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REVIEW ARTICLE

Integrating Yoga and Physical Exercise to Modulate the Nucleus Tractus Solitarii–Hypothalamus Circuit: Convergent Cardiovascular, Neuroendocrine, and Neuroimmune Mechanisms

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ABSTRACT

Background: The nucleus tractus solitarii (NTS) in the dorsal medulla is the principal relay for all visceral afferent information and the main circuitry with the hypothalamic paraventricular nucleus (PVN) and other forebrain regions. This NTS–hypothalamus circuit regulates cardiovascular reflexes, hormone release, and immune signalling. Yoga and aerobic exercise are common lifestyle interventions with overlapping benefits, but their combined effects on this circuit are rarely studied.

Objective: Integrate data on individual as well as combination effects of yoga and aerobic exercise over the NTS-hypothalamus circuit.

Methods: This narrative review compiles mechanistic and physiological evidence on how aerobic exercise and yoga affect NTS–hypothalamus signalling, including descending and ascending pathways, neuroendocrine and metabolic mediators, and vagal immune circuits.

Key Findings: Exercise modulates the descending PVN-to-NTS oxytocin and vasopressin pathways, increases the excitability of PVN–NTS-projecting neurons, and resets baroreflex. This lowers resting heart rate and blunts the exercise of heart rate response. Yoga mainly affects the ascending sensory pathways. Slow, deep breathing upregulates vagal activity in the NTS, improving heart rate variability. It also enhances thalamic GABA, which inhibits HPA-axis activity and sympathetic discharge. Both interventions overlap in hormonal and metabolic signalling through NTS GLP-1, catecholamine, and oxytocin systems projecting to the PVN and arcuate nucleus, regulating appetite and glucose. They also share immune signaling effects via the vagal anti-inflammatory pathway, which lowers inflammation and balances autonomic tone.

Conclusions: Yoga and exercise modulate the NTS–hypothalamus circuit through distinct, simultaneously complementary pathways. Combination practices may enhance cardiovascular, endocrine, and immune benefits, supporting targeted and personalised lifestyle interventions. We discuss how these mechanistic differences can guide practice selection and combination and outline current limitations and future research needs.

KEYWORDS: *Autonomic nervous system, Glucagon-like peptide-1, Hypothalamus, Inflammatory reflex, Nucleus tractus solitarii, Oxytocin, Yoga, exercise training.*

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INTRODUCTION

Many chronic diseases, such as hypertension, type 2 diabetes, and stress-related mental and inflammatory disorders, have a common feature: disrupted communication between the body and the brain. The nucleus tractus solitarii (NTS) in the dorsal medulla of the brainstem is crucial to this process. It receives sensory information and taste signals primarily from cranial nerves VII, IX, and X. The NTS, once considered merely a reflex relay for functions like heart rate and breathing, is now understood as a crucial hub. It processes diverse sensory inputs and orchestrates appropriate responses through its connections to networks governing bodily functions and internal sensations.[1] The NTS's lower region integrates forebrain, vagus nerve, and spinal cord signals, linking thoughts and emotions with bodily states.[2]

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The NTS-hypothalamus circuit, primarily involving the hypothalamic paraventricular nucleus (PVN), facilitates significant bidirectional integration.[3] This adaptable circuit likely mediates the broad effects of lifestyle interventions. Yoga, a common practice, often equals or exceeds aerobic

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exercise in various health benefits, with the exception of cardiorespiratory fitness.[4] However, they are rarely studied together in relation to their effects on the brain's central circuits. This review brings together evidence from the cited sources showing that both yoga and exercise influence the NTS–hypothalamus circuit, each in partly different but complementary ways. Knowing how these mechanisms work can help guide the choice, combination, and customisation of these practices for specific problems with the autonomic or metabolic systems.

The NTS–hypothalamus circuit as an integrative substrate

The NTS acts as the main centre for cardiovascular reflexes, such as the baroreflex and the exercise pressor reflex. It gets input from baroreceptors, chemoreceptors, and muscle metaboreceptors. Second-order NTS neurons control vagal outflow and, via the ventrolateral medulla, set sympathetic tone. The PVN and amygdala send signals that provide predictive control. During intense endurance exercise, the amygdala–PVN–NTS circuit is activated together. This circuit can limit performance, showing how emotion, autonomic drive, and this pathway are closely linked .[5]

Figure 1 shows this structure. Two features are important. First, most of the PVN input to the NTS comes from parvocellular neurons: about 60% of these neurons make corticotropin-releasing hormone, and a smaller group makes oxytocin.[2] Second, the oxytocin and vasopressin neurons that return to the brainstem terminate on the same catecholaminergic groups that ascend to the PVN, completing the circuit.[3]

The NTS contains many types of neurons. These neurons produce noradrenaline, GABA, GLP-1, prolactin-releasing peptide, pro-opiomelanocortin, and cholecystokinin and belong to groups that connect to the hypothalamus and limbic system. This variety allows the NTS to coordinate cardiovascular, neuroendocrine, metabolic, and neuroimmune functions. It also offers several molecular targets for behavioural treatments.[2,6] The balance between these neuron types is important: NTS GABAergic neurons affect blood glucose, while glutamatergic neurons control sympathetic nerve activity.[2]

Exercise training remodels the circuit: cardiovascular and neuroendocrine effects.

Oxytocin and vasopressin. Exercise training clearly changes this circuit. The only sources of oxytocin (OT) and vasopressin (VP) in the dorsal brainstem are PVN projections. Exercise activates these PVN-to-NTS neurons.3 In trained normotensive animals, hypothalamic OT messenger RNA levels increase by about 3.5-fold. NTS OT levels increase, and resting bradycardia emerges.[3,7]

Opposing, complementary actions. In the NTS, OT enhances baroreflex sensitivity and range, and in trained animals,

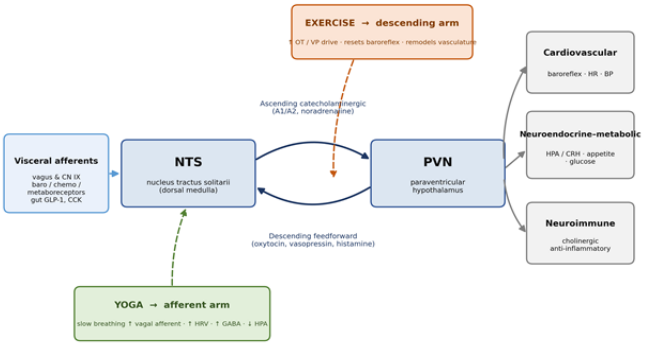


Figure 1: The NTS–hypothalamus circuit integrates visceral sensations, driving cardiovascular, neuroendocrine-metabolic, and neuroimmune responses. The NTS and PVN are connected by a reciprocal loop; visceral afferents signal the NTS, which in turn signals the PVN. The PVN then projects back to the NTS. Exercise mainly affects the descending arm, while yoga primarily impacts the afferent arm. BP, blood pressure; CCK, cholecystokinin; CN IX, glossopharyngeal nerve; GABA, γ-aminobutyric acid; GLP-1, glucagon-like peptide-1; HPA, hypothalamic–pituitary–adrenal; HR, heart rate; HRV, heart-rate variability; OT, oxytocin; VP, vasopressin.

it mitigates exercise-induced tachycardia. Conversely, VP shifts the baroreflex to higher pressure and heart rate, thereby increasing heart rate during exercise. Studies show that dynamic exercise elevates VP levels in the brainstem; injecting VP into the NTS boosts exercise tachycardia, while blocking V1-receptors diminishes it, with neither affecting blood pressure. This PVN-to-NTS pathway, by altering the baroreflex set point, links central command to baroreceptor feedback, and its continuous activity is crucial for normal baroreflex heart rate control.[8]

Cellular remodelling. Exercise changes cellular pathways, making PVN neurons projecting to the NTS more excitable and responsive, with less afterhyperpolarization. At the same time, neurosecretory magnocellular neurons become less excitable. As a result, OT and AVP are released into the dorsal brainstem, improving cardiovascular control.[9] Structural alterations occur in catecholaminergic inputs to the paraventricular nucleus (PVN) and in dendrites of cardiorespiratory regions within the medulla and hypothalamus. These physiological adaptations account for the primary effects of training, including decreased resting heart rate, diminished exercise-induced tachycardia, and enhanced baroreflex regulation.[3]

Additional descending and vascular effects. In addition to OT and VP, descending control during exercise involves histaminergic neurons of the tuberomammillary nucleus projecting to the NTS.

Activation of H1-receptors leads to pressor and tachycardic responses. Chronic exercise alters this pressor response, indicating a tuberomammillary–NTS feedforward pathway.[10] Exercise also changes the blood supply to these

nuclei. Aerobic training increases capillary growth in the PVN and NTS. It normalises the brain artery wall-to-lumen ratio in hypertensive animals, preserving blood flow in the preautonomic network.[11] At the network level, these changes alter the PVN–NTS–ventrolateral medulla circuit, resulting in decreased sympathetic activity and increased parasympathetic tone.[12] This matches broader adaptations in the brainstem, hypothalamus, and HPA-axis structures.[13]

Yoga modulates the circuit: autonomic, GABAergic and neuroendocrine effects

Afferent activation by slow breathing. Yoga mainly affects this circuit from the afferent side. Slow pranayamic breathing increases parasympathetic activity and reduces sympathetic dominance. As a result, both heart rate and blood pressure drop, while heart-rate variability (HRV) rises due to increased vagal afferent input. Yoga rebalances stress-affected systems—autonomic nervous system, GABA, and HPA axis—by stimulating the vagus nerve and reducing allostatic load.[14] The evidence supports these findings. Iyengar yoga increased parasympathetic modulation of the heart and heart rate variability (HRV) compared with walking. A single 60-minute session increased thalamic gamma-aminobutyric acid (GABA) by 34% in experienced practitioners and by by 15% in novices after 12 weeks, which correlated with improved mood. Walking did not produce these changes. This suggests that slow breathing, not just movement, drives vagal afferent activation.[14] Human studies support these findings. In adults with migraine, 12 weeks of structured pranayama, in addition to standard care, improved heart rate variability (HRV) and baroreflex sensitivity, and reduced heart rate and blood pressure. Cardiac autonomic balance also shifted toward parasympathetic dominance compared to controls.[15]

Downregulating the stress axis. Yoga reduces activity in neural pathways influencing the hypothalamus. Reviews and meta-analyses show that adjuvant yoga reduces blood pressure, heart rate, cortisol, and cytokine levels more than usual care alone. It also alters sympathetic nervous system and hypothalamic-pituitary-adrenal (HPA) axis activity (16-18). The benefits go beyond physical health. A meta-analysis of randomised trials found that yoga reduces self-reported post-traumatic stress and depressive symptoms. This supports its use as a complementary treatment for stress-related mental illnes.[19,20] Mind–body practices in general improve vagal tone and autonomic balance. They also cause measurable neuroendocrine and epigenetic changes in stress-regulatory genes, which match the physiological effects.[21]

Neuroendocrine–metabolic convergence: oxytocin, GLP-1 and the gut–brain axis

A shared GLP-1 node. The NTS is a crucial part of the gut-brain axis, where both systems influence shared pathways. GLP-1 is produced by enteroendocrine L-cells and NTS preproglucagon neurons. Gut-derived GLP-1 is rapidly degraded and mainly signals via vagal afferents. Brain-derived GLP-1 acts as a neurotransmitter. NTS GLP-1 neurons project to the paraventricular, supraoptic, arcuate, and dorsomedial hypothalamic nuclei, regulating feeding, glucose, and cardiovascular function. This node has two roles: activating NTS GLP-1 neurons reduces food intake but also increases heart rate and lowers liver glucose production.[2] Exercise activates the oxytocin pathway, leading to oxytocin neurons in the PVN projecting to and activating GLP-1 neurons in the NTS.[22] Exenatide, a GLP-1 analogue, promotes functional connectivity between the NTS, thalamus, and hypothalamus in humans, reducing hunger, particularly in obese individuals.[23]

Mapping the feeding circuit. The NTS-to-hypothalamus feeding circuit is now well understood. Noradrenergic A2 and GLP-1 neurons, two key NTS neuron groups, densely innervate the medial parvocellular PVN and connect to corticotropin-releasing hormone neurons at the HPA axis apex. Gut signals, such as cholecystokinin, activate these neurons depending on feeding status.[24] Activating NTS A2 neurons projecting to the PVN decreases food intake without discomfort, engaging both PVN oxytocin and corticotropin-releasing factor neurons.[25] Another cholecystokinin NTS-to-PVN pathway controls appetite without negative effects.[26] Nutrient sensing also uses this circuit. Dietary amino acids activate NTS prolactin-releasing peptide neurons. These neurons inhibit hypothalamic AgRP neurons through leptin receptor neurons in the dorsomedial hypothalamus. This process contributes to high-protein satiety and long-term energy balance.[27] The system also works in reverse. When glucose is low, NTS catecholamine neurons projecting to the arcuate nucleus stimulate feeding by affecting both AgRP and pro-opiomelanocortin neurons.[28] So, one catecholaminergic hub can either suppress or promote intake, depending on its hypothalamic target. Exercise affects this circuit through metabolic changes. As a metabolic challenge, it alters activity in arcuate pro-opiomelanocortin and AgRP neurons, as well as other feeding populations, thereby influencing appetite and glucose levels.[29]

Translation to metabolic benefit. These circuits translate into measurable benefit. Both exercise and yoga lower not only fasting but also postprandial glucose levels. Also improve lipid profiles in individuals with diabetes and other chronic conditions. Head-to-head trials show comparable falls in fasting blood glucose.[4] Yoga additionally lowers body mass index and improves insulin sensitivity, and mind–body practices modulate metabolic and neuroendocrine signalling.[4,18] The GLP-1, catecholaminergic and oxytocin systems of the circuit thus bridge the cardiovascular effects of exercise and the neuroendocrine effects of yoga to shared metabolic outcomes.

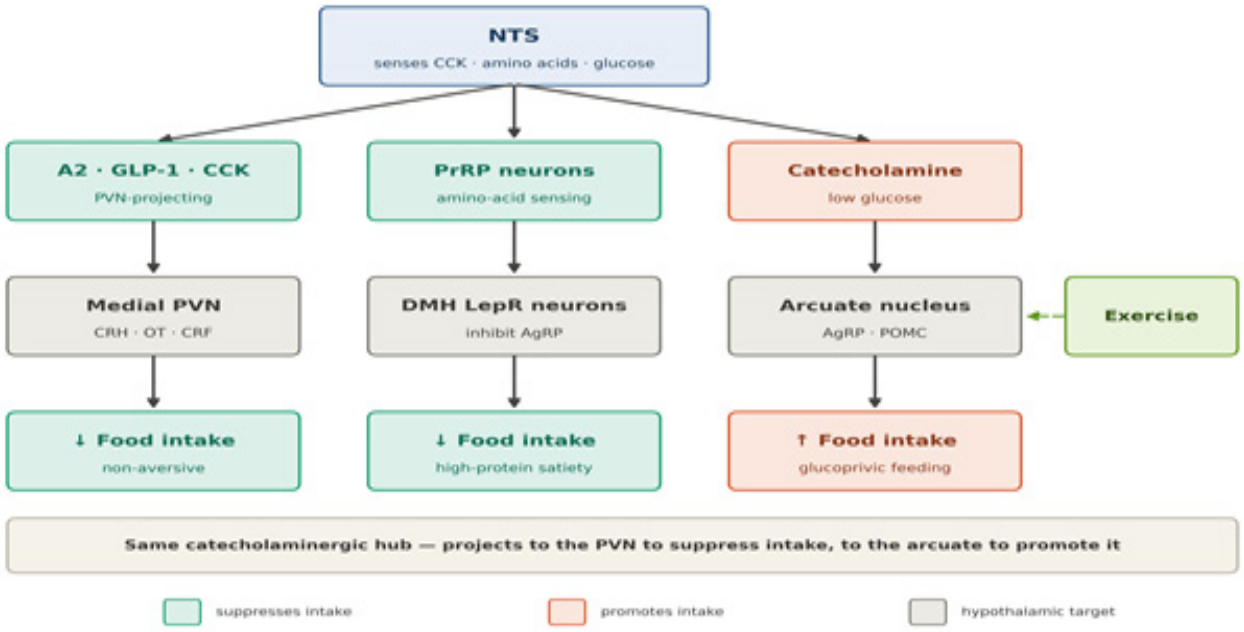


Figure 2: The NTS-to-hypothalamus feeding circuit acts as a central hub with different effects depending on its target. The nucleus tractus solitarii (NTS) gathers gut and metabolic signals, such as cholecystokinin, dietary amino acids, and glucose. Different groups of NTS neurons connect to specific hypothalamic areas. A2 noradrenergic, GLP-1, and cholecystokinin neurons send signals to the medial parvocellular paraventricular nucleus (PVN), which includes corticotropin-releasing hormone, oxytocin, and corticotropin-releasing factor neurons, to reduce food intake without causing discomfort. PrRP neurons, activated by amino acids, signal through LepR neurons in the DMH to inhibit AgRP neurons, promoting satiety after protein consumption. When glucose levels drop, NTS catecholamine neurons instead signal the arcuate nucleus (AgRP and pro-opiomelanocortin, POMC) to encourage eating. This means the same catecholaminergic hub can either suppress or promote food intake depending on its target. Exercise changes arcuate POMC and AgRP neurons, which affects appetite and glucose levels. In the diagrams, teal indicates intake-suppressing roles, coral indicates intake-promoting roles, and grey indicates hypothalamic targets.

NEUROIMMUNE CONVERGENCE: THE INFLAMMATORY REFLEX

The inflammatory reflex. The third arm of convergence is neuroimmune. The vagus supports a cholinergic anti-inflammatory pathway. Afferents detect peripheral inflammatory signals and activate the NTS–dorsal motor nucleus of the vagus axis. Efferent cholinergic signalling, relayed through the spleen, releases acetylcholine. This binds α7 nicotinic receptors on macrophages, suppressing nuclear factor-κB and pro-inflammatory cytokine production.[30] The reflex is bidirectional. Peripheral interleukin-1β and interleukin-6 can suppress NTS-to-hypothalamus transmission and drive RVLM sympathetic premotor neurons, raising sympathetic output and inflammation; restoring vagal tone rebalances the axis.[30] The anatomy of this cytokine-to-hypothalamus link is well established. Interrupting the ascending catecholaminergic projections from the medulla to the PVN abolishes interleukin-1β-stimulated activation of paraventricular corticotropin-releasing hormone neurons. The NTS catecholaminergic relay is therefore required for the neuroendocrine response to a circulating inflammatory cytokine.[31]

Both practices strengthen the reflex. Exercise strongly affects this system. Regular aerobic exercise reduces monocyte Toll-like receptor-4 and nuclear factor-κB signalling. It lowers interleukin-6 and tumour necrosis factor-α. Exercise-induced cytokines, like IL-15, enhance the α7 nicotinic cholinergic pathway, reducing oxidative stress in the RVLM and restoring GABAergic inhibitory tone, thereby improving sympathovagal balance.[31] In the heart, exercise works through interleukin-10 and STAT3 signalling. It also shifts the renin–angiotensin balance toward the protective angiotensin type-2 receptor, which is found on GABAergic NTS neurons. Voluntary exercise lowers the pro-hypertensive angiotensin type-1 receptor, interleukin-1β, and tumour necrosis factor-α, while increasing the angiotensin type-2 receptor and interleukin-10 in the PVN.[32] Yoga has similar effects. Reviews and trials show lower C-reactive protein and pro-inflammatory cytokines (interleukin-1β, interleukin-6, tumour necrosis factor-α), less stress-induced C-reactive protein and interleukin-6 and reduced nuclear factor-κB signalling.[16-18] Mind–body practices also reverse pro-inflammatory gene-expression profiles.[21] Both interventions strengthen the same inflammatory-reflex circuit, lowering systemic inflammation and restoring vagal–sympathetic balance.

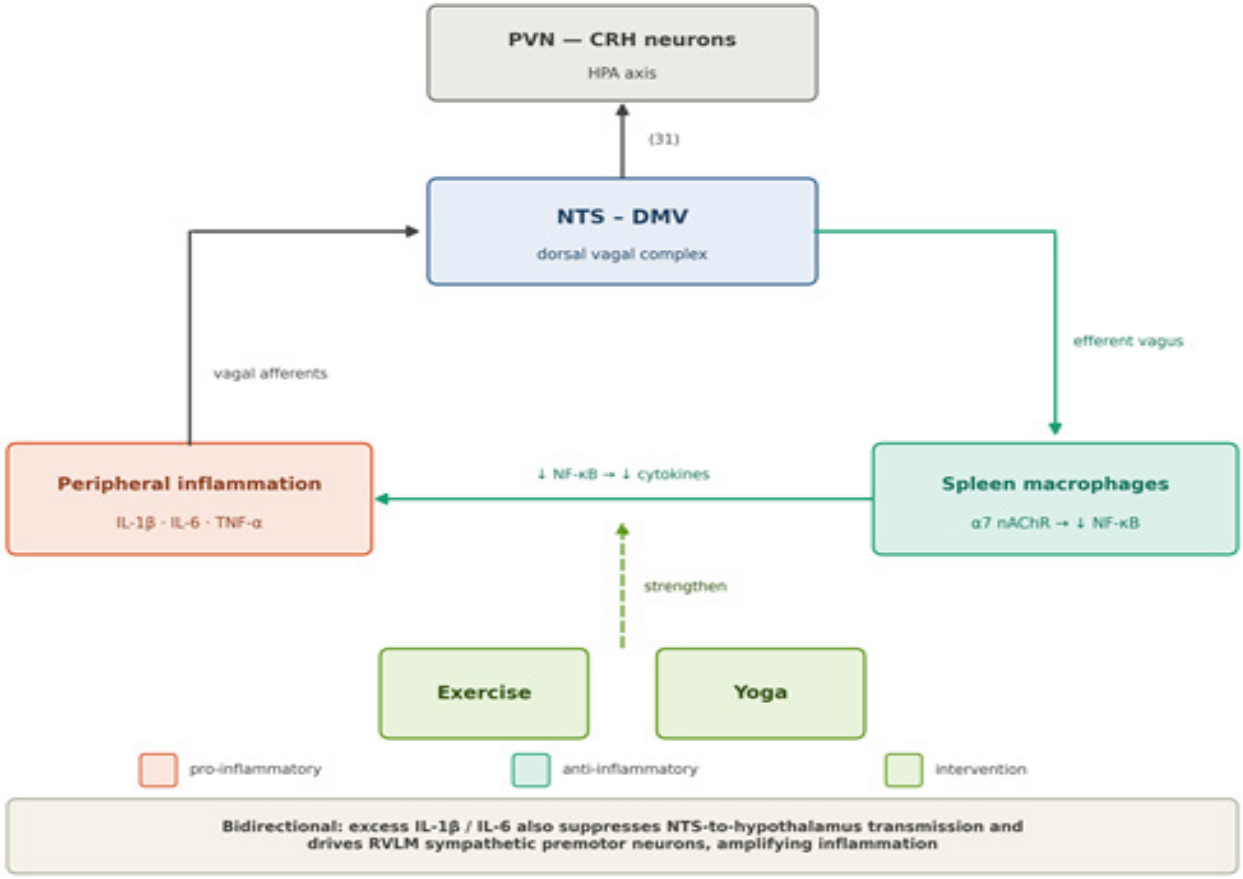


Figure 3: Exercise and yoga can modulate the inflammatory reflex. Vagal afferents detect peripheral inflammatory cytokines (e.g., IL-1β, IL-6, TNF-α) and transmit signals to the NTS and DMV. The anti-inflammatory response, mediated by the spleen, releases acetylcholine, which binds to α7 nicotinic receptors on macrophages, suppressing NF-κB and reducing pro-inflammatory cytokine production, thus completing a feedback loop. Another pathway signals the PVN, releasing CRH in response to cytokines. The reflex is bidirectional: high IL-1β and IL-6 can reduce NTS-hypothalamus communication and activate RVLM sympathetic premotor neurons, increasing inflammation. Exercise and yoga strengthen this reflex by lowering cytokine levels, supporting the α7-cholinergic pathway, and balancing vagal and sympathetic activity. Diagrams use coral for pro-inflammatory, teal for anti-inflammatory, and green for behavioural interventions.

Table 1: Differential and convergent actions of aerobic exercise and yoga on the NTS–hypothalamus circuit. Arrows denote the direction of change. Abbreviations as in Figure 1; AgRP, agouti-related peptide; AT1R/AT2R, angiotensin type-1/type-2 receptor; BMI, body mass index; CRP, C-reactive protein; IL, interleukin; NF-κB, nuclear factor-κB; POMC, pro-opiomelanocortin; TNF-α, tumour necrosis factor-α.

	Physical (aerobic) exercise	**Yoga**
Primary entry point	Descending PVN→NTS arm (feedforward)	Ascending vagal→NTS arm (afferent)
Key mediators	Oxytocin, vasopressin, histamine, catecholaminergic and vascular remodelling	Vagal afferent drive; thalamic GABA; ↓ cortisol
Cardiovascular	↑ baroreflex gain; resting bradycardia; blunted exercise tachycardia; PVN/NTS angiogenesis	↑ HRV; ↑ baroreflex sensitivity; ↓ HR and BP; parasympathetic shift
Neuroendocrine–metabolic	Remodels arcuate POMC/AgRP feeding circuits; ↓ glucose	↓ HPA / cortisol; ↓ glucose; ↑ insulin sensitivity; ↓ BMI
Neuroimmune	↓ IL-6, ↓ TNF-α; ↑ α7 cholinergic pathway; ↑ AT2R and IL-10 in PVN	↓ CRP, ↓ IL-6, ↓ TNF-α; ↓ NF-κB signalling
Most direct evidence	Hypertension; exercise intolerance; cardiorespiratory fitness	Stress-related, anxious and inflammatory conditions; PTSD and depression

Integration and clinical implications

Complementary entry points. Table 1 compares the two practices. Exercise mainly acts on the descending feedforward pathway by strengthening PVN-to-NTS oxytocin and vasopressin signals. It enhances PVN neuronal excitability that projects to the NTS and resets the baroreflex.[3,7,9] Yoga mainly acts on the ascending, afferent pathway by using slow breathing to boost vagal input, raise HRV and thalamic GABA, and reduce HPA-axis and sympathetic activity (14-16). These two pathways meet at shared neuroendocrine and neuroimmune points.[22,30] The same NTS catecholaminergic and GLP-1 projections to the PVN that control appetite and the HPA axis also carry inflammatory signals.[25,31] Their effects are mechanistically linked, not just additive.

Clinical implications. This complementarity is useful in practice. When the main goal is to improve descending cardiovascular drive, baroreflex function, and fitness—as in hypertension or exercise intolerance—exercise has the strongest evidence.[3] When the focus is on increasing vagal, GABAergic, and anti-inflammatory tone—as in stress-related, anxiety-related, or inflammatory conditions—yoga has the strongest evidence base. Yoga's benefits, compared with exercise, for stress, mood, and some physical symptoms are well documented.[4,14] For metabolic problems, both practices lower glucose and improve lipids and insulin sensitivity, and they can be combined.[4] A precision-medicine approach to exercise is being developed, using genomic, metabolomic, and autonomic biomarkers to tailor the "dose".[30] In the future, this could be used to prescribe yoga and exercise together, matched to a patient's autonomic and metabolic profile.

Limitations and future directions. Several limitations influence these conclusions. Most detailed information regarding NTS–PVN plasticity and the feeding circuit is derived from rodent studies. Direct human evidence linking either intervention to specific changes in NTS circuits remains limited.[3,9,28] Yoga trials differ in style, dosage, and participant characteristics. Findings for inflammatory markers and clinician-rated outcomes are inconsistent.[16,17,19] Few studies have directly compared yoga and exercise across diverse patient groups, and even fewer have evaluated their combined effects.[4] Future research should assess circuit-level outcomes, including HRV, baroreflex sensitivity, thalamic GABA, and neuroendocrine and inflammatory biomarkers, in adequately powered trials that compare and combine these interventions. Additionally, research should determine whether biomarker-guided prescriptions enhance outcomes.

CONCLUSION

Integrating these two practices modulates the NTS–hypothalamus circuit at multiple levels, improving cardiovascular, neuroendocrine, and neuroimmune outcomes. Elucidating these mechanisms supports the development of precision exercise prescriptions tailored to specific autonomic and metabolic dysfunctions, enhancing the effectiveness and individualisation of patient care.

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