# Anti-*Helicobacter pylori* activity and inhibition of gastritis by *Magnolia officinalis* extract

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## *Abstract*

The potential inhibitory effect of *Magnolia officinalis* extract (MOE) on *Helicobacter pylori* was studied by using (i) disk agar diffusion assay, (ii) *in vitro* inhibition assays of *H. pylori* adhesion to human gastric adenocarcinoma epithelial cell line AGS cells, (iii) inhibition of *H. pylori*-induced inflammation in AGS cell, and (iv) *in vivo H. pylori* SS1 mouse model of infection over a period of 4 weeks. Results indicated that MOE had a wide range of inhibitory effect against *H. pylori* growth. The inhibition of *H. pylori* adherence to gastric epithelial cells was identified by MOE. In addition, MOE inhibited *H. pylori*-induced inflammatory response significantly by reduced interleukin (IL)-8 expressions in *H. pylori*-infected AGS cells. Also, significant inhibition activity of MOE against *H. pylori* was identified in a mouse model. *In vivo* experiments were performed by per oral inoculation of MOE over a period of 4 weeks to fifty C57BL/6 mice previously infected with *H. pylori* SS1. Rapid urease tests of the mice stomachs demonstrated a significant reduction in *H. pylori* colonization. In addition to the therapeutic effect against *H. pylori* infection, the MOE reduced mucosal inflammation and epithelial damages in the stomach of the *H. pylori*-infected mice. These results demonstrate that the MOE successfully cured *H. pylori* infection and prevented *H. pylori*-induced pathology. Therefore, MOE could be a promising treatment for patients with gastric complaints including gastritis caused by *H. pylori*.

## Introduction

*Helicobacter pylori* is the most important etiological agent of chronic gastritis and peptic ulcers and also increases the risk for gastric cancer (Sugiyama et al., 1998; Maruta et al., 2001). Accumulating evidence has demonstrated that eradicating *H. pylori* in the stomach by administering oral antimicrobial agents results in the resolution of *H. pylori*-associated gastroduodenal diseases (Asaka et al., 2001; Salih et al., 2005). In addition, the eradication of *H. pylori* decreases the risk for gastric carcinogenesis (Maruta et al., 2005; Kim, 2012). Triple therapy regimens are currently accepted as first-line therapy. They combine a proton pump inhibitor (PPI) with either metronidazole and clarithromycin, or amoxicillin and clarithromycin (Misiewicz et al., 1997); however, such combination therapy does not always successfully eradicate *H. pylori* (Kim, 2012).

In recent years, the increased occurrence of clarithromycin- and/or metronidazole-resistant strains of *H. pylori* has become a problem (Midolo et al., 1996; Kim, 2012). Furthermore, antibiotics cannot be used due to drug allergies in some patients, and antibiotic therapy is also occasionally associated with adverse events. Moreover, it is considered inappropriate to administer long-term antimicrobial agents to prevent *H. pylori* infection. Antibiotic chemotherapy occasionally produces side effects and fails to eliminate bacterial infection (Buenz et al., 2007). The occurrence of strains resistant to antibiotics is expected to increase; thus, it is important to search for nonantibiotic substances to cure these infections (Liu et al., 2007; Kim, 2012).

*Magnolia officinalis* is grown in Asian countries such as China, Thailand and Korea. Its bark has been used for many years in traditional Chinese medicines and Japanese remedies for the treatment of a variety of mental disorders, including depression (Maruyama et al., 2003). It is well known that *M. officinalis* is a major component in herbal formulations such as Banxia-houpu decoction and Saiboku-to used as remedies for depression and other illnesses, e.g. cough, asthma, liver disease, shoulder pain, urinary problems and diarrhea (Wang et al., 2005). Magnolia extract, produced primarily from the dried stem, root or branch bark of *M. officinalis* (Chang and But, 1986), is a constituent of dietary supplements and topically applied cosmetic products. It contains the active compounds magnolol and honokiol, which have various pharmacological activities, including anti-inflammatory effects (Wang et al., 1995), antimicrobial activity (Chang et al., 1998; Ho et al., 2001), antioxidant activity and free radical-scavenging activity (Lo et al., 1994). The inhibitory effects of *M. officinalis* extract (MOE) against *Listeria monocytogenes*, *Streptococcus faecalis*, *Escherichia coli*, *Salmonella typhimurium*, *S. aureus* and *Bacillus anthracis* were reported (Hu et al., 2011).

The goal of this study was to determine the antibacterial activity and inhibition of *Helicobacter pylori*-induced inflammation effects of the *Magnolia officinalis* extract (MOE).

## Materials and Methods

***Preparation of Magnolia officinalis extract:*** The dry bark of *Magnolia officinalis*, purchased from Oriental Pharmacy (Iksan, Korea), was according to the standard mentioned in Korean Pharmacopoeia and Korean Herbal Pharmacopoeia, which are the official compendia of standard. The procedures for preparing MOE were as follows. Plant extract preparations and tests were conducted as triple repetition. The air-dried bark of *M. officinalis* (1 kg) was cut into pieces and extracted twice with 70% (v/v) ethanol (three times as much as the weight of the dried plants) for 3 hours at 100°C. After filtration through a 400-mesh filter cloth, the filtrate was refiltered through filter paper (Whatman, No. 5) and concentrated on a rotary evaporator (EYELA, Tokyo, Japan), then the concentrated filtrate was evaporated to dryness under vacuum with a freezing dryer (Labconco, USA). Finally, the solid residue was collected, placed in sealed bottles and stored at -20°C.

***Ultra-Performance Liquid Chromatography (UPLC) analysis of MOE:*** Magnolol (5, 5'-di-2-propenyl-(1,1'biphenyl)-2,2'-diol), which was used for the standard material of MOE composition, was purchased from the Korean Food & Drug Administration (Cheongwon-gun, Korea). Figure 1 shows the chemical structures of magnolol. The magnolol concentration of MOE was analyzed by UPLC. Waters ACQUITY UPLC system (Waters Corp., Milford, USA) was used for UPLC system. The column was C18 type ACQUITY UPLC BEH (2.1 × 50 mm, 1.7 μm Waters Corp., Milford, USA). A Waters Nova Pack C-18 column (ACQUITY UPLC BEH (2.1 × 50 mm 1.7 μm, Waters Corp., Milford, USA) was employed. The wavelength of the UV detector was set at 300 nm. The column temperature was set at 30°C with a flow rate of mobile phase at 0.6 mL/min (0.1% $H_3PO_4$/Acetonnitrile).

***Determination of anti-H. pylori activity:*** The *in vitro* anti-*H. pylori* activities of MOE were determined by disk agar diffusion method (Castillo-Juarez et al., 2007). Briefly, a total volume of 100 μl of *H. pylori* suspension ($1 × 10^8$ colony forming units (CFUs)/ml) was spread onto Mueller Hinton agar plates (BBL) containing 10% sheep blood. Sterile paper disks (6 mm, BBL) were placed on the agar surface with 3 different concentrations of MOE extracts (12.5, 25 and 50 μg/ml) individually. DMSO was used as negative control and antibiotics amoxicillin (AMX, 0.05 mg/ml), clarithromycin (CLR, 0.05 mg/ml), and metronidazole (MTZ, 0.8 mg/ml) were used as positive controls. After 72 h of incubation at 37°C under a microaerophilic condition with humidity, the inhibition zone was determined in diameter.

***AGS cell culture:*** AGS cells (ATCC CRL 1739; human gastric adenocarcinoma epithelial cell line) were cultured in RPMI (Sigma) supplemented with 10% de-complement fetal bovine serum (Invitrogen). Penicillin and streptomycin (GIBCO BRL) were also added if needed. In the *H. pylori*-induced IL-8 secretion, the cell

culture medium was not supplemented with antibiotics.

***Inhibition of H. pylori adhesion to AGS cells:*** *H. pylori* adhesion to cultured AGS cells was done using a standard gentamicin assay as previously described (Lai et al., 2006). MOE extracts (12.5, 25 and 50 µg/ml) and DMSO diluted in cell culture medium were added (to reach the indicated dilutions) directly to the cell culture medium for 10 min prior to inoculation of wells with *H. pylori* in log-phase. *H. pylori* was added to the AGS cells at MOI of 50, then the cells were incubated at 37°C for 6 h. To determine the number of cell-adhesion bacteria, the infected cells were washed three times to remove unbound bacteria and then lysed with distilled water for 10 min. Lysates were diluted in PBS, plated onto Brucella blood agar plates and cultured for 4-5 days, after which the CFUs were counted. To determine the number of viable intracellular bacteria, the infected cells were washed three times in PBS and incubated with 100 µg/ml of the membrane-impermeable antibiotic gentamicin (Sigma-Aldrich) for 1.5 h at 37°C to remove extracellular bacteria, followed by the same procedures as above to obtain CFUs. Adhesion activity was determined as the mean of at least six experiments performed in duplicate. The controls containing *H. pylori*-infected AGS cells without the test samples were used to establish 100% adhesion. Results were expressed as percentage of relative inhibition of *H. pylori* adhesion, as compared with the controls.

***Determination of interleukin-8:*** AGS cells ($2 \times 10^4$ cells) were seeded in 96-well plates. The cells were pretreated with MOE (12.5, 25, 50 and 100 µg/mL) for 24 hr. The cells were then infected with *H. pylori* for 24 hrs. The culture medium was collected, and IL-8 measurement was performed using ELISA kits (Enzo Life Sciences, Farmingdale, NY, USA). Each sample was tested in triplicate.

***Animals:*** Specific pathogen-free (SPF) male C57BL/6 mice (body weights of 20-24 g) were procured from Samtako Co. (Osan, Korea). All animals were kept at the inspecting facility of Wonkwang University (Iksan, South Korea) for 1 week to allow acclimation before experimentation. Thereafter, they were kept in an isolated SPF barrier room with regulated temperature (23 ± 1°C), humidity (50 ± 5%) and light/dark cycle (12/12 h). The animals were fed a sterilized (2 M rad radiation) pellet diet (Purina, Seoul, Korea) and sterilized water *ad libitum*. All studies were performed in accordance with the Guide for Animal Experimentation of Wonkwang University and approved by the Institutional Animal Care and Use Committee of Wonkwang University (WKU16-265). All efforts were made to minimize pain or discomfort to the animals used.

***Bacterial inoculation:*** *H. pylori* SS1 (B0890, Helicobacter pylori Korean Type Culture Collection, HPKTCC) was incubated in brain-heart infusion broth containing 10% fetal bovine serum at 37°C overnight under a microaerophilic atmosphere and allowed to grow to a density of ~$2.0 \times 10^9$ CFU/ml culture broth.

The animals were inoculated 3 times at 3-day intervals by intragastric inoculation of $1.0 \times 10^9$ CFU *H. pylori* suspended in 0.5 ml broth. The challenged animals were confirmed to be *H. pylori*-positive by the stool antigen analysis with SD Bioline *H. pylori* Ag kit (Standard Diagnostics, Inc) as described previously (Moon et al., 2013).

***In vivo study protocol:*** The inhibition effect of MOE on *H. pylori* infection was investigated using a mouse model. Fifty mice were divided into 5 groups: negative control (group I, n = 10); *H. pylori*-infected, MOE-untreated animals (group II, n = 10); *H. pylori*-infected, MOE (25 mg/kg)-treated animals (group III, n = 10); *H. pylori*-infected, MOE (50 mg/kg)-treated animals (group IV, n = 10); and *H. pylori*-infected, MOE (100 mg/kg)-treated animals (group V, n = 10). The MOE was administered orally daily at 25, 50 and 100 mg/kg/day dose during a 4-week treatment period.

At 4 weeks after *H. pylori* inoculation, all animals were sacrificed and their stomachs were dissected after ether euthanasia. Each stomach was opened along the greater curvature and washed with saline. Then, half of the glandular mucosa was scraped for detection of colonizing *H. pylori*, and the residual part was formalin-fixed and embedded in paraffin for histological observations. *H. pylori* colonization was confirmed by rapid urease tests, as described previously (Lee et al., 2010a; 2010b). Mucosal damage was evaluated grossly and histologically according to previously described criteria (Lee et al., 2010b).

***Statistical analysis:*** Values for all parameters under study were recorded for each experimental unit, and statistical analysis was performed using a general linear model. Values are reported as average ± standard deviation, when appropriate. Student's *t*-test was used for pair-wise comparisons. Incidence percentages (95% confidence intervals [CIs]) were calculated with the MiniTab statistical software program (Minitab, State College, PA, USA). A *P*-value < 0.05 was considered significant.

## Results

***Yield of M. officinalis extract (MOE):*** The extract yield of dry bark of *M. officinalis* with 70% ethanol was 30%. MOE composition was analyzed by UPLC. The retention time of magnolol in the specified UPLC condition was 7.886 min. The concentration of magnolol in MOE was 10.21 ± 0.10%.

***Growth inhibition of H. pylori:*** The investigation into relative inhibitory potency of MOE against *H. pylori* SS1 growth was conducted using the disk agar diffusion assay. The MOE extracts were tested at a maximum concentration of 100 µg/ml. For referencing, the inhibitory effects of standard antimicrobial agents for the treatment of *H. pylori* infection, clarithromycin (CLR), amoxicillin (AMX), both at 50 µg/ml, and metronidazole (MTZ), at 800 µg/ml, were determined as well. To quantify the inhibitory effect of *H. pylori*, the diameter of growth inhibition area was measured and expressed in millimeters, as presented in Table 1. As shown in Table 1, the tested agents showed a wide

range of inhibitory effect against *H. pylori* growth with inhibition zone ranging from 0 to 20.9 mm. The antibiotics CLR, AMX and MTZ inhibited the bacterial growth with the inhibition zone of 19.9, 15.8 and 7.6 mm, respectively. The 100 ug/ml MOE showed an inhibition zone value of 20.9 mm (Table 1).

***Inhibition of H. pylori adhesion to AGS cells:*** The MOEs (12.5, 25, 50 and 100 µg/ml individually) were assayed with regard to its ability to inhibit the adhesion of *H. pylori* to AGS cells. As shown in Fig. 1, 12.5, 25, 50 and 100 µg/ml of MOE exhibited significant decrease in *H. pylori* infection percentages and a marked anti-adhesion activity against *H. pylori* by 27.5%, 45.7%, 60.5% and 90.2%, respectively, compared to the *H. pylori*-infected cells without MOE. The results from this study demonstrate that MOE has the ability to inhibit bacterial adhesion of AGS cells.

***Effect of MOE on H. pylori-induced IL-8:*** IL-8 production in gastric epithelial cells infected with *H. pylori* with or without pre-incubation of MOE from 12.5 to 100 µg/ml was measured. The pretreatment of *H. pylori*-infected AGS cells with 12.5, 25, 50 and 100 µg/ml of MOE for 24 hrs significantly decreased IL-8 production and a marked anti-inflammatory activity

against *H. pylori*-induced IL-8 by 26.0%, 36.5%, 54.6% and 71.0%, respectively, compared to the *H. pylori*-infected cells without MOE (Fig. 2).

***Results of rapid urease test CLO:*** Repeated intragastric inoculation ($1.0 \times 10^9$ CFU/mouse, 3 times) of *H. pylori* to C57BL/6 mice revealed positive reaction (red color) in CLO test on the gastric mucosa (Table 2). The stomachs of *H. pylori*-infected mice orally treated with 25, 50 and 100 mg/kg/day doses of MOE during the 4-week treatment period displayed positive reaction in 70%, 30% and 10%, respectively.

***Results of histopathological analysis:*** Pathological changes in the gastric mucosa were negligible in the animals from non-*H. pylori*-inoculated groups (I). In contrast, gastric atrophy and ulceration developed in the gastric mucosa of the mice in group II (*H. pylori* inoculation only), indicating marked mucosal destruction. However, the mice in groups III, IV and V (*H. pylori* + MOEs of 25, 50 and 100 mg/kg/day individually) revealed markedly improved villi lesions. The histopathological lesion score in the MOE-treated animals was significantly lower than that in the infection control group II (*H. pylori* inoculation only) (Table 3).

**Table 1**    Anti-*H. pylori* activities of *Magnolia officinalis* extract (MOE) using disk agar diffusion method

| Treatment (µg/ml) | | Clear zone (mm) |
|---|---|---|
| Clarithromycin | (50) | 19.9 ± 0.40 |
| Amoxicillin | (50) | 15.8 ± 0.49 |
| Metronidazole | (800) | 7.6 ± 0.55 |
| MOE | (0) | 0 ± 0 |
| | (12.5) | 5.5 ± 0.76 |
| | (25) | 11.4 ± 0.71 |
| | (50) | 15.4 ± 0.61 |
| | (100) | 20.9 ± 0.10 |

**Table 2**    Reactivity in CLO test on gastric mucosa of mice infected with *H. pylori* followed by treatment with or without *Magnolia officinalis* extract (MOE)

| Group | Treatment | n | Positive %[a] | Therapeutic % |
|---|---|---|---|---|
| I | No treatment | 10 | 0% (CI[b]0-27.6) | - |
| II | *H. pylori* | 10 | 100% (CI 72.2-100) | 0% (CI 0-27.6) |
| III | *H. pylori*+MOE 25 mg/kg | 10 | 70% (CI 39.7-89.2) | 30% (CI 10.8-60.3) |
| IV | *H. pylori*+MOE 50 mg/kg | 10 | 30% (CI 10.8-60.3) | 70% (CI 39.7-89.2) |
| V | *H. pylori*+MOE 100 mg/kg | 10 | 10% (CI 1.8-40.4) | 90% (CI 60.0-98.2) |

[a]The positive percent reveals *H. pylori* colonization, which was observed as the red color change from yellow color medium.
[b]Incidence percentage (95% confidential interval) was calculated with MiniTab statistic software program.

**Table 3**    Histopathological lesions scores of mice infected with *H. pylori* followed by treatment with or without *Magnolia officinalis* extract (MOE)

| Group | Treatment | n | Histopathological lesion score |
|---|---|---|---|
| I | No treatment | 10 | 0 ± 0 |
| II | *H. pylori* | 10 | 6.9 ± 0.74 |
| III | *H. pylori*+MOE 25 mg/kg | 10 | 3.9 ± 0.88* |
| IV | *H. pylori*+MOE 50 mg/kg | 10 | 2.6 ± 0.84* |
| V | *H. pylori*+MOE 100 mg/kg | 10 | 0.9 ± 0.57** |

*, **Significantly different from the control group II (* $P < 0.05$, ** $P < 0.01$)
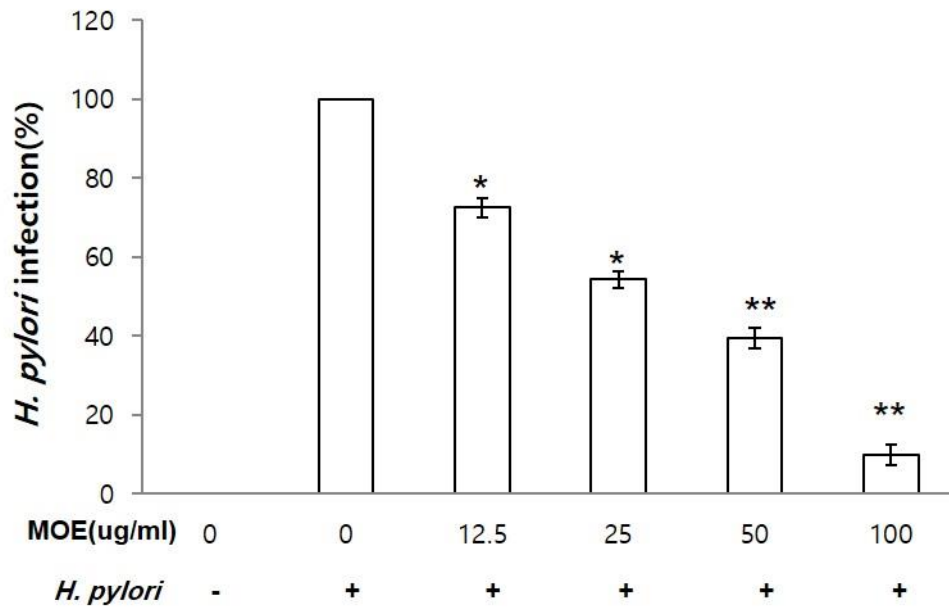
**Figure 1** Inhibition assay of *H. pylori* adhesion to human gastric mucosal AGS cell. *Magnolia officinalis* extract (MOE) of 12.5, 25, 50 and 100 µg/ml were individually assayed with regard to its ability to inhibit the adhesion of *H. pylori* to AGS cells.
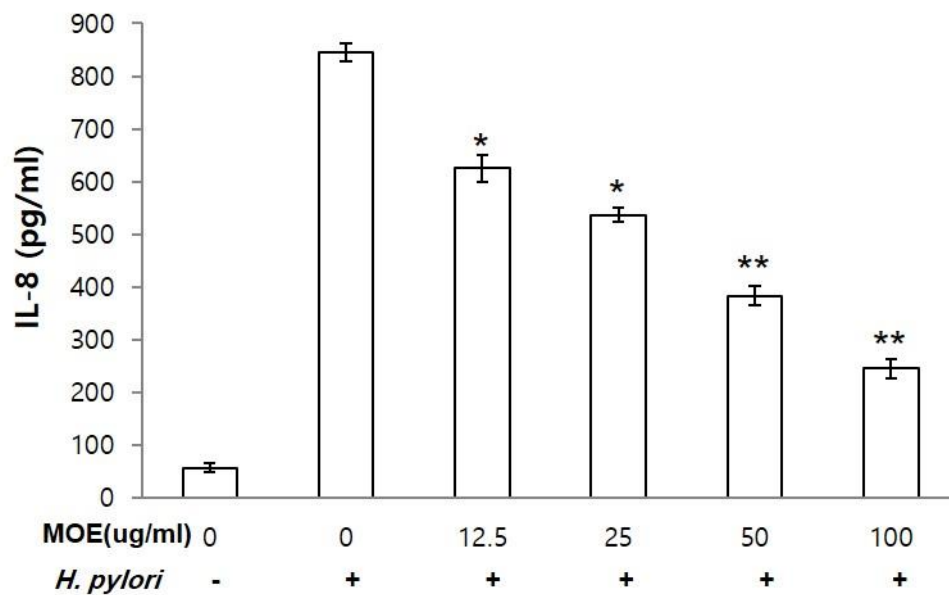


**Figure 2** Results of IL-8 inflammatory cytokine in human gastric mucosal AGS cell line. *Magnolia officinalis* extract (MOE) induced decrease in IL-8 cytokine in *H. pylori*-infected gastric epithelial AGS cells.

## Discussion

*H. pylori* highly colonize the human gastric mucosa and perfectly adapt to that environment. Concerning pathology, *H. pylori* causes gastritis and is classified as a major primary risk factor for the development of gastric or peptic ulcers, gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma (Wroblewski and Peek, 2010; Kim, 2012).

In the present study, the inhibition of *H. pylori* adherence to gastric epithelial cells was identified by MOE. In addition, MOE inhibited the *H. pylori*-induced inflammatory response by the reduced IL-8 expressions in *H. pylori*-infected AGS cells. Also, the inhibition activity of MOE against *H. pylori* was

identified in a mouse model. The rapid urease tests of the mice stomachs demonstrated a significant reduction in *H. pylori* colonization. In addition to the therapeutic effect against *H. pylori* infection, the MOE reduced mucosal inflammation and epithelial damages in the stomach of *H. pylori*-infected mice. It is likely that *H. pylori* eradication caused a decrease in the degree of inflammation in the stomach, although there is a possibility that MOE itself has an anti-inflammatory effect on gastric mucosa.

MOE contains a large amount of polyphenolic compounds (magnolol, honokiol, tetrahydromagnolol, isomagnolol, etc.). Previous studies have shown that phenolic compounds can affect microbial growth by altering microbial cell permeability and permitting the loss of macromolecules inside the cell. Once the

phenolic compounds have crossed the cell membrane, interactions with membrane enzymes and proteins will cause an opposite flow of protons, affecting cellular activity. It has also been suggested that cell membrane of *Pseudomonas aeruginosa*, causing an increase in permeability (Bernheim, 1972). This theory was substantiated by rapid swelling of *P. aeruginosa* cells due to phenol (Puupponen-Pimia et al., 2001). Davidson (1993) reported that phenolic compounds might act on microbial cell walls or membranes, proposing that they inhibit microbial growth by altering microbial cell permeability, which leads to the loss of intracellular molecules such as proteins, DNA, RNA and ATP. Phenolic compounds could also interact with membrane proteins, causing damage to their structure and functionality. Conner and Beuchat (1984) suggested that the antimicrobial activity of essential oils against yeasts could be a result of the disturbance of several enzymatic systems involved in energy production and the synthesis of structural components.

IL-8 is a chemoattractant factor for neutrophil recruitment and a critical immune mediator for the pathogenesis of chronic gastritis caused by *H. pylori* infection. In addition, various studies have reported that high expression of IL-8 correlates with poor prognosis of gastric cancers or gastrointestinal tumorigenesis, including angiogenesis (Asfaha et al., 2013). Therefore, IL-8 has been suggested as a therapeutic target in gastric cancer. The present study revealed that the *in vitro* treatment of MOE effectively inhibited *H. pylori*-induced production of IL-8 in gastric epithelial cells, suggesting that MOE may have preventive as well as therapeutic effects on *H. pylori*-mediated inflammation.

Although triple therapy, using two antibacterial antibiotics and a proton pump inhibitor, is effective and its short duration helps maintain patient compliance, a considerable number of patients experience undesirable side effects, such as diarrhea, epigastric pain, nausea, and bloating (Sakamoto et al., 2001). In contrast, MOE is relatively safe. These safe characteristics of MOE may, therefore, be appropriate for use in prevention and therapy against *H. pylori* infection. As chronic inflammation and increased cell proliferation are features common to the pathogenesis of many human cancers, and as these features seem to play a central role in initiating and promoting carcinogenesis, the suppression of inflammation and cell proliferation by MOE may be an effective modality for the prevention of *H. pylori*-induced carcinogenesis of the stomach.

In conclusion, MOE showed a significant inhibition effect against *H. pylori* infection. It could be a promising MOE treatment for patients with gastric complaints including gastric ulcers caused by *H. pylori*. MOE may be useful to treat patients with an *H. pylori* infection with high therapeutic efficacy and safety.

## *Acknowledgements*

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# บทคัดย่อ

## การออกฤทธิ์และการยับยั้งกระเพาะอาหารอักเสบที่เกิดจากเฮลิโคแบกเตอร์ ไพโลไร โดยสารสกัดจากแมกโนเลีย ออฟฟิสินาลิส

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การศึกษาศักยภาพของสารสกัดจากแมกโนเลีย ออฟฟิสินาลิส (MOE) ในการยับยั้งเฮลิโคแบกเตอร์ ไพโลไร ด้วยวิธี (i) ดิสก์อะกาดิฟฟิวชั่น  (ii) การยับยั้งเอช ไพโลไรในการจับกับเซลล์ไลน์ AGS ของมะเร็งกระเพาะอาหารของมนุษย์ (iii) การยับยั้งเอช ไพโลไรที่เหนียวนำให้เกิดการอักเสบใน AGS และ IV การศึกษาSS1 ในหนูไมซ์เกี่ยวกับการทำให้ติดเชื้อ เอช ไพโลไร โดยศึกษาเป็นเวลา 4 สัปดาห์ ผลการศึกษาบ่งชี้ว่า MOE มีผลยับยั้งการเจริญเติบโตของเอช ไพโลไร MOE ยับยั้งการจับของเอช ไพโลไร ที่เยื่อบุกระเพาะอาหาร  นอกจากนี้ MOE ยับยั้งขบวนการอักเสบที่เหนียวนำโดย เฮช ไพโลไรโดยมีผลลดอินเตอร์ลิวคิน -8 ใน AGS ที่ติดเชื้อนี้  การยับยั้งเอช ไพโลไรในการทดลองในหนูโดย MOE มีนัยสำคัญ  การศึกษาภายในร่างกายทำโดยป้อน MOE แก่หนูไมซ์ ที่ติดเชื้อเฮช ไพโลไร SS1 เป็นเวลา 4 สัปดาห์  นำกระเพาะอาหารของหนูทดลองมาตรวจด้วยวิธี ยูรีเอสเร่งด่วนพบว่าการสร้างโคโลนีของเอช ไพโลไร ลดลงอย่างมีนัยสำคัญ  นอกจากนี้ยังพบว่า MOE ลดการอักเสบของมิวโคซาและลดการเสียหายของเยื่อบุกระเพาะอาหารในหนูไมซ์ที่ติดเชื้อ  เอช  ไพโลไร  ผลการศึกษานี้แสดงให้เห็นถึงความสำเร็จของ MOE ในการรักษาการติดเชื้อเอช ไพโลไร และป้องกันการเหนี่ยวนำให้เกิดรอยโรคทางพยาธิวิทยาของเอช ไพโลไร  ดังนั้น MOE สามารถใช้รักษาผู้ป่วยโรคกระเพาะอาหารรวมถึงกระเพาะอาหารอักเสบจาก เอช ไพโลไร

**คำสำคัญ:** แมกโนเลีย ออฟฟิสินาลิส เฮลิโคแบกเตอร์ เอช ไพโลไร ไอแอล-*8* กระเพาะอาหารอักเสบ

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