (per episode)	0.38 (0.15 to 0.96)	0.02
Use of mycophenolate mofetil	0.36 (0.16 to 0.84)	0.02



Abstract No. 31

TARGETING INFLAMMATORY MONOCYTES BY IMMUNE-MODIFYING NANOPARTICLES PREVENTS KIDNEY ALLOGRAFT REJECTION

LAI Sum Wing Christina[1,2], WU Huiling[1], LOH Yik Wen[1], SINGER Julian[1,3], NIEWOLD Paula[1], GETTS Daniel[1], KING Nicholas[1], CHADBAN Steve[1,3]

[1]University of Sydney, [2]Renal & Transplantation Unit, Royal Prince Alfred Hospital, Sydney, [3]Royal Prince Alfred Hospital, Sydney

Aim: We previously reported the capacity of immune-modifying nanoparticles (IMPs) to bind to circulating inflammatory monocytes/macrophages(MΦ) via the specific scavenger receptor MARCO, thereby causing inflammatory MΦ removal in the spleen with subsequent protection in models of infection, autoimmunity and ischemia injury. Here we investigated the therapeutic potential of IMPs to target MΦ in kidney allograft rejection in a murine model of kidney transplantation.

Methods: Kidney transplants were performed from BALB/c to C57BL/6 mice as WT allografts. A group of WT allograft mice received a daily intravenous injection of 300ul ($1.46x10^{10}$particles/ml, 500nm in diameter) of negatively-charged IMPs from day 1 post-transplant for 15 days (WT+IMP). Samples were collected at days 14 and 100 post-transplantation.

Results: WT+IMP allografts had prolonged survival compared to WT allografts (Fig.1, $p<0.05$), and were protected from acute rejection with lower creatinine (23.8±1.4 vs. 48.1±18.7μmol/L, $p<0.01$) and less tubulitis (95.6±12.6 vs. 140.4±33.9scores, $p<0.001$). Histologically, accumulation of $CD4^+$ (37.8±13.3 vs. 73.1±31.7cells/HPFs, $p<0.01$), $CD8^+$ (39.1±17.7 vs. 75.9±39.2cells/HPFs, $p<0.05$), $CD68^+$ (12.4±4.5 vs. 21.8±6.9%field positive, $p<0.01$) and CD11c (1.802±0.8573 vs. 12.56±4.658%field positive, $p<0.001$) cells were reduced in WT+IMP allografts, compared to WT allografts. High dimensional flow cytometry analysis of cells isolated from allografts showed significantly reduced T cells and myeloid cells in WT+IMP. WT+IMP allografts expressed significantly less pro-inflammatory (IL6) and Th1 (IFN□) cytokines and cytotoxic molecules (Perforin, GranzymeB & iNOS).

Conclusion: IMP infusion affords significant protection from acute allograft rejection, indicating therapeutic potential. Diverting inflammatory MΦ away from the allograft may be a useful strategy to prevent subsequent adaptive alloimmunity.

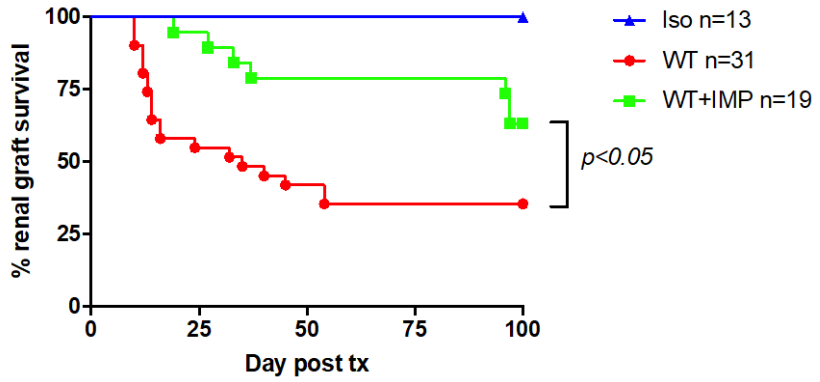


Figure 1: WT+IMP prolongs kidney allograft survival.

Abstract No. 32

EFFECT OF PROTON PUMP INHIBITOR ON MYCOPHENOLIC ACID EXPOSURE IN KIDNEY AND LIVER TRANSPLANT RECIPIENTS: A DOUBLE-BLIND RANDOMISED CROSS-OVER TRIAL

SUNDERLAND Andrew[1], RUSS Graeme[2], SALLUSTIO Benedetta[3], CERVELLI Matthew[3], JOYCE David[4], JEFFREY Gary[5], BOUDVILLE Neil[6], CHAKERA Aron[6], DOGRA Sharan[6], CHAN Doris[6], WONG Germaine[7], LIM Wai[6]

[1]Sir Charles Gairdner Hospital, Perth, [2]Nephrology and Renal Transplant, Royal Adelaide Hospital, [3]Department of Clinical Pharmacology, Royal Adelaide Hospital, [4]School of Medicine & Pharmacology, University of Western Australia, Perth, [5]Hepatology and Liver Transplant Medicine Unit, Sir Charles Gairdner Hospital, Perth, [6]Nephrology and Renal Transplant, Sir Charles Gairdner Hospital, Perth, [7]School of Medicine, University of Sydney

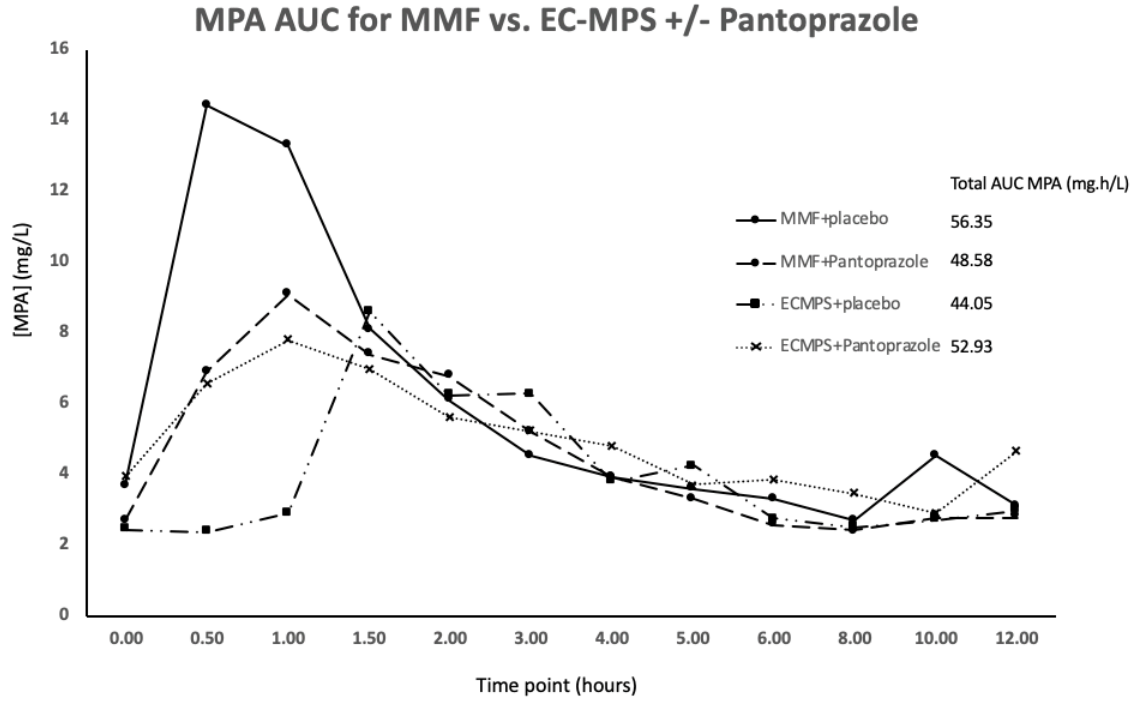
Aim: To determine the effect of the proton-pump inhibitor (PPI) pantoprazole on mycophenolic acid (MPA) exposure in stable kidney/liver transplant recipients maintained on Mycophenolate Mofetil (MMF) or enteric-coated mycophenolate sodium (EC-MPS).

Methods: A multicentre randomised, prospective, double-blind placebo-controlled cross-over study was conducted to determine the effect of gastric acid suppression (pantoprazole 40mg daily or matching placebo) on the MPA-AUC over 12 hours (MPA-AUC12h) and maximum concentration (MPA-Cmax) in recipients ≥6 months post-transplant maintained on MMF (≥1g/daily in equally divided doses) or EC-MPS (≥1080mg/daily in equally divided doses), with calcineurin-inhibitor and corticosteroids.

Results: Of 40 participants randomised, 19 (47.5%) and 21 (52.5%) were maintained on MMF and EC-MPS, respectively. The mean (SD) age was 58 (11) years and 67% of participants were males. Almost 50% were maintained on PPI, which was ceased for 2-weeks prior to randomisation. Almost 70% of participants were maintained on tacrolimus-based immunosuppressive regimen. In recipients maintained on MMF, concomitant treatment with pantoprazole significantly reduced mean MPA-AUC12h by 19% (absolute reduction of 10.8 [95%CI 4.1, 17.5] mg.h/L; paired *t-test* 0.003) and MPA-Cmax by 33% (absolute reduction of 6.0 [2.3,9.7] mg/L; paired *t-test* 0.003). In contrast, pantoprazole significantly increased the MPA-AUC12h (19% absolute increase p=0.037) but not for MPA-Cmax in recipients maintained on EC-MPS (Figure 1).

Conclusion: The co-administration of pantoprazole substantially reduced the bioavailability of MPA in patients maintained on MMF, and therefore, clinicians should be cognisant of this drug interaction, which may have important clinical implications.

Figure 1.



Abstract No. 33

A20 IS PROTECTIVE AGAINST ACUTE KIDNEY INJURY REGARDLESS OF IMMUNOLOGICAL ENVIRONMENT

LI Jennifer[1,2], NGUYEN-NGO Danny[1,2], ZAMMIT Nathan[3], EL-RASHID Maryam[1,2], MINHAS Nikita[4], GREY Shane[3], ROGERS Natasha[1,2]

[1]Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney, [2]Westmead Clinical School, University of Sydney, [3]Transplant Immunobiology Laboratory, Garvan Institute of Medical Research, Sydney, [4]Centre for Transplant and Renal Research, University of Sydney

Inflammation is an important mechanism for acute kidney injury, which contributes to overall mortality of hospitalized patients. The pro-inflammatory cytokine, tumor necrosis factor (TNF-α) upregulates A20 (a zinc finger protein), which has an important role in maintaining homeostasis in inflammation and immunity. Various single nucleotide polymorphisms (SNP) within the A20 gene have been associated with increased susceptibility to inflammatory diseases.

Aim: Examine the effect of I325N single nucleotide polymorphism within the A20 locus on inflammation and acute kidney injury.

Methods: Age- and gender-matched littermate control (wild-type), heterozygous ($A20^{\Delta/+}$) and homozygous ($A20^{\Delta/\Delta}$) I325N SNP mutant mice were challenged by renal ischemia reperfusion injury (IRI) and had subsequent analysis of renal function and biomolecular phenotyping.

Results: Mutant $A20^{\Delta/+}$ and $A20^{\Delta/\Delta}$ mice were protected from renal IRI, with lower serum creatinine compared to littermate controls at 24hr reperfusion, reduced TUNEL-positive staining and histological damage of kidney tissue. Paradoxically, these A20 mutant mice demonstrated upregulation of pro-inflammatory cytokines TNF-α, CCL2 and CXCL2, increased CD11c+ dendritic cells (DC), F4/80+CD11b+ macrophages (expressing maturation markers CD80 and CD40), and both CD103+ DC and CD8+ T cells. $A20^{\Delta/\Delta}$ mice also demonstrated upregulation of nitrosative stress, downregulation of superoxide dismutase expression, and was associated with increased phospho-NFκB expression in renal parenchymal and tubular epithelial cells. Cell lines transfected with A20 constructions (wild-type or I325N variant) and incubated with TNF-α did not change cell viability. **Conclusions**: The I325N SNP variant of A20 leads to NF-κB activation and augmented inflammation but was paradoxically protective in ischemic AKI.

Abstract No. 34

EVEROLIMUS IN THE PREVENTION OF CALCINEURIN-INHIBITOR-INDUCED LEFT VENTRICULAR HYPERTROPHY IN HEART TRANSPLANTATION (RAD-TAC STUDY)

ANTHONY Chris, IMRAN Mohammad, EMMANUEL Sam, ILIFF James, KOTLYAR Eugene, MUTHIAH Kavitha, KEOGH Anne, HAYWARD Christopher, MACDONALD Peter, JABBOUR Andrew
Department of Cardiology, St Vincent's Hospital, Sydney

Purpose: To determine whether low-dose everolimus in combination with low-dose tacrolimus compared to full-dose tacrolimus without everolimus safely attenuates left ventricular hypertrophy after heart transplantation.
Method: In this prospective study, orthotopic heart transplant (OHT) transplant recipients were randomized at 12 weeks post-transplant to a combination of everolimus, tacrolimus (low dose), mycophenolate and prednisolone (RAD-TAC group) OR tacrolimus (normal dose) mycophenolate and prednisolone (TAC group). Left ventricular mass (LVM), mass index (LVMi), function and fibrosis markers (T1 mapping) were assessed by Cardiac MRI (CMR) at baseline and at 52 weeks post-transplant.
Results: Forty patients were randomized. Patient characteristics, including LVMi, age and gender were evenly matched between groups at baseline. Patients in the RAD-TAC group had significantly lower tacrolimus levels (5.5 (1.5) ug/L vs. 8. 3 (3.1) ug/L, mean (SD); p=0.03). The mean everolimus level was 6.8 (1.8) ug/L. LV Mass in the RAD-TAC group significantly reduced over the 40-week study period; in contrast, LV Mass in the TAC group increased (LV Mass change -11.1g (18.3) vs. +0.4g (6.3); p=0.001, LVMi change -6.1g/m2 (8.8) vs.+1.4g/m2 (5.8); p=0.001). Interstitial fibrosis (by CMR T1 mapping) in the RAD-TAC group significantly reduced over the 40-week period; in contrast, interstitial fibrosis in the TAC group increased (fibrosis change -31 (51) ms vs. +33 (-94) ms; p=0.002). No significant differences were observed in measures of myocardial function, total rejection episodes, hypertension (systolic BP 131 (9.2) mmHg vs. 132 (8.5) mmHg; p=0.7) or serious adverse events between groups.
Conclusion: Low-dose everolimus in combination with low-dose tacrolimus compared to full-dose tacrolimus safely attenuates left ventricular hypertrophy after heart transplantation. The reduction in CMR-determined fibrosis also provides insights into a potential mechanistic role of everolimus in the pathophysiology of cardiac hypertrophy after transplantation.

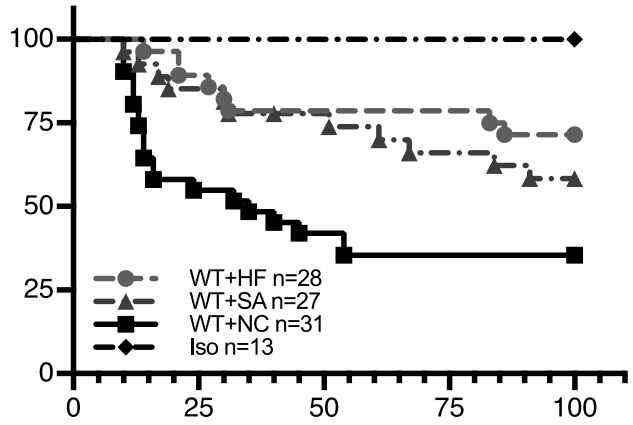
Abstract No. 35

HIGH FIBRE DIET PREVENTS KIDNEY ALLOGRAFT REJECTION AND PROTECTS AGAINST TRANSPLANT-ASSOCIATED DYSBIOSIS

SINGER Julian[1,2,3], WU Huiling[1,3,2], KWAN Tony[1,3], LOH Yik Wen[4,3], WANG Chuanmin[1,3], TAN Jian[1,3], LI Yan J[1], LAI Sum Wing Christina[1], MACIA Laurence[4,3], ALEXANDER Stephen I[5,3], CHADBAN Stephen J[1,2,3]

[1]Kidney Node Laboratory, Charles Perkins Centre, [2]Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney, [3]School of Medicine, University of Sydney, [4]Nutritional Immunometabolism Lab, Charles Perkins Centre, [5]Centre for Kidney Research, The Children's Hospital at Westmead, Sydney

Aim: To investigate the impact of dietary supplementation with high-fiber (HF), or the short-chain fatty acid sodium acetate (SA) on kidney allograft rejection in a murine model.
Methods: Kidney transplants were performed from BALB/c($H2^d$) to B6($H2^b$) or B6($H2^b$):$GPR43^{-/-}$ mice as allografts. Allograft mice received normal chow (WT+NC), a HF diet (WT+HF), or SA supplementation (WT+SA; $GPR43^{-/-}$+SA). Gut microbiota composition was assessed by 16S rRNA sequencing.
Results: WT+HF allografts had prolonged survival compared to WT+NC allografts (Figure 1, $p<0.01$), and were protected from acute (day 14: lower creatinine ($p<0.01$), less tubulitis ($p<0.001$)) and chronic (day 100: lower creatinine ($p<0.05$), less proteinuria ($p<0.01$) and glomerulosclerosis ($p<0.001$)) rejection. Transplantation led to dysbiosis in WT+NC mice, with gut microbial diversity decreased at day 14, but not in WT+HF mice where diversity was enhanced ($p<0.05$). Following transplant, bacteria known to produce SA and induce Tregs were enhanced (*Clostridiales* $p<0.0001$) or remained dominant in WT+HF mice (*Bifidobacterium* $p<0.05$) as compared to WT+NC animals. Similarly, WT+SA allografts exhibited superior survival to WT controls (Figure 1, $p<0.05$), were protected from rejection and exhibited donor specific tolerance confirmed by acceptance of donor strain but rejection of 3rd party skin grafts ($p<0.01$). The survival benefit conferred by SA was broken by depletion of CD25+ Tregs and was ineffective in $GPR43^{-/-}$ allograft recipients ($p<0.05$). **Conclusions:** HF diet prevented transplant-associated dysbiosis and afforded protection against allograft rejection. Protection was mediated, at least in part, by SA and was dependent on a CD4+CD25+Foxp3+ regulatory mechanism and signalling via GPR43.



Abstract No. 36

EFFECT OF LANGUAGE AND COUNTRY OF BIRTH ON MEDICAL SUITABILITY AND CONSENT IN SOLID ORGAN DONOR REFERRALS IN NEW SOUTH WALES 2010-2015 – A LINKED-DATA COHORT STUDY

WALLER Karen[1,2], HEDLEY James[1,3], DE LA MATA Nicole[1,3], ROSALES Brenda[1,3], WYBURN Kate[2,4], KELLY Patrick[1,3], O'LEARY Michael[5], CAVAZZONI Elena[5], WEBSTER Angela[1,3,6]

[1]Centre for Organ Donor Evidence, University of Sydney, [2]Sydney School of Medicine, Faculty of Health and Medicine, University of Sydney, [3]Sydney School of Public Health, Faculty of Health and Medicine, University of Sydney, [4]Renal Unit, Royal Prince Alfred Hospital, Sydney, [5]NSW Organ and Tissue Donation Service, [6]Centre for Transplant and Renal Research, Westmead Hospital, Sydney

Introduction: Culturally and linguistically diverse populations in Australia are over-represented on transplant waiting lists but under-represented in actual organ donor populations.

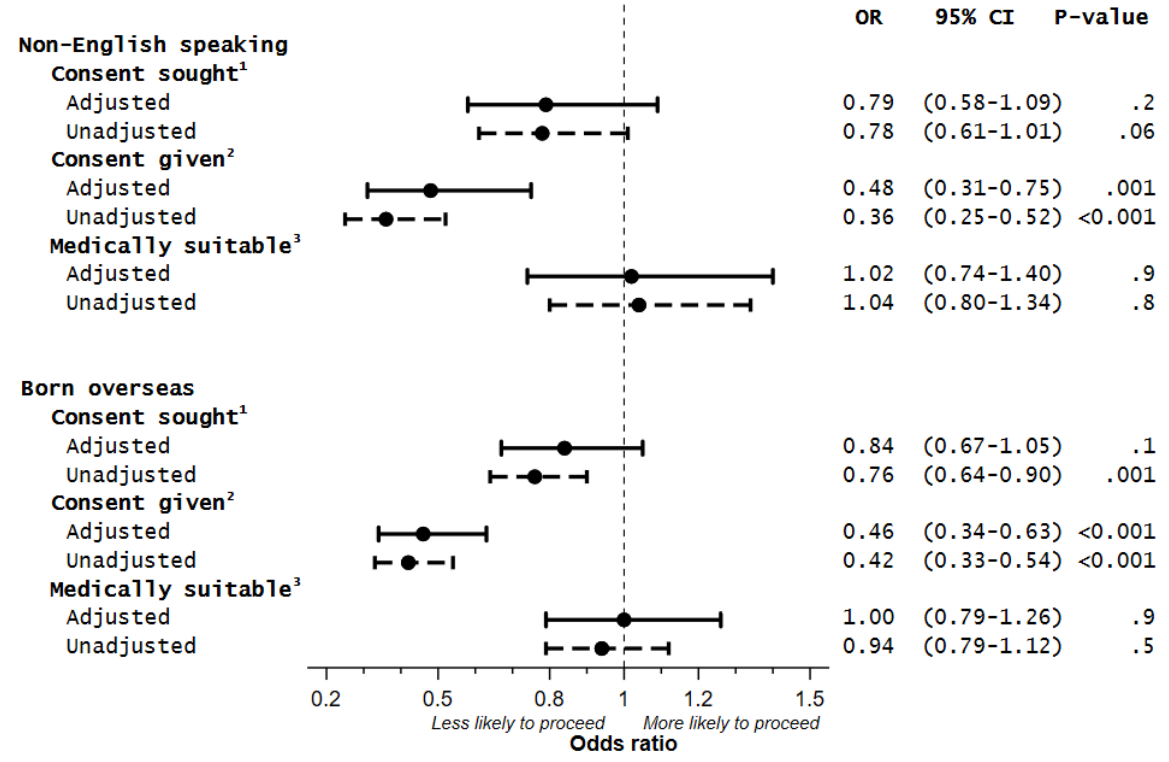
Aims: We sought to compare medical suitability and family consent outcomes between donor referrals based on primary language and country of birth.

Methods: We used linked-data from the NSW Biovigilance Register. This Register linked NSW donor referrals from 2010-2015 with the NSW Admitted Patient Data Collection and Emergency Department Data Collection. Effects of primary language (English vs. non-English) and country of birth (Australian vs. overseas born) on referral outcomes were determined using logistic regression (odds ratios with 95%CI).

Results: Of 2,957 referrals from NSW, 2,644 (89%) were likely to proceed (donor did not recover, no donor registered refusal, and no coroner refusal), and of these 2,383 (90%) had complete data for analysis. Family consent was sought for 1,302 (55%) and was granted for 846 (65%) referrals. There were 991 (42%) referrals medically suitable for donation. Language and country of birth were not associated with families being asked for consent ($p>0.1$) or being deemed medically suitable ($p>0.9$). Families of Non-English speakers were less likely to consent to donation if asked (adjusted OR 0.48; 95%CI 0.31-0.75; $p=0.001$). Families of overseas born referrals were also less likely to consent to donation (adjusted OR 0.46; 95%CI 0.34-0.63; $p<0.001$). There were no interaction effects ($p=0.4$).

Conclusions: Culturally and linguistically diverse referrals were less likely to obtain family consent to donation, but there were no differences in being asked for consent or medical suitability.

Figure 1: Forest plot of adjusted logistic regression results



[1] Adjusted for age, sex, cause of death, length of stay, cardiovascular disease, cancer, infection, smoking status, IV drug use, non-IV drug use, and high risk partner

[2] Adjusted for age, sex, cause of death, other same-day referrals, comorbidities, cardiovascular disease, respiratory disease, smoking status, IV drug use, and non-IV drug use

[3] Adjusted for age, sex, cause of death, cancer, infection, smoking status, IV drug use, non-IV drug use, and high risk partner

Abstract No. 37

INCIDENCE AND PREDICTORS OF INFECTIOUS-RELATED MORTALITY IN RECIPIENTS OF A KIDNEY TRANSPLANT: A REGISTRY STUDY

CHAN Samuel[1,2,3,4], PASCOE Elaine Mary[2,3,4], CLAYTON Philip Andrew[1,5,6], MCDONALD Stephen Peter[1,5,6], LIM Wai Hon[1,7], SYPEK Matthew Peter[1,5,6], PALMER Suetonia Cressida[8], ISBEL Nicole Maree[9,3], FRANCIS Ross Simon[9,3], CAMPBELL Scott Bryan[1,9,3], HAWLEY Carmel Mary[1,9,3,4], JOHNSON David Wayne[1,9,3,4]

[1]ANZDATA, [2]Princess Alexandra Hospital, Brisbane, [3]Australasian Kidney Trials Network, University of Queensland at the Princess Alexandra Hospital, [4]Translational Research Institute, University of Queensland at the Princess Alexandra Hospital, [5]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [6]School of Medicine, University of Adelaide, [7]Sir Charles Gairdner Hospital Unit, University of Western Australia, Perth, [8]Department of Medicine, University of Otago, Christchurch, New Zealand, [9]Department of Nephrology, Princess Alexandra Hospital, Brisbane

Background: The burden of infectious disease is high among kidney transplant recipients due to concomitant immunosuppression. In this study, the incidence of infectious-related mortality and associated factors were evaluated.

Methods: In this registry-based retrospective, longitudinal cohort study, recipients of a first kidney transplant in Australia and New Zealand between 1997-2015 were included. Cumulative incidence of infectious-related mortality was estimated using competing risk regression (using non-infectious mortality as a competing risk event), and compared with age and sex-matched populated-based data using standardised incidence ratios (SIR).

Results: Among 12,519 patients, (median age 46yrs, 63% male, 15% persons with diabetes, 6% Indigenous), 416 (3.3%) died from infection. Infection-related mortality reduced over time from 15.6 per 100,000-person-years in 1997-2000 to 4.7 per 10,000-person-years in 2011-2015 ($p<0.001$). Compared with the age-matched general population, kidney transplant recipients had a markedly higher risk of infectious-related death (SIR 7.8, 95%CI 7.1-8.6). Infectious mortality was associated with older age (≥60yrs adjusted subdistribution hazard ratio [SHR] 4.16, 95%CI 2.15-8.05; reference 20-30yrs;), female sex (SHR 1.62, 95%CI 1.19-2.29), Indigenous ethnicity (SHR 2.87, 95%CI 1.84-4.46; reference Caucasian), earlier transplant era (2011-2015 SHR 0.39, 95%CI 0.20-0.76; reference 1997-2000), and use of T-cell depleting therapy (SHR 2.43, 95%CI 1.36-4.33). Live donor transplantation was associated with lower risk of infectious-related mortality (SHR 0.53, 95%CI 0.37-0.76).

Conclusions: Infectious-related mortality in kidney transplant recipients is significantly higher than the general population, but has reduced over time. Risk factors include older age, female sex, Indigenous ethnicity, T-cell depleting therapy and deceased donor transplantation.

Abstract No. 38

A LONGITUDINAL STUDY OF GAMMA DELTA T CELLS POST LUNG TRANSPLANT: POTENTIAL PLAYERS IN IMMUNITY TO CYTOMEGALOVIRUS

SULLIVAN Lucy[1,2], SHAW Evangeline[1], STANKOVIC Sanda[1], SNELL Gregory[2], BROOKS Andrew[1], WESTALL Glen[2]

[1]Microbiology and Immunology at The Peter Doherty Institute for Infection and Immunity, University of Melbourne, [2]Lung Transplant Service, Alfred Hospital, Melbourne

Aims: Cytomegalovirus (CMV) reactivation in lung transplant recipients is linked with chronic rejection and there is a need to develop biomarkers that indicate CMV immunity. To this end, we aimed to assess the role of γδ T cells in CMV immunity following lung transplantation.

Methods: Peripheral blood mononuclear cells (PBMC) were isolated from lung transplant patients from the Alfred Hospital by written informed consent at pre-transplant, 0.5, 1.5, 3, 6, 9, 12- and 18-months post-transplant. Patients were stratified based on the risk of CMV disease into low risk (CMV donor (D) negative (-), CMV recipient (R) -, n=6), moderate risk (CMV R positive (+), n=13) or high risk (CMV D+, R-, n =12). CMV reactivation was classified as CMV PCR positive (>150 copies/ml) in blood and/or BAL within the first 12-months post-transplant. The phenotype of γδ T cells in PBMC was assessed by multi-colour flow cytometry and compared to healthy controls (n=12).

Results: Over the post-transplant period, there was an increase in the proportion of Vδ1 γδ T cells all lung transplant groups. There was a higher expression of NKG2C and CD16 on γδ T cells of moderate risk recipients without CMV reactivation.

Conclusions: Our results showed that subsets of γδ T cells are linked with the control of CMV following lung transplantation. The NK cell receptor CD94-NKG2C could be important in the control of CMV on multiple cell types. We demonstrated that γδ T cells may have utility as a biomarker of CMV immunity following lung transplantation.

Abstract No. 39

DOES EARLY REMOVAL OF URETERIC STENT SIMULTANEOUSLY WITH INDWELLING URETHRAL CATHETER POST KIDNEY TRANSPLANTATION REDUCE INFECTION RATES AND HEALTHCARE COSTS?

TRAPNELL FRANK[1], HEIN Martin[1], MOU Lingjun[2], JAQUES Bryon[2,1], BOUDVILLE Neil[3,1], CHAKERA Aron[3,1], HE Bulang[2,1]

[1]School of Medicine, Faculty of Health Sciences, University of Western Australia, Perth, [2]Renal Transplant Unit, Sir Charles Gairdner Hospital, Perth, [3]Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth

Aim: Ureteric stents have proven efficacy in reducing major urological complications (MUCs) when inserted prophylactically during kidney transplantation. However, ureteric stents are associated with an increased risk of UTIs and require an additional procedure for removal. It is theorised that an earlier stent removal time may reduce UTI risk whilst still protecting against MUCs. This research aims to investigate if early ureteric stent removal at 4-5 days post-transplantation results in a significant reduction in UTIs and health care costs, without a significant increase in MUCs.

Methods: This is a single centre prospective study, with a retrospective control group. The control group has received a kidney transplant within the past 2 years, and had their ureteric stents removed 4-6 weeks post transplantation with cystoscopy. The prospective group will have their ureteric stents removed 4-5 days post-transplantation using a non-cystoscopic traction technique (Figure 1) aiming for a 20% UTI reduction. Both cohorts will consist of 80 participants with a 12 month follow up.

Results: Preliminary data from 14 early removal participants and 35 control participants demonstrates significantly lower UTI rates in the prospective early removal group (0% vs 36%, p=0.01). Thus far, one ureteric stenosis and one urine leak has been recorded in the control group and zero in the early removal group. Estimated savings of $713AUD per case have been achieved.

Conclusion: Preliminary data demonstrates that early non-cystoscopic ureteric stent removal may reduce the incidence of UTIs and reduce costs without increasing the risk of urological complications.

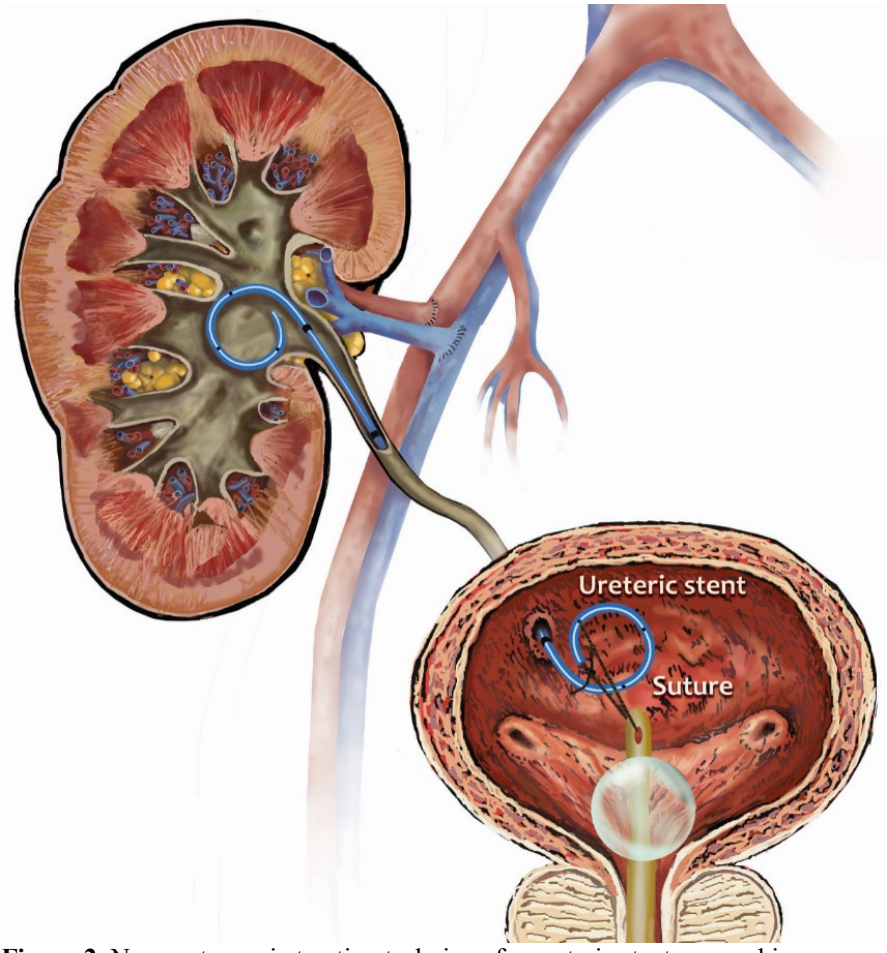


Figure 2. Non-cystoscopic traction technique for ureteric stent removal in prospective early removal group**.**

Abstract No. 40

CARDIAC MORTALITY IN TRANSPLANT PATIENTS; A POPULATION-BASED COHORT STUDY 1988-2013 IN AUSTRALIA AND NEW ZEALAND

WYLD Melanie[1,2], DE LA MATA Nicole[3], MASSON Philip[4], O'LONE Emily[5,3], WEBSTER Angela[6,3]

[1]Renal Unit, Royal Prince Alfred Hospital, Sydney, [2]Sydney Medical School, University of Sydney, [3]School of Public Health, University of Sydney, [4]Renal Unit, Royal Free London NHS Foundation Trust, [5]Renal Unit, Royal North Shore Hospital, Sydney, [6]Renal Unit, Westmead Hospital, Sydney

Background: Transplant recipients experience excess cardiac mortality. We compared cardiac death rates in Australian and New Zealand kidney transplant recipients to the general population and identified risk factors for cardiac death in kidney transplant recipients.

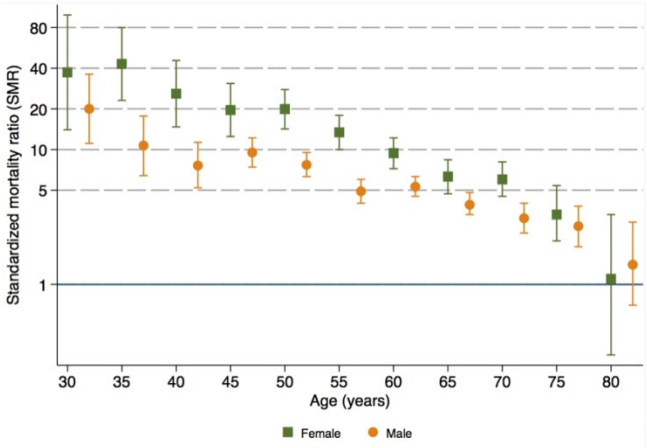
Methods: Primary cause of death for kidney transplant recipients was established through ICD-10 codes and data linkage between ANZDATA and national death registers. We used indirect standardisation to estimate standardised mortality ratios(SMR) and Fine-Gray competing risks models to determine risk factors for cardiac mortality (subhazard ratios, SHR).

Results: During 1988-2013, there were 5286 deaths in 17,628 kidney transplant recipients over 175,084 person-years of follow up. 936(17.8%) of these deaths were cardiac. The crude cardiac death rate halved from >800/100,000 person-years in the early 1990s to <400/100,000 person-years in the 2010s ($p<0.001$). The cardiac SMR was 5.46(95%CI: 5.12, 5.82). SMRs fell from a high of 16.92(95%CI: 9.61, 29.80) in 1984 to 4.03(95%CI: 4.03, 7.05) in 2013($p<0.001$). Females had significantly *higher* relative cardiac mortality than men at all age groups until age 75 when the differences were no longer statistically significant(Figure 1).

Risk factors for cardiac death included male sex(SHR 1.61,95%CI:1.39,1.85), age >55(SHR 2.97,95%CI:2.48,3.55), >36 months on dialysis SHR 1.63,95%CI:1.30,2.05), pre-existing coronary artery disease(SHR 1.49,95%CI:1.20,1.85), and either diabetes(SHR 1.6,95%CI:1.28, 2.0) or hypertension(SHR 1.53,95%CI:1.16,2.02) as the cause of kidney failure.

Conclusions: Kidney transplant recipients have greatly increased risk of cardiac mortality compared to the general population. Excess risk is much higher for females than males suggesting cardiac risks may be under-recognized, and/or prevention and treatment interventions less effective in females with ESKD.

Figure 1: Cardiac SMRs by age and sex



Abstract No. 41

ASSOCIATION BETWEEN THE SIDE OF LIVE DONOR KIDNEYS AND TRANSPLANT RECIPIENT OUTCOMES

DOBRIJEVIC Ellen[1,2], WONG Germaine[2,3], CLAYTON Phil[4,5], ALLEN Richard[6,2]

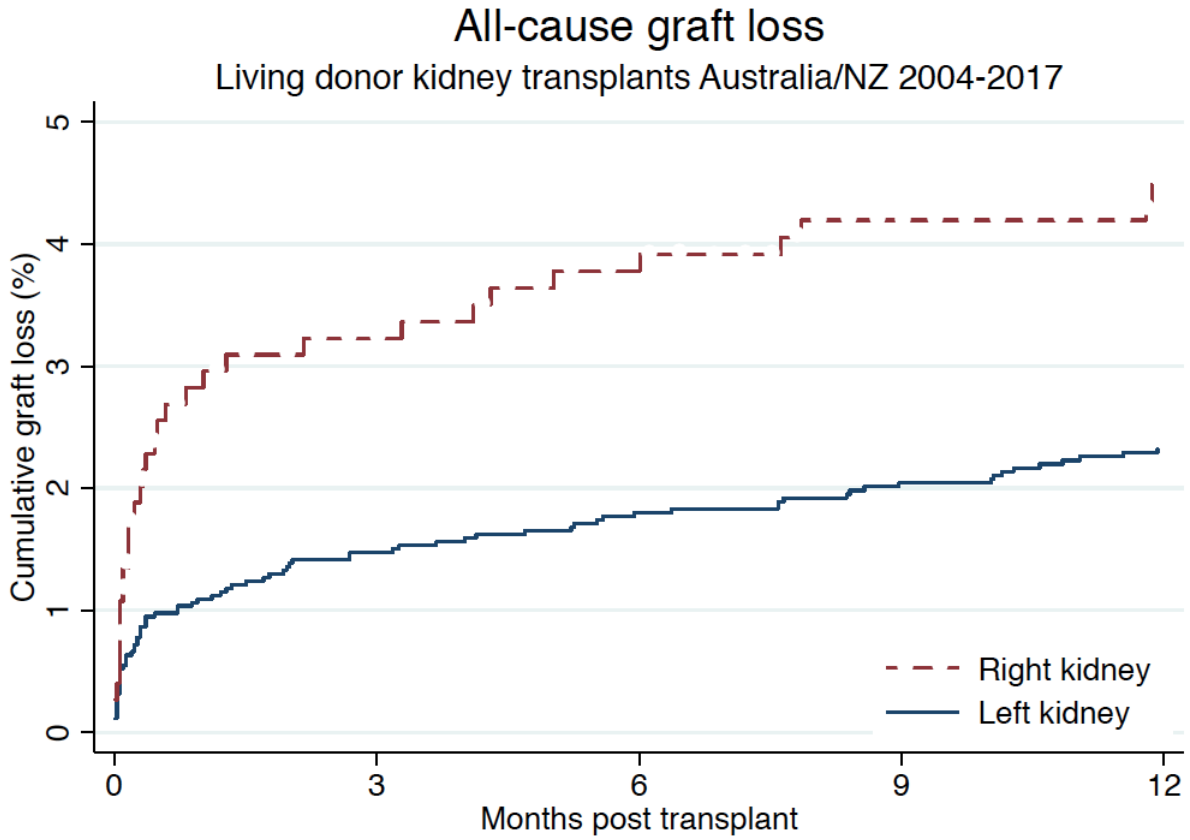
[1]School of Medicine, University of Sydney, [2]Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney, [3]School of Public Health, University of Sydney, [4]ANZDATA, [5]Nephrology and Renal Transplant, Royal Adelaide Hospital, [6]Department of Surgery, University of Sydney

For ease of surgery, donor and recipient surgeons prefer left-sided living donor kidneys (LDKs). **Aim**: Determine the impact of left versus right LDKs on recipient outcomes, including delayed graft function (DGF), death-censored graft loss (DCGL) and overall graft survival.

Methods: Using ANZDATA and ANZOD registry data, a piecewise multivariable Cox regression model was performed using recipient data matched with donor details.

Results: 4,222 LDKs were transplanted into adult recipients in 25 centers from 2004-2017 and followed for a median of 6.5 years, of which 17.6% were right kidneys. Overall graft survival (%) (95%CI) at one and five years after transplantation were 97.3 (96.8-97.8) and 89.2 (88.1-90.2). Incidence of DGF was 2.7% for left versus 5.7% for right (p<0.001). 59 (1.4%) LDKs were lost within 30 days of transplantation. Nine deaths occurred in the first month, 6 from cardiovascular events. Grouped causes of graft loss within 30 days of transplantation were technical (59.3%), non-technical (25.4%) and death with functioning graft (15.3%). Compared with left donor kidneys, adjusted HR (95%CI) for DCGL among recipients of right kidneys was 2.45 (1.31-4.57), within 30 days after transplantation, and 0.89 (0.67-1.18) beyond 30 days. Within 30 days after transplantation, primary non-function (PNF) accounted for 7.9% of left kidneys lost compared to 28.6% of right kidneys (p=0.05).

Conclusion: Adult right LDKs recipients experience an increased risk of PNF, DGF and DCGL within 30 days of transplantation. This information should be included in the recipient patient consent process and factored into immunosuppression trial analyses.



Abstract No. 42

LONG-TERM OUTCOMES OF UTILISING DONATION AFTER CIRCULATORY DEATH GRAFTS IN LIVER TRANSPLANTATION – AN AUSTRALIAN 12-YEAR COHORT STUDY

SASTRY Vinay, PANDYA Keval, PANLILIO Mara, WEST Claire, VIRTUE Susan, WELLS Mark, CRAWFORD Michael, PULITANO Carlo, STRASSER Simone, MCCAUGHAN Geoff, MAJUMDAR Avik, LIU Ken

Australian National Liver Transplantation Unit, Royal Prince Alfred Hospital, Sydney

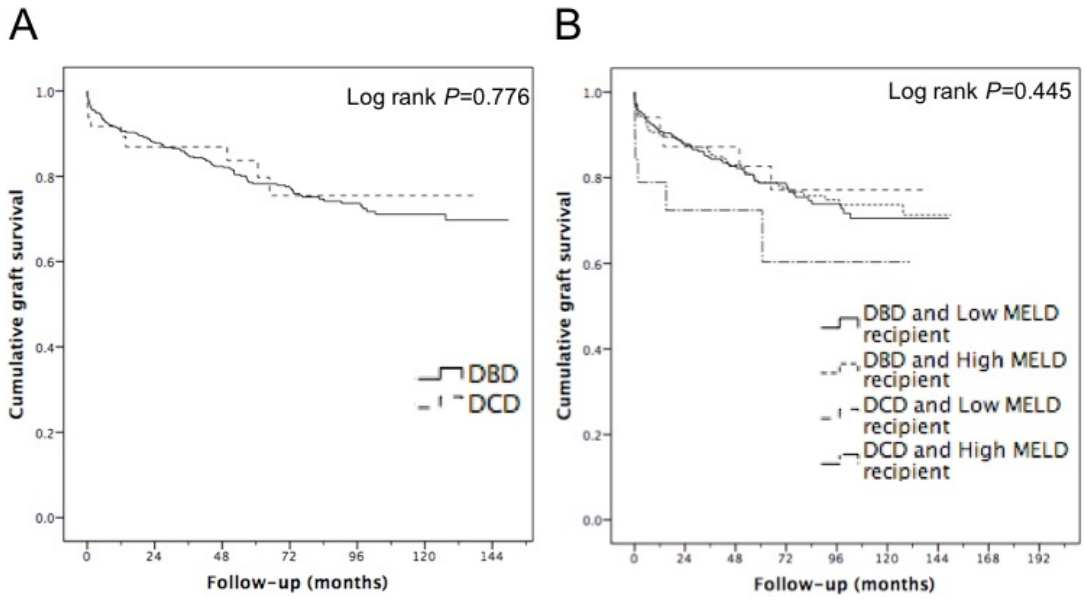
Aims: Use of donation circulatory death donors (DCDs) has been one strategy to expand the donor pool, however data on long-term outcomes in recipients of DCD grafts are mixed. We studied the characteristics, utilisation and recipient outcomes of DCDs.

Methods: We retrospectively studied consecutive adults who underwent deceased-donor LT between 2006-2018. Donor and recipient data at time of LT and recipient outcomes were collected. The primary outcome of interest was graft survival (time to re-transplantation or death).

Results: During the study period, 739 donors were utilised for LT. Of these, 53 (7.2%) were DCDs. Compared to donation after brain death donors (DBDs), DCDs were younger (30vs.50years), more likely to have history of pre-donation cardiac arrest (71.2%vs.34.8%), had longer intubation time (3vs.2days), less inotrope requirements (32.7%vs.7.8% on no agents) and higher AST (59vs.46U/L) (median values presented, $P<0.01$ for all). DCDs also had shorter cold ischaemia time (5.75vs.6.85hours,$P=0.005$) and higher donor risk index (1.68vs.1.56, $P<0.001$). As per our unit policy, recipients of DCD grafts were less likely to be PSC (1.9vs.9.0%, $P=0.076$) or re-transplant patients (0vs.6%, $P=0.067$). Similarly, DCD recipients did not receive split grafts (0vs.15.3%, $P<0.001$). The proportion of DCDs among utilised grafts increased from 4.8%(2006-2009) to 7.8%(2010-2012) and remained stable afterwards. DCD grafts had similar long-term graft survival compared to DBDs, although DCDs recipients with high pre-LT MELD scores>20 appeared to have worse outcomes (**Figure1**).

Conclusion: Long-term outcomes of DCD grafts are similar to DBD grafts especially when matched with appropriately selected recipients (first transplant, non-PSC patients with low MELD<20).

Figure 1



Abstract No. 43

LONG-TERM OUTCOMES OF UTILISING EXTENDED CRITERIA DECEASED DONORS IN LIVER TRANSPLANTATION – AN AUSTRALIAN 12-YEAR COHORT STUDY

PANDYA Keval, SASTRY Vinay, PANLILIO Mara, WEST Claire, VIRTUE Susan, WELLS Mark, CRAWFORD Michael, PULITANO Carlo, STRASSER Simone, MCCAUGHAN Geoff, MAJUMDAR Avik, LIU Ken

Australian National Liver Transplantation Unit, Royal Prince Alfred Hospital, Sydney

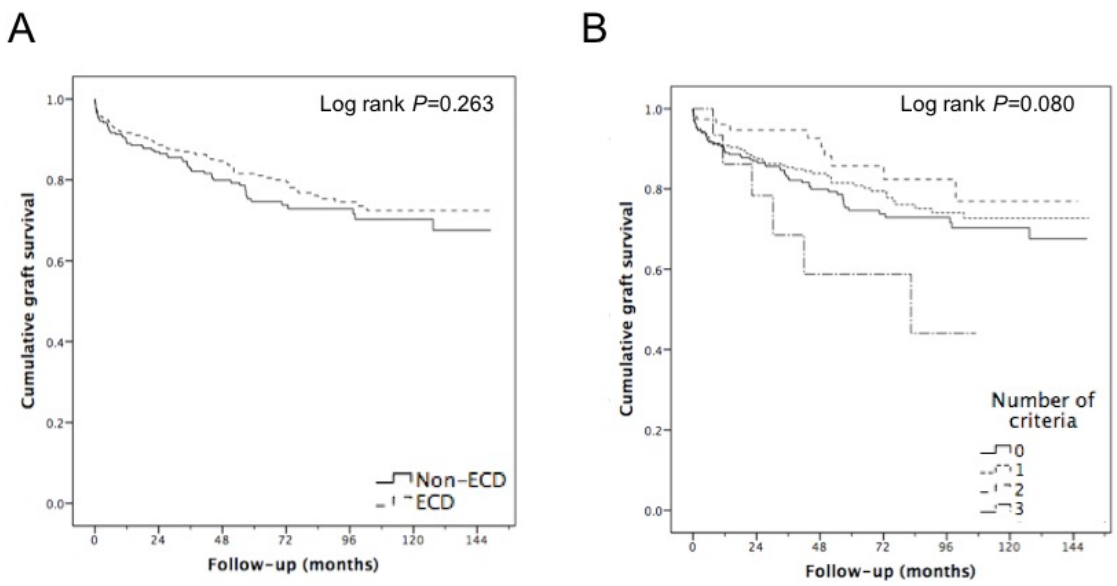
Aims: Use of extended criteria donors (ECDs) has been one strategy to increase the donor pool. Long-term outcomes in recipients of ECD livers have not been well studied. We studied the characteristics, utilisation and recipient outcomes of ECDs.

Methods: We retrospectively studied consecutive adults who underwent deceased-donor LT between 2006-2018. Donor and recipient data at time of LT and recipient outcomes were collected. ECD was defined using Eurotransplant criteria. The primary outcome of interest was graft survival (time to re-transplantation or death).

Results: During the study period (median follow-up 50.6months), 739 donors were utilised for LT. Of these, 432 (58.4%) were ECDs. Elevated ALT/AST was the most common ECD criterion (31.5%), followed by age>65 (16.5%) and BMI>30 (16.0%). ECDs were older (51vs.47years), with higher BMI (26.4vs.24.5kg/m^2), transaminases (ALT 42vs.24U/L) and graft steatosis (11.4%vs.0% ≥S2) compared to non-ECDs (median values presented, P<0.001 for all). ECDs had worse renal function (creatinine 80vs.67umol/L, P<0.001) and higher donor risk index (1.65vs.1.52, P<0.001). There were no differences in recipient characteristics of ECD vs. non-ECD grafts except ECD recipients were less likely receive a split graft (10.9%vs.18.9%, P=0.002). The proportion of ECDs among utilised grafts did not change over time. 157 patients experienced graft loss during follow-up (31 re-transplants, 126 deaths). ECDs had similar long-term graft survival compared to non-ECDs, although outcomes appeared to be worse when ≥3 criteria were met (**Figure1**).

Conclusion: ECD livers meeting up to 2 Eurotransplant criteria can be safely used without impacting long-term graft survival. This has implications for organ utilisation.

Figure 1



Abstract No. 44

IGLS CRITERIA APPLIED TO THE AUSTRALIAN ISLET TRANSPLANT PROGRAM.

KAY Thomas[1], GOODMAN David[2], LOUDOVARIS Thomas[3], RADFORD Toni[4], ANDERSON Patricia[5,6], HOWE Kathy[2], COATES Toby[7], O'CONNELL Philip[5,6]

[1]St Vincent's Institute, Melbourne, [2]Department of Nephrology, St Vincent's Hospital, Melbourne, [3]Islet Transplantation Facility, St Vincent's Institute, Melbourne, [4]Royal Adelaide Hospital, [5]Centre for Transplant and Renal Research, Westmead Hospital, Sydney, [6]Westmead Millennium Institute, Westmead Hospital, Sydney, [7]Department of Medicine, Royal Adelaide Hospital

The Igls criteria have been proposed as a way of documenting outcome of islet transplantation that is simple and clinically useful and can be compared to other treatments including both transplant and technology approaches. **Aims:** We aimed to apply the Igls criteria to the database for the Australian Islet Transplant Consortium and to use the criteria as a way to define outcomes of transplants carried between 2007 and 2018. In this period the Consortium carried out transplants in three centres and islet isolations in two centres. A total of 105 islet infusions were done into 50 recipients.

Results: 12 months after the initial islet infusion 71% (30/42) of recipients met the Igls criteria for a successful transplant ie with either an "optimal" or "good" outcome. The percent successful was 64%(25/39) at 2y, 50% (16/32) at 3y, 54% (15/28) at 4y, 42% (8/19) at 5y and 62% (8/13) at 8y. In addition CGM parameters were recorded and compared with optimal and good outcomes. As expected CGM parameters were improved most in the "optimal" group.

	Mean glucose	sd glucose	%>7.8	3.9-7.8	%<3.9
pre	8.20	3.49	38.93	45.47	10.73
optimal	6.83	1.33	17.96	80.83	1.26
good	7.40	2.16	34.53	58.28	5.89
marginal	8.80	3.49	56.71	36.29	6.71

Conclusion: In the majority of T1D patients receiving an islet transplant for severe hypoglycaemia with hypoglycaemia unawareness, HbA1c of <7% and resolution of hypoglycaemia can be achieved and in most cases this is stable for several years.

Abstract No. 45

FACTORS INFLUENCING ISLET ISOLATION OUTCOMES: 20-YEAR DATA FROM THE WESTMEAD ISLET TRANSPLANT PROGRAM

CHEW Yi Vee[1], WILLIAMS Lindy[1], BURNS Heather[2], JIMENEZ-VERA Elvira[1], HUANG Dandan[2], O'CONNELL Philip[1], HAWTHORNE Wayne[1,3]

[1]Centre for Transplant and Renal Research, Westmead Hospital, Sydney, [2]Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney, [3]Department of Surgery, Westmead Hospital, Sydney

Aims: Successful islet cell isolation to treat type 1 diabetes is influenced by multiple factors including donor selection, organ procurement and isolation parameters. This study aimed to identify factors that affected the outcome of an islet preparation in order to proceed to transplantation.

Methods: Islets were isolated from DBD donor pancreata using collagenase and neutral protease (SERVA). Donor characteristics, procurement data, isolation yield and outcomes were analysed to determine variables associated with transplantable yields. Data was further divided into Transplanted (Tx) and Non-transplanted (NTx) to identify factors significantly related to successful outcomes.

Results: Data collected from 250 islet isolations between July 2000 and February 2019 were evaluated. On average, 28% of islet preparations were transplanted, with 54% of isolations in 2016-2019 reaching release criteria. Transplantable yields (defined as 300,000 IEQ; 4,000 IEQ/kg for a 75kg recipient) were obtained from donors aged between 20-60 years, with BMI >20kg/m^2, and weight >60kg. Cold ischaemia times exceeding 10hrs were found to negatively affect isolation yields.

Compared to NTx (n=180), Tx (n=70) had significantly higher total IEQ (573,977±29,352 VS 325,563±14,625 IEQ) and IEQ/g pancreas (7,018±388 VS 5,085±274 IEQ/g). The Tx group was significantly associated ($p<0.01$) with higher donor weight, BMI, pancreas weight, and lower CIT compared to NTx. Tx islets also exhibited significantly higher viability, purity, beta cell viability and stimulation indices compared to Non-tx ($p<0.05$).

Conclusion: A focus on increased donor BMI/weight and lower CIT would contribute significantly to successful islet isolation outcomes, resulting in transplantable yields of islets for treatment of Type 1 diabetes.

Abstract No. 46

A SYSTEMATIC REVIEW AND META-ANALYSIS TO IDENTIFY THE RISK FACTORS FOR PANCREATIC ALLOGRAFT THROMBOSIS FOLLOWING SIMULTANEOUS PANCREAS-KIDNEY TRANSPLANTATION

BLUNDELL Jian[1,2], SHAHRESTANI Sara[1,2], LENDZION Rebecca[2], HAWTHORNE Wayne[1,2,3]

[1]School of Medicine, University of Sydney, [2]Department of Surgery, Westmead Hospital, Sydney, [3]Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney

Simultaneous pancreas-kidney (SPK) transplantation remains the most effective treatment option for achieving consistent and long-lasting euglycaemia in Type 1 diabetic patients with associated renal failure. Thrombosis of the pancreatic vasculature continues to contribute significantly to early graft failure and loss.

Aim: To compare the rate of thrombosis to graft loss, and systematically review the risk factors for early thrombosis of the pancreas allograft following SPK transplantation.

Method: We systematically searched the MEDLINE, EMBASE, The Cochrane Library and PREMEDLINE databases for studies reporting thrombosis following pancreas transplantation, and additional studies were compiled through relevant reference lists. Identified publications were screened for inclusion and synthesised accordingly into a data extraction sheet. The Newcastle-Ottawa Scale was used to appraise included studies. 51 studies satisfied the eligibility criteria; 32 cohort studies, 18 conference abstracts and one meta-analysis. Bias assessment revealed well conducted cohort studies of low bias risk.

Results: Meta-analysis of these studies revealed a 9% event rate for thrombosis, and 7% rate of graft loss secondary to thrombosis. This review established significant associations between donor and recipient characteristics, procurement and preservation methodology, transplantation technique, post-operative management and an increased risk of early thrombosis in the pancreas allograft.

Conclusion: Further investigation into HTK preservation fluid, prophylactic heparin protocol and exocrine drainage method is necessary to clarify their thrombotic influence on the pancreas graft. By continuing to investigate these contributory factors, it is hoped that the high thrombosis rates plaguing pancreas transplants can be appropriately addressed, ultimately resulting in improved patient outcomes following SPK transplantation.

Abstract No. 47

RECOMBINANT SOLUBLE CR1 TREATMENT IS PROTECTIVE IN A MOUSE MODEL OF RENAL ISCHEMIA REPERFUSION INJURY

BONGONI Anjan K[1], MCRAE Jennifer L[1], SALVARIS Evelyn J[1], VIKSTROM Ingela[2], MORELLI Adriana Baz[2], WYMANN Sandra[2], HARDY Matthew P[2], PEARSE Martin J[2], COWAN Peter J[1,3]

[1]Immunology Research Centre, St Vincent's Hospital, Melbourne, [2]CSL Ltd. Melbourne, [3]Department of Medicine, University of Melbourne

Background: Complement, an arm of the innate immune system, is a potent mediator of ischemia-reperfusion (IR) injury (IRI), which significantly affects function and survival of transplanted kidneys. A recombinant truncated and soluble form of complement receptor type 1 (sCR1) with complement inhibitory activity has been generated as a potential therapeutic candidate.

Aims: To test sCR1 as a potential agent for the suppression of complement-mediated IR-induced organ damage in a mouse model of renal IRI.

Methods: Male 10-12 week-old C57BL/6 mice were subjected to right nephrectomy and 22 minutes left renal ischemia at 37ºC. Mice (n=8-14/group) were treated with i.p. administration of 60 mg/kg sCR1, or vehicle, 1 hr prior to ischemia. Mice were sacrificed 24 hrs after reperfusion, and blood and kidney samples were collected to assess renal function (serum creatinine, urea), complement activation (plasma C3b, C5a) and deposition (C9), and neutrophil and macrophage infiltration.

Results: Compared to Sham, severe renal injury was induced following IR in the vehicle-treated mice as indicated by significantly increased serum creatinine and urea, plasma C3b and C5a, and tissue C9 deposition, and cellular infiltration. sCR1 treatment significantly protected against IR-induced damage, manifested by significantly lowered renal dysfunction, complement activation and deposition and cellular infiltration. **Conclusion:** Complement inhibition using sCR1 protected against IR-mediated renal damage and this was associated with markedly reduced renal dysfunction, as well as lowered complement activation and deposition, and cell infiltration. Blockade of complement activation by sCR1 is thus a promising therapeutic approach to reduce IRI and improve organ transplant function.

	Sham	Vehicle	sCR1	p value (Vehicle vs. sCR1)
Creatinine (μM)	18.5±1.1	181.1±36.2	64.9±72.4	0.003
Urea (mg/dL)	55.5±6.3	384.8±52.5	142.4±145.1	0.02
Plasma C3b (AU/ml)	725.0±239.5	2681.0±478.6	1576.0±526.9	0.009
Plasma C5a (ng/mL)	42.9±15.8	388.8±104.1	267.3±93.7	0.03
C9 deposition (RawIntDen)	$0.3\pm0.9\times10^6$	$7.2\pm1.7\times10^6$	$4.3\pm2.2\times10^6$	0.02
Neutrophils (counts/HPF)	2.0±1.0	55.0±11.0	37.0±16.0	0.03
Macrophages (counts/HPF)	2.0±1.0	52.0±10.0	33.0±18.0	0.03

Abstract No. 48

NECROPTOSIS IN RENAL ISCHAEMIA REPERFUSION INJURY

PEFANIS A[1,2], NACHBUR U[3], MCRAE JL[2], BONGONI AK[4], SALVARIS EJ[2], MURPHY JM[5,6], IERINO FL[1,7], COWAN PJ[2,1]

[1]Department of Medicine, University of Melbourne, [2]Immunology Research Centre, St Vincent's Hospital, Melbourne, [3]Cell signalling and cell death division, Walter and Eliza Hall Institute of Medical Research, Melbourne, [4]Immunology Research Centre, St Vincent's Institute, Melbourne, [5]Cell Signalling, Walter and Eliza Hall Institute of Medical Research, Melbourne, [6]Department of Medical Biology, University of Melbourne, [7]Department of Nephrology, St Vincent's Hospital, Melbourne

Background: Ischemia-reperfusion injury (IRI) negatively impacts graft outcomes in kidney transplantation. Necroptosis, a form of regulated necrosis, is triggered by death receptor-mediated recruitment of the kinases RIPK1 and RIPK3 and activation of the pseudokinase MLKL. Active MLKL causes cell death by plasma membrane permeabilisation, driving "necroinflammation".

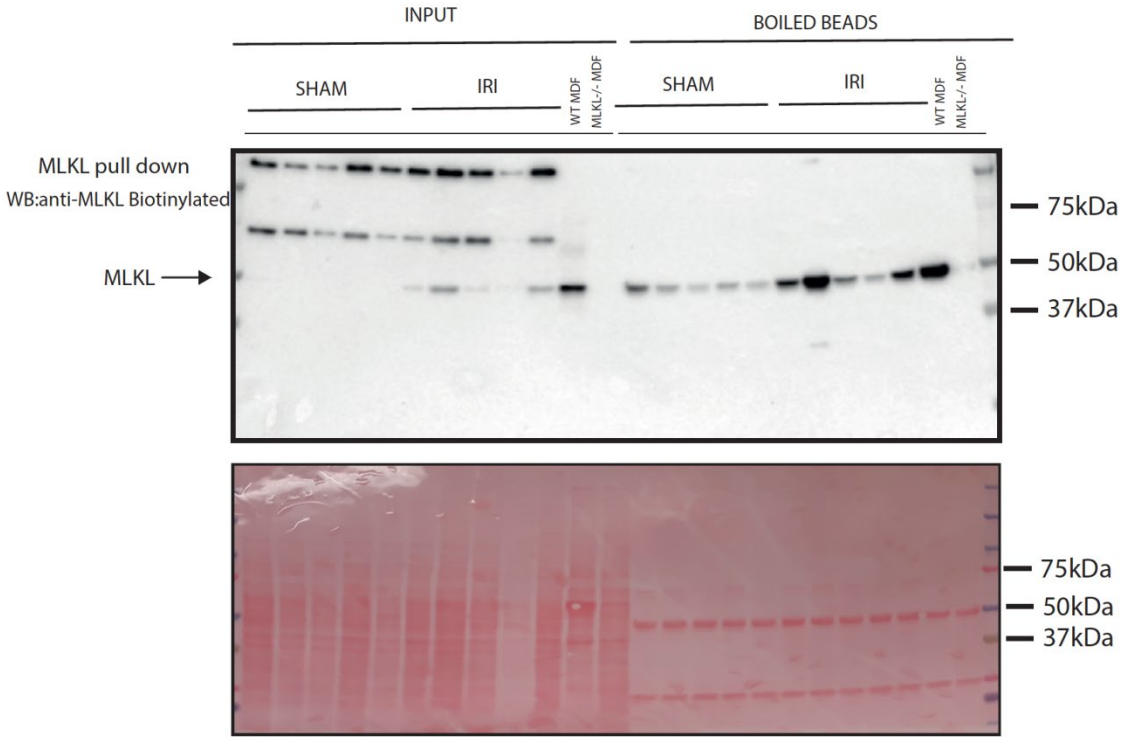
Aim: To investigate the role of necroptosis in a mouse model of renal IRI.

Methods: 10-12 week old male mice (MLKL knockout and wild-type littermates) underwent right nephrectomy and 22 min left renal ischemia. A separate cohort of WT mice were treated before ischemia with the novel RIPK1 inhibitor Nec-1s. Samples were collected at 24 hrs to assess MLKL expression (Western blot) and renal injury (serum creatinine, μM).

Results: MLKL was upregulated following IRI in WT mice (Figure 1). Creatinine was not significantly different in MLKL KO and WT mice (116.76±14.24 vs 127.82±14.43, p=0.59; Sham 19.50±0.72). Creatinine in Nec-1s-treated mice was significantly higher than in vehicle-treated mice (179±18.26 vs 107.17±22.27, p=0.034; Sham 18.29±0.29).

Conclusion: The upregulation of MLKL suggests that necroptosis may contribute to renal IRI. However, serum creatinine was not reduced in MLKL KO mice, possibly reflecting the temporal relationship between MLKL upregulation and this readout of kidney injury. We hypothesise that the increase in serum creatinine upon RIPK1 inhibition may be a consequence of RIPK1's pleotropic nature, as it can stimulate both cell death and cell survival pathways. Histological assessment of injured kidneys and variation of experimental settings will be performed to further investigate the contribution of necroptosis to renal IRI.

Figure 1. Western Blot assessing for MLKL in mice kidneys following IRI and sham procedures.



Abstract No. 49

SELECTIVE RETENTION OF DONOR MYELOID CELLS IN CONGENIC LIVER TRANSPLANTS

DART SJ[1], PROSSER A[1,2], HUANG WH[3], LIU L[1], DE BOER B[4], JEFFREY G[1], DELRIVIERE L[3,5], KALLIES A[6], LUCAS M[1,7]

[1]School of Medicine, University of Western Australia, Perth, [2]School of Human Sciences, University of Western Australia, Perth, [3]School of Surgery, University of Western Australia, Perth, [4]Department of Anatomical Pathology, Fiona Stanley Hospital, PathWest, Perth, [5]WA Liver & Kidney Transplant Service, Sir Charles Gairdner Hospital, Perth, [6]Department of Microbiology and Immunology, The University of Melbourne, The Peter Doherty Institute for Infection and Immunity, Melbourne, [7]Department of Immunology, Sir Charles Gairdner Hospital, PathWest, Perth

Introduction: Transplantation of solid organs involves the simultaneous transfer of donor immune cells, and myeloid cells play important roles in transplantation outcome. However, the dynamics between donor and recipient myeloid cells and their retention over time has not been investigated in detail using congenic models.
Aims: To characterise and quantify donor and recipient myeloid cell populations in congenic liver transplantation.
Methods: Orthotopic liver transplants were performed between congenically matched mouse strains, expressing either CD45.1 or CD45.2. Leukocytes from donor ($CD45.1^+$) and recipient ($CD45.2^+$) mice were analysed by multi-parameter flow cytometry to quantify M1 and M2 macrophages, $CD11b^-$ and $CD11b^+$ dendritic cells, eosinophils, monocytes and neutrophils at 0, 1, 4, 7 and 28 days post-transplantation in the graft and peripheral lymphoid organs.
Results: Following transplantation, the number of donor myeloid cells in the graft rapidly decreases. $CD11b^+$ dendritic cells, eosinophils and M1 macrophages are depleted to less than 98% of their total number at day 0. However, $CD11b^-$ dendritic cells, neutrophils, M2 macrophages and monocytes are retained up to day 28 at 10-30% of their total baseline number. Post-transplantation, the liver is infiltrated by all analysed subsets of recipient myeloid cells. Recipient neutrophils and M1 macrophages are the predominant myeloid populations in the liver on day 1 and day 28 post-transplantation.
Conclusions: Following congenic liver transplantation there is selective retention of donor myeloid cell populations within the transplanted organ and periphery. The level of donor cell retention may be important in transplantation outcome, thus further studies with mismatch models are underway.

Abstract No. 50

THE P2X7 ANTAGONIST BRILLIANT BLUE G PRESERVES REGULATORY T CELLS AND REDUCES SERUM HUMAN INTERFERON-GAMMA IN A HUMANISED MOUSE MODEL OF GRAFT-VERSUS-HOST DISEASE

CUTHBERTSON Peter[1,2], ADHIKARY Sam[1,2], SLUYTER Ronald[3,2], WATSON Debbie[1,2]

[1]School of Chemistry and Molecular Bioscience, University of Wollongong, [2]Illawarra Health and Medical Research Institute, [3]School of Chemistry and Molecular Bioscience, Univeristy of Wollongong

Background: Graft-versus-host disease (GVHD) is a severe and often lethal complication arising from donor stem cell transplantation. In allogeneic and humanised mouse models of GVHD, extracellular ATP from damaged tissue activates the P2X7 receptor, leading to increased activation of donor T cells and worsened disease. **Aim:** This study aimed to investigate the effects of a modified P2X7 blockade regime in a humanised NOD-scid IL2Rγnull (NSG) mouse model of GVHD.
Methods: NSG mice were injected i.p. with $10x10^6$ human peripheral blood mononuclear cells (day 0), and daily i.p. injections of P2X7 antagonist Brilliant Blue G (BBG) (50mg/kg) or saline (day 0-10). Mice were monitored for clinical GVHD for 3 or 10 weeks. Human cell engraftment and serum human interferon-gamma (hIFNγ) were measured by flow cytometry and ELISA, respectively.
Results: BBG did not affect engraftment of $hCD45^+$ cells. However, the $hCD4^+CD25^+CD127^{lo}$ regulatory T cells (Treg) frequency was significantly increased in the spleens of BBG (4.9±3.2%, n=8) compared to saline (1.9±1.4%, n=7) mice at week 3 (P=0.03). Serum hIFNγ was significantly decreased in BBG (21.6±52.8pg/uL, n=8) compared to saline (110.0±54.6pg/uL, n=7) mice at week 3 (P=0.01). BBG also reduced clinical score (P=0.05, n=8), but did not significantly affect weight loss or overall survival over 10 weeks.
Conclusion: P2X7 blockade with a modified BBG regime preserved Tregs and reduced serum hIFNγ in humanised mice. Therefore P2X7 blockade using this new regime in combination with other therapies may be of benefit to prevent GVHD.

Abstract No. 51

A SURVEY OF THE CONTEMPORARY USE OF JJ STENTS IN RENAL TRANSPLANTATION IN AUSTRALIA AND NEW ZEALAND AND THE FEASIBILITY OF A CLINICAL TRIAL OF OPTIMAL TIMING OF STENT REMOVAL.

JAMBOTI Jagadish[1,2,3], IRISH Ashley[2,4], BHANDARI Mayank[5,6], HAWLEY Carmel[7,8,9]

[1]Fiona Stanley Hospital, [2]University of Western Australia, Perth, [3]Curtin University, Perth, [4]Renal & Transplantation Unit, Fiona Stanley Hospital, [5]Department of Surgery, Fiona Stanley Hospital, [6]Nephrology and Renal Transplant, [7]Renal & Transplantation Unit, Princess Alexandra Hospital, Brisbane, [8]University of Queensland, Brisbane, [9]AKTN,

A survey of the contemporary use of JJ stents in renal transplantation in Australia and New Zealand and the feasibility of a clinical trial of optimal timing of stent removal.

AIMS: Assess the current practice of JJ stent use, timing of removal and potential complications (Urinary Tract Infection, BK Virus infection) in renal transplant recipients (RTRs) and the feasibility of a clinical trial to test the optimal timing of stent removal.

METHODS: An online survey approved by ANZSN and TSANZ councils and facilitated via AKTN directed to the 20 recognised renal transplant centers in Australia and New Zealand in July 2018.

RESULTS: A total of 18/20 (90%) transplant centers responded, with an estimated annual 1232 RTRs.

1) 100% of the participating renal transplant centers used JJ stents intra-operatively and removed them by cystoscopy.

2) 67% (12/18) centers removed JJ stents after 4 weeks following transplant; 33% (6/18) removed at 2-4 weeks.

3) Estimated Prevalence of UTI requiring antibiotic treatment in the first 3 months was <10% in 39% (7/18) centers and between 10-20% in 61% centers (11/18).

4) 78% (14) of transplant centers screened for BK virus Post-transplant by serum and 17% (3) centers by both urine and serum. Reported BKV prevalence varied widely from 1-20%.

5) 83% of participating centers expressed willingness to be part of a study involving early removal of JJ stents.

CONCLUSIONS and FUTURE DIRECTIONS: JJ stents are routine in RTR in Australia and New Zealand and the majority are removed cystoscopically as outpatients after 4 weeks. There is a significant burden of early post-transplant UTIs. A randomised, controlled study comparing early inpatient removal of JJ stents with current practice is feasible and is widely supported.

Abstract No. 52

CLOSED INCISION NEGATIVE PRESSURE WOUND THERAPY IS SAFE AND FEASIBLE IN KIDNEY TRANSPLANT PATIENTS

HERLIHY David, TRAN Quoc, PUTTASWAMY Vikram, FISHER Charles

Department of Vascular Surgery, Royal North Shore Hospital, Sydney

Closed incision negative pressure wound therapy (ciNPWT) has been shown to reduce the rates of surgical site infection (SSI) and dehiscence in high risk vascular, cardiac and orthopaedic patients. As yet there has been no reported assessment of the use of ciNPWT in renal transplant patients.

Aim: We aim to explore the feasibility, techniques and difficulties of ciNPWT in kidney transplant patients prior to undertaking a multi-centre randomised-controlled trial.

Method: A retrospective review of 16 kidney transplant patients receiving ciNPWT assessing SSIs, wound dehiscence and complications, as well as exploring the challenges of maintaining ciNPWT for 7 days post-transplantation.

Results: ciNPWT dressings were used in 16 of 20 kidney transplants from August 2018 to March 2019. There was an average length of stay of 10 days, with an average ciNPWT duration of 7 days. There was 1 transplant nephrectomy and 1 unrelated mortality. There was 1 (6.25%) wound haematoma and there were no SSIs, wound dehiscences or lymphocoeles. ciNPWT was well tolerated by the patients, with no increase in pain or analgesia requirements. Initially, dressings were removed and replaced for duplex ultrasound imaging, however with correct dressing positioning and adjustments in technique, adequate imaging could be obtained without removing the dressing.

Conclusion: ciNPWT is feasible and safe for use in kidney transplant patients, however further research is required to clarify the full extent of its benefits in this cohort.

Abstract No. 53

CONTEMPORARY MANAGEMENT OF PROSTATE CANCER IN RENAL TRANSPLANT PATIENTS

PEREIRA Ryan[1,2], ROBERTSON Ian[1], BYRNE Sarah[1], GRIFFIN Anthony[1], LAWSON Malcolm[1,2], PRESTON John[1,2], WOOD Simon[1,2], RHEE Handoo[1,2]

[1]Renal Transplant Unit, Princess Alexandra Hospital, Brisbane, [2]Urology, Princess Alexandra Hospital, Brisbane

Aim: Due to the anatomical proximity of the prostate to pelvic positioned renal graft, management of Prostate Cancer (PCa) with surgery or radiation presents a challenge. Traditionally, definitive PCa management was considered high risk due to potential injury to the allograft, vasculature or ureter. The aim of this study was to investigate whether recent advancements in PCa management result in acceptable risks to the RTx graft. **Method:** A retrospective chart review between 2010-2018 of RTx patients referred to our unit for assessment and management of PCa was performed.

Results: The mean age was 68.43+/-7.98 years. The mean referring PSA was 98.48 ng/ml (1.1-1260) with a median value 11ng/ml. Reviewing the histology, the median International Society of Urological Pathology grading of PCa was 3 (range 1-5). Mean time to diagnosis from RTx was 6.35+/-4.65 years. Six patients had locally advanced or metastatic disease requiring systemic therapy at presentation. Of those with localized disease, external beam radiotherapy (EBRT) was used in 8 (29.62%) and radical surgery in 6 (22.22%). No significant change in creatinine was identified following EBRT or surgery. Androgen deprivation therapy was used in 10 patients (37.04%). To date, 13 of 27 (48.15%) patients are alive. Three patients (11.11%) died from progressive PCa.

Conclusion: This series from a large tertiary transplant unit did not identify any significant adverse effects of contemporary PCa treatment. As patients who receive RTx are advancing in age, it is reasonable to offer age and risk factor specific PCa screening and treatment.

Abstract No. 54

LAPAROSCOPIC TRANSPLANT NEPHRECTOMY FOR A FAILED INTRA-PERITONEAL TRANSPLANT KIDNEY

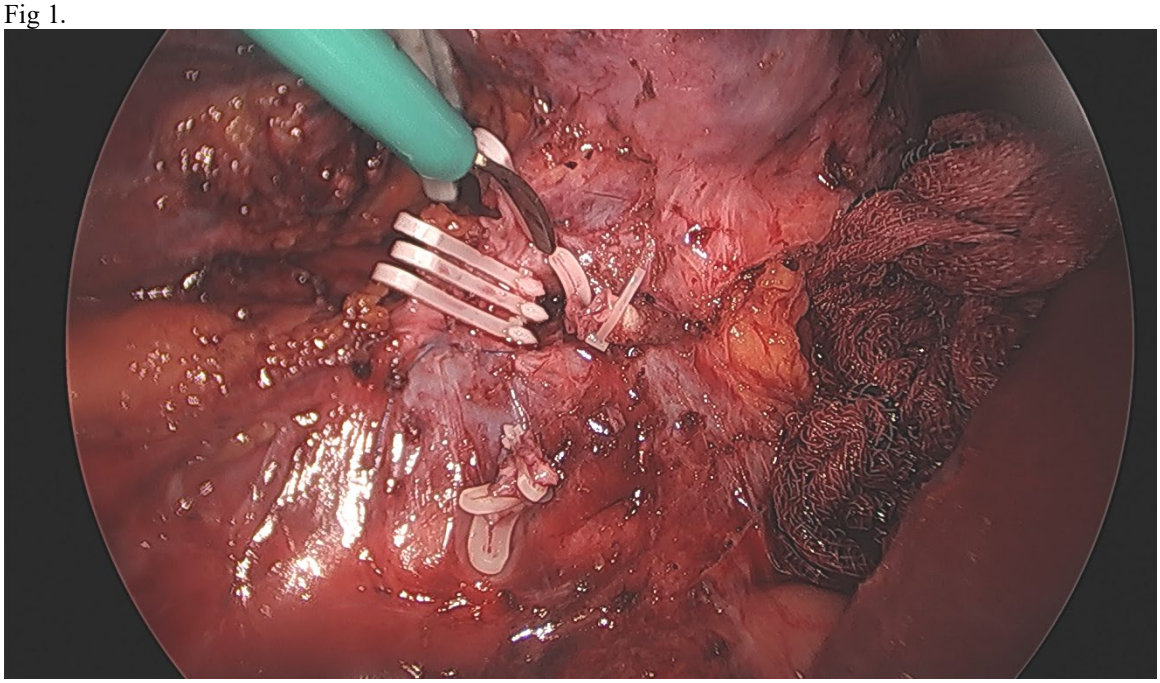
HEER MK[1], BULL Nick[2], TREVILLIAN PR[1]

[1]Renal Transplant Unit, John Hunter Hospital, Newcastle, [2]Department of Surgery, John Hunter Hospital, Newcastle

Transplant nephrectomy (TN) for failed renal transplant is performed as an open surgery because of adhesions and inflammation which makes dissection challenging. There is risk of significant morbidity due to bleeding, injury to viscera and infection. Minimally invasive surgery has improved outcomes in many other surgeries, but few authors have reported limited success with this approach to TN. We hereby present first case report of laparoscopic transplant nephrectomy from Australia. The recipient is 19 years old, who had received an orthotopic intraperitoneal right renal allograft transplant at the age of 12 years. She lost transplant function to chronic rejection after 6 years and was established on dialysis. An attempt to reduce her immunosuppression resulted in acute graft pain and graft intolerance syndrome. She was referred for TN. We used a laparoscopic approach with standard three ports for mobilization and fourth port for retraction. The graft had dense adhesions especially under the liver and renal hilum but also had peri-renal edema around mid and lower pole which facilitated the dissection. We were able to complete TN after obtaining control of renal allograft vessels with Hem-o-lock® clips (fig1). We also encountered her native renal vessels adherent to graft which needed further ligation. Operation took just below 180 minutes and around 50 ml of blood loss. Patient was discharged home on day 3.

Conclusion: Laparoscopic TN is feasible though challenging and can be safely performed in selected patients.

Fig 1.



Abstract No. 55

KIDNEY TRANSPLANTATION USING DONORS WITH SINGLE AND MULTIPLE RENAL ARTERIES – IS THERE A DIFFERENCE?

TRAN Quoc, HERLIHY David, MORITZ Peter, PUTTASWAMY Vikram
Department of Vascular Surgery, Royal North Shore Hospital, Sydney

Aims: Donor kidneys with multiple renal arteries (MRA) have previously been reported to be associated with increased complications and poorer outcomes in recipients. The objective of this study was to investigate the incidence of complications and the impact on the functionality of the transplanted kidney.
Methods: From 2017 to 2019, a total of 100 consecutive kidney transplantations that occurred at Royal North Shore Hospital, Sydney were retrospectively analysed. Patients were assigned to two groups: donors with single renal artery (SRA) and donors with multiple renal arteries (MRA, 26% of cohort). The impact of anatomical abnormalities on short-term outcomes of the transplantation were analysed with respect to warm ischaemic time, biochemical markers, resistive index, and complications requiring return to theatres.
Results: Mean warm ischemia time (in minutes) were similar with 54.2±25.3 vs 46.6±11.5 for transplants using MRA and SRA kidneys respectively. Change in urea at post-operative day 1 was -5.1±39.6% vs -9.6±55.9% and day 7 14.6±97.2% vs 18.7±110.4%. Change in estimated glomerular filtration rate (eGFR) at post-operative day 1 was 71.6±95.0% vs 95.7±130.2% and day 7 456.0±550.6% ± 386.9±443.7%. Change in creatinine clearance at post-operative day 1 was 25.6±28.3% vs 27.7±32.6% and 7 57.0±31.3% vs 49.4±41.1%. On table resistive index were 0.609±0.063 vs 0.607±0.074. Operative complications requiring take back to theatre showed 15.3% vs 9.5%.
Conclusion: Although kidney grafts with MRA have been considered a relative contraindication, this study suggests that kidney transplants using allografts with multiple versus single arteries have similar early outcomes.

Abstract No. 56

RISK FACTORS AND OUTCOMES OF VERY EARLY ACUTE REJECTION COMPLICATING KIDNEY TRANSPLANTATION IN AUSTRALIA AND NEW ZEALAND – A REGISTRY ANALYSIS

PUTRINO Samantha[1], FRANCIS Ross[1], JOHNSON David[1], CHO Yeoungjee[1], HAWLEY Carmel[1], CAMPBELL Scott[1], ISBEL Nikky[1], FAHIM Magid[1], PASCOE Elaine[2], MUDGE David[1]
[1]Nephrology and Renal Transplant, Princess Alexandra Hospital, Brisbane, [2]Princess Alexandra Hospital, Brisbane

Aims: We aimed to evaluate risk factors and outcomes of very early acute rejection (VEAR) in Australian and New Zealand kidney transplant recipients.
Methods: This registry-based, retrospective observational cohort study included all adult patients with ESKD undergoing transplantation in Australia and New Zealand between 2004 and 2014, using data from the ANZDATA and ANZOD registries. The primary outcome was VEAR, defined as rejection within the first week post-transplant. Secondary outcomes included patient and graft survivals.
Results: 8405 grafts were transplanted into 8260 recipients with a median follow-up of 4.2 years. VEAR occurred in 183 (6.0%) living and 353 (6.6%) deceased donor kidney grafts. Risk factors in living donor grafts were more class II HLA mismatches (adjusted odds ratio [aOR] for two mismatches 6.65, 95% CI 3.19-13.87), older donor age (aOR per year 1.02, 1.00-1.03), and higher peak PRA (aOR per percentage 1.01, 1.01-1.02). Deceased donor grafts had an increased risk with repeat transplantation (aOR 2.23, 1.42-3.49), more class I (aOR for three or four mismatches 2.58, 1.29-5.13) and class II (aOR for two mismatches 2.22, 1.58-3.13) HLA mismatches, donor hypertension (aOR 1.65, 1.25-2.17), ATSI recipient race (aOR 1.55, 1.01-2.39), donation after circulatory death (aOR 1.44, 1.06-1.96) and higher peak PRA (aOR per percentage 1.01, 1.00-1.01). VEAR was associated with inferior patient, overall graft, and death-censored graft survival rates.
Conclusions: VEAR complicates a small but significant proportion of kidney transplants and is associated with identifiable predictors and inferior outcomes. Further knowledge of this entity may help guide graft allocation, recipient counselling and management strategies.

Abstract No. 57

EVALUATION OF KIDNEY FUNCTION IN LIVING DONORS

GUO Henry, MCGINN Stella, LI Yan

Department of Nephrology & Transplantation, Royal North Shore Hospital, Sydney

Background: Assessment of kidney function is important during evaluation of potential living kidney donors (LKD). Compensatory hyper-filtration post donation results in 30% GFR loss and a small increase to future risk of ESRD. Current methods of assessing GFR have their own pitfalls.

Aim: To compare concordance between 24-hour Creatinine Clearance (CrCl) and estimated GFR (eGFRcr) using the CKD-EPI or MDRD equation against measured GFR (mGFR) using ^{99m}Tc-DTPA clearance.

Methods: Potential donors who underwent two or more tests and proceeded to donation between 2008-2018 were included. Reference mGFR was determined using ^{99m}Tc-DTPA clearance. Performance of 24hr CrCl and eGFRcr were assessed for accuracy, adjusted for age, gender, weight and ethnicity.

Results: 99 LKDs were included with a mean age of 51 yrs. Most were Caucasian (80%) and female (61%) with mean serum creatinine of 74.3±13.3 μmol/L. Across the entire cohort, bias was greatest using CrCl, with overestimation of GFR (20mL/min, $p<0.0001$), whilst eGFRcr (CKD-EPI) underestimated GFR (7.53mL, $p<0.001$) compared to ^{99m}Tc-DTPA. Multivariable regression showed significant overestimation with 24hr CrCl in men versus women ($p=0.05$) and with decreasing age ($p=0.03$). The KDIGO guidelines now recommend an ideal pre-donation GFR>90mL/min. 32% of our donors would not meet these new criteria using mGFR. 80% of our donors meet criteria when using CrCl in isolation versus 58% with eGFRcr (CKD-EPI) and 42% using eGFRcr (MDRD).

Conclusions: Creatinine based eGFR and 24-hour urinary CrCl are both accompanied by bias and should be interpreted in conjunction with measured GFR and clinical context when assessing potential LKDs.

Abstract No. 58

IMPACT OF DONOR HOSPITAL LOCATION ON LUNG ACCEPTANCE AND TRANSPLANT OUTCOMES

CAREW AM[1], YERKOVICH ST[2], HOPKINS PMA[2], CHAMBERS DC[2,3]

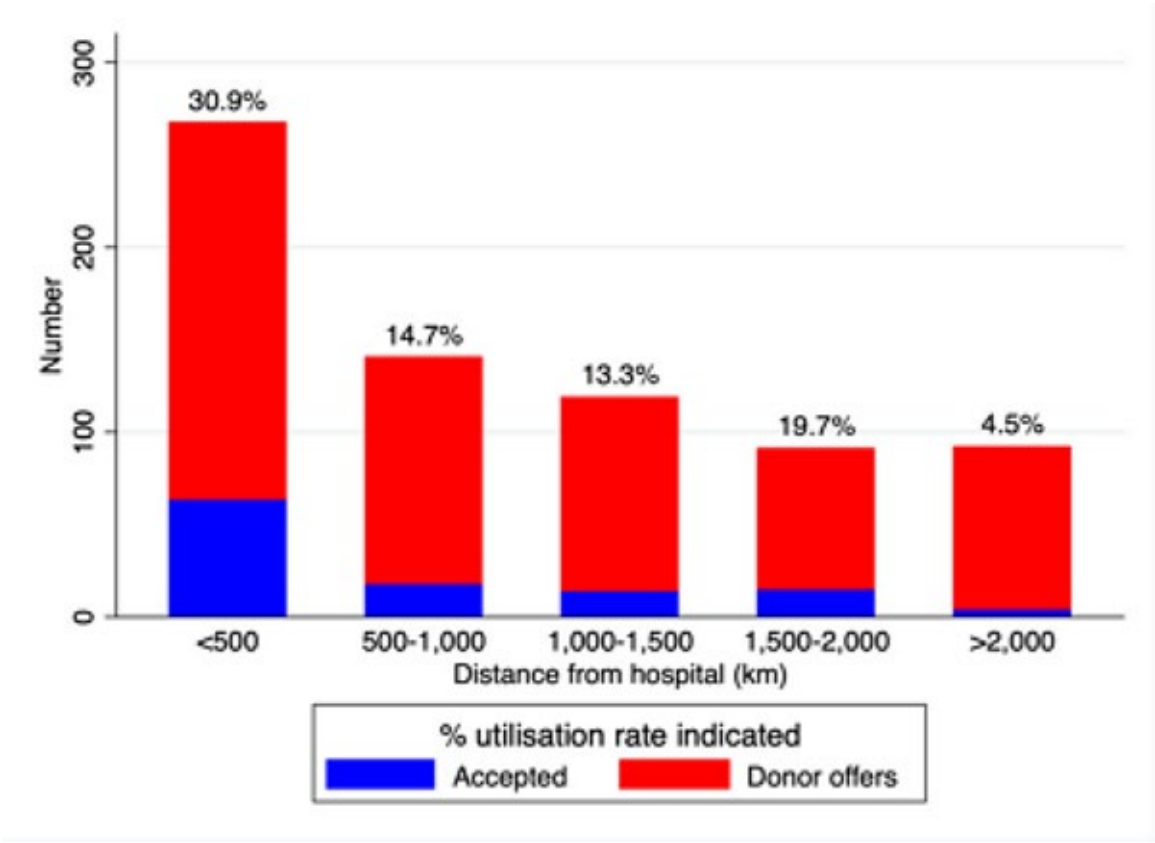
[1]Prince Charles Hospital, Brisbane, [2]Queensland Centre for Pulmonary Transplantation and Vascular Disease, Prince Charles Hospital, Brisbane, [3]School of Clinical Medicine, University of Queensland, Brisbane

Introduction: Donor lungs in Australia and New Zealand are initially offered to the lung transplant unit in their home state, and if declined are offered to non-home state units on a rotating basis. We hypothesized that increasing donor hospital distance would translate into a lower likelihood of organ acceptance. We examined whether increasing distance presents a higher risk of high-grade primary graft dysfunction (PGD), bronchiolitis obliterans syndrome (BOS), or death.

Methods: We retrospectively reviewed lung donor offers made to the Queensland Lung Transplant Service over a 3-year period (2015-2017), measuring the linear distance between the donor and recipient hospitals. We linked donor hospital distances to lung transplant recipients in the same period and examined rates of grade 3 PGD at 72 hours, BOS and death. We employed univariate and multivariate logistic regression and survival analyses to investigate the effect of distance on outcomes, together with basic donor or recipient demographic data.

Results: 593 lung donor offers were considered from distances of up to 3617.7km. Per additional 100km from the donor hospital, the odds ratio of lung acceptance was 0.93 (95% CI, 0.91-0.96). There was no appreciable impact of increasing distance on the outcome measures of grade 3 PGD at 72 hours (OR 1.05, 95% CI 0.99-1.11), development of BOS (HR 1.05, 95% CI 0.99-1.11), or death (HR 1.05, 95% CI 0.99-1.11).

Conclusion: Increasing lung donor hospital distance was associated with a lower likelihood of organ acceptance, but not with a higher risk of developing high grade PGD, BOS, or death.



Abstract No. 59

ALLOSEQ CFDNA ASSAY MEASURES DONOR-DERIVED CELL-FREE DNA IN SOLID ORGAN TRANSPLANT RECIPIENTS

BALTIC Svetlana[1], VIARD Thierry[1], AMOKRANE Kahina[2], DRABBELS Jos JM.[3], FAÉ Ingrid[4], FORTIER Catherine[2], MATHERS Simon[5], WENDA Sabine[4], LEE Deanna[2], O'CONNOR Timothy[1], ROSS David[1], WANG Sarah[2], GRSKOVIC Marica[1], ROELEN Dave[4], BURLINSO1N Natalia Diaz[6], TAUPIN Jean-Luc[1], FISCHER Gottfried F[5]

[1]CareDx, [2]Hôpital Saint Louis, Paris, France, [3]Leiden University Medical Center, Leiden, Netherlands, [4]Medical University Vienna, Vienna, Austria, [5]Manchester Royal Infirmary, Manchester, United Kingdom

Aim: Circulating donor-derived cell-free DNA (dd-cfDNA) is a biomarker of allograft injury that may enable more frequent, quantitative, and safe assessment of allograft rejection and injury status.

AlloSeq cfDNA is a new, non-invasive, targeted NGS assay that uses 202 single-nucleotide polymorphisms to accurately quantify dd-cfDNA in plasma of transplant recipients, without a need for separate donor and recipient genotyping. The assay combines a clinical laboratory compatible protocol and a fully automated analysis software. The aim of this multi-center study was to evaluate the assay performance.

Methods: AlloSeq cfDNA performance was assessed using independently validated 15 reference samples. Reference samples were developed by mixing genomic DNA from cell lines. DNA was fragmented by sonication to approximately 160-bp fragments to simulate the size profile of cell-free DNA. Three separate reference material panels were constructed using a donor cell line containing a single copy of EGFR T790M gene and three recipient cell lines. Trace amounts of the donor DNA were mixed with the recipient DNA at target levels from 0% to 25% to simulate different amounts of cfDNA originating from donor. The amount of donor DNA was verified using digital PCR and a SNP genotyping assay for EGFR T790M.

Results: AlloSeq cfDNA accurately and reproducibly quantified the fraction of dd-cfDNA in all 15 samples.

Conclusion: This noninvasive streamlined sequencing assay requires minimal hands-on time and can be completed within 24 hours post obtaining recipients cfDNA, on site, providing a critical practical turnaround time preferred in post transplantation surveillance.

Abstract No. 60

PATIENT AND GRAFT OUTCOMES FOLLOWING SIMULTANEOUS LIVER-KIDNEY TRANSPLANTATION: AN ANZDATA REGISTRY ANALYSIS

TANGIRALA NISHANTA[1], GRACEY David[1,2], WONG Germaine[3,2], FINK Michael[4], WYBURN Kate[1,2], CHADBAN Steven[1,2], MCCAUGHAN Geoff[5,2], ADAMS Leon[6], JEFFREY Gary[7], FAWCETT Jonathan[8], BYRNE Mandy[9], CATALAN Aimee[10], LIM Wai[11,12]

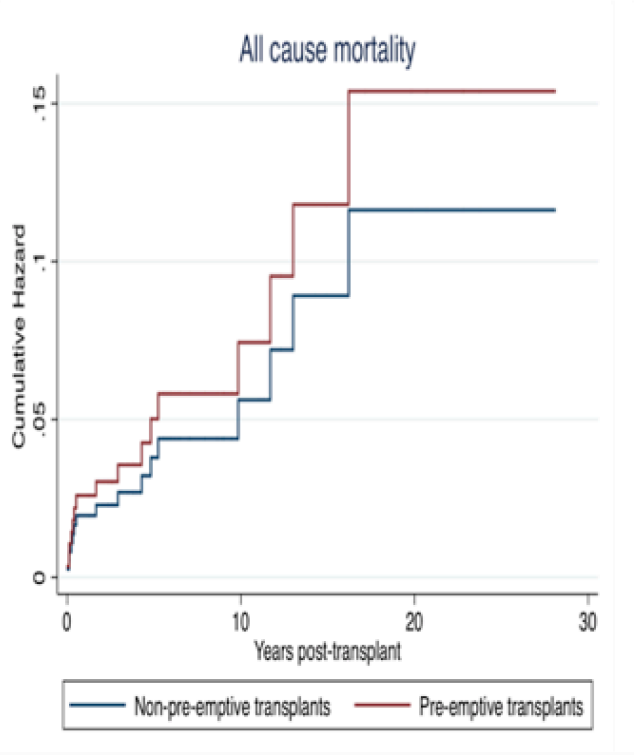
[1]Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney, [2]University of Sydney, [3]Department of Renal Medicine, Westmead Hospital, Sydney, [4]Hepatobiliary and Liver Transplant Surgery, University of Melbourne, [5]Department of Liver Transplantation, Royal Prince Alfred Hospital, Sydney, [6]Department of Liver Transplantation, University of Western Australia, Perth, [7]Hepatology and Liver Transplant Medicine Unit, University of Western Australia, Perth, [8]General, Visceral and Transplant Surgery, University of Queensland at the Princess Alexandra Hospital, [9]Liver Transplant Unit Victoria, Austin Hospital, Melbourne, [10]Mater Institute, Brisbane, [11]Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth, [12]University of Western Australia, Perth

Aims: To compare patient and kidney allograft outcomes of simultaneous liver-kidney (SLK) transplant recipients in Australia and New Zealand who received pre-emptive kidney transplants (SLK-PET) with those who were dialysis dependent (SLK-D) prior to transplantation.

Methods: We used multivariable-adjusted Cox regression shared frailty modeling to examine associations between pre-emptive status, all-cause mortality and kidney allograft failure, accounting for the potential intra-cluster correlation within transplanting centres.

Results: Of the 84 SLK transplants performed between 1989-2017, 20 (24%) received PET. The leading cause of ESKD was polycystic disease (27%), followed by glomerulonephritis (17%), oxalosis (10%) and amyloidosis (6%). The mean (SD) age of SLK-PET and SLK-D recipients were 48.7 (13.8) and 44.2 (16.2) years respectively (p=0.27). The median (IQR) waiting time for SLK-D recipients was 35.3 (13.6-73.1) months. For those who received PET, the median (IQR) eGFR at time of transplant was 21.7 (11.5-29.4) ml/min/1.73m^2. The 3-year patient and kidney allograft survivals for SLK-PET recipients were 79% (52%-91%) and 79% (52%-91%), respectively; compared with 91% (81%-96%; p=0.3) and 90% (79%-95%; p=0.4), respectively for SLK-D recipients. Compared to SLK-D recipients, the adjusted HRs for all-cause mortality and kidney allograft failure were 1.32 (0.40-4.35) and 1.06 (0.25-4.45), respectively for SLK-PET recipients. Figure 1 shows the adjusted cumulative failure curves for all-cause mortality and kidney allograft failure according to pre-emptive status.

Conclusion: Kidney allograft and patient survival following SLK transplants are acceptable. No patient or graft survival benefit from receiving SLK-PET compared to SLK-D was observed.



Abstract No. 61

RESPONSE PROCESS OF A PROPOSED CORE OUTCOME MEASURE FOR LIFE PARTICIPATION FOR TRIALS IN KIDNEY TRANSPLANT RECIPIENTS: A COGNITIVE PILOT STUDY

BAUMGART Amanda[1,2], TONG Allison[1,2], HOWELL Martin[1,2], CRAIG Jonathan C[1,2], JOSEPHSON Michelle[3], JU Angela[1,2]

[1]School of Public Health, University of Sydney, [2]Centre for Kidney Research, The Children's Hospital at Westmead, Sydney, [3]Department of Medicine, University of Chicago

Background: Life participation, the ability to engage in meaningful activities, has been identified by the Standardised Outcomes in Nephrology – Kidney Transplantations initiative as a critically important core outcome for all clinical trials. However, with no specific life-participation measure validated in this population, we examined the suitability of the PROMIS Short Form v2.0 – Ability to Participate in Social Roles and Activities 4a (Figure 1).

Methods: Semi-structured interviews were conducted with 20 kidney transplant recipients from seven countries. The four domains of comprehension, retrieval of relevant cognitive information, processes of judgement and response scale usability were explored using descriptive synthesis.

Results: Participants interpreted items consistently, although some found the first and fourth to be similar. Various information retrieval cognitive strategies were used, including recalling incidents (e.g., avoiding gatherings with ill relatives) and relying on a 'general idea'. The majority indicated a recall period greater than one month (three months most commonly recommended) was necessary to capture variation in activities which could be affected by fluctuating health or seasons. Most found formulating responses to be 'straightforward' and the response scale to be clear; however, some considered the middle response option difficult to differentiate. Six preferred a response scale assessing the severity of restricted life participation instead of frequency. **Conclusions:** The PROMIS measure for life participation appears to be comprehensible and meaningful. Nonetheless, modifications are warranted to ensure kidney transplant recipients can clearly distinguish between all items and response options and that the recall period is relevant to recipients yet appropriate for clinical trials.

Figure 1. Patient-Reported Outcomes Measurement Information System (PROMIS) Short Form v2.0 – Ability to Participate in Social Roles and Activities 4a

Please respond to each item by marking one box per row.

		Never	Rarely	Sometimes	Usually	Always
SRPPER11_ CaPS	I have trouble doing all of my regular leisure activities with others	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1
SRPPER18_ CaPS	I have trouble doing all of the family activities that I want to do.....	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1
SRPPER23_ CaPS	I have trouble doing all of my usual work (include work at home).....	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1
SRPPER46_ CaPS	I have trouble doing all of the activities with friends that I want to do.....	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1

Abstract No. 62

FACTORS INFLUENCING SOUTH WESTERN SYDNEY LOCAL HEALTH DISTRICT (SWSLHD) DECEASED DONOR RENAL TRANSPLANT WAITING LIST ACTIVATION

CHEUNG Jason, ZAHOROWSKA Beata, SHANMUGALINGAM Renuka, MUNRO Colleen E, WONG Jeffrey KW

Renal Unit, Liverpool Hospital

Introduction: SWSLHD is Australia's largest non-transplanting (dialysis patients) renal unit (ANZDATA 2017). Most transplant candidates are worked up locally per protocol for assessment at a SWSLHD clinic run by an external transplanting hospital team.

Aims: Review patients referred for transplant wait-listing in SWSLHD over a 5-year period to examine time from clinic review to wait-listing and factors influencing delays.

Methods: Patients whose first pre-transplant clinic visit occurred between January 2014 and December 2018 were identified. Their characteristics and wait-listing date were obtained from electronic medical records and the National Organ Matching Systems database. Dual organ transplants and living-donor transplant assessments were excluded.

Results: 146 patients were referred to the SWSLHD clinic, of which 96 were wait-listed, 19 deemed unsuitable and 31 pending wait-listing. Eight patients (4.6%) were activated at first clinic review, with overall mean activation time of 173 days. Patients activated early (<30 days) were younger than those activated after >30 days (age 46 versus 52, p=0.03) and were less likely to be diabetic (19% versus 40%, p=0.03). On multivariate analysis, BMI>30kg/m2 was the significant characteristic of patients not accepted for wait-listing. Patients whose wait-listing was pending were more likely to have BMI>25kg/m2 compared those that had been activated (p<0.01).

Conclusions: Despite protocoled transplant work-up, the mean activation time was almost 6 months. Our findings suggest targeted work-up is required in our diabetic and overweight-obese dialysis population to accelerate and increase waiting list activation.

Abstract No. 63

CLINICAL CHARACTERISTICS OF SIMULTANEOUS LIVER KIDNEY TRANSPLANT RECIPIENTS AND AN ANALYSIS OF THEIR LONG-TERM RENAL ALLOGRAFT AND PATIENT OUTCOMES COMPARED TO KIDNEY TRANSPLANT ALONE RECIPIENTS: AN ANZDATA REGISTRY ANALYSIS

TANGIRALA Nishanta[1], LIM Wai[2,3], WONG Germaine[4,5], FINK Michael[6], CHADBAN Steven[1,5], WYBURN Kate[1,5], MCCAUGHAN Geoffrey[7,5], ADAMS Leon[8], FAWCETT Jonathan[9], JEFFREY Gary[10], CATALAN Aimee[11], BYRNE Mandy[12], GRACEY David[1,5]

[1]Department of Renal Medicine, Royal Prince Alfred Hospital, Sydney, [2]Department of Renal Medicine, University of Western Australia, Perth, [3]Sir Charles Gairdner Hospital, Perth, [4]Department of Renal Medicine, Westmead Hospital, Sydney, [5]University of Sydney, [6]Department of Liver Transplantation, Austin Hospital, Melbourne, [7]Department of Gastroenterology, Royal Prince Alfred Hospital, Sydney, [8]Hepatology and Liver Transplant Medicine Unit, University of Western Australia, Perth, [9]General, Visceral and Transplant Surgery, University of Queensland at the Princess Alexandra Hospital, [10]Department of Liver Transplantation, University of Western Australia, Perth, [11]Mater Institute, Brisbane, [12]Liver Transplant Unit Victoria, Austin Hospital, Melbourne

Aims: To describe the characteristics and long-term outcomes of simultaneous liver-kidney (SLK) transplant recipients compared to kidney transplant alone (KTA) recipients.

Methods: Using the Australia and New Zealand Dialysis and Transplant Registry (1989-2017), characteristics of SLK recipients were examined. Unadjusted 5-year kidney allograft and patient survivals were calculated for SLK (n=84) and first deceased donor KTA recipients (n=12,231).

Results: Of the 84 SLK transplants, 53/84 were performed in the most recent transplant era (2010-2017); a six-fold increase since 1989. The leading causes of ESKD in SLK recipients were polycystic disease (27%), glomerulonephritis (17%), oxalosis (10%) and amyloidosis (6%). In the SLK group, 20 (24%) received pre-emptive kidney transplants. The two groups were similar in age (mean [SD] 45[16]years SLK vs 48[15]years KTA) and BMI (24[5]kg/m^2 SLK vs 26[6]kg/m^2 KTA) at transplantation. SLK recipients were less sensitised (peak PRA>50%, 4.8% vs 10%; p<0.001), more likely to have received kidneys from younger donors (mean[SD] donor age 35[16]years vs 42[17]years), more likely to have received HLA-mismatched kidneys (mean[SD] HLA-mismatches 4.4 vs 3.3; p>0.001) but have lower burden of vascular disease or diabetes compared to KTA recipients. The 5-year kidney allograft and patient survivals for SLK recipients were 83% (95%CI 72%-90%) and 84% (73%-91%), respectively. These compared with 78% (78-79%; log-rank p=0.02) and 88% (87-88%; log-rank p=0.57), respectively for KTA recipients.

Conclusion: Long-term kidney allograft and patient outcomes were comparable between SLK and KTA recipients, although the donor, recipient and transplant characteristics between the two cohorts were dissimilar.

Abstract No. 64

HUMAN LEUKOCYTE ANTIGEN MATCHING IN DECEASED DONOR ALLOCATION DOES NOT PREDICT ACUTE REJECTION EPISODES

GRAMLICK Madelyn E[1,2], YAUSIS Samuel[3], TREVILLIAN Paul R[3,2,4,5], HEER Munish K[3,6,4]

[1]Department of Surgery, John Hunter Hospital, Newcastle, [2]Faculty of Health and Medicine, The University of Newcastle, [3]Newcastle Transplant Unit, John Hunter Hospital, Newcastle, [4]Hunter Transplant Research Foundation, Newcastle, [5]Hunter Medical Research Institute, Hunter Medical Research Institute, Newcastle, [6]The University of Newcastle, New South Wales, Australia

AIMS: Human leukocyte antigen (HLA) matching has been a fundamental element of kidney allocation policies. With increasing knowledge of additional immunological risk factors the significance of HLA matching appears to be reducing. We aimed to assess the impact of HLA matching on graft rejection in patients undergoing deceased donor kidney transplant.

METHODS: The study population included all adult deceased donor kidney recipients at our unit from 2006 to 2018. Data was extracted from electronic health records. Patients were divided in two groups based on number of HLA mismatches (HLAMM) on HLA A, B and DR loci. Group 1 = HLAMM <=2 and Group 2 = HLAMM =>3. Data was analysed using SPSS.

RESULTS: 206 patients were included for analysis: 90/206 (43.7%) in Group 1 and 116/206 (56.3%) in Group 2. The demographic profile was comparable in two groups (table 1). Group 1 patients had no previous transplant, whilst 7/116 (6.03%) of Group 2 patients had received prior renal transplant (p=0.018). There was no significant difference in the number of patients suffering rejection: group 1 = 19/90 (21.1%), group 2 = 37/116 (31.9%) (p=0.084). However, patients in group 2 suffered rejection episodes sooner after transplant. There was also no significant difference in mean creatinine at 1 and 3 years.

CONCLUSIONS: A high proportion of patients received HLA-matched kidneys however this did not confer a significant benefit in terms of decreased episodes of rejection or mean creatinine at 1and 3 years compared to patients who received a kidney based on wait-time allocation.

TABLE 1:
Variables and outcomes of transplant recipients by HLAMM grouping, 2006 - 2018

	Group 1	Group 2	Significance (p)
Variable			
Recipient age (median [IQR], years)	57.50 [20]	53 [19]	0.161
Recipient gender (female, %)	42.2	35.3	0.314
Donor age (median [IQR], years)	50.46 [30]	50.76 [25]	0.763
Donor gender (female, %)	50	46.6	0.463
Heart beating donors (%)	78.16	67.89	0.110
Cold ischaemic time (mean, minutes)	842.21	854.87	0.797
Warm ischaemic time (median [IQR], minutes)	29(36)	32(37)	0.138
Previous transplant (%)	0	6.03	0.018
Outcome			
Patients with rejection (%)	19/90 (21.1)	37/116 (31.9)	0.084
Weeks to first rejection (median [IQR])	13 [17]	1 [7]	0.044
Weeks to second rejection (median [IQR])	24.50 [62]	9 [19]	0.085
Weeks to third rejection (median [IQR])	189 [215]	26 [19]	0.011
1 year creatinine (median [IQR], micromol/L)	138.07[61.25]	151[63.50]	0.172
3 year creatinine (median [IQR], micromol/L)	125.50[89.25]	147.25[66.50]	0.251

Abstract No. 65

VACCINATION SERORESPONSE AS A PREDICTOR FOR SUBSEQUENT KIDNEY TRANSPLANT REJECTION AND SERIOUS INFECTION

TA'EED A[1], POLKINGHORNE KR[1,2], MULLEY WR[1,2]

[1]Department of Nephrology, Monash Medical Centre, Melbourne, [2]Centre for Inflammatory Disease, Monash University, Melbourne

Aims: We hypothesized that seroconversion to vaccination in renal transplant recipients (RTRs) could provide a functional measure of immunosuppression adequacy with seroconversion predicting future rejection episodes and failure of seroconversion predicting subsequent infection.

Methods: We followed 151 RTRs who received the monovalent pandemic H1N1 vaccine in 2009 until December 2016. The incidence of rejection, serious infection (requiring hospitalisation), patient and graft survival was compared between seroconverters and non-seroconverters by Cox regression. Data were obtained from ANZDATA and chart review. Additionally, we assessed whether rejection episodes prior to vaccination were associated with the likelihood of seroconverting to vaccination by logistic regression.

Results: Twenty-six rejection episodes and fifty-eight serious infections occurred in the follow-up period. Seroconversion was not associated with an increased risk of subsequent rejection (HR 1.20, 95% CI 0.50-2.83, p=0.678) or serious infection (HR 0.94, 95% CI 0.50-1.75, p=0.835) on univariable or multivariable analysis. Seroconversion did not predict patient or graft survival and using a mixed model, there was no difference in eGFR at 5 years post-vaccination between seroconverters and non-seroconverters (p=0.256). Rejection prior to vaccination was associated with half the odds of seroconversion, however this association had reduced significance after adjustment for eGFR and mycophenolate dose (OR 0.51, 95%CI 0.23-1.14, p=0.10).

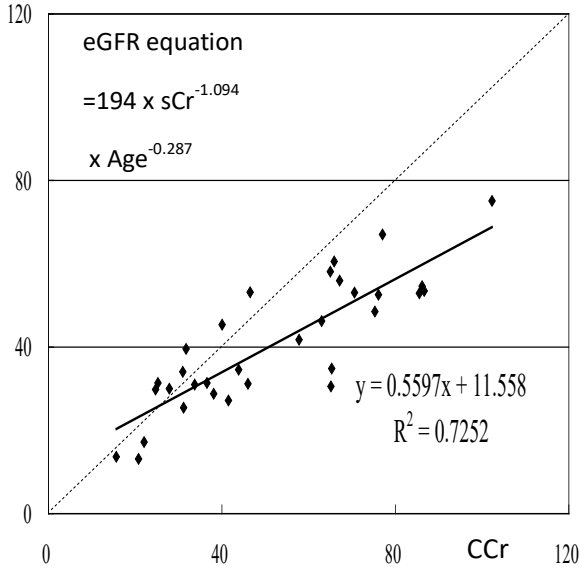
Conclusion: Vaccine response was not associated with the immunologic events of rejection or infection post-vaccination. It will be of interest to examine whether seroresponses to other vaccine types, such as polysaccharide vaccines, are more valuable in predicting immune events.

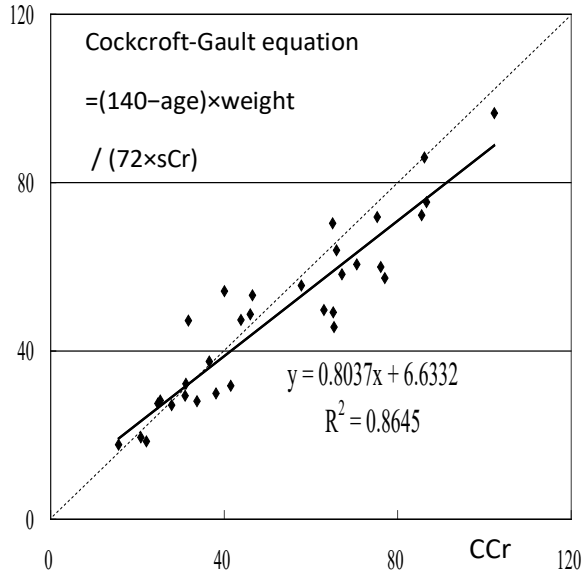
Abstract No. 66

EVALUATION OF THE EQUATIONS TO ESTIMATE THE RENAL FUNCTION OF KIDNEY TRANSPLANT RECIPIENTS FOCUSING ON CREATININE CLEARANCE

MARUI Yuhji, YOZA Naoto, SATO Yoshitsugu, MATSUMURA Kaori, USUBA Wataru, AOKI Naoto, NISHI Tomohiro, KATSUOKA Yuichi, NAKAZAWA Ryuto, SASAKI Hideo, CHIKARAISHI Tatsuya
Department of Urology, St marianna University School of Medicine

Background: Currently in Japan, the estimated glomerular filtration rate (eGFR) calculated by the 3-variable Japanese equation has been widely incorporated into clinical practice to recognize the renal function of CKD patients including kidney transplant recipients. In the meanwhile, creatinine clearance (CCr) is an important index of renal function as contributors to total renal drug clearance, and Cockcroft-Gault equation (CG) has been used to estimate CCr.
Aim: To examine the correlation of eGFR and CG to CCr of the kidney transplant recipients.
Methods: From 2016-2018, 27 kidney transplant recipients of our hospital were evaluated about age, weight, height, serum Cr (sCr), eGFR, CG and CCr. The regression lines were calculated to evaluate the correlation of eGFR and CG to CCr.
Results: With mean follow-up time of 107 (1-237) months 32 data sets were examined. The mean of eGFR, CG and CCr were 40.7±15.3ml/min/1.73m^2, 48.5±20.2ml/min and 52.0±23.3ml/min respectively. The regression lines were shown in figure. The correlation coefficients of eGFR and CG with cCCr were 0.73 and 0.86 respectively. The regression coefficient of CG was closer to 1 than eGFR.
Conclusions: For our kidney transplant recipients CG demonstrated greater concordance with CCr than eGFR, which tended to be underestimated against CCr. As CCr is a composite index of renal function including glomerular filtration and tublar secretion, CG appears to be more beneficial in kidney transplant recipients, especially with regard to the use of narrow-therapeutic-window drug.





Abstract No. 67

CANCER INCIDENCE IN DONOR REFERRALS - A NSW COHORT STUDY 2010-2015 USING DATA LINKAGE

HEDLEY James[1,2], DE LA MATA Nicole[1,2], ROSALES Brenda[1,2], O'LEARY Michael[3], CAVAZZONI Elena[3], KELLY Patrick[1,2], WYBURN Kate[4,5], WEBSTER Angela[1,2,6]

[1]Centre for Organ Donor Evidence, University of Sydney, [2]Sydney School of Public Health, Faculty of Health and Medicine, University of Sydney, [3]Other, NSW Organ and Tissue Donation Service, [4]Sydney Medical School, Faculty of Health and Medicine, University of Sydney, [5]Renal Unit, Royal Prince Alfred Hospital, Sydney, [6]Centre for Transplant and Renal Research, Westmead Hospital, Sydney

Introduction: Detailed medical history is not always available at donor referral, impeding suitability assessment. Donor referrals with possible cancer history may be rejected if details are uncertain. Timely cancer verification could reduce potential missed donor opportunities.

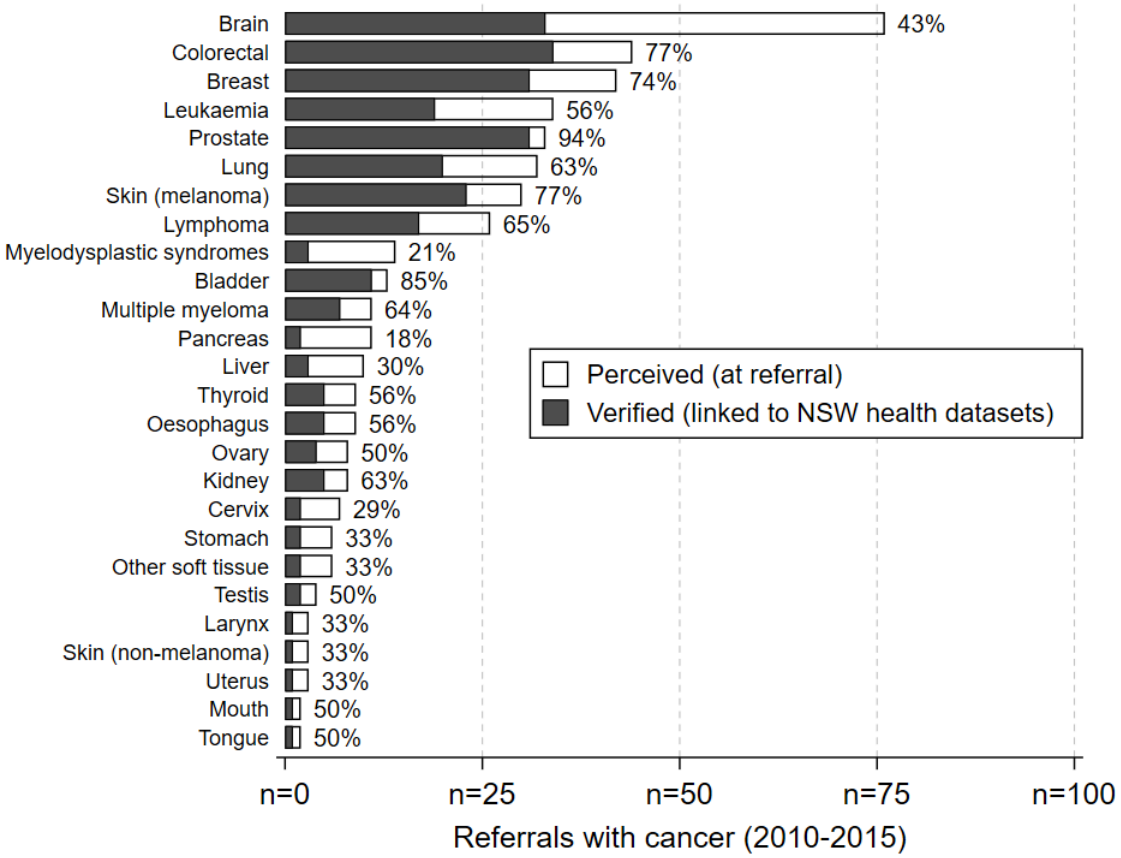
Aims: We sought to determine cancer incidence in donor referrals by primary site, and to compare the information available at time of referral with linked administrative health-data.

Methods: We used organ donor referrals 2010-2015 from the NSW Biovigilance Register. This Register linked donor referrals and transplant recipients to the NSW Admitted Patient Data Collection and Central Cancer Registry. Primary cancer site was grouped using ICD-10-AM codes.

Results: Of 2,957 referrals for donation, 433 (15%) had cancers reported at time of referral (458 cancers). Of these, 303 cancers (70%) were also reported in NSW health datasets. The most common cancers reported at time of referral were 76 (3%) brain, 44 (1%) colorectal, 42 (1%) breast, 34 (1%) leukaemia, 33 (1%) prostate, 32 (1%) lung, and 30 (1%) melanoma. Among these 291 most common cancers, 191 (66%) were verified. Agreement was highest in cancers of the prostate (94%), colorectal (77%), melanoma (77%) and breast (74%). Lowest agreement was found in cancers of the brain (43%) and leukaemia (56%) (Figure 1).

Conclusions: Cancers are quite commonly reported for donor referrals, but many cancers cannot be verified in the cancer registry, suggesting misclassification. Real-time health record access at referral could clarify uncertainty and potentially increase donation.

Figure 1: Perceived and verified cancers in NSW donor referrals 2010-2015



Abstract No. 68

GENETIC TESTING SHOWS HIGH FREQUENCY OF MENDELIAN DISORDERS IN PAEDIATRIC KIDNEY TRANPLANT RECIPIENTS

MCCARTHY Hugh[1], HOLMAN K[2], HO G[2], BENNETTS B[2], ALEXANDER SI[3]

[1]Centre for Kidney Research,, [2]Molecular Genetics, children's Hospital at Westmead, [3]Centre for Kidney Research, Children's Hospital at Westmead

Abstract: Testing and identification of underlying genetic aetiologies of end stage renal failure in children, has been improved with high throughput genetic testing.

Aim: To identify the genetic aetiology of end stage kidney failure in the paediatric kidney transplant population in an unselected, real-world cohort.

Methods: We reviewed the charts and genetic testing results of 103 children less than 18 years at the time of transplant who had received kidney transplants between 2009-19 at the Children's Hospital at Westmead.

Results: The clinical presentations included Congenital Abnormalities of Kidney and Urinary Tract (CAKUT) 28 patients; Nephrotic Syndrome (NS) 17 patients; polycystic kidney disease 7 patients; tubular disorders 3 patients; Nephronophthisis 21 patients; glomerulonephritis 11 patients; VACTERL 3 patients; ESRF no obvious cause 2 patients; econdary in 11 patients including a number with Wilm's tumours.

Genetic causes were identified in 41 of them, including *NPHS1, NPHS2, ADCK4, PLCE1* and *TRPC6* in children with NS, *NPHP1, NPHP3, NPHP4 CEP290, CEP164, INVS*, and *OFD1* in patients with nephronophthisis, *AGXT* and *CTNS* in patients with hyperoxaluria and cystinosis and CAKUT related mutations including *PAX2* and a number of copy number variations including Chr 6 duplication and Trisomy 21.

Conclusions: A high rate of genetic disorders is found in paediatric transplant recipients as the underlying cause of their kidney failure. These results may have important prognostic and clinical significance for them and their families.

Abstract No. 69

EHEALTH INTERVENTIONS FOR SOLID ORGAN TRANSPLANT RECIPIENTS: A SYSTEMATIC REVIEW AND META-ANALYSIS OF RANDOMISED CONTROLLED TRIALS

TANG James[1,2,3], JAMES Laura[1,2], HOWELL Martin[1,2], TONG Allison[1,2], WONG Germaine[1,2,3]

[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 Hospital, Sydney

Background: Self-management after organ transplantation consists of complex regime of tasks. Lack of support for self-management may contribute to adverse health outcomes. eHealth has the potential to support self-management, but evidence in solid organ transplantation remains unclear. This review aims to summarise the evidence for eHealth interventions after solid organ transplantation.

Methods: CENTRAL, MEDLINE and Embase databases were searched for randomised trials of eHealth interventions in solid organ transplant recipients. Risk ratios or standardised mean difference of outcomes of the intervention compared to standard care were calculated, and summary estimates determined using the random effects model. The quality of evidence was assessed by Cochrane risk of bias and Grading of Recommendations, Assessment, Development and Evaluations.

Results: Fourteen trials from 5 countries involving 1348 participants were included. eHealth interventions were grouped into functionality (Table 1). Outcomes were categorised into domains: clinical, adherence, healthcare utilisation, patient-reported outcomes, and knowledge/attitudes/behavioural change. Compared with standard care, eHealth improved adherence to medicine taking (RR 1.63, CI: 1.32 to 2.01) and self-monitoring responsibilities (RR 2.86, CI: 2.05 to 3.99), up to 12 months post-transplant. There were no differences in other outcomes including acute rejection and graft loss. Five studies reported harms including stress, anxiety and failure of intervention. Overall risk of bias was considered low or unclear, and the quality of evidence was low to very low for all outcomes.

Conclusion: eHealth interventions may improve adherence to medicine taking and self-monitoring in the short-term, but it is uncertain whether eHealth will improve long-term patient relevant outcomes.

Table 1: Definitions of eHealth intervention functionality	
Educational	Interventions aimed at improving knowledge and skills through learning. These may include websites, mobile 'apps' and computer programs.
Reminder/recall	Interventions used to aid memory. Examples may include computer reminders, mobile texting, telephone calls, Internet of things (devices with internet capability and the ability to communicate with other devices) such as wearables, sensors, and electronic pillboxes.
Self-monitoring/feedback	Interventions aimed at measuring a current behaviour and comparing this to a goal with resulting change in behaviours. These may include Telehealth, mobile 'apps', websites and Internet of things.
Behavioural counselling	Interventions aimed to enable patients to manage their condition through education and supportive tools to increase skills and confidence in areas including regular assessment, goal setting and problem solving. Examples may include Telehealth, mobile 'apps' and websites.
Clinical decision support systems	Interventions providing patients with targeted knowledge in a timely manner to improve health decision-making. Examples may include websites, mobile 'apps' and computer programs.
Multifunctional	Interventions encompassing a wide range of functionalities such as educational, reminder, self-monitoring, behavioural counselling and clinical decision support system. Examples may include single modality or combination of modalities such as websites, mobile 'apps' and computer programs.

Abstract No. 70

A CASE OF ABO INCOMPATIBLE RENAL TRANSPLANTATION FOLLOWING BLOOD GROUP SWITCHING IN THE RENAL TRANSPLANT PATIENT

LAM Susanna[1], HULTIN Sebastian[2], PRESTON John[1], CAMPBELL Scott[2]

[1]Urology and Renal Transplant, Princess Alexandra Hospital, Brisbane, [2]Division of Medicine, Princess Alexandra Hospital, Brisbane

Aim: We describe an unanticipated ABO incompatible renal transplant in a recipient with a previous allogeneic stem cell transplant, who was found to have reverted to his original blood group, after organ allocation.

Case: A 66 year old male with ESKD secondary to membranous nephropathy was listed to undergo deceased donor renal transplantation at our centre. His blood group changed from B to A following allogeneic stem cell transplantation for thrombocythaemia in 2007. It had been confirmed to be A several times. The recipient was unsensitised with a cPRA of 0%. He was offered a donation after circulatory death (DCD) 0/6 HLA mismatch organ with a donor blood group A. Blood tested at admission revealed reversion to his original blood group B. An urgent anti A titre was 1:2. We proceeded to transplantation following ABOi protocol with standard immunosuppression. Following delayed graft function attributed to a prolonged cold ischaemic time of 17 hours, his creatinine fell spontaneously on day 5. His post-operative anti-A titre was persistently 1:1.

Conclusion: Despite confirmed ABO conversion after stem cell transplantation, the recipient's original blood group B cells had regrafted with only low anti-A titres. We demonstrate a successful DCD ABOi transplant with standard immunosuppression in a recipient with possible bone marrow chimerism. Although a rare circumstance, it is important to consider regrafting of patient's own stem cells with altered blood group in this clinical situation.

Abstract No. 71

FREQUENCY AND OUTCOMES OF KIDNEY DONATION FROM INTENSIVE CARE PATIENTS WITH ACUTE RENAL FAILURE REQUIRING RENAL REPLACEMENT THERAPY

SANDERS Jo[1], OPDAM Helen[2], FURNISS Hayley[3], HUGHES Peter[4], KANELLIS John[5], JONES Daryl[2]

[1]Intensive Care, Holmesglen Private Hospital, [2]Intensive Care, Austin Hospital, Melbourne, [3]Intensive Care, Better Care VIctoria, [4]Renal Transplant Unit, Royal Melbourne Hospital, [5]Renal Transplant Unit, Monash Medical Centre, Melbourne

Kidney transplantation is the preferred treatment for end-stage renal failure. Unfortunately, donor organ shortages prevent many individuals receiving a renal transplant and there is a need to increase the pool of appropriate donors. The presence of acute kidney injury (AKI) in deceased donors has traditionally been a relative contraindication to renal transplantation, even though renal recovery may be favorable in the absence of chronic renal disease.

We undertook an eight-year retrospective observational study of potential deceased organ donors with AKI requiring renal replacement therapy (RRT). We evaluated the rate of successful transplantation, as well as short-term and longer term (median of 19.5 [13.0-52.7) months) outcomes following donation.

Amongst 1058 consented potential organ donors, 39 patients had AKI requiring RRT, of whom 19 became donors (13 not medically suitable, 7 didn't proceed to donation). The median (IQR) donor age was 41 (34-50) years and norepinephrine, epinephrine and vasopressin were given to 18, 14 and 9 donors, respectively. From the 38 donated kidneys 34 were transplanted. The median (IQR) age of recipients was 53 (42.8-58.5) years and they were dialysis free in a median (IQR) of 5.5 (2.3-10.8) days. Only minor abnormalities were found at 3 and 6 month renal biopsies, and 2 patients experienced graft failure in the first 12 months.

Amongst deceased donors with AKI receiving RRT and vasoactive medications outcomes of renal transplantation seems acceptable in the absence of pre-existing renal failure and other donor co-morbidity. Such patients may be an important additional source of kidneys for transplantation.

Abstract No. 72

SATISFACTORY PERFORMANCE OF THE ABBOTT ARCHITECT I2000 TACROLIMUS IMMUNOASSAY AGAINST THE LC-MS/MS TACROLIMUS ASSAY DEMONSTRATED IN KIDNEY TRANSPLANT PATIENTS FROM THE NORTHERN TERRITORY, AUSTRALIA

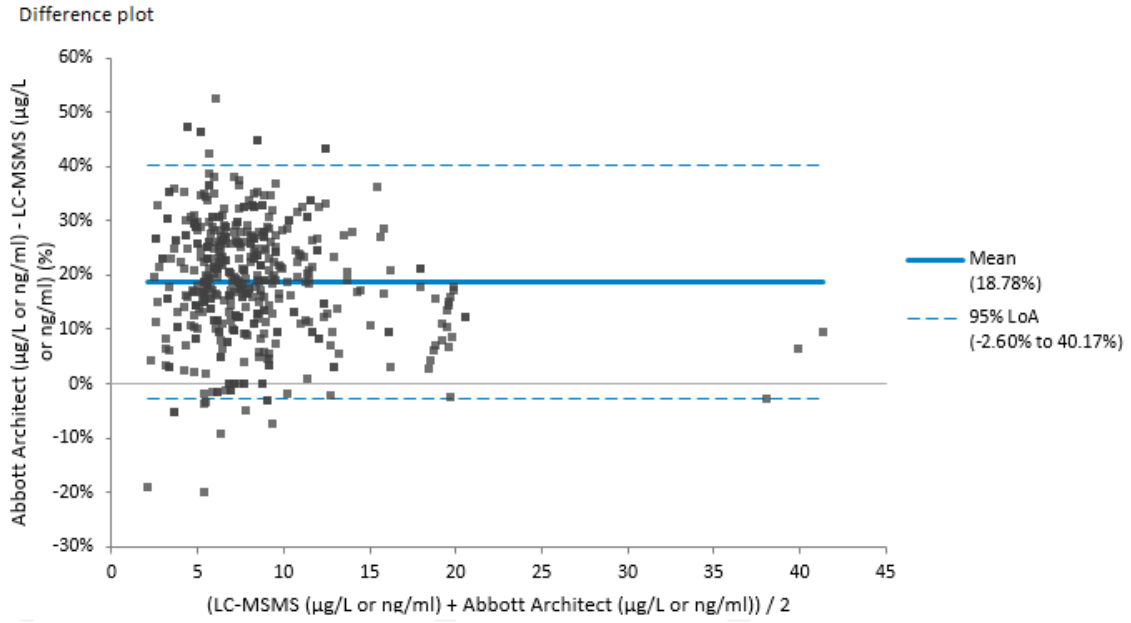
KAREPALLI Vijay K[1], RATHNAYAKE Geetha[2], MOGULLA Manohar[1], ASHFORD Jenna[2], MAJONI Sandawana William[1,3,4], SALLUSTIO Benedetta C[5]

[1]Department of Nephrology, Royal Darwin Hospital, [2]Department of Pathology, Royal Darwin Hospital, [3]Flinders University, Northern Territory Medical program, Royal Darwin Hospital, [4]Menzies Research Institute, Royal Darwin Hospital, [5]Department of Clinical Pharmacology, The Queen Elizabeth Hospital, Adelaide

Background: Tacrolimus is a post-transplant immunosuppressive drug with a narrow therapeutic window requires close monitoring of levels to avoid under-immunosuppression or toxicity. Top End Renal Services in the Northern Territory (NT) refer to South Australia (SA) for drug levels as there was no local service. We aimed to evaluate the Abbott ARCHITECTi2000 immunoassay against the liquid chemistry tandem mass spectroscopy (LC-MS/MS) used in SA for measuring tacrolimus levels to provide on-site service in NT. **Methods:** 465 specimens collected over 5 months and performed over several reagent lots. We used Passing-Bablok regression plots and Bland–Altman plots to assess the agreement between tacrolimus results on both platforms.

Results: The Passing-Bablok regression plot demonstrated a slope of 1.172 (CI 1.136 to 1.207) with an intercept of 0.262 (CI 0.040 to 0.472). In Deming analysis the slope was 1.095 (CI 1.074 to 1.116) with an intercept of 0.773 (CI 0.592 to 0.955), correlation coefficient (r) was 0.9782. Bland-Altman plot demonstrated positive bias for Abbott ARCHITECT results. The mean absolute bias was 1.494 ug/L and the mean percentage bias was 18.78%. Within run imprecision, Co-efficient of Variation (%) was 5.1, 2.7, 4.3, 3.4 and 3.5 at tacrolimus concentration levels of 4.2, 6.5, 9.5, 17.2 and 24.4 µg/L. TAT has improved by 80% from local assay.

Conclusion: The results demonstrate that Abbott ARCHITECT i2000 is an acceptable method to monitor levels of tacrolimus. The positive bias could be justifiable if the drug levels are initially based and then monitored on results from the same platform.



Graph: Bland-Altman bias plot for percentage difference for Abbott ARCHITECT i2000 tacrolimus results vs LC-MS/MS results. Mean= mean bias, 95% LOA=95% of Limit of agreement.

Abstract No. 73

TRAJECTORY OF DECLINE IN KIDNEY FUNCTION AND ASSOCIATION WITH ALL-CAUSE GRAFT LOSS IN AUSTRALIAN KIDNEY TRANSPLANT PATIENTS: JOINT LATENT CLASS MIXED MODELS

VON HUBEN Amy[1,2], TEIXEIRA-PINTO Armando[1,2], AU Eric[2,1], WONG Germaine[2,1]

[1]School of Public Health, University of Sydney, [2]Centre for Kidney Research, The Children's Hospital at Westmead, Sydney

Background: End-stage kidney disease (ESKD) is a serious and costly chronic disease in Australia. Kidney transplantation is not a cure for ESKD; with one-year/five-year all-cause graft survival rates remaining approximately 95%/85% over 2007 to 2016.

Aim: To identify classes of patients with estimated glomerular filtration rate (eGFR) trajectory progression associated with poorer survival times after kidney transplant for the purposes of early intensive risk factor management.

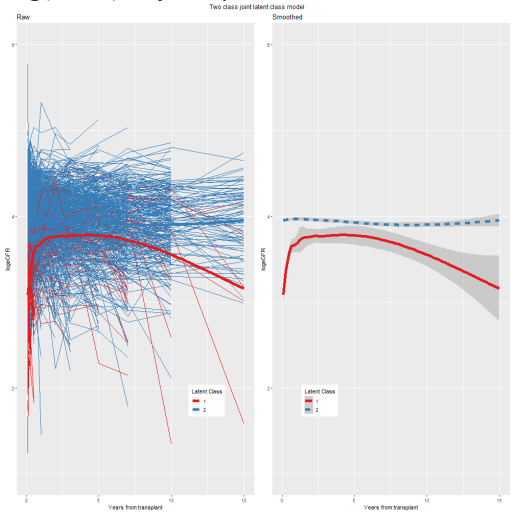
Method: We have applied a joint latent class mixed model (*jlcmm)* to kidney transplant patients from the national registry ANZDATA (1995 to 2014), to identify patterns of decline in renal function and investigate their association with clinical factors and all-cause graft loss.

Results: We have found evidence of at least two latent classes of patients with eGFR trajectories associated with differing all-cause graft survival curves (see Figure 1). Approximately 90% of patients have a stable eGFR trajectory after transplant, whilst the remaining patients have a curved trajectory associated with a more than doubling of the hazard of all-cause graft loss; hazard ratio 2.13 (95% CI: 1.93, 2.37). Of the patients with a curved trajectory, 91% had a deceased kidney donor.

Conclusion: The application of a joint latent class mixed model to Australian kidney transplant patients over 1995 to 2014 identifies two latent classes of patients: Class 1 has approximately 10% of patients, a curved eGFR trajectory, is highly associated with having a deceased kidney donor, and results in a more than doubling of the hazard of all-cause graft loss compared with Class 2.

Joint modelling: Two class joint latent class mixed model (jlcmm)

Log(eGFR) trajectory raw and smoothed



Kaplan Meier survival curves by latent class

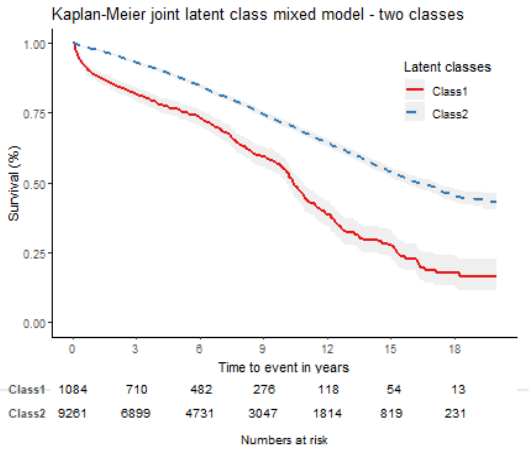


Figure 3 Results of applying the joint latent class mixed model to Australian kidney transplant patients, 1995 to 2014. Observed log(eGFR) trajectories by class, raw and smoothed. Kaplan-Meier survival curves for all-cause graft loss by latent class.

Abstract No. 74

DONOR REFERRALS WITH PRIMARY BRAIN TUMOUR – PERCEIVED VS. VERIFIED RISK

THOMSON Imogen[1,2], HEDLEY James[1,3], DE LA MATA Nicole[1,3], ROSALES Brenda[1,3], O'LEARY Michael[4], CAVAZZONI Elena[4], KELLY Patrick[1,3], WYBURN Kate[2,5], WEBSTER Angela[1,3,6]

[1]Centre for Organ Donor Evidence, University of Sydney, [2]Sydney Medical School, Faculty of Health and Medicine, University of Sydney, [3]Sydney School of Public Health, Faculty of Health and Medicine, University of Sydney, [4]Other, NSW Organ and Tissue Donation Service, [5]Renal Unit, Royal Prince Alfred Hospital, Sydney, [6]Centre for Transplant and Renal Research, Westmead Hospital, Sydney

Introduction: In Australia, organ donation is not contraindicated for referred donors with most types of primary brain tumour (PBT). However, the perceived risk of transmission is based only on information available at referral, which may be incomplete or inaccurate.

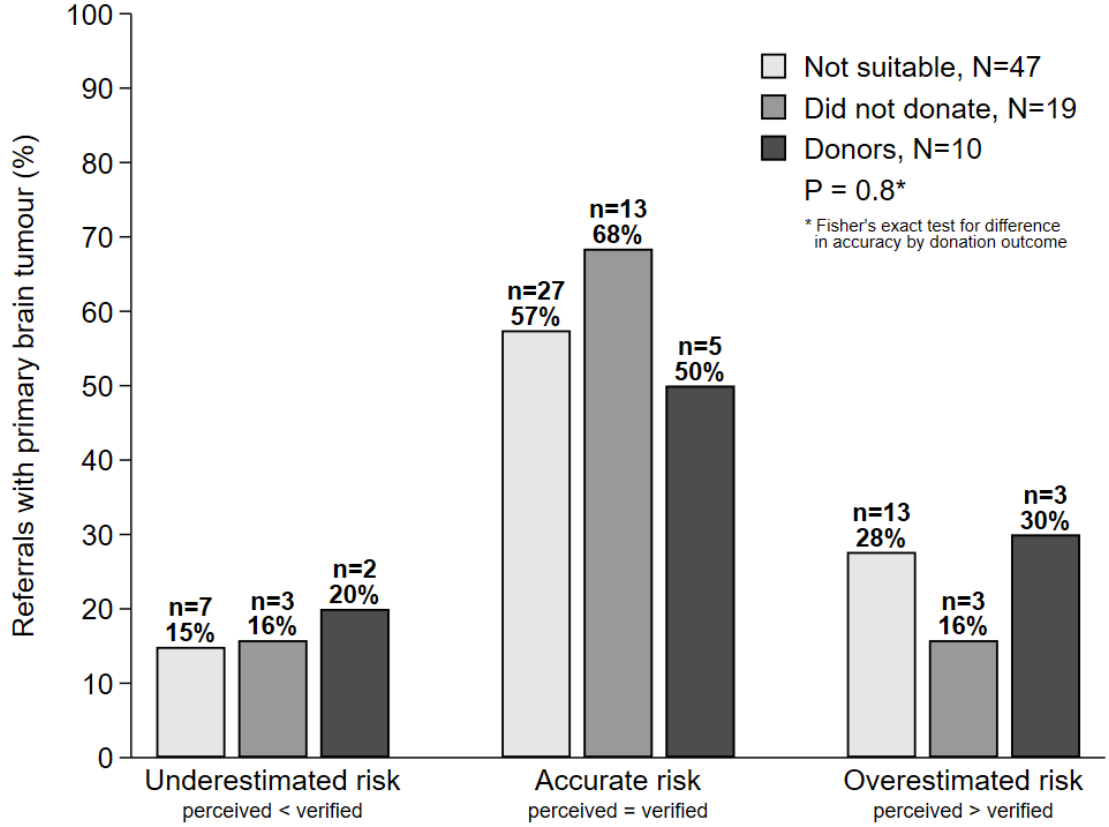
Aims: We sought to determine the accuracy of perceived risk of PBT transmission at the time of referral compared to verified risk using linked-data.

Methods: We used organ donor referrals 2010-2015 from the NSW Biovigilance Register. This Register linked NSW organ donor referrals and transplant recipients to NSW Admitted Patient Data Collection and the Central Cancer Registry. We classified risk based on tumour type using the Transplant Society of Australia and New Zealand (TSANZ) guidelines. Perceived risk was determined from referral logs, with verified risk based on NSW Biovigilance register.

Results: We analysed 2,957 referrals for donation and identified 76 (3%) with PBT; 11 (14%) from referral logs, 5 (6%) from other health records, and 62 (79%) from both. The perceived risk agreed with verified risk in 45 referrals (59%), was overestimated in 19 (25%), and underestimated in 12 (16%). There was no difference in agreement based on donation outcome (p=0.8). Cohen's Kappa was 0.39 suggesting fair agreement. All 10 (14%) actual donors were verified minimal risk with disagreement in 1 (9%) perceived low risk.

Conclusions: Assessment of PBT transmission risk cannot always be determined accurately at referral. Perceived risk is more frequently overestimated than verified risk, suggesting risk averse decision-making. Evidence based decision support may have a future role.

Figure 1: Perceived vs. verified risk for referrals with primary brain tumour by donation outcome



Abstract No. 75

ANALYSING THE EFFECTS OF CLINICAL PREDICTIVE VARIABLES ON KIDNEY TRANSPLANT OUTCOMES IN RANDOM FOREST MODELS

PAIZIS Kathy[1], SLOGGETT Clare[2], SYPEK Matthew[3,4,5], IERINO Francesco[6,7]

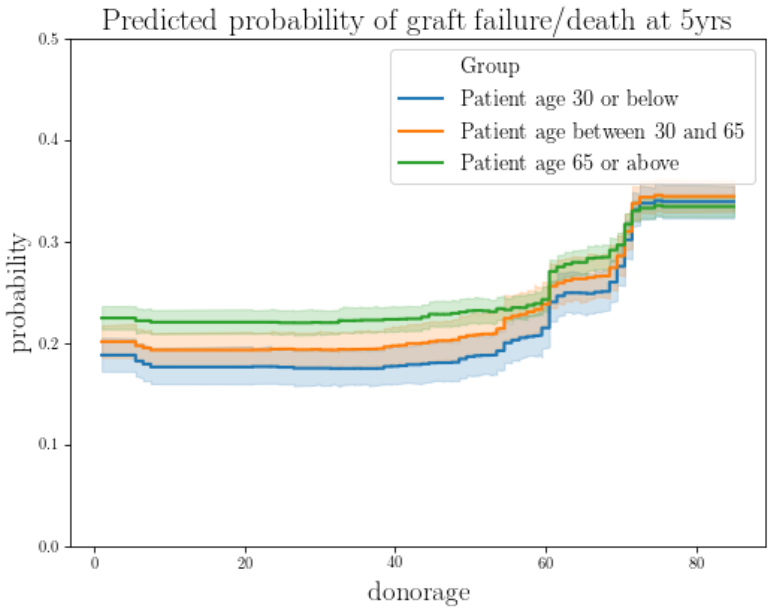
[1]Renal & Transplantation Unit, Austin Hospital, Melbourne, [2]Melbourne Bioinformatics, University of Melbourne, [3]ANZDATA, [4]Department of Nephrology, Melbourne Health, [5]Faculty of Medicine, Dentistry and Health Sciences, University of Melbourne, [6]Department of Nephrology, St Vincent's Hospital, Melbourne, [7]University of Melbourne

Aims: Machine learning approaches have become of interest in studying clinical data. Random forests are a popular approach due to their flexibility and wide applicability. Compared to regression models, ML approaches have the capacity to capture more complex associations and detect variable interactions without previous specification of interaction terms. The disadvantage can be that the resulting model is more difficult to interpret. We present a method for investigating the effects of clinical variables on transplant outcomes in a Random Forest model.

Methods: A Random Forest model was applied to ANZDATA/ANZOD renal transplant data 1994-present, incorporating a wide range of potentially predictive pre-transplant variables. By tracing predicted probabilities, we are able to examine the role of these variables in detail, and to compare the effects in different graft cohorts under the same model.

Results: We plot the effects of variable manipulation within the model to explore interactions and assist in hypothesis generation (see Figure for example). We are also able to examine non-trivial behaviour for some predictive variables, such as the non-linear risk associated with patient age.

Conclusions: The method has to date suggested interesting hypotheses including: (a) older kidneys may have a noticeably lower detrimental effect in older patient cohorts; (b) Noradrenaline may have a protective effect on lower-quality or older kidneys in particular; (c) patient age may begin to affect outcomes more severely at a critical "threshold" age. Further investigation of predictive variables as well as validation of effects discovered so far would be valuable.



Abstract No. 76

HBA1C AS A PREDICTOR OF POSTOPERATIVE KIDNEY COMPENSATORY HYPERTROPHY IN MALE LIVING DONORS

KOSUKE Tanaka[1], KOHEI Kinoshita[1], YUJI Hidaka[1], MARIKO Toyoda[2], AKITO Inadome[3], ASAMI Takeda[4], JUN Shoji[5], SHIGEYOSHI Yamanaga[1]

[1]Department of Surgery, Japanese Red Cross Kumamoto Hospital, [2]Department of Medicine, Japanese Red Cross Kumamoto Hospital, [3]Urology, Japanese Red Cross Kumamoto Hospital, [4]Department of Nephrology, Japanese Red Cross Nagoya Daini Hospital, [5]Department of Nephrology, UCSF Connie Frank Transplant Center

Aims: Renal function of the remaining kidney in living donors recovers up to 60~70% of pre-donation estimated-glomerular filtration rate (eGFR). Degree of compensatory renal function recovery is related with chronic interstitial fibrosis and tubular atrophy (ci+ct) score and arteriolar hyalinosis (ah) score. We examined living donor characteristics to identify potential clinical predictors of chronic renal lesions and postoperative hypertrophy.

Methods: This was a single-center, retrospective analysis of 155 living donors. We reviewed 1-hour post-perfusion renal biopsies and divided the 155 donors into two groups; chronic change (CC) group (n=21) with ci+ct > 1 and ah > 1 as well as the rest of donors as a control group (n=134). We examined age, sex, tobacco use, blood pressure, HbA1c, aortic calcification index, BMI as possible predictive factors. The recovery rate of eGFR was measured at one year after surgery.

Results: Out of the 155 donors, 53 (34.2%) were male. There was a significant difference in HbA1c between the CC group and the control group in male donors (6.02±0.18 vs 5.7±0.54, p=0.032). Multivariate analysis also showed that HbA1c was significantly different for male donors (odds ratio 1.25 per 0.1%, p=0.039). Cut-off value of HbA1c was 6.05% (AUC 0.737, p=0.034). There was a higher recovery rate of eGFR among males whose HbA1c was lower than 6.05% (60.7±6.3% vs 53.6±7.4%, p=0.013)

Conclusions: Although HbA1c value may meet criteria for living donation, this may serve as a possible marker for the insufficient recovery of postoperative renal function.

Abstract No. 77

COMBATING LOSS TO FOLLOW UP OF LIVING KIDNEY DONORS

GUO Henry, MCGINN Stella, LI Yan

Department of Nephrology & Transplantation, Royal North Shore Hospital, Sydney

Background: Whilst great efforts and resources are placed on identifying suitable living donors to minimize their future risk, there remains incomplete post donation follow up with little long-term data of living kidney donors in Australia.

Aim: To assess factors associated with loss to follow up post kidney donation at our transplant centre.

Methods: We conducted a single centre retrospective cohort study of living kidney donors between 2008 and 2018 at Royal North Shore Hospital, Sydney. Date of transplantation, date of last follow up, demographics, renal function and prevalence of cardiovascular risk factors were collected from medical records.

Results: Out of 98 donors, 60% were female, with 26% travelling from interstate or overseas to donate. The mean duration of follow up was 4.1 ± 2.9 years. Follow up rates were 76.3% and 42.7% at 1 and 5 years respectively. Tendency to follow up declined with younger age and increasing distance from our transplant centre. Follow up rates at 5 years did not differ between related, unrelated or altruistic donors. Of the donors still being followed up at 5 years, 28% had eGFR<60mL/min, 20% had hypertension and 67% had treated hypercholesterolemia.

Conclusions: Despite the risks associated with kidney donation, long term follow up remains poor. Future interventions should target improving long term follow up care for all living kidney donors.

Abstract No. 78

ENDOTHELIAL GLYCOCALYX DYSFUNCTION IN ORGAN DONORS IS ASSOCIATED WITH DELAYED GRAFT FUNCTION IN RENAL TRANSPLANT RECIPIENTS BUT NOT EARLY ALLOGRAFT DYSFUNCTION IN LIVER RECIPIENTS

SLADDEN TM[1,2], YERKOVICH S[1], JAFFREY L[3], REILING J[4], FAWCETT J[4], ISBEL N[3], CHAMBERS D[1,2]

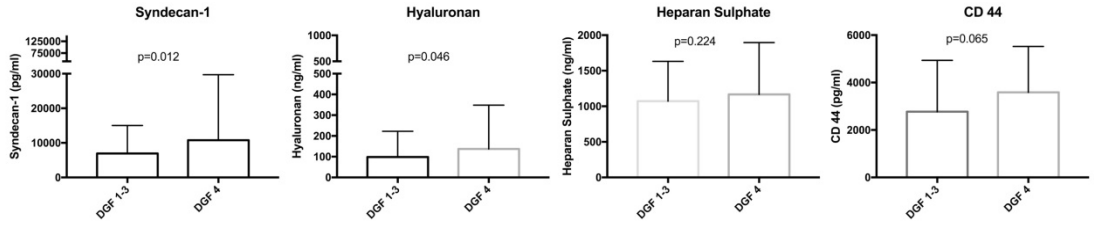
[1]Queensland Centre for Pulmonary Transplantation and Vascular Disease, Prince Charles Hospital, Brisbane, [2]School of Medicine, University of Queensland, Brisbane, [3]Department of Nephrology, Princess Alexandra Hospital, Brisbane, [4]Lung Transplant Service, Princess Alexandra Hospital, Brisbane

Aims: Early post-transplant organ dysfunction is associated with deleterious long term outcomes, with the incidence increasing with the use of extended criteria organs. The endothelial glycocalyx, a fragile luminal structure, has been demonstrated to be essential in vascular function and shed post-transplant. We hypothesised injury to the glycocalyx in organ donors, as determined by increased peripheral blood levels of glycocalyx breakdown products, would predispose grafts to the developed of delayed graft function (DGF) in renal recipients and early allograft dysfunction (EAD) in liver recipients.

Methods: Kidney and/or liver organ donors, where consent for research was obtained, between 2009 -2015 and who had a blood sample available were included. Recipients all received their transplant at the Princess Alexandria Hospital. Endothelial glycocalyx breakdown products: hyaluronan, syndecan-1, heparan sulphate and CD44 were measured in the peripheral blood of organ donors.

Results: From 192 organ donors, recipient follow-up was available for 111 liver recipients with 15 recipients (13.5%) developing EAD and 321 renal recipients, 86 (26.8%) developing stage 4 DGF. Elevated hyaluronan (OR:1.16 (0.99-1.35); p=0.05), CD44 (OR:1.21 (0.98-1.49); p=0.07) and syndecan-1(OR:1.02 (1.00-1.04); p=0.03) were all associated with the development of Stage 4 DGF in renal recipients by univariate analysis. In multivariate analysis, hyaluronan was independently associated with Stage 4 DGF (per 100ng/ml OR:1.23; p=0.04). No there was no association between glycocalyx breakdown products with liver EAD.

Conclusions: Endothelial glycocalyx shedding in renal organ donors was associated with the development of severe DGF highlighting endothelial glycocalyx dysfunction as a novel injury pathway.



Abstract No. 79

DIAGNOSTIC TESTS FOR DELAYED GRAFT FUNCTION: A SYSTEMATIC REVIEW

LAI Sum Wing Christina[1,2], YEE, Seow Yeing[1], YING, Tracey[1], CHADBAN Steve[1,3]

[1]University of Sydney, [2]Renal & Transplantation Unit, Royal Prince Alfred Hospital, Sydney, [3]Royal Prince Alfred Hospital, Sydney

Background: To date, there is no consensus in definition of delayed graft function (DGF). The most commonly used definition is the requirement of dialysis within 7 days post-transplant. This definition is imperfect as dialysis indication is subjective. It lacks sensitivity and specificity to correctly identify patients with DGF and to predict clinical outcome. We therefore sought to evaluate other potential diagnostic tests for DGF by meta-analysis.
Methods: A systematic literature search from inception to December 2018 was performed via Ovid MEDLINE and EMBASE. All studies evaluating diagnostic tests with the primary endpoint of DGF, defined as requirement of dialysis within 7 days post-transplant, were included.
Results: Of 3,185 citations, we identified 23 studies describing novel diagnostic tests for DGF. A total of 15 potential tests were identified which include blood tests (neutrophil gelatinase-associated lipocalin, cystatin C, thromboxane, IL-1, IL-18, aminoacylase-1, C-terminal argin fragment, liver-type fatty acid-binding protein, leptin and adiponectin, malondialdehyde), urine tests (clusterin, tissue inhibitor of metalloproteinases-2, insulin-like growth factor-binding protein 7, targeted urine proteome assay), renal biopsy and ultrasound. There was a high variability in diagnostic threshold, test methods and study quality with majority of the trials performed in a single centre setting.
C**onclusions:** The results of our systematic review have not identified a superior test. A large study comparing the strength of association between the current gold standard and alternative definitions with short- and long-term outcomes is needed.

Abstract No. 80

IDENTIFICATION OF BARRIERS FOR INDIGNEOUS AUSTRALIANS GAINING ACCESS TO THE KIDNEY TRNASPLANT WAITING LIST: A VICTORIAN PILOT STUDY

ATKINSON Amy[1], FORD Sharon[2], GOCK Hilton[1], IERINO Frank[1], GOODMAN David[1]

[1]Department of Nephrology, St Vincent's Hospital, Melbourne, [2]Department of Nephrology, St Vincent's Institute, Melbourne

Aims To identify the barriers to Indigenous Australians gaining access to the kidney transplant waiting list.
Methods All Indigenous Australians with end stage renal failure linked to a single Victorian hospital over a 10-year period were included in the study. All living participants were interviewed, and hospital medical and tissue typing records were examined to plot the patients transplant journey. Reasons why patients were not listed for transplantation and delays in transplant work-up pathway were recorded.
Results: Of the 39 patients in the study, 7 have received a kidney transplant, 11 remain on dialysis and 21 are deceased. Currently there are no patients on waiting list. Most of the patients had diabetes mellites (>70%). Those receiving a transplant were younger, had less ischaemic heart disease and had ceased smoking. Seven of 9 patients listed for transplantation received a transplant with good outcomes. Psychosocial factors such as fear, lack of interest, depression and adherence to medications and dialysis attendance accounted for up to one-third of patients not being listed for transplant. Only 1 of 29 patients achieved the state KPI (list for transplant within 3 months of starting dialysis, target 30%). It took between 2-3 years before 9 of 29 patients (31%) were listed for transplant.
Conclusion: Medical co-morbidities and psychosocial factors are the main barriers to Indigenous Australians gaining access to the transplant list. This early data supports a focused attention to early culturally appropriate education and management of co-morbidities before commencement of dialysis to provide Indigenous Australians greater access to transplantation.

Abstract No. 81

HEALTHCARE PROFESSIONAL AND COMMUNITY PREFERENCES IN DECEASED DONOR KIDNEY ALLOCATION: DO THE PRIORITIES ALIGN?

SYPEK MP[1,2,3,4], HOWELL M[5,6], DUNCANSON E[1], HUGHES P[2,3], WONG G[5,6,7], CLAYTON P[1,8,9], HOWARD K[5], MCDONALD S[1,8,9]

[1]ANZDATA, [2]Nephrology and Renal Transplant, Melbourne Health, [3]Faculty of Medicine, Denistry and Health Sciences, University of Melbourne, [4]Nephrology and Renal Transplant, Royal Children's Hospital, Melbourne, [5]School of Public Health, University of Sydney, [6]Centre for Kidney Research, Children's Hospital at Westmead, [7]Centre for Transplant and Renal Research, Westmead Hospital, Sydney, [8]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [9]Department of Medicine, University of Adelaide

Background: The allocation of kidneys from deceased donors requires a balance between optimising the utility derived from this finite resource and ensuring equitable access. We sought to identify the preferences of health care professionals on the principles that should guide organ allocation and compare these to the priorities of the general community.

Methods: A best worse scaling choice experiment was conducted amongst healthcare professionals working in organ donation and kidney transplantation in Australia. Twenty-nine allocation principles were presented in a balanced incomplete block design with respondents required to select the most and least important principles from 6 options. Sequential best worst multinomial logistic regression modelling was used to determine relative preferences and compared to outcomes from a similar survey previously conducted amongst the general community.

Results: 206 healthcare professionals completed the survey. There was a strong preference for principles that maximise utility, either through allocating kidneys with the best predicted survival to the healthiest/youngest recipients or as an explicitly stated goal. Priority was also given to principles addressing equality in access for disadvantaged groups. In contrast, members of the general community were more likely to value principles prioritizing patients with the greatest need. Both groups did not prioritize principles that rewarded individuals for past actions or choices, or favoured older patients or local organ allocation.

Conclusion: Healthcare professionals favour principles that maximise utility in kidney allocation. This may be in conflict with priorities of the general community and consideration of a broad range of opinions is necessary when considering the design of organ allocation systems.

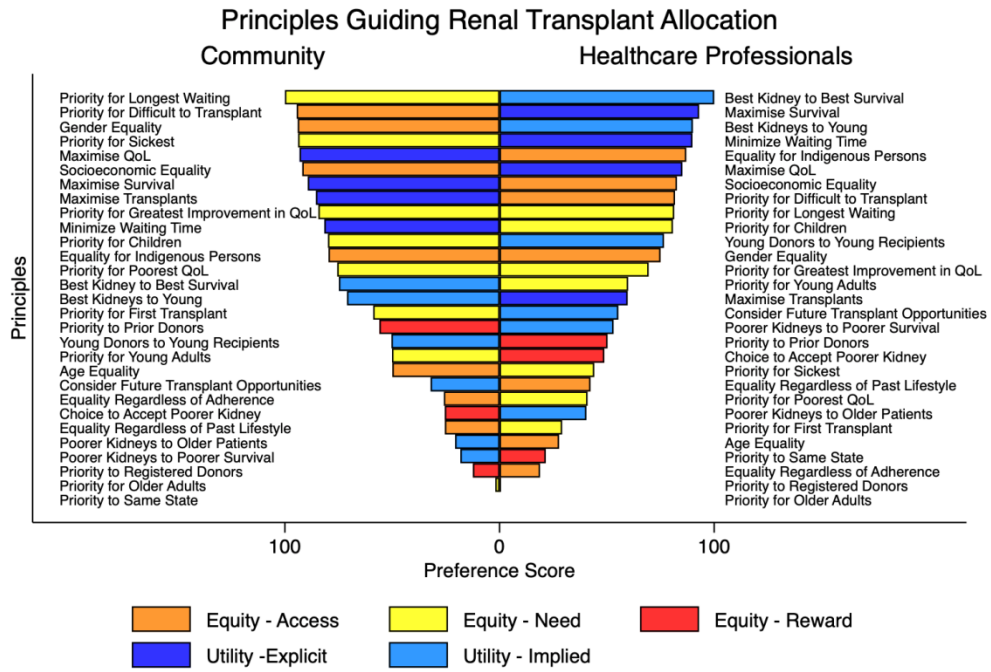


Figure 1: Comparison of community and healthcare professional preferences in principles guiding deceased donor kidney transplant allocation. Preference scores are derived from scaled coefficients of a sequential best worst multinomial logistic regression model and range from 0-100, with higher scores indicating greater prioritisation. QoL: Quality of Life.

Abstract No. 82

IATROGENIC LIVER INJURY SUSTAINED DURING DECEASED DONOR ORGAN PROCUREMENT: AN ANALYSIS OF THE RISK FACTORS AND CONSEQUENCES IN AN AUSTRALIAN TRANSPLANT CENTRE

WALCOTT James[1,2], FINK Michael[1], MURALIDHARAN Vijayaragavan[1], CHRISTOPHI Christopher[1,2]
[1]Department of Surgery, Austin Hospital, Melbourne, [2]Department of Surgery, University of Melbourne

Introduction: Liver transplantation is an established treatment for various liver diseases, and its success relies on the quality of the donated organ. Studies on procurement-related liver injury may not apply to modern day practice. This is the first Australasian study examining risk factors and consequences of procurement-related injury.

Method: The Victorian Liver Transplant database was examined for injuries from deceased liver donors for the calendar years 2010 – 2017. Information regarding the donor, the procurement surgery and subsequent transplantation was obtained. Injury information was sought from the "organ retrieval report form" (ORRF). Risk factors for injury were calculated using multivariate regression. Outcomes of complications and survival were calculated.

Results: A total of 639 offers for whole liver donation resulted in 468 transplantations of which there were 420 accompanying ORRF forms. There were 46 injuries in 45 livers. Aberrant anatomy increased the risk of vascular injury (OR 4.80 CI 1.99-611.60, $p<0.001$). Surgeon inexperience increased the risk of parenchymal injury (OR 16.24, CI 2.15-122.64, $p<0.01$). There was no difference in overall or graft survival for injured grafts. Complication rates were the same in the presence of injury with the exception of a decreased risk of anastomotic biliary strictures in the presence of a vascular injury (OR 0.13, CI 0.02-0.95, $p=0.04$).

Conclusion: This study shows that procurement related liver injuries are common, and that aberrant anatomy and surgeon inexperience increase the risk of injury. Similar outcomes for transplantation despite the presence of injury indicate that injuries are being appropriately managed in the Australasian setting.

Abstract No. 83

MISSED OPPORTUNITIES FOR ORGAN DONATION AMONG DONORS WITH PRIMARY BRAIN MALIGNANCIES (PBMS): NEW SOUTH WALES (NSW) COHORT STUDY 2010-2015

THOMSON Imogen[1,2], HEDLEY James[1], ROSALES Brenda[1], WYBURN Kate[3,1], WEBSTER Angela[1,4]

[1]School of Medicine, Faculty of Health Sciences, University of Sydney, [2]JMO Unit, John Hunter Hospital, Newcastle, [3]Renal Unit, Royal Prince Alfred Hospital, Sydney, [4]Centre for Transplant and Renal Research, Westmead Hospital, Sydney

Aims: Most PBMs are not a contraindication to organ donation as risk of tumour transmission is low. We sought to characterise organ donor referrals with PBMs in NSW, identify any transmission events, and describe missed opportunities for donation for referrals declined due to PBMs.

Methods: We undertook a retrospective review of 2010-2015 donor referrals in SAFEBOD, a register linking NSW Organ and Tissue Donation Service referral logs to donor and recipient health records, identifying referrals with PBMs. Tumours were classified using World Health Organization (WHO) grading and risk-assessed using Transplant Society of Australia and New Zealand (TSANZ) guidelines.

Results: Of 2957 referrals, 76 (3%) with PBMs were identified. Issues other than PBM meant that 47 (62%) were unsuitable for donation. 10 (13%) donors donated organs to 23 recipients in NSW. The remaining 19 (25%) were missed donor opportunities, and were significantly younger (47.7 vs. 58.6 years, p<0.001) and had fewer comorbidities (0.8 vs. 2.1, p<0.001) than referrals overall. WHO-I tumours were most common among PBM referrals (36, 47%) and PBM donors (8, 80%), but WHO-IV tumours predominated among missed opportunities (12, 68%) including glioblastoma multiforme (GBM) (11, 63%). All PBM donors had TSANZ 'low risk' or 'not contraindicated' malignancies. No referrals with GBM or ventriculo-peritoneal shunt donated organs. No transmission events occurred after 860 months total follow up.

Conclusion: There exist opportunities to increase organ donation rates in NSW through greater consideration of donors with PBM. This must be balanced against risk of transmission, especially when evaluating referrals with GBM.

Table: Characteristics of referrals and donors with and without PBMs

	Non-donors, N (%) p-value								Donors, N (%) p-value				
Characteristic	**All**		**All PBMs**			**Missed opportunities**			**All**		**PBM donors**		
Total	2398	-	66	-		19	-		559	-	10	-	
Age, mean (SD)	58.6	(18.8)	50.9	(21.4)	<0.001	47.7	(17.6)	0.01	50	(18.2)	44.4	(17.7)	0.4
Comorbidities, mean (SD)	2.1	(1.6)	1.6	(1.7)	0.01	0.8	(1.2)	<0.001	1.6	(1.4)	0.9	(0.7)	0.1
Cardiovascular disease	1595	(67)	29	(44)	<0.001	5	(26)	<0.001	325	(58)	3	(30)	0.1
Respiratory disease	328	(14)	6	(9)	0.4	1	(5)	0.05	70	(13)	1	(10)	0.9
Diabetes	418	(17)	9	(14)	0.5	0	(0)	0.06	18	(3)	0	(0)	0.6
Hypertension	798	(33)	16	(24)	0.1	3	(16)	0.1	170	(30)	3	(30)	0.9
Malignancy[1]	343	(14)	9	(14)	0.9	3	(16)	0.7	42	(8)	0	(0)	0.9
Liver disease	129	(5)	2	(3)	0.6	0	(0)	0.6	10	(2)	0	(0)	0.9
Kidney disease	206	(9)	5	(8)	0.9	0	(0)	0.4	12	(2)	0	(0)	0.9
High-risk behaviours, mean (SD)	0.1	(0.4)	<0.1	(0.2)	0.05	0	(0)	0.2	0.2	(0.6)	0	(0)	0.2
PBM Grade (WHO)[3]													
WHO-I	-		4	(72)		2	(10)		-		8	(80)	
WHO-II	-		6	(9)		1	(5)		-		2	(20)	
WHO-III	-		2	(3)		1	(5)		-		0	(0)	
WHO-IV	-		26	(39)		13	(68)		-		0	(0)	
PBM risk (TSANZ)													
Not contraindicated	-		38	(58)		3	(16)		-		10	(100)	
Low risk	-		2	(3)		1	(5)		-		0	(0)	
Intermediate	-		26	(39)		13	(68)		-		0	(0)	

[1] Presence of malignancy excluding PBM. [2] High-risk behaviours included drug use (IVDU non-IVDU), recent incarceration, sex work, high-risk partners and MSM). [3] Two missed opportunities with unclassified PBMs have been excluded from this section as grade and transmission risk could not be determined.

Abstract No. 84

IMPLEMENTATION OF INCREASED VIRAL RISK DONOR WAITING LIST IN VICTORIA – A USEFUL ADDITION TO THE DONOR POOL

LEE Darren[1,2], SENG Nina[3], GRAMNEA Indra[3], HUDSON Fiona[4], D'COSTA Rohit[3], MCEVOY Leanne[3], SASADEUSZ Joe[5], GOPAL Basu[6], KAUSMAN Joshua[7], MASTERSON Rosemary[8], PAIZIS Kathy[2], KANELLIS John[9], HUGHES Peter[8], GOODMAN David[10], WHITLAM John[2]

[1]Department of Renal Medicine, Eastern Health, Melbourne, [2]Department of Nephrology, Austin Hospital, Melbourne, [3]DonateLife Victoria, [4]Victorian Transplantation and Immunogenetics Service, Australian Red Cross Blood Service, [5]Department of Infectious Diseases, Royal Melbourne Hospital, [6]Department of Renal Medicine, Alfred Hospital, Melbourne, [7]Department of Nephrology, Royal Children's Hospital, Melbourne, [8]Department of Nephrology, Royal Melbourne Hospital, [9]Department of Nephrology, Monash Medical Centre, Melbourne, [10]Department of Nephrology, St Vincent's Hospital, Melbourne

Aims: Increased viral risk donors (IVRDs) with at-risk behaviours for blood borne virus infection and negative nucleic acid testing (NAT) have a low absolute risk of window period infection. A program allocating these kidneys to pre-consented recipients was recently implemented in Victoria. We reviewed the performance after seven months in operation.

Methods: We retrospectively compared the characteristics of IVRDs (defined by PHS 2013 criteria and open window periods) and non-IVRDs (31/07/2018-28/02/2019) and examined the serological and NAT results of IVRDs at donation and recipients post-transplant. Continuous data was expressed as median (IQR).

Results: 24.4% of waitlisted patients were pre-consented. Twelve IVRDs (23 kidneys) were utilised, comprising 13.5% of all kidneys transplanted. No suitable recipients were found for two IVRDs. Risk factors included intravenous drug use (58%) and increased risk sexual behaviour (42%). NAT was performed 3 (1-4) days after admission. Three IVRDs had abnormal serology results (2 HCV Ab positive, 1 HBcAb and HCV Ab positive) but negative NAT. Recipients of HCV Ab positive IVRDs seroconverted, but no viraemia was detected in recipients from all IVRDs to date. Compared with non-IVRDs (n=82), IVRDs were significantly younger (36 (29-43) versus 51 (36-60) years; $P<0.01$). Kidney donor profile index (KDPI) was significantly lower (21 (10-39) versus 60 (26-76); $P<0.01$), more likely ≤20 (50% versus 18%; $P<0.05$) and none had a KDPI >85 (0% versus 12%; $P=0.35$).

Conclusions: IVRDs appear to offer better quality kidneys from a historically under-utilised donor pool. Increasing the pre-consent rate may improve utilisation and benefit more waitlisted recipients.

Abstract No. 85

THE IMPORTANCE OF DONOR CANCER HISTORY IN DETERMINING USE OF KIDNEYS FROM CONSENTED DONORS

AU Eric HK[1,2], PHILLIPS Jessica[3], OPDAM Helen[4,5], MCDONALD Mark[6], CHAPMAN Jeremy R[7], MCDONALD Stephen[8,9], JOHNSON David[10,11], KANELLIS John[12], WONG Germaine[1,2,7], LIM Wai H[3,13]

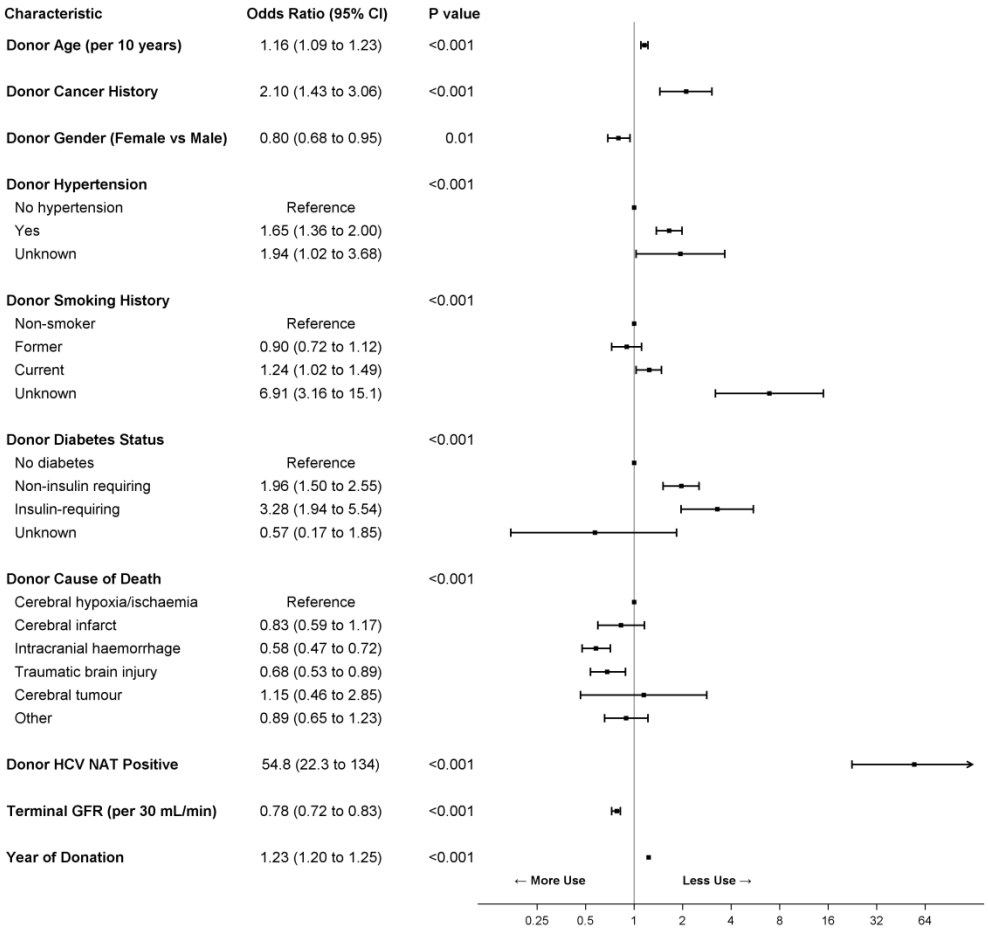
[1]School of Public Health, University of Sydney, [2]Centre for Kidney Research, The Children's Hospital at Westmead, Sydney, [3]Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth, [4]Organ and Transplant Authority, [5]Department of Intensive Care, Austin Hospital, Melbourne, [6]Organ and Tissue Authority, [7]Centre for Transplant and Renal Research, Westmead Hospital, Sydney, [8]ANZDATA, South Australian Health and Medical Research Institute, [9]Adelaide Medical School, University of Adelaide, [10]Department of Nephrology, Princess Alexandra Hospital, [11]Faculty of Medicine, University of Queensland, Brisbane, [12]Department of Nephrology, Monash Medical Centre, Melbourne, [13]Faculty of Health and Medical Sciences, University of Western Australia, Perth

Aim: To determine whether a history of prior donor cancer influences the decision to use kidneys from consented donors in Australia and New Zealand.

Methods: This study included all consented (intended and actual) donors recorded in the ANZDATA and ANZOD registries between 1989 and 2017. The relationship between donor factors [including prior cancer (excluding non-melanoma skin cancers), age, gender, ethnicity, HCV status, body mass index (BMI), comorbid conditions and terminal eGFR] and non-use of donor kidney (i.e. not retrieved or utilised) were examined using Least Absolute Shrinkage and Selection Operator (LASSO) penalised logistic regression and random forests.

Results: A total of 9485 donors (865 intended and 8620 actual) were identified [57.8% female, median age 42.9 years (IQR 28.0-57.0)]. Kidneys from 1645 (17.3%) donors were not retrieved or utilised. The most common cause of donor death was intracranial haemorrhage (21.0%). Donor factors associated with non-use [Odds Ratio (95% CI)] included increasing age [1.16 per 10 years (1.09-1.23)], cancer history [2.10 (1.43-3.06)], hypertension [1.65 (1.36-2.00)], current smoker [1.24 (1.02-1.49)], insulin-requiring diabetes mellitus [3.28 (1.94-5.54)], cause of death, HCV NAT positivity [54.8 (22.3-134)] and terminal eGFR [0.78 per 30 mL/min/1.73^2 increase (0.72-0.83)]. Donor ethnicity and BMI were not associated with non-use. The most important donor factors in determining non-use using random forest classifiers were terminal GFR and donor age.

Conclusion: Prior cancer history was associated with a 2-fold increased risk of non-use of consented donor kidneys. However, the most important donor factors in determining non-use were terminal eGFR and donor age.



Abstract No. 86

MISSED OPPORTUNITIES FOR ORGAN DONATION? USE OF LINKED HEALTH DATA TO VERIFY INCREASED BLOODBORNE VIRUS (BBV) RISK AMONG NSW ORGAN DONOR REFERRALS, 2010-2015

DE LA MATA Nicole[1], WALLER Karen[1], HEDLEY James H[1], ROSALES Brenda[1], KELLY Patrick J[1], WYBURN Kate[2,3], O'LEARY Michael[4], CAVAZZONI Elena[4], WEBSTER Angela C[1,5]

[1]School of Public Health, University of Sydney, [2]Sydney Medical School, University of Sydney, [3]Renal Unit, Royal Prince Alfred Hospital, Sydney, [4]NSW Organ and Tissue Donation Service, [5]Centre for Transplant and Renal Research, Westmead Hospital, Sydney

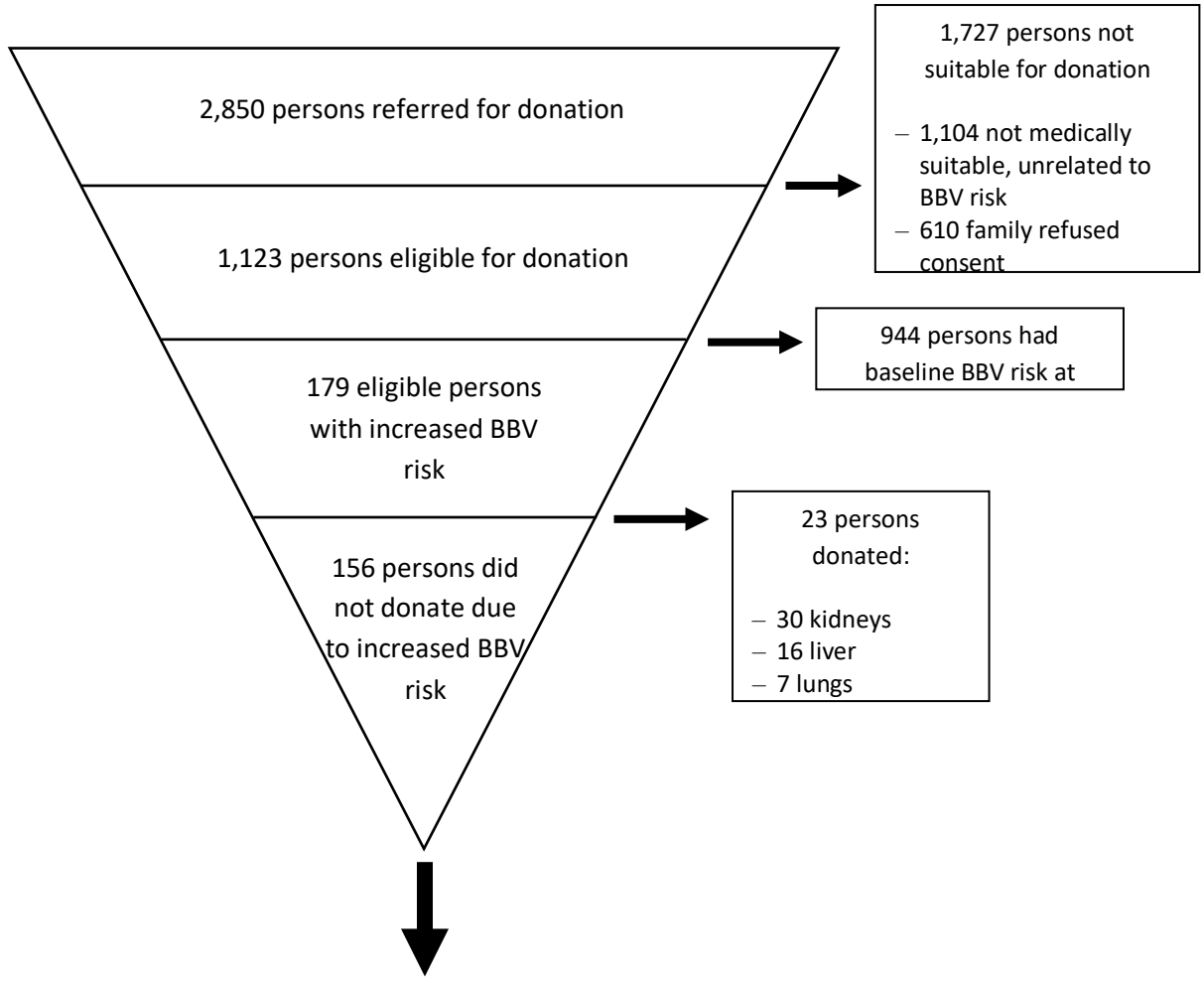
Background: Donors with bloodborne viruses (BBV) may transmit infection to transplant recipients. Often medical information is limited at the time of referral, and donors may be declined when social or medical history suggests increased risk of BBV without serology or NAT confirmation.

Aims: To determine potential missed opportunities for organ donation using linked health data when referrals were declined due to increased BBV risk at referral.

Methods: We included all donor referrals 2010-2015 from the NSW Biovigilance Public Health Register, SAFEBOD. This Register linked donor referrals in NSW to administrative health databases, including the hospital admissions data, notifiable conditions information management system and the death register. Increased BBV risk at referral was defined as an active infection, past infection or high-risk behaviour for HIV, HBV or HCV. In the Register, active or past infection were indicated by ICD-10-AM codes in NSW health datasets. **Results:** Of 2,850 persons referred for organ donation in NSW, 156 persons did not donate due to increased BBV risk at referral (Figure 1). Among these, no evidence of active infection was found in 150 persons for HIV, 141 persons for HBV and 84 persons for HCV. Overall, 75 of 156 persons referred who did not donate due to increased BBV risk at referral had no evidence of active BBV infection up to their terminal contact with health services.

Conclusions: Utilizing routinely collected administrative health data has identified potential missed donation opportunities and, if available in real-time, may provide a useful additional information source to aid decision-making.

Figure 1. Potential missed opportunities for organ donation due to increased bloodborne virus (BBV) risk in NSW, 2010-2015.



BBV Infection	Known active infection at referral	Additional active infection in SAFEBOD	No evidence of active infection
Any BBV	69	12	75
HIV	4	2	150
HBV	11	4	141
HCV	60	12	84

Abstract No. 87

ADDRESSING TRANSPLANT TOURISM INTO CHINA

MATAS David
Law firm, Barrister and Solicitor

Aims: The aim of the presentation would be to set out recommendations for action by the Transplantation Society of Australia in addressing transplant tourism into China.
Methods: The presentation would consider 1) the open letter to Xi Jinping, President of China, on organ transplantation abuse in China, signed on behalf of the Transplantation Society by, amongst others, Australian transplant doctors and leaders Jeremy Chapman and Philip O'Connell, published in April 2014, and, in particular, the component of that letter which refers to the recruitment of international patients seeking organ transplants by the then Tianjin Web site http://www.cntransplant.com ; 2) the claims by the Chinese government of changes in their transplantation system to address the concerns set out by the Transplantation Society; 3) the Parliament of the Commonwealth of Australia Human Rights Sub-Committee House of Representatives Joint Standing Committee on Foreign Affairs, Defence and Trade report of November 2018 titled "Compassion, Not Commerce: An Inquiry into Human Organ Trafficking and Organ Transplant Tourism" and in particular the recommendation that the Government of Australia continue to express concern to China regarding allegations of organ trafficking in that country; and 4) the current version of the Tianjin Web site at http://www.tran-kid.com/index.html .
Results: The concerns expressed by the Transplantation Society in April 2014 remain valid today. The claims of the Chinese government of change are not independently verifiable and more cosmetic than real. **Conclusions:** The Transplantation Society needs to update and continue its expression of concerns set out in April 2014.

Abstract No. 88

BLOODBORNE VIRUS (BBV) INFECTIONS IN NSW ORGAN DONOR REFERRALS USING LINKED HEALTH DATA: THE SAFEBOD COHORT, 2010-2015

DE LA MATA Nicole[1], WALLER Karen[1], HEDLEY James H[1], ROSALES Brenda[1], KELLY Patrick J[1], WYBURN Kate[2,3], O'LEARY Michael[4], CAVAZZONI Elena[4], WEBSTER Angela C[1,5]

[1]School of Public Health, University of Sydney, [2]Sydney Medical School, University of Sydney, [3]Renal Unit, Royal Prince Alfred Hospital, Sydney, [4]Other, NSW Organ and Tissue Donation Service, [5]Centre for Transplant and Renal Research, Westmead Hospital, Sydney

Background: Misclassification of bloodborne viruses (BBV) may lead to potential donors being unnecessarily declined, or to infected donor organs being used.

Aims: To compare perceived BBV risk at referral with verified BBV risk from linked health datasets.

Methods: We included all donor referrals, 2010-2015, from the NSW Biovigilance Public Health Register (SAFEBOD). This Register linked all organ donor referrals in NSW to administrative health databases, including the hospital admissions data and notifiable conditions information management system. Perceived BBV risk was based on information available at referral, including serology, nucleic acid testing, and high-risk behaviour. Verified BBV risk was based on ICD-10-AM codes indicating active infection, past infection and exposure to HIV, HBV or HCV in SAFEBOD. We used cross tabulations and Cohen's Kappa to compare perceived and verified BBV risk.

Results: Of 2,850 donor referrals, 1,727 were either not medically suitable or their family declined consent. Among the 1,123 remaining referrals, agreement between perceived and verified BBV risk occurred for 1,010 persons (934 baseline BBV risk; 75 active BBV infection; 1 past BBV infection) (Table 1). There was substantial agreement for any active BBV infection (Kappa=0.69, p value<0.001). Eight referrals perceived to have baseline risk were verified with active BBV infection (1 HIV; 3 HBV; 4 HCV). 15 active infections at referral (1 HIV; 4 HBV; 10 HCV) were not verified from linked health data.

Conclusions: Data linkage to existing administrative health data can aid assessment of donor referrals, revealing unrealised biovigilance risk to recipients and potential additional donor opportunities

Table 1. Agreement of bloodborne virus (BBV) risk perceived at the time of referral compared to verified from linked health data in NSW, 2010-2015.

		Verified BBV risk from linked health data				
		Baseline	**Exposed**	**Past**	**Active infection**	**Total**
Perceived BBV risk at referral	**Baseline**	934	1	1	8	944
	High risk behavior	26	0	5	7	38
	Past	20	0	1	8	29
	Active infection	15	0	22	75	112
	Total	995	1	29	98	1,129

Note: Grey cells are disagreements and white cells are agreements between perceived and verified BBV risk.

Abstract No. 89

EXTENDING LIVING KIDNEY DONOR SELECTION CRITERIA: A BIOETHICAL PERSPECTIVE

WEIGHTMAN AC[1,2]

[1]Renal & Transplantation Unit, Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [2]Monash Bioethics Centre, Monash University, Melbourne

Current guidelines for the assessment of living kidney donors are one-dimensional: there is exclusive focus on risks to the donor's health while benefits for donors and recipients are intentionally excluded. The donor must have negligible risk for detrimental health outcomes as assessed by the transplant physician, resulting in the rejection of many willing people based on their potential to develop kidney or cardiovascular disease later in life. There is no opportunity for donors to consent to transplants with a higher risk of long-term complications. As well as raising issues of unjustified paternalism, this process also perpetuates social inequity by excluding donors in lower socioeconomic and Indigenous populations due to their pre-existing disproportionate burden of cardiovascular disease, diabetes and kidney failure. I argue that a more liberal approach to donor assessment would be ethically justifiable. It is not unreasonable for a person to willingly sacrifice their own health in order to help another, particularly their child or spouse. Donor assessment should incorporate a broader understanding of health, including the relationship with the recipient and the benefits of donation (psychological, financial and vicarious). The transplant physician still has moral responsibilities to prevent excessively dangerous or coercive transplants from taking place, but should be guided by the donor when determining the significance of procedural risks. Donor assessment should be used to facilitate autonomous decision-making with the opportunity for appropriately motivated donors to consent to riskier transplants.

Abstract No. 90

INCIDENCE AND OUTCOMES OF ANTIBODY MEDIATED REJECTION IN THE AUSTRALIAN AND NEW ZEALAND RENAL TRANSPLANT POPULATION

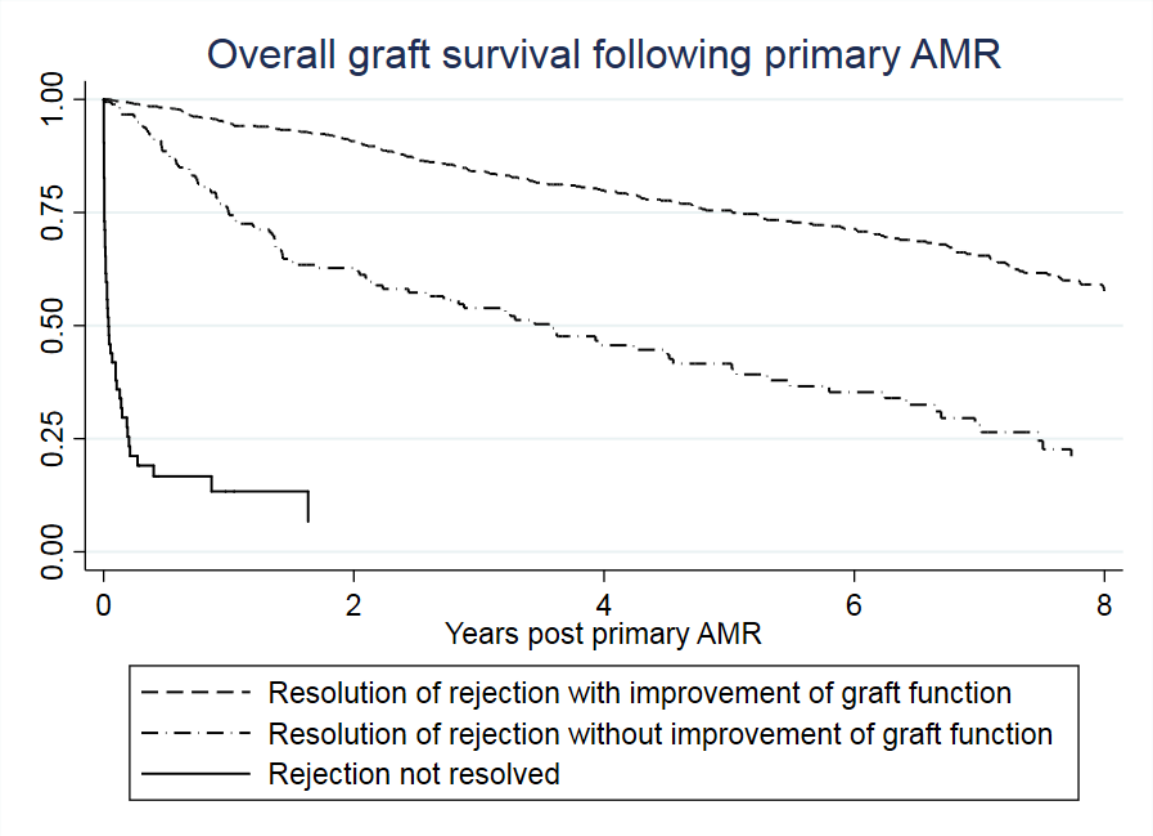
DANSIE Kathryn[1], CLAYTON Philip[2]

[1]Australia and New Zealand Dialysis and Transplant Registry (ANZDATA), South Australian Health and Medical Research Institute (SAHMRI), [2]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital

Aim: To describe the incidence and outcomes of antibody mediated rejection (AMR) in the Australian and New Zealand renal transplant population.

Methods: Using data from the Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry, we included all antibody mediated rejection episodes for patients transplanted from 2005-2017. Incidence of AMR was calculated separately for less than and greater than 6 months following transplantation. Treatment regimens were examined through descriptive analysis and graft survival was observed from Kaplan-Meier curves. **Results:** There were 1426 AMR cases for transplants conducted between 2005 and 2017, with an incidence rate of 12.87 rejections per 100 transplant years for less than 6 months following transplantation and 1.07 rejections per 100 transplant years for greater than 6 months following transplantation. A large variation in treatment regimens was observed (76 different treatment combinations) across 80 treating hospitals in Australia and New Zealand, with "steroids, plasmapheresis and intravenous immunoglobin"; "plasmapheresis and intravenous immunoglobin" and "steroids only" being most frequently employed (18.6%, 11.0% and 8.8% respectively). Median graft survival following primary AMR was 8 years which varied with response to treatment. Patients whose rejection episode resolved, with or without improvement of graft function, fared better than patients whose rejection episode did not resolve median survival for resolved: 8.5 years; not resolved: 0.04 years)(figure).

Conclusions: There is considerable variation in the treatment of AMR episodes in the Australian and New Zealand renal transplant population. Graft survival following AMR is greater for patients whose AMR episodes were completely or partially resolved.



Abstract No. 91

SUCCESSFUL SIMULTANEOUS LIVER-KIDNEY TRANSPLANT WITH HIGH LEVEL DSA USING LOW-INTENSITY IMMUNOSUPPRESSIVE REGIMEN

HOWSON Prue[1], KULKARNI Hemant[2], CHAKERA Aron[1], LIM Wai H[1]

[1]Nephrology and Renal Transplant, Sir Charles Gairdner Hospital, Perth, [2]Renal Unit, Royal Perth Hospital

Simultaneous liver kidney (SLK) transplantation in the presence of a positive CDC-crossmatch is not considered a contraindication, with the expectation that the liver allograft exerts an immunoprotective effect on the kidney allograft. However, there are case reports suggesting that highly-sensitised SLK recipients with strong pre-transplant donor-specific anti-HLA antibody at high titres may experience early antibody mediated rejection. Consequently, induction therapy using T cell depleting antibody and higher intensity maintenance immunosuppression are often advocated for these patients.

We present a case of a sensitised 51 year old woman with polycystic liver kidney disease who had received a SLK transplant. Although pre-transplant CDC was negative, she had moderate to high titres of pre-transplant donor-specific anti-HLA antibody (antibody to B*07:02 with MFI of 17,570 and to DQB1*03:03 with MFI of 2333). Induction therapy was not given and she was initiated on standard tacrolimus (target trough 5-8ng/ml), mycophenolate (1g bd) and oral prednisolone. Protocol biopsy at 3-months post-transplant was unremarkable. The development of CMV syndrome and diarrhoea at 8-months post-transplant necessitated anti-viral therapy with subsequent switch from mycophenolate to azathioprine. Both B*07:02 and DQB1*03:03 anti-HLA antibodies were no longer detected at 3 weeks post-transplant, although there was a re-emergence of moderate B1*03:03 at 10-months post-transplant (MFI of 1419). At 18 months post-transplant, she has normal liver and kidney function and has not experienced acute rejection. This case suggests that low-intensity immunosuppressive regimen without induction therapy may be safe in moderate immunological risk SLK transplant recipients.

Abstract No. 92

IDENTIFICATION OF DONOR ANTI-HLA ANTIBODIES IN MULTIPLE TRANSPLANT RECIPIENTS

HIHO Steven[1,2], KUMMROW Megan[2], LEVVEY Bronwyn[3], SULLIVAN Lucy[4,5], WESTALL Glen[1], SNELL Greg[1]

[1]Lung Transplant Unit, Alfred Hospital, Melbourne, [2]Transplantation Laboratory, Australian Red Cross Blood Service, [3]Lung Transplant Service, Alfred Hospital, Melbourne, [4]Department of Microbiology, University of Melbourne, [5]Department of Microbiology, Doherty Institute

Monitoring for anti-HLA antibodies post-transplant is an important tool in early detection of rejection episodes and may direct clinical treatment therapies. We recently reported a case of the passive transfer of HLA antibodies in all organ recipients from a cadaveric donor. These anti-HLA antibody profiles were not directed towards donor HLA, but matched the donors HLA antibody profile. Here we describe this and another case and the clinical management used.

Method: Patient and donor HLA antibody testing was performed with Luminex single antigen beads (One Lambda) with a MFI cut-off of 1000. HLAMatchmaker (V2.1) was used for eplet analysis, and HLA typing was performed using Luminex LABType (One Lambda) for recipients and donors.

Results: The HLA class I antibody specificities were identified to be directed at the 65GKA & 166ES eplets and were identified in all organ recipients of each donor and were not Donor Specific Antibodies (DSAs). Pregnancy and sensitising history of donors were unable to be confirmed but donor antibody profiles suggest at least one sensitising event. Lung recipients had normal lung function at 8 and 12 months post-transplant, however one patient died of pancreatitis (with normal lung function).

Conclusion: We suggest that donor B cells, were passengers with the transplanted organs, and appear to be a variant of Passenger lymphocyte syndrome (PLS) that may complicate the post-transplant treatment and identification of DSA. Further studies are continuing to identify cases and confirm origin of these antibodies.

Abstract No. 93

SURGICAL APPROACHES FOR MANAGING THE DIFFICULT ABDOMINAL WALL IN KIDNEY TRANSPLANT RECIPIENTS: CASE REPORTS

LAM Susanna, LAURENCE Jerome, VERRAN Deborah
Transplantation Services, Royal Prince Alfred Hospital, Sydney

Introduction: The surgical approaches for managing early deep abdominal wall complications in the setting of infection in kidney transplant patients are not well described. Managing wound complications is difficult and the use of mesh can be controversial. We describe successful management of wound dehiscence and renal allograft compartment syndrome (RACS) in the context of contamination or infection, using a combination of mesh and negative pressure wound therapy (NPWT) in a series of kidney transplant recipients.
Aims: To describe the surgical treatment of abdominal wall dehiscence and RACS in the setting of infection in kidney transplant recipients.
Methods: Retrospective analysis of kidney transplant recipients from 2015-2017 was performed.
Results: Out of 341 recipients, 5(1.5%) required reoperation for dehiscence or suspected RACS. Average age was 54-years and BMI: 33kg/m^2. Average time to dehiscence was 20-days. Surgical repair involved securing preperitoneal mesh to fascia, with NPWT placed over mesh either primarily or after failed primary wound closure secondary to wound infection. Average time to wound healing was 79-days. Time to reoperation for RACS was 3 days. Once allograft perfusion is confirmed, onlay polyglactin mesh was sutured into fascia, over the allograft and NPWT placed over mesh. Time to wound healing was 72-days.
Conclusion: We describe an approach to managing the difficult abdominal wall in the context of fascial dehiscence and RACS by using a combination of abdominal wall mesh repair and NPWT which has allowed for successful wound healing in complex, immunosuppressed kidney transplant patients.

Abstract No. 94

USING DONOR BILE DUCT INJURY SCORES TO PREDICT BILIARY STRICTURES AFTER LIVER TRANSPLANTATION: RESULTS FROM THE AUSTRALIAN NATIONAL LIVER TRANSPLANTATION UNIT

LY Mark[1], PULITANO Carlo[2,3], MCKENZIE Catriona[4], KENCH James[4,3], CRAWFORD Michael[2]
[1]Royal Prince Alfred Hospital, Sydney, [2]Australian National Liver Transplantation Unit, Royal Prince Alfred Hospital, Sydney, [3]University of Sydney, [4]Department of Tissue Pathology and Diagnostic Oncology, Royal Prince Alfred Hospital, Sydney

Introduction: Donor histological bile duct injury scores have been correlated with the incidence of Biliary Strictures (BS) after liver transplantation, particularly peribiliary gland injury, mural necrosis and vascular plexus injury. We performed a clinical study to evaluate the correlation between bile duct injury scores and the development of BS after liver transplantation in Australian donors.
Methods: Bile duct samples were taken from brain dead donors retrieved and transplanted in NSW, Australia from March 2016 and June 2017. Samples were taken at the backtable, prior to transplantation. Samples were scored according to the grading system developed by Op Den Dries et al by two independent pathologists who were blinded from outcomes. Recipients were followed up and the primary outcome was BS at 12 months. Data on donor, recipient characters and post-operative complication data was extracted.
Results: Bile duct samples were taken from 58 grafts and the overall incidence of BS was 25.9% (n=15). There were 13 Anastomotic Strictures (AS) and 3 Non-Anastomotic Strictures. Decreased levels of inflammation in bile duct samples was associated with BS ($p=0.005$) and AS ($p=0.005$). All samples had evidence of biliary epithelial injury however this was not associated with BS. No association was observed for mural stromal necrosis, vascular plexus injury or peribiliary gland injury, and developing BS at 12 months ($p>0.05$).
Conclusions: Decreased inflammation scores were associated with Biliary and Anastomotic Strictures which may reflect poorer microvascular circulation. Our study did not observe the same correlations between bile duct injury scores as previous studies.

Abstract No. 95

COMPLICATIONS RESULTING FROM THE USE OF A SURGICAL MESH PATCH IN PAEDIATRIC RENAL TRANSPLANT RECIPIENTS

DURKAN Anne[1,2], TAHER Amir[1], THOMAS Gordon[3,2], SHUN Albert[3,2]

[1]Department of Nephrology, The Children's Hospital at Westmead, Sydney, [2]School of Medicine, University of Sydney, [3]Department of Surgery, The Children's Hospital at Westmead, Sydney

Aims: To review the complications encountered in paediatric renal transplant recipients in whom a surgical mesh patch, Surgisis, (Cook Surgical, Bloomington, IN) was used to aid closure of the abdomen and prevent abdominal compartment syndrome.
Methods: A retrospective manual case note review was performed for all paediatric renal transplant recipients at a single centre between Sept 2006 and Sept 2018. Clinical and epidemiological data were collected.
Results: 111 transplant recipients were identified of whom seven had a surgical mesh placed. The median age at transplantation of these seven patients was 2.8 years and the median weight was 9.8kg compared to 10.8 years and 27kg, respectively, in those without mesh. Five children had a living related donor and just one had a pre-emptive transplant. Three children developed large fluid collections between the subcutaneous layers and the mesh, requiring surgical drainage in the early post-operative period. Three children presented 2, 10 and 26 weeks after transplantation with vomiting and abdominal pain, including one child who had required surgical drainage of a fluid collection. At laparotomy, all three were found to have bowel obstruction related to the mesh, necessitating bowel resection in one child. A fourth child presented 11 years post transplantation with bowel obstruction requiring bowel resection, but an association with the mesh could not be established in this patient.
Conclusions: The use of a surgical mesh patch in young renal transplant recipients is associated with a high complication rate. The routine use of these patches should be avoided.

Abstract No. 96

ATYPICAL HAEMOLYTIC URAEMIC SYNDROME POST-TRANSPLANTATION

SO Sarah, SPICER Timothy

Renal Unit, Liverpool Hospital

Case: A 72-year-old lady, seven months post-renal transplant for polycystic kidney disease, was admitted with diarrhoea and acute kidney injury. Her creatinine was 250 umol/L (baseline 150 umol/L) and haemoglobin 106 g/L. Stool cultures and CMV PCR were negative. Immunosuppression included Prednisone 12.5 mg daily, Sirolimus 4 mg daily and Tacrolimus 5 mg twice daily (intolerant of Mycophenolate due to leukopaenia). After one week, she developed recurrent episodes of pulmonary oedema and creatinine rose from 271 to 500 umol/L over 72 hours, requiring CVVHDF. She developed microangiopathic haemolytic anaemia with a haemoglobin drop to 79 g/L, thrombocytopaenia (84x10^9/L), and fragments on blood film. At D12, Sirolimus was ceased, Prednisone increased and Mycophenolate recommenced. Differentials included acute rejection, thrombotic thrombocytopaenic purpura or haemolytic uraemic syndrome. Repeat stool cultures were negative. ADAMTS13 level normal (18%). Tacrolimus was ceased at D23. At D26, she commenced intravenous methylprednisolone to empirically treat rejection and received her first dose of eculizumab for possible aHUS. A renal biopsy subsequently demonstrated acute tubular necrosis. Renal function improved and dialysis was ceased at D37. Tacrolimus was recommenced at D51 without further haemolysis. At discharge, her creatinine was 140 umol/L and she remains on fortnightly eculizumab.
Conclusions: This case demonstrates post-transplant aHUS in a patient being treated with combined mTOR and calcineurin inhibitors. Combination treatment may be higher risk for causing aHUS compared to usage of either agent alone. The successful reintroduction of Tacrolimus suggests that graft outcomes can be reasonable after drug-associated aHUS, if the mTOR inhibitor is ceased.

Abstract No. 97

METABOLIC HEALTH POST LIVING KIDNEY DONATION

GUO Henry, MCGINN Stella, LI Yan

Department of Nephrology & Transplantation, Royal North Shore Hospital, Sydney

Background: Kidney transplantation remains the best treatment for ESRD, with living donation now accounting for 24% of transplants in Australia. With recent evidence pointing towards a small increased lifetime risk of developing ESRD, estimating pre-existing cardiovascular burden and identifying modifiable risk factors is increasingly important.

Aim: To analyse metabolic health of living kidney donors (LKD) pre and post donation.

Methods: LKDs from 2008-2018 who were followed up for at least 12 months were included. Demographics, baseline weight, body mass index (BMI), glycaemic status and cardiovascular risk factors were obtained from medical records.

Results: 78 LKDs were included with mean follow up of 38 months. Median age at donation was 53±10.4 years with majority being Caucasian (80%) and female (61%). Mean baseline weight was 75.4kg, corresponding to BMI of 26, which increased to 82kg (BMI=29) by 1-year post donation. Five LKDs gained ≥10kg in the first year post donation. Mean reduction in eGFR at 1-year was 25±10 mL/min/1.73m^2 with no difference between overweight and healthy donors. Twelve donors developed new hypercholesterolemia, all of which were associated with weight gain in the first year post donation. Mean pre-donation BP was 121/76 mmHg with 11% diagnosed as hypertensive. Blood pressure and incidence of hypertension remain unchanged at 1 year. At baseline, 4 LKDs had impaired glucose tolerance, however none developed diabetes during the follow up period.

Conclusions: Despite stringent screening practices pre-donation, continued vigilance with long term monitoring of weight, glycaemic status and other risk factors remains important after living kidney donation.

Abstract No. 98

ASPERGILLUS PROSTATITIS FOLLOWING RENAL TRANSPLANTATION

HEPBURN Kirsten[1], ELIAS Anthony[2], KIM Dana[1], KOTSIOU George[2,3], MCGINN Stella[1,3]

[1]Department of Nephrology, Royal North Shore Hospital, Sydney, [2]Department of Infectious Diseases, Royal North Shore Hospital, Sydney, [3]School of Medicine, University of Sydney

Background: Urinary tract infections are a common complication following renal transplantation. We report a case of fungal granulomatous prostatitis presenting as pyuria with severe dysuria in a renal transplant patient.

Case: A 68-year-old male, who had a low risk deceased donor renal transplant, presented with severe dysuria and urinary frequency on a background of standard immunosuppression with no rejection episodes. He developed post-transplant diabetes, had prolonged catheterization following lymphocele marsupialization and had a prolonged course of antibiotics for pneumonia. Serial urine microscopies demonstrated heavy pyuria (>100x10^6/L) and haematuria without significant growth on culture. Despite empiric antimicrobial treatment and reduction of mycophenylate, his symptoms worsened and C-RP (141mg/L) and WCC (25.6x10^9/L) increased. Ultrasound demonstrated urothelial thickening of the pelvicalyceal system and a markedly enlarged prostate (98ml). Screens for mycobacteria, CMV, adenovirus, BK virus and STI causes of urethritis were negative. PSA was normal (3.8µg/L). Cystoscopy revealed a large prostate and normal transplant retrograde pyelogram. Ureteric aspirate revealed no white cells. Examination of multiple bladder biopsies and prostate secretions was unremarkable. MRI prostate demonstrated innumerable abscesses. A TURP was performed. Histopathology of prostate chips demonstrated granulomatous and suppurative inflammation associated with fungal hyphae and small foci of adenocarcinoma. Prolonged culture yielded *Aspergillus fumigatus*. He was commenced on voriconazole with improvement in symptoms.

Conclusion: Persistent urinary symptoms without positive urine cultures in an immunosuppressed patient should prompt further investigation for unusual causes. Fungal granulomatous prostatitis is rare, and in this case required dedicated MRI imaging followed by biopsy for diagnosis.

Abstract No. 99

AN UNUSUAL CAUSE OF HEADACHE IN A RENAL TRANSPLANT PATIENT

HEPBURN Kirsten[1], KIM Dana[1], DARBAR Archie[2,3], POLLOCK Carol[1,3]

[1]Department of Renal Medicine, Royal North Shore Hospital, Sydney, [2]Department of Infectious Diseases, Royal North Shore Hospital, Sydney, [3]School of Medicine, University of Sydney

Background: Aggressive, potentially fatal, fungal infections are increasing in frequency in immunosuppressed patients. We report a case of *Aspergillus Fumigatus* sinusitis with extension into the pituitary fossa in a renal transplant patient.

Case: A 53-year-old male with a previous splenectomy, presented with a subacute history of bifrontal headaches, 7-months post renal transplant for IgA nephropathy complicated by early T cell mediated rejection managed with IV methylprednisolone. Maintenance immunosuppression was tacrolimus, prednisolone and mycophenolate. He was admitted with an unrelated small bowel obstruction, but a CT sinuses revealed extensive opacification in the left sphenoid sinus with erosion of the adjacent anterior wall of the pituitary fossa. Dedicated pituitary MRI revealed fungal sinusitis with invasion through the sellar floor, displacing the normal pituitary gland superiorly. Pituitary function was normal except a sick euthyroid pattern, and cortisol suppression consistent with prednisone treatment. There was no ocular or cranial nerve involvement. Urgent left trans-nasal sphenoidotomy revealed inflamed mucosa, granulation tissue with congealed pus and white fungal spores. Biopsy demonstrated fungal hyphae with evidence of osteomyelitis, with *Aspergillus fumigatus* identified on culture. He was treated with 4 weeks of liposomal amphotericin in combination with voriconazole, then voriconazole monotherapy for a further 6 weeks. Immunosuppression was reduced. Follow up imaging confirmed marked improvement following antifungal therapy.

Conclusion: Fungal sinus infections should be considered as a cause of headaches in transplant patients. Dedicated MRI imaging with gadolinium enhancement and biopsy is essential for diagnosis. Combination antifungal therapy may offer the greatest treatment efficacy in these severe infections.

Abstract No. 100

CRYPTOCOCCUS INFECTION IN RENAL TRANSPLANT RECIPIENTS: A CASE CONTROL STUDY

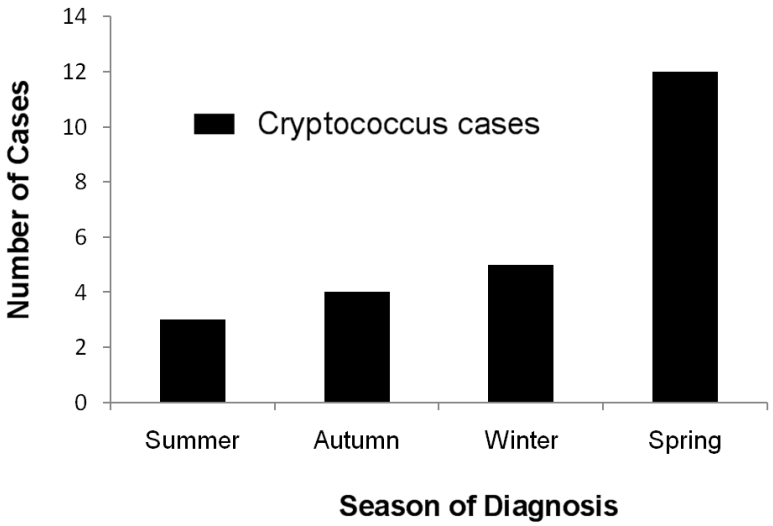
ZAHOROWSKA Beata[1], CHEN Sharon CA[2], KABLE Kathryn[3], NANKIVELL Brian J[3]

[1]Renal Unit, Liverpool Hospital, [2]Department of Infectious Diseases, Westmead Hospital, Sydney, [3]Renal & Transplantation Unit, Westmead Hospital, Sydney

Background: Renal transplant recipients have among the lowest incidence of invasive fungal infections compared with other solid organ transplants, however where fungal infection does occur *Cryptococcus* infection predominates and is associated with significant morbidity and mortality. We undertook to investigate the risk factors for *Cryptococcus* infection in renal and renal and pancreas transplant recipients.

Methods: A retrospective, matched case control analysis of *Cryptococcus* infection cases occurring between June 2005 and June 2017 post transplantation at our centre was performed.

Results: 24 cases of *Cryptococcus* infection post renal transplant were analysed with 2:1 control transplant recipients. Tacrolimus level and prednisone dose were identified as risk factors for *Cryptococcus* infection in this population (p=0.038 and p=0.019, respectively) and *Cryptococcus* infection was significantly associated with the outcome of death or graft loss at 12 months (odds ratio 4.53 [95% CI 1.15-14.89]). A novel finding of increased case diagnosis in Australian spring months was observed.



Conclusion: *Cryptococcus* infection risk factors confirmed in this study can be used to inform clinical practice of immunosuppression management and guide further research into *Cryptococcus* screening and potential prophylaxis design.

Abstract No. 101

A CASE OF MALAKOPLAKIA MIMICKING MALIGNANCY IN A RENAL TRANSPLANT PATIENT

SRINIVASA Vinay, GOVINDARAJULU Sridevi

Department of Nephrology, Darling DOwns Hospital and Health Services

Introduction: Malakoplakia is a rare granulomatous disease, occurring in immunocompromised patients. It is associated with immunosuppression in solid organ transplantation, particularly with kidney transplantation.

The true incidence of this condition is not known. 700 cases have been reported in the literature. Over 40 of these patients are kidney transplant patients. In Australia, 8 cases of malakoplakia have been described in the literature; only 2 cases including ours, reported bladder involvement.

Case Presentation: A 53-year-old male, renal transplant recipient, on standard triple immunosuppressive therapy, presented to clinic with frank macroscopic haematuria and history of two episodes of urinary tract infection in 3 months. Other positive history included sigmoid diverticulitis necessitating antibiotic treatment 3 months ago. His background renal disease was Autosomal Dominant Polycystic Kidney Disease.

Bladder ultrasound and abdominal CT confirmed a suspicious mass in the sigmoid colon and bladder. Primary malignancy of sigmoid colon with urinary bladder invasion and metastases was suspected. PET reported a FDG avid mass involving the sigmoid colon invading into the bladder. An enlarged FDG avid portocaval lymph node was observed. Colonoscopy showed no endoluminal mass within the sigmoid colon. Cystoscopy and biopsy followed. Surprisingly, histology revealed Michaelis-Guttman bodies typical of Malakoplakia. The patient underwent high anterior resection of rectum and resection of the colovesical fistula.

Conclusion: Malakoplakia is a rare granulomatous disorder associated with standard immunosuppressive therapy in transplant patients. Clinical presentation may mimic malignancy and awareness of this condition is important.

Abstract No. 102

NOT JUST A SIMPLE LUMP

SCOTT Tahira[1,2], YAXLEY Julian[3,4]

[1]Townsville Hospital, [2]Other, University of Queensland, Brisbane, [3]Renal Unit, Gold Coast University Hospital, [4]University of Queensland, Brisbane

Background: *Cryptococcus neoformans* is a common fungal infection in immunosuppressed individuals. Patients on calcineurin inhibitors and corticosteroids have weak innate and T-cell mediated immunity which limits host defence against Cryptococcus (1). Cryptococcal infection has protean manifestations, including meningitis, pneumonia and less frequently skin lesions.

Skin manifestations are uncommon, affecting 10-20% of patients, and are mostly observed in patients that have undergone solid organ transplantation. If present, skin involvement usually indicates disseminated Cryptococcus. The presentation of Cryptococcal skin disease is variable and includes papules, pustules, nodules, ulcers, and cellulitis (2). Inflammatory fungal cellulitis may occur concomitantly with bacterial cellulitis and is clinical indistinguishable, often leading to delayed diagnosis.

Clinical Case: A 43-year-old male presents with a lower thigh lump that had rapidly progressed in size within a week. He had undergone living donor kidney transplantation seven years prior for End-Stage Kidney Disease from IgA nephropathy. His maintenance immunosuppression comprised daily mycophenolate mofetil 2g, tacrolimus 4mg, and prednisolone 5mg. The transplant had been complicated prior with pneumocystis jirovecii pneumonia and cytomegalovirus viraemia.

Examination revealed an erythematous elevated hyperaemic plaque over the anterolateral aspect of the distal thigh. Magnetic resonance imaging showed cutaneous and subcutaneous oedema with subcutaneous fat stranding. Punch biopsy was performed. Histology demonstrated florid granulomatous reaction to numerous yeast organisms in the dermis and subcutis, and microscopy and culture detected *Cryptococcus neoformans* with a reactive serum Cryptococcal antigen assay of 1:256. Disseminated disease was confirmed with chest computer tomography findings suggestive of Cryptococcoma. There was no evidence of central nervous system involvement on further testing. Induction antimicrobial therapy was commenced with amphotericin B and 5-fluorocytosine, and mycophenolate mofetil was reduced to 1.5mg daily. Following induction treatment the patient was then continued on life-long maintenance fluconazole.

Conclusion: This case outlines the importance of suspecting cutaneous lesions in transplants patients as manifestations of opportunistic infections namely disseminated cryptococcus infection.

References

1. Singh N, Dromer F, Perfect JR, Lortholary O. Cryptococcosis in solid organ transplant recipients: current state of the science. Clin Infect Dis. 2008;47(10):1321-7.

2. Hayashida MZ, Seque CA, Pasin VP, Enokihara M, Porro AM. Disseminated cryptococcosis with skin lesions: report of a case series. An Bras Dermatol. 2017;92(5 Suppl 1):69-72.

Abstract No. 103

SUCCESSFUL KIDNEY TRANSPLANTATION OF A HEPATITIS B SURFACE ANTIGEN MUTANT POSITIVE DONOR TO A HEPATITIS B NEGATIVE RECIPIENT

WILSON Gregory[1,2], FRANCIS Ross[1,2]

[1]Department of Nephrology, Princess Alexandra Hospital, Brisbane, [2]School of Medicine, University of Queensland, Brisbane

Background: The use of kidney donors with active hepatitis B (HBV) infection into uninfected recipients is typically contraindicated except in exceptional circumstances. HBV serology is mandatory in all donors however this may not detect HBV surface antigen mutant infections.

Case Report: We describe the case of a HBV negative, 30-year-old male who received a deceased donor kidney transplant from a 64-year-old donor with occult HBV surface antigen mutant infection (negative HBV sAg, low level HBV viraemia (62 IU/mL)). The recipient was highly sensitised (cPRA 99%), and had been on the deceased donor list for several years with no offers. The transplant had significant immunological benefits with 2 HLA mismatches and no donor specific antibodies. He had previously been immunized against HBV but at time of transplantation did not have detectable HBV sAb.

A 2-into-1 kidney transplant was performed with informed consent including the very high likelihood of HBV transmission. He received standard induction and maintenance immune suppression. He was commenced on entecavir immediately following his transplant and HBV immunoglobulin (400 IU) was administered at time of transplantation, daily for 1 week, then weekly for 4 weeks. HBV DNA PCR was routinely tested and remains negative at 6 months. Allograft function remains stable (serum creatinine 197 μmol/L).

Conclusion: We report the case of a kidney transplant from a donor with an active HBV surface antigen mutant infection into an uninfected recipient. This case highlights the importance of prospective HBV NAT testing for deceased organ donors – even if HBV sAg is negative.

Abstract No. 104

A RARE PRESENTATION OF CMV DISEASE IN A KIDNEY TRANSPLANT RECIPIENT

YAXLEY Julian[1], TITUS Thomas[2]
[1]Gold Coast University Hospital, [2]Department of Renal Medicine, Gold Coast University Hospital

Background: Cytomegalovirus (CMV) is a widespread latent virus which reactivates commonly in the immunosuppressed kidney transplant population. CMV infection can manifest with systemic symptoms such as fever and arthralgia, or with tissue-invasive disease such as enteritis, nephritis, or pneumonitis. In such instances acute CMV infection may lead to acute allograft rejection or death.

Case: A 67-year-old man presented with acute abdominal pain. He had undergone a living non-related donor kidney transplantation 12 years earlier in Pakistan, for end-stage renal failure secondary to IgA nephropathy. Both the donor and recipient were CMV-seronegative. The post-operative period was complicated by severe CMV disease with pneumonitis, hepatitis and colitis, requiring admission to the intensive care unit and treatment with intravenous ganciclovir. Following recovery his subsequent long-term graft function was good. In the current presentation, non-contrast computed tomography showed a thickened ileocecal junction with surrounding gas locules and pericolic stranding consistent with a perforated viscus. Emergency right hemi-colectomy was performed, complicated by anastomotic leak and intra-abdominal sepsis requiring reoperation. Bleeding persisted from the anastomosis despite colonoscopic intervention. Histopathology tissue stains were positive for CMV and the serum CMV titre was 2.43×10^3 IU/ml. The patient's condition stabilised rapidly after commencing treatment with intravenous ganciclovir. He eventually made a successful recovery with intact graft function after completing a long course of oral valganciclovir.

Discussion: Tissue-invasive CMV disease is uncommon in kidney transplantation when both donor and recipient are seronegative.[1] The most frequent manifestation of CMV disease is gastrointestinal involvement.[2] Viral reactivation typically occurs less than 6 months after surgery and late CMV disease more than 12 months after solid organ transplant surgery is unusual.[3] Furthermore, bowel perforation as a feature of CMV enterocolitis is rarely encountered. Intravenous ganciclovir is the antiviral agent-of-choice for severe cases. This report highlights are rare and challenging presentation of CMV disease in a kidney transplant patient.

References:

1. Hartmann A, Sagedal S, Hielmesaeth J. The natural course of cytomegalovirus infection and disease in renal transplant recipients. Transplantation 2006; 82(2): 15.
2. Arthurs S, Eid A, Pedersen R, Kremers W, Cosio F, Patel R, et al. Delayed-onset primary cytomegalovirus disease and the risk of allograft failure and mortality after kidney transplantation. Clin Infect Dis 2008; 46(6): 840.
3. Slifkin M, Tempesti P, Poutsiaka D, Snydman D. Late and atypical cytomegalovirus disease in solid-organ transplant recipients. Clin Infect Dis 2001; 33(7): 62-68.

Abstract No. 105

SAFETY, TOLERABILITY AND EFFECT ON HIGH-SENSITIVITY CRP OF LOW-DOSE COLCHICINE IN KIDNEY TRANSPLANT RECIPIENTS

SORARU Jacqueline[1], WONG Germaine[2], THOMPSON Peter[3], JOYCE David[3], TEIXEIRA-PINTO Armando[4], LIM Wai H[1]

[1]Department of Renal Medicine, Sir Charles Gairdner Hospital, Perth, [2]National Pancreas Transplant Unit, Westmead Hospital, Sydney, [3]University of Western Australia, Perth, [4]University of Sydney

Aims: In view of the high prevalence of cardiovascular disease (CVD) in kidney transplant recipients, and the potential for colchicine to improve CVD outcomes, we determined the safety, tolerability and effect on high-sensitivity C-reactive protein (hsCRP) of low-dose colchicine in stable kidney transplant recipients.

Methods: This pilot study emulated a phase I dose escalation strategy to determine the effect of low-dose colchicine (0.5mg 3 days/week for 2-weeks, escalating to 0.5mg/daily for 2-weeks) on the tolerability and effects on hsCRP and intra-individual variability of tacrolimus level in stable kidney transplant recipients >3 months post-transplant maintained on tacrolimus-based immunosuppression and with eGFR >20ml/min/1.73m2. An interim safety analysis of the first 10 participants is presented.

Results: Of the 10 participants, the mean (SD) age was 60.7 (6.6) years, 8 (80%) were maintained on statin and 6 (60%) had diabetes. Mean (SD) hsCRP reduced from 1.91 (1.69) mg/L at baseline to 1.17 (1.03) mg/L after the colchicine protocol (i.e. mean 29% [95%CI -9%, 66%] absolute reduction; paired t-test p=0.25). The haemoglobin, white cell count and creatinine kinase levels remained unchanged in the 4-week period. The tacrolimus trough displayed a low intra-patient variability over the 4-week period with a median (IQR) coefficient of variation of only 11% (7-16%). Two participants experienced loose bowel actions but continued the colchicine until study completion.

Conclusion: Low dose colchicine was safe and well tolerated in this small cohort of stable kidney transplant recipients, and appeared to have little impact on the trough tacrolimus levels.

Abstract No. 106

CANCER TRANSMISSION FROM DECEASED ORGAN DONORS WITH PRIOR CANCERS

PHILLIPS Jessica[1], WONG Germaine[2], AU Eric[2], MCDONALD Stephen[3], CHAPMAN Jeremy[2], OPDAM Helen[4], MCDONALD Mark[5], PILMORE Helen[6], KANELLIS John[7], LIM Wai[8]

[1]Renal Medicine, Sir Charles Gairdner Hospital, Perth, [2]Westmead Hospital, Sydney, [3]Royal Adelaide Hospital, [4]Austin Hospital, Melbourne, [5]Organ Transplant Australia, [6]Auckland City Hospital, [7]Monash Medical Centre, Melbourne, [8]Sir Charles Gairdner Hospital, Perth

Aims: We aimed to describe the characteristics of deceased organ donors with prior cancers and the outcomes of kidney transplant recipients who received kidneys from donors with prior cancers or were reported to have lost their grafts/died from donor cancers.

Methods: The study included all actual donors with prior cancers recorded in the ANZOD registry between 1989-2017. Donor cancer characteristics and rate of donor-transmitted cancer are described. The characteristics of recipients of kidneys from donors without prior cancers but were recorded to have lost their grafts/died from donor cancer are also described.

Results: Of 259 actual donors with a prior cancer history, kidneys from 197(76%) donors were transplanted into 366 recipients. The most common cancer type was non-melanoma skin cancers(30%), followed by gynaecological cancers(16%), brain cancers(9%), melanoma, breast and prostate cancers(all 7%). The median(IQR) time from cancer diagnosis to donation was 6.5(0.8-14.8)years. Of the 366 recipients who have received kidneys from donors with cancers, 5(1.4%) were recorded as experiencing graft loss from donor cancer, with 1 developed cancer in the allograft 255-days post-transplant(Table). There were 12 recipients from 10 donors without prior cancer history recorded as experiencing graft loss from donor cancer, with 2 cases of melanoma (same donor) detected in the allografts at 12-months post-transplant. Data relating to the ascertainment of donor-transmitted cancer were not collected.

Conclusion: There appears to be a low-rate of donor cancer transmission but the establishment of structured reporting to collect more detailed/accurate records of donor cancer history and characteristics of donor-transmitted cancers is required.

CASES OF PRIOR DONOR CANCER (YEAR)	TYPE OF DONOR CANCER	DONOR AGE	CAUSE OF DONOR DEATH	RECIPIENT CANCER TYPE/SITE (DAYS FROM TRANSPLANT)	TIME TO ALLOGRAFT FAILURE FROM TRANSPLANT (DAYS)
1 (1998) [#]	NHL	72	Intracranial haemorrhage	None	11
2 (1998) [#]	NHL	72	Intracranial haemorrhage	None	10
3 (2001)	Melanoma	47	Intracranial haemorrhage	None	155
4 (2003)	Renal cell cancer (excised)	56	Intracranial haemorrhage	Adenocarcinoma – allograft (255)	270
5 (2008)	NHL	68	Intracranial haemorrhage	None	3

#Same donor. NHL – Non-Hodgkin lymphoma.

Abstract No. 107

POST TRANSPLANT LYMPHOPROLIFERATIVE DISEASE (PTLD) PRESENTING AS ENTEROCOLITIS IN A RENAL TRANSPLANT PATIENT

HEPBURN Kirsten[1], JAHAN Sadia[1], MARSH Bree[1], FRANCIS Leo[2], JOHN George[1]

[1]Department of Nephrology, Royal Brisbane Hospital, [2]Department of Anatomical Pathology, Royal Brisbane Hospital

Introduction: PTLD is a rare, potentially fatal, complication of renal transplantation, with variable presentation depending on organ involvement. We present a case of PTLD presenting as acute enterocolitis.

Case: The patient, a 29-year-old male, was 11 months post renal transplant for reflux nephropathy. He was donor EBV positive to recipient negative, 6/6 HLA mismatch, with early T-cell mediated rejection requiring IV methylprednisolone. Maintenance immunosuppression was tacrolimus, mycophenolate and prednisone. He presented with abdominal pain, decreased appetite, and diarrhoea without fevers. Investigations revealed an acute kidney injury with creatinine 320µmol/L and CRP 143mg/L. Non-contrast CT abdomen demonstrated terminal ileitis/colitis with backwash ileitis, and para-aortic and mesenteric lymphadenopathy (Figure 1a). Surgical impression was enterocolitis rather than appendicitis. He was treated with 5-days of piperacillin-tazobactam. CMV and faecal culture returned negative, however EBV DNA was detected (1.7x10^3 copies/ml). Immunosuppression was reduced. He was readmitted 6-days later with ongoing pain and culture negative diarrhoea. Colonoscopy revealed deeply ulcerated caecum and distorted ileocaecal valve (Figure 1b). Biopsy demonstrated an EBV-driven lymphoproliferative process composed of large B-lymphocytes, with strong CD20 immunohistochemical and EBV-EBER reactivity (Figure 1c). PET revealed intense FDG activity in the distal ileum, proximal right colon, lymph nodes (above and below diaphragm), liver and bone marrow. Bone marrow biopsy is awaited and he is planned for R-CHOP. Adoptive T-cell therapy may be considered.

Conclusions: This case highlights the importance of considering lymphoproliferative disease in seemingly infective presentations, especially in patients with EBV donor recipient mismatch, and that EBV prophylaxis does not prevent PTLD.

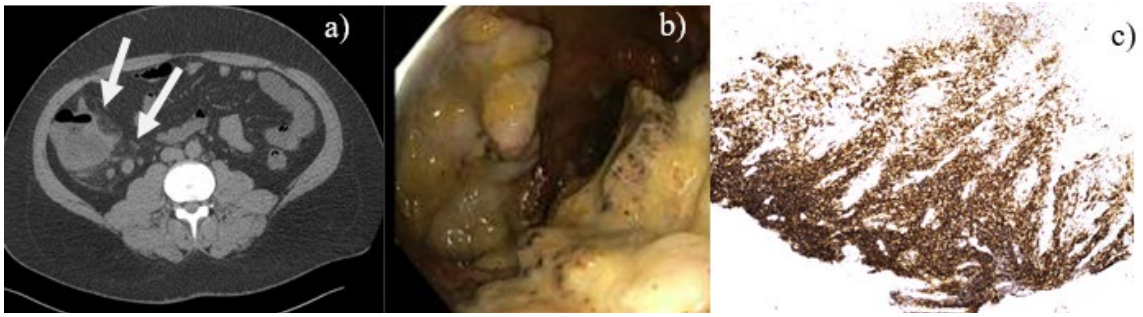


Figure 1 a) CT abdomen demonstrating colitis/ileitis, mesenteric stranding and lymphadenopathy, b) Colonoscopy image of the caecum c) Immunohistochemistry demonstrating CD20 sheet like reactivity.

Abstract No. 108

VALIDATION OF THE METROTICKET 2.0 MODEL FOR 5-YEAR HCC SPECIFIC SURVIVAL IN AN AUSTRALIAN LIVER TRANSPLANT COHORT

STOKLOSA Ted[1], SANTHAKUMAR Positha[1], FERNANDES Brian[1], PERERA Nadia[1], HU Xinxin[1], TATIANA Tsoutsman[1], LIU Ken[1,2,3], MCCAUGHAN Geoffrey[1,2,3], STRASSER Simone[1,2], MAJUMDAR Avik[1,2]

[1]Department of Gastroenterology, Royal Prince Alfred Hospital, Sydney, [2]University of Sydney, [3]Centenary Institute of Cancer Medicine and Cell Biology, Sydney

Background: Predicting the prognosis of patients with hepatocellular carcinoma (HCC) who undergo liver transplant (LT) is complex. The Metroticket 2.0 model (1) has recently been described to predict HCC-specific survival. We aimed to evaluate this model in an Australian context.

Methods: All adult patients who underwent LT for HCC between 01/01/1998 and 15/03/2014 at our centre were included (N=182). Milan and Metroticket 2.0 criteria were compared to predict post LT five-year HCC-specific survival (5Y-HSS). Data were collected at waitlisting and at the time of last imaging prior to LT.

Results: Hepatitis C was the most common aetiology (55%) and 128 (70%) had prior locoregional therapy. Median post LT follow-up was 7.2 [IQR 5.0- 11.1] years. 59 (32%) patients died with 14 due to HCC recurrence. At waitlisting, 180/182 (99%) were within Milan, 179/182 (98%) within Metroticket 2.0. 5-year overall survival was 77%. At waitlisting, Metroticket 2.0 predicted median 5YHSS was 95% (IQR 93%-98%), similar to the observed 5Y-HSS of 93% (Figure 1a). Prior to LT (median time last imaging and LT was 1 month [1-2]), there was a difference in 5Y-HSS for patients outside Metroticket 2.0, unlike Milan criteria (Figure 1b&c). The sensitivity and specificity for 5Y-HSS were respectively: Metroticket 2.0; 98% and 17%, Milan; 96% and 17%. The positive and negative predictive values were respectively: Metroticket 2.0; 94% and 33%, Milan; 94% and 22%.

Conclusions: Metroticket 2.0 predicted 5Y-HSS is similar to observed outcomes. The Metroticket 2.0 model has better discriminatory value for 5Y-HSS prior to LT compared with Milan.

Mazzaferro , et al. Gastroenterology. 2018;154(1):128-39.

Abstract No. 108ctd

Figure 1a. 5-year post LT HCC specific survival from waitlisting

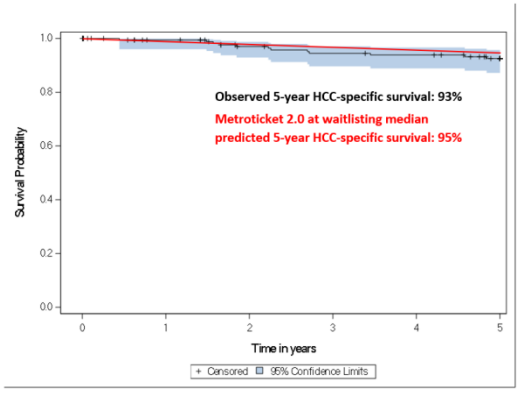


Figure 1b. 5-year post LT HCC specific survival from last imaging prior to LT by Metroticket 2.0 criteria

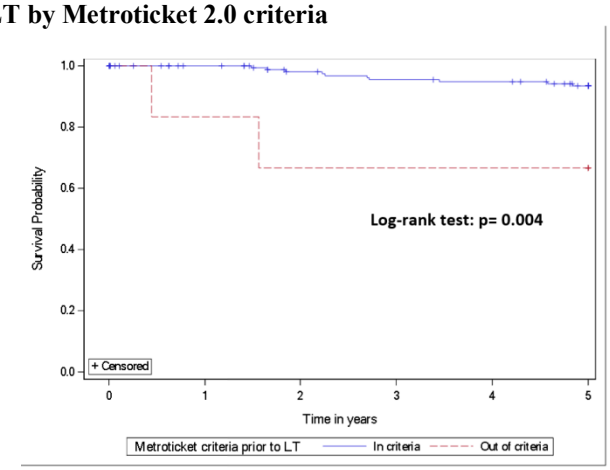
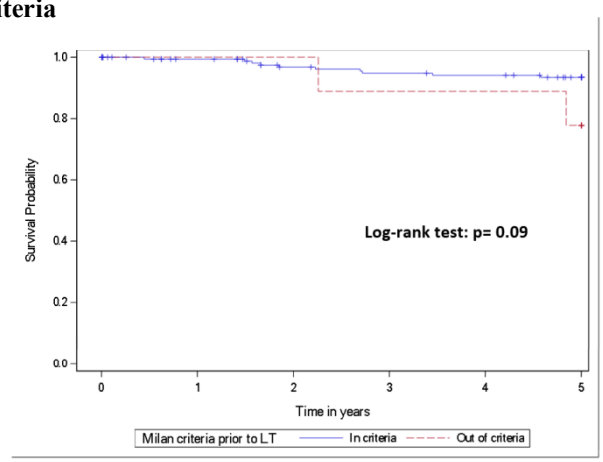


Figure 1c. 5-year post LT HCC specific survival from last imaging prior to LT by Milan criteria



Abstract No. 109

METASTATIC ROUND CELL SARCOMA IN A RENAL TRANSPLANT RECIPIENT

SHETTIGAR Reshma[1], PUTT Tracey[2], SCHOLLUM John[2], WALKER Robert[2]

[1]Department of Renal Medicine, Dunedin Public Hospital, [2]Department of Nephrology, Dunedin Public Hospital

57-year-old male, end stage renal failure due to polycystic kidneys, underwent deceased donor renal transplantation. Patient received standard induction immunosuppression with basiliximab and maintainence immunosuppression of mycophenolate, tacrolimus and prednisone. 6 months post transplant patient noticed a lump about 4 cm in diameter on the medial side of the lower end of his thigh. Ultrasound showed that this mass was arising from the Sartorius muscle. He further underwent a biopsy of this mass, which showed un-differentiated round cell sarcoma. Staging CT scan did not show any evidence of metastatic disease. It was decided patient would have radiation therapy followed by surgical removal of sarcoma. A week prior to surgery, he presented with severe back pain and epistaxis. Blood test revealed thrombocytopenia with platelet count of 9* 10^9/litre . CT scan showed several metastatic lesions in lumbar spine and femur and lungs. Patient was deemed to unwell to tolerate chemotherapy. Patient was managed with platelet transfusion and analgesia with a plan to offer radiation therapy for pain. Patient passed away 2 weeks later.

Discussion: Sarcomas are rare and heterogeneous group of aggressive malignant tumors of mesenchymal origin that make up less than 1% of adult malignancies. Unlike Kaposis sarcoma, which is associated with immunosuppression, round cell sarcoma has never been reported in renal transplant recipient. Role of immunosuppression contributing to the genesis and progression of the tumor is unknown. Despite treatment with aggressive chemotherapy and radiation therapy, prognosis with metastatic round cell sarcoma is extremely poor.

Abstract No. 110

RAPIDLY PROGRESSIVE METASTATIC ADENOCARCINOMA OF UNKNOWN PRIMARY 2 MONTHS FOLLOWING DECEASED DONOR RENAL TRANSPLANTATION, RECIPIENT OR DONOR DERIVED – A CASE REPORT

MCMICHAEL Lachlan[1,2], CARROLL Robert[1]

[1]Central Northern Adelaide Renal and Transplantation Service, Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [2]Discipline of Medicine, University of Adelaide

Background: Malignancy is the most common cause of death with a functioning graft but is rarely encountered within the first-year post-transplantation. Differentiating donor versus recipient derived malignancies remains a clinical challenge with significant clinical implications if incorrectly identified.

Case Report: 72-year-old man with a background history of end-stage renal failure secondary to diabetic kidney disease, ischaemic heart disease, hypertension and type 2 diabetes mellitus. Underwent deceased donor renal transplantation from a 66-year-old female donor with a background history of chronic pancreatitis, hypertension and diabetes mellitus. Day 54 post-transplant admitted for further investigation of deranged liver function tests. Initial investigations included upper abdominal ultrasonography and magnetic resonance cholangiopancreatography identifying diffuse heterogenous change of the liver parenchyma. Liver biopsy was performed identifying invasive adenocarcinoma. Immunohistochemistry consistent with an upper gastrointestinal tract adenocarcinoma with strong staining for CK19 and CK7, weak CK20 staining and negative staining for Hepar-1, TTF-1 and CDX2. Given the rapidity of onset and donor history of chronic pancreatitis further evaluation for a donor derived malignancy was performed including tissue karyotyping, fluorescence in situ hybridization (FISH), CA 19-9 tumour markers on stored donor/recipient sera and formal DNA sequencing of the malignant tissue. Testing was consistent with a recipient-derived malignancy.

Conclusions: Development of malignancy within the acute period following transplantation is a rarely described entity. Appropriate investigation to identify whether the malignancy is recipient or donor derived remains an important process to ensure appropriate management of the recipient affected and recipients of other organs from the same donor.

Abstract No. 111

COLONIC MUCOSA-ASSOCIATED LYMPHOID TISSUE LYMPHOMA IN A KIDNEY TRANSPLANT RECIPIENT

MARTIN Kylie[1], TAYLOR Andrew[2], TAM Constantine[3], HILL Prue[4], MACHET David[5], GOODMAN David[1]

[1]Department of Nephrology, St Vincent's Hospital, Melbourne, [2]Department of Gastroenterology, St Vincent's Hospital, Melbourne, [3]Department of Haematology, St Vincent's Hospital, Melbourne, [4]Department of Anatomical Pathology, St Vincent's Hospital, Melbourne, [5]Anatpath

Case: We describe a case of a 62-year-old male with colonic extra-nodal marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma) 14 years after kidney transplantation. Colonoscopy following a positive faecal occult blood test identified MALT lymphoma in 1 of 3 sessile sigmoid polyps. A clone on bone marrow biopsy had the same peripheral blood flow cytometric characteristics as seen on peripheral blood flow cytometry and immunoprofile of the colonic MALT lymphoma. Positron Emission Tomography scan was negative for other sites of disease. Gastric biopsy showed mild chronic gastritis with focal intestinal metaplasia with no evidence of *Helicobacter pylori* (*H. pylori*). However his *H.pylori* IgG was positive. He received *H.pylori* eradication treatment and is being managed expectantly. Immunosuppression was unchanged with prednisolone, mycophenolate mofetil and cyclosporine A with stable renal allograft function.

Discussion: MALT lymphoma is rare in the solid-organ transplant recipient. MALT lymphoma It is most commonly found in the gastrointestinal tract. Colonic involvement is found in only 2.5% of cases. Although complete remission occurs in 70-80% of localised gastric MALT lymphoma cases after *H.pylori* eradication, there is no standardised therapy for extra-gastric sites. This case highlights the importance of histopathological diagnosis of colonic lesions in immunosuppressed solid-organ transplant recipients and consideration of conservative management in extra-gastric MALT lymphoma as MALT lymphoma in the immunosuppressed solid organ-transplant recipient does not appear to be clinically aggressive and behaves like MALT lymphoma in the immunocompetent patient.

Abstract No. 112

YOUNG WOMAN WITH TYPE 1 DIABETES AND SEVERE SUB-CUTANEOUS SKIN REACTION TO INSULIN, BECOMES INSULIN INDEPENDENT AFTER ISLET TRANSPLANT

RADFORD Toni[1,2], DROGEMULLER Christopher[1,2,3], LOUDOVARIS Thomas[4,2], KAY Thomas[5,2], O'CONNELL Philip[6,2], HAWTHORNE Wayne[7,2], TORPY David[1,2], COATES TOBY[1,2]

[1]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [2]Australian Islet Transplantation Consortium, Australian Islet Transplantation Consortium, [3]Centre for Transplant and Renal Research, University of Adelaide, [4]Islet Transplantation Facility, St Vincent's Institute, Melbourne, [5]Islet Transplantation Facility, Tom Mandel Islet Program, Melbourne, [6]Islet Transplantation Facility, Westmead Hospital, Sydney, [7]Islet Transplantation Facility, Westmead Millennium Institute, Westmead Hospital, Sydney

While allergies to insulin are less common since the introduction of human insulins, they can still occur. In this case study I report a patient who presented with a severe insulin allergy which was treated with an islet transplant.

TD is a 37 year old woman diagnosed with type 1 diabetes in 2002. Initially using multiple injections, she changed to an insulin pump in 2008. In 2011, she noticed small lumps around the cannula injection sites. Over the following 5 years, she trialled re-siting the cannula 2 to 3 times a week with various insulins, with no benefit. A trial of subcutaneous injections resulted in immediate formation of lumps that took 2-6 weeks to resolve. The patient felt well with no other systemic effects.

A skin biopsy in 2016 diagnosing mixed lobular panniculitis induced by possible phagocytosis of insulin with clearly defined, deeply indurated nodules with minimal surface erythema. No microorganisms or foreign bodies were detected in the tissue.

Hydrocortisone was administered simultaneously with insulin using an insulin pump. Antiprotease inhibitor Nafamostat was trialled to reduce insulin resistance and for its anti-inflammatory effect. Topical steroid cream was applied and systemic glucocorticoids were commenced. An insulin desensitisation protocol was completed. Oral Azathioprine 50mg daily was also trialled. These all had no effect.

The condition worsened and doses of insulin up to 75u was required to control blood glucose levels. The HbA1c was >10%. It was suspected that insulin was not being absorbed through the inflamed subcutaneous tissue.

A central catheter enabled intravenous insulin via a pump to be administered after 2 admission to hospital with DKA. The risks of using intravenous insulin prompted her endocrinologist to refer her to the islet transplant team.

TD was listed in early 2017. Her first islet infusion yielded 9218 islet equivalents per kg (IEq/Kg) of body weight. Her insulin requirement dropped from 75u to 16u within 3 months. Sub-cutaneous insulin was re-trialled after 4 weeks due to her highly immunosuppressed state with no evidence of nodules recurring. A second infusion 5 months after her first, provided another 6179 IEq/Kg.

TD became insulin independent on day 56 post second infusion. Her HbA1c is stable at 5.5%, with C-peptide levels >300picomols/L. Her immunosuppressive regimen is Tacrolimus 5mg BD and Myfortic 360 mg BD.

This presents an interesting case of a patient allergic to exogenous insulin but not endogenous insulin produced by an allo-islet transplant.

More broadly this study presents an example where the usual criteria of hypoglycaemia unawareness for islet transplantation, has been extended.

Abstract No. 113

THE USE OF FREESTYLE LIBRE FLASH GLUCOSE MONITORING SYSTEM TO MONITOR BLOOD GLUCOSE LEVELS IN LARGE ANIMAL PORCINE MODEL

PENKO Daniella[1,2], NITSCHKE Jodie[1,3], JOHNSTON Julie[1,3], MATHEWS Loren[4], JOHNS Daniel[4], MUIRHEAD Robb[4], PULLAN Caitlin[4], DROGEMULLER Christopher[1,3], TORPY David[5], KUCHEL Timothy[4], COATES Patrick[1,3]

[1]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [2]The Discipline of Medicine, University of Adelaide, [3]Department of Medicine, University of Adelaide, [4]Preclinical Imaging and Research Laboratories, South Australian Health and Medical Research Institute, [5]3Endocrine and Metabolic Unit, Royal Adelaide Hospital

Introduction: Abbott Laboratories Flash Glucose Monitoring System, FreeStyle Libre, is a recent clinical innovation to monitor blood glucose levels (BGL) of Diabetics. The system involves self-application of a sensor with attached probe onto upper arm measuring interstitial glucose levels. The system continuously monitors glucose-levels, identifying fluctuations improving regulation. The sensor can be worn for up to 2 weeks, reducing need for repetitive self-monitoring using blood glucose strips. However, accuracy discrepancies were noted between measuring interstitial versus blood glucose-levels. This study assessed the device in a large diabetic porcine model, to investigate the ease of using the FreeStyle Libre monitoring glucose levels and its accuracy

.**Method:** Landrace pigs were rendered diabetic by removal of the pancreas (N=3). The sensor was inserted into the skin adjacent to dorsal neck vertebrae. Intra-venous glucose tolerance test (IVGTT, 0.5g glucose/kg) was performed over 120 minutes, recording BGL readings via sensor and self-monitoring using a glucose meter. Glucose-levels were monitored twice daily using the scanner up to 75 days post-pancretectomy.

Results: The device successfully continuously monitored porcine glucose-levels over time, enabling management of induced diabetes via exogenous insulin. A few failures lead to momentary read errors, requiring replacement of the sensor. However, this may be due to the nature of working with large animals and site of application. Scanner IVGTT data followed a similar trend to traditional self-monitoring using test strips. A lag was observed in scanner glucose-levels versus test strip, as reported elsewhere.

Conclusion: The device was well-tolerated, demonstrating a novel application and off-label use of this system in a large animal setting.

Abstract No. 114

REVISITING PANCREAS TRANSPLANTATION IN SOUTH AUSTRALIA

BHATTACHARJYA Shantanu, OLAKKENGIL Santosh, RUSSELL Christine, COATES Patrick Toby, RUSS Graeme

Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital

Aims: The first Simultaneous Pancreas and Kidney Transplant in South Australia were performed in 1990. There was immediate pancreatic graft function, however kidney failed as a complication of recipient iliac artery thrombosis. The recipient underwent further surgeries, but died due to cardiac issues. The high complexity of the surgery at this early stage meant that the unit made no further attempts. In 2016 South Australia appointed 2 new Consultant Transplant Surgeons; with kidney and pancreas transplant experience.

In 2018, it became apparent that some potential SA recipients were declining SPK transplants due to the need for travel interstate. Also 53 potential pancreas donors in South Australia did not proceed to donation in the preceding 3 years for logistic reasons.

Methods: The program in South Australia was commenced in August 2018. Four pancreas transplants have been performed; Three have been simultaneous pancreas and kidney transplants and one a Pancreas alone transplant The 3 recipients for SPK were all female with median age of 44 (range 43- 55). The recipient of the pancreas alone transplant was a male of 41 years. The median donor age was 39 (range 25 – 53 years). Mean cold ischemic time to pancreas reperfusion was 7hrs 54min (range 6.06 to 10.13).

Results: There has been 100% patient and graft survival and all four recipients are insulin independent.

Conclusion: Our early results are consistent with Registry data in Australasia where the reported 1 year patient survival is 98%; Pancreas graft survival is 93% and kidney graft survival is 97%.

Abstract No. 115

NORMOTHERMIC MACHINE PERFUSION OF DCD LIVERS – A TOOL THAT COULD HELP EXPAND THE DONOR POOL

REILING Janske[1,2,3,4], HODGKINSON Peter[1,5], COCO Tina[6], BUTLER Nicholas[1,5], FACWETT Jonathan[1,5]

[1]Faculty of Medicine, University of Queensland, Brisbane, [2]Gallipoli Medical Research Institute, Greenslopes private hospital, Brisbane, [3]Princess Alexandra Research Foundation, Princess Alexandra Hospital, Brisbane, [4]Queensland Liver Transplant Servivce, Princess Alexandra Hospital, Brisbane, [5]Queensland Liver Transplant Service, Princess Alexandra Hospital, Brisbane, [6]DonateLife Queensland

Introduction: Concerns over unacceptably poor outcomes relating to ischaemic injury currently limit the utilisation of donation after circulatory death (DCD) livers in Australasia. Normothermic machine perfusion (NMP) might enable safer use of DCD livers because it allows for *ex-vivo* assessment of graft function prior to implantation. We describe our early experience following the first three NMP preserved liver transplants in Australasia using a "back-to-base" approach.

Methods: From July 2018 three DCD livers were procured using standard cold preservation techniques prior to repatriation and NMP using the OrganOx *Metra* device. Each graft had one or more elements outside of established criteria (age >50; BMI >30, non-metropolitan). The functional warm ischaemic times were 12,16,27 and cold ischaemic times were 5h, 5h15min, and 7h4min respectively. NMP duration was >6 (range 9 – 17.5) hours. Recipient MELD score ranged from 18-31.

Results: Each liver met pre-established criteria for implantation during NMP: lactate clearance within two hours, glucose utilisation within 6 hours, pH maintenance >7.2, satisfactory haemodynamic flows (portal vein 0.9 – 1.2 L/min, hepatic artery 0.5 – 0.6 L/min). Immediate graft function was unanimously observed following implantation.

Conclusion: The OrganOx *Metra* using a "back-to-base" approach has enabled the safe use of three DCD grafts outside of established Australasian criteria. Whilst early graft dysfunction can be successfully predicted, the impact of NMP on ischaemic biliopathy rates is yet to be established. Nevertheless, this technology undoubtedly improves transplant logistics and should enable a safer expansion of the DCD program across Australasia.

Abstract No. 116

EMPAGLIFLOZIN AS A SODIUM-HYDROGEN EXCHANGE INHIBITOR (NHEI) FOR DONOR HEART PRESERVATION

VILLANUEVA Jeanette[1], GAO Ling[1], SCHEUER Sarah[1], DOYLE Aoife[1], BOKKERS Kim[1], HICKS Mark[2,1], MACDONALD Peter[3,1]

[1]Transplantation Laboratory, Victor Chang Cardiac Research Institute, Sydney, [2]Department of Clinical Pharmacology, St Vincent's Hospital, Sydney, [3]Heart & Lung Transplant Unit, St Vincent's Hospital, Sydney

Background: Many donor hearts are unsuitable for transplantation after long periods of cold storage or donation after circulatory death. Despite evidence supporting improved donor heart recovery when NHEI cariporide/zoniporide supplement heart preservation solutions containing erythropoietin and glyceryl trinitrate (E+G), NHEI are not clinically used due to neurotoxicity observed in cariporide clinical trials. A safe alternative to NHEI that promises to provide similar cardioprotection is the sodium-glucose cotransporter inhibitor (SGLT2i) empagliflozin. Empagliflozin is the first anti-diabetic drug to yield significant positive cardiovascular outcomes.

Aims: To assess the ability of the SGLT2i Empagliflozin to protect the heart during prolonged cold donor heart preservation.

Methods: Wistar rat (320-420g; n=6-7) hearts were perfused ex-vivo and baseline haemodynamic measurements acquired. Hearts were arrested and stored in Celsior±1μM Empa (6h, 4°C). Triple supplementation with E+G+zoniporide was used as a positive control. Hearts were then reperfused (37 °C, Langendorff 15min, working 30min). Post-reperfusion aortic flow (AF), coronary flow (CF), cardiac output (CO), and pulse pressure (PP) was expressed as a percentage of baseline measurements (mean±SEM).

Results: 1μM Empa supplementation showed a trend towards increased CO (AF+CF, Figure 1) compared to unsupplemented hearts. Whereas 4/7 unsupplemented hearts showed no AF recovery, only 2/7 Empa-hearts failed to have AF recovery. Compared to Celsior alone, functional recovery after triple supplementation with E+G+zoniporide showed significant improvement ($p<0.05$), consistent with our previous studies.

Conclusion: Hearts supplemented with 1μM Empa during 6h cold storage showed non-significant improvements to functional cardiac recovery however further dose optimization studies are required.

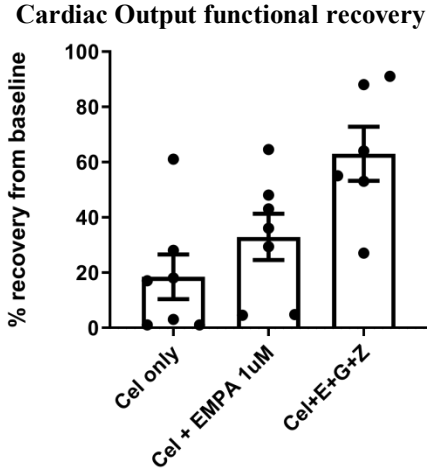


Figure 4 Cardiac output functional recovery after 6h cold storage and 45 minutes reperfusion.

Abstract No. 117

ASSESSMENT OF 3D-BIOPRINTED HUMAN REGULATORY T-CELLS FOR CO-PRINTING WITH HUMAN PANCREATIC ISLETS

KIM Juewan[1], HOPE Christopher M[2], PERKINS Griffith B[1], YUE Zhilian[3], LIU Xiao[3], DROGEMULLER Christopher J[4,5], CARROLL Robert P[4,5], BARRY Simon C[2,6], WALLACE Gordon G[3], COATES P Toby[4,5]

[1]Department of Molecular and Cellular Biology, University of Adelaide, [2]Discipline of Paediatrics, University of Adelaide, [3]Intelligent Polymer Research Institute, University of Wollongong, [4]Central Northern Adelaide Renal and Transplantation Service, Royal Adelaide Hospital, [5]Discipline of Medicine, University of Adelaide, [6]Molecular Immunology Group, University of Adelaide

Introduction: 3D-bioprinting facilitates the fabrication of complex 3D-architectures. 3D-bioprinting of regulatory T-cells (Tregs) with islets may overcome the immunosuppressive shortcomings inherent in islet transplantation. This project aims to characterize 3D-bioprinted human Tregs using alginate-GelMA bioink supplemented with IL-2 and CCL1.

Method: Natural Tregs were FACS sorted from peripheral blood, whereas induced Tregs were induced from naïve $CD4^+$ T-cells. Tregs were suspended in media ('non-printed') or printed in a photo- and chemically-crosslinked alginate-GelMA bioink, with and without IL-2 and CCL1. Tregs were analyzed by flow cytometry for viability (propidium iodide), phenotype (CD25 and FOXP3) and functionality (TGF-β, CD69, CD39 and CTLA-4). CD154 suppression assay evaluated Treg function. Trans-well migration assays were performed to evaluate the bioprinted Treg migration and recruitment capability of CCL1-supplemented bioink.

Results: In the presence of IL-2, 3D-bioprinted Tregs retained viability above 80% with no significant decreases compared to non-printed cells, up to 3 days post-bioprinting. However, in the absence of IL-2, 3D-bioprinted Treg viability significantly decreased below 50%, by day 3 ($p<0.0001$). 3D-bioprinting maintained expression of Treg phenotypic and functionality markers and suppressive capacity. Furthermore, 3D-bioprinting was shown to halt migration of Tregs. CCL1-supplemented bioink demonstrated recruitment of Tregs and enhancement of Treg function.

Conclusion: 3D-bioprinting had a minimal impact on viability, phenotype and function of Tregs. Secondly, bioink supplementation with IL-2 displayed a positive impact on 3D-bioprinted Tregs. Furthermore, we demonstrated printing prevents Treg migration from the 3D-bioprinted structures. Lastly, bioink supplementation with CCL1 enabled Treg recruitment and enhanced Treg function.

Abstract No. 118

IL-2 ALONE DOES NOT MAINTAIN ACTIVATED HUMAN CD4+CD25+CD127LOCD45RA FOXP3HI TREG POPULATION IN CULTURE

VERMA Niruapama D[1,2], LAM Andrew[1,2], CHIU Chris[1,2], ROBINSON Catherine M[1], TRAN Giang T[1,2], HODGKINSON Suzanne J[1,2,3], HALL Bruce M[1,2,4]

[1]Immune Tolerance Laboratory, Ingham Institute for Applied Medical Research, [2]Department of Medicine, University of New South Wales, Sydney, [3]Department of Neurology, Liverpool Hospital, [4]Department of Nephrology, Liverpool Hospital

Background: Human T regulatory cells express CD4, CD25, Foxp3 but are $CD127^{lo}$. Naïve Treg express CD45RA (Population I) and after activation have less CD45RA. Some $CD45RA^{lo}$ Treg have high expression of Foxp3 and CD25 (PopII). The majority of $CD45RA^{lo}$ cells have normal Foxp3 and CD25 expression (PopIII). Activated Treg express chemokine receptors of activated T cells, including CXCR3 (Th1) and CCR6 (Th17) indicating they can migrate to sites of inflammation. We examined if culture of Treg with rIL-2 and alloantigen increased the number of activated Treg.

Methods: Healthy human blood $CD4^{+}CD127^{lo}CD25^{+}$Regulatory T cell were isolated by kits that either required PBMC isolation first or direct from whole blood or cell sorting. These tTreg were cultured with rIL-2 (200Units/ml) and irradiated allogeneic stimulator cells. Multicolour flow cytometry assessed changes in Treg populations, especially PopII.

Results: tTreg cultured alone had reduced PopII (1.3% vs 8.6%). Culture with allostimulators alone preserved the PopII (12% vs 8.6%). IL-2 alone increased PopII in 5 of 8 of experiments similar to the culture with allostimulators and rIL-2 (6/8 experiments). CXCR3 expression in PopII was similar in fresh Treg to those cultured with IL-2 alone or IL-2 with allostimulators suggesting IL-2 alone does not support activated Treg expressing CXCR3.

Conclusions: These results suggest that activated Treg in PopII, need allostimulation or IL-2. Human Treg activation, similar to our rat studies, may be a 2-step process that requires cytokines other than IL-2. Understanding these tTreg activation pathways may produce potent antigen-specific Treg for therapy.

Abstract No. 119

INTERLEUKIN-6 RECEPTOR BLOCKADE IMPROVES SURVIVAL AND REDUCES GRAFT-VERSUS-HOST DISEASE IN HUMANISED MICE TREATED WITH POST-TRANSPLANT CYCLOPHOSPHAMIDE

MILES Nicole[1,2,3], ADHIKARY Sam[1,2,3], GERAGHTY Nicholas[1,2,3], SLUYTER Ronald[1,2,3], ALEXANDER Stephen[4], WATSON Debbie[1,2,3]

[1]School of Chemistry and Molecular Bioscience, Faculty of Science, Medicine and Health, University of Wollongong, [2]Illawarra Health and Medical Research Institute, [3]Molecular Horizons, University of Wollongong, [4]Centre for Kidney Research, The Children's Hospital at Westmead, Sydney

INTRODUCTION: Graft-versus-host disease (GVHD) is a major complication of donor stem cell transplantation, where donor immune cells in the graft attack host tissues. We have shown depletion of proliferating immune cells using post-transplant cyclophosphamide (PTCy) can reduce signs of GVHD in humanised mice. However regulatory T cells (Tregs) are reduced over time. Interleukin-6 receptor (IL6R) blockade is known to reduce activated T cells and increase Tregs.
AIM: To investigate the effect of adding IL6R blockade to PTCy on GVHD development in humanised mice.
METHODS: NOD-SCID-IL2Rγ^{null} mice were injected (i.p.) with 20 x 10^6 human peripheral blood mononuclear cells (day 0). PTCy (33mg/kg) was injected (days 3, 4) and tocilizumab ($n = 6$) or control IgG antibody ($n = 3$) (0.5mg/mouse) twice a week for 4 weeks. Mice were monitored for clinical signs of GVHD for 10 weeks. Histological damage to target organs and serum cytokines were assessed.
RESULTS: Tocilizumab, which blocks human IL6R, did not affect human T cell engraftment ($CD4^+$, $CD8^+$), or the proportion of Tregs ($CD4^+CD25^+CD127^{lo}$), but increased $CD39^{hi}$ Tregs. Humanised mice treated with tocilizumab demonstrated lower clinical scores (Toc; 4.2±0.8 vs Ctl; 8.3±0.3), ($p < 0.05$) and prolonged survival (Toc; median survival time (MST) >70 days vs Ctl; MST, 53.5 days), ($p < 0.05$). Tocilizumab mice showed reduced histological damage to the gut and skin. Tocilizumab did not affect human serum cytokine levels (hIFN-γ, TNF-α, IL-6, IL-10, IL-2).
CONCLUSION: Additional therapy using tocilizumab reduces GVHD in humanised mice treated with PTCy, and further studies are warranted.

Abstract No. 120

LOW-DOSE INTERLEUKIN-2 INCREASES HUMAN REGULATORY T CELLS IN HUMANISED MICE WITH GRAFT-VERSUS-HOST DISEASE TREATED WITH POST-TRANSPLANT CYCLOPHOSPHAMIDE

ADHIKARY Sam[1,2], GERAGHTY Nicholas[1,2], SLUYTER Ronald[1,2], ALEXANDER Stephen[3], WATSON Debbie[1,2]

[1]Immunology and Cell Signalling Group, Illawarra Health and Medical Research Institute, [2]School of Chemistry and Molecular Bioscience, University of Wollongong, [3]Centre for Kidney Research, The Children's Hospital at Westmead, Sydney

INTRODUCTION: Graft-versus-host disease (GVHD) is a life threatening complication of donor stem cell transplantation where reactive donor T cells in the graft attack the host. We have shown that post-transplant cyclophosphamide (PTCy) depletes reactive donor cells and significantly reduces GVHD in humanised mice. In humans, low-dose interleukin (IL)-2 reduces GVHD by boosting regulatory T cells (Tregs). **AIM:** To investigate the effect of combinational treatment of PTCy and low-dose IL-2 on GVHD in humanised mice. **METHODS:** NOD-SCID-IL2Rγ^{null} mice were injected (i.p.) with 20 x 10^6 human peripheral blood mononuclear cells (day 0), followed by 33 mg/kg PTCy (days 3 and 4) and 0.3 IU IL-2 (or PBS) (day 0-5, then thrice weekly). Mice were monitored for clinical GVHD for 10 weeks, with flow cytometric assessment of human cell engraftment, qPCR analysis of regulatory genes and H&E assessment of damage to target organs. **RESULTS:** Engraftment of $hCD45^+$ leukocytes were similar between PTCy+PBS- and PTCy+IL-2-mice ($P>0.05$), the majority being $hCD3^+$ T cells in each group ($P=0.75$). Both groups demonstrated similar $hCD4^+$:$hCD8^+$ T cell ratios ($P=0.561$), however PTCy+IL-2-mice showed significantly greater $hCD4^+hCD25^+hCD127^{lo}$ Tregs ($P=0.045$). PTCy+PBS- and PTCy+IL-2-mice demonstrated similar survival (MST=65 days vs. 53 days, respectively), ($P=0.613$), which was prolonged compared to IL-2-mice (MST=38 days) ($P<0.05$ for both). Finally, PTCy+PBS- and PTCy+IL-2-mice demonstrated reduced infiltrating liver leukocytes compared to IL-2-mice ($P<0.05$ for both), but similar relative expression of splenic human interferon-γ, hFoxP3 and hIL-17 ($P>0.05$ for all)
CONCLUSION: This study indicates that combined treatment of PTCy with low-dose IL-2 increases human Tregs that survive long-term in humanised mice.

Abstract No. 121

CHARACTERISATION OF TISSUE-RESIDENT T CELLS BY TRANSPLANTATION

PROSSER Amy[1,2], HUANG Wen Hua[3], LIU Liu[1], DE BOER Bastiaan[4], WATSON Monalyssa[1,2], DART Sarah[1,2], LUCAS Andrew[1], JEFFREY Gary[1], DELRIVIERE Luc[3,5], KALLIES Axel[6], LUCAS Michaela[1,7]

[1]School of Medicine, University of Western Australia, Perth, [2]School of Human Sciences, University of Western Australia, Perth, [3]School of Surgery, University of Western Australia, Perth, [4]Department of Anatomical Pathology, Fiona Stanley Hospital, [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]Immunology Department, Sir Charles Gairdner Hospital, Perth

Introduction: Tissue-resident (TR) T cells are located in all tissues tested thus far and exhibit rapid responses to stimulation. Organ transplantation involves not only transfer of organ tissue, but of TR T cells as well. Infiltration of the transplanted organ by recipient-derived lymphocytes is also likely to result in alloreactive TR T cells in the organ. Phenotypic characterisation of TR T cells is incomplete, as there appear to be marked subset and context differences in marker expression.

Methods: Using orthotopic liver transplants between congenically matched mouse strains, we have characterised donor and recipient T cells after transplantation. In depth phenotyping by flow cytometry was performed with 19 markers related to memory, activation, exhaustion, transcription factors, and chemokine and co-inhibitory receptors.

Results: Donor-derived T cell subsets exhibit varying degrees of tissue-residency within the liver after transplantation. Subsets which were retained to a higher degree displayed a distinct TR phenotype, as opposed to the heterogeneous phenotypes of low retention subsets. All markers, including those classically associated with tissue-residency were expressed to varying degrees in a subset-dependent manner. Proportions of infiltrating recipient T cell subsets became phenotypically similar to retained donor-derived TR T cells, indicating their residency within the transplanted organ.

Conclusions: A 'one-size fits all' descriptive phenotype of TR T cells does not reflect the true heterogeneity of these cells. Subset-specific differences in expression of all markers examined herein confirms the potential for varying localisation, retention and function of each cell type, warranting further investigation.

Abstract No. 122

CXCR3 DEPLETION IN PROLONGING ISLET GRAFT SURVIVAL IN MOUSE XENOTRANSPLANTATION

NICHOLSON Leigh[1], HU Min[1], PILGRIM Suzanna[2], HAWTHORNE Wayne[1], GREY Shane[2], O'CONNELL Philip[1]

[1]Centre for Transplant and Renal Research, Westmead Millennium Institute, Westmead Hospital, Sydney, [2]Transplantation Immunology Laboratory, Garvan Institute of Medical Research, Sydney

Aims: CXCR3 is a chemokine receptor expressed on activated T cells and is involved in the recruitment of effector T cells to facilitate immune responses. This project aimed to identify the therapeutic benefits of an anti-CXCR3 antibody in prolonging graft xenograft survival, using a pig to mouse islet transplant model.

Methods: BALB/c mice received pancreatic neonatal islet cell cluster (NICC) transplantations under the kidney capsule. Mice were then treated with an anti-CXCR3 antibody in combination with a very low dose of Rapamycin (0.1mg/kg, 4-10 times less than normal use), Rapamycin in isolation, or an anti-CXCR3 isotype control antibody. Subsequent and ongoing groups included anti-CXCR3 antibody in combination with Rapamycin and CTLA-4 Ig and CTLA-4 Ig in isolation. The first anti-CXCR3 antibody dosage plan was administered once a week for nine weeks. The present dosage plan included a second treatment in Week 1 post-transplantation. Graft survival was assessed up to 100 days post-transplant. Data is analyzed using flow cytometry, histology and C-Peptide measurement via assay.

Results: Mice receiving anti-CXCR3 antibody in combination with Rapamycin exhibited partial graft survival with positive insulin staining and low serum C-peptide over 100 days, while grafts were rejected in isotype control before 30 days. Flow cytometry data suggested initial anti-CXCR3 treatment reduced CXCR3+ CD8+ and CXCR3+ CD4+ T cells, but these were not effectively reduced at first two week post transplantation. Preliminary data from the present project shows that most CXCR3+ CD8+ and CXCR3+ CD4+ were effectively reduced in all groups receiving anti-CXCR3 treatment after an additional dosage in Week 1, when compared to groups receiving either Rapamycin or CTLA-4 Ig alone. The second cohort of animals are ongoing.

Conclusions: Reduction of CXCR3 expression in xenotransplantation recipients may contribute to the prolonging of graft survival.

AUTHOR INDEX

NOTE: Numbers refer to abstract number, *not* page number. Bold numbers indicate presenting author.

ABOU-DAHER Frederika 23
ADAMS Leon 60, 63
ADHIKARY Sam 50, 119, **120**
AKITO Inadome 76
ALEXANDER Ian 27
ALEXANDER Stephen 12, 35, 68, 119, 120
ALLEN Richard 17, 30, 41
AMOKRANE Kahina 59
ANDERSON Patricia 44
ANTHONY Chris 34
AOKI Naoto 66
APTE Simon 11
ASAMI Takeda 76
ASHFORD Jenna 72
ATKINSON Amy 80
AU Eric 8, **30**, 73, **85**, 106

BALTIC Svetlana 59
BARRY Simon 117
BAUMGART Amanda 61
BEDI Sukhandeep 28
BENNETTS B 68
BERTOLINO Patrick 26, 27
BHANDARI Mayank 51
BHATTACHARJYA Shanthanu 5, **114**
BISHOP Alex 26
BLUNDELL Jian 46
BOKKERS Kim 116
BONGONI Anjan 14, **47**, 48
BOUDVILLE Neil 7, 32, 39
BOURKE Michael 30
BOWEN David 26, 27
BROOKS Andrew 38
BULL Nick 54
BURLINSON Natalia Diaz 59
BURNS Heather 13, 45
BUTLER Nicholas 115
BYRNE Mandy 60, 63
BYRNE Sarah 18, **19**, 53

CAMPBELL Scott 7, 21, 37, 56, 70
CAO Lu 13
CAREW A 58
CARROLL Robert 22, 117, 110
CASTELLS Antoni 30
CATALAN Aimee 60, 63
CAVAZZONI Elena 36, 67, 74, 86, 88
CERVELLI Matthew 32
CHADBAN Steve 7, 23, 24, 30, 31, 35, 60, 63, 79
CHAKERA Aron 20, 32, 39, 91
CHAMBERS Daniel 11, 58, 78
CHAN Doris 32
CHAN Samuel 37
CHAPMAN Jeremy R 30, 85, 106
CHAU Quy Thuan 1
CHEN Sharon 100
CHEUNG Jason 62
CHEW Yi Vee 45
CHIKARAISHI Tatsuya 66
CHIU Chris 118
CHO Yeoungjee 56
CHRISTOPHI Christopher 82
CLAYTON Philip 5, 6, 7, 16, 17, **21**, 22, 37, 41, 81, 90
COATES Toby 5, 21, 22, 25, 44, 112, **113**, 114, 117
COCO Tina 115
COHNEY Solomon 21
COWAN Peter 14, 47, 48
CRAIG Jonathan 30, 61
CRAWFORD Michael 3, 42, 43, 94
CROMPTON Charles 4
CROSS Nicholas 15, 17, 30
CUTHBERTSON Peter 50

D'COSTA Rohit 84
D'ORSOGNA L 20
DANSIE Kathryn 21, **90**
DARBAR Archie 99
DART Sarah 49, 121
DE BOER Bastiaan 49, 121
DE LA MATA Nicole 36, 40, 67, 74, **86**, **88**
DE ROO Ronald 18
DE SANTIS D 20
DE SILVA Tharushi 11
DELRIVIERE Luc 49, 121
DIAS Brendan 5
DIEKMAN Fritz 30
DIVITHOTAWELA Chandima 11
DOBRIJEVIC Ellen 17, **41**
DOGRA Sharan 32
DONNELLAN Sine 15
DOYLE Aoife 116
DRABBELS Jos Jm. 59
DROGEMULLER Christopher 112, 113, 117
DUNCANSON E 81
DURKAN Anne 95

EASTMENT Jacques 2
EL-RASHID Maryam 33
ELIAS Anthony 98
EMMANUEL Sam 34
FACWETT Jonathan 115
FAÉ Ingrid 59
FAHIM Magid 56
FARIDI Pouya 27

FAWCETT Jonathan 60, 63, 78
FERNANDES Brian 108
FINK Michael 60, 63, 82
FISCHER Gottfried 59
FISHER Charles 52
FORD Sharon 80
FORTIER Catherine 59
FRANCIS Leo 107
FRANCIS Ross 2, 37, 56, 103
FURNISS Hayley 71

GAO Ling 116
GERAGHTY Nicholas 29, 119, 120
GETTS Daniel 31
GOCK Hilton 80
GOODMAN David 9, **10**, 44, 80, 84, 111
GOPAL Basu 84
GOVINDARAJULU Sridevi 101
GRACEY David 23, 60, 63
GRAMLICK Madelyn 64
GRAMNEA Indra 84
GREY Shane 33, 122
GRIFFIN Anthony 2, 18, 19, 53
GRSKOVIC Marica 59
GULYANI Aarti 6
GUO Henry 57, **77**, **97**

HALL Bruce 28, 118
HARDY Matthew 47
HAWLEY Carmel 37, 51, 56
HAWTHORNE Wayne J 18
HAWTHORNE Wayne 13, 18, 45, 46, 112, 122
HAYWARD Christopher 34
HE Bulang 4, 39
HEDLEY James 36, **67**, 74, 83, 86, 88
HEER Munish 54, 64
HEIN Martin 39
HEPBURN Kirsten 98, **99**, **107**
HERLIHY David 52, 55
HICKS Mark 116
HIHO Steven 92
HILL Prue 111
HITOS Kerry 18
HOANG Khac Chuan 1
HODGKINSON Peter 115
HODGKINSON Suzanne 28, 118
HO G 68
HOLMAN K 68
HOPE Christopher M 117
HOPKINS Peter 11, 58
HORT Amy 18
HOWARD Kirsten 30, 81
HOWE Kathy 44
HOWELL Martin 61, 69, 81
HOWSON Prue 91
HUANG Dandan 13, 45
HUANG Wen Hua 49, 121
HUDSON Fiona 84
HUGHES Peter 16, 21, 71, 84
HULTIN Sebastian 70
HU Min 12, **13**, 122
HURTADO Plinio 25
HU Xinxin 108

IERINO Frank 21, 48, 75, 80
ILIFF James 34
IMRAN Mohammad 34
IRISH Ashley 20, 21, 51
IRISH Georgina 7
ISBEL Nikky 2, 21, 37, 56, 78

JABBOUR Andrew 34
JAFFREY L 78
JAHAN Sadia 107
JALALONMUHALI Maisarah 22
JAMBOTI Jagadish 51
JAMES Laura 30, 69
JAQUES Bryon 39
JEFFREY Gary 32, 60, 63, 121
JEFFREY G 49
JESUDASON Shilpa 6
JIMENEZ-VERA Elvira 45
JOHN George 107
JOHNS Daniel 113
JOHNSON David 2, 37, 56, 85
JOHNSTON Julie 113
JONES Daryl 71
JOSEPHSON Michelle 61
JOYCE David 32, 105
JU Angela 61
JUN Shoji 76

KABLE Kathryn 100
KALLIES Axel 49, 121
KANAGARAJAH Vijay 2, 18. 19
KANELLIS John 7, 21, 71, 84, 85, 106
KAREPALLI Vijay 72
KATSUOKA Yuichi 66
KAUSMAN Joshua 84
KAY Thomas 44, 112
KELLY Patrick 36, 67, 74, 86, 88
KENCH James 94
KEOGH Anne 34
KIEU Anh 30
KIM Dana 98, 99
KIM Juewan 117
KING Nicholas 31
KOHEI Kinoshita 76
KOSUKE Tanaka 76
KOTLYAR Eugene 34
KOTSIOU George 98
KUCHEL Timothy 113
KULKARNI Hemant 91
KUMMROW Megan 92
KWAN Tony 35

LAI Sum Wing Christina 23, **31**, 35
LAM Andrew 118
LAM Susanna 70, **93**

LAM Vincent 18
LARKINS Nick 4, **20**
LAURENCE Jerome 93
LAWSON Malcolm 18, 19, 53
LEE Darren 84
LEE Deanna 59
LEE VWS 8
LENDZION Rebecca 46
LEONG Mario 26, 27
LEVVEY Bronwyn 92
LEW Andrew 14
LI Jennifer 33
LIM Wai 20, 21, 30, 32, 37, 60, 63, 85, 91, 105, 106
LIU Ken 42, 43, 108
LIU Liu 49, 121
LIU Xiao 117
LI Yan 35, 57, 77, 97
LOCKWOOD David 18, 19
LOH Yik Wen 31, 35
LOK Charmaine 30
LOUDOVARIS Thomas 44, 112
LU B 12
LUCAS Andrew 121
LUCAS Michaela 49, 121
LY Mark 3, **94**

MACASKILL Petra 30
MACDONALD Peter 34, 116
MACHET David 111
MACIA Laurence 35
MAJONI Sandawana 72
MAJUMDAR Avik 42, 43, 108
MARIKO Toyoda 76
MARSH Bree 107
MARTIN Kylie 111
MARUI Yuhji 66
MASSON Philip 40
MASTERSON Rosemary 84
MATAS David 87
MATHERS Simon 59
MATHEWS Loren 113
MATSUMURA Kaori 66
MAWSON Jane 23
MCCARTHY Hugh 68
MCCAUGHAN Geoff 42, 43, 60, 63, 108
MCDONALD Mark 85, 106
MCDONALD Stephen 6, 9, 10, 16, 37, 81, 85, 106
MCEVOY Leanne 84
MCGINN Stella 57, 77, 97, 98
MCKENZIE Catriona 94
MCMICHAEL Lachlan 110
MCRAE Jennifer 47, 48
MIFSUD Nicole 27
MILES Nicole 119
MINCHAM Christine 4
MINHAS Nikita 33
MOGULLA Manohar 72
MORELLI Adriana Baz 47
MORITZ Peter 55
MOU Lingjun 39
MUDGE David 56
MUIRHEAD Robb 113
MULLEY William 21, 65
MUNN Stephen 2
MUNRO Colleen E 62
MURALIDHARAN Vijayaragavan 82
MURPHY J 48
MUTHIAH Kavitha 34

NACHBUR U 48
NAKAZAWA Ryuto 66
NANKIVELL Brian J 100
NEWMAN Allyson 23
NGO Xuan Thai 1
NGUYEN-NGO Danny 33
NGUYEN Duy Dien 1
NGUYEN Thanh-Tuan 1
NGUYEN Trong Hien 1
NICHOLSON Leigh 122
NIEWOLD Paula 31
NISHI Tomohiro 66
NITSCHKE Jodie 113
O'CONNELL Phillip 13, 44, 45, 112, 122
O'CONNOR Timothy 59
O'LEARY Michael 36, 67, 74, 86, 88
O'LONE Emily 40
O'SULLIVAN Brendan 11
OLAKKENGIL Santosh 5, 114
OPDAM Helen 71, 85, 106

PAIZIS Kathy 75, 84
PALMER Suetonia 15, 37
PANDYA Keval 42, **43**
PANLILIO Mara 42, 43
PASCOE Elaine 37, 56
PAUL-HENG Moumita 26, 27
PEARSE Martin J 47
PEFANIS A 48
PENKO Daniella 113
PEREIRA Ryan 18, 19, **53**
PERERA Nadia 108
PERKINS Griffith 25, 117
PHILLIPS Jessica 85, **106**
PILGRIM Suzanna 122
PILMORE Helen 21, 106
PLEASS Henry 17, 18
POLKINGHORNE K R 65
POLLOCK Carol 30, 99
PRESTON John 2, 18, 19, 53,70
PROSSER Amy 49, **121**
PULITANO Carlo 3, 42, 43, 94
PULLAN Caitlin 113
PURCELL Anthony 27
PUTRINO Samantha 56
PUTTASWAMY Vikram 52, 55
PUTT Tracey 109

QUACH Do La 1

RADFORD Toni 44, **112**
RAKESH Prateek 28
RANA Abdul Ahad 5
RATHNAYAKE Geetha 72
RAY Mark 18, 19
REILING Janske 78, **115**
RHEE Handoo 18, 19, 53
ROAKE Justin 17
ROBERTSON Ian 18, 19, 53
ROBERTSON Paul 18
ROBINSON Catherine 28, 118
ROBINSON S 12
ROELEN Dave 59
ROGER Simon 30
ROGERS Natasha 33
ROSALES Brenda 36, 67, 74, 83, 86, 88
ROSS David 59
RUSSELL Christine 114
RUSS Graeme 21, 32, 114
RYAN Brendan 18

SALLUSTIO Benedetta 32, 72
SALVARIS Evelyn 14, 47, 48
SANDERS Jo 71
SANTHAKUMAR Cositha 108
SASADEUSZ Joe 84
SASAKI Hideo 66
SASTRY Vinay 42, 43
SATO Yoshitsugu 66
SCHEUER Sarah 116
SCHOLLUM John 109
SCOTT Tahira 102
SENG Nina 84
SEN Shaundeep 30
SHAHRESTANI Sara 18, 46
SHANMUGALINGAM Renuka 62
SHARLAND Alexandra 26, 27, 28
SHARMA A 20
SHAW Evangeline 38
SHETTIGAR Reshma 109
SHIGEYOSHI Yamanaga 76
SHUN Albert 95
SINGER Julian 31, **35**
SLADDEN T 78
SLOGGETT Clare 75
SLUYTER Ronald 29, 50, 119, 120
SNELL Gregory 38, 92
SON Eric 27
SON Taeyoung 26
SORARU Jacqueline 105
SO Sarah 8, **96**
SPICER Timothy 96
SRINIVASA Vinay 101
STANKOVIC Sanda 38
STOKLOSA Ted 108
STRASSER Simone 42, 43, 108
SULLIVAN Lucy 38, 92
SUNDERLAND Andrew 32
SYPEK Matthew 6, **16**, 37, 75, **81**

TA'EED A 65
TAHER Amir 95
TAM Constantine 111
TAN Ai Lin 18, 19
TAN Ai Lin 2
TANGIRALA Nishanta 60, **63**
TANG James 69
TAN Jian 35
TAN Maxine 11
TATIANA Tsoutsman 108
TAUPIN Jean-Luc 59
TAVERNITI A 20
TAYLOR Andrew 111
TEIXEIRA-PINTO Armando 30, 73, 105
THAI Kinh Luan 1
THAI Minh Sam 1
THOMAS Gordon 95
THOMPSON Peter 105
THOMSON Imogen 74, **83**
TITUS Thomas 104
TONG Allison 30, 61, 69
TONG Marcus Chen Yee 4
TORPY David 112, 113
TRAN Giang 28, 118
TRAN Ngoc Sinh 1
TRAN Quoc 52, **55**
TRAN Trong Tri 1
TRAPNELL Frank 39
TREVILLIAN Paul 21, 54, 64
TURNER Robin 30
ULLAH Shahid 9, 10
USUBA Wataru 66

VASILARAS Arthur 17
VERMA Nirupama 28, **118**
VERRAN Deborah 93
VIARD Thierry 59
VIKSTROM Ingela 47
VILLANUEVA Jeanette 116
VIRTUE Susan 42, 43
VOISEY Joanne 11
VON HUBEN Amy 73
VU Duc Huy 1

WALCOTT James 82
WALKER Robert 109
WALLACE Gordon 117
WALLER Karen 36, 86, 88
WALTON Duncan 23
WANG Chuanmin 26, 28, 35
WANG Sarah 59
WANG YM 12
WAN H 12
WATSON Debbie 29, 50, 119, 120
WATSON Monalyssa 121
WATSON Narelle 23
WEBSTER Angela 36, 40, 67, 74, 83, 86, 88
WEIGHTMAN AC 89
WELLS Mark 42, 43

WENDA Sabine 59
WESTALL Glen 38, 92
WEST Claire 42, 43
WHITLAM John 84
WILARUS A 12
WILCOX Paul 28
WILLIAMS Gabrielle 30
WILLIAMS Lindy 45
WILLIAMS Narelle 30
WILLIMAN Jonathon 15
WILLIS Frank 4
WILSON Gregory 103
WONG Germaine 8, 17, 20, 21, 30, 32, 41, 60, 63, 69, 73, 81, 85, 105, 106
WONG Jeffrey 62
WOOD Simon 18, 19, 53
WU Huiling 31, 35
WYBURN Kate 21, 23, 24, 36, 60, 63, 67, 74, 83, 86, 88
WYLD Melanie 24, **40**
WYMANN Sandra 47

XIAOQIAN Ma 13

YANG Jean YH 30
YAUSIS Samuel 64
YAXLEY Julian 102, **104**
YEE Seow Yeing 79
YERKOVICH S 58, 78
YING Tracey 24, 79
YI Shounan 13
YOZA Naoto 66
YUEN Lawrence 18
YUE Zhilian 117
YUJI Hidaka 76

ZAHOROWSKA Beata 62, **100**
ZAMMIT Nathan 33
ZHANG Gy 12
ZHAO Yuanfei 13

T • S • A • N • Z

The Transplantation Society of Australia and New Zealand Inc.

ABN 90 796 930 798

EXECUTIVE OFFICER
Sommer Twycross
145 Macquarie Street
Sydney NSW 2000
Australia
Tel: (02) 9256 5461
Fax: (02) 9241 4083
E-mail:tsanz@tsanz.com.au
Web: www.tsanz.com.au

NOTICE OF ANNUAL GENERAL MEETING

The Annual General Meeting of the Transplantation Society of Australia and New Zealand held on Monday 30th April 2018 in Room 106, Level 1, Melbourne Convention Centre at 5.00pm.

Present: 51 members of the Society were present at the meeting which was chaired by the President, Professor Steve Alexander, who welcomed everyone to the meeting.

Also in attendance: Ms Sommer Twycross, Executive Officer and Mrs Nieves Piaggio, Administrative Officer

1. APOLOGIES

Dr Robert Carroll, Professor Patrick Coates and Professor Peter Macdonald

2. CONFIRMATION OF THE MINUTES

The minutes of the Annual General Meeting held on 8th May 2017 were passed as a true record.

Moved: Peter Cowan
Seconded: Robert Jones

3. BUSINESS ARISING FROM THE MINUTES

Nil.

4. PRESIDENT'S REPORT

Professor Steve Alexander thanked everyone for attending. He said that attendance to all meetings was up from last year with the turnout at the Masterclass increasing by over 50%. He then went on to thank the convenors Lucy Sullivan and Rosemary Masterson (ASM), John Whitlam and Rhonda Holdsworth (PGC) and Darren Lee and Veena Roberts (Masterclass).

He told members that, thanks to Rob Carroll, the financial standing of the Society was going well.

He then summarised the various projects that TSANZ had undertaken with assistance from OTA (Infections – developing new Guidelines, getting involved with the Transplant review with both National and State Governments, Indigenous outcomes, and Equity). He confirmed that TSANZ continued to be supportive of associated meetings such as VSOT (Professor Richard Allen) and the Liver Meeting (Professor Geoff McCaughan)

With regard to the Advisory Committees, Steve Alexander thanked all the committee members. He informed membership that TSANZ were working towards forming an Indigenous Working Group as well as changing the Paediatric Working Group to an Advisory Committee.

Members were told that TSANZ continues its good relationship with OTA through the Transplant Liaison and Reference Group (TLRG).

He reminded members that Council elections would take place in 2019 and that nominations would begin in October 2018. He encouraged all put their name forward.

He then thanked the current Council members: Professor Patrick (Toby) Coates (President Elect), A/Prof. Kelli MacDonald, Dr Christine Russell (surgeon), Dr Natasha Rogers (Honorary Secretary) and Associate Professor Bronwyn Levvey, Dr Rob Carroll (Treasurer), Dr Nick Cross (NZ), A/Prof. Nick Shackel (Liaison officer with the College of Physicians) and Nigel Palk (ATCA representative) for their talent, expertise and skills and highlighted its gender representation. He also acknowledged the Administration staff of Sommer Twycross, Nieves Piaggio and Kim Rawson (new Project's Officer)

5. TREASURER'S REPORT – Given by Natasha Rogers

Natasha Rogers confirmed stability in terms of the finances of TSANZ even though there has been a fall in sponsorship during the last 3 years. However, expenses for meetings and awards had remained stable. She stated that Council were looking at new ways to generate revenue. Society funds were sitting in low risk term deposits and therefore no making much of a return.

She then referred the membership to the Financial Report of 31st December 2017 and proposed that it be accepted:

Moved: Bronwy Levvey
Seconded: Nikki Isbel
Passed unanimously.

6. SECRETARY'S REPORT

Natasha Rogers, the Honorary Secretary, confirmed that the number of members was stable at 592 with an approximate 50/50-member split. She pointed out that of those, only a small number were students. Therefore, to encourage more student membership, Council had discussed at their last Council meeting that student rates (full-time students without TTS) be reduced from $55 to $22. She called for a show of hands where more than 50% of the quorum accepted. She encouraged all to let their students know.

She said that following up from the workshop facilitated by Novartis in November 2016, Novartis were planning a mentoring and networking workshop on August 3, 2018 at their headquarters in Macquarie Park Sydney. She stressed that it was not gender exclusive and that Novartis were not subsidising travel. She stated

that attendees did not have to be members and was free.

Natasha Rogers then presented members with the proposed constitution change (to be voted on at the AGM 2019) with regard to the representation on Council. The wording in Section 2(b) (vi) would include *"at least 3 men and 3 women"*.

7. REPORT ON ADVISORY COMMITTEES/WORKING GROUPS – given by Nick Cross

He advised membership that the Advisory Committees and Working Groups were working well as was the TLRG meetings with OTA. He notified members of the retirement of Eva Mehakovic.

He went on to inform membership that the meeting held on March 16, 2018 with the Minister for Health, Kenneth Wyatt, regarding improving Indigenous health, especially access to kidneys, had gone well.

Work was being planned on creating an Advisory Group around Ethics (incorporating the Declaration of Istanbul), changes to the Organ transplantation guidelines as well as developing guidelines for infectious transmitted diseases.

The proposal for an MBS task force for solid liver transplantation had been submitted.

8. SCIENTIFIC PROGRAM & EDUCATION COMMITTEE REPORT (SPEC)

A/Professor Daniel Chambers, Co-Chair of SPEC, thanked this year's organisers of all 3 meetings. He said the attendance figures were exceeding 2017 and there had been positive feedback especially about the basic science component of the Masterclass.

He then advised that both himself and A/Professor Kelli MacDonald term as co- chairs of SPEC would conclude at the AGM in July 2019 (Sydney) with Council choosing Dr Wai Lim and Dr Lucy Sullivan to take their place.

9. LIAISON WITH SCIENTIFIC SOCIETIES

No discussion.

10. GENERAL BUSINESS

Nil

There being no further business the meeting closed at 5.20pm.

Steve Alexander	Natasha Rogers
President	Honorary Secretary

PRESIDENT'S MESSAGE

It is with great pleasure I write to let you know about the many achievements and developments for the Transplant community and for the families and patients we care for. This is my second year as President and one where Council, SPEC, the working groups and the many people involved in the organisation of meetings and reports and the staff of TSANZ have done an enormous amount of work to strengthen transplantation at all levels and to improve the Society.

Last year we had a wonderful ASM in Melbourne that brought a host of international and national speakers, but also one that encapsulated the fun and joy of the TSANZ. Much of this can be sheeted home to Rosemary Masterson and Lucy Sullivan as the organisers and the admirable work of SPEC in ensuring that new science, good ideas, camaraderie welcomed everyone throughout the transplant community. We saw excellent presentations in some of the cutting edge of transplant science, with Vijay Kuchroo, Sandy Feng, Stan Jordan and Kathryn Tinckam. Carmel Hawley led us into translational medicine and trials, and Misty Jenkins illuminated us on implicit bias. Local speakers such Axel Kallies provided world leading research. The young investigator session stood out as a showcase of clinical and basic research in our society. It was wonderful to see the first Aviva award for patient care and to have Charmaine Simeonovic, who had known Lafferty, win the Lafferty Award.

This year's meeting organised by Natasha Rogers and Bill Mulley in Sydney offers to be equally exciting returning to Sydney, at the University of Sydney, with a range of outstanding local and international speakers as well as a focus on patient engagement. There will also be preceding post graduate course and Masterclass and a weekend heart lung meeting organised by Bronwyn Levvey and Andrew Jabbour on organ perfusion. SPEC, which has been a great success and provided support for all the meetings, with Dan Chambers and Kelli McDonald as chairs who are now handing over to Wai Lim and Lucy Sullivan, the incoming chairs.

We continue to work with colleagues and friends in KHA, ANZSN, OTA, Beat CKD, the Menzies Institute, the Red Cross and patient groups such as Transplant Australia on a variety of initiatives; in particular, two major initiatives are of note.

The introduction of Organ Match in April was a major stepping-stone in improving organ allocation and meeting the needs of transplanting units and their patients. Major credit goes to Rhonda Holdsworth and John Kanellis and the team supported by OTA that put this together and as ongoing rollouts of this system occur the opportunity exists to move prior and ongoing work on allocation to maximise equity and utility for the benefit of patients.

Second is the strong work in indigenous transplantation by the indigenous working group. The report listed on the TSANZ website outlines the severe state of waiting list and transplantation outcomes for indigenous patients. At the recent ISN, TSANZ hosted indigenous patients from Canada and New Zealand as well as local patients who shared their stories and with great warmth and humour addressed barriers that exist in each of these jurisdictions. This work in this area led by Stephen McDonald and Toby Coates and their working group who building on a large body of work by many colleagues led to ongoing funding of $2.3 million dollars to address strategies to improve indigenous transplant outcomes and was strongly supported by the Minister Ken Wyatt.

Work with the OTA through the TLRG chaired by Steve Chadban has progressed well. Work orders on clinical guidelines, paediatric donation and allocation, and infectious issues have all been completed or are well under way. The 10 year meeting by OTA in Sydney earlier this year was a great success and brought together many components of the transplant sector. All of the Advisory groups have made significant achievements in their areas in particular RTAC under John Kanellis and now Kate Wyburn which have had a major impact on Organ Match. Of note is the exceptional work of the Women in Transplantation group who have provided ongoing advice and support for issues around equity.

Collaboration with ANZSN is strong with close collaboration with their transplant group headed by Ross Francis and William Mahoney.

The Council has been strong and united with a diversity of talent. In particular, I would thank Toby Coates for his tireless role as President-elect, his support and his work in multiple areas. Administratively, Council and the Advisory groups have been strongly supported by our administrative team. Sadly, Sommer Twycross has left after 8 years to work in Queensland but has been admirably replaced by Nieves Piaggio and Katie Graham and Kim Rawson as our project officer. The newly elected members, include Kate Wyburn and Phil Clayton as well as Helen Pilmore as our first New Zealand President-elect, leave Council in tremendous shape for future activities. We continue to discuss the possibility of a further TTS meeting in ANZ and the possibility of a paediatric IPTA meeting and welcome the intestinal rehabilitation meeting next year in Auckland.

The ASM will continue to rotate around the capital cities, which has proved a great success with next year's ASM in Adelaide combining with the Liver transplant meeting.

Our platinum sponsors Novartis and Astellas have provided enormous support not just to the society but also to a variety of initiatives enhancing equity, assisting adolescents and transition and linking us with our Asian neighbours.

We noted with sadness the Christchurch attacks, and have felt for our New Zealand members. We have been exceptionally lucky with the work Nick Cross has done in New Zealand and with Council and TSANZ and TLRG and OTA with benefits including the paired kidney exchange moving to include New Zealand. We also are excited to have a New Zealand president, and look forward to the benefits of further collaboration across the Tasman.

In summary it has been a great pleasure to be part of TSANZ and see so many people engaged across Australia and New Zealand to assist patients and donors and to do the very best for transplantation in all its manifestations.

Good luck for the upcoming ASM,

Best Wishes

Steve Alexander

TSANZ Membership List

AUSTRALIAN CAPITAL TERRITORY

CARNEY, G
*CUNNINGHAM, J
FALK, M
KWAN, T
MEHAKOVIC, E
SIMEONOVIC, C

NEW SOUTH WALES

ADHIKARY, S R
ALEXANDER, S I
ALLEN, R D M
ANDERSON, P F
ANTHONY, C
AOUAD, L
AU, E H K
BURNS, H
#BURTON, M
CALISA,V
CAO, J
CAO, L
*CELCER, J
CHADBAN, S J
CHAN, E
CHANDA, S
CHEN, J
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
CUTHBERTSON, P
*DATSON, L
*DAVIDSON, D
DCUNHA, U
DE LA MATA, N L
DHITAL, K
DIDSBURY, M
DIEP, J
DOBRIJEVIC, E L K
DURKAN, A

EL-RASHID
M ERLICH, J H
FAZEKAS DE ST GROTH, B
FERNANDO, M R
*FRITIS-LAMORA, R
GALLAGHER, M P
GALLAGHER, A
GAO, L
GEORGE, C R P
GERAGHTY, N
GILLIES, A H B
GLANVILLE, A R
GRACEY, D
GRAHAM, A R
GRANGER, E K
GREY, S T
GROOBY, K
#HABIJANEC (NEE BELMAR), B
HAGHIGHI, K
HAHN, D
HALOOB, I
HAMEED, A M
HANSON, C
HARKESS, M
HAVRYK, A P
HAWTHORNE, W J
HEDLEY, 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
JARDINE, M
JAYASINGHE, K
JHA, S
JIMENEZ-VERA, E
JU, X
KABLE, K
KELLY, J J
KELLY, P
KENNEDY, S E
KEUNG, K
KIM, S
KUBITSKIY, A
KWAN, J C
LAI, C
LAM, V
LAN, P
LAURENCE, J
LE PAGE, A K
LEE, V
LEONG, M
LEWIS, D
LI, J
LIN, R
LIUWANTARA, D
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
MONTEVERDE, R A
*MONTGOMERY, E
#MUNRO, C
MURUGASU, R
MUTHIAH, K
NABB, L
NANKIVELL, B J
NARESH, C N
NATFAJI, A
NICHOLSON, L
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
RAKESH, P K
RALPH, A
RAMAN, A
RAWLINSON, W D
REIMANN, F

*** Denotes ATCA/TSANZ Member**
 # Denotes TNA/TSANZ Member

RICHARDS (NEE MACKAY), K
RITCHIE, A
ROBERTSON, M R
*ROBERTSON, P R
ROBINSON, C
ROGERS, N
ROSALES, B M
*ROSS, C M
ROXBURGH, S
SANDERY, B J
SCHEUER, S
SCHOLES-ROBERTSON, N J
*SEIFERT, N
SEN, S
#SGORBINI, M
SHACKEL, N A
SHAHRESTANI, S
SHARLAND, A F
SHARMA, A
SHEN, Y Y H
SHINGDE, R
SHUN, A
SIEW, S M
SILVEIRA, P A
SIMPSON, A M
SINGER, J
SKALICKY, D
*SMITH, A
SON, E T
SPICER, T
SPROTT, P
*STEIN, A M
STORMON, M O
STRASSER, S I
SUD, K
TALAEI ZANJANI, N
TANG, J
TANGIRALA, N
THOMSON, I K
TONG, A
TREVILLIAN, P R
TUCH, B E
#UTSIWEGOTA, M
VAUGHAN, E
VERMA, N
VERRAN, D J
VILLANUEVA, J
VIRTUE, S
VOLOVETS, A
WALLER, K
WALTERS, S N
WAN, S
WANG, C
#WARE, J
WATSON, D
WEBSTER, A C
WEST, C A
WILLIAMS, L J
WONG, G
WONG, M G
WONG, N L
WOODHOUSE, E
WRIGHT, J
WU, H
WYBURN, K
WYLD, M
YAO, J
YI, S
YING, T
YONG, K
ZAHOROWSKA, B
ZAMMIT, N
ZHANG, G Y
ZHAO, Y

NORTHERN TERRITORY

BROWN, M
CAIRNES, S E
CASS, A HUGHES, J
ELLIOTT, R
KAREPALLI, V K
LAWTON, P D
THIELE, I

QUEENSLAND

ABBOTT, W J
#BETTENS, P
BROOKS, E
BURKE, M
BYRNE, S J
CAMPBELL, S B
CAREY, J M
CATERSON, R J
CHAMBERS, D
CHAN, S
*COCO, T
COSSART, A R
DIVITHOTAWELA, C
#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 RHOLLETT, P
HOPKINS, P
HULTIN, S
ISBEL, N M
#JARRETT, M
JAVORSKY, G
JEGATHEESAN, D K
KANAGARAJAH, V
LE TEXIER, L
#LEISFIELD, P M
LOCKWOOD, D
LYNCH, S V
MACDONALD, K P A
MACDONALD, G A
MALLETT, A
MARKEY, K A
MARTINS, P
MCSWEENEY, W T
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
ROBSON, A
*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
WINKS (NEE BRUCE), L A
WONG, J S C

SOUTH AUSTRALIA

BARBARA, J A J
BATEMAN, S
BHATTACHARJYA, S
BIMAL GUJARI, D
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
HOLMES-LIEW, C

*** Denotes ATCA/TSANZ Member**
 # Denotes TNA/TSANZ Member

HOPE, C
IRISH, G L
JESSUP, C F
JESUDASON, S
*JOHN, E V
*JONES, P
JUNEJA, R
KANG, K
KAPOJOS, J
KETTE, F
KIM, J
KRIGE, A J
LADHANI, M
LETT, B
MCDONALD, S P
MCMICHAEL, L
MILLS, R A
MOHAN RAO, M
MULLER, K
NEO, E L
PALK, N
PASSARIS, G
PAVATHUPARAMBIL ABDUL MANAPH, N
PERKINS, G B
RADFORD, T M
RAO, N
ROJAS-CANALES, D
RUSS, G R
RUSSELL, C H
SALEHI, T
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

VICTORIA
ANGUS, P W
ANDREW, N
ATKINSON, A R
BARRACLOUGH, K
BATEMAN, K
BERGIN, P
BLAIR, S L
BONGONI, A K
BROWN, F
BYRNE, A
CANTWELL, L S
CHEONG, 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
DWYER, K M
*DWYER, B M
#ELLIS, C
FANG, D
FINK, M
FISICARO, N
FORD, S L
#GARDNER-DIXON, P
GOCK, H
GOH, S K
GOLDMAN, A R
GOODMAN, D J
GOPAL, B
GOW, P J
GRYNBERG, K
HARDIKAR, W
*HERD, L
HIHO, S J
*HOBSON, J
HODGSON, A
HOLDSWORTH, R F
HUGHES, P
*HUGHES, T
IDEL, I
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
LANTERI, M B
LEE, D
LEE, S
LEONG, K G
LEUNG, P Y M
LEVVEY, B
LEW, A
LIAN, M
LOUDOVARIS, T
#LUSCOMBE, B
MACKNAMARA, E
MARIANA, L
MARTIN, D
MARTIN (NEE NGU), K
MASTERSON, R
MCGIFFIN, D C
MCRAE, J
MENAHEM, S
METZ, D
MIACH, P J
MICHELL, I D
MULLEY, W R
MULLINS, K
MURPHY, S S
NICHOLLS, K M
PAIZIS, K
PARASKEVA, M
PAVLOVIC, J
PEFANIS, A
PERINI, M
PIMENTEL, A L
RAMESSUR, S
*REA, A
ROBERTS, V
ROBERTSON, A J
ROZENKOV, V
RYAN, J
SALTER, S
SALVARIS, E
SANDIFORD, M
SANDRIN, M S
SAUNDER, A
*SHIPP, A
SINCLAIR, M
SNELL, G I
SOMERVILLE, C A
STANKOVIC, S
STARKEY, G D
STEDMAN, H A
STEINBERG, A G
STEVEN, M
SUH, N
SULLIVAN, L C
TA, J
TA-EED, A
TAIT, B D
TAN, S J
TESTRO, A
THOMAS, H
THOMSON, N M
TRAPANI, J A
TUPPER, K
#VAGO, A
WALCOTT, J C
WALKER, R G
WANG, B Z
WESTALL, G
WHITLAM, J
WILSON, S

*** Denotes ATCA/TSANZ Member**
 # Denotes TNA/TSANZ Member

#YIP, D

WESTERN AUSTRALIA

ADAMS, L
GO, S
GODDARD, K A
HE, B
HOWSON, P E
IRISH, A B
JAMBOTI, J S
#JARY, C
LARBALESTIER, R
LARKINS, N G
LAVENDER, M
#LAWRENCE, S
LIM, W H
LUCAS, M
MANICKAVASAGAR, R
MOU, L
MUSK, M
NANAYAKKARA, C
NG, Z Q
PERRY, G
PHILLIPS, J L
PROSSER, A
PUTRINO (NEE CHUA), S
PUTTAGUNTA, H
*SCHUAMANN, M A
*SMITH, M S
SORARU, J
SUNDERLAND, A
SWAMINATHAN, R
TASSONE, G
TING, C
VIECELLI, 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
*LANGLANDS,
J LEIKIS, M J
BOAN, P
DART, S
DELRIVIERE, L D D
DEMBO, L
DO NGUYEN, H
MANLEY, P
MCNALLY, A
MCWILLIAMS, T
MUNN, S R
MUTHU, C
PILMORE, H L
PRESTIDGE, C
ROAKE, J A
ROBERTS, A
RUYGROK, P N
HETTIGAR, R P
*SPRENGER, L
WALKER, R J
WASYWICH, C A
YASUTOMI, M

OVERSEAS MEMBERS

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

HONORARY LIFE MEMBERS

BIGGS, J C
BISHOP, G A
CHAPMAN OAM, J R
D'APICE, A
HALL, B M
MARSHALL, V C
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
#FAZACKERLEY, C A
FIDLER, S
GARAS, G

*** Denotes ATCA/TSANZ Member**
 # Denotes TNA/TSANZ Member

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)

*** Denotes ATCA/TSANZ Member**
 # Denotes TNA/TSANZ Member