

Turmeric (curcumin) remedies gastroprotective action

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ABSTRACT

The purpose of this review is to summarize the pertinent literature published in the present era regarding the antiulcerogenic property of curcumin against the pathological changes in response to ulcer effectors (*Helicobacter pylori* infection, chronic ingestion of non-steroidal anti-inflammatory drugs, and exogenous substances). The gastrointestinal problems caused by different etiologies was observed to be associated with the alterations of various physiologic parameters such as reactive oxygen species, nitric oxide synthase, lipid peroxidation, and secretion of excessive gastric acid. Gastrointestinal ulcer results probably due to imbalance between the aggressive and the defensive factors. In 80% of the cases, gastric ulcer is caused primarily due to the use of non-steroidal anti-inflammatory category of drug, 10% by *H. pylori*, and about 8-10% by the intake of very spicy and fast food. Although a number of antiulcer drugs and cytoprotectants are available, all these drugs have side effects and limitations. In the recent years a widespread search has been launched to identify new antiulcer drugs from synthetic and natural resources. An Indian dietary derivative (curcumin), a yellow pigment found in the rhizome of *Curcuma longa*, has been widely used for the treatment of several diseases. Epidemiologically, it was suggested that curcumin might reduce the risk of inflammatory disorders, such as cancer and ulcer. These biological effects are attributed to its anti-inflammatory and antioxidant activities. It can, therefore, be reported from the literature that curcumin prevents gastrointestinal-induced ulcer and can be recommended as a novel drug for ulcer treatment.

Key words: Curcumin, gastroprotective, *Helicobacter pylori*, non-steroidal anti-inflammatory drugs, oxidative stress

INTRODUCTION

The pathogenesis of gastrointestinal problems caused by different etiologies was observed to be associated with the alterations of various physiologic parameters such as reactive oxygen species (ROS), nitric oxide synthase (NOS), lipid peroxidation, and secretion of excessive gastric acid.^[1] Interference with normal protective mechanisms can result in mucosal injury, including erosions or ulceration, which may cause significant gastrointestinal bleeding. Gastrointestinal ulcer can be a life-threatening complication in patients with gastric mucosal injury.^[2] It is a deep lesion penetrating through the entire thickness of the gastrointestinal mucosa and muscularis mucosa.

Ulcer disease, which can occur in the esophagus, stomach, and/or in the duodenum, is one of the main prevalent still unresolved medical problems facing numerous patients in a wide range of age of both sexes worldwide.^[3] Ulceration involves the full thickness of the gastrointestinal mucosa. It is caused by disruption of the normal balance between the corrosive effect of the gastric juice and the protective effect of the mucus on the gastric epithelial cells.^[4] It results probably due to imbalance between the aggressive (acid, pepsin, bile, and *Helicobacter pylori*) and the defensive (gastric mucus, bicarbonate secretion, prostaglandin, nitric oxide, and innate resistance of the mucosal cells) factors.^[5] Ulceration refers to a site of inflammation where an epithelial surface of the skin, gastric epithelium, colonic mucosa, and bladder epithelium has become necrotic and eroded, often associated with subepithelial acute and chronic inflammation.^[6] Gastric mucosal layers play the role of a barrier by limiting the exposure of the gastric mucosal cells to numerous injurious luminal agents and irritants of both exogenous and endogenous origin. The mucosal surface epithelium is subject to attack by physical, chemical, or microbiological agents from the gastric lumen, which are involved in multiple pathologies, such as gastritis, peptic ulcer, or gastric cancer. Pretreatment with different substances could effectively prevent the gastric mucosa from developing erosions and ulcerations.^[7]

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In 80% of the cases, gastric ulceris caused primarily due to the use of nonsteroidal anti-inflammatory category of drugs, 10% by *H. pylori*, and about 8-10% by use of very spicy and fast food.^[8] Although a number of antiulcer drugs, such as H₂ receptor antagonists, proton pump inhibitors, and cytoprotectants, are available, all these drugs have side effects and limitations.^[9] In the recent years, a widespread search has been launched to identify new antiulcer drugs from synthetic and natural resources. An Indian dietary derivative – curcumin– has been proved an invaluable therapeutic agent that has no side effects and is cheaper to treat gastric diseases.^[10] Curcumin (C₂₁H₂₀O₆), the principal curcuminoid, a yellow pigment found in the rhizome of *Curcuma longa*, also known as turmeric, has been used since ancient times in China to treat various human disorders, and it is well documented for its medicinal properties in the Indian and Chinese systems of medicine.^[11] The presence of both phenolic OH and CH₂ groups in β-diketone moiety of this natural compound contributes significantly to its potent antioxidant properties.^[12] The gastro protective potentials of curcumin might protect patients from the adverse gastric side effects of many anti-inflammatory drugs, thereby improving the quality of life for patients and decreasing the treatment costs significantly.^[13] Both *in vitro* and *in vivo* studies have shown curcumin to possess a wide range of pharmacological activities including, antiprotozoa, antimicrobial, antivenom, anti-HIV, antitumor, and antiangiogenic activities.^[14,15] This article, therefore, intends to document only the gastro protective potentials of curcumin that have been reported in both *in vitro* and *in vivo* studies.

Curcumin

Curcumin (curcuminoid), a yellow pigment obtained from the rhizomes of *Curcuma longa* (Family: Zingiberaceae), is a major component of turmeric and is commonly used as a spice and food-coloring agent.^[16,17] Curcumin (diferuloylmethane), a polyphenol, is an active element of the perennial herb *C. longa* (commonly known as turmeric). The major curcuminoids present in turmeric are demethoxycurcumin (curcumin II) and bisdemethoxycurcumin (curcumin III), and the recently identified cyclocurcumin.^[18] The major components of commercial curcumin are curcumin I (77%), curcumin II (17%), and curcumin III (3%).^[3] The coloring nature of turmeric was isolated in the 19th century and was named curcumin. Curcuminoids refer to a group of phenolic compounds present in turmeric, which are chemically related to its principal ingredient curcumin.^[14]

Three curcuminoids were isolated from turmeric: Curcumin, demethoxycurcumin, and bisdemethoxycurcumin [Figure 1]. All three impart the hallmark yellow pigmentation to the *C. longa* plant and particularly to its rhizomes. The chemical structure of curcumin was determined in the 1970s and the 1980s; recently, the potential uses of curcuminoids in medicine have been studied extensively. It was shown

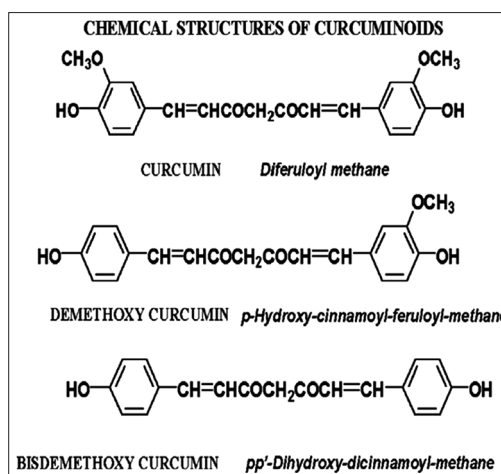


Figure 1: Chemical structure of curcuminoids curcumin, demethoxycurcumin, and bisdemethoxy curcumin that have shown antioxidant and anti-inflammatory properties^[15]

that oral consumption of curcumin in rats resulted in approximately 75% of it being excreted in the feces and only traces appearing in the urine, whereas intra-peritoneal administration of curcumin accounted for similar levels of fecal excretion of curcumin, with only 11% found in bile, suggesting the poor absorption of curcumin from the intestine.^[19] It has also been shown to be bio-transformed to dihydrocurcumin and tetrahydrocurcumin. Subsequently, these products are converted to monoglucuronide conjugates, and reported that the main biliary metabolites of curcumin are glucuronide conjugates of tetrahydrocurcumin and hexahydrocurcumin.^[20]

Gastric diseases and its significance

A gastric ulcer is a hole in the gut lining of the stomach, duodenum, or esophagus. An ulcer is a gastric ulcer of the stomach, a duodenal ulcer is an ulcer of the duodenum, and an esophageal ulcer is an ulcer of the esophagus. An ulcer occurs when the acidic digestive juices that are secreted by the stomach cells corrode the lining of these organs. For many years, excess acid has been believed to be the major factor responsible for ulcer disease. Accordingly, treatment emphasis was on neutralizing and inhibiting the secretion of stomach acid. Although acid is considered a primary factor in ulcer formation, the leading cause of ulcer disease currently is believed to be infection of the stomach by the bacteria called *H. pylori*. Another major cause of ulcers is the chronic use of anti-inflammatory medications, commonly referred to as non-steroidal anti-inflammatory drugs (NSAIDs) including aspirin. Cigarette smoking and ingestion of exogenous substances such as alcohol and fast foods are also important causes of ulcer formation and ulcer treatment failure.^[8,10,21] These major effectors of gastric ulcer trigger the secretion of messenger chemicals called the ulcer chemical mediators (e.g., gastric acid, histamine, acetylcholine, Prostaglandin I₂ etc.).

Helicobacter pylori

The interaction of this bacterium with the host cells (epithelial cells) had been reported to be implicated in various gastric diseases, such as gastric ulcer, adenocarcinoma, and lymphoproliferative disorders.^[22] *H. pylori*-infected gastric mucosa showed infiltration of polymorphonuclear leukocytes, lymphocytes, monocytes, and intraepithelial severe neutrophil infiltration.^[23] These changes would increase apoptosis and proliferation in the mucosal layer.^[24]

NSAIDS

NSAIDs-induced ulcer is an intricate process involving inhibition of prostaglandin synthesis in the gastric tract, which causes increased gastric acid secretion, diminished bicarbonate secretion, diminished mucus secretion, diminished trophic effects on epithelial mucosa,^[25] and increased microvascular permeability, nitric oxide imbalance, and free radical production. These anti-inflammatory drugs inhibit the activities of cyclooxygenase (COX) enzymes, block prostaglandin synthesis, increase gastric acid secretion, cause mucus depletion, and increase mucosal wall damage.^[21]

Oxidative stress

This is caused by exogenous substances such as cigarette smoking, and consumption of alcohol and fast foods. This factor causes gastric lesion by hemorrhage, resulting from the intense production of free radicals ROS, ruptured lysosome membrane, released hydrolytic enzymes, thereby causing damage to gastric epithelial cells.^[10] This damage leads to the constriction of gastric mucosa veins and arteries, causing congestion, inflammation, and tissue injury.^[26]

Curcumin gastroprotective potentials

C. longa has been used in traditional remedy for a wide range of ailments, including wound healing, urinary and gastrointestinal tract infections, and liver ailments.^[27] Curcumin has been defined as the most active component in *C. longa* and has considerable gastroprotective and antiulcerogenic effect. Its antiulcer potential activity was recently confirmed and reviewed in our laboratory.^[28] The antiulcer activity of curcumin was displayed by attenuating the different ulcerative effectors including gastric acid hypersecretion, total peroxides, myeloperoxidase activity, IL-6, and apoptotic incidence, along with its inhibitory activity for pepsin.^[21]

One study carried out with curcumin and dimethoxycurcumin to investigate the major functional group in curcumin reported that phenolic OH plays a major role in the activity of curcumin.^[29] It is evident that the antiulcer activity of curcumin arises from its antioxidant activity. Since, the antioxidant or scavenging reactive free radicals ability of curcumin arise whether from the phenolic OH group or from the CH₂ group of the b-diketone moiety.^[30] Free radical-mediated peroxidation of membrane lipids and oxidative damage of cellular molecules are

believed to be associated with various chronic pathological complications such as cancer, ulcer, and other inflammatory diseases.^[31] Curcumin is assumed to play a vital role against these pathological conditions, and could be an antiulcer potent agent.

A study also indicated that indomethacin inactivates gastric peroxidase to induce reactive-oxygen-mediated gastric mucosa injury, and concluded that indomethacin-induced oxidative damage by ROS as shown by increased lipid peroxidation and thiol depletion was almost completely blocked by curcumin.^[32] That is, curcumin protects gastric peroxidase from inactivation by indomethacin for efficient enzymatic removal of H₂O₂ to block gastric damage by ROS.^[33]

Surprisingly, curcumin showed immense therapeutic potential against *H. pylori* infection, as it was highly effective in the eradication of *H. pylori* from infected mice as well as in restoration of *H. pylori*-induced gastric damage. Curcumin does this by preventing the growth of *H. pylori* cagA + Strain to control *H. pylori*-mediated ulcer, suggesting its antiulcer potential.^[34]

It has been suggested that NSAIDs could induce gastric injury through increases in inflammatory cytokines and leukocyte adhesions. Curcumin, an antioxidant herbal substance, can prevent these adverse effects and hence might be used as a preventive method for NSAIDs-induced gastropathy.^[33] It was also reported that curcumin is more active against COX-2 and TXA₂ compared to COX-1.^[35] This is supported by the findings of Morimoto *et al.*^[36] that curcumin possesses COX-2 and TXA₂ inhibitory activity without affecting COX-1 activity. That is, curcumin can only block the inflammatory prostaglandin (PGI₂) synthesis without affecting the synthesis of protective prostaglandin (PGE₂), which is a protective mediator against gastric-induced damage. Equally, it was reported that the anti-inflammatory effect of curcumin is most likely mediated through its ability to inhibit COX-2, lipoxygenase (LOX), and inducible nitric oxide synthase (iNOS). COX-2, LOX, and iNOS are important enzymes that mediate inflammatory processes.^[33,34] Recently, it has been suggested that curcumin affected arachidonic acid metabolism by blocking the phosphorylation of cytosolic phospholipase and decreasing the expression of COX-2. Furthermore, it also inhibited the catalytic activities of 5-LOX.^[37] These activities may contribute to the anti-inflammatory and antiulcer actions of curcumin and its analogs.^[37] This could also serve as an evidence that curcumin is a potent antiulcer agent.

A research also reported that turmeric powder has beneficial effect on the stomach. It increases mucin secretion in rabbits and may thus act as a gastroprotectant against irritants.^[38] Curcumin has been shown to protect the stomach from the ulcerogenic effects of phenylbutazone

in guinea pigs at 50 mg/kg doses.^[39,40] It also protects from 5-hydroxytryptamine-induced ulceration at 20 mg/kg doses.^[39] Recently, it was confirmed that curcumin can block indomethacin, ethanol, and stress-induced gastric ulcer and can prevent pylorus-ligation-induced acid secretion.^[40] Rafatullah *et al.* (1990) reported that an oral dose of 500 mg/kg of the ethanol extract of turmeric produced significant antiulcerogenic activity in rats subjected to hypothermic restraint stress, pyloric ligation indomethacin, and reserpine administration. He suggested that turmeric extract not only increased gastric wall mucus but also restored the non-protein sulfhydryl content in the glandular stomachs of rats, and finally concluded that the extract has significant antiulcer, antisecretory, and gastroprotective effects in rats.^[2]

CONCLUSION

Gastrointestinal ulcer could be induced by infection of the stomach by a bacterium called *H. pylori*, by chronic ingestion of NSAIDs, and exogenous substances such as cigarette, alcohol, and fast foods. A dietary natural drug (curcumin) could be used to protector treat the inflammation induced by the ulcer effectors. The biological effect of curcumin to combat these induced pathological disorders is due to its anti-inflammatory and antioxidant activities. Therefore, this review confirmed curcumin as an antiulcer potent agent.

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