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Research report

Social modeling of eating mediated by mirror neuron activity: A causal model moderated by frontal asymmetry and BMI☆

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ABSTRACT

The social modeling of eating effect refers to the consistently demonstrated phenomenon that individuals tend to match their quantity of food intake to their eating companion. The current study sought to explore whether activity within the mirror neuron system (MNS) mediates the social modeling of eating effect as a function of EEG frontal asymmetry and body mass index (BMI). Under the guise of rating empathy, 93 female undergraduates viewed a female video confederate "incidentally" consume either a low or high intake of chips while electroencephalogram (EEG) activity was recorded. Subsequent ad libitum chip consumption was quantified. A first- and second-stage dual moderation model revealed that frontal asymmetry and BMI moderated an indirect effect of model consumption on participants' food consumption as mediated by MNS activity at electrode site C3, $a_3b_3 = -0.718$, $SE = 0.365$, 95% CI [−1.632, −0.161]. Left frontal asymmetry was associated with greater mu activity and a positive association between model and participant chip consumption, while right frontal asymmetry was associated with less mu activity and a negative association between model and participant consumption. Across all levels of frontal asymmetry, the effect was only significant among those with a BMI at the 50th percentile or lower. Thus, among leaner individuals, the MNS was demonstrated to mediate social modeling of eating, as moderated by frontal asymmetry. These findings are integrated within the normative account of social modeling of eating. It is proposed that the normative framework may benefit from consideration of both conscious and unconscious operation of intake norms.

1. Introduction

The influence of social factors on how much individuals eat has been robustly established. Evidence consistently demonstrates that individuals eat more when their eating companion eats more, while eating less when their eating companion eats less [10,51]. Vartanian et al. [51] recent meta-analysis of 38 articles demonstrated a large modeling effect, $r = 0.39$, in literature to date. Despite the consistency with which this so-called social modeling of eating effect emerges, a definitive explanation as to why this occurs has yet to be elucidated. A number of moderators have been explored to delineate the mechanisms that may strengthen or minimize this effect with limited success. The tendency to model the food intake of one's companion appears to be reliably elicited across heterogeneous participant characteristics and situational contexts [10,51]. The durable quality of this behaviour arguably suggests that it may be partially attributable to inherent mechanisms within the brain. The central aim of this study was thus to explore a feasible neural mechanism of action through which social modeling of eating may emerge and attempt to identify for whom this effect may be most likely to emerge.

Preliminary support from the social modeling of eating literature implicates the importance of unconscious behavioural mimicry processes in the social modeling of eating effect. Hermans et al. [32] found that women who ate with a companion were more likely to eat bites that were congruent with their eating companion (i.e. within 5 s) rather than incongruent bites (i.e. outside the 5s interval). This may arguably insinuate the role of a neural link between perception and action, a link made possible via the mirror neuron system (MNS). The MNS refers to a conglomerate of neurons in the human premotor and parietal cortices responsive to both action-execution and action-observation [19]. Mirror neurons fire both when an individual performs an action themselves and when solely observing an action performed by another individual [34]. Mirror neurons were first localized in the ventral premotor cortex, inferior parietal lobe, and part of the inferior frontal gyrus of the macaque brain using microelectrode recordings of single neurons [13]. A human homologue of the MNS has since been identified in the aforementioned areas, in addition to the dorsal premotor cortex, superior parietal lobe, temporal gyrus, and the cerebellum, primarily

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with functional magnetic resonance imaging (fMRI) and other non-invasive brain imaging techniques, such as electroencephalogram (EEG; [19]. MNS activity is measured with EEG via mu rhythm, with alpha-mu (8–12 Hz) frequency bands generated over the sensorimotor cortex desynchronizing, or decreasing in amplitude, during both execution and observation of actions [23]. A recent meta-analysis by Fox et al. [19] found consistent support for EEG mu rhythm desynchronization during both action execution ($d = 0.46$) and action observation ($d = 0.31$) in 85 EEG studies of the MNS.

This neural mirroring mechanism has been implicated in behavioural imitation capabilities [40]. Upon observing an action, MNS activation of corresponding motor areas in the brain can facilitate a response in the observer and elicit repetition of similar actions [17]. Particularly relevant to social modeling of eating, studies with macaques have shown that response facilitation occurs in the observation of eating. In one study, macaques who observed a conspecific eating demonstrated a greater frequency of eating behaviour than those that did not [17]. Activation of mirror neurons in response to bite observation may in turn increase the likelihood of initiating a parallel action [32], thereby resulting in approximating a companion's intake.

If the MNS underlies social modeling of eating, factors that enhance MNS activity should also enhance the tendency towards social modeling. The current study thus hypothesized that significant moderators of the social modeling of eating effect may ostensibly be limited by factors that lead to differential activation of the MNS. Notably, greater MNS activation has been associated with increased attention to stimuli [39]. Research has also demonstrated that observation of rewarding actions generated significantly greater mu rhythm desynchronization than punishing or neutral actions [4]. Ergo, variables that influence individuals' attention and response to rewards may be capable of altering social modeling effects. Frontal asymmetry is thus one viable contender.

1.1. Frontal asymmetry and approach motivation

Frontal asymmetry is a well-studied phenomenon referring to the differential lateralization of cortical activity between the left and right frontal brain regions. With EEG, frontal asymmetry is defined as the difference between alpha activity within the left and right frontal sites [49]. A longstanding literature suggests that differences in frontal asymmetry reflect an underlying propensity towards certain emotional or motivational trait response tendencies [49]. Greater left frontal activity has been deemed characteristic of an approach-motivated tendency associated with heightened responsivity to appetitive stimuli, whereas greater right frontal activity has been associated with the predominance of avoidance of aversive stimuli [49]. Corroborating this notion, it has been demonstrated that left frontal asymmetry is associated with greater bias to respond to reward-related cues [45].

Evidence has also indicated that left frontal asymmetry is associated with attentional narrowing towards appetitive stimuli [25]. Bandura's fundamental social learning account of modeling distinctly recognizes that attention is essential for any act of modeling to occur. Attention serves to regulate the sensory registration of modeled actions [2]. One must necessarily be paying attention to a model to register their behaviour enough so as to be able to subsequently mimic that behaviour. Greater attention towards food stimuli would then hypothetically be expected to enhance the likelihood of the occurrence of modeling through heightened awareness of the model's eating behaviour. As noted, greater attentional allocation to stimuli has been found to enhance mu rhythm desynchronization [39]. Frontal asymmetry was thus explored for its capacity to strengthen or weaken MNS activity in response to the observation of a model's consumption. Individuals' body mass index (BMI) was also explored as a moderator within the model.

1.2. BMI as a second-stage moderator

Though previous studies in the literature on the social modeling of eating effect suggest participants' weight status does not moderate this effect (i.e., [9,47]), weight status in these studies has been classified categorically and dichotomized into normal weight vs. overweight classes. It has been noted that dichotomizing variables can lead to negative consequences, such as loss of information about individual differences and loss of statistical power [37]. Thus, it was reasoned that previous studies may have been unable to detect an effect of BMI due to undesirable statistical methodology. Moreover, overweight and obese individuals appear to exhibit an attenuated reward response with respect to dopamine release upon consuming food relative to their anticipatory reward response to food cues when compared to those of a normal weight [14]. In a social eating scenario, observing one's companion eating is often a cue for imminent consumption of food for oneself (i.e., at a restaurant when one's companion is served first). As heavier individuals would still experience a reward response to anticipatory cues, they would not be expected to display differential MNS activity in response to observing eating. However, given that consumption itself may be less rewarding, individuals may be less likely to continue eating to match their companion's consumption. Higher-order cognitive control may play a role to dampen the effect of MNS activation on explicit behaviour. In addition, the "right brain hypothesis," posits that obesity may be related to dysfunction in the right prefrontal cortex [1]. Compared to leaner counterparts, individuals classified as obese tend to display lower gray matter density in the right frontal operculum and frontal gyri [41]. Such findings may implicate differences in frontal asymmetry across BMI classes that may alter the strength of the indirect effect as moderated by frontal asymmetry. The current study therefore hypothesized that frontal asymmetry would moderate the first stage of the proposed indirect association between model and participant consumption via mu rhythm desynchronization, while BMI would moderate the second stage when observing a model eating as displayed in Fig. 1.

1.3. The normative account of social modeling of eating

According to the normative account of the social modeling of eating effect, individuals look to others to determine how much food is appropriate to eat in the absence of clear intake guidelines [29]. Given that internal signals for satiety may be unreliable regulatory controls of food intake [29], the normative account argues individuals may consciously rely on socially-derived norms to determine when to stop eating to avoid eating excessively [30]. This normative interpretation appears to account for individuals' tendency to match others' intake when they are merely told how much food previous individuals have consumed in a similar situation (e.g., [36]). Such situations cannot be explained by MNS activity and involve more conscious efforts to regulate intake. The current study does not intend to suggest that the MNS can explain the social modeling of eating phenomenon in its totality. Mimicry is, of course, by no means an inevitability [54]. However, evidence to date suggests that modeling can both be accessible to conscious control, as well as automatic [10]. The normative model may thus operate on two separate levels: one, a conscious decision-making process, and the other, a hard-wired inclination mediated by activity within the MNS (Fig. 2)

Cruwys et al. [11] argue that the social identity approach offers a parsimonious model for understanding the existence of normative effects on modeling such that, in order for modeling to occur, shared group membership must be perceived. A particular model's food intake may only be interpreted as a valid reference point for oneself to the extent that shared group membership exists. Parallels between social modeling of eating and the MNS notably emerge in regards to the importance of similarity across both literatures. Evidence to date suggests similarity is a significant moderator of social modeling of eating.

Specifically, individuals exhibited a greater likelihood of modeling in-group members rather than out-group members for psychologically salient features such as university affiliation [11], gender [9], and weight [12,38]. Eating norms displayed by an in-group member such as one's eating companion may be deemed to be self-relevant, and thus, individuals may selectively attend to this consumption information to guide appropriate behaviour [10].

The mirror neuron literature likewise indicates that the MNS is more active when the model and observer are more similar [34]. A growing body of evidence suggests that perception-action coupling within the MNS is strengthened by shared group membership [16]. Gutsell and Inzlicht [24] reported that individuals displayed greater mu rhythm desynchronization when passively observing the actions of in-group members, and comparatively less when observing out-group members' actions. Evidence derived from studies exploring unconscious mimicry further stresses the importance of social context and shared group membership. Bourgeois and Hess [3] found that individuals only displayed facial mimicry of negative emotions when they were displayed by an in-group member, but not when displayed by an out-group member. Yabar et al. [54] similarly reported that participants displayed greater unconscious mimicry of face touching behaviours when exhibited by an in-group member than when those same actions were performed by an out-group member. Thus, the extant literature suggests that individuals may be more likely to respond to behavioural norms created by those one perceives as more similar to oneself, regardless of whether this occurs consciously or without conscious effort. Exploration of the MNS in relation to the social modeling of eating effect may thus provide further explanatory power to determine the circumstances under which modeling will occur.

2. Method

2.1. Participants

A total of 107 female participants, $M = 20.01$, $SD = 3.60$, range 17–36, were recruited from Lakehead University's Department of Psychology undergraduate participant pool through Sona Experiment Manager. The majority of the sample self-identified their ethnicity as Caucasian (71.0%), with the remainder reporting either Aboriginal (7.5%), South Asian (7.5%), African-Canadian (3.7%), East Asian (1.9%), Middle Eastern (1.9%), or "Other" (4.7%). Participants' BMI ranged from 17.09 to 44.61, $M = 24.76$, $SD = 4.62$. Despite random assignment to experimental conditions, those in the low-intake condition demonstrated a significantly lower BMI, $M = 23.50$, $SD = 3.97$, $t(91) = -2.51$, $p < 0.05$, than those in the high-intake condition, $M = 25.67$, $SD = 4.38$. However, BMI was not significantly correlated with chip consumption $r = -0.01$, $p = 0.94$. No other significant differences emerged between conditions. All participants had normal or corrected-to-normal vision. Participants were required to be non-smoking, not taking cold/hypertensive medication, and right-handed. They were also to refrain from exercising, drinking caffeine, or eating 2 h prior to the laboratory session and abstain from alcohol 12 h prior. The procedure was approved by the Lakehead University Research Ethics Board.

Of the total 107 participants who completed the experimental protocol, fourteen were excluded for various reasons. Three participants were removed due to fewer than 25% of utilizable EEG epochs collected in the recording blocks of interest. Four participants were excluded on the basis of answering less than 50% of attentional assessment questions correctly. Based on raw EEG spectral power, five participants were defined as outliers (z-score = ± 3.29; [18]) and removed for excessive distortion in EEG signals. Two additional participants were excluded on the basis of a laterality quotient suggestive of ambidextrous handedness on the short form of the Edinburgh Handedness Inventory (EHI; [52]), in accordance with evidence of differences in the lateralization of parietal-premotor coherence among such individuals [35]. The final remaining sample utilized in statistical analyses consisted of 93 participants.

2.2. Materials

2.2.1. Food intake

Participants in each condition were provided access to a preweighed 56-g bowl of LAY'S® Classic chips to eat (i.e., approximately 30–40 chips). The bowl was weighed at the end of the session using a Denver Instrument Summit Series S-2002 laboratory scale, and the weight of the remaining chips was subtracted from the initial weight of the chips.

2.2.2. Electroencephalogram (EEG) recording

EEG signals were recorded using a 24-channel Waveguard EEG cap from Advanced Neuro Technology (ANT; Enschede, Netherlands) with electrodes embedded in the cap at scalp positions F3, F4, C3, Cz, C4, O1, O2, M1, and M2 in accordance with the International Electrode Placement System. The EEG signals were filtered through a 72-channel amplifier and into a computer with ASA 4.7 Experiment Manager (Version 9.2) software. Since mu rhythm is defined as oscillations measured over the sensorimotor cortex, data from C3 and C4 were of particular interest, as is typical in EEG studies of the MNS [40]. Cortical EEG activity was sampled continuously for the duration of recording at a sampling frequency of 1024 Hz. Two electrodes were also placed above and below the right eye to provide an index of vertical electro-oculogram (VEOG) activity to assess eye blinks and optical artifacts.

Offline EEG processing was conducted using Advanced Source Analysis (ASA; Version 4.8.0) software to prepare the EEG signals for analyses. EEG data was re-referenced to the average of the mastoid electrodes (i.e., M1 and M2). Artifacts in the signal exceeding ± 100 μV in amplitude, as well as those coinciding with eye blinks from the VEOG signal were detected. Additional artifacts were identified in the signal through visual inspection. Epochs encompassing these artifacts were excluded from analyses. EEG data was high-pass filtered at 24 Hz, with a low cut-off frequency of 0.49 Hz and a high cut-off frequency of 100 Hz. Data were segmented into epochs of 1 s duration, with an interval of 0.5 s between epochs and 50% overlap. To extract power spectral densities in the 8–12 Hz alpha-mu frequency band, a Fast Fourier Transform was performed at 0.5 Hz intervals using a Hanning window.

An index of mu rhythm desynchronization was calculated as the ratio of change in EEG amplitude from the baseline condition to the eating-view recording block (i.e., eating-view mu power/baseline mu power) for the central sites. The use of a ratio optimally controls for variability in absolute mu power due to individual differences in scalp thickness and electrode impedance [40]. Given that ratio data are non-normal as a result of lower-bounding, the ratio was log transformed, as is customary for mu suppression [42]. A log ratio of less than zero indicates suppression, values of zero indicate no suppression, and values greater than zero are reflective of enhancement [42]. Concerns have notably been raised as to whether activation at central electrode sites is truly reflective of MNS activation or merely an artifact of spreading activation from alpha activity in occipital regions [33]. The mu frequency band overlaps with alpha, which is particularly sensitive to attentional fluctuations. Suppression ratios were thus additionally calculated for sites O1 and O2 to enable examination of the localization of the effect.

To calculate frontal asymmetry scores, alpha power at the left (F3) and right (F4) electrode sites during the "eating-view" recording was first natural log transformed, in keeping with conventions for this type of data [49]. A difference score was subsequently calculated to summarize the relative activity across the right and left frontal cortex (i.e. $F4ln - F3ln$) in accordance with convention. As alpha power is inversely associated with cortical activity (i.e., greater power equates to less cortical activity, while lower power equates to more activity), higher asymmetry scores indicated greater left-hemispheric activity

whereas negative scores reflect greater right-hemispheric activity [20]. Frontal asymmetry from the eating-view block, rather than the baseline, was utilized to provide more power to detect more stable differences in individuals' response tendencies. Frontal asymmetry in response to motivationally-relevant challenges can reduce uncontrolled subject factors that may arise during resting state recordings, thereby increasing the power to discover associations between neural activity and subjective responding [49]. However, the use of baseline frontal asymmetry did not appreciably change the results.

2.2.3. Video stimuli

The baseline video for EEG recording was comprised of two alternating video clips sourced from YouTube™ edited and combined into one 5-min video using Movie Studio Platinum (version 12.0). The first video clip depicted a cat playing with puppies for a 1.5 min, followed by a video of a fireworks show for the subsequent 2 min, and ended with 1.5 min of the remainder of the first video clip. These clips were chosen in accordance with Hobson & Bishop's [33] assertion that the baseline stimulus should not activate the MNS. Even stimuli illustrating the opening of a flower, as some studies have utilized, may arguably be imitable by imagining the opening of a closed hand, for example. Tangwiriyasakul et al. [50] assert that the optimal baseline for calculation of mu rhythm suppression ought to block the imagination of movement, induce a relatively relaxed state, and maintain attention.

The confederate videos were recorded using a Canon EOS 7D camera with a Canon EF-S 17–85 mm image stabilizer lens and an Olympus E-M5 camera, each mounted on a tripod. The confederate was observed eating in the same laboratory setting and sitting in the same position that participants in the current study sat during their laboratory sessions. The confederate was shown eating either a small or large amount of LAY'S® Classic chips (5.7 kcal/g). In both the low- and high-intake conditions, the confederate began with 56 g of chips in the bowl, the equivalent of two single-serving bags of chips. In the low-intake condition, the confederate ate 5 chips (6.96 g) over the duration of the 10-min 27 s video segment. In the high-intake condition, the model ate 35 g, or approximately 33 chips. The disparity in consumption between conditions was noticeable in both the number of times the model reached for a chip (5 vs. 33) and the quantity of chips visibly remaining in the transparent bowl. To ensure that the emotional expressions of the confederate did not differ across the conditions, an earbud was placed in the confederate's left ear out of view of the camera during recording, which dictated instructions as to when to smile, frown, laugh, and so forth during the 10-min film clip.

The same confederate modeled both intake conditions. Her age was 24 years and BMI was 26.3 (weight 144 lbs, height 158 cm). The videos displayed the confederate from the shoulders up and she donned a loose-fitting sweatshirt to minimize comparison of body shape and size and to ensure ambiguity of body size to enhance the likelihood of perceived similarity in weight. For both the low- and the high-intake conditions, two camera angles were simultaneously filmed. There was no sound to accompany either video. The first camera angle (i.e. "eating-view" video) recorded a front-facing view of the confederate to enable visibility of her facial expressions and eating behaviour. This first camera was placed in front of the confederate at approximately a 45° angle to the right of where she was seated. From this perspective, the television screen was not visible to participants. The second camera angle (i.e. "movie-view" video) recorded a side-angled view of the confederate, recorded with the camera marginally behind and to the right side. This angle displayed the right side of her face and facilitated a view of the bowl of chips on the table, as well as the film *Up* (© 2009 Walt Disney Studios Motion Pictures) playing on the television in the background. This perspective enabled temporal contiguity between observation and subsequent modeling of food consumption when food was available ad libitum. The confederate grabbed each chip with her right hand.

A coarse behavioural measure of attention was utilized akin to that used by Hobson and Bishop [33] to confirm that participants adequately attended to the video stimuli for the video's duration. This was included in light of recommendations that attentional engagement is critical to monitor to assist in disentangling the involvement of attentional processes in identifying mirror neuron activity [44]. The baseline and confederate videos were edited in Movie Studio Platinum (version 12.0) to insert symbols to be periodically displayed in an area of the visual space. Following each video, participants were asked to indicate what symbol appeared, how many times it was seen, and its location on the screen.

2.3. Procedure

Once informed consent was obtained, participants were fit with an appropriately-sized EEG cap (large: 56–61 cm; medium: 51–56 cm; small: 47–51 cm) and connected to a 72-channel amplifier (ANT; Enschede, Netherlands). ElectroGel was applied to the scalp to attain impedance levels below 10 kΩ prior to recording. Two bipolar electrodes were placed above and below the right eye to record ocular artifacts throughout the procedure. One electrode was attached below the left clavicle to serve as the ground electrode. Once connected, participants were told the cover story to mask the true aim of the study. Participants were purposely deceived to believe that the study sought to examine MNS activation in response to mental judgments about the level of empathy others possess. This deception was necessary, as previous research has indicated that participants' eating behaviour may be altered if they feel their food intake is under scrutiny [30].

The entire procedure was viewed on a 72-in. diagonal wide Samsung DLP television located 2 m in front of the seated participant. Once participants were briefed on the flow of experimental tasks, the researcher retreated to a back room in the laboratory for the majority of the study's duration to control the presentation of tasks and EEG recording. As level of hunger could affect the amount of food consumed during ad libitum access, participants rated their hunger on the Grand Hunger Scales [21] prior to EEG recordings. Additional questions to assess thirst, fatigue, and discomfort were affixed to these scales to uphold the ruse of the study, each measured on a 7-point Likert scale. The 5-min baseline recording was then taken as participants watched the baseline video, followed by completing the attentional assessment.

Participants were then instructed that they would watch a video of a fellow university student, in reality the confederate, while she was watching an emotional film as part of a previous study conducted to rate her perceived level of empathy based on facial expressions alone. Participants had been randomized to view either: (1) the low-intake condition, in which the confederate depicted the consumption of a small quantity of chips, or (2) the high-intake condition in which the confederate consumed a large quantity. Individuals then watched the "eating-view" video displaying the front-facing view of the model's eating behaviour and facial expressions. Participants proceeded to answer the attention assessment questions and a contrived empathy rating questionnaire to purportedly assess the model's empathy after the video.

Upon completion of these questionnaires, participants were informed that the study's last task would take a few minutes to set up. Participants were told that whilst waiting, they would see the film the confederate watched to satisfy any curiosity about the accuracy of their empathy rating. The researcher stated that to make the wait more pleasant, they should feel free to help themselves to some chips. At this time, the researcher brought out the same 56 g bowl of LAY'S® Classic chips the confederate ate from the back room and set it down on the table in front of them. The researcher again retreated to the back room to supposedly continue setting up the next task. Participants were not aware that this portion of the session was of interest to the experimenter. This video displayed the "movie-view" side-angled view of the model in which her eating behaviour and the television screen were both visible.

Once the video concluded, participants again completed the attentional assessment and a questionnaire to assess their emotional valence in response to the film and how much they liked the chips offered before proceeding to the final task of the study. The details of this task have been omitted, as they are not of interest to the present study. Upon its completion, participants answered a demographic questionnaire and the EDI [52]. Before leaving, the researcher measured participants' weight using a Brecknell (LPS-400) digital scale and their height. The bowl of chips was weighed at the end of the laboratory session once the participant had left. The study's true purpose was not revealed to participants at this time to ensure that they were not able to discuss the study's deception with others in advance of their participation. This was an important consideration given the small size of the university campus. A debriefing email was sent indicating the true purpose of the study once the study had concluded.

3. Results

3.1. Data analytic strategy

Hayes [28] PROCESS model 21 was utilized to examine the dual moderated mediation. The model hypothesized that the first stage of the indirect effect of model consumption (*X*) on participant consumption (*Y*) through mu desynchronization (*M*) would be moderated by frontal asymmetry (*W*) (i.e. exerting its influence between *X* and *M*), and that the relationship between frontal asymmetry and the indirect effect would be conditioned on the value of BMI (*V*) in the model's second stage (i.e. exerting its influence between *M* and *Y*). Model 21 generates an index of dual moderated mediation (a_3b_3) which quantifies the rate of change in the moderation of the indirect effect by each moderator as the other changes [26]. To discern that the index is significantly different from zero, PROCESS generates a bias-corrected bootstrap confidence interval (CI) identifying the upper and lower bounds of a 95% CI. When this interval does not include zero, this indicates a statistically significant index of dual moderated mediation, enabling a claim of a first- and second-stage dual moderated mediation [27]. The model is then probed via inferential tests of the index of conditional moderated mediation for values of both moderators using the pick-a-point quantile method to integrate into a substantive interpretation [26]. Following examination of the hypothesized model, a simple mediation model was analyzed using Hayes' [28] PROCESS model 4 to report on the indirect effect of model consumption (*X*) on participant consumption (*Y*) through mu rhythm desynchronization (*M*).

Participants' baseline frontal asymmetry was controlled for. Four additional covariates were included in the model: chip liking, level of discomfort, positively-valenced emotional state, and age. In preliminary analysis, these variables were found to be significantly associated with variation in mu rhythm desynchronization or chip consumption. It is crucial in forming a causal argument that one is able to rule out epiphenomenality or spurious associations as alternative explanations [28]. As a general rule in mediation analyses, one ought to include covariates in the models of mediators and *Y* unless there is a principled reason to exclude such variables, even if there is only a demonstrable association with the mediator (A.F. Hayes, personal communication, March 8, 2017). Variables spuriously associated with key variables in the causal system therefore must be controlled for [28]. Moreover, it was discerned that the literature reasonably substantiated the inclusion of each of the covariates included to ensure that they were not merely artifacts of the current sample.

Participants consumed an average of 19.76 g, $SD = 17.01$, out of 56 g chips served (i.e., 35%). The quantity of chips consumed was positively skewed, $z_{skewness} = 2.83$, and was therefore subjected to a square root transformation, which was utilized in the analysis. BMI was also positively skewed, $z_{skewness} = 4.39$. Only a reciprocal transformation effectively attenuated the skew. However, the reciprocal reverses the scores such that values initially large in the data become small and vice versa [18]. As the untransformed BMI did not appreciably change the results obtained, untransformed BMIs are reported for ease of interpretation.

Table 1
Indices of Dual Moderated Mediation, Standard Errors, and Bias-Corrected Bootstrap 95% CIs of PROCESS Model 21 Testing the Effect of Model Consumption (X) on Participant Consumption (Y) through Mu/Alpha Suppression (M) as Moderated by Frontal Asymmetry (W) and BMI (V).

Electrode Site	a_3b_3 (*SE*)	95% CI
C3	−0.718 (0.365)	**−1.632, −0.161**
C4	−0.440 (0.303)	−1.091, 0.124
O1	−0.327 (0.469)	−1.298, 0.532
O2	−0.283 (0.433)	−1.238, 0.454

Note. $N = 93$. Bolded 95% CI do not straddle zero.

3.2. Social modeling of eating effect

In line with the social modeling of eating effect, participants' consumption of chips was positively correlated with the model's consumption, $r = 0.25$, $p < 0.05$. A one-way ANOVA additionally revealed that individuals who viewed the model consume a low intake consumed significantly fewer chips, $M = 3.28$, $SD = 2.20$, than those who viewed the model consume a high intake, $M = 4.41$, $SD = 2.19$, $F(1, 91) = 6.12$, $p < 0.05$. As per Cohen's classification [18], the obtained effect size $d = -0.52$ is considered medium. This is comparable to the effect reported by Vartanian et al. [51], $r = 0.39$ (equivalent to $d = 0.85$), though not quite as large.

3.3. Dual moderated mediation model

The overall dual moderated mediation model was significant at electrode site C3, $a_3b_3 = -0.718$, $SE = 0.365$, 95% CI [−1.632, −0.161]. As the CI does not include zero, frontal asymmetry and BMI significantly moderated the indirect effect of model consumption on participant consumption via mu rhythm desynchronization. The results were not altered when the untransformed value of participants' consumption was utilized in analyses, C3, $a_3b_3 = -5.621$, $SE = 3.181$, 95% CI [−12.979, −0.405]. This index can be interpreted to mean that the moderation of the indirect effect of model consumption on participant consumption via mu rhythm desynchronization by frontal asymmetry decreased by 0.718 units as BMI increased. Table 1 displays the indices of dual moderated mediation for each model across electrode sites. The dual moderated mediation model was not significant at either C4, O1, or O2. Exclusion of the aforementioned covariates in the model led to a nonsignificant index of dual moderated mediation, C3, $a_3b_3 = -0.448$, $SE = 0.340$, 95% CI [−1.398, 0.018]. However, when chip liking was then included as the sole covariate given its strong association with participants' consumption, the effect was significant, C3, $a_3b_3 = -0.487$, $SE = 0.295$, 95% CI [−1.319, −0.081], suggesting that the effect was not merely an artifact of overfitting the model.

Fig. 1 depicts the regression coefficients for each pathway in the model estimated at site C3.[1] The model can be separated for interpretive purposes into two separate moderation analyses. The regression coefficient for $X \times W$ (a_3 in Fig. 1) indicates a statistically significant interaction between model consumption and frontal asymmetry predicted C3 mu suppression. For two cases that differed by a unit on *X* (i.e. low- vs. high-intake condition), mu suppression changed by −2.382 units as frontal asymmetry changed by one unit. Thus, observation of greater eating behaviour was associated with more mu

[1] Unstandardized regression coefficients, standard errors, and 95% CIs generated from the model tested at electrode sites C3, C4, O1, and O2 can befound in Supplemental Table 1.

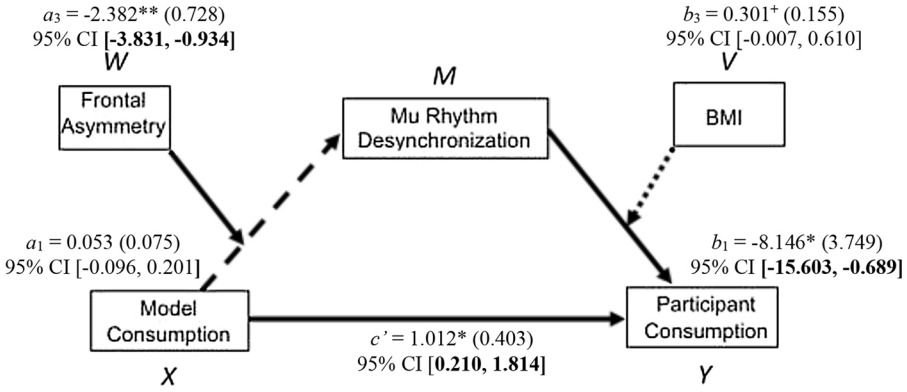

Fig. 1. Unstandardized regression coefficients (*SE*) and 95% CIs for the simple effects and interaction terms in the dual moderated mediation of participant consumption (*Y*) from model consumption (*X*) through mu rhythm desynchronization (*M*) as moderated by frontal asymmetry (*W*) and BMI (*V*) at site C3. Broken lines depict nonsignificant effects. Dotted lines depict trending effects. $^{+}$ $p < 0.06$, $^{*}p < 0.05$, $^{**}p < 0.01$.

suppression (i.e., more negative values on *M*) among those with higher levels of frontal asymmetry. The first stage of this model was followed up with a simple moderation analysis. At frontal asymmetry values corresponding to −1 *SD* (i.e., right frontal asymmetry), the association between model consumption and mu suppression at C3 was positive, $b = 0.230$, $SE = 0.095$, $p < 0.05$, 95% CI [0.040, 0.419], indicative of less activity in the MNS. For values +1 *SD* (i.e., left frontal asymmetry), it was negative, $b = -0.282$, *SE*, 0.123, $p < 0.05$, 95% CI [-0.526, −0.038], suggestive of greater MNS activity.

Moreover, mu suppression significantly predicted chip consumption, $b_1 = -8.146$, $SE = 3.749$, 95% CI [−15.603, −0.689]. The negative regression coefficient between mu suppression and participant consumption suggests that greater suppression was associated with a larger quantity of chips consumed. Although the interaction between mu suppression (*M*) and BMI (*V*) was not statistically significant, $b_3 = 0.301$, $SE = 0.155$, 95% CI [−0.007, 0.610], $p = 0.06$, there was a trend towards significance. This coefficient for $M \times V$ represents that as BMI increased, greater mu suppression was associated with *fewer* chips consumed by the participant.

Table 2 presents the conditional indirect effect of *X* on *Y* at values corresponding to the 10th, 25th, 50th, 75th, and 90th percentiles of frontal asymmetry and BMI. The indirect effect quantifies how much two cases that differ by a unit on *X* are estimated to differ on *Y* as a result of *X*'s influence on *M* [28]. The conditional indirect effect was significant for values of frontal asymmetry corresponding to the 10th, 75^{th,} and 90th percentiles. The 75th and 90th percentiles correspond to frontal asymmetry values of 0.090 and 0.160, respectively. Positive frontal asymmetry reflects left frontal asymmetry. At these values, the indirect effect of model consumption on participant consumption was positive, suggesting that model consumption was associated with greater chip consumption as mediated by mu suppression. The 10th percentile of frontal asymmetry corresponded to a value of −0.103, suggesting right frontal asymmetry. For such individuals, the negative indirect effect suggested that greater model consumption was associated with less chip consumption as mediated by mu suppression. With respect to BMI, the conditional indirect effects were only significant for those with a BMI at the 50th percentile or lower (i.e., 20.08–23.72), and was increasingly attenuated as BMI increased.

3.4. Simple mediation model

Analyses were also run to examine the indirect effect of model

Table 2
Unstandardized Regression Coefficients, Standard Errors, and Bias-Corrected Bootstrap 95% Confidence Intervals of the Conditional Indirect Effect of Model Consumption (X) on Participant Consumption (Y) through Mu Suppression (M) at Levels of Frontal Asymmetry (W) and BMI (V).

		Model A: C3 Mu Suppression (M_1)		Model B: C4 Mu Suppression (M_1)	
FA (*W*)	BMI (*V*)	*b* (*SE*)	95% CI	*b* (*SE*)	95% CI
−0.103	20.075	−0.624 (0.343)	**−1.487, −0.104**	−0.516 (0.326)	**−1.332, −0.034**
	21.419	−0.504 (0.286)	**−1.241, −0.083**	−0.436 (0.279)	**−1.139, −0.027**
	23.724	−0.297 (0.212)	−0.882, 0.001	−0.298 (0.219)	−0.853, 0.030
	26.456	−0.051 (0.206)	−0.499, 0.347	−0.135 (0.206)	−0.603, 0.238
	29.778	0.247 (0.314)	−0.184, 1.088	0.063 (0.282)	−0.438, 0.688
−0.033	20.075	−0.273 (0.216)	−0.862, 0.011	−0.249 (0.199)	−0.809, 0.001
	21.419	−0.220 (0.177)	−0.718, 0.007	−0.210 (0.169)	−0.689, 0.000
	23.724	−0.130 (0.121)	−0.506, 0.009	−0.144 (0.126)	−0.520, 0.013
	26.456	−0.023 (0.099)	−0.286, 0.139	−0.065 (0.107)	−0.359, 0.100
	29.778	0.108 (0.162)	−0.066, 0.614	0.031 (0.146)	−0.205, 0.398
0.023	20.075	0.003 (0.160)	−0.365, 0.287	−0.038 (0.131)	−0.412, 0.147
	21.419	0.002 (0.130)	−0.295, 0.238	−0.032 (0.110)	−0.352, 0.123
	23.724	0.001 (0.081)	−0.174, 0.168	−0.022 (0.077)	−0.271, 0.079
	26.456	0.000 (0.047)	−0.103, 0.101	−0.010 (0.049)	−0.191, 0.048
	29.778	−0.001 (0.091)	−1.223, 0.176	0.005 (0.062)	−0.085, 0.184
0.090	20.075	0.322 (0.198)	**0.029, 0.847**	0.205 (0.154)	−0.008, 0.635
	21.419	0.260 (0.164)	**0.024, 0.715**	0.173 (0.132)	−0.008, 0.547
	23.724	0.153 (0.120)	−0.001, 0.538	0.118 (0.104)	−0.014, 0.438
	26.456	0.027 (0.114)	−0.159, 0.324	0.054 (0.092)	−0.063, 0.352
	29.778	−0.127 (0.175)	−0.723, 0.074	−0.025 (0.120)	−0.349, 0.175
0.160	20.075	0.690 (0.324)	**0.193, 1.536**	0.485 (0.273)	**0.069, 1.186**
	21.419	0.557 (0.274)	**0.143, 1.294**	0.410 (0.238)	**0.047, 1.032**
	23.724	0.328 (0.216)	**0.024, 0.939**	0.281 (0.195)	−0.009, 0.796
	26.456	0.057 (0.227)	−0.378, 0.569	0.127 (0.194)	−0.196, 0.595
	29.778	−0.273 (0.336)	−1.241, 0.197	−0.060 (0.260)	−0.624, 0.441

Note. $N = 93$. Bolded 95% CI do not straddle zero. BMI and frontal asymmetry levels correspond to the 10th, 25th, 50th, 75th, and 90th percentiles, respectively.

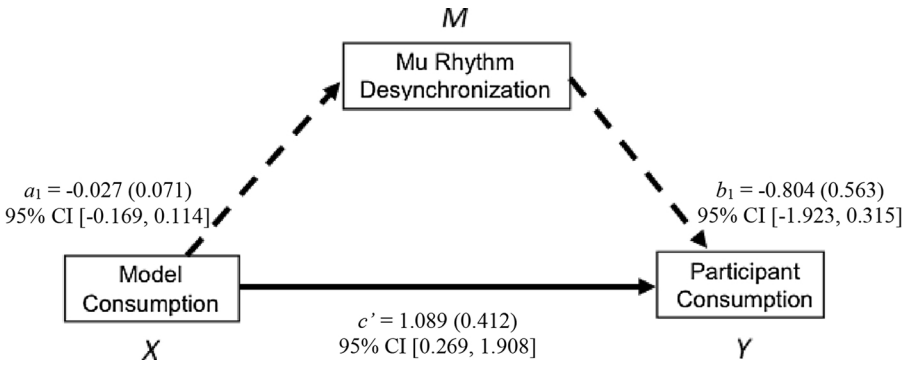

Fig. 2. Unstandardized regression coefficients (*SE*) and 95% CIs for the simple effects in the mediation of participant consumption (*Y*) from model consumption (*X*) through mu rhythm desynchronization (*M*) at site C3. Broken lines depict nonsignificant effects. *$p < 0.01$.

consumption (*X*) on participant consumption (*Y*) through mu desynchronization (*M*). The simple mediation model was not significant at either of the central sites, C3, $b = 0.045$, $SE = 0.068$, 95% CI [−0.037, 0.256] or C4, $b = 0.031$, $SE = 0.060$, 95% CI [−0.045, 0.228] (see Fig. 2). The effect was also not significant at O1 or O2 .

4. Discussion

In a recent review of the social modeling of eating effect, it was highlighted that "…a key agenda for future research must be the question of which mechanism(s) underlie(s) modeling effects on eating" [10] (p.13). The results of the current study provide preliminary evidence for the putative causal involvement of mirror neurons within the social modeling of eating effect as moderated by both frontal asymmetry and BMI. Specifically, evidence was found for a significant index of dual moderated mediation at electrode site C3 which resides over central sensorimotor regions associated with the MNS. Though the model was not significant at C4, significant conditional indirect effects were still apparent, suggesting merely an attenuated effect in the right hemisphere. Previous studies have found that mu suppression appears to be more pronounced in the contralateral hemisphere relative to the hand observed to be moving [42]. As the right hand was observed to perform the actions, evidence of a significant effect in the left hemisphere (i.e., C3) but not in the right (i.e., C4) appears to conform to this pattern. Given that motor functions are generally lateralized to the contralateral hemisphere, suppression at C3 would more closely mimic activation akin to if one were executing the action, as all participants were right-handed. Ergo, suppression over the left central areas may be more significantly linked to one's actual eating behaviours. Topographic specificity was additionally demonstrated with respect to a lack of suppression within occipital sites. Contrary to previously noted criticisms that mu rhythm desynchronization may merely reflect alpha originating in the occipital lobes, the effects were not significant at the occipital sites (i.e., O1 and O2). Thus, evidence of mu suppression was not merely an artifact of attentional processing demands reflected in alpha.

For those who exhibited right frontal asymmetry, greater model consumption was associated with lower participant consumption through mu rhythm desynchronization. Of note, Silva et al. [48] previously found that restrained eaters exhibited greater right frontal asymmetry compared to unrestrained eaters. The current results similarly suggest that right frontal asymmetry was associated with a propensity towards restraining one's eating, particularly when it would otherwise be expected that an individual would consume more. Moreover, the indirect effect as moderated by frontal asymmetry was only significant among those with low to average BMIs (i.e., 20.08–23.72), and was increasingly attenuated as BMI increased. These values notably correspond to the "healthy" BMI range for adults [5]. This gradual attenuation may be explained by diminished reward in response to chip consumption among those of a higher BMI, thereby reducing the likelihood of continued consumption when observing a model eating more. To provide a rudimentary test of this notion, a one-tailed Pearson correlation was conducted between BMI and participants' rating of chip liking. There was a trend towards a significant negative correlation, $r = -0.16$, $p = 0.07$, such that those with a higher BMI tended to report liking the chips less. While this may reflect social desirability, it may be informative for future studies to examine whether reward responses alter this modeling effect.

It is also possible that participants possessing a higher BMI may have been motivated to suppress eating activity to a greater extent than those with lower BMIs. Though differences in consumption were not statistically significant, those with a BMI greater than or equal to 25.0 ate a smaller quantity of chips (in g) on average in the low-intake condition, $M = 11.05$, $SD = 12.20$, than those with a BMI less than 25.0, $M = 16.71$, $SD = 16.55$, as well as slightly fewer chips in the high-intake condition, $M = 22.66$, $SD = 15.56$, compared to leaner individuals, $M = 23.31$, $SD = 18.54$. Those with a higher BMI may feasibly experience a greater baseline urge to engage in hedonically-driven eating when provided ad libitum access to palatable foods. Greater cognitive control may thus be utilized to suppress eating when it is perceived that one should moderate food intake, either for social desirability or based on the model's consumption. Conger et al. [9] proposed that overweight women may be particularly sensitive to social context cues in the presence of others due to self-consciousness and concern about deviating from a desired societal norm of weight. This may suggest that higher-order cognitive processes may determine the degree of influence the MNS may have in eliciting social modeling of eating.

Of note, although the weight status of the participant themselves has not been shown in previous studies to moderate the social modeling of eating effect [9], literature has suggested that it is the interaction between individuals' weight status and the weight status of the model that is most crucial [10]. Specifically, modeling has been found to be more likely when the model was similar in weight status to the participant. Normal-weight individuals have been found to be more likely to adapt their intake to a model of normal-weight appearance, rather than a model who appeared slim [31] or a model who was medically obese [38]. By contrast, obese participants were found only to model the intake of an obese participant [12]. While the current study attempted to make the weight status of the confederate as ambiguous as possible within the parameters, the slenderness of both her face and hands could arguably be used as an index of her overall body weight. Thus, with the use of only one confederate of average weight status, it is possible that the attenuated effect among participants with higher BMIs may be attributable to lower perceived similarity with the model. This further bolsters the importance of similarity and shared group membership on both the social modeling of eating effect and MNS activity.

4.1. Non-significance of the simple mediation model

Analyses revealed that the simple indirect effect between model and participant consumption was not significantly mediated by mirror

neuron activity. However, this does not negate the role of the MNS as a mediator of this effect. When a mediation is moderated, the underlying mechanism differs in size or strength as a function of a set of variables. Thus, it is not possible to quantify the indirect effect as a constant [28]. As the social modeling of eating effect is a robust effect subject to both conscious and unconscious influences, it may not be reasonable to expect that MNS activity alone can reliably account for the full causal operation of the effect across individuals. As indicated above, the moderated mediation effect disappeared among those of a higher BMI, of which 35 participants of the total 93 had a BMI greater than 25.0. Moreover, the moderation in the first step of the model revealed a reversal of the mediated effect across those with either left or right frontal asymmetry. As a result of the effect that such individual differences may have on the emergence of social modeling, it is perhaps not surprising that a single constant was not capable of significantly quantifying this effect.

4.2. Contribution to the social modeling of eating effect

Cruwys et al. [10] criticized that the social modeling of eating effect lacks a parsimonious model to account for why moderators of this phenomenon may or may not exist. The potential existence of a neurophysiological causal mechanism appears to offer a relatively promising model to explain the seemingly automatic, unconscious instances of mimicry that occur in social eating scenarios. According to the current conceptualization of the normative account, individuals look to others to consciously determine how much food is appropriate to eat in the absence of clear intake guidelines [30]. Participants in the current study, however, were unaware that their eating behaviour was of any interest. Thus, it is unlikely that participants consciously sought to match their eating behaviour to the model. The current findings suggest that individuals automatically appeared to engage in social modeling of eating, thereby conforming to normative levels of consumption displayed in the social context, without conscious deliberation to do so. The normative account proposes that individuals in one's in-group are deemed to provide a more relevant reference point, and thus modeling is more likely to occur when individuals perceive shared group membership. Previous studies have found automatic influences of shared group membership on both social modeling of eating [10] and MNS activity (i.e., [24]). That BMI emerged as a significant moderating variable may corroborate the importance of shared group membership in eliciting this effect. As mentioned above, the effect was no longer significant among individuals with a higher BMI, an effect that may feasibly be related to low perceived similarity with the model.

The modeling effect was also notably reversed across those with left and right frontal asymmetry. Those with left frontal asymmetry were more likely to consume more when the confederate ate more, whereas those with right frontal asymmetry were more likely to eat *less*. The current experimental procedure did not prompt participants to consider their level of similarity to the model prior to the observation of eating. However, self-other similarity is a potent factor that can lead to relatively instant formation of attitudes of liking towards others [22]. As left frontal asymmetry has reliably been associated with a tendency towards positive affect [49], it is possible that shared group membership may have influenced frontal asymmetry and subsequently influenced the degree of modeling. Cruwys et al. [10] have suggested that attending to shared group membership may explain why individuals react against an eating norm if it is displayed by out-group members. Such processes may feasibly be reflected on a neurophysiological level. This reflects an intriguing proposition worthy of future exploration through efforts to manipulate salience of group membership. It ought to be noted, however, that the model remained significant when individuals' baseline frontal asymmetry was utilized as the moderator in the first stage of the model, though the size of the index of dual moderated mediation was attenuated. Thus, the current findings suggest that there is likely to be an influence of dispositional individual differences that impact individuals' likelihood of engaging in modeling, perhaps in addition to potential effects of perceived similarity.

Within the normative account, it has also been argued that individuals' desire to affiliate and be liked by one's eating companion may be a primary motive underlying adherence to social norms [10]. Though the current experimental design did not provide an opportunity to consciously form a social bond, involvement of the MNS does not preclude the possibility that social bonding may also influence the strength of this effect. For example, one study demonstrated that external administration of intranasal oxytocin, a central hormone involved in social bonding, enhanced mu rhythm suppression [43]. Evidence suggests that mimicry can establish liking and enhance affiliation [6]. Ergo, it appears as though the MNS may be reasonably integrated into the current normative framework and offer to provide incremental explanatory power to identify when and for whom the social modeling of eating effect may be most likely to occur. Involvement of cognitive processes and conscious efforts to adhere to norms in this effect cannot be negated. The effect may still be malleable to higher-order cognitive processes and exertion of control over one's desired goals. The findings of the current study suggest, however, that the normative account of the social modeling of eating phenomenon may benefit from consideration of the way in which automatic, unconscious processes may contribute to the adherence to social norms. Nevertheless, these findings necessitate replication to be substantiated prior to the development of such an expanded account. The current findings must also be considered in the context of the study's strengths and limitations.

4.3. Strengths and limitations

The present study was limited by the inclusion of only female undergraduate participants. This was founded upon evidence of a stronger social modeling of eating effect within females [51], as well as the importance of perceived similarity to the model for both MNS activity and social modeling. If a neurophysiological mechanism is truly at play, however, this effect should not be specific to females, though the effect may differ in strength across the sexes. Future studies ought to explore whether similar conclusions can be derived when males view a male model, as well as when the model differs in sex from the observer.

Moreover, EEG methodology is inherently associated with particular limitations. As a neurophysiological index, EEG has relatively poor spatial resolution compared to techniques such as fMRI [19]. EEG activity recorded at particular sites may not necessarily reflect cortical activity directly below those electrodes [19]. Cohen [56] notes, however, EEG is capable of reasonably accurate anatomical localization within centimeters and states the preponderance of conclusions linking EEG topography to cognitive, perceptual, or motor processes only necessitate precision at this scale [56]. Nevertheless, the use of neuroimaging techniques with superior spatial resolution (i.e., fMRI), in concert with EEG is desirable to specify localization of functional activity to the MNS in order to substantiate current findings.

Examining the involvement of MNS activity via EEG event-related desynchronization (ERD) in future studies may help to establish that mu suppression occurred specifically in response to the observation of a bite. In the current study, the model's observed eating activity was interspersed with emotional facial displays to uphold the ruse of the study. This deception was necessary to adequately assess the social modeling of eating effect to ensure that participants were not aware that the model's eating behaviour was of interest. However, mirror neuron literature has indicated that individuals exhibit mu suppression in response to facial expressions [15]. Though facial expressions were standardized across both high- and low-intake conditions, MNS activation due to facial expressions may have confounded suppression in response to the observation of eating.

This study had a relatively large sample size compared to other studies within the EEG mirror neuron literature. The majority of studies examining mirror neuron activity typically suffer from small sample

sizes [19], which may result in underpowered study designs. Based on the effect sizes of studies in Fox et al.'s [19] meta-analysis, the authors determined that a sample size of at least 66 participants was necessary to detect a moderate effect size with power of 0.80. However, the current study may have been moderately underpowered with the inclusion of the four predictor variables of interest, as well as the five listed covariates. The typical rule of thumb based on simulation studies has suggested that for linear models, there ought to be a minimum of 10–15 observations per predictor variable to allow for adequate estimates of effects [55]. With these nine predictors, the study thus minimally met the low end of desired sample size. Exclusion of all five covariates led to a nonsignificant effect (see Section 3.3). Including chip liking as the sole covariate, however, the effect was significant, which suggests the effect was not merely due to overfitting the model.

4.4. Future directions & clinical implications

It would be of primary interest for future studies to explore whether systematically altering frontal asymmetry impacts the indirect social modeling of eating effect in the expected direction. Such empirical analyses could bolster confidence in the purported causal process underlying the model. Research has demonstrated that neurofeedback via EEG can enable individuals to modulate patterns of frontal asymmetry. Within such training protocols, individuals receive feedback based on real-time analysis of EEG signals and receive rewarding feedback when asymmetry changes in the desired direction [46]. If this causal model can be corroborated, it may be possible to apply these findings in a clinical setting. Neurofeedback training prior to a meal may be able to attenuate or enhance this effect, depending on the circumstances and desired impact. Advancements in smartphone technology may provide the opportunity for brief, rudimentary neurofeedback sessions to make that feasible. For example, the Muse Monitor app [8] utilizes a wireless 4-channel EEG headband and can show moment-to-moment changes in EEG power.

The current study suggests that inducing right frontal asymmetry prior to social eating scenarios may be able to minimize maladaptive modeling of excessive food intake. This may have implications for attenuating the risk of obesity within families. It has been demonstrated that parental obesity status early in childhood is a strong predictor of later adult obesity [53]. Children's modeling of excessive parental intake is posited to contribute to this association [53]. Targeting children at high familial risk of obesity early may decrease the likelihood of modeling parents' excessive consumption. Conversely, these findings could have implications in the context of treatment for eating disorders. Chronic restrained eaters tend to exhibit right frontal asymmetry at rest [48]. It could be explored whether inducing left frontal asymmetry prior to a meal could encourage approximating a more appropriate intake and aid in the establishment of more regular patterns of consumption. However, it would be necessary to explore whether this effect holds true among those with a BMI less than 20.0, as only one tenth of the current sample had such a low BMI.

To the knowledge of the authors, this study is the first to examine the MNS in relation to human eating behaviour, and more specifically, in relation to the social modeling of eating effect. Though Hermans et al. [32] alluded to the involvement of "a mirroring network" (p. 4) in explaining the close correspondence of bites when women ate together, there have been no studies to date directly testing the involvement of the MNS to explain such effects. More research is warranted to elucidate the influence of the MNS on human social eating behaviour and to attempt to replicate the current findings. Further investigation may substantiate an expansion of the normative account to acknowledge the automatic operation of social norms in the influence on individual's consumption. Overall, the evidence from the current study suggests that among low to average weight individuals, those with a predisposition towards an approach-motivation style may be more susceptible to engage in the social modeling of eating effect through the enhancement of mirror neuron activity.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.bbr.2017.10.009.

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