PARALLEL ORAL PRESENTATIONS 1C
TRANSPLANTATION

O41 PROLONGED ALLOGRAFT SURVIVAL MEDIATED BY INTERLEUKIN-10 EXPRESSING REGULATORY B CELLS REQUIRES B CELL RECEPTOR LIGATION BUT NOT COGNATE T CELL HELP
M Mallik, T M Conlon, C J Callaghan, E M Bolton, J A Bradley, G J Pettigrew
University Department of Surgery, Cambridge Biomedical Research Centre, United Kingdom

Introduction
Late graft loss is a major problem in clinical transplantation. We have previously demonstrated that IL-10-expressing regulatory B cells (Bregs) promote long-term allograft survival in a murine model of autoantibody-mediated chronic cardiac allograft rejection. Here we investigate further their mechanisms of action.

Methods
Bregs were generated in vitro by culturing naïve C57BL/6 (B6) B cells with anti-CD40 monoclonal antibody for 3 days. The importance of cognate T cell interaction was assessed by treatment of B6 recipient mice of minimally mismatched B6.H-2bm12 hearts with similarly prepared B cells unable to express the major histocompatibility class II (MHCII) molecule; and the importance of Breg antigen-specificity examined by treatment with a monoclonal population of similarly prepared B cells bearing B cell receptors (BCRs) specific for only hen egg lysozyme (HEL).

Results
Equivalent IL-10 expression by MHCII/- and HEL-specific "Bregs" was confirmed by enzyme-linked immunosorbent assay. Surprisingly, MHCII/- Bregs (n=6) were as effective as B6 Bregs at prolonging allograft survival (median graft survival time, MST > 100 days), and reducing the development of harmful autoantibody (<200 units throughout) and cardiac allograft vasculopathy (mean luminal stenosis, MLS = 11.8%, day 100); a lack of BCR ligation rendered HEL-specific "Bregs" ineffective (MST < 50 days, peak autoantibody > 600, MLS = 53.5%, day 50).

Conclusions
Bregs can act independently of cognate T cell help, but require BCR ligation. Breg-cell therapy may supplement T cell depletion strategies, and tailored Bregs specific for donor MHC may prove equally effective while avoiding the harmful effects of non-specific immunosuppression.

Take-home message
IL-10 expressing regulatory B cells can prolong allograft survival in a murine model of autoantibody-mediated chronic allograft rejection in the absence of cognate T cell help but require B cell receptor ligation.

O42 STIMULATING MACROPHAGES TO ENHANCE LIVER REGENERATION
MRC Centre for Regenerative Medicine, University of Edinburgh (1), Institute of Translational Medicine, University of Liverpool (2), Roslin Institute, University of Edinburgh (3), Department of Surgery and Surgical Sciences, Royal Infirmary Edinburgh (4), MRC Centre for Reproductive Health, University of Edinburgh (5)

Introduction
Macrophages are central to liver regeneration, showing potential for therapeutic manipulation. Using human data and mouse models effects of macrophage colony stimulating factor (CSF1) on outcome and its therapeutic potential in liver injury are explored.

Methods
Serum was analysed from patients with acute liver injury (paracetamol intoxication(PI)) or pre/post partial hepatectomy(PH). C57Bl/6 mice or similar background were used in all preclinical studies (n=8/group). Ethical committee and home office approval was granted.

Results
Following PI serum CSF1 level was higher in survivors (n=31) than those who died or required liver transplantation (n=47)(p<0.0001;ROC-AUC=0.84(0.74-0.93)). In mice hepatic CSF1 gene expression increased following PI (p<0.01) and blockade of the CSF1
receptor (CSF1R) lead to persistent increase in liver injury markers (ALT/AST/alkphos, p<0.01). Following PH in humans (n=12) and mice (4 time points) serum CSF1 level remained low, however blockade of the CSF1R in mice lead to massive impairment of hepatocyte proliferation (p<0.001). Multiphoton microscopy of the CSF1R-eGFP mouse and lineage tracing with the CSF1R-Cre reporter showed CSF1R expression restricted to macrophages. Administration of a novel CSF1-Fc conjugate to uninjured mice elevated proregenerative cytokine expression, resulting in liver growth. CSF1-Fc administration enhanced liver weight and hepatocyte proliferation (Ki67) in PI, 2/3 PH and 2/3 PH with chronic liver injury (p<0.01). Flow cytometry of hepatic macrophage populations identified the predominant regenerative macrophage phenotype (F480-int;CD11b-high;CD45-high;Ly6C-high/int;CD206-neg) induced by CSF1-Fc administration.

**Conclusion**
CSF1 shows promise as a biomarker in acute liver failure and CSF1-Fc shows potential as a therapeutic agent to enhance acute liver regeneration.

**Take-home message**
Macrophage colony stimulating factor (CSF1) has a major role in liver regeneration and shows therapeutic potential.

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**O43 TARGETING THE BAFF/APRIL SIGNALLING AXIS FOR PREVENTION OF ANTIBODY-MEDIATED ALLOGRAFT REJECTION**
M Chhabra, M Mallik, J Garland, M Negus, S Rehavoka, J Ali, EM Bolton, JA Bradley, GJ Pettigrew
University of Cambridge, Department of Surgery

**Introduction**
B cell activating factor (BAFF) and its related cytokine APRIL (a proliferation-inducing ligand), are fundamental for B cell maturation. Here we study how their blockade influences hepatic allograft rejection.

**Methods**
BAFF/APRIL signalling was blocked using TACI-Ig fusion protein in a mouse model of alloantibody-mediated rejection, in which T-cell-deficient (TCRKO) B6 recipients of BALB/c heart allografts are reconstituted with TCR75 CD4 helper T cells that recognise processed Kd-alloantigen. Kinetics of graft rejection were monitored and humoral alloimmunity assessed by: immunohistochemical appraisal of splenic germinal centre (GC) activity; quantification of splenic and bone marrow (BM) plasma cells (PCs); and assay of serum anti-Kd IgG alloantibody.

**Results**
TACI-Ig treatment of naive TCRKO mice induced profound depletion of mature B cells, similar to the B cell profile observed in mice genetically-deficient for BAFF-receptor. Additional administration of TACI-Ig to TCR75-reconstituted TCRKO recipients of BALB/c heart grafts nevertheless accelerated allograft rejection, with preservation of a strong alloantibody response despite negligible total numbers of IgG-secreting splenic and BM PCs. This response was confined to the extrafollicular arm, because neither Kd-specific GC activity nor BM PC deposition was detectable. To target this escape, TACI-Ig was administered in conjunction with anti-CD20. Anti-CD20 alone prolonged graft survival modestly with BM PC deposition, but when used in conjunction with TACI-Ig, grafts survived long-term without BM deposition of Kd-specific PCs.

**Conclusions**
Despite BAFF-blockade, alloantigen challenge permits escape of alloreactive B cells and generation of effector alloantibody. BAFF targeting strategies may nevertheless synergise with conventional B cell depleting agents. [GC-germinat centre; BM-bone marrow; PC-plasma cell]

**Take-home message**
B cell activating factor (BAFF) and its related cytokine APRIL, are fundamental for B cell maturation and hence, are emerging targets in the treatment of antibody-mediated rejection. Addition of anti-BAFF/APRIL therapy to conventional anti-B cell agents was found to abolish alloantigen-specific bone marrow plasma cell deposition and significantly ameliorate antibody-mediated graft outcomes in a murine model of heart transplantation.

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**O44 IMPAIRED ENDOTHELIUM DEPENDENT RELAXATION—A PROXY OF VIABILITY FOR DONATION AFTER CIRCULATORY DEATH (DCD) KIDNEYS**
Introduction

DCD kidneys are at increased risk of delayed and primary non function. This study investigated the endothelial response to acetylcholine with increasing cold time in a porcine DCD model. The response of a blood vessel to acetylcholine is the resultant of endothelially secreted nitric oxide and direct vasoconstrictive effects on smooth muscle.

Methods

Kidneys were harvested with 15 minutes of warm ischaemia and then underwent 2 (n=7) or 16 (n=8) hours of static cold storage. Kidneys were reperfused with a normothermic oxygenated autologous blood based solution containing creatinine on an isolated organ perfusion circuit.

Results

Mean (±SD), urine output (2h 624±37ml vs 16h 335±164; P=0.0014) and creatinine clearance (2h: 12±5.4ml/min/100g.h vs 16h 5.1±3.7; P=0.0159) were higher in the 2h group. Renal blood flow (2h 441.3±153.5ml/min/100g.h vs 16h: 573±255; P=0.244) and intra-renal resistance (2h 7.5±2.8, vs 16h 8.7±8.2 mmHg/min/100g; P= 0.714) were equivalent. There was a significant acetylcholine-induced vasodilatation in the 2h (Renal blood flow baseline 67.0ml/min/100g±26.2 vs 10-8 mM acetylcholine 81.2±34.7; P=0.0338) but not the 16h group (P>0.05).

Conclusions

The reduction of function in the 16h group may result from greater cold ischaemic injury. The loss of acetylcholine-induced vasodilatation after 16 hours of cold storage suggests that endothelium is more sensitive to cold ischaemia than vascular smooth muscle. Endothelial response to acetylcholine may form the basis for a test to determine viability of DCD kidneys after prolonged cold ischaemia. Abbreviation. DCD: Donation after Circulatory Death.

Take-home message

The loss of acetylcholine-induced vasodilatation after 16 hours of cold storage in a porcine DCD kidney model suggests that endothelium is more sensitive to cold ischaemia than vascular smooth muscle. Endothelial response to acetylcholine may form the basis for a test to determine viability of DCD kidneys after prolonged cold ischaemia.
Results
The ratio of urine protein/creatinine was significantly lower in the group treated with CHBP compared with that in the IR group with or without CsA treatment at both time points. The tubulointerstitial damage and interstitial fibrosis were improved by CHBP in CsA treated group only at 2 weeks (P<0.05). In addition, the expression of mTOR, HSP27 and SAPK/JNK protein was decreased by CHBP in the IR kidneys at 2 weeks (P<0.05).

Conclusions
CHBP protected the kidney against IR and CsA induced injury in the mouse model, which might be associated with the reduction of mTOR, HSP27 and SAPK/JNK protein.

Take-home message
CHBP exhibited protective effects on ischemia-reperfusion and cyclosporine A induced renal injury in a mouse model, which might be associated with the reduction of mTOR, HSP27 and SAPK/JNK protein.

046 AMELIORATION OF ISCHAEMIA-REPERFUSION INJURY IN A MOUSE MODEL OF CARDIAC TRANSPLANTATION USING A NOVEL MITOCHONDRIA-TARGETED ANTIOXIDANT
AJ Dare (1,2), A Logan (2), TA Prime (2), M Goddard (3), EA Bolton (1), JA Bradley (1), GJ Pettigrew (1), MP Murphy (2), K Saeb-Parsy (1)
Department of Surgery, University of Cambridge (1), MRC Mitochondrial Biology Unit, Cambridge (2), Papworth Hospital NHS Foundation Trust (3)

Introduction
Accumulating evidence supports a key role for mitochondrial oxidative damage in ischemia reperfusion injury (IRI), a major cause of early graft dysfunction in transplantation. We therefore investigated the efficacy of a mitochondria-targeted antioxidant, MitoQ, in ameliorating IRI.

Methods
To induce a minimal and a severe ischaemic injury in a syngeneic mouse model of heterotopic cardiac transplantation, donor hearts were flushed with Soltran(± 50μM MitoQ), then stored at 4°C for 30min or 4h in UW solution(± 50μM MitoQ) prior to transplant. MitoQ uptake was confirmed using mass spectrometry. IRI severity was assessed by cardiac troponin-I levels and histology. Mitochondrial reactive oxygen species(ROS) generation was measured using mass spectrometry. Oxidative damage was assessed by protein carbonyls (ELISA) and mitochondrial DNA(mtDNA) damage (qPCR). Serum cytokine responses were determined by immunoassay.

Results
Prolonged cold preservation (4h vs.30min) resulted in higher troponin (4.9ng/mL±1.2 vs.0.6±0.2) and worse histological injury 24h post-transplantation. This was associated with a 2-fold increase in mitochondrial ROS, increased oxidative damage to myocardial proteins and mtDNA and a heightened IL-6 response. MitoQ was successfully taken up into the graft mitochondria at 4°C and reduced the severity of IRI posttransplant: there was a reduction in troponin (2.4ng/mL ±0.6 vs.4.9±1.2; 4h group), mitochondrial ROS and oxidative damage to proteins and mtDNA. This was accompanied by a diminished pro-inflammatory cytokine response.

Conclusions
Prolonged cold preservation of donor organs leads to increased mitochondrial oxidative damage at reperfusion and greater IRI, which can be successfully ameliorated with MitoQ. MitoQ represents a promising therapeutic candidate for transplant-related IRI.

Take-home message
The mitochondria-targeted antioxidant MitoQ represents a promising therapeutic approach to ameliorating post-transplant ischaemia reperfusion injury. Importantly, it has imminent potential for translation to the clinic, having already undergone Phase I-II clinical trials in a non-transplant setting.

047 CYCLOSPORINE A INDUCED IMMEDIATE VASOCONSTRICTION DEMONSTRATED DURING AND IMPAIRED ENDOTHELIUM DEPENDENT RELAXATION DEMONSTRATED AFTER REPERFUSION IN A PORCINE DONATION AFTER CIRCULATORY DEATH (DCD) MODEL.
G Lee, SA Hosgood, M Patel, CAM Crotty, ML Nicholson
University of Leicester, Department of Infection, Immunity and Inflammation, Leicester General Hospital
Introduction
The immediate nephrotoxic impact of cyclosporine is unknown in DCD kidneys. This was investigated in a porcine DCD model, during 3 hours of normothermic reperfusion.

Methods
Kidneys were harvested with 15 minutes of warm and 16 hours of cold ischaemia. They were reperfused with normothermic oxygenated autologous blood to which creatinine had been added on an isolated organ perfusion circuit. Cyclosporine (300ng/ml) (n=5) was compared with control (n=6). Dose response of renal blood flow after addition of acetylcholine was used as an index of endothelial function.

Results
Mean (±SD) AUC renal blood flow (control 573±255ml/min/100g.h vs cyclosporine 306±65; P=0.022) and oxygen consumption (control 47±13.3ml/min/g vs cyclosporine 25±5.5; P=0.002) were significantly lower in the cyclosporine group. Creatinine clearance (control 5.1±3.7ml/min/100g.h vs cyclosporine 3.5±1.4; P=0.299) and fractional excretion of sodium (control 106±54%.h vs cyclosporine 105 ± 49; P=0.978) were equivalent. Acetylcholine had a vasoconstrictive effect in the cyclosporine group (Renal blood flow: baseline 38.4±8.7ml/min/100g vs 23.8±4.4 10⁻⁵ mM acetylcholine; P=0.0003) but not control (P>0.05) Concentrations of urinary IL-1β, TNF-α, IL-8 and ET-1 were equivalent, (P>0.05).

Conclusions
We have demonstrated that the vasoconstrictive effects of cyclosporine occur immediately on reperfusion in a porcine DCD kidney model. The failure of endothelium dependent relaxation after cyclosporine suggests functional endothelial impairment also occurs during this timescale. Calcineurin inhibitor avoidance may reduce delayed graft function in DCD kidney transplants. Abbreviations: DCD: Donation after circulatory death. AUC: Area under the curve

Take-home message
We have demonstrated that the vasoconstrictive effects of cyclosporine occur immediately on reperfusion in a porcine DCD kidney model. The failure of endothelium dependent relaxation after cyclosporine suggests functional endothelial impairment also occurs during this timescale. Calcineurin inhibitor avoidance may reduce delayed graft function in DCD kidney transplants.

O48 THE EFFECT OF COLD ISCHAEMIC TIME ON CYTOKINE AND DIABETES MARKERS IN PANCREAS TRANSPLANTATION
HA Khambalia (1,3), Z Moinuddin (1), D van Dellen (1), M Nirmalan (2), Y Alexander (3), T Augustine (1)
Departments of Transplantation (1) and Anaesthetics (2), Manchester Royal Infirmary, Manchester, Cardiovascular Research Unit, University of Manchester, (3) Manchester, UK

Introduction
Prolonged cold ischaemic time (CIT) correlates to greater morbidity and poorer outcomes post pancreas transplantation. The biochemical nature and response on pancreatic function have not been delineated.

Methods
A prospective study was performed, investigating the relationship of CIT on cytokines and diabetes markers in the peri-operative period. Inflammatory markers (Interleukin (IL)-6 and -10) and pancreatic endocrine markers (Insulin and C-peptide) were measured (pre-operatively, at reperfusion, and 30minutes, 6, 12, 24, 48 and 72 hours post-perfusion). Repeated measures ANOVA was used to compare biomarker levels at these time points.

Results
18 consecutive simultaneous pancreas and kidney transplant recipients (9, CIT less than 14hours; 9, CIT greater than 14 hours (p<0.001, t-test)); matched for co-variables were included. Patients in the shorter CIT group had higher levels of IL-6 up to 48 hours postsurgery (significant at 12 hours (p=0.007)) and higher levels of IL-10 up to 72 hours post-surgery (significant at 12, 24, 48 and 72 hours (p=0.023, 0.023, 0.020 and 0.048 respectively)). Patients in the shorter CIT group demonstrated higher C-peptide levels, up to 72 hours postsurgery (p=0.023) and higher levels of insulin from 12 to 72 hours postsurgery (significant at 48 and 72 hours (p=0.004 and p=0.003 respectively)).

Conclusions
Shorter CIT offers protective benefits to the pancreas allograft, reflected in increased IL-6 and consequently IL-10, which have potent anti-inflammatory effects. This correlates with
improved short-term graft function in the shorter CIT cohort and suggests impaired graft function in the longer CIT cohort. This may translate to reduced long-term graft function.

**Take-home message**
This is the first study to show that prolonged Cold Ischaemic Time affects the systemic inflammatory response and reduces early pancreatic function post transplantation. This may translate in reduced long-term graft function.

**O49  EX-VIVO NORMOTHERMIC PERFUSION: A QUALITY ASSURANCE SYSTEM FOR KIDNEY TRANSPLANTATION**
SA Hosgood, AD Barlow, ML Nicholson
University of Leicester

**Introduction**
The suitability of marginal kidneys for transplantation is often questioned. Ex-vivo normothermic perfusion (EVNP) may allow us to quality-assure kidneys prior to transplantation.

**Methods**
Sixty discarded kidneys underwent 60 minutes of EVNP with an oxygenated red blood cell based solution at 36.0°C. During EVNP, kidney were categorised into grades based on their appearance; graded I (n=27) (good), II (n=22) (moderate) and III (n=11) (poor). Receiving operating characteristics (ROC) were used to identify thresholds of perfusion parameters according to the grade. The thresholds were applied to a series of 30 transplanted kidneys that underwent EVNP and the outcome analysed.

**Results**
The renal blood flow (RBF) and urine output (U/O) had the highest predictive values (area under the ROC 0.9 and 0.9 respectively). A threshold of RBF <64ml/min/100g and U/O <50ml were identified as cut off parameters. The RBF and U/O fell below the threshold in 5 kidneys in the transplant series, 4/5 of these kidneys were grade II compared to 2/25 in kidneys with perfusion parameters above the threshold. All of the other kidneys fell into the grade I category. Serum creatinine levels were significantly higher at day 7 and 14 (P=0.026, 0.046) and eGFR lower at 1 month (32±18 vs 50±16ml/min;P=0.04) in the lower threshold kidneys. Hospital stay was also significantly longer 14±8, 9±4days;P=0.044).

**Conclusion**
The quality of a kidney can be assessed using EVNP prior to transplantation. Kidneys with a low RBF and low U/O during EVNP have inferior early graft function and the recipients have prolonged hospitalisation.

**Take-home message**
The quality of a kidney can be assessed using ex-vivo normothermic perfusion prior to transplantation.

**O50  HUMAN LEUKOCYTE ANTIGEN B-CELL EPITOPE TERTIARY STRUCTURE AND SURFACE ELECTROSTATIC POTENTIAL REVEAL THE MOLECULAR BASIS FOR ALLOANTIBODY BINDING AND EPITOPE IMMUNOGENICITY**
DH Mallon, P Winn, E Bolton, JA Bradley, C Taylor, V Kosmoliaptsis
Department of Surgery, University of Cambridge

**Introduction**
The potential of donor HLA B-cell epitopes to induce recipient humoral alloimmunity depends on their structural and physiochemical properties. We determined the three-dimensional structure and electrostatic potential of two widely-expressed HLA B-cell epitopes and examined the impact of amino acid mutations on HLA antibody reactivity.

**Methods**
Tertiary protein models of high frequency HLA B alleles (n=24) expressing either the Bw4 or Bw6 epitope (defined by sequence motifs at positions 77-83) were generated using comparative structure prediction (Modeller) based on crystallographically-resolved HLA structures. The electrostatic potential in three-dimensional space encompassing the Bw4/Bw6 epitope, for each HLA-B molecule, was computed by solving the Poisson-Boltzmann equation for macromolecules and quantitatively compared with one another to form dendrograms that cluster epitopes with similar electrostatics properties. Amino acid (aa) mutations within the 77-83 sequence motifs were also examined.

**Results**
Comparison of the electrostatic potential in the space surrounding residues 77-83 allowed tight clustering of HLA-B molecules according to Bw4 or Bw6 epitope expression, independent of epitope aa composition and variability in the structural context of epitope expression, providing a molecular basis for known patterns of serological cross-reactivity. Critical amino acid mutations that abrogated antibody binding to Bw6-expressing HLA-B*07:02 (G83R, R79G, R82L) induced distinct electrostatic potential changes displacing the mutants from the Bw6 epitope cluster; in contrast, mutation N80T did not affect antibody binding and had a negligible physiochemical effect.

**Conclusion**
This study suggests that HLA B-cell epitopes are characterized by distinct topographic patterns of electrostatic potential, explaining HLA-specific antibody binding and enabling novel insights into HLA immunogenicity.

**Take-home message**
Characterizing HLA B-cell epitopes by their physiochemical properties can give greater insight into immunogenicity than by examining sequence alone.

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**O51 EX-VIVO NORMOTHERMIC PERFUSION IN MARGINAL DONOR KIDNEY TRANSPLANTATION**

SA Hosgood, AD Barlow, CA Crotty, ML Nicholson
University of Leicester

**Introduction**
Ex-vivo normothermic perfusion (EVNP) is a novel method of preservation that restores circulation and allows an organ to regain function prior to transplantation. This study reports the outcome of EVNP in kidneys from marginal donors.

**Methods**
Thirty kidneys from marginal donors underwent a short period of EVNP immediately before transplantation. Kidneys were perfused with a plasma free red cell based solution at a mean temperature of 34.7°C.

**Results**
Twenty one kidneys were from extended criteria donors (ECD), 4 from donation after circulatory death (DCD) donors and 5 from standard criteria donors (SCD) that suffered a hypoxic brain injury. The average donor age was 58 ± 11 yr and recipient age 59 ± 11. Kidneys were perfused for an average of 62.4 ± 12.2min. The mean renal blood flow during perfusion was 67.3 ± 29.3ml/min and the total amount of urine produced 168 ± 105ml. All kidneys were transplanted successfully with no complications. The total cold ischaemic time was 11.4 ± 4.3h and total ischaemic time 13.2 ± 4.5h. There were no incidences of primary non function and the delayed graft function rate (DGF) was 2/30 patients (6.7%). eGFR at day 7 and at 1 and 12 months was 44.7, 47.4 and 50.9ml/min respectively. Patient survival was 100% and graft survival at 12 months 93.3%.

**Conclusion**
This first series of EVNP in ECD, DCD and marginal donor kidney transplantation supports the concept that restoring circulation and function prior to transplantation is a safe and feasible method of kidney preservation.

**Take-home message**
Ex-vivo normothermic perfusion is a safe and feasible method of kidney preservation.

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**O52 DEPLETION OF MURINE INTRA-ALLOGRAFT B-LYMPHOCYTES INHIBITS RENAL CHRONIC ALLOGRAFT DAMAGE**

GH Tse, D Kluth, M Gray, J Hughes, D Gray, L Marson
MRC Centre for Inflammation Research, University of Edinburgh

**Introduction**
In the UK 4% of renal transplants are lost annually to interstitial fibrosis and tubular atrophy, which characterize chronic allograft damage (CAD). B-lymphocytes have been identified in human allografts however their significance is unclear. We aim to investigate the role of intra-allograft B-lymphocyte using a mouse model of renal CAD, furthermore we hypothesized that depletion of these cells could inhibit the development of CAD.

**Methods**
CAD was modelled using congenic strains, donor C57BL/6BM12 mice kidneys transplanted into C57BL/6 recipients, a single MHC-II mismatch. B-lymphocytes were depleted at 4 weeks following transplantation by intravenous anti-CD20 monoclonal antibody compared
to placebo. Mice were culled at 8 and 12 weeks and histological outcomes determined (n=6 per group).

**Results**

Administration of a single dose of anti-CD20 monoclonal antibody at 4 weeks following renal transplantation significantly inhibited the accumulation of B-lymphocytes and T-lymphocytes within the renal allograft cortex compared to placebo injection when evaluated at 8 weeks and 12 weeks (p<0.0001). Furthermore B-lymphocyte depletion reduced the number of germinal centres per mm² tissue area (p<0.05). B-lymphocytes in the germinal centres co-localised on immunofluorescence for B220+ IgM+ IgG+ GL7+ furthermore plasma cells were apparent based on CD138+. B-lymphocyte depletion protected against the development of CAD with more viable tubules (p<0.05) and reduced collagen deposition (p<0.05).

**Conclusions**

Treatment of renal allograft transplanted mice with anti-CD20 reduces the density of B-lymphocytes and T-lymphocytes. Intra-allograft B-lymphocytes form germinal centres and this is inhibited by anti-CD20. Intra-allograft B-lymphocytes play a key role in mediating chronic damage in the transplanted kidney.

**Take-home message**

Intra-allograft B-lymphocytes play a key role in mediating chronic damage in the transplanted kidney. Intra-allograft B-lymphocytes may be important targets to inhibit interstitial fibrosis and tubular atrophy in human kidney transplants.

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**O53 VIABILITY ASSESSMENT OF DISCARDED HUMAN KIDNEYS USING EX-VIVO NORMOTHERMIC PERFUSION**

SA Hosgood, AD Barlow, ML Nicholson
University of Leicester

**Introduction**

Ex-vivo normothermic perfusion (EVNP) is a new technique of preservation that involves circulating an oxygenated blood based solution through the kidney at normal body temperature. Kidney function is regained and therefore it may be a valuable device on which to assess the viability of marginal donor kidneys prior to transplantation.

**Methods**

From October 2012 to June 2013, 60 discarded human kidneys underwent 60 minutes of EVNP. Kidneys were perfused with an oxygenated red blood cell based solution at a normal body temperature. Kidneys were graded based on a visual assessment as follows; Grade I: Good perfusion (pink and evenly perfusion), Grade II: moderate (pink/purple) and Grade III: poor perfusion (mottled purple). Renal functional parameters were recorded throughout perfusion.

**Results**

Grade I kidneys performed significantly better across all of the perfusion parameters compared to grade III kidneys. They had a significantly higher renal blood flow (I; 92 ± 26, II; 63 ± 25, III; 32 ± 15ml/min/100g;P<0.0001), higher level of oxygen consumption (67.7 ± 20.9, II; 42.2 ± 15.4, III; 22.3 ± 10.0ml/min/g;P<0.0001), higher urine output (I; 119 ± 73, II 105 ± 76, III 12 ± 13ml;P<0.0001) and higher creatinine clearance (I; 2.2 ± 2.0, II; 1.5 ± 1.5, III 0.1 ± 0.1ml/min/100g;P=0.002) compared to grade III. Grade II kidneys had improved function compared to grade III (P<0.05).

**Conclusion**

EVNP may potentially allow us to quality-assure kidneys prior to transplantation. Based on appearance and functional parameters we consider that grade I and II kidneys would be suitable for transplantation.

**Take-home message**

Ex-vivo normothermic perfusion may potentially allow us to quality-assure kidneys prior to transplantation.