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Journal:	*BMJ Open*
Manuscript ID	bmjopen-2023-082725
Article Type:	Original research
Date Submitted by the Author:	04-Dec-2023
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Keywords:	Cardiovascular Disease, Renal transplantation < NEPHROLOGY, TRANSPLANT MEDICINE, End stage renal failure < NEPHROLOGY

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Aortic stiffness after living kidney donation:

A Systematic Review and Meta-analysis

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Running title: *Aortic stiffness in living kidney donors*

Total word count: 4,656 words

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Abstract

Objectives: Increased aortic stiffness measured with carotid-femoral pulse wave velocity (cf-PWV) has been associated with adverse cardiovascular outcomes. Some studies have reported increased cf-PWV in living kidney donors after nephrectomy. This review aimed to determine the effects of living kidney donation on cf-PWV, glomerular filtration rate (GFR), systolic (SBP), diastolic blood pressure (DBP) and their differences versus non-nephrectomized healthy individuals.

Design: Systematic review and Meta-analysis.

Data sources: Electronic databases (MEDLINE, EMBASE, Cochrane library, OVID, EBM reviews, grey literature).

Eligibility criteria: We searched for studies that measured cf-PWV in living kidney donors before and/or after nephrectomy. Non-nephrectomized healthy individuals included as controls were the comparators. Studies that provided age-adjusted cf-PWV reference values in normotensive healthy individuals were also included.

Outcome measures: The mean differences in cf-PWV, GFR, and BP before and after nephrectomy and their mean differences versus non-nephrectomized healthy comparators. We also explored differences in yearly-adjusted cf-PWV changes between donors and normotensive healthy individuals.

Data extraction/synthesis: Two independent reviewers extracted data and assessed risk of bias (ROBINS-I) and quality of evidence (GRADE). Pooled effect estimates were calculated using the inverse variance method and analyzed with random effect models.

Results: Nine interventional (652 donors; 602 controls) and 7 reference studies (8,436 individuals) were included. cf-PWV increased at 1-year post-donation (p=0.03) and was on average 0.4 m/s (95% CI: 0.07; 0.60) higher than in healthy controls (p=0.01). These differences were non-significant 5 years post-nephrectomy (p=0.54). GFR decreased after nephrectomy (p<0.001) and remained reduced compared to healthy controls (p<0.001), but SBP and DBP were not significantly different (p≥0.14). Yearly changes in cf-PWV post-nephrectomy were similar to age-adjusted reference values in healthy normotensive individuals (p=0.76).

Conclusions: Aortic stiffness increases independent of BP one year after kidney donation, but the long-term effects seem minimal. These findings may impact future consent of prospective living kidney donors.

PROSPERO Registration number: CRD42020185551.

Key words: *living kidney donors, aortic stiffness, cardiovascular disease, pulse wave velocity, nephrectomy.*

Strengths and limitations of this study

- ⇒ Comprehensive systematic review using multiple electronic databases and rigorous assessment of study quality and certainty of the evidence**.**
- ⇒ The Risk of Bias tool for non-Randomized studies (ROBINS-I) was used to independently evaluate risk of bias and study quality.
- ⇒ The inclusion of 2 comparator groups of non-nephrectomized healthy controls was used to assess changes in carotid-femoral pulse wave velocity in living kidney donors after donation.
- ⇒ The inclusion of blood pressure as outcome allowed to assess if changes in carotid femoral pulse wave velocity were related to changes in blood pressure.
- ⇒ The study was limited by the small number of studies and the paucity of well-designed cohort studies with long-term follow ups.

Introduction

Living kidney donors (LKD) are exposed to perioperative and long-term risks, including potential adverse effects on kidney health.[1] Although kidney hypertrophy is a recognized physiologic response to unilateral nephrectomy, LKD ultimately lose on average 30% of their pre-donation total glomerular filtration rate (GFR).[1,2] Although this reduction in GFR may be of concern to donors and clinicians,[3] the absolute risk increase for kidney failure, cardiovascular disease or death after donation is small and even lower than in the general population.[2,4-5]

Recently, several prospective studies involving measurements of carotid-femoral pulse wave velocity (cf-PWV) have documented that LKD have increased aortic stiffness after nephrectomy when compared to healthy controls of similar age.[6-12] Although most of these investigations involved small samples and limited follow-up times,[13-14] these findings are relevant since increased cf-PWV is associated with adverse cardiovascular outcomes and all-cause mortality in the general population.[15] Since most of these studies did not detect increases in systemic blood pressure (BP) post-nephrectomy,[14] a reduction in GFR may be an independent graded risk factor for cardiovascular remodeling in LKD.[16] Moreover, this phenomenon may be particularly important for young LKD who have the longest risk exposure to the effects of reduced kidney mass.

To determine the effects of living kidney donation on aortic stiffness and their differences relative to non-nephrectomized healthy individuals, we conducted a systematic review and meta-analysis to evaluate the progression of cf-PWV, changes in arterial BP and GFR in LKD before and after nephrectomy. We also gathered data on differences in cf-PWV, BP and GFR between LKD and their non-nephrectomized healthy comparators. Finally, we explored whether yearly changes in aortic stiffness in LKD determined by cf-PWV, differed from age-adjusted reference values in normotensive healthy individuals. We hypothesized that living kidney donation would decrease kidney function and increase aortic stiffness and arterial blood pressure compared to non-nephrectomized healthy individuals.

Materials and Methods

The review was conducted in accordance with the Cochrane Collaboration Methods, Systematic Reviews standards and PRISMA guidelines.[17] The study protocol has been published[18] and registered in PROSPERO (CRD42020185551) (www.crd.york.ac.uk/prospero).[19] The Preferred Reporting items for systematic Reviews and Meta-Analysis guidelines were followed and a checklist file is included.[20]

Data Sources, searching criteria and eligibility

We conducted a comprehensive search (Appendix 1) to retrieve all observational studies published to December 2022 that included healthy individuals participating in a kidney donation program who underwent measurements of cf-PWV before and/or after nephrectomy. The broad nature of our original search captured studies with additional metrics of arterial stiffness.[18] However, these secondary outcomes were not considered in this review as we focused on cf-PWV. The search was applied to several electronic databases including MEDLINE, EMBASE, Cochrane Central databases, Cochrane register of controlled trials, Cochrane Methodology Register, Health Technology Database, "Grey Matters Light", Technologies in health, OVID, EBM Reviews and grey literature. We also searched for studies that included cf-PWV in healthy individuals from the general population that evaluated age effects and aortic stiffness. Population-based studies were searched using the following key words and filters: aortic stiffness, arterial stiffness, cf-PWV, PWV, age, adults, humans, reference or normal values, healthy participants or subjects, and normal volunteers. There were no language restrictions in the initial search although during screening only studies published in English, French, Spanish, Portuguese, and Italian were included. We also identified data sources from manual searches of references in some relevant citations. All search results were downloaded into an Excel spreadsheet and screened by title and authors to remove duplicates.

Study inclusion and exclusion criteria

Our target population included healthy adult individuals (>18 years of age) who met standard institutional kidney donation criteria and had aortic stiffness evaluated with cf-PWV before and/or after nephrectomy. Non-nephrectomized healthy individuals included as healthy controls within the same study were used as comparators. Since prospective randomized clinical trials of kidney donation would never be possible for ethical reasons, we included prospective non-randomized (cohort, case-control, case series, before-and-after) and retrospective studies, provided that ≥ 10 subjects per study were enrolled.

Outcomes

The primary outcomes were the mean differences in cf-PWV before and after nephrectomy in LKD, and the mean differences versus their non-nephrectomized healthy comparators. Secondary outcomes were the pre- and post-donation mean differences in systolic and diastolic BPs and GFRs in LKD and the mean differences versus their non-nephrectomized healthy comparators. Exploratory outcomes were the differences in the yearly-adjusted changes in cf-PWV between LKD and a group of normotensive healthy individuals who participated in population-based studies of aortic stiffness.

Screening and study selection

Two independent reviewers screened abstracts and titles. We excluded non-human, *in-vitro* or modeling studies, narrative/systematic reviews, pediatric investigations, and letters to the editor. After screening was completed, reviewers examined the study methods to confirm that cf-PWV measurements were performed with validated automatic devices. The selected studies underwent full text review by two independent reviewers according to pre-defined inclusion and exclusion criteria (Appendix 2). In case of disagreement, a third reviewer was available to achieve consensus by discussion. We also screened for studies that included healthy individuals from the general population where age-adjusted values for cf-PWV were reported (reference studies). The 2 reviewers selected those studies that explicitly included healthy normotensive individuals (>18 years) with no history of cancer, cardiovascular, neurologic, inflammatory, or kidney disease. To clarify missing information, we contacted study authors by electronic mail. We declared a null response if no reply was obtained after three e-mail attempts within a 4-month period.

Data extraction

A data extraction form was prepared *a priori* from consensus amongst investigators and piloted for optimization. Two reviewers independently performed full data extraction (Appendix 3). Published secondary analyses associated with an original study were considered part of a single study.

Study quality

The risk of bias was assessed using the Risk of Bias tool in non-Randomized studies (ROBINS-I) and each study was independently evaluated by 2 reviewers according to seven domains including confounding, selection, classification of the intervention, deviation from intended intervention, missing data, outcome measurement and reporting.[21] Each reviewer classified the risk of bias for each domain as low, moderate, serious, critical or no information available. A final consensus produced an overall risk of bias for each study. Since the purpose of including reference studies was to provide normative values, their study quality was not assessed.

Quality of evidence

Quality was evaluated according to the 5 domains of the GRADE recommendations, and the overall assessment was reported as very low, low, moderate or high.[22]

Statistical analyses

The weighted mean differences and their 95% confidence intervals (95% CI) were calculated using the reported means and standard deviations (SD) from each study. In cases where different measures of central tendency (i.e., median) and distribution (i.e., inter-quartile) were reported, means and SD were estimated according to the algorithms described by Luo et al.[23] For studies [6,10] that did not include pre-donation values, post-donation differences between LKD and healthy controls were estimated using the mean absolute cf-PWV. To explore statistical heterogeneity between studies, the Q test and the I^2 statistic was used (with a value of I^2 >65 considered to be a highly important heterogeneity). To find potential sources of heterogeneity, we stratified studies by sub-groups according to the duration of follow up and study design. Sensitivity analyses included examination of effect model, parameter estimates and methodological quality. If suitable, the pooled effect estimates were calculated using the method of the inverse variance and data was modeled according to the DerSimonian-Laird Method (random effects model) ($p<0.05$). To reduce "double-counting" error on the effect estimates in LKD cohorts (before-and-after donation), we reduced by 50% the number of study participants for each measurement. Inter-group differences were analyzed using the Cochrane Q test with p value less than 0.10. Publication bias was investigated by Funnel plots, and asymmetry was evaluated if the number of studies in the meta-analysis was greater than 10. Yearly changes in cf-PWV (m/s/year) for kidney donors and healthy controls were estimated using the mean differences between pre- and post-donation values divided by the number of years of observation. In reference studies, the yearly changes in cf-PWV (m/s/year) were estimated according to the age-decade average differences reported at the 90-to-97.5 percentile of the distribution. This cutoff would ensure that the area under the normal curve would fall within 1.282 to 1.960 SD from the mean cf-PWV for each decade. If this data was not available, we used the beta coefficient of the age and cf-PWV regression function. Independent t-tests (2-tailed) evaluated the significance of the between-group differences ($p<0.05$). The differences in cf-PWV are reported as the means and their 95% CI (or their SD, if noted), while for absolute cf-PWV values, medians and quartiles are described. All meta-analyses utilized RevMan 5.4 (Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014).

Results

Study characteristics

The search strategy found 568 citations. After screening and full-text review, 9 studies met the final eligibility criteria (Figure 1). Five studies [6-12] compared LKD and healthy controls, but only 3 of those had measurements before-and-after donation.[8-9,11-12] Four additional studies included single cohorts of LKD with measurements pre- and post-donation.[24-27] We identified 3 reports based on secondary analyses [7,8,28] that were considered part of their original publication [6-9] (Figure 1). Three of our included studies [9,11,12] that were published by the same research group (UK) had participants evaluated at different time periods and some degree of overlap was assumed. In the absence of confirmation, these studies were analyzed independently. Table 1 and Appendix 4 summarize the characteristics of studies, participants and country of origin.

Inset Table 1 here.

Table 1. Demographic characteristics of kidney donors and healthy controls in eligible studies.

Study characteristics		Living Kidney donors					Healthy controls		
Reference ID	Country	Age at donation (years)	Duration of follow-up post-donation	Females/total number of donors (%)	Hypertensive donors/total number of donors (%)	Current or past smokers/total number of donors (%)	Females/ total number of controls (%)	Hypertensive controls/total number of controls (%)	Current or past smokers/ total number of controls (%)
Fesler et al. 2015	France	51.0 ± 10.0	12 mo	39/45 (87)	0/45 (0)	NR/45	N/A	N/A	N/A
DeSeigneux et al. 2015	Switzerland	54.1 ± 10.2	12 mo	12/21 (57.1)	5/21 (23.8)	7/21 (33.0) NR/21	N/A	N/A	N/A
Moody et al. 2015, 2016	UK	46.5 ± 12.1	12 mo	45/68 (66.0)	3/68 (5.0)	8/68 (12.0) 21/68 (30.0)	29/56 (52.0)	3/68 (5.0)	8/68 (12%); 21/68 (30%)
Bahous et al. 2006, 2015	France	41.0 ± 11.0	111 ± 42 mo	66/101 (65.3)	0/101 (0)	NR/101	143/263 (54.4)	0/263 (0)	NR/263
Buus et al. 2019	Denmark	49.5 ± 12.0	12 mo	28/52 (54.0)	17/52 (32.0)	15/52 (29.0); NR/52	N/A	N/A	N/A
Gokalp et al. 2020	Turkey	51.0 ± 13.0	6 mo	20/34 (59.0)	NR/34	NR/34	N/A	N/A	N/A
Price et al. 2020	UK	51.0 ± 12.0	12 mo	90/168 (54.0)	17/169 (10.0)	74/168 (44.0) NR/168	81/138 (59.0)	9/138 (7.0)	38/138 (28.0); NR/138
Kasiske et al. 2020	USA	98% between 18 to 64 years	6 years; and 9 years	6 year visit: 70/109 (64.0) 9 year visit: 72/113 (64.0)	6/203 (3.0)	24/203 (11.8) 40/203 (19.7)	6 year visit: 34/84 (40.0) 9 year visit: 40/100 (40.0)	9/ 201 (4.5)	24/201 (11.9); 45/201 (22.4)
Price et al. 2021	UK	48.0 ± 12.5	12 mo; and 60 mo	27/50 (64.0)	2/50 (4.0)	4/49 (8.1); 15/49 (30.6)	28/45 (62.0)	3/43 (7.0)	2/43 (4.6); 12/43 (28.0)

UK: United Kingdom, NR: not reported, N/A not applicable; mo: months;

Population characteristics

Living kidney donors

A total of 652 LKD had measurements of cf-PWV after kidney donation, but only 438 LKD (in 7 studies) [8,9,11,12,24-27] had examinations before and after nephrectomy. The remaining 214 LKD (in 2 additional studies),[6,7,10] did not have pre-donation assessment. The cf-PWV was measured in 2 studies at 6 months after donation,[8,9,27] in 6 studies at 12 months,[8-9,11-12,24-26] and in 3 studies at 5 years or longer (5, 6 and 9 years) [6,10,12] (Table 1). Amongst all studies, average age at donation was 48.0 years (± 5.0 years) (range: 41.0 to 54.1 years) with most organs donated by females with an average proportion of 63.4% (range: 54% to 87%) per study. Only 3 studies [8,9,10,12] reported the ethnic composition of LKD. Donors were predominantly white Caucasian (range: 90% to 94.6%) with a minority of Asian (range: 6% to 7%) and Black heritage (range: 0% to 3%). In 7 of the 9 studies, an average of 12.5% (range: 0% to 32%) of LKD were hypertensive at the time of donation and this rate increased to an average of 18.6% (range: 4% to 32%) after nephrectomy. Moreover, an average of 32.9% of donors (range: 28% to 44%) were current smokers and/or individuals with a history of previous smoking, although the duration of exposure was not reported. The most common medications prescribed for LKD prior to organ donation were antihypertensives and lipid reducing drugs (e.g., statins). The most common antihypertensive medications were angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARB) (range: 0% to 19% in 5 studies), calcium channel blockers (range: 2% to 5%; in 6 studies) and beta blockers (2%; in 3 studies). Statins were reported with an average rate between 0% and 12% in 6 studies. There was no information on cardiovascular risk assessment pre-donation and hypertension management with diuretics.

Healthy controls

A total of 602 healthy individuals were included as comparators in 5 studies (Table 1). Two studies had comparative assessments at 12 months after nephrectomy,[8,911] one at 12 and 60 months,[12] and two at 5 years or longer (5, 6 and 9 years).[6,7,10] The average age of healthy controls in these studies was 46 years (range: 43 to 49 years) compared to 49 years (range: 46 to 51 years) in kidney donors. The incidence of hypertension, history of cardiovascular disease and diabetes mellitus was higher in kidney donors post-donation, relative to controls. The average proportion of hypertension, history of cardiovascular disease and diabetes mellitus was 6.3 % (range: 0% to 9% in 5 studies), 16.7% (range: 0% to 28% in 3 studies) and 0.5 % (range: 0% to 2% in 4 studies) in healthy controls compared to 11.0 % (range: 5% to 18.8% in 5 studies), 19.6% (range: 4.9% to 34% in 3 studies) and 1.6% (range: 0% to 5.9% in 4 studies) respectively in LKD. Only 3 studies [8,9,11,12] documented the proportions of current and previous smokers between these two sub-populations ranging between 2% and 28% in controls versus 6% and 44% in donors. The most frequent medications prescribed to healthy controls as reported in 2 studies [11,12] were ACE inhibitors/ARBs, statins and calcium channel blockers. Their proportions at the time of initial recruitment ranged from 3% to 7%, 7% to 13% and 2% to 3% respectively. Ethnicity in healthy controls was only reported in 3 studies.[8,9,10,12] The ethnic distribution of these participants was white Caucasian (range: 84% to 95%), Asian (9%) and black heritage (6% to 7%). None of the studies reported cardiovascular risk in healthy controls.

Outcome measures

Aortic Stiffness

The primary outcome analysis included 7 studies [8,9,24-27] with non-adjusted cf-PWV values plus 2 studies[11,12] whose values were adjusted according to mean BP and heart rate. Due to limited information in these 2 studies, their adjusted values were not transformed. Tables 2 and 3 summarize the unadjusted cf-PWV values in donors and controls respectively. The median unadjusted cf-PWV prior to nephrectomy was 7.10 m/s (quartiles: 6.80, 7.52) and this value increased to a median of 7.21 m/s (quartiles: 7.14, 7.27) at 6 months, 7.30 m/s (quartiles: 7.22, 7.68) at 12 months and 7.69 m/s (quartiles: 7.50, 8.60) at 5 years. Figure 2 shows the Forest plots of the effect estimates for the unadjusted cf-PWV in LKD before and after nephrectomy (panel "a") and their differences against healthy comparators (panel "b"). The unadjusted cf-PWV in LKD increased with time after nephrectomy (Z=3.1, p=0.002; I^2=0%). While these effects were statistically significant at 12 months after nephrectomy (Z=2.2, p=0.03; I^2=10%; 6 studies), they were not significant at 6 months (Z=1.3, p=0.20; I^2= 0%; 2 studies) or 5 years and longer (Z=1.8; p=0.07; one study). The mean difference in the unadjusted cf-PWV before and after donation was 0.23 m/s (95% CI: -0.12; 0.58) at 6 months, 0.30 m/s at 12 months (95% CI: 0.03; 0.57) and 0.60 m/s at 5 years (95% CI: -0.04; 1.24). At 12 months post-donation, unadjusted cf-PWV values in LKD were on average 0.4 m/s (95% CI: 0.08; 0.72) higher than in healthy controls (Z=2.43; p=0.01; 3 studies), but this difference became non-significant (mean: 0.15 m/s; 95% CI: -0.32; 0.62) at 5 years or longer after donation (Z=0.62; p=0.54). Statistical heterogeneity between studies was high at 12 months (I^2=78%; p=0.01) and at 5 years (I^2=65%; p=0.02).

Insert Tables 2 and 3 here

Table 2. Hemodynamic characteristics in living kidney donors before and after nephrectomy.

study	LKD Pre-donation baseline				LKD Post-donation follow-up				
Citation	cf-PWV (m/s)	GFR (ml/min/ 1.73m²)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Follow-up post-donation	cf-PWV (m/s)	GFR (ml/min/ 1.73m²)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Fesler et al. 2015	7.2 ± 1.3	107 ± 19	122 ± 12	70 ± 9	12 mo	6.8 ± 1.1	73 ± 15;	122 ± 13	70 ± 8
DeSeigneux et al. 2015	8.5 ± 1.2	95 ± 10	116.6 ± 13	76.0 ± 7.3	12 mo	9.4 ± 1.8	61 ± 11	N/R	N/R
Moody et al. 2015, 2016	6.8 ± 1.1	89 ± 19	121 ± 8.0	75 ± 6.0	12 mo	7.3 ± 1.5	59 ± 13	122 ± 12.0	76 ± 9
Bahous et al. 2006, 2015	N/R	107.5 ± 20	114.0 ± 18	69.1 ± 19.4	111 ± 42 mo	9.5 ± 2.5	86.2 ± 18	129.6 ± 20	81.6 ± 11.8
Buus et al. 2019	7.5 ± 1.3	100.6 ± 15	120 ± 14	74 ± 8	12 mo	7.8 ± 1.6	64.7 ± 10.6	119 ± 13	74.9 ± 9.7
Gokalp et al. 2020	7.6 ± 1.8	99.8 ± 19	120 ± 15.6	74.3 ± 9.2	6 mo	7.3 ± 1.5	61.9 ± 15.1	122.1 ± 20.1	74.9 ± 9.7
Price et al. 2020	7.0 ± 1.3	91 ± 15	124 ± 10	79 ± 8	12 mo	7.3 ± 1.4	64 ± 14	124 ± 10	79 ± 8
Kasiske et al. 2020 *	N/R	91 ± 17.7	120 ± 13.6	69.7 ± 8.8	6 years	7.1 ± 1.6	64 ± 8.6	117.5 ± 11.2	73.1 ± 8.6
	N/R	-	-	-	9 years	7.7 ± 1.7	62 ± 8.9	117.1 ± 13.0	73.9 ± 10.0
Price et al. 2021 *	6.7 ± 1.0	95 ± 15	121 ± 9	73±7	12 mo	7.2 ± 1.1	65.8 ± 10.3	121.2 ± 15.1	74 ± 10.2
	-	-	-	-	60 mo	7.3 ± 1.3	67.4 ± 12.4	123.6 ± 11.5	78 ± 9.0

BP: blood pressure; cf-PWV: carotid-femoral pulse wave velocity (m/s); GFR: glomerular filtration rate; LKD: living kidney donors; mo: months; N/R: not reported; * Kasiske et al. (6 and 9 years) and Price et al. (12 months and 60 months) used the same pre-donation baseline for each of their 2 follow-up points after donation.

Table 3. Hemodynamic characteristics of healthy comparators at baseline and follow-up.

study	Healthy comparators – Measurement at enrollment				Healthy comparators - Measurement at follow-up				
	cf-PWV (m/s)	GFR (ml/min/ 1.73m²)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Time point at follow-up	cf-PWV (m/s)	GFR (ml/min/ 1.73m²)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Moody et al. 2015, 2016	6.7 ± 1.1	89 ± 19	122 ± 11	74 ± 8	12 mo	6.7 ± 1.1	86 ± 19	121 ± 10	76±9
Bahous et al. 2006, 2015 (NRR group)[&]	N/R	N/R	N/R	N/R	111 ± 42 mo	8.5 ± 1.5	N/R	125.2 ± 9.5	75.7 ± 6.1
Bahous et al. 2006, 2015 (RR group)[&]	N/R	N/R	N/R	N/R	111 ± 42 mo	8.9 ± 1.3	N/R	123 ± 15.1	78.5 ± 10.6
Price et al. 2020	7.0 ± 1.4	94 ± 16	122 ± 10	77 ± 8	12 mo	7.2 ± 1.4	96 ± 17	123 ± 12	78 ± 9
Kasiske et al. 2020 *	N/R	90 ± 16.2	117.1 ± 13.0	69.7 ± 8.8	6 years	7.29 ± 2.5	86 ± 15.7	118.8 ± 14.2	73.1 ± 8.6
					9 years	7.90 ± 2.3	84 ± 13.5	120 ± 14.9	73.9 ± 10.0
Price et al. 2021 *	6.78 ± 0.17	99 ± 16	122±11	75±9	12 mo	6.73 ± 0.16	96.9 ± 11.9	120.8 ± 7.6	75.4 ± 7.6
					60 mo	7.54 ± 0.22	94.1 ± 16.7	122.7 ± 11.8	78.4 ± 9.0

BP: blood pressure; cf-PWV: carotid-femoral pulse wave velocity; GFR: glomerular filtration rate; mo: months; N/R: not reported; * Kasiske et al. (6 years and 9 years) and Price et al. (12 months and 60 months) used the same baseline at enrollment for each of their 2 follow-up points; NRR group: not recipient related group; RR group: recipient related group; [&] Bahous et al. 2006, 2015, identified 2 sub-groups (NRR and RR) within their comparator group relative to the kidney recipient.

Kidney function

GFR in LKD was measured in one study at 6 months post-nephrectomy,[27] in 6 studies at 12 months [8,9,11,12,24-26] and in 3 studies at 5 years or longer.[6,7.10,12] Six studies estimated GFR using the CKD Epidemiology Collaboration equation (CKD-EPI) based on the ^{Cr51}EDTA clearance,[8,9.11,12,24-26] one study[27] estimated GFR from 24-hour urine creatinine clearance, and 2 additional studies [6,7.10] used both the modification of Diet in renal disease (MDRD) and CKD-EPI from Iohexol clearance. Supplementary Figure S-1 shows the Forest plots of the effects estimates on GFR in LKD (panel "a") and their differences against healthy controls (panel "b"). Relative to before nephrectomy, GFR decreased by an average of 30 ml/min/1.73 m^2 (95% CI: -32; -28) throughout the 5-year follow-up period ($Z=27.4$; $p<0.001$). In particular, GFR decreased by 38 ml/min/1.73 m^2 (95% CI: -49; -26) within the first 6 months after nephrectomy (one study; $Z=6.5$; $p<0.001$), by 31 ml/min/1.73 m^2 (95% CI: -34; -27) at 12 months (6 studies; $Z=19.3$; $p<0.0001$), and by 28 ml/min/1.73 m^2 (95% CI: -31; -25) at 5 years or longer (3 studies; $Z=17.3$; $p<0.0001$). When these values were compared to healthy controls, LKD had significantly lower GFRs (mean differences: -26 ml/kg/1.73 m^2; 95% CI: -28; -23; $Z=22.1$; $p<0.001$).

Systemic BP

In LKD, systolic and diastolic BP were measured non-invasively at 6 months post-donation in 1 study,[27] at 12 months in 4 studies, [8,9,11,25,26] at 1 and 5 years in another,[12] and longer than 5 years in 2 studies.[6,7,10] A single study [24] did not report BP post-nephrectomy. Five studies [8,9,10,11,12,26] reported the daily average BP derived from 24-hour BP monitoring, while four studies [6,7,24,25, 27] reported BP values from the average of 3 measurements taken at the time of the office visit. Most studies except one,[6,7] measured BP in controls at initial recruitment and follow-up. The Forest plots of the effect estimates on the systolic and diastolic BP are represented in Supplementary Figures S-2 and S-3 respectively. Diastolic BP (Z=2.6; p=0.009), but not systolic BP (Z=0.8; p=0.44) increased with time after donation. This effect was only significant at 5 years post-nephrectomy, when diastolic BP increased by an average of 5 mm Hg (95% CI: 2.1, 8.8; I^2=63%; Z=3.2; p=0.001) relative to pre-donation values. When these time-related changes were compared to healthy controls, differences in systolic (mean differences: 0.8 mm Hg 95% CI: -1.2; 2.7) and diastolic BP (mean differences: 1.1 mm Hg; 95% CI: -0.4; 2.6) at 5 years or longer were non-significant (systolic: Z=0.8; p=0.43; diastolic: Z=1.48, p=0.14). Overall, statistical heterogeneity was moderate for systolic (I^2=44%; $\chi 2$=12.5; p=0.08) and marginal for diastolic BP (I^2=53%; $\chi 2$=14.1; p=0.04).

Comparison with reference values

Supplementary Table S-1 shows the yearly changes in cf-PWV for seven reference studies that included 8,436 normotensive healthy participants (>18 and <70 years).[29-35] Supplementary Table S-2 shows the estimated yearly changes in non-adjusted cf-PWV for LKD and healthy controls. The non-adjusted cf-PWV increased by an average of 0.174 m/s per year (± 0.720) in LKD (8 studies) and 0.090 m/s per year (± 0.951) in healthy controls (4 studies). The yearly increases in LKD and their controls were comparable to the 0.118 m/s per year (± 0.134) average increase from normotensive healthy individuals (>18 to <70 years) (donors: t=0.20; p=0.84; controls: t=0.078; p=0.93). Since previous studies have indicated a larger yearly increase in cf-PWV for older age groups, we performed a sub-group analysis for individuals ≤ 60 years and > 60 years. The average yearly increase in cf-PWV in reference studies for individuals ≤ 60 years was 0.0754 m/s (± 0.047) compared to 0.158 m/s (± 0.171) in those > 60 years (Supplementary Table S-1). Our analysis showed that there was no difference in the average yearly change in cf-PWV between LKD (t=-0.301; p=0.76) or healthy controls (t=-0.026; p=0.97) against normotensive healthy individuals ≤ 60 years.

Sensitivity analyses

The effect of overlapping on the effect estimates between LKD and healthy controls was tested by sequential exclusion/inclusion of the involved studies.[8,9,12] Exclusion decreased the mean cf-PWV difference at 12 months (full model: 0.40 m/s, partial models: 0.34 m/s, 0.31m/s) and increased statistical heterogeneity (full model: 78%; partial models: 81%, 86%), but there was no effect on the overall estimates (χ^2=0.32; df=1, p=0.57). Our assessment of parameter estimates, quality and effect model did not change the final analysis.

Risk of bias

Supplementary Table S-3 summarizes the assessment of the risk of bias with the ROBINS-I tool. Four of the 5 studies that included a control group [8-12] had moderate risk of bias (80%) and one serious risk of bias.[6,7] Three single cohort studies [24,25,27] had serious risk of bias (75%) and one moderate risk of bias. [26] No study was classified as low-risk or critical risk of bias. Risk of bias was associated with the presence of confounding bias, selection bias due to relaxation of inclusion criteria for donors and controls, missing data and selective reporting.

Funnel plots of asymmetry

The small number of studies (<10) in the meta-analysis and the likelihood that any test on asymmetry would be underpowered precluded using any test for reporting bias. Supplementary

Figure S-4 shows effect estimates and sample sizes for studies with cf-PWV between LKD and controls. A large asymmetry for both small and large sample size studies was evident and suggested potential risk for publication bias.

Certainty of the Evidence

Supplementary Table S-4 summarizes certainty of the evidence for all outcomes according to the GRADE methodology. Confidence on the effect estimates was low to moderate for the cf-PWV, low for systemic BP and moderate to high for GFR.

Discussion

In this systematic review, we pooled data from 652 LKD, 602 healthy controls and 8,436 normotensive healthy participants with standard cf-PWV measurements to evaluate the effects of nephrectomy on aortic stiffness after living kidney donation. Based on low to moderate quality of evidence, our findings suggest that the impact of nephrectomy on aortic stiffness at 5 years post-donation or longer is minimal, despite a reduction in kidney function. On the other hand, cf-PWV increases within the first year after nephrectomy, exceeding values observed in selected groups of non-nephrectomized healthy individuals (average difference: 0.4 m/s), although these differences are negligible at 5 years post-donation (average difference: 0.15 m/s). Additionally, the yearly changes in cf-PWV after donation were similar to those in healthy normotensive individuals from the general population. Our review also suggests that 5 years after donation, systolic and diastolic BPs increased by an average of 3- and 5-mm Hg respectively, but these changes were similar to those identified in healthy control groups. Thus, we hypothesize that vascular remodeling occurs within the first year post-nephrectomy, leading to discrete elevation of aortic stiffness with no changes in systemic BP. Five years after nephrectomy however, progression of aortic stiffness in LKD is similar to the age-dependent effects observed in a healthy normotensive population.

Compared to values before donation, GFR in LKD decreases by an average of 30 ml/min/1.73 m^2 between 6 months and 5 years after nephrectomy. These results are comparable to previous studies that reported reductions in kidney function between 30% and 50% after kidney donation.[1,2,11,12,36] Our current analysis supports that a reduction in kidney function of such magnitude after donation is insufficient alone to cause significant effects on aortic stiffness at least 5 years post-donation. In contrast, similar reductions in kidney function in early-stage chronic kidney disease (CKD) are associated with increased aortic stiffness and reduced vascular distensibility.[37-40] Inflammation-mediated endothelial injury,[12,37] increased upregulation of matrix metallo-proteinase-2,[41] abnormal calcium/phosphorous mineral balance[42] and extracellular fluid excess[37] are mechanisms of vascular injury more likely found in CKD patients, which may play a role on the increased aortic stiffness in CKD, but not after kidney donation.[6,36,41,43]

Studies on the progression of aortic stiffness after kidney donation have had contradictory results. While some studies have shown an increase in aortic stiffness [6-12,26] others have documented a negligible effect.[24,25,27] Varying study designs, small sample sizes and short-term follow-ups may have contributed to the heterogeneity in results. Our findings confirm that there is a paucity of well-designed cohort studies with large sample sizes and long-term follow ups. In addition, although our meta-analysis increased the robustness of the comparisons between donors and controls, this analysis may have been underpowered to detect small differences. A difference of 0.4 m/s (SD: 3) in cf-PWV between donors and controls would have required at least 883 participants per group with 80% power and level of significance of 5%. Although our analysis was adjusted for duration of follow-up and study quality, heterogeneity between studies was still present. We speculate that relaxation of study inclusion criteria may have led to unbalanced distribution of risk determinants (i.e., hypertension, smoking, diabetes, dyslipidemia) between the two cohorts. Because these confounders may decrease comparability, baseline differences should be minimized in future studies.

The effect of reduced kidney function, independent of increased BP, on aortic stiffness in LKD is controversial.[14,44,45] In partially nephrectomized rats, reduced kidney function modified the viscoelastic properties of large arteries independent of the effects of age and BP.[46] However, since serum creatinine increased more than double compared to control animals, the magnitude of reduction in GFR may have not been similar to what is observed in LKD. Our review suggests that except for a small increase in cf-PWV within the first year post-donation, there were no differences in BP between healthy donors and controls. These findings support previous studies that have reported a reduction in the Magnetic Resonance Imaging-detected aortic distensibility in LKD but not in healthy controls at one year post-donation,[9] with these differences becoming negligible at 5 years post-nephrectomy.[12] Furthermore, these changes in donor's aortic stiffness may be associated with an increase in left ventricular mass one year post nephrectomy,[9,27] which is no longer noticeable at 5 years.[12,47]

Several risk factors (e.g., African American or Hispanic ethnicity, obesity, age, diabetes) may increase the risk for elevated BP and aortic stiffness post-donation.[14,25,48,49,50] However, few studies have documented the role of genetics or ethnicity factors in the development of CKD and increased aortic stiffness.[6,7,51,52] Kidney donors of African ancestry with mutations in the Apolipoprotein L1 gene (APOL1) are at higher risk for developing CKD, imposing new challenges to the process of donor selection and consent.[53,54] Bahous et al[6,7] who explored differences in cf-PWV between recipient and non-recipient-related healthy volunteers of Lebanese ancestry, found a significantly higher rate of elevated aortic stiffness in recipient-related healthy controls. Moreover, Muzaale et al.[52] and Wu et al.[55] reported marked differences in the risk for kidney failure across different types of donor-recipient and ethnicity relationships, suggesting genetic factors. Consequently, the role of genetic determinants in modifying risk of aortic stiffness post-donation cannot be ruled out.

Beyond biological effects of reduced kidney function, nephrectomy may also result in alterations of the arterial network that are associated with changes in hemodynamics and functional stiffness of the arterial tree.[56] Although few studies have documented that compensatory growth of the remaining kidney is commonly seen after unilateral nephrectomy,[57] the relationship of this phenomenon with cardiovascular remodeling and vascular stiffness remains elusive. Interestingly, several circulating growth factors released during compensatory kidney hypertrophy,[58] have been associated with myocardial and central vascular remodeling.[59] In particular, growth hormone (GH) and its main mediator insulin growth factor-1 (IGF-1) are implicated in the early stages of compensatory renal hypertrophy [60] and increase aortic wall thickness in transgenic mice models without any significant change in arterial BP.[61] Thus, we speculate that these circulating growth factors may be linked to the cardiovascular remodeling process and transient increase in aortic stiffness early after nephrectomy.

Limitations

The strength of this review includes a rigorous systematic methodology and assessment of study quality and certainty of the evidence. Nevertheless, our conclusions may be limited by the small number of studies and participants, and the restricted access to information for data standardization.[8,9,11,12] Furthermore, our sensitivity analysis on studies where overlapping was suspected,[8,9,11,12] suggested a reduced mean difference in cf-PWV at 12 months post-donation.

Thus, the likelihood that overlapping might have influenced our effect estimates cannot be completely excluded. Since cf-PWV is an operator-dependent technique,[62] an important issue is the comparability between medical devices and technical reproducibility of measurements. All selected studies utilized standard devices (Supplementary Table S-1), although no information was given on their reproducibility.[49,62] Despite our efforts to detect potential sources of heterogeneity, residual confounding was still present, and this may have impacted comparability between cohorts. Additionally, we recognize that the different techniques utilized in the measurement of GFR (estimated versus direct measurement), and BP (24-hour monitoring versus office) may have contributed to the variability on these outcomes.[63,64] Moreover, the confounding effects of anti-hypertensive therapy on the control of BP after donation cannot be ignored. Finally, publication bias cannot be entirely ruled out.

Conclusions

Our systematic review and meta-analysis documented that reduced kidney function after living kidney donation is associated with a small elevation in aortic stiffness within the first year, independent of changes in systemic BP. These effects however, become negligible 5 years post-donation. The data suggest that vascular remodeling occurs within the first year post-nephrectomy, but is no longer detected after 5 years. In the absence of other critical cardiovascular risk factors, the effects of nephrectomy on aortic stiffness in LKD at least 5 years after donation is insignificant. These results may have implications for the future evaluation and consent of prospective living kidney donors.

Acknowledgements

We acknowledge the assistance and support of Ms. Risa Shorr from the Learning services at the Ottawa Hospital during the development of our searching strategy. The authors express appreciation for "all individuals that donate a kidney for the benefit of another human being".

Author contributions

RAR and KDB conceived the study; RAR designed the study, created the analytical plan, synthesized, analyzed, interpreted the evidence and drafted the manuscript; RAR and KM were involved in study screening, data extraction, verification and quality appraisal; RAR, MA, AB, EC and KDB provided comments and reviews to initial drafts. All authors have read, reviewed and approved the final version of the manuscript.

Funding

This research received no specific grant from any funding agency in the public, commercial or not-for profit sectors.

Competing interests

None declared.

Patient and Public involvement

Patients and/or the public were not involved in the design, conduct, reporting or dissemination plans for this study.

Patient consent for publication

Not required.

Data availability statement

All data relevant to the study are included in the article or uploaded as supplementary information

Supplementary information

File 1; Supplementary Tables (Tables S1, S2, S3 and S4); Supplementary Figures (Figures S1, S2, S3, S4); Appendices (Appendix 1, 2, 3, 4)

File 2: PRISMA checklist.

File 3: MOOSE checkist

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Figure Legends

Figure 1: PRISMA flow chart.

Figure 2: Pooled effect estimates on the carotid-femoral pulse wave velocity (cf-PWV) in living kidney donors from before to after nephrectomy (Panel "a") and on their differences relative to healthy comparators (Panel "b"). Because pre-donation values for Bahous et al. (2006) and Kasike et al. (2020) were not provided (Panel "b"), mean differences between living kidney donors and controls were calculated using their mean absolute cf-PWV values. In single cohort studies with before-and-after design (Panel "a") and in the study by Bahous et al. 2006 (Panel "b"), the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors during the analysis. NRR: non-recipient related, RR: recipient related.

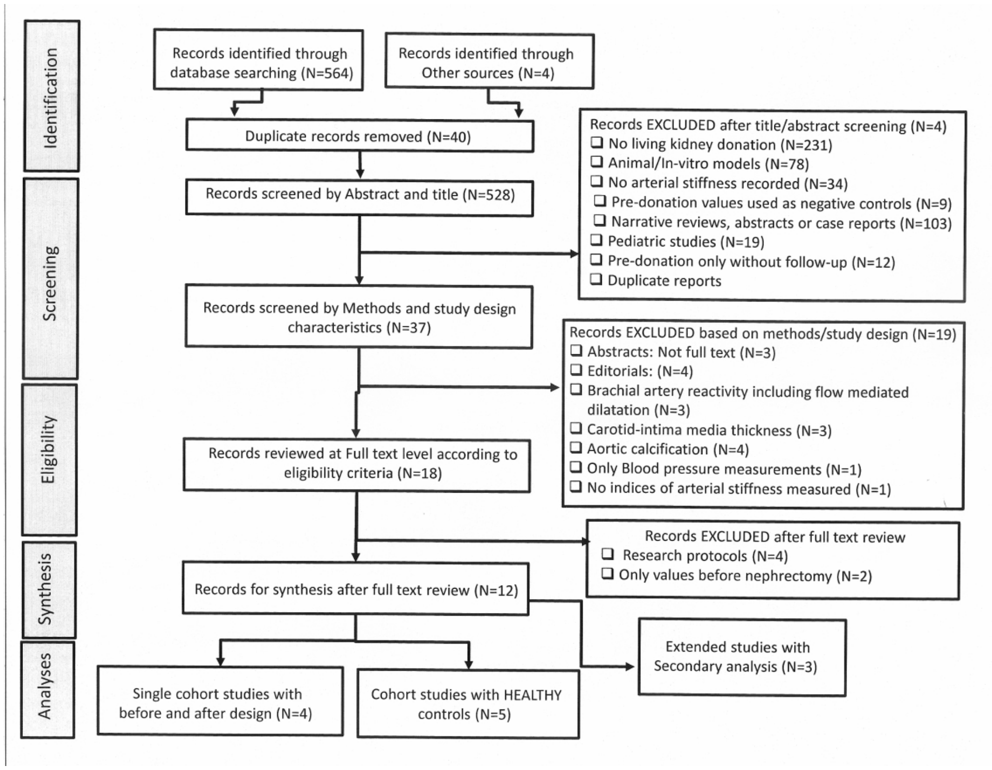

Figure 1. Flow chart of studies identified in the systematic review.

273x208mm (300 x 300 DPI)

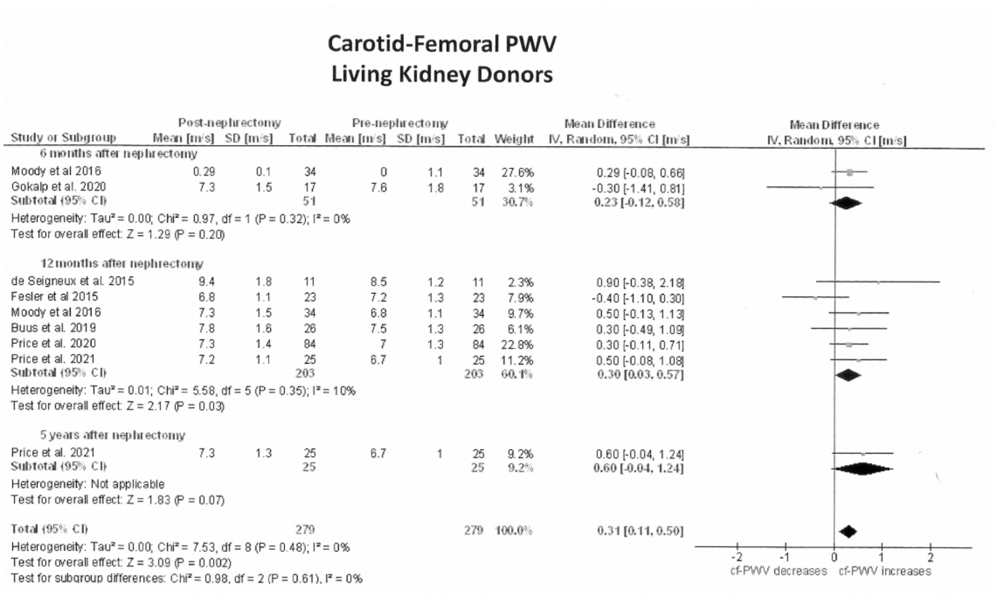

Figure 2 - Panel A - Pooled effect estimates on the carotid-femoral pulse wave velocity of living kidney donors from before to after nephrectomy.

273x163mm (300 x 300 DPI)

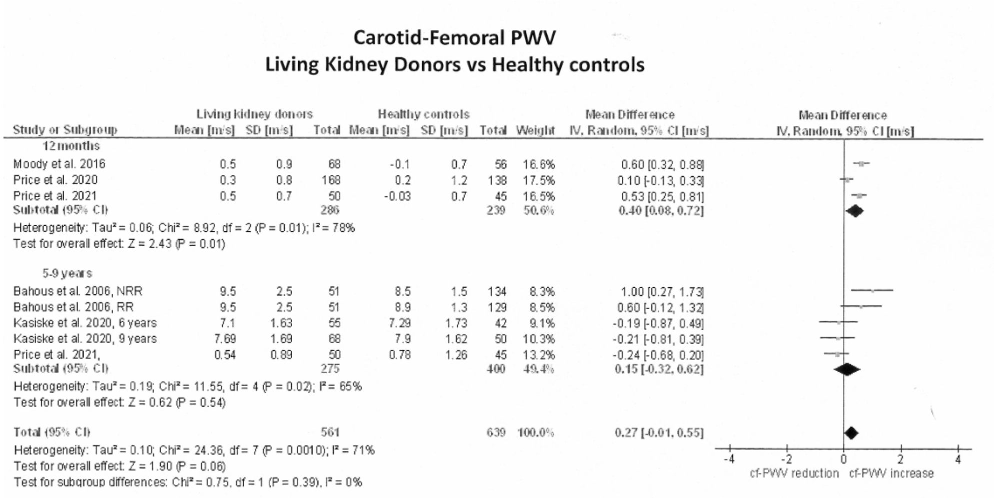

Figure 2- Panel B: Pooled effect estimates on the differences in carotid-femoral pulse wave velocity between living kidney donors and healthy controls.

275x141mm (300 x 300 DPI)

Aortic stiffness after living kidney donation:

A Systematic Review and Meta-analysis

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SUPPLEMENTAL MATERIAL

I - Supplementary Tables

II - Supplementary Figures and Figure legends

III - Appendices

Supplementary Tables

Table S-1 Estimated average yearly change (± SD) in cf-PWV (m/s/year) for normotensive healthy individuals participating in population-based studies

Table S-2. Estimated average yearly change (± SD) in cf-PWV (m/s/year) for living kidney donors and healthy comparators.

Table S-3. Risk of bias assessment according to the ROBINS-I scale for eligible studies

Table S-4. Assessment of the certainty of the evidence on the primary and secondary outcomes according to the GRADE guidelines.

Table S-1 Estimated average yearly change (± SD) in cf-PWV (m/s/year) for normotensive healthy individuals participating in population-based studies with age-adjusted values.

variables	Arterial stiffness' collaboration 2010	Farro et al 2012	Baier et al. 2018	Elias et al. 2011	Baldo et al 2018	Kozakova et al. 2015	Gomez-Sanchez et al. 2020
Age (years) (range)	>30 to ≥70	>18 to 69	>18 to 80	40 to 90	35 to 74	18 to 78	35 to 75
Measurement device	SphygmoCor	SphygmoCor	Vicorder	SphygmoCor	Complior	SphygmoCor	SphygmoCor
Total number of participants	1,455	429	3,092	502	2,158	307	493
Average cf-PWV (m/s/year) in all participants	0.1500 ± 0.0967 (#)	0.1580 ± 0.0864	0.1000 ± 0.1208 (&&)	0.105 ± 0.2913 (&)	0.1047 ± 0.0526 (*)	0.090 ± 0.005	0.1188 ± 0.2915
Average cf-PWV in ≤ 60 years (m/s/year)	0.0967 ± 0.0404 (#)	N/R	0.0733 ± 0.0321	N/R	0.0763 ± 0.0359 (*)	0.088 ± 0.007	0.0425 ± 0.1645 (**)
Average cf-PWV in > 60 years (m/s/year)	0.2300 ± 0.1131 (#)	N/R	0.0550 ± 0.0212	N/R	0.1615 ± 0.0163 (*)	0.150 ± 0.019	0.1950 ± 0.418 (**)
cf-PWV in Females (m/s/year)	N/R	N/R	N/R	N/R	0.0960 ± 0.173	0.099 ± 0.005	0.1603 ± 0.1472
cf-PWV in males (m/s/year)	N/R	N/R	N/R	N/R	0.0919 ± 0.182	0.076 ± 0.005	0.1805 ± 0.1515

(*) Estimates are based on the 95% percentile values from all males and females included in this study.

(**) Data represent average values from reported males and females sub-groups in this study. The reported 95% CI were transformed to SD using the method of Wu et al (2018).

(&) Estimates are based on the regression coefficient (b) of the relationship between age and cf-PWV in all participants from this study (Table 4) and adjusted by mean arterial pressure, weight, height, glucose and creatinine. The reported standard error (SE) was converted to SD according to the formula: SD=SE*(SQRT sample size).

(#) Estimates are based on the reported 90th percentile of the distribution in the sub-sample defined as "normal values" in this study (Table 4)

&& Estimates are based on the 97.5th percentile values from the normotonic subgroup in this study (Table 4).

Table S-2. Estimated average yearly change (± SD) in cf-PWV (m/s/year) for living kidney donors and healthy comparators.

Living Kidney Donors									
	Buus et al. 2019	Gokalp et al 2020	De Seigneux et al 2015	Moody et al 2016	Fesler et al. 2015	Price et al 2020	Price et al. 2021	Price et al 2021	Kasiske et al. 2020
Age at measurement (years)	51.0 ± 11.6	50.97 ± 13	55.1 ± 10.2	47.5 ± 12.1	52 ± 10	52 ± 12	49 ±12.5	54.3 ± 12.3	98.6% (age: 18 to 64)
Time frame of observation	12 mo	12 mo	12 mo	12 mo	12 mo	12 mo	12 mo	60 mo	36 mo
Device	SphygmoCor	SphygmoCor	SphygmoCor	SphygmoCor	Sphygmocor	SphygmoCor	SphygmoCor	SphygmoCor	Sphygmocor
Number of subjects	51	34	21	68	45	168	50	42	109
Average yearly change in cf-PWV (m/s/year)	+ 0.30 ± 0.900	- 0.24 ± 0.401	+ 0.3 ± 0.469	+ 0.5 ± 0.900	- 0.4 ± 0.250	+ 0.3 ± 0.821	+ 0.50 ± 0.717	+ 0.108 ± 0.920	+ 0.197 ±0.435

Healthy comparators									
				Moody et al 2016		Price et al 2020	Price et al. 2021	Price et al 2021	Kasiske et al. 2020
Age at measurement (years)	N/A	N/A	N/A	45.1 ± 12.8	N/A	49.0 ± 14	45.3 ± 13.07	50.3 ± 12.91	95.6% (age range: 18 to 64)
Time frame of observation	N/A	N/A	N/A	12 mo	N/A	12 mo	12 mo	60 mo	36 mo
Number of subjects	N/A	N/A	N/A	56	N/A	138	45	42	84
Average yearly change in cf-PWV (m/s/year)	N/A	N/A	N/A	- 0.1 ± 0.700	N/A	+ 0.2 ± 1.190	− 0.03 ± 0.680	+ 0.16 ± 1.23	+ 0.203 ± 0.52

cf-PWV: carotid-femoral pulse wave velocity; mo: months; N/A not applicable;

Table S-3. Risk of bias assessment according to the ROBINS-I scale for eligible studies.

study	1. Bias due to confounding	2. Selection Bias	3. Classification Bias	4. Bias due to deviations from intended intervention	5. Bias due to missing data	6. Bias in outcome measurement	7. Bias in selection of the reported result	OVERALL RISK OF BIAS
Fesler et al. 2015	SERIOUS	LOW	SERIOUS	MODERATE	MODERATE	MODERATE	LOW	SERIOUS
De Seigneux et al. 2015	SERIOUS	SERIOUS	MODERATE	MODERATE	SERIOUS	MODERATE	SERIOUS	SERIOUS
Moody et al. 2016	LOW	MODERATE	LOW	LOW	MODERATE	LOW	LOW	MODERATE
Bahous et al. 2006	SERIOUS	MODERATE	MODERATE	MODERATE	SERIOUS	MODERATE	SERIOUS	SERIOUS
Buus et al. 2019	MODERATE	LOW	MODERATE	MODERATE	LOW	MODERATE	MODERATE	MODERATE
Gokalp et al. 2020	SERIOUS	SERIOUS	MODERATE	SERIOUS	NO INFORMATION	MODERATE	MODERATE	SERIOUS
Price et al. 2020	LOW	MODERATE	LOW	LOW	MODERATE	MODERATE	LOW	MODERATE
Kasiske et al. 2020	MODERATE	MODERATE	LOW	LOW	MODERATE	MODERATE	MODERATE	MODERATE
Price et al. 2021	LOW	LOW	LOW	LOW	MODERATE	MODERATE	LOW	MODERATE

Definition and interpretation of individual items for the ROBINS-I scale: [21]

1.- Confounding of intervention effects occurs when one or more prognostic variables (variables that predict the outcome of interest) also predict whether an individual receives one or the other of the interventions of interest.

2.- When exclusion or inclusion of some participants, or the initial follow up time of some participants, or some outcome events, is related to both intervention and outcome, there will be an association between interventions and outcome even if the effects of the interventions are identical. This type of bias is called selection bias.

3.- Non-differential misclassification is unrelated to the outcome and will usually bias the estimated effect of intervention towards the null. Differential misclassification however, may occur when misclassification of intervention status is related to the outcome or the risk of the outcome, and is likely to lead to bias. It is therefore important that, wherever possible, interventions are defined and categorized without knowledge of subsequent outcomes. Differential misclassification can also occur if information (or availability of information) on intervention status is influenced by outcomes.

4.- This domain (sometimes known as "performance bias") relates to biases that arise when there are systematic differences between the care provided to experimental intervention and comparator groups, beyond the assigned interventions. Bias may occur when these differences arise because of knowledge of the intervention applied and the expectation of finding a difference between experimental intervention and comparator consistent with the hypothesis being tested in the study. Deviations from intended interventions may arise because an intervention was not implemented successfully because participants did not adhere to interventions, or because important co-interventions were not balanced between intervention and comparator groups.

5.-Reasons for missing data include attrition (loss to follow up), missed appointments, incomplete data collection and participants being excluded from analysis by primary investigators. If the proportion of missing data is low and the reasons for missing data are similar across intervention groups, then the risk of bias is likely to be low. As the proportion of missing data rises, differences in treatment response between available and missing participants may increase the potential for bias.

6.- This bias (referred as detection bias) may be introduced if outcomes are misclassified or measured with error. Differential measurement errors (those related to intervention status) will bias the estimated effect of intervention-outcome relationship. Detection bias can arise when outcome assessors are aware of intervention status or if different methods are used to assess outcomes in different intervention groups, or if measurement errors are related to intervention status or effects (or to a confounder of the intervention-outcome relationship).

7.- Selective reporting will lead to bias if it is based on the direction, magnitude or statistical significance of intervention effect estimates. Selective outcome reporting occurs when an effect estimate for a particular outcome measurement is selected from among multiple measurements. Selective analysis reporting occurs when the reported results are selected from intervention effects estimated in multiple ways, or in the selection of a subgroup of participants, selected from a larger cohort, for which results are reported on the basis of a more interesting finding.

Sterne JAC, Hernán MA, Reeves BC, Savović J, Berkman ND, Viswanathan M, Henry D, et al. ROBINS-I: a tool for assessing risk of bias in non-randomized studies of interventions. BMJ 2016; 355; i4919; doi: 10.1136/bmj.i4919

Table S-4. Assessment of the certainty of the evidence on the primary and secondary outcomes according to the GRADE guidelines.

Outcome	Study design	Overall Risk of bias (ROBINS-I)	Inconsistency	Indirectness	Imprecision	Publication bias	Other considerations	Quality of the body of evidence
Carotid-Femoral Pulse Wave Velocity	Single cohort	Serious risk (low quality)	Not serious	Not serious	Not serious	Serious risk (-1) **	Upgrade + 1 Due to estimated effect sizes	**Low ⊕⊕**
	Cohort with controls (#)	Moderate risk (moderate quality)	Serious * (-1)	Not serious	Not serious	Not serious	Upgrade +1 Due to estimated effect sizes	**Moderate ⊕⊕⊕**
Systolic and diastolic blood pressure	Single cohort	Serious risk (low quality)	Not serious	Not serious	Not serious	Serious risk (-1) [&&&]	Upgrade + 1 Due to estimated effect sizes	**Low ⊕⊕**
	Cohort with controls (#)	Moderate risk (moderate quality)	Serious [&&] (-1)	Not serious	Serious (-1) [&]	Not serious	Upgrade +1 Due to estimated effect sizes	**Low ⊕⊕**
Glomerular Filtration rates	Single cohort	Serious risk (low quality)	Not serious	Not serious	Not serious	Not serious	Upgrade + 1 Due to estimated effect sizes	**Moderate ⊕⊕⊕**
	Cohort with controls (#)	Moderate risk (moderate quality)	Not serious	Not serious	Not serious	Not serious	Upgrade +1 Due to estimated effect sizes	**high ⊕⊕⊕⊕**

Considerations for this assessment [22]:

(#) Best evidence in non-randomized studies of living kidney donation as randomized studies are unethically to practice. The use of a comparator of healthy controls of comparable age enhances the confidence on the level of evidence.

* inconsistency rated as serious due to a consistent and significant heterogeneity between studies.

** small studies with short follow ups more likely to be associated with variable results.

*** over or under-estimation of effects due to selective publication (risk of overlapping in 3 studies)

*** upgraded due to narrow 95% CI and greater than 30% estimated effect size.

& imprecision rated as serious due to wide 95% CI.

&& inconsistency rated as serious due to presence of significant heterogeneity.

&&& publication bias rated as serious due to serious reporting bias (absence of pre-nephrectomy baseline in some studies)

Supplementary Figures and Figure legends:

S-1:Forest Plots of the Effect estimates for Glomerular Filtration rate (Panels A and B)

S-2: Forest Plots of the Effect estimates for Systolic Blood pressure (Panels A and B)

S-3: Forest Plots of the Effect estimates for Diastolic Blood pressure (Panels A and B)

S-4: Funnel plots for cf-PWV

Figure S1-a

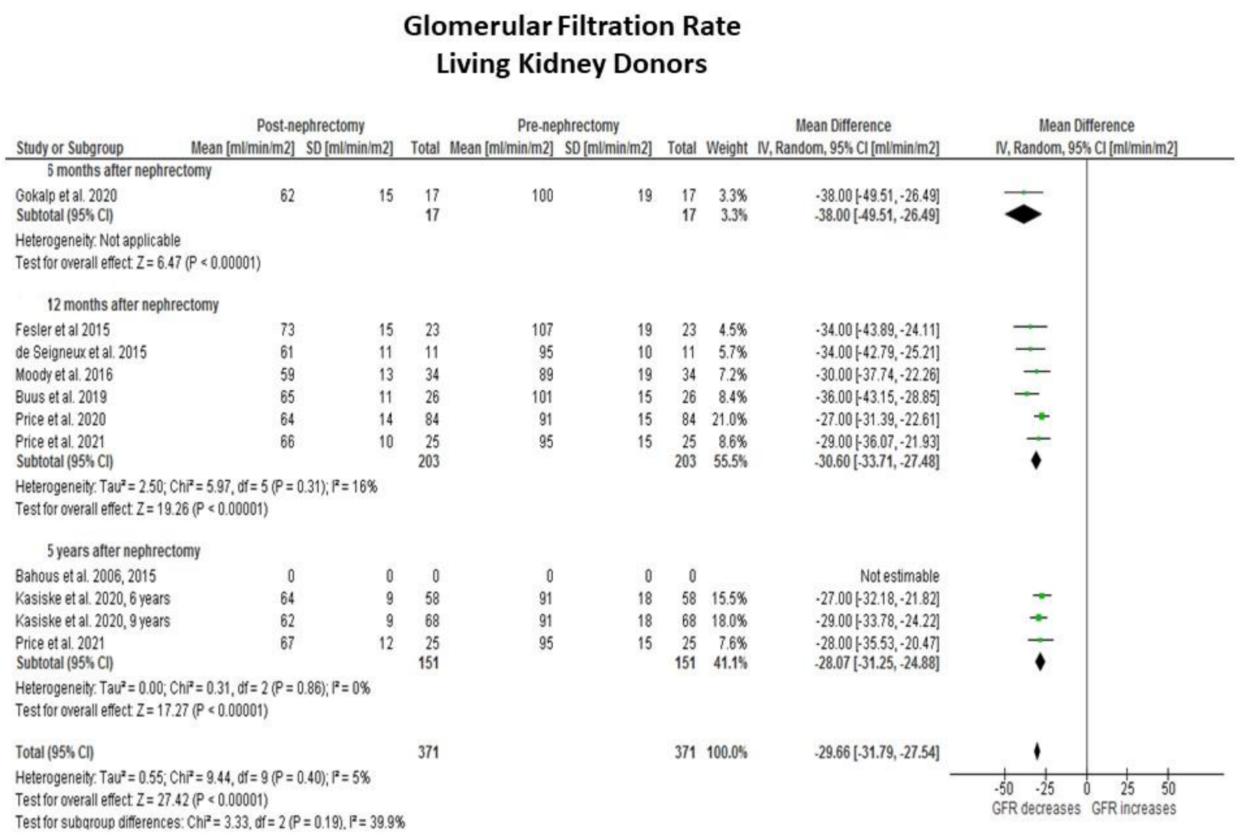
Glomerular Filtration Rate
Living Kidney Donors
Post-nephrectomy
Pre-nephrectomy
Mean Difference
Study or Subgroup Mean [ml/min/m2] SD [ml/min/m2] Total Mean [ml/min/m2] SD [ml/min/m2] Total Weight IV, Random, 95% CI [ml/min/m2]
Mean Difference IV, Random, 95% CI [ml/min/m2]
6 months after nephrectomy
Gokalp et al. 2020 62 15 17 100 19 17 3.3% -38.00 [-49.51, -26.49]
Subtotal (95% CI) 17 17 3.3% -38.00 [-49.51, -26.49]
Heterogeneity: Not applicable
Test for overall effect: Z = 6.47 (P < 0.00001)
12 months after nephrectomy
Fesler et al 2015 73 15 23 107 19 23 4.5% -34.00 [-43.89, -24.11]
de Seigneux et al. 2015 61 11 11 95 10 11 5.7% -34.00 [-42.79, -25.21]
Moody et al. 2016 59 13 34 89 19 34 7.2% -30.00 [-37.74, -22.26]
Buus et al. 2019 65 11 26 101 15 26 8.4% -36.00 [-43.15, -28.85]
Price et al. 2020 64 14 84 91 15 84 21.0% -27.00 [-31.39, -22.61]
Price et al. 2021 66 10 25 95 15 25 8.6% -29.00 [-36.07, -21.93]
Subtotal (95% CI) 203 203 55.5% -30.60 [-33.71, -27.48]
Heterogeneity: Tau² = 2.50; Chi² = 5.97, df = 5 (P = 0.31); I² = 16%
Test for overall effect: Z = 19.26 (P < 0.00001)
5 years after nephrectomy
Bahous et al. 2006, 2015 0 0 0 0 0 0 Not estimable
Kasiske et al. 2020, 6 years 64 9 58 91 18 58 15.5% -27.00 [-32.18, -21.82]
Kasiske et al. 2020, 9 years 62 9 68 91 18 68 18.0% -29.00 [-33.78, -24.22]
Price et al. 2021 67 12 25 95 15 25 7.6% -28.00 [-35.53, -20.47]
Subtotal (95% CI) 151 151 41.1% -28.07 [-31.25, -24.88]
Heterogeneity: Tau² = 0.00; Chi² = 0.31, df = 2 (P = 0.86); I² = 0%
Test for overall effect: Z = 17.27 (P < 0.00001)
Total (95% CI) 371 371 100.0% -29.66 [-31.79, -27.54]
Heterogeneity: Tau² = 0.55; Chi² = 9.44, df = 9 (P = 0.40); I² = 5%
Test for overall effect: Z = 27.42 (P < 0.00001)
Test for subgroup differences: Chi² = 3.33, df = 2 (P = 0.19), I² = 39.9%
-50 -25 0 25 50
GFR decreases GFR increases

Figure S1-b

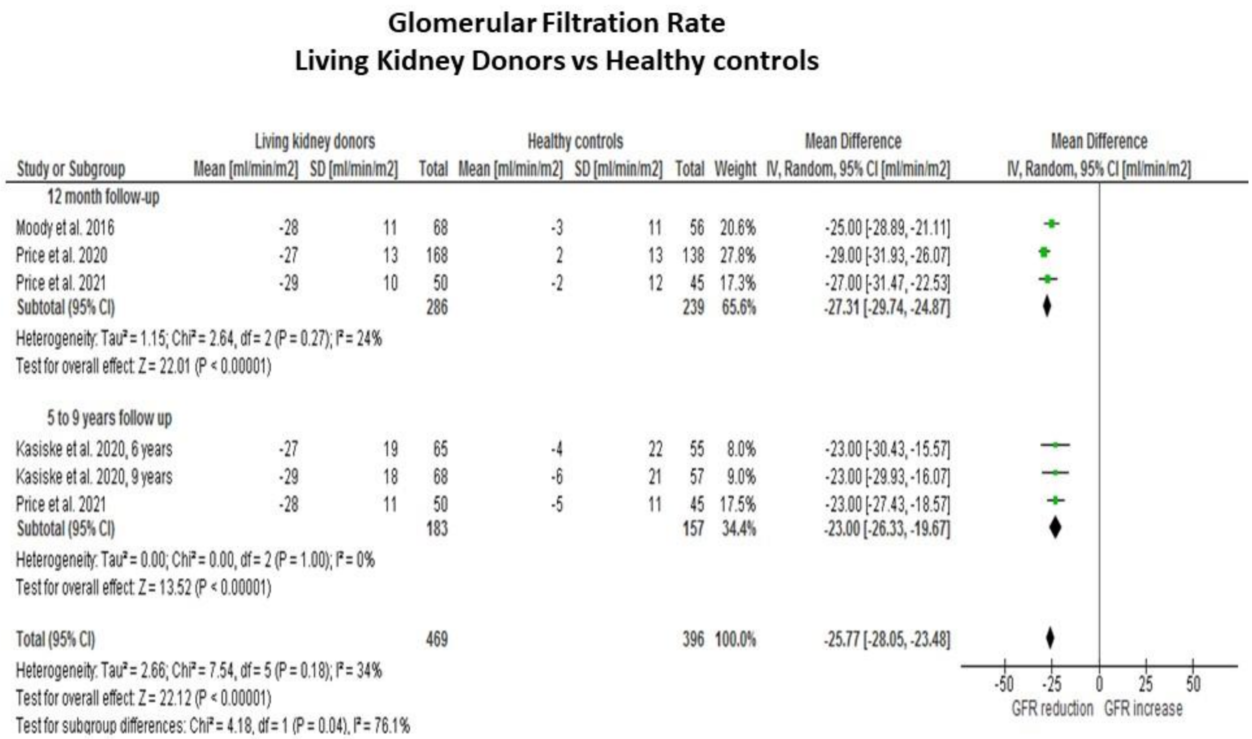

Figure S-1: Pooled effect estimates on Glomerular filtration rate (GFR) (ml/min/1.73 m^2) in living kidney donors from before to after nephrectomy (Panel "a") and on their differences relative to healthy comparators (Panel "b"). In all single cohort studies with before-and-after design (Panel "a") and in the study by Bahous et al. 2006 (Panel b), the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors during the analysis. NRR: non-recipient related, RR: recipient related.

Figure S2-a

Systolic Blood Pressure
Living Kidney Donors

Study or Subgroup	Post-nephrectomy Mean [mm Hg]	Post-nephrectomy SD [mm Hg]	Post-nephrectomy Total	Pre-nephrectomy Mean [mm Hg]	Pre-nephrectomy SD [mm Hg]	Pre-nephrectomy Total	Weight	Mean Difference IV, Random, 95% CI [mm Hg]
6 months after nephrectomy								
Gokalp et al. 2020	122	20	17	120	16	17	4.2%	2.00 [-10.18, 14.18]
Subtotal (95% CI)			**17**			**17**	**4.2%**	**2.00 [-10.18, 14.18]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 0.32 (P = 0.75)								
12 months after nephrectomy								
Fesler et al 2015	122	13	23	122	12	23	8.4%	0.00 [-7.23, 7.23]
Moody et al. 2016	122	12	34	121	8	34	12.1%	1.00 [-3.85, 5.85]
Buus et al. 2019	119	13	26	120	14	26	8.3%	-1.00 [-8.34, 6.34]
Price et al. 2020	124	10	60	124	10	60	14.4%	0.00 [-3.58, 3.58]
Price et al. 2021	121	15	25	121	9	25	8.9%	0.00 [-6.86, 6.86]
Subtotal (95% CI)			**168**			**168**	**52.1%**	**0.13 [-2.23, 2.49]**
Heterogeneity: Tau² = 0.00; Chi² = 0.22, df = 4 (P = 0.99); I² = 0%								
Test for overall effect: Z = 0.11 (P = 0.91)								
5 to 9 years after nephrectomy								
Bahous et al. 2006, 2015	130	20	50	114	18	50	8.1%	16.00 [8.54, 23.46]
Kasiske et al. 2020, 6 years	118	11	58	120	14	58	12.6%	-2.00 [-6.58, 2.58]
Kasiske et al. 2020, 9 years	117	13	68	120	14	68	12.6%	-3.00 [-7.54, 1.54]
Price et al. 2021	124	12	25	121	9	25	10.4%	3.00 [-2.88, 8.88]
Subtotal (95% CI)			**201**			**201**	**43.7%**	**3.00 [-4.13, 10.14]**
Heterogeneity: Tau² = 44.67; Chi² = 20.61, df = 3 (P = 0.0001); I² = 85%								
Test for overall effect: Z = 0.82 (P = 0.41)								
Total (95% CI)			**386**			**386**	**100.0%**	**1.10 [-1.72, 3.93]**
Heterogeneity: Tau² = 11.07; Chi² = 21.08, df = 9 (P = 0.01); I² = 57%								
Test for overall effect: Z = 0.77 (P = 0.44)								
Test for subgroup differences: Chi² = 0.62, df = 2 (P = 0.73), I² = 0%								

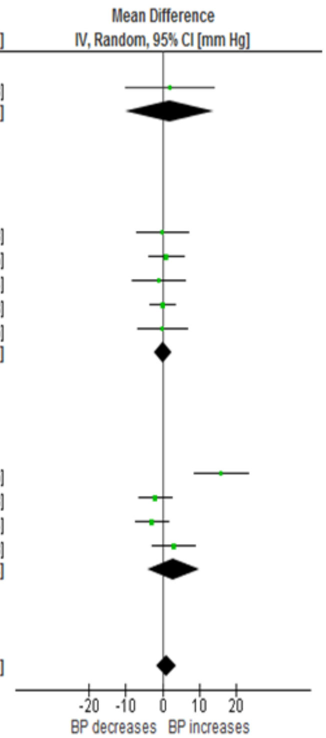

Figure S2-b

Systolic Blood Pressure
Living Kidney Donors vs Healthy controls

Study or Subgroup	Living kidney donors Mean [mm Hg]	SD [mm Hg]	Total	Healthy controls Mean [mm Hg]	SD [mm Hg]	Total	Weight	Mean Difference IV, Random, 95% CI [mm Hg]
12 month follow up								
Moody et al. 2016	1	12.9	46	-0.5	6.1	36	12.4%	1.50 [-2.73, 5.73]
Price et al. 2020	0.1	12	119	0.6	8	111	19.3%	-0.50 [-3.12, 2.12]
Price et al. 2021	0.2	15	50	-1.25	8	45	10.7%	1.45 [-3.32, 6.22]
Subtotal (95% CI)			215			192	42.3%	0.30 [-1.71, 2.32]
Heterogeneity: Tau² = 0.00; Chi² = 0.89, df = 2 (P = 0.64); I² = 0%								
Test for overall effect: Z = 0.30 (P = 0.77)								
5 to 9 years follow-up								
Bahous et al. 2006, NRR	130	20	51	125	10	134	8.3%	5.00 [-0.74, 10.74]
Bahous et al. 2006, RR	130	20	51	123	15	129	7.6%	7.00 [0.93, 13.07]
Kasiske et al. 2020, 6 years	118	11	128	119	14	107	16.2%	-1.00 [-4.27, 2.27]
Kasiske et al. 2020, 9 years	117	13	136	120	15	114	15.1%	-3.00 [-6.52, 0.52]
Price et al. 2021	2.57	12	50	0.66	12	45	10.5%	1.91 [-2.92, 6.74]
Subtotal (95% CI)			416			529	57.7%	1.36 [-2.06, 4.78]
Heterogeneity: Tau² = 9.68; Chi² = 11.67, df = 4 (P = 0.02); I² = 66%								
Test for overall effect: Z = 0.78 (P = 0.44)								
Total (95% CI)			631			721	100.0%	0.78 [-1.17, 2.73]
Heterogeneity: Tau² = 3.34; Chi² = 12.57, df = 7 (P = 0.08); I² = 44%								
Test for overall effect: Z = 0.78 (P = 0.43)								
Test for subgroup differences: Chi² = 0.27, df = 1 (P = 0.60), I² = 0%								

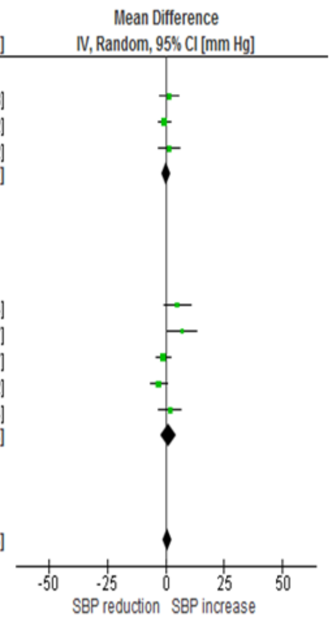

Figure S-2: Pooled effect estimates on Systolic Blood pressure (mm Hg) in living kidney donors from before to after nephrectomy (Panel "a") and on their differences relative to healthy comparators (Panel "b"). In all single cohort studies with before-and-after design (Panel "a") and in the study by Bahous et al. 2006 (Panel "b"), the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors during the analysis. NRR: non-recipient related, RR: recipient related.

Figure S-3-a

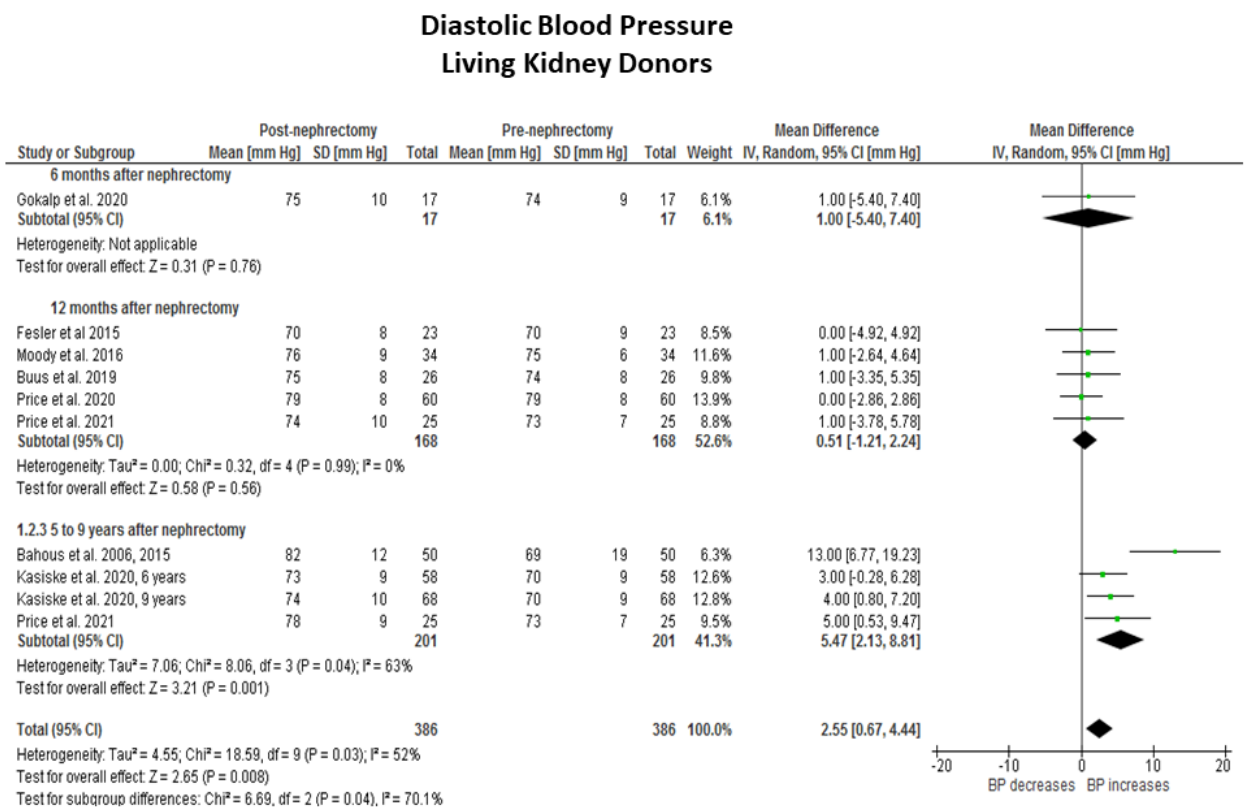
Diastolic Blood Pressure
Living Kidney Donors
Post-nephrectomy
Pre-nephrectomy
Mean Difference
Mean Difference
Study or Subgroup Mean [mm Hg] SD [mm Hg] Total Mean [mm Hg] SD [mm Hg] Total Weight IV, Random, 95% CI [mm Hg]
IV, Random, 95% CI [mm Hg]
6 months after nephrectomy
Gokalp et al. 2020 75 10 17 74 9 17 6.1% 1.00 [-5.40, 7.40]
Subtotal (95% CI) 17 17 6.1% 1.00 [-5.40, 7.40]
Heterogeneity: Not applicable
Test for overall effect: Z = 0.31 (P = 0.76)
12 months after nephrectomy
Fesler et al 2015 70 8 23 70 9 23 8.5% 0.00 [-4.92, 4.92]
Moody et al. 2016 76 9 34 75 6 34 11.6% 1.00 [-2.64, 4.64]
Buus et al. 2019 75 8 26 74 8 26 9.8% 1.00 [-3.35, 5.35]
Price et al. 2020 79 8 60 79 8 60 13.9% 0.00 [-2.86, 2.86]
Price et al. 2021 74 10 25 73 7 25 8.8% 1.00 [-3.78, 5.78]
Subtotal (95% CI) 168 168 52.6% 0.51 [-1.21, 2.24]
Heterogeneity: Tau² = 0.00; Chi² = 0.32, df = 4 (P = 0.99); I² = 0%
Test for overall effect: Z = 0.58 (P = 0.56)
1.2.3 5 to 9 years after nephrectomy
Bahous et al. 2006, 2015 82 12 50 69 19 50 6.3% 13.00 [6.77, 19.23]
Kasiske et al. 2020, 6 years 73 9 58 70 9 58 12.6% 3.00 [-0.28, 6.28]
Kasiske et al. 2020, 9 years 74 10 68 70 9 68 12.8% 4.00 [0.80, 7.20]
Price et al. 2021 78 9 25 73 7 25 9.5% 5.00 [0.53, 9.47]
Subtotal (95% CI) 201 201 41.3% 5.47 [2.13, 8.81]
Heterogeneity: Tau² = 7.06; Chi² = 8.06, df = 3 (P = 0.04); I² = 63%
Test for overall effect: Z = 3.21 (P = 0.001)
Total (95% CI) 386 386 100.0% 2.55 [0.67, 4.44]
Heterogeneity: Tau² = 4.55; Chi² = 18.59, df = 9 (P = 0.03); I² = 52%
Test for overall effect: Z = 2.65 (P = 0.008)
Test for subgroup differences: Chi² = 6.69, df = 2 (P = 0.04), I² = 70.1%
-20 -10 0 10 20
BP decreases BP increases

Figure S-3-b

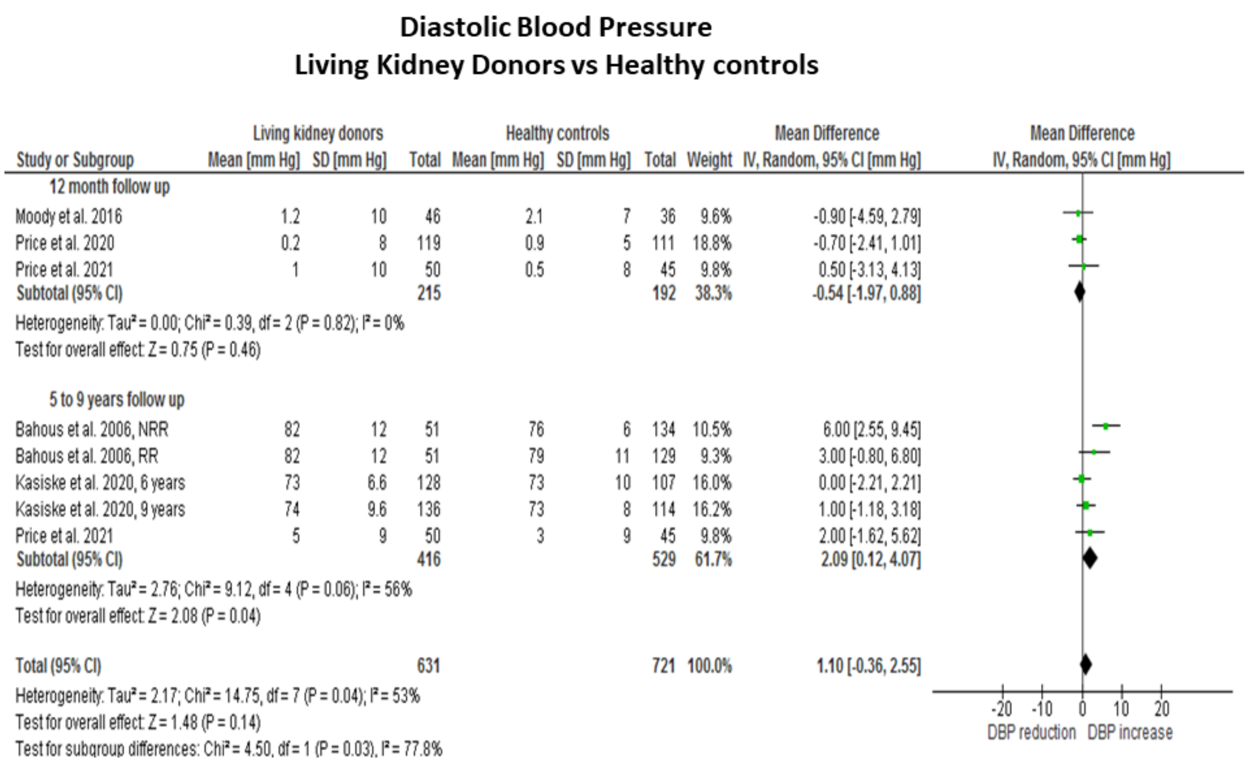

Figure S-3: Pooled effect estimates on Diastolic Blood pressure (mm Hg) in living kidney donors from before to after nephrectomy (Panel "a") and on their differences relative to healthy comparators (Panel "b"). In all single cohort studies with before-and-after design (Panel "a") and in the study by Bahous et al. 2006 (Panel "b"), the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors during the analysis. NRR: non-recipient related, RR: recipient related.

Figure S-4

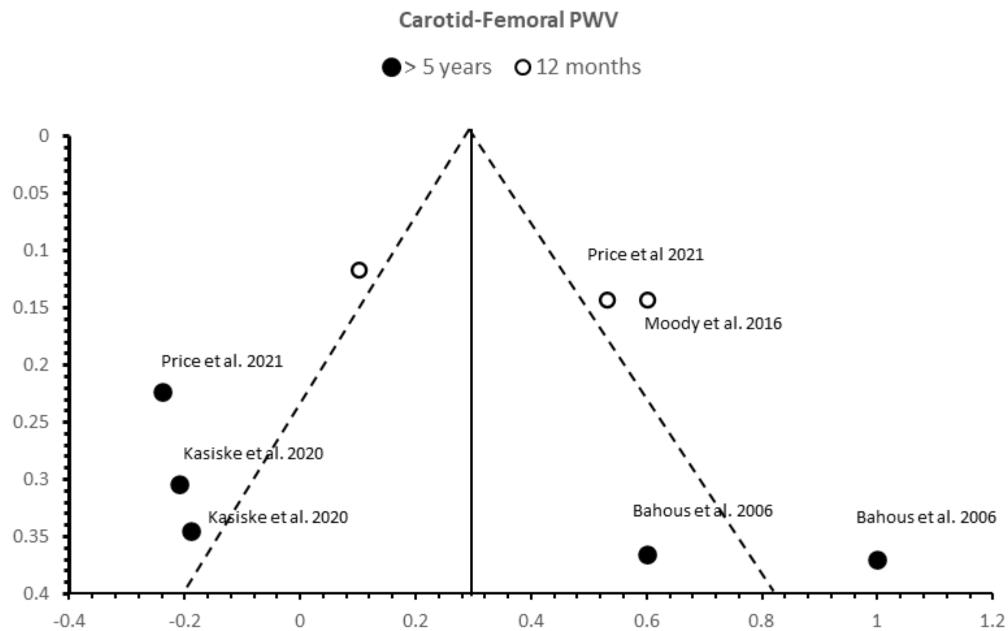

Figure S-4. Funnel plots of asymmetry in 5 studies who evaluated living kidney donors and controls. Data is stratified by time of follow up (12 months and > 5 years). Vertical black line represents the mean difference of the pooled effect estimates between kidney donors and controls (0.3 m/s). Dotted lines represent the 95% CI of the effect estimates. Filled circles represent studies > 5 years while non-filled circles those with measurements at 12 months.

Appendices

Appendix 1: Search strategy

Appendix 2: List of inclusion and exclusion criteria

Appendix 3: Summary of data extraction themes

Appendix 4: Complementary summary of study characteristics and country of origin.

Appendix 1. Search strategy.

Database: Embase Classic+Embase **<1947 to 2022 Dec 06>**, Ovid MEDLINE(R) ALL <1946 to Dec 06, 2022>, EBM Reviews - Cochrane Central Register of Controlled Trials <February 2020>
Search Strategy:
----
1 Nephrectomy/ (88255)
2 nephrectom*.tw,kw. (96086)
3 Kidney Transplantation/ and Living Donors/ (14605)
4 ((renal or kidney) adj3 (donor* or donation*)).tw. (32914)
5 or/1-4 (161070)
6 Vascular Stiffness/ (25452)
7 ((vascular or arter* or aort*) adj3 (stiff* or rigid*)).tw. (39922)
8 stiffness.tw,kw. (169270)
9 exp Pulse Wave Analysis/ (30543)
10 Cardio Ankle Vascular Index.tw,kw. (1710)
11 Augmentation index.tw,kw. (10033)
12 central pulse pressure.tw,kw. (1410)
13 Ankle-brachial index.tw,kw. (12896)
14 Ankle Brachial Index/ (13675)
15 (aort* adj2 (distensibilit* or elasticit*)).tw. (2677)
16 ((pulse or pulsation) adj2 (curve* or tracing* or wave*)).tw. (42852)
17 pulse wave.kw. (5300)
18 (pwv or apwv or bapwv or cfpwv).tw,kw. (20707)
19 (pulse adj2 (analys#s or velocit* or transit time)).tw. (37462)
20 (vascular stiff* or aortic stiff* or arter* stiff).kw. (1790)
21 ((decreased or reduced or diminished or lessened or lowered) adj3 ((vascular or aortic or arter*) adj compliance)).tw. (1257)
22 blood flow velocity/ (101381)
23 ((blood or circulation) adj2 (flow or rate) adj velocit*).tw. (21273)
24 (central adj (pulse or aortic or arterial) adj pressure).tw. (2881)
25 (central pressure or pulse pressure or pulse tension).tw,kw. (25275)
26 (central pulse pressure or blood flow velocit*).kw. (3437)
27 aasi.tw,kw. (560)
28 applanation tonomet*.tw,kw. (10330)
29 (sphygmocor* or vicorder*).tw,kw. (3318)
30 ((assess* or measur* or determin* or evaluat*) adj3 ((vascular or aortic or arter*) adj elasticit*)).tw. (656)
31 (Carotid adj3 intima-media thickness).tw. (23970)
32 Carotid-intima media thickness.kw. (2894)
33 Carotid artery ultrasonography.tw,kw. (292)
34 Carotid Arteries/dg (8914)
35 (Carotid arter* adj3 ultrasonograph*).tw. (1410)
36 (Ultrasonography, Doppler/ or Ultrasonography/) and Carotid Artery Diseases/ (4100)
37 calcinosis/ or exp vascular calcification/ (77074)
38 Vascular calcification.tw,kw. (11072)

39 vascular calcinosis.tw,kw. (40)
40 Flow mediated dilatation.tw,kw. (5904)
41 Brachial Artery/ and Vasodilation/ (6138)
42 brachial artery reactivity.tw,kw. (483)
43 or/6-42 (467264)
44 5 and 43 (1078)
45 exp animals/ not exp humans/ (10091587)
46 44 not 45 (692)
47 46 use medall (302) Medline
48 46 use cctr (13) Cochrane
49 nephrectomy/ or radical nephrectomy/ (91023)
50 nephrectom*.tw. (93900)
51 kidney donor/ (11405)
52 living donor/ and kidney transplantation/ (16482)
53 ((renal or kidney) adj3 (donor* or donation*)).tw. (32914)
54 49 or 50 or 51 or 52 or 53 (165157)
55 arterial stiffness/ (27294)
56 ((vascular or arter* or aort*) adj3 (stiff* or rigid*)).tw. (39922)
57 stiffness.tw. (166817)
58 pulse wave/ (26256)
59 Cardio Ankle Vascular Index.tw. (1685)
60 augmentation index/ (5503)
61 Augmentation index.tw. (9834)
62 Augmentation index.tw. (9834)
63 pulse pressure/ (313849)
64 central pulse pressure.tw. (1389)
65 ankle brachial index/ (13675)
66 Ankle-brachial index.tw. (12479)
67 (aort* adj2 (distensibilit* or elasticit*)).tw. (2677)
68 ((pulse or pulsation) adj2 (curve* or tracing* or wave*)).tw. (42852)
69 (pwv or apwv or bapwv or cfpwv).tw. (20649)
70 (pulse adj2 (analys#s or velocit* or transit time)).tw. (37462)
71 ((decreased or reduced or diminished or lessened or lowered) adj3 ((vascular or aortic or arter*) adj compliance)).tw. (1257)
72 blood flow velocity/ (101381)
73 ((blood or circulation) adj2 (flow or rate) adj velocit*).tw. (21273)
74 (central adj (pulse or aortic or arterial) adj pressure).tw. (2881)
75 (central pressure or pulse pressure or pulse tension).tw. (24926)
76 aasi.tw. (558)
77 applanation tonomet*.tw. (10187)
78 (sphygmocor* or vicorder*).tw. (3310)
79 ((assess* or measur* or determin* or evaluat*) adj3 ((vascular or aortic or arter*) adj elasticit*)).tw. (656)
80 arterial wall thickness/ (21793)
81 (Carotid adj3 intima-media thickness).tw. (23970)
82 (Carotid arter* adj3 ultrasonograph*).tw. (1410)

83 exp blood vessel calcification/ (19784)
84 vascular calcinosis.tw. (36)
85 Vascular calcification.tw. (10295)
86 dilatation/ and brachial artery/ (846)
87 Flow mediated dilatation.tw. (5753)
88 brachial artery reactivity.tw. (471)
89 or/55-88 (683344)
90 54 and 89 (3439)
91 exp animals/ not exp humans/ (10091587)
92 90 not 91 (1320)
93 92 use emczd (342) Embase
94 47 or 48 or 93 (657)
95 remove duplicates from 94 (524)
96 95 use medall (300) Medline
97 95 use emczd (221) Embase
98 95 use cctr (3) Cochrane

Appendix 2. List of inclusion and exclusion criteria.

PICO definitions	Inclusion criteria	Exclusion criteria
Population	Healthy subjects (≥18 years) that met standard criteria for kidney donation, who underwent unilateral simple nephrectomy and consented to measurements of carotid-femoral PWV before and/or after nephrectomy.	• Healthy subjects that underwent unilateral simple nephrectomy for other reason than kidney donation. • Children and adolescents with solitary kidney after unilateral nephrectomy.
Intervention	Open or laparoscopic unilateral simple nephrectomy	• Unilateral nephrectomy combined to other surgical procedures
Comparator	• Healthy adult subjects (≥18 years) with measurements of carotid-femoral PWV who participated as healthy comparative controls in kidney donor studies. • Healthy subjects from the general population with measurements of carotid-femoral PWV included in reference studies.	• Kidney recipients
Outcome	• Changes in carotid-femoral PWV	• Other indices of vascular stiffness (augmentation index, carotid-radial PWV, brachial-ankle PWV, cardio-ankle vascular index, carotid-intima media thickness, calcification index)
Study design	• Prospective non-randomised (cohort, case–control, case series and before-and-after studies) and retrospective studies if 10 or more participants have been included in the primary analysis. • Articles reported in English, French, Italian, Portuguese and Spanish languages.	• Paediatric and non-human studies • Narrative reviews • In vitro or mathematical modelling reports. • Duplicates • Sub-studies of previously published trials.

PWV: pulse wave velocity

Appendix 3. Summary of data extraction themes.

The process of data extraction included the following themes:

a) ***Study characteristics*** included authors, country of origin, publication date, title, language of publication, study design, inclusion and exclusion criteria, pre-and post-nephrectomy time measurement points, duration of follow-up, study design, use of a control group and individual study inclusion and exclusion criteria.

b) ***Characteristics of participants*** including sample size for donors and controls, proportion of female and males, donor's age at the time of nephrectomy and testing, control's age at the time of initial testing (i.e., recruitment) and at follow up, body mass index for donors and controls. If available, we documented the participant's clinical history (donors and controls) including the proportion of subjects with hypertension, cardiovascular disease, diabetes, hypercholesterolemia, obesity, history of cancer, smoking, as well as the proportion of subjects receiving antihypertensive therapy and type of medication [i.e. Angiotensin converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARB), beta or alpha blockers, calcium channel blockers, diuretics, statins and aspirin]. In addition, if accessible, we estimated the proportion of recipient-related and non-recipient related donors and controls, and the ethnicity of participants.

c) ***Renal chemistry profile*** including: plasma glucose, plasma creatinine, calculated creatinine clearance (MDRD or CKD-EPI), urinary albumin/creatinine and blood urea nitrogen in kidney donors (before and after nephrectomy) and healthy controls (recruitment and follow-up).

d) ***Carotid-Femoral PWV (cf-PWV)*** including instrumentation and technique of measurement, absolute values and post-donation changes relative to their pre-donation baseline, adjusted or non-adjusted values and type of adjusting factor (i.e., mean arterial pressure, heart rate).

e) **H*emodynamic characteristics*** including systolic, diastolic and mean blood pressure, heart rate and pulse pressure, techniques of measurement (i.e., office, 24 hours monitoring); absolute values and post-donation changes relative to pre-donation baseline.

Appendix 4. Complementary summary of study characteristics and country of origin.

Most studies (n=7) were completed in Europe, one in the USA and another in the Middle East. All studies reported that the process of screening and selection for kidney donors followed institutional protocols. In 5 studies, participants in the control group were screened as if they would be fit for kidney donation, but they were not actual donors.[6-12] Two of these studies [6,7,10] reported that 90.0% and 21.2% of healthy controls, respectively, were first-degree relatives of recipients, but only one study[10] documented that 51.5% of donors were biologically related to the recipients. Only one study provided information on clinical outcomes [6,7] and reported that 4.9% (5/101) of donors developed at least one adverse cardiovascular event (coronary, cerebral, aortic or peripheral artery disease) after nephrectomy (follow-up range: 43 to 219 months).

BMJ Open

Aortic stiffness after living kidney donation: A Systematic Review and Meta-analysis

Journal:	*BMJ Open*
Manuscript ID	bmjopen-2023-082725.R1
Article Type:	Original research
Date Submitted by the Author:	25-Sep-2024
Complete List of Authors:	Rodriguez, Rosendo; Ottawa Hospital, Department of Medicine McNeill, Kylie; The Ottawa Hospital, Department of Medicine Agharazii, Mohsen; CHU de Québec-Université Laval, Nephrology Bugeja, Ann; The Ottawa Hospital, Nephrology; The Ottawa Hospital, Department of Medicine Clark, Edward; The Ottawa Hospital, Nephrology; The Ottawa Hospital, Department of Medicine Burns, Kevin; The Ottawa Hospital, Nephrology; University of Ottawa, Kidney Research Centre
Primary Subject Heading:	Renal medicine
Secondary Subject Heading:	Cardiovascular medicine, Surgery, Medical management
Keywords:	Cardiovascular Disease, Renal transplantation < NEPHROLOGY, TRANSPLANT MEDICINE, End stage renal failure < NEPHROLOGY

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Aortic stiffness after living kidney donation:

A Systematic Review and Meta-analysis

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Running title: *Aortic stiffness in living kidney donors*

Total word count: 5,267 words

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Abstract

Objectives: Increased aortic stiffness measured with carotid-femoral pulse wave velocity (cf-PWV) has been associated with adverse cardiovascular outcomes. Some studies have reported increased cf-PWV in living kidney donors after nephrectomy. This review aimed to determine the effects of living kidney donation on cf-PWV, glomerular filtration rate (GFR), systolic (SBP), diastolic blood pressure (DBP) and their differences versus non-nephrectomized healthy individuals.

Design: Systematic review and Meta-analysis.

Data sources: Electronic databases (MEDLINE, EMBASE, Cochrane Central databases, Cochrane Register of controlled trials, Cochrane Methodology Register, Health Technology Database, Technologies in Health, EBM Reviews and “Grey Matters Light”).

Eligibility criteria: We searched for studies that measured cf-PWV in living kidney donors before and/or after nephrectomy. Non-nephrectomized healthy individuals included as controls were the comparators. Studies that provided age-adjusted cf-PWV reference values in normotensive healthy individuals were also included.

Outcome measures: The mean differences in cf-PWV, GFR, and BP before and after nephrectomy and their mean differences versus non-nephrectomized healthy comparators. We also explored differences in yearly-adjusted cf-PWV changes between donors and normotensive healthy individuals.

Data extraction/synthesis: Two independent reviewers extracted data and assessed risk of bias (ROBINS-I) and quality of evidence (GRADE). Pooled effect estimates were calculated using the inverse variance method and analyzed with random effect models.

Results: Nine interventional (652 donors; 602 controls) and 6 reference studies (6,278 individuals) were included. cf-PWV increased at 1-year post-donation (p=0.03) and was on average 0.4 m/s (95% CI: 0.07; 0.60) higher than in healthy controls (p=0.01). These differences were non-significant 5 years post-nephrectomy (p=0.54). GFR decreased after nephrectomy (p<0.001) and remained reduced compared to healthy controls (p<0.001), but SBP and DBP were not significantly different (p≥0.14). Yearly changes in cf-PWV post-nephrectomy were similar to age-adjusted reference values in healthy normotensive individuals (p=0.76).

Conclusions: Aortic stiffness increases independent of BP one year after kidney donation, but the long-term effects seem minimal. These findings may impact future consent of prospective living kidney donors.

PROSPERO Registration number: CRD42020185551.

Key words: *living kidney donors, aortic stiffness, cardiovascular disease, pulse wave velocity, nephrectomy.*

Strengths and limitations of this study

⇒ Comprehensive systematic review using multiple electronic databases and rigorous assessment of study quality and certainty of the evidence.

⇒ The Risk of Bias tool for non-Randomized studies (ROBINS-I) was used to independently evaluate risk of bias and study quality.

⇒ The inclusion of 2 comparator groups of non-nephrectomized healthy controls was used to assess changes in carotid-femoral pulse wave velocity in living kidney donors after donation.

⇒ The inclusion of blood pressure as outcome permitted to assess if changes in carotid femoral pulse wave velocity were dependent on changes in blood pressure.

⇒ The study was limited by the small number of studies and the paucity of well-designed cohort studies with long-term follow ups.

Introduction

Living kidney donors (LKD) are exposed to perioperative and long-term risks, including potential adverse effects on kidney health.[1] Although kidney hypertrophy is a recognized physiologic response to unilateral nephrectomy, LKD ultimately lose on average 30% of their pre-donation total glomerular filtration rate (GFR).[1,2] Although this reduction in GFR may be of concern to donors and clinicians,[3] the absolute risk increase for kidney failure, cardiovascular disease or death after donation is small and even lower than in the general population.[2,4-5]

Carotid-femoral Pulse wave velocity (cf-PWV) is a surrogate of the intrinsic stiffness of the arterial wall and has been reported highly predictive of cardiovascular events in high risk populations.[6,7] The prognostic value of cf-PWV has been associated to the integrated measure of the impact of cardiovascular risk factors on the arterial wall and to the adverse hemodynamic effect of aortic stiffness.[6-8] Recently, several prospective studies involving measurements of cf-PWV have documented that LKD have increased aortic stiffness after nephrectomy when compared to healthy controls of similar age.[9-15] Although most of these investigations involved small samples and limited follow-up times,[16-17] these findings are relevant since increased cf-PWV is associated with adverse cardiovascular outcomes and all-cause mortality in the general population.[18] Since most of these studies did not detect increases in systemic blood pressure (BP) post-nephrectomy,[17] a reduction in GFR may be an independent graded risk factor for cardiovascular remodeling in LKD.[19] Moreover, this phenomenon may be particularly important for young LKD who have the longest risk exposure to the effects of reduced kidney mass.

To determine the effects of living kidney donation on aortic stiffness and their differences relative to non-nephrectomized healthy individuals, we conducted a systematic review and meta-analysis to evaluate the progression of cf-PWV, changes in arterial BP and GFR in LKD before and after nephrectomy. We also gathered data on differences in cf-PWV, BP and GFR between LKD and their non-nephrectomized healthy comparators. Finally, we explored whether yearly changes in aortic stiffness in LKD determined by cf-PWV, differed from age-adjusted reference values in normotensive healthy individuals. We hypothesized that living kidney donation would decrease kidney function and increase aortic stiffness and arterial blood pressure compared to non-nephrectomized healthy individuals.

Materials and Methods

The review was conducted in accordance with the Cochrane Collaboration Methods, Systematic Reviews standards, and reported according to PRISMA guidelines.[20] The study protocol has been published[21] and registered in PROSPERO (CRD42020185551) (www.crd.york.ac.uk/prospero).[22] The Preferred Reporting items for systematic Reviews and Meta-Analysis guidelines were followed and a checklist file is included.[23]

Data Sources, searching criteria and eligibility

We conducted a comprehensive search (Appendix 1) to retrieve all observational studies published to December 2022 that included healthy individuals participating in a kidney donation program who underwent measurements of cf-PWV before and/or after nephrectomy. Our initial search during protocol registration was undertaken until December 2020 and it was subsequently updated until March 2021 at the time of protocol publication.[21] The broad nature of our original search captured studies with additional metrics of arterial stiffness.[21] However, these secondary outcomes were not considered in this review as we focused on cf-PWV. The search was applied to several electronic databases including MEDLINE, EMBASE, Cochrane Central databases, Cochrane Register of controlled trials, Cochrane Methodology Register, Health Technology Database, Technologies in Health, and EBM Reviews. EMBASE, MEDLINE, EBM reviews were searched through the OVID platform and the Cochrane Register searched via EBM. We searched for grey literature through the "Grey Matters Light" platform from the Canadian Agency for Drugs and Technology in Health (CADTH) and the ProQuest website for dissertations and theses. We also searched for studies that included cf-PWV in healthy individuals from the general population that evaluated age effects and aortic stiffness. Population-based studies were searched using the following key words and filters: aortic stiffness, arterial stiffness, cf-PWV, PWV, age, adults, humans, reference or normal values, healthy participants or subjects, and normal volunteers. There were no language restrictions in the initial search although during screening only studies published in English, French, Spanish, Portuguese, and Italian were included. We also identified data sources from manual searches of references in some relevant citations. All search results were downloaded into an Excel spreadsheet and screened by title and authors to remove duplicates. Ethical approval was not required since our study did not involve participation of human subjects.

Study inclusion and exclusion criteria

Our target population included healthy adult individuals (>18 years of age) who met standard institutional kidney donation criteria and had aortic stiffness evaluated with cf-PWV before and/or after nephrectomy. Non-nephrectomized healthy individuals included as healthy controls within the same study were used as comparators. Since prospective randomized clinical trials of kidney donation would never be possible for ethical reasons, we included prospective non-randomized (cohort, case-control, case series, before-and-after) and retrospective studies, provided that ≥ 10 subjects per study were enrolled.

Outcomes

The primary outcomes were the mean differences in cf-PWV before and after nephrectomy in LKD, and the mean differences versus their non-nephrectomized healthy comparators. Secondary outcomes were the pre- and post-donation mean differences in systolic and diastolic BPs and GFRs in LKD and the mean differences versus their non-nephrectomized healthy comparators. Exploratory outcomes were the differences in the yearly-adjusted changes in cf-PWV between LKD and a group of normotensive healthy individuals who participated in population-based studies of aortic stiffness.

Screening and study selection

Two independent reviewers screened abstracts and titles. We excluded non-human, *in-vitro* or modeling studies, narrative/systematic reviews, pediatric investigations, and letters to the editor. After screening was completed, reviewers examined the study methods to confirm that cf-PWV measurements were performed with validated automatic devices. The selected studies underwent full text review by two independent reviewers according to pre-defined inclusion and exclusion criteria (Appendix 2). In case of disagreement, a third reviewer was available to achieve consensus by discussion. We also screened for studies that included healthy individuals from the general population where age-adjusted values for cf-PWV were reported (reference studies). The 2 reviewers selected those studies that explicitly included healthy normotensive individuals (>18 years) with no history of cancer, cardiovascular, neurologic, inflammatory, or kidney disease. To clarify missing information, we contacted study authors by electronic mail. We declared a null response if no reply was obtained after three e-mail attempts within a 4-month period.

Data extraction

A data extraction form was prepared *a priori* from consensus amongst investigators and piloted for optimization. Two reviewers independently performed full data extraction (Appendix 3). Published secondary analyses associated with an original study were considered part of a single study.

Study quality

The risk of bias was assessed using the Risk of Bias tool in non-Randomized studies (ROBINS-I) and each study was independently evaluated by 2 reviewers according to seven domains including confounding, selection, classification of the intervention, deviation from intended intervention, missing data, outcome measurement and reporting.[24] Each reviewer classified the risk of bias for each domain as low, moderate, serious, critical or no information available. A final consensus produced an overall risk of bias for each study. Since the purpose of including reference studies was to provide normative values, their study quality was not assessed.

Certainty of the evidence

Quality of the certainty of the evidence was evaluated according to the 5 domains of the GRADE recommendations, and the overall assessment was reported as very low, low, moderate or high.[25]

Statistical analyses

Meta-analysis

The weighted mean differences and their 95% confidence intervals (95% CI) were calculated using the reported means and standard deviations (SD) from each study. In cases where different measures of central tendency (i.e., median) and distribution (i.e., inter-quartile) were reported, means and SD were estimated according to the algorithms described by Luo et al.[26] For studies [9,13] that did not include pre-donation values, post-donation differences between LKD and healthy controls were estimated using the mean absolute cf-PWV. To determine the level of skewness in small sample size studies (n <35), we subtracted the extreme value of the reported range or quartile distribution from the estimated means calculated by the Luo et al's method[26] and divided by the estimated standard deviation according to Altman and Bland.[27] Only cases with a ratio less than 1 (suggesting severe skewness) were log transformed. To explore statistical heterogeneity between studies, the Q test and the I^2 statistic was used (with a value of I^2 >65 considered to be a highly important heterogeneity). To find potential sources of heterogeneity, we stratified studies by sub-groups according to the duration of follow up and study design. Sensitivity analyses included examination of effect model, parameter estimates and methodological quality. If suitable, the pooled effect estimates were calculated using the method of the inverse variance and data was modeled according to the DerSimonian-Laird Method (random effects model) (p<0.05). To minimize the risk of artificially increasing the precision on the effect estimates due to counting the same patient twice in before-and-after studies ("double counting" error),[28] we reduced by 50% the number of study participants for each measurement.[29] To determine the strength of this approach, a sensitivity analysis between the models with and without adjustment was performed. Inter-group differences were analyzed using the Cochrane Q test with p value less than 0.10. Publication bias was investigated by Funnel plots, and asymmetry was evaluated if the number of studies in the meta-analysis was greater than 10. All meta-analyses utilized RevMan 5.4 (Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014).

Reference studies

Yearly changes in cf-PWV (m/s/year) for kidney donors and healthy controls were estimated using the mean differences between pre- and post-donation values divided by the number of years of observation. In reference studies, the yearly changes in cf-PWV (m/s/year) were estimated according to the age-decade average differences reported at the 90-to-97.5 percentile of the distribution. This cutoff would ensure that the area under the normal curve would fall within 1.282 to 1.960 SD from the mean cf-PWV for each decade. If this data was not available, we used the beta coefficient of the age and cf-PWV regression function. The significance of between-group comparisons was assessed by independent t-tests (2-tailed) ($p<0.05$). The differences in cf-PWV are reported as the means and their 95% CI (or their SD, if noted), while for absolute cf-PWV values, medians and quartiles are described. Quantitative analyses utilized IBM SPSS statistics, version 29 (Armonk, NY, USA).

Results

Study characteristics

The search strategy found 568 citations. After screening and full-text review, 9 studies met the final eligibility criteria (Figure 1). Five studies [9-15] compared LKD and healthy controls, but only 3 of those had measurements before-and-after donation.[11-12,14-15] Four additional studies included single cohorts of LKD with measurements pre- and post-donation.[30-33] We identified 3 reports based on secondary analyses [10,11,34] that were considered part of their original publication [9,12] (Figure 1). Three of our included studies [12,14,15] that were published by the same research group (UK) had participants evaluated at different time periods and some degree of overlap was assumed. In the absence of confirmation, these studies were analyzed independently. Supplementary Table S-1 and Appendix 4 summarize the characteristics of studies, participants and country of origin.

Insert Figure 1 here.

Population characteristics

Living kidney donors

A total of 652 LKD had measurements of cf-PWV after kidney donation, but only 438 LKD (in 7 studies) [11,12,14,15,30-33] had examinations before and after nephrectomy. The remaining 214 LKD (in 2 additional studies),[9,10,13] did not have pre-donation assessment. The cf-PWV was measured in 2 studies at 6 months after donation,[11,12,33] in 6 studies at 12 months,[11-12,14-15,30-32] and in 3 studies at 5 years or longer (5, 6 and 9 years) [9,13,15] (Supplementary Table S-1). Amongst all studies, average age at donation was 48.0 years (± 5.0 years) (range: 41.0 to 54.1 years) with most organs donated by females with an average proportion of 63.4% (range: 54% to 87%) per study. Only 3 studies [11,12,13,15] reported the ethnic composition of LKD. Donors were predominantly white Caucasian (range: 90% to 94.6%) with a minority of Asian (range: 6% to 7%) and Black heritage (range: 0% to 3%). Only 2 studies [9,31] reported a detailed definition of hypertension characterized as SBP >140 mm Hg and/or DBP >90 mm Hg; or by the use of antihypertensive therapy due to previously diagnosed hypertension. In 7 of the 9 studies, an average of 12.5% (range: 0% to 32%) of LKD were hypertensive at the time of donation and this rate increased to an average of 17.2 .% (range: 4% to 32%; 4 studies) and 12.8% (range: 5.4% to 18.8%; 4 studies) at 12 months and 5-to-9 years after donation respectively. Moreover, an average of 32.9% of donors (range: 28% to 44%) were current smokers and/or individuals with a history of previous smoking, although the duration of exposure was not reported. The most common medications prescribed for LKD prior to organ donation were antihypertensives and lipid reducing drugs (e.g., statins). The most common antihypertensive medications were angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARB) (range: 0% to 19% in 5 studies), calcium channel blockers (range: 2% to 5%; in 6 studies) and beta blockers (2%; in 3 studies). Statins were reported with an average rate between 0% and 12% in 6 studies. There was no information on cardiovascular risk assessment pre-donation and hypertension management with diuretics.

Healthy controls

A total of 602 healthy individuals were included as comparators in 5 studies (Table S-1). Two studies had comparative assessments at 12 months after nephrectomy,[11,12,14] one at 12 and 60 months,[15] and two at 5 years or longer (5, 6 and 9 years).[9,10,13] The average age of healthy controls in these studies was 46 years (range: 43 to 49 years) compared to 49 years (range: 46 to 51 years) in kidney donors. The incidence of hypertension, history of cardiovascular disease and diabetes mellitus was higher in kidney donors post-donation, relative to controls. The average proportion of hypertension, history of cardiovascular disease and diabetes mellitus was 6.3 % (range: 0% to 9% in 5 studies), 16.7% (range: 0% to 28% in 3 studies) and 0.5 % (range: 0% to 2% in 4 studies) in healthy controls compared to 11.0 % (range: 5% to 18.8% in 5 studies), 19.6% (range: 4.9% to 34% in 3 studies) and 1.6% (range: 0% to 5.9% in 4 studies) respectively in LKD. Only 3 studies [11,12,14,15] documented the proportions of current and previous smokers between these two sub-populations ranging between 2% and 28% in controls versus 6% and 44% in donors. The most frequent medications prescribed to healthy controls as reported in 2 studies [14,15] were ACE inhibitors/ARBs, statins and calcium channel blockers. Their proportions at the time of initial recruitment ranged from 3% to 7%, 7% to 13% and 2% to 3% respectively. Ethnicity in healthy controls was only reported in 3 studies.[11,12,13,15] The ethnic distribution of these participants was white Caucasian (range: 84% to 95%), Asian (9%) and black heritage (6% to 7%). None of the studies reported cardiovascular risk in healthy controls. Additional baseline characteristics were either part of the exclusion criteria or were not sufficiently reported.

Outcome measures

Aortic Stiffness

The primary outcome analysis included 7 studies [11,12,30-33] with non-adjusted cf-PWV values plus 2 studies[14,15] whose values were adjusted according to mean BP and heart rate. Due to limited information in these 2 studies, their adjusted values were not transformed. Supplementary Tables S-2 and S-3 summarize the unadjusted cf-PWV values in donors and controls respectively. The median unadjusted cf-PWV prior to nephrectomy was 7.10 m/s (quartiles: 6.80, 7.52) and this value increased to a median of 7.21 m/s (quartiles: 7.14, 7.27) at 6 months, 7.30 m/s (quartiles: 7.22, 7.68) at 12 months and 7.69 m/s (quartiles: 7.50, 8.60) at 5 years. Figure 2 shows the Forest plots of the effect estimates on the unadjusted cf-PWV in LKD before and after nephrectomy and Figure 3 illustrates their differences against healthy comparators . The unadjusted cf-PWV in LKD increased with time after nephrectomy (Z=3.1, p=0.002; I^2=0%). While these effects were statistically significant at 12 months after nephrectomy (Z=2.2, p=0.03; I^2=10%; 6 studies), they were not significant at 6 months (Z=1.3, p=0.20; I^2= 0%; 2 studies) or 5 years and longer (Z=1.8; p=0.07; one study). The mean difference in the unadjusted cf-PWV before and after donation was 0.23 m/s (95% CI: -0.12; 0.58) at 6 months, 0.30 m/s at 12 months (95% CI: 0.03; 0.57) and 0.60 m/s at 5 years (95% CI: -0.04; 1.24). At 12 months post-donation, unadjusted cf-PWV values in LKD were on average 0.4 m/s (95% CI: 0.08; 0.72) higher than in healthy controls (Z=2.43; p=0.01; 3 studies), but this difference became non-significant (mean: 0.15 m/s; 95% CI: -0.32; 0.62) at 5 years or longer after donation (Z=0.62; p=0.54). Statistical heterogeneity between studies was high at 12 months (I^2=78%; p=0.01) and at 5 years (I^2=65%; p=0.02).

Insert Figures 2 and 3 here

Kidney function

GFR in LKD was measured in one study at 6 months post-nephrectomy,[33] in 6 studies at 12 months [11,12,14,15,30-32] and in 3 studies at 5 years or longer.[9,10.13,15] Six studies estimated GFR using the CKD Epidemiology Collaboration equation (CKD-EPI) based on the ^{Cr51}EDTA clearance,[11,12.14,15,30-32] one study[33] estimated GFR from 24-hour urine creatinine clearance, and 2 additional studies [9,10.13] used both the modification of Diet in renal disease (MDRD) and CKD-EPI from Iohexol clearance. Figure 4 shows the Forest plots of the effects estimates on GFR in LKD and Figure 5 exhibits their differences against healthy controls. Relative to before nephrectomy, GFR decreased by an average of 30 ml/min/1.73 m^2 (95% CI: -32; -28) throughout the 5-year follow-up period (Z=27.4; p<0.001). In particular, GFR decreased by 38 ml/min/1.73 m^2 (95% CI: -49; -26) within the first 6 months after nephrectomy (one study; Z=6.5; p<0.001), by 31 ml/min/1.73 m^2 (95% CI: -34; -27) at 12 months (6 studies; Z=19.3; p<0.0001), and by 28 ml/min/1.73 m^2 (95% CI: -31; -25) at 5 years or longer (3 studies; Z=17.3; p<0.0001). When these values were compared to healthy controls, LKD had significantly lower GFRs (mean differences: -26 ml/kg/1.73 m^2; 95% CI: -28; -23; Z=22.1; p<0.001).

Insert Figures 4 and 5 here

Systemic BP

In LKD, systolic and diastolic BP were measured non-invasively at 6 months post-donation in 1 study,[32] at 12 months in 4 studies, [11,12,14,31,32] at 1 and 5 years in another,[15] and longer than 5 years in 2 studies.[9,10,13] A single study [30] did not report BP post-nephrectomy. Five studies [11,12,13,14,15,32] reported the daily average BP derived from 24-hour BP monitoring, while four studies [9,10,30,31, 33] reported BP values from the average of 3 measurements taken at the time of the office visit. Most studies except one,[9,10] measured BP in controls at initial recruitment and follow-up. The Forest plots of the effect estimates on the systolic BP are represented in Supplementary Figures S-1 and S-2 and on the diastolic BP are presented in Supplementary Figures S-3 and S-4 respectively. Diastolic BP (Z=2.6; p=0.009), but not systolic BP (Z=0.8; p=0.44) increased with time after donation. This effect was only significant at 5 years post-nephrectomy, when diastolic BP increased by an average of 5 mm Hg (95% CI: 2.1, 8.8; I^2=63%; Z=3.2; p=0.001) relative to pre-donation values. When these time-related changes were compared to healthy controls, differences in systolic (mean differences: 0.8 mm Hg 95% CI: -1.2; 2.7) and diastolic BP (mean differences: 1.1 mm Hg; 95% CI: -0.4; 2.6) at 5 years or longer were non-significant (systolic: Z=0.8; p=0.43; diastolic: Z=1.48, p=0.14). Overall, statistical heterogeneity was moderate for systolic (I^2=44%; $\chi2$=12.5; p=0.08) and marginal for diastolic BP (I^2=53%; $\chi2$=14.1; p=0.04).

Comparison with reference values

Supplementary Table S-4 shows the yearly changes in cf-PWV for six reference studies that included 6,278 normotensive healthy participants (>18 and <70 years).[35-40] Supplementary Table S-5 shows the estimated yearly changes in non-adjusted cf-PWV for LKD and healthy controls. The non-adjusted cf-PWV increased by an average of 0.174 m/s per year (± 0.720) in LKD (8 studies) and 0.090 m/s per year (± 0.951) in healthy controls (4 studies). The yearly increases in LKD and their controls were comparable to the 0.1203 m/s per year (± 0.1486) average increase from normotensive healthy individuals (>18 to <70 years) (donors: t=0.20; p=0.84; controls: t=0.078; p=0.93). Since previous studies have indicated a larger yearly increase in cf-PWV for older age groups, we performed a sub-group analysis for individuals ≤ 60 years and > 60 years. The average yearly increase in cf-PWV in reference studies for individuals ≤ 60 years was 0.0751 m/s (± 0.061) compared to 0.158 m/s (± 0.143) in those > 60 years (Supplementary Table S-4).

Our analysis showed that there was no difference in the average yearly change in cf-PWV between LKD (t=-0.301; p=0.76) or healthy controls (t=-0.026; p=0.97) against normotensive healthy individuals ≤ 60 years.

Sensitivity analyses

The effect of overlapping on the effect estimates between LKD and healthy controls was tested by sequential exclusion/inclusion of the involved studies.[11,12,15] Exclusion decreased the mean cf-PWV difference at 12 months (full model: 0.40 m/s, partial models: 0.34 m/s, 0.31m/s) and increased statistical heterogeneity (full model: 78%; partial models: 81%, 86%), but there was no effect on the overall estimates (χ^2=0.32; df=1, p=0.57). Our assessment of parameter estimates, quality and effect model did not change the final analysis.

We evaluated the impact of adjusting our model for "double counting" errors on the effect estimates in studies with before-and-after design [11,12,14,15,30-33] by investigating the differences in the model with and without adjustment. The Forest plots for the non-adjusted analyses (primary and secondary outcomes) are illustrated in Supplementary Figures S-5, S-6, S-7 and S-8. The mean differences and statistical heterogeneity for the model with and without adjustment are summarized in Table S-6. The pooled mean differences and their precision were not significantly different between the two quantitative models. Although the standard error in the non-adjusted model increased only by 3% (quartiles: -5.1% to 7.4%), its statistical heterogeneity (I^2 value) notably increased by 35% (range: 22% to 47%) compared with the adjusted model.

Risk of bias

Supplementary Table S-7 summarizes the assessment of the risk of bias with the ROBINS-I tool. Four of the 5 studies that included a control group [11-15] had moderate risk of bias (80%) and one serious risk of bias.[9,10] Three single cohort studies [30,31,33] had serious risk of bias (75%) and one moderate risk of bias.[32] No study was classified as low-risk or critical risk of bias. Risk of bias was associated with the presence of confounding bias, selection bias due to relaxation of inclusion criteria for donors and controls, missing data and selective reporting.

Funnel plots of asymmetry

The small number of studies (<10) in the meta-analysis and the likelihood that any test on asymmetry would be underpowered precluded using any test for reporting bias. Supplementary Figure S-9 shows effect estimates and sample sizes for studies with cf-PWV between LKD and controls. A large asymmetry for both small and large sample size studies was evident and suggested potential risk for publication bias.

Certainty of the Evidence

Supplementary Table S-8 summarizes certainty of the evidence for all outcomes according to the GRADE methodology. Confidence on the effect estimates was low to moderate for the cf-PWV, low for systemic BP and moderate to high for GFR.

Discussion

In this systematic review, we pooled data from 652 LKD, 602 healthy controls and 6,278 normotensive healthy participants with standard cf-PWV measurements to evaluate the effects of nephrectomy on aortic stiffness after living kidney donation. Based on low to moderate quality of evidence, our findings suggest that the impact of nephrectomy on aortic stiffness at 5 years post-donation or longer is minimal, despite a reduction in kidney function. On the other hand, cf-PWV increases within the first year after nephrectomy, exceeding values observed in selected groups of non-nephrectomized healthy individuals (average difference: 0.4 m/s), although these differences are negligible at 5 years post-donation (average difference: 0.15 m/s). Additionally, the yearly changes in cf-PWV after donation were similar to those in healthy normotensive individuals from the general population. Our review also suggests that 5 years after donation, systolic and diastolic BPs increased by an average of 3- and 5-mm Hg respectively, but these changes were similar to those identified in healthy control groups. Thus, we hypothesize that vascular remodeling occurs within the first-year post-nephrectomy, leading to discrete elevation of aortic stiffness with no changes in systemic BP. Five years after nephrectomy however, progression of aortic stiffness in LKD is similar to the age-dependent effects observed in a healthy normotensive population.

Compared to values before donation, GFR in LKD decreases by an average of 30 ml/min/1.73 m^2 between 6 months and 5 years after nephrectomy. These results are comparable to previous studies that reported reductions in kidney function between 30% and 50% after kidney donation.[1,2,14,15,41] Our current analysis supports that a reduction in kidney function of such magnitude after donation is insufficient alone to cause significant effects on aortic stiffness at least 5 years post-donation. In contrast, similar reductions in kidney function in early-stage chronic kidney disease (CKD) are associated with increased aortic stiffness and reduced vascular distensibility.[42-45]

Inflammation-mediated endothelial injury,[15,42] increased upregulation of matrix metallo-proteinase-2,[46] abnormal calcium/phosphorous mineral balance[47] and extracellular fluid excess[42] are mechanisms of vascular injury more likely found in CKD patients, which may play a role on the increased aortic stiffness in CKD, but not after kidney donation.[9,41,46,48]

Studies on the progression of aortic stiffness after kidney donation have had contradictory results. While some studies have shown an increase in aortic stiffness [9-15,32] others have documented a negligible effect.[30,31,33] Varying study designs, small sample sizes, short-term follow-ups and differences between BP-adjusted and non-adjusted cf-PWV values may have contributed to the heterogeneity in the results. Our findings confirm that there is a paucity of well-designed cohort studies with large sample sizes and long-term follow ups. In addition, although our meta-analysis increased the robustness of the comparisons between donors and controls, this analysis may have been underpowered to detect small differences. A difference of 0.4 m/s (SD: 3) in cf-PWV between donors and controls would have required at least 883 participants per group with 80% power and level of significance of 5%. Although our analysis was adjusted for duration of follow-up and study quality, heterogeneity between studies was still present. We speculate that relaxation of study inclusion criteria may have led to unbalanced distribution of risk determinants (i.e., hypertension, smoking, diabetes, dyslipidemia) between the two cohorts. Because these confounders may decrease comparability, baseline differences should be minimized in future studies.

The effect of reduced kidney function, independent of increased BP, on aortic stiffness in LKD is controversial.[17,44,50] In partially nephrectomized rats, reduced kidney function modified the viscoelastic properties of large arteries independent of the effects of age and BP.[51] However, since serum creatinine increased more than double compared to control animals, the magnitude of reduction in GFR may have not been similar to what is observed in LKD. Our review suggests that except for a small increase in cf-PWV within the first-year post-donation, there were no differences in BP between healthy donors and controls. These findings support previous studies that have reported a reduction in the Magnetic Resonance Imaging-detected aortic distensibility in LKD but not in healthy controls at one year post-donation,[12] with these differences becoming negligible at 5 years post-nephrectomy.[15] Furthermore, these changes in donor's aortic stiffness may be associated with an increase in left ventricular mass one year post nephrectomy,[12,33] which is no longer noticeable at 5 years.[15,52]

Several risk factors (e.g., African American or Hispanic ethnicity, obesity, age, diabetes) may increase the risk for elevated BP and aortic stiffness post-donation.[17,31,53,54,55] However, few studies have documented the role of genetics or ethnicity factors in the development of CKD and increased aortic stiffness.[9,10,56,57] Kidney donors of African ancestry with mutations in the Apolipoprotein L1 gene (APOL1) are at higher risk for developing CKD, imposing new challenges to the process of donor selection and consent.[58,59] Bahous et al[9,10] who explored differences in cf-PWV between recipient and non-recipient-related healthy volunteers of Lebanese ancestry, found a significantly higher rate of elevated aortic stiffness in recipient-related healthy controls. Moreover, Muzaale et al.[57] and Wu et al.[60] reported marked differences in the risk for kidney failure across different types of donor-recipient and ethnicity relationships, suggesting genetic factors. Consequently, the role of genetic determinants in modifying risk of aortic stiffness post-donation cannot be ruled out.

Beyond biological effects of reduced kidney function, nephrectomy may also result in alterations of the arterial network that are associated with changes in hemodynamics and functional stiffness of the arterial tree including those associated with the effect of the different types of yuxta-aortic vascular surgeries.[61] Although few studies have documented that compensatory growth of the remaining kidney is commonly seen after unilateral nephrectomy,[62] the relationship of this phenomenon with cardiovascular remodeling and vascular stiffness remains elusive. Interestingly, several circulating growth factors released during compensatory kidney hypertrophy,[63] have been associated with myocardial and central vascular remodeling.[64] In particular, growth hormone (GH) and its main mediator insulin growth factor-1 (IGF-1) are implicated in the early stages of compensatory renal hypertrophy [65] and increase aortic wall thickness in transgenic mice models without any significant change in arterial BP.[66] Thus, we speculate that these circulating growth factors may be linked to the cardiovascular remodeling process and transient increase in aortic stiffness early after nephrectomy.

Limitations

The strength of this review includes a rigorous systematic methodology and assessment of study quality and certainty of the evidence. Nevertheless, our conclusions may be limited by the small number of studies and participants, and the restricted access to information for data standardization.[11,12,14,15] In particular, over-representation of the Caucasian population in these studies, prevents the applicability of our conclusions to other ethnicity groups. Furthermore, our sensitivity analysis on studies where overlapping was suspected,[11,12,14,16] suggested a reduced mean difference in cf-PWV at 12 months post-donation. Thus, the likelihood that overlapping might have influenced our effect estimates cannot be completely excluded. Since cf-PWV is an operator-dependent technique,[67] important issues in the interpretation of these results are the comparability between medical devices,[68] the variation due to the different calculating algorithms[68,69] and the technical reproducibility of these measurements.[67] All selected studies utilized standard devices (Supplementary Table S-1), although no information was given on their reproducibility.[54,67]

Despite our efforts to detect potential sources of heterogeneity, residual confounding was still present, and this may have impacted comparability between cohorts. Additionally, we recognize that the different techniques utilized in the measurement of GFR (estimated versus direct measurement), and BP (24-hour monitoring versus office) may have contributed to the variability on these outcomes.[70,71] Moreover, the confounding effects of anti-hypertensive therapy on the control of BP after donation and the limitations for adjusting the effects of gender and age [72,73] in our analysis cannot be ignored. Age in particular, may have a differential effect on arterial stiffness for males and females.[72] Although both sexes experience an increase in arterial stiffness with aging, the increase seems to be steeper in males than females.[72,73]We believe that an individual participant data meta-analysis would have been a more appropriate way to synthesize our data and adjust aortic stiffness according to the different risk factors. Finally, the risk of publication and selection bias cannot be entirely ruled out.

Conclusions

Our systematic review and meta-analysis documented that reduced kidney function after living kidney donation is associated with a small elevation in aortic stiffness within the first year, independent of changes in systemic BP. These effects however, become negligible 5 years post-donation. The data suggest that vascular remodeling occurs within the first year post-nephrectomy but is no longer detected after 5 years. In the absence of other critical cardiovascular risk factors, the effects of nephrectomy on aortic stiffness in LKD at least 5 years after donation is insignificant. These results may have implications for the future evaluation and consent of prospective living kidney donors.

Acknowledgements

We acknowledge the assistance and support of Ms. Risa Shorr from the Learning services at the Ottawa Hospital during the development of our search strategy. We express our appreciation for "all individuals that donate a kidney for the benefit of another human being" and recognize the inspiring motivation of Adolfo M. Rodriguez-Trejo.

Contributors

RAR and KDB conceived the study; RAR designed the study, created the analytical plan, synthesized, analyzed, interpreted the evidence and drafted the manuscript; RAR and KM were involved in study screening, data extraction, verification and quality appraisal; RAR, MA, AB, EC and KDB provided comments and reviews to initial drafts. All authors have read, reviewed and approved the final version of the manuscript. RAR is the guarantor of this work.

Funding

This research received no specific grant from any funding agency in the public, commercial or not-for profit sectors.

Competing interests

None declared.

Patient and Public involvement

Patients and/or the public were not involved in the design, conduct, reporting or dissemination plans for this study.

Patient consent for publication

Not required.

Data availability statement

All data relevant to the study are included in the article or uploaded as supplementary information.

Supplementary information

File 1; Supplementary Tables (Tables S1, S2, S3, S4, S5, S6, S7, S8); Supplementary Figures (Figures S1, S2, S3, S4, S5, S6, S7, S8, S9); Appendices (Appendix 1, 2, 3, 4, 5)

File 2: PRISMA checklist.

File 3: MOOSE chekist

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Figure Legends

Figure 1: PRISMA flow chart.

Figure 2: Pooled effect estimates on the carotid-femoral Pulse Wave Velocity (cf-PWV) (m/s) in living kidney donors from before to after nephrectomy. In single cohort studies with before-and-after design, the number of living kidney donors allocated to each measurement was reduced by 50% to decrease “double counting” errors during the analysis.

Figure 3: Pooled effect estimates on the differences in carotid-femoral Pulse Wave Velocity (cf-PWV) (m/s) between living kidney donors and their respective healthy comparators. Because pre-donation values for Bahous et al. (2006) and Kasiske et al. (2020) were not provided, mean differences between living kidney donors and controls were calculated using their mean absolute cf-PWV values. In the study by Bahous et al. (2006), the number of living kidney donors allocated to each measurement was reduced by 50% to decrease “double counting errors” during the analysis. NRR: non-recipient related, RR: recipient related.

Figure 4: Pooled effect estimates on the Glomerular Filtration rate (GFR) (ml/min/1.73 m^2) in living kidney donors from before to after nephrectomy. In all single cohort studies with before-and-after design, the number of living kidney donors allocated to each measurement was reduced by 50% to decrease “double counting” errors during the analysis.

Figure 5: Pooled effect estimates on the differences in Glomerular Filtration rate (GFR) (ml/min/1.73 m^2) between living kidney donors and their healthy comparators. In the study by Bahous et al. (2006), the number of living kidney donors allocated to each measurement was reduced by 50% to decrease “double counting errors” during the analysis.

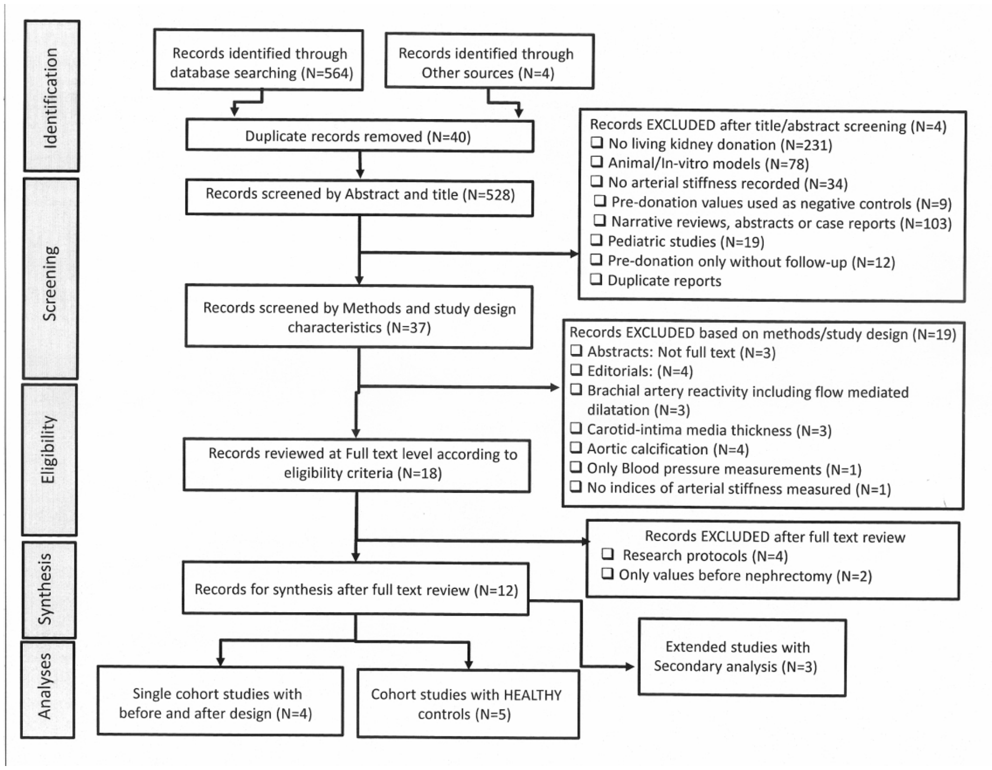

Fig 1: PRISMA Flow Chart.

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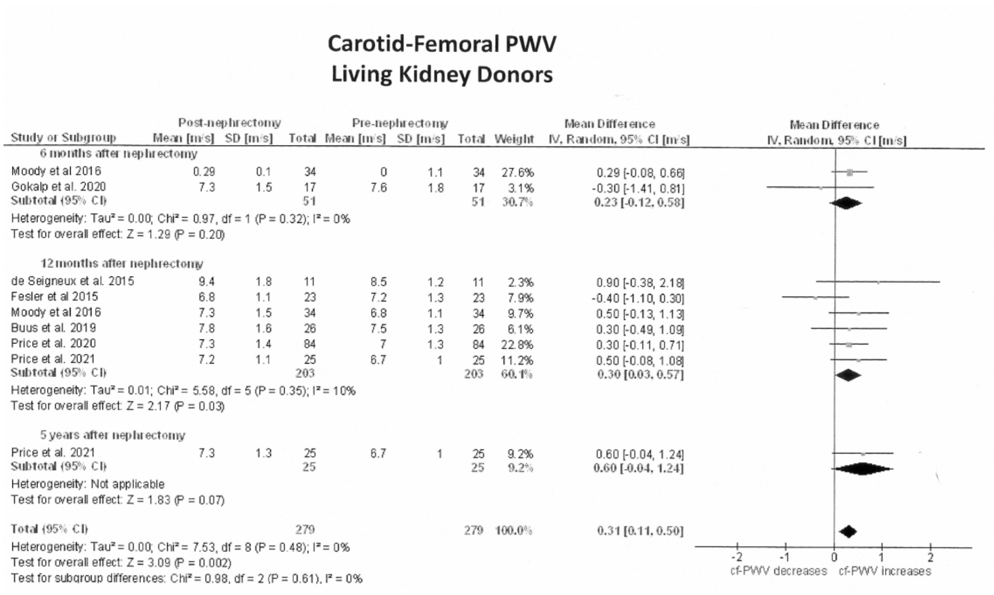

Figure 2: Pooled effect estimates on the carotid-femoral Pulse Wave Velocity (cf-PWV) (m/s) in living kidney donors from before to after nephrectomy.

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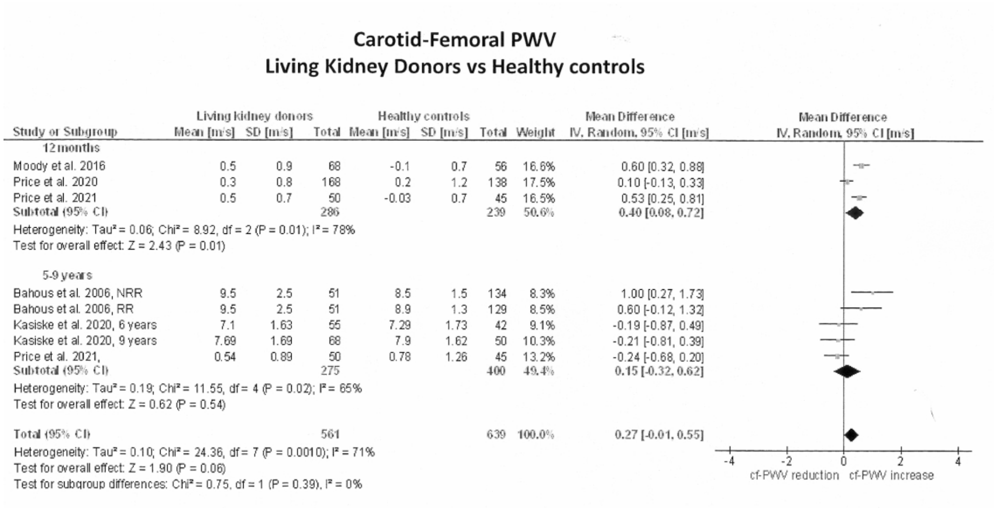

Figure 3: Pooled effect estimates on the differences in carotid-femoral Pulse Wave Velocity (cf-PWV) (m/s) between living kidney donors and their respective healthy comparators.

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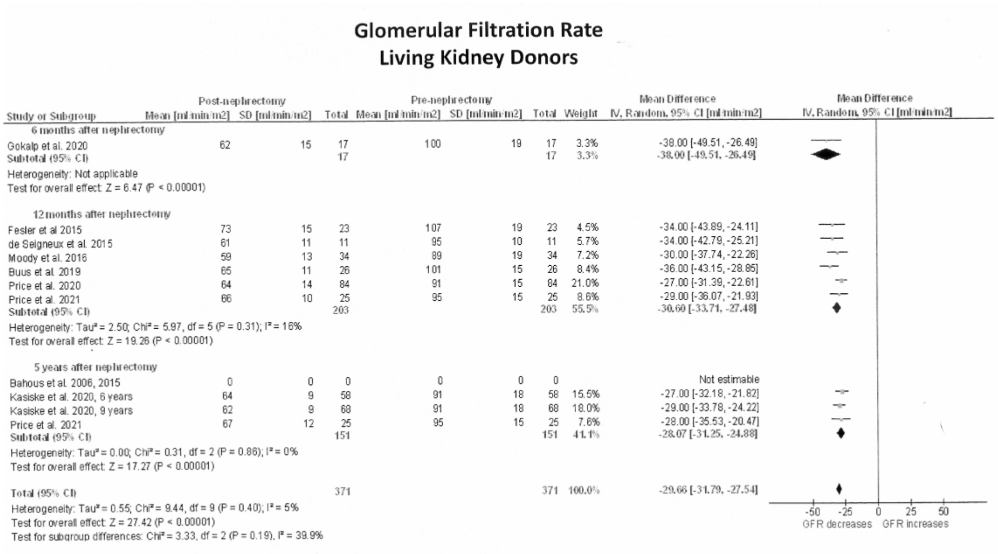

Figure 4: Pooled effect estimates on the Glomerular Filtration rate (GFR) (ml/min/1.73 m2) in living kidney donors from before to after nephrectomy.

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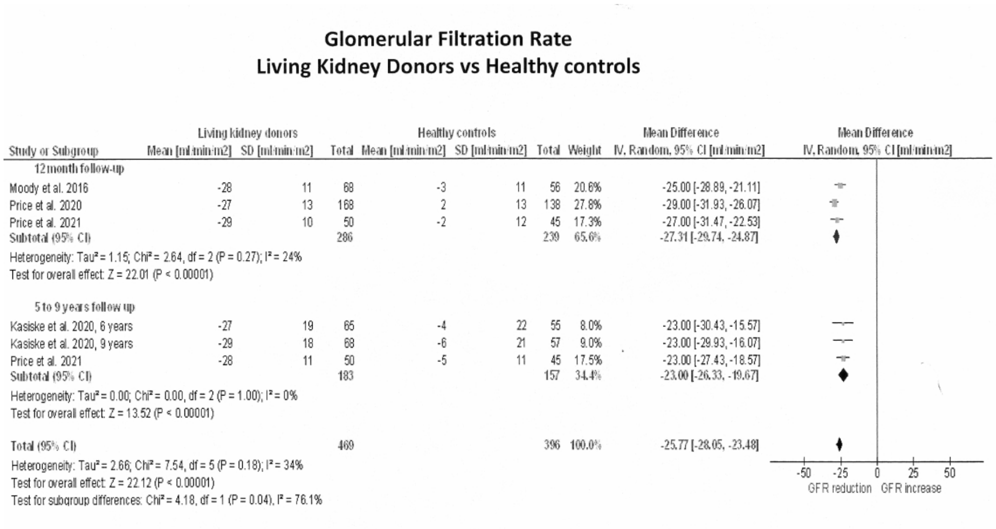

Figure 5: Pooled effect estimates on the differences in Glomerular Filtration rate (GFR) (ml/min/1.73 m2) between living kidney donors and their healthy comparators. In the study by Bahous et al. (2006), the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting errors" during the analysis.

271x144mm (300 x 300 DPI)

Aortic stiffness after living kidney donation:

A Systematic Review and Meta-analysis

Rosendo A. Rodriguez, MD, PhD; Kylie McNeill, PhD; Mohsen Agharazii, MD; Ann Bugeja, MD; Edward G. Clark, MD, MSc; Kevin D. Burns. MD, CM.

SUPPLEMENTAL MATERIAL

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II - Supplementary Figures and Figure legends

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Supplementary Tables

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Table S-2 Hemodynamic characteristics in living kidney donors before and after nephrectomy.

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Table S-5. Estimated average yearly change (± SD) in cf-PWV (m/s/year) for living kidney donors and healthy comparators.

Table S-6. Summary of the pooled mean differences for the primary and secondary outcomes and their statistical heterogenity in the adjusted and full meta-analysis models (with and without 50% reduction on the sample size for each arm) in before-and-after study designs.

Table S-7. Risk of bias assessment according to the ROBINS-I scale for eligible studies

Table S-8. Assessment of the certainty of the evidence on the primary and secondary outcomes according to the GRADE guidelines.

Table S-1. Demographic characteristics of kidney donors and healthy controls in eligible studies.

Study characteristics		Living Kidney donors					Healthy controls		
Reference ID	Country	Age at donation (years)	Duration of follow-up post-donation	Females/total number of donors (%)	Hypertensive donors/total number of donors (%)	Current or past smokers/total number of donors (%)	Females/ total number of controls (%)	Hypertensive controls/total number of controls (%)	Current or past smokers/ total number of controls (%)
Fesler et al. 2015	France	51.0 ± 10.0	12 mo	39/45 (87)	0/45 (0)	NR/45	N/A	N/A	N/A
DeSeigneux et al. 2015	Switzerland	54.1 ± 10.2	12 mo	12/21 (57.1)	5/21 (23.8)	7/21 (33.0) NR/21	N/A	N/A	N/A
Moody et al. 2015, 2016	UK	46.5 ± 12.1	12 mo	45/68 (66.0)	3/68 (5.0)	8/68 (12.0) 21/68 (30.0)	29/56 (52.0)	3/68 (5.0)	8/68 (12%); 21/68 (30%)
Bahous et al. 2006, 2015	France	41.0 ± 11.0	111 ± 42 mo	66/101 (65.3)	0/101 (0)	NR/101	143/263 (54.4)	0/263 (0)	NR/263
Buus et al. 2019	Denmark	49.5 ± 12.0	12 mo	28/52 (54.0)	17/52 (32.0)	15/52 (29.0); NR/52	N/A	N/A	N/A
Gokalp et al. 2020	Turkey	51.0 ± 13.0	6 mo	20/34 (59.0)	NR/34	NR/34	N/A	N/A	N/A
Price et al. 2020	UK	51.0 ± 12.0	12 mo	90/168 (54.0)	17/169 (10.0)	74/168 (44.0) NR/168	81/138 (59.0)	9/138 (7.0)	38/138 (28.0); NR/138
Kasiske et al. 2020	USA	98% between 18 to 64 years	6 years; and 9 years	6-year visit: 70/109 (64.0) 9-year visit: 72/113 (64.0)	6/203 (3.0)	24/203 (11.8) 40/203 (19.7)	6-year visit: 34/84 (40.0) 9-year visit: 40/100 (40.0)	9/ 201 (4.5)	24/201 (11.9); 45/201 (22.4)
Price et al. 2021	UK	48.0 ± 12.5	12 mo. and 60 mo	27/50 (64.0)	2/50 (4.0)	4/49 (8.1); 15/49 (30.6)	28/45 (62.0)	3/43 (7.0)	2/43 (4.6); 12/43 (28.0)

UK: United Kingdom, NR: not reported, N/A not applicable; mo: months;

Table S-2. Hemodynamic characteristics in living kidney donors before and after nephrectomy.

study	LKD Pre-donation baseline				LKD Post-donation follow-up				
Citation	cf-PWV (m/s)	GFR (ml/min/ 1.73m²)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Follow-up post-donation	cf-PWV (m/s)	GFR (ml/min/ 1.73m²)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Fesler et al. 2015	7.2 ± 1.3	107 ± 19	122 ± 12	70 ± 9	12 mo	6.8 ± 1.1	73 ± 15;	122 ± 13	70 ± 8
DeSeigneux et al. 2015	8.5 ± 1.2	95 ± 10	116.6 ± 13	76.0 ± 7.3	12 mo	9.4 ± 1.8	61 ± 11	N/R	N/R
Moody et al. 2015, 2016	6.8 ± 1.1	89 ± 19	121 ± 8.0	75 ± 6.0	12 mo	7.3 ± 1.5	59 ± 13	122 ± 12.0	76 ± 9
Bahous et al. 2006, 2015	N/R	107.5 ± 20	114.0 ± 18	69.1 ± 19.4	111 ± 42 mo	9.5 ± 2.5	86.2 ± 18	129.6 ± 20	81.6 ± 11.8
Buus et al. 2019	7.5 ± 1.3	100.6 ± 15	120 ± 14	74 ± 8	12 mo	7.8 ± 1.6	64.7 ± 10.6	119 ± 13	74.9 ± 9.7
Gokalp et al. 2020	7.6 ± 1.8	99.8 ± 19	120 ± 15.6	74.3 ± 9.2	6 mo	7.3 ± 1.5	61.9 ± 15.1	122.1 ± 20.1	74.9 ± 9.7
Price et al. 2020	7.0 ± 1.3	91 ± 15	124 ± 10	79 ± 8	12 mo	7.3 ± 1.4	64 ± 14	124 ± 10	79 ± 8
Kasiske et al. 2020 *	N/R	91 ± 17.7	120 ± 13.6	69.7 ± 8.8	6 years	7.1 ± 1.6	64 ± 8.6	117.5 ± 11.2	73.1 ± 8.6
	N/R	-	-	-	9 years	7.7 ± 1.7	62 ± 8.9	117.1 ± 13.0	73.9 ± 10.0
Price et al. 2021 *	6.7 ± 1.0	95 ± 15	121 ± 9	73±7	12 mo	7.2 ± 1.1	65.8 ± 10.3	121.2 ± 15.1	74 ± 10.2
	-	-	-	-	60 mo	7.3 ± 1.3	67.4 ± 12.4	123.6 ± 11.5	78 ± 9.0

BP: blood pressure; cf-PWV: carotid-femoral pulse wave velocity (m/s); GFR: glomerular filtration rate; LKD: living kidney donors; mo: months; N/R: not reported; * Kasiske et al. (6 and 9 years) and Price et al. (12 months and 60 months) used the same pre-donation baseline for each of their 2 follow-up points after donation.

Table S-3. Hemodynamic characteristics of healthy comparators at baseline and follow-up.

	Healthy comparators – Measurement at enrollment				Healthy comparators - Measurement at follow-up				
study	cf-PWV (m/s)	GFR (ml/min/ 1.73m²)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Time point at follow-up	cf-PWV (m/s)	GFR (ml/min/ 1.73m²)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)
Moody et al. 2015, 2016	6.7 ± 1.1	89 ± 19	122 ± 11	74 ± 8	12 mo	6.7 ± 1.1	86 ± 19	121 ± 10	76±9
Bahous et al. 2006, 2015 (NRR group)[&]	N/R	N/R	N/R	N/R	111 ± 42 mo	8.5 ± 1.5	N/R	125.2 ± 9.5	75.7 ± 6.1
Bahous et al. 2006, 2015 (RR group)[&]	N/R	N/R	N/R	N/R	111 ± 42 mo	8.9 ± 1.3	N/R	123 ± 15.1	78.5 ± 10.6
Price et al. 2020	7.0 ± 1.4	94 ± 16	122 ± 10	77 ± 8	12 mo	7.2 ± 1.4	96 ± 17	123 ± 12	78 ± 9
Kasiske et al. 2020 *	N/R	90 ± 16.2	117.1 ± 13.0	69.7 ± 8.8	6 years	7.29 ± 2.5	86 ± 15.7	118.8 ± 14.2	73.1 ± 8.6
					9 years	7.90 ± 2.3	84 ± 13.5	120 ± 14.9	73.9 ± 10.0
Price et al. 2021 *	6.78 ± 0.17	99 ± 16	122±11	75±9	12 mo	6.73 ± 0.16	96.9 ± 11.9	120.8 ± 7.6	75.4 ± 7.6
					60 mo	7.54 ± 0.22	94.1 ± 16.7	122.7 ± 11.8	78.4 ± 9.0

BP: blood pressure; cf-PWV: carotid-femoral pulse wave velocity; GFR: glomerular filtration rate; mo: months; N/R: not reported; * Kasiske et al. (6 years and 9 years) and Price et al. (12 months and 60 months) used the same baseline at enrollment for each of their 2 follow-up points; NRR group: not recipient related group; RR group: recipient related group; [&] Bahous et al. 2006, 2015, identified 2 sub-groups (NRR and RR) within their comparator group relative to the kidney recipient.

Table S-4 Estimated average yearly change (± SD) in cf-PWV (m/s/year) for normotensive healthy individuals participating in population-based studies with age-adjusted values.

variables	Arterial stiffness' collaboration 2010	Farro et al 2012	Baier et al. 2018	Elias et al. 2011	Kozakova et al. 2015	Gomez-Sanchez et al. 2020
Age (years) (range)	>30 to ≥70	>18 to 69	>18 to 80	40 to 90	18 to 78	35 to 75
Measurement device	SphygmoCor	SphygmoCor	Vicorder	SphygmoCor	SphygmoCor	SphygmoCor
Total number of participants	1,455	429	3,092	502	307	493
Average cf-PWV (m/s/year) in all participants	0.1500 ± 0.0967 (#)	0.1580 ± 0.0864	0.1000 ± 0.1208 (&&)	0.105 ± 0.2913 (&)	0.090 ± 0.005	0.1188 ± 0.2915
Average cf-PWV in ≤ 60 years (m/s/year)	0.0967 ± 0.0404 (#)	N/R	0.0733 ± 0.0321	N/R	0.088 ± 0.007	0.0425 ± 0.1645 (**)
Average cf-PWV in > 60 years (m/s/year)	0.2300 ± 0.1131 (#)	N/R	0.0550 ± 0.0212	N/R	0.150 ± 0.019	0.1950 ± 0.418 (**)
cf-PWV in Females (m/s/year)	N/R	N/R	N/R	N/R	0.099 ± 0.005	0.1603 ± 0.1472
cf-PWV in males (m/s/year)	N/R	N/R	N/R	N/R	0.076 ± 0.005	0.1805 ± 0.1515

(*) Estimates are based on the 95% percentile values from all males and females included in this study.

(**) Data represent average values from reported males and females sub-groups in this study. The reported 95% CI were transformed to SD using the method of Wu et al (2018).

(&) Estimates are based on the regression coefficient (b) of the relationship between age and cf-PWV in all participants from this study (Table 4) and adjusted by mean arterial pressure, weight, height, glucose and creatinine. The reported standard error (SE) was converted to SD according to the formula: SD=SE*(SQRT sample size).

(#) Estimates are based on the reported 90th percentile of the distribution in the sub-sample defined as "normal values" in this study (Table 4)

&& Estimates are based on the 97.5th percentile values from the normotonic subgroup in this study (Table 4).

Table S-5. Estimated average yearly change (± SD) in cf-PWV (m/s/year) for living kidney donors and healthy comparators.

Living Kidney Donors									
	Buus et al. 2019	Gokalp et al 2020	De Seigneux et al 2015	Moody et al 2016	Fesler et al. 2015	Price et al 2020	Price et al. 2021	Price et al 2021	Kasiske et al. 2020
Age at measurement (years)	51.0 ± 11.6	50.97 ± 13	55.1 ± 10.2	47.5 ± 12.1	52 ± 10	52 ± 12	49 ±12.5	54.3 ± 12.3	98.6% (age: 18 to 64)
Time frame of observation	12 mo	12 mo	12 mo	12 mo	12 mo	12 mo	12 mo	60 mo	36 mo
Device	SphygmoCor	SphygmoCor	SphygmoCor	SphygmoCor	Sphygmocor	SphygmoCor	SphygmoCor	SphygmoCor	Sphygmocor
Number of subjects	51	34	21	68	45	168	50	42	109
Average yearly change in cf-PWV (m/s/year)	+ 0.30 ± 0.900	- 0.24 ± 0.401	+ 0.3 ± 0.469	+ 0.5 ± 0.900	- 0.4 ± 0.250	+ 0.3 ± 0.821	+ 0.50 ± 0.717	+ 0.108 ± 0.920	+ 0.197 ±0.435
Healthy comparators									
				Moody et al 2016		Price et al 2020	Price et al. 2021	Price et al 2021	Kasiske et al. 2020
Age at measurement (years)	N/A	N/A	N/A	45.1 ± 12.8	N/A	49.0 ± 14	45.3 ± 13.07	50.3 ± 12.91	95.6% (age range: 18 to 64)
Time frame of observation	N/A	N/A	N/A	12 mo	N/A	12 mo	12 mo	60 mo	36 mo
Number of subjects	N/A	N/A	N/A	56	N/A	138	45	42	84
Average yearly change in cf-PWV (m/s/year)	N/A	N/A	N/A	- 0.1 ± 0.700	N/A	+ 0.2 ± 1.190	− 0.03 ± 0.680	+ 0.16 ± 1.23	+ 0.203 ± 0.52

cf-PWV: carotid-femoral pulse wave velocity; mo: months; N/A not applicable;

Table S-6. Summary of the pooled mean differences for the primary and secondary outcomes and their statistical heterogeneity in the adjusted and full meta-analysis models (with and without a 50% reduction in the sample size on each arm) in before-and-after design studies.

		Pooled Effect estimates			
		Overall model		12 months post-donation	
Outcome variable	Model tested	Mean difference (95% CI)	I^2 values	Mean difference (95% CI)	I^2 values
cf-PWV (m/s)	Adjusted-model	0.31 (0.011; 0.50)	0%	0.30 (0.03; 0.57)	10%
	Full-model	0.30 (0.10; 0.51)	46%	0.30 (0.02;0.59)	54%
GFR (ml/min/m²)	Adjusted-model	-29.7 (-31.8; -27.5)	5%	-30.6 (-33.7; -27.5)	16%
	Full-model	-30.3 (-32.6; -28.1)	52%	-31.2 (-34.4; -28.0	58%
SBP (mm Hg)	Adjusted-model	1.10 (-1.7; 3.9)	57%	0.13 (-2.2; 2.5)	0%
	Full-model	1.32 (-1.6; 4.2)	79%	0.13 (-1.5; 1.8)	0%
DBP (mm Hg)	Adjusted-model	2.6 (0.7; 4.4)	52%	0.51 (-1.2; 2.2)	0%
	Full-model	2.7 (0.8, 4.6)	76%	0.51 (-0.7, 1.7)	0%

cf-PWV: carotid-femoral Pulse Wave Velocity; GFR: Glomerular Filtration rate; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; 95% CI: 95% confidence interval.

Table S-7. Risk of bias assessment according to the ROBINS-I scale for eligible studies.

study	1. Bias due to confounding	2. Selection Bias	3. Classification Bias	4. Bias due to deviations from intended intervention	5. Bias due to missing data	6. Bias in outcome measurement	7. Bias in selection of the reported result	OVERALL RISK OF BIAS
Fesler et al. 2015	SERIOUS	LOW	SERIOUS	MODERATE	MODERATE	MODERATE	LOW	SERIOUS
De Seigneux et al. 2015	SERIOUS	SERIOUS	MODERATE	MODERATE	SERIOUS	MODERATE	SERIOUS	SERIOUS
Moody et al. 2016	LOW	MODERATE	LOW	LOW	MODERATE	LOW	LOW	MODERATE
Bahous et al. 2006	SERIOUS	MODERATE	MODERATE	MODERATE	SERIOUS	MODERATE	SERIOUS	SERIOUS
Buus et al. 2019	MODERATE	LOW	MODERATE	MODERATE	LOW	MODERATE	MODERATE	MODERATE
Gokalp et al. 2020	SERIOUS	SERIOUS	MODERATE	SERIOUS	NO INFORMATION	MODERATE	MODERATE	SERIOUS
Price et al. 2020	LOW	MODERATE	LOW	LOW	MODERATE	MODERATE	LOW	MODERATE
Kasiske et al. 2020	MODERATE	MODERATE	LOW	LOW	MODERATE	MODERATE	MODERATE	MODERATE
Price et al. 2021	LOW	LOW	LOW	LOW	MODERATE	MODERATE	LOW	MODERATE

Definition and interpretation of individual items for the ROBINS-I scale: [21]

1.- Confounding of intervention effects occurs when one or more prognostic variables (variables that predict the outcome of interest) also predict whether an individual receives one or the other of the interventions of interest.

2.- When exclusion or inclusion of some participants, or the initial follow up time of some participants, or some outcome events, is related to both intervention and outcome, there will be an association between interventions and outcome even if the effects of the interventions are identical. This type of bias is called selection bias.

3.- Non-differential misclassification is unrelated to the outcome and will usually bias the estimated effect of intervention towards the null. Differential misclassification however, may occur when misclassification of intervention status is related to the outcome or the risk of the outcome, and is likely to lead to bias. It is therefore important that, wherever possible, interventions are defined and categorized without knowledge of subsequent outcomes. Differential misclassification can also occur if information (or availability of information) on intervention status is influenced by outcomes.

4.- This domain (sometimes known as “performance bias”) relates to biases that arise when there are systematic differences between the care provided to experimental intervention and comparator groups, beyond the assigned interventions. Bias may occur when these differences arise because of knowledge of the intervention applied and the expectation of finding a difference between experimental intervention and comparator consistent with the hypothesis being tested in the study. Deviations from intended interventions may arise because an intervention was not implemented successfully because participants did not adhere to interventions, or because important co-interventions were not balanced between intervention and comparator groups.

5.-Reasons for missing data include attrition (loss to follow up), missed appointments, incomplete data collection and participants being excluded from analysis by primary investigators. If the proportion of missing data is low and the reasons for missing data are similar across intervention groups, then the risk of bias is likely to be low. As the proportion of missing data rises, differences in treatment response between available and missing participants may increase the potential for bias.

6.- This bias (referred as detection bias) may be introduced if outcomes are misclassified or measured with error. Differential measurement errors (those related to intervention status) will bias the estimated effect of intervention-outcome relationship. Detection bias can arise when outcome assessors are aware of intervention status or if different methods are used to assess outcomes in different intervention groups, or if measurement errors are related to intervention status or effects (or to a confounder of the intervention-outcome relationship).

7.- Selective reporting will lead to bias if it is based on the direction, magnitude or statistical significance of intervention effect estimates. Selective outcome reporting occurs when an effect estimate for a particular outcome measurement is selected from among multiple measurements. Selective analysis reporting occurs when the reported results are selected from intervention effects estimated in multiple ways, or in the selection of a subgroup of participants, selected from a larger cohort, for which results are reported on the basis of a more interesting finding.

Sterne JAC, Hernán MA, Reeves BC, Savović J, Berkman ND, Viswanathan M, Henry D, et al. ROBINS-I: a tool for assessing risk of bias in non-randomized studies of interventions. BMJ 2016; 355; i4919; doi: 10.1136/bmj.i4919

Table S-8. Assessment of the certainty of the evidence on the primary and secondary outcomes according to the GRADE guidelines.

Outcome	Study design	Overall Risk of bias (ROBINS-I)	Inconsistency	Indirectness	Imprecision	Publication bias	Other considerations	Quality of the body of evidence
Carotid-Femoral Pulse Wave Velocity	Single cohort	Serious risk (low quality)	Not serious	Not serious	Not serious	Serious risk (-1) **	Upgrade + 1 Due to estimated effect sizes	**Low** □□
	Cohort with controls (#)	Moderate risk (moderate quality)	Serious * (-1)	Not serious	Not serious	Not serious	Upgrade +1 Due to estimated effect sizes	**Moderate** □□□
Systolic and diastolic blood pressure	Single cohort	Serious risk (low quality)	Not serious	Not serious	Not serious	Serious risk (-1) &&&	Upgrade + 1 Due to estimated effect sizes	**Low** □□
	Cohort with controls (#)	Moderate risk (moderate quality)	Serious && (-1)	Not serious	Serious (-1) &	Not serious	Upgrade +1 Due to estimated effect sizes	**Low** □□
Glomerular Filtration rates	Single cohort	Serious risk (low quality)	Not serious	Not serious	Not serious	Not serious	Upgrade + 1 Due to estimated effect sizes	**Moderate** □□□
	Cohort with controls (#)	Moderate risk (moderate quality)	Not serious	Not serious	Not serious	Not serious	Upgrade +1 Due to estimated effect sizes	**high** □□□□

Considerations for this assessment [22]:

(#) Best evidence in non-randomized studies of living kidney donation as randomized studies are unethically to practice. The use of a comparator of healthy controls of comparable age enhances the confidence on the level of evidence.

* inconsistency rated as serious due to a consistent and significant heterogeneity between studies.

** small studies with short follow ups more likely to be associated with variable results.

*** over or under-estimation of effects due to selective publication (risk of overlapping in 3 studies)

*** upgraded due to narrow 95% CI and greater than 30% estimated effect size.

& imprecision rated as serious due to wide 95% CI.

&& inconsistency rated as serious due to presence of significant heterogeneity.

&&& publication bias rated as serious due to serious reporting bias (absence of pre-nephrectomy baseline in some studies)

Supplementary Figures and Figure legends:

S-1: Pooled effect estimates on the Systolic Blood Pressure in living kidney donors from before to after nephrectomy.

S-2: Pooled effect estimates on the differences in Systolic Blood pressure (mm Hg) between living kidney donors and their respective healthy comparators.

S-3: Pooled effect estimates on the Diastolic Blood pressure (mm Hg) in living kidney donors from before to after nephrectomy.

S-4: Pooled effect estimates on the differences in Diastolic Blood pressure (mm Hg) between living kidney donors and their respective healthy comparators.

S-5: Pooled effect estimates on the carotid-femoral Pulse Wave Velocity (cf-PWV) in living kidney donors from before-to-after nephrectomy in the unadjusted-model.

S-6: Pooled effect estimates on the Glomerular Filtration rate in living kidney donors from before-to-after nephrectomy without adjusting in the unadjusted-model.

S-7: Pooled effect estimates on the Systolic Blood Pressure (mm Hg) in living kidney donors from before-to-after nephrectomy in the unadjusted-model.

S-8: Pooled effect estimates on the Diastolic Blood Pressure (mm Hg) in living kidney donors from before-to-after nephrectomy in the unadjusted-model.

S-9: Funnel plots of asymmetry in 5 studies who evaluated living kidney donors and controls.

Figure S-1

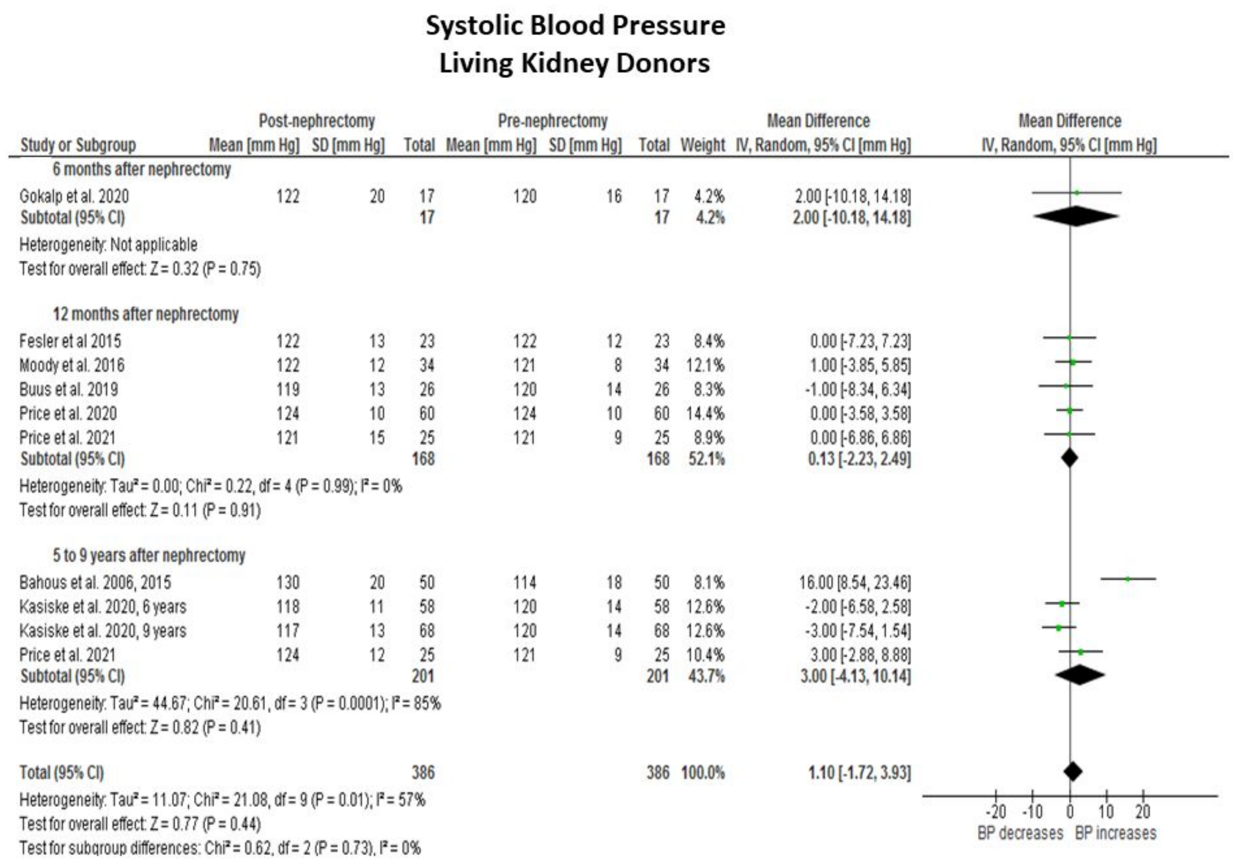

Figure S-1: Pooled effect estimates on the Systolic Blood pressure (mm Hg) in living kidney donors from before to after nephrectomy. In all single cohort studies with before-and-after design the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors during the analysis.

Figure S-2

Systolic Blood Pressure
Living Kidney Donors vs Healthy controls

Study or Subgroup	Living kidney donors Mean [mm Hg]	SD [mm Hg]	Total	Healthy controls Mean [mm Hg]	SD [mm Hg]	Total	Weight	Mean Difference IV, Random, 95% CI [mm Hg]
12 month follow up								
Moody et al. 2016	1	12.9	46	-0.5	6.1	36	12.4%	1.50 [-2.73, 5.73]
Price et al. 2020	0.1	12	119	0.6	8	111	19.3%	-0.50 [-3.12, 2.12]
Price et al. 2021	0.2	15	50	-1.25	8	45	10.7%	1.45 [-3.32, 6.22]
Subtotal (95% CI)			215			192	42.3%	0.30 [-1.71, 2.32]
Heterogeneity: Tau² = 0.00; Chi² = 0.89, df = 2 (P = 0.64); I² = 0%								
Test for overall effect: Z = 0.30 (P = 0.77)								
5 to 9 years follow-up								
Bahous et al. 2006, NRR	130	20	51	125	10	134	8.3%	5.00 [-0.74, 10.74]
Bahous et al. 2006, RR	130	20	51	123	15	129	7.6%	7.00 [0.93, 13.07]
Kasiske et al. 2020, 6 years	118	11	128	119	14	107	16.2%	-1.00 [-4.27, 2.27]
Kasiske et al. 2020, 9 years	117	13	136	120	15	114	15.1%	-3.00 [-6.52, 0.52]
Price et al. 2021	2.57	12	50	0.66	12	45	10.5%	1.91 [-2.92, 6.74]
Subtotal (95% CI)			416			529	57.7%	1.36 [-2.06, 4.78]
Heterogeneity: Tau² = 9.68; Chi² = 11.67, df = 4 (P = 0.02); I² = 66%								
Test for overall effect: Z = 0.78 (P = 0.44)								
Total (95% CI)			631			721	100.0%	0.78 [-1.17, 2.73]
Heterogeneity: Tau² = 3.34; Chi² = 12.57, df = 7 (P = 0.08); I² = 44%								
Test for overall effect: Z = 0.78 (P = 0.43)								
Test for subgroup differences: Chi² = 0.27, df = 1 (P = 0.60), I² = 0%								

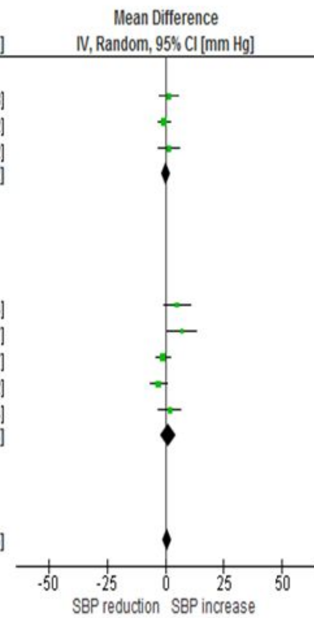

Figure S-2: Pooled effect estimates on the differences in Systolic Blood pressure (mm Hg) between living kidney donors and their respective healthy comparators. In the study by Bahous et al. 2006, the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors during the analysis. NRR: non-recipient related, RR: recipient related.

Figure S-3

Diastolic Blood Pressure
Living Kidney Donors

Study or Subgroup	Post-nephrectomy Mean [mm Hg]	Post-nephrectomy SD [mm Hg]	Post-nephrectomy Total	Pre-nephrectomy Mean [mm Hg]	Pre-nephrectomy SD [mm Hg]	Pre-nephrectomy Total	Weight	Mean Difference IV, Random, 95% CI [mm Hg]
6 months after nephrectomy								
Gokalp et al. 2020	75	10	17	74	9	17	6.1%	1.00 [-5.40, 7.40]
Subtotal (95% CI)			**17**			**17**	**6.1%**	**1.00 [-5.40, 7.40]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 0.31 (P = 0.76)								
12 months after nephrectomy								
Fesler et al 2015	70	8	23	70	9	23	8.5%	0.00 [-4.92, 4.92]
Moody et al. 2016	76	9	34	75	6	34	11.6%	1.00 [-2.64, 4.64]
Buus et al. 2019	75	8	26	74	8	26	9.8%	1.00 [-3.35, 5.35]
Price et al. 2020	79	8	60	79	8	60	13.9%	0.00 [-2.86, 2.86]
Price et al. 2021	74	10	25	73	7	25	8.8%	1.00 [-3.78, 5.78]
Subtotal (95% CI)			**168**			**168**	**52.6%**	**0.51 [-1.21, 2.24]**
Heterogeneity: Tau² = 0.00; Chi² = 0.32, df = 4 (P = 0.99); I² = 0%								
Test for overall effect: Z = 0.58 (P = 0.56)								
1.2.3 5 to 9 years after nephrectomy								
Bahous et al. 2006, 2015	82	12	50	69	19	50	6.3%	13.00 [6.77, 19.23]
Kasiske et al. 2020, 6 years	73	9	58	70	9	58	12.6%	3.00 [-0.28, 6.28]
Kasiske et al. 2020, 9 years	74	10	68	70	9	68	12.8%	4.00 [0.80, 7.20]
Price et al. 2021	78	9	25	73	7	25	9.5%	5.00 [0.53, 9.47]
Subtotal (95% CI)			**201**			**201**	**41.3%**	**5.47 [2.13, 8.81]**
Heterogeneity: Tau² = 7.06; Chi² = 8.06, df = 3 (P = 0.04); I² = 63%								
Test for overall effect: Z = 3.21 (P = 0.001)								
Total (95% CI)			**386**			**386**	**100.0%**	**2.55 [0.67, 4.44]**
Heterogeneity: Tau² = 4.55; Chi² = 18.59, df = 9 (P = 0.03); I² = 52%								
Test for overall effect: Z = 2.65 (P = 0.008)								
Test for subgroup differences: Chi² = 6.69, df = 2 (P = 0.04), I² = 70.1%								

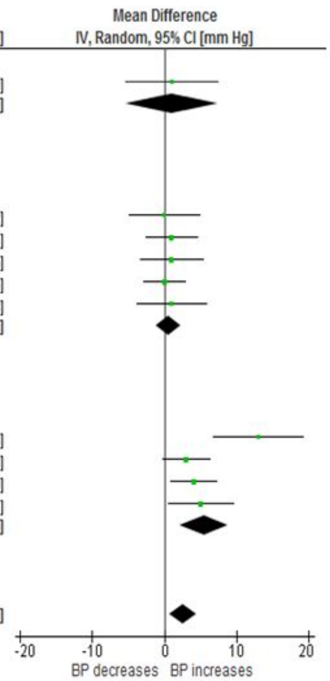

Figure S-3: Pooled effect estimates on the Diastolic Blood pressure (mm Hg) in living kidney donors from before to after nephrectomy. In all single cohort studies with before-and-after design. the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors during the analysis.

Figure S-4

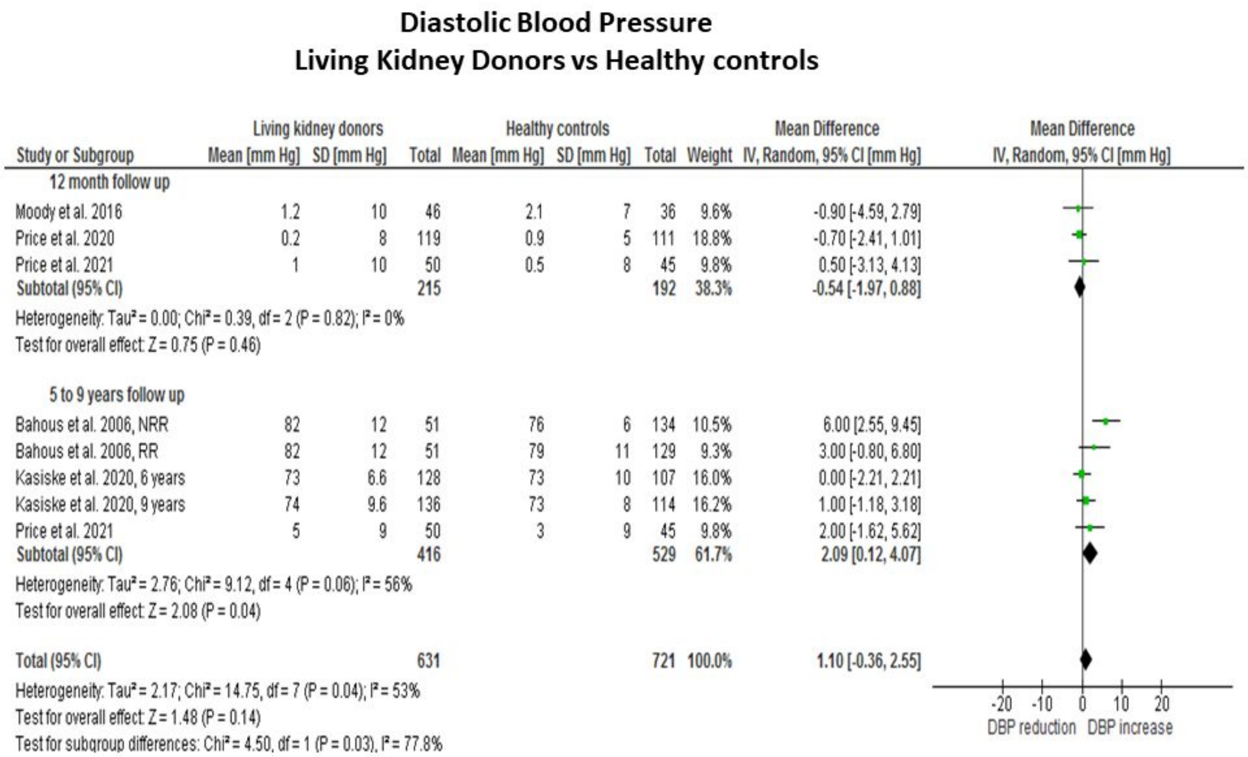

Figure S-4: Pooled effect estimates on the differences in Diastolic Blood pressure (mm Hg) between living kidney donors and their respective healthy comparators. In the study by Bahous et al. 2006, the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors during the analysis. NRR: non-recipient related, RR: recipient related.

Figure S-5

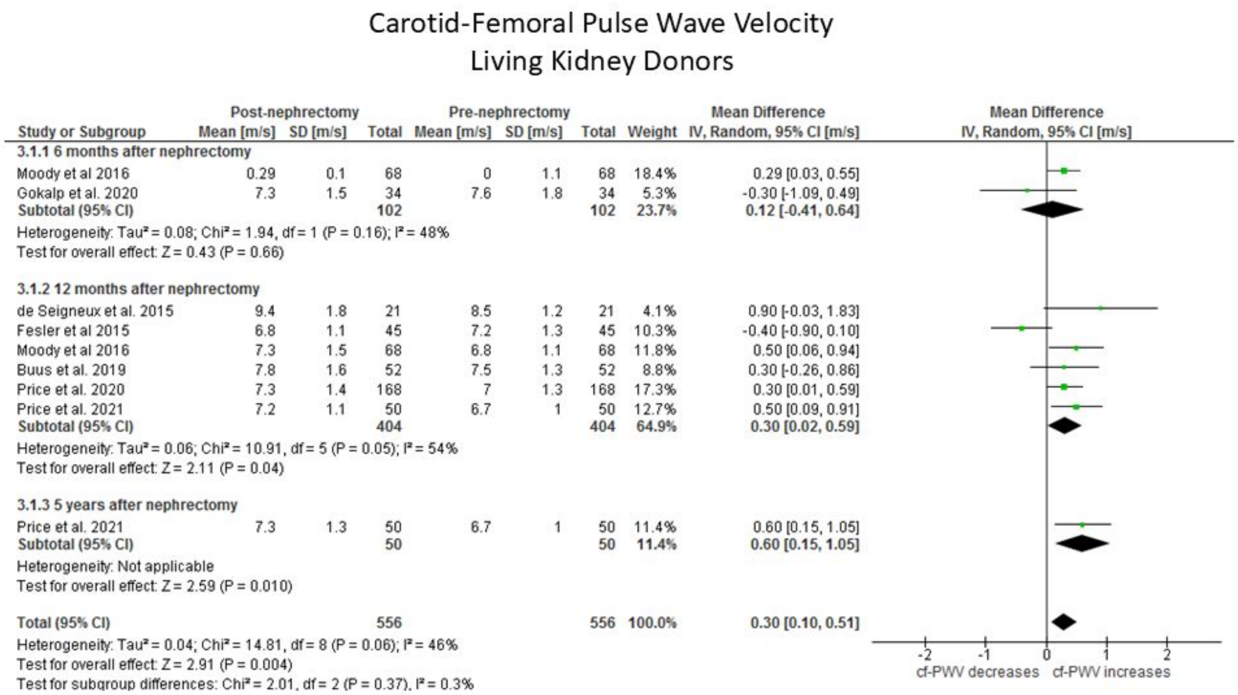

Figure S-5: Pooled effect estimates on the carotid-femoral Pulse Wave Velocity (cf-PWV) (m/s) in living kidney donors from before-to-after nephrectomy without adjusting for double counting errors (unadjusted-model). The pooled mean differences in the unadjusted model did not differ from the mean differences in the adjusted model where the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors.

Figure S-6

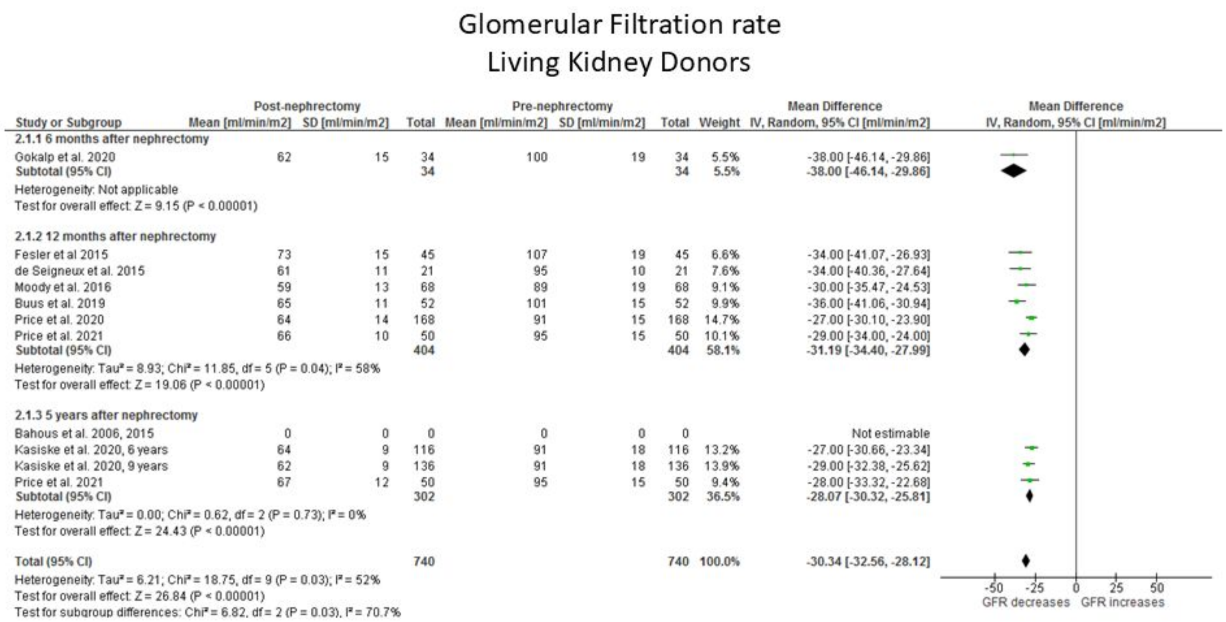

Figure S-6: Pooled effect estimates on the Glomerular Filtration rate (GFR) (ml/min/1.73 m^2) in living kidney donors from before-to-after nephrectomy without adjusting for double counting errors (unadjusted-model). The pooled mean differences in the unadjusted model did not differ from the mean differences in the adjusted model where the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors.

Figure S-7

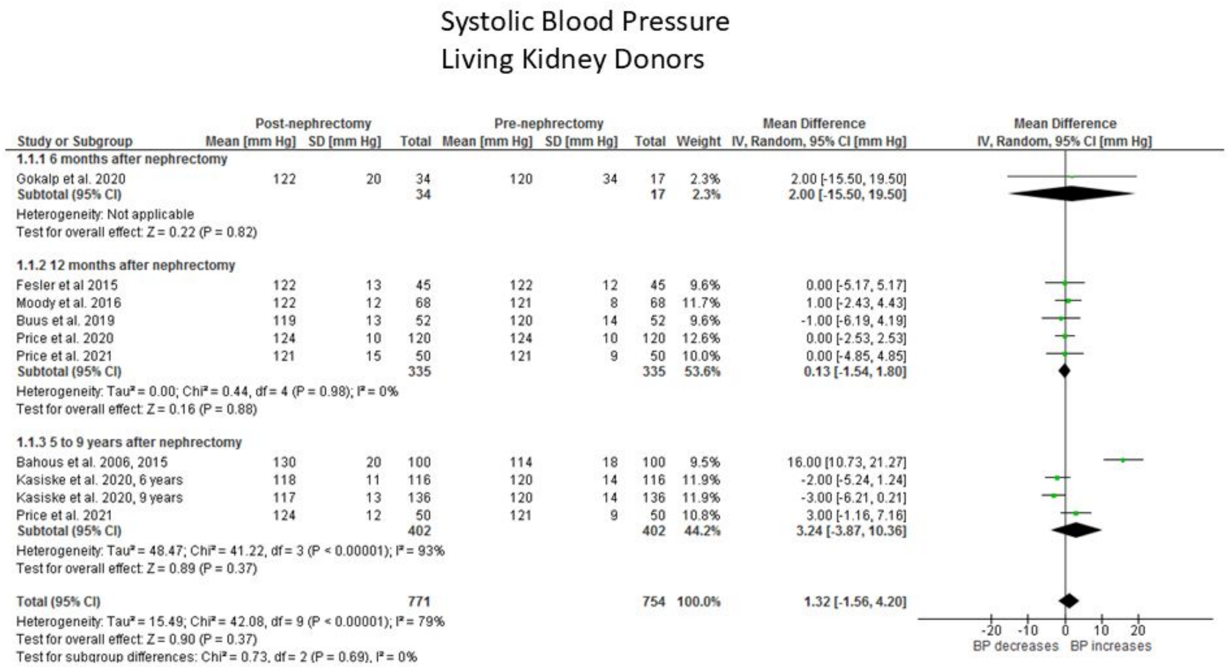

Figure S-7: Pooled effect estimates on the Systolic Blood Pressure (mm Hg) in living kidney donors from before-to-after nephrectomy without adjusting for double counting errors (unadjusted-model). The pooled mean differences in the unadjusted model did not differ from the mean differences in the adjusted model where the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors.

Figure S-8

Diastolic Blood pressure
Living Kidney Donors

Study or Subgroup	Post-nephrectomy Mean [mm Hg]	Post-nephrectomy SD [mm Hg]	Post-nephrectomy Total	Pre-nephrectomy Mean [mm Hg]	Pre-nephrectomy SD [mm Hg]	Pre-nephrectomy Total	Weight	Mean Difference IV, Random, 95% CI [mm Hg]
1.2.1 6 months after nephrectomy								
Gokalp et al. 2020	75	10	34	74	9	34	7.7%	1.00 [-3.52, 5.52]
Subtotal (95% CI)			**34**			**34**	**7.7%**	**1.00 [-3.52, 5.52]**
Heterogeneity: Not applicable								
Test for overall effect: Z = 0.43 (P = 0.66)								
1.2.2 12 months after nephrectomy								
Fesler et al 2015	70	8	45	70	9	45	9.3%	0.00 [-3.52, 3.52]
Moody et al. 2016	76	9	68	75	6	68	11.0%	1.00 [-1.57, 3.57]
Buus et al. 2019	75	8	52	74	8	52	10.1%	1.00 [-2.08, 4.08]
Price et al. 2020	79	8	120	79	8	120	11.9%	0.00 [-2.02, 2.02]
Price et al. 2021	74	10	50	73	7	50	9.5%	1.00 [-2.38, 4.38]
Subtotal (95% CI)			**335**			**335**	**51.7%**	**0.51 [-0.71, 1.74]**
Heterogeneity: Tau² = 0.00; Chi² = 0.64, df = 4 (P = 0.96); I² = 0%								
Test for overall effect: Z = 0.83 (P = 0.41)								
1.2.3 5 to 9 years after nephrectomy								
Bahous et al. 2006, 2015	82	12	100	69	19	100	7.8%	13.00 [8.60, 17.40]
Kasiske et al. 2020, 6 years	73	9	116	70	9	116	11.4%	3.00 [0.68, 5.32]
Kasiske et al. 2020, 9 years	74	10	136	70	9	136	11.5%	4.00 [1.74, 6.26]
Price et al. 2021	78	9	50	73	7	50	9.9%	5.00 [1.84, 8.16]
Subtotal (95% CI)			**402**			**402**	**40.6%**	**5.80 [2.47, 9.14]**
Heterogeneity: Tau² = 9.15; Chi² = 16.11, df = 3 (P = 0.001); I² = 81%								
Test for overall effect: Z = 3.41 (P = 0.0006)								
Total (95% CI)			**771**			**771**	**100.0%**	**2.70 [0.82, 4.58]**
Heterogeneity: Tau² = 6.69; Chi² = 37.15, df = 9 (P < 0.0001); I² = 76%								
Test for overall effect: Z = 2.81 (P = 0.005)								
Test for subgroup differences: Chi² = 8.53, df = 2 (P = 0.01), I² = 76.6%								

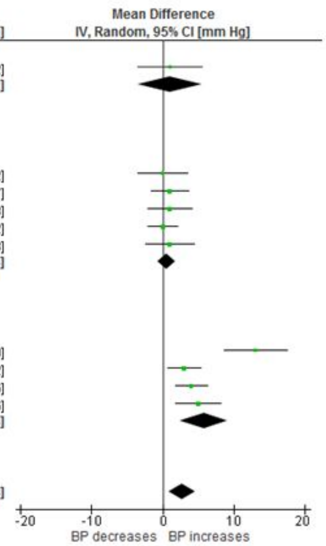

Figure S-8: Forest plots of the pooled effect estimates on Diastolic blood pressure in living kidney donors from before to after nephrectomy without adjusting for double counting errors (unadjusted-model). The pooled mean differences in the full model did not differ from the mean differences in the adjusted model where the number of living kidney donors allocated to each measurement was reduced by 50% to decrease "double counting" errors.

Figure S-9

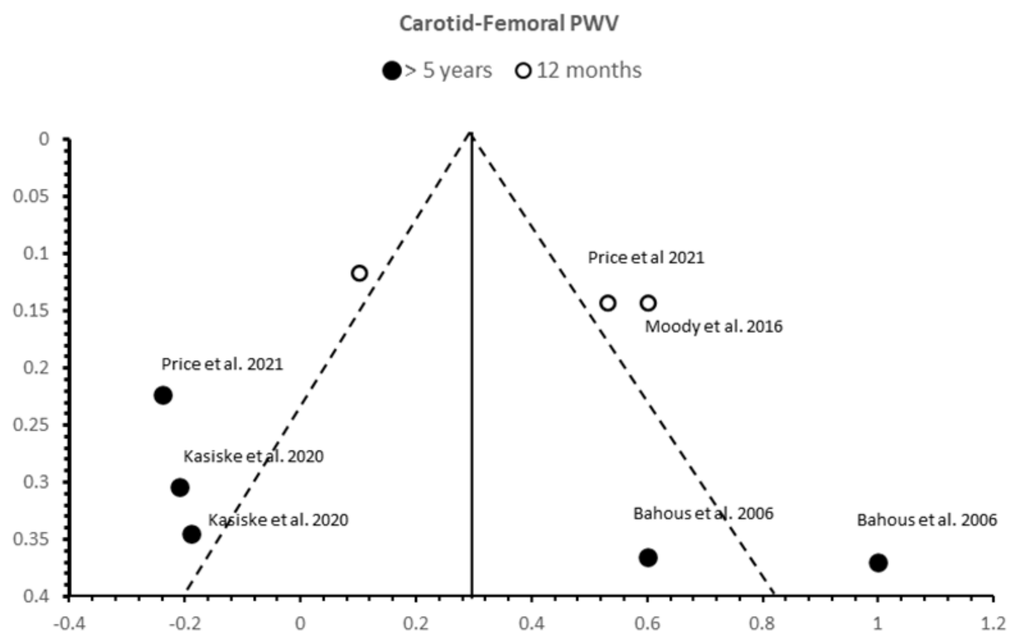

Figure S-9. Funnel plots of asymmetry in 5 studies who evaluated living kidney donors and controls. Data is stratified by time of follow up (12 months and > 5 years). Vertical black line represents the mean difference of the pooled effect estimates between kidney donors and controls (0.3 m/s). Dotted lines represent the 95% CI of the effect estimates. Filled circles represent studies > 5 years while non-filled circles those with measurements at 12 months.

Appendices

Appendix 1: Search strategy

Appendix 2: List of inclusion and exclusion criteria

Appendix 3: Summary of data extraction themes

Appendix 4: Complementary summary of study characteristics and country of origin.

Appendix 5: Comprehensive list of excluded studies.

Appendix 1. Search strategy.

Database: Embase Classic+Embase **<1947 to 2022 Dec 06>**, Ovid MEDLINE(R) ALL <1946 to Dec 06, 2022>, EBM Reviews - Cochrane Central Register of Controlled Trials <February 2020>
Search Strategy:
----
1 Nephrectomy/ (88255)
2 nephrectom*.tw,kw. (96086)
3 Kidney Transplantation/ and Living Donors/ (14605)
4 ((renal or kidney) adj3 (donor* or donation*)).tw. (32914)
5 or/1-4 (161070)
6 Vascular Stiffness/ (25452)
7 ((vascular or arter* or aort*) adj3 (stiff* or rigid*)).tw. (39922)
8 stiffness.tw,kw. (169270)
9 exp Pulse Wave Analysis/ (30543)
10 Cardio Ankle Vascular Index.tw,kw. (1710)
11 Augmentation index.tw,kw. (10033)
12 central pulse pressure.tw,kw. (1410)
13 Ankle-brachial index.tw,kw. (12896)
14 Ankle Brachial Index/ (13675)
15 (aort* adj2 (distensibilit* or elasticit*)).tw. (2677)
16 ((pulse or pulsation) adj2 (curve* or tracing* or wave*)).tw. (42852)
17 pulse wave.kw. (5300)
18 (pwv or apwv or bapwv or cfpwv).tw,kw. (20707)
19 (pulse adj2 (analys#s or velocit* or transit time)).tw. (37462)
20 (vascular stiff* or aortic stiff* or arter* stiff).kw. (1790)
21 ((decreased or reduced or diminished or lessened or lowered) adj3 ((vascular or aortic or arter*) adj compliance)).tw. (1257)
22 blood flow velocity/ (101381)
23 ((blood or circulation) adj2 (flow or rate) adj velocit*).tw. (21273)
24 (central adj (pulse or aortic or arterial) adj pressure).tw. (2881)
25 (central pressure or pulse pressure or pulse tension).tw,kw. (25275)
26 (central pulse pressure or blood flow velocit*).kw. (3437)
27 aasi.tw,kw. (560)
28 applanation tonomet*.tw,kw. (10330)
29 (sphygmocor* or vicorder*).tw,kw. (3318)
30 ((assess* or measur* or determin* or evaluat*) adj3 ((vascular or aortic or arter*) adj elasticit*)).tw. (656)
31 (Carotid adj3 intima-media thickness).tw. (23970)
32 Carotid-intima media thickness.kw. (2894)
33 Carotid artery ultrasonography.tw,kw. (292)
34 Carotid Arteries/dg (8914)
35 (Carotid arter* adj3 ultrasonograph*).tw. (1410)
36 (Ultrasonography, Doppler/ or Ultrasonography/) and Carotid Artery Diseases/ (4100)
37 calcinosis/ or exp vascular calcification/ (77074)
38 Vascular calcification.tw,kw. (11072)

39 vascular calcinosis.tw,kw. (40)
40 Flow mediated dilatation.tw,kw. (5904)
41 Brachial Artery/ and Vasodilation/ (6138)
42 brachial artery reactivity.tw,kw. (483)
43 or/6-42 (467264)
44 5 and 43 (1078)
45 exp animals/ not exp humans/ (10091587)
46 44 not 45 (692)
47 46 use medall (302) Medline
48 46 use cctr (13) Cochrane
49 nephrectomy/ or radical nephrectomy/ (91023)
50 nephrectom*.tw. (93900)
51 kidney donor/ (11405)
52 living donor/ and kidney transplantation/ (16482)
53 ((renal or kidney) adj3 (donor* or donation*)).tw. (32914)
54 49 or 50 or 51 or 52 or 53 (165157)
55 arterial stiffness/ (27294)
56 ((vascular or arter* or aort*) adj3 (stiff* or rigid*)).tw. (39922)
57 stiffness.tw. (166817)
58 pulse wave/ (26256)
59 Cardio Ankle Vascular Index.tw. (1685)
60 augmentation index/ (5503)
61 Augmentation index.tw. (9834)
62 Augmentation index.tw. (9834)
63 pulse pressure/ (313849)
64 central pulse pressure.tw. (1389)
65 ankle brachial index/ (13675)
66 Ankle-brachial index.tw. (12479)
67 (aort* adj2 (distensibilit* or elasticit*)).tw. (2677)
68 ((pulse or pulsation) adj2 (curve* or tracing* or wave*)).tw. (42852)
69 (pwv or apwv or bapwv or cfpwv).tw. (20649)
70 (pulse adj2 (analys#s or velocit* or transit time)).tw. (37462)
71 ((decreased or reduced or diminished or lessened or lowered) adj3 ((vascular or aortic or arter*) adj compliance)).tw. (1257)
72 blood flow velocity/ (101381)
73 ((blood or circulation) adj2 (flow or rate) adj velocit*).tw. (21273)
74 (central adj (pulse or aortic or arterial) adj pressure).tw. (2881)
75 (central pressure or pulse pressure or pulse tension).tw. (24926)
76 aasi.tw. (558)
77 applanation tonomet*.tw. (10187)
78 (sphygmocor* or vicorder*).tw. (3310)
79 ((assess* or measur* or determin* or evaluat*) adj3 ((vascular or aortic or arter*) adj elasticit*)).tw. (656)
80 arterial wall thickness/ (21793)
81 (Carotid adj3 intima-media thickness).tw. (23970)
82 (Carotid arter* adj3 ultrasonograph*).tw. (1410)

83 exp blood vessel calcification/ (19784)
84 vascular calcinosis.tw. (36)
85 Vascular calcification.tw. (10295)
86 dilatation/ and brachial artery/ (846)
87 Flow mediated dilatation.tw. (5753)
88 brachial artery reactivity.tw. (471)
89 or/55-88 (683344)
90 54 and 89 (3439)
91 exp animals/ not exp humans/ (10091587)
92 90 not 91 (1320)
93 92 use emczd (342) Embase
94 47 or 48 or 93 (657)
95 remove duplicates from 94 (524)
96 95 use medall (300) Medline
97 95 use emczd (221) Embase
98 95 use cctr (3) Cochrane

Appendix 2. List of inclusion and exclusion criteria.

PICO definitions	**Inclusion criteria**	**Exclusion criteria**
Population	Healthy subjects (≥18 years) that met standard criteria for kidney donation, who underwent unilateral simple nephrectomy and consented to measurements of carotid-femoral PWV before and/or after nephrectomy.	• Healthy subjects that underwent unilateral simple nephrectomy for other reason than kidney donation. • Children and adolescents with solitary kidney after unilateral nephrectomy.
Intervention	Open or laparoscopic unilateral simple nephrectomy	• Unilateral nephrectomy combined to other surgical procedures
Comparator	• Healthy adult subjects (≥18 years) with measurements of carotid-femoral PWV who participated as healthy comparative controls in kidney donor studies. • Healthy subjects from the general population with measurements of carotid-femoral PWV included in reference studies.	• Kidney recipients
Outcome	• Changes in carotid-femoral PWV	• Other indices of vascular stiffness (augmentation index, carotid-radial PWV, brachial-ankle PWV, cardio-ankle vascular index, carotid-intima media thickness, calcification index)
Study design	• Prospective non-randomised (cohort, case–control, case series and before-and-after studies) and retrospective studies if 10 or more participants have been included in the primary analysis. • Articles reported in English, French, Italian, Portuguese and Spanish languages.	• Paediatric and non-human studies • Narrative reviews • In vitro or mathematical modelling reports. • Duplicates • Sub-studies of previously published trials.

PWV: pulse wave velocity

Appendix 3. Summary of data extraction themes.

The process of data extraction included the following themes:

a) ***Study characteristics*** included authors, country of origin, publication date, title, language of publication, study design, inclusion and exclusion criteria, pre-and post-nephrectomy time measurement points, duration of follow-up, study design, use of a control group and individual study inclusion and exclusion criteria.

b) ***Characteristics of participants*** including sample size for donors and controls, proportion of female and males, donor's age at the time of nephrectomy and testing, control's age at the time of initial testing (i.e., recruitment) and at follow up, body mass index for donors and controls. If available, we documented the participant's clinical history (donors and controls) including the proportion of subjects with hypertension, cardiovascular disease, diabetes, hypercholesterolemia, obesity, history of cancer, smoking, as well as the proportion of subjects receiving antihypertensive therapy and type of medication [i.e. Angiotensin converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARB), beta or alpha blockers, calcium channel blockers, diuretics, statins and aspirin]. In addition, if accessible, we estimated the proportion of recipient-related and non-recipient related donors and controls, and the ethnicity of participants.

c) ***Renal chemistry profile*** including: plasma glucose, plasma creatinine, calculated creatinine clearance (MDRD or CKD-EPI), urinary albumin/creatinine and blood urea nitrogen in kidney donors (before and after nephrectomy) and healthy controls (recruitment and follow-up).

d) ***Carotid-Femoral PWV (cf-PWV)*** including instrumentation and technique of measurement, absolute values and post-donation changes relative to their pre-donation baseline, adjusted or non-adjusted values and type of adjusting factor (i.e., mean arterial pressure, heart rate).

e) **H*emodynamic characteristics*** including systolic, diastolic and mean blood pressure, heart rate and pulse pressure, techniques of measurement (i.e., office, 24 hours monitoring); absolute values and post-donation changes relative to pre-donation baseline.

Appendix 4. Complementary summary of study characteristics and country of origin.

Most studies (n=7) were completed in Europe, one in the USA and another in the Middle East. All studies reported that the process of screening and selection for kidney donors followed institutional protocols. In 5 studies, participants in the control group were screened as if they would be fit for kidney donation, but they were not actual donors.[6-12] Two of these studies [6,7,10] reported that 90.0% and 21.2% of healthy controls, respectively, were first-degree relatives of recipients, but only one study[10] documented that 51.5% of donors were biologically related to the recipients. Only one study provided information on clinical outcomes [6,7] and reported that 4.9% (5/101) of donors developed at least one adverse cardiovascular event (coronary, cerebral, aortic or peripheral artery disease) after nephrectomy (follow-up range: 43 to 219 months).

Appendix 5: List of excluded studies.

ORN	UI	TITLE	CITATION
1	33399510	Noninvasive Staging of Liver Fibrosis Using 5-Minute Delayed Dual-Energy CT: Comparison with US Elastography and Correlation with Histologic Findings.	Radiology. 298(3):600-608, 2021 Mar.
2	32243494	Circulating uromodulin inhibits vascular calcification by interfering with pro-inflammatory cytokine signalling.	Cardiovascular Research. 117(3):930-941, 2021 Feb 22.
3	31899189	Calcification of the cavernosal bodies may be responsible for development of erectile dysfunction in uremic apolipoprotein E deficient (apoE-/-) mice.	Revista Internacional de Andrologia. 19(1):25-33, 2021 Jan-Mar.
4	33545931	Renal tissue elasticity by acoustic radiation force impulse: A prospective study of healthy kidney donors.	Medicine. 100(3):e23561, 2021 Jan 22.
5	634413230	Effects of living kidney donation on arterial stiffness: A systematic review protocol.	BMJ Open. 11 (3) (no pagination), 2021. Article Number: e045518. Date of Publication: 08 Mar 2021.
6	2007884313	Concomitant Aorto-Caval Reconstruction for Inferior Vena Cava Leiomyosarcoma.	Annals of Vascular Surgery. 70 (pp 567.e13-567.e17), 2021. Date of Publication: January 2021.
7		Pulse pressure variation guided fluid therapy during kidney transplantation: a randomized controlled trial	Brazilian journal of anesthesiology. 2020.
8	33367746	The receptor activator of nuclear factor kappaBeta ligand receptor leucine-rich repeat-containing G-protein-coupled receptor 4 contributes to parathyroid hormone-induced vascular calcification.	Nephrology Dialysis Transplantation. 2020 Dec 26
9	32398767	The protective effects of renin-angiotensin system compons on vascular calcification.	Journal of Human Hypertension. 2020 May 12
10	33377886	Kidney Hyperfiltration After Nephrectomy: A Mechanism to Restore Kidney Function in Living Donors.	Acta Medica Indonesiana. 52(4):413-419, 2020 Oct.
11	33293769	Effects of dietary fiber on vascular calcification by repetitive diet-induced fluctuations in plasma phosphorus in early-stage chronic kidney disease rats.	Journal of Clinical Biochemistry & Nutrition. 67(3):283-289, 2020 Nov.

12	33192538	OGT-Mediated KEAP1 Glycosylation Accelerates NRF2 Degradation Leading to High Phosphate-Induced Vascular Calcification in Chronic Kidney Disease.	Frontiers in Physiology. 11:1092, 2020.
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