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The effect of Ginger (Zingiber officinale) On *Helicobacterpylori* at Different concentrations, Mechanisms

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Abstract

Helicobacter pylori (H. pylori) is a gastric pathogen with long-term reactions that is a significant causative agent of chronic gastritis, peptic ulcer disease (PUD), gastric mucosa-associated lymphoid tissue (MALT) lymphoma and gastric adenocarcinoma. Claritytracyclin, clarithromycin, metronidazole, and levofloxacin resistance has also created interest in exploring alternative antimicrobial agents. Ginger contains phytochemicals like gingerols, shogaols, paradols, zingerone, terpenes and more that have antimicrobial, antioxidant, anti-inflammatory, anti-adhesive and gastroprotective properties. This review gives insight into the evidence presented on ginger extracts and ginger constituents to combat *H. pylori* and focuses on the concentration response relationship, extraction solvents, susceptibility testing, anti-biofilm activity, modulation of *H. pylori* virulence, gastric inflammation, animal evidence and limited human evidence. It also evaluates agar-well diffusion as a potential screening test, and offers a robust model for subsequent experiments. There is reproducible in-vitro evidence, potential mechanistic effects, and there are animal data of attenuation of gastric inflammation. Thus the current state of human evidence is but tentative and incomplete, and ginger is not an option for standalone eradication therapy. Further, ginger should be considered as a standardized add-on to guideline-based treatment, not a replacement for antibiotics, until studies can be conducted with adequate numbers of patients to establish sufficient evidence for efficacy in eradicating the bacteria and for their dose, safety, pharmacokinetics and possible interactions.

Keywords: Zingiberofficinale;gingerol;*Helicobacterpylori;*antimicrobialresistance;biofilm;agar-well diffusion.

Reviewapproach

This narrative review combines peer-reviewed biomedical literature, systematic reviews, mechanistic insights and clinical experience relating to the question at hand. Inference of evidence based on study design, methodological quality, biological plausibility and applicability to clinical practice. Numerical citations reference the reference list.

Key evidence map

Evidencelevel	Representativefinding	Interpretation
Invitro	Ginger extracts and gingerols inhibit *H. pylori* strains and may affect membranes, biofilms, and virulence.	Supportsbiologicalactivity;doesnot establish clinical eradication.
Animal	Standardized extracts reduced bacterial load and gastric inflammation in infected gerbils.	Supportsgastricexposureandhost effects; translation remains uncertain.
Human	A small trial reported symptom improvementandincreasednegativestool antigen results.	Preliminary;requireslargerindependent randomized trials.
Clinicalstandard	Modern eradication uses multidrug, acid-suppressive regimens with test of	Gingerisinvestigationalasanadjunct, not a replacement.

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	cure.	

I. Introduction

Helicobacter pylori is the causation agent of gastritis, a Gram-negative helical-shaped bacterium which is a microaerophilic organism adapted to the highly acidic gastric environment. Its urease activity, motility, chemotaxis, urease, and ability to change host immunity allows for long term colonization of the mucus layer and gastric epithelium. Mostly infants and children are infected and unobserved infections can last for decades. Many of those infected never experience any symptoms, but the organism has been causally associated with chronic active gastritis, the majority of gastric ulcers, a significant proportion of gastric ulcers, gastric mucosa-associated lymphoid tissue (MALT) lymphoma and non-cardia gastric adenocarcinoma [1-4]. With the carcinogenic role played by *H. pylori*, accurate diagnosis, effective eradication, and confirmation post *H. pylori* treatment have become significantly important to care of the patient.

In standard eradication, two or more antimicrobial agents will be combined with acid suppression. In many parts of the world, however, the effectiveness of historical triple therapy with clarithromycin is decreasing due to antimicrobial resistance. Current recommendations therefore prefer testing of cure at the end of the therapy and 14-day optimized bismuth quadruple therapy, or an alternative treatment regimen based on susceptibility [5]. These changes highlight the need to always view herbal products as a proposed alternative to proven eradication therapy and not as a proven alternative therapy. However, natural products could afford lead compounds, modulate virulence of the bacterial pathogen, increase the tolerability of the mucosa or increase the efficacy of the traditional drug.

Ginger is considered to be one of the most widely used medicinal rhizomes. It is being traditionally used on gastrointestinal applications, such as Nausea, Dyspepsia, Abdominal discomfort, and inflammatory effects. The effects of the phenolics are investigated in modern pharmacology, with 6-gingerol, 8-gingerol, 10-gingerol, 6-shogaol, and derivatives being cited. Whether ginger has general antimicrobial activity; whether reproducible, chemically characterized extracts can inhibit clinically important strains of *H. pylori* at concentrations likely to be attainable and safe in the stomach [5-9,16-18].

1. *H.pylori*Pathogenesis and Therapeutic Challenges

The ability of *H. pylori* to survive involves synergistic mechanisms of *H. pylori* and host interactive. Urease breaks down the stomach acid of urea and neutralizes it. Flagellar motility is used for motility in mucus. The roles of adhesins in attaching to epithelial glycoconjugates include BabA and SabA. The pathogenicity island of cag is responsible for the transport of CagA into gastric epithelial cells by a type IV secretion system which disrupts, signaling, junctional integrity, proliferation, and inflammatory responses to these cells. VacA is involved in vacuolation, in injury to mitochondria, immune modulation, and in impaired functions of epithelial cells [2-4]. All of these factors will generate multiple hypothetical avenues for phytochemical activity other than bactericidal activity.

Adverse drug effects are only one of the causes of treatment failure. Poor adherence, poor acid suppression, excessive bacterial load, re-infection, heteroresistance, and bacterial tolerance due to biofilm formation play a role in antibiotic resistance but may not be as apparent as we think. Biofilms have the ability to alter the penetration of antimicrobials and produce metabolically diverse bacterial communities. Even if the candida is merely bacteriostatic, it could also interfere with its ability to adhere, enhance the development of biofilms, inhibit urease activity, modify inflammatory signalling, or decrease gastric oxidative stress, and thus it may be beneficial [2,3,23,26,27,29].

Though careful, interpretation of plant-extract studies is needed. Diffusion as well as potency can influence the results of zone-of-inhibition assays; a hydrophobic component can have biological activity in the test materials but be diffuse in the agar. On the other hand, inhibition zones can be over-inflated due to residual solvent, low pH or contamination. The broth microdilution minimum inhibitory concentration (MIC), minimum bactericidal concentration (MBC), time-kill kinetics, solvent controls, standardized inocula, strain panels, chemical characterization and proper statistical analysis provide strong evidence [2, 3, 23, 26, 27, 29].

2. PhytochemistryofGinger and the Importance of Extraction

Dried ginger is a different type of ginger

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from the fresh ginger. The hot or dried rhizome converts gingerols to shogaols whilst heating or drying fresh rhizome can lead to formation of paradols and zingerone. The volatile oils are zingiberene, beta-sesquiphellandrene, ar-curcumene and related terpenes. These compounds readily occur depending the cultivar, geographical location, maturity, storage, drying temperature, extracting solvent, extraction period and analytical technique [6-9]. The 100% can therefore only give chemically an ambiguous description of an extract if the amount of plant material, number of mls of extraction solvent, amount of material retrieved, and marker compound content are reported.

Water extracts are clinically intuitive and relatively safe, but much more of the phenolic and lipophilic constituents are extracted by ethanol (or other organic solvents). A wider range of substance may be recovered than is possible by water alone. Solvent selection can significantly affect the measured activity as shown in published comparisons for antimicrobial activity [10]. If aqueous solutions are used for *H. pylori* studies, then ethanol extracts can be compared but ethanol must be dry or used as a control extract containing the same amount of ethanol, as ethanol could inhibit the bacteria.

For a "repeatable" preparation, the botanical identity and vouchering specimen, fresh or dry mass of the particles, solvent composition, solid to solvent ratio, temperature, time, filtration, method of concentration, yield, and method of sterility should be reported. Gingerols and shogaols must be quantified at least by HPLC or HPLC-MS. Concentration-response relationships are difficult to compare at different laboratories if not standardized [6-10].

3. DirectAntibacterialEvidenceAgainst*H.pylori*

As part of this preliminary in-vitro study, it was found that extracts from ginger root and gingerols themselves demonstrated inhibitory activity against a number of strains of *H. pylori* that had the CagA toxin (causing gastric alteration) [11]. A further study on standardized ginger extract saw a reduction of *H. pylori*-induced gastric inflammation in a Mongolian gerbil model with the selected strains showing MIC values in a low microgram-per-milliliter range [12]. These findings have been found to be good for both direct antibacterial function and host protective.

Mechanistic studies are considered to have spurred the interest in recent times, in the derivatives of 6-gingerol with similar structure. Qian and colleagues found high activity against *H. pylori* with a correlation to the destruction of the bacteria's cell walls and metabolic disturbance [13]. Steamed ginger extract was also found to suppress *H. pylori* infection and inflammatory responses in H. pylori-infected gastric epithelial models [14]. All of these studies show that processing can affect the potency due to changes in the ratios of gingerols and shogaols.

There is a great deal of variability in the evidence. The various studies employ different strains, media, incubation methods, definitions of extracts and endpoints. Some will report zones of inhibition, others will report MICs, changes in optical density, a change in colony counts, or molecular markers. These zones of slope at higher nominal extract percentage values are very suitable for overlap and work with activity concentration dependent, but they do not provide a Doses of relevance to Pharmacology! The results should be presented as mass concentration (e.g. mg/mL dry extract) and preferably as concentration of recognized marker compounds [11-14,18].

4. *ProposedMechanismsofAnti-H.pyloriActivity*

The antibacterial effect of ginger seems to be poly-targeted. Bacterial constituents, including phenols, affect the permeability of bacterial membranes, leading to leakage of both intracellular components from the bacterium and energy metabolism disturbance. Hydroxyl and carbonyl groups are able to interact with protein and membrane lipids. Other constituents of ginger are found to inhibit ATP related processes in other bacteria which suggest a possible energetic action, although this is yet to be confirmed in H. pylori [12-14,19,20,22,24].

Particularly remarkable is the anti-adhesive and anti-biofilm properties. *H. pylori* can form biofilms which can promote persistence and antimicrobial tolerance. Compounds from ginger can inhibit early attachment, ECM stability, and/or mature biofilms biomass. Future studies may be directed toward prevention of biofilm growth versus disruption of an existing biofilm as they are significantly different biological outcomes. Viable counts and microscopy should accompany analysis of biomass by Crystal-violet [12-14,19,20,22,24].

Futhermore, ginger may help to modify virulence and inflammation in the host. *H. pylori* stimulate nuclear factor-kappa b pathways and mitogen-activated protein kinase pathways which

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triggers the production of inflammatory mediators such as interleukin-8. Experimental systems indicate that the inflammatory markers are reduced in standard or steamed ginger preparations [12,14]. Antioxidant activity might help to control lipid peroxidation via ROS and inflammatory signaling pathways, which could help to inhibit mucosal injury. The action taken may help improve symptoms or damage of tissue without eradicating it entirely.

5. Concentration-ResponseTestingandInterpretation

Testing 10%, 25%, 50%, 75 and pure stock can give a preliminary gradient; percentages will have to be defined. The term 100% stock can refer to a variety of products: crude juice, reconstituted dry extract, or evaporated ethanolic extract, which are different products. It is recommended to use serial 2-fold dilutions for the determination of the MIC, due to the fact that they offer quantitative endpoints. Agar well diffusion should be used as an initial screening technique but should not be the only technique used [23-25].

To conduct a rigorous experiment, at least one *H. pylori* strain should be included as a reference and several clinical isolates with known antimicrobial susceptibility profile should be included. Inoculum should be standardized — for example, a density equivalent to a standard McFarland turbidity, which is determined by the number of counts on the plate. Plates should be incubated using microaerophilic conditions at approximately 35-37 degrees C for a specific amount of time in an *H. pylori* supportive enriched medium. Positive control should include clinically relevant antibiotics and a negative control should consist of sterile water, the same concentration of residual solvent, and an uninoculated sterility control [23-25].

Copying wells (replication) within a plate is not biologically replicating wells. Independent preparations of extract and independent bacterial cultures must be performed. The zones of inhibition should be reported as the mean, standard deviation and confidence interval. If assumptions are met, one-way ANOVA with an appropriate post-hoc test may be used, otherwise robust or non-parametric tests are recommended. A dose response model can estimate EC50 like parameters but MIC/MBC are more easily interpreted antimicrobial endpoints [23-25].

6. *HumanEvidenceandClinicalTranslation*

So far, there is limited clinical evidence. The use of ginger powder supplementation in functional dyspepsia was shown to improve dyspeptic symptoms and streptozotocin-treated stool antigen testing was also more negative in the ginger group compared to the control group in one randomized probion in *H. pylori* positive patients [15]. The study is significant because it breaks away from in-vitro activity, but the relatively small sample size and the broad 95% confidence intervals may be due to the ginger being formulated differently with different dosing, and need additional replication to draw any conclusions about the ability of ginger to eradicate *H. pylori* on its own.

It is important to influence the symptoms and eliminate the microbes. Ginger has effects on gastric motility, nausea and inflammatory symptoms which are not associated with its antibacterial properties. Persistent infection is still associated with an increased risk of ulcer and cancer and can cause the affected patient to feel well. Validated diagnostic tests before treatment and guideline concordant test-of-cure testing after a proper interval after treatment – avoiding pump inhibitors or antibiotics for appropriate TOT pre-test periods should be performed in future trials [5,15,26].

For the time being, the most readily argued clinical position is as an adjunct. Trials might involve comparing ginger to placebo and eradication as primary end points, while adherence rates, adverse events, dyspepsia scores, inflammatory markers and microbiome effects are recorded as secondary end points. These trials should be powered for the ability to detect clinically meaningful difference, and should prespecify rescue treatment for treatment failure [5,15,26].

7. *Safety,QualityControl,andInteractions*

However, typically ginger is safe to take in the usual amounts found in cooking, but highly concentrated products may lead to heartburn, abdominal discomfort, bloating or diarrhea. The quality of products produced is also variable and issues of contamination, adulteration, incorrect botanical identification and unstable marker levels are possible. Highly concentrated extracts should not be considered "safe" based on traditional culinary applications [16,7,8].

Any potential interactions should be considered, especially with patients taking an antiplatelet agent or an anticoagulant drug, a glucose-lowering agent, or a drug that is influenced

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by gastrointestinal motility. There is conflicting evidence regarding clinically relevant bleeding interactions but conservative monitoring should be considered in high risk persons. Special attention for pregnant patients, patients with severe underlying diseases, and surgical patients pending surgery [7,8,16].

Gingivitis should NOT be used as an excuse not to diagnose an alarm symptom, including gastrointestinal bleeding, loss of weight, increasing weight, anemia, dysphagia or abdominal mass. Nor should it be the substitute for evidence based eradication in a confirmed infection [7,8,16].

8. RecommendedExperimentalProtocol

Prospective research can be enriched with a sequential design. Identification of the chemical constituents of 70% Ethanolic and Aqueous ginger extract is to be done in Phase 1. Phase 2 should try agar-well diffusion as a screening test. The sterile broth/agar dilution method of standardising *H. pylori* should be used for the detection of MIC and MBC in phase 3. Time-kill activity, urease inhibition capability, adherence, prevention of formation/dissolution of biofilms, toxicity to protozoa, and interactions with certain antibiotic(s) should be assessed for Phase 4. Cytotoxicity is to be assessed in gastric epithelial cells for Phase 5 and a selectivity index is to be calculated [23-25].

However, the diffusion assay is only possible if both the volume and diameters of the wells are equal. Extract concentrations should only be reported in mg/mL, not percentages. The ethanol extract must be evaporated and then brought back into solution in a defined solvent (vehicle). Vehicles-only control is a must. Measurement should be blind or use digital image analysis if the presence of pigment or turbidity may prevent accurate endpoint determination. Apparent inhibition zones can be confirmed with subculture in a confirmatory culture [23-25] which can help differentiate bacteriostatic from bactericidal effects.

The checkerboard assay, fractional inhibitory concentration indices (FICI) and time-kill testing are important to consider when evaluating synergy. Larger diffusion zone claims should not be given the first bite of the cherry by themselves. In the case of the ultimate goal being therapeutic use, it is important to consider the simulated gastric conditions, low pH stability and interaction with mucus, as well as intragastric levels achieved during treatment [23-25].

9. Limitations of the Evidence

However, the existing literature suffers from heterogeneity, small assay size, inconsistent methodologies, incomplete reporting of phytochemicals and publication bias for positive results. This may not be achievable in vivo at in-vitro concentrations. Animal models can be used to model specific components of infection but can't confidently predict eradication in humans. Conflicting recommendations exist from the small human evidence base for optimal formulation, dose, persistence and for compatibility with current eradication treatment regimens [11-18,23,26].

Another conceptual limitation has to do with the other dimensions of therapy: inhibition of bacterial growth is only one therapeutic dimension. A useful adjunct should be tolerated in the stomach, get to the mucous niche, be not harmful to the host, and not interfere with the absorption and adherence of antibiotics. On the other hand, if only symptomatic treatment is given, the underlying infection could remain undetected. These restrictions dictate ongoing research; but it must be noted that careful language has to be employed in abstracts and conclusions [11-18,23,26].

II. Conclusion

Chemically plausible and experimentally active compounds in ginger are active against *H. pylori* active. Concentration dependent in-vitro inhibition, activity of gingerols and processed ginger derivatives, attenuation of inflammatory signaling, and anti-biofilm activity has been demonstrated. The initial animal and preliminary human studies show promise but aren't conclusive.

Based on available data, it is possible to conclude that standardized ginger preparations have a potential to develop as complementary agents and/or source of anti-*H. pylori* lead compounds. They are not at the present time acceptable as stand-alone treatment. Rigorous phytochemical standardization, quantitative susceptibility testing, mechanistic assay research, and adequate-powered RCTs in addition to guideline based therapy should be taken together for future research.

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