



**KDIGO CLINICAL PRACTICE GUIDELINE  
ON THE EVALUATION AND MANAGEMENT OF CANDIDATES  
FOR KIDNEY TRANSPLANTATION**

**SUMMARY TABLES & EVIDENCE PROFILES**

KDIGO - Transplant Candidate  
Guideline Topic: KTx vs WL  
Categorical outcomes

Pubmed id	Authors	Year	Name of database	Study design	Country	Period of patient recruitment	Length of follow-up	N analyzed	Inclusion criteria: General	Inclusion criteria: CKD specific	Inclusion criteria: Other	Age at evaluation/listing	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian, %	Race, Hispanic, %	Race, Other, %	Time on waitlist: WL group	Time on waitlist: KTx group	Primary renal diagnosis: GN, %	Primary renal diagnosis: HTN, %	Primary renal diagnosis: DM, %	Primary renal diagnosis: Other, %	Outcome	
15857921	Oniscu	2005	Scottish Renal Registry and UK Transplant	Retrospective	Scotland	1989-1999	1 year	1736	Txp vs. WL	dialysis and waitlisted	nd	46.6 (14.1)	61	nd	nd	nd	nd	nd	nd	nd	26	87	15	Interstitial nephritis 30%, multisystem 15%	All cause mortality	
17452897	Rao	2007	Organ Procurement and Transplantation Network	Retrospective	US	1990-2004	nd	5667	Txp vs. WL	pt who started dialysis before KTx	age>=70	70-74 79.0%, >=75 21.0%	68	70	16	5	8	Native American 0.5%	nd	nd	12	30	22	nd, 30%	All cause mortality	
15031354	Oniscu	2004	nd (sociodemographic, listing, transplant and comorbidity data partly from national renal (Scottish Renal Registry) and transplant (United Kingdom Transplant) databases)	Retrospective	Scotland	1989-1999	>1 year	325	Txp vs. WL	on dialysis when waitlisted	>60 yo	median (IQR) WL 66.3 (63.0, 72.9), Txp 64.0 (58.5, 69.5)	62	nd	nd	nd	nd	nd	median (IQR) 529 days (181, 877)	median (IQR) 252.5 days (21, 484)	24	nd	10	interstitial nephritis 24%, multisystem 22%	All cause mortality	
10755528	Johnson	2000	nd (Queensland cadaveric renal transplant waiting list)	Retrospective	Australia	1993-1997	2.8 years	174	Txp vs. WL	nd	>60 yo	66.1 (0.5)	44	89	nd	nd	nd	nd	nd	nd	5	nd	10	analgesic nephropathy 21%, ADPKD 14%, FSGS 4%, IgAN 6%, idiopathic 18%	All cause mortality	
20038521	Heldal	2010	Norway Renal Registry	Retrospective	Norway	1990-2005	nd (till May 2008)	286	Txp vs. WL	on dialysis when waitlisted, first Txp	>=70 yo	median (range) 73.6 (70.0, 81.0)	70	nd	nd	nd	nd	n	nd	nd	31	nd	4	pyelonephritis 10%, hereditary renal disease 9%, vascular diseases 38%, other/unknown 8%	All cause mortality	
18808405	Gillen	2008	USRDS	Retrospective	US	1990-2003	nd (till Dec 2003)	5961	Txp vs. WL	on dialysis at entry of the study, first Txp, no combined Txp	<=18 yo	11.2 (5.1)	57	67	26	nd	nd	nd, 7%	nd	7.9 months (11.8)	37	nd	nd	congenital 34%, vascular/interstitial 7%, nephrotoxic/tumor related 1%, other 7%, unknown 13%	All cause mortality	
26765937	Roland	2016	nd (national registry database)	Retrospective	US	2003-2010	4.0 years	317	Txp vs. WL	nd	HIV +	median (IQR): candidates 45 (39-52)	84	25	68	nd	nd	nd	nd	nd	nd	focal GN: 4	23	diabetic nephropathy: 11	nd	Survival
12631130	Glanton	2003	USRDS	Retrospective	US	1995-1999	51 months (accrual), 29 months (additional)	7443	Txp (living and deceased donors respectively) vs. WL	pt who started ESRD Rx, excluded transplant without preceding dialysis	excluded other organ Txp	48.1 (12.0)	54	nd	35	nd	nd	nd	nd	nd	nd	24	18	40	nd	All cause mortality

Graft loss

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Pubmed id	Authors	Year	Outcome definition	Predictor	Predictor definition	Full model (Txp vs. WL with interaction term)		Subgroup model 1 (Txp vs. WL in predictor subgroups)				P value in predictor group	Adjustment in predictor group, Other covariates	Comparison in non-predictor group	Estimate in non-predictor group, mean (95% CI)	P value in non-predictor group	Adjustment in non-predictor group, Other covariates	Overall Quality	
						Adjustment, Other covariates	P value	Metrics	Comparison in predictor group	Estimate in predictor group, mean (95% CI)									
15857921	Oniscu	2005	nd	age	18-34	N/A	N/A	RR	KTxp vs. WL	0.23 (0.05, 1.14)	nd	age, gender, primary renal disease, social deprivation, time since wait-listing, and comorbidity	nd	nd	nd	nd	nd	A	
				age	35-49	N/A	N/A	RR	KTxp vs. WL	0.26 (0.11, 0.57)	nd		nd	nd	nd	nd			
				age	50-59	N/A	N/A	RR	KTxp vs. WL	0.12 (0.05, 0.27)	nd		nd	nd	nd	nd	nd		
				age	60-64	N/A	N/A	RR	KTxp vs. WL	0.19 (0.04, 0.98)	nd		nd	nd	nd	nd	nd		
				age	>65	N/A	N/A	RR	KTxp vs. WL	0.34 (0.14, 0.83)	nd		nd	nd	nd	nd	nd		
17452897	Rao	2007	nd	Elderly (age)	>=70	N/A	N/A	RR	KTxp vs. WL	0.59 (0.53, 0.65)	<0.0001	causes of ESRD, WL time	nd	nd	nd	nd	nd	A	
				Elderly (age)	70-74	N/A	N/A	RR	KTxp vs. WL	0.58 (0.52, 0.65)	<0.0001		nd	nd	nd	nd	nd		
15031354	Oniscu	2004	nd	Elderly (age)	>=75	N/A	N/A	RR	KTxp vs. WL	0.67 (0.53, 0.86)	<0.05		nd	nd	nd	nd	nd	A	
				Elderly (age)	age>60	N/A	N/A	RR	KTxp vs. WL	0.35 (0.22, 0.54)	nd	sex, age, social deprivation, primary renal disease, dialysis modality, distance from pts' home to the Txp center	nd	nd	nd	nd	nd		
10755528	Johnson	2000	nd	Elderly (age)	age>60	N/A	N/A	HR	KTxp vs. WL	0.16 (0.06, 0.42)	nd		nd	nd	nd	nd	B		
20038521	Heldal	2010	nd	Elderly (age)	age>=70	N/A	N/A	HR	KTxp vs. WL	0.78 (0.52, 1.18)	0.25 (starting dialysis 1990-1999: 1.01 (0.58, 1.75); starting dialysis after 2000: 0.40 (0.19, 0.83))	age, sex, primary kidney disease, type of center where dialysis was initiated (university vs not university hospital), time on dialysis before waitlisting and dialysis modality	nd	nd	nd	nd	nd	A	
18808405	Gillen	2008	nd	Pediatric (age)	age=0-5 yo	N/A	N/A	RR	KTxp vs. WL	0.76 (0.32, 1.79) (12-18 months f/u); 0.52 (0.14, 1.91) (30-36 months f/u)	nd	age, sex, race, cause of ESRD, time of placement on wl	nd	nd	nd	nd	nd	A	
				Pediatric (age)	age=6-12 yo	N/A	N/A	RR	KTxp vs. WL	0.29 (0.08, 1.03) (12-18 months f/u); 0.09 (0.02, 0.54) (30-36 months f/u)	nd		nd	nd	nd	nd	nd		
				Pediatric (age)	age=13-18 yo	N/A	N/A	RR	KTxp vs. WL	0.36 (0.19, 0.69) (12-18 months f/u); 0.30 (0.15, 0.62) (30-36 months f/u)	nd		nd	nd	nd	nd	nd		
26765937	Roland	2016	nd	transplantation in HIV+ candidates	receiving transplant versus remaining on waitlist	age (by decade), BMI at enrollment (<21)	0.23	HR	KTxp vs. WL	0.6 (95% CI 0.3, 1.4)	nd		nd	nd	nd	nd	B		
12631130	Glanton	2003	nd	obesity	BMI>=30	N/A	N/A	HR	Obese: KTxp (deceased donor) vs. WL	0.39 (0.33, 0.47)	<0.0001	factors associated with obesity in patients placed on the renal transplant waiting list: race, age, gender, year of first dialysis session, cause of ESRD, additional variables	Non-Obese: KTxp (deceased donor) vs. WL	0.99 (0.35, 0.43)	<0.0001	factors associated with obesity in patients placed on the renal transplant waiting list: race, age, gender, year of first dialysis session, cause of ESRD, additional variables	A		
				obesity	BMI>=30	N/A	N/A	HR	Obese: KTxp (living donor) vs. WL	0.23 (0.16, 0.34)	<0.0001		Non-obese: KTxp (living donor) vs. WL	nd	nd	nd			
				obesity	BMI>=41	N/A	N/A	HR	Obese: KTxp vs. WL (all)	0.47 (0.17, 1.25)	0.13		nd	nd	nd	nd			
			nd	obesity	BMI>=30	N/A	N/A	HR	Obese: KTxp (deceased donor) vs. WL	0.35 (0.29, 0.42)	<0.0001		Non-Obese: KTxp (deceased donor) vs. WL	0.33 (0.30, 0.37)	<0.0001				

Pubmed id	Authors	Year	Population: Non-biased selection of study participants without inappropriate restrictions or selection. All eligible participants included or a random selection of these. No biased or large loss to follow-up.	Predictors/Variables: All predictors or study variables are well-defined and appropriately measured.	Outcome: Clearly longitudinal (incident outcome) [only if relevant]. Outcome blindly adjudicated or equivalent. Measured completely and the same for all participants.	Confounders: Important potential confounding factors appropriately accounted for.
15857921	Oniscu	2005	low	low	unclear	low
17452897	Rao	2007	low	low	unclear	low
15031354	Oniscu	2004	low	low	unclear	low
10755528	Johnson	2000	low	low	unclear	unclear
20038521	Heldal	2010	low	low	unclear	low
18808405	Gillen	2008	low	low	unclear	low
12631130	Glanton	2003	low	low	unclear	low
26765937	Roland	2016	low	low	unclear	unclear

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Quality Assessment

Pubmed id	Authors	Year	Model: Multivariable. All included variables reported. Appropriate model and methods for variable selection used. Reported results interpretable.	OVERALL: high if Population, Outcome, Model biased/bad; maybe high if predictors and confounders alone are high
15857921	Oniscu	2005	low	low
17452897	Rao	2007	low	low
15031354	Oniscu	2004	low	low
10755528	Johnson	2000	low	unclear
20038521	Heldal	2010	low	low
18808405	Gillen	2008	low	low
12631130	Glanton	2003	low	low
26765937	Roland	2016	low	unclear

## Evidence Profile: Kidney transplantation vs. waitlisting

Predictor	Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence	Other Considerations	Summary of Findings		
								Quality of Evidence	Description of Findings	Outcome Importance
<b>Age</b>	<b>Death</b>	6	14149 (174-5961)	No limitations (0)	No important inconsistencies (0)	Direct (0)	None (0)	High	Txp superior to waitlist in almost all age groups*†	Critical
<b>Transplantation in HIV+</b>	<b>Death</b>	1	317	Serious limitations (-1)	N/A	Direct (0)	Sparse (-2)	Very Low	Txp was comparable to waitlist in patients who were HIV+	
<b>Obesity</b>	<b>Death</b>	1	7443	No limitations (0)	N/A	Direct (0)	Sparse (-2)	Low	Txp similarly superior to waitlist among obese and nonobese	
	<b>Graft loss</b>	1	7443	No limitations (0)	N/A	Direct (0)	Sparse (-2)	Low	Txp similarly superior to waitlist among obese and nonobese	Critical
<b>Overall summary:</b>								<b>Quality of Overall Evidence:</b>		
Transplant generally found to be superior to continued waitlist status regardless of age or obesity								Variable		

GL = Guideline, N/A = not applicable, NS = nonsignificant predictor, Txp = transplantation.

\* 1 study found similar RR (0.12-0.34) of transplant vs. waitlist across age groups 18-34 years through >65 years (lowest age cohort was non-significant, likely due to lack of statistical power). 4 studies restricted to elderly (>60-70 years) all found significantly lower death with transplant (RR/HR=0.36-0.67), including in a subgroup restricted to ≥75 years old. 1 study of children found large differences in death, favoring transplant over waitlist across 3 age strata (0-5, 6-12, 13-18 years; HR=0.09-0.52); however, in the small subset of 0/5 year olds, the RR was not statistically significant.

### Evidence Profile: Effect of pre-emptive transplantation on post-transplant outcomes (from registry studies)

Outcome	Registries (No. Studies)	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence	Other Considerations	Summary of Findings		
							Quality of Evidence	Description of Findings	Outcome Importance
Death-censored graft loss	SRTR, USRDS (2)	26503	No limitations (0)	Important inconsistencies (-2)*	Direct (0)	Sparse (-1)	Very low	Lower risk c/t transplant w/in 1 y (HR ~0.94†) but higher c/t all post-dialysis transplants (HR=1.69)	Critical
Death	SRTR, USRDS (2)	26503	No limitations (0)	Important inconsistencies (-2)*	Direct (0)	Sparse (-1)	Very low	Lower risk c/t transplant w/in 1 y (HR ~0.83†) but higher c/t all post-dialysis transplants (HR=1.32)	Critical
<b>Overall summary:</b> Unclear whether pre-emptive transplantation lowers risk of graft loss or death.							<b>Quality of Overall Evidence:</b> Very Low		

\* One study compared pre-emptive transplant with transplant within 1 year of starting dialysis, while the other compared pre-emptive transplant vs. transplant any time after start of dialysis. These studies had different findings.

† Inverse of reported hazard ratio.

Scientific Registry of Transplant Recipients (SRTR), USRDS = United States Renal Data System

**KDIGO - Transplant Candidate**  
 Guideline Topic: Registry studies  
 Categorical outcomes

Pubmed id	Authors	Year	Name of database	Country	Period of patient recruitment	Length of follow-up (mean/median)	N analyzed	Eligibility criteria: General	Eligibility criteria: CKD specific	Eligibility criteria: Age specific	Eligibility criteria: Others	Age, mean (SD)/ median (range), years	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian (total), %	Race, East Asian, %	Race, South Asian, %	Race, Middle Eastern, %	Race, Hispanic, %	Race, Other, %	Primary kidney disease, GN, %	Primary kidney disease, HTN, %	Primary kidney disease, DM, %	Primary kidney disease, Other, %	Dialysis duration	Dialysis modality	Repeat or h/o KTxp, %	Panel reactive antibody, % or mean (SD)			
<b>PREDICTORS OF MORTALITY</b>																																
23295317	Cannon	2012	UNOS	US	2004-2009	nd	74983	All Txp	Kidney-alone transplant	nd	BMI 10-60	48 (16)	60	55	24	5	nd	nd	nd	14	nd, 2%	nd	21	16	nd	nd	nd	nd	16.0% (29.1)			
24138318	Farrugia	2014	HES/ONS	UK	2001-2012	4.4 years	19103	All Txp	KTxp alone	nd	excluded cases with incomplete demographic info	median (IQR) 45 (34, 55)	61	72	5	9	nd	nd	nd	nd	mixed 1%, other not reported 11%	nd	nd	16% reported as comorbidity	nd	nd	nd	nd	nd			
20814353	Huang	2010	OPTN/UNOS	US	2000-2008	at least 2 years	31179	All Txp	nd	>=60 yo	nd	median (IQR): 60-69yo 64 (61, 66), 70-79yo 63 (61, 66), >=80yo 81 (80, 82)	63	63	20	nd	nd	nd	nd	10	nd	11	26	12	other not reported 51%	preemptive 17%, >3y 32%	nd	nd	peak PRA>20%: 4%			
26660200	Ilori	2015	OPTN/UNOS	US	1996-2010	nd	44013	All Txp	nd	>=60 years	nd	median (IQR) 65 (7.0)	63	62	20	5	nd	nd	nd	11	nd, 2%	15	25	34	cystic kidney disease 9%, other unknown 17%	median (IQR) 2.47 years (2.81)	nd	nd	nd			
24009216	Kainz	2013	OEDTR	Austria	1992-2011	median 7.41 years	553	All Txp	First KTxp	nd	Underwent echo 1 year before KTxp (all pt who were potentially eligible for renal allograft wait-listing underwent a baseline echo with annual f/u while being listed)	52 (13)	58	100 (based on study conducted in Austria)	0	0	0	0	0	0	0	23	nd	14	vascular 9%, other unknown 55%	median (IQR), LA2D<=53mm 1.9 yr (0.8, 3.2), LA2D>53mm 1.8 (0.9, 3.2)	nd	0	median (IQR), LA2D<=53mm 0% (0, 0), LA2D>53mm 0% (0, 4)			
27336396	Kang	2016	UNOS	US	2005-2013	3.9 years	104632	All Txp	KTxp alone, not foreign donor kidney recipients	>=18 yo	excluded recipients with a pretransplant cancer other than skin cancer without coexisting skin cancer	median (IQR): w/o pre-Txp skin cancer 53 (42, 61), w/ pre-Txp skin cancer 64 (57, 70)	61	50	27	6	nd	nd	nd	15	nd	nd	nd	34% reported as comorbidity	nd	nd	nd	0	nd			
26147285	Krishnan	2015	RR/NHSBT	UK	2004-2010	nd	8082	All Txp	First KTxp	nd	nd	>70 yo: 2%, 50-70: 41%	63	85	4	9	nd	nd	nd	nd	nd, 2%	21	6	8	pyelonephritis 10%, polycystic disease 16%, uncertain 36%	nd	nd	0	nd			
26720436	Lynch	2016	USRDS	US	2000-2010	nd	37623	All Txp	nd	medicare population	included only pt with continuous primary coverage through medicare for at least 1 year before and after Txp	48.6 (13.6)	60	58	34	nd	nd	nd	nd	16	other not reported 8%	21	23	31	Cystic/hereditary /congenital 8%, Neoplasms/tumors 2%, other 15%	5.7 years (4.5)	nd	prior organ Txp 17%	nd			



Pubmed id	Authors	Year	Name of database	Country	Period of patient recruitment	Length of follow-up (mean/median)	N analyzed	Eligibility criteria: General	Eligibility criteria: CKD specific	Eligibility criteria: Age specific	Eligibility criteria: Others	Age, mean (SD)/ median (range), years	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian (total), %	Race, East Asian, %	Race, South Asian, %	Race, Middle Eastern, %	Race, Hispanic, %	Race, Other, %	Primary kidney disease, GN, %	Primary kidney disease, HTN, %	Primary kidney disease, DM, %	Primary kidney disease, Other, %	Dialysis duration	Dialysis modality	Repeat or h/o KTxp, %	Panel reactive antibody, % or mean (SD)
21449945, 27391198, 22156753	Molnar	2011, 2015, 20	SRTR, DaVita	US	2001-2007	median: 717 days, IQR (356, 1206)	14508	All Txp	First KTxp, on HD or PD before Txp	nd	nd	48 (14)	61	nd	27	nd	nd	nd	nd	nd	nd	nd	nd	27 reported as comorbidity	nd	0-6 m 12%, 6-24 m 29%, 2-5 y 36%, >5 y 23%	HD 86%, PD 14%	0	10.1% (24.0)
							8961	All Txp	First KTxp, on HD before Txp	nd	Excluded pts without electronically recorded serum albumin levels in the last quarter prior to transplantation, lacked data from the baseline quarter, with outlier values for age																		
							10083	All Txp	First KTxp, on HD before Txp	>=18 yo	nd																		
26102616	Opelz	2016	CTS	Germany	1995-2012	10 years	46548	All Txp	First KTxp	>=18 years	No h/o combined organ Txp, smoking status was documented at the time of Txp	>60 yo: 18%	62	73	nd	nd	nd	nd	nd	nd	nd, 30%	nd	nd	nd	nd	nd	nd	0	nd
24070588	Pieloch	2014	UNOS	US	2001-2006	3 years	30132	All Txp	First Txp	adults	excluded pt with multiorgan Txp	48.4 (13.9)	57	56	22	nd	nd	nd	nd	13	unknown 8%	nd	nd	nd	nd	nd	82	0	nd
25758804	Pieloch	2015	OPTN/UNOS	US	2000-2008	3 years	100261	All Txp	nd	adults	excluded pt with multiorgan Txp	18-49 49%, 50-64 38%, >=65 13%	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	29% reported as comorbidity	nd	0 years 10%, 0-4 55%, 4-24%	nd	nd	nd	
21566110	Reddy	2011	OPTN/UNOS	US	2001-2007	3 years	75681	All Txp	First Txp	>18 yo	excluded multiorgan Txp, pt with pre-Txp HCV infection, included pt with at least one follow-up visit reported to OPTN/UNOS	>= 60 yo 26%	60	56	23	5	nd	nd	nd	13	unknown 2.2%	20	23.4	25	unknown 21%	no 19%, < 1 year 18%, 1-3 years 31%, >= 3 years 33%	nd	0	>= 10% 18%
21415312	Streja	2011	SRTR/MHD	US	2001-2007	2.3 years	10090	All Txp	First Txp	nd	nd	49 (13)	51	nd	27	4	nd	nd	nd	15	nd	nd	nd	45% as comorbidity	nd	< 6 m 12%, 6-24 m 29%, 2-5 y 37%, > 5y 23%	0	10.3 (24.0)	
25135680	Wightman	2014	UNOS	US	2008-2011	nd	2076	All Txp	First Txp, excluded multi-organ Txp	children	nd	<5 y 10%, 5-12 y 31%, 13-18 y 59%	57	50	19	3	nd	nd	nd	27	American Indian/Alaska Native 0.7%, Hawaiian/Other 0.4%, multiracial 1%	15	0	0	structural 37%, FSGS 15%, other not reported 34%, missing 1.4%	nd	0	nd	

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25098499	Xia	2014	SRTR/OPTN	US	2000-2013	3 years	486	All Txp	Kidney alone Txp, deceased donor	adults	Excluded HBVsAg +, had missing or unknown HIV or HCV serostatus or received a previous liver transplant	50.7 (11.4)	72	nd	62	nd	nd	nd	nd	nd	nd	nd	nd	21	nd	> 3 years 72%	nd	8	>30%, 30%	
26636735	Barracough	2016	ANZDATA	Australia, New Zealand	2000-2012	nd	7826	All Txp	Kidney txp alone, recipients of multiple organ transplants were excluded	adults	Recipients of multiple organ transplants were excluded	18-44: 38.6% 46-64: 52.6% 65+: 8.8%	62.8	nd	nd	nd	nd	nd	nd	nd	Indigenous: Australian: nonIndigenous Australian: 96.5%	nd	nd	14	nd	nd	nd	2+ graft number: 8.0%	0-9%: 72.3% 10-49%: 17.2% ≥50%: 10.1%	
28010785	Lim	2017	ANZDATA	Australia, New Zealand	1994-2012	median 6.5 years	10,714	All txp	All primary living and deceased donor kidney transplant recipients	nd	recipients of multiple-organ transplants, recipients of kidney transplants who had received two or more grafts between 1994 and 2012, recipients with type 1 diabetes, and those without documented diabetes status were included	49.2	62.1	80.6	nd	nd	nd	nd	nd	nd	nd	Indigenous: 8.2% other: 11.2%	44.2	4.5	9.2	42.1	nd	nd	nd	nd
28361229	Ladhani	2017	ANZDATA	Australia, New Zealand	1994-2013	median 8.4 years	750	Children receiving first txp	Kidney txp	2-18 years	registry is a comprehensive database of all children and adults who have received renal replacement therapy since 1965 in Australia and New Zealand	2-6: 23.6% 7-10: 20.9% 11-15: 28.8% 16+: 26.7%	58.3	79.6	nd	nd	nd	nd	nd	nd	nd	Indigenous: 8.3% other: 16.7%	30.8	nd	nd	69.2	nd	nd	0	0-25: 87.2% 26-50: 4.1% 51-75: 4.3% 76-100: 2.9%
26924061	Pruthi	2016	UKRR (UK Renal Registry)	UK	1997-2009	nd (through December 2012)	4750	Incident renal transplant patients in the UK, aged >16 years with a primary renal diagnosis of GN or APKD	Incident renal transplant patients with primary renal diagnosis of GN or APKD	>16 years	nd	GN group: median 45 ADPKD group: median 53	62	89	4	5	nd	nd	nd	nd	2	62.6	nd	nd	ADPKD: 37.4	GN group: median 1.9 years ADPKD group: median 1.6 years	nd	nd	nd	nd

Pubmed id	Authors	Year	Name of database	Country	Period of patient recruitment	Length of follow-up (mean/median)	N analyzed	Eligibility criteria: General	Eligibility criteria: CKD specific	Eligibility criteria: Age specific	Eligibility criteria: Others	Age, mean (SD)/ median (range), years	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian (total), %	Race, East Asian, %	Race, South Asian, %	Race, Middle Eastern, %	Race, Hispanic, %	Race, Other, %	Primary kidney disease, GN, %	Primary kidney disease, HTN, %	Primary kidney disease, DM, %	Primary kidney disease, Other, %	Dialysis duration	Dialysis modality	Repeat or h/o KTxp, %	Panel reactive antibody, % or mean (SD)					
<b>PREDICTORS OF GRAFT LOSS</b>																																		
24370342	Tancredi	2014	OPTN	US	2000-2010	1 year (for graft failure)	6032	All Txp	First KTxp	<18 years	No h/o combined organ Txp, had a functioning graft on postop day 1; albumin, HLA mismatch level, h/o dialysis available	10.9 (5.2)	59	53	19	nd	nd	nd	nd	23	nd	nd	nd	nd	nd	nd	nd	nd	nd	congenital/ structural causes in 47%, FSGS in 14%, other glomerular diseases in 26%, malignancies in 1%, other causes in 7%, and unknown cause in 5%	nd	HD 34%, PD 34%, no dialysis 31%	0	nd
12110738	Briganti	2002	ANZDATA	Australia, New Zealand	1988-1997	10 y	1505	biopsy-proven GN	first KTxp	nd	nd	median 46, IQR 36-57	68	nd	nd	nd	nd	nd	nd	nd	nd	100	0	0	0	median 15, IQR 8-20	nd	0	median 6, IQR (0-45)					
23295317	Cannon	2012	UNOS	US	2004-2009	nd	74983	All Txp	Kidney-alone transplant	nd	BMI 10-60	48 (16)	60	55	24	5	nd	nd	nd	14	nd, 2%	nd	21	16	nd	nd	nd	nd	16.0% (29.1)					
21797974	Clayton	2011	ANZDATA	Australia, New Zealand	1988-2007	median 6.7y	1521	biopsy-proven IgAN	>=16 years	primary kidney-only txp	nd	43 (11.9)	76	80	nd	nd	nd	nd	nd	nd	20	0	0	0	IgAN 100%	0-6 months 0%, 6 months-<1 year 17%, 1 to <5 years 49%, >=5 years 14%	nd	0	<=50% 92%, >50% 7%					
22124283	Foster	2011	USRDS	US	1988-2009	median 5.9 y	90689	first Ktxp	<40 y	nd	nd	0-4y 2.6%, 5-9y 2.8%, 10-12y 2.4%, 13-16y 5.5%, 17-20y, 7.0%, 21-24y 9.3%, 25-29y 17.9%, 30-34y 24.1%, 35-39y 28.5%	57.8	69.2	23.5	nd	nd	nd	nd	nd	7.3	28.3	nd	nd	CAKUT 8.2%, FSGS 8.3%, unknown 22.9%, other 32.3%	median 13.8 months (IQR: 4.2-31.0)	nd	0	nd					

Pubmed id	Authors	Year	Name of database	Country	Period of patient recruitment	Length of follow-up (mean/median)	N analyzed	Eligibility criteria: General	Eligibility criteria: CKD specific	Eligibility criteria: Age specific	Eligibility criteria: Others	Age, mean (SD)/ median (range), years	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian (total), %	Race, East Asian, %	Race, South Asian, %	Race, Middle Eastern, %	Race, Hispanic, %	Race, Other, %	Primary kidney disease, GN, %	Primary kidney disease, HTN, %	Primary kidney disease, DM, %	Primary kidney disease, Other, %	Dialysis duration	Dialysis modality	Repeat or h/o KTxp, %	Panel reactive antibody, % or mean (SD)
23406350	Heaphy	2013	SRTR	US	1995-2010	nd	109392	deceased-donor ktxp	first Txp of any organ	>18 y	exclusions: cold ischemia times less than 1 h (n = 156) or greater than 60 h (n = 378); donors listed as less than 1 year or greater than 80 years of age (n = 602); recipients with a creatinine level greater than the 99th percentile of the sample equivalent to a value of 4.0 (n = 1340); missing donor height (n = 323), donor weight (n = 3) and donor creatinine (n = 618), donors with a BMI less than 13 or greater than 50 (n = 3403); and multiorgan transplants (n = 3265)	median 52, IQR 42-61	60.7	48.4	31.7	5.3	nd	nd	nd	12.8	multiracial 0.2%, american indian/alaskan native 1.1%, hawaiian/other pacific islander 0.5%	nd	nd	nd	nd	nd	nd	0	median 0, IQR 0-0 (0.73.1%, 1-30.15.9%, 31-80.5.3%, >=81.2.4%)
20814353	Huang	2010	OPTN/UNOS	US	2000-2008	at least 2 years	31179	All Txp	nd	>60 yo	nd	median (IQR): 60-69yo 64 (61, 66), 70-79yo 63 (61, 66), >=80yo 81 (80, 82)	63	63	20	nd	nd	nd	nd	10	nd	11	26	12	other not reported 51%	preemptive 17%, >3y 32%	nd	nd	peak PRA>20%: 4%
26660200	Ilori	2015	OPTN/UNOS	US	1996-2010	nd	44013	All Txp	nd	>=60 years	nd	median (IQR) 65 (7, 0)	63	62	20	5	nd	nd	nd	11	nd, 2%	15	25	34	cystic kidney disease 9%, other unknown 17%	median (IQR) 2.47 years (2.81)	nd	nd	nd
24009216	Kainz	2013	OEDTR	Austria	1992-2011	median 7.41 years	553	All Txp	First KTxp	nd	Underwent echo 1 year before KTxp (all pt who were potentially eligible for renal allograft wait-listing underwent a baseline echo with annual f/u while being listed)	52 (13)	58	100 (based on study conducted in Austria)	0	0	0	0	0	0	0	23	nd	14	vascular 9%, other unknown 55%	median (IQR), LA2D<=53mm 1.9 yr (0.8, 3.2), LA2D>53mm 1.8 (0.9, 3.2)	nd	0	median (IQR), LA2D<=53mm 0% (0, 0), LA2D>53mm 0% (0, 4)
27336396	Kang	2016	UNOS	US	2005-2013	3.9 years	104632	All Txp	KTxp alone, not foreign donor kidney recipients	>=18 yo	excluded recipients with a pretransplant cancer other than skin cancer without coexisting skin cancer	median (IQR): w/o pre-Txp skin cancer 53 (42, 61), w/ pre-Txp skin cancer 64 (57, 70)	61	50	27	6	nd	nd	nd	15	nd	nd	nd	34% reported as comorbidity	nd	nd	nd	0	nd
20801565	Kasike	2010	USRDS/OPTN	US	2000-2006	5 years?	59001	All Txp	deceased donor	>=18 yo	excluded multiorgan recipients and prior recipients of nonkidney organs	50 (13)	nd	61	31	6	nd	nd	nd	nd	unknown 2.4%	25	22	25	syctic disease 8.8%, other not reported 18.7%	nd	nd	nd	nd

Pubmed id	Authors	Year	Name of database	Country	Period of patient recruitment	Length of follow-up (mean/median)	N analyzed	Eligibility criteria: General	Eligibility criteria: CKD specific	Eligibility criteria: Age specific	Eligibility criteria: Others	Age, mean (SD)/ median (range), years	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian (total), %	Race, East Asian, %	Race, South Asian, %	Race, Middle Eastern, %	Race, Hispanic, %	Race, Other, %	Primary kidney disease, GN, %	Primary kidney disease, HTN, %	Primary kidney disease, DM, %	Primary kidney disease, Other, %	Dialysis duration	Dialysis modality	Repeat or h/o KTxp, %	Panel reactive antibody, % or mean (SD)
21449945, 27391198, 22156753	Molnar	2011, 2015, 20	SRTR, DaVita	US	2001-2007	median: 717 days, IQR (356, 1206)	14508	All Txp	First KTxp, on HD or PD before Txp	nd	nd	48 (14)	61	nd	27	nd	nd	nd	nd	nd	nd	nd	nd	27 reported as comorbidity	nd	0-6 m 12%, 6-24 m 29%, 2-5 y 36%, >5 y 23%	HD 86%, PD 14%	0	10.1% (24.0)
							8961	All Txp	First KTxp, on HD before Txp	nd	Excluded pts without electronically recorded serum albumin levels in the last quarter prior to transplantation, lacked data from the baseline quarter, with outlier values for age																		
							10083	All Txp	First KTxp, on HD before Txp	>=18 yo	nd																		
19353768	Mulay	2009	USRDS	US	1990-2003	median 51 months	41272	primar cause of renal failure was primary or secondary GN	first KTxp	nd	nd	40.2 (14.9)	56.7	70.8	21.9	nd	nd	nd	nd	nd	7.3	100	0	0	0	0-12 months 27.8%, 12-36 months 37.3%, >36 month, 26.2%	nd	0	>50% 7.8%
26569067	Naik	2016	OPTN/UNOS	US	2001-2009	median 5.5-6.0 years	108654	All Txp	First KTxp	Adults	No h/o other organ Txp (BMI data available, although not mentioned in the article)	49 (13)	58	54	26	5	nd	nd	nd	14	2	nd	nd	nd	nd	nd	nd	0	<=30% 60%, >20% 9%

Pubmed id	Authors	Year	Name of database	Country	Period of patient recruitment	Length of follow-up (mean/median)	N analyzed	Eligibility criteria: General	Eligibility criteria: CKD specific	Eligibility criteria: Age specific	Eligibility criteria: Others	Age, mean (SD)/ median (range), years	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian (total), %	Race, East Asian, %	Race, South Asian, %	Race, Middle Eastern, %	Race, Hispanic, %	Race, Other, %	Primary kidney disease, GN, %	Primary kidney disease, HTN, %	Primary kidney disease, DM, %	Primary kidney disease, Other, %	Dialysis duration	Dialysis modality	Repeat or h/o KTxp, %	Panel reactive antibody, % or mean (SD)
26102616	Opelz	2016	CTS	Germany	1995-2012	10 years	46548	All Txp	First KTxp	>=18 years	No h/o combined organ Txp, smoking status was documented at the time of Txp	>60 yo: 18%	62	73	nd	nd	nd	nd	nd	nd	nd, 30%	nd	nd	nd	nd	nd	nd	0	nd
24070588	Pieloch	2014	UNOS	US	2001-2006	3 years	30132	All Txp	First Txp	adults	excluded pt with multiorgan Txp	48.4 (13.9)	57	56	22	nd	nd	nd	nd	13	unknown 8%	nd	nd	nd	nd	nd	82	0	nd
25758804	Pieloch	2015	OPTN/UNOS	US	2000-2008	3 years	100261	All Txp	nd	adults	excluded pt with multiorgan Txp	18-49 49%, 50-64 38%, >=65 13%	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	29% reported as comorbidity	nd	0 years 10%, 0-4 55%, 4-24%	nd	nd	nd	nd
21566110	Reddy	2011	OPTN/UNOS	US	2001-2007	3 years	75681	All Txp	First Txp	>18 yo	excluded multiorgan Txp, pt with pre-Txp HCV infection, included pt with at least one follow-up visit reported to OPTN/UNOS	>= 60 yo 26%	60	56	23	5	nd	nd	nd	13	unknown 2.2%	20	23.4	25	unknown 21%	no 19%, < 1 year 18%, 1-3 years 31%, >= 3 years 33%	nd	0	>= 10% 18%
21415312	Streja	2011	SRTR/MHD	US	2001-2007	2.3 years	10090	All Txp	First Txp	nd	nd	49 (13)	51	nd	27	4	nd	nd	nd	15	nd	nd	nd	45% as comorbidity	nd	< 6 m 12%, 6-24 m 29%, 2-5 y 37%, > 5y 23%	0	10.3 (24.0)	
25135680	Wightman	2014	UNOS	US	2008-2011	nd	2076	All Txp	First Txp, excluded multi-organ Txp	children	nd	<5 y 10%, 5-12 y 31%, 13-18 y 59%	57	50	19	3	nd	nd	nd	27	American Indian/Alaska Native 0.7%, native Hawaiian/Other 0.4%, multiracial 1%	15	0	0	structural 37%, FSCS 15%, other not reported 34%, missing 1.4%	nd	0	nd	
25098499	Xia	2014	SRTR/OPTN	US	2000-2013	3 years	486	All Txp	Kidney alone Txp, deceased donor	adults	Excluded HBVsAg +, had missing or unknown HIV or HCV serostatus or received a previous liver transplant	50.7 (11.4)	72	nd	62	nd	nd	nd	nd	nd	nd	nd	nd	21	nd	> 3 years 72%	nd	8	>30%, 30%
26636735	Barracough	2016	ANZDATA	Australia, New Zealand	2000-2012	nd	7826	All Txp	Kidney txp alone, recipients of multiple organ transplants were excluded	adults	Recipients of multiple organ transplants were excluded	18-44: 38.6% 46-64: 52.6% 65+ 8.8%	62.8	nd	nd	nd	nd	nd	nd	nd	indigenous 3.5% nonindigenous Australian: 96.5%	nd	nd	14	nd	nd	nd	2+ graft number: 8.0%	0-9%: 72.3% 10-49%: 17.2% ≥50%: 10.1%

Pubmed id	Authors	Year	Name of database	Country	Period of patient recruitment	Length of follow-up (mean/median)	N analyzed	Eligibility criteria: General	Eligibility criteria: CKD specific	Eligibility criteria: Age specific	Eligibility criteria: Others	Age, mean (SD)/ median (range), years	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian (total), %	Race, East Asian, %	Race, South Asian, %	Race, Middle Eastern, %	Race, Hispanic, %	Race, Other, %	Primary kidney disease, GN, %	Primary kidney disease, HTN, %	Primary kidney disease, DM, %	Primary kidney disease, Other, %	Dialysis duration	Dialysis modality	Repeat or h/o KTxp, %	Panel reactive antibody, % or mean (SD)	
28361229	Ladhani	2017	ANZDATA	Australia, New Zealand	1994-2013	median 8.4 years	750	Children receiving first txp	Kidney txp	2-18 years	registry is a comprehensive database of all children and adults who have received renal replacement therapy since 1965 in Australia and New Zealand	2-6: 23.6% 7-10: 20.9% 11-15: 28.8% 16+: 26.7%	58.3	79.6	nd	nd	nd	nd	nd	nd	nd	indigenous: 8.3% other: 16.7%	30.8	nd	nd	69.2	nd	nd	0	0-25: 87.2 26-50: 4.1 51-75: 4.3 76-100: 2.9
26924061	Pruthi	2016	UKRR (UK Renal Registry)	UK	1997-2009	nd (through December 2012)	4750	Incident renal transplant patients in the UK, aged >16 years with a primary renal diagnosis of GN or APKD	Incident renal transplant patients with primary renal diagnosis of GN or APKD	>16 years	nd	GN group: median 45 ADPKD group: median 53	62	89	4	5	nd	nd	nd	nd	2	62.6	nd	nd	ADPKD: 37.4	GN group: median 1.9 years ADPKD group: median 1.6 years	nd	nd	nd	

Pubmed id	Authors	Year	Name of database	Country	Period of patient recruitment	Length of follow-up (mean/median)	N analyzed	Eligibility criteria: General	Eligibility criteria: CKD specific	Eligibility criteria: Age specific	Eligibility criteria: Others	Age, mean (SD)/ median (range), years	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian (total), %	Race, East Asian, %	Race, South Asian, %	Race, Middle Eastern, %	Race, Hispanic, %	Race, Other, %	Primary kidney disease, GN, %	Primary kidney disease, HTN, %	Primary kidney disease, DM, %	Primary kidney disease, Other, %	Dialysis duration	Dialysis modality	Repeat or h/o KTxp, %	Panel reactive antibody, % or mean (SD)
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**PREDICTORS OF OTHER OUTCOMES**

27336396	Kang	2016	UNOS	US	2005-2013	3.9 years	104632	All Txp	KTxp alone, not foreign donor kidney recipients	>=18 yo	excluded recipients with a pretransplant cancer other than skin cancer without coexisting skin cancer	median (IQR): w/o pre-Txp skin cancer 53 (42, 61), w/ pre-Txp skin cancer 64 (57, 70)	61	50	27	6	nd	nd	nd	15	nd	nd	nd	34% reported as comorbidity	nd	nd	nd	0	nd
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17198258	Shah	2006	OPTN/UNOS	US	2004-2005	306 days	15309	All Txp	First KTxp	>20 yo	included those had at least on f/u and non-diabetic	>60 yo, 17.8%	59	55.8	23.3	5.1	nd	nd	nd	11.9	not specified 3.9%	nd	77.6% reported as comorbidity	0	nd	nd	nd	0	nd
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**PRE-EMPTIVE vs. EARLY DIALYSIS**



Pubmed id	Authors	Year	Name of database	Country	Period of patient recruitment	Length of follow-up (mean/median)	N analyzed	Eligibility criteria: General	Eligibility criteria: CKD specific	Eligibility criteria: Age specific	Eligibility criteria: Others	Age, mean (SD)/ median (range), years	Sex, male, %	Race, White, %	Race, Black, %	Race, Asian (total), %	Race, East Asian, %	Race, South Asian, %	Race, Middle Eastern, %	Race, Hispanic, %	Race, Other, %	Primary kidney disease, GN, %	Primary kidney disease, HTN, %	Primary kidney disease, DM, %	Primary kidney disease, Other, %	Dialysis duration	Dialysis modality	Repeat or h/o KTxp, %	Panel reactive antibody, % or mean (SD)
27653837	Amaral	2016	USRDS	US	2000-2012	4.8 years	7527	All Txp	nd	<18 yo	included those entered medicare program	10.8 (5.3)	59	71	17	nd	nd	nd	nd	22 (hispanic white)	12	15 (including 6% secondary GN)	nd	nd	CAKUT 46%, FSGS 13%, lupus 2%, others unknown 25%	nd	nd	nd	<20% 73%, 20-80% 19%, >80% 8%
23371953	Grams	2013	SRTR	US	1995-2011	nd	18976	deceased-donor ktxp	first KTxp	adults	nd	52.7 (12.5)	44.8*	57.3*	20.3*	nd	nd	nd	nd	nd	22.4*	7.7*	5*	5.5*	81.8*	nd	nd	0	<=40% 55%

**KDIGO - Transplant Candidate**  
**Guideline Topic: Registry studies**  
**Categorical outcomes**

Pubmed id	Authors	Year	Outcome	Outcome definition	% w/ outcome	% w/o outcome	Primary Predictor	Predictor definition	% w/ predictor	% w/o predictor	Metric	Estimate, mean (95% CI)	P value	Adjustment, Other covariates (list once)	Methodological quality	Notes
<b>PREDICTORS OF MORTALITY</b>																
23295317	Cannon	2012	Mortality	all-cause mortality	3.9% 1 year, 7.5% 3 years, 13.4% 5 years	96.9% 1 year, 92.5% 3years, 86.6% 5 years	Class I Obesity	BMI 30-35	20	70.3	HR	0.92 (0.86, 0.99)	0.025	recipient age, race, gender, CVA as donor cause of death, donor type, cold ischemic time, HLA mismatch, other causes of renal failure, previous KTxp	A	
							Class II Obesity	BMI 30-35	7.7		HR	1.06 (0.96, 1.18)	0.244			
							Class III Obesity	BMI>=40	2.1		HR	1.15 (0.95, 1.39)	0.151			
							Diabetic nephropathy	nd	21	79	HR	1.61 (1.50, 1.73)	<0.001			
							Hypertensive nephropathy	nd	16	84	HR	1.10 (1.02, 1.19)	0.012			
24138318	Farrugia	2014	Mortality	1-year mortality postkidney Txp	566 deaths	nd	Age 50-59 (vs <50)	Age 50-59 (vs <50)	nd	nd	HR	2.38 (1.95, 2.90)	<0.001	sex, race, living donor Txp, allograft failure	A	
							Age 60-69 (vs <50)	Age 60-69 (vs <50)	nd	nd	HR	4.46 (3.68, 5.39)	<0.001			
							Age 70-79 (vs <50)	Age 70-79 (vs <50)	nd	nd	HR	7.62 (5.84, 9.94)	<0.001			
							Age >=80 (vs <50)	Age >=80 (vs <50)	nd	nd	HR	15.72 (4.98, 49.60)	<0.001			
							Socioeconomic deprivation 2 (vs 1)	Socioeconomic deprivation 2 (IMD 2010) (vs 1), 1- most deprived	alive 21.9%, death 25.6%	alive 21.9%, death 25.6%	HR	0.84 (0.68, 1.05)	0.124			
							Socioeconomic deprivation 3 (vs 1)	Socioeconomic deprivation 3 (IMD 2010) (vs 1), 1- most deprived	alive 19.8%, death 17.7%	alive 19.8%, death 17.7%	HR	0.86 (0.69, 1.08)	0.193			
							Socioeconomic deprivation 4 (vs 1)	Socioeconomic deprivation 4 (IMD 2010) (vs 1), 1- most deprived	alive 18.2%, death 18.9%	alive 18.2%, death 18.9%	HR	0.86 (0.68, 1.08)	0.19			
							AMI	acute myocardial infarction	alive 2.4%, death 9.7%	alive 97.6%, death 91.3%	HR	1.52 (1.15, 2.01)	0.003			
							CHF	congestive hear failure	alive 0.6%, death 2.7%	alive 99.4%, death 97.3%	HR	1.51 (0.77, 2.93)	0.229			
							PVD	perpheral vascular disease	alive 0.7%, death 2.7%	alive 99.3%, death 97.3%	HR	1.70 (1.17, 2.47)	0.006			
							CVA	cerebral vascular accident	alive 1.4%, death 4.6%	alive 98.6%, death 95.4%	HR	1.66 (0.91, 3.03)	0.097			
							DM	diabetes	alive 15.2%, death 25.4%	alive 84.8%, death 74.6%	HR	1.64 (1.38, 1.93)	<0.001			
20814353	Huang	2010	Mortality	all-cause mortality?	nd	nd	Age >= 80 (vs. 60-69)	nd	0.6	79.8	HR	2.42 (1.91, 3.06)	nd	transplant year, recipient age, recipient gender, recipient race, dialysis duration, retransplantation, peak PRA, recipient comorbidities (diabetes, cardiovascular disease, peripheral vascular disease, and cerebrovascular disease), donor type, donor age, degree of human leukocyte antigen mismatch, induction therapy, tacrolimus use, mycophenolate use, and steroid use	A	
							Age 70-79 (vs. 60-69)	nd	19.6		HR	1.42 (1.34, 1.51)	nd			
26660200	Ilori	2015	Death	nd	37.4%	62.6%	Age	10-year change, all pts >= 60 yo	nd	nd	HR	1.47 (1.42, 1.52)	nd	race and ethnicity, any acute rejection, end-stage renal disease (ESRD) etiology, sex, human leukocyte antigen (HLA) mismatch, pretransplantaiton dialysis, type of donor, donor age, cold ischemia time, insurance, neighborhood poverty, and period of transplantation	A	
24009216	Kainz	2013	Death	death with and without censored graft loss	33.6% in upper LA2D stratum in 10 years, 16.3% in lower LA2D stratum in 10 years	66.4% in upper LA2D stratum in 10 years, 83.7% in lower LA2D stratum in 10 years	left atrial diameter (mm)	continuous variable by echo in mm	na	na	HR	1.06 (1.03, 1.08)	<0.001	left atrial diameter, right ventricular diameter, perihervascular disease, HBG, immunosuppression, calcineurin inhibitor use, afib	B	
							right ventricular diameter (mm) perihervascular disease (yes versus no)	continuous variable by echo in mm nd, yes vs no	na	na	HR	0.95 (0.90, 1.01)	0.12			HR
27336396	Kang	2016	Mortality	all-cause mortality?	8-years post-Txp w/ pre-Txp skin cancer 42.8%, w/o pre-Txp skin cancer 28.4%	8-years post-Txp: w/ pre-Txp skin cancer 57.2%, w/o pre-Txp skin cancer 71.6%	Pre-Txp skin cancer (vs. no pre-Txp skin cancer)	nd	1.6	98.4	HR	1.20 (1.07, 1.34)	<0.001 (from log-rank test)	adjusted for sex, age, BMI, ethnicity, EBV, HBV, HCV, serostatus, dialysis duration, and induction therapy	B	
							Pre-Txp skin cancer excluding those with solid cancer (vs. no pre-Txp skin cancer)	nd	1.4	98.6	HR	1.17 (1.04, 1.32)	<0.001 (from log-rank test)			adjusted for sex, age, BMI, ethnicity, EBV, HBV, HCV, serostatus, dialysis duration, and induction therapy
26147285	Krishnan	2015	Death	all cause mortality	2.8%	97.2%	BMI <18.5	BMI <18.5	2.9	41.1	HR	1.96 (0.90, 4.30)	0.0912	recipient gernder, age, race, primary diagnosis, donor status, age, sex, race, rejection, HLA mismatch	B	
							BMI 25-<30	BMI 25-<30	35.5		HR	0.94 (0.68, 1.29)	0.6858			
							BMI 30-<35	BMI 30-<35	16.8		HR	0.73 (0.47, 1.13)	0.1628			
							BMI 35-<40	BMI 35-<40	3.3		HR	0.48 (0.15, 1.53)	0.2163			
26720436	Lynch	2016	Mortality	all-cause mortality	nd	nd	Age	as continuous variable, per yr	na	na	HR	1.04 (1.04, 1.04)	<0.0001	sex, race, h/o Txp, dialysis vintage, donor type, new onset of comorbidity, no. inpatient days in pre-Txp year	B	
							Diabetes	nd	29.2	70.8	HR	1.39 (1.31, 1.47)	<0.0001			
							CHF	nd	12.7	87.3	HR	1.22 (1.13, 1.31)	<0.0001			
							CVD	nd	3.1	96.9	HR	1.16 (1.02, 1.32)	0.02			
PVD	nd	4.5	95.5	HR	1.15 (1.03, 1.27)	0.01										
COPD	nd	1.7	98.3	HR	1.20 (1.02, 1.41)	0.03										

Pubmed id	Authors	Year	Outcome	Outcome definition	% w/ outcome	% w/o outcome	Primary Predictor	Predictor definition	% w/ predictor	% w/o predictor	Metric	Estimate, mean (95% CI)	P value	Adjustment, Other covariates (list once)	Methodological quality	Notes			
21449945, 27391198, 22156753	Molnar	2011, 2015, 20	Mortality	Graft failure censored all-cause death	7.0	93.0	PD vs. HD	nd	14.0	86.0	HR	0.57 (0.38, 0.87)	0.009		A				
			Death, all-cause, graft loss censored	all-cause mortality	8.0	92.0	albumin	by 0.2 g/dl, as continuous	nd	nd	HR	0.87 (0.82, 0.93)	<0.001	age, gender, race-ethnicity, diabetes mellitus, dialysis vintage, primary insurance, marital status, standardized mortality ratio of the dialysis clinic during entry quarter, dialysis dose as indicated by KU/V (single pool), presence or absence of a dialysis catheter and residual renal function during the entry quarter, body mass index (BMI), the normalized protein nitrogen appearance (nPNA) and serum or blood concentrations of TIBC, ferritin, phosphorus, calcium, bicarbonate, peripheral white blood cell count (WBC), lymphocyte percentage and hemoglobin, donor type, donor age, panel reactive antibody (PRA) titer (last value prior to transplant), number of HLA mismatches, cold ischemia time and extended donor criteria	albumin has no significant interacting effect with age, gender, race, hemoglobin, BMI, DM				
			Mortality	all-cause mortality?	9.9% in the entire cohort of 15125 pt	90.1	Age 18-34 (vs. 50-64)	nd	nd	nd	HR	0.41 (0.31, 0.54)	<0.001	recipient race, type of insurance, time on dialysis, donor's age, DM					
							Age 35-49 (vs. 50-64)	nd	nd	nd	HR	0.60 (0.50, 0.71)	<0.001						
							Age >=65 (vs. 50-64)	nd	nd	nd	HR	1.63 (1.40, 1.90)	<0.001						
							DM (presence vs. absence)	nd	37.0	63.0	HR	1.53 (1.34, 1.74)	<0.001						
							CAD (presence vs. absence)	nd	7.0	93.0	HR	1.38 (1.15, 1.65)	<0.001						
							PVD (presence vs. absence)	nd	7.0	93.0	HR	1.38 (1.13, 1.69)	0.002						
							Serum albumin	by 1 g/dl, as continuous	nd	nd	nd	HR	0.62 (0.52, 0.75)	<0.001					
			26102616	Opelz	2016	Death, all-cause	nd	6068 events	nd	Stopped smoking (vs. Never smoking)	nd	22.1	67.6	HR	1.1 (1.0, 1.2)	<0.001			
Death with a functioning graft due to CVD	nd	nd				nd	Continued smoking (vs. Never smoking)	nd	10.3	HR	1.6 (1.5, 1.8)	<0.001							
Death with a functioning graft due to malignancy	nd	nd				nd	Stopped smoking (vs. Never smoking)	nd	22.1	67.6	HR	1.1 (1.0, 1.3)	0.075						
							Continued smoking (vs. Never smoking)	nd	10.3	HR	1.6 (1.4, 1.9)	<0.001							
							Stopped smoking (vs. Never smoking)	nd	22.1	67.6	HR	1.4 (1.2, 1.7)	0.001						
24070588	Pieloch	2014	Mortality	3 year, all cause mortality?	nd	nd	Morbid obesity	BMI 35-40 kg/m2	20	80	HR	2.6 (2.1, 3.1)	<0.001		C				
25758804	Pieloch	2015	Mortality	all-cause mortality	KTMI score=0 1.8%, 1 3.4%, 2 6.3%, 3 10.3%, 4 15.2%, 5 19.2%, 6 24.0%, >=7 25.3%	KTMI score=0 98.2%, 1 96.6%, 2 93.7%, 3 89.7%, 4 84.8%, 5 80.8%, 6 76.0%, >=7 74.7%	KTMI score 1 (vs score 0)	Kidney Transplant Morbidity Index score= 1	22.2	6.4	HR	1.85 (1.45, 2.36)	<0.001	human leukocyte antigen mismatch, cold ischemic time, donor age, and donor type					
							KTMI score 2 (vs score 0)	Kidney Transplant Morbidity Index score= 2	27.6	HR	3.11 (2.46, 3.94)	<0.001							
							KTMI score 3 (vs score 0)	Kidney Transplant Morbidity Index score= 3	22.8	HR	5.00 (3.96, 6.31)	<0.001							
							KTMI score 4 (vs score 0)	Kidney Transplant Morbidity Index score= 4	13.3	HR	7.37 (5.83, 9.32)	<0.001							
							KTMI score 5 (vs score 0)	Kidney Transplant Morbidity Index score= 5	5.5	HR	9.41 (7.41, 11.94)	<0.001							
							KTMI score 6 (vs score 0)	Kidney Transplant Morbidity Index score= 6	1.7	HR	12.51 (9.45, 15.83)	<0.001							
							KTMI score >= 7 (vs score 0)	Kidney Transplant Morbidity Index score>= 7	0.5	HR	13.03 (9.68, 17.54)	<0.001							
21566110	Reddy	2011	Mortality in living donor Txp	all cause mortality?	5 years all recipients, HBV+ 14.7%, HBV- 14.4%	5 years all recipients, HBV+ 85.3%, HBV- 85.6%	HBV infection (vs. HBV-)	HBsAg +ve	all recipients 1.8%	all recipients 98.2%	HR	0.98 (0.59, 1.63)	nd						
			Mortality in deceased donor Txp	all cause mortality?	5 years all recipients, HBV+ 14.7%, HBV- 14.4%	5 years all recipients, HBV+ 85.3%, HBV- 85.6%	HBV infection (vs. HBV-)	HBsAg +ve	all recipients 1.8%	all recipients 98.2%	HR	1.09 (0.88, 1.36)	nd						
21415312	Streja	2011	Mortality	graft failure censored death	7.8%	92.2%	BMI	as continuous variable, based on each 1 kg/m2 higher BMI	na	na	HR	0.99 (0.98, 1.02)	0.91	age, sex, race, ethnicity, diabetes mellitus, dialysis vintage, primary insurance, marital status, the standardized mortality ratio of the dialysis clinic during entry quarter, dialysis dose as indicated by KU/V (single pool), presence or absence of a dialysis catheter, and residual renal function during the entry quarter (i.e., urinary urea clearance)	B				
							Creatinine	as continuous variable, based on each 1 mg/dl higher scr	na	na	HR	0.91 (0.86, 0.95)	<0.001						
25135680	Wightman	2014	Mortality	all cause mortality	0.9%	99.1%	Definite intellectual disability	identified as "definitely cognitive delay/impairment" by their center	5.6	84.1	HR	0.3 (0.2, 12.2)	0.752	age in years (<5, 5-12, 13-18), male gender, race (white/nonwhite), etiology (structural, FSGS, GN, other), deceased donor (Y/N), cold ischemia time >24 hrs (Y/N), HLA match, PRA/CPRA (<10%, 10-80%, 80-100%)					
							Probable intellectual disability	"probable" or "questionable" cognitive delay/impairment, "reduced academic load/nonparticipation," or "delayed grade level/special education"	10.3	HR	0.2 (0.1, 1.3)	0.752							

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25098499	Xia	2014	Patient survival	patient mortality from any cause following transplantat ion	12.6	87.4	HIV seropositive (vs. negative)	nd	50.0	50.0	HR	0.80 (0.39, 1.64)	nd			
26636735	Barracough	2016	Overall survival	Patient death	333	7171	≥Obese	nd	nd	nd	HR	0.96 (0.77, 1.20)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term	A	
							Overweight	nd	nd	nd	HR	0.91 (0.75, 1.10)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term		
							Smoker	nd	nd	nd	HR	1.20 (1.01, 1.43)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term		
							CVD	nd	nd	nd	HR	1.39 (1.01, 1.91)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term		
							DM	nd	nd	nd	HR	1.43 (1.14, 1.78)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term		
							Age 45-64	nd	52.6	47.4	HR	0.63 (0.56, 0.71)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term		
							Age ≥65	nd	8.8	91.2	HR	0.47 (0.37, 0.60)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term		
28010785	Lim	2017	All-cause mortality	nd	nd	nd	Age <40 years (DM vs. no DM)	nd	34.7	65.3	HR	5.16 (2.84, 9.35)	nd	donor age, donor type, waiting time, prevalent cardiovascular disease, ethnic origin, total ischemic time, prevalent peripheral vascular disease, prevalent cerebrovascular disease, BMI, smoking, era, and peak panel reactive antibody	A	
							Age 40-55 years (DM vs. no DM)	nd	37.9	62.1	HR	2.08 (1.62, 2.66)	nd	donor age, donor type, waiting time, prevalent cardiovascular disease, ethnic origin, total ischemic time, prevalent peripheral vascular disease, prevalent cerebrovascular disease, BMI, smoking, era, and peak panel reactive antibody		
							Age >55 years (DM vs. no DM)	nd	27.4	72.6	HR	1.41 (1.17, 1.71)	nd	donor age, donor type, waiting time, prevalent cardiovascular disease, ethnic origin, total ischemic time, prevalent peripheral vascular disease, prevalent cerebrovascular disease, BMI, smoking, era, and peak panel reactive antibody		
28361229	Ladhani	2017	Overall death	all-cause mortliaty	53	697	Obese	nd	8.1	91.9	HR	0.80 (0.25, 2.61)	nd	adjusted for age at transplant, HLA mismatch, and year of transplant	A	
							Overweight	nd	17.2	82.8	HR	0.85 (0.38, 1.92)	nd	adjusted for age at transplant, HLA mismatch, and year of transplant		
							Underweight	nd	64.4	35.6	HR	1.18 (0.25, 2.61)	nd	adjusted for age at transplant, HLA mismatch, and year of transplant		
26924061	Pruthi	2016	Patient survival	nd	nd	nd	ADPKD	nd	nd	nd	HR	reference	reference	nd	A	
							Crescentic GN	nd	nd	nd	HR	1.11 (0.65, 1.90)	0.7	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							FSGS	nd	nd	nd	HR	1.12 (0.75, 1.66)	0.6	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							GN histologically not examined	nd	nd	nd	HR	1.13 (0.78, 1.63)	0.5	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							GN histologically proven	nd	nd	nd	HR	1.13 (0.86, 1.49)	0.4	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							IgA nephropthy	nd	nd	nd	HR	1.18 (0.92, 1.52)	0.2	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		

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							Lupus nephritis	nd	nd	nd	HR	1.81 (1.13, 2.90)	0.013	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							Membranous nephropathy	nd	nd	nd	HR	0.91 (0.61, 1.36)	0.7	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							MPGN type II	nd	nd	nd	HR	1.03 (0.65, 1.62)	0.9	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							MPGN type II	nd	nd	nd	HR	4.68 (2.03, 10.81)	0.0003	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							GPA	nd	nd	nd	HR	0.78 (0.47, 1.29)	0.3	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							Preemptive transplantation	nd	nd	nd	HR	0.72 (0.49, 1.06)	0.1	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							<1 year on dialysis	nd	nd	nd	HR	0.68 (0.51, 0.90)	0.01	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		
							1-3 years on dialysis	nd	nd	nd	HR	reference	reference	nd		
							>3 years on dialysis	nd	nd	nd	HR	1.57 (1.29, 1.92)	<0.0001	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, HLA mismatch, cold ischemic time, and graft failure.		

**PREDICTORS OF GRAFT LOSS**

24370342	Tancredi	2014	Graft loss, 1 year	nd	0.05	0.95	Serum albumin < 2.5 (vs. >=3.5)	<2.5 g/dl	5.1	72	HR	1.71 (1.09, 2.70)	nd	recipient age, sex, ethnicity, cause of CKD, OPTN region where transplant occurred, year of transplant, need for pretransplantation dialysis, time on the deceased donor wait list, donor source (deceased or living), donor age and cause of death, HLA mismatch level, and cold ischemia time.	A	
12110738	Briganti	2002	graft loss due to GN recurrence	nd	3.5%	96.5%	Serum albumin 2.5-3.4 (vs. >=3.5)	2.5-3.4 g/dl	22.9		HR	1.36 (1.04, 1.78)	nd	Hazard ratios for factors that remained independently predictive in multivariable analysis were adjusted for all other independently predictive factors	A	
							FSGS vs. mean risk for all categories of GN	nd			HR	2.03 (1.19, 3.44)	0.009			
							membranous GN vs. mean risk for all categories of GN	nd			HR	nd	ns			
							IgA nephropathy vs. mean risk for all categories of GN	nd			HR	nd	ns			
							Pauci-immune crescentic glomerulonephritis vs. mean risk for all categories of GN	nd			HR	nd	ns			
							other types of GN	nd			HR	0.30 (0.13, 0.66)	0.003			
							Age	10-year change	nd	nd	HR	nd	ns			
							peak PRA	per 10% increment	nd	nd	HR	1.10 (1.00, 1.21)	0.05			
							Dialysis duration	per 1-year increment	nd	nd	HR	nd	ns			
23295317	Cannon	2012	Graft loss (not death-censored)	patients who either died or experienced graft failure were considered to have failed	6% 1 year, 26% 5 years	93% 1 year, 74% 5 years	Class I Obesity	BMI 30-35	20	70.3	HR	1.00 (0.95, 1.05)	0.901	recipient age, race, gender, CVA as donor cause of death, donor type, peak PRA, cold ischemic time, HLA mismatch, other causes of renal failure, previous kidney transplant	A	
							Class II Obesity	BMI 30-35	7.7		HR	1.15 (1.07, 1.24)	<0.001			
							Class III Obesity	BMI >=40	2.1		HR	1.26 (1.11, 1.43)	<0.001			
							Diabetic nephropathy	nd	21	79	HR	1.34 (1.27, 1.42)	<0.001			
							Hypertensive nephropathy	nd	16	84	HR	1.09 (1.04, 1.15)	0.001			
	Clayton	2011	graft loss due to IgAN recurrence	nd	3.6%	96.4%	Age	10-year change	nd	nd	SHR	0.87 (0.67, 1.13)	0.31	age, sex, HLA mismatch, dialysis duration, transplant era, steroid use	A	
21797974							Dialysis duration 6 months to <1 year (vs. <6months)	nd	46.4	53.6	SHR	0.73 (0.35, 1.49)	nd			
							Dialysis duration 1 y to <5 years (vs. <6months)	nd	71.3	28.7	SHR	0.50 (0.25, 0.98)	nd			
							Dialysis duration >=5 years (vs. <6months)	nd	40.8	59.2	SHR	0.40 (0.09, 1.74)	nd			
							Era 1998-2007 (vs. 1988-1992)	nd	63%	37%	SHR	0.26 (0.10, 0.66)	nd			
22124283	Foster	2011	death-censored graft loss	nd	35.1	64.9	Age 0-4y vs. 25-29y	age at time of graft loss, not time of transplant	nd	nd	HR	0.94 (0.79, 1.13)	0.5	age, sex, SES, primary disease, race, donor age, living donor, duration of dialysis, HLA mismatch, era of transplant	A	
							Age 5-9y vs. 25-29y	age at time of graft loss, not time of transplant	nd	nd	HR	0.60 (0.53, 0.68)	<0.0001			
							Age 10-12y vs. 25-29y	age at time of graft loss, not time of transplant	nd	nd	HR	0.56 (0.49, 0.64)	<0.0001			
							Age 13-16y vs. 25-29y	age at time of graft loss, not time of transplant	nd	nd	HR	0.91 (0.84, 0.98)	0.01			

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							Age 17-20y vs. 25-29y	age at time of graft loss, not time of transplant	nd	nd	HR	1.20 (1.13, 1.27)	<0.0001			
							Age 21-24y vs. 25-29y	age at time of graft loss, not time of transplant	nd	nd	HR	1.20 (1.13, 1.26)	<0.0001			
							Age 30-34y vs. 25-29y	age at time of graft loss, not time of transplant	nd	nd	HR	0.83 (0.80, 0.87)	<0.0001			
							Age 35-39y vs. 25-29y	age at time of graft loss, not time of transplant	nd	nd	HR	0.73 (0.70, 0.76)	<0.0001			
							Age >=40y vs. 25-29y	age at time of graft loss, not time of transplant	nd	nd	HR	0.65 (0.62, 0.68)	<0.0001			
							SES low-mid quartile vs. lowest quartile	nd	18.9	18.8	HR	0.95 (0.91, 0.98)	0.003			
							SES High-mid quartile vs. lowest quartile	nd	26.3	18.8	HR	0.91 (0.88, 0.94)	<0.0001			
							SES Highest quartile vs. lowest quartile	nd	36	18.8	HR	0.83 (0.80, 0.86)	<0.0001			
							GN vs. CAKUT	congenital anomalies of the kidneys or urinary tract	28.3	8.2	HR	1.03 (0.98, 1.09)	0.2			
							FSGS vs. CAKUT	congenital anomalies of the kidneys or urinary tract	8.3	8.2	HR	1.13 (1.07, 1.20)	<0.0001			
							DM vs. CAKUT	congenital anomalies of the kidneys or urinary tract	nd	8.2	HR	1.02 (0.96, 1.07)	0.6			
							Other primary disease vs. CAKUT	congenital anomalies of the kidneys or urinary tract	32.3	8.2	HR	1.01 (0.96, 1.07)	0.6			
							Unknown primary disease vs. CAKUT	congenital anomalies of the kidneys or urinary tract	22.9	8.2	HR	0.85 (0.80, 0.90)	<0.0001			
							Dialysis duration	per 1-year increment	nd	nd	HR	1.02 (1.01, 1.02)	<0.0001			
23406350	Heaphy	2013	graft loss	nd	71.8	28.2	PKD vs. no PKD	nd	9.6	90.4	HR	0.75 (0.72, 0.78)	<0.0001	recipient age, race, gender, PKD status, diabetes status, serum PRA percent, income, primary insurance, obesity status and interactions between ECD status and recipient characteristics	A	
							PRA 1-30% vs. 0%	nd	15.9	73.1	HR	1.04 (1.00, 1.07)	0.0245			
							PRA 31-80% vs. 0%	nd	5.3	73.1	HR	1.14 (1.09, 1.21)	<0.0001			
							PRA >=81% vs. 0%	nd	2.4	73.1	HR	1.21 (1.12, 1.31)	<0.0001			
							BMI >30 vs. <= 30	nd	26.1	73.9	HR	1.13 (1.10, 1.16)	<0.0001			
							High school education/GED vs. none/grade school	nd	38.5	6.4	HR	1.09 (1.04, 1.14)	0.0002			
							Some college vs. bachelor degree vs. none/grade school	nd	29.2	6.4	HR	0.96 (0.92, 1.01)	0.1044			
							Graduate degree vs. none/grade school	nd	4.3	6.4	HR	0.95 (0.89, 1.02)	0.1282			
20814353	Huang	2010	Graft failure	Death-censored graft loss	2 year 60-69yo 7%, 70-79yo 8%, >=80yo 9%	2 year 60-69yo 33%, 70-79yo 92%, >=80yo 91%	Age >= 80 (vs. 60-69)	nd	0.6	79.8	HR	0.89 (0.57, 1.39)	nd	transplant year, recipient age, recipient gender, recipient race, dialysis duration, retransplantation, peak PRA, recipient comorbidities (diabetes, cardiovascular disease, peripheral vascular disease, and cerebrovascular disease), donor type, donor age, degree of human leukocyte antigen mismatch, induction therapy, tacrolimus use, mycophenolate use, and steroid use	A	
							Age 70-79 (vs. 60-69)	nd	19.6		HR	1.02 (0.93, 1.11)	nd			
26660200	Ilori	2015	Graft loss	nd	14.1%	85.9%	Age	10-year change, all pts >= 60 yo	nd	nd	HR	0.94 (0.89, 1.00)	nd	race and ethnicity, any acute rejection, end-stage renal disease (ESRD) etiology, sex, human leukocyte antigen (HLA) mismatch, pretransplantation dialysis, type of donor, donor age, cold ischemia time, insurance, neighborhood poverty, and period of transplantation	A	
24009216	Kainz	2013	Graft loss	the need for retransplantation or permanent return to dialysis	N=119	nd	right atrial diameter (mm)	continuous variable by echo in mm	na	na	HR	1.04 (1.02, 1.07)	0.001	right atrial diameter, cerebrovascular disease, peripheral vascular disease, coronary heart disease, HbG, age at Txp, donor age, immunosuppression, calcineurin inhibitor use, afib, year of Txp	A	
							cerebrovascular disease (yes versus no)	nd, yes vs no	5	95	HR	2.52 (0.61, 10.36)	0.16			
							peripheral vascular disease (yes versus no)	nd, yes vs no	13	87	HR	2.29 (0.57, 9.41)	0.06			
							coronary heart disease (yes versus no)	nd, yes vs no	15	85	HR	0.60 (0.18, 1.99)	0.34			
27336396	Kang	2016	Graft failure	nd	8-years post-Txp: w/ pre-Txp skin cancer 47.6%, w/o pre-Txp skin cancer 41.4%	8-years post-Txp: w/ pre-Txp skin cancer 52.4%, w/o pre-Txp skin cancer 58.6%	Pre-Txp skin cancer excluding those with solid cancer (vs. no pre-Txp skin cancer)	nd	1.6	98.4	HR	1.14 (1.02, 1.27)	0.03 (from log-rank test)	adjusted for sex, age, BMI, ethnicity, EBV, HBV, HCV serostatus, dialysis duration, use of induction therapy	A	
20801565	Kasike	2010	Graft loss	5-year post-Txp, return to maintenance dialysis therapy, preemptive retransplantation, or death with a functioning graft	nd	nd	Primary cause of CKD: HTN vs. DM	nd	22.4	24.9	HR	0.84 (0.79, 0.89)	<0.001	donor age, race, RRT, recipient age, HCV, donor history of HTN, primary insurance, trauma as donor cause of death, HLA	B	
							Primary cause of CKD: GN vs. DM	nd	25.2		HR	0.77 (0.73, 0.82)	<0.001			
							Primary cause of CKD: Cystic disease vs. DM	nd	8.8		HR	0.59 (0.54, 0.65)	<0.001			

Pubmed id	Authors	Year	Outcome	Outcome definition	% w/ outcome	% w/o outcome	Primary Predictor	Predictor definition	% w/ predictor	% w/o predictor	Metric	Estimate, mean (95% CI)	P value	Adjustment, Other covariates (list once)	Methodological quality	Notes		
21449945, 27391198, 22156753	Molnar	2011, 2015, 20	Graft loss	Death censored graft failure	11.4	88.6	PD vs. HD	nd	14.0	86.0	HR	1.08 (0.79, 1.47)	0.63	age, sex, recipient race/ethnicity, diabetes mellitus, dialysis vintage, primary insurance, marital status, standardized mortality ratio of the dialysis clinic during entry quarter, and eight comorbidities (atherosclerotic heart disease, congestive heart failure, cancer, chronic obstructive pulmonary disease, cerebrovascular disease, hypertension, peripheral vascular disease, and tobacco use), body mass index (BMI) and nine laboratory variables: serum or blood concentrations of total iron binding capacity, ferritin, phosphorus, calcium, bicarbonate, peripheral white blood cell count (WBC), lymphocyte percentage, albumin, and hemoglobin, donor type, donor age, donor sex, panel reactive antibody (PRA) titer (last value before transplant), number of HLA mismatches, and cold ischemia time	A			
				Graft loss, death censored	graft failure	8.8	91.2	albumin	by 0.2 g/dl, as continuous	nd	nd	HR	0.96 (0.90, 1.02)				0.15	
				Graft loss	death censored allograft loss	10.9% in the entire cohort of 15125 pt	89.1	Age 18-34 (vs. 50-64)	nd	nd	nd	HR	1.64 (1.37, 1.96)				<0.001	recipients' race, type of insurance, time on dialysis, Hgb, donor's DM, HLA mismatch
								Age 35-49 (vs. 50-64)	nd	nd	nd	HR	1.25 (1.07, 1.45)				0.004	
								Age>=65 (vs. 50-64)	nd	nd	nd	HR	0.82 (0.67, 1.01)				0.06	
								Primary cause of ESRD: HTN vs. DM	nd	23.0	25.0	HR	1.51 (1.21, 1.89)				<0.001	
								Primary cause of ESRD: GN vs. DM	nd	23.0		HR	1.58 (1.25, 2.00)				<0.001	
				Primary cause of ESRD: Cystic disease vs. DM	nd	8.0		HR	1.14 (0.83, 1.58)	0.42								
				DM (presence vs. absence)	nd	37.0	63.0	HR	1.35 (1.14, 1.61)	<0.001								
19353768	Mulay	2009	graft loss due to GN recurrence	nd	2.6%	97.4%	FSGS vs. "other"	type of GN	20.6	nd	HR	1.53 (1.16, 2.03)	<0.001	donor and recipient age; donor and recipient gender; donor and recipient race; duration of dialysis prior to transplant; peak panel reactive antibody; donor type (living or deceased); donor cause of death if deceased donor; cold ischemia time; HLA antigen match; delayed graft function; acute rejection; hepatitis B surface antigen status; employment status; recipient body mass index and transplant year	A	**"other" includes IgM nephropathy; rapidly progressive glomerulonephritis; Goodpasture's syndrome; Henoch-Schonlein purpura; scleroderma; hemolytic uremic syndrome; polyarteritis; Wegener's granulomatosis; vasculitis; other proliferative glomerulonephritis; postinfectious and subacute bacterial endocarditis-induced glomerulonephritis.		
							IgA nephropathy vs. "other"	type of GN	6.6	nd	HR	1.02 (0.65, 1.58)	0.95				**"other" includes IgM nephropathy; rapidly progressive glomerulonephritis; Goodpasture's syndrome; Henoch-Schonlein purpura; scleroderma; hemolytic uremic syndrome; polyarteritis; Wegener's granulomatosis; vasculitis; other proliferative glomerulonephritis; postinfectious and subacute bacterial endocarditis-induced glomerulonephritis.	
							Membranous GN vs. "other"	type of GN	3.9	nd	HR	1.75 (1.15, 2.67)	0.01				**"other" includes IgM nephropathy; rapidly progressive glomerulonephritis; Goodpasture's syndrome; Henoch-Schonlein purpura; scleroderma; hemolytic uremic syndrome; polyarteritis; Wegener's granulomatosis; vasculitis; other proliferative glomerulonephritis; postinfectious and subacute bacterial endocarditis-induced glomerulonephritis.	
							MPGN vs. "other"	type of GN	3.9	nd	HR	2.57 (1.84, 3.58)	<0.001				**"other" includes IgM nephropathy; rapidly progressive glomerulonephritis; Goodpasture's syndrome; Henoch-Schonlein purpura; scleroderma; hemolytic uremic syndrome; polyarteritis; Wegener's granulomatosis; vasculitis; other proliferative glomerulonephritis; postinfectious and subacute bacterial endocarditis-induced glomerulonephritis.	
							Lupus nephritis vs. "other"	type of GN	10.6	nd	HR	0.72 (0.49, 1.06)	0.1				**"other" includes IgM nephropathy; rapidly progressive glomerulonephritis; Goodpasture's syndrome; Henoch-Schonlein purpura; scleroderma; hemolytic uremic syndrome; polyarteritis; Wegener's granulomatosis; vasculitis; other proliferative glomerulonephritis; postinfectious and subacute bacterial endocarditis-induced glomerulonephritis.	
							unspecified pathology vs. "other"	type of GN	42.2	nd	HR	0.59 (0.44, 0.78)	<0.001				**"other" includes IgM nephropathy; rapidly progressive glomerulonephritis; Goodpasture's syndrome; Henoch-Schonlein purpura; scleroderma; hemolytic uremic syndrome; polyarteritis; Wegener's granulomatosis; vasculitis; other proliferative glomerulonephritis; postinfectious and subacute bacterial endocarditis-induced glomerulonephritis.	
							Age	10-year change	nd	nd	HR	0.86 (0.80, 0.91)	<0.001					
							Dialysis duration 1-12 months vs. 0 months	nd	nd	nd	HR	2.08 (1.46, 2.96)	<0.001					
							Dialysis duration 12-36 months vs. 0 months	nd	37.3	nd	HR	1.71 (1.18, 2.48)	<0.001					
							Dialysis duration >36 months vs. 0 months	nd	26.2	nd	HR	1.26 (0.83, 1.93)	0.28					
			BMI	continuous?	nd	nd	HR	0.98 (0.96, 1.00)	0.02									
			peak PRA >50% vs. <50%	nd	7.8	92.2	HR	1.24 (0.87, 1.78)	0.24									
			Era 2001-2003 vs. 1990-1994	nd	nd	nd	HR	0.39 (0.24, 0.64)	<0.001									
26569067	Naik	2016	Graft loss	center-reported return to dialysis or retransplantation	nd	nd	Underweight	BMI<18.5	2.4	32.8	HR	0.96 (0.88, 1.05)	0.41	Recipient and donor age, race, sex, dialysis time, cold ischemia time, HLA mismatch levels, PRA, era of transplantation, donor BMI, type of kidney (living, SCD, ECD), delayed graft function, induction therapy and immunosuppression at discharge	A			
				Overweight			BMI 25-30	33.5	HR	1.05 (1.01, 1.08)	0.01							
				Class I obesity			BMI 30-35	20.3	HR	1.15 (1.10, 1.19)	<0.001							
				Class II obesity			BMI 30-35	7.7	HR	1.21 (1.15, 1.28)	<0.001							
				Class III obesity			BMI >=40	3.4	HR	1.13 (1.04, 1.22)	0.002							

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26102616	Opelz	2016	Graft loss	nd	10521 events	nd	Stopped smoking (vs. Never smoking)	nd	22.1	67.6	HR	1.1 (1.0, 1.1)	<0.001	year of transplant, recipient age, sex and race, donor type (deceased or living), donor age, original disease leading to end-stage renal failure, time on dialysis, pretransplant panel reactive antibodies, HLA-A + B + DR mismatches, increased cardiovascular risk (yes/no as identified by investigator), pretransplant cancer, type of immunosuppressive therapy (calcineurin inhibitors, antemetabolites, steroids, mechanistic target of rapamycin inhibitor, antibody induction therapy) and smoking status (never smoked, history of smoking but patient stopped before receiving a transplant, ongoing smoking at time of transplant).	C	
							Continued smoking (vs. Never smoking)	nd	10.3	HR	1.5 (1.4, 1.6)	<0.001				
							Stopped smoking (vs. Never smoking)	nd	22.1	HR	1.0 (1.0, 1.1)	0.19				
							Continued smoking (vs. Never smoking)	nd	10.3	HR	1.4 (1.3, 1.5)	<0.001				
24070588	Pieloch	2014	Graft loss	3 year graft loss	nd	nd	Morbid obesity	BMI 35-40 kg/m2	20	80	HR	1.04 (0.98, 1.11)	0.209	age, gender, race, functional status, DM, PVD, dialysis dependency, HLA matching, cold ischemia time, donor type	A	
25758804	Pieloch	2015	Graft failure	a permanent return to dialysis or death with functioning graft	KTM score=0 8.8%, 1 11.8%, 2 14.6%, 3 18.3%, 4 22.2%, 5 26.0%, 6 30.2%, >= 7 31.3%	KTM score=0 91.2%, 1 88.2%, 2 85.4%, 3 81.7%, 4 77.8%, 5 74.0%, 6 69.8%, >= 7 68.7%	KTM score 2 (vs score 0)	Kidney Transplant Morbidity Index score= 1	22.2	6.4	HR	1.30 (1.16, 1.45)	<0.001	human leukocyte antigen mismatch, cold ischemic time, donor age, and donor type	C	
							KTM score 3 (vs score 0)	Kidney Transplant Morbidity Index score= 2	27.6	HR	1.44 (1.29, 1.60)	<0.001				
							KTM score 4 (vs score 0)	Kidney Transplant Morbidity Index score= 3	22.8	HR	1.74 (1.56, 1.94)	<0.001				
							KTM score 5 (vs score 0)	Kidney Transplant Morbidity Index score= 4	13.3	HR	2.08 (1.87, 2.33)	<0.001				
							KTM score 6 (vs score 0)	Kidney Transplant Morbidity Index score= 5	5.5	HR	2.46 (2.19, 2.77)	<0.001				
							KTM score 7 (vs score 0)	Kidney Transplant Morbidity Index score= 6	1.7	HR	2.97 (2.58, 3.41)	<0.001				
							KTM score >= 7 (vs score 0)	Kidney Transplant Morbidity Index score= 7	0.5	HR	3.11 (2.55, 3.80)	<0.001				
21566110	Reddy	2011	Graft loss in living donor Txp	Death-censored graft failure	5 years all recipients, HBV+ 74.9%, HBV- 75.1%	5 years all recipients, HBV+ 74.9%, HBV- 75.1%	HBV infection (vs. HBV-)	HBsAg +ve	all recipients 1.8%	all recipients 98.2%	HR	0.74 (0.45, 1.24)	nd	recipient age, gender, body mass index, race, comorbid (diabetes, hypertension, cerebrovascular disease), dialysis duration, donor HbCAb, expanded criteria donor, HLA DR mismatch, cold ischemia time (in deceased donor), induction therapy, and immunosuppressants at discharge	A	
			Graft loss in deceased donor Txp	Death-censored graft failure	5 years all recipients, HBV+ 74.9%, HBV- 75.1%	5 years all recipients, HBV+ 74.9%, HBV- 75.1%	HBV infection (vs. HBV-)	HBsAg +ve	all recipients 1.8%	all recipients 98.2%	HR	1.06 (0.85, 1.33)	nd			
21415312	Streja	2011	Graft failure	death-censored graft failure	7.1%	92.9%	BMI	as continuous variable, based on each 1 kg/m2 higher BMI	na	na	HR	1.01 (0.99, 1.03)	0.34		C	
							Creatinine	as continuous variable, based on each 1 mg/dl higher scr	na	na	HR	0.96 (0.81, 1.00)	0.061			
25135680	Wightman	2014	Graft failure	death-censored graft failure	5.7%	94.3%	Definite intellectual disability	Identified as "definitely cognitive delay/impairment" by their center	5.6	84.1	HR	1.1 (0.5, 2.5)	0.698	age in years (<5, 5-12, 13-18), male gender, race (white/nonwhite), etiology (structural, FSGS, GN, other), deceased donor (Y/N), cold ischemia time >24 hrs (Y/N), HLA match, PRA/CPRA (<10%, 10-80%, 80-100%)	C	
							Probable intellectual disability	"probable" or "questionable" cognitive delay/impairment, "reduced academic load/nonparticipation," or "delayed grade level/special education"	10.3	HR	0.5 (0.3, 2.0)	0.698				
25098499	Xia	2014	Death-censored graft survival	the earliest of re-transplantation or return to dialysis	13.3	86.7	HIV seropositive (vs. negative)	nd	50.0	50.0	HR	0.85 (0.48, 1.51)	nd	HIV/HCV coinfection, age, race, sex, etiology of ESRD, BMI, PRA, prior KTxp, insurance, dialysis duration, Txp year, comorbidity, HLA mismatch, cold ischemia time	A	
26636735	Barracough	2016	Graft failure	nd	7177	327	≥Obese	nd	nd	nd	HR	1.14 (0.94, 1.38)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term	A	
							Overweight	nd	nd	nd	HR	1.05 (0.89, 1.23)	nd			
							Smoker	nd	nd	nd	HR	1.30 (1.13, 1.49)	nd			
							CVD	cerebrovascular disease	nd	nd	HR	0.92 (0.65, 1.30)	nd			



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							DM	nd	nd	nd	HR	1.27 (1.02, 1.58)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term		
							Age 45-64	nd	nd	nd	HR	1.03 (0.92, 1.15)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term		
							Age ≥65	nd	nd	nd	HR	1.17 (0.97, 1.40)	nd	age, comorbidities, BMI, smoking status, transplant era, graft number, HLA mismatches, PRA, ischemic time, donor source, and transplant state, and included a race by rural interaction term		
28361229	Ladhani	2017	Graft loss	nd	31.3	68.7	Obese	nd	8.1	91.9	HR	1.61 (1.05, 2.47)	nd	adjusted for age at transplant, racial origin, primary renal disease, HLA mismatch, and year of transplant	A	
							Overweight	nd	17.2	82.8	HR	1.03 (0.71, 1.49)	nd	adjusted for age at transplant, racial origin, primary renal disease, HLA mismatch, and year of transplant		
							Underweight	nd	64.4	35.6	HR	1.05 (0.70, 1.60)	nd	adjusted for age at transplant, racial origin, primary renal disease, HLA mismatch, and year of transplant		
26924061	Pruthi	2016	Graft failure	return to dialysis or preemptive retransplantation	nd	nd	ADPKD	nd	nd	nd	HR	reference	reference	nd	A	
							Crescentic GN	nd	nd	nd	HR	1.53 (0.90, 2.61)	0.12	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHBST) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							FGSG	nd	nd	nd	HR	2.39 (1.78, 3.22)	<0.0001	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHBST) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							GN histologically not examined	nd	nd	nd	HR	0.93 (0.61, 1.41)	0.7	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHBST) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							GN histologically proven	nd	nd	nd	HR	1.68 (1.31, 2.17)	<0.0001	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHBST) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							IgA nephropathy	nd	nd	nd	HR	1.59 (1.27, 1.99)	<0.0001	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHBST) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							Lupus nephritis	nd	nd	nd	HR	1.64 (1.13, 2.40)	0.01	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHBST) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		

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							Membranous nephropathy	nd	nd	nd	HR	1.99 (1.38, 2.86)	0.0002	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHSBT) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							MPGN type II	nd	nd	nd	HR	2.33 (1.63, 3.33)	<0.0001	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHSBT) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							MPGN type II	nd	nd	nd	HR	3.50 (1.87, 6.55)	<0.0001	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHSBT) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							GPA	nd	nd	nd	HR	1.16 (0.68, 1.98)	0.6	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHSBT) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							Preemptive transplantation	nd	nd	nd	HR	0.72 (0.53, 0.97)	0.03	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHSBT) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							<1 year on dialysis	nd	nd	nd	HR	1.02 (0.82, 1.26)	0.9	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHSBT) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		
							1-3 years on dialysis	nd	nd	nd	HR	reference	reference	nd		
							>3 years on dialysis	nd	nd	nd	HR	1.41 (1.17, 1.70)	0.0003	adjusted for age, gender, type of transplant, ethnicity, donor age, time on dialysis pretransplantation, year of transplantation, HLA mismatch, and cold ischemic time. HLA mismatch was categorized (as per NHSBT) into 4 groups ranging from low to high levels of mismatch: group 1, 0 mismatch; group 2, 0DR and 0/1B mismatches; group 3, 0DR and 2B or 1DR and 0/1B mismatches; and group 4, 1DR and 2B or 2DR mismatches		

**PREDICTORS OF OTHER OUTCOMES**

27336396	Kang	2016	Post-transplant malignancy overall	nd	5-years post-Txp: w/ pre-Txp cancer 31.6%, w/o pre-Txp cancer 7.4%	5-years post-Txp: w/ pre-Txp cancer 68.4%, w/o pre-Txp cancer 92.6%	Pre-Txp skin cancer (vs. no pre-Txp cancer)	nd	1671 pt	nd	HR	2.60 (2.27, 2.98)	<0.001	adjusted for sex, age, ethnicity, hypertension, BMI, induction therapy, tacrolimus use at discharge, HLA DR, diabetes, serostatus of CMV, EBV, HBV, HCV, and serum creatinine	A	
							Pre-Txp NMSC alone (vs. no pre-Txp cancer)	nd	1024 pt	nd	HR	2.89 (2.47, 3.40)	<0.001			
							Pre-Txp melanoma skin cancer alone (vs. no pre-Txp cancer)	nd	398 pt	nd	HR	1.77 (1.30, 2.40)	<0.001			
17198258	Shah	2006	New-onset diabetes mellitus	nd	12 mo 8.9%, 24 mo 14.8%	12 mo 91.1%, 24 mo 85.2%	Age	by 10 years, as continuous variable	na	na	HR	1.29 (1.24, 1.34)	<0.001	sex, race, donor- ECD vs SCD, living vs deceased, HLA mismatching, immunosuppressive Rx	A	
							HTN (yes vs. no)	na	77.6	22.4	HR	1.26 (1.11, 1.44)	<0.001			
							BMI 25-30 (vs. <25)	na	32.4	42.5	HR	1.39 (1.24, 1.57)	<0.001			
							BMI >30 (vs. <25)	na	23.1		HR	1.84 (1.63, 2.08)	<0.001			
							HCV antibody (+ vs. -)	na	4.2	79.6	HR	1.42 (1.15, 1.74)	0.001			

**PRE-EMPTIVE vs. EARLY DIALYSIS**

Pubmed id	Authors	Year	Outcome	Outcome definition	% w/ outcome	% w/o outcome	Primary Predictor	Predictor definition	% w/ predictor	% w/o predictor	Metric	Estimate, mean (95% CI)	P value	Adjustment, Other covariates (list once)	Methodological quality	Notes
27653837	Amaral	2016	Graft failure	death-censored graft failure, in which death with graft function was treated as graft failure, and mortality	5 years 14.6% for preemptive, 23.6% for non-preemptive	5 years 85.4% for preemptive, 76.4% for non-preemptive	Pre-emptive KTxp (yes vs no)	a transplant with no history of dialysis	13.6	86.4	HR	1.32 (1.10, 1.56)	nd	sex, race/ethnicity, age at time of transplantation, etiology of end-stage renal disease, panel reactive antibody, insurance status at the time of transplantation, neighborhood poverty, donor type (in combined donor type models), and cold ischemia time (in deceased donor recipient models).	A	
			Mortality	all cause mortality	4.4%	95.6%	Pre-emptive KTxp (yes vs no)	a transplant with no history of dialysis	13.6	86.4	HR	1.69 (1.22, 2.33)	nd			
23371953	Grams	2013	death		nd	nd	early dialysis vs. preemptive	early: <=1 year	nd	nd	HR	1.06 (0.99, 1.14)	0.06	propensity matched on UNOS region, recipient and donor age, recipient sex, ethnicity, impaired functional status, PRA, Hep C status, previous non-kidney transplant, insurance type, etiology of renal disease, transplant year, ECD, DCD, zero-antigen mismatch, cold ischemia time, wait time	B	*baselines estimated from table
			death-censored graft loss	nd	nd	nd	early dialysis vs. preemptive	early: <=1 year	nd	nd	HR	1.21 (1.12, 1.30)	<0.001			

Pubmed id	Authors	Year	Population: Non-biased selection of study participants without inappropriate restrictions or selection. All eligible participants included or a random selection of these. No biased or large loss to follow-up.	Predictors/Variables: All predictors or study variables are well-defined and appropriately measured.	Outcome: Clearly longitudinal (incident outcome) [only if relevant]. Outcome blindly adjudicated or equivalent. Measured completely and the same for all participants.	Confounders: Important potential confounding factors appropriately accounted for.
24370342	Tancredi	2014	low	unclear	unclear	low
23295317	Cannon	2012	low	low	low	low
26569067	Naik	2016	low	low	low	low
26102616	Opelz	2016	unclear	high	high	low
24009216	Kainz	2013	low	high	low	low
26660200	Ilori	2015	low	low	low	low
26147285	Krishnan	2015	high (excluded all pts w/o BMI data)	low	low	low
27653837	Amaral	2016	low	low	low	low
25758804	Pieloch	2015	low	low	low	high (some important confounders not adjusted, also not reported as baseline)
21415312	Streja	2011	low	low	low	low
25135680	Wightman	2014	low	low	low	low
24138318	Farrugia	2014	low	low	low	low
27336396	Kang	2016	low	unclear	unclear	low
20814353	Huang	2010	low	low	unclear	low
26720436	Lynch	2016	high	unclear	low	low
20801565	Kasiske	2010	high	unclear	low	low
24070588	Pieloch	2014	low	low	unclear	low
21566110	Reddy	2011	low/unclear	low	unclear	low
21449945	Molnar	2011	low	low	unclear	low
17198258	Shah	2006	low	unclear	unclear	low
25098499	Xia	2014	low	unclear	low	low
21797974	Clayton	2011	low	low	low	low
19353768	Mulay	2009	low	low	low	low
12110738	Briganti	2002	low	low	low	low
3406350	Heaphy	2013	low	low	low	low
23371953	Grams	2013	low	low	low	low
22124283	Foster	2011	low	low	low	low
26636735	Barracough	2016	low	low	low	low
28010785	Lim	2017	low	low	low	low
28361229	Ladhani	2017	low	low	low	low
26924061	Pruthi	2016	low	low	unclear	low

Pubmed id	Authors	Year	Model: Multivariable. All included variables reported. Appropriate model and methods for variable selection used. Reported results interpretable.	OVERALL: high if Population, Outcome, Model biased/bad; maybe high if predictors and confounders alone are high
24370342	Tancredi	2014	low	low
23295317	Cannon	2012	low	low
26569067	Naik	2016	low	low
26102616	Opelz	2016	low	high
24009216	Kainz	2013	low	low
26660200	Ilori	2015	low	low
26147285	Krishnan	2015	high (for using uncertain primary diagnosis)	high
27653837	Amaral	2016	low	low
25758804	Pieloch	2015	high (some important confounders not adjusted, not gave reasons)	high
21415312	Streja	2011	low/high (about 50% pt were excluded in the multivariate model)	high
25135680	Wightman	2014	low/high (about 50% pt were excluded in the multivariate model)	high
24138318	Farrugia	2014	low	low
27336396	Kang	2016	low	low/unclear
20814353	Huang	2010	low	low
26720436	Lynch	2016	low	high
20801565	Kasiske	2010	low	high/unclear
24070588	Pieloch	2014	low	low
21566110	Reddy	2011	low	low
21449945	Molnar	2011	low	low
17198258	Shah	2006	low	low
25098499	Xia	2014	low	low
21797974	Clayton	2011	low	low
19353768	Mulay	2009	low	low
12110738	Briganti	2002	low	low
3406350	Heaphy	2013	low	low
23371953	Grams	2013	unclear	low
22124283	Foster	2011	low	low
26636735	Barraclough	2016	low	low
28010785	Lim	2017	low	low
28361229	Ladhani	2017	low	low
26924061	Pruthi	2016	low	low

### Evidence Profile: Pre-Transplant Predictors of Post-Transplant Mortality (from Registry Studies)\*

Predictor	Registries (No. Studies)	Percent w/Predictor	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence	Other Considerations	Summary of Findings		
							Quality of Evidence	Description of Findings	Outcome Importance
<b>Elderly (age ≥60 yo)</b>	ANZDATA, OPTN/UNOS, SRTR†, HES/ONS, USRDS (5)	≥60 100%; ≥70 yo: 20% in 1 study; ≥65 yo: 9% in 1 study	Serious limitations (-1)‡	No important inconsistencies (0)	Direct (0)	None	Moderate	Among elderly, higher risk with increased age (categorical: HR= 1.42-15.7, HR increased as the age increased; continuous: HR= 1.47)	Critical
<b>Other ages</b>	ANZDATA, OPTN/UNOS, SRTR†, HES/ONS (4)	Age 45-64 yo: 53% in 1 study	No limitations (0)	No important inconsistencies (0)	Direct (0)	None	High	Among patients younger than 60, older age associated with higher risk (categorical: HR= 1.67-3.22, continuous: HR= 1.04)	
<b>BMI/Obesity</b>	ANZDATA, SRTR†, UNOS RR/NHSBT (6)	BMI>35: 3.3-20%	Very serious limitations (-2)§	No important inconsistencies (0)	Direct (0)	None	Low	Neither high BMI (HR= 0.48-1.96) nor low BMI (HR= 1.96) is significant associated with poor survival outcome, except for BMI 30-35 vs. <30 Sig (HR= 0.92) associated with better outcome in one study	
<b>DM</b>	ANZDATA, SRTR†, HES/ONS, USRDS (4)	15.2- 37.0%	Serious limitations (-1)‡	No important inconsistencies (0)	Direct (0)	None	Moderate	DM consistently associated with higher risk of mortality (HR= 1.39-1.64)	
<b>PVD</b>	SRTR†, HES/ONS, OEDTR, USRDS (4)	0.7-13%	Very serious limitations (-2)#	No important inconsistencies (0)	Indirect (-1)#	None	Very low	PVD consistently associated with higher risk of mortality (HR= 1.15-4.60)	
<b>CVD (including AMI and CAD)</b>	ANZDATA, SRTR†, HES/ONS, USRDS (4)	2.4-7.0%	Serious limitations (-1)‡	No important inconsistencies (0)	Direct (0)	None	Moderate	CVD consistently associated with higher risk of mortality (HR= 1.16-1.52)	
<b>CHF</b>	HES/ONS, USRDS (2)	0.6-12.7%	Very serious limitations (-2)#	Important inconsistencies (-1)	Indirect (-1)#	None	Very low	Unclear: association between CHF and mortality significant in one study (HR= 1.22), NS in one (HR= 1.51); LAD as continuous variable, Sig, HR= 1.06; RLD as continuous variable, NS, HR= 0.95	

Predictor	Registries (No. Studies)	Percent w/Predictor	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence	Other Considerations	Summary of Findings			
							Quality of Evidence	Description of Findings	Outcome Importance	
<b>GN</b>	UKRR/NHS Blood and Transplant (1)	nd	No limitations (0)	N/A	Direct (0)	Sparse (-1)	Moderate	Lupus nephritis (HR = 1.81, p=0.013) and MPGN type II (HR = 4.68, p=0.0003) had a greater reduction in 10-year patient survival than APKD. DM types I and II (HR=2.24, 1.59, p<0.0001, 0.001) and other or not reported kidney disease (HR = 1.28, 1.28, p= 0.007, 0.004) had higher risk of mortality than GN. Polycystic kidney disease had no significant difference (HR=0.81, p=0.56).		
<b>Time on dialysis</b>	UKRR (1)	nd	No limitations (0)	N/A	Direct (0)	Sparse (-1)	Moderate	Over 3 years on dialysis (HR=1.57, p<0.0001) significantly reduced 10-year patients survival, while less than 1 year on dialysis (HR=0.68, p=0.01) significantly improved 10-year patient survival compared to 1-3 years on dialysis.		
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>			
Older age, DM, CVD, GN, and time on dialysis are associated with higher risk of death. Higher BMI and obesity may not be associated with higher risk of death. For other predictors, the evidence was unclear or there was insufficient evidence.*							Moderate Low Very Low			

\* See list of predictors evaluated by a single study each below the footnotes.

† Linked with DaVita.

‡ Biased selection of patient population in one study.

§ Biased selection of patient population in one study. Some important confounders not adjusted and no reasoning described for the selection of co-variables in one study.

Approximately 50% patients were excluded in the multivariate analysis due to the lack of data in one study.

# No specific definition or diagnostic criteria provided for the predictor in one study.

N/A= Not available or not applicable, BMI= Body mass index, DM= Diabetes mellitus, PVD= periphervascular disease, CVD= Cardiovascular disease, AMI= Acute myocardial infarction, CAD= Coronary artery disease, CHF= Chronic heart failure, LAD= Left atrium diameter, RVD= Right ventricle diameter

OPTN = Organ Procurement and Transplantation Network- other names of the database include the Scientific Registry of Transplant Recipients (SRTR) and United Network for Organ Sharing (UNOS), DaVita = Kidney disease and dialysis information, USRDS = The United States Renal Data System, ANZDATA = The Australian and New Zealand Dialysis and Transplantation Registry, OEDTR = Österreichische Gesellschaft für Nephrologie, CTS = Collaborative Transplant Study, HES = Hospital Episode Statistics, ONS = Office for National Statistics, RR = the UK Renal Registry, NHSBT = the National Health Service Blood and Transplant, UKRR = United Kingdom Renal Registry, MPGN = membranoproliferative glomerulonephritis, APKD = adult polycystic kidney disease

**Only one study for each of the following predictors:**

- Albumin, by 0.2 g/dl DaVita/ SRTR HR= 0.87 (0.82, 0.93)
- Albumin, by 1 g/dl DaVita/ SRTR HR= 0.62 (0.52, 0.75)
- Cerebral vascular accident, presence (vs. absence) HES/ONS HR= 1.66 (0.91, 3.03)
- COPD, presence vs. absence USRDS HR= 1.20 (1.02, 1.41)
- Creatinine, by 1 mg/dl SRTR/MHD HR= 0.91 (0.86, 0.95)
- Current smoker, vs. never smoker CTS HR= 1.6 (1.5, 1.8)
- Definite intellectual disability, presence vs. absence UNOS HR= 0.3 (0.2, 12.2)
- Diabetic nephropathy, presence (vs. absence) UNOS HR= 1.61 (1.50, 1.73)
- Dialysis modality, peritoneal dialysis (vs. hemodialysis) DaVita/ SRTR HR= 0.57 (0.38, 0.87)
- Ever smoker, vs. never smoker CTS HR= 1.1 (1.0, 1.2)
- Hepatitis B infection, HBV + (vs. HBV -) OPTN/UNOS HR= 0.98 (0.59, 1.63) (in recipients of living donors), 1.09 (0.88, 1.36) (in recipients of deceased donors)
- HIV infection, HIV + (vs. HIV -) SRTR/OPTN HR= 1.25 (0.61, 2.56)
- Hypertensive nephropathy, presence (vs. absence) UNOS HR= 1.10 (1.02, 1.19)
- Kidney transplant morbidity index score, score 1, score 2, score 3, score 4, score 5, score 6, score 7 (vs. score 0) OPTN/UNOS HR= 1.85 (1.45, 2.36), 3.11 (2.46, 3.94), 5.00 (3.96, 6.31), 7.37 (5.83, 9.32), 9.41 (7.41, 11.94), 12.51 (9.45, 15.63), 13.03 (9.68, 17.54)
- Pre-transplant skin cancer excluding patients with solid cancers, vs. no pre-transplant skin cancer UNOS HR= 1.17 (1.04, 1.32)
- Pre-transplant skin cancer, vs. no pre-transplant skin cancer UNOS HR= 1.20 (1.07, 1.34)
- Probable intellectual disability, presence vs. absence UNOS HR= 0.2 (0.1, 1.3)
- Socioeconomic deprivation, score 2, score 3, score 4 (vs. score 1) HES/ONS HR= 0.84 (0.68, 1.05), 0.86 (0.69, 1.08), 0.86 (0.68, 1.08)



### Evidence Profile: Pre-Transplant Predictors of Graft Loss (from Registry Studies)\*

Predictor (Suboutcome)	Registries (No. Studies)	Percent w/Predictor	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence	Other Considerations	Summary of Findings		
							Quality of Evidence	Description of Findings	Outcome Importance
<b>Elderly</b>	ANZDATA, SRTR†, OPTN/UNOS (4)	≥60 100%; ≥70 20%	No limitations (0)	No important inconsistencies (0)	Direct (0)	Sparse (-2)	Low	Among elderly, no difference by age (categorical), although one continuous analysis found lower risk with increasing age	Critical
<b>Other ages</b>	ANZDATA, SRTR†, USRDS (3)	nd	No limitations (0)	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very low	Unclear: Risk of graft loss varies by age, but pattern is not consistent across studies	
<b>(2ary GN recurrence)</b>	USRDS, ANZDATA (2)	N/A	No limitations (0)	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very low	Unclear: Risk of graft loss due to GN recurrence decreases with higher age in one study (HR=0.86 per decade), but no significant association in another study	
<b>Albumin</b>	SRTR†, OPTN (2)	Low albumin: 28% in one study	No limitations (0)	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very low	Hypoalbuminemia is significantly associated with increased graft loss (HR 1.36-1.71), but one study found NS association when evaluated as a continuous variable.	
<b>BMI/Obesity</b>	ANZDATA, OPTN/UNOS, SRTR†, USRDS (7)	Obesity/overweight: 2-64%	Serious limitations (-1)‡	No important inconsistencies (0)	Direct (0)	None (0)	Moderate	Morbid obesity (BMI ≥40) associated with higher graft loss (HR =1.13-1.26); other evaluations of BMI (including underweight) NS	
<b>DM as cause of ESRD</b>	ANZDATA, SRTR†, USRDS (3)	37% in one study	No limitations (0)	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very Low	Unclear: One study each found significant and NS associations	
<b>GN as cause of ESRD</b>	ANZDATA, OPTN/USRDS, SRTR†, UKRR, USRDS (6)	3.9-28.3%	Serious limitations (-1)§	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very low	Unclear: One study each found lower, no, or higher risk of graft loss	
<b>Membranous GN as cause of ESRD (2ary GN recurrence)</b>	ANZDATA, UKRR, USRDS (3)	20.6% in one study	No limitations (0)	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very Low	Unclear: One study each found significant and NS associations.	

Predictor (Suboutcome)	Registries (No. Studies)	Percent w/Predictor	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence	Other Considerations	Summary of Findings		
							Quality of Evidence	Description of Findings	Outcome Importance
<b>FSGS as cause of ESRD (2ary GN recurrence)</b>	USRDS, ANZDATA (2)	20.6% in one study	No limitations (0)	No important inconsistencies (0)	Direct (0)	Sparse (-2)	Low	Risk of graft loss due to GN recurrence is associated with FSGS as the primary cause of ESRD (HR=1.53, 2.03).	
<b>IgA nephropathy as cause of ESRD (2ary GN recurrence)</b>	UKRR, USRDS, ANZDATA (3)	6.6% in one study	No limitations (0)	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very Low	Unclear: One study each found significant and NS associations.	
<b>HTN as cause of ESRD</b>	OPTN/USRDS, SRTR†, UNOS (3)	16.0-23.0%	Serious limitations (-1)§	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very low	Unclear: One study each found lower, no, or higher risk of graft loss.	
<b>Cystic disease as cause of ESRD</b>	OPTN/USRDS, SRTR† (2)	8.0-8.8%	Serious limitations (-1)§	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very low	Unclear: One study each found significant and NS associations.	
<b>PRA (2ary GN recurrence)</b>	ANZDATA, USRDS, SRTR† (2)	2.4-15.9%	No limitations (0)	Important inconsistencies (-1)	Direct (0)	Sparse (-2)	Very Low	Unclear: One continuous analysis found increased risk of graft loss due to GN recurrence per 10% increment, but another found no significant association as a categorical variable (>50% vs. <50%).	
<b>Dialysis duration (2ary GN recurrence)</b>	ANZDATA, USRDS, UKRR (5)	26.2-71.3%	No limitations (0)	No important inconsistencies (0)	Direct (0)	None (0)	High	Per 1-y increment, NS; 1-12 months vs. 0 months, HR=2.08, sig, 12-36 months vs. 0 months, HR=1.71, sig; >36 months vs. 0 months, HR=1.26, NS; >36 months vs. 12-36 months HR=1.41, sig	
<b>Primary kidney diagnosis</b>	UKRR (1)	nd	Serious limitations (-1)**	N/A	Direct (0)	Sparse (-2)	Very Low	FSGS (HR=2.39, p<0.0001), GN histologically proven (HR=1.68, p<0.0001), IgA nephropathy (HR=1.59, p<0.0001), lupus nephritis (HR = 1.64, p=0.01), membranous nephropathy (HR=1.99, p=0.0002), MPGN type I (HR=2.33, p<0.0001) and MPGN type II (HR = 3.50, p<0.0001) had a greater reduction in 10-year graft loss than APKD.	

Predictor (Suboutcome)	Registries (No. Studies)	Percent w/Predictor	Methodological Quality of Studies	Consistency Across Studies	Directnes s of the Evidence	Other Considerations	Summary of Findings		
							Quality of Evidence	Description of Findings	Outcome Importance
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>		
Dialysis duration is a predictor of graft loss due to GN recurrence.							High		
Morbid obesity (BMI ≥40 kg/m <sup>2</sup> ) is a predictor of graft loss.							Moderate		
Among elderly, older age may not be a predictor of graft loss.							Low		
FSGS may be a predictor of graft loss due to GN recurrence.							Low		
For other predictors, the evidence was unclear or there was insufficient evidence.*							Very Low		

\* See list of predictors evaluated by a single study each below the footnotes.

† Linked with DaVita

‡ Approximately 50% patients were excluded in the multivariate analysis in one study due to missing data. Important confounders were not adjusted for with no further explanations in another study.

§ Biased selection of patient population in one study.

\*\* Database poorly described.

N/A= Not available or not applicable, BMI= body mass index, NS= non-significant, Sig= significant, ESRD= end-stage kidney disease, HR= hazard ratio, CAKUT= congenital anomalies of the kidney and the urinary tract, GN= glomerulonephritis, DM= diabetes mellitus, HTN= hypertension

OPTN = Organ Procurement and Transplantation Network- other names of the database include the Scientific Registry of Transplant Recipients (SRTR) and United Network for Organ Sharing (UNOS), DaVita = Kidney disease and dialysis information, USRDS = The United States Renal Data System, ANZDATA = The Australian and New Zealand Dialysis and Transplantation Registry, UKRR = United Kingdom Renal Registry, MPGN = membranoproliferative glomerulonephritis, APKD = adult polycystic kidney disease  
FSGS = focal segmental glomerulosclerosis, GN = glomerulonephritis

#### Only one study for each of the following predictors:

Outcome = Graft loss (all cause)

- Cerebrovascular disease, presence (vs. absence) OEDTR HR= 2.52 (0.61, 10.36)
- Coronary heart disease, presence (vs. absence) OEDTR HR= 0.60 (0.18, 1.99)
- Creatinine, per 1 mg/dl SRTR/MHD HR= 0.96 (0.81, 1.00)
- Current smoker, vs. never smoker CTS HR= 1.5 (1.4, 1.6)
- Diabetes mellitus, presence (vs. absence) SRTR/DaVita HR= 1.35 (1.14, 1.61)
- Dialysis duration, per 1 year USRDS HR= 1.02 (1.01, 1.02)
- Dialysis modality, peritoneal dialysis (vs. hemodialysis) SRTR/DaVita HR= 1.08 (0.79, 1.47)
- Former smoker, vs. never smoker CTS HR= 1.1 (1.0, 1.1)
- FSGS as cause of ESRD, FSGS (vs. congenital anomalies of the kidneys or urinary tract) USRDS HR= 1.13 (1.07, 1.20)
- Hepatitis B infection, HBV + (vs. HBV -) OPTN/UNOS HR= 0.74 (0.45, 1.24) (in recipients of living donors), 1.06 (0.85, 1.33) (in recipients of deceased donors)
- HIV infection, HIV + (vs. HIV -) SRTR/OPTN HR= 1.18 (0.66, 2.08)

- Intellectual disability, definite intellectual disability, probable intellectual disability (vs. no intellectual disability) UNOS HR= 1.1 (0.5, 2.5), 0.5 (0.3, 2.0)
- Kidney transplant morbidity index, score 1, score 2, score 3, score 4, score 5, score 6, score 7 (vs. score 0) OPTN/UNOS HR= 1.30 (1.16, 1.45), 1.44 (1.29, 1.60), 1.74 (1.56, 1.94), 2.08 (1.87, 2.33), 2.46 (2.19, 2.77), 2.97 (2.58, 3.41), 3.11 (2.55, 3.80)
- Level of education, High school education, some college or bachelor degree, graduate degree (vs. none/grade school) SRTR HR= 1.09 (1.04, 1.14), 0.96 (0.92, 1.01), 0.95 (0.89, 1.02)
- Penal reactive antibody (PRA), PRA 1-30%, 31-80%, >=81% (vs. 0%) SRTR HR= 1.04 (1.00, 1.07), 1.14 (1.09, 1.21), 1.21 (1.12, 1.31)
- Periphervascular disease, presence (vs. absence) OEDTR HR= 2.29 (0.97, 5.41)
- Polycystic kidney disease, presence (vs. absence) SRTR HR= 0.75 (0.72, 0.78)
- Pre-transplant cancer, pre-transplant skin cancer (vs. no pre-transplant skin cancer) UNOS HR= 1.14 (1.02, 1.27)
- Right atrial diameter, per mm OEDTR HR= 1.04 (1.02, 1.07)
- Socioeconomic status (SES), SES low-mid quartile, high-mid quartile, highest quartile (vs. lowest quartile) USRDS HR= 0.95 (0.91, 0.98), 0.91 (0.88, 0.94), 0.83 (0.80, 0.86)
- Young age (pediatric), age 0-4, age 5-9, age 10-12, age age 13-16 (vs. age 25-29) USRDS HR= 0.94 (0.79, 1.13), 0.60 (0.53, 0.68), 0.56 (0.49, 0.64), 0.91 (0.84, 0.98)

*Outcome = Graft loss secondary to GN recurrence*

- Era (2001-2003 vs. 1990-1994) USRDS 0.39 (0.24, 0.64)
- Lupus nephritis as cause of ESRD (vs. other) USRDS HR=0.72 (0.49, 1.06)
- Mesangiocapillary glomerulonephritis type I as cause of ESRD (vs. mean risk for all categories of GN) ANZDATA HR=2.91 (1.53, 5.55)
- MPGN as cause of ESRD (vs. other) USRDS HR=2.57 (1.84, 3.58)
- Pauci-immune crescentic glomerulonephritis as cause of ESRD (vs. mean risk for all categories of GN) ANZDATA HR=nd, NS
- Unspecified pathology of ESRD (vs. other) USRDS HR=0.59 (0.44, 0.78)
- "Other" pathology of ESRD (vs. mean risk for all categories of GN) ANZDATA HR=0.30 (0.13, 0.66)

*Outcome = Graft loss secondary to IgAN recurrence*

- Age (10y increment) ANZDATA HR=0.87 (0.67, 1.13)
- Dialysis duration, 6 months to <1y vs. <6 months, 1y to 5 years vs. <6 months, ≥5 years vs. <6 months ANZDATA HR= 0.73 (0.35, 1.49), 0.50 (0.25, 0.98), 0.40 (0.09, 1.74)
- Era (1998-2007 vs. 1988-1992) ANZDATA HR=0.26 (0.10, 0.66)

### Evidence Profile: Pre-Transplant Predictors of Post-Transplant Outcomes Other Than Death and Graft Loss (from Registry Studies)

Outcome	Predictor	Registries (No. Studies)	Percent w/Predictor	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence	Other Considerations	Summary of Findings			
								Quality of Evidence	Description of Findings	Outcome Importance	
<b>Post-transplant malignancy</b>	Pre-tpx skin cancer	UNOS (1)	1.6	No limitations (0)	NA	Direct (0)	Sparse (-2)	Low	Pre-transplant skin cancer, pre-transplant NMSC, and pre-transplant melanoma were significant predictors of post-transplant malignancy (HR=2.60, 2.89, 1.77)	High	
	<b>New-onset DM</b>	Age	OPTN/UNOS (1)	NA	No limitation (0)	NA	Direct (0)	Sparse (-2)	Low	Increased age, per decade, is significantly associated with new-onset DM (HR=1.29).	Moderate
		HTN	OPTN/UNOS(1)	77.6	No limitation (0)	NA	Direct (0)	Sparse (-2)	Low	Hypertension is a statistically significant predictor of new-onset DM (HR=1.26).	
		BMI	OPTN/UNOS (1)	nd	No limitation (0)	NA	Direct (0)	Sparse (-2)	Low	Obesity (BMI 25-30) and morbid obesity (BMI >30) significantly predict new-onset DM (HR=1.39, 1.84)	
HCV Antibody	OPTN/UNOS (1)	4.2	No limitation (0)	NA	Direct (0)	Sparse (-2)	Low	Positive hepatitis C virus antibody is a statistically significant predictor of new-onset DM (HR=1.42).			
<b>Overall summary:</b>								<b>Quality of Overall Evidence:</b>			
Sparse data suggest that pre-transplant skin cancers predict post-transplant malignancies, and that increasing age, hypertension, obesity, and HCV significantly predict new-onset DM after transplantation								Low			

BMI = body mass index, DM = diabetes mellitus, HCV = hepatitis C virus, HR = hazard ratio, nd = no data; NMSC=Non-melanoma skin cancer

OPTN = Organ Procurement and Transplantation Network- other names of the database include the Scientific Registry of Transplant Recipients (SRTR) and United Network for Organ Sharing (UNOS)



PMID	Author	Year	Test	Subgroup description	Outcome	Definition	Outcome measurement timepoint	Sample size (N)	Frequency (event) rate, %	Relative effect	P value	Overall quality
26517474	Maldonado	2015	Stanford Integrated Psychosocial Assessment for Transplantation	SIPAT score= 0-6	Mortality	nd	1 y post-Txp	54	(5) 9.3%	HR 0.98 (0.92, 1.06)	0.652	B
				127				(18) 14.2%				
				SIPAT score>= 7-20	Organ failure	nd		36	(3) 8.3%	HR 0.99 (0.96, 1.04)	0.803	
				SIPAT score>= 21				54	(3) 5.6%			
				SIPAT score= 0-6	Nonadherence	nd		127	(8) 6.3%	AUC 0.60 (0.50, 0.71)	0.058	
				SIPAT score= 7-20				36	(1) 2.8%			
				SIPAT score>= 21	Mortality			54	(6) 11.5%			
				SIPAT score= 0-6				127	(20) 15.9%			
				SIPAT score= 7-20	Organ failure			36	(10) 27.8%			
				SIPAT score>= 21				55	0.0%			
SIPAT score any	Nonadherence			55	0.0%							
SIPAT score any				55	(12) 22.2%							
SIPAT score any	Graft failure	nd	nd	55	(12) 22.2%	nd	nd					
SIPAT score any				33	10 (30.3%)							
21620037	Calia	2011	Eysenck Personality Questionnaire [other tests (Fear Inventory, MOCQ-R, STAI) and other items in EPQ were not associated with graft failure]	median score: w/graft failure 3.5 ± 1.6 vs. w/o graft failure 2.3 ± 1.3	Graft failure	nd	nd	33	10 (30.3%)	nd	nd	B
				Higher scores on the psychoticism factor suggested solitude and difficulty adapting to the external environment.								
			CBA-2,0 "Primary Scale" includes EPC, Fear Inventory, MOCQ-R, STAI	nd	Graft rejection	nd	nd	33	nd	OR 2.088 (1.083, 1.025)	0.028	

			RCT: Adequate generation of a randomized sequence	RCT.....Allocation concealment	RCT.....Blinding of PATIENTS	RCT.....Blinding of PROVIDERS	RCT.....Intention-to-treat-analysis	NonRCT.....Representativeness of the case?	NonRCT.....Selection of the exposed cohort
			<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly unsealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>truly representative; not representative; OR no description</p>	<p>drawn from the same source; not drawn from a different source; OR no description</p>
26517474	Maldonado	2015	na	na	na	na	na	unclear	low
21620037	Calia	2011	NA	NA	NA	NA	NA	truly representative	drawn from the same source



			NonRCT.....Ascertainment of exposure	NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
PMID	Author	Year	secure record or self report; not a secure record or self-report; OR no description	yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
26517474	Maldonado	2015	low	low for mortality and graft loss	unclear	unclear	unclear	unclear	none
21620037	Calia	2011	secure record	unclear	NA	NA	unclear	low	none

## Evidence Profile: Psychosocial testing

Outcome (Test)	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
<b>Death (SIPAT)</b>	1	217 (55)*	Serious limitations (-1)	N/A	Indirect* (-1)	Sparse (-1)	Very low	No association between pre-Txp SIPAT score and post-Txp mortality (across organ transplants)	Critical
<b>Graft loss (SIPAT)</b>	1	217 (55)*	Serious limitations (-1)	N/A	Indirect* (-1)	Sparse (-1)	Very low	No association between pre-Txp SIPAT score and post-Txp graft loss (across organ transplants)	Critical
<b>Graft loss (EPQ)</b>	1	33 (33)	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	Psychoticism, as assessed by the Eysneck Personality Questionnaire, was associated with a 30% rate of graft failure.	Critical
<b>Non-adherence (SIPAT)</b>	1	217 (55)*	Serious limitations (-1)	N/A	Indirect* (-1)	Sparse (-1)	Very low	"Minimally acceptable to high risk" SIPAT score possibly associated with increased risk of post-Txp non-adherence (across organ transplants) (AUC P=0.058)	Moderate
<p><b>Overall summary:</b> Pre-transplant SIPAT score not associated with post-transplant mortality or graft loss (across organ transplants); possible association between high risk SIPAT score and non-adherence. Psychoticism on Eysneck Personality Questionnaire associated with higher risk of graft failure.</p>							<p><b>Quality of Overall Evidence:</b> Very low</p>		

EPQ = Eysenck Personality Questionnaire; SIPAT = Stanford Integrated Psychosocial Assessment for Transplantation, Txp = transplant

\* 55 of 217 had kidney transplants. Others had heart (n=36), lung (n=68), and liver (n=58). No kidney transplant patients died or had graft loss at 1 year post-transplant.

PMID	Author	Year	Type of article	Country	Era (Study years)	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Intervention-specific characteristic 1	Intervention-specific characteristic 2	Arm (Intervention)
19459828	Dunn	2009	peer-reviewed journal article	USA	1982-2006	unclear	nd	nd	CKD 5	HD			<b>1st graft loss due to Non-adherence, Retransplanted per protocol</b> 1st graft loss not due to non-adherence <b>1st graft loss due to Non-adherence, Retransplanted per protocol</b> 1st graft loss not due to non-adherence <b>1st graft loss due to Non-adherence, Retransplanted per protocol</b>  1st graft loss not due to non-adherence

PMID	Author	Year	Intervention description	Outcome	Definition	Outcome measurement timepoint	Sample size (N)	Frequency (event) rate, %	Relative effect	P value	Overall quality		
19459828	Dunn	2009	Selective retransplant protocol	Graft loss	death censored	8 years	35	45%	HR 1.51	0.11	C	Also data at 1, 3, 5 years	
				Death		8 years	35	68%	HR nd	0.25 (multivariate)			
				Graft loss due to non-adherence			35	72%			0.0001		Also data at 1, 3, 5 years 2nd graft loss due to similar reasons in these 5 patients
						552	68%						
						552	72%						
							552	14% (5)					
							552	2% (10)					

			RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Representativeness of the case?	NonRCT:.....Selection of the exposed cohort
PMID	Author	Year	<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly un concealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>truly representative; not representative; OR no description</p>	<p>drawn from the same source; not drawn from a different source; OR no description</p>
19459828	Dunn	2009	N/A	N/A	N/A	N/A	N/A	truly representative	drawn from the same source

			NonRCT.....Ascertainment of exposure	NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
PMID	Author	Year	secure record or self report; not a secure record or self-report; OR no description	yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
19459828	Dunn	2009	secure record	no	low	low	low	low	Poor reporting. Omitted their patients transplanted elsewhere (against their "protocol")

## Evidence Profile: Nonadherence

Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
<b>Death</b>	1	587	Very serious limitations (-2)	N/A	Direct (0)	Sparse (-1)	Very low	No difference between patients retransplanted after non-adherence vs. after adherence. No comparison with patients with non-adherence who were not retransplanted.	Critical
<b>Graft loss</b>	1	587	Very serious limitations (-2)	N/A	Direct (0)	Sparse (-1)	Very low	No difference between patients retransplanted after non-adherence vs. after adherence. No comparison with patients with non-adherence who were not retransplanted.	Critical
<b>Graft loss due to nonadherence</b>	1	587	Very serious limitations (-2)	N/A	Direct (0)	Sparse (-1)	Very low	Among originally non-adherent, 14% lost 2 <sup>nd</sup> graft due to non-adherence; among originally adherent 2% lost 2 <sup>nd</sup> graft due to non-adherence (P=0.0001). Among non-adherent, same reasons for non-adherence.	High
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>		
Overall patients who lost first graft due to non-adherence do as well after retransplantation as patients who lost first graft for other reasons. No comparison with those who were not retransplanted							Very low		

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean (SD) or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	BMI or weight	Diabetes measures pretransplant	Pre-Txp Category	Category description
16499590	Shishido	2006	peer-reviewed publication	Japan	1999-2003	prospective observational study	9.7 (5.4) (2.5, 18)	64	ND	CKD 4-5	16.5 kg/m <sup>2</sup> (12.2, 26.8)	HbA1c: 4.5	IGT, pre-Txp	Impaired glucose tolerance – 2 h PG ≥140 mg/dL and <200 mg/dL
20169406	Iida	2010	peer-reviewed publication	Japan	2001-2006	retrospective observational study	37.5 (19.7, 51.2)	64	ND	CKD 4-5	20.9 kg/m <sup>2</sup>	248 (65.6%) patients showed the normal IFG pattern (Group 1) 115 (30.4%) showed the IFG or IGT pattern (IFG/IGT; Group 2) 15 (4.0%) showed the DM pattern (Group 3)	IGT, pre-Txp NGT, pre-Txp IGT, pre-Txp NGT, pre-Txp	Normal glucose tolerance – 2 h PG < 140 mg/dL IFG/IGT pattern was defined as a fasting blood glucose level between 100 and 125 mg/dl or a 2-h glucose level between 140 mg/dl and 199 mg/dl in the OGTT OGTT involved the administration of 75 g of glucose, was performed 2 weeks before transplantation. Normal pattern was defined as a fasting blood glucose level <100 mg/dl or a 2-h glucose level <140 mg/dl in the OGTT. OGTT involved the administration of 75 g of glucose, was performed 2 weeks before transplantation. IFG/IGT pattern was defined as a fasting blood glucose level between 100 and 125 mg/dl or a 2-h glucose level between 140 mg/dl and 199 mg/dl in the OGTT OGTT involved the administration of 75 g of glucose, was performed 2 weeks before transplantation. Normal pattern was defined as a fasting blood glucose level <100 mg/dl or a 2-h glucose level <140 mg/dl in the OGTT. OGTT involved the administration of 75 g of glucose, was performed 2 weeks before transplantation.
21949218	Chakkerla	2011	peer-reviewed publication	US	1999-2008	retrospective observational study	49 (15)	57	ND	CKD 4-5	27 (6) kg/m <sup>2</sup>	FPG 92 (11) mg/dL	IGT, pre-Txp NGT, pre-Txp	patients with FG ≥100 mg/dL No FG ≥100 mg/dL
21336240	Caillard	2011	peer-reviewed publication	France	2005-2008	retrospective observational study	50 (14)	67	ND	CKD 4-5	25.4 (4.4) kg/m <sup>2</sup>	IGT was diagnosed in 37 patients (15%)	IGT, pre-Txp NGT, pre-Txp	pretransplant IGT on pretransplant OGTT normal GT on pretransplant OGTT
12480976	Mathew	2003	peer-reviewed publication	India	1996-1998	prospective observational study	32.9 (9.7)	83.6	ND	CKD 4-5	18.3 (2.4) kg/m <sup>2</sup>	2-h glucose >140 mg/dL 1-h glucose > 156mg/dL	IGT, pre-Txp NGT, pre-Txp	IGT or PTDM on pretransplant OGTT NGT on pretransplant OGTT



PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean (SD) or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	BMI or weight	Diabetes measures pretransplant	Pre-Txp Category	Category description
24468096	Tokodai	2014	peer-reviewed publication	Japan	2000-2011	retrospective observational study	43.9	68	ND	CKD 4-5	21.3 kg/m <sup>2</sup>	HbA1c: 5.07%	<b>IGT, pre-Txp</b>	<b>abnormal FPG on OGTT</b>
NA	<b>Ramesh Prasad</b>	2009	peer-reviewed publication	Canada	2003-2006	case-control analysis	49.8 (10.5)	64	SCr 132 {34} μmol/L	CKD 4-5	75.6 {18} kg	OGTT abnormalities pretransplant: (12 of 78) 15%	NGT, pre-Txp <b>Impaired fasting glucose</b>  Normal fasting glucose  <b>Impaired glucose tolerance</b>  Normal glucose tolerance  <b>Abnormal random blood glucose</b>  Normal random blood glucose  <b>OGTT</b>	normal FPG on OGTT <b>FBG between 6.1 and 6.9 mmol/l</b>  FBG <6.1 mmol/l  <b>2-h glucose between 7.8 and 11.0 mmol/l</b>  2-h glucose <7.8 mmol/l  <b>RBG &gt;6.0 mmol/L</b>  RBG ≤6.0 mmol/L

PMID	Author	Year	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Relative effect	P value	Sn (95% CI)	Sp (95% CI)	PPV (95% CI)	NPV (95% CI)	Overall Quality	
16499590	Shishido	2006	PTDM	The definition and diagnosis of diabetes after transplantation was based on the currently accepted definition of DM and IGT recently defined by the WHO	1.5 years	18	(0) 0%	ND	ND					A	Children
20169406	Iida	2010	Permanent NODAT	Patients who developed permanent antidiabetic agent-dependent DM	>2 years	115	(2) 5.4% (7) 6.1%	ND OR 2.59 (0.85, 7.88)	ND 0.084					A	
						248	(6) 2.4%								
			Transient NODAT	Patients who had required transient antidiabetic therapy more than once during the follow-up period	>2 years	115	(11) 9.6%	OR 1.71 (0.80, 3.66)	1.16						
						248	(17) 6.9%								
21949218	Chakkerla	2011	NODAT	NODAT was diagnosed if a patient had HbA1c $\geq$ 6.5%, fasting venous plasma glucose $\geq$ 126 mg/dL, or was receiving diet or medical therapy for diabetes between 1 month and 1 year post transplant	1 year	72	(30) 42%	ND	ND					B	
				Multivariate analysis using a standard model, in which both continuous and discrete variables were included and weighted according to the $\beta$ -coefficients in the multivariate logistic model		246	(55) 22% pretransplant FPG per 10 mg/dL increase	ND OR 1.35 (1.06, 1.73)	ND 0.02						
				Multivariate analysis using a dichotomous model, in which continuous variables were dichotomized based on clinically relevant cut points (values below and above the cut point were assigned a value of 0 and 1, respectively) and were weighted according to the $\beta$ -coefficients in the multivariate logistic model		FG $\geq$ 100 mg/dL	ND	OR 2.07 (1.12, 3.85)	0.02						
21336240	Caillard	2011	NODAT	Diagnosed if one of the following was present: a fasting glucose level more than 126 mg/dL (7 mM/L) on at least two occasions; a nonfasting glucose level more than 200 mg/dL (11.1 mM/L); a 2-hr glucose level of a standard OGTT more than 200 mg/dL; or the need for antidiabetic medication. IGT was defined based on ADA guidelines (2-hr glucose level of a standard OGTT between 140 and 200 mg/dL)	3 years	22	(11) 50%	ND	ND					A	
				Multivariate analysis, the risk of developing NODAT increase in recipients with one risk factor from age (more than or less than 50 years), type of nephropathy (ADPKD or not), and the result of pretransplant OGTT (IGT or normal)		98	(20) 20%	ND	ND	2.4-fold (0.8, 7)	0.1				
				Multivariate analysis, the risk of developing NODAT increase in recipients with two risk factor from age (more than or less than 50 years), type of nephropathy (ADPKD or not), and the result of pretransplant OGTT (IGT or normal)		ND	ND	ND	5.2-fold (1.8, 15)	0.02					
				Multivariate analysis, the risk of developing NODAT increase in recipients with three risk factor from age (more than or less than 50 years), type of nephropathy (ADPKD or not), and the result of pretransplant OGTT (IGT or normal)		ND	ND	ND	14-fold (3,67)	0.01					
12480976	Mathew	2003	PTDM	PTDM based on 1-h glucose value >50th percentile	25.6 months	80	ND	OR 2.9 (1.2, 6.9)	0.01					B	
						76	ND	ref	ref						

PMID	Author	Year	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Relative effect	P value	Sn (95% CI)	Sp (95% CI)	PPV (95% CI)	NPV (95% CI)	Overall Quality
24468096	Tokodai	2014	NODAT	Defined according to the American Diabetes Association: as the presence of diabetes symptoms plus casual plasma glucose concentrations $\geq 11.1$ mmol/L (200 mg/dL) or FPG concentrations $\geq 7$ mmol/L (126 mg/dL); fasting was defined as the absence of caloric intake for at least 8 h. Impaired fasting glucose was defined as $5.6 \leq \text{FPG} < 7$ mmol/L; multivariate logistic regression analyses adjusted by recipient age, gender, hepatitis C virus, and use of tacrolimus	1 year	ND	ND	OR 1.03 (0.97, 1.09)	0.38					A
NA	Ramesh Prasad	2009	NODAT	Defined based on a minimum of two FBG measurements $\geq 7.0$ mmol/L and/or RBG $\geq 11.1$ mmol/L, obtained on separate days in the absence of acute illness	6 months	8	ND (4) 50%	ref ND	ref 0.03					B
				Defined based on a minimum of two FBG measurements $\geq 7.0$ mmol/L and/or RBG $\geq 11.1$ mmol/L, obtained on separate days in the absence of acute illness	6 months	143	(27) 18%	ND	ref					
				Defined based on a minimum of two FBG measurements $\geq 7.0$ mmol/L and/or RBG $\geq 11.1$ mmol/L, obtained on separate days in the absence of acute illness	6 months	4	(3) 75%	ND	0.0006					
				Defined based on a minimum of two FBG measurements $\geq 7.0$ mmol/L and/or RBG $\geq 11.1$ mmol/L, obtained on separate days in the absence of acute illness	6 months	147	(28) 19%	ND	ref					
				multivariate analysis of pretransplant RBG $> 6.0$ mmol/L adjusted for acute rejection and age per 10 years	6 months	ND	ND	OR 6.1 (2.1, 18.2)	0.001					
				multivariate analysis of pretransplant RBG $> 6.0$ mmol/L adjusted for acute rejection and age per 10 years	6 months	ND	ND	ref	ref					
			Performance characteristics	values for OGTT	6 months	151				23%	96%	58%	83%	



			ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Dx test studies.....Reference standard	Dx test studies.....Same reference standard	Dx test.....Independent reference standard	Dx test.....Interpretation of results	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
PMID	Author	Year	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability); there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding; >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers; >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	Did the whole sample or a random selection of the sample, receive verification using a reference standard of diagnosis? [yes/no/unclear]	Did patients receive the same reference standard regardless of the index test result? [yes/no/unclear]	Was the reference standard independent of the index test (i.e. the index test did not form part of the reference standard)? [yes/no/unclear]	Were the index test results interpreted without knowledge of the results of the reference standard? [yes/no/unclear]	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
1649590	Shishido	2006	unclear	low	NA	NA	NA	NA	none
17302602	Joss	2007	unclear	low	NA	NA	NA	NA	none
20169406	Iida	2010	unclear	low	NA	NA	NA	NA	none
21949218	Chakkerla	2011	unclear	low	NA	NA	NA	NA	No description of the test as an intervention pretransplant
21336240	Caillard	2011	unclear	low	NA	NA	NA	NA	none
12480976	Mathew	2003	unclear	low	NA	NA	NA	NA	Some inconsistencies in the number of patients analyzed by OGTT and those receiving OGTT
24468096	Tokodai	2014	unclear	low	NA	NA	NA	NA	none
ND	Nam	2001	unclear	low	NA	NA	NA	NA	none
NA	Ramesh Prasad	2009	unclear	low	unclear	unclear	unclear	yes	none

## Evidence Profile: Glucose tolerance testing pre-transplantation

Test (Outcome)	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
IGT/IFG [vs. NGT] (NODAT)	7	>1163*	No limitations (0)	No important inconsistencies (0)	Direct (0)	None (0)	High	IGT pre-transplantation imparts an approximately double risk of NODAT 6 months to 3 years.	Moderate
RBG [vs. normal] (NODAT)	1	≤151	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	Abnormal RBG has significant association with NODAT (OR=6.1)	
OGTT† (NODAT)	1	151	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	Abnormal OGTT† has sensitivity = 23% for NODAT and specificity = 96%.	
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>		
Patients with pre-transplant IGT or IFG are at increased risk of NODAT. However, pre-transplant OGTT has poor sensitivity, but high specificity for NODAT.							High		

FG = fasting glucose, IGT/IFG = impaired glucose tolerance and/or impaired fasting glucose (e.g., FG 5.6-6.9 mmol/L (100-125 mg/dL), 2-hour glucose 7.8-10.9 mmol/L (140-196 mg/dL), N/A = not applicable,

NGT = normal glucose tolerance, NODAT = new-onset diabetes after transplantation, OGTT = oral glucose tolerance test, RBG = random blood glucose >6.0 mmol/L (108 mg/dL).

\* 1 study did not report sample sizes.

† FBG between 6.1 and 6.9 mmol/L (110-124 mg/dL) and/or 2-h glucose between 7.8 and 11.0 mmol/L (140-199).

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean (SD) or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Intervention-specific characteristic 1	Intervention-specific characteristic 2	Arm (Intervention)	Intervention description
22958221	Zuber	2012		France		Retrospective case series  (including data from previously published case reports)	0.8-33 y	ND	5D	NA			Eculizumab peri-transplant for aHUS (and high risk for recurrence based on complement mutation analysis)	1. Dose <24 h before transplant and 2nd dose <24 h after transplant; 2. Dose 1 week before transplant; 3. Plasma exchange therapy at time of transplant and converted to eculizumab therapy
24933457	Matar	2014		USA		Retrospective case series	0.9-57 y	33%	5D	NA			Eculizumab peri-transplant for aHUS (and high risk for recurrence based on complement mutation analysis) No eculizumab Eculizumab peri-transplant for aHUS (and high risk for recurrence based on complement mutation analysis) No eculizumab	Dose < 24 h before living donor transsplant  Dose < 24 h before living donor transsplant

KDIGO - Transplant Candidate  
 Guideline Topic: Recurrence aHUS  
 Categorical outcomes

PMID	Author	Year	Intervention Duration	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Relative effect	P value	Overall Quality
22958221	Zuber	2012	Lifetime of allograft	Recurrent aHUS	ND	variable	9 with peri-transplant eculizumab	11%			C
24933457	Matar	2014	6 m in 3 patients, lifelong in 1 patient	Graft loss	ND	variable	4	0%			B
				Recurrent aHUS	ND	variable	8	50%			
							4	0%			
8	38%										



PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Intervention	Intervention specifics
11292291	Ohta	2001		Japan		Retrospective, non-randomized control study	4.6 +/- 2.2 y	ND	5D	NA	Plasmapheresis before transplant for FSGS	5, 3 and 1 day before transplantation
21338460	Gonzalez	2011		USA		Retrospective, non-randomized with historical controls	12.8 y	59%	5D	NA	Plasmapheresis before transplant for FSGS	
15605284	Hubsch	2005		USA	1999-2003 (daclizumab induction)	Retrospective, non-randomized	7.0 +/- 4.0 y	63%	5D	NA	Plasmapheresis before transplant for FSGS	
					1979-1998 (pre-daclizumab)		7.0 +/- 4.0 y	50%	5D	NA	No plasmapheresis	
					1999-2003 (daclizumab induction)						Plasmapheresis before	
					1979-1998 (pre-daclizumab)						No plasmapheresis	
16303004	Gohh	2005		USA		Non-randomized, non-comparative	35 +/- 12 y	40%	5D	NA	Preemptive plasmapheresis	8 sessions of peri-operative plasmapheresis in patients at high risk of FSGS recurrence (prior recurrence in allograft or rapid progression to ESRD)
25715638	Lionaki	2015		Greece		Non-randomized, non-comparative	30.9 y	72%	5D	NA	immunoabsorption	3 sessions of immunoabsorption in the week prior to transplant and 3 sessions in the week after transplant for kidney transplant candidates with FSGS with a scheduled live donor transplant



PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Intervention-specific characteristic 1	Intervention-specific characteristic 2	Arm (Intervention)
11292291	Ohta	2001		Japan		Retrospective, non-randomized control study	4.6 +/- 2.2	ND	5-D	NA	Plasmapheresis (prophylactic)	5, 3 and 1 day before transplantation	Plasmapheresis No plasmapheresis

PMID	Author	Year	Intervention description	Intervention Duration	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Baseline Value	Final Value	Change	P value	Overall Quality
11292291	Ohta	2001			Proteinuria	g/d	Variable	15	ND 6 ND	16.9 51.2	NA NA	NA NA	C

## Evidence Profile: Treatments to prevent kidney disease recurrence

Disease (Treatment)	Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
								Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
FSGS (plasmapheresis)	Graft loss	2	49	Very serious limitations (-2)	No important inconsistencies (0)	Direct (0)	Small, sparse, old (-2)	Very low	No difference plasmapheresis vs. none	Critical
	Recurrent FSGS	5	111	Very serious limitations (-2)	No important inconsistencies (0)	Direct (0)	Small, mostly old (-2)	Very low	No difference plasmapheresis vs. none	High
aHUS (eculizumab)	Graft loss	1	12	Serious limitations (-1)	N/A	Direct (0)	Small, sparse (-2)	Very low	Possible lower rate with eculizumab	Critical
	Recurrent aHUS	2	21*	Very serious limitations (-2)	No important inconsistencies (0)	Direct (0)	Small, sparse (-1)	Very low	Possible lower rate with eculizumab	High
<p><b>Overall summary:</b> Unclear evidence that plasmapheresis does not affect FSGS recurrence or graft loss, but that eculizumab may lower rates of aHUS recurrence and graft loss.</p>								<p><b>Quality of Overall Evidence:</b> Very low</p>		

aHUS = atypical hemolytic uremic syndrome, FSGS = focal segmental glomerulosclerosis, N/A = not applicable.

\* Includes case reports

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Reason for nephrectomy	Arm (Intervention)
9610554	Erturk	1998	peer-reviewed publication	US	1984-1995	prospective cohort study	31 (10-67)	55.5	ND	ND	vesicoureteral reflux	nephrectomy prior to txp corrected reflux persistent reflux nephrectomy prior to txp corrected reflux persistent reflux nephrectomy prior to txp corrected reflux persistent reflux nephrectomy prior to txp corrected reflux persistent reflux reflux no reflux reflux reflux nephrectomy of first graft
14724448	Ramos	2004	peer-reviewed publication	US	ND	retrospective cohort study	45.3	100	ND	CKD 5	BK virus-associated nephropathy	



				RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Selection of treated and control cohort?
				<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly unsealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>drawn from the same source; drawn from a different source;            OR            no description</p>
9610554	Erturk	1998	NA		NA	NA	NA		low
1472448	Ramos	2004	NA		NA	NA	NA		NA



				NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
				yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
9610554	Erturk	1998	low		unclear	low	unclear	low	none
14724448	Ramos	2004	NA		NA	NA	unclear	low	none

## Evidence Profile: Transplantation outcomes after pre-transplant nephrectomy for UTI or BKAN

Outcome (Kidney Disease)	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
<b>Death (UTI)</b>	1 (UTI)	36	Very serious limitations (-2)*†	N/A	Indirect (-1)*	Sparse, small (-2)	Very low	Overall survival rate in patients with reflux was high at long-term follow-up (>90% at 5 years)	Critical
<b>Graft Loss (UTI)</b>	(UTI)	36	Very serious limitations (-2)*†	N/A	Direct (0)	Sparse, small (-2)	Very low	No significant difference was shown between nephrectomy and no nephrectomy at long-term follow-up	Critical
<b>Complicated UTI (UTI)</b>	1 (UTI)	36	Very serious limitations (-2)*†	N/A	Direct (0)	Sparse, small (-2)	Very low	No significant difference was shown between nephrectomy and no nephrectomy at long-term follow-up	High
<b>Uncomplicated UTI (UTI)</b>	1 (UTI)	36	Very serious limitations (-2)*†	N/A	Direct (0)	Sparse, small (-2)	Very low	No significant difference was shown between nephrectomy and no nephrectomy at long-term follow-up	Moderate
<b>BKAN Recurrence (BKAN)</b>	1 (BKAN)	10	Very serious limitations (-2)*†	N/A	Indirect (-1)*	Sparse, small (-2)	Very Low	Recurrence rate was 10% in short-term follow-up after transplantation with pre-transplant nephrectomy	High
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>		
Post-transplant survival high after nephrectomy for vesicoureteral reflux; graft loss rates similar. A percentage of patients with BKAN have recurrence post-transplant after nephrectomy.							Very Low		

BKAN = BK virus associated nephropathy; UTI = Urinary tract infection / vesicoureteral reflux , N/A = Not applicable, BKAN = BK virus-associated nephropathy

\* The study did not compare the effect of nephrectomy with no nephrectomy on patient outcome.

† Potential confounding effects were not adjusted in the non-randomized controlled study. BKAN study was noncomparative.

PMID	Author	Year	Type of article	Country	Era (Study years)	Study design	Age [mean (SD) or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Arm (Cohort)	Cohort description	Outcome
22236928	Jung	2012	peer-reviewed journal article	South Korea	2000-2010	retrospective	[42 (17-23)]	62%	CKD 5	HD	<p><b>Tuberculin Skin Test</b></p> <p>Tuberculin Skin Test  Tuberculin Skin Test +  Tuberculin Skin Test -  Tuberculin Skin Test + / TB exposure or h/o TB  Tuberculin Skin Test - / TB exposure or h/o TB</p> <p>Tuberculin Skin Test NA / TB exposure or h/o TB  Tuberculin Skin Test + / no TB exposure, no h/o TB</p> <p>Tuberculin Skin Test - / no TB exposure, no h/o TB  Tuberculin Skin Test NA / no TB exposure, no h/o TB  TB exposure or h/o TB  no TB exposure, no h/o TB  h/o TB  no h/o TB  h/o TB  no h/o TB</p>	<p>TST was conducted before transplant according to the pre-transplant evaluation protocol.</p> <p>Previously healed TB on CXR</p> <p>Previous TB history</p>	<p>TST Positive</p> <p>post-transplant TB</p>
22802098	Kim	2013	peer-reviewed journal article	South Korea	2010-2012	prospective	[47 (20-69)]	56%	CKD 5	HD	<p><b>Tuberculin Skin Test</b></p> <p>QuantiferON-TB Gold In-Tube test (QFT-GIT)  QuantiferON-TB Gold In-Tube test (QFT-GIT)  QuantiferON-TB Gold In-Tube test (QFT-GIT)  Post-tpx check</p>	<p>One-step TST was conducted before elective transplant surgery.</p> <p>One-step TST was conducted before elective transplant surgery.</p> <p>Check for respiratory symptoms, physical examination, chest radiography, and sputum analysis very 1-2 months for mean follow-up of 387 days (13-661).</p>	<p>TST Positive</p> <p>TST Positive</p> <p>TB-specific Ag +  TB-specific Ag -  TB-specific Ag indeterminate</p> <p>TB+</p>

PMID	Author	Year	Definition	Sample size (N)	Frequency (event) rate, %	Relative effect	Adjusted for	P value	Overall quality
22236928	Jung	2012	Induration of $\geq 5$ mm diameter	729	0.313	nd		nd	B
				729	2%	nd		nd	
				228	3.5%	aOR 3.50 (1.12, 10.93)	age, sex, BMI, DM, previously healed TB on CXR, TB history, CMV serological mismatching, HLA mismatching	0.031	
				501	1.0%	ref		ref	
				18	0.0%	nd		nd	
				20	10.0%	RR 4.21 (1.67, 10.61) (vs TST-negative/no TB exposure and no h/o TB)		0.002	
				17	11.8%	nd		nd	
				210	3.8%	RR 6.31 (1.66, 24.03) (vs TST-negative/no TB exposure and no h/o TB)		0.007	
				481	0.6%	ref		ref	
				351	2.3%	nd		nd	
				55	7.3%	RR 4.22 (1.39, 12.87)		0.011	
				1042	1.8%	ref		ref	
				nd	nd	aOR 8.69 (1.00, 75.51)	age, sex, BMI, DM, TST +, TB history, CMV serological mismatching, HLA mismatching	0.05	
				nd	nd	ref		ref	
				nd	nd	aOR 0.24 (0.01, 4.11)	age, sex, BMI, DM, previously healed TB on CXR, TST +, CMV serological mismatching, HLA mismatching	0.322	
22802098	Kim	2013	TST $\geq 5$ mm	119	29%	nd		nd	B
			TST $\geq 10$ mm	119	19%	nd		nd	
				126	42%	nd		nd	
				126	53%	nd		nd	
				126	5%	nd		nd	
			positive result	126	0%	nd		nd	
								nd	

			RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Representativeness of the case?	NonRCT:.....Selection of the exposed cohort	
	PMID	Author	Year	<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly un concealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. i.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>truly representative; not representative; OR no description</p>	<p>drawn from the same source; not drawn from a different source; OR no description</p>
	22236928	Jung	2012	N/A	N/A	N/A	N/A	N/A	no description	drawn from the same source
	22802098	Kim	2013	N/A	N/A	N/A	N/A	N/A	truly representative	drawn from the same source

PMID	Author	Year	NonRCT.....Ascertainment of exposure  secure record or self report; not a secure record or self-report; OR no description	NonRCT.....Demonstration that outcome of interest was not present at start of study  yes; no; unclear	COMPARATIVE....Baseline differences between groups accounted for  For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	COMPARATIVE...Outcome assessment timing (across interventions)  There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	ALL.....Blinding of OUTCOME ASSESSORS  There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	ALL.....Dropouts/missing data (attrition bias)  There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.  There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
22236928	Jung	2012	secure record	no	N/A	N/A	low	low	
22802098	Kim	2013	secure record	no	N/A	N/A	low	low	TST data missing for n= 9 patients.

### Evidence Profile: Tuberculosis testing

Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
TB post-Txp	2	848	Serious limitations (-1)	Important inconsistencies (-2)	Direct (0)	None	Very low	One study from South Korea found TST to be strong predictor of post-Txp TB. However, another study from South Korea found equal rates of post-Txp TB regardless of pre-Txp TST (0% in South Korea).	High
<b>Overall summary:</b> TST pre-transplant does not consistently predict post-transplant tuberculosis							<b>Quality of Overall Evidence:</b> Very low		

TB = tuberculosis, TST = tuberculin skin test, Txp = transplant.

\* A third study from South Korea found no incidence of post-transplant tuberculosis

KDIGO - Transplant Candidate  
Guideline Topic: TB Treatment  
Categorical outcomes

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Arm (Intervention)	Intervention description
27548035	Simkins	2016	peer-reviewed publication	US	2012-2014	retrospective cohort study	59.81 {10.22}	66%	CKD 4: 2% CKD 5-ND: 5% CKD 5-D: 94%	ND	Short course TB treatment	RPT 900 mg + INH 15 mg/kg
10970979	Vachharajani	2000	peer-reviewed publication	India	198-1991	Retrospective "NRCS"	39.9 {12.7}	67%	HD 100%	nd	Full course TB treatment Short course TB treatment	INH 5 mg/kg H 200 mg, R 450 mg, Z 750 mg, E 800 mg, dose adjusted for pt with liver dysfunction
	Malhotra	1986	peer-reviewed article	India	nd	nd	[28.9]	82%	CKD 5	HD	Short course TB treatment	Isoniazid 200 mg/day + EMB 7.5 mg/kg/day + Rifampin 450-600 mg/day + Pyridoxine 10 mg/day
24142036	Lopez de Castilla	2014	peer-reviewed publication	US	2012	prospective cohort study	total: 57 (33-75)	total: 82%	ND	ND	Short course TB treatment	Rifapentine 750-900 mg + Isoniazid 15 mg/kg QW



KDIGO - Transplant Candidate  
 Guideline Topic: TB Treatment  
 Categorical outcomes

PMID	Author	Year	Intervention Duration	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Relative effect	P value	Overall Quality
27548035	Simkins	2016	12 week	TB reactivation	ND	mean 2.5 years	43	(0) 0%	Same: No events either arm	ND	B
			9 months				110	(0) 0%			
	Vachharajani	2000	6 mo in 2 pt, 3 mo in 2 pt	TB reactivation	post-transplant	ND	4	(0) 0%	nd	nd	C
10970979			HR 1 y, ZE 2-3 mo,	TB reactivation	post-transplant	ND	4	(0) 0%	nd	nd	
	Malhotra	1986	3 to 6 months	patient survival		1.5-6.5 years	11	64%	nd	nd	C
			3 to 6 months	TB reactivation	post-transplant	1.5-6.5 years	11	9%	nd	nd	
			4 to 6 months	graft loss	chronic rejection	1.5-6.5 years	11	18%	nd	nd	
24142036	Lopez de Castilla	2014	12 weeks	TB reactivation	ND	ND	8	(0) 0%	NA		B

			RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Selection of treatment and control cohort?	NonRCT:.....Demonstration that outcome of interest was not present at start of study
PMID	Author	Year	<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly unconcealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>drawn from the same source; drawn from a different source; OR no description</p>	<p>yes; no; unclear</p>
27548035	Simkins	2016	NA	NA	NA	NA	NA	low	NA
24142036	Lopez de Castilla Malhorta	2014 1986	NA nd	NA nd	NA nd	NA nd	NA nd	low no description	NA no description
10970979	Vachharajani	2000	NA	NA	NA	NA	NA	High	High

			COMPARATIVE....Baseline differences between groups accounted for	COMPARATIVE....Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
PMID	Author	Year	<p>For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).</p>	<p>There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias</p>	<p>There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.; or:          &gt;&gt; for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. &gt;&gt; for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. &gt;&gt; for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.</p>	<p>There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.</p>	<p>There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere</p>
27548035	Simkins	2016	low	low	unclear	high	
24142036	Lopez de Castilla	2014	NA	NA	unclear	low	
	Malhorta	1986	secure record or self report	no	nd	nd	low
10970979	Vachharajani	2000	Low	No	Unclear	Unclear	<p>Unclear</p> <p>Low</p> <p>Pyridoxine was given (10 mg/day) in patients receiving isoniazid during pre-transplant chemotherapy. Number of chemo patients not defined.            All patients completed full course of treatment, while half of them had KTx in the middle of treatment course in the shorter course group</p>

### Evidence Profile: Tuberculosis treatment, short vs. full course

Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings			
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome	
<b>Death</b>	1	11	Very serious limitations (-2)	N/A	Direct (0)	Sparse, small (-2)	Very low	1/3 dead up to 6.5 years after short course (3-6 mo) TB treatment and KTx	Critical	
<b>Graft loss</b>	1	11	Very serious limitations (-2)	N/A	Direct (0)	Sparse, small (-2)	Very low	2/11 with graft loss up to 6.5 years after short course (3-6 mo) TB treatment	Critical	
<b>TB activation</b>	4	180 (4-110)	Very serious limitations (-2)	No important inconsistencies (0)	Direct (0)	None (0)	Low	No reactivations in 2 comparative studies 3-6 mo vs. 1 year TB treatment; 1 reactivation among 66 patients with short course TB treatment (3-6 mo)	High	
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>			
TB is rare post-transplantation in patients treated with short course (3-6 months) of TB treatment							Low			

KTx = kidney transplantation, TB = tuberculosis

PMID	Author	Year	Type of article	Country	Era (Study years)	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Arm (Intervention)	Intervention description
21114569	Potsangbam	2011	peer-reviewed journal article	India	2007-2008	unclear	[35.38 {9.48}]	94	CKD 5	HD	recombinant HBV vaccine, 2 doses	2 doses at 40 micro grams each
											recombinant HBV vaccine, 3 doses	3 doses at 40 micro grams each
											recombinant HBV vaccine, 4 doses	4 doses at 40 micro grams each
28457920	Kauke	2017	peer-reviewed publication	Germany	2005-2012	retrospective cohort study	49.68	34.6	CKD 5	nd	HBV vaccination	administered during dialysis prior to transplantation

KDIGO - Transplant Candidate  
 Guideline Topic: HBV Vaccine  
 Categorical outcomes

PMID	Author	Year	Intervention duration	Outcome	Definition	Outcome measurement timepoint	Sample size (N)	Frequency (event) rate, %	Relative effect	P value	Overall quality
21114569	Potsangbam	2011	12 months	Anti-HBsAg titres (IU/L) >100	>100	12 months	17	84%	nd	NS overall	C
				Anti-HBsAg titres (IU/L) >100	>100	12 months	17	5%	nd		
				Anti-HBsAg titres (IU/L) >100	>100	12 months	17	11%	nd		
			12 months	Anti-HBsAg titres (IU/L) <10	<10	12 months	17	61.1%	nd		
				Anti-HBsAg titres (IU/L) 10-100	10-100	12 months	17	5.6%	nd		
				Anti-HBsAg titres (IU/L) >100	>100	12 months	17	33.3%	nd		
			12 months	Anti-HBsAg titres (IU/L) <10	<10	12 months	12	61.5%	nd		
				Anti-HBsAg titres (IU/L) 10-100	10-100	12 months	12	5.6%	nd		
	Anti-HBsAg titres (IU/L) >100	>100	12 months	12	23.1%	nd					
28457920	Kauke	2017	median 5.5 years	Anti-HBsAg titres (IU/L) >10	>10	nd	188	141 (75%)	nd	nd	B
				5-year graft survival	nd	5 years	188	93.6%	nd	nd	

			RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Representativeness of the case?	NonRCT:.....Selection of the exposed cohort
PMID	Author	Year	<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly unconcealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>truly representative; not representative; OR no description</p>	<p>drawn from the same source; not drawn from a different source; OR no description</p>
0	Potsangbam	2011	N/A	N/A	N/A	N/A	N/A	no description	drawn from the same source
28457920	Kauke	2017	nn	N/A	N/A	N/A	N/A	truly representative	drawn from the same source

			NonRCT.....Ascertainment of exposure	NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE.....Baseline differences between groups accounted for	COMPARATIVE.....Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.	
	PMID	Author	Year	secure record or self report; not a secure record or self-report; OR no description	yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability); there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
0		Potsangbam	2011	secure record	no	N/A	N/A	low	Pre-transplant vaccine patients not separated out. HBV vaccine type not mentioned.	
28457920		Kauke	2017	secure record	no	N/A	N/A	unclear	low	none



PMID	Author	Year	Type of article	Country	Era (Study years)	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Arm (Intervention)	Intervention description	Intervention notes	Intervention duration
15637753	Lapinski	2005	peer-reviewed journal	Poland	<=2004	unclear	[35-66]	75%	CKD 5	HD	Lamivudine	100 mg after each dialysis (3 times/wk)		12 months
24997462	Ow	2014	peer-reviewed journal	United Kingdom	2000-2008	retrospective	51 [IQR 43-59]	69%	CKD 5	HD	Lamivudine	first dose 35 mg then 10 mg once daily	Lamivudine resistance developed in five patients—two were switched to adefovir; three were changed to combination lamivudine and adefovir.	58 months, median (IQR 37-81)
											Lamivudine	first dose 35 mg then 10 mg once daily	Lamivudine resistance developed in five patients—two were switched to adefovir; three were changed to combination lamivudine and adefovir.	58 months, median (IQR 37-81)
											No treatment			

PMID	Author	Year	Outcome	Definition	Outcome measurement timepoint	Sample size (N)	Frequency (event) rate, %	Relative effect	P value	Overall quality
15637753	Lapinski	2005	elimination of HBV-DNA elimination of HBeAg	the absence of HBs antigens detected in sera the absence of HBe antigens in sera	12 months after treatment 12 months after treatment	16 16	56% 38%	NA NA	NA NA	B
24997462	Ow	2014	complete viral supression	<12 IU/mL	122.4 months	21 (0 at baseline)	48%	+48% c/t baseline	NA	C
			viral supression	1.2-9.9x10 <sup>1</sup>		21 (0 at baseline)	10%	+10% c/t baseline		
			viral supression	1.0x10 <sup>2</sup> -9.9x10 <sup>3</sup>		21 (2 at baseline)	29%	+19% c/t baseline		
			viral supression	1.0x10 <sup>4</sup> -9.9x10 <sup>6</sup>		21 (14 at baseline)	14%	-52% c/t baseline		
			viral supression	>=1.0x10 <sup>7</sup>		21 (5 at baseline)	0%	-24% c/t baseline		
			Death, all cause		122.4 months	21	29% (6)	nd	nd	Cause of death: In patients with complete suppression, two deaths occurred due to non-hepatic causes (one dialysis withdrawal, one sepsis). In patients with incomplete suppression, there were two liver-related deaths (HCC, spontaneous bacterial peritonitis) and two deaths due to dialysis withdrawal. Cause of death: sepsis (five cases), dialysis withdrawal (four cases), cardiac (two cases), non-hepatic malignancy (two cases) and hepatocellular carcinoma (HCC; one case).
						31	45% (14)			

			RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Representativeness of the case?	NonRCT:.....Selection of the exposed cohort	
	PMID	Author	Year	<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly un concealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. i.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>truly representative; not representative; OR no description</p>	<p>drawn from the same source; not drawn from a different source; OR no description</p>
	15637753	Lapinski	2005	nd	nd	nd	nd	nd	not representative	no description
	24997462	Ow	2014	nd	nd	nd	nd	nd	not representative	no description

			NonRCT.....Ascertainment of exposure	NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
PMID	Author	Year	secure record or self report; not a secure record or self-report; OR no description	yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
15637753	Lapinski	2005	secure record	no	nd	nd	low	low	8 (50%) of the subjects were coinfectd with HCV Lamivudine resistance developed in 5 patients—2 were switched to adefovir; 3 were changed to combination lamivudine and adefovir.
24997462	Ow	2014	secure record	no	nd	nd	low	low	

### Evidence Profile: Hepatitis B treatment (lamivudine)

Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
Death, all-cause	1	52	Very serious limitations (-2)	N/A	Direct (0)	Sparse (-1)	Very low	45% (no treatment) vs. 29% (treatment), NS	Critical
Death, hepatic	1	52	Very serious limitations (-2)	N/A	Direct (0)	Sparse (-1)	Very low	3% (no treatment; HCC) vs. 10% (treatment; HCC, spontaneous bacterial peritonitis), NS	High
Viral elimination / suppression	2	37	Very serious limitations (-2)	No important inconsistencies (0)	Direct (0)	Small studies (-1)	Very low	HBV DNA elimination 56% (12 mo), HBeAg elimination 38% (12 mo), complete viral suppression 48% (10 y)	High
<p><b>Overall summary:</b> Lamivudine results in long-term viral elimination in about 50% of patients on HD. Lower death and hepatic-death rate with treatment, but underpowered to show statistical significance.</p>							<p><b>Quality of Overall Evidence:</b> Very low</p>		

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean (SD) or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	CD4+ T cell [mean (SD) or median (range)]	HIV RNA undetectable (%)	Arm (Intervention/Predictor)	Arm description
26765937	Roland	2016	peer-reviewed publication	US	2003-2010	prospective cohort study	45 [39-52]	84	CKD 4-5	ND	465 [313-600] Nadir CD4+ T-cells: 257 [117-428]	100	HIV+ KTxp	kidney transplant in HIV infected patients
													HIV+ No KTx	Transplant candidates did not receive a transplant due to lack of organ availability, no longer meeting study eligibility requirements, being transplanted off-study, dying before an organ became available, inability to adhere to the study requirements, their own decision, or the study reaching its enrollment cap.
													HIV+ KTxp	kidney transplant in HIV infected patients
25807035	Sawinski	2015	peer-reviewed publication	US	1996-2003	retrospective cohort study	HIV- 52 (IQR: 41-61), HIV+ 46 (IQR: 41-56)	60	CKD 4-5	ND	ND	ND	HIV+ KTxp	First kidney transplant in HIV infected patients
													HIV- KTxp	First kidney transplant in HIV uninfected patients
													HIV+ KTxp	First kidney transplant in HIV infected patients
15153575	Abbott	2004	peer-reviewed publication	US	1996-2001	retrospective cohort study	48.2 {10.6}	ND	CKD 4-5	ND	ND	ND	HIV+ KTxp	kidney transplant in HIV infected patients
													HIV- KTxp	kidney transplant in HIV uninfected patients
													HIV+ vs. HIV- KTxp	kidney transplant in HIV infected patients
24621536	Malat	2014	peer-reviewed publication	US	1987-2012	case-control analysis	47.4 {9.4}	7800%	CKD 4-5	ND	ND	ND	HIV+ KTxp	kidney transplant in HIV infected patients
													HIV- KTxp	kidney transplant in HIV uninfected patients
													HIV+ KTxp	kidney transplant in HIV infected patients
	Xia	2014	peer-reviewed publication	US	2000-2013	retrospective observational study	48.1 {8.8}	77	CKD 4-5	ND	ND	ND	HIV+ KTxp	kidney transplant in HIV infected patients; 14.8% were also HCV+
													HIV- KTxp	kidney transplant in HIV or HCV uninfected patients
													HIV+ KTxp	kidney transplant in HIV infected patients; 14.8% were also HCV+
													HIV- KTxp	kidney transplant in HIV or HCV uninfected patients
													HIV+ KTxp	kidney transplant in HIV infected patients; 14.8% were also HCV+
													HIV- KTxp	kidney transplant in HIV or HCV uninfected patients
													HIV+ vs. HIV- KTxp	kidney transplant in HIV infected patients; 14.8% were also HCV+

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean (SD) or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	CD4+ T cell [mean (SD) or median (range)]	HIV RNA undetectable (%)	Arm (Intervention/Predictor)	Arm description
25791727	Locke	2015	peer-reviewed publication	US	2002-2011	registry	nd	79.2	nd	nd	nd	nd	<a href="#">HIV+ KTxp</a> <a href="#">HIV- KTxp</a> <a href="#">HIV+ KTxp</a> <a href="#">HIV+ / HCV- KTxp</a> <a href="#">HIV- / HCV- KTxp</a> <a href="#">HIV+ / HCV- KTxp</a> <a href="#">HIV- / HCV- KTxp</a> <a href="#">HIV+ / HCV+ KTxp</a> <a href="#">HIV- / HCV+ KTxp</a> <a href="#">HIV+ / HCV+ KTxp</a> <a href="#">HIV- / HCV+ KTxp</a> <a href="#">HIV+ KTxp</a> <a href="#">HIV- KTxp</a> <a href="#">HIV+ KTxp</a> <a href="#">HIV- KTxp</a> <a href="#">HIV+ / HCV- KTxp</a> <a href="#">HIV- / HCV- KTxp</a> <a href="#">HIV+ / HCV- KTxp</a> <a href="#">HIV- / HCV- KTxp</a> <a href="#">HIV+ / HCV+ KTxp</a> <a href="#">HIV- / HCV+ KTxp</a> <a href="#">HIV+ / HCV+ KTxp</a> <a href="#">HIV- / HCV+ KTxp</a>	kidney transplant in HIV infected patients matched controls (kidney transplant in HIV uninfected patients) <b>kidney transplant in HIV infected patients</b> <b>matched controls (kidney transplant in HIV uninfected patients)</b> kidney transplant in HIV infected and HCV uninfected patients matched controls (kidney transplant in HIV uninfected and HCV uninfected patients) <b>kidney transplant in HIV infected and HCV uninfected patients</b> <b>matched controls (kidney transplant in HIV uninfected and HCV uninfected patients)</b> kidney transplant in HIV infected and HCV infected patients matched controls (kidney transplant in HIV uninfected and HCV infected patients) <b>kidney transplant in HIV infected and HCV infected patients</b> <b>matched controls (kidney transplant in HIV uninfected and HCV infected patients)</b> kidney transplant in HIV infected patients matched controls (kidney transplant in HIV uninfected patients) <b>kidney transplant in HIV infected patients</b> <b>matched controls (kidney transplant in HIV uninfected patients)</b> kidney transplant in HIV infected and HCV uninfected patients matched controls (kidney transplant in HIV uninfected and HCV uninfected patients) <b>kidney transplant in HIV infected and HCV uninfected patients</b> <b>matched controls (kidney transplant in HIV uninfected and HCV uninfected patients)</b> kidney transplant in HIV infected and HCV infected patients matched controls (kidney transplant in HIV uninfected and HCV infected patients) <b>kidney transplant in HIV infected and HCV infected patients</b> <b>matched controls (kidney transplant in HIV uninfected and HCV infected patients)</b>
27305590	Shelton	2017	peer-reviewed publication	US	2004-2013	registry of re-transplants in HIV+ vs. I47 (37-57)		59.3	CKD 4-5	nd	nd	nd	<a href="#">HIV+ re-KTxp</a>  <a href="#">HIV- re-KTxp</a> <a href="#">HIV+/HCV+ re-KTxp</a> <a href="#">HIV+/HCV- re-KTxp</a> <a href="#">HIV+ re-KTxp</a>  <a href="#">HIV- re-KTxp</a> <a href="#">HIV+/HCV+ re-KTxp</a> <a href="#">HIV+/HCV- re-KTxp</a>	<a href="#">HIV+ retransplantation candidates</a>  <a href="#">HIV- retransplantation candidates</a> <a href="#">HIV/HCV coinfection retransplantation</a> <a href="#">HIV+ retransplantation candidates</a> <a href="#">HIV+ retransplantation candidates</a>  <a href="#">HIV- retransplantation candidates</a> <a href="#">HIV/HCV coinfection retransplantation</a> <a href="#">HIV+ retransplantation candidates</a>

PMID	Author	Year	Outcome	Definition	Outcome		Event rate, %	Relative effect	Variables adjusted in multivariate analysis	P value	Overall Quality	
					Measurement	Sample size (N)						
26765937	Roland	2016	death (risk matched)	ND	Timepoint median 4 years	150	17 (11.3%)	HR 1.172 (0.669, 2.055)	recipient sex, ethnicity, age at transplant, diabetes, hypertension, BMI, hepatitis C antibody, hepatitis B core antibody, hepatitis B surface antigen, CMV antibody status, work status education, and primary method of payment; human leukocyte antigen match, cold ischemic time, time of transplant; donor sex, ethnicity, age, diabetes, hypertension, and cause of death	0.58	A	
							71 (11.8%)					
			graft loss (risk-matched)	ND		600	46 (30.7%)	HR 1.418 (0.997, 2.017)	recipient sex, ethnicity, age at transplant, diabetes, hypertension, BMI, hepatitis C antibody, hepatitis B core antibody, hepatitis B surface antigen, CMV antibody status, work status education, and primary method of payment; human leukocyte antigen match, cold ischemic time, time of transplant; donor sex, ethnicity, age, diabetes, hypertension, and cause of death	0.052		
25807035	Sawinski	2015	death		3 years	600 492	11	aHR 0.90 (0.66, 1.24)	HCV+, age, sex, race, DM, pre-Txp dialysis, dialysis vintage, type of donor, donor HCV+, acute rejection in 1st year, CDC high risk donor, antibody induction use	0.53	A	
						117791	10					
			graft loss		3 years	492	19	aHR 0.60 (0.40, 0.88)	HCV+, age, sex, race, DM, PRA>=30%, pre-Txp dialysis, type of donor, donor HCV+, acute rejection in 1st year, CDC high risk donor, antibody induction use	0.01		
15153575	Abbott	2004	death		2.62 years 2.99 years 5 years	47 27851	4.3 12.8	ND aHR 0.36 (0.05, 2.53)	ND donor and recipient age, race, gender, duration of dialysis before transplantation, donor and recipient HCV status, use of mycophenolate immunosuppression, delayed graft function, and body mass index	ND 0.31	B	
			graft loss	return to dialysis after transplantation and did not include death with a functioning graft	2.62 years 2.99 years	47 27851	2.1 6.8	ND	ND	ND		
24621536	Malat	2014	graft loss		1.92 years	400 1904	26.5 20.1	ND	ND	ND	B	
				Kidney Donor Risk Index as a predictor of graft loss	1.92 years			aHR 1.28 (0.83, 1.98) aHR 2.10 (1.70, 2.61)	ND ND	0.27 <0.001		
24621536	Xia	2014	graft loss	death-censored graft survival	10 years	243	ND	ND	ND	0.0928	A	
						243	ND					
				death-censored graft survival	3 years	243	86.9	ND	ND	ND		
						243	86.4					
				multivariate HR adjusted for age, race, sex, DM, BMI, PRA, prior transplant, insurance, dialysis duration, transplant year, comorbidity, HLA mismatch, and cold ischemia time	3 years			aHR 0.85 (0.48, 1.51)	age, race, sex, DM, BMI, PRA, prior transplant, insurance, dialysis duration, transplant year, comorbidity, HLA mismatch, and cold ischemia time	ND		
			death	survival in months	10 years	243	ND	ND	ND	0.4276		
			243	ND								
	death	3 years	243	85.1	ND	ND	ND	ND				
			243	89.6								
	multivariate HR adjusted for age, race, sex, DM, BMI, PRA, prior transplant, insurance, dialysis duration, transplant year, comorbidity, HLA mismatch, and cold ischemia time	3 years	243	ND	aHR 0.80 (0.39, 1.64)	age, race, sex, DM, BMI, PRA, prior transplant, insurance, dialysis duration, transplant year, comorbidity, HLA mismatch, and cold ischemia time	ND					



PMID	Author	Year	Outcome	Definition	Outcome		Event rate, %	Relative effect	Variables adjusted in multivariate analysis	P value	Overall Quality		
					Measurement	Timepoint							
25791727	Locke	2015	death		5 years	467	16.5	ND	ND	0.06	A	Also data for 1 and 3 years	
						4670	13.8						
					10 years	467	48.4	HR 1.34 (1.08, 1.68)	ND	0.01			
						4670	27.9						
					5 years	362	11.3	ND	ND	0.5			
						3620	10.9						
					10 years	362	36.5	HR 1.26 (0.98, 1.69)	ND	0.13			
						3620	22.4						
					5 years	105	33	ND	ND	<0.01			
					1050	21.4							
			10 years		105	70.7	HR 1.57 (1.11, 2.23)	ND	0.01				
					1050	43.77							
			graft loss		5 years	467	30.8	ND	ND	0.003			
					4670	24.7							
					10 years	467	50.2	HR 1.37 (1.15, 1.64)	ND	<0.001			
					4670	45.6							
					5 years	362	25	ND	ND	0.58			
					3620	24.2							
	10 years	362	44.1	HR 1.06 (0.85, 1.33)	ND	0.61							
	44												
	3620												
	5 years	105	48	ND	ND	0.02							
	36												
	1050												
	10 years	105	73	HR 1.38 (1.08, 1.77)	ND	0.01							
	63.8												
	1050												
27305590	Shelton	2017	graft loss	nd	3 years	22	33.3	HR 1.96 (1.14, 3.36)	recipient age, race, HIV status, HCV status; donor age, race, type; time frame between first graft loss and re-KTxp; and era of re-KTxp (2004-2007 vs. 2008-2013)	0.01	A		
						4127	17.3						
						7	85.7	HR 5.40 (1.3, 21.84)	nd	nd			
						13	15.4						
						22	19.8	HR 3.11 (1.82, 5.34)	recipient age, race, HIV status, HCV status; donor age, race, type; time frame between first graft loss and re-KTxp; and era of re-KTxp (2004-2007 vs. 2008-2013)	<0.01			
						4127	7.9						
			death		nd	3 years	4127	7.9					
							7	42.9	HR 1.21 (0.30, 4.90)	nd		nd	
							13	15.4					

				RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Selection of treated and control cohort?
				<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly unsealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>drawn from the same source; drawn from a different source; OR          no description</p>
26765937	Roland	2016	NA	NA	NA	NA	NA	NA	low
25807035	Sawinski	2015	NA	NA	NA	NA	NA	NA	low
15153575	Abbott	2004	NA	NA	NA	NA	NA	NA	low
24621536	Malat	2014	NA	NA	NA	NA	NA	NA	low, although 5x as many controls were enrolled versus cases.
0	Xia	2014	NA	NA	NA	NA	NA	NA	low
25791727	Locke	2015	NA	NA	NA	NA	NA	NA	low
27305590	Shelton	2017	NA	NA	NA	NA	NA	NA	low

				NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
				yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
26765937	Roland	2016	low		low	low	unclear	low	none
25807035	Sawinski	2015	low		low	unclear	low	low	none
15153575	Abbott	2004	low		low	NA	unclear	low	none
						high, there was significant difference between follow-up times for the cases versus controls.			
24621536	Malat	2014	low		low		unclear	low	none
0	Xia	2014	low		low		unclear	low	none
25791727	Locke	2015	low		low		unclear	low	none
27305590	Shelton	2017	low		low		unclear	low	none

## Evidence Profile: Transplantation outcomes in patients with HIV

Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
<b>Death</b>	6	1421 HIV+ (155282 HIV-)	Serious limitations (-1)*	Important inconsistencies (-1)	Direct (0)	Imprecise estimates (-1)	Very low	Studies inconsistent about risk of death among HIV+ vs. HIV- with HR ranging from 0.36 to 3.11	Critical
<b>Graft loss</b>	7	1821 HIV+ (157186 HIV-)	Serious limitations (-1)*	Important inconsistencies (-2)†	Direct (0)	None	Very low	Studies inconsistent about risk of graft loss among HIV+ vs. HIV- with HR ranging from 0.60 to 1.96	Critical
<b>Overall summary:</b> Unclear whether HIV status associated with post-transplantation death or graft loss.							<b>Quality of Overall Evidence:</b> Very Low		

HIV = human immunodeficiency virus, HR = hazard ratio.

\* It is unknown whether results were based on multivariate analysis (and the covariates). Some studies have relatively short length of follow-up (shorter than three years in two studies for each outcome respectively).

KDIGO - Transplant Candidate  
Guideline Topic: Vaccine Measles  
Categorical outcomes

PMID	Author	Year	Type of article	Country	Era (Study years)	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Arm (Intervention)	Intervention description
19438829	Mori	2009	peer-reviewed journal article	Japan	1990-2002	retrospective	[7.9 {4.8}]	60%	CKD 5	HD	live measles vaccine	

KDIGO - Transplant Candidate  
Guideline Topic: Vaccine Measles  
Categorical outcomes

PMID	Author	Year	Outcome	Definition	Outcome measurement timepoint	Sample size (N)	Frequency (event) rate, %	Relative effect	P value	Overall quality
19438829	Mori	2009	Seroconversion	seroconversion	1 year after transplant 2 years after transplant	19 9	89.5% (17) 100% (9)		nd	C

			RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Representativeness of the case?	NonRCT:.....Selection of the exposed cohort
PMID	Author	Year	<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly un concealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>truly representative; not representative; OR no description</p>	<p>drawn from the same source; not drawn from a different source; OR no description</p>
19438829	Mori	2009	N/A	N/A	N/A	N/A	N/A	truly representative	drawn from the same source

			NonRCT.....Ascertainment of exposure	NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
PMID	Author	Year	secure record or self report; not a secure record or self-report; OR no description	yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
19438829	Mori	2009	secure record	no	N/A	N/A	low	high (only 19/42 evaluated at 1 year and 9/42 at 2 years)	



## Evidence Profile: Pre-transplant vaccination

Vaccine (Outcome)	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
HBV (Post-Txp titers)	2	234	Very serious limitations (-2)	N/A	Direct (0)	Sparse (-1)	Very Low	Higher titers with 3 or 4 pre-Txp doses than with 2, but underpowered so nonsignificant. Vaccination during dialysis prior to transplantation lead to positive responses and great survival post-transplant.	High
Measles (Post-Txp seroconversion)	1	19	Very serious limitations (-2)	N/A	Direct (0)	Sparse, small (-2)	Very low	90% retained seroconversion 1 year after Txp	High
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>		
Pre-transplantation vaccination for HBV and measles is successful to maintain post-transplantation immunity. Three or four HBV doses may be more effective than only two.							Very low		

HBV = hepatitis B vaccination, N/A = not applicable, Txp = transplant

KDIGO - Transplant Candidate  
 Guideline Topic: Cancer Screening  
 Categorical outcomes

PMID	Author	Year	Type of article	Country	Era (Study years)	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Arm (Intervention)	Intervention description
8116110	Yang	1994	peer-reviewed journal	US	1990-1991	prospective cohort study	mean 43 (50-68)	61	CKD 5	HD	renal ultrasonography (RUS) cystoscopic examination digital rectal examination	
9884257	Gulanikar	1998	peer-reviewed journal	US	1995-1997	prospective cohort study	35 {2.4}	62	CKD 5	HD	renal ultrasound	
26069893	Al Ameen	2015	peer-reviewed journal	Saudi Arabia	2008-2014	retrospective cohort study	mean 57.9 (50-74)	61	CKD 5	HD	colonoscopy	
25247014	Therrien	2014	peer-reviewed journal	Canada	2007-2009	retrospective cohort study	55.6 {8.7}	75	CKD 5	HD	colonoscopy	

KDIGO - Transplant Candidate  
 Guideline Topic: Cancer Screening  
 Categorical outcomes

PMID	Author	Year	Outcome	Definition	Sample size (N)	Frequency (event) rate, %	Relative effect	P value	Overall quality
8116110	Yang	1994	Renal cell carcinoma Bladder transitional cell carcinoma Prostate cancer		100	1%	ND	ND	C
					100	1%	ND	ND	
					100	1%	ND	ND	
9884257	Gulanikar	1998	renal cell carcinoma		206	4%	ND	ND	B
26069893	Al Ameel	2015	colorectal cancer	1 polyp	169	15%	ND	ND	B
				2 polyps	169	5%	ND	ND	
				3 polyps	169	2%	ND	ND	
				>4 polyps	169	2%	ND	ND	
25247014	Therrien	2014	colorectal cancer	1 polyp	64	13%	ND	ND	B
				≥2 polyps	64	20%	ND	ND	

PMID	Author	Year	RCT: Adequate generation of a randomized sequence	RCT.....Allocation concealment	RCT.....Blinding of PATIENTS	RCT.....Blinding of PROVIDERS	RCT.....Intention-to-treat-analysis	NonRCT.....Representativeness of the case?	NonRCT.....Selection of the exposed cohort
			<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly un concealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>truly representative; not representative; OR no description</p>	<p>drawn from the same source; not drawn from a different source; OR no description</p>
8116110	Yang	1994	NA	NA	NA	NA	NA	low	low
9884257	Gulanikar	1998	NA	NA	NA	NA	NA	low	unclear
26069893	Al Ameen	2015	NA	NA	NA	NA	NA	low	low
25247014	Therrien	2014	NA	NA	NA	NA	NA	low	low

PMID	Author	Year	NonRCT.....Ascertainment of exposure  secure record or self report; not a secure record or self-report; OR no description	NonRCT.....Demonstration that outcome of interest was not present at start of study  yes; no; unclear	COMPARATIVE.....Baseline differences between groups accounted for  For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	COMPARATIVE...Outcome assessment timing (across interventions)  There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	ALL.....Blinding of OUTCOME ASSESSORS  There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	ALL.....Dropouts/missing data (attrition bias)  There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.  There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
8116110	Yang	1994	low	no, but since this is a screening study the risk of bias is low	NA	NA	low	low	none
9884257	Gulanikar	1998	low	no, but since this is a screening study the risk of bias is low	NA	NA	low	low	ACKD vs non-ACKD reported results
26069893	Al Ameen	2015	low	no, but since this is a screening study the risk of bias is low	NA	NA	low	low	none
25247014	Therrien	2014	low	no, but since this is a screening study the risk of bias is low	NA	NA	low	low	none

## Evidence Profile: Cancer screening in kidney transplant candidates

Screening Test (Outcome)	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
<b>Colonoscopy (Colon cancer)</b>	2	233	Serious limitations (-1)	No important inconsistencies (0)	Indirect (-1)*	Sparse (-1)	Very low	Pretransplant screening by colonoscopy found at least one polyp in 22%-33% of kidney transplant candidates.	High
<b>Ultra-sonography (Renal cell carcinoma)</b>	2	306	Very serious limitations (-2)	No important inconsistencies (0)	Indirect (-1)*	Sparse, old (-2)	Very low	Pretransplant screening by kidney ultrasonography found abnormalities consistent with renal cell carcinoma in 5% of kidney transplant candidates.	High
<b>Cystoscopy (Transitional cell carcinoma)</b>	1	100	Very serious limitations (-2)	N/A	Indirect (-1)*	Sparse, old (-2)	Very low	Pretransplant screening by cystoscopic examination found stage TA transitional cell carcinoma of the bladder in 1% of kidney transplant candidates.	High
<b>Digital Rectal Exam (Prostate cancer)</b>	1	100	Very serious limitations (-2)	N/A	Indirect (-1)*	Sparse, old (-2)	Very low	Pretransplant screening by digital rectal exam found stage A prostate cancer in 1% of kidney transplant candidates.	High
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>		
Screening kidney transplant candidates for cancer found cancer and pre-cancer in a percentage of patients.							Very low		

\* All studies evaluated only incidence of positive screening test results with no clinical outcomes and no outcomes related to transplantation.

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Type of cancer	Arm (Intervention)	Intervention description
9422410	Goldfarb	1997	peer-reviewed publication	US and EU	1974-1996	retrospective cohort study	36 (9.6)	72	CKD 4-5	ND	renal cell carcinoma	removal of localized renal cell carcinoma	patients who had Von Hippel-Lindau disease rendered anephric due to the removal of localized renal cell carcinoma and who subsequently underwent renal transplantation. Thirteen patients underwent bilateral nephrectomy (5 synchronous and 8 asynchronous), whereas 5 patients underwent nephron-sparing surgery followed by remnant nephrectomy for tumor recurrence renal transplant recipients without VHL
												No RCC (or VHL) removal of localized renal cell carcinoma	patients who had Von Hippel-Lindau disease rendered anephric due to the removal of localized renal cell carcinoma and who subsequently underwent renal transplantation. Thirteen patients underwent bilateral nephrectomy (5 synchronous and 8 asynchronous), whereas 5 patients underwent nephron-sparing surgery followed by remnant nephrectomy for tumor recurrence renal transplant recipients without VHL
												No RCC (or VHL) removal of localized renal cell carcinoma	patients who had Von Hippel-Lindau disease rendered anephric due to the removal of localized renal cell carcinoma and who subsequently underwent renal transplantation. Thirteen patients underwent bilateral nephrectomy (5 synchronous and 8 asynchronous), whereas 5 patients underwent nephron-sparing surgery followed by remnant nephrectomy for tumor recurrence renal transplant recipients without VHL
												No RCC (or VHL) removal of localized renal cell carcinoma	patients who had Von Hippel-Lindau disease rendered anephric due to the removal of localized renal cell carcinoma and who subsequently underwent renal transplantation. Thirteen patients underwent bilateral nephrectomy (5 synchronous and 8 asynchronous), whereas 5 patients underwent nephron-sparing surgery followed by remnant nephrectomy for tumor recurrence renal transplant recipients without VHL
												No RCC (or VHL) removal of localized renal cell carcinoma	patients who had Von Hippel-Lindau disease rendered anephric due to the removal of localized renal cell carcinoma and who subsequently underwent renal transplantation. Thirteen patients underwent bilateral nephrectomy (5 synchronous and 8 asynchronous), whereas 5 patients underwent nephron-sparing surgery followed by remnant nephrectomy for tumor recurrence renal transplant recipients without VHL
9869873	Penn	1997	peer-reviewed publication	US	until August 1997	retrospective cohort study	ND	ND	ND	ND	incidental renal carcinoma: 72	treatment of incidental renal cell carcinoma	treatment of renal cell carcinoma pre-transplant, at the time of transplant, or after transplant (n=99)
											carcinoma of the body of the uterus: 26	treatment of carcinoma of the body of the uterus	treatment of carcinoma of the body of the uterus pre-transplant, at the time of transplant, or after transplant (n=99)
											testicular tumors: 43	treatment of testicular tumors	treatment of testicular tumors pre-transplant, at the time of transplant, or after transplant (n=99)
											carcinoma of uterine cervix: 93	treatment of carcinoma of the uterus	treatment of carcinoma of the uterus pre-transplant, at the time of transplant, or after transplant (n=99)

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Type of cancer	Arm (Intervention)	Intervention description
											carcinoma of the thyroid gland: 54	treatment of carcinoma of the thyroid gland	treatment of carcinoma of the thyroid gland pre-transplant, at the time of transplant, or after transplant (n=99)
											lymphomas: 37	treatment of lymphomas	treatment of lymphomas pre-transplant, at the time of transplant, or after transplant (n=99)
											Wilms' tumor: 78	treatment of Wilms' tumors	treatment of Wilms' tumors pre-transplant, at the time of transplant, or after transplant (n=99)
											carcinoma of the prostate gland: 33	treatment of carcinoma of the prostate gland	treatment of carcinoma of the prostate gland pre-transplant, at the time of transplant, or after transplant (n=99)
											colorectal carcinoma: 53	treatment of colorectal cancers	treatment of colorectal cancer pre-transplant, at the time of transplant, or after transplant (n=99)
											melanoma: 29	treatment of skin cancers	treatment of skin cancer pre-transplant, at the time of transplant, or after transplant (n=99)
											carcinoma of the breast: 90	treatment of carcinoma of the breast cancers	treatment of carcinoma of the breast pre-transplant, at the time of transplant, or after transplant (n=99)
											other symptomatic renal carcinoma: 222	treatment of other symptomatic renal carcinoma	treatment of other symptomatic renal carcinoma pre-transplant, at the time of transplant, or after transplant (n=99)
											carcinoma of the urinary bladder: 55	treatment of carcinoma of the urinary bladder	treatment of carcinoma of the urinary bladder pre-transplant, at the time of transplant, or after transplant (n=99)
											sarcomas: 17	treatment of sarcomas	treatment of sarcomas pre-transplant, at the time of transplant, or after transplant (n=99)
											non-melanoma skin cancer: 125	treatment of skin cancer	treatment of skin cancer pre-transplant, at the time of transplant, or after transplant (n=99)



PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Type of cancer	Arm (Intervention)	Intervention description
											myelomas: 12	treatment of myelomas	treatment of myeloma pre-transplant, at the time of transplant, or after transplant (n=99)

KDIGO - Transplant Candidate  
 Guideline Topic: Cancer recurrence risk  
 Categorical outcomes

PMID	Author	Year	Intervention Duration	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Note	Relative effect	P value	Overall Quality	
9422410	Goldfarb	1997	NA	graft survival	ND	1 year	32	(32) 100%		ND	0.52	B	
			NA				32	87.5%		ND			
			NA			5 years	32	62.6%		ND	0.52		
			NA	patient survival	ND	1 year	32	76.1%	(32) 100%		ND		0.37
			NA			5 years	32	96.8%	65.0%		ND		0.37
			NA	death, cancer-related	deaths from metastatic disease	5 years	32	93.0%	(3) 9.3%	ND	ND		
9869873	Penn	1997	ND	cancer recurrence	incidental renal	ND	72	1%	low recurrence rate (1-7%) tumors	ND	ND	C	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	--		ND	ND		
			ND	cancer recurrence	body of uterus	ND	26	4%	low recurrence rate (1-7%) tumors	ND	ND		
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	50%		ND	ND		
			ND	cancer recurrence	testicular	ND	43	5%	low recurrence rate (1-7%) tumors	ND	ND		
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	58%		ND	ND		
			ND	cancer recurrence	cervix if the uterus	ND	93	6%	low recurrence rate (1-7%) tumors	ND	ND		

PMID	Author	Year	Intervention Duration	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Note	Relative effect	P value	Overall Quality
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	54%		ND	ND	
			ND	cancer recurrence	thyroid	ND	54	7%	low recurrence rate (1-7%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	35%		ND	ND	
			ND	cancer recurrence	lymphoma, Hodgkins disease and non-Hodgkins lymphoma	ND	37	11%	intermediate recurrence rate (11-21%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	76%		ND	ND	
			ND	cancer recurrence	Wilms' tumor	ND	78	13%	intermediate recurrence rate (11-21%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	33%		ND	ND	
			ND	cancer recurrence	prostate	ND	33	18%	intermediate recurrence rate (11-21%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	insufficient		ND	ND	
			ND	cancer recurrence	colon	ND	53	21%	intermediate recurrence rate (11-21%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	42%		ND	ND	
			ND	cancer recurrence	melanoma	ND	29	21%	intermediate recurrence rate (11-21%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	41%		ND	ND	
			ND	cancer recurrence	breast	ND	90	23%	high recurrence rate (>23%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	51%		ND	ND	
			ND	cancer recurrence	symptomatic renal carcinomas	ND	222	27%	high recurrence rate (>23%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	22%		ND	ND	
			ND	cancer recurrence	bladder	ND	55	29%	high recurrence rate (>23%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	22%		ND	ND	
			ND	cancer recurrence	sarcomas	ND	17	29%	high recurrence rate (>23%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	insufficient		ND	ND	
			ND	cancer recurrence	nonmelanoma skin carcinomas	ND	125	53%	high recurrence rate (>23%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	11%		ND	ND	

PMID	Author	Year	Intervention Duration	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Note	Relative effect	P value	Overall Quality
			ND	cancer recurrence	myeloma	ND	12	67%	high recurrence rate (>23%) tumors	ND	ND	
			ND		treated >5 years prettransplant, % of recurrent patients	ND	ND	0%		ND	ND	

			RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Selection of treated and control cohort?
			<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly unconcealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>drawn from the same source; drawn from a different source; OR no description</p>
9869873	Penn	1997	NA	NA	NA	NA	NA	NA
8475546	Penn	1993	NA	NA	NA	NA	NA	NA
9422410	Goldfarb	1997	NA	NA	NA	NA	NA	NA

			NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE.....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.	
	PMID	Author	Year	yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
9869873	Penn		1997	low	NA	unclear	unclear	high, no description of methods	
8475546	Penn		1993	low	NA	unclear	low	none	
9422410	Goldfarb		1997	low	unclear	moderate, for patient survival control group had a longer follow-up than disease group	unclear	low	not sure these are truly cancer patients going into transplant

### Evidence Profile: Cancer recurrence risk (pre-transplant cancer treatment)

Outcome (Treated Cancer)*	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
<b>Death (RCC)</b>	1	64	Serious limitations (-1)	N/A	Direct (0)	Sparse, small (-2)	Very low	Similar death rates post-transplantation in patients with VHL treated for RCC as patients without RCC. Cancer-related death in 9% of patients treated for RCC	Critical
<b>Graft loss (RCC)</b>	1	64	Serious limitations (-1)	N/A	Direct (0)	Sparse, small (-2)	Very low	Similar graft loss rates post-transplantation in patients with VHL treated for RCC as patients without RCC.	Critical
<b>Cancer recurrence (multiple)</b>	1	1039	Very serious limitations (-2)	N/A	Direct (0)	Sparse (-1)	Very low	Low recurrence cancers (1-7%): RCC, uterus, testicular, cervix, thyroid. Intermediate recurrence cancers (11-21%): lymphoma, Wilm's tumor, prostate, colorectal, melanoma. High recurrence cancers (>23%): breast, other renal, bladder, sarcoma, non-melanoma skin, myeloma	High
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>		
Patients treated for RCC (with VHL) have similar post-transplant death and graft loss rates as patients without RCC. Cancers have different frequencies of recurrence.							Very low		

RCC = renal cell carcinoma, VHL = Von Hippel-Lindau disease.

\* Treatment pre-transplantation

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Coronary artery disease	Valvular disease	Arm (Intervention)	Intervention description
18154800	Bechtel	2008	peer-reviewed publicatio	Germany	1989-2003	retrospective multicenter study	61 {11}	69.5	CKD 5D	SCr 568 {229} mmol/L	100% (of analyzed)	192 (36.8%)	CABG with subsequent txp	Coronary artery bypass grafting, either with (n=103) or without (n=326) valve surgery.
													CABG without subsequent txp	



PMID	Author	Year	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Relative effect	P value	Overall Quality
18154800	Bechtel	2008	Patient survival	Multivariate analysis of long-term survival with a subsequent renal transplant (after exclusion of all perioperative deaths) - adjusted for emergency surgery, DM, age, number of allogenic transfusions, use of internal thoracic artery graft, sinus rhythm	5 years	17	93.8% (81.9, 100)	Death: HR 0.14 (0.03, 0.58)	0.007	B
					5 years	412	39.4% (34.0, 44.7)			

				RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Selection of treated and control cohort?
				<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly unsealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>drawn from the same source; drawn from a different source; OR no description</p>
18154800	Bechtel	2008	NA		NA	NA	NA		low, however only 17 of the 552 patients received subsequent transplant

				NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE.....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
	PMID	Author	Year	yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
18154800	Bechtel	2008	low		unclear	low	unclear	low, 94.3% completeness of f/up	none

### Evidence Profile: Cardiac revascularization pre-transplantation

Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
<b>Death</b>	2	455	Serious limitations (-1)	No important inconsistencies (0)	Direct (0)	None	Moderate	Small trial found no difference in death rates with or without revascularization, but sparse (1 death in each group). In a retrospective study, dialysis patients who had CABG, those who had subsequent kidney transplantation had better survival than those who didn't; HR = 0.14 (95% CI 0.03, 0.58)	Critical
<b>Myocardial infarction or unstable angina</b>	1	26	Very serious limitations (-2)	N/A	Direct (0)	Sparse (-1)	Very Low	HR = 0.43 (~0.2, 0.90)	
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>		
Patients who have kidney transplant after CABG have higher survival than those who do not receive a transplant							Low		

Abbreviations: CI = confidence interval, HR = hazard ratio.

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	LV function	Arm (Intervention)	Intervention description
11472607	Mitsnefes	2001	peer-reviewed publication	US	1998-2000	prospective observational study	15.4 {5.1} (5.9, 20.8)	57	CKD 5D	GFR 55.0 {21.4} mL/min/1.73m2 (41, 121)	LVEDD: 4.24 ± 0.69 cm LVPW: 0.77 ± 0.24 cm LV SF: 37.10% ± 8.3 LVM: 110.50 ± 55.2 gm LVM index: 43.90 ± 17.8 gm/m2.7 LVH: 12 (53%) LV geometry: concentric LVH: 5 (22%) eccentric LVH: 7 (30%) concentric remodeling: 2 (9%) normal: 9 (39%)	Echocardiography	Each patient had two complete echocardiographic evaluations. The first was performed after the initial diagnosis of ESRD but after at least 6 weeks of chronic dialysis. The second echocardiographic evaluation was performed at least 6 months after successful (i.e. measured GFR at least 40 mL/min/1.73 m2) renal Tx
23542473	Stallworthy	2013	peer-reviewed publication	New Zealand	2000-2009	retrospective observational study	53 (42, 61)	64	CKD 4-5	ND	Subjective LV function: Normal: 613 (86%) Mildly impaired: 57 (8%) Moderately impaired: 30 (4%) Severely impaired: 17 (2%)	Echocardiography	The last echocardiogram before transplantation or the most recent echocardiogram (for individuals not transplanted) was analyzed as representing the most relevant data available to the transplanting physician

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	LV function	Arm (Intervention)	Intervention description
7491692	Parfrey	1995	peer-reviewed publication	Canada	1982-1991	prospective cohort	37 {12}	72	CKD 4-5	ND	Left atrial diameter: 39 ± 6 mm LV end diastolic diameter: 52 ± 7 mm LV end systolic diameter: 34 ± 7 mm Ventricular septal wall thickness in diastole: 12.2 ± 3 Posterior LV wall thickness in diastole: 12.1 (2.5) Fractional shortening: 35% ± 8.5 LV mass index: 152 ± 50 g/m <sup>2</sup> LV volume: 84 ± 35 mL/m <sup>2</sup> Diagnosis: concentric LV hypertrophy: 41 (41%) LV dilation: 32 (32%) systolic dysfunction: 12 (12%) normal echocardiogram: 17 (17%)	Echocardiography	baseline and annual echocardiography were performed using M-mode and two-dimensional ultrasonography.
24009216	Kainz	2013	peer-reviewed publication	Austria	1992-2001	registry study	52 {13}	58	CKD 5D	6.8 {2.8}	LVEDD: 48 {6} mm LVESD: 29 {6} mm LVF (<50%): 4%	Echocardiography	Standard two-dimensional echocardiographic and M-mode pictures were performed by a cardiologist using either a Vivid i or Vivid 7 Cardiovascular Ultrasound System
27841080	Ozkul	2016	peer-reviewed publication	Turkey	2004-2014	retrospective observational	~38 {nd}	68.2	ND	ND	n=162 <55%, n=1601 >=55%	Echocardiography	
26750652	Bang	2016	peer-reviewed publication	South Korea	2006-2013	retrospective observational	44.4 {11.3}	63	ND	eGFR 7 (5-9)	60.4 {6.5}	Echocardiography	preoperative echocardiography, E/e calculated

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	LV function	Arm (Intervention)	Intervention description
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PMID	Author	Year	Predictor	Definition	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Relative effect	P value	Overall Quality				
11472607	Mitsnefes	2001			LV function	left ventricular hypertrophy (LVH)	baseline	23	(12) 52%	ND	ref	A				
							1.9 year posttransplant	23	(13) 56%	ND	NS					
							concentric LVH	baseline	23	(5) 22%	ND		ref			
							1.9 year posttransplant	23	(4) 17%	ND	NS					
							eccentric LVH	baseline	23	(7) 30%	ND		ref			
							1.9 year posttransplant	23	(9) 39%	ND	NS					
							concentric remodeling	baseline	23	(2) 9%	ND		ref			
							1.9 year posttransplant	23	(2) 9%	ND	NS					
							normal	baseline	23	(9) 39%	ND		ref			
							1.9 year posttransplant	23	(8) 35%	ND	NS					
			(all patients)		all-cause death		4.2 years	739	(217) 29%	ND	ND					
23542473	Stallworthy	2013					4.2 years	739	ND	0.84 (0.79, 0.89)	<0.001	A				
							4.2 years	739	ND	1.21 (1.09, 1.36)	<0.001					
							4.2 years	739	ND	1.14 (1.02, 1.28)	0.02					
							4.2 years	739	ND	0.82 (0.72, 0.94)	0.004					
							4.2 years	739	ND	1.14 (0.70, 1.85)	0.61					
							Mild impairment	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA	4.2 years	739	ND		1.36 (0.71, 2.59)	0.35		
							Moderate impairment	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA	4.2 years	739	ND		2.71 (1.36, 5.39)	0.005		
							Severe impairment	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA	4.2 years	739	ND		1.91 (1.28, 2.83)	0.001		
							Pulmonary hypertension/right ventricular dysfunction	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA	4.2 years	739	ND		1.95 (1.32, 2.88)	<0.001		
							Regional wall motion abnormalities	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA	4.2 years	739	ND		1.95 (1.32, 2.88)	<0.001		
							(all patients)			cardiovascular death	4.2 years		739	(98) 13%	ND	ND
							LVEF	per 5% increase, univariate	4.2 years	739	ND		0.83 (0.77, 0.90)	<0.001		
							LVESD	per 5% increase, univariate	4.2 years	739	ND		1.25 (1.07, 1.47)	0.006		
							LVEDD	per 5% increase, univariate	4.2 years	739	ND		1.13 (0.95, 1.34)	0.17		
FS	per 5% increase, univariate	4.2 years	739	ND	0.8 (0.65, 0.99)	0.04										



PMID	Author	Year	Predictor	Definition	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Relative effect	P value	Overall Quality
			Mild impairment	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA			4.2 years	739	ND	1.47 (0.73, 2.95)	0.28	
			Moderate impairment	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA			4.2 years	739	ND	1.26 (0.47, 3.39)	0.65	
			Severe impairment	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA			4.2 years	739	ND	4.60 (1.66, 12.72)	0.003	
			Pulmonary hypertension/right ventricular dysfunction	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA			4.2 years	739	ND	1.45 (0.76, 2.74)	0.26	
			Regional wall motion abnormalities	Per echo reading, adjusted for age, dialysis duration, DM, waitlisting status, subjective LV function, PHT/RVD, RWMA			4.2 years	739	ND	2.30 (1.31, 4.04)	0.004	
7491692	Parfrey	1995			LV function	concentric LV hypertrophy	baseline	102	(41) 41%	ND	ref	
						LV dilation	47 months	102	(37) 37%	ND	NS	A
							baseline	102	(32) 32%	ND	ref	
						systolic dysfunction	47 months	102	(29) 29%	ND	NS	
							baseline	102	(12) 12%	ND	ref	
							47 months	102	(0) 0%	ND	0.001	
						normal echocardiogram	baseline	102	(17) 17%	ND	ref	
							47 months	102	(36) 36%	ND	0.004	
24009216	Kainz	2013	LAD	>53 mm	Death		10 years	287	33.60%	ND	ND	
				≤53mm			10 years	266	16.30%	ND	ND	A
				per mm, adjusted for RVD, PVD, HBG, immunosuppression, calcineurin inhibitor use, atrial fibrillation			10 years	ND	ND	HR 1.06 (.03, 1.08)	<0.001	
				per mm, adjusted for LAD, PVD, HBG, immunosuppression, calcineurin inhibitor use, atrial fibrillation			10 years	ND	ND	HR 0.95 (0.90, 1.01)	0.12	
			RVD		Graft loss		10 years	ND	ND	HR 1.04 (1.02, 1.07)	0.001	
			RAD	per mm, adjusted for HBG, cerebroVD, PVD, age, donor factors, immunosuppression, calcineurin inhibitor use, CHD, year								
26750652	Bang	2016		Early diastolic transmitral flow velocity (E) in combination with early diastolic mitral annular velocity (e')	Graft failure		3.4 years	821	ND			
			E/e' <15	>=15 is indicative of an increase in LV filling pressure			3.4 years	224	ND	OR 1.51 (1.02-2.23)	0.039	
			E/e' >=15		postTxp hemodialysis		3.4 years	821	ND	OR 1.69 (1.05-2.73)	0.032	
			E/e' <15				3.4 years	224	ND			
			E/e' >=15		Mortality, overall		3.4 years	821	ND			
			E/e' <15				3.4 years	224	ND	OR 3.38 (1.78-6.48)	<0.001	
			E/e' >=15				3.4 years	224	ND			
27841080	Ozkul	2016	LVEF <55%		Death		~10 years	162	6.8%	ND		

PMID	Author	Year	Predictor	Definition	Outcome	Definition	Outcome Measurement Timepoint	Sample size (N)	Frequency (Event) Rate, %	Relative effect	P value	Overall Quality
			LVEF >= 55%		<b>Survival time, median</b>		~10 years	1601	2%	ND	<0.001	
			LVEF <55%				114.1 months	162				
			LVEF >= 55%				123.5 months	1601			0.002	

PMID	Author	Year	Type of article	Country	Era	Study design	Age [mean (SD) or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	LV function	Arm (Intervention)
11472607	Mitsnefes	2001	peer-reviewed publication	US	1998-2000	prospective observational study	15.4 (5.1) [5.9, 20.8]	57		GFR 55.0 (21.4) mL/min/1.73m <sup>2</sup> (41, 121)	LVEDD: 4.24 ± 0.69 cm IVS: 0.80 ± 0.18 cm LVPW: 0.77 ± 0.24 cm LV SF: 37.10% ± 8.3 LVM: 110.50 ± 55.2 gm LVM index: 43.90 ± 17.8 gm/m <sup>2.7</sup> LVH: 12 (53%) LV geometry: concentric LVH: 5 (22%) eccentric LVH: 7 (30%) concentric remodeling: 2 (9%) normal: 9 (39%)	Echocardiography
23542473	Stallworthy	2013	peer-reviewed publication	New Zealand	2000-2009	retrospective observational study	53 (42, 61)	64	CKD 4-5	ND	Subjective LV function: Normal: 613 (86%) Mildly impaired: 57 (8%) Moderately impaired: 30 (4%) Severely impaired: 17 (2%)	Echocardiography
7491692	Parfrey	1995	peer-reviewed publication	Canada	1982-1991	prospective cohort	37 (12)	72	CKD 4-5	ND	Left atrial diameter: 39 ± 6 mm LV end diastolic diameter: 52 ± 7 mm LV end systolic diameter: 34 ± 7 mm Ventricular septal wall thickness in diastole: 12.2 ± 3 Posterior LV wall thickness in diastole: 12.1 (2.5) Fractional shortening: 35% ± 8.5 LV mass index: 152 ± 50 g/m <sup>2</sup> LV volume: 84 ± 35 mL/m <sup>2</sup> Diagnosis: concentric LV hypertrophy: 41 (41%) LV dilation: 32 (32%) systolic dysfunction: 12 (12%) normal echocardiogram: 17 (17%)	Echocardiography



				RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Selection of treated and control cohort?
				<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly unsealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>drawn from the same source; drawn from a different source; OR no description</p>
11472607	Mitsnefes	2001	NA	NA	NA	NA	NA	NA	low
23542473	Stallworthy	2013	NA	NA	NA	NA	NA	NA	low
7491692	Parfrey	1995	NA	NA	NA	NA	NA	NA	low
24009216	Kainz	2013	NA	NA	NA	NA	NA	NA	low

			NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.	
	PMID	Author	Year	yes; no; unclear	<p>For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).</p>	<p>There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias</p>	<p>There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.; or:          &gt;&gt; for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. &gt;&gt; for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. &gt;&gt; for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.</p>	<p>There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.</p>	<p>There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere</p>
	11472607	Mitsnefes	2001	low	NA	low	unclear	low	none
	23542473	Stallworthy	2013	low	NA	low	unclear	low	none
	7491692	Parfrey	1995	low	NA	low	unclear	low	none
	24009216	Kainz	2013	low	NA	n	unclear	low	none

## Evidence Profile: Pre-transplantation echocardiography

Outcome	Predictor	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
								Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
<b>Death*</b>	<b>Echo parameters</b>	4	4100	No limitations (0)	No important inconsistencies (0)	Direct (0)	Sparse, per parameter (-2)	Low	Pre-Txp echo parameters predict post-Txp death: lower LVEF and FS, higher LVESD, LVEDD, LAD (e.g., >53 mm)	Critical
	<b>Impairment</b>	1	739	No limitations (0)	N/A	Indirect† (-1)	Sparse (-2)	Very low	Severe impairment is a significant predictor of post-Txp death	
	<b>Pulm HTN</b>	1	739	No limitations (0)	N/A	Direct (0)	Sparse (-2)	Low	PTH on pre-Txp echo doubles risk of post-Txp death	
	<b>RWMA</b>	1	739	No limitations (0)	N/A	Direct (0)	Sparse (-2)	Low	PTH on pre-Txp echo doubles risk of post-Txp death	
<b>Graft loss</b>	<b>Echo parameters</b>	2	1598	No limitations (0)	No important inconsistencies (0)	Direct (0)	Sparse (-2)	Low	Higher RAD associated with higher risk of graft loss, E/e' >=15 associated with higher risk of graft loss	Critical
<b>LV function</b>	<b>Echo</b>	2	125	No limitations (0)	No important inconsistencies (0)	Indirect‡ (-1)	Small sample (-1)	Low	Prevalence of LVH (and subtypes) remains stable pre-Txp vs. 2 & 4 years post-Txp. Syst dysfxn fully resolves by 4 years post-Txp. Prevalence of normal echo doubles by 4 years post-Txp.	High
<b>Overall summary:</b>								<b>Quality of Overall Evidence:</b>		
Pre-transplant echo parameters and findings are associated with post-transplantation death and graft loss.								Low		

Echo = echocardiography, E/e' = Early diastolic transmitral flow velocity (E) in combination with early diastolic mitral annular velocity (e'), FS = fractional shortening, LAD = left atrial diameter, LVEDD = left ventricular end diastolic diameter, LVEF = left ventricular ejection fraction, LVESD = left ventricular end systolic diameter, Pulm HTN = pulmonary hypertension, RAD = right atrial diameter, RWMA = regional wall motion abnormalities, Syst dysfxn = systolic dysfunction, Txp = kidney transplant.

\* Overall similar findings for cardiovascular death from 1 study (N=739); LVEF <55% associated with shorter survival time (P=0.002) from 1 study (N=1763).

† Impairment defined variably by sonographers.

‡ Only comparisons of prevalence of LV function pre- and post-Txp.

PMID	Author	Year	Type of article	Country	Era (Study years)	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Intervention description	Arm (Intervention)
18045824	Aull-Watschinger, S. and Konstantin, H. and Demetriou, D. and Schillinger, M. and Habicht, A. and Horl, W. H. and Watschinger, B.	2008	peer-reviewed	Austria	1995-2005	retrospective single-center	>18	66%	ESRD	nd	Carotid duplex ultrasound	<a href="#">plaques</a>  <a href="#">stenosis 25-50%</a> <a href="#">stenosis 51-70%</a> <a href="#">stenosis &gt;70%</a>



PMID	Author	Year	Outcome	Definition	Outcome measurement timepoint	Sample size (N)	Frequency (event) rate, %	Relative effect	P value	Overall quality
18045824	Aull-Watschinger, S. and Konstantin, H. and Demetriou, D. and Schillinger, M. and Habicht, A. and Horl, W. H. and Watschinger, B.	2008	TIA/Stroke		4 y post-Txp (median)	809	4.9% (40)	Reference (no stenosis)		B
			TIA/Stroke			44	18.2% (8)	HR: 1.68 (0.59, 4.78)		
			TIA/Stroke			50	4.0% (2)	HR: 1.54 (0.47, 2.76)		
			TIA/Stroke			9	11.1% (1)	HR: 1.71 (0.20, 15.06)		



			NonRCT.....Demonstration that outcome of interest was not present at start of study	COMPARATIVE.....Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL.....Blinding of OUTCOME ASSESSORS	ALL.....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
PMID	Author	Year	yes; no; unclear	For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
18045824	Aull-Watschinger, S. and Konstantin, H. and Demetriou, D. and Schillinger, M. and Habicht, A. and Hori, W. H. and Watschinger, B.	2008	yes	na	na	unclear	there are 10 missing patients (sample size is 922 but table numbers add to 912)	low

### Evidence Profile: Carotid artery testing

Intervention	Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
								Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
Carotid duplex ultrasound	TIA or Stroke	1	912	Serious limitation (-1)	N/A	Direct (0)	Imprecise, sparse (-2)	Very low	Pre-transplant carotid stenosis not associated with post-transplant events	Critical
<b>Overall summary:</b>								<b>Quality of Overall Evidence:</b>		
Imprecise evidence that pre-transplantation carotid stenosis is not associated with post-transplantation stroke or TIA								Very low		

N/A = not applicable, TIA = transient ischemic attack.

PMID	Author	Year	Type of article	Country	Era	Study design	Sample size (N)	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	% Hypertension	Arm (Intervention)	Intervention description
23449651	Niemczyk	2013	peer-reviewed publicatio	Poland	2009-2012	prospective cohort study	83 ADPKD	46 {15}	38.6	1: 27.7%, 2: 24.1%, 3: 30.1%, 4: 16.9%, 5: 1.2%	ND	arterial hypertension: 96.4%	MRA study for intracranial aneurysms, confirmed by CTA	MR results were verified by use of CT angiography and were then referred to a specialist in neurosurgery.
11981069	Graf	2002	peer-reviewed publicatio	Germany	ND	prospective cohort study	43 ADPKD	45.7 (12.9)	48.8	ND	normal: 37.2%, impaired: 34.9%, ESRD: 25.6%		MRA study for intracranial aneurysms	MRA performed using 3D phase-contrast imagine sequences and 2D inflow image sequence.
	Wakabayashi	1983	peer-reviewed publicatio	Japan	1981-1982	prospective cohort study	17 ADPKD	mean 42 (32-66)	41.2	ND	ND	52.9	Angiography for intracranial aneurysm	four vessel angiography
15086900	Gibbs	2004	peer-reviewed publicatio	US	1989-2002	retrospective cohort study	21 (ADPKD, known unruptured aneurysm)	47.9 (calculated)	33.30%	ND	ND	ND	MR angiographic screening	three-dimensional time-of-flight MR angiography

KDIGO - Transplant Candidate  
 Guideline Topic: ADPKD-related cerebral aneurysm  
 Categorical outcomes

PMID	Author	Year	Outcome	Outcome Measurement Timepoint	Definition	Subgroup	Sample size (N)	Frequency (Event) Rate, %	Relative effect	P value	Overall Quality
23449651	Niemczyk	2013	cerebral aneurysms	ND	Any aneurysm	All	83	(14) 16.9%			B
						Newly diagnosed	34	(1) 2.9%	ND	<0.05	
11981069	Graf	2002	death	ND	Newly diagnosed death due to subcranial hemorrhage	<=45 years old (post hoc threshold)	49	(11) 22.4%			
						>45 years old	6	(2) 33.3%	ND	ND	B
	Wakabayashi	1983	cerebral aneurysms	ND	cerebral aneurysms	Dolichoectasia	2	0%			
						Normal	35	0%			
	Gibbs	2004	Aneurysm growth	81 (13-160) mo post-first eval	Aneurysm rupture	Family hx of stroke	32	(3) 9.4%	ND	ND	
						Family hx of ICA or intracranial bleed	11	(3) 27.2%			
15086900	Gibbs	2004	Aneurysm growth	81 (13-160) mo post-first eval	Aneurysm rupture	All	17	(7) 41.2%			B
						Hypertension	9	(2) 22.2%	ND	ND	
	Gibbs	2004	Aneurysm growth	81 (13-160) mo post-first eval	Aneurysm rupture	No hypertension	8	(5) 62.5%			
						Follow-up study	18	1 (5.6%)			B
	Gibbs	2004	Aneurysm growth	81 (13-160) mo post-first eval	Aneurysm rupture	Follow-up study	18	1 (5.6%)			
						All	21	0%			

PMID	Author	Year	RCT: Adequate generation of a randomized sequence  There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.	RCT:.....Allocation concealment  There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly unconcealed procedures.	RCT:.....Blinding of PATIENTS  There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.	RCT:.....Blinding of PROVIDERS  There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.	RCT:.....Intention-to-treat-analysis  There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).	NonRCT:.....Selection of treated and control cohort?  drawn from the same source; drawn from a different source; OR no description	NonRCT:.....Demonstration that outcome of interest was not present at start of study  yes; no; unclear
23449651	Neimczyk	2013	NA	NA	NA	NA	NA	NA	low, although aneurysms were suspected
11981069	Graf	2002	NA	NA	NA	NA	NA	NA	low, although aneurysms were suspected
0	Wakabayashi	1983	NA	NA	NA	NA	NA	NA	NA, no follow-up (screening)
15086900	Gibbs	2004	NA	NA	NA	NA	n		low, although aneurysms were suspected

PMID	Author	Year	COMPARATIVE...Baseline differences between groups accounted for	COMPARATIVE...Outcome assessment timing (across interventions)	ALL....Blinding of OUTCOME ASSESSORS	ALL....Dropouts/missing data (attrition bias)	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.
			<p>For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).</p>	<p>There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias</p>	<p>There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; or &gt;&gt; for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. &gt;&gt; for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. &gt;&gt; for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.</p>	<p>There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.</p>	<p>There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere</p>
23449651	Neimczyk	2013	NA	NA	unclear	low	none
11981069	Graf	2002	NA	NA	unclear	low	none
0	Wakabayashi	1983	NA	NA	unclear	low	none
15086900	Gibbs	2004	NA	NA	unclear	low	none



## Evidence Profile: Intracranial imaging in patients with ADPKD

Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings			
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome	
<b>Death</b>	1	43	Serious limitations (-1)	N/A	Direct (0)	Sparse, small (-2)	Very low	2/43 (4.7%) died of subcranial hemorrhage; both among 6 patients found to have aneurysm; no deaths among other 37 patients	Critical	
<b>Aneurysm rupture</b>	1	21	Serious limitations (-1)	N/A	Direct (0)	Sparse, small (-2)	Very low	0% after a mean of 7.7 years since aneurysm found.	Critical	
<b>Aneurysm</b>	3	143	Serious limitations (-1)	No important inconsistencies (0)	Direct (0)	Small studies, intermediate outcome (-1)	Low	Approximately 20-40% ADPKD patients found to have aneurysms. One study found only 1 small newly diagnosed aneurysm among 34 patients ≤45 years old compared to 22% of 49 older patients. One study each found higher prevalence in patients with family history of ICA or bleed than those with family history of stroke, and in patients without hypertension than with (both NS).	High	
<b>Change in aneurysm</b>	1	18	Serious limitations (-1)	N/A	Direct (0)	Sparse, small (-2)	Very low	Among 18 ADPKD patients found to have aneurysm, only 1 each had aneurysm growth or new aneurysms over a mean of 7 years	High	
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>			
Evidence does not directly address whether ADPKD patients benefit from intracranial testing for aneurysms. Data on death from intracranial bleeding and on rate of aneurysm rupture are inconsistent. Possibly patients ≤45 years old are very unlikely to have aneurysm. Some evidence that aneurysms rarely change over time,							Very low			

KDIGO - Transplant Candidate  
Guideline Topic: Thrombophilia  
Categorical outcomes

PMID	Author	Year	Type of article	Country	Era (Study years)	Study design	Age [mean (SD) or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Testing/Subgroup	Intervention	Outcome
10798752	Vaidya	2000	peer-reviewed journal article	USA	1995-1998	unclear	nd	55%	CKD 5	HD	APAS screening		ACA elevated APAS diagnosis
											APAS & KTx	Pre-Txp anticoagulation Peri-Txp anticoagulation No anticoagulation	Graft loss, 1 year Graft loss, 1 year Graft loss <1 week
											High ACA (not APAS) & KTx Normal ACA, no APAS & KTx		Graft loss Graft loss
15476477	Forman	2004	peer-reviewed journal article	USA	1996-2001	retrospective	[44.9 {2.1}]	61%	CKD 5	HD	ACA Screening ACA positive ACA negative ACA pos vs. neg		ACA elevated delayed graft function
											APAS	Peri-Txp anticoagulation No anticoagulation	graft loss Graft loss, 1 month
22507396	Vaidya	2012	peer-reviewed journal article	USA	1992-2009	unclear	nd	52%	CKD 5	HD	APAS Screening		APAS diagnosis
											APAS & KTx	LMWH post-Txp No anticoagulation	Graft loss, 1 year ACA IgG or IgM or both Graft loss, 10 year
11502996	Wuthrich	2001	peer-reviewed journal article	Switzerland	1996-1999	unclear	nd	nd	CKD 5	HD	Factor V Leiden FVL mutation & KTx		FVL mutation graft loss
19845577	Ghisdal	2010	peer-reviewed journal article	Belgium	2001-2006	prospective	[47.8 {0.2}]	66.5%	CKD 5	HD	Testing on day of transplant		Antithrombin Protein C deficiency Protein S deficiency APC resistance Factor VIIIc Factor IX Lupus anticoagulant Antiphospholipid antibodies PT (G20210A) variant GPIIa (T1565C) variant FV (G1691A) variant Graft survival, 4 years
											>=1 thrombophilic factor No thrombophilic factors >=1 thrombophilic factor No thrombophilic factors		Patient survival, 4 years
17032424	Kranz	2006	peer-reviewed journal article	Germany	1998-2003	prospective	[10.1 {1.5}]	33%	CKD 4-5	PD/HD	Thrombophilia testing		Thrombophilic risk factors C667T mutation of the MTHFR gene

PMID	Author	Year	Type of article	Country	Era (Study years)	Study design	Age [mean {SD} or median (range)]	% Male	Baseline CKD stage	Baseline kidney function	Testing/Subgroup	Intervention	Outcome
											>=1 thrombophilic factor No thrombophilic factors		<b>factor V Leiden mutation (FV506Q)</b> <b>antiphospholipid antibodies (anti cardiolipin antibodies, lupus anticoagulant)</b> <b>prothrombin mutation (G20210A)</b> <b>protein C deficiency</b> Graft loss, 3.3 y

PMID	Author	Year	Definition	Sample size (N)	Frequency (event) rate, %	Relative effect	P value	Overall quality
10798752	Vaidya	2000	IgG >10 units, IgM >15 units, IgA >7 units documented lupus, frequent abortions, AV shunt thrombosis, thrombocytopenia, cerebrovascular thrombosis, microrenal angiopathy	502	19% (93)	nd	nd	B
				502	4.6% (23)			
				2	0%			
				2	50% (1 at day 5)			
				7	100% (7)			
				37	27% (10), none due to thrombosis			
				207	86%, none due to thrombosis			
15476477	Forman	2004		337	18% (61)	1.65 (0.69, 3.97), adjusted for post-Txp coumadin	0.53	B
				60	10% (60)			
				274	14% (38)			
				337				
				8	0%			
				1	100% (1), at day 4			
				337				
22507396	Vaidya	2012	patients were required to have a history of clotting disorders of one or more of the following: (i) biopsy-established micro-renal angiopathy, (ii) more than six A-V shunt thromboses, (iii) a history of lupus, (iv) frequent spontaneous abortions, and (v) thrombocytopenia.	1625	2.4% (39)		NS vs. cadaveric (ACA/APAS neg, P=0.051); "Lower" vs. living donor (ACA/APAS neg, P=0.0036)	C
				10	20% (2)			
				11	27% (3)			
				1625	5.8% (94)			
				46	72%			
11502996	Wuthrich	2001		202	4.0% (8)			B
				8	25% (2)			
19845577	Ghisdal	2010		309	14.2%			B
				301	13.0%			
				302	5.3%			
				310	2.6%			
				309	20.4%			
				214	1.4%			
				304	38.2%			
				286	26.9%			
				291	2.4%			
				289	29.8%			
				291	2.4%			
				250	81.2%			
				60	83.7%			
				250	91.7%			
				60	95.9%			
17032424	Kranz	2006		66 children	27.3%			B
				66 children	10.6%			

PMID	Author	Year	Definition	Sample size (N)	Frequency (event) rate, %	Relative effect	P value	Overall quality
				66 children	7.6%			
				66 children	4.5%			
				66 children	1.5%			
				66 children	1.5%			
				18 children	5.6% (1, from de novo GN)		NS	
				48 children	4.2% (2, from chronic rejection, recurrence of oxalosis)			

			RCT: Adequate generation of a randomized sequence	RCT:.....Allocation concealment	RCT:.....Blinding of PATIENTS	RCT:.....Blinding of PROVIDERS	RCT:.....Intention-to-treat-analysis	NonRCT:.....Representativeness of the case?	NonRCT:.....Selection of the exposed cohort	
	PMID	Author	Year	<p>There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimization (minimization may be implemented without a random element, and this is considered to be equivalent to being random). There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number; or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.</p>	<p>There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomization); sequentially numbered drug containers of identical appearance; or sequentially numbered, opaque, sealed envelopes. There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or other explicitly un concealed procedures.</p>	<p>There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.</p>	<p>There is low risk of bias if all randomized patients were reported/analyzed in the group to which they were allocated by randomization. I.e., no dropouts or they state analyzed as ITT (unless there's an obvious problem).</p>	<p>truly representative; not representative; OR no description</p>	<p>drawn from the same source; not drawn from a different source; OR no description</p>
	11502996	Wuthrich	2001	N/A	N/A	N/A	N/A	N/A	truly representative	drawn from the same source
	19845577	Ghisdal	2010	N/A	N/A	N/A	N/A	N/A	truly representative	drawn from the same source
	15476477	Forman	2004	N/A	N/A	N/A	N/A	N/A	truly representative	drawn from the same source
	22507396	Vaidya	2012	N/A	N/A	N/A	N/A	N/A	no description	drawn from the same source

PMID	Author	Year	NonRCT.....Ascertainment of exposure  secure record or self report; not a secure record or self-report; OR no description	NonRCT.....Demonstration that outcome of interest was not present at start of study  yes; no; unclear	COMPARATIVE....Baseline differences between groups accounted for  For RCT, LOW RoB unless there are important baseline differences that are not adjusted for. For nRCS, HIGH RoB if unadjusted or adjusted only for age and sex; LOW RoB if multivariate adjustment (more than age/sex) or propensity score analysis. There is low risk of selection bias if groups are similar at baseline for demographic factors, value of main outcome measure(s), and important prognostic factors (examples in the field of back and neck pain are duration and severity of complaints, vocational status, percentage of patients with neurological symptoms).	COMPARATIVE...Outcome assessment timing (across interventions)  There is low risk of detection bias if outcome assessments for all intervention groups were measured at the same time. If they report results at mean follow-up times, then HIGH risk of bias	ALL.....Blinding of OUTCOME ASSESSORS  There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; or: >> for patient-reported outcomes in which the patient was the outcome assessor (e.g., pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding. >> for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between patients and care providers (e.g., co-interventions, length of hospitalization, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers. >> for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.	ALL.....Dropouts/missing data (attrition bias)  There is a low risk of attrition bias if there were no missing outcome data. The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias.	Additional Bias: Bias due to problems not covered elsewhere in the table. If yes, describe them in the Notes.  There is a low risk of bias if the study appears to be free of other sources of bias not addressed elsewhere
11502996	Wuthrich	2001	secure record	no	N/A	N/A	low	low	
19845577	Ghisdal	2010	secure record	no	N/A	N/A	low	low	
15476477	Forman	2004	secure record	no	N/A	N/A	low	low	
22507396	Vaidya	2012	secure record	no	N/A	N/A	low	low	

## Evidence Profile: Thrombophilia testing

Outcome	# of Studies	Total N of Patients	Methodological Quality of Studies	Consistency Across Studies	Directness of the Evidence, including Applicability	Other Considerations	Summary of Findings		
							Quality of Evidence for Outcome	Description of Findings	Importance of Outcome
<b>Death, ≥1 thrombophilia factor</b>	1	310	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	Thrombophilia factors not a predictor of post-transplant death	Critical
<b>Graft loss, APAS +</b>	3	41	Serious limitations (-1)	No important inconsistencies (0)	Direct (0)		Moderate	Pre-transplant anticoagulation mostly prevents acute graft loss	Critical
ACA +	3	420	Serious limitations (-1)	No important inconsistencies (0)	Direct (0)		Moderate	Not a significant predictor of graft loss	
FVL mutation	1	8	Serious limitations (-1)	N/A	Direct (0)	Sparse, small sample (-2)	Very low	25% graft loss	
≥1 thrombophilia factor	2	376 (66 children)	Serious limitations (-1)	No important inconsistencies (0)	Direct (0)		Moderate	Not a significant predictor of graft loss	
<b>Prevalence, Anticardiolipin Ab</b>	3	2464	Serious limitations (-1)	Important inconsistencies (-1)	Direct (0)		Low	6-19%	
Antiphospholipid Ab adults	1	286	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	27%	
children	1	66	Serious limitations (-1)	N/A	Direct (0)	Sparse, small sample (-1)	Very low	4.5%	
Antiphospholipid Ab syndrome	2	2127	Serious limitations (-1)	No important inconsistencies (0)	Direct (0)		Moderate	2.4-4.6%	
Antithrombin	1	309	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	14%	
APC resistance	1	310	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	2.6%	
Factor IX	1	214	Serious limitations (-1)	N/A	Direct (0)	Spars (-1)	Low	1.4%	



FVL variant (FV506Q) children	1	66	Serious limitations (-1)	N/A	Direct (0)	Sparse, small sample (-1)	Very low	7.6%
FVL variant (G1691A)	1	291	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	2.4%
Factor VIIIc	1	309	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	20%
GP1IIa variant (T1565C)	1	289	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	30%
Lupus anticoagulant	1	308	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	38%
MTHFR variant (C667T), children	1	66	Serious limitations (-1)	N/A	Direct (0)	Sparse, small sample (-1)	Very low	11%
Protein C deficiency, adults	1	301	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	13%
children	1	66	Serious limitations (-1)	N/A	Direct (0)	Sparse, small sample (-1)	Very low	1.5%
Protein S deficiency	1	302	Serious limitations (-1)	N/A	Direct (0)	Sparse (-1)	Low	5.3%
Prothrombin variant (G20210A)	2	357 (66 children)	Serious limitations (-1)	No important inconsistencies (0)	Direct (0)	Sparse (-1)	Moderate	1.5-2.4%
<b>Overall summary:</b>							<b>Quality of Overall Evidence:</b>	
Antithrombotic factors are not predictors of post-transplantation death or graft loss. Except that patients with APAS who do not receive pre-transplantation anti-coagulation are at high risk of graft loss.							Low to moderate	

**Summary Table: MGUS**

Study Country Year	Sample	N	Pre-Txp MGUS, %	Post-Txp MGUS, n (%) [type]	Hematologic outcomes (post-Txp)	Other
Bancu, 2014 (Conf abstr) Spain 1996-2011	KTx, all	587 <sup>1</sup>	9 (1.5%)	8 (1.4%) [de novo]	Pre-Txp MGUS: MM 1/9 (11%) Post-Txp MGUS: MM 0/8 (1/8 MGUS resolved) [6 y median f/up]	
Cuéllar-García 2015 (25645776) Spain 1992-2012	KTx, all	1016 <sup>2</sup>	5 (4.9%)	11 (10.8%) [de novo, probably]	Pre-Txp MGUS: PTLD 1/5 (20%) Post-Txp MGUS: MALTL 1/11 (9.1%) All: MM 0/16 [30 mo median f/up]	
Fenoglio 2013 (Conf abstr) Italy 1998-2012	KTx, all	851 <sup>3</sup>	16 (1.9%)	26 (3.2%) [de novo]	Pre-Txp MGUS: MM 1/16 (6.3%) Post-Txp MGUS: MM 1/26 (3.8%) [4.1 y median f/up]	
Gagnon 2017 (Conf abstr) Canada 2000-2016	KTx, SPEP available	755 <sup>4</sup>	13 (1.7%)	43 (5.8% <sup>5</sup> ) [de novo]	Pre-Txp MGUS: LCDD 2/13 (15.4%) Pre-Txp MGUS: SMM 2/13 (15.4%) Post-Txp MGUS: LCDD 1/43 (2.3%) Post-Txp MGUS: MM 1/43 (2.3%) <sup>6</sup> [7.5 y median f/up]	None of 7 cases of PTLD identified in study was preceded by MGUS
Goebel 2015 (26194021) US 2005-2011	KTx, all	14,076 <sup>7</sup>	45 (0.3%)		Pre-Txp MGUS: PTLD 0/45 (0%) Pre-Txp MGUS: MM "<10"/45 (<22%) Pre-Txp MGUS: lymphoma 0/45 (0%)	NR <sup>8</sup>
Heymans 2016 (Conf abstr) Belgium 2015	KTx, with SPEP data	304 <sup>9</sup>	6 (2.0%)	44 (14.8%) [de novo]	Pre-Txp MGUS: PTLD 1/6 (16.7%) Post-Txp MGUS: PTLD 6/44 (13.6%) [f/up NR]	

<sup>1</sup> Of 587 patients with kidney transplants (100%).

<sup>2</sup> Of 1016 patients with kidney transplants (100%).

<sup>3</sup> Of 851 patients with kidney transplants (100%).

<sup>4</sup> Of 1009 patients with kidney transplants (75%).

<sup>5</sup> 5 year incidence rate of 2.7%

<sup>6</sup> None of these 6 patients with post-transplant malignancy had systematic hematologic workup prior to transplantation to rule out these conditions.

<sup>7</sup> Of 14,076 patients with kidney transplants (100%).

<sup>8</sup> Presented post-MGUS outcomes for all solid organ transplantation, no kidney-specific data

<sup>9</sup> Of unreported number with kidney transplants.

Study Country Year	Sample	N	Pre-Txp MGUS, %	Post-Txp MGUS, n (%) [type]	Hematologic outcomes (post-Txp)	Other
Jimenez-Zepeda (21712755) US 1999-2009	KTx, all	823 <sup>10</sup>	14 (1.7%)		Pre-Txp MGUS: PTLD 0/14 [7 y median f/up]	None of ≤6 cases <sup>11</sup> of PTLD identified in study was preceded by MGUS
Kaur 2017 (Conf abstr) US 2001-2015	KTx, all	2890 <sup>12</sup>	23 (0.8%)		Pre-Txp MGUS: Proximal tubulopathy 1/23 (4.3%) Pre-Txp MGUS: PTLD 2/23 (8.7%) [f/up NR]	
Naina 2012 (22473253) US 1963-2006	KTx, with SPEP data pre-Txp, adult	3491 <sup>13</sup>	23 (0.7%)	19 (0.5%) [de novo]	Pre-Txp MGUS: SMM 2/23 (8.7%) Pre-Txp MGUS: PTLD 2/23 (8.7%) Post-Txp MGUS: MM or SMM 0/19 Post-Txp MGUS: PTLD 2/19 (10.5%) [8.5 y median f/up]	
Rostaing 1994 (7977478) France 1984~1994	KTx, all	502 <sup>14</sup>	4 (0.8%)		Pre-Txp MGUS: SMM 2/5 (40%) <sup>15</sup> [3-8 y f/up]	
Soltero 2012 (22044717) US 2000-2007	Evaluated for KTx, ≥50 y, SPEP available	336 <sup>16</sup>	31 (9.2%)			
Younes 2013 (Conf abstr) NR 2000-2010	KTx w/MGUS pre-Txp	31 <sup>17</sup>	NR		Pre-Txp MGUS: MM 0/31 [45.6 mo median f/up]	

KTx = kidney transplant recipient,  
Txp = transplantation,  
MG = monoclonal gammopathy,

<sup>10</sup> Of 823 patients with kidney transplants (100%).

<sup>11</sup> Among 1199 with kidney (69%), liver (31%), or pancreas (0.7%) transplant

<sup>12</sup> Of 2890 patients with kidney transplants (100%).

<sup>13</sup> Of 3518 patients with kidney transplants (99%).

<sup>14</sup> Of 502 patients with kidney transplants (100%).

<sup>15</sup> Among 4 patients with kidney transplant and 1 patient with heart transplant. Unclear phrasing (italics added): “In 2 patients, MGUS had *probably* progressed to smoldering myeloma (stage I of Durie and Salmon).”

<sup>16</sup> Of 675 patients with kidney transplants ≥50 years old (49.8%).

<sup>17</sup> Of unknown number of patients with kidney transplants.

MGUS = monoclonal gammopathy of undetermined significance  
MPGN = membranoproliferative glomerulonephritis  
MIDD = monoclonal immunoglobulin deposition disease  
Ig = immunoglobulin,  
C3GN = C3 glomerulonephritis  
c/t = compared to,  
NS = nonsignificant,  
MBCL = Monoclonal B cell lymphocytosis,  
MM = multiple myeloma  
LPL = lymphoplasmacytic lymphoma  
HLA = human leukocyte antigen,  
PTLD = post-transplantation lymphoproliferative disorder,  
MALT = mucosa-associated lymphoid tissue lymphoma,  
LCDD = light chain deposition disease,  
SMM = smoldering multiple myeloma  
TMA = thrombotic microangiopathy  
Conf Abst = conference abstract,  
NR = not reported,  
f/up = follow-up (since MGUS diagnosis)

**Summary Table: MGUS, continued**

Study	N	Kidney outcomes	Survival
Bancu, 2014 (Conf abstr)		NR	NR
Cuéllar-García 2015 (25645776)		NR	NR
Fenoglio 2013 (Conf abstr)		NR	NR
Gagnon 2017 (Conf abstr)		NR	NR
Goebel 2015 (26194021)		NR	NR
Heymans 2016 (Conf abstr)		NR	NR
Jimenez-Zepeda (21712755)		NR	NR
Kaur 2017 (Conf abstr)			
Naina 2012 (22473253)		NR	NR
Rostaing 1994 (7977478)	5 MGUS <sup>18</sup>	Pre-Txp MGUS: 2/5 SCr>150 µmol/L (1.7 mg/dL) (but with no evidence of light chain deposition on kidney biopsy) [3-8 y f/up]	
Soltero 2012 (22044717)	9 vs. 25 MGUS <sup>19</sup>		Pre-Txp MGUS, 9 Txp vs. 25 non-Txp: P=0.13 (from date of MGUS diagnosis) “After the date of transplant, patients with MGUS had a decreased survival compared with patients who were not transplanted” P=0.0008 [Median f/ups: 18.7 mo since Txp, 39.1 mo since MGUS diagnosis (among Txp), 18.9 mo since MGUS diagnosis (among non-Txp)]
Younes 2013 (Conf abstr)			

MGUS = monoclonal gammopathy of undetermined significance,

NR = not reported

c/t = compared to,

w/o = without

Conf Abst = conference abstract,

<sup>18</sup> 5 patients with kidney transplant and 1 patient with heart transplant.

<sup>19</sup> Includes 3 patients <50 years of age, not accounted for above.

**Summary Table: MGRS**

Study Country Year	Sample	N	Pre-Txp MGRS, %	Post-Txp MGRS, n (%) [type]	Hematologic outcomes (post-Txp)	Other
Kaur 2017 (Conf abstr) US 2001-2015	KTx, all	2890 <sup>20</sup>	14 (0.5%): MCN 4 <sup>21</sup>  MIDD 7  MPGN 1 TMA 1  SM 1		Pre-Txp MM: MM 2/2 (100%) w/o ASCT, 0/2 w/ASCT [16 mo median f/up] Pre-Txp MIDD: MM 2/7 (29%), MIDD 1/7 (14%), LPL 1/7 (14%), PT 1/7 (14%) [84 mo median f/up] Pre-Txp MPGN: NR Pre-Txp TMA: C3GN 1/1 (100%) [3 week f/up] Pre-Txp SM: Amyloidosis 1/1 (100%) [90 mo f/up]	

MGRS = monoclonal gammopathy of renal significance,  
 KTx = kidney transplant recipient,  
 Txp = transplantation,  
 Conf Abst = conference abstract,  
 MIDD = monoclonal immunoglobulin deposition disease,  
 MPGN = membranoproliferative glomerulonephritis,  
 TMA = thrombotic microangiopathy,  
 SM = smoldering myeloma,  
 ASCT = autologous stem cell transplantation,  
 LPL = lymphoplasmacytic lymphoma,  
 PT = proximal tubulopathy,  
 f/up = follow-up,  
 C3GN = C3 glomerulonephritis,  
 MCN = myeloma cast nephropathy (called "myeloma kidney" in article),

**Table 2b. MGRS, continued**

Study	N	Kidney outcomes	Survival
Kaur 2017 (Conf abstr)	14 MRGR	Graft failure (alive) 1/14 (7%) [4.7 yr median f/up] <sup>22</sup>	Death: 4/14 (28%) [4.7 yr median f/up]

<sup>20</sup> Of 2890 patients with kidney transplants (100%).

<sup>21</sup> 2 of the 4 patients with myeloma kidney had combined autologous stem cell and kidney transplantations.

<sup>22</sup> Graft failure might have occurred between 1 and 3 years (although it might have been the case that one patient died between 1 and 3 years).

**Summary Table: MGUS Study Limitations**

Study	Who received workup for gammopathy?	Diagnosis	Analysis	Other
Bancu, 2014 (Conf abstr)	No data (Unclear RoB)	No data (Unclear RoB)	No analyses	
Cuéllar-García 2015 (25645776)	Systematically screened (Low RoB)	Electrophoresis (Unclear RoB)	No analyses	
Fenoglio 2013 (Conf abstr)	No data (Unclear RoB)	No data (Unclear RoB)	No analyses	
Gagnon 2017 (Conf abstr)	Not all KTx had SPEP available, implicitly (high RoB)	SPEP, implied (Unclear RoB)	No analyses	
Goebel 2015 (26194021)	MGUS recorded in state database (high RoB)	ICD-9-CM diagnosis code 273.1 (high RoB)	No analyses	
Heymans 2016 (Conf abstr)	Those with data available (high RoB)	SPEP and sIF (low RoB)	No analyses	
Jimenez-Zepeda (21712755)	SPEP as part of pre-Txp workup (low RoB)	SPEP and sIF (low RoB)	No analyses	
Kaur 2017 (Conf abstr)	No data (Unclear RoB)	sIF, uIF, or ICD 10 code (low RoB)	No analyses	Poorly reported, hard to interpret results (poor quality)
Naina 2012 (22473253)	Those with monoclonal protein study pre-Txp, but only 1% were missing data (low RoB),	SPEP and sIF (low RoB)	No analyses	
Rostaing 1994 (7977478)	All had SPEP before transplantation (low RoB)	SPEP, sIF only since ~1991 (Unclear RoB)	No analyses	
Soltero 2012 (22044717)	Only 50% had SPEP available (high RoB)	SPEP and sIF (low RoB)	Unadjusted; unclear methodology, particularly for time since transplant analysis (high RoB)	
Younes 2013 (Conf abstr)	No data (Unclear RoB)	No data (Unclear RoB)	No analyses	

SPEP = serum protein electrophoresis,

sIF = serum immunofixation,

RoB = risk of bias

MGUS = monoclonal gammopathy of undetermined significance,

Conf Abst = conference abstract,

KTx = kidney transplant,

Txp = transplant

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