Casting a long shadow: Infection drives stomach cancer inequalities in Māori and Pacific peoples

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In this Blog we discuss our recently published study that shows that infection from the bacteria *Helicobacter pylori* is the major driver of stomach cancer inequalities borne by Māori and Pacific peoples in NZ. We also discuss a possible next step which could be for one DHB to pilot a 'test and treat' screening programme that seems likely to help reduce such inequalities.

Māori and Pacific people have 3 to 6 times the rates of stomach cancer as NZ Europeans. These are some of the starkest relative inequalities between ethnic groups in New Zealand (1). This disparity is particularly concerning given poor stomach cancer survival. In 2013 the Ministry of Health reported that 80 Māori and 35 Pacific people died from stomach cancer, out of a total of 371 stomach cancer deaths. Although stomach cancer rates are declining here and around the world, we estimate that there would be up to 85 fewer deaths every year (61 Māori, 24 Pacific) if stomach cancer occurred in Māori and Pacific people at the same incidence as in European/Other. The majority of these deaths are probably preventable. While *H. pylori* is known to cause much of stomach cancer around the world, there has been speculation in NZ that high Māori and Pacific rates are not largely explained by differing rates of *H. pylori*. Our findings published in the journal *Gastric Cancer* this week underline *H. pylori* as the major driver of ethnic inequalities in stomach cancer (2) (http://link.springer.com/article/10.1007/s10120-016-0671-8/fulltext.html). This new study has substantial implications for how we might address excess stomach cancer deaths by ethnicity and how we address stomach cancer risk for all New Zealanders.

There has been longstanding interest in reducing the high rates of stomach cancer in Māori (3). In the early 1980s *H. pylori* was identified (4) and subsequently recognised as a driver of ethnic differences (5). However, much attention has been given to a genetic risk of stomach cancer in some Māori whanau (6), with some commentators suggesting that the excess risk of stomach cancer may be largely due to this genetic mutation (7). In fact, the E-cadherin mutation does not explain many of the excess deaths in Māori and Pacific. Our new study clearly places *H. pylori* as the major driver of stomach cancer inequalities (2).

H. pylori is an infection that is normally acquired in childhood and passed down through families. It's prevalence is associated with poverty and household crowding (8) and may be linked to these risk factors over generations; marking this infection with a historical legacy

of poverty, crowding and colonisation. With today's rise in homelessness and increasing rates of household crowding (9), particularly for the least well off, problems with housing affordability and child poverty have marked implications and can cast a very long shadow. Preventing children getting infected with *H. pylori* may be achievable through interventions to reduce household crowding and child poverty. Acquisition of *H. pylori* has declined in younger generations (10) but it will take decades to eradicate *H. pylori* at the current rate of decline.

Adults with chronic *H. pylori* infection are likely to be asymptomatic. Infection with *H. pylori* is treatable with triple therapy (two antibiotics and a proton pump inhibitor for 7-14 days) but this is not current practice for asymptomatic individuals. Several randomised controlled trials (RCT) have shown that testing and treating asymptomatic people is successful in reducing stomach cancer by about a third, although there is a wide range of uncertainty. The evidence is also unclear about the potential harms from screening, and this question needs further investigation, for example impacts on antibiotic resistance, and whether there is increased risk of any other complications.

The WHO and IARC Working Group (11) recommends that all countries consider including gastric cancer in their national cancer control programmes and that they conduct detailed assessments of its current and future human and economic impacts and of the potential value of prevention strategies. A cost-effectiveness analysis we have undertaken (under review with a journal) suggests that a targeted *H. pylori* test and treat screening programme is likely to be cost effective given current NZ settings particularly for Māori and Pacific adults (12). A watchful brief should therefore be placed on RCT evidence emerging from Europe, Asia and elsewhere that is evaluating the harms and benefits of *H. pylori* test and treat screening strategies. A screening feasibility pilot could be trialled in one DHB, for example to identify the highest risk groups (by age, sex and ethnicity) and the optimal test and treat strategy. Further information is also needed about the changing incidence of *H. pylori*, which groups are at highest risk of future stomach cancer, any changes in treatment success rates, and to identify the age that a test and treat strategy would be most beneficial.

In summary, we consider that stomach cancer inequalities should be better addressed in New Zealand. It may be time for health authorities to start considering piloting a *H. pylori* test and treat screening programme eg, in one DHB with relatively high proportions of Māori and Pacific peoples.

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