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Association Between Appetite-Regulating Hormonal Levels and Body Composition Among Adults With Pre-operative Bed Undergoing Bariatric Surgery: A Prospective Cohort Study Protocol

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Abstract

Introduction: Severe obesity, defined by a body mass index (BMI) of >35 kg/m^2, is a health concern affecting one in eight individuals worldwide and one in three Canadians (1, 2, 3). Metabolic bariatric surgery (MBS) is a safe and effective treatment that offers long-term success compared to conventional methods like pharmacotherapy and behavioural control. However, post-operative weight regain remains common, particularly among MBS candidates with binge-eating disorder (BED). The objective of this study is to comparatively examine the association of appetite-regulation hormones, namely ghrelin and leptin, with post-laparoscopic sleeve gastrectomy (LSG) and post-Roux-en-Y gastric bypass (RYGB) weight maintenance and binge-eating behaviours among individuals with preoperative BED.

Methods: This is a prospective cohort study to examine changes in ghrelin and leptin levels in adult patients with pre-operative BED undergoing LSG or RYGB. A total of 90 will be recruited from bariatric centers of excellence in Ontario, Canada. Summary statistics and anthropometrics will be measured pre-operatively and at 1, 3-, 6-, 12-, and 24-months post-surgery. Binge eating will be assessed using the Binge Eating Scale (BES).

Anticipated Results: Ghrelin and leptin levels will vary by surgery type, due to RYGB's association with higher 24-month ghrelin levels than LSG. Lower 12-month post-MBS BES scores will predict lower weight regain 24-months post-MBS. Weight loss is anticipated in both groups, with no significant difference based on surgery type.

Discussion: The study aims to examine the influence of surgery type-specific ghrelin and leptin levels on binge-eating behavior and post-operative weight maintenance. Differences in hormonal regulation and weight regain between LSG and RYGB patients may be correlated, given that RYGB patients are reported to experience more favourable long-term outcomes. Elevated ghrelin levels post-RYGB are paradoxically associated with lower BED symptoms, potentially promoting long-term weight stability. These findings could inform personalized post-operative care strategies and promote long-term hormonal monitoring/therapy.

Conclusion: Understanding the hormonal mechanisms underlying post-MBS weight regain and binge-eating behaviours helps optimize long-term patient outcomes. Long-term research on the stability of ghrelin and leptin levels, and the influences of other appetite-regulating hormones, would provide insight into sustained metabolic changes post-MBS.

Keywords: eating behaviour; bariatric surgery; binge-eating; ghrelin; leptin; postoperative outcomes; weight regain

Introduction

Globally, one in eight people have obesity [1]. The prevalence of obesity has increased in Canada especially, affecting one in three Canadians aged 18 and older in 2022 as opposed to around one in five in 2003[2]. Severe obesity is classified as having a body mass index (BMI) ≥ 35 kg/m^2 when associated with comorbidities [3]. These comorbidities can include, but are not limited to: cardiovascular disease, type 2 diabetes, hypertension, and musculoskeletal issues [2]. Despite the availability of behavioural and pharmacological interventions, studies report around 74% of individuals fail to sustain total body weight loss of 10% or greater after four years with these methods [3]. Metabolic bariatric surgery (MBS) is a safe and effective treatment option for severe obesity. Reported benefits of the surgery include increased mobility, self-esteem, and psychosocial functioning , contributing to greater psychological well-being and quality of life [3]. Specifically, the laparoscopic sleeve gastrectomy (LSG) and the Roux-en-Y gastric bypass (RYGB) are two restrictive and malabsorptive procedures of MBS that alter the anatomy and physiology of the gastrointestinal (GI) tract (Figure 1). LSG and RYGB are associated with high 24-month success rates of 74% and 88% (success being

defined as ≥60% EWL), respectively [4]. Preventing excessive weight regain is ideal to avoid late reoperation. Thus, the contribution of pre-operative and post-operative eating behaviours to post-MBS weight maintenance and associated health outcomes is a major point of interest in scientific literature.

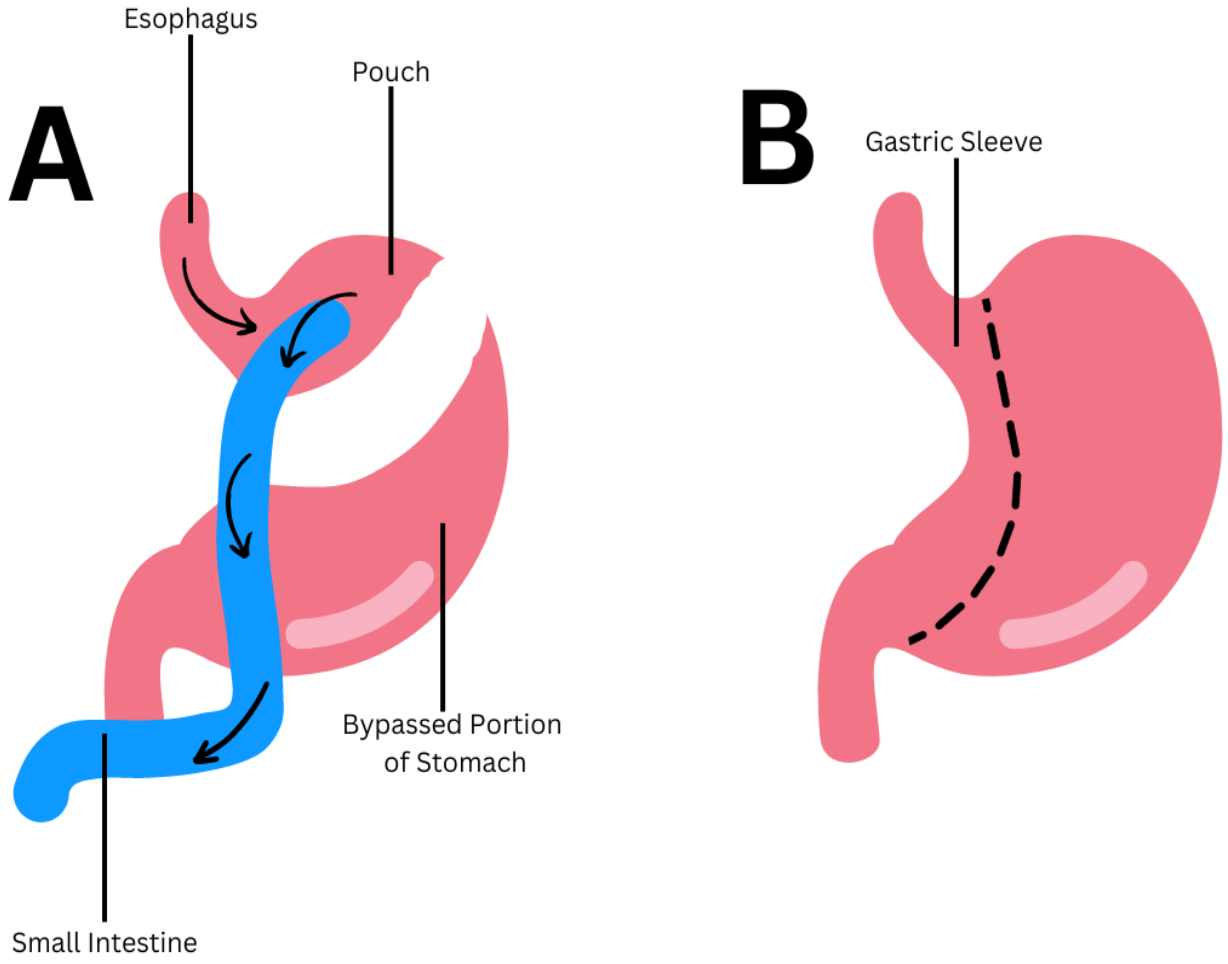

Figure 1. Simplified anatomical rearrangement of GI Tract pictured (A) RYGB and (B) LSG. Figure created using Canva.

Psychiatric conditions are frequently observed among patients seeking MBS. Depression, binge-eating disorder (BED), and anxiety are the three most recognized disorders in this regard [5]. BED is prevalent in 15.7-26.6% of individuals seeking bariatric surgery and has been heavily associated with long-term weight regain [6]. BED is defined by frequent, uncontrolled consumption of large quantities of food without compensatory actions like vomiting or laxative use. Research findings are mixed, however, with some studies demonstrating direct associations between preoperative BED and unfavourable postoperative outcomes while others fail to observe significant correlations. Potential post-operative dysregulation of appetite hormones, such as ghrelin and leptin, due to anatomical changes post-MBS could influence the persistence of binge-eating behaviours. Figure 2 displays the physiological effects of ghrelin.

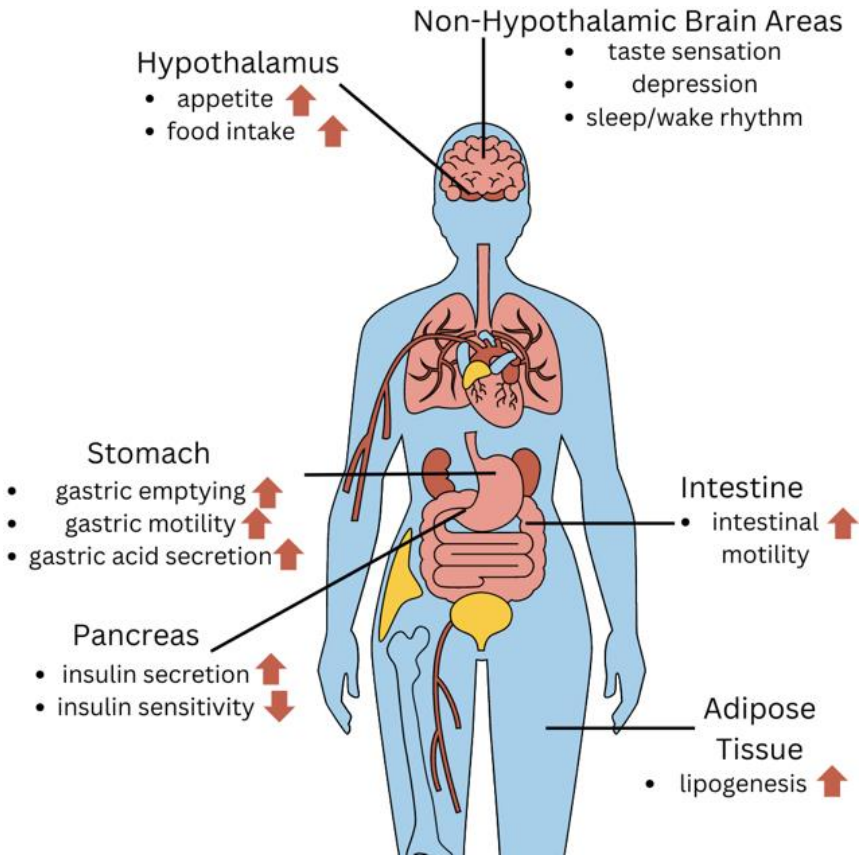

Figure 2. Simplified physiological effects of ghrelin discovered over the past 15 years. Created using Canva and adapted from Müller et al. [7].

In 2002, a foundational study by Cummings et al. suggested that ghrelin production increases in response to weight loss to maintain energy homeostasis, causing weight regain after conventional treatment (i.e. pharmacotherapy and behavioural changes) in 80% of cases [8]. However, ghrelin is secreted by cells in the gastric fundus, which is commonly removed or bypassed in various MBS procedures [7]. Post-MBS ghrelin suppression may help sustain long-term weight loss and reduce vulnerability to binge-eating behaviour. Leptin, in contrast, is synthesized by white adipose tissue and acts on the hypothalamus to inhibit unnecessary energy intake [9]. Decreased adipose tissue post-surgery results in decreased leptin production, antagonistically reducing hunger suppression. Current literature lacks a comprehensive understanding of how changes in appetite hormones like ghrelin and leptin specifically impact patients with binge-eating disorder (BED) after bariatric surgery. The influence of these hormonal changes on post-operative weight management, eating behaviours, and body composition in this population remain unclear. The objectives of this proposed study are to examine the post-MBS changes in ghrelin and leptin levels in adult patients pre-operatively diagnosed with BED. Associations of post-MBS changes in appetite hormone levels with post-operative BED symptoms and body composition/weight outcomes will also be determined.

Methods

Study Design

This protocol follows the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines to ensure accurate and applicable findings can be concluded from conducted observational research [10]. A prospective cohort study to examine the effect of post-MBS changes in ghrelin and leptin levels on weight maintenance in SG and RYGB patients with pre-operative BED will be conducted. Assessment of patients will take place up to three months pre-surgery and at the following 5 timepoints post-surgery: 1, 3, 6, 12, and 24 months. Patient care will follow the Enhanced Recovery After Surgery (ERAS) Society guidelines [11].

Recruitment

A total of 90 participants aged 18-65 will be identified by random sampling from bariatric centers of excellence in Ontario. These recognized centers include: Guelph General Hospital, London Health Sciences Centre, St Joseph's Healthcare Hamilton, Unity Health Toronto, and Humber

River Health. Informed by the success of multiple comparative studies conducted on long-term changes in appetite-regulating hormones in patients undergoing RYGB and SG with similar sample sizes, a sample size of 90 would be ideal [3, 6, 12]. This sample size will ensure accuracy in statistical analyses of changes in hormone levels and behavioural outcomes while accounting for an estimated 22% participant dropout rate and adequately controlling confounding variables. Alignment in sample sizes with other studies that focus on hormonal changes, binge-eating, or both outcomes enables direct comparisons and validation of findings.

Bias

Random sampling and the diversity in participant recruitment with a wide age range prevents selection bias and demographic underrepresentation. Measurement bias is addressed by anonymously using validated tools like the Binge Eating Scale (BES) to reduce tendencies of patients to answer dishonestly in fear of judgement. Standardized procedures will be used for hormone level measurements, such as consistent blood sample collection times and the use of Enzyme-Linked Immunosorbent Assay (ELISA) kits. To ensure participant safety and maximize accuracy, three categories of potential confounding variables must be controlled: demographic, medical history, and lifestyle factors. Baseline demographic characteristics would be collected to understand age and sex distribution. The aim would be an even participant distribution by sex and a mean age of 45.0 +/- 11.5 to accurately represent the varied effects on young, middle-aged, and older males and females.

Eligibility

Patients who are about to undergo MBS and pre-operatively diagnosed with BED using the Structured Clinical Interview for DSM-IV Disorders (SCID) are eligible. Participants are to be excluded if they have a history of cancer, are currently pregnant, or have been dieting and/or used hormonal contraceptives within the last six months. Participants are to be excluded if they had tobacco for >4 weeks and alcohol 1-2 years before surgery.

Measures

Binge-eating Behaviour

The Binge Eating Scale (BES) is a 16-item self-report questionnaire designed to assess behavioral and emotional/cognitive symptoms of binge eating post-MBS [13]. As a Likert-type scale, each item on the BES offers 3 to 4 response options, spanning a spectrum of severity. Total scores on the BES can range from 0 to 46, based upon which individuals can be categorized into three groups: a) no binge eating (score $\leq$ 17), b) mild to moderate binge eating (score of 18 – 26), and c) severe binge eating (score $\geq$ 27). Patients must pre-operatively complete the BES and fall in group b) or c). BES scores will be updated 12-months and 24-months post-operatively. A change in score of 3 points or more that causes participants to change group classifications will be considered clinically meaningful.

Plasma Ghrelin and Leptin Levels

For each patient, a blood sample will be collected at a designated clinical laboratory in Ontario between 7:00 a.m. and 9:00 a.m. by a qualified healthcare professional. Serum total ghrelin (pg/mL) and serum leptin (pg/mL) levels will be determined using commercially available ELISA (Enzyme-Linked Immunosorbent Assay) kits. Participants will be required to fast for at least 8-12 hours before blood collection.

Anthropometrics

The Eufy by Anker scale will be used to record body composition statistics (i.e. body fat, BMI, and weight) during physician consultations at each timepoint. The Eufy scale uses bioelectrical impedance analysis (BIA), which involves measuring resistance to current flow, to estimate body fat percentage. Height will be measured using a stadiometer.

Statistical Analysis

Descriptive statistics will be recorded for demographic characteristics (age, gender, surgery type) pre-operatively. Summary statistics will be reported as means +/- standard deviations for plasma ghrelin and leptin levels, weight, BMI, and BES scores at each time point. Statistical analyses will be performed using MedCalc version 10.3.0.0 (MedCalc Software, Ostend, Belgium) and SPSS for Windows version 16.0 (IBM Corporation, Armonk, New York, NY, USA). Graphical visualization will be facilitated by Python's matplotlib software for the trajectories of ghrelin and leptin levels, weight changes, and BES scores over time. Pearson's correlation coefficient (r) will be computed to examine the relationships between ghrelin and leptin levels, weight loss outcomes, and changes in binge-eating behaviors. Multiple regression analysis will be conducted to assess the predictive value of: a) BES scores on weight regain, and b) ghrelin and leptin levels on sustained weight loss and improvements in binge-eating behaviors. Results will be adjusted for pre-operative BMI.

Anticipated Results

Participant Characteristics

A sample size N=90 will be recruited, ideally with an even distribution of participants by sex and surgery type. However, a higher proportion of females and SG patients is expected (Table 1). Means and standard deviations of demographic statistics determining similarities in body composition and anatomical function of the study group are summarized in Table 1. Of 90 patients, around 78% (n = 70) are expected to attend all post-operative clinics [14].

Table 1. Participant Characteristics at Baseline (N=90)

Parameters	Mean +/- SD *
LSG (%)	54 (60%)
RYGB (%)	36 (40%)
Male (%)	34 (37.78%)
Female (%)	56 (62.22%)*
Age (years)	45.0 +/- 11.51
Height (m)	1.7 +/- 0.37
Weight (kg)	137.1 +/- 13.44
BMI (kg/m$^{2)}$	49.3 +/- 7.98
BES score	34 ± 5.1

Values with * indicate units of N (%).
**Anticipated values were calculated based on participant recruitment in existing studies [5, 14, 15].

Plasma Ghrelin Levels (Figure 3)

At 3-months pre-operation, individuals with BED are expected to have low baseline ghrelin levels (109.6 ± 32.6 fmol/ml) due to ghrelin hyposecretion in individuals with obesity [16]. 3 months post-operatively, ghrelin levels are anticipated to enter a plateau phase, stabilizing at 43.7 ± 11.3 fmol/ml. A mild rise in mean ghrelin levels, though statistically insignificant, is anticipated to a concentration of 44.8 ± 13.2 fmol/ml 6 months post-operatively [16]. 12 months and 24-months post-LSG, a gradual but significant increase to a concentration of 50.2 ± 10.9 fmol/ml and 54.3 ± 10.6 fmol/ml [16], respectively, is anticipated.

At 1-month post-operatively, mean ghrelin levels in post-RYGB patients are expected to be stable around 75.27 ± 15.6 fmol/ml [16]: significantly lower than baseline, but higher than post-LSG levels. No significant difference is expected at the 3-, 6-month and 12-month checkpoints. 24 months post-RYGB, ghrelin levels are expected to increase to concentrations comparable to baseline values around 101.9 +/- 30.3 fmol/ml [16].

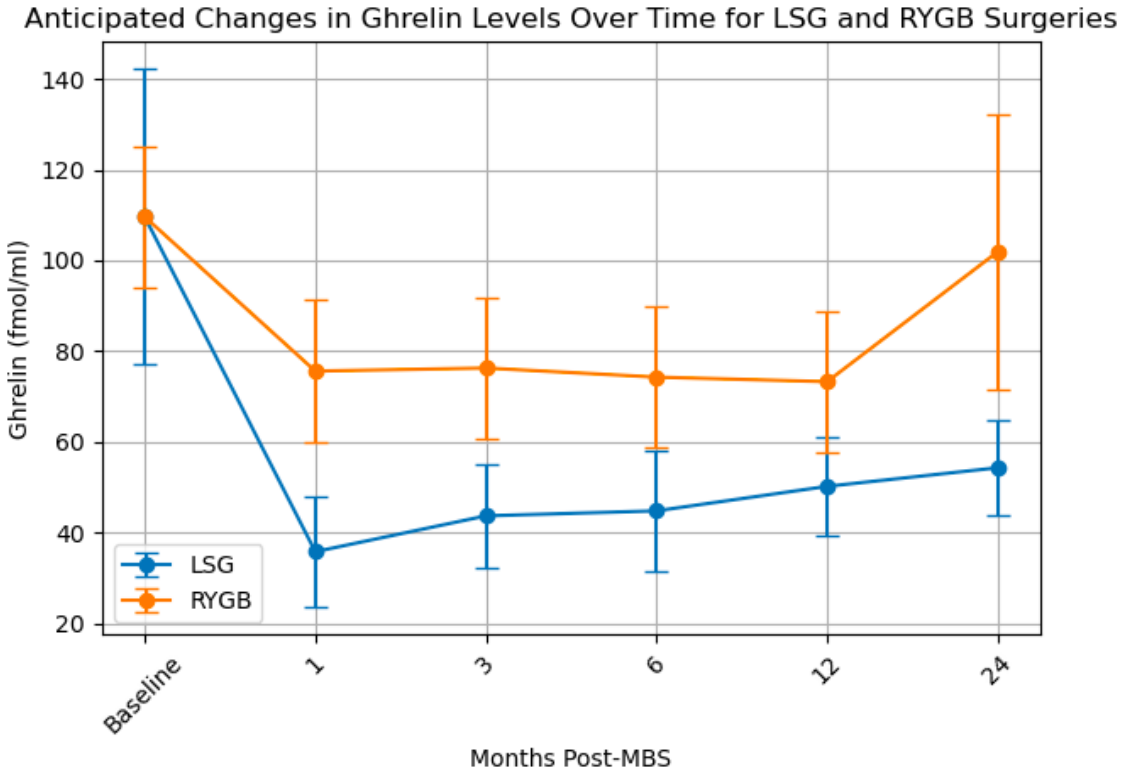

Figure 3. Anticipated changes in post-LSG and post-RYGB mean ghrelin levels +/- standard deviation contrasted across six timepoints. Figure created using Python software.

Plasma Leptin Levels (Table 2)

3-months pre-surgery, mean leptin levels are anticipated to be 251.9 +/- 45.5 ng/mL [17]. 1-month post-LSG and post-RYGB levels are expected to be half of baseline concentrations at 125.5 +/- 35.3 ng/mL and 105.5 +/- 23.3 ng/mL, respectively. Leptin levels are anticipated to continuously decrease in proportion to weight loss until 12-months post-MBS [12]. Predicted values and trends are listed in Table 2. A mean change +/- SD from baseline of –168.7 +/- 29.4 ($p < 0.001$, highly significant) is expected for both LSG and RYGB patients, resulting in mean leptin levels stabilizing at 83.2 +/- 29.3 ng/mL at 12-months post-MBS. No significant difference is anticipated between the 12-month and 24-month checkpoints, regardless of surgery type [12].

Table 2. Anticipated Changes in post-LSG and Post-RYGB Mean Leptin Levels Contrasted Across the Six Timepoints

Time passed post-MBS (months)	Leptin levels (ng/mL)	
	LSG	RYGB
Baseline	251.9 +/- 45.5	
1-month	125.5 +/- 35.3	105.5 +/- 23.3
3-months	111.3 +/- 31.2	97.3 +/- 25.7
6-months	96.7 +/- 20.1	90.2 +/- 26.9
12-months	83.2 +/- 29.3*	
24-months	85.7 +/- 26.2*	

*The mean leptin levels for all participants have been reported as one value at the 12- and 24-month checkpoints due to the lack of a significant difference requiring division.
**Values were calculated by referencing existing studies [12, 15].

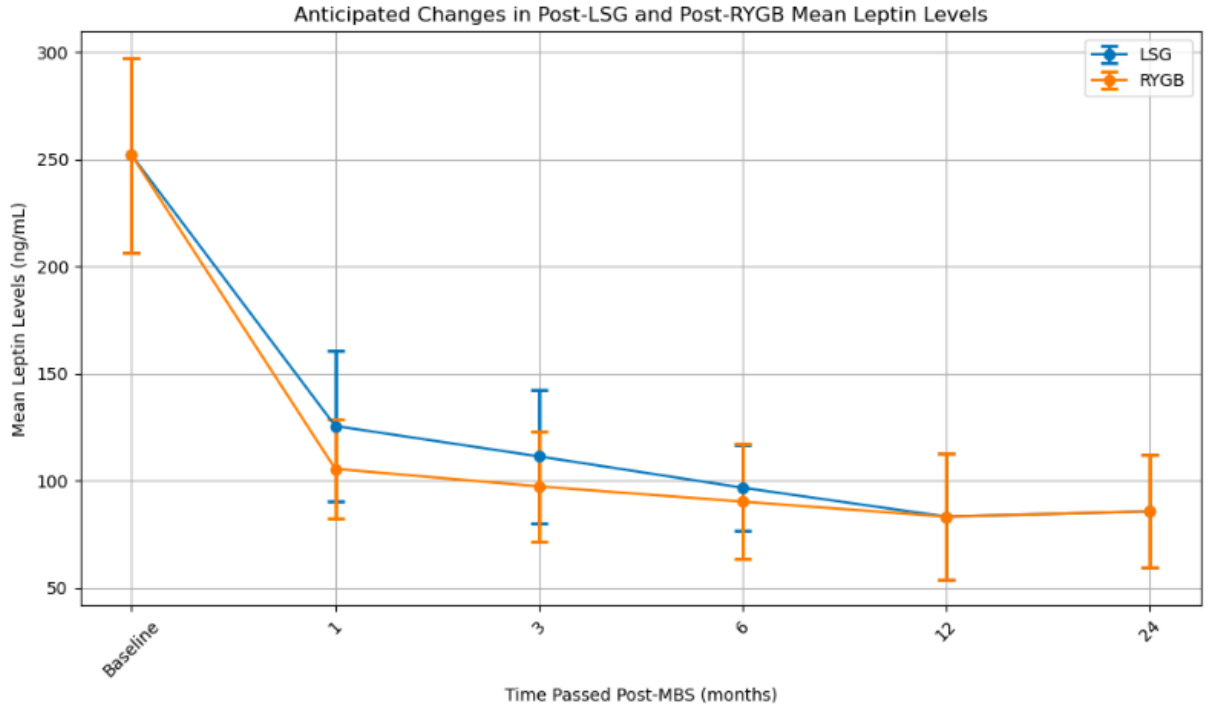

Figure 4. Anticipated changes in post-LSG and post-RYGB mean leptin levels +/- standard error contrasted across the six timepoints. Figure 4, constructed from the anticipated values in Table 2, displays the stabilization of leptin levels over time. Figure created using Python software.

Weight Outcomes

Pre-operatively the mean weight and BMI are expected to be 137.14 +/- 13.10 kg and 49.3 +/- 4.93 kg/m^2 respectively [5, 14, 15]. Weight loss is anticipated to be statistically significant between each checkpoint over the first 12 months post-operatively (Table 3) [6]. There is no

predicted difference between weight, BMI, or %EWL outcomes based on surgery type [6], resulting in means calculated including both post-LSG and post-RYGB patients (Figure 5).

Weight Regain

The anticipated nadir weight is 86.5 kg ± 8.9 [19]. 10% of patients are expected to regain more than 15% of nadir weight by the final 24-month checkpoint [18]. Median (range) weight regain will be measured as the percentage of maximum weight lost; it is expected to be 35.0% post-LSG and 24.7% post-RYGB [18].

Table 3. Anticipated Mean Weight (kg) and % Excess Weight Loss (%EWL) of Study Population at All Checkpoints

Parameters (mean ± SD)	Baseline	1 month	3 months*	6 months*	12 months*	24 months*
Weight (kg)	137.14 ± 13.10	128.24 ± 15.73	111.03 ± 13.24*	104.02 ± 11.61*	86.5 ± 8.9*	90.0 ± 7.6*
BMI	49.3 +/- 4.92	45.3 +/- 3.21	41.2 +/- 3.82	38.6 +/- 2.92	32.3 +/- 2.76	34.2 +/- 2.88
%EWL		9.80 ± 2.29	29.5 ± 4.58*	50.11 ± 7.10*	69.5 ± 6.7*	63.7 ± 5.3*

Values with * are statistically significant.

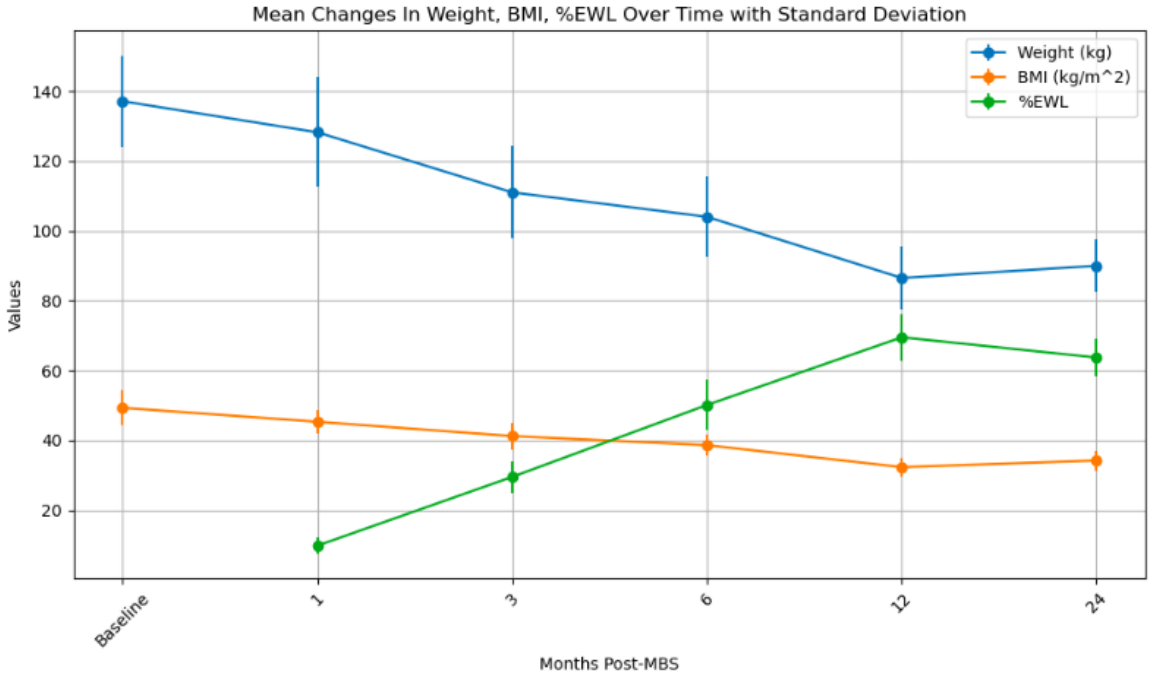

Figure 5. Anticipated changes in weight, BMI, and %EWL +/- standard error contrasted across the six timepoints. Figure created using Python software.

Binge-Eating Behaviour (BE):

A significant decrease in BE post-MBS is expected, along with a positive correlation to postoperative weight loss [20]. 3-months post-MBS, the change in BES scores is expected to be too incoherent between participants toevaluate their correlations with ghrelin and leptin levels or weight loss. 6-months post-MBS, weight loss is anticipated to be proportional to observed decreases in BES scores, resulting in mean BES scores +/- SD of 22.0 +/- 6.4 [20]. 12-months post-MBS, mean BES scores are expected to stabilize around 13 +/- 7.2. Post-MBS, 80% of initial participants are expected to fall in group (a): the category of normalized eating [20]. 15% are expected to fall in group (b): moderate binge-eating, followed by 5% in group (c): severe binge-eating [20]. BES scores are expected to have predictive value at the 12-month checkpoint regarding weight regain at the 24-month checkpoint (Figure 6). No significant difference in BES scores between the 12- and 24-month checkpoints is anticipated.

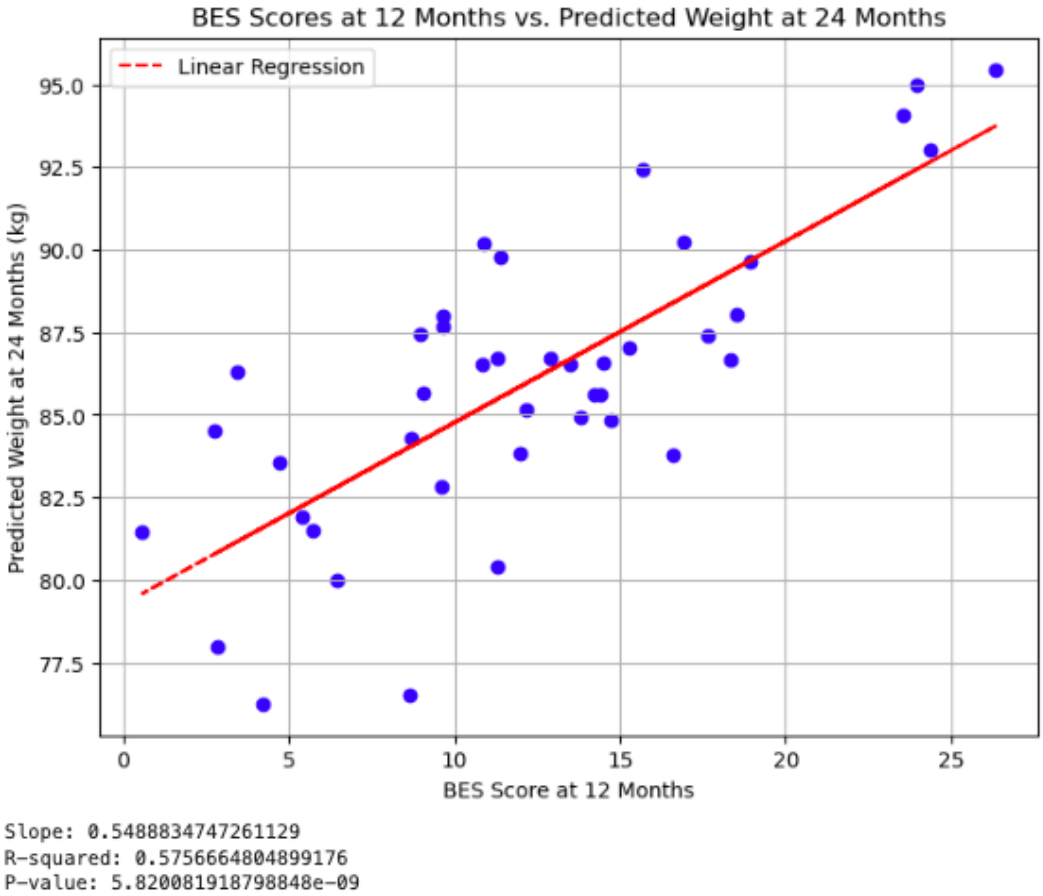

Figure 6. Anticipated positive correlation of BES scores at the 12-Month checkpoint with lower weights observed at the 24-Month checkpoint. Figure created using Python software.

Discussion

This study will demonstrate the influence of surgery type-dependent ghrelin and leptin levels on 12-months post-MBS binge-eating behaviour, which is a statistically significant predictor of weight regain at the 24-month checkpoint. Lower 12-month post-MBS BES scores are expected to predict lower weight regain [20]. The expected R-squared value of 0.575 suggests that the correlation between these two factors is moderately positive and statistically significant (Graph 4). Higher ghrelin levels are anticipated 24-months post-RYGB as opposed to post-LSG, although paradoxically associated with lower BES scores [20]. This could contribute to the median percentage of weight regain being greater post-LSG than post-RYGB. No significant difference in leptin levels between patients of various surgery types 24-months post-MBS is anticipated [20].

Anatomically, the RYGB procedure forms a vertical pouch bypassing a large portion of the stomach, including the ghrelin-producing fundus [3]. The reduction in ghrelin-producing cells post-RYGB is not as significant as post-LSG. A statistically significant decrease in ghrelin levels should be observed 1-month post-RYGB, although still expected to be double the ghrelin levels 1-month post-LSG. Initially, the pouch formed through the RYGB procedure retains 1/16th of the standard pre-operative stomach capacity but can stretch over time, increasing hormonal secretion and food consumption [17]. This increases the probability of post-RYGB ghrelin levels returning to baseline values 24 months post-MBS [9]. Anatomically, the LSG procedure involves removing a large portion of the fundus to create a banana-shaped stomach sleeve[5]. This decreases gastric capacity, causing satiety and discomfort with limited food intake.

Ghrelin is a 28-amino acid peptide produced by the proximal intestine and the fundus [16]. Conventional weight loss results in increased ghrelin production with the purpose of increasing appetite and preventing starvation. However, due to the removal of most ghrelin-producing cells, baseline levels are expected to be halved 1-month post-LSG and maintained for 24 months [16]. Baseline plasma ghrelin levels are lower in individuals with binge-eating behaviours [19]. Lower ghrelin levels post-LSG initially reduce hunger, increasing cognitive restraint and enabling adherence to a low-calorie diet sustaining weight loss [20]. However, continually low ghrelin levels could

decrease one's ability to recognize feelings of satiety due to smaller postprandial declines in ghrelin. This could promote impulsive, uncontrolled bouts of consumption, forcibly increasing gastric capacity and dysregulating hormonal secretion relating to meal initiation and metabolic health (i.e. insulin, leptin...etc.) [20]. This potentially explains the paradoxical association of lower 24-month ghrelin levels with higher BES scores and greater weight regain as opposed to post-RYGB. This study affirms the need to research challenges made by recent studies to the currently understood role of peripheral ghrelin in appetite regulation [12, 25], especially concerning the neglected implications for MBS candidates with or without BED.

Leptin is an anorexigenic adipokine secreted in proportion to adipose tissue size [22]. A study by Talalaj et al. reports a 50% and 39-44% decrease in adipose tissue 12-months post-RYGB and post-LSG, respectively [22]. The surgical reduction in leptin-producing cells decreases blood leptin levels, consequently promoting binge-eating behaviours like frequent overconsumption from a lack of satiety. Following weight regain and an increase in adipose tissue between the 12-month and 24-month checkpoints, leptin levels are expected to rise gradually, potentially aiding with cognitive restraint related to binge-eating 24-months post-MBS [22]. In this study, %TWL is expected to be around 30% at the 24-month checkpoint [22]. Studies by Chang et al., Adams et al. report %TWL to be around 20-30% 10 and 12-years post MBS, respectively, suggesting that the initial weight regain may be necessary to elevate leptin levels [23, 24, 25]. When sufficient leptin levels are reached 24- or 36-months post-MBS, long-term weight stability is expected after the initial weight regain of around 5-10kg [24].

A limitation of this study is the prediction of correlations based on short-term hormonal secretion, binge-eating, and weight loss outcomes. Further research must be done on the stability of ghrelin and leptin levels, and their consequent influence on binge-eating behaviours, past the 24-month checkpoint until 10-years post MBS. Another limitation is the omission of post-MBS measurements of other hormones and adipokines involved in appetite regulation and metabolic health. Hormones able to influence post-MBS binge-eating behaviours and weight regain, alone or in combination with others, include: GLP1, an incretin that reduces appetite and regulates glucose metabolism; peptide YY (PYY), an incretin that slows gastric emptying; adiponectin; insulin; and cholecystokinin [25]. Strengths of this study include: its heavy focus on ghrelin, leptin, RYGB, and LSG allowing for a comparative and comprehensive hormonal analysis; validated tools to assess binge-eating behaviours; serum markers; detailed timepoint analysis of changes in weight, BMI, %EWL, and hormonal levels; graphical representation for ease of visual comprehension; and correlation with binge-eating behaviour adding depth to the understanding of post-operative eating behaviours.

Conclusions

This study proposes to compare changes in ghrelin and leptin levels post-LSG and post-RYGB, revealing their roles in post-operative weight maintenance and binge-eating behavior in patients with pre-operative BED. The anticipated findings suggest that surgery type-specific differences in ghrelin and leptin levels may influence weight regain and the persistence of binge-eating behaviors, with RYGB offering more favorable outcomes 24-months post-operatively compared to LSG in this regard.

Understanding the hormonal mechanisms underlying weight regain and binge-eating behaviors post-MBS could aid with the personalization of post-operative care. Additional psychological support, pharmacotherapy targeting appetite regulation, or modifications to surgical techniques could be based upon predicted hormonal correlations.

Future research should extend beyond the 24-month post-operative period to evaluate the long-term stability of ghrelin and leptin levels, and their continued impact on weight and eating behaviors. The interactions of other hormones and adipokines, such as GLP-1, PYY, adiponectin, insulin, and cholecystokinin, should be investigated for a more accurate and comprehensive understanding of appetite regulation.

These insights could optimize both weight outcomes and quality of life for bariatric patients by enhancing recovery protocols and long-term management strategies. The replication and expansion of these findings in larger, more diverse populations and across different types of bariatric procedures should be encouraged to validate, refine, and increase application of the predictive models established in this study.

List of Abbreviations Used

BE: binge-eating behaviour
BED: binge-eating disorder
BES: binge-eating scale
BMI: body mass index
DSM: diagnostic and statistical manual of mental disorders
ELISA: enzyme-linked immunosorbent assay
EWL: excess weight loss
GLP-1: glucagon-like peptide 1
LSG: laparoscopic sleeve gastrectomy
MBS: metabolic bariatric surgery
PYY: peptide YY
RYGB: roux-en-Y gastric bypass
STROBE: strengthening the reporting of observational studies in epidemiology

Conflicts of Interest

The author declares that they have no conflict of interests.

Ethics Approval and/or Participant Consent

The study is approved by the provincial Health Research Ethics Authority. Participants are to provide written consent to take part in the study.

Authors' Contributions
RA: made substantial contributions to study design and planning, analysis and interpretation of existing data to interpolate and infer anticipated results; drafted the work and revised it critically; and gave final approval of the version to be published with full accountability taken.

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