INTERNATIONAL JOURNAL OF PHARMACEUTICAL SCIENCES
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: https://www.ijpsjournal.com

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Review Article

Curcuma amada: Exploring ITS Phytochemical Diversity, Multifaceted Pharmacological Activities and Gastroprotective Mechanism -an Integrated Review.

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ARTICLE INFO

Published: 25 Nov 2026
Keywords:
Curcuma amada, mango ginger, intestinal barrier, tight junctions, gut microbiota, antioxidant, gastroprotection
DOI:
10.5281/zenodo.24525053

ABSTRACT

Curcuma amada Roxb. (mango ginger), a rhizomatous herb of the family Zingiberaceae, resembles ginger in appearance but carries the aroma of raw mango. It is used as a spice, a pickle ingredient and in Ayurvedic and Unani practice for digestive complaints, inflammation, skin disorders and fever. The rhizome contains curcuminoids, essential-oil terpenoids, phenolic acids, starch and many other constituents, and extracts have shown antioxidant, anti-inflammatory, antimicrobial and gastric-protective activity in experimental systems. Gut integrity depends on the mucus layer, a single layer of epithelium sealed by tight junctions, mucosal immunity and a balanced microbiota. Oxidative stress, NF-κB-driven inflammation and dysbiosis damage this barrier and allow bacterial products such as lipopolysaccharide (LPS) to enter the circulation. Strong evidence that curcumin, the best-studied curcuminoid, protects barrier function comes from Curcuma longa studies, and C. amada shares some of the same chemistry. Direct evidence for C. amada on epithelial permeability, tight-junction proteins or the microbiota is, however, still scarce. Most available data concern antioxidant assays, H+,K+-ATPase inhibition, Helicobacter pylori growth inhibition and ethanol- or aspirin-induced ulcer models. This review summarises the botany, phytochemistry and gut-relevant pharmacology of C. amada, separates direct evidence from extrapolation, notes safety considerations, and outlines the studies needed to establish whether mango ginger can support gut integrity.

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

INTRODUCTION

Curcuma amada Roxb., known as mango ginger, belongs to the family Zingiberaceae and is native to the Indo-Malayan region, with cultivation across the tropics of Asia [1,2]. The name "amada" reflects the raw-mango flavour of the rhizome. Traditional systems use it as an appetiser, carminative, antipyretic, expectorant and emollient, and for biliousness, itching, bronchitis, hiccough and inflammation from injuries [2,3]. A comprehensive review of the species recorded more than 130 constituents, with the rhizome rich in essential oils [1].

The intestinal barrier is a multilayered defence. The physical barrier is formed by the epithelium, the microbiota and the mucus secreted by goblet cells, and it works alongside immune and chemical components [4]. When this barrier is weakened, microbes and their products reach the lamina propria and activate resident immune cells, a process implicated in ulcerative colitis, Crohn's disease and metabolic disease [4,5]. Natural products that reduce oxidative stress, restrain NF-κB signalling and support the microbiota are therefore of interest as barrier-protective agents [4].

Most of the barrier-focused literature on the genus *Curcuma* concerns turmeric (*C. longa*) and its pigment curcumin [5–8]. Mango ginger is a related, culinary species whose gut-specific biology is far less documented. This review gathers what is known about C. amada, places it beside the curcumin evidence, and identifies what remains unproven.

BOTANICAL AND TAXONOMICAL DESCRIPTION OF CURCUMA AMADA

Taxonomical classifiication

Table 1: Taxonomical Classification of *C. Amada*

Rank	Name
Kingdom	Plantae
Phylum	Tracheophyta
Class	Liliopsida (monocots)
Order	Zingiberales
Family	Zingiberaceae

Table 2: Common Names of *C. Amada* in Different Languages [2]

Rank	Name
Genus	Curcuma L.
Species	Curcuma amada Roxb.

Common names

Language	Name
English	Mango ginger
Sanskrit	Amrardrakam, Karpuraharida
Hindi	Amahaldi
Malayalam	Mangainchi
Tamil	Mankayinci
Telugu	Mamidi allam

Habitat and distribution

The genus is widely distributed in the tropics of Asia, Africa and Australia and is thought to have originated in the Indo-Malayan region [2]. C. amada is cultivated in India (including Kerala, West Bengal and Odisha) and neighbouring countries. It grows best in moist but well-drained, fertile soil under warm, humid conditions [3,9].

Botanical description

C. amada is a perennial, rhizomatous herb. The rhizome is the economic part: it is branched, ginger-like in outline, pale yellow to cream inside, and gives off a distinct raw-mango smell. Aerial shoots bear a pseudostem of overlapping leaf sheaths and large lanceolate leaves, and the inflorescence is a spike with bracts. Starch granules make up a large share of the rhizome, and its morphological and biochemical profile differs from that of ginger [9,10]. *Authors should confirm the detailed morphology (dimensions, floral and anatomical characters) against a taxonomic flora or herbarium voucher before submission.*

PHYTOCHEMICAL COMPOSITION OF CURCUMA AMADA

The rhizome yields a wide range of compounds. The groups most relevant to gut health are listed below.

Curcuminoids. Curcumin, demethoxycurcumin and bisdemethoxycurcumin have been reported in acetone extracts of C. amada [2]. Their content is generally

described as much lower than in *C. longa*, which matters when reading any claim that borrows curcumin data for mango ginger.

Phenolic acids. Free phenolics from mango ginger are dominated by caffeic (about 26%), gentisic (24%), ferulic (20%), gallic (10%), cinnamic (7%) and protocatechuic (7%) acids, whereas bound phenolics are mainly ferulic (47%), cinnamic (29%), p-coumaric (11%) and syringic (5%) acids [11].

Essential oil and terpenoids. The rhizome is oil-rich, and the volatile fraction (monoterpenes and sesquiterpenes) gives the mango note [1,12].

Other constituents. Starch, sterols, a novel bioactive molecule with antimicrobial, antioxidant, cytotoxic and platelet-aggregation-inhibitory activity, and numerous minor compounds have been described [1,12,13].

INTESTINAL BARRIER INTEGRITY: A BRIEF FRAMEWORK

The barrier has four interacting layers: the mucus layer, the epithelium with its tight junctions (claudins, occludin and ZO-1), the mucosal immune system, and the microbiota with its metabolites [4]. Barrier failure raises permeability, and bacteria-derived LPS crossing into the blood is proposed as a driver of chronic low-grade inflammation and metabolic disease [6,7]. Experimental markers include histological colitis score, tight-junction protein expression, FITC-dextran flux, serum D-lactate and plasma LPS [5,6]. Oxidative stress, NF-κB and MAPK signalling, TLR4 and NLRP3 inflammasome activation, and dysbiosis are the main mechanistic targets of barrier-protective compounds [4,8,14].

PROTECTIVE MANIFESTATIONS OF CURCUMA AMADA RELEVANT TO GUT INTEGRITY

Antioxidant activity

Phenolic fractions of mango ginger scavenge free radicals, show reducing power, inhibit lipid peroxidation and protect DNA in vitro [11]. Different rhizome extracts have also been tested for antioxidant capacity in comparative studies [2,3]. Because reactive oxygen species damage epithelial cells and tight junctions, this activity is a plausible, but indirect, basis for barrier protection. It has not been shown in intestinal epithelial models for C. amada.

Anti-infilammatory activity

An ethanolic rhizome extract showed anti-inflammatory effects in acute and chronic models in albino rats [2], and reviews list inflammation among its traditional and experimental uses [3,9]. These data come from paw-oedema-type systems and not from intestinal inflammation, so applicability to colitis is unconfirmed.

Gastric protection and anti-ulcer activity

This is the area with the most direct gastrointestinal data. Phenolic fractions of C. amada inhibited gastric H+,K+-ATPase with IC50 values of 2.2 and 0.7 µg/mL for free and bound fractions, reported as [9–27] times more potent than lansoprazole in the same assay, and also inhibited H. pylori growth [11]. A postgraduate dissertation reported that fresh rhizome juice (2 mL/kg) had antiulcer effects in ethanol- and aspirin-induced ulcer models in rats without mortality in acute toxicity testing [15]; as an unpublished thesis this should be treated as preliminary. Related species support the concept of Curcuma-mediated mucosal protection, with *C. xanthorrhiza* leaf extract, *C. aromatica* water extract and *C. purpurascens* rhizome extract each protecting the gastric mucosa of rats against ethanol injury through antioxidant, mucus-preserving and prostaglandin-related effects [16–18].

These findings are for the stomach, not the small or large intestine.

Antimicrobial activity

Aqueous and organic extracts of mango ginger inhibited *Escherichia coli*, *Bacillus subtilis* and *Staphylococcus aureus* [2]. For the gut this cuts both ways: it may limit pathogens, but broad antimicrobial activity could also disturb commensals, and no study has examined the effect of C. amada on a complex microbiota.

Evidence borrowed from curcumin and *Cùìcùm Yoùg*

Because C. amada contains curcuminoids, curcumin studies indicate which barrier mechanisms deserve testing in mango ginger. They should not be read as evidence that C. amada behaves the same way.

- **Tight junctions and permeability.** In rat enteritis, curcumin protected mucosal barrier function by activating MKP-1 and reducing p38 and NF-κB activation [6]. Curcumin is also proposed to lower plasma LPS by maintaining the paracellular barrier, acting mainly on enterocytes and the gut lumen despite poor systemic absorption [7,19].
- **Colitis models.** Curcumin ameliorated DSS-induced colitis in mice, partially restoring barrier integrity and altering the microbiota and fecal metabolites [14]. Bisdemethoxycurcumin, a curcuminoid also present in C. amada, improved barrier function, reduced epithelial apoptosis, inhibited NLRP3 inflammasome activation and modulated the microbiota in DSS colitis [8].
- **Reviews and clinical data.** Reviews describe curcumin as an adjuvant for intestinal inflammatory disease through effects on barrier function and inflammation [5,20]. A randomised placebo-controlled trial of a curcumin extract in adults with digestive complaints found no significant change in microbiota or SIBO compared with placebo [21], and a 2026 study in ulcerative colitis, Crohn's disease and healthy participants found only transient microbiota shifts, in an uncontrolled design [22]. Human evidence for microbiota-mediated benefit is therefore still weak.

Table 3: Strength of Evidence for Gut-Relevant Effects of *C. Amada*

Effect	Evidence for C. amada	Evidence level
Antioxidant (cell-free)	Phenolic fractions, extracts	In vitro
H+,K+-ATPase and H. pylori inhibition	Phenolic fractions	In vitro
Anti-ulcer (ethanol/aspirin)	Fresh rhizome juice	Animal, unpublished thesis
Anti-inflammatory	Ethanolic extract	Animal, non-intestinal
Tight-junction / permeability	None found	Gap
Colitis models	None found	Gap
Microbiota modulation	None found	Gap
Human gut outcomes	None found	Gap

TOXICOLOGICAL AND SAFETY CONSIDERATIONS

C. amada is eaten as a spice and pickle, and a methanol rhizome extract showed no toxic effects in mice at high doses in a hypoglycaemic study [2], while the fresh-juice thesis reported no mortality in acute testing [15]. These observations come from short experiments and do not constitute a full safety profile. Chronic toxicity, genotoxicity, reproductive safety and drug interaction data for concentrated extracts are lacking [3]. For curcumin, poor solubility, rapid metabolism and low systemic bioavailability are the usual limits, and safety work on *C. longa* extract in mice has been reported separately [19,23]. Concentrated mango ginger supplements should therefore be treated as unevaluated, and people with biliary disease, those taking anticoagulants or antiplatelet drugs (given the platelet-aggregation-inhibitory activity of a C. amada molecule [13]), and pregnant or breastfeeding women should seek medical advice before use. Unlike *Aristolochia* species, C. amada is not known to contain aristolochic acids, but botanical authentication remains essential because adulteration and misidentification occur in the *Curcuma* trade.

FUTURE RESEARCH PERSPECTIVES

The central gap is the absence of barrier-specific studies. Priorities include:

1. **Standardised extracts.** Chemically fingerprinted extracts (curcuminoid, phenolic acid and terpenoid content) from authenticated material, tested alongside *C. longa* extract and pure curcumin.
2. **Epithelial models.** Caco-2, HT-29 and organoid monolayers to measure transepithelial electrical resistance, FITC-dextran flux, and ZO-1, occludin and claudin expression after inflammatory or oxidative challenge.

3. **In vivo colitis and permeability models.** DSS, TNBS and NSAID-enteropathy models with histology, plasma LPS, D-lactate, cytokines and NF-κB, MAPK and NLRP3 readouts.
4. **Microbiota and metabolites.** 16S/metagenomic profiling and short-chain fatty acid analysis, including effects on commensals given the antimicrobial activity.
5. **Pharmacokinetics and herb–drug interactions.** ADME, gut-lumen exposure, metabolism by gut bacteria, and interaction with antiplatelet, anticoagulant and acid-suppressing drugs.
6. **Safety.** Sub-chronic and chronic toxicity of concentrated extracts.
7. **Clinical studies.** Small, controlled human trials with permeability and symptom endpoints, only after adequate preclinical and safety data.

CONCLUSION

Curcuma amada is a culinary and traditional medicinal plant whose rhizome contains curcuminoids, phenolic acids and terpenoids, and its extracts show antioxidant activity, inhibition of gastric proton pump activity and H. pylori growth, and preliminary anti-ulcer and anti-inflammatory effects. These properties make it a reasonable candidate for gut-protective research. The claim that mango ginger improves intestinal barrier integrity is, however, currently an extrapolation from curcumin and related *Curcuma* species rather than a demonstrated finding. No study located for this review measured tight-junction function, permeability, colitis outcomes or microbiota change for C. amada, and the human evidence for curcumin itself is mixed. Rigorous, standardised and safety-conscious studies are needed before C. amada can be recommended for gut health.

ABBREVIATIONS

- AA – Aristolochic acid
- ADME – Absorption, distribution, metabolism, excretion
- DSS – Dextran sodium sulfate
- FITC – Fluorescein isothiocyanate
- H+,K+-ATPase – Gastric proton pump
- IC50 – Half-maximal inhibitory concentration
- LPS – Lipopolysaccharide
- MAPK – Mitogen-activated protein kinase
- NF-κB – Nuclear factor kappa B
- NLRP3 – NOD-like receptor family pyrin domain-containing 3
- SIBO – Small intestinal bacterial overgrowth
- TLR4 – Toll-like receptor 4
- TNBS – Trinitrobenzene sulfonic acid
- ZO-1 – Zonula occludens-1

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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HOW TO CITE: Syamjith P., *F. Ameena* Fida, Muhammed Muflih K. M., Nasmiya A. P., Rabeeha P. K., Thasni, *Curcuma Amada*: Exploring ITS Phytochemical Diversity, Multifaceted Pharmacological Activities and Gastroprotective Mechanism -an Integrated Review., Int. J. of Pharm. Sci., 2026, Vol 4, Issue 11, 4428-4433. https://doi.org/10.5281/zenodo.24525053