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CINNAMON AGAINST OBESITY – A SYSTEMATIC REVIEW

Mikołaj Zbrożek *(Corresponding Author, Email: mikolaj.zbrozek@gmail.com)*
Prof. W. Orłowski Independent Public Clinical Hospital, Warsaw, Poland
ORCID ID: 0009-0007-8873-6184

Hanna Maruchniak
Czerniakowski Hospital, Warsaw, Poland
ORCID ID: 0009-0003-8735-9912

Wiktoria Marzec
Specialist Hospital named after Florian Ceynowy in Wejherowo, Wejherowo, Poland
ORCID ID: 0009-0006-6395-6263

Paulina Biedroń
Praski Hospital of the Transfiguration of the Lord, Warsaw, Poland
ORCID ID: 0009-0008-8461-2958

Anna Skrzypek
Czerniakowski Hospital, Warsaw, Poland
ORCID ID: 0009-0007-6771-3186

Maciej Hutkowski
Independent Public Healthcare Complex in Pruszków, Poland
ORCID ID: 0009-0006-9811-8681

Zuzanna Chwostek
Medical Center, Łańcut, Poland
ORCID ID: 0009-0001-8177-4168

Bartłomiej Kosiarski
Central Clinical Hospital, Medical University, Warsaw, Poland
ORCID ID: 0009-0005-2499-1245

Patrycja Markowicz
Praski Hospital of the Transfiguration of the Lord, Warsaw, Poland
ORCID ID: 0009-0001-3986-1751

Krzysztof Biłyk
Independent Public Specialist Western Hospital named after St. John Paul II, Poland
ORCID ID: 0009-0006-2374-4231

ABSTRACT

Introduction: Obesity is a major global health problem associated with metabolic disturbances, including insulin resistance, dyslipidemia, and chronic inflammation. Due to various limitations of pharmacological therapies, increasing attention has been directed toward natural compounds with potential metabolic benefits. This review aimed to evaluate the role of cinnamon and its bioactive constituents in obesity management.
Methodology: A comprehensive literature search was conducted using PubMed, Scopus, Web of Science, and Google Scholar. Studies investigating cinnamon and its bioactive compounds in relation to obesity, body weight, and metabolic parameters were included. Both experimental and clinical studies were analyzed according to predefined inclusion and exclusion criteria.
Results: Cinnamon contains bioactive compounds, particularly cinnamaldehyde and polyphenols, that influence key metabolic pathways. Experimental studies demonstrate inhibition of adipogenesis, stimulation of thermogenesis, and improvement of insulin sensitivity. Clinical studies suggest modest reductions in body weight and selected metabolic parameters, although results remain heterogeneous.
Conclusion: Cinnamon appears to be a promising supportive strategy in obesity and metabolic syndrome management; however, its clinical effectiveness remains limited. Further well-designed studies are required to determine optimal dosing, safety, and long-term efficacy.

KEYWORDS

Cinnamon Supplementation; Obesity Management; Cinnamaldehyde; Polyphenols; Metabolic Syndrome; Insulin Resistance

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Introduction

Obesity has emerged as a global epidemic and represents one of the most significant public health challenges of the 21st century. According to the World Health Organization, the prevalence of obesity has nearly tripled since 1975, with over 650 million adults classified as obese worldwide, and projections suggesting that more than one billion individuals may be affected by 2030. Obesity is defined as excessive accumulation of adipose tissue that impairs health and is commonly assessed using body mass index (BMI), with values ≥30 kg/m² indicating obesity. It is strongly associated with increased morbidity and mortality, particularly due to its role in the development of type 2 diabetes mellitus, cardiovascular diseases, and certain malignancies [1-4]. The pathophysiology of obesity is complex and multifactorial, involving an imbalance between caloric intake and energy expenditure, as well as genetic, environmental, and behavioral determinants. Importantly, obesity is now recognized as a chronic inflammatory and metabolic disorder rather than solely a condition of excess fat accumulation. Adipose tissue functions as an endocrine organ that secretes adipokines such as leptin and adiponectin, as well as pro-inflammatory cytokines including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which contribute to insulin resistance, dyslipidemia, and systemic inflammation [4-7]. In response to the increasing burden of obesity, pharmacological interventions have gained substantial importance, particularly in individuals who do not respond adequately to lifestyle modifications. Among the most notable recent advances are incretin-based therapies, including glucagon-like peptide-1 receptor agonists (GLP-1 RAs) such as liraglutide and semaglutide, which promote weight loss by reducing appetite, delaying gastric emptying, and improving glycemic control. More recently, dual agonists such as tirzepatide, targeting both GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) receptors, have demonstrated superior efficacy in reducing body weight and improving metabolic outcomes in clinical trials [8-11]. Despite their effectiveness, these pharmacological treatments are associated with several limitations. The most commonly reported adverse effects include gastrointestinal symptoms such as nausea, vomiting,

diarrhea, and constipation, which may lead to treatment discontinuation. Additionally, concerns have been raised regarding the risk of pancreatitis, gallbladder disease, and potential long-term safety issues. The high cost of these therapies and the need for long-term administration further limit their accessibility, particularly in low- and middle-income countries [8,10,12-14]. Consequently, increasing attention has been directed toward dietary interventions and natural bioactive compounds with potential anti-obesity effects. Among these, polyphenols have been extensively studied due to their ability to modulate key metabolic pathways. Experimental and clinical studies have demonstrated that polyphenols may reduce food intake, inhibit adipogenesis, enhance fatty acid oxidation, decrease lipogenesis, and attenuate inflammation and oxidative stress [4,15-17]. Cinnamon, a widely consumed spice obtained from the bark of Cinnamomum species, has attracted significant scientific interest due to its diverse biological properties. It contains a complex mixture of bioactive compounds, including cinnamaldehyde, cinnamic acid, eugenol, and various polyphenols, which have been shown to exert antioxidant, anti-inflammatory, and metabolic regulatory effects. Studies suggest that cinnamon may improve insulin sensitivity, enhance glucose uptake, modulate lipid metabolism, and influence adipocyte differentiation, thereby contributing to weight regulation and metabolic health [17-21]. However, despite the growing body of evidence, the effects of cinnamon on obesity remain inconsistent across studies. Variability in cinnamon species, preparation methods, dosage, and duration of supplementation contributes to heterogeneous outcomes. Moreover, safety considerations, particularly related to the presence of coumarin in cassia cinnamon, must be taken into account when evaluating its clinical applicability. Coumarin intake exceeding the tolerable daily intake of 0.1 mg/kg body weight may lead to hepatotoxic effects [22-24]. Therefore, the aim of this systematic review is to comprehensively evaluate the current evidence on cinnamon and its bioactive compounds in the context of obesity, with particular emphasis on its botanical sources, chemical composition, molecular mechanisms of action, and findings from experimental and clinical studies.

Methodology

This systematic review was conducted in accordance with established methodological principles for evidence synthesis and was designed to identify, evaluate, and summarize current evidence on the effects of cinnamon and its bioactive compounds on obesity and metabolic regulation. A comprehensive literature search was performed using major scientific databases, including PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar. The search strategy included combinations of keywords and Medical Subject Headings (MeSH) terms related to cinnamon and obesity, such as “cinnamon”, “cinnamaldehyde”, “polyphenols”, “obesity”, “body weight”, “adipogenesis”, “lipid metabolism”, “insulin resistance”, and “metabolic syndrome”. Boolean operators (AND, OR) were used to refine the search and maximize retrieval of relevant studies. In addition, reference lists of selected articles and relevant review papers were manually screened to identify further eligible studies. The inclusion criteria were defined prior to the literature search. Studies were included if they met the following criteria: (i) original research articles or systematic reviews published in peer-reviewed journals; (ii) studies investigating cinnamon or its bioactive compounds (e.g., cinnamaldehyde, polyphenols) in relation to obesity, body weight, adiposity, or metabolic parameters; (iii) studies conducted on in vitro models, animal models, or human subjects; and (iv) articles published in English. Both experimental and clinical studies were considered to provide a comprehensive overview of available evidence. Exclusion criteria included: (i) studies not related to obesity or metabolic outcomes; (ii) articles lacking full-text availability; (iii) conference abstracts, editorials, or non-peer-reviewed publications; and (iv) studies focusing exclusively on unrelated pharmacological effects of cinnamon without metabolic relevance. Duplicate records identified across databases were removed prior to screening. The study selection process consisted of two stages. First, titles and abstracts were screened for relevance. Subsequently, full-text articles were assessed for eligibility based on predefined inclusion and exclusion criteria. Studies that met all criteria were included in the qualitative synthesis. Particular emphasis was placed on studies providing mechanistic insights, as well as randomized controlled trials and meta-analyses. The methodology of this review was aligned with established guidelines for systematic reviews and evidence synthesis [25,26].

Results

SOURCES OF CINNAMON

Cinnamon is a spice obtained from the dried inner bark of evergreen trees belonging to the genus *Cinnamomum* (family Lauraceae). Although the term "cinnamon" is commonly used as if it referred to a single botanical product, in practice it includes bark derived from several species that differ in geographical origin, morphology, chemical composition, and safety profile. The main commercially relevant species are *Cinnamomum verum* (syn. *C. zeylanicum*), *Cinnamomum cassia* (also referred to as *C. aromaticum* in some sources), *Cinnamomum burmannii*, and *Cinnamomum loureiroi* [18,20,23,27]. The spice is produced from the inner bark rather than from the whole stem or outer bark. After harvesting, young stems are cut, the outer corky layer is removed, and the softened inner bark is loosened, detached, and dried. During drying, the bark curls naturally into quills, which may then be sold as sticks or ground into powder. In the case of Ceylon cinnamon, several thin pieces of inner bark are often placed one inside another to form compound quills, which are regarded as a characteristic feature of high-quality commercial material. This processing method is labor-intensive and strongly influences the final quality of the spice [27-29]. From a geographical perspective, *C. verum* is traditionally associated with Sri Lanka and southern India and is commonly marketed as "Ceylon cinnamon" or "true cinnamon." By contrast, *C. cassia* is mainly associated with China, *C. burmannii* with Indonesia, and *C. loureiroi* with Vietnam. These species dominate different segments of the global market, with cassia-type cinnamons generally being cheaper and more widely available than *C. verum*. As a consequence, many retail cinnamon products, especially powdered cinnamon, consist predominantly of cassia-type material rather than true cinnamon [18,22,23,27]. The botanical source has a substantial impact on the physical characteristics of the spice. *C. verum* produces thin, fragile, multilayered quills with a light brown color and delicate texture, whereas cassia-type cinnamons are usually thicker, harder, darker, and composed of a single rolled bark layer. These morphological differences are not only relevant for sensory quality, but also help distinguish species in the market. Ceylon cinnamon is generally described as having a milder, sweeter, and more complex flavor, while cassia-type cinnamons are typically more pungent, hotter, and more astringent due to their volatile composition [18,20,21,27,29]. The most important differences between cinnamon species concern their chemical composition. Cinnamon bark contains volatile compounds, phenolic compounds, and other secondary metabolites, but their proportions vary significantly by species. Across most commercial species, cinnamaldehyde is the dominant volatile constituent of the bark and is largely responsible for the characteristic aroma and many of the biological effects attributed to cinnamon. However, *C. verum* tends to contain relatively higher amounts of eugenol and a lower amount of coumarin, whereas cassia-type cinnamons, especially *C. cassia*, *C. burmannii*, and *C. loureiroi*, are characterized by substantially higher coumarin concentrations [17,18,20,23]. Besides cinnamaldehyde, cinnamon bark may contain cinnamic acid, cinnamyl acetate, polyphenols such as procyanidins, and smaller amounts of terpenes and aromatic compounds including camphor, linalool, and caryophyllene derivatives, depending on the species and analytical method used. Available compositional data indicate that *C. loureiroi* and *C. cassia* are often particularly rich in cinnamaldehyde and volatile oil content, which contributes to their strong flavor intensity, whereas *C. verum* is generally considered chemically milder but safer for long-term consumption due to its much lower coumarin content [17,18,20,23]. This distinction is especially important from a nutritional and toxicological perspective. Coumarin is a naturally occurring compound that can exert hepatotoxic effects when consumed chronically in excessive amounts. For this reason, the botanical authenticity of cinnamon used in food products and supplementation is highly relevant. *C. verum* is generally regarded as the preferred source when repeated or long-term intake is intended, while cassia-type cinnamons may pose a greater safety concern because of their naturally higher coumarin levels [22-24]. Therefore, any interpretation of the biological effects of cinnamon in obesity research should take into account not only the administered dose, but also the botanical species from which the spice or extract was obtained. From the perspective of experimental and clinical research, the distinction between species is crucial because differences in chemical composition may influence both efficacy and safety. Studies that do not clearly identify the botanical source of cinnamon are difficult to compare and may contribute to inconsistent findings. Consequently, accurate characterization of the cinnamon species should be regarded as an essential methodological element in studies investigating the anti-obesity and metabolic effects of cinnamon [17,18,20,23]. These interspecies differences have important implications for both efficacy and safety, and are summarized in Table 1. to facilitate comparison across commonly used cinnamon types.

Table 1. Key interspecies differences in cinnamon relevant to metabolic research [26,30,31]

Species	Compositional tendency	Research relevance	Safety implication
Cinnamomum verum	Balanced profile (cinnamaldehyde, eugenol, polyphenols); very low coumarin	Suitable for long-term nutritional and clinical studies	Low hepatotoxic risk
Cinnamomum cassia	High cinnamaldehyde; low eugenol	Common in mechanistic and metabolic studies	High coumarin content → hepatotoxic risk
Cinnamomum burmannii	Cinnamaldehyde-rich; lower polyphenol diversity	Relevant for population-level dietary exposure	High coumarin content
Cinnamomum loureiroi	Very high cinnamaldehyde; variable chemotypes	Emerging relevance in thermogenesis research	Variable coumarin content

CHEMICAL COMPOSITION

The chemical composition of cinnamon bark is complex and includes a wide range of volatile and non-volatile compounds that contribute to its sensory properties and biological activity. The major groups of constituents include essential oils, polyphenolic compounds, phenolic acids, and coumarins, with their relative abundance depending strongly on the botanical species, geographical origin, and processing methods [17,18,20,23]. The essential oil fraction represents the most characteristic component of cinnamon and is dominated by cinnamaldehyde, which is responsible for the distinctive aroma and many of the pharmacological effects of the spice. In most species, cinnamaldehyde constitutes between 60% and 80% of the volatile oil, although this proportion varies depending on species and extraction technique. Other important volatile constituents include cinnamyl acetate, eugenol, linalool, β-caryophyllene, and camphor. Notably, eugenol is present in higher concentrations in *Cinnamomum verum*, contributing to its milder flavor profile, whereas cassia-type cinnamons are characterized by a higher cinnamaldehyde content and a more intense, pungent aroma [17,18,20,23]. In addition to volatile compounds, cinnamon is rich in polyphenols, which play a central role in its metabolic and antioxidant properties. Among these, proanthocyanidins, particularly type-A polymers, are considered key bioactive constituents. These compounds have been shown to exert insulin-mimetic effects, enhance glucose uptake, and modulate signaling pathways related to lipid metabolism. Other polyphenolic compounds identified in cinnamon include catechins, epicatechins, and various phenolic acids such as gallic acid, caffeic acid, and ferulic acid. These compounds contribute to the antioxidant capacity of cinnamon by scavenging reactive oxygen species and reducing oxidative stress, which is closely linked to obesity-related metabolic dysfunction [17-19]. Phenolic acids, including cinnamic acid and its derivatives, represent another important group of compounds. These substances are involved in the phenylpropanoid pathway and contribute to both antioxidant and metabolic regulatory effects. Although present in smaller amounts compared to cinnamaldehyde and polyphenols, they may act synergistically with other constituents to enhance the overall biological activity of cinnamon [19,20]. A critical compositional difference between cinnamon species concerns the presence of coumarin. Cassia-type cinnamons (*C. cassia*, *C. burmannii*, *C. loureiroi*) contain significantly higher levels of coumarin compared to *C. verum*, in which this compound is present only in trace amounts. This difference has important toxicological implications, as coumarin has been associated with hepatotoxic effects when consumed in excessive amounts over prolonged periods. Regulatory authorities have therefore established a tolerable daily intake of 0.1 mg/kg body weight, highlighting the need to consider both dosage and species when evaluating cinnamon as a dietary supplement [22-24]. The overall composition of cinnamon is influenced by multiple factors, including plant species, geographical origin, climate conditions, harvesting time, and processing techniques. For example, variations in maturity stage of plant material have been shown to affect antioxidant capacity and phenolic content, with more mature plant tissues generally exhibiting higher levels of bioactive compounds. These variations may contribute to inconsistencies observed across experimental and clinical studies and should be taken into account when interpreting results related to the anti-obesity effects of cinnamon [17,18,32]. Importantly, the biological activity of cinnamon cannot be attributed to a single compound, but rather to the synergistic interaction of its constituents. The combination of cinnamaldehyde, polyphenols, and other minor compounds enables cinnamon to influence multiple metabolic pathways simultaneously, including glucose metabolism, lipid homeostasis, inflammation, and oxidative stress. This multi-target mechanism is particularly relevant in the context of obesity, which is itself a multifactorial metabolic disorder [18,19].

MOLECULAR MECHANISMS

The anti-obesity effects of cinnamon are mediated through a complex interplay of bioactive compounds, including cinnamaldehyde, polyphenols, cinnamic acid, and other minor constituents such as eugenol and cinnamyl acetate, which act on multiple metabolic pathways simultaneously. These compounds influence key processes involved in obesity, including glucose metabolism, lipid homeostasis, adipocyte differentiation, thermogenesis, inflammation, and oxidative stress [7,17,33]. Cinnamaldehyde, the principal bioactive compound of cinnamon, plays a central role in metabolic regulation by modulating both energy balance and adipose tissue function. It has been shown to suppress adipogenesis through downregulation of transcription factors such as peroxisome proliferator-activated receptor gamma (PPARγ) and CCAAT/enhancer-binding protein alpha (C/EBPα), which are essential for adipocyte differentiation. Additionally, cinnamaldehyde inhibits lipogenesis by reducing the expression of sterol regulatory element-binding protein-1c (SREBP-1c) and associated enzymes, including acetyl-CoA carboxylase, while simultaneously promoting fatty acid β-oxidation. These effects collectively lead to reduced lipid accumulation and decreased adipocyte hypertrophy [17,34-36]. Furthermore, cinnamaldehyde has been shown to activate AMP-activated protein kinase (AMPK), a key regulator of cellular energy homeostasis. Activation of AMPK enhances glucose uptake, stimulates fatty acid oxidation, and inhibits anabolic processes such as lipid and cholesterol synthesis. In addition, cinnamaldehyde acts as an agonist of transient receptor potential ankyrin 1 (TRPA1), which has been implicated in appetite regulation and energy expenditure. Activation of TRPA1 in gastrointestinal tissues may contribute to reduced food intake and delayed gastric emptying, further supporting its anti-obesity potential [7,17,36]. Polyphenolic compounds present in cinnamon, particularly proanthocyanidins, exert complementary metabolic effects. These compounds have been shown to enhance insulin signaling by increasing insulin receptor phosphorylation and promoting translocation of glucose transporter type 4 (GLUT4) to the cell membrane. As a result, glucose uptake is increased in insulin-sensitive tissues, leading to improved glycemic control. In addition, cinnamon polyphenols modulate gene expression related to glucose metabolism, including increased transcription of glucose transporter type 1 and decreased expression of glycogen synthase kinase 3 beta and phosphatidylinositol 3-kinase regulatory subunit alpha. These molecular changes contribute to improved insulin sensitivity and reduced hyperglycemia [17,19]. Polyphenols also play a significant role in regulating lipid metabolism and adipocyte function. They have been shown to inhibit adipocyte differentiation and lipid accumulation in cellular models, such as 3T3-L1 adipocytes, while simultaneously promoting lipolysis and fatty acid oxidation. In vivo studies confirm that cinnamon supplementation can reduce lipid accumulation in adipose tissue and liver, indicating a systemic effect on fat distribution and storage [33-35]. Another important mechanism involves the induction of thermogenesis and the browning of white adipose tissue. Cinnamaldehyde has been demonstrated to increase the expression of thermogenic genes, including uncoupling protein 1 (UCP1), leading to enhanced energy expenditure through heat production. This browning process shifts adipose tissue function from energy storage to energy dissipation, which may contribute to a negative energy balance and weight reduction. Experimental studies in animal models have shown that cinnamaldehyde improves both white and brown adipose tissue metabolism and promotes long-term metabolic benefits [36,37]. In addition to metabolic regulation, cinnamon exerts significant anti-inflammatory and antioxidant effects, which are critical in the context of obesity. Chronic low-grade inflammation is a hallmark of obesity and contributes to insulin resistance and metabolic dysfunction. Cinnamon compounds inhibit key inflammatory pathways, including nuclear factor kappa B (NF-κB), and reduce the production of pro-inflammatory cytokines such as tumor necrosis factor-alpha and interleukin-6. Simultaneously, their antioxidant properties reduce reactive oxygen species and oxidative damage, thereby improving cellular function and metabolic homeostasis [7,33]. Cinnamic acid and other minor phenolic compounds contribute to these effects through their antioxidant activity and supportive role in metabolic pathways. Although less extensively studied, they are believed to act synergistically with cinnamaldehyde and polyphenols, enhancing the overall biological activity of cinnamon. This synergistic interaction is particularly important given the multifactorial nature of obesity, which involves multiple dysregulated pathways rather than a single target [7,17]. Overall, the anti-obesity effects of cinnamon result from the coordinated modulation of multiple molecular pathways, including improved insulin signaling, activation of AMPK, inhibition of adipogenesis, stimulation of thermogenesis, and reduction of inflammation and oxidative stress. This multi-target mechanism highlights the potential of cinnamon as a complementary approach in obesity management, although variability in composition and bioavailability remains a challenge for its clinical application [7,17,36]. Due to the multi-target nature of cinnamon-derived compounds, a pathway-oriented synthesis of their metabolic effects is provided in Table 2.

Table 2. Integrated metabolic pathways targeted by cinnamon bioactive compounds [17,21,33,38]

Pathway	Molecular target	Effect	Metabolic implication
Insulin signaling	Insulin receptor / GLUT4	Upregulation	Improved glucose uptake
Energy sensing	AMPK	Activation	Increased energy expenditure
Adipogenesis	PPARγ / C/EBPα	Inhibition	Reduced adipocyte formation
Lipogenesis	SREBP-1c / ACC	Downregulation	Decreased lipid synthesis
Fat oxidation	β-oxidation pathways	Upregulation	Increased lipid utilization
Thermogenesis	UCP1	Activation	Browning of adipose tissue
Inflammation	NF-κB	Inhibition	Reduced pro-inflammatory cytokines
Oxidative stress	ROS pathways	Reduction	Improved cellular homeostasis

ANIMAL AND CLINICAL RESEARCH

Experimental studies (in vitro and animal models)

A substantial body of evidence supporting the anti-obesity effects of cinnamon originates from in vitro and animal studies, which provide mechanistic and physiological insights into its metabolic activity. In cellular models, particularly 3T3-L1 adipocytes, cinnamon extracts and their bioactive compounds have been shown to inhibit adipocyte differentiation, reduce lipid accumulation, and promote lipolysis. These effects are mediated through suppression of adipogenic gene expression, including PPARγ and C/EBPα, and modulation of metabolic pathways involved in lipid storage and utilization [7,33]. In addition to inhibiting adipogenesis, cinnamon has demonstrated the ability to influence whole-body metabolism in animal models of diet-induced obesity. For instance, studies conducted on zebrafish exposed to high-calorie diets have shown that cinnamon supplementation significantly reduces blood glucose levels, triglycerides, and body mass index (BMI), with treated animals exhibiting metabolic profiles comparable to non-obese controls. These findings suggest that cinnamon may effectively counteract the metabolic consequences of excessive caloric intake [35]. Similarly, in rodent models, cinnamon and its major bioactive component, cinnamaldehyde, have been shown to improve adipose tissue function and systemic metabolism. Supplementation in early-life obesity models resulted in reduced adipocyte hypertrophy, decreased visceral fat accumulation, and enhanced thermogenic activity in adipose tissue. Notably, these effects persisted even after cessation of treatment, indicating potential long-term metabolic reprogramming [36]. Another important mechanism observed in animal studies is the induction of adipose tissue browning. Cinnamaldehyde has been shown to increase the expression of thermogenic markers such as uncoupling protein 1 (UCP1), promoting the conversion of white adipose tissue into metabolically active brown-like tissue. This shift enhances energy expenditure through heat production and may contribute to the development of a negative energy balance, which is critical in obesity management [37]. Furthermore, cinnamon supplementation has been associated with improvements in hepatic metabolism. In high-fat diet-fed rats, cinnamon has been shown to reduce hepatic steatosis by enhancing fatty acid β-oxidation, inhibiting lipogenesis, and decreasing oxidative stress and inflammation. These effects are particularly relevant given the strong association between obesity and non-alcoholic fatty liver disease (NAFLD) [34]. Collectively, findings from experimental models indicate that cinnamon exerts multifaceted anti-obesity effects by modulating adipogenesis, lipid metabolism, thermogenesis, and inflammatory pathways. However, the translational relevance of these findings to human populations requires careful consideration.

Clinical studies (human evidence)

Clinical studies investigating the effects of cinnamon supplementation on obesity-related outcomes have produced promising but heterogeneous results. Several randomized controlled trials and meta-analyses suggest that cinnamon may contribute to modest reductions in body weight, body mass index (BMI), and fat mass, particularly when administered at doses exceeding 2–3 g per day and over longer durations (≥12 weeks) [39,40]. A meta-analysis of randomized controlled trials demonstrated that cinnamon supplementation significantly reduces body weight and BMI, with more pronounced effects observed in individuals under 50 years of age and those with baseline obesity (BMI ≥30 kg/m²). These findings suggest that cinnamon may be particularly effective in populations with existing metabolic disturbances [39]. In addition to anthropometric outcomes, cinnamon has been shown to influence hormonal regulators of energy balance. A systematic review of clinical trials reported that cinnamon supplementation may decrease circulating levels of leptin and visfatin while increasing ghrelin levels, although the results were not entirely consistent across studies. Interestingly, cinnamon did not significantly affect adiponectin levels in most cases, despite improvements in insulin sensitivity and glucose metabolism, suggesting that its metabolic effects may be mediated through adiponectin-

independent pathways [40]. Individual clinical trials further support these findings. For example, supplementation with approximately 3 g of cinnamon daily in individuals with metabolic syndrome resulted in significant reductions in body weight, total adiposity, and abdominal fat. These changes were accompanied by improvements in glycemic control and lipid profiles, indicating a broader metabolic benefit [41]. Moreover, recent studies suggest that cinnamon may enhance the effects of lifestyle interventions. In a randomized trial combining high-intensity interval training with cinnamon supplementation, participants receiving both interventions exhibited greater reductions in body fat percentage compared to those undergoing exercise alone. This indicates that cinnamon may act synergistically with physical activity in improving body composition [42]. Despite these promising findings, the clinical evidence remains inconsistent, largely due to variability in study design, cinnamon species, dosage, and duration of supplementation. Additionally, differences in baseline metabolic status among participants may influence the magnitude of observed effects. Therefore, while cinnamon shows potential as a supportive intervention in obesity management, further well-designed, large-scale clinical trials are needed to establish its efficacy and optimal use.

Limitations

Despite the growing body of evidence supporting the anti-obesity effects of cinnamon, several important limitations must be considered when interpreting the available data. One of the most significant challenges is the relatively limited number of high-quality clinical studies. Although numerous in vitro and animal studies have demonstrated promising metabolic effects, including reduced adipogenesis, improved insulin sensitivity, and enhanced lipid metabolism, the translation of these findings into human populations remains insufficiently validated. Many clinical trials are characterized by small sample sizes, short intervention durations, and heterogeneous study populations, which limits the generalizability of the results [7,39,40]. Furthermore, considerable variability exists in study design, including differences in cinnamon species, preparation methods, dosage, and duration of supplementation. In many cases, studies do not specify the botanical source of cinnamon, which is particularly problematic given the significant differences in chemical composition between *Cinnamomum verum* and *cassia*-type species. This lack of standardization complicates comparisons across studies and may contribute to inconsistent findings regarding efficacy and safety [7,23]. Another important limitation concerns the pharmacokinetics and bioavailability of cinnamon-derived compounds. The absorption, distribution, metabolism, and excretion of key bioactive components, particularly cinnamaldehyde and polyphenols, are not yet fully understood. Cinnamaldehyde is rapidly metabolized to cinnamic acid and subsequently to benzoate derivatives, which may alter its biological activity in vivo. Additionally, polyphenols often exhibit low bioavailability due to extensive metabolism in the gastrointestinal tract and liver, as well as interactions with gut microbiota. These factors may significantly influence the extent to which the observed in vitro effects can be replicated in human subjects [17,21]. The route of administration and formulation of cinnamon also represent critical factors affecting its efficacy. Most studies utilize powdered cinnamon or crude extracts, which may differ substantially in composition and concentration of active compounds. The lack of standardized preparations and controlled delivery systems further complicates the assessment of dose–response relationships and optimal therapeutic dosing [21]. Safety considerations also represent a significant limitation, particularly in relation to coumarin content. *Cassia*-type cinnamon, which is commonly available in the commercial market, contains relatively high levels of coumarin, a compound associated with hepatotoxicity when consumed in excessive amounts. Chronic intake exceeding the tolerable daily intake of 0.1 mg/kg body weight may pose a health risk, especially in individuals consuming cinnamon as a supplement over extended periods. The frequent lack of species identification in studies and commercial products increases the risk of unintentional overexposure [22-24]. In addition, potential interactions between cinnamon and pharmacological treatments should be considered. Given its effects on glucose metabolism and insulin sensitivity, cinnamon may potentiate the action of antidiabetic drugs, potentially leading to hypoglycemia if not properly monitored. However, data on drug–nutrient interactions remain limited, highlighting the need for further investigation in clinical settings [7,40]. Finally, long-term safety and efficacy data are lacking. Most available studies evaluate relatively short-term interventions, and there is insufficient evidence regarding the sustained effects of cinnamon supplementation on body weight, metabolic health, and potential adverse outcomes over prolonged periods. Therefore, further well-designed, large-scale randomized controlled trials are required to establish standardized dosing strategies, clarify pharmacokinetic properties, and confirm long-term safety and clinical relevance.

Discussion

The present review highlights the potential of cinnamon and its bioactive compounds as modulators of metabolic pathways associated with obesity. The collected evidence indicates that cinnamon exerts its effects through multiple mechanisms, including improvement of insulin sensitivity, inhibition of adipogenesis, enhancement of lipid metabolism, and reduction of inflammation and oxidative stress. These findings are consistent with the multifactorial nature of obesity, which involves dysregulation across several interconnected biological systems [7,17]. One of the most important strengths of cinnamon as a potential anti-obesity agent is its multi-target mode of action. Unlike conventional pharmacological treatments, which often focus on a single pathway, cinnamon compounds such as cinnamaldehyde and polyphenols simultaneously influence glucose metabolism, lipid homeostasis, and inflammatory signaling. For instance, activation of AMPK, inhibition of PPARγ-mediated adipogenesis, and modulation of NF-κB-driven inflammation collectively contribute to improved metabolic outcomes. This pleiotropic activity may be particularly advantageous in addressing complex metabolic disorders such as obesity and metabolic syndrome [17,20]. Experimental studies provide strong support for these mechanisms. In vitro and animal models consistently demonstrate reductions in adipocyte differentiation, lipid accumulation, and inflammatory markers, as well as improvements in thermogenesis and energy expenditure. The induction of adipose tissue browning and enhancement of mitochondrial activity observed in animal studies represent especially promising mechanisms, as they directly target energy balance and fat utilization [33,36,37]. However, despite robust preclinical evidence, the translation of these findings into clinical outcomes remains limited. Human studies suggest that cinnamon supplementation may lead to modest reductions in body weight, BMI, and fat mass, particularly in individuals with metabolic disorders. Nevertheless, the magnitude of these effects is generally smaller than those observed in pharmacological interventions such as GLP-1 receptor agonists, which can produce substantial and clinically significant weight loss [8,10,39,40]. This discrepancy highlights the need to consider cinnamon primarily as a supportive or adjunctive intervention rather than a standalone therapeutic strategy. Another important issue is the heterogeneity of clinical evidence. Differences in cinnamon species, dosage, duration of supplementation, and baseline characteristics of study populations contribute to variability in reported outcomes. In particular, the lack of standardization in cinnamon preparations makes it difficult to establish clear dose–response relationships or identify optimal treatment protocols. Moreover, many studies do not specify whether Ceylon or *cassia* cinnamon was used, which is critical given the differences in chemical composition and safety profiles [7,23]. The pharmacokinetic properties of cinnamon compounds further complicate the interpretation of results. The rapid metabolism of cinnamaldehyde and the relatively low bioavailability of polyphenols may limit their systemic effects in humans. Emerging research suggests that advanced delivery systems could enhance the stability and bioavailability of these compounds, potentially improving their therapeutic efficacy. However, this area remains underexplored and requires further investigation [21]. Safety considerations also play a crucial role in evaluating the clinical applicability of cinnamon. While generally regarded as safe when consumed in dietary amounts, higher doses used in supplementation may pose risks, particularly due to coumarin content in cassia cinnamon. Chronic exposure to coumarin has been associated with hepatotoxicity, emphasizing the importance of species selection and quality control in both research and practical applications [22-24]. In comparison with emerging pharmacological treatments for obesity, cinnamon offers certain advantages, including low cost, wide availability, and a favorable safety profile at moderate doses. However, its efficacy is considerably lower than that of modern anti-obesity drugs, such as GLP-1 receptor agonists or dual incretin therapies. These pharmacological agents have demonstrated substantial weight reduction in large-scale clinical trials but are associated with high costs and adverse effects, including gastrointestinal disturbances and potential long-term risks [8,10]. Therefore, cinnamon may be best positioned as a complementary intervention that enhances the effects of lifestyle modification or pharmacotherapy rather than replacing them. Overall, the current evidence suggests that cinnamon has promising anti-obesity potential, particularly through its multi-target metabolic effects. However, significant gaps remain, including insufficient clinical data, lack of standardization, and limited understanding of pharmacokinetics. Future research should focus on well-designed randomized controlled trials, standardized cinnamon preparations, and improved delivery systems to fully elucidate its role in obesity management.

Conclusions

Cinnamon and its bioactive compounds, particularly cinnamaldehyde and polyphenols, demonstrate considerable potential in the modulation of metabolic pathways associated with obesity. Evidence from experimental studies indicates that cinnamon can influence key mechanisms, including improved insulin sensitivity, inhibition of adipogenesis, stimulation of fatty acid oxidation, induction of thermogenesis, and reduction of inflammation and oxidative stress. These multi-target effects are particularly relevant given the complex and multifactorial nature of obesity. Clinical studies suggest that cinnamon supplementation may lead to modest improvements in body weight, body mass index, and metabolic parameters, especially in individuals with metabolic disturbances. However, the magnitude of these effects is generally limited and less pronounced compared to pharmacological interventions currently used in obesity treatment. Several important limitations must be considered, including the lack of standardization of cinnamon preparations, variability in botanical species, limited number of high-quality human studies, and insufficient understanding of pharmacokinetics and bioavailability of active compounds. Additionally, safety concerns related to coumarin content, particularly in *cassia* cinnamon, should be taken into account when considering long-term or high-dose supplementation. Overall, cinnamon appears to be a promising adjunctive strategy in obesity management, particularly as a component of dietary interventions or in combination with lifestyle modification. Further well-designed randomized controlled trials and studies focusing on standardized formulations and delivery systems are necessary to fully establish its efficacy, optimal dosing, and long-term safety.

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