Obesity and Responsiveness to Food Marketing Before and After Bariatric Surgery

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Although food marketing is often accused of increasing population obesity, the relationship between individual responsiveness to marketing and obesity has yet to be established: Are people with obesity more responsive to food marketing and, if so, is it a stable trait or can it be reversed by bariatric surgery? We studied the responses to three common marketing tactics that frame foods and portions as healthier than they really are in three groups of women: (a) a group of patients with obesity before, 3 months, and 12 months after bariatric surgery, (b) a control group of lean women, and (c) another control group of women with obesity but not seeking any treatment for their obesity. People with obesity were initially more responsive to food marketing, but bariatric surgery reduced their responsiveness down to the level of lean people. In addition to documenting another potential psychological consequence of bariatric surgery, our study suggests that the higher responsiveness of people with obesity is not a stable individual predisposition and supports the notion of a reciprocal relationship between obesity and sensitivity to environmental influences.

Keywords  Attributions and Inference Making; Beliefs and Lay Theories; Field Experiments; Food and Nutrition; Health Psychology; Neuroscience and Physiological Methods; Personality; Public Policy Issues

Introduction

Rising obesity rates worldwide have been linked to the relentless marketing of calorie-dense and nutrient-poor foods (Harris, Pomeranz, Lobstein, & Brownell, 2009). If food marketing contributes to obesity, people who are particularly responsive to marketing tactics should be more at risk of becoming obese, and marketing responsiveness should be higher among people with obesity than among lean people. In line with this reasoning, children with overweight or obesity have been found to be more responsive to television food advertising than lean children (Russell, Croker, & Viner, 2019). Whether that is also the case in adults and for other marketing tactics is unknown.

Indeed, marketers often employ tactics that frame foods or portion sizes as healthier than they are, leading people to increase their energy intake
more than they realize (for a review, see Chandon, 2013). One study (Wansink & Chandon, 2006) examined the relationship between people’s body mass and their responsiveness to one of these marketing tactics, but it only compared lean people with people with overweight, who differ from people with obesity on a host of medical and socioeconomic dimensions (Moore & Cunningham, 2012). Our first goal is thus to establish whether people with chronic obesity are more responsive to marketing-based framing than lean people.

If responsiveness to food marketing is higher among people with obesity, an important question is whether it reflects a stable dispositional trait—suggesting a one-way causal relationship between responsiveness to marketing and the risk of becoming obese—or whether it can change after weight fluctuations—suggesting a more complex reciprocal relationship, as shown previously for impulsivity (Sutin et al., 2013). Indeed, bariatric surgery—a procedure that involves making changes to the digestive system to promote weight loss—has been found to increase preferences for healthier food by altering reward and taste processing (Behary & Miras, 2015; Ochner et al., 2011). Hence, our second goal is to study whether bariatric surgery impacts the responsiveness to food marketing of people with obesity.

To achieve the first goal, we measured the responsiveness to three marketing framing tactics in three matched groups: (a) patients with severe obesity, before, 3 months, and 12 months after bariatric surgery, (b) a control group of people with obesity but not candidate for surgery, and (c) another control group of lean individuals, whose responsiveness was measured twice, at six-month intervals, in order to measure the effects of test repetition.

**Conceptual Development**

*Effects of Marketing Framing of Food and Portion Sizes*

While many marketing actions can promote unhealthy eating (Cadario & Chandon, 2020), this research focuses on three marketing framing tactics that have been shown to lead to biased food judgements and decisions by either being objectively erroneous (e.g., underestimating the amount of calories in a food) or by lacking internal consistency (e.g., choosing larger portion sizes depending on the framing). These tactics have been investigated in cross-sectional studies of lean people, but their relationship with obesity has yet to be established.

**Brand framing effects**

People underestimate the energy content of foods framed as healthy—the so-called “health halo” effect—and overestimate the energy content of foods framed as indulgent—the “health horn” effect (Burton, Cook, Howlett, & Newman, 2014; Chernev & Gal, 2010), which then influences their energy intake (Provancher, Polivy, & Herman, 2009; Wansink & Chandon, 2006). For example, because Subway® was marketed as being healthier than other fast-food restaurant brands, people erroneously expect a Subway sandwich to contain fewer calories than a McDonald’s® burger with an identical calorie count, leading them to choose higher-calorie side dishes, drinks, or desserts with the Subway sandwich than with the McDonald’s burger (Chandon & Wansink, 2007).

**Size labeling effects**

Despite a significant increase in portion sizes over the past 40 years, consumers often ignore the portion size information available on labels (Lennard, Mitchell, McGoldrick, & Betts, 2001) and neglect quantity information when judging food healthiness (Liu et al., 2019; Rozin, Ashmore, & Markwith, 1996). In particular, Aydinoglu and Krishna (2011) showed that labeling the same food portion as “small” rather than “large” led to an underestimation of its size and to greater food intake.

**Size range effects**

Research has shown that portion size selection is influenced by the largest or smallest sizes available in a choice set. Sharpe, Staelin, and Huber (2008) found that removing the smallest size “S” from a S, M, L, and XL set reduced the chance that size M would be chosen. This occurred because some of the people who had chosen size M in the original set avoided it once it had become the smallest size of the new set, choosing instead to upsize to L, the new “medium” size. Likewise, removing the largest size from the set led people to choose smaller portions.

**Obesity and Responsiveness to Marketing Framing**

*Are individuals with obesity more responsive to food marketing framing?*

Building on existing results showing that, compared with people with a normal weight, those with overweight eat more chocolate candies when they are labeled “low fat” (Wansink & Chandon,
2006), we hypothesize that people with obesity (compared with lean people) are more responsive to marketing framing effects: Their calorie estimates are more likely to be influenced by the use of healthy or indulgent-sounding brand names (H1a); their choice of portion size is more likely to be influenced by the language used to label the portion (H1b), and by the range of sizes available (H1c).

Is responsiveness to food marketing framing dispositional?

If people with obesity are more responsive to food marketing framing (as hypothesized in H1), one explanation may be that this responsiveness reflects stable, dispositional traits in people with obesity. Extant research suggests that personality traits such as low self-control and high reward responsiveness—which are largely dispositional (Takahashi et al., 2007; Tangel, Baumeister, & Boone, 2004)—predict individuals’ responsiveness to food marketing (e.g., Baumeister, 2002; Kidwell, Hardesty, & Childers, 2008; Wadhwa, Shiv, & Nowlis, 2008), but also higher obesity risks (Gerlach, Herpertz, & Loeber, 2015). If responsiveness to food marketing is a stable individual trait, its relationship with obesity is a simple one-directional causal link (responsiveness causes obesity), and people with obesity should remain equally responsive to food marketing after undergoing bariatric surgery.

However, the association between responsiveness to food marketing and obesity may be more complex than a mere one-way causality, such that the responsiveness to food marketing framing may also be a consequence of obesity. Supporting this contention, burgeoning research has demonstrated that bariatric surgery does not only modify patients’ biology, but also their psychology, including changes in cognitive function (Handley, Williams, Caplin, Stephens, & Barry, 2016), and in reward and motivation processing (Schmidt et al., 2020; Volkow, Wise, & Baler, 2017), which are typically at play in framing effects (Covey, 2014). While it is outside the scope of this research to investigate the precise mechanism of action, we expect that, among people with obesity, bariatric surgery reduces the effects of brand framing on calorie estimations (H2a) and the effects of portion size labeling (H2b) and of the range of sizes available (H2c) on portion size choices.

Methods

Participants were enrolled in a clinical trial protocol studying gut microbiota from some of the authors of this manuscript. The same participants were then recruited to study various aspects of decision-making in people with obesity. In this manuscript, we focus on tasks and research questions related to marketing responsiveness. Details about the stimuli, these tasks, and about other tasks pertaining to other research questions are described on the Open Science Framework (OSF): https://osf.io/pgxvh/?view_only=c5aec66ceb4d42bc8a4fa5283f473794. Data and code are available at https://osf.io/qpcj/?view_only=344c16fa481418ea2f30c582d04e908.

Participants

Data collection took place between August 2011 and March 2017 in Paris, France. The “patients with obesity” consisted of 73 women with severe obesity (BMI = 45.9 ± 5.2) scheduled for Roux-en-Y-gastric bypass (RYGB) or adjustable gastric banding surgery (AGB). The “lean controls” group consisted of 41 lean women (BMI = 22.2 ± 1.5), matched to the group of patients with obesity in terms of age, gender, income, employment, and marital status. The sample size of these two groups was determined by the clinical trial protocol studying gut microbiota.

As the patients who qualified for bariatric surgery were more obese than the obese population in general (leading to higher risk of comorbid conditions) and also had to meet specific eligibility criteria for bariatric surgery, we collected data from 29 women with obesity (BMI = 33.1 ± 3.2) who were not scheduled to undergo bariatric surgery or any other weight-loss procedure (the “controls with obesity”). The sample size was limited by the local availability of female participants with obesity who were not seeking treatment and who were willing to participate in the study.

Patients with obesity were tested before surgery (session 1), 3 months (session 2), and 12 months after surgery (session 3). To account for the effects of test repetition, we surveyed the lean control group over two sessions, 6 months apart, in the same hospital setting as the patients with obesity. The group of controls with obesity was surveyed only once. No observation was excluded from the statistical analyses.

Table 1 shows the sample size and sociodemographic characteristics of the three groups. The different sample sizes are due to attrition and to nonresponse to some tasks. Supplementary analyses reported in the Appendix S1 show that the matching was successful, except on education, which was higher in the lean group than in the two obese groups, as is typically the case in the population
Education was, therefore, included as a control variable in the group comparison analyses. Table 2 shows the physical and psychological characteristics of each group at each session. The BMI and body fat mass (evaluated by DXA-scan) of patients with obesity were higher than those of the two control groups before surgery and strongly decreased after surgery. We measured impulse-related psychological characteristics with the Brief Self-Control scale (Tangney et al., 2004) and the Behavioral Inhibition/Approach System (BIS/BAS) questionnaire (Carver & White, 1994). Patients with obesity rated as high as the lean controls and higher than the controls with obesity on Self-Control; there were also differences across groups on three dimensions of BIS/BAS (Drive, Reward Sensitivity, and Fun Seeking). Self-control and BIS/BAS remained stable over time for all groups. Appetite, measured at the start of each session, varied both across groups and over time within the groups. Therefore, the analyses of marketing responsiveness across samples include models controlling for appetite, self-control, and the three dimensions of BIS/BAS varying across groups.

### Procedure

All groups participated in the following three tasks adapted from existing studies, repeated across sessions, in the same order. A study with participants matched to the lean controls (reported in the Appendix S1) shows that all three tasks successfully manipulated the perceived healthiness of food options or portions.
The participants were asked to estimate the number of calories in one portion of eight branded snacks on a Visual Analog Scale ranging from 0 to 500 calories and including the label “one McDonald’s cheeseburger” at the 300 calories mark. We selected four brands framed through advertising and packaging cues as being healthy: One can of Minute Maid® 100% apple juice (149 calories), one can of Minute Maid® limon and nada (178 calories), two Balisto® honey almond granola bars (194 calories), and two Yoplait® fruit yogurts (242 calories). The other four brands were framed as indulgent: One can of Coca-Cola® (139 calories), one can of Oasis® Tropical (149 calories), two mini Mars® bars (160 calories), and four McDonald’s® Chicken McNuggets® (179 calories). Note that, compared with the four “indulgent” snacks, the four “healthy” snacks actually contained 22% more calories, 14% more fat, 33% more carbohydrates, and 8% less protein.

We adapted the paradigm developed by Sharpe et al. (2008) by creating a detailed scenario asking participants to imagine that they were on a three-day road trip in the United States with friends during which they visited a different burger restaurant every day. Participants were shown the menus of three restaurants that are unfamiliar in their country, White Castle, A&W, and Hardee’s, presented in random order. After one filler choice (of burger), they selected a portion of fries—which tested size labeling effects. After another filler task (choice of drink), they selected a drink size—which served to test size range effects.

Size labeling effects were assessed by adapting the procedure used by Aydinoğlu and Krishna (2011) to a within-subject task. All three menus featured the same three portion sizes of fries, explicitly indicating the weight of each: 71, 117, and 154 g. In the control menu, the three sizes were labeled “small”, “medium”, and “large”. In the
“understated” menu, they were labeled “mini”, “small”, and “medium”—which should lead to upsizing because the descriptions minimize the size of the portions. In the “overstated” menu, they were labeled “medium”, “large” and “extra-large”—which should lead to downsizing because the descriptions are inflating the size of the portions.

We examined size range effects by adapting the procedure used by Sharpe et al. (2008) to a within-subject task. We asked participants to select a drink size from the three menus. In the control menu, the drink sizes were 24, 47, 65, and 95 cl. In the “left-truncated” menu, the drink sizes were 47, 65, and 95 cl. In the “right-truncated” menu, the drink sizes were 24, 47, and 65 cl. Note that the “right-truncated” menu was objectively healthier, and the “left-truncated” menu was objectively less healthy. Still, this size range task allowed us to test whether participants made inconsistent choices across menus (say, choosing a different size when the same size is available, as described later).

**Results**

**Are Participants with Obesity more Responsive to Food Marketing Framing?**

**Brand framing effects**

Table 3 shows that calorie content was systematically underestimated for the four snacks framed as healthy, as predicted by the “health halo” effect (e.g., by −15.1% among patients with obesity). Calorie content was systematically overestimated for the four brands framed as indulgent, consistent with the “health horn” effect (e.g., by +49.4% among patients with obesity). We created an overall brand framing index by adding the absolute value of both estimation errors (e.g., 15.1% + 49.4% = 64.5%).

Table 3

<table>
<thead>
<tr>
<th>Marketing effects</th>
<th>Patients with obesity</th>
<th>Lean controls</th>
<th>Controls with obesity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>t₀</td>
<td>t₃</td>
<td>t₁₂</td>
</tr>
<tr>
<td>Mean (SD) N</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brand framing effects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Halo effect: Calorie estimation of “healthy” brands (% error)</td>
<td>−15.1ₐ,ₓ</td>
<td>−10.3ₓ</td>
<td>−14.₃ₓ</td>
</tr>
<tr>
<td>(38.7)</td>
<td>(40.9)</td>
<td>(41.9)</td>
<td>(39.4)</td>
</tr>
<tr>
<td>Horn effect: Calorie estimation of “indulgent” brands (% error)</td>
<td>49.₄ₐ,ₓ</td>
<td>47.₈ₓ</td>
<td>38.₉ₓ</td>
</tr>
<tr>
<td>(58.₇)</td>
<td>(52.₈)</td>
<td>(52.⁰)</td>
<td>(51.₇)</td>
</tr>
<tr>
<td>Brand framing index</td>
<td>6₄₅ₐ,ₓ</td>
<td>5₈₁ₓ</td>
<td>5₃₂ₓ</td>
</tr>
<tr>
<td>(3₄.₇)</td>
<td>(3₁.₆)</td>
<td>(2₇.₈)</td>
<td>(2₇.₁)</td>
</tr>
<tr>
<td>[Halo effect] + [Horn effect]</td>
<td></td>
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</tr>
<tr>
<td>Size labeling effects</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Change in size order from control to understated description (grams)</td>
<td>1₆.₀₃ₐ,ₓ</td>
<td>3₁.₅ₓ</td>
<td>4₄.₉ₓ</td>
</tr>
<tr>
<td>(2₇.₆₃)</td>
<td>(1₈.₇₈)</td>
<td>(1₃.₅₁)</td>
<td>(2₄.₁₇)</td>
</tr>
<tr>
<td>Change in size order from overstated to control description (grams)</td>
<td>1₁.₁₈ₐ,ₓ</td>
<td>2₃.₆ₓ</td>
<td>2₃.₃ₓ</td>
</tr>
<tr>
<td>(2₇.₂₁)</td>
<td>(1₄.₇₃)</td>
<td>(₉.₃₃)</td>
<td>(2₃.₂₃)</td>
</tr>
<tr>
<td>Size labeling index</td>
<td>₂₇₃₈ₐ,ₓ</td>
<td>₆₆₈ₓ</td>
<td>₆₆₂ₓ</td>
</tr>
<tr>
<td>(3₂.₄₇)</td>
<td>(2¹.₃₇)</td>
<td>(₁₈.₄₂)</td>
<td>(2₄.₁₂)</td>
</tr>
<tr>
<td>(total gram change)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Size range effects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control (full) versus left-truncated menu (% participants making inconsistent choice)</td>
<td>₂₅.₄ₐ,ₓ</td>
<td>₁₅.₆ₓ,ᵧ</td>
<td>₄₅.₈ₓ</td>
</tr>
<tr>
<td>(4₃.₈)</td>
<td>(3₆.₇)</td>
<td>(2₁.₆)</td>
<td>(3₆.₆)</td>
</tr>
<tr>
<td>Control (full) versus right-truncated menu (%participants making inconsistent choice)</td>
<td>₂₀₉ₐ,ₓ</td>
<td>₂₂.₂ₓ</td>
<td>₁₁.₉ₓ</td>
</tr>
<tr>
<td>(₄₀.₉)</td>
<td>(₄².₀)</td>
<td>(₃₂.₈)</td>
<td>(₃₆.₆)</td>
</tr>
<tr>
<td>Size range index (% participants making at least one inconsistent choice)</td>
<td>₄₃.₃ₐ,ₓ</td>
<td>₃₁.₁ₓ,ᵧ</td>
<td>₁₆.₇ₓ</td>
</tr>
<tr>
<td>(₄₉.₉)</td>
<td>(₄₆.₈)</td>
<td>(₃₇.₇)</td>
<td>(₄₄.₂)</td>
</tr>
</tbody>
</table>

Note. Means with the same a or b subscript are not statistically different across groups at t₀; whereas, means with the same x or y subscript are not statistically different across sessions within the same group (at the 5% level, two tailed). For patients with obesity, t₀ occurred before bariatric surgery and t₃ and t₁₂ occurred 3 and 12 months after surgery, respectively.

Italics: Values are standard errors.

Bold Values are summary indices constructed based on some of the other measures also reported in the table.
As hypothesized (H1a), the brand framing index was larger among patients with obesity (\( M = 64.5\% \pm 34.7\)%) than among lean controls (\( M = 49.9\% \pm 27.1\)%). This difference is statistically significant in the regression without covariate (Model 1: \( b = -0.15, \ t(138) = -2.15, \ p = .03, \ \eta^2 = 0.06\)), as well as in the three regressions controlling for education, appetite, and impulse-related psychological traits (see Table 4). The difference between patients with obesity and controls with obesity was never statistically different, regardless of the model (all \( ps > .25\)), which is also consistent with H1a.

### Size labeling effects

We computed the change in the amount (in g) of fries ordered between the control and the “understated” conditions, and between the “overstated” and the control conditions. In both cases, an increase indicates that respondents ordered more fries in response portion size labeling. We used the sum of both effects as an overall size labeling index.

Table 3 and 4 show that, as hypothesized (H1b), size labeling effect was larger among patients with obesity (\( M = 27.4 \text{ g} \pm 32.5\)) than among lean controls (\( M = 12.5 \text{ g} \pm 21.4\)) and that these effects were statistically significant regardless of the model (Model 1: \( b = -14.9, \ t(126) = -2.53, \ p = .01, \ \eta^2 = 0.05\), see Table 4 for the other models). Also consistent with H1b, the difference between patients with obesity and controls with obesity was never statistically different, regardless of the model (all \( ps > .23\)).

### Size range effects

We measured size range effects using three binary variables capturing choice inconsistencies across the three menus. We first compared the control menu (24, 47, 65, and 95 cl) and the more indulgent “left-truncated” menu (47, 65, and 95 cl). Participants who chose the same sizes or those who chose the smallest size in both menus made consistent choices (since those who chose 24 cl in the control menu could not choose it in the “left-truncated” menu). All the other choices are considered
inconsistent and indicate that respondents were influenced by the range of sizes in the menu. Similarly, we considered that participants made inconsistent choices between the control and the healthier “right-truncated” menu (24, 47, and 65 cl) when they did not choose the same size or when they did not choose the largest size in both menus. The aggregate size range index indicates whether participants made at least one inconsistent choice across all menus.

As shown in Table 3, the differences across groups were smaller for size range effects than for the other two effects and were never statistically significant at the 5% level (two tailed). Logistic regressions reported in Table 4 show that patients with obesity were only marginally more likely than lean controls to make inconsistent choices (Model 1: $b = -0.79$, $z = -1.80$, $p = .07$, OR = 0.45; see Table 4 for the other models), therefore rejecting $H_{1c}$. The difference between patients with obesity and controls with obesity was never statistically different, regardless of the model (all $p$s $> 0.30$), which is consistent with $H_{1c}$.

**Additional robustness checks**

Analyses reported in Table S1 in the Appendix S1 show that there were no significant differences between the different types of participants with obesity (patients who underwent RYGB, patients who underwent AGB, and controls with obesity). Table S2 shows that the correlation between the responses to the three marketing tactics are low and not significant at the 5% level, indicating that they are not redundant. Table S2 further shows that marketing responsiveness is positively and statistically correlated to body fat and, to a lower extent, to BMI, which is a less precise measure of obesity at the individual level (Neovius, Linné, Barkeling, & Rossner, 2004). Finally, Table S3 shows that across-group comparisons are robust when controlling for individual deviations from the group average in terms of body fat or BMI.

**Does Bariatric Surgery Reduce Responsiveness to Food Marketing Framing?**

To account for the panel structure of the data, we estimated mixed-effects linear regressions for the brand framing and size labeling indices and a mixed-effects logistic regression for the size range index among patients with obesity and among lean controls. Because some patients with obesity skipped one of the two postsurgery sessions, the regressions used pairwise deletion. Table 5 shows the results of the regressions without covariate

### Table 5

**Effects of Marketing Framing Across Sessions**

<table>
<thead>
<tr>
<th></th>
<th>Brand framing index</th>
<th>Size labeling index</th>
<th>Size range index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patients with obesity</td>
<td>Lean controls</td>
<td>Patients with obesity</td>
</tr>
<tr>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
<td>Model 1</td>
</tr>
<tr>
<td>$t_{13}$ versus $t_0$ (patients with obesity)</td>
<td>$-0.05$</td>
<td>$-0.04$</td>
<td>$-20.73^{**}$</td>
</tr>
<tr>
<td>$t_{12}$ versus $t_0$ (patients with obesity)</td>
<td>$-0.11^{*}$</td>
<td>$-0.10^{†}$</td>
<td>$-20.46^{**}$</td>
</tr>
<tr>
<td>$t_{14}$ versus $t_0$ (lean controls)</td>
<td>$-0.03$</td>
<td>$-0.03$</td>
<td>$-1.58$</td>
</tr>
<tr>
<td>Appetite</td>
<td>$0.01$</td>
<td>$0.01$</td>
<td>$0.10$</td>
</tr>
<tr>
<td>Intercept</td>
<td>$0.64$</td>
<td>$0.62$</td>
<td>$0.49$</td>
</tr>
<tr>
<td>N</td>
<td>160</td>
<td>157</td>
<td>75</td>
</tr>
</tbody>
</table>

**Note.** Values are unstandardized regression coefficients and standard errors (in parentheses).

**$^{**}p < .01,$**

**$^{*}p < .05,$**

**$p < .10$** (all tests are two tailed).

*Italics Values are standard errors.*
(Model 1) and controlling for appetite, the only time-varying covariate (Model 2). Additional analyses provided in the Appendix S1 show that the results are similar across the two types of weight-loss surgeries, for all three marketing tactics (Table S4).

Brand framing effects

As shown in Figure 1 and Tables 3 and 5, compared to its presurgery level, the average brand framing index of patients with obesity was directionally (but not significantly) lower 3 months after surgery (Model 1: $b = -0.05, z = -1.13, p = .26$, Cohen’s $f^2 = 0.01$, Model 2: $b = -0.04, z = -0.74, p = .46, f^2 = 0.01$) and was significantly or marginally lower 12 months after surgery depending on the model used (Model 1: $b = -0.11, z = -2.25, p = .03, f^2 = 0.04$, Model 2: $b = -0.10, z = -1.79, p = .07, f^2 = 0.03$), partially supporting H2a. Among lean controls, brand framing effects did not vary significantly over time regardless of the model (Model 1: $p = .47$; Model 2: $p = .51$). This suggests that the decline found among patients with obesity was not simply due to test repetition or to the passing of time between sessions. Twelve months after the surgery, the brand framing index was no longer statistically different between patients with obesity and lean controls ($t(82) = 0.56, p = .58$) and was significantly lower in patients with obesity than in controls with obesity ($t(70) = 2.39, p = .02$).

Size labeling effects

Compared to its presurgery level, the size labeling index among patients with obesity was significantly lower 3 months after surgery (Model 1: $b = -20.73, z = -4.24, p < .001, f^2 = 0.196$, Model 2: $b = -22.37, z = -4.19, p < .001, f^2 = 0.17$) and after 12 months (Model 1: $b = -20.46, z = -4.16, p < .001, f^2 = 0.18$; Model 2: $b = -22.02, z = -4.13, p < .001, f^2 = 0.16$), supporting H2b. Size labeling effects did not significantly vary across sessions for lean controls (Model 1: $p = .76$, Model 2: $p = .59$). Twelve months after the surgery, the size labeling index was no longer statistically different between patients with obesity and lean controls ($t(76) = 1.31, p = .19$) and was significantly lower in patients with obesity than in controls with obesity ($t(62) = 2.09, p = .04$).

Size range effects

Compared to its presurgery level, the size range index among patients with obesity was directionally (but not significantly) lower 3 months after surgery (Model 1: $b = -0.80, z = -1.46, p = .14$, OR = 0.45; Model 2: $b = -0.45, z = -0.75, p = .45$, OR = 0.64) and was statistically lower after 12 months (Model 1: $b = -1.88, z = -2.77, p = .01$, OR = 0.15, Model 2: $b = -1.56, z = -2.23, p = .03$, OR = 0.21), supporting H2c. Among lean control participants, size range effects did not significantly vary across sessions regardless of the model (Model 1: $p = .34$; Model 2: $p = .47$). Twelve months after...
surgery, the size range index was no longer statistically different between patients with obesity and lean controls (\( z = -0.98, p = .33 \)) and was significantly lower in patients with obesity than in controls with obesity (\( z = -2.07, p = .04 \)).

General Discussion

We find that people with obesity are more responsive than lean people to two of three marketing tactics aimed at framing foods or portions as healthier than they really are, but that the responsiveness of people with obesity to all three tactics decreases 12 months after bariatric surgery down to the level of lean people. These findings rule out that people with obesity are more responsive to food marketing tactics purely as a result of a stable individual disposition, such as low self-control or high reward sensitivity, as suggested by research cited earlier. One possible interpretation for these findings is that the relationship between obesity and marketing responsiveness reflects a two-step process whereby people who are more exposed (but who may have been originally equally responsive) to food marketing are more likely to become obese, which then increases their responsiveness to food marketing in a reinforcing loop. Our findings also offer a novel insight into how bariatric surgery—a procedure undertaken by 600,000 people each year around the world (Angrisani et al., 2018)—may be inducing weight loss.

Why would bariatric surgery reduce responsiveness to food marketing? Research that has investigated the psychological consequences of bariatric surgery has tended to focus on neuroscientific explanations. Indeed, brain regions involved in reward and motivation processing show more activity in people with obesity than in lean people (Volkow et al., 2017), while bariatric surgery alters such neural processes through changing the connectivity within the brain’s reward system (Ochner et al., 2011; Schmidt et al., 2020). That being said, it is also possible that a heavy surgical intervention like bariatric surgery strongly increased patients’ motivation to eat healthily and to process food information in a more systematic manner, thus decreasing the impact of marketing framing, which may lead to changes also in other brain processes in addition to the established changes in the reward and motivation circuits.

What remains also unclear is whether the decrease in responsiveness to marketing that followed bariatric surgery is strictly the result of weight loss, or whether it is also the result of the host of the biological and psychological modifications induced by the surgery itself and that would not occur through non-surgery-induced weight loss, such as changes in people’s own beliefs about the cause of obesity (McFerran & Mukhopadhyay, 2013). In line with this reasoning, 12 months after surgery, the responsiveness to marketing of patients with obesity had decreased to the level of lean controls, and below the level of control individuals with obesity, even though most of these patients were still obese (mean BMI = 33.35). Further research should thus examine how obesity-related dysfunctions in metabolic and hormonal signaling processes (Aron-Wisnewsky et al., 2019; Palmiter, 2007; Volkow et al., 2017) may be influenced by bariatric surgery and weight loss, and whether relationships could be found between metabolic and hormonal signals and response to food marketing.

In addition to uncertainty about the underlying mechanism, we acknowledge the following limitations in our study. First, because we surveyed the patients with obesity up to only 1 year after surgery, the durability of the effects beyond that period is unknown. Also, there were unequal sample sizes across participants, and we do not know whether our findings would hold in the absence of such attrition in the group of patients with obesity. Also, all our participants were French women. Future research should thus examine a larger and more diverse sample of people with obesity, including men and adolescents. Further, in our study, responsiveness to portion size range decreased after bariatric surgery, but its association with presurgery obesity status was weaker than for the other two marketing tactics and not statistically significant. Additional research is thus necessary to determine whether this was caused by fatigue (it was the last task), by the binary nature of the responsiveness index, or by other factors. More generally, more research is needed to explore the interactions between biology, the environment, and eating behavior, which are more complex than previously thought.

References


Covey, J. (2014). The role of dispositional factors in moderating message framing effects. *Health Psychology, 33,* 52.


**Supporting Information**

Additional supporting information may be found in the online version of this article at the publisher’s website:

*Appendix S1*. Methodological Details Appendix.