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The presence of real food usurps hypothetical health value judgment in overweight people

Hypothetical and real food choices in obesity

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Abstract

To develop more ecologically valid models of the neurobiology of obesity, it is critical to determine how the neural processes involved in food-related decision-making translate into real-world eating behaviours. We examined the relationship between goal-directed valuations of food images in the MRI scanner and food consumption at a subsequent ad libitum buffet meal. We observed that 23 lean and 40 overweight human participants showed similar patterns of value-based neural responses to health and taste attributes of foods. In both groups, these value-based responses in the ventromedial PFC were predictive of subsequent consumption at the buffet. However, overweight participants consumed a greater proportion of unhealthy foods. This was not predicted by in-scanner choices or neural response. Moreover, in overweight participants alone, impulsivity scores predicted greater consumption of unhealthy foods. Overall, our findings suggest that, while the hypothetical valuation of health of foods is predictive of eating behaviour in both lean and overweight people, it is only the real-world food choices that clearly distinguish them.

Significance statement

Do overweight people make unhealthier food choices than lean people because they value the healthiness of foods less than lean people do? We show that fMRI markers of valuation of healthiness of foods do not differ between the lean and overweight groups. While these markers do predict healthy food choices at an ad libitum buffet, they do not account for an overall greater selection of unhealthy food choices in the overweight group. This suggests that a fundamental shift in obesity may lie in how the presence of food overcomes prior value-based decision-making.

Introduction

It is recognised that a major driver of excess weight gain operates at the higher cognitive levels that control eating behaviour rather than at the level of metabolic regulation. It is important therefore to develop a more sophisticated understanding of the neural bases of food valuation and choice. Data from epidemiological and laboratory studies suggest that obesity is associated with a greater consumption of foods with high sugar and/or fat content (Hooper et al., 2012; Malik et al., 2013; Morenga et al., 2013), or high energy density (Johnson et al., 2009), all of which are widely perceived as unhealthy (National Obesity Observatory, 2011). This does not seem to be driven by differences in the perception of foods' healthiness between lean and overweight people (O'Brien and Davies, 2007). This raises a key question: is obesity associated with a fundamental change in the processes of valuation, such that the consideration of healthiness of foods plays a smaller role in their valuation in people who are overweight than in people who are lean?

There is robust evidence for the existence of food-related goal value signals in the brain (Bartra et al., 2013; Clithero and Rangel, 2013), but there are two key limitations of this data. First, there is no evidence that neural responses associated with subjective valuation of foods presented in the experimental setting of the MRI scanner correlate with real-world eating behaviour outside the scanner. This is necessary to demonstrate if we are to use within-scan measures as surrogates of real-world food valuation, and as predictors of eating behaviour. Second, it is not known if this valuation process differs in relation to weight status.

Alternatively, maladaptive eating in people who are overweight might not be driven by reduced valuation of foods' healthiness. This would be consistent with large-scale surveys that report a high importance attached to the goal of healthy eating for the vast majority of the population, but persistent discrepancies between food intake and dietary recommendations for health (The UK Food Standards Agency, 2009). Maladaptive food choices and the susceptibility to develop obesity have been linked to the personality trait of impulsivity (French et al., 2012), characterised by a reduced ability to inhibit prepotent responses, and a greater tendency to act without forethought, potentially leading to behaviours that might be in conflict with our goals and values.

Distinguishing between these possibilities will contribute to a fuller understanding of the neurobiology of obesity and may identify new targets for intervention. In this study, we set out to explore whether the extent to which subjective ratings of food's healthiness contributes to the neural computation of goal value of foods (health valuation) is predictive of food choices in a buffet lunch served after the scanning session. We predicted that overweight participants would choose fewer healthy, and more unhealthy foods at the buffet, and we sought to investigate whether neural indices of value predicted choice behaviour and distinguished between lean and overweight people.

Materials and Methods

Participants

We recruited 69 healthy, right-handed participants (age M = 30.1, SD = 6.1, range 18-40; BMI M = 27.9, SD = 5.9, range 19.9–44.5 kg/m2; 39 females) in two groups: lean (BMI <25 kg/m^2) and overweight (BMI >25 kg/m), matched for age, gender, education, income, and IQ. All participants had normal or corrected to normal vision, had no history of psychiatric or other significant medical history and reported no contraindications to MRI scanning. Engaging in high intensity workout more than three hours per week was also one of the exclusion criteria; the reason for including the limit of weekly exercise was to exclude athletes whose BMI would, due to increased muscle mass, falsely classify them as overweight. Furthermore, we excluded vegetarians and people with any other specific dietary preferences or allergies relating to the food items used in the study. Particular effort was invested to make the sample of participants representative of the UK population and participants were recruited from the wider community rather than exclusively from the [Author University]. Specifically, given that greater prevalence of overweight and obesity is found in lower socioeconomic groups (Department of Health Public Health Research Consortium et al., 2007; National Obesity Observatory, 2012), effort was made to recruit groups of lean and overweight people with an overall comparable variability of education levels and yearly incomes (in order to dissociate the adiposity-linked differences in food choices and valuation from the potential confound of socioeconomic status).

The study was approved by the [Author University] Psychology Research Ethics Committee and was conducted at two departments of [Author University]. It was carried out in accordance with the principles of the Declaration of Helsinki. All participants provided written, informed consent.

Six participants were excluded from the analysis: three of them did not complete the study and the behavioural data were inadvertently not saved for two participants, which prevented the analysis of their fMRI data. One participant was involved in rigorous physical training (bodybuilding), which was not detected during the screening process. The demographics of the remaining 63 participants (23 lean and 40 overweight), whose data were processed and analysed, are presented in Table 1.

Study design

Before coming to take part in the study, participants were instructed to eat their standard breakfast at home before 8am. All aspects of the study were conducted on a single day in the same order (Figure 1.A). The study session started at 9am, after which the health and taste ratings of the scanner task foods were collected, the scanner tasks were thoroughly explained and practiced, and additional cognitive measures were collected. These included tasks that examined response inhibition (Stop Signal Reaction Time (SSRT) (Logan, 1994), Stroop interference (SI) (Golden and Freshwater, 2002)), a self-report questionnaire assessing impulsivity (BIS-11) (Patton et al., 1995)), and an eating behaviour questionnaire (Dutch Eating Behaviour Questionnaire (van Strien et al., 1986)). The scanning session started at 10:30am, and the buffet lunch was served from 1 to 1:30pm. After lunch, subjects rated the healthiness and taste of the foods offered to them in the buffet, and completed an IQ test (test of G (Cattell and Cattell, 1950)).

The food choice task

The task used to explore food valuation was based on Hare et al. (2009). Prior to the scanning session, participants rated 50 food items (common snack foods), presented on a computer screen, on a five-point scale for their healthiness (Very Unhealthy', 'Unhealthy', 'Neutral', 'Healthy', and 'Very Healthy', coded in the behavioural and fMRI analysis as 1, 2, 3, 4, 5, respectively) and tastiness ('Very Bad', 'Bad', 'Neutral', 'Good', and 'Very Good', coded in the behavioural and fMRI analysis as 1, 2, 3, 4, 5, respectively). This was conducted in two separate blocks, the order of which was counter-balanced across participants (Figure 1.B). Before the taste-rating block, participants were instructed to 'rate the tastiness of each food item without regard for its healthiness', and correspondingly, before the health-rating block they were instructed to 'rate the healthiness of each food item without regard for its tastiness'.

Following the two rating blocks, one item that was rated as neutral on both health and taste scales was selected as the reference food item for that participant (for participants who did have an item rated as neutral on both scales, we selected an item that was rated neutral on the taste scale and healthy on the health scale as the reference item). Given that the reference item was kept consistent throughout for each participant, the valuation was ultimately expressed with reference to this individually specific constant.

Participants were shown a picture of the reference food item at the beginning of the task and told that on each trial they would have to choose between the food item shown on that trial and the reference food item (Figure 1.C). They were told to imagine that each offered swap constitutes a real food choice, and to treat each swap as if it was the only one offered. We note, that in contrast to the task used by Hare et al. (2009), due to our overall study design that included a buffet lunch, our in-scanner food choices were completely hypothetical. To indicate how willing they would be to accept the swap, participants selected (on a sliding scale below the picture of the offered food) between five options: 'Strong No', 'No', 'Neutral', 'Yes', 'Strong Yes', which was taken as a behavioural measure of goal value, and coded in the behavioural and fMRI analysis as 1, 2, 3, 4, 5, respectively.

Since each trial presented a food stimulus (offered to be swapped for the reference food) and therefore entailed a number of perceptuomotor components, we included control trials (in keeping with previous work (Medic et al., 2014; Plassmann et al., 2007)). In the control task, the same 50 foods were presented in 'forced' trials (as opposed to the 'free' trials), in which participants were instructed to select one out of five responses that were randomly shown on the screen ('Please select "Strong No"/"No"/"Neutral"/'"Yes"/"Strong Yes" '). These trials required participants to engage in all the processes involved in the free trials with the critical difference of requiring no subjective valuation. Thus, the aim was to match the free and forced trials as closely as possible, with the exception that the former required participants to indicate the relative value of the food by indicating how willing they were to swap it for the reference item.

Altogether, 50 trials of each trial type (free and forced), of duration 8 seconds, were presented in a randomised order. The picture of the food was presented throughout the entire 8-second duration of the trial. The initial position of the cursor on the sliding scale varied randomly between all of the five positions of the scale. Participants made responses using a standard button box, with the first and second buttons serving to move the cursor down or up the sliding scale, and the third button serving to confirm their response. Once the confirmation button had been pressed, the cursor could not be moved further until the next trial. When the 8-second trial was over, a feedback screen showing the final decision was presented. If the response was not confirmed within 8 seconds, the feedback screen stated 'Not quick enough'. In the analysis, these trials were considered missed trials.

Buffet

Following the scanning session, participants were provided with an ad libitum buffet lunch consisting of a range of sweet and savoury foods that were previously rated as healthy and unhealthy by an independent panel and pair-matched for energy densities (Table 2). After participants had finished eating, the remaining food was weighed.

fMRI analysis

fMRI data were analysed in spm8, using three models to examine distinct experimental questions. First, we sought to identify brain circuitry involved in valuation of the presented food; second, we explored the relationship between pre-scan health and taste ratings and the neural responses related to valuation. Additionally, in the third model, we investigated group differences in the BOLD signal during food valuation.

In model 1, separate regressors were created for free and forced trials. Free and forced behavioural measures of value, i.e. willingness to accept the swap, were used as parametric modulators of these regressors. To examine processes specifically associated with valuation, we calculated the first-level contrasts as the difference between the free and forced parametric modulators. To determine which brain regions are involved in valuation across all participants, at the second-level analysis, we computed a one-sample t-test on the single-participant contrast coefficients from all participants.

In model 2, we investigated the extent to which the health and taste ratings contributed to neural activity underlying goal value computation. We therefore restricted our analysis to the value-coding cluster established in the previous analysis (goal-value coding functional ROI). Health and taste ratings of the foods were used as parametric modulators of the free trial regressors. To determine the contribution of each individual's health and taste ratings to their pattern of neural activity associated with goal value computation, we extracted individual-level health and taste betas from the individual peak goal-value coding voxels within the value-coding functional ROI. To validate the results of this fMRI analysis, we additionally estimated the degree to which each participant's health and taste ratings contributed to the behavioural measure of value inferred from food swaps.

In model 3, we explored the group differences in the BOLD response during food valuation. To examine BOLD response specifically related to valuation, we calculated the first-level contrast as the difference in BOLD responses between the free and forced trials. To examine the differences between lean and overweight participants, we conducted two t-tests (lean < overweight, overweight >lean) on the first-level contrast estimates. We restricted our analysis to the previously defined goal-value coding functional ROI, and also explored the existence of significant clusters across the whole brain.

Statistical analyses and model visualisation

Behavioural data were analysed using linear models (lm package in R) and linear mixed effects models (nlme package (Pinheiro et al., 2013)), in which participants were modelled as a random effect. To perform stepwise linear model selection, we used the stepAIC function, available in the MASS package (Venables and Ripley, 2002). Fitted linear multiple regression models (Figure 4) were visualised using the visreg function (package visreg). Cross-validation of the multiple regression models was performed using the CVlm function (package DAAG).

<u>Results</u>

Behavioural results

Food choice task

Lean and overweight participants did not differ in their health ratings for the food items ($t(61) = -1.47$, $p = 0.15$[a]), suggesting a similar perception of healthiness of these foods. They also did not differ in their taste ratings for the same food items ($t(61) = 1.22$, $p = 0.23$[b]). Based on individual health and taste ratings, foods were classified as healthy or unhealthy (health factor), and as tasty or nontasty (taste factor), resulting in four food categories (healthy-tasty, healthy-nontasty, unhealthy-tasty, unhealthy-nontasty); given that the categorisation of foods was done separately per each participant, based on their individual ratings, foods representing each category differed across participants. Per each participant, foods were designated as tasty if the tastiness of the food was rated as 'Very Good' or 'Good'; or non-tasty, if the participant rated the tastiness of food as 'Neutral', 'Bad' or 'Very Bad'. Analogously, based on the health ratings, each food was designated as either healthy, if the healthiness of the food was rated as 'Very Healthy' or 'Healthy'; or unhealthy, if the participant rated the healthiness of that food as 'Neutral', 'Unhealthy' or 'Very Unhealthy'. We estimated a linear mixed effects model to explore the effect of the health and taste factors, and group (lean and overweight), on the proportion of swaps accepted ('Yes' or 'Strong Yes'). The analysis revealed a single main effect of the taste factor ($F(1,180) = 309.11$, $p < 0.0001$[c]), with participants accepting more swaps for tasty than nontasty foods (Figure 2.A). An analogous analysis of the time taken to decide about the swap as a function of the health and taste factors, and group, found no significant main or interaction effects[d].

Neurocognitive measures of impulsivity

We examined the differences between lean and overweight participants for three measures of impulsivity, namely SSRT[e], SI[f], and the self-report questionnaire BIS-11[g]. None of these measures differed between lean and overweight participants (Table 3).

Buffet consumption

To increase specificity, per each participant, buffet foods were categorised based on individual health and taste ratings, and following the same protocol as with the scanner foods, into healthy and unhealthy, and tasty and nontasty (the participants' health and taste ratings were overall closely aligned with the panel's ratings). For each participant, we summed consumption (in grams) for each of the four food categories. We then estimated a linear mixed effects model to explore the effect of the health and taste factors, and group (lean and overweight), on the weight of food consumed. This analysis revealed a main effect of taste factor ($F(1,169) = 219.13$, $p < 0.0001$[h]), and a smaller, but significant effect of health factor ($F(1,169) = 4.35$, $p = 0.04$[h]) on consumption (Figure 2.B). The group factor did not affect consumption ($F(1,60) = 0.29$, $p = 0.59$[h]), demonstrating that overall, lean and overweight participants did not differ in their total consumption. However, they differed in their food choices within the buffet: consumption was significantly influenced by a three-way interaction between the health and taste of foods, and group ($F(1,169) = 9.29$, $p = 0.003$[h]). This interaction was driven by significant health-by-taste ($F(1,169) = 8.23$, $p = 0.005$[h]) and health-by-group interactions ($F(1,169) = 13.09$, $p < 0.001$[h]). Tukey post-hoc tests within the four food categories revealed that lean participants consumed significantly more healthy-tasty foods than the overweight participants ($p = 0.005$), while the overweight participants consumed significantly more unhealthy-tasty foods than the lean participants ($p < 0.001$). Similar results were seen when consumption was examined separately for solid foods[i] and drinks[j] .

Additionally, we conducted a linear mixed effects analysis of the energy intake at the buffet, analogously to the analysis of weight of consumed foods. Similarly as with the analysis of consumed weight, this analysis revealed main effects of taste of foods ($F(1,169) = 137.84$, $p < 0.0001$[k]) and health of foods ($F(1,169) = 16.2$, $p = 0.0001$[k]) on energy intake. The group factor on its own did not affect energy intake ($F(1,60) = 0.26$, $p = 0.61$[k]). However, energy intake was significantly influenced by a three-way interaction between the health and taste factors, and group ($F(1,169) = 9.98$, $p = 0.002$[k]). This interaction was driven by significant health-by-taste ($F(1,169) = 4.76$, $p = 0.03$[k]) and health-by-group interactions ($F(1,169) = 11.86$, $p < 0.001$[k]). Tukey post-hoc tests within the four food categories revealed that the lean participants consumed significantly more energy from healthy-tasty foods than the overweight participants ($p = 0.004$), while the overweight participants consumed significantly more calories from unhealthy-tasty foods than the lean participants ($p < 0.001$).

fMRI results

As described above, three analyses were performed. The first analysis sought to identify regions involved in the computation of goal value. In the second analysis, we examined the extent to which taste and health attributes contributed to the neural computation of goal value. In the third analysis, we explored group differences in BOLD signal during food valuation.

Model 1: Brain circuitry involved in goal valuation

As expected from previous work (Bartra et al., 2013; Clithero and Rangel, 2013), the strongest goal value signal was detected in the activity of the ventromedial prefrontal cortex (vmPFC)($p < 0.05$, FWE corrected for multiple comparisons at the cluster level, Figure 3.A). Further, activity correlating with goal value was found in the regions of the posterior cingulate cortex and cuneus (Table 4). For completeness, we conducted two additional analyses. Firstly, we explored the correlation of neural activity with free and forced decisions separately. Whereas the neural activity correlating with free decision strength in free trials mimicked the pattern of neural activity in the main contrast, there was no region, even at a liberal threshold of $p < 0.001$ uncorrected, whose activity correlated with forced decision strength in forced trials. This confirms that the effects established in the main contrast were not driven by activity associated with forced trials. Secondly, we investigated whether there was a region whose activity tracked the mismatch between free decision and the randomly ascribed forced decision for the same food item during forced trials. In other words, we examined whether being forced to make decisions that deviated from how one would normally decide in relation to a given food item was associated with enhanced responses. However, no such region was detected, even at a liberal threshold of $p < 0.001$ uncorrected.

Model 2: The contribution of health and taste attributes to goal value computation

In the second analysis, the value-coding cluster in the vmPFC established in the previous analysis was used as a functional ROI, given its most consistent association with goal value computation in the literature. To determine the contribution of health and taste attributes to the neural activity associated with goal value computation, we extracted individual-level health and taste betas from the individual peak goal-value coding voxels within the vmPFC functional ROI.

Individual-level taste betas for this sample were significantly greater than zero (t(62) = 6.42, p < 0.0001[l], Figure 3.B) indicating a significant contribution of taste rating to the neural activity in the vmPFC. In contrast, foods' health ratings on average did not predict neural activity in the vmPFC (t(62) = 0.88, p = 0.38[m], Figure 3.B), though there was considerable inter-individual variability (coefficient of variation (CV) = 800, compared to CV of 123.68 for the taste betas). Furthermore, in a linear mixed effects model exploring the effect of attribute (health and taste) and group (lean and overweight) on the magnitude of neural betas, a significant main effect of attribute was established (F(1, 61) = 23.24, p < 0.0001[n]), with neural taste betas being significantly greater than neural health betas in both lean and overweight participants. No main effect of group (F(1,61) = 0.21, p = 0.65[n]) or attribute-by-group interaction (F(1,61) = 1.54, p = 0.22[n]) was detected. A separate, single-attribute analysis revealed that neither health (t(61) = -1.69, p = 0.09[o]) nor taste betas (t(61) = 0.45, p = 0.66[p]) differed between the groups. Additionally, given the significant interaction between BIS-11 measure of impulsivity and food consumption in the buffet (see below), we expanded the current model of neural betas by including BIS-11 scores. While the attribute remained a significant predictor of neural betas (F(1,59) = 22.5, p < 0.0001[q]), no other main or interaction effects were detected[q].

To validate the analysis of neural betas, the contributions of health and taste attributes of foods to the behavioural measure of food's goal value, i.e. the behavioural health and taste betas, were extracted separately for each participant. Across all the participants, the mean taste beta was significantly greater than zero (t(62) = 21.53, p < 0.0001[r]), whereas the mean health beta was not significantly different from zero (t(62) = 1.92, p = 0.06[s]). The behavioural analysis therefore replicated the results of the fMRI analysis in showing that the taste attribute, but not the health attribute, was a significant contributor to goal valuation of foods. Furthermore, in a linear mixed effects model exploring the effect of attribute (health, taste) and group (lean, overweight) on the magnitude of behavioural betas, results analogues to the analysis of the neural betas were obtained: a significant main effect of attribute was established ($F(1, 61) = 100.92$, $p < 0.0001$[t]), with behavioural taste betas significantly greater than health betas in both lean and overweight participants. No main effect of group ($F(1,61) = 0.52$, $p = 0.47$[t]) or attribute-by-group interaction ($F(1,61) = 0.01$, $p = 0.94$[t]) were detected. A separate, single-attribute analysis revealed that neither health ($t(61) = -0.39$, $p = 0.69$[u]) nor taste betas ($t(61) = -0.73$, $p = 0.47$[v]) differed between the groups. Similarly as in the case of neural betas, the inclusion of BIS-11 as an additional predictor did not explain more variance in behavioural betas: the attribute remained a significant predictor of behavioural betas ($F(1,59) = 100.9$, $p < 0.0001$[w]), while no other main or interaction effects were detected[w].

Model 3: Exploring group differences in BOLD response during valuation

Additionally, we investigated the group differences in the BOLD response during valuation. We conducted an ROI-based analysis in the vmPFC functional ROI, and explored the existence of significant clusters at the whole brain level. T-tests, exploring the difference between lean and overweight participants (lean > overweight, overweight > lean) failed to a find significant activation in the vmPFC (p, 0.025, FWE small volume correction, Bonferroni-corrected for 2 tests), or any significant clusters at the whole brain level ($p<0.025$, FWE corrected for multiple comparisons at the cluster level, Bonferroni-corrected for 2 tests).

Model of healthy food consumption

Finally, we explored whether the pattern of food consumption in the buffet could be predicted by the individual-level neural betas, and if this relationship was modulated by group. Further, we examined whether the inclusion of measures of impulsivity in such a model would capture more variance of the buffet food consumption.

Given that the greatest variability in food consumption across all participants was driven by the health attribute of foods, we used the proportion of healthy foods consumed in the buffet as our main outcome variable (i.e. the consumed weight of foods individually perceived as healthy out of the total consumption of all foods). We conducted a linear multiple regression analysis in two stages, performing a stepwise model selection at each stage. We used the stepAIC function implemented in the MASS package in R, which selects the best model fit by minimising the Akaike's information criterion (AIC) (Venables and Ripley, 2002). Both a forward and backward model selection were used, allowing for interactions between variables. To reduce colinearity, all of the continuous predictors were mean-centred.

In the first stage of this analysis, the neural health beta and group (overweight minus lean) were included as predictors of the proportion of healthy foods consumed. The stepwise procedure returned a model in which the neural health beta and group were identified as independent, non-interacting predictors of the proportion of healthy foods consumed (model 1 in Table 5). The model captured 22.09% of the variance of healthy food consumption ($F(2,59) = 9.65$, $p < 0.001$); the 10-fold cross-validation of the model returned a mean square of prediction error (ms) of 0.0596. The neural health beta positively predicted the proportion of healthy foods consumed across all participants ($\beta = 0.26$, $p = 0.03$[x]), however, over and above this association, the overweight participants consumed a significantly smaller proportion of healthy foods (i.e. a greater proportion of unhealthy foods) than the lean participants ($\beta = -0.37$, $p = 0.002$[x]).

In the second stage of the analysis, in addition to the predictors above, we included the three measures of impulsivity: SSRT, SI and BIS-11 scores. In this case, the stepwise procedure revealed a best fitting-model that explained 43% of the variance of healthy food consumption ($F(4,55) = 12.12$, $p < 0.0001$) (model 2 in Table 5, Figure 4.A and 4.B), with the cross-validation ms = 0.0451. The neural health beta ($\beta = 0.22$, $p = 0.03$[y]) and group (β = -0.47, p < 0.0001[y]) remained as significant independent predictors of the proportion of healthy food consumed (Figure 4.A). Only the BIS-11 remained as a measure of impulsivity in the best fitting model, and there was a significant interaction between BIS-11 impulsivity scores and group (β = -0.43, p = 0.02[y]). In overweight participants, increasing BIS-11 impulsivity was predictive of a smaller proportion of healthy foods consumed (i.e. greater consumption of unhealthy foods), but there was no such association in the lean participants (Figure 4.B).

To validate the above models, the same model procedures were repeated substituting the neural health betas with the behavioural health betas, and these resulted in analogous best-fitting models, with similar parameter estimates[z,α] (Table 6). The analogous analysis for the proportion of tasty food consumption, with neural or behavioural taste betas, and all other predictors as above, failed to find a significant model of tasty food consumption predicted by any combination of these variables.

Discussion

Our findings in lean and overweight people offer intriguing insights into food valuation; its relationship to neural signals and the impact on decision-making. To summarise, we confirmed that value-based decision-making is related to vmPFC activity, with activity in this region reflecting the goal value of presented foods. The degree to which the health and taste attributes of foods contributed to this vmPFC activity (the neural health and taste 'betas') did not differ between lean and overweight participants. Importantly, the contribution of health attributes to the neural value signal was predictive of the proportion of healthy foods consumed in the buffet, demonstrating its validity as a measure of real-world valuation and choice. In both lean and overweight groups, those with higher health betas chose a greater proportion of healthy foods, and critically, this relationship did not differ between the groups. This is demonstrated by the similar slopes for the two groups in the graph (Figure 4.A). However, the overall proportion of healthy foods consumed in the buffet was significantly greater in lean participants, i.e., overweight participants consumed a significantly greater proportion of unhealthy foods. This is demonstrated by the differing intercepts for the two groups (Figure 4.A). Our results therefore indicate that the increased real-world consumption of unhealthy foods by people who are overweight is not driven by reduced valuation of food's healthiness, as assessed by subjective or neural responses. Rather, for a given level of such value placed upon health, there is less actual consumption of healthy food in the overweight people. Intriguingly, in the overweight participants, the proportion of healthy foods consumed was further modulated by impulsivity scores: participants who were overweight and who were highly impulsive consumed the largest proportion of unhealthy foods in the buffet. Below, we consider the implications of these findings.

At the group level, the taste attribute significantly contributed to the neural computation of goal value of foods (in line with previous work by Hare et al. (2009)), and was also a major factor affecting food choices at the buffet. It is important to note that while in the scanner food choice task, participants made binary forced choices, in the buffet lunch, they freely selected foods to consume, and unsurprisingly, predominantly chose foods that they rated as tasty. In other words, there was practically no inter-individual variability in the proportion of tasty foods consumed in the buffet, which explains why the contribution of the taste attribute to the goal valuation of foods in the vmPFC at the individual level did not predict individual consumption of tasty foods in the buffet lunch.

In contrast, the health attribute was not, at the group level, a significant contributor to the goal value computation of foods in the vmPFC in either lean or overweight participants. This was because there was, as might be expected, appreciable inter-individual variability in the contribution of the health attribute to the goal value computation within each group, with no differences between the groups. Capitalising on this variability, we show that, in both groups, it predicted the proportion of healthy foods consumed in the buffet. In other words, the neural signal of health valuation – the weight given to the health attribute in the goal value computation of foods – predicts real-world choices. This provides support for the use of such measures in studying goal valuation in relation to eating choices.

However, while the hypothetical choice offered in the scanner produced a neural signal for health valuation that was strongly predictive of subsequent individual-level eating behaviour, it did not predict differences between lean and overweight people (as demonstrated by parallel slopes on Figure 4A). Importantly, however, overweight participants ate a significantly smaller proportion of foods they individually regarded as healthy, compared to their lean counterparts (as demonstrated by the difference in intercepts on figure 4A). This suggests that over and above the effect of hypothetical health valuation, which doesn't differ between the groups, and equally affects their real-world behaviour, a real-world bias towards unhealthy foods is present in people who are overweight.

What might drive this effect? One possibility is that a different behavioural construct - other than goal-directed valuation – may mediate the differences between lean and overweight participants in real-world food choices. It is relevant, in this respect, that impulsivity scores showed their effects only in the overweight individuals in the context of actual consumption. In children, impulsivity scores have been linked to greater BMI and greater food consumption, however this relationship is less clear in adults (French et al., 2012), where several studies suggest that greater impulsivity scores per se do not confer risk to maladaptive eating or obesity. More often, impulsivity scores have been reported to interact with implicit measures of motivation for foods in predicting food intake and obesity (Epstein et al., 2014; Hofmann et al., 2009; Nederkoorn et al., 2010; Rollins et al., 2010). This suggests that the combination of a high motivation for food and a reduced capacity to inhibit prepotent responses act together in raising the risk of over-eating and obesity.

According to the theory of incentive salience, such implicit motivation, or 'wanting', can be dissociated from the explicit valuation of rewards, and is induced upon encountering rewards, or their associated stimuli, that have previously been experienced as pleasant, or liked (Berridge, 2007). Highly palatable foods – which are often perceived as unhealthy – are thus likely to induce the strongest implicit motivation. In line with these theoretical perspectives, there is evidence that such motivation is most strongly induced in the physical presence of rewards (Bushong et al., 2010; Mischel and Moore, 1973; Woelbert and Goebel, 2013), consequently affecting our decisions and often promoting divergence from our goals in many decision-making scenarios, including eating. For example, the expression of such motivation might explain the effects of food cues to increase appetite (Ferriday and Brunstrom, 2008). Critically, its dependence on the physical presence of rewards provides a good conceptual fit to our data, where differences in food choices between lean and overweight participants were only observed in the buffet, i.e. once participants were presented with foods to choose for immediate consumption.

Several studies indicate that the effects of physical presence of foods on consumption, and motivation for foods, might be more pronounced in overweight than in lean participants. Schachter (Schachter and Rodin, 1974) argued that overweight participants are more sensitive to external cues of food proximity than lean participants. More recently, it was demonstrated that overweight participants express a comparatively greater motivation/desire for food following exposure to food cues (Ferriday and Brunstrom, 2011; Tetley et al., 2009). Studies exploring the effects of food cues on eating behaviour in children demonstrated that overweight children, upon smelling food (Jansen et al., 2003) or watching food TV commercials (Halford et al., 2004), increase their consumption to a greater extent than lean participants. Furthermore, it has been reported that overweight participants are willing to work harder to obtain food rewards (Saelens and Epstein, 1996; Temple et al., 2008). Overall then, the between-group difference in food choices in the real versus hypothetical condition, which we observed here, could reflect group differences in health valuation across the two conditions, as well as differences in the implicit motivation for food, and the extent to which trait impulsivity manifests in the presence of food.

Another possibility that we should consider is that it is differential valuation that drives differing choices across groups. Indeed, it is known that different choices may be made in the hypothetical compared to the real condition. Despite the demonstration that the same neural circuitry encodes both hypothetical and real decisions (Kang et al., 2011), a number of studies have described a hypothetical bias, i.e. the tendency to overstate hypothetical valuations (List and Gallet, 2001; Little and Berrens, 2004; Murphy et al., 2005). In the study by Kang et al. (2011), while the indifference curves for hypothetical and real choices had the same shape (reminiscent of the parallel slopes in Fig 4A), the indifference point in the hypothetical condition was shifted towards a larger value. The reported existence of such a bias provides one way of interpreting our data: while we have demonstrated the predictive validity of hypothetical valuation, we acknowledge the possibility that overweight participants might have attributed greater weight to food's healthiness in the hypothetical than in the real-world condition. We note that, compared to the hypothetical scanner condition, in the buffet, participants were not constrained by limited time to make choices, and were also in a hungrier state, all of which could have been factors that contributed to a change in health valuation in the real condition. Such an account is in line with sequential sampling models of decision-making, which describe valuation as a sequential process, in which the recollection of new information or a change in conditions can gradually modify the initial value estimate (Otter et al., 2008).

We were only able to study a limited range of foods and it is not possible to study eating behaviour in this detail in naturalistic settings. We cannot be certain how the scanner or the buffet meal affected individual behavior, despite our efforts to create a relaxed eating environment for the latter. One thing is clear, while fMRI signals were meaningful and predictive of real-world behaviours, it was only with the presentation of real food choices that the group differences emerged. The study thus provides an important indication that, while fMRI experiments offer precise and predictive measures of key processes related to value, choice and consumption, they must be complemented by other, more naturalistic measures.

In summary, we show that the individual variability in the weights given to health attributes in goal value computation of foods in the vmPFC predicts food choices in a buffet lunch. More specifically, we demonstrated that people who are overweight make fewer real-world healthy food choices compared to their lean counterparts, in contrast to the hypothetical condition, where their health valuations of foods are indistinguishable from those of lean participants. While impulsivity did not fully account for these differences, it was striking that, in overweight participants only, increased impulsivity scores were associated with a greater proportion of unhealthy foods consumed. Importantly, these results suggest that the bias towards consumption of unhealthy foods among participants who are overweight is expressed primarily in the presence of readily available foods. They add further weight to existing evidence that interventions to reduce food consumption in those who are overweight are more likely to be effective when targeted at the processes, often automatic and non-conscious, that get activated by the omnipresence of highly palatable unhealthy foods in our everyday environments.

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Figure legends

Figure 1. Study design and experimental task. A. Study design. B. Before the scanner session, participants rated 50 foods for their healthiness and tastiness, in two separate ratings blocks, the order of which was counterbalanced across participants. For each participant, the health- and taste-neutral food was selected as the reference food for the scanner task. C. The scanner food choice task featured the same 50 items presented as part of free and forced trials. Free and forced trials, of duration 8s, were presented in a randomised order. After the decision trial was over, a 1s feedback screen presented the decision that was made. This was followed by a 0.5s blank screen. On 30 random occasions during the course of the task, a 6s null trial with a fixation cross was presented after the blank screen.

Figure 2. Food choices in the scanner task and in the buffet lunch. A. The proportion of acceptance of food swaps (selecting 'yes' or 'strong yes') in the scanner food choice task, across four categories of foods, in lean ($n = 23$) and overweight participants ($n = 40$). B. Buffet consumption (expressed as weight of consumed foods) across four food categories, in lean and overweight participants. ** $p < 0.01$, *** $p < 0.001$. Error bars represent SEM.

Figure 3. Neural measures of food's goal value. A. The neural representation of goal value in the vmPFC. The results of the fMRI analysis were rendered onto a standard SPM8 T1 template image, with corronal and sagittal sections presented at the coordinates appropriate for displaying the vmPFC cluster ($p_{FWE} < 0.05$, corrected at the cluster level, $p < 0.001$ uncorrected threshold). B. Health and taste betas extracted from the vmPFC activity, in lean and overweight participants. Error bars represent SEM.

Figure 4. Model of healthy food consumption. Visual depiction of the multiple linear regression model 2 (Table 2). A. A partial residual plot of the proportion of healthy foods consumed as a function of the neural health beta, in lean and overweight participants. B.

A partial residual plot of the proportion of healthy foods consumed as a function of BIS-11 impulsivity scores, in lean and overweight participants. Each dot represents one participant.

Table legends

Table 1. Study sample demographics

Table 2. Foods comprising the buffet lunch

Table 3. Mean scores of neurocognitive measures of impulsivity in lean and overweight participants

Table 4. Brain regions correlated with goal value

Table 5. Regression coefficients and corresponding p-values of the best-fitting models of healthy food consumption in the buffet, as a function of neural health betas, group and impulsivity scores

Table 6. Regression coefficients and corresponding p-values of the best fitting models of healthy food consumption in the buffet, as a function of behavioural health betas, group and impulsivity scores

Table 7. Statistical table

Table 1

Measure	Lean (n=23) Mean (SD)/n	Overweight/Obese (n=40) Mean (SD)/n	t/χ^2	p
BMI	21.88 (1.3)	30.84 (4.82)	8.70	<0.001
Age	29.78 (6.00)	29.85 (5.75)	0.04	0.97
Gender				
Female	13	23	0.01	0.99
Male	10	17		
Education				
University degree	13	21	0.01	0.96
No university degree	10	19		
Average yearly income (£)				
≤ 9,999	7	11	2.41	0.49
10,000 – 19,999	10	13		
20,000 – 29,999	3	12		
30,000 – 39,999	3	3		
Ethnicity				
White	20	35	0.90	0.82
Black	1	2		
Asian	2	2		
Other	0	1		
IQ	107.45 (12.78)	111.28 (17.45)	0.90	0.37

DEBQ				
Restraint	22.86 (8.35)	26.58 (5.87)	2.05	0.05
Emotional	27.23 (8.15)	31.58 (9.58)	1.80	0.08
External	30.73 (4.58)	32.45 (6.15)	1.15	0.26

Table 2

Food	kcal/100g	Fat /100g	Sat fat/ 100g	Weight/Volume as served	Calories available
Cheddar crackers	509	27.7	16.0	200g	1018
Oatcake crackers	449	21.8	8.4	200g	898
Chocolate mini bites	440	19.8	3.5	200g	880
Eat natural cereal bar	456	24.7	16.4	200g	912
Fruit pastille sweets	330	Trace	-	100g	330
Dried mixed fruit	280	0.6	0.2	100g	280
Scotch eggs	235	15.3	8.0	400g	940
Broccoli and tomato quiche	215	13.2	4.3	400g	860
BLT sandwich	225	10.0	2.2	354g	797
Chicken salad Sandwich	195	7.5	1.0	400g	780
Trifle	160	5.4	3.4	600g	960
Strawberry yoghurt	111	2.6	1.7	600g	666
Coke	42	-	-	1 litre	420

Orange juice	48	-	-	1 litre	480
Diet coke	-	-	-	1 litre	-
Water	-	-	-	1 litre	-

Table 3

Measure	Lean Mean (SD)	Overweight Mean (SD)	t	p
SSRT (n = 61)	161.09 (39.5) ms	172.1 (58) ms	-0.80	0.43[e]
SI (n = 62)	229.03 (231.07) ms	243.71 (249.23) ms	0.23	0.82[f]
BIS-11 (n=63)	66.74 (7.79)	62.3 (9.11)	1.96	0.06[g]

Table 4

Region	Side	Cluster size (voxels)	Peak MNI coordinates			Peak Scores	
			x	y	z	T	Z
Medial Frontal Gyrus	L/R	1556	-8	44	-4	6.3	5.55
Cuneus	R	663	18	-92	20	5.25	4.78
Posterior Cingulate	L/R	544	-8	-46	36	4.48	4.16

$p<0.05$ whole-brain FWE correction for multiple comparisons at the cluster-level ($p<0.001$ uncorrected threshold).

Table 5

	Predictor	β	p
Model 1[x]	Neural health beta	0.26	0.03
	Group (Overweight - Lean)	-0.37	0.002
Model 2[y]	BIS-11	0.04	0.83
	Neural health beta	0.22	0.03
	Group (Overweight - Lean)	-0.47	< 0.001
	BIS-11:Group (Overweight - Lean)	-0.43	0.02

[x] $F(2,59) = 9.65$, $p < 0.001$; $R^2 = 0.22$, ms = 0.0596

[y] $F(4,55) = 12.12$, $p < 0.000$; $R^2 = 0.43$, ms = 0.0451

Table 6

	Predictor	β	p
Model 1[z]	Behavioural health beta	0.44	< 0.0001
	Group (Overweight - Lean)	-0.4	< 0.001
Model 2[α]	BIS-11	0.04	0.81
	Behavioural health beta	0.26	0.03
	Group (Overweight - Lean)	-0.47	< 0.001
	BIS-11:Group (Overweight - Lean)	-0.41	0.02

[z] $F(2,59) = 17.61$, $p < 0.0001$, $R^2 = 0.35$, ms = 0.0521

[α] $F(4,55) = 12.3$, $p < 0.0001$, $R^2 = 0.43$, ms = 0.0457

Table 7

Test	Data structure	Type of test	Test statistic	p-value	[Confidence intervals]/Power
a: Overweight - Lean	Normal distribution	Linear mixed-effects model	t(61) = -1.47	0.15	[-0.25, 0.04]
b: Overweight - Lean	Normal distribution	Linear mixed-effects model	t(61) = 1.22	0.23	[-0.09, 0.37]
c: Main effect of Taste	Normal distribution	Linear mixed-effects model	F(1,180) = 309.11	< 0.0001	1
c: Main effect of Health	Normal distribution	Linear mixed-effects model	F(1,180) = 2.78	0.1	0.39
c: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1, 61) = 0.74	0.39	0.14
c: Health x Taste interaction	Normal distribution	Linear mixed-effects model	F(1,180) = 0.51	0.48	0.11
c: Health x Group interaction	Normal distribution	Linear mixed-effects model	F(1,180) = 0.2	0.66	0.07
c: Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,180) = 0.03	0.87	0.05
c: Health x Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,180) = 0.17	0.68	0.07
d: Main effect of Taste	Normal distribution	Linear mixed-effects model	F(1,180) = 1.88	0.17	0.28
d: Main effect of Health	Normal distribution	Linear mixed-effects model	F(1,180) = 0.96	0.33	0.17
d: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1,61) = 1.74	0.19	0.27
d: Health x Taste interaction	Normal distribution	Linear mixed-effects model	F(1,180) = 0.37	0.54	0.09
d: Health x Group interaction	Normal distribution	Linear mixed-effects model	F(1,180) = 0.61	0.43	0.12
d: Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,180) = 2.19	0.14	0.32

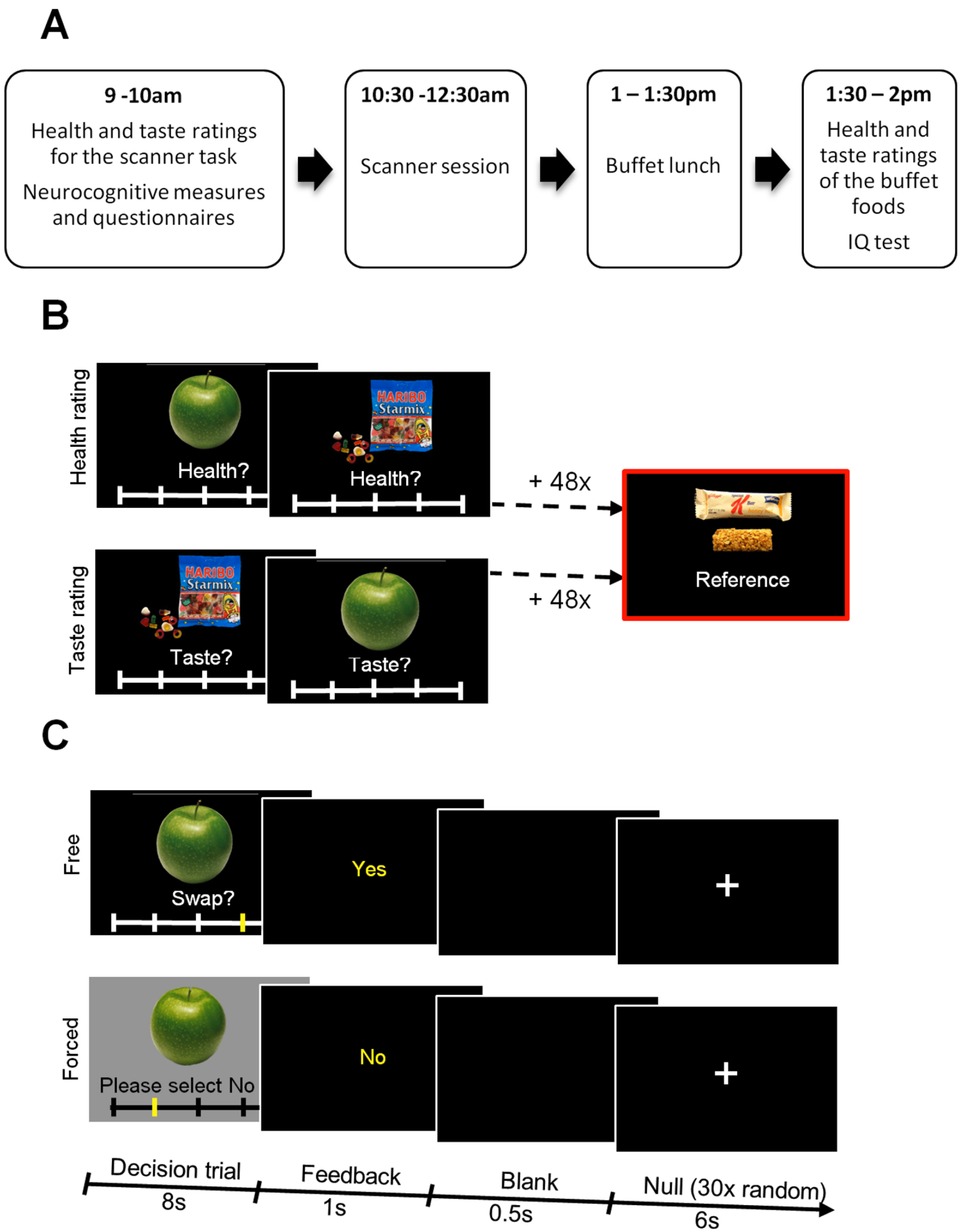
Test	Data structure	Type of test	Test statistic	p-value	[Confidence intervals]/Power
d: Health x Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,180) = 0.04	0.85	0.05
e: Overweight - Lean	Normal distribution	Two-sample t-test	t(1,59) = -0.8	0.43	[-38.4, 16.4]
f: Overweight - Lean	Normal distribution	Two-sample t-test	t(1,60) = -0.24	0.81	[-156, 122]
g: Overweight - Lean	Normal distribution	Two-sample t-test	t(1,61) = 1.96	0.06	[-0.09, 8.97]
h: Main effect of Taste	Normal distribution	Linear mixed-effects model	F(1,169) = 219.13	<0.0001	1
h: Main effect of Health	Normal distribution	Linear mixed-effects model	F(1,169) = 4.35	0.04	0.56
h: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1,60) = 0.29	0.59	0.08
h: Health x Taste interaction	Normal distribution	Linear mixed-effects model	F(1,169) = 8.23	0.005	0.83
h: Health x Group interaction	Normal distribution	Linear mixed-effects model	F(1,169 = 13.09	0.0004	0.96
h: Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,169) = 0.13	0.72	0.07
h: Health x Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,169) = 9.29	0.003	0.87
i: Main effect of Taste	Normal distribution	Linear mixed-effects model	F(1,162) = 135.05	< 0.0001	1
i: Main effect of Health	Normal distribution	Linear mixed-effects model	F(1,162) = 6.2	0.01	0.71
i: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1,60) = 0.01	0.97	0.05
i: Health x Taste interaction	Normal distribution	Linear mixed-effects model	F(1,162) = 0.48	0.49	0.11
i: Health x Group interaction	Normal distribution	Linear mixed-effects model	F(1,162 = 8.04	0.005	0.82

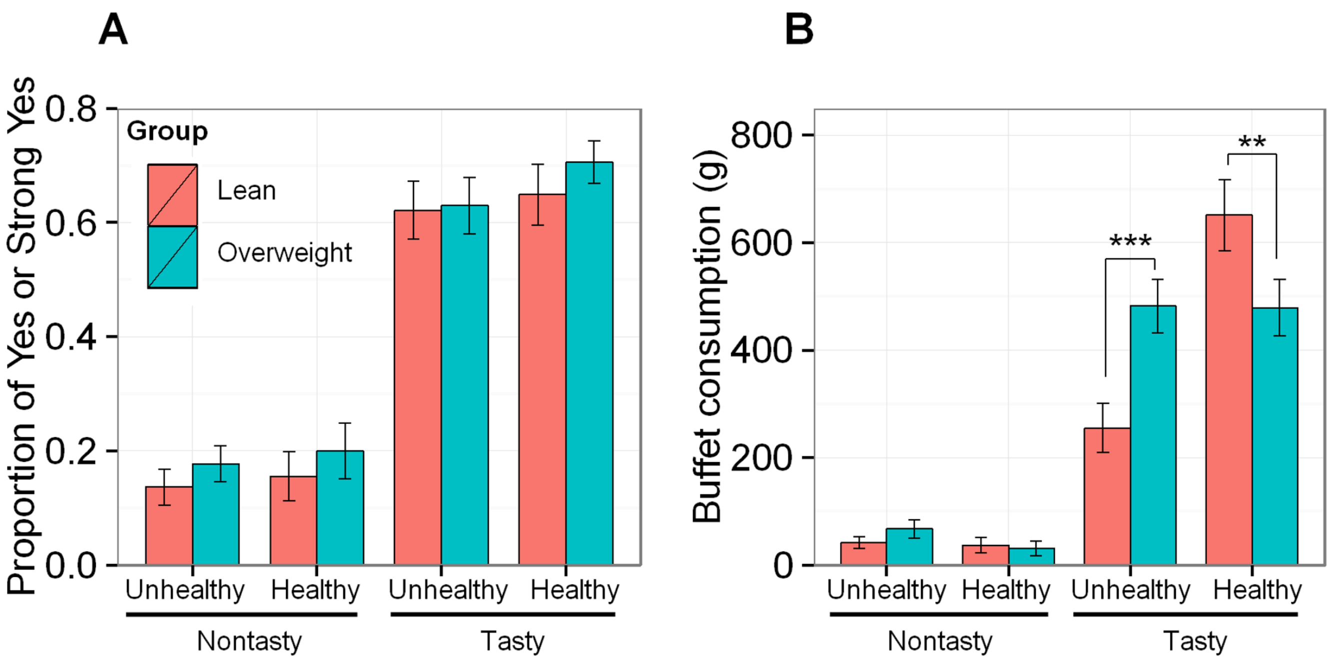
Test	Data structure	Type of test	Test statistic	p-value	[Confidence intervals]/Power
i: Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,162) = 0.04	0.84	0.05
i: Health x Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,162) = 7.06	0.009	0.77
j: Main effect of Taste	Normal distribution	Linear mixed-effects model	F(1,92) = 59.26	< 0.0001	1
j: Main effect of Health	Normal distribution	Linear mixed-effects model	F(1,92) = 41.04	< 0.0001	1
j: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1,60) = 1.1	0.29	0.19
j: Health x Taste interaction	Normal distribution	Linear mixed-effects model	F(1,92) = 1.52	0.22	0.24
j: Health x Group interaction	Normal distribution	Linear mixed-effects model	F(1,92) = 3.21	0.08	0.44
j: Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,92) = 0.59	0.44	0.12
j: Health x Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,92) = 2.52	0.12	0.36
k: Main effect of Taste	Normal distribution	Linear mixed-effects model	F(1,169) = 137.84	<0.0001	1
k: Main effect of Health	Normal distribution	Linear mixed-effects model	F(1,169) = 16.2	0.0001	0.98
k: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1,60) = 0.26	0.61	0.08
k: Health x Taste interaction	Normal distribution	Linear mixed-effects model	F(1,169) = 4.76	0.03	0.59
k: Health x Group interaction	Normal distribution	Linear mixed-effects model	F(1,169 = 11.86	0.0007	0.94
k: Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,169) = 0.05	0.83	0.06
k: Health x Taste x Group interaction	Normal distribution	Linear mixed-effects model	F(1,169) = 9.98	0.002	0.89

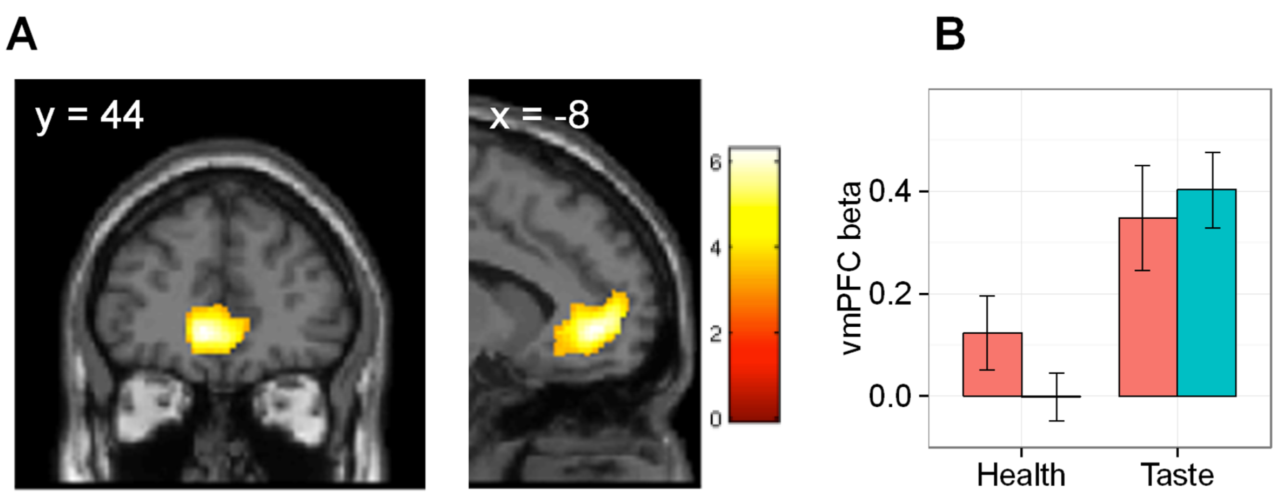
Test	Data structure	Type of test	Test statistic	p-value	[Confidence intervals]/Power
l	Normal distribution	One-sample t-test	t(62) = 6.42	<0.0001	[0.26, 0.5]
m	Normal distribution	One-sample t-test	t(62) = 0.88	0.38	[-0.04, 0.12]
n: Main effect of Attribute	Normal distribution	Linear mixed-effects model	F(1,61) = 23.24	<0.0001	0.99
n: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1,61) = 0.21	0.65	0.07
n: Attribute x Group interaction	Normal distribution	Linear mixed-effects model	F(1,61) = 1.54	0.22	0.24
o: Overweight - Lean	Normal distribution	Two-sample t-test	t(61) = -1.69	0.09	[-0.03, 0.3]
p: Overweight - Lean	Normal distribution	Two-sample t-test	t(61) = 0.45	0.66	[-0.3, 0.19]
q: Main effect of Attribute	Normal distribution	Linear mixed-effects model	F(1,59) = 22.5	<0.0001	0.99
q: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1,59) = 0.2	0.65	0.07
q: Main effect of BIS-11	Normal distribution	Linear mixed-effects model	F(1,59) =0.01	0.83	0.06
q: Attribute x Group interaction	Normal distribution	Linear mixed-effects model	F(1,59) = 1.5	0.23	0.24
q: Attribute x BIS-11 interaction	Normal distribution	Linear mixed-effects model	F(1,59) = 0.1	0.75	0.06
q: Group x BIS-11 interaction	Normal distribution	Linear mixed-effects model	F(1,59) = 0.01	0.93	0.05
q: Attribute x Group x BIS-11 interaction	Normal distribution	Linear mixed-effects model	F(1,59) = 0.01	0.93	0.05
r	Normal distribution	One-sample t-test	t(62) = 21.53	< 0.0001	[0.51, 0.61]
s	Normal distribution	One-sample t-test	t(62) = 1.92	0.06	[0, 0.15]

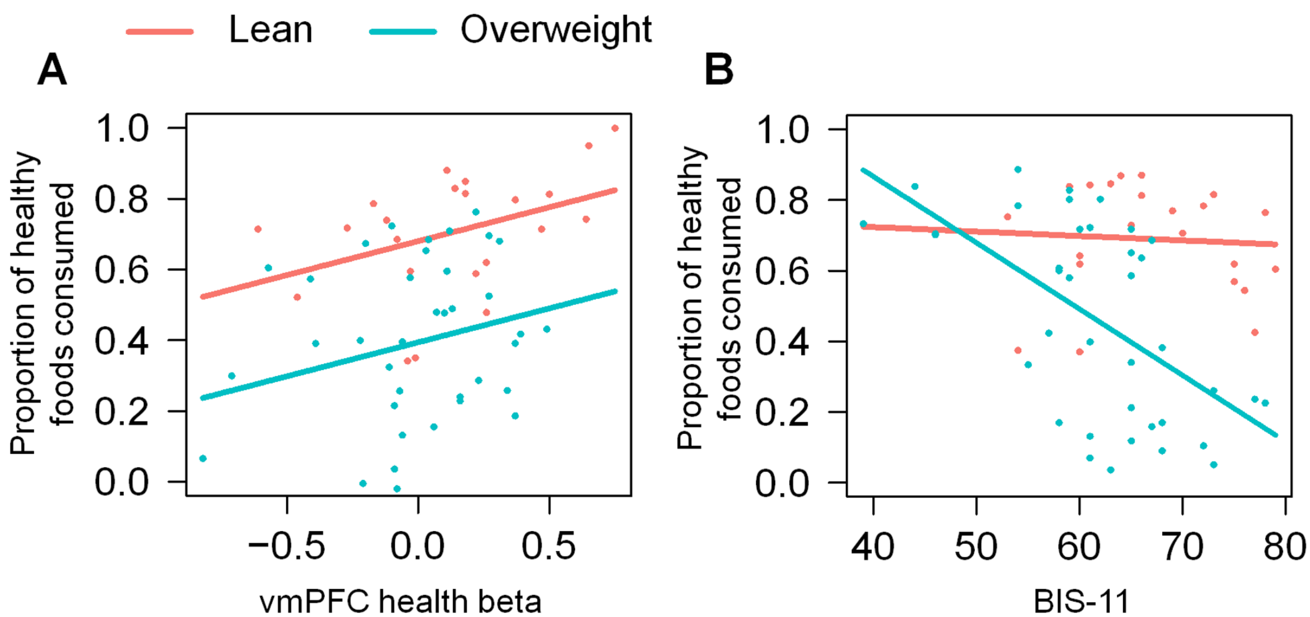
Test	Data structure	Type of test	Test statistic	p-value	[Confidence intervals]/Power
t: Main effect of Attribute	Normal distribution	Linear mixed-effects model	F(1,61) = 100.92	< 0.0001	1
t: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1,61) = 0.47	0.47	0.11
t: Attribute x Group interaction	Normal distribution	Linear mixed-effects model	F(1,61) = 0.01	0.94	0.05
u: Overweight - Lean	Normal distribution	Two-sample t-test	t(61) = - 0.39	0.69	[-0.13, 0.19]
v: Overweight - Lean	Normal distribution	Two-sample t-test	t(61) = -0.73	0.47	[-0.07, 0.15]
w: Main effect of Attribute	Normal distribution	Linear mixed-effects model	F(1,59) = 100.9	< 0.0001	1
w: Main effect of Group	Normal distribution	Linear mixed-effects model	F(1,59) = 0.5	0.47	0.11
w: Main effect of BIS-11	Normal distribution	Linear mixed-effects model	F(1,59) = 0.4	0.54	0.1
w: Attribute x Group interaction	Normal distribution	Linear mixed-effects model	F(1,59) = 0.01	0.94	0.05
w: Attribute x BIS-11 interaction	Normal distribution	Linear mixed-effects model	F(1,59) = 3.2	0.08	0.44
w: Group x BIS-11 interaction	Normal distribution	Linear mixed-effects model	F(1,59) = 0.2	0.65	0.07
w: Attribute x Group x BIS-11 interaction	Normal distribution	Linear mixed-effects model	F(1,59) = 0.2	0.67	0.07
x: Neural beta	Normal distribution	Linear model	t(1,59) = 2.24	0.03	[0.02, 0.43]
x: Overweight - Lean	Normal distribution	Linear model	t(1,59) = -3.24	0.002	[-0.35, -0.08]
y: BIS-11	Normal distribution	Linear model	t(1,55) = -0.21	0.83	[-0.01, 0.01]
y: Neural beta	Normal distribution	Linear model	t(1,55) = 2.21	0.03	[0.02, 0.36]

Test	Data structure	Type of test	Test statistic	p-value	[Confidence intervals]/Power
y: Overweight - Lean	Normal distribution	Linear model	$t(1,55) = -4.35$	< 0.0001	[-0.39, -0.15]
y: BIS-11 x (Overweight - Lean) interaction	Normal distribution	Linear model	$t(1,55) = -2.45$	0.02	[-0.03, 0]
z: Behavioural beta	Normal distribution	Linear model	$t(1,59) = 4.25$	< 0.0001	[0.2, 0.57]
z: Overweight - Lean	Normal distribution	Linear model	$t(1,59) = -3.9$	0.0003	[-0.36, -0.11]
α: BIS-11	Normal distribution	Linear model	$t(1,55) = 0.24$	0.81	[-0.01, 0.01]
α: Behavioural beta	Normal distribution	Linear model	$t(1,55) = 2.29$	0.03	[0.03, 0.43]
α: Overweight – Lean	Normal distribution	Linear model	$t(1,55) = -4.35$	< 0.0001	[-0.39, -0.15]
α: BIS-11 x (Overweight – Lean) interaction	Normal distribution	Linear model	$t(1,55) = -2.34$	0.02	[-0.03, 0]


A
9 -10am
Health and taste ratings for the scanner task
Neurocognitive measures and questionnaires
10:30 -12:30am
Scanner session
1 – 1:30pm
Buffet lunch
1:30 – 2pm
Health and taste ratings of the buffet foods
IQ test
B
Health rating
Health?
Health?
+ 48x
Reference
Taste rating
Taste?
Taste?
+ 48x
C
Free
Swap?
Yes
+
Forced
Please select No
No
+
Decision trial
8s
Feedback
1s
Blank
0.5s
Null (30x random)
6s


A
B
Proportion of Yes or Strong Yes
0.8
0.6
0.4
0.2
0.0
Group
Lean
Overweight
Unhealthy
Healthy
Unhealthy
Healthy
Nontasty
Tasty
Buffet consumption (g)
800
600
400
200
0
***
**
Unhealthy
Healthy
Unhealthy
Healthy
Nontasty
Tasty


A
y = 44
x = -8
6
4
2
0
B
vmPFC beta
0.4
0.2
0.0
Health
Taste



Lean
Overweight
A
B
Proportion of healthy foods consumed
1.0
0.8
0.6
0.4
0.2
0.0
−0.5
0.0
0.5
vmPFC health beta
40
50
60
70
80
BIS-11