REVIEW

Alcohol and Other Addictive Disorders Following Bariatric Surgery: Prevalence, Risk Factors and Possible Etiologies

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Abstract

Bariatric surgery is currently the most effective intervention for significant and sustained weight loss in obese individuals. While patients often realize numerous improvements in obesity-related comorbidities and health-related quality of life, a small minority of patients have less optimal outcomes following bariatric surgery. The literature on the emergence of alcohol use disorders (AUDs) following bariatric surgery has grown in the past several years and collectively provides convincing evidence that a significant minority of patients develop new-onset AUDs following bariatric surgery. Rouxen-Y gastric bypass (RYGB) has generally been associated with the risk of developing an AUD, while laparoscopic adjustable gastric banding generally has not, in several large studies. One theory that has been discussed at some length is the idea of 'addiction transfer' wherein patients substitute one 'addiction' (food) for a new 'addiction' (alcohol) following surgery. Animal work suggests a neurobiological basis for increased alcohol reward following RYGB. In addition, several pharmacokinetic studies have shown rapid and dramatically increased peak alcohol concentrations following RYGB. The prevalence of alcohol and other addictive disorders and potential etiological contributors to post-operative AUDs will be explored.

Keywords
alcohol; bariatric surgery

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Prevalence of alcohol use disorders following bariatric surgery

A growing number of studies have provided information regarding the frequency of alcohol consumption and possible changes in the effects of consuming alcohol following bariatric surgery. Buffington (2007), for example reporting findings from a Web-based questionnaire, found that of the 318 patients who completed their survey, 83% said they consumed alcohol 'regularly' or 'upon occasion'. Of those patients who reported drinking one or more alcoholic drinks per week, 84% stated they were far more sensitive to the effects of alcohol than they were before surgery. Additionally, nearly half (45%) reported that they could 'feel the effects of alcohol after having only a few sips of alcohol'. Finally, 52% reported that the effects of alcohol lasted for a shorter period of time after surgery compared with before surgery. Buffington suggests that this could be due to changes in the rate of alcohol clearance following surgery. Another possible explanation is that following Roux-en-Y gastric bypass (RYGB), patients may not accurately perceive their level of intoxication. Significant changes in the pharmacokinetic profile of alcohol following RYGB lead to very rapidly attained maximum blood alcohol concentrations (BAC) that are disproportionately high in relation to the dose of alcohol consumed. After patients experience this very rapid and high maximum BAC, the more modest BAC levels present during the alcohol elimination process may feel lower by comparison than they actually are.

Ertelt et al. (2008) also surveyed postbariatric surgery patients to investigate self-reported alcohol abuse and dependence before and after bariatric surgery. Of the 70 respondents, 83% consumed alcohol after surgery, and of those, 84% reported increased sensitivity to the effects of alcohol after surgery. In contrast to the study by Buffington (2007), a significant percentage of patients in this study (29%) indicated that they felt the intoxicating effects of alcohol for a longer time after surgery than before. Six (8.6%) individuals were identified who met self-reported criteria for a diagnosis of alcohol dependence, and one participant (1.4%) met criteria for alcohol abuse after surgery. In this sample, only two patients reported developing alcohol dependence after surgery, after not reporting such a problem prior to surgery. Additionally, two patients reported that they did not drink alcohol prior to surgery but did engage in alcohol consumption after

surgery. Two other patients reported that alcohol consumption had increased after surgery. However, approximately two-thirds of the sample (67.1%) reported no change in their alcohol consumption, and 16 participants (22.9%) reported a decrease in alcohol use after bariatric surgery.

Suzuki et al. (2010) surveyed 530 postbariatric surgery patients about their alcohol consumption. From these 530 patients, 51 agreed to be interviewed and assessed more thoroughly. Two key findings from this study were that (1) individuals with a lifetime history of an alcohol use disorder (AUD) were more likely to have an AUD after surgery (compared with those who did not have such a history) and (2) patients who underwent RYGB were more likely to have an AUD after surgery than those who received a laparoscopic adjustable gastric banding (LAGB).

Saules et al. (2010) examined 7199 patients in a substance abuse treatment facility, 54 of whom had undergone bariatric surgery. Information regarding alcohol and drug use and abuse was collected for the group of postsurgical participants by matching them with control subjects in the facility (who had not undergone any form of bypass surgery). All alcohol intake information was collected as a range (e.g. 'a pint to a fifth of vodka per day'). According to this study, there were no differences between the surgery and nonsurgery groups at the time of programme entry for rates of alcohol or substance abuse, but there was a difference for alcohol withdrawal symptoms—members of the postsurgery group were more likely to suffer alcohol withdrawal. Of the 54 surgery participants, 83.3% currently drank alcohol and did report drinking a significantly larger minimum number of drinks per drinking day (13.1 ± 9.9 for surgery group vs 9.3 ± 6.7 for controls). Sixty-two percent of the surgery group came to the facility specifically for alcohol treatment, while 9.4% came for alcohol and drug abuse, 13.2% for opiates, 7.5% for benzodiazepines, 5.7% for polysubstance abuse and 1 participant for amphetamines. Compared with their matched control group, the postsurgery group was more likely to be seeking treatment for alcohol abuse, coincident alcohol and drug abuse, or benzodiazepine use. Approximately 36% of the postsurgery group reported heavy use of alcohol or drugs presurgery, 43.4% reported initiating heavy substance use only after surgery and 20.8% reported heavy use of alcohol and/or drugs presurgery and commenced heavy use of another drug and/or alcohol postsurgery.

Heinberg and Ashton (2010) reported that patients with a history of substance abuse or dependence experienced a greater per cent excess weight loss at 6 and 9 months following bariatric surgery compared with patients with no history of abuse/dependence prior to surgery. The reason for these findings is unclear, although the authors hypothesized that the group with a substance abuse/dependence history received more intense treatment and monitoring around the time of surgery, which may have led to improved outcomes. They further suggest that patients with a history of a substance use disorder in remission have already successfully changed their substance use behaviour and therefore may be better equipped to make the lifestyle changes necessary to be successful after bariatric surgery.

Welch et al. (2011) assessed 75 RYGB patients 2 years after bariatric surgery using the CAGE questionnaire (Ewing, 1984) where CAGE is an acronym used as a reminder of the four questions on the screening tool. Although not the primary focus of the study, the authors provided data on self-reported levels of alcohol abuse in this sample. Of the 75 participants who completed follow-up assessments, only 1.3% responded in a manner suggesting that they abused alcohol.

Woodard, Downey, Hernandez-Boussard and Morton, 2011 conducted a longitudinal study that assessed 19 RYGB patients at three time points: presurgery, 3 months postsurgery and 6 months postsurgery. Of these patients, 74% drank alcohol before RYGB, and that number decreased post-operatively to 44% at both 3 and 6 month postsurgery assessment periods. All patients who drank alcohol after surgery had also reported consuming alcohol in the presurgery assessment. Patients who drank alcohol after surgery reported a decline in the number of drinking events at 6 months postsurgery; these participants declined from drinking on average of 1.9 days per week to drinking on average 0.9 days per week. Additionally, patients drank less alcohol in one sitting postsurgery; the average number of drinks declined from 2.4 drinks to 1.5 drinks at 6 months postsurgery. Total drinks per week also declined, from 4.4 presurgery to 1.8 drinks at the final assessment.

Rates of AUDs among a large sample of bariatric surgery patients enrolled in the Longitudinal Assessment Bariatric Surgery 2 (LABS-2) study have been reported (King et al., 2012). Among 1945 participants, the prevalence of AUDs as assessed by the Alcohol Use Disorders Identification Test (AUDIT) did not differ between presurgery (7.6%) and 1 year postsurgery (7.3%). At year 2, however, there was a significant increase in the number of AUDs noted (9.6%). Variables independently associated with an increased odds of a reported AUD post-operatively included male sex, younger age, regular alcohol consumption prior to surgery, prior AUD, recreational drug use prior to surgery and lower interpersonal support pre-operatively. Consistent with other research, undergoing RYGB was independently associated with an increased risk of a post-operative AUD relative to undergoing adjustable LAGB. Of participants who reported a pre-operative AUD, 62.3% had continued or developed recurrent AUD symptoms following surgery. Interestingly, 7.9% of participants who did not report an AUD at the pre-operative assessment had a post-operative AUD. This underscores other literature that suggests *de novo* alcohol use problems may develop following bariatric surgery.

Another large cohort, the Swedish Obese Subjects study (SOS), was examined for long-term rates of alcohol problems after bariatric surgery (Svensson et al., 2013). Unlike the LABS-2 cohort, in which approximately 70% of patients underwent RYGB, a minority of the 2010 patients in the SOS study underwent RYGB (13%), while the majority underwent vertical banded gastroplasty (68%) or gastric banding (19%). The follow-up period of participants was up to 22 years (median 15 years, range 8–22 years). Information about alcohol consumption was assessed from a validated SOS dietary questionnaire, which was used to collect data regarding habitual food and beverage consumption over the prior 3 months. From participant responses, total dose in grams of alcohol consumed per day was calculated. Medium-risk alcohol consumption was classified as >40 g (approximately three standard drinks) and >20 g (approximately 1.5 standard drinks) of alcohol per day for men and women, respectively. Participants were also asked whether they perceived that they had an alcohol

problem. Despite the lower prevalence of RYGB in the SOS study, the findings concur with the results from the LABS-2 cohort. Participants who underwent RYGB in the SOS study had an increased risk of an alcohol abuse diagnoses (adjusted hazard ratio = 4.97) versus controls, as well as an increased risk of having at least World Health Organization-defined medium risk (adjusted hazard ratio = 2.69), and an elevated risk of self-reported perception of having an alcohol problem (adjusted hazard ratio = 5.91). Vertical banded gastroplasty increased the risk of these conditions to a lesser degree, and LAGB did not increase risk relative to controls. Additional predictors of an alcohol abuse diagnosis were male sex, baseline smoking and baseline alcohol consumption, similar to predictors identified in the LABS-2 cohort.

Conason et al. (2013) administered the Compulsive Behaviors Questionnaire to assess substance use before and at 1, 3, 6, 12 and 24 months following bariatric surgery among a sample of 100 participants who underwent RYGB and 55 participants who underwent LAGB. A composite substance use score, which represented drug use, alcohol use and cigarette smoking, was increased significantly by 24 months postsurgery. The frequency of alcohol intake was significantly increased at 24 months among patients who underwent RYGB, whereas alcohol use frequency did not increase over time among patients who underwent LAGB. The frequency of cigarette use and drug use did not change significantly over time following surgery. However, the response rate declined over time following surgery and was only 24% at the 24-month follow-up in this study.

Östlund et al. (2013) presented data on alcohol use from a database of 11, 115 patients who had undergone bariatric surgery. The rates of hospital admissions for alcohol-related problems were examined in patients who underwent RYGB ($n = 4161$) or a purely restrictive procedure ($n = 6954$; either vertical banded gastroplasty or gastric banding) in Sweden between 1980 and 2006. The mean follow-up times were 3.8 (±4.5) years and 11.5 (±6) years for the gastric bypass and restrictive groups, respectively. The risk of hospitalization for alcohol abuse following RYGB was increased twofold versus those who underwent a purely restrictive procedure (hazard ratio = 2.3).

A survey of alcohol use following RYGB was performed by Lent et al. (2013). Of the 899 patients surveyed pre-operatively, 155 returned a post-operative survey with at least one question filled out concerning recent alcohol use and smoking status. Participants were surveyed with an alcohol questionnaire created by the investigators, administered on average, 34.9 ± 12.8 months following RYGB. Alcohol use decreased significantly from presurgery (72.3%) to postsurgery (63.2%). As the amount of alcohol consumed pre-operatively increased, the odds of drinking alcohol post-operatively increased by sixfold. Higher body mass index (BMI) increased the odds of high alcohol consumption, and older age decreased odds of both alcohol consumption and smoking. Neither alcohol use nor smoking was associated with the amount of weight loss achieved following surgery. Smoking status did not change from presurgery to postsurgery. The findings in this study are somewhat discrepant from other recent prospective longitudinal studies of alcohol use in RYGB patients. The reason for the differences among studies is unclear but may be a result of the different assessment tools for alcohol use in this study compared with others. The questionnaire used in this study was not validated, and the amount of alcohol consumed (frequency of drinking and total drinks/week) was compared between presurgery and postsurgery. Following surgery, however, patients reported experiencing greater intoxication and increased problems associated with a reduced total volume of alcohol ingested.

Wee et al. (2014) performed an interview-based study of patients before ($n = 541$) and 1 ($n = 375$) and 2 ($n = 328$) years following RYGB or LAGB surgery. The investigators used the AUDIT-Consumption. Prior to surgery, 17% of the patients in this sample reported high-risk drinking, whereas 1 year after surgery, this number decreased to 13% ($p = 0.10$). Among the sample interviewed prior to surgery and at 2 years following surgery, the percentage of patients who reported high-risk drinking decreased slightly from 15% to 13% ($p = 0.39$). In agreement with prior literature, this study showed that 6–7% of patients reported new high-risk drinking following surgery. In this study, the rate of new-onset high-risk alcohol consumption did not differ by surgical procedure. Unlike several prior studies, Wee et al. also evaluated the percentage of patients who no longer experienced high-risk drinking after surgery and found that approximately half of patients with high-risk drinking presurgery stopped this behaviour following surgery. As the authors point out, the rate of improvement in high-risk drinking postsurgery is rarely examined, even though surgery may have beneficial effects on alcohol consumption for a subgroup of patients.

The Structured Clinical Interview for DSM-IV Axis I (SCID-1), along with the AUDIT, was used to assess alcohol and other addictive disorders following weight loss surgery among a subgroup ($n = 201$) of participants from the LABS-2 cohort (Mitchell et al., 2015). Using SCID criteria, 16 participants (8%) developed an AUD within 3 years following RYGB. Of these, 43.8% did not have a pre-operative history of an AUD. Overall, 3.5% of participants developed a new-onset AUD postsurgery using SCID criteria only. Using AUDIT scores as well to examine problematic alcohol use, 7.5% of the sample had an AUD post-operatively who did not have an AUD before surgery. While these data again support that a similar percentage of patients develop an AUD following bariatric surgery (~7%), these data also suggest that the method of assessment has a significant influence on this percentage. Given that the rates of new-onset post-operative AUDs were much lower using the SCID (3.5%), it is worth considering whether the SCID-1 criteria for DSM-IV were overly conservative. As discussed by Mitchell et al. (2015), the version of the SCID used in this study required consumption of at least five drinks on one occasion to meet initial screening criteria, which may not be feasible given anatomical and pharmacokinetic changes post-operatively, and may not adequately capture all AUDs in post-operative patients. Conversely, the AUDIT may lead to overdiagnosis as it is meant to serve as a screening tool prior to further patient evaluation.

Rates of alcohol abuse before and after bariatric surgery were also recently examined by self-report in the Portuguese population (de Araujo Burgos et al., 2015). The authors found that among 659 patients who underwent RYGB (32.6%) or LAGB (67.4%), of whom 42% completed 2-year follow-up, the frequency of alcohol use was 24.2% in the pre-operative period and 9.4% in the post-operative period. Daily alcohol intake was similar before and after surgery. There were no associations

between alcohol use and surgical type. In this study, there was no increase in the frequency of alcohol intake observed postsurgery. This study relied on a series of self-report questions that were created for this study.

Cuellar-Barboza et al. (2014) performed a retrospective study of electronic medical records of patients treated at the Mayo Clinic Addiction Treatment Program between 2004 and 2012. Of the 823 patient charts examined, 4.9% had a history of undergoing RYGB. Patients who had undergone RYGB reported resuming alcohol consumption approximately 1.4 ± 0.2 years postsurgery, meeting AUD criteria 3.1 ± 0.5 years following surgery, and seeking treatment 5.4 ± 0.3 years post-operatively.

Taken together, most, though not all, research suggests that there is a small but significant minority of patients who undergo bariatric surgery develop new-onset AUDs following surgery. Of importance, literature suggests that AUDs may also improve in a subgroup of patients following surgery (Wee et al., 2014). Of the surgical types, RYGB has been associated with greater risk of developing an AUD compared with LAGB in multiple studies (King et al., 2012; Suzuki, Haimovici & Chang, 2012; Conason et al., 2013; Östlund et al., 2013; Svensson et al., 2013). The method of alcohol assessment is highly likely to influence the reported rates of AUDs (Mitchell et al., 2015) and may account for a large portion of the variability in findings noted across studies. Limited data are available on long-term outcomes with respect to alcohol use following RYGB, with most studies only providing data for 2–3 years postsurgery. The SOS study provides long-term data on alcohol use suggesting continued increase in rates over several years; however, only a minority of patients in this study underwent RYGB. Patterns of alcohol use over the long-term will be important to examine. In addition, etiological contributors and predictors of post-operative AUDs are important considerations, and a limited amount of research has been conducted to address these issues.

Animal models of bariatric surgery and alcohol

Although the consensus is generally positive regarding bariatric surgery as a means to treat severe obesity and its associated comorbidities, the continued concerns of increased alcohol intake following the procedure have been examined in several studies using animal models. In order to address the question of whether RYGB increases preference towards alcohol intake, Hajnal et al. (2012) examined whether RYGB increased self-administration of alcohol in dietary obese rats. Comparing the results of the RYGB rats with their sham counterparts, the study revealed that RYGB rats significantly engaged in behaviours to receive greater amounts of ethanol. Interestingly, when RYGB rats were treated with a predose of the ghrelin-1a-receptor antagonist, D-[Lys3]-GHRP-6, researchers found decreases in alcohol intake following the procedure. These findings suggest that augmentation of the ghrelin system may impact the rewarding effects of alcohol following RYBG. Davis et al. (2012) found that in a rodent model of ethanol-preferring rats, the RYGB procedure decreased ethanol consumption, along with a decrease in the effectiveness of the reinforcing properties of ethanol. This change in behaviour was associated with ethanol-induced increases in plasma levels of the gut hormone glucagon-like peptide-1 (GLP-1). When a GLP-1 agonist was administered to a sham counterpart, decreased ethanol intake was found. Interestingly, when ghrelin was replaced in the RYGB sample, the rats showed a restored desire for ethanol consumption and drinking behaviour. These findings, which are dissimilar to the findings of Hajnal et al. (2012), would suggest that RYGB actually attenuates alcohol intake by reducing the reinforcing properties of ethanol and by the changes in the levels of gut hormones GLP-1 and ghrelin.

Although alterations of the ghrelin system have been considered by some to be the primary reason for the increase in alcohol intake following RYGB, new studies from Davis et al. (2013) may suggest otherwise. Using a rodent model of alcohol nonpreferring rats, these researchers examined the postsurgical effects of RYGB on ethanol consumption. Controlling for presurgical weight and dietary consumption, the study shows that RYGB directly increased alcohol intake independent of ethanol metabolism or postsurgical shifts in release of ghrelin. The researchers suggest that this observation may be due to altered genetic expression in the regions of the brain associated with the reward circuit following RYGB surgery. Consequently, the mechanism for increased alcohol consumption following RYGB needs further investigation.

Although it has been less of a focus in the literature, there is clearly a subgroup of patients who experience problematic alcohol use prior to bariatric surgery and then report a reduction in use following surgery. An important point was made in a commentary by Sogg and Stoeckel (2014). The authors comment on the variation in alcohol use trajectories after bariatric surgery and suggest that presurgical alcohol use may affect the brain and behaviour in differing ways. They discuss the idea that neurobiology may differ between patients who have a high-risk alcohol profile prior to surgery and those who do not, and that weight loss surgery may interact in different ways to produce varying patterns of alcohol use following surgery. More research is needed to explore factors that are associated with improvement or worsening of alcohol use following bariatric surgery.

Addiction transfer (cross-addiction) model

The popular media has devoted a considerable amount of attention to postbariatric surgery patients developing 'addictive behaviours', such as alcohol abuse or dependence. Some of the sources of this information have included the *Wall Street Journal* (Spencer, 2006), *People Magazine* (Souter, Shapiro & Sheff-Cahan, 2007) and the Oprah Winfrey Show (The Oprah Winfrey Show, 2006). Their common message is that when faced with an inability to engage in a past coping behaviour (e.g. eating large amounts of food), postbariatric surgery patients frequently adopt new inappropriate coping behaviours (e.g. alcohol misuse) to help deal with negative emotions. This phenomenon has been commonly referred to in the literature as 'cross addiction' and 'addiction transfer'. Data collected from a study conducted with obese, binge eating patients show that participants' negative mood appears to rise precipitously in the time immediately preceding a binge eating episode (Engel et al., 2008). Following the binge

eating episode, participants' negative mood improved, suggesting a negative reinforcement model and further suggesting that the function of the binge eating episode is to help ameliorate negative emotional states (Engel et al., 2008). These data come from the participants' natural environment also implying that this relationship between negative emotional state and eating behaviour occurs outside of the laboratory environment.

The addiction transfer model is functionally similar to the relationship described in the preceding text, with the key differences being that the group of interest is a postbariatric group (rather than nonbariatric surgery obese individuals) and that the key behaviour of interest (binge eating) has been replaced with a different behaviour (alcohol consumption), because of the patients' inability to eat large amounts of food. This lack of ability to eat a large volume of food in one sitting therefore reduces the patients' ability to regulate their emotions through this mechanism and theoretically leads them to find a new behaviour that can serve this purpose. This model has not been adequately developed or empirically validated and should be explored in future research. Mechanisms through which the addiction transfer model may operate (e.g. psychological and neurobiological) need to be studied.

Neurobiology and alcohol

Neurobiological contributors may play a role in the development or maintenance of both alcohol abuse and obesity. Food, alcohol, drugs of abuse and various other positively reinforcing substances lead to dopamine release in the nucleus accumbens (Martel & Fantino, 1996; Koob & Le Moal, 1997). Dopamine has been termed the 'pleasure neurotransmitter' (Nash, 1997). Neuroimaging studies have shown that dopamine release occurs during binge eating (Wang et al., 2011) and also during alcohol consumption (Aalto et al., 2015). Theoretically, changes in the ability to stimulate this neurotransmitter system postsurgically through palatable foods could provide a neurobiological basis for the addiction transfer hypothesis. That is, when faced with an inability to attain reward through overeating postgastric bypass, other dopamine-releasing stimuli, such as alcohol, may be substituted in an attempt to achieve reward. As research seeks to examine the validity of the addiction transfer model, neurobiological factors, including dopamine and other neuro-hormonal moderators of mood and reward, should also be investigated. Taylor, Curtis and Davis (2010) propose that as in the traditional view of addiction, both overeating and substance abuse activate the mesolimbic reward system, specifically the caudate nucleus, the hippocampus and the insula. This results in a release of striatal dopamine, which plays an important part in neural circuits involved in reward. The authors also suggest that predisposition to addictions, including food addiction, is a result of downregulation of dopamine receptors, which would cause individuals with decreased dopamine receptor availability, who are more sensitive to stimuli, to overeat as a means to produce pleasure or reduce pain. Southon et al. (2003) found that the D2 dopamine receptors in the central nervous system contribute to the regulation of food consumption.

Results from imaging studies provide additional data to support the relationship between obesity and changes in the dopaminergic systems. For example, Wang, Volkow and Fowler (2002) reported that overeating in obese individuals presents in a fashion similar to that of compulsive drug-taking behaviour observed in subjects with substance abuse. Using Positron Emission Tomography (PET) scans, Wang et al. observed that obese subjects displayed reduced levels of dopamine D2 receptor (DA D2) availability. Also, DA D2 receptor levels were found to be inversely related to the BMI of the participants. Wang et al. suggested that these dopaminergic deficits may create a need to overeat to compensate for decreased activation of these reward circuits (2001).

Recently, neuroimaging data have described changes in DA D2 receptors following weight loss surgery. Using a prospective design and using Magnetic Resonance Imaging (MRI) and PET, Dunn et al. (2010) assessed five patients pre-operatively and again 6–11 weeks following RYGB ($n = 4$) or vertical sleeve gastrectomy ($n = 1$). The results revealed decreased DA D2 receptor availability after surgery in several regions that may affect eating behaviour. This suggests enhanced dopaminergic neurotransmission, which the authors interpreted as contributing to reduced hunger and an improved sense of satiety following surgery. These data may suggest that obese individuals consume excess food in an effort to obtain food-related stimulation and reinforcement, which is minimized because of their dopaminergic deficits.

However, Steele et al. (2010) argue against the idea of 'food addiction' wherein a deficiency of dopamine receptors leads individuals to engage in compensatory behaviour such as overeating. These investigators also used PET to examine dopamine receptor availability in the brains of five females who had undergone bariatric surgery and found a significant relationship between the amount of weight lost and dopamine receptor availability, which is generally consistent with previous work by Wang et al. (2002). At 6 weeks after RYGB, D2 receptor availability was increased as assessed by PET. Although these findings are generally consistent with previous studies (Wang et al., 2001), Steele et al. (2010) offer an alternative interpretation to these data, suggesting that the findings imply that improvements in appetitive behaviour are a result of increased available dopamine receptor binding. Overeating in the context of obesity appears to result in a reversible downregulation of D2 receptors. Thus, these findings suggest that dopamine binding increases in parallel with weight loss, which would imply that decreased dopamine receptors are a result of being obese, and not a cause contributing or risk factor for obesity.

Pharmacokinetics of alcohol following bariatric surgery

Research has shown heightened subjective sensitivity to alcohol following bariatric surgery (Buffington, 2007; Ertelt et al., 2008), and alcohol administration studies have generally shown altered pharmacokinetics of alcohol following RYGB, mixed results following sleeve gastrectomy and no change following gastric banding.

Following RYGB, the extant research suggests that the pharmacokinetic parameters of alcohol are altered (Klockhoff, Näslund & Jones, 2002; Hagedorn, Encarnacion, Brat & Morton, 2007; Maluenda et al., 2010; Woodard et al., 2011; Steffen, Engel, Pollert, Li & Mitchell, 2013). Klockhoff et al. (2002) performed a cross-sectional comparison of BAC between 12 post-RYGB patients and 12 controls matched on BMI and age. Following a single weight-based dose (0.30 g/kg) of 95% v/v alcohol, the time to maximum BAC (Tmax) was shorter in the RYGB group

(10 minutes vs 30 minutes), and the maximum BAC (Cmax) was higher in the RYGB group (0.74 vs 0.58 g/l). Also, the BAC was higher in the RYGB group at 10 and 20 minutes postdose relative to the control group, although the area-under-the plasma concentration time curve did not differ significantly between the surgery and control groups.

Hagedorn et al. (2007) found similar results in a cross-sectional comparison of 19 post-RYGB patients with 17 nonsurgical controls. Consumption of 5 oz of red wine produced a higher peak breath alcohol level in the RYGB group versus controls (0.08% vs 0.05%), as well as a longer time to reach a breath alcohol level of zero (108 vs 72 minutes). Similarly, Woodard et al. (2011) conducted a within-subject study, examining 19 postsurgical patients at 3- and 6-month follow-ups after their surgery. Participants in this study also consumed 5 oz of red wine. The results suggested that at 6-month follow-up, the percent breath alcohol concentration was significantly greater than at the other assessment points (presurgery = 0.024%, 3-month follow-up = 0.059%, 6-month follow-up = 0.088%). Woodard et al. also noted a significantly longer time to reach a zero breath alcohol level following surgery (presurgery = 49 minutes, 3-month follow-up = 61 minutes, 6-month follow-up = 88 minutes). Steffen et al. (2013) administered a weight-based dose of alcohol (0.3 g/kg) to five patients following RYGB and collected blood samples every minute for the first 5 minutes following ingestion of the drink and periodically thereafter through an indwelling intravenous catheter. The mean maximum BAC was 138.4 ± 28.6 mg/dl and the mean time to maximum BAC was 5.4 ± 3.1 minutes, suggesting a much earlier and higher peak that was not seen in earlier studies because of the onset of blood and/or breath sampling at 10 or 15 minutes.

There are significant differences between study approaches that likely contribute to the variability among results observed. Blood (Klockhoff et al., 2002; Steffen et al., 2013) versus breath alcohol concentration (Hagedorn et al., 2007; Woodard et al., 2011) determination is one major difference between the existing studies, and BAC is generally considered to be more accurate. Some studies have relied upon a fixed dose of alcohol with no adjustment for changes in body weight following surgery (Hagedorn et al., 2007; Woodard et al., 2011), whereas other studies have used a weight-based dose of alcohol (Klockhoff et al., 2002; Steffen et al., 2013). Comparison groups have also varied and include within-subject prospective, longitudinal designs (Woodard et al., 2011), cross-sectional designs with BMI-matched (Klockhoff et al., 2002) or BMI-unmatched (Hagedorn et al., 2007) nonsurgical controls and one study with no control group (Steffen et al., 2013). Despite the methodological variability in these reports, it is apparent that following surgery RYGB patients rapidly achieve peak BAC concentrations that are significantly higher than expected in matched nonsurgical or presurgical comparison groups. Studies that administered a fixed dose of red wine to all participants (Hagedorn et al., 2007; Woodard et al., 2011) also reported a longer time to eliminate alcohol following RYGB, although this finding is not consistent across the literature (Klockhoff et al., 2002) and may be due, at least in part, to the variability in gram-per-kilogram dose of alcohol administered when a fixed alcohol dose is given.

Pharmacokinetic data have been inconsistent following sleeve gastrectomy. Maluenda et al. (2010) performed a within-subject study of 12 morbidly obese patients in which participants consumed an individualized dose of alcohol based upon total body water (average of 187 ml of red wine prior to surgery and an average of 159 ml post sleeve gastrectomy). Following the consumption of the wine, the maximum BAC was significantly greater in the postsurgical assessment than in the presurgical assessment (4.0 vs 1.5 g/l, respectively), along with a significantly longer time to reach the elimination of breath alcohol to zero (204 vs 177.4 minutes). Changchein, Woodard, Hernandez-Boussard and Morton (2012) performed a prospective, longitudinal study and reported no difference before and after laparoscopic sleeve gastrectomy (LSG) surgery (3 and 6 months) in peak BAC or time to eliminate alcohol. This research group also evaluated the pharmacokinetics of alcohol in a group who underwent LAGB in which similar results were reported. Gallo et al. (2014) examined patients who underwent sleeve gastrectomy and demonstrated that peak BAC and alcohol elimination rate did not differ between the presurgical evaluation and the post-operative (3 and 12 months) assessment points. Similar to the data with RYGB, these data are difficult to extrapolate between studies as some used individualized dosing (Maluenda et al., 2010) whereas others used fixed alcohol doses (Changchien et al., 2012; Gallo et al., 2014).

Mechanistically, pharmacokinetic changes likely result from the dramatic anatomical and physiological changes that result from RYGB or sleeve gastrectomy. There is a reduction in available gastric surface area, and therefore a decrease in the availability of the metabolic enzyme gastric alcohol dehydrogenase is likely, which is normally responsible for a small but significant amount of presystemic alcohol metabolism (Meier & Seitz, 2008). The gastric emptying rate associated with liquids is also generally accelerated by RYGB (Dirksen et al., 2013) and sleeve gastrectomy (Melissas et al., 2013). This allows alcohol to reach the jejunum rapidly after ingestion where it is readily absorbed. There are also obvious changes in body weight that result from surgery, which alter the gram-per-kilogram dose of a fixed amount of alcohol given before and after surgery. Alcohol follows a non-linear pharmacokinetic profile where approximately 120 mg/kg/hour is metabolized regardless of the BAC present (Schuckit, 2011). Therefore, the higher the milligram-per-kilogram dose ingested, the longer it would be expected to take to return to baseline (BAC of 0 mg/dl). Whether the pharmacokinetic changes that follow RYGB account for some of the increased risk of AUDs following surgery is unclear. Theoretically, higher and more rapid BAC achievement may be more reinforcing to patients, and additional research is needed to explore this area. Animal work, however, has shown that rats that underwent RYGB worked harder to receive alcohol compared with sham operated rats even when the alcohol was delivered intravenously, suggesting that neurobiological mechanisms are involved in alcohol reward and it is not simply a pharmacokinetic phenomenon (Polston et al., 2013).

Other addictive disorders following bariatric surgery

The concept of behavioural addiction has been discussed in the literature for decades (Clark, 2014; Marazziti, Presta, Baroni, Silvestri & Dell'Osso, 2014), although there is ongoing discussion within the field with regard to the underlying neurobiology and categorization of these types of disorders. With the

publication of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), there came a shift in the classification of pathological gambling from an impulse control disorder listing to an addictive disorder listing. Several other previously identified impulse control disorders, including trichotillomania and skin-picking disorder (excoriation disorder), have also been reclassified, although these now fall into obsessive–compulsive and related disorders. DSM-5 also mentions other behavioural addictions such as 'sex addiction', 'exercise addition' and 'shopping addiction' but indicates that at this time, these still lack sufficient peer-reviewed evidence to be fully identified as psychiatric disorders. However, the conceptualization that there are repetitive behaviours that rise to the level of pathological addictions exists.

Considerable data have demonstrated that obesity is associated with an increase in impulsive behaviours and/or reduced impulse control (Chamberlain, Derbyshire, Leppink & Grant, 2015; Davis, 2010; Schmidt, Körber, de Zwaan & Müller, 2012). In addition, binge eating disorder, which is commonly present in the obese population, is defined in part by significant sense of loss of control over eating (First & Ward, 2013; Pollert et al., 2013). While it is generally agreed that obesity is not primarily a disease of impulse control or an addiction, there is ongoing discussion within the field in this regard (Dileone, Taylor & Picciotto, 2012; Volkow, Wang, Tomasi & Baler, 2013).

Several groups have studied various impulsive behaviours in obese populations, although little data exist regarding the prevalence of these behaviours in association with bariatric surgery. A study performed in 2012 by Schmidt et al. examined 100 morbidly obese patients who were candidates for bariatric surgery. This study demonstrates a relatively high current and lifetime prevalence rate of some impulse control disorders in these patients prior to surgery although no follow-up data after surgery were collected. Interestingly, the rates of impulse control disorders in these morbidly obese patients paralleled the rates of impulse control disorders seen in inpatient psychiatric patients, suggesting that obese patients have a tendency towards pathological levels of impulse control (Muller et al., 2011). In terms of specific impulse control disorders, skin picking and compulsive buying were found to be the most common with current prevalence rates of 8% and 6% and lifetime prevalence rates of 9% and 8%, respectively. Other disorders and current/lifetime prevalence rates found in this study were intermittent explosive disorder 5% and 10%, pathological gambling 1% and 3%, and pathological Internet use 2% and 5%. None of the participants endorsed pyromania, kleptomania, trichotillomania or compulsive sexual behaviour. Excessive exercising was not addressed in the study (Schmidt et al., 2012). While these data highlight the apparently substantial prevalence rates of impulsive disorders prior to surgery, the question whether there are any measurable changes in these behaviours postsurgery was not addressed.

Early data suggest that there is no change in impulsive behaviours following surgical intervention (Ryden et al., 2004). More recent data suggest that there might be some increase in certain behaviours, but there are no clear data to suggest that there is a significant problem with 'addiction transfer'. A 2014 study by Georgiadou, Gruner-Labitzke, Kohler, de Zwaan and Muller looking at both cognitive function and nonfood-related impulsivity does not demonstrate a difference between postbariatric surgery patients and matched controls regarding impulsive symptoms and behaviours. Furthermore, a study of 201 postbariatric surgery patients by Mitchell et al. (2015) demonstrates a 3% increase in the occurrence of any nondrug-related addictive behaviour following surgery relative to presurgery behaviours. In terms of specific behaviours, impulsive–compulsive buying and impulsive–compulsive Internet use had the largest percentage increases, although there were no matched controls, and therefore, interpretation of these findings is limited. In addition, not all addictive behaviours were assessed in this study. As previously mentioned, excoriation disorder or skin picking has one of the highest prevalence rates of all addictive behaviours in the obese population in general and was not included in this study.

In conclusion, while there are no definitive data at this time to demonstrate that there is an increase in behavioural addictions or impulse control disorders following bariatric surgery, there is sufficient anecdotal evidence and underlying theoretical reason to believe that an increase in addictive behaviours could occur. Certainly, more studies are needed at this time to further explore this phenomenon.

Conclusion

A significant minority of patients who undergo bariatric surgery develop post-operative AUDs. It is important to note, however, that the literature also suggests that alcohol-related problems may decrease in a subgroup of patients following bariatric surgery. Currently, the variability in study methodology prohibits us from drawing firm conclusions regarding prevalence rates of, and risk factors associated with, AUDs after bariatric surgery. The length of follow-up across studies is highly variable as is the method of assessment used to determine the presence of an AUD. Response rates at follow-up also vary across studies and diminish the ability to draw conclusions in some datasets. Pharmacokinetic studies have also produced varying results and have relied upon a wide range of methodological designs, dosing regimens and assessment approaches. One finding that is more clear is that RYGB patients experience more rapid absorption and increased maximum BACs following surgery. Limited intravenous alcohol administration animal data shows that neurobiological mechanisms play a role in alcohol-related reward following RYGB and that the phenomenon may not be purely pharmacokinetic (Polston et al., 2013).

Most of the current data suggest that the risk of an AUD after bariatric surgery is increased following RYGB; AUD following LAGB does not appear to be significantly increased based on the current available literature. Further elucidating the role that surgical procedure plays in the risk of post-operative AUDs should also be an important focus of future research, as there are few data with LSG (and other newer bariatric procedures). The mechanisms responsible for the association between bariatric surgery and AUDs are not understood at the present time and should be a continued focus of future research. Research should also continue to explore whether particular patients or groups of patients are at increased risk for AUDs postsurgery.

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