**Correspondence**

**Effects of Lactobacillus gasseri OLL 2716 (LG21) on Helicobacter pylori infection in children**

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Sir,

Eradication therapies for *Helicobacter pylori* infection are not yet satisfactory, although many therapeutic strategies have been studied to improve the eradication rate for this infection. Children rarely have symptoms of this infection and, therefore, are a suitable group in which to assess therapies that are non-aggressive compared with those used for adults.1 The use of probiotics in the field of *H. pylori* infection has been proposed for improving eradication rates and to attenuate antibiotic-related complications.2 However, the scientific basis for the prophylactic and therapeutic actions of probiotics has yet to be established.

Michetti *et al.*3 reported that a whey-based culture supernatant of *Lactobacillus acidophilus* La1 had a partial, acid-independent, long-term suppressive effect on *H. pylori* in humans. Sakamoto *et al.*4 reported recently that *Lactobacillus gasseri* OLL 2716 (LG21) is effective in both suppressing *H. pylori* infection and reducing gastric mucosal inflammation in patients with *H. pylori* infection. There is, however, no study that examines the effects of probiotics in children with *H. pylori* infection, who are known to have a different gastric inflammation from that in adults and need non-aggressive therapies for *H. pylori* infection. Therefore, to investigate the effects of probiotics on *H. pylori* infection in children, we administered yogurt containing LG21 to 12 children with *H. pylori* infection, for 8 weeks, and then performed a [13C]urea breath test (UBT). We also measured the levels of faecal *H. pylori* antigens and serum pepsinogen I and II before, and at 4 and 10 weeks after, ingestion.

The subjects, five boys and seven girls, ranged in age from 7.4 to 15.8 years (mean age 12.1 years). All subjects were diagnosed with *H. pylori* infection based on positive results from a [13C]UBT and histological and culture examinations of the gastric mucosa. The study was approved by our institutional review board, and informed parental consent was obtained before all examinations. The subjects each received 120 g of yogurt containing LG21 twice daily for 8 weeks. Compliance with the ingestion of yogurt was monitored by parents using a diary. None of the subjects had undergone previous eradication therapy for *H. pylori* nor had any used antimicrobial drugs or proton-pump inhibitors during the previous month or during the study.

The [13C]UBT was performed by administering 2 mg/kg (maximum 100 mg) of [13C]urea before, and at 4 and 10 weeks after, the onset of yogurt ingestion for 8 weeks. Faecal *H. pylori* antigens were measured using the HpSA enzyme immunoassay (Premier Platinum HpSA; Meridian Diagnostics, Cincinnati, OH, USA), and serum pepsinogen I and II (PG I/II) determination was performed using a radioimmunoassay kit (Pepsinogen Riabead; Dainabot, Tokyo, Japan) following the manufacturer’s instructions.

There were no significant differences in the [13C]UBT values before, and at 4 and 10 weeks after, ingestion (Table 1). The HpSA level 4 weeks after the onset of ingestion was significantly lower than that before ingestion, although there were no significant differences in the HpSA values before and at 2 weeks after completing therapy. The PG I/II ratio 4 weeks after the onset of ingestion was significantly higher than that before ingestion, although no significant difference in this ratio was observed before and 2 weeks after the end of therapy.

A significant increase in the PG I/II ratio and a significant decrease in the HpSA level at 4 weeks after the onset of the LG21-containing yogurt diet compared with the pre-diet levels were noted. Because the value of the PG I/II ratio is considered to be inversely correlated with the level of inflammation in *H. pylori*-infected gastric mucosa, an increase in the PG I/II ratio might indicate suppression of *H. pylori*-associated gastric mucosal inflammation, which is in accordance with an adult study by Sakamoto *et al.*4 HpSA values do not provide quantitative assessments of the density of *H. pylori* colonization in the gastric mucosa, and we could not find a significant decrease in the [13C]UBT values after the ingestion of LG21 in the present study. Thus, further studies are needed to clarify the effect of LG21 on the density of *H. pylori* in gastric mucosa.

However, there were no significant differences in the values of [13C]UBT, HpSA and PG I/II before and at 2 weeks after the end of the 8-week yogurt diet, which is in contrast to...
the results of the adult study of LG21 administration (Sakamoto et al.\textsuperscript{4}), although the amount of LG21 ingestion was much greater in our study (240 g/day versus 180 g/day). There are two possible reasons for this discrepancy. First, as the immunopathogenesis of \textit{H. pylori}-associated gastritis and gastric inflammation in children is considered to be different from that in adults\textsuperscript{5} this may account for the different responses to LG21 administration. Secondly, we performed \textsuperscript{13}C$\text{UBT}$ and serum PG I/II 2 weeks after the end of therapy as opposed to the adult study, which measured these immediately after the end of the treatment. It is thus possible that LG21 can suppress \textit{H. pylori}-associated gastric inflammation only during active administration and that relapses may occur following cessation of therapy.

**References**


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**Table 1.** Results of tests for \textsuperscript{13}C$\text{UBT}$, HpSA and the serum PG I/II ratio in children with \textit{H. pylori} infection before, and at 4 and 10 weeks after, the onset of administration of LG21-containing yogurt, for 8 weeks (n = 12, mean ± S.D.)

<table>
<thead>
<tr>
<th>Test</th>
<th>Before</th>
<th>After 4 weeks</th>
<th>After 10 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>\textsuperscript{13}C$\text{UBT}$ (%)</td>
<td>28.1 ± 14.2</td>
<td>26.2 ± 13.1</td>
<td>30.2 ± 17.1</td>
</tr>
<tr>
<td>HpSA (OD)</td>
<td>0.62 ± 0.39</td>
<td>0.41 ± 0.28*</td>
<td>0.52 ± 0.48</td>
</tr>
<tr>
<td>PG I/II ratio</td>
<td>2.21 ± 0.98</td>
<td>2.59 ± 1.02*</td>
<td>2.36 ± 0.98</td>
</tr>
</tbody>
</table>

*P < 0.05 compared with before administration.

OD, optical density was measured by spectrophotometry (450 nm).