



Māori have a much higher incidence of community-acquired pneumonia and pneumococcal pneumonia than non-Māori: findings from two New Zealand hospitals

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Abstract

To determine the incidence rates of community-acquired pneumonia and pneumococcal pneumonia requiring hospitalisation among Māori and non-Māori, an observational study was conducted in Christchurch and Hamilton. Self-reported data were collected using an interviewer-administered questionnaire. Routine clinical, radiological, and microbiological techniques were used apart from the BinaxNow pneumococcal antigen test for diagnosis of this infection. Census data was used to determine the denominator for statistical analyses. The pneumonia rate overall was 3.03 times higher among Māori than non-Māori ($p < 0.001$). Differences were significant for each 10-year age group from age 45–74 years ($p < 0.05$). The rate of pneumococcal pneumonia was 3.23 fold higher for Māori than non-Māori ($p < 0.001$), but it did not reach statistical significance in the age-related comparisons.

These ethnic disparities are of major concern, and policy planners should consider further interventions to improve the efficacy of current anti-smoking campaigns and to undertake studies of conjugate pneumococcal vaccines for Māori.

Community-acquired pneumonia (CAP) is the most common cause of admission to hospital for adults in New Zealand; it has a reported mortality of between 6.5% and 8%.^{1–3} *Streptococcus pneumoniae* is the most frequently identified pathogen in CAP in New Zealand^{1,2,4} and worldwide.^{3,5,6}

Invasive forms of pneumococcal disease are associated with a high mortality, thus immunisation with the polysaccharide vaccine is recommended for the elderly and those with chronic disease or impaired immunity in New Zealand and elsewhere.⁷

Despite these recommendations, pneumococcal immunisation is very uncommon in New Zealand.⁴ Some ethnic groups are also at increased risk from invasive pneumococcal infection. For instance, a population-based survey in Auckland found that Māori and Pacific Island adults in New Zealand had increased rates of invasive pneumococcal disease, and Māori children have a higher rate of invasive pneumococcal disease than Caucasian (New Zealand European) children.^{8,9}

High rates of invasive pneumococcal disease have been reported in native Americans, African Americans, and indigenous Australians.^{10–14} In response, the Australian guidelines now recommends immunisation of indigenous peoples and Torres Strait Islanders from age of 50 years.¹⁵

There is no convincing evidence that pneumonia can be prevented by the polysaccharide vaccine in older Western populations,¹⁶ but the spread of penicillin-resistant strains of *S. pneumoniae* has renewed interest in the prevention of

pneumococcal infections in order to reduce antibiotic pressure as well as reduce morbidity, mortality, and hospitalisation.^{17,18}

Possible strategies include smoking reduction and the use of the newer conjugate vaccines.^{19,20} Because of the potential importance of such strategies for prevention of pneumonia and pneumococcal infection in New Zealand, in this study we determined the age-specific rates of CAP and pneumococcal pneumonia in Māori and non-Māori populations in two major regional centres.

Methods

Participants—All patients over 18 years of age admitted to Christchurch and Waikato Hospitals between 27 July 1999 and 27 July 2000 with a diagnosis of community-acquired pneumonia were screened for inclusion into the study. Christchurch Hospital and Waikato Hospital each have approximately 600 beds.

Both hospitals are the only hospitals in their respective regions that admit patients with CAP, although both act as tertiary referral centres for larger populations. This study is a further evaluation of patients described previously,⁴ and the inclusion and exclusion criteria for this study were those used in a previous CAP study.¹

Pneumonia was defined as an acute illness with radiographic pulmonary shadowing (at least segmental or present in one lobe), which was neither pre-existing or of another known cause. Patients were excluded from the study when pneumonia was not the principle reason for their admission, when they were moribund at presentation (as relevant history microbiological samples and ethnicity could not be obtained), and when the pneumonia was associated with bronchial obstruction or bronchiectasis (as underlying abnormalities alter host susceptibility). Patients with known tuberculosis were also excluded.

Patients with severe immunosuppression—neutropaenia, individuals with AIDS, or those currently receiving cancer chemotherapy were excluded. Patient characteristics and admission clinical and physical findings were recorded on a standardised proforma. Patients identified their own ethnicity by answering the question. “How do you identify ethnically? You may identify more than one—Pakeha/European, New Zealand Māori, Pacific Islander, Asian, European, Other.”

Recent antibiotic use and pneumococcal vaccination status were self reported by the patient, and comorbidities were reported by the patient and verified by reference to medical records. At the time of enrolment, blood was drawn for haematological, biochemical, and microbiological analysis. Sputum and urine samples were sought from all patients. All chest radiographs were reviewed by a designated radiologist in each centre to confirm radiological entry criteria. Severity of pneumonia was determined by the method of Fine et al.²¹

Microbiological methods—Blood cultures were incubated aerobically and anaerobically using the BacT/Alert Microbial Detection System (Organon Teknika, Durham, NC, USA). Respiratory samples were cultured on sheep-blood agar, chocolate agar, buffered charcoal yeast extract agar supplemented with (-ketoglutarate, and modified Wadowsky-Yee medium. Urine samples were tested using the NOW™ *Streptococcus pneumoniae* Urinary Antigen Test (Binax, Portland, ME) according to the manufacturer's recommendations.

Criteria for diagnosis as pneumococcal pneumonia—This diagnosis was made if the clinical and radiological criteria for pneumonia were fulfilled and *S. pneumoniae* was isolated from a sterile sample (such as blood, pleural fluid, or lung aspirate sample), or when *S. pneumoniae* antigen was detected in the urine.

Statistical analysis—Subjects were excluded from statistical analysis of the rates of pneumococcal disease if a urine antigen test had not been done, unless a blood culture was positive. Rates were calculated from the 2001 primary self declared ethnicity census data for the catchment areas of the two hospitals. A standardised morbidity ratio (SMR) was calculated for the Māori/non-Māori comparison. The expected values for the Māori group were calculated from the observed rates within each age-sex group for the non-Māori group.

Hypothesis testing and 95% confidence intervals for SMRs were derived from the standard Poisson approximation. All data was entered into a specifically designed Microsoft Access database. The SPSS for Windows 10.0 statistical package was used for the analysis (SPSS Inc., Chicago, USA). The level of significance was set at $p < 0.05$.

Results

Patient characteristics—During the 12-month study period, 545 patients were eligible for the study of whom 474 (87%) participants were enrolled. Of these 474 patients, 304 were from Christchurch Hospital and 170 from Waikato Hospital. Of the 71 unenrolled patients, 37 individuals declined study enrollment, 18 were missed for logistic reasons, 10 were unable to give consent and had no available next of kin, and 6 died prior to consent being obtained.

The mean age of those enrolled in the study was 63.7 years (range 18–99 years) and 53% were men. 274 participants (58%) were recorded as having significant comorbidity at time of presentation: 100 (21%) were smokers, 123 (26%) had chronic obstructive pulmonary disease (COPD), 66 (14%) asthma, 52 (11%) diabetes, 95 (20%) heart failure, 28 (6%) renal disease, and 5 (1%) liver disease. Twenty-four patients (5%) were immunosuppressed, 128 (27%) had received an antibiotic prior to admission, 237 (50%) had received influenza vaccine prior to admission, and (19) 4% received pneumococcal vaccine in the previous 5 years. Fifty-seven study participants (12%) identified as being Māori, of whom 41 (72%) were admitted to Waikato Hospital.

Compared with non-Māori, the Māori population were significantly younger (mean age of 50 vs 66 years, $p < 0.001$, difference = 15 years, 95%CI 10–20 years) and they had a significantly higher rate of smoking (35% vs 19%, $p = 0.004$, difference 16%, 95%CI 5%–28%). The mean pneumonia severity index score (PSI)²¹ for CAP was similar among Māori and non-Māori (56 vs 49, difference = 7.5, CI -6%–21%).

The mean PSI for pneumococcal pneumonia was less among Māori (80 v 95, $p = 0.042$, difference = 15, 95% CI 4–25) but this was dependent on the lower age among Māori. There was no statistical difference between the rates of comorbidity other than asthma, antecedent antibiotic use, morbidity, or mortality at 6 weeks follow-up between these two groups (Table 1).

Incidence of community acquired pneumonia—The 2001 Census populations for the Christchurch and Waikato regions for Māori were 27,000 and 39,000, respectively; for non-Māori, they were 327,000 and 198,000 respectively. The population age-specific rates of CAP are shown by ethnicity in Figure 1. The age-specific rates of CAP were statistically significant for each of the three 10-year age groups from 45–54, 55–64, and 65–74 years. The pneumonia rate was 3.03 times higher among Māori than non-Māori in the whole population (Table 2). There was no significant difference in incidence rates by gender or centre in any of the decade groups.

Incidence of pneumococcal community acquired pneumonia—The population age-specific rates of pneumococcal CAP are shown by ethnicity in Figure 2. There was no statistically significant difference in pneumococcal CAP for any of the 10-year age groups, but the rate was higher in the population overall (Table 2). There was no significant difference in incidence rates by gender or centre.

Figure 1. Population age-specific rate of community-acquired pneumonia for Maori and non-Maori (p<0.05)

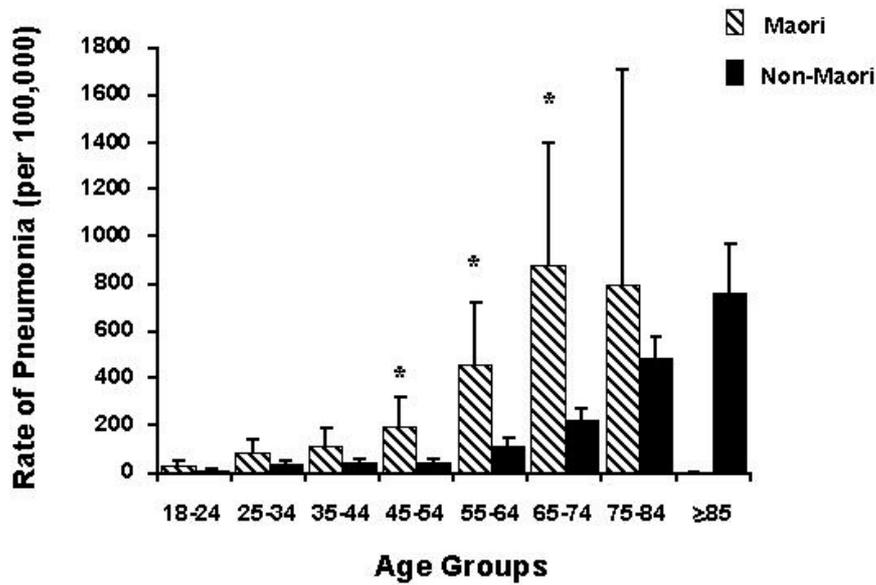


Figure 2. Population age-specific rates of pneumococcal community-acquired pneumonia for Maori and non-Maori

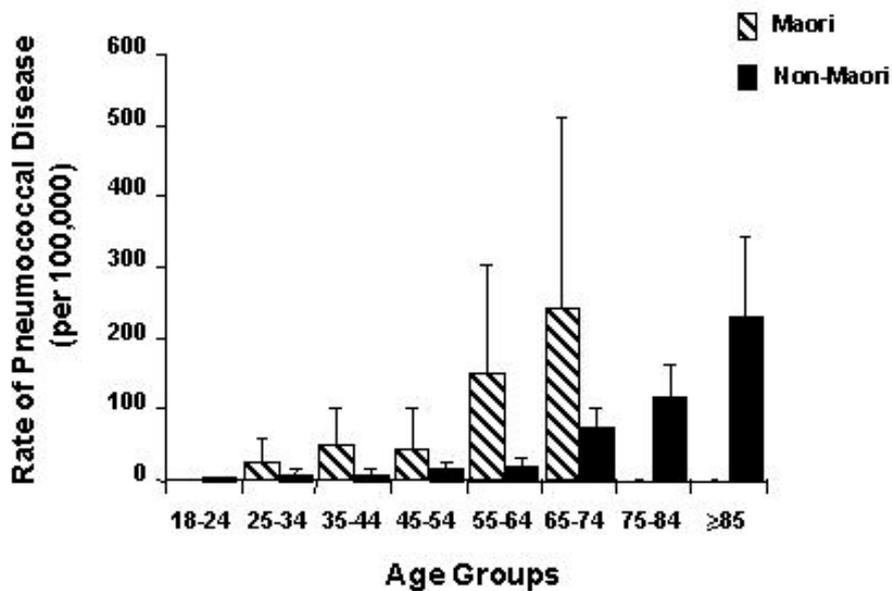


Table 1. Study population patient characteristics and outcome at 6 weeks

Variable	Māori N (%)	Non-Māori N (%)
Community acquired pneumonia	57	417
Pneumococcal community-acquired pneumonia	16 (28)	110 (26)
Prevalence of smoking	20 (35)	76 (19)*
Co-morbidities	36 (63)	238 (57)
Chronic obstructive pulmonary disease (COPD)	12 (21)	109 (26)
Asthma	16 (28)	52 (13)**
Heart failure	15 (26)	81 (19)
Diabetes	8 (14)	46 (11)
Cardiovascular disease	5 (8)	49 (12)
Renal failure	3 (5)	25 (6)
Liver disease	1 (2)	5 (1)
Admission to ICU	1 (2)	15 (4)
Survival at 6 weeks	55 (97)	390 (94)

*p=0.004; **p=0.002.

Table 2. Standardised incidence rates of pneumonia and pneumococcal pneumonia: Māori and non-Māori compared

Variable	Maori	Non-Maori	Standardised morbidity ratio	P value
Pneumonia				
Christchurch	16	8.2	1.94	0.01
Waikato	41	10.6	3.88	0.001
All	57	18.8	3.03	0.001
Pneumococcal pneumonia				
Christchurch	3	1.93	1.55	0.13
Waikato	13	3.01	4.31	0.001
All	16	4.95	3.23	0.001

Discussion

In the catchment areas studied, we have shown that Māori have an increased (3–4 fold) incidence rate of CAP and pneumococcal pneumonia compared with non-Māori. Moreover, the increase in CAP incidence rates seen in non-Māori at aged 65 occurred 20 years earlier among Māori, and pneumococcal pneumonia incidence rates showed a similar pattern.

These incidence rates results cannot be generalised to the general population as the catchment areas of Christchurch and Waikato Hospitals is not representative of the New Zealand population. However, the clear discrepancy in the rates of CAP and pneumococcal pneumonia between Māori and non-Māori (as demonstrated by the disease ratios) probably represents a disease discrepancy present in the general population. These findings are consistent with previous studies that have shown Māori (and other Polynesian) children in South Auckland have increased rates of admission to hospital for pneumonia, and adult Māori have increased rates of invasive pneumococcal disease in the Auckland area compared to Europeans.^{8,9,22}

Identification of ethnicity is important for interpretation of the results of this study. To cross-check that the ethnicity data had been collected correctly, we approached 50% of those who identified as Māori at the conclusion of the study, and found complete correlation with the ethnicity as originally recorded. This reassured us that the ethnicity recorded originally in response to the study ethnicity question was correct.

The denominator for calculation incidence rates was derived from the 2001 Census ethnicity data. Although the 2001 Census does not cover the period of the study, we chose data from this Census rather than an interpolation of the ethnicity data between 1996 and 2001 because the question asked was different in those years.

The ethnicity question asked in this study was slightly different from the question used in both the 1996 and the 2001 census. The 2001 census question reads “Which ethnic group do you belong to? Mark the space or spaces which apply to you.”

This instruction is followed by a list of ethnicities as follows: New Zealand European, Māori, Samoan, Cook Island Māori, Tongan, Niuean, Chinese, Indian, Other. In the Census analysis, people who have recorded more than one ethnic group have been counted in each applicable group.

People answering the Census questionnaire may have been more likely to include more than one ethnicity than those answering the study questionnaire. If so, this will have introduced some systematic bias into the results as Māori ethnicity would have been under-reported in the present study compared with the census data. Such an effect is likely to have reduced the difference, however, and thus would strengthen our study conclusions.

Eighty-seven percent of patient eligible for the study participated, but 71 subjects could not be enrolled. Of these, 34 were unlikely to introduce any ethnic bias, as they could not be enrolled for logistic reasons, death, or an inability to obtain consent. The other 37 declined to be enrolled. This could in part be ethnicity related, but we doubt this was sufficiently strong an influence to compromise the results.

There is also a potential bias in that the non-Māori group included non-Māori Polynesians who have increased rates of childhood pneumonia and invasive pneumococcal disease compared with European New Zealanders).^{8,22} This would tend to decrease observed differences between the groups and thus increase the robustness of the observations, however.

The determination of incidence rates for pneumococcal pneumonia is difficult, as isolation of *S. pneumoniae* from blood or another sterile fluid is insensitive although highly specific, and isolation for sputum has low specificity because of potential contamination from organisms colonising the upper respiratory tract.

In this study, the diagnosis of pneumococcal infection depends largely on the detection of urinary pneumococcal antigen. The method used differs from previous urinary antigen tests in that it detects a soluble cell wall pneumococcal antigen common to all strains. There have been several studies published on the performance of this test,²³⁻²⁶ and it has been licensed by the Federal Drug Administration (FDA) in the US. In an adult population, we estimated the sensitivity to be 80% (and specificity 100%) compared with blood culture, and found the test reliable in the presence of previous antimicrobial therapy.²³ It was highly specific in adults as no positive were

found in a large control population, however others have reported positive results in children with nasopharyngeal carriage of *S. pneumoniae*.²⁶

Taken together these results suggest the test sufficiently robust to be used to estimate the incidence of pneumococcal disease in adult populations.

The reasons why there is an increased rate of CAP and pneumococcal disease in Māori needs to be examined in further epidemiological studies. Indeed, indigenous peoples worldwide have increased rates of pneumococcal disease compared with others in the same geographic region—including Alaskan and Greenland natives, African Americans, and Australian aborigines.^{14–15,26–30}

It is very likely that socioeconomic factors play an important role in this discrepancy. Some of the increase is attributable to smoking, a powerful risk factor for pneumococcal disease, and Māori have higher rates of smoking than the general population.³¹

Other factors such as crowded living conditions, economic status, and access to medical care may contribute to the observed discrepancy but it was not the intention of this study to look for these specific effects and we do not have comprehensive information on the population from which these cases were drawn on which to base any comparisons.

It is possible that some genetic factors exist which contribute to increased susceptibility to pneumonia. Recently Yee et al demonstrated that a homozygous state for FcRIIa-R131 gene is associated with increased mortality for bacteraemic pneumococcal disease, thus suggesting inherited host factors play a role in the pathogenesis of pneumococcal disease,³² and Roy et al observed homozygotes for mannose-binding lectin codon variants were at increased risk of invasive pneumococcal disease.³³ It is likely that other polymorphisms in host genes influence the outcome of pneumonia.

Increased rates of CAP and pneumococcal disease demonstrated in an ethnic group increases the potential benefit of targeted prevention strategies. Such an intervention could include improved antismoking campaigns,¹⁹ and consideration could be given to improved influenza and pneumococcal immunisation rates for Māori. While doubts remain over the efficacy of the polysaccharide vaccine for the prevention of pneumonia,¹⁶ it is effective against bacteraemic disease (about 90% of which is from pneumonia).

At present, this vaccine is scarcely used in any group, presumably because of cost and access considerations, although it is recommended by the New Zealand Ministry of Health for at-risk populations.⁷ There is also evidence that the conjugate vaccine reduces pneumonia in children and has a secondary effect in decreasing pneumococcal disease in adults.²⁰

Careful consideration should be given to evaluating the potential value of both pneumococcal polysaccharide vaccine among Māori adults and the conjugate vaccine in children.

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