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죽여 추출물의 항염증 효과 및 EtOH/HCl 유도 위 점막 손상에 대한 보호 효과

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Anti-inflammatory Effects and Gastric Mucosal Protection of Phyllostachyos Caulis in Taeniam Extract against EtOH/HCl-Induced Gastric Mucosal Injury

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ABSTRACT

Background: *Phyllostachyos Caulis in Taeniam* (PCT), commonly known as bamboo shavings, is a traditional medicinal herb widely used in Korea and China for its heat-clearing, phlegm-resolving, and anti-inflammatory properties. This study investigated the antioxidant, anti-inflammatory, and gastroprotective effects of the hot-water extract of PCT (PCTD).
Methods and Results: PCTD exhibited radical-scavenging activity in DPPH and ABTS assays and anti-inflammatory effects in lipopolysaccharide (LPS)-stimulated RAW264.7 cells. Its gastroprotective effects were evaluated in a mouse model of acute gastric mucosal injury induced by EtOH/HCl. In LPS-stimulated RAW264.7 cells, PCTD reduced nitric oxide and prostaglandin E_2 levels without compromising cell viability. At a concentration of 200 μg/mL, PCTD also modulated the expression of inflammation-related cytokines and proteins. Furthermore, oral administration of PCTD significantly reduced gross mucosal injury in the EtOH/HCl-induced model. Representative histological sections revealed differences in mucosal morphology and PAS-positive and TUNEL-positive staining following PCTD treatment.
Conclusions: These findings indicate that PCTD exhibits antioxidant and anti-inflammatory activities *in vitro* and attenuates EtOH/HCl-induced acute gastric mucosal injury in mice. Its ability to suppress pro-inflammatory mediators in macrophages and protect against gastric mucosal injury highlights its therapeutic potential as a natural anti-inflammatory and gastroprotective agent.

Key Words: Anti-inflammatory, Antioxidant, Gastritis, Phyllostachyos Caulis in Taeniam, Plant Resource, Traditional Medicine

INTRODUCTION

Phyllostachyos Caulis in Taeniam (PCT) is derived from the middle layer of the stems of *Phyllostachys nigra* var. *henonis* or *Phyllostachys bambusoides*. PCT is a commonly used medicinal material in traditional Chinese and Korean medicine (Kim *et al*., 2013; An *et al*., 2025). Since ancient times, PCT has been utilized for clearing heat, resolving phlegm, relieving adverse gastric symptoms, stopping vomiting, and calming the liver and nerves (Kim *et al*., 2013; An *et al*., 2025). Modern pharmacological studies indicate that PCT contains multiple active compounds, such as chlorogenic acid, ferulic acid, p-coumaric acid, and triterpenoids, which contribute to its notable antioxidant and anti-inflammatory activities (Lim *et al*., 2017; An *et al*., 2025). Jin *et al*. (2012) indicated that the ethyl acetate fraction of bamboo significantly suppressed levels of inflammatory mediators such as TNF-α, IL-1β, and IL-6 in RAW264.7 macrophages stimulated by *Porphyromonas gingivalis* LPS. Furthermore, the fraction activated the Nrf2/p38 MAPK pathway and induced heme oxygenase-1 (HO-1) expression, suggesting a role in the regulation of oxidative stress and inflammatory responses (Jin *et al*., 2012). Its triterpenoid-

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enriched extract (EBS) has been reported to possess antitumor and anti-lipid peroxidation properties (Lu *et al*., 2010). These findings suggest that bamboo shavings may have potential as a natural source of antioxidant and anti-inflammatory agents.

Gastritis is a common gastrointestinal disorder characterized by inflammation and structural damage to the gastric mucosa and may occur in acute or chronic forms (Kim *et al*., 2023). Major causes include *Helicobacter pylori*, chronic heavy drinking, and long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs) (Yang *et al*., 2024). Alcohol consumption not only stimulates and damages gastric epithelial cells, but excessive intake also weakens the mucosal barrier, making it more vulnerable to irritation (Oh *et al*., 2005; Kim *et al*., 2021). The gastric mucosa is protected by several defense mechanisms, including the mucus-bicarbonate barrier, prostaglandin synthesis, mucosal blood flow, antioxidant defense, and epithelial repair (Hagen, 2021). Impairment of these protective mechanisms can further aggravate mucosal erosion and inflammatory damage. Natural plant extracts have been found to maintain gastric mucosal integrity by modulating oxidative stress and inflammatory pathways (Lee *et al*., 2022; Chen *et al*., 2025). This study aimed to confirm the anti-inflammatory activity of PCTD at the cellular level and investigate its inhibitory effects on inflammation and its protective effects on the gastric mucosa using an EtOH/HCl-induced acute gastric mucosal injury model.

MATERIALS AND METHODS

1. Plant material and extract

Phyllostachyos Caulis in Taeniam (PCT) used in this experiment was purchased from Kwangmyungdang Pharmaceutical Co. (Ulsan, South Korea), a product certified under the Good Manufacturing Practice (GMP) standards of the Ministry of Food and Drug Safety, South Korea. Documentation of the plant material includes a voucher specimen (No. 2-20-0025) deposited in the Korean Herbarium of Standard Resources at the Korea Institute of Oriental Medicine (KIOM) (Supplementary Fig. S1). The PCT was subjected to reflux extraction in distilled water for 3 hours. The solution was concentrated under reduced pressure, and freeze-dried. The extract was stored at –70℃.

2. Antioxidant activity

2.1. DPPH radical scavenging activity

To evaluate the antioxidant activity of PCTD, the DPPH (2,2-diphenyl-1-picrylhydrazyl) radical scavenging assay was performed. PCTD was prepared at working concentrations of 25, 50, and 100 ㎍/mL. Gallic acid was used as a positive control at the same concentrations (25, 50, and 100 ㎍/mL). A volume of 100 μL of 0.2 mM DPPH solution was mixed with an equal volume of PCTD solution or gallic acid solution in a 96-well plate. The mixture was incubated in the dark for 30 minutes (Nam *et al*., 2021). Absorbance was measured at 517 ㎚ using a microplate spectrophotometer (SpectraMax i3x, Molecular Devices, San Jose, CA, USA).

2.2. ABTS radical scavenging activity

To evaluate the antioxidant capacity of PCTD, the ABTS [2,2'-azinobis-(3-ethylbenzothiazoline-6-sulfonic acid)] radical scavenging assay was employed (Bibi Sadeer *et al*., 2020). A 7 mM ABTS solution was mixed with an equal volume of 2.45 mM potassium persulfate solution and reacted in the dark. The mixture's absorbance at 734 ㎚ was quantified using a microplate reader, proceeding with the assay only when readings reached 0.70 ± 0.02. Subsequently, 180 μL of $ABTS^{+}$ working solution was mixed with PCTD at various concentrations (25, 50, 100 ㎍/mL) and reacted for 10 min. Gallic acid was used as a positive control at the same concentrations (25, 50, and 100 ㎍/mL). After the reaction, absorbance was measured at 734 ㎚ using a microplate spectrophotometer (SpectraMax i3x, Molecular Devices, San Jose, CA, USA).

Radical scavenging activity (%) = [1 − (mean absorbance of sample / mean absorbance of DW control)] × 100

3. Anti-inflammatory activity

3.1. Cell culture

RAW264.7 macrophages, obtained from the American Type Culture Collection (ATCC, Manassas, VA, USA), were maintained in Dulbecco's Modified Eagle Medium (DMEM) containing 10% fetal bovine serum (FBS) along with 1% penicillin-streptomycin (P/S). Cells were cultured at 37℃ with 5% CO_2 and used during the exponential growth phase.

3.2. Cell viability and nitric oxide (NO) production

RAW 264.7 cells were seeded in 96-well plates at a density of 2×10^5 cells/well and incubated for 24 h. The cells were pretreated with PCTD (50, 100, or 200 ㎍/mL) for 1 h and then stimulated with 1 ㎍/mL lipopolysaccharide (LPS) for 24 h. Cell viability was assessed using the MTS assay by

measuring absorbance at 490 ㎚. For NO analysis, nitrite levels in the culture supernatant were measured using Griess reagent at 540 ㎚ and calculated from a nitrite standard curve.

3.3. Inhibition of inflammatory cytokine and PGE2 production

RAW264.7 cells were pretreated with PCTD (100 or 200 ㎍/mL) and then stimulated with LPS (1 ㎍/mL). After 24 h, supernatants were collected for analysis. To assess the effects of LPS and PCTD on PGE_2 production, an ELISA kit (R&D Systems, Minneapolis, MN, USA) was used. Absorbance was measured at 450 ㎚ using a SpectraMax i3x microplate spectrophotometer (Molecular Devices, San Jose, CA, USA), and PGE_2 production was calculated based on the standard curve.

3.4. IL-1β, IL-6, G-CSF, and GM-CSF ELISA assay

The concentrations of IL-1β, IL-6, G-CSF, and GM-CSF in the cell culture supernatants were quantified using specific commercial ELISA kits (R&D Systems, Minneapolis, MN, USA) following the manufacturer's protocols. Cytokine concentrations are expressed as pg/mL.

3.5. Western blot analysis

RAW264.7 cells were pretreated with PCTD (100 or 200 ㎍/mL) for 1 h and then stimulated with LPS (1 ㎍/mL) for 18 h. Total cellular proteins were extracted, and the expression levels of COX-2, COX-1, IL-1β, and IL-10 were analyzed by western blotting. β-actin was used as a loading control. Protein band intensities were quantified using Image Lab software (Bio-Rad Laboratories, Hercules, CA, USA) and normalized to β-actin. The experiment was independently performed three times.

4. Protective effect against gastric mucosal injury

4.1. Experimental animals

Six-week-old C57BL/6 male mice (20 ± 2 g) were purchased from Doolyul Bio (Seocho-gu, Seoul, Korea). After one week of acclimatization under standard laboratory conditions (temperature 22 ± 2℃, humidity 50 ± 5%, 12-hour light/dark cycle) with free access to food and water, they were used for experiments. All experimental equipment underwent sterilization. All animal experiments complied with global welfare guidelines and received formal approval from the KIOM Animal experiment ethics committee under protocol number (Approval No: 22-080).

4.2. Induction of gastric mucosal injury and drug administration

To evaluate the gastroprotective effect of PCTD, gastric mucosal injury was induced by administration of 70% ethanol containing 150 mM HCl. To analyze changes in gastric mucosal injury severity, 24 mice were randomly divided into four groups (n = 6 per group): Normal control group (NC, receiving saline), EtOH/HCl group (receiving EtOH/HCl), positive control group (R25, receiving EtOH/HCl + ranitidine 25 ㎎/㎏), and PCTD-treated group (PCTD50, receiving EtOH/HCl + PCTD 50 ㎎/㎏). For one week before induction, the NC and EtOH/HCl groups received saline, the R25 group received ranitidine (25 ㎎/㎏), and the PCTD50 group received PCTD (50 ㎎/㎏) orally. The 50 ㎎/㎏ dose used in the present study falls within the range previously evaluated for Bambusae Caulis in Taeniam extracts *in vivo* (Ra *et al.*, 2010; Lim *et al.*, 2017). This pretreatment protocol was designed to evaluate the protective effect of PCTD against acute gastric mucosal injury rather than the post-injury recovery process. On the day of the experiment, after a 4-hour fast, the respective pretreatments (saline, ranitidine, PCTD) were administered. After two hours, EtOH/HCl solution was orally administered to establish gastric lesions, and the animals were euthanized two hours later.

4.3. Assessment of gastric mucosal injury

To analyze changes in gastric mucosal injury, the stomach was removed, incised, rinsed with saline, pinned in place, and photographed. ImageJ software (National Institutes of Health, Bethesda, MD, USA) was employed to measure both total and injured area (㎟) from the gastric mucosal images. The injury rate was calculated using the following formula:

$$\text{Lesion area (\%)} = (\text{Damaged area} / \text{Total mucosal area}) \times 100$$

4.4. Histopathological analysis

To observe the extent of gastric mucosal damage and histopathological changes induced by PCTD administration, gastric tissue samples were fixed, paraffin-embedded, and sectioned. Hematoxylin and eosin (H&E) staining was performed to evaluate gastric mucosal structure, hemorrhage, and inflammatory cell infiltration. To evaluate the distribution and function of mucus-secreting cells within the gastric mucosa, periodic acid-Schiff (PAS) staining was employed. To assess DNA fragmentation in gastric tissue, a terminal deoxynucleotidyl transferase-mediated dUTP nick-end labeling (TUNEL) assay

was performed following the manufacturer's protocol (Roche, Mannheim, Germany). TUNEL-positive staining was observed and recorded using an optical microscope.

5. Statistical analysis

All data are presented as mean ± SD. Group differences were assessed using one-way ANOVA with Tukey's multiple comparisons test in GraphPad Prism (GraphPad Software, San Diego, CA, USA). For the *in vitro* experiments, comparisons of interest were made between the unstimulated control (NC) and LPS-stimulated groups and between the LPS-stimulated and PCTD-treated groups. For the *in vivo* experiment, comparisons of interest were made between the normal control group (NC) and EtOH/HCl groups and between the EtOH/HCl group and ranitidine (R25) or PCTD-treated groups. Differences were considered statistically significant at $p < 0.05$.

RESULTS

1. Antioxidant effects

1.1. DPPH and ABTS radical scavenging activities

DPPH and ABTS radical scavenging activities of PCTD were evaluated using gallic acid as a positive control. As shown in Fig. 1, PCTD exhibited concentration-dependent radical scavenging activity in both assays. Gallic acid exhibited higher radical scavenging activity than PCTD in both assays. At a concentration of 100 μg/mL, PCTD reached approximately 69.1% in the DPPH and 46.5% in the ABTS assay. The IC_{50} values of PCTD were approximately 55.3 μg/mL for DPPH and 106.9 μg/mL for ABTS. These results indicate that PCTD possesses *in vitro* antioxidant activity.

2. Anti-inflammatory Effects

2.1. Cell viability

To assess potential cytotoxicity, cell viability was measured after treatment with different concentrations of PCTD. LPS stimulation increased the MTS signal compared with the untreated control. However, PCTD treatment at concentrations up to 200 μg/mL did not significantly reduce the MTS signal compared with the LPS-stimulated group (Fig. 2A).

2.2. Inhibition of nitric oxide (NO) production

Nitric oxide (NO) is a reactive species produced during cellular inflammatory responses and is widely recognized as a represen-

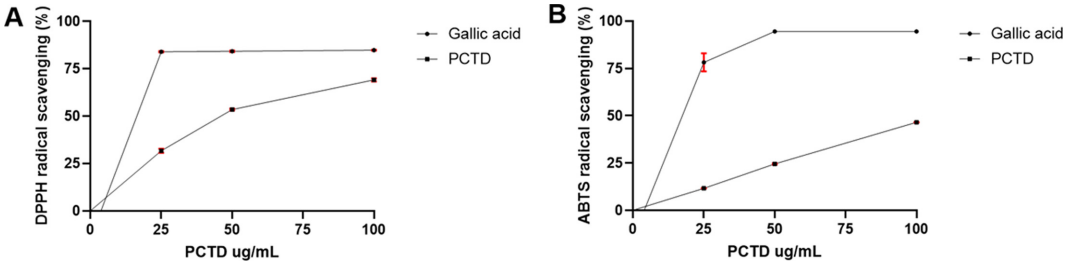


Fig. 1. DPPH (A) and ABTS (B) radical-scavenging activities of PCTD. Gallic acid was used as a positive control. Values are presented as mean ± SD (n = 3).

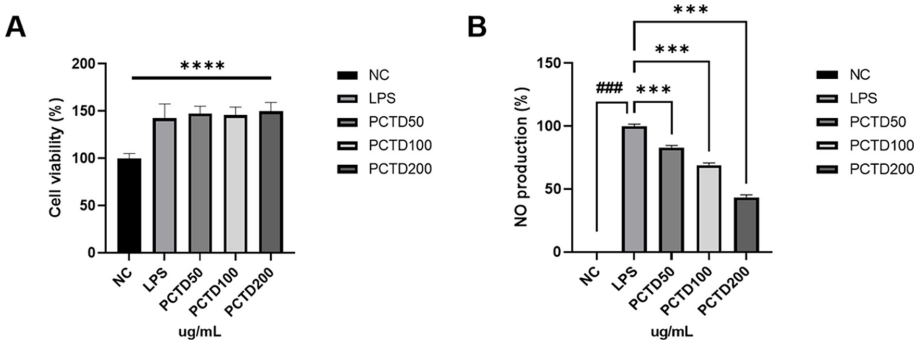


Fig. 2. Cell viability (A) and nitrite levels (B) in LPS-stimulated RAW264.7 cells treated with PCTD. Values represent the mean ± SD (cell viability, n = 6; nitrite, n = 3). ###$p < 0.001$ vs. unstimulated cells; ****$p < 0.0001$, ***$p < 0.001$ vs. LPS-stimulated cells.

tative indicator for evaluating anti-inflammatory activity. The LPS-treated group produced a large amount of nitrite compared with the control group. However, when extracts at concentrations of 50, 100, and 200 μg/mL were added under LPS-stimulated conditions, NO production exhibited a concentration-dependent inhibitory trend. Significant differences were observed between all treated groups and the LPS-treated group. Notably, at 200 μg/mL, PCTD reduced NO production by approximately 56.6% compared with the LPS-treated group (Fig. 2B).

2.3. Inhibition of prostaglandin E2 (PGE2) production

PGE_2 is an important inflammatory mediator associated with cyclooxygenase activity and inflammatory responses (Wallace, 2008; Halter, 2001). To evaluate the inhibitory effect of PCTD on LPS-induced macrophage inflammation, PGE_2 levels were measured after treatment with different concentrations. The study demonstrated that PGE_2 production in the LPS-treated group significantly increased compared to the control group. Compared with the LPS-treated group, PGE_2 production was reduced by approximately 31.2% and 36.6% following treatment with 100 and 200 μg/mL PCTD, respectively. These results indicate that PCTD reduced LPS-induced PGE_2 production (Fig. 3).

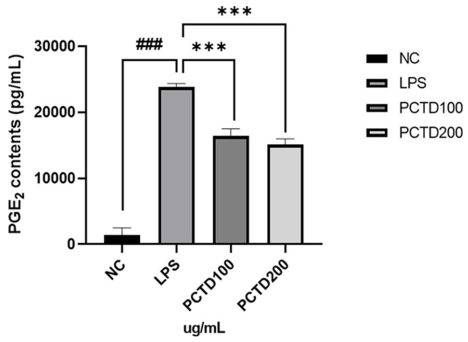


Fig. 3. Effects of PCTD on prostaglandin E_2 (PGE_2) levels in the LPS-stimulated RAW264.7 cells. Values are presented as mean ± SD (n = 3). ###$p < 0.001$ vs. unstimulated cells; ***$p < 0.001$ vs. LPS-stimulated cells.

2.4. Inhibition of IL-6, IL-1β, G-CSF, and GM-CSF secretion levels

To further validate the anti-inflammatory properties of PCTD, pro-inflammatory cytokine levels in culture supernatants were measured via ELISA. LPS stimulation markedly increased the secretion of IL-6, IL-1β, G-CSF, and GM-CSF compared with the untreated control group.

PCTD treatment showed a decreasing trend in the levels of these cytokines, with a significant reduction in GM-CSF at 200 μg/mL. Specifically, GM-CSF secretion was reduced by approximately 17.3% compared with the LPS-treated group. These results indicate that PCTD attenuated LPS-induced inflammatory mediator production in RAW264.7 cells (Fig. 4).

2.5. Effects of PCTD on inflammatory protein expression

LPS stimulation significantly increased COX-2 and IL-1β expression, whereas COX-1 and IL-10 expression were

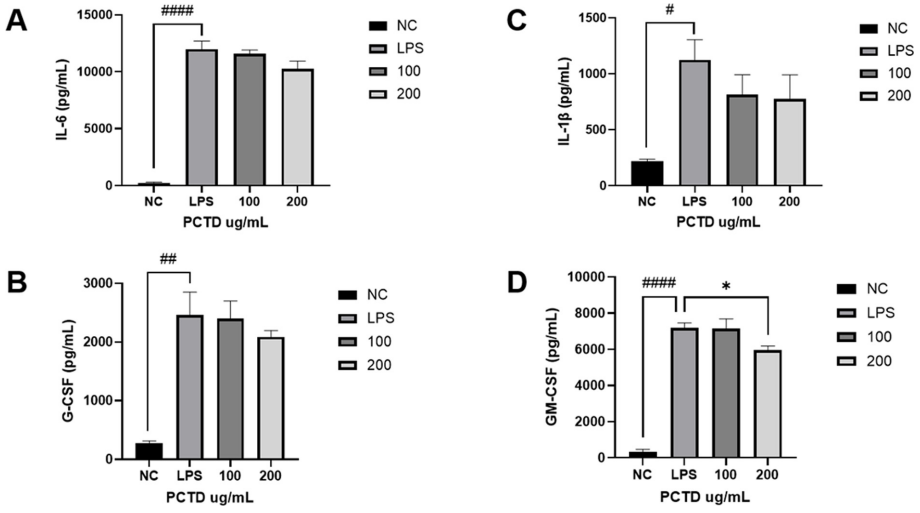


Fig. 4. Effects of PCTD on the production of IL-6 (A), G-CSF (B), IL-1β (C), and GM-CSF (D) in RAW264.7 cells. Data are expressed as mean ± SD from three independent experiments (n = 3). ####$p < 0.0001$, ##$p < 0.01$, #$p < 0.05$ vs. unstimulated cells; *$p < 0.05$ vs. LPS-stimulated cells.

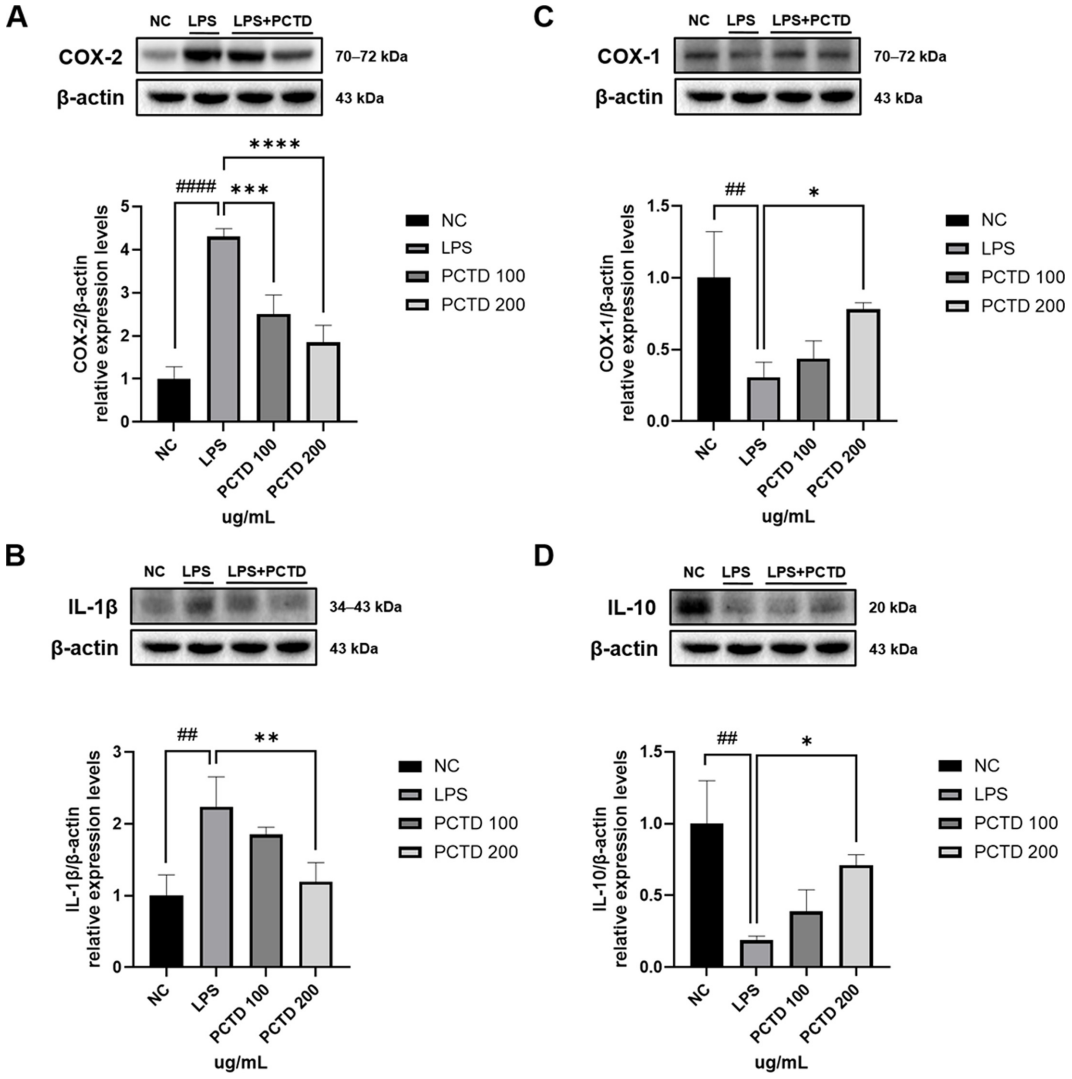


Fig. 5. Effects of PCTD on COX-2 (A), IL-1β (B), COX-1 (C), and IL-10 (D) protein expression in LPS-stimulated RAW264.7 cells. Representative Western blot images and densitometric analyses are shown. Protein expression was normalized to β-actin. Data are expressed as mean ± SD from three independent experiments (n = 3). ####$p < 0.0001$, ##$p < 0.01$ vs. unstimulated cells; ****$p < 0.0001$, ***$p < 0.001$, **$p < 0.01$, *$p < 0.05$ vs. LPS-stimulated cells.

significantly decreased compared with the normal control group. PCTD treatment significantly decreased COX-2 expression at 100 and 200 ㎍/mL, by approximately 41.7% and 57.1%, respectively, compared with the LPS-stimulated group, while IL-1β expression was significantly decreased at 200 ㎍/mL, by approximately 47.4%. In contrast, COX-1 and IL-10 expression were significantly increased by PCTD treatment at 200 ㎍/mL compared with the LPS-stimulated group, reaching approximately 2.54-fold and 3.75-fold of the LPS-treated levels, respectively (Fig. 5).

3. Gastric mucosal protective effects

3.1. Analysis of gastric mucosal injury

To evaluate the protective effect of PCTD against acute gastric mucosal injury, an EtOH/HCl-induced acute gastric mucosal injury model was employed in mice. As shown in Fig. 6, the EtOH/HCl group exhibited a significantly higher gastric damage ratio than the normal control (NC) group. Compared with the EtOH/HCl group, the treatment group administered 50 ㎎/㎏ PCTD exhibited significantly reduced mucosal damage, with lesion area decreasing by approximately 13.70% to 6.61%, corresponding to a 51.7% reduction. The

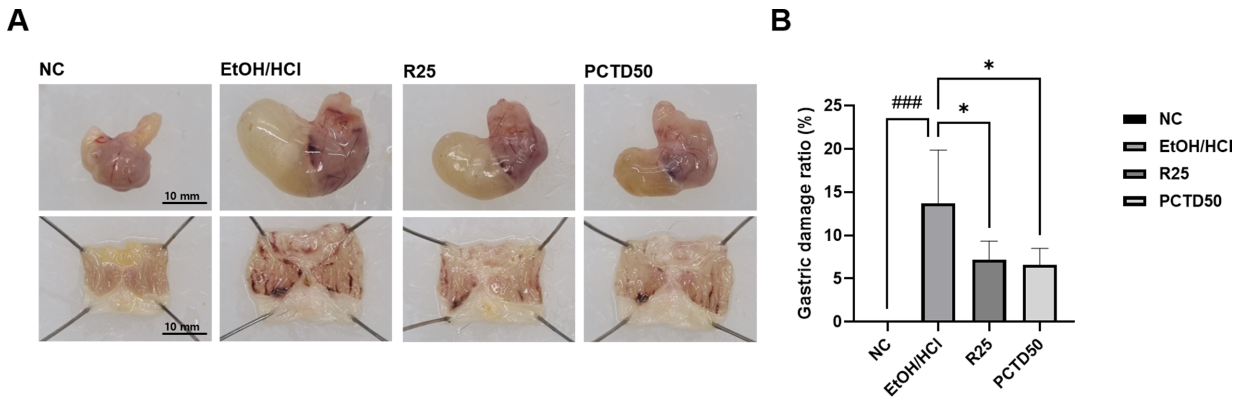


Fig. 6. Effects of PCTD on EtOH/HCl-induced gastric mucosal injury in mice. Gross appearance (A) and gastric mucosal injury ratio (B). NC–Normal control group; EtOH/HCl–EtOH/HCl group; R25–EtOH/HCl-treated group administered ranitidine (25 ㎎/㎏); and PCTD50–EtOH/HCl-treated group administered PCTD (50 ㎎/㎏). ###$p < 0.001$ vs. NC group; *$p < 0.05$ vs. EtOH/HCl group.

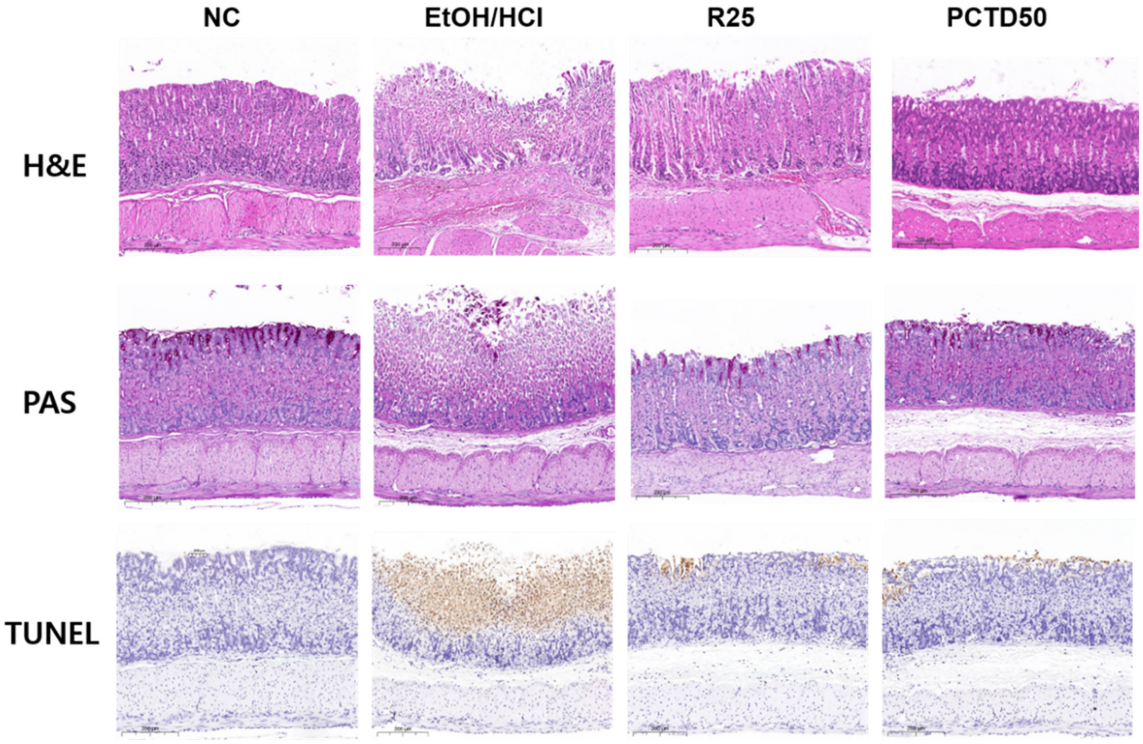


Fig. 7. Effects of PCTD on histopathological changes in the stomach of mice with EtOH/HCl-induced gastric mucosal injury. Representative H&E-, PAS-, and TUNEL-stained gastric tissue sections are shown. NC–Normal control group; EtOH/HCl–EtOH/HCl group; R25–EtOH/HCl-treated group administered ranitidine (25 ㎎/㎏); and PCTD50–EtOH/HCl-treated group administered PCTD (50 ㎎/㎏).

positive control group (R25) demonstrated a similar degree of improvement to the treatment group.

3.2. Histopathological analysis

H&E staining was performed to observe histopathological changes in the damaged gastric mucosa and those induced by PCTD administration. The EtOH/HCl group exhibited characteristic damage, including surface epithelial loss, mucosal folding, hemorrhage, and inflammatory cell infiltration. In the positive control group treated with 25 ㎎/㎏ ranitidine and the PCTD 50 ㎎/㎏ group, although epithelial damage, muscularis hemorrhage, and inflammatory cell infiltration were still

visible, the histopathological changes appeared less pronounced compared with the EtOH/HCl group (Fig. 7).

Analysis of mucus secretion function is an important indicator for evaluating gastric mucosal protective efficacy. Periodic acid-Schiff (PAS) staining enables visualization of mucus-secreting cells in the epithelial layer as purple-stained areas. PAS-positive staining appeared less extensive in the damaged areas than in the normal group. In contrast, representative sections from the PCTD-treated group showed more extensive PAS-positive staining than those from the EtOH/HCl group, with a staining pattern similar to that observed in the positive control group.

To assess DNA fragmentation associated with gastric mucosal injury, TUNEL staining was performed. Representative sections from the EtOH/HCl group showed more extensive TUNEL-positive staining than those from the normal group. In contrast, TUNEL-positive staining appeared less extensive in the ranitidine- and PCTD-treated groups than in the EtOH/HCl group.

DISCUSSION

Inflammation is a complex defensive physiological response triggered by the immune system to tissue injury, infection, or harmful stimuli (Zarrin *et al*., 2021). Gastritis is characterized by inflammatory damage to the gastric mucosa. Excessive apoptosis or necrosis of gastric epithelial cells can contribute to mucosal damage, leading to structural disruption and hemorrhage (Yan *et al*., 2024). Current therapeutic options for gastritis include antacids, proton pump inhibitors (PPIs), and histamine H_2 receptor antagonists (Kim *et al*., 2025). Recently, pharmacologically active plant-derived compounds have received attention as promising therapeutic and preventive candidates for gastric mucosal injuries (Lee *et al*., 2022; Chen *et al*., 2025).

Ethanol and HCl aggravate gastric mucosal damage by inducing inflammatory responses, while hydrochloric acid further damages the gastric mucus layer and gastric tissue (Li *et al*., 2008). Therefore, to investigate the anti-inflammatory and gastric mucosal protective effects of PCTD, this study employed LPS-stimulated RAW264.7 macrophage inflammation models *in vitro* and an EtOH/HCl-induced acute gastric mucosal injury model in mice.

Oxidative stress is caused by an imbalance between oxidation and antioxidant defense systems. Excess ROS production can activate inflammatory pathways and promote the expression of inflammatory mediators and cytokines (Lingappan, 2018; Nam *et al*., 2021). Numerous studies have reported that plant extracts exhibit antioxidant activity through free radical scavenging (Hwang *et al*., 2023; Heckmann *et al*., 2024). In the present study, PCTD showed DPPH and ABTS radical scavenging activity, and the scavenging activity increased with PCTD concentration (Fig. 1). These results indicate that PCTD possesses antioxidant potential.

The anti-inflammatory efficacy of bioactive agents is commonly screened through LPS-induced macrophage models. LPS, a component of the outer membrane of Gram-negative bacteria, stimulates macrophages to release a range of inflammatory cytokines and mediators (Wang *et al*., 2017). Produced by inducible nitric oxide synthase (iNOS), NO serves as a key inflammatory mediator, with its overproduction exacerbating inflammatory pathways (Alderton *et al*., 2001). PCTD treatment did not significantly reduce the MTS signal at concentrations up to 200 ㎍/mL (Fig. 2A) and significantly reduced LPS-induced NO production (Fig. 2B).

PGE_2 is another important inflammatory mediator associated with cyclooxygenase activity (Wallace, 2008), and regulation of excessive PGE_2 production has been associated with the attenuation of inflammatory responses. Simultaneously, it drives the inflammatory cascade forward by stimulating immune cells to upregulate inflammatory cytokine production (Halter, 2001). IL-1β and IL-6 are representative pro-inflammatory cytokines involved in the amplification of inflammatory responses (Wang *et al*., 2017; Gong *et al*., 2021). Also, granulocyte colony-stimulating factor (G-CSF) and granulocyte-macrophage colony-stimulating factor (GM-CSF) are produced by macrophages during inflammatory responses and can contribute to the amplification of inflammation together with inflammatory cytokines (Lee and Park, 2015; Hwangbo *et al*., 2021). Recently, it has been reported that natural anti-inflammatory flavonoids such as quercetin not only inhibit major pro-inflammatory cytokines in stimulated macrophages but also downregulate growth factors such as G-CSF and GM-CSF (Kim and Park, 2016). In this study, PCTD significantly reduced LPS-induced PGE_2 production (Fig. 3). Also, PCTD treatment showed decreasing trends in IL-6, IL-1β, and G-CSF levels, whereas GM-CSF was significantly reduced at 200 ㎍/mL (Fig. 4). These results support the anti-inflammatory activity of PCTD. COX-2 is associated with inflammatory prostaglandin production, whereas IL-10 plays an important role in limiting excessive inflammatory responses (DuBois *et*

al., 1998; Saraiva *et al*., 2020). Similar changes in COX-2, PGE_2, and IL-10 have been reported in LPS-stimulated RAW264.7 macrophages following anti-inflammatory treatment (Chou *et al*., 2003). Consistent with these findings, PCTD reduced COX-2 and IL-1β expression and increased COX-1 and IL-10 expression (Fig. 5).

Gastritis refers to inflammatory damage to the gastric mucosa caused by exogenous stimuli, often accompanied by features such as mucosal hyperemia, hemorrhage, and erosion (Kim *et al.,* 2023). The gastric mucosal protective effect of PCTD was further evaluated in the EtOH/HCl-induced acute gastric mucosal injury model. EtOH/HCl administration caused extensive hemorrhagic lesions and mucosal damage, whereas PCTD treatment reduced the damaged gastric mucosal area (Fig. 6). These findings indicate that PCTD attenuated acute gastric mucosal injury induced by EtOH/HCl.

Histological findings further supported the protective effect of PCTD against EtOH/HCl-induced gastric mucosal injury. EtOH/HCl treatment caused epithelial disruption, hemorrhage, and inflammatory cell infiltration, whereas these histopathological changes were attenuated in the PCTD-treated group (Fig. 7). To protect the gastric mucosa from external stimuli, mucus-secreting cells in normal tissue secrete mucus. However, when stimulated, increased pepsin production and reduced mucosal blood flow can impair mucus secretion. Decreased mucus secretion weakens protection against external stimuli, leading to damage and inflammation in the mucosa, submucosa, and muscularis layers (Hagen, 2021; Jia *et al*., 2023). Previous studies have shown that ethanol-induced gastric injury is accompanied by disruption of the gastric epithelium and impairment of the mucus barrier (Li *et al*., 2021; Hagen, 2021). Consistent with these findings, representative PAS-stained sections showed less extensive positive staining in the EtOH/HCl group than in the normal group, whereas more extensive PAS-positive staining was observed following PCTD treatment (Fig. 7). In addition, to assess DNA fragmentation associated with gastric mucosal injury, TUNEL staining was performed. Representative TUNEL-stained sections showed more extensive positive staining in the EtOH/HCl group than in the normal control group, whereas staining appeared less extensive following PCTD treatment, with a pattern similar to that observed in the positive control group (Fig. 7). These qualitative observations suggest that PCTD treatment was associated with less pronounced gastric mucosal damage.

Because the present *in vivo* study evaluated a single PCTD dose, further studies using multiple doses would be useful to characterize the dose-response relationship. In addition, as ranitidine was used as the sole positive control, the present study could not distinguish acid-suppressive effects from direct mucosal-protective effects, which should be considered a limitation of the present study.

In this study, the antioxidant and anti-inflammatory activities of the hot-water extract of Phyllostachyos Caulis in Taeniam (PCTD) were confirmed through *in vitro* and *in vivo* assays. Together, these findings demonstrate that PCTD attenuates EtOH/HCl-induced acute gastric mucosal injury in mice. Further studies are needed to clarify the molecular mechanisms underlying these effects.

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