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Weight Regain After Bariatric Surgery: Scope of the Problem, Causes, Prevention, and Treatment

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Abstract

Purpose of Review Although bariatric surgery is the most effective treatment of severe obesity, a proportion of patients experience clinically significant weight regain (WR) with further out from surgery. The purpose of this review is to summarize the prevalence, predictors, and causes of weight regain.

Recent Findings Estimating the prevalence of WR is limited by a lack of consensus on its definition. While anatomic failures such as dilated gastric fundus after sleeve gastrectomy and gastro-gastric fistula after Roux-en-Y gastric bypass can lead to WR, the most common causes appear to be dysregulated/maladaptive eating behaviors, lifestyle factors, and physiological compensatory mechanisms. To date, dietary, supportive, behavioral, and exercise interventions have not demonstrated a clinically meaningful impact on WR, and there is limited evidence for pharmacotherapy.

Summary Future studies should be aimed at better defining WR to begin to understand the etiologies. Additionally, there is a need for non-surgical interventions with demonstrated efficacy in rigorous randomized controlled trials for the prevention and reversal of WR after bariatric surgery.

Keywords Obesity · Bariatric surgery · Weight regain · Behavior therapy · Antiobesity drugs

Introduction

During 2017–2018, 42% of adults in the USA had obesity (body mass index, BMI ≥ 30 kg/m^2) and 9% had severe obesity (BMI ≥ 40 kg/m^2) [1] with these prevalence rates projected to rise to 49% and 24%, respectively, by 2030 [2]. Obesity is associated with numerous chronic diseases including type 2 diabetes (T2D), hypertension, dyslipidemia, cardiovascular disease, nonalcoholic fatty liver disease, obstructive sleep apnea [3], and increased risk of hospitalization for COVID-19 with poorer outcomes [4–6]. Weight loss of 5–10%, if sustained long-term, can lead to improvement of obesity-related comorbidities [3, 7]. Whereas lifestyle interventions are recommended, they are hard to implement in primary care practices, and have limited long-term efficacy, primarily due to poor adherence [8–11]. Currently approved antiobesity drugs have yielded an average placebo-subtracted weight loss of 3–11% at 1 year in phase 3 clinical trials with daily oral phentermine/topiramate or weekly subcutaneous semaglutide demonstrating the most efficacy [12, 13].

Currently, bariatric surgery is the most effective treatment for obesity with clinically significant long-term weight loss [14–16] along with amelioration or resolution of obesity-related comorbidities including T2D [17–20], hypertension [21, 22], and dyslipidemia [23]. There is evidence from observational studies that bariatric surgery is associated with a reduction in major adverse cardiovascular events [24–27] and mortality [28–31]. The increased popularity of bariatric surgery is evidenced by the fact that > 600,000 surgeries are performed annually worldwide and > 256,000 in the USA alone in 2019, with sleeve gastrectomy (SG; 59%) and

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Roux-en-Y gastric bypass (RYGB; 18%) being the most common procedures [32].

While bariatric surgery is generally superior to non-surgical weight loss interventions, a significant proportion of patients achieve less-than-expected benefit due to suboptimal weight loss (SWL) or weight regain (WR). Sub-optimal weight loss, defined as not achieving a weight loss of 40% to 60% of baseline excess body weight (EWL) over 1–2 years, occurs in about 11% to 22% of patients following bariatric surgery [33–38]. Conversely, weight regain, defined as initially achieving expected weight loss after surgery but regaining weight, has a much higher prevalence in the bariatric population and as such will be the focus of this review.

Weight Regain (WR) After Bariatric Surgery

Although the prevalence rates for WR vary depending on the weight parameters defining as "regain" [39], it is now well established that a large proportion of patients experience significant WR during long-term follow-up [33, 40–53]. In a study of 300 RYGB patients, 37% had significant weight regain at 7-year follow-up using the definition of ≥ 25% increase from nadir weight [50]. A systematic review revealed that up to 76% of SG patients had significant weight regain at 6-year follow-up [51]. In the largest prospective cohort study of 1406 RYGB patients, the average weight regain, as a percentage of nadir weight (lowest post-operative weight), was 5.7% at 1 year after reaching the nadir weight, increasing to 10.1% after 2 years, 12.9% after 3 years, 14.2% after 4 years, and 15% after 5 years, thus revealing that the largest change in weight regain occurs 2 years after reaching nadir weight, but continues for increase out to 5 years post-op [53••]. In the same study, the incidence of ≥10% weight regain was 23%, 51%, 64%, 69%, and 72% after 1 to 5 years respectively.

Causes of Weight Regain

While procedural failures such as slippage of the gastric band, gastro-gastric fistulas, dilated gastric fundus, and enlargement of the gastric pouch or gastro-jejunal stoma can result in weight regain [48, 51, 54–56], the most common causes are thought to be dysregulated (e.g., loss-of-control eating) or maladaptive (e.g., grazing) eating, noncompliance to dietary recommendations, return to previous eating habits, sedentary lifestyle, and physiological compensatory mechanisms such as changes in hormones that regulate energy intake leading to increased appetite and food cravings, and increased caloric intake [51, 57–69]. Comorbid psychiatric disorders, especially history of depression, have also been implicated as potential causes of treatment failure [70, 71].

Three maladaptive eating behaviors—grazing, loss-of-control eating, and binge eating—have been commonly reported among bariatric surgery patients. Grazing is described as uncontrolled, unplanned, repetitive eating of small amounts of food between mealtimes. In one study, more than of half of the patients who reported binge eating before surgery shifted to grazing behavior after surgery [60]. The prevalence of grazing behavior among post-bariatric surgery patients ranges from 17 to 47%, depending on the method of assessment, including structured interviews and/or validated questionnaires, and time after surgery [72, 73], and has been correlated with weight regain in several studies [63, 74–76]. Loss-of-control eating (LOCE) is a subjective perception of being compelled to eat, or unable to resist or stop eating, that leads to eating when not intended and/or difficulty stopping. It is often related to subjective distress [77] and has been associated with poorer outcomes including weight regain among post-bariatric surgery patients [78–84]. There is evidence of the increased prevalence of binge eating disorder (BED) among patients seeking bariatric surgery [80, 85, 86] which persists post-surgery and is associated with WR. However, because of their physical inability to rapidly eat large quantities of food in a short period of time [87, 88], post-bariatric surgery patients are unlikely to meet the binge eating threshold set by the Diagnostic and Statistical Manual of Mental Disorder, 5th edition (DSM-5). Nevertheless, an argument could be made that eating an amount of food that is unusually large for post-bariatric surgery patients might constitute a binge episode that is relevant to this population [89].

Predictors of Weight Regain

While several pre-operative patient characteristics have been found to be predictive of SWL after bariatric surgery [35, 37], there is a relative paucity for WR, with one study reporting that African American patients had greater post-operative weight regain than White or Hispanic patients [90]. Post-operative factors associated with weight regain include larger gastrojejunal stoma diameter, larger gastric volume following SG, longer post-operative follow-up, presence of diabetes, binge eating, LOCE, increased food urges, excessive nocturnal eating, lower physical activity, lower social support, life stresses, problematic alcohol use, and depressive symptoms [60, 72, 78–80, 83, 91–95]. Additionally, weight regain was associated with higher pre-prandial ghrelin and lower post-prandial GLP-1 levels although the data are limited [67, 68, 92].

Clinical Consequences of Weight Regain

Weight regain leads to the recurrence of obesity-related comorbidities including T2D, hypertension, and dyslipidemia, increases health care costs, and has a negative effect on the quality of life and emotional health [41, 96–101].

Therefore, it is imperative that adjunctive therapies with proven efficacy are available for optimal management of weight regain and to maximize the long-term benefits of surgery.

Interventions for Prevention of Weight Regain or to Improve Overall Outcomes

Barring two small ($N = 18–30$) studies [102, 103], nutritional, cognitive-behavioral, supportive, and other psychological and lifestyle interventions started at the time of bariatric surgery or up to 2 years post-operatively, have not demonstrated a significant effect on overall weight loss [104–112]. In a study that enrolled subjects who had bariatric surgery between 3 months and 8.5 years, weight changes after 12 weeks did not differ between those who received high-volume exercise intervention vs control group (4.2 kg vs 4.7 kg; $P = 0.46$) [113]. Systematic reviews and meta-analyses have concluded that these interventions have a marginal or no effect on post-operative weight loss or maintenance [114, 115].

Interventions for Reversal of Weight Regain

Revisional Surgery

Certain anatomical causes of weight regain after Roux-en-Y gastric bypass (i.e., pouch/stoma dilation, or gastro-gastric fistula) sleeve gastrectomy (i.e., antrum or fundus dilation) or gastric band can be corrected with revisional surgery. Indeed, while the evidence is based on retrospective cohort analyses, reversal of weight gain, as percent excess body mass index loss (%EBMIL) after RYGB ranges from 43.3–63.7% at 1 year, and 14–76% at 3 years post-revision, depending on the procedure employed [116]. Further, conversion of SG to RYGB for WR results in a 40% excess body weight loss (%EBWL) at 12 months [117], albeit this is based on 2 studies. Finally, %EBWL after conversion of the gastric band to either RYGB or SG ranged from 23 to 74 %, with a mean follow-up between 7.3 and 44.4 months [118]. However, since most revisional surgical procedures carry a higher morbidity than the primary procedures [119, 120], non-surgical interventions should be tried first [48].

Behavior Therapy/Lifestyle Interventions

In 3 small trials ($N = 11$ to 28) which specifically enrolled patients with WR after bariatric surgery, behavioral interventions resulted in weight loss ranging from 1.6 to 5.1% over 6–10 weeks, but the absence of control groups limits the interpretation of efficacy [121–123]. The largest study ($n = 71$) to date of behavior therapy for post-surgery WR reported no meaningful benefit for the therapy group vs the wait-listed control group (average weight change: −0.8% vs 0.3%) after 10 weeks [124].

A retrospective chart-review study noted that 44 patients with post-surgery WR who participated in lifestyle intervention without pharmacotherapy lost an average of 2.1 kg after an average duration of 14.7 months [125]. In a non-randomized study of surgical revision procedures and pharmacotherapy for reversal of WR, the control group given diet/lifestyle counseling achieved no weight loss (75 ± 15 kg baseline vs 76 ± 14 kg) at 1 year [126].

Finally, a 5-month supervised high-intensity exercise program led to a small average weight loss of 1.2 kg; however, at 2 months after the intervention ended, the subjects had an average 1.1 kg weight regain [127].

In summary, dietary, behavioral and exercise interventions have not demonstrated efficacy in reversing WR after bariatric surgery.

Pharmacotherapy

As shown in Table 1, published studies to date of pharmacotherapy for reversal of weight regain after bariatric surgery consisted of 8 retrospective studies and 2 open-label trials. Some studies also included patients for whom pharmacotherapy was believed to have been prescribed for SWL or weight-loss plateau to promote additional weight loss. Not included in Table 1 are publications reporting (a) pharmacotherapy specifically targeting SWL, or initiated around the time of bariatric surgery to boost overall weight loss, (b) sub-group analyses using the previously published data, (c) those reporting ‘associations’ between weight change and anti-obesity drug prescriptions via electronic medical record search without determining whether the prescriptions were aimed at SWL, prevention or reversal of WR, or to improve overall weight loss, and (d) publications not reporting the change of weight with treatment. Topiramate (TPM) and phentermine (PHEN) were the most prescribed drugs and there is limited evidence for the effectiveness of topiramate and liraglutide. As narrated in the ‘Comments’ section, these studies were limited by their retrospective data collection, lack of predetermined censoring of the beginning and end of data collection, lack of a priori hypotheses, lack of randomization, small sample sizes, lack of control group, unclear definitions of WR, insufficient or missing data, lack of safety data, improper interpretation of results, and generally poor quality of reporting.

Conclusions

Although bariatric surgery remains the most effective treatment for obesity leading to sustained weight loss and amelioration of most weight-related comorbidities, especially

Table 1 Published studies of pharmacotherapy for weight regain after bariatric surgery

Reference	Interventions	Design	*N*	Duration (months)	Weight loss	Comments
Jester et al. (1996) [128]	PHEN 15 mg FEN 20 mg PHEN/FEN 15/20	Prospective open-label	34	3	WL = 4.5 kg to 22.7 kg	Data collected in one private clinic. 20 of 34 completed 3 months. No information regarding how many were treated with each medication.
Pajecki et al. (2012) [129]	Liraglutide 1.2 mg–1.8 mg	Retrospective	15	2–7; avg 3	WL = 2 kg to 18 kg	40% experienced nausea. Included patients with SWL.
Schwartz et al. (2016) [130]	PHEN 37.5 mg PHEN/TPM 1200 kcal/d diet for all	Retrospective	65 (PHEN 52; PHEN/TPM 13)	3	PHEN group averaged 6.3kg WL.	Only 30 patients had assessment at 3 months (24 PHEN, 6 PHEN/TPM). Included patients reaching weight loss plateau. Very few treated with PHEN/TPM.
Stanford et al. (2017) [131]	PHEN TPM Zonisamide Metformin Bupropion Orlistat Sibutramine Liraglutide Exenatide Pramlintide Naltrexone Lorcaserin PHEN/TPM Canagliflozin BUP/Naltrexone	Retrospective EMR review	319 Prescribed at WR =249 Prescribed at weight plateau = 68	Variable, not reported	56% had ≥ 5% W Mean WL = 7.6%	TPM, PHEN, metformin, bupropion, and zonisamide were most frequently prescribed. TPM was reported as the only medication that was effective based on OR 1.9 (95% CI: 1.1, 3.2; $P = 0.02$) for achieving ≥ 10% weight loss. However, TPM was not effective for achieving ≥ 5% weight loss based on OR 1.03 (95% CI: 0.65, 1.64; $P = 0.90$). In fact, none of the medications were associated with a significant OR for achieving ≥ 5% weight loss. Unclear how the investigators determined whether the medications were prescribed at weight plateau or for reversal of WR, or for treating other conditions. For example, metformin, exenatide, pramlintide, liraglutide, and canagliflozin might have been prescribed for diabetes, topiramate for migraine, bupropion for depression, and naltrexone for alcohol or opiate use disorder. Weight loss was calculated as the difference between weight when medication was started and nadir weight at any timepoint during medication treatment. Ideally, weight change should be the difference between start and the end of treatment and not the difference between the start of treatment and maximum weight loss. This method of data extraction disregarded weight regain while the same medication was continued beyond the nadir weight loss, thus overestimating the treatment effect. No clear explanation of which medication the weight loss was credited to when subjects took several antiobesity medications on and off or had a new medication added. No information on dose or duration of antiobesity medication therapy.

Table 1 (continued)

Reference	Interventions	Design	N	Duration (months)	Weight loss	Comments
Nor Hanipah et al. (2018) [132]	PHEN PHEN/TPM Lorcaserin BUP/Naltrexone	Retrospective	209	12	Avg WL = 3.2% at 3 m, and 2.2% at 1 yr; 37% had 1 yr WL > 5%	PHEN (75%) and PHEN/TPM (12%) were the most frequently prescribed medications. Included patients with SWL.
Srivastava et al. (2018) [133]	Metformin PHEN TPM PHEN/TPM BUP/Naltrexone Lorcaserin Zonisamide GLP-1RAs	Retrospective	48	6	2.2% WL at 3 m; 4.2% WL at 6 m	High attrition rate of 37% at 3 months and 68% at 6 months. Greater weight loss in those taking 2 or more meds compared to only one med or no meds.
Rye et al. (2018) [134]	Liraglutide 3.0 mg	Retrospective	20	7	Median WL 7.1% at 16 wks and 9.7% at 28 wks	Small sample. Included patients with SWL.
Wharton et al. (2019) [125]	Liraglutide 3.0 mg	Retrospective	117	7.6 ± 7.1	Avg WL = 5.5%	Variable duration. 29% experienced nausea. Only 37% were taking liraglutide at 1 yr.
Horber et al. (2021) [126]	Liraglutide 3.0 mg (LG) Fobi Ring with pouch resizing (FP) Endosurgery (ES) Diet counseling (DC)	Prospective open-label	Total = 95 LG = 34 FP = 16 ES = 15 DC = 30	24	Ave WL Liraglutide = 12 kg FP = 17 kg DC = 0.0 kg	Single obesity clinic. Non-randomized. Self-selection bias. Treatments were paid by public health insurance. ES group had an average weight loss of 3 kg after 12 months at which time all patients demanded additional drug therapy. Hence, 24-month weight loss is not available for the ES group.
Gazda et al. (2021) [135]	Intensive Lifestyle Modification (ILM) Non-GLP-1RAs: orlistat, TPM, PHEN, PHEN/TPM, lisdexamfetamine, naltrexone, BUP/Naltrexone, lorcaserin GLP-1RAs: liraglutide, semaglutide, exenatide, dulaglutide, albiglutide, lixisenatide	Retrospective EMR review 6.1	Total = 207 3m = 201 6m = 112 9m = 67 12m = 53	3–12	Mean (SD) WL% At 3 m: ILM = 1.4 (4.1)% Non-GLP-1RAs = 2.2 (3.6)% GLP-1RAs = 4.5 (3.1)% $P < 0.001$ At 12 m: ILM = 2.4 (4.9)% Non-GLP-1RAs = 5.4 (15.8)% GLP-1RAs = 8.9 (7.2)% $P = 0.12$	Medications were added to ILM intervention. Unknown whether GLP-1RAs were prescribed for weight management or diabetes control. Total sample dropped from 207 at the start of treatment to 53 (10 each in ILM and non-GLP-1 groups, and 33 in GLP-1 group) at 12 m. Large variability in weight loss. The investigators did not perform individual chart reviews to determine reasons for treatment discontinuation which might have included change in health insurance, affordability, lack of efficacy, adverse events, change in treatment group, lost to follow-up, or censoring by cut-off date.

Table 1 (continued)

Reference	Interventions	Design	*N*	Duration (months)	Weight loss	Comments
Rubio et al. (2021) [136]	Liraglutide 3.0 mg	Prospective open-label	23	12	Mean (SD) WL% Daily dosing: 17.9 (6.1)% Alternate day dosing: 17.2 (5.0)%	36 non-diabetic patients with obesity who had bariatric surgery and regained weight were treated with daily liraglutide 3.0 mg s.c. During the first 12 weeks, 13 patients were excluded (lack of effectiveness = 8; voluntary withdrawal due to economic reasons = 5). Of the 23 patients who completed 6 months, 11 were switched to liraglutide 3.0 mg alternate day dosing and 12 continued daily liraglutide 3.0 mg. The average weight loss of 17.9% with daily liraglutide 3.0 mg was unusually large (twice as much as the average weight loss observed with daily liraglutide 3.0 mg in RCTs in patients with obesity who never had surgery). Exclusion of patients for lack of effectiveness during the first weeks, small sample size, open-label nature of treatment, and lack of adequate description of the ancillary treatments the patients received are notable limitations.

EMR electronic medical record, *BUP* bupropion, *FEN* fenfluramine, *GLP-1RA* glucagon-like peptide-1 receptor agonist, *PHEN* phentermine, *PHEN/FEN* phentermine+fenfluramine, *PHEN/TPM* phentermine+topiramate, *SWL* suboptimal weight loss, *TPM* topiramate, *WL* total weight loss

T2D, it is now well recognized that a large proportion of patients experience significant WR during long-term follow-up. The prevalence rates of WR vary widely depending on the definition and the time since surgery. While WR could be attributed to anatomic and surgical causes in a small percentage of cases, the major causes of WR seem to be postoperative increased caloric intake due to increased appetite and maladaptive or dysregulated eating, inadequate physical activity, and psychosocial stresses. Unfortunately, WR is associated with the recurrence of previously controlled T2D, hypertension, and other weight-related comorbidities, with lowered quality of life and emotional health. Prevention of WR would be ideal, but unfortunately, the behavioral and lifestyle interventions aimed at prevention have not demonstrated efficacy. For reversal of WR, revisional surgery can be effective in some cases but is generally associated with a higher rate of complications than primary bariatric surgery. Dietary, behavioral, and exercise interventions have demonstrated null or marginal efficacy in reversing WR. Published studies of pharmacotherapy for WR have been mostly retrospective reviews of medical records or small open-label trials, and it appears that antiobesity drugs induce less weight loss in patients with a history of bariatric surgery than those without. There is a dire need for demonstration of efficacy in RCTs for cost-effective pharmacotherapy combined with lifestyle modification for the management of weight regain after bariatric surgery.

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Declarations

Conflict of Interest Kishore M. Gadde reports grants to his institution from AstraZeneca, BioKier, Indiana University Foundation, and National Institutes of Health, outside the submitted work. Ninh T Nguyen receives honorarium as a speaker from Olympus and Endogastric Solutions.

Human and Animal Rights This is a review article which reports previously published studies with human subjects. No animal experiments are reported in this article.

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