

The burden of secondary antibiotic resistance in *Helicobacter pylori* in Auckland, Aotearoa New Zealand

Cameron Schauer, Marius van Rijnsoever, Susan Taylor, Michael TM Wang, Russell Walmsley, Jonathan Koea, Stephen Inns, Tom Hills

ABSTRACT

AIM: *Helicobacter pylori* (*H. pylori*) infection is the principle modifiable risk factor for gastric cancer and a key driver of ethnic disparities in gastric disease within Aotearoa New Zealand. Increasing antibiotic resistance threatens eradication success, yet secondary resistance patterns have not previously been described in New Zealand. This study aimed to describe, for the first time, secondary *H. pylori* antibiotic resistance rates in New Zealand and identify demographic predictors to inform eradication strategies.

METHODS: A retrospective review was conducted of *H. pylori* isolates referred for antibiotic susceptibility testing in Auckland between January 2018 and December 2023. Testing was performed at Middlemore Hospital Laboratory using gradient diffusion minimum inhibitory concentration (MIC) strips for amoxicillin, clarithromycin, metronidazole and tetracycline. Demographic data were obtained from the Auckland TestSafe database and analysed using univariate and multivariable logistic regression.

RESULTS: Of 3,234 patients tested, 644 (20%) were culture positive. Resistance rates were clarithromycin 68%, metronidazole 68.5%, amoxicillin 2.5% and tetracycline 0%. Male sex was associated with lower odds of metronidazole resistance ($p < 0.001$). Māori and Pacific peoples had lower odds of both metronidazole and clarithromycin resistance compared with Europeans ($p < 0.05$), while Asian ethnicity was associated with higher clarithromycin resistance ($p = 0.02$).

CONCLUSION: This first-in-New Zealand description of secondary *H. pylori* resistance shows high rates of clarithromycin and metronidazole resistance. These findings lend support to use of non-clarithromycin-based salvage regimens. Future prospective studies of secondary and also, most importantly, primary resistance data are essential to inform testing and treatment guidelines and improve eradication outcomes.

Helicobacter pylori (*H. pylori*) remains one of the most prevalent chronic bacterial infections in the world. It colonises gastric mucosa, and while most infections remain asymptomatic, it is well established as the primary cause of a number of diseases ranging from simple gastritis to gastroduodenal ulceration and gastric adenocarcinoma.^{1,2}

Both gastric ulceration and gastric cancer mortality in Aotearoa New Zealand show marked ethnic disparities. Hospitalisation due to peptic ulcers are three to four times higher for Māori and Pacific peoples respectively, compared to solely European individuals.³ Similarly, the incidence of gastric cancer for these ethnicities is three times that of non-Māori.^{4,5} *H. pylori* is identified as the principle modifiable risk factor and the single largest driver of these inequities.⁶ Infection is estimated to account for over half of the excess gastric cancer incidence in Māori men and more than two-thirds in Pacific men.⁶

Testing within New Zealand has remained a topic of interest, with evidence in both the

North⁷ and South Island^{8,9} of increased testing and positivity rates. Despite the aforementioned inequity of disease and two to three times higher seroprevalence,¹⁰ there are new concerning data demonstrating lower testing rates in Māori (odds ratio [OR] 0.69) and Pacific peoples (OR 0.81) compared to people of European ethnicity.⁷

Internationally, increasingly robust evidence has established *H. pylori* eradication as a strategy for prevention of gastric cancer in high-risk populations, with mounting pressure for countries to explore the possibility of introducing population-based *H. pylori* screen-and-treat programmes.¹¹ This has been estimated to be a cost-effective strategy when targeted to those who are high risk in New Zealand.¹² We have recently laid out a framework and action plan for the implementation of such a programme in New Zealand.¹³ However, the effectiveness of eradication regimens has been increasingly threatened by rising antibiotic resistance, which is also characterised by vast ethnic and socio-economic disparity, in part driven by antibiotic overuse.¹⁴

The mainstays of *H. pylori* eradication therapy are commonly used antibiotics: amoxicillin, clarithromycin, metronidazole and tetracycline. A recently published meta-analysis examining antibiotic resistance of *H. pylori* in Australia and New Zealand demonstrated an increase in primary clarithromycin resistance with stable or low resistance to other commonly used antibiotics.¹⁵ However, there were only 15 total studies, including just three from New Zealand, published between 1996 and 2013.^{16–18} These studies examined primary resistance only, defined as being present before any *H. pylori* targeted therapy is administered. Local work to prospectively update primary resistance estimates in our symptomatic population is ongoing.¹⁹ Five studies have reported secondary resistance rates from Australia,¹⁵ defined as resistance detected after failure of one or more eradication attempts. No descriptions of secondary resistance in *H. pylori* have ever been reported from New Zealand.

This investigation aims to describe results of *H. pylori* resistance testing to inform our understanding of the rates of secondary resistance, which may improve understanding of local resistance patterns and guide eradication regimens to optimise eradication rates.

Methods

Currently, in Auckland New Zealand, *H. pylori* antibiotic resistance testing is only suggested if two rounds of empirical antibiotic treatment, which would typically include a total of five different antimicrobial agents over at least 24 days, is unsuccessful.²⁰ We performed a retrospective review of these samples sent for resistance testing between January 2018 and December 2023.

Recommended protocols in Auckland for *H. pylori* treatment are as follows:²⁰

- omeprazole, 20mg twice daily; and
- clarithromycin, 500mg twice daily; and
- amoxicillin, 1,000mg twice daily; or metronidazole, 400mg twice daily for 14 days if penicillin allergic.

If this is not successful, the second-line treatment suggested is:

- omeprazole, 20mg twice daily; and
- tripotassium dicitratobismuthate (bismuth), 120mg four times daily; and
- tetracycline, 500mg four times daily; and
- metronidazole, 400mg three times daily.

If unsuccessful after two attempts at eradication, the chance of achieving that goal is low. The next step suggested is to request non-acute adult gastroenterology assessment for gastroscopy.²¹

At gastroscopy, culture samples were placed in saline solution and sent to a single laboratory unit in Auckland, the IANZ-accredited Middlemore Hospital Laboratory, for processing.

Culture sample processing during the study period was as follows: samples were macerated using a sterile scalpel blade and inoculated onto two *Brucella* agar plates of 5% sheep blood (Fort Richard Laboratories). The inoculated plates were then incubated at 37 degrees Celsius in microaerophilic conditions using a CampyGen generator (Oxoid) and examined for typical colonies for up to 10 days.

Susceptibility testing was performed on any identified curved gram-negative bacilli that was both oxidase and urease positive. A suspension of organism was prepared in saline to a two McFarland standard density. This suspension was then used to inoculate Mueller–Hinton 5% sheep blood agar plates (Fort Richard Laboratories). One gradient diffusion minimum inhibitory concentration (MIC) strip was applied per plate and incubated at 37 degrees Celsius for 72 hours in microaerophilic conditions before reading the MIC.²² Standardised MICs of amoxicillin, clarithromycin, tetracycline and metronidazole were performed using gradient diffusion MIC strips to report either susceptibility or resistance.

Results from these cultures only were then extracted between January 2018 and December 2023 from the Auckland TestSafe database, using Qlik Data Integration software (Pennsylvania, United States of America). This provided a full set of tests completed in the Auckland Region (hospital, community and private institutions). Data were linked with available demographic variables including prioritised ethnicity, which were as recorded in the clinical records (Regional Clinical Portal, Orion Health, New Zealand) at the time of extraction. To ensure completeness, this dataset was separately and independently cross referenced to clinical records kept by the Middlemore Hospital Laboratory team. Any discrepancies were reviewed by two investigators (CS and MVR).

This study was approved by the Waitemata Research & Knowledge Centre (reference: WAI20005), and as part of our wider work on management of *H. pylori* (New Zealand Health and Disability Ethics Committee reference: 2024 EXP 21063). This was supported by the Health New Zealand – Te Whatu Ora Waitemata District

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Statistics

Statistical analysis was performed using IBM SPSS Statistics version 30.0 (New York, United States of America). Univariate inter-group comparisons were performed using the unpaired *t*-test, where normal distribution had been confirmed by Shapiro–Wilk testing ($p > 0.05$). Non-normally distributed data were analysed using the Mann–Whitney U test, and categorical data were analysed using the Chi-squared test or Fisher’s exact test. Multivariable logistic regression was performed to assess for predictors of antibiotic resistance, incorporating all demographics and variables. All tests were two-tailed, with $p < 0.05$ considered statistically significant.

Results

A total of 3,234 patients (mean±SD age, 51.1±15.1 years; 1,397 males and 1,837 females) had *H. pylori* culture testing between January 2018 and December 2023, of which there were 644 culture-positive cases. Demographics and results of patients with culture-positive *H. pylori* infection are summarised in Table 1.

Rates of resistance to clarithromycin was 68%, metronidazole 68.5%, amoxicillin 2.5% and tetracycline 0%. Antibiotic resistance by year of testing is presented in Table 2.

Demographic predictors of metronidazole and clarithromycin resistance are presented in Table 3. Multivariable logistic regression demonstrated that male sex was associated with lower odds of metronidazole resistance ($p < 0.001$). Patients of Māori and Pacific ethnicity had reduced odds of both metronidazole and clarithromycin resistance compared with those of European ethnicity ($p < 0.05$), while Asian ethnicity was associated with higher rates of clarithromycin resistance than European ethnicity ($p = 0.02$).

Discussion

This study provides the first description of *H. pylori* secondary antibiotic resistance rates in New Zealand, offering some insight into local treatment challenges.

These data represent the largest number of *H. pylori* secondary resistance isolates tested in any Australasian study to date, close to the combined total ($n = 710$) from the five previously

published studies in the region (one from 2006 and four between 2012 and 2018).¹⁵ Compared to these earlier data, our prevalence estimates for secondary resistance are broadly similar for metronidazole (69% vs 68%), amoxicillin (2.5% vs 3%) and tetracycline (0% vs 0.5%). Our secondary clarithromycin resistance rate (68%) was somewhat lower than the pooled Australian estimate (79%). However, this difference is likely influenced by a single Western Australian study contributing 310 isolates between 2007 and 2011, which reported a strikingly high clarithromycin resistance rate of 94%.²³

In a global context, our secondary clarithromycin resistance rate is lower compared to China, which has a pooled secondary resistance to clarithromycin of 77% (with primary resistance reported as 37%).²⁴ However, there is considerable geographical variability, with the highest rates reported from Lanzhou being 94%.²⁵ However, our rates are substantially higher than estimates from an analysis of the World Health Organization regions between January 2007 and June 2017, in which secondary clarithromycin resistance was noted to be 18% in the Americas, 17% in the Eastern Mediterranean, 48% in Europe and 15% in Southeast Asia.²⁶ In this study, the Western Pacific Region rate was noted to be 67%. A meta-analysis between 2000 and 2023 of global rates of secondary clarithromycin resistance among children was 69%.²⁷

Many factors may be associated with these considerable levels of secondary resistance to clarithromycin. Certainly, our estimates may be inflated due to Auckland’s testing protocol requiring failure of two eradication regimens prior to culture and susceptibility testing (as opposed to at least one per the accepted definition), with patients almost certainly having received clarithromycin as part of this treatment. Clarithromycin resistance has been found to be the single most important predictor of eradication failure with standard triple therapy,²⁸ which is also seemingly confirmed by these results in our study population.

The last published study from Auckland (2012), which included 73 newly diagnosed patients from South Auckland, reported a 16% primary clarithromycin resistance rate. A recent study from Wellington detecting clarithromycin resistance genes using droplet digital polymerase chain reaction (PCR) in 84 patients also suggested a rate of 16%.²⁹ Since then, resistance rates may have risen in part due to New Zealand’s high macrolide consumption, which is known to correlate with *H. pylori* treatment failure when clarithromycin

is used.^{30,31} Importantly, concerns about rising macrolide resistance were raised even before azithromycin funding was broadened in New Zealand in 2012.³²

In our cohort, metronidazole resistance was similarly high at 69%. This is as compared to 30% in the Americas, 65% in the Eastern Mediterranean, 48% in Europe and 44% in Southeast Asia.²⁶ It is thought these high rates may also be related to metronidazole use in the treatment of parasitic infections,³³ although unlikely to be a factor in our cohort given low rates of parasitic infection in New Zealand.

In contrast, the absence of tetracycline resistance is reassuring, consistent with global trends and supports its continued inclusion in quadruple salvage therapy.²⁶ Similarly, the persistently low rates of amoxicillin resistance remain encouraging. Future studies should also include resistance data for fluoroquinolones, which are currently only partially reported. As the prevalence of multidrug-resistant organisms increases, the availability of alternative therapies such as levofloxacin—which at present is limited and self-funded—will become increasingly important.

We also observed demographic differences in resistance patterns. Māori and Pacific patients had lower odds of clarithromycin and metronidazole resistance compared with European patients. This contrasts with previous reports of higher primary clarithromycin resistance in these groups.¹⁸ These findings should be interpreted cautiously, given the low overall rates of testing and positivity among Māori and Pacific peoples. A recent publication similarly reported that Māori and Pacific peoples had the lowest likelihood of retesting (OR 0.57 and 0.47, respectively). In contrast, retesting was most common in Asian patients (39%), consistent with our study in which Asian patients also had the highest number of positive cultures. The significantly higher rates of clarithromycin resistance among Asian patients compared with Europeans ($p=0.02$) may reflect differing prior antibiotic exposures, both within New Zealand and through overseas birthplace, residence or travel.

The overall success rates of eradication therapy in New Zealand are unknown, as routine retesting is not recommended in the Auckland Region unless symptoms persist. Data from the Northern Region indicate that only 34% of patients with an initial positive stool test underwent retesting, of whom 30% remained positive.⁷ Access to culture-based susceptibility testing in Auckland

clearly requires persistence with repeated testing and engagement, further limiting uptake. These challenges are compounded by well-documented inequities in access to primary care for Māori and Pacific peoples,³⁴ raising particular concern that these populations with the highest burden of gastric cancer are also the least likely to be retested or referred for susceptibility testing.¹³

It is noteworthy that most patients undergoing culture testing in our cohort had negative results (80%). We are currently studying success rates of *H. pylori* culture before first-line treatment at Waitemata using culture testing.¹⁹ Culture-based testing is hampered by practical challenges. *H. pylori* is a fastidious organism that requires up to 10 days of incubation, is highly sensitive to transport conditions³⁵ (often reliant on taxi transport to the laboratory), and yields are further reduced in the context of partial treatment or concurrent proton pump inhibitor use or low bacterial loads.^{36,37} These factors, alongside the high costs, limit the feasibility of culture as the sole diagnostic tool. Endoscopic biopsy sampling patterns were also not standardised, which may have contributed to reduced culture yield.³⁸

For these reasons, we are currently evaluating molecular methods,¹⁹ such as PCR assays, which identify specific resistance-conferring mutations in *H. pylori* DNA.³⁹ These tests are rapid, highly sensitive and comparatively inexpensive.⁴⁰ They are already in clinical use overseas, for example in Northeast China, where secondary resistance to clarithromycin (83%) and levofloxacin (70%) is high. In that setting, PCR-guided therapy significantly outperformed empirical treatment.⁴¹ In Wellington, New Zealand, eradication success was significantly lower in patients with clarithromycin resistance genes (38.5%) compared to those without (97.2%, $p<0.001$).²⁹ Importantly, PCR can also be performed on stool samples, avoiding the need for gastroscopy and biopsy.

An important limitation of this retrospective dataset is the inability to capture detailed prior antibiotic exposure histories and adherence, and demographic details including place of birth and immigration status, which may have aided interpretation of resistance mechanisms. Incomplete ethnicity recording (27%) may also reflect differential engagement with the health system. Further data are required on these important aspects, and we are now actively consenting patients for enrolment in the Worldwide Registry on the management of *H. pylori*,⁴³ which allows for collection of these factors.

We wish to re-emphasise that our cohort reflects patients referred for endoscopy and culture, having failed multiple therapies. This selects for more complex, refractory cases and doesn't reflect the broader *H. pylori* population. These rates may be further enriched as re-testing is infrequently performed (30%), with Māori and Pacific peoples half as likely to be retested as solely European individuals.⁷ Because repeat testing in these groups is so infrequent, these resistance data may disproportionately reflect patients more engaged with the healthcare system, potentially under-representing marginalised groups.

We were also only able to analyse completed clinician test requests. Therefore, the proportion of incomplete or unperformed tests remains unknown. While it is possible that some testing included in our study was performed to assess for primary resistance, this is unlikely a large effect given the strict criteria for gastroscopy access. Furthermore, data from the Northern Region indicate that 84% of the over 8,000 patients reviewed with *H. pylori* stool antigen positivity were treated with first- and second-line therapies in accordance with guidelines, suggesting high levels of adherence.⁷

Finally, this study was restricted to the Auckland Region. Susceptibility profiles may vary

geographically across New Zealand, given district-level differences in antibiotic dispensing⁴⁴ and population ethnicity composition. This makes extrapolation to other regions, especially the South Island or rural areas, uncertain. Regular surveillance, review and dissemination of resistance data at both regional and national levels will be essential to inform policy and optimise treatment strategies.

As the first published study of its kind in New Zealand, it is not yet possible to determine whether secondary resistance rates are rising over time, as has been reported internationally. We hope these findings can serve as a benchmark for future comparison.

Conclusion

This study highlights high rates of secondary *H. pylori* resistance to clarithromycin and metronidazole in Auckland, New Zealand. These findings lend support to use of non-clarithromycin-based salvage regimens. Future work should focus on nationwide surveillance of both primary and secondary resistance and on integrating these data into an evidence-based framework for targeted eradication, particularly in high gastric cancer risk groups.

Table 1: Demographic and microbiological characteristics of patients tested and positive cases for *H. pylori*. Data are presented as mean±SD, or number of participants (% of participants).

Parameter		Patients with positive <i>H. pylori</i> culture (n=644)
Age (years)		51.4±15.1
Male sex		281 (43.6%)
Ethnicity	European	77 (12.0%)
	Māori	19 (3.0%)
	Pacific	50 (7.8%)
	Asian	271 (42.1%)
	Other	52 (8.1%)
	Data not available	175 (27.2%)
Year of testing	2018	39 (6.1%)
	2019	86 (13.4%)
	2020	76 (11.8%)
	2021	147 (22.8%)
	2022	166 (25.8%)
	2023	130 (20.2%)
Antibiotic susceptibility testing	Amoxicillin resistance	16/644 (2.5%)
	Metronidazole resistance	441/644 (68.5%)
	Clarithromycin resistance	438/644 (68.0%)
	Tetracycline resistance	0/644 (0.0%)

Table 2: Antibiotic resistance by year. Asterisks denote statistically significant values (p<0.05).

Antibiotic resistance	2018 (n=39)	2019 (n=86)	2020 (n=76)	2021 (n=147)	2022 (n=166)	2023 (n=130)	p-value
Amoxicillin	0/39 (0.0%)	1/86 (1.2%)	0/76 (0.0%)	7/147 (4.8%)	5/166 (3.0%)	3/130 (2.3%)	0.23
Metronidazole	24/39 (61.5%)	61/86 (70.9%)	55/76 (72.4%)	104/147 (70.7%)	112/166 (67.5%)	85/130 (65.4%)	0.75
Clarithromycin	29/39 (74.4%)	61/86 (70.9%)	52/76 (68.4%)	109/147 (74.1%)	108/166 (65.1%)	79/130 (60.8%)	0.19
Tetracycline	0/39 (0.0%)	0/86 (0.0%)	0/76 (0.0%)	0/147 (0.0%)	0/166 (0.0%)	0/130 (0.0%)	>0.99

Table 3: Logistic regression of metronidazole and clarithromycin resistance by demographic factors. Asterisks denote statistically significant values ($p < 0.05$).

		Multivariable-adjusted logistic regression	
		OR (95% CI)	p-value
Metronidazole resistance			
Age (per year)		1.01 (1.00–1.02)	0.18
Male sex		0.47 (0.32–0.72)	<0.001*
Ethnicity	Māori versus European ethnicity	0.31 (0.11–0.92)	0.04*
	Pacific versus European ethnicity	0.36 (0.17–0.80)	0.01*
	Asian versus European ethnicity	1.26 (0.69–2.29)	0.45
	Other versus European ethnicity	1.36 (0.59–3.13)	0.47
Year of testing (per year)		1.01 (0.90–1.14)	0.83
Clarithromycin resistance			
Age (per year)		0.99 (0.97–1.00)	0.08
Male sex		0.73 (0.46–1.15)	0.17
Ethnicity	Māori versus European ethnicity	0.18 (0.06–0.58)	0.003*
	Pacific versus European ethnicity	0.33 (0.15–0.71)	0.005*
	Asian versus European ethnicity	1.99 (1.11–3.57)	0.02*
	Other versus European ethnicity	1.59 (0.69–3.66)	0.28
Year of testing (per year)		0.91 (0.82–1.03)	0.14

COMPETING INTERESTS

Nil.

AUTHOR INFORMATION

Dr Cameron Schauer: Department of Gastroenterology, Health New Zealand – Te Whatu Ora Waitematā; The University of Auckland, Auckland.

Dr Marius van Rijnsoever: Department of Gastroenterology, Health New Zealand – Te Whatu Ora Waitematā.

Dr Susan Taylor: Middlemore Hospital Laboratory, Health New Zealand – Te Whatu Ora Counties Manukau.

Dr Michael TM Wang: Faculty of Medicine, The University of Auckland, Auckland.

A/Prof Russell Walmsley: Department of Gastroenterology, Health New Zealand – Te Whatu Ora Waitematā; The University of Auckland, Auckland.

Prof Jonathan Koea: Department of Surgery, Health New Zealand – Te Whatu Ora Waitematā; The University of Auckland, Auckland.

Dr Stephen Inns: Department of Medicine, University of Otago, Wellington; Health New Zealand – Te Whatu Ora Capital Coast and Hutt Valley; Department of Gastroenterology, Hutt Hospital, Lower Hutt.

Dr Tom Hills: Medical Research Institute of New Zealand, Wellington; Department of Infectious Diseases, Auckland City Hospital, Auckland.

CORRESPONDING AUTHOR

Dr Cameron Schauer: Gastroenterology Unit, Level 1, Tōtara Haumarū, 124 Shakespeare Road, Takapuna, Auckland 0620, New Zealand.
E: cameron.schauer@gmail.com

URL

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