To the editor:

**Fcγ receptor IIB gene polymorphism in adult Japanese patients with primary immune thrombocytopenia**

Several studies have indicated that platelet recovery occurs in a subgroup of immune thrombocytopenia (ITP) patients after successful *Helicobacter pylori* (*H pylori*) eradication.\(^1,2\) Interestingly, a higher response rate to *H pylori* eradication therapy has been reported in Japan and Italy than in the United States and European countries other than Italy,\(^2\) suggesting that the efficacy of *H pylori* eradication is influenced by ethnicity, probably through genetic and environmental factors. In addition, Asahi et al observed that monocytes from Japan and Italy than in the United States and European countries other than Italy,\(^2\) suggesting that the efficacy of *H pylori* eradication is influenced by ethnicity, probably through genetic and environmental factors. In addition, Asahi et al observed that monocytes from

To the editor:

**Fcγ receptor IIB gene polymorphism in adult Japanese patients with primary immune thrombocytopenia**

Several studies have indicated that platelet recovery occurs in a subgroup of immune thrombocytopenia (ITP) patients after successful *Helicobacter pylori* (*H pylori*) eradication.\(^1,2\) Interestingly, a higher response rate to *H pylori* eradication therapy has been reported in Japan and Italy than in the United States and European countries other than Italy,\(^2\) suggesting that the efficacy of *H pylori* eradication is influenced by ethnicity, probably through genetic and environmental factors. In addition, Asahi et al observed that monocytes from Japan and Italy than in the United States and European countries other than Italy,\(^2\) suggesting that the efficacy of *H pylori* eradication is influenced by ethnicity, probably through genetic and environmental factors. In addition, Asahi et al observed that monocytes from

**Table 1. Genotype distributions of the FcγRIIB 232I/T polymorphism**

| FcγRIIB 232I/T polymorphism | Healthy controls, n = 193 | Total ITP patients, n = 206 | P, vs healthy controls | H pylori–infected ITP patients, n = 100* | P, vs healthy controls | H pylori–uninfected ITP patients, n = 82* | P, vs healthy controls | H pylori eradication therapy† | Responders, n = 21 | Nonresponders, n = 21 | P
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>I/I genotype</td>
<td>134 (69.4)</td>
<td>113 (54.8)</td>
<td>.01‡</td>
<td>51 (51.0)</td>
<td>.01‡</td>
<td>47 (57.3)</td>
<td>.2</td>
<td>7 (33.3)</td>
<td>18 (85.7)</td>
<td>.01‡</td>
<td></td>
</tr>
<tr>
<td>I/T genotype</td>
<td>56 (29.0)</td>
<td>84 (40.8)</td>
<td></td>
<td>51 (44.0)</td>
<td></td>
<td>51 (60.0)</td>
<td>.002</td>
<td>12 (57.2)</td>
<td>3 (14.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T/T genotype</td>
<td>3 (1.6)</td>
<td>9 (4.4)</td>
<td></td>
<td>5 (5.0)</td>
<td></td>
<td>5 (5.0)</td>
<td>.2</td>
<td>2 (9.5)</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-T carriers§</td>
<td>134 (69.4)</td>
<td>113 (54.8)</td>
<td>.003</td>
<td>51 (51.0)</td>
<td>.002</td>
<td>47 (57.3)</td>
<td>.053</td>
<td>7 (33.3)</td>
<td>18 (85.7)</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>T carriers§</td>
<td>59 (30.6)</td>
<td>93 (45.2)</td>
<td>.35 (42.7)</td>
<td>14 (66.7)</td>
<td>.35 (42.7)</td>
<td>14 (66.7)</td>
<td>3 (14.3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Genotype distributions of the FcγRIIB 232I/T polymorphism in ITP patients and healthy controls, in *H pylori*–infected and –uninfected ITP patients, and in responders and nonresponders to *H pylori* eradication therapy. Genotype distributions were tested for statistical significance using the χ-square or Fisher exact test when 1 or more variables was <5.

*Of 182 patients with ITP evaluated for *H pylori* infection status, 100 were confirmed positive for *H pylori* infection based on a positive urea breath test and/or serum anti–*H pylori* antibodies measured by an enzyme-linked immunosorbent assay kit.

†Forty-two *H pylori*–infected ITP patients were administered amoxicillin (750 mg twice daily), clarithromycin (400 mg twice daily), and lansoprazole (30 mg twice daily) for 7 days. Twenty-one ITP patients were responders, defined as having a platelet count higher than 50 × 10⁹/L and doubling of the baseline level at 24 weeks after initiation of the eradication regimen.

‡The corrected *P* (*Pcorr*) values were calculated by multiplying the observed *P* value by the number of comparisons made. *Pcorr* = .03.

§FcγRIIB receptors encoded by the FcγRIIB 232I/T polymorphism are unable to interact with activating receptors and exert inhibitory activity.\(^6,7\) In addition, only few subjects were T/T genotype in this study. Therefore, we compared non-T carriers (I/I genotype) to T carriers (I/T + T/T genotype).


ITP patients (Table 1). ITP patients who were FcγRIIB 232T carriers contained significantly higher frequencies of responders than did noncarriers (66.7% vs 14.3%; \( P = .001; \text{OR} = 12.0; 95\% \text{CI, 2.62-54.99} \)). Thus, more efficient eradication therapy in 232T carriers may improve *H pylori* infection-related immunoregulatory systems, such as activating and inhibiting FcγR balance, thereby interrupting phagocytosis and antigen presentation.

In summary, our data suggest that FcγRIIB 232I/T polymorphisms may play an important role in susceptibility to *H pylori* infection-related immunoregulatory systems, such as activating and inhibiting FcγR balance,3 thereby interrupting phagocytosis and antigen presentation.

In summary, our data suggest that FcγRIIB 232I/T polymorphisms may play an important role in susceptibility to *H pylori* infection-related immunoregulatory systems, such as activating and inhibiting FcγR balance, thereby interrupting phagocytosis and antigen presentation.

The online version of this article contains a data supplement.

Acknowledgments: We thank Dr Toshio Okazaki for helpful discussions.

This work was supported by research grants from the Yokohama Foundation for Advancement of Medical Science 2011, the Kanagawa Nanbyou Study Foundation 2010, a Kitasato University Research Grant for Young Researchers 2011, the Kitasato University School of Allied Health Sciences (Grant-in-Aid for Research Project, No. 2011-1053), and the Kitasato University Graduate School of Medical Sciences (Integrative Research Program 2011).

Contribution: T.S., A.S., N.A., Y.O., and T.N. performed experiments; T.S., K.M., A.S., and M.K. analyzed the data; T.S., K.M., T.A., S.M., Y.K., Y.I., M.H., and M.K. collected patients and healthy control samples; K.M., Y.I., and M.H. collected the clinical data; S.T. contributed essential tools; M.K. helped revise the paper; and T.S. designed the research and wrote the paper.

Conflict-of-interest disclosure: The authors declare no competing financial interests.

Correspondence: Takashi Satoh, Division of Hematology, Kitasato University School of Allied Health Sciences, 1-15-1 Kitasato, Minami-ku, Sagamihara 252-0373, Japan; e-mail: takashis@kitasato-u.ac.jp.

References


Fcγ receptor IIb gene polymorphism in adult Japanese patients with primary immune thrombocytopenia

Takashi Satoh, Koji Miyazaki, Asako Shimohira, Naoki Amano, Yuka Okazaki, Tetsuya Nishimoto, Tohru Akahoshi, Shinichi Munekata, Yuhsaku Kanoh, Yasuo Ikeda, Masaaki Higashihara, Shinichiro Takahashi and Masataka Kuwana