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The influence of nutrients and nutrition's methods on the autonomic nervous system activity

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Abstract: The autonomic nervous system (ANS) plays a key role in maintaining physiological homeostasis by regulating cardiovascular activity, gastrointestinal motility, and stress responses.
The aim of this review was to analyze how selected dietary components, and complete nutritional models affect ANS activity, with particular emphasis on underlying physiological mechanisms and practical dietary recommendations in cases of sympathetic or parasympathetic hyperactivity.
This paper discusses how caffeine, simple sugars, and highly processed foods tend to enhance sympathetic output, whereas omega-3 fatty acids, magnesium, dietary fiber, promote autonomic balance by reducing sympathetic tone and enhancing parasympathetic function.
Based on a review of recent scientific studies, it can be concluded that nutrition and lifestyle interventions can significantly modulate ANS function and serve as valuable tools in the management of chronic stress, anxiety, hypertension, and functional gastrointestinal disorders.

Keywords: autonomic nervous system activity, nutrition, dietary habits.

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Introduction

The autonomic nervous system (ANS), along with the endocrine system, plays a crucial role in maintaining the body's homeostasis. It operates based on a reflex arc, which consists of three main elements: the sensory (afferent) component that receives stimuli, the efferent (motor) component that executes responses, and the central integrating system that processes and coordinates the body's responses [1]. This mechanism helps to maintain homeostasis, or the state of internal physiological balance, providing stable conditions for the functioning of organs and tissues [2].

ANS consists of two main components: the sympathetic and parasympathetic nervous system: the sympathetic nervous system (SNS) is responsible for initiating the body's defensive responses,

commonly known as the "fight or flight" response [3, 4]. The parasympathetic nervous system (PNS) is responsible for coordinating processes related to recovery and relaxation. It acts antagonistically to the SNS and facilitates the return to a resting state after the "fight or flight" response. Although the actions of the sympathetic and parasympathetic systems are generally antagonistic, their influence on various organs is not symmetrical [3–5].

Another crucial part of the ANS that collaborates in regulation is the enteric nervous system (ENS), which is essential for the proper functioning of the digestive system and maintaining its homeostasis. It comprises a complex network of neurons and glial cells connected to both the sympathetic and parasympathetic nervous systems. ENS is divided into the myenteric plexus, located between the longitudinal and circular muscle layers of the intestine, and the submucosal plexus, situated in the submucosal layer. Neurons of the myenteric plexus primarily regulate gastrointestinal motility, while neurons of the submucosal plexus control fluid balance. The function of the intestines is managed by the ENS through reflex arcs involving the longitudinal and circular muscles. Signal transmission in these circuits occurs via nicotinic synapses, which are associated with the action of acetylcholine [6, 7].

Research on the impact of diet on autonomic nervous system activity has gained increasing interest, especially in the context of rising metabolic disorders, stress, and neurodegenerative diseases. Nutrition significantly affects the functioning of the entire body. Nutrients such as macronutrients (proteins, fats, carbohydrates), micronutrients, vitamins, and fiber can modify nervous system activity through various biochemical, hormonal, and neurotransmitter mechanisms. An appropriate diet, rich in foods that support the proper functioning of the nervous system, can be part of the prevention and treatment of many disorders, such as excessive sympathetic activation, chronic stress, and cardiovascular diseases [8, 9].

The main objective of this study was to review recent scientific research describing the effect of nutrients and diets on autonomic nervous system activity. The aim was to find answers to whether food components can increase or decrease the tone of the sympathetic or parasympathetic components of the ANS and to understand the correlation between the ANS, which works alongside the endocrine system in regulating the body's homeostasis, and dietary habits.

The role of the ANS in the process of nutrition

ANS by regulating the body's responses independently of conscious control, orchestrates the process of food intake. The centers for hunger and satiety are located in the hypothalamus, which is a part of the diencephalon, within the nuclei of the hypothalamus such as the arcuate nucleus, the ventromedial hypothalamic nucleus (VMH), the lateral hypothalamic area (LHA), and the nucleus of the solitary tract [10]. The arcuate nucleus plays a key role in integrating signals that regulate appetite. It contains two distinct systems: the anorexigenic system, which suppresses appetite, and the orexigenic system, which stimulates food intake. The regulation of these processes is based on the action of neuropeptides, hormones, neurotransmitters, and signals from adipose tissue, insulin, and gastrointestinal hormones, which inform the brain about the body's energy needs. This regulation occurs at two levels: metabolic, primarily driven by signals originating from the gastrointestinal tract and hormonal activity, and non-metabolic, based on sensory stimuli [11, 12].

Regulation of food intake occurs through two mechanisms: long-term and short-term control. Long-term regulation is based on mechanisms that monitor the levels of lipids, carbohydrates, and amino acids in the blood [13]. An increase in blood glucose, amino acids, and leptin activates

the satiety center and suppresses the hunger center. Additionally, ANS is directly connected to adipocyte tissue, thus significantly affecting the body's metabolic homeostasis [14]. Short-term regulation, on the other hand, is closely associated with the degree of gastric wall distension and the stimulation of receptors that inhibit hunger. The type of consumed food and its physicochemical properties influence this process [15].

Following food ingestion (short-term regulation), anorexigenic substances are released in the gastrointestinal tract, which, via the vagus nerve and the nucleus of the solitary tract (NTS), influence appetite-regulating centers, leading to the suppression of hunger. Just before a meal, ghrelin is secreted, which, through endocrine pathways and via the hypothalamic centers, stimulates appetite. In mechanisms of long-term regulation of food intake, leptin, secreted by adipocytes, and insulin, present in the blood, play key roles [16]. They act on neurons in the arcuate nucleus (ARC) to inhibit appetite, producing an anorexigenic effect. Physical activity increases anorexigenic signaling, evidenced by elevated levels of PYY, GLP-1, and PP, while simultaneously suppressing orexigenic signals, such as a decrease in ghrelin concentration [17, 18].

Equally important in the regulation of food intake is the influence of hormones acting at various stages of this process. Peripheral mechanisms involved in this regulation include the action of gut hormones such as ghrelin, which stimulates appetite and is secreted by mucosal cells of the stomach during fasting. Conversely, leptin primarily produced by the stomach and pancreas exerts an anorexigenic effect by suppressing hunger. Corticotropin-releasing hormone (CRH), widely distributed in the central nervous system, also reduces appetite. Orexins (A and B), released from afferent neurons of the gut, act on OX R1 receptors located on nerve cells, increasing appetite similarly to ghrelin. Other peptides involved in appetite regulation include peptide YY (PYY), pancreatic polypeptide (PP), glucagon-like peptide-1 (GLP-1), cholecystokinin (CCK), and neuropeptide Y (NPY), all of which stimulate hunger [18–20] (Table 1).

Table 1. Central and peripheral regulation of body mass [18, 19].

Central regulation of body mass	Peripheral regulation of body mass — acting centrally
POMC — α-MSH	CCK
CART	GLP-1
NPY	PYY
AgRP	LEP
OXA	GHR
	INS
	Adiponectin
	Resistin
	OXM

POMC, α-MSH, Proopiomelanocortin — α-melanocyte-stimulating hormone, CART — Cocaine- and Amphetamine-Regulated Transcript, NPY — Neuropeptide Y, AgRP — Agouti-Related Peptide, OXA — Orexin A, CCK — Cholecystokinin, GLP-1 — Glucagon-like peptide-1, PYY — Peptide YY, LEP — Leptin, GHR — Ghrelin, INS — Insulin, OXM — Oxyntomodulin.

The effect of a meal on the ANS functions

ANS plays a crucial role in regulating food intake and digestion. Therefore, food consumption constitutes a significant factor inducing changes in ANS activity [18, 21]. The digestion process, especially the distension of the stomach wall by ingested food, activates regulatory mechanisms involving both the parasympathetic and sympathetic nervous systems [22]. The most important among them is the activation of the vagus nerve, which is the main nerve of the parasympathetic component. Increased parasympathetic activity leads to enhanced gastrointestinal motility, increased secretion of digestive juices, and improved blood flow in visceral organs. As a result, the digestive system is prepared for efficient digestion and nutrient absorption [23]. Simultaneously with the activation of the parasympathetic system, changes also occur in the sympathetic nervous system. The sympathetic system functions in a balancing role, preventing excessive drops in blood pressure due to redistribution of blood volume toward the gastrointestinal tract [21].

In healthy individuals, the response to a meal involves an increase in parasympathetic activity, which manifests through features such as a decreased heart rate and an increase in HRV within the high-frequency (HF) component (Table 2).

Table 2. Comparison of normal and abnormal ANS responses to a meal [24, 25].

Mechanism	Normal ANS response	Abnormal ANS response
Parasympathetic activity	Increased activity	Lack of activity
Sympathetic activity	Moderate activation (maintaining normal blood pressure)	High activation
Heart rate variability (HRV)	Increased in the high-frequency (HF) band	Reduced HRV, dominance of LF/HF ratio
Gastrointestinal symptoms	None (physiological sensation of fullness)	Abdominal pain, discomfort, bloating
Return to baseline	Rapid return to autonomic balance after digestion	Delayed return to balance

AUN — autonomic nervous system, HRV — heart rate variability, HF — high-frequency component of HRV, LF — low-frequency component of HRV.

Concurrently, there is a moderate activation of the sympathetic nervous system [26]. However, this response may be impaired in individuals with ANS dysfunction, which is observed in patients with functional dyspepsia. Such patients typically exhibit reduced parasympathetic activity and a persistent sympathetic dominance, which may contribute to symptoms such as early satiety, discomfort, or epigastric pain [25, 27].

The effect of water intake on the ANS functions

ANS activity can be stimulated by various stimuli, including physiological ones such as water consumption [28]. Recent years have seen increasing research focusing on the indirect hemodynamic effects of drinking water, which may have significant clinical implications, particularly in individuals with autonomic failure [29].

In patients with orthostatic hypotension, a significant rise in blood pressure has been observed within several minutes after water intake. This increase has been described as a pressor reflex, which is thought to result from activation of peripheral afferent nerve fibers that respond to changes in fluid volume [30]. Studies suggest that this response is especially pronounced in older adults and patients with ANS dysfunction, whereas in younger individuals, there is no significant increase in blood pressure. This indicates a compensatory mechanism in this group, involving increased activity of the vagus nerve, which helps balance sympathetic activation [31].

A non-invasive method used to evaluate ANS response is the water load test (WLT). The test involves drinking a specified volume of water within 5 minutes (the 5-minute water load test) until the subject reports a feeling of fullness. This procedure allows assessment of gastric signal perception (visceral hypersensitivity), particularly in patients with functional gastrointestinal disorders [32, 33].

The impact of water intake on the ANS can be investigated using HRV parameters. Studies have shown that after water consumption, changes occur in the HRV spectrum. The most frequently observed response is an increase in the low-frequency (LF) component, which indicates sympathetic activation. In some individuals, an increase in parasympathetic activity (high-frequency component, HF) has also been noted, which may reflect an attempt to restore balance in autonomic regulation [34, 35].

Nutrients and ANS functions

Dietary modulation of the sympathetic ANS activity

Activation of the sympathetic nervous system is a key physiological response to stress. It triggers the "fight or flight" reaction, mobilizing the body for rapid action by initiating a series of physiological changes [36]. These include an increase in heart rate and blood pressure, slowed gastrointestinal motility, bronchodilation, and stimulation of glycogenolysis, lipolysis, and thermogenesis [37]. During a stress response, the hypothalamus also activates the hypothalamic-pituitary-adrenal (HPA) axis, which is one of the most important systems regulating endocrine responses, functioning in a complementary manner to the sympathetic system. However, chronic activation of these systems can lead to the development of numerous pathologies. Therefore, understanding these mechanisms is crucial for developing effective prophylactic and therapeutic strategies for related disorders [38, 39]. In this context, growing interest is directed toward the role of environmental factors, including diet, in modulating ANS activity.

Previous research has indicated that certain dietary components can influence the activation of the SNS, either by enhancing or suppressing its activity. This modulation may have adaptive significance in the context of stress management or cognitive enhancement. Diet impacts the SNS through various mechanisms, including the regulation of neurotransmitter and hormone release, activation of the HPA axis, and direct effects on the central nervous system and gut microbiota [40, 41].

Caffeine is an alkaloid belonging to the methylxanthine group, present in coffee and tea, which acts as an antagonist of adenosine receptors (subtypes A1 and A2), whose activation leads to increased activity of the SNS, resulting in enhanced thermogenesis, elevated blood pressure, and the occurrence of tachycardia [42]. Furthermore, at high concentrations, caffeine inhibits phosphodiesterases, which increases the level of cyclic adenosine monophosphate (cAMP) within cells, facilitating neurotransmitter release and participating in the regulation of nerve impulse

transmission [43]. Clinical studies have demonstrated that a single dose of 250–300 mg of caffeine significantly increases the activity of sympathetic muscle fibers — MSNA and raises blood pressure [44]. Additionally, regular caffeine consumption among habitual coffee drinkers is associated with sustained long-term activation of the SNS. The substance may also amplify the body's response to stress stimuli by enhancing the activity of the hypothalamic-pituitary-adrenal axis. Such effects can result in more frequent and prolonged increases in blood pressure, which over time may constitute a risk factor for the development of cardiovascular diseases such as ischemic heart disease [45].

Theobromine, the main methylxanthine component of cocoa and dark chocolate, acts similarly to caffeine but with a lesser intensity. It also increases intracellular cAMP levels and blocks adenosine receptors. Both substances activate the SNS and elevate the plasma concentrations of stress hormones: cortisol and catecholamines. Additionally, theobromine leads to activation of the renin-angiotensin-aldosterone system (RAAS), resulting in a slight increase in renin and aldosterone levels particularly observed after consuming large quantities of dark chocolate, while no significant effect is seen following white chocolate intake [46]. The consequence of this activity includes stimulation of β-adrenergic receptors in the heart and blood vessels, which increases cardiac output, and activation of α1-adrenergic receptors in the vasculature, causing vasoconstriction. High consumption of methylxanthines may therefore induce transient sympathetic activation, tachycardia, increased blood pressure, and thermogenesis [45, 46].

Salt intake influences the SNS through volume-pressure and neuroendocrine mechanisms. Excess salt initially leads to an increase in blood volume and blood pressure, triggering baroreceptor reflexes that decrease sympathetic tone. However, under conditions of chronic high sodium intake, a significant increase in SNS activity is observed, particularly in individuals with hypertension and renal diseases [47]. At the central level, salt also influences the activation of the RAAS [48]. It has been demonstrated, that in animal models, a high-salt diet impairs the salt-induced suppression of renin and catecholamine activity. In rats fed with a 0.9% NaCl solution, stronger activation of the SNS and a higher rise in blood pressure were noted following angiotensin II (Ang II) administration into the rostral ventrolateral medulla (RVLM), compared to rats drinking water without added salt. Additionally, blockade of AT1 receptors in the RVLM in these animals resulted in a considerable decrease in renal and visceral SNS activity and lowered blood pressure. These findings suggest that excess salt enhances the sensitivity of hypothalamic and brainstem neurons to angiotensin II, thereby amplifying sympathetic activation, which may contribute to iatrogenic hypertension [48, 49]. Moreover, there is evidence that high sodium intake also activates the HPA axis. In mice on a high-sodium diet, increased baseline corticosterone secretion was observed, and in humans, elevated cortisol levels, along with increased expression of CRH and POMC in the hypothalamus and pituitary, were reported [47]. This indicates that regular high salt consumption may sensitize stress mechanisms excess salt amplifies the HPA axis response to stressors. As a result, there is a sustained increase in glucocorticoids and catecholamines, which, together with SNS activation, promotes increased sodium and water retention and raises blood pressure [50].

Capsaicin is the primary pungent compound found in chili peppers. It is widely recognized for its anti-inflammatory and neuroprotective effects, largely due to its antioxidant properties [51]. This compound activates the TRPV1 receptor located on sensory nerves of the gastrointestinal tract. The stimulation is transmitted to the brain, leading to an increase in sympathetic nerve activity targeting brown adipose tissue (BAT) [52]. Studies on animals and humans have shown that a diet rich in capsaicin can modestly increase total energy expenditure and support the reduction

of adipose tissue. Although the effect is modest, regular consumption of capsaicin may aid in weight management [53]. In addition to capsaicin, other compounds can stimulate thermogenesis, including piperine from black pepper and gingerols from ginger [52].

Carbohydrate intake strongly activates the SNS. The postprandial sympathetic response is necessary for compensating the influx of glucose and lipids, as well as for maintaining cardiovascular and metabolic homeostasis. This mechanism partially involves stimulation of sensory receptors in the intestine and the portal system of the liver, which transmit signals to central nervous system centers, leading to increased peripheral sympathetic activity [21]. Animal studies have shown that administration of glucose or fructose significantly elevates the rate of norepinephrine (NA) breakdown in brown and visceral adipose tissue, indicating enhanced NA release. In contrast, galactose does not induce such SNS stimulation [54].

Insulin plays a crucial role here. It is rapidly secreted following glucose intake and increases systemic sympathetic activity through central action in the hypothalamus. Insulin stimulates the paraventricular nucleus of the hypothalamus, leading to increased glucose uptake by tissues [55]. This accelerates metabolism in the liver and muscles and increases thermogenesis. Some effects of carbohydrate consumption are also due to ascending reflexes from the gastrointestinal tract (e.g., neuropeptide release) and hepatotoxic changes, which can modulate baroreceptors and axially influence sympathetic tone. In summary, carbohydrates stimulate sympathetic neurons through a combination of metabolic and hormonal signals, as illustrated by the increase in sympathetic parameters (blood pressure, catecholamines) following their consumption [21, 54].

Dietary modulation of the parasympathetic ANS activity

The parasympathetic system is a part of the ANS responsible for the body's state of rest. Parasympathetic activation slows the heart rate, lowers blood pressure, enhances digestion by stimulating saliva and digestive juice secretion, and promotes regeneration processes. PNS activity is influenced by many external factors, including diet. Meal consumption itself stimulates the component of the autonomic nervous system responsible for digestion through stomach wall distension. Furthermore, certain dietary components can increase parasympathetic activity through various mechanisms such as influencing neurotransmitters, modulating receptors, or interacting with the gut-brain axis [56].

Omega-3 fatty acids, found among others in oily marine fish (eicosapentaenoic acid and docosahexaenoic acid) and flaxseed (alpha-linolenic acid), play a vital role in nervous system functioning. In particular, docosahexaenoic acid (DHA) is a key component of neuronal phospholipid membranes, with its content being significantly increased in the central nervous system [57]. As a result, omega-3 fatty acids can modulate autonomic control affecting the balance between sympathetic and parasympathetic activity. Studies have shown that higher dietary intake of omega-3s is associated with increased parameters of HRV, which are the indicators of vagal nerve activity and parasympathetic dominance. The mechanisms through which these fatty acids enhance PNS activity include their anti-inflammatory effects and improvement of baroreceptor reflex sensitivity. Incorporation of DHA into the membranes of cardiac muscle cells and neurons stabilizes electrical activity in the heart and strengthens vagal tone, which may be protective against arrhythmias and rhythm disturbances. Enhanced vagal activity induced by omega-3 fatty acids is considered one of the cardioprotective mechanisms, as it correlates with a lower incidence of sudden cardiac death and arrhythmias in individuals with high intake of these fats [58, 59].

Magnesium (Mg) is an essential mineral for proper functioning of the nervous and muscular systems, and adequate dietary intake may enhance parasympathetic activity. It acts as a cofactor in numerous enzymatic reactions, participates in nerve conduction, and modulates neurotransmitter receptor activity [60]. Importantly, Mg helps maintain the balance between excitation and inhibition within the nervous system; its ions exert inhibitory effects on glutamatergic NMDA receptors and indirectly support GABA-ergic neurotransmission.

Physiologically, magnesium is known for its muscle-relaxant and calming effects, which are associated with activation of the PNS, aiding in stress reduction. Past investigation indicate that individuals with higher serum magnesium levels tend to have a more favorable HRV profile. For example, research on healthy subjects has demonstrated a significant positive correlation between magnesium levels and HRV parameters reflecting vagal tone. Conversely, magnesium deficiency is linked to reduced HRV and a dominance of sympathetic components [61].

Supplementation has been shown to reverse these adverse changes. Clinical studies have found that magnesium administration in patients (e.g., those with arrhythmias) increases parasympathetic activity markers and improves autonomic balance. This mechanism involves, among other effects, decreased secretion of stress hormones and direct facilitation of cholinergic conduction. Magnesium also plays a role in the synthesis and release of acetylcholine, the primary neurotransmitter of the parasympathetic nervous system, making its proper level crucial for optimal vagus nerve function. Magnesium maintains ion homeostasis within the ANS, affecting neurotransmission and modulating brain-derived neurotrophic factor (BDNF), which also positively influences the integrity of the blood-brain barrier and exerts neuroprotective effects [62] (see Fig. 1).

In clinical practice, this mineral is recommended as a remedy to alleviate stress symptoms and improve sleep quality. In the diet, its sources include green leafy vegetables, nuts, and seeds; therefore, it is very important to incorporate these foods into daily nutrition [62, 63].

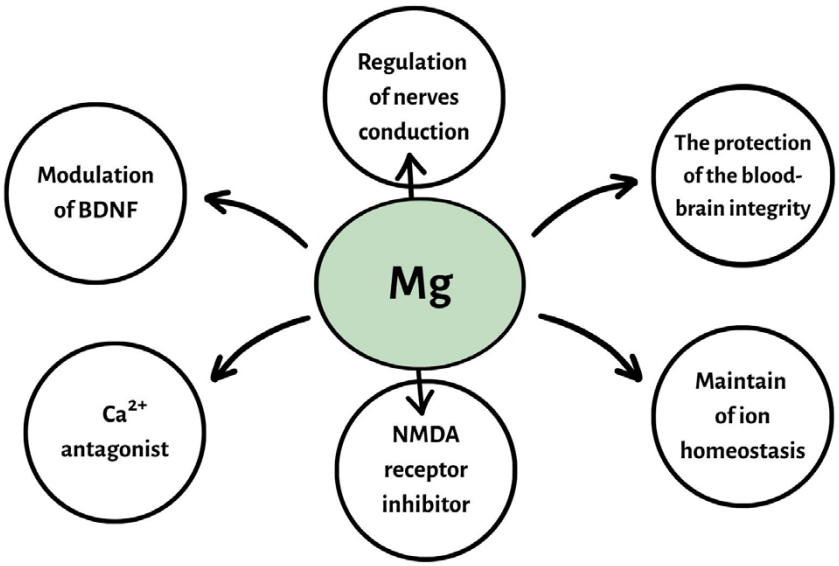

Fig. 1. The influence of magnesium on the ANS.
BDNF — brain-derived neurotrophic factor.

Tryptophan is an exogenous amino acid found in foods such as eggs, fish, turkey meat, sesame seeds, and pumpkin seeds. It is widely known as a precursor to serotonin — a neurotransmitter playing a key role in regulating mood, sleep, and digestion [64].

The influence of tryptophan on the parasympathetic nervous system primarily stems from its conversion to serotonin and subsequently to melatonin.

Serotonin participates in stimulating the gut-brain axis; approximately 90% of serotonin is produced in the gastrointestinal tract (in enterochromaffin cells), where it regulates intestinal motility and digestive secretion. Activation of serotonergic receptors in the gastrointestinal system increases myoelectric activity and secretion, supporting efficient digestion [64]. Moreover, some serotonin molecules indirectly influence brain centers via neural signals, affecting mood and stress responses. Higher dietary tryptophan availability promotes serotonin synthesis in the brain, which can exert anxiolytic and calming effects, reducing excessive sympathetic activity.

Experimental studies have shown that lowering tryptophan levels results in decreased serotonin concentrations, accompanied by a significant reduction in HRV and an increase in anxiety symptoms. These findings suggest that adequate dietary tryptophan intake is essential for maintaining proper parasympathetic activity. The produced serotonin activates brain stem regions (such as the nucleus of the solitary tract and dorsal vagal nucleus), which are responsible for controlling the vagus nerve, thereby facilitating parasympathetic signals to organs. Additionally, the end product of this pathway, melatonin, supports cyclic PNS activity during sleep and regeneration. It is also worth noting that vitamin B6 is important for this process, as its active form acts as a cofactor in the conversion of tryptophan to serotonin [65, 66].

L-theanine is a non-protein amino acid found in the leaves of green tea. It is known for its relaxing and anxiolytic effects. Structurally, it resembles glutamic acid but functionally acts as an antagonist of glutamate receptors (NMDA and AMPA) in the brain, which limits excessive neuronal excitation. At the same time, L-theanine stimulates the release of gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter that promotes "calmness" of the nervous system activity [67]. As a result, this amino acid may shift the autonomic balance toward parasympathetic dominance, especially under stress conditions. Controlled human studies have shown that administration of L-theanine prior to a stressful situation reduces physiological stress responses, including heart rate, cortisol levels, and salivary immunoglobulin A, compared to placebo. Heart rate variability analysis in these studies indicated that L-theanine decreases excessive sympathetic activity, reflected by a lower heart rate while maintaining or even increasing parasympathetic component indices. In other words, this amino acid prevents the typical rise in heart rate and blood pressure associated with stress by suppressing adrenaline release and enhancing vagal tone [68].

This mechanism explains why consuming green tea induces a feeling of calmness and improves concentration without causing feelings of anxiety. Additionally, L-theanine increases alpha wave activity in the brain, which correlates with a relaxed state, by blocking excessive glutamatergic stimulation in stress centers and activating GABA-ergic neurons. It allows the body to recover more quickly to a state of balance after exposure to a stressor [69].

Scientific evidence has demonstrated that the **gut microbiota**, along with **probiotics and prebiotics supplied** through diet, can influence the functioning of the enteric nervous system and the brain, including increasing the activity of the vagus nerve (n. X) [70]. The gut-brain axis is a bidirectional communication system between the gastrointestinal tract and the central nervous system, in which the vagus nerve and chemical signals play a key role. Probiotics — beneficial

live bacteria present in fermented foods or supplements — can modulate this axis by producing neuroactive substances and interacting with gut cells. For example, some bacteria from the Lactobacillus and Bifidobacterium genera produce butyric acid and GABA, which act on n. X receptors in the gut wall, sending inhibitory signals to the brain.

In preclinical studies, administration of the Lactobacillus rhamnosus JB-1 strain to mice over several weeks resulted in decreased levels of stress hormones and anxiety symptoms; however, this effect was abolished after vagotomy [71]. This suggests a direct role of parasympathetic stimulation driven by vagal signals from the gut. The mechanisms by which probiotics influence the vagus nerve include: the production of neurotransmitters in the gut that activate n. X receptors; reduction of inflammation through strengthening of the intestinal barrier; and modulation of sensory nerves in the gut, which physiologically increases afferent signals via the vagus nerve [72]. From a dietary perspective, consuming fermented foods supports a healthy microbiota, which indirectly translates into higher vagal activity and promotes a state of rest and recovery, essential for optimal daily functioning and overall health.

Meal frequency and the function of the ANS

Regular eating habits play a crucial role in maintaining ANS balance. Irregular meal patterns, especially prolonged intervals between meals, can lead to fluctuations in blood glucose levels, which stimulate sympathetic nervous system activity. This response resembles a stress reaction and may result in an increase in blood pressure to enhance blood flow to vital organs. Chronic sympathetic activation promotes the development of hypertension, may suppress appetite, disrupt digestive processes, and increase catabolism, thereby negatively affecting overall health. Conversely, regular meal intake stabilizes blood glucose levels and supports PNS activity, which is responsible for relaxation and proper functioning of the gastrointestinal system. Parasympathetic activation facilitates blood pressure reduction, lowers the risk of cardiovascular diseases, and promotes efficient digestion and nutrient absorption [73, 74]. Consistent meal timing can also support the synchronization of biological rhythms, which positively influences ANS function.

Chrononutrition adapting meal timing to circadian rhythms — may improve metabolic health and balance. The regularity and frequency of meals have a significant impact on the functioning of the ANS. Maintaining consistent, healthy eating habits can reinforce the balance between sympathetic and parasympathetic systems, contributing to better well-being [75]. The concept of chrononutrition, which encompasses the timing of meals and their composition, and its impact on the circadian rhythm of the organism. The endogenous and exogenous factors can modulate the function of the biological clock, such as age, sleep, and shift work. The importance of meal timing and quality in regulating circadian rhythms is confirmed. The consequences of synchronization or desynchronization of this rhythm will be for metabolic health, including glycemic control, body mass index (BMI), and the risk of developing diabetes type 2 [75].

Conclusion

This study demonstrated that certain dietary components, such as caffeine, simple sugars, and high-glycemic-index foods enhance the activity of the sympathetic nervous system, leading to increased blood pressure, accelerated heart rate, and elevated catecholamine secretion. Conversely, substances such as omega-3 polyunsaturated fatty acids, magnesium, and choline exert

a protective effect on the nervous system and strengthen parasympathetic activation, promoting relaxation, reducing nervous tension, and improving digestive processes. The data obtained suggest that appropriate modifications in dietary habits can serve as an effective, non-invasive component of the prevention and treatment of ANS balance disorders. An interdisciplinary approach combining knowledge from dietetics, neurophysiology, and health psychology enables the optimization of nutritional therapy in the context of improving overall homeostasis and reducing the symptoms of excessive sympathetic or parasympathetic activity.

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Conflict of interest

None declared.

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