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Pharmacokinetic, toxicological, and bioactivity profile of taurine in cardiovascular health and disease management

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ABSTRACT

Taurine (2-aminoethanesulfonic acid) is a naturally occurring sulfur-containing amino acid that plays critical roles in cellular homeostasis, particularly in the cardiovascular and central nervous systems. It functions in osmoregulation, membrane stabilization, and ion transport. Taurine's antioxidant and anti-inflammatory properties have garnered attention for their potential to protect against cardiovascular diseases, where oxidative stress and inflammation are key contributors. This study evaluates taurine's pharmacokinetic properties using in silico methods, including ADME (absorption, distribution, metabolism, and excretion) prediction and STopTox toxicity analysis, as well as *in vitro* anti-inflammatory and antioxidant assays. The results reveal taurine's favorable ADME profile, with high water solubility, moderate permeability, and minimal metabolic interactions, suggesting a promising safety profile for clinical use. The STopTox analysis further supports its low toxicity risk, aside from minor concerns regarding skin and eye irritation. *In vitro* assays demonstrated taurine's ability to inhibit protein denaturation and scavenge free radicals, including hydrogen peroxide, superoxide anions, and nitric oxide. These findings align with taurine's well-established cytoprotective and anti-inflammatory properties, highlighting its therapeutic potential for cardiovascular health. Taurine exhibits strong anti-inflammatory and antioxidant activities, a promising pharmacokinetic profile, and low toxicity, positioning it as a valuable candidate for managing cardiovascular diseases. Further clinical research is needed to confirm its therapeutic efficacy.

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INTRODUCTION

Taurine (2-aminoethanesulfonic acid) is a naturally occurring sulfur-containing amino acid (Fig. 1) derivative that is widely distributed in mammalian tissues. It is particularly abundant in metabolically active organs such as the heart, retina, brain, and skeletal muscle (Zhao and Qi, 2025). Unlike most amino acids, taurine is not incorporated into proteins during translation; instead, it exists mainly in its free form within intracellular fluids. This distinctive property enables taurine to participate in a wide range of physiological processes essential to cellular homeostasis. Taurine plays important roles in osmoregulation, stabilization of biological membranes, modulation of ion transport, and bile acid conjugation in the liver (Zhao and Qi, 2025; Kp and Martin, 2023). Through these functions, taurine helps maintain normal metabolic activity and cellular integrity.

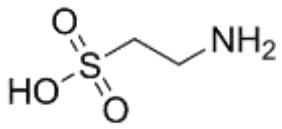

Fig. 1. Structure of taurine

Moreover, taurine is involved in the development and protection of the central and cardiovascular systems, underscoring its importance for human health and physiological regulation (Kp and Martin, 2023).

In recent years, taurine has gained significant attention in biomedical and pharmacological research due to its potential protective effects against cardiovascular diseases. Cardiovascular disorders remain a leading cause of global morbidity and mortality, and oxidative stress, along with chronic inflammation, are considered major factors in the development of endothelial dysfunction and atherosclerosis (Donia and Khamis, 2021; Jim *et al.*, 2025). Taurine exhibits strong antioxidant and anti-inflammatory properties, enabling it to scavenge reactive oxygen species and suppress inflammatory signaling pathways. By reducing oxidative damage and inflammation in vascular tissues, taurine helps preserve endothelial function and prevents vascular injury.

In addition to its antioxidant properties, taurine also plays a crucial role in regulating vascular tone and lipid metabolism. These functions are particularly important in the prevention and management of hypertension and coronary heart disease. Studies have demonstrated that taurine supplementation may improve lipid profiles, reduce blood pressure, and enhance endothelial function, thereby lowering the risk of cardiovascular complications (Nie *et al.*, 2025).

Furthermore, taurine contributes to cytoprotection by stabilizing protein structures and regulating intracellular calcium homeostasis, both of which are essential for maintaining proper cardiac contractility and muscle function (Schaffer *et al.*, 2010). Calcium regulation is particularly important in cardiac cells because it directly influences the contraction and relaxation cycles of the heart.

Despite its well-documented benefits, a comprehensive evaluation of its pharmacokinetic and toxicological profile is essential for clinical substantiation. This study investigates the ADME properties of taurine, alongside a StopTox analysis to evaluate its safety across multiple toxicity endpoints. Furthermore, we assess its bioactivity using a series of *in vitro* assays, including protein denaturation inhibition and various free radical scavenging tests, to characterize its anti-inflammatory and antioxidant potency relative to standard therapeutic agents such as aspirin and ascorbic acid.

MATERIALS AND METHODS

Chemicals and reagents

Taurine was purchased from Sigma-Aldrich, USA. Aspirin and ascorbic acid were purchased from HiMedia, Mumbai, India. All chemicals were of analytical grade, and solutions were freshly prepared prior to each experiment.

In silico pharmacokinetic and toxicity prediction

The physicochemical properties, pharmacokinetic profile, and drug-likeness of taurine were assessed

using the SwissADME web server (http://www.swissadme.ch) and ADMETlab 3.0 (https://admetlab3.scbdd.com). The canonical SMILES string for taurine was submitted to the server to generate key parameters, including lipophilicity (iLOGP), water solubility, molecular weight, and topological polar surface area. The BOILED-Egg model was used to predict gastrointestinal absorption and blood-brain barrier penetration.

Furthermore, the acute toxicity profile, covering oral, dermal, and inhalation toxicity, as well as skin and eye irritation/corrosion, was evaluated using the STopTox web portal (https://stoptox.mml.unc.edu/).

In vitro anti-inflammatory activity: Protein denaturation assay

The anti-inflammatory activity of taurine was evaluated using the egg albumin denaturation method. A reaction mixture (5 mL) was prepared by combining 0.2 mL of egg albumin (fresh hen's egg), 2.8 mL of phosphate-buffered saline (pH 6.4), and 2 mL of taurine at varying concentrations (20–320 µM). The mixture was incubated at 37°C for 15 minutes, then heated to 70°C for 5 minutes to induce denaturation. After cooling, absorbance was measured at 660 nm using a UV-Vis spectrophotometer. Aspirin (100 µM) was used as the reference standard in this assay.

The percentage protein denaturation was calculated using the formula:

$$\text{Inhibition\%} = \frac{\text{Absorbance of control} - \text{Absorbance of sample}}{\text{Absorbance of control}} \times 100$$

The IC_{50} values, representing the concentration required to inhibit 50% of the activity, were determined using regression analysis for each assay.

In vitro antioxidant assays

All *in vitro* antioxidant assays for taurine and the reference standard, Ascorbic acid, were performed at concentrations ranging from 20 to 320 µM.

The percentage free radical inhibition for each assay was calculated using the formula:

$$\text{Inhibition\%} = \frac{\text{Absorbance of control} - \text{Absorbance of sample}}{\text{Absorbance of control}} \times 100$$

The IC_{50} values, representing the concentration required to inhibit 50% of the activity, were determined using regression analysis for each assay.

Ferric reducing power assay

This assay measures taurine's ability to reduce ferricyanide (Fe^{3+}) to ferrocyanide (Fe^{2+}). The reaction mixture containing potassium ferricyanide and phosphate buffer was incubated at 50°C for 20 minutes. After the addition of trichloroacetic acid and ferric chloride, the absorbance was recorded at 700 nm.

Hydrogen peroxide scavenging assay

A 40 mM solution of H_2O_2 was prepared in phosphate buffer (pH 7.4). Taurine at concentrations ranging from 20–320 µM was added to the H_2O_2 solution. The decrease in H_2O_2 concentration was monitored by measuring absorbance at 230 nm.

Superoxide anion scavenging assay

Superoxide radicals were generated using the NADH/PMS system or photo-reduction of riboflavin. The scavenging activity was determined by inhibiting Nitroblue tetrazolium reduction, with absorbance measured at 560 nm.

Nitric oxide scavenging assay

Nitric oxide was generated from sodium nitroprusside in phosphate-buffered saline (pH 7.4). Scavenging activity was evaluated by measuring the absorbance of the resulting purple azo dye at 546 nm with the Griess reagent.

Statistical analysis

All experiments were performed in triplicate, and data are expressed as mean ± standard deviation.

RESULTS

ADME profile of taurine

The ADME profile (Fig. 2A) of taurine indicates favorable pharmacokinetic characteristics. The molecular weight of taurine is 125.01 g/mol, which falls within the optimal range for drug-like properties. Its logP value is significantly negative at -2.762, suggesting good water solubility and low membrane permeability. Taurine's topological polar surface area (TPSA) is 80.39 Å^2, indicating moderate polarity. The compound shows low permeability in both Caco-2 and MDCK cell lines, which are typically used to evaluate drug absorption across cell membranes, and has a low potential for P-glycoprotein (Pgp) inhibition or substrate activity, suggesting a reduced likelihood of interactions with drug transporters.

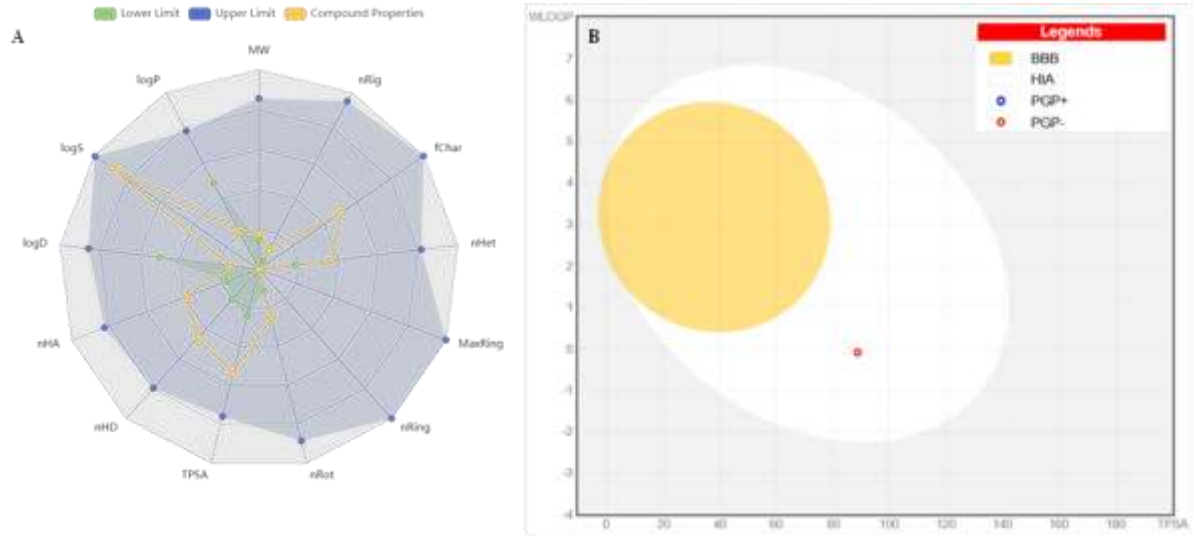

Fig. 2. A) Bioavailability radar and B) BOILED-Egg model

Additionally, taurine demonstrates high bioavailability, with a probability of over 50% bioavailability in human intestinal absorption (HIA 1.0) and a high predicted fraction unbound in plasma (Fu 89.5%). Regarding metabolism, taurine is unlikely to inhibit key cytochrome P450 enzymes (CYP1A2, CYP2C19, CYP2D6, CYP3A4, etc.), suggesting a low potential for drug-drug interactions via metabolic pathways. Its predicted stability in human liver microsomes is high, further suggesting a stable metabolic profile. Taurine's distribution is characterized by moderate plasma protein binding (PPB = 9.198%) and a very low potential to cross the blood-brain barrier (BBB = 0.026), indicating limited central nervous system exposure. Excretion predictions show that taurine has a moderate plasma clearance (3.959 ml/min/kg), and its half-life is relatively short at 1.88 hours. In terms of toxicity, taurine exhibits a low risk for many common toxicological concerns, including hERG inhibition, genotoxicity, and liver toxicity, although there is some indication of potential skin sensitization.

Fig. 2B displays a pharmacokinetic profile for Taurine, where various properties like TPSA and LogP (Octanol/Water Partition Coefficient) are plotted to evaluate its ability to cross biological barriers. The highlighted regions indicate the prediction zones for the Blood-Brain Barrier (BBB), Human Intestinal Absorption (HIA), and PGP (P-Glycoprotein) efflux, with Taurine marked as a red dot within these areas. The plot suggests that Taurine is likely a PGP substrate, implying it may not be extensively effluxed by the P-Glycoprotein system, allowing potential absorption and distribution. The positioning within the BBB and HIA zones indicates a favorable absorption profile, with a high likelihood of crossing the blood-brain barrier and being absorbed in the intestine.

STopTox analysis

StopTox analysis (Fig. 3) for taurine across various toxicity endpoints suggests that it is generally non-toxic. Specifically, taurine was predicted to be non-toxic for acute inhalation, acute oral, and acute dermal toxicity,

with prediction confidence values of 70.0%, 83.0%, and 90.0%, respectively. Additionally, taurine was classified as a non-sensitizer for skin sensitization, with a high prediction confidence of 90.0%. However, Taurine showed a potential risk for eye irritation and corrosion, with a prediction of toxic at 61.0% confidence, as well as for skin irritation and corrosion, which was predicted as positive (irritant) with 60.0% confidence.

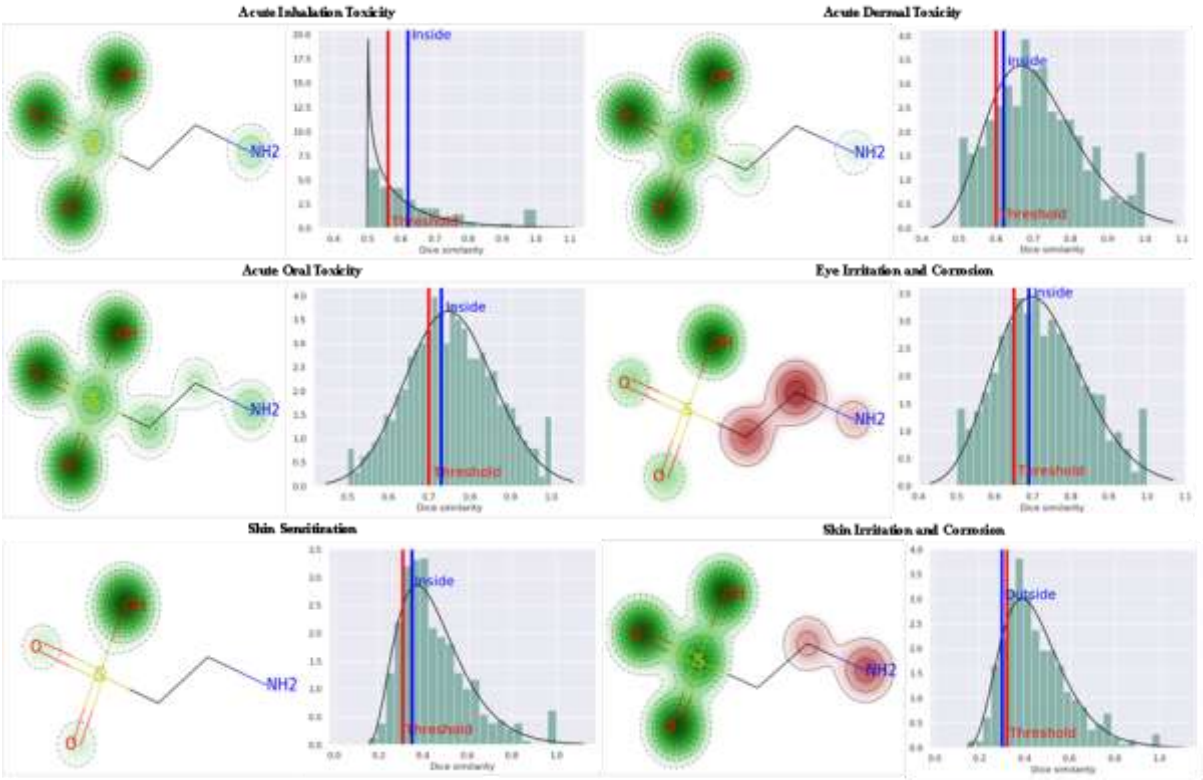

Fig. 3. Toxicity predictions for taurine based on STopTox analysis across multiple endpoints

Effect of taurine on protein denaturation

The protein denaturation assays (Fig. 4) revealed that both taurine and aspirin exhibit anti-inflammatory activity, with IC50 values of 103.59 µM for taurine and 92.61 µM for aspirin.

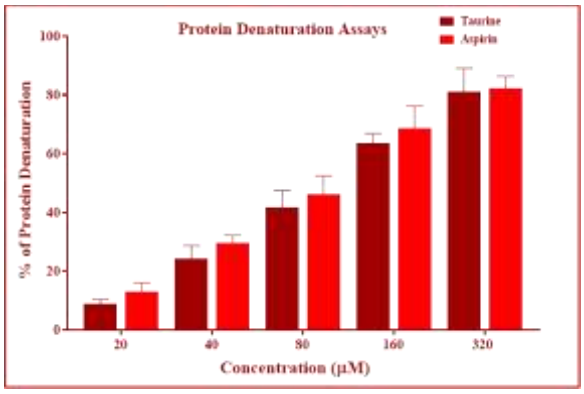

Fig. 4. Anti-inflammatory activity of taurine assessed by using the protein denaturation assay

Data represented as mean ± SD of triplicate experiments.

At a concentration of 20 µM, taurine inhibited 8.79% (± 1.64) of protein denaturation, while aspirin showed a higher inhibition of 12.94% (± 3.17). As the concentration increased to 40 µM, taurine's inhibition rose to 24.07% (± 4.61), while aspirin's inhibition increased to 29.46% (± 2.75). At 80 µM, taurine exhibited 41.64% (± 5.87) inhibition, and aspirin showed 46.01% (± 6.49). At 160 µM, taurine reached 63.58% (± 3.19) inhibition, compared to aspirin's 68.72% (± 7.49), and at 320 µM, taurine achieved 80.92% (± 8.16), while aspirin demonstrated a comparable inhibition of 82.16% (± 4.18).

Effect of taurine on ferric reducing power

In the ferric reducing power assay (Fig. 5), taurine and ascorbic acid both exhibited antioxidant activity, with taurine showing an IC50 value of 90.10 µM, slightly higher than ascorbic acid's IC50 value of 72.99 µM. At lower concentrations, taurine (20 µM) achieved a

reduction of 13.87% (± 2.15), while ascorbic acid (20 µM) showed a slightly higher reduction of 16.24% (± 1.59). As concentrations increased, taurine demonstrated a progressive increase in reduction: at 40 µM, it reached 26.92% (± 4.07); at 80 µM, it was 47.65% (± 6.84); and at 160 µM, it achieved 65.02% (± 7.48), with the highest concentration (320 µM) showing 83.18% (± 3.85). Ascorbic acid followed a similar pattern but exhibited slightly higher reductions across all concentrations, with 31.74% (± 6.41) at 40 µM, 52.19% (± 8.15) at 80 µM, and 70.84% (± 6.98) at 160 µM, reaching 92.52% (± 7.15) at 320 µM.

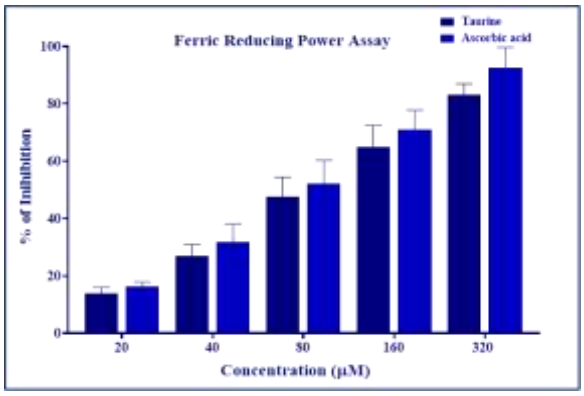

Fig. 5. Antioxidant activity of taurine assessed by using ferric reducing power assay
Data represented as mean ± standard deviation (SD) of triplicate experiments.

Effect of taurine on hydrogen peroxide scavenging

In the hydrogen peroxide scavenging assay (Fig. 6), taurine demonstrated an IC50 value of 83.71 µM, indicating moderate scavenging potential, while ascorbic acid had a slightly lower IC50 of 63.63 µM, reflecting a stronger ability to neutralize hydrogen peroxide. At 20 µM, taurine showed 19.24% (± 5.41) scavenging activity, whereas ascorbic acid scavenged 24.69% (± 4.68) of hydrogen peroxide. At 40 µM, taurine's activity increased to 33.45% (± 5.48), while ascorbic acid reached 39.37% (± 6.49). The trend continued at 80 µM, with taurine achieving 51.67% (± 7.87) and ascorbic acid 58.46% (± 5.45). At higher concentrations, taurine exhibited 63.48% (± 8.42) inhibition at 160 µM and 76.87% (± 3.14) at 320 µM. In comparison, ascorbic acid reached 67.18% (± 6.18) at 160 µM and 81.47% (± 5.06) at 320 µM.

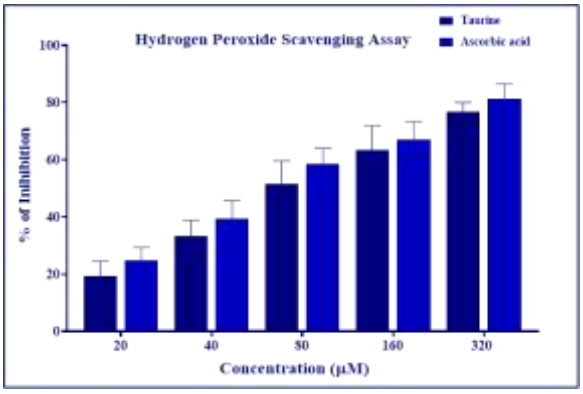

Fig. 6. Antioxidant activity of taurine assessed by using the hydrogen peroxide scavenging assay
Data represented as mean ± standard deviation (SD) of triplicate experiments.

Effect of taurine on superoxide anion scavenging

Taurine and ascorbic acid were evaluated for their ability to scavenge superoxide anions (Fig. 7), with taurine displaying an IC50 value of 90.82 µM, which was higher than ascorbic acid's IC50 of 64.14 µM, indicating a stronger scavenging effect for ascorbic acid. At 20 µM, taurine exhibited 15.78% (± 4.15) scavenging activity, while ascorbic acid showed 20.64% (± 2.16). At 40 µM, taurine's scavenging increased to 34.61% (± 2.45), whereas ascorbic acid reached 41.75% (± 3.18). At 80 µM, taurine achieved 47.08% (± 5.81), and ascorbic acid showed a higher scavenging of 55.38% (± 4.85). At 160 µM, taurine's activity reached 61.43% (± 7.45), while ascorbic acid demonstrated 70.57% (± 6.49). Finally, at 320 µM, taurine exhibited 77.92% (± 8.19), while ascorbic acid achieved 83.18% (± 5.02).

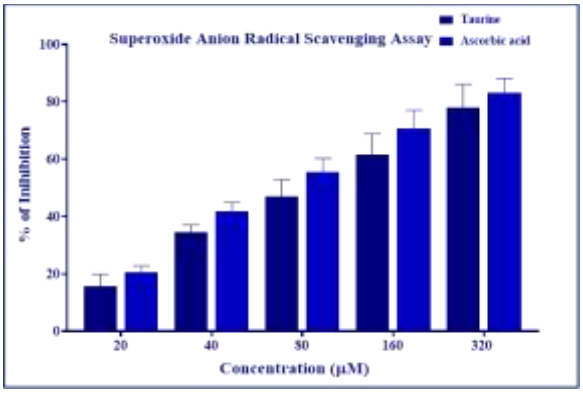

Fig. 7. Antioxidant activity of taurine assessed by using the superoxide anion scavenging assay
Data represented as mean ± standard deviation (SD) of triplicate experiments

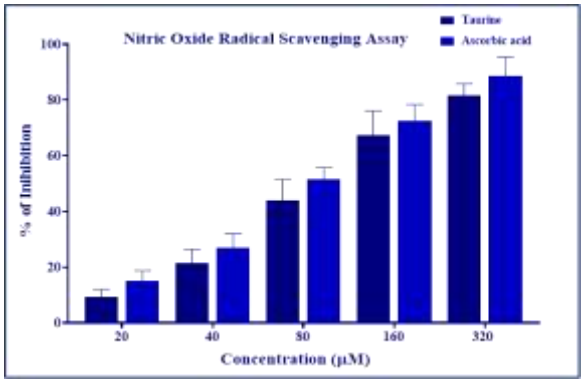

Fig. 8. Antioxidant activity of taurine assessed by using the nitric oxide radical scavenging assay
Data represented as mean ± standard deviation (SD) of triplicate experiments

Effect of taurine on nitric oxide radical scavenging

The nitric oxide radical scavenging assay (Fig. 8) revealed that both taurine and ascorbic acid possess the ability to scavenge nitric oxide radicals, with taurine demonstrating an IC50 value of 98.21 µM, while ascorbic acid exhibited a lower IC50 of 77.61 µM, indicating stronger efficacy for ascorbic acid. At a concentration of 20 µM, taurine showed a scavenging activity of 9.42% (± 2.61), compared to ascorbic acid's 14.94% (± 3.87). As the concentration increased to 40 µM, taurine's scavenging improved to 21.54% (± 4.86), while ascorbic acid reached 26.81% (± 5.16). At 80 µM, taurine demonstrated 44.06% (± 7.51) scavenging, while ascorbic acid exhibited 51.64% (± 4.16). At higher concentrations, taurine achieved 67.19% (± 8.95) at 160 µM and 81.67% (± 4.18) at 320 µM, while ascorbic acid reached 72.62% (± 5.78) at 160 µM and 88.75% (± 6.79) at 320 µM.

DISCUSSION

Taurine's pharmacokinetic and therapeutic profiles indicate it has promising potential for clinical applications. Its molecular weight of 125.01 g/mol and a negative LogP of -2.762 suggest high water solubility, although they typically indicate limited passive membrane permeability. Despite these theoretical characteristics, taurine is effectively absorbed from the gastrointestinal tract within 1.0 to 2.5 hours, reaching peak plasma concentration around 1.5 hours, as demonstrated by human studies (Bondarenko *et al.*, 2018). The analysis further predicts a high fraction unbound in plasma (Fu 89.5%) and low protein binding (9.198%), suggesting a favorable pharmacokinetic profile. However, it is important to note that physiological conditions, such as nutritional status, can alter taurine's clearance and tubular reabsorption, as observed in animal studies (Catalán-Latorre *et al.*, 2018). Additionally, the prediction that taurine is unlikely to inhibit key cytochrome P450 enzymes suggests minimal risk for metabolic drug-drug interactions, supporting its safety and stability as a supplement (Bondarenko *et al.*, 2018).

The protein denaturation assay reveals taurine's potent anti-inflammatory properties, with an IC50 of 103.59 µM, comparable to that of aspirin, a standard anti-inflammatory agent. The ability of taurine to inhibit protein denaturation (up to 80.92% at 320 µM) aligns with its known role as an organic osmolyte, stabilizing proteins under thermal stress (Mastrella *et al.*, 2022). Furthermore, taurine exhibits antioxidant activity, effectively scavenging hydrogen peroxide (IC50 = 83.71 µM) and superoxide anions (IC50 = 90.82 µM). These effects are consistent with taurine's high concentration in tissues exposed to oxidative stress, where it serves a cytoprotective function (Marcinkiewicz and Kontny, 2014). These findings reinforce taurine's multifaceted biological roles, particularly its antioxidant defense mechanisms and protein stabilization properties.

Taurine's mechanisms of action extend beyond its antioxidant capacity. It modulates the activity of endogenous antioxidant enzymes, such as superoxide dismutase and catalase, enhancing the body's defense against oxidative stress. Additionally, taurine inhibits NADPH oxidase, thereby further decreasing reactive oxygen species production (Han *et al.*, 2016). Beyond its antioxidant effects, taurine also exerts cardiovascular benefits by inhibiting the renin-angiotensin system (RAS), which plays a key role in regulating blood pressure and vascular tone (Abebe and Mozaffari, 2011). Taurine also stabilizes endothelial function by modulating signaling pathways, including the protein kinase B and extracellular-signal-regulated kinase pathways, and contributes to calcium homeostasis and

cell membrane stability, which are crucial for cellular health (Marcinkiewicz and Kontny, 2014; Abebe and Mozaffari, 2011; Baliou *et al.*, 2021).

The pharmacological profile of taurine positions it as a potential protective agent against cardiovascular diseases. Taurine's ability to promote endothelium-dependent vasorelaxation and protect endothelial cells from damage induced by oxidized low-density lipoprotein (LDL) highlights its role in vascular health (Ulrich-Merzenich *et al.*, 2007). Furthermore, taurine has demonstrated its ability to mitigate oxidative stress and improve endothelial function, particularly when combined with nutrients such as magnesium (Katakawa *et al.*, 2016). Taurine's lipid-regulating effects are also noteworthy; experimental evidence shows that it can significantly reduce total cholesterol, triglycerides, and LDL-C levels, addressing dyslipidemia, a major cardiovascular disease risk factor (Eskandrani *et al.*, 2025).

Moreover, taurine offers myocardial protection, particularly in models of heart failure and diabetes, where it helps prevent oxidative damage and mitochondrial dysfunction, both of which are critical for maintaining cardiac output and preventing tissue injury (Han *et al.*, 2016; Mastrella *et al.*, 2022). Taurine also exerts anti-inflammatory effects by inhibiting protein denaturation and modulating inflammatory markers, which could help reduce chronic inflammation, a key factor in the progression of atherosclerosis and hypertensive heart disease (Donia and Khamis, 2021; Abebe and Mozaffari, 2011). These findings collectively underscore taurine's therapeutic potential in the prevention and management of cardiovascular conditions.

CONCLUSION

Taurine demonstrates a promising pharmacokinetic and toxicological profile, coupled with potent anti-inflammatory and antioxidant activities, making it a valuable therapeutic candidate for the management of cardiovascular diseases. Its ability to modulate oxidative stress and inflammatory pathways highlights its protective role in vascular and myocardial health. The favorable pharmacokinetic properties and low toxicity risk further support taurine's potential as a safe and effective therapeutic agent. Future clinical trials will be crucial to substantiate its therapeutic applications in cardiovascular medicine.

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