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Taurine supplementation as a therapeutic strategy for cellular senescence and chronic inflammation in long COVID: a systematic review and meta-analysis

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Abstract

Background SARS-CoV-2 infection can induce cellular senescence, resulting in chronic inflammation and senescence-associated secretory phenotype observed in post-acute sequalae of COVID-19 (PASC). Taurine, a conditionally essential amino acid with potent anti-inflammatory and antioxidant properties, is naturally upregulated during COVID-19 convalescence. Preclinical evidence suggests taurine protects against cellular senescence, telomerase deficiency, DNA damage, and mitochondrial dysfunction, indicating its potential therapeutic role in PASC.

Methods We systemically searched MEDLINE, Embase, Cochrane Library, and Scopus through 21st March 2025 for clinical trials investigating taurine supplementation in systemic perturbations associated with PASC. Outcomes of interest included markers of glycemic control, lipid metabolism, inflammation, oxidative stress, cardiopulmonary function, and neurocognition. In a parallel analysis, we systematically searched six databases (MEDLINE, Embase, Cochrane Library, CINAHL, Web of Science, and Scopus) for studies reporting plasma taurine levels during COVID-19 convalescence. Random-effects models were used to pool effect sizes, and meta-regression was employed to explore study heterogeneity.

Results Analysis of 27 clinical trials ($n=1,030$) demonstrated that taurine supplementation significantly improved markers of metabolic dysfunction (including hemoglobin A1c, fasting blood glucose, fasting insulin, HOMA-IR, total cholesterol, triglycerides, and low-density lipoprotein), inflammation (C-reactive protein, TNF-α, and IL-6), and oxidative stress (malondialdehyde). Supplementation also improved blood pressure and exercise capacity, though no significant effects on neurocognition were observed. Given the dose-response relationship identified between taurine and inflammatory markers TNF-α and IL-6, a daily dose of 3,000 mg appears to offer an optimal balance between clinical efficacy and tolerability. Furthermore, a pooled analysis of six studies ($n=308$) revealed significantly lower plasma taurine levels in individuals with PASC compared to recovered, symptom-free counterparts (SMD - 0.35, 95% CI: -0.63 to -0.08).

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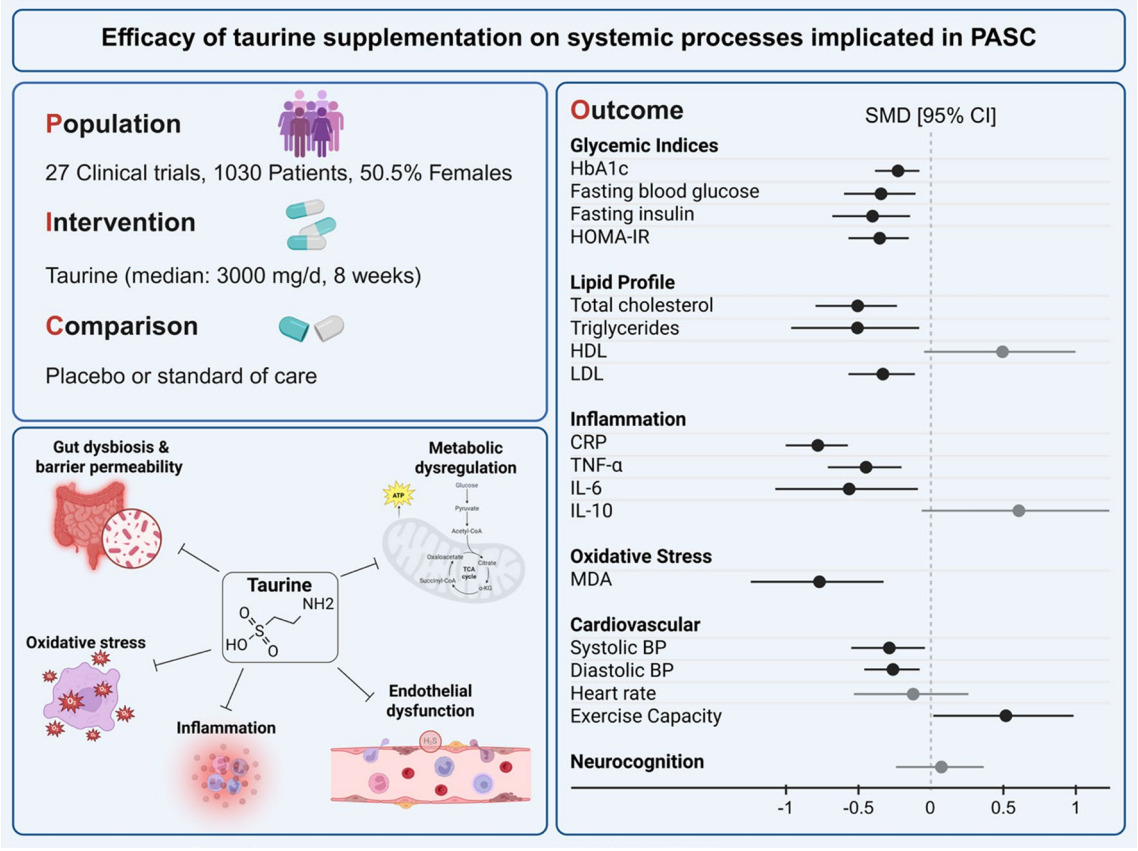
Conclusions Taurine supplementation effectively ameliorates key pathological features of PASC, including metabolic perturbation, endothelial dysfunction, and oxidative stress. The observed relative taurine deficiency in individuals with PASC further supports its potential as a therapeutic strategy to reduce senescence burden and chronic inflammation underlying this debilitating condition.

Trial registration CRD420251011508 (Registration Date: 16 March 2025).

Keywords Post-acute sequalae of COVID-19, Long COVID, Inflammation, Senescence, Taurine, Meta analysis, Metabolic syndrome, Cardiopulmonary function, Cognition

Graphical Abstract



Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) utilizes the ubiquitously expressed angiotensin-converting enzyme 2 (ACE2) as its cellular receptor, leading to multisystemic manifestations [1–3]. The resulting post-acute and long-term health consequences of SARS-CoV-2 infection are referred to as long COVID, also known as post-acute sequalae of COVID-19 (PASC). Cardinal symptoms include brain fog, fatigue, post-exertional malaise, sleep disturbances, and shortness of breath amongst many others [4, 5]. Epidemiological studies suggest that 6% to 30% of COVID-19 survivors develop long COVID, translating to at least 65 million affected individuals worldwide [4, 6]. Higher risk is associated with female sex, advanced age, smoking, elevated body mass index, multimorbidity, and greater acute disease severity [7, 8].

Although the overall risk of PASC complications has declined over time, many individuals continue to grapple with persistent neurological, pulmonary, cardiovascular, metabolic, and gastrointestinal disorders years beyond their initial infection [9, 10]. These lingering conditions contribute to substantial disability-adjusted life-years and poor clinical outcomes. Mechanistically, PASC is linked to accelerated biological aging, characterized by chronic inflammation, gut dysbiosis, cellular senescence, epigenetic alterations, mitochondrial dysfunction, oxidative stress, and telomere attrition [11–13]. Strikingly, these processes mirror the hallmarks of inflammaging, a key driver of metabolic, neurocognitive, and cardiovascular

diseases [14, 15]. However, despite growing insights into its mechanisms, effective treatments for long COVID remain elusive.

Taurine (2-aminoethanesulfonic acid) is a sulfonate-containing amino acid abundantly distributed across tissues such as the brain, heart, and skeletal muscles [16]. As a conditionally essential micronutrient, it has been safely incorporated into various nutraceutical drinks and infant formulas. Taurine plays a critical role in cellular energetics by stabilizing mitochondrial function, enhancing antioxidant activity, and inhibiting apoptosis [17]. These properties underlie its broad therapeutic potential against cardiovascular, metabolic, and neurodegenerative disorders. Notably, taurine deficiency has been implicated in the aging process and cardiovascular disease [18, 19], whereas taurine supplementation has shown promising effects in preclinical models to reduce depression-like behaviours, inflammation, DNA damage, and telomerase deficiency, while enhancing bioenergetics and countering cellular senescence [20, 21].

Relevant to PASC, metabolomic analyses of plasma samples from individuals convalescing from COVID-19 have identified taurine as one of the top upregulated metabolites six months post-infection [22–24]. Importantly, higher taurine levels correlate negatively with the incidence of long COVID symptoms, including cognitive impairment, mood disturbances, dyspnea, and general malaise [22, 25]. During the convalescence phase, patients exhibiting a significant rise in plasma taurine also experienced fewer adverse clinical outcomes [24].

Given taurine's beneficial effects on pathways implicated in cellular senescence and its natural upregulation during COVID-19 convalescence, we hypothesize that taurine supplementation could alleviate symptom burden and improve outcomes in individuals affected by PASC. To date, however, no study has directly investigated the effects of taurine supplementation in this population. Therefore, this study aims to (1) investigate the efficacy of taurine supplementation on conditions sharing core pathological features with PASC across metabolic, cardiovascular, gastrointestinal, and neurocognitive domains; (2) evaluate the safety of taurine supplementation and sources of heterogeneity in trial results; and (3) analyzed differences in plasma taurine levels during convalescence between recovered, symptom-free individuals and those experiencing persistent PASC.

Methods

Protocol and registration

This systematic review and meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines. The protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO, CRD420251011508) on March 16, 2025 [26]. The completed PRISMA checklist is available in Supplemental Table 1.

Search strategy

In collaboration with the research team, the medical librarian (J.Y.K) developed and executed comprehensive searches in Ovid MEDLINE, Ovid Embase, Cochrane Library (via Wiley), and Scopus on March 21, 2025. To capture all relevant literature on taurine supplementation for systemic perturbations associated with PASC, relevant keywords and controlled vocabulary were carefully selected. There were no language or date limits applied and the searches were restricted to clinical trials by using a broad clinical trials search filter developed by Canada's Drug Agency [27]. The comprehensive search strategies are detailed in Supplemental Table 2. For studies investigating the clinical efficacy of taurine supplementation, a total of 3,151 results were retrieved, and they were exported to EndNote for an initial duplicate removal process before being imported into the Covidence (www.covidence.org) web-based tool for screening. This yielded 2,010 unique records for title and abstract screening.

A separate, targeted literature search was conducted on April 8, 2025, to identify studies reporting plasma taurine levels during convalescence from acute SARS-CoV-2 infection. This search encompassed Ovid MEDLINE, Ovid Embase, Cochrane Library (via Wiley), CINAHL, Web of Science Core Collection, and Scopus (Supplemental Table 3). From 42 retrieved records, 23 unique references remained after duplicate removal and were advanced to title and abstract screening.

Study selection process

Randomized and non-randomized clinical trials evaluating oral taurine supplementation in adults 18 years of age or older were eligible for inclusion. We included studies investigating outcomes across neurocognitive, metabolic, cardiovascular, and gastrointestinal domains, as defined by epidemiological studies of PASC [6, 28, 29]. Given no clinical trials to date have directly examined taurine supplementation in patients diagnosed with PASC, we elected to include trials conducted in populations with conditions sharing core pathological features with PASC, such as chronic inflammation, immune dysregulation, and endothelial dysfunction (Table 1). Studies were excluded if taurine was co-administered with other nutrients, delivered via non-enteric routes, or evaluated solely for performance enhancement in athletes or healthy young individuals. Studies were also excluded from the meta-analysis if essential data could not be extracted from publications and not provided by corresponding authors following multiple requests.

Table 1 Characteristics of included studies for oral taurine supplementation in systemic processes implicated in post-acute sequalae of COVID-19

Study	Sample size	Sex (% female)	Mean age (years)	Study population	Daily taurine dose	Treatment duration (weeks)	Comparator
Rosa et al. 2014	16	100	32.5	Obese participants	3000 mg	8	Placebo (Starch)
Abud et al. 2022	24	100	61.4	Post-menopausal women	1500 mg	16	Placebo (Starch)
Haidari et al. 2020	38	100	35.9	Obese participants	3000 mg	8	Placebo (Identical capsules)
Chauncey et al. 2003	32	N/A	N/A	T2DM	3000 mg	16	Placebo (Identical capsules)
Moludi et al. 2022	120	20	52.6	T2DM	3000 mg	8	Placebo (Starch)
Masouleh et al. 2021	20	100	53.0	T2DM	2500 mg	8	Control
Brøns et al. 2004	18	0	40.0	Overweight participants with predisposition for T2DM	1500 mg	8	Placebo (Cellulose)
Zhang et al. 2004	30	53	20.3	Obese participants	3000 mg	7	Placebo (Starch)
Moloney et al. 2010	18	0	28.0	T1DM	1500 mg	2	Placebo (Identical capsules)
Esmaeili et al. 2020	41	70	43.1	T2DM	3000 mg	8	Placebo (Microcrystalline cellulose)
Maleki et al. 2020	45	72	42.8	T2DM	3000 mg	8	Placebo (Microcrystalline cellulose)
Buonani et al. 2019	21	100	60.5	Post-menopausal women	1500 mg	8	Control
Shari et al. 2019	80	45	49.5	T2DM	1000 mg	12	Placebo (Starch)
Shari et al. 2020	50	60	37.9	Obese participants	3000 mg	8	Placebo (Starch)
Chupel et al. 2018	24	100	83.6	Elderly participants in nursing home	1500 mg	14	Control
Bae et al. 2019	31	100	80.7	Dementia	3000 mg	4	Control
Vahdat et al. 2021	32	23	32.9	Total brain injury	30 mg/kg	2	Control
O' Donnell et al. 2016	86	30	21.4	Psychosis	4000 mg	12	Placebo (Identical capsules)
Hsieh et al. 2014	30	43	59.5	Chronic alcohol use disorder	6000 mg	12	Placebo (Unspecified)
Schwarzer et al. 2018	22	36	52.0	Liver cirrhosis	6000 mg	4	Placebo (Identical capsules)
Hu et al. 2008	24	46	58.0	Chronic hepatitis C	6000 mg	12	Placebo (Unspecified)
Ahmadian et al. 2017	16	N/A	60.1	Heart failure	1500 mg	2	Placebo (Starch)
Beyranvand et al. 2011	29	10	60.6	Heart failure	1500 mg	2	Placebo (Starch)
Azuma et al. 1985	14	36	66.6	Heart failure	6000 mg	4	Placebo (Unspecified)
Sun et al. 2016	120	58	56.8	Prehypertensive participants	1600 mg	12	Placebo (Identical capsules)
Fujita et al. 1987	19	0	22.0	Prehypertensive participants	6000 mg	1	Placebo (Unspecified)
Elmokadem et al. 2015	30	43	46.0	Sepsis	30 mg/kg	2	Control

Study selection was performed independently and in duplicate by two reviewers (K.W. and C.M.) using the Covidence systematic review software (Veritas Health Innovations, Melbourne, VIC, Australia). Titles and abstracts were screened for predefined criteria relating to study design, demographics, analytical method, and intervention. Any study that was identified as potentially eligible at this first stage was advanced to full-text review for assessment of eligibility. Any disagreements between reviewers were resolved through consultation with a third reviewer (G.Y.O.) when present.

Data extraction and quality assessment

Two reviewers (K.W. and C.M.) independently extracted the following variables: author name, year of publication, study design, geographic location, participant demographics, sample size, intervention details, inclusion and exclusion criteria, adverse events associated with taurine supplementation, and outcomes of interest before and after intervention. For studies on convalescent taurine levels, we additionally extracted the analytical method, duration since SAR-CoV-2 infection, and mean ± standard deviation of taurine levels in recovered and PASC cohorts. If the outcomes of interest were only presented graphically, we extracted the data using the software WebPlotDigitizer 5.2.

The risk of bias in clinical trials was assessed independently by two reviewers using the revised Cochrane Collaboration risk of bias tool (RoB 2) [30]. This tool evaluates five potential domains of bias, including the randomization process, deviations from intended interventions, missing outcome data, measurement of outcome, and selection of the reported result. Each domain is assessed using signalling questions, leading to an overall judgement of low, some concerns or high risk of bias. Disagreements between reviewers were resolved through discussion until a consensus was reached.

Data analysis

All analyses were conducted using the R package *metafor* (R version 4.5.0, Vienna, Austria, *metafor* version 4.8.0) with random effects restricted maximum likelihood method to pool effect sizes for all outcomes of interest. For meta-analysis of continuous variables, standardized mean differences (SMD) with corresponding 95% confidence intervals were calculated. Data reported as mean ± standard error or median with interquartile range were converted to mean ± standard deviation using established methods outlined by the Cochrane Collaboration [31].

Heterogeneity was quantified using the I^2 statistic, tau-squared, and chi-square test. An I^2 value greater than 50% was considered to indicate substantial heterogeneity among the included studies. The influence of individual studies on the model was assessed using DFBETA and Cook's distance. A one-study-removal sensitivity analysis was performed for any studies deemed influential to assess the impact on the overall effect size. Meta-regression was conducted to investigate the effects of variables, including total daily taurine dose, age, sex, and treatment or follow-up duration on outcomes of interest when appropriate. To assess for small study effects, the rank correlation and Egger's regression test using standard error of the observed outcomes as predictor were used to check for funnel plot asymmetry. Statistical significance was considered based on two-tailed $p < 0.05$.

Results

Study selection and characteristics

For clinical trials involving oral taurine supplementation, the initial database search identified 3,151 potentially eligible records (Fig. 1). After removing duplicates, we screened 2,010 records, ultimately including 27 trials involving 1,030 participants in the meta-analysis (Table 1). Sample size ranged from 14 to 120 participants, with a median of 30 participants per study. Females comprised 50.5% of the overall participants, and seven studies enrolled exclusively female participants [32–38]. The median taurine dosage was 3,000 mg/day (ranging from 1,000 mg/day to 6,000 mg/day) administered over a median of eight weeks (ranging from 1 week to 16 weeks). Taurine appeared to be generally well-tolerated, with no major adverse events reported except for mild gastrointestinal discomfort, increased fatigue, and flatulence at 6,000 mg/day as noted in one study (Supplemental Table 4) [39]. Seventeen studies used a placebo for comparison, while six studies employed a non-intervention or standard of care control group. Of the 27 studies, 15 investigated taurine supplementations in metabolic disorders, including obesity, diabetes, and post-menopausal state (Table 1). Four examined neurocognitive effects in nursing home residents, patients with dementia, or psychosis, and three focused on gastrointestinal disorders, such as cirrhosis, chronic alcohol use or hepatitis. Finally, five studies assessed taurine's effects on cardiovascular disease, with two studies in prehypertensive individuals and three in heart failure patients.

For the analysis of taurine levels during COVID-19 convalescence, our database search identified 42 potentially eligible records (Supplemental Fig. 1). After screening, six studies comprising 308 participants (104 recovered and 204 with persistent PASC) were included (Supplemental Table 5). The average participant age ranged from 32 to 61 years, with 51% female representation. Follow-up duration varied from 3 to 24 months, and all studies used liquid chromatography-mass spectrometry for taurine measurement with COVID-19 diagnosis

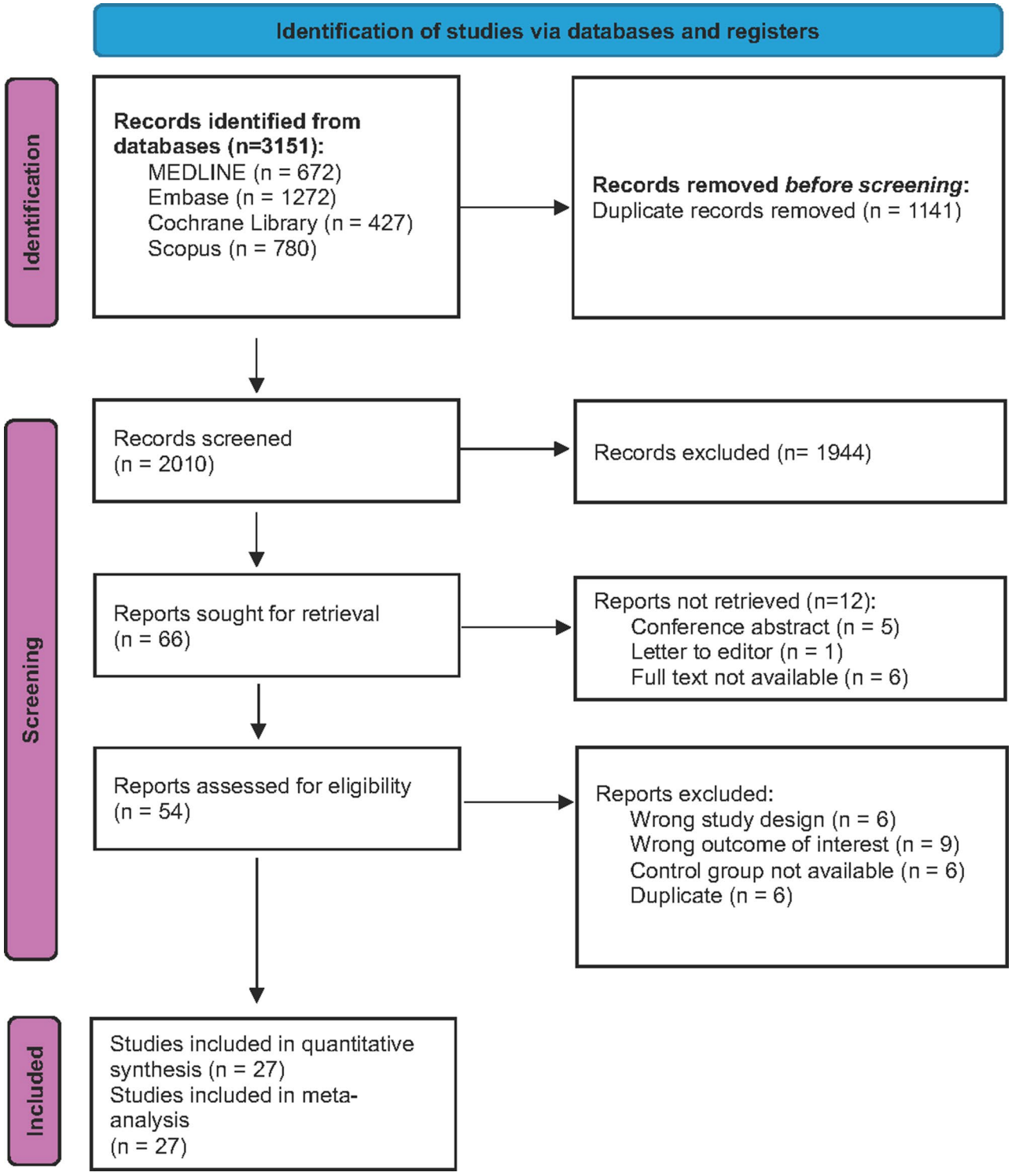


Fig. 1 Preferred reporting items for systematic reviews and meta-analyses (PRISMA) flowchart for oral taurine supplementation in systemic processes implicated in PASC

confirmed based on positive SARS-CoV-2 real time PCR assay.

Risk of bias assessment

Risk of bias assessments for the included trials are summarized in Fig. 2 and Supplemental Fig. 2. Of the 27 studies, 12 studies (44.4%) had a low risk of bias [37, 39–49], 11 studies (40.7%) raised some concerns [32, 35, 36, 38, 50–56], and 4 studies (14.8%) were at high risk [33, 34, 57, 58]. Among the high-risk studies, two used non-randomized group allocation [33, 34], one study had concerns with blinding of study personnel [57], and the fourth study had significant missing outcome data due to participant noncompliance or medication changes [58].

Effects of taurine supplementation on metabolic parameters

Taurine supplementation was associated with improved glycemic indices, evidenced by reduced glycated hemoglobin (HbA1c, 10 studies: SMD -0.23, 95% CI: -0.42 to -0.03), fasting blood glucose (FBG, 8 studies: SMD -0.38, 95% CI: -0.59 to -0.17), fasting insulin (7 studies: SMD -0.40, 95% CI: -0.63 to -0.16), and homeostatic model assessment for insulin resistance (HOMA-IR, 7 studies: SMD -0.36, 95% CI: -0.59 to -0.13, Fig. 3A).

Additionally, taurine supplementation also promoted better lipid profiles, reducing total cholesterol (12 studies: SMD -0.50, 95% CI: -0.85 to -0.15), triglycerides (12 studies: SMD -0.50, 95% CI: -0.98 to -0.03), and low-density lipoprotein (LDL, 8 studies: SMD -0.33, 95% CI: -0.55 to -0.11, Fig. 3B). However, supplementation did not demonstrate a significant effect on high-density lipoprotein (HDL) compared to controls (10 studies: SMD 0.49, 95% CI: -0.09 to 1.08).

There was significant heterogeneity observed between studies for total cholesterol (I^2 = 66.75, p = 0.001), triglycerides (I^2 = 82.01, $p < 0.001$), and HDL (I^2 = 86.50, $p < 0.001$). According to Cook's distance, one study was deemed as overly influential [51]. A sensitivity analysis excluding this individual study preserved the observed effects while substantially reducing heterogeneity (Supplemental Fig. 3). Funnel plots showed no evidence of asymmetry (Supplemental Fig. 4), and meta-regression revealed no significant association between outcomes and age, sex, daily taurine dose or treatment duration.

Effects of taurine supplementation on inflammation and oxidative stress

Taurine supplementation significantly reduced markers of inflammation, including C-reactive protein (CRP, 7 studies: SMD -0.77, 95% CI: -1.01 to -0.54) and TNF-α (6 studies: SMD -0.42, 95% CI: -0.65 to -0.19, Fig. 4A). Moreover, there was a trend towards reduction in IL-6 levels (6 studies: SMD -0.54, 95% CI: -1.07 to -0.01) in the pooled analysis with a wide confidence interval. Similarly, taurine supplementation resulted in a uniform direction towards increase in IL-10 levels (3 studies: SMD 0.62, 95% CI: -0.11 to 1.34), but the overall pooled effect did not reach statistical significance due to the limited sample size.

The effects of taurine supplementation on oxidative stress have been assessed through various markers including advanced oxidation protein products, ferric-reducing antioxidant power, lipid peroxidation product (malondialdehyde, MDA), glutathione, superoxide dismutase, pentosidine, and methylglyoxal [36, 38, 47, 55, 59]. Of these markers assessed, only MDA was consistently reported across 6 trials and was reduced by taurine supplementation (SMD -0.71, 95% CI: -1.17 to -0.24, Fig. 4B).

Meta-regression identified a dose-response relationship with reduction in TNF-α (β -0.006, p = 0.03) and IL-6 (β -0.0008, p = 0.02). Despite the heterogeneity observed for IL-6, IL-10, and MDA, none of the studies were deemed overly influential. Additionally, we found no asymmetry through the rank correlation and Egger's regression test (Supplemental Fig. 5).

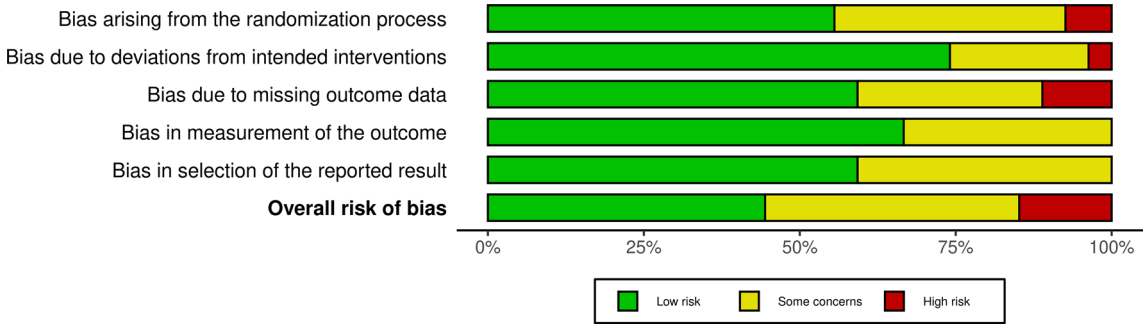


Fig. 2 Summary of risk of bias assessment using the Cochrane Risk of Bias 2 tool

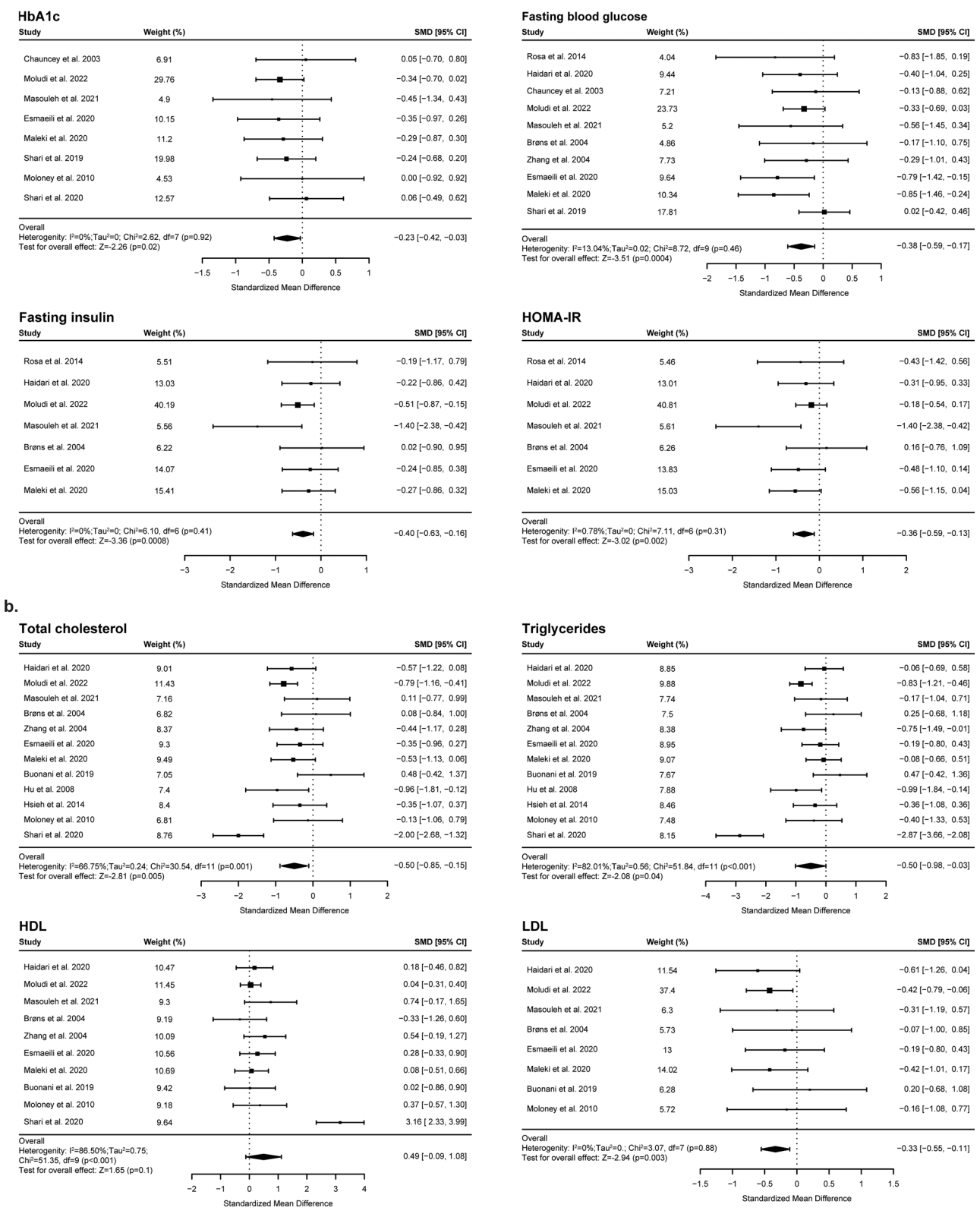


Fig. 3 Forest plot of the overall effects of taurine supplementation on metabolic parameters

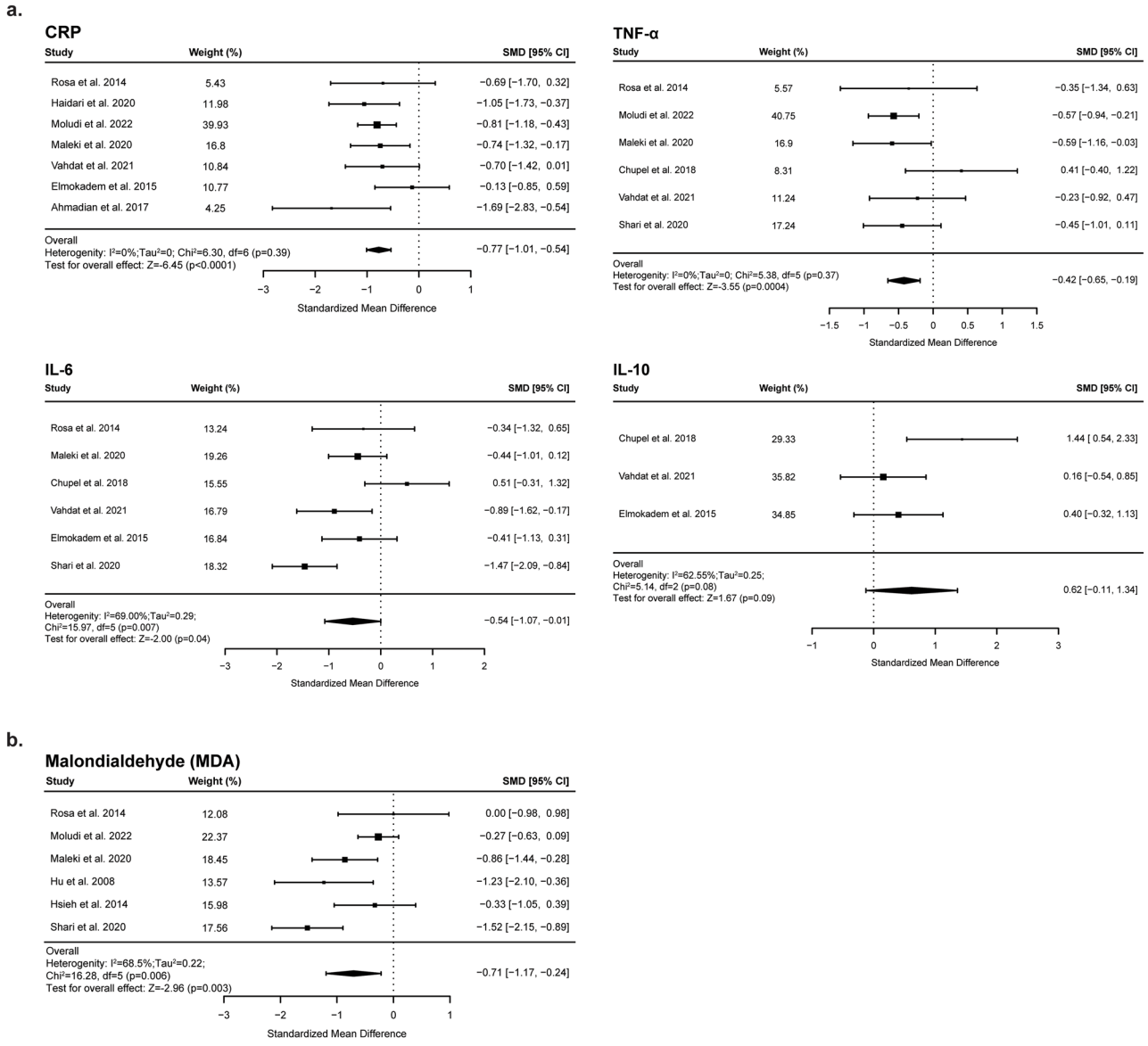


Fig. 4 Forest plot of the overall effects of taurine supplementation on inflammatory and oxidative stress markers

Effects of taurine supplementation on cardiopulmonary status

There were 9 studies that examined the effect of taurine supplementation on blood pressure (Fig. 5). In pooled analyses, taurine supplementation was associated with reduction in systolic blood pressure (SMD -0.30, 95% CI: -0.56 to -0.05) and diastolic blood pressure (SMD -0.29, 95% CI: -0.49 to -0.10). However, no significant effect on heart rate was observed (SMD -0.15, 95% CI: -0.59 to 0.30). Since shortness of breath, post-exertional malaise, and fatigue are frequently reported as symptoms of PASC, we sought to analyze taurine supplementation on exercise capacity. Two studies reported metabolic equivalents (MET) pre- and post-supplementation, while one study employed the 2-minute step-test to assess aerobic endurance [57, 60, 61]. A suggestive association was observed between taurine supplementation and an increase in exercise capacity based on the pooled analysis (SMD 0.50, 95% CI: 0.02 to 0.98). No significant asymmetry was present on the funnel plots (Supplemental Fig. 6).

Effects of taurine supplementation on neurocognitive function

Three of the four studies that examined taurine supplementation effects on neurocognitive function were in institutionalized settings, comprised entirely of elderly female participants and were deemed to be at a high risk of bias [33, 34, 61]. These studies utilized either the Montreal Cognitive Assessment or Mini-Mental State Examination to assess for domains of cognitive function.

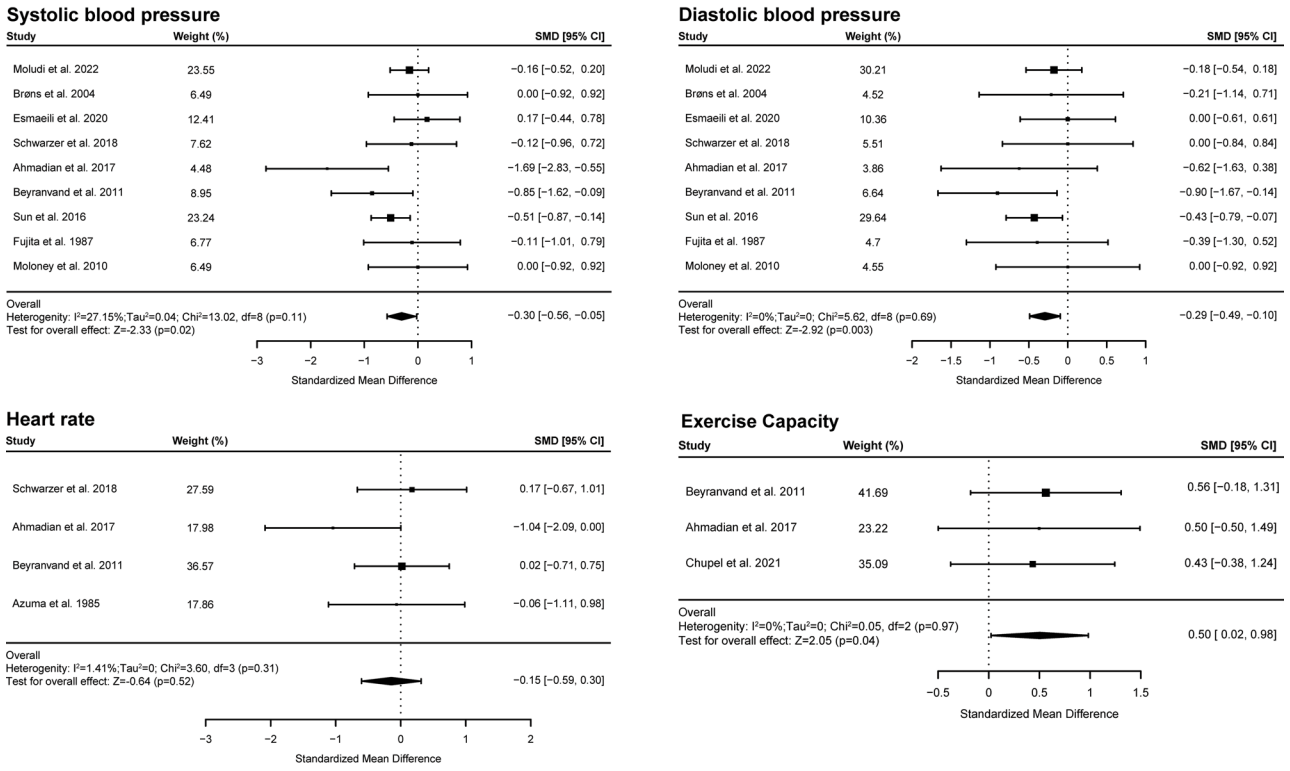


Fig. 5 Forest plot of the overall effects of taurine supplementation on cardiopulmonary status

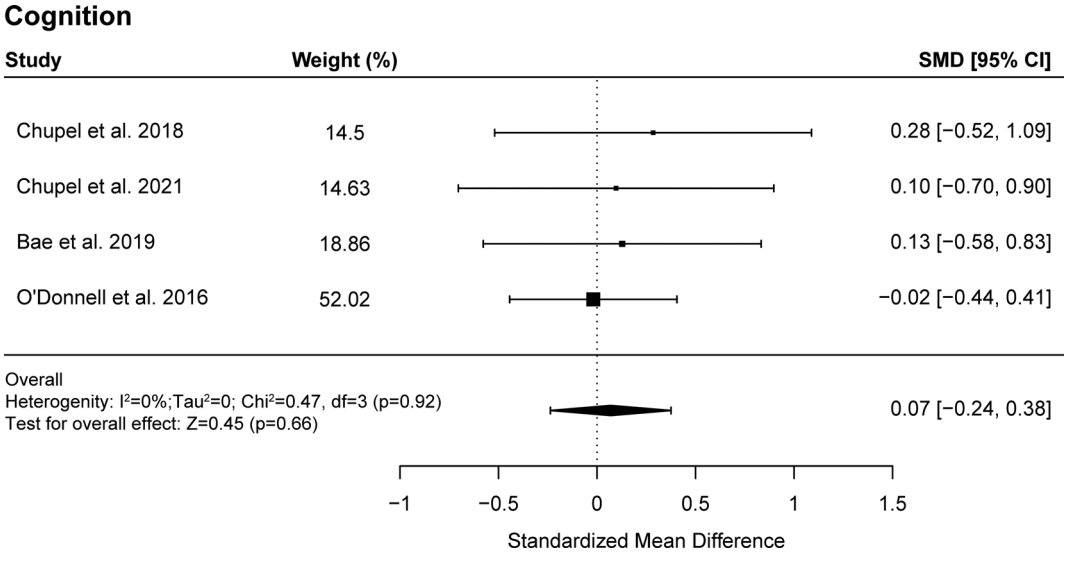


Fig. 6 Forest plot of the overall effects of taurine supplementation on neurocognitive function

Another study was in the setting of first-episode psychosis among young adults aged 18–25 years with cognition assessed using the MATRICS Consensus Cognitive Battery [44]. Individually, neither of these studies demonstrated an improvement in cognitive function from taurine supplementation, which was further reflected in the pooled estimate (SMD 0.07, 95% CI: -0.24 to 0.38, Fig. 6 and Supplemental Fig. 7).

Plasma taurine levels in COVID-19 convalescence

We next sought to evaluate the link between plasma taurine levels and PASC symptoms. A total of six studies were initially examined for plasma taurine levels during convalescence, comparing patients who reported complete symptom resolution with those experiencing persistent PASC symptoms (SMD -0.20, 95% CI: -0.60 to 0.20, Supplemental Fig. 8) [23, 24, 62–65]. However, we

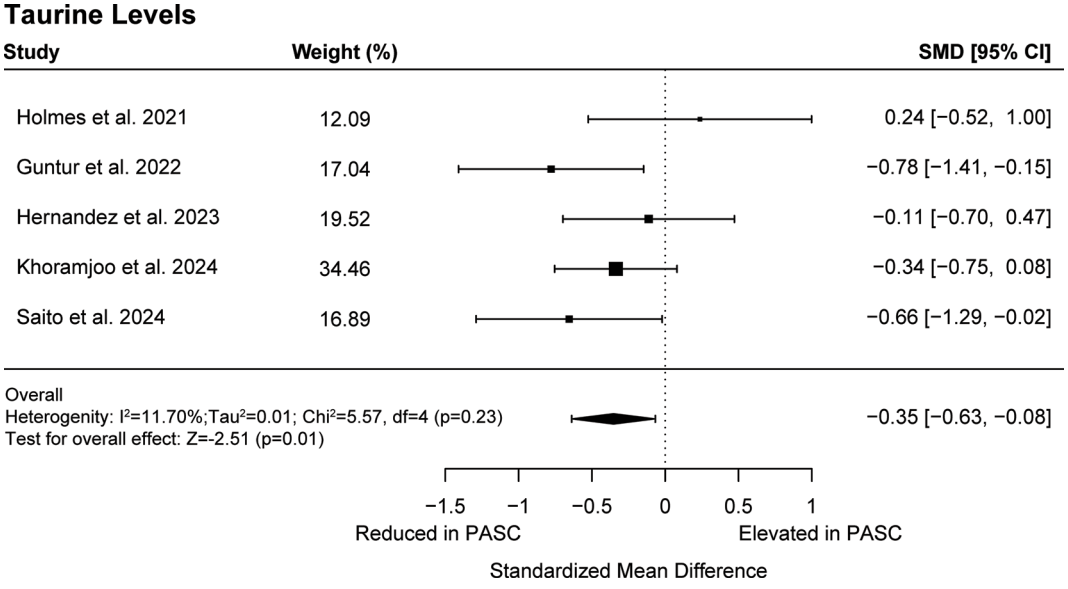


Fig. 7 Forest plot of plasma taurine levels in individuals with persistent post-acute sequalae of COVID-19 compared to recovered individuals during COVID-19 convalescence

noted substantial heterogeneity (I^2 = 61.68%, tau^2=0.15, Chi^2 = 11.99) and one study was identified to have a significant influence on the pooled effects (Cook's d = 0.55, ΔSMD = 1.15) [23]. A notable difference was found with the mean age of the study population being 32 years [23], whereas the mean age ranged between 50 and 60 years for other studies [24, 62–65]. Although data from three independent longitudinal and cross-sectional cohorts suggest that taurine levels does not decline with age in healthy individuals, the relationship between taurine and functional measures of health status showed substantial age-related differences [66]. As such, the physiological response of taurine metabolism to stress from SARS-CoV-2 infection may be influenced by the aging process. Exclusion of this study in sensitivity analyses revealed a substantial reduction in the pooled SMD, suggesting its findings disproportionately contributed to the overall effect. When the remaining studies were examined, we observed reduced plasma taurine levels in those with PASC compared to recovered individuals (SMD -0.35, 95% CI -0.63 to -0.08, I^2 = 11.70, tau^2 = 0.01, Chi^2 = 5.57, Fig. 7). Meta-regression analyses did not demonstrate substantial variability based on age, sex or follow-up duration in taurine levels from the included studies.

Discussion

SARS-CoV-2 infection triggers virus-induced cellular senescence in human epithelial and immune cells, characterized by cell-cycle arrest, impaired mitochondrial energetics, and a pro-inflammatory senescence-associated secretory phenotype that persists long after viral clearance [13, 67–69]. This lingering senescent state fuels systemic inflammation, promoting tissue fibrosis and microthrombosis, which are processes that pathologically mirror aging-related decline and correlate strongly with the debilitating symptoms observed in long COVID [68, 69]. Supporting this connection, epigenetic studies reveal striking evidence of accelerated biological aging in patients with COVID-19, demonstrated by DNA methylation age acceleration and telomere attrition, particularly following severe infection [70]. These findings position both acute and long COVID as conditions of accelerated aging, highlighting aging-related pathways as prime therapeutic targets [12]. In this context, our meta-analysis identifies taurine supplementation as a promising intervention that effectively mitigates key features of cellular senescence and inflammaging, including metabolic dysfunction, chronic inflammation, and oxidative stress while enhancing cardiopulmonary function in PASC-related disease conditions, warranting its investigation as a targeted therapy for PASC.

The relative taurine deficiency we observed in individuals with PASC compared to their recovered counterparts is deeply rooted in the pathophysiology of long COVID and the complex, bidirectional relationship between taurine and chronic inflammation [71]. During an inflammatory response, taurine is redistributed and accumulates at sites of inflammation, where it reacts with hypochlorous acid produced by neutrophils to form taurine chloramine [72]. Release of taurine chloramine from neutrophil apoptosis suppresses the overproduction of proinflammatory cytokines, including IL-1β, TNF-α, IL-6, and IL-8 [73]. Furthermore, it promotes nuclear translocation of the redox-sensitive transcription factor Nrf2, stimulating the production of antioxidant enzymes like heme oxygenase-1, glutathione peroxidase, peroxiredoxin, and

thioredoxin [73]. However, persistent inflammation and oxidative stress, as seen in conditions such as inflammatory bowel disease, diabetes or obesity, chronically increase taurine consumption, leading to systemic depletion [20, 74]. Similarly, the lingering low-grade inflammation of long COVID creates a sustained demand for taurine, depleting its reserves [75]. This pathophysiological process underscores the potential of taurine supplementation, which at 3,000 mg/day reduces inflammatory (CRP, TNFα) and oxidative stress (MDA) markers, replenishing these depleted reserves and halting the cycle of inflammation [75, 76].

Gut dysbiosis represents another critical link between PASC and taurine deficiency. SARS-CoV-2 infection induces tissue inflammation and cell death, contributing to microbiome alterations, intestinal mucosal barrier breakdown, and increased tight junction permeability, changes that persist into the convalescent phase [77]. Taurine homeostasis relies on the enterohepatic circulation, where it conjugates with bile acids in the liver. Under physiologic conditions, approximately 95% of these conjugated bile acids are reabsorbed in the terminal ileum and recycled [78]. However, infection reshapes the microbiota, increasing the abundance of taurine-utilizing bacteria taxa, which has been proposed to serve as an adaptive host response to enhance colonization resistance, as microbial taurine metabolism releases hydrogen sulfide that suppresses pathogen respiration [79, 80]. Despite these potential benefits, the increased microbial consumption of taurine, coupled with reduced intestinal uptake due to barrier disruption, contributes to systemic taurine deficiency. Although clinical evidence is still emerging, experimental models show that oral taurine supplementation can restore intestinal barrier integrity, increase tight junction proteins, and suppress aging-related intestinal inflammation [81, 82].

Metabolic dysregulation, characterized by hyperglycemia, insulin resistance, and beta-cell dysfunction, is an established consequence of COVID-19 that persists well beyond the acute infection. This impairment in glucose and lipid metabolism translates to an increased long-term risk of incident diabetes and dyslipidemia [83, 84]. The clinical significance of addressing this metabolic underpinning is highlighted by the COVID-OUT trial, where early metformin treatment (ivermectin and fluvoxamine were non-efficacious) reduced the incidence of long COVID diagnoses over a 10-month follow-up period [85]. Our results align with this strategy, demonstrating that taurine supplementation may alleviate PASC-related metabolic syndrome through improvement in glycemic indices, including HbA1c, FBG, fasting insulin, HOMA-IR and lipid profiles (total cholesterol, triglycerides, and LDL levels). The mechanisms behind these cholesterol-lowering and anti-hyperglycemic properties of taurine are well-documented in prior reviews [86, 87]. Importantly, taurine's benefits extend beyond metabolic regulation as it has been shown to enhance health span and reduce inflammaging through modulation of NLRP3 inflammasome activation, a canonical pathway driving the chronic low-grade inflammation implicated in both type 2 diabetes mellitus and accelerated aging [88–90].

Neuroinflammation is a central tenet of aging-related neurodegenerative disorders and has also been linked to brain fog and cognitive impairment observed in patients with long COVID [91]. Despite the plethora of evidence from experimental models showing protective effects of taurine supplementation in conditions like Alzheimer's disease [92], Parkinson's disease [93], depression [94], and post-hemorrhagic neuroinflammation [95], its clinical efficacy remains to be established. The neutral finding on cognitive function in our meta-analysis is corroborated by other studies showing no additional cognitive benefits of taurine supplementation beyond standard therapies such as donepezil [96]. Nevertheless, it is worth noting that the clinical evidence from human studies is very limited and derived largely from high risk-of-bias studies in elderly, institutionalized women with known cognitive impairment using brief cognitive screening tools (MoCA and MMSE) [33, 34, 61]. Given taurine's proposed mechanism to mitigate neuronal cell death through limiting glutamate-mediated excitotoxicity, reducing the aggregation of misfolded proteins, and inhibiting pro-neuronal apoptotic pathways, future trials should aim to investigate its efficacy in earlier stages of cognitive decline, such as in those associated with PASC [97, 98].

ACE2 is highly expressed in the endothelial cells and pericytes, contributing to the increased systemic thrombosis risk observed during acute SARS-CoV-2 infection [2]. However, in long COVID, the microvascular system is characterized by a persistent hypercoagulable state driven by endothelial dysfunction [99]. There is strong evidence to suggest that taurine may counteract this dysfunction through increasing plasma hydrogen sulfide and upregulating its synthesis, which inhibits calcium influx into smooth muscle cells and stimulates endothelial nitric oxide synthase to promote both endothelial and non-endothelial vasodilation [40, 100]. Indeed, our study recapitulates taurine's blood pressure-lowering effects and suggests it may improve cardiopulmonary function, corroborating previous meta-analyses demonstrating improved cardiac function in patients with heart failure [101, 102].

Although the current meta-analysis supports the cardiometabolic and anti-inflammatory benefits of taurine supplementation, whether these effects are mediated through taurine's modulation of the senescent phenotype remains to be established in clinical studies. Network analysis has shown that taurine-related genes most

frequently interact with senescence-associated secretory phenotype (SASP) genes such as p16 and p21 [20]. Supporting this, in vitro experiments demonstrated that taurine treatment significantly reduced SA-β-gal activity in irradiated osteoblasts [20]. Mechanistically, taurine interacts directly with CDKN2AIP, a positive regulator of the p53 signalling pathway that promotes p53 degradation, thereby suppressing the p53-p21 and p16INK4a signalling axes [103, 104]. Moreover, the SASP comprises a heterogenous secretome of pro-inflammatory cytokines, chemokines, growth factors, and matrix-remodeling proteases, driven primarily by chronic NF-κB activation. By inhibiting the NF-κB signaling cascade, taurine reduces inflammation, oxidative stress, mitochondrial dysfunction, and apoptosis, underscoring its broad cytoprotective role against cellular senescence that warrants further clinical exploration [81, 105, 106].

We recognize several limitations of our study. First and foremost, the clinical evidence synthesized in this meta-analysis is not derived from a PASC patient population and our findings therefore require formal validation in dedicated clinical trials within this population. Second, we pooled data from a heterogenous patient population with various comorbidities including obesity, diabetes, liver disease, hypertension, and heart failure. The response to taurine supplementation is likely dependent on baseline health status, and the substantial statistical heterogeneity in some analyses suggests that treatment effects may vary according to specific study-level characteristics. Furthermore, heterogeneity in placebo composition across the included trials represents an additional methodological consideration. Third, most of the included studies had relatively small sample sizes (ranging from 14 to 120 participants), which limits the robustness of our summary estimates. We hope that our findings will encourage larger, more robust prospective studies to better evaluate taurine's efficacy in conditions associated with PASC and inflammaging. Fourth, six of the 27 included studies were non-randomized clinical trials, with two in patients with metabolic syndrome, two in elderly women with cognitive impairment, one in patients with brain injury, and one in the setting of sepsis [32–35, 43, 50]. The inclusion of non-randomized trials may introduce bias due to residual confounding. However, the extend of this potential bias on treatment effects is difficult to ascertain given the diversity of patient populations and outcome measures across these studies. Finally, we excluded studies that combined taurine supplementation with other nutrients. Consequently, we are unable to infer potential synergistic effects with other amino acids, despite the prevalence of such combination therapies in the existing literature.

Conclusions

This integrated analysis of 27 clinical trials, encompassing 1,030 participants demonstrates that taurine supplementation improves markers of endothelial function, metabolic homeostasis, and chronic inflammation, which are pathological processes central to the cellular senescence observed in PASC. A daily dose of approximately 3,000 mg appears to balance clinical efficacy with tolerability. Furthermore, the observed reduction in plasma taurine levels in individuals with PASC compared to their recovered counterparts suggests a deficit in the body's capacity to manage oxidative stress and inflammation, key features of PASC, possibly through increased consumption and reduced enterohepatic recycling. Given this compelling evidence, future studies should consider taurine supplementation for individuals grappling with the debilitating effects of PASC and other virus-induced senescent conditions.

Abbreviations

SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2
ACE2	Angiotensin-converting enzyme 2
PASC	Post-acute sequalae of COVID-19
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
SMD	Standardized mean differences
CI	Confidence interval
HbA1c	Glycated hemoglobin
FBG	Fasting blood glucose
HOMA-IR	Homeostatic model assessment for insulin resistance
HDL	High-density lipoprotein
LDL	Low-density lipoprotein
CRP	C-reactive protein
MDA	Malondialdehyde

Supplementary Information

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Supplementary Material 1

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Author contributions

Conception and design of the study: K.W. and G.Y.O.; Data acquisition, analysis or interpretation: K.W., C.M., M.K., and J.Y.K.; Drafted the manuscript or substantively revised it: K.W., C.M., M.K., J.Y.K, and G.Y.O. All authors reviewed and approved the final manuscript.

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Data availability

This review is based on data extracted from publicly available studies. The authors confirm that the data supporting the findings of this study are available within the article and its supplementary materials. The characteristics of all included studies can be found in Table 1 and Supplemental Table 4. Supplemental Tables 2 and 3 included all search terms used to access the clinical databases.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
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Competing interests
The authors declare no competing interests.

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