ORIGINAL ARTICLE

# Impact of donor obesity on allograft outcomes after kidney transplantation adjusted for kidney donor profile index – a national cohort study

Piyavadee Homkrailas[1,2], Marcelo Sampaio[1], Nakul Datta[1], Gabriel M. Danovitch[1] & Suphamai Bunnapradist[1]

1 Division of Nephrology, Department of Medicine, Kidney and Pancreas Transplant Research Center, David Geffen School of Medicine at UCLA, Los Angeles, CA, USA

2 Division of Nephrology, Department of Medicine, Bhumibol Adulyadej Hospital, Bangkok, Thailand

**Correspondence**
Suphamai Bunnapradist MD, MS, 1015 Gayley Ave., Suite 220, Los Angeles, CA 90024, USA.
Tel.: (310) 794-8516;
fax: (310) 794-8589;
e-mail: bunnapradist@mednet.ucla.edu

## ABSTRACT

Obesity in deceased kidney donors is a known risk factor for poor allograft outcomes. The Kidney Donor Profile Index (KDPI) has been introduced to predict graft survival in deceased donor kidney transplantation (DDKT). Obesity, however, is not included in KDPI. We study the impact of donor obesity on DDKT outcomes after adjusting for organ quality by KDPI. The Organ Procurement Transplantation Network/United Network for Organ Sharing (OPTN/UNOS) data of DDKT from 2005 to 2017, with donor BMI $\geq$ 18.5 kg/m$^2$ and weight >80 kg were included. There was a total of 66 382 DDKTs with 10 917 death-censored graft failures. For KDPI $\leq$ 30%, the 10-year death-censored graft survival (DCGS) rates among donor BMI < 30, 30–35, 35–40, 40–45 and $\geq$45 kg/m$^2$ groups were 75.9%, 75.4%, 76.1%, 74.9% and 79.6%, respectively. For KDPI > 30%, 10-year DCGS rates were 67.5%, 66.1%, 65.9%, 62.6% and 63.2%, respectively. After adjusting for known confounding factors including KDPI, donor obesity was not independently associated with an increased risk for graft failure. In DDKT with donor weight >80 kg, donor obesity was not associated with a lower long term DCGS compared to non-obesity when KDPI $\leq$ 30%.

*Transplant International* 2021; 34: 681–688

**Key words**
allograft outcomes, donor obesity, kidney donor profile index, kidney transplantation

Received: 2 September 2020; Revision requested: 6 October 2020; Accepted: 18 January 2021; Published online: 7 March 2021

## Introduction

In the last twenty years, there has been a significant increase in the prevalence of obesity (defined by body mass index; BMI $\geq$ 30 kg/m$^2$) from 30.5% to 42.2% in the United States [1]. The prevalence of severe obesity (defined by BMI $\geq$ 40 kg/m$^2$) has also increased from 4.7% to 9.2% [1]. As the obesity epidemic worsens, 30% of deceased kidney donors are obese (based on The Organ Procurement Transplantation Network; OPTN data as of January 12, 2020). Obesity is associated with various structural, hemodynamic, and metabolic alterations in the kidney. Kidneys from obese individuals are associated with "obesity-related glomerulopathy" defined morphologically as glomerulomegaly (glomerular hypertrophy), focal segmental glomerulosclerosis, and focal podocyte foot process effacement [2,3]. Donor obesity is also associated with surgical technical difficulties resulting in increased nephrectomy operation times, and cold and warm ischemic times [4–6].


doi:10.1111/tri.13826

These factors are known to have a negative impact on delayed graft function (DGF) rates and reduced long-term graft survival after deceased donor kidney transplantation (DDKT) [7–11].

Previous studies have shown that donor obesity is associated with DGF, primary non-function (PNF), acute rejection and inferior long-term kidney allograft survival [4,12,13]. Naik *et al.* [13] demonstrated that with both living and deceased kidney donors, having a BMI $\geq$ 35 kg/m$^2$, was associated with an increased risk of long-term allograft failure (hazard ratio 1.22; 95% CI 1.14–1.31, $P < 0.01$). Alhamad *et al.* [14] study of donors after brain death (DBD) found that a BMI 35–50 kg/m$^2$ was associated with a significantly increased risk of kidney allograft failure compared with a BMI 20–25 kg/m$^2$ (hazard ratio 1.36; 95% CI 1.02–1.82, $P = 0.04$) while a donor BMI 30–35 kg/m$^2$ did not impact graft survival.

The Kidney Donor Profile Index (KDPI), is a scoring system based on ten donor factors. It is used as a measure of the quality of deceased donor kidneys and has been shown to be predictive of both short- and long-term graft survivals [15–19]. The KDPI is derived by first calculating the Kidney Donor Risk Index (KDRI), which maps from a relative risk scale to a cumulative percentage scale. The reference population used for this mapping is all deceased donors in the United States with a kidney recovered for the purpose of transplantation in the prior calendar year. Lower KDPI values are associated with increased donor quality and expected longevity [20]. Kidneys with KDPI $\leq$ 20% have 10-year graft survival rate 65% compared to 35% in kidneys with KDPI > 85% (based on OPTN data as of May 10, 2019). Although obesity is not included in the current KDPI scoring system, donor weight <80 kg results in increased KDPI whereas donor weight $\geq$80 kg does not affect KDPI score. According to the negative impact of donor obesity, we hypothesize that obese DDKTs are associated with inferior graft survival compared with non-obese DDKTs, even after adjusted by KDPI. Here we study the impact of donor obesity on kidney transplant outcomes. We evaluated the outcomes of allograft survival, DGF, PNF, and serum creatinine at the first-year post-transplant.

## Materials and methods

### Data source and study population

The Organ Procurement Transplantation Network/United Network for Organ Sharing (OPTN/UNOS) database as of June 10, 2019 was used in this study. All kidney transplant recipients from deceased donors between January 2005 and December 2017 were included. Recipients with multiple organ transplants and donors with missing BMI, KDPI or a BMI < 18.5 kg/m$^2$ were excluded. There is a collinearity issue when using KDPI score which contains donor weight and height to adjust the effect of donor BMI on allograft outcomes. In KDPI score calculation, donor weight <80 kg results in increased KDPI while donor weight $\geq$80 kg is not included in the calculation. For this reason, donors with body weight $\leq$80 kg were excluded from the study. This study was institutional review board exempt due to its use of publicly available data and absence of identification of individual donors and recipients. We grouped donors based on their calculated BMI as follows: <30, 30–35, 35–40, 40–45 and $\geq$45 kg/m$^2$.

### Outcome measures

The study population was analyzed to determine the impact of donor obesity adjusted for KDPI on kidney graft outcomes. The primary outcome of interest was death-censored graft survival (DCGS; defined as the time from transplant to the earliest of allograft loss, kidney re-transplantation, re-initiation of dialysis, or loss to followed up with a functioning graft, censored for death). Secondary outcomes included DGF (defined as dialysis within the first week post-transplantation), PNF (defined as permanent loss of allograft function starting immediately after transplantation), and serum creatinine at first-year post-transplant.

### Statistical analysis

Donor and recipient characteristics were evaluated. Variables were analyzed by using median and interquartile range for continuous variables, and using proportions for categorical variables. Demographic differences and post-transplant outcomes related to kidney allograft between groups were compared. Post-transplant outcomes related to kidney allograft were identified and compared between the study groups using Kruskal-Wallis or Pearson's chi-squared test as appropriate. Serum creatinine levels at the first-year post-transplant were only available for individuals who were alive, being followed clinically, and did not experience graft failure at one-year followed-up. The Kaplan-Meier method was used to generate survival curves, and log rank test was used to compare graft



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survival between groups. Kaplan-Meier survival curves were plotted and compared between groups of donor BMI for each level of KDPI (0, 1–10, 10–20, 20–30, 30–40, 40–50, 50–60, 60–70, 70–80, 80–90 and 90–100). Pairwise correlations between KDPI and death censored graft failure were tested. There were small negative correlations when KDPI ≤ 30% while there were small positive correlations when KDPI > 30%. From both Kaplan-Meier plots and pairwise correlations, we separated survival curves into KDPI ≤ 30% and >30%. In regression analysis, donor BMI was modeled with restricted cubic splines with five knots located at 25, 30, 35, 40 and 45. Cox-proportional hazards model was used to calculate hazards ratio (HR) and 95% confidence interval (CI) to examine risks associated with graft loss. Logistic regression model was used to calculate odds ratio (OR) and 95% CI to examine risks associated with DGF and PNF. Donor BMI of 25 kg/m$^2$ was used as a reference. Due to a low number of donors with a BMI > 50 kg/m$^2$ (1.8%), we grouped donors with a BMI ≥ 50 kg/m$^2$ together in the regression analysis plot. In the multivariable model, we adjusted for (i) donor gender and KDPI (ii) recipient age, gender, ethnicity, diabetes, cytomegalovirus (CMV) status, hepatitis C virus (HCV) status and previous kidney transplant and (iii) transplant variables which included cold ischemic time, panel reactive antibody (PRA) and human leukocyte antigen (HLA) mismatch. STATA version 13 (Statacorp, College Station, TX, USA) was used in all statistical analyses.

## Results

There were 149 209 DDKTs between January 1, 2005 and December 31, 2017. We excluded 82 827 DDKTs from the study (72 916 had donor weight ≤80 kg, 8977 had donor BMI < 18.5 kg/m$^2$, 725 had missing KDPI values and 209 had missing donor BMI values). A total of 66 382 DDKTs were included in our analysis. Median follow up time was 4.1 (0–14.3) years. Among these, 11 204 (16.9%) recipients died and 10 917 (16.5%) recipients experienced death censored graft failure of whom 1912 were attributed to repeat kidney transplant. There were 6852 (10.3%) DDKTs receiving kidneys from severely obese donor (BMI ≥ 40 kg/m$^2$), which was increased from 8.8% in year 2005 to 12.1% in year 2017. The distribution of donor BMI is shown in Fig. 1. Baseline donor, recipient and transplant characteristics are shown in Table S1. There were higher known high-risk donor factors resulting in a higher KDPI within groups of obese donors. These factors included an older age, a lower height, female gender, black ethnicity, history of hypertension, diabetes, cerebrovascular cause of death and donation after cardiac death (DCD). Recipient and transplant characteristics were not clinically different among the groups.

### Graft survival

Among donors with a BMI < 30, 30–35, 35–40, 40–45 and ≥45 kg/m$^2$, respectively, there were 4475 (15.7%),

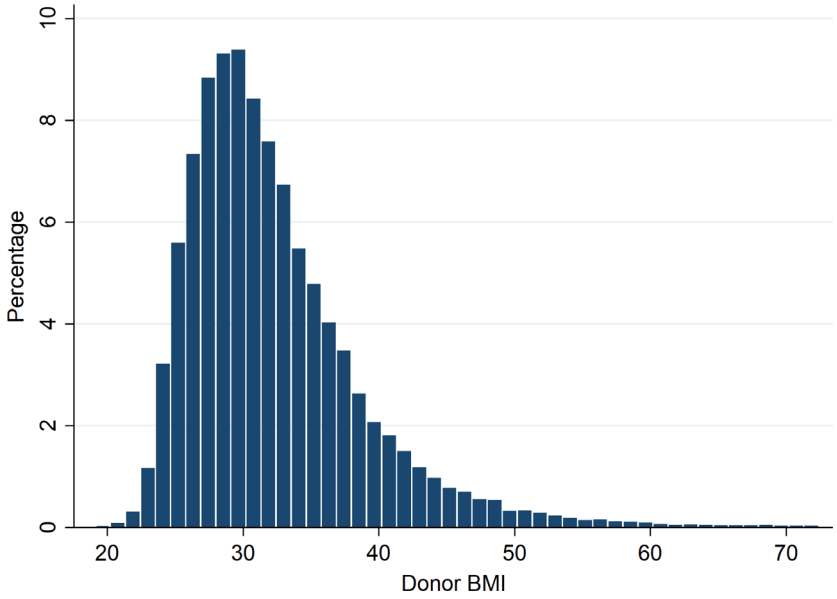
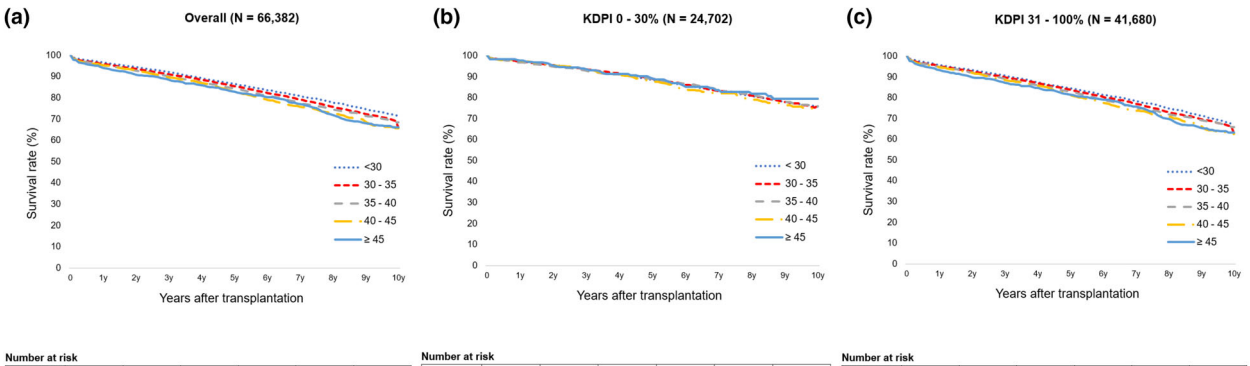



**Figure 1** Distribution of donor body mass index.




Number at risk

| BMI (kg/m2) | 0 | 2y | 4y | 6y | 8y | 10y |
|---|---|---|---|---|---|---|
| <30 | 28,441 | 21,645 | 14,911 | 9,525 | 5,597 | 2,805 |
| 30 - 35 | 20,855 | 15,564 | 10,491 | 6,480 | 3,623 | 1,702 |
| 35 - 40 | 9,906 | 7,342 | 4,848 | 2,921 | 1,588 | 730 |
| 40 - 45 | 3,995 | 2,881 | 1,857 | 1,105 | 613 | 263 |
| ≥45 | 2,797 | 1,959 | 1,229 | 751 | 401 | 183 |

Number at risk

| BMI (kg/m2) | 0 | 2y | 4y | 6y | 8y | 10y |
|---|---|---|---|---|---|---|
| <30 | 13,905 | 10,834 | 7,520 | 4,961 | 2,958 | 1,523 |
| 30 - 35 | 6,747 | 5,169 | 3,553 | 2,231 | 1,314 | 645 |
| 35 - 40 | 2,533 | 1,932 | 1,332 | 841 | 484 | 236 |
| 40 - 45 | 928 | 679 | 462 | 276 | 170 | 80 |
| ≥45 | 519 | 391 | 233 | 138 | 84 | 39 |

Number at risk

| BMI (kg/m2) | 0 | 2y | 4y | 6y | 8y | 10y |
|---|---|---|---|---|---|---|
| <30 | 14,536 | 10,811 | 7,391 | 4,564 | 2,639 | 1,282 |
| 30 - 35 | 14,108 | 10,395 | 6,938 | 4,249 | 2,309 | 1,057 |
| 35 - 40 | 7,373 | 5,410 | 3,516 | 2,080 | 1,104 | 494 |
| 40 - 45 | 3,067 | 2,202 | 1,395 | 829 | 443 | 183 |
| ≥45 | 2,278 | 1,568 | 996 | 613 | 317 | 144 |

**Figure 2** Kaplan-Meier plots of death-censored graft survival according to donor body mass index (BMI). (a) Overall donors. (b) Donors with kidney donor profile index (KDPI) 0–30%. (c) Donors with KDPI 31–100%.

3467 (16.5%), 1712 (17.2%), 732 (18.2%) and 531 (18.8%) recipients experienced graft failure during the followed-up time. Kaplan-Meier plots of overall DCGS are shown in Fig. 2a. Ten-year DCGS rates were 71.8% (95% CI 70.8–72.7), 69.2% (95% CI 68.0–70.3), 68.6% (95% CI 66.9–70.2), 65.6% (95% CI 62.8–68.3) and 66.1% (95% CI 62.7–69.3), respectively. There were 24 702 DDKT recipients (37.2%) receiving a kidney with KDPI ≤ 30% and 41 680 DDKT recipients (62.8%) receiving a kidney with KDPI > 30%. There was no difference in DCGS between groups if KDPI was 0–30%, which were 75.9% (95% CI 74.8–77.1), 75.4% (95% CI 73.5–77.2), 76.1% (95% CI 72.9–78.8), 74.9% (95% CI 69.7–79.4) and 79.6% (95%CI 72.8–84.8), respectively (Fig. 2b). A significantly lower DCGS among severely obese donor groups was observed if KDPI was 31–100%, which were 67.5% (95% CI 66.1–68.8), 66.1% (95% CI 64.5–67.5), 65.9% (95% CI 63.8–67.8), 62.6% (95% CI 59.2–65.8) and 63.2% (95% CI 59.3–66.7), respectively (Fig. 2c). Comparing with donor BMI 25 kg/m$^2$, a higher donor BMI was associated with an increased risk for graft failure in an unadjusted cox-proportional hazards model which HR were 1.12 (95% CI 1.05–1.18), 1.22 (95% CI 1.15–1.30), 1.31 (95% CI 1.22–1.44), 1.37 (95% CI 1.28–1.48) and 1.43 (95% CI 1.28–1.60), respectively, among donors with a BMI 30, 35, 40, 45, ≥50 kg/m$^2$ (Fig. 3a). After adjustment by KDPI (Fig. 3b) and KDPI along with other factors as described in the methods (Fig. 3c), donor obesity was not associated with an increased risk for graft failure if donor weight was >80 kg.

## Delayed graft function

Overall incidence rate of DGF was 29.0%. There was a higher incidence of DGF among donors with higher BMI which were 25.5%, 30.1%, 32.8%, 33.3% and 35.5% among donor BMI < 30, 30–35, 35–40, 40–45 and ≥45 kg/m$^2$ groups, respectively (Table 1). Comparing with donor BMI 25 kg/m$^2$, a higher donor BMI was associated with a significantly increased risk for DGF (Fig. 4a,b).

## Primary non-function

Overall incidence rate of PNF was 1.3% which has been decreasing by year of DDKT from 1.9% in 2005 to 0.8% in 2017. The incidence of PNF among donor BMI < 30, 30–35, 35–40, 40–45 and ≥45 kg/m$^2$ groups were 1%, 1.3%, 1.4%, 1.4% and 2.3%, respectively (Table 1). After adjustment by KDPI along with other factors, a higher donor BMI was associated with a significantly lower risk of PNF compared to donor BMI of 25 kg/m$^2$ (Fig. 4c,d).

## Serum creatinine at 1-year post-transplant

There were no clinical differences in median serum creatinine at 1-year after DDKT which were 1.3 (IQR 1–1.6), 1.3 (1.1–1.7), 1.3 (1.1–1.7), 1.3 (1.1–1.7) and 1.4 (1.1–1.7) mg/dl among donors with a BMI of <30, 30–35, 35–40, 40–45 and ≥45 kg/m$^2$ groups (Table 1).



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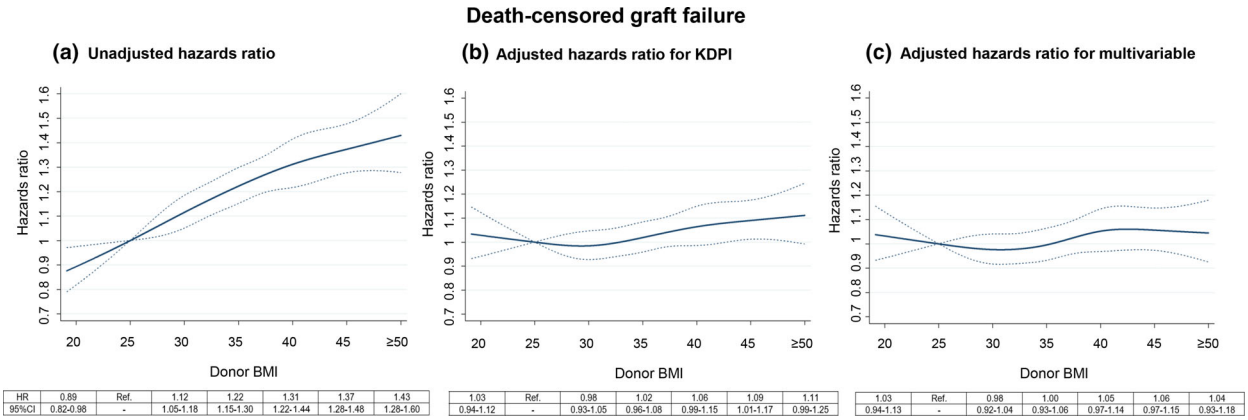


**Figure 3** Risk of donor body mass index (BMI) on death censored graft failure. Donor BMI was modeled with restricted cubic splines with fives knots located at 25, 30, 35, 40, and 45. Hazards ratio (HR) were plotted against donor BMI, and 95% confidence intervals were included (dot-line). Donor BMI of 25 was used as a reference. (a) Unadjusted HR. (b) Adjusted HR for Kidney Donor Profile Index (KDPI). (c) Adjusted HR for donor variables (gender and KDPI), recipient variables (age, ethnicity, diabetes, cytomegalovirus status, hepatitis C virus status, previous kidney transplant) and transplant variables (cold ischemic time, panel reactive antibody and human leukocyte antigen mismatch).

**Table 1.** Post-transplant outcomes related to donor BMI.

| Outcomes | BMI < 30 kg/m$^2$ *n* = 28 575 (43.0%) | BMI 30–35 kg/m$^2$ *n* = 20 985 (31.6%) | BMI 35–40 kg/m$^2$ *n* = 9970 (15.0%) | BMI 40–45 kg/m$^2$ *n* = 4020 (6.1%) | BMI ≥ 45 kg/m$^2$ *n* = 2828 (4.3%) |
|---|---|---|---|---|---|
| Delayed graft function, *n* (%) | 7301 (25.5) | 6324 (30.1) | 3273 (32.8) | 1341 (33.3) | 1003 (35.5) |
| Primary non-function, *n* (%) | 298 (1.0) | 275 (1.3) | 140 (1.4) | 55 (1.4) | 66 (2.3) |
| Remaining study population at 1-year followed up, *n* (%) | 23 608 (82.6%) | 18 051 (86.0%) | 8701 (87.3%) | 3525 (87.7%) | 2436 (86.1%) |
| Median serum creatinine at 1-year post-transplant of the remaining study population, mg/dl (IQR) | 1.3 (1–1.6) | 1.3 (1.1–1.7) | 1.3 (1.1–1.7) | 1.3 (1.1–1.7) | 1.4 (1.1–1.7) |

BMI, body mass index; IQR, interquartile range.

## Discussion

Using OPTN/UNOS data of DDKTs from donors with a body weight >80 kg between 2005 and 2017, donors with a higher BMI were more likely to have risk factors resulting in a higher KDPI. There was no difference in 10-year DCGS among donor BMIs if KDPI was ≤30% whereas there were lower DCGS rates among severely obese donors if KDPI was >30%. After adjusting for known confounding factors including KDPI, donor obesity was not independently associated with an increased risk for graft failure.

There have been previous studies illustrating association of donor BMI with long-term graft outcomes. A US-based registry of 97 090 DDKTs observed no association between donor BMI and DCGS in DBD donors. However, DCD donors with BMI ≥ 45 kg/m$^2$ had a significantly increased risk of graft failure [4]. A study from US-based registry of 118 734 DDKT and 84 377 living donor kidney transplants (LDKT) observed the effect of donor BMI on outcomes. They showed a significant and graded increase in graft failure risk among overweight (BMI 25–30 kg/m$^2$), mildly obese (BMI 30–35 kg/m$^2$) and very obese (BMI > 35 kg/m$^2$) in both living and deceased donor types [13]. A study that included 9916 simultaneous pancreas-kidney transplantation from DBD donors demonstrated that only donor BMI 35–50 kg/m$^2$ was associated with significantly

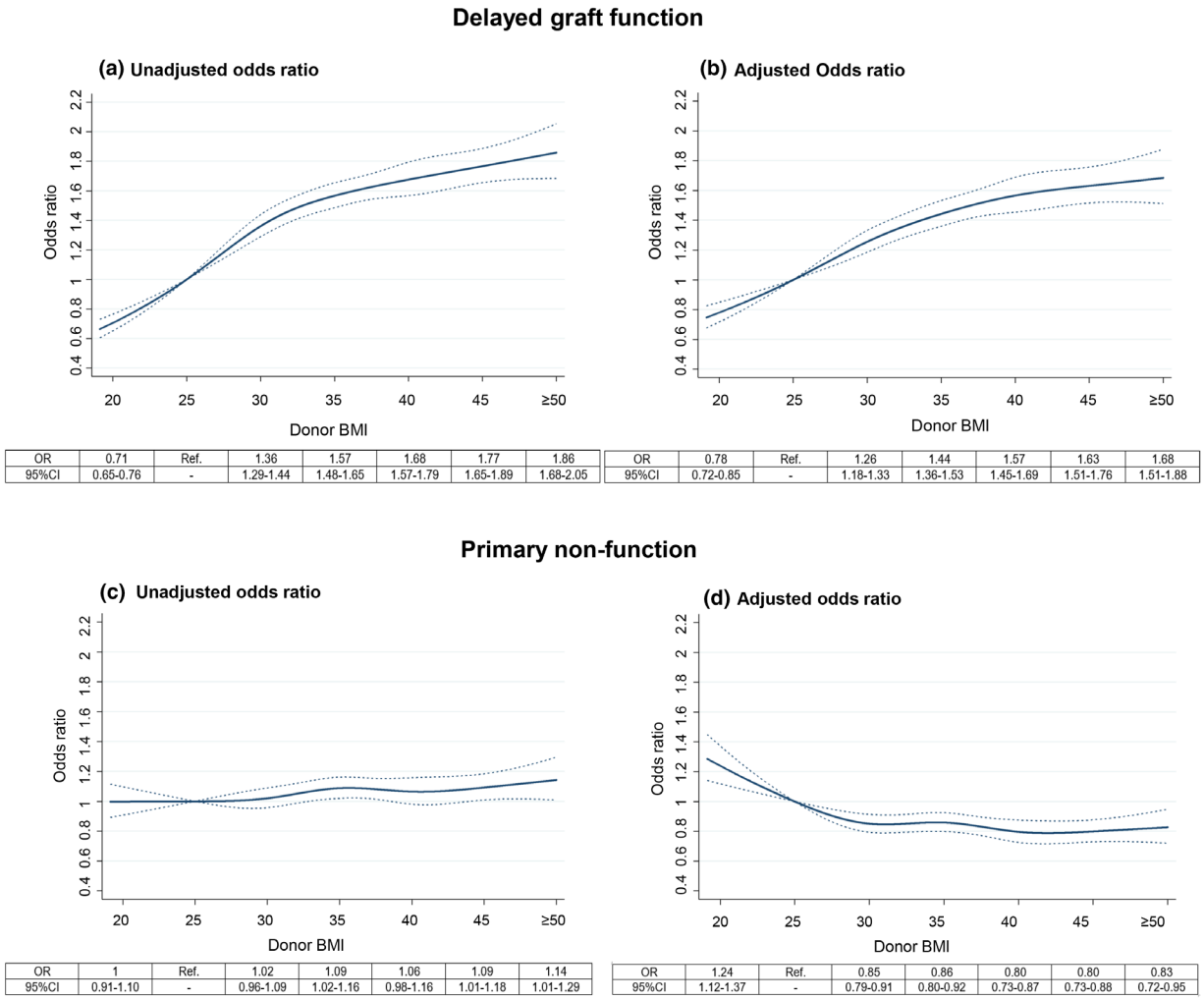


**Figure 4** Risk of donor body mass index (BMI) on delayed graft function (DGF) and primary non-function (PNF). Donor BMI was modeled with restricted cubic splines with fives knots located at 25, 30, 35, 40, and 45. Odds ratio (OR) were plotted against donor BMI, and 95% confidence intervals were included (dot-line). Donor BMI of 25 was used as a reference. (a) Unadjusted odds ratio (OR) for DGF. (b) Adjusted OR for DGF. (c) Unadjusted OR for PNF (d) Adjusted OR for PNF. Multivariable adjustment included donor variables (gender and KDPI), recipient variables (age, ethnicity, diabetes, cytomegalovirus status, hepatitis C virus status, previous kidney transplant) and transplant variables (cold ischemic time, panel reactive antibody and human leukocyte antigen mismatch).

higher risk of kidney graft failure while donor BMI 30–35 kg/m$^2$ did not impact graft survival [14]. These studies showed the negative impact of donor obesity on graft survival among various donor types. Despite that, DBD donor obesity affected graft survival differently between the studies. The causes of graft failure are complex and are often be multifactorial, which include pre-transplant, peri-transplant and post-transplant factors. Organ quality is one of the most important pre-transplant factors which is currently evaluated by the KDPI scoring system. Increasing KDPI was a more powerful predictor of DGF and graft survival than using DBD or DCD status [21]. In distinction from previous studies, we used KDPI to determine the deceased donor organ quality, to demonstrate the actual impact of donor obesity on graft survival.

Several factors related to donor obesity might have contributed to effect the allograft outcomes. First, obesity associated underlying pathology made these grafts more prone to the ischemic and immune injury. Obesity, even with or without metabolic syndrome was a potent risk factor for the development of kidney disease [22,23]. Compensatory glomerular hyperfiltration, proteinuria and obesity related glomerulopathy were reported in association with obesity [24]. However, there was no data supporting how long the obesity persists until structural kidney damage occurs. Histologic evaluation at organ recovery could provide this



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information. Secondly, cold and warm ischemic time are known to have a negative impact on both DGF and DCGS following DDKT. In obese donors, surgical technical challenges in performing nephrectomy may result in longer operation time and warm ischemic time [4,12]. Thirdly, organs from severely obese donors may be more difficult to cool since adipose tissue acts as an thermal insulator and poor conductor, thus proving superior thermal properties [25].

Among the higher quality kidneys (KDPI < 30%), we found that obese donor kidneys were not associated with a significantly lower DCGS compared to non-obese donor kidneys. A younger age and fewer risk factors in these donors may lead to less kidney structural damage resulting in the same long-term graft survival regardless of an obesity status. In contrast, there were more donor risk factors among the lower quality kidneys (KDPI > 30%). Older obese donors with hypertension or diabetes may have a metabolic syndrome or other comorbidities which are not included in the KDPI. These additional comorbidities may result in more kidney damage and a greater tendency to ischemic injury during kidney transplant, which in turn affect long-term graft survival. DGF was impacted by donor obesity regardless of KDPI level. After adjustment for donor factors including KDPI, recipient factors and transplant factors including cold ischemic time, donor obesity was an independent risk for DGF. Warm ischemic time may play a significant role among these donors [4–6]. Donor obesity, however, was not associated with an increased risk for PNF. In the past, the most common causes of PNF were acute rejection and surgical complications [26]. Current induction therapy and advanced surgical technique have led to a decrease an incidence of PNF. Overall donor quality, not only donor obesity, is associated with PNF.

There are limitations to a retrospective database study. First, there was missing kidney biopsy data at organ recovery (48.65% missing data). Because subtle histologic changes could occur despite normal kidney function, we could not demonstrate the association between donor obesity, other additional comorbidities, percentage of kidney glomerulosclerosis and allograft outcomes. Second, the definition of DGF was based on the need for dialysis within the first week after kidney transplant. This standard epidemiologic definition from the registry missed the milder degree of graft dysfunction or slow graft function that made DGF rates appear to be lower. Future prospective studies including histologic change at organ recovery and the recognition of mild degree of graft dysfunction would provide more detail about the effect of obesity and graft outcomes.

In conclusion, we found that DDKTs from obese donors were not associated with a lower DCGS compared to non-obese donors if the quality of kidneys were good (KDPI ≤ 30%). A significantly decreased graft survival among obese donors was observed if KDPI > 30% where other donor risk factors and additional comorbidities may survivable at play. Therefore, donor BMI should be considered while determining if to transplant these donor kidneys. Donor obesity was also associated with an increased risk for DGF but not PNF.

## Authorship

PH: participated in the research design, performed the data analysis and interpretation and the writing of paper (drafting and critical revision of article). MS: participated in data analysis and interpretation. ND: participated in the writing of paper (drafting of article). GMD: participated in the writing of paper (drafting of article). SB: participated in the research design, performed the data analysis and interpretation, the performance of the research, critical revision of the article and contributed to the writing of paper.

## Funding

The authors have declared no funding.

## Conflicts of interest

The authors have declared no conflicts of interest.

## Acknowledgements

This work was supported in part by Health Resources and Services Administration contract 234-2005-37011C. The content is the responsibility of the authors alone and does not necessarily reflect the views or policies of the Department of Health and Human Services, nor does mention of trade names, commercial products, or organizations imply endorsement by the U.S. Government.

## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

**Table S1.** Baseline donor, recipient, and transplant characteristics by donor BMI.

## REFERENCES


1. Hales CM, Carroll MD, Fryar CD, Ogden CL. *Prevalence of Obesity and Severe Obesity Among Adults: United States, 2017–2018*. NCHS Data Brief, no 360. Hyattsville, MD: National Center for Health Statistics, 2020. Available at https://www.cdc.gov/nchs/products/databriefs/db360.htm. Accessed May 10, 2020.
2. Kambham N, Markowitz GS, Valeri AM, *et al*. Obesity-related glomerulopathy: an emerging epidemic. *Kidney Int* 2001; **59**: 1498.
3. D'Agati VD, Chagnac A, de Vries AP, *et al*. Obesity-related glomerulopathy: clinical and pathologic characteristics and pathogenesis. *Nat Rev Nephrol* 2016; **12**: 453.
4. Ortiz J, Gregg A, Wen X, *et al*. Impact of donor obesity and donation after cardiac death on outcomes after kidney transplantation. *Clin Transplant* 2012; **26**: E284.
5. Heimbach JK, Taler SJ, Prieto M, *et al*. Obesity in living kidney donors: clinical characteristics and outcomes in the era of laparoscopic donor nephrectomy. *Am J Transplant* 2005; **5**: 1057.
6. Lafranca JA, Hagen SM, Dols LF, *et al*. Systematic review and meta-analysis of the relation between body mass index and short-term donor outcome of laparoscopic donor nephrectomy. *Kidney Int* 2013; **83**: 931.
7. Siedlecki A, Irish W, Brennan DC. Delayed graft function in the kidney transplant. *Am J Transplant* 2011; **11**: 2279.
8. Kayler LK, Srinivas TR, Schold JD. Influence of CIT-induced DGF on kidney transplant outcomes. *Am J Transplant* 2011; **11**: 2657.
9. Tennankore KK, Kim SJ, Alwayn IP, *et al*. Prolonged warm ischemia time is associated with graft failure and mortality after kidney transplantation. *Kidney Int* 2016; **89**: 648.
10. Brennan C, Sandoval PR, Husain SA, *et al*. Impact of cold and warm ischemia time on outcomes for kidneys donated after cardiac death [abstract]. *Am J Transplant* 2019; **19**(suppl 3): 441.
11. Osband AJ, James NT, Segev DL. Extraction time of kidneys from deceased donors and impact on outcomes. *Am J Transplant* 2016; **16**: 700.
12. Reese PP, Feldman HI, Asch DA, *et al*. Short-term outcomes for obese live kidney donors and their recipients. *Transplantation* 2009; **88**: 662.
13. Naik AS, Zhong Y, Parasuraman R, *et al*. The temporal and long-term impact of donor body mass index on recipient outcomes after kidney transplantation – a retrospective study. *Transpl Int* 2020; **33**: 59.
14. Alhamad T, Malone AF, Lentine KL, *et al*. Selected mildly obese donors can be used safely in simultaneous pancreas and kidney transplantation. *Transplantation* 2017; **101**: 1159.
15. Dahmen M, Becker F, Pavenstädt H, Suwelack B, Schütte-Nütgen K, Reuter S. Validation of the Kidney Donor Profile Index (KDPI) to assess a deceased donor's kidneys' outcome in a European cohort. *Sci Rep* 2019; **9**: 11234.
16. Lehner LJ, Kleinsteuber A, Halleck F, *et al*. Assessment of the Kidney Donor Profile Index in a European cohort. *Nephrol Dial Transplant* 2018; **33**: 1465.
17. Gupta A, Francos G, Frank AM, Shah AP. KDPI score is a strong predictor of future graft function: moderate KDPI (35–85) and high KDPI (> 85) grafts yield similar graft function and survival. *Clin Nephrol* 2016; **86**: 175.
18. Jay C, Pugh J, Halff G, Abrahamian G, Cigarroa F, Washburn K. Graft quality matters: Survival after simultaneous liver-kidney transplant according to KDPI. *Clin Transplant* 2017; **31**: e12933.
19. Zens TJ, Danobeitia JS, Leverson G, *et al*. The impact of kidney donor profile index on delayed graft function and transplant outcomes: a single-center analysis. *Clin Transplant* 2018; **32**: e13190.
20. OPTN. A Guide to Calculating and Interpreting the Kidney Donor Profile Index (KDPI). https://optn.transplant.hrsa.gov/media/1512/guide_to_calculating_interpreting_kdpi.pdf. Updated March 23, 2020. Accessed May 15, 2020.
21. Choung HG, Bomback AS, Stokes MB, *et al*. The spectrum of kidney biopsy findings in patients with morbid obesity. *Kidney Int* 2019; **95**: 647.
22. Cao X, Zhou J, Yuan H, *et al*. Chronic kidney disease among overweight and obesity with and without metabolic syndrome in an urban Chinese cohort. *BMC Nephrol* 2015; **16**: 85.
23. Kovesdy CP, Furth SL, Zoccali C, *et al*. Obesity and kidney disease: hidden consequences of the epidemic. *Am J Nephrol* 2017; **45**: 283.
24. Savastano DM, Gorbach AM, Eden HS, *et al*. Adiposity and human regional body temperature. *Am J Clin Nutr* 2009; **90**: 1124.
25. Zens T, Leverson G, Redfield R, *et al*. Is there a difference between DCD and DBD kidney transplantation with similar KDPI?. *Am J Transplant* 2016; **16** (suppl 3): 563.
26. Woo YM, Jardine AG, Clark AF, *et al*. Early graft function and patient survival following cadaveric renal transplantation. *Kidney Int* 1999; **55**: 692.