

Green Tea Kombucha Protects Against Murine Colitis by Averting Oxidative Colon Damage

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Introduction: Ulcerative colitis (UC) is a chronic inflammatory bowel disease (IBD) associated with intestinal inflammation, oxidative stress, intestinal barrier disruption, and increased risk of colorectal cancer. Although 5-aminosalicylic acid (5-ASA) preparations are commonly used in the management of IBD, their use may be limited by cost and adverse effects, including nausea and vomiting. These limitations highlight the need for safer and more affordable therapeutic alternatives. Kombucha, a fermented tea beverage, possesses considerable antioxidant properties and may therefore have therapeutic potential in oxidative stress-associated disorders. We hypothesised that green tea Komboucha (GTK) could attenuate colonic injury by mitigating oxidative stress.

Methods: The antioxidant capacity of GTK was initially evaluated using the 2,2-diphenyl-1-picrylhydrazyl (DPPH) free-radical scavenging assay. Colitis was subsequently induced in male Wistar rats by a single intrarectal administration of 500 μ L of 4% (v/v) acetic acid. Following induction, rats received daily oral administration of water, Pentasa mesalazine (30 mg/kg), or GTK at doses of 100 and 500 mg/kg for seven days. Animals were euthanized on day eight, the colons were excised, weighed, and their weight-to-length ratios determined. Colonic homogenates were also analyzed for the antioxidant enzymes superoxide dismutase (SOD) and catalase (CAT). Histopathological changes were evaluated following hematoxylin and eosin staining.

Results: GTK demonstrated significant antioxidant activity, as evidenced by its ability to scavenge DPPH free radicals. Intrarectal administration of acetic acid produced marked colonic injury, characterized histologically by crypt loss, oedema, and pronounced leukocyte infiltration into the lamina propria. Treatment with GTK attenuated these inflammatory and morphological alterations, with effects comparable to those observed following mesalazine treatment. The increased colon weight-to-length ratio associated with acetic acid-induced colitis was also reduced following GTK administration. In addition, GTK reversed the colitis-associated reduction in colonic CAT activity, suggesting an improvement in antioxidant defense.

Conclusion: These findings suggest that GTK ameliorates murine ulcerative colitis by enhancing antioxidant defense and reducing colon inflammation and tissue injury. GTK may therefore represent a promising natural therapeutic candidate for the management of UC.