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## **Why is *Helicobacter pylori* disappearing? More questions than answers**

**Brendan Drumm,<sup>1</sup> Marion Rowland<sup>2</sup>**

After almost 30 years of research into *Helicobacter pylori*, we still are uncertain how infection is transmitted from person to person or more specifically from stomach to stomach. This is an important epidemiological question, as *H. pylori* is a fascinating enteric pathogen which is disappearing from many developed countries in the absence of any therapeutic or environmental strategy to reduce the prevalence of infection. The prevalence of *H. pylori* remains high in the developing world but its incidence is also decreasing in these countries.<sup>1</sup> The rapid decline in the prevalence of *H. pylori* infection in developed countries cannot be explained simply by the treatment of infection since the 1980s. The decline has been obvious since the early years of the 20th century, as evidenced by the unexplained decline in peptic ulcer disease and gastric cancer.<sup>2,3</sup> The reason for this decrease in one of the most common infectious diseases worldwide must be a change in some critical factor in the transmission pathway of *H. pylori* in humans.

There is no known animal or environmental reservoir of *H. pylori* infection. Epidemiology and molecular fingerprinting studies suggest that infection is spread from person to person during childhood. Apart from a few studies of transmission in primates, there is little evidence to date on the mechanism(s) of transmission.<sup>4,5</sup> Understanding transmission is essential if we are to develop strategies to limit or hinder spread of infection.

The study by Escobar-Pardo et al.<sup>6</sup> in this issue of *Jornal de Pediatria* is interesting and suggests that the prevalence of *H. pylori* remains high in children from the

Xingu Indian Reservation in Brazil. There has been only one long-term prospective study on the acquisition of *H. pylori* infection and this was in a developed country.<sup>7</sup> That study showed that most children who became infected with *H. pylori* were infected by the age of 3 years, and that new infection was uncommon after the age of 5 years.<sup>7</sup> These

findings were confirmed by the study in the Xingu Reservation, where the prevalence of infection was the same in the 4-5 year age group as in the 8-9 year age group.<sup>6</sup> The fact that *H. pylori* is acquired at a very young age is also supported by the finding that, following treatment, reinfection does not occur in older children, but younger

children may be at risk of reinfection when treated before the age of 5 years.<sup>8</sup>

*H. pylori*, in common with most other enteric infections, is acquired in the pediatric age group,<sup>7</sup> and we know that *H. pylori* is clustered in families.<sup>9</sup> However, unlike many other gastrointestinal infections which are transmitted by the fecal-oral route, new infection with *H. pylori* is uncommon in older children and adults, suggesting that fecal-oral transmission is unlikely. In addition, epidemics or outbreaks of infection even in institutions have not been documented. While *H. pylori* can survive in water, there is no evidence that water provides a reservoir for infection, and studies of Rhesus macaques confirm that even with a clean water supply *H. pylori* is acquired at a very young age.<sup>4,5</sup>

There is good evidence from the work of Parsonnet et al. that transmission in emesis is much more likely than fecal-oral transmission.<sup>10</sup> Those authors induced both vomiting and diarrhea in *H. pylori*-infected healthy volunteers. While

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*H. pylori* was cultured easily from all samples of emesis of infected individuals, it was found in only 21% of fecal samples. Recently, Perry et al. have shown that 75% of new *H. pylori* infections in children were associated with exposure to an *H. pylori*-infected person with gastroenteritis, and where vomiting was the predominant symptom of the gastroenteritis, the risk of new *H. pylori* infections was markedly increased in young children.<sup>11</sup> Similarly, studies on Rhesus macaques found large concentrations of *H. pylori* in emesis, but no *H. pylori* was isolated from their feces. Furthermore, when fecal samples from *H. pylori* infected animals were gavaged into uninfected macaques, there was no evidence of transmission of infection.<sup>5</sup>

What then is responsible for the rapid decline in the prevalence of *H. pylori* worldwide? What factors in early childhood, most likely in the infant and toddler age group, have changed in the last 100 years to cause such a decline in what was once the most common bacterial infection of humans? This decline has occurred on all continents and in all socioeconomic groups.<sup>1</sup> The indigenous population studied by Escobar-Pardo et al. may provide an exciting opportunity to determine the reason for the decline in the incidence of *H. pylori*, particularly in developing countries.

In the study, the prevalence of infection in 2-3 year old children was reported to be 60%; this prevalence increased to 81% in those over 5 years of age. The numbers found in the study are too small to confirm that this indicates a decline in the incidence of *H. pylori* in this population. However, our studies indicating that infection or reinfection after the age of 3-4 is very unlikely<sup>7,8</sup> suggest that the findings of Escobar-Pardo et al. could indicate a decreasing incidence of infection in this indigenous population.<sup>6</sup>

It is therefore hoped that the authors of this study will have an opportunity to prospectively follow this 2-3 year old group of children for the next number of years. If the prevalence of infection remains around 60% over subsequent years, it will confirm a decline in the incidence of *H. pylori* and also that new *H. pylori* infections are uncommon after the age of 3 years, even in a developing country. The challenge then is to identify the factors which explain this decline in the incidence of *H. pylori*.

The differences in the prevalence of infection in the different villages are a very interesting feature of the study. The Ikpeng ethnic group had a much lower prevalence of *H. pylori* than the other ethnic groups assessed. An in-depth examination of the factors responsible for the different rates of infection among children from the different tribes and/or villages would be very worthwhile. Since 2003, the provision of wells with solar-power panels to pump water to individual households in these indigenous communities may provide new insights into the transmission of *H. pylori*.

The provision of a good supply of clean water may in some way interrupt the transmission of *H. pylori* by routes such as emesis or saliva.

Alternatively, differences in prevalence between the different tribal groups could be related to host/genetic factors, environmental factors, or child rearing practices. In the context of evidence supporting gastric-oral or oral-oral transmission, pre-mastication of food is of major interest. It is important to determine the exact mechanism of transmission for *H. pylori*, and further studies from this population may prove very important in elucidating the transmission pathways of *H. pylori*.

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