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Neuro-Adipose Signaling Pathways: How Brain–Adipose Communication Breakdowns Lead to Diabesity

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ABSTRACT

Adipose tissue and the brain are linked by dense neuroendocrine circuits that continuously coordinate appetite, energy expenditure, thermogenesis and glucose–lipid handling. This bidirectional "brain–fat axis" uses sympathetic and sensory nerves, together with circulating adipokines and gut-derived signals, to match fuel availability with storage and oxidation. In obesity, this communication progressively fails. Hypothalamic and brainstem networks that normally integrate leptin, insulin and nutrient cues become inflamed and resistant; sympathetic and sensory innervation of white and brown adipose tissue (WAT, BAT) is remodeled; and adipose-derived endocrine and immune signals feed back to further impair central control. The result is a self-reinforcing state of "diabesity," where chronic positive energy balance, adipose dysfunction and type 2 diabetes (T2D) become tightly coupled.This review frames diabesity as a disorder of neuro–adipose signaling. First, we outline the anatomical and molecular architecture of brain–adipose circuits, emphasizing sympathetic efferents, emerging roles of PIEZO2-positive sensory afferents and key adipokine pathways. We then summarize how hypothalamic and brainstem nuclei use these channels to regulate lipolysis, adipogenesis, browning and thermogenesis in WAT and BAT. Next, we examine how obesogenic diets, chronic overnutrition and stress trigger hypothalamic inflammation, leptin and insulin resistance, and degeneration or functional impairment of adipose innervation. These changes uncouple caloric intake from expenditure and disrupt adipose endocrine outputs, promoting ectopic lipid deposition, insulin resistance and β-cell stress. Finally, we discuss emerging strategies to restore brain–adipose communication, including weight loss, structured sleep and circadian alignment, pharmacologic leptin and GLP-1–based therapies, and experimental neuromodulation of sympathetic outflow or adipose sensory circuits. We highlight open questions around human adipose innervation, sex- and depot-specific neuro–adipose wiring, and how to integrate neurobiological measures into precision care for obesity-related T2D.

Keywords: neuro–adipose axis; sympathetic innervation; hypothalamic inflammation; leptin resistance; diabesity

INTRODUCTION

Energy balance and glucose homeostasis are not merely the sum of local tissue processes; they emerge from a coordinated conversation between the central nervous system and peripheral metabolic organs. The hypothalamus and brainstem integrate sensory, hormonal and nutrient signals that report on energy stores, acute nutrient inflow and environmental demands. In turn, these nuclei control behavior and autonomic outflow to direct when we eat, how much we move and how adipose tissue stores or burns energy[1,2-5].

Adipose tissue is a central node in this system. White adipose depots buffer energy excess by storing triglycerides in adipocytes and releasing fatty acids during fasting. Brown adipose tissue and beige adipocytes dissipate energy as heat and act as high-capacity sinks for glucose and lipids, thereby improving systemic insulin sensitivity. Both WAT and BAT are richly innervated by sympathetic efferent fibers, and more recently recognized sensory afferents, providing a direct neural channel for brain–fat communication in addition to endocrine signaling via adipokines such as leptin and adiponectin[2–4,6-9].

On the efferent side, pre-autonomic neurons in the hypothalamus (notably within the arcuate nucleus, paraventricular nucleus and lateral hypothalamus) and brainstem send descending projections to sympathetic preganglionic neurons. These preganglionic neurons synapse on sympathetic ganglia whose postganglionic fibers innervate WAT and BAT. Norepinephrine released from these terminals regulates lipolysis, adipogenesis, blood flow and thermogenesis by engaging β-adrenergic and other receptors on adipocytes and stromal cells.

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In BAT and beige fat, this pathway controls UCP1-mediated thermogenesis and broader oxidative programs that influence whole-body energy expenditure and glycemic control[5,10-14]. On the afferent side, adipose tissue sends information back to the brain through circulating hormones and metabolites, and via somatosensory and vagal afferent fibers that detect mechanical and chemical cues. Recent work has identified PIEZO2-expressing sensory neurons that innervate adipose depots and respond to mechanical properties such as tissue distension, providing a neural channel through which fat pads can signal their filling state and modulate sympathetic outflow in a feedback loop[6,15-20].

Leptin epitomizes endocrine communication in this axis. Secreted in proportion to fat mass, leptin acts on hypothalamic POMC and AgRP neurons to inhibit appetite and stimulate energy expenditure, partly by increasing sympathetic drive to BAT and WAT. In parallel, adiponectin, resistin, and other adipokines influence hypothalamic inflammation, insulin and glucose sensing, and autonomic tone[7, 8,21-25]. Gut hormones (GLP-1, PYY, ghrelin) and pancreatic signals such as insulin and amylin converge on overlapping hypothalamic and brainstem circuits, shaping the overall pattern of brain–adipose communication[9,26-30]. Under healthy conditions, this network maintains a "set domain" for body weight and glycemia, with flexibility around a stable mean rather than a rigid set point. When caloric intake increases transiently, leptin and insulin rise, hypothalamic melanocortin signaling ramps up, sympathetic outflow to thermogenic fat and other tissues increases, and energy expenditure is boosted to limit weight gain. Conversely, during negative energy balance, reduced leptin and insulin signal fuel scarcity; orexigenic AgRP/NPY neurons are activated while catabolic POMC tone falls, feeding is stimulated, and sympathetic tone to adipose tissue is reduced, favoring fat conservation[9,31-36].

Diabesity reflects a chronic breakdown of this adaptive dialogue. Persistent intake of energy-dense, highly palatable diets, often in the context of circadian disruption and stress, induces early inflammatory and oxidative responses in the hypothalamus. Within days of high-fat feeding, microglia and astrocytes in the mediobasal hypothalamus become activated, and inflammatory signaling via JNK and IKKβ is engaged, even before substantial weight gain[10,37-43]. This inflammation impairs leptin and insulin signaling in key energy-regulating nuclei, blunting anorexigenic responses and uncoupling caloric intake from energy expenditure. As a result, higher levels of fat mass and leptin are required to generate the same hypothalamic response, a state of central leptin resistance that stabilizes the system at a higher weight[11,44-47].

At the same time, obesity reshapes adipose tissue itself. WAT becomes hypertrophic, hypoxic and inflamed, with altered adipokine profiles and increased release of free fatty acids and extracellular vesicles that feed back on brain circuits. BAT and beige depots lose thermogenic capacity, often due in part to altered sympathetic innervation and local inflammatory remodeling[12,48-53]. Sensory feedback from adipose tissue may become distorted as PIEZO2-positive afferents and other sensory fibers undergo structural and functional changes, further compromising closed-loop control[13,54-57]. This multi-layered disruption transforms the brain–adipose axis from a stabilizing network into one that reinforces excess fat storage, reduced thermogenesis and impaired glucose regulation. Hyperphagia persists despite abundant energy stores; sympathetic tone to BAT is inadequate or mistimed; WAT lipolysis is dysregulated and releases lipids at inappropriate times; and inflammatory adipokines and adipose-derived EVs amplify hypothalamic inflammation and systemic insulin resistance[14,58-63].

The remainder of this article explores these processes in detail. Section 2 describes the anatomical and molecular wiring of neuro–adipose pathways. Section 3 examines how hypothalamic and brainstem circuits use this wiring to regulate adipose function. Section 4 explains how obesogenic environments and genetic predisposition disturb these pathways, creating diabesity. Section 5 links neuro–adipose dysfunction to systemic insulin resistance and T2D. Section 6 reviews therapies that partially restore brain–adipose communication.

2. Anatomical and Molecular Architecture of Neuro–Adipose Circuits

White, brown and beige adipose tissues are richly innervated by sympathetic and sensory fibers. Sympathetic efferents originate from preganglionic neurons in the intermediolateral column of the spinal cord, which receive input from hypothalamic and brainstem nuclei such as the arcuate nucleus, paraventricular nucleus, dorsomedial hypothalamus and rostral medullary raphe. These preganglionic neurons synapse onto postganglionic neurons projecting to WAT and BAT, where they release norepinephrine to regulate lipolysis, blood flow and thermogenesis[15, 64-69]. Anatomical tracing and optogenetic studies have delineated hypothalamic–BAT circuits in rodents, showing that POMC neurons, certain GABAergic neurons and brainstem autonomic centers converge on spinal sympathetic networks that innervate interscapular BAT and beige depots. Activation of these pathways increases UCP1 expression, mitochondrial respiration and energy expenditure, while inhibition promotes positive energy balance[15, 70-76].

Sensory innervation of adipose tissue has only recently come into focus. Adipose depots are innervated by somatosensory fibers that course alongside sympathetic fibers and blood vessels. These afferents project to dorsal root ganglia and higher centers and express markers such as CGRP, TRPV1 and, notably, the mechanosensitive ion channel PIEZO2. Genetic and chemogenetic manipulation of PIEZO2-positive adipose sensory neurons in mice reveals that they sense mechanical changes such as tissue expansion and modulate

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sympathetic output, acting as a local feedback controller that restrains excessive sympathetic drive and influences lipolysis and browning[16, 77-84].

On the molecular side, leptin and insulin are the primary adiposity and nutrient signals to the brain. Leptin receptors are highly expressed in POMC and AgRP neurons, as well as in other hypothalamic and extra-hypothalamic populations. Binding of leptin activates JAK2–STAT3 and PI3K pathways, altering transcription of neuropeptides and acutely modulating neuronal firing. Insulin receptors in similar regions provide a partially overlapping but distinct signal of nutrient status, influencing sympathetic outflow and hepatic glucose production[7, 17,85-86]. Adiponectin, resistin, visfatin, chemerin and other adipokines act on central and peripheral targets, modulating hypothalamic inflammation, AMPK activity, and autonomic regulation. Adipose-derived cytokines such as TNF-α and IL-6 can cross or signal across the blood–brain barrier and affect microglia and astrocytes, linking adipose inflammation to hypothalamic signaling changes[18, 87-93].

Together, these neural and hormonal pathways create a dense, reciprocal network. The brain sends pattern-coded sympathetic commands to adipose depots, while adipose tissue returns information via sensory fibers and endocrine outputs. Disruption at any node, such as nerve terminals, sensory receptors, adipokine secretion, or receptor signalling, can degrade the fidelity of brain–fat communication and contribute to metabolic disease[12, 19,94-97].

3. Central Control of Appetite, Sympathetic Outflow and Adipose Metabolism

Within the hypothalamus, the arcuate nucleus integrates peripheral signals of energy status. POMC/CART neurons respond to leptin, insulin and GLP-1 by increasing firing and releasing α-MSH onto melanocortin 4 receptor (MC4R)–expressing neurons in the paraventricular and other nuclei. This melanocortin pathway suppresses food intake and increases sympathetic outflow to BAT and WAT, enhancing thermogenesis and lipolysis. In contrast, AgRP/NPY neurons are activated by fasting, ghrelin and low leptin; they inhibit POMC neurons and MC4R targets, stimulate feeding, and reduce sympathetic tone[20]. These circuits project to downstream autonomic centers such as the paraventricular nucleus, lateral hypothalamus, dorsal vagal complex and rostral ventrolateral medulla, which in turn control sympathetic preganglionic neurons in the spinal cord. In response to cold exposure or overfeeding, melanocortin and related circuits increase sympathetic tone to BAT, stimulating thermogenesis and glucose and lipid uptake; they also adjust sympathetic drive to WAT to modulate lipolysis and adipogenesis[20, 21].

Brainstem centers integrate gut-derived satiety and nutrient signals with hypothalamic cues. The nucleus tractus solitarius receives vagal afferents conveying information about gastric distension and nutrient content, and is modulated by circulating GLP-1, PYY and CCK. These signals influence feeding behavior and autonomic outflow, including sympathetic drive to adipose tissue[22]. Under healthy conditions, the timing and magnitude of this central control are tuned to the sleep–wake and feeding–fasting cycles. During the active phase, increased sympathetic tone to BAT and higher melanocortin activity promote thermogenesis and nutrient disposal; during the rest phase, sympathetic drive to adipose tissue is reduced, allowing for efficient storage and limiting unnecessary heat production[22, 23].

This framework reveals how disruptions in central circuits through inflammation, genetic mutations in MC4R or leptin pathways, stress or sleep and circadian disturbances can simultaneously affect appetite and adipose metabolism. Hyperphagia, reduced BAT activation and inappropriate WAT lipolysis are not independent phenomena but different manifestations of impaired central command over adipose tissue[22].

4. Neuro–Adipose Communication Breakdowns in Obesity

Obesity emerges when environmental pressures overwhelm the resilience of brain–adipose circuits. High-fat, high-sugar diets rapidly trigger hypothalamic inflammation, characterized by activation of microglia, astrocyte proliferation and engagement of inflammatory kinases such as JNK and IKKβ in POMC and AgRP neurons[24]. This inflammatory milieu impairs leptin and insulin signal transduction, leading to central leptin and insulin resistance. As a consequence, rising adiposity and circulating leptin no longer produce appropriate anorexigenic and thermogenic responses. Adipose tissue itself becomes an active driver of this process. Hypertrophic adipocytes in WAT secrete increased leptin but also more pro-inflammatory adipokines and cytokines such as TNF-α, IL-6 and resistin, which can access the hypothalamus and further promote glial activation and neuronal stress[7]. Adipose-derived extracellular vesicles and lipids add to this inflammatory load, altering neuronal membrane composition and gene expression.

At the level of neural wiring, obesity and overnutrition induce structural and functional changes in adipose innervation. Studies using denervation, viral tracing and advanced imaging show that chronic high-fat feeding reduces sympathetic fiber density in certain WAT depots, alters neurotransmitter receptor expression on adipocytes, and modifies neurovascular coupling. BAT innervation can become blunted, contributing to thermogenic failure[25–27]. Sensory innervation is also affected. Recent work reveals that obesity alters the density and mechanosensitivity of PIEZO2-positive adipose sensory neurons, degrading the fidelity of afferent feedback on tissue expansion and metabolic state. This may lead to inappropriate suppression or activation of sympathetic outflow, further uncoupling adipose behavior from central needs[28–30].

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At the systems level, these changes result in a new, maladaptive homeostatic state. Increased adiposity is defended by altered hypothalamic circuits that perceive weight loss attempts as threat, driving hunger and reducing energy expenditure. Sympathetic drive to WAT and BAT is either inappropriately low or chronically dysregulated, supporting fat retention and diminished thermogenesis. Adipose-derived inflammatory and endocrine signals keep hypothalamic inflammation active, sustaining central resistance to leptin and insulin. This feedback loop defines the neurobiological core of diabesity[31, 32].

5. From Brain–Adipose Dysregulation to Systemic Insulin Resistance and Type 2 Diabetes

Disrupted neuro–adipose communication drives T2D through multiple, converging pathways. Central leptin and insulin resistance, together with altered reward and cognitive control circuits, promote chronic overeating of energy-dense foods. Caloric surplus amplifies adipose hypertrophy and inflammation, leading to increased lipolysis and flux of free fatty acids to liver and muscle[33]. Sympathetic misregulation of WAT and BAT contributes to both energy imbalance and glucose dysregulation. Blunted sympathetic drive to BAT reduces thermogenic energy expenditure and glucose and lipid clearance, promoting ectopic fat accumulation in liver and muscle. Impaired or mistimed sympathetic control of WAT can result in elevated nocturnal lipolysis and free fatty acid release, exacerbating hepatic steatosis and interfering with insulin signaling via DAG–PKC and ceramide pathways[34].

Hypothalamic inflammation and altered autonomic output also affect hepatic glucose production. Normally, insulin acting in the hypothalamus suppresses hepatic gluconeogenesis via a neural relay; in obesity, hypothalamic insulin resistance blunts this pathway, contributing to elevated fasting glucose and a flattened diurnal rhythm of hepatic glucose output[35]. Peripheral insulin-sensitive tissues are not passive recipients. Skeletal muscle insulin resistance, partly driven by ectopic lipid deposition, further reduces postprandial glucose disposal, forcing β-cells to secrete more insulin. The resulting hyperinsulinemia may worsen sympathetic imbalance and central insulin resistance, closing another vicious loop. Over time, β-cells exposed to chronic glucolipotoxic stress and altered central signals (including changes in autonomic innervation of the pancreas) lose functional mass, tipping compensated insulin resistance into overt T2D[11, 35].

Thus, diabesity is not simply "obesity plus diabetes" but a neuro-metabolic integrative state in which brain–adipose communication breakdowns are central drivers of both excessive fat storage and defective glucose control.

6. Restoring Brain–Adipose Communication: Lifestyle, Pharmacologic and Neuromodulatory Approaches

Because neuro–adipose circuits are plastic, at least partly, diabesity is modifiable. Weight loss via caloric restriction, increased physical activity or bariatric surgery reduces adipose inflammation, improves adipokine profiles and partially reverses hypothalamic gliosis and inflammation in both animal models and humans. These changes are associated with improved leptin and insulin sensitivity, more appropriate sympathetic responses and better glycemic control[36]. Lifestyle interventions that restore circadian alignment like regular sleep–wake cycles, earlier meal timing and reduced light at night, also appear to normalize aspects of brain–adipose signaling. Aligning feeding with periods of higher endogenous insulin sensitivity and thermogenic capacity may enhance sympathetic responses to meals and improve adipose handling of nutrients[36].

Pharmacologic therapies can indirectly or directly modulate the neuro–adipose axis. GLP-1 receptor agonists and dual/triple incretin agonists reduce appetite through central and vagal mechanisms and promote weight loss, which in turn improves adipose and hypothalamic inflammation. Some evidence suggests they also enhance BAT activity and browning, possibly via central sympathetic mechanisms[37]. SGLT2 inhibitors shift substrate utilization and may favor adipose remodeling and sympathetic rebalancing. Thiazolidinediones improve adipose insulin sensitivity and expand subcutaneous fat, thereby altering leptin and adipokine signaling to the brain, although their use is constrained by side effects.[37].

Direct neuromodulation of autonomic circuits is an emerging frontier. Experimental approaches include chemogenetic or optogenetic activation of hypothalamic neurons that drive BAT thermogenesis, spinal cord stimulation and peripheral neuromodulation targeting sympathetic outflow. While these are not yet clinically applicable, they demonstrate that manipulating neural traffic to adipose tissue can markedly alter body weight and glucose homeostasis in animals[37]. At the periphery, preserving or restoring adipose sensory innervation, particularly PIEZO2-positive neurons, may become a therapeutic goal. Strategies that protect peripheral nerves from metabolic injury, akin to approaches in diabetic neuropathy, could maintain accurate afferent feedback and improve closed-loop control of adipose function[37].

Taken together, current interventions already act, often unintentionally, on brain–adipose circuits. Future therapies may more explicitly aim to recalibrate this axis, combining weight loss, circadian hygiene, adipose-specific pharmacology and perhaps targeted neuromodulation.

7. Neuro–Adipose-Informed Precision Medicine for Diabesity: Outlook

As understanding of neuro–adipose signaling deepens, diabesity can be reframed as a disease of network architecture rather than of isolated organs. This perspective opens possibilities for precision medicine grounded in brain–fat communication metrics.

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One avenue is phenotyping patients by central and peripheral markers of neuro–adipose function. Neuroimaging of hypothalamic structure and inflammation, combined with functional MRI during food cues, could identify individuals with pronounced central leptin and insulin resistance. Peripheral readouts such as BAT activity on PET–CT, skin or supraclavicular thermography, and nerve imaging of adipose depots might quantify sympathetic and sensory integrity. Circulating adipokine profiles, extracellular vesicle signatures and genetic variants in melanocortin, leptin and clock genes could further refine stratification[28, 30, 34].

Such profiling could guide therapy choices. Patients with marked hypothalamic inflammation and low BAT activity might benefit most from aggressive weight loss, GLP-1–based agents and cold- or drug-induced BAT activation. Those with substantial circadian misalignment might respond particularly well to time-restricted eating and structured light and sleep interventions. Individuals with preserved BAT and sympathetic tone but severe hedonic overeating could require treatments that preferentially target reward and cognitive control circuits[29, 38]. Key challenges include translating complex neurobiological measurements into clinically usable tools, avoiding over-medicalization of behavioral variability, and ensuring interventions are feasible in real-world social and occupational contexts, including shift work and constrained environments. Further, much of our mechanistic knowledge comes from rodent studies; human adipose innervation patterns, sensory pathways and hypothalamic microcircuitry are less accessible and may differ in important ways[39].

CONCLUSION

Nevertheless, the central message is clear: brain–adipose communication is not a peripheral detail of obesity and T2D but a core determinant of who develops diabesity, how severe it becomes and how reversible it is. By making these signaling pathways visible through imaging, electrophysiology, molecular biomarkers and careful clinical phenotyping, we can move toward interventions that do more than lower numbers on a scale or glucose meter. We can aim to re-establish a resilient, adaptive dialogue between the brain and adipose tissue, restoring the system's ability to flexibly respond to changing environments without sliding into chronic disease.

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