

Social Modeling of Eating and the Mirror Neuron System:
The Role of Neurophysiological Mechanisms in Predicting Susceptibility to Modeling
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Abstract

The social modeling of eating effect refers to the consistently demonstrated phenomenon that individuals tend to match the quantity of their food intake to their eating companion. The current study aimed to explore the mirror neuron system (MNS) as a causal mechanism through which this effect may occur. Factors demonstrated to enhance MNS activity were additionally explored as moderators. The current study thus aimed to examine whether: (1) external eating moderated social modeling of eating as mediated by MNS activity, (2) external eating was associated with attentional bias to food, and (3) attentional bias towards food moderated the indirect modeling effect. Under the guise of rating empathy, 93 female undergraduates at Lakehead University viewed a female video confederate “incidentally” consume either a low or high intake of chips while electroencephalogram (EEG) activity was recorded. Subsequent ad libitum potato chip consumption was quantified. Neither external eating nor attentional bias towards food moderated the hypothesized indirect social modeling of eating effect. An exploratory first- and second-stage dual moderation model, however, revealed that frontal asymmetry and body mass index (BMI) moderated an indirect effect. 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. 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 mediated social modeling of eating, as moderated by frontal asymmetry. Left frontal asymmetry enhanced MNS activity and subsequent modeling, while right frontal asymmetry attenuated MNS activity and led to a reversal of the effect.

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Social Modeling of Eating and the Mirror Neuron System: The Role of Neurophysiological Mechanisms in Predicting Susceptibility to Modeling

As the majority of one's meals are often consumed in the presence of others, the consumption of food is as much a social event as it is a biological necessity (Cruwys, Bevelander, & Hermans, 2015). As such, it is not surprising that social factors exert an influence on food intake. The influence of social modeling on how much individuals eat has been robustly established throughout the literature. Evidence consistently demonstrates that individuals eat more when their eating companion eats more, while eating less when their eating companion eats less (Cruwys et al., 2015; Vartanian, Spanos, Herman, & Polivy, 2015). 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 in attempts to delineate the mechanisms that may strengthen or weaken 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 (Cruwys et al., 2015; Vartanian et al., 2015). The robust quality of this behaviour and existent literature suggest that this unconscious process may be partially attributable to inherent mechanisms operating within the human brain.

The mirror neuron system (MNS) refers to a conglomerate of neurons in the human premotor and parietal cortices responsive to both action-execution and action-observation (Fox et al., 2015). The current study proposed that the MNS may be a viable contender to account for the robust quality of the social modeling of eating effect. The literature to date has demonstrated that the MNS is associated with behavioural imitation capabilities (Bernier, Dawson, Webb, & Murias, 2007; Oberman et al., 2005). Upon observing an action, the MNS activates

corresponding motor areas in the brain and can facilitate a response in the observer to elicit a repetition of similar actions (Ferrari, Maiolini, Addessi, Fogassi, & Visalberghi, 2005). Though previously unexplored, such a mechanism may thus feasibly underlie the tendency to model the amount of food consumed by one's companion. The posited neurophysiological role of the MNS in evoking this effect may additionally be able to account for the numerous factors found to unsuccessfully alter the strength of the social modeling of eating effect. In accordance, only factors capable of magnifying or attenuating mirror neuron activity may be able to systematically moderate this effect. The current study served as a preliminary attempt to explore whether activity in the MNS may causally account for the social modeling of eating effect, with an emphasis on attempting to establish individual-level factors which may modulate the strength of this phenomenon.

The Social Modeling of Food Effect

Heightened interest in the social modeling of eating effect is suggested by the publication of both qualitative (Cruwys et al., 2015) and quantitative (Vartanian et al., 2015) reviews of this phenomenon in recent years. Vartanian and colleagues' (2015) conducted a meta-analysis of 38 studies to quantify the effect of social models on individuals' food intake. A large modeling effect ($r = .39$) was found on the amount of food individuals consume in the presence of an eating companion. Both the qualitative and quantitative review concluded that the available evidence consistently demonstrates a tendency for individuals to consume an amount that approximates that of one's eating companion, indulging more when companions ate more and limiting when companions ate less (Cruwys et al., 2015; Vartanian et al., 2015).

The influence of social modeling on eating appears to be so ingrained that both live- and remote-confederate designs induce modeling effects (Cruwys et al., 2015). In the remote-

confederate paradigm, the model is not physically present with the observer (Cruwys et al., 2015). The remote confederate's intake may be communicated to the observer via written narrative about the amount a previous participant consumed in the situation, or through exposure to a video model (Cruwys et al., 2015). Of interest to the MNS account for the social modeling of eating effect, studies have demonstrated that individuals will model the amount of food consumed by a peer who is observed eating via video recording (Bevelander, Anschutz, Creemers, Kleinjan, & Engels, 2013; Romero, Epstein, & Salvy, 2009). In these studies, food was incidental to the task being performed by the model. Participants were engaged in the same task as the video model in the laboratory and provided access to the same food that the model was consuming (Bevelander et al., 2013; Romero et al., 2009).

Though one study reported that participants did not adjust their intake to that of a video model, definitive conclusions are limited due the chosen methodology and an indication that the manipulation was not accurately perceived by participants. Under the pretext of examining observational strategies, Hermans, Salvy, Larsen, and Engels (2012) exposed participants to a female confederate in two separate experiments. In the first experiment, the confederate ate either four winegums (i.e., pastille-type sweets), or nothing, while performing work-related tasks such as working on a computer, reading, highlighting a textbook, and stapling papers together. The participants had access to peanut M&Ms (i.e. candy-coated chocolate peanuts) while observing the model, under the rationale of making the task more pleasant. This first experiment in the two-part study concluded that participants exposed to the eating confederate did not consume significantly more than those in the non-eating confederate condition. The authors noted, however, that the participants had access to a different type of snack food and the amount consumed may not have been noticeable enough to induce an effect. In an attempt to address

these limitations, Hermans and colleagues set up Experiment 2 in which participants were offered the same food as the video model and the disparity between the amount consumed across the two intake conditions was enhanced. The confederate was shown consuming either a small portion of eight M&Ms, a large portion of 20 M&Ms, or not eating anything while watching television, reading and writing in an agenda, and having a telephone call.

Despite these methodological adjustments, participants in the large portion condition did not eat more than those in the small portion or no-intake conditions. A manipulation check revealed, however, that those in the large portion condition did not perceive that the video confederate reached for M&Ms on 10 different occasions. On average, these participants estimated that the confederate had reached to grab M&Ms from the bowl five times. By contrast, participants in the small portion condition perceived that the confederate reached a similar number of times ($M = 3.16$) to grab M&Ms. The authors note that the confederate was engaging in a number of activities throughout the duration of the video that may have drawn attention away from the confederate's eating. Once again, the eating behaviour of the confederate may not have been salient and distinct enough in the large portion condition to influence individuals' behaviour. The limitations of this study arguably do not permit the conclusion put forth by Hermans and colleagues (2012) that a video model is ineffective. Furthermore, it appears to suggest that both salience of, and individuals' attention to, the food cues are particularly important factors in order for the social modeling of eating effect to emerge.

Investigations aimed to identify moderators of this effect have been relatively unsuccessful. Variables examined include: individuals' level of restraint (i.e., active constraint of food consumption; Polivy, Herman, Younger, & Erskine, 1979), dieting status (Rosenthal & Marx, 1979), body mass index (BMI; Hermans, Larsen, Herman, & Engels, 2008), and hunger,

even when food deprived up to 24 hours (Goldman, Herman, & Polivy, 1991). None of these factors have shown a capacity to alter the magnitude of this effect. Regardless of variations evident in the aforementioned factors, individuals consistently matched their food intake with that of their eating companion. Nevertheless, a small subset of significant moderators has been found thus far. Accumulated evidence suggests that there is a greater modeling effect among women compared to men ($r = .39$ vs. $.17$) within literature to date (Vartanian et al., 2015).

Greater perceived similarity between the model and the participant has also been demonstrated to enhance the social modeling of eating effect (Cruwys et al., 2015). Specifically, individuals have exhibited a greater likelihood of modeling in-group members rather than out-group members for psychologically salient features such as university affiliation (Cruwys et al., 2012), gender (Conger, Conger, Costanzo, Wright, & Matter, 1980), and weight (de Luca & Spigelman, 1979; McFerran, Dahl, Fitzsimons, & Morales, 2010).

Although the weight status of the participant themselves has not been shown to moderate the social modeling effect (Conger et al., 1980), the literature indicates that individuals' weight status interacts with the weight status of the model to exert an effect. Normal-weight individuals were more likely to adapt their intake to a model of normal-weight appearance, rather than a model who appeared slim (Hermans et al., 2008) or a model who was obese (McFerran et al., 2010). This interaction of weight status between the model and observer further bolsters the importance of similarity with one's eating companion in eliciting modeling of food intake. Overall, both the robustness of the social modeling effect on food intake combined with the limited number of significant moderators thus far has spurred curiosity as to the mechanisms that may induce such behaviour so reliably. Cruwys et al. (2015) note that despite the repeated replication of this effect, an answer as to why modeling occurs remains relatively elusive;

however, several speculations have been advanced.

According to a normative account of the social modeling of eating effect, individuals look to others to determine how much food is appropriate to eat in order to manage the impressions of others in the absence of clear intake guidelines (Herman, Roth, & Polivy, 2003). Given that internal signals for satiety may be unreliable regulatory controls of food intake (Herman & Polivy, 2005), individuals may consciously rely on socially-derived norms to determine when to stop eating (Herman et al., 2003). This normative interpretation appears to adequately account for individuals' tendency to match the intake of others when they are merely told how much food previous individuals have consumed in a similar situation (e.g., Leone, Pliner, & Herman, 2007). However, it is debatable whether a normative account can fully explain in vivo modeling situations. Normative accounts arguably cannot adequately explain the persistence of social modeling effects even when participants have been food deprived for over 24 hours (Goldman et al., 1991). The sheer magnitude of hunger and knowledge of one's current level of deprivation would be expected to encourage greater consumption and override this social modeling phenomenon. Yet those who are food deprived for over 24 hours and paired with a minimal intake model eat an amount approximating their companion (Goldman et al., 1991). Considering that the observation of others often leads to unconscious behavioural mimicry (Hermans et al., 2012), social modeling of eating may reflect automatic mimicry processes in kind (Cruwys et al., 2015). The social modeling of eating effect then may not be dependent on a conscious decision-making process about conforming to norms, but rather on a hard-wired inclination.

Preliminary evidence implicates the importance of unconscious behavioural mimicry processes in the social modeling of eating effect. Hermans et al. (2012) 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 seconds) rather than incongruent bites (i.e. outside the 5-second interval). One may speculate that mimicry of bite frequency may account for the likelihood that one will approximately match the amount of food consumed with that of one's eating companion. The authors acknowledged that such patterns of behavioural mimicry may insinuate the role of a neural link between perception and action. Activation of one's own motor cortex via bite observation may in turn increase the likelihood of initiating a parallel action (Hermans et al., 2012).

The findings of this study indirectly corroborate evidence of mirroring networks in the human brain and arguably allude to the role of mirror neurons in this phenomenon. The MNS has been proposed to promote the development of mimicry or imitative behaviour in animals and humans alike (Iacoboni & Dapretto, 2006), and can facilitate the performance of observed motor actions (Ferrari et al., 2005). Thus, the current study proposed that activation within the MNS may elicit social modeling of food intake and mediate the relationship between the amount of food consumed by a model and that of the observer. To the author's knowledge, no studies to date have examined the potential role of the MNS in the social modeling of eating effect.

The Mirror Neuron System (MNS)

The initial discovery of mirror neurons in the Rhesus macaque brain revealed the existence of a conglomerate of neurons in the brain which fired both when the macaque performed an action and when solely observing another individual perform an action (Iacoboni & Dapretto, 2006). This correspondence in activation suggested the existence of a common neural code between perceptual and motor processes within the brain (Molenberghs, Cunnington, & Mattingly, 2009). Experimental studies first localized mirror neurons in the ventral premotor cortex, inferior parietal lobe, and part of the inferior frontal gyrus in the macaque brain using

microelectrode recordings of single neurons (Fox et al., 2015). 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 with the use of functional magnetic resonance imaging (fMRI) and other noninvasive brain imaging techniques (Fox et al., 2015). An increasing number of studies conducted in recent years have examined the human MNS through electroencephalogram (EEG) technology (Fox et al., 2015). In contrast to fMRI, EEG offers the unique capability to examine the temporal nature of activation in response to both observation and execution (Fox et al., 2015). Simultaneous temporal activation is particularly important in order to infer a correspondence between observed actions and the activation of parallel motor areas.

MNS activity is measured with EEG through 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 (Gros, Panasiti, & Chakrabarti, 2015). At rest, sensorimotor neurons fire in synchrony, producing large amplitude EEG oscillations in the mu frequency band (Pineda & Oberman, 2006). During observation or execution of an action, mirror neurons fire asynchronously, resulting in decreased amplitude and power of the mu band (Pineda & Oberman, 2006). Mirror neurons discharge more during the execution than during the observation of an action; however, similar areas of activation are evident (Iacoboni & Dapretto, 2006). This similar pattern of mu desynchronization during action and observation has been interpreted as demonstrating evidence of mirroring properties in these regions of the brain (Fox et al., 2015). A recent meta-analysis conducted by Fox and colleagues (2015) provided a comprehensive analysis of 85 EEG studies and concluded that there is consistent support for EEG mu rhythm desynchronization during both action execution ($d = .46$)

and action observation ($d = .31$). It was concluded that robust evidence indicates that altered EEG mu rhythm is a valid index of neural mirroring.

Mu rhythm desynchronization is calculated as a ratio between mu power during the condition of interest (i.e. action observation or execution) and a baseline period of activity. Calculating mu rhythm desynchronization as a ratio of change in the EEG amplitude optimally controls for variability in absolute mu power due to individual differences in scalp thickness and electrode impedance (Oberman et al., 2005). Different baseline EEG stimuli have been used throughout the literature and can theoretically alter the magnitude of desynchronization. Tangwiriyaakul et al. (2013) investigated the effect of different types of baseline stimuli (i.e. static, dynamic, quasi-static) on baseline mu power and overall desynchronization calculations as a result. Among their sample, there was evidence of high variation in the type of baseline that created maximal baseline mu power across participants. The strength of power in baseline mu rhythm recordings subsequently affected the magnitude of desynchronization demonstrated. Tangwiriyaakul and colleagues (2013) concluded that the optimal baseline measurement for studies of the MNS may vary across individuals. Conversely, Fox et al.'s (2015) meta-analysis concluded that type of baseline (i.e. static vs. dynamic; biological vs. nonbiological) utilized across EEG studies did not moderate the effect size for mu rhythm desynchronization during either observation or execution. Thus, the type of baseline utilized may be a significant point for consideration in examining and conducting studies of MNS activity. More research is necessary consequently to ascertain the optimal baseline to implement for all participants. Quantifying the ratio of change to a single baseline stimulus appears to be the best available method to minimize individual differences in the magnitude of baseline mu.

Concerns within the literature have been raised as to whether mu rhythm

desynchronization ought to be considered a valid index of mirror neuron activity, particularly calling into question whether mu suppression can be reliably distinguished from changes in alpha activity (Hobson and Bishop, 2016). Notably, mu rhythms oscillate in the same 8 – 12 Hz frequency range as alpha rhythms that culminate over the occipital cortex (Perry, Stein, & Bentin, 2011). Alpha rhythms are heavily influenced by states of expectancy and awareness (Pineda & Oberman, 2006). Suppression of alpha waves is thought to reflect greater neural activity induced by a perceptual event, with increasing demands of attention, alertness, episodic memory, and task-load linked to decreased alpha power (Perry et., 2011). It has been argued that alpha suppression, as measured by alpha activity within the occipital lobe, may be easily confounded with mu. Some researchers have suggested that evidence of mu suppression may merely reflect alterations in the attentional demands across conditions in studies of mirror neuron activity. Nevertheless, alpha and mu are considered to be distinguishable based on their topography and reactivity. Specifically, alpha activity arises in more posterior and occipital regions, while mu originates in the sensorimotor cortex in more central brain regions (Hobson & Bishop, 2016). Findings with respect to the localization of suppression effects documented in studies of the MNS have been relatively mixed.

While some studies have found evidence for suppression in both occipital and central electrode sites (e.g., Perry & Bentin, 2010; Perry, Stein, & Bentin, 2011; Perry et al., 2010), others have found that suppression effects were constrained to the central sites (e.g., Oberman et al., 2005; Oberman, McCleery, Ramachandran, & Pineda, 2007). Given that studies of MNS activity necessarily entail perceiving motion, it is possible that alpha and mu suppression may be modulated in parallel (Perry et al., 2011), rather than reflecting an artifact of attentional processes. Moreover, evidence within the literature has indicated that greater attention towards

stimuli and task demands may enhance MNS activity (Muthukumaraswamy & Singh, 2008). Such findings may indicate that greater occipital suppression would be expected among those demonstrating greater mu suppression in response to the observation of actions as a tangential, but related, phenomenon. In light of concerns, it has been recommended that analyses of the MNS via mu rhythm suppression ought to include experimental effects at both central and occipital regions to adequately understand the phenomenon under study (Perry & Bentin, 2010). Furthermore, control of attentional engagement ought to be a primary concern in mu suppression studies (Hobson & Bishop, 2016). Thus, the current study included an examination of alpha activity at the occipital sites to explore whether mu rhythm desynchronization could be evidenced above and beyond mere attention-related activation. A rudimentary index of attentional engagement was also integrated into the design of the methodological protocol to ensure that individuals attended sufficiently to all conditions presented.

The MNS and social modeling. A tentative link between the MNS and the social modeling of eating effect appears to emerge in research that has been conducted to date. Correspondence between perceived and executed actions essentially creates a neural substrate of motor simulation which may facilitate particular responses by triggering the repetition of an observed action that is already in the observer's motor repertoire (Ferrari et al., 2005). For example, activation of the MNS has been found to be associated with the phenomenon of contagious yawning, a process of behavioural mimicry that occurs relatively frequently and automatically outside of conscious awareness (Haker, Kawohl, Herwig, & Rössler, 2013). Particularly relevant to social modeling of eating, studies with macaques have shown that response facilitation occurs in the observation of eating actions. In one study, macaques who observed a conspecific eating exhibited a greater frequency of eating behaviour than those that

did not observe eating behaviour (Ferrari et al., 2005). In conjunction with the aforementioned evidence of bite mimicry among female humans eating with a companion (Hermans et al., 2012), such evidence indirectly implicates the role of mirroring neural networks in eliciting a stronger match between an observer and model's eating behaviours.

Further parallels between the MNS and social modeling of eating effect can be seen within the literature in regards to the importance of similarity. As previously noted, similarity has emerged as a significant moderator of the social modeling of food intake, whereby individuals tend to match their intake to a model who is more similar in terms of sex, weight, and age. The mirror neuron literature likewise indicates that the human MNS is more active when the model and observer are more similar to one another (Iacoboni & Dapretto, 2006). By extension, if the MNS underlies the social modeling of eating effect, factors that enhance MNS activity should subsequently enhance the tendency towards social modeling. Accordingly, the enhancement of social modeling due to similarity may be reflected at the neural level by greater MNS activity. Additional evidence bolstering the capacity of the MNS to account for the tendency towards modeling one's companions' intake can be derived from the literature on empathy.

One study reported that individuals who scored higher on a measure of trait empathy exhibited a greater tendency to match their food intake to that of their eating companion (Robinson, Tobias, Shaw, Freeman, & Higgs, 2011). The authors of the study suggested that such matching may reflect the tendency of empathic individuals to use behavioural mimicry to increase liking and rapport with their eating companion (Robinson et al., 2011). However, evidence from the MNS literature offers an alternative explanation. In light of facilitating resonance between the actions of others and oneself, the MNS has been associated with facilitating understanding of others' intentions, goals, and emotional state, capabilities which

form the foundation of empathy. Investigations have found that individuals who endorsed higher levels of empathy tended to exhibit evidence of greater activity within the MNS (Gros et al., 2015; Iacoboni & Dapretto, 2006; Kaplan & Iacoboni, 2006). This evidence suggests that the greater tendency of more empathic individuals to model the intake of their eating companion may be induced by enhanced MNS activation. Such parallels between the MNS and social modeling of eating, though not explicitly explored, provide impetus to explore the potential role of differential MNS activation in individuals' proclivity towards modeling food intake.

If the MNS underlies the social modeling phenomenon—as hypothesized—the neurologically-based activation of mirror neuron areas within the brain may also help to explain why a number of proposed moderators have counterintuitively not yielded significant effects. For example, recall that even individuals who had been food deprived up to 24 hours were demonstrated to down-regulate their intake to match a companion who ate a small amount, despite the mounting physiological urge to eat. Top-down regulation from cortical brain areas in response to environmental stimuli can override gut-based neuroendocrine signaling systems that signal hunger and satiety (Mayer, 2011). It is plausible that activation of the MNS may lead to such top-down regulatory effects to account for the lack of moderation previously found for hunger. The current study hypothesized that significant moderators of this phenomenon, such as similarity, may be limited to factors that differentially activate the MNS. To further support the MNS's influence in the expression of the social modeling of eating effect, it ought to be demonstrated that variables associated with enhanced MNS activity similarly enhance social modeling. For example, greater MNS activation has been associated with increased attention to stimuli (Muthukumaraswamy & Singh, 2008). As a result, variables that impinge on individuals' attention should theoretically be capable of influencing the tendency to engage in the social

modeling of eating effect when observing a companion eat.

External Eating

External eating is one possible moderator of the social modeling of eating phenomenon given its potential capacity to influence individuals' attention during social eating scenarios. External eating refers to an increased tendency to eat in response to external cues (Hou et al., 2011). The notion of external eating harkens back to the externality hypothesis originally put forth by Schachter (1971), who proposed that individuals who were overweight or obese were more externally controlled or stimulus-bound in their eating behaviour. A myriad of studies examining various external cues have since qualified that weight class does not directly determine one's level of responsivity towards external food cues (Herman & Polivy, 2008). Rather, it has been put forth that externality is a general response style that may be acquired biologically or through early life experience (Rodin & Slochower, 1976). Externality has been theorized to represent a relatively stable trait characteristic among individuals (Nijs, Franken, & Muris, 2009). This response tendency has been associated with individuals' vulnerability towards overeating, which may subsequently cause weight gain (e.g., Davis et al., 2007; Rodin & Slochower, 1976) and feasibly explain the greater incidence of a more external eating style among those who are overweight. Across normal weight as well as overweight or obese individuals, however, there is evidence of wide variation in individuals' level of external reactivity to food (Rodin & Slochower, 1976; van Strien, Herman, & Anschutz, 2011). Individuals notably exhibit differential tendencies towards a more external eating style. With respect to the social modeling of eating effect, the amount of food one's eating companion consumes is a salient external cue that has been found to universally influence individuals' food intake (Herman & Polivy, 2008). Those who are characterized by a greater proclivity towards

external eating may thus theoretically be more highly influenced by the amount of food their companion consumes and may be more likely to engage in this phenomenon.

External eating, the MNS, and the social modeling of food. To date, there have been no studies that have directly examined an association between the tendency towards external eating and social modeling as a determinant of food consumption. One may reasonably infer that those higher in external eating would be more sensitive to the eating behaviour of their companion, a salient external cue, and thus demonstrate a greater tendency to adjust their intake in concert with the amount of food consumed by their partner. Higher levels of external eating also exhibit a rational association with MNS activity. Numerous studies have demonstrated that external eating tends to be associated with a heightened attentional bias for food cues (e.g., Hou et al., 2011), which is in accordance with evidence that increased attention leads to an enhancement of MNS activation (Muthukumaraswamy & Singh, 2008). The tendency to eat in response to external cues has also been acknowledged to occur regardless of one's internal state of hunger or satiety (van Strien, Frijters, Bergers, & Defares, 1986). In parallel, the social modeling of eating effect has been found to occur regardless of hunger (Cruwys et al., 2015). Thus, a cogent argument can be made for the role of external eating as a moderator in the social modeling of eating effect within the proposed model.

Accordingly, the current study proposed that a greater proclivity towards external eating would elicit greater activation within the MNS, as evidenced by greater mu rhythm desynchronization. It was hypothesized that this activation would subsequently manifest as a greater tendency to engage in social modeling to match one's food intake to one's eating companion. The aforementioned hypothesis hinges critically on the enhancement of the MNS through greater attentional bias towards food stimuli among external eaters. It was thus

necessary to determine if individuals characterized by high levels of external eating in the current study demonstrated an attentional bias towards food cues. It was reasoned that if external eating was found to moderate the proposed causal association in actuality, such evidence could further elucidate the mechanism of action through which external eating operated to alter the strength of the social modeling effect through MNS activation.

Attentional Bias to Food Cues and the Social Modeling of Food

Despite the number of moderators investigated for their capacity to influence the social modeling of eating effect to date, there is a surprising paucity of evidence examining the role of attention in this phenomenon. The aforementioned study by Hermans and colleagues (2012) in which a video model was unsuccessful in eliciting the effect clearly suggests that the salience of, and attention towards, food cues may be particularly important factors in determining whether or not social modeling of food intake occurs. Bandura's 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 (Bandura & Jeffery, 1973). 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 stimuli would then hypothetically be expected to enhance the likelihood of the occurrence of modeling.

Attentional bias towards food, or the degree to which individuals exhibit selective attention towards food cues, may lead to greater modeling of eating in social scenarios. Attentional bias towards food provides an index of individuals' differential reactivity to food reward (Hou et al., 2011). Consequently, hypersensitivity towards the reward value of food cues may increase vulnerability towards overeating (Brignell, Griffiths, Bradley, & Mogg, 2009), or eating regardless of one's level of physiological hunger. To the knowledge of the author, only one study

has been conducted to specifically examine whether increased attentional bias towards food leads to a greater tendency to model one's consumption on that of one's eating companion. Hermans and colleagues (2013) used an eye-tracking paradigm to assess participants' attentional bias towards food as they watched clips involving food consumption from the movie "Eat Pray Love" (© 2010 Columbia Pictures Industries, Inc.). Attentional bias was determined via the number of fixations, gaze duration, and latency of initial fixations on food during the observed portions of the film. Hermans and colleagues (2013) acknowledged that in assessing attentional bias, it was important to make a distinction between food cues and dynamic eating cues. While food cues can be thought to refer to characteristics of the food itself (i.e., the sight, smell, or taste), dynamic eating cues would refer to stimuli involving individuals' interactions with food (i.e., the sight of someone eating). It was proposed that attention towards dynamic food cues would be a more ecologically valid measure to approximate real-life eating scenarios.

In this study by Hermans and colleagues (2013), the measurement of attentional bias for dynamic food cues occurred in an initial individual laboratory session. Participants subsequently returned for a second laboratory session a few days later, during which they were paired with a female confederate to explore the social modeling of eating behaviour (Hermans et al., 2013). When individuals' tendency to attend to dynamic food cues was explored as a moderator of the amount of food participants had consumed during the subsequent laboratory session, their level of attentional bias did not significantly moderate their proclivity to model the female confederate. While this lack of association would suggest that attentional bias towards food does not influence one's tendency to engage in social modeling, there are a number of methodological considerations to note within this study that may account for this finding.

Though more ecologically valid, the authors mentioned that dynamic eating cues may have

lower attention-grabbing power than passive food cues (Hermans et al., 2013). As a result, it is possible that other external factors or characteristics of the particular scenes shown in the study may have influenced individuals' eating behaviour instead. The scene, or image, complexity may have attenuated the strength of the attentional bias evident in such a scenario. Miller & Fillmore (2010) examined the role of image complexity on attentional bias towards alcohol-related images among drinkers using a visual-probe task. Complex images were those depicting real-life scenes that involved alcohol, such as a party in which individuals were consuming alcohol. The simple images displayed a solitary alcoholic beverage. Results from this study indicated that an attentional bias was only evident for the simple alcohol images. Overall, response times (RTs) to detect probes replacing all complex images was slower than in comparison to simple images. The authors concluded that this finding suggested that complex images may have been more taxing to process cognitively, thereby reducing evidence of an attentional bias. Eye-tracking evidence also demonstrated that participants spent less time fixating their focus and more time scanning the complex images as compared to simple images (Miller & Fillmore, 2010). It may be theorized that when individuals must orient themselves to a scene displayed on screen quickly, the stimuli of interest may be less apparent to grab one's attention.

Real-life eating scenarios would arguably be better classified as complex scenes. Thus, dynamic food cues such as those used by Hermans and colleagues (2013) may provide a more appropriate assessment of attentional bias towards food as a predictive measure of individuals' behaviour in vivo. The context of viewing such scenes, however, ought to be considered as well. In a true social eating scenario, there is no need for individuals to rapidly orient to what is occurring in the scene in front of them. The centrality of food to this scenario is more readily apparent and individuals would generally be more attuned to their eating companion while

filtering out extraneous stimuli prior to the presentation of food. The social nature of such an interaction would warrant greater focus on the proximal stimuli to the exclusion of distracting stimuli in one's surroundings. Relatively more simple stimuli may thus be more appropriate to utilize in an assessment of attentional bias towards food to examine its relevance to the social modeling of eating effect.

With respect to dynamic food cues, there is an additional lack of clarity as to whether individuals would be more likely to attend to the eater or to the food in social eating situations. The sight of one's eating companion necessarily involves exposure to the food being consumed. Hermans et al. (2013) thus argued that dynamic eating cues cannot be entirely separated from food cues. As such, assessing attentional bias towards dynamic eating cues cannot disentangle the independent contributions that properties of the food itself versus the sight of someone eating or interacting with food may have on determining the extent of one's attention. An additional consideration in interpreting the results of Hermans and colleagues (2013) and their conclusion that attentional bias did not moderate the social modeling of eating effect is the use of a Hollywood movie as the dynamic eating stimulus. It is possible that individuals' familiarity with the movie or familiarity with the face of the famous actress, Julia Roberts, may have influenced the degree of attention that participants allotted to the film.

Previous literature has shown that processing highly familiar faces requires less attention to elevate processing above a recognition threshold in facial identification tasks (Jackson & Raymond, 2006). It should be noted that the procedural description provided by Hermans and colleagues (2013) did not explicitly indicate what participants were instructed to do when told to watch the film clip. If participants were simply told to watch the clip while their attention was to be assessed with no particular directive, it is possible that individuals may have disengaged from

the stimuli and engaged in more passive viewing, particularly if otherwise engaged mentally.

Further complicating the conclusions derived by Hermans and colleagues is the unexamined role of hunger during the attentional bias task.

Participants' subjective hunger ratings were given prior to the second laboratory session in which they were expected to model the food intake of the confederate and controlled for in statistical analyses. Hermans and colleagues (2013), however, did not provide a measure of participants' hunger prior to administering the attentional bias task. Appraisal theories suggest that the extent to which a stimulus is relevant to an individual's goals or well-being determines the attention that is allocated to particular stimuli (Tapper, Pothos, & Lawrence, 2010). All things considered, food is more motivationally relevant for an individual who is hungry relative to an individual who is well-sated (Tapper et al., 2010). Given the heightened biological value of food stimuli when depleted, hungry individuals would be expected to allocate greater attention towards food (Tapper et al., 2010).

Previous studies have indicated that increased subjective hunger was associated with enhanced attentional bias towards food-related words (Mogg, Bradley, Hyare, & Lee, 1998), as well as greater attentional bias towards photographs of food, regardless of whether the food was appetizing or bland (Tapper et al., 2010). Rather than capturing a stable or characteristic tendency to display an attentional bias towards food cues, the attentional bias measured during the first portion of the study may have solely reflected the state motivational relevance of food as influenced by participants' level of hunger at the time of the laboratory session. Ergo, this initial measure of attentional bias, if reflective of variable hunger states, could not be expected to reliably exhibit a consistent association with participants' subsequent modeling behaviour on a later occasion. Without a mechanism through which state variability could have been controlled,

it is unlikely that the attentional bias towards food measured in the first experiment was purely indicative of one's trait tendency to attend to food cues. It is perhaps unsurprising then to note that social modeling behaviour during the subsequent laboratory session a few days later was not significantly associated with attentional bias. Thus, although evidence from Hermans and colleagues' (2013) study would suggest that attention does not moderate the effect of model's consumption on participants' food intake, the extent to which such conclusions may be replicated in alternative circumstances is unclear. Collectively, these findings imply a need to clarify the potential association between attention and the social modeling of eating effect.

Attentional bias and external eating. There is a notable lack of research into the role of attention in the tendency to model a companion's food intake. However, a number of studies have demonstrated that external eating, purported by the current study to enhance the social modeling of eating phenomenon, is associated with enhanced attentional bias towards food cues (e.g., Brignell, Griffiths, Bradley, & Mogg, 2009; Hepworth, Mogg, Brignell, & Bradley, 2010; Hou et al., 2011). A small number of previous studies have utilized the modified Stroop task as a measure of attentional bias to food cues, which compares the time taken to colour-name food-related and control words. However, Stroop tasks can be difficult to interpret, as interference effects can be reflective of a bias either towards or perhaps away from food words (Hepworth et al., 2010). The visual-probe task, by contrast, provides a more direct measure of the allocation of visual attention to food cues (Hepworth et al., 2010). Pictorial stimuli additionally have better ecological validity than simply using food words (Brignell et al., 2009). Thus, visual-probe tasks appear to be a better-suited method to assess attentional bias in this context. Though dynamic food cues proffer the best ecological validity, as noted earlier, it may be difficult in such instances to disentangle attention towards features of the food cues and towards those eating the

food.

In a visual-probe task, each trial begins with the presentation of a fixation cross for 500 ms which is subsequently replaced by the simultaneous presentation of two pictures (e.g. a food-related picture and a control picture) that appear on opposite sides of a screen (Hepworth et al., 2010). Following presentation on the screen for a predetermined duration of time, the pictures disappear from the screen and are replaced by a dot, the target probe, in the location of one of the previously shown pictures. Participants must indicate the location of the probe as quickly as possible by pressing either a left or right arrow on the keyboard (Hepworth et al., 2010). An attentional bias to food cues would be indicated by faster RTs to target probes replacing food versus control pictures (Brignell et al., 2009). When a probe appears in a location that is already attended to, the rationale is that one's attention would not need to be redirected to locate the probe and thus, responses to such probes would typically occur faster than when responding to probes appearing in unattended locations (Brignell et al., 2009).

In the literature concerning external eating and attentional bias, two different stimulus durations have typically been used: 500 ms and 2,000 ms. These time durations have been previously utilized in research designed to assess the time course of attentional bias for both appetitive and aversive motivational states (Hepworth et al., 2010). The attentional bias at 500 ms has been theorized to be reflect attentional enhancement due to greater initial orienting towards the stimuli, with evidence that this measure of bias is positively correlated to the direction of individuals' initial shift in gaze (Hepworth et al., 2010). By contrast, a duration of 2,000 ms provides greater opportunity for participants' attention to shift between the pictures, and thus is thought to provide a measure of the maintenance of attention towards stimuli (Hepworth et al., 2010). Two previous studies to date that examined attentional bias among

external eaters found no significant main effect or interaction of picture duration. However, the current study included both durations to further elucidate whether the attentional bias among external eaters may be differentially associated with initial orienting or maintenance of attention among a sample of female undergraduate students. As noted, the role of attention is central to the proposition that external eating may enhance the social modeling of eating effect through mu rhythm desynchronization. To fully substantiate the importance of external eating as a moderator, it was thus critical to include an assessment of attentional bias towards food cues in the present study.

Approach Motivation, External Eating, and Attentional Bias to Food

Attentional bias towards food, and more distally external eating, may reflect a manifestation of a constitutionally- and approach-based motivation. Research has demonstrated that individuals tend to exhibit stable characteristic differences in motivational bias. This motivational bias may be expressed as either a proclivity to engage in approach-motivated behaviours towards positive stimuli or avoidance-motivated behaviours away from negative stimuli (Tomer et al., 2014). Attentional bias towards food and external eating may feasibly represent trait expressions of an underlying tendency to approach appetitive stimuli. Previous studies have indicated that external eating is associated with a systematic cognitive bias towards food-relevant information, with respect to not only bias in attention, but also with respect to an approach tendency as well (Brignell et al., 2009). Using a stimulus-response compatibility task, a task sensitive to approach biases towards appetitive stimuli, Brignell and colleagues (2009) demonstrated that those higher in external eating displayed a greater approach tendency for food cues than those in a low external eating group. It has also been shown that approach-motivated positive affective states can cause a narrowing of cognition and attention (Gable & Harmon-

Jones, 2010). Whereas attentional bias and external eating are behavioural manifestations suggestive of an approach-motivation, this motivational bias has been found to exist at a neurological level represented by differential lateralization of frontal brain activity.

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. Using EEG, frontal asymmetry is defined as the difference between alpha activity within the left and right frontal sites (Smith, Reznik, Stewart, & Allen, 2017). There is a longstanding literature that suggests that frontal asymmetry reflects an underlying propensity towards certain emotional (i.e. positive or negative) or motivational (i.e. appetitive/approach or avoidant) trait response tendencies (Smith et al., 2017). 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 withdrawal (Pizzagalli, Sherwood, Henriques, & Davidson, 2005) or avoidance of aversive stimuli (Smith et al., 2017). Corroborating this notion, it has been demonstrated that greater left frontal activity is associated with a greater bias to respond to reward-related cues (Pizzagalli et al., 2005).

Furthermore, evidence suggests that observed differences in motivational response tendencies exemplified by those who differ in relative frontal asymmetry may be linked to an asymmetry of frontal dopamine binding. Greater dopamine binding has notably been documented within left frontal regions (Tomer et al., 2014). Such findings can help to account for the identifiable magnification of reward responsivity associated with lateralization of activity to the left frontal cortex. Moreover, it has recently been established that greater left frontal activity during reward anticipation converges with fMRI evidence of increased blood

oxygenation level-dependent (BOLD) activity in brain regions associated with reward (i.e., the left anterior cingulate cortex, medial prefrontal cortex, and left orbitofrontal cortex; Gorka, Phan, & Shankman, 2015). These regions are implicated in the mesolimbic dopaminergic pathway responsible for the processing of rewards. This further substantiates the contention that asymmetry in activity across frontal brain regions is reflective of individuals' response tendency to rewards associated with dopamine release, such as food.

Of particular interest to the exploratory analyses within the present study, it has additionally been demonstrated that greater relative left frontal activity is associated with attentional narrowing towards appetitive stimuli (Harmon-Jones & Gable, 2009). Thus, it is plausible that those who demonstrate greater attentional bias towards food may theoretically do so as a function of possessing greater left frontal asymmetry. Furthermore, those who endorse higher levels of hedonic hunger or drive for highly palatable foods have been shown to exhibit greater left frontal asymmetry (Winter et al., 2016). External eating similarly refers to a drive to consume palatable food for hedonic reasons in the absence of hunger. Both external eating and attentional bias towards food may thus reasonably suggest an underlying neural vulnerability towards the manifestation of approach-motivated behaviour resulting from left frontal asymmetry. In addition to the hypothesized links with external eating and attentional bias towards food, the literature reviewed here suggest that frontal asymmetry may theoretically hold the capacity to influence the MNS. In light of statistical trends emerging from the data, frontal asymmetry was examined in exploratory analyses as a potential moderator of this effect.

The Current Study

Fundamentally, the current study sought to replicate the robust social modeling of eating effect found in the literature to further substantiate the finding that individuals tend to eat more

when observing an individual who eats more and eat less when the observed model eats less (Cruwys et al., 2015; Vartanian et al., 2015). The primary objective of the current study, however, was to elucidate whether MNS activation, a neurophysiological mechanism, may underlie this social modeling of eating effect. Activation of the MNS in response to the observation of eating may present a plausible answer to the elusive question as to why this effect emerges with such consistency and could perhaps clarify why previously explored moderators have not yielded significant results. The current study aimed to examine the moderating effect of external eating, proposed to be a proxy for heightened attentional bias towards food, to the enhancement of the mediated relationship between model and observer consumption through mu rhythm desynchronization. In order to further delineate whether attentional bias was responsible for the purported enhancement of social modeling among external eaters, attentional bias towards food was assessed via a visual-probe task. The current study aimed to replicate previous studies demonstrating an association between external eating and attentional bias towards food (Brignell et al., 2009; Hepworth et al., 2010; Hou et al., 2011).

Previous literature has indicated that it may be necessary to distinguish between attentional bias towards food cues and dynamic food cues, or eating cues, in order to understand the influence of attentional bias on the social modeling of eating effect. To do so, the current study used four different categories of pictorial stimuli: food-related cues (e.g., spaghetti), eating-related cues (e.g., a hand holding a fork with spaghetti), non-food cues (e.g., a tissue box), and non-food human cues (e.g., a hand taking a tissue from the box). Individuals tend to prioritize animate environmental stimuli and allocate greater attention towards animate stimuli during attention tasks (Altman, Khislavsky, Coverdale, & Gilger, 2016). Consequently, in order to minimize inherent bias towards the eating-related cues due to the inclusion of human hands,

these photos were contrasted with non-food cues that also included hands interacting with neutral non-food stimuli. Finally, the current study sought to explore whether attentional bias towards food itself would moderate the mediated relationship between model consumption and participant consumption through mu rhythm desynchronization.

Based on the literature review conducted, the current study proposed the following three hypotheses:

- 1) External eating would moderate an indirect relationship between model consumption and participant consumption through mu rhythm desynchronization, such that the amount of food consumed by the model would be more strongly related to the degree of mu rhythm desynchronization among more externally-controlled eaters (see Figure 1), thereby resulting in greater modeling;
- 2) Those higher on external eating would exhibit a greater attentional bias towards food cues, and;
- 3) Attentional bias towards food would moderate the indirect effect between the model consumption on participant consumption through mu rhythm desynchronization, such that the amount of food consumed by the model would be more strongly related to the degree of mu rhythm desynchronization among those more attentive to food cues (see Figure 2), thereby enhancing modeling of food intake.

Method

Participants

In the literature, greater effect sizes of social modeling on food intake have been found among females compared to males (Vartanian et al., 2015), and perceived similarity between the observer and the model has been acknowledged to be a critical factor in the occurrence of social

modeling. The current study therefore included only female participants with the use of a female confederate as the model to maximize perceived similarity. A total of 107 female participants were recruited from Lakehead University's Department of Psychology undergraduate participant pool at the Thunder Bay campus through the SONA system (see Appendix A for SONA study description). Participants ranged in age from 17 to 36 ($M = 20.01$, $SD = 3.60$). The majority of the sample self-identified their ethnicity as Caucasian (71.0%), with the remainder of the sample split amongst Aboriginal (7.5%), South Asian (7.5%), African-Canadian (3.7%), East Asian (1.9%), Middle Eastern (1.9%), and "Other" (4.7%) ethnicities. Of the total sample, participants' BMIs ranged from 17.09 to 44.61, with a mean of 24.76 ($SD = 4.62$). All participants had normal or corrected-to-normal vision.

To participate, females were also required to be non-smoking, not currently taking any cold or hypertensive medication, and right-handed. Differences in hemispheric laterality of parietal-premotor network-based coherence patterns have been noted in previous studies between right- and left-handed individuals (Kelly, Mizelle, & Wheaton, 2015). Inclusion of only right-handed individuals was thus done to minimize confounding inherent to the aggregation of EEG data from left- and right-handers. Participants were also told to refrain from exercising, drinking caffeine, or eating 2 hr prior to the laboratory session and to abstain from alcohol 12 hr prior. The experimental procedure was approved by the Lakehead University Research Ethics Board and informed consent was obtained prior to the experiment's commencement. Participants received two bonus points for participating in the study to count towards their final grade in an undergraduate course eligible to receive bonus points.

Of the total 107 participants who completed the experimental protocol, fourteen participants 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 the attentional assessment questions correctly following the baseline and eating view video recordings of central interest to examining the hypotheses. One of these participants also answered 96% of trials on the visual-probe task incorrectly with a 50% chance of a correct response for each trial, suggesting that their responses across all study tasks had questionable integrity. It was concluded that these participants had not adequately attended to the stimuli and they were thus excluded from subsequent hypothesis testing. EEG data were additionally examined for outliers, defined as a z score of ± 3.29 (Field, 2013). Based on raw EEG spectral power, five participants were removed for excessive distortion in the EEG signals as indicated by extreme μV^2 values within the recording blocks. As the inclusion criteria explicitly required right-handed individuals, two additional participants were excluded on the basis of a laterality quotient suggestive of ambidextrous handedness. The final remaining sample utilized in statistical analyses consisted of 93 participants.

Materials

Demographics questionnaire (Appendix I). Participants completed a demographics questionnaire to collect information as to their age, marital status, ethnicity, declared program of study, school enrolment status, whether they were currently taking any prescribed or over-the-counter medications, and any dietary restrictions that they may have had (e.g., vegan, vegetarian, etc.).

Edinburgh Handedness Inventory – Short Form (Veale, 2014; Appendix J). The Edinburgh Handedness Inventory – Short Form (EHI – Short Form) is a brief, 4-item version of the original 10-item EDI. The measure is designed to assess handedness, or one's preference to use one hand with greater frequency than the other when using certain objects or performing

given daily activities (Veale, 2014). The 4-item version offers greater brevity of administration, as well as empirically-validated factorial validity, and clearer instructions than its predecessor. The EDI – Short Form was administered to verify handedness of participants. The measure results in laterality quotients ranging from -100 (left-handed) to 100 (right-handed). Self-reported handedness ranged from laterality quotients of -25.00 to 100.00 ($M = 88.90$, $SD = 18.99$), indicative of a predominantly right-handed sample. Those who received a laterality quotient between -40 and 40 inclusive were deemed to be ambidextrous, showing no distinct hand preference. In accordance with evidence of differences in the lateralization of parietal-premotor coherence (Kelly et al., 2015), 2% ($n = 2$) of the total sample of participants was excluded on the basis of ambidextrous handedness.

Grand Hunger Scales (Grand, 1968; Appendix E). Prior evidence has demonstrated that increased subjective hunger is associated with an enhanced attentional bias towards food-related stimuli (Mogg et al., 1998). Hunger could also affect the amount of food an individual may consume when provided with ad libitum access for consumption. Participants were thus required to rate their hunger by filling in the Grand Hunger Scales (Grand, 1968) prior to the presentation of food and the completion of all laboratory tasks. The Grand Hunger Scales have been used in previous studies of attentional bias to food cues (e.g., Brignell et al., 2009; Hepworth et al., 2010; Hou et al., 2011). Four indices of hunger are assessed by the measure: the number of hours since last eating, estimated to the nearest 15 minutes; a rating of their subjective hunger on a 7-point Likert scale anchored by “*not hungry at all*” and “*extremely hungry*”; a rating of the amount of their favourite food they would be able to eat at the present time on a 6-point Likert scale anchored by the points “*none at all*” and “*as much as I could get*”; and an estimate of the time duration until their next expected meal, estimated to the nearest 15 minutes (Grand, 1968).

A composite score for the Grand Hunger scales was not derived given the discrepancy in the weighting of each numeric index. Rather, each of the four hunger items were separately analyzed for their potential impact on participant consumption and attentional bias. Additional questions to assess level of thirst, fatigue, and discomfort were affixed to these scales in order to uphold the ruse of the study, each measured on a 7-point Likert scale from “*none at all*” or “*not thirsty/tired at all*” to “*extreme discomfort*” or “*extremely thirsty/tired.*”

Food intake. Participants in each condition were provided access to a preweighed 56-gram bowl of LAY'S® Classic chips to eat (i.e., approximately 30-40 chips). Any uneaten chips in the bowl were weighed at the end of the session using a Denver Instrument Summit Series S-2002 laboratory scale. To quantify how much the participants ate in grams, the weight of the remaining chips was subtracted from the initial weight of the chips.

Dutch Eating Behaviour Questionnaire (DEBQ; van Strien, Frijters, Bergers, & Defares, 1986; Appendix K). The DEBQ is a 33-item self-report questionnaire comprised of three subscales designed to assess Restrained, Emotional, and External eating. The Restrained eating subscale consists of 10 items to assess restrictive control of food intake (e.g., “Do you try to eat less at mealtimes than you would like to eat?”). Emotional eating is assessed by 13 items and provides a measure of individuals’ tendency to respond to emotional arousal states such as angry, fear, or anxiety with excessive eating (e.g., “Do you have a desire to eat when you are emotionally upset?”). The External eating subscale, comprised of 10 items, assesses proclivity to eat in response to external food-related stimuli, regardless of one’s internal state of hunger or satiety (e.g., “If you see others eating, do you also have the desire to eat?”). Items are responded to on a 5-point Likert scale, ranging from 1 (*never*) to 5 (*very often*). The total score and scores on each of the subscales were obtained by dividing the sum of the items by the total number of

items contained on that scale. Preliminary validation of the DEBQ revealed high internal consistency of each subscale, with Cronbach's alpha ranging from 0.80 to 0.95, as well as high factorial validity. Though external eating was of central interest, all three subscales were completed, as the subscales have been shown to significantly correlate with one another (van Strien, Frijters, Bergers, & Defares, 1986). In the current study, the DEBQ subscales exhibited internal consistency comparable to that of previous studies, with Cronbach's alpha of .84 on the External Eating subscale, .90 on Restrained Eating, and .94 on Emotional Eating.

The external validity of the DEBQ - External Eating subscale has been called into question in a study by Jansen and colleagues (2011) where *low* scorers on external eating ate more after food cue exposure whereas high scorers did not. However, the conclusions derived from that study about the validity of the External Eating subscale may be questionable. While the increase in high external eaters' intake in the food cue exposure condition relative to a control condition did not reach statistical significance, they ate more in both the cue exposure and control conditions than those who scored low on external eating in terms of overall food intake (Jansen et al., 2011). Thus, regardless of experimental manipulation of food cue salience, high external eaters were arguably more responsive to food cues overall when presented with food. The cue exposure condition, which involved taking food items to the nose and smelling them intensely for 10 min, may have lacked ecological validity (van Strien, Herman, & Anschutz, 2012). Arguably, demand characteristics and socially desirable responding may have further confounded the results as well (van Strien et al., 2012), as such a scenario may heighten the artificial nature of the task and remind participants that their behaviours are being monitored.

As external eating has been associated with increased attentional bias to food (Hou et al., 2011; Nijs, Franken, & Muris, 2009), conditions aimed to explicitly enhance the salience of food

cues may have a limited capacity to enhance external eaters' attention to food cues. External eaters are already more likely to attend to food when it is present. Ergo, increasing cue salience further may reap minimal attentional enhancement among such individuals. In contrast, individuals who score lower on external eating, and who are generally less attentive to food cues, may exhibit a more substantial increase in food intake as a result of an experimental manipulation designed to heighten attention to food. van Strien, Herman, and Anschutz (2012) additionally suggested that Jansen and colleagues' (2011) use of a median split for classifying low versus high external eaters may have led to misclassification. In their sample, there was a relatively insufficient proportion of participants exhibiting particularly low values on external eating. In response, van Strien and colleagues (2012) conducted an additional study and found that when more extreme scores on the subscale were used to classify individuals, high external eaters in a food commercial condition ate more than high external eaters in a neutral commercial condition, whereas low external eaters did not eat more in one condition than the other. The DEBQ – External eating subscale has also been shown to predict level of food craving among individuals (Burton, Smit, & Lightowler, 2007). There is thus sufficient evidence to suggest that the External Eating subscale of the DEBQ provides a satisfactory measure reflective of individuals' external eating behaviour.

Contrived “Empathy Rating” scale (Appendix F). Participants were required to fill in a contrived “Empathy Rating” scale after watching the video of the model (discussed below) in order to bolster the cover story masking the true aim of the study. The scale was created solely to have face validity. To enhance believability, the construction of the scale was based on available measures designed to assess empathy. The Communication of Affect Receiving Ability Test (CARAT; Buck, 1976) for example, assesses individuals' ability to decode affect in others. This

measure was used to generate a question about how pleasant the model's emotional reactions were, as well as what genre of film they believed the model was watching. In addition, the Questionnaire of Cognitive and Affective Empathy (QCAE; Reniers, Corcoran, Drake, Shryane, & Völlm, 2011) assesses one's self-reported level of empathy. One question from this measure assessing emotional detachment while watching films was reformulated to refer to an observed individual. A question was also included to examine how similar the participant considered herself in relation to the model, as similarity has been reported to be an important factor in the social modeling of eating effect (Cruwys et al., 2015).

Toronto Empathy Questionnaire (TEQ; Spreng, McKinnon, Mar, & Levine, 2009; Appendix G). The TEQ was administered to bolster the cover story which indicated that the study was interested in how one's own level of empathy may influence mental judgments about others' empathy. The TEQ is a 16-item self-report measure that conceptualizes empathy as a unidimensional factor (Spreng et al., 2009). Items are responded to on a 5-point Likert scale, ranging from "*never*" to "*always*." The scale has been found to demonstrate good internal consistency, $\alpha = .85$, adequate test-retest reliability, $r = .81$, and strong convergent validity, correlating with behavioural measures of social decoding and other self-report measures of empathy. The TEQ exhibited high internal consistency in the current study, $\alpha = .82$. The TEQ was not of interest to the central hypotheses. It was used solely for purposes of bolstering the fabricated aim of the current study.

Film Task Questionnaire (Appendix H). Following the presentation of the film clip, participants filled out a questionnaire to provide their reactions to both the film clip and the food presented during this portion of the laboratory session. Participants were asked to indicate how much they liked the chips that were offered while they watched the film clip on a 6-point Likert

scale anchored by “Strongly disliked” and “Strongly liked.” Their familiarity with the film clip was specified by checking one of the following options: “I have never watched the movie before,” “I have watched clips of the movie, but have never seen the film in its entirety,” “I have watched the movie once before,” or “I have watched the movie more than once.” This post-film questionnaire also asked participants to indicate the intensity of various emotions that they experienced while they watched the film clip on a 9-point Likert scale anchored by “Not at all/none” to “Extremely/a great deal”; how pleasant they would rate the feelings they had during the film on a 9-point Likert ranging from “Unpleasant” to “Pleasant”; and whether they looked away or closed their eyes during any scenes.

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 (Oberman et al., 2005). 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 electrooculogram (VEOG) activity to assess eye blinks and optical artifacts over the course of the recording.

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 $\pm 100 \mu\text{V}$

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. Resulting EEG spectral data were manually transferred into SPSS v.23 for analyses. Participants with fewer than 25% of the maximum number of possible epochs in the baseline and eating-view recording blocks were excluded from analyses. This resulted in the removal of three participants (2.8% of participants). Due to technical recording difficulties with the recording, one participant was excluded on the basis of extreme μV^2 values across all recording blocks, and another three were excluded due to extreme μV^2 values isolated to the eating-view block.

An index of mu rhythm desynchronization was calculated in SPSS as the ratio of change in the 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. Though EEG data had also been recorded in the movie-view block, this data was largely characterized by extreme μV^2 values due to the artifacts created by the consumption of food. Therefore, this data was not utilized in subsequent analyses. Given that ratio data are non-normal as a result of lower-bounding, the ratio was log transformed for analyses, as is customary when examining mu suppression (Perry & Bentin, 2009). A log ratio of less than zero indicates suppression, values of zero indicate no suppression, and values greater than zero are reflective of enhancement (Perry & Bentin, 2009). The log ratio data for mu rhythm desynchronization was utilized as the mediating variable in

analyses of the moderated mediation hypotheses (hypotheses one and three) and the exploratory model. Suppression ratios were additionally calculated for electrode sites F3, F4, O1, and O2 to enable an examination of the localization of the effect. These ratios were computed in an identical fashion to the mu rhythm ratios. The power in the 8-12 Hz frequency band generated over each of these sites in the eating-view block was divided by the power in this band during the baseline. It is convention to refer to suppression in the 8-12 Hz frequency band recorded over central sites as mu suppression (Gros, Panasiti, & Chakrabarti, 2015), while activity at the frontal and occipital sites in this band is referred to as alpha suppression. This terminology is upheld in the current study. Findings presented across scalp sites thus refer to mu/alpha suppression.

Visual-probe task stimuli. Four different categories of pictorial stimuli were viewed by the participant during the visual-probe: 20 food-related cues (e.g., strawberries), 20 eating-related cues (e.g., an individual holding a strawberry), 20 non-food cues (e.g. a hammer), and 20 non-food human cues (e.g., an individual holding a hammer). All food and non-food items were presented on the same shallow, white serving dish. The eating-related and non-food human cues depicted an individual holding the item in the foreground of the image with the serving dish present in the background of the image. The same female hand model was used for all eating-related and non-food human photographs. The model's hands were free of any jewellery or nail polish to ensure minimal attentional distraction.

In order to determine the food stimuli that would comprise the food-related and eating-related cues, the current study examined the normative data contained within the *food-pics* database. The *food-pics* database consists of 568 food images and 315 non-food images made available under the creative commons license to facilitate standardization of research on food cues (Blechert, Meule, Busch, & Ohla, 2014). Accompanying these images are normative

participant ratings of valence, arousal, palatability, complexity, and craving for each given food image. The DEBQ – External Eating scale has been previously found to predict level of food craving (Burton, Smit, & Lightowler, 2007). It has also been demonstrated that individuals who report high levels of food craving (i.e., external eaters) tend to exhibit a stronger approach tendency towards food (Brockmeyer, Hahn, Reetz, Schmidt, & Friederich, 2015). Thus, to maximize the likelihood that all individuals would crave and attend to the food items utilized, normative craving ratings guided the selection of food stimuli. Food craving was indicated by participants' response on a visual analogue scale to the question, "How much would you like to eat this food right now if it was in front of you?" anchored by "not at all" to "extremely," scored from 0 – 100 (Blechert et al., 2014). Only those images that received a normative rating of 50 or higher were considered for inclusion.

Another important variable considered in the selection of the specific food items for the visual-probe task was image complexity. Complexity was explained to participants as images characterized by "many components, details and subobjects" (Blechert et al., 2014, p.5) and rated on a visual analogue scale anchored by "very low" to "very high," scored from 0 – 100. Only food items receiving a normative complexity rating below 50 were considered for inclusion to minimize complexity. Furthermore, food items that contained meat were also excluded in order to minimize the effect that a vegetarian or vegan diet may have on individuals' attention towards food items in the current study. Thus, the food items selected to be photographed for the visual-probe task were those that received the highest ratings on food craving in conjunction with a low complexity rating within the normative female vegetarian sample in the initial establishment of the *food-pics* database.

The images were photographed specifically for the task in the laboratory prior to study

commencement using an Olympus E-M5 camera against a white background. The photographs were edited in Adobe Lightroom (Adobe Lightroom, Version 6.0) to have a resolution of 1920 x 1080 pixels (240 dpi, sRGB colour format). Photographs were edited to ensure standardized appearance with respect to the brightness, exposure, shadowing, and white balance. The pictorial stimuli were shown in visual-probe trials consisting of four different types of picture pairs:

1) food-related cues versus non-food cues, 2) eating-related cues versus non-food human cues, 3) non-food cues vs. non-food human cues, and 4) non-food cues vs. non-food cues. For each picture category, the 20 pairs of stimuli were shown four times each throughout the 320 trials.

RT data from the visual-probe task were transferred from Inquisit™ summary data for each participant into SPSS. RT data from trials with incorrect responses (2.7% of the data) were excluded from analyses. As is customary in the literature, RTs less than 200 ms and 2,000ms were considered outliers and excluded accordingly (0.2% of the data). Two attentional bias scores were calculated for each participant: an attentional bias towards food score and an attentional bias towards eating score. The attentional bias towards food score was calculated by subtracting participants' mean RT to probes replacing food-related cues from the mean RT to probes replacing non-food cues based on previous methodology (i.e. Hou et al., 2011; Brignell et al., 2009; Hepworth et al., 2010). A positive value indicated faster response times towards probes replacing food-related cues, indicative of an attentional bias towards food cues. The attentional bias towards eating score was calculated by subtracting participants' mean RT to probes replacing eating-related cues from the mean RT to probes replacing non-food human cues. Similarly, a positive value indicated attentional bias towards eating cues.

Video Stimuli

Baseline video. The present study opted to utilize a baseline measure of mu activity

derived from an eyes-open condition in which participants watched a video of dynamic, non-human stimuli. Barry et al. (2007) reported evidence that power reductions in across-scalp averages occur in alpha and beta frequency bands from eyes-closed to eyes-open conditions. An eyes-open condition was decided upon to ensure that mu desynchronization was not artificially inflated due to variations in EEG activity across eyes-closed to eyes-open conditions. The baseline video was comprised of two alternating video clips sourced from YouTube™. The two videos were edited and combined into one 5-min video using Movie Studio Platinum (version 12.0). The first video clip depicted a cat playing with young 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 of the animals. These clips were chosen in accordance with Hobson & Bishop's (2016) assertion that the control stimulus should not activate the MNS. It has been noted that even stimuli illustrating the opening of a flower, as some studies have previously utilized, may arguably be imitable by imagining the opening of a closed hand, for example. Tangwiriyasakul and colleagues (2013) 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 video stimuli used in the present study were thus chosen to be visually appealing enough to maintain participants' attention for the 5-min recording duration. It was reasoned that alternating the two video clips would assist in sustaining individuals' attention for the 5-min duration.

Confederate videos. The confederate videos were recorded using a Canon EOS 7D camera with a Canon EF-S 17-85mm 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 in during their laboratory

sessions. The confederate was shown eating either a small or large amount of LAY'S® Classic chips (5.7kcal/g). Chips were chosen in accordance with the focus on the intake of high-energy dense palatable foods (i.e., snacks) among most studies examining the social modeling of eating effect (Cruwys et al., 2015). LAY'S® Classic chips are also gluten-free, lactose-free, devoid of common food allergens, and kosher. This was strategically done to minimize the potential that personal food restrictions of the participants on these grounds would interfere with volume of consumption when presented with the same food item. 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 was instructed to eat as much of the chips in the bowl as possible during the film. In actuality, the model ate 35 g, or approximately 33 chips in this condition. The disparity in consumption between the conditions was noticeable in both the number of times the model reached for a chip (5 vs. 33) and the quantity of chips remaining in the bowl. To ensure that the emotional expressions of the confederate did not differ across the low- and high-intake conditions, an earbud was strategically 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 confederate was chosen to have a BMI within the normative range for the student demographic at the university. The videos displayed the confederate from the shoulders up to minimize comparison of body shape and size. She also donned a loose-fitting sweatshirt to further ensure slight ambiguity of body size. This was

deliberately done to enhance the likelihood of perceived similarity in weight status between the confederate and a broader range of participants. For both the low- and the high-intake conditions, two camera angles were simultaneously filmed. 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, which was recorded with the camera marginally behind and to the confederate's 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. The confederate grabbed each chip with her right hand. Given that all participants recruited were right-hand dominant, this second angle created an egocentric perspective. That is, the view of the right hand reaching for the chips would mimic the same perception as if they themselves were eating. Previous literature has suggested that an egocentric perspective enables greater action simulation and action understanding (Kelly et al., 2015).

Attention to video stimuli. A coarse behavioural measure of attention was utilized akin to that of Hobson and Bishop (2016) to confirm that participants adequately attended to the video stimuli for the duration of the clip. This was included in light of recommendations that attentional engagement is critical to monitor in studies of mu suppression to assist in disentangling the involvement of alpha and mu suppression in identifying mirror neuron activity. Though previous studies have relied upon counting tasks as an index of attentional engagement, such tasks may themselves influence alpha activity; thus, less cognitively-engaging tasks are

preferable (Hobson & Bishop, 2016). The baseline and confederate videos were edited in Movie Studio Platinum (version 12.0) to insert symbols that would appear periodically throughout the video clip in a designated area of the visual space. Following the viewing of each clip during the experiment, participants were asked three simple questions to indicate what symbol appeared (i.e., triangle, flower, square, star, arrow), how many times it was seen (i.e., 1-10), and where it appeared when it was seen on the screen (i.e., upper right, center, or left; bottom right, center, or left). In both of the eating-view videos for the low- and high-intake conditions, a symbol appeared on the screen five times in the bottom centre of the screen (☼ for high-intake; ☼ for low-intake). For the baseline (▲ in the upper right corner) and both the high- and low-intake movie-view videos, a symbol appeared on the screen three times (→ for high-intake; ■ for low-intake) in the screen's upper centre.

Procedure

Participants were invited to sign up for the study through the Sona Experiment Manager system (Appendix A). Interested participants signed up for one of the available laboratory appointments listed on Sona. All timeslots were scheduled at either 9:30 a.m., 11:30 a.m., 1:30 p.m., 3:30 p.m., or 5:30 p.m. Participants were informed that they must not drink alcohol 12 hr prior, nor were they to exercise, eat, or drink caffeine 2 hr prior to the laboratory session, as such factors can alter activity in the central nervous system and impact EEG recording. Upon arrival to the laboratory, participants read an information letter (Appendix B) to explicate the timeline of the tasks involved in the study (as depicted in Figure 3) and were provided with an informed consent form (Appendix C) to sign if they agreed to participate. Once their consent had been obtained and any questions had been answered, participants' head circumference was measured to fit them with an appropriately-sized 24 channel-EEG cap (large: 56-61 cm; medium: 51-56

cm; small: 47-51 cm). Participants were then connected to a 72-channel amplifier (Advanced Neuro Technology, Netherlands) for EEG and VEOG recording. ElectroGel was applied to the scalp to attain impedance levels below 10 k Ω prior to recording. Two self-adhesive bipolar electrodes were applied, with the positive and negative polarities placed above and below the right eye, respectively, to record and subsequently correct for ocular artifacts throughout the procedure. Participants were additionally instructed to attach one electrode below their left clavicle to serve as the ground electrode.

Once fully connected for electrophysiological recording, participants were told an elaborated version of the cover story alluded to in the Sona sign-up description to mask the true aim of the study (Appendix D). Participants were correctly informed that the brain area of interest was the MNS, with a brief description of the literature associating activity within the MNS and empathy. Participants were purposely led to believe, however, that the study sought to examine brain activation in response to mental judgments about the level of empathy others possess. As explained to participants, the study was also interested in exploring how MNS activity during such judgments may be associated with one's own level of empathy. This deliberate deception was necessary, as awareness of the research hypothesis may have otherwise encouraged participants to act in a way that was not true to their natural response tendency in their environment. Previous research examining the social modeling of eating effect has indicated that participants' eating behaviour may be altered if they feel their food intake is under scrutiny (Herman, Roth, & Polivy, 2003). It was thus critical to ensure that participants were unaware that their food consumption was of any interest to the experimenter.

Participants were instructed that they would be led through the study's procedure via instructions presented through a SurveyMonkey™ link. The entire procedure was viewed on the

72-inch 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 the tasks and EEG recording. A bell was provided to participants to ring if they had any need for clarification throughout the study or if they necessitated being unhooked from the recording devices to leave the laboratory. Participants rated their level of hunger as measured by the Grand Hunger scales in addition to their current level of thirst, fatigue, and discomfort on 7-point Likert scales (Appendix E) prior to the baseline electrophysiological recordings. These measures were requested under the guise that these variables associated with their current physical state could affect the level of brain activity recorded during the task. Following the completion of this questionnaire, participants were reminded to sit comfortably and remain as still as possible, and the 5-min baseline EEG and VEOG recording was then taken while participants watched the baseline video. This was followed by completion of the attention questions.

Participants were then instructed on screen that they would be watching a supposed video of a Lakehead University student, in reality the confederate, while she was watching an emotional film as part of a previous study conducted in the laboratory. They were informed that the film she was watching would not be visible to them, nor would there be any sound to accompany the video. Participants were told to rely solely on the visual cues of the emotive facial expressions displayed to rate the level of empathy they perceived. To minimize interference with the recordings, participants were once again instructed solely to observe the video and to engage in their assessment with minimal movement. They were informed that their empathy rating would be provided on a questionnaire after the video had concluded. Prior to entering the laboratory, participants had been randomized to the independent variable referred to

as model's consumption that comprised two levels: the low-intake condition, in which the confederate depicted the consumption of a small quantity of chips, and the high-intake condition in which the confederate depicted the consumption of a large quantity. At this time, they watched the eating-view video displaying the front-facing view of the confederate's eating behaviour and facial expressions without the television in view. Following this video, participants filled out the attention assessment questions, the contrived Empathy Rating questionnaire to purportedly assess the confederate's empathy level (Appendix F), and the TEQ (Appendix G) to provide a measure of their own self-reported empathy, in accordance with the cover story.

Upon completion of these questionnaires, the researcher emerged from the back room to inform participants that the so-called "reaction time task" (i.e., the visual-probe task) would take a few minutes to set up for the last portion of the laboratory session. Participants were told that while they were waiting, they would be able to view the film that the confederate had been watching to satisfy any curiosity they may have had to either confirm or disconfirm the accuracy of their empathy judgment. The researcher additionally stated that to make the wait more pleasant, they should feel free to help themselves to some chips. At this time, the same 56 g bowl of LAY'S® Classic chips the confederate ate was brought out from the back room and set down on the table in front of them. The researcher then retreated to the back room to supposedly continue setting up the next task. Participants were not aware that this portion of their time in the laboratory was of interest to the experimenter. This movie-view video displayed the side-angled view of the confederate in which her eating behaviour and the television screen was visible. This perspective enabled temporal contiguity between the observation and subsequent modeling of food consumption when food was available ad libitum. Recall that previous studies have found that socially-derived norms can influence participants' consumption. The rationale for providing

this second view of chip consumption was to enable greater confidence in the inference that mirror neuron activation was driving consumption rather than such norms. EEG and VEOG activity was additionally recorded during the observation of this video and chip consumption.

Once the video concluded, participants completed the attention assessment questions and the film task questionnaire (Appendix H). The researcher then emerged to remove the EEG cap, VEOG electrodes, and ground electrode from the participants prior to the visual-probe task for participants' comfort. The uneaten bowl of chips was taken away as the researcher returned to the back room. The bowl of chips was weighed at the end of the laboratory session once the participant had left. The visual-probe task was then presented using Inquisit™ (Version 4.0) software and presented on the television screen. The visual-probe task was modelled on those previously used to assess attentional bias towards food cues (e.g., Brignell et al., 2009; Hepworth et al., 2010; Hou et al., 2011), with the inclusion of two additional picture pair categories to explore attention towards eating-related and food-related cues. The pictures were shown in trials consisting of four different types of picture pairs: (1) a food-related cue versus a non-food cue, (2) an eating-related cue versus a non-food human cue, (3) a non-food cue versus a non-food human cue, and (4) a non-food cue versus a non-food cue.

The visual-probe task began with 12 practice trials, followed by two blocks of 160 test trials in which all of the three picture pairs were presented. Of the total 320 experimental trials, 240 were critical trials in which 80 presented a food-related and a non-food cue, 80 which presented an eating-related and a non-food human cue, and 80 which presented a non-food and non-food human cue. The remaining 80 trials were filler trials, which presented two non-food cues. Each type of cue had an equal probability of being shown on either the left or right side of the screen. All of the visual-probe trials consisted of the same picture pairings shown in the same

order for each participant. Each trial presented a fixation cross in the center of the screen for 500 ms, followed by the presentation of one of the four picture pairs simultaneously shown for either 500 or 2,000 ms. These durations were in accordance with previous methodology to ascertain attentional bias towards food among external eaters (i.e., Brignell et al., 2009; Hepworth et al., 2010) to discern between initial orienting and maintenance of attention, respectively. Each picture pair had an equal probability of being shown for either time duration. At the end of the set duration, the pictures disappeared to be replaced by the presentation of an 'X', the target probe, on either the left or the right of the screen. Participants were instructed to indicate the location of the target probe as quickly as possible by pressing either the "<" key to indicate its presentation to the left side of the screen, or the ">" key to indicate presentation on the right side. Subsequent trials began after the participant's response or, if they did not respond, a new trial began after 2,000 ms had elapsed. There were an equal number of trials in each of the four trial types as a function of stimuli duration, location of the cue, and probe location.

Upon completion of the visual-probe task, participants filled out the demographic questionnaire (Appendix I), the EHI – Short Form (Appendix J), and the DEBQ (Appendix K), as well as two questionnaires associated with a second study, the Reward-based Eating Drive scale (Van den berg, Franken, & Muris, 2010) and the Reward Responsiveness scale (Epel et al., 2014). Once participants clicked "Done" on the SurveyMonkey form, the researcher emerged from the back room once again to measure participants' weight using a Brecknell (LPS-400) digital scale and their height. Participants were invited to ask any questions that they may have had about the study, and were then thanked and dismissed. The study's true purpose was not revealed to participants at this time to ensure that participants were not able to discuss the study's deception with others in advance of their participation, thereby invalidating the results.

This was an important consideration given the small size of the university campus. Once the study concluded, a debriefing email (Appendix L) was sent to participants to indicate the true purpose of the study. Participants were encouraged to contact the research team if they had any concerns about their data upon the revelation of the study's purpose and were informed they retained the right to withdraw their data.

Results

Data Preparation

All data from the psychometric measures and demographic questionnaires were entered into SPSS v.23 and examined for missing values. On the DEBQ – External Eating subscale, two individuals were missing responses across two items; three responses were missing across two items on the DEBQ – Restrained Eating subscale; and seven responses were missing across three items on the DEBQ – Emotional Eating subscale (4 individuals missing item #16 “Do you have a desire to eat when you are cross?”). Missing values were replaced with prorated scores based on individuals' mean score on the scale or subscales in question. Notably, Hayes' (2013) PROCESS macro does not provide an internal procedure for dealing with missing values outside of listwise deletion. As age was included as a covariate in the models, missing values for five participants were replaced with the mean age of the sample to retain their data in analyses. In addition, despite a number of participants consuming 0 g of chips, only two participants did not indicate how much they liked the chips offered. For these individuals, the missing value for chip liking was replaced with the mean liking reported in the sample ($M = 5.04$).

Data Analysis Strategy

Hypotheses one and three. Analyses of hypotheses one and three to assess the hypothesized moderated mediation relationships were conducted with the use of model seven of

Hayes' (2013) PROCESS macro. PROCESS utilizes an ordinary least squares (OLS) analytic framework to estimate the unstandardized regression coefficients (b), standard errors (SE), direct and indirect effects, and an index of moderated mediation. A moderated mediation refers to a mediated effect of X on Y that varies in strength in accordance with the value of a moderator (Hayes, 2015) or, put differently, an instance in which the size of the indirect effect depends upon another variable (Hayes & Rockwood, 2016). Moderation "...speaks to the circumstances, contexts, or types of people for whom an effect exists and for whom it does not" (Hayes & Rockwood, 2016, p.1).

The conceptual models illustrating hypotheses one and three (depicted in Figure 1 and Figure 2) are known as a first-stage moderated mediation (Hayes, 2015). A first-stage moderated mediation tests the effect of X (model consumption) on Y (participant consumption) through M (mu rhythm desynchronization), in which the effect of X on M is hypothesized to be moderated by W (i.e., external eating or attentional bias to food). Thus, model seven estimates the indirect effect of X on Y through M at various levels of W , which is referred to as the conditional indirect effect. The method, as articulated by Hayes (2015), quantifies the linear relationship between the indirect effect and the proposed moderator of that effect to generate an "index of moderated mediation" through the product of regression coefficients. Figure 4 depicts the statistical model of a moderated mediation analysis. The indirect effect is a linear function of the moderator (Hayes & Rockwood, 2016). Subsequently, it must be discerned that the index of moderated mediation is significantly different from zero to enable the claim that there is evidence of a significant moderated mediation (Hayes & Rockwood, 2016).

PROCESS enables the generation of a bootstrap confidence interval by randomly resampling n cases from the data with replacement, where n represents the sample size employed

in the study (Hayes & Rockwood, 2016). The model is repeatedly re-estimated k times, with 10,000 iterations utilized in the current study. A bias-corrected bootstrap confidence interval is then constructed which defines the 2.5th and 97.5th percentiles of this distribution adjusted as a function of the proportion of k values less than the point estimate calculated in the original data (Hayes, 2013). These values define the upper and lower bounds of a 95% confidence interval (CI). When this interval does not straddle zero, but rather is entirely above or below, this is interpreted as evidence to support a statistically significant index of moderation, enabling a claim of moderated mediation (Hayes & Rockwood, 2016). Though alternative methods are available for inferences about the product of regression coefficients and estimates of effects, the use of bootstrapping circumvents the assumption of normality of the sampling distribution, provides relatively higher statistical power, and reduces the likelihood of Type I error (Preacher & Hayes, 2008).

To probe any significant interactions in the moderated mediation models, the pick-a-point approach was implemented. The pick-a-point approach involves estimating the conditional indirect effect of X on Y at specified values of W , typically defined as the mean and 1 SD above or below the mean. PROCESS can alternatively be used to estimate the conditional indirect effect at various percentiles in the sample's distribution of the moderator (W). While the Johnson-Neyman (JN) technique can be used to probe simple moderations, and is notably less arbitrary in the selection of values of the moderator at which to estimate the effect, it cannot be utilized in the case of a moderated mediation. The JN technique requires normality of the sampling distribution, which is known to be violated for the indirect effect (Hayes & Rockwood, 2016).

The above analytical strategy differs from the traditional approach expounded by Baron

and Kenny (1986) for analyzing mediation, which involves demonstration of statistically significant regression coefficients for each of the causal pathways from X to M (path a), from M to Y (path b), and from X to Y (path c) in sequence. Under this approach, if the three pathways were significant, and it could be shown that the direct effect of X on Y when M was held constant was closer to zero than X 's effect without controlling for M , then it was considered appropriate to deem M as a mediator (Hayes & Rockwood, 2016). Contemporary thinking, however, focuses on conducting an inferential test of the indirect effect, which is estimated as the product of regression coefficients for paths a and b , rather than tests of significance for each individual pathway. Hayes and Rockwood (2016) explicate that this shift is founded upon three central principles of statistical inference.

Firstly, empirical claims should be based on quantifying the effect most directly relevant to the hypothesis (Hayes & Rockwood, 2016). The piecemeal approach of Baron & Kenny (1986), however, does not formally quantify and perform an inferential test of the indirect effect. Empirical claims also ought to be based upon as few inferential tests as necessary to retain statistical power and avoid excessive risk of Type I and Type II error (Hayes & Rockwood, 2016). Lastly, directly quantifying the indirect effect enables the generation of a confidence interval, thereby providing more information than a simple claim of mediation. This more modern approach to empirically testing causal mediational processes is thus primarily interested in estimating and interpreting the direct and indirect effects, along with their corresponding inferential tests (Hayes, 2013). Though the focus is largely directed towards the indirect effect in this method, however, it should be noted that pathways a and b cannot be ignored entirely. The pattern of signs necessarily entail different substantive interpretations (Hayes & Rockwood, 2016). The signs must correspond to the theorized associations to represent support for one's

inferences.

For all analyses examining mu rhythm suppression and chip consumption, four covariates were included to ensure that variance attributable to these extraneous variables was eliminated: chip liking, level of discomfort, positively-valenced emotional state, and age. In preliminary analyses, these variables were found to be significantly associated with variation in either mu rhythm desynchronization (M) or chip consumption (Y). One of the central assumptions for a correctly specified mediation model is that the model must not have any misspecification due to unmeasured variables that cause variables in the mediation analysis (MacKinnon, Fairchild, & Fritz, 2007). It is crucial in forming a causal argument that one is able to rule out epiphenomenality or spurious associations as alternative explanations (Hayes, 2013). 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 that are spuriously associated with key variables in the causal system therefore must be controlled for (Hayes, 2013). Moreover, in the case of each covariate included in the model, the research literature reasonably substantiated their inclusion for their capacity to influence particular variables.

One might expect that individuals' taste preference for the chips offered would potentially influence the volume of chips consumed in the laboratory. Following ad libitum access, participants rated how much they liked the chips on a 7-point Likert scale from 1 (*Strongly disliked*) to 7 (*Strongly liked*). Chip liking was shown to be strongly correlated with the quantity of chips consumed, $r = .58$, $p < .001$, and was accordingly controlled for in analyses. Individuals' discomfort rating following EEG cap fitting and prior to the start of the experimental tasks was

also significantly associated with how many chips they consumed in the laboratory, $r = .23, p < .05$. Previous literature has indicated that approximately 40-50% of the population tend towards eating in response to stress (Sproesser, Schupp, & Renner, 2014). Furthermore, a recent mediational analysis revealed that higher levels of stress tend to be associated with higher levels of emotional eating through greater eating dysregulation (Tan & Chow, 2014). Though often neglected as a trigger for consumption, positive emotions may also elicit greater caloric intake (Evers, Marieke, de Ridder, & de Witt, 2013). Ergo, evidence of hyperphagia, or increased eating behaviour, in response to negative (e.g., Tan & Chow, 2014) or positive (e.g., Evers et al., 2013) affect may only capture a transitory fluctuation in eating behaviour. Such changes may provide limited insight with regards to the dynamics of one's eating more characteristically (Sproesser et al., 2014). In the mirror neuron literature, it has additionally been shown that both positively- and negatively-valenced states can augment MNS activity (Hill et al., 2013). The current study found that endorsement of an overall positive emotional state was significantly associated with enhanced MNS activity at central sites C3, $r = -.32, p < .01$, and C4, $r = -.31, p < .01$. Thus, both discomfort and a composite average of positively-valenced affective state variables endorsed were additionally included as covariates.

With respect to the inclusion of age as a covariate, evidence has suggested that mu suppression in response to the observation of actions increases with age from childhood to adolescence (Oberman et al., 2013). In the current study, there was evidence of a significant association between age and mu suppression at both C3, $r = .20, p < .05$, and C4, $r = .19, p < .05$. Previous research examining changes in EEG power spectra has indicated that from childhood to early adolescence, the amplitudes of lower frequencies decrease while those of higher frequencies increase (e.g., Cragg et al., 2011; Somsen et al., 1997). These changes appear to

correspond to general maturational processes within the brain, though the precise processes they reflect are unclear. It has been posited that such changes may reflect developmental maturation in white matter, such as an increase in myelination (Cragg et al., 2011). It has not been delineated whether and at what point in development this enhancement may slow, stall, or begin to decrease. However, it has been shown that maturational changes in brain regions continue throughout adolescence and into adulthood with respect to cortical thickness and volume (Tamnes et al., 2009). As ages in the present study ranged from 17-36, age was included as a covariate to eliminate the variance in mu rhythm activity across participants that may have arisen due to differential maturational stages in the brain.

All analyses that included scores on the DEBQ - External Eating scale also included participants' scores on the Emotional and Restrained Eating scales of the DEBQ as covariates, given high correlations amongst the subscales. Though the a priori analysis plan reasoned to include hunger as a covariate to control for potential state-based changes in the motivational relevance of food, post hoc analyses indicated that hunger was unrelated to participants' consumption and it was thus not included in order to retain greater statistical power. Concern was given to ensuring that the inclusion of covariates did not create the risk of overfitting the model beyond its capacity with respect to using up too many degrees of freedom in relation to the number of observations. The typical rule of thumb based on simulation studies has suggested that for linear models, there ought to be a minimum of 10 to 15 observations per predictor variable to allow for adequate estimates of effects (Babyak, 2004). At most, the model included two distinct predictors (i.e., model consumption, external eating) while controlling for six covariates. Thus, there were at least 12 or more observations per variable.

Hypothesis two. To analyze hypothesis two, attentional bias towards food and attentional

bias towards eating scores were each examined separately in paired-samples *t*-tests to discern whether RTs differed significantly across both time durations utilized (i.e., 500 ms and 2,000 ms) within each stimuli category. As no significant differences emerged between RTs for food stimuli, $t(92) = 0.185, p = 0.854$, or RTs for eating stimuli, $t(92) = -0.136, p = 0.892$ across the two durations, the RTs were collapsed into two composite scores for attentional bias towards food and attentional bias towards eating cues, respectively (see Table 1 for *Ms* and *SDs* of visual probe data). Hypothesis two predicted that those who were higher on external eating would exhibit a greater attentional bias towards food and was analyzed via separate regression analyses. External eating was conceptualized as the independent variable and both attentional bias towards food and attentional bias towards eating scores were separately tested as the dependent variables. As external eating was theorized to represent a proxy for attentional bias towards food, this analysis sought to determine which task was capable of eliciting evidence of an attentional bias among external eaters, as has been previously demonstrated. The bias score correlated with external eating was subsequently utilized as the moderator variable in hypothesis three.

BMI was included as a covariate in the analysis of hypothesis two for one of the regression models tested. Prior evidence has demonstrated that BMI is associated with greater attentional bias to food stimuli (Yokum, Ng, & Stice, 2011). The current study similarly found a significant association between BMI and attentional bias towards food-related cues, $r = .22, p < .05$. Thus, BMI was included as a covariate in the regression model predicting attentional bias towards food-related cues to control for its effect on the criterion variable. There was no significant association between BMI and attentional bias towards eating-related cues, $r = .01, p = .92$. Therefore, BMI was not controlled for in the test of the regression model to predict attentional bias towards eating-related cues. As the DEBQ – External Eating subscale was used to quantify

external eating, both the Restrained and Emotional Eating subscales were additionally included as covariates given correlations among subscales.

Variable coding. In all analyses, the independent variable (X) model consumption was dichotomously coded as 1 (high intake condition) and 0 (low intake condition), representing that a higher value on model consumption was associated with the observation of greater food consumption. Participant consumption (Y) was quantified as the amount of chips participants ate in grams, such that higher levels of participant consumption reflected a greater intake. Recall that the log ratio calculation utilized for mu rhythm desynchronization translates into negative values for suppression, zero if there is no evidence of alteration in mu rhythm activity, and positive values for hypersynchronization or enhancement (Perry & Bentin, 2009). Thus, more negative values of mu rhythm desynchronization (M) were indicative of increased mirror neuron activity over baseline. For hypothesis one, external eating (W) was quantified as individuals' score on the External Eating scale of the DEBQ, with higher scores reflective of a greater tendency towards eating in response to external stimuli. For hypothesis three, attentional bias (W) was calculated such that positive values indicated greater attention towards food- or eating-related stimuli.

Parametric assumptions. Prior to conducting analyses of the experimental hypotheses, the data were assessed for violations of parametric assumptions to ensure that the findings would be unbiased and therefore exhibit a greater likelihood of generalizability (Field, 2013). Moderated mediation requires the fulfillment of the assumptions of homoscedasticity, normality, linearity, and independence of errors. The assumption of homoscedasticity indicates that the variance of residuals is constant across values of the predictor and implies that the residual variance is independent of the predictor variables (Hayes & Cai, 2007). Violations of this assumption can effectively inflate Type I error rates, reduce statistical power, and bias estimates

of the confidence intervals (Hayes & Cai, 2007). As recommended by Field (2013), the assumptions of homoscedasticity and linearity were assessed visually via a scatterplot of the residuals against the predicted values. Given the complexity of the moderated mediation model, it was parsed into separate models to explore the assumption from X to M , M to Y , and X to Y . As there was no systematic relationship between the models' errors and models' predictions (Field, 2013), it was concluded that there were no violations of the assumption of homoscedasticity or linearity for any of the proposed analyses. Based on recommendations by Hayes & Cai (2007), heteroscedasticity-consistent standard error estimates were also utilized in PROCESS to conduct the moderated mediations to provide greater assurance as to the validity and power of tests.

With respect to normality, the data were first examined for outliers across the variables of interest, defined as cases with a z score of ± 3.29 (Field, 2013). All outliers on raw EEG power were removed from analyses, as it was deemed to be reflective of technical recording error. One outlier was identified on BMI and replaced with the next highest nonoutlier value (Field, 2013). All continuous variables were additionally tested for normality using z_{skewness} , calculated as $\text{skewness}/SE$. Any z_{skewness} scores equal to or greater than ± 1.96 by convention were considered significantly skewed at $p < .05$ (Field, 2013). Given the nature of the data, there were no theoretical concerns about the independence of errors and thus this assumption was not formally tested.

Descriptive Statistics

Descriptive information pertaining to relevant psychometric variables and sample characteristics are presented in Table 2. Among these variables are those included as moderators or criterion variables in analyses of the study's hypotheses. As exemplified by the reported z_{skewness} , none of the psychometric variables exhibited significant skew (± 1.96). Overall, the

mean external eating score as measured by the DEBQ – External Eating subscale was 3.24 ($SD = 0.6$). Participants consumed an average of 19.76 g ($SD = 17.01$) out of 56 g chips served (i.e., 35%). The quantity of chips participants consumed was positively skewed, $z_{\text{skewness}} = 2.83$, and was therefore subjected to a natural log transformation which did not correct the skew. Chip consumption was subsequently transformed using a square root transformation which effectively corrected the skew. The square root transformed value of chip consumption was thus utilized in subsequent analyses. The distribution of BMI was also positively skewed in the sample, $z_{\text{skewness}} = 4.39$. As both the log and square root transformations did not reduce the skew, a reciprocal transformation was attempted. Though this attenuated the skew, the reciprocal transformation reverses the scores such that values initially large in the data become small and vice versa (Field, 2013). Analyses revealed that the untransformed BMI did not appreciably change the results obtained. Thus, for ease of interpretation, untransformed BMIs are reported for all analyses.

Table 3 displays the means, standard deviations, and skewness of the raw EEG power values during the baseline and eating-view recording blocks, as well as the mu/alpha suppression ratios at each electrode site. Raw EEG power in the mu and alpha frequency bands was positively-skewed as is typical of untransformed power values in the 8-12 Hz frequency band (Smith et al., 2017). The reported suppression ratios were calculated by performing a logarithmic transformation on the ratio of the raw EEG power values. All subsequent analyses used these transformed ratio values. Skew remained for the distributions of C4 and F4 data. However, further transformation of these data was deemed undesirable for the following reasons. The log ratio is the conventional measurement utilized for mu rhythm desynchronization. Field (2013) recommends that using robust procedures (i.e. bootstrapping) is often preferable over transforming the data where possible when there are implications for interpreting the data. All

models including suppression ratios used bootstrapped moderated mediation analyses.

Group Comparisons

To explore whether there were any unintended differences in the sociodemographic characteristics of the low- and high-intake groups, independent-groups, two-tailed *t*-tests were examined for all psychometric and demographic variables. No significant differences between the two conditions emerged on characteristics such as age, marital status, ethnicity, DEBQ subscale scores, DEBQ total scores, or any of the four indices of hunger as measured by the Grand Hunger scale. Despite random assignment to conditions, those in the low-intake condition demonstrated a significantly lower BMI, $M = 23.50$, $SD = 3.97$, $t(91) = -2.51$, $p < .05$, than those in the high-intake condition, $M = 25.67$, $SD = 4.38$. However, BMI was not significantly correlated with chip consumption $r = -.01$, $p = .94$. Thus, BMI was not controlled as a covariate in the analyses of the social modeling of eating effect (i.e. hypothesis one or hypothesis three).

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, $r = .25$, $p < .05$. 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, $M = 4.41$, $SD = 2.19$, $F(1, 91) = 6.12$, $p < .05$. As per Cohen's classification (Field, 2013), the obtained effect size $d = -0.52$ is considered to be medium. This effect size is comparable to that reported by Vartanian et al. (2015), $r = .39$ (equivalent to $d = 0.85$) in their meta-analysis, though not quite as large.

Hypothesis One

The first hypothesis predicted external eating (W) would moderate the strength of the

relationship between model consumption (X) and mu rhythm desynchronization (M), thereby influencing the level of participant consumption (Y). As central electrode sites C3 and C4 are of particular interest to activity in the MNS, the model was first estimated at these sites. Data were subjected to Hayes' (2013) PROCESS macro model 7 to test for a first-stage moderated mediation. There was no evidence of a significant index of moderated mediation at either C3, $a_3b_1 = .097$, $SE = .147$, 95% CI [-.071, .582] or C4, $a_3b_1 = .039$, $SE = .120$, 95% CI [-.131, .389]. The model was additionally estimated at F3, F4, O1, and O2 to determine whether evidence of suppression was demonstrated at any of these other comparative cortical sites. None of these analyses proved significant. Table 4 displays the regression coefficients obtained within each of the six moderated mediation analyses with external eating as the moderator.

Interestingly, the regression coefficient for the effect of suppression at electrode site F3 on chip consumption was at trend level of probability, $b = -1.066$, $p = .07$, suggesting that there was perhaps an association between alpha suppression and participant consumption as moderated by external eating at frontal sites. However, none of the indices of moderated mediation were significant, as evidenced by the inclusion of zero within each 95% CI for each of the six electrode sites (see Table 5). As revealed by the pick-a-point approach in Table 5, the indirect effect was not significant at any level of external eating ($\pm 1 SD$ or M). Thus, results do not support the hypothesis that mu rhythm would mediate the observed effect of model consumption on participant consumption, which remained regardless of one's level of external eating.

Hypothesis Two

The second hypothesis postulated that those higher on external eating (X) would exhibit greater attentional bias towards food (Y). Two simple linear regression analyses were run, one each for attentional bias towards food-related cues and eating-related cues. The following

covariates were entered: DEBQ Restrained and Emotional eating subscales in both regression models, as well as BMI in the model examining attentional bias towards food-related cues. External eating significantly predicted attentional bias towards food-related cues, $b = 8.40$, $t(88) = 1.97$, $p = .052$, though just at the cusp of significance. Conversely, level of external eating did not significantly predict attentional bias towards eating-related cues, $b = -2.38$, $t(88) = -0.61$, $p = .54$. A paired-samples t -test in the overall sample revealed a trend towards greater attentional bias towards food-related cues, $M = 5.89$, $SD = 20.87$, than towards eating-related cues $M = 0.72$, $SD = 18.44$, $t(92) = 1.80$, $p = .08$, suggesting that food cues may hold greater salience or attention-grabbing power than eating cues. The magnitude of this attentional bias towards food notably approximates that found in previous studies (i.e., Hou et al., 2011). As external eating was predictive of attentional bias towards food-related cues, this bias score was utilized as the moderating variable in the moderated mediation model of hypothesis three (see Figure 2).

Hypothesis Three

The third hypothesis predicted that the indirect effect of model consumption (X) on participant consumption (Y) via mu rhythm desynchronization (M) would be conditional on a first-stage moderation of attentional bias towards food (W). That is, it was hypothesized that the indirect effect would be conditional upon individuals' tendency to allocate their attention towards food, as measured by the visual-probe task. This model was tested using model 7 of Hayes' (2013) PROCESS macro. Once again, the model was analyzed first at central electrode sites C3 and C4. There was no evidence of a significant index of moderated mediation at either C3, $a_3b_1 = .003$, $SE = .003$, 95% CI [-.001, .014], or C4, $a_3b_1 = .003$, $SE = .004$, 95% CI [-.001, .015]. The results do not support the hypothesis that attentional bias towards food would moderate an indirect association between model and participant consumption via mu rhythm.

The model was additionally estimated at F3, F4, O1, and O2. None of these analyses yielded statistically significant effects. Table 6 displays the regression coefficients for each of six moderated mediation analyses with attentional bias towards food as the moderator. Interestingly, once again the pathway between alpha suppression at F3 and chip consumption was at trend level of probability $b = -1.017, p = .08$. However, as evidenced by the indices of moderation mediation shown in Table 7, none of the models yielded statistically significant effects. All of the confidence intervals around the six indices of moderated mediation straddled zero.

Summary. The results indicate robust evidence of the social modeling of eating effect. However, contrary to hypothesis one and three, mu rhythm suppression at central sites did not mediate this relationship, regardless of external eating or attentional bias towards food. Interestingly, an association was observed at trend level between alpha suppression at site F3 (M) and chip consumption (Y) in analyses of both hypotheses one and three. This coefficient represented the relationship between alpha suppression at F3 and chip consumption when controlling for model consumption (X) and external eating (W) or attentional bias towards food (W), respectively. Notably, this association was only apparent at F3 and not at F4. This seemed to suggest that there was a differential pattern of activity present across the frontal sites which may have influenced individuals' intake. As the strength of the association appeared to differ across these frontal sites, frontal asymmetry was postulated to be a potential moderator. Thus, it was explored whether differential activation across the frontal sites altered the strength of mu/alpha suppression's capacity to mediate the established social modeling of eating effect.

Exploratory Analyses

Frontal asymmetry data preparation. Recall from the introduction, frontal asymmetry reflects the differential lateralization of cortical power recorded at the frontal F3 and F4 sites. To

calculate frontal asymmetry scores, alpha power at the left (F3) and right (F4) electrode sites during the baseline and eating-view recording blocks were first natural log transformed, in keeping with conventions for this type of data (Smith et al., 2017). A difference score was subsequently calculated to summarize the relative activity across the left and right frontal cortex (i.e. $F4ln - F3ln$) in accordance with convention for each of the baseline and eating-view blocks, respectively. 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 (Harmon-Jones & Gable, 2009). Prior to analyses, frontal asymmetry scores were examined for outliers defined as cases with a z score of ± 3.29 (Field, 2013). One outlier was identified on baseline frontal asymmetry scores and was replaced with the next highest outlier, as per recommendations for winsorizing outliers (Field, 2013). No outliers were established for the eating-view block. Frontal asymmetry scores during both the baseline recording, $M = 0.03$, $SD = 0.12$, $z_{skewness} = 1.0$, and during the eating-view recording, $M = 0.03$, $SD = 0.11$, $z_{skewness} = 1.24$ did not exhibit significant skew.

Exploratory analytic strategy. Frontal asymmetry calculated during the eating-view recording block was opted to be examined for its capacity to moderate the first stage of the mediated social modeling of eating effect via mu rhythm desynchronization. It was reasoned that utilizing the eating-view frontal asymmetry would be more appropriate than baseline frontal asymmetry to enable valid causal inference. It has been indicated that dynamic shifts in relative asymmetry occur within subjects across time (Smith et al., 2017). As one's motivational state may alter frontal asymmetry, it would be most pertinent to assess how the current state of asymmetry was able to influence activation occurring simultaneously in other regions of the

brain (i.e., the MNS). Analyses revealed that BMI was significantly associated with frontal asymmetry, $r = .22, p < .05$, indicative of greater left frontal asymmetry as BMI increased. Ergo, the moderation of the indirect social modeling of eating effect by frontal asymmetry was posited to differ in strength across varying levels of BMI. Figure 5 depicts the exploratory first- and second-stage dual moderated mediation with BMI placed in the second-stage of the model.

To examine the model, Hayes (2013) PROCESS model 21 was utilized to examine whether there was evidence of a dual moderated mediation. This model hypothesized that the first stage of the indirect effect of model consumption (X) on participant consumption (Y) through mu rhythm 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 second stage of the model (i.e. exerting its influence between M and Y). The index of dual moderated mediation (a_3b_3) for this model “...quantifies the rate of change in the moderation by W of the indirect effect of X as V changes, as well as the rate of change in the moderation by V of the indirect effect of X as W changes” (Hayes, in press). Figure 6 displays the statistical model underlying PROCESS model 21.

The dual moderated mediation can subsequently be probed by estimating and conducting an inferential test of the index of conditional moderated mediation for various values of both moderators to integrate into a substantive interpretation (Hayes, in press). Using the pick-a-point approach, PROCESS can generate an estimate of the conditional indirect effect at values of the moderators corresponding to the 10th, 25th, 50th, 75th, and 90th percentiles in the distribution of the moderators (Hayes, 2013). This option was used rather than the mean and $\pm 1 SD$ above and below the mean, as it was expected that the interpretation would necessarily require a more

nuanced view of the data. Akin to the prior analyses of the social modeling of eating effect in hypotheses one and three, chip liking, level of discomfort, positively-valenced emotional state, and age were included as covariates in model 21. This model also controlled for participants' baseline frontal asymmetry.

Exploratory results. The overall dual moderated mediation model was significant at electrode sites C3 and F3. Table 8 displays the unstandardized regression coefficients, standard errors, and 95% CIs generated from the model tested at electrode sites C3, C4, F3, F4, O1, and O2. Examining the model tested with C3 mu suppression as the mediator, a statistically significant interaction between model consumption and frontal asymmetry predicted C3 mu suppression, $b = -2.382$, $SE = 0.728$, 95% CI [-3.831, -0.934], and mu suppression significantly predicted chip consumption, $b = -8.146$, $SE = 3.749$, 95% CI [-15.603, -0.689]. Although this model did not reveal a statistically significant interaction between mu suppression (M) and BMI (V), $b = 0.301$, $SE = 0.155$, 95% CI [-0.007, 0.610], $p = .06$, there was a trend towards significance. With respect to frontal site F3, there was evidence of a statistically significant interaction between model consumption and frontal asymmetry in the prediction of F3 alpha suppression, $b = -2.142$, $SE = 0.703$, 95% CI [-3.540, -0.743]. For site F3, alpha suppression indicated a trend towards significance in predicting chip consumption, $b = -8.665$, $SE = 4.454$, 95% CI [-17.302, 0.686], $p = .07$. There was no evidence of a trend towards significance or significant interaction between alpha suppression and BMI at F3. However, the lack of a significant interaction does not imply that the indirect effect is not moderated (Hayes, 2015). It is more relevant to compute an index estimating the overall model and to quantify the relationship between the proposed moderator and the size of the indirect effect. Table 9 displays these indices of dual moderated mediation for each of the six models tested across electrode sites.

As noted, the index of dual moderated mediation was statistically significant for the model at central electrode site C3, $a_3b_3 = -0.718$, $SE = 0.365$, 95% CI [-1.632, -0.161]. As the CI does not include zero, it is appropriate to conclude that frontal asymmetry and BMI moderated the indirect effect of model consumption on participant consumption via mu rhythm desynchronization. The index of dual moderated mediation can be interpreted to mean that the moderation of the indirect effect of model consumption on participant consumption via mu rhythm desynchronization at electrode site C3 by frontal asymmetry decreased by 0.718 units as BMI increased. The index of dual moderated mediation (a_3b_3) reflects the multiplication of $X \times W$ and $M \times V$, as can be inferred from the statistical diagram in Figure 7. The model can be separated for interpretive purposes into two separate moderation analyses. Examining the regression coefficient for $X \times W$ at site C3 in Table 8, $a_3 = -2.382$, this means that for two cases that differed by a unit on X (i.e. low-intake vs. high-intake condition), mu rhythm desynchronization changed by -2.382 units as frontal asymmetry (M) increased by one unit. Thus, observation of greater eating behaviour by the model was associated with more mu suppression among those with higher levels of frontal asymmetry, which corresponds to greater left frontal cortical activity. The first stage of this model was followed up with a simple moderation analysis. At frontal asymmetry values corresponding to -1 SD , the association between model consumption and mu suppression at C3 was positive, $b = 0.230$, $SE = 0.095$, $p < .05$, 95% CI [0.040, 0.419] indicative of less activity in the MNS, whereas for values +1 SD , it was negative, $b = -0.282$, $SE, 0.123$, $p < .05$, 95% CI [-0.526, -0.038], suggestive of greater MNS activity.

Overall, the regression coefficient between mu rhythm desynchronization (M) and participant consumption (Y) was significant and negative, suggesting that greater mu suppression

was associated with a larger quantity of chips consumed. The coefficient for $M \times V$, $b_3 = 0.301$, represents the fact that for two cases that differed by a unit on mu desynchronization (M), chip consumption changed by 0.301 units as BMI (V) increased by one unit. In this case, the positive coefficient reflects the fact that as BMI increased, greater mu suppression (i.e., more negative values on M) was associated with *fewer* chips consumed by the participant (Y). This suggests that the influence of the social modeling of eating effect as mediated by mu rhythm desynchronization was attenuated amongst those with a higher BMI. Thus, the overall index of dual moderated mediation was positive when multiplying $X \times W$ and $M \times V$ to generate an estimate of a_3b_3 . Figure 8 depicts the regression coefficients for each pathway in the model estimated at site C3. The dual moderated mediation model was also significant at frontal site F3, $a_3b_3 = -.636$, $SE = .411$, 95% CI [-1.642, -.023], with a similar pattern of interpretation.

Tables 10 through 14 present the conditional indirect effect of X on Y at values of both moderators using the pick-a-point quantile method. Each of the five tables represent the 10th, 25th, 50th, 75th, and 90th percentiles of frontal asymmetry, respectively, and list the conditional indirect effect at each quantile of BMI for each of the electrode sites. 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 (Hayes, 2013). As seen across these five tables, the conditional indirect effect was significant for values of frontal asymmetry corresponding to the 10th, 75th, and 90th percentiles within the current sample. The 75th and 90th percentiles correspond to positive 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/alpha suppression at central and frontal electrode sites. The 10th

percentile of frontal asymmetry corresponded to a negative value of -0.103, suggesting right frontal asymmetry. For such individuals, the indirect effect was negative, suggesting that greater model consumption was associated with less chip consumption as mediated by mu/alpha suppression. With respect to BMI, the conditional indirect effects were only significant for those with a BMI at the 50th percentile or lower, corresponding to 20.08 to 23.72 in the current sample.

Summary of exploratory analyses. The exploratory dual moderated mediation model revealed that frontal asymmetry and BMI moderated the indirect effect of model consumption on participants' chip consumption through mu rhythm desynchronization. The effect was significant for those at the 10th, 75th, and 90th percentiles of frontal asymmetry. Left frontal asymmetry was associated with greater mu rhythm desynchronization (i.e. greater MNS activity) and a positive association between model and participant chip consumption. Right frontal asymmetry was associated with less mu rhythm desynchronization (i.e. less MNS activity) and a negative association between model and participant chip consumption. Across all levels of frontal asymmetry, the effect was only significant for those with a BMI at the 50th percentile or lower.

Discussion

The social modeling of eating effect refers to the well-established phenomenon whereby individuals tend to match their food intake with that of their companion (Cruwys et al., 2015; Vartanian et al., 2015). In a recent review of this effect, it was highlighted that it is critical for future research to explore the mechanisms underlying modeling effects on eating (Cruwys et al., 2015). The current study sought to explore the MNS as a potential mechanism through which this effect may be elicited. In light of substantial efforts to delineate the effect's boundary conditions, it was hypothesized that external eating would moderate an indirect effect of model consumption on participant consumption via mu rhythm desynchronization (i.e., MNS activity).

This hypothesis was founded on evidence that MNS activity is enhanced through attentional focus on stimuli (Muthukumaraswamy & Singh, 2008), and corresponding findings that external eating appears to be associated with greater attentional bias towards food (e.g. Brignell et al., 2009; Hepworth et al., 2010; Hou et al., 2011). It was thus additionally posited that there would be an association evident between external eating and attentional bias towards food, and that attentional bias towards food would similarly moderate the hypothesized indirect causal process. The discussion that follows will begin with a brief overview of the results of these hypotheses followed by a consideration of the ecological validity of the proposed moderators. Subsequently, the results of the exploratory analyses will be examined and interpreted in greater depth. The discussion will conclude with a consideration of the contributions to the social modeling of eating literature, the strengths and limitations of this study, and suggested directions for future research and potential clinical implications.

External Eating and Attentional Bias as Moderators

The results from the present analyses did not support either of the hypothesized moderated mediation models. Neither external eating, nor attentional bias towards food, significantly moderated an indirect social modeling of eating effect through mu rhythm desynchronization. Though the regression coefficients in the models for hypotheses one and three did not reach significance, it is still informative to examine the direction of the signs of each pathway to explore the theoretical predictions about how the causal mechanism may operate (Hayes & Rockwood, 2016). Examining the coefficients linking mu rhythm desynchronization (M) with participants' consumption (Y), there was a nonsignificant negative association between mu rhythm desynchronization and chip consumption when controlling for model consumption (X) and external eating (W) or attentional bias (W), respectively, across both

hypotheses one and three. The direction of this association was in line with the assertion that greater suppression would be associated with greater chip consumption. For both external eating and attentional bias towards food, the coefficient representing the interaction between each respective moderator with model consumption ($X \times W$) and mu rhythm desynchronization (M) was negative. This observation was consistent with the notion that higher levels of both external eating and attentional bias towards food would be associated with greater desynchronization, and may provide some indication that MNS activity may be amenable to be altered by individual-level characteristics. Overall, data from hypotheses one and three revealed a significant direct effect between model and participant consumption when controlling for mu rhythm desynchronization. Greater model consumption was associated with greater chip consumption among participants. At the most fundamental level, there was thus evidence to replicate the social modeling of eating effect, regardless of one's level of external eating or attentional bias towards food.

External Eating Predicting Attentional Bias

Support was additionally demonstrated for hypothesis two. External eating significantly predicted attentional bias towards food cues ($p = .05$) when controlling for restrained and emotional eating, as well as BMI. Despite including both “dynamic” eating-related cues (i.e., a human interacting with food) and food-related cues (i.e., food alone), an association was only evidenced between external eating and attentional bias towards food-related cues. Previous literature has suggested that attentional bias as measured by the visual-probe task may depend largely on image complexity. Miller and Fillmore (2010) demonstrated that individuals only displayed an attentional bias towards simple, but not complex, images of appetitive stimuli (i.e.

alcohol). The current study lends credence to such findings, suggesting that attentional bias assessed via the visual-probe task may only be apparent for simple stimuli.

Ecological Validity of Attentional Bias and External Eating

In light of the aforementioned findings, it may be argued that the visual-probe paradigm may lack ecological validity in the measurement of attentional bias, which may thus reduce the ability to predict actual behavioural tendencies related to food. Complex “eating-related cues” would conceivably more closely approximate what one would observe within real-life eating scenarios. As those higher in external eating would be expected to be more sensitive to food cues in their everyday lives, bias towards complex food stimuli would thus be expected. However, there was no evidence of an attentional bias towards these “eating” stimuli. This begs the question as to whether evidence of attentional bias towards simple food stimuli can truly predict behavioural manifestations of the apparent bias in vivo. The null findings of hypothesis three in which attentional bias towards food was proposed as a moderator corroborate this notion.

Moreover, external eating was posited as a moderator of the social modeling of eating effect based upon previous studies that have established a link between external eating and attentional bias to food. Such studies, however, have relied upon visual-probe tasks using simple stimuli depicting items of food in isolation (e.g., Brignell et al., 2009; Hepworth et al., 2010; Hou et al., 2011). The null findings of hypothesis one examining external eating as a moderator may similarly reflect that attentional bias towards simple food stimuli may not necessarily predict appetitive behaviours. Thus, external eating and attentional bias to food may have been flawed in their capacity to enable inferences to the dynamic nature of real-life eating scenarios.

A more appropriate method to examine attentional bias may be with the use of eye-tracking. Future studies ought to examine attentional bias through eye-tracking to establish a

more ecologically-valid measure of attentional allocation through the assessment of the number and duration of fixations on food (Hermans et al., 2013). This may provide a more reliable determination as to: whether attention towards food can affect the strength of the social modeling of eating effect, whether those higher in external eating exhibit greater attention towards food in dynamic eating scenarios in vivo, and whether attention may influence the extent of mu rhythm desynchronization in response to the observation of eating behaviour. It may also be argued that underlying differences in individuals' level of external eating and attentional bias to food can be better captured by concepts that can be more reliably operationalized, such as frontal asymmetry.

Mediation Conditional on Frontal Asymmetry and BMI

The results of the exploratory analyses 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 sites C3 and F3. Within the overall first- and second-stage dual moderated mediation model, the coefficient between mu suppression at C3 and chip consumption (i.e., b_1 in Figure 6) was negative, in support of the notion that greater mu rhythm desynchronization (i.e., more negative values of M) recorded over sensorimotor brain regions was associated with greater chip consumption (i.e., higher values on Y). Thus, greater activity in the region associated with the MNS predicted a greater quantity of chips consumed. This indirect social modeling of eating effect, however, was conditional upon a first-stage moderation by individuals' level of frontal asymmetry and a second-stage moderation of BMI.

As mentioned previously, left frontal asymmetry has been associated with attentional narrowing towards appetitive stimuli (Harmon-Jones & Gable, 2009), as well as with higher levels of hedonic hunger and drive for palatable foods (Winter et al., 2016). External eating and

attentional bias towards food, the initially hypothesized moderators, may thus be markers of this underlying neural vulnerability to approach rewarding stimuli. External eating and attentional bias may be thought to conceptualize approach motivation more distally as a personality trait or behavioural manifestation. It is feasible that these distal variables may not exhibit enough power to influence neurophysiological activity. By contrast, frontal asymmetry represents a distinct neurological predisposition indicative of approach motivation (Smith et al., 2017). It may thus be more potent in its capacity to influence other neural processes. The use of frontal asymmetry recorded from the eating-view block provides further 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 (Smith et al., 2017). Regardless of its potential association with both external eating and attentional bias towards food, frontal asymmetry was found to significantly moderate the model in the pathway between model consumption and mu rhythm desynchronization.

The conditional indirect effect was only statistically significant among those with frontal asymmetry values in the 10th, 75th, and 90th percentiles. Interestingly, there was also a reversal of the indirect effect across these levels of frontal asymmetry. The indirect effect was negative for those who exhibited negative frontal asymmetry at the 10th percentile, reflective of right frontal asymmetry. Greater model consumption was associated with lower participant chip consumption through mu/alpha desynchronization among such individuals. Notably, Silva and colleagues (2002) found that restrained eaters exhibited greater right frontal asymmetry when compared to unrestrained eaters. The results of the present study 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. By contrast, the indirect effect was positive among those at the 75th and 90th percentiles of frontal asymmetry, indicative of left frontal asymmetry. For such individuals, greater model consumption was associated with a higher intake of chips, as mediated by mu/alpha desynchronization.

Exploration of the simple interaction between model consumption and frontal asymmetry ($X \times W$) in predicting mu/alpha suppression (M) suggested that left frontal asymmetry was associated with greater MNS activity, whereas right frontal asymmetry was associated with less MNS activity in response to the observation of eating. The presence or absence of reward may influence the degree of excitability in the MNS during action execution and observation (Brown & Brüne, 2014). Specifically, observing rewarding actions generates significantly greater mu suppression as compared to punishing or neutral actions (Brown, Wiersema, Pourtois, & Brüne, 2013). Notably, those with left frontal asymmetry tend to be more responsive to rewards (Pizzagalli et al., 2005). Thus, it may reasonably be expected that left frontal asymmetry would be associated with greater MNS excitability. Right frontal asymmetry, by contrast, has been associated with withdrawal-related motivation and avoidant responses (Smith et al., 2017). Theoretically, such individuals would be less likely to orient themselves towards, and perhaps even direct attention away, from rewarding appetitive stimuli. This pattern of avoidance could feasibly result in a reduction of MNS excitability. This first-stage simple moderation suggests that the influence of frontal asymmetry in either attenuating or enhancing MNS activity may be partially responsible for observed differences in the manifestation of social modeling of eating.

Within the second stage of the dual moderated mediation model, the positive moderation coefficient for $M \times V$ (i.e., b_3) indicates that as BMI increased, mu rhythm desynchronization was associated with fewer chips consumed. The indirect social modeling of eating effect as

moderated by frontal asymmetry was only significant among those with low to average BMIs (i.e., 20.08 to 23.72) in the sample. These values notably correspond to the “healthy” BMI range for adults (CDC, 2017). Across these BMI values, the moderated indirect effect was increasingly attenuated as BMI increased. Tentative speculations are subsequently advanced to explore possible reasons why this association may have demonstrated a progressive diminution among higher levels of BMI.

Frontal asymmetry, BMI, and the MNS. The gradual attenuation of the social modeling of eating effect among those with a higher BMI may be explained by the experience of diminished reward in response to chip consumption. Evidence indicates that repeatedly consuming palatable food for hedonic rather than homeostatic reasons can alter individuals’ reward response to food due to chronic activation of reward systems (Ely, Winter, & Lowe, 2013). Overconsumption of highly palatable foods appears to downregulate dopamine receptors. Among individuals who are overweight and obese, dopamine signaling appears to shift such that a greater reward response occurs in anticipation of food rather than in response to consumption itself, relative to those of average weight (Ely et al., 2013). That is, there is an attenuated dopamine release for food consumption. This may have effectively reduced the likelihood that individuals would have continued to engage in consumption when observing a model eating more compared to their leaner counterparts.

In a social eating scenario, observing one’s companion eating is often an anticipatory cue for imminent consumption of food for oneself (i.e., at a restaurant when one’s companion is served first). Given that individuals who are obese or overweight would still experience a similar reward response to anticipatory food cues, those with a higher BMI would not be expected to display differential MNS activity in response to the observation of eating compared to those of a

lower BMI. Thus, BMI would not be expected to influence the first-stage of the causal model. Notably, no differences in mu rhythm desynchronization emerged between those with a BMI above 25.0 compared to those with a BMI below 25.0, $t(91) = 0.18$, $p = 0.86$. To provide a rudimentary test of the speculative notion that reward from consumption attenuated the effect, a one-tailed Pearson correlation was conducted between BMI and participants' rating of how much they liked the chips. Recall that chip liking was rated immediately following ad libitum access to the chips. Interestingly, there was a trend towards a significant negative correlation, $r = -0.16$, $p = 0.07$, suggesting that those with a higher BMI tended to indicate liking the chips less. While this may reflect socially desirable responding, it may be informative for future studies to examine whether differential dopamine responses alter this modeling effect.

These findings may also be understandable with respect to motor resonance. Motor resonance is defined as specific muscle-to-action facilitation of the primary motor cortex during action observation (Cross & Iacoboni, 2014). The MNS is causally involved in motor resonance, such that neural pathways between the sensorimotor regions of the MNS and the primary motor cortex are activated when observing actions (Koch et al., 2010). Motor resonance is essentially responsible for the execution of imitation. However, it has been suggested that one may be able to strategically suppress motor resonance and therefore the activation of an imitative response if it is perceived to interfere with task goals (Cross & Iacoboni, 2014). For example, individuals displayed greater motor resonance when they were told to prepare to imitate a response compared to when they were told to counterimitate (i.e. perform a behaviour different from that observed; Cross & Iacoboni, 2014). In the present study, it is 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 and motor resonance when it is perceived that one should moderate food intake, either for social desirability or based on the model's consumption. Conger and colleagues (1980) proposed that overweight women may be particularly sensitive to social context cues in the presence of others due to greater self-consciousness and concern about deviating from a desired societal norm of weight. This may suggest that higher-order cognitive processes may determine the potential degree of influence the MNS may have in eliciting social modeling of eating.

It may be beneficial for future studies to examine the role of motor resonance within this effect using transcranial magnetic stimulation (TMS). More generally, this could further substantiate the potential causal role of the MNS in eliciting social modeling of eating via unconscious processes. Examination of motor resonance within this effect could also help to elucidate whether attenuated motor resonance can account for the apparent diminishing strength of the effect as a function of BMI. When utilizing TMS, an individual's response to a conditioning pulse depends on the level of excitability in the neural pathway at the time of stimulus application. Differences in the efficacy of the conditioning pulse can thus be used to ascertain the relative activation within certain cortical networks (Koch et al., 2010). Future studies could also benefit from including a measure of social desirability or body image concern

to delineate whether higher-order cognitive influences arising from concern about displaying control over food intake contribute to overriding unconscious mimicry.

Localization of the MNS

The findings from the exploratory analyses may additionally raise questions regarding the localization of the effect in the MNS. One may note that though the index of dual moderated mediation was significant at central site C3, it was not significant at C4. Previous studies have found that mu suppression appears to be more pronounced in the contralateral hemisphere relative to the hand that is observed to be moving (Perry & Bentin, 2009). As the right hand was performing the action of grabbing the chips and bringing them to the mouth, evidence of a significant effect in the left hemisphere (i.e., C3) but not in the right hemisphere (i.e., C4) appears to conform to this pattern. In addition, since 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 themselves, given that all participants were right-handed. Thus, mu suppression over the left central sensorimotor areas at C3 may therefore be expected to be more significantly linked to one's actual eating behaviours through motor resonance processes. This contralateral pattern was also evident at the frontal sites, with a significant effect only at F3 and not F4. Questions may additionally be raised, however, as to whether the existence of a significant effect at frontal electrode site F3 invalidates the findings as being suggestive of MNS involvement in the social modeling of eating phenomenon.

Mirror neuron studies via EEG have traditionally observed and reported mu suppression solely at central electrode sites (i.e. C3 and C4), defining mirror neuron activity as mu suppression over the central sensorimotor cortex (Gros et al., 2015). There is a notable paucity of research examining the extent to which suppression may be reliably evidenced across other scalp

regions (Frenkel-Toledo, Bentin, Perry, Liebermann, & Soroker, 2013). Fox and colleagues' (2015) recent meta-analysis concluded, however, that mu rhythm desynchronization in response to action observation does not appear to exhibit topographic specificity to central scalp locations. Unfortunately, vast inconsistencies exist across studies of mirror neurons with respect to the location and number of EEG channels in which activity has been examined and reported. This variability in methodology complicates the interpretation and localization of MNS effects. The focus on central electrodes within EEG studies additionally stands in contrast to the established involvement of regions in the frontal and premotor cortices in the MNS across other neurophysiological methodology, such as fMRI (Fox et al., 2015).

In a meta-analysis of 125 fMRI studies, Molenberghs, Cunnington, and Mattingley (2012) revealed that the inferior frontal gyrus, ventral premotor cortex, dorsal premotor cortex, inferior and superior parietal lobes, temporal gyrus, and cerebellum demonstrated mirroring properties. These regions displayed activity in response to both the execution and observation of motor actions, supporting the existence of a MNS in areas dispersed across the cortical mantle. fMRI activation in the posterior inferior frontal gyrus and Brodmann Area 6 in response to the observation and execution of actions has also been implicated to indicate mirror neuron activity (Press, Weiskopf, & Kilner, 2012). This area notably corresponds to frontal electrode sites. Additionally, in a meta-analysis of 20 fMRI studies, Molenberghs, Cunnington, and Mattingley (2009) found consistent evidence of suppression in frontal regions in studies investigating imitative actions, specifically within the dorsal premotor cortex.

Within the smaller subset of EEG studies of the MNS that have reported on areas beyond central sites, frontal sites have also generally been found to demonstrate mirroring properties. Frenkel-Toledo and colleagues (2013) specifically sought to examine the distribution of mu

suppression over the scalp and found significant suppression was indexed at frontal, central, occipital, and parietal sites in response to action observation. Mu suppression was notably strongest, however, at the central and frontal sites (Frenkel-Toledo et al., 2013). Puzzo and colleagues (2010) also found EEG evidence of suppression over the premotor cortex and supplementary motor area (i.e., FC3, FC1, FCz, FC2, and FC4), frontal brain regions, during the observation of hand actions. Perhaps then the question should be raised as to whether it is methodologically appropriate based on what is known about the MNS to focus solely on central electrode sites in EEG studies (Frenkel-Toledo et al., 2013). It should, however, be emphasized that the findings of the current study suggestive of suppression at site F3 do not constitute substantive evidence to attest to reliable involvement of frontal sites in the MNS.

In the current study, some degree of topographic specificity was also demonstrated with respect to a lack of suppression within occipital sites. Contrary to previously noted criticisms that mu rhythm desynchronization may merely reflect attentional processes originating in the occipital lobes, none of the effects were significant at the occipital electrode sites (i.e., O1 and O2). In fact, coefficient estimates were considerably attenuated in comparison to those of the frontally- and centrally-located sites. Thus, it cannot be argued that evidence of mu suppression was merely an artifact of attentional processing demands in the present study. It is possible that studies of the MNS that have demonstrated suppression at occipital sites (e.g., Perry & Bentin, 2010; Perry, Stein, & Bentin, 2011; Perry et al., 2010), may be those in which the baseline condition was significantly less attentionally-engaging than the experimental condition (i.e. a ball moving across the screen, a static image, or eyes-closed). An effect may feasibly only arise at occipital sites when there is a greater discrepancy between the attention captured by the baseline and experimental conditions. In such instances, greater attentional engagement to the

experimental stimuli could manifest as evidence of significant suppression in the occipital lobes when compared to baseline activity. However, it may be separate from MNS activity and merely modulated in parallel. Vast variability in the type of baseline measures utilized across MNS studies makes such a speculation possible. Studies have begun to examine whether the type of baseline utilized (i.e., within-trial, between-trial, etc.) alters whether an effect is additionally discovered at occipital sites (i.e. Hobson & Bishop, 2016). Further research may also be beneficial to elucidate whether the specific baseline stimuli (i.e., biological vs. non-biological, dynamic vs. static, etc.) influences whether or not occipital suppression emerges to help elucidate the meaning behind occipital involvement in the context of the MNS.

There is a definite need for widespread agreement as to how to quantify MNS activation, with specificity as to the key regions to explore. Future studies examining MNS activity via EEG ought to report on scalp sites beyond central electrodes to help in clarifying the localization of suppression effects over the cortical mantle to discern the expanse of the MNS. Given that areas beyond central sites have been implicated in fMRI studies, efforts should be made to establish convergence between the fMRI and EEG literature. The National Institute on Mental Health's (NIMH) Research Domain Criteria initiative has stated that constructs ought to be examined across multiple units of analysis to verify convergence (Gorka, Phan, & Shankman, 2015). Agreement between complimentary brain mapping techniques can further bolster the existence of a distinctive system of mirror neurons in the human brain. Greater methodological consensus can provide more reliable analyses of MNS activity and allow for progression in this field.

Contribution to the Social Modeling of Eating Effect

Cruwys and colleagues (2015) criticized that the social modeling of eating effect lacks a parsimonious model that can account for why moderators of this phenomenon may or may not

exist and why the effect is so robust. The potential existence of a neurophysiological causal mechanism appears to offer a relatively promising parsimonious model to explain instances of the seemingly automatic, unconscious mimicry process that occurs in social eating scenarios. One previous explanatory account of the social modeling of eating effect proposed that modeling may arise due to concerns about conforming to social norms. Participants in the current study, however, were unaware that their eating behaviour was of any interest and still exhibited this pattern of modeling. Thus, it is unlikely that participants consciously sought to match their eating behaviour to the model. Such an explanation would be more plausible if participants engaged in a taste-test paradigm, thereby creating greater uncertainty as to how much would be appropriate to consume in the experimental context.

An alternative explanatory account has proposed that social modeling of eating may reflect individuals' desire to affiliate and strengthen the social bond with one's eating companion (Cruwys et al., 2015). For example, women who endorsed a higher need to please others and maintain social harmony ate more when they believed their eating companion wanted them to eat more (Exline, Zell, Bratslavsky, Hamilton, & Swenson, 2012). A remote confederate design such as the one in the current study, however, challenges this explanation. In such instances, there is no opportunity for a social bond to be created and yet the effect remains. Involvement of the MNS also does not preclude the possibility that social bonding may influence the strength of this effect. One study demonstrated that external administration of intranasal oxytocin, a central hormone involved in social bonding, enhanced mu rhythm suppression (Perry et al., 2010). Thus, the facilitation of this effect by social bonding processes may still be possible to understand with respect to the MNS. Nevertheless, the involvement of cognitive processes and the role of normative information in this effect cannot be entirely negated. As demonstrated in the current

study, the MNS's role in this effect may still be malleable to higher-order cognitive processes and the exertion of control over one's desired goals. These findings also necessitate replication to be substantiated and must be considered in the context of the study's strengths and limitations.

Strengths, Limitations, and Future Directions

The current findings are primarily limited by the inclusion of only female undergraduate participants. This was founded upon evidence of a stronger social modeling of eating effect within females (Vartanian et al., 2015), as well as the documented importance of 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 it 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 has particular limitations. As a neurophysiological index, EEG has relatively poor spatial resolution compared to techniques such as fMRI (Fox et al., 2015). In fact, some studies have called into question the validity of examining the MNS with mu rhythm, as EEG activity recorded at particular sites may not necessarily reflect cortical activity directly below those electrodes (Fox et al., 2015). Cohen (2017) notes, however, that EEG is capable of reasonably accurate anatomical localization within centimeters. The preponderance of conclusions linking EEG topography to cognitive, perceptual, or motor processes only necessitate precision at the centimeter scale (Cohen, 2017). As an alternative, the use of neuroimaging techniques with superior spatial resolution, such as fMRI, in concert with EEG may also provide greater specificity with respect to the localization of mirror neurons. However, fMRI is notably more expensive (Fox et al., 2015). An additional alternative to improve EEG studies of the MNS may be the use of the frequency-domain measure of

coherence, which “...quantifies the degree of covariance between two spatially separate signals” (Miskovic & Schmidt, 2010).

Complex neurological processes necessitate coordination among various neuronal assemblies (Miskovic & Schmidt, 2010). Coherence between two cortical areas is thus thought to reflect neural connectivity and functional communication. One recent study examined MNS activity using imaginary coherence and calculated the linear dependency of two signals phase-lagged to each other in the mu/alpha band (Kelly et al., 2015). Using this method, pairs of electrodes were selected and regions of interest were identified to derive parietal-frontal networks for examination (i.e. right parietal and premotor pair P6 + F4). Kelly and colleagues (2015) found evidence indicative of coherence between parietal-premotor networks when participants observed hand motions. Measurement of coherence proffers the ability to remove overestimation biases that may occur due to volume conduction and other artifacts (Kelly et al., 2015). Such methodology may thus be desirable in future studies to discern whether suppression effects at multiple electrode sites merely reflect feedforward or backward activation across the scalp or a true expression of a cortical network reflective of the MNS.

Alternatively, examining the involvement of MNS activity via EEG event-related desynchronization (ERD) in future studies may help to establish that 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 to the study. However, mirror neuron literature has indicated that individuals may also exhibit mu suppression in response to facial expressions, particularly when those expressions appear directed towards

the viewer (Ensenberg, Perry, & Aviezer, 2016). Though the facial expressions displayed were standardized across both the high- and low-intake conditions, MNS activation resulting from facial expressions may have confounded suppression in response to the observation of eating. This may have diminished the power of mu suppression to predict eating behaviour, perhaps helping to account for the null findings of hypotheses one and three.

One of the notable strengths of the current study was its 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 (Fox et al., 2015), which may result in underpowered study designs. In Fox and colleagues' (2015) meta-analysis examining EEG studies of mu rhythm desynchronization, the smallest sample size for an action observation condition consisted of six participants, while the largest included 40 participants. The current study's sample of 93 participants is thus a notable strength. Based on the effect sizes of studies in Fox and colleagues' (2015) 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. It is thus reasonable to conclude that the study was adequately powered to detect an effect.

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 (Quaedflieg et al., 2016). It has also been demonstrated that encouraging individuals to adopt an action-oriented mindset can elicit

increased left frontal asymmetry (Harmon-Jones, Harmon-Jones, Fearn, Sigelman, & Johnson, 2008). This suggests that it may be possible to temporarily modify frontal asymmetry. If this causal model can be further substantiated, it may be possible to apply these findings in a clinical setting. Speculative musings of clinical applications are briefly noted henceforth.

Clinical Implications

Investigations of social influences on eating suggest there are real consequences of modeling at a population level. The basic social influence process that undergirds the modeling of food intake can influence the adoption of dangerous dieting behaviours as well as patterns of overeating, which may contribute to disordered eating or obesity (Cruwys et al., 2015). If future studies can replicate the present findings indicating that frontal asymmetry alters one's susceptibility to social influences, 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, making such hypothetical applications feasible. For example, the eBrainAvatar Muse Mood app (BrainMaster Technologies, Inc., Canada) utilizes a wireless 4-channel EEG headband to show moment-to-moment changes in power asymmetry. This technology could thus potentially be harnessed for such neurofeedback training.

Heightened concern regarding the "obesity epidemic" and evidence of the spread of obesity among social networks (Christakis & Fowler, 2007) arguably creates an impetus to understand and manage factors that make individuals susceptible to social influences on eating. The present 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 and overeating within families. It has been

demonstrated, for example, that parental obesity status early in childhood is a strong predictor of later adult obesity (Wardle, Sanderson, Guthrie, Rapoport, & Plomin, 2002). Children's modeling of excessive parental intake is posited to contribute to this association (Wardle et al., 2002). Targeting children at high familial risk of obesity early may decrease the likelihood that they will model parents' excessive consumption or achieve an unhealthy weight. Conversely, these findings could have implications in the context of treatment for eating disorders. As mentioned, chronic restrained eaters tend to exhibit right frontal asymmetry at rest (Silva et al., 2002). It could be explored whether inducing left frontal asymmetry prior to a meal could encourage approximating a more appropriate intake in severe inpatient cases. This could perhaps help to encourage the establishment of more regular patterns of consumption. However, it would be necessary to explore whether this effect can be evidenced among those with a BMI less than 20.0, as only one tenth of the current sample had such a low BMI.

To this author's knowledge, 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 and colleagues (2012) alluded to the involvement of "a mirroring network" (p. 4) in explaining the close correspondence of bites, or bite mimicry, 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 needed in order to elucidate the influence of the MNS on human social eating behaviour and to attempt to replicate the current findings. Overall, the current study suggests that among low- to average-weight individuals, those with a predisposition towards an approach-motivation style due to left frontal asymmetry may be more susceptible to engage in social modeling of eating through the corresponding enhancement of mirror neuron activity.

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Table 1

Means and Standard Deviations of Reaction Times (ms) Across Visual Probe Variables and Stimuli Durations

| Variables | 500 ms <i>M (SD)</i> | 2,000 ms <i>M (SD)</i> |
|---|-------------------------|---------------------------|
| Attentional Bias Scores | | |
| Food-related cues | 5.73 (28.46) | 4.96 (27.87) |
| Eating-related cues | 0.16 (25.20) | 0.75 (30.50) |
| Food-related cue vs. non-food cue | | |
| Probe replacing food-related cue | 430.44 (51.86) | 425.93 (50.42) |
| Probe replacing non-food cue | 436.17 (52.01) | 430.89 (54.75) |
| Eating-related cue vs. non-food human cue | | |
| Probe replacing eating-related cue | 435.22 (54.12) | 431.11 (54.58) |
| Probe replacing non-food human cue | 435.38 (54.82) | 431.86 (58.86) |
| Non-food cue vs. a non-food human cue | | |
| Probe replacing non-food human cue | 434.38 (59.23) | 429.39 (54.67) |
| Probe replacing non-food cue | 438.88 (56.34) | 431.17 (56.26) |
| Non-food cue vs. non-food cue | | |
| Probe replacing non-food cue on left | 439.71 (61.56) | 431.10 (55.47) |
| Probe replacing non-food cue on right | 434.32 (58.03) | 426.97 (55.79) |

Note. $N = 93$. Non-food cue vs. non-food cue blocks were considered neutral and thus RTs were differentiated based on where the probe was located.

Table 2

Means, Standard Deviations, Reliability Coefficients, and Skew of Psychometric Variables and Sample Characteristics

| | Mean (<i>SD</i>) | No. of items | α | z_{skewness} |
|------------------------------|--------------------|--------------|----------|-----------------------|
| DEBQ – External eating | 3.24 (0.60) | 10 | .84 | 0.21 |
| DEBQ – Emotional eating | 2.51 (0.82) | 13 | .94 | 1.20 |
| DEBQ – Restrained eating | 2.36 (0.75) | 10 | .90 | 0.31 |
| DEBQ – Total score | 2.67 (0.56) | 33 | .93 | 1.02 |
| AB towards food cues | 5.89 (20.87) | -- | -- | 1.88 |
| AB towards eating cues | 0.72 (18.44) | -- | -- | 1.73 |
| BMI | 24.57 (4.30) | -- | -- | 4.39 |
| Chip consumption in g | 19.76 (17.01) | -- | -- | 2.83 |
| Chip consumption in g (sqrt) | 3.84 (2.25) | -- | -- | -0.91 |

Note. $N = 93$. DEBQ = Dutch Eating Behaviour Questionnaire; AB = Attentional bias; BMI = Body mass index; α = Cronbach's index of internal consistency. $z_{\text{skewness}} = \text{skewness}/SE$; ± 1.96 considered skewed. Chip consumption in g (sqrt) reflects the square root transformation of participants' chip consumption in grams used in all analyses.

Table 3

Means, Standard Deviations, and $z_{skewness}$ of Raw EEG Power of Baseline and Eating-View Recording Blocks and EEG Mu/Alpha Suppression Ratios

| Electrode site | Baseline raw power | | Eating-view raw power | | Mu/alpha suppression ratio | |
|----------------|--------------------|----------------|-----------------------|----------------|----------------------------|----------------|
| | $M(SD)$ | $z_{skewness}$ | $M(SD)$ | $z_{skewness}$ | $M(SD)$ | $z_{skewness}$ |
| C3 | 8.10 (5.89) | 4.88 | 10.26 (8.03) | 4.99 | 0.20 (0.34) | 2.01 |
| C4 | 7.96 (5.40) | 6.00 | 10.39 (8.16) | 5.82 | 0.21 (3.22) | 2.46 |
| F3 | 7.53 (4.40) | 7.34 | 8.87 (6.32) | 8.11 | 0.12 (0.28) | 1.01 |
| F4 | 7.78 (4.49) | 7.04 | 9.08 (6.10) | 6.95 | 0.12 (0.28) | 2.62 |
| O1 | 4.70 (3.90) | 14.08 | 6.80 (6.16) | 9.80 | 0.27 (0.42) | 1.90 |
| O2 | 5.03 (6.78) | 29.78 | 6.38 (5.31) | 8.34 | 0.24 (0.40) | 0.79 |

Note. $N = 93$. Raw EEG mu/alpha power expressed in μV^2 within the 8-13 Hz band. EEG mu/alpha suppression ratios reflect the log transformed ratio of power at each electrode site during the eating-view divided by the baseline.

Table 4

Unstandardized Regression Coefficients, Standard Errors, and 95% Confidence Intervals of PROCESS Model 7 Estimating Participant Consumption (Y) from Model Consumption (X) through Mu/Alpha Suppression (M) as Moderated by External Eating (W)

| Antecedent | Consequent | | | |
|-----------------------|-----------------------------|---------------|-----------------------------|---------------------|
| | Model A: C3 mu (M_I) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.341 (0.504) | -0.662, 1.343 | 1.007* (0.404) | 0.204, 1.810 |
| Mu suppression (M) | -- | -- | -0.769 (0.549) | -1.861, 0.323 |
| External eating (W) | -0.010 (0.100) | -0.209, 0.189 | -- | -- |
| $X \times W$ | -0.123 (0.151) | -0.427, 0.175 | -- | -- |
| Constant | 0.352 (0.373) | -0.390, 1.095 | -0.319 (2.886) | -6.059, 5.420 |
| | $R^2 = 0.187$ | | $R^2 = 0.479$ | |
| | $F(9, 83) = 2.190^*$ | | $F(8, 84) = 15.657^{***}$ | |
| Antecedent | Consequent | | | |
| | Model B: C4 mu (M_I) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.132 (0.507) | -0.876, 1.141 | 1.023* (0.399) | 0.231, 1.816 |
| Mu suppression (M) | -- | -- | -0.702 (0.527) | -1.749, 0.346 |
| External eating (W) | -0.030 (0.101) | -0.231, 0.171 | -- | -- |
| $X \times W$ | -0.055 (0.153) | -0.359, 0.249 | -- | -- |
| Constant | 0.336 (0.363) | -0.387, 1.058 | -0.394 (2.854) | -6.070, 5.282 |
| | $R^2 = 0.183$ | | $R^2 = 0.476,$ | |
| | $F(9, 83) = 2.399^*$ | | $F(8, 84) = 15.775^{***}$ | |
| Antecedent | Consequent | | | |
| | Model D: F3 alpha (M_I) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.259 (0.398) | -0.533, 1.051 | 1.025* (0.395) | 0.239, 1.811 |
| Alpha suppression (M) | -- | -- | -1.066 ⁺ (0.582) | -2.222, 0.091 |
| External eating (W) | -0.076 (0.083) | -0.241, 0.090 | -- | -- |
| $X \times W$ | -0.089 (0.120) | -0.328, 0.149 | -- | -- |
| Constant | 0.377 (0.354) | -0.328, 1.081 | -0.335 (2.855) | -6.013, 5.342 |
| | $R^2 = 0.168,$ | | $R^2 = 0.483,$ | |
| | $F(9, 83) = 1.235$ | | $F(8, 84) = 15.724^{***}$ | |
| Antecedent | Consequent | | | |
| | Model E: F4 alpha (M_I) | | Y (Participant consumption) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.218 (0.436) | -0.649, 1.085 | 0.994* (0.401) | 0.198, 1.791 |
| Alpha suppression (M) | -- | -- | -0.935 (0.595) | -2.119, 0.248 |
| External eating (W) | -0.060 (0.088) | -0.236, 0.115 | -- | -- |
| $X \times W$ | -0.088 (0.130) | -0.347, 0.171 | -- | -- |
| Constant | 0.350 (0.371) | -0.387, 1.087 | -0.364 (2.871) | -6.074, 5.345 |
| | $R^2 = 0.168,$ | | $R^2 = 0.480,$ | |
| | $F(9, 83) = 1.278$ | | $F(8, 84) = 15.387^{***}$ | |

| Antecedent | Consequent | | | |
|---------------------------|-----------------------------|---------------|-----------------------------|---------------------|
| | Model F: O1 alpha (M_1) | | Y (Participant consumption) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.216 (0.621) | -1.018, 1.450 | 1.097** (0.396) | 0.308, 1.885 |
| Alpha suppression (M) | -- | -- | -0.575 (0.443) | -1.456, 0.306 |
| External eating (W) | -0.052 (0.116) | -0.282, 0.178 | -- | -- |
| $X \times W$ | -0.044 (0.185) | -0.412, 0.324 | -- | -- |
| Constant | 0.415 (0.585) | -0.748, 1.578 | -0.412 (2.781) | -5.941, 5.118 |
| | $R^2 = 0.090$ | | $R^2 = 0.478$ | |
| | $F(9, 83) = 0.613$ | | $F(8, 84) = 15.072^{***}$ | |

| Antecedent | Consequent | | | |
|---------------------------|-----------------------------|---------------|---------------------------------|---------------------|
| | Model F: O2 alpha (M_1) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.109 (0.543) | -0.971, 1.188 | 1.082** (0.399) | 0.289, 1.874 |
| Alpha suppression (M) | -- | -- | -0.453 (0.449) | -1.345, 0.439 |
| External eating (W) | -0.064 (0.108) | -0.278, 0.150 | -- | -- |
| $X \times W$ | -0.015 (0.164) | -0.340, 0.311 | -- | -- |
| Constant | 0.542 (0.579) | -0.610, 1.693 | -0.407 (2.805) | -5.985, 5.172 |
| | $R^2 = 0.107$ | | $R^2 = 0.473$ | |
| | $F(9, 83) = 0.579$ | | $F(8, 84) = 14.921^{***}$ | |

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

⁺ $p < .07$, * $p < .05$, *** $p < .001$.

Table 5

Unstandardized Regression Coefficients, Standard Errors, and Bias-Corrected Bootstrap 95% Confidence Intervals Testing the Conditional Indirect Effect of Model Consumption (X) on Participant Consumption (Y) through Mediating Mu /Alpha Suppression at Moderating Levels of External Eating (W: DEBQ – External Eating)

| External Eating (W) | Model A: C3 Mu Suppression (M_I) | | Model B: C4 Mu Suppression (M_I) | | Model C: F3 Alpha Suppression (M_I) | |
|------------------------|--|---------------|--|---------------|--|---------------|
| | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| a_3b_1 | 0.097 (0.147) | -0.071, 0.582 | 0.039 (0.120) | -0.131, 0.389 | 0.100 (0.146) | -0.117, 0.492 |
| -1 <i>SD</i> = 2.636 | -0.007 (0.103) | -0.271, 0.168 | 0.009 (0.090) | -0.147, 0.233 | -0.025 (0.110) | -0.306, 0.151 |
| <i>M</i> = 3.237 | 0.052 (0.068) | -0.027, 0.278 | 0.032 (0.062) | -0.038, 0.257 | 0.032 (0.072) | -0.068, 0.252 |
| +1 <i>SD</i> = 3.838 | 0.110 (0.119) | -0.034, 0.508 | 0.055 (0.100) | -0.067, 0.366 | 0.089 (0.116) | -0.069, 0.431 |
| External Eating (W) | Model D: F4 Alpha Suppression (M_I) | | Model E: O1 Alpha Suppression (M_I) | | Model F: O2 Alpha Suppression (M_I) | |
| | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| a_3b_1 | 0.082 (0.137) | -0.108, 0.474 | 0.025 (0.114) | -0.161, 0.326 | 0.007 (0.093) | -0.157, 0.234 |
| -1 <i>SD</i> = 2.636 | 0.013 (0.103) | -0.166, 0.272 | -0.057 (0.101) | -0.366, 0.070 | -0.032 (0.079) | -0.300, 0.064 |
| <i>M</i> = 3.237 | 0.063 (0.072) | -0.023, 0.299 | -0.042 (0.072) | -0.268, 0.040 | -0.028 (0.058) | -0.242, 0.037 |
| +1 <i>SD</i> = 3.838 | 0.112 (0.115) | -0.038, 0.450 | -0.027 (0.098) | -0.372, 0.081 | -0.024 (0.083) | -0.362, 0.056 |

Note. $N = 93$. DEBQ = Dutch Eating Behaviour Questionnaire; a_3b_1 = Index of moderated mediation. None of the 95% CIs excluded zero in the model.

Table 6

Unstandardized Regression Coefficients, Standard Errors, and 95% Confidence Intervals of PROCESS Model 7 Estimating Participant Consumption (Y) from Model Consumption (X) through Mu/Alpha Suppression (M) as Moderated by Attentional Bias towards Food (W)

| Antecedent | Consequent | | | |
|-----------------------|-----------------------------|---------------|-----------------------------|---------------------|
| | Model A: C3 mu (M_I) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | -0.031 (0.077) | -0.185, 0.123 | 1.030* (0.411) | 0.213, 1.847 |
| Mu suppression (M) | -- | -- | -0.758 (0.570) | -1.892, 0.376 |
| Attentional bias (W) | 0.001 (0.002) | -0.003, 0.005 | -- | -- |
| $X \times W$ | -0.003 (0.003) | -0.010, 0.003 | -- | -- |
| Constant | 0.372 (0.290) | -0.205, 0.948 | -0.932 (2.932) | -6.760, 4.897 |
| | $R^2 = 0.171$ | | $R^2 = 0.449$ | |
| | $F(7, 85) = 1.820^+$ | | $F(6, 86) = 16.870^{***}$ | |
| Antecedent | Consequent | | | |
| | Model B: C4 mu (M_I) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | -0.005 (0.071) | -0.145, 0.136 | 1.044 (0.404) | 0.240, 1.847 |
| Mu suppression (M) | -- | -- | -0.734 (0.540) | -1.808, 0.340 |
| Attentional bias (W) | 0.001 (0.002) | -0.004, 0.005 | -- | -- |
| $X \times W$ | -0.005 (0.003) | -0.011, 0.002 | -- | -- |
| Constant | 0.279 (0.268) | -0.254, 0.812 | -0.998 (2.888) | -6.738, 4.743 |
| | $R^2 = 0.197,$ | | $R^2 = 0.447,$ | |
| | $F(7, 85) = 2.168^*$ | | $F(6, 86) = 17.230^{***}$ | |
| Antecedent | Consequent | | | |
| | Model D: F3 alpha (M_I) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | -0.014 (0.065) | -0.142, 0.115 | 1.050* (0.400) | 0.255, 1.845 |
| Alpha suppression (M) | -- | -- | -1.017 ⁺ (0.564) | -2.138, 0.105 |
| Attentional bias (W) | 0.000 (0.002) | -0.003, 0.003 | -- | -- |
| $X \times W$ | -0.001 (0.002) | -0.006, 0.004 | -- | -- |
| Constant | 0.280 (0.308) | -0.333, 0.892 | -0.943 (2.875) | -6.658, 4.772 |
| | $R^2 = 0.105,$ | | $R^2 = 0.453,$ | |
| | $F(7, 85) = 0.911$ | | $F(6, 86) = 20.014^{***}$ | |
| Antecedent | Consequent | | | |
| | Model E: F4 alpha (M_I) | | Y (Participant consumption) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | -0.048 (0.065) | -0.178, 0.081 | 1.021* (0.407) | 0.213, 1.829 |
| Alpha suppression (M) | -- | -- | -0.871 (0.572) | -2.008, 0.265 |
| Attentional bias (W) | -0.000 (0.001) | -0.003, 0.003 | -- | -- |
| $X \times W$ | -0.002 (0.002) | -0.006, 0.003 | -- | -- |
| Constant | -0.266 (0.308) | -0.347, 0.878 | -0.994 (2.889) | -6.738, 4.749 |
| | $R^2 = 0.121,$ | | $R^2 = 0.449,$ | |
| | $F(7, 85) = 0.911$ | | $F(6, 86) = 18.532^{***}$ | |

| Antecedent | Consequent | | | |
|---------------------------|-----------------------------|---------------|-----------------------------|---------------------|
| | Model F: O1 alpha (M_1) | | Y (Participant consumption) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.095 (0.097) | -0.098, 0.288 | 1.111** (0.401) | 0.314, 1.908 |
| Alpha suppression (M) | -- | -- | -0.443 (0.395) | -1.229, 0.343 |
| Attentional bias (W) | 0.000 (0.002) | -0.003, 0.004 | -- | -- |
| $X \times W$ | -0.002 (0.004) | -0.009, 0.006 | -- | -- |
| Constant | 0.350 (0.549) | -0.740, 1.441 | -1.081 (2.828) | -6.702, 4.540 |
| | $R^2 = 0.025$ | | $R^2 = 0.445$ | |
| | $F(7, 85) = 0.343$ | | $F(6, 86) = 15.703***$ | |
| Antecedent | Consequent | | | |
| | Model G: O2 alpha (M_1) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.067 (0.086) | -0.103, 0.237 | 1.094** (0.402) | 0.296, 1.893 |
| Alpha suppression (M) | -- | -- | -0.285 (0.392) | -1.064, 0.494 |
| Attentional bias (W) | 0.001 (0.002) | -0.003, 0.005 | -- | -- |
| $X \times W$ | -0.001 (0.003) | -0.008, 0.006 | -- | -- |
| Constant | 0.425 (0.539) | -0.647, 1.496 | -1.126 (2.844) | -6.780, 4.528 |
| | $R^2 = 0.050$ | | $R^2 = 0.440$ | |
| | $F(7, 85) = 0.625$ | | $F(6, 86) = 16.352***$ | |

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

⁺ $p = .08$, * $p < .05$, *** $p < .001$.

Table 7

Unstandardized Regression Coefficients, Standard Errors, and Bias-Corrected Bootstrap 95% Confidence Intervals Testing the Conditional Indirect Effect of Model Consumption (X) on Participant Consumption (Y) through Mediating Mu /Alpha Suppression at Moderating Levels of Attentional Bias towards Food (W)

| AB (W) | Model A: C3 Mu Suppression (M_1) | | Model B: C4 Mu Suppression (M_1) | | Model C: F3 Alpha Suppression (M_1) | |
|-----------------|--|---------------|--|---------------|--|---------------|
| | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| a_3b_1 | 0.003 (0.003) | -0.001, 0.014 | 0.003 (0.004) | -0.001, 0.015 | 0.001 (0.003) | -0.003, 0.009 |
| -1 SD = -14.981 | -0.015 (0.094) | -0.306, 0.113 | -0.048 (0.094) | -0.366, 0.057 | -0.003 (0.093) | -0.235, 0.157 |
| $M = 5.893$ | 0.039 (0.068) | -0.044, 0.249 | 0.024 (0.060) | -0.058, 0.214 | 0.021 (0.067) | -0.100, 0.174 |
| +1 SD = 26.766 | 0.094 (0.101) | -0.030, 0.422 | 0.095 (0.102) | -0.025, 0.422 | 0.045 (0.088) | -0.091, 0.275 |
| AB (W) | Model D: F4 Alpha Suppression (M_1) | | Model E: O1 Alpha Suppression (M_1) | | Model F: O2 Alpha Suppression (M_1) | |
| | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| a_3b_1 | 0.001 (0.003) | -0.002, 0.010 | 0.001 (0.002) | -0.002, 0.008 | 0.000 (0.002) | -0.002, 0.005 |
| -1 SD = -14.981 | 0.023 (0.085) | -0.113, 0.246 | -0.053 (0.086) | -0.375, 0.035 | -0.023 (0.056) | -0.281, 0.030 |
| $M = 5.893$ | 0.050 (0.065) | -0.032, 0.249 | -0.038 (0.067) | -0.266, 0.032 | -0.018 (0.048) | -0.218, 0.030 |
| +1 SD = 26.766 | 0.077 (0.087) | -0.033, 0.350 | -0.023 (0.077) | -0.284, 0.070 | -0.013 (0.061) | -0.231, 0.057 |

Note. $N = 93$. AB = Attentional bias towards food; a_3b_1 = Index of moderated mediation. None of the 95% CIs excluded zero in the model.

Table 8

Unstandardized Regression Coefficients, Standard Errors, and 95% Confidence Intervals of PROCESS Model 21 Estimating Participant Consumption (Y) from Model Consumption (X) through Mu/Alpha Suppression (M) as Moderated by Frontal Asymmetry (W) and BMI (V)

| Antecedent | Consequent | | | |
|-----------------------|-----------------------------|--|---|------------------------|
| | Model A: C3 mu (M_I) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.053 (0.075) | -0.096, 0.201 | 1.012* (0.403) | 0.210, 1.814 |
| Mu suppression (M) | -- | -- | -8.146* (3.749) | -15.603, -0.689 |
| Frontal asymmetry (W) | 1.773* (0.877) | 0.030, 3.516 | -- | -- |
| X × W | -2.382* (0.728) | -3.831, -0.934 | -- | -- |
| BMI (V) | -- | -- | -0.050 (0.056) | -0.162, 0.063 |
| M × V | -- | -- | 0.301 ⁺ (0.155) | -0.007, 0.610 |
| Constant | 0.349 (0.301) | -0.250, 0.947 | 0.266 (3.052) | -5.805, 6.337 |
| | | $R^2 = 0.295,$ $F(8, 84) = 3.714^{**}$ | $R^2 = 0.485,$ $F(9, 83) = 11.947^{***}$ | |
| Antecedent | Consequent | | | |
| | Model B: C4 mu (M_I) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.078 (0.069) | -0.060, 0.215 | 1.061* (0.410) | 0.246, 1.876 |
| Mu suppression (M) | -- | -- | -5.322 (3.305) | -11.897, 1.252 |
| Frontal asymmetry (W) | 2.255** (0.781) | 0.701, 3.809 | -- | -- |
| X × W | -2.374*** (0.640) | -3.646, -1.102 | -- | -- |
| BMI (V) | -- | -- | -0.018 (0.064) | -0.145, 0.109 |
| M × V | -- | -- | 0.185 (0.130) | -0.073, 0.444 |
| Constant | 0.286 (0.288) | -0.287, 0.860 | -0.586 (3.219) | -6.989, 5.817 |
| | | $R^2 = 0.328,$ $F(8, 84) = 4.448^{***}$ | $R^2 = 0.463,$ $F(9, 83) = 11.393^{***}$ | |
| Antecedent | Consequent | | | |
| | Model C: F3 alpha (M_I) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.037 (0.060) | -0.082, 0.156 | 1.073* (0.407) | 0.264, 1.883 |
| Alpha suppression (M) | -- | -- | -8.665 ⁺⁺ (4.454) | -17.302, 0.686 |
| Frontal asymmetry (W) | 0.318 (0.724) | -1.121, 1.758 | -- | -- |
| X × W | -2.142** (0.703) | -3.540, -0.743 | -- | -- |
| BMI (V) | -- | -- | -0.020 (0.061) | -0.141, 0.101 |
| M × V | -- | -- | 0.297 (0.187) | -0.074, 0.668 |
| Constant | 0.204 (0.310) | -0.412, 0.819 | -0.632 (3.108) | -6.813, 5.550 |
| | | $R^2 = 0.284,$ $F(8, 84) = 2.261^*$ | $R^2 = 0.472,$ $F(9, 83) = 12.832^{***}$ | |

| Antecedent | Consequent | | | |
|---------------------------|-----------------------------|---|---|---------------------|
| | Model D: F4 alpha (M_1) | | Y (Participant consumption) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.036 (0.060) | -0.084, 0.155 | 1.020* (0.407) | 0.211, 1.829 |
| Alpha suppression (M) | -- | -- | -7.650 ⁺ (3.956) | -15.519, 0.218 |
| Frontal asymmetry (W) | 1.343 ⁺⁺ (0.718) | -0.086, 2.771 | -- | -- |
| $X \times W$ | -2.228** (0.680) | -3.580, -0.876 | -- | -- |
| BMI (V) | -- | -- | -0.012 (0.060) | -0.132, 0.106 |
| $M \times V$ | -- | -- | 0.267 (0.161) | -0.054, 0.588 |
| Constant | 0.218 (0.310) | -0.398, 0.834 | -0.843 (3.118) | -7.045, 5.358 |
| | | $R^2 = 0.293,$ $F(8, 84) = 2.989^{**}$ | $R^2 = 0.468,$ $F(9, 83) = 13.473^{***}$ | |

| Antecedent | Consequent | | | |
|---------------------------|-----------------------------|---|---|---------------------|
| | Model E: O1 alpha (M_1) | | Y (Participant consumption) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.215 (0.093) | -0.031, 0.399 | 1.124** (0.427) | 0.276, 1.972 |
| Alpha suppression (M) | -- | -- | -3.103 (3.990) | -11.039, 4.833 |
| Frontal asymmetry (W) | 1.878 (1.050) | -0.210, 3.965 | -- | -- |
| $X \times W$ | -3.158** (1.019) | -5.184, -1.132 | -- | -- |
| BMI (V) | -- | -- | -0.005 (0.084) | -0.173, 0.162 |
| $M \times V$ | -- | -- | 0.104 (0.163) | -0.221, 0.428 |
| Constant | 0.272 (0.591) | -0.904, 1.447 | -0.725 (3.997) | -8.675, 7.226 |
| | | $R^2 = 0.178,$ $F(8, 84) = 2.832^{**}$ | $R^2 = 0.454,$ $F(9, 83) = 11.171^{***}$ | |

| Antecedent | Consequent | | | |
|---------------------------|-----------------------------|--------------------------------------|---|---------------------|
| | Model F: O2 alpha (M_1) | | Participant consumption (Y) | |
| | Coeff. (SE) | 95% CI | Coeff. (SE) | 95% CI |
| Model consumption (X) | 0.181* (0.087) | 0.007, 0.354 | 1.090* (0.426) | 0.242, 1.938 |
| Alpha suppression (M) | -- | -- | -2.728 (3.631) | -9.951, 4.495 |
| Frontal asymmetry (W) | 1.422 (1.072) | -0.710, 3.555 | -- | -- |
| $X \times W$ | -2.947** (1.107) | -5.149, -0.746 | -- | -- |
| BMI (V) | -- | -- | -0.001 (0.080) | -0.160, 0.159 |
| $M \times V$ | -- | -- | 0.096 (0.152) | -0.207, 0.399 |
| Constant | 0.316 (0.578) | -0.834, 1.466 | -0.842 (3.879) | -8.556, 6.873 |
| | | $R^2 = 0.194,$ $F(8, 84) = 2.094$ | $R^2 = 0.448,$ $F(9, 83) = 11.618^{***}$ | |

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

⁺ $p = .06$, ⁺⁺ $p = .07$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 9

Indices of Dual Moderated Mediation, Standard Errors, and Bias-Corrected Bootstrap 95% Confidence Intervals of PROCESS Model 21 Testing 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 |
| F3 | -0.636 (0.411) | -1.642, -0.023 |
| F4 | -0.595 (0.371) | -1.468, 0.017 |
| 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.

Table 10

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 /Alpha Suppression (M) at the 10th Percentile of Frontal Asymmetry (W) and Levels of BMI (V)

| FA (W) | BMI (V) | Model A: C3 Mu Suppression (M _I) | | Model B: C4 Mu Suppression (M _I) | | Model C: F3 Alpha Suppression (M _I) | |
|--------|---------|--|-----------------------|--|-----------------------|--|-----------------------|
| | | b (SE) | 95% CI | 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 | -0.605 (0.342) | -1.439, -0.093 |
| | 21.419 | -0.504 (0.286) | -1.241, -0.083 | -0.436 (0.279) | -1.139, -0.027 | -0.502 (0.279) | -1.200, -0.085 |
| | 23.724 | -0.297 (0.212) | -0.882, 0.001 | -0.298 (0.219) | -0.853, 0.030 | -0.326 (0.202) | -0.878, -0.032 |
| | 26.456 | -0.051 (0.206) | -0.499, 0.347 | -0.135 (0.206) | -0.603, 0.238 | -0.118 (0.212) | -0.593, 0.262 |
| | 29.778 | 0.247 (0.314) | -0.184, 1.088 | 0.063 (0.282) | -0.438, 0.688 | 0.136 (0.346) | -0.352, 1.035 |
| FA (W) | BMI (V) | Model D: F4 Alpha Suppression (M _I) | | Model E: O1 Alpha Suppression (M _I) | | Model F: O2 Alpha Suppression (M _I) | |
| | | b (SE) | 95% CI | b (SE) | 95% CI | b (SE) | 95% CI |
| -0.103 | 20.075 | -0.606 (0.333) | -1.410, -0.097 | -0.553 (0.452) | -1.597, 0.175 | -0.388 (0.378) | -1.262, 0.225 |
| | 21.419 | -0.511 (0.279) | -1.192, -0.087 | -0.477 (0.361) | -1.319, 0.106 | -0.326 (0.301) | -1.042, 0.159 |
| | 23.724 | -0.348 (0.209) | -0.918, -0.047 | -0.349 (0.246) | -0.943, 0.062 | -0.219 (0.210) | -0.721, 0.136 |
| | 26.456 | -0.155 (0.201) | -0.629, 0.186 | -0.196 (0.269) | -0.786, 0.291 | -0.092 (0.249) | -0.591, 0.384 |
| | 29.778 | -0.081 (0.302) | -0.394, 0.819 | -0.010 (0.474) | -0.993, 0.909 | -0.038 (0.435) | -0.719, 0.975 |

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

Table 11

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 /Alpha Suppression (M) at the 25th Percentile of Frontal Asymmetry (W) and Levels of BMI (V)

| FA (W) | BMI (V) | Model A: C3 Mu Suppression (M _I) | | Model B: C4 Mu Suppression (M _I) | | Model C: F3 Alpha Suppression (M _I) | |
|--------|---------|--|---------------|--|---------------|--|---------------|
| | | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| -0.033 | 20.075 | -0.273 (0.216) | -0.862, 0.011 | -0.249 (0.199) | -0.809, 0.001 | -0.251 (0.209) | -0.823, 0.018 |
| | 21.419 | -0.220 (0.177) | -0.718, 0.007 | -0.210 (0.169) | -0.689, 0.000 | -0.208 (0.170) | -0.686, 0.012 |
| | 23.724 | -0.130 (0.121) | -0.506, 0.009 | -0.144 (0.126) | -0.520, 0.013 | -0.135 (0.114) | -0.487, 0.005 |
| | 26.456 | -0.023 (0.099) | -0.286, 0.139 | -0.065 (0.107) | -0.359, 0.100 | -0.049 (0.098) | -0.322, 0.091 |
| | 29.778 | 0.108 (0.162) | -0.066, 0.614 | 0.031 (0.146) | -0.205, 0.398 | 0.056 (0.169) | -0.125, 0.577 |
| FA (W) | BMI (V) | Model D: F4 Alpha Suppression (M _I) | | Model E: O1 Alpha Suppression (M _I) | | Model F: O2 Alpha Suppression (M _I) | |
| | | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| -0.033 | 20.075 | -0.248 (0.198) | -0.770, 0.015 | -0.325 (0.288) | -1.027, 0.093 | -0.222 (0.234) | -0.793, 0.124 |
| | 21.419 | -0.209 (0.164) | -0.640, 0.011 | -0.281 (0.231) | -0.852, 0.053 | -0.186 (0.187) | -0.664, 0.084 |
| | 23.724 | -0.142 (0.115) | -0.483, 0.003 | -0.205 (0.157) | -0.611, 0.033 | -0.125 (0.129) | -0.467, 0.070 |
| | 26.456 | -0.053 (0.093) | -0.340, 0.061 | -0.115 (0.164) | -0.503, 0.166 | -0.052 (0.145) | -0.354, 0.220 |
| | 29.778 | 0.033 (0.142) | -0.148, 0.452 | -0.006 (0.287) | -0.604, 0.551 | 0.036 (0.255) | -0.408, 0.590 |

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

Table 12

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 /Alpha Suppression (M) at the 50th Percentile of Frontal Asymmetry (W) and Levels of BMI (V)

| FA (W) | BMI (V) | Model A: C3 Mu Suppression (M _I) | | Model B: C4 Mu Suppression (M _I) | | Model C: F3 Alpha Suppression (M _I) | |
|--------|---------|--|---------------|--|---------------|--|---------------|
| | | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| 0.023 | 20.075 | 0.003 (0.160) | -0.365, 0.287 | -0.038 (0.131) | -0.412, 0.147 | 0.027 (0.152) | -0.304, 0.316 |
| | 21.419 | 0.002 (0.130) | -0.295, 0.238 | -0.032 (0.110) | -0.352, 0.123 | 0.023 (0.125) | -0.242, 0.268 |
| | 23.724 | 0.001 (0.081) | -0.174, 0.168 | -0.022 (0.077) | -0.271, 0.079 | 0.015 (0.081) | -0.132, 0.208 |
| | 26.456 | 0.000 (0.047) | -0.103, 0.101 | -0.010 (0.049) | -0.191, 0.048 | 0.005 (0.049) | -0.068, 0.146 |
| | 29.778 | -0.001 (0.091) | -1.223, 0.176 | 0.005 (0.062) | -0.085, 0.184 | -0.006 (0.083) | -0.290, 0.096 |
| FA (W) | BMI (V) | Model D: F4 Alpha Suppression (M _I) | | Model E: O1 Alpha Suppression (M _I) | | Model F: O2 Alpha Suppression (M _I) | |
| | | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| 0.023 | 20.075 | 0.034 (0.141) | -0.239, 0.329 | -0.147 (0.177) | -0.672, 0.040 | -0.091 (0.140) | -0.524, 0.051 |
| | 21.419 | 0.029 (0.118) | -0.196, 0.286 | -0.127 (0.144) | -0.560, 0.027 | -0.077 (0.113) | -0.439, 0.038 |
| | 23.724 | 0.019 (0.081) | -0.116, 0.225 | -0.092 (0.098) | -0.405, 0.016 | -0.052 (0.076) | -0.323, 0.027 |
| | 26.456 | 0.009 (0.049) | -0.055, 0.165 | -0.052 (0.088) | -0.330, 0.063 | -0.022 (0.073) | -0.250, 0.073 |
| | 29.778 | -0.005 (0.064) | -0.246, 0.073 | -0.003 (0.149) | -0.353, 0.280 | 0.015 (0.127) | -0.190, 0.329 |

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

Table 13

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 /Alpha Suppression (M) at the 75th Percentile of Frontal Asymmetry (W) and Levels of BMI (V)

| FA (W) | BMI (V) | Model A: C3 Mu Suppression (M_I) | | Model B: C4 Mu Suppression (M_I) | | Model C: F3 Alpha Suppression (M_I) | |
|--------|---------|--|---------------------|--|---------------|--|---------------------|
| | | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| 0.090 | 20.075 | 0.322 (0.198) | 0.029, 0.847 | 0.205 (0.154) | -0.008, 0.635 | 0.349 (0.199) | 0.037, 0.840 |
| | 21.419 | 0.260 (0.164) | 0.024, 0.715 | 0.173 (0.132) | -0.008, 0.547 | 0.290 (0.167) | 0.035, 0.708 |
| | 23.724 | 0.153 (0.120) | -0.001, 0.538 | 0.118 (0.104) | -0.014, 0.438 | 0.188 (0.128) | 0.009, 0.562 |
| | 26.456 | 0.027 (0.114) | -0.159, 0.324 | 0.054 (0.092) | -0.063, 0.352 | 0.068 (0.128) | -0.118, 0.411 |
| | 29.778 | -0.127 (0.175) | -0.723, 0.074 | -0.025 (0.120) | -0.349, 0.175 | -0.079 (0.191) | -0.614, 0.211 |
| FA (W) | BMI (V) | Model D: F4 Alpha Suppression (M_I) | | Model E: O1 Alpha Suppression (M_I) | | Model F: O2 Alpha Suppression (M_I) | |
| | | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| 0.090 | 20.075 | 0.360 (0.195) | 0.053, 0.851 | 0.060 (0.144) | -0.143, 0.467 | 0.060 (0.127) | -0.102, 0.434 |
| | 21.419 | 0.303 (0.167) | 0.047, 0.740 | 0.052 (0.120) | -0.122, 0.386 | 0.050 (0.103) | -0.082, 0.344 |
| | 23.724 | 0.206 (0.132) | 0.021, 0.582 | 0.038 (0.085) | -0.076, 0.292 | 0.034 (0.072) | -0.047, 0.273 |
| | 26.456 | 0.092 (0.125) | -0.081, 0.426 | 0.021 (0.067) | -0.045, 0.286 | 0.014 (0.068) | -0.059, 0.261 |
| | 29.778 | -0.050 (0.171) | -0.462, 0.245 | 0.001 (0.098) | -0.185, 0.237 | -0.010 (0.114) | -0.345, 0.165 |

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

Table 14

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 /Alpha Suppression (M) at the 90th Percentile of Frontal Asymmetry (W) and Levels of BMI (V)

| FA (W) | BMI (V) | Model A: C3 Mu Suppression (M_I) | | Model B: C4 Mu Suppression (M_I) | | Model C: F3 Alpha Suppression (M_I) | |
|--------|---------|--|---------------------|--|---------------------|--|---------------------|
| | | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| 0.160 | 20.075 | 0.690 (0.324) | 0.193, 1.536 | 0.485 (0.273) | 0.069, 1.186 | 0.721 (0.335) | 0.162, 1.507 |
| | 21.419 | 0.557 (0.274) | 0.143, 1.294 | 0.410 (0.238) | 0.047, 1.032 | 0.599 (0.281) | 0.138, 1.276 |
| | 23.724 | 0.328 (0.216) | 0.024, 0.939 | 0.281 (0.195) | -0.009, 0.796 | 0.389 (0.225) | 0.050, 0.991 |
| | 26.456 | 0.057 (0.227) | -0.378, 0.569 | 0.127 (0.194) | -0.196, 0.595 | 0.140 (0.249) | -0.289, 0.716 |
| | 29.778 | -0.273 (0.336) | -1.241, 0.197 | -0.060 (0.260) | -0.624, 0.441 | -0.162 (0.378) | -1.089, 0.462 |
| FA (W) | BMI (V) | Model D: F4 Alpha Suppression (M_I) | | Model E: O1 Alpha Suppression (M_I) | | Model F: O2 Alpha Suppression (M_I) | |
| | | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI | <i>b</i> (SE) | 95% CI |
| 0.160 | 20.075 | 0.736 (0.337) | 0.167, 1.518 | 0.299 (0.271) | -0.067, 1.029 | 0.234 (0.248) | -0.097, 0.920 |
| | 21.419 | 0.620 (0.289) | 0.148, 1.321 | 0.258 (0.220) | -0.034, 0.874 | 0.197 (0.199) | -0.062, 0.755 |
| | 23.724 | 0.422 (0.234) | 0.063, 1.024 | 0.188 (0.156) | -0.016, 0.664 | 0.132 (0.142) | -0.048, 0.570 |
| | 26.456 | 0.188 (0.241) | -0.209, 0.763 | 0.106 (0.160) | -0.101, 0.615 | 0.056 (0.166) | -0.167, 0.550 |
| | 29.778 | -0.098 (0.341) | -0.851, 0.530 | 0.005 (0.267) | -0.475, 0.630 | -0.038 (0.283) | -0.711, 0.499 |

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

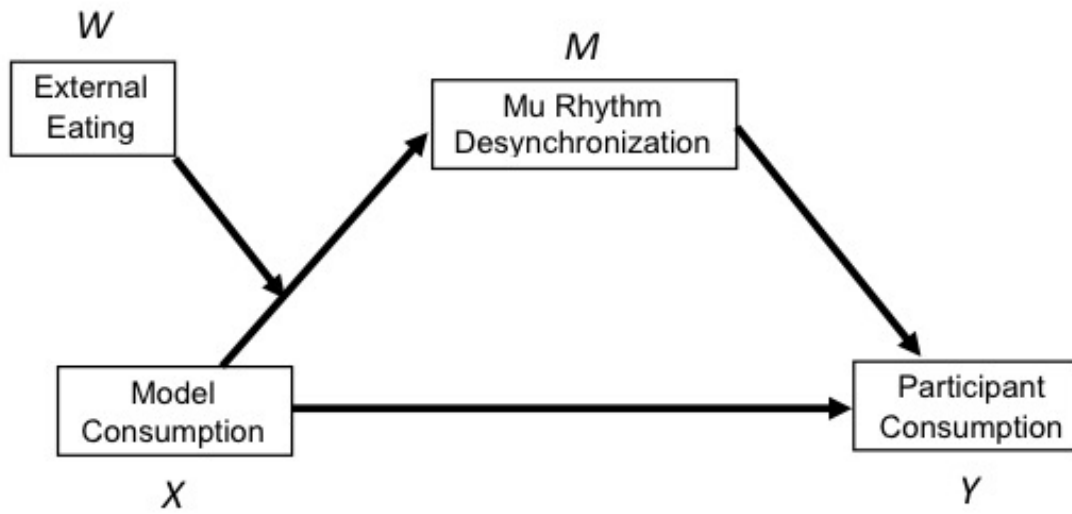


Figure 1. Conceptual model in which the effect of model consumption on participant consumption via mu rhythm desynchronization is moderated by external eating.

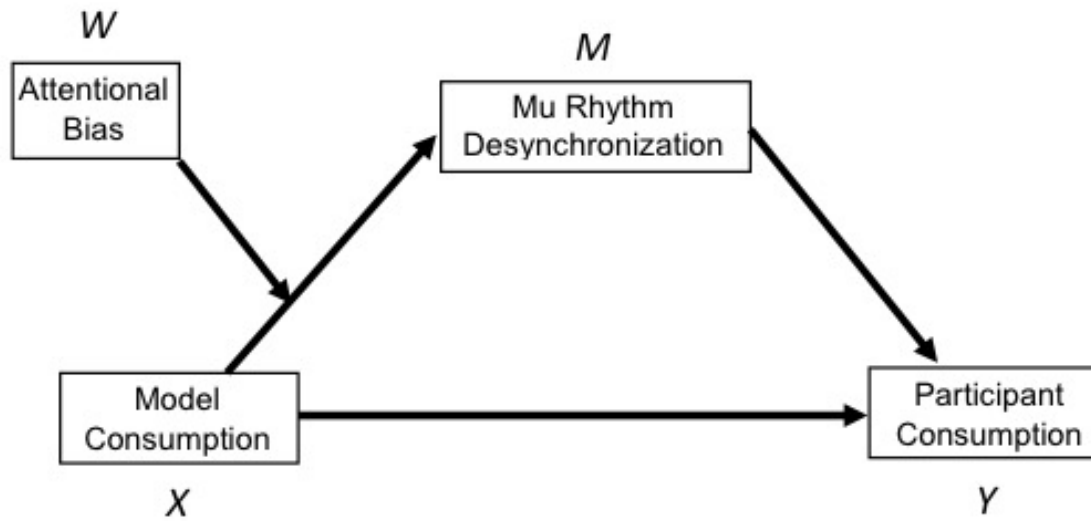


Figure 2. Conceptual model in which the effect of model consumption on participant consumption via mu rhythm desynchronization is moderated by attentional bias.

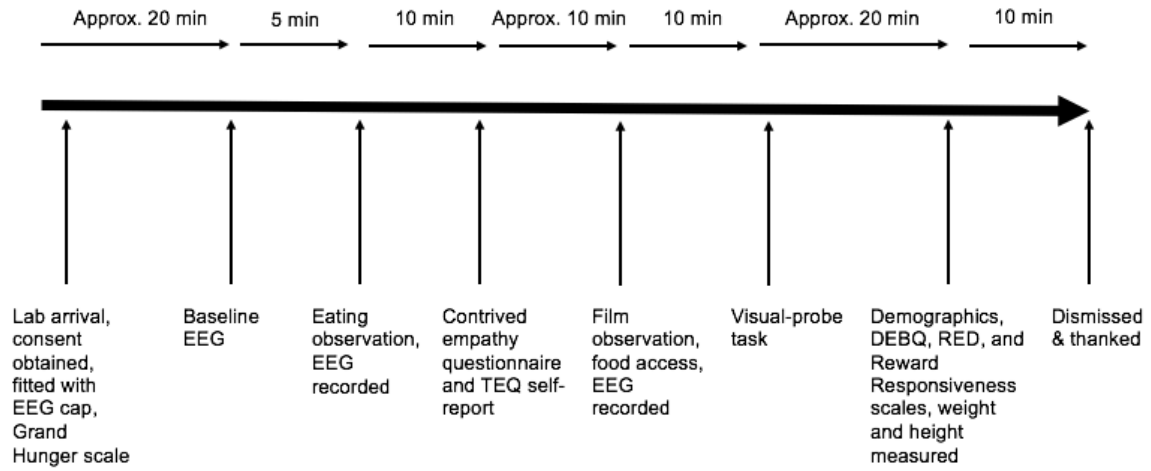


Figure 3. Timeline of activities during laboratory visit.

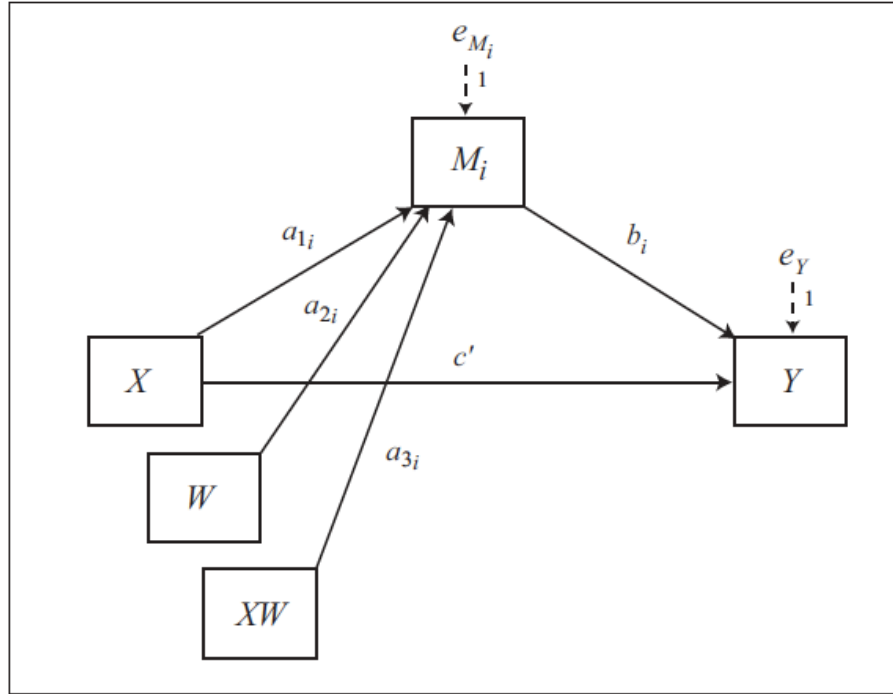


Figure 4. Statistical diagram of the moderated mediation model (Hayes, 2013) underlying the conceptual models of hypotheses one and three as presented in Figures 1 and 2.

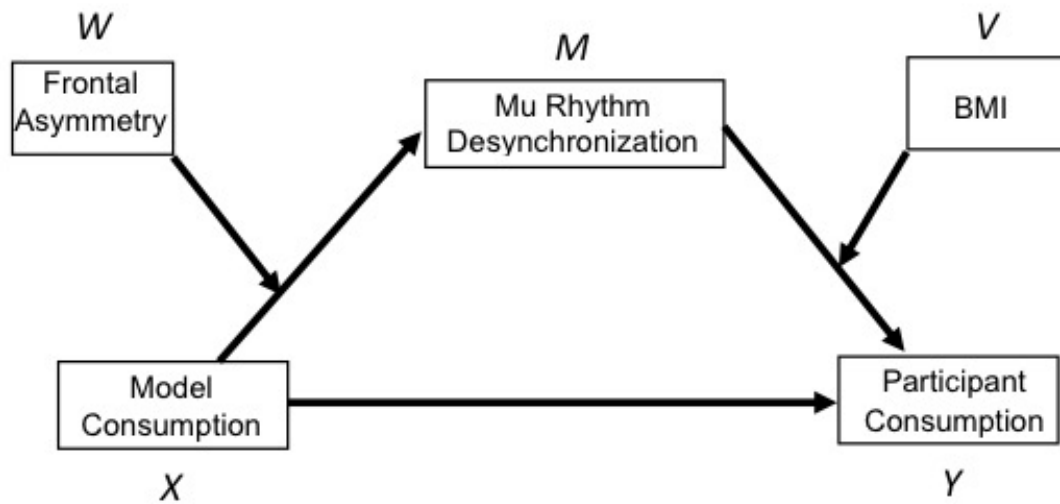


Figure 5. Conceptual model in which the effect of model consumption on participant consumption via mu rhythm desynchronization is hypothetically moderated by frontal asymmetry in the first stage of the model and BMI in the second stage.

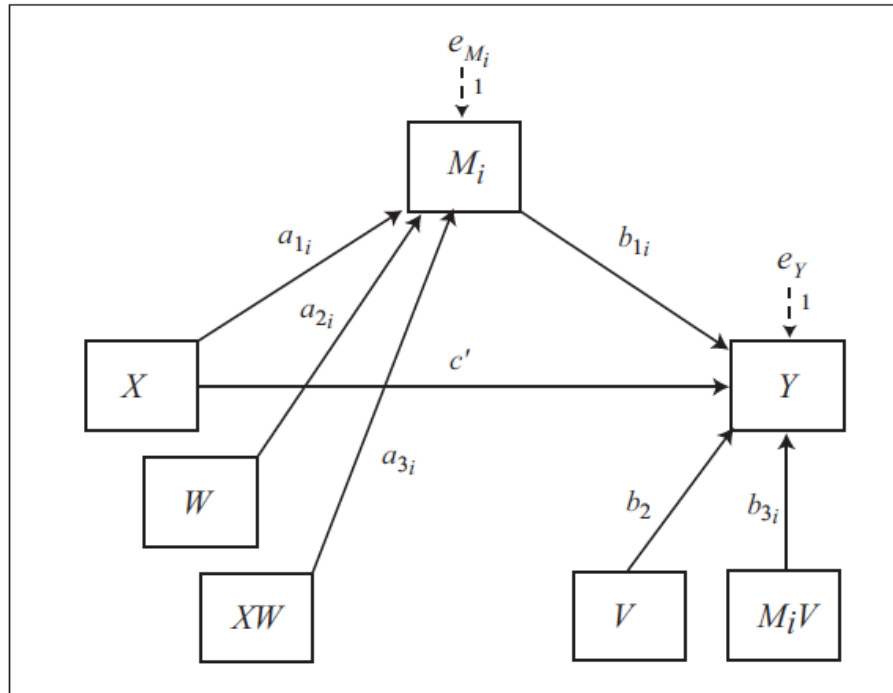


Figure 6. Statistical diagram of the first- and second-stage dual moderated mediation model (Hayes, 2013) underlying the exploratory analysis.

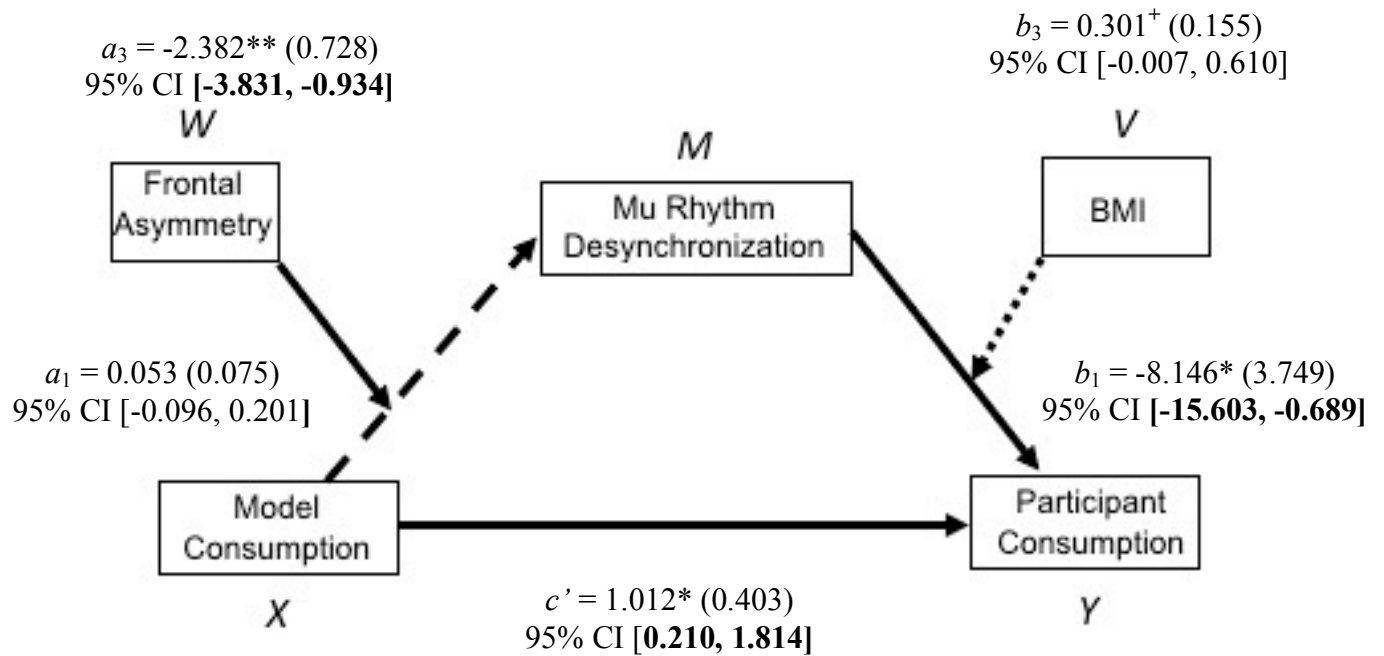


Figure 7. Unstandardized regression coefficients (SE) and 95% CIs for the simple effects and interaction terms in the dual moderated mediation of participant consumption (Y) from model consumption (X) through mu rhythm desynchronization (M) at differing levels of frontal asymmetry (W) and BMI (V) at site C3. Broken lines depict nonsignificant effects. Dotted lines depict trending effects.
⁺ $p < .06$, * $p < .05$, ** $p < .01$.



Appendix A Sona Study Description

Study Name: Social Processes and the Brain

Description:

You are invited to participate in a research study being conducted by Laura McGeown under the supervision of Dr. Ron Davis in the Department of Psychology at Lakehead University. If you are a non-smoking, right-handed female at the Thunder Bay campus, you are eligible to participate in this study that examines the changes in brain and visual activity that occur when a person is making judgments about particular aspects of another person's character. If you choose to enroll, you can now sign up to attend a laboratory session over the next week(s) in the Department of Psychology that will take approximately 90 minutes of your time. You must refrain from exercising, drinking caffeine, or eating 2 hours prior to the laboratory session and you must not drink alcohol 12 hours prior, as this may interfere with the electroencephalogram (EEG) and electrooculogram (EOG) recordings that will be used to assess your electrophysiological activity. Please also ensure that you are not currently taking any cold or hypertensive medication for the same reason. The EEG recording will require that you wear a tight-fitting cap. Be aware that the application of the EEG cap requires a small amount of conductive gel be applied to your scalp. This gel can be easily removed from the hair.

Upon arrival to the lab, you will be fitted with an EEG cap and you will place a ground electrode on your chest. Two electrodes will be placed around your right eye, one above and below, to record your visual activity for the EOG recording. Your electrophysiological activity will be recorded while watching a 10-minute video of another female student. You will be asked to silently assess how empathic you believe she is based on facial cues as she watches a film. Once the video has completed, you will provide your written rating of her and complete a questionnaire about your own level of empathy. You will then be asked to engage in an unrelated reaction time task to assess the speed with which your typical judgments are made in response to pictures of common objects, people, and food. Once this task is completed, you will be asked to fill out demographic and additional questionnaires, and your height and weight will also be measured.

Your participation in the study is completely voluntary and you may withdraw at any time, with no penalty. All of the information provided will be kept completely confidential. Only myself, research assistant Dasuel Shin, and Dr. Ron Davis will be permitted to view your information. However, Dr. Davis will not be aware of the identities of any students who volunteer to participate in this study. The information you provide will be assigned a code that is unattached to your name and any identifying information provided will be kept confidential in any reports or publications of the results that arise. All of the information provided will be securely stored on a password protected computer located in a double-locked research office in the Department of Psychology at Lakehead University Thunder Bay Campus for 5 years, as per University regulations.



A risk associated with your participation in this study is the possibility that the act of completing our personal questionnaires may cause an emotional reaction depending upon how you think about yourself in light of the questions you are answering. You may choose not to answer any question asked in the questionnaires without penalty or consequences. If at any point during or after this study you would like to speak to a mental health professional, feel free to contact the Student Health and Counseling Centre located in the Prettie Residence in person or by telephone at 807-343-8361.

If you are registered in a Psychology undergraduate course eligible for bonus points, your participation by way of attending the laboratory visit would lead to 2 bonus points credited to your final grade in that course. Please feel free to contact Laura McGeown at lmcgeown@lakeheadu.ca or Dr. Ron Davis at ron.davis@lakeheadu.ca if you have any questions regarding your participation in this study. This study has been approved by the Lakehead University Research Ethics Board. If you have any questions related to the ethics of the research and would like to speak to someone outside of the research team, please contact Sue Wright at the Research Ethics Board at 807-343-8283 or research@lakeheadu.ca

Sincerely,

Laura McGeown lmcgeown@lakeheadu.ca
Dr. Ron Davis ron.davis@lakeheadu.ca (807) 343-8646



Appendix B

Participant Information Letter

Dear Potential Participant:

You have signed up to participate in a research study being conducted by Laura McGeown, a Masters student under the supervision of Dr. Ron Davis in the Department of Psychology at Lakehead University. This study is interested in examining the changes in brain and visual activity that occur while you will be making judgments about particular aspects of another's character. This laboratory session will take approximately 90 minutes of your time.

Once you have provided your consent on the consent form that follows, you will be fitted with an EEG cap to record brain activity, and electrodes will be placed above and below your eye for electrooculogram (EOG) visual activity. This will record your electrophysiological activity while you watch a video of a female student and silently assess how empathic you believe she is. After a baseline EEG and EOG recording is taken, the video will begin and you will be asked to remain as still as possible while you watch, as any movement may interfere with the electrophysiological recordings. You will provide your written rating and complete a questionnaire about your own level of empathy once the video has completed. You will then engage in an unrelated reaction time task to assess the speed with which typical judgments are made in response to pictures of common objects, people, and food. Once this task is completed, you will be asked to fill out demographic and additional questionnaires, and your height and weight will also be measured. You will then be debriefed, thanked, and dismissed from the lab.

Your participation in this study is completely voluntary and you may withdraw your consent at any time without penalty. All of the information provided will be kept completely confidential by assigning a code to your data that is unattached to your name. Only myself, research assistant Dasuel Shin, and Dr. Ron Davis will be permitted to view your information. A potential conflict of interest may arise for participants enrolled in a class taught by Dr. Davis who may feel pressure to participate. However, Dr. Davis will not be aware of the identities of any students who volunteer to participate in this study and he will not see any information that could be used to identify participants. Any identifying information provided will also be kept confidential in reports of the results and any publications that result. All of the information provided will be securely stored on a password protected computer located in a double-locked research office in the Department of Psychology at Lakehead University Thunder Bay Campus for 5 years, as per University regulations.

This study has the potential to benefit you from the learning experience of participating in psychological research. A risk associated with your participation in this study is the possibility that the act of completing our personal questionnaires may cause an emotional reaction depending upon how you think about yourself in light of the questions you are answering. You may choose not to answer any question asked in the questionnaires without penalty or consequences. If at any point during or after this study you would like



to speak to a mental health professional, feel free to contact the Student Health and Counseling Centre located in the Prettie Residence in person or by telephone at 807-343-8361.

If you are registered in a Psychology undergraduate course eligible for bonus points, your participation by way of attending the laboratory visit would lead to 2 bonus points credited to your final grade in that course. Please feel free to contact Laura McGeown and/or Dr. Ron Davis with any questions you may have. If you wish to receive a summary of the findings of this study, you will have the opportunity to indicate your interest to do so during the laboratory session, as well as during a final debriefing email that will be sent once all participants have participated. You will be asked to provide an email address to indicate where the results should be sent. The results will be sent at the conclusion of the study once all participants have completed their participation and all analyses have been conducted. You will not be identified directly or indirectly through this process.

This study has been approved by Lakehead University Research Ethics Board. If you have any questions related to the ethics of this study and would like to speak to someone outside of the research team, please contact Sue Wright at the Research Ethics Board at 807-343-8283 or research@lakeheadu.ca.

Sincerely,

Laura McGeown lmcgeown@lakeheadu.ca
Dr. Ron Davis ron.davis@lakeheadu.ca (807) 343-8646



Appendix C Participant Consent Form

By providing my name and signature below, I indicate that I have read the “Participant Information Letter” and that I have had the opportunity to receive satisfactory answers from the researchers concerning any questions that I might have about my participation in the **Social Processes and the Brain**. I understand and agree to the following:

1. I understand all of the information on the “Participant Information Letter”;
2. I agree to participate in this study;
3. I am a volunteer and can withdraw at any time from this study without penalty or consequence;
4. I may choose not to answer any question asked in the questionnaires without penalty or consequence;
5. There are no anticipated physical risks associated with participation in this study. Should I experience any personal distress or discomfort during or following my participation, I know that I may personally contact the Health and Counselling Centre at Lakehead University (Thunder Bay campus) to speak to a mental health professional;
6. My personal information will be securely stored in the Department of Psychology at Lakehead University for 5 years as per University regulations;
7. Dr. Ron Davis is never aware of the identities of those who volunteer to participate in this study;
8. My personal information will remain anonymous should any publications or public presentations come out of this project;
9. I may receive a summary of this research upon completion if I so request;
10. I give my permission to be contacted by telephone and/or email for the purpose of participation in this study; and
11. I understand and agree to this “Consent to Participate”

Full Name (*please print*)

Date

Signature (*please sign*)

Appendix D

Laboratory Cover Story Script

The mirror neuron system is a group of neurons in the brain which fire both when individuals perform an action and when observing actions performed by others. This system has been proposed to underlie the development of imitation and behavioural mimicry, as well as empathy. It has been found that individuals with higher levels of empathy exhibit greater activity within the mirror neuron system. Empathy is an important part of social cognition that helps us understand and respond adaptively to the emotions of others by accurately assessing how another individual is feeling. One cue that can be used to infer the emotions of others is the emotional facial cues they express. Empathic individuals are more likely to mimic the behaviour of others to ease social interaction and assist in emotional communication. To judge the level of empathy that another individual possesses then, one may assess the nature of the emotional reactions they display. The current study is interested in examining whether the mirror neuron system in the brain is active during a task in which one assesses the empathic quality of another individual. In line with previous literature, it is expected that self-reported empathy will be related to one's mirror neuron activity during an empathic processing task such as the one you are about to engage in our lab.

Appendix E
Grand Hunger Scales & Additional Ratings

Please provide an estimate (to the nearest 15 minutes) of the amount of time that has passed since you last ate:

How hungry do you currently feel? Please indicate on the following scale by circling the number in the box that best matches your current level of hunger:

| | | | | | | |
|----------------------|---|---|---|---|---|---------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| Not hungry at all | | | | | | Extremely hungry |

How much of your favourite food would you be able to eat at the present time?

| | | | | | |
|-------------|---|---|---|---|---------------------------|
| 1 | 2 | 3 | 4 | 5 | 6 |
| None at all | | | | | As much as I could get |

Please provide an estimate (to the nearest 15 minutes) of the amount of time until your next expected meal:

How thirsty do you currently feel? Please indicate on the following scale by circling the number in the box that best matches your current level of thirst:

| | | | | | | |
|-----------------------|---|---|---|---|---|----------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| Not thirsty at all | | | | | | Extremely thirsty |

How tired do you currently feel? Please indicate on the following scale by circling the number in the box that best matches your current level of fatigue:

| | | | | | | |
|---------------------|---|---|---|---|---|--------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| Not tired at all | | | | | | Extremely tired |

How much discomfort do you currently feel? Please indicate on the following scale by circling the number in the box that best matches your current level of discomfort:

| | | | | | | |
|----------------|---|---|---|---|---|-----------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
| None at all | | | | | | Extreme discomfort |

Appendix F
Contrived “Empathy Rating” Scale

Below is a list of statements about the person you just finished watching. Please read each statement carefully and circle your answer on the scale below each item.

1) The emotions she expressed seemed genuine.

| | | | | | | | | |
|----------------------|---|---|---|----------------------------------|---|---|---|-------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

2) She seemed emotionally detached while watching the video.

| | | | | | | | | |
|----------------------|---|---|---|----------------------------------|---|---|---|-------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

3) She expressed emotion with great ease.

| | | | | | | | | |
|----------------------|---|---|---|----------------------------------|---|---|---|-------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

4) She seemed caught up in the emotions expressed in the film.

| | | | | | | | | |
|----------------------|---|---|---|----------------------------------|---|---|---|-------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

5) She appeared to be accurately perceiving the emotional tone of the film.

| | | | | | | | | |
|----------------------|---|---|---|----------------------------------|---|---|---|-------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

6) She displayed more positive emotions towards the film overall.

| | | | | | | | | |
|----------------------|---|---|---|----------------------------------|---|---|---|-------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

7) There were some periods in which the emotions she was expressing in the video were unclear.

| | | | | | | | | |
|-------------------|---|---|---|----------------------------|---|---|---|----------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

8) She seemed like a likeable person.

| | | | | | | | | |
|-------------------|---|---|---|----------------------------|---|---|---|----------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

9) She appeared to be more engaged in thinking rather than feeling.

| | | | | | | | | |
|-------------------|---|---|---|----------------------------|---|---|---|----------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

10) She seemed to be emotionally invested in the film.

| | | | | | | | | |
|-------------------|---|---|---|----------------------------|---|---|---|----------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

11) She seemed to be in tune with her own emotions.

| | | | | | | | | |
|-------------------|---|---|---|----------------------------|---|---|---|----------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

12) She did not seem to be following the emotions expressed by the characters in the film.

| | | | | | | | | |
|-------------------|---|---|---|----------------------------|---|---|---|----------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

13) I would be likely to become friends with someone like her.

| | | | | | | | | |
|----------------------|---|---|---|----------------------------------|---|---|---|-------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

14) She appeared distracted while watching the film.

| | | | | | | | | |
|----------------------|---|---|---|----------------------------------|---|---|---|-------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

15) Please rate how pleasant or unpleasant her subjective responses were overall.

| | | | | | | | | |
|--------------------|---|---|---|--------------------------------------|---|---|---|------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Very unpleasant | | | | Neither pleasant or unpleasant | | | | Very pleasant |

16) She seemed similar to me in some ways.

| | | | | | | | | |
|----------------------|---|---|---|----------------------------------|---|---|---|-------------------|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| Strongly disagree | | | | Neither agree nor disagree | | | | Strongly agree |

17) Based on her general emotional responses, I believe the genre of film being watched is likely a:

_____ Comedy

_____ Drama

_____ Romantic Comedy

_____ Thriller

_____ Horror Movie

Appendix G
Toronto Empathy Questionnaire

Below is a list of statements. Please read each statement carefully and rate how frequently you feel or act in the manner described. Circle your answer on the response form. There are no right or wrong answers or trick questions. Please answer each question as honestly as you can.

1. When someone else is feeling excited, I tend to get excited too

Never Rarely Sometimes Often Always

2. Other people's misfortunes do not disturb me a great deal

Never Rarely Sometimes Often Always

3. It upsets me to see someone being treated disrespectfully

Never Rarely Sometimes Often Always

4. I remain unaffected when someone close to me is happy

Never Rarely Sometimes Often Always

5. I enjoy making other people feel better

Never Rarely Sometimes Often Always

6. I have tender, concerned feelings for people less fortunate than me

Never Rarely Sometimes Often Always

7. When a friend starts to talk about his/her problems, I try to steer the conversation towards something else

Never Rarely Sometimes Often Always

8. I can tell when others are sad even when they do not say anything

- | | Never | Rarely | Sometimes | Often | Always |
|--|-------|--------|-----------|-------|--------|
| 9. I find that I am “in tune” with other people’s moods | | | | | |
| | Never | Rarely | Sometimes | Often | Always |
| 10. I do not feel sympathy for people who cause their own serious illnesses | | | | | |
| | Never | Rarely | Sometimes | Often | Always |
| 11. I become irritated when someone cries | | | | | |
| | Never | Rarely | Sometimes | Often | Always |
| 12. I am not really interested in how other people feel | | | | | |
| | Never | Rarely | Sometimes | Often | Always |
| 13. I get a strong urge to help when I see someone who is upset | | | | | |
| | Never | Rarely | Sometimes | Often | Always |
| 14. When I see someone being treated unfairly, I do not feel very much pity for them | | | | | |
| | Never | Rarely | Sometimes | Often | Always |
| 15. I find it silly for people to cry out of happiness | | | | | |
| | Never | Rarely | Sometimes | Often | Always |
| 16. When I see someone being taken advantage of, I feel kind of protective towards him/her | | | | | |
| | Never | Rarely | Sometimes | Often | Always |

**Appendix I
Demographics Questionnaire**

Age: _____ **Height:** _____ **Weight:** _____

Marital Status (please circle one below):

Married/Common law Divorced/Separated Single Widowed

Ethnicity (please check one or indicate below)

_____ Caucasian/White

_____ Aboriginal/First Nation

_____ South Asian

_____ Hispanic

_____ African-Canadian/Black

_____ East Asian

_____ Middle Eastern

_____ Other (please identify): _____

School Enrolment (please circle one)

Full time

Part time

Not enrolled

What subject are you majoring in? _____

Please list the name(s) of prescribed medication(s) and/or over-the-counter medications you are currently taking:

Please indicate if you have any dietary restriction (i.e. vegan, vegetarian, food allergies/intolerances):

Please check the box below if you would like to receive a summary of the findings upon completion of the study and provide an email where they can be sent to.

Appendix J
Edinburgh Handedness Inventory – Short Form (EHI – Short Form)

Please indicate your preference in the use of hands in the following activities or objects:

0 = Always Right; 1 = Usually Right; 3 = Both Equally; 4 = Usually Left; 5 = Always Left

| | | | | | |
|---------------|---|---|---|---|---|
| 1. Writing | 0 | 1 | 2 | 3 | 4 |
| 2. Throwing | 0 | 1 | 2 | 3 | 4 |
| 3. Toothbrush | 0 | 1 | 2 | 3 | 4 |
| 4. Spoon | 0 | 1 | 2 | 3 | 4 |

Appendix K

Dutch Eating Behavior Questionnaire (DEBQ)

Items are responded to on a 5-point Likert scale response format, where 1 (*never*), 2 (*seldom*), 3 (*sometimes*), 4 (*often*), and 5 (*very often*).

Restrained Eating items

1. If you have put on weight, do you eat less than you usually do?
2. Do you try to eat less at meal times than you would like to eat?
3. How often do you refuse food or drink offered because you are concerned about your weight?
4. Do you watch exactly what you eat?
5. Do you deliberately eat food that are slimming?
6. When you have eaten too much, do you eat less than usual the following day?
7. Do you deliberately eat less in order not to become heavier?
8. How often do you try not to eat between meals because you are watching your weight?
9. How often in the evenings do you try not to eat because you are watching your weight?
10. Do you take into account your weight with what you eat?

Emotional Eating items

11. Do you have the desire to eat when you are irritated?
12. Do you have the desire to eat when you have nothing to do?
13. Do you have the desire to eat when you are depressed or discouraged?
14. Do you have a desire to eat when you are feeling lonely?
15. Do you have a desire to eat when somebody lets you down?
16. Do you have a desire to eat when you are cross?
17. Do you have a desire to eat when you are expecting something unpleasant to happen?
18. Do you get the desire to eat when you are anxious, worried or tense?
19. Do you have a desire to eat when things are going against you or when things have gone wrong?
20. Do you have a desire to eat when you are frightened?
21. Do you have the desire to eat when you are disappointed?
22. Do you have a desire to eat when you are bored or restless?
23. Do you have a desire to eat when you are emotionally upset?

External Eating items

24. If food tastes good to you, do you eat more than usual?
25. If food smells and looks good to you, do you eat more than usual?
26. If you see or smell something delicious, do you have the desire to eat it?
27. If you have something delicious to eat, do you eat it straight away?
28. If you walk past the baker, do you have the desire to buy something delicious?
29. If you walk past a snackbar or café, do you have the desire to buy something delicious?
30. If you see others eating, do you also have the desire to eat?
31. Can you resist eating delicious foods?

32. Do you eat more than usual when you see others eating?
33. When your mother or father are preparing a meal, are you inclined to eat something?



Appendix L Participant Debriefing Email

Dear Participant:

During your participation in the study entitled, “Social Processes and the Brain,” you were informed that the purpose of the study was to examine whether the mirror neuron system in the brain is active during a task in which one assesses another individual’s level of empathy. Though the study *was* interested in examining the activation of the mirror neuron system while watching the female in the video, you will likely recall that she was also eating potato chips. The true purpose of the study was to examine activity in the mirror neuron system while watching another individual eating and to assess how this activation may be related to how much you chose to eat when provided with potato chips later in that study.

Previous literature has shown evidence of a social modeling effect on food intake, in which individuals tend to eat more when an observed individual eats more and eat less when the observed individual eats less. The current study aims to examine whether activity in the mirror neuron system may underlie this effect. Specifically, it is proposed that individuals who show greater mirror neuron activity may be more likely to adjust the amount of food that they consume to the amount that an observed individual consumes.

The true purpose of the study was not able to be revealed to you at the time you participated in the study for multiple reasons. Firstly, previous literature has indicated that individuals may alter their eating behaviour when they know that it is under scrutiny in a laboratory situation. In addition, when the predicted response under particular conditions is clearly stated, it is possible that some individuals participating in a psychological study may act in a way that is contrary to what their natural response would have been. Some individuals may feel inclined to deliberately act in a way that confirms predictions and to do what they believe the researchers want them to do. By contrast, others may deliberately act in a way that would purposefully contradict predictions. In either situation, the reactions expressed by individuals would not provide a good indication of their natural response tendency. Therefore, to properly address the research question and ensure valid results were obtained, it was necessary to provide an alternative cover story about the purpose of this study.

This study has the potential to benefit society, as the social influence on eating has very real consequences at a population level. The basic social influence process that underlies the modeling of food intake could potentially influence the adoption of dangerous dieting behaviours as well as patterns of overeating, which may lead to eating disorders or obesity. Developing a better understanding of the factors that may contribute to both of these issues may help to point towards more effective treatment options.

You are reminded that your original consent included your right to withdraw at any time from this study without penalty or consequence. If you wish, you may request the



withdrawal of your data and choose to withdraw your participation from the study. If this is your wish, please indicate so in your response to this email on or before March 13th (i.e. within 14 days of receiving this email. You are also reminded that any of the information provided during the laboratory session will not be attached to your name.

Please feel free to contact Laura McGeown and/or Dr. Ron Davis if you have any concerns or questions about your participation or the study in light of this disclosure, and we will be happy to answer any questions you may have. If you are experiencing any personal distress or discomfort as a result of this study or this disclosure of deception and you would like to speak to a mental health professional, please feel free to contact the Student Health and Counseling Centre in person located in the Prettie Residence or by telephone at 807-343-8361. If you have any questions related to the ethics of this study or your rights as a research participant and would like to speak to someone outside of the research team, please contact Sue Wright at the Research Ethics Board at 807-343-8283 or research@lakeheadu.ca.

Your participation in this study is greatly appreciated. If you would like to receive a summary of the findings and did not already indicate your interest to receive the results during your laboratory session, please indicate your interest and provide an email address that you can be reached at in your reply to this email. We hope that you found your participation in this study an interesting and valuable experience.

Sincerely,

Laura McGeown lmcgeown@lakeheadu.ca
Dr. Ron Davis ron.davis@lakeheadu.ca (807) 343-8646