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Matched weight loss induced by sleeve gastrectomy or gastric bypass similarly improves metabolic function in obese subjects

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Abstract

Objective—We evaluated the effects of marked weight loss, induced by Roux-en-Y gastric bypass (RYGB) or sleeve gastrectomy (SG) surgeries, on insulin sensitivity, β-cell function and the metabolic response to a mixed meal.

Design and Methods—Fourteen non-diabetic insulin-resistant patients who were scheduled to undergo SG (n=7) or RYGB (n=7) procedures completed a hyperinsulinemic-euglycemic clamp procedure and a mixed-meal tolerance test before surgery and after losing ∼20% of their initial body weight.

Results—Insulin sensitivity (insulin-stimulated glucose disposal during a clamp procedure), oral glucose tolerance (postprandial plasma glucose area under the curve), and β-cell function (insulin secretion in relationship to insulin sensitivity) improved after weight loss, and were not different between surgical groups. The metabolic response to meal ingestion was similar after RYGB or SG, manifested by rapid delivery of ingested glucose into the systemic circulation and a large early postprandial increase in plasma glucose, insulin and C-peptide concentrations in both groups.

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Conflicts of Interest: SK is a shareholder of Aspire Bariatrics, Metro Midwest Biotech and Human Longevity Inc, serves as a consultant for Aspire Bariatrics, served on the scientific advisory boards of Takeda Pharmaceuticals, Vivus, Danone/Yakult, Bristol-Myers Squibb, NovoNordisk, Dairy Research Institute, and the Egg Nutrition Council, and is a member of the Merck Speakers Bureau.

Author contributions: DB performed the experiments, collated and analyzed data and wrote the manuscript; FM assisted with data analysis and reviewed/edited the manuscript; JCE performed the surgeries and reviewed/edited the manuscript; AG assisted with data analysis and reviewed/edited the manuscript; ALO processed samples and reviewed/edited the manuscript; BWP analyzed data and reviewed/edited the manuscript; SK designed the study, obtained funding, reviewed the data, contributed to the discussion and reviewed/edited the manuscript.

Conclusions—We conclude that, when matched on weight loss, RYGB and SG surgeries result in similar improvements in the two major factors involved in regulating plasma glucose homeostasis, insulin sensitivity and β-cell function in obese people without diabetes.

Keywords

Bariatric surgery; sleeve gastrectomy; insulin resistance; gastric bypass

Introduction

Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy (SG) are the most commonly performed bariatric surgery procedures in the United States and worldwide (1). Both procedures cause considerable weight loss and a marked improvement in glycemic control in patients who have type 2 diabetes (T2D). However, the results from two recent randomized controlled trials found RYGB surgery was more effective than SG in treating T2D, manifested by a higher rate of diabetes remission (defined as glycated hemoglobin <6.5% or ≤6% without diabetes medications) at 1 year after surgery (2, 3). The mechanism(s) responsible for this observed difference in therapeutic efficacy between procedures is unclear, because weight loss was greater after RYGB than SG, which could have been responsible for the differences in diabetes remission between groups. In addition, it is not clear whether weight loss induced by RYGB and SG has the same or different effects on metabolic function, because of conflicting results from different studies, which found the improvement in insulin sensitivity after surgery-induced weight loss was greater after RYGB than SG (4), greater after SG than RYGB (5), or the same after both procedures (6).

The primary aim of this study was to test the hypothesis that RYGB surgery causes a greater improvement in skeletal muscle insulin sensitivity than does SG after the same amount of marked weight loss. The secondary aim was to evaluate the effect of RYGB and SG on β-cell function and the metabolic response to the ingestion of a mixed meal. Obese, insulin-resistant subjects were studied before RYGB or SG surgery and after they lost the same large amount (∼20%) of their body weight to avoid the confounding effect of differences in weight loss between surgical procedures on our metabolic outcome measures.

Methods

Study subjects

Fourteen consecutive eligible patients who were scheduled to undergo SG (n=7; 1 man, 6 women, 37±10 yrs old) or RYGB (n=7; 2 men, 5 women; 40±7 yrs old) procedures at Barnes-Jewish Hospital in St. Louis, MO participated in this study, after they provided written informed consent. Some of the subjects in the RYGB group also participated in a study that was published previously (7). All subjects were required to have evidence of insulin resistance, based on the homeostasis model assessment of insulin resistance (HOMA-IR) score >3.0 (8), but those who had diabetes were excluded to avoid the confounding effects of differences in baseline glycemic control, glucose toxicity, and postsurgical changes in diabetes medications on our outcome measures. The study was approved by the Washington University Institutional Review Board.

Study Design and Experimental Procedures

Body fat mass and fat-free mass (FFM) (determined by dual-energy X-ray absorptiometry), insulin sensitivity (determined by a hyperinsulinemic-euglycemic clamp [HEC] procedure), and the metabolic response to a mixed meal were assessed before bariatric surgery and after 20% surgery-induced weight loss.

Hyperinsulinemic-euglycemic Clamp Procedure—Subjects were admitted to the Clinical Research Unit (CRU) and consumed a standard evening meal (12 kcal/kg FFM; 50% of calories as carbohydrate, 30% as fat and 20% as protein). The following morning, a catheter was inserted into a forearm vein for infusion and a second catheter was inserted into a radial artery to obtain blood samples. At 0600 h, a primed-continuous infusion of [6,6-$^2H_2$]glucose (priming dose: 22 μmol/kg; infusion rate: 0.22 μmol/kg/min) was started and maintained until the end of the study. At 0930 h, insulin was infused at a rate of 50 mU·m^2·min^{-1} for 4 hours. Euglycemia (plasma glucose ∼100 mg/dl) was maintained by infusing 20% dextrose enriched to 2.5% with [6,6-$^2H_2$]glucose. Blood samples were obtained immediately before starting the tracer infusion and during the final 30 minutes of the basal period and the clamp procedure to determine plasma substrate and insulin concentrations and glucose tracer-to-tracee ratio (TTR).

Mixed-Meal Metabolic Study—Subjects were admitted to the CRU and consumed a standard evening meal. The following morning, a catheter was inserted into a forearm vein for infusion and a second catheter was inserted into a radial artery to obtain blood samples. A primed, continuous infusion of [6,6-$^2H_2$]glucose (priming dose: 22 μmol/kg; infusion rate: 0.22 μmol/kg/min) was started and maintained until the end of the study. After 3.5 h of tracer infusion, subjects ingested a liquid meal (containing 46 g of glucose mixed with 0.9 g of [U-^{13}C]glucose, 9 g of fat, and 9 g of protein) which was provided in 7 equally divided aliquots given every 5 minutes over 30 minutes. Blood samples were obtained immediately before starting the glucose tracer infusion, every 10 minutes for 30 minutes just before starting the meal ingestion, and then every 15 minutes for the first hour and every 20 minutes for the subsequent 5 hours after starting the meal, to determine plasma substrate and hormone concentrations, and glucose TTRs.

Surgical Procedures—Bariatric surgeries were performed by using standard laparoscopic approaches. The RYGB procedure involved creating a small (∼20 ml) proximal gastric pouch, a 30 cm biliopancreatic limb and a 100 cm Roux limb. The SG procedure involved dividing the gastrocolic ligament, initiating the gastrectomy 6 cm proximal to the pylorus along the greater curve, and creating the sleeve along the lesser curve over a 40 French Bougie.

Weight Management after Surgery—A supervised dietary weight loss program was instituted to help subjects in both groups consume a similar energy-deficit diet and achieve a 20% weight loss within 4-6 months after surgery. All subjects were instructed to consume a no-added-sugar liquid diet (400-600 kcal/day) for the first week after surgery, a pureed diet (2-3 oz/meal providing 700-800 kcal/day) for weeks 2-3, a soft diet (3-4 oz/meal providing 800-1000 cal/day) for weeks 4-5, followed by a regular-food diet containing 1000-1200 kcal/day and 1.0 g of protein/kg body weight per day. After subjects achieved a 20% weight loss, a balanced weight maintenance diet was prescribed, and subjects maintained a stable body weight (<2% change) for at least 2 weeks before repeat studies were performed.

Analyses of Blood Samples—Plasma insulin and C-peptide concentrations were measured by using enzyme-linked immunosorbent assays (EMD Millipore Corporation, St. Charles, MO). Plasma glucose TTRs were determined by using gas chromatography-mass spectrometry (9), and plasma glucose concentrations were measured by the glucose oxidase method on an automated analyzer (Yellow Springs Instrument Co., Yellow Springs, OH).

Calculations

Insulin sensitivity: Glucose total rate of appearance (Ra) into the systemic circulation was calculated by dividing the tracer infusion rate by the average plasma glucose TTR during the last 30 min of the basal and insulin infusion periods (10). Endogenous glucose production (EGP) was determined by subtracting exogenous glucose infusion rate (from 20% dextrose) from total Ra. Glucose rate of disappearance (Rd) from plasma was equal to endogenous glucose Ra plus the rate of exogenously infused dextrose and glucose tracer. Insulin-stimulated glucose disposal, primarily in skeletal muscle, was used as an index of insulin sensitivity.

Metabolic response to the mixed-meal: Plasma glucose and insulin concentration areas-under-the-curve (AUCs) for 6 h after initiating meal consumption were calculated by using the trapezoid rule (11). Total glucose Ra into the systemic circulation during the meal was calculated by using Steele's equation for non-steady-state conditions (10). Glucose Ra into the systemic circulation from ingested glucose and from EGP was calculated as previously described (12). Total postprandial insulin secretion rate (ISR) was calculated by using stochastic deconvolution (13). The principle of the disposition index (insulin secretion in relationship to insulin sensitivity) (14) was used to evaluate β-cell function, calculated as the product of the rate of insulin-stimulated glucose disposal normalized for plasma insulin concentration (μmol/kgFFM/min per μIU/mL, assessed during the HEC procedure) and total ISR AUC (pmol/L, assessed after meal ingestion).

Statistical Analysis

Data were examined for normality according to the Shapiro-Wilks criteria. A two-way repeated measures analysis of variance with Tukey's post-hoc test was used to compare the effects of SG and RYGB surgeries on study outcome measures (except for postprandial metabolic outcomes). A mixed model repeated measures analysis of variance was used to evaluate differences between SG and RYGB groups in weight loss-induced changes in postprandial metabolic outcomes. A p-value of ≤ 0.05 was considered statistically significant. All data are presented as means±SD for normally distributed data or means and 95% confidence intervals for log-transformed data, unless otherwise noted.

Based on the variability of glucose rate of disappearance (Rd) during a HEC procedure in non-diabetic obese subjects we have studied previously (15), we estimated that 7 subjects in each group (RYGB and SG) would be needed to detect a 50% difference in insulin-stimulated glucose Rd between the RYGB and the SG groups with a β-value of 0.20 (i.e., 80% power) and an α-value of 0.05. This proposed difference between groups is a conservative estimate based on the results from the only previous study that compared the effect of SG versus RYGB on glucose Rd by using the HEC procedure, which found more than a 250% greater increase in insulin-stimulated glucose Rd after SG than after RYGB (5).

Results

Body composition and basal metabolic variables

Subjects in the RYGB and SG groups were studied before and after losing 22.0±2.0% and 21.0±2.6% of their body weight, respectively (Table 1). Weight loss induced by RYGB and SG caused a marked decrease in FFM, fat mass, fasting plasma glucose and insulin and concentrations, and HOMA-IR score, but values did not differ between surgical groups (Table 1).

Insulin sensitivity

Weight loss caused a two-fold increase in insulin-stimulated glucose disposal in both RYGB and SG groups, but there were no differences between the surgical groups (Figure 1).

Metabolic response to mixed meal ingestion

Plasma hormones—Surgery-induced weight loss decreased the postprandial plasma insulin AUC in both RYGB and SG groups, but values were not different between groups (Table 1). Both RYGB and SG altered the shape of the insulin and C-peptide concentration curves; the increases in the postprandial peak plasma insulin and C-peptide concentrations above baseline were greater after surgery than before surgery in both groups ($p \leq 0.05$), without a difference between groups ($p \geq 0.60$) (Figure 2).

β-cell function—Total ISR AUC in response to the mixed-meal decreased after both RYGB (30138 [18034, 50373] to 22555 [11948, 42579]) pmol/L) and SG (26633 [18454, 38432] to 24648 [14976, 40570] pmol/L) surgery-induced weight loss ($p=0.03$), and the decrease was not different between groups ($p=0.21$ for interaction). β-cell function increased three to fourfold after weight loss in the RYGB (26.9±11.6 to 105.2±24.1 μmol/kgFFM/min per μIU/mL × pmol/L × 10^3) and the SG (29.3±24.1 to 94.1±42.0 μmol/kgFFM/min per μIU/mL × pmol/L × 10^3) groups ($p<0.001$), but there was no difference between groups ($p=0.651$) (Figure 1).

Glucose kinetics—Postprandial plasma glucose AUC decreased to a similar extent in both groups after surgery-induced weight loss (Table 1). However, both RYGB and SG surgeries resulted in a greater peak in the early rise of plasma glucose, because of a marked increase in the early rate of appearance (Ra) of ingested glucose into the systemic circulation (Figure 3). The percentage of total meal-derived glucose that appeared in the circulation within 60 min after initiating meal ingestion increased from 53±19% before to 85±10% after surgery in the RYGB group and from 55±10% before to 78±13% after surgery in the SG group (p=0.01 for both surgery groups, with no difference between groups). Meal ingestion caused a marked suppression of EGP, so that EGP accounted for less than 10%, while ingested glucose accounted for more than 90%, of postprandial total glucose Ra (Figure 3). Both SG and RYGB surgeries resulted in a more rapid and greater postprandial suppression of EGP and a more rapid return toward baseline (Figure 3).

Discussion

This study was conducted to evaluate whether marked (∼20%) weight loss induced by RYGB has greater therapeutic effects on metabolic function than the same weight loss induced by SG in obese people without T2D. Our findings indicate that RYGB and SG cause similar improvements in both whole body (primarily skeletal muscle) insulin sensitivity, assessed by using the HEC procedure and stable isotopically labeled tracer infusion, and β-cell function, assessed as the relationship between insulin sensitivity and the insulin response to mixed meal ingestion. Both RYGB and SG caused similar changes in postprandial glucose metabolism, manifested by a more rapid delivery of ingested glucose into the systemic circulation, and greater early postprandial suppression of EGP with a more rapid return of EGP toward baseline. These results demonstrate that despite the anatomical differences between RYGB and SG, weight loss induced by both procedures results in rapid glucose absorption and similar improvements in the two major factors involved in regulating glucose homeostasis, namely insulin sensitivity and β-cell function.

The results from our study do not help explain the potential mechanism(s) responsible for the greater rate of diabetes remission observed after RYGB than SG (2, 3). In our subjects, weight loss induced by either RYGB or SG caused the same improvement in two of the major factors involved in the pathogenesis of T2D, i.e. insulin sensitivity and β-cell function. Our results are consistent with those from a previous study, conducted in both diabetic and non-diabetic obese subjects, which found similar improvements in HOMA-IR score after either RYGB or SG (16, 17). In contrast, two other studies conducted in subjects with T2D found conflicting results; insulin sensitivity assessed by applying a mathematical model (18) to data from a mixed meal tolerance test improved more after RYGB than SG in one study (4), whereas insulin sensitivity assessed by using the HEC procedure improved more after SG than RYGB in the other (5). In the former study, Kashyap and colleagues found β-cell function, assessed by the response to a mixed meal, increased to a much greater extent after RYGB than SG despite similar weight loss in both groups (4). The reason for the differences in results among studies is not clear, but could be related to differences in study population and experimental techniques used to assess metabolic function. These discrepant results underscore the need for additional mechanistic studies in patients with T2D.

Our study has several important limitations. First, we might have missed statistically significant differences between groups in some of our outcome measures, because of the small number of subjects who participated in our study. However, the marked improvement in insulin sensitivity, our primary study outcome, was nearly identical after both RYGB and SG, making it unlikely that that we missed a clinically important effect. Second, our study was conducted in subjects who did not have T2D, so our results might not necessarily apply to patients with T2D. Nonetheless, our data have important physiological implications in understanding the effect of RYGB and SG surgeries on metabolic outcomes.

In conclusion, the results from the present study provide additional insights into the effects of marked weight loss induced by RYGB and SG-induced weight loss on insulin sensitivity and the metabolic response to a mixed meal in obese, insulin-resistant people. Our data demonstrate potent, but similar, beneficial effects of weight loss, induced by either surgical procedure, on whole-body insulin sensitivity, β-cell function, and the metabolic response to a mixed meal. Additional studies conducted in patients with T2D are needed to determine whether the observations made in non-diabetic subjects in this study can be extrapolated to people with T2D.

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What is already known about the subject

- Roux-en-Y gastric bypass (RYGB) and sleeve gastrectomy (SG) are the most commonly performed bariatric surgery procedures worldwide.
- Both RYGB and SG cause considerable weight loss and a marked improvement in glycemic control in patients who have type 2 diabetes.
- The results from recent randomized controlled trials demonstrate RYGB is more effective than SG in causing remission of type 2 diabetes.

What does this study add to existing knowledge

- The mechanism(s) responsible for the observed difference in therapeutic efficacy between RYGB and SG procedures is not known. This is the first study to evaluate the major mechanisms involved in the pathogenesis of type 2 diabetes, namely insulin sensitivity, β-cell function, and the metabolic response to a mixed meal, in obese subjects after the same, marked RYGB and SG induced weight loss.
- Our data demonstrate potent, but similar, beneficial effects after both RYGB and SG procedures on whole-body insulin sensitivity (assessed by using the hyperinsulinemic-euglycemic clamp procedure in conjunction with stable isotopically labeled tracer infusion), β-cell function, and the metabolic response to a mixed meal in subjects matched for weight loss.
- The results from the present study provide new insights into the effects of RYGB and SG induced weight loss on insulin sensitivity, β-cell function beta and the metabolic response to meal ingestion in obese, insulin-resistant people.

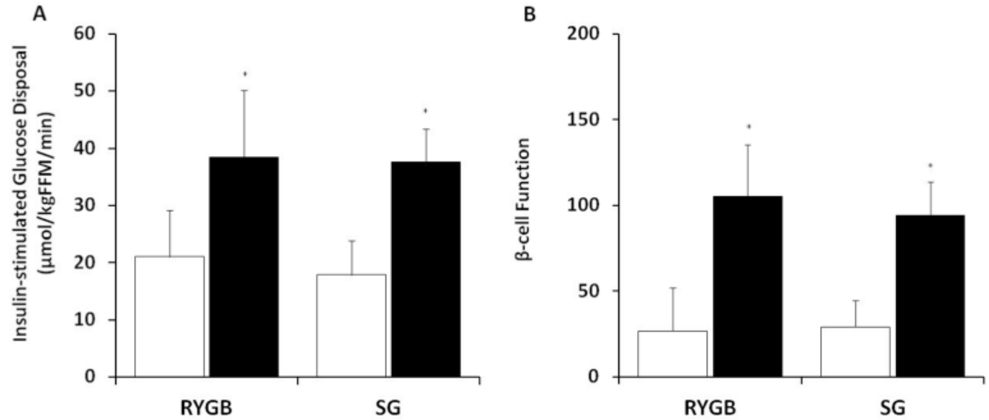

Figure 1.

Insulin-stimulated glucose disposal (assessed by using the hyperinsulinemic-euglycemic clamp procedure) (A) and β-cell function (based on the relationship between insulin secretion during a mixed meal and insulin sensitivity) (B) before (white bars) and after (black bars) ~20% weight loss induced by Roux-en-Y gastric bypass (RYGB) or sleeve gastrectomy (SG) surgeries. *Value significantly different from value before surgery, $p<0.005$. Values are means±SEM.

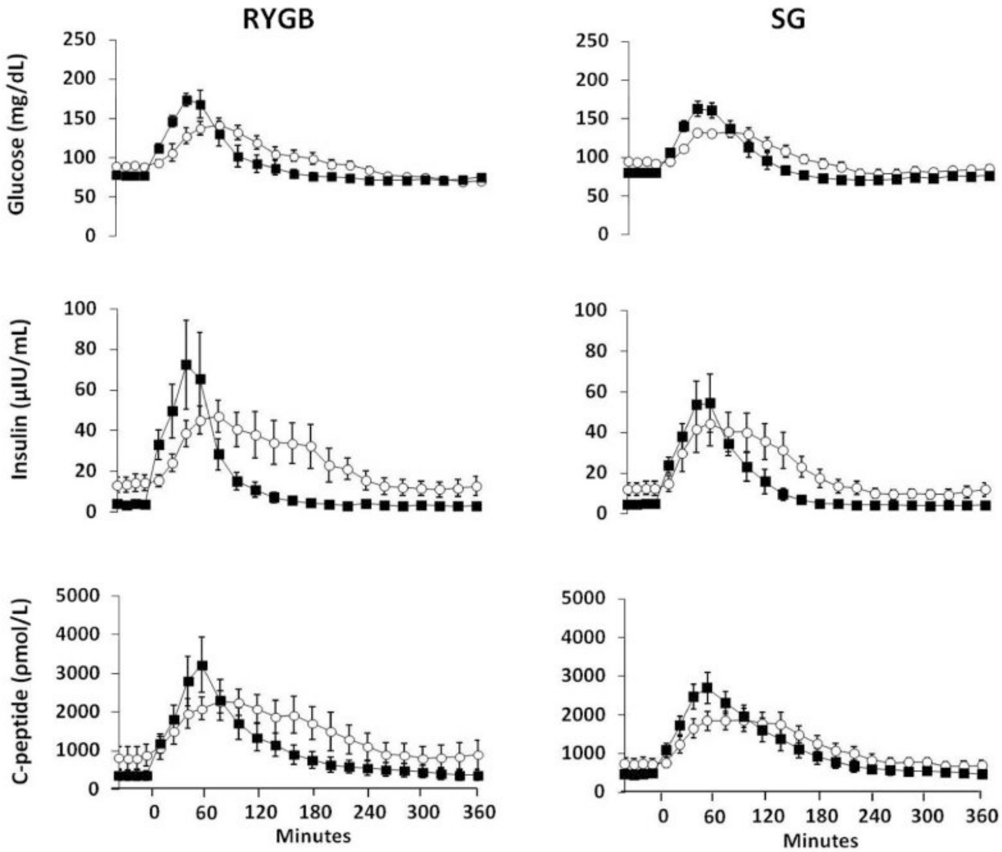

Figure 2.

Plasma hormone concentrations after ingestion of a mixed meal (consumed from 0-30 min) before (white circles) and after (black squares) ~20% weight loss induced by roux-en-Y gastric bypass (RYGB) or sleeve gastrectomy (SG) surgeries. Plasma glucose, insulin and C-peptide concentrations areas under the curve were significantly different after than before surgery in both RYGB and SG groups (all p-values <0.001), but there were no significant differences between surgical groups (all p-values for interaction >0.42). Values are means ±SEM.

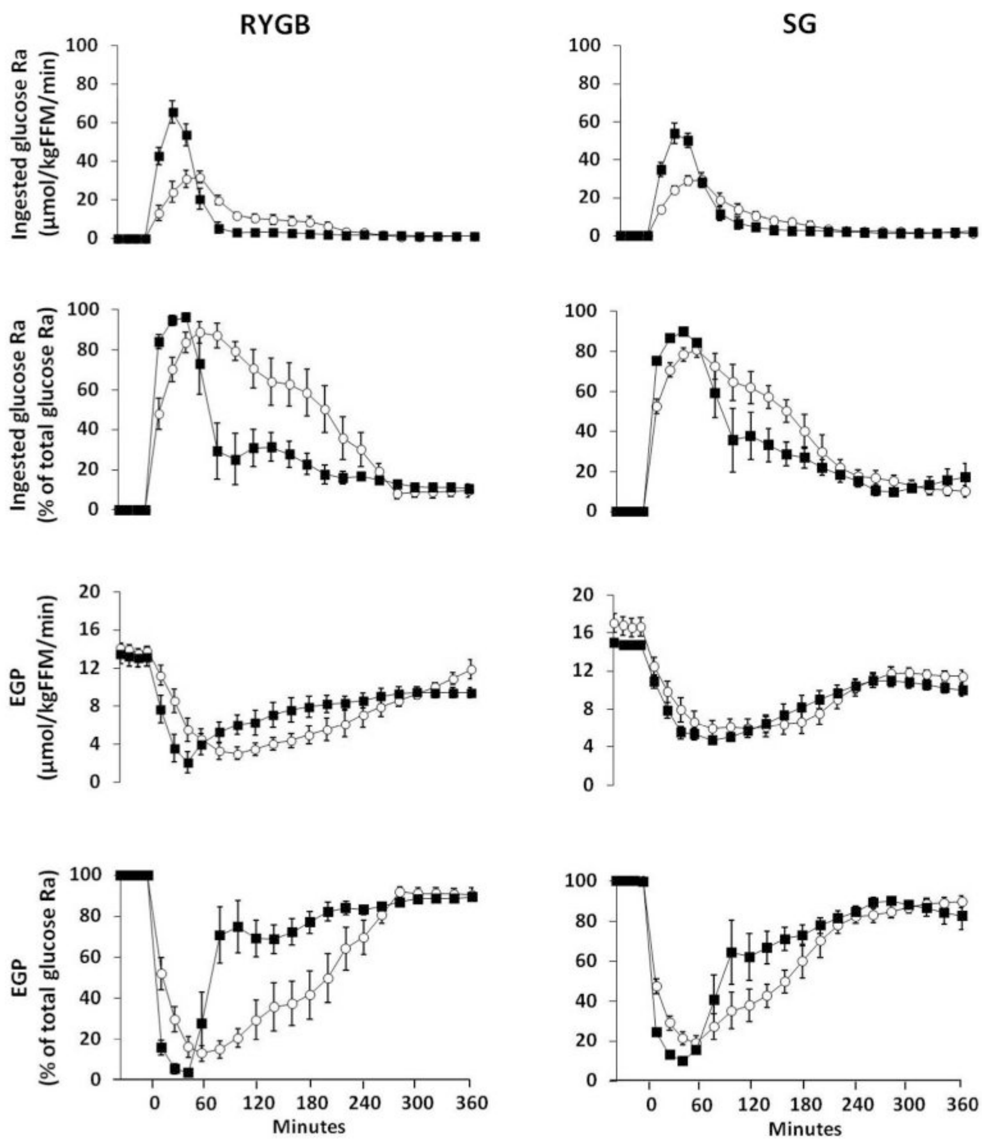

Figure 3.

Rate of appearance (Ra) of ingested glucose into the systemic circulation, ingested glucose Ra as a percentage of total glucose Ra, rate of endogenous glucose production (EGP), and EGP as a percentage of total glucose Ra after ingestion of a mixed meal (consumed from time 0-30 min) before (white circles) and after (black squares) ~20% weight loss induced by Roux-en-Y gastric bypass (RYGB) or sleeve gastrectomy (SG) surgeries. The changes in ingested glucose Ra and EGP after surgery were not different between RYGB and SG groups (all p-values for interaction >0.25). Values are means±SEM.

Table 1

Body composition and metabolic variables before and after weight loss induced by roux-en-Y gastric bypass and sleeve gastrectomy.

	RYGB		SG	
	Before	After	Before	After
Body weight (kg)	147.5±23.0	115.3±20.4*	146.5±14.3	115.9±13.0*
BMI (kg/m^2)	50.0±3.9	38.9±3.5*	54.9±8.5	43.5±7.6*
Fat-free mass (kg)	68.1±13.0	60.5±12.8*	64.9±10.2	60.1 ±6.7*
Fat mass (kg)	79.2±12.2	55.5±10.2*	78.8±14.6	56.5±13.1*
Fat mass (% body weight)	53.9±3.6	48.0±4.0*	54.7±7.5	48.1±2.7*
Plasma glucose (mg/dL)	89.6±7.2	78.8±7.7*	94.5±9.8	80.0±3.9*
Plasma insulin (μU/mL)	13.4±10.2	4.1±2.1*	12.2±7.4	4.7±2.5*
HOMA-IR score	5.9±2.6	1.8±0.9*	6.5±3.4	2.9±1.5*
Glucose AUC (mg/dL*360 min)*10^3	38.1±2.8	36.0±3.8*	38.3±4.2	35.9±2.9*
Insulin AUC (μU/mL*360 min)*10^3	9.5±5.8	5.7±3.9*	8.1±4.7	5.5±2.7*

Values are means±SD.

*Value significant different from value before surgery ($p < 0.05$). There were no significant main effects of group or time x group interactions.

RYGB: Roux-en Y gastric bypass; SG: sleeve gastrectomy; HOMA-IR: homeostasis model assessment of insulin resistance.