AOR CODE: AOR04201

Premium

Gastro Relief

Revolutionary Natural Formula for Heartburn & the Damage it Causes

- Relieves symptoms while addressing possible causes
- Heals the gastric lining, protects the esophagus, and kills H. pylori
- Provides fast-acting relief of heartburn

Gluten Free  Vegan  Non-GMO  Gastrointestinal Health Heartburn

AOR Code  Variant
AOR04201  60 VEGI-CAPS

Details
Gastro Relief is a unique and revolutionary supplement for heartburn, indigestion, gastritis and other concerns of the upper gastrointestinal tract. Mastic gum, ginger, zinc-carnosine, vitamin C, nitric oxide-producing potassium nitrate, and sodium alginate provide fast symptomatic relief while promoting the healing of the stomach lining.

Gastro Relief is designed to quickly and effectively provide natural support against the pain of heartburn while providing nutrients to target potential causes of heartburn and help heal the cells lining the stomach. Mastic gum has been shown to inhibit H. pylori, a bacterium that is at the root of the majority of heartburn cases and stomach issues. Vitamin C increases the acidity of the stomach, since most gastric issues stem from a lack of stomach acid as opposed to too much. Ginger calms the stomach during digestive upset, while zinc-carnosine and nitric oxide promote healing of stomach lining cells that may have been damaged from a bacterial infection or from inappropriate acid levels. Finally, sodium alginate prevents gastric contents from backing up into the esophagus, preventing painful heartburn symptoms and protecting the esophagus against damage from acidity.

All in all, Gastro Relief is an excellent alternative to antacids or conventional medications to reduce heartburn symptoms, promote proper digestion and decrease stomach pain while actually promoting healing to the damaged tissues.

Label Info
**Discussion**
Gastro Relief helps relieve digestive upset/disturbances including dyspepsia.

**Product Variation**

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<th>Product Code</th>
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<td>AOR04201</td>
<td>60 VEGI-CAPS</td>
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**Supplements Facts**

<table>
<thead>
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<th>Serving Size: 2 Capsules</th>
<th>Amount</th>
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<tr>
<td>Ginger extract (10:1)</td>
<td>50 mg</td>
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<tr>
<td>Zinc L-carnosine</td>
<td>75 mg</td>
</tr>
<tr>
<td>Mastic gum (Pistacia lentiscus)</td>
<td>400 mg</td>
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<tr>
<td>Vitamin C (ascorbic acid)</td>
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Non-medical ingredients:
potassium nitrate (202 mg), sodium alginate, microcrystalline cellulose, silicon dioxide, sodium stearyl fumarate, maltodextrin. Capsule: hypromellose.

**Guarantees**
AOR™ guarantees that all ingredients have been declared on the label. Contains no wheat, gluten, nuts, peanuts, sesame seeds, sulphites, mustard, soy, dairy, eggs, fish, shellfish or any animal byproduct.

**Adult Dosage**
Take 2 capsules daily with food, or as directed by a qualified health care practitioner.

**Cautions**
Consult a health care practitioner prior to use if you have hypochlorhydria/achlorhydria, stomach lesions, diabetes mellitus, Crohn’s disease, or if you are taking hypoglycemic agents or hypolipidemic agents. Consult a health care practitioner if symptoms persist or worsen. Hypersensitivity (ie. allergy) has been known to occur, in which case discontinue use. Methemoglobinemia has been reported on rare occasions following an accidental overdose of potassium nitrate; intravenous methylene blue is the specific therapy for this condition. Do not use if pregnant or breastfeeding, if you are taking erectile dysfunction-type products, or if you have an allergy to plants of the Anacardiaceae family such as pistachio, terebinth, Chinese pistache, and Schinus terebinthifolius (Brazilian pepper).

**Source**
Natural botanical extracts

**Pharmaceutical synthesis**

**Main Application**
Heartburn
Gastric and duodenal ulcers

Helicobacter pylori infection

Disclaimer

The information and product descriptions appearing on this website are for information purposes only, and are not intended to provide or replace medical advice to individuals from a qualified health care professional. Consult with your physician if you have any health concerns, and before initiating any new diet, exercise, supplement, or other lifestyle changes.

Research

Background

Heartburn

Heartburn is a very common condition that is characterized by a burning sensation that starts in the upper abdomen and moves into the chest and that is worse when lying down or bending forward. Heartburn sufferers can often associate the problem with specific foods, with fatty foods, acidic foods, alcohol and chocolate being known offenders. Heartburn is not mere acid indigestion but usually a symptom of gastroesophageal reflux – a condition where the contents of the stomach back up into the esophagus because the sphincter between the stomach and esophagus is not functioning properly. Since the cells of the esophagus are not meant to handle this level of acidity, heartburn is extremely painful and can have serious long term consequences such as esophageal cancer, narrowing of the esophagus from scaring and Barrett’s esophagus. It is not only the acid which causes problems. The stomach also contains pepsin and bile acids which are equally dangerous for the cells of the esophagus. Pepsin is a digestive enzyme which degrades proteins and the presence of bile acids in the esophagus has been shown to increase the likeliness of more serious injuries.

Quick Relief

The premise behind Gastro Relief™ is to provide quick and effective relief from the symptoms of heartburn while addressing any fundamental pathology which would cause the problem.

How Do the Ingredients in Gastro Relief Help?

Bactericidal Mastic Gum

Gastro Relief™ contains Mastic Gum which has been shown to wipe out bacteria called Helicobacter pylori, the cause behind the majority of gastric and duodenal ulcers. Untreated H. pylori also increases the risk of gastric cancer.

Protective Alginic Acid

When it comes to relieving heartburn, the key ingredient in Gastro Relief™ is alginic acid. Alginic acid is a viscous substance found in algae which absorbs water extremely quickly to form a “raft” on top of the gastric contents. This “raft” has two effects; it prevents the gastric contents from being pushed back up the esophagus and also coats the esophagus if the gastric contents were to reach the
esophagus. Since it acts as a barrier and not simply by neutralizing the acidity or by preventing acid from being generated in the stomach, the alginic acid also protects the esophagus from the pepsin and bile salts found in the stomach and also known to damage the upper digestive tract.

**Acidic Vitamin C**

Treating heartburn in isolation with heartburn medication targeting the acidity of the stomach is narrow minded and only provides temporary relief. Indeed, the problem with reflux is not that the stomach is too acidic; often the reverse is actually true. The valve that releases the contents of the stomach into the intestinal tract is triggered by acidity, if the stomach contents are not acidic enough, this valve does not open and the stomach contents back up into the esophagus causing pain and irritation. This is why Gastro Relief™ contains Vitamin C which is acidic, instead of the typical acidity buffers.

**Healing Nitric Oxide & Zinc-Carnosine**

Nitrates and their reduced products have been consumed for thousands of years. For the last fifty years nitrates have been the subject of serious propaganda and regulatory restrictions. There have been suggestions that intake of these ingredients can cause cancers due to the link between cured meats and cancer. Yet population studies and various toxicological studies show that nitrates are important nutrients with significant health benefits of which we are being deprived. In fact various researchers suggest that nitrates are probably responsible for the health benefits associated with various fruits and vegetables and the so called “Mediterranean Diet”. In addition various vegetables are rich sources of nitrates and an intake of a plate of salad or a glass of beetroot juice offers many times the nitrate level that the regulatory bodies have set as an upper limit.

Nitrates produce nitric oxide (NO) in the gut, which improves blood flow and nutrient delivery to the stomach lining to help heal it. Zinc-Carnosine also helps to heal the cells lining the gut that have been damaged either by H. pylori, stomach acids or harsh pharmaceuticals.

**Research**

**Mastic Gum & H. pylori**

Clinical studies have clearly shown the effectiveness of this resin with 80% of patients receiving mastic gum for two weeks reporting significant improvements in their symptoms. Further test tube studies confirmed that Mastic Gum kills H. pylori in concentrations easily attainable through supplementation.

**Zinc-Carnosine & H. pylori-related gastritis**

Zinc Carnosine has received much attention lately thanks to its ability to up-regulate key antioxidant enzymes thereby preventing free radicals from damaging cells. Studies have confirmed that Zinc Carnosine has anti-ulcer properties and prevents gastric mucosal injury. Animal studies also show that Zinc Carnosine is indicated in H. pylori infections as the molecule prevents the development of H. pylori related gastritis.
Nitric Oxide & Inflammation

Studies also clearly demonstrate that Nitric oxide precursors are effective anti-inflammatory agents with protective effects against gastritis. Potassium Nitrate is a precursor to Nitric Oxide. Nitric Oxide is a potent vasodilator and increases blood flow to the gastric mucosa enhancing repair and the inflow of nutrients and oxygen. Higher nitric oxide levels in the stomach were also shown to increase effective peristalsis movements.

Supportive Ascorbic Acid

The acidic nature of Vitamin C helps lower the pH of the stomach, which increases the chances of the LES closing properly and reducing the risk of heartburn. The presence of ascorbic acid also enhances the conversion of dietary nitrate and salivary nitrite to NO by gastric juices. Those with H. pylori infections often have been found to have reduced ascorbic acid levels in their gastric secretions.

Alginic Acid & Reflux

Clinical studies clearly demonstrate the protective role of alginic acid, protecting the esophagus from the damaging potential of the refluxate, not merely the acid. This has led some experts in the field to lean towards natural products such as alginate rafts to address the problem instead of using more conventional approaches. An added bonus is that alginic acid works extremely quickly providing relief within a few minutes. Researchers have also confirmed that Alginic acid is more effective than ranitidine and omeprazole, two drugs used in reflux, during the first hour after dosing. It is not surprising that this method of treatment for heartburn has been around for more than 30 years.

Market Trends

Antacid medications such as proton pump inhibitors (PPIs) and H2-receptor antagonists are among the most sold medications in the world. This means that and overwhelming percentage of people suffer from gastric disturbances and heartburn. Unfortunately, these medications that are designed to reduce hydrochloric acid (HCL) secretion in the stomach have some very serious implications. Stomach acid is required to properly digest food, which releases nutrients for absorption by the body. It also kills swallowed pathogens and reduces the risk of some infections. The use of antacid medications and resulting low HCL secretion has been associated with an increased risk of bone fractures (likely due to malabsorption of nutrients), deficiencies in various nutrients like vitamin B12 which can contribute to the development of heart, brain and nerve diseases to name a few, food allergies due to undigested proteins, drug hypersensitivities, increased cases of pneumonia, and much more.

AOR Advantage

When it comes to the treatment of heartburn, it is essential to address the underlying problem associated with the symptom. Heartburn is usually merely a symptom of a more serious problem such as an infection or an incompetent sphincter. Addressing only the symptoms can result in long-term problems. Gastro Relief™ was formulated specifically to alleviate heartburn while addressing the root cause of the problem. Try Gastro Relief™ today to experience quick, natural and effective relief of
heartburn while benefiting from its long-term healing effects.

References


**Abstract**

A review of the gastroprotective effects of ginger (Zingiber officinale Roscoe).


Haniadka R, Saldanha E, Sunita V, Palatty PL, Fayad R, Baliga MS.

The rhizomes of Zingiber officinale Roscoe (Zingiberaceae), commonly known as ginger is an important kitchen spice and also possess a myriad health benefits. The rhizomes have been used since antiquity in the various traditional systems of medicine to treat arthritis, rheumatism, sprains, muscular aches, pains, sore throats, cramps, hypertension, dementia, fever, infectious diseases, catarrh, nervous diseases, gingivitis, toothache, asthma, stroke and diabetes. Ginger is also used as home remedy and is of immense value in treating various gastric ailments like constipation, dyspepsia, belching, bloating, gastritis, epigastric discomfort, gastric ulcerations, indigestion, nausea and vomiting and scientific studies have validated the ethnomedicinal uses. Ginger is also shown to be effective in preventing gastric ulcers induced by nonsteroidal anti-inflammatory drugs [NSAIDs like indomethacin, aspirin], reserpine, ethanol, stress (hypothermic and swimming), acetic acid and Helicobacter pylori-induced gastric ulcerations in laboratory animals. Various preclinical and clinical studies have also shown ginger to possess anti-emetic effects against different emetogenic stimuli. However, conflicting reports especially in the prevention of chemotherapy-induced nausea and vomiting and motion sickness prevent us from drawing any firm conclusion on its effectiveness as a broad spectrum anti-emetic. Ginger has been shown to possess free radical scavenging, antioxidant; inhibition of lipid peroxidation and that these properties might have contributed to the observed gastroprotective effects. This review summarizes the various gastroprotective effects of ginger and also emphasizes on aspects that warrant future research to establish its activity and utility as a gastroprotective agent in humans.

Ginger extract and polaprezinc exert gastroprotective actions by anti-oxidant and growth factor modulating effects in rats.


Ko JK, Leung CC.

BACKGROUND AND AIM: Contemporary medications used in the treatment of gastric ulcers involve the use of novel mucosal protective drugs. The present study aimed to investigate the gastroprotective effect of ginger extract and polaprezinc in a rat model of acetic acid-induced gastric ulcer.
METHODS: ‘Kissing’ ulcers were induced in male Sprague-Dawley rats by using 60% acetic acid. Rhizoma Zingiber officinale (ginger) extract (1.5-5 g/kg) or polaprezinc (30 and 60 mg/kg) was orally given to the animals once daily for three consecutive days after ulcer induction. All animals were killed on day 5 by an overdose of ketamine.

RESULTS: Both ginger extract and polaprezinc significantly reduce the gastric ulcer area in a dose-dependent manner, with concomitant attenuation of the elevated activities of xanthine oxidase and myeloperoxidase, as well as malondialdehyde level in the ulcerated mucosa. Nevertheless, only polaprezinc could restore the mucosal glutathione level. Polaprezinc also causes the overexpression of basic fibroblast growth factor, vascular endothelial growth factor and ornithine decarboxylase, whereas ginger extract only increases the expression of the two growth factors in the gastric mucosa. Furthermore, polaprezinc could consistently downregulate the protein expression of tumor necrosis factor (TNF)-?, interleukin-1?, macrophage inflammatory protein-2 and cytokine-induced neutrophil chemoattractant-2? that have been activated in the ulcerated tissues, whereas ginger extract mainly inhibits the expression of the chemokines and to some extent TNF-?.

CONCLUSION: Ginger extract and polaprezinc both show anti-oxidation that consequently alleviates gastric mucosal damage and promotes ulcer healing, which together serve as effective mucosal protective agents.


Ueda K, Ueyama T, Oka M, Ito T, Tsuruo Y, Ichinose M.

Heme oxygenase (HO)-1 is implicated in cytoprotection in various organs. We tested a possibility that polaprezinc (PZ), an anti-ulcer drug, could induce HO-1 in the gastric mucosa. Male 6-week-old Wistar rats were intragastrically administered PZ. Gastric expression of HO-1 was assessed by real time RT-PCR and western blotting, and localization of HO-1 was observed by in situ hybridization and immunohistochemistry. The levels of HO-1 mRNA were increased in a dose-dependent manner. The levels of HO-1 mRNA were increased 4-fold by PZ at the dose of 200 mg/kg at 3 h as compared with control levels. The levels of immunoreactive HO-1 were increased 3-fold at 6 h. Signals for HO-1 mRNA and immunoreactivity were detected strongly in the surface gastric mucosal cells and moderately in the gastric macrophages. Treatment with an HO-1 inhibitor, stannous mesoporphyrin (SnMP) significantly worsened the HCl-induced acute gastric mucosal lesions and increased the apoptosis of mucosal cells. Mucosal lesions were decreased by pretreatment with PZ, while they were increased by co-administration with SnMP. These data indicate for the first time that PZ is an effective inducer of HO-1 in the stomach. PZ-induced HO-1 functions as a part of the mucosal protective effects of PZ.
The role of an alginate suspension on pepsin and bile acids – key aggressors in the gastric refluxate. Does this have implications for the treatment of gastro-oesophageal reflux disease?


Strugala V, Avis J, Jolliffe IG, Johnstone LM, Dettmar PW.

OBJECTIVES: During a reflux event the oesophagus is exposed to a heterogeneous mixture of gastric juice components. The role of non-acid components of the refluxate in causing damage to the oesophagus is now well established but no therapeutic option exists to address this.

METHODS: The role of Gaviscon Advance (GA), a raft-forming alginate suspension, in protecting the oesophagus from damage by pepsin and bile acids (aggressors) was investigated using a series of in-vitro models.

KEY FINDINGS: GA was able to dose-dependently inhibit pepsin activity over and above the neutralisation effect of the formulation. This was evident against both protein and collagen substrates using two distinct colorimetric assays. GA was able to retard the diffusion of pepsin and multiple bile acids using a Franz cell model. Using the raft-forming mode of action GA was able to remove both pepsin and multiple bile acids from a simulated reflux event. There was capacity in the GA raft to accommodate aggressors from multiple reflux events.

CONCLUSIONS: GA can specifically remove both pepsin and bile acids from the refluxate, limit their diffusion and affect enzymatic activity of pepsin. There is a role for GA to reduce the damaging potential of the refluxate and thus protect the oesophagus.

Dietary nitrite prevents hypercholesterolemic microvascular inflammation and reverses endothelial dysfunction


Stokes KY, Dugas TR, Tang Y, Garg H, Guidry E and Bryan NS.

The nitrite anion is an endogenous product of mammalian nitric oxide (NO) metabolism, a key intermediate in the nitrogen cycle in plants, and a constituent of many foods. Research over the past 6 years has revealed surprising biological and cytoprotective activity of this anion. Hypercholesterolemia causes a proinflammatory phenotype in the microcirculation. This phenotype appears to result from a decline in NO bioavailability that results from a reduction in NO biosynthesis, inactivation of NO by superoxide, or both. Since nitrite has been shown to be potently cytoprotective and restore NO biochemical homeostasis, we investigated if supplemental nitrite could attenuate microvascular inflammation caused by a high cholesterol diet. C57Bl/6J mice were fed either a normal diet or a high cholesterol diet for 3 wk to induce microvascular inflammation. Mice on the high cholesterol diet received either nitrite-free drinking water or supplemental nitrite at 33 or 99 mg/l ad libitum in their drinking water. The results from this investigation reveal that mice fed a cholesterol-enriched diet exhibited significantly elevated leukocyte adhesion to and emigration through the venular endothelium as well as impaired endothelium-dependent relaxation in arterioles.
Administration of nitrite in the drinking water inhibited the leukocyte adhesion and emigration and prevented the arteriolar dysfunction. This was associated with sparing of reduced tetrahydrobiopterin and decreased levels of C-reactive protein. These data reveal novel anti-inflammatory properties of nitrite and implicate the use of nitrite as a new natural therapy for microvascular inflammation and endothelial dysfunction associated with hypercholesterolemia.

**Nitrate in foods: harmful or healthy?**


Martijn B Katan

Nitrate and nitrite are considered hazardous, and there are legal limits to their concentration in food and drinking water. Nitrate from fertilizer accumulates in vegetables and fruit, and largescale livestock production yields huge amounts of manure rich in nitrate that seeps into groundwater. Therefore, keeping nitrate concentrations below legal limits is a struggle for farmers. In this issue of the Journal, Hord et al (1) challenge these limits. Other authors have already pointed out that the evidence for adverse effects of nitrate is inconsistent and that nitrate may actually be beneficial (2, 3). Hord et al (1) go one step further: they claim that nitrate and nitrite should be considered as nutrients.

**Zinc carnosine, a health food supplement that stabilises small bowel integrity and stimulates gut repair processes.**


**BACKGROUND:** Zinc carnosine (ZnC) is a health food product claimed to possess health-promoting and gastrointestinal supportive activity. Scientific evidence underlying these claims is, however, limited.

**AIM:** To examine the effect of ZnC on various models of gut injury and repair, and in a clinical trial.

**METHODS:** In vitro studies used pro-migratory (wounded monolayer) and proliferation ([3H]-thymidine incorporation) assays of human colonic (HT29), rat intestinal epithelial (RIE) and canine kidney (MDCK) epithelial cells. In vivo studies used a rat model of gastric damage (indomethacin/restraint) and a mouse model of small-intestinal (indomethacin) damage. Healthy volunteers (n = 10) undertook a randomised crossover trial comparing changes in gut permeability (lactulose:rhamnose ratios) before and after 5 days of indomethacin treatment (50 mg three times a day) with ZnC (37.5 mg twice daily) or placebo coadministration.

**RESULTS:** ZnC stimulated migration and proliferation of cells in a dose-dependent manner (maximum effects in both assays at 100 micromol/l using HT29 cells), causing an approximate threefold increase in migration and proliferation (both p CONCLUSION: ZnC, at concentrations likely to be found in the gut lumen, stabilises gut mucosa. Further studies are warranted.
Inhibition of gastric H, K-ATPase and Helicobacter pylori growth by phenolic antioxidants of Zingiber officinale.

Mol Nutr Food Res. 2007 Mar;51(3):324-32.

Siddaraju MN, Dharmesh SM.

Ulcer is a common global problem characterized by acute gastric irritability, bleeding, etc. due to either increased gastric cell proton potassium ATPase activity (PPA) or perturbation of mucosal defence. Helicobacter pylori has been identified as a major ulcerogen in addition to oxidative stress and nonsteroidal anti-inflammatory drugs. In this paper, we report ginger-free phenolic (GRFP) and ginger hydrolysed phenolic (GRHP) fractions of ginger (Zingiber officinale) as potent inhibitors of PPA and H. pylori growth. GRFP and GRHP inhibited PPA at an IC(50) of 2.9 ± 0.18 and 1.5 ± 0.12 microg/mL, exhibiting six- to eight-fold better potency over lansoprazole. GRFP is constituted by syringic (38%), gallic (18%) and cinnamic (14%) acids and GRHP by cinnamic (48%), p-coumaric (34%) and caffeic (6%) acids as major phenolic acids. GRFP and GRHP further exhibited free radical scavenging (IC(50) 1.7 ± 0.07 and 2.5 ± 0.16), inhibition of lipid peroxidation (IC(50) 3.6 ± 0.21 and 5.2 ± 0.46), DNA protection (80% at 4 microg) and reducing power abilities (80-338 U/g) indicating strong antioxidative properties. GRFP and GRHP may thus be potential in-expensive multistep blockers against ulcer.

Concomitant alterations in intragastric pH and ascorbic acid concentration in patients with Helicobacter pylori gastritis and associated iron deficiency anaemia.


BACKGROUND: Seroepidemiological and clinical studies suggest that Helicobacter pylori may cause iron deficiency anaemia (IDA) in the absence of peptic lesions by undefined mechanisms, which still remain to be fully elucidated. Gastric acidity and ascorbic acid (AA) promote iron absorption. AA is lowered in the presence of H pylori infection. H pylori can cause atrophic body gastritis with achlorhydria, decreased iron absorption, and consequent IDA. Whether alterations in intragastric acidity and AA concentrations play a role in IDA developing in patients with H pylori gastritis remains to be determined. AIM: To evaluate gastric juice pH and gastric juice and plasma AA in patients with H pylori infection and unexplained IDA, compared with controls with IDA and a healthy stomach or with controls with H pylori infection and no IDA.

RESULTS: Patients with IDA and H pylori gastritis were characterised by concomitant increased intragastric pH (median value 7) and decreased intragastric AA (median value 4.4 micro g/ml) compared with controls with a healthy stomach (median pH 2; median intragastric AA 17.5 micro g/ml) and with H pylori positive controls without IDA (median pH 2.1; median intragastric AA 7.06 micro g/ml). Intragastric AA was inversely related to pH (r=-0.40, p=0.0059) and corporal degree of gastritis (r=-0.53, p=0.0039). Plasma AA concentrations were lower in all infected groups than in
healthy controls.

CONCLUSIONS: Patients with unexplained IDA and H pylori gastritis present concomitant changes in intragastric pH and AA that may justify impaired alimentary iron absorption and consequent IDA.

Protective effect of dietary nitrate on experimental gastritis in rats.


Larauche M, Anton PM, Garcia-Villar R, Theodorou V, Frexinos J, Buéno L, Fioramonti J.

Nitrates have long been considered as harmful dietary components and judged responsible for deleterious effects on human health, leading to stringent regulations concerning their levels in food and water. However, recent studies demonstrate that dietary nitrate may have a major role in human health as a non-immune mechanism for host defence, through its metabolism to NO in the stomach. NO is a versatile molecule and although evidence exists showing that administration of low doses of exogenous NO protects against gastrointestinal inflammation, higher NO doses have been shown to exacerbate injury. So, the effect of an ingestion of nitrates in doses corresponding to a normal diet in human consumers on an experimental gastritis induced by iodoacetamide in rats was investigated. During gastritis one of the following compounds was given orally: water; KNO3; the NO donor sodium nitroprusside; the NO scavenger haemoglobin given with either water or KNO3. N(G)-nitro-l-arginine methyl ester (l-NAME), a non-specific NO synthase inhibitor, was administered with either water, iodoacetamide alone, or combined with KNO3. After killing, the stomach was resected and microscopic damage scores, myeloperoxidase and NO synthase activities were determined. Iodoacetamide-induced gastritis was significantly reduced by KNO3 administration, an effect which was reproduced by sodium nitroprusside and reversed by haemoglobin. l-NAME induced gastric mucosal damage in itself, and KNO3 did not prevent the gastritis induced by iodoacetamide associated with l-NAME. In conclusion, dietary nitrate exerts a protective effect against an experimental gastritis in rats by releasing NO in the stomach but such an effect requires the production of endogenous NO.

Bactericidal activity of Pistacia lentiscus mastic gum against Helicobacter pylori


Marone P, Bono L, Leone E, Bona S, Carretto E, Perversi L.

In this study we evaluated the antibacterial activity of mastic gum, a resin obtained from the Pistacia lentiscus tree, against clinical isolates of Helicobacter pylori. The minimal bactericidal concentrations (MBCs) were obtained by a microdilution assay. Mastic gum killed 50% of the strains tested at a concentration of 125 microg/ml and 90% at a concentration of 500 microg/ml. The influence of sub-MBCs of mastic gum on the morphologies of H. pylori was evaluated by transmission electron microscopy. The lentiscus resin induced blebbing, morphological abnormalities and cellular fragmentation in H. pylori cells.
Review article: alginate-raft formulations in the treatment of heartburn and acid reflux.


Mandel KG, Daggy BP, Brodie DA, Jacoby Hl.

Alginate-based raft-forming formulations have been marketed word-wide for over 30 years under various brand names, including Gaviscon. They are used for the symptomatic treatment of heartburn and oesophagitis, and appear to act by a unique mechanism which differs from that of traditional antacids. In the presence of gastric acid, alginates precipitate, forming a gel. Alginate-based raft-forming formulations usually contain sodium or potassium bicarbonate; in the presence of gastric acid, the bicarbonate is converted to carbon dioxide which becomes entrapped within the gel precipitate, converting it into a foam which floats on the surface of the gastric contents, much like a raft on water. Both in vitro and in vivo studies have demonstrated that alginate-based rafts can entrap carbon dioxide, as well as antacid components contained in some formulations, thus providing a relatively pH-neutral barrier. Several studies have demonstrated that the alginate raft can preferentially move into the oesophagus in place, or ahead, of acidic gastric contents during episodes of gastro-oesophageal reflux; some studies further suggest that the raft can act as a physical barrier to reduce reflux episodes. Although some alginate-based formulations also contain antacid components which can provide significant acid neutralization capacity, the efficacy of these formulations to reduce heartburn symptoms does not appear to be totally dependent on the neutralization of bulk gastric contents. The strength of the alginate raft is dependent on several factors, including the amount of carbon dioxide generated and entrapped in the raft, the molecular properties of the alginate, and the presence of aluminium or calcium in the antacid components of the formulation. Raft formation occurs rapidly, often within a few seconds of dosing; hence alginate-containing antacids are comparable to traditional antacids for speed of onset of relief. Since the raft can be retained in the stomach for several hours, alginate-based raft-forming formulations can additionally provide longer-lasting relief than that of traditional antacids. Indeed, clinical studies have shown Gaviscon is superior to placebo, and equal to or significantly better than traditional antacids for relieving heartburn symptoms. Alginate-based, raft-forming formulations have been used to treat reflux symptoms in infants and children, and in the management of heartburn and reflux during pregnancy. While Gaviscon is effective when used alone, it is compatible with, and does not interfere with the activity of antisecretory agents such as cimetidine. Even with the introduction of new antisecretory and promotility agents, alginate-rafting formulations will continue to have a role in the treatment of heartburn and reflux symptoms. Their unique non-systemic mechanism of action provides rapid and long-duration relief of heartburn and acid reflux symptoms.

A double-blind controlled clinical trial of mastic and placebo in the treatment of duodenal ulcer.


Al-Habbal MJ, Al-Habbal Z, Huwez FU.
A double-blind clinical trial was carried out on thirty-eight patients with symptomatic and endoscopically proven duodenal ulcer to compare the therapeutic responses to mastic (1 g daily, twenty patients) and placebo (lactose, 1 g daily, eighteen patients) given orally over a period of 2 weeks. Symptomatic relief was obtained in sixteen (80%) patients on mastic and in nine (50%) patients on placebo, while endoscopically proven healing occurred in fourteen (70%) patients on mastic and four (22%) patients on placebo. The differences between treatments were highly significant (P less than 0.01). Mastic was well tolerated and did not produce side effects. It is concluded that mastic has an ulcer healing effect, but further studies are needed to establish its role in treating peptic ulcer.