HHS Public Access

Author manuscript

Surg Obes Relat Dis. Author manuscript; available in PMC 2022 March 01.

Published in final edited form as:

Surg Obes Relat Dis. 2021 March ; 17(3): 508–515. doi:10.1016/j.soard.2020.11.010.

The Relationship Between Kidney Function and Body Mass Index Before and After Bariatric Surgery in Patients with Chronic Kidney Disease

Heidi Fischer, PhD[*],
Department of Research and Evaluation, Kaiser Permanente Southern California, Pasadena, CA

Robert E. Weiss, PhD,
Department of Biostatistics, UCLA Fielding School of Public Health, Los Angeles, CA

Allon N. Friedman, MD,
Department of Medicine, Indiana University School of Medicine, Indianapolis, IN

Talha H. Imam, MD,
Department of Nephrology, Fontana Medical Center, Kaiser Permanente Southern California, Fontana

Karen J. Coleman, PhD
Department of Research and Evaluation, Kaiser Permanente Southern California, Pasadena, CA

Abstract

Background & Objectives—Improvements in kidney function post-bariatric surgery may be related to weight loss-independent effects. We characterize the dynamic relationship between body mass index (BMI) and estimated glomerular filtration rate (eGFR) before and after bariatric surgery in patients with chronic kidney disease (CKD).

Design, Setting, Participants and Measurements—Observational retrospective cohort study of patients with CKD stage 3 or higher who received bariatric surgery at the Kaiser Permanente Southern California (KPSC) health system between 2007-2015. Bariatric surgery procedures included primary Roux-en-Y gastric bypass (RYGB) or sleeve gastrectomy (SG) procedures. Outcomes consisted of mean trajectory estimates and correlations of BMI and eGFR taken between two years before and three years after surgery. Multivariate functional mixed models were used to estimate how BMI and eGFR trajectories evolved jointly.

Results: A total of 619 RYGB and 474 SG patients were included in the final analytic sample. The median time measurements were available before surgery was 1.9 years for SG and 1.8 years

[*]Corresponding author 100 S. Los Robles, 6th Floor, Pasadena, CA 91101, (626) 564-3290 (office), (626) 564-7872 (fax), Heidi.Fischer@kp.org.

Disclosures
The authors have no conflicts of interest to disclose.

for RYGB. Median follow-up times after surgery were 2.8 years for both SG and RYGB. Mean age at time of surgery was 58 years, 77% were women, 56% were non-Hispanic whites, mean BMI was 44 kg/m^2, 60% had diabetes mellitus, and 84% had hypertension. Compared to pre-surgery eGFR decline, post-surgery decline in eGFR was 57% slower (95% CI, 33%-81%) for RYGB patients and 55% slower (95% CI, 25%-75%) for SG patients. Mean correlation between BMI and eGFR was negligible at all time points.

Conclusion: Though bariatric surgery slowed decline in eGFR up to three years after surgery, changes in eGFR tracked poorly with changes in BMI. This study provides evidence that the kidney-related benefits of bariatric surgery may at least be partly independent of weight loss. Confirming this hypothesis could lead to mechanistic insights and new treatment options for CKD.

Keywords

Chronic kidney disease; bariatric surgery; obesity; glomerular filtration rate

1. INTRODUCTION

The presence of chronic kidney disease (CKD) confers a greatly increased risk of morbidity and death as well as a disproportionately high cost burden for society.[1,2] Obesity is arguably the most important modifiable cause of CKD since it affects nearly half of patients with CKD in the US and is the root cause of many of the most consequential CKD risk factors including type 2 diabetes, hypertension, and cardiopulmonary disease as well as having direct adverse effects.[3,4]

Currently the most effective treatment for severe obesity is bariatric surgery. Bariatric surgery results in significantly greater and more durable long-term weight loss compared with non-surgical weight loss methods.[5,6] Bariatric surgery may also have kidney protective effects. Numerous observational studies find that bariatric surgery improves major risk factors for CKD[7-9] and parameters of kidney disease.[4,10-16] However, only one such study was performed in patients with pre-existing CKD.[17]

Although the putative benefits of bariatric surgery on kidney function are commonly assumed to be directly related to weight loss, this assumption is debatable. A growing literature finds that bariatric surgery improves the diabetic milieu even before significant weight is lost thus demonstrating that its effects are at least partially weight-loss independent (and explaining its alternative moniker “metabolic surgery”).[18,19] It is possible that similar weight-independent effects may also be contributing to improvements in kidney function. In fact, preliminary research supports this hypothesis, though these studies were not performed in patients with CKD nor included analyses that directly estimated the dynamic association between weight loss and kidney function over time.[20-22] Confirming such a phenomenon could lead to new mechanistic insights and treatment options for CKD.

To address these limitations, we conducted an observational retrospective cohort study of patients with CKD stage 3 or higher who had undergone bariatric surgery to evaluate weight and estimated glomerular filtration rate (eGFR) trajectories both before and after surgery and

characterize the dynamic relationship. We hypothesized that changes in eGFR and weight would closely correlate both before and after surgery.

2. MATERIALS AND METHODS

2.1. Setting

Data were obtained from the Kaiser Permanente Southern California (KPSC) system, which delivers integrated care to more than 4.5 million members through outpatient clinics and hospitals. Details of the bariatric surgery program at KPSC have been published elsewhere.[23] Briefly, nearly 3,000 primary weight loss procedures are performed annually by 23 surgeons at nine hospitals. Data on bariatric surgery patients at KPSC are maintained in a registry that contains electronic information from several sources (described in the Measures section). Bariatric surgery patients at KPSC are generally representative of the national bariatric surgery population with the exception that they have a much higher proportion of ethnic/racial minorities.[24,25] All analyses were approved by the Institutional Review Board for Human Subjects. A waiver of signed consent was approved due to minimal study risk.

2.2. Participants

The selection process for patients in the current study is shown in Figure 1. Bariatric surgery patients were eligible for the study if they had the following characteristics: (1) a primary Roux-en-Y gastric bypass (RYGB) or sleeve gastrectomy (SG) procedure from January 1, 2007, through December 31, 2015; (2) a body mass index (BMI) ≥ 30 kg/m^2 at time of surgery; (3) an eGFR < 60 mL/min/1.73 m^2 (CKD stages 3 or higher) at time of surgery; and (4) no record of kidney transplant or dialysis in the 5 years before surgery. A total of 619 RYGB and 474 SG patients were included in the final analytic sample.

2.3. Measurements

Weight (lbs) and eGFR (mL/min/1.73 m^2) measurements were obtained from electronic medical records and outside claims sources for 2 years before the index bariatric operation through 3 years after the operation. These time periods were selected to allow enough time to compare trends before and after surgery and to insure that the SG patients had enough time to accumulate meaningful follow-up time since it was not performed regularly at KPSC until 2011. Data collected from patients was entered into the electronic medical record by clinical staff as part of routine care. Date of birth, sex, and race/ethnicity were self-reported. Weight was measured by clinical staff and height was typically self-reported. Diagnoses, procedures, and laboratory results were available from all healthcare settings including outpatient, inpatient, and emergency departments.

2.4. Analyses

2.4.1. Outcome Definitions—Serum creatinine was used to calculate eGFR using the CKD Epidemiology Collaboration (CKD- EPI) creatinine equation.[26] KPSC labs adjusted serum creatinine levels by whether or not the original measurement assay was IDMS-traceable. Height and weight were used to calculate BMI (kg/m^2).

2.4.2. Statistical Models—Bayesian multivariate functional mixed models were used to estimate how BMI and eGFR trajectories evolved jointly before and after bariatric surgery for both RYGB and SG. These models incorporate random effects to model trajectories and estimate the correlation between trajectories of different outcomes.[27,28]

BMI and eGFR trajectories are highly non-linear over time both before and after surgery. To accurately capture these non-linear trajectories, we model both the population mean and random trajectories using splines, or piece-wise polynomials, which can model highly non-linear time trends. We utilized a specific type of spline, the cubic B-spline, for the fixed and random effects due to its stable numeric properties.[29] Knots were chosen to smoothly model trends in individual trajectories without picking up random variation.

Simultaneous and cross-time correlations of the trajectories for eGFR and BMI were estimated to evaluate the association of the two outcomes throughout the period of 2 years before to 3 years after surgery. Absolute correlation estimates less than .2 are considered to be negligible while those over .3 are considered moderate.[30] Credible intervals (CI), the Bayesian analog of confidence intervals, are provided for all estimates of interest. The complete model is detailed in Section 1 of the Appendix A.

Posterior means and standard deviations were obtained from Markov Chain Monte Carlo (MCMC) simulations[31,32] using R package MCMCglmm.[33] To supplement understanding of BMI trajectory changes before and after surgery, trajectory changes are also presented in terms of percent excess BMI lost, with excess BMI > 25 kg/m^2, and percent total body weight lost. The RYGB and SG patient populations were analyzed separately. Results are displayed graphically whenever possible to aid in interpretation. All statistical analyses were conducted using R, version 3.2.3 (R Foundation for Statistical Computing).

3. RESULTS

3.1. Participants

Descriptive characteristics are presented in Table 1 for patients undergoing both types of bariatric surgery. In general, mean age at surgery was 58 years, 77% were women, 56% were non-Hispanic whites, mean BMI was 44 kg/m^2, 60% had diabetes mellitus, and 84% had hypertension. The median time that measurements were available before bariatric surgery was 1.9 years for SG and 1.8 years for RYGB. Of SG and RYGB patients, 98% and 90% recorded BMI measurements more than one year before surgery while 91% and 80%, respectively, recorded at least one serum creatinine measurement more than one year before bariatric surgery. Median follow-up times after bariatric surgery were 2.8 years for both SG and RYGB (86% had at least one BMI measurement in the third year following surgery for both procedures, while 81% and 79%, respectively, had at least one serum creatinine measurement in the third year following surgery).

3.2 Mean Trajectories

Mean BMI and eGFR trajectories for RYGB and SG procedures are shown in Figures 2A - 2D. Pre-surgical mean BMI trajectories for both procedures increased over time from 2 years to 1 year before surgery at an average of 1.12 kg/m^2 per year for RYGB (95%

CI, .76-1.71 kg/m^2) and .92 kg/m^2 per year for SG (95% CI, .39-1.48 kg/m^2). Beginning 1 year before surgery, there was a small downward trend in BMI until the date of surgery. Following surgery, there was a steep decrease in BMI before reaching the nadir at 1.28 (95% CI, 1.23-1.35) and 1.10 (95% CI, 1.04-1.17) years following surgery for RYGB and SG, respectively. Weight gain then resumed at .71 (95% CI, .52-.92) kg/m^2 per year for RYGB and .79 (95%CI, .59-.98) kg/m^2 per year for SG until the end of the 3-year follow-up period. The percent difference between pre- and post-surgery annual weight gain was similar with ninety-five percent credible intervals overlapping zero for both surgery types. To supplement understanding of BMI trajectory changes before and after surgery, we present trajectories for percent excess BMI lost and percent total body weight lost in Figure A.1 of Appendix A.

Mean eGFR trajectories declined steadily in the 2 years before surgery for both procedures at an average of 4.83 mL/min/1.73 m^2 (95%CI, 3.73-5.91) per year for RYGB and 4.12 mL/min/1.73 m^2 (95% CI, 2.98-5.25) per year for SG. After an initial sharp increase in eGFR in the 2 months following each operation, a gradual increase in eGFR continued until .96 (95%CI, .76, 1.28) years and .81 (95%CI, .24-1.09) years following surgery for RYGB and SG, respectively. Decline in eGFR then resumed at an average rate of 2.08 mL/min/1.73 m^2 (95%CI, .96-3.01 mL/min/1.73 m^2) per year for RYGB and 1.84 mL/min/1.73 m^2 (95%CI, 1.06-2.78 mL/min/1.73 m^2) per year for SG. Compared to the 2-years prior to surgery, this post-surgery decline in eGFR for was 57% slower (95% CI, 33%-81%) for RYGB patients and 55% slower (95% CI, 25%-75%) for SG patients.

3.3 Correlation of Trajectories

Marginal correlations over time between simultaneously measured eGFR and BMI outcomes (i.e. the correlation of BMI measured at time t with eGFR measured at time t) for each operation can be found in Figures 3A-3B. Correlation remained negative but negligible both before and after surgery. Both surgery types showed a small downward trend immediately following surgery, with correlations reaching a minimum of −.13 (95% CI, −.20,−.053) for RYGB patients (Fig 3A) and −0.08 (95% CI, −0.19, .03) for SG patients (Fig 3B), but the magnitude of this trend was not meaningful. Trends otherwise appeared similar both before and after surgery. Correlations across time points, which would measure a delayed association of weight loss and eGFR (eg: correlation of BMI at 6 months with eGFR at 1 year, BMI at 1 year with eGFR at 6 months), showed similar weak relationships in both directions and are presented in Tables A.1 and A.2 of Appendix A.

4. DISCUSSION

Our study carefully analyzed the relationship between fluctuations in weight and eGFR in patients with CKD stages 3 or higher who underwent RYGB or SG, the two predominant types of bariatric surgery in the US.[34] While previous observational studies have demonstrated an association between bariatric surgery and a slowing in the decline of kidney function or kidney risk indices post-surgery[4,12-17], this study provides additional insight by comparing patient post-surgical weight and eGFR trajectories with their own pre-surgical trajectories. Trajectory information immediately following bariatric surgery, when changes in eGFR may also reflect reduced muscle mass, were not included in these comparisons.

[17,35] One major finding was that the decline in eGFR was over 50% slower up to 3 years post-surgery as compared to the 2 years prior to surgery regardless of the type of bariatric surgery performed.

That bariatric surgery slows progression of CKD is biologically plausible. Bariatric surgery ameliorates major intermediate risk factors for CKD such as type 2 diabetes and systemic hypertension (the first and second most common causes, respectively, of kidney failure in the US), pulmonary hypertension, sleep apnea, and heart dysfunction.[7,8,36] Direct adverse effects of obesity on the kidney like glomerular hyperfiltration, intraglomerular hypertension leading to shear and podocyte stress, obesity related glomerulopathy, and fat infiltration may also be ameliorated by bariatric surgery but confirmatory data in humans are less available.[20,37,38] Our study indicates that bariatric surgery may reset the decline in eGFR to a flatter slope in the patients with CKD. Studies with longer follow-up are needed to confirm our findings and determine if the benefits last longer than three years and slow progression to kidney failure.

The relationship between changes in eGFR and BMI is also revealing. In the year prior to bariatric surgery the mean BMI decreased slightly, most likely due to a requirement that patients lose some weight to be eligible for surgery.[39] The profound drop in weight that occurred immediately after bariatric surgery is typical and confirms that this intervention is effective in patients with CKD. Modest weight regain such as what occurred after the weight nadir is also common.[40] Within the first two months following surgery, we also observed an immediate and large rise in eGFR that could reflect a real improvement in glomerular filtration, a spurious effect from reduced muscle mass leading to a lower serum creatinine and higher eGFR estimation, or a combination of the two.[35]

Significant mean improvements in both weight and eGFR occurred during the first year after surgery. However, while correlations were in the expected direction (i.e. decreases in BMI were associated with increases in eGFR), correlations between these two measures both before and after either type of bariatric surgery procedure were negligible. While in general patients with CKD receiving bariatric surgery experienced weight loss and improved eGFR, the measures' negligible correlation indicates that those patients that lost more weight than average were not more likely to see improvements in eGFR and vice versa. Negligible correlations were observed during the entire 3-year post-surgical period so cannot be fully explained by possible eGFR measurement error during the initial period of weight loss following surgery, when changes in serum creatinine may also measure changes in body composition, including muscle mass.[17,35]

While our findings require confirmation, this is not the first time that weight has been observed to track poorly with parameters of kidney function or damage. Friedman et al reported that GFR directly measured before and after bariatric surgery in 36 individuals with near-normal kidney function was not statistically associated with change in weight.[20] In a systematic review and meta-analysis of persons with type 2 diabetes without CKD Martin et al found that reductions in urinary albumin excretion after bariatric surgery were independent of BMI.[22] Similarly, in a murine model comparing RYBG to dietary restriction, Neff and colleagues found that RYBG provided an anti-proteinuric effect that

was independent of weight loss.[35] Our findings also echo previous studies in animals and humans that find bariatric surgery offers metabolic benefits that are independent of weight loss, perhaps through the activation of gut hormones, bile acid signaling, changes in the gut microbiome, or reprogramming of gut tissue. However the controversy over putative weight-independent effects has still not been fully settled.[41] Clearly further research is needed in this area that is specific to kidney disease.

Our findings are directly relevant to clinical care as they reinforce the idea of bariatric surgery as a renoprotective intervention and offer novel quantitative data in the form of changes in eGFR slope to support this contention. Notably, the improvement in the slope of decline of eGFR after bariatric surgery compares favorably to mainstay therapies for CKD such as renin-angiotensin-aldosterone axis blockers.[42,43] This in turn further supports integrating anti-obesity strategies such as bariatric surgery into routine nephrology practice. In addition, the results provide further evidence supporting earlier suggestions in the medical literature that some of the kidney-related benefits of renoprotective surgery may be explained by weight-independent effects. This will require further exploration and confirmation in human physiology studies and clinical trials

Our study has several limitations. Our data were obtained from clinical electronic medical records so neither directly measured GFR or the surrogate serum filtration marker cystatin C, which is more closely associated than serum creatinine with measured GFR in persons with obesity, were available for analysis.[20] The effects we found may not reflect what would have occurred if GFR was directly measured. Our study follow-up time was also limited to 3-years following surgery, so we were unable to determine if the benefits observed last longer than three years and slow progression to kidney failure. In addition, this study was not meant be a formal comparative effectiveness study between SG and RYGB so formal conclusions about differences between SG and RYGB cannot be made. Comparative effectiveness methods with multiple comorbid conditions in the same statistical model would further complicate an already complex model and are beyond the scope of this investigation. This study was intended to estimate and compare means and correlations for BMI and eGFR trajectories before and after surgery within RYGB and SG operations to better understand the pattern of change and relationship between weight and eGFR following surgery. Major strengths included the use of sophisticated multivariate mixed models to, for the first time, analyze the joint evolution of eGFR and BMI before and after two common bariatric surgery procedures in a population with CKD.

5. CONCLUSION

In conclusion our results support the contentions that bariatric surgery offers kidney protection in patients with established kidney disease and that at least part of this protection is afforded by weight-loss independent mechanisms.. Further study is necessary to definitively confirm these findings.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding for this study was provided by the Biostatistics Methodology Research Proposals of Department of Research & Evaluation, Kaiser Permanente Southern California and the National Institute for Diabetes and Digestive and Kidney Diseases, R01 DK105960. Funders had no role in study design; collection, analysis, and interpretation of data; writing the report; and the decision to submit the report for publication

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Highlights

- Bariatric surgery improves kidney function for patients with chronic kidney disease.
- Kidney-related benefits of bariatric surgery may at least be partly independent of weight loss.
- Confirming this hypothesis could lead to mechanistic insights and new treatment options.

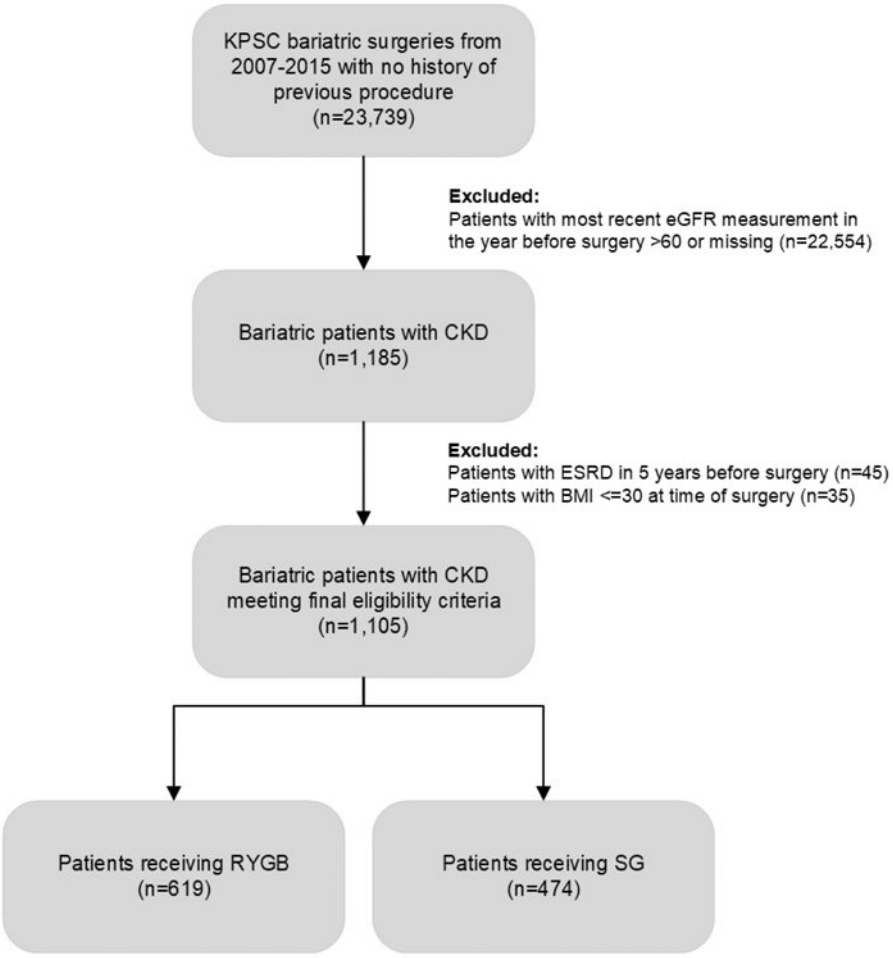

Figure 1:
Selection process for study participants.

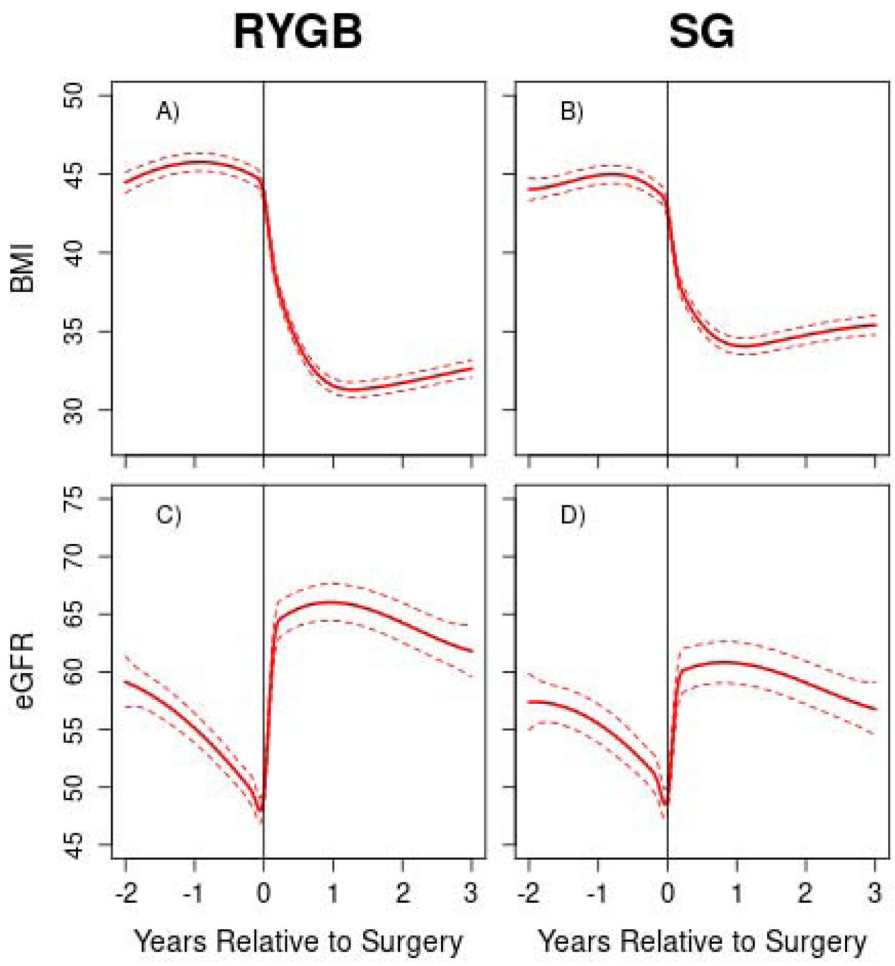

Figure 2:

Mean trajectories from two years before surgery to three years after surgery for A) Body mass indices (BMI) in Roux-en-Y gastric bypass (RYGB) cohort, B) BMI in Sleeve-Gastrectomy (SG) cohort, C) estimated glomerular filtration rate (eGFR) in RYGB cohort, and D)eGFR in SG cohort. Dotted lines indicate 95% credible intervals, solid vertical line indicates time of surgery.

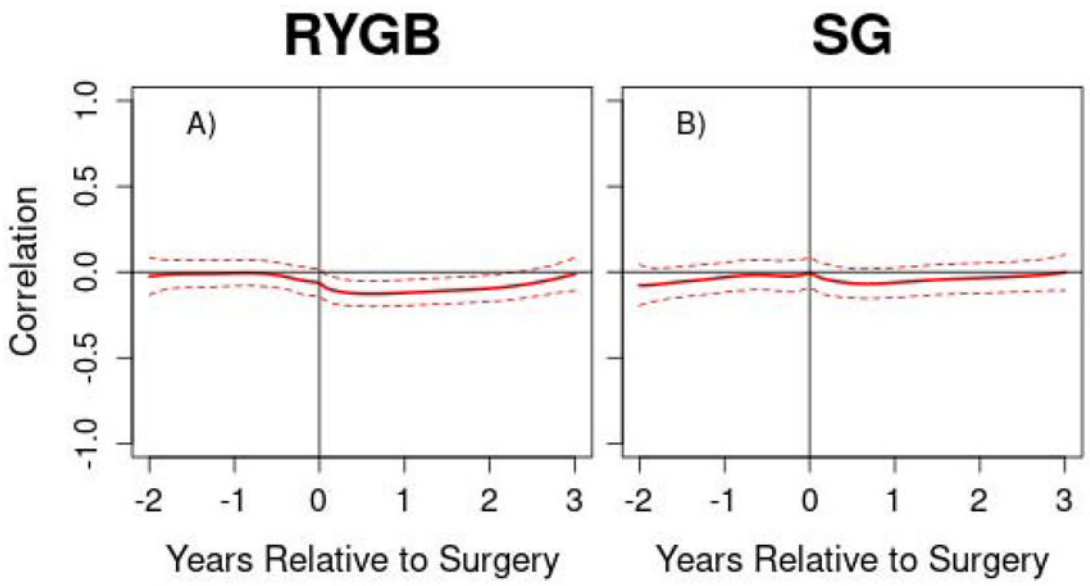

Figure 3:
Correlation trajectories over time between eGFR at time *t* and BMI at time *t* for A) RYGB cohort, and B) SG cohort. Dotted lines indicate 95% credible intervals, solid vertical line indicates time of surgery.

Table 1:

Patient Characteristics at Surgery[a]

	SG (n = 474)	RYGB (n = 619)	Overall (n=1093)
Age (years)	58.7 (8.89)	57.3 (8.33)	57.9 (8.60)
Weight (kg)	120.0 (21.08)	124.6 (24.41)	122.6 (23.13)
BMI (kg/m^2)	43.4 (6.09)	44.5 (7.05)	44.0 (6.67)
% Women	380 (80.2%)	458 (74%)	838 (76.7%)
Race/Ethnicity			
Non-Hispanic White	265 (55.9%)	350 (56.5%)	615 (56.3%)
Non-Hispanic Black	97 (20.5%)	100 (16.2%)	197 (18%)
Hispanic	95 (20%)	154 (24.9%)	249 (22.8%)
Other/Missing/Unknown	17 (3.6%)	15 (2.4%)	32 (2.9%)
% with hypertension	385 (81.2%)	528 (85.3%)	913 (83.5%)
% with diabetes	220 (46.4%)	430 (69.5%)	650 (59.5%)
Serum creatinine (mg/dL)	1.5 (1.01)	1.4 (0.63)	1.5 (0.82)
eGFR (ml/min/1.73 m^2)	48.0 (12.22)	47.9 (10.20)	47.9 (11.12)
CKD stage 3 (%)	428 (90.3%)	575 (92.9%)	1003 (91.8%)
CKD stage 4 (%)	29 (6.1%)	38 (6.1%)	67 (6.1%)
CKD stage 5 (%)	17 (3.6%)	6 (1%)	23 (2.1%)

[a]Values for categorical variables are given as count (proportion); for continuous variables, as mean ± standard deviation. Abbreviations: Roux-en-Y gastric bypass (RYGB), sleeve gastrectomy (SG), body mass index (BMI), chronic kidney disease (CKD).