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A Polyphenol-Rich Ginger, Licorice and Pomegranate Blend Strengthens Gatrointestinal Antioxidant Defenses While Preserving Gastric Acid Homeostasis

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Abstract: Food-derived botanical combinations may modulate gastrointestinal physiology through complementary effects on oxidative homeostasis and digestive function. This study investigated the effects of an oral blend of ginger, liquorice and pomegranate extracts in healthy male rats. Eighteen animals were randomly allocated to vehicle control, 50 mg/kg blend and 100 mg/kg blend groups and treated orally for 14 days. Body weight, food intake, gastric volume and acidity, serum and gastric tissue malondialdehyde (MDA), antioxidant enzyme activities, glutathione status, digestive enzyme activities and gastric histology were evaluated. The blend produced no mortality or treatment-related adverse changes in final body weight, gastric volume, free acidity or total acidity. Food intake showed a modest increase in both treatment groups. Serum MDA was reduced by 44.5% and 58.9% at 50 and 100 mg/kg, respectively, while gastric tissue MDA was also lower than that in the control group. Serum and intestinal superoxide dismutase, catalase, glutathione peroxidase and glutathione reductase activities increased following blend administration, with generally greater responses at 100 mg/kg; intestinal glutathione levels were also increased. Gastric pepsin activity increased at 100 mg/kg, whereas trypsin, chymotrypsin, amylase and lipase activities were not significantly altered. Gastric histological architecture remained preserved across all groups. Overall, the ginger–liquorice–pomegranate blend enhanced endogenous antioxidant defence and produced selective effects on gastric digestive activity without materially altering gastric secretory parameters or major small-intestinal digestive enzyme activities. These findings support further investigation of this botanical combination as a food-derived strategy for modulating gastrointestinal oxidative and digestive responses.

Keywords: Functional food; ginger; Zingiber officinale; liquorice; Glycyrrhiza glabra; pomegranate; Punica granatum; gastric mucosa; oxidative stress; digestive enzymes.

1. Introduction

The gastrointestinal tract is continuously exposed to dietary constituents, gastric acid, digestive enzymes, microbial metabolites and other potentially damaging factors. Maintenance of gastrointestinal integrity therefore depends on a dynamic balance between aggressive and protective mechanisms. Major protective processes include mucus and bicarbonate secretion,

maintenance of epithelial integrity and renewal, adequate mucosal blood flow, and endogenous antioxidant defence.[1,2] Disruption of this balance can compromise the gastric mucosa and contribute to gastritis, peptic ulceration and other gastrointestinal disorders. Oxidative stress and inflammation are important components of these pathological processes, as excessive generation of reactive oxygen species can damage cellular membranes, proteins and other biological structures and can weaken mucosal defence.[1]

Dietary botanicals have attracted increasing interest as potential sources of bioactive compounds capable of supporting gastrointestinal function. Unlike single pharmacological agents, botanical preparations contain diverse phytochemicals that may influence multiple biological pathways simultaneously. Ginger (Zingiber officinale Roscoe) is a widely used dietary spice and medicinal plant containing gingerols, shogaols and related phenolic compounds. Experimental and clinical studies have reported that ginger and its constituents can influence gastric motility, digestive function and nausea, while also exhibiting antioxidant and anti-inflammatory activities.[3–6] These effects suggest that ginger may contribute to gastrointestinal homeostasis through both functional and redox-related mechanisms.

Liquorice (Glycyrrhiza glabra L.) is another traditionally used botanical with a long history of application in gastrointestinal health. Its principal bioactive constituents include glycyrrhizin, glycyrrhetinic acid and several flavonoids. Experimental evidence suggests that liquorice-derived compounds may influence gastric mucosal defence by supporting mucus production, reducing oxidative damage and modulating inflammatory responses.[7–9] Such activities may be relevant to the maintenance of the gastric barrier, particularly under conditions in which endogenous protective mechanisms are challenged.

Pomegranate (Punica granatum L.) is particularly rich in polyphenolic compounds, including ellagitannins, punicalagin, anthocyanins and related phenolics. These constituents possess substantial antioxidant and anti-inflammatory properties and have been investigated for their potential effects on gastrointestinal tissues.[10,11] Pomegranate-derived polyphenols may reduce oxidative stress and influence cellular signalling pathways associated with inflammation and tissue protection. Collectively, the reported activities of ginger, liquorice and pomegranate provide a biological rationale for investigating their combined use as a food-derived botanical formulation.

A combination of these extracts could potentially influence several complementary components of gastrointestinal defence. Ginger may contribute to digestive motility and regulation of oxidative and inflammatory pathways, liquorice may support the mucosal mucus barrier and gastric defence, while pomegranate polyphenols may provide additional antioxidant and anti-inflammatory effects. Such complementary activities provide a rationale for developing a multi-botanical formulation rather than relying on a single plant extract. However, the biological activity of a combination cannot be predicted solely from the established effects of its individual components. Interactions among phytochemicals, differences in extract composition, relative proportions, dose, bioavailability and the physiological context may modify the overall response. A combined formulation may therefore produce additive, synergistic or even attenuated effects compared with its individual constituents. Direct experimental evaluation of the complete formulation is consequently required.

In the present study, a formulation containing extracts of ginger, liquorice and pomegranate was evaluated in healthy male rats. The investigation was designed to provide an initial functional assessment of the formulation following 14 days of oral administration. The study examined a range of parameters relevant to gastrointestinal and systemic physiology, including food intake, gastric secretory indices, gastric wall protection, antioxidant status in serum and intestinal tissue, digestive enzyme activity and microscopic integrity of the gastric tissue. Assessment of these endpoints allowed the formulation to be evaluated from complementary physiological, biochemical and histological perspectives rather than through a single outcome measure.

The use of healthy animals was intentional because the primary objective was to determine whether repeated administration of the botanical combination could influence normal gastrointestinal function and endogenous protective mechanisms. Evaluation under physiological conditions can provide preliminary evidence regarding functional activity and biological tolerance before investigation in pathological models. Changes in gastric secretion, antioxidant defence or digestive enzyme activity may indicate biological effects of the formulation even in the absence of experimentally induced gastrointestinal injury.

Importantly, the present work was designed as an initial functional assessment and should not be interpreted as a therapeutic gastroprotection study. No ulcer-induction, chemically induced mucosal injury or experimentally triggered inflammatory challenge was employed. Consequently, the findings cannot establish whether the formulation prevents or treats established gastric ulcers, gastritis or other pathological gastrointestinal conditions. Instead, the study addresses whether short-term oral administration of the ginger–liquorice–pomegranate combination is associated with measurable changes in gastric physiology, mucosal defence, oxidative balance, digestive activity and tissue integrity in healthy animals. This distinction is essential when interpreting the biological significance of the findings and provides an appropriate basis for subsequent studies using disease-relevant models.

2. Materials and methods

2.1 Test formulation

The test product was a blend of ginger extract containing 40–50% gingerol, liquorice extract at 20–30%, and pomegranate extract at 5–10%. Batch 001 was manufactured on 10 October 2024 and had a stated shelf life of two years.

2.2 Animals and housing

Eighteen male albino rats weighing 120 ± 10 g were obtained from Saha Enterprise, Kolkata, India. Animals were housed in six cages under standard laboratory conditions at 25 ± 2 °C, 50–70% relative humidity and a 12 h light and 12 h dark cycle. They were acclimatised for seven days and received standard pellet diet and water ad libitum.

2.3 Experimental design and treatment

Animals were randomly divided into three groups of six. The control group received vehicle orally for 14 days. The low-dose group received 50 mg/kg body weight per day and the high-dose group received 100 mg/kg body weight per day. On day 15, animals were euthanised using humane procedures. Blood, gastric contents, stomach and small intestinal tissue were collected. Gastric contents were stored at −20 °C, and a section of stomach was fixed in Bouin's solution for histology.

2.4 Gastric and general health measurements

Body weight and food intake were monitored during the experiment. Gastric volume, pH, free acidity and total acidity were assessed according to the method of Devi et al.[12] Ulcer score was evaluated using the scoring approach of El-Ashmawy et al.[13] and gastric wall mucus was assessed by Alcian blue binding.[14] Mortality and visible signs of intolerance were recorded.

2.5 Oxidative stress and antioxidant measurements

Malondialdehyde (MDA) was measured in serum and gastric tissue as an index of lipid peroxidation.[15] Superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) and glutathione reductase (GR) activities were measured in serum and small intestinal tissue using established spectrophotometric methods.[16–19] Reduced glutathione (GSH) was determined using the method of Moron et al.[20]

2.6 Digestive enzyme activity

Pepsin activity was measured in stomach tissue. Trypsin, chymotrypsin, amylase and lipase activities were measured in small intestinal mucosa using established spectrophotometric or enzymatic procedures.[21–25]

2.7 Histological assessment

Bouin-fixed stomach samples were processed for light microscopy. The mucosa, submucosa, muscularis propria and serosa were examined qualitatively for structural change, inflammation, ulceration and tissue damage at ×200 magnification. An additional magnified view was used to examine the mucosal surface.

2.8 Ethical considerations

The study was approved by the Institutional Animal Ethics Committee of Vidyasagar University and was conducted according to the requirements of the Committee for the Purpose of Control and Supervision of Experiments on Animals, Government of India. Animal welfare and reporting principles were followed in accordance with the ARRIVE 2.0 guidelines.26

2.9 Statistical analysis

Values are presented as mean ± standard error of the mean (SEM), with six animals per group. Between-group differences were evaluated by one-way analysis of variance followed by Dunnett's multiple-comparison test. Within-group differences between day 0 and day 14 were evaluated using paired t-tests. A two-sided P value below 0.05 was considered statistically significant. Statistical analysis was performed using SPSS. Percentage changes were calculated descriptively from the group means.

3. Results

3.1 Body weight, food intake and survival

All 18 animals completed the 14-day intervention and no mortality was recorded. Baseline and final body weight were comparable across groups. Food intake was higher than control by 5.3% at 50 mg/kg and 8.4% at 100 mg/kg. These differences were significant at $P < 0.05$ and $P < 0.001$, respectively. The increase in food intake was not accompanied by a significant between-group difference in final body weight.

*Fig. 1 Body weight and daily food intake. Bars show mean ± SEM (n = 6). *P < 0.05 and ***P < 0.001 versus control.*

Values are mean ± SEM, n = 6. *P < 0.05 and ***P < 0.001 versus control after one-way ANOVA with Dunnett's comparison.

Group	Body weight, day 0 (g)	Body weight, day 14 (g)	Food intake (g/rat/day)
Control	121.33 ± 1.28	127.16 ± 1.05	11.97 ± 0.16
50 mg/kg	122.66 ± 2.36	128.83 ± 1.33	12.60 ± 0.16*
100 mg/kg	123.83 ± 2.15	129.16 ± 1.03	12.98 ± 0.19***

Table 1. Body weight and food intake after oral administration of the botanical blend.

3.2 Gastric secretory parameters and mucosal observations

Ulcer score was zero in every group. Gastric volume, free acidity and total acidity did not differ significantly between treated and control animals. Gastric pH increased significantly at both doses, and Alcian blue-bound gastric wall mucus showed a dose-related increase. Histological examination showed intact gastric layers in all groups, without visible ulceration or treatment-related structural damage.

Group	Ulcer score	Gastric volume (mL)	Free acidity (µEq/L)	Total acidity (µEq/L)
Control	0	1.55 ± 0.03	29.10 ± 0.31	40.23 ± 0.24
50 mg/kg	0	1.54 ± 0.03	28.30 ± 0.70	39.50 ± 0.38
100 mg/kg	0	1.54 ± 0.02	28.06 ± 0.79	39.40 ± 0.40

Table 2. Gastric secretory parameters at the end of treatment.

Values are mean ± SEM, n = 6. No significant between-group differences were observed for gastric volume, free acidity or total acidity.

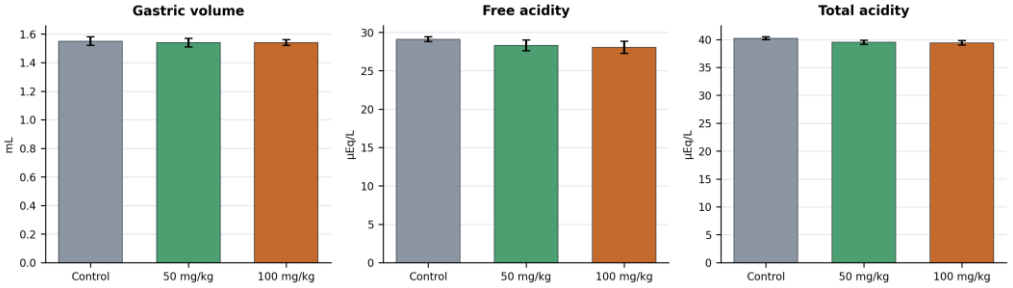

Fig. 2 Gastric volume, free acidity and total acidity after 14 days. Bars show mean ± SEM (n = 6). No significant differences were observed.

3.3 Lipid peroxidation

Serum MDA changed from 9.98 ± 0.20 to 5.54 ± 0.40 nM/mL at 50 mg/kg and from 8.02 ± 0.19 to 3.30 ± 0.30 nM/mL at 100 mg/kg. These values represent reductions of 44.5% and 58.9%, respectively. Control serum MDA increased by 2.7%. Gastric tissue MDA at day 14 was 27.9% lower than control in the 50 mg/kg group and 67.5% lower in the 100 mg/kg group. Both treated groups differed from control at P < 0.001 for serum and gastric tissue MDA at day 14.

Group	Serum MDA, day 0 (nM/mL)	Serum MDA, day 14 (nM/mL)	Within-group P	Gastric MDA, day 14 (nM/mL)
Control	8.29 ± 0.18	8.51 ± 0.37	0.011	5.38 ± 0.46
50 mg/kg	9.98 ± 0.20	5.54 ± 0.40***	0.0004	3.88 ± 0.38***
100 mg/kg	8.02 ± 0.19	3.30 ± 0.30***	<0.0001	1.75 ± 0.22***

Table 3. Serum and gastric tissue malondialdehyde concentrations.

Values are mean ± SEM, n = 6. Within-group P values were calculated using paired t-tests. ***P < 0.001 versus control at day 14.

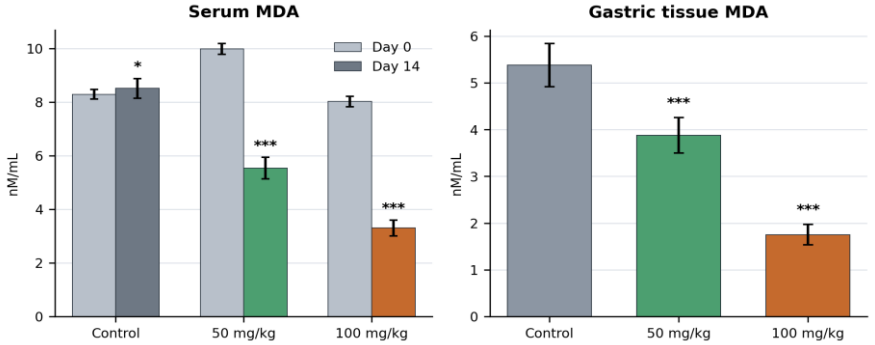

*Fig. 3 Serum and gastric tissue MDA. Bars show mean ± SEM (n = 6). *P < 0.05 for the within-group control change; ***P < 0.001 for treated-group day 14 values. Day 0 and day 14 are shown for serum; gastric tissue was evaluated at day 14.*

3.4 Superoxide dismutase and catalase

Serum SOD and CAT remained stable in controls. At 50 mg/kg, serum SOD increased by 8.0% and CAT by 13.0% from baseline. At 100 mg/kg, the corresponding increases were 38.6% and 42.2%. Intestinal SOD was 14.9% and 31.8% above control at 50 and 100 mg/kg, while intestinal CAT was 14.7% and 94.2% above control. The response was generally greater at the higher dose.

Outcome	Control	50 mg/kg	100 mg/kg
Serum SOD, day 0 (U/mL)	9.52 ± 0.24	10.44 ± 0.38	10.25 ± 0.36
Serum SOD, day 14 (U/mL)	9.55 ± 0.23	11.28 ± 0.13***	14.21 ± 0.36***
Serum CAT, day 0 (mol/s/L)	37.69 ± 1.85	39.28 ± 1.10	40.33 ± 0.95
Serum CAT, day 14 (mol/s/L)	37.87 ± 1.84	44.38 ± 1.18*	57.34 ± 1.60***
Intestinal SOD, day 14 (U/mg issue)	12.37 ± 0.29	14.21 ± 0.39**	16.30 ± 0.44***
Intestinal CAT, day 14 (μM H_2O_2 consumed/mg/min)	67.51 ± 2.22	77.45 ± 2.42*	131.1 ± 1.89***

Table 4. Serum and intestinal superoxide dismutase and catalase activity.

Values are mean ± SEM, n = 6. *P < 0.05, **P < 0.01 and ***P < 0.001 versus control after one-way ANOVA with Dunnett's comparison.

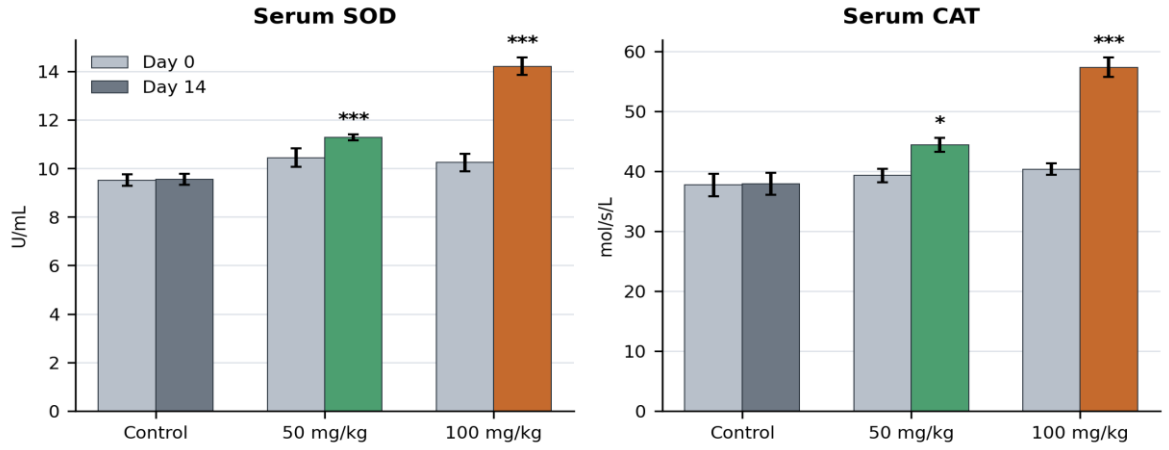

*Fig. 4 Serum SOD and CAT before and after treatment. Bars show mean ± SEM (n = 6). *P < 0.05 and ***P < 0.001 versus control at day 14.*

3.5 Glutathione-dependent antioxidant system

Serum GPx, GR and GSH remained stable in control animals. At 50 mg/kg, GPx increased by 36.8%, GR by 20.1% and GSH by 14.2% from baseline. At 100 mg/kg, GPx increased by 125.2%, GR by 101.0% and GSH by 16.3%. In intestinal tissue, both doses increased GPx, GR and GSH relative to control. The high dose produced the largest values for all three tissue measures.

Outcome	Control	50 mg/kg	100 mg/kg
Serum GPx, day 0 (U/mL)	2.70 ± 0.14	2.28 ± 0.10	2.78 ± 0.23
Serum GPx, day 14 (U/mL)	2.67 ± 0.15	3.12 ± 0.08	6.26 ± 0.17***
Serum GR, day 0 (U/mL)	6.26 ± 0.08	6.23 ± 0.16	6.28 ± 0.06
Serum GR, day 14 (U/mL)	6.39 ± 0.17	7.48 ± 0.21**	12.62 ± 0.28***
Serum GSH, day 0 (μM/L)	7.43 ± 0.14	7.18 ± 0.24	7.44 ± 0.19
Serum GSH, day 14 (μM/L)	7.51 ± 0.15	8.20 ± 0.24	8.65 ± 0.24**
Intestinal GPx, day 14 (U/mg tissue)	5.57 ± 0.21	7.55 ± 0.22***	9.34 ± 0.31***
Intestinal GR, day 14 (U/mg tissue)	15.58 ± 0.53	26.03 ± 0.45***	33.46 ± 0.74***
Intestinal GSH, day 14 (μM/mg tissue)	5.32 ± 0.16	7.52 ± 0.30***	10.83 ± 0.21***

Table 5. Serum and intestinal glutathione-related antioxidant measures.

Values are mean ± SEM, n = 6. **P < 0.01 and ***P < 0.001 versus control after one-way ANOVA with Dunnett's comparison.

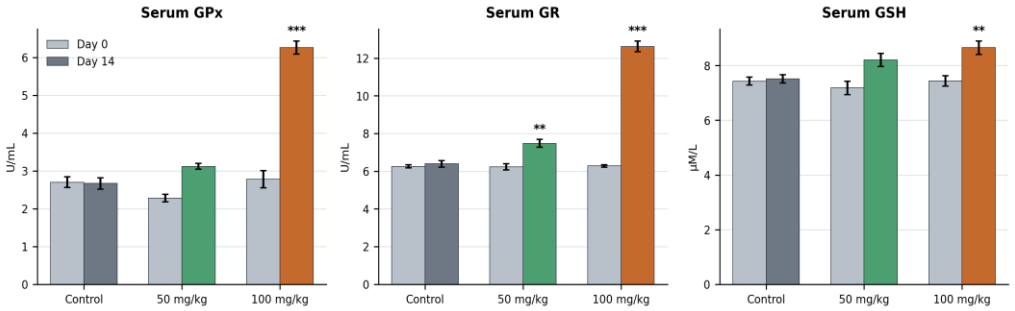

*Fig. 5 Serum GPx, GR and GSH before and after treatment. Bars show mean ± SEM (n = 6). **P < 0.01 and ***P < 0.001 versus control at day 14.*

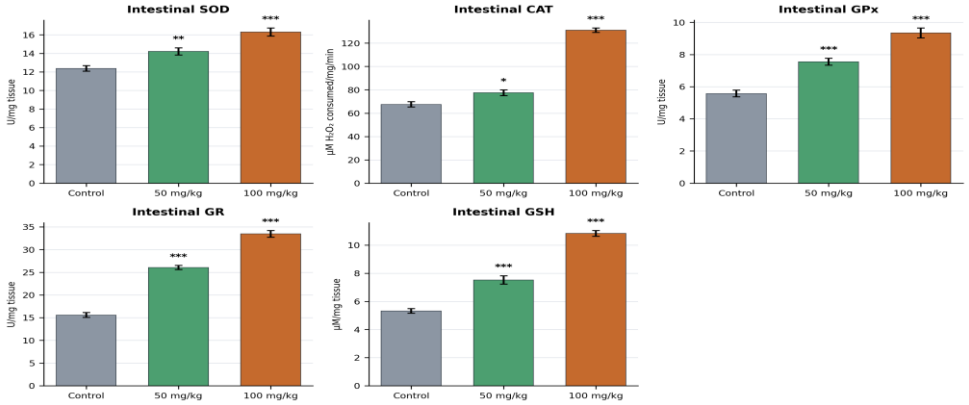

*Fig. 6 Intestinal SOD, CAT, GPx, GR and GSH after 14 days. Bars show mean ± SEM (n = 6). *P < 0.05, **P < 0.01 and ***P < 0.001 versus control.*

3.6 Digestive enzyme activity

Gastric pepsin activity was 171.4% higher than control at 100 mg/kg and the difference was significant. The small increase at 50 mg/kg was not significant. Trypsin, chymotrypsin, amylase and lipase activities did not differ significantly among groups. Thus, the response was selective for gastric pepsin under the conditions examined and did not indicate broad stimulation or suppression of small intestinal digestive enzymes.

Group	Pepsin (U/mg protein)	Trypsin (U/g protein)	Chymotrypsin (U/mg protein)	Amylase (U/g protein)	Lipase (U/mg protein)
Control	0.14 ± 0.002	30.43 ± 1.81	0.12 ± 0.003	477.70 ± 3.25	69.09 ± 1.62
50 mg/kg	0.15 ± 0.003	30.83 ± 1.54	0.13 ± 0.006	485.90 ± 9.28	68.54 ± 1.83
100 mg/kg	0.38 ± 0.006*	31.11 ± 0.90	0.14 ± 0.002	485.70 ± 5.30	68.79 ± 1.17

Table 6. Digestive enzyme activity in stomach and small intestinal tissue.

Values are mean ± SEM, n = 6. *P < 0.05 versus control. No significant differences were observed for the four small intestinal enzymes.

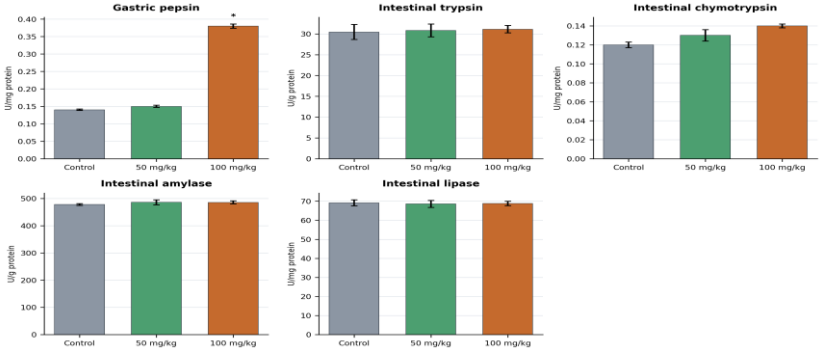

*Fig. 7 Gastric and small intestinal digestive enzyme activity after treatment. Separate panels are used because the enzyme assays have different units and scales. Bars show mean ± SEM (n = 6). *P < 0.05 versus control for pepsin.*

4. Discussion

The principal finding of the present study was that 14-day oral administration of the ginger, liquorice and pomegranate extract blend produced coordinated changes in biochemical markers associated with gastric mucosal defence and redox homeostasis without inducing macroscopic gastric injury or materially altering the major gastric secretory parameters [19]. The magnitude of the response was generally greater at 100 mg/kg than at 50 mg/kg, particularly for serum and gastric and intestinal tissue malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) and glutathione reductase (GR). The formulation additionally increased gastric pepsin activity at the higher dose, whereas the principal small-intestinal digestive enzymes remained comparable with the control group [20]. Collectively, these findings suggest that the formulation exerted a predominantly mucosal and redox-modulatory effect rather than acting as a potent inhibitor of gastric acid secretion or a broad stimulant of digestive enzyme activity.

The absence of significant changes in gastric volume, free acidity and total acidity is particularly relevant for interpreting the gastroprotective profile of the formulation. These findings indicate that the observed effects cannot be attributed primarily to suppression of gastric acid production during the 14-day treatment period [21]. Instead, the increase in gastric pH together with enhanced gastric wall mucus suggests modulation of the luminal and pre-epithelial components of the mucosal defence system. Gastric mucosal protection is maintained through a multilayered barrier involving the mucus–bicarbonate layer, epithelial integrity, adequate mucosal perfusion, rapid epithelial restitution and endogenous antioxidant and cytoprotective mechanisms. The present findings are therefore more consistent with reinforcement of defensive mechanisms than with classical antisecretory activity. Liquorice-derived constituents have been associated with enhancement of mucus secretion and mucosal protection, whereas ginger phenolics, particularly gingerols and shogaols, and pomegranate-derived ellagitannins and their metabolites have been reported to modulate oxidative stress, inflammatory signalling and cellular antioxidant responses[23].The simultaneous presence of these phytochemical classes provides a plausible basis for complementary effects on different components of gastrointestinal defence. Nevertheless, because the present study evaluated the formulation as a combination, the relative contribution of individual extracts and the possibility of pharmacological additivity or synergy cannot be established [24].

5. Conclusion

The present study demonstrates that the oral combination of ginger, liquorice, and pomegranate extracts exerts a multifaceted effect on gastrointestinal physiology, particularly through modulation of antioxidant defence and gastric functional activity. Fourteen days of treatment did not produce apparent gastric mucosal injury or adverse alterations in the evaluated secretory parameters, supporting the preliminary gastrointestinal tolerability of the formulation under the experimental conditions. The formulation significantly modulated serum and gastric tissue malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase (GR), indicating an influence on oxidative stress and endogenous antioxidant defence. The comparatively greater response at 100 mg/kg suggests a dose-associated biological effect, while the increase in gastric pepsin activity at this dose indicates selective modulation of gastric digestive function. In contrast, the absence of major changes in small-intestinal digestive enzymes suggests that the formulation does not broadly disrupt digestive enzyme activity. Collectively, these findings support the potential of the combined botanical formulation to enhance gastrointestinal antioxidant protection while maintaining physiological digestive function. Future studies should establish detailed dose–response relationships, long-term safety, and mechanisms involving Nrf2/HO-1, NF-κB, inflammatory mediators, mucosal integrity, mucus and bicarbonate secretion, and tight-junction proteins. Evaluation in disease-relevant models, including NSAID-induced gastric injury and ulceration, together with histopathological and molecular analyses, is warranted. Comparative studies of individual versus combined extracts should determine possible synergistic interactions. Phytochemical standardization, pharmacokinetic characterization, and appropriately designed clinical trials will ultimately be required to establish reproducibility, safety, efficacy, and translational potential of the formulation for gastrointestinal health.

Conflicts of interest

None

Data availability

The numerical summary data supporting the findings are presented in Tables 1–6.

Ethics statement

The protocol was approved by the Institutional Animal Ethics Committee of Vidyasagar University and complied with CPCSEA requirements for the care and use of laboratory animals.

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