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Cape gooseberry (*Physalis peruviana* L.) as a functional food for metabolic health: A narrative review of glucose and lipid regulatory effects

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ABSTRACT

Metabolic syndrome is a multifactorial and progressive disorder characterized by obesity, dyslipidemia, type 2 diabetes, and other metabolism-related conditions. Cape gooseberry (CP) (*Physalis peruviana* L.), an exotic fruit rich in bioactive compounds, has attracted growing attention for its potential benefits on metabolic health. This narrative review critically examines preclinical and clinical studies evaluating the metabolic effects of CP consumption, with a focus on biomarkers related to glucose and lipid metabolism, oxidative stress, and inflammation. A comprehensive search was conducted in four major scientific databases. A total of 40 studies were analyzed, including 28 preclinical investigations, 2 clinical trials, and 10 complementary studies addressing compound characterization and the impact of processing. While preclinical studies consistently report beneficial effects in animal models, clinical evidence is limited to two studies with heterogeneous results, both of which are affected by methodological limitations such as small sample sizes, short intervention durations, and inadequate dosing. Additionally, this review explores processing techniques aimed at preserving the bioactive profile of the fruit, which may enhance its application in functional food formulations. Despite encouraging evidence from preclinical studies, robust randomized controlled trials are required to validate the therapeutic potential of CP in the prevention and management of metabolic syndrome in humans.

1. INTRODUCTION

Metabolic syndrome is a multifactorial and progressive condition characterized by the simultaneous presence of central obesity, insulin resistance, dyslipidemia, and hypertension, all of which contribute to an increased risk of type 2 diabetes mellitus, cardiovascular disease, and other chronic non-communicable diseases [1,2]. The global prevalence of metabolic disorders such as diabetes mellitus and obesity continues to rise at an alarming rate. Limitations associated with pharmacological therapies, including cost, side effects, and long-term adherence, have led to growing interest in alternative and complementary approaches, particularly dietary strategies involving functional foods rich in bioactive compounds [3,4].

Functional foods, defined as those providing health benefits beyond basic nutrition, have emerged as an innovative and preventive strategy in this context [3,4]. Among the promising candidates is *Physalis peruviana* L., commonly known as cape gooseberry (CP) or goldenberry, an exotic fruit that is gaining scientific attention due to its rich phytochemical composition and associated bioactivities [5].

CP, an edible fruit native to the Andes and widely cultivated in South America, Africa, and Asia, has been identified as a promising functional food owing to its diverse phytochemical profile and traditional medicinal uses [6,7]. It has been traditionally used to treat respiratory, gastrointestinal, and inflammatory disorders [8–10]. These ethnopharmacological applications have sparked scientific interest, leading to numerous investigations identifying its

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bioactive constituents, including β-carotene, lycopene, lutein, quercetin, rutin, peruviosides, β-sitosterol, stigmasterol, lupeol, linalool, and various withanolides, which have been associated with biological activities relevant to metabolic health [11].

Such interest has been reinforced by preclinical findings, in which various preparations of the fruit, such as fresh pulp, juice, freeze-dried pomace, and methanolic extracts, have demonstrated antioxidant, anti-inflammatory, hypoglycemic, and hypolipidemic effects, mainly through modulation of metabolic and cellular pathways associated with oxidative stress and insulin resistance [12–15]. However, the clinical translation of these findings remains limited, with only two human studies conducted to date, both reporting heterogeneous results and significant methodological limitations [16,17].

This narrative review aims to critically synthesize the existing preclinical and clinical evidence on the metabolic effects of CP consumption, focusing on its role in the management of diabetes and obesity. Additionally, the review highlights methodological inconsistencies across reported studies and discusses current gaps in clinical findings. Furthermore, it explores preservation strategies to extend the shelf life of the fruit and compiles data supporting the development of CP–based functional foods.

2. MATERIALS AND METHODS

This paper is structured as a narrative review, aimed at synthesizing current scientific evidence regarding the potential of CP as a functional food in the management of metabolic disorders, particularly diabetes mellitus and obesity. It adopts a comprehensive and qualitative approach to information gathering, organization, and interpretation, allowing for integration of diverse findings across both preclinical and clinical contexts.

2.1. Search strategy and data sources

A literature search was conducted using four major scientific databases, including PubMed, ScienceDirect, SciELO, and Google Scholar, to identify relevant publications from January 2005 to May 2025. The timeframe was selected to cover the most recent 20 years of research on the topic and capture both early and contemporary findings.

Search terms included combinations of the following keywords: "*Physalis peruviana* L.", "*Cape gooseberry*", "*Goldenberry*", "*Functional food*", "*Metabolic diseases*", "*Diabetes mellitus*", "*Obesity*", "*In vivo*", "*Folk medicine*", and "*Bioactive compounds*". Boolean operators (AND, OR, NOT) were applied to refine the results.

2.2. Inclusion and exclusion criteria

To guide our analysis, we applied the following inclusion criteria: (1) original clinical or preclinical studies evaluating the effects of CP or its derivatives on metabolic parameters related to diabetes or obesity; (2) articles published in English or Spanish; and (3) studies published in peer-reviewed journals between 2005 and 2025. The exclusion criteria were: (1) studies based solely on *in vitro* experiments without *in vivo* or clinical relevance; (2) review articles, conference abstracts, editorials, or opinion pieces; and (3) studies lacking explicit data on metabolic biomarkers or adequate methodological detail.

2.3. Selection and data extraction

An initial screening of titles and abstracts was performed to identify potentially relevant articles. Full-text articles were then retrieved and assessed in detail based on the predefined inclusion and exclusion criteria. The final selection comprised 40 primary studies: 28 preclinical and 2 clinical investigations reporting on metabolic outcomes, bioactive compounds, or food processing methods related to CP. The remaining 10 studies, while not directly evaluating metabolic biomarkers, provided complementary insights into the characterization of bioactive compounds, technological processing, or formulation strategies relevant to the functional food applications of CP.

The selected studies were analyzed narratively, focusing on the biological effects of the fruit and its constituents, the mechanisms of action proposed, and their implications for the prevention or management of metabolic diseases. Critical appraisal was applied to compare findings, highlight inconsistencies, and identify research gaps.

3. RESULTS AND DISCUSSION

3.1. Overview of the evidence

A total of 40 articles were included in this review, comprising 28 preclinical studies, 2 clinical trials, and 10 supporting studies. Most of the preclinical research used rodent models to evaluate the metabolic effects of CP in the context of diabetes mellitus, obesity, and dyslipidemia. The formulations varied considerably, ranging from fresh fruit and juice to extracts and processed pulp. In contrast, the clinical evidence remains limited, both in volume and quality, which restricts the ability to draw strong conclusions about translational applicability.

3.2. Analysis of reported information

3.2.1. Benefits of CP consumption in the management of metabolic syndrome-related diseases: diabetes and obesity

Metabolic syndrome is a chronic and progressive pathophysiological condition that increases the risk of diseases such as dyslipidemia, type 2 diabetes mellitus, and obesity [1,2]. The pathophysiology of diabetes and obesity is complex and multifactorial, involving chronic inflammation, oxidative stress, insulin resistance, and deregulated lipid metabolism. Dysfunctional adipose tissue releases pro-inflammatory mediators that promote the infiltration of various immune cells, mainly macrophages, generating a pro-inflammatory environment with the participation of cytokines such as Tumor Necrosis Factor Alpha (TNF-α) and Interleukin 6 (IL-6). These cytokines play a key role in impairing insulin signaling, which in the long term leads to systemic insulin resistance and the exacerbation of metabolic dysfunction [18–21]. Oxidative stress, defined by an imbalance between pro-oxidants and antioxidant defenses, is another critical factor contributing to the development of insulin resistance, β-cell dysfunction, and

lipid peroxidation. These processes culminate in progressive metabolic deterioration and increase the risk of cardiovascular disease and type 2 diabetes [22,23].

Given their high morbidity and mortality rates, type 2 diabetes mellitus and obesity represent a major public health challenge [1,2]. Based on data from the International Diabetes Federation Diabetes Atlas, it is estimated that around 537 million adults between the ages of 20 and 79 are living with diabetes globally, a number expected to rise to 643 million by 2030 [24]. Conversely, the World Health Organization reported that in 2022, approximately 2.5 billion adults were overweight, with around 890 million (16%) classified as obese [25]. A key strategy to prevent and control these pathologies is the adoption of a nutritionally balanced diet rich in vegetables and fruits. These foods contain secondary metabolites with well-documented biological activities [3,4].

CP, a fruit that has gained increasing attention in recent decades, is an example of a nutrient-rich food with potential health benefits [5]. Historically, this fruit has been used to treat digestive disorders such as indigestion and gastric acidity, as well as to strengthen the immune system. In Indonesia, it is commonly used for the treatment of diabetes mellitus and related conditions [6], while in China, it is employed for its anticancer, detoxifying, and anti-inflammatory properties [7]. In Latin America, CP has traditionally been used to treat conditions such as asthma, dermatitis, fever, ulcers, and inflammation, as well as to help reduce blood cholesterol levels [8–10].

The ethnopharmacological use of CP has generated great scientific interest, resulting in numerous *in vitro* and *in vivo* studies. These investigations have provided evidence supporting the therapeutic properties of compounds present in the fruit, which may play a crucial role in regulating glucose metabolism and lipid profiles, both of which are key factors in metabolic syndrome.

3.2.1.1. Preclinical studies

To evaluate the metabolic effects of functional foods, several biomarkers have been employed, including fasting blood glucose, HbA1c, triglycerides, high-density lipoprotein (HDL)/low-density lipoprotein (LDL) cholesterol, insulin, as well as markers of inflammation and oxidative stress. Animal studies consistently show that CP consumption exerts beneficial effects in models of obesity and dyslipidemia by improving lipid profiles, lowering blood glucose, and mitigating inflammation and oxidative damage. A summary of the main findings is provided in Table 1.

The effects of filtered and freeze-dried CP juice were evaluated in Wistar rats with diabetes induced by streptozotocin (STZ), followed by 24 hours of *ad libitum* access to a 20% sucrose solution to exacerbate hyperglycemia, as described by Mora *et al.* [12]. The juice was administered orally at a dose of 250 mg/kg/day for 15 days. The treatment led to a reduction in blood glucose levels exceeding 30%, along with increased ferric reducing ability of plasma and enhanced activity of key antioxidant enzymes, including superoxide dismutase (SOD) and catalase (CAT). Additionally, a decrease in lipid peroxidation and protein oxidation was observed. These findings support the hypoglycemic and antioxidant potential of CP in diabetic models.

In a separate study, Dewi *et al.* [26] investigated the effects of filtered CP juice (excluding seeds and skins) and quercetin on insulin resistance and oxidative stress in 36 Wistar rats. The animals were fed a high-fat diet and received STZ to induce insulin resistance. Over a 28-day period, the rats were treated via oral gavage with doses of 5 and 25 ml/kg/day, as well as quercetin at 2.2 and 30 mg/kg/day. The administration of the higher doses (25 ml/kg/day of CP juice and 30 mg/kg/day of quercetin) resulted in a significant increase in total antioxidant capacity and adiponectin levels. This enhancement in adiponectin was suggested to be mediated through the upregulation of peroxisome proliferator-activated receptor-γ (PPAR-γ) mRNA, potentially induced by the flavonoid content of the treatments [26].

In a comparable approach, the protective role of CP on oxidative damage was assessed in a type 1 diabetes model using 27 Wistar rats, as detailed by Fazilet *et al.* [13]. The intervention involved the intraperitoneal administration of a CP extract (1 ml/kg, twice weekly) combined with 2 g of CP powder dissolved in drinking water, provided ad libitum over a 60-day period. This combined treatment resulted in a significant increase in glutathione levels and a reduction in blood glucose and malondialdehyde (MDA), a biomarker of lipid peroxidation. These findings support the potential of CP as a therapeutic agent for managing hyperglycemia and oxidative stress in diabetes.

A study conducted in an acute hyperlipidemia model using 24 *Mus musculus* var. Swiss mice demonstrated the hypolipidemic effects of CP, as reported by Campos *et al.* [27]. Hyperlipidemia was induced by Triton, and the intervention consisted of a single oral dose of dehydrated CP juice, prepared from filtered fresh fruit, administered at 0.05 g/100 g and 0.2 g/100 g of body weight. After 24 hours, a significant reduction in plasma cholesterol and triglyceride levels was observed. These effects were attributed to the flavonoid content of CP, suggesting its potential utility as a rapid-acting agent in the management of hyperlipidemia.

Furthermore, two studies conducted by Ramadan *et al.* [28] explored the hypolipidemic and antioxidant properties of different CP preparations in male albino rats with a high-fat diet. In the first study, Ramadan *et al.* [28] supplemented the diet with filtered and pasteurized juice at concentrations of 5% and 15% for 60 days. This intervention led to significant reductions in total cholesterol, triglycerides, and LDL levels, as well as an increase in HDL levels. Histological analyses also revealed normalization of liver tissues, suggesting that the observed effects may be attributed to the flavonoid content of the juice and its capacity to inhibit LDL oxidation [28]. In a subsequent study, Ramadan [29] administered freeze-dried CP pomace (containing skins and seeds) at dietary concentrations of 10% and 30% for 60 days. Similar improvements in lipid profiles were observed, along with protection against hepatic oxidative stress and the development of fatty liver in rats fed a high-fat diet [29].

In addition, the metabolic effects of whole CP pulp (including peel and seeds) were evaluated in male C57BL/6 mice with diet-induced obesity in a study conducted by Pino-de

Table 1. Primary outcomes of preclinical trials on the effects of CP consumption in diabetes management and lipid profile modulation.

Preclinical studies					
Form of the fruit used and dosage	**Disease(s)**	**Method applied**	**Duration of the treatment**	**Main effect and/or mechanism of action**	**References**
CP juice (filtered and freeze-dried) Dose: 250 mg/kg/day (Orally)	Diabetes mellitus	Female Wistar rats with diabetes induced by STZ, followed by *ad* access to a 20% sucrose solution for 24 hours/day over 15 days	15 days	↓ Blood glucose ↓ Lipid peroxidation ↓ Protein oxidation ↑ Activity of antioxidant enzymes (SOD and CAT)	[12]
CP juice (Undiluted, filtered to remove seeds and skins) Dose: 5 and 25 ml/kg/day (Oral gavage)	Diabetes mellitus	Wistar rats with a high-fat diet and STZ	28 days	↑ Total antioxidant capacity ↑ Adiponectin levels	[26]
CP powder (extracted with hexane–isopropanol, filtered, and dissolved in DMSO for injection) Dose: 1 ml extract/kg, twice a week (Intraperitoneally) + 2 g CP powder dissolved in water	Diabetes mellitus	Male albino Wistar rats with STZ-induced type 1 diabetes	60 days	↓ Glucose levels ↓ MDA ↑ Glutathione levels	[13]
CP juice (Dehydrated juice from filtered fresh fruit) Single dose: 0.05 g/100 g and 0.2 g/100 g of body weight	Hyperlipidemia	Male *Mus musculus* var. *Swiss* with acute hyperlipidemia induced by Triton	1 day	↓ Cholesterol levels ↓ Triglyceride levels	[27]
CP juice (Filtered pasteurized juice) Dose: Diet supplemented with 5% and 15% of juice	Hypercholesterolemia	Male albino rats with a high-fat diet	60 days	↓ GPT ↓ Cholesterol levels ↓ Triacylglycerols ↓ LDL ↑ HDL	[28]
CP freeze-dried pomace (skins and seeds) Dose: Diet supplemented with 10% and 30% of pomace	Hypercholesterolemia	Male albino rats with a high-fat diet	60 days	↓ Cholesterol levels ↓ Triglycerides ↓ LDL ↑ HDL	[29]
CP pulp from whole fruit (including peel and seeds) Dose: 300 mg/kg/day (administered by gavage)	Obesity and insulin resistance	Obese male C57BL/6 mice induced by a high-fat diet	56 days	↓ Fasting blood glucose and insulin levels ↓ Proinflammatory markers in the liver [mRNA of TNF-α, IL-6, IL-1β, and TLR4] ↓ Hepatic and skeletal muscle lipid peroxidation	[14]

(Continued)

Preclinical studies					
Form of the fruit used and dosage	**Disease(s)**	**Method applied**	**Duration of the treatment**	**Main effect and/or mechanism of action**	**References**
Fresh CP finely chopped Dose: Diet supplemented with 8% (w/w) of fresh fruit	Obesity	Obese Wistar rats with metabolic syndrome induced by a high-fat diet	16 days	↓ Body weight, liver, pancreas, visceral and subcutaneous adipose tissue ↓ Blood glucose ↓ FasN expression ↓ LPL expression ↑ INSR expression ↑ PPARγ expression	[30]
CP methanolic extract (70% MeOH, vacuum evaporated and lyophilized) Dose: 200 and 400 mg/kg/day of extract (Orally)	Obesity	Female Wistar rats with obesity induced by a high-fat and sucrose diet	56 days	↓ BMI ↓ Cholesterol levels ↓ Triglycerides ↓ Glucose levels ↓ Insulin resistance index ↓ Leptin levels ↓ Inflammatory markers (TNF-α, IL-2, IL-6, and C-reactive protein) ↑ HDL levels ↑ Adiponectin levels	[15]
CP juice (Freshly prepared, filtered pulp from ripe fruits) Dose: 5 ml/kg/day (Orally)	Diabetes mellitus	Male rats with STZ-induced diabetes	56 days	↓Glucose ↓MDA ↑CAT, GR Improved serum AGE levels Improved lipid profile	[31]
CP fruit powder methanolic dried extract Dose: 200 and 400 mg/kg/day (Orally)	Hyperglycemia	NMRI white mice with hyperglycemia induced by oral administration of a glucose solution (4 g/kg body weight/day)	213 days	↓Glucose The antihyperglycemic effect is observed at 30 minutes	[32]

STZ: streptozotocin; SOD: superoxide dismutase; CAT: catalase; GPT: glutamic pyruvic transaminase; LDL: low-density lipoprotein; HDL: high-density lipoprotein; mRNA: messenger ribonucleic acid; TNF-α: tumor necrosis factor alpha; IL: interleukin, TLR4: toll-like receptor 4; FasN: fatty acid synthase; INSR: insulin receptor; LPL: lipoprotein lipase; INSR: insulin receptor; PPARγ: peroxisome proliferator-activated receptor gamma; BMI: body mass index; MDA: malondialdehyde; AGE: advanced glycation end-products.

la Fuente *et al.* [14]. In this study, 32 obese mice were fed a high-fat diet and treated for 56 days with a dietary supplementation of 300 mg/kg/day of CP pulp. The intervention effectively prevented visceral fat accumulation, reduced fasting blood glucose levels, and decreased lipid peroxidation in both liver and muscle tissues. Furthermore, the treatment led to a marked reduction in the expression of pro-inflammatory markers, including TNF-α mRNA, IL-6, IL-1β, and TLR4. These findings suggest that the daily consumption of whole CP pulp may serve as a promising nutritional strategy to prevent insulin resistance and liver damage in the context of diet-induced obesity.

In a complementary study, Ángel-Martín *et al.* [30] supplemented the diet of 64 obese Wistar rats with 8% (w/w) of finely chopped fresh CP for 16 days. The intervention not only prevented excessive weight gain but also significantly improved lipid profiles and reduced visceral and subcutaneous fat accumulation. Notably, beneficial effects were also observed in vital organs such as the liver and pancreas, likely due to the high content of polyphenols and vitamins A and C. Furthermore, modulation of key metabolic genes, including INSR, PPARγ, FasN, and LPL, was reported, reinforcing the fruit's potential role in improving metabolic health [30].

Furthermore, the effects of a methanolic extract of CP on hepatic oxidative stress and metabolic syndrome were examined in obese female Wistar rats by Moussa *et al.* [15]. The extract was administered orally at doses of 200 and 400 mg/kg/day for 56 days. The intervention led to significant improvements in multiple metabolic parameters, including reductions in body mass index (BMI), total cholesterol, triglycerides, glucose levels, and the insulin resistance index. In addition, HDL cholesterol levels increased, accompanied by favorable modulation of adipokines, as evidenced by decreased leptin and elevated adiponectin levels. The treatment also attenuated oxidative stress and systemic inflammation, as indicated by lower levels of TNF-α, IL-2, IL-6, and C-reactive protein. Collectively, these findings highlight the potential of CP methanolic extract as a therapeutic agent for alleviating key features of metabolic syndrome, such as hyperlipidemia, diabetes mellitus, and fatty liver disease.

Additionally, Aljadani *et al.* [31] investigated the effects of freshly prepared CP juice, obtained from filtered pulp of ripe fruits, on rats with STZ-induced type 1 diabetes. The juice was administered orally at a dose of 5 ml/kg/day for 56 days, either alone or in combination with metformin. The intervention led to a significant reduction in blood glucose levels, improved lipid profiles, and renal protection, as evidenced by decreased levels of kidney damage markers. Oxidative stress was also markedly attenuated, with lower MDA concentrations and enhanced activity of antioxidant enzymes such as CAT and glutathione reductase. Furthermore, the juice inhibited the formation of advanced glycation end-products (AGEs), which are closely associated with diabetic complications. Notably, co-administration with metformin amplified these protective effects, underscoring the potential of CP juice as an effective adjunct therapy for glycemic control and renal protection in diabetes [31].

Most recently, Tshibangu *et al.* [32] evaluated the antihyperglycemic potential of a methanolic dried extract derived from CP fruit powder Naval Medical Research Institute (NMRI) white mice with glucose-induced hyperglycemia. Hyperglycemia was triggered by the oral administration of a glucose solution (4 g/kg body weight/day), and the extract was administered daily at doses of 200 and 400 mg/kg over a 213-day treatment period. Control groups received distilled water, glibenclamide (5 mg/kg), or metformin (25 mg/kg). Blood glucose levels were assessed at 30 and 150 minutes after glucose loading. Both extract doses significantly reduced glycemia at both time points, with the 400 mg/kg dose exhibiting effects comparable to those of glibenclamide and metformin. These findings further support the antidiabetic potential of CP and are consistent with previous studies highlighting its therapeutic properties [32].

3.2.1.1.1. Analysis of preclinical studies focused on diabetes.

Preclinical studies consistently demonstrate that CP exerts beneficial metabolic effects, including reductions in fasting glucose, triglycerides, total cholesterol, and LDL cholesterol, along with increases in HDL levels and antioxidant enzyme activity (Table 1). However, despite this general consistency, the interpretation of the findings is challenged by notable methodological heterogeneity.

To assess the impact of CP on blood glucose regulation, most studies employed STZ to induce diabetes in Wistar rats. An exception was the study by Tshibangu *et al.* [32], who induced hyperglycemia in NMRI white mice *via* oral glucose administration.

Instead of oral supplementation, the study by Fazilet *et al.* [13] administered an intraperitoneal extract supplemented with 2 g of CP fruit powder in water to male Wistar rats with STZ-induced type 1 diabetes for 60 days. This intervention resulted in decreased blood glucose and (MDA, a marker of lipid peroxidation), alongside elevated glutathione levels. A similar treatment duration was used by Aljadani *et al.* [31], who administered CP juice orally (5 ml/kg/day) for 56 days in a comparable diabetic model. While both studies reported improvements in glycemic control and oxidative stress, Aljadani *et al.* [31] observed broader benefits, including enhanced lipid profiles, reduced levels of serum advanced AGEs, and increased activity of antioxidant enzymes such as CAT and glutathione reductase.

Although the study by Pino-de la Fuente *et al.* [14] employed a treatment duration comparable to that of Fazilet *et al.* [13] and Aljadani *et al.* [31], their study diverged in key methodological aspects. Unlike the others, Pino-de la Fuente *et al.* [14] did not induce diabetes via STZ; instead, insulin resistance was triggered through a high-fat diet in obese male C57BL/6 mice. Additionally, different matrices were used for fruit delivery: fruit powder dissolved in water [13], juice [31], and pulp incorporated into the diet [14]. These discrepancies limit direct comparisons. Nevertheless, all three studies consistently reported reductions in blood glucose and lipid peroxidation, two key biomarkers across the investigations, suggesting that both Wistar rats and C57BL/6 mice are suitable models and that various CP preparations (juice, powder, and pulp) retain biologically active compounds with hypoglycemic and antioxidant properties.

The experimental design proposed by Dewi *et al.* [26] closely resembles that of Aljadani *et al.* [31], as both employed STZ to induce diabetes and administered CP juice at comparable doses (5 and 25 ml/kg/day). However, Dewi *et al.* [26] applied the treatment for only 28 days, half the duration used by Aljadani *et al.* [31]. Moreover, the studies assessed different sets of biomarkers, preventing a comprehensive comparison. Dewi *et al.* [26] primarily reported increased total antioxidant capacity, without evaluating other parameters such as AGEs or antioxidant enzyme activity. Therefore, conclusions regarding treatment duration and effect consistency remain limited.

A methodology similar to that of Dewi *et al.* [26] was proposed by Mora *et al.* [12], as the same STZ-induced diabetic model and short intervention periods (15 and 28 days, respectively) were used in both studies. While the preparation methods were largely analogous, Mora *et al.* [12] administered a dehydrated juice form, which resulted in dosage units being expressed in milligrams rather than milliliters. Due to differences in the selected biomarkers, the results could not be directly compared. Nonetheless, both studies demonstrated that CP exerted significant antioxidant activity.

3.2.1.1.2. Analysis of preclinical studies focused on obesity.

As shown in Table 1, to explore the effects of CP on obesity and dyslipidemia, most experimental models employed

high-fat diets to induce metabolic alterations. An exception was the study by Campos *et al.* [27], who induced hyperlipidemia acutely using Triton in Swiss mice. These studies also varied in the rodent species used, which adds to the methodological diversity observed across the literature. Nevertheless, a common trend emerged: CP consistently demonstrated the ability to reduce total cholesterol and triglyceride levels, and in several cases, to improve the lipoprotein profile (LDL/HDL).

Whole CP fruit was administered to obese rodents in both studies conducted by Pino-de la Fuente *et al.* [14] and Ángel-Martín *et al.* [30], using C57BL/6 mice and Wistar rats, respectively. However, differences in the form of administration and treatment duration (56 days in the former and 16 days in the latter) preclude direct comparison of dosages. Despite these differences, both studies reported reductions in blood glucose levels, indicating that the hypoglycemic effects of CP can emerge within a short timeframe (16 days) and be sustained with longer interventions (56 days). Comparison of other biomarkers was not possible, as the two studies targeted different physiological pathways: Pino-de la Fuente *et al.* [14] focused on inflammation and oxidative stress, whereas Ángel-Martín *et al.* [30] investigated lipid metabolism through enzyme activity and gene expression. Importantly, both studies yielded favorable results for their respective endpoints.

Supporting these findings, Moussa *et al.* [15] conducted a study with a similar treatment duration (56 days), in which a methanolic extract of CP was administered to obese Wistar rats. This study stands out for its use of 70% methanol during sample preparation, an approach that effectively extracted bioactive constituents responsible for the observed biological effects. Their results, particularly regarding reductions in pro-inflammatory cytokines TNF-α and IL-6, closely aligned with those reported by Pino-de la Fuente *et al.* [14], reinforcing the anti-inflammatory potential of the fruit in obesity-related contexts.

Additional evidence comes from studies where the whole fruit was not administered. For instance, Ramadan *et al.* [28] administered filtered juice devoid of seeds and skin, while Ramadan [29] used only the skin and seeds. Despite these differences in sample composition, both studies reported significant improvements in lipid profiles, including reductions in total cholesterol, triacylglycerols, and LDL, alongside increases in HDL. These findings suggest that the bioactive compounds responsible for these effects are distributed across different parts of the fruit. Moreover, both studies employed a treatment duration of 60 days, similar to the 56-day protocol used by Pino-de la Fuente *et al.* [14]. However, due to differences in biomarker selection, cross-study comparisons remain limited.

In contrast to these long-term studies, Campos *et al.* [27] implemented a single-dose, acute model of hyperlipidemia. They administered dehydrated CP juice at two doses (0.05 g/100 g and 0.2 g/100 g body weight) and observed significant reductions in cholesterol and triglyceride levels within 24 hours. These results demonstrate that the beneficial effects of CP on lipid metabolism may be observed rapidly, following even a single administration, a particularly promising finding.

3.2.1.2. Clinical studies

Despite the existing gaps in preclinical research, the promising results reported in individual studies targeting diabetes and obesity have led to the initiation of clinical trials investigating the therapeutic potential of CP in the management of metabolic diseases, particularly diabetes. To date, clinical evidence on the effects of CP in humans remains limited and preliminary, with only two available studies reporting heterogeneous outcomes regarding insulin sensitivity and blood glucose levels. A summary of the main findings from these studies is provided in Table 2.

The effects of CP on insulin signaling and its potential antidiabetic properties were evaluated in a group of 18 healthy male volunteers aged 20–50 years by Vaillant *et al.* [16]. During the first phase of the study, blood samples were collected after fasting and again 6 hours after participants consumed 250 g of fresh CP along with a standardized breakfast and lunch. Subsequently, the participants consumed 150 g of CP daily for 19 days, after which new blood samples were analyzed. The results showed that CP intake was associated with the modulation of biological pathways related to insulin signaling,

Table 2. Primary results of clinical trials on the effects of CP consumption on diabetes management.

Clinical studies					
Form of the fruit used and dosage	**Disease(s)**	**Method applied**	**Duration of the study**	**Main effect and/or mechanism of action**	**Reference**
Fresh CP Dose: 150 and 250 g	Diabetes mellitus	18 healthy male volunteers	19 days	CP consumption was associated with biological pathways involving insulin, EGFR, and the PI3K/Akt/mTOR signaling cascade	[16]
Fresh CP Dose: 50 g	Diabetes mellitus	28 overweight and obese young adults	21 days	There was no significant reduction in glucose levels.	[17]

EGFR: Epidermal growth factor receptor

including the epidermal growth factor receptor (EGFR) and the PI3K/Akt/mTOR cascade. A total of 74 plasma metabolites were identified, with 11 metabolites common to both phases of the study. These findings suggest that CP exerts a measurable influence on insulin-related pathways, although this effect appears to diminish after cessation of consumption, yet remains detectable 24 hours post-intake.

In contrast, Ludeña-Meléndez *et al.* [17] conducted a quasi-experimental study involving 28 young adults (aged 18–26) with overweight or obesity, who were randomly assigned to either a control group or an intervention group. The intervention group consumed 50 g of CP daily for 21 days, while both groups followed a standardized low-carbohydrate diet. Although a significant reduction in fasting blood glucose was observed in both groups, no statistically significant difference was found between them. These results suggest that in non-diabetic individuals under dietary control, CP does not exert an additional hypoglycemic effect. This contrasts with previous findings in diabetic populations, where CP consumption has been associated with significant improvements in glycemic control [17].

3.2.1.2.1. Analysis of clinical studies.

A comparison of the two clinical studies that met the inclusion criteria reveals that, while both were based on preclinical evidence indicating that CP consumption can improve insulin sensitivity and reduce blood glucose levels, they pursued different objectives. The study by Vaillant *et al.* [16] investigated the acute and medium-term effects of CP intake on plasma metabolites related to insulin signaling pathways. The findings demonstrated a positive modulation of the PI3K/Akt/mTOR signaling cascade. Although the study did not evaluate clinical parameters such as blood glucose or insulin sensitivity, its findings suggest that compounds present in CP may exert subtle metabolic effects in healthy individuals.

In contrast, the study conducted by Ludeña-Meléndez *et al.* [17] aimed to evaluate the impact of CP consumption on fasting blood glucose in young adults with overweight or obesity. While a reduction in blood glucose was observed in both the intervention and control groups, no statistically significant difference was detected between them, and therefore, no glycemic benefit could be attributed to CP intake. This lack of effect may be explained by several methodological factors. First, both groups followed a standardized low-carbohydrate diet, which likely contributed to reduced glucose levels in both cases, diminishing the ability to isolate the specific effects of the fruit. Second, the participants did not present with marked metabolic impairment (e.g., type 2 diabetes), limiting the potential to observe clinically relevant improvements in glycemic control. Additionally, the mode of administration, the moderate daily dose, and the short intervention period may have further reduced the bioavailability and efficacy of the active compounds. The limited sample size also constrained the statistical power of the study.

3.2.1.3. Limitations of preclinical and clinical studies

The analysis of both preclinical and clinical studies revealed several limitations inherent to each type of study. In the case of preclinical evaluation of effects of CP on glucose metabolism, substantial variability in study design, including differences in disease induction methods, animal models, delivery matrices (pulp, extract, juice, and powder), doses, durations (ranging from 1 to 213 days), and biomarkers assessed, was observed. Despite this methodological diversity, the collective evidence supports the hypoglycemic and antioxidant effects of CP, often in line with conventional treatments. Nonetheless, greater standardization in experimental protocols is essential to improve the comparability and interpretability of future findings.

Similarly, in the context of preclinical studies focused on obesity, it is important to highlight a key insight from the collective analysis: despite notable differences in experimental models, animal species, administration forms, and biomarker panels, CP consistently showed beneficial effects on plasma lipid levels, glycemia, oxidative stress, and inflammation. This consistency supports the hypothesis that the bioactive compounds of the fruit retain their efficacy across various biological contexts. Nevertheless, further research is warranted to evaluate CP across short-, medium-, and long-term treatment durations, and using standardized formulations (e.g., whole pulp, freeze-dried pomace, and filtered pasteurized juice). Future studies should harmonize the selection of biomarkers for lipid metabolism, oxidative stress, and inflammation to facilitate cross-study comparisons and clarify the mechanisms underlying the therapeutic potential of CP.

Turning to clinical evidence, it is important to highlight that both studies included small sample sizes and short intervention periods. For instance, in the study by Ludeña-Meléndez *et al.* [17], the daily dose of CP administered was substantially lower than the recommended thresholds, considering both the glycemic index of the fruit and the World Health Organization's guidelines on daily fruit consumption [33]. These limitations weakened the statistical robustness and clinical significance of the findings. As a result, current clinical evidence remains inconclusive, and firm conclusions regarding the effects of CP on insulin sensitivity and glycemic regulation cannot yet be drawn.

Accordingly, these deficiencies underscore the need for well-designed randomized controlled trials with increased sample size and clearly defined CP doses and formulations. Consideration should also be given to including populations with clearly defined metabolic disorders in addition to healthy individuals. Future studies should explore the short- and long-term effects of CP consumption on oxidative stress, inflammation, glycemic control, and lipid metabolism. Notably, a recent study by Ndahura *et al.* [76] proposed a protocol for a randomized controlled trial aimed at evaluating the effect of CP consumption on blood glucose control in patients with type 2 diabetes.

The above strategies would contribute to providing a more accurate assessment of the potential of fruit as an adjuvant in the prevention and/or treatment of metabolic disorders such as diabetes and obesity.

3.2.2. Bioactive compounds of cape gooseberry fruit and their role in diabetes and obesity management

In individuals with obesity, dysfunctional adipose tissue persistently secretes pro-inflammatory cytokines such

as IL-6, IL-1β, and TNF-α, which activate immune cells, particularly macrophages, thereby initiating a state of chronic inflammation accompanied by oxidative stress [18,19]. Elevated TNF-α levels have been directly linked to the onset of insulin resistance, contributing to the development and progression of type 2 diabetes mellitus and further amplifying the systemic inflammatory response [20–22].

Phytochemical analyses of CP have revealed the presence of key secondary metabolites, including carotenoids, flavonoids, phytosterols, terpenes, and withanolides, which exhibit antioxidant, anti-inflammatory, antidiabetic, and anti-obesity activities. These bioactive compounds are considered the principal mediators of the potential health benefits associated with CP consumption, particularly in the prevention and/or management of type 2 diabetes mellitus and obesity (Fig. 1).

The collective findings from preclinical and clinical investigations suggest potential molecular mechanisms by which the secondary metabolites of CP exert their biological activities (Table 3).

As shown in Table 3, the carotenoids, flavonoids, phytosterols, terpenes, and withanolides present in CP exhibit significant antioxidant and anti-inflammatory activity through multiple mechanisms. These bioactive compounds act as potent antioxidants by stimulating key enzymes, including SOD, CAT, glutathione reductase (GR), and glutathione peroxidase (GPx), while simultaneously reducing lipid peroxidation and the generation of nitric oxide (NO) and reactive oxygen species (ROS). Additionally, they have been observed to increase the levels of the T-Nrf2 protein and heme oxygenase-1 (*HO-1)* mRNA.

The anti-inflammatory response is mediated through the inhibition of various pro-inflammatory mediators, such as NO, prostaglandin E2 (PGE2), and multiple cytokines. Furthermore, these compounds suppress the activity of enzymes including cyclooxygenase-2 (COX-2), lipoxygenase (LOX), inducible nitric oxide synthase (iNOS), and myeloperoxidase (MPO), while activating glutathione reductase (GR), glutathione S-transferase A4 (GSTA4), and HO-1. Consequently, the expression of key inflammatory markers such as *NLRP3, TLR4, MCP-1, PPARα,* and *PPARγ* is downregulated, while crucial signaling pathways, including JAK2/STAT3, JNK/p38 MAPK, and TLR4/MyD88/NF-κB P65, are modulated. These antioxidant and anti-inflammatory mechanisms may help explain the regulatory effects of these metabolites on glucose levels and lipid profiles. Several studies have established a direct correlation between oxidative stress, chronic inflammation, and the onset or progression of metabolic disorders such as obesity and diabetes [19,23].

Research has further demonstrated that flavonoids, phenolic acids, phytosterols, and terpenes exert antidiabetic effects by lowering blood glucose and HbA1c levels, enhancing insulin secretion, and promoting glycogen synthesis in the liver. These compounds activate the PI3K/p-Akt signaling pathway, facilitate glucose uptake by stimulating the translocation of glucose transporters (GLUT)-4, GLUT-2, and GLUT-1 transporters, and inhibit key enzymes involved in carbohydrate metabolism, such as α-glucosidase, α-amylase, and maltase. Additionally, they regulate the phosphorylation of IκBα/NF-κB and normalize the expression of key metabolic proteins, including protein kinase B (PKB), insulin receptor substrates (IRS-1 and IRS-2), and (GLUT-2 and GLUT-4). These bioactive compounds also enhance the phosphorylation of AMP-activated protein kinase (AMPK) and acetyl-CoA carboxylase (ACC) in adipose tissue, liver, and muscles, while increasing glucokinase (GCK) levels and boosting the activity of enzymes such as glucose-6-phosphate dehydrogenase and phosphohexose isomerase. Moreover, they help balance gluconeogenic

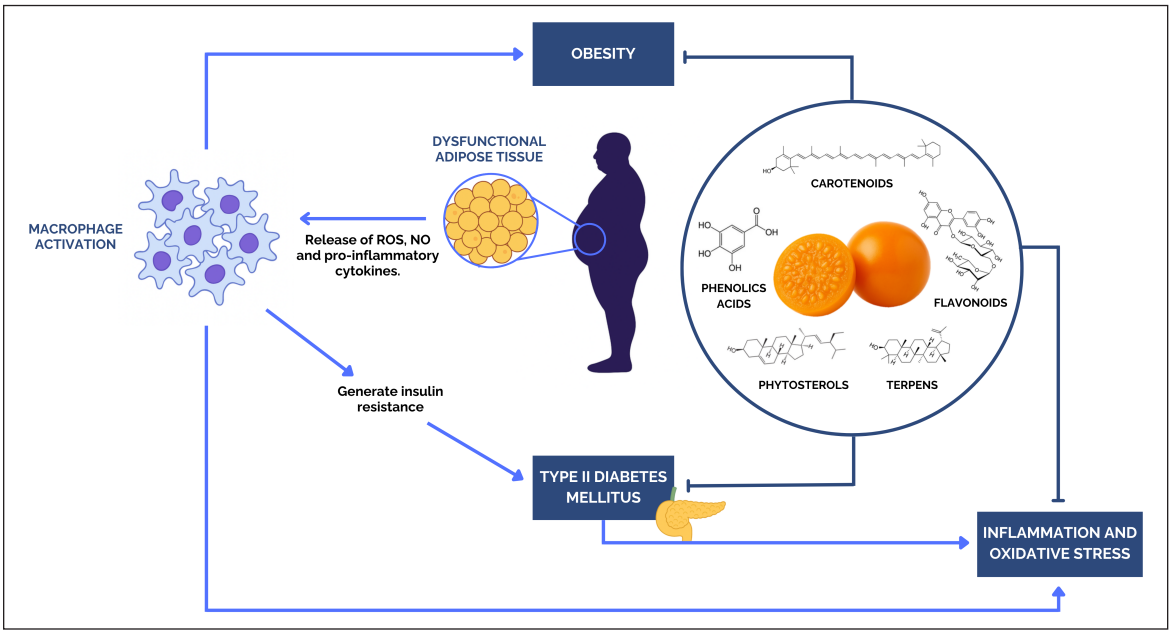

Figure 1. Bioactive secondary metabolites identified in CP including carotenoids, flavonoids, phytosterols, terpenes, and withanolides, and their associated antioxidant, anti-inflammatory, antidiabetic, and anti-obesity properties.

Table 3. Biological effects of bioactive secondary metabolites present in CP as complementary therapies for diabetes and lipid profile disorders.

Metabolite	Name	Activity	Mechanism of Action	Reference
Carotenoids	β-cryptoxanthin	Antioxidant, anti-inflammatory, antihyperlipidemic.	↓Inflammatory markers (IL-1β, IL-4, IL-6, IFN-γ, NF-κB) Suppresses lipid peroxidation. Inhibits lipid accumulation.	[42–44]
	β-carotene	Antioxidant, anti-inflammatory	↓IL-6, IL-1β, TNF-α Regulates JAK2/STAT3 and JNK/p38 MAPK signaling pathways. Prevents NF-κB translocation. Inhibits NLRP3. Suppresses PPARα and PPARγ expression and activity.	[11, 42, 45, 46]
	Lycopene	Antioxidant, anti-inflammatory	↓Inflammatory markers (TNF-α, IFN-γ, IL-2, IL-6, COX-2) ↓PGE2 production ↓NO production ↑IL-10	[11, 44, 47]
		Antihyperlipidemic	↓LDL ↓Cholesterol synthesis ↑HDL	[44]
	Lutein	Antioxidant, anti-inflammatory	↓IL-1β, IL-6, IL-12, COX-2 ↓ROS ↓NO ↑IL-10 Inhibits NF-κB	[11, 42, 44, 48]
		Antihyperlipidemic	↓LDL ↓VLDL ↓IDL ↓Triglycerides Inhibits MPO	[44]
Phenolic acid	Gallic acid	Antioxidant, anti-inflammatory	↓Lipid peroxidation ↓ROS production ↓Inflammatory markers (TNF-α, IFN-γ, IL-1β, IL-6, IL-17, IL-21, IL-23, COX-2, iNOS)	[11, 49, 59]
		Anti-diabetic	↑Activity of antioxidant enzymes (SOD, CAT, GR, GPx) ↑Stimulates the PI3K/p-Akt signaling pathway ↑Stimulates the translocation of GLUT-4, GLUT-2, and GLUT-1	[50]
Flavonoids	Quercetin	Antioxidant, anti-inflammatory	↓Inflammatory markers (TNF-α, IFN-γ, IL-1β, IL-6, IL-8, COX, LOX) ↓NO production	[11, 51, 52]
		Anti-diabetic	↓Glucose ↓Maltase activity ↓GLUT-2 activity	[51, 53]
		Antihyperlipidemic	↓Cholesterol ↓Triacylglycerides ↑Plasma adiponectin ↑HDL	[51]

Metabolite	Name	Activity	Mechanism of Action	Reference
		Antioxidant, anti-inflammatory	↓TNF-α, IL-1β, IL-6, IL-8, COX-2, NF-κB ↓MDA, ROS, NO, GSSG, iNOS ↓NO ↓ROS ↑CAT, SOD, GPx ↑IL-10	[11, 54, 55]
	Rutin	Anti-diabetic	↓Glucose ↑Insulin	[54]
		Antihyperlipidemic	↓Total cholesterol ↓Triglycerides ↓LDL	[54]
		Antioxidant, anti-inflammatory	↓Inflammatory markers (TNF-α, IL-6, IL-1β, COX-2) ↓NO ↑SOD, GPx	[11, 56]
	Myricetin	Anti-diabetic	↓Glucose ↓Suppresses α-glucosidase and α-amylase activity ↓IκBα/NF-κB phosphorylation Normalizes the expression of PKB, IRS-1, IRS-2, GLUT-2, and GLUT-4	[56]
		Anti-obesity	↓Body weight ↓Leptin ↓Cholesterol ↓Triglycerides ↓LDL ↓PPARγ expression ↑HDL ↑Energy expenditure Activates BAT	[56, 57]
	Kaempferol	Antioxidant, anti-inflammatory	↓ROS ↓NO ↓IL-1β, IL-6, TNF-α ↓mRNA of IL-1β, IL-6, TNF-α, COX-2, MCP-1, iNOS, TLR4, NLRP3, MAPK, NF-κB ↑T-Nrf2 protein ↑mRNA of GSR, GSTA4, HO-1	[11, 58, 59]
		Anti-diabetic	↓Glucose Inhibits α-glucosidase ↑Insulin ↑GCK levels ↑Glycogen synthesis ↑Phosphorylation of AMPK and ACC in adipose tissue, liver, and muscles	[60]

(Continued)

Metabolite	Name	Activity	Mechanism of Action	Reference
Phytosterols	Peruvioses A, B, C, D, F	Anti-diabetic	Inhibits α-amylase	[11, 61]
			↓Inflammatory markers (TNF-α, IL-5)	
			↓NF-κB translocation to the nucleus	
		Antioxidant, anti-inflammatory	Inhibits MPO	[11, 62]
			↓NO	
	β-sitosterol		↑IL-10	
			↓Glucose	
			↓HbA1c	
		Anti-diabetic	↑Insulin	[62]
			↑Activity of glucose-6-phosphate dehydrogenase and phosphohexose isomerase	
			↓Expression of TNF-α, IL-6, IL-1β, iNOS, COX-2	
		Antioxidant, anti-inflammatory	↓NO	[11, 63]
			↑IL-10	
			↓Glucose	
	Stigmasterol		↓HbA1c	
		Anti-diabetic	↑Insulin	[62]
			Normalizes levels of gluconeogenic enzymes (glucose-6-phosphatase, fructose-1,6-bisphosphatase, and lactate dehydrogenase)	
Terpenes			↓Inflammatory markers (IL-1, IL-2, IL-6, IFN-γ, TNF-α)	
		Antioxidant, anti-inflammatory	Suppresses levels of inflammatory gene and protein expression through the TLR4/MyD88/NF-κB P65 signaling pathway	[11, 64]
			↓ROS	
	Lupeol		Activates Nrf2	
			↓Glucose	
		Anti-diabetic	↑Insulin	[64]
			↑GLUT-4 translocation	
			↓Inflammatory markers (IL-1β, IL-1, IL-6, IL-8, COX-2, TNF-α)	
			↓ ROS	
		Antioxidant, anti-inflammatory	↓ iNOS	[11, 65]
			↓ NF-κB	
	Linalool		↑ SOD, CAT, GPx	
			↓ Glucose	
		Anti-diabetic	↑ Insulin	[66]
			↑ Hepatic glycogen	
Withanolides	W4, W5, W8, W36, W39, W59, W60, W61	Anti-inflammatory, antioxidant, anti-obesity	The exact mechanisms through which these compounds exert their therapeutic activity are not yet fully understood.	[67]

IL: interleukin; IFN-γ: interferon-gamma; NF-κB: nuclear factor-κB; TNF-α: tumor necrosis factor alpha; NLRP3: nod-like receptor protein 3; COX-2: cyclooxygenase-2; PGE2: prostaglandin E 2; NO: nitric oxide; LDL: low-density lipoprotein; HDL: high-density lipoprotein; VLDL: very-low-density lipoprotein; IDL: intermediate-density lipoprotein; MPO: myeloperoxidase; ROS: reactive oxygen species; iNOS: inducible nitric oxide synthase; SOD: superoxide dismutase; CAT: catalase; GR: glutathione reductase; GPx: glutathione peroxidase; GLUT: glucose transporter; LOX: lipoxygenase; PKB: protein kinase B; IRS: insulin receptor substrate; BAT: brown adipose tissue; MCP-1: monocyte chemoattractant protein-1; MAPK: mitogen-activated protein kinase; GSR: glutathione-disulfide reductase; GSTA4: glutathione S-transferase 4; HO-1: heme oxygenase-1; GCK: glucokinase; AMP: activated protein kinase; ACC: acetil-CoA carboxilasa; HbA1c: glycated hemoglobin.

enzymes, including glucose-6-phosphatase, fructose-1,6-bisphosphatase, and lactate dehydrogenase, contributing to improved glycemic control (Table 3 for further details).

Carotenoids and certain flavonoids, including quercetin, rutin, and myricetin, have also attracted attention for their notable antihyperlipidemic and anti-obesity effects. These bioactive compounds reduce cholesterol, LDL, very-low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), triglyceride, and triacylglycerol levels, while downregulating PPARγ expression. Simultaneously, they enhance HDL levels and promote increased energy expenditure. Notably, these compounds contribute to weight loss and activate brown adipose tissue, thereby amplifying their impact on lipid metabolism (Table 3 for further details).

Table 4. Functional food prototypes based on CP.

Formulation	Preparation method	Observations	References
Minimally processed gooseberries fortified with vitamin E	Vacuum impregnation	- The method was carried out using an isotonic sucrose solution at 20°Brix. - The firmness and color of the fruit experienced alterations. - The vitamin E content exceeded the recommended daily intake (RDI) levels. - A shelf life of 15 days was set.	[68]
Minimally processed gooseberry inoculated with *Lactobacillus plantarum*	Vacuum impregnation	- An isotonic glucose solution at 14% w/w concentration was used. - Fresh gooseberries with characteristics of probiotic food were obtained.	[41]
Gooseberry pulp fortified with oligofructose calcium and vitamin C	Fortified pulp	- The method promoted an increase in vitamin C and calcium levels. - The fortified pulp exhibited acceptable sensory characteristics.	[69]
Gooseberries fortified with vitamin C, vitamin D3, vitamin B9, and vitamin E	Vacuum impregnation	- The method was applied using an aqueous phase emulsion with sucrose. - Vacuum impregnation did not result in a significant alteration of the physicochemical characteristics of the fruit. - Changes in color and texture were observed.	[70]
Gooseberry powder fortified with vitamin C, iron, folic acid, textured soy protein, and fiber	Spray drying	- Maltodextrin was incorporated into the formulation. - The resultant powder exhibited low hygroscopicity, high solubility, and elevated levels of added active compounds. - The process of spray drying has been shown to minimize the loss of compounds during the drying stage.	[71]
CP waste powder (seeds, skin, pulp parts)	Dehydration and crushing	- The analysis revealed a high content of fiber, potassium, sodium, and phosphorus. - The resultant powder was deemed an ideal additive for bakery products and cereal-based snacks due to its optimal techno-functional characteristics.	[72]
CP nectar	Nectar preparation	- The beverage contains 8% sucrose. - It has been identified as a source of provitamin A and vitamin C. - It possesses antioxidant properties, with β-carotene being one of its key compounds.	[73]
Enriched beverage with agraz pulp (*Vaccinium meridionale* Swartz) and CP (*Physalis peruviana* L.)	Preparation of a beverage from a hydrolyzed whey permeate	- The stabilizers have been demonstrated to enhance the sensory presentation of the beverage. - Sensory tests indicated a general acceptance of over 80%. - There were losses of 4% in carotenoids and 17% in total phenols by the end of the storage period.	[74]
CP compote and jams	CP compote Traditional CP jam Suggested CP jam Mixed CP jam	- Most of the quality characteristics and attributes remained stable for 32 days. - The suggested CP jam showed a higher total phenolic and flavonoid content compared to the fresh fruit. - The traditional CP jam exhibited the lowest phenolic and flavonoid content. - The processed products were ranked in descending order of functionality as follows: suggested jam, compote, traditional jam, mixed jam, fresh fruit.	[75]

RDI: Reference Daily Intake; w/w: Weight per weight.

3.2.3. Development and processing of functional foods based on cape gooseberry: a supporting role

To promote the inclusion of CP in the diet of individuals with diabetes and dyslipidemia, it is not enough to provide scientific evidence supporting its health benefits in both patients and healthy populations. It is also essential to improve the shelf life of the fruit and develop multiple formulations that retain its bioactive and medicinal properties.

CP is a highly perishable fruit with a short post-harvest shelf life, requiring rapid commercialization [34]. Due to this limitation, various preservation and processing methods have been evaluated [35].

The implementation of these strategies should not only aim to extend shelf life but also to broaden the market reach, facilitating access to a wider consumer base and enabling international commercialization. Among the methods developed to preserve CP are blanching and osmotic dehydration [36], hot air drying [37], high hydrostatic pressure [38], combined with heat dehydration and freeze-drying [39,40]. These methods offer specific advantages and limitations depending on their ability to preserve the functional, nutritional, and sensory properties of the fruit.

Techniques such as hot air drying, high hydrostatic pressure, freeze-drying, and convection–infrared drying have been identified as viable alternatives for maintaining the bioactive compounds in CP, particularly phenolics, which are closely associated with the antioxidant and anti-inflammatory activity of the fruit [36–40]. Thus, CP preserved using these techniques may continue to provide health benefits to individuals with diabetes and obesity. Nevertheless, further preclinical and clinical trials are required to assess the effects of preserved CP on biomarkers related to oxidative stress, inflammation, diabetes, and dyslipidemia.

It is important to emphasize that in methods such as hot air drying and freeze-drying, standardizing processing temperature and time is critical to optimizing outcomes and minimizing alterations in nutritional quality, sensory attributes, and bioactive compound content [37,39,40].

Finally, although osmotic dehydration effectively preserves certain nutritional components, its use is contraindicated in people with diabetes and obesity due to the addition of an 80° Brix sucrose syrup, making it unsuitable for these populations [36]. In contrast, processing strategies that increase the concentration of beneficial compounds, such as spray drying optimized to preserve carotenoids and flavonoids, could contribute to the formulation of functional foods with targeted metabolic effects.

Despite the limitations of some of the preservation methods, several researchers have proposed that these techniques could serve as a foundation for the development of functional foods derived from CP. In recent years, a number of these methods have been explored and applied in the formulation of various CP-based products, as shown in Table 4. However, many of these formulations contain added sugars, such as sucrose, glucose, and maltodextrin, which contradict their intended purpose, as they may be harmful to individuals with diabetes and metabolic disorders.

Conversely, formulations designed specifically for patients with diabetes and lipid profile disorders have focused on nutritional enhancement through the enrichment of vitamins and minerals. While such products may contribute to overall health, they often fail to go beyond meeting basic nutritional needs. One notable exception is the formulation incorporating *Lactobacillus plantarum*, which has demonstrated probiotic properties [41].

Prospective evaluations of the long-term effects will be crucial to determine the actual functional potential of CP-based formulations. Specifically, it will be necessary to assess their impact on key biomarkers associated with oxidative stress, inflammation, diabetes, and dyslipidemia. These studies will provide essential evidence to support the rational development of functional foods targeted at individuals with metabolic disorders.

4. CONCLUSION

The findings gathered and analyzed in this review highlight the promising potential of CP as a functional food for managing metabolic syndrome. Preclinical studies consistently report significant improvements in glycemic control, lipid metabolism, and inflammatory status. These effects are primarily attributed to the presence of bioactive compounds such as carotenoids, flavonoids, phenolic acids, phytosterols, and terpenes. These compounds have been shown to modulate key signaling pathways, inhibit pro-inflammatory enzymes, enhance antioxidant defenses, stimulate glucose uptake, and reduce lipid accumulation. Such mechanisms support the development of CP-based functional food products, which could improve market accessibility, increase consumer interest, and contribute to broader public health strategies.

Nonetheless, despite the strength and consistency of preclinical evidence, clinical validation is still limited. Current human studies are scarce and affected by methodological limitations. Therefore, well-designed randomized controlled trials are urgently needed to confirm the therapeutic efficacy of CP and to support its inclusion in evidence-based nutritional interventions aimed at preventing and managing metabolic disorders.

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6. AUTHOR CONTRIBUTIONS

All authors made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; took part in drafting the article or revising it critically for important intellectual content; agreed to submit to the current journal; gave final approval of the version to be published; and agree to be accountable for all aspects of the work. All the authors are eligible to be an author as per the International Committee of Medical Journal Editors (ICMJE) requirements/guidelines.

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8. CONFLICTS OF INTEREST

The authors report no financial or any other conflicts of interest in this work.

9. ETHICAL APPROVALS

This study does not involve experiments on animals or human subjects.

10. DATA AVAILABILITY

All data generated and analyzed are included in this research article.

11. PUBLISHER'S NOTE

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ChatGPT (OpenAI, San Francisco, CA) was used as a language assistance tool during the preparation and editing of the manuscript. The tool was exclusively applied for grammar refinement and clarity improvements. The scientific content, data interpretation, and conclusions are entirely the work of the authors.

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