# Reliance on model-based and model-free control in obesity

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# Abstract

Consuming more energy than is expended may reflect a failure of control over eating behaviour in obesity. Behavioural control arises from a balance between two dissociable strategies of reinforcement learning: model-free and model-based. We hypothesized that weight status relates to an imbalance in reliance on model-based and model-free control, and that it may do so in a linear or quadratic manner. To test this, 90 healthy participants in a wide BMI range (normal-weight (n=31), overweight (n=29), obese (n=30)) performed a sequential decision-making task. The primary analysis indicated that obese participants relied less on model-based control than overweight and normal-weight participants, with no difference between overweight and normal-weight participants. In line, secondary continuous analyses revealed a negative linear, but not quadratic, relationship between BMI and model-based control. Computational modelling of choice behaviour suggested that a mixture of both strategies was shifted towards less model-based control in obese participants. Furthermore, exploratory analyses of separate weights for model-free and model-based control showed stronger reliance on model-free control with increased BMI. Our findings suggest that obesity may indeed be related to an imbalance in behavioural control as expressed in a phenotype of less model-based control potentially resulting from enhanced reliance on model-free computations.

# Introduction

Obesity is the result of systematically consuming more energy than is expended. This can be seen as a failure of control over eating behaviour [1–3] and could result from altered processing of reward [4]. As a consequence, appetitive and often high-caloric foods are over-consumed despite negative consequences, such as the uncomfortable feeling of being full, feelings of regret, or long-term health risks. Such failures of behavioural control in obesity may arise from alterations in reinforcement learning [5]. Indeed, obesity-related impairments in reward- and punishment-based cue-conditioning have been observed in the context of both food and monetary outcomes [6], as well as impairments in appetitive conditioning in the context of chocolate rewards [7] (but see [8]). Furthermore, obese participants exhibited impairments in learning from negative outcomes when money or points served as an incentive [6,9,10]. These studies have focused on forms of learning that mostly resemble retrospective model-free 'trial-and-error' reinforcement learning. However, behavioural control arises from a balance between model-based and model-free control [11,12]. Model-based control relies on an internal model of the environment to enable forward planning. As a result, this system is flexible (but cognitively costly), allowing us to be goal-directed even when the environment changes, e.g. abrupt change in the current outcome value, changes. In contrast, the model-free system is cognitively inexpensive and fast (but inflexible) and is thought to underlie habitual control. To better understand this balance in obesity, the current study investigates relative reliance on model-based and model-free control of choice behaviour.

Indirect evidence links obesity to reduced model-based, or rather, goal-directed control. Previous outcome devaluation studies tapping into goal-directed and habitual control of food choices in obesity have shown a negative correlation between goal-directed control and degree of obesity in humans [13,14]. That is, the higher the BMI, the less participants adjusted their food choices after devaluation of one of the two choices. Behavioural adjustment after outcome devaluation of non-food rewards related positively to model-based, but not model-free control, in healthy human participants performing a two-step decision-making task [15–17] (but see [18]). Alterations in model-based vs. model-free control have been associated with behavioural inflexibility as observed in clinical populations such as metamphetamine addiction, obsessive compulsive disorder, and binge eating disorder [19,20], as well as in a general population sample reporting symptoms of the same disorders and of other eating disorders [21]. However, Voon et al. [19] did not find differences in model-based and model-free control between obese participants without binge eating disorder and non-obese control participants. The absence of an association between obesity and model-based or model-free control seems surprising, given the above-mentioned obesity-related performance differences in simple

reinforcement learning tasks and outcome devaluation tasks, resembling more model-free and model-based control, respectively.

We propose two reasons why the study by Voon et al. [19] might have lacked power to detect obesity-related group differences in model-based and model-free control. First, rather subtle behavioural alterations are to be expected in obese individuals that are physically healthy. With a relatively low contrast in body mass index (BMI) between the obese and non-obese group (BMI [kg/m$^2$]: obese: M=31.49, SD=3.6; non-obese: M=23.54, SD=2.9), and an average BMI for the obese group only slightly above the cutoff for obesity (>30 kg/m$^2$), such behavioural alterations may be difficult to detect. Second, the relationship between BMI and model-based and model-free control may in fact be quadratic in nature, thus masking potential obesity-related differences. A quadratic relationship with degree of obesity has indeed been observed for reward sensitivity [22] and cognitive restraint of eating behaviour [23]. Furthermore, obesity may quadratically relate to alterations in striatal dopamine tone [24]. This is relevant because there is accumulating evidence that different measures and manipulations of dopamine transmission overall relate positively to model-based control as measured in the two-step task [25–29].

In the current study, we aimed to address the two issues raised above by including (1) more highly obese individuals to boost the contrast between groups, and (2) an intermediate overweight group for more sensitivity to detect the existence of potential linear or quadratic relationships between weight status and behavioural control. The original two-step task was implemented to disentangle and directly compare the reliance on model-based and model-free control [16,25,30]. We hypothesized that weight status relates to the degree to which individuals rely on model-based and model-free learning, and that it may do so in a linear or quadratic manner.

# Materials and methods

## Participants

The results reported in this study are based on data from 90 healthy right-handed participants in a wide BMI range (45 women; age [years]: M=26.9; SD=3.6; range: 21-35; BMI [kg/m$^2$]: M=27.9, SD=6.4, range = 18.4 - 47.6). Participants were recruited based on their BMI status, i.e., normal-weight (n(women) = 31(16), BMI [kg/m$^2$] = 18.5-24.9), overweight (n(women) = 29(14), BMI [kg/m$^2$] = 25-29.9) and obese (n(women) = 30(15), BMI > 30)(**Table 1**). Note that the reported data were acquired in two parts. Fifty-seven datasets were acquired as a part of several studies running in the department between October 2012 and August 2014. Data acquisition of overweight and obese participants was not completed at the time due to logistic

reasons. To finally conclude the study, the remaining participants were tested between February and March 2018 (n=37, for details see **Supplemental Figure 1**). Part of the reported data have previously been published in a study comparing relative reliance on model-based and model-free control to habit propensity in a slips-of-action task in specifically normal-weight women and men (n=28) [16]. Participants were tested at the Department of Neurology of the Max Planck Institute for Human Cognitive and Brain Sciences (Leipzig, Germany) and received monetary compensation on an hourly basis, as well as a bonus based on their task performance (between 3 to 10 Euros). All participants gave written consent prior to the study. The study was carried out in accordance with the Declaration of Helsinki and approved by the Ethics Committee at the University of Leipzig, Germany.

After having provided informed consent, weight and height of the participants was measured, followed by the two-step task (for details see **Experimental paradigm**). Participants were then asked to complete a number of self-report questionnaires – validated in German – for characterizing the sample: Beck's Depression Inventory (BDI) [31] to assess possible depressive symptoms (cut-off for exclusion >18, indicating possibility of moderate to severe depression), the Behavioural Inhibition System / Behavioural Activation System questionnaire (BIS/BAS) [32,33] to assess punishment and reward sensitivity, the Three-Factor Eating Questionnaire (TFEQ)[34,35] to assess eating behaviour in terms of cognitive restraint, disinhibition and hunger, the UPPS Impulsive Behaviour Scale[36,37] to assess impulsive behaviour in terms of Urgency, lack of Premeditation, lack of Perseverance, and Sensation seeking, and the Yale Food Addiction Scale (YFAS) [38,39] to assess symptoms that could be indicative of food addiction. Finally, participants performed several cognitive tests to examine their potential relation to performance on the task: the Viennese Matrices Test (VMT) [40] to assess non-verbal IQ. We also administered a computerized version of the Visual Paired Associates test of the Wechsler Memory Scale (VPA) [41,42] to assess visual short term memory. Participants were included if none of the following exclusion criteria applied: estimated non-verbal IQ (<85 based on the VMT), known metabolic disorders (e.g., diabetes), smoking, (history of) neurological, psychiatric, or eating disorders, symptoms of depression, drug or alcohol dependence, current pregnancy, and psychological treatment. In total 94 participants were tested of which 3 participants did not complete the experimental paradigm and 1 participant was excluded from analysis because of an estimated non-verbal IQ below 85.

## Experimental paradigm

We administered a sequential decision making task [16,25,30], in which participants were asked to make two subsequent decisions on each trial to earn a monetary reward (20 cents) or no

reward (**Figure 1a**). At the first stage, participants were asked to choose between two grey stimuli, which would bring them to one of two second-stage stimulus pairs (the green or yellow pair). One of the grey first-stage stimuli was connected commonly (70%) to the green and rarely (30%) to the yellow stimulus pair, and vice versa for the other grey stimulus (**Figure 1b**). The first-stage stimuli and transition probabilities were fixed throughout the experiment. After selecting one of the two second-stage stimuli, participants either received the monetary reward or not (**Figure 1c**). The probability of receiving reward for each of the four second-stage stimuli changed slowly and continuously according to Gaussian random walks to ensure continuous learning. The changes were kept consistent for all participants performing the experiment. Participants completed a total of 201 trials. Prior to the experiment, participants went through elaborate computer-based instructions and were then asked to explain the task including its first-stage transition probabilities to the experimenter. Open questions were addressed by the experimenter. The instructions included a detailed knowledge of common (70%) and rare (30%) transitions after first-stage choices, and the slowly changing probabilities after second-stage choices. After the instructions participants performed 56 training trials with a different set of stimuli. Participants were made aware that the height of their financial bonus depended on the accumulated reward in the task.

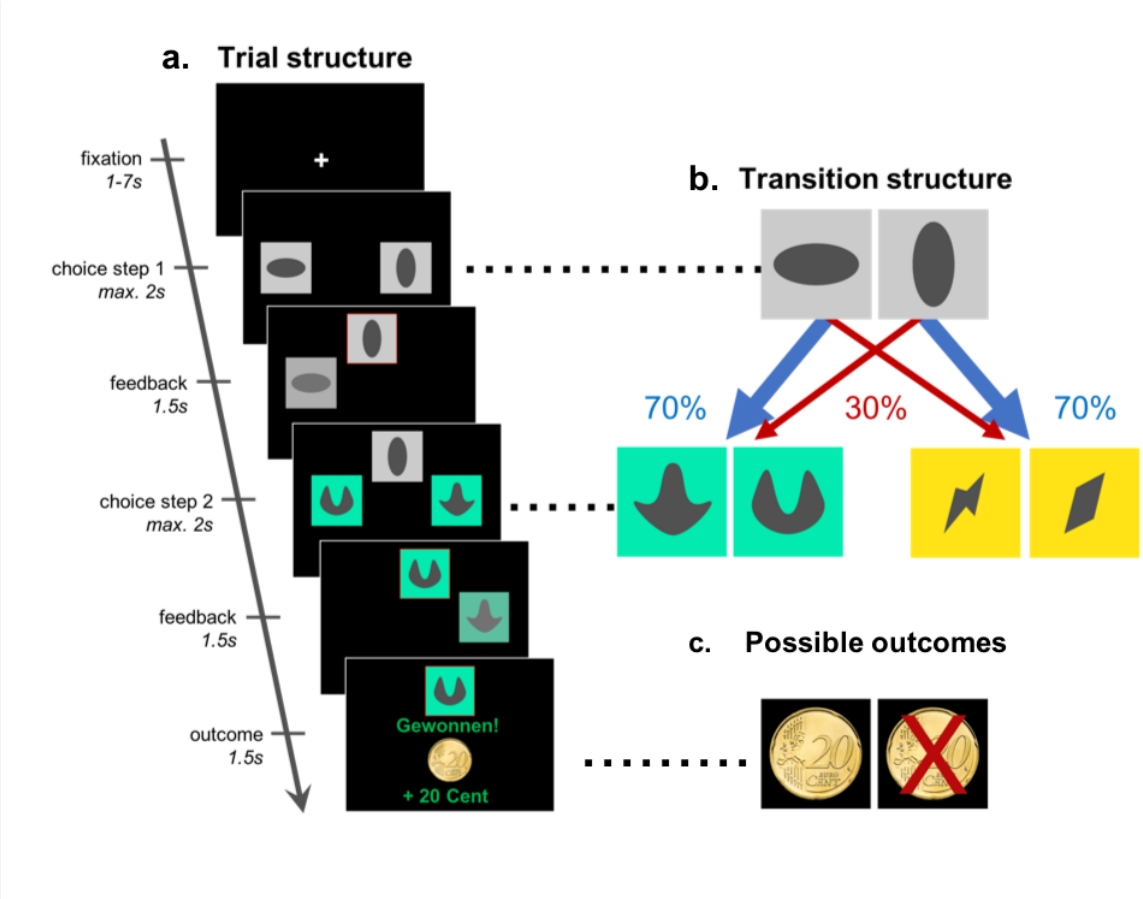



**Figure 1. The two-step task [25,30]. (a) Trial structure of an example trial with a rare transition, which allows for the dissociation of model-based and model-free control of**

**behaviour. (b) Transition structure showing how each first-stage stimulus (grey) leads to one of the two second-stage stimulus pairs (green or yellow) in 70% of the trials (common, blue arrows) and to the other pair in 30% of the trials (rare, red arrows). (c) Possible outcomes (reward, no reward). Reward probability for the four second-stage stimuli varies throughout the task according to random walks to encourage continuous learning.**

## Data analysis

Calculation of first-stage stay probabilities on the two-step task, as well as computational modeling of participants' choice behaviour were performed using in-house scripts in Matlab (version 2017b, The MathWorks, Inc.). Statistical analyses of self-reported, behavioural, and computational data were run in R Studio (version 3.4.4., R Core Team, 2018) and SPSS (version 24, IBM Corp., 2018). The R package ggplot2 was used to plot the results [45].

Shapiro-Wilk's test of normality and Levene's test of equality of variance were ran for all group characteristics, including scores on self-reported questionnaires and neuropsychological tests, as well as for the raw stay probabilities (per condition), and for the estimated model parameters.

The alpha level was set to .05 ($\alpha$ = .05) for all *a priori* analyses of interest. Note that for *post hoc* analyses, we did not correct for multiple comparisons as these results are exploratory and should be interpreted as such.

Partial $\eta^2$ ($\eta_p^2$) is reported as an effect size for all parametric univariate analyses because it meaningfully describes effects in a design in which multiple measures have been experimentally manipulated (as in the two-step task), and it yields very similar estimates as $\eta^2$ for analyses that only include a between-group variable [46,47]. Note that $\eta_p^2$ does not depend on the number of variables in the model and, thus, can be compared across studies. For non-parametric Kruskal-Wallis tests, $\eta^2_H$ was calculated as follows: (H – k +1) / (n – k), with *H* reflecting the test statistic, *k* the number of groups, and *n* the total sample size [48].

To check the robustness of our findings and rule out that any observed effect of group on behaviour could have been driven by age [21,49,50] or IQ [16,21,51,52] rather than weight status, we reran all models *post hoc* including age and non-verbal IQ as covariates of no interest.

### *Characterization of the groups*

We tested for group differences in age and sex to confirm that the groups were well-matched. BMI was analysed to confirm the grouping of participants into normal-weight, overweight and obese participants. Group analysis of cognitive tests (including non-verbal IQ) and self-reported questionnaire data were run to further characterize the sample.

For normally distributed data (age, VPA score, BIS/BAS, UPPS), we ran a one-way ANOVA with between-subjects factor weight group for each measure. Upon violation of the assumption of normality or equality of variance (BMI, non-verbal IQ, BDI, TFEQ, YFAS symptom score), the Kruskal-Wallis test by ranks was performed. Sex distribution between groups was analysed using Chi-Square Test. Group differences were followed up by *post hoc* parametric (independent T-test) or nonparametric (Mann-Whitney U Test) pairwise comparisons.

### *Raw behaviour according to first-stage stay probabilities*

Investigating the likelihood with which participants choose a first-stage stimulus depending on the previous trial type (Rewarded/Unrewarded, Common/Rare), gives an insight into how much they relied on model-based or model-free control. Therefore, we calculated first-stage stay probabilities as the proportion of trials in which participants chose the same first-stage stimulus as in the previous trial (coded as 'stay') for each of the conditions (Rewarded Common, Rewarded Rare, Unrewarded Common, Unrewarded Rare). We then analysed participants' stay probabilities using ANOVA with the between-subject factor Group (Normal-weight, Overweight, Obese), and within-subject factors Reward (Rewarded, Unrewarded) and Transition (Common, Rare). Because the aim was to test for a three-way interaction and the group sizes are well balanced, type III sums of squares were calculated in this analysis.

A purely model-free agent relies on whether or not the previous trial was rewarded, irrespective of transition probability (Common/Rare). If rewarded, the previous first-stage choice should be repeated. If not, it may be better for the model-free agent to switch to the other first-stage stimulus. As a consequence, model-free control is reflected in a main effect of Reward. On the other hand, a purely model-based agent optimally relies both on reward and transition probability of the previous trial. A model-based agent will also stay with a previous first-stage choice when a common trial was rewarded, and switch when a common trial was not rewarded. However, the model-based agent differs in choice behaviour following rare trials. That is, in contrast to a purely model-free agent, a model-based agent can infer that when a rare trial was rewarded, reward probability on the current trial is higher if one chooses the other first-stage stimulus (switch), and vice versa for unrewarded rare trials (stay). Model-based control is

therefore reflected in the interaction between Reward and Transition. Here, we were mainly interested in group differences in model-based and model-free control and thus focused on the Group x Reward x Transition interaction and Group x Reward interaction on stay probabilities, respectively.

We hypothesized that the relationship between weight status and model-based or model-free control might be linear or quadratic in nature. To investigate the nature of these relationships, we next performed planned pairwise group comparisons on the Reward x Transition interaction term (i.e., (Rewarded Common – Rewarded Rare) – (Unrewarded Common – Unrewarded Rare)) and on the main effect of Reward (i.e., (Rewarded Common + Rewarded Rare) – (Unrewarded Common + Unrewarded Rare)) on stay probabilities.

Finally, we ran two *post hoc* linear models (lm() from the R stats package): (1) on the Reward x Transition interaction term, and (2) on the main effect of Reward to investigate the existence of a linear and quadratic relationship with BMI on a continuous scale. Both models included BMI and $BMI^2$ as orthogonal predictors.

### *Computational modeling*

To investigate how participants' choices were affected by reward and transition probability throughout the experiment rather than in the previous trial alone, we computationally modeled choice behaviour. We implemented a hybrid of a model-free and model-based reinforcement algorithm as is described in detail in our previous work [16,25] and in the original paper [30].

In short, the model-free algorithm (SARSA($\lambda$)) included a learning rate for each stage ($\alpha_1$, $\alpha_2$) and a parameter $\lambda$, which allows the second stage prediction error to affect the next first-stage values (Q). The model-based algorithm learns values by planning forward and computes first-stage values by multiplying the value of the better second-stage option with the associated transition probabilities. Then, the model-free and model-based first-stage decision values are connected in the hybrid algorithm:

$$Q(s_A, a_j) = \omega\, Q_{MB}(s_A, a_j) + (1 - \omega)\, Q_{MF}(s_A, a_j)$$

where $Q(s_A, a_j)$ denotes the decision value of the chosen stimulus $a_j$ from the first stage stimulus pair $s_A$, and $\omega$ captures the relative weighting of the model-based ($Q_{MB}(s_A, a_j)$) and model-free algorithm ($Q_{MF}(s_A, a_j)$). The weighting parameter $\omega$ is the main parameter of interest and can take a value between 0 and 1. If $\omega$ = 1, first-stage choices are purely controlled

by model-based control, and if $\omega$ = 0, they are purely controlled by model-free control. Note that at the second stage $Qnet = Q_{MB} = Q_{MF}$ because reward probabilities are not fixed.

Finally, the decision values were transformed into action probabilities using the softmax function for $Qnet$:

$$P(a_{i,t} = a|s_{i,t}) = \frac{exp(\beta_i[\, Q_{net}(s_{i,t}, a) + \rho \cdot rep(a)])}{\sum_{a'} \exp\,(\beta_i[\, Q_{net}(s_{i,t}, a') + \rho \cdot rep(a')]}$$

where $\beta_i$ controls the stochasticity of choices at stage $i$ = 1 or 2, and repetition parameter $\rho$ reflects choice perseveration at the first stage.

The model had a total of seven parameters that were bounded by transforming them to a logistic $(\alpha_1, \alpha_2, \lambda, \omega)$ or exponential $(\beta_1, \beta_2)$ distribution. To infer the maximum-a-posteriori estimate of each parameter for each subject, the (empirical) Gaussian prior distribution was set to the maximum-likelihood estimates given the data of all participants and then expectation-maximization was used [53]. We report the negative log-likelihood (-LL) as a measure of model fit. Lower values reflect better model fit.

To assess reliance on model-based and model-free control over first-stage choices separately, we calculated $\beta_{MB}$ and $\beta_{MF}$ by multiplying the first-stage stochasticity parameter $\beta_1$ with weighting parameter $\omega$, such that $\beta_{MB} = \beta_1{}^* \omega$ and $\beta_{MF} = \beta_1{}^* (1 - \omega)$[16,54]. Note, the resulting parameters were not normally distributed. For the sake of completeness, we also inferred the equivalent version of the model with separate ß's for MF and MB directly from the data [54] instead of re-computing the ß's from $\omega$.

We assessed group differences in $\omega$ using ANOVA with between-group factor weight status, as well as in $\beta_{MB}$ and $\beta_{MF}$ using Kruskal-Wallis test by ranks. Planned pairwise comparisons were performed as part of the ANOVA or using Mann-Whitney U test as a nonparametric alternative. For each of these analyses, the alpha level was set at .05. Finally, we investigated the relationship between these performance measures and weight status on a continuous scale by running a *post hoc* linear regression model for each. Each model included BMI and BMI$^2$ as orthogonal predictors. The dependent variables in the three models were $\omega, \beta_{MB}$ and $\beta_{MF}$.

After having detected between-group differences on the model parameters' of interest, an important sanity check is whether the inferred parameters actually reproduce the observed behavioural data in terms of stay probabilities. To do so, we re-ran the model based on each individual's inferred parameters to generate data for each individual (1000 simulations per subject) and performed the original ANOVA.

# Results

## Characterization of the groups

**Table 1** summarizes the weight groups (normal-weight (NW), overweight (OW), and obese (OB)) in terms of age, sex and BMI, as well as in terms of their scores on the cognitive tests and self-report questionnaires. The groups were well matched on sex and age, and did not differ in visual short-term memory (VPA), or non-verbal IQ as measured on the Viennese Matrices Test (VMT). However, a trend-level group difference was observed for non-verbal IQ, with numerically higher IQ scores for the normal-weight and overweight relative to the obese group (**Table 1**). We did observe a group difference in the average number of depressive symptoms (*KW*(2) = 11.5, $p$ = .003, $\eta^2_H$ = .11) even though the scores are not clinically relevant in the current sample. This difference was driven by the obese participants having a higher symptom score relative to normal-weight, but not overweight, participants (*post hoc* pairwise comparisons: NW vs. OB, $p$ = .004; OW vs. OB, $p$ = .137; NW vs OW, $p$ = .254). The average number of food addiction symptoms also differed between the groups (KW(2) = 17.3, $p$ < .001, $\eta^2_H$ = .18), again, driven by a higher number of symptoms for obese relative to normal-weight, but not overweight, participants (*post hoc* pairwise comparisons: NW vs. OB, $p$ < .001; OW vs. OB, $p$ = .159; NW vs OW, $p$ = .242). In terms of self-reported eating behaviour (TFEQ) the groups differed in disinhibition (KW(2) = 16.9, $p$ < .001, $\eta^2_H$ = .17) and restraint (KW(2) = 7.2, $p$ = .027, $\eta^2_H$ = .06). Disinhibition scores were higher for obese relative to both normal-weight and overweight participants and somewhat higher for overweight relative to normal-weight participants (*post hoc* pairwise comparisons: NW vs. OB, $p$ < .001 ; OW vs. OB, $p$ = .010; NW vs OW, $p$ = .076). Restraint scores were highest for overweight participants and lower for normal-weight, but not obese participants (*post hoc* pairwise comparisons: NW vs. OB, $p$ < .375 ; OW vs. OB, $p$ = .374; NW vs OW, $p$ = .013). No other group differences were observed.

## Raw behaviour according to first-stage stay probabilities

Analysis of stay probabilities (**Figure 2a**) revealed that participants' first-stage choices were significantly affected by reward (main effect Reward: F(1,87) = 27.2, $p$ < .001, $\eta_p^2$ = .238) as well as by the combination of reward and transition probability (interaction Reward x Transition: F(1,87) = 183.4, $p$ < .001, $\eta_p^2$ = .678) on the previous trial. This is in line with previous research [25,30] and suggests that, across groups, the participants relied on both model-based and model-free choice strategies, respectively. Transition probability alone did not significantly affect participants' first-stage choices (Transition: F(1,87) = 3.4, $p$ = .070, $\eta_p^2$ = .037).

The weight groups significantly differed in the use of a model-based choice strategy (**Figure 2b**) as reflected by a significant three-way Group x Reward x Transition interaction on stay

probabilities ($F$ (2,87) = 4.3, $p$ = .017, $\eta_p^2$ = .090), but not in the use of a model-free choice strategy (Group x Reward: $F$ (2,87) = 1.8, $p$ = .174, $\eta_p^2$ = .039, **Figure 2c**). Planned comparisons of the Reward x Transition interaction between groups showed that the three-way interaction was driven by a significantly higher interaction term for normal-weight relative to obese ($p$ = .017) and for overweight relative to obese ($p$ = .010) participants, whereas normal-weight and overweight participants did not differ from each other ($p$ = .817).

We observed no Group x Transition interaction ($F$ (2,87) = 1.2, $p$ = .297, $\eta_p^2$ = .028), nor a main effect of Group ($F$ (2,87) = 1.7, $p$ = .187, $\eta_p^2$ = .038) on stay probabilities. These results suggest that choices of obese participants relied relatively less on model-based control than those of normal-weight and overweight participants.

**Table 1. Group characteristics displaying mean (standard deviation) and range if not otherwise stated, followed by the test-statistic and p-value of group comparison for each measure.**

| | Normal-weight | | | Overweight | | | Obese | | | *p* | *test-statistic* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| n | 31 | | | 29 | | | 30 | | | | |
| sex (F:M) | 16:15 | | | 14:15 | | | 15:15 | | | ns | $0.07^{\chi}$ |
| age | 26.9 | (3.3) | 21-34 | 26.0 | (3.7) | 21-35 | 27.8 | (3.8) | 22-34 | .166 | $1.8^{F}$ |
| BMI (kg/m$^2$) | 21.6 | (1.8) | 18.4-24.8 | 26.9 | (1.3) | 25.1-29.9 | 35.4 | (4.5) | 30.2-47.6 | <.001 | $79.1^{KW}$ |
| **Cognitive tests** | | | | | | | | | | | |
| Non-verbal IQ[$] | 119.9 | (12.1) | 95.0-136.5 | 117.3 | (10.6) | 93.0-136.5 | 111.2 | (16.4) | 85.0-136.5 | .084 | $5.0^{KW}$ |
| VPA score | 12 | (3.9) | 3-18 | 13.2 | (3.1) | 7-18 | 12.2 | (3.2) | 6-18 | .381 | $1.0^{F}$ |
| **Self-report questionnaires** | | | | | | | | | | | |
| BDI | 3.6 | (3.3) | 0-14 | 4.9 | (3.2) | 0-11 | 6.8 | (4.2) | 0-17 | .003 | $11.5^{KW}$ |
| BIS/BAS | | | | | | | | | | | |
| BIS | 20.0 | (3.4) | 14-28 | 19.8 | (4.3) | 11-27 | 19.5 | (4.0) | 7-27 | .884 | $0.1^{F}$ |
| BAS drive | 12.1 | (2.1) | 7-16 | 12.0 | (1.8) | 9-16 | 11.5 | (1.7) | 8-16 | .449 | $0.8^{F}$ |
| BAS fun | 12.1 | (1.8) | 9-16 | 12.0 | (1.8) | 8-15 | 12.0 | (1.9) | 8-16 | .972 | $0.03^{F}$ |
| BAS reward | 16.8 | (2.0) | 12-20 | 17.0 | (2.1) | 11-20 | 16.0 | (2.0) | 10-19 | .141 | $2.0^{F}$ |
| TFEQ | | | | | | | | | | | |
| Restraint | 5.0 | (3.2) | 0-15 | 8.1 | (4.6) | 0-18 | 6.4 | (4.7) | 0-18 | .027 | $7.2^{KW}$ |
| Disinhibition | 4.9 | (2.1) | 0-9 | 6.3 | (3.3) | 2-15 | 8.3 | (3.3) | 3-16 | <.001 | $16.9^{KW}$ |
| Hunger | 5.7 | (3.3) | 1-13 | 5.1 | (4.1) | 0-13 | 7.1 | (3.4) | 1-14 | .066 | $5.4^{KW}$ |
| UPPS | | | | | | | | | | | |
| Urgency | 26.8 | (5.8) | 15-42 | 25.4 | (5.1) | 17-36 | 27.7 | (6.6) | 13-39 | .314 | $1.2^{F}$ |
| (lack of) | 22.2 | (4.1) | 12-31 | 22.7 | (4.6) | 16-36 | 22.3 | (4.0) | 12-29 | .904 | $0.1^{F}$ |
| Premeditation (lack of) | 20.1 | (6.0) | 12-44 | 19.4 | (5.6) | 10-34 | 21.3 | (5.3) | 12-34 | .438 | $0.8^{F}$ |
| Perseverance | | | | | | | | | | | |
| Sensation seeking | 31.8 | (6.6) | 18-44 | 31.6 | (8.5) | 17-48 | 28.0 | (7.4) | 14-40 | .090 | $2.5^{F}$ |
| YFAS (#symptoms) | 0.8 | (0.7) | 0-2 | 1.3 | (1.4) | 0-7 | 1.9 | (1.0) | 0-4 | <.001 | $17.3^{KW}$ |

*Abbreviations: n = number of participants; F:M = the ratio of females to males; VPA = Visual Paired Associates test of the Wechsler Memory Scale; BDI = Beck's Depression Inventory; BIS/BAS = Behavioural Inhibition System / Behavioural Activation System; TFEQ = Three-Factor Eating Questionnaire; UPPS = Urgency, lack of Premeditation, lack of Perseverance, and Sensation seeking; YFAS = Yale Food Addiction Scale.*

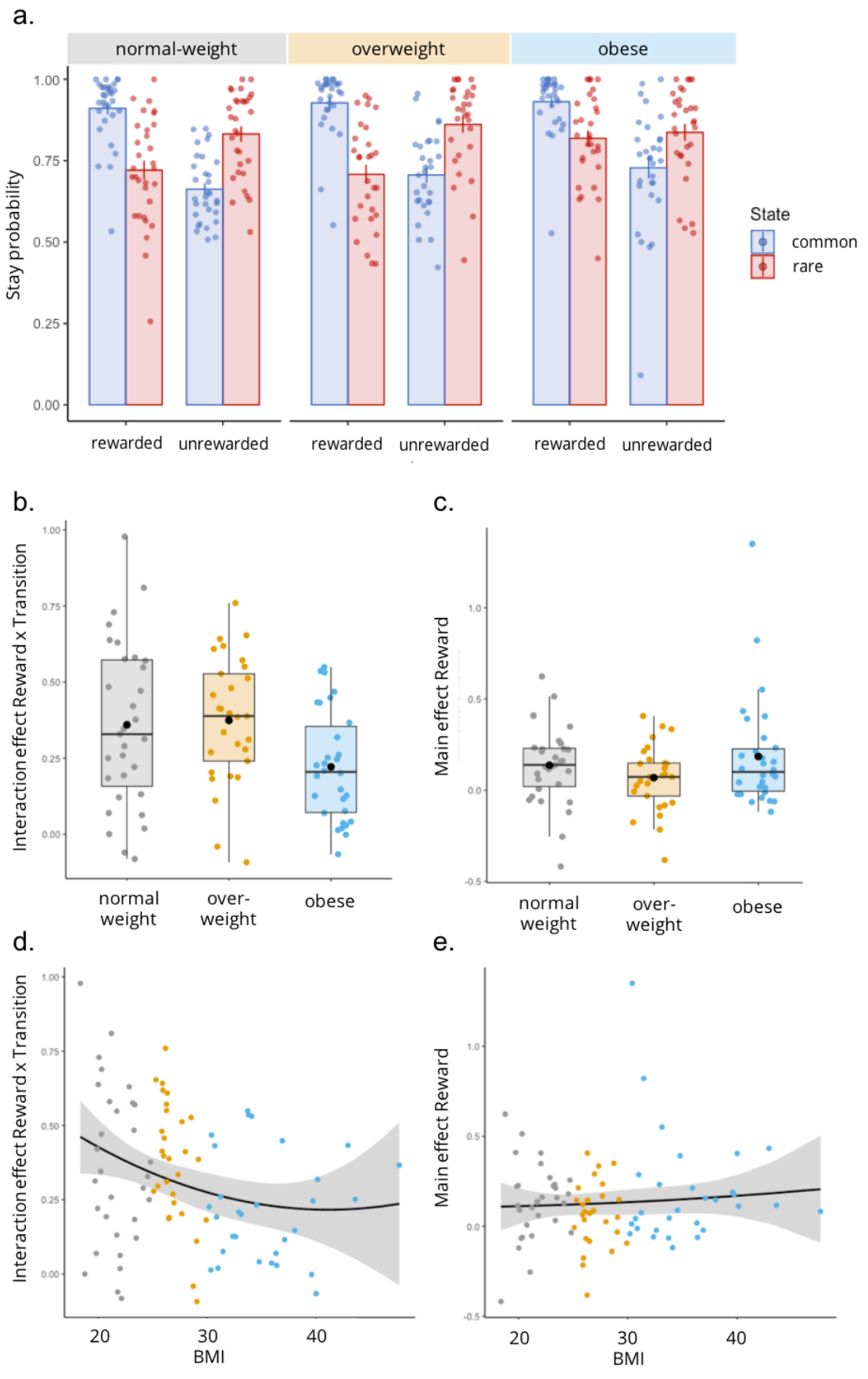
*[$]Non-verbal IQ was calculated based on the Viennese Matrices Test (VMT).*

*[χ] Chi square test for frequency data (degrees of freedom: 2).*

*[F] F-test with for normally distributed scores (degrees of freedom: 2,87).*
*[KW] Independent-Samples Kruskal-Wallis Test of distributions for non-normally distributed scores (degrees of freedom: 2).*

*Post hoc* simple effects analyses were performed to further investigate the three-way interaction on stay probabilities and revealed a striking difference between the groups. Interestingly, we observed a Group x Reward interaction for rare ($F(2,87) = 4.2$, $p = .018$), but not common trials ($F(2,87) < 1$, $p = .497$). This in turn was driven by a simple main effect of Group on stay probabilities following rewarded rare trials ($F(2,87) = 4.6$, $p = .012$), but not unrewarded rare trials ($F(2,87) < 1$, $p = .688$). The simple effect of Group was also reflected in a Group x Transition interaction for rewarded ($F$ (2,87) = 3.8, $p = .026$), but not unrewarded trials ($F$ (2,87) = 2.4, $p = .100$). Finally, pairwise group comparisons of rewarded rare trials showed that obese participants were more likely to stay with their previous first-stage choices when a rare trial had been rewarded relative to normal-weight ($t(59) = -2.5$ , $p = .014$) and overweight participants ($t(57) = -2.9$ , $p = .006$), with no difference between normal-weight and overweight participants ($t(58) = 0.3$, $p = .766$). This is of interest because it is participants' behaviour following rare trials that allows us to dissociate model-based from model-free control. Increased staying after a rare rewarded trial hints at more model-free control, even though this effect was not sufficiently strong to come out as a significant interaction between Group and Reward. Nevertheless, it seems that the observed group difference in model-based control may in fact be driven by enhanced reliance on model-free computations (see **Discussion** for more).

Next, we addressed the question if reliance on model-based and model-free control related to obesity in a linear and/or quadratic manner. Because the traditional weight categories of normal-weight, overweight and obese individuals reflect unequal intervals in terms of BMI, we turned to BMI as a continuous variable, even though the study was designed for group-based analyses. We ran two linear regression models including BMI and $BMI^2$ as orthogonal predictors in each, and investigated their relationship with the (1) Reward x Transition interaction term, and (2) the main effect of Reward on stay probabilities. BMI related negatively to the Reward x Transition interaction term ($\beta_{BMI} = -.28$, $p = .007$), but no additional quadratic relationship was observed ($\beta_{BMI^2} = .10$, $p = .319$)(**Figure 2d**). Together, BMI and $BMI^2$ explained a significant proportion of variance in the effect of Reward and Transition on choice strategy (adjusted $R^2 = .069$, $F(2,87) = 4.3$, $p = .017$). In line with the absence of a Group x Reward effect on stay probabilities, we did not observe a linear or quadratic relationship between BMI and the main effect of Reward on stay probabilities ($\beta_{BMI} = .08$, $p = .463$; $\beta_{BMI^2} = .01$, $p = .892$)(**Figure 2e**), nor did the model explain a significant proportion of variance ($R^2 = -.016$, $F$ (2,87) = 0.3, $p = .756$).


a.
normal-weight
overweight
obese
Stay probability
1.00
0.75
0.50
0.25
0.00
rewarded
unrewarded
State
common
rare
b.
Interaction effect Reward x Transition
normal weight
over-weight
obese
c.
Main effect Reward
d.
BMI
e.

396

**Figure 2. Stay probabilities. (a) Average stay probabilities per condition for each group. Error bars represent ±1 SEM. (b) On the group level, the use of a model-based choice strategy (i.e., the Reward x Transition interaction term) was lower for obese relative to normal-weight and overweight participants, whereas (c) the use of a model-free choice strategy (i.e., the main effect of Reward) did not differ significantly between groups. The box plots in (b) and (c) show the median and interquartile range for each group, with the black dot denoting the mean. (d) On the continuous level, the Reward x Transition interaction term was negatively related to BMI, with no additional significant quadratic relationship. (e) No linear or quadratic relationship was observed between BMI and the main effect of Reward. The scatter plots in (d) and (e) show the model fit (black line) and confidence interval (shaded) of the respective regression models with predictors BMI and BMI$^2$. Individual data points are color-coded based on weight group for illustrative purpose.**

## Computational modeling of choice behaviour

Computational modeling of behaviour allowed us to take into account participants' choices throughout the experiment rather than only considering the effect of the previous trial. For a summary of all parameters and group comparisons, see **Table 2**.

The parameter $\omega$ was of initial interest because it reflects participants' relative reliance on model-based vs. model-free control. A purely model-based agent has an $\omega$ of 1, whereas a purely model-free agent has an $\omega$ of 0. As expected, we observed a significant group effect on $\omega$ ($F$ (2,87) = 5.3, $p$ = .007, $\eta_p^2$ = .109)(**Figure 3a**). Planned comparisons showed that the group effect on $\omega$ was driven by higher values for normal-weight relative to obese ($t$(59) = 2.1, $p$ = .042) and overweight relative to obese participants ($t$(57) = 3.1, $p$ = .003). Although overweight participants numerically had the highest $\omega$ values, there was no statistical difference with normal-weight participants ($t$(58) = -1.1, $p$ = .265).

To investigate the nature of the relationship between $\omega$ and weight on a continuous scale (i.e., BMI), we again ran a *post hoc* regression model including the linear term BMI and quadratic term BMI$^2$ as predictors. The linear term related negatively to values of $\omega$ with lower values in individuals with a higher BMI ($\beta_{BMI}$ = -.23, $p$ = .030), whereas the quadratic term did not significantly add to the model ($\beta_{BMI}{}^2$ = -.005, $p$ = .964)(**Figure 3b**). In total, the model explained 3.1% of variance in $\omega$ (adjusted $R^2$ = .031, $F$ (2,87) = 2.4, p = .093), which reflects only a small effect of BMI on reliance on model-based vs. model-free control.

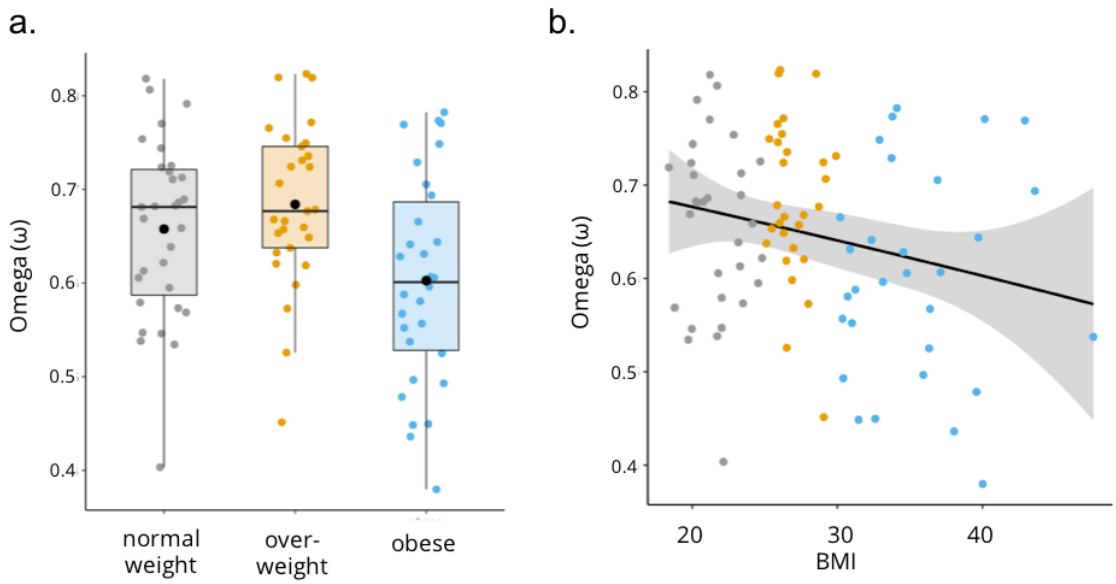



**Figure 3. Relative reliance on model-based and model-free control (omega). (a) On the group level, omega was significantly lower for obese relative to normal-weight and overweight participants. The box plot reflects the median, interquartile range, and mean value (black dot) for each weight group. (b) On the continuous level, omega was negatively related to BMI, with no additional significant quadratic relationship. The scatter plot shows the model fit (black line) and confidence interval (shaded) of the regression model. Individual data points are color-coded based on weight group for illustrative purposes.**

Next, we investigated the reliance on model-based and model-free control separately by deriving $\beta_{MB}$ and $\beta_{MF}$ from the model parameters $\beta_1$ and $\omega$. These ß's reflect the stochasticity with which participants made first-stage choices; a high (low) value reflects low (high) stochasticity and thus stronger (weaker) reliance on that type of control. Because the resulting ß's were not normally distributed, we performed non-parametric group analysis for each ß ($\beta_{MB}$ and $\beta_{MF}$). Surprisingly, no group difference was observed for $\beta_{MB}$ ($KW(2) = 1.7$, $p = .434$, $\eta^2_H < .001$)(**Figure 4a**), nor for $\beta_{MF}$ ($KW(2) = 3.9$, $p = .144$, $\eta^2_H = .02$)(**Figure 4b**). However, continuous analyses revealed a significant positive linear, but not quadratic, relationship between $\beta_{MF}$ and BMI ($\beta_{BMI} = .247$, $p = .019$; $\beta_{BMI}{}^2 = -.133$, $p = .199$; adjusted $R^2 = .06$, $F(2,87) = 3.7$, $p = .028$)(**Figure 4d**), whereas no significant relationship between $\beta_{MB}$ and BMI was observed ($\beta_{BMI} = -.008$, $p = .939$; $\beta_{BMI}{}^2 = -.147$, $p = .169$; adjusted $R^2 = -.0008$, $F(2,87) < 1$, $p = .386$)(**Figure 4c**).

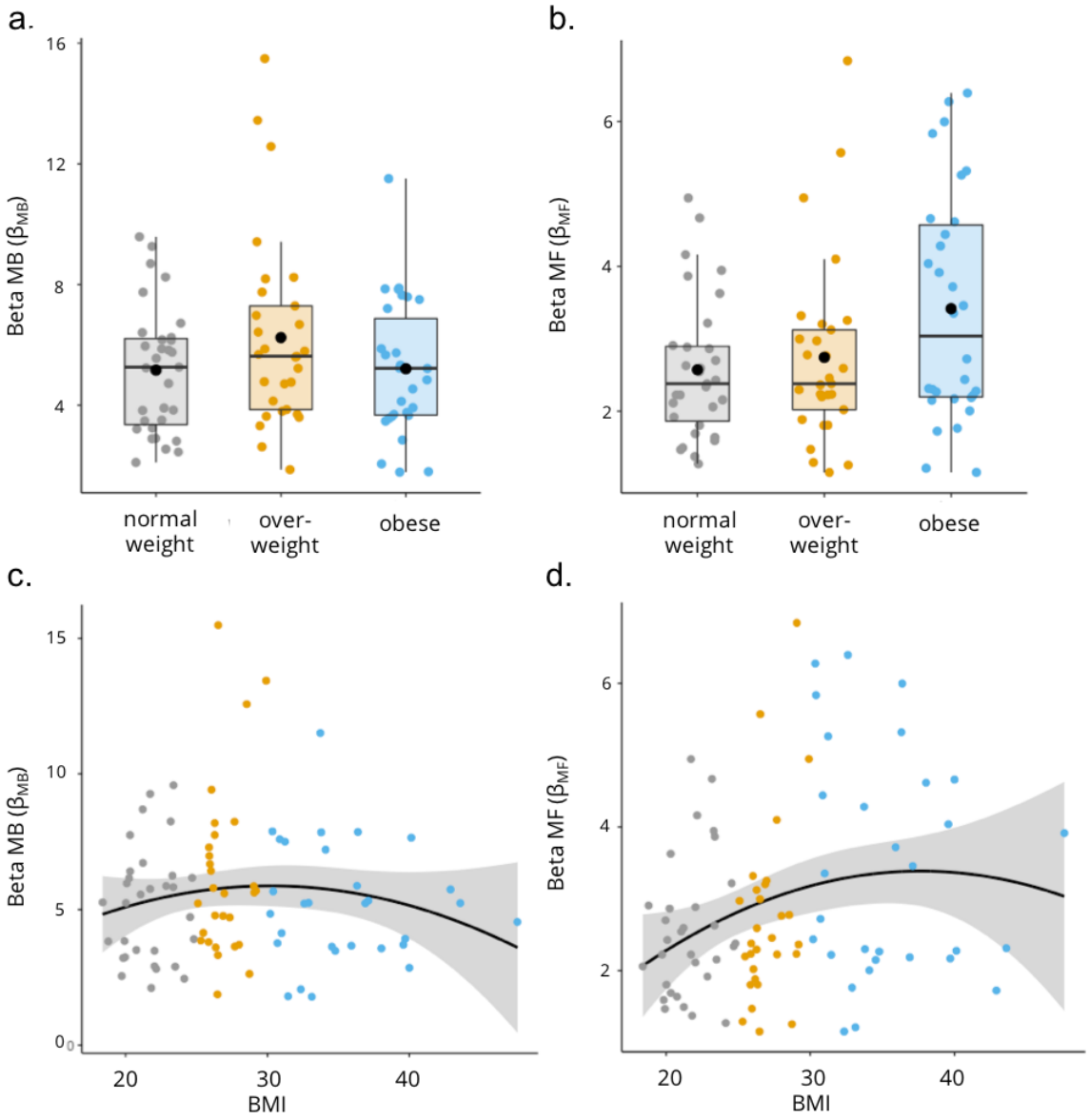



**Figure 4. Stochasticity of model-based and model-free choices (model 1). (a) No group difference was observed for $\beta_{MB}$ nor (b) $\beta_{MF}$. The box plots in (a) and (b) reflect the median, interquartile range, and mean value (black dot) for each weight group. (c) On the continuous level, no linear or quadratic relationship was observed between BMI and $\beta_{MB}$. (d) A positive linear relationship was observed between BMI and $\beta_{MF}$ reflecting reduced stochasticity of model-free choices with higher BMI. The scatter plots in (c) and (d) show the model fit (black line) and confidence interval (shaded) of the regression models. Individual data points are color-coded based on weight group for illustrative purposes.**

For the sake of completeness, we also inferred the equivalent version of the model with separate ß's for MF and MB directly from the data [54] instead of re-computing the ß's from $\omega$. The model was otherwise identical to the original model including $\omega$ (**Supplemental Table 1**) and yielded highly similar results as described in the previous paragraph. There was a slight group difference in $\beta_{MB}$ *at trend level* (KW(2) = 5.4, p = .067, $\eta^2_H$ = .04), but no difference in $\beta_{MF}$ (KW(2) = 3.6, p = .165, $\eta^2_H$ = .02). On a continuous level, $\beta_{MF}$ was again linearly related to BMI ($\beta_{BMI}$ = .249, *p* = .018; $\beta_{BMI}{}^2$ = -.072, *p* = .492; adjusted $R^2$ = .046, *F* (2,87) = 3.1, *p* = .046), and no relationship was observed between $\beta_{MB}$ and BMI ($\beta_{BMI}$ = -.084, *p* = .432; $\beta_{BMI}{}^2$ = -.077, *p* = .470; adjusted $R^2$ = -.010, *F* (2,87) < 1, *p* = .565)(**Supplemental Figure 2**).

None of the other model parameters differed significantly between the groups (model 1: **Table 2;** model 2: **Supplemental Table 1**). This indicates that the groups did not differ in terms of first or second stage learning rates ($\alpha_1$, $\alpha_2$), stochasticity of first or second stage choices ($\beta_1$, $\beta_2$), the tendency to persevere independent of reward or transition ($\rho$), the eligibility parameter ($\lambda$), and importantly, how well the model fit participants' data (-LL).

**Table 2. Summary and group comparisons of all model parameters**

| Para-meter | Group | Mean | (SD) | Quantiles 25% | 50% | 75% | test-statistic | p |
|---|---|---|---|---|---|---|---|---|
| $\omega$ | NW | 0.66 | (0.09) | 0.58 | 0.68 | 0.72 | | |
| | OW | 0.68 | (0.09) | 0.63 | 0.68 | 0.75 | $5.3^F$ | **.007** |
| | OB | 0.60 | (0.11) | 0.52 | 0.60 | 0.70 | | |
| $\alpha_1$ | NW | 0.47 | (0.17) | 0.33 | 0.48 | 0.63 | | |
| | OW | 0.47 | (0.21) | 0.34 | 0.55 | 0.65 | $<1^{KW}$ | .867 |
| | OB | 0.46 | (0.29) | 0.23 | 0.42 | 0.73 | | |
| $\alpha_2$ | NW | 0.54 | (0.23) | 0.41 | 0.62 | 0.70 | | |
| | OW | 0.56 | (0.19) | 0.47 | 0.56 | 0.68 | $<1^F$ | .560 |
| | OB | 0.50 | (0.21) | 0.35 | 0.50 | 0.70 | | |
| $\beta_1$ | NW | 7.7 | (2.6) | 5.3 | 7.8 | 9.0 | | |
| | OW | 9.0 | (4.0) | 6.3 | 8.1 | 10.7 | $1.6^{KW}$ | .447 |
| | OB | 8.6 | (3.2) | 6.9 | 7.6 | 11.3 | | |
| $\beta_2$ | NW | 4.3 | (1.7) | 2.8 | 4.1 | 5.4 | | |
| | OW | 4.0 | (1.2) | 3.2 | 4.2 | 4.8 | $<1^{KW}$ | .955 |
| | OB | 5.2 | (3.7) | 3.3 | 4.0 | 5.4 | | |
| $\lambda$ | NW | 0.53 | (0.23) | 0.36 | 0.54 | 0.71 | | |
| | OW | 0.53 | (0.18) | 0.38 | 0.56 | 0.69 | $<1^F$ | .967 |
| | OB | 0.55 | (0.22) | 0.41 | 0.55 | 0.69 | | |
| $\rho$ | NW | 0.14 | (0.05) | 0.10 | 0.14 | 0.18 | | |
| | OW | 0.14 | (0.04) | 0.11 | 0.13 | 0.16 | $3.0^{F,\#}$ | .057 |
| | OB | 0.15 | (0.06) | 0.13 | 0.16 | 0.18 | | |
| -LL | NW | 175.7 | (40.9) | 141.1 | 174.0 | 207.6 | | |
| | OW | 169.8 | (44.8) | 137.8 | 157.4 | 188.6 | $1.5^F$ | .225 |
| | OB | 155.7 | (50.7) | 128.3 | 157.9 | 193.3 | | |

*NW = normal-weight*
*OW = overweight*
*OB = obese*
*[F] F-test for normally distributed parameters (degrees of freedom: 2,87).*
*[KW] Independent-Samples Kruskal-Wallis Test of distributions for non-normally distributed parameters (degrees of freedom: 2). [#]One formal outlier was observed in the obese group and excluded from the analysis of this parameter (degrees of freedom: 2,86).*

Finally, we ran simulation recovery analyses for both models to assess whether the model parameters captured the observed behavioural data. Based on the estimated parameters, we simulated choice behaviour on the task and investigated stay probabilities. For both models, the reported significant Group x Reward x Transition interaction was fully reproduced indicating that the model captured important aspects of the data.

### Correcting for age and IQ

To check the robustness of our findings and rule out that the observed group differences could be explained by age [21,49,50] or IQ [16,21,51,52] rather than weight status, we reran all models *post hoc* including age and non-verbal IQ as covariates of no interest. In case of nonparametric tests, the analyses were performed after having regressed out age and non-verbal IQ from the dependent variables using linear regression.

Adding the covariates did not change the results qualitatively - the outcomes were largely in line with the original analyses and suggest that weight status, over and above age and IQ, explains unique variance in the degree to which individuals rely on measures of model-based, and possibly model-free, control (see **Supplemental Table 2** for a graphical overview of the outcomes of all analyses of interest). Notably, the reported group differences in model-based control, as observed in stay probabilities, and the relative reliance on model-based and model-free control, as reflected in the model parameter $\omega$, were relatively robust when correcting for age and non-verbal IQ. However, the pairwise comparison in model-based control between normal-weight and obese participants did not reach significance. Furthermore, on the continuous level we observed a similar negative relationship between BMI and model-based control (stay probabilities) and again a positive relationship between BMI and model-free control ($\beta_{MF}$) for both computational models (see **Supplemental Materials** for statistics).

## Discussion

The aim of this study was to investigate the relationship between weight status (i.e., normal-weight, overweight, and obese) and reliance on model-based and model-free control in the two-step task [16,25,30]. Our results indicate that obese participants relied less strongly on model-based control than overweight and – to a lesser extent – normal-weight participants, with no difference in performance between overweight and normal-weight participants. This was observed in group analysis of participants' choice behaviour (i.e., stay probabilities), as well as in the continuous analysis where BMI negatively related to model-based choice behaviour.

No quadratic relationship with BMI was observed. Furthermore, computational modeling of participants' choices revealed a similar group difference in the weighting of model-based and model-free control (i.e., $\omega$) that was driven by less model-based (vs. model-free) control for obese relative to overweight and normal-weight participants. Secondary continuous analyses of the randomness of participants' choices, captured by the model parameters $ß_{MB}$ and $ß_{MF}$, instead revealed a positive linear relationship with model-free, not model-based, control. This relationship was not observed at the group level.

Although seemingly contradictory, together these findings may in fact suggest that the observed obesity-related difference in model-based control is driven, in part, by enhanced reliance on model-free computations. This interpretation concurs with our *post hoc* simple effects analyses of stay probabilities, which revealed that the group difference in model-based control was driven by an increased inclination of obese (relative to normal-weight) to stay with their choice specifically after trials on which a rare transition led to reward. Rare trials are the trials of interest in this task, because performance following rare trials is used to dissociate model-based from model-free choices. Common trials on the other hand lead to the same decision in model-based and model-free agents. The group difference was only observed for rewarded, not unrewarded rare trials. We speculate that obese individuals may more easily fall back on model-free control, or in other words be more reactive after having been rewarded than normal-weight participants, whilst relying similarly on model-based control in the case of no reward. The current task is not designed to address this subtle effect, which could explain why it was not reflected in a group difference in model-free control in the analysis of stay probabilities as well as of $ß_{MF}$.

Our findings are in contrast to those of a previous study by Voon et al. [19] using the same paradigm. When comparing non-obese controls and obese participants with and without binge-eating disorder, Voon et al. [19] reported no difference in the weighting parameter $\omega$ between obese participants *without* binge-eating disorder and non-obese controls, whereas $\omega$ was on average lower for obese participants *with* binge-eating disorder relative to matched non-obese controls. Interestingly, our findings in healthy obese participants better match the previous findings in obese participants *with* binge-eating disorder. It should be noted however that $\omega$, and thus the reliance on model-based over model-free control, was much higher in the current study (mean (SD) omega: 0.6 (0.11) vs. 0.3 (0.24), range = 0-1). The discrepancy between the studies can be explained by several factors. First, the current study tested a more severely obese group than the Voon-study with a mean BMI of 35.4 kg/m$^2$ (SD: 4.5) vs. 31.5 kg/m$^2$ (SD: 3.6). In fact, in terms of BMI our sample was closer to the binge-eating group (mean BMI[kg/m$^2$]: 35.0, SD: 5.6). It may thus be the case that the reported finding of a lower weighting parameter $\omega$ in binge-eating disorder in the Voon-study can partially be explained

by the severity of obesity. Alternatively, the obese participants in our sample might unbeknownst fulfill criteria for binge-eating disorder, as we did not conduct a full psychiatric screening. Second, we included an intermediate weight group for increased sensitivity to detect group differences and potential quadratic effects that might otherwise remain uncovered. The group difference in model-based control in the current study was indeed mostly driven by the difference between overweight and obese participants. We therefore recommend that cognitive studies of obesity should include a wide BMI range, preferably also sampling severe to morbid obesity to assess for quadratic relationships, and to carefully disentangle between contributions of weight status and compulsive measures such as binge-eating symptoms.

The observed difference in reliance on model-based control in obesity generally concurs with previous outcome devaluation studies in relation to obesity that found reduced goal-directed control [13,14]. Goal-directed and model-based control are often equated [11] and have been found to relate, albeit weakly [15–17]. However, the concepts measured in the two types of tasks do not reflect the exact same constructs. Whereas the two-step task is designed to dissociate model-based and model-free control, it is difficult to disentangle reliance on goal-directed and habitual control in outcome devaluation paradigms in humans. Goal-directed and habitual control are thought to be organized hierarchically rather than in parallel. That is, the goal-directed system may benefit from habits in goal-pursuit and thus rely on the habit system [55], and the habit system may affect what goals are selected and pursued by the goal-directed system [56]. Empirical evidence for the existence of such hierarchies comes from a new generation of sequential decision-making tasks [57–59]. It will be relevant for future studies to focus on habitual goal-selection in the context of obesity, as has been suggested for addiction and other disorders of compulsivity [56], and investigate if it relates more closely to maladaptive eating behaviour in daily life.

The current study has several limitations. First, the dataset was collected in two parts with a sampling bias in terms of group and sex (see **Supplemental Figure 1**). Due to this bias we could not meaningfully account for sex and sample (2012-2014 vs. 2018) as covariates of no interest, because variance explained by sample and weight group or sample and sex cannot be disentangled in our design [60]. However, the task was identical in both sampling periods and administered in very similar lab spaces within the department. More importantly, extensive computerized instructions were implemented to minimize variability in performance due to differences in instructions between experimenters. We are therefore fairly confident that the observed group differences in model-based and model-free control in the task are not confounded by sampling period. Second, as emphasized above, the observed group differences are subtle with modest effect sizes and await replication. We speculate that these

differences may be more pronounced when taking into account participants' diet rather than obesity. Rodent studies suggest that rather than obesity, the intake of high fat and/or sugar diets may better predict alterations in dopamine-transmission [61–66]. We expect these changes to be at the heart of the maladaptive behavioural control in obesity [24] and there is accumulating evidence that different measures and manipulations of dopamine transmission overall related positively to model-based control as measured in the two-step task [25–29]. Whether diet rather than obesity relates to maladaptive behavioural control needs to be addressed in further studies. A third limitation is that, although the continuous analyses converge with the observed group differences in model-based control and strengthens the conclusion that obesity is indeed associated with altered reliance on model-based vs. model-free control, the design of the current study was not optimal for this type of analysis. BMI was not equidistributed across the complete sample due to the group-based recruitment-strategy. Hence, the current study might have been underpowered to robustly show true effects between BMI and behavioural control strategies on a continuous level. In particular the linear relationship between BMI and model-free control needs to be interpreted with care, as we did not observe this effect in the group-based analysis. Despite these limitations, the findings from our two independent analysis approaches did converge. That is, analysis of raw choice behaviour in terms of stay probabilities and of parameters from the computational modeling ($\omega$, $ß_{MB}$, $ß_{MF}$) both point to alterations in the reliance on model-based vs. model-free control in obesity. Simulation recovery analysis of the parameter estimates of the computational models further strengthened our confidence in the observed findings, because it recovered the observed three-way interaction between group, reward and transition probability on stay probabilities.

In conclusion, we found evidence for a relationship between the degree of obesity and reliance on model-based and model-free control relative to overweight and normal-weight participants, which was linear rather than quadratic in nature. Obesity was associated with relatively lower model-based control compared to normal-weight and overweight. The estimates of model-free control from the computational modeling approach were consistently higher with increased BMI. Together, these findings suggest that it is a combination of decreased model-based and increased model-free control in this task that characterizes the obese group. Whether or not the observed effects are dopamine-mediated, as hypothesized, remains an open question that warrants further investigation, for example, by pharmacologically manipulating dopamine transmission, or investigating the interaction between BMI and individual differences in dopamine transmission in terms of genetic or epigenetic variation.

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## Author contributions statement

AH, LD, and FS, designed the experiment. FM and LJ set up the study. FM collected data. LJ, LD, and FM analysed the data. LJ, LD, and AH wrote the manuscript. All authors read and approved the manuscript.

## Competing interests

The author(s) declare no competing interests.

## Data availability

The datasets analysed during the current study are available from the corresponding author on reasonable request.

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# Supplemental Materials

Reliance on model-based and model-free control in obesity

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**Supplemental Table 1. Summary and group comparisons of all parameters of model 2**

| Para-meter | Groups | Mean | (SD) | Quantiles 25% | 50% | 75% | test-statistic | p |
|---|---|---|---|---|---|---|---|---|
| $\alpha_1$ | NW | 1.5 | (2.5) | 0.54 | 0.80 | 1.3 | | |
| | OW | 0.62 | (2.5) | 0.48 | 0.80 | 1.5 | $<1.0^{KW}$ | .279 |
| | OB | 6.1 | (26.9) | 0.26 | 0.54 | 1.1 | | |
| $\alpha_2$ | NW | 1.6 | (6.3) | 0.32 | 1.0 | 3.9 | | |
| | OW | 0.90 | (2.4) | 0.40 | 1.2 | 1.7 | $<1.0^{KW}$ | .640 |
| | OB | -1.1 | (8.3) | -0.12 | 0.65 | 1.1 | | |
| $\beta_{MB}$ | NW | 5.0 | (2.5) | 2.7 | 4.3 | 6.9 | | |
| | OW | 5.9 | (3.0) | 3.8 | 5.3 | 7.8 | $5.4^{KW}$ | .067 |
| | OB | 4.4 | (2.3) | 2.8 | 3.9 | 5.3 | | |
| $\beta_{MF}$ | NW | 2.5 | (0.74) | 1.9 | 2.3 | 2.8 | | |
| | OW | 2.5 | (0.95) | 2.0 | 2.3 | 2.8 | $3.6^{KW}$ | .165 |
| | OB | 3.1 | (1.3) | 2.1 | 2.8 | 3.9 | | |
| $\beta_2$ | NW | 4.3 | (1.7) | 2.9 | 4.2 | 5.3 | | |
| | OW | 4.1 | (1.2) | 3.1 | 4.2 | 4.9 | $<1.0^{KW}$ | .979 |
| | OB | 5.2 | (3.8) | 3.2 | 4.0 | 5.4 | | |
| $\lambda$ | NW | 1.4 | (12.7) | 0.38 | 0.73 | 1.7 | | |
| | OW | 2.0 | (4.7) | 0.53 | 0.75 | 3.2 | $<1.0^{KW}$ | .990 |
| | OB | -1.9 | (13.3) | 0.39 | 0.80 | 1.7 | | |
| $\rho$ | NW | 1.0 | (0.44) | 0.70 | 0.97 | 1.4 | | |
| | OW | 1.2 | (0.56) | 0.89 | 1.2 | 1.4 | $5.3^{KW}$ | .069 |
| | OB | 1.2 | (0.66) | 0.88 | 1.4 | 1.6 | | |
| -LL | NW | 175.5 | (40.9) | 139.7 | 173.5 | 207.3 | $2.3^{KW}$ | .316 |
| | OW | 166.5 | (41.4) | 137.5 | 157.4 | 188.6 | | |
| | OB | 155.6 | (50.5) | 128.8 | 157.4 | 192.8 | | |

*NW = normal-weight*
*OW = overweight*
*OB = obese*
$^{KW}$ *Independent-Samples Kruskal-Wallis Test of distributions for non-normally distributed parameters (degrees of freedom: 2).*

**Supplemental Table 2. Overview of the outcomes of the analyses of interest. The study was designed primarily for group-based analysis (left column), but also permitted secondary continuous analysis of BMI (right column). The colors highlight whether the measure reflects model-based (blue) or model-free control (yellow), or relative reliance on model-based and model-free control (green). For each measure, the original analysis is reported (bright shade) as well as the covariate analysis with covariates age and non-verbal IQ (light shade).**

| | | Group-based (primary) | | Continuous (secondary) | |
|---|---|---|---|---|---|
| **Behavioral** | | | | | |
| Stay probabilities | | | | | |
| Interaction Reward x Transition | MB | ANOVA | ✓ OB<[OW,NW] | BMI & BMI$^2$ | ✓ negative linear |
| | | with covariates | ✓^ OB<[OW,NW^] | with covariates | ✓ negative linear |
| Main effect Reward | MF | ANOVA | X | BMI & BMI$^2$ | X |
| | | with covariates | X | with covariates | X |
| **Computational modelling** | | | | | |
| Model 1 | | | | | |
| $\omega$ | MB vs. MF | ANOVA | ✓ OB<[OW,NW] | BMI & BMI$^2$ | ✓ negative linear |
| | | with covariates | ✓ OB<OW | with covariates | X |
| $\beta_{MB}$ | MB | Kruskal-Wallis | X | BMI & BMI$^2$ | X |
| | | Covariates regressed out | X | with covariates | ✓^ negative quadratic |
| $\beta_{MF}$ | MF | Kruskal-Wallis | X | BMI & BMI$^2$ | ✓ positive linear |
| | | Covariates regressed out | ✓^ OB>NW | with covariates | ✓ positive linear |
| Model 2 | | | | | |
| $\beta_{MB}$ | MB | Kruskal-Wallis | ✓^ | BMI & BMI$^2$ | X |
| | | Covariates regressed out | X | with covariates | X |
| $\beta_{MF}$ | MF | Kruskal-Wallis | X | BMI & BMI$^2$ | ✓ positive linear |
| | | Covariates regressed out | X | with covariates | ✓ positive linear |

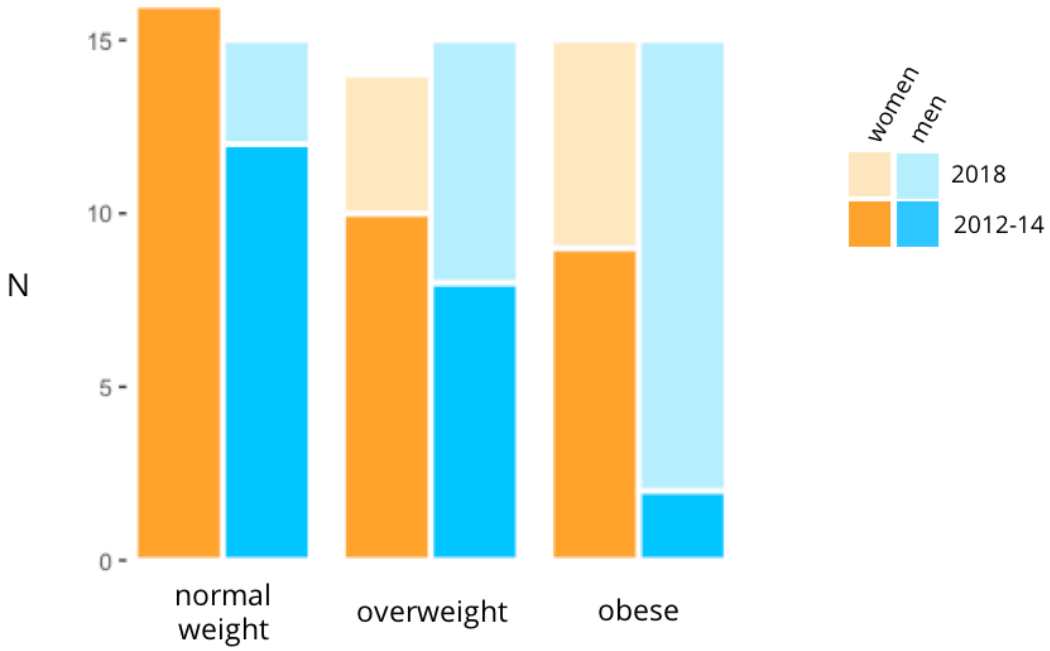
*MB = model-based*
*MF = model-free*
*NW = normal-weight*
*OW = overweight*
*OB = obese*
*✓ = a statistical difference is observed*
*X = no statistical difference is observed*
*^ observed difference at trend level*

### *Post hoc* covariate analysis

See **Supplemental Table 2** for a graphical overview of the outcomes of all analyses of interest. We found that the reported group differences in model-based control as observed in stay probabilities and the relative reliance on model-based and model-free control as reflected in the model parameter $\omega$ were robust when correcting for age and non-verbal IQ. That is, we still observed a Group x Reward x Transition interaction at trend level ($F$ (2,85) = 2.6, $p$ = .080, $\eta_p^2$ = .058), that was driven by a larger interaction term for overweight relative to obese participants ($p$ = .048), and a similar trend for normal-weight relative to obese participants ($p$ = .052). The three-way interaction was again complemented by the continuous analysis, which showed a negative linear, but no quadratic relationship between BMI and the Reward x Transition interaction term ($\beta_{BMI}$ = -.533, $p$ = .018, $\beta_{BMI}^2$ = .143, $p$ = .512, $R^2$ = .141, $F$(4,85) = 4.7, $p$ = .002). Also the absence of a group difference on model-free control in terms of stay probabilities was unaltered, as no Group x Reward interaction was observed ($F$ (2,85) = 2.3, $p$ = .121, $\eta_p^2$ = .048), nor a significant relationship between BMI and the main effect of reward in continuous analysis ($\beta_{BMI}$ = .083, $p$ = .456; $\beta_{BMI}^2$ = .006, $p$ = .956, $R^2$ = -.030, $F$ (4,85) = 0.3, $p$ = .844). Furthermore, the group difference in $\omega$ was still significant ($F$(2,85) = 3.3, $p$ = .044, $\eta_p^2$ = .071) and was driven by lower reliance on model-based vs. model-free control for obese relative to overweight individuals ($p$ = .013). In contrast to the original analysis, no significant difference was observed between obese and normal-weight participants ($p$ = .119). On the continuous level, the linear relationship between BMI and $\omega$ was no longer significant when adding the covariates ($\beta_{BMI}$ = -.17, $p$ = .102; $\beta_{BMI}^2$ = -.053, $p$ = .605, adjusted $R^2$ = .111, $F$ (4,85) = 3.8, p = .007).

Covariate analysis of the model parameters $\beta_{MB}$ and $\beta_{MF}$ now revealed a group difference in $\beta_{MF}$ at trend level ($KW$(2) = 5.0, $p$ = .081), which was driven by a significantly higher $\beta_{MF}$ for obese relative to normal-weight ($p$ = .023), but not overweight participants, whereas still no group difference was observed for $\beta_{MB}$ ($KW$(2) = 2.2, $p$ = .336)**.** These findings were complemented by continuous analysis of BMI and $\beta_{MB}$ and $\beta_{MF.}$ That is, the group difference in $\beta_{MF}$ was reflected in a significant positive relationship with BMI as before ($\beta_{BMI}$ = .269, $p$ = .010; $\beta_{BMI}^2$ = -.128, $p$ = .202; adjusted $R^2$ = .141, $F$ (4,85) = 4.6, p = .002), whereas the absence of a group effect on $\beta_{MB}$ could in fact be explained by a quadratic relationship with BMI at trend level ($\beta_{BMI}$ = .061, $p$ = .570; $\beta_{BMI}^2$ = -.184, $p$ = .083; adjusted $R^2$ = .051, $F$ (4,85) = 2.2, p = .077). However, only the positive relationship between BMI and $\beta_{MF}$ was robust against a slight change in the computational model (i.e., model 2) in which $\beta_{MF}$ and $\beta_{MB}$ were estimated separately($\beta_{BMI}$ = .253, $p$ = .016; $\beta_{BMI}^2$ = -.056, $p$ = .582; adjusted $R^2$ = .042, $F$ (2,87) = 2.9, p = .059).

**Supplemental Figure 1. Overview of participants per group for the two test time frames. A large part of the dataset was acquired between 2012 and 2014 [1,2] and consisted predominantly of normal-weight and overweight participants. Data acquisition was finally completed in 2018 by testing the remaining obese and overweight participants.**

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**Supplemental Figure 2. Stochasticity of model-based and model-free choices (model 2). (A) On the group level, $\beta_{MB}$ exhibited a group difference at trend level. (B) No group difference was observed for $\beta_{MF}$. The box plots in A and B reflect the median, interquartile range, and mean value (black dot) for each weight group. (C) On the continuous level, no linear or quadratic relationship was observed between BMI and $\beta_{MB}$. (D) A positive linear relationship was observed between BMI and $\beta_{MF}$. The scatter plots in C and D show the model fit (black line) and confidence interval (shaded) of the regression models. Individual data points are color-coded based on weight group for illustrative purposes.**