

Helicobacter pylori infection in Indigenous Australians: a serious health issue?

If Indigenous Australians are at high risk of H. pylori infection, the associated risks of peptic ulceration and gastric cancer may make screening and treatment necessary

Interest in asymptomatic *Helicobacter pylori* infection seems largely to have waned in much of the developed world.¹⁻³ It is almost universally accepted that *H. pylori* causes chronic gastritis and peptic ulcer disease,¹⁻³ and the evidence that the infection also causes gastric adenocarcinoma is virtually unassailable.¹⁻⁴ In high-risk populations, at least, prevention of gastric cancer by *H. pylori* eradication is theoretically feasible, although convincing clinical-trial evidence is lacking.²⁻⁵ However, perhaps because the incidence of gastric cancer is steadily declining in developed nations (and because *H. pylori* infection seems to be much less often acquired these days), interest in its prevention remains remarkably low key.^{1,2}

Australian Aboriginals and Torres Strait Islanders are much more likely to suffer ill health and die at a younger age than non-Indigenous Australians.⁶ However, one health scourge that the Indigenous population seemed to have missed, despite their lower socioeconomic status, was *H. pylori* infection and its complications.⁷ For example, seroprevalence studies suggested *H. pylori* infection was very rare.⁷ The report by Windsor and colleagues in this issue of the Journal (page 210) is at variance with these earlier findings,⁸ and strongly suggests that *H. pylori* infection is as common in Australia's Indigenous population as it is in developing countries in South America, Africa and parts of Asia.³ If confirmed, these findings may have major health implications; indeed, the high prevalence identified in children⁸ implies a public health risk which will continue long into the future unless this situation is rectified.

Notably, the findings of Windsor et al are limited to a small and possibly unique Indigenous group, and may not be more widely

generalisable — more data are needed. However, the fact that over half the population in the remote rural communities studied were tested gives some confidence that *H. pylori* is remarkably widespread in this community (where 91% were found to be infected by means of a “gold standard” test). On the other hand, in urban Perth, referral bias can not be ruled out as accounting for the high *H. pylori* prevalence (60%), although the burden of infection remains strikingly high — if bias is the sole explanation, it would have to be particularly pervasive.

What are the potential implications of these findings? Gastric adenocarcinoma remains a major health problem in the developing world.² In the developed world, the consequences of *H. pylori* infection have arguably been underappreciated. In the United Kingdom, it has been calculated on the basis of relatively conservative assumptions that by age 85, and allowing for competing causes of mortality, the cumulative risk of dying from gastric cancer arising because of *H. pylori* infection is a staggering 1 in 51 for men and 1 in 96 for women.⁹ Data from the Northern Territory suggest that the rate of mortality from gastric cancer among Indigenous people has not increased.¹⁰ However, gastric cancer may become a much greater burden for the Indigenous population if, as is hoped, life expectancy improves from the current unacceptable median age at death of just 51 years.⁶

Older studies have suggested that peptic ulcer is uncommon in the Indigenous population,⁸ but, if Indigenous people undergo fewer endoscopies, this impression might be driven purely by referral bias. It has been calculated that, in the UK population, 1 in 154 men and 1 in 173 women die from peptic ulcer caused by *H. pylori*.⁹ The ulcer rates may, however, be truly

low in Indigenous Australians, as undernutrition and lower socioeconomic status are associated with lower gastric acid secretion. This allows *H. pylori* to spread throughout the stomach (causing pan-gastritis and increasing the risk of cancer if people live long enough), but protects against ulcer disease.^{2,3}

Circulatory disease, particularly ischaemic heart disease, is a major cause of mortality in the Indigenous population.^{6,10} Is *H. pylori* an important and modifiable risk factor for ischaemic heart disease? Unfortunately, the evidence for this is mixed and, at best, equivocal.¹¹ Overall, widespread eradication of *H. pylori* to try to reduce ischaemic heart disease in this population is not currently justifiable.

Other health problems in the Indigenous population just might be attributable to *H. pylori*, at least in part. Indigenous Australians have been reported to be more likely to give birth to infants who are small for gestational age,¹² and *H. pylori* infection has been associated with growth restriction in young children.¹³ A Sydney study also found (among 448 pregnant women attending for routine examinations in the third trimester) that intrauterine growth restriction was twice as common in *H. pylori*-seropositive women than in seronegative women.¹⁴ This association was not explained by smoking or maternal height, although these were also independent risk factors for intrauterine growth restriction.¹⁴ While the data clearly need confirmation, the general health risks of a high carriage rate of *H. pylori* in Indigenous people may have been underestimated and may be another antenatal risk factor.

Might *H. pylori* offer protection from some other diseases, and, if so, might attempts at its widespread eradication lead to more ill health in the Indigenous population? Such concerns have been raised,¹⁵ but seem overexaggerated and unlikely. In particular, recent data suggest that *H. pylori* eradication in peptic ulcer disease neither provokes reflux oesophagitis nor aggravates symptoms of gastroesophageal reflux.¹⁶ We need large, population-based intervention trials to define any risks, but whether it is wise to wait for such results before actively intervening remains debatable.

H. pylori may be very common in Indigenous Australians, particularly among those living in remote parts of Australia. Future research needs to focus on the prevalence of *H. pylori* (and strains of different virulence) and the burden of dyspepsia in these populations. Although the exact route of transmission of *H. pylori* remains controversial, close contact within families is probably at least one key risk factor, and faecal-oral spread is feasible.^{2,3} Thus, strong consideration needs to be given to trying to reduce the acquisition of the infection in Indigenous children by reducing family crowding, and providing adequate sanitation and clean water. Doctors need to be aware that there may be a high risk of *H. pylori* infection in subpopulations within Australia. They should consider testing and treating Indigenous patients who present with dyspepsia (or non-ulcer dyspepsia), have a history of peptic ulcer complications, or have a family history of gastric cancer.^{2,3,17} However, to reduce the

disease burden of *H. pylori* in the next 50 years, population screening and treatment may be the only solution. Assuming the data of Windsor et al are generalisable, a randomised controlled trial to test the health benefits (and risks) of population-based screening and antibiotic treatment in Indigenous Australians should be a government research-funding priority.

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- 1 Talley NJ. *Helicobacter pylori* management: how to improve the therapeutic confusion in practice. *Can J Gastroenterol* 2003; 17 Suppl B: 21B-24B.
- 2 Malfertheiner P, Megraud F, O'Morain C, et al. Current concepts in the management of *Helicobacter pylori* infection — the Maastricht 2-2000 consensus Report. *Aliment Pharmacol Ther* 2002; 16: 167-180.
- 3 Lam SK, Talley NJ. Report of the 1997 Asia Pacific Consensus Conference on the management of *Helicobacter pylori* infection. *J Gastroenterol Hepatol* 1998; 13: 1-12.
- 4 Uemura N, Okamoto S, Yamamoto S, et al. *Helicobacter pylori* and the development of gastric cancer. *N Engl J Med* 2001; 345: 784-789.
- 5 Wong BC, Lam SK, Wong WM, et al. *Helicobacter pylori* eradication to prevent gastric cancer in a high-risk region of China: a randomized controlled trial. *JAMA* 2004; 291: 187-194.
- 6 Ring IT, Brown N. Indigenous health: chronically inadequate responses to damning statistics. *Med J Aust* 2002; 177: 629-631.
- 7 Dwyer B, Sun NX, Kaldor J, et al. Antibody response to *Campylobacter pylori* in an ethnic group lacking peptic ulceration. *Scan J Infect Dis* 1988; 20: 63-68.
- 8 Windsor HM, Abioye-Kuteyi EA, Leber JM, et al. Prevalence of *Helicobacter pylori* in Indigenous Western Australians: comparison between urban and remote rural populations. *Med J Aust* 2005; 182: 210-213.
- 9 Axon A, Forman D. *Helicobacter* gastroduodenitis: a serious infectious disease. *BMJ* 1997; 314: 1430-1431.
- 10 Condon JR, Barnes T, Cunningham J, Armstrong BK. Long-term trends in cancer mortality for Indigenous Australians in the Northern Territory. *Med J Aust* 2004; 180: 504-507.
- 11 Pellicano R, Fagoonee S, Rizzetto M, Ponzetto A. *Helicobacter pylori* and coronary heart disease: which directions for future studies? *Crit Rev Microbiol* 2003; 29: 351-359.
- 12 Roberts CL, Lancaster PA. Australian national birthweight percentiles by gestational age. *Med J Aust* 1999; 170: 114-118.
- 13 Bravo LE, Mera R, Reina JC, et al. Impact of *Helicobacter pylori* infection on growth of children: a prospective cohort study. *J Pediatr Gastroenterol Nutr* 2003; 37: 614-619.
- 14 Eslick GD, Yan P, Xia HH, et al. Foetal intrauterine growth restrictions with *Helicobacter pylori* infection. *Aliment Pharmacol Ther* 2002; 16: 1677-1682.
- 15 Blaser MJ. Hypothesis: the changing relationships of *Helicobacter pylori* and humans: implications for health and disease. *J Infect Dis* 1999; 179: 1523-1530.
- 16 Raghunath A, Hungin AP, Wooff D, Childs S. Systematic review: the effect of *Helicobacter pylori* and its eradication on gastro-oesophageal reflux disease in patients with duodenal ulcers or reflux oesophagitis. *Aliment Pharmacol Ther* 2004; 20: 733-744.
- 17 Moayyedi P, Deeks J, Talley NJ, et al. An update of the Cochrane systematic review of *Helicobacter pylori* eradication therapy in nonulcer dyspepsia: resolving the discrepancy between systematic reviews. *Am J Gastroenterol* 2003; 98: 2621-2626. □