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Review / Derleme

The Effects of Taurine on Energy Homeostasis and Health: A Nutritional Perspective

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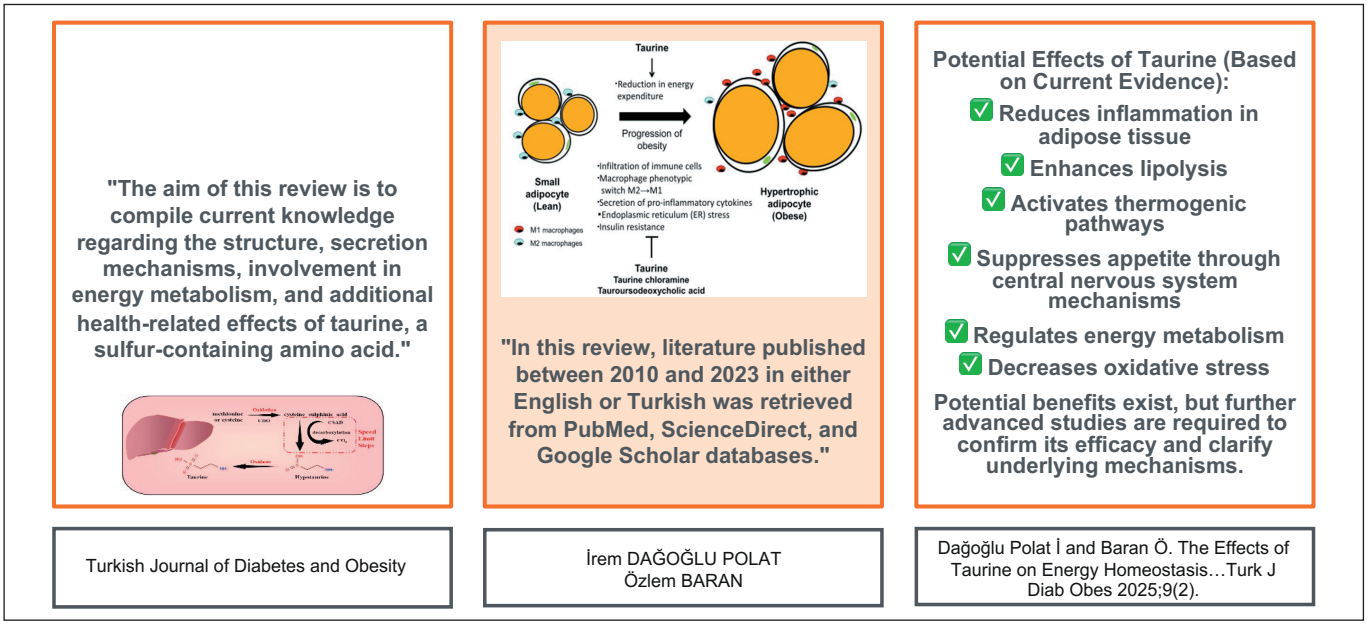
GRAPHICAL ABSTRACT



ABSTRACT

Taurine is a sulfur-containing β-amino acid that structurally differs from standard amino acids. It is found in high concentrations across various tissues of the human body and plays a role in numerous physiological processes, including antioxidant defense, energy regulation, and modulation of the central nervous system. Globally increasing obesity is accompanied by serious health problems such as metabolic syndrome, insulin resistance, dyslipidemia, and hyperglycemia. While traditional approaches have regarded adipose tissue as a passive structure solely involved in lipid storage, it is now recognized as an active endocrine organ. In this context, the biologically active molecules secreted by adipocytes and their roles in metabolic processes are critically important in the pathogenesis of obesity. The primary aim of this review article is to systematically evaluate the functional effects of taurine in adipose tissue and the mechanisms by which it counteracts obesity. Taurine possesses a structure that distinguishes it from typical amino acids and is present in high concentrations in the body, contributing to various biological functions such as reducing oxidative stress, suppressing inflammation, and regulating energy metabolism. Studies in animal models have demonstrated that taurine supplementation reduces inflammation in adipose tissue, increases lipolysis, activates thermogenic pathways, and suppresses appetite via central nervous system mechanisms,

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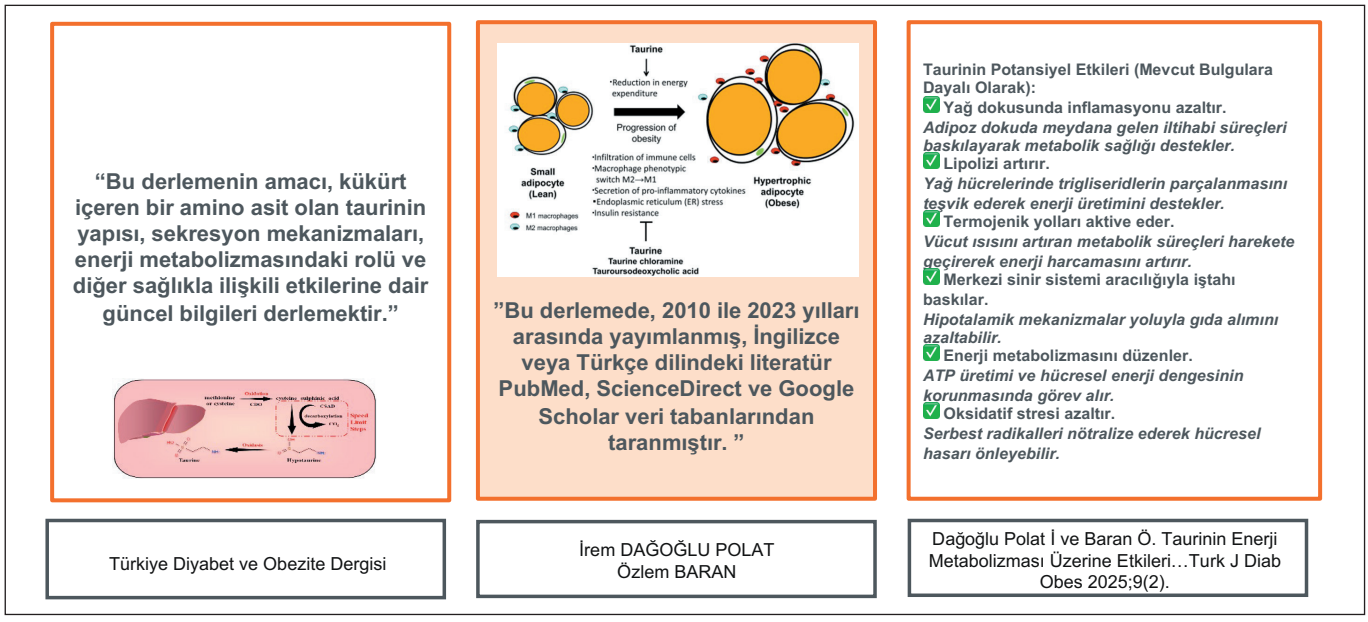
thereby preventing the development of obesity. Moreover, findings of low plasma taurine levels in individuals living with obesity and diabetes suggest that taurine deficiency may disrupt metabolic balance. Although animal and epidemiological studies indicate that taurine is promising for alleviating metabolic disorders, further advanced research is necessary to fully elucidate its mechanisms and confirm its efficacy in humans.

Keywords: *Adipose Tissue, Obesity, Taurine*

Taurinin Enerji Homeostazi ve Sağlık Üzerindeki Etkileri: Beslenme Perspektifi

GRAFİKSEL ÖZET



ÖZ

Taurin, standart amino asitlerden yapısal olarak farklılık gösteren, sülfür içeren bir β-amino asittir. İnsan vücudunun çeşitli dokularında yüksek konsantrasyonlarda bulunur ve antioksidan savunma, enerji regülasyonu ve merkezi sinir sisteminin düzenlenmesi gibi çok sayıda fizyolojik süreçte rol alır. Küresel olarak artan obeziteye metabolik sendrom, insülin direnci, dislipidemi ve hiperglisemi gibi ciddi sağlık sorunları eşlik etmektedir. Geleneksel yaklaşımlar yağ dokusunu sadece lipid depolamada rol alan pasif bir yapı olarak görürken, artık aktif bir endokrin organ olarak kabul edilmektedir. Bu bağlamda, adipositler tarafından salgılanan biyolojik olarak aktif moleküller ve bunların metabolik süreçlerdeki rolleri obezitenin patogenezinde kritik öneme sahiptir. Bu derleme makalesinin temel amacı, taurinin adipoz dokudaki fonksiyonel etkilerini ve obeziteye karşı koyma mekanizmalarını sistematik olarak değerlendirmektir. Taurin, onu tipik amino asitlerden ayıran bir yapıya sahiptir ve vücutta yüksek konsantrasyonlarda bulunur, oksidatif stresi azaltmak, inflamasyonu bastırmak ve enerji metabolizmasını düzenlemek gibi çeşitli biyolojik işlevlere katkıda bulunur. Hayvan modellerinde yapılan çalışmalar, taurin takviyesinin yağ dokusundaki enflamasyonu azalttığını, lipolizi artırdığını, termojenik yolları aktive ettiğini ve merkezi sinir sistemi mekanizmaları yoluyla iştahı baskıladığını ve böylece obezite gelişimini önlediğini göstermiştir. Ayrıca, obezite ve diyabetle yaşayan bireylerde düşük plazma taurin düzeylerinin saptanması, taurin eksikliğinin metabolik dengeyi bozabileceğine işaret etmektedir. Her ne kadar hayvan ve epidemiyolojik çalışmalar taurinin metabolik bozuklukları hafifletmek için umut verici olduğunu gösterse de, mekanizmalarını tam olarak aydınlatmak ve insanlardaki etkinliğini doğrulamak için daha ileri araştırmalara ihtiyaç vardır.

Anahtar Sözcükler: *Obezite, Taurin, Yağ dokusu*

INTRODUCTION

Taurine, a sulfur-containing β-amino acid structurally distinct from standard amino acids, accumulates in high concentrations in various human tissues and contributes to key physiological processes such as antioxidant defense, energy regulation, and modulation of the central nervous system (1).

Researchers have directly linked the rapid global increase in obesity to metabolic syndrome, which encompasses metabolic disorders such as dyslipidemia, insulin resistance, and hyperglycemia. The expansion of adipose tissue reflects a persistent positive energy balance resulting from a sedentary lifestyle shaped by genetic predisposition and environmental factors. In earlier models, scientists regarded adipose tissue as a passive lipid reservoir. However, current scientific paradigms recognize it as a metabolically active endocrine organ that synthesizes a wide range of bioactive molecules (2).

These biologically active molecules released from adipose tissue are termed adipocytokines, which include leptin, adiponectin, and resistin. Leptin, predominantly secreted by white adipose tissue, plays a central role in the regulation of energy balance by acting on the hypothalamus to inhibit appetite and increase energy expenditure. In the context of obesity, circulating leptin levels are paradoxically elevated; however, this is often accompanied by leptin resistance, a condition in which target tissues fail to respond adequately to leptin's anorexigenic effects, thereby contributing to persistent hyperphagia and weight gain. Adiponectin, in contrast, exhibits anti-inflammatory, anti-atherogenic, and insulin-sensitizing properties. It enhances fatty acid oxidation, improves glucose uptake in skeletal muscle, and suppresses hepatic gluconeogenesis. Notably, adiponectin levels are inversely correlated with adiposity and are significantly reduced in individuals with obesity and type 2 diabetes. Resistin, originally identified as an adipocyte-derived hormone linked to insulin resistance, is now known to be primarily produced by macrophages in humans. It promotes the expression of pro-inflammatory cytokines such as TNF-α and IL-6 and is implicated in the development of insulin resistance, systemic inflammation, and endothelial dysfunction. Collectively, these adipocytokines serve as key mediators linking adipose tissue dysfunction to metabolic and inflammatory pathways involved in obesity-related complications (3-5).

These molecules play key roles in insulin sensitivity, nutrient metabolism, inflammatory responses, and stress regulation. In obesity, macrophages infiltrate adipose tissue and trigger chronic low-grade inflammation through increased production of proinflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and monocyte chemoattractant protein-1 (MCP-1). This chronic inflammatory state significantly contributes to the development of insulin resistance and obesity-related complications (6).

As outlined in the previous section, certain bioactive compounds that adipose tissue secretes play a pivotal role in the development of chronic inflammation and obesity-related complications. In this context, recent studies have identified taurine as a promising regulatory molecule with therapeutic potential for managing metabolic disorders. Among all free amino acids in mammals, taurine exhibits the highest intracellular concentrations, typically ranging from 20 to 50 mM—markedly higher than the micromolar levels seen for most other amino acids. Taurine plays a central role in maintaining cellular homeostasis, as the body synthesizes it from cysteine and also obtains it through dietary sources. Beyond its classical function in bile acid conjugation, recent research has emphasized taurine's diverse physiological and pharmacological effects. These include anti-obesity properties, enhanced insulin sensitivity, suppression of macrophage infiltration into adipose tissue, regulation of endoplasmic reticulum stress, antioxidant and anti-inflammatory effects, and neuromodulatory actions (7,8).

This study aims to investigate the functional roles of taurine in adipose tissue, with a particular emphasis on its involvement in the development of obesity and the regulation of energy homeostasis in both human and animal models.

TAURINE

Taurine derives its name from the Latin word taurus, meaning bull, and is etymologically linked to the Ancient Greek term ταῦρος (taûros). It was first isolated in 1827 by German scientists Leopold Gmelin and Friedrich Tiedemann from the bile of Bos taurus (ox bile), a discovery that not only contributed to the understanding of bile constituents but also gave the compound its name. Taurine is a β-amino sulfonic acid found in high concentrations in the brain, retina, heart, skeletal muscle, and leukocytes, where it plays essential roles in various physiological processes (9).

Taurine is predominantly concentrated in oxidative tissues that are rich in mitochondria, such as cardiac and skeletal muscle, whereas its levels are comparatively lower in glycolytic tissues. The distribution of taurine varies markedly across human tissues, reflecting its involvement in tissue-specific metabolic functions. Chemically, taurine ($C_2H_7NO_3S$) is classified as a β-amino acid with a molecular weight of 125.15 g/mol. Its molecular structure consists of

an amino group (NH_2) and a sulfonic acid group (SO_3H) attached to the β-carbon, notably lacking a carboxyl group. This unique structural configuration enables taurine to remain unbound as a free amino acid within cells, thereby allowing significant functional versatility, particularly in cellular signaling, osmoregulation, and mitochondrial homeostasis (9,10).

Taurine's isoelectric point is approximately pH 5.12. Its strong acidic nature allows it to remain highly soluble in water across physiological pH ranges. Although taurine is structurally similar to amino acids, it lacks a carboxyl group and possesses a beta-amino configuration, features that prevent its incorporation into protein synthesis (11).

Dietary Sources and Biosynthesis of Taurine

Taurine is present in numerous food sources, and daily intake ranges from approximately 40 to 400 mg depending on dietary habits. Rich dietary sources include fish, milk, eggs, and meat. Consequently, individuals adhering to vegetarian diets typically have lower taurine intake. The primary dietary sources of taurine and the corresponding concentrations found in each food item are detailed in Figure 1 (9,12, 13).

Taurine is chiefly produced in the liver and kidneys, yet it is also distributed across various tissues, including the brain, retina, heart, placenta, leukocytes, and skeletal muscles. Its biosynthesis originates from the metabolic processing of cysteine and methionine. In the principal pathway, cysteine undergoes oxidation via cysteine dioxygenase (CDO), yielding cysteine sulfinic acid. This compound is subsequently decarboxylated by cysteine sulfinic acid decarboxylase (CSAD) to generate hypotaurine, which is further oxidized to taurine through the action of hypotaurine dehydrogenase. An alternative trans-sulfuration route converts homocysteine into cystathionine, followed by sequential enzymatic reactions involving cystathionine γ-lyase (CGL), CDO, and CSAD, ultimately leading to taurine formation (Figure 2) (13,14).

Toxicological Effects of Taurine

Taurine, a naturally occurring amino acid, shows minimal adverse effects in the body. Toxicity studies have reported that taurine is neither genotoxic, carcinogenic, nor teratogenic (15). Some studies have defined tolerable upper intake levels based on the no-observed-adverse-effect level (NOAEL). For instance, intravenous administration of 1000–2000 mg/kg/day for 13 weeks to increased water con-

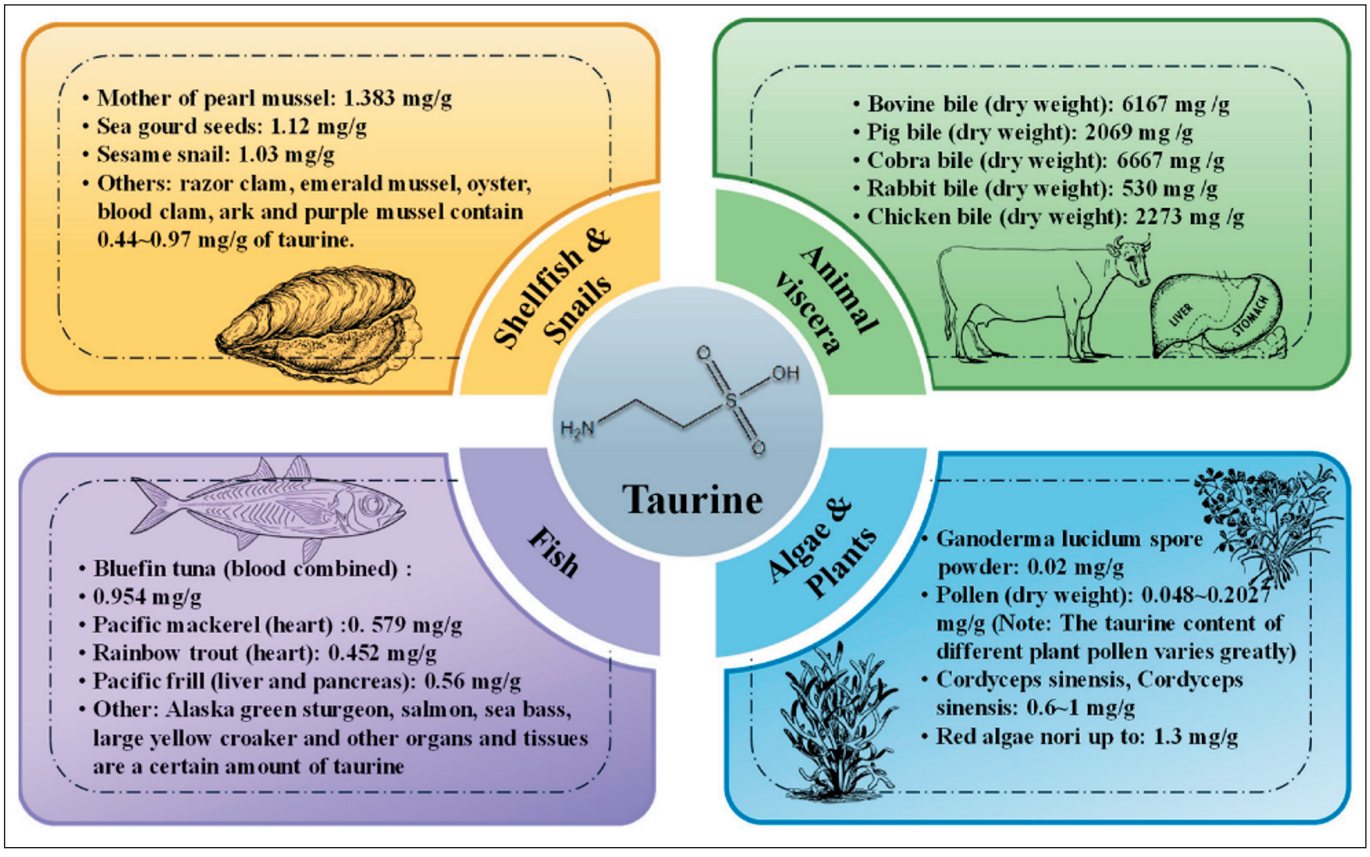


Figure 1: Main dietary sources of taurine and the proportion of taurine in each food.

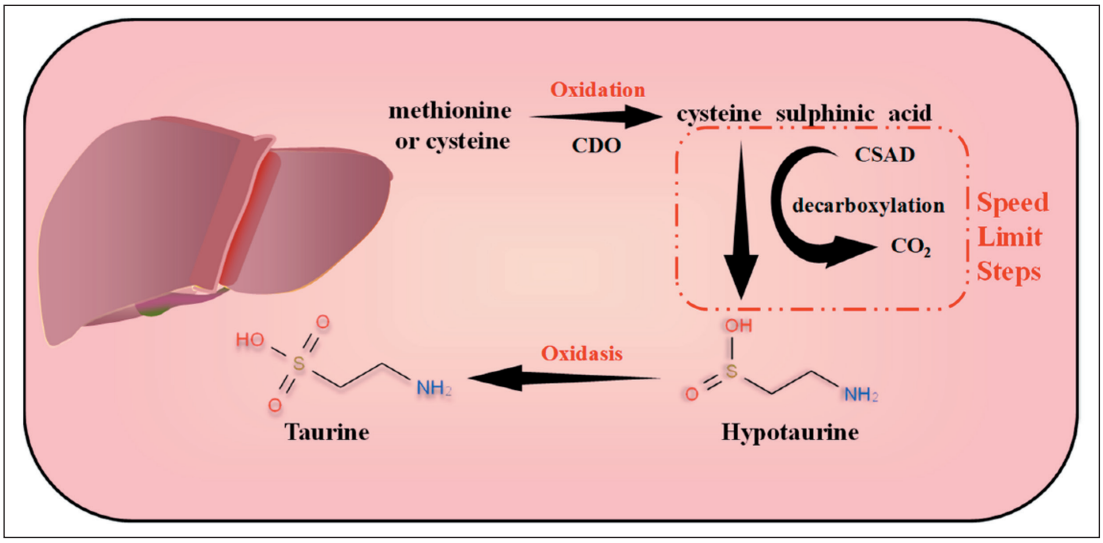


Figure 2: Taurine synthesis pathway in the liver.

sumption and hemosiderin deposition in rat lungs. Almohaimeed et al. reported a NOAEL of 500 mg/kg/day, whereas observed hepatic lipid infiltration in guinea pigs at 462 mg/kg/day over two weeks (16). A risk assessment concluded that a maximum dose of 3 g/day posed no toxicological concern in clinical studies. According to the VKM Report 2015:22, titled Risk Assessment of "Other Substances" Taurine, a NOAEL of 1000 mg/kg/day was identified for taurine based on subchronic toxicity studies in animals. (17).

A study investigated the effects of taurine on the brain tissue energy status and lipid peroxidation in a guinea pig model of endotoxemia. Endotoxemia significantly reduced the ATP/ADP ratio and taurine levels in the brain, while increasing malondialdehyde (MDA) levels, a marker of lipid peroxidation. However, taurine administration did not exhibit the anticipated antioxidant and neuroprotective effects; on the contrary, it may have contributed to neuronal damage at the administered dose. These findings suggest that taurine, when used at certain doses, may exert toxic rather than neuroprotective effects (18).

In another experimental trial, the administration of a high taurine dose (6 g) was assessed for its influence on performance-related variables, namely time to exhaustion (TTE), maximal oxygen uptake (VO_2max), maximal heart rate (HRmax), and perceived exertion (RPE), during treadmill exercise of progressively increasing intensity. The study included 10 well-trained male endurance athletes and employed a double-blind, randomized, crossover design. Participants were given a sugar-free lemonade mixed with either taurine or placebo approximately 90 minutes before the exercise trial. The treadmill protocol began at a speed of 6 km/h, with both speed and incline increasing over time. The results indicated that taurine had no significant effect on TTE or HRmax, although a non-significant 2% increase in VO_2max was observed. These findings suggest that high-dose taurine supplementation does not confer a notable benefit to endurance performance (19).

Biological Functions of Taurine

Taurine's distinct physicochemical characteristics enable it to act as a modulator in a variety of essential biological processes, including protection against oxidative stress, regulation of protein phosphorylation and calcium homeostasis, antioxidant defense, membrane stabilization, bile acid conjugation, lipid metabolism, and glucose control. Notably, taurine exerts pleiotropic effects across multiple organ systems, including the cardiovascular system, skeletal muscle, retina, liver, kidneys, and central nervous system (20,21).

Taurine exerts its cellular antioxidant effects primarily through scavenging peroxyl radicals, nitric oxide, and superoxide anions—though it is ineffective against hydrogen peroxide. This free radical scavenging activity increases with concentration and reaches its peak efficacy at approximately 60 mM (22).

A recent study comparatively investigated the antiproliferative, antimigratory, and antioxidant effects of the compounds capsaicin, β-carotene, melatonin, and taurine on healthy cells (L929 fibroblasts) and breast cancer cells (MCF-7). Cell viability, migration, and oxidant/antioxidant levels were assessed following treatment with various concentrations of these compounds. The results demonstrated that all compounds significantly reduced the migratory capacity of MCF-7 cells, particularly after 48 hours of exposure. Furthermore, β-carotene emerged as the most effec-

tive compound in terms of antioxidant activity, although it also exhibited pro-oxidant properties at high concentrations. These findings suggest that the aforementioned compounds may hold potential as anticancer agents for breast cancer treatment (23).

Taurine has been shown to play a significant cytoprotective role in reproductive physiology. Experimental studies on rabbit spermatozoa have demonstrated that taurine exerts a strong antioxidant effect, particularly against highly reactive species such as hypochlorite (HOCl) molecules and hydroxyl (OH) radicals. It also provides moderate protection against hydrogen peroxide (H_2O_2) and superoxide (O_2^-) radicals. These findings suggest that taurine may contribute to the preservation of sperm function and viability by mitigating oxidative stress in the male reproductive system (24).

In skeletal muscle physiology, taurine serves multiple essential functions related to contractile and regenerative capacity. It plays a key role in calcium ion–dependent excitation–contraction coupling, regulates cellular volume, and enhances myogenesis. These effects become especially critical in aging or disease states, where a decline in endogenous taurine levels is commonly observed. Under such conditions, taurine supplementation has been shown to support muscle repair and maintain functional integrity (25).

The role of taurine in skeletal muscle function has been widely explored in scientific research. According to De Luca et al., taurine contributes to the modulation of muscle cell excitability and contractile activity by stabilizing cellular membranes, maintaining osmotic balance, and regulating intracellular calcium homeostasis. Additionally, taurine has been implicated in influencing metabolic pathways and gene expression within muscle tissue, thereby promoting adaptive and cytoprotective responses. Notably, in pathological conditions such as disuse-induced muscle atrophy and various myopathies, disturbances in intramuscular taurine concentrations have been observed. Preclinical evidence indicates that taurine supplementation may help restore muscle performance by enhancing contractile function and sustaining electrical excitability through its effects on ion channel regulation. These findings suggest that taurine holds therapeutic promise for managing muscle-related disorders (26).

In an experimental study, the effects of taurine on inflammatory muscle injury were investigated using a myositis model induced by acetic acid in Wistar albino rats. The animals were divided into three groups: control, myositis, and myositis + taurine (500 mg/kg/day), and taurine treatment was administered for 15 days. Histopathological and immunohistochemical analyses revealed significant improvements in parameters such as degeneration, necrosis, inflammation, and structural disorganization of muscle tissue, particularly in the taurine-treated group ($p<0.01$). Moreover, a significant reduction was observed in the levels of caspase-3, a marker of apoptosis. These findings suggest that taurine, through its anti-inflammatory and anti-apoptotic properties, may serve as a potential therapeutic agent in mitigating myositis-induced muscle damage (27).

In the cardiovascular system, taurine modulates apolipoprotein B100 levels and enhances myocardial contractility, which may reduce the severity of atherosclerosis and coronary heart disease. A recent meta-analysis suggests taurine may exert antihypertensive effects in humans (28).

An experimental study assessing taurine's protective role against oxidative stress, inflammation, and endothelial dysfunction in the cardiac tissue of diabetic rats reported several favorable outcomes. In the diabetic state, upregulation of the proinflammatory transcription factor NF-κB was accompanied by diminished expression of pivotal antioxidant and endothelial regulatory molecules, including nuclear factor erythroid 2–related factor 2 (Nrf2), heme oxygenase-1 (HO-1), endothelial nitric oxide synthase (eNOS), and sirtuin-1 (SIRT-1). Taurine supplementation partially reinstated the expression of these molecular targets, highlighting its regulatory influence on redox-sensitive signaling mechanisms. Additionally, cardiac oxidative injury associated with diabetes—indicated by elevated malondialdehyde (MDA) concentrations and reduced activities of antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx)—was markedly alleviated following taurine administration. Taken together, these results indicate that taurine mitigates oxidative injury, downregulates inflammatory mediators, and enhances endothelial function in the heart, supporting its therapeutic potential for preventing and managing cardiovascular complications linked to diabetes (29).

Taurine is integral to kidney homeostasis, serving as an efficient osmolyte while influencing fundamental cellular mechanisms such as apoptosis and regulation of the cell cycle.Its presence contributes to fluid–electrolyte balance and cytoprotection under physiological and pathological conditions. Additionally, taurine is essential for normal retinal development and calcium signaling, highlighting its broad physiological relevance across multiple organ systems (20). A recent experimental study further elucidated taurine's renoprotective effects in the setting of cold ischemia, a major challenge in kidney transplantation. In this model, kidneys harvested from rats were stored for 72 hours at 4 °C in University of Wisconsin (UW) solution with taurine supplementation. The findings indicated that taurine ad-

ministration led to a significant decline in malondialdehyde (MDA) concentrations, reflecting reduced lipid peroxidation and mitigation of oxidative damage. Concurrently, the activities of key antioxidant enzymes—superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx)—were substantially enhanced. Histopathological assessment further demonstrated that taurine treatment lessened morphological alterations, including tubular necrosis and glomerular lesions. Collectively, these outcomes imply that taurine confers protection to renal tissue by counteracting oxidative stress and preserving both its architecture and functional capacity. Within the framework of advanced transplantation techniques and extended organ preservation, taurine supplementation could represent a viable approach to minimizing post-transplant complications and enhancing graft survival (30).

Though it cannot scavenge hydrogen peroxide or hydroxyl radicals directly, taurine prevents reactive oxygen species generation, protects mitochondria from superoxide overload, and enhances electron transport chain activity. It also reduces endoplasmic reticulum (ER) stress and supports protein folding (31-33).

Taurine and Adipose Tissue: Mechanistic Insights into Anti-Obesity Effects

Recent research has highlighted taurine's pivotal influence on adipose tissue physiology, especially in the context of obesity and related metabolic impairments. Within adipose depots, taurine mitigates obesity-associated changes by regulating adipogenesis, lipolysis, adipokine release, inflammatory signaling, and oxidative stress (7,34). Additionally, taurine participates in the modulation of ligand-activated transcription factors that govern lipid handling, energy utilization, and redox equilibrium. Of particular importance, taurine influences the function of peroxisome proliferator-activated receptor gamma (PPARγ), a nuclear receptor that plays a critical role in adipocyte maturation and the storage of lipids. Through downregulation of PPARγ, taurine suppresses adipogenesis and promotes a metabolically favorable adipocyte phenotype In addition, taurine-induced activation of nuclear factor erythroid 2–related factor 2 (NRF2) enhances antioxidant defense mechanisms, while its interaction with bile acid receptors such as farnesoid X receptor (FXR) and Takeda G-protein–coupled receptor 5 (TGR5) may further influence energy metabolism, thermogenesis, and inflammatory signaling pathways (35,36).

Evidence indicates that taurine suppresses the differentiation of adipocytes by downregulating peroxisome proliferator-activated receptor gamma (PPARγ) and CCAAT/enhancer-binding proteins (C/EBPs), leading to decreased lipid deposition in preadipocytes. In a study by Kim et al., taurine treatment markedly inhibited adipogenesis in white adipose tissue (WAT) while leaving thermogenic brown adipose tissue (BAT) unaffected, underscoring its tissue-specific modulatory actions (37,38).

In models of high-fat diet–induced obesity, taurine supplementation ameliorated systemic inflammation by reducing the expression of TNF-α, IL-6, and MCP-1 in adipose tissue (33). This anti-inflammatory action is partly attributed to taurine chloramine, an endogenous metabolite known to inhibit nuclear factor kappa B (NF-κB) signaling and suppress proinflammatory gene transcription (39). Furthermore, taurine-treated adipocytes exhibit enhanced expression of adiponectin, an insulin-sensitizing and anti-inflammatory adipokine often reduced in obesity (34).

Neonatal exposure to monosodium glutamate (MSG) in rodents impairs the regulation of appetite and energy balance, promoting the onset of obesity, glucose intolerance, and insulin resistance. The potential anti-obesity actions of taurine have been examined using this experimental model. In Wistar rats with MSG-induced obesity, dietary taurine supplementation (2.5% in drinking water for 70 days) led to reduced fat deposition in retroperitoneal and periepididymal adipose depots, alongside lowered lipid concentrations in both plasma and hepatic tissues. These results indicate that taurine may confer favorable metabolic effects by influencing lipid storage mechanisms and enhancing lipid profiles in obesity models (40).

Epidemiological Research Has İncreasingly Highlighted Taurine's Potential Anti-Obesity And Cardiometabolic Regulatory Properties

Adipose tissue is now understood to function not solely as a passive reservoir for energy storage but as an active endocrine organ with diverse regulatory roles. While the liver and kidneys are classically regarded as the primary sites of taurine production, experimental evidence has revealed that epididymal and perirenal white adipose depots exhibit markedly elevated expression of cysteine dioxygenase (CDO) and cysteine sulfinic acid decarboxylase (CSAD)—the two key enzymes involved in taurine biosynthesis. This observation indicates that white adipose tissue may play a meaningful role in maintaining systemic taurine balance. In obesity, however, the expression of these enzymes is markedly diminished, occurring in parallel with a reduction in circulating taurine concentrations. In vitro studies using 3T3-L1 preadipocytes have shown that CDO and CSAD protein levels increase markedly during differentiation into mature adipocytes, whereas hypertrophic adipocytes associated with obesity exhibit a diminished capacity for taurine

synthesis. This decline in taurine production mirrors the reduced adiponectin levels frequently observed in obesity, supporting the hypothesis that taurine may function as a novel adipokine. Thus, reduced taurine production within adipose tissue may constitute an important mechanistic contributor to the development of obesity and its related metabolic derangements. Figure 3 provides a schematic overview illustrating taurine's regulatory influence on adipose tissue dysfunction and the inflammatory processes linked to obesity (41,43)

Figure 3 presents a mechanistic overview of taurine's role in modulating adipose tissue function along the continuum from metabolic health to obesity. In metabolically healthy, lean individuals, small adipocytes demonstrate active taurine biosynthesis, which supports systemic energy balance and sustains an anti-inflammatory milieu dominated by M2 macrophages. As obesity develops, adipocytes enlarge (hypertrophy), accompanied by reductions in both energy expenditure and taurine production. This pathological shift fosters increased immune cell infiltration—particularly of pro-inflammatory M1 macrophages—alongside a transition from the M2 to the M1 macrophage phenotype. Consequently, local inflammation intensifies, with elevated secretion of cytokines such as TNF-α and IL-6, activation of endoplasmic reticulum (ER) stress pathways, and the emergence of insulin resistance. Taurine and its bioactive derivatives—taurine chloramine and tauroursodeoxycholic acid (TUDCA)—act to mitigate these adverse changes by promoting M2 macrophage polarization, inhibiting pro-inflammatory signaling cascades, and reducing ER stress, thereby restoring adipose tissue functionality and improving overall metabolic health (41).

In the context of obesity, mitochondrial dysfunction in white adipose tissue contributes to impaired fatty acid oxidation and reactive oxygen species (ROS) accumulation. Taurine mitigates these adverse effects by stabilizing mitochondrial membranes, enhancing electron transport chain efficiency, and reducing superoxide generation. Surai et al. showed that taurine at physiological concentrations scavenges ROS including nitric oxide and superoxide anions, thus preserving redox balance (21,32).

Importantly, the CARDIAC (Coronary Artery Disease in Asian Communities) Study—conducted across 61 population groups in 25 countries—assessed 24-hour urinary taurine excretion as an indicator of dietary taurine intake. Findings demonstrated a strong inverse correlation between urinary taurine concentrations and mortality from coronary heart disease, supporting the notion that taurine may exert a protective influence on cardiovascular health outcomes (44).

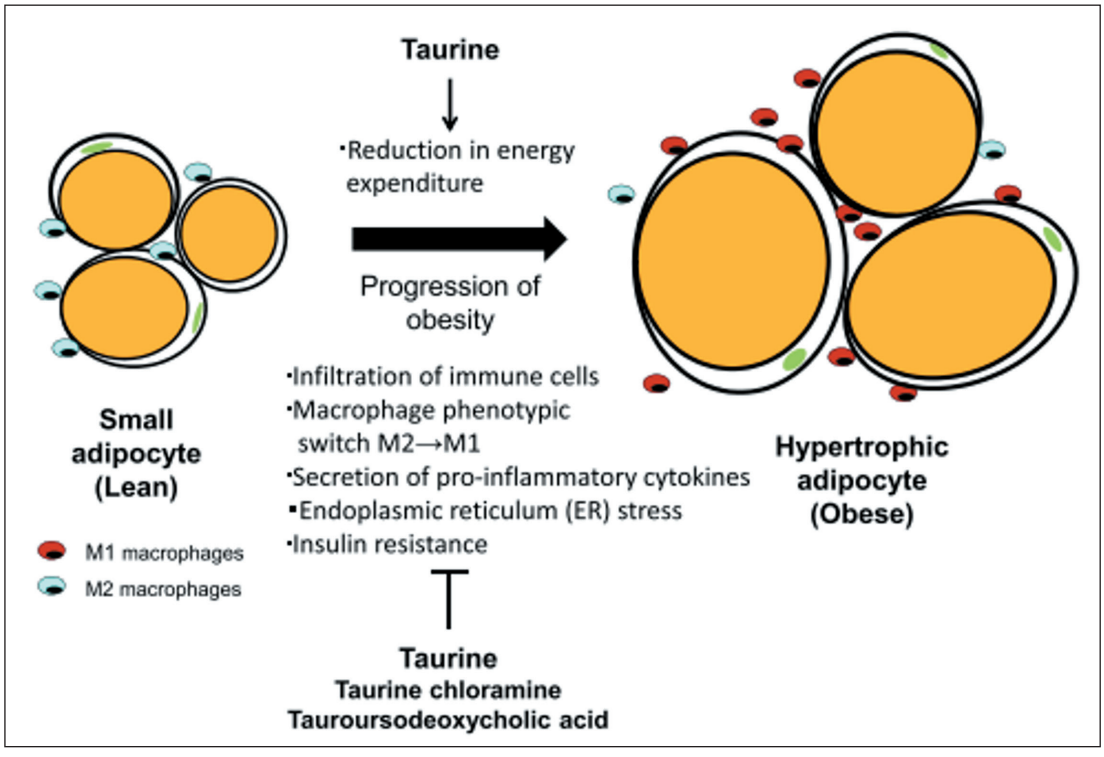


Figure 3: Taurine and its derivatives attenuate adipose tissue inflammation, macrophage polarization, and metabolic dysfunction during obesity progression.

In addition to cardiovascular outcomes, higher urinary taurine excretion was also associated with lower body mass index (BMI), systolic and diastolic blood pressure, and plasma total cholesterol levels, suggesting a protective effect on metabolic health (45).

Moreover, individuals with higher fish consumption—known to be a primary dietary source of taurine—exhibit greater systemic taurine levels compared to those following predominantly meat-based or vegetarian diets. These findings point to the dietary origin and inter-individual variability in taurine status, which may modulate the compound's metabolic efficacy. In this context, the therapeutic potential of taurine in humans is likely to be influenced by factors such as dosage, duration of supplementation, baseline taurine status, and genetic or lifestyle-related metabolic risk factors (46)

Taurine has been shown to improve insulin signaling in adipose tissue by increasing the phosphorylation of Akt and reducing endoplasmic reticulum (ER) stress–related markers such as CHOP and GRP78. These effects collectively contribute to enhanced glucose uptake and improved insulin responsiveness, particularly under metabolic stress conditions (47).

Moreover, taurine alleviates endoplasmic reticulum (ER) stress, a key contributor to insulin resistance and metabolic inflammation in obesity. ER stress triggers the unfolded protein response (UPR), leading to upregulation of stress markers such as GRP78, CHOP, and XBP1 (48). Taurine suppresses these markers by modulating redox homeostasis and preventing calcium dyshomeostasis between the ER and mitochondria (49). Taurine supplementation significantly reduced ER stress markers and improved hepatic insulin signaling in models of nonalcoholic fatty liver disease, suggesting systemic benefits beyond adipose tissue (50).

Mitochondrial biogenesis and oxidative metabolism in adipose tissue are further influenced by taurine via upregulation of peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α) (51).

Taurine has been increasingly recognized for maintaining mitochondrial integrity and regulating energy expenditure, particularly within metabolically active tissues such as adipose tissue. Taurine enhances mitochondrial biogenesis and promotes oxidative phosphorylation by modulating transcriptional regulators such as PGC-1α, nuclear respiratory factors (NRFs), and mitochondrial transcription factor A (TFAM) (52).

Regulation of Appetite and Energy Balance: The Role of Taurine, Central Nervous System, and Bile Acid Signaling

Specifically, taurine has been reported to modulate the hypothalamic arcuate nucleus—a central hub for controlling energy intake—by enhancing the responsiveness of anorexigenic signaling pathways and concurrently downregulating orexigenic mediators, including neuropeptide Y (NPY) and agouti-related peptide (AgRP). This dual action ultimately contributes to a decrease in food consumption (53).

Taurine plays a role in bile acid signaling pathways by modulating bile acid metabolism and receptor activation. Bile acids function not only in digestion but also act as endocrine regulators via G-protein-coupled receptors (e.g., TGR5) and nuclear receptors such as farnesoid X receptor (FXR) (54). Taurine-conjugated bile acids (e.g., taurocholic acid) have high affinity for TGR5, which is expressed in the CNS, adipose tissue, and intestinal epithelium. Activation of TGR5 enhances energy expenditure, mitochondrial function, and secretion of glucagon-like peptide-1 (GLP-1), contributing to improved metabolic homeostasis (7,55).

Choudhuri and Klaassen reported that specific bile acid-sensitive G-protein coupled receptors respond more strongly to taurine-conjugated bile acids than to glycine-conjugated forms (56). This receptor activity promotes brown adipose tissue thermogenesis, increases mitochondrial uncoupling protein 1 (UCP1) expression, and facilitates glucose disposal, making taurine a potential modulator of bile acid–mediated metabolic effects (57).

These neurometabolic mechanisms collectively suggest that taurine may serve as a central and peripheral integrator of signals involved in energy intake and expenditure, positioning it as a candidate for novel interventions targeting neuroendocrine pathways in obesity management (53).

CONCLUSION

Taurine is a multifaceted amino sulfonic acid that exhibits a wide range of physiological functions relevant to metabolic health, particularly in the context of obesity and energy regulation. This review highlights taurine's significant influence on adipose tissue remodeling, anti-inflammatory and antioxidant defense, mitochondrial and endoplasmic reticulum homeostasis, insulin sensitivity, and central appetite regulation.

Both experimental and clinical data support taurine's potential to ameliorate obesity-related metabolic disorders.

Its ability to modulate adipocyte differentiation, suppress macrophage-driven inflammation, enhance mitochondrial function, and improve insulin signaling positions it as a promising agent in the nutritional and pharmacological management of metabolic syndrome. Notably, taurine's interaction with bile acid receptors such as TGR5 and its effects on gut-brain axis signaling pathways further expand its relevance in energy homeostasis.

Despite compelling evidence from animal and cellular studies, human trials examining taurine's effects on adiposity and metabolic risk remain limited. The variability in dietary taurine intake across populations, particularly in vegetarians and individuals with chronic diseases, necessitates further evaluation of optimal intake levels and supplementation protocols. Moreover, the precise molecular mechanisms through which taurine regulates transcription factors, adipokines, and nutrient sensors in adipose and neural tissues warrant deeper investigation.

In future research, randomized controlled trials with well-characterized cohorts are essential to confirm taurine's efficacy and safety in treating obesity and its comorbidities. Advances in omics technologies may also provide new insights into taurine's personalized applications in metabolic health. Ultimately, taurine's diverse biological effects and low toxicity profile make it a compelling candidate for integrative strategies targeting metabolic homeostasis.

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Conflict of Interest
The authors declare no conflict of interest.

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