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Table S1. CyTOF Antibodies.

Metal	Marker	Clustering
Rh103Di	Viability	
Nd142Di	CD19	Major Immune Compartments
ND143Di	CD45RA*	
Nd145Di	CD4	
Nd146Di	CD8	
Nd148Di	CD16	
Nd150Di	CD1c	
Eu141Di	CD123	
Sm152Di	CD66b	
Gd155Di	CD27*#	
Gd160Di	CD14	
Dy161Di	CD56	
Er168Di	CD3	
Er170Di	CD38*#	
Yb174Di	HLADR*	
In113Di	CD57*	Lymphocyte Compartments *$CD4^+$ and $CD8^+$ T cell subclustering #B cell subclustering
In115Di	CD11c#	
Nd144Di	KLRG1*#	
Pr141Di	IgD#	
Sm147Di	2B4*	
Sm149Di	CD127*#	
Eu153Di	PD-1*	
Sm154Di	ICOS*	
Gd156Di	TIM3*	
Gd158Di	CCR6*#	
Dy163Di	CXCR5*#	
Dy164Di	CD21#	
Er166Di	CD25*#	
Er167Di	CCR7*#	
Er169Di	OX40*	
Yb171Di	CD95*#	
Yb172Di	IgM#	
Yb173Di	CXCR3*#	
Lu175Di	TIGIT*	
Yb176Di	CD45RO*#	

Table S2. Baseline Characteristics of Study Donor and Recipients for Patients that Developed DSA.

	Total DSA^{POS} (n=10)	ABMR^{POS} (n=5)	ABMR^{NEG} (n=5)	p-value
Donors				
Age (years)	11.0 ± 5.8	12.0 ± 5.4	10.0 ± 6.6	0.61
Male no. (%)				1.00
Male	8 (80)	4 (80)	4 (80)	
Recipients				
Age (years)	10.8 ± 6.4	11.6 ± 7.2	10.0 ± 6.2	0.72
Male no. (%)				1.00
Male	8 (80)	4 (80)	4 (80)	
Race (%)				1.00
White	9 (90)	4 (80)	5 (100)	
Hispanic	1 (10)	1 (20)	0 (0)	
Primary Renal Disease no. (%)				1.00
Alport Syndrome	1 (10)	0 (0)	1 (20)	
ARPKD	2 (20)	1 (20)	1 (20)	
CAKUT	6 (60)	4 (80)	2 (40)	
Nephronophthisis	1 (10)	0 (0)	1 (20)	
Months on Dialysis	17.2 ± 10.2	17.4 ± 11.0	17.0 ± 10.7	0.95
Cold ischemia time (min)	816.8 ± 184.9	723.2 ± 91.8	910.4 ± 215.9	0.13
Year of Transplant	2009 ± 3.2	2010 ± 3.4	2008 ± 4.5	0.45
HLA Mismatch (A+B+DQ+DR)	4.6 ± 1.3	4.6 ± 1.1	4.6 ± 1.5	1.00

All donors were white and deceased. ARPKD: Autosomal recessive polycystic kidney disease; CAKUT: congenital anomalies of the kidney and urinary tract; HLA: human leukocyte antigen. Continuous variables represented as mean ± SD. Categorical variables are expressed as percentages. * $p<0.05$, ** $p<0.01$, *** $p<0.001$ by unpaired t-test for continuous variables or Fisher exact test for categorical variables.

Table S3. Allograft Outcomes of Patients by Development of ABMR.

	Total DSA^{POS} (n=10)	ABMR^{POS} (n=5)	ABMR^{NEG} (n=5)	p-value
eGFR post-transplant ¤				
3 months	98.8 ± 24.9	99.1 ± 28.8	98.4 ± 23.8	0.97
6 months	97.7 ± 24.9	97.6 ± 28.2	97.7 ± 24.5	0.99
9 months	94.7 ± 22.7	93.6 ± 22.4	95.8 ± 25.6	0.89
12 months	95.2 ± 23.2	94.2 ± 23.0	96.2 ± 26.1	0.90
DSA HLA Class reactivity	10 (100)	5 (100)	5 (100)	1.00
Class I	3[ƒ] (30)	2[ƒ] (40)	1[ƒ] (20)	
Class II				
DGF no. (%)	1 (10)	0 (0)	1 (20)	1.00
ACR no. (%)	3 (30)	2 (40)	1 (20)	1.00
ACR time post-transplant (months)	17.0 ± 1.0	16.5 ± 0.7	18.0 ± 0.0	1.00
Graft Loss no. (%)	3 (30)	3 (30)	0 (0)	0.17
Graft loss time post-transplant (months)		48.0 ± 25.1	N/A	
DSA Titers (MFI)	12531 ± 6920.9	13686.6 ± 9300.8	11375.4 ± 4234.5	0.63

¤eGFR: estimated glomerular filtration rate calculated by the Schwartz formula (Schwartz, Haycock et al. 1976); DSA: DGF: delayed graft function; ACR: acute cellular rejection. [ƒ]2 patients did not have Class I DSA at onset but developed them after detection of Class II DSA. Continuous variables represented as mean ± SD. Categorical variables as a percentage. *p<0.05, **p<0.01, ***p<0.001 by unpaired t-test for continuous variables or Fisher exact test for categorical variables.

Figure S1.

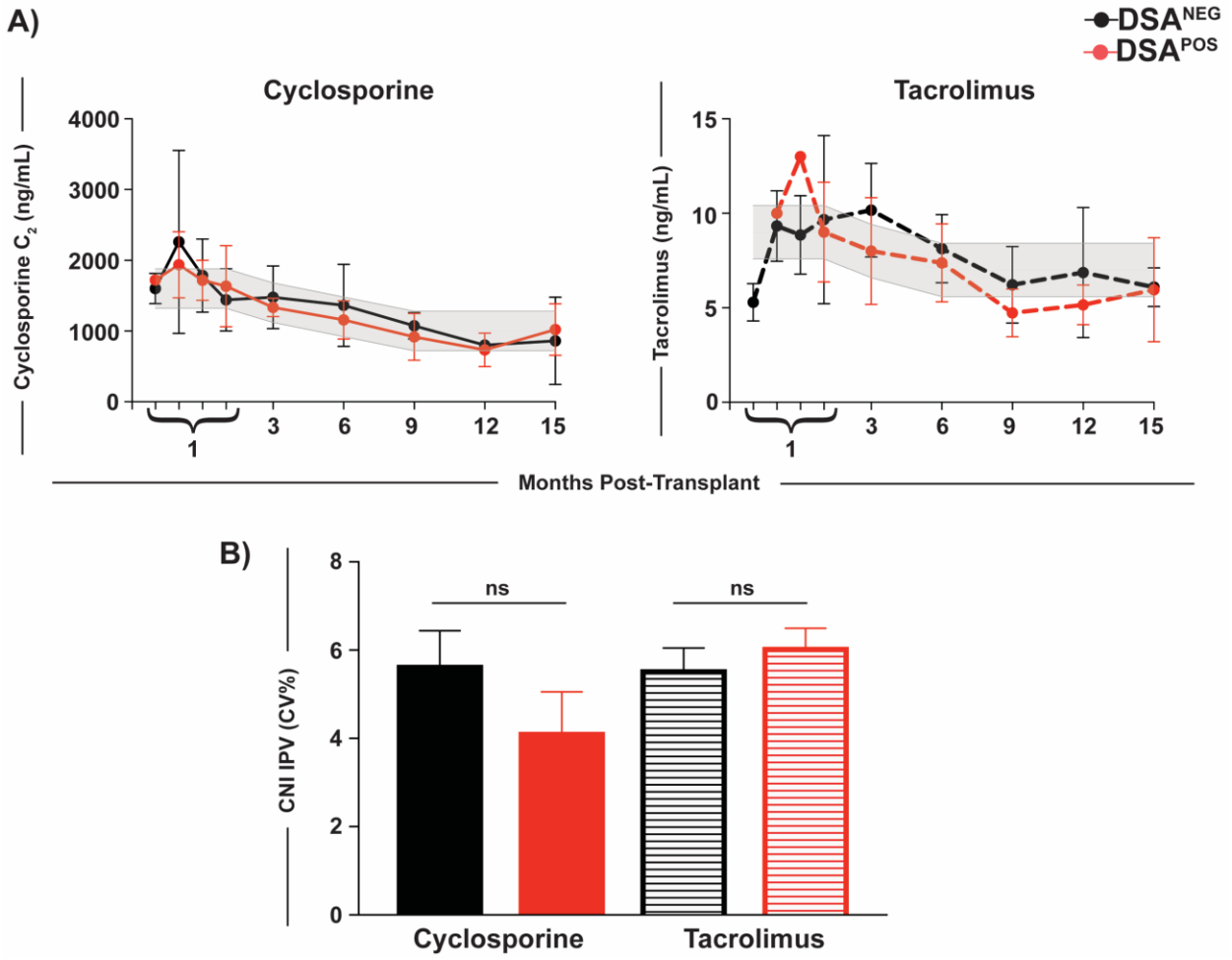

Figure S1. CNI Adherence was Similar Between DSA^{POS} and DSA^{NEG} Recipients. All patients received mycophenolate mofetil (MMF) and a calcineurin inhibitor (CNI), with or without steroids, as maintenance immunosuppression. **A**) Twelve patients received cyclosporine as the CNI (left; 7 DSA^{POS}, 4 DSA^{NEG}) and nine patients received tacrolimus (right;3 DSA^{POS}, 6 DSA^{NEG}). Error bars represent standard deviation (SD) and target values of the CNI are shown in grey. **B**) CNI intra-patient variability (IPV), as measured by the coefficient of variation (CV%), was not different in patients on tacrolimus during the "stable" phase of immunosuppression (≥6-months post-transplant). Error bars represent standard error of the mean (SD). *p<0.05, **p<0.01 by unpaired t-test.

Three patients switched from cyclosporine to tacrolimus; 2 were DSA^{NEG} and 1 was DSA^{POS}ABMR^{POS}. Coefficient of variation was calculated by formula: $(CV\%) = \sqrt{\{\Sigma(X_j - \bar{X})^2/(n-1)/\bar{X}\} * 100}$.

Figure S2.

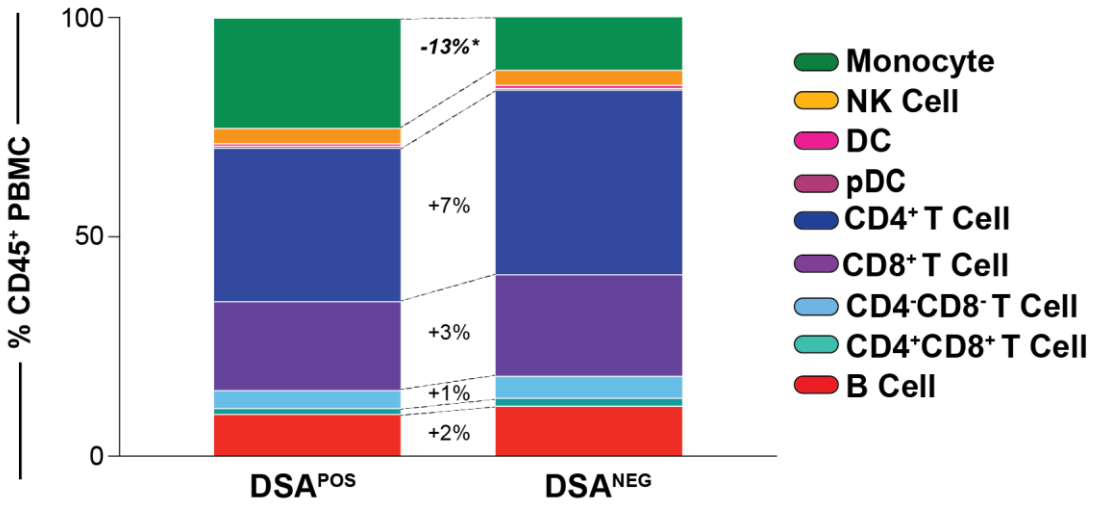

Figure S2. Increases in several lymphocyte populations account for the decreased monocytes seen in DSA^{NEG} recipients prior to DSA detection. Analysis of the breakdown of major immune cell classes prior to DSA detection in DSA^{POS} (left) and DSA^{NEG} (right) recipients. Percentage change in larger cell subsets annotated between bars. *p<0.05, **p<0.01 by unpaired t-test.

REFERENCES

Schwartz, G. J., et al. (1976). "A simple estimate of glomerular filtration rate in children derived from body length and plasma creatinine." Pediatrics **58**(2): 259-263.