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Inhibition of urease as a central mechanism underlying the anti-*Helicobacter pylori* activity of chios mastic gum

Abstract

Mastic gum, a natural resin derived from *Pistacia lentiscus* var. *Chia*, has been traditionally used for gastrointestinal disorders. *Helicobacter pylori* is a Gram-negative bacterium that chronically colonizes the human stomach and is strongly associated with peptic ulcer disease and gastric malignancies. The increasing resistance to conventional antibiotic therapy has prompted the search for alternative treatments. This paper explores the antibacterial activity of Chios mastic gum (CMG) against *H. pylori*, with particular emphasis on its mechanism as a natural urease inhibitor. CMG is rich in bioactive triterpenic acids, including masticadienonic and isomasticadienonic acids, which exhibit significant anti-inflammatory and antimicrobial properties. Urease plays a critical role in *H. pylori* survival by neutralizing gastric acidity through ammonia production. We hypothesize that the anti-*H. pylori* effect of mastic gum is primarily mediated through inhibition of urease activity, thereby impairing bacterial colonization and pathogenicity. This mechanism highlights the therapeutic potential of CMG as a natural, safer alternative to synthetic urease inhibitors.

Keywords: Mastic gum; *Helicobacter pylori*; urease inhibition; triterpenic acids; natural products; gastric ulcer; antimicrobial activity.

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Introduction

Mastic gum is a natural aromatic resin obtained from the trunk and branches of *Pistacia lentiscus* var. *Chia*. It is uniquely produced on the island of Chios in Greece, where traditional cultivation methods have been preserved for centuries.[1] The resin is collected as tear-shaped droplets following controlled incisions in the tree bark and has been historically used for its medicinal properties.

Helicobacter pylori is a Gram-negative bacterium that persistently colonizes the human gastric mucosa. It infects approximately 50% of the global population and is associated with chronic gastritis, peptic ulcer disease, gastric adenocarcinoma, and mucosa-associated lymphoid tissue (MALT) lymphoma.[2] Despite eliciting both humoral and cellular immune responses, the bacterium often evades eradication and persists lifelong without treatment.

In Sudan, resin harvesting is a traditional practice centered on key trees such as Acacia senegal (locally known as Hashab), Acacia seyal (Talh), Boswellia papyrifera (Luban), and Commiphora myrrha (Mur), where farmers use a tapping method involving shallow incisions in the bark—typically after the rainy season—to stimulate exudation of gum or resin as a protective response; the exuded material is left to dry on the tree for 2–3 weeks before manual collection, with some species like Boswellia requiring repeated incisions and sequential harvesting cycles every 10–15 days to improve quality, while sustainable practices such as rotational tapping are essential to prevent tree damage and ensure continued production, making this process both economically and ecologically significant in Sudan.[3–5]

Mastic gum has been reported as an effective treatment for peptic and duodenal ulcers, largely attributed to its antibacterial activity against *H. pylori*.[6–9] However, the precise molecular mechanisms underlying this activity remain incompletely understood.

Pathogenesis of *Helicobacter pylori* and role of urease

Urease is a key virulence factor in *H. pylori* pathogenesis. It catalyzes the hydrolysis of urea into ammonia and carbon dioxide, enabling the bacterium to survive in the highly acidic gastric environment.[10] The ammonia produced not only neutralizes gastric acid but also contributes to epithelial cell damage and inflammation.

Given its essential role in bacterial survival and colonization, urease represents a critical therapeutic target in *H. pylori* infection.

Phytochemical composition of chios mastic gum

Chios mastic gum contains a high proportion (40–55%) of triterpenic acids, including:

I. 24Z-masticadienonic acid (MNA)
II. 24Z-isomasticadienonic acid (IMNA)
III. 24Z-masticadienolic acid (MLA)
IV. 24Z-isomasticadienolic acid (IMLA)
V. Oleanonic acid
VI. Moronic acid

These compounds belong to pentacyclic and tetracyclic triterpenoids and exhibit potent anti-inflammatory and antimicrobial activities.[11]

Anti-*Helicobacter pylori* activity of mastic gum

Several in vitro and in vivo studies have demonstrated that mastic gum exhibits bactericidal activity against *H. pylori*.[6–9] Clinical evidence also suggests its beneficial role in reducing bacterial load and improving gastrointestinal symptoms.

The antibacterial effects of mastic gum are likely multifactorial, involving:

I. Direct bactericidal activity

II. Disruption of bacterial cell membranes

III. Anti-inflammatory effects on gastric mucosa

Urease inhibition as a mechanism of action

Triterpenic acids present in CMG are recognized as natural inhibitors of urease enzyme activity.[12] Unlike synthetic urease inhibitors, which may have toxicity and adverse effects, natural compounds provide a safer alternative.

Inhibition of urease leads to:

a. Reduced ammonia production.

b. Increased bacterial susceptibility to gastric acidity.

c. Impaired colonization and survival of *H. pylori*.

Hypothesis

We hypothesize that the bioactivity of mastic gum against *Helicobacter pylori* is primarily mediated through inhibition of urease enzyme activity. By targeting this essential virulence factor, mastic gum disrupts the bacterium's ability to survive in the acidic gastric environment, thereby reducing its pathogenicity.

Conclusion

Chios mastic gum represents a promising natural therapeutic agent against *H. pylori* infection. Its rich composition of bioactive triterpenic acids contributes to its antibacterial and anti-inflammatory properties. The proposed mechanism of urease inhibition offers a novel and safer alternative to conventional therapies. This hypothesis requires validation through in vitro urease inhibition assays, molecular docking, and enzyme kinetics studies.

Acknowledgment

None.

Competing interests

The author declares no conflicts of interest.

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