AST | AMERICAN SOCIETY OF TRANSPLANTATION

CEOT

CUTTING EDGE OF TRANSPLANTATION

ONE TRANSPLANT FOR LIFE:

MANY PATHWAYS TO SUCCESS

FEBRUARY 23 — 25, 2017

ARIZONA BILTMORE • PHOENIX, AZ

NAME: _____

For more information, visit *myAST.org/meetings/CEOT*

Veloxis PHARMACEUTICALS

Expressors of the *CYP3A5*1* gene variant are rapid metabolizers of tacrolimus[1]

Race alone is not a sufficient clinical predictor

Prevalence of *CYP3A5*1* among patient types

45%-80%	15%-35%	5%-30%	13%-26%
African-American[2,3]	Asian[2]	Caucasian[2,4]	Hispanic[5]

Rapid metabolizers are at risk of underimmunosuppression[1]

Learn more about a potential therapeutic approach for these patients at the Veloxis booth

References: 1. Jacobson PA et al. *Transplantation*. 2011;91(3):300-308. **2.** Staatz CE et al. *Clin Pharmacokinet*. 2010;49(3):141-175. **3.** Trofe-Clark J et al. *Am J Transplant*. 2015;15(suppl 3). **4.** Kuehl P et al. *Nat Genet*. 2001;27(4):383-391. **5.** Claudio-Campos K et al. *Drug Metab Pers Ther*. 2015;30(2):87-105.

ONE LAMBDA
A Thermo Fisher Scientific Brand

SATELLITE BREAKFAST SYMPOSIUM

Precision Medicine in Transplantation:
Reclassifying Graft Rejection and Injury with the Molecular Microscope and Integrative Diagnostics

Friday, February 24th

7:00-8:00am

Frank Lloyd Wright Salon ABC

SPEAKERS

Philip F. Halloran, MD, PhD
Alberta Transplant Applied Genomics Centre
Edmonton, Canada

Alexandre Loupy, MD, PhD
Nectar Hospital
Paris, France

Carmen Lefaucheur, MD
Saint Louis Hospital
Paris, France

Gaurav Gupta, MD
Virginia Commonwealth University
Richmond, VA

(*This is not an official function of the CEOT Meeting and is not endorsed by AST.)
Breakfast provided by AST.

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HEART INSTITUTE

We are pleased to sponsor

CUTTING EDGE OF TRANSPLANTATION

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American Society of Transplantation

General Information

REGISTRATION DESK

Thursday, Feb. 23	10:00 AM – 7:00 PM
Friday, Feb. 24	6:30 AM – 5:00 PM
Saturday, Feb. 25	7:30 AM – 5:00 PM

EXHIBIT HALL (POSTERS, INDUSTRY DISPLAYS)

Thursday, Feb. 23	4:45 pm – 6:00 pm
Friday, Feb. 24	10:30 am – 3:00 pm
Saturday, Feb. 25	10:00 am – 5:00 pm

INDUSTRY DISPLAYS

Be sure to visit the following companies in the exhibit hall during breaks and receptions:

Novartis Pharmaceuticals Corporation

Sanofi Genzyme

Veloxis Pharmaceuticals

EVENING EVENTS

Reception and Posters
Thursday 4:45 PM – 6:00 PM

Join your colleagues for a warm welcome to the Cutting Edge of Transplantation meeting. View the posters and visit industry exhibit booths, and enjoy ample food and drinks with AST.

Poolside Reception, Paradise Pool,
Saturday 6:00 PM – 7:00 PM

Conclude your CEOT experience by winding down Poolside with your colleagues

MEALS

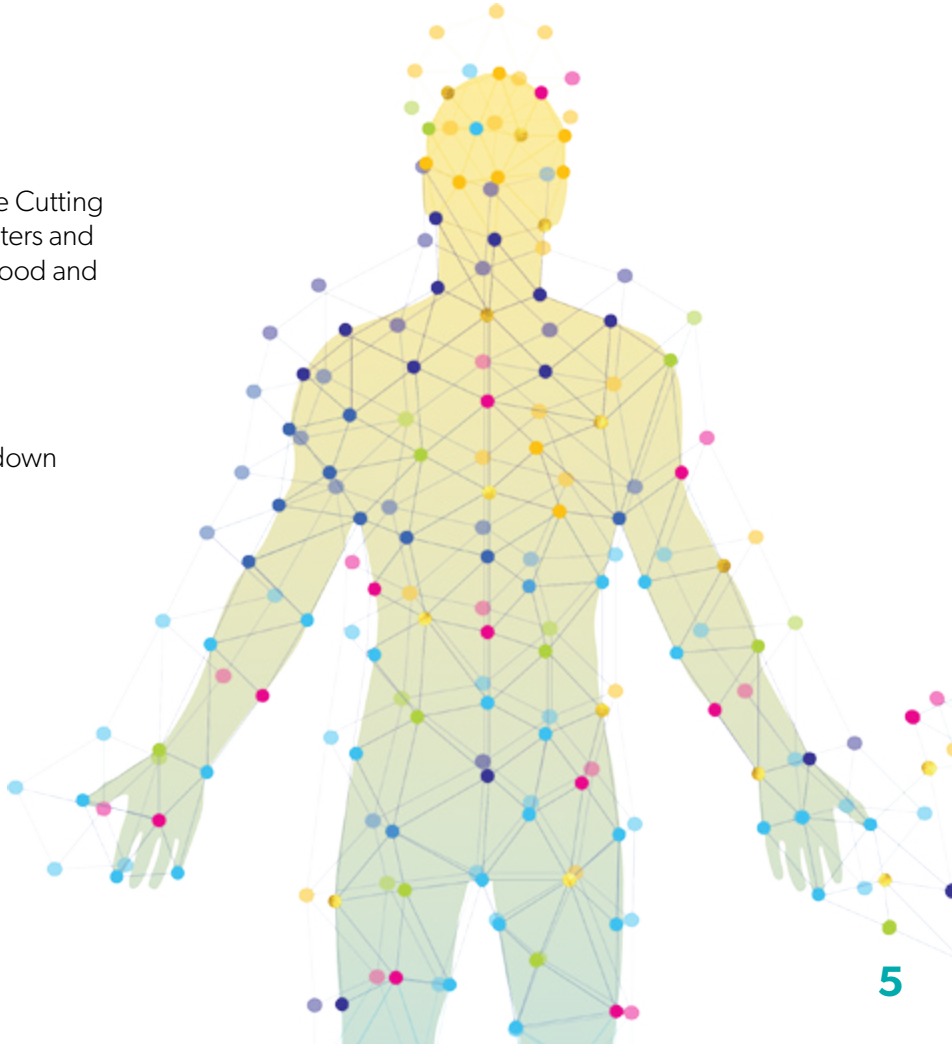
Breakfast is provided by AST Friday at 7:00 AM at the satellite symposia. A continental breakfast served Saturday at 7:30AM. Lunch will be provided by AST during the luncheon workshops. Breaks and evening refreshments will also be provided. Please visit the hotel concierge or the AST registration desk for dining suggestions for dinner.

NAME BADGE

All attendees must wear the AST-provided name badge at all times to gain access to CEOT events and sessions.

GUESTS

All guests must be registered and wear the AST-provided guest name badge at all times to gain access to the evening reception on Thursday. All other sessions and events are educational in nature and we request that guests do not attend.



Program Planning Committee

Anil Chandraker, MD, FAST, **2017 Co-chair**
Brigham and Women's Hospital

Kenneth Newell, MD, PhD, FAST, **2017 Co-chair**
Emory University School of Medicine

Andrew Adams, MD, PhD
Emory University School of Medicine

James S. Allan, MD, MBA, FAST
Massachusetts General Hospital

Aji Djamali, MD
University of Wisonsin-Madison

David P. Foley, MD, FACS
University of Wisconsin School of Medicine and Public Health

Richard Formica, MD, FAST
Yale University School of Medicine

Lorenzo Gallon, MD
Northwestern University, Division of Organ Transplant

Robert S. Gaston, MD, FAST
University of Alabama at Birmingham

John Gill, MD, MS, FAST
The University of British Columbia

Jon Kobashigawa, MD, FAST
Cedars-Sinai Heart Institute

Gwen McNatt, MS, CNN
Northwestern Memorial Hospital

Jacqueline G. O'Leary, MD, MPH
Baylor Health

Emilio Poggio, MD
Cleveland Clinic

Luke Preczewski
Jackson Health System

Melissa Yeung, MD
Brigham and Women's Hospital

Invited Faculty and Moderators

Andrew B. Adams, MD, PhD
Emory University School of Medicine

Tolulope Adesiyun, MD
Johns Hopkins

James S. Allan, MD, MBA
Massachusetts General Hospital

Paulina Alvarez
Cleveland Clinic

Francisco Arabia, MD
Cedars Sinai

Sharon Bartosh, MD
University of Wisconsin

Gerald Berry, MD
Stanford University

Sangeeta Bhorade, MD
Northwestern University

Roy D. Bloom, MD
Hospital of the University of Pennsylvania

Christopher D. Blosser, MD
University of Washington

Emily Blumberg, MD
Hospital of the University of Pennsylvania

Robert A. Bray, PhD
Emory University

Congressman Michael Burgess, M.D., (R-TX),
Chairman of the U.S. House of Representatives' Energy & Commerce Subcommittee on Health

Patricia Campbell, MBChB
University of Alberta

Darrell A. Campbell, Jr., MD
University of Michigan

Tim Caulfield, BSc, LL.B., LL.M
University of Alberta

Anil Chandraker, MD
Brigham & Women's Hospital

Patricia Chang, MD
University of North Carolina

Monica Colvin, MD
University of Michigan

Howard Eisen, MD
Drexel University

Jeremy Feldman, MD
Arizona Pulmonary Specialists

Sandy Feng, MD, PhD
University of California, San Francisco

Jay Fishman, MD
Massachusetts General Hospital Transplant Center

Richard N. Formica, Jr., MD
Yale University School of Medicine

Jon Friedman, MD
Optum Health Complex Medical Conditions

Nicholas Furiasse, MD
Cedars Sinai

Robert S. Gaston, MD
University of Alabama Birmingham

Howard M. Gebel, PhD
Emory University Hospital

John Gill, MD, MS
Providence Health Care

Ronald G. Gill, MD
University of Colorado of Denver HSC

David Goldberg, MD, MSCE
University of Pennsylvania

Hilary Goldberg, MD
Brigham and Women's Hospital

Gaurav Gupta, MD
Virginia Commonwealth University

Ramsay Hachem, MD
Washington University

Philip F. Halloran, MD, PhD
Alberta Transplant Applied Genomics Centre

Michael G. Ison, MD, MS
Northwestern University Feinberg School of Medicine

Annette Jackson, PhD
Johns Hopkins

Anthony M. Jevnikar, MD
London Health Sciences Center

Aarya Kafi, MD
Mayo Clinic

Bruce Kaplan, MD
Cedars-Sinai Heart Institute

Jon Kobashigawa, MD
Cedars-Sinai Heart Institute

Kevin Koomalsingh, MD
Cedars Sinai

Hrishikesh Kulkarni, MD
Washington University

Deepali Kumar, MD
University Health Network

Carmen LeFlaucheur, MD
Saint Louis Hospital, Paris

Chris Larsen MD, PhD
Emory University

Joseph R. Leventhal, MD, PhD
Northwestern University Feinberg School of Medicine

Deborah Levine, MD
University of Texas

Josh Levitsky, MD, MS
Northwestern University

Alex Loupy, MD, PhD
Necker Hospital Paris

Roslyn B. Mannon, MD
University of Alabama, Birmingham

James Markmann, MD, PhD
Massachusetts General Hospital

Arthur J. Matas, MD
University of Minnesota

Amit K. Mathur, MD, MS
Mayo Clinic Scottsdale

Invited Faculty and Moderators *(continued)*

Sumeet Mitter, MD
Northwestern

Dianne McKay, MD
University of California San Diego

Ulf Meier-Kriesche, MD
Bristol-Myers Squibb

Joan Merrill, MD
Oklahoma Medical Research Foundation

Robert A. Montgomery, MD, D.Phil, FACS
NYU Langone Medical Center

Kenneth Newell, MD, MS
Emory University School of Medicine

Peter Nickerson, MD
University of Manitoba Canadian Blood Services

Philip O'Connell, PhD, FRACP
University of Sydney at Westmead Hospital

Jignesh Patel, MD
Cedars-Sinai Heart Institute

Thomas Pearson, MD, DPhil
Emory University, Emory Transplant Center

Sean Pinney, MD
Icahn School of Medicine at Mt. Sinai

Ishna Poojary, MD
College of Medicine, Tuscon Arizona

Susan Prockop, MD
Memorial Sloan Kettering Hospital

Elaine Reed, PhD
David Geffen School of Medicine at UCLA

Nancy Reinsmoen, PhD
Cedars-Sinai

Rajeev Saggar, MD
Banner – University Medical Center Phoenix

Jesse Schold, PhD, M.Stat, M.Ed.
Cleveland Clinic Foundation

Daniel Schwartz, MD, MBA, CMS
U.S. Dept. Health and Human Services

John Sedor, MD
MetroHealth Medical Center

Jon Snyder, PhD
Chronic Disease Research Group

Laurie Snyder, MD
Duke University

Christopher D. Sonnenday, MD, MHS
University of Michigan Health System

Randall Starling, MD
Cleveland Clinic

Darren Stewart, MS
United Network for Organ Sharing

Samir Sultan, DO
Banner Health

Manikkam Suthanthiran, MD
Weill Cornell Medical Center

Anat Tambur, DMD, PhD
Northwestern University

Kathryn Tinckam, MD
University of Toronto

Nicole Turgeon, MD
Emory University

Dolly Tyan, PhD
Stanford University

Alex Wiseman, MD
University of Colorado of Denver HSC

The Honorable Kevin Yoder
United States House of Representatives

Lorenzo Zaffiri, MD
Duke University

Martin Zamora, MD
University of Colorado

Adriana Zeevi, MD
University of Pittsburgh

All general sessions take place in Frank Lloyd Wright Salon E-F.

Sessions in the heart track take place in Frank Lloyd Wright Salon A-B.

Sessions in the lung track take place in Frank Lloyd Wright Salon C-D.

Breaks will be held in the Frank Lloyd Wright Salon G.

Other sessions and event locations are noted within the program.

THURSDAY, FEBRUARY 23

1:00 PM - 2:30 PM — **Lunch Workshop – Tolerance Shark Tank***
Moderators: *Anthony M. Jevnikar, MD*
London Health Sciences Center
James S. Allan, MD, MBA
Massachusetts General Hospital

1:00 PM — Treg Studies in Clinical Transplant - Your Safest Investment
Sandy Feng, MD, PhD
University of California, San Francisco

1:30 PM — Who Needs T Regs if You Have Facilitator Cells and Chimerism?
Joseph R. Leventhal, MD, PhD
Northwestern University Medical School

2:00 PM — Tregs Are Not the Only ONE to Study
James Markmann, MD, PhD
Massachusetts General Hospital

2:30 PM — **Welcome Remarks**
Anil Chandraker, MD, FAST
Brigham and Women's Hospital
Kenneth Newell, MD, PhD, FAST
Emory University School of Medicine

2:45 PM – 4:45 PM — **Session 1: Time to Refocus from Early to Late Outcomes***
Moderators: *Alexander Wiseman, MD*
University of Colorado of Denver HSC
Roy D. Bloom, MD
Hospital of the University of Pennsylvania

2:45 PM — Observations from DEKAF
Arthur J. Matas, MD
University of Minnesota

3:15 PM — Can We Predict the Future and Change it?: Gocar
Phillip O'Connell, PhD, FRACP,
University of Sydney at Westmead Hospital

3:45 PM — Novel Strategies to Improve Adherence in Transplant Recipients
Christopher D. Blosser, MD
University of Washington

4:15 PM — Treatment of Chronic Antibody Mediated Rejection
Robert A. Montgomery, MD, D. Phil, FACS
NYU Lagone Medical Center

4:45 PM – 6:00 PM — **Poster Presentations and Welcome Reception**
Frank Lloyd Wright Salon G

6:00 PM – 7:30 PM — **Session 2: Challenges to Therapeutic Innovation in Transplant: Multiple Perspective***
Moderators: *Roslyn B. Mannon, MD*
University of Alabama, Birmingham
Andrew B. Adams, MD, PhD
Emory University School of Medicine

6:00 PM — Investigators Perspective
Chris Larsen, MD, PhD
Emory University

6:30 PM — Industry's Perspective
Herwig-Ulf Meier-Kriesche, MD
Bristol-Myers Squibb

7:00 PM — The Perspective of a Successful Outsider Path Forward
Joan Merrill, MD
Oklahoma Medical Research Foundation

FRIDAY, FEBRUARY 24

7:00 am – 8:00 am — **Satellite Symposium presented by One Lambda Inc., A Thermo Fischer Scientific Brand†**
This is not an official function of the CEOT meeting and is not endorsed by the AST.
Breakfast is provided by AST.
Frank Lloyd Wright Salon E-7

Precision Medicine in Transplantation: Reclassifying Graft Rejection and Injury with the Molecular Microscope and Integrative Diagnostics
Philip F. Halloran, MD, PhD,
Alberta Transplant Applied Genomics Centre, Edmonton, Canada
Alexandre Loupy, MD, PhD
Nectar Hospital, Paris, France
Carmen LeFaucheur, MD
Saint Louis Hospital, Paris France
Gaurav Gupta, MD
Virginia Commonwealth University, Richmond, VA

8:00 am — **Break**

**Continuing education credit offered. See separate packet.*
†No continuing education credit offered.

8:15 am – 10:45 am **Session 3:**
Select one of three sessions

Option One: No Transplantable Organ Left Behind*
Frank Lloyd Wright Salon E-7

Moderators: *Richard N. Formica, Jr., MD*
Yale University School of Medicine
Nicole Turgeon, MD, Emory University

8:15 AM — Waste not want not: Analysis of discarded organs.
Darren Stewart, M.S.
United Network for Organ Sharing

8:45 AM — New SRTR Metrics for Organ Acceptance
Jon Snyder, PhD
Chronic Disease Research Group

9:15 AM — What is the Risk in a PHS Increased Risk Kidney and How Do We Safely Use Them?
Michael G. Ison, MD, MS
Northwestern University

9:45 AM — Who Gets the Kidney at Risk for Discard: Decisions Under Pressure
Amit K. Mathur, MD, MS
Mayo Clinic Scottsdale

Option Two: **Antibodies Pre Heart Transplantation***
Frank Lloyd Wright Salon A-B

Moderators: *Howard Eisen, MD*
Drexel University
Patricia Campbell, University of Alberta

8:15 AM — Detection and Monitoring of Antibodies Pre-Heart Transplantation
Elaine Reed, PhD
David Geffen School of Medicine at UCLA

8:35 AM — Outcomes of Pre Transplant Antibodies: Non-VAD and VAD
Jon Kobashigawa, MD
Cedars-Sinai Heart Institute

8:55 AM — Should Sensitized Patients Have Allocation Priority?
Howard Eisen, MD, Drexel University

9:15 AM — Managing Sensitized Patients Pre-Heart Transplantation
Patricia Chang, MD
University of North Carolina

9:35 AM — Debate: Pre-Heart Transplant Conundrum: Desensitization Vs. Transplanting Against a Positive Flow Crossmatch

Pro Desensitization
Jignesh Patel, MD
Cedars-Sinai Heart Institute

Pro Transplanting Against a Positive Flow Crossmatch
Monica Colvin, MD, University of Michigan

Panel Discussion

Option Three: Antibodies Pre-Lung Transplantation*
Frank Lloyd Wright Salon C-D

Moderators: *Martin Zamora, MD,*
University of Colorado
Katherine Tinckam, MD
University of Toronto

8:15 AM — Strategies for the Assessment of Sensitization in Candidates for Lung Transplantation
Dolly Tyan, PhD, Stanford University

8:35 AM — Should the Presence of DSA Influence Recipient Selection in the Setting of Lung Transplantation?
Hilary Goldberg, MD
Brigham and Women's Hospital

8:55 AM — Complement Fixation and Risk Stratification in the Sensitized Lung Transplant Candidate
Adriana Zeevi, University of Pittsburgh

9:15 AM — Managing Sensitized Patients Pre-Lung Transplantation
Deborah Levine, MD, University of Texas

9:35 AM — Debate: Pre-Lung Transplant Conundrum: Desensitization Vs. Transplanting Against a Positive Flow Crossmatch

Pro Desensitization
Laurie Snyder, MD, Duke University

Pro Transplanting Against a Positive Flow Crossmatch
Ramsey Hachem, MD
Washington University

Panel Discussion

10:45 AM — Break

11:15 AM **Session 4: AM Keynote***

Historical Perspectives on the Failure to Enact the Immunosuppression Bill and Why this Goal Remains Important
Moderators: *Anil Chandraker, MD,*
Brigham & Women's Hospital
Dianne B. McKay, MD
University of California San Diego

11:15 AM — Historical Perspectives
Robert S. Gaston, MD
University of Alabama @ Birmingham

Plan Moving Forward
Congressman Michael Burgess, M.D., (R-TX), Chairman of the U.S. House of Representatives' Energy & Commerce Subcommittee on Health.

**Continuing education credit offered. See separate packet.*
†No continuing education credit offered.

12:30 PM **Buffet lunch served and proceed into Session**

12:45 PM **Satellite Symposium†**
Presented by Sanofi Genzyme
This is not an official function of the CEOT meeting and is not endorsed by the AST. Lunch is provided by AST.
Frank Lloyd Wright Salon A

A Critical Appraisal of Induction Therapy in Kidney Transplantation
Moderator: *Kenneth Newell, MD, PhD*
Emory University School of Medicine

Induction in 2017: Who Receives It and Who Needs It?
Alex Wiseman, MD
University of Colorado of Denver HSC

Induction With Depleting Agents: What Are the Anticipated and Actual Benefits?
Bruce Kaplan, MD, Mayo Clinic

Depleting Induction and Steroid Avoidance: Strategies and Benefits
E. Steven Woodle, MD

2:00 PM – 4:00 PM **Session 5:**
Select one of three sessions

Option One: Recipient Candidacy: Do the Right Thing!*
Frank Lloyd Wright E

Moderators: *Robert S. Gaston, MD*
University of Alabama @ Birmingham
Thomas Pearson, MD, D.Phil
Emory University – Emory Transplant Center

2:00 PM Saying No, When Is It Necessary?
John Gill, MD, MS
Providence Health Care

2:30 PM Approaches to Transplant the High - Risk Recipients
Alexander Wiseman, MD
University of Colorado of Denver HSC

3:00 PM Prehabilitation - Training the Frail
Christopher Sonnenday, MD, MHS
University of Michigan Health System

3:30 PM Which Hat Am I wearing: Public Steward or Patient Advocate?
Timothy Caufield, BSc, LL.B., LL.M
University of Alberta

Option Two: Antibodies Post Heart Transplantation*
Frank Lloyd Wright Salon A

Moderators: *Anat Tambur, DMD, PhD*
Northwestern University
Jon Kobashigawa, MD
Cedars-Sinai Heart Institute

2:00 PM Antibodies as a Marker for Poor Outcome Post-Heart Transplantation
Monica Colvin, MD, University of Michigan

2:20 PM Prevention of Antibody Development Post-Transplantation: Results from CTOT-11 Trial
Randy Starling, MD, Cleveland Clinic

2:40 PM Prevention of Complement Activation and Antibody Development: Results from the Duet Trial
Jignesh Patel, MD, PhD
Cedars-Sinai Heart Institute

3:00 PM Treatment of Detected Antibodies
Sean Pinney,MD
Icahn School of Medicine at Mt. Sinai

3:20 PM Non-HLA Antibodies Post-Heart Transplantation
Annette Jackson, PhD, Johns Hopkins

3:40 PM Post Heart-Transplant Conundrum: Do All Post HTx DSA Require Treatment? What About HLA Abs?
Adriana Zeevi, MD, University of Pittsburgh

Option Three: The Challenges of Post-Lung Transplant Antibodies*
Frank Lloyd Wright Salon C-D

Moderators: *Laurie Snyder, MD*
Duke University
Sangeeta Bhorade, MD
Northwestern University

2:00 PM Antibodies as a Marker for Poor Outcome in Lung Transplantation
Deborah Levine, MD, University of Texas, San Antonio

2:20 PM Prevention of Antibody Development Post Lung Transplantation
Martin Zamora, University of Colorado

2:40 PM Non-HLA Antibodies (K-alpha 1 tubulin, collagen V)
Nancy Reinsmoen, PhD, Cedars-Sinai

3:00 PM Treatment of Detected Antibodies in Lung Transplantation
Ramsey Hachem, MD
Washington University in St. Louis

**Continuing education credit offered. See separate packet.*
†No continuing education credit offered.

3:20 PM	Connection of Circulating Antibodies to Pathology AMR on Lung Biopsy *Gerald Berry, Stanford University*
3:40 PM	Post-Lung Transplant Conundrum: Do All Post Lung Transplant DSA Require Treatment? *Katherine Tinckam, University of Toronto*
4:00 PM	Break
4:15 PM	**Afternoon Keynote†** *The Honorable Kevin Yoder, United States House of Representatives*

SATURDAY, FEBRUARY 25

8:00 AM – 10:00 AM **Session 6:**
Select one of three sessions

Option One - Transplant Value- How to Survive the Next Decade*
Frank Lloyd Wright Salon E

Moderators: *Kenneth A. Newell, MD, PhD*
Emory University School of Medicine
John Gill, MD, MS
Providence Health Care

8:00 AM	Big Picture Healthcare Value/Reform in the US *John Sedor, MD* *Metrohealth Medical Center*
8:30 AM	Learning from the World Outside Transplantation *Darrell A. Campbell, Jr., MD* *University of Michigan*
9:00 AM	Value the Private Payor Perspective *Jon Friedman, MD* *Optum Health Complex Medical Conditions*
9:30 AM	Value the Public Payor Perspective *Daniel Schwartz, MD, MBA, CMS* *U.S. Dept. of Health & Human Services*

Option Two - Antibody Case Studies in Heart Transplantation*
Frank Lloyd Wright Salon A

Moderators: *Patricia Chang, MD*
University of North Carolina
Jignesh Patel, MD, PhD,Cedars-Sinai

8:00 AM	Pre-Transplant Treatment of the Sensitized Patient *Sean Pinney, MD, Mount Sinai* *Sumeet Mitter, MD, Northwestern*
8:20 AM	Sensitization in the VAD Patient Awaiting Transplant: When to Ignore and When to Treat *Annette Jackson, PhD, Johns Hopkins* *Tolulope Adesiyun, MD, Johns Hopkins*
8:40 AM	Does Persistence Pay Off? Treatment of Recurrent AMR Acutely and Longitudinally *Randall Starling, MD, Cleveland Clinic* *Paulino Alvarez, Cleveland Clinic*
9:00 AM	An Immune System Never Forgets: Treatment of Delayed Hyperacute Rejection Post Transplant *Monica Colvin, MD* *University of Michigan* *Nicholas Furiasse, MD, Cedars Sinai*
9:20 AM	A Case of Graft Dysfunction: Are Donor Specific Antibodies Bystanders or Instigators? *Francisco Arabia, MD,Cedars Sinai* *Kevin Koomalsingh, MD, Cedars Sinai*
9:40 AM	Panel Discussion

Option Three- Case Studies in Lung Transplantation: Challenges in Treatment*
Frank Lloyd Wright D

Moderators: *Deborah Levine, MD,*
University of Texas
Sangeeta Bhorade, MD,
Northwestern University

8:00 AM	Post Transplant Treatment fo DSA With & Without Lung Dysfunction *Martin Zamora, MD* *University of Colorado* *Aarya Kafi, MD, Cedars Sinai*
8:20 AM	Severe PAH with Cardiogenic Shock: Is Heart/Lung Transplant the Best Option? *Rajeev Saggar, MD* *Banner - University Medical Center Phoenix* *Samir Sultan, DO, Banner Health*
8:40 AM	Supporting the Decompensated PH Patient Awaiting Lung Transplant *Jeremy Feldman, MD* *Arizona Pulmonary Specialists* *Ishna Poojary, MD* *College of Medicine Tuscon Arizona*
9:00 AM	Acute AMR After Lung Transplantation *Ramsey Hachem, MD* *Washington University* *Hrishikesh Kulkarni, MD* *Washington University*
9:20 AM	Highly Sensitized Patient Awaiting Lung Transplant *Laurie Snyder, MD, Duke University* *Lorenzo Zaffiri, MD,* Duke University
9:40 AM	Panel Discussion
10:00 AM	Break

**Continuing education credit offered. See separate packet.*
†No continuing education credit offered.

10:15 AM – 11:45 AM	**Session 7: Post - Transplant Infections: Jeopardizing Long-Term Success***
	Moderators: *Emily A. Blumberg, MD*
	Hospital of the University of Pennsylvania
10 :15 AM	The Impact of Infections: Aspergillus to Zika
	Emily Blumberg, MD
	Hospital of the University of Pennsylvania
10:45 AM	The Microbiome: Implications on Graft Survival
	Jay A. Fishman, MD
	Massachusetts General Hospital Transplant Center
11:15 AM	T-Cell Therapies: Promise and Practice
	Susan E. Prockop, MD
	Memorial Sloan Kettering Hospital
11: 45 AM	Buffet Lunch served and proceed into session
Noon	**Lunch Workshop***
	Session 8: Hepatitis C- Death of an Old Foe
	Moderators: *Josh Levitsky, MD, MS*
	Northwestern U Feinberg School of Medicine
	Richard N. Formica, Jr., MD
	Yale University School of Medicine
12:00 PM	HCV Consensus Conference Summary
	Josh Levitsky, MD, MS
	Northwestern U Feinberg School of Medicine
	Richard N. Formica, Jr., MD
	Yale University School of Medicine
12:30 PM	Treatment of HCV Pre vs Post- transplant
	Roy D. Bloom, MD
	Hospital of the University of Pennsylvania
1:00 PM	HCV + donors to HCV – Recipients
	David Goldberg, MD, MSCE
	University of Pennsylvania
1:30 PM – 3:30 PM	**Session 9: Biomarkers and Personalized Medicine: Why Can't I Order It?**
	Moderators: *Ronald G. Gill, PhD*
	University of Colorado - Denver
	Jon Kobashigawa, MD
	Cedars Sinai Heart Institute
1:30 PM	Surrogates: Why Do We Need Them and Why Haven't We Developed Them?
	Jesse Schold, PhD, M.Stat, M.Ed.
	Cleveland Clinic Foundation
2:00 PM	Molecular Biomarkers - Why are There so Many Biomarkers to Predict Medicine and None are Used Clinically?
	Manikkam Suthanthiran, MD
	Weill Cornell Medical Center
2:30 PM	Surrogates as Clinical Endpoint and for Enrichment in Trials
	Alexander Loupy, MD, PhD
	Necker Hospital Paris
3:30 PM –4:00 PM	**Break**
4:00 PM – 5:30 PM	**Session 10: Donor Specific Antibody: Should We Avoid or Confront?***
	Moderators: *Peter Nickerson, MD*
	University of Manitoba Canadian Blood Services
	Sharon M. Bartosh, MD
	University of Wisconsin
4:00 PM	Understanding Risks and Consequences
	Kathryn J. Tinckam, MD, MMSc, FRCPC
	University Health Network
4:30 PM	Finding the Best Match: Looking to the Epitopes
	Anat R. Tambur, DMD, PhD
	Northwestern University
5:00 PM	Increased Matching the Impact on Equity
	Howard M. Gebel, PhD
	Emory University Hospital
	Robert A. Bray, PhD
	Emory University Hospital
5:30 PM	**Closing**
	Anil Chandraker, MD
	Brigham & Women's Hospital
	Kenneth A. Newell, MD, PhD
	Emory University School of Medicine
6:00 PM	Poolside Reception
	Paradise Pool, Cabana Club

**Continuing education credit offered. See separate packet.*
†No continuing education credit offered.

Young Innovator Award

This award is given to emerging professionals in transplantation with the top-scoring abstracts, as determined by the CEOT Program Committee.

CONGRATULATIONS

TO THE 2017 CEOT YOUNG INNOVATOR AWARD WINNERS:

Dina Abdelwahab
Yale University
New Haven, CT

Tamar Aintablian
Heart Institute, Cedars Sinai Medical Center
Los Angeles, CA

Songjie Cai
Osaka University Graduate School of Medicine
Osaka, Japan

Cindy Yip
University of Buffalo,
Buffalo, NY

Carolina Johnson
Mayo Clinic Hospital,
Phoenix, AZ

Nadeen Khoury
Mayo Clinic,
Rochester, MN

Magdalena Kwapisz
Medical University of Warsaw, Poland

Rihito Nagata
Tokyo University,
Tokyo, Japan

Caren Rose
University of British Columbia
Vancouver, BC

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POSTER ABSTRACTS # 1-30

Poster Session
Thursday, February 23, 2016
4:45 pm – 6:00 pm

Frank Lloyd Wright Salon G

ABSTRACT #1

TITLE: Belatacept Conversion in Kidney Recipients with Low GFR Doesn't Improve Renal Function

AUTHORS (FIRST NAME, LAST NAME): Dina Abdelwahab[1], Byron Smith[2], Bruce Kaplan[1], Raymond L. Heilman[1]

INSTITUTIONS (ALL): 1. Transplant Nephrology, Mayo Clinic Arizona, Phoenix, AZ, United States.
2. Biostatistics and Informatics, Mayo Clinic Rochester, Rochester, MN, United States.

Background: Belatacept interferes with signal 2 of T cell activation via blockade CD80/86 which doesn't adversely impact renal function. We tested a hypothesis that Belatacept conversion might improve GFR in kidney transplant patients with low GFR. We used propensity matching to select the control group. To our knowledge this is the first study which compared the Belatacept recipients to a matched control cohort on tacrolimus regimen.

Methods: In our center we have a clinical protocol to switch patients with low GFR to Belatacept if a biopsy shows no rejection. We exclude patients who are EBV or have a DSA.We included all kidney recipients who were converted from CNI to Belatacept between 1/2012 to 9/2016. We used propensity matching to control for age, sex, race, KDPI,DGF, donor source, eGFR at conversion, baseline biopsy cv score. The primary outcome was change in eGFR 4 months after conversion. We also looked at de novo DSA, rejection and graft survival. For these analyses, paired tests were used. Continuous data was tested using the paired t-test while categorical data was tested using McNemar s test.

Results: 31 patients were converted from tacrolimus to Belatacept were compared to a paired matched control. There was no difference in the change of eGFR at 4 months in the bela and control group (see the table). There was no significant difference in rejection rate, denovo DSA and graft survival between the groups.

Conclusions: We found no evidence Belatacept conversion improves eGFR 4 months after conversion in kidney transplant recipients with low eGFR.

Disclosure: Dina Abdelwahab: No | Byron Smith: No | Bruce Kaplan: No | Raymond Heilman: No

KEYWORDS: acute graft failure, CNI nephrotoxicity, co-stimulation blockade.

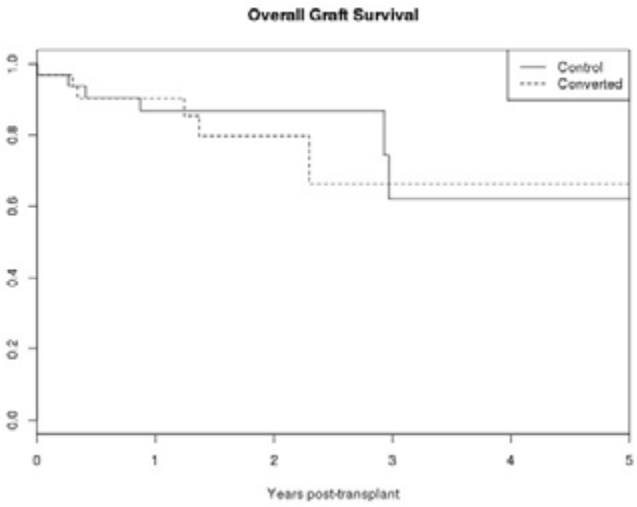


TABLE:

variable	Belatacept	control	difference	paired.test
Time.Zero.GFR	23.3(12.31)	26.73(12.61)	-3.43(4)	0.28
Four.Month.GFR	32.67(17.12)	33.82(13.74)	-1.15(19)	0.7754
Age	54.29(12.33)	55.35(12.04)	-1.06(5)	0.733
Gender	13(42%)	12(39%)	11(35%)	1
African American Recipient	2(6%)	1(3%)	1(3%)	1
Living Donor	6(19%)	7(23%)	5(16%)	1
Re-Transplant	3(10%)	3(10%)	2(6%)	1
Change in GFR	9.07(14.45)	6.91(9.89)	2.16(18)	0.5032
Post-Conversion	6(20%)	6(20%)	6(20%)	1
Acute Rejection Survival Analysis	669(819)	687(521)	0.8	0.739
de Novo DSA post conversion	12(41%)	6(21%)	12(41%)	0.15

ABSTRACT #2

TITLE: Primary Graft Dysfunction Post Heart Transplantation: Is There Greater Risk for De Novo Donor-Specific Antibody Development?

AUTHORS (FIRST NAME, LAST NAME): Jignesh Patel[1], Michelle Kittleson[1], Lawrence Czer[1], Tamar Aintablian[1], Tina Kao[1], Dael Geft[1], David H. Chang[1], Fardad Esmailian[1], Jon Kobashigawa[1]

INSTITUTIONS (ALL): 1. Heart Institute, Cedars-Sinai Medical Center, Los Angeles, CA, United States.

Background: Primary graft dysfunction (PGD) may occur in up to 30% of heart transplant recipients and has been associated with increased mortality. The development of *de novo* donor specific antibodies (DSA) has also been associated with increased mortality and risk of rejection after heart transplantation (HTx). It has not been established whether patients with PGD are predisposed to development of *de novo* DSA post-HTx.

Methods: Between 2011 and 2015 we assessed 494 patients for development of PGD at a single center. 43 patients were identified with PGD (left or bi-ventricular) and were compared to a non-PGD control. Outcomes for each group included 1-year survival, 1-year freedom from non-fatal major adverse cardiac events (NF-MACE: defined as myocardial infarction, congestive heart failure, percutaneous cardiac intervention, placement of pacemaker/defibrillator, stroke), 1-year freedom from treated rejection, and 1-year freedom from *de novo* DSA development. The time to DSA development was also assessed.

Results: Patients with PGD had longer ischemic times compared to the control (181.1 ± 58.3 vs 154.8 ± 57.2 min, p=0.005). PGD was associated with reduced 1-year survival, 1-year freedom from *de novo* DSA, antibody-mediated rejection (AMR), and NF-MACE compared to the non-PGD control (see table). These patients were more sensitized prior to HTx and had a greater prevalence of anti-thymocyte globulin induction therapy post-HTx. There was no significant difference in the average time to *de novo* DSA development in the PGD and non-PGD cohorts (4.4 months ± 5.2 vs 4.6 months ± 4.2, p=0.806). Both groups appear to have a greater occurrence of Class II *de novo* DSA upon initial development (71.4% vs 76.9%, p=1.0).

Conclusions: Patients with PGD post-HTX appear to be at greater risk for *de novo* DSA development and AMR. Further studies are warranted with a larger population size and longer follow-up to confirm these results.

Disclosure: Jignesh Patel: Yes;Alexion Pharmaceuticals:Grant:Research;Pfizer:Grant:Research;Alnylam Pharmaceuticals:Grant:Research | Michelle Kittleson: No | Lawrence Czer: Yes;St. Jude Medical:Grant:Research | Tamar Aintablian: No | Tina Kao: No | Dael Geft: No | David Chang: Yes;Abbott Laboratories:Ownership Interest:Other;AbbVie Inc:Ownership Interest:Other;Repligen:Ownership Interest:Other;Teva Pharmaceutical Industries:Grant:Research | Fardad Esmailian: Yes;SynCardia Systems:Grant:Research;TransMedics Inc:Grant:Research;TransMedics Inc:Honoraria:Advisory Committee | Jon Kobashigawa: Yes;Alexion Pharmaceuticals:Consulting Fee:Consulting;CSL Behring:Consulting Fee:Consulting;Novartis:Grant:Research;CareDx Inc:Grant:Research;CareDx Inc:Honoraria:Other

KEYWORDS: heart transplantation, donor-specific antibodies.

TABLE:

Endpoints	PGD (n=42)	No PGD (n=452)	P-Value
1-Year Survival	80.4%	93.1%	0.001
1-Year Freedom from NF-MACE	67.8%	90.3%	<0.001
1-Year Freedom from Any-Treated Rejection	69.0%	85.5%	0.005
1-Year Freedom from Acute Cellular Rejection	87.1%	92.5%	0.332
1-Year Freedom from Antibody-Mediated Rejection	88.3%	96.3%	0.039
1-Year Freedom from Biopsy Negative Rejection	89.8%	94.8%	0.105
1-Year Freedom from *de novo* DSA	78.8%	90.0%	0.042
Pre-Transplant PRA ⇢ 10%	47.6% (20/42)	28.1% (127/452)	<0.001
ATG Induction Therapy	71.4% (30/42)	42.0% (190/452)	<0.001

ABSTRACT #3

TITLE: iPSCs Derived Regulatory Dendritic Cells Induce Murine Cardiac Allografts Acceptance via Generating Donor-specific Regulatory T Cells

AUTHORS (FIRST NAME, LAST NAME): Songjie Cai[2, 1], Jiangang Hou[2, 4], Masayuki Fujino[2, 3], Naotsugu Ichimaru[1], Lina Lu[5], Shiro Takahara[1], Xiao-Kang Li[2]

INSTITUTIONS (ALL): 1. Advanced Technology for Transplantation, Osaka University Graduate School of Medicine, Osaka, Japan.
2. Division of Transplantation Immunology, National Research Institute for Child Health and Development, Tokyo, Japan.
3. AIDS Research Center, National Institute of Infectious Diseases, Tokyo, Japan.
4. Huashan Hospital, Fudan University, Shanghai, China.
5. Department of Immunology, Lerner Research Institute, Cleveland Clinic, Cleveland, OH, United States.

Background: Our group has reported that we successfully generated and characterized DCregs from murine induced pluripotent stem cells (iPSCs). We found that iPS-DCregs could keep in the 'stable immature stage' even under the strong stimulation. Harnessing this characteristic, we hypothesized that donor-type iPS-DCregs expressing donor-antigen worked as a negative vaccine to generate antigen-specific regulatory T cells (Tregs), and induced donor-specific allo-graft acceptance in murine experimental cardiac transplantation.

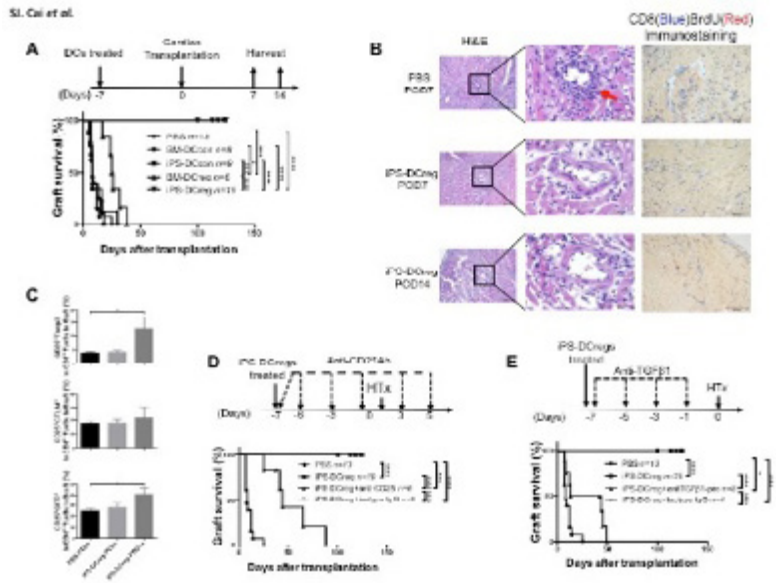
Methods: Recipients (CBA/N, H2K^{k}) were pre-treated with donor-type (C57BL/6, B6, H2K^{b}) iPS-DCregs seven days previous to transplantation. Graft survival and allo-rejection/tolerance associated biomarker were measured.

Results: Recipient pretreated with B6 iPS-DCregs accepted B6 cardiac grafts permanently (**Image A**). The number of graft infiltrating CD8^{+} T cells in iPS-DCregs treated recipients was significantly decreased compared with no-treat group, and the Foxp3^{+} Tregs were significantly increased (**Image B, C**). Further, the naïve secondary recipients (CBA/N) treated with the adoptive transfer of splenocytes from iPS-DCregs treated primary recipients on POD100 accepted B6 cardiac grafts but rejected BALB/c grafts. The above protective effects were reversed by infusion of anti-TGFβ or anti-CD25 mAb (**Image D, E**).

Conclusions: The data indicated that preconditioning of donor-type iPS-DCregs to recipients generated antigen-specific Tregs in association with TGFβ. These findings highlight the iPS-DCregs will be a key cell therapy in clinic transplantation.

Disclosure: Songjie Cai: No | Jiangang Hou: No | Masayuki Fujino: No | Naotsugu Ichimaru: No | Lina Lu: No | Shiro Takahara: No | Xiao-Kang Li: No

KEYWORDS: dendritic cells, donor-specific hyporesponsiveness, acute allograft rejection, CD4 regulatory cells.



(**A**) CBA/N (recipient) preteated with B6 derived iPS-DCregs accepted B6 cardiac allograft. (**B**) Allografts were harvested on POD7 and 14 for H&E and CD8/BrdU immunostaining. (**C**)Graft infiltration lymphocytes (GIL) were isolated and stained with CD4/CD25/Foxp3/CTLA4/GITR for FCM. (**D**) Anti-CD25 mAb and (**E**) anti-TGFβ mAb treatment disrupted iPS-DCregs induced allo-tolerance.

ABSTRACT #4

TITLE: Outcome of Systemic Monitoring of Donor Specific Antibody (DSA) and Protocol Management to Optimize Renal Allograft Outcome in a Single Transplant (Tx) Center

AUTHORS (FIRST NAME, LAST NAME): Shirley Chang[1], Aijaz Gundroo[1], Cindy Yip[1], Diping Wang[4], Michael Williams[2], Indika Mallawaarachchi[3], Alok Dwivedi[3], Sunil Patel[2], Thomas Shanahan[5], Mareena Zachariah[1]

INSTITUTIONS (ALL): 1. Internal Medicine, Univ of Buffalo, Buffalo, NY, United States.
2. Surgery, Univ of Buffalo, Buffalo, NY, United States.
3. Biomedical Sciences, Texas Tech University, El Paso, TX, United States.
4. Pathology, Univ of Buffalo, Buffalo, NY, United States.
5. Immco Diagnostics, Buffalo, NY, United States.

Background: De novo (dn) DSA development has been shown to adversely affect long-term allograft survival. To improve long-term renal graft function, ECMC has taken aggressive approach to systemic screening DSA in post-Tx patients (pts) at 1 month (m), 2 m, 3 m, 6 m, & yearly (yr) intervals. If dn DSA with MFI>3000 present, Tx kidney (K) biopsy (bx) is performed, management decision is based on bx findings. ACR is treated with steroid bolus (2 gm IV total), AMR treated with steroid bolus ± thymoglobulin & plasmapheresis & IVIG. Pts without rejection on bx are treated with IVIG (2 gm/kg), with close DSA follow-up until becomes -(MFI<1500).

Methods: This is a single center retrospective study at Univ. of Buffalo from Jan 2012 to Sept 2016. All 379 K Tx recipients were included, pancreas tx alone pts were excluded. Induction immunosuppressive was Thymo 3 mg/kg, with maintenance of pred/tacrolimus/MMF. DSA monitoring is as above, more frequently if dn DSA +.

Results: 49 recipients developed dn DSA, & 330 did not. Of those who developed dn DSA, 57% were females, 92% had DDKT, 27% developed HLA I DSA, 88% developed HLA II DSA,12% had both HLA I & II DSA. Median onset of dn Class I DSA occurred after 40 days (range 15-642 days) post-Tx, for dn Class II DSA was at 180 days (range 11-1367 days, $p<0.05$). Dn Class II DSA occurred more frequently, with more DR, DRw, & DQ mismatches. 4 pts did not have Tx K Bx at time of dn DSA. Of those who had bx, 36% had ACR, 20% had AMR, 15% had both ACR + AMR and more likely to have both Class I & II dn DSA. 9 pts had graft failure, median onset 2.5 yrs post-tx, and all had HLA II DSA. There were no differences in eGFR in pts with Class I *vs.* II DSA. Pts with higher cPRA developed more HLA I compared to HLA II DSA ($p<0.02$). Comparing pts who developed dn DSA *vs.* those who did not, recipients with dn DSA have higher cPRA ($p<0.001$), & lower EPTS ($p<0.02$). Re-Tx did not play a role in dn DSA development. Serum Cr in those without DSA were lower at 1 m, and 1 yr ($p=0.01$, $p=0.05$), but no different at 2 yr & 3 yr when compared to pts with DSA.

Conclusions: 12.9% Tx K recipients develop dn DSA at ECMC, with earlier DSA development associated with HLA Class I, and later DSA developement with HLA Class II. Recipients who are more sensitized, & younger age were likely to develop dn DSA post-tx; in turn, they tend to have higher serum Cr at 1 m & 1 yr, but the difference no longer present at 2-3 yrs in our tx center with aggressive DSA management.

Disclosure: Shirley Chang: No | Aijaz Gundroo: No | Cindy Yip: No | Diping Wang: No | Michael Williams: No | Indika Mallawaarachchi: No | Alok Dwivedi: No | Sunil Patel: No | Thomas Shanahan: No | Mareena Zachariah: No

KEYWORDS: alloantibodies, allograft function, acute rejection, allograft monitoring.

ABSTRACT #5

TITLE: Discrepant HLA Antibody Reactivity Detected with Different Solid-Phase Luminex-Based Assays

AUTHORS (FIRST NAME, LAST NAME): Andres Jaramillo[1], Lora J. Nelson[1], John Lunz[3], Raymond L. Heilman[2], Hasan A. Khamash[2], Marcelo J. Pando[1]

INSTITUTIONS (ALL): 1. Department of Laboratory Medicine and Pathology, Mayo Clinic Hospital, Phoenix, AZ, United States.
2. Department of Medicine, Mayo Clinic Hospital, Phoenix, AZ, United States.
3. Gift of Hope Organ & Tissue Donor Network, Itasca, IL, United States.

Background: Precise determination of HLA antibodies is essential for patients awaiting transplantation. HLA antigens that are recognized by a patient are listed in UNET as unacceptable antigens (UA) and donors having those antigens are excluded from offering the patient an organ. The use of solid-phase assays (SPA) for detecting HLA antibody has been extremely important in identifying UA and improving organ allocation. However, in some cases, SPA can display non-specific antibody binding that is not

detected in the flow cytometry crossmatch (FCXM). The aim of this study was to compare the different commercial HLA antibody analysis assays to overcome the potential non-specific antibody binding problem in our testing algorithm.

Methods: In our institution, the HLA antibody profile of a patient is determined by means of the LABScreen Single Antigen assays (One Lambda, Inc.) and confirmed with the LABScreen PRA assays (One Lambda, Inc.). Confirmed specificities with MFI > 2000 are listed as UA. For this study, 5 patients were found to have high reactivity by the single antigen bead (SAB) assays but were negative by the PRA assays.

Results: All the patients showed high reactivity to all the HLA-DRB1 antigens and selected HLA class I antigens (Table 1). In order to unquestionably define the presence or absence of HLA antibodies, surrogate FCXM were performed on all patients with strong donor-specific antibodies (DSA) (MFI > 5000) to selected donors. Interestingly, none (0%) of the FCXM (n=50) displayed positive results despite the presence of high DSA levels (Table 1). Subsequently, the patients' HLA antibody profiles were analyzed by means of the LIFECODES LSA Single Antigen assays (Immucor, Inc.). It is noteworthy that the results from these SAB assays were negative in all patients (Table 1).

Conclusions: This study indicates that the use of the LABScreen Single Antigen assay as the sole method for UA determination can lead to a significant disadvantage for patients with non-specific antibody binding. Therefore, the use of 2 different SAB platforms or 2 different bead assays (PRA and SAB) in the HLA antibody testing algorithm would prevent patients displaying non-specific antibody binding from being inappropriately excluded from receiving an organ due to false-positive SPA results.

Disclosure: Andres Jaramillo: No | Lora Nelson: No | John Lunz: No | Raymond Heilman: No | Hasan Khamash: No | Marcelo Pando: No

KEYWORDS: alloantibodies, kidney transplantation, kidney allocation.

TABLE:

	Calculated PRA	SAB Assay					FCXM
		HLA Class I		HLA Class II			
Patient	**One Lambda SAB Assay**	**Immucor SAB Assay**	**No. of Specificities* (One Lambda)**	**No. of Specificities* (Immucor)**	**No. of Specificities* (One Lambda)**	**No. of Specificities* (Immucor)**	**No. of Positive Results**
1	85%	0%	3	0	10	0	0/10 (0%)
2	100%	0%	0	0	17	0	0/15 (0%)
3	96%	0%	4	0	22	0	0/10 (0%)
4	56%	0%	0	0	4	0	0/4 (0%)
5	100%	0%	5	0	22	0	0/11 (0%)

*MFI > 2000

ABSTRACT #6

TITLE: Kidney Transplantation in Octogenarians: The Mayo Clinic Experience.

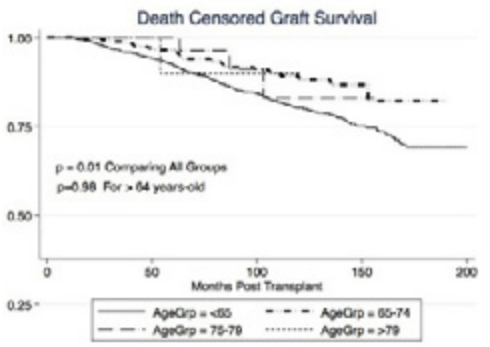
AUTHORS (FIRST NAME, LAST NAME): Nadeen Khoury[1], Hatem Amer[1]

INSTITUTIONS (ALL): 1. Nephrology and Hypertension , Mayo Clinic, Rochester , MN, United States.

Background: Kidney transplantation (KTx) is the preferred renal replacement therapy in selected individuals. There is limited data on transplantation of octogenarians. The purpose of this study was to examine our program's experience transplanting individuals in their 80's.

Methods: We searched the transplant database for patients who were >79 years of age at the time of transplant. End points were death censored graft survival and all cause graft loss. Control groups for survival analysis were patients with age groups <65 years, 65-74 years and 75-79 years. There were 11 transplants performed in octogenarians in our program. As the first octogenarian was transplanted in 2000, we censored transplants before 2000. We excluded all patients with <12 months follow-up.

Results: In our cohort of 2896 transplants, 10 (0.35%) were octogenarians, 43 (1.5%) were [75-79], 473 (16%)[65-74] and 2370 (82%)< 65 years old. Octogenarians were 80-84 years old at the time of transplant. Nine were males and 9 received a living donor KTx. Family members were donors in 7 cases. All recipients had a post graduate degree, 4 of them were physicians. Seven patients received preemptive transplants while the rest were on dialysis for <1 year. Thymoglobulin was used for induction before 2005 and basiliximab thereafter. None of the patients were diabetic. Native kidney disease was hypertensive nephrosclerosis in 9 and reflux nephropathy in one. One recipient had a non-fatal postoperative MI. Mean follow-up was 87 ± 22 [54-121] months. Median post-transplant survival was 87 months. 1 patient lost his graft from transplant glomerulopathy and 5 died from deconditioning and infections. Comparative survival is shown below.



Death Censored Graft Survival.

Conclusions: Our single-center experience shows that KTx can benefit a selected group of octogenarians . Information about good outcomes should be spread to providers and individuals to encourage transplant referrals in this patient population.

Disclosure: Nadeen Khoury: No | Hatem Amer: No

KEYWORDS: kidney graft survival, living donor transplantation.

ABSTRACT#7

TITLE: The Efficacy of the Follow-Up System of Living Kidney Donor in the Monitoring of the Remaining Kidney Function.

AUTHORS (FIRST NAME, LAST NAME): Magdalena Kwapisz[1], Rafal Kieszek[1], Kalina Jedrzejko[1], Jolanta Gozdowska[2], Artur Kwiatkowski[1]

INSTITUTIONS (ALL): 1. Department of General and Transplant Surgery, Medical University of Warsaw, The Infant Jesus Teaching Hospital, Warsaw, Poland.
2. Department of Transplantation Medicine, Nephrology and Internal Diseases, Medical University of Warsaw, The Infant Jesus Teaching Hospital, Warsaw, Poland.

Background: The possibility of an increased risk of end-stage renal disease is considered as a main concern related with living kidney donation. This makes the monitoring of the remaining kidney function the most important purpose of the follow-up system. The long-term safety of nephroureterctomy procedure with the absence of accelerated loss of renal function is generaly expected result.

Methods: The retrospective cohort study including 172 patients (aged 24 – 72; mean 43) that underwent unilateral nephroureterectomy for organ donation in 2003-2016 was conduced. Medical files was analysed in terms of efficacy of the long-term care system in the aspect of monitoring of the remaining kidney function.

Results: The analysed group consisted of 110 (63.9%) women (24 – 72 y of age) and 64 (36.1%) men (25-64 y of age). Mean follow-up period was 5.44 years (range: 6 months – 10 years). Despite of the rise of its value directly after the donation, mean serum creatinine level in all age groups was within the range of the normal (referred to 0.6 – 1.3 mg/dl) during the observation. In 26 cases serum creatinine level above the laboratory testing standard was found (up to 2,2 mg/dl). Mean postdonation MDRD GFR was measured at about 68.3% of predonation value. Despite of its initial declining after nephrectomy, mean GFR remained above 60 ml/min/1.73m2. A MDRD GFR below 60 ml/min/ 1.73m2 was found in 40 donors (23.3%). However, a MDRD GFR below 45 ml/min/1.73m2 was observed only in 7 cases (4%), down to 33 ml/min/1.73m2. During that time, no one developed end-staged renal disease or required dialysis treatment. When GFR was determined based on CKD-EPI equation, only 21.5% of analyzed had a GFR below 60 ml/min/1.73 m2 , down to 33,7 ml/min/1.73 m2 . It was found below 45 ml/min/1.73m2 in 5 cases (2.9%) only. Mean postdonated CKD-EPI estimated GFR was 66 ml/min/1.73 m2 and it was measured at about 69.99% of its predonation value.

Conclusions: An exact qualification process, when carried out properly, minimizes the probability of being a kidney donor by a person with an increased risk of chronic kidney disease. No one has developed end-stage renal failure after donation in evaluated group. Estimated GFR below 60 ml/min/1.73 m2 is a threshold value for detection of chronic kidney failure and when reported by MDRD equation, it may lead to inappropriate diagnosis in some cases. CKD-EPI equation seems to be more accurate than MDRD in people with higher levels of GFR and it should be used for kidney donors, as their low GFR is a result of nephrectomy, not kidney or systemic disease.

Disclosure: Magdalena Kwapisz: No | Rafal Kieszek: No | Kalina Jedrzejko: No | Jolanta Gozdowska: No | Artur Kwiatkowski: No

KEYWORDS: Donor evaluation, living donor transplantation, chronic kidney disease (CKD).

ABSTRACT #8

TITLE: De Novo Malignancies After Living-Donor Liver Transplantation in the University of Tokyo

AUTHORS (FIRST NAME, LAST NAME): Rihito Nagata[1], Nobuhisa Akamatsu[1], Junichi Togashi[1], Sumihito Tamura[1], Junichi Arita[1], Junichi Kaneko[1], Yoshihiro Sakamoto[1], Kiyoshi Hasegawa[1], Norihiro Kokudo[1]

INSTITUTIONS (ALL): 1. Artificial organ and transplant surgery, Tokyo University, Tokyo, Japan.

Background: Back grounds: De novo malignancies are the important cause of death among liver transplant recipients over 3 years after transplantation. The aim of this study was to investigate the de novo malignancies after living-donor liver transplantation (LDLT) in our institute.

Methods: Methods: 467 adult patients who underwent LDLT using cryopreserved homologous veins at the University of Tokyo since 1996 to 2014 were the subjects of the study. The patient who died within 1 year after LDLT (n=46) were excluded, therefore, those who survived more than 1 year (n=421) were retrospectively reviewed. Posttransplant recipients were screened by routine medical checkup and optional surveillance including serum tumor marker, abdominal ultrasound, computed tomography, and gastrointestinal endoscopy to detection of post-transplant malignancy.

Results: Results: Totally 32 cases of de novo malignancies were observed after LDLT. Of these, the largest majority were 15 gastrointestinal (GI) malignancies including colorectal (n=8), stomach (n=5), oral cavity, esophagus (n=1, respectively), followed by 7 hematological malignancy cases. Other malignancy types were skin (n=3), breast (n=3), prostate (n=2), and kidney (n=2). When adjusted with standardized incident ratios (SIR), the trend seen in Japanese nation-wide survey was also seen in our center, as we presented high standardized incident rates related to the sites of esophagus (SIR=16.9) and leukemia (SIR=15.6) . Regarding the treatment for de novo cancers, surgical resection was indicated for 17 (53%) cases. 6 cases of GI malignancies received curative endoscopic resection. Of the 421 recipients, 54 (13%) patients died after 1 years of LDLT, 11 patients died of de novo malignancies and that account for 20% of all cause of death. The 1-, 3-, 5-year overall survival after the diagnosis of de novo malignancies were 95%, 80%, 62% respectively. Meanwhile, 22 (69%) cases were diagnosed without distant metastases by the posttransplant surveillance.

Conclusions: Conclusion: De novo malignancy impairs posttransplant patient outcome, whereas early diagnosis with routine surveillance might improve its prognosis.

Disclosure: Rihito Nagata: No | Nobuhisa Akamatsu: No | Junichi Togashi: No | Sumihito Tamura: No | Junichi Arita: No | Junichi Kaneko: No | Yoshihiro Sakamoto: No | Kiyoshi Hasegawa: No | Norihiro Kokudo: No

KEYWORDS: malignancy, liver transplantation.

ABSTRACT #9

TITLE: Is kidney transplantation (KTX) justified in patients with more than 10 years of pre-KTX dialysis exposure?

AUTHORS (FIRST NAME, LAST NAME): Caren Rose, Jagbir Gill, Julie Lesage, Yayuk Joffres, John S. Gill

Background: The new kidney allocation system led to a sharp increase in KTX among patients with 10 years of pre-KTX dialysis exposure. We undertook the current analysis to determine the survival benefit of KTX in such patients.

Methods: The study cohort consisted of n =2820 prevalent dialysis patients who were wait-listed for KTX and < 75 years of age, 10 years after initiating chronic dialysis treatment between 1998-2000 (the most contemporary cohort that could be assembled in which every patient would have at least 5 years of potential follow-up after the date of their 10 year dialysis anniversary to determine the survival benefit of subsequent KTX).

Survival was determined from the date of each patient's 10 year dialysis anniversary until the date of permanent removal from the waiting list, living donor KTX, death or end of follow up Dec 31, 2015 using a multivariate non-proportional hazards analysis with KTX treated as a time dependent covariate to account for the fact that patients switched treatment from dialysis to KTX at different times during follow-up.

Results: The Table shows the hazard ratio for death in the n=1021 (36%) of study patients who underwent deceased donor KTX after a mean ± STD: 12.5 ± 1.9 years after dialysis initiation compared to the n=1799 patients who remained on dialysis. The median (Q1,Q3) deceased donor kidney profile index (KDPI) of the transplanted kidneys was 45% (26%, 64%).

TABLE:

	% of cohort	HR	95% CI
Deceased donor transplantation	36	0.49	0.41, 0.59
Age (years) after 10 years of dialysis initiation			
<40			
40-49	18	1.00	--
50-59	25	1.55	1.20, 2.00
⇢ 60	30	2.62	2.07, 3.32
	27	3.93	3.10, 4.98
Female Sex	48	1.06	0.95, 1.20
Race			
White (ref)	33	1.00	
Black	59	0.84	0.74, 0.95
Other	8	0.96	0.77, 1.19
Cause of ESRD			
GN (ref)	23	1.00	
Diabetes	26	1.70	1.43, 2.03
Other	51	1.15	0.98, 1.35
Comorbid conditions			
History of CVD (ref none)	12	1.03	0.87, 1.22
History of PVD (ref none)	2	1.22	0.89, 1.68
History of CVA (ref none)	2	1.13	0.78, 1.63

Discussion and Conclusions: Using the most available contemporary cohort of patients with 10 years of pre-KTX dialysis exposure and sufficient potential follow up, deceased donor KTX was associated with a significant survival benefit compared to continued treatment with dialysis. Of note, the KDPI of kidneys transplanted was relatively low but similar to that of recipients with 10 years of pre-KTX dialysis exposure in the post KAS era (KDPI median (Q1,Q3):51 (35,68). Whether recipients of higher KDPI kidneys would derive a similar survival benefit remains uncertain. We conclude that KTX in patients with 10 years of pre-KTX exposure is justifiable on the basis of a survival benefit.

ABSTRACT #10

TITLE: The OPO Offer Tool(TOOT) Developed to Help Transplant Centers Monitor the Organ Offers

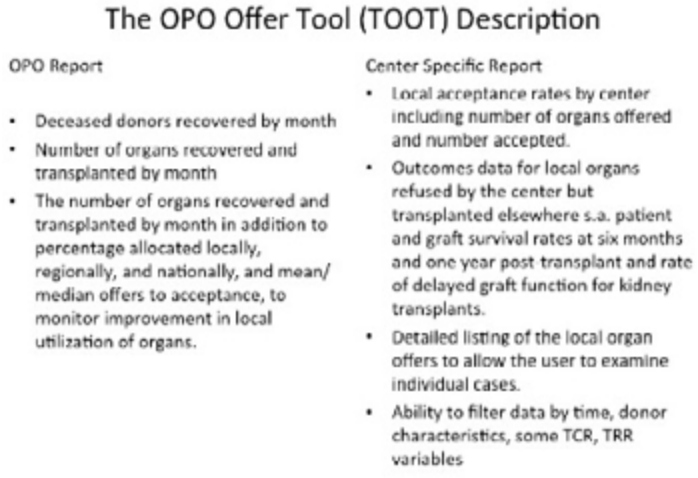
AUTHORS (FIRST NAME, LAST NAME): Fernando Torres[2], Matthias Peltz[3], Yvette Chapman[1], Patti Niles[1]

INSTITUTIONS (ALL): 1. Southwest Transplant Alliance, Dallas, TX, United States.
2. Pulmonary and Critical Care,, UT Southwestern Medical Center,, Dallas, TX, United States.
3. Cardio Thoracic Surgery,, UT Southwestern Medical Center, Dallas, TX, United States.

Background: Organ transplantation continues to grow in the United States, but is outpaced the growing demand for transplantation. Organ utilization rates vary among organ procurement organizations (OPOs) and transplant centers. For years, transplant centers have asked OPOs about the outcomes of organs they decline. We hypothesized that providing local centers with outcomes data for declined organs that were transplanted elsewhere would influence acceptance behavior and increase local organ recovery rates.

Methods: We contracted with the business analytics arm of UNOS to build a Tableau workbook that reviews offers and outcomes for deceased donor organs recovered for transplantation by specified OPOs. The OPO Local Offer Tool (TOOT) was used to provide transplant centers within our OPO 24 month outcome data on organs declined by local centers that were ultimately

transplanted as shared organs elsewhere. Subsequently, we will prospectively monitor local and shared organ transplantation rates to assess the influence of center specific outcomes data reports of rejected organs on local center donor acceptance rates for all solid organs. Only organs declined for donor or organ quality were considered (Codes 830, 837).



Results: We will report historical local transplantation rates, shared transplantation rates, and discarded organs. These data will be compared to utilization and discard rates after implementation of the tool. De-identified local center specific behavior data will be reviewed.

Conclusions: TOOT provides transplant centers with outcomes data for organs declined at their center. We believe that centers with high survival rates of organs from donors they declined will alter their approach to donor selection and increase local transplantation rates. TOOT, if applied nationally, may significantly increase the number of organs transplanted and limit discarding otherwise suitable organs.

Disclosure: Fernando Torres: No | Matthias Peltz: No | Yvette Chapman: No | Patti Niles: No

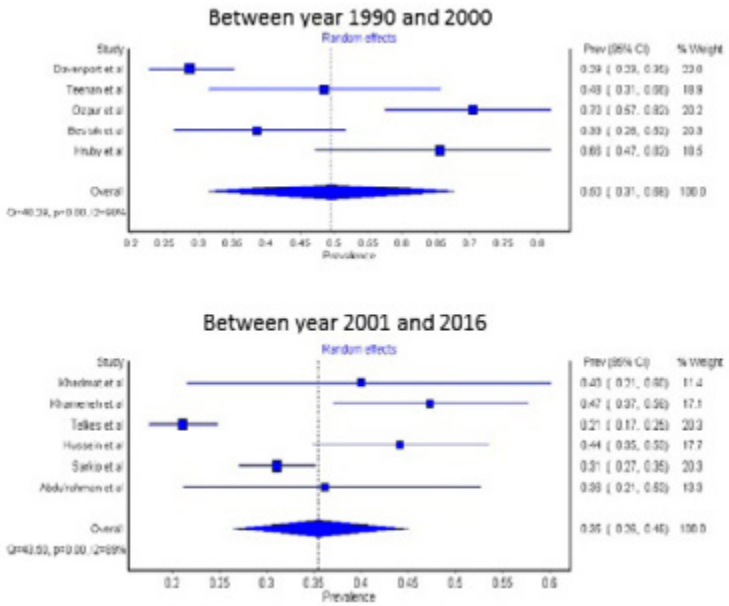
KEYWORDS: Donor evaluation, long-term outcomes.

ABSTRACT #11

TITLE: Helicobacter Pylori Infection in Kidney Transplant Recipients: A Meta-Analysis

AUTHORS (FIRST NAME, LAST NAME): Wisit Cheungpasitporn[1,3], Charat Thongprayoon[2], Michael Mao[1], Karn Wijarnpreecha[2], Donald Mitema[1,3], Stephen B. Erickson [1]

INSTITUTIONS (ALL): 1. Nephrology and Hypertension, Mayo Clinic, Rochester, MN, United States.
2. Internal Medicine, Bassett Medical Center, Cooperstown, NY, United States.
3. William J. von Liebig Transplant Center, Mayo Clinic, Rochester, MN 2Department of Laboratory Medicine and Pathology, Mayo Clinic, Rochester, MN, United States.



The estimated prevalence of *H. pylori* among kidney transplant patients.

Background: The aims of this study were 1) to examine the prevalence of *Helicobacter pylori* (*H. pylori*) infection in kidney transplant recipients and 2) assess the risk of *H. pylori* infection in kidney transplant recipients compared with non-transplant patients.

Methods: A comprehensive literature search was performed from inception until September 2016. Studies that reported prevalence, relative risks, odd ratios, hazard ratios or standardized incidence ratio of *H. pylori* among kidney transplant recipients were included. Pooled risk ratios and 95%CI was calculated using a random-effect model. Adjusted point estimates from each study were combined by the generic inverse variance method of DerSimonian and Laird.

Results: Eleven observational studies with 2,545 kidney transplant recipients met all inclusion criteria and were enrolled in this study. From study periods between year 1990 and 2000, the estimated prevalence of *H. pylori* among kidney transplant patients was 50% (95%CI: 31%-68%) [Figure], with a prevalence of 46% (95%CI: 23%-70%) in high-income countries and 55% (95%CI: 22%-86%) in middle-income countries, respectively. From year 2001 through 2016, the estimated prevalence of H. pylori among kidney transplant patients was 35% (95%CI: 26%-45%), with a prevalence of 28% (95%CI:

19%-37%) in high-income countries and 45% (95%CI: 38%-51%) in middle-income countries. Data regarding prevalence of *H. pylori* infection in low-income countries were limited. The pooled RR of *H. pylori* infection in kidney transplant recipients was 0.57 (95%CI: 0.33-1.00) when compared with the non-transplant patients.

Conclusions: There was a decline in prevalence of *H. pylori* in kidney transplant recipients in both high-income and middle-income countries with time. The overall estimated prevalence of *H. pylori* in kidney transplant recipients is 35%. In addition, our meta-analysis demonstrates a potential decreased risk of *H. pylori* infection in kidney transplant recipients compared with non-transplant populations.

Disclosure: Wisit Cheungpasitporn: No | Charat Thongprayoon: No | Michael Mao: No | Karn Wijarnpreecha: No | Donald Mitema: No | Stephen Erickson : No

KEYWORDS: kidney transplantation, infectious diseases, post transplant infections, racial and ethnic disparities.

ABSTRACT #12

TITLE: Association Between Urinary Retention and Polyomavirus (BK) Infection in Kidney Transplant Recipients

AUTHORS (FIRST NAME, LAST NAME): Wisit Cheungpasitporn[1, 2], Hatem Amer[1, 2], Fernando G. Cosio[1, 2], Carrie A. Schinstock[1, 2]

INSTITUTIONS (ALL): 1. Nephrology and Hypertension, Mayo Clinic, Rochester, MN, United States.
2. William J. von Liebig Transplant Center, Mayo Clinic, Rochester, MN 2Department of Laboratory Medicine and Pathology, Mayo Clinic, Rochester, MN, United States.

Background: BK nephropathy is a major risk factor for allograft dysfunction and loss. BK viuria and viremia are early markers of BK nephropathy in kidney transplant recipients.

Methods: We performed a retrospective cohort study of solitary kidney transplant recipients from 10/2007 - 5/2014 to examine the association between history of urinary retention (post-void residuals >200cc) and BK viuria, viremia, and nephropathy. All kidney transplant patients at our center had post-void residuals tested by ultrasound or urinary catheter when they had iothalamate renal clearance testing.

Results: Of 962 kidney transplant recipients, 49.7% had BK viruria, 29.5% had BK viremia, 4.5% had BK nephropathy and 24.9% had urinary retention during mean follow-up of 4.1+/-1.9 years post-transplant. BK viuria was associated with urinary retention (30.5% in BK viuria vs 19.4% no BK viuria, p<0.001). Peak blood BK PCR was also higher in those with urinary retention [mean+/-SD of 113,955+/-317,511 vs 37,746+/-105,661 copies, p=0.04]. Patients with BK viremia and BK nephropathy also had numerically higher rates of urinary retention, but this did not reach statistical significance.

Conclusions: Kidney transplant recipients with urinary retention are more likely to develop BK viuria and a higher degree of BK viremia than those without urinary retention. Therefore, urinary retention may be a potentially modifiable risk factor for BK nephropathy and potential allograft loss.

Disclosure: Wisit Cheungpasitporn: No | Hatem Amer: No | Fernando Cosio: No | Carrie Schinstock: No

KEYWORDS: BK virus, risk factors, kidney transplantation, infectious diseases.

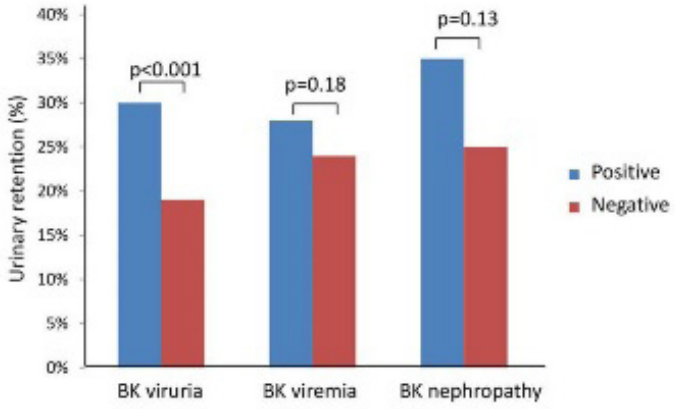


Figure 1. Rate (%) of Urinary Retention by Status of BK Viruria, Viremia, and Nephropathy.

Figure 1. Rate (%) of Urinary Retention by Status of BK Viruria, Viremia, and Nephropathy.

ABSTRACT #13

TITLE: Outcomes of Renal Transplantation in Patients with Previous Hematologic Malignancies

AUTHORS (FIRST NAME, LAST NAME): Jessica Hedvat[1], Leandra Miko[1], Andrew Santeusanio[1], Madhav Menon[1], Vinay Nair[1]

AUTHORS/INSTITUTIONS: J. Hedvat, L. Miko, A. Santeusanio, M. Menon, V. Nair, Mount Sinai Hospital, New York, New York, UNITED STATES

Background: Recommendations regarding the appropriateness of organ transplantation in patients with prior hematologic malignancies are limited given the lack of available data. Based on retrospective studies, a minimum waiting period of 2 to 5 years post-remission is currently suggested. Further studies are required to better assess which patients with prior hematologic malignancies will maximally benefit from renal transplant.

Methods: This was an IRB-approved, single center, retrospective study of adults who received renal transplants between 1/2009 and 1/2016 with a prior diagnosis of multiple myeloma (MM), leukemia, lymphoma, light chain deposition disease, amyloidosis, or myeloproliferative disorder. The primary endpoint was the incidence of malignancy 1-year post-transplant.

Results: Ten patients were identified for inclusion; 6 received chemotherapy and 5 had a previous hematopoietic stem cell transplant (HSCT). Median age at time of transplant was 58 years. Median waiting time post-remission was 2.6 years, and median follow-up time post-transplant was 2.7 years.

Five patients received lymphocyte depleting induction, 4 received IL-2 inhibitor therapy, and 1 received no induction. All patients received tacrolimus and mycophenolate, and 6 received steroids as part of maintenance. Overall 1-year patient and graft survival were 100% and 90% respectively, with one episode of acute cellular rejection that did not result in graft loss. Eight patients had bacterial, 2 had viral, and 2 had fungal infections within 1-year post-transplant. Two patients were diagnosed with new malignancies; both died from complications of cancer with functioning allografts (Table 1). There was suspicion for recurrent malignancies in 2 patients based on concerning laboratory values, but with equivocal cytopathology. All 4 of these patients received chemotherapy and HSCT prior to renal transplant.

Conclusions: In this review, 4/10 patients developed new/recurrent concern for malignancies and 2/10 patients died from cancer-related complications 2.8 years post-transplant. Although this raises some concerns, it is unclear whether the new malignancies were associated with the patients' history of cancer. Despite concerns for recurrence in 2 patients, they remain alive with functioning allografts 3.2 and 2.7 years post-transplant. Future studies should attempt to clarify the role of transplant in patients with prior chemotherapy and HSCT and the need for lymphocyte depleting induction in these immunologically complex patients.

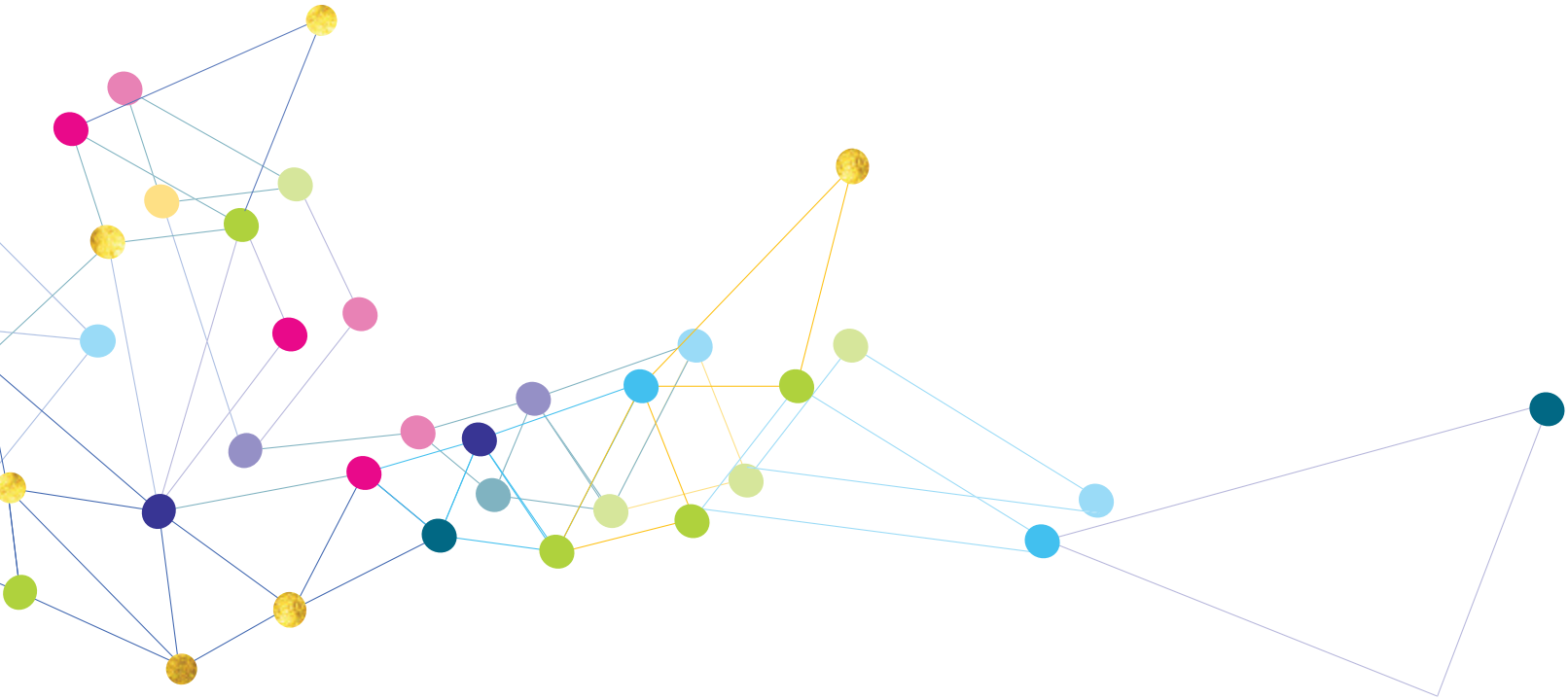


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TABLE:

Patient	Malignancy	Time from hematologic remission to renal transplant (years)	Recurrence	New malignancy	Time to new malignancy (years)	Induction	Outcome
1	MM	3.8	No	Yes (pancreatic cancer)	2.4	Thymoglobulin	Died 2.5 years posttransplant, SCr 1.4 mg/dL
2	Acute lymphocytic leukemia	1.9	No	Yes (acute myeloid leukemia)	2	Daclizum ab	Died 3.2 years posttransplant, SCr 1.2-1.8 mg/dL
3	MM	1.9	Concern for recurrence 2.5 years postrenal transplant	No	N/A	Basilixim ab	Alive 3.2 years posttransplant, SCr 1.3 mg/dL
4	Light chain (AL) amyloidosis	3.3	Concern for recurrence 1.7 years postrenal transplant	No	N/A	Thymoglobulin	Alive 2.7 years posttransplant, SCr 1.8 mg/dL

ABSTRACT #14

TITLE: Results of the usage of biological mesh in the repair of complex abdominal wall defects in patients posttransplantation and general surgery. The feasibility of using MRI in follow up.

AUTHORS (FIRST NAME, LAST NAME): Kalina Jedrzejko[1]

AUTHORS/INSTITUTIONS: K. Jedrzejko, General and Transplantation Surgery, Baby Jesus Clinical Hospital in Warsaw, Warsaw, POLAND

Background: Immunosuppressive therapy, inflammation, and surgical site or general infection make the use of traditional methods for abdominal wall closure ineffective and preclude the application of synthetic mesh. In such difficult cases, the utilization of the biological material, such as PermacolTM, a porcine acellular dermal collagen implant, may be a solution. The purpose of this study was to analyze the use of biological mesh in patients in whom the closure of abdominal wall defect with other methods was not possible and to compare outcomes in patients after transplantation and general surgery.

Methods: The study group consisted of 14 patients, including 6 patients after transplantation. The evaluation of wound healing was based on a clinical examination and in selected patients on magnetic resonance imaging (MRI).

Retrospectively, we analysed data as follows: age, sex, the main reason for the usage of the biological implant, comorbidities, procedural details (the size of the implant, technique of the procedure, the type of the implant), type of bacteria (if infected). Follow-up period ranged from 6 to 32 months.

Results: Only 3 uneventful outcomes were observed. None of the patient had hernia recurrence during the follow-up period. Serum leakage from the wound (8 cases), wound dehiscence (5) that required secondary subcutaneous tissue and skin sewing, and surgical site infection (4 patients) were the most common complications post biological mesh implementation. There were no statistical differences between the groups as long as the complications are concerned. Magnetic resonance imaging (MRI) that is an excellent examination however it should not be considered as an examination of choice in such cases.

Conclusions: Biological meshes may be used in patients in whom other ways of treatment had failed; still a prolonged time of wound healing should be expected. It seems, that it is safe to use PermacolTM in post transplantational patients. Implanting PermacolTM and negative pressure wound therapy can be combined. The way of treatment in such complicated cases should be considered individually. More trials, with bigger group of patients should be performed.

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ABSTRACT #15

TITLE: Immunosuppressive Ability of Pancreas Derived Mesenchymal Stem Cells

AUTHORS (FIRST NAME, LAST NAME): Mazhar A. Kanak[1], Faisal Kunnathodi[2], Marlon F. Levy[1], Michael C. Lawrence[2], Bashoo Naziruddin[3]

INSTITUTIONS (ALL): 1. Surgery, Virginia Commonwealth University, Richmond, VA, United States.
2. Baylor Research Institute, Dallas, TX, United States.
3. Simmons Transplant Institute, Baylor Scott and White Health, Dallas, TX, United States.

Background: The introduction of a steroid-free protocol by the Edmonton group revived islet transplantation therapy for Type-1 Diabetic patients. However, long-term islet dysfunction remains a challenge. Immunosuppression used after transplantation contribute to islet graft failure. Islet transplantation requires alternative approaches to improve immunosuppression. We hypothesized that remnant tissue after islet isolation could be used to recover MSCs. The purpose of this study is to determine if pancreas-derived MSCs have the capacity to be used as immunosuppression during islet co-transplantation.

Methods: Pancreatic tissue from COBE bag remnants was cultured in RPMI medium and plastic-adherent cells were isolated and identified as MSCs by cell morphology and surface marker expression. MSCs (2x10^5 cells) were cultured in 24 well plates overnight. T cells from human PBMCs were stained with 10 uM CFSE for 10 mins and then stimulated with CD3/CD28 beads and cultured with or without MSCs for 5 days. Cells were co-cultured either in direct contact or separated by a transwell membrane of 1um pore size. T Cells were separated from beads and analyzed by flow cytometry for proliferation.

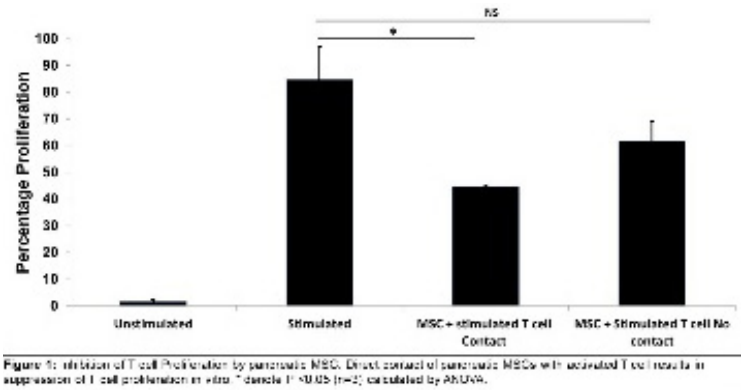


Figure 1: Inhibition of T cell Proliferation by pancreatic MSC. Direct contact of pancreatic MSCs with activated T cell results in suppression of T cell proliferation in vitro. * denote P <0.05 (n=3) calculated by ANOVA.

Results: The culture of remnant tissue after islet isolation resulted in plastic-adherent cells of fibroblast-like morphology after 3 passages. Flow cytometry analysis showed homogeneous expression of markers (CD90, CD73, CD29, CD105) and negative expression of CD14, CD34, and CD45 as classically characterized by cultured MSCs. Co-culture of pancreas-derived MSCs and human T cells stimulated with CD3/CD28 beads resulted in suppression of T cell proliferation in vitro. Moreover, cell-cell contact of MSCs with activated T cells was required for effective suppression of T cell proliferation [Figure 1].

Conclusions: Pancreas-derived MSCs significantly inhibited T cell proliferation demonstrating immunosuppressive properties by direct contact with activated T cells. Co-transplantation models should be further studied to assess the true potential of an MSC-derived immunosuppressive strategy in allogenic islet cell transplants.

Disclosure: Mazhar Kanak: No | Faisal Kunnathodi: No | Marlon Levy: No | Michael Lawrence: No | Bashoo Naziruddin: No

KEYWORDS: immunosuppression, pancreatic islet transplantation, stem cells, T-cell activation.

ABSTRACT #16

TITLE: Immunosuppressive Role of Withaferin A: An NFkB Inhibitor

AUTHORS (FIRST NAME, LAST NAME): Mazhar A. Kanak[1], Michael C. Lawrence[2], Marlon F. Levy[1], Bashoo Naziruddin[3]

INSTITUTIONS (ALL): 1. Surgery, Virginia Commonwealth University, Richmond, VA, United States.
2. Baylor Research Institute, Dallas, TX, United States.
3. Simmons Transplant Institute, Baylor Scott and White Health, Dallas, TX, United States.

Background: The advent of steroid-free protocol by the Edmonton group encouraged islet transplantation as a therapy to cure type-1 Diabetes. However, long-term dysfunction in islet transplantation is a major challenge that needs to be addressed. Immunosuppressive drugs used currently are toxic to the beta cells resulting in graft loss. Withaferin A (WA) is a plant-derived molecule which has been used as an anti-inflammatory molecule because of its ability to block Nuclear Factor Kappa B (NFkB) pathway. Since immune cell activation is combinedly regulated by NFAT, AP-1, and NFkB, we hypothesized that WA can be used to suppress this activation. The purpose of this study is to demonstrate the immunosuppressive ability of WA *in vitro*.

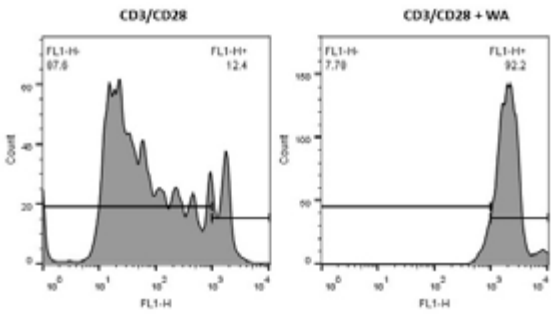


Figure 1: Human T cell proliferation inhibited by Withaferin A: CFSE stained T cells proliferated after stimulation with CD3/CD28 beads for 5 days. Presence of WA significantly abolished proliferation of T cells. Percentage of proliferated and unproliferated T cells shown on top left and top right corners respectively.

Methods: Human and mouse islets were cultured with or without WA at various concentrations and viability was measured to demonstrate safety. Splenocytes were extracted from C57BL/6 mice and stained with 10uM CFSE for 10 mins. Stained cells were stimulated with equal numbers of CD3/CD28 beads in the presence or absence of 1ug/mL WA and cultured for 5 days. T cell proliferation and surface marker analysis for CD4, CD8 and CD25 was determined using flow cytometric analysis. Experiments were also repeated using purified mouse T cells from spleen and human T cells purified from PBMCs.

Results: There was no significant change in the viability of islets up to a concentration of 1ug/ml WA. Flow cytometric analysis revealed that the proliferation of T cells was significantly inhibited by WA in mouse splenocytes, purified T cells and human T cells [Figure 1]. Further analysis of markers revealed a significant reduction in CD4 and CD8 T cell counts.

Conclusions: Low dose of WA showed no toxic effect on Islets. T cell activation was significantly inhibited by WA. The use of WA with no beta cell toxicity as an immunosuppressive agent in islet transplantation may result in promising outcomes.

Disclosure: Mazhar Kanak: No | Michael Lawrence: No | Marlon Levy: No | Bashoo Naziruddin: No

KEYWORDS: pancreatic islet transplantation, immunosuppression, T-cell activation, long-term outcomes.

ABSTRACT #17

TITLE: Photopheresis for Recalcitrant Rejection After Heart Transplantation: Worthwhile?

AUTHORS (FIRST NAME, LAST NAME): Jignesh Patel[1], Michelle Kittleson[1], Xiaohai Zhang[1], Ellen Klapper[1], Tamar Aintablian[1], Kellee Murayama[1], Lawrence Czer[1], Dael Geft[1], David H. Chang[1], Jon Kobashigawa[1]

INSTITUTIONS (ALL): 1. Heart Institute, Cedars Sinai Medical Center, Los Angeles, CA, United States.

Background: Rejection is the leading cause of mortality after heart transplantation (HTx). While the rates of rejection have declined over the past few decades from improvement in immunosuppression, recalcitrant rejection still poses as a major problem for HTx recipients with high mortality. Photopheresis (Pph) is an immunomodatory therapy which involves treatment of leukocytes with psoralen and ultraviolet light. This therapy has been shown to be effective in patients with acute cellular rejection (ACR), antibody-mediated rejection (AMR), and biopsy negative rejection (BNR). We sought to establish the effectiveness of this immunosuppressive modality in our single center.

Methods: Between 2010 and 2015 we assessed 458 HTx patients and found 17 patients who were treated with Pph for severe/recurrent rejection. Pph was administered for 2 consecutive days, weekly x4, and monthly x5. Patients were assessed for cardiac dysfunction and PRA pre-therapy and post-therapy. Also assessed was subsequent 1-year survival, 1-year freedom from rejection (ACR, AMR, BNR), and cardiac allograft vasculopathy as defined by stenosis ⇢ 30% by angiography.

Results: For 17 patients treated with Pph, average time from Htx was 14 ± 10 months. 35% of patients had elevated Class I PRA prior to HTx, with a change from 43 ± 40% pre-Pph to 21 ± 37% post-Pph (p=0.339). 53% of patients who had elevated Class II PRA prior to HTx had a trend from 66 ± 27% pre-Pph lowered to 22 ± 37% post-Pph (p=0.058). (See figure). There was no significant difference before and after therapy in LVEF (43 ± 14% vs 44 ± 16%, p=0.742). No patients developed ACR at subsequent 1-year post-Pph and 1-year freedom from AMR (94%) and BNR (79%) were less than pre-Pph. Subsequent 1-year post-treatment survival and 1-year freedom from CAV were not unexpected (see table).

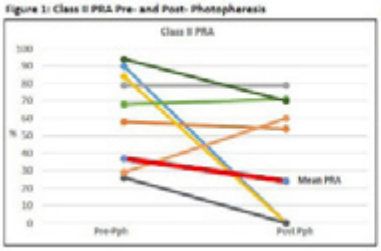


Table 1

Endpoints	n=17
Average Time of Pph Initiation from Transplant, Mean Months ± SD, Median	13.8 ± 10.1, 12.8
Subsequent Actuarial 1-Year Survival	71.7%
Subsequent Actuarial 1-Year Freedom from CAV	93.7%
Subsequent Actuarial 1-Year Freedom from NF-AMVT	93.9%
Subsequent Actuarial 1-year Freedom from Any-Treated Rejection	72.3%
Subsequent Actuarial 1-Year Freedom from Acute Cellular Rejection	100.0%
Subsequent Actuarial 1-Year Freedom from Antibody-Mediated Rejection	94.1%
Subsequent Actuarial 1-Year Freedom from Biopsy Negative Rejection	79.3%

Conclusions: Pph for severe/recurrent rejection is associated with reasonable survival and appears effective at suppressing cellular/humoral responses. Further studies are warranted with a larger population size and longer follow-up to confirm these results.

Disclosure: Jignesh Patel: Yes;Alexion Pharmaceuticals: Grant:Research;Pfizer:Grant:Research;Alnylam Pharmaceuticals:Grant:Research | Michelle Kittleson: No | Xiaohai Zhang: No | Ellen Klapper: No | Tamar Aintablian: No | Kellee Murayama: No | Lawrence Czer: Yes;St. Jude Medical:Grant:Research | Dael Geft: No | David Chang: Yes;Abbott Laboratories:Ownership Interest:Other;AbbVie Inc:Ownership Interest:Other;Repligen:Ownership Interest:Other;Teva Pharmaceutical Industries:Grant:Research | Jon Kobashigawa: Yes;Alexion Pharmaceuticals:Consulting Fee:Consulting;CSL Behring:Consulting Fee:Consulting;Novartis:Grant:Research;CareDx Inc:Grant:Research;CareDx Inc:Honoraria:Other

KEYWORDS: heart transplantation, immunosuppression.

ABSTRACT #18

TITLE: Outcome of Heart Transplant Patients with Rapidly Progressive Cardiac Allograft Vasculopathy

AUTHORS (FIRST NAME, LAST NAME): Jignesh Patel[1], Michelle Kittleson[1], Babak Azarbal[1], Tamar Aintablian[1], Genevieve Rodriguez[1], Michele Hamilton[1], Dael Geft[1], David H. Chang[1], Lawrence Czer[1], Alfredo Trento[1], Jon Kobashigawa[1]

INSTITUTIONS (ALL): 1. Heart Institute, Cedars Sinai Medical Center, Los Angeles, CA, United States.

Background: Cardiac allograft vasculopathy (CAV) is a heterogeneous disease which can be quite indolent and quite rapid in progression. It is not known what mechanism underlies this rapidly progressive disease. Therefore, we sought to evaluate these patients whose underlying CAV progressed within 6 months after the sentinel coronary angiogram which revealed CAV for the first time.

Methods: Between 2009 and 2013 we assessed 517 heart transplant patients of which 92 had established CAV. Eight out of 92 patients had rapidly progressive CAV. These patients were assessed for risk factors including sensitization, diabetes, age of the donor, history of CMV and first year rejection episodes. Endpoints included 1-year survival, 1-year freedom from Non-Fatal Major Adverse Cardiac Events (NF-MACE: myocardial infarction, new congestive heart failure, percutaneous coronary intervention, implantable cardioverter defibrillator/pacemaker implant, stroke) and 1-year freedom from *de novo* donor-specific antibodies (DSA).

Results: The 8 patients with rapidly progressive CAV had a similar percentage of circulating DSA compared to those 84 patients with non-rapidly progressive CAV. One-year survival was significantly less in the rapidly progressive CAV group but NF-MACE was similar between groups. There were no significant differences in demographics.

Conclusions: Heart transplant patients who develop rapidly progressive CAV have worse survival and do not appear to have more circulating DSA. Further investigation into the mechanisms of rapidly progressive CAV may be important in reducing its development.

Disclosure: Jignesh Patel: Yes;Alexion Pharmaceuticals:Grant:Research;Pfizer:Grant:Research;Alnylam Pharmaceuticals:Grant:Research | Michelle Kittleson: No | Babak Azarbal: No | Tamar Aintablian: No | Genevieve Rodriguez: No | Michele Hamilton: Yes;St. Jude Medical:Consulting Fee:Other;Abbott Laboratories:Consulting Fee:Consulting | Dael Geft: No | David Chang: Yes;Abbott Laboratories:Ownership Interest:Other;AbbVie Inc:Ownership Interest:Other;Repligen:Ownership Interest:Other;Teva Pharmaceutical Industries:Grant:Research | Lawrence Czer: Yes;St. Jude Medical:Grant:Research | Alfredo Trento: No | Jon Kobashigawa: Yes;Alexion Pharmaceuticals:Consulting Fee:Consulting;CSL Behring:Consulting Fee:Consulting;Novartis:Grant:Research;CareDx Inc:Grant:Research;CareDx Inc:Honoraria:Other

KEYWORDS: heart transplantation, allograft vasculopathy.

TABLE:

Endpoints	Non-Rapid CAV (n=84)	Rapid CAV (n=8)	P-Value
Subsequent 1-Year Actual Survival	94.9%	75.0%	0.025
Subsequent 1-Year Actual Freedom from NF-MACE	88.0%	87.5%	0.970
Subsequent 1-Year Actual Freedom from *de novo* DSA	100.0%	100.0%	1.000

ABSTRACT #19

TITLE: A Comparison of Driveline Infections: Left Ventricular Assist Device Vs. Total Artificial Heart

AUTHORS (FIRST NAME, LAST NAME): Carmelita Runyan[1], Heather Barone[1], Newman Huie[1], Jennifer Hajj[1], Rhodora Jocson[1], Lee Lam[1], Elizabeth Passano[1], Lawrence Czer[1], Jon Kobashigawa[1], Jaime Moriguchi[1], Francisco Arabia[1]

INSTITUTIONS (ALL): 1. Heart Institute, Cedars-Sinai Medical Center, Los Angeles, CA, United States.

Background: Mechanical Circulatory Support (MCS) driveline infections (DLI) remain one of the major limiting factors to successful long term support after MCS implantation. There are limited data specifically examining the incidence and predictors of driveline infections across devices. Does the larger cannula size and presence of two drivelines (pneumatic tubes) equate to a higher infection rate in the total artificial heart population? We sought to answer this question by evaluating our MCS patients for driveline infections and comparing those with a Left Ventricular Assist Device (LVAD) to those with a Total Artificial Heart (TAH).

Methods: Between 2012 and 2016 we evaluated 192 MCS patients. Driveline infections were identified and these patients were divided into 2 groups by device type (LVAD or TAH). Outcomes included driveline infection rates, mean length of support, days from implant to first infection, pre-implant infection and diabetes, unplanned readmissions, dressing change compliance, and days from first infection to transplant in patients who were transplanted.

Results: We identified 8 LVAD patients with 11 driveline infections and 4 TAH patients with 7 driveline infections. Pre-implant diabetes and pre-implant infection do not offer clear indicators of increased risk. Non-compliance in driveline management suggests a greater risk for driveline infections. There was no difference in the average number of days to first infection between these groups. Of the 7 transplant patients, TAH patients were transplanted an average of 100 days sooner than LVAD patients.

Conclusions: The large cannula size and the presence of two pneumatic drivelines in patients with a TAH does not lead to a higher driveline infection rate.

Disclosure: Carmelita Runyan: No | Heather Barone: No | Newman Huie: No | Jennifer Hajj: No | Rhodora Jocson: No | Lee Lam: No | Elizabeth Passano: No | Lawrence Czer: Yes;St. Jude Medical:Grant:Research | Jon Kobashigawa: Yes;Alexion Pharmaceuticals:Consulting Fee:Consulting;CSL Behring:Consulting Fee:Consulting;Novartis:Grant:Research;CareDx Inc:Grant:Research;CareDx Inc:Honoraria:Other | Jaime Moriguchi: Yes;HeartWare International Inc:Grant:Research;Thoratec:Grant:Research | Francisco Arabia: Yes;SynCardia Systems:Consulting Fee:Consulting

KEYWORDS: heart preservation, long-term outcomes.

TABLE:

Endpoints	LVAD with DLI (n=8)	TAH with DLI (n=4)
DLI Rate	7.0% (8/115)	5.2% (4/77)
Days of Support, Mean ± SD	752.1, 358.6	575.5, 187.7
Days from Implant to First DLI, Mean ± SD	365.25, 327.31	333.50, 192.83
Days from First DLI to Transplant, Mean ± SD	363.50, 222.34 (n=4)	263.00, 219.06 (n=3)
Diabetes Pre-Implant, %	12.5	25.0
Infection Pre-Implant, %	12.5	25.0
Unplanned Readmission Rate (per 100 pt months)	21.7	14.5
Non-Compliant with Dressing Changes, %	75.0	25.0

ABSTRACT #20

TITLE: Race, Admissions, and Renal Transplant Waitlisting

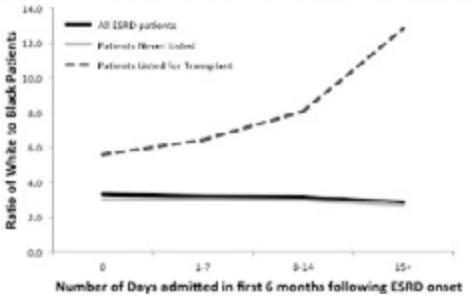
AUTHORS (FIRST NAME, LAST NAME): Raymond J. Lynch[1], Rebecca Zhang[1], Andrew Adams[1], Rachel Patzer[1]

INSTITUTIONS (ALL): 1. Surgery, Emory University, Atlanta, GA, United States.

Background: We have previously shown that increased hospitalization while listed for renal transplantation is associated with excess waitlist mortality, reduced odds of transplantation, and inferior recipient and graft survival. We sought to determine whether patterns of baseline admissions might be used to identify incident ESRD patients with potential for good waitlist and transplant outcomes.

Methods: We used United States Renal Data Systems files to perform a retrospective review of all adult new-onset ESRD cases, limited to individuals with primary Medicare coverage over the first 6 months after ESRD onset to capture number of days spent inpatient. Socioeconomic status was approximated by ZIP code poverty rate. Cox multivariable analysis was performed to determine impact of demographics, comorbidities and hospitalization history on survival and successful transplant listing.

Figure 1: Ratio of White to Black Patients by Eventual Listing Status and Number of Days Hospitalized in First Six Months Following ESRD Onset:



Results: A total of 470,084 patients were studied. EPTS score at ESRD onset was lower in black than white patients, and higher among those in poorer areas. The overall rate of waitlisting was 11.8% for white and 5.3% for black patients. Baseline admissions reduced likelihood of waiting approximately equally by race, but the ratio of white to black patients listed was significantly greater among more-heavily admitted groups (figure 1). In multivariable Cox modeling, >20% poverty by ZIP code was associated with decreased likelihood of waitlisting (HR 0.754, 95% CI 0.734-0.776). Black patients who were never listed had superior survival to white patients at each level of admissions (3 year survival 75.2% vs. 67.2% for non-admitted patients, 53.2% vs. 45.9% for patients with 15+ days inpatient, $p < 0.0001$ for both comparisons). Excess admissions significantly reduced likelihood of listing (HR for 15+ days 0.753 (95%CI 0.732-0.774)) and increased likelihood of death among non-listed patients (HR 1.58 for 15+ days (95% CI 1.57-1.60)).

Conclusions: Black ESRD patients are less likely to be waitlisted than whites, especially in the setting of high baseline admissions. Use of admissions as a candidate performance metric could improve disparities in access to the waitlist for black patients and extend the utility of the organ supply.

Disclosure: Raymond Lynch: No | Rebecca Zhang: No | Andrew Adams: No | Rachel Patzer: No

KEYWORDS: kidney allocation, race.

ABSTRACT #21

TITLE: Development of an evidence-based video series to improve education before kidney transplant

AUTHORS (FIRST NAME, LAST NAME): Holly Mansell[1], Nicola Rosaasen[2], Rahul Mainra[1, 2], Azaad Kukha-Bryson[3], Ahmed Shoker[1, 2], Dave Blackburn[1], Jay Wilson[1], Maya Obadia[4], Gail MacKay[1], Paraag Trivedi[5]

INSTITUTIONS (ALL): 1. University of Saskatchewan, Saskatoon, SK, Canada.
2. Saskatchewan Transplant Program, Saskatoon, SK, Canada.
3. Safeway Pharmacy, Sobeys Inc., Saskatoon, SK, Canada.
4. University of Toronto, Toronto, ON, Canada.
5. Shopper's Drug Mart, Regina, SK, Canada.

Background: Non-adherence to immunosuppressive medications after kidney transplant is prevalent and responsible for significant morbidity and mortality. It is currently unknown whether education in the pre-transplant period can positively impact post-transplant adherence, since educational interventions are unstandardized, poorly described in transplant literature, and not rigorously studied.

Objective: To develop a standardized, evidence-based intervention that educates transplant candidates on the importance of medication adherence.

Methods: A literature search and needs assessment was undertaken to determine format and content for the educational intervention. Consultations with stakeholders occurred in the form of 3 studies in a Canadian center:1)A qualitative study investigating what kidney recipients wished they would have known before transplant 2)A qualitative analysis of health-care provider perspectives on transplant education 3)A mixed-methods study assessing health literacy, transplant knowledge, beliefs of medicines and education satisfaction in patients on the kidney waitlist. Nvivo software was used to code qualitative data and identify themes, and descriptive and univariate statistics were calculated on demographics and assessments in the 3rd study. A list of guiding principles was generated from the needs assessment and literature review. The intervention was developed and reviewed by experts in medication adherence, video education, motivational psychology, and cultural education; transplant patients and health care providers.

Results: Video-based education was determined to be the preferred format since it is effective and can be provided at low cost with minimal impact to health care personnel, and replayed as needed for patients and families. The intervention consists of 6 videos ranging from 3 to15min in length, and outlines the transplant process with emphasis on adherence. Animations are incorporated to illustrate complex information for patients with low health literacy and patient testimonials align the content with principles of adult learning theory. The project can be viewed at: https://youtu.be/lqO3hgKX5R8

Conclusions: A 6-part video series has been developed to provide standardized education and has met the approval of stakeholders. Next steps include piloting the intervention in a group of transplant candidates to determine whether it improves transplant knowledge, and subsequently undertaking a multicenter trial to investigate whether education before transplant impacts post-transplant medication adherence.

Disclosure: Holly Mansell: No | Nicola Rosaasen: No | Rahul Mainra: No | Azaad Kukha-Bryson: No | Ahmed Shoker: No | Dave Blackburn: No | Jay Wilson: No | Maya Obadia: No | Gail MacKay: No | Paraag Trivedi: No

KEYWORDS: kidney transplantation, end-stage renal disease, immunosuppression.

ABSTRACT #22

TITLE: Development and Validation of the Kidney Transplant Understanding Tool (K-TUT)

AUTHORS (FIRST NAME, LAST NAME): Holly Mansell[1], Nicola Rosaasen[2], Rahul Mainra[1,2], Ahmed Shoker[1,2], Jeff Taylor[1], Dave Blackburn[1]

INSTITUTIONS (ALL): 1. University of Saskatchewan, Saskatoon, SK, Canada.
2. Saskatchewan Transplant Program, Saskatoon, SK, Canada.

Background: Several educational interventions have been designed to improve patient knowledge before and after kidney transplantation. However, evaluation of such interventions has been difficult because validated instruments to measure knowledge-based outcomes are lacking. This study sought to develop and validate an instrument to assess patient knowledge of kidney transplantation.

Methods: Two main purposes were defined for the tool a priori: 1.to be used as a tool for identifying and targeting education deficiencies 2.to measure knowledge as an outcome in educational research. Development of the Kidney Transplant Understanding Tool (K-TUT) took place over two phases: Phase 1: Tool Development: Determination of the concepts of interest and the formulation of the questions involved an extensive literature search and a focus group of transplant recipients. Establishment of content validity occured by review from 39 members of the Saskatchewan Transplant Program and Saskatoon Health Region. The questionnaire was reworded to meet acceptable reading level according to Flesh Kincaid and SMOG formulas, and piloted on 10 kidney transplant recipients.
Phase 2: Testing of the K-TUT: The K-TUT was tested in two separate cohorts of patients: i) a pre-transplant cohort, and ii) a post-transplant cohort. Experts and transplant recipients were consulted to establish content validity, and the tool was analyzed for internal consistency, reproducibility and construct validity.

Results: Surveys were offered to 106 pre-transplant patients and 235 transplant recipients. Response rates were 39% (41/106) and 63% (149/235), and mean scores were 53±8.5 (77%), and 56±6.3 (81%), respectively. Cronbach's alpha of the items in the tool ranged from 0.79 to 0.88. Interclass correlation coefficient (ICC) of the test-retest was 0.937 (95%CI,0.763-0.985) and 0.762 (95% CI,0.566-0.877) in the pre and post-transplant cohorts. Health literacy as measured by the Short Test of Functional Health Literacy (S-TOHFLA) in the pre-transplant group was significantly associated with increased knowledge (r=0.52;P<0.001). The majority of transplant recipients (98/148=67%) believed the questionnaire adequately assessed knowledge, 24% (36/148) were 'unsure', and 85% (126/148) agreed that no questions should be removed.

Conclusions: Content and construct validity, internal consistency and reproducibility of the K-TUT have been established. While more study is warranted to further assess psychometric properties, the Kidney Transplant Understanding Tool (K-TUT) appears to be a promising tool to measure transplant knowledge.

Disclosure: Holly Mansell: No | Nicola Rosaasen: No | Rahul Mainra: No | Ahmed Shoker: No | Jeff Taylor: No | Dave Blackburn: No

KEYWORDS: kidney transplantation, end-stage renal disease.

ABSTRACT #23

TITLE: Implementation of a Live Donor Champion Program to Increase Living Donation

AUTHORS (FIRST NAME, LAST NAME): Dana M. Parker[1]

INSTITUTIONS (ALL): 1. Transplant, University of Colorado Health, Aurora, CO, United States.

Background: Background: There are currently over 100,000 patients awaiting kidney transplant, and the average wait for a cadaver organ is over 5 years in many areas. While strategies for increasing deceased donor organ availability have been largely exhausted, living donation is associated with decreased wait time, improved recipient outcomes, and represents an organ pool with the highest potential for expansion. . Oftentimes, however, potential kidney recipients are overwhelmed with their own health needs and are hesitant to reach out to others regarding living donation. Friends, family, and often strangers can be the best advocates for candidates to expand the living donor pool.

Methods: With the above mentioned study in mind, our group sought to provide a similar DC training program offered in a more streamlined fashion. Originally consisting of two 2-hour sessions over a 2 week period, our DC program has evolved to a single 4-hour session occurring in a single day. This program aims to offer valuable resources to those in a position to identify potential living donors for a person in need but are limited by time or travel constraints. Education session interventions include; donor evaluation education, social media tutorials, role play, and a panel of previous recipients and donors. We evaluate DC overall

comfort level with initiating a conversation prior to and after sessions via questionnaire. Since inception, we have held three DC sessions with a total of nine participants surveyed.

Results: Preliminary data based on pre- and post-session questionnaires suggest overall living donor knowledge and comfort level in discussing donation increased significantly after each DC session. Participants also received social media tutorials and resources for use after completing the session. Role play experience helped increase the comfort level of DC in discussing living donation and connecting potential donors to the center. To date, in three DC sessions, three live donor surgeries have been completed and another DC session is scheduled.

Conclusions: Living Donor Champion Programs can work to quickly identify and move potential living donors through the evaluation process and ultimately on to live donation surgery. These people maynot have been identified through recipient alone as we know they are often uninformed or reluctant to discuss the need for donation with family members.

Disclosure: Dana Parker: No

KEYWORDS: Ethics, living donor transplantation, paired kidney exchange, organ acquisition.

ABSTRACT #24

TITLE: Trafficking of donor leukocytes and recipient cells cross-dressed with donor MHC molecules after transplantation of cardiac allografts in nonhuman primates.

AUTHORS (FIRST NAME, LAST NAME): Kortney Robinson, Jose Marino, Joshua Paster, Isabel Hanekamp, Joren C Madsen and Gilles Benichou

B**ackground**: Direct allorecognition, or activation of recipient T cells by allogeneic MHC molecules on donor leukocytes is known to trigger acute allograft rejection. Recently, we have documented the presence of recipient cells bearing donor MHC molecules on their surface in the spleens of murine heart transplant recipients. The display of donor MHC molecules on the surface of recipient cells has been termed "cross-dressed." Convincing preliminary evidence suggests that the presentation of donor MHC by recipient cells (cross-dressed cells) plays a key role in the initiation of the T cell alloresponse, which ultimately leads to allograft rejection.

Methods: We have evaluated this phenomenon in our well-characterized nonhuman primate model of heart transplantation, by detecting donor leukocytes (passenger leukocytes) and recipient cells displaying donor MHC molecules (cross-dressed) with imaging flow cytometry (Amnis) as shown in Figure 1. Blood samples were collected at early time points post vascular clamp removal (5-120 minutes) in a heterotopic heart transplant. As shown in Table 1, within minutes after graft placement, thousands of donor leukocytes and cross-dressed cells had migrated out of the transplanted heart. This is the first demonstration of the presence and trafficking of recipient cells cross-dressed with donor MHC molecules in a primate solid organ transplant model. This finding could serve as a reference for the timing of therapeutic protocols aimed at preventing allorecognition, instead of controlling its deleterious effect once it has been initiated.

Table 1.

Timepoints	Passenger Leukocytes	Cross-dressed Cells	
Pre-Transplantation	0	0	Number of cells per million recipient cells
5 minutes post-transplantation	2410	5780	
25 minutes post-transplantation	980	3220	
45 minutes post-transplantation	840	2530	
120 minutes post-transplantation	800	2100	

Figure 1. Cross-dressed Cell

ABSTRACT #25

TITLE: Pre-Transplant Obesity: Opportunity for Intervening during Long Organ Transplant Waiting time

AUTHORS (FIRST NAME, LAST NAME): Ekamol Tantisattamo[1], Haritha Mopuru[2]

INSTITUTIONS (ALL): 1. Multi-Organ Transplant Center, Division of Nephrology, Department of Internal Medicine, Oakland University William Beaumont School of Medicine, Royal Oak, MI, United States.
2. Multi-Organ Transplant Center, William Beaumont Hospital, Royal Oak, MI, United States.

Background: Obesity is one of the main reasons for excluding patients for kidney transplantation. A longitudinal nature of weight change in patients who are on kidney transplant waiting list is unknown.

Methods: Seventy consecutive kidney transplant recipients were reviewed. BMI were retrieved at 1 year pre-transplant and annually prior up to 6 years pre-transplantation.

Results: The majority of the recipients were male 41 patients(male 58.6%) Mean age was 52.7±1.4(mean±SEM). BMI at the time of transplant ranged 16.6-48.3 kg/m^2. The majority of the patients(39%) were overweight and 1/3 were obese(BMI⇢30 kg/m^2). The remaining had normal weight. All patients had ⇢1-measured weight at 1-year pre-transplantation and the followed-up weight ranged 1-6 years pre-kidney transplantation. Only BMI at 6 years pre-transplantation was significantly lower than the BMI at the time of transplantation(31.4±2.4 VS 27.6±0.7,p=0.0479;Figure1). Compared to BMI at the time of transplantation, more than half of the obese patients(55-75%) between 1 and 6 years pre-transplantation except only 33.3% of obese patients at 3 years pre-kidney transplant lost weight; whereas, <half of non-obese patients(31.8-45.5%) lost weight(Figure2). Moreover, at 2 and 4 years pre-kidney transplantation, obese patients lost more weight than non-obese patients at the time of kidney transplantation(p=0.0490 and 0.0492).

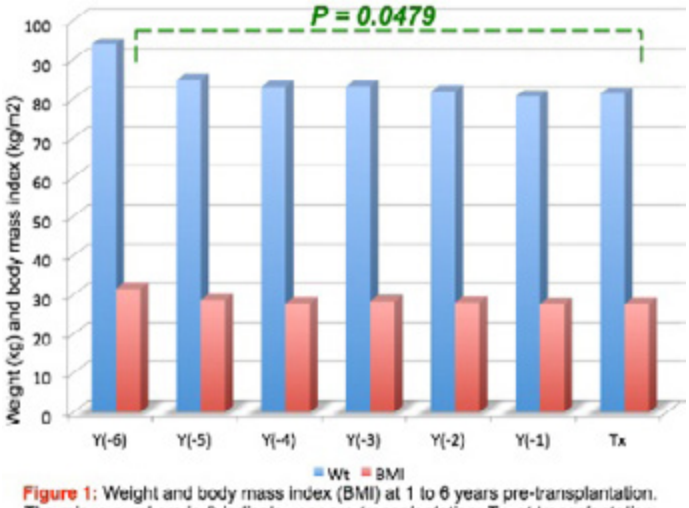
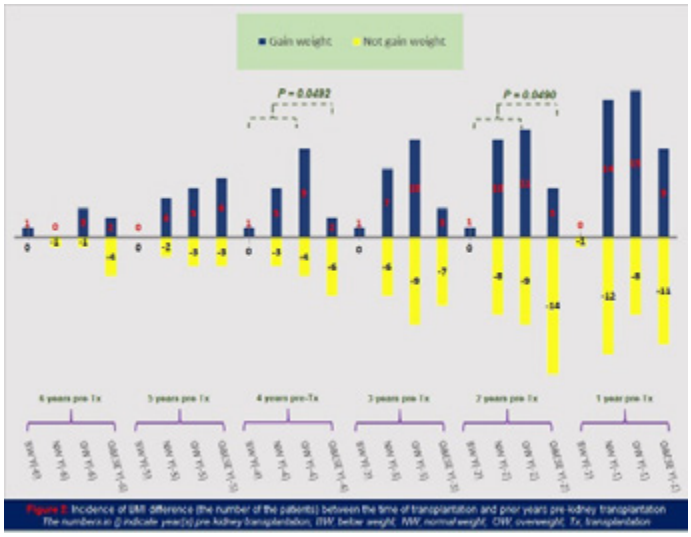


Figure 1: Weight and body mass index (BMI) at 1 to 6 years pre-transplantation.
The minus numbers in () indicate year pre-transplantation; Tx, at transplantation.



Conclusions: Obese patients who are on kidney transplant waiting list appear to lose more weight than non-obese patients particularly during the last 4 years prior to transplantation. Although a common problem, obesity should not be discouraged the patients from kidney transplantation especially those listed in the States with long organ transplant waiting time.

Disclosure: Ekamol Tantisattamo: No | Haritha Mopuru: No

KEYWORDS: kidney transplantation, kidney allocation, public policy, risk factors.

ABSTRACT #26

TITLE: Body Mass Index and Blood Pressure After Kidney Transplantation

AUTHORS (FIRST NAME, LAST NAME): Ekamol Tantisattamo[1], Haritha Mopuru[2]

INSTITUTIONS (ALL): 1. Multi-Organ Transplant Center, Division of Nephrology, Department of Internal Medicine, Oakland University William Beaumont School of Medicine, Royal Oak, MI, United States.
2. Multi-Organ Transplant Center, William Beaumont Hospital, Royal Oak, MI, United States.

Background: Correlation between body mass index (BMI) and blood pressure (BP) after kidney transplantation is unclear. Improved BMI after kidney transplantation may improve BP control.

Methods: A 27 living donor renal transplant recipients were reviewed. Demographic data, systolic blood pressure (SBP), diastolic blood pressure (DBP), and BMI were retrieved.

Results: Of all 27 patients, mean age was 50.8±2.87 years old (21.42-79.53) and 14 patients were female. The majority was Caucasian (20 patients). Mean BMI was 27.99±1.02 kg/m^2. Pre-transplant SBP and DBP were 135.48±3.39 and 81.26±2.72 mmHg, respectively. After 1, 3, and 6 months post-transplant, BMI, SBP, and DBP were lower than those measured pre-transplant; however, only SBP 1 month post-transplant significantly decreased from pre-transplant SBP (124.89±2.71 vs. 135.48±3.39 mmHg; p = 0.0181) (Figure 1). Among 12 patients with BMI ⇢30 kg/m^2, both SBP and DBP decreased as same as BMI at 1 and 6 months post-transplant, whereas all SBP, DBP, and BMI increased at 3 month post-transplant without statistical significance (Figure 2A). Similarly, SBP, DBP, and BMI of the remaining 15 patients with BMI <30 kg/m^2 had the same pattern as the patients with BMI ⇢30 kg/m^2 with further increased SBP, DBP, and BMI at 6 months post-transplant (Figure 2B).

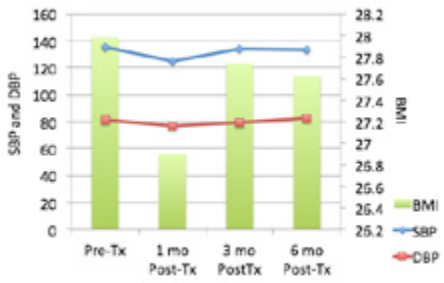


Figure 1: SBP, DBP, and BMI in all recipients

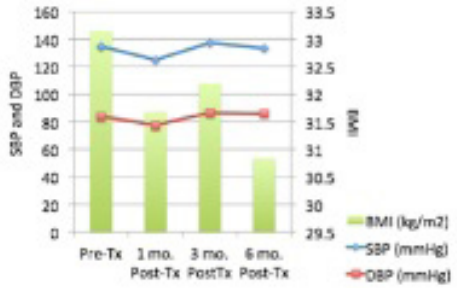


Figure 2A: SBP, DBP, and BMI in recipient with BMI >/=30

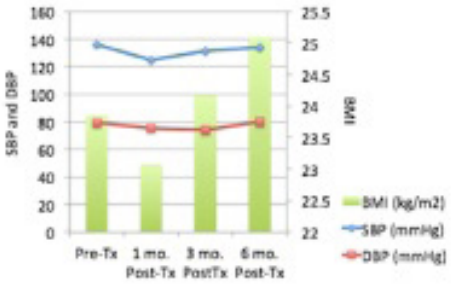


Figure 2B: SBP, DBP, and BMI in recipients with BMI <30

Conclusions: Although BMI decreased during early post-transplant period, rebounded obesity especially in the pre-transplant non-obese patients appears correlated with SBP and DBP during late post-transplant period. Weight control during post-transplant period should be one of the strategies for blood pressure management particularly in non-obese patients.

Disclosure: Ekamol Tantisattamo: No | Haritha Mopuru: No

KEYWORDS: kidney transplantation, kidney graft survival, end-stage renal disease, kidney disease.

ABSTRACT #27

TITLE: Post-Kidney Transplant Weight Changes and Induction Immunosuppression

AUTHORS (FIRST NAME, LAST NAME): Ekamol Tantisattamo[1], Haritha Mopuru[2]

INSTITUTIONS (ALL): 1. Multi-Organ Transplant Center, Division of Nephrology, Department of Internal Medicine, Oakland University William Beaumont School of Medicine, Royal Oak, MI, United States.
2. Multi-Organ Transplant Center, William Beaumont Hospital, Royal Oak, MI, United States.

Background: The effects of different induction immunosuppression on weight change after kidney transplantation is unclear.

Methods: Reviewing consecutive kidney and transplant recipients in the year 2015 yields 67 patients of whom received one of the following 3 induction immunosuppressive medications including rabbit-antithymocyte globulin (rATG), basiliximab, and alemtuzumab. The choice of these induction immunosuppressions depended on the patients' characteristic and using steroid-sparing strategies for alemtuzumab.

Results: The majority of the recipients were male (40 patients; 59.7%) and the mean age was 52.72±1.47 (Mean±SEM). Overall, mean weight and body mass index (BMI) at the time of kidney transplantation was 81.24±2.29 kg and 27.67 ± 0.68 kg/m^2 (16.62-48.29), respectively. At the time of kidney transplantation, the patients in basiliximab and alemtuzumab groups had equal mean weights and BMI; whereas, patients in the rTAG group had the lowest mean weight and BMI, but no statistical significance. Patients receiving rATG induction appear to lose weight; whereas, weight initially increased significantly at the early but subsequently decreased significantly in basiliximab and alemtuzumab groups. By using ANOVA with taking pre-transplant BMI into the consideration (non-obese and obese group with BMI of <30 and ⋯≥30 kg/m^2), both induction immunosuppression and obesity influent on weight change (βWt) only at 24, 48, and 72 weeks post-transplantation (p=0.014, 0.015, and 0.001). In rATG group patients significantly lost weight at 24 and 48 weeks post-transplantation (mean βWt = -19.01±5.88; p=0.015 and -27.32±8.13; p=0.013) compared with basiliximab group and at 48 week post-transplantation compared with alemtuzumab group (mean βWt = -20.76±7.47; p=0.042). Compared with alemtuzumab group, patient in basiliximab group gained weight (+DWt) at both 24 and 48 weeks, but no statistical significance (mean βWt 7.31±3.6; p=0.175 and 6.56±5.24; p=0.690) (Figure 1).

Conclusions: In obese patients, the rATG group lost significant weight compared to the remaining groups, and basiliximab group appeared to gain weight compared to alemtuzumab group. Since patients receiving rATG lost weight the most among of 3 induction immunosuppression, rATG should be considered as an induction immunosuppression for obese patients.

Disclosure: Ekamol Tantisattamo: No | Haritha Mopuru: No

KEYWORDS: kidney transplantation, induction immunosuppression, basiliximab, rabbit anti-thymocyte globulin.

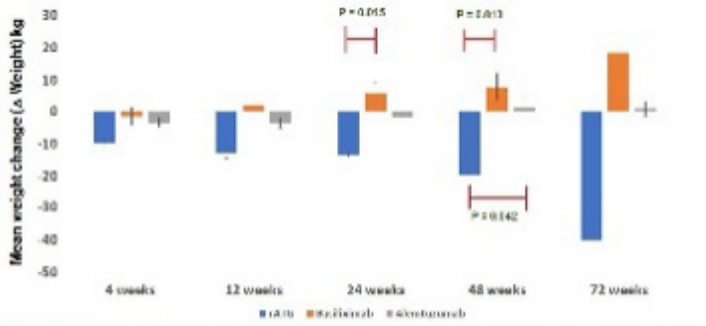


Figure 1: Mean weight changes (a Weight−Weight at Post transplant Weight at the time of transplant) at different time post-transplantation between 3 groups of induction immunosuppressions

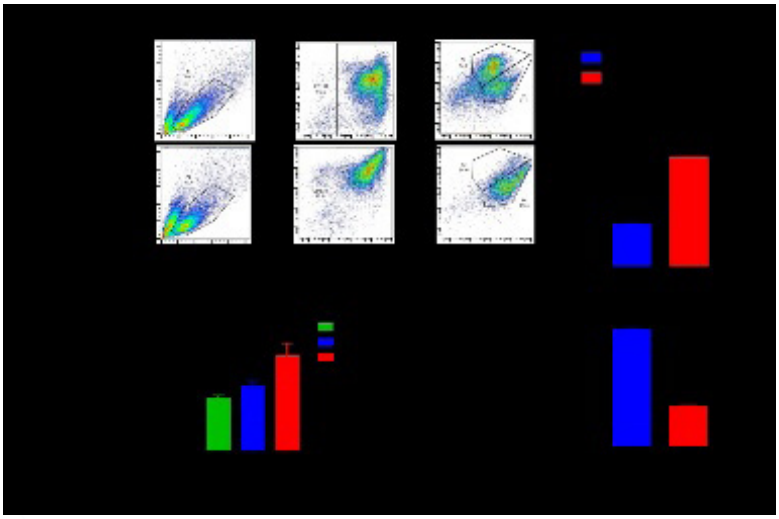
ABSTRACT #28

TITLE: Soluble Fibrinogen-like Protein 2 Regulates Differentiation and Enhances Immunosuppressive Function of Myeloid-derived Suppressor Cells in Allograft Immunity

AUTHORS (FIRST NAME, LAST NAME): Cheng Yang[1], Ming Xu[1], Tongyu Zhu[1]

INSTITUTIONS (ALL): 1. Urology, Zhongshan Hospital, Fudan University, Shanghai, China.

Background: Soluble fibrinogen-like protein 2 (sFGL2) is a novel immunoregulatory molecule, secreted mainly by regulatory T cells. CD11b$^+$ Gr1$^+$ myeloid-derived suppressor cells (MDSCs) are an important regulatory innate cell population and have significant inhibitory effect on T cell-mediated responses.



Methods: Here, we synthesized murine full length sFGL2 by eukaryotic expression system, and investigated the impact on differentiation and function of MDSCs. Bone marrow cells from BABL/c mice were cultured with or without 10 μg/ml sFGL2 for 3 days and 5 days under 10 ng/ml GM-CSF stimulation. Skin transplant was performed from BABL/c mice to C57/B6 mice with or without sFGL2-induced MO-MDSCs infusion.

Results: Compared with PBS, sFGL2 significantly induced CD11b$^+$Ly6G$^-$Ly6C^{high} MDSC (MO-MDSC) differentiation but inhibited CD11b$^+$Ly6G$^+$Ly6C^{low} MDSC (PMN-MDSC) differentiation. The sFGL2-induced MO-MDSCs significantly inhibited T cells proliferation compared with those induced by PBS. Besides, sFGL2-induced MO-MDSCs demonstrated higher expression of arginase-1 and iNOS at both

mRNA and protein level. Furthermore, adoptive transfer sFGL2-induced MO-MDSCs prolonged the skin allograft survival in mice. In the sFGL2-induced MO-MDSCs infusion group, the transplanted skin allograft showed mild inflammatory immune cell infiltration, less apoptosis and necrosis, and lower pro-inflammatory cytokines expression. T cells in the recipient mouse displayed a lower autoimmune phenotype (lower TCR^+ $CD44^{high}$ $CD62^{low}$ cells).

Conclusions: Taken together, our results indicate sFGL2 prompts MO-MDSCs differentiation and enhances their immunosuppressive function.

Disclosure: Cheng Yang: No | Ming Xu: No | Tongyu Zhu: No

KEYWORDS: tolerance, innate immunity.

ABSTRACT #29

TITLE: The mTOR Signal Regulates Myeloid Derived Suppressor Cells Differentiation and Immunosuppressive Function in Acute Kidney Injury

AUTHORS (FIRST NAME, LAST NAME): Cheng Yang[1], Chao Zhang[1], Tongyu Zhu[1]

INSTITUTIONS (ALL): 1. Urology, Zhongshan Hospital, Fudan University, Shanghai, China.

Background: The mammalian target of rapamycin (mTOR) signal controls innate and adaptive immune response in multiple immunoregulatory contexts. Myeloid derived suppressor cells (MDSCs) are a heterogeneous population of myeloid cells of potent immunosuppressive capacity. In this study, we aimed to investigate the role of MDSCs in the protection of acute kidney injury (AKI) and the regulation of mTOR signal on MDSC's protective role in this context.

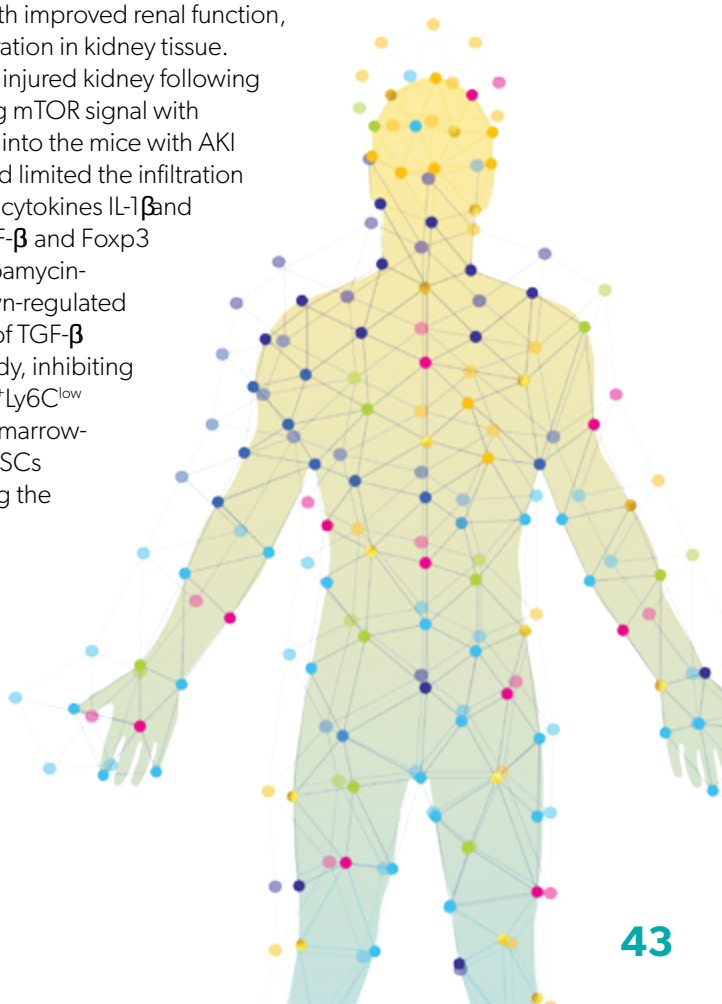
Methods: Rapamycin was administered in mice ischemia reperfusion injury model. Renal function and tissue injury were assessed. Different subsets of MDSCs were examined by flow cytometry. For in vitro experiment, MDSCs differentiation and immunosuppressive function were investigated with or without rapamycin treatment. We also adoptive transferred rapamycin-treated MDSCs into mice with AKI.

Results: In mice AKI model, rapamycin administration was associated with improved renal function, restored histological damage and decreased $CD4^+$ and $CD8^+$ T cell infiltration in kidney tissue. MDSCs, especially $CD11b^+Ly6G^+Ly6C^{low}$ G-MDSCs were recruited to the injured kidney following the interaction of CXCL1, CXCL2 and their receptor CXCR2 after inhibiting mTOR signal with rapamycin treatment. The adoptive transfer of rapamycin-treated MDSCs into the mice with AKI significantly improved renal function, ameliorated histologic damages and limited the infiltration of T cells in kidney tissue. In addition, the expression of pro-inflammatory cytokines IL-1β and IFN-βmRNA and protein was down-regulated while the expression of TGF-β and Foxp3 mRNA and protein was up-regulated in kidney tissue after transferring rapamycin-treated MDSCs. Adoptive transfer of rapamycin-treated MDSCs also down-regulated the serum levels of IL-1β IL-6 and IFN-βand up-regulated the serum levels of TGF-β compared with the IR group and PBS-treated MDSC group. In *in vitro* study, inhibiting mTOR signal regulated the induction of MDSC towards the $CD11b^+Ly6G^+Ly6C^{low}$ G-MDSC subset. The ability to suppress T cell proliferation of both bone marrow-derived $CD11b^+Ly6G^+Ly6C^{low}$ G-MDSCs and $CD11b^+Ly6G^-Ly6C^{high}$ M-MDSCs was enhanced by inhibiting mTOR signal with rapamycin via up-regulating the expression of arginase-1 and iNOS mRNA.

Conclusions: Taken together, our results demonstrated that MDSCs ameliorated AKI and the protective effect was enhanced by mTOR signal inhibition via promoting MDSCs recruitment, regulating the induction of MDSCs and strengthening their immunosuppressive activity.

Disclosure: Cheng Yang: No | Chao Zhang: No | Tongyu Zhu: No

KEYWORDS: ischemia/reperfusion injury, innate immunity.



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