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Social support influences effective neural connections during food cue processing and overeating: A bottom-up pathway

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ABSTRACT

Background: Social support helps prevent the onset and progression of overeating. However, few studies have explored the neural mechanisms underlying this pathway. This study used functional magnetic resonance imaging (fMRI) and dynamic causal modeling (DCM) analysis to elucidate the general neural mechanisms and effective neural pathways linking social support to alterations in food cue processing and overeating.
Methods: This study included 58 healthy, premenopausal female participants (mean age, 20.92 years), divided into social support (SS) and non-social support (NSS) groups. Participants underwent fMRI scans while performing the Food Incentive Delay (FID) task. We investigated group differences in brain activation and effective connections, as well as correlations with food consumption.
Results: When exposed to food cues, the SS group showed increased activation in the Executive Control Network (ECN), Salience Network, and Reward Network, specifically in response to high-calorie foods in the ECN. DCM analysis demonstrated enhanced excitatory effects in the SS group, including pathways from the right caudate to the right insula, right insula to right DLPFC, and left putamen to left VMPFC, under high-calorie conditions. The effective connectivity between the caudate and insula was negatively correlated with food choices.
Conclusion: Social support modulates a bottom-up neural pathway connecting intrinsic networks related to reward sensitivity, emotional salience, and inhibitory control, which helps suppress excessive cravings and intake of high-calorie foods. This study provides the first neural evidence for a shared neural basis between social reward and food reward.

Introduction

Overeating is characterized by the consumption of food in quantities exceeding physiological hunger, often accompanied by a subjective sense of loss of control (Goldschmidt et al., 2016). Such behaviors may lead to eating disorders and obesity (Stice et al., 2009), and can result in severe mental health issues, including an increased risk of suicide (Bibbins-Domingo et al., 2007; Lamerz et al., 2005; Reilly et al., 2010). Social support is defined as the resources provided by social networks that enhance an individual's ability to cope with challenges (Cohen et al., 2004). It helps prevent the progression of eating disorders (Bertera, 2005) and mitigates unhealthy eating behaviors (Gruber, 2008; Kim et al., 2008). For instance, a seven-year longitudinal study involving 496 adolescent females found that high levels of social support can effectively prevent the future onset of overeating (Stern et al., 2023). Rat models found operant social reward prevented drug self-administration, highlighting the need for incorporating social factors into neuroscience-based addiction research (Venniro et al., 2018). However, few studies have investigated the neural mechanisms by which social support influences overeating.

According to the risk model of eating disorders, negative affect, self-control and reward sensitivity are three important internal psychological factors affecting overeating, while interpersonal environment is an important external factor affecting overeating (Tanofsky-Kraff et al., 2020). Previous evidence demonstrated that participants who received support from their friends reported lower levels of negative emotion,

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high levels of self-control, chose fewer food portions, and consumed fewer snacks during acute stress (Xiao et al., 2023). These results can be explained by subsequent studies on neural mechanisms, which found that social support modulates synergistic interactions both within and between intrinsic networks involved in inhibitory control (Executive Control Network, ECN), emotional salience (Salience Network, SN), and reward sensitivity (Reward Network, RN). And social support predicted overeating through the ECN−SN network connectivity and negative emotions, especially in young woman (Xiao et al., 2023). Therefore, the neural mechanisms by which social support reduces overeating may operate through enhancing inhibitory control and improving emotional regulation.

In recent studies, social support has also been recognized as a kind of social reward that helps overcome cravings for addictive substances, such as inhibit opioid and psychostimulant self-administration and relapse (Venniro et al., 2022, 2018). These findings can be explained by the reward substitutability hypothesis (Carver, 2004), which suggests that the abundance (or scarcity) of one survival resource reduces (or increases) the need for another survival resource (Briers et al., 2006). For example, in a taste test task, participants who experienced social isolation reported an increase in ice cream consumption (Chang et al., 2021). Similarly, research on economic rewards shows that enhanced social connections reduce the desire for monetary compensation (Lasaleta et al., 2021). Another study found that after 10 hours of social isolation and 10 hours of fasting, participants exhibited activation in the same brain regions (VTA/SN) when craving food and social connection, suggesting that food and social rewards may share common cognitive processing pathways (Tomova et al., 2020). Additionally, in recent years, Gu and colleagues (2019) proposed the "common neural currency hypothesis", which posits that anticipation of both social and monetary rewards engages shared neural circuits, including the ventral tegmental area (VTA), ventral striatum (VS), and anterior insula (AI) (Gu et al., 2019). In summary, considering the rewarding nature of social support, it is likely that social support also influences the processing of food cues and decision-making related to eating behavior through brain regions and connections associated with the reward network.

Although social support and food reward behaviorally complement each other and share, in part, cognitive processing circuits, it is not known how social support affects neural activity and effective connectivity during food reward processing. We used a Food Incentive Delay task with low-calorie and high-calorie food cues and then applied a dynamic causal modeling (DCM) analysis to the data (Friston et al., 2003) to examine the causal architecture of coupled or distributed dynamics of central brain regions of the inhibitory control, reward processing, and emotion regulation brain systems. Our study aims to test the following 3 hypotheses: First, participants who received social support exhibited heightened activation in the ECN, SN, and RN brain regions when viewing food cues, compared to those in the non-social support group. Second, social support-associated brain reactivity is correlated with decreased food choices and consumptions. Third, as a survival resource, social support could play the role of social reward, replacing food reward seeking and craving, and then affecting eating decision and behaviors.

Methods

Participants

We recruited 58 healthy female participants (mean age=20.92±1.59 years; age range, 18.43–25.21 years) from a university in the southwest region of China. Participants with metal objects in the body, neurologic or psychiatric disorders, taking medications that affect cognitive function and birth control pills for the past 3 months, irregular menstrual cycles, and reported never have once emotional eating behavior were excluded. All participants gave informed consent to the study procedures, which were approved by the University Institutional Review Board. Participant data included self-reported ethnicity, age, body mass index (BMI). Multimodal data, including functional magnetic resonance imaging (fMRI) and clinical and behavioral measures, were also collected.

Procedure

In Session 1, participants were randomly assigned to one of two groups: the social support (SS) group and the non-social support (NSS) group. All participants completed a set of baseline questionnaires (see the "Behavioral assessments" section below). Participants in the SS group were instructed to bring their best friend, explicitly defined as someone with whom they did not have a sexual relationship. Participants in the NSS group attended the Session 1 alone.

Session 2 was conducted 2–3 days after Session 1. Participants first reported their current levels of stress, positive and negative emotions, food craving, hunger, thirst, and time since their last meal. They then completed a stress induction task, after which they reported their stress levels again to assess the success of the stress manipulation. Subsequently, both groups underwent different social support manipulation tasks and the Food Incentive Delay (FID) task while undergoing fMRI scanning to capture neural activity during these tasks. After the fMRI session, participants completed a food portion choice task and a bogus tasting task, which were used to quantify overeating behavior. Finally, participants were taken to another room to complete the presentation and receive their compensation.

Behavioral assessments

Body mass index (BMI). Participants self-reported their body height (in m) and weight (in kg). Based on this self-reported information the body mass index (kg/m^2) was calculated.

Eating behaviors. The Three-Factor Eating Questionnaire (TFEQ-R18) is a scale that measures three domains of eating behavior: cognitive restraint (e.g., "I deliberately take small helpings to control my weight"), uncontrolled eating (e.g., "Sometimes when I start eating, I just can't seem to stop") and emotional eating (e.g., "I start to eat when I feel anxious") (Karlsson et al., 2000). The items are measured using a 4-point response scale (1 = definitely false, 4 = definitely true). Cronbach's coefficient α of the TFEQ in this study ranged from 0.74 to 0.87.

Trait perceived social support. The Multidimensional Scale of Perceived Social Support (MSPSS) (Wang et al., 1999) used 12 item to assesses participants' perceived social support (e.g., "when I have problems, some people [i.e., relatives, friends, classmates] were there accompanying me"). Responses were rated using a 5-point Likert-type scale (1 = never to 5 = always). Cronbach's coefficient α of the MSPSS in the current study was 0.88.

Trait/state perceived stress. Trait perceived stress level was filled in during the first experiment and measured using the Perceived Stress Questionnaire (PSQ) (S. Cohen et al., 1983), which contains a total of 14 questions (e.g., how often did you feel upset about something unexpected happening in the last month?), rated on a 5-point scale from 1 (never) to 5 (always). Cronbach's coefficient α of the PSQ in the current study was 0.87. State perceived stress was measured by three items (feeling of anxiety/worried/fearful) (Tomiyama et al., 2011), and it's Cronbach's coefficient α in this study ranged from 0.83 to 0.86.

Trait/state positive and negative emotions. The Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988) was used to measure trait and state levels of 9 positive emotions (e.g., energetic) and 11 negative emotions (e.g., distracted) with ratings ranging from 1 (almost none) to 5 (extremely many), where alertness was classified from positive emotions in the original version to negative emotions in this study because of the negative meaning in the Chinese culture. Trait positive-negative mood was measured in Session 1, where participants responded based on their emotional state in the most recent month. And the state mood level was measured in Session 2, where participants responded based on

their current mood state. Cronbach's coefficient α of the PANAS in this study ranged from 0.87 to 0.92.

Trait Self-efficacy. Self-efficacy was measured using the General Self-Efficacy Scale (GSES) (Luszczynska et al., 2005). It contains 10 questions about the individual's self-efficacy when encountering setbacks or difficulties. For example, "I can always find a solution to a problem when I encounter it". A scale of 1 (not at all correct) to 4 (completely correct) on a 4-point Likert scale was used. Cronbach's coefficient α of the GSES in the current study was 0.91.

Reward sensitivity. Reward sensitivity was measured using the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (Torrubia et al., 2001). The reward sensitivity subscale contains 24 questions (e.g., Does encouragement from friends or family enable you to perform well at work and school?), including a 0 (no) and 1 (yes) secondary rating. Cronbach's coefficient α of the questionnaire in the current study was 0.70.

Self-rating Depression. Depression symptoms were measured using the Self-rating Depression Scale (SDS) developed by Zung (Zung, 1965). The scale consists of 20 items (e.g., "I feel down-hearted and blue"), rated on a 4-point Likert scale ranging from 1 (a little of the time) to 4 (most of the time). Cronbach's coefficient α for the SDS in the current study was 0.72.

Self-rating Anxiety. Anxiety symptoms were measured using the Self-rating Anxiety Scale (SAS) developed by Zung (Zung, 1971). The scale consists of 20 items (e.g., "I feel more nervous and anxious than usual"), rated on a 4-point Likert scale ranging from 1 (a little of the time) to 4 (most of the time). Cronbach's coefficient α for the SAS in the current study was 0.79.

Experimental manipulation and measurements

Stress Manipulation. Perceived stress levels were manipulated using a speech task, a widely used and effective method for stress induction (Kwan & Gordon, 2016; Plessow et al., 2011). After completing the initial state questionnaires, participants were given five minutes to prepare a presentation on the topic of "global warming." The presentation was evaluated in real-time via video conference by two judges (one male and one female). The judges, impersonated by two of the main experimenters, were instructed to maintain expressionless faces throughout the presentation to enhance the stress-inducing effect. Note that the stress task served only as a means of inducing emotional overeating and was not a variable primarily explored in the study.

Social Support Manipulation. We manipulated the perceived support level using a social support task published in the previous literature (Morawetz et al., 2021). In Session 1, friends of the participants in the social support group were asked to provide five supportive statements for use in emotionally challenging situations, with the understanding that these statements would be presented to the participants in Session 2. However, in Session 2, all participants were presented with the same set of statements (details of the stimulus sentences are provided in Method S1 in Supplementary Material). Participants in the social support group were shown a picture of their best friend on the screen (details of the stimulus pictures are provided in Method S1 in Supplementary Material), accompanied by a supportive statement. They were asked to cope with stressful thoughts related to the speech using the support depicted. In the non-social support group, participants first saw a scrambled image with instructions to attentively view the subsequent aversive image and experience any emotional responses without manipulating their emotions. This served as the control condition. The material details of the social support manipulation are described in Method S1 in Supplementary Material. Notedly, previous study has set up four groups in the Social Support Manipulation task, including support from friend, support from stranger, self-regulation, and control (Morawetz et al., 2021). However, it has been demonstrated that support from stranger brought higher negative emotions or choice more foods (Morawetz et al., 2021; Xiao, Luo, Zeng et al., 2023), self-regulation activated more disinterested brain regions. Given that we focus on the influence of social support, the present study only set two groups that with or without social support from the best friends.

Food-related tasks

Food Incentive Delay (FID) task. This study employed a modified version of the FID task proposed by Simon (Simon et al., 2015). During each trial, participants were first presented with a geometric figure (cue), followed by a fixed interval (anticipation), and then responded to the direction of an arrow with a button press (reaction). Immediately after their response, feedback appeared indicating whether the participant had won a food reward (feedback). Participants were informed that they could win high-calorie foods (e.g., chips), low-calorie foods (e.g., cherry tomatoes), or nothing (pixelated control) based on their reaction performance. These outcomes were signaled by square, triangle, and circle cues, respectively.

Because each cue corresponded to a specific class of food or object, participants had to learn and remember the association between each cue and its corresponding food/object before beginning the task. Only those who reached 90% accuracy in matching these associations were allowed to proceed to the formal trials. The formal trials included 60 trials in total, with 20 trials for each cue. Food reward sensitivity was defined as the reaction times to the target in the food reward trials, with shorter reaction times reflecting higher reward sensitivity.

Food Portion Choice task. A food portion choice task was used in this study. In each trial, participants were asked to choose how much food they wanted to eat based on their current state. The study included a total of 20 high-calorie foods (e.g., chips) and 20 low-calorie foods (e.g., cherry tomatoes). Each trial presents a type of food with portion sizes from 0 to 4.

Bogus Tasting task. As a behavioral measure of actual high- and low-calorie food consumption, participants were offered a bowl of tomato-flavored potato chips and a bowl of cherry tomatoes. To minimize the influence of social desirability on eating behavior, participants were left alone and instructed to eat as much of the cherry tomatoes and chips as they liked within the next ten minutes to accurately appraise these foods. At the end of the experiment, the experimenter returned to conduct a manipulation check. The amount of food consumed was documented and further analyzed to assess actual consumption of high- and low-calorie foods.

Food incentive delay (FID) task MRI acquisition, processing, and analyses

Brain data were acquired using a 3.0-T Prisma MRI scanner (Siemens). Acquisition details are provided in the Method S2 in Supplementary Material. Participants were asked to fast for approximately 1 hours prior to scanning. Participants performed an FID task while in the scanner to assess neural responses to various foods (high-calorie, low-calorie) and nonfood (pixelated control) images. Neuroimaging data were processed using Statistical Parametric Mapping 12 (SPM12) software (http://www.fil.ion.ucl.ac.uk/spm/) implemented in Matlab R2014b (Mathworks Inc. Sherborn MA, USA). Details regarding preprocessing are provided in the Method S2 in Supplementary Material.

Group differences of brain reactivity to anticipate food cues

To determine social support differences in whole-brain food cue reactivity, we specified the following contrasts: (1) SS vs NSS (main effect of group), (2) high-calorie food vs low-calorie (main effect of calorie), and (3) interaction effect between group and calorie. The corresponding reversed contrasts for the above were also specified. There were four conditions in the first level model, representing each cell in the 2 group (SS vs. NSS) * 2 stimuli (low-calorie vs. high-calorie food cues) within and between-subject factor design. We used a relatively

lenient correction approach (cluster level uncorrected $p < 0.005$, peak level $p < 0.001$, cluster size >10 voxel) to retain as many potential activation areas as possible, to provide valuable seed point coordinates for the subsequent DCM analysis.

Stochastic DCM

DCM with SPM12 (revision 7771) was used for connectivity analysis. DCM is described in detail elsewhere (Friston et al., 2003). We hypothesized that the modulation effect on effective connectivity between brain regions sensitive to executive control, salience, reward would show different patterns when comparing between the social support with the NSS group under high- or low-calorie food cues.

Regions of interest (ROI) were regions that met all the following criteria: 1) showed significant activation in the univariate second-level analysis; 2) showed activation in previous fMRI studies using reward-related Incentive Delay Tasks (Gu et al., 2019); and 3) had a theoretical reason to be activated as per the Risk Model of Eating Disorders (Tanofsky-Kraff et al., 2020). Eight nodes met these criteria and were consequently used in the DCM analyses: (1) right insula, (2) left putamen, (3) right dorsolateral PFC (DLPFC, x=36, y=21, z=30), (4) right caudate, (5) right globus pallidus internus (GPi), (6) left superior parietal lobule (SPL), (7) left ventromedial PFC (VMPFC), and (8) another coordinate of right DLPFC (DLPFC2, x=36, y=36, z=21).

Volumes of interest (VOIs) were obtained by significant activation clusters, which were determined by second-level random effects univariate analysis. Cluster maxima locations were used as VOI centers around which 6-mm spheres were extracted as the VOI regions. The standard SPM procedure was followed by using the principal eigenvariate of each VOI as a summary of the functional activity time series in that VOI (Ma et al., 2015), and each principal eigenvariate time series was adjusted for the F-contrast of effects of interest (Stephan et al., 2010). The same VOIs were used across subjects.

DCM structure inference was conducted using DCM network discovery (Friston et al., 2011). Before the DCM network discovery analysis was conducted, a single full model was specified for each subject. Group-level post hoc optimization was conducted by selecting all inverted full models (one per subject). The optimal sparse model was found at the group level by using Bayesian parameter averaging.

Results

Participant characteristics

Compared to the NSS group (N = 30), the SS group (N = 28) did not show significant differences in age, BMI, time since the last meal, head motion, and psychometric measures related to eating behavior, intrapersonal and interpersonal emotion regulation, the relationship between friends, and personality (see Table S1 in Supplementary Material). These factors could potentially affect the fMRI activity and behavioral eating index.

Group differences of brain reactivity to anticipate food cues

fMRI second-level analyses revealed several statistically significant clusters for main effects and interactions (Table 1 and Fig. 1). The results indicated that the right insula, left putamen, and right DLPFC were significantly activated in the SS group compared to the NSS group. Additionally, the right caudate, right GPi, and left SPL showed greater activation when anticipating low-calorie foods compared to high-calorie foods. Significant activation was also observed in the left VMPFC and right DLPFC in the interaction between task group and food calorie content. The percent signal changes in the significant clusters showing interaction effects were extracted and plotted for different groups. Further analysis demonstrated that the VMPFC and DLPFC (Fig. 1) were more strongly activated in the SS group during anticipation of high-calorie food trials, with no significant difference during anticipation of low-calorie food trials.

Table 1
Summary of univariate fMRI results.

Brain Region	L/R	MNI			*F*	Cluster size (mm^3)
		x	y	z		
Main Effect of Group (SS > NSS)						
Insula	R	45	-12	-6	14.09	621
Putamen	L	-33	-6	0	16.14	405
DLPFC	R	36	21	30	11.89	297
Main Effect of Calorie (High < Low)						
Caudate	R	12	24	0	15.28	513
GPi	R	15	-3	-3	12.65	270
SPL	L	-36	-57	60	16.78	1944
Interaction Effect (Group × Calorie)						
VMPFC	L	-12	45	-6	14.16	324
DLPFC	R	36	36	21	16.19	648

Note. The x, y, and z coordinates and the *F* values were reported on the peak voxel. SS, social support; NSS, non-social support; DLPFC, dorsolateral prefrontal cortex; GPi, globus pallidus internus; SPL, superior parietal lobule; VMPFC, ventromedial prefrontal cortex; L, left; R, right; MNI, Montreal Neurological Institute.

The extracted percent signal changes were also correlated with food cue reaction time and food consumption. Results indicated that, in the SS group, the right GPi activity was negatively correlated with chips consumption (r = -0.454, $p < 0.05$), the right caudate activity was negatively correlated with cherry tomato consumption (r = -0.406, $p < 0.05$). In the non-SS group, the left SPL activity was negatively correlated with chips consumption (r = -0.388, $p = 0.055$). Additionally, the activity of the right insula, left putamen, and the left SPL were positively correlated with reaction times to high- and low-calorie food cues, respectively (high: all $r > 0.409$, $p\langle 0.05$; low: all $r\rangle 0.434$, $p < 0.05$). Age, BMI, time since the last meal, and head motion were included as covariates.

DCM analysis results

Starting from a full model, post hoc optimization revealed a sparse model structure at the group level. The group-level sparse structure regarding modulation effects is shown in Table 2 and Fig. 2, which show the posterior mean strength and posterior probability for each modulatory input and each connection. Only connections that had posterior probability of modulation effect greater than 0.999 are shown in Table 2. Results demonstrated that three connections were modulated by social support under high-calorie food condition, including right insula to right DLPFC, left putamen to left VMPFC, and right caudate to right insula [this connection was negatively correlated with food choices (q[1]= -0.279, $p < 0.05/2$=0.25)]. There were five connections were modulated by social support under low-calorie food condition, including right DLPFC2 to right DLPFC, right DLPFC2 to left VMPFC [this connection was negatively correlated with eating craving after FID task (q = -0.269, $p < 0.05/2$=0.25) and after experiment (q = -0.289, $p < 0.05/2$=0.25)], left SPL to right DLPFC, right caudate to right DLPFC, and right GPi to right insula. The group-level sparse structure regarding the endogenous connections is present in Table 3, which shows the posterior mean strength and posterior probability of each endogenous connection.

Additional analyses results

The Pearson correlation analysis showed that BMI was positively correlated with high-calorie food choices (r = 0.262, p =0.047), and

[1] The effective connection values are not normally distributed; thus, Spearman's correlation coefficients are reported here.

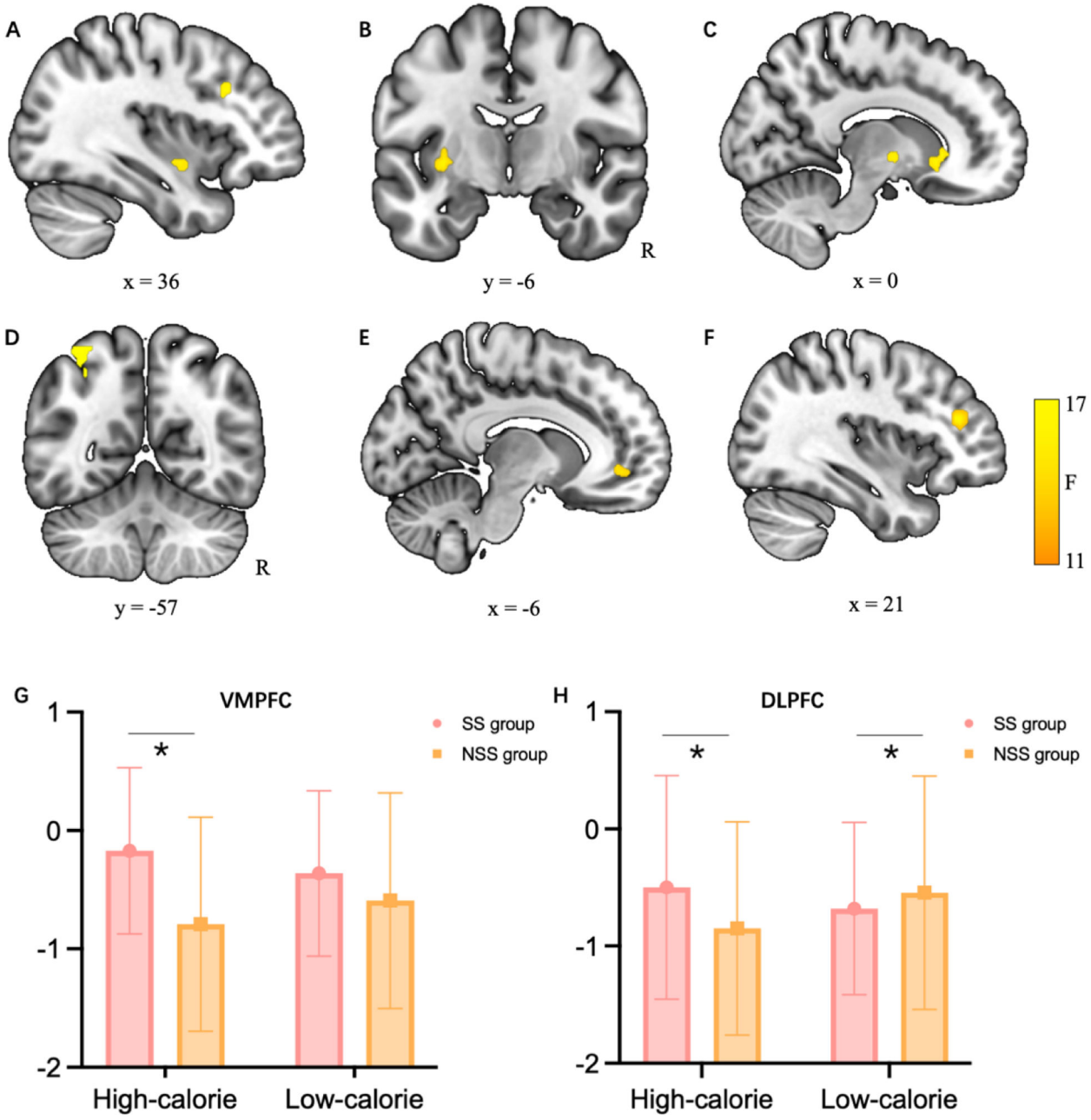

Fig. 1. Univariate two-way functional magnetic resonance imaging analysis of variance results. (A) Right insula, right dorsolateral prefrontal cortex (DLPFC), and (B) left putamen showed significant higher activation in social support (SS) group than non-social support (NSS) group. (C) Right caudate, right globus pallidus internus (GPi), and (D) left superior parietal lobule (SPL) showed significant higher activation when facing low-calorie food cue than high-calorie food cue. (E) Left ventromedial prefrontal cortex (VMPFC) and right dorsolateral prefrontal cortex (DLPFC) showed significant activation in interaction between group and food calorie. R, right hemisphere. Brain activation pattern illustrated in bar graphs for (G) left VMPFC and (H) right DLPFC. The percent signal change for each region was extracted using MarsBaR region of interest toolbox for SPM. Asterisk denotes the significant activation difference between SS group and NSS group in that food calorie condition. Error bars represent SE. *p <0.05.

negatively correlated with the accuracy rate of low-calorie food cues during the FID task (r =-0.293, p =0.026). These results seem convincing because a higher BMI is often associated with greater cravings for high-calorie foods and lower interest in low-calorie foods. In addition, trait perceived stress (r = -0.276, p=0.036), depression symptoms (r = -0.306, p = 0.020), and anxiety symptoms (r = -0.299, p = 0.023) were also negatively correlated with accuracy rate of low-calorie food cues during the FID task, suggesting potential factors that may influence the decision to choose healthy foods. Moreover, emotional eating and uncontrolled eating measured by the TFEQ-R18 were positively correlated with both high-calorie food choices (rs > 0.390, ps < 0.003) and low-calorie food choices (rs > 0.266, ps < 0.043), as well as with food craving after the experiment (rs > 0.280, ps < 0.034). Consider we have controlled the group differences of the above measures before the experiment, it might not influence the main results we focus on, but it still has some enlightening significance for future research.

Additionally, we explored the interaction effect of these measures with the social support condition. The moderation analyses showed that the social support condition moderated the relationship between high-calorie food choices and BMI (interaction β coefficients = 0.175, p = 0.025), emotional eating (β = 0.179, p = 0.012), cognitive restraint (β = -0.169, p = 0.033), and anxiety symptoms (β = 0.175, p = 0.026).

Table 2
Sparse matrix structure—modulation effect.

Connection	High-calorie Food		Low-calorie Food	
	Strength (Hz)	Posterior Probability	Strength (Hz)	Posterior Probability
R Insula → R DLPFC	0.56	0.9999		
L Putamen → L VMPFC	0.57	0.9999		
R Caudate → R Insula	0.75	0.9999		
R DLPFC2 → R DLPFC			-0.65	0.9999
R DLPFC2 → L VMPFC			0.53	0.9999
L SPL → R DLPFC			0.47	0.9999
R Caudate → R DLPFC			-0.57	0.9999
R GPi → R Insula			0.72	0.9999

Among the 56 connections, only those that were identified (posterior probability > 0.999) to be modulated by one of the two food calorie conditions are listed. Posterior mean strength (Hz) and posterior probability of each identified (posterior probability > 0.999) modulation effect is demonstrated. DLPFC, dorsolateral prefrontal cortex; VMPFC, ventromedial prefrontal cortex; SPL, superior parietal lobule; GPi, globus pallidus internus; L, left; R, right.

Specifically, BMI, emotional eating, and anxiety symptoms were significantly and positively correlated with high-calorie food choices, whereas cognitive restraint was significantly and negatively correlated with high-calorie food choices only in the NSS group. None of these relationships were significant in the SS group. These findings suggest that social support may protect against the inevitable pathway from obesity or unhealthy eating habits to actual high-calorie food decisions and consumption.

Furthermore, social support condition moderated the relationship between the time since the last meal and the consumption of cherry tomatoes (β = -4.85, p = 0.020), as well as between baseline perceived stress and reaction time to high-calorie food cues during the FID task (β = 20.82, p = 0.012).

In summary, these behavioral measures were closely related to eating behavior outcomes, which is why we ensured there were no significant group differences between the SS and NSS groups. Nevertheless, we also observed the powerful influence of social support in attenuating several effects of obesity, eating habits or traits, and even stress levels. These findings offer new perspectives for future research and underscore the importance of emotional health in making healthy eating decisions.

Discussion

This study elucidates the neural mechanisms and effective neural pathway linking social support to alterations in neural responses to food cue processing. Individuals who receiving social support exhibited heightened activation in brain regions in the executive control, salience, and reward networks when processing food cues, and displayed a bottom-up effective cooperative pattern among the regions of the three networks. These findings provide evidence for how social rewards affect food cue processing, highlighting interactions among brain systems in substituting social for food rewards. This interaction may be crucial in preventing eating disorders, providing insights for understanding and intervention.

Social support association with brain activity during food cue processing

Through intergroup comparisons between the SS group and the control group, we initially found that when exposed to food cues, participants in SS group activated more areas related to ECN (i.e., right DLPFC, left VMPFC), SN (i.e., right insula), and RN (i.e., left putamen). Notably, the high activation of the VMPFC and DLPFC was specific to high-calorie foods, as the SS group only exhibited greater activation of these regions when confronted with high-calorie food cues.

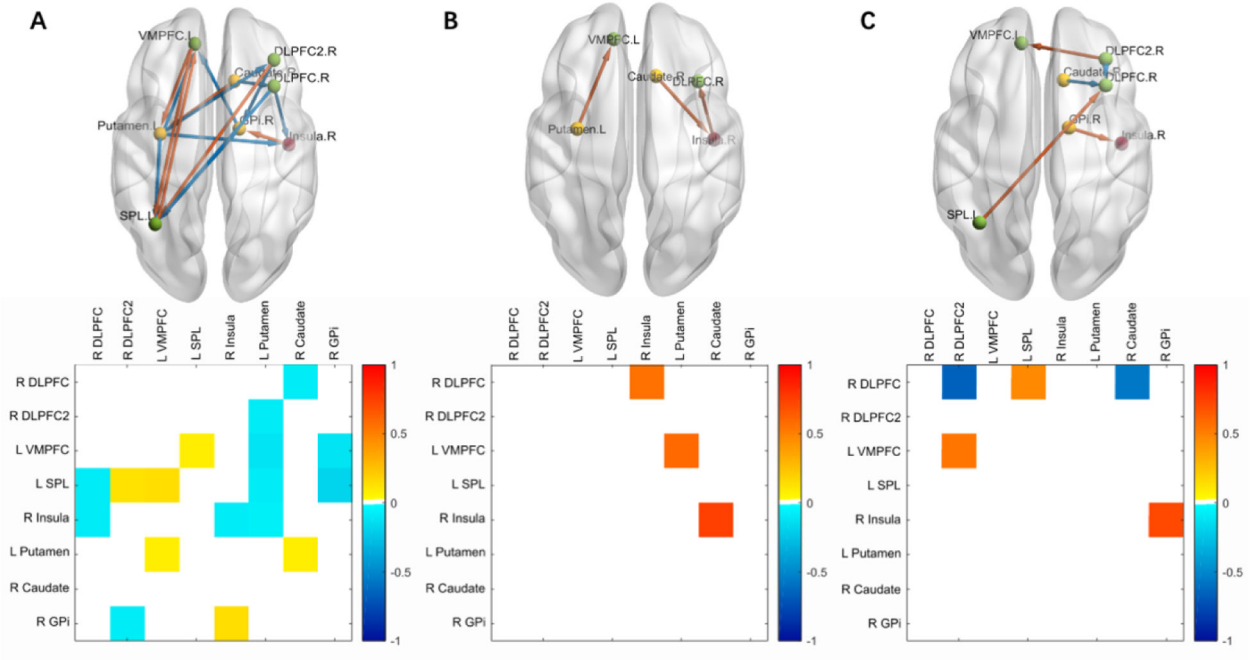

Fig. 2. Schematic diagram representing (A) endogenous connections, (B) effective connectivity modulated by high-calorie food, and (C) effective connectivity modulated by low-calorie food, illustrating the differences between the social support group and the non-social support group. For clarity, in (B) and (C) not all nodes are shown. The positive modulations are shown in orange, and negative modulations are shown in blue. The brain diagram was visualized with the BrainNet Viewer. The matrixes showed the details of these connections (posterior probability > 0.999). DLPFC, dorsolateral prefrontal cortex; GPi, globus pallidus internus; SPL, superior parietal lobule; VMPFC, ventromedial prefrontal cortex; L, left; R, right.

Table 3
Sparse matrix structure—endogenous connection.

Connection	Strength (Hz)	Posterior Probability	Connection	Strength (Hz)	Posterior Probability
R DLPFC → L SPL	-0.08	0.9999	L Putamen → R DLPFC2	-0.08	0.9999
R DPLFC → R Insula	-0.09	0.9999	L Putamen → L VMPFC	-0.12	0.9999
R DLPFC2 → L SPL	0.11	0.9999	L Putamen → L SPL	-0.09	0.9999
R DLPFC2 → R GPi	-0.08	0.9999	L Putamen → R Insula	-0.07	0.9999
L VMPFC → L SPL	0.12	0.9999	R Caudate → R DLPFC	-0.09	0.9999
L VMPFC → L Putamen	0.07	0.9999	R Caudate → L Putamen	0.08	0.9999
L SPL → L VMPFC	0.07	0.9999	R GPi → L VMPFC	-0.11	0.9999
R Insula → R Insula	-0.08	0.9999	R GPi → L SPL	-0.18	0.9999
R Insula → R GPi	0.13	0.9999			

Posterior mean strength (Hz) and posterior probability of each significant endogenous connection are outlined. DLPFC, dorsolateral prefrontal cortex; SPL, superior parietal lobule; GPi, globus pallidus internus; VMPFC, ventromedial prefrontal cortex; L, left; R, right.

Specifically, DLPFC (part of ECN) is responsible for motor planning and inhibitory control. Previous studies have shown that increased DLPFC activity is linked to stronger self-control in food-related decisions (Chen et al., 2018), and stimulating the DLPFC with tDCS enhances its activation and reduces snack consumption and hunger after the experiment (Stinson et al., 2022). In this study, social support activated the DLPFC, aligning with previous findings that support from close others can enhance DLPFC activation (Coan et al., 2017; Morawetz et al., 2021; Nishiyama et al., 2015), suggesting that social support may influence the neural mechanisms underlying emotional regulation strategies (Morawetz et al., 2021), and in turn, impact negative emotion-driven overeating. Another key brain result in ECN is the VMPFC, which play a crucial role in reward-based decision-making, negative emotion regulation, and theory of mind capabilities (Hiser & Koenigs, 2018). Besides, VMPFC play a crucial role in reward-based decision-making (Hiser & Koenigs, 2018). In women with binge eating, this brain region exhibits hypoactivity in response to interpersonal stressors in experimental settings (Jarcho et al., 2015). Multiple studies have found that social rewards (e.g., smiling faces) activate the VMPFC, like monetary and food rewards (Gu et al., 2019; Ruff & Fehr, 2014; Tomova et al., 2020). In this study, social support may restore VMPFC activity under stress, normalizing reward response and reducing cravings for high-calorie foods. This suggests that social support can influence individuals' reward processing by affecting higher-order reward regions involved in social information and decision-making (such as the paralimbic cortex, which including the VMPFC).

We also observed increased insula (part of SN) activation in the social support group during anticipant of foods. Previous studies have shown the important role of insula in integrating emotion processing with interoception (Blom et al., 2015). It's also crucial in converting interoceptive signals into a person's subjective experiences of desire, anticipation, or impulse (Droutman et al., 2015; Noel et al., 2013). In this study, social support increased insula activation, potentially enhancing individuals' accuracy in detecting interoceptive states, thereby allowing for more precise recognition of hunger signals and reducing binge eating driven by psychological factors.

Additionally, individuals with social support exhibited heightened brain reactivity in the putamen (part of RN). In multiple studies related to eating, the putamen is consistently activated during the anticipation of rewards, and its activation levels are generally lower in individuals with eating disorders or those at risk (Hartogsveld et al., 2022). Higher levels of putamen activation correspond to greater reward sensitivity, facilitating a quicker return to normal decision-making and reward responses under stress. In this study, the acquisition of social rewards could influence individuals' value assessments of food rewards, thereby affecting their responses to food cues and outcomes in food selection.

Correlation analysis revealed that in the social support group, right GPi and caudate nucleus activation levels were negatively correlated with chip and cherry tomato intake, respectively, while left SPL activation was negatively correlated with chip consumption in the non-social support group. These regions showed greater activation when anticipating low-calorie foods. More activation in the right insula, left putamen, and SPL was associated with slower response times to food cues in the social support group. These findings indicate a complex relationship between social support, reward sensitivity, and food choices. Social support may influence individuals' reaction times and actual consumption behavior by modulating the activity of these brain regions. This may occur because social support provides a certain level of reward value (social reward), which both alleviates the dampening effect of stress on reward-related brain regions—restoring their activity—and reduces the drive to seek and crave food.

Bottom-up effective connection during high-calorie food cue processing in SS group

Further DCM analysis revealed how social support modulate effective connections. Specifically, under high-calorie food conditions, individuals in SS group exhibited enhanced excitatory effects from right insula to right DLPFC, potentially reflecting the influence of social support on self-control and decision-making, suggesting that social support enhances the activity of the prefrontal cortex via the insula, which is responsible for alertness, thereby improving self-regulation of emotions and behaviors (Strayhorn Jr, 2002). Secondly, the connection from the putamen to the VMPFC indicate enhanced excitatory effects. VMPFC is crucial for reward-based decision-making through its interactions with the striatum, which include putamen (Hiser & Koenigs, 2018). Based on the experimental design and results of this study, we can speculate that social support may enhance VMPFC activation through bottom-up modulation from lower-order reward areas to higher-order reward areas, influencing individuals' reward decisions. Finally, the enhanced excitatory effect of the caudate nucleus on the insula may represent bottom-up modulation of interoception by the basic reward areas, increasing the accuracy of interoceptive signals, which in turn influences the executive control region (DLPFC) via the insula. What's more, this connection negatively predicted the portion size chosen in the subsequent food selection task, which reflect specific neural circuits connecting reward and salience areas that contribute to reduced binge eating behavior. In response to low-calorie food cues, the SS group showed stronger reward-to-insula excitation enhancement, as well as excitation enhancement within the ECN. This may represent a greater involvement of brain regions involved in executive control in the selection of low-calorie foods. Overall, by enhancing the functioning of the reward system, social support facilitates the functionality of the alertness system from the bottom up, which in turn enhances the efficacy of the executive control system.

Limitation

Several limitations of this study are worth noting. First, the sample only included healthy female individuals. While our study aligns with previous reports indicating that women with binge eating exhibit weaker VMPFC functional connectivity (Hiser & Koenigs, 2018), we cannot extend our findings to individuals with clinical eating disorders. Second, the generalizability of the conclusions drawn about female participants may be influenced by the phase of the menstrual cycle

(Frank et al., 2010; Van Vugt, 2010). Although we excluded participants with irregular menstrual cycles during the recruitment phase, we did not capture the specific phase of their cycle at the time of the experiment. Third, we did not find significant results in other emotion-related regions, such as the amygdala. Combined with the authors' previous research (Xiao, Luo, Ding et al., 2023), it is possible that the insula, which acts as a "sentinel", may be more critical than amygdala in the influences of social support on eating behavior.

Conclusion

This study highlights the association between social support and altered brain processing of food cues in regions related to the ECN, SN, and RN. This association is characterized by an enhanced recognition of appetite-related internal homeostasis, heightened sensitivity to food reward cues, and inhibitory control over high-calorie food selection. Ultimately, a bottom-up neural pathway is formed between food sensitivity, interoceptive awareness, and inhibitory control to suppress excessive cravings and consumption for high-calorie foods. While the benefits of social support have long been recognized by people worldwide and by clinical intervention specialists, the actual mechanisms through which social support operates remain poorly understood. The findings of this study underscore the significant role of social support in altering brain function and behavior, providing the first effective neural evidence of a shared neural basis between social reward and food reward, and backing the substitution theory of reward (Carver, 2004). More importantly, these results might provide new perspectives and empirical evidence for interventions and recovery strategies not only for individuals with eating disorders but also for those struggling with other forms of substance addiction.

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Ethics statement

Ethical approval for this study was granted by the Ethics Committee of the Southwest University. All participants were given informed consent before taking part in the experiments.

Declaration of competing interest

The authors declare no conflict of interest.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ijchp.2025.100545.

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