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Research report

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

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ABSTRACT

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

1. Introduction

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

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

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

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

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

1.1. Frontal asymmetry and approach motivation

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

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

1.2. BMI as a second-stage moderator

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

1.3. The normative account of social modeling of eating

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

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