Prevalence of *Helicobacter pylori* vacA, cagA, cagE, iceA and babA2 genotypes in Thai dyspeptic patients

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**KEYWORDS**  
*Helicobacter pylori*; Prevalence; vacA; cagA; cagE; iceA; babA2; Thai dyspeptic patients

**Summary**  
Objectives: To investigate the prevalence of the vacA, cagA, cagE, iceA, and babA2 genotypes in *Helicobacter pylori* strains isolated from Thai dyspeptic patients, and to determine whether any correlation exists between these genotypes and clinical manifestations.  
Methods: *Helicobacter pylori* was examined in 112 patients (62 with non-ulcer dyspepsia (gastritis), 34 with peptic ulcer disease, and 16 with gastric cancer (GCA)), detected by culture or direct detection from gastric biopsies. Allelic variants of the vacA, cagA, cagE, iceA, and babA2 genotypes were identified by using the polymerase chain reaction.  
Results: The positive rates for the vacAs1, vacAs2, cagA, cagE, iceA1, iceA2, and babA2 genes in *H. pylori* of dyspeptic patients were 100%, 0%, 98.2%, 88.4%, 45.5%, 33.1%, and 92%, respectively. The allelic variant vacAs1m1 was more prevalent (58%) than vacAs1m2 (42%). The cagA and cagE genes were commonly found together (87.5%). The most predominant genotypes were vacAs1m1, cagA, cagE, iceA1, and babA2. The various genes alone or in combination had no statistically significant association with the clinical outcomes (p > 0.05).  
Conclusion: Neither single gene nor combination of vacA, cagA, cagE, iceA, and babA2 genes was significantly helpful in predicting the clinical outcome of *H. pylori* infection in Thai patients. The high prevalence of these genes in *H. pylori* isolated from Thai patient groups suggests that *H. pylori* strains are geographically dependent.

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Introduction

Helicobacter pylori is considered an important etiological agent in the development of gastritis, peptic ulcers and gastric carcinoma.\(^1,2\) The occurrence of such diverse diseases with \(H. pylori\) may depend on specific properties of the organism, host genetic factors, and environmental factors.\(^3\) Although more than 50% of the world's population is infected with \(H. pylori\), a minority of carriers develop serious gastrointestinal diseases;\(^3-5\) however, increasing evidence suggests that the genetic variability of \(H. pylori\) may itself be of clinical importance.\(^3-7\) Several putative genes, such as \(vacA, cagA, cagE, iceA\) and \(babA2\), have been identified and may play important roles in the pathogenesis of \(H. pylori\) infection.\(^8-10\)

The vaculating cytotoxin gene (\(vacA\)) is present in all \(H. pylori\) strains.\(^4\) The \(vacA\) genotype comprises a hypervariable signal sequence and a middle region allele. The \(vacA\) subtypes are determined by the combination of \(s1a, s1b, s1c\) and \(s2\), and \(m1, m2a\) and \(m2b\).\(^4,11\) Although all strains of \(H. pylori\) contain the \(vacA\) gene, they vary in terms of their ability to produce cytotoxin. Type \(m1\) strains demonstrate more toxic activity than \(m2\), type \(s1a\) is more active than \(s1b\), and type \(s2\) is less active than \(s1\).\(^6,12\)

The cytotoxin associated gene A (\(cagA\)) has been proposed as a marker for a genomically pathogenic island (\(cag-PAI\)) of approximately 40 kbp whose presence is associated with more severe clinical outcomes.\(^13,14\) The \(cagA\)-positive \(H. pylori\) strains are known to induce interleukin-8 (IL-8) production and mucosal inflammation.\(^13,14\) Other members of the \(cag-PAI\) have also been evaluated for their involvement in virulence, and \(cagE\) is one of the marker genes in \(cagA\) of the \(cag-PAI\). It is essential for \(cagA\) translocation and phosphorylation.\(^15,16\) The presence of the \(cagE\) gene has also been associated with a more severe clinical outcome.\(^17\)

The induced by contact with epithelium (\(iceA\)) gene has recently been discovered. The two main allelic variants of the gene are \(iceA1\) and \(iceA2\). The expression of \(iceA1\) is up-regulated on contact between \(H. pylori\) and human epithelial cells, and may be associated with peptic ulcer disease.\(^7,18,19\)

The blood group antigen-binding adhesin gene (\(babA\)) is involved in the binding activity between bacterial adhesin and human Lewis-b blood group antigens on gastric epithelial cells.\(^20\) Although three \(bab\) alleles have been identified (\(babA1, babA2, babB\)), only the \(babA2\) gene product is necessary for Lewis-b binding activity. Several researchers suggest that the presence of \(babA2\) is related to the occurrence of peptic ulcers and gastric cancer.\(^20,21\)

In Thailand, an average 48% of dyspeptic patients are infected with \(H. pylori\), but a higher prevalence has been found in gastric ulcer patients and peptic ulcer patients than in gastritis patients.\(^22-24\) Some virulence-related gene products such as \(VacA\) and \(CagA\) in the isolated strains have been studied;\(^25,26\) however, the involvement of \(H. pylori\) genotypes in specific diseases remains controversial.\(^8,9,21,27\) A mainstream challenge for researchers to identify the particular \(H. pylori\) genes, including \(vacA, cagA, cagE, iceA\) and \(babA2\), has been elucidated;\(^9\) however, no study in Thailand has simultaneously investigated the prevalence and relationship to clinical outcomes of these putative genes. To understand the clinical relevance of \(H. pylori\) genotyping in predicting infection outcomes, and \(H. pylori\) genes in different geographical regions for the basic knowledge of Thai dyspeptic patients, we investigated the prevalence of \(vacA, cagA, cagE, iceA\), and \(babA2\) genes of \(H. pylori\) obtained from 112 Thai patients with gastritis, peptic ulcers, and gastric cancer. The correlation between the genetic status of the isolates and the occurrence of gastrointestinal diseases was assessed.

Materials and methods

Patients

Gastric biopsies and \(H. pylori\) isolates were obtained from 112 patients who had undergone routine endoscopy for symptoms of dyspepsia at the hospitals in central and northeast Thailand. We included 34 patients with peptic ulcer disease (PUD; 20 with gastric ulcers (GU), 14 with duodenal ulcers (DU)), 62 with non-ulcer dyspepsia or gastritis (GT), and 16 with gastric cancer (GCA). The patients were 55 males and 57 females with an age range of 18 to 88 years (mean 49.5 years).

The study was approved by the ethics committee of Khon Kaen University and performed in accordance with the Declaration of Helsinki. Informed consent was obtained from each patient prior to entering the study.

Clinical samples and culture

Three gastric mucosal biopsy specimens from the antrum and corpus were obtained from each patient and divided into three parts. Both antral and corpus specimens were used for culture, the rapid urease test (RUT), and histological examination.

Culture was performed according to the method of Hazell,\(^28\) with modifications. Briefly, each antral and corpus specimen was immediately placed into Stuart's transport medium and brought to the laboratory within 2 h at 4 °C. Each of the biopsy specimens was homogenized separately in medium and brought to the laboratory within 24 hours. A positive RUT was indicated when the color changed from yellow to pink. The positive RUTs were confirmed by Gram staining, oxidase, catalase and urease tests. The \(H. pylori\) colonies were further used for DNA extraction.

Commercial rapid urease test (RUT, Pronto Dry test)

The RUT was performed according to the manufacturer's instructions (Medical Instruments Corp., Solothurn, Switzerland). Briefly, one antral and one corpus specimen together were directly inoculated onto the commercial RUT agar gel. The results were observed and recorded within 24 hours. A positive RUT was indicated when the color changed from yellow to pink. The positive RUTs were used for chromosomal DNA extraction if the culture was negative.
Genomic DNA extraction

DNA from 30 *H. pylori* isolates and 82 of each antrum and corpus gastric biopsy positive by the RUT, were extracted using the genomic DNA purification kit (Puregene, Gentra Systems, USA), according to the manufacturer’s instructions. Briefly, a loop full of cell culture or the gastric biopsy samples (obtained from the urease test agar assay homogenized with 200 µl of normal saline) were incubated with 450 µl cell lysis solution and 2.5 µl proteinase K solution for 3 h at 55 °C. The lysate was incubated at 98 °C for 10 min and then 2.5 µl RNase A solution was added to the cell lysate and incubated at 37 °C for 60 min. Then, 200 µl protein precipitation solution was added and centrifuged at 13 000 g for 3 min. The supernatant was collected and 400 µl of 100% isopropanol was added and centrifuged at 13 000 g for 5 min. The supernatant was carefully discarded. Then, 300 µl of 70% ethanol was added to the pellet and centrifuged. The ethanol was poured off and left to dry for 3 h. Then, 50 µl DNA hydration solution was added and incubated for 1 h at 65 °C. DNA was stored at –20 °C until used.

**PCR assays for *glmM* gene and virulence genes (*vacA*, *cagA*, *cagE*, *iceA*, *babA2*)**

Primer sequences, sizes, and conditions of PCR amplifications of the *glmM* gene — for detection and confirmation of *H. pylori* — and the virulence genes (i.e., *vacA*, *cagA*, *cagE*, *iceA*, and *babA2*) were designed based on published papers with a modification of PCR mixtures and PCR conditions (Table 1).

<table>
<thead>
<tr>
<th>Genes</th>
<th>Primer sequence (5’ → 3’)</th>
<th>PCR product (bp)</th>
<th>PCR conditions</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>glmM</em></td>
<td>AGGTTTTTAGGGTGTAGGCTTTTTCTAACTACCACCTAAGGC</td>
<td>294</td>
<td>93 °C, 1 min; 55 °C, 1 min; 72 °C, 1 min (35 cycles)</td>
<td>29</td>
</tr>
<tr>
<td><em>vacA</em></td>
<td>ATGGAAATACAAACACACCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>259/286</td>
<td>94 °C, 1 min; 52 °C, 1 min; 72 °C, 1 min (35 cycles)</td>
<td>12,30</td>
</tr>
<tr>
<td><em>cagA</em></td>
<td>TTGAAAACCTTACACCTGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>190</td>
<td>94 °C, 1 min; 52 °C, 1 min; 72 °C, 1 min (35 cycles)</td>
<td>30</td>
</tr>
<tr>
<td><em>cagE</em></td>
<td>GTGTTTCTAACCCAGAGGTAGTAAACAGGC CTGACTGTTAATCGCCATACCGCATGCCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>187</td>
<td>94 °C, 1 min; 52 °C, 1 min; 72 °C, 1 min (35 cycles)</td>
<td>30</td>
</tr>
<tr>
<td><em>iceA1</em></td>
<td>CTCGCTCGCTTTAGGTTTCGTTGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>213</td>
<td>94 °C, 1 min; 52 °C, 1 min; 72 °C, 1 min (35 cycles)</td>
<td>31</td>
</tr>
<tr>
<td><em>iceA2</em></td>
<td>CAACTGGTGGCTTGAACCGGAGGCTGCTGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>567/642</td>
<td>94 °C, 1 min; 52 °C, 1 min; 72 °C, 1 min (35 cycles)</td>
<td>30</td>
</tr>
<tr>
<td><em>babA2</em></td>
<td>CAAACGAAAAACAGGCTGCTGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>298</td>
<td>94 °C, 1 min; 60 °C, 1 min; 72 °C, 1 min (45 cycles)</td>
<td>32</td>
</tr>
<tr>
<td><em>babA2</em></td>
<td>TTAGAATTAAATACCAACACATGCTGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>508</td>
<td>94 °C, 1 min; 53 °C, 45 s; 72 °C, 45 s (35 cycles)</td>
<td>16</td>
</tr>
<tr>
<td><em>babA2</em></td>
<td>GTTGGTGTGATTACCATTTTCGTTGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>247</td>
<td>95 °C, 1 min; 57 °C, 1 s; 72 °C, 1 min (35 cycles)</td>
<td>7</td>
</tr>
<tr>
<td><em>babA2</em></td>
<td>GTTGGTGTGATTACCATTTTCGTTGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>229</td>
<td>95 °C, 1 min; 57 °C, 1 s; 72 °C, 1 min (35 cycles)</td>
<td>7</td>
</tr>
<tr>
<td><em>babA2</em></td>
<td>CAAACGAAAAACAGGCTGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAACGCTGCTTGAAGGCGCCAAAC</td>
<td>271</td>
<td>94 °C, 1 min; 45 °C, 1 min; 72 °C, 1 min (30 cycles)</td>
<td>33</td>
</tr>
</tbody>
</table>

Each PCR of *glmM*, *vacA*, *cagA*, *cagE*, *iceA*, and *babA2* was performed in a total volume of 50 µl containing 100 ng genomic DNA from *H. pylori* culture or 400 ng genomic DNA from gastric biopsies in which the RUT was positive, 200 µM each of dNTP (Gibco BRL, USA), 1 × PCR buffer (20 mM Tris-HCl, pH 8.4), 50 mM KCl, 1.5 mM MgCl2 (2 mM MgCl2 for *cagA*), 0.5 µM of each primer (0.2 µM for *babA2* and 0.3 µM for *cagA*), and 1.5 units of Taq polymerase (Gibco BRL, USA). For each batch of PCR assay, distilled water instead of the genomic DNA templates was used as a negative control.

The reaction mixtures were cycled in an automated thermal cycler (GeneAmp, PCR 2400, Perkin-Elmer, USA) under the conditions shown in Table 1. After amplification, 10 µl of PCR product was electrophoresed on 1.5–2% agarose gel, stained with ethidium bromide, and examined under UV illuminator.

**Data analysis**

Fisher’s exact test or the Chi-square test was used for analysis of categorical data. A *p*-value of <0.05 was considered statistically significant.

**Results**

*Helicobacter pylori*-infected patients were evaluated for the relation of age, gender, and ethnic group with the severity of disease as shown in Table 2. The severity of disease was diagnosed by endoscopic findings and a pathologist. The results show that there was no significant difference among these parameters with regard to the gastroduodenal patient disease as shown in Table 2. The severity of disease was diagnosed by endoscopic findings and a pathologist. The results show that there was no significant difference among these parameters with regard to the gastroduodenal patient.
patients, only two (1.8%) showed an absence of cag (data not shown).

The frequency distribution of the combination genotypes of H. pylori are presented in Table 4. The four major genotypes found were: (1) vacAs1m1, cagA, cagE, iceA1, and babA2 (22.3%); (2) vacAs1m2, cagA, cagE, iceA1, and babA2 (16.1%); (3) vacAs1m1, cagA, cagE, iceA2, and babA2 (15.2%); and (4) vacAs1m2, cagA, cagE, iceA2, and babA2 (11.6%). No significant difference was found among the patient groups (p > 0.05) (Table 4).

**Discussion**

The clinical relevance of the putative virulence-associated genes of H. pylori and geographical region is still a matter of controversy. The present study reported the relationship between some virulence genes (vacA, cagA, cagE, iceA, baby) of H. pylori and the clinical status among Thai patients.

All strains of H. pylori contain the vacA gene, but they vary in terms of their ability to produce cytotoxin. Type s1 and m1 strains demonstrate more toxin activity than s2 and m2 strains. In Western studies, the presence of vacAs1 patients, iceA1 and iceA2 were detected in 45.5% (51/112) and 33.1% (37/112), respectively. iceA1 was most commonly found in the duodenal ulcer patients (57.1%; 8/14), whereas iceA2 was most commonly found in the gastric ulcer patients (40%; 8/20). The iceA-negative strain, in which neither iceA1 nor iceA2 was detected, was found in 21.4% of the 112 H. pylori strains. This finding is similar to that reported by Han et al.

The babA2 gene was detected in 92% (103/112) of the H. pylori-infected patients. The babA2 gene was commonly found in all patient groups; however, there was no statistically significant difference in each of the individual genes among the patient groups (p > 0.05) (Table 3).
and cagA has been shown to be significantly associated with peptic ulcers.\(^1,10\) However, several studies in Asian populations have not confirmed this relationship, indicating that there are important geographic differences.\(^{19,27,36}\) All vacA genotypes from our 112 H. pylori-infected dyspeptic patients contained the s1 signal region while 58% and 42% of H. pylori strains possessed the m1 and m2 middle region, respectively. Our results are in agreement with previous reports that show a predominance of s1 in Asian populations; the s1 and m1 genotypes from our 112 H. pylori strains obtained from 112 patients with different clinical outcomes.

<table>
<thead>
<tr>
<th>Genotypes</th>
<th>Clinical status</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-ulcer GT(^a) (%) (N = 62)</td>
<td>Peptic ulcer</td>
</tr>
<tr>
<td></td>
<td>GU(^b) (%) (N = 20)</td>
<td>DU(^c) (%) (N = 14)</td>
</tr>
<tr>
<td>vacA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>s1m1</td>
<td>36 (58.1)</td>
<td>12 (60)</td>
</tr>
<tr>
<td>s1m2</td>
<td>26 (41.9)</td>
<td>8 (40)</td>
</tr>
<tr>
<td>cagA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>60 (96.8)</td>
<td>20 (100)</td>
</tr>
<tr>
<td>Negative</td>
<td>2 (3.2)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>cagE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>54 (87.1)</td>
<td>18 (90)</td>
</tr>
<tr>
<td>Negative</td>
<td>8 (12.9)</td>
<td>2 (10)</td>
</tr>
<tr>
<td>iceA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>iceA1</td>
<td>29 (46.8)</td>
<td>7 (35)</td>
</tr>
<tr>
<td>iceA2</td>
<td>19 (30.6)</td>
<td>8 (40)</td>
</tr>
<tr>
<td>iceA−</td>
<td>14 (22.6)</td>
<td>5 (25)</td>
</tr>
<tr>
<td>babA2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>57 (91.9)</td>
<td>17 (85)</td>
</tr>
<tr>
<td>Negative</td>
<td>5 (8.1)</td>
<td>3 (15)</td>
</tr>
<tr>
<td>cagA, cagE</td>
<td>53 (85.5)</td>
<td>18 (90)</td>
</tr>
</tbody>
</table>

\(^a\) Helicobacter pylori-infected gastritis patients.

\(^b\) Helicobacter pylori-infected gastric ulcer patients.

\(^c\) Helicobacter pylori-infected duodenal ulcer patients.

\(^d\) Helicobacter pylori-infected gastric cancer patients.

and cagA has been shown to be significantly associated with peptic ulcers.\(^1,10\) The cagA-positive vacA\(^c\)m1 and iceA1 have been shown to be predominant in Japan and Korea,\(^{16,20}\) while Qiao et al.\(^{40}\) reported that type s1 of vacA is more common than type s2, and m1 and m2 are equally represented in the Xi’an area of China.

The cagA gene has been shown to be present in about 60–70% of H. pylori strains isolated from Western populations including those of Europe and America.\(^6,9,10\) H. pylori cagA-positive strains have been reported to be associated with more severe H. pylori disease such as gastric mucosal atrophy and gastric cancer.\(^9,32,38\) In this study, cagA was found in 98.2% of H. pylori-infected dyspeptic patients. This prevalence is similar to that reported by others, who have shown cagA-positive H. pylori in more than 90% of cases in East Asian countries, and no association with severity of disease.\(^{37,39}\) There was no significant association between either the vacA subtype or cagA gene and severity of gastroduodenal diseases.\(^37,39\) However, recent studies have reported the importance of the diversity of CagA in relation to gastrointestinal diseases. They have indicated that the diversity of the tyrosine CagA phosphorylation occurs at the unique Glu–Pro–Ile–Ala (EPIYA) motifs present in the C-terminal region, affected by protein-tyrosine phosphatase (SHP-2), and actively involved in the regulation of the spreading, migration, and adhesion of cells. It may induce abnormal proliferation and movement of gastric epithelial cells and be associated with the mortality rate of gastric cancer in Asia.\(^{40}\) These findings should be further studied in H. pylori cagA-positive strains isolated from Thai dyspeptic patients in the future.

The cagE gene, also within the pathogenicity island and shown to stimulate production of several cytokines from infected epithelial cells, was found in 88.4% of H. pylori and 87.5% of cagA-positive H. pylori in this study. This result corresponds to those found in a previous report on children in the USA;\(^9\) however, we found that when cagA was positive, cagE was negative in 10.7% of samples (12/112), whereas only one sample was cagE-positive and cagA-negative.

The iceA gene may be associated with peptic ulcer disease;\(^9,18\) however, some studies have failed to confirm this correlation, and some groups have suggested a reverse relationship.\(^9\) There are two distinct allelic variants of iceA, namely iceA1 and iceA2.\(^7,19,37\) One study has suggested that iceA1 is associated with the development of peptic ulcers, and that iceA1-positive strains produce more of the proinflammatory factor IL-8 than iceA1-negative strains.\(^41\) Peek et al.\(^{18}\) demonstrated that iceA1 expression is significantly related to the host mucosal response, which led to the
hypothesis that the levels of transcription within the host environment may contribute to disease development. In contrast, iceA2 expression may be more influenced by gene structure, which has a repeated protein structure but it does not have homology with known proteins. Indeed, our H. pylori study of the iceA allele demonstrated that iceA1 (45.5%) was the most frequent genotype detected in our population. This finding agrees with previous reports that have shown the iceA1 allele more frequently found than the iceA2 allele in Chinese, Japanese, Korean and Dutch patients;7,19,34,37 iceA2 has been found to be predominant among Brazilian, European and American patients.9,19,42

The babyA2 gene has been shown to be associated with a higher risk of ulcer or adenocarcinoma development, and has been strongly associated with vacAs1 (79% babyA2-positive) and cagA genotypes (80% babyA2-positive) in German adults.21 H. pylori with triple positive genotypes, vacAs1, cagA and babyA2, has been reported in only 31% of dyspeptic children in Shanghai isolates from Korean A. Our results agree with Kim et al.6 Rudi J, Kolb C, Maiwald M, Kuck D, Sieg A, Galle PR, Hopkins RJ, Morris Jr JG.5 The vacAs1m1, cagA, cagE, iceA1, babyA2 genotypes in Thai dyspeptic patients 35

Table 4  Frequency distribution of combination genotypes of 112 H. pylori in the gastrointestinal dyspeptic patients

<table>
<thead>
<tr>
<th>Combination genotypes</th>
<th>Non-ulcer GTa (%) (N = 62)</th>
<th>Peptic ulcer</th>
<th>GCAb (%) (N = 16)</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>babyA2 positive</td>
<td>13 (21)</td>
<td>4 (20)</td>
<td>5 (35.7)</td>
<td>3 (18.8)</td>
</tr>
<tr>
<td>babyA2 positive</td>
<td>11 (17.7)</td>
<td>2 (10)</td>
<td>2 (14.3)</td>
<td>3 (18.8)</td>
</tr>
<tr>
<td>babyA2 positive</td>
<td>11 (17.7)</td>
<td>1 (5)</td>
<td>1 (7.1)</td>
<td>4 (25)</td>
</tr>
<tr>
<td>babyA2 positive</td>
<td>4 (6.5)</td>
<td>5 (25)</td>
<td>2 (14.3)</td>
<td>2 (12.5)</td>
</tr>
<tr>
<td>Others</td>
<td>23 (37.1)</td>
<td>8 (40)</td>
<td>4 (28.6)</td>
<td>4 (25)</td>
</tr>
<tr>
<td>Total</td>
<td>62 (100)</td>
<td>20 (100)</td>
<td>14 (100)</td>
<td>16 (100)</td>
</tr>
</tbody>
</table>

a Helicobacter pylori-infected gastritis patients. 

b Helicobacter pylori-infected gastric ulcer patients.

c Helicobacter pylori-infected duodenal ulcer patients.

d Helicobacter pylori-infected gastric cancer patients.

The development of gastric cancer, as was found in Japan and Korea.

In conclusion, this is the first report of the high prevalence of H. pylori virulence genes in Thai dyspeptic patients. There was no significant difference in any one specific bacterial gene and the gene pattern being associated with a particular clinical outcome. It is therefore necessary to define both the environmental and host factors in association with the bacterial characteristics for use in the prediction of the severity of disease.

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Conflict of interest: No conflict of interest to declare.

References


