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Review Article

Cinnamon pharmacological mechanisms and clinical translation

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Abstract

Cinnamon, obtained from the bark of Cinnamomum trees, has a millennia-long history as a culinary spice and traditional medicine. Contemporary pharmacological investigations have revealed that its therapeutic potential is underpinned by a remarkably polypharmacological profile: a single natural product simultaneously engages the Nrf2/Keap1/ARE antioxidant axis, suppresses NF-κB/MAPK inflammatory cascades, and activates AMPK-dependent metabolic regulation. In this review, we synthesize the current evidence on these multi-target mechanisms and argue that such network-level activity challenges the reductionist assumption that complex diseases necessitate highly specific monotherapies, positioning cinnamon as an exemplary model of evolutionarily shaped multi-faceted therapeutics. Further we have evaluated the clinical evidence with particular emphasis on type 2 diabetes, cardiovascular disease, and inflammatory conditions. While preclinical findings are promising, we highlight that translational progress is substantially hindered by unresolved issues in product standardization, limited bioavailability of key constituents, and considerable inter-individual variability. We conclude that the path toward evidence-based therapeutic protocols requires a concerted focus on rationally designed delivery systems, personalized medicine strategies, and rigorous biomarker-driven clinical trials.

1. Introduction

Cinnamon, derived from the bark, leaves, and flowers of trees, belonging to the genus Cinnamomum (family Lauraceae), is one of the oldest and most economically important species in human history [1]. Archaeological evidence traces the use of cinnamon to approximately 2000 BCE in ancient Egypt, where it was employed in embalming rituals and valued as a luxury commodity [2]. Throughout classical antiquity, cinnamon was traded along the Spice Routes connecting Southeast Asia to the Mediterranean world, and its medicinal applications have been extensively documented in traditional medical systems, including Ayurveda, Traditional Chinese Medicine (TCM), and Greco-Arabic Unani medicine [3]. The genus *Cinnamomum* comprises over 300 species distributed across the tropical and subtropical regions of Asia, Australia, and the Pacific Islands, with *C. verum* (true cinnamon or Ceylon cinnamon), *C. cassia* (Chinese cinnamon), *C. burmannii* (Korintje cinnamon), and *C. loureiroi* (Saigon cinnamon or Vietnamese cinnamon) being the most commercially significant species [4].

The renewed scientific interest in cinnamon over the past two decades has been driven by the convergence of several factors: the global rise in chronic metabolic diseases, growing consumer demand for natural and plant-based therapeutics, and the development of advanced analytical techniques that have enabled the detailed characterization of cinnamon's complex phytochemical profile [5]. Modern pharmacological research has revealed that cinnamon contains a rich array of bioactive compounds, including volatile

essential oil components (cinnamaldehyde, eugenol, linalool, and camphor), polyphenolic compounds (procyanidins, catechins, and epicatechins), and various other secondary metabolites (coumarin, cinnamic acid, and cinnamyl alcohol) [6]. These compounds exhibit a remarkable breadth of biological activities, from potent antioxidant and anti-inflammatory effects to more specific actions, such as insulin sensitization, tumor suppression, and antimicrobial activity [7-12].

The molecular mechanisms underlying the pharmacological activities of cinnamon have been extensively investigated [8]. Key signaling pathways identified as primary targets include the Nrf2/Keap1/ARE antioxidant defense system [9] (Fig. 2), NF-kappaB/IKK inflammatory cascade [10] (Fig. 3), MAPK family (ERK, JNK, p38) stress response pathways [11], and AMPK energy-sensing metabolic regulator [12] (Fig. 4). These pathways are interconnected and form a complex regulatory network that governs cellular responses to oxidative stress, inflammation, metabolic dysregulation and oncogenic transformation. The ability of the bioactive constituents of cinnamon to modulate multiple targets within this network provides a mechanistic basis for its pleiotropic therapeutic effects and distinguishes it from single-target pharmaceutical agents.

Despite the substantial preclinical evidence supporting the therapeutic potential of cinnamon, the translation of these findings into clinical practice has been uneven [13]. Clinical trials evaluating cinnamon supplementation in type 2 diabetes, cardiovascular risk reduction, and other conditions have yielded mixed results, reflecting challenges related to product standardization, bioavailability limitations, inter-individual variability in response, and differences in study design and cinnamon preparations used [14]. This review aims to provide a comprehensive synthesis of the current evidence on the pharmacological mechanisms and clinical translation of cinnamon identify critical knowledge gaps, and propose future research directions that may accelerate the development of evidence-based cinnamon therapeutics.

2. Materials and methods

2.1. Literature search strategy

A comprehensive and systematic literature search was conducted to identify relevant studies on the pharmacological mechanisms and clinical translation of cinnamon. The search was performed across multiple electronic databases, including PubMed/MEDLINE, Scopus, Web of Science, Embase, and Google Scholar, covering publications from January 2000 to December 2023. The search strategy employed a combination of Medical Subject Headings (MeSH) terms and free-text keywords, including: 'cinnamon,' 'Cinnamomum,' 'cinnamaldehyde,' 'cinnamic acid,' 'eugenol,' 'coumarin,' 'proanthocyanidins,' 'pharmacological mechanisms,' 'antioxidant,' 'anti-inflammatory,' 'antidiabetic,' 'Nrf2,' 'NF-kappaB,' 'AMPK,' 'insulin sensitivity,' 'clinical trial,' and 'meta-analysis.' Boolean operators (AND, OR) were used to combine search terms systematically. The search was restricted to articles published in English in peer-reviewed journals.

2.2. Inclusion and exclusion criteria

Studies were included in this review if they met the following criteria: (1) original research articles, randomized controlled trials (RCTs), systematic reviews, and meta-analyses investigating the pharmacological activities of cinnamon or its bioactive constituents; (2) studies published in SCI-indexed peer-reviewed journals; (3) studies providing mechanistic insights into antioxidant, anti-inflammatory, antidiabetic, or other pharmacological effects of cinnamon; (4) clinical trials evaluating cinnamon supplementation in human subjects with metabolic or inflammatory conditions. Studies were excluded if they: (1) were non-English language publications without available translations; (2) were conference abstracts, letters, editorials, or non-peer-reviewed preprints; (3) lacked sufficient methodological detail or reported only preliminary data; (4) investigated cinnamon only as a flavoring agent without pharmacological evaluation; (5) were duplicate publications or contained overlapping datasets.

2.3. Data extraction and synthesis

Data extraction was performed independently by two reviewers using a standardized data extraction form. For preclinical studies, the following information was

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recorded: study model (in vitro or in vivo), cinnamon species and preparation used, bioactive compounds investigated, dose ranges, duration of treatment, molecular targets and signaling pathways examined, and key pharmacological outcomes. For clinical trials, additional data points included: study design (RCT, crossover, etc.), sample size, participant demographics, cinnamon preparation and dosage, intervention duration, primary and secondary endpoints, and main findings. Discrepancies between reviewers were resolved through discussion and consensus. The extracted data were synthesized thematically, organized by pharmacological mechanism and clinical application, and presented in a narrative review format supplemented with summary tables and mechanistic pathway figures.

3. Results and discussion

3.1. Phytochemical composition

The phytochemical complexity of cinnamon is a defining characteristic that underpins its diverse pharmacological activities [15]. Comprehensive phytochemical analyses using gas chromatography-mass spectrometry (GC-MS), liquid chromatography-tandem mass spectrometry (LC-MS/MS), and nuclear magnetic resonance (NMR) spectroscopy have identified hundreds of compounds in various Cinnamomum species [16] (Fig. 1). These compounds can be broadly categorized into volatile essential oil constituents and non-volatile polyphenolic compounds, each contributing to distinct biological activities and therapeutic potentials.

3.2. Essential oil components

The essential oil fraction of cinnamon bark typically constitutes 1-2.5% of the dry weight and is dominated by cinnamaldehyde (3-phenyl-2-propenal), which accounts for 50-95% of the volatile fraction, depending on the species and processing conditions [17]. Cinnamaldehyde is the principal bioactive compound responsible for cinnamon's characteristic aroma and many of its pharmacological activities, including antimicrobial, anti-inflammatory, and antidiabetic effects [18]. Other significant volatile constituents include eugenol (4-allyl-2-methoxyphenol), which is particularly abundant in *C. verum* and *C. loureiroi*, and reported for analgesic and anti-inflammatory properties. Also, linalool (3,7-dimethyl-1,6-octadien-3-ol), a monoterpene alcohol with anxiolytic and sedative activities. However, camphor, reported as local counterirritant and rubefacient properties [19].

3.3. Non-volatile polyphenolic compounds

In addition to the volatile essential oil fraction, cinnamon contains a diverse array of non-volatile polyphenolic compounds that are increasingly recognized as major contributors to its health-promoting properties [20]. The most important of these are proanthocyanidins (condensed tannins), which are oligomeric or polymeric flavonoids and composed of catechin and epicatechin subunits [21]. C. verum is particularly rich in B-type proanthocyanidins, whereas C. cassia predominantly contains A-type proanthocyanidins with characteristic double interflavan bonds. These proanthocyanidins have been shown to possess potent antioxidant activity, insulin-mimetic properties, and the ability to inhibit the formation of advanced glycation end-products (AGEs) [22].

3.4. Species-specific differences in phytochemical profiles

Significant variations in phytochemical composition exist among commercially important Cinnamomum species, reflecting differences in genetics, growing conditions, harvest timing, and processing methods [23]. These species-specific differences have important implications for both therapeutic efficacy and safety. *C. verum* (Ceylon cinnamon) is characterized by a lower cinnamaldehyde content (49-65%), higher eugenol levels (4-15%), minimal coumarin content, and abundant B-type proanthocyanidins, making it the safest species for regular consumption [24]. *C. cassia* (Chinese cinnamon) contains the highest cinnamaldehyde concentration (70-95%), trace eugenol, the highest coumarin content, and A-type proanthocyanidins, which are associated with stronger insulin-potentiating activity but greater hepatotoxicity risk [25].

3.5. Pharmacological mechanisms

The pharmacological activities of cinnamon are mediated through a complex interplay of molecular targets and signaling pathways [26]. This section provides a detailed examination of the major pharmacological mechanisms, with particular emphasis on the antioxidant, anti-inflammatory, and antidiabetic pathways that have been extensively

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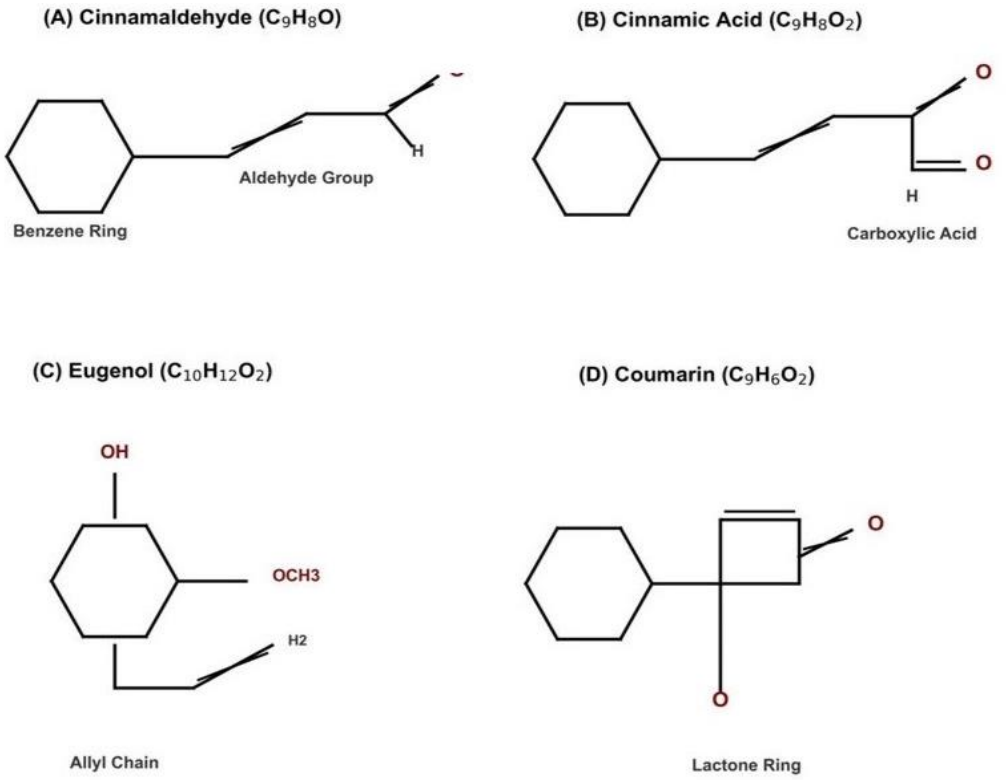

Figure 1. Chemical structures of major bioactive compounds in cinnamon: (A) cinnamaldehyde, (B) cinnamic acid, (C) eugenol, and (D) coumarin.

studied and are most relevant to clinical translation.

3.6. Antioxidant mechanisms and the nrf2/keap1/are pathway

Oxidative stress, resulting from an imbalance between the production of reactive oxygen species (ROS) and the capacity of endogenous antioxidant defense systems, is a fundamental pathological mechanism underlying numerous chronic diseases, including diabetes, cardiovascular disorders, neurodegenerative conditions, and cancer [27]. Cinnamon and its bioactive constituents have demonstrated potent antioxidant activity through both direct free radical scavenging and indirect modulation of endogenous antioxidant defense pathways [28]. The direct antioxidant effects are primarily attributable to polyphenolic compounds, particularly proanthocyanidins and flavonoids, which can neutralize superoxide anions, hydroxyl radicals, peroxyl radicals, and peroxynitrite through hydrogen atom transfer and single electron transfer mechanisms [3].

The indirect antioxidant mechanism, which is considered more physiologically relevant, involves the activation of the Nrf2/Keap1/ARE signaling pathway [29] (Fig. 2). Under basal conditions, Nrf2 (nuclear factor erythroid 2-related factor 2) is sequestered in the cytoplasm by its negative regulator Keap1 (Kelch-like ECH-associated protein 1) and targeted for ubiquitin-mediated proteasomal degradation. Cinnamaldehyde and other electrophilic cinnamon compounds can modify critical cysteine residues (particularly Cys151, Cys273, and Cys288) on Keap1 through Michael addition reactions, causing a conformational change that disrupts the Keap1-Nrf2 interaction and allows Nrf2 to translocate to the nucleus [30]. Once in the nucleus, Nrf2 heterodimerizes with small Maf proteins and binds to antioxidant response elements (AREs) in the promoter regions of numerous cytoprotective genes, including heme oxygenase-1 (HO-1), NAD(P)H quinone oxidoreductase 1 (NQO1), glutathione S-transferases (GSTs), glutamate-cysteine ligase catalytic subunit (GCLC), and superoxide dismutase (SOD) [31].

Extensive experimental evidence supports the Nrf2-activating properties of cinnamon. In HepG2 hepatocellular carcinoma cells, cinnamaldehyde

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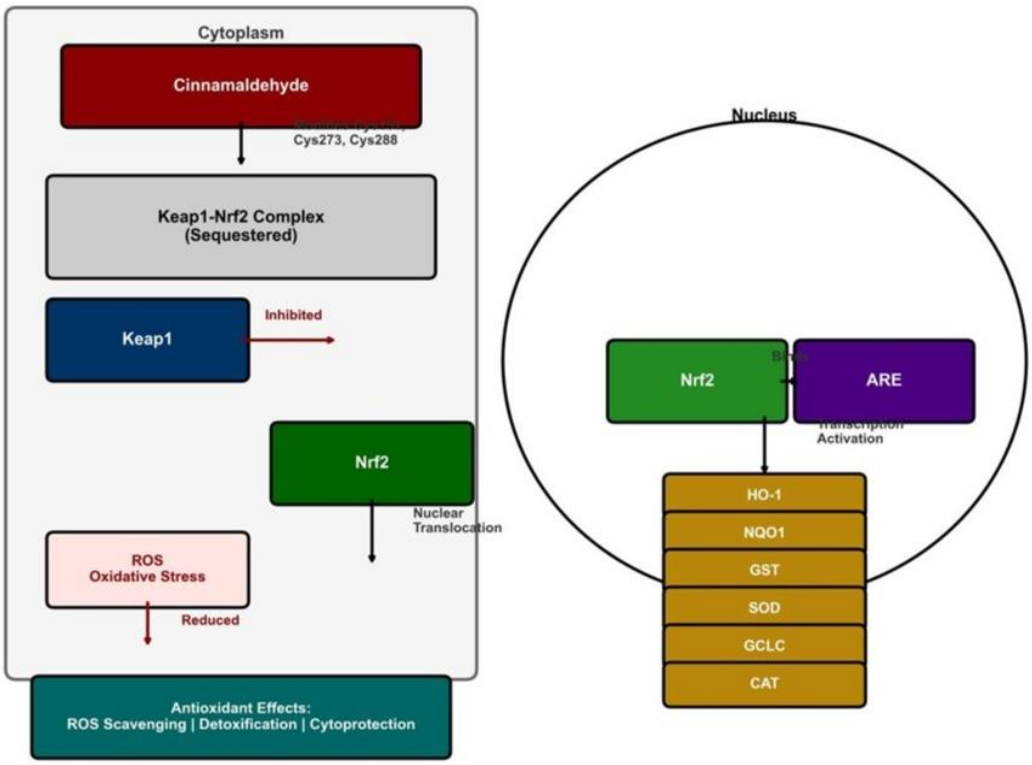

Figure 2. Schematic representation of the Nrf2/Keap1/ARE antioxidant signaling pathway activated by cinnamaldehyde and cinnamon polyphenols. Based on data from Zhang et al. [17] and Yu et al. [18].

treatment (20-80 microM) significantly increased nuclear Nrf2 accumulation, upregulated HO-1 and NQO1 expression, and enhanced cellular resistance to tert-butyl hydroperoxide-induced oxidative damage [32]. Similar findings have been reported in RAW264.7 macrophages, where cinnamaldehyde suppressed LPS-induced ROS production and nitric oxide (NO) generation through the Nrf2-dependent induction of antioxidant enzymes [33]. In vivo studies in streptozotocin-induced diabetic rats demonstrated that cinnamon bark extract (200-400 mg/kg/day) restored hepatic and renal glutathione levels, increased SOD and catalase activities, and reduced malondialdehyde (MDA) formation, effects that were associated with enhanced Nrf2 nuclear translocation and ARE-driven gene expression [34].

3.7. Anti-inflammatory mechanisms and nf-kappab/mapk signaling

Chronic inflammation is a central pathological feature of numerous diseases, including metabolic syndromes, atherosclerosis, rheumatoid arthritis, inflammatory bowel diseases, and neurodegenerative disorders [35]. Cinnamon has demonstrated significant anti-inflammatory activity across a wide range of experimental models, targeting multiple steps in the inflammatory (Q1) Qacascade [36] (Fig. 3). The primary molecular targets include the NF-kappaB (nuclear factor kappa-light-chain-enhancer of activated B cells) pathway and the MAPK (mitogen-activated protein kinase) signaling cascades, which serve as master regulators of pro-inflammatory gene expression.

The NF-kappaB pathway is activated by diverse stimuli, including pathogen-associated molecular patterns (PAMPs), damage-associated molecular patterns (DAMPs), and pro-inflammatory cytokines. In resting cells, NF-kappaB dimers (typically p50/p65 heterodimers) are retained in the cytoplasm by inhibitory IkappaB proteins [37]. Upon activation, the IkappaB kinase (IKK) complex phosphorylates IkappaBalpha, targeting it for ubiquitin-dependent proteasomal degradation and liberating NF-kappaB for nuclear translocation. In the nucleus, NF-kappaB activates the 1` transcription of hundreds of pro-

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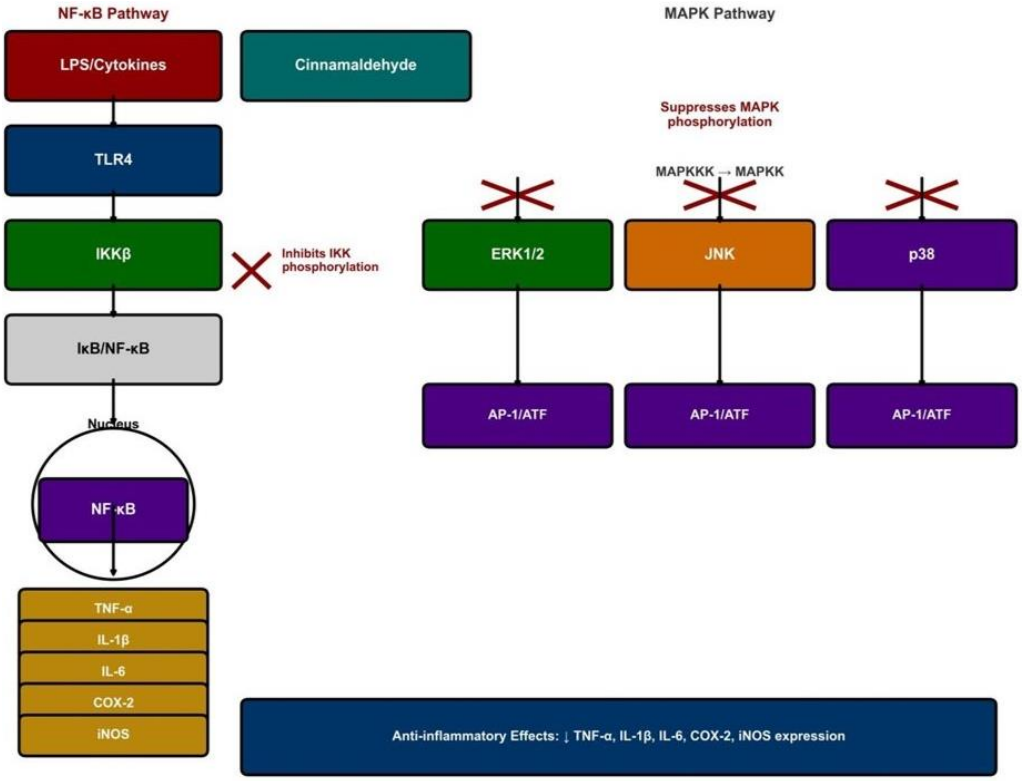

Figure 3. Overview of the NF-kappaB and MAPK inflammatory signaling pathways and their modulation by cinnamon bioactive compounds. Based on data from Kwon et al. [20], Liao et al. [21], and Tung et al. [22].

inflammatory genes, including tumor necrosis factor-alpha (TNF-alpha), interleukin-1beta (IL-1beta), interleukin-6 (IL-6), cyclooxygenase-2 (COX-2), and inducible nitric oxide synthase (iNOS) [38]. Cinnamaldehyde has been shown to inhibit NF-kappaB activation through multiple mechanisms: direct modification of the p65 subunit via Michael addition to critical cysteine residues (Cys38), inhibition of IKKbeta phosphorylation, and prevention of IkappaBalpha degradation [39].

The MAPK signaling pathways, comprising extracellular signal-regulated kinase (ERK), c-Jun N-terminal kinase (JNK), and p38 MAPK, represent parallel inflammatory signaling cascades that converge on the activation of the transcription factors AP-1 and CREB [40]. Cinnamon extracts and cinnamaldehyde have been shown to suppress LPS-induced phosphorylation of ERK1/2, JNK, and p38 in macrophages and other inflammatory cell types, thereby reducing the production of pro-inflammatory mediators [41]. The inhibition of MAPK signaling by cinnamon compounds is mediated through both direct antioxidant effects (reduction of upstream ROS signaling) and modulation of the upstream kinases. Notably, the dual targeting of NF-kappaB and MAPK pathways by cinnamon compounds produces synergistic anti-inflammatory effects that are more potent than the inhibition of either pathway alone [42].

3.8. Antidiabetic mechanisms and AMPK/metabolic regulation

Type 2 diabetes mellitus (T2DM) is a complex metabolic disorder characterized by chronic hyperglycemia resulting from insulin resistance, progressive beta-cell dysfunction, and inadequate insulin secretion [43]. Cinnamon has attracted particular attention for its potential antidiabetic properties, with a growing body of preclinical evidence demonstrating its effects on multiple aspects of glucose homeostasis [44] (Fig. 4). The antidiabetic mechanisms of cinnamon involve the modulation of insulin signaling, glucose uptake and metabolism, pancreatic beta-cell protection, and AMPK-mediated metabolic regulation.

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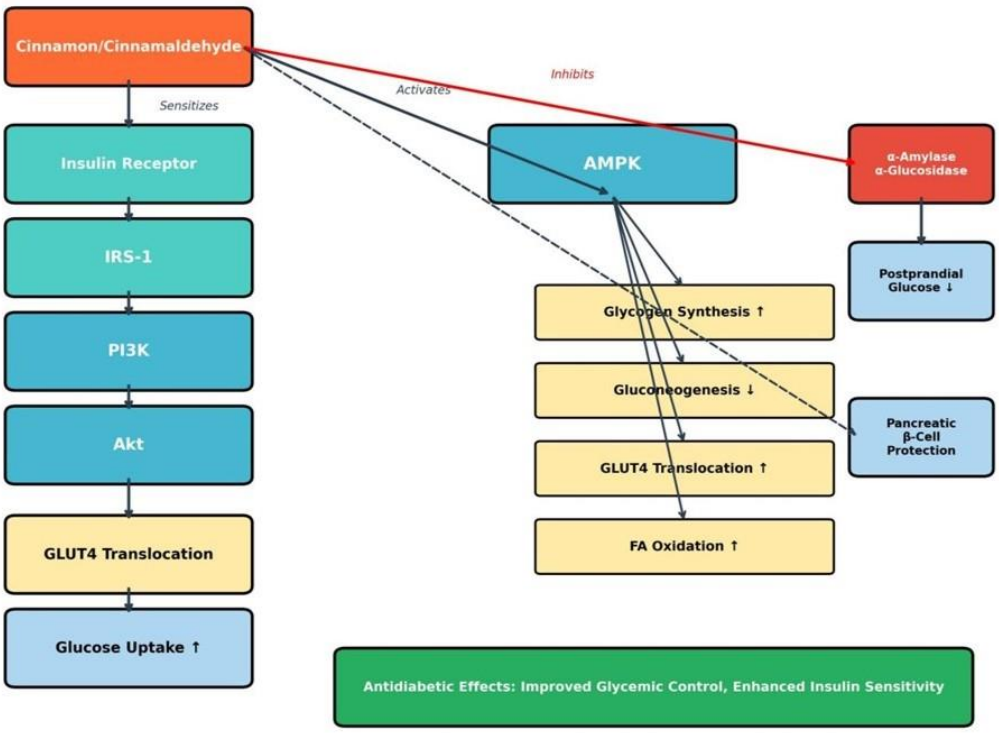

Figure 4. AMPK-mediated antidiabetic mechanisms of cinnamon bioactive compounds. Based on data from Sartorius et al. [26], Qin et al. [27], and Cao et al. [28].

One of the most extensively studied mechanisms is the insulin-sensitizing effect of cinnamon polyphenols, particularly the double-linked A-type proanthocyanidins found in C. cassia [45]. These compounds have been shown to enhance insulin receptor tyrosine kinase activity, increase insulin receptor substrate-1 (IRS-1) phosphorylation, and activate downstream phosphatidylinositol 3-kinase (PI3K)/Akt signaling, leading to increased translocation of glucose transporter 4 (GLUT4) to the plasma membrane and enhanced glucose uptake in skeletal muscle and adipose tissue [46]. In 3T3-L1 adipocytes, cinnamon polyphenol extract (0.01-0.1 mg/mL) significantly increased insulin-stimulated glucose uptake by 20-60%, effects comparable to those of the thiazolidinedione class of insulin sensitizers [47].

The AMP-activated protein kinase (AMPK) pathway has emerged as a central mediator of the metabolic effects of cinnamon [48]. AMPK serves as a cellular energy sensor that is activated under conditions of energy depletion (increased AMP:ATP ratio) and coordinates metabolic reprogramming to restore energy homeostasis. Cinnamon compounds, particularly cinnamaldehyde and cinnamic acid derivatives, activate AMPK through both AMP-dependent and AMP-independent mechanisms [49]. Activation of AMPK by cinnamon leads to enhanced fatty acid oxidation, inhibition of hepatic gluconeogenesis, stimulation of glucose uptake in skeletal muscle, and improvement of mitochondrial biogenesis and function [50].

3.9. Clinical translation

The translation of preclinical findings on the pharmacological activities of cinnamon into clinical evidence has been an active area of research, particularly in the fields of metabolic diseases and cardiovascular risk reduction [51]. This section reviews the current state of clinical evidence, emerging drug delivery strategies, and safety considerations relevant to the clinical development of cinnamon-based therapeutics.

3.10. Glycemic control in type 2 diabetes

The largest body of clinical evidence for cinnamon concerns its effects on glycemic control in patients with type 2 diabetes mellitus. Since the seminal study by Khan et al. in 2003 [52], which reported dramatic reductions in fasting blood glucose (18-29%), serum triglycerides (23-30%), LDL cholesterol (7-27%), and total cholesterol (12-26%) in T2DM patients

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consuming 1-6 g/day of cinnamon, numerous clinical trials and meta-analyses have been conducted with variable results. An early randomized controlled trial (RCT) by Akilen et al. [53] demonstrated a significant reduction in HbA1c of 0.36% and a decrease in systolic blood pressure of 3.8 mmHg after 12 weeks of supplementation with 2 g/day of cinnamon in 58 patients with type 2 diabetes mellitus (T2DM). Recently, Shishehbor et al. [54] reported that cinnamon supplementation significantly lowered fasting blood glucose and improved lipid profiles in patients with T2DM. Khosravi et al. [55] further showed that cinnamon improved glycemic control and antioxidant status. These consistent glycemic benefits corroborated by a 2024 systematic review and meta-analysis by Li et al. [56], confirmed that cinnamon significantly reduces fasting blood glucose and HbA1c across multiple RCTs. Collectively, recent evidence reinforces and extends the findings of Akilen et al. supporting the potential of cinnamon as a complementary intervention for T2DM.

Meta-analyses have attempted to reconcile these discrepancies. Allen et al. analyzed 10 RCTs with 543 T2DM patients and found a modest but statistically significant HbA1c reduction of 0.09%, noting substantial heterogeneity across studies [57]. Leach and Kumar [58], in a Cochrane review of 8 RCTs involving 435 patients, reported a more robust fasting blood glucose reduction of 24.59 mg/dL, with greater effects observed in studies using C. cassia preparations. The most comprehensive review by Sharma et al. encompassing 18 studies with over 1,100 participants, concluded that cinnamon supplementation produces dose-dependent improvements in glycemic control, with optimal effects observed at doses of 1.5-3 g/day for durations of 8-16 weeks [59].

3.11. Safety and toxicological considerations

While cinnamon is generally recognized as safe (GRAS) for culinary use, safety concerns arise at higher doses required for therapeutic effects [60]. The primary safety issue is the coumarin content, particularly in C. cassia preparations. Coumarin is hepatotoxic in susceptible individuals, causing reversible elevation of liver transaminases at daily doses exceeding 0.1 mg/kg body weight. It has been classified as a possible human carcinogen (Group 2B) by the International Agency for Research on Cancer (IARC) [61]. The European Food Safety Authority (EFSA) has established a tolerable daily intake (TDI) of 0.1 mg coumarin/kg body weight, which can be exceeded by consuming as little as 1-2 teaspoons of C. cassia powder daily [62-63]. C. verum, with its naturally low coumarin content, is strongly preferred for therapeutic applications requiring sustained supplementation.

Clinical studies have largely corroborated these toxicological concerns and informed subsequent risk–benefit assessments. In a randomized controlled trial involving patients with type 2 diabetes, Romeo et al. [64] reported that cinnamon supplementation at 1 g/day for 12 weeks was well tolerated, with no serious adverse events and no clinically meaningful changes in liver transaminases. Similarly, Hajimonfarednejad et al. observed stable hepatic function and an absence of significant adverse effects during a 12-week trial using a cinnamon extract [65]. These findings were further reinforced by a 2024 systematic review and meta-analysis of RCTs conducted by Li et al. [56], which confirmed that adverse events associated with cinnamon were predominantly mild and occurred at rates comparable to placebo, with no detectable signal of hepatotoxicity when trial-grade products were administered. Nevertheless, it should be noted that these studies predominantly utilized preparations with controlled or undisclosed coumarin levels, underscoring the critical importance of product standardization. Consequently, the translational imperative moving forward is the exclusive use of low-coumarin cinnamon species or coumarin-depleted extracts in future clinical trials, thereby maximizing safety without compromising the therapeutic efficacy.

3.12. Future perspectives

The translational journey of cinnamon from a traditional remedy to an evidence-based therapeutic agent has reached a pivotal stage. Its multi-target mechanisms, including activation of the Nrf2 antioxidant pathway, suppression of NF-κB/MAPK inflammatory signalling, and modulation of AMPK-dependent metabolism, provide a compelling rationale for its glucose-lowering and anti-

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inflammatory effects. However, clinical adoption is still hindered by inadequate standardisation, poor bioavailability of key constituents such as procyanidins, and substantial inter-individual variability. Moving forward, low-coumarin, marker-quantified preparations must become mandatory, and advanced delivery systems, such as nano-encapsulation, should be employed to overcome absorption barriers. Given that gut microbiota composition influences the bioconversion of cinnamon polyphenols, integrating metagenomic stratification into future trials will help identify likely responders and enable a personalised approach. The evidence base also requires strengthening through large-scale, long-duration randomised controlled trials employing hard clinical endpoints, such as diabetic complications or cardiovascular events—and incorporating health-economic analyses to establish real-world value. Beyond glycaemic control, cinnamon's unique polypharmacology, which simultaneously targets oxidative stress, inflammation, and metabolic dysregulation, aligns with network pharmacology concepts and supports its investigation as a multi-risk-factor modifier in conditions like metabolic syndrome and non-alcoholic fatty liver disease. In summary, only by addressing these translational bottlenecks through rigorous standardisation, innovative formulation, microbiome-informed stratification, and clinically meaningful trial designs can cinnamon evolve from a controversial supplement into a scientifically credible, regulation-ready adjunctive therapy for metabolic and inflammatory diseases.

4. Conclusions

Cinnamon represents a promising natural therapeutic agent with a complex phytochemical profile and diverse pharmacological activities that target multiple disease-relevant pathways. The preclinical evidence for the antioxidant, anti-inflammatory, antidiabetic, and cardioprotective effects of cinnamon is substantial and well-supported by mechanistic studies demonstrating the modulation of Nrf2/Keap1/ARE, NF-kappaB/MAPK, and AMPK signaling pathways. However, clinical translation remains challenging due to issues of product standardization, bioavailability limitations, and inter-individual variability in response. Future research should focus on standardized cinnamon preparations with defined marker compound content, optimized delivery systems to enhance bioavailability, and well-designed clinical trials with appropriate power and duration to establish evidence-based therapeutic protocols. With continued scientific investigation and rigorous clinical evaluation, cinnamon may fulfill its potential as a valuable adjunctive therapy for metabolic and inflammatory diseases.

Disclaimer (artificial intelligence)

Author(s) hereby state that no generative AI tools such as Large Language Models (ChatGPT, Copilot, etc.) and text-to-image generators were utilized in the preparation or editing of this manuscript.

Authors' contributions

Conceptualization, Z.J.L., J.P.D., J.W.S; methodology, Z.J.L., J.P.D, M.Q.L.; software, Z.J.L., J.P.D., J.W.S; formal analysis, S.M.F.; investigation, J.P.D, M.Q.L., resources, J.P.D, M.Q.L., J.W.S; data curation, J.P.D, M.Q.L., J.W.S; writing–original draft and preparation, J.P.D, M.Q.L.; visualization: Z.J.L., J.P.D, M.Q.L.; supervision, Z.J.L., J.P.D, M.Q.L.; project administration, Z.J.L., J.P.D, M.Q.L.; funding acquisition: Z.J.L., J.P.D, M.Q.L.

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Availability of data and materials

All relevant data are within the paper and its supporting information files. Additional data will be made available on request according to the journal policy.

Conflicts of interest

The authors declare no conflict of interest.

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