Review Articles

Acupuncture and Herbal Medicine

# From mechanisms to applications: a new perspective on the clinical implementation of acupuncture-induced weight loss *via* eating behavior regulation

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**Abstract**

Aberrant eating behavior constitutes a critical bottleneck in both the development of obesity and the maintenance of weight loss. This review focused on the role of acupuncture in regulating eating behavior for weight loss. Most studies have focused on the regulation of body weight or body mass index but have overlooked the role of eating behaviors. As a non-pharmacological intervention for weight loss, acupuncture offers multitarget advantages that call for an in-depth analysis from the perspective of eating. This review focused on the novel perspective that the core mechanism of acupuncture for weight loss relies on the synergy of multiple pathways involving the central nervous system, gut microbiota, and vagus nerve. The central mechanism of acupuncture for weight loss may lie in restoring the imbalance between the neuropeptide Y/agouti-related protein and pro-opiomelanocortin neurons in the hypothalamic arcuate nucleus, as well as improving the function of the prefrontal–limbic system, thereby inhibiting emotional eating. At the peripheral level, acupuncture may maintain leptin–ghrelin homeostasis, inhibit tumor necrosis factor-alpha or interleukin-6–mediated inflammation, and affect intestinal barrier repair and reconstruction of eating signaling pathways. To address the limitations of current studies, such as the lack of functional magnetic resonance imaging or positron emission tomography neuroimaging evidence, small sample sizes, short follow-up durations, and non-individualized protocols, this review proposes integrated research strategies, including incorporating eating behavior assessment scales, utilizing multi-omics to identify core targets, and conducting multicenter, large-sample, randomized controlled trials. These solutions aim to advance acupuncture from an empirical therapy to precise management, thereby providing novel strategies for obesity prevention and treatment that integrate mechanistic innovations with clinical translational value.

**Keywords:** Acupuncture, Eating behavior, Gut microbiota, Vagus nerve, Weight loss

**Graphical Abstract:** https://links.lww.com/AHM/A236

## Introduction

According to World Health Organization (WHO) data from 2025, 66% of adults worldwide are overweight or obese, and more than 2.9 billion adults globally will be affected by 2030. Furthermore, by 2023, the global annual loss due to obesity is estimated to be approximately 2.5 trillion US dollars (accounting for 2.8% of the global GDP that year), and obesity-related metabolic syndromes have become the third-largest global public health challenge[1]. The standardized average body mass index (BMI) of Chinese adults increased from 22.7 kg/m$^2$ in 2004 to 24.4 kg/m$^2$ in 2018, with the obesity rate increasing from 3.1% to 8.1%[2]. A study on adult primary care patients revealed that nearly 47% of respondents reported experiencing a loss of control over eating (LOCE). Among these individuals, 42.2% were obese, compared to only 9.4% of those who had never experienced such eating control issues[3]. Additionally, the prevalence of emotional eating among overweight and obese populations worldwide is 44.9%[4]. Collectively, these data indicate that aberrant eating behaviors significantly increase the difficulty of achieving weight loss.

As the core regulatory link for energy intake, aberrant eating behaviors are initiating factors for the development of obesity. Eating behavior is regulated by coordinated

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central–peripheral mechanisms. Obesity arises from an imbalance between energy intake and expenditure, which occurs when hypothalamic leptin signaling is impaired (ie, leptin resistance) in the central nervous system (CNS) or when peripheral intestinal ghrelin secretion is abnormally elevated[5]. Consequently, interventions targeting eating behaviors are fundamental for obesity management. However, current weight loss strategies have limitations. The inhibitory effect on eating of GLP-1 receptor (GLP-1R) agonists, such as semaglutide[6], can be counteracted by dopaminergic neuron activity, and maintaining long-term stability at normal levels is difficult[7]. Therefore, normalizing eating behaviors to sustain weight loss efficacy is crucial for obesity management.

Acupuncture is a non-pharmacological weight loss intervention capable of directly or indirectly modulating the CNS[8], and related clinical studies have gradually shifted from regulating body weight to standardizing eating behavior. Traditional weight loss studies have predominantly focused on the effects of acupuncture on body weight and BMI, overlooking its critical role in modulating eating behaviors to promote weight loss. Acupuncture improves appetite scores and restores a normal eating time, with effects superior to those of pharmacological treatments[9]. Therefore, analyzing the clinical effects and mechanisms of acupuncture on weight loss from the perspective of eating behavior normalization is important for refining the theoretical framework of acupuncture-based weight management and optimizing treatment protocols.

In this review, we searched PubMed, Embase, Web of Science, CNKI, Wanfang Data, and VIP from their inception to October 15, 2025, using search terms related to acupuncture, obesity, and eating behavior. Studies with clearly defined interventions and outcome measures were prioritized. This review aimed to analyze the mechanisms of action of acupuncture in weight loss from the perspective of behavioral regulation. The abnormal eating behaviors align with the current neurobehavioral framework, primarily encompassing homeostatic eating imbalance, emotional eating dysregulation, and eating disorders[10]. Homeostatic eating imbalance refers to hypothalamic dysregulation of the perception of the body's energy[11]. Emotional eating dysregulation mainly involves impulsive eating triggered by negative emotions[12]. Furthermore, severe eating disorders, such as binge eating disorder, represent a pathological manifestation of dysregulation across the aforementioned dimensions and fall within the scope of clinical interventions[13]. For quantitative assessments, the studies cited in this review employed various tools. The Food Craving Questionnaire (FCQ) assesses the craving intensity for specific foods[14]; the Emotional Eater Questionnaire (EEQ) focuses on eating tendencies triggered by negative emotions[15]; and the Visual Analog Scale (VAS) is used for real-time quantification of subjective hunger and satiety[16]. This conceptual framework lays the groundwork for a subsequent systematic analysis of the regulatory effects and mechanisms of acupuncture on different dimensions of eating behaviors. A conceptual framework integrating the multipathway mechanisms of acupuncture, regulation of eating behaviors, and translation from clinical evidence to future research is shown in Figure 1.

## Evidence for acupuncture exerting weight loss effects by regulating the eating regulatory center: focusing on the effect of standardizing eating behaviors

Multiple randomized controlled trials (RCTs) have suggested that acupuncture may normalize eating behaviors by modulating food intake, meal frequency, satiety perception, and food preferences[17]. The regulatory effects of acupuncture on food intake and meal frequency are particularly evident[18]. One RCT involving 60 participants who were overweight demonstrated that acupuncture applied to the auricular Shenmen (TF4) and Stomach (CO4) points significantly suppressed appetite[19]. Similarly, auricular acupuncture has been shown to markedly reduce appetite in patients with obesity and counteract the development of binge eating disorder[20]. Another large-scale multicenter RCT involving 278 participants demonstrated that 4 weeks of acupuncture at points such as Zhongwan (CV12) and Zusanli (ST36) significantly improved postprandial fullness and early satiety, with superior effects compared to sham acupuncture[21]. These findings suggest that acupuncture has the potential to regulate appetite, restore normal transmission of appetite-regulating signals, and maintain satiety signals, thereby helping maintain stable meal intervals[22].

Appetite can be deconstructed into key components, such as satiety perception, food preference, eating motivation, and dietary behavior regulation[23]. Acupuncture not only affects food preferences and satiety perception (eg, by reducing the intake of high-sugar and high-fat foods) but may also improve eating motivation (eg, by enhancing appetite in patients with anorexia). Acupuncture may improve the perception of satiety and regulate food preferences, thereby reducing the intake of high-sugar and high-fat foods. Individuals with impaired prefrontal[24–25] and limbic cortex functions exhibit a significantly stronger preference for high-sugar and high-fat foods, suggesting that these brain regions play a role in reducing immediate reward impulses and stabilizing food preferences. Functional magnetic resonance imaging (fMRI) studies have revealed that stimulating auricular points such as the Pancreas and Gallbladder (CO11), Liver (CO12), and Shenmen (TF4) activates both the prefrontal and limbic cortices in humans. This indicates that acupuncture may reduce cravings for high-fat foods among individuals with obesity. In patients with anorexia, auricular acupuncture at points including Shenmen (TF4), Stomach (CO4), and Subcortex (AT4) significantly improves depressive mood and increases appetite and food intake, with superior effects to those of conventional nutritional support alone[26]. Furthermore, an open-label randomized crossover study involving patients with anorexia and bulimia demonstrated that 8 weeks of acupuncture treatment led to significant improvements in eating disorder-related quality of life scores[20], suggesting that acupuncture may alleviate eating disorder symptoms and enhance quality of life in patients with anorexia.

The interventional effect of acupuncture on emotional eating has been associated with improvements in anxiety and depressive symptoms. In a study involving acupuncture treatment in 1,330 participants with obesity



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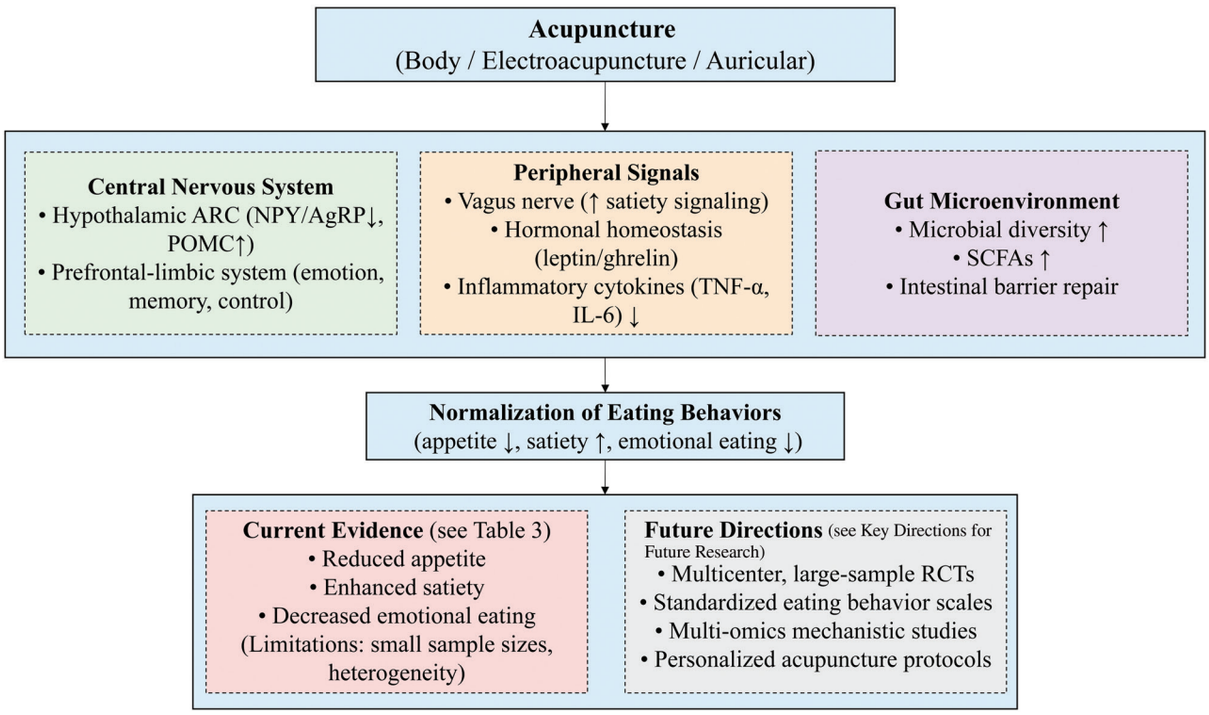



**Figure 1.** Conceptual framework of acupuncture-induced weight loss *via* eating behavior regulation. This diagram illustrates the core structure of the review. Acupuncture (body, EA, or auricular) acts on three interconnected systems: the central nervous system (hypothalamic ARC and prefrontal–limbic network), peripheral signals (vagus nerve, hormones, and inflammatory cytokines), and the gut microenvironment (microbiota, SCFAs, and intestinal barrier). Their synergistic effects lead to the normalization of eating behaviors (reduced appetite, increased satiety, and decreased emotional eating), ultimately contributing to weight loss. The framework also summarizes the key clinical outcomes and limitations of the current evidence (left box) and points toward future high-quality research directions (right box). Created using BioRender. AgRP: Agouti-related protein; ARC: Arcuate nucleus; EA: Electroacupuncture; IL-6: Interleukin-6; NPY: Neuropeptide Y; POMC: Pro-opiomelanocortin; RCTs: Randomized controlled trials; SCFAs: Short-chain fatty acids; TNF-α: Tumor necrosis factor-alpha.

complicated by hyperlipidemia, the total effective rate was 97.52% in patients whose condition was triggered by emotional factors[27]. Research utilizing the Hamilton Anxiety Scale (HAMA) and Hamilton Depression Scale (HAMD) demonstrated that in human patients with obesity and comorbid emotional eating (HAMA score ≥ 14 points), an 8-week acupuncture intervention significantly reduced HAMA scores from (25.61 ± 4.14) to (19.46 ± 3.97) points and HAMD scores from (24.08 ± 4.85) to (19.21 ± 3.86) points. Concurrently, the incidence rate of emotional eating episodes decreased from 68.8% to 21.9% ($P < 0.001$)[28]. Another cohort study of 165 females with obesity showed a 4.8% reduction in body weight in those receiving electroacupuncture (EA) treatment. Furthermore, compared with the sham EA and diet restriction groups, the EA group showed significantly greater alleviation of psychological symptoms, including fear, anger, anxiety, compulsivity, paranoia, and depression. These findings indicate that EA may be an effective method for treating obesity and the associated psychological symptoms[29].

These independent studies have suggested that acupuncture has a regulatory effect on appetite. However, owing to challenges in conducting such studies, including long follow-up periods, difficulty in controlling experimental variables, and limited patient compliance, the number of published studies remains limited. Nevertheless, these clinical phenomena provide clues for uncovering the underlying mechanisms. A structured summary of key clinical studies is shown in Table 1.

## Physiological regulatory network of eating behaviors: synergistic mechanisms from central to peripheral

### *Core CNS pathways for the regulation of eating*

The CNS governs eating behaviors through a hierarchical regulatory network. The hypothalamus serves as the core regulatory center, whereas the limbic system and prefrontal cortex integrate complex signals related to emotions, memory, and other factors. Neurotransmitters and peptides are the key regulatory molecules. This intricate network offers substantial potential for acupuncture in terms of weight loss. The hypothalamic arcuate nucleus (ARC) functions as the first gateway for eating regulation and primarily comprises two functionally antagonistic neuronal populations: neuropeptide Y (NPY)/agouti-related protein (AgRP) neurons and pro-opiomelanocortin (POMC)/cocaine- and amphetamine-regulated transcript (CART) neurons. NPY and AgRP are potent orexigenic factors. During energy deficits, NPY or AgRP neurons are activated and project to the paraventricular nucleus (PVN) to release NPY, which stimulates appetite and suppresses energy expenditure[31]. In contrast, POMC is cleaved to form α-melanocyte-stimulating hormone (α-MSH), which exerts anorexigenic effects by binding to melanocortin-4 receptors (MC4R) in the PVN. CART enhances the anorexigenic effects of POMC[32]. The activity of ARC neurons is also modulated *via* autocrine feedback mechanisms. For instance, AgRP can inhibit the electrical activity of POMC neurons, thereby establishing a dynamic balance[33].



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**Table 1**

**Summary of key clinical studies on acupuncture for eating behaviors and weight loss**

| Study (first author, year) | Study design | Sample size | Acupuncture intervention | Control | Key eating-related outcomes | Major limitations/bias sources |
|---|---|---|---|---|---|---|
| Richards and Marley 1998[19] | Randomized controlled trial | 60 overweight subjects | Transcutaneous electrical stimulation of auricular Shenmen and stomach points, twice daily for 4 weeks | Electrical stimulation on thumb (non-acupoint) | Appetite suppression: 95% in active group *vs.* 0% in control group | Analysis may have included only responders; no sham device; subjective appetite measure; short duration |
| Fogarty et al. 2010[20] | Randomized crossover pilot; 2-week washout | 10 randomized, 9 analyzed (5 AN, 4 BN) | Up to 10 sessions of individualized TCM acupuncture over 13 weeks (avg. 11 needles/session; shallow/light needling; de qi not obligatory) | Usual multidisciplinary care alone | Significant improvement in physical/cognitive domain of EDQoL ($P = 0.0009$) | Very small sample size; open-label; no sham; allocation not concealed; possible carryover; weight not measured; pilot; one dropout excluded |
| Wang et al. 2021[21] | Secondary analysis of a multicenter RCT | 278 patients with postprandial distress syndrome (acupuncture: 138, sham: 140) | Acupuncture at GV20, CV17, CV12, CV6, PC6, and ST36, 20 min/session, 3×/week for 4 weeks | Sham acupuncture (non-meridian, non-acupoint, shallow needling) | Postprandial fullness, early satiety, upper abdominal bloating, belching: significantly improved *vs.* sham during treatment and follow-up ($P < 0.05$) | Secondary analysis (not primary outcome); study not specifically designed for weight loss or eating behaviors; short treatment duration |
| Sun et al. 2020[26] | Pilot randomized controlled trial (two-arm parallel) | 55 patients with stage III/IV cancer and appetite loss (acupuncture: 27, control: 28) | Auricular acupuncture (main points: Shenmen, stomach, spleen, and subcortex; accessory: sympathetic, liver, small intestine, and Sanjiao); intradermal needles on one ear, changed every 2–3 days; patients pressed points 2–3 times/day; 4 weeks treatment + 4 weeks follow-up | Usual nutritional supportive care alone | SNAQ score: acupuncture group improved by 4.48 points *vs.* control group decreased by 0.07 points at week 4; 51.4% appetite improvement; weight gain in acupuncture group *vs.* weight loss in control group | Pilot study; no sham control; no blinding; control received usual care only (not placebo); possible expectancy bias; relatively small sample size; advanced cancer population may limit generalizability to general obesity |
| Wang et al. 2016[27] | Randomized controlled trial | 110 patients with spleen deficiency/damp blockage obesity and hyperlipidemia (observation: 55, control: 55); plus 52 healthy controls | Observation: warm needling (SP3, ST42, SP9, and ST36) + auricular acupuncture (CO13, CO4, CO14, and CO10), warm needling every 2 days, ear acupuncture every 2–3 days, for 3 months | Control: warm needling only (same body points and frequency) | Total effective rate: observation, 96.4% *vs.* control, 87.3% ($P < 0.01$); both groups improved obesity indices similarly; observation group had superior improvements in levels of blood lipids, leptin, and insulin sensitivity (all $P < 0.01$) | No sham acupuncture; no blinding; specific TCM pattern may limit generalizability; no long-term follow-up reported |
| Min et al. 2022[28] | Randomized controlled trial (three arms) | 90 patients with IBS-D (liver depression/spleen deficiency pattern); 30 per group | (1) Acupuncture group: PC6, LR3, ST25, SP6, ST36, ST37, and EX-HN3; even method, 30 min, daily, 5×/week, 3 weeks. (2) Acupuncture + moxibustion group: same acupuncture plus ginger-separated moxibustion at CV8 (3 cones, ~30 min), daily, 5×/week, 3 weeks | Drug group: montmorillonite powder + flupentixol/melitracen tablets, daily, 3 weeks | Both acupuncture groups significantly improved HAMA and HAMD scores *vs.* drug group ($P < 0.05$); acupuncture + moxibustion showed best long-term stability (6-month follow-up) and greatest reduction in serum CRH, ACTH, CORT, and SP levels ($P < 0.05$) | No sham acupuncture/moxibustion; no blinding; no placebo control for drugs; specific TCM pattern may limit generalizability; outcomes focus on psychological symptoms rather than direct eating behavior measures; no weight or BMI reported |

*(Continued)*



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**Table1**
**(Continued)**

| Study (first author, year) | Study design | Sample size | Acupuncture intervention | Control | Key eating-related outcomes | Major limitations/bias sources |
|---|---|---|---|---|---|---|
| Cabioglu et al. 2007[29] | Randomized controlled trial (three-arm parallel) | 165 females with obesity | EA (3 auricular + 6 body points); parameters not clearly specified | (1) Sham EA, (2) diet restriction alone | Weight reduction: EA, 4.8% *vs.* sham EA, 2.7% *vs.* diet, 2.5%; EA significantly reduced phobia, anger, anxiety, obsession, paranoid symptoms, and depression compared to other groups | Blinding unclear; no long-term follow-up; subjective psychological outcomes |
| Chen et al. 2025[30] | Randomized controlled trial (two-arm parallel, sham-controlled) | 68 patients with abdominal obesity (EA, 34 and SA, 34) + 34 healthy controls | EA at bilateral ST36, SP6, CV12, ST25, CV9, SP15, GB26, ST28, ST24, SP14, and CV9; 30 min/session, 3×/week for 8 weeks | Sham EA: superficial needling at non-acupoints (approximately 2 cm lateral to real points), no electrical stimulation | Significantly improved waistline, weight, BMI, hipline, VFA, IWQOL-Lite, and Kessler 10 (all $P < 0.05$) | Small sample size; no microbiota or lipidomics analysis in sham group; single-center design |

ACTH, Adrenocorticotropic hormone; AN, Anorexia nervosa; BMI: Body mass index; BN, Bombesin; CORT, Corticosterone/cortisol; CRH, Corticotropin-releasing hormone; EA: Electroacupuncture; EDQoL, Eating Disorders Quality of Life; HAMA: Hamilton Anxiety Scale; HAMD: Hamilton Depression Scale; IBS-D, Irritable bowel syndrome with diarrhea; IWQOL, Impact of Weight on Quality of Life; RCT: Randomized controlled trials; SA, Sialic acid; SNAQ, Simplified Nutritional Appetite Questionnaire; SP, Substance P; TCM, Traditional Chinese Medicine; VFA, Visceral fat area.

The limbic system and prefrontal cortex serve as higher-level regulatory centers for eating behaviors, with distinct nuclei focusing on different dimensions to coordinate the modulation of eating behavior and maintain energy homeostasis. The amygdala, at the core of emotional processing, acts as an emotion-to-eating converter that specializes in stress- or anxiety-induced emotional eating. The basolateral amygdala (BLA) projects glutamatergic fibers to the ARC, mediating anxiety- and stress-driven overeating[34]. When the body is in a state of chronic stress, excessive activation of BLA neurons promotes the release of orexigenic factors from NPY or AgRP neurons, leading to emotional eating[35]. The hippocampus functions as a memory–reward integrator that regulates the association between memory and rewards to influence food preferences. Specifically, mice with hippocampal lesions exhibit significantly increased cravings for high-sugar diets, suggesting that the hippocampus may regulate eating decisions by inhibiting midbrain reward pathways, such as the ventral tegmental area–nucleus accumbens pathway[36]. Finally, the prefrontal cortex, particularly the dorsolateral prefrontal cortex, serves as an executive controller responsible for the implementation and regulation of eating behaviors. It integrates peripheral energy signals and environmental information to suppress impulsive eating, and its hypofunction is closely associated with overeating in individuals with obesity[37]. These three structures interact *via* downstream pathways to collectively regulate eating behaviors. They coordinate relevant regulatory targets, such as emotions, memories, and executive functions, and act through central and peripheral pathways.

Chemical messengers involved in central regulation primarily include neurotransmitters and peptide substances such as leptin, dopamine, neurotensin, and raptin. These molecules act on central nuclei, such as those in the hypothalamus, to inhibit or promote eating, regulate food preferences, and influence emotional eating. Leptin, a satiety signal secreted by adipose tissue, crosses the blood–brain barrier and binds to leptin receptors (LepRs) in the ARC, which inhibits NPY/AgRP neurons and activates POMC neurons, thereby reducing food intake[38]. Dopamine, a core neurotransmitter in the midbrain reward pathway, influences food preferences by modulating reward thresholds. Reduced expression of dopamine D2 receptors leads to increased consumption of high-sugar and high-fat foods to achieve satisfaction[39]. Downregulated expression of the neurotensin gene (*NTS*) in neurons projecting from the nucleus accumbens lateral shell (NAcLat) to the ventral tegmental area (VTA) reduces the release of neurotensin in the VTA. This reduction diminishes the excitatory regulation of dopamine neurons in the VTA, thereby affecting hedonic eating behaviors[40]. Neurons in the hypothalamic PVN secrete raptin, which acts on both PVN neurons and metabotropic glutamate receptor 3 (GRM3) in the gastric wall. It enhances the appetite-inhibiting activity of neurons while slowing gastric emptying, thereby prolonging the sense of satiety[41]. Such chemical messengers are increasingly being discovered, offering additional therapeutic targets for controlling eating behaviors.

### *Interactive effects between peripheral signals and eating behaviors*

Peripheral tissues relay information on the energy status to the CNS *via* the gut–brain and hormone–neural axes, in which gut microbiota metabolites, pancreatic and adipose tissue hormones, and the vagus nerve serve as key regulatory carriers. Complex interrelationships exist between these components, offering numerous possibilities for obesity interventions. The gut–brain axis is a core channel for peripheral signals to reach the CNS, with the gut microbiota and their metabolites acting as critical regulatory factors. Gut microbiota produce short-chain



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fatty acids (SCFAs), such as acetate, propionate, and butyrate, through the fermentation of dietary fiber. Propionate and butyrate regulate eating behaviors *via* two primary pathways. First, after entering the bloodstream and reaching the hypothalamus, they activate G protein-coupled receptor 41 (GPR41) or G protein-coupled receptor 43 (GPR43) and inhibit the activity of NPY/AgRP neurons[42]. Second, they stimulate intestinal endocrine cells to secrete GLP-1, which directly delays gastric emptying and transmits satiety signals to the brain *via* the vagus nerve[43]. Bile acids, another important group of substances synthesized in the liver and metabolized by gut microbiota, bind to intestinal Takeda G protein–coupled receptor 5 (TGR5) and promote the secretion of GLP-1 and peptide YY (PYY). PYY acts directly on neuropeptide Y receptor type 2 (Y2) in the ARC to suppress appetite[44]. Additionally, the gut microbiota can directly modulate eating behaviors through the vagus nerve–CNS pathway. For instance, bifidobacteria enhance vagal sensitivity, thereby improving the ability of the CNS to perceive satiety signals[45].

In contrast to the complex regulation of eating behaviors, the core organs involved in glucose metabolism are more delineated and have been the focus of several major research teams. The pancreas serves as the central organ that regulates glucose metabolism based on peripheral signals. The hormones secreted by the pancreas interact with the adipose tissue in the feedback regulation of eating behaviors. Pancreatic β-cells secrete insulin, which crosses the blood-brain barrier and binds to insulin receptors (INSR) in the hypothalamus. This binding inhibits NPY/AgRP neurons, activates POMC neurons, and simultaneously enhances the anorexigenic effect of leptin, thereby forming a coordinated eating regulation pattern involving insulin and leptin[46]. In addition, adipose tissues secrete adiponectin, which activates the AMP-activated protein kinase (AMPK) pathway in the hypothalamus to promote POMC expression while suppressing the reward response in appetite centers, thereby reducing high-sugar food intake. Adiponectin also forms a feedback loop with the pancreas[47]. Notably, crosstalk exists between peripheral hormones and gut microbiota metabolites. For example, insulin modulates the composition of the gut microbiota by increasing the abundance of *Lactobacillus*, thereby promoting the production of SCFAs, which further enhance anorexigenic signaling. This represents a typical example of a peripheral–central interaction[48], which is particularly prominent in high-sugar–related eating behaviors.

The vagus nerve serves as a critical pathway for the transmission of peripheral signals to the CNS. Its afferent fibers detect the energy status of the gut, pancreas, and adipose tissues. Mechanical distension of the intestine, such as increased gastric volume after eating, activates mechanoreceptors in the vagus nerve that project to the hypothalamus *via* the nucleus tractus solitarius to convey satiety signals[49]. Meanwhile, intestinal endocrine cell–derived hormones such as GLP-1 and PYY bind to receptors on vagal nerve terminals, activate chemoreceptors, and enhance the appetite-inhibiting effect of the CNS[50]. In addition, the vagus nerve regulates peripheral organ functions through efferent fibers. For example, the hypothalamus modulates ghrelin secretion *via* vagal efferent activity, forming a central–peripheral feedback loop[51]. Under conditions of a high-fat diet (HFD) or energy surplus, pancreatic parenchymal cells, particularly in the exocrine compartment, accumulate free fatty acids such as palmitic and oleic acids. These fatty acids activate free fatty acid receptor 4 (FFA4) on vagal afferent terminals[52], triggering a $G_q$-type G protein–mediated signaling pathway[53]. This promotes phospholipase C (PLC) activation, elevates intracellular inositol trisphosphate (IP3) levels, and mobilizes calcium release from the endoplasmic reticulum, thereby enhancing neural firing[54]. This signal alerts the CNS to pancreatic lipid overload, influencing subsequent eating preferences such as reducing high-fat food intake and promoting energy expenditure to facilitate weight loss. Simultaneously, adenosine triphosphate (ATP) released by pancreatic cells under metabolic stress binds to P2X purinergic receptor subtype 3 (P2X3) on afferent nerve terminals[55], rapidly activating neural impulses[56] and contributing to central regulation during pancreatic stress. Together, these vagus-mediated pathways help stabilize appetite and maintain body weight.

### *Dysregulation of the eating regulatory network in pathological states*

Obesity disrupts the homeostasis of the central–peripheral eating regulatory network, primarily manifesting as central leptin resistance, gut microbiota dysbiosis, and emotional stress–mediated inflammatory responses. These pathological changes are not mutually independent but involve complex interactive processes, which is one explanation for the low success rates of weight loss. Central leptin resistance and abnormally elevated peripheral ghrelin levels represent core dysregulatory factors in the regulation of eating in obesity. Under physiological conditions, the binding of leptin to LEPR activates the Janus kinase 2-signal transducer and activator of transcription 3 signaling pathway (JAK2-STAT3), thereby inhibiting NPY/AgRP neurons. However, adipose tissue hyperplasia leads to chronically elevated leptin levels in individuals with obesity. The long-term hyperleptinemic environment causes downregulation of LEPR expression in the ARC and impairs the phosphorylation of STAT3, leading to the development of leptin resistance[57]. Leptin resistance results in sustained activation of NPY/AgRP neurons and inhibition of POMC neurons, leading to an intense appetite even when the body has an energy surplus. Additionally, obesity disrupts the rhythm of ghrelin secretion in the gastric fundus. In individuals with a normal weight, ghrelin levels increase before meals and decrease after eating. In contrast, patients with obesity exhibit a blunted postprandial reduction in ghrelin levels, accompanied by significantly elevated nocturnal ghrelin levels. This aberrant profile promotes overeating before meals and nighttime snacking[58].

Emotional stress and chronic inflammation exacerbate the dysregulation of the eating regulatory network. Chronic emotional stress, such as work pressure and anxiety, activates the hypothalamic–pituitary–adrenal (HPA) axis, leading to increased cortisol secretion. Cortisol acts on glucocorticoid receptors in the ARC, activating NPY/AgRP neurons and suppressing



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POMC neurons, thereby promoting emotional eating[59]. Furthermore, chronic low-grade inflammation in obesity, characterized by elevated serum levels of tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), disrupts eating regulation through two principal mechanisms. First, inflammatory factors cross the blood-brain barrier, activate hypothalamic microglia, and trigger the release of proinflammatory factors such as IL-1β, which impair leptin signaling[60]. Second, inflammatory factors reduce the sensitivity of the intestinal vagus nerves and weaken the transmission of peripheral satiety signals to the CNS, leading to delayed satiety perception[61]. Collectively, these processes may lead to a vicious cycle of negative emotions, excessive food intake, and exacerbated negative emotions[62].

Gut microbiota dysbiosis is an important consequence of aberrant eating behaviors associated with obesity. During the fermentation of dietary fiber, gut microbiota produce acetate, which interacts with the hypothalamus to suppress AgRP neuronal activity and activate POMC neurons, thereby reducing food intake[63]. Patients with abdominal obesity exhibit reduced gut microbial diversity[30], which may contribute to delayed satiety and an unstable appetite. Gut microbiota dysbiosis leads to a decreased production of SCFAs[64], weakening their anorexigenic effects on the hypothalamus[65]. Concurrently, the overgrowth of harmful bacteria such as *Escherichia coli* promotes the release of endotoxins such as lipopolysaccharides (LPS). LPS disrupts the intestinal barrier, enters the bloodstream, and triggers chronic low-grade inflammation[66]. Furthermore, microbial disruption affects bile acid metabolism. In individuals with obesity, the proportion of secondary bile acids, such as deoxycholic acid, increases in the gut, with excessive activation of TGR5 receptors, resulting in abnormal secretion of GLP-1 and further exacerbation of dysregulated eating behaviors[67] A summary of core network nodes and signals and an overview of the integrated regulatory network in physiological and pathological states are presented in Table 2 and Figure 2, respectively.

## Putative mechanisms and experimental evidence for acupuncture-mediated regulation of eating behaviors

### *Effect of acupuncture on central eating regulatory pathways*

#### *Animal experimental evidence*

In HFD-induced models of obesity, the expression of the orexigenic NPY/AgRP neurons in the ARC is upregulated, while the expression of the anorexigenic POMC/CART neurons and their downstream product α-MSH is reduced, leading to hyperphagia. EA stimulation at acupoints such as Zusanli (ST36) and Sanyinjiao (SP6) can significantly downregulate both messenger ribonucleic acid (mRNA) and protein levels of NPY and AgRP in the hypothalamus, while upregulating POMC expression and increasing α-MSH levels in the cerebrospinal fluid (CSF). Notably, 2 Hz EA has demonstrated superior efficacy in this regulation[68]. This process is closely associated with the demethylation of the tuberous sclerosis complex 1 gene promoter and inhibition of the mammalian target of rapamycin complex 1 (mTORC1) signaling pathway[69], ultimately reducing food intake, body weight, and body fat percentage. ARC lesions completely abolish the satiety-inducing effects of EA, confirming its core mediating role in this regulatory process.

Animal studies have demonstrated that after 4 weeks of acupuncture intervention, HFD-induced obese rats show increased hypothalamic dopamine D2 receptor expression and 5-hydroxytryptamine (5-HT) levels[70], and the increase in dopamine levels is negatively correlated with reduced high-sugar food intake. Furthermore, in obese type 2 diabetic (T2DM) rats, acupuncture intervention increases the hypothalamic phosphorylated signal transducer and activator of transcription 3 (p-STAT3)/signal transducer and activator of transcription 3 (STAT3) ratio, which is negatively correlated with leptin levels[71].

#### *Human mechanistic observations*

fMRI has revealed that EA at points such as Zusanli (ST36) and Hegu (LI4) activates the hypothalamus and nucleus accumbens (ie, the reward system) while inhibiting the amygdala (ie, emotional regulation)[72]. Acupuncture at acupoints such as Taichong (LR3) and Xingjian (LR2) broadly suppresses the limbic–paralimbic–neocortical network, including the medial prefrontal cortex, amygdala, and hippocampus, while activating the insula and anterior cingulate cortex[73]. Clinical research has found that after 12 weeks of auricular acupuncture, serum leptin and ghrelin levels decrease significantly compared with pre-intervention levels, and the leptin–ghrelin ratio is restored to near-normal levels.

#### *Mechanistic inferences and synthesis*

Research suggests that acupuncture may restore eating-related signaling pathways, thereby affecting weight loss and improving emotional eating through the specific modulation of hypothalamic eating regulatory nuclei, repair of central eating networks (eg, hypothalamus activation and amygdala inhibition), and regulation of central neurotransmitters and peripheral peptides. Acupuncture may exert a relatively specific modulatory effect on certain eating-related nuclei within the hypothalamus, which may contribute to appetite stabilization and weight loss.

Furthermore, acupuncture can restore the central eating network by coordinating multiple eating-related brain centers. Network remodeling is hypothesized to ameliorate emotional eating disorders by modulating emotional memory and visceral sensations. The dynamic balance between activation and inhibition likely helps restore the emotion–eating connection, thereby normalizing the dysfunctional eating network[74]. These findings indicate that different acupoints can produce convergent modulatory effects on brain networks, supporting the notion that acupuncture primarily acts through network-level restoration.

Acupuncture modulates the levels of central neurotransmitters and peripheral peptides, which may restore eating signaling pathways. In summary, the modulation of the hypothalamic ARC nucleus by acupuncture primarily corresponds to the regulation of satiety, directly influencing the food intake volume and meal



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**Table 2**

**Core network nodes, signals for eating behavior regulation, and obesity-related pathological alterations**

| Category | Core components | Primary functions | Pathological alterations in obesity |
|---|---|---|---|
| Central regulatory nodes | ARC (NPY/AgRP and POMC/CART neurons) | Regulates appetite; NPY/AgRP stimulates eating, POMC/CART inhibits eating[31]. Forms dynamic balance *via* autocrine feedback[33]. Integrates peripheral energy signals to maintain energy homeostasis[38,46,49] | Increased NPY/AgRP and decreased POMC activities. Neuronal imbalance leads to persistent hunger[31] |
| | Amygdala | Emotion–eating converter: processes stress/anxiety-induced emotional eating. BLA influences ARC *via* glutamatergic projections[34] | BLA hyperactivation under chronic stress promotes the release of orexigenic factors (eg, NPY/AgRP), exacerbating emotional eating[35] |
| | Hippocampus | Memory–reward integrator: links food memories with rewards, influencing food preferences[36] | Impaired function may increase cravings for high-sugar foods[36] |
| | Prefrontal cortex (especially dorsolateral) | Executive controller: regulates eating behavior, integrates peripheral energy signals and environmental information, and inhibits impulsive eating[37] | Hypofunction is associated with overeating |
| | Nucleus accumbens (lateral) to ventral Tegmental area | Core of the reward pathway: dopamine regulates food reward threshold, influencing eating preferences[40] | Reduced dopamine D2 receptor expression, leading to a need for more high-fat/high-sugar food to achieve satisfaction[39] |
| Peripheral key signals | SCFAs (gut microbiota metabolites) | Enter circulation to directly suppress appetite in the hypothalamus[42]. Stimulate intestinal endocrine cells to secrete GLP-1, delaying gastric emptying and transmitting satiety signals to the CNS[43] | Gut dysbiosis leads to decreased SCFA production, weakening satiety signals[65] |
| | Bile acids (synthesized in liver and metabolized by gut microbiota) | Bind to intestinal TGR5 receptors, promoting GLP-1 and PYY release, exerting anorectic effects[44] | Altered composition may disrupt GLP-1 secretion patterns[67] |
| | Leptin (adipose tissue) | Satiety signal, inhibits appetite[38] | Leptin resistance: impaired signal transduction despite high leptin levels, loss of anorectic effect[57] |
| | Ghrelin (stomach) | Hunger signal: increases preprandially, decreases postprandially[51] | Disrupted rhythm: insufficient postprandial decline, elevated nocturnal levels, promoting pre-meal overeating and night eating[58] |
| | Insulin (pancreatic β-cells) | Binds to hypothalamic INSR, suppresses appetite; enhances anorectic effect of leptin[46]. Modulates gut microbiota composition, promoting SCFA production and enhancing satiety signaling[48] | Central insulin resistance may contribute to appetite dysregulation |
| | Adiponectin (adipose tissue) | Activates the AMPK pathway, suppressing the reward response in appetite centers; forms a feedback loop with the pancreas[47] | Levels are often reduced in obesity, diminishing its protective effect |
| Obesity-related pathological alterations | Central leptin resistance | Hyperleptinemia with impaired LEPR-JAK2-STAT3 signaling in the hypothalamus leads to leptin resistance, sustained activation of NPY/AgRP neurons, and inhibition of POMC neurons[57] | Strong appetite persists despite energy surplus[57] |
| | Disrupted ghrelin secretion rhythm from gastric fundus | Delayed postprandial decline, elevated nocturnal levels[58] | Disrupts normal intermeal intervals, promoting additional food intake[58] |
| | Emotional stress and chronic low-grade inflammation | Emotional stress activates the HPA axis, increasing cortisol and promoting emotional eating[59]<br>Elevated serum levels of inflammatory factors (eg, TNF-α and IL-6). Reduces intestinal vagal nerve sensitivity, delaying satiety[60]. Activates hypothalamic microglia, releasing proinflammatory cytokines and inhibiting leptin signaling[61] | Forms a vicious cycle of "negative emotion–excessive food intake–worsened negative emotion"[62] |
| | Gut microbiota dysbiosis | Decreased diversity[30], reduction in beneficial bacteria such as SCFA producers[65], increase in harmful bacteria[66] | Reduces SCFA production[30]; induces chronic inflammation[65]; impairs bile acid metabolism, aggravating eating dysregulation[66] |

AMPK: AMP-activated protein kinase; AgRP: Agouti-related protein; ARC: Arcuate nucleus; BLA: Basolateral amygdala; CART: Cocaine- and amphetamine-regulated transcript; CNS: Central nervous system; GLP-1: Glucagon-like peptide 1; HPA: Hypothalamic–pituitary–adrenal; IL-6: Interleukin-6; NPY: Neuropeptide Y; POMC: Pro-opiomelanocortin; PYY: Peptide YY; SCFA: Short-chain fatty acids; TNF-α: Tumor necrosis factor-alpha.



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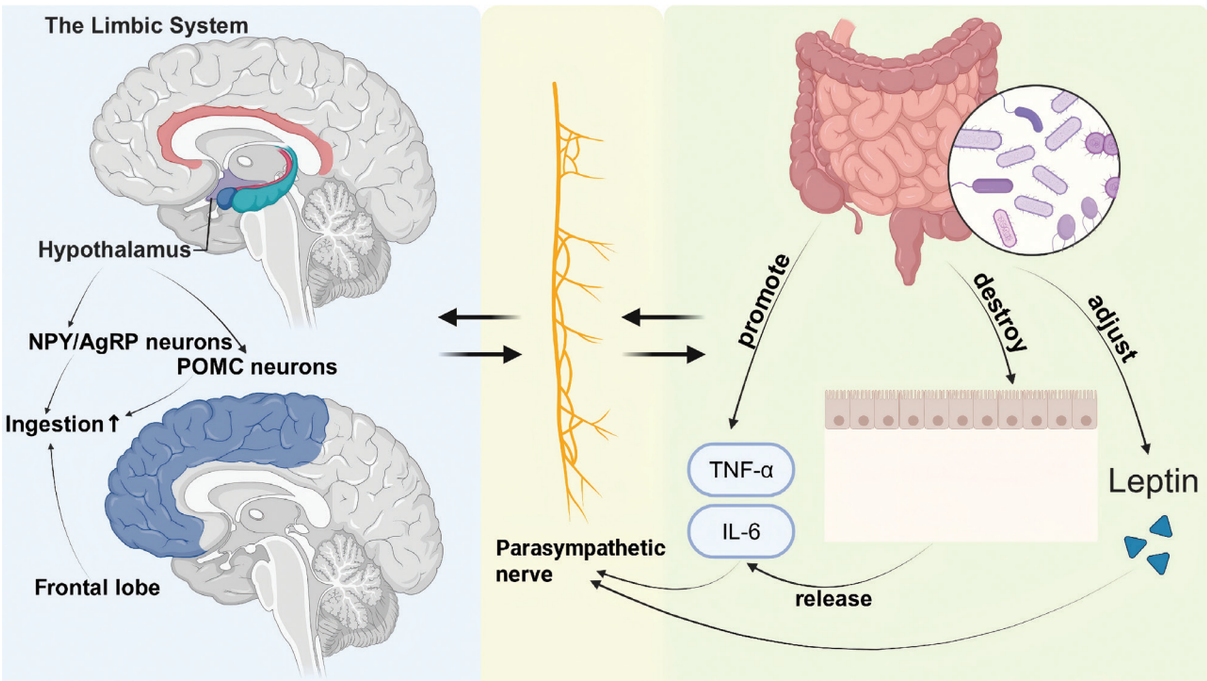



**Figure 2.** Effect of intestinal flora on the central vagal system in obesity. This schematic illustrates the bidirectional communication between the central nervous system, gut microbiota, and vagus nerve in energy homeostasis. Central dysregulation involves an imbalance between orexigenic (NPY/AgRP) and anorexigenic (POMC) neurons in the hypothalamic ARC, coupled with impaired prefrontal–limbic system function, promoting emotional eating. Peripherally, altered gut microbiota composition, impaired intestinal barrier, and chronic inflammation (eg, TNF-α and IL-6) disrupt leptin–ghrelin homeostasis. These peripheral signals are relayed to the brain *via* humoral pathways and to the vagus nerve, which serves as a critical neural conduit. The resulting dysregulation of this axis drives aberrant feeding behaviors and obesity pathogenesis. AgRP: Agouti-related protein; ARC: Arcuate nucleus; IL-6: Interleukin-6; NPY: Neuropeptide Y; POMC: Pro-opiomelanocortin; TNF-α: Tumor necrosis factor-alpha.

frequency. Its regulation of the prefrontal cortex–limbic system network is closely associated with inhibiting impulsive eating, improving emotional eating, and modulating food preferences, such as reducing cravings for high-fat and high-sugar foods. The reduction in food intake observed in animal experiments has been directly correlated with the decreased appetite VAS scores and reduced total caloric intake recorded in human dietary logs.

Notably, different acupuncture modalities, including manual acupuncture, EA, and auricular acupuncture, may involve overlapping but not identical neural pathways in regulating eating behaviors. Body acupuncture (eg, ST36, SP6) primarily exerts its effects by modulating hypothalamic ARC neuronal activity and vagal afferent or efferent functions, whereas auricular acupuncture (eg, Shenmen, Stomach) may project more directly to the limbic system because of the anatomical distribution of the auricular branch of the vagus nerve. Further research is warranted to elucidate the relative specificity and synergistic effects of different acupuncture modalities in the regulation of eating behaviors. Additionally, EA parameters may exert differential regulatory effects on eating behaviors. Specific EA frequencies may influence energy metabolism, and in terms of acupoint selection, the abdominal and limb acupoints may affect eating behaviors through distinct peripheral or central mechanisms. These findings suggest that future EA treatment strategies should be individualized based on the specific eating behavior subtypes.

## *Modulation of the vagus nerve–peripheral signaling pathway by acupuncture*

### *Animal experimental evidence*

*In vivo* vagus nerve electrical activity recordings of acupuncture interventions performed in HFD-induced obese rats show an increased discharge frequency of vagal afferent fibers in the acupuncture group and a positive correlation between this frequency and delayed gastric emptying[75]. Animal studies have demonstrated that 4 weeks of acupuncture intervention in obese T2DM rats results in a significantly increased pancreatic insulin sensitivity index (ISI) and insulin secretion[17]. The effect of acupuncture on insulin balance is abolished after sympathetic[76] or sensory[18] nerve transection, indicating the essential role of autonomic nerves in mediating peripheral hormonal regulation during acupuncture. Additionally, acupuncture in obese model rats reduces the expression of NPY in the hypothalamus and decreases peripheral NPY secretion, thereby suppressing appetite, reducing food intake and hunger sensations, and ultimately influencing energy intake[77].

### *Human mechanistic observations*

Acupuncture inhibits sham eating-induced gastric acid secretion by modulating vagal activity[78], demonstrating its role in regulating gastrointestinal function through the vagus nerve. This finding supports the involvement of the vagus nerve in the acupuncture-mediated regulation of eating behaviors.



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#### *Mechanistic inferences and synthesis*

Acupuncture may restore peripheral–central eating signaling pathways by enhancing vagus nerve activity, modulating the secretion of peripheral hormones, and suppressing chronic inflammation. Acupuncture facilitates the transmission of peripheral satiety signals by enhancing vagus nerve activity and may regulate insulin secretion *via* the autonomic nerve–pancreatic axis. In obesity, the body is in a state of low-grade chronic inflammation, and acupuncture may ameliorate the inflammation-mediated disruption of eating signals.

As the core of eating signal integration, the hypothalamus exhibits microglial activation *via* increased expression of ionized calcium-binding adapter molecule 1 (Iba-1), which significantly increases the release of proinflammatory cytokines such as TNF-α and IL-6. These cytokines directly inhibit the activity of ARC POMC neurons and activate NPY/AgRP neurons, resulting in the neurocircuitry dysfunction underlying abnormal eating[79]. Acupuncture may interrupt this circuit through neural regulation: hypothalamic microglial activation is inhibited, reducing the neurotoxicity of proinflammatory cytokines[80], while the downregulated phosphorylation of nuclear factor-κB p65 (NF-κB p65) blocks the cascade amplification of inflammatory signals within neurons. This ultimately restores the regulatory function of leptin in the hypothalamic appetite center[81].

The enhancement of vagal nerve activity by acupuncture and its regulation of the rhythms of peripheral hormones, such as those of leptin and ghrelin, primarily affect the timely initiation and maintenance of postprandial satiety, thereby influencing single-meal food intake and intermeal intervals. The modulation of nocturnal ghrelin rhythms may improve nighttime eating behaviors. Evidence from animal experiments indicates that acupuncture delays gastric emptying and alters postprandial hormone profiles. These physiological changes have been associated with enhanced satiety (measured by VAS) and decreased snack intake (recorded *via* 24-hour dietary recall) in human studies included in this review.

### *Acupuncture regulates eating behaviors via the gut microbiota–gut–brain axis*

#### *Animal experimental evidence*

Acupuncture at the Daimai (GB26) acupoint in HFD-fed rats significantly reduces body weight, decreases food intake, enhances lipid metabolism, increases the abundance of beneficial bacteria, such as those belonging to the phylum Bacteroidetes, and reduces the proportion of harmful bacteria[82]. Tian et al.[83] also confirmed that EA stimulation of HFD-fed rats at acupoints, including Zhongwan (CV12), Guanyuan (CV4), Zusanli (ST36), and Fenglong (ST40), regulates the structure of small intestinal microbiota and promotes its rebalance, thereby reducing body weight, systemic chronic inflammatory responses, and excessive eating. Fecal microbiota transplantation (FMT) experiments further confirm the mediating role of microbiota; when feces from acupuncture-treated obese rats are transplanted into non-intervened obese rats, the recipient rats show a significantly greater reduction in food intake and body weight than those that received feces from untreated obese model rats[84].

A previous study used 16S rRNA gene sequencing combined with high-performance liquid chromatography-mass spectrometry (HPLC-MS) metabolomics to demonstrate that EA treatment significantly alters 10 bacterial genera and restores 11 metabolites to normal levels in the intestines of HFD-induced obese mice[85]. EA at Dachangshu (BL25) and Tianshu (ST25) can alleviate pathological manifestations in the colon of HFD-fed mice, including reducing inflammatory cell infiltration, epithelial loss, crypt structure destruction, and goblet cell damage, thereby decreasing food intake[86]. Mechanistically, animal studies have demonstrated that EA increases the expression of colonic tight junction proteins, reduces intestinal inflammation and cell apoptosis, regulates the ghrelin and PYY levels in colonic and hypothalamic tissues, improves gut microbiota structure, and stabilizes appetite[87].

#### *Human mechanistic and clinical observations*

Clinical studies have demonstrated that EA treatment significantly increases the gut microbiota α-diversity in patients with obesity, optimizes the Firmicutes/Bacteroidetes ratio, and promotes the production of SCFAs, such as acetate, propionate, and butyrate, by gut microbiota. These metabolites activate G protein–coupled receptors (GPCRs), stabilize appetite, and regulate host energy metabolism and immune homeostasis[30].

#### *Mechanistic inferences and synthesis*

Animal and clinical studies have suggested that acupuncture ameliorates obesity-related gut microbiota dysbiosis. Acupuncture may exert weight loss effects by promoting the production of beneficial metabolites, suggesting that EA can inhibit appetite and ameliorate obesity by regulating gut microbiota composition and metabolite levels. Acupuncture not only repairs intestinal barrier function but may also reduce chronic inflammation mediated by obesity and its complications and improve eating regulation. Acupuncture-induced remodeling of the gut microbiota and the promotion of its metabolites (eg, SCFAs) may exert a multipathway influence on eating behaviors. Notably, SCFAs and signaling molecules, such as GLP-1, enhance satiety[88]. The amelioration of the microbiota–immune–brain axis alleviates emotional eating, and the modulation of bile acid metabolism by the microbiota may indirectly affect food preferences. In animal studies, reduced food intake and weight loss accompanied by improved gut microbiota composition have been consistently observed. These physiological changes are consistent with the improved eating behavior outcomes reported in the human studies included in this review, including reduced FCQ and EEQ scores. Table 3 provides an overview of physiological roles and potential acupuncture targets. The integrated pathways through which acupuncture normalizes eating behaviors and subsequently reduces body weight are summarized in Figures 3 and 4, respectively.



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**Table 3**

**Physiological roles of key nodes in eating regulation and potential targets and evidence for acupuncture interventions**

| Node/signal | Primary physiological role in eating behaviors | Potential acupuncture target/evidence |
|---|---|---|
| Central nodes | | |
| Hypothalamic ARC (NPY/AgRP, POMC) | Core of energy homeostasis: NPY/AgRP neurons stimulate appetite; POMC neurons produce α-MSH, suppressing appetite. Integrates signals (eg, leptin and insulin) to maintain the long-term energy balance | Target: ARC neuronal activity<br>Evidence: 2 Hz EA at acupoints (eg, ST36, SP6, and LI4) downregulates NPY and AgRP expression and upregulates POMC expression and cerebrospinal fluid α-MSH levels in obesity models, reducing food intake[68–69,72] |
| Amygdala | Emotion–eating converter: processes stress/anxiety-induced emotional eating. The BLA influences ARC *via* glutamatergic projections | Target: amygdala hyperactivity<br>Evidence: EA at acupoints (eg, ST36, LI4, LR3, and LR2) suppresses amygdala activity[72–73] |
| Prefrontal cortex | Executive controller: regulates eating behavior, integrates peripheral energy signals and environmental information, and inhibits impulsive eating | Target: prefrontal cortical function<br>Evidence: acupuncture at auricular points (eg, CO11, CO12, and TF4) may activate the prefrontal cortex, potentially enhancing cognitive control over eating impulses[26] |
| Nucleus accumbens | Core of the reward pathway: dopamine modulates the food reward threshold, influencing eating preferences | Target: reward circuitry<br>Evidence: EA at ST36 and LI4 activates the nucleus accumbens[72]. Acupuncture increases dopamine D2 receptor and 5-HT levels in the hypothalamus and nucleus accumbens, affecting reward perception[70] |
| Peripheral signals | | |
| Vagus nerve | Transmits gastrointestinal satiety signals; regulates gastrointestinal function and hormone secretion | Target: vagal afferent activity<br>Evidence: acupuncture enhances vagal afferent discharge frequency[75] and inhibits sham eating-induced gastric acid secretion[78], indicating an ability to modulate vagally mediated gastrointestinal feedback and appetite control |
| Leptin | Satiety signal: secreted by adipocytes, acts on the hypothalamus to suppress appetite | Target: leptin sensitivity<br>Evidence: acupuncture intervention reduces serum leptin levels, ameliorates leptin resistance, and normalizes the leptin–ghrelin ratio[71] |
| Ghrelin | Hunger signal: secreted by the stomach; increases preprandially to stimulate appetite, and decreases postprandially | Target: ghrelin secretion rhythm<br>Evidence: acupuncture regulates abnormal ghrelin secretion patterns and reduces its levels[71] |
| SCFAs | Gut microbiota metabolites: produced by the fermentation of dietary fiber, such as acetate, propionate, and butyrate. Stimulates intestinal secretion of GLP-1/PYY; may travel to the brain and directly suppress appetite | Target: gut microbiota and SCFA production<br>Evidence: acupuncture stimulation remodels the gut microbiota and promotes SCFA production[85] |
| Gut microbiota | Core modulator of the gut–brain axis: communicates with the CNS *via* metabolites (eg, SCFAs, bile acids), immune, and neural pathways, affecting appetite, inflammation, and energy metabolism | Target: microbiota composition<br>Evidence: acupuncture significantly improves obesity-associated microbial dysbiosis, increases diversity, and optimizes community structure[30,82–83] |
| Chronic inflammation (TNF-α and IL-6) | Pathological disruptor: peripheral and central inflammation induces leptin and insulin resistance, damages hypothalamic neurons, and reduces vagal nerve sensitivity | Target: inflammatory signaling<br>Evidence: acupuncture reduces serum and hypothalamic levels of proinflammatory cytokines (TNF-α and IL-6), inhibits hypothalamic microglial activation, and improves inflammation-mediated eating dysregulation[79–80,83] |

α-MSH: α-Melanocyte-stimulating hormone; 5-HT: 5-Hydroxytryptamine; AgRP: Agouti-related protein; ARC: Arcuate nucleus; BLA: Basolateral amygdala; CNS: Central nervous system; EA: Electroacupuncture; GLP-1: Glucagon-like peptide 1; IL-6: Interleukin-6; NPY: Neuropeptide Y; POMC: Pro-opiomelanocortin; PYY: Peptide YY; SCFA: Short-chain fatty acids; TNF-α: Tumor necrosis factor-alpha.

## Limitations of current research and future research directions

### *Limitations of existing research*

Clinical studies face challenges such as small sample sizes, short follow-up durations, and inconsistent evaluation indicators. Most current RCTs on acupuncture for weight loss have small sample sizes, and multicenter studies are rare, which may limit the generalizability of the results. The follow-up durations have been relatively short, failing to fully assess the long-term regulatory effects of acupuncture on eating behaviors. Furthermore, unified standards for evaluating eating behaviors are lacking[89]. For instance, some studies assessed only food intake without considering dimensions such as satiety and food preferences. Additionally, studies have used



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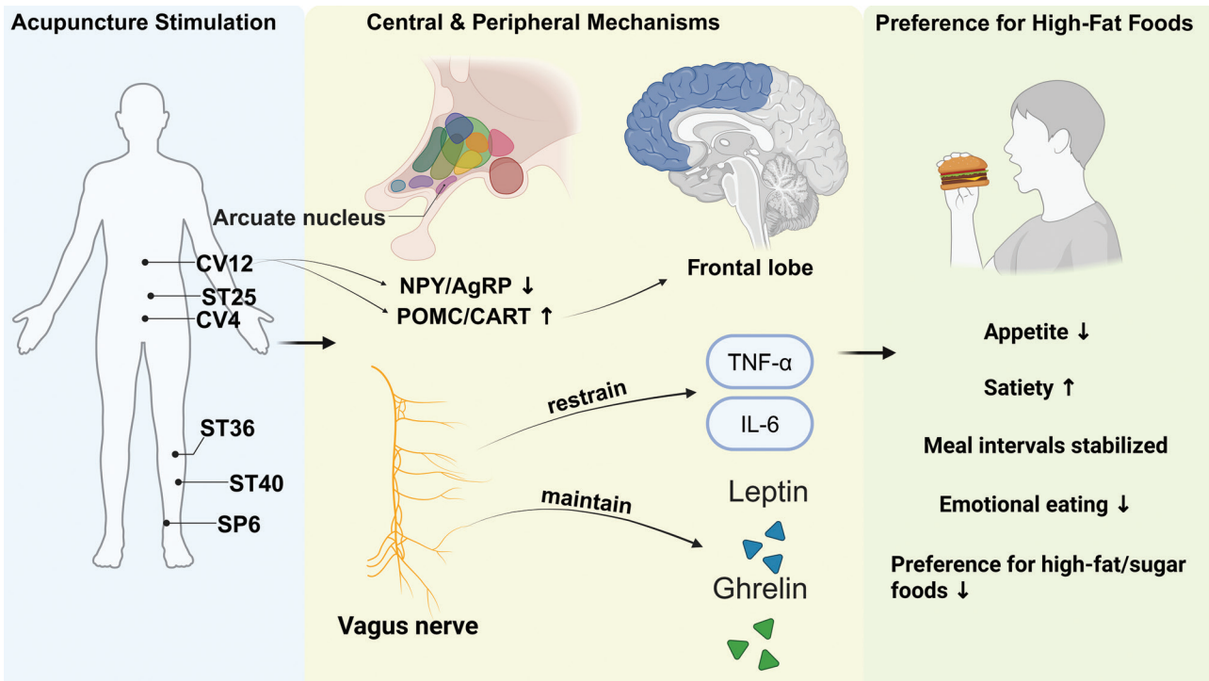



Figure 3. Mechanisms of acupuncture in regulating eating behaviors. Acupuncture at ST36, SP6, and ST25 modulates the central and peripheral pathways to normalize eating behaviors. Centrally, acupuncture rebalances hypothalamic ARC neurons (downregulating NPY/AgRP and upregulating POMC/CART), inhibits amygdala activity, enhances prefrontal cortical function, and modulates the reward system (NAc-VTA), thereby reducing emotional eating and cravings for high-fat and high-sugar foods. Peripherally, acupuncture increases vagal afferent activity, restores leptin/ghrelin homeostasis, and suppresses chronic inflammation (TNF-α and IL-6). These integrated effects lead to reduced appetite, increased satiety, stabilized meal intervals, and decreased emotional eating. AgRP: Agouti-related protein; ARC: Arcuate nucleus; CART: Cocaine- and amphetamine-regulated transcript; IL-6: Interleukin-6; NAc-VTA: Nucleus accumbens-ventral tegmental area; NPY: Neuropeptide Y; POMC: Pro-opiomelanocortin; TNF-α: Tumor necrosis factor-alpha.

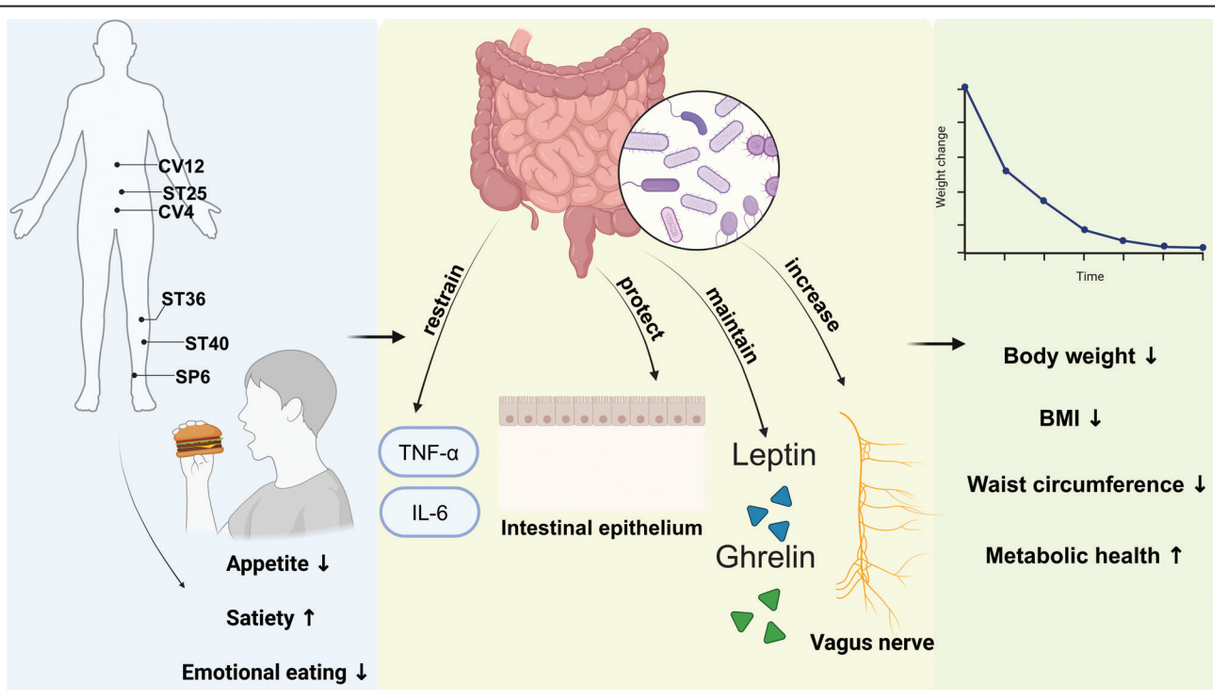



Figure 4. Mechanisms of acupuncture-induced weight loss. Building on normalized eating behaviors (Figure 1), acupuncture promotes weight loss through integrated multisystem mechanisms. Acupuncture restructures the gut microbiota (increases diversity and SCFA production), repairs intestinal barrier integrity, and reduces LPS-induced inflammation. It enhances insulin sensitivity, restores leptin signaling, normalizes ghrelin rhythms, and increases vagal efferent activity. Suppression of chronic inflammatory cytokines (TNF-α and IL-6) alleviates hypothalamic leptin resistance. These coordinated effects lead to sustained reductions in body weight, BMI, waist circumference, and overall metabolic improvements. BMI: Body mass index; IL-6: Interleukin-6; LPS: Lipopolysaccharides; SCFAs: Short-chain fatty acids; TNF-α: Tumor necrosis factor-alpha.



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different versions of scales (eg, FCQ and VAS), making it difficult to conduct pooled analyses of results.

Mechanistic studies face issues such as a disconnect between animal models and clinical scenarios, as well as fragmented molecular mechanisms. Most current animal experiments adopt HFD-induced obesity models, which fail to simulate complex clinical scenarios such as emotional eating. Research on molecular mechanisms mostly focuses on single pathways, such as hypothalamic neurons and gut microbiota, and a systematic analysis of multipathway synergistic regulation involving the CNS, gut microbiota, and vagus nerve is lacking. Moreover, core molecular targets such as key enzymes for acupuncture-regulated SCFA synthesis have not been identified.

Methodological limitations are primarily reflected in the insufficient standardization of sham acupuncture control designs and the difficulty in implementing blinding. Various sham acupuncture controls have been implemented, including non-acupoint acupuncture, shallow needling, and EA without current. There is no unified standard for selecting non-acupoints; therefore, sham acupoints chosen in previous studies may have exerted certain physiological effects. In addition, acupuncture treatment depends on the performance of the practitioner, and achieving complete blinding is difficult. This may introduce assessment bias; for example, patients' trust in acupuncture could influence eating behavior scores.

Meanwhile, clinical and basic research are disconnected; regulatory pathways revealed by basic research are difficult to translate directly into clinical optimization strategies. For instance, the gut microbiota–SCFA–CNS regulatory mechanism identified in animal experiments has not been clearly linked to the logic of acupoint selection in clinical practice, nor has it guided the precise setting of key parameters, such as the EA frequency and needle retention time. Basic research has focused on single molecular targets, such as hypothalamic NPY or beneficial gut bacteria such as *Akkermansia*, but has lacked correlation analysis with clinical eating behavior assessments (eg, FCQ subscales) and therefore fails to provide actionable optimization strategies for improving food preferences *via* acupuncture. This translational gap between basic mechanisms and clinical practice prevents basic research findings from addressing core clinical issues such as sample heterogeneity and therapeutic efficacy stability, restricting the precise development of acupuncture for weight loss.

A structured summary of the main clinical studies, including their design, sample size, intervention, control, outcomes, and key methodological limitations, is provided in Table 1. Notably, the available clinical evidence exhibits considerable heterogeneity in the study design, intervention protocols, control conditions, outcome measures, and follow-up durations. This heterogeneity, together with the methodological limitations outlined in Table 1, underscores the urgent need for future high-quality trials with greater homogeneity in key design elements, such as standardized acupuncture regimens, sham controls, validated eating behavior assessments, and long-term follow-ups, to strengthen the evidence base and facilitate more definitive conclusions.

### *Key directions for future research*

Clinical research should include multicenter, large-sample RCTs and establish a standardized system for assessing eating behaviors. It would be advisable to conduct multicenter RCTs with sample sizes exceeding 500 cases and extend the follow-up durations to 12 to 24 months, with a focus on evaluating the long-term stability of eating behaviors. Meanwhile, the evaluation system should include a comprehensive framework that comprises food intake, satiety VAS, FCQ, and EEQ to objectively monitor eating behaviors.

Mechanistic research could benefit from analyzing core pathways using multi-omics techniques and establishing animal models that are more closely aligned with clinical scenarios. Combining transcriptomics, metabolomics, and microbiomics may help screen core molecular targets, such as SCFA synthases and vagus nerve receptors, that are involved in acupuncture-regulated eating behaviors. Dual-factor obesity models combining HFD and chronic stress can be established to simulate clinical emotional eating scenarios. Additionally, organoid models, such as hypothalamic and intestinal organoids, can be used to verify the direct regulatory effects of acupuncture on eating behaviors.

Translational research should explore personalized acupuncture protocols and develop auxiliary technologies that target eating regulation. Personalized acupoint schemes can be formulated based on eating behavior classifications, such as excessive or emotional eating. For example, calming acupoints such as Neiguan (PC6) and Shenmen (HT7) are used for patients with emotional eating. Portable devices (eg, EA patches) can be developed to improve long-term patient compliance. Furthermore, strategies that combine acupuncture with low-glycemic index diets and exercise should be explored to enhance the synergistic effects on regulating eating behaviors.

## Conclusion

This review synthesized the clinical evidence and mechanistic insights into the efficacy of acupuncture for weight management, with a focus on standardized eating behavior as a pivotal mediator. Clinical studies have suggested that acupuncture may promote weight loss by reducing food intake, enhancing satiety, regulating food preferences, and inhibiting emotional eating, which are mediated by the normalization of eating behaviors. Mechanistically, acupuncture may act through a synergistic multipathway involving the CNS, gut microbiota, and vagus nerve. These effects include restoring the balance of hypothalamic ARC neurons, improving prefrontal cortex–hypothalamus connectivity, remodeling gut microbiota to promote SCFA production and intestinal barrier repair, and enhancing vagus nerve activity to regulate peripheral hormones and suppress chronic inflammation. Despite current limitations, such as small sample sizes and fragmented mechanisms, future research employing multicenter RCTs, multi-omics techniques, and personalized protocols is expected to advance the clinical translation of acupuncture, offering a novel, evidence-based strategy for obesity management to address the global obesity burden and align with precision medicine goals.



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## Conflict of interest statement

The authors declare no conflict of interest.

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## Author contributions

Yun Liu, Mengying Zhao, Siyu Zhuang, and Jiahui Xie were involved in the review of the concept, drafting of the manuscript, editing of the manuscript, and preparing figures of the manuscript. Yun Liu, Yujue Wang, WingYi Ng, Zhi Yu, Mengjiang Lu, and Xinyue Jing were involved in drafting and editing the manuscript. Gerhard Litscher, Guanhu Yang, Tiancheng Xu, and Bin Xu provided linguistic polishing support for the manuscript. Tiancheng Xu finalized the manuscript and confirmed that it represents the integration of relevant medical knowledge and acupuncture research. All authors contributed to the revision of the manuscript and approved the final submitted version.

## Ethical approval of studies and informed consent

Not applicable.

## Acknowledgments

None.

## Data availability

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

## Declaration of generative AI in scientific writing

During the preparation of this work, the authors did not use any generative artificial intelligence (AI) or AI-assisted technologies in the writing, data analysis, figure creation, or content generation process. All authors take full responsibility for the originality, accuracy, and integrity of the manuscript.

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