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Effects of Bee Bread on Paracetamol-Induced Intestinal Injury in Rats

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ABSTRACT

Although paracetamol (PS) is widely used as a safe analgesic and antipyretic agent in both humans and animals at therapeutic doses, high-dose administration may cause damage not only to the liver and kidneys but also to the gastrointestinal system. The present study aimed to evaluate the protective effects of bee bread (BB) on intestinal integrity and inflammation in a paracetamol-induced intestinal injury model in Wistar rats using histopathological and immunohistochemical methods. The expression levels of the tight junction protein *zonula occludens-1* (*ZO-1*) and the inflammatory marker myeloperoxidase (MPO) were evaluated by immunohistochemistry. A total of 32 male rats (8-10 weeks old, 250-300 g) were randomly assigned to four groups (n = 8 per group), consisting of the control group, the BB group (750 mg/kg/day orally for 10 days), the PS group (1000 mg/kg administered as a single oral dose on day 10), and the PS plus BB group. The BB group exhibited normal intestinal histological architecture, preserved *ZO-1* expression, and low MPO immunoreactivity, comparable to those observed in the control group. In contrast, the PS group showed marked mucosal irregularities, villus degeneration, increased inflammatory cell infiltration, decreased *ZO-1* expression, and increased MPO immunoreactivity. Compared with the PS group, BB treatment alleviated paracetamol-induced intestinal injury, increased *ZO-1* expression, and reduced MPO immunoreactivity in the PS plus BB group. In conclusion, BB administration alone did not adversely affect intestinal morphology or the evaluated immunohistochemical markers and partially protected against paracetamol-induced intestinal injury by preserving intestinal barrier integrity and attenuating inflammatory responses.

Keywords: Bee bread, Histology, Paracetamol, *Zonula occludens-1*

INTRODUCTION

Paracetamol is a widely used antipyretic and analgesic agent and is considered safe at therapeutic doses (Jaeschke and Ramachandran, 2024). However, overdose may induce toxicity primarily affecting the liver, while the kidneys, gastrointestinal tract, and, in severe cases, the central nervous and cardiovascular systems may also be involved (Jaeschke and Ramachandran, 2024). During paracetamol metabolism, the reactive metabolite N-acetyl-p-benzoquinone imine (NAPQI) is generated, leading to glutathione depletion, mitochondrial oxidative stress, and subsequent cellular injury (Jaeschke and Ramachandran, 2024).

Emerging evidence indicates that paracetamol overdose may impair intestinal integrity by disrupting the epithelial barrier, increasing intestinal permeability, and promoting mucosal injury (Perananthan et al., 2024). The intestinal mucosa serves as a dynamic physical and immunological barrier that prevents the translocation of luminal microorganisms, toxins, and antigens into the systemic circulation (Horowitz et al., 2023). Intercellular tight junctions are essential for regulating paracellular permeability and preserving the integrity of the intestinal epithelial barrier (Arumugam et al., 2025). *Zonula occludens-1* (*ZO-1*) is a cytoplasmic scaffolding protein that anchors transmembrane tight junction proteins, including claudins and occludin, to the actin cytoskeleton, thereby ensuring tight junction assembly and epithelial barrier stability (Horowitz et al., 2023). In mice, experimental loss of intestinal epithelial *ZO-1* has been shown to modestly increase tight junction permeability and impair mucosal repair (Kuo et al., 2021).

Myeloperoxidase (MPO), a neutrophil-derived heme enzyme predominantly stored in neutrophil granules and released upon neutrophil activation, is widely recognized as a reliable biomarker of tissue inflammation and neutrophil infiltration (Cartwright et al., 2024; Clough et al., 2024). Furthermore, oxidative stress can increase tight junction permeability and alter the localization of tight junction proteins in intestinal epithelial cells, thereby impairing epithelial barrier function (Hasegawa et al., 2021).

Bee bread (BB) is a naturally fermented bee product containing abundant flavonols (quercetin, kaempferol, and isorhamnetin) together with phenolic acids including caffeic, ferulic, p-coumaric, gallic, and protocatechuic acids, all of which are recognized for their antioxidant and anti-inflammatory properties (Bakour et al., 2022). Simulated gastrointestinal digestion studies demonstrated that BB exhibits higher intestinal bioaccessibility of phenolic compounds than bee pollen, suggesting improved delivery of these bioactive molecules to the intestinal epithelium (Aylanc et al., 2021).

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Although direct in vivo evidence regarding the protective effects of BB against paracetamol-induced intestinal injury remains limited, its high intestinal bioaccessibility together with its well-documented antioxidant and anti-inflammatory properties provides a strong rationale for investigating its therapeutic potential (Aylanc et al., 2021; Bakour et al., 2022). Therefore, the present study aimed to investigate the potential protective effects of bee bread against paracetamol-induced duodenal injury in rats by evaluating histopathological changes and the immunohistochemical expression of *ZO-1* and MPO.

MATERIALS AND METHODS

Ethical approval

The present study was approved by the Local Animal Ethics Committee of Siirt University, Siirt, Türkiye (Approval No. 2026.03.23). All procedures involving tissue collection and histopathological and immunohistochemical evaluations were conducted in accordance with the approval granted by the committee and the institutional guidelines for the care and use of laboratory animals. No additional experimental animals were used, in accordance with the principles of the 3Rs (Replacement, Reduction, and Refinement).

Study design and tissue collection

The present study was a randomized controlled experiment using animals. A total of 32 male Wistar albino rats were randomly assigned to four experimental groups (8 per group), including a control, a BB group, paracetamol (PS group), and paracetamol with bee bread (PS + BB group). The control group received 0.5 mL sterile saline (0.9% NaCl) by oral gavage once daily for 10 consecutive days. The second group received 750 mg/kg/day of bee bread by oral gavage for 10 consecutive days (BB group). The BB, a naturally fermented bee-pollen product, was stored in its original sealed container at room temperature (approximately 22°C), protected from direct sunlight, according to the manufacturer's instructions until use. The selected dose was based on previous experimental studies demonstrating the antioxidant and anti-inflammatory efficacy of BB in rats (Suleiman et al., 2021; Bakour et al., 2022). In the third group, rats received sterile saline by oral gavage for 10 days, followed by a single oral administration of paracetamol (1000 mg/kg) on day 10 (PS group). This dose was selected because it is a well-established hepatotoxic dose in rats and has been widely used to induce acute oxidative tissue injury (Jaeschke and Ramachandran, 2024). In the fourth group, rats received BB (750 mg/kg/day) by oral gavage for 10 consecutive days; two hours after the final BB administration on day 10, a single oral dose of paracetamol (1000 mg/kg) was administered (PS + BB group). The 2-hour interval was chosen to ensure gastrointestinal absorption of BB prior to giving paracetamol, aligning with prophylactic pretreatment strategies used in experimental studies (Suleiman et al., 2021).

Animals were fasted for 12 hours before paracetamol administration and tissue collection, while water was provided *ad libitum*. Duodenal tissues were collected 24 hours after paracetamol administration for histopathological and immunohistochemical analyses. Throughout the experimental period, all animals had free access to tap water and a standard commercial pellet diet (Arden Feed Industry, Turkey).

Histopathological examination

Histopathological examination was performed using standard hematoxylin and eosin staining procedures as previously described by Bancroft and Gamble (2008). Paraffin sections were stained with Harris hematoxylin solution and alcoholic eosin Y solution (Ref. No. BS-009, Histomed, Türkiye) and examined using a light microscope (Olympus CX23, Japan). Histopathological assessment included evaluation of the overall architecture of the intestinal mucosa, epithelial integrity, villus morphology, inflammatory cell infiltration, and evidence of mucosal injury. Histopathological and immunohistochemical evaluations were performed by an expert investigator blinded to the experimental groups.

Immunohistochemical examination

Immunohistochemical examination was performed as previously described by Ramos-Vara and Miller (2014), with minor modifications. Paraffin-embedded tissue sections (4-6 μm thick) were placed on poly-L-lysine-coated glass slides. The sections were subsequently deparaffinized in xylene and rehydrated through graded ethanol solutions. Heat-induced antigen retrieval was carried out in ethylenediaminetetraacetic acid (EDTA) buffer. To eliminate endogenous peroxidase activity, the sections were incubated with 3% hydrogen peroxide, after which nonspecific protein binding was blocked using Ultra V Block. Sections were incubated overnight at +4°C with primary antibodies against MPO (STJA0010464, St John's Laboratory, UK; 1:500) and *ZO-1* (21773-1-AP, Proteintech, USA; 1:500). After washing with PBS, biotinylated secondary antibody and streptavidin–peroxidase were applied. Immunoreactivity was detected using 3,3′-diaminobenzidine (DAB) as the chromogenic substrate, after which the sections were counterstained with Harris

hematoxylin. The stained slides were subsequently evaluated and digitally photographed using a light microscope (Olympus CX23, Japan; Ramos-Vara and Miller, 2014). MPO immunoreactivity was used to assess intestinal inflammation, whereas *ZO-1* immunoreactivity was used to evaluate intestinal barrier integrity.

Statistical analysis

Since immunohistochemical scores represented ordinal data, results were expressed as median (minimum–maximum). Statistical analyses were performed using Jamovi software (Version 2.6.44). The normality of data distribution was assessed using the Shapiro–Wilk test. As the data were not normally distributed, differences among the four experimental groups were analyzed using the Kruskal–Wallis test. When a significant overall difference was detected, pairwise comparisons were performed using the Dwass–Steel–Critchlow–Fligner (DSCF) multiple comparison test. Data are presented as median (minimum–maximum), and a P-value less than 0.05 was considered statistically significant.

RESULTS

Histopathology

Histopathological examination demonstrated preserved duodenal architecture in the control and BB groups. Paracetamol administration resulted in marked intestinal mucosal injury, whereas BB treatment substantially attenuated these pathological changes. Although the PS plus BB group showed clear histological improvement compared with the PS group, the duodenal morphology was not completely restored to that observed in the control group (Figure 1).

Immunohistochemical results of myeloperoxidase

Low MPO immunoreactivity was observed in the control and BB groups, whereas the PS group exhibited markedly increased *MPO* expression. Post hoc DSCF analysis demonstrated significantly higher MPO immunoreactivity in the PS group than in the control ($p = 0.003$) and BB ($p = 0.003$) groups. Bee bread treatment significantly reduced MPO expression compared with the PS group (median: 2 [2-3] versus 4 [3-4]; $p = 0.018$). However, MPO immunoreactivity in the PS plus BB group remained significantly higher than that in the control ($p = 0.049$) and BB ($p = 0.049$) groups (Figure 2).

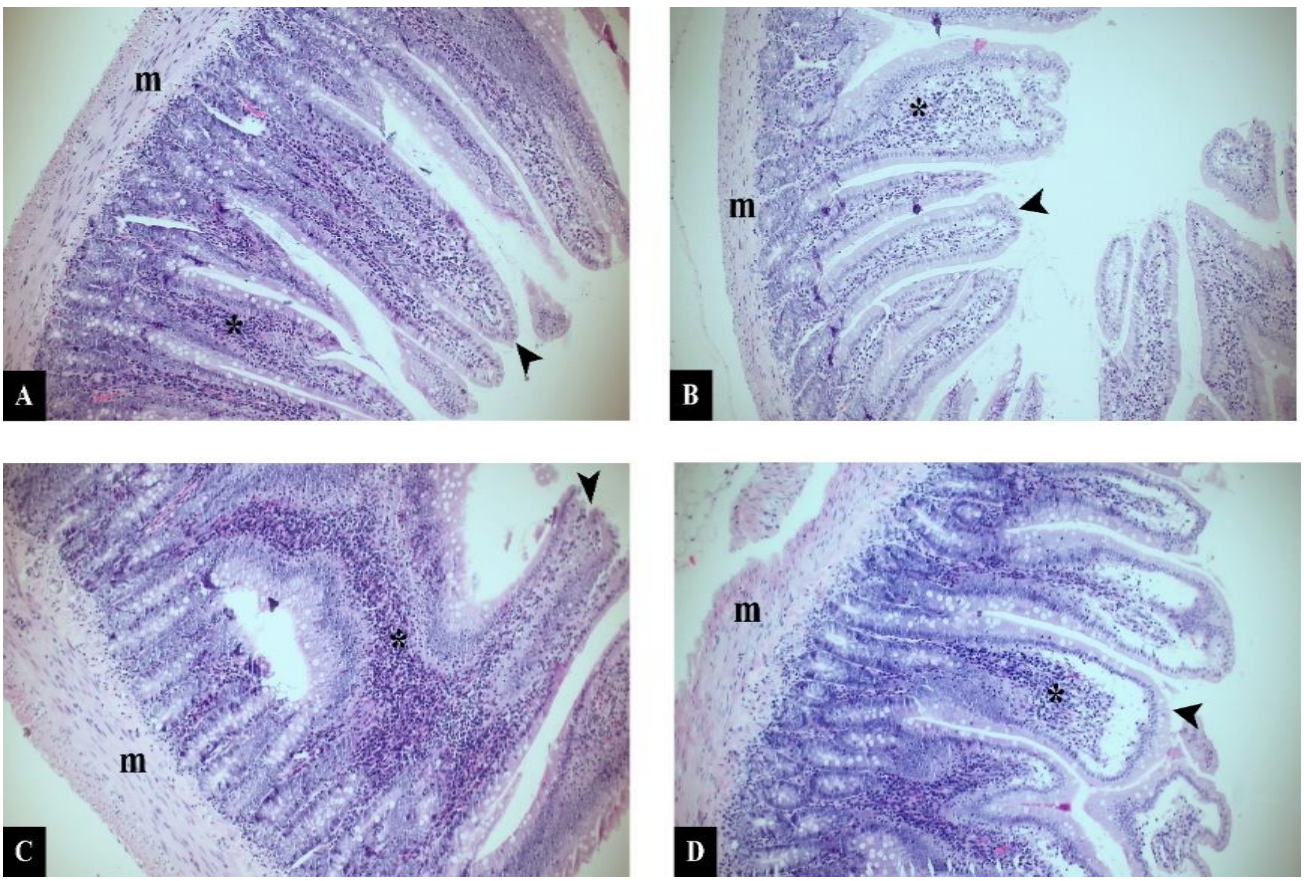

Figure 1. Representative hematoxylin and eosin (H&E)-stained sections of the duodenum in male rats. **A:** Control, **B:** Bee bread, **C:** Paracetamol, and **D:** Paracetamol plus bee bread. Black arrowheads: Epithelial alterations, *: Inflammatory cell infiltration, m: Muscularis layer, original magnification ×20. Scale bar: 100 µm.

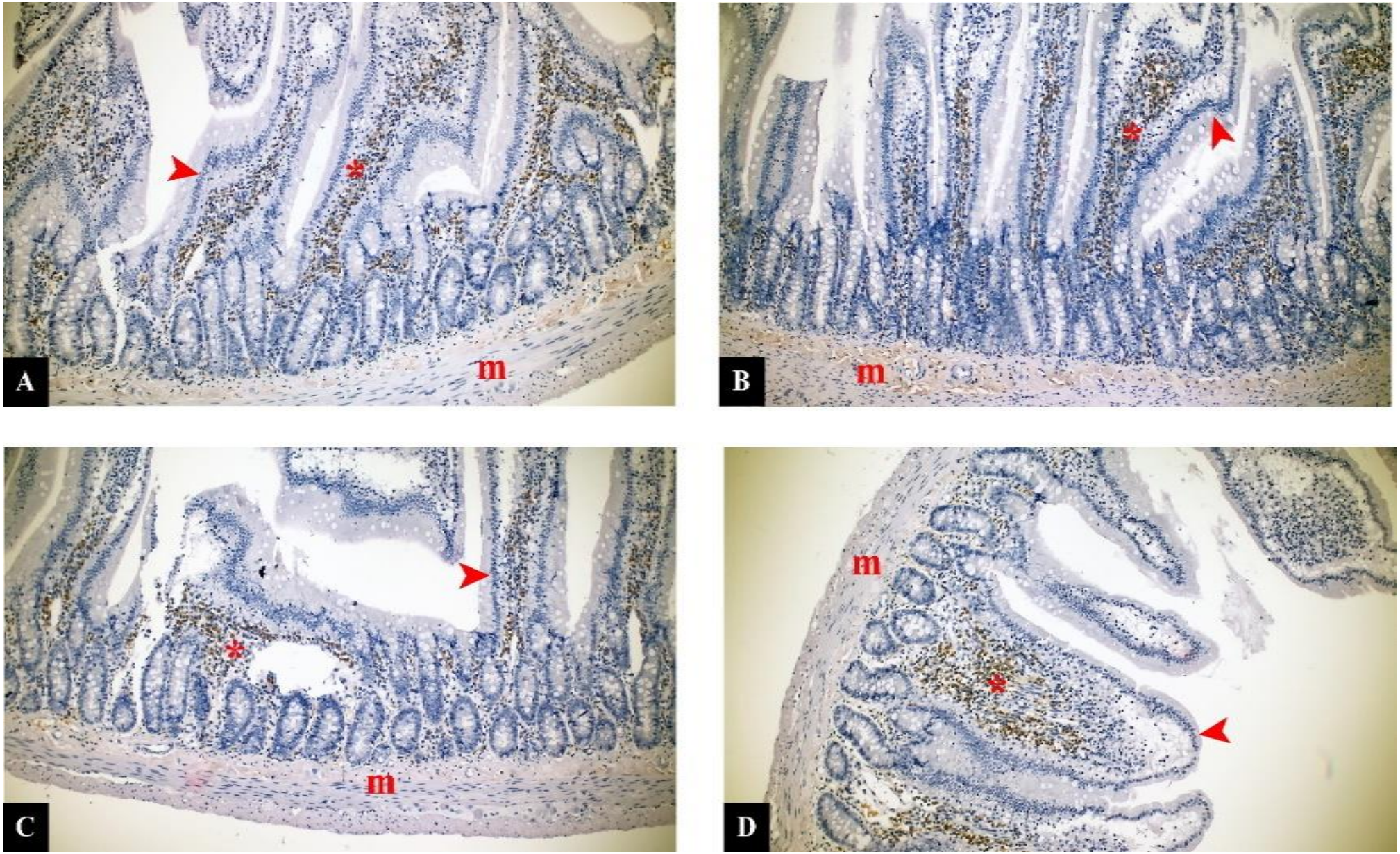

Figure 2. Immunohistochemical staining of myeloperoxidase in the duodenum of male rats. **A**: Control, **B**: Bee bread, **C**: Paracetamol, and **D**: Paracetamol plus bee bread. *: Inflammatory cell infiltration, m: Muscularis layer, DAB chromogen with Harris hematoxylin counterstaining, original magnification ×20. Scale bar: 100 μm.

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Immunohistochemical results of *Zonula occludens-1*

Strong and continuous membranous *ZO-1* immunoreactivity was observed along the villus epithelium in the control and BB groups. In contrast, the PS group exhibited weak and discontinuous epithelial staining. Overall differences among the experimental groups were statistically significant ($p = 0.002$). Post hoc DSCF analysis demonstrated significantly lower *ZO-1* immunoreactivity in the PS group than in the control ($p = 0.012$) and BB ($p = 0.012$) groups. Bee bread treatment significantly increased *ZO-1* immunoreactivity in the PS plus BB group compared with the PS group. No significant differences were detected between the PS plus BB group and either the control or BB groups (both $p = 0.941$; Figure 3).

Collectively, the findings of the present study indicated that BB attenuates paracetamol-induced intestinal injury and inflammation while partially restoring intestinal barrier integrity through reduced MPO expression and improved *ZO-1* immunoreactivity.

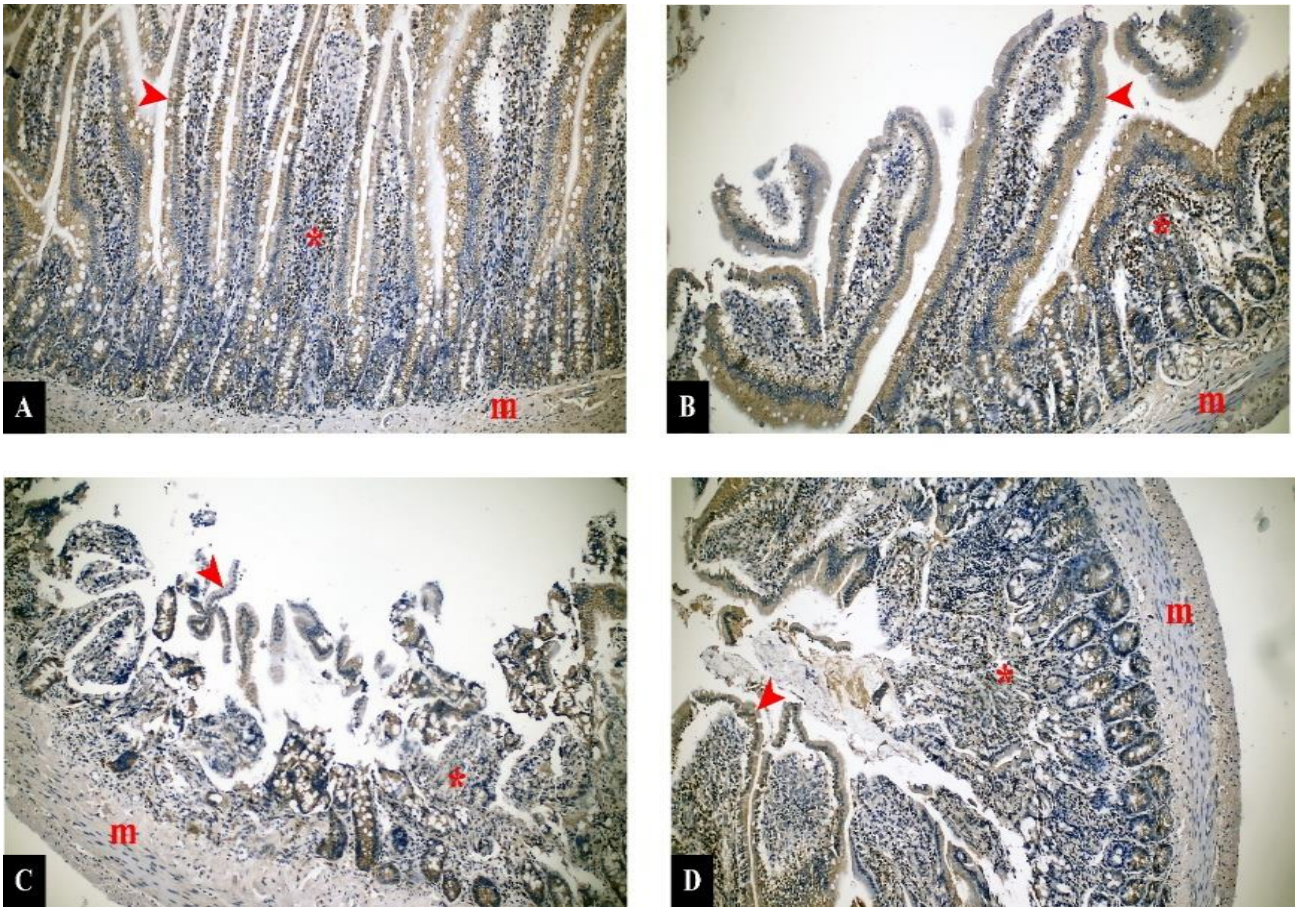

Figure 3. Immunohistochemical staining of *zonula occludens-1* (*ZO-1*) in the duodenum of male rats. **A**: Control, **B**: Bee bread, **C**: Paracetamol, **D**: Paracetamol plus bee bread. Arrowheads indicate *ZO-1*-positive epithelial cells; m: Muscularis layer. *: Inflammatory cell infiltration, DAB chromogen with Harris hematoxylin counterstaining. Original magnification ×20, Scale bar: 100 µm.

Table 1. Semi-quantitative myeloperoxidase and *zonula occludens-1* immunoreactivity scores in the duodenum of 8–10-week-old male rats

Parameter	C	BB	PS	PS+BB	P-value
MPO	1.5 (1–2)^{a}	2 (1–2)^{a}	4 (3–4)^{c}	2 (2–3)^{b}	<0.001
ZO-1	3 (2–4)^{a}	3 (2–4)^{a}	1 (1–2)^{b}	2.5 (2–3)^{a}	0.002

C: Untreated control rats, BB: Bee bread-treated rats, PS: Paracetamol-treated rats, PS+BB: Paracetamol plus bee bread-treated rats, MPO: Myeloperoxidase, *ZO-1*: *Zonula occludens-1*. ^{abc}Different superscript letters in the same row were significantly different at $p < 0.05$.

DISCUSSION

In the present study, paracetamol administration produced marked duodenal injury characterized by villus degeneration, inflammatory cell infiltration, increased myeloperoxidase immunoreactivity, and reduced *ZO-1* expression. These findings are consistent with those of Alabbas et al. (2023), who found that acetaminophen induced intestinal injury, increased intestinal permeability, and reduced *ZO-1* expression in the colonic epithelium of mice. Furthermore, Alabbas et al. (2023) demonstrated bacterial translocation to the liver and spleen, emphasizing the role of intestinal barrier

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dysfunction in acetaminophen-related systemic toxicity. Unlike the current results indicating the duodenum as a direct target of paracetamol-induced injury, Alabbas et al. (2023) mainly concentrated on the colon.

The present findings were further supported by recent clinical evidence demonstrating intestinal epithelial injury following acute paracetamol overdose. Perananthan et al. (2024) demonstrated that patients with acute paracetamol overdose exhibited increased circulating biomarkers of enterocyte injury, indicating that intestinal epithelial damage also occurs in humans. Therefore, the decreased *ZO-1* expression observed in the present study provides histopathological and immunohistochemical evidence that complements these clinical observations.

The marked increase in MPO immunoreactivity in the PS group indicates enhanced neutrophil-associated inflammation within the duodenal mucosa. The present findings extend the observations of Alabbas et al. (2023) by demonstrating that acetaminophen-induced intestinal injury is not limited to the colon but also involves the proximal small intestine. Importantly, BB administration significantly reduced MPO immunoreactivity while restoring *ZO-1* expression toward control levels, suggesting attenuation of intestinal inflammation together with preservation of epithelial barrier integrity.

Paracetamol-induced intestinal injury shares several pathogenic mechanisms with paracetamol-induced hepatotoxicity, including oxidative stress, inflammatory responses, mitochondrial dysfunction, and disruption of the intestinal epithelial barrier (Alabbas et al., 2023; Jaeschke and Ramachandran, 2024; Perananthan et al., 2024). Following overdose, increased metabolism of paracetamol by cytochrome P450 enzymes leads to the excessive formation of the highly reactive metabolite N-acetyl-p-benzoquinone imine (NAPQI; McGill and Jaeschke, 2013; Jaeschke and Ramachandran, 2024). Although hepatocytes are the primary site of NAPQI generation, intestinal epithelial cells also express cytochrome P450 enzymes capable of producing this reactive metabolite, thereby contributing to local intestinal injury (Alabbas et al., 2023; Perananthan et al., 2024; Jaeschke and Ramachandran, 2024). Excessive NAPQI formation depletes intracellular glutathione, promotes mitochondrial oxidative stress, induces enterocyte apoptosis, and disrupts epithelial tight junctions, ultimately increasing intestinal permeability (Horowitz et al., 2023; Jaeschke and Ramachandran, 2024; Arumugam et al., 2025). Impairment of the intestinal barrier facilitates bacterial translocation and amplifies local inflammatory responses through activation of the gut–liver axis (McGill and Jaeschke, 2013; Alabbas et al., 2023).

Oxidative stress and inflammation act synergistically to impair intestinal barrier function and promote mucosal injury (Bhattacharyya et al., 2014). Bee bread is a natural fermented bee product rich in polyphenols, flavonoids, phenolic acids, amino acids, vitamins, minerals, and antioxidant enzymes, including quercetin, kaempferol, caffeic acid, gallic acid, and *p*-coumaric acid (Bakour et al., 2022). Bioactive compounds in BB enhance endogenous antioxidant defense through activation of the Nrf2/HO-1 pathway while suppressing NF-κB and MAPK-mediated inflammatory signaling, thereby reducing oxidative stress, limiting pro-inflammatory cytokine production, and preserving epithelial tight-junction proteins (Bakour et al., 2022). The observed effects of BB were consistent with the proposed mechanisms, as evidenced by significantly reduced MPO immunoreactivity and restored *ZO-1* expression in the present study. Similar protective effects on intestinal epithelial barrier integrity and inflammatory responses have been reported for fermented bee pollen, which enhances tight-junction protein expression and suppresses NF-κB-mediated inflammatory signaling (Zhang et al., 2024).

The concordance between the histopathological and immunohistochemical findings supports the close relationship between preservation of intestinal morphology and maintenance of epithelial barrier integrity. Alabbas et al. (2023) demonstrated that acetaminophen-induced intestinal injury was accompanied by disruption of tight-junction proteins, increased intestinal permeability, and inflammatory alterations. Likewise, Zhang et al. (2024) reported that fermented bee pollen enhanced epithelial barrier integrity by increasing tight-junction protein expression through inhibition of the NF-κB-mediated MLCK-MLC signaling pathway. The present findings support the hypothesis that BB protects against paracetamol-induced intestinal injury through coordinated preservation of epithelial barrier integrity and attenuation of inflammatory responses.

CONCLUSION

Paracetamol administration induced marked duodenal injury characterized by mucosal alterations, increased MPO immunoreactivity, and decreased *ZO-1* expression. Bee bread administration attenuated these changes by reducing MPO immunoreactivity and improving *ZO-1* expression, together with partial preservation of intestinal morphology. The findings of the present study indicated that BB exerts a partial protective effect against paracetamol-induced intestinal injury by attenuating inflammation and supporting epithelial barrier integrity. The main limitation of the current study was the lack of biochemical and molecular analyses of oxidative stress and inflammatory pathways. Future studies

incorporating biochemical, molecular, and intestinal permeability analyses are recommended to further clarify the mechanisms underlying the protective effects of BB.

DECLARATIONS

Author contributions

Günsel Kirman conceptualized and designed the study, developed the methodology, conducted the investigation, curated the data, performed the formal statistical analyses, prepared the figures and visualizations, wrote the original draft of the manuscript, and reviewed and edited the final version of the manuscript. The author has read and approved the final edition of the manuscript before publication in the present journal.

Availability of the data and materials

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Competing interests

The author declares that there are no competing interests.

Ethical considerations

Ethical issues, including plagiarism, consent to publish, scientific misconduct, data fabrication and/or falsification, duplicate publication and/or submission, and redundancy, have been carefully considered and checked by the author. The author used ChatGPT (OpenAI) solely to improve the language, grammar, and readability of the manuscript. The AI tool was not used for data collection, data analysis, interpretation of results, or generation of scientific conclusions. The author reviewed, edited, and approved all content and accepts full responsibility for the accuracy and integrity of the manuscript before submission, revision, and publication.

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