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Review Article

New perspective on acupuncture treatment for obesity: mechanisms in the brain based on multidimensional neural circuits

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Abstract: Obesity, characterized by pathologic adipose accumulation resulting from an imbalance between energy intake and expenditure, is a major global public health burden. Lifestyle interventions are prone to lead to weight regain, whereas pharmacotherapy and bariatric surgery are constrained by adverse effects or postoperative complications, underscoring an urgent need for safe, effective, and durable strategies. Acupuncture/electroacupuncture can reduce body weight and improve metabolic homeostasis, yet the underlying mechanisms involving central neural circuits remain to be elucidated. This review summarizes evidence that acupuncture modulates appetite through coordinated neuroendocrine actions and whole-brain circuit remodeling, including reprogramming the pro-opiomelanocortin (POMC)/agouti-related protein (AgRP) homeostatic axis and ameliorating leptin resistance; regulating the ventral tegmental area (VTA)-nucleus accumbens (NAc)-prefrontal cortex (PFC) dopaminergic reward pathway to suppress reward-driven feeding; attenuating hyperactivation of the hypothalamic-pituitary-adrenal (HPA) axis and influencing limbic circuitry to relieve stress-induced binge eating; and strengthening prefrontal-hippocampal network connectivity to enhance executive control. Future directions include integrating whole-brain imaging, dynamic monitoring of neurotransmitters and hormones, and causal circuit interrogation to advance efficacy prediction and optimization of individualized protocols.

Keywords: Obesity, acupuncture, appetite regulation, multidimensional neural circuits, dopamine reward system

Introduction

Obesity is typically defined as a body mass index (BMI) ≥ 30 kg/m^2, representing a highly heterogeneous disease state arising from an imbalance between energy intake and energy expenditure [1], and has evolved into a major global public health crisis. Nearly two-fifths of adults worldwide currently have overweight or obesity; by 2060, the economic burden attributable to obesity is projected to reach 3.29% of global gross domestic product (GDP), underscoring its substantial socioeconomic effect [2]. Obesity markedly increases the risk of cardiovascular disease, type 2 diabetes, and cancer, among other disorders [3, 4]. Its key pathophysiologic basis lies in dysregulation of the multilayered central nervous system (CNS) control of energy homeostasis. Specifically, this dysregulation manifests as disrupted homeostatic regulation in the hypothalamic arcuate nucleus (ARC), characterized by hyperactivation of orexigenic agouti-related protein (AgRP) neurons and reduced activity of anorexigenic pro-opiomelanocortin (POMC) neurons [5]; this process may be associated with impaired function of the mesolimbic dopaminergic system that mediates reward and motivation [6, 7]. However, mainstream interventions remain limited: lifestyle interventions are constrained by inadequate long-term adherence and frequent weight regain [8]; pharmacotherapy is often accompanied by gastrointestinal and neuropsychiatric adverse effects [9]; and bariatric sur-

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gery, although effective, is limited by the risk of complications [10]. Therefore, there is an urgent need to develop novel therapeutic strategies that are both safe and effective and that enable sustained long-term weight maintenance.

As a non-pharmacologic therapy, acupuncture has demonstrated favorable clinical efficacy in weight-loss interventions [11]. Studies suggest that acupuncture exerts its effects primarily by neuroendocrine pathways, by modulating obesity-related neural circuits and regulating endocrine functions, thereby producing therapeutic effects [12]. Notably, acupuncture may have the advantage of promoting multi-target neural circuit remodeling [13]. For instance, acupuncture may alleviate hypothalamic leptin resistance and modulate POMC neurons within the ARC [14], and regulate dopamine signaling in the mesolimbic pathway [15]. However, prior studies have largely focused on modulation mechanisms within a single pathway or specific brain regions [16, 17], and a systems-level understanding of the anti-obesity effects of acupuncture remains limited: there is still a lack of in-depth elucidation of how neural circuits responsible for homeostatic regulation, reward processing, emotion regulation, and cognitive control coordinate and interact at the whole-brain level to jointly mediate the anti-obesity effects of acupuncture. Accordingly, this study aims to systematically integrate and elucidate the multilevel, whole-brain coordinated neural mechanisms involved in the anti-obesity effects of acupuncture, with a focus on the interconnected neural dimensions of homeostasis, reward, emotion, and cognitive control.

Relationship between obesity and appetite

The pathophysiologic basis of obesity lies in obesogenic environment-driven dysregulation of appetite control, leading to a persistently imbalanced "appetite-metabolic" state [18, 19]. Feeding behavior is jointly regulated by the homeostatic and hedonic systems [20, 21]. The homeostatic system is centered on the ARC and maintains energy homeostasis through two antagonistic neuronal populations: AgRP/neuropeptide Y (NPY) neurons promote food intake, whereas POMC/cocaine- and amphetamine-regulated transcript (CART) neurons integrate peripheral signals such as leptin and insulin to suppress feeding and relay satiety information by the ARC-paraventricular nucleus (PVN)-parabrachial nucleus (PBN) pathway [22]. The hedonic system is primarily driven by the mesolimbic dopaminergic reward pathway: dopaminergic projections from the ventral tegmental area (VTA) to the nucleus accumbens (NAc) can, through the VTA→NAc→lateral hypothalamic area (LHA) circuit, potentiate motivational craving for highly rewarding foods [23]. Complex crosstalk exists between these two systems: AgRP neurons project to the LHA to enhance hunger motivation, whereas POMC neurons participate in modulating reward value evaluation within the NAc to reduce food intake [24]. Meanwhile, the orbitofrontal cortex, dorsolateral prefrontal cortex, insula, amygdala, and hippocampus further regulate reward-driven feeding by integrating sensory cues, affective states, and mnemonic information [25]. As a key hub, the LHA integrates afferent inputs from the ARC, VTA/NAc, and limbic system and directly participates in the regulation of feeding output. Notably, LHA neurons expressing preproenkephalin can be activated under stress and selectively drive stress-induced binge eating and preference for high-fat, high-sugar foods [26], thereby establishing a "stress-binge eating" positive-feedback circuit that promotes obesity progression. Collectively, coupled dysregulation of impaired homeostatic control and aberrant hedonic/reward processing synergistically drives overeating and constitutes an important mechanistic basis for the onset and persistence of obesity.

Central regulatory network of obesity: pathologic basis of multiregional functional imbalance

Obesity is not attributable to abnormalities in a single brain region; rather, under high-fat diet (HFD) and environmental stress, neural circuits across multiple dimensions-including homeostatic regulation, reward motivation, emotional stress, and cognitive executive control-undergo plastic remodeling, culminating in a cross-regional pathologic network imbalance (**Figure 1**).

ARC: a key integrative node for energy homeostatic regulation

ARC integrates peripheral signals through an antagonistic neuronal network to regulate

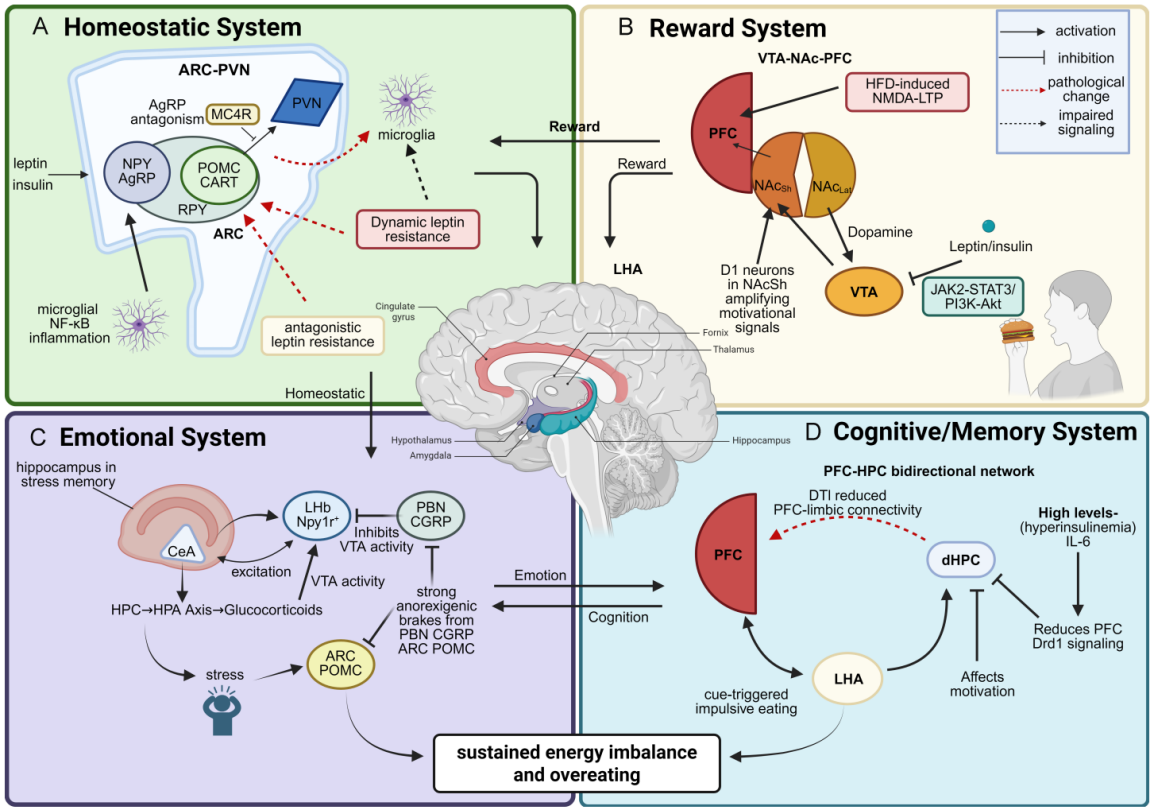

Figure 1. Pathologic basis of obesity-induced functional imbalance across multiple brain regions. A. Homeostatic system: ARC malfunctions, with hunger neurons (NPY/AgRP) becoming overactive. B. Reward system: VTA-NAc is hyperactivated by high-calorie foods, creating addiction-like cravings. C. Emotional system: Stress activates the 'HPA axis', releasing hormones that promote eating for comfort. D. Cognitive/Memory system: The 'prefrontal-hippocampal' network for decision-making is impaired, leading to impulsive eating and poor resistance to cues. PVN: Paraventricular Nucleus; ARC: Arcuate Nucleus; POMC/CART: Pro-opiomelanocortin/Cocaine- and Amphetamine-Regulated Transcript; AGRP/NPY: Agouti-Related Protein/Neuropeptide Y; MC4R: Melanocortin 4 Receptor; LHA: Lateral Hypothalamic Area; VTA: Ventral Tegmental Area; NAc: Nucleus Accumbens; PFC: Prefrontal Cortex; NAcLat: Nucleus Accumbens Lateral Shell; HFD: High-Fat Diet; HPC: Hippocampus.

appetite. Within the ARC, orexigenic NPY/AgRP neurons and anorexigenic POMC/CART neurons receive satiety signals from the gastrointestinal tract, adipokines, and peripheral hormonal signals such as leptin and insulin, and project to downstream nuclei including the PVN, thereby exerting opposing influences on feeding behavior and energy expenditure [27, 28]. Obesity-related pathologic processes in the ARC mainly include: (1) Imbalance of the melanocortin receptor pathway, where AgRP antagonizes melanocortin 4 receptor (MC4R)-mediated signaling to promote feeding [29], whereas MC4R gene knockout leads to increased food intake and the development of obesity [30]. (2) Dynamic evolution of reduced leptin sensitivity-in the early stage of HFD exposure, compensatory enhancement of leptin receptor signaling may, to some extent, delay weight gain; however, with prolonged exposure, POMC system activity declines and central leptin resistance emerges, ultimately resulting in sustained energy imbalance [31]. (3) Activation of inflammatory responses: energy surplus can activate the nuclear factor κB (NF-κB) pathway, inducing proliferation and activation of hypothalamic microglia, thereby further exacerbating leptin resistance [32]. (4) Novel targets and circuit mechanisms-leptin can rapidly inhibit AgRP neuronal activity through Basonuclin 2-expressing neurons within the ARC [33]. Peptide YY can enhance satiety responses through neuropeptide Y receptor type 2 (Y2 receptor)-related signaling, providing a strategy for anti-obesity interventions targeting the gut-hypothalamus axis [34].

Reward system: dopaminergic circuitry from homeostatic feeding to reward-driven overeating

The reward system is mediated primarily by the mesolimbic dopaminergic circuit, with key nodes including VTA, NAc, and prefrontal cortex (PFC). This system integrates signals related to metabolic state and external sensory stimuli, encoding the reward value of food into motivational signals, thereby promoting reward-driven feeding beyond homeostatic intake [35]. These motivational signals can be further amplified in dopamine D1 receptor (Drd1)-expressing neurons in the NAc shell (NAcSh), increasing the propensity to seek and consume highly rewarding foods [36]. The LHA is an important integrative hub linking homeostatic and reward pathways; it converges metabolic-related inputs from the ARC, including projections from AgRP/NPY neurons, and reward-related inputs from the NAc, and cooperatively regulates feeding output through its connections with the nucleus tractus solitarius (NTS) and the VTA [37].

Under pathologic conditions, the dopaminergic circuit can exhibit multi-level plasticity changes and network imbalance: (1) Abnormal dopaminergic signaling-HFD can induce N-methyl-D-aspartate receptor-dependent long-term potentiation in the VTA, thereby enhancing preference for energy-dense foods [38]. Meanwhile, epigenetic modifications in the lateral NAc (NAcLat), including those involving DNA methyltransferase 3A, can suppress the NAcLat→VTA projection pathway [39]. (2) Reward circuit dysfunction-suppression of the NAcLat→VTA pathway leads to dysregulated dopamine release and loss of control over feeding behavior [40]. (3) Aberrant coupling between homeostatic and reward signals-Drd1 receptors expressed by AgRP neurons participate in the induction of compulsive feeding [6]; in addition, leptin and insulin can suppress VTA dopaminergic neuronal activity through the Janus kinase 2 (JAK2)-signal transducer and activator of transcription 3 (STAT3) and phosphoinositide 3-kinase (PI3K)-protein kinase B (Akt) signaling pathways [41]. (4) Novel circuit mechanisms-neurons in the NAcSh expressing serine protease inhibitor B2 can attenuate leptin-mediated inhibitory effects by inhibiting leptin receptor (LepR)-positive γ-aminobutyric acid (GABA)ergic neurons in the LHA [42]; impairment of the glutamatergic projection from the medial prefrontal cortex (mPFC) to the LHA can exacerbate stress-related increases in food intake [7]. Collectively, pathologic remodeling of dopaminergic circuits constitutes an important neural mechanism underlying reward-driven overeating and compulsive feeding in obesity, providing a theoretical basis for circuit-specific precision interventions.

Emotional system: neural basis of limbic system-stress axis-induced feeding

Clinical studies indicate that, in individuals with obesity, anxiety symptoms are positively correlated with intake of energy-dense foods [43]. This association may reflect functional remodeling of the limbic system-stress axis in stress responses and feeding regulation. The neurobiological basis includes: (1) the amygdala participates in integrating fear-related cues and reward motivation, and activation of NPYergic neurons in the medial subdivision of the central amygdala (CeA) can promote overeating under stress conditions [44]; (2) the hippocampus contributes to stress memory formation and can modulate glucocorticoid release via the hypothalamic-pituitary-adrenal (HPA) axis, thereby influencing inflammatory response levels [45]; (3) chronic stress perturbs energy homeostasis and feeding behavior through bidirectional mechanisms: on the one hand, NPY-related pathways enhance feeding drive; on the other hand, neurons in the lateral habenula expressing neuropeptide Y receptor type 1 (Npy1r) can suppress VTA activity, thereby reducing intake of highly palatable foods [44]. Conversely, activation of POMC neurons and calcitonin gene-related peptide-expressing neurons in the PBN, particularly the subpopulation projecting to the CeA, can markedly suppress feeding [46]. In addition, HFD may further remodel this network, accompanied by aberrant regulation of dopamine release in the NAc, thereby promoting anxiety-related overeating and forming a self-reinforcing positive-feedback loop [47].

Cognitive/memory system: executive control impairment mediated by the fronto-hippocampal network

Studies suggest that both the hippocampus and the PFC are key hubs within the brain's

functional network, yet they differ in functional specialization: the hippocampus serves as a driver of whole-brain functional activity, predominating in the dynamic regulation of information flow, whereas the prefrontal cortex acts as an integrative center for whole-brain information, converging diverse sensory signals [48].

The PFC and hippocampus are connected by extensive bidirectional projections, forming a fronto-hippocampal functional network that jointly supports the coupled integration of executive control and memory processes. The PFC can integrate multimodal cues such as olfactory and visual inputs and, through functional coupling with the NAc, participates in the representation of feeding-related decisions and the maintenance of goal-directed behavior [49, 50]. The hippocampus plays a dominant role in episodic and spatial memory, and also participates in the processing of feeding-related contextual cues and motivation regulation. Studies have reported that specific neuronal populations can encode food-related spatial cues and their reward associations, and influence feeding motivation by connectivity between the LHA and the dorsal hippocampus (dHPC) [51]. The cooperative action of these regions can regulate feeding behavior by coordinating cognitive control and memory retrieval processes.

Within neural circuits related to reward, emotion, and cognitive control, repeated observations have demonstrated differences in brain activity between obese and non-obese individuals; meanwhile, the oral feeding process can also modulate neural activity patterns in these circuits [52]. Diffusion tensor imaging studies show that white matter structural connectivity between the PFC and the limbic system is reduced in individuals with obesity, thereby leading to decreased cognitive flexibility [53]. Moreover, HFD can induce hyperinsulinemia and be accompanied by elevated levels of inflammatory factors such as interleukin-6 (IL-6), thereby impairing hippocampal synaptic plasticity and reducing Drd1-related signaling activity within the PFC [54, 55]. Other studies have indicated that the dHPC contains orexigenic neuronal subpopulations that are selectively sensitive to lipid and sugar signals; these neurons may participate in the regulation of dietary choice by influencing spatial memory representations and feeding motivation, among other mechanisms [56]. Overall, environmental stress and HFD can both damage the fronto-hippocampal network, leading to memory bias and weakened executive control, thereby increasing the risk of cue-triggered impulsive feeding and potentially establishing a self-reinforcing cycle among metabolic dysfunction, neuroinflammation, and cognitive impairment.

Mechanistic exploration of acupuncture-mediated appetite modulation for the treatment of obesity

In obesity, imbalance of the central regulatory network is mainly manifested as hypothalamic dysfunction, hyperexcitability of the reward system, dysregulated emotional processing and stress responses, and impaired cognitive control. Through multidimensional modulation of CNS neurotransmission, gastrointestinal function, and the endocrine system, acupuncture enables targeted intervention in appetite regulation, thereby ameliorating obesity-associated disturbances in energy metabolism (**Figure 2**). The following sections delineate these mechanisms from the perspectives of neuromodulation, gut hormones, activity changes in central nuclei, and integration of the neuroendocrine-immune network.

Homeostatic system remodeling: targeted modulation of hypothalamic energy balance by acupuncture needling

As the central regulatory hub for whole-body energy homeostasis, the hypothalamus coordinates energy intake and expenditure by integrating afferent inputs from the brainstem and peripheral tissues such as adipose tissue, the pancreas, and the stomach. When acupuncture needles stimulate acupoints, this local mechanical stimulation can activate somatosensory afferent nerve fibers; signals ascend through the spinal cord and brainstem into the hypothalamic energy-homeostasis network, thereby inducing plastic changes in neuronal activity within specific nuclei. Experimental studies show that electroacupuncture at Zusanli (ST36) and Sanyinjiao (SP6) enhances melanocortin system activity in ARC, characterized by increased α-melanocyte-stimulating hormone (α-MSH) release and enhanced POMC neuronal function, accompanied by feeding-suppres-

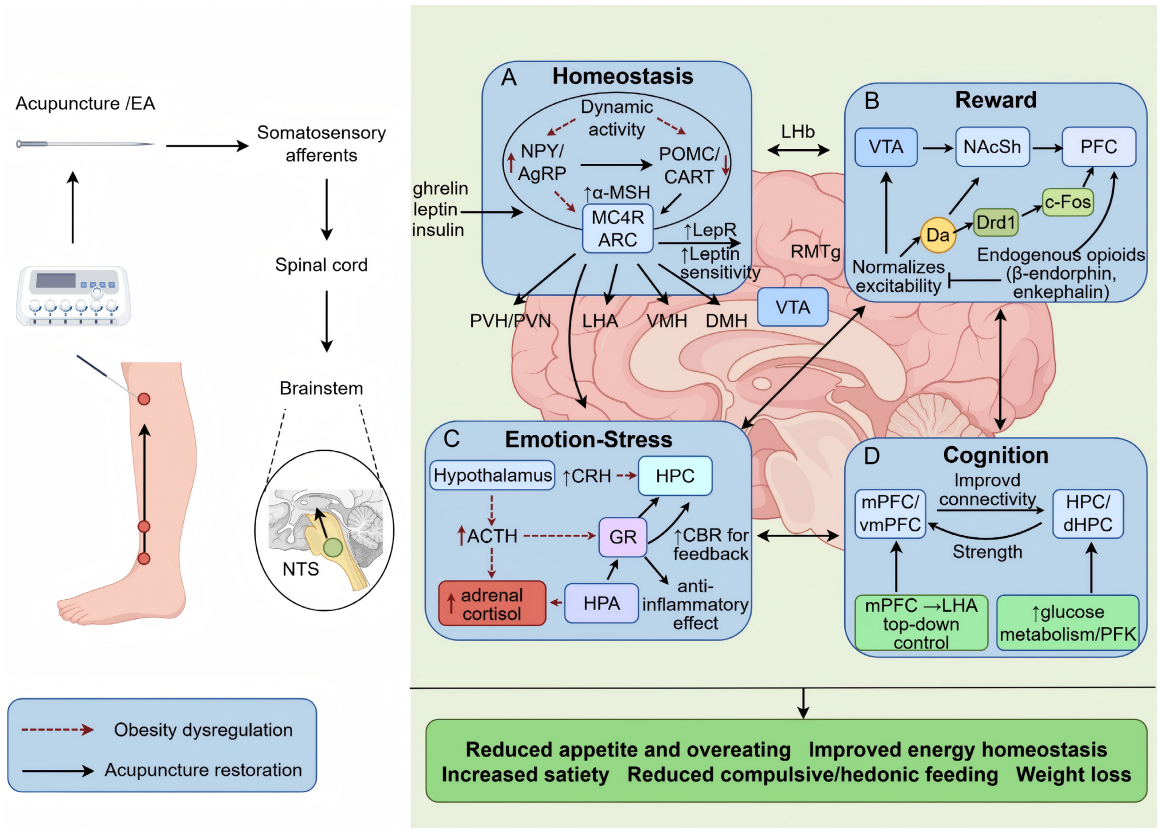

Figure 2. Acupuncture intervention in systemic dimensions: homeostasis, reward, emotional stress, and cognitive control. A. ARC homeostasis; B. Reward circuit; C. Emotion/stress; D. Cognitive control axis. NPY/AgRP: Neuropeptide Y/Agouti-Related Protein; POMC/CART: Proopiomelanocortin/Cocaine- and Amphetamine-Regulated Transcript; MC4R: Melanocortin Receptor 4; ARC: Arcuate Nucleus; LepR: Leptin Receptor; VTA: Ventral Tegmental Area; PFC: Prefrontal Cortex; NAc: Nucleus Accumbens; DA: Dopamine; LHA: Lateral Hypothalamic Area; DMH: Dorsomedial Hypothalamic Nucleus; mPFC: Medial Prefrontal Cortex; vmPFC: Ventromedial Prefrontal Cortex.

sive effects [57]. In addition, acupuncture may promote lipid metabolism and weight loss by inhibiting the production of leptin and prostaglandin E [58]. Acupuncture needling not only reduces leptin secretion but also upregulates LepR expression, thereby enhancing leptin sensitivity [59]. Needling-induced changes in neurotransmitter/hormonal signaling are coupled with the activity of antagonistic neuronal populations within the ARC, including inhibition of NPY/AgRP neurons and activation of POMC/CART neurons, and may act in concert with rebalancing of feeding-related hormonal signals such as ghrelin, thereby shifting energy intake and metabolic regulation toward restoration of homeostasis. Mechanistically, altered ARC neuronal activity can influence feeding output through downstream projection networks: NPY/AgRP neurons project to the LHA, ventromedial hypothalamus (VMH), dorsomedial hypothalamus (DMH), and PVN to promote feeding, whereas POMC/CART neurons relay satiety information and suppress feeding through pathways involving the VMH, DMH, NTS, and PVN [14]. Collectively, acupuncture needling can remodel hypothalamic control of energy homeostasis through a multilevel cascade-peripheral sensory afferents, activity of key hypothalamic nuclei, neuropeptide/hormonal signaling, and downstream circuit outputs-thereby providing a neurobiological basis for needling-based intervention in obesity and other metabolic disorders.

Rebalancing of the reward system: dynamic modulation of dopaminergic pathways by acupuncture needling

Acupuncture rebalances the reward system by modulating the mesolimbic dopaminergic circuit (VTA⟶NAc⟶PFC) and its associated neu-

ral networks. At the peripheral level, needling stimulation can activate small myelinated nerve fibers in muscle; impulses ascend through the spinal cord, further eliciting neuronal activity changes in midbrain and hypothalamus-pituitary-related centers and triggering the release of three endogenous opioid peptides (enkephalins, β-endorphin, and dynorphin) as well as other monoaminergic mediators, thereby exerting broad neuromodulatory effects on reward pathways [60]. At the central circuit level, needling at Taichong (LR3) can upregulate c-Fos expression in the NAc shell [61]; in a mouse model of atopic dermatitis, needling at Quchi (LI11) can increase tyrosine hydroxylase and Drd1 protein expression in reward-related regions including the NAc, dorsolateral striatum, and the VTA [62]. Moreover, needling at Shenmen (HT7) and Zusanli (ST36) can attenuate cocaine-induced hyperexcitability of VTA dopaminergic neurons, thereby reducing reward signal transmission associated with drug- or food-related cues [63]. Needling can also suppress endogenous opioid peptide inputs and dopaminergic neuronal activity within circuits upstream of the NAc and VTA, thereby improving substance dependence-related motor abnormalities [64]. Mechanistic studies further suggest that acupuncture-evoked afferent signals may recruit the lateral habenula-rostromedial tegmental nucleus circuit through peripheral tactile pathways, thereby exerting inhibitory control over dopaminergic neurons [65]. Collectively, acupuncture promotes reward-system rebalancing by dynamically modulating neuronal activity within the mesolimbic dopaminergic system, suppressing dopamine release in the VTA, and reshaping receptor balance and neurotransmitter interactions within the NAc. Such modulation not only helps alleviate addictive behaviors but also aids in obesity treatment by suppressing overeating and improving energy homeostasis.

Emotional-stress axis modulation: integrative effects of acupuncture needling on the limbic system

Neuroendocrine regulation and autonomic nervous system responses that link obesity and stress are important interventional targets for stress-related obesity. After needling stimulation activates peripheral somatosensory afferents, impulses ascend through the spinal cord and brainstem and can influence neuronal activity in relevant nuclei including the hypothalamus and limbic system, thereby treating obesity and affective disorders by mechanisms such as rebalancing neurotransmission, enhancing neuroplasticity, attenuating inflammatory responses, and modulating neuroendocrine function [66]. Available evidence indicates that acupuncture needling can suppress hyperactivation of the HPA axis, which is considered a key mechanism underlying its therapeutic effects on mood disorders and obesity [67]. In addition, acupuncture can increase glucocorticoid receptor (GR) protein expression, inhibit hypothalamic corticotropin-releasing hormone (CRH) secretion, and thereby reduce adrenocorticotropic hormone (ACTH) and glucocorticoid levels, attenuating the stress response [68]. Further studies suggest that acupuncture needling may modulate HPA axis function by regulating key components such as hypothalamic CRH, GR, and cortisol, and promote the release of anti-inflammatory cytokines, thereby alleviating peripheral and central inflammatory responses [69, 70]. Electroacupuncture at Sanyinjiao (SP6), Zhubin (KI9), Qimen (LR14), and Shenshu (BL23) can effectively modulate HPA axis function [71].

Beyond the HPA axis, needling can also influence limbic circuits involved in reward and anti-reward processing. Reward networks with the VTA as a key node play an important role in the onset and progression of mood disorders such as depression. Studies show that stimulation at Zusanli (ST36) and Yanglingquan (GB34) can enhance dopamine synthesis and release within the amygdala and activate related signal transduction processes, accompanied by improvement of depression-like negative affect [72]. In summary, the effects of acupuncture needling on emotion- and feeding-related phenotypes may involve both endocrine modulation of the HPA axis and remodeling of dopaminergic signaling within the limbic system. Their coordinated changes may jointly contribute to stress-induced regulation of feeding, alleviating chronic stress-related overeating while mitigating appetite enhancement associated with emotional fluctuations, ultimately improving energy homeostasis and metabolic status. On this basis, integrated modulation of the emotion-appetite-metabolism pathway by acupuncture needling may constitute a mechanistic

foundation for its intervention in psychosomatic or stress-related obesity.

Cognitive system restoration: enhancing effects of acupuncture needling on the fronto-hippocampal network

Individuals with obesity often exhibit reduced prefrontal cortical gray matter volume, which is associated with cognitive impairment [74]. Acupuncture stimulation can increase regional cerebral blood flow and metabolic activity in the hippocampus and improve functional connectivity between the hippocampus and other brain regions [73]. With respect to improvement of cognitive impairment, the potential mechanisms of acupuncture needling involve multiple processes, including attenuation of synaptic and neuronal functional damage, suppression of neuroinflammatory responses, enhancement of cerebral energy metabolism, and promotion of functional restoration of neural circuits [75]. At the circuit level, connectivity between the mPFC and the LHA is considered to participate in cortical regulation of feeding behavior and motivational reward seeking; modulation of this pathway by the mPFC is associated with feeding suppression and reduced motivational drive. Under mild stress conditions, inhibition of this pathway can increase food intake and motivational behaviors, whereas chronic lesioning is associated with weight gain [76]. At the metabolic level, electroacupuncture can upregulate phosphofructokinase activity in the hippocampus and ameliorate aberrant glucose metabolism, thereby enhancing glycolytic capacity in cognition-related brain regions and increasing glucose metabolic activity in the hippocampus, hypothalamus, frontal lobe, and temporal lobe, ultimately improving memory function [77]. In addition, acupuncture needling can inhibit activation of glutamatergic neurons in the ventromedial PFC; given the critical role of the ventromedial PFC in attention, decision-making, and cognitive control, this effect may be related to improvement in executive control-related phenotypes [78]. These multi-level mechanisms suggest that acupuncture needling may achieve comprehensive regulation of appetite by repairing stress-sensitive neural circuits, resetting metabolic systems, and rebalancing excitatory/inhibitory neurotransmitter networks, thereby offering a novel interventional strategy for obesity and compulsive binge eating.

Summary and perspectives

Obesity poses a serious health threat; although patients often attempt to lose weight through lifestyle modification, many experience repeated relapse and weight regain. The use of western anti-obesity pharmacotherapies is limited by adverse effects, including mood changes, suicidal ideation, and gastrointestinal or cardiovascular complications [79]. Therapeutic modalities in traditional Chinese medicine include acupuncture, electroacupuncture, moxibustion, and other interventions, and have been shown to be effective in treating a range of neurological disorders [80]. However, most prior studies have been confined to modulatory mechanisms within a single pathway or specific brain regions. The novel perspective on acupuncture needling for obesity proposed in this work primarily focuses on central regulatory mechanisms involving multidimensional neural circuits, thereby distinguishing it from previous studies that emphasized single-pathway systems.

The most commonly used techniques for investigating desire-related processes in the human brain include neurocognitive testing and functional magnetic resonance imaging (fMRI). fMRI has been widely applied to study the neurophysiologic mechanisms of acupuncture, enabling noninvasive observation of brain processes associated with acupuncture [81]. Studies have shown that resting-state functional connectivity of the insula differs between preprandial and postprandial states: in the fasting state, insula-frontal cortical connectivity is stronger, whereas in the sated state, insula connectivity with default-mode regions is stronger [82]. A significant enhancement of hippocampal, amygdalar, and insular function following acupuncture needling can effectively alleviate mild cognitive impairment [83]. In addition, acupuncture needling can strengthen coordination between bilateral sensorimotor networks, activate the contralateral sensorimotor network, and modulate executive control deficits in the affected hemisphere [84]. With respect to emotion regulation, acupuncture treatment may alleviate symptoms by stimulating limbic structures such as the amygdala and anterior cingulate cortex [85]. Reduced functional connectivity between the hypothalamus and thalamus after acupuncture needling is negatively correlated with the needling-induced

modulation of hunger and can effectively restrain reward-driven overeating [86].

Within the "multidimensional neural network" framework established in this study, the anti-obesity effects of acupuncture needling are more likely to arise from coordinated remodeling across system dimensions including homeostatic regulation, reward processing, emotional stress, and cognitive control, rather than isolated modulation of a single pathway: on the one hand, suppression of HPA axis hyperactivation by needling (accompanied by reduced CRH/ACTH and glucocorticoid levels and increased GR expression) may reduce stress load and, potentially by influencing limbic system- and NAc-associated dopaminergic signaling, attenuate reward-driven feeding tendencies under stress. Concurrently, needling-induced anti-inflammatory effects may couple with improved hypothalamic leptin sensitivity, thereby reinforcing restoration of energy homeostasis mediated by the antagonistic POMC and NPY/AgRP network within the ARC. On the other hand, enhancement of metabolism and functional connectivity within the fronto-hippocampal network may strengthen executive control and augment top-down cortical regulation of feeding-output circuits, including the mPFC-LHA pathway, thereby suppressing reward cue-triggered impulsive feeding and indirectly reducing the translation of hypothalamic hunger signals into behavioral output. Future studies should integrate whole-brain network imaging, dynamic monitoring of neurotransmitters and neuroendocrine measures, and causal circuit validation within a unified experimental and analytical framework to systematically delineate the coupling mechanisms among the HPA axis, dopaminergic reward network, hypothalamic homeostatic circuits, and prefrontal cognitive control network, thereby more fully capturing the system-level features of acupuncture needling interventions and promoting efficacy prediction and optimizing individualized protocols.

Although the evidence synthesized in this work indicates that acupuncture can comprehensively modulate feeding behavior through multilevel mechanisms involving regulation of energy homeostasis, reward circuits, emotional state, and cognitive function, this complexity poses major challenges for elucidating its coordinated regulatory mechanisms and defining the specific contributions of brain region-specific circuits to appetite regulation. Future research should prioritize the following directions: (1) integrating neural circuit analytical techniques, such as viral tracing and *in vivo* calcium imaging, to dynamically monitor and resolve activity patterns of key circuits under acupuncture intervention; (2) combining multi-omics approaches, such as spatial transcriptomics and spatial metabolomics, to precisely identify synergistic regulatory targets and to construct computational models for quantifying circuit synergy effects, such as a "circuit synergy index"; (3) developing individualized acupuncture protocols guided by real-time neurofeedback to drive a shift in research paradigms from reductionist studies targeting single pathways toward system-level network-based investigations. Moreover, existing animal models of obesity often fail to fully recapitulate the complex pathologic features of human obesity, including the influences of psychosocial factors and higher-order cognitive functions [87]. Therefore, future studies are needed.

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Disclosure of conflict of interest

None.

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