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Unlocking the Therapeutic Potential of *Momordica charantia* Linn.: From Traditional Use to Modern Drug Discovery

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Abstract

Momordica charantia Linn., commonly known as bitter melon or bitter gourd, is an important medicinal plant extensively used in traditional Asian medical systems for the prevention and management of various diseases. This review aims to compile and critically summarise updated information on its phytochemistry, pharmacological activities, toxicological profile, ethnomedicinal uses, and commercial and phytopharmaceutical relevance to support future research and drug development. A comprehensive literature survey was conducted using English-language publications, focusing on pharmacological evaluations, isolation and characterisation of bioactive compounds, toxicological studies, and both traditional and modern applications of *M. charantia*. Pharmacological investigations have demonstrated a wide range of bioactivities, including hepatoprotective, cardioprotective, and anti-inflammatory effects, as well as other therapeutic effects, from extracts of different plant parts. Numerous bioactive constituents have been identified, such as triterpenes, proteins, steroids, alkaloids, lipids, inorganic components, and phenolic compounds. Several novel molecules, including kuguacin-J, momordicine-I, α-eleostearic acid, momordicoside A, quercetin-3-O-glycoside, cucurbitacin E, and goyaglycoside-G, exhibit significant therapeutic potential. The review also highlights traditional and contemporary uses, phytopharmaceutical formulations, toxicity considerations, and other applications. Despite its long-standing traditional use, many therapeutic claims remain scientifically unvalidated, emphasising the need for further mechanistic and clinical studies to identify active principles and their synergistic effects.

Major Findings: *M. charantia* contains bioactive compounds like triterpenoids, saponins, flavonoids, and peptides with diverse pharmacological effects. Studies show antidiabetic, anticancer, hepatoprotective, cardioprotective, anti-inflammatory, and antioxidant activities, but further mechanistic studies and clinical trials are needed for validation and standardisation.

Keywords: *Momordica charantia* Linn., Pharmacological Activity, Phytochemistry, Phytopharmaceutical Formulation, Toxicity, Traditional Medicinal Uses

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1. Introduction

Momordica charantia Linn. is a widely cultivated vegetable crop belonging to the Cucurbitaceae family, commonly known as the cucumber, gourd, melon, or pumpkin family. These medium-sized plants thrive in warm regions worldwide and are characterised by their distinct bitter taste, attributed to alkaloid phytochemicals, along with a broad spectrum of medicinal properties[1]. The genus *Momordica* includes

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47 species in Africa and 12 species in Asia and Australia[2]. *M. charantia* grows naturally in tropical and subtropical areas of Africa, Asia, the Americas, and the Caribbean, and is also extensively cultivated[3]. It serves as an important commercial vegetable in many countries with tropical or subtropical climates and continues to be valued for its traditional medicinal uses[4]. Historically, bitter melon has been used as an herbal remedy for diabetes across various cultures, and it remains integral to diabetes prevention and management in many developing nations[5]. Extracts from its fruits, vines, leaves, and roots have been traditionally employed to relieve toothache, diarrhoea, and furuncles, and to treat conditions such as dysmenorrhea, eczema, gout, jaundice, leprosy, piles, pneumonia, psoriasis, rheumatism, and scabies[6]. Extensive research has documented a wide range of biological activities of *M. charantia*, including hypoglycaemic, antibacterial, antiviral, antitumour, immunomodulatory, antioxidant, antidiabetic, anthelmintic, antimutagenic, antilipolytic, antifertility, hepatoprotective, and anti-ulcerogenic effects[7]. These pharmacological properties arise from its rich and diverse chemical composition, which includes tannins, terpenoids, carbohydrates, resins, saponins, flavonoids, sterols, phylobatamins, anthraquinones, glycosides, amino acids, fatty acids, and phenolic compounds[8]. A variety of phyto-formulations have been developed using *M. charantia*, such as polyherbal formulations, tablets, capsules, suspensions, nanoparticles, and gels[9]. This review aims to provide an updated overview of the phytochemical profile, pharmacological activities, toxicological aspects, and ethnomedicinal significance of *M. charantia*.

2. Botanical Description and Distribution

2.1 Taxonomy and Botanical Classification

The plant was first classified by Linnaeus in 1753[10] and belongs to the Kingdom: Plantae; Division: Magnoliophyta; Class: Magnoliopsida; Order: Cucurbitales; Family: Cucurbitaceae; Genus: Momordica; Species: *M. charantia*[11]. The genus *Momordica* comprises approximately 47 species native to Africa and another 12 species distributed across Asia and Australia[12]. Botanically, the genus name *Momordica*, derived from the Latin word *momordere,* meaning "to bite," alludes to the irregularly toothed or jagged margins of the leaves, which appear as though they have been bitten[13].

2.2 Morphological Characteristics

Momordica charantia is a monoecious, slender, tendril-bearing annual climbing vine, reaching 2-4 meters in height. The plant is distinguished by its deeply lobed, serrated leaves, giving a "bitten" appearance. Leaves are simple, alternate, 3-7-lobed, measuring 4-12 cm across, with broad lobes often terminating in pointed tips and lacking stipules. Flowers are unisexual, actinomorphic, and yellow, with male and female flowers borne separately[14]. The fruit exhibits considerable morphological variation, including discoid, ovoid, ellipsoid, and oblong shapes, often pointed at the ends[15]. Typically 2-10 cm long, the fruits possess a warty exterior and a hollow interior lined by a thin layer of flesh, enclosing flattened seeds embedded in pith[16]. Immature fruits are whitish or pale green, maturing to shades of light to dark green, and turning orange-yellow upon full ripening within 45-80 days. Seeds are 8-15 mm long, straw-coloured, and encased in fleshy pulp, white in unripe fruits and red when mature[17]. In India, fruits are characteristically narrow with pointed ends and jagged, triangular ridges, and are renowned for their distinctly strong bitter taste[16].

2.3 Geographical Distribution and Cultivation Practices

Momordica charantia is believed to have originated in the tropical regions of the ancient world. It is widely cultivated as both a food and medicinal plant across India and other parts of the Indian subcontinent, as well as in Southeast Asia, China, Africa, the Caribbean, and South America[18]. *M. charantia* is an herbaceous perennial vine native to India and can be grown at elevations up to 1500 meters. Cultivation is typically carried out by sowing seeds in pits during the warm season, from April to July. The soil is enriched with manure, and seeds are planted at a spacing of about half a meter. After germination, only one healthy seedling is retained per pit, and watering is done once or twice a week. The plants begin to flower about 30-35 days after sowing, and fruits become ready for harvest approximately 15-20 days after flowering[19].

3. Traditional and Ethnomedicinal Uses

For centuries, *M. charantia* has been extensively used across various Asian traditional medicine systems for the prevention and treatment of numerous ailments[20]. The fruits and leaves of *Momordica* species are abundant in phytochemicals that contribute to their nutritional and nutraceutical value, offering diverse health-promoting effects. Recognised since ancient times, this plant has played a vital role in traditional and folk medicine systems[21], being employed for managing conditions such as Type 2 Diabetes Mellitus (T2DM), hypertension, obesity, cancer, bacterial and viral infections, and even AIDS[22]. Traditional Ayurvedic formulations, particularly for metabolic disorders such as *Prameha* (diabetes) and dyslipidemia, continue to incorporate *M. charantia* based on classical descriptions of its bitter taste (*tikta*), light quality (*laghu*), and warming potency (*ushna virya*), which are believed to underlie its therapeutic effects in metabolic homeostasis[23].

In Ayurveda, *M. charantia* (bitter melon) has been utilised for millennia, with seeds, roots, leaves, and particularly unripe fruits recognised for their pharmacological significance[24]. Traditionally, the fruits are employed in managing asthma, colic, constipation, cough, diabetes, fever (including malaria), gout, helminthiasis, inflammation, leprosy, skin disorders, ulcers, and wounds. Leaf juice is used for piles, as a bitter tonic and blood purifier, and to treat boils and other dermatological conditions, while also supporting liver health and preventing hepatic diseases[25,26]. Leaves are further applied in managing menstrual irregularities, burning sensations, constipation, fever, colic, infections, parasitic infestations, measles, hepatitis, and helminthiasis[27]. In Guyanese traditional medicine, leaf tea is consumed to regulate blood glucose, promote menstruation, relieve intestinal gas, and as an antiviral for measles, hepatitis, and fever, with both internal and external applications for sores, wounds, infections, worms, and parasites[28]. Seeds are traditionally used for ulcers, liver and spleen disorders, diabetes, hypercholesterolemia, flatulence, and wound healing, while roots address syphilis, rheumatism, boils, ulcers, septic swellings, ophthalmia, and vaginal prolapse. Bitter melon juice is also believed to relieve pyorrhea[29].

In Turkish folk medicine, oil from ripe *M. charantia* fruits macerated in olive oil with honey is used to prevent and heal gastric ulcers[30]. In African traditional practices, bitter melon treats worm infestations, inflammation, fever, menstrual disorders, syphilis, rheumatism, and skin diseases, with leaf decoctions used for diabetes, jaundice, liver ailments, ulcers, and burns. It is also prescribed for gonorrhea, measles, chickenpox, scabies, and malaria. In Caribbean medicine, leaf decoctions or fruit juice manage diabetes, hypertension, womb infections, malaria, dysentery, and parasitic diseases, while leaf baths are employed to alleviate rheumatism[21].

4. Phytochemistry of *M. charantia*

Momordica charantia is a nutrient-dense plant that has a diverse range of therapeutic chemicals. Triterpene, protein, steroid, alkaloid, inorganic, lipid, and phenolic chemicals are the primary elements of *M. charantia*[31]. Saponins in *M. charantia* are broadly categorised into two major groups: oleanane and cucurbitane-type triterpenoids[32]. To date, more than 100 saponins have been isolated and characterised from the fruit, stem, and leaves of the plant. Among these, bittersweet, a natural cucurbitane triterpenoid found in the fruit, leaves, and seeds, has demonstrated notable hypoglycemic potential[33]. Several key saponins, including momordicoside L, 3β,7β,25-trihydroxycucurbita-5,23(E)-dien-19-al, momordicoside K, momordicine I, momordicoside I, momordicoside F2, and momordicoside F1, have been identified using UPLC-ESI-MS/MS[34]. Furthermore, momordicoside U (Namsa N D et al., 2011), kuguaglycoside G[35], 3-hydroxycucurbita-5, 24-dien-19-al-7, 23-di-*O*-*β*-glucopyranoside[36], momordicine I[37], 3ß, 7ß, 25-tri-hydroxycucurbita-5, 23 (*E*)-dien-19-al[38], momordicine II[39] had also been isolated. More than 30 structurally distinct volatile constituents have been identified from the flowers of *M. charantia*, with laurene being the predominant component, followed by methyl jasmonate and 1-octadecanol[40].

Momordica charantia consists of the several chemical constituents, including alkaloids, gentilic acid, guanylate cyclase inhibitors, gypsogenin, hydroxytryptamines, karounidiols, lanosterol, lauric acid, linoleic acid, linolenic acid, momorcharasides,

momorcharins, momordenol, momordicillin, momordicinin, momordicosides, momordin, momordolo, multiflorenol, myristic acid, nerolidol, oleanolic acid, oleic acid, oxalic acid, pentadecans, peptides, petroselinic acid, polypeptides, proteins, ribosome-inactivating proteins, rosmarinic acid, rubixanthin, spinasterol, steroidal glycosides, stigmastadiols, stigmasterol, taraxerol, trehalose, trypsin inhibitors, uracil, vacine, v-insuline, verbascoside, vicine, zeatin, zeatinriboside, zeaxanthin, zeaxanthin, amino acids, aspartic acid, serine, glutamic acid, thscinne, alanine, g-amino butyric acid and pipecolic acid, ascorbigen, bsistosterol-d-glucicide, citruline, elasterol, flavochrome, lutein, lycopene, pipecolic acid[41,42]. The fruits and leaves are also rich sources of B-complex vitamins, providing thiamine (B1), riboflavin (B2), niacin (B3, 2%), vitamin B6 (3%), and folate (B9, 13%)[43,44]. The seeds of *M. charantia* are notable for their high lipid content, particularly polyunsaturated fatty acids (approximately 45% by weight) and conjugated linolenic acid (63-68%, predominantly as eleostearic acid), making them one of the few natural sources of this fatty acid. Essential oil extracted from dried seeds contains sesquiterpenes, phenylpropanoids, and monoterpenes, along with beneficial compounds such as tocopherols and polyphenols[45].

5. Pharmacological Activities

Recent pharmacological studies using cell lines, microbial assays, and animal models of human diseases have demonstrated that various parts and extracts of *M. charantia* possess a broad spectrum of bioactivities.

5.1 Anticancer Activity

Momordica charantia has demonstrated anticancer potential across multiple preclinical models, with several extracts and isolated constituents exhibiting selective cytotoxicity toward malignant cells while sparing non-malignant counterparts. Bioactive components such as saponins, Kuguacin-J, Momordicine-I, and α-Eleostearic acid-rich seed oil have shown growth-inhibitory effects against liver, breast, colon, lung, leukaemia, and head and neck cancer models. In hepatocellular carcinoma (HepG2) cells, *M. charantia* extract enhanced the anticancer efficacy of rosuvastatin through additive cytotoxic interactions, accompanied by apoptosis induction, modulation of oxidative stress, and alterations in lipid metabolism[46]. Ethanolic fruit extracts and Kuguacin-J significantly reduced viability and increased caspase-3 activation in breast cancer cells (MCF-7 and MDA-MB-231), while displaying minimal toxicity toward normal mammary epithelial cells and potentiating cisplatin activity[47]. Momordicine-I effectively suppressed tumour growth in head and neck cancer xenograft models by inhibiting c-Met/STAT3 signalling and downstream oncogenic regulators, including c-Myc and cyclin D1, without apparent systemic toxicity[48]. Seed oil enriched in α-eleostearic acid attenuated hepatocellular carcinoma progression in rats through modulation of tumour-related gene expression[49]. Additionally, seed extracts exhibited greater potency than pericarp fractions against colorectal cancer (HT-29) cells[50]. Recently, bitter-melon-derived extracellular vesicles have emerged as novel anticancer entities, induced ROS-mediated mitochondrial dysfunction and significantly inhibiting breast tumour growth *in vivo* with negligible toxicity[51].

5.2 Antidiabetic Activity

Methanolic and aqueous extracts markedly reduce fasting and postprandial hyperglycaemia, accompanied by improvements in body weight, haemoglobin, lipid profile, and endogenous antioxidant defences. Methanolic extracts generally exhibit superior glucose-lowering efficacy, comparable to standard antidiabetic drugs[52]. Mechanistically, *M. charantia* normalises elevated serum triglycerides and cholesterol, restores hepatic glycogen, enhances hexokinase activity, and inhibits intestinal α-glucosidase, thereby improving glucose utilisation and metabolic homeostasis. Saponin-rich fractions further enhance antihyperglycemic activity by increasing circulating C-peptide levels, reinforcing antioxidant enzyme systems, correcting dyslipidaemia, and promoting pancreatic β-cell repair[53]. Recent *in silico* studies have identified bioactive peptides and triterpenoids with high affinity for key molecular targets involved in glucose regulation, including the insulin receptor, SGLT1, DPP-IV, and GLUT2, exhibiting favourable drug-likeness and ADMET properties[54].

5.3 Antimalarial Activity

In *Plasmodium berghei*-infected mice, aqueous fruit extracts (50-100 mg/kg) produced dose-dependent chemo suppression exceeding 60%, with co-administration alongside chloroquine further enhancing parasite clearance and prolonging survival[55]. Commercial fruit extracts similarly demonstrated significant *in vivo* antimalarial activity, with dose-related parasite suppression and improved median survival, highlighting the plant's potential as a low-cost, accessible therapeutic resource[56]. Beyond direct parasite inhibition, *M. charantia* modulates host immune responses. *In vitro*, extracts suppressed *Plasmodium falciparum* 3D7 growth with a favourable selectivity index (>10), while *in vivo* treatment enhanced Akt and GSK3β phosphorylation, suppressed NF-κB activation, decreased pro-inflammatory cytokines (IFN-γ, TNF-α), and elevated anti-inflammatory IL-10, indicating regulation of malaria-associated inflammatory pathways[57]. Stem-derived methanolic extracts and fractions achieved 80-91% chemo suppression at 500 mg/kg, with the ethyl acetate fraction exhibiting efficacy comparable to chloroquine and arteether[58].

5.4 Hepatoprotective Activity

In streptozotocin-induced diabetic rats, oral administration of fruit extract (200 mg/kg) formulated with cow urine normalised elevated hepatic markers, including AST, ALT, cholesterol, and triglycerides, while enhancing endogenous antioxidant enzymes such as SOD, GPx, and CAT. Histopathological analyses confirmed regeneration of hepatic architecture, indicating superior protection compared with glibenclamide[59]. In acetaminophen-induced hepatotoxicity, *M. charantia* extract reduced serum ALT, AST, ALP, and LDH, with prophylactic treatment providing greater protection against enzyme elevation[60]. Aqueous leaf extracts (200-400 mg/kg) attenuated CCl4-induced hepatic damage by normalising liver weight, haematological indices, serum transaminases, bilirubin, and total protein, while reducing oxidative stress markers, including malondialdehyde, and enhancing SOD, CAT, and GSH activity[61]. Hydroalcoholic extracts (100-200 mg/kg) similarly mitigated SGOT, SGPT, ALP, and bilirubin elevations, with histology confirming reduced necrosis and vacuolar degeneration[62]. Solvent fractions of methanolic extracts also improved liver function and corrected dyslipidaemia in alloxan-diabetic rats[63]. Comparative studies demonstrate hepatoprotective efficacy comparable to standard drugs such as Liv-52[64], highlighting *M. charantia* as a promising natural hepatoprotective agent mediated primarily through antioxidant and cytoprotective mechanisms.

5.5 Nephroprotective Activity

Momordica charantia demonstrates nephroprotective potential through both molecular and physiological mechanisms. D-Galacturonic Acid (DGA), a major phytoconstituent, has been shown to interact with key residues of caspase-3 (GLU124, LYS137, ARG164), suggesting inhibition of apoptosis in renal cells[65]. Oral administration of the extract (250 mg/kg) significantly reduced serum urea and creatinine, normalised hepatic and renal biomarkers (ALT, AST, ALP, bilirubin), improved glycaemic control, and supported body weight maintenance. Histopathological analyses confirmed restoration of glomerular and tubular architecture, indicating mitigation of renal damage. Notably, co-administration with *Citrullus colocynthis* produced enhanced protective effects, reflecting potential synergistic interactions in traditional polyherbal formulations and highlighting *M. charantia* as a promising candidate for the prevention and management of renal injury[66].

5.6 Cardioprotective Activity

Extracellular vesicle-like nanovesicles derived from *M. charantia* (MCELNs) were efficiently internalised by cardiomyocytes, promoting proliferation, suppressing apoptosis, attenuating mitochondrial reactive oxygen species, stabilising mitochondrial membrane potential, and reducing DNA damage. In irradiated mice, MCELNs markedly ameliorated myocardial injury and fibrosis while restoring phosphorylation of ROS-associated signalling pathways, including AKT and ERK, highlighting their therapeutic potential in radiation-induced cardiac toxicity[67]. In isoproterenol-induced myocardial infarction, fruit extracts, particularly in combination with *Spirulina platensis*, reduced serum cardiac injury markers (LDH, CK-MB, CK, SGOT) and preserved myocardial membrane integrity, with bioactive constituents such as Momordicoside A

contributing to synergistic effects[68]. Aqueous fruit extracts (250 mg/kg) normalised cardiac biomarkers, reduced lipid peroxidation, enhanced glutathione-mediated antioxidant defences, and mitigated fibrosis in offspring of diabetic dams[69]. Similarly, ethanolic extracts in cholesterol-fed rats lowered LDL cholesterol, reversed myocardial degeneration, aortic ulceration, and inflammatory infiltration, restoring cardiac and vascular integrity[70]. In acute myocardial infarction models, *M. charantia* (100-500 mg/kg) corrected ECG abnormalities, suppressed TNF-α, IL-6, and CRP, and upregulated cytoprotective mediators (HO-1, Nrf2, IL-10), collectively reducing cardiac injury[71].

5.7 Neuroprotective Activity

In aspartame-induced neurotoxicity, ethanolic fruit extracts effectively prevented spatial memory impairment and hippocampal neuronal degeneration, as evidenced by improved performance in the Morris water maze, increased neuronal density in the CA1-CA3 regions, reduced lipid peroxidation, enhanced glutathione peroxidase activity, and inhibition of acetylcholinesterase activity. Co-administration with *Curcuma longa* produced additive neuroprotective effects, suggesting synergistic interactions between *M. charantia* flavonoids and curcumin[72]. In cerebral ischemia-reperfusion models, *M. charantia*-derived exosome-like nanoparticles effectively crossed the blood-brain barrier and preferentially accumulated in ischemic regions, resulting in reduced infarct volume, neurological deficits, and blood-brain barrier disruption. These effects were mediated through suppression of matrix metalloproteinase-9, restoration of tight-junction proteins, attenuation of neuronal apoptosis, and activation of the AKT/GSK3β signalling pathway, with miRNA5266 identified as a key molecular regulator[73].

5.8 Anti-obesity Activity

In high-fat diet-fed animals, aqueous extracts (1-2%, w/v) administered via drinking water markedly reduced body-weight gain, feed efficiency, and adipose tissue accumulation. Notably, combined supplementation of *M. charantia* (1%) with *Chrysanthemum zawadskii* (1%) produced synergistic effects, resulting in pronounced reductions in retroperitoneal and inguinal white adipose tissue mass, suggesting enhanced regulation of adipose deposition[74]. Oral administration of aqueous leaf extract (200 mg/kg/day) further decreased body weight, lipid accumulation, and adipose tissue volume, while significantly improving glucose tolerance, insulin sensitivity, and serum lipid profiles. These metabolic benefits were mechanistically associated with downregulation of lipogenic genes and upregulation of genes involved in fatty acid oxidation, indicating coordinated modulation of hepatic and adipose lipid metabolism[75]. Consistent with these findings, hydroalcoholic fruit extracts significantly attenuated hyperglycaemia and dyslipidaemia in cafeteria diet-induced obesity models, outperforming several comparator plant extracts. Collectively, these results support the ethnopharmacological relevance of *M. charantia* as a modulator of energy balance and metabolic homeostasis, while highlighting the need for standardised preparations and clinical validation to confirm its anti-obesity potential[76].

5.9 Immunomodulatory Activity

In cyclophosphamide-induced immunosuppressed mice, *M. charantia* Polysaccharides (MCP) effectively restored immune function, as evidenced by increased thymus and spleen indices, enhanced macrophage phagocytic activity, and significantly elevated lymphocyte proliferation. MCP treatment also upregulated key immunostimulatory cytokines, including interferon-γ, interleukin-6, and interleukin-12, at both gene and protein levels, indicating coordinated activation of innate and adaptive immune responses and reversal of delayed-type hypersensitivity suppression[77]. Beyond polysaccharides, whole-fruit extracts of *M. charantia* have demonstrated immunoregulatory activity in infectious disease models. In *Schistosoma mansoni*-infected mice, extract administration improved immune homeostasis by increasing anti-inflammatory interleukin-10 levels, suppressing excessive interferon-γ responses, and expanding $CD4^+$ and $CD8^+$ T-cell populations. Notably, co-administration with praziquantel enhanced parasite clearance, reduced hepatic granuloma formation, and improved systemic antioxidant status, suggesting beneficial modulation of immune-mediated pathology alongside conventional therapy[78]. Collectively, these findings indicate that *M. charantia* can strengthen innate immune defences, regulate cytokine networks,

and support adaptive immune cell function. However, heterogeneity in extraction methods, structural variability of polysaccharides, and limited mechanistic and translational studies underscore the need for standardised preparations and deeper structure-activity investigations to substantiate its immunotherapeutic potential[79].

5.10 Wound Healing Activity

In vivo studies report significantly enhanced wound contraction, with extract-loaded nanofiber dressings achieving up to 94% wound closure, highlighting their promise as bioactive wound-care materials[80]. Topical application of 1% *M. charantia* gel and cream formulations further accelerated wound repair in murine models, with cream preparations showing superior wound contraction and histological restoration. These effects were associated with increased fibroblast proliferation, collagen deposition, and elastic fibre formation, likely mediated by the plant's antioxidant and antimicrobial constituents that support epithelialisation and modulate inflammatory responses[9]. Importantly, *M. charantia* also exhibits efficacy in diabetic wound models, promoting epithelial regeneration, angiogenesis, and fibroblast activity while attenuating chronic inflammation, suggesting potential utility as an adjunct therapy for impaired wound healing[81].

5.11 Antioxidant Activity

In vitro cell suspension culture studies have demonstrated that exposure to copper nanoparticles induces oxidative stress-mediated activation of antioxidant defences, resulting in elevated biosynthesis of phenolic and flavonoid compounds. Increased hydrogen peroxide and malondialdehyde levels stimulated both enzymatic (phenylalanine ammonia-lyase and polyphenol oxidase) and non-enzymatic antioxidant responses, leading to enhanced DPPH scavenging activity[82]. Complementary agronomic approaches have shown that foliar application of amino acids, particularly phenylalanine at 300 mg/L, significantly augments phenolic and flavonoid accumulation and improves antioxidant capacity, highlighting the influence of nutritional modulation on phytochemical quality[83]. Extraction optimisation studies consistently identify 80% ethanol as an efficient solvent for maximising antioxidant recovery from *M. charantia* fruits, yielding robust activity in DPPH and Ferric Reducing Antioxidant Power (FRAP) assays[84]. Metabolomics analyses using GC-MS and LC-MS attribute this activity to a diverse spectrum of compounds, including ascorbic acid, quercetin glycosides, Cucurbitane triterpenoids, fatty acids, and newly identified metabolites such as Brevifolincarboxylic acid[85,86]. Notably, *M. charantia*-derived extracellular vesicles exhibit potent antioxidant activity both *in vitro* and *in vivo* by upregulating endogenous antioxidant enzymes and attenuating oxidative and inflammatory stress, representing a novel antioxidant delivery modality[87].

5.12 Anti-inflammatory Activity

Momordica charantia exhibits significant anti-inflammatory activity mediated by diverse Cucurbitane-type triterpenoids and polyphenolic constituents. Kaguacidine A demonstrated moderate inhibition of Lipopolysaccharide (LPS)-induced nitric oxide production in RAW 264.7 macrophages, indicating suppression of pro-inflammatory mediator release[88]. Structurally related triterpenoids, including Momordicines I, II, IV, and TCD, displayed distinct cytotoxic and anti-inflammatory profiles, with TCD emerging as the most pharmacologically favourable candidate. Mechanistic studies revealed that TCD suppresses inflammatory signalling primarily through inhibition of the IKK/NF-κB pathway, while concomitantly activating cytoprotective Nrf2/HO-1 and ERK1/2-p38 MAPK pathways, suggesting a dual anti-inflammatory and antioxidant mode of action[89]. Aqueous extracts of *M. charantia* also exerted pronounced anti-inflammatory effects by inhibiting protein denaturation, heat-induced haemolysis, and proteinase activity, with these effects strongly correlated with total phenolic content, underscoring the role of polyphenols in mediating immunomodulatory responses[90]. In addition, several triterpene glycosides, including Karavilosides, Momordicosides, and Charantoside XV, significantly downregulated key inflammatory mediators such as IL-6, TNF-α, inducible nitric oxide synthase, and cyclooxygenase-2, with IL-6 showing the greatest suppression[91]. Methanolic leaf extracts further attenuated inflammation in cyanide-intoxicated rat models by reducing pro-inflammatory cytokine levels, restoring redox balance,

and limiting tissue damage[92]. Notably, Charantadiol A effectively suppressed *Porphyromonas gingivalis*-induced inflammatory responses *in vitro* and *in vivo*, highlighting potential utility in infection-associated inflammatory conditions[93].

5.13 Antiparasitic Activity

Comparative assessments of methanolic, aqueous, and dichloromethane extracts against *Haemonchus contortus* larvae demonstrated significant anthelmintic efficacy, with methanolic extracts of *M. charantia* showing among the highest inhibition of larval migration, comparable to other traditionally used species. However, the observed cytotoxicity of the crude methanolic extract underscores the necessity for further fractionation to isolate safer, bioactive constituents[94]. Four cucurbitacin-type triterpenoids were isolated, exhibiting strong leishmanicidal effects (IC_{50} = 2.11-3.25 µg/mL) alongside favourable selectivity indices (8.5-17.2). Notably, a cucurbitacin-enriched fraction demonstrated enhanced potency with minimal mammalian cytotoxicity, reinforcing its potential as a natural antileishmanial lead compound[95].

5.14 Antiviral Activity

Momordica charantia leaf extracts, compounds such as quercetin-3-galactopyranoside, rutin, and hyperin, demonstrated strong binding affinity to the SARS-CoV-2 main protease (Mpro), surpassing that of the standard antiviral agent Nirmatrelvir[96]. Additionally, fruit, seed, and pith extracts of *M. charantia* from diverse geographical sources displayed potent inhibition of HIV-1 reverse transcriptase. Notably, Cucurbitane glycosides such as Karavilosides K1 and K3 inhibited reverse transcriptase by 70-80%, with molecular docking analyses revealing affinities comparable to established antiretrovirals, including Rilpivirine[97].

5.15 Antimicrobial and Antibacterial Activity

Leaf extracts exhibit inhibitory effects against *Staphylococcus aureus*, methicillin-resistant *S. aureus* (MRSA), *Escherichia coli*, *Pseudomonas aeruginosa*, *Salmonella typhi*, *Proteus mirabilis*, and *Klebsiella pneumoniae*[98]. Among different preparations, methanolic and ethanolic extracts, rich in alkaloids, flavonoids, tannins, terpenoids, glycosides, and phenolic compounds, show the strongest antibacterial efficacy, reflected by low minimum inhibitory and bactericidal concentrations[99]. Ethyl acetate fractions enriched in glycosylated flavones exhibit pronounced activity against *K. pneumoniae*[100]. Notably, several extracts retain antibacterial potency under acidic and elevated temperature conditions, indicating physicochemical stability relevant to oral formulations[101]. Seed extracts further demonstrate significant antibacterial and anti-biofilm activities, supported by *in silico* docking analyses showing favourable interactions of seed-derived compounds with *S. aureus* biofilm-associated proteins[102]. In addition, green-synthesised zinc oxide and chromium oxide nanoparticles derived from *M. charantia* extracts exhibit enhanced antibacterial activity against *E. coli*, *S. aureus*, *P. aeruginosa*, *Bacillus subtilis*, and *Vibrio cholerae*, often surpassing conventional antibiotics at higher concentrations[103,104]. Methanolic extracts also inhibit *Leptospira interrogans*, suggesting potential adjunct applications in leptospirosis management[105].

6. Phytopharmaceutical Formulations

Plant extracts have long attracted interest in pharmaceutical development for improving formulation stability and patient compliance[106]. Accordingly, *M. charantia* has been formulated into various dosage forms, including oral tablets, capsules, and topical gels, tailored to specific therapeutic applications. An overview of reported *M. charantia*-based phyto-formulations is summarised in Table 1.

Although the majority of pharmacological investigations of *M. charantia* have been conducted in *in vitro* and animal models, several clinical and translational studies provide supportive evidence for its therapeutic potential, particularly in the management of metabolic disorders. Clinical investigations involving patients with type 2 diabetes have demonstrated that standardised bitter melon preparations can reduce fasting blood glucose levels by approximately 10-25% and improve glucose tolerance, supporting its traditional antidiabetic use. Moreover, systematic reviews and meta-analyses of polyphenol-rich plant extracts, including *M. charantia*, suggest moderate but clinically meaningful improvements in glycaemic control when used as complementary therapy. These findings indicate that the plant possesses clinically relevant bioactivity, although larger randomised controlled trials

are still required to establish standardised dosing and long-term safety profiles.

Recent formulation strategies have also explored polyherbal and nanoparticulate delivery systems to enhance the therapeutic performance of *M. charantia*. For example, optimised solid formulations combining *M. charantia* with other antidiabetic plants such as *Abelmoschus esculentus* have demonstrated enhanced synergistic hypoglycaemic activity, improving glucose-lowering efficacy compared with individual extracts. Similarly, nanoparticle-based formulations (*e.g.*, silver nanoparticles, solid lipid nanoparticles, and nanoliposomes) derived from *M. charantia* extracts have shown significantly improved antimicrobial, antioxidant, and drug-delivery properties due to enhanced bioavailability, stability, and targeted cellular uptake. These synergistic and nanotechnological approaches highlight promising strategies to bridge the gap between traditional phytotherapy and modern phytopharmaceutical development, although clinical validation of these advanced formulations remains limited.

7. Toxicological and Safety Evaluation

Extensive toxicological investigations have been conducted to delineate the safety profile and potential toxic thresholds of *M. charantia*. Acute toxicity assessments using zebrafish (*Danio rerio*) embryo models revealed concentration-dependent reductions in embryo survival, with notable variability between cultivars and extraction methods. Hot aqueous extracts of Chinese and Indian varieties exhibited LC_{50} values of 144.54 and 199.53 µg/mL, respectively, while cold aqueous extracts were comparatively less toxic (LC_{50} 251.19 µg/mL for the Chinese variety). The cold aqueous Indian extract showed the lowest toxicity, with no LC_{50} observed within the tested concentration range, highlighting the influence of extraction conditions on safety outcomes[107].

Table 1. Phyto-formulations containing *M. charantia* extract

Plant part utilised	Extract	Phyto-formulations	Polymers/excipients utilised	Application	References
Leaf	Milli-Q water	Silver nanoparticles	Silver nitrate	Antimicrobial and catalytic activity	111
Fruit	50% ethanol	Nanoliposomes	Lecithin, sunflower oil, deionised water, 2% glycerol	--	112
--	70% ethanol	Self- nanoemulsifying DDS	Snakehead fish powder, tween 20, span 80, glycerine, olive oil	--	113
Stem	Distilled water	Silver nanoparticles	Silver nitrate	Antimicrobial activity	114
Fruit	Ethanol	Effervescent granule	Citric acid, tartaric acid, sodium bicarbonate, gelatine, aspartame	--	115
Fruit	--	Tablet	Avicel pH 102, primojel, amprotab, lactose, talcum	--	116
Leaf	Deionised water	TiO_2 nanoparticles	Titanium tetrachloride	Antimalarial activity	31
--	--	Chewable lozenges	Candy base, lubricants, humectants, binders, whipping agents, acidulants	Antidiabetic activity	117
Fruit	Ultrapure water	Silver nanoparticles	Silver nitrate	--	118
Fruit	1-butyl-3-methylimidazolium tetrafluoroborate	Solid lipid nanoparticles	Glyceryl monostearate, span 80, capric caprylic triglyceride, tween 80	--	119

Table 1. Continued...

Plant part utilised	Extract	Phyto-formulations	Polymers/excipients utilised	Application	References
Leaf	Distilled water	Zinc oxide nanoparticles	Zinc sulphate heptahydrate	Antibacterial activity	120
Leaf	Chloroform	Mouth dissolving tablets	Microcrystalline cellulose, manetole, aspertine, croscarmellose sodium, crosspovidone	Anti-hyperglycaemic activity	121
Fruit peels	Distilled water	Silver nanoparticles	1 mM aqueous solution of silver nitrate	Helminthicide and larvicidal activity	122
Fruit	Ethanol	Microemulsion	Tween 20, sorbitan laurate, tween 80, sorbitan oleate, caprylic/capric triglyceride	Antioxidant and 5α-reductase inhibitory activity	123
Fruit	--	Exosome	--	Neuroprotective activity	73
Fruit	Distilled water	Silver nanoparticles	Silver nitrate	Antibacterial and antibiofilm activity	124
Fruit	Distilled water	Silver nanoparticles	Silver nitrate	Anthelmintic activity	125
Fruit	Deionised water	Zinc oxide nanoparticles	Zinc acetate dihydrate	Antibacterial and antioxidant activity	104
Fruit	Double-distilled water	Zinc oxide nanoparticles	Zinc acetate dihydrate, sodium hydroxide	Antibacterial activity	126
Fruit	80% ethanol	Phytosomes	Stigmasterol glucoside, ß-sitosterol glucoside, phosphatidylcholine	--	127
Fruit	80% ethanol	Silver Nanoparticles	Silver nitrate	Antioxidant and antibacterial activity	128
Fruit	70% ethanol	Herbal capsule	Magnesium carbonate, magnesium stearate, sodium hydrogen carbonate	--	129
Leaf	70% ethanol	Microemulsion	Tween 80, soybean oil, and water	Antibacterial activity	130
Fruit	Distilled water	Copper oxide nanomaterial	copper (ii) sulfate pentahydrate, sodium hydroxide	Antimicrobial activity	131
Fruit	--	Silver nanoparticles	Silver nitrate	α-Amylase inhibition activity	132
Leaf	Acetone	Gel	Carbopol, triethanolamine, liquid petrolatum, propylene glycol	Wound healing activity	133
Leaf	96% ethanol	Vanishing cream	Stearic acid, triethanolamine, glycerine, beeswax, cetaceum, liquid paraffin, propylene glycol, sorbitol, butylated hydroxytoluene	Skincare cream preparation	134

Sub-chronic toxicity studies in male albino mice receiving petroleum ether, chloroform, and ethanolic seed extracts (25 mg/100 g body weight, intraperitoneally for 48 days) demonstrated no mortality or behavioural abnormalities in the petroleum ether and ethanolic extract groups. In contrast, the chloroform extract induced mild weight loss, suggesting the presence of solvent-extractable toxic constituents[108]. Acute oral toxicity testing conducted according to OECD Guideline 423 showed that ethanolic fruit extracts administered to Sprague Dawley rats at doses up to 2000 mg/kg produced only mild, transient signs of toxicity without mortality or major behavioural alterations, indicating an LD_{50} exceeding 2000 mg/kg and a favourable safety margin at therapeutic doses[109].

Developmental toxicity studies further underscored tissue-specific safety differences. Zebrafish embryo assays revealed pronounced toxicity of seed extracts ($LD_{50} \approx 50$ µg/mL), inducing multiple developmental abnormalities even at sub-lethal concentrations. In contrast, fruit extracts were considerably safer up to 200 µg/mL, although signs of cardiac hypertrophy were observed, indicating potential cardiotoxic risk during early development[110].

Several studies also report the protective effects of *M. charantia* against chemically induced toxicities. In methotrexate-induced reproductive toxicity models, oral administration of fruit extract (600 mg/kg for 5 days) significantly mitigated testicular and epididymal damage, reduced oxidative stress and inflammatory biomarkers, and restored antioxidant enzyme activity, supporting its cytoprotective potential[135]. Similarly, leaf extracts demonstrated marked hepatoprotective activity against carbon tetrachloride induced liver injury in Wistar rats, with biochemical and histopathological improvements comparable to the reference drug Liv-52[63]. Nevertheless, caution is warranted for certain preparations. Methanolic extracts of seeds and whole plant exhibited dose-dependent toxicity in Wistar rats, with intraperitoneal LD_{50} values of 288 mg/kg and 714 mg/kg, respectively, and demonstrated significant uterotonic activity *in vitro*, indicative of abortifacient potential[136]. Furthermore, the D-galactose-specific seed lectin (MCL) displayed selective antitumour activity *in vitro* and *in vivo* but showed moderate cytotoxicity in brine shrimp assays (LC_{50} = 49.7 µg/mL), highlighting the need for careful dose optimisation and safety evaluation[137].

8. Challenges and Future Perspectives

Despite the growing body of literature describing the pharmacological potential of *M. charantia*, several methodological limitations should be carefully considered when interpreting the reported findings. One of the major challenges is the significant variability in phytochemical composition, which may arise from differences in plant cultivars, geographical origin, harvesting stage, and extraction techniques. Such variability can lead to inconsistencies in biological activity and complicate the standardisation of extracts for therapeutic use. In addition, many pharmacological studies rely primarily on *in vitro* experiments or small-scale animal models, often with limited sample sizes and heterogeneous experimental designs, which may reduce the statistical robustness and reproducibility of the findings. Another important limitation is the lack of quantitative meta-analyses and comparative studies that systematically evaluate the efficacy of different extracts, isolated compounds, or formulations. Furthermore, inconsistencies in dosing strategies, extract characterisation, and experimental endpoints across studies make it difficult to directly compare results and establish clear therapeutic benchmarks. Therefore, future research should prioritise standardised extraction protocols, well-powered *in vivo* studies, and rigorous clinical trials, along with systematic meta-analytical approaches, to generate more reliable and translatable evidence for the therapeutic applications of *M. charantia*.

9. Conclusion

Momordica charantia represents a pharmacologically versatile medicinal plant with substantial ethnopharmacological relevance and a well-documented diversity of bioactive constituents. Preclinical studies provide convincing evidence for its antidiabetic and multiple adjunct therapeutic effects, largely supporting its traditional applications. Phytochemical research has identified Cucurbitane-type triterpenoids and related secondary metabolites as key contributors to its bioactivity, while emerging formulation strategies have improved their pharmacological performance. Toxicological data suggest an acceptable safety profile for fruit-derived preparations at therapeutic doses, although

certain seed-derived and solvent-based extracts require careful safety evaluation. Notwithstanding extensive experimental evidence, clinical data remain limited and heterogeneous. Future investigations should prioritise standardised preparations, robust pharmacokinetic and toxicological assessments, and well-designed clinical trials to facilitate the rational translation of *M. charantia* into evidence-based phytotherapeutic applications

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Author Contribution

Ms. Ketaki Shinde and Dr. Trupti Durgawale jointly conceptualised and designed the study. Data collection was carried out by Ms. Vaishnavi Shedge and Mr. Omkar Tipugade, while Ms. Ketaki Shinde performed the data analysis and drafted the initial version of the manuscript. Dr. Durgawale also supervised the overall study, contributed to data interpretation, and provided critical revisions. All authors have read and agreed to the published version of the manuscript.