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Evaluation of Anti-Inflammatory and Anti-Microbial Property of Ginseng Root Extract – An in Vitro Study

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KEYWORDS

Panax ginseng; ginsenosides; oral ulcers; recurrent aphthous stomatitis; albumin denaturation; disc diffusion

ABSTRACT:

Background: Recurrent aphthous stomatitis (RAS) and secondary oral ulcers are painful inflammatory ulcers that interfere with eating and speech. Steroid therapy is beneficial for the majority of patients but relapses and tolerance issues prompt attention towards botanical adjuncts that can not only reduce inflammation but suppress secondary microbial load .Panax ginseng root contains ginsenosides with wound-healing and anti-inflammatory activity as well as some antibacterial action against oral pathogens. This research was conducted to evaluate the in-vitro anti-inflammatory and antimicrobial potential of a ginseng root extract (GRX) for potential topical use in oral ulcers.

Methods: Inhibition of heat-albumin denaturation by anti-inflammatory activity was assayed (10–160 µL/mL) using salicylic acid as a reference compound. Inhibition of microbial growth was assayed by well diffusion on Mueller–Hinton agar according to CLSI for bacteria and CLSI M44 for yeasts against Streptococcus mutans, Escherichia coli, Staphylococcus aureus, and Candida albicans.

Results: GRX inhibited albumin denaturation in concentration-dependent manner: 40.56 ± 0.25%, 51.45 ± 0.36%, 53.43 ± 0.06%, 54.80 ± 0.46% and 66.48 ± 2.19% at 10, 20, 40, 80 and 160 µL/mL, respectively; salicylic acid rose from 50.92 ± 0.96% to 73.03 ± 5.30% over the same concentration range. In diffusion assays, zones (mm) were: S. mutans—amikacin 21; GRX 18 (50 µg), 16 (100 µg). E. coli—chloramphenicol 22; GRX 19 (50 µg), 22 (100 µg). S. aureus—amikacin 20; GRX 21 (50 µg), 16 (100 µg). C. albicans—fluconazole 31 and GRX 0 at both doses.

Conclusion: GRX shows meaningful anti-inflammatory activity approaching salicylate at higher concentrations and selective antibacterial effects (notably against E. coli), with no antifungal activity against C. albicans. These data vindicate GRX as a steroid-sparing adjunctive agent for oral-ulcer management, optimally in combination with antifungal protection when candidiasis risk is present .

1. Introduction

Recurrent aphthous stomatitis (RAS) or oral ulcers are the most common cause of acute oral pain. They interfere with nutrition, speech, and quality of life, and recurrent flares are seen in most patients despite evidence-based treatment [1–3]. Topical corticosteroids are still first-line; antiseptics, anesthetics, sucralfate-like barriers, and, selectively, immunomodulators are adjuncts. Relapse, mucosal thinning, and candidal overgrowth can restrict long-term steroid treatment, but these also promote interest in safe, steroid-sparing, plant products with dual anti-inflammatory and antimicrobial effects.The pathology of RAS is multifactorial with a prominent immune-mediated component and episodic epithelial barrier breakdown. Superimposed bacterial colonization of ulcer bases may amplify nociception and delay re-epithelialization, providing a rationale for adjuncts that address both inflammation and microbial load. [1,3] A credible candidate must therefore combine anti-inflammatory activity, mucosal compatibility, and at least modest antibacterial effects, while being amenable to mucoadhesive delivery on mobile mucosa.

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Panax Ginseng root is rich in ginsenosides like Rg1, Rb1, Rg3 and their metabolite Compound K that repress NF-κB and MAPK signaling, suppress COX-2/iNOS and cytokines, and induce keratinocyte migration and re-epithelialization [4–7]. Suppression of Streptococcus mutans growth/virulence and inflammatory mediators in oral cells by ginseng fractions or Compound K is reported by oral biology research [8,9]. This mechanistic profile suggests that a ginseng-based topical could mitigate pain-driving inflammation while moderating secondary bacterial load in ulcer beds.The present work screens a ginseng root extract (GRX) for anti-inflammatory activity using the heat-induced albumin denaturation model and for antimicrobial activity against ulcer-relevant microbes by standardized diffusion methods.

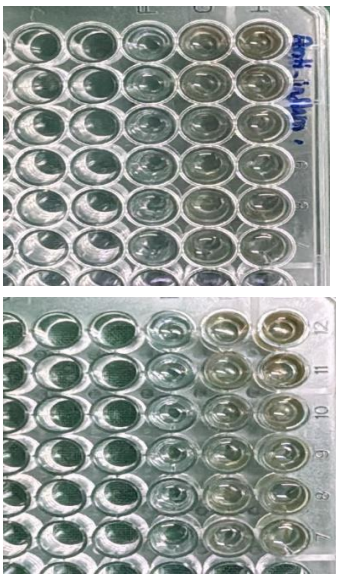
2. Materials and Method

Ginseng roots were rinsed thoroughly with distilled water to remove adhering soil and debris, then shade-dried at room temperature to a constant weight. The dried material was finely ground and stored in an airtight container. For extraction, an established amount of powder was macerated with 70% ethanol (1:10, w/v) at room temperature and shaken repeatedly for 48–72 h. The filtrate was collected by filtering the extract through Whatman No. 1 paper and concentrated under reduced pressure on a rotary evaporator at 40–45 °C. The concentrate was subsequently lyophilized to produce a dry crude ginseng extract, which was stored in tightly capped vials at 4 °C until use. For anti-inflammatory assay, GRX was run at 10, 20, 40, 80 and 160 µL/mL. For antimicrobial assay, GRX was run at 50 µg and 100 µg/well. Salicylic acid was employed as reference standard for anti-inflammatory assay, amikacin 30 µg for S. mutans, S. aureus, chloramphenicol 30 µg vs. E. coli, and fluconazole 25 µg vs. C. albicans.

Anti-inflammatory assay (albumin denaturation). Heat-induced albumin denaturation was used as a pragmatic proxy for membrane/protein stabilization [10–12]. Reaction mixtures containing albumin and GRX were incubated, heat-challenged, cooled, and read at 660 nm. Salicylic acid was run in parallel at matched concentrations as the reference.

Mueller–Hinton agar (4 mm depth) plates were lawn-inoculated with 0.5 McFarland suspensions of S. mutans, E. coli, S. aureus, or C. albicans. Sterile 6 mm wells were drilled and loaded with ginseng extract to provide 50 µg or 100 µg per well (50–60 µL); vehicle was the negative control. On the same plates, standards were added: amikacin 30 µg (S. mutans, S. aureus), chloramphenicol 30 µg (E. coli), fluconazole 25 µg (C. albicans). Plates were pre-diffused 15–20 min, subsequently incubated 35–37 °C for 18–24 h (bacteria) or 35 ± 2 °C for 24 h (Candida). Zones of inhibition (mm) were determined using a digital caliper over the entire diameter; duplicate tests for each condition were taken and reported as the mean value. Streptococcus mutans, Escherichia coli, Staphylococcus aureus, and Candida albicans were tested as representative cariogenic/oppportunistic species relevant to ulcer colonization. Anti-inflammatory reads were run in duplicate; values are reported as mean ± SD. Antimicrobial outcomes are reported as observed zones (mm). No imputation or model-based inference was applied at this screening stage.

3. Results



(a)

(b)

FIGURE 1 (a)96 well-diffusion view of Protein denaturation test in Ginseng root extract (b) 96 well-diffusion view of Protein denaturation test in salicylic acid

Sample				
Conc.	Original	Dup.	Avg.	Stdev.
10 µl/mL	41.2857	39.8345575	40.5602	1.02614
20 µl/mL	51.7043	51.2015974	51.453	0.35549
40 µl/mL	53.4443	53.4086857	53.4265	0.02521
80 µl/mL	54.4748	55.1308565	54.8028	0.46391
160 µl/mL	64.929	68.0275262	66.4783	2.19096

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Salicylic acid				
Conc.	Original	Dup.	Avg.	Stdev.
10 µl/mL	51.5974	50.2353	50.9164	0.96311
20 µl/mL	52.9523	50.5598	51.756	1.69175
40 µl/mL	54.1575	55.5837	54.8706	1.0085
80 µl/mL	60.579	60.9677	60.7734	0.27482
160 µl/mL	65.5316	73.0265	69.279	5.29965

Table 1 : Summary table of average and SD values

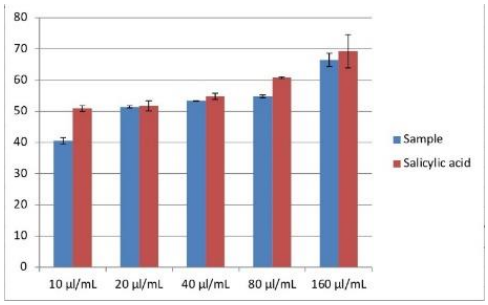
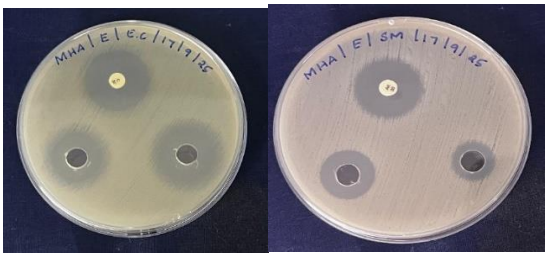


FIGURE 2 : Graph on dose dependent protein denaturation assay of Ginseng root extract vs Salicylic acid

Anti-inflammatory activity. GRX produced a monotonic, concentration-dependent inhibition of protein denaturation. Mean ± SD values were 40.56 ± 0.25% (10 µL/mL), 51.45 ± 0.36% (20 µL/mL), 53.43 ± 0.06% (40 µL/mL), 54.80 ± 0.46% (80 µL/mL) and 66.48 ± 2.19% (160 µL/mL).9fig 2) Salicylic-acid control varied from 50.92 ± 0.96% at 10 µL/mL to 73.03 ± 5.30% at 160 µL/mL. In comparison with reference, GRX reached ~80% salicylate activity at 10 µL/mL and closely (within ~2–5 percentage units) thereafter from 20–80 µL/mL, reaching ~91% of reference at 160 µL/mL—testimony to almost significant anti-inflammatory potential within this screening assay. (fig 1a,b) Low variation was present across concentrations (SD ≤ 0.46 up to 80 µL/mL) with slight increase at 160 µL/mL (SD = 2.19) attesting to reproducibility of assay. (table 1)

(a) (b)



(c) (d)

FIGURE 3 : (a) well diffusion test to check anti microbial activity against Staphylococcus Aureus (b) against Candida Albicans (c) against E. Coli (d) against Streptocoocus Mutans

Antimicrobial activity. Diffusion testing revealed organism-selective inhibition. Against Streptococcus mutans, the amikacin control measured 21 mm; GRX zones were 18 mm at 50 µg and 16 mm at 100 µg (≈ 85.7% and 76.2% of the standard, respectively). (fig 3d) For Escherichia coli, chloramphenicol yielded 22 mm; GRX produced 19 mm (50 µg; ≈ 86.4% of standard) and 22 mm (100 µg; 100%, i.e., matched the standard). (fig 3c) For Staphylococcus aureus, amikacin measured 20 mm; GRX gave 21 mm at 50 µg (105% of standard) but 16 mm at 100 µg (80% of standard). (fig 3a) Candida albicans was highly susceptible to fluconazole (31 mm) while GRX showed 0 mm at both doses, indicating no antifungal activity at tested loads.(fig 3d) Dose-directionality differed by species: E. coli increased 19→22 mm from 50→100 µg, whereas S. mutans and S. aureus decreased (18→16 mm and 21→16 mm), and C. albicans remained inactive. The reduced zones for S. mutans and S. aureus at 100 µg likely reflect diffusibility/solubility or matrix interactions that limit radial spread in agar despite higher nominal loading—an effect recognized for polyphenol-rich botanicals in diffusion assays. [13–15]

4. Discussion

This study demonstrates two properties of GRX that are relevant to oral-ulcer care. First, the anti-inflammatory effect rose steadily with concentration and approached the salicylate reference at 160 µL/mL in the albumin-denaturation model. While this assay is a screening proxy, its positive signal is congruent with substantial evidence that ginsenosides suppress NF-κB/MAPK signaling and downstream mediators (COX-2, iNOS, TNF-α, IL-1β) and can enhance keratinocyte migration and mucosal repair [4–7]. In oral contexts, Rg1-containing hydrogels have accelerated mucosal wound healing,

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lending biological plausibility to symptomatic relief and faster re-epithelialization with ginseng-based topicals [6].

Second, the antibacterial profile was species-selective—strongest for E. coli (parity with chloramphenicol at 100 µg), moderate for S. mutans and S. aureus (particularly at 50 µg), and absent against C. albicans. These findings align with literature reporting antibacterial effects of red-ginseng fractions and Compound K against oral pathogens, with variable antifungal activity unless specific saponin fractions are enriched or combined with azoles [8,9]. The bell-shaped response for S. mutans and S. aureus (smaller zones at the higher load) is consistent with diffusion-limited behavior of complex plant extracts in agar; follow-up MIC/MBC in broth systems and time-kill/biofilm assays are warranted to clarify intrinsic potency independent of diffusibility [13–17].

Clinical implications. For painful RAS lesions where inflammation predominates and bacterial colonization may exacerbate pain, a mucoadhesive GRX gel or film could offer steroid-sparing symptomatic benefit and modest antibacterial coverage. Since there is no antifungal activity here, products to be used in patients at risk for candidiasis should be combined with antifungal therapy where needed (e.g., fluconazole). Furthermore, as oral ulcers are multifactorially caused, GRX would be an addition to existing treatment, not a replacement.

Albumin denaturation is a convenient screen but not an assay with defined pathways; cytokine panels and COX-2/iNOS protein measurements must confirm anti-inflammatory mechanisms. Diffusion assays confuse potency with diffusibility. Hence broth microdilution MIC/MBC, biofilm inhibition against S. mutans, and synergy tests against antibiotics must conform to CLSI guidelines. Standardization to an extract fingerprint of ginsenosides (e.g., Rg1, Rb1, CK content) will improve reproducibility.In-vivo tolerability and mucosal-healing models must be performed prior to clinical study.

5. Conclusion

Ginseng root extract demonstrated a dose-dependent anti-inflammatory effect approaching the salicylate reference at higher concentrations and selective antibacterial activity—particularly against E. coli—with no antifungal effect against Candida albicans under the tested conditions. These findings support GRX as a promising adjunct for oral-ulcer management, ideally within a mucoadhesive formulation and, where needed, combined with antifungal coverage. Standardization and mechanistic/biofilm studies are the next steps toward translation.

Conflict of Interest : NIL

Funding : NIL

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