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Systematic Review

Systematic Review of Literature Up to 31 December 2025 on the Potential Health Benefits of Cinnamon

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Abstract

Cinnamon (*Cinnamomum*) is often marketed as a natural remedy for various health conditions, which requires verification. This systematic review evaluates the clinical and preclinical evidence behind cinnamon's purported health benefits. Following a database search of literature published up to 31 December 2025 in PubMed and followed by six inclusion criteria, 26 of the original 201 studies were included. These studies were grouped into four categories: (i) cardiometabolic health and glycemic regulation; (ii) anti-inflammatory, antioxidant and tissue protection; (iii) other bioactive effects including antimicrobial and longevity; and (iv) safety and toxicology. The most robust finding across the literature is that cinnamon acutely blunts post-prandial glucose spikes in healthy and overweight adults, while significantly reducing post-prandial insulin specifically in overweight and obese populations. Beyond this short-term effect, the clinical evidence is inconsistent. Results for long-term blood sugar control (HbA1c) are mixed. Though animal studies show cardiovascular benefits, human trials do not. Furthermore, claims regarding brain health, gut health, antimicrobial effects, and reproduction have not been proven in humans. Safety data is incomplete. Low doses (up to 500 mg/day) appear safe for healthy adults but providing standard clinical doses to lactating animals cause metabolic issues in their offspring. In conclusion, cinnamon is a useful dietary addition for managing post-meal blood sugar but current evidence does not support broader medical claims. To establish true clinical utility, future research requires chemically standardized extracts, thorough safety testing, and larger human trials lasting at least 12 weeks.

Keywords: *Cinnamon (Cinnamomum); Health Benefits; HbA1c*

Introduction

Cinnamon is the dried inner bark of trees in the *Cinnamomum* genus (Lauraceae family). Two species dominate commercially: Ceylon cinnamon (*Cinnamomum zeylanicum* or *Cinnamomum verum*), grown mainly in Sri Lanka [1,2], and Chinese cinnamon (*Cinnamomum cassia*) [3,4]. While used interchangeably in cooking, they differ chemically. Notably, *C. cassia* contains higher levels of coumarin, a compound that can cause liver damage at high doses, making this an important factor for supplement safety [1]. Both species share key bioactive compounds, primarily cinnamaldehyde. This is the main bark constituent, the source of cinnamon's flavor and aroma, and a TRPA1-receptor activator [2,5]. Other shared compounds include A-type procyanidins [6], cinnamic acid, and eugenol [7].

Historically, cinnamon has been used in cooking, traditional medicine, and religious practices across Asia, the Middle East, and Europe. Its traditional applications for digestive issues, respiratory conditions, and diabetes-like symptoms have shaped much of the current cardiometabolic research agenda [2,8]. This long history is a useful starting point for thinking about safety but it does not by itself establish efficacy for any specific outcome. Global rates of type 2 diabetes, metabolic syndrome, obesity, and cardiovascular disease have risen sharply in recent decades. In 2024, an estimated 589 million adults had diabetes, a number projected to reach 853 million by 2050 [9]. Adult obesity has doubled since 1990, affecting 890 million people in 2022 [10], and cardiovascular disease remains the leading cause of mortality, causing 17.9 million deaths annually [11]. Culinary spices are increasingly promoted as low-cost additions to medical treatment to help manage these conditions, with cinnamon featuring prominently due to its traditional use and reported metabolic activity. However, habitual cinnamon intake is very low, averaging just 71 mg/day in one large Iranian cohort [12]. This falls far short of the 1.5 to 6 g/day typically tested in clinical trials.

The current evidence base for cinnamon is highly varied in design, dose, and quality. Research spans human randomized controlled trials [8,13], animal models [5,14], cell-based mechanistic studies [4], and large epidemiological cohorts [12]. The results are inconsistent. Some trials show glycemic or cardiovascular improvements, while others using similar doses in comparable populations report null results [15-17]. Existing reviews tend to focus narrowly on a single outcome, usually blood-glucose control, leaving a gap in evaluating broader claims around cardiovascular, anti-inflammatory, neuroprotective, antimicrobial, and anti-obesity benefits. Furthermore, because many striking findings originate from animal studies untested in humans, the strength of the evidence varies significantly. A systematic review is necessary to map this evidence, evaluate study quality, and separate proven benefits from preliminary claims. Hence, this systematic review evaluates the available evidence regarding cinnamon's health benefits. The 26 included studies were initially coded against ten themes: glycemic control, cardiovascular health, obesity and metabolic syndrome, anti-inflammatory and antioxidant effects, neuroprotection, gut health, antimicrobial activity, longevity, safety and toxicology, and reproductive health. To account for biological overlap, these themes were consolidated into four broad categories: (i) cardiometabolic health and glycemic regulation, (ii) anti-inflammatory, antioxidant, and tissue-level protection, (iii) other bioactive effects (including antimicrobial and longevity), and (iv) safety and toxicology. Within each category, this review synthesizes the findings, applies critical methodological appraisal, and distinguishes effects proven in adequately designed human trials from those based solely on preclinical work, ultimately establishing what can responsibly be concluded for human health.

Methodology

Search strategy: A comprehensive literature search was conducted on 1 April 2026 using the PubMed database for studies up to 31 December 2025, with the following search string: "cinnamon AND health AND benefi*". This resulted in the following search URL https://pubmed.ncbi.nlm.nih.gov/?term=cinnamon+AND+health+AND+benefi*&filter=dates.1000/1/1-2025/12/31.

Inclusion/exclusion criteria: Following PRISMA [18] guidelines, six eligibility criteria were applied sequentially. First, full-text articles were required for a complete evaluation of study methods. Second, publications had to be in English for accurate interpretation, acknowledging a minor risk of language bias. Third, only primary research was included to allow direct analysis of original data. Fourth, cinnamon had to be either the primary intervention or a central focus of the study. Studies using cinnamon only peripherally in complex multi-ingredient blends were excluded. Fifth, the research needed to measure a specific health outcome, excluding studies that did not assess health effects or were otherwise irrelevant. Finally, the studies had to involve human or animal subjects.

Results and Discussion

26 of the original 201 studies were included and initially coded against ten predefined health-benefit themes: (1) glycemic control, (2) cardiovascular health, (3) obesity and metabolic syndrome, (4) anti-inflammatory and antioxidant effects, (5) neuroprotection, (6) gut health, (7) antimicrobial activity, (8) longevity, (9) safety and toxicology and (10) reproductive health. A study could be assigned to more

than one theme where it reported multiple outcomes, with the primary theme set by the study's stated objective. Because ten descriptive themes were too fragmented for coherent critical discussion, they were consolidated into four overarching categories: cardiometabolic health and glycemic regulation, anti-inflammatory, antioxidant and tissue-level protection, other bioactive effects (antimicrobial activity and longevity), and safety and toxicology.

Table 1 outlines the thematic landscape of the 26 included studies, providing the predominant study design and the ratio of human to animal models per category. While the evidence base is broad, its distribution is uneven. The cardiometabolic category contains 15 of the 26 studies and is the only area dominated by human randomized controlled trials. In contrast, the remaining three categories rely primarily on preclinical evidence.

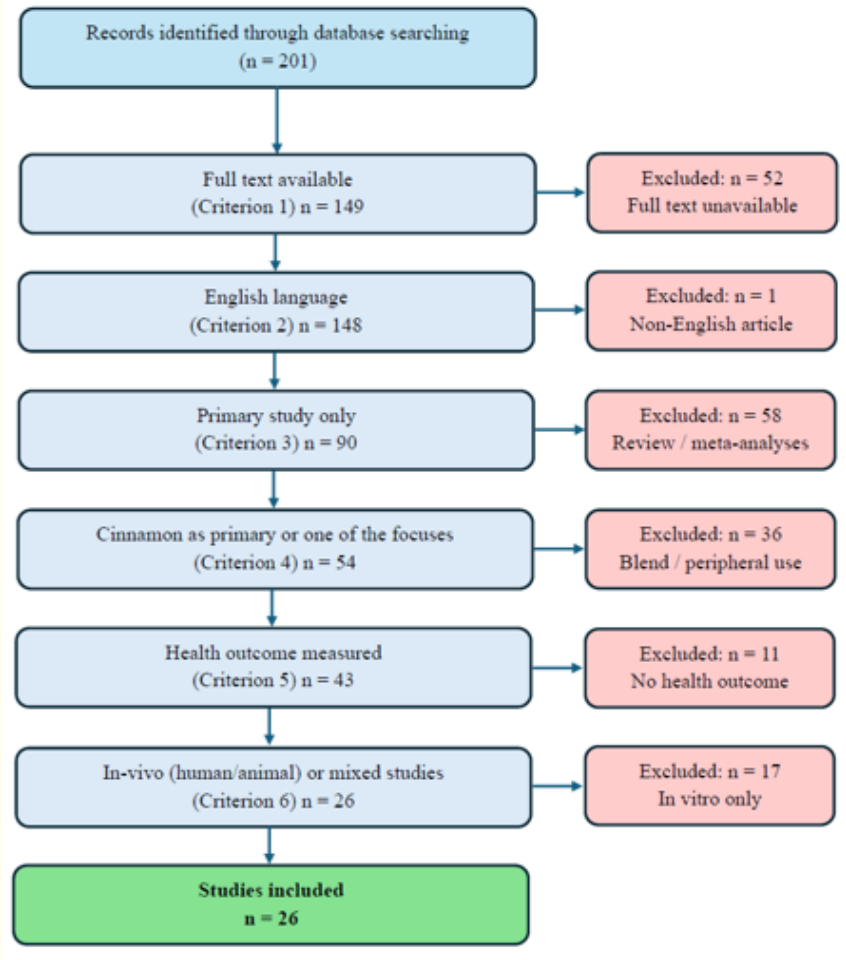

Figure 1: *PRISMA flow diagram of the six sequential eligibility criteria screening process.*

Theme 1: Cardiometabolic health and glycemic regulation

This category brings together the three most heavily studied themes: glycemic control (n = 7), cardiovascular health (n = 4) and obesity and metabolic syndrome (n = 4). They were grouped because they form an interlocking metabolic-syndrome cluster: insulin resistance, central adiposity, abnormal blood lipids, raised blood pressure and vascular dysfunction tend to co-occur and share underlying mechanisms.

Reference	Study Design	Population/Model	Dose, Duration and Species	Key Findings
[21]	12-week double-blind RCT (4-arm spice trial)	97 completing adults with or at risk of metabolic syndrome (UAE)	3 g/day cinnamon powder, 12 weeks. Species unidentified	Largest HbA1c reduction across spices (-0.596%, p ≤ 0.001), small FBG reduction (-1.27 mg/dL), LDL/TC unchanged.
[20]	Acute single-blind cross-over RCT	25 overweight / obese adults	Cinnamon-containing test meal (~1 g per 135 kcal), single 24-h challenge. Species unidentified	Reduced post-prandial glycemic response (p = 0.01), reduced post-prandial insulin at 0.5h (p = 0.04)
[3]	Three-arm dose-response RCT (no placebo)	41 healthy adults	*C. cassia* 1, 3 or 6 g/day, 40 days	Post-prandial blood glucose fell at all three doses (p < 0.05), fasting blood glucose fell only at 6 g/day, HbA1c, weight and BMI unchanged
[2]	120-day double-blind, placebo-controlled RCT	154 T2DM patients (Northern India)	*C. zeylanicum* (Alba grade) 1.5 g/day, 120 days.	FBG -35.50 mg/dL (95% CI -173 to 58.4), HbA1c -0.85%, significant PTP1B inhibition in PBMCs (p = 0.039)
[8]	60-day double-blind, placebo-controlled RCT	99 T2DM patients (Thailand)	*C. cassia* 1.5 g/day, 60 days	P ≤ 0.001 for HbA1c, triglycerides, and blood pressure, p = 0.026 for fasting glucose
[15]	6-week double-blind, placebo-controlled RCT	25 postmenopausal T2DM women (Netherlands)	*C. cassia* 1.5 g/day, 6 weeks	No significant change in insulin sensitivity, OGTT or fasting lipid profile
[19]	Acute open-label cross-over pilot	Adults stratified by weight (normal weight, overweight/obese)	6 g cinnamon (Korintje cinnamon, ground from cassia bark) in oatmeal, single meal. Species: *Cinnamomum cassia*	Overweight/obese: post-prandial insulin iAUC reduced ~18.7% (p = 0.001), 60-min glucose rose, normal-weight: glucagon fell
[16]	8-week parallel single-blind RCT (5 arms)	~204-208 T2DM adults (Iran)	*C. verum* 3 g/day, dissolved in black tea, 8 weeks	No between-group difference in SBP, DBP or sICAM-1 in cinnamon arm, ginger and saffron reduced sICAM-1, cinnamon did not

[23]	Animal experimental (*in vivo* + *ex vivo* Langendorff)	30 male Wistar rats, 5 groups, 8 weeks training/supplementation	*C. zeylanicum* methanolic bark extract 200 mg/kg/day, 8 weeks,	Enhanced cardiac contractility, lower TC, LDL, MDA, higher HDL and HDL/LDL ($p < 0.01$), reduced exercise-induced MDA ($p < 0.01$)
[17]	8-week double-blind, placebo-controlled RCT	39 completing T2DM adults (Iran), same cohort as Davari., *et al*. 2020 (20 subjects in cinnamon and 19 in placebo)	Cinnamon 3 g/day capsules, 8 weeks. Species unidentified	Within-group reductions in sICAM-1 and sVCAM-1 in both arms ($p < 0.001$), no between-group difference (sICAM-1 $p = 0.69$, sVCAM-1 $p = 0.72$)
[24]	Animal experimental (*in vivo* regional ischaemia-reperfusion)	32 male Sprague-Dawley rats, 4 groups (8/arm)	*C. zeylanicum* HPLC-standardised ethanolic extract 50, 100, 200 mg/kg/day, 14 days pre-treatment	Reduced infarct size, ventricular tachycardia and ectopic beats, stabilised ECG, higher SOD/GPx, lower cTnI, LDH, MDA ($p < 0.05$)
[5]	Mixed mouse experiments + cell line	C57BL/6 mice (lean acute, DIO chronic), MGN3-1 ghrelin-secreting cells	Cinnamaldehyde 250 mg/kg acute (oral gavage), CIN-supplemented diet for 5 weeks chronic. Species unidentified	Reduced food intake and gastric emptying, *in vitro* ghrelin secretion fell ~44% (TRPA1-mediated), chronic: lower weight gain, improved GTT, higher fatty-acid oxidation gene expression
[26]	Animal experimental (developmental obesity rat model)	Male Wistar rats, small-litter overfed model	Cinnamaldehyde 40 mg/kg/day, postnatal days 30-60, outcomes at days 60 and 180. Species unidentified	Reduced adipocyte hypertrophy, lower lipogenesis genes, higher oxidative-metabolism genes, higher BAT thermogenesis, effects persisted to PND 180
[22]	8-week 4-arm RCT (no placebo)	40 male overweight/obese soldiers, 10 per arm	Cinnamon 1.5 g/day ± Tabata exercise, 8 weeks. Species unidentified	Tabata + cinnamon arm: largest reductions in body mass, BMI, body-fat %, FBS, HOMA-IR, liver enzymes, CRP, TNF-α, higher adiponectin and irisin ($p < 0.001$ vs other arms)
[25]	Animal experimental (HFD-induced obesity)	C57BL/6J mice on high-fat diet for 8 weeks	Cinnamon bark extract supplemented in HFD, 8 weeks	Lower fat-mass gain, adipose-tissue inflammation, liver steatosis, improved GTT and IR index, lower Peptococcus, higher antimicrobial peptides and tight-junction proteins

Table 2: *Studies in the cardiometabolic category (n = 15) ordered by underlying theme: glycemic control, cardiovascular health, then obesity and metabolic syndrome.*

Across the seven studies coded under glycemic control, one of the most consistently potential benefits of cinnamon is its ability to consistently improve the body's metabolic response after a meal, specifically glucose and insulin regulation. For example, one study found that adding 6g of cinnamon to a heavy, high-fat meal significantly lowered both post-meal glucose ($p = 0.01$) and insulin ($p = 0.04$) in overweight and obese adults [20]. However, this glucose-lowering effect is not always guaranteed. Another study testing with the same dose of cinnamon with an oatmeal breakfast found that it successfully lowered post-meal insulin in overweight adults ($p = 0.001$) but it did not lower their glucose and that the cinnamon had no significant impact on either insulin or glucose for normal-weight participants [19]. Looking beyond single meals, a 40-day trial showed that consuming 1, 3, or 6g of cinnamon daily consistently reduced 2-hour post-meal glucose spikes for healthy adults [3]. Despite these reliable post-meal benefits, only the maximum 6 g/day dose lowered fasting blood glucose, and long-term blood sugar levels (HbA1c) remained completely unchanged across all doses [3].

While these three different designs, a single-meal challenge, a single-dose crossover, and a 40-day dose-response trial do not yield identical glycemic outcomes, they converge on demonstrating acute improvements in post-prandial metabolic responses. This consistency makes the post-prandial effect the most robustly supported conclusion in this review. Clear mechanisms support these dual clinical observations: cinnamaldehyde activates the TRPA1 receptor on enteroendocrine cells, slowing gastric emptying to blunt glucose absorption [5], and cinnamon compounds inhibit PTP1B, a negative regulator of insulin signaling, in the peripheral blood mononuclear cells of type 2 diabetes patients ($p = 0.039$) [2]. Clinically, cinnamon can mitigate glucose spikes from certain meals and reliably improves the early insulin response. This is a real benefit for people who struggle with post-meal metabolic spikes but it is far narrower than the popular claim that cinnamon is a general diabetes treatment.

Results for long-term glycemic control are far less consistent. A trial of 99 Thai patients with type 2 diabetes reported significant HbA1c reductions after 1.5 g/day *C. cassia* for 60 days ($p < 0.005$), alongside falls in triglycerides and blood pressure [8]. Yet the same dose given for six weeks to 25 postmenopausal women with type 2 diabetes produced no change in insulin sensitivity, oral glucose tolerance or lipids [15]. A 120-day trial reported a 35.50 mg/dL fall in fasting blood glucose but the 95% confidence interval (-173 to 58.4 mg/dL) crossed zero, indicating the trial was underpowered to confirm the effect [2]. In a 12-week four-spice trial, fasting glucose fell by a statistically significant but clinically negligible -1.27 mg/dL, alongside an HbA1c reduction of -0.596% ($p \leq 0.001$) [21]. The long-term evidence is therefore mixed: the positive effects are modest, and the null trials are of similar methodological quality to the positive ones. A consistent acute effect does not yet translate into a reliable long-term HbA1c benefit at the doses and durations tested.

The four cardiovascular studies divide sharply: animal data are encouraging but the human data show no effect. In male Wistar rats, 200 mg/kg/day of methanolic *C. zeylanicum* bark extract for eight weeks enhanced cardiac contractility and improved lipid profiles (lower total cholesterol and LDL, $p < 0.01$, HDL raised, $p < 0.05$, mainly with concurrent training), and reduced exercise-induced lipid peroxidation (lower MDA, $p < 0.01$) [23]. An HPLC-standardized *C. zeylanicum* extract at 50, 100 and 200 mg/kg/day protected against induced ischemia-reperfusion injury, reducing infarct size and arrhythmias, stabilizing the electrocardiogram, and lowering serum cardiac troponin I, lactate dehydrogenase and MDA ($p < 0.05$) [24].

By contrast, both human trials returned null between-group results. A five-arm spice trial in patients with type 2 diabetes found cinnamon had no effect on blood pressure or the adhesion molecule sICAM-1 [16]. An 8-week protocol that also produced inflammatory data [17] reported within-group reductions in sICAM-1 and soluble vascular cell adhesion molecule-1 (sVCAM-1) of $p < 0.001$ in both arms but between-group p-values of 0.75 and 0.72 indicate no cinnamon-specific advantage. Methodologically, when treatment and placebo participants improve equally, a within-group reduction does not prove the treatment worked. Both human trials were limited by small cohorts, and one delivered cinnamon in black tea, a vehicle carrying caffeine and tea polyphenols with their own cardiovascular effects [16]. The cardiovascular benefits seen in animals therefore remain biologically plausible but clinically unproven.

Three of the four studies on obesity and metabolic syndrome rely on animal models, yet they outline a highly consistent set of mechanisms. At the cellular level, cinnamaldehyde suppressed the hunger hormone ghrelin by roughly 44%. In mice, this suppression directly reduced food intake, delayed gastric emptying, and limited weight gain while activating fat-burning genes (*ACSL4*, *Cpt1a*) [5]. Beyond appetite control, cinnamaldehyde appears to alter how fat is physically stored. When adolescent rats received 40 mg/kg/day from postnatal day 30 to 60, the treatment prevented fat cells from enlarging, suppressed fat-storage pathways, and increased calorie-burning thermogenesis (brown-adipose-tissue thermogenesis, *Ucp1*). Notably, the physiological improvements to white fat persisted up to day 180, long after the supplementation stopped [26]. Finally, the whole cinnamon bark extract delivered similar systemic benefits. It reduced overall fat accumulation, lowered tissue inflammation, and prevented fatty liver, while simultaneously improving blood sugar control and fortifying tight junction proteins by promoting beneficial gut bacteria [25].

The only human trial in this group reported the largest reductions in body-fat percentage, fasting blood sugar, HOMA-IR, liver enzymes, CRP and TNF-α in a combined Tabata exercise and cinnamon group, which significantly exceeded the benefits of taking cinnamon alone [22]. However, the trial lacked a placebo arm and recruited only 10 male soldiers per group. While the study's cinnamon-only arm showed modest benefits, the most dramatic improvements relied heavily on the high-intensity Tabata exercise, meaning those outsized effects cannot be attributed to cinnamon. The obesity findings are mechanistically rich but predominantly preclinical, meaning the human evidence does not yet prove a clinically meaningful effect.

Synthesizing this category, the cardiometabolic evidence supports one narrow and well-replicated finding: doses of 1 to 6g lower post-meal blood glucose. The long-term glycemic effect remains inconsistent despite being biologically plausible, and current human data do not support a cardiovascular or anti-obesity benefit at the doses and durations tested.

In summary, cinnamon may help manage post-meal blood-sugar spikes in some adults but the evidence does not support its use as a primary treatment for type 2 diabetes, hypertension, abnormal lipids or obesity. Nevertheless, the mechanisms shared across cell, animal, and short-term human studies, particularly TRPA1 activation, PTP1B inhibition, and gut-barrier improvement, mark the most promising targets for future large-scale human trials.

Theme 2: Anti-inflammatory, antioxidant and tissue protection

This category brings together four themes that share one biological thread, namely the effect of cinnamon on oxidative stress and inflammation: anti-inflammatory and antioxidant effects (n=1) [13], neuroprotection (n=2) [6, 27], gut health (n=2) [4, 28] and reproductive health (n=1) [14]. Although these studies examine different tissues such as the blood, brain, gut and male reproductive tract, they measure the same two sets of biomarkers: markers of oxidative stress (reactive oxygen species, malondialdehyde, and the antioxidant enzymes superoxide dismutase, glutathione peroxidase and catalase) and markers of inflammation (NF-κB signalling and the cytokines TNF-α and IL-6). Grouping by shared biology provides a clearer interpretation of this small dataset.

Only one human randomized controlled trial sits in this category. It gave 3 g/day of cinnamon, with the species not stated, or a matching placebo for eight weeks to 39 patients with newly diagnosed type 2 diabetes [13]. Of five inflammation and antioxidant markers, only plasma NF-κB, a master switch of the inflammatory response, was significantly lower in the cinnamon group than in placebo at endpoint ($p = 0.02$). Meanwhile, hs-CRP ($p = 0.29$), TNF-α ($p = 0.27$), IL-6 ($p = 0.52$) and SIRT-1 ($p = 0.51$) showed no difference from placebo. One positive result against four null results, no correction for multiple testing, and an unidentified cinnamon species mean this is best read as a single isolated signal, not evidence of a broad anti-inflammatory effect in type 2 diabetes. A therapeutic dose may nudge one inflammatory pathway but it does not deliver the wide reduction in markers expected of a genuinely clinically effective anti-inflammatory treatment.

Reference	Study Design	Population / Model	Dose, Duration and Species	Key Findings
[13]	8-week double-blind, placebo-controlled RCT	39 completing newly diagnosed T2DM patients (Iran), 22 cinnamon, 22 placebo	Cinnamon 3 g/day capsules, 8 weeks. Species unidentified	Plasma NF-κB significantly reduced in cinnamon group ($p = 0.02$), no between-group difference for hs-CRP, TNF-α, IL-6 or SIRT-1
[27]	Animal experimental (non-transgenic AD rat model)	MSG-induced non-transgenic Alzheimer's disease rats	*C. zeylanicum* aqueous bark extract 50 mg/kg/day, 20 weeks	Improved maze learning, inhibited cholinesterase activity, higher phosphorylated GSK-3β, higher neuron count in hippocampal dentate gyrus
[6]	Animal (MPTP-PD mouse model) + *in vitro* SH-SY5Y	30 male C57BL/6 mice (10/group), SH-SY5Y dopaminergic-like cells	A-type cinnamon procyanidin oligomers (CPO-A) 150 mg/kg/day, 15 days. Species unidentified	Attenuated MPTP-induced loss of TH+ neurons and Th mRNA in substantia nigra, inhibited p38MAPK/P53/BAX cascade, *in vitro* blocked MPP+-induced ROS ($p < 0.01$)
[28]	Animal experimental (DSS-induced colitis)	Mice, 6 groups (control, DSS, ginger, cinnamon, ginger + cinnamon low, ginger + cinnamon high)	Cinnamon subcritical-water extract ± ginger, 21 days. Species unidentified	High-dose ginger+cinnamon (GCH): lower DAI, histology score, MPO, lower IL-1β, IL-6, TNF-α mRNA, cinnamon-only arm less effective than GCH
[4]	*In vitro* Caco-2 + *in vivo* DSS-colitis mice	Caco-2 human intestinal cells, DSS-colitis mice	*C. cassia* hot-water extract (HPLC-quantified for cinnamic acid)	*In vitro*: higher TEER and tight-junction protein expression, lower cytokine mRNA, *in vivo*: improved histological index, lower cytokine concentrations
[14]	Animal experimental (STZ-induced diabetic rat model)	80 male Wistar rats, 8 groups (n = 10 each), 56-day duration	*C. zeylanicum* 75 mg/kg/day ± ginger 100 mg/kg/day, 56 days	Cinnamon increased sperm number, viability, motility ($p < 0.05$), higher testosterone, LH, FSH, higher TAC, SOD, catalase, GPx, lower MDA, combination produced largest effects

Table 3: *Studies in the anti-inflammatory, antioxidant and tissue-level protection category (n = 6).*

Both neuroprotection studies are preclinical yet point the same way. The first study uses a rat model of Alzheimer's disease created with monosodium glutamate (MSG), which produces damage from excess glutamate rather than the amyloid-beta plaques and tau tangles that define the human disease [27]. Rats given 50 mg/kg/day of a water-based *C. zeylanicum* bark extract for 20 weeks learned mazes better, showed reduced activity of cholinesterase and had higher phosphorylated GSK-3β and more neurons in the hippocampal dentate gyrus, a region central to memory. The second study uses a mouse model of Parkinson's disease created with the toxin MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine), alongside SH-SY5Y cells that behave like dopamine-producing neurons [6]. MPTP causes a sudden chemical injury and does not reproduce the slow course of human Parkinson's disease. Treating the mice for 15 days with A-type cinnamon procyanidin oligomers at 150 mg/kg/day reduced the loss of dopamine neurons by blocking a cell-death pathway, p38MAPK/

P53/BAX. In the cell experiment, the same compounds prevented the build-up of reactive oxygen species ($p < 0.01$). Both studies rest on disease models of doubtful human relevance, use doses far above realistic supplement levels once scaled to body weight, and used only male animals in the Parkinson's model. With no human data, neuroprotection remains a research hypothesis rather than an established benefit of cinnamon.

Both gut-health studies used a mouse model of colitis; an inflammation of the colon produced with dextran sulphate sodium (DSS). The first tested a combined ginger and cinnamon preparation over 21 days. At a high dose, the mixture restored lost body weight, lowered the disease activity index and histology score, reduced myeloperoxidase, and suppressed the genes for IL-1β, IL-6 and TNF-α [28]. Because the study uses a multi-arm design, it was able to isolate the effects of the spices. While the high-dose combination produced the strongest suppression of inflammatory cytokines, the cinnamon-only arm was highly effective by itself, driving the majority of the improvements in weight recovery and physical disease symptoms.

The second gut study tested a hot-water extract of *Cinnamomum cassia*, its cinnamic acid content quantified by HPLC, in both Caco-2 cells and the same DSS colitis model [4]. In the cells, cinnamon strengthened the intestinal barrier, raising trans-epithelial electrical resistance, increasing tight-junction proteins and lowering cytokine genes. In mice, it improved the tissue-damage score and reduced colonic cytokine levels.

The two studies agree that cinnamon, alone or with ginger, eases DSS-induced colitis by protecting the gut barrier and dampening inflammation, consistent with the gut-microbiota and tight-junction findings in the cardiometabolic models [25]. The limits are clear: there are no human gut-health data, and while Park., *et al*. (2023) clearly identified their intervention as *C. cassia*, the first study failed to report the exact species used [28]. Despite this, because Im., *et al*. (2021) utilized a multi-arm design with a cinnamon-only control, both studies successfully isolated the independent effects of each spices. Cinnamon's effect on the gut barrier is plausible enough to justify human trials in inflammatory bowel disease but it is not yet proof of clinical benefit.

Reproductive health rests on a single animal study using male Wistar rats made diabetic with streptozotocin (STZ), in an eight-group design of 80 rats, 10 per group [14]. STZ causes diabetes by abruptly destroying the insulin-producing beta-cells of the pancreas, a sudden loss that is biologically different from the gradual insulin resistance and slow beta-cell decline of human type 2 diabetes, which limits how far the results carry over to people. Cinnamon alone at 75 mg/kg/day for 56 days, ginger alone at 100 mg/kg/day, and the two combined all raised sperm count, survival and motility relative to untreated diabetic rats. These gains came with higher testosterone, luteinizing hormone and follicle-stimulating hormone, and with stronger antioxidant defences, raising total antioxidant capacity, superoxide dismutase, catalase and glutathione peroxidase while lowering malondialdehyde ($p < 0.05$).

Although the combined ginger and cinnamon group improved the most, cinnamon's individual efficacy was clearly demonstrated because the study design explicitly included isolated spice arms. The pattern still fits the antioxidant theme, since much of the reproductive damage in diabetic rats is oxidative but with no human data a single animal study cannot support any reproductive-health claim for people.

Across the six studies, this category tells a consistent biological story: cinnamon's polyphenols and cinnamaldehyde act on the same oxidative-stress and inflammatory pathways in several tissues. The evidence, however, is almost entirely preclinical. The one human trial produced a single positive biomarker against four null results, and the animal and cell studies depend on disease models of doubtful relevance to human conditions. The anti-inflammatory and tissue-protective effects of cinnamon are therefore biologically plausible but should not be presented as proven, what they justify is properly powered human trials using clearly defined cinnamon preparations. The shared oxidative-stress mechanism running across these studies [4,6,13,14,22,28] is what the four-theme grouping is designed to make visible.

Theme 3: Other bioactive effects: Antimicrobial activity and longevity

This section reviews a theme that falls outside cinnamon's primary cardiometabolic and inflammatory effects: antimicrobial activity [7,29] and longevity [12].

Reference	Study Design	Population/Model	Dose, Duration and Species	Key Findings
[7]	Pharmaceutical formulation, *in vitro* + rat *in vivo*	Oral microbiota *in vitro*, rats for hot-plate analgesia	Cinnamon-oil nano-emulgel (NEG1) with 2.5% hydroxy-propyl cellulose. Species unidentified	Optimized globule size 92 nm, 95% stability, 23 mm zone of inhibition, enhanced mucosal permeation, 45-min hot-plate analgesia
[29]	Animal experimental (*E. stiedae*-induced hepatic coccidiosis)	60 New Zealand white rabbits, 6 groups (10/arm)	Cinnamon essential oil 100 mg/kg vs toltrazuril (15 ppm), artemisinin (200 ppm). Species unidentified	Cinnamon oil produced "partial" protection across clinical, parasitological and biochemical endpoints, clearly inferior to both toltrazuril and artemisinin
[12]	Prospective cohort study (Golestan, Iran)	~44,398 adults aged 40-75, up to ~10 years follow-up	Habitual dietary cinnamon (mean ~71 mg/day among consumers, FFQ-assessed). Species unidentified	No significant association between cinnamon and all-cause, cardiovascular or cancer mortality (turmeric, pepper and saffron showed inverse associations)

Table 4: *Studies in the "other bioactive effects" category, antimicrobial activity and longevity (n = 3).*

Antimicrobial evidence currently rests on laboratory and animal models rather than human trials. One study evaluated an optimized nano-emulsion gel containing 20% cinnamon oil, reporting a 23 mm zone of inhibition against the oral bacterium *Streptococcus mutans* and about 45 minutes of pain relief in a rat hot-plate test [7]. However, the research primarily tested the delivery technology rather than defining cinnamon's natural antimicrobial spectrum. A second study demonstrated that cinnamon essential oil provided only partial protection against parasitic liver infections in rabbits, performing significantly worse than standard pharmacological treatments such as toltrazuril and artemisinin [29]. These findings confirm baseline *in vitro* and animal-model activity but place cinnamon firmly below established pharmacological treatments. Research on cinnamon and human longevity currently shows no evidence of clinical benefit. An analysis of over 44,398 adults in the Golestan cohort found no significant association between regular cinnamon consumption and reduced mortality [12]. Although the average cohort intake (71 mg/day) was substantially lower than typical clinical doses (1.5 to 6.0 g/day), the data confirms that standard dietary consumption does not extend human lifespan. Ultimately, while these studies highlight diverse biological activities, neither the antimicrobial nor the longevity claims currently possess the clinical evidence required for practical health recommendations.

Theme 4: Safety and toxicology

Safety is kept as a standalone category because it acts as the analytical counterweight to the benefit claims of the previous three sections. Two studies address it directly: a Phase I human dose-escalation trial [1] and an animal developmental-programming study [30].

Reference	Study Design	Population/Model	Dose, Duration and Species	Key Findings
[1]	Open-label Phase I dose-escalation trial	28 healthy adults (Sri Lanka), mean age 38.8 years	*C. zeylanicum* water extract: 85 mg/day (month 1), 250 mg/day (month 2), 500 mg/day (month 3)	No serious adverse events or hepatotoxicity, significant lower SBP, DBP, TC, LDL-c by month 3, 2 withdrawals (dyspepsia), compliance 85-95%
[30]	Animal experimental (developmental programming)	Lactating Wistar dams (control n = 9, cinnamon n = 11), male offspring assessed at PND 180	*C. zeylanicum* aqueous extract 400 mg/kg/day to lactating dams (~3-5 g/day human-equivalent), 20-day lactation	Adult male offspring: visceral obesity (p = 0.001), hyperleptinaemia (p = 0.002), hyperinsulinaemia (p = 0.016), mild hepatic steatosis (p = 0.001), lower PPAR-α mRNA

Table 5: *Safety and toxicology studies (n = 2).*

A three-month trial provides reassuring safety data for Ceylon cinnamon in healthy adults [1]. Among participants taking escalating doses from 85 mg/day to 500 mg/day, there were no serious adverse events or hepatotoxicity reported. However, four participants did experience non-serious dyspeptic symptoms, which necessitated the withdrawal of two individuals from the study, leaving 28 participants to complete the trial. While the remaining cohort showed modest cardiometabolic improvements and tolerated a maximum dose of 500 mg/day, it is important to note that this was a highly concentrated extract (produced with an 8 to 9% manufacturing yield) that corresponds exactly to the upper standard therapeutic dose of 6.0 g/day of raw cinnamon powder. Therefore, while this upper therapeutic equivalent was generally well-tolerated, long-term safety data beyond three months remains limited.

Preclinical evidence from a maternal-exposure model is more concerning [30]. Lactating Wistar rats were given a water-based *C. zeylanicum* extract at 400 mg/kg/day across a 20-day lactation period, a dose equivalent to roughly 3 to 5 g/day for a 70 kg human (64 mg/kg) and squarely within the range used in human trials. By day 180, the adult male offspring showed visceral obesity ($p = 0.001$), high leptin ($p = 0.002$), high insulin ($p = 0.016$) and mild hepatic steatosis ($p = 0.001$), together with reduced liver expression of PPAR-alpha mRNA ($p = 0.005$). This highlights a delayed developmental programming effect resulting from maternal supplementation. While animal models require cautious translation, the direct overlap with human clinical doses indicates that cinnamon supplementation during lactation is not yet proven safe for breastfed infants, and safety data for pregnant or breastfeeding women across full clinical doses are missing.

Two further safety points emerge in this review. First, distinguishing between cinnamon species is critical because *C. cassia* contains substantially more hepatotoxic coumarin than *C. zeylanicum*, posing greater risks to liver function and coagulation with regular use [1]. Second, even when evaluating the safer *C. zeylanicum* species, current clinical safety data remains restricted by methodological limitations. For instance, the safety trial in healthy adults was limited by a small sample size, a short three-month duration, a lack of a control group, and an inability to quantify the exact active compounds in the extract used [1]. Without knowing the exact species and chemical composition tested, safety and efficacy findings cannot be confidently generalized.

The animal-to-human translation problem: A major limitation in cinnamon research is its heavy reliance on animal studies that often do not translate to human outcomes. The gap is clearest in cardiovascular research, where animal studies show strong cardiac benefits such as in this two studies: [23,24]. However, outcome in human trials [16,17] showed none. Three factors are identified why animal findings so often fail in people. The first is unrealistic dosing. As detailed in earlier sections, many preclinical models utilize doses that scale to

an impractical 10.5 to 14 grams a day for an average human [6,23], rather than maintaining realistic therapeutic ranges of 3.5 to 5.0 grams a day, this can be seen in both [24,30]. The second is abrupt disease modeling. Several studies use harsh chemicals to instantly replicate conditions like Alzheimer's, Parkinson's, or diabetes [6,14,27], which fails to capture the slow, gradual progression of these diseases in humans. While diet-induced obesity models are closer to real life [5,25], interspecies differences in gut microbiota still complicate direct comparisons. The third is the preparation of the cinnamon extract. Describing an intervention merely as a water- or alcohol-based extract is too vague, as active compounds vary widely. Studies such as [2,4,24] quantifying active compounds via HPLC or GC-MS are significantly more reliable and more readily translated to human doses.

Methodological limitations and standardization: Many human trials rely on samples that are too small to produce reliable results [13,15,17]. When studies lack a placebo control group [3], it becomes impossible to prove that cinnamon is the reason for the health improvements, rather than natural data fluctuations or dietary shifts. In addition, trials lasting only 6 to 12 weeks are simply too short to capture meaningful shifts in long-term markers like HbA1c.

An even bigger issue is the failure to standardize the cinnamon itself. Out of the 26 studies reviewed, nearly half either used isolated compounds or failed to identify the exact *Cinnamomum* species tested. Since active compounds like cinnamaldehyde and coumarin vary wildly between species, leaving this information out makes it incredibly difficult to replicate the research or translate it into human interventions. To fix this, future trials must chemically profile their interventions, following the standard set by studies using analytical methods like HPLC to track specific marker compounds [4].

Conclusion

Across the 26 included studies, the most robust clinical benefit of cinnamon is the acute reduction of post-meal glucose and insulin spikes. However, evidence for long-term glycemic control remains highly inconsistent. The claims on cardiovascular and anti-inflammatory benefits rely predominantly on preclinical models, while neuroprotective, antimicrobial, and reproductive benefits lack human validation entirely. This weak efficacy profile is compounded by pervasive methodological flaws, including small sample sizes, short trial durations, uncharacterized *Cinnamomum* species, and poor placebo controls.

Safety data is also unresolved. While low doses up to 500 mg/day appears to be well tolerated, the maternal exposure to a standard clinical dose of *C. zeylanicum* in lactating rats produced visceral obesity and hepatic steatosis in the offspring, raises a serious concern for high-dose supplementation during breastfeeding. This also points to a crucial difference between using cinnamon as a spice and taking it as a high-dose supplement. The average dietary consumption of cinnamon is approximately 71 mg/day and shows no measurable health impact. This means that the potential health benefits seen in clinical trials are strictly limited to concentrated supplements of 1.5 to 6 g/day and not to standard cooking.

Due to the risks observed in developmental programming models, clinical guidelines should advise against high-dose cinnamon supplementation during lactation until further preclinical toxicology is established. Future research should prioritize rigorously controlled human trials lasting at least 12 weeks with chemically standardized, species-identified preparations. Based on the current evidence, cinnamon is a useful dietary adjunct for managing post-meal glucose spikes but it cannot be recommended as a primary therapeutic intervention for type 2 diabetes or any other chronic metabolic conditions.

Supplementary Materials

Supplementary materials can be downloaded from https://bit.ly/Cinnamon_SR.

Conflict of Interest

The authors declare no conflict of interest.

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Bibliography

1. Ranasinghe P., *et al*. "Evaluation of pharmacodynamic properties and safety of Cinnamomum zeylanicum (Ceylon cinnamon) in healthy adults: a phase I clinical trial". *BMC Complementary and Alternative Medicine* 17.1 (2017): 550.

2. Mandal A., *et al*. "Efficacy of young *Cinnamomum zeylanicum* blume bark on hyperglycemia and PTPase activity in type 2 diabetes". *Cureus* 15.2 (2023): e35023.

3. Kizilaslan N and Erdem NZ. "The effect of different amounts of cinnamon consumption on blood glucose in healthy adult individuals". *International Journal of Food Science* (2019): 4138534.

4. Park TG., *et al*. "Cinnamon (*Cinnamomum cassia*) hot water extract improves inflammation and tight junctions in the intestine *in vitro* and *in vivo*". *Food Science and Biotechnology* 32.13 (2023): 1925-1933.

5. Camacho S., *et al*. "Anti-obesity and anti-hyperglycemic effects of cinnamaldehyde via altered ghrelin secretion and functional impact on food intake and gastric emptying". *Scientific Reports* 5.1 (2015): 7919.

6. Xu Q., *et al*. "A-type cinnamon procyanidin oligomers protect against 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine-induced neurotoxicity in mice through inhibiting the P38 mitogen-activated protein kinase/P53/BCL-2 associated X protein signaling pathway". *The Journal of Nutrition* 150.7 (2020): 1731-1737.

7. Hosny KM., *et al*. "Development and optimization of cinnamon oil nanoemulgel for enhancement of solubility and evaluation of antibacterial, antifungal and analgesic effects against oral microbiota". *Pharmaceutics* 13.7 (2021): 1008.

8. Sengsuk C., *et al*. "Effect of cinnamon supplementation on glucose, lipids levels, glomerular filtration rate, and blood pressure of subjects with type 2 diabetes mellitus". *Diabetology International* 7.2 (2016): 124-132.

9. International Diabetes Federation. IDF Diabetes Atlas (2025).

10. World Health Organization. "Obesity and overweight" (2024).

11. World Health Organization. "Cardiovascular diseases (CVDs)" (2021).

12. Hashemian M., *et al*. "Turmeric, pepper, cinnamon, and saffron consumption and mortality". *Journal of the American Heart Association* 8.18 (2019): e012240.

13. Davari M., *et al*. "Effects of cinnamon supplementation on expression of systemic inflammation factors, NF-kB and Sirtuin-1 (SIRT1) in type 2 diabetes: a randomized, double blind, and controlled clinical trial". *Nutrition Journal* 19.1 (2020): 1.

14. Khaki A., *et al*. "The anti-oxidant effects of ginger and cinnamon on spermatogenesis dys-function of diabetes rats". *African Journal of Traditional, Complementary and Alternative Medicines* 11.4 (2014): 1.

15. Vanschoonbeek K., *et al*. "Cinnamon supplementation does not improve glycemic control in postmenopausal type 2 diabetes patients". *The Journal of Nutrition* 136.4 (2006): 977-980.

16. Azimi P., *et al*. "Effect of cinnamon, cardamom, saffron and ginger consumption on blood pressure and a marker of endothelial function in patients with type 2 diabetes mellitus: A randomized controlled clinical trial". *Blood Pressure* 25.3 (2016): 133-140.

17. Mirmiran P., *et al*. "A randomized controlled trial to determining the effect of cinnamon on the plasma levels of soluble forms of vascular adhesion molecules in type 2 diabetes mellitus". *European Journal of Clinical Nutrition* 73.12 (2019): 1605-1612.

18. Liberati A., *et al*. "The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration". *PLoS Medicine* 6.7 (2009): e1000100.

19. Wang J., *et al*. "Acute effects of cinnamon spice on post-prandial glucose and insulin in normal weight and overweight/obese subjects: a pilot study". *Frontiers in Nutrition* 7 (2021): 619782.

20. Huang Y., *et al*. "Endothelial function and postprandial glucose control in response to test-meals containing herbs and spices in adults with overweight/obesity". *Frontiers in Nutrition* 9 (2022): 811433.

21. Al Dhaheri AS., *et al*. "The effect of therapeutic doses of culinary spices in metabolic syndrome: a randomized controlled trial". *Nutrients* 16.11 (2024): 1685.

22. Sabzevari Rad R., *et al*. "The combined effects of Tabata training and cinnamon supplementation on metabolic changes and body composition in soldiers with overweight or obesity". *Journal of the International Society of Sports Nutrition* 22.1 (2025): 2564237.

23. Badalzadeh R., *et al*. "The effect of cinnamon extract and long-term aerobic training on heart function, biochemical alterations and lipid profile following exhaustive exercise in male rats". *Advanced Pharmaceutical Bulletin* (2014).

24. Sedighi M., *et al*. "Protective effects of cinnamon bark extract against ischemia-reperfusion injury and arrhythmias in rat". *Phytotherapy Research* 32.10 (2018): 1983-1991.

25. Van Hul M., *et al*. "Reduced obesity, diabetes, and steatosis upon cinnamon and grape pomace are associated with changes in gut microbiota and markers of gut barrier". *American Journal of Physiology-Endocrinology and Metabolism* 314.4 (2018): E334-E352.

26. Neto JGO., *et al*. "Cinnamaldehyde treatment during adolescence improves white and brown adipose tissue metabolism in a male rat model of early obesity". *Food and Function* 13.6 (2022): 3405-3418.

27. Madhavadas S and Subramanian S. "Cognition enhancing effect of the aqueous extract of *Cinnamomum zeylanicum* on non-transgenic Alzheimer's disease rat model: Biochemical, histological, and behavioural studies". *Nutritional Neuroscience* 20.9 (2017): 526-537.

28. Im JA., *et al*. "Animal model of intestinal anti-inflammatory effect of ginger-cinnamon complex". *Food Science and Biotechnology* 30.9 (2021): 1249-1256.

29. Sorour S., *et al*. "Anticoccidial and hepatoprotective effects of artemisinin liquid extract, cinnamon essential oil and clove essential oil against *Eimeria stiedae* infection in rabbits". *Tropical Biomedicine* 35.4 (2018): 926-943.

30. Neto JGO., *et al*. "Maternal cinnamon intake during lactation led to visceral obesity and hepatic metabolic dysfunction in the adult male offspring". *Endocrine* 63.3 (2019): 520-530.

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