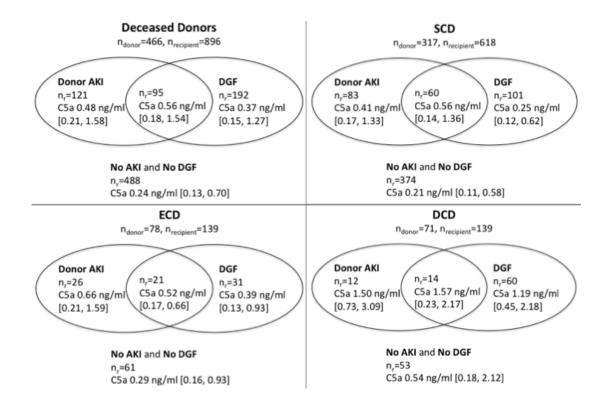
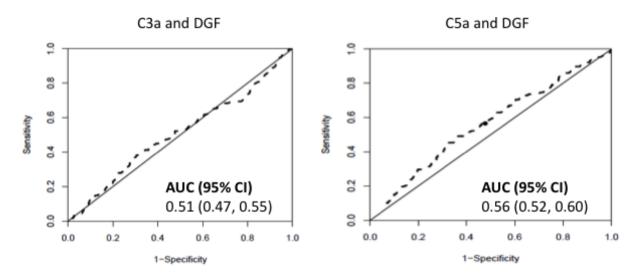


**Figure S1**: Complement is activated via the alternative or classical pathways. These pathways result in the assembly of an active C3 convertase and engage the alternative pathway through the C3b amplification loop. This leads to C5a generation and inflammatory reactions as well as to the assembly of the membrane attack complex cC5b-9.



**Figure S2:** Rate of donor AKI and DGF according to donor type. SCD, Standard criteria donor; ECD, expanded criteria donor; DCD, donation-after-cardiac-death; DGF, delayed graft function; AKI, acute kidney injury. Reported are median C5a concentrations [IQR].



**Figure S3:** ROC curves for urinary C3a and C5a and recipient delayed graft function (DGF).

**Table S1**: Urinary C3a and C5a levels by donor AKI with and without correction for urine creatinine

Urinary Biomarker	<b>ALL</b> (n=466)	<b>No AKI</b> (n=352)	AKI stage 1 (n=71)	AKI stage 2 or 3 (n=43)	P*
C3a (ng/ml)	0.33 [0.22, 0.5]	0.33 [0.22, 0.5]	0.30 [0.21, 0.45]	0.38 [0.2, 0.64]	0.22
C5a (ng/ml)	0.32 [0.15, 1]	0.27 [0.14, 0.83]	0.33 [0.13, 1.33]	0.92 [0.34, 1.62]	<0.001
C3a (corrected for urine creatinine)	0.01 [0, 0.02]	0.01 [0, 0.02]	0.01 [0, 0.02]	0.01 [0, 0.03]	0.77
C5a (corrected for urine creatinine)	0.01 [0, 0.03]	0.01 [0, 0.03]	0.01 [0, 0.06]	0.04 [0.01, 0.06]	0.002

AKI, acute kidney injury. AKI defined as stage 1 or higher with at least an increase in serum creatinine of ≥0.3 mg/d/l or at least an increase of 1.5-fold from admission to terminal value.

Statistics reported are median [IQR]. \*Wilcoxon rank sum test.

**Table S2**: Spearman correlations between complement and urinary injury biomarker (\*p<0.01)

Urinary biomarker	C5a	C3a
NGAL	0.60*	0.13*
KIM-1	0.15*	0.02
IL-18	0.44*	0.06

Table S3. Positive and negative predictive values of C5a with the outcome of DGF

Biomarker	Donors w	thout AKI	Donors with AKI		
	PPV	NPV	PPV	NPV	
C5a <sup>1</sup>	32%	78%	44%	56%	

AKI, acute kidney injury; PPV, positive predictive value; NPV, negative predictive value. 

¹ First tertile (C5a ≤0.3 ng/ml as negative test) vs. 2<sup>nd</sup> and 3<sup>rd</sup> tertile (C5a >0.3 ng/ml as positive test). The DGF prevalence rate was 28% in donors without AKI and 44% in donors with AKI.

Table S4. Urinary C5a and renal function 12-months after transplantation

	<b>C5a</b> <b>T1</b> (n=301)	<b>C5a T2 &amp; T3</b> (n=601)	P*
12-month eGFR (ml/min/1.73 m <sup>2</sup> )			
All	59.4 [47.3, 74.8]	56.5 [43.7, 72.8]	0.09
Donors without AKI	59.7 [46.9, 74.4]	56.9 [42.9, 74.0]	0.22
Donors with AKI	57.2 [47.7, 82.0]	55.8 [44.4, 68.2]	0.35

First (T1) vs. 2<sup>nd</sup> and 3<sup>rd</sup> tertile (T2,3). AKI, acute kidney injury; eGFR, estimated glomerular filtration rate. Statistics reported are median [IQR]. \* Wilcoxon rank sum test.