THE NEW ZEALAND MEDICAL JOURNAL
Journal of the New Zealand Medical Association

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In this Issue of the Journal

Rurality and pandemic influenza: geographic heterogeneity in the risks of infection and death in Kanagawa, Japan (1918–1919)
Hiroshi Nishiura, Gerardo Chowell

Mortality (i.e. deaths/population) of pandemic influenza has been reported to be smaller in rural areas than cities and towns, potentially suggesting the possible protective effect of rurality. We tested the hypothesis analyzing historical data of Spanish influenza in Kanagawa Prefecture, Japan, from 1918–19, which precisely documented both the numbers of cases and deaths by geographic area. We found that the morbidity (i.e. infections/population) was highest in villages, demonstrating that rurality did not show a predictive value of protection. High morbidity in rural areas highlights the importance of social distancing (i.e. contact interventions) in order to minimise infections in the event of the next influenza pandemic.

Benchmarking home parenteral nutrition in Scotland and New Zealand: disparities revealed
Lyn Gillanders, Janet Baxter, Patrick Ball, Arend Merrie, Ruth F McKee

The Home Parenteral Nutrition Registers for NZ and Scotland showed that over 3 times as many people in Scotland received this type of nutrition support. Crohn's disease is the leading cause of gut failure in both countries and the incidence is very high in both countries. It is difficult to see why the provision of this potentially life-saving therapy should be so different between the two countries.

Sociodemographic characteristics of New Zealand adult smokers, ex-smokers, and non-smokers: results from the 2006 Census
Sharon Ponniah, Ashley Bloomfield

This paper updates the prevalence of regular-, ex-, and never-smokers in New Zealand between the 1996 and 2006 Census. Prevalence refers to the proportion of people who smoke in New Zealand (relative to the size of the population), therefore changes between 1996 and 2006 take changing population numbers into account. Overall, the prevalence of smoking has between 1996 and 2006 has decreased from 23.7% to 20.7%. Maori and Pacific ethnic groups continue to demonstrate higher rates of smoking, along with less economically advantaged groups and the unemployed. This research highlights the importance of including questions on smoking in the New Zealand census in order to monitor changes in smoking in response to various prevention, cessation, and policy initiatives implemented in New Zealand over time.

Low and declining cigarette smoking rates among doctors and nurses: 2006 New Zealand Census data

Richard Edwards, Tom Bowler, June Atkinson, Nick Wilson

This paper describes trends in cigarette smoking among doctors and nurses by gender and specialty area using data from the 1976, 1981, 1996, and 2006 population censuses. Smoking among doctors and nurses has declined steadily since 1976, and is now less than 5% among doctors and 13% among female and 20% among male nurses. Among nurses, smoking among psychiatric nurses remains high (26% male, 30% female). The findings suggest that substantial decreases in smoking are possible and a smokefree cultures can be established among substantial occupational groups who are well informed about the degree of risk, are aware of the reality of the health consequences of smoking, and work in a substantially non-smoking environment. Further research and targeted interventions are needed to address smoking among psychiatric nurses.

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The clioepidemiology of pandemic influenza and next steps for pandemic influenza research in New Zealand

Nick Wilson, Michael G Baker, Lance C Jennings

This issue of the *Journal* includes an article[1] in the realm of clioepidemiology—epidemiology using historical data (after Clio, the muse of history).[2] The article examines Japanese experience with pandemic influenza during 1918–19 and is notable for studying both morbidity and mortality data. It demonstrates that the issues are complex and that just because rural areas may have sometimes been found to experience lower pandemic-related mortality rates, this may not necessarily correspond with lower rural infection and morbidity rates.

Another notable feature of this article is the possibility that the lower urban morbidity rates were attributable to public health (e.g. social distancing) interventions and/or individual level interventions (e.g. mask use) in this part of Japan. Other recent evidence from this pandemic has also indicated that another island nation (Iceland) successfully used public health measures to reduce disease spread.[3] Maritime quarantine used by mainland Australia, Tasmania, and some Pacific Islands also prevented or delayed arrival of this pandemic.[4]

Various recent studies in the United States provide additional evidence that quarantine and "protective sequestration" were effective for some communities for the 1918 pandemic.[5] Similarly, social distancing interventions (e.g. school and workplace closures) were sometimes successful when implemented early and if sustained.[6–8]

Social distancing interventions appear to also have helped when the pandemic eventually reached Sydney.[9] But the Australia experience also warns of the problems of people circumventing the quarantine blockade at state borders and refusing to wear masks.[10]

These general successes with pandemic prevention and mitigation contrast with most other countries for 1918, including New Zealand. Our country hardly attempted serious public health measures to control the pandemic[11] and there were only a few isolated examples of local control successes.[12] Furthermore, errors by New Zealand officials resulted in the spread of the pandemic to Samoa with catastrophic results, a failure for which there has been an official New Zealand Government apology.[13]

More recently New Zealand has invested substantially in pandemic influenza planning[14,15] including extensive simulation exercises.[16] Furthermore, a new revision ("version 17") of the New Zealand pandemic plan is currently underway. Yet the New Zealand health sector still lacks a defined agenda for pandemic influenza research.

The need for such a research agenda has been articulated elsewhere (e.g. for the USA[17]) and actively developed and funded in Australia by their National Health and Medical Research Council (NHMRC).[18]

To start the discussion for New Zealand we provide some initial thoughts on potential pandemic influenza research priorities:

Clioepidemiology—Much remains to be learnt about New Zealand's 1918 pandemic experience. Further analysis of individual level data from citizens and military personnel could be rewarding. For example, to understand why some communities appeared to manage the epidemic much better than others and to investigate differential impacts by socioeconomic position, ethnicity and for healthcare workers.

Modelling spread and containment—Expansion of previous modelling work done in New Zealand[19] to utilise freely available modelling software could be performed (e.g. using *InfluSim*[20]). If detailed travel and time-use data were collated then this could even be fed into one of the supercomputer models available (as used in the US[21]). Such work may clarify the scope for travel restrictions to control spread (e.g. between the North and South Island), and inform when best to institute and lift school and other closures.

Seasonal influenza epidemiology—New Zealand has a comprehensive surveillance system for seasonal influenza that includes primary care, laboratory, hospitalisation, mortality, and immunisation coverage data. Findings from this system are already providing insights into the potential impact of influenza immunisation at a population level.[22–24]

Internationally, such surveillance has provided information on the emergence of adamantane resistance in influenza A viruses[25] and the global spread and seasonality of influenza.[26] With some further refinements, the New Zealand surveillance system could give additional insights into the real burden of disease from seasonal influenza, seasonality,[27] and the spread within the country—which could have implications for a pandemic control. It may also be able to clarify the impact of interventions such as routine school holidays on influenza transmission (as per a study in France[28]).

Population vulnerability to influenza—Seasonal influenza can also be used to investigate patterns of population vulnerability to influenza according to demographic, socioeconomic, and environmental factors. Research in New Zealand is currently using a large cohort-study of social housing applicants and tenants to investigate the effects of housing conditions on hospitalisation with seasonal influenza and pneumonia.

Initial findings suggest that while household crowding has a modest effect on the risk of hospitalisation, having young children living in a household is a key risk factor for adult hospitalisation.[29] A limitation of all work based on seasonal influenza is that pandemic influenza may behave somewhat differently.

Border screening for influenza—New Zealand is currently researching screening instruments for arriving airline passengers to evaluate the performance of methods such as declaration cards, temperature testing, throat/nasal swab collection, and post-arrival follow-up.[30] Given the critical importance of the "keep it out" component of New Zealand's pandemic influenza plan, this research area seems particularly urgent.

Current social distancing phenomena—Research could be done on studying the lessons arising from home detention for prisoners in terms of informing New Zealand responses to social distancing non-pharmaceutical interventions (NPIs). Utilising this "natural experiment" would be much less expensive than paying a random selection of people to adopt such social distancing measures for a trial period. Similarly, there

could be work to explore the impact on New Zealand families with children that arise from sudden school closures—e.g. associated with norovirus outbreaks.

Experiments to assess potential interventions—The Centers for Disease Control in the US is funding experiments to investigate the effectiveness of NPIs—e.g. on mask use and hand hygiene.[31] In New Zealand, there is also a Health Research Council funded study on using hand sanitisers to reduce illness absences in primary school children.[32]

Other health initiatives such as "the Sneeze Safe programme" (www.sneezesafe.co.nz) promoting respiratory hygiene among pre-school and primary school children, based on early intervention studies in Antarctica,[33] provide a basis for further systematic research. Such studies are expensive, but the interventions being investigated may have wide benefits in reducing the morbidity from a wide range of infectious diseases.

Stockpile management—As some of the New Zealand stockpiles of antibiotics, antiviral medication, and of H5N1 vaccine start to expire, there will increasingly be a need for testing to determine if some of these supplies may still be worth retaining for emergency use (i.e. while efficacy persists over a certain minimal level).

Public attitudes to NPIs—Research on public attitudes to NPIs among New Zealanders could help assess acceptability and likely uptake. This research could use surveys (e.g. as per overseas studies[34,35]), focus groups, hui, and citizens' panels or citizens' juries. There is some experience with the latter in New Zealand.[36]

It might even be reasonable to plan on conducting short-duration and rapid turn-around public surveys during a pandemic to help guide the acceptability of public health recommendations and to identify problems with various social distancing NPIs. Indeed, this idea has been suggested in the US for pandemic influenza.[35]

Studying virtual worlds—Virtual worlds may partially reflect real-world human behaviour and are therefore attracting the interest of epidemiologists and social scientists.[37–39] Therefore, researchers could study a typical New Zealand "virtual hospital" or emergency department with avatars controlled by real hospital staff for a day. This process could help inform how the health system responds to various pandemic scenarios.

Identifying co-benefits of influenza pandemic planning—Research could detail the spin-off benefits from pandemic planning for improvements in seasonal influenza control, improvements in other infectious disease control (e.g. if basic hygiene standards are improved) and for improving response to other natural disasters.

In conclusion, this brief preliminary research agenda is far from complete but it does suggest that there is plenty of scope for further worthwhile research in this country. Many of these research outputs will also be of value to New Zealand society even if the next influenza pandemic is far in the future.

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Painting a new picture for practice nurses in a capitated environment: who holds the brush?

Barbara Docherty, Nicolette Sheridan, Tim Kenealy

Perspective is the rein and rudder of painting; Leonardo da Vinci (1452-1519)

The 2001 New Zealand Primary Health Care Strategy (the Strategy) signalled official intention to develop a national primary healthcare nursing workforce of 'well trained primary health care nurses'.[1]

Capitation funding was introduced for general practice, progressively replacing the previous fee-for-service system. Independent Practitioner Associations (IPAs), which were principally business support organisations for general practitioners (GPs), were replaced by Primary Health Organisations (PHOs) with a brief requiring all practitioners, including nurses, to be able to 'influence the organisation's decision-making'.[1]

For nurses working in general practice, the Strategy now provided an opportunity to gain advanced nursing practice qualifications and give credence to a role which had struggled over almost four decades to achieve autonomy and specialty status.

Seven years on from that first flush of expectation, have practice nurses finally been liberated? To answer such a question it is timely to look at the historical genesis of practice nursing, the funding mechanism which established this new nursing category and the resultant implications for today's practice nursing environment.

It is now nearly 40 years since practice nursing entered New Zealand general practice settings via the 1970 Government Practice Nurse Subsidy Scheme (PNSS) which supported GPs employing registered nurses to 'provide a doctor's assistant'.[2]

While the intention of the subsidy was that some aspects of patient care could be delegated to practice nurses, 'giving the doctor more time for work which only he can do',[3] the subsidy as a direct payment to the GP was not directly linked to patient need and it was common for practice nurses to be engaged in non-patient activities such as receptionist duties.

By 1974, the Department of Health, which administered the PNSS, endorsed a role embracing only nursing-related responsibilities and supported this by regulatory subsidy and nursing oversight via the principal public health nurse. But unlike New Zealand's closest paradigm, Great Britain, where practice nursing became recognised as an area of community nursing with a specialist qualification at degree level,[4] New Zealand practice nurses were not given support to develop a nationally standardised clinical or educational framework nor role consistency reinforced by a model for implementation.

By the 1980s, in spite of the multi millions of dollars invested in the PNSS, murky accountability, epitomised by large portions of the subsidy being moved to cover

services such as ACC nursing provision,[5] hastened subtle and progressive erosion of the spirit and intent of the PNSS.

In 1991, with new guidelines introduced for the PNSS, [6] any optimism that practice nursing professional development would progress, disappeared along with any hope of a funding mechanism to support change. The future for practice nursing in New Zealand had now become critically compromised. This nursing entity, with a foundation based purely on a government subsidy, with a name that bears no relationship to any particular specialty area of nursing practice, had become the casualty of a new health marketing model, [7] rather than a potentially rich example of nursing provision in general practice.

Mixed reaction from practice nurses, with many blissfully unaware of the subsidy's shifting status and others perceiving it as doing 'nothing to enhance health gains to patients',[8] meant that with the advent of the Strategy many practice nurses were oblivious to the huge potential to turn around the PNSS misfortunes.

The capitation formula introduced in 2001, now absorbed not only the PNSS monies but also the General Medical Subsidy (GMS), signalling freedom for practice nurses to make clinical decisions by prioritising patient needs before seeing the GP: '…nurses are able to provide the needed services, thereby freeing the GP to handle more complex cases…'.[9]

But previous tension between nurses and IPAs over lack of nursing representation in governance and imposed rather than negotiated practice nurse provision, soon surfaced again with PHOs. Although PHOs were required to demonstrate multidisciplinary involvement 'rather than one group being dominant',[1] insufficient numbers of practice nurses in strategic positions such as PHO boards resulted in a nursing group that was collectively powerless to influence and change national policy.

Many practice nurses have attempted to contribute meaningfully to primary health care but, at times, ambiguous articulation to policymakers and funders has exposed contradictions. In the main, practice nurses are still content to accept delegated nursing tasks aligned with a traditional medical model. There is no defined supervision structure and nursing practice relies on years of experience and accumulated knowledge with no connection to a national primary health care nursing framework requiring specific qualifications.

Acceptance of this status quo has left successive governments, and some general practitioners and PHOs, with the firm belief that supporting the need for more nursing autonomy, professional recognition, and accountability, does not need to be reflected within any funding structure, particularly capitation.

For other practice nurses, requirements of NZ Nursing Council under the HPCA Act, together with an injection of $8.1 million from the Ministry of Health (MoH) committed for the development of the primary health care nursing workforce (scholarships and nursing innovation projects),[10,11,] has provided a major boost to gain advanced nursing qualifications. Even so, application of this knowledge into practice is often constrained by a mismatch with general practice and PHO environments in which the nurse is an employee. Even nurse practitioners, in spite of their legislated scope of practice, cannot yet claim capitation funding.

Several alternative funding models have been proposed by Docherty,[8] amongst others, [12] mostly sharing a common theme that primary health care nursing services and governance should be nurse-defined and nurse-led.

Docherty asserts that PNSS payments have amounted to well over $1000 million since inception and contends that as a failed experiment the now 'invisible' PNSS monies should be removed from the capitation formula and, along with PNSS monies still available through Section 88 Ministry of Health requirements, be initially ring-fenced and directly applied to a new model.

Practice nursing in New Zealand has now come almost full circle and continues to be shaped by specific models of general practice funding. Today, as in 1970, the practice nurse role remains variable and contingent on the needs of the employing general practitioner; even worse, health workers in a capitated practice can be employed without registered nurse status.

Meanwhile, some nurses still act as receptionists, the majority of new practice nurses still learn on the job and judging by recruitment advertisements, computer skills are rated more highly than practice nursing experience.[13–15]

Despite agreement at a national level that a uniform national primary health care nursing education framework is essential, no such framework yet exists. Furthermore, consensus on the future development of practice nursing is unlikely as long as nursing activity continues to be negotiated by general practitioners without accountability, mandate, or reference to an expert base of a national nursing framework.

Practice nursing in 2008 looks like this: an ageing workforce without primary health care qualifications; no standardised career pathway; unacceptable variations in the quality of practice; and a capitation funding system that does not reflect the intent of the Strategy for nurses while continuing to fund PHOs and general practitioners. Painting a new picture for practice nurses in a capitated environment now demands a new perspective.

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Health in New Zealand—overview of Labour Party policy

The New Zealand Labour Party

Labour is committed to a public health system that people can trust, one that is there for them regardless of their ability to pay. We will build upon the positive changes to our health system over this decade to offer the best possible service for all New Zealanders.

General practitioners (GPs) and Primary Health Organisations

We will keep primary healthcare subsidies universal, so that everyone is able to get cheaper doctors visits and cheaper prescription medicines. A million Kiwis now pay no more than $16 to see a GP and the cost of most prescription medicines has been cut to no more than $3. Young families are saving around $550 each year and older couples are saving around $440. We are committed to addressing workforce issues in primary healthcare so that every kiwi has access to quality public healthcare when they need it.

Hospital and District Health Boards

We will continue to invest in increasing surgery that improves people's quality of life. More New Zealanders now receive elective operations than ever before—over 300 every day—and we're committed to lifting that still further.

We will continue to maintain and improve hospital buildings and campuses so they are better able to cope with high demand in the public health system. Five major hospital redevelopments have now been approved and are underway, on top of eight major refurbishments and seven new hospitals built since 1999.

We are committed to retain the right for local communities to have their say on local health issues. Every 3 years you can now vote for your local representatives as part of local government elections. Labour believes that District Health Boards make better decisions for their communities when they are part of that community.

Labour will also ensure that District Health Boards work together to ensure that a consistently high standard of healthcare is available throughout the country. We will ensure that public health resources are used effectively for maximum benefit.

The health workforce

We will continue investing in more frontline staff, building on the 4000 additional nurses and 1000 additional doctors since 1999. Labour will continue to honour its commitment to valuing the role of nurses, following on from the historic pay increase for nurses that took place in 2005. We will retain the recent doubling in the number of GPs to be trained, up from 50 to 104 each year. We will also preserve changes to primary care services that have improved incomes for GPs and are making the area increasingly attractive to work in.

Caring for older people

Labour is committed to the progressive removal of asset testing for older people in long-stay residential care. We will build on major funding increases for aged residential care by looking at ways to improve the funding model, and improve quality controls in the aged care sector to ensure all of our older people get the standard of care they deserve.

We will also maintain the significant increases to home-based support funding, so older New Zealanders can be the ones to choose to stay in their own homes—not have the choice made for them.

We will continue to invest in funding of elective surgery, especially hip and knee replacements and cataract operations, all vital for maintaining independence. And from October 2008 we are lifting the subsidy for hearing aids from $198 to $500.

Child health

Labour is dedicated to making sure our kids get the best start in life. We will continue to roll out the new free *School Ready* check up for all children before they start school. We will also keep supporting successful measures like the universal newborn hearing screening programme and *Well Child* checks for pre-schoolers.

Prevention and promotion

Health is not only about helping people who fall ill, it's also about keeping people well. We're starting to see a halt to the previously relentless advance of obesity in children. Labour will continue to promote healthy, active lifestyles, including through *Fruit in Schools* and *Mission On*, which encourages our children to think and act in ways that keep them fit and healthy.

Labour is committed to fighting against cervical cancer with the introduction of an immunisation programme which is expected to save around thirty lives a year. The human papillomavirus (HPV) immunisation programme will be offered to women aged 12–18.

Dental/oral health

Labour believes that good oral health is fundamental to the overall health of New Zealanders. We will continue with our multi-million dollar investment programme to ensure oral health facilities are up to scratch. We will also keeping building the oral health workforce and continue the fundamental shift towards focussing on preventive oral health.

Protecting the oral health of our children is a top priority. Labour will continue to shift services for children and teenagers from the existing School Dental Service to Community Oral Health Services. These community based clinics will not just provide treatment services, but will also a base to 'reach out' with oral health promotion activities. We will also maintain the increased funding in this area that has enabled there to be more dental assistants and ensured more pre-schoolers receive dental care.

Improved rural services

Labour will continue to support the substantial improvement in rural health services that has occurred in recent years. We will maintain schemes to retain and recruit GPs and nurses, and help medical professionals in rural areas improve access to health services. We will also encourage young doctors to spend time working in rural areas through the Rural Immersion Fund.

We're committed to providing funding to support midwives who practise and work in rural areas, and the mobile surgical bus will continue to bring elective surgeries to rural communities.

Labour policies are available at www.labour08.co.nz

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Rurality and pandemic influenza: geographic heterogeneity in the risks of infection and death in Kanagawa, Japan (1918–1919)

Hiroshi Nishiura, Gerardo Chowell

Abstract

Aim To characterise the impact of rurality on the spread of pandemic influenza by exploring both the numbers of cases and deaths in Kanagawa Prefecture, Japan, from October 1918 to April 1919 inclusive.

Method In addition to the numbers of influenza cases and deaths, population sizes were extracted from census data, permitting estimations of morbidity, mortality, and case fatality by 199 different regions (population 1.4 million). These outcomes were compared between four groups; cities (n=6), larger towns (38), smaller towns (101), and villages (54).

Results Whereas crude mortality in villages was lower than those of other population groups, the morbidity appeared to be the highest in villages, revealing significant difference compared to all cities and towns [risk ratio=0.601 (95% confidence interval: 0.600–0.602)]. Villages also yielded the lowest case fatality, the difference of which was statistically significant among four population groups (p=0.02).

Conclusion Rurality did not show a predictive value of protection against pandemic influenza in Kanagawa. Lower morbidity in the towns and cities is likely explained by effective preventive measures in urban areas. High morbidity in rural areas highlights the potential importance of social distancing measures in order to minimise infections in the event of the next influenza pandemic.

An increase in the number of outbreaks caused by highly pathogenic avian influenza type A (H5N1) virus in poultry, and its transmission in humans, has raised a considerable public health concern over the next pandemic.[1]

Although it is difficult to offer valid prediction of the forthcoming influenza pandemic, exploring previous pandemics is crucial for identifying specific patterns of transmission and suggesting optimal intervention strategies. Influenza caused by type A (H1N1) virus in 1918–19 is known to have caused the world's worst-known influenza pandemic, the so-called 'Spanish influenza' (which did not originate in Spain), causing an estimated 50 million deaths worldwide.

Quantification of the spread and transmission of pandemic influenza should provide valuable suggestions to improve the effectiveness of future pandemic preparedness plans.[2,3]

The mechanisms of transmission that may be deduced from the pattern of geographic spread of pandemic influenza have been demonstrated in several recent studies.[4–8] During the influenza pandemic it has been reported that severity (particularly mortality) differed considerably by geographic locations.[9,10]

Recently, historical data of Spanish influenza in New Zealand was revisited; it suggested that the mortality estimate was significantly smaller in rural areas than cities and towns.[11] Similarly, mortality has been suggested to be high in urban settings in other countries,[12,13] as supported by mathematical models attributing the differential mortality to sociodemographic conditions and public health measures.[14,15]

However, different epidemiologic outcomes have not been comparatively explored to examine the impact of rurality on 1918–19 influenza pandemic (e.g. infection and death). This is mainly owing to limited availability and scarce information of historical data which usually document the number of deaths alone. It is therefore fruitful to discuss this issue, explicitly distinguishing the implications of rurality between infection and death.

In the present study, we uncover a historical record of pandemic influenza in Kanagawa Prefecture, Japan, from October 1918 to April 1919, which precisely recorded both the numbers of cases and deaths by region. This study was aimed at characterising the impact of rurality on influenza by exploring three different outcomes—i.e. morbidity, mortality, and case fatality.

Methods

We extracted historical epidemiologic data of the influenza pandemic in Kanagawa, Japan, from 1918–19.[16] The historical data show numbers of cases and deaths in 199 different administrative regions; the total numbers between October 1918 and April 1919 were documented.

Prior to the pandemic, Kanagawa had suffered only sporadic outbreaks of bubonic plague at different times and places; thus it was believed that the prefectural government had been well trained and particularly successful in precisely tracing the spread of Spanish influenza in the prefecture.[16] In addition to influenza data, population sizes and mean household sizes (i.e. mean number of members per household) by region, as of the end of 1917, were obtained from a census report.[17]

Kanagawa is in the southern Kanto region of Honshu Island; and lies to the north between Yokohama and Tokyo. Ninety years ago the prefecture was very unique in that the capital city Yokohama played a key role as the major port of Kanto region; the main railway lines from Tokyo to southern Japan also passed through that city. Its population at the end of 1917 was 1,359,451, which covered 2415 km^2.

Detailed statistical record was independently summarised only in this prefecture in Japan, which was briefly revisited in a historical study introducing the report as containing the higher quality data.[18]

The present study used population size as a measure of assessing geographic heterogeneity. The populations were categorised as cities (population>20,000), larger towns (5,000<population≤20,000), smaller towns (2,000<population≤5,000), and villages (population≤2,000).

The cut-off values 2000 and 20,000 followed a previous study in New Zealand,[11] and 5000 was the minimum population size prerequisite to legally become a town as indicated by Japanese law.

Since we have access to cases, deaths and population sizes by region, morbidity (cases/population), mortality (deaths/population), and case fatality (deaths/cases) were comparatively examined.

- First, crude estimates of three outcomes were obtained by population group. These estimates were compared between groups using ratio of the outcome variables; i.e. incidence rate ratio (IRR), mortality rate ratio (MRR), and ratio of case fatality proportion (RCF).
- Second, distributions of the outcomes were compared between population groups, using one-way analysis of variance (ANOVA) followed by post-hoc test, employing Dunnett's method.

  When Dunnett's method was applied, villages were set as a control variable. Moreover, mean household sizes were similarly compared by population group, followed by test of within-group correlation by means of the Pearson's product-moment correlation between outcome variables and household size.

All statistical data were analysed using JMP v7.0 statistical software (SAS Institute Inc., Cary, NC, USA).

Results

In total, 292,139 cases and 5021 deaths were recorded during the period of observation, yielding overall morbidity, mortality, and case fatality estimates of 214.9 (95% confidence interval (CI): 214.2–215.6) per 1,000, 3.69 (3.59–3.79) per 1,000, and 1.72 (1.67–1.77)%, respectively.

Estimating the outcomes by region, median (25–75% quartile) morbidity and mortality were 182.7 (87.4–317.5) per 1,000 and 1.62 (0.84–3.06) per 1,000, respectively. Similarly, median (25–75% quartile) case fatality was estimated as 3.1 (1.6–5.1)%, ranging from 0 to 14.2%. Table 1 shows crude estimates by population group.

Table 1. Epidemiologic outcomes of influenza pandemic in Kanagawa, Japan: October 1918--April 1919

Population grouping (N)	Population (N)	Cases (N)	Deaths (N)	Morbidity† (95% CI‡)	Mortality† (95% CI‡)	Case fatality¶ (95% CI‡)
Cities (6)	634107	137028	2290	216.1 (215.1–217.1)	3.61 (3.46–3.76)	1.67 (1.60–1.74)
Larger towns (38)	319346	54901	1166	171.9 (170.6–173.2)	3.65 (3.44–3.86)	2.12 (2.00–2.24)
Smaller towns (101)	346034	79381	1366	229.4 (228.0–230.8)	3.94 (3.74–4.16)	1.72 (1.63–1.81)
Villages (54)	59964	20829	199	347.4 (343.5–351.2)	3.31 (2.86–3.78)	0.96 (0.82–1.09)

† Morbidity and mortality are calculated as rate per 1000 inhabitants for a period between October 1918 and April 1919; ¶Case fatality is proportion of deaths among the total number of cases; ‡ CI, confidence interval.

Morbidity was highest in villages, followed by smaller towns and cities. The risk of infection (measured as IRR) in all cities and towns was 0.601 (95% CI: 0.600–0.602) times that in villages. On the contrary, mortality was lowest in villages, and three other groups yielded a significantly higher estimate [MRR = 1.12 (1.11, 1.12)].

Case fatality was highest in larger towns followed by smaller towns and cities. Villages appeared to yield the lowest case fatality with an estimated 0.96 (0.82–1.09)%. Comparison of detailed ratios is summarised in Table 2.

In villages, crude estimates of mortality and case fatality were significantly lower compared to other population groups, whereas morbidity was significantly higher. Larger towns showed significantly higher case fatality [RCF=1.23 (1.18–1.29)] than smaller towns, but morbidity and mortality were significantly smaller in larger towns [IRR and MRR were 0.749 (0.744, 0.755) and 0.92 (0.89, 0.96), respectively].

Moreover, cities yielded higher morbidity [IRR=1.257 (1.253, 1.261)] than larger towns.

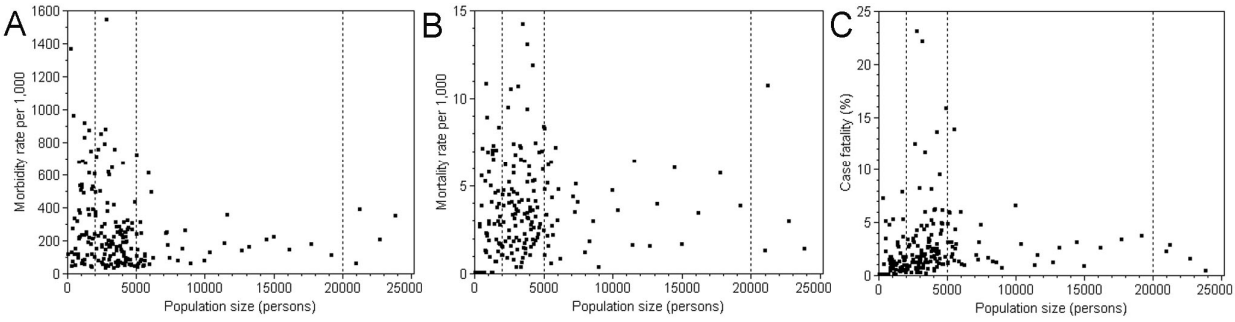
Within cities, the capital Yokohama showed significantly lower morbidity [IRR=0.990 (0.986, 0.993)] compared with five other cities. Within each group, we did not find any significant correlation between the outcomes and population size.

Table 2. Differential risks of influenza pandemic by population groups in Kanagawa, Japan, from October 1918--April 1919

Population grouping (N)	IRR† (95% CI‡)	MRR† (95% CI‡)	RCF† (95% CI‡)
Cities (6)	0.622 (0.621–0.623)	1.09 (1.08–1.10)	1.75 (1.73–1.77)
Larger towns (38)	0.495 (0.493–0.497)	1.10 (1.08–1.12)	2.22 (2.17–2.27)
Smaller towns (101)	0.660 (0.658–0.663)	1.19 (1.17–1.21)	1.80 (1.77–1.84)
Villages (54)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)

†IRR, incidence rate ratio; MRR, mortality rate ratio; RCF, ratio of case fatality proportions; ‡CI, confidence interval.

Figure 1. Morbidity, mortality, and case fatality of influenza pandemic as a function of population size, Kanagawa, Japan: October 1918--April 1919



Three outcomes (A) morbidity, (B) mortality and (C) case fatality of Spanish influenza pandemic are shown in relation to the population size. Each dot represents estimate of a single administrative region. In each panel, three vertical dashed lines represent cut-off values of population size for grouping (i.e. population sizes of 2,000, 5000, and 20,000). Yokohama (n=469,868) and Yokosuka (n=75,325) are excluded from the figure as the population sizes are large. Morbidity, mortality, and case fatality in these cities were 215.5 per 1,000, 3.74 per 1,000, and 1.73 % and 180.0 per 1,000, 2.4 per 1,000, and 1.33 %, respectively.

Figure 1 shows the distributions of the outcomes by population size and group. One-way ANOVA revealed that morbidity was significantly different between population groups (p=0.01), where villages appeared to have experienced significantly higher morbidity than larger towns (p<0.01). Mortality did not differ significantly between

population groups (p=0.33), but we found a significant difference in case fatality (p=0.02) between the groups.

Following the post-hoc test, smaller towns appeared to yield higher case fatality than villages (p=0.01). Unlike the observation using the crude case fatality, case fatality in larger towns was not significantly different from that of villages (p=0.12).

Mean household size significantly differed by population group (p<0.01), which was characterised by significantly smaller household sizes in cities (p<0.01) and larger towns (p=0.02). However, we did not find any significant within-group correlations between mean household size and morbidity as well as mortality.

Discussion

The present study analysed differences in the risks of infection and death of Spanish influenza by population size. Using historical data in Kanagawa, Japan, the numbers of cases and deaths as well as population size were extracted, enabling us to analyse three different outcomes.

To the best of our knowledge, this study is the first to investigate geographic differences in morbidity, mortality, and case fatality, explicitly separating the role of outcomes. Although our case fatality estimates were smaller than those of hospitalised cases among young adult armies in Tokyo,[19] the higher estimate in the hospital most likely highlights more severe cases (i.e. those who were hospitalised) and age (i.e. young adults who were at high risk of death), and our estimates are consistent with that of entire Japan ranging from 0.5–13.7 % with the mean estimate of 1.0%[20] (mortality and morbidity estimates for all prefectures in Japan are given in English in p. 397 of Rice and Palmer[21]).

With regard to crude mortality estimates by population group, MRRs were smaller than those in New Zealand,[11] but the consistent pattern was seen with lowest estimate in villages and highest in smaller towns. However, morbidity was highest in villages. Case fatality proportion bridges the relationship between morbidity and mortality, and villages appeared to yield the lowest estimate, which was significantly different by population group in both comparisons of crude estimates and the corresponding distributions by region.

In other words, the low mortality in villages appeared to be greatly influenced by case fatality, the conditional probability of death given infection (or onset), at least in the unique dataset of Kanagawa. Moreover, when we comparatively examined the distributions of mortality using ANOVA, no significant difference in mortality was found by population group. That is, although our analysis of crude mortality by population group implied a possible protection of the population by remoteness, the difference reflected differing fatality of disease by region, and rather, morbidity appeared highest in villages.

Kanagawa was one of the prefectures where the administrative regions were moderately affected by the influenza pandemic.[21] In a location where extreme remoteness may not be expected,[22] geographic heterogeneity in the risk of infection (i.e. morbidity) revealed opposite pattern of our expectation, showing higher risks of infection in smaller population groups.[14]

Although the data in Kanagawa differs from that of New Zealand in aspects such as the time period and areas of observation, the present study suggests that rurality was not predictive of protection against pandemic influenza when we measured both morbidity and mortality. Considering the similar variations between smaller and larger towns, larger towns showed lower morbidity than smaller towns, and accordingly, smaller towns were also not protected from infection in Kanagawa.

It is difficult to intuitively suggest the definitive reasons why significantly high incidence was seen in villages. Mean household size tended to be higher as population size decreases, but this was not correlated with the risk of infection. Heterogeneous patterns of transmission would not be clarified unless the relevant social and biological backgrounds are explicitly clarified.

As a potential mechanism of intensive within-regional transmission in rural areas, it should be noted that each village was a small community of farmers who lived closely together and were well-connected to each other, and perhaps, this permitted the spread of the disease once the community experienced the introduction of an influenza case.

In practical terms, in order to minimise the risk of infection, high morbidity in rural areas highlights importance of social distancing measures in the event of the forthcoming next pandemic. Provided that rural areas are at high risk of transmission, and given that communities in the present day are more densely connected to each other than those in 1918–19, it would be critically important to protect the community from interregional introduction of cases.

If rural areas indeed prevent themselves from inter-regional introduction of cases by means of social distancing, it will be possible to expect lower risk of infection in these areas.

In addition, towns and cities could have been potentially protected against influenza due to population and individual countermeasures.[18,23] Indeed, public health authorities in Kanagawa were better-prepared for an epidemic than almost any other prefecture in Japan.[24] For example, spinning (cotton) mills in Kanagawa initially suffered from outbreaks in October 1918 and thus the prefecture decided to close similar factories and restricted the movements of individuals in crowded dormitories at an early phase of the pandemic.[16]

The prefecture was also a leader in warning the public of the dangers of influenza and its mode of transmission through the use of pamphlets and posters.[20,24] At the individual level, the use of several different types of mask was recommended not only for those participating in medical practices but also the general population.[20,25]

Mathematical analysis of Spanish influenza data in the US suggests not only that intervention effectively reduced the disaster size, but also that individuals reactively reduced the number of infectious contacts, perhaps by behavioural changes.[14]

Morbidity and mortality with time and place in addition to any information of the timing of implementing public health measures would permit explicit analyses of the effectiveness of countermeasures. To achieve precise estimation of the effectiveness, it is essential to address heterogeneous contact patterns and risk of severe manifestations, and thus further studies are needed to precisely estimate the impact of interventions in heterogeneously mixing populations with varying risks of death.

How about the lower case fatality in villages than in other locations? As a possible reason, differential case fatality could be explained by different levels of previous exposures. A historical study suggests low frequency of previous exposures in town areas by previous pandemic of type A (H1N1) influenza.[18] The similar argument of the impact of acquired immunity on the risk of influenza death (i.e. partial protection) has been made historically.[26] However, if this was the case, not only the risk of death but also that of onset (given infection) should have been more or less inhibited by previous exposure in villages.

In line with this, age-related heterogeneity and underlying diseases have to be remembered as factors generating heterogeneous risks of death. We postulate that some underlying diseases and sociodemographic characteristics have modified case fatality, which in general varies widely by region.[10,27] For example, it is likely that proportion of young adults were higher in cities and towns than that in villages where middle-aged farmers constituted the core of rural population. Moreover, as a potential reason, poorer health and nutrition in towns and cities as well as limited social supports and healthcares in urban areas (e.g. limited nursing care offered by neighbours) could have also contributed to higher case fatality in urban areas.

Further data on socioeconomic status could be useful in testing whether poverty levels in urban areas contributed to higher case fatality than in rural areas.

A limitation must be noted in relation to the interpretation of morbidity and case fatality. If historical survey included many false diagnoses of influenza (e.g. febrile illness caused by different disease), disease misclassification (i.e. non-differential misclassification) must have been present.

Although the historical record in Kanagawa explicitly documents clinical pictures of influenza with the characteristic flu-like symptoms (e.g. fever, myalgia, severe malaise),[16] it is fairly difficult even today to achieve population-based diagnoses of influenza with high sensitivity and specificity. Therefore, if the diagnoses of cases in rural areas included more false negatives than those in cities, reported estimates of morbidity and case fatality in rural areas might be deemed, respectively, overestimate and underestimate, which cannot be fully addressed using the historical record of Spanish influenza alone. Besides, as we briefly discussed, the prefecture had suffered from plague outbreaks prior to the pandemic, and Kanagawa was one of the prefectures where the epidemiologic data by region were most precisely recorded in Japan.

It is worth documenting that agreement between pneumonia and influenza death with time were visually and implicitly examined in the original report.[16] Also, it should be remembered that it is not rare to observe that the regional pattern of influenza morbidity goes in the opposite direction to that of mortality.[28]

As another technical issue, the present study did not account for other variables except for population size. Investigations over age and gender would be desirable, and analyses of similar data in other locations are called for. In particular, historical record in a geographically isolated area (e.g. small island) with both the numbers of cases and deaths has a potential to inspire new knowledge to the world on this issue.

As an epidemiologic implication, the present study would be deemed typical to indicate the critical importance in explicitly distinguishing the roles of outcomes (e.g.

infection and death).[28] It is usually the case that we can obtain death data alone from historical literature. If this is the case, the underlying assumption to make an interpretation and its validity would play key roles to offer valid conclusions.

Specifically, although mortality data are frequently used even for performing predictions,[29] it should be noted that mortality reflects two separate epidemiologic steps (i.e. infection and death) which are differently modified by numerous factors. To decipher the mechanisms of transmission using death data only, some reasonable adjustment or additional case data are needed.

So, weren't rural areas protected against pandemic influenza? Unfortunately, the present study cannot offer explicit general conclusion on this issue. At least, our analysis of the data in Kanagawa suggests high incidence in rural areas, and in this prefecture rural areas were not protected from pandemic influenza in terms of both mortality and morbidity.

Our result was suggestive of potential protectiveness of individuals in rural areas from severe disease (i.e. death given infection), but it has to be clarified more in detail with other variables.[30] Accordingly, the potential importance of social distancing (to minimise the risk of infection) and an epidemiologic need in measuring different outcomes were highlighted. Further studies with different datasets measuring both the numbers of cases and deaths are therefore crucial.

In addition, mathematical and statistical models with spatiotemporal components can be useful tools for deciphering the mechanisms of observing different outcomes by region.

In conclusion, the present study analysed the role of rurality during the 1918–19 Spanish influenza pandemic in Kanagawa, Japan, using numbers of cases and deaths by region. Villages had the highest reported incidence.

If the geographic patterns of morbidity were valid, lower morbidity in the towns and cities might be potentially explained by effective preventive measures in urban areas. However, provided that morbidity data were not sufficiently accurate, slightly smaller estimates of mortality in rural areas still imply the potential protectiveness of remote areas.

In future studies, high resolution spatiotemporal morbidity and mortality data in addition to any information on the timing of public health measures would be crucial for offering the most effective pandemic preparedness plans in heterogeneously mixing populations with varying risks of severe manifestation.

Competing interests: None known.

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Benchmarking home parenteral nutrition in Scotland and New Zealand: disparities revealed

Lyn Gillanders, Janet Baxter, Patrick Ball, Arend Merrie, Ruth F McKee

Abstract

Aim Home parenteral nutrition (HPN) remains the treatment of choice for severe intestinal failure. These patients are few in number but consume significant resource in funding and personnel. Patients receiving HPN in Scotland and New Zealand (NZ) are both tracked through HPN registers which enable clinical audit for identifying important variations in practice. Scotland and NZ have similar demographics, healthcare systems, and populations (Scotland 5.1 million, NZ 4.1 million).

Methods The HPN registers for Scotland and New Zealand for 2005 were examined for patients who received HPN during 2005 together with the diagnostic category identified (ICD-10) that resulted in provision of HPN.

Results The diagnostic categories for the 2005 HPN patients were similar in both countries but rates of provision were much higher in Scotland (71 patients vs 14 patients).

Conclusions Despite similar demographics, healthcare systems, and population size, HPN is utilised to a significantly lesser extent in NZ. The reasons for this are not clear. However, it is possible that there is a lack of recognition of the need for HPN and/or under provision of HPN, which may lead to poorer treatment outcomes.

Home parenteral nutrition (HPN) is a low-volume high-expenditure treatment which allows some patients with inadequate intestinal function or an inaccessible intestinal tract to sustain nutritional and fluid status while remaining at home.[1] Monitoring characteristics, standards of care, and outcomes of HPN patients varies worldwide with some European centres maintaining comprehensive ongoing records through to no national reporting in other countries with similar health standards.[2]

The Scottish Home Parenteral Nutrition Managed Clinical Network (SHPNMCN) has been in existence since 2000 and has maintained an HPN register as part of its activities.[1] The New Zealand (NZ) register was started in 2004 as part of an initiative by the Australasian Society of Parenteral and Enteral Nutrition (AuSPEN) to develop an Australasian HPN register.[3]

The AuSPEN Register was developed in conjunction with the SHPNMCN to allow annual benchmarking. The NZ portion of the register obtained ethical approval in NZ for patients to consent to annual data collection.

Early comparison of register data between NZ and Scotland revealed that patient numbers were much greater in Scotland. This survey was designed to see if underlying diseases leading to provision of HPN were different between Scotland and NZ.

Method

HPN registers for Scotland and NZ were examined early in 2006 for patients who received HPN in each country during 2005 together with the indications for provision of HPN.

To allow direct comparison rates of utilisation, the underlying diseases were mapped to the relevant ICD-10[4] diagnostic classification for digestive diseases. Although both registers include children only adults (18years and over) were included in this analysis.

Results

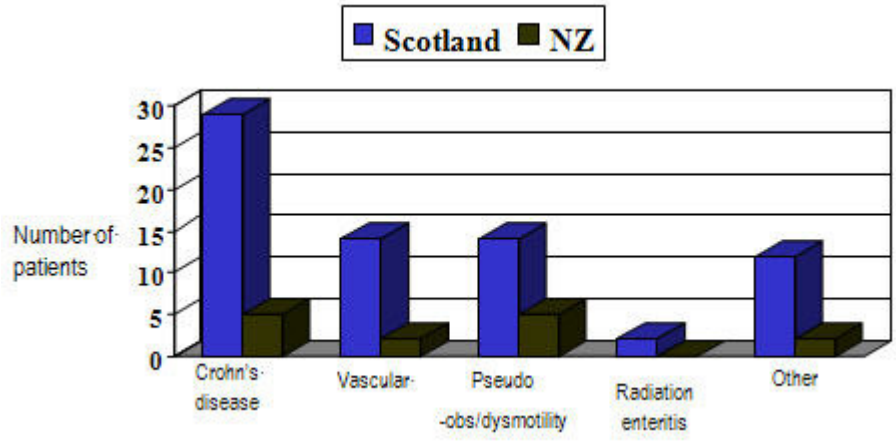
Rates of provision of HPN in 2005 were much higher in Scotland (71 patients vs 14 patients) as shown in Table 1.

Table 1. Rates of provision of home parenteral nutrition (HPN) in Scotland vs New Zealand

Variables	Scotland	New Zealand
Adult patients on HPN	71	14
Population (million)	5.1	4.1
HPN/million total population	14	3.4

The diagnostic categories for the 2005 HPN patients were similar in both countries (Figure 1)

Figure 1. Comparison of the diagnostic categories between Scotland and New Zealand (NZ) HPN patients



Discussion

There is wide variation in practice throughout the world in relation to patient selection for HPN. Within Scotland and NZ patient referral patterns appear to be inconsistent. Some regions in both countries have very few HPN patients. This variation may

reflect availability of treatment as well as differences in prevalence of diseases such as Crohn's disease in different areas.

The prevalence of HPN patients in the UK as a whole is approximately 5/million, although this is probably under-reported according to the British Artificial Nutrition Survey (BANS) Register.[5] The BANS Register also shows that the prevalence of HPN patients in Scotland is similar to regions in the United Kingdom which have recognised intestinal failure units. The UK prevalence varies from 6.2/million to 16.2/million patients. In Scotland the prevalence is 14/million[5] and in NZ it is 3.4/million.[3]

The requirements for the safe delivery of HPN have been outlined both by the National Institutes for Clinical Excellence[6]and the SHPNMCN.[1] Patients should be supervised in units where there is experience in the management of HPN, as well as knowledge and experience in the management of intestinal failure. Units providing this service should have a nutrition support team with at least a clinician, pharmacist, specialist nurse, and dietitian.

The widespread geographical areas of Scotland and NZ make it difficult for patients to travel to recognised centres of expertise for their treatment and some large centres have developed outreach services including protocols and standards of care. When the NZ arm of the Australasian HPN register was established, the SHPNMCN was recognised as a model with established expertise in collection and publication of standards of care and HPN protocols.

Scotland and NZ have similar public health systems, demographics, and population (Table 2). In addition, in NZ the Health Practitioners Competency Assurance Act (2004) introduced the 'comparable health system' path to registration of medical practitioners—a list of 22 countries (including Scotland) regarded as similar to NZ on indicators such as life expectancy and infant mortality.[7] The similar population sizes in each country together with these other factors thus make inter-country benchmarking for a specialist service such as HPN a reasonable process, particularly from the NZ perspective.

Table 2. Demographics compared: New Zealand and Scotland

Country	Population (million)	Life expectancy at birth 2004–2006 (years)[8,9]	2005 GDP (US$) per capita[8,9]
New Zealand	4.1	Males 77.9 Females 81.9	24,400
Scotland	5.1	Males 74.6 Females 79.6	29,923

The diagnostic categories for the 2005 HPN patients were similar in both countries and are also broadly similar to some other European countries according to older data in a survey preformed by the ESPEN-HAN group between December 1998 and March 1999.[10] Nine centres in five European countries participated in this study including only patients with benign diseases who had been receiving HPN for at least

2 years. This survey included 228 adult patients including 141 females and 87 males, with a median age of 49 (19–92) years. The underlying conditions were:

- Crohn's disease (33%),
- Mesenteric vascular diseases (25%),
- Post-surgical (19%),
- Intestinal pseudo-obstruction (8%),
- Radiation enteritis (4%),
- Abdominal trauma (2%), and
- Miscellaneous (8%)

Crohn's disease was the leading diagnostic category for both Scotland and NZ, although the small numbers in NZ make this a more uncertain conclusion. The nature of Crohn's disease can lead to many surgical resections and thus intestinal failure as a result of short bowel syndrome.[11] Some studies have reported an incidence of short bowel syndrome associated with Crohn's disease of between 0.1 and 4%.[12,13]

Inflammatory bowel disease, especially Crohn's disease has increased greatly over the last 50 years. It was estimated recently that the incidence of Crohn's disease was 0.7–14.6 per 100,000 population in Europe and North America.[14] A recent survey in NZ has shown that Crohn's disease incidence and prevalence in the Canterbury region are amongst the highest ever reported.[15] Scottish data is older[16] but the pattern appears similar in the north-eastern region of Scotland (Table 3). A more recent review of Crohn's disease in the east of Scotland confirms this data.[17]

There are large differences in the rates of provision between Scotland and NZ which are difficult to explain on the basis of demographics, healthcare systems, and population. Crohn's disease is most likely the leading cause of intestinal failure in both countries and the prevalence and incidence is high in both countries. Whilst enteral feeding is a common method of nutrition support it is not a successful means of support in the setting of intestinal failure and therefore cannot account for the disparity seen. Thus the indications are similar and in comparison with other European countries such as Italy[18] provision of HPN for cancer is rare.

Table 3. Comparison of point prevalence of Crohn's disease

Country	Date studied	Prevalence/100,000
New Zealand	1 June 2005	155.2
Scotland (North East)	31 December 1988	147
Scotland (East)	2003–2007	157

On this basis, it would be reasonable to assume that HPN use should be similar, particularly for Crohn's disease. These results lead to the question of whether there is over-utilisation of HPN in Scotland or under-utilisation of HPN in New Zealand.

This is not a straightforward question to answer. The Scottish HPN network has adhered well to guidelines about patient selection for HPN.[19] The prevalence of HPN in Scotland is also similar to that in the areas around the two large intestinal failure centres in England and Wales.[5]

Although it may be that the lower prevalence in New Zealand is due to more emphasis on enteral feeding, it is possible that there is under-utilisation of HPN in New Zealand. This might be due to a lack of recognition of the indications for HPN, conservative attitudes on the part of clinicians or under-resourcing of nutrition support and nutrition support teams.

Conclusions:

Despite similar demographics, healthcare systems, and population size, HPN is utilised to a significantly lesser extent in NZ compared to Scotland. The reasons for this difference are not clear. There may be different rates of the underlying indications for HPN although they are similar for Crohn's disease.

It is possible that there is lack of recognition of the need for HPN and/or under provision of HPN, which may lead to poorer treatment outcomes for New Zealanders. Ongoing prospective registration of HPN cases and benchmarking with networks such as the SHPNMCN will clarify this situation further.

Competing interests: None known.

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Sociodemographic characteristics of New Zealand adult smokers, ex-smokers, and non-smokers: results from the 2006 Census

Sharon Ponniah, Ashley Bloomfield

Abstract

Aim To analyse adult smoking rates in New Zealand using 2006 Census data

Methods Data from the 2006 Census of Population and Dwellings were analysed for smoking status against various demographic variables. Data were compared against 1996 Census data to identify changes in smoking prevalence. Age standardised rates were calculated for gender and ethnicity using the WHO Population Standard.

Results The prevalence of regular smokers aged 15 years and over in New Zealand in 2006 was 20.7%, a 3% decrease since 1996. People identifying with Māori and Pacific ethnic groups as well as people who reside in areas of greater deprivation, who earn less, and who are unemployed continue to have the highest rates of smoking. The prevalence of never having smoked regularly has increased among 15 to 19 year olds; increases are not reflected to the same extent among 20 to 24 year olds, however, thus suggesting a possible increase in the age of initiation over the last 10 years.

Conclusion While the prevalence of smoking in New Zealand continues to decline, the decrease is gradual—3% over 10 years, largely due to a reduction in initiation rather than increased cessation—and significant ethnic and socioeconomic inequalities in smoking persist. These findings are being used to inform policy and practice in tobacco control, including ensuring that programmes and initiatives are accessible and reliably delivered to groups with the highest smoking rates.

New Zealand is recognised internationally as having a comprehensive tobacco control programme, structured around evidence-based interventions proven to reduce smoking.[1] Smoking, however, remains the single largest preventable cause of death and disease, with approximately 5000 deaths attributable to smoking in New Zealand each year.[2]

Reducing harm caused by tobacco is a key health target,[3] championed by the Ministry of Health and a range of initiatives around policy, prevention, and cessation have been implemented in recent years. As part of a comprehensive survey programme to monitor progress towards reducing tobacco-related harm, information on smoking was collected in the 1996 and 2006 New Zealand Census of Population and Dwellings.

The Census provides the most accurate representation of smoking in the population and therefore is a vital tool for policymakers and practitioners to monitor the contribution of smoking to premature disease and death and to monitor the effectiveness of tobacco control measures over time.[1–6]

This paper utilises 2006 Census data to describe and analyse the current smoking 'picture' in New Zealand. Comparisons are made with data from the 1996 Census to identify changes over the last 10 years.

Methods

Two questions relating to tobacco smoking from the New Zealand 2006 Census of Populations and Dwellings were analysed. The two questions asked in the 2006 Census were "Do you smoke cigarettes regularly?" and "Have you ever been a regular smoker of one or more cigarettes a day?"

Prevalence estimates were calculated for regular smokers, ex regular smokers, and people who have never smoked regularly; people not answering the question were excluded from the denominator. Age-standardised estimates using the direct method were calculated for gender and ethnic groups using the WHO Population Standard.

Descriptive data from the Census 2006 are presented by gender, age, ethnic group, and a sociodemographic score for level of deprivation (NZDep06).[7] Data are also presented for workforce, labour status, and personal income. Data previously analysed from the 1996 Census were used to compare changes in the prevalence of regular smokers, ex-smokers, and never-smokers between the two censuses.

The 2006 Census reported a non-response rate of 5.2% for compared with 7.2% in the 1996 Census.

Results

The prevalence of smoking in the New Zealand adult population (aged 15 years and over) was 20.7% in 2006—representing around 597,792 people who regularly smoke.

Table 1. Prevalence (% of total population) of *regular smokers*, 2006 Census—by gender, ethnic group, and age group

Age group	European		Māori		Pacific Peoples		Asian		Other Ethnicity		Total	
	female	male	female	male	female	male	female	male	female	male	female	male
15–19 years	19.0%	17.2%	40.6%	31.4%	21.3%	20.1%	3.7%	6.7%	11.3%	12.6%	19.7%	17.8%
20–24 years	29.4%	30.9%	52.9%	47.4%	37.8%	40.3%	8.1%	23.5%	20.9%	25.3%	28.1%	31.6%
25–29 years	26.8%	29.8%	50.9%	44.9%	36.6%	41.3%	6.8%	23.9%	19.6%	24.9%	26.6%	30.9%
30–39 years	23.2%	25.8%	50.5%	43.7%	31.3%	38.0%	4.8%	20.1%	19.2%	22.8%	23.9%	27.5%
40–49 years	21.6%	22.6%	50.2%	41.9%	27.9%	36.6%	3.9%	19.6%	18.3%	19.5%	22.3%	24.3%
50–59 years	17.3%	18.7%	40.6%	34.2%	19.5%	32.8%	3.2%	16.2%	14.1%	14.7%	17.8%	19.6%
60–64 years	14.0%	16.1%	32.5%	26.4%	13.2%	26.9%	2.6%	13.0%	10.7%	11.5%	14.2%	16.3%
65+ years	6.9%	8.6%	18.4%	16.1%	7.8%	19.7%	2.3%	9.6%	6.0%	6.7%	7.1%	8.9%
15+ years	18.6%	20.3%	45.5%	38.5%	27.3%	33.5%	4.9%	18.1%	15.4%	17.6%	19.6%	21.9%
Age-standardised rate	19.4%	21.0%	44.8%	37.9%	26.6%	33.2%	4.7%	17.7%	15.5%	17.8%	20.1%	22.4%

Gender and age group—The prevalence of smoking was 19.6% for females and 21.9% for males (Table 1). By age group, the highest rate of smoking was seen in 20–24 year olds (29.8%), decreasing gradually with increasing age. Male smoking rates were higher than those for females in every age group, except for the 15 to 19 year age group.

Ethnic group—Ethnicity in the 2006 Census was reported using a grouped, 'total response' method where one individual may be recorded more than once for each ethnic group they identify with.

Rates of smoking were highest for Māori (42.2%) and Pacific (30.3%) ethnic groups, with lowest rates among Asian ethnic groups (11.1%) (Table 2). The prevalence of smoking among European ethnic groups was 19.4%.

Table 2. Prevalence of regular smokers, ex-smokers, and never-smokers, 2006 Census—by ethnic group and age group

Age group	European	Māori	Pacific Peoples	Asian	Other ethnicity	Total
Regular smoker						
15–19	18.1%	36.1%	20.7%	5.2%	11.9%	18.8%
20–24	30.1%	50.3%	39.1%	15.7%	23.3%	29.8%
25–29	28.2%	48.2%	38.8%	14.9%	22.4%	28.6%
30–39	24.4%	47.4%	34.5%	11.4%	21.1%	25.6%
40–49	22.1%	46.3%	32.1%	10.9%	18.9%	23.3%
50–59	18.0%	37.5%	26.1%	9.4%	14.4%	18.7%
60–69	13.4%	26.3%	18.4%	7.0%	9.8%	13.6%
65+	7.7%	17.3%	12.9%	5.7%	6.3%	7.9%
Total (15+)	19.4%	42.2%	30.3%	11.1%	16.5%	20.7%
Age standardised	20.2%	41.6%	29.8%	10.8%	16.7%	21.2%
Ex-smoker						
15–19	4.9%	6.00%	3.9%	2.0%	3.9%	4.6%
20–24	13.4%	11.8%	9.4%	5.2%	12.9%	11.3%
25–29	19.1%	15.1%	11.8%	7.0%	18.3%	16.2%
30–39	21.6%	18.3%	11.7%	7.5%	21.6%	19.1%
40–49	24.3%	23.6%	12.6%	8.3%	25.8%	22.3%
50–59	29.2%	29.1%	13.1%	10.1%	31.4%	27.6%
60–69	34.5%	34.1%	14.1%	11.3%	37.1%	33.1%
65+	36.3%	35.1%	16.4%	11.9%	38.3%	35.1%
Total (15+)	24.5%	19.1%	10.8%	7.2%	25.6%	22.1%
Age standardised	23.6%	19.8%	11.1%	7.5%	24.4%	21.4%
Never smoked regularly						
15–19	76.9%	57.9%	75.4%	92.8%	84.2%	76.7%
20–24	56.5%	37.9%	51.5%	79.1%	63.8%	58.9%
25–29	52.8%	36.7%	49.4%	78.0%	59.3%	55.2%
30–39	53.9%	34.3%	53.9%	81.0%	57.3%	55.3%
40–49	53.6%	30.1%	55.3%	80.7%	55.3%	54.5%
50–59	52.8%	33.4%	60.8%	80.6%	54.1%	53.7%
60–69	52.1%	39.7%	67.5%	81.7%	53.1%	53.3%
65+	56.1%	47.6%	70.7%	82.3%	55.4%	56.9%
Total (15+)	56.1%	38.7%	58.9%	81.7%	57.9%	57.3%
Age standardised	56.3%	38.7%	59.1%	81.8%	58.8%	57.5%

Level of Deprivation (NZDep06)—The prevalence of smoking increases steadily with increasing deprivation, with a more than three-fold difference between the least and most deprived decile. In the most deprived areas (decile 10), the prevalence of smoking is 36.5%, compared with 10.7% in the least deprived areas (decile 1). See Table 3.

The prevalence of smoking increases from decile 1 to 10 in all ethnic groups. For Māori, the prevalence of smoking increases sharply from a relatively high rate of 21.9% in decile 1 (least deprived) to 52.9% in decile 10 (most deprived).

The prevalence of smoking among Pacific peoples does not vary as greatly across decile as it does for other ethnic groups, with a smoking rate of 19.9% in decile 1 and 32.8% in decile 10. The smoking rate for European New Zealanders increases from 10.8% in decile 1 to 33.9% in decile 10.

Table 3. Prevalence of regular smokers, 2006 Census—by ethnic group and level of deprivation (NZDep06)

Deprivation decile	European	Māori	Pacific Peoples	Asian	Other Ethnicity	Total
Decile 1	10.8%	21.9%	19.9%	7.5%	9.7%	10.7%
Decile 2	13.7%	28.2%	24.2%	8.9%	12.1%	13.6%
Decile 3	15.4%	30.3%	25.8%	9.7%	13.3%	15.3%
Decile 4	17.1%	33.4%	25.9%	9.7%	15.1%	17.0%
Decile 5	18.8%	35.9%	26.9%	10.6%	16.3%	18.8%
Decile 6	20.8%	38.8%	28.8%	11.4%	18.3%	21.0%
Decile 7	22.8%	41.0%	28.9%	11.9%	19.7%	23.1%
Decile 8	25.5%	43.7%	30.6%	12.5%	21.9%	26.1%
Decile 9	28.1%	46.8%	31.2%	12.9%	23.9%	29.5%
Decile 10	33.9%	52.9%	32.8%	15.1%	27.6%	36.5%
Missing	7.4%	33.9%	33.3%	16.7%	10.8%	8.6%
Total	**19.4%**	**42.2%**	**30.3%**	**11.1%**	**16.5%**	**20.7%**

Workforce, labour status, and personal income—The prevalence of smoking is highest among the unemployed (36.5%) and this is consistent for all ethnic groups (Table 4). Higher rates of smoking are observed among those earning between $15,000 and $30,000 a year, with rates of smoking lower for those earning greater than $50,000 a year. This pattern is consistent across all ethnic groups.

Among Māori earning less than $30,000 a year, the rate of smoking is more than double that of non-Māori; this difference reduces with increasing income. See Table 5.

Table 4. Prevalence of regular smokers, 2006 Census by ethnic group, and workforce and labour status (WFLS)

WFLS	European	Māori	Pacific Peoples	Asian	Other Ethnicity	Total
Employed full-time	21.8%	40.7%	33.7%	12.72%	18.1%	22.6%
Employed part-time	16.2%	37.4%	25.9%	9.6%	13.2%	17.2%
Unemployed	35.0%	57.5%	41.0%	13.9%	27.4%	36.5%
Not in the labour force	15.7%	43.1%	24.7%	9.4%	13.5%	17.5%
Total	**19.4%**	**42.2%**	**30.3%**	**11.1%**	**16.5%**	**20.7%**

Table 5. Prevalence of regular smokers, 2006 Census—by ethnic group and personal income (in NZ dollars)

Personal income	European	Māori	Pacific Peoples	Asian	Other Ethnicity	Total
<$15,000	18.0%	42.8%	27.0%	10.0%	15.0%	19.4%
$15,000–30,000	23.2%	48.4%	35.4%	13.7%	19.9%	24.9%
$30,000–50,000	21.9%	39.2%	33.5%	12.4%	19.2%	22.8%
>$50,000	12.7%	24.4%	25.3%	8.7%	11.6%	12.9%
Total	**19.4%**	**42.2%**	**30.3%**	**11.1%**	**16.5%**	**20.7%**

Ex regular smokers—The proportion of ex regular smokers in New Zealand was 22.1% in 2006 and increases almost linearly with age. A higher percentage of ex-

smokers aged between 15 and 39 years was female, while higher percentages of ex-smokers aged 40+ years was male. The prevalence of ex regular smokers was 24.5% among Europeans, 19.1% among Māori, 10.8% for Pacific peoples, and 7.2% for Asian ethnic groups (Table 1).

Never smoked regularly—The proportion of people who have never smoked regularly was 57.2% in 2006. Although over three-quarters (76.7%) of people aged 15 to 19 years have never smoked regularly, this percentage decreases with age, with just over one in every two people aged between 20 and 59 having never smoked regularly. The prevalence of Māori who have never smoked regularly is 38.7% (Table 1).

Changes in the prevalence of regular smoking between 1996 and 2006—The prevalence of smoking in New Zealand declined by three percentage points between 1996 and 2006 (Table 6). The greatest reduction in smoking prevalence occurred in the 25 to 29 year age group (3.8% points), followed by 30 to 39 year olds (by 3% points). Greater reductions in prevalence were seen in females than in males, particularly in the 20–24 and 25–29 year age groups (5% and 5.8% respectively).

Table 6. Prevalence of regular smokers, ex-smokers, and never-smokers, 1996 and 2006 Census—by gender and age group

Age group	1996			2006		
	Female	Male	Total	Female	Male	Total
Regular smoker						
15–19	22.5%	20.1%	21.3%	19.7%	17.8%	18.8%
20–24	33.1%	31.2%	32.2%	28.1%	31.6%	29.8%
25–29	32.3%	32.6%	32.5%	26.6%	30.9%	28.6%
30–39	27.5%	29.8%	28.6%	23.9%	27.5%	25.6%
40–49	23.1%	25.7%	24.3%	22.3%	24.3%	23.3%
50–59	19.9%	23.1%	21.5%	17.8%	19.6%	18.7%
60–64	15.2%	19.0%	17.1%	14.2%	16.3%	15.2%
65+	9.1%	12.5%	10.6%	7.1%	9.0%	8.0%
15+	22.8%	24.8%	23.7%	19.5%	21.9%	20.7%
Ex-smoker						
15–19	6.9%	4.7%	5.8%	5.4%	3.8%	4.6%
20–24	12.2%	8.5%	10.4%	12.3%	10.3%	11.3%
25–29	17.0%	12.9%	15.0%	17.8%	14.4%	16.2%
30–39	20.9%	18.9%	19.9%	20.6%	17.5%	19.1%
40–49	22.0%	27.1%	24.5%	21.9%	22.7%	22.3%
50–59	23.1%	34.7%	28.8%	24.5%	30.8%	27.6%
60–64	24.2%	41.7%	32.9%	27.6%	38.4%	32.9%
65+	22.8%	51.0%	35.0%	25.2%	47.3%	35.1%
15+	19.4%	24.9%	22.0%	20.3%	23.9%	22.1%
Never smoker						
15–19	70.6%	75.1%	72.9%	74.9%	78.4%	76.7%
20–24	54.7%	60.2%	57.4%	59.5%	58.1%	58.8%
25–29	50.7%	54.4%	52.5%	55.6%	54.7%	55.2%
30–39	51.6%	51.3%	51.5%	55.5%	55.1%	55.3%
40–49	54.9%	47.2%	51.1%	55.8%	53.0%	54.5%
50–59	57.0%	42.2%	49.7%	57.7%	49.6%	53.7%
60–64	60.6%	39.3%	50.0%	58.2%	45.3%	51.8%
65+	68.1%	36.6%	54.4%	67.7%	43.7%	56.9%
15+	57.9%	50.3%	54.2%	60.1%	54.1%	57.2%

The prevalence of smoking among Māori declined by only 1.5% between 1996 and 2006. The greatest decrease in the prevalence of smoking in Māori was among people

aged 25 to 39 years (by around 2.8%). The prevalence of smoking among Māori aged 20–24 years has increased by 1.4%. Among non-Māori, the prevalence of smoking has decreased less than among Māori (around 1%). The greatest decreases are observed among those aged 65+ years (by 2.4%). See Table 7.

Table 7. Prevalence of regular smokers, 1996 and 2006 Census—by Māori, non-Māori, and age group

Age group	Māori		Non-Māori	
	1996	2006	1996	2006
15–19	35.6%	36.1%	17.7%	18.0%
20–24	48.9%	50.3%	28.5%	29.1%
25–29	51.0%	48.2%	28.8%	27.9%
30–39	50.1%	47.4%	25.1%	24.8%
40–49	44.7%	46.3%	22.0%	22.5%
50–59	37.5%	37.5%	20.0%	18.2%
60–64	28.3%	29.6%	16.1%	14.9%
65+	19.7%	17.3%	10.2%	7.8%
15+	43.7%	42.2%	21.0%	20.1%

The proportion of ex-smokers in the population has not changed between 1996 and 2006, although the absolute number will have increased given the increase in the size of the New Zealand population (Table 7). Among males, the prevalence of ex-smokers has decreased, particularly among those aged 40+ years. In contrast, the prevalence of ex smokers among females in these age groups has increased. See Table 6.

The prevalence of people who have never smoked regularly in New Zealand increased by 3.0% between 1996 and 2006. The prevalence of females who have never smoked regularly has increased by 2.2%; the prevalence of males who have never smoked regularly has increased by 3.8% (Table 6).

Greater increases in the prevalence of never-smokers are evident among 15 to 19 years, with similar increases among males and females. The prevalence of never-smokers aged 20–24 years did not increase to the same extent as 15 to 19 year olds (Table 6).

Discussion

There are two key findings from this analysis of 1996 and 2006 Census smoking data.

First, smoking among people aged 15+ years declined from 23.7% in 1996 to 20.7% in 2006, with a reduction seen in every age group. Encouragingly, smoking rates among Māori have reduced more than smoking rates among non-Māori, largely driven by greater reductions in prevalence of smoking in Māori aged 25 to 29 years compared with non-Māori in the same age group.

A 3% reduction in smoking prevalence represents about 100,000 less smokers, half of whom would have died prematurely as a result of their smoking. However, it is also a relatively slow rate of change and if change continues at the same rate, it will be a

further 60 years before the smoking rate in the general population reaches the level it is currently among doctors in New Zealand (3.4%).[8]

The second key finding is that most of the reduction in smoking prevalence between 1996 and 2006 is explained by an increase in never-smokers, with little change in the proportion of ex-smokers in the population. This implies that efforts to reduce smoking initiation have been relatively more successful than those for smoking cessation. This finding is behind a renewed focus on improving the delivery of cessation advice and support to smokers throughout the health sector, and new initiatives to prompt quit attempts including the introduction of graphic pictorial warnings on tobacco packets.

People aged 20 to 49 years and people who identify with Māori and Pacific ethnic groups have higher regular smoking rates than other age and ethnic groups. People with lower personal incomes and people who are unemployed also have higher rates of smoking than those on higher incomes, a pattern that is consistent across all ethnic groups. This evidence highlights the continuing ethnic and socioeconomic inequalities associated with smoking, thus reinforcing the need to target initiatives towards these groups.

Smoking rates increase with increasing deprivation, most markedly for Māori and less so for Pacific peoples than other ethnic groups. Interestingly, the differences between Pacific peoples and European New Zealanders is much less evident in deprived than in less deprived areas—a pattern that is worth further investigation.

Although the overall rates of ex regular smokers are comparable between Māori and non-Māori, it highlights the issue of increasing inequalities in this country.[9–13] Higher rates of ex-smokers identify with European ethnic groups rather than Māori and/or Pacific groups, reiterating that either more initiatives need to available and/or existing initiatives be better targeted towards these groups to increase the prevalence of ex-smokers.

There appears to have been a shift in prevalence of 15–19 and 20–24 year old never-smokers between 1996 and 2006. The prevalence of never-smokers increased by almost 4% between 1996 and 2006 among 15–19 year olds; the prevalence of never having smoked regularly among 20–24 year olds only increased by 1.4 percentage points, however.

Similar declines in the prevalence of regular smoking between these two age groups suggest that the age of smoking initiation in New Zealand is increasing. Evidence from an annual census of secondary schools illustrates significant reductions in smoking among Year 10 students (aged 14 and 15) since 1999,[14] thus supporting the theory for a delay in smoking initiation. This finding warrants further investigation and has important implications for the target groups and content of future prevention and/or cessation initiatives.

The Census provides the most accurate and representative information on smoking prevalence in New Zealand. A nationally representative survey, the New Zealand Tobacco Use Survey (NZTUS), was also conducted in 2006 and recorded a smoking prevalence of 23.5% for the population aged 15 to 64 years.[6] The equivalent figure for this age band from the 2006 Census is 23.0%, which is comparable to the NZTUS estimate.

Limitations of the 2006 Census data include the way smoking status was derived and defined, and changes in coding of ethnic group. The Census definition of regular smoking equates most closely to daily smoking. Internationally, this is seen as an underestimate of the true prevalence of smoking in the population as it excludes non-daily and occasional smokers.

The New Zealand Tobacco Use Survey[6] indicates that the prevalence of current, non-daily smoking is around 2% and this should be taken into consideration when making comparisons between different data sources.

Between 1996 and 2006, there was a change to the way ethnic group was recorded and analysed, from 'prioritised' to 'total response' ethnicity classification.[15] This change means it is not appropriate to compare rates for different ethnic groups between the two censuses, and it is for this reason that only Māori/non-Māori comparisons have been made.

Smoking information was not collected in the 2001 census so it is not possible to more fully explore changing smoking patterns between 1996 and 2006; whether these occurred early or late in the period, for example. The collection of smoking information in subsequent Censuses is therefore essential to provide an accurate picture of changes in smoking in the population to inform policy and practice and should be advocated for in order to provide accurate and consistent monitoring of changes in smoking prevalence over time.

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Low and declining cigarette smoking rates among doctors and nurses: 2006 New Zealand Census data

Richard Edwards, Tom Bowler, June Atkinson, Nick Wilson

Abstract

Aims To examine smoking status among doctors and nurses using data from the 2006 Census and to describe recent trends in smoking prevalence among doctors and nurses.

Methods Analysis of smoking status in 2006 New Zealand Census among medical practitioners and midwifery and nursing professionals, and comparison of cigarette smoking prevalences with findings of previous analyses of the census and surveys of doctors.

Results There were 6312 male and 4197 female doctors, and 2469 male and 32,682 female nurses included in the 2006 Census. Non-response to the smoking status questions were less than 5%. Only 4% of male doctors and 3% of female doctors were regular cigarette smokers in 2006. Among specialist groups, the highest smoking prevalence was 12% among male obstetricians and gynaecologists, and 10% among female radiologists and radiotherapists.

13% of female and 20% of male nurses were smokers. The highest smoking prevalences were among psychiatric nurses (26% male and 30% female nurses). There has been a steady decline in cigarette smoking among doctors and nurses in New Zealand since the 1960s and 1970s.

Conclusions The results from the 2006 Census demonstrate that non-smoking among doctors and nurses is increasingly the norm, around 90% of younger doctors have never been regular smokers. The results show that it is possible to achieve very substantial decreases in smoking prevalences and to establish smokefree cultures among substantial occupational groups who are well informed about the degree of risk, are aware of the reality of the health consequences of smoking, and work in a substantially non-smoking environment.

Smoking among health care workers, particularly doctors and nurses, has been a commonly researched topic since the health effects of smoking became widely known in the 1950s. Recent systematic reviews found 81 and 73 English language papers describing smoking among doctors and nurses respectively.[1,2] Health care workers are a key potential role model group for promoting a smokefree society, and due to their very high level of awareness of the major health effects of smoking are a potential vanguard group for achieving very low smoking prevalence.

In New Zealand, surveys of smoking among doctors on the medical register were carried out in 1963 and 1972.[3,4] These were followed by a series of analyses conducted by Sir David Hay of smoking among doctors and nurses based on data from the 1976, 1981, and 1996 censuses.[5–8] Each of these censuses included a

question on smoking status. On the basis of the encouraging trends in these analyses, Sir David speculated that by 2000 there could be a generation of non-smoking doctors.[7]

A recent letter to the *Journal* noted that no such similar analysis had been carried out using the 2006 Census data,[9] in response to which the prevalence among broad categories of health professionals has recently been reported.[10] There has also been a recent report on smoking among nurses in New Zealand giving overall prevalence of smoking and smoking by area of practice in the 2006 Census.[11] This report also covered a more detailed survey of beliefs and practices about smoking cessation among 371 nurses.

The purpose of this paper is to examine in more detail the smoking status of doctors and nurses using data from the 2006 Census and to describe recent trends in cigarette smoking prevalence among these health professionals.

Methods

Analyses are based on responses to two questions on smoking in the 2006 New Zealand Census. [12] Responses to the questions are used to categorise individuals as current regular smoker, ex-regular smoker, and never-regular smoker. We excluded from the denominator all subjects who did not have valid data for the smoking questions.

- Do you smoke cigarettes regularly (that is, one or more a day)? Count only tobacco cigarettes. Don't count pipes, cigars, or cigarillos. (Yes / No)
- Have you ever been a regular smoker of one or more cigarettes a day? (Yes / No)

Responses to the smoking questions were analysed by age in four groups (15–24 years, 25–44 years, 45–64 years, 65+ years), by sex, and by occupation using levels 4 and 5 of the Australian and New Zealand Standard Classification of Occupations.[13] Student nurses and medical students were not separately coded in the census analyses, and are not included in the data presented.

For examining trends we considered data from surveys of doctors in 1963 and 1972,[3,4] and from censuses that included questions on smoking (in 1976, 1981, 1996, and 2006).[5–8]

There are minor discrepancies in the totals between and within the tables as all the numbers were random rounded to a multiple of three as per Statistics New Zealand protocol.

Results

Number of respondents and response—There were 6312 male and 4197 female doctors, and 2469 male and 32,682 female nurses included in the census. Non-response to the smoking status questions was 2.9% in male doctors, 3.3% for female doctors, 4.5% for male nurses, and 3.5% among female nurses. This compared with a non-response for the smoking questions of 5.2% in all census respondents.[12] Percentages of current smokers, ex-smokers, and never-smokers in analyses presented exclude subjects who did not respond to either or both of the smoking status questions.

Smoking among doctors in 2006—The prevalence of smoking among all doctors and all those in the census with an occupational classification stratified by age and sex is shown in Table 1. Only 4.0% of male doctors and 3.0% of female doctors were regular smokers, with minor variations by age group. Smoking prevalence among doctors is substantially less than among the total employed population for males and females and all age groups. Around 90% of male doctors aged less than 25 years and of female doctors aged less than 45 years had never been regular smokers.

Table 1. Smoking prevalence among doctors in the 2006 New Zealand Census*

	Doctors				Total employed population	
	N	Regular Smoker (%)	Ex-smoker (%)	Never smoked regularly (%)	N	Regular smoker (%)
Male						
15-24	186	4.8	4.8	90.3	165,876	27.3
25-44	2,793	4.2	11.8	84.0	451,929	25.4
45-64	2,790	3.9	23.0	73.2	381,213	18.5
65+	360	2.5	44.2	52.5	50,748	9.2
All	6,132	4.0	18.5	77.4	1,049,766	22.4
Female						
15-24	300	3.0	7.0	91.0	150,579	23.4
25-44	2,445	2.7	8.8	88.5	411,699	22.1
45-64	1,263	3.6	15.9	80.8	343,110	17.4
65+	48	0.0	18.8	81.3	30,621	8.8
All	4,059	3.0	10.9	86.1	936,009	20.2

*There may be minor discrepancies in the totals within this table (and between other tables) as all the numbers were random rounded to a multiple of three as per Statistics New Zealand protocol.

Table 2 shows smoking among doctors by speciality:

- Among male doctors, the highest smoking prevalence was among obstetricians and gynaecologists (11.8%) and the lowest among anaesthetists (2%).
- Among female doctors, the highest smoking prevalence was among radiologists and radiotherapists (9.7%) and the lowest among GPs (1.9%).

Smoking among nurses in 2006—The prevalence of smoking among all nurses stratified by age and sex is shown in Table 3.

Only 13.2% of female nurses were smokers, and 19.6% of male nurses. These compare with 20.2% of females and 22.4% of males among the total employed population. Smoking among nurses was higher than in the total employed population for the male nurses aged 45–64 and >65 years, and for female nurses over 65 years. Otherwise regular smoking was less common among nurses than in the total employed population in all other age groups for both genders. The proportion of smokers was highest in male and female nurses in the 15–24 years age group. However, there was also a very high proportion of female nurses in this age group who had never smoked regularly (72%).

Table 2. Smoking prevalence among doctors by specialty in the 2006 New Zealand Census*

	N	Regular Smoker (%)	Ex-smoker (%)	Never smoked regularly (%)
Male				
General Practitioner	2,313	3.4	20.8	75.9
Resident Medical Officer	1,710	4.2	14.7	80.9
Surgeon	606	3.5	20.3	76.2
Physician	990	5.5	19.4	74.8
Gynaecologist and Obstetrician	51	11.8	23.5	64.7
Radiologist, Radiation Oncologist	171	3.5	21.1	77.2
Anaesthetist	300	2.0	16.0	81.0
Female				
General Practitioner	1,572	1.9	9.5	88.4
Resident Medical Officer	1,560	2.5	8.5	88.7
Surgeon	63	4.8	14.3	81.0
Physician	612	5.4	19.6	75.0
Gynaecologist and Obstetrician	39	0.0	7.7	92.3
Radiologist, Radiation Oncologist	93	9.7	12.9	80.6
Anaesthetist	120	2.5	15.0	82.5

* There may be minor discrepancies in the totals within this table (and between other tables) as all the numbers were random rounded to a multiple of three as per Statistics New Zealand protocol.

Table 3. Smoking prevalence among nurses in the 2006 New Zealand Census

	Nurses				Total employed population	
	N	Regular Smoker (%)	Ex-smoker (%)	Never smoked regularly (%)	N	Regular smoker (%)
Male						
15-24	63	23.8	23.8	57.1	165,876	27.3
25-44	1,176	18.6	23.2	57.7	451,929	25.4
45-64	1,068	20.8	36.2	43.3	381,213	18.5
65+	51	11.8	41.2	47.1	50,748	9.2
All	2,358	19.6	29.5	50.9	1,049,766	22.4
Female						
15-24	1,080	15.8	11.9	72.2	150,579	23.4
25-44	14,076	13.8	23.1	63.1	411,699	22.1
45-64	15,558	12.6	30.4	57.0	343,110	17.4
65+	837	9.7	33.0	57.0	30,621	8.8
All	31,548	13.2	26.6	60.2	936,009	20.2

* There may be minor discrepancies in the totals within this tables (and between other tables) as all the numbers were random rounded to a multiple of three as per Statistics New Zealand protocol.

Table 4 shows smoking among nurses by speciality. Smoking prevalence was higher among male "principal" and psychiatric nurses. Among female nurses, smoking prevalence was 15% or lower for all groups of nurses, except for psychiatric nurses who had a particularly high prevalence (30%). Male and female psychiatric nurses had higher smoking prevalences than the overall male and female census population.

The lowest smoking prevalences (<10%) were among midwives and Plunket nurses (the latter being community-based nurses who focus particularly on child health and development). Midwives and Plunket nurses provide smoking cessation support and advice about smokefree homes, so are potentially key smokefree role models within communities.

Table 4. Smoking prevalence among nurses by specialty* in the 2006 New Zealand Census #

	N	Regular Smoker (%)	Ex-smoker (%)	Never smoked regularly (%)
Male				
Principal Nurse	111	21.6	27.0	54.1
Registered Nurse	1,632	17.6	28.7	53.9
Psychiatric Nurse	525	26.3	33.7	40.6
Other male nurse	81	15.4	26.9	57.7
Female				
Principal Nurse	1293	15.3	29.9	55.0
Registered Nurse	25,011	12.8	25.9	61.3
Psychiatric Nurse	1,140	30.0	29.7	40.0
Plunket Nurse	474	9.5	27.8	62.0
Public Health and District Nurse	1,236	10.9	31.1	58.0
Occupational Health Nurse	174	13.8	31.0	55.2
Midwife	2,217	9.3	28.0	62.7

* Principal nurses are senior nurses including charge nurses. Registered nurses are general staff nurses working mainly in a hospital setting. See text for a description of Plunket nurses.

There may be minor discrepancies in the totals within this tables (and between other tables) as all the numbers were random rounded to a multiple of three as per Statistics New Zealand protocol.

Trends in smoking prevalence among doctors and nurses

Census data shows that regular cigarette smoking among doctors has declined steadily since 1976 (Figure 1). Smoking prevalence was only 5% by the 1996 census for male and female doctors, and by 2006 had declined to 4% and 3% respectively.

Cigarette smoking prevalence was also assessed in samples of doctors in 1963 and 1972 (2623 in 1963 and 3113 in 1972). In 1963 only occasional and regular cigarette

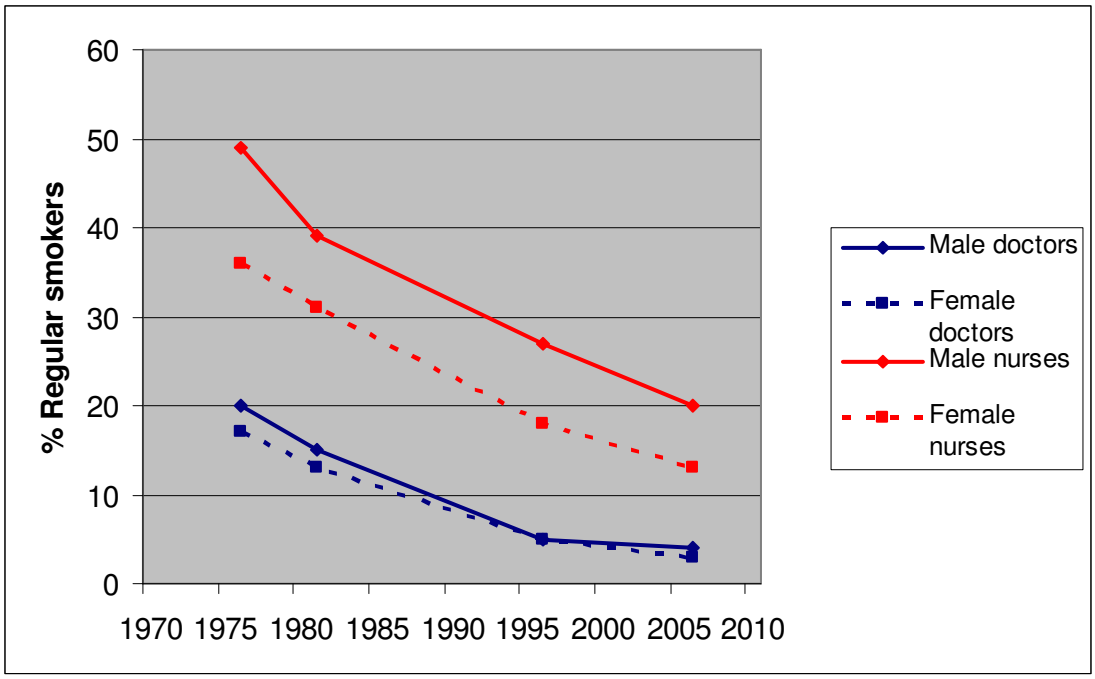
smoking combined was measured, and was 35.5% among men and 28.8% among women. [4]

By 1972 this had declined to 22.0% in men and 14.6% among women doctors. Regular cigarette smoking was 17.4% for male doctors and 12.2% among female doctors in 1972. [3]

Smoking among nurses has also decreased regularly since first assessed in 1976, and has declined further in 2006. Smoking prevalence among psychiatric nurses has remained high. In 1976 49% of female and 50% of male psychiatric nurses were regular cigarette smokers. This reduced to 46% of females and 38% males in 1981, and to 31% for males and female psychiatric nurses in 1996.

Table 4 shows that there has been little further decline among female psychiatric nurses, though male smoking prevalence has reduced to 26%.

Figure 1. Trends in regular cigarette smoking prevalence among doctors and nurses in New Zealand from New Zealand census data



Sources: 1976, 1981, 1996, and 2006 censuses.[5–8]

Discussion

The results from the 2006 Census demonstrate that non-smoking among doctors and nurses is increasingly the norm. The vast majority of doctors are now non-smokers, and among younger doctors, around 90% have never been regular smokers. Among nurses there has been a steady decline, so that female nurses now have a smoking prevalence well below the population level, in contrast to the situation in 1976 and 1981.[5,7] Male nurses formerly had a very high smoking prevalence. This too has also

decreased and is now slightly below overall male smoking prevalence in New Zealand. If recent trends continue, smoking will soon also be a rarity among nurses.

A major strength of this study is that uniquely it is based on a census rather than a sample of health care workers. A potential limitation is that there was a proportion who did not complete the smoking status question (less than 5% in the occupational groups included in this study). It is plausible that these individuals may be more likely to be smokers (and not report this due to social desirability bias) and hence the smoking prevalence figures may be a slight underestimate. However, given the low non-response rate this is unlikely to greatly affect the findings. Another limitation is that even the level 5 occupational categories are aggregations of a broad range of types of doctors and nurses, so a more finely focused assessment of smoking status by particular specialties was not possible.

In the international review of smoking among doctors, from 15 studies which have been published since 2000, smoking prevalences varied between 2% and 40% in all doctors, 5% to 32% among male doctors, and 0 to 23% among female doctors.[1] Smoking prevalence among New Zealand doctors is therefore probably one of the lowest in the world. Among studies of nurses published between 1996 and 2006, the mean smoking prevalence was 20%.[2] Since this prevalence was largely among female nurses, this shows that the New Zealand smoking prevalence for female nurses of 13% is also lower than in most other countries where surveys have been performed.

These figures are encouraging as health professionals, particularly doctors and nurses are likely to be important role models to the rest of the community for health-related behaviours. These health workers are also important for delivering smoking cessation services and the credibility of this service delivery may be undermined if the provider is known to be a smoker.

The persisting high smoking prevalence among psychiatric nurses is of concern, and has been reported in other countries.[14-17] It identifies a high risk group who may particularly benefit from targeted smoking cessation advice and support. It is also of concern because of the possible impact on smoking among patients attending mental health services, which have been shown to be very high in numerous studies around the world.[18] Further research would be useful to explore the reasons for continued high smoking prevalence in this group and to examine the effect of interventions to reduce smoking among all those working and living in mental health service settings given the important links between mental health and smoking. [19]

Nevertheless, all health workers who continue to smoke could benefit from targeted smoking cessation support using evidence-based pharmacotherapy and counselling support.[20] These can be delivered (and ideally paid for) by employing health sector agencies. This could be justified on the grounds of reducing absenteeism levels, improving work productivity and reducing the adverse role model of have smoking health professionals in healthcare settings and in the community.

In conclusion, data from the 2006 census in New Zealand confirms the very low prevalence of smoking among doctors and low and decreasing smoking prevalence among nurses. The results show that it is possible to achieve very substantial decreases in smoking prevalence and that smokefree cultures can become established and be maintained among substantial occupational groups who are well informed

about the degree of risk, are aware of the reality of the health consequences of smoking, and work in a substantially non-smoking environment. This provides hope that with strong tobacco control policies a similar smokefree culture and very low prevalence of smoking could be established much more widely in the population.

Competing interests: Wilson and Edwards have previously worked for NGOs and the Ministry of Health on tobacco control issues.

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Has the prevalence and severity of symptoms of asthma changed among children in New Zealand? ISAAC Phase Three

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Abstract

Aim To investigate time trends in prevalence of symptoms of asthma by repeating, during 2001–3 (Phase Three), the International Study of Asthma and Allergies in Childhood (ISAAC) Phase One study that was conducted in New Zealand in 1992–3.

Methods ISAAC Phase Three involved repeating the cross-sectional questionnaire survey of two age groups of school children (6–7 years and 13–14 years, children and adolescents respectively) using the same methodology as Phase One. In New Zealand it was conducted in Auckland, Bay of Plenty, Christchurch, Nelson, and Wellington.

Results After 9 years, reported asthma ever increased from 24.6% to 30.2% in children and from 24.1% to 32.4% in adolescents ($p<0.001$). Current wheeze (written questionnaire) significantly decreased in children from 23.6% to 22.2% ($p=0.002$) and in adolescents from 29.7% to 26.7% ($p=0.047$), and for the video questionnaire from 18.1% to 11.1% ($p<0.001$). There was a significant reduction in wheezing limiting speech from 5.0% to 3.7% in children, and 7.9% to 6.2% in adolescents. Little regional variation was found. A higher proportion of children with asthma symptoms now report having ever had asthma.

Conclusions The decrease in prevalence and severity of symptoms of asthma is encouraging, but the reasons for these trends are currently unclear. Increases in asthma labelling are likely to be due to greater awareness of asthma. A trend of decreasing prevalence of asthma symptoms, if maintained, has positive implications for lessened burden of disease among asthmatics and lowered cost of treatment.

Asthma has been a national concern in New Zealand since the late 1970s when an asthma mortality epidemic was identified.[1] At the same time, admissions to hospital for asthma increased.[2] Since that time several studies in New Zealand children have shown an increase in asthma symptom prevalence during the period 1969-2000.[3-5]

The International Study of Asthma and Allergies in Childhood (ISAAC) Phase One found a high burden of asthma symptoms in New Zealand children in 1992–3 compared with most other countries,[6] and we hypothesised that the prevalence of asthma symptoms would continue to increase.

ISAAC has completed a worldwide study of the time trends of asthma and allergies in which New Zealand data is included.[7] The time trends in asthma symptoms in the New Zealand ISAAC study are reported in more detail here. New Zealand time trends data for symptoms of rhinoconjunctivitis and eczema will be presented elsewhere.

The objective of this study was to assess recent time trends in asthma symptom prevalence in New Zealand using standardised methodology.

Methods

The details of the study design and methods for ISAAC Phase Three are described in detail elsewhere.[8] ISAAC Phase Three was conducted in 2001–3 in five centres in New Zealand which had participated in ISAAC Phase One: Auckland, Bay of Plenty, Christchurch, Nelson, and Wellington.

Each centre conducted Phase Three in the same way as Phase One to ensure comparable data. The study centre investigators chose the schools either by new random samples of schools in that area or by going back to the same schools chosen at random in Phase One. The two age groups of children selected were 6–7 and 13–14 year olds, here called children and adolescents respectively.

The older age group completed written questionnaires on asthma, rhinitis, and eczema symptoms at school, and also completed an asthma symptoms video questionnaire. The younger age group took the questionnaire home for parental completion. The key question used for assessing asthma symptom prevalence for both age groups ('current wheeze') was: *'Have you (Has your child) had wheezing or whistling in the chest in the past 12 months?'*.

The severity of asthma symptoms was assessed by three questions that asked about the following symptoms in the past 12 months: number of attacks of wheezing; sleep disturbed due to wheezing; and wheezing severe enough to limit speech to only one or two words at a time between breaths. The video questionnaire showed five scenes of young people with asthma symptoms; wheezing at rest, wheezing with exercise, waking with wheeze, waking with cough, and a severe attack of asthma, and asked if they had experienced these symptoms at any time in their life, if *yes* in the past year, if *yes* one or more times a month.

In Phase Three the core questions were followed by an additional environmental risk factor questionnaire.[8] Methodology and data from each centre were examined for adherence to protocol, and comparability in methodology between Phases 1 and 3. Ethics Committee approval was obtained for each centre, and centres obtained their own funding.

As in Phase One, the two age groups were analysed separately. Symptom prevalence values in each centre were calculated by dividing the number of positive responses to each question by the number of completed questionnaires for the written and video questionnaires separately. For each centre, the annual change in symptom prevalence was calculated by taking the difference between the Phase One and Phase Three prevalence values and dividing by the number of years between the two surveys.

An estimate of the absolute rate of change per year in asthma symptoms was derived for each centre and also the standard error of the change per year (SE), adjusted for the effect of cluster sampling[9] from which significant changes ≥2 SE up or down could be derived. The other assessments of change with time were made using a generalised mixed model with a logit link and a binomial error distribution, and modelling the schools as a random effect.

Other factors in the model were gender, ethnicity, school decile, and month of interview as our previous work has shown that a higher (non-statistically significant) rate of positive responses for asthma symptoms were found among adolescents responding in winter months.[10]

Results

The ISAAC Phase Three study was completed to the standards of the ISAAC protocol[8] in all five centres in children and adolescents. However because Wellington had a low response rate (47.2%) in children, this centre was excluded from the analyses for this age group. For the centres included in the analyses, there were 10,873 children (response rate 85.2%) and 13,317 adolescents (response rate 89.2%) (Table 1).

Phase One was conducted in 1992–3 and Phase Three in 2001–3. Thus, the time period between the phases averaged 9 years (range 8–10 years). As for Phase One, Auckland, Wellington, and Christchurch collected the data over 1 year, and in Bay of

Plenty and Nelson over one school term.[6] The later starting times for data collection in two centres were due to delays in obtaining ethical approval where ethics committees originally favoured written consent, but finally approved passive consent so as to be consistent with Phase One methodology.

Table 1. Phase One year of study and number of participants (N), and Phase Three year of study, number of participants and response rate (%)

Centre	6–7 year age group				
	Phase One		Phase Three		
	Year	N	Year	N	Response
Auckland	1993	3526	2002	3541	84.6
Bay of Plenty	1993	2681	2002	2150	79.9
Nelson	1993	1868	2003	1867	92.0
Christchurch	1993	3318	2003	3315	86.0
Total	1993	11393	2003	10873	85.2
Centre	**13–14 year age group**				
Auckland	1993	3206	2001	2870	92.3
Bay of Plenty	1993	2813	2002	1976	76.2
Wellington	1993	4417	2001	3050	96.9
Nelson	1993	1838	2003	2305	90.5
Christchurch	1993	3186	2003	3116	88.2
Total	1993	15460	2002	13317	89.2

Table 2 shows the findings for changes in asthma symptom prevalence between Phase One and Phase Three. When these results were adjusted for gender, ethnicity, school decile, and month of interview, lifetime asthma (asthma ever) significantly increased from 26.3% to 31.9% ($p<0.001$) in children and 22.8% to 31.3% ($p<0.001$) in adolescents. However there was a completely different picture for reported symptoms in the last 12 months, which generally did not change or even decreased (Figures 1 and 2).

Table 2. Asthma symptoms Phase Three prevalence (%), change per year (%), and standard error (SE, %)

Symptom		Auckland	Bay of Plenty	Wellington	Nelson	Christchurch	Total
		6–7 year age group					
Current wheeze	Phase One	22.5	24.0		18.7	27.2	23.6
	Phase Three	22.4	23.7		20.2	22.3	22.2
	Change per year	-0.02	-0.04		0.15	-0.49*	-0.11
	SE	0.12	0.15		0.13	0.13	0.07
4 or more attacks of wheeze	Phase One	7.9	8.2		7.1	10.2	8.5
	Phase Three	7.4	6.8		6.4	7.8	7.2
	Change per year	-0.05	-0.15		-0.07	-0.24*	-0.13*
	SE	0.07	0.08		0.08	0.08	0.04
Sleep disturbance	Phase One	3.7	3.6		2.2	3.9	3.5
	Phase Three	3.8	3.8		2.4	2.6	3.2
	Change per year	0.01	0.03		0.01	-0.13*	-0.04
	SE	0.07	0.07		0.05	0.05	0.03
Wheeze affecting speech	Phase One	5.3	4.8		4.6	5.0	5.0
	Phase Three	3.8	4.4		3.1	3.6	3.7
	Change per year	-0.17*	-0.04		-0.15*	-0.14*	-0.13*
	SE	0.07	0.08		0.06	0.06	0.03

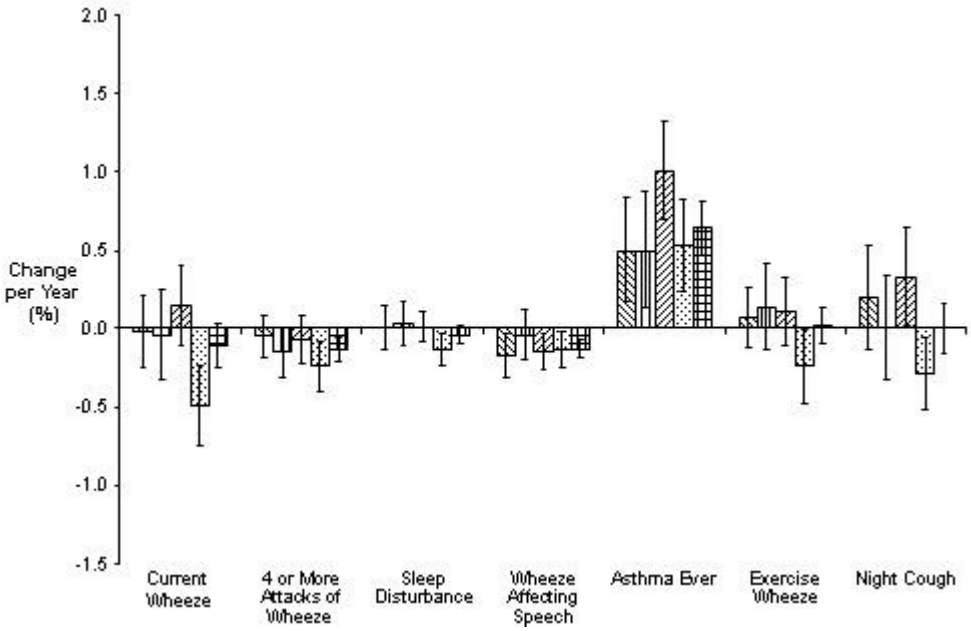
		Auckland	Bay of Plenty	Wellington	Nelson	Christchurch	Total
Asthma ever	Phase One	23.8	25.7		17.6	28.4	24.6
	Phase Three	28.3	30.2		27.7	33.6	30.2
	Change per year	0.50*	0.50*		1.01*	0.53*	0.64*
	SE	0.17	0.19		0.16	0.15	0.09
Exercise wheeze	Phase One	14.8	15.9		13.2	19.2	16.1
	Phase Three	15.4	17.1		14.2	16.8	16.0
	Change per year	0.07	0.14		0.11	-0.24*	0.02
	SE	0.10	0.14		0.11	0.12	0.06
Night cough	Phase One	27.5	28.9		21.1	32.2	28.1
	Phase Three	29.3	29.0		24.4	29.2	28.4
	Change per year	0.20	0.01		0.33*	-0.29*	0.00
	SE	0.17	0.17		0.16	0.12	0.08
		13–14 year age group					
Current wheeze	Phase One	26.5	29.5	31.7	31.0	29.6	29.7
	Phase Three	22.5	20.6	32.6	28.0	27.9	26.7
	Change per year	-0.51	-0.98*	0.11	-0.29	-0.17	-0.39*
	SE	0.34	0.21	0.25	0.19	0.23	0.13
4 or more attacks of wheeze	Phase One	8.0	9.0	11.1	10.2	9.8	9.7
	Phase Three	4.9	4.4	7.8	6.6	6.7	6.2
	Change per year	-0.38*	-0.51*	-0.41*	-0.35*	-0.31*	-0.38*
	SE	0.12	0.10	0.13	0.14	0.07	0.06
Sleep disturbance	Phase One	2.7	3.3	3.0	2.6	2.9	2.9
	Phase Three	2.9	2.3	3.7	1.7	2.2	2.6
	Change per year	0.02	-0.11	0.08	-0.09	-0.07	-0.05
	SE	0.07	0.07	0.08	0.05	0.07	0.03
Wheeze affecting speech	Phase One	8.1	7.1	8.3	8.2	7.5	7.9
	Phase Three	5.6	4.1	8.1	6.3	6.2	6.2
	Change per year	-0.31*	-0.33*	-0.03	-0.19	-0.13	-0.21*
	SE	0.12	0.09	0.14	0.11	0.08	0.05
Asthma ever	Phase One	22.9	22.3	26.4	20.2	25.9	24.1
	Phase Three	27.9	28.3	36.3	29.4	37.6	32.4
	Change Per Year	0.63*	0.67*	1.24*	0.91*	1.17*	0.93*
	SE	0.26	0.14	0.17	0.15	0.19	0.11
Exercise wheeze	Phase One	36.1	39.4	41.2	43.4	40.3	39.9
	Phase Three	32.4	31.6	42.5	41.6	37.8	37.5
	Change per year	-0.45	-0.86*	0.17	-0.17	-0.25	-0.29*
	SE	0.30	0.32	0.31	0.25	0.22	0.14
Night cough	Phase One	29.7	31.3	30.3	26.3	27.4	29.3
	Phase Three	30.8	26.9	31.5	27.6	26.8	28.9
	Change per year	0.14	-0.49	0.15	0.13	-0.06	-0.01
	SE	0.26	0.28	0.26	0.28	0.32	0.13
Current wheeze (Video)	Phase One	16.3	18.6	19.5	19.1	17.4	18.2
	Phase Three	11.2	13.4	12.1	11.5	8.8	11.2
	Change per year	-0.64*	-0.57*	-0.93*	-0.76*	-0.86*	-0.76*
	SE	0.14	0.15	0.16	0.16	0.11	0.07
Exercise wheeze (video)	Phase One	28.4	28.4	31.1	32.4	32.2	30.4
	Phase Three	15.9	13.5	17.1	15.9	16.2	15.9
	Change per year	-1.56*	-1.66*	-1.75*	-1.64*	-1.60*	-1.64*
	SE	0.23	0.28	0.23	0.32	0.20	0.11
Sleep disturbance (video)	Phase One	11.3	11.4	12.2	10.5	11.3	11.5
	Phase Three	5.1	6.7	5.4	3.9	4.6	5.1
	Change per year	-0.79*	-0.53*	-0.86*	-0.65*	-0.67*	-0.73*
	SE	0.10	0.14	0.08	0.08	0.12	0.05
Night cough (video)	Phase One	20.7	25.2	23.1	23.3	22.5	22.9
	Phase Three	20.0	17.2	22.5	18.4	17.9	19.4
	Change per year	-0.09	-0.88*	-0.08	-0.49*	-0.46	-0.32*
	SE	0.26	0.40	0.22	0.23	0.30	0.13

		Auckland	Bay of Plenty	Wellington	Nelson	Christchurch	Total
Severe wheeze (video)	Phase One	11.4	12.8	14.9	11.7	13.2	13.0
	Phase Three	6.3	8.3	6.8	9.4	7.1	7.4
	Change per year	-0.63*	-0.50*	-1.01*	-0.23*	-0.61*	-0.56*
	SE	0.14	0.13	0.14	0.11	0.10	0.06

*Change ≥2 standard errors.

Current wheeze reported with the written questionnaire in Phase Three significantly decreased from Phase One in children from 25.7% to 23.5% (p=0.002) and in adolescents from 28.3% to 26.3% (p=0.047). For the video questionnaire, current wheeze significantly decreased from Phase One from 20.5% to 12.4% (p<0.001), and there was a decrease ≥2SE for all symptoms in all centres except night cough in Auckland and Wellington where no change was seen (Figure 3).

Figure 1. Change per year in prevalence of symptoms for the 6-7 year age group for Auckland, Bay of Plenty, Nelson, Christchurch, and Total. The vertical bars indicate 95% confidence intervals.



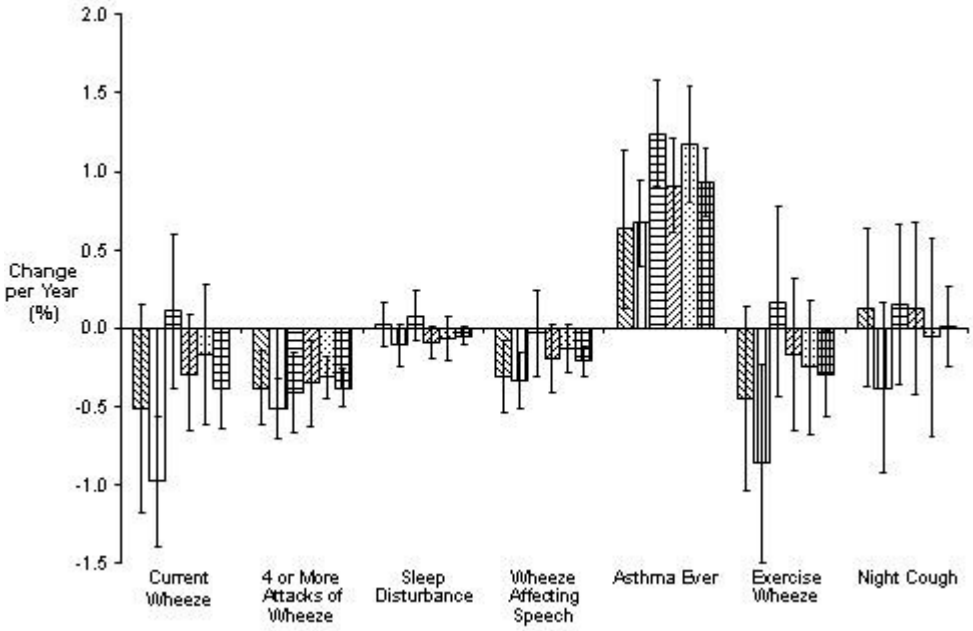
There was a reduction in symptoms of severe asthma in the last 12 months for both age groups: for wheezing limiting speech there was a decrease from 6.4% to 4.7% (p<0.001) in children and a decrease from 8.4% to 6.5% (p<0.001) in adolescents. For 4 or more attacks there was a decrease in adolescents from 7.5% to 4.8% (p<0.001), but the reductions in 4 or more attacks for children, and wheezing disturbing sleep in both age groups were non-significant.

The prevalence of current wheezing in ISAAC Phase Three was more common in boys (50.4%) than girls (49.6%) of the 6–7 year age group (p<0.001), but more

common in girls (52.1%) than boys (47.9%) of the 13–14 year age group (p<0.001), similar to the observations in Phase One.

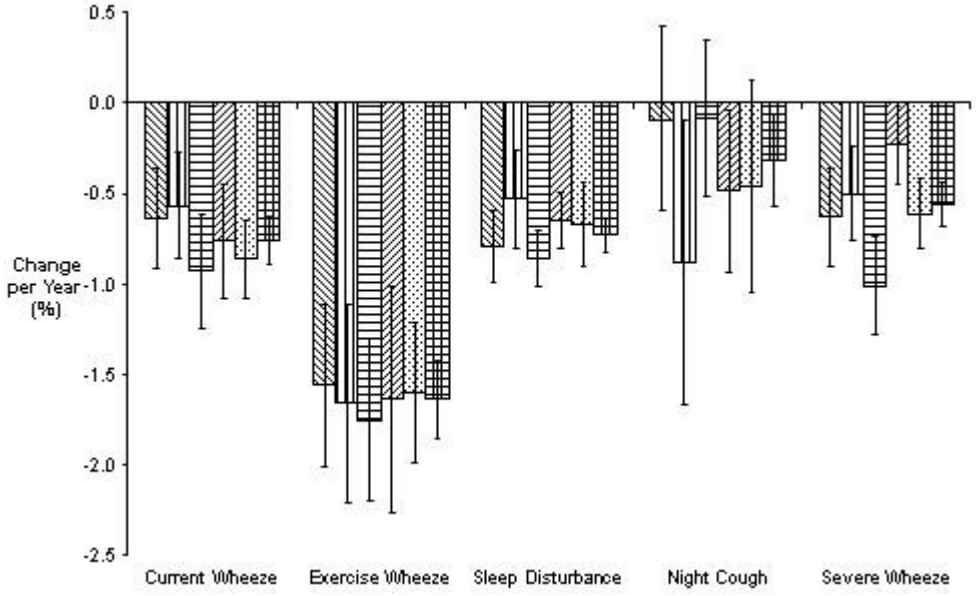
Among children with current wheeze, reported asthma ever increased from 70.4% to 77.3%, a change of 7.0% (95%CI 6.70–7.26%) and among adolescents from 54.4% to 63.7%, a change of 9.3% (95%CI 9.00–9.65%). However of those with reported asthma ever, the proportion with current wheezing fell from 67.6% to 56.9% and from 67.1% to 52.5% in children and adolescents respectively.

Figure 2. Change per year in prevalence of symptoms (written questionnaire) for the 13-14 year age group for Auckland, Bay of Plenty, Wellington, Nelson, Christchurch, and Total. The vertical bars indicate 95% confidence intervals.



Among children, the relative risk of current wheeze in those with atopy (allergic rhinoconjunctivitis or eczema) was 2.52 (95%CI 2.22–2.87) and among adolescents was 2.13 (95%CI 2.06–2.21). Among those with atopy the proportion with current wheezing did not change between phases (data not shown).

Figure 3. Change per year in prevalence of symptoms (video questionnaire) for the 13-14 year age group Auckland, Bay of Plenty, Wellington, Nelson, Christchurch, and Total. The vertical bars indicate 95% confidence intervals.



Conclusions

This is the first multicentre study of time trends of the prevalence of asthma symptoms within New Zealand, and the only study examining trends beyond 2000. In contrast with reports of an increase in the prevalence of asthma symptoms from the 1960s to 2000,[4,5,11,12] we found no evidence of any increase in symptoms from 1993 to 2002. In both age groups, there were significant decreases in current wheezing, wheeze limiting speech, and for the adolescent group the video scenes of asthma symptoms. The prevalence of symptoms reported with the video questionnaire were lower than from the written questionnaire, similar to the findings from ISAAC Phase One.[13] The video questionnaire is likely to detect more severe symptoms due to the visual and auditory nature of the signs.

Despite the fact that the prevalence values of most current symptoms including symptoms of severe asthma decreased, reported asthma ever increased in both age groups. This could be due to several factors, including a greater awareness of asthma by parents and adolescents, a greater use of the diagnostic label by doctors, or better asthma control where more children with asthma have gained complete symptom control. Unfortunately information on individual medication use was not collected in this study to explore this further.

In the 1980s several studies in the UK concluded that asthma was being significantly under diagnosed. At the same time, concerns about rising asthma prevalence, increased hospital admissions for asthma and asthma mortality resulted in a push for increased recognition and treatment of asthma in developed countries. The findings

presented above suggest that the trends established in the 1980s to increase the diagnosis of asthma in those with symptoms of asthma has continued into the new millennium so that fewer children with wheeze are not diagnosed with asthma.

There may be an increase in the labelling of asthma within preschool children who have viral-induced wheeze which has a good prognosis and which does not progress to the classical asthma phenotype in school age children or mislabelling of asthma earlier in life. There is no evidence that preventative treatment of asthma symptoms in preschool children improves the prognosis of asthma.[14,15]

There was no evidence that the relationship between asthma and atopic disease is changing—the proportion of children with current wheeze reporting other atopic disease did not change between phases. The higher prevalence of current wheeze among boys in the younger age group and among adolescent girls is consistent with earlier reports.[13]

The strengths of this study are the inclusion of several New Zealand centres, three in the North Island and two in the South Island, and the ability to make valid comparisons with the rest of the world due to the standardised methodology used. The numbers of subjects and response rates of the centres included in the analyses are high, and centres completed rigorous data and methodology checks. The time interval (8–10 years) was similar to previous New Zealand studies where increases over time have been found.[3–5,16]

The 6–7 year age group in Wellington was excluded from the analyses to avoid possible bias due to a low response rate. In the Bay of Plenty, lower participation rates were found for both age groups compared with other centres for Phase Three, and a slightly lower response rate for the 6–7 year age group for Phase One, but within the allowable range. In general, most centres reported less enthusiasm from schools to participate in the Phase Three study than was experienced in Phase One because of curriculum pressures (especially for the secondary schools), the change to a four term year, as well as difficulties in interpreting the Privacy Act. These factors may have contributed to the lower response rate (85%, 89% in children and adolescents respectively) than that achieved in Phase One (91%, 93%).

There are limitations of the study which should be considered. Symptom prevalence has been examined at two time points only, so estimates of mean yearly changes during the time period of the study cannot be interpreted with confidence as a consistent linear change. There is the possibility of recall bias for wheezing ever and asthma ever. This may be worse for adolescents who may not recall events in early life. Also a diagnosis of asthma may have been made and later retracted, but still reported by parents or adolescents as ‘asthma ever’.

There was a real concern that the study would not be able to be completed in two centres due to ethics committees favouring active written consent, rather than the passive consent used in Phase One. In the event, approval for passive consent was granted, but the commencement of the study was delayed in those centres. Active consent is very likely to result in lower response rates and importantly the Phase One protocol would not have been duplicated.[17]

In December 2006 the New Zealand National Ethics Advisory Committee developed guidelines on conducting observational studies in an ethical manner that are intended

to facilitate high quality studies, protect the interests of participants, and underpin public assurance of good study conduct.[18] These guidelines allow passive consent to be the model of choice for observational studies such as ISAAC.

Possible reasons for the observed decrease in symptom prevalence after a period of increase include a decrease in intensity of an aggravating environmental factor or a protective environmental/management factor. There may also be improved management of individuals with asthma. For example, a growing number of studies suggest that ingestion of antioxidants may be associated with fewer asthma symptoms,[19,20] and health messages from education programmes targeted at the prevention of heart disease may have also influenced the prevalence of asthma. However, there is currently no evidence that New Zealand children are in fact eating more healthily; rather there is increased concern about unhealthy diets.

Obesity is linked with wheezing,[21] but evidence points to an increase in obesity in New Zealand children,[22] rather than a decrease which might accompany a decrease in reported asthma symptoms. Could there be an increase in prevalence which is eventually limited by the genetic potential for asthma in the population? Or a cohort effect where a historical event has increased prevalence within a group born during a given period, and that exposure has now stopped? Could the increase, and now decrease be influenced by perception of symptoms due to a decrease in awareness programmes?

Hospital admissions relate mostly to more severe asthma, and from 1993 to 2002 there was no change in hospital admissions due to asthma among children (3.6 per 1000) but among adolescents admissions nearly halved (2.4 to 1.3 per 1000) (New Zealand Health Information Service). Decreased severity of asthma is often attributed to wider use of inhaled corticosteroids (ICS). Data on dispensing of ICS were available only for the whole age group (6-17 years) from 1993 to 2001 and showed that total adjusted amount of ICS was stable during this period.[23] There are no data available by centre and age group, or on usage by individuals.

Long acting β-agonists usage is unlikely to have had an impact on asthma prevalence reported from symptoms because since their funding in 1997 they were accessible only to the few children on high dose ICS ≥800 μgBDP equivalents/day. While the increased use of effective treatment, especially ICS, may be important in reducing the severity of episodes, it is unlikely to explain the decrease in mild wheeze symptoms.[24]

From 1993 to 2002 the number of tonnes of tobacco released in New Zealand was stable (767.0, 771.2 respectively), but the number of cigarettes sold fell from 3.5 to 2.7 million (NZ Customs Service, New Zealand Overseas Trade Statistics 2007); the number of cigarette equivalents sold as tobacco is not measured. While it is tempting to speculate that a reduction in cigarette smoking may have resulted in a reduction in asthma symptoms, this is implausible as there was an increase in prevalence of asthma symptoms seen in earlier decades at the same time as cigarette smoking was falling. There has also been an increased attendance at childcare facilities, which has been associated with a reduced risk of developing asthma.[25]

It is also highly unlikely that any genetic influences, sensitisation to environmental allergens and respiratory syncytial virus infection in early life changed over that period, and thus these potential explanations remain highly speculative.

The prevalence of symptoms of asthma remains high by international standards, with New Zealand ranked in the top five countries among ISAAC Phase Three time trend centres,[7,26] with levels similar to the levels in Australia, the United Kingdom, Ireland, Canada, and the United States. The prevalence of current symptoms has decreased not only in New Zealand but also in Australia, the United Kingdom, and parts of Western Europe.[24,26,27] However symptom prevalence has increased in other parts of the world, including Latin America and parts of Europe, and especially in low prevalence centres so that the differences between the English-speaking countries and the rest of the world have lessened.

To summarise, the prevalence of asthma symptoms in New Zealand is mainly decreasing which is good news. The explanation for this trend is unknown, but it is likely to be due to a combination of factors, including changes in unknown environmental causes of current asthma symptoms, or changes in asthma awareness or asthma treatment.

A trend of decreasing prevalence of asthma symptoms, if maintained, has positive implications for lessened burden of disease among asthmatics and lowered cost of treatment.

Competing interests: None known.

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Are antibiotics indicated as an initial treatment for patients with acute upper respiratory tract infections? A review

Bruce Arroll, Tim Kenealy, Karen Falloon

Abstract

Aim To determine the effect of antibiotic treatment versus placebo in patients with acute upper respiratory tract infections.

Methods A systematic review and meta-analysis examined the effect of antibiotics versus placebo in randomised controlled trials to initially treat acute upper respiratory tract infections.

Results. Eight studies of children from age 6 months and adults up to the age of 49 years were found. The main outcome measures were general improvement and adverse effects. No benefit was demonstrated in terms of overall improvement from the use of antibiotics compared to placebo for patients with acute upper respiratory tract infections RR=0.89 (95%CI 0.77–1.04). There was also a significant increase in adverse effects (mainly gastrointestinal) using a random effects model with a relative risk RR=2.71 (95%CI 1.08–6.83).

Conclusions Initial use of antibiotics do not benefit patients with acute upper respiratory tract infections and are associated with an increase in adverse effects.

Upper respiratory tract infections (URTIs, syn common cold) are the most common acute illness worldwide;[1,2] it is estimated that adults have 2 to 3 colds per year. They also represent one of the most common causes of antimicrobial use and a frequent reason for prescribing antibiotics in ambulatory practice and primary care.[2,3]

In this setting, antibiotics are considered to be overprescribed,[4] broad-spectrum varieties tend to be overused,[5] and primary care physicians (in some jurisdictions) treat over half of these colds with antibiotics.[6]

Despite this frequent antibiotic usage, the vast majority of URTIs have a viral aetiology. The most common virus is rhinovirus when symptoms are mild and influenza or parainfluenza viruses when symptoms are more severe, particularly when accompanied by muscle aches and fatigue.[7] Most patients tend to recover spontaneously in 1–2 weeks without the need for antibiotics. However, there is a small risk of serious complications (e.g. otitis media) with all the upper respiratory tract infections and this in part influences clinicians in their desire to prescribe antibiotics.[8]

Patients' perceived expectations and other non-clinical factors can also influence the decision to prescribe antibiotics.[9]

Due to the fact that URTIs are such a prevalent condition worldwide, it is important to obtain an estimate of the effectiveness of antibiotics. If they are ineffective there is a concern that the widespread use of antibiotics is not only a poor utilisation of health

resources but also that they cause adverse effects and hasten development of resistant strains of bacteria.[10,11]

A recent study out of Europe has warned that with current prescribing practices we risk losing the miracle drugs of the 20th Century.[12] The study investigated outpatient antibiotic use in 26 countries in Europe and demonstrated correlations which showed that resistance to microorganisms (*S. pneumoniae, S. pyogenes,* and *E. coli*) increased with consumption of antibiotics. It also noted that in most countries there was a growing use of the newer (i.e. broad-spectrum) antibiotics, such as combination amoxicillin and clavulanic acid, the new macrolides and quinolones.

A subsequent study looked into this relation further by investigating two macrolides—azithromycin and clarithromycin—which are among the medications commonly used in respiratory tract infections.[13] Their results showed that (inappropriate) macrolide use is the single most important driver of the emergence of macrolide resistance *in vivo*.

This systematic review aims ascertain the effectiveness of antibiotics as the initial treatment for patients with upper respiratory tract infections. In order to assess the benefits and harm of antibiotics in upper respiratory tract infections we systematically examined the antibiotic versus placebo literature and conducted a meta-analysis in terms of the outcomes of general improvement and adverse effects.

Methods

Literature search

A Medline search covering the years 1966 to January 2007 was undertaken using the following MeSH terms: common cold, respiratory tract infections (upper), random allocation, double blind method, or single blind method.

A similar search strategy was used on EMBASE and a search of the Cochrane Controlled Trials Register. The Family Medicine Database was searched through the Canadian College of Family Physicians Library in London Ontario including a search on FAMLI Vol. 1, 1980 to Vol. 13, 1993 (this database was discontinued in 1993). We also searched the reference lists of relevant trials, review articles, and textbook chapters. Authors of the studies finally retrieved by the above method were approached in writing to enquire about unpublished or unretrieved articles.

Other investigators have approached the British Pharmaceutical industry for papers on respiratory tract infections but received no unpublished material (T Fahey, personal communication).

Disease definition

The International Classification of Health Problems in Primary Care (ICHPPC-2) defines a URTI as an illness with evidence of acute inflammation of nasal or pharyngeal mucosa and the absence of other specifically defined respiratory infections e.g. streptococcal tonsillitis, laryngitis, bronchitis, pneumonia, asthma, and hayfever.[14]

Lower respiratory tract signs were accepted in patients with the above symptoms so long as the majority of patients in the study did not have these signs and that pneumonia was ruled out. The reviewers recognised that there would be some undetermined overlap with the alternative diagnoses of bronchitis and pharyngitis. However, this was not necessarily a disadvantage as it reflected the real life situation faced by practitioners making decisions on the use of antibiotics.

Criteria for including studies for this review

All randomised controlled trials of antibiotic versus placebo as initial treatment for acute upper respiratory tract infection.

Studies were excluded:

- If they involved the use of an active substance instead of a placebo (e.g. aspirin) as these substances may exert a beneficial effect thereby nullifying any beneficial effect of the antibiotic
- If antibiotics were given prophylactically
- If more than seven percent of participants had throat swabs positive for beta haemolytic streptococcal infection (This cutoff was chosen to distinguish this review from the review on sore throat by Del Mar which had 8% streptococcal culture rate as the lowest rate).[15]
- If there was concern about the process of randomisation
- If there was a known bacterial diagnosis at the time of treatment initiation
- If patients had a past history of serious illness
- If most of the patients had been given the diagnosis of bronchitis
- If patients had more than 7 days of symptoms at the time of study entry.

Statistical analysis

We used Cochrane review manager v4.2 software for the analysis. A fixed effects method was used for all analyses except for side-effects. Here a random effects model was used as a Chi squared test indicated heterogeneity of the findings.[16]

Results

Eight trials were eligible for inclusion in our review.[17–24] All eight studies were double blind evaluations comparing antibiotic with placebo.

The proportion of drop outs ranged from 0% in the study by Hoagland et al (1950) to 6.5% in the Kaiser et al study (1996).

Six of the studies provided a global measure of improvement form day 1 to day 7 from onset of treatment. These are summarised in Figure 1, which shows a non-significant benefit from antibiotics compared with placebo, with a relative risk of 0.89 (95%CI 0.77–1.04).

Two studies did not contribute to this analysis.[17,19] The study by Gordon et al (1974) expressed their results as p values with placebo being better at relief of symptoms than ampicillin (p=0.05) and no significant difference for placebo versus erythromycin nor placebo versus penicillin. The study by Howie and Clark (1970) found no significant benefit for a number of outcomes including illnesses with no purulent spit, with purulent spit of 10 days or more, cough, spit and purulent nasal discharge.[19]

Only two of the studies had outcomes as measured by the effects of antibiotics on specific symptoms.[19,22] Unfortunately the denominator for one of these was based on episodes of illness rather than individual patients which did not allow the data to be added to the other studies.[19] This study by Howie and Clark did analyse side effects by individual and hence this data could be used in the combined analysis.

Adverse effects

Four studies reported adverse effects and the summary relative risk was significantly increased 2.71 (95%CI 1.08–6.83) (Figure 2); a random effects model was used due to heterogeneity. The adverse effects in the adult patient studies was 4.06 (95%CI 2.34–

7.04) with no significant heterogeneity using the fixed effects model. It would appear that mixing the child and adult results created the heterogeneity.

Figure 1 Persisting Symptoms

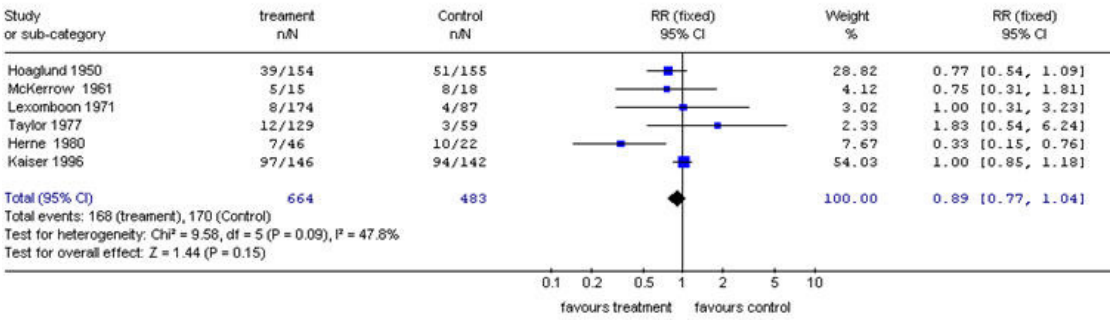
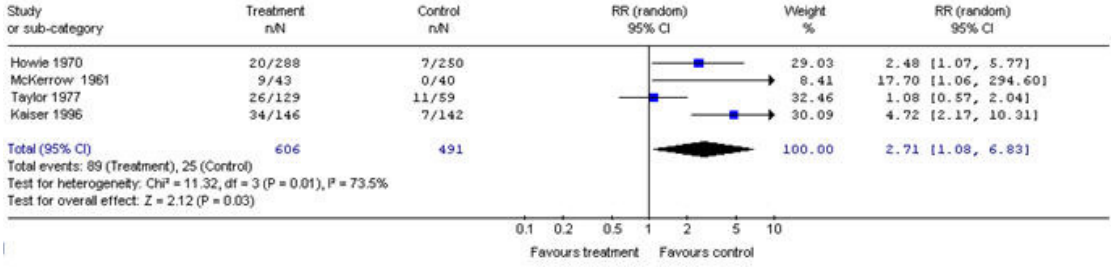


Figure 2 Adverse effects in adults and children



Possible benefit from antibiotics in subgroups

One study in this review found a significant benefit for antibiotics in the subset of patients (20% of whole group) who had positive nasopharyngeal aspirates for three respiratory pathogens *H. influenzae, M. catarrhalis,* and *S. pneumoniae*; p=0.001.[20] Another study that found an overall benefit from antibiotics may have had a large number of patients with streptococcal tonsillitis but the presentation of the findings made this difficult to determine.[24]

Discussion

The results of this meta-analysis show that there is no benefit in terms of overall improvement in prescribing antibiotics as an initial treatment for patients with upper respiratory tract infections. The number of well conducted studies is small for such a common condition. The suggestion that there may be some benefit in terms of resolution of nasal discharge has been reviewed elsewhere.[25]

Antibiotics were associated with more than twice the risk of side effects than were placebos. This was similar to the side effects in the meta-analysis of antibiotics versus placebo for children with otitis media.[26] There are some possible explanations for the heterogeneity in the side effects data. The two studies that were statistically significant were conducted in adults.[19,20] The duration of treatment was identical while there were some differences in the antibiotics.

The Howie and Clark (1970) paper reported on demethylchlortetracycline, the Kaiser et al (1996) paper reported on amoxycillin with clavulanic acid, while the Taylor et al paper (1977) in children[22] reported on cotrimoxazole and amoxycillin. The side effect rate for the cotrimoxazole was 24% which was higher than the 15% for amoxycillin while the placebo rate was 19%. The fact that the side effect rate for amoxycillin was lower than that for placebo may explain the heterogeneity.

There is no gold standard definition of upper respiratory tract infection and the diagnosis is made on clinical grounds. Although the range of inclusion criteria appear wide, we believe that the majority of patients were suffering from viral upper respiratory tract infections. In effect, we accept the clinical judgement of the doctors examining the patients from the various studies. Only two studies reported the presence of lower respiratory tract findings.

One of these studies found lower respiratory tract signs in 13% of under-2 year olds and in 13% of those over 6 years of age.[17] In the other study, 43% of patients had auscultatory evidence of more extensive peripheral airways disease.[22] We assumed that most of these signs were due to bronchospasm.

Two of the studies included in this meta-analysis may underestimate the benefit of antibiotics. One study reported findings at 24 hours which would be regarded as being too soon to show any effect for antibiotics treatment but the results have been included here for the sake of being systematic.[18] The other study used 15 mg of tetracycline or equivalent given three times daily which would be considered sub-therapeutic today.[23] However both studies were included as they met the pre-stated inclusion criteria.

We excluded studies with more than 7% of patients with streptococci found on throat swab but kept the paper by Gordon et al (1974) which had 2 of 89 patients with cultures of beta haemolytic streptococci.[17] Other studies did not test for throat bacteria. The issue of bacterial involvement is a concern for reviews of bronchitis, sore throats and upper respiratory tract infections. There is only one overlap between our study and the bronchitis Cochrane review by Fahey et al (2006)[1] and that is the study by Howie et al 1970.[2]

The sore throat review by Del Mar (1992) included the Taylor et al (1977) study that is in our review.[3,4] The role of bacteria in upper respiratory tract infections either as a causal factor or as a complication is highlighted in the study by Kaiser et al (1996). They make a case for the role of three bacteria, *H. influenzae, M. catarrhalis,* and *S. pneumoniae*, and it would be helpful to see this work repeated in another centre.

Only two of the studies were conducted in general practice.[19,22] The other studies were at a military base[18] used research clinic staff,[23] at a casualty clinic[17,20] and hospital outpatients.[21] If the assumption is made that patients can self refer to these secondary care settings and use them as primary care providers then our results are generalisable to the wider primary care setting.

Conclusions

Antibiotics appear to have no benefit in the initial treatment of acute upper respiratory tract infections. The implications for practice are that prescriptions of antibiotics

should not be given as an initial treatment as they will not affect overall improvement and carry the risk of adverse effects.

Physicians prescribing antibiotics are encouraged to consider the appropriateness of their use in order to minimise the potential ecological side effect of antibiotic-resistant organisms. Further research is needed on the role of pathogenic bacteria from nasopharyngeal aspirates in upper respiratory tract infections.

Note: A electronic version of this paper is available at the Cochrane website www.cochrane.org This will be updated over time and represent the most recent evidence on this topic.

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The development of CPR

Stuart McLennan

Abstract

Cardiopulmonary resuscitation (CPR) is now the standard treatment for someone having a cardiac arrest. It is, however, a procedure that has emerged only relatively recently. For a number of scientific and religious reasons, it was long considered impossible, even blasphemous, to attempt to reverse 'death'. Because of these factors, the area of resuscitation failed to progress until the Enlightenment in the 18th Century. The main elements of resuscitation were then developed over the next 200 years, and eventually brought together to create CPR in the early 1960s. The increased demands that morality was seen to place on the medical profession to combat sudden cardiac death subsequent to this development may have been an important factor in why CPR has come to be used in the widespread manner it currently is.

O, that I could but call these dead to life!
William Shakespeare, King Henry VI

Cardiopulmonary resuscitation (CPR) is now the standard treatment for someone having a cardiac arrest. It is, however, a procedure that has emerged only relatively recently. An understanding of how CPR developed highlights a number of important factors. This paper will first examine the changes that needed to take place for progress in the area of resuscitation to occur, before moving on to examine how the various elements of resuscitation were then developed, and eventually brought together to create CPR in the early 1960s. It will finish by suggesting an important outcome of this development for the medical profession.

Changes needed for progress in the area of resuscitation

While there are sporadic accounts of attempted resuscitation since antiquity,[1–5] before 1960, successful resuscitation was largely limited to occasional victims of respiratory arrest.[6]

For much of recorded history, human beings understood death in a rather fatalistic manner—for the most part beyond human control, and "as much an inevitability in their lives as the death of every other organic creature".[7] The loss of a person's life signs was considered the victory of death. It is, in fact, only a relatively recent development that the medical profession has "regularly attempted to wrest such patients from death".[8]

For a number of scientific and religious reasons, it was long considered impossible, even blasphemous, to attempt to reverse 'death'. One of the most important of these factors was the influence of Galen of Pergamum (129–200), whose writings on medicine became accepted as the authority on the subject for 1400 years.[9]

While Galen's erroneous anatomy and views on the circulation played a part in preventing progress in the area of resuscitation, it was his physiology that caused the

most damage; which required a belief in 'vital spirits' and 'innate heat'.[2,9] Galen believed that the 'innate heat of life' was produced in the 'furnace of the heart'; being turned on at birth and extinguished at death, never to be lit again. As Eisenberg writes, "this strongly held belief, passed on through the centuries, is one reason why no one believed that death could be reversed".[2]

The fall of the Roman Empire in 476 ushered in what is widely considered to be a millennium of intellectual stagnation, known as the 'Middle Ages'. Throughout the Middle Ages there was no divergence from the 'truth of Galen'. It was not until the Renaissance that the work of Galen began to be challenged by the likes of anatomists Andreas Vesalius and William Harvey. However, while the Renaissance saw a stirring of groundbreaking intellectual activity, the thinkers of the time still generally worked under the framework of Christian orthodoxy.[2]

Orthodox belief held that individual spiritual judgment took place at death, followed by salvation or eternal damnation. The prevailing attitude of the time was that the wisdom of the Lord should be accepted in all things, and that any effort to alter the inevitable was contrary to God's will, and, therefore, blasphemous.[2]

However, with the rapid increase of scientific discovery and the rise of secularism during the Enlightenment in the 18th Century, and with good evidence that it was at this time that the doctor's duty to prolong life first emerged,[10] the stage was finally set for progress in the area of resuscitation.

All of the main elements of resuscitation had been started to be investigated in some form, and over the next 200 years these elements would be developed and eventually brought together to create CPR.[2]

Artificial respiration

The search for artificial respiration lead to various techniques being developed over the centuries, however it has ultimately come to be defined by a technique that was described as early as the 18th Century.

In 1744, William Tossach gave one of the first accounts of successful mouth-to-mouth ventilation. However, while the mouth-to-mouth technique was already know at this time, it was instead bellows that were being advocated for artificial respiration during the 18th Century.[2]

Scientists had been able to establish that exhaled air contained a poisonous gas, which they called 'fixed air' (carbon dioxide). The finding of increased levels of 'fixed air' in exhaled air lent support to the belief that mouth-to-mouth ventilation might not provide enough oxygen to the victim. With the means to answer these concerns not available at the time, the advocates of bellows were able to make a compelling case for the inadequacies of exhaled air.[2,5]

By the middle of the 19th Century, however, a major change had occurred in artificial respiration, with the mechanical expansion and compression of the chest wall being advocated. For the next 100 years, this approach defined artificial respiration, with dozens of manual ventilation methods being promoted.[2]

Even after James Elam had established in 1954 that exhaled air was sufficient to maintain adequate oxygenation, manual ventilation methods continued to prevail;

with many still not convinced that mouth-to-mouth ventilation was superior over the other techniques.

All this was about to change, however, after a chance meeting between Elam and Peter Safar in 1956. Having been stimulated to become involved in resuscitation research by the meeting with Elam, Safar soon began a series of experiments to establish whether or not mouth-to-mouth ventilation was effective.[2,5]

By the spring of the following year, Safar had been able to conclusively establish three important points:

- First, simply tilting the person's head backward would usually open the airway.
- Second, most of the manual ventilation methods provided little air, whereas mouth-to-mouth ventilation provided excellent artificial respiration.
- Third, anyone could perform mouth-to-mouth ventilation easily and effectively.[11]

Within a year of these findings, Elam and Safar had been able to convince the world to switch from the manual ventilation methods to mouth-to-mouth ventilation.

Artificial circulation

Artificial circulation has also come to be defined by a technique that was described as early as the 18th Century, but was then widely ignored until the mid-1900s.

While John Howard first described external chest compression in the 18th Century,[3] it was not until 1878 that one of the first successful attempts to achieve artificial circulation via external chest compression was reported in animals by Dr Boehm, and not until 1891 that Friedrich Maass reported one of the first successful uses of external chest compression on humans.[2,5,12–15]

Despite the fact that external chest compression had been well described in several case reports, and that a form of it was already in use as part of the manual ventilation methods for artificial respiration mentioned above, external chest compression did not catch on as a means of artificial circulation.

While there could be a number of reasons why this happened, one of the most likely explanations, Eisenberg suggests,[2] is that while people at the time could accept external chest compression as a means of aiding ventilation, they may have found it difficult to envisage an artificial circulation role for chest compression as well. Indeed, many at the time did not even believe artificial circulation was possible. As a doctor in 1890 wrote, "we are powerless against paralysis of the circulation, while asphyxia can be treated through artificial respiration as long as the heart keeps beating".[2]

As a result of these factors, the role of external chest compression in artificial circulation was widely ignored until 1960, when William Kouwenhoven, Guy Knickerbocker, and James Jude accidentally discovered that they could achieve adequate artificial circulation by applying pressure to the chest with their hands. They published their findings that year in a very straightforward manner. As the authors

wrote in the article, “anyone, anywhere, can now initiate cardiac resuscitative procedures. All that is needed is two hands”.[15]

The birth of CPR

It had now been established that mouth-to-mouth ventilation was an effective technique for artificial respiration, and that external chest compression was an effective technique for artificial circulation. Now all that was needed was for the two techniques to be brought together to create the CPR.

This formally took place on 16 September 1960, when Safar, Jude, and Kouwenhoven presented their findings at the Maryland Medical Society. In this presentation, Safar stressed the importance of combining artificial respiration and artificial circulation, stating that the two techniques of mouth-to-mouth ventilation and external chest compression “cannot be considered any longer as separate units, but as parts of a whole and complete approach to resuscitation”.[2]

While this “whole and complete approach to resuscitation” was so new that it was still without a name, CPR had been born. For CPR to gain widespread acceptance, however, there also needed to be a way of re-establishing a normal cardiac rhythm.[2]

Defibrillation

The primary means of re-establishing a normal cardiac rhythm is, of course, to pass an electric current through the heart to cause electrical cardioversion For this ability to develop, however, it required, inter alia, the discovery of a connection between ventricular fibrillation and electrical cardioversion.[1,16]

In 1899, Jean Louis Prevost and Frederic Battelli discovered this connection while researching on animals. Prevost and Battelli established that a weak electric current passed through the heart, directly or through the chest, would fibrillate the heart, and that a stronger electric current was capable of terminating fibrillation. The significance of Prevost and Battelli’s findings, and its relevance to humans, were not, however, appreciated for another three decades.[2,5,16]

In 1926, electrical companies began seeking advice on how to deal with the alarming number of fatal electric accidents among their workers. It was with funding from one of these electrical companies that Donald Hooker, William Kouwenhoven, and Orthello Langworthy began to study the effects of electricity directly on the heart, in 1930.[2] The findings from their research, also conducted on animals, backed up the earlier work of Prevost and Battelli.[17]

While the Second World War prevented Hooker, Kouwenhoven, and Langworthy from developing the ability to defibrillate human hearts, it was on the basis of their work that Claude Beck constructed the first defibrillator, an internal defibrillator.[2] It was with this internal defibrillator that Beck was able to successfully defibrillate a patient’s heart for the first time in 1947.[18] However, the medical profession still lacked the ability to defibrillate the heart without having the victim’s chest open. All this changed when Paul Zoll developed external defibrillation in 1955.[19]

However, the defibrillator that Zoll designed (just like the earlier internal defibrillator developed by Beck) utilised alternating current (AC). Because of this, these

defibrillators were very large and heavy. While some manoeuvrability could be attained by storing the defibrillator in a large cabinet with wheels attached to the bottom, it was essentially not practical to bring the defibrillator to the patient. This lack of portability was a serious limitation of the AC defibrillators, and meant that not many lives would be able to be saved until this portability issue was resolved.[2]

Bernard Lown addressed the portability issue in the early 1960s when he developed a defibrillator that used direct current (DC) instead of AC. This eliminated the need for a heavy transformer, thus allowing the defibrillator to be battery operated. With this came portability, and meant that the defibrillator could now travel to the patient.[2,20]

CPR and defibrillation: a new hope

Thus, by the early 1960s, the medical profession was in the possession of CPR, and also, importantly, defibrillation. The development of portable defibrillation was particularly significant as it coincided with the emergence of CPR.

With CPR now providing a means of keeping someone having a cardiac arrest alive longer, and the defibrillator now being able to be rushed to the patient, there were now many more opportunities for the defibrillator to be used to save lives.[2] Indeed, these two procedures have become so closely associated that the use of defibrillation is generally assumed when CPR is spoken of.

The Increased demands of morality

An important outcome of this development of CPR, just like the development of so many other life-prolonging technologies, was the increased demands that morality was seen to place on the medical profession to combat death.

As Daniel Callahan has argued, as death became more and more under the control of humans, as it became increasingly possible for medicine to save and prolong life, to manipulate the conditions of dying, the fatalistic view of death was rejected, and morality was called upon to increase its demands to struggle against death.[7,21]

Embedded in this change is what Callahan calls the 'moral logic' of medical progress; where the scientific imperative of progress comes to take on the force of a moral imperative: what can be done medically to struggle against death, ought to be done, and what ought to be done, ought to be available to all.[7,21] If not, then "…we are open to moral blame. People will die who need not die. If we do not use our newly available technologies to save lives, we can be held accountable for the loss of those lives".[7]

Thus, once in possession of CPR, the medical profession was perhaps seen to be under a moral obligation to use CPR to combat sudden cardiac death; that they could now not sit idly by and allow *any* patient having a cardiac arrest to simply die when there was the slightest chance they could now prevent death by using CPR.[22]

While there have clearly been many reasons why CPR has come to be used in the widespread manner it currently is, it is suggested that this ethical dimension was an important factor.

Summary

In summary, CPR is now the standard treatment for someone having a cardiac arrest. It is, however, a procedure that has emerged only relatively recently. For several scientific and religious reasons it was long considered impossible, even blasphemous, to attempt to reverse 'death'. Because of these factors, the area of resuscitation failed to progress until the Enlightenment in the 18th Century.

The main elements of resuscitation were then developed over the next 200 years, and eventually brought together in the early 1960s to create the procedure of CPR. The increased demands that morality was seen to place on the medical profession to combat sudden cardiac death subsequent to this development may have been an important factor in why CPR has come to be used in the widespread manner it currently is.

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THE NEW ZEALAND MEDICAL JOURNAL
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Dyspnoea in a 17-year-old swim instructor: a diagnosis of hot tub lung

Tzu-Chieh Yu, Rashid Ahmed, Elaine Yap, Sunil Kumar

Hot tub lung has recently been recognised as a granulomatous lung disease, more commonly perceived as a hypersensitivity response to non-tuberculous mycobacteria carried by water aerosols from hot tubs/spas, showers, and indoor heated swimming pools. The responsible pathogens belong to the *Mycobacterium avian* intra-cellulare complex (MAIC).

Although there is ongoing debate in the literature regarding its pathogenesis[1] (infectious versus hypersensitivity reaction related to the MAIC organisms), it has many features of extrinsic allergic alveolitis (EAA).

We report a case of presumed hot tub lung in a seventeen-year old woman.

Case report

A 17-year-old New Zealand European woman, presented with 6 weeks of worsening dyspnoea and 2 weeks of left-sided pleuritic chest pain, with a dry cough.

As a previously fit and well swim instructor and lifeguard, she reported becoming short of breath after climbing one flight of stairs and struggling to walk on flat surfaces. She denied symptoms of fever, night sweats, and weight loss, and had no risk factors for venous thromboembolic disease.

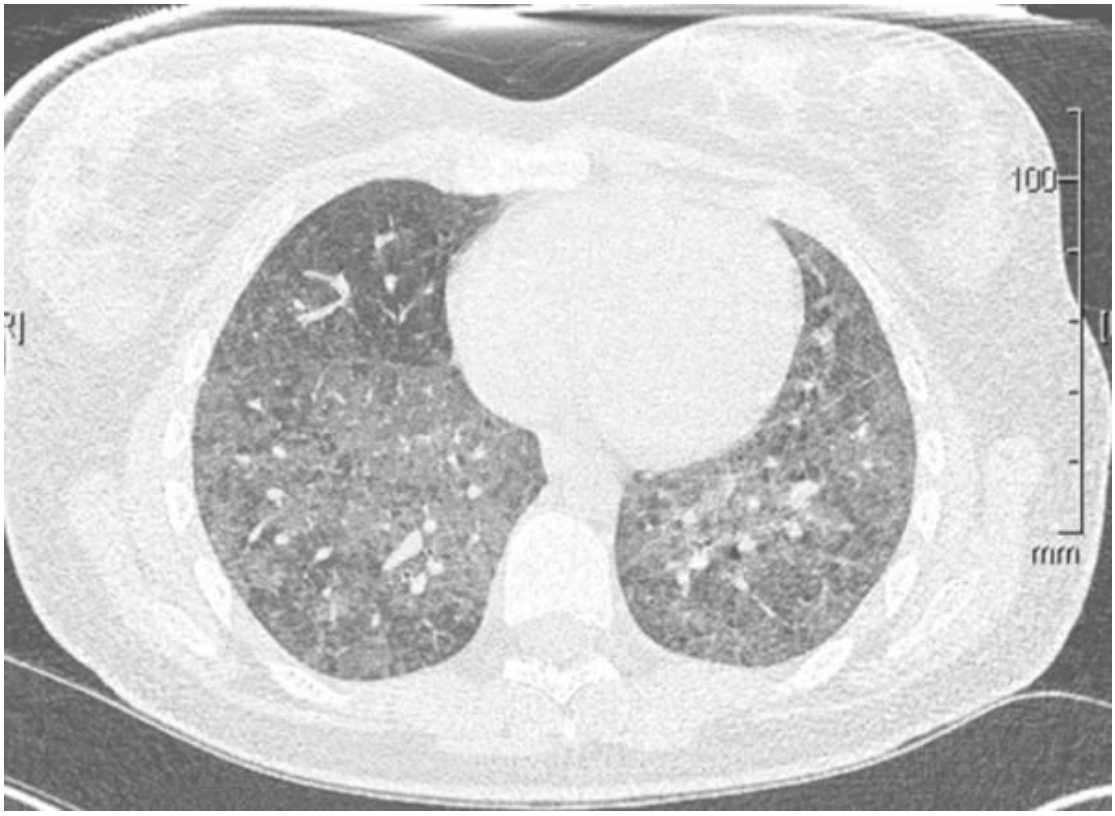
She was a non-smoker, who was swim instructor for 2 years at an indoor heated swimming pool which was non-chlorinated. She noticed dyspnoea 6 months after starting work there and an exacerbation of this symptom with each visit to the pool.

On examination, she was afebrile and tachypnoeic with oxygen saturation of 89% on air. There were bi-basal inspiratory crackles but chest was resonant on percussion and rest of examination was unremarkable.

Initial chest X-ray showed increased interstitial lung markings bilaterally, corresponding with extensive areas of nodular ground-glass opacification seen on high-resolution CT scan, most marked in the lung apices (Figure 1).

Blood tests were largely normal but C-reactive protein was mildly elevated at 16 mg/L. Vasculitis and connective tissue disease screening were negative. Trans-bronchial lung biopsies showed scattered non-necrotising granulomas, with an inflammatory mixed leucocytic infiltrate in the interstitium but no interstitial fibrosis. Inadequate tissue samples meant that culture was not performed. Broncho-alveolar lavage fluid (BALF) failed to reveal evidence of infection.

Figure 1. High-resolution CT showing extensive areas of nodular ground-glass opacification with scattered areas of mosaic attenuation, which reflects air trapping consistent with bronchiolitis[2]



Based on the clinical and investigative features, a diagnosis of EAA was made: presumed to be hot tub lung as symptoms were exacerbated with each visit to the indoor swimming pool although no evidence of MAIC was obtained.

With instructions to avoid further exposure to indoor swimming pools, she was commenced on 40 mg prednisone daily for 3 weeks, after which her symptoms significantly improved.

Discussion

To our knowledge, this is the first reported case of hot tub lung in New Zealand, a disease which has only been described in a few case reports since 1997.[1] This emerging disease is largely believed to be a form of EAA, a group of related inflammatory interstitial lung diseases that result from hypersensitivity immune reactions to various inhaled environmental antigens.

Related and better documented conditions include Farmer's lung and Bird fancier's lung. It is synonymous with hypersensitivity pneumonitis (HP).

As a newly recognised disease with few documented cases, the theory of hot tub lung being consistent with a hypersensitivity reaction has been based on these observations:

- All cases have history of exposure to the known antigen (heated pools of water); symptoms reoccur with repeated exposure;
- Affected individuals have all been immunocompetent;
- Some have shown spontaneous improvement with cessation of hot tub exposure; those treated with corticosteroids show improvement rather than worsening; and
- M any experience natural resolution of symptoms and radiographic abnormalities regardless of treatment.[1]

There are keys to diagnosing hot tub lung. Firstly, when reviewing respiratory symptoms, consider EAA and ask about exposure to indoor heated pools of water. Although symptoms of hot tub lung can be non-specific such as dyspnoea and chest pain, diagnosis should be suspected when high-resolution CT demonstrates ground-glass opacities[2] and there are histological findings of non-necrotising granulomas.

When MAIC-positive cultures from sputum, BALF, or biopsied lung tissue are obtained, diagnosis can be confirmed.[2] Treatment primarily involves avoidance of further antigen exposure, with the use of steroids only if symptoms are persistent or initially severe.[3]

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THE NEW ZEALAND MEDICAL JOURNAL
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Cryptogenic organising pneumonia in a 92 year old

Helen Kenealy, Geoffrey Green

Abstract

We report a case of a 92-year-old lady with cryptogenic organising pneumonia (COP). She was referred for assessment by her family doctor with raised inflammatory markers. She gave a history of a severe viral illness 2 months prior. Her symptoms at the time of review were a dry cough with minimal sputum production. Respiratory examination was unremarkable, except for mild shortness of breath on exertion. Her series of chest X-rays revealed patchy, bilateral, migratory areas of consolidation. The diagnosis of COP was made on a combination of clinical, radiological, and bronchoscopic alveolar lavage findings. Treatment with high dose steroids saw improvement clinically and radiologically within 6 weeks. COP is a rare but modifiable disease. Presentations such as these should prompt early respiratory consultation.

Cryptogenic organising pneumonia is a rare but modifiable condition. This is in contrast to idiopathic pulmonary fibrosis which is a progressive and lethal disease. We describe a representative presentation of this condition in an elderly woman. There is no data in the literature about the prevalence of this condition in the very elderly and it is therefore possible that it is under-diagnosed.

Case report

Mrs C is a 92 year old with known moderate cognitive impairment. She was referred by her family doctor with a raised erthyrocyte sedimentation rate (ESR) of 112 mm/hour. She and her husband gave a clear history of a severe viral illness 2 months prior to review which had left her bed bound.

At the time she had a bad cough productive of small amounts of sputum with a low grade fever, sore throat, upper arm myalgias, no appetite, mild nausea, confusion, and lethargy. Her general practitioner treated her with a course of antibiotics.

When reviewed at clinic she was slowly improving with appetite and weight increasing. Her main complaint was a persisting dry cough productive of minimal amounts of sputum with no haemoptysis. She had a past history of pulmonary tuberculosis treated in a sanatorium at age 5 and was a lifelong non smoker. There was no suggestion of aspiration on history. She did not appear acutely unwell. Her respiratory examination revealed mild shortness of breath on exertion, but was otherwise completely unremarkable, including the absence of clubbing. There was no evidence of connective tissue diseases on examination.

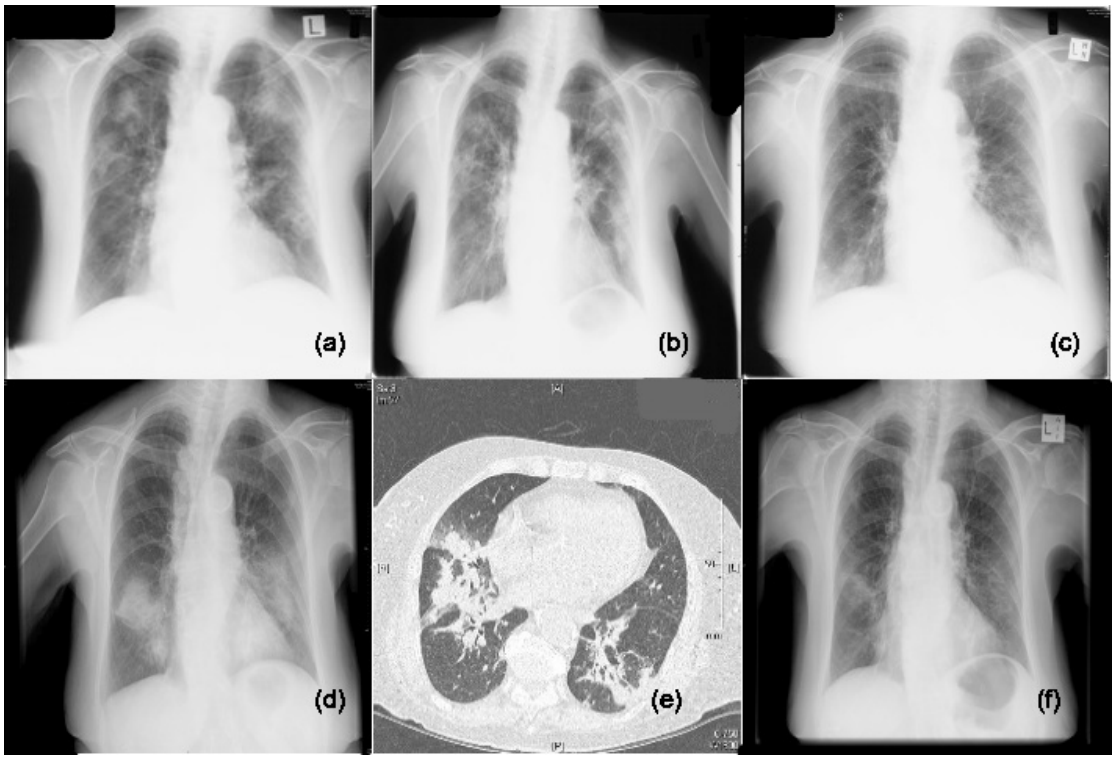
Her series of chest X-rays (CXR) revealed patchy, bilateral, migratory areas of consolidation, firstly in the upper to mid zones, then in the bases (Figure 1 [a,b,c,d]). It was on this basis that cryptogenic organising pneumonia was considered a differential and referral to our respiratory colleagues was made.

A bronchoscopic alveolar lavage (BAL) was performed which excluded both bacterial and mycobacterial infections and no malignant cells were seen. The lavage was hypercellular with a predominance of foamy histiocytes (57.5%). The other cells seen were: lymphocytes 27.3%, neutrophils 9.3%, eosinophils 3%, multinucleate histiocytes 0.7%, and histiocytes containing particulate material 0.2%.

Her peripheral eosinophil count was always normal when measured. A serum autoantibody screen (including both p and c-ANCA) was negative. High resolution CT (HRCT) chest revealed multiple peripheral bilateral areas of consolidation with air bronchograms and some cylindrical bronchiectasis (Figure 1 [e]).

Review by a speech and language therapist (SLT) excluded aspiration. On the advice of the SLT no formal investigations were performed as bedside assessment was unremarkable.

Figure 1. Radiological findings



This initial chest X-ray (a) at time of viral illness shows patchy bilateral mid and upper zone opacities, a second X-ray 1 month later (b) shows non resolution despite a course of oral amoxycillin-clavulanic acid. The third X-ray 2 months later (c) shows bilateral basal opacities. A fourth shows worsening opacities bibasally another 2 months later (d). HRCT at this time shows bilateral dense consolidation with air bronchograms (e). Last chest X-ray (f) shows marked improvement after 6 weeks of steroids.

With the results of the investigations described above she was diagnosed with cryptogenic organising pneumonia (COP) and started on 60 mg oral prednisone by the respiratory specialists and reviewed 6 weeks later. This treatment saw a marked

improvement in her clinical state, settling of her inflammatory markers to normal and near normalisation of her CXR (Figure 1 (f)). No lung function tests were done. She was monitored in respiratory outpatient clinic using inflammatory markers and chest X-rays.

The patient was started on oral co-trimoxazole for *Pneumocystis carnii* prophylaxis and weekly alendronate for osteoporosis prevention.

Discussion

Cryptogenic organising pneumonia is generally thought of as a diagnosis of exclusion.[1] It is part of a larger group of idiopathic interstitial pneumonias and is said to have first been described in 1983.[2] The mean age of onset is 55;[2] it most commonly occurs in the sixth decade.[1]

This disease can be contrasted with interstitial pulmonary fibrosis in which the fibrosis is generally irreversible and progressive. Histologically, the organising pneumonia pattern of lung injury exhibits plugs or buds of granulation tissue that fill bronchiolar lumen and extend into the alveolar ducts and spaces.[1]

Experimental data suggest that the histopatholological hallmarks are the result of an imbalance of the matrix metalloproteinases and the tissue inhibitors of metalloproteinases.[3] The migratory pattern of the patchy, bilateral, peripheral opacities as seen in this case are not uncommon. Both BAL and HRCT are important investigations in the diagnostic process.

Relapses are said to be common, however they can be reduced with medium-term high-dose oral steroid treatment.[1,3]

The key learning points are to seek respiratory opinion early when X-ray changes evolve, or do not resolve; secondly to always consider treatable causes of symptoms, including in the elderly; thirdly a chest X-ray should always be part of the work up in a patient with an ESR >100.

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THE NEW ZEALAND MEDICAL JOURNAL
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A 14-year-old boy with left leg osteomyelitis and acute hypoxaemic respiratory failure

Navneet Singh, Gyanendra Agrawal

Clinical

A 14-year old previously healthy boy presented with fever and painful swelling of the left leg for 10 days. There was no history of local trauma prior to the onset of symptoms. Osteomyelitis of the left tibia was diagnosed at a local hospital and local debridement performed. He was then referred to the authors' institute to manage progressively worsening breathlessness of 5 days duration.

Chest radiograph and high resolution computed tomography (HRCT) were performed (Figures 1 and 2).

Figure 1. Chest radiograph tomography

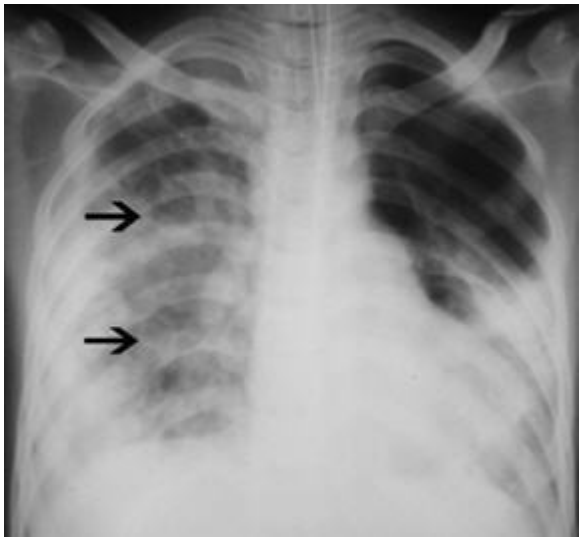
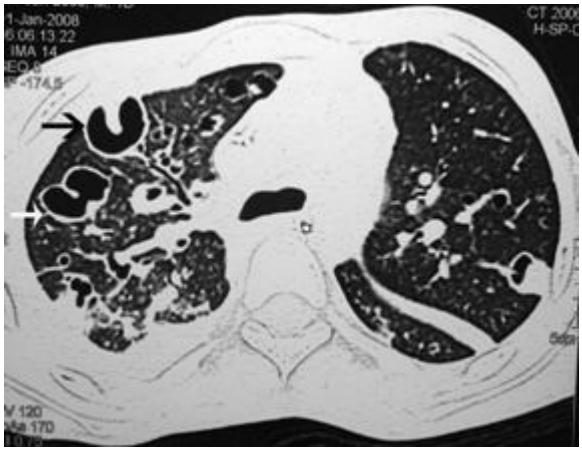


Figure 2. High resolution computed (HRCT)



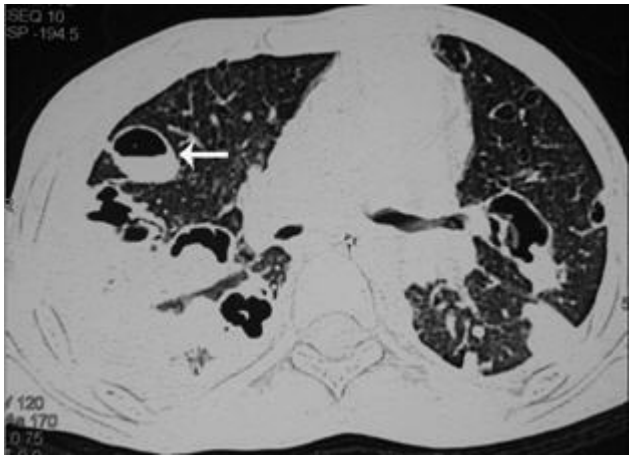
What is the diagnosis?

Answer

Disseminated staphylococcal disease

The chest radiograph shows consolidation in both lungs with multiple cystic lesions (Figure 1, arrows) and computed tomography confirmed the presence of multiple pneumatocoeles in both lungs (Figure 2, arrows), including some with air-fluid levels (Figure 3).

Figure 3. HRCT showing patchy consolidation and pneumatocoeles in both lungs with more severe involvement on the right. One of the pneumatocoeles shows presence of air fluid level (white arrow)



Percutaneous needle aspiration from the right pleural cavity revealed frank pus and an intercostal tube drainage was performed. Intravenous cloxacillin and amikacin were initiated in view of clinical suspicion of disseminated staphylococcal disease. The patient developed hypoxemic respiratory failure subsequently and was therefore intubated and shifted to the respiratory intensive care unit (RICU) for mechanical ventilation. A bedside echocardiography did not reveal any vegetations.

Growth of methicillin-sensitive *Staphylococcus aureus* on pus and endotracheal aspirate cultures later confirmed the presence of disseminated staphylococcal infection. Following treatment with parenteral antibiotics and supportive care, he was weaned off mechanical ventilatory support and shifted out of the RICU after 14 and 17 days of hospital stay respectively.

Discussion

Serious cardiopulmonary complications are well known after suppurative arthritis and osteomyelitis caused by *S. aureus*. Severe pulmonary involvement can present as bilateral bronchopneumonia that often may be associated with the development of pneumatocoeles, pneumothorax and empyema. This commonly occurs even without any demonstrable evidence of immunodeficiency.

Chest radiographs reveal empyema and pleural effusion with/without associated pneumothorax in as many as 93% of patients. However, the more characteristic radiological finding namely the presence of pneumatocoeles is identified in less than one-third of patients and in the majority, these would be apparent only on radiographs taken a few days after admission.

Deterioration after hospital admission occurs in almost two-third of cases. Although staphylococcal pneumonia is commonly seen in infants and young children where it occurs as a primary event, adolescents and young adults may develop the same following skin, soft tissue, and osteoarticular infection.

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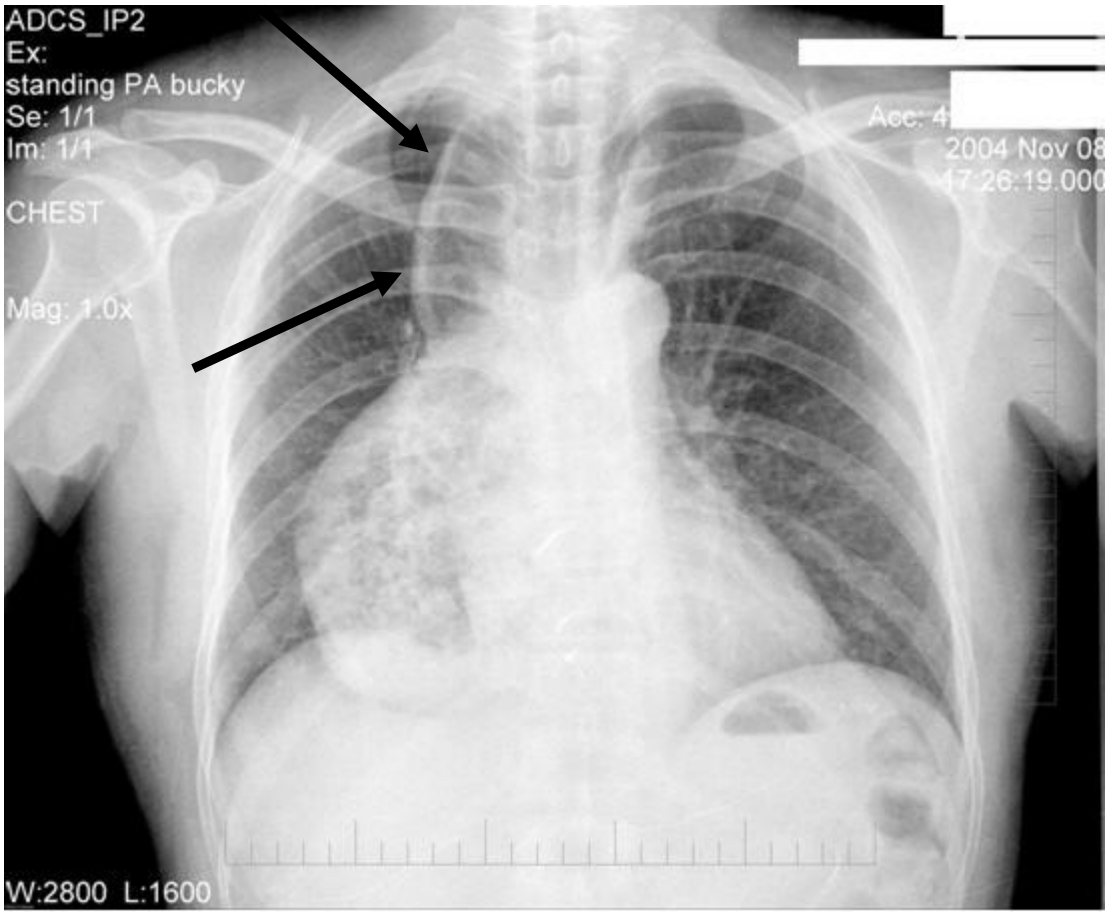
An incidental thoracic mass

Philip Finny, Jubbin J Jacob, Nihal Thomas

A 64-year-old asymptomatic gentleman with Type 2 diabetes mellitus underwent a routine chest radiograph (Figure 1).

Figure 1



What is the diagnosis?

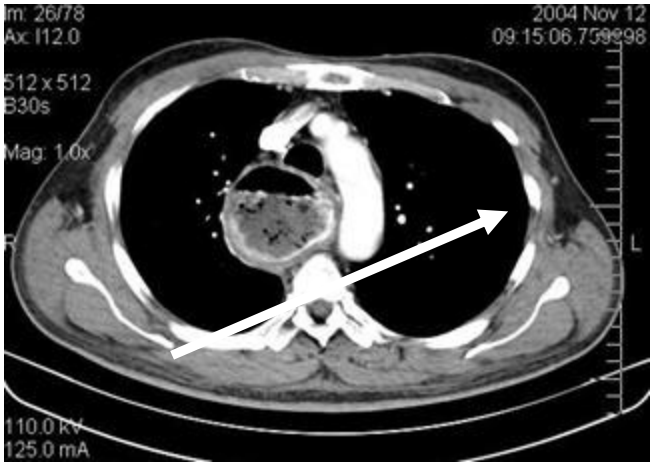
Answer

The chest radiograph shows substantial dilatation of the oesophagus (arrows). These findings are suggestive of an asymptomatic *achalasia cardia*.

Usually chest radiograph findings in achalasia cardia include the absence of the gastric air bubble and finding of an air/fluid level in the thorax. However, in this case both these findings were absent. The diagnosis of achalasia can be confirmed by barium swallow in which an air fluid level mixed with barium can be seen in the thorax at the region of the aortic arch. When in doubt about the nature of obstruction at the gastro-oesophageal junction the patient can be asked to drink a glass of hot water in the erect position. This causes immediate and pronounced dilatation of the gastro-oesophageal junction and the whole barium suddenly passes into the stomach.

In our case the diagnosis was confirmed by a computed tomographic scan of the thorax (Figure 2) which revealed dilated lower oesophagus with a clear air fluid level.

Figure 2



Discussion

The cause of achalasia cardia is unknown but histologically there is degeneration of the myenteric plexus at the gastro-oesophageal junction. In this particular patient endoscopic dilatation was attempted which was unsuccessful. Subsequently a successful Heller's myotomy was performed.

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Bertillon's Nomenclature of Disease and of Cause of Death (part 2)

Written by Dr Colquhoun, Dunedin, and published in N Z Med J. 1907;5(23).2–3.

Continued from part 1 at http://www.nzma.org.nz/journal/121-1283/3281

If we look at our own system as given in the year book of New Zealand, we shall see how imperfect it is and how much it calls for reform. Specific Febrile or Zymotic diseases includes six orders. First under the heading of "Miasmatic" we have the common Zymotic Diseases—"Miasmatic" is of course an obsolete word which means nothing. Then we have under Diarrhoeal diseases, Dysentery; under Malarial, Beriberi; in neither case does this represent our knowledge on the subject.

In Clause 6, we come to local diseases, and the first order includes diseases of the nervous system. This includes Apoplexy and Hemiplegia—a bad classification, as these conditions when causing death are terminal diseases, usually implying vascular degeneration due to Bright's and some other general disease.

This suggests the idea that there ought to be some means of indicating whether in such cases the immediate cause of death might not be indicated by some such word as "terminal," and the actual disease causing the terminal condition be given and registered as the' cause of death.

Take a case of chronic Bright's, which ends in sudden death by Apoplexy. Such a case is not one which ought to be registered as death from disease of the nervous system. Or take a patient' with general paralysis who dies of Aspirative Pneumonia—such a case should not be shown as one of death due to Pneumonia. It ought obviously to go under "Diseases of the Nervous System".

Under diseases of the Respiratory System, we have in our own and Bertillon's classification, Pneumonia. It is difficult to understand why this should be so, and Phthisis pulmonalis be put under the general heading. In order 5, of Local Diseases, we have Diseased of the Digestive system. The most striking feature of our list is the absence of Appendicitis. This disease has only appeared in the English list since 1900.

Enteritis, responsible for 232 deaths in 1902, is a fair enough heading, but what about "Simple Cholera and Diarrhoea," which appear in the first class, i.e. Zymotic Diseases. What, is the difference in their cases? This surely points to the need of an official nomenclature which ought to be in the hands of every practitioner.

Under the last heading. Ill-defined Diseases, there appear, Dropsy, with two deaths in 1902, Marasmus with 323 deaths. Surely "Marasmus" ought to go the way of Dropsy and cease to appear as a cause of death. If it means anything, it means "wasting," and there must be something at the back of that. As a matter of fact most of such cases are due to Enteritis from improper feeding. It has no place in Bertillon's nomenclature.

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Pharmacologic treatment of children who have hypercholesterolaemia?

The American Academy of Pediatrics (AAP) has recently recommended that children as young as 8 years who have hypercholesterolaemia may be treated with statins. This has provoked a storm of criticism.

The critics point out that cholesterol serves as the building block for all steroid hormones, including cortisol, aldosterone, oestrogen, and testosterone. In addition, it has a key role in a variety of fundamental cell functions, particularly in the brain and nervous system. The point being that more harm than good may follow wide statin usage in childhood.

The authors of this editorial agree with the criticism and regard the AAP recommendation as a knee-jerk response to the American childhood obesity epidemic. They prefer attention to lifestyle and dietary changes as a more scientific and safe option.

N Eng J Med 2008;359,1309–12.

Chlamydia epidemic in the UK

In the UK, the Medicines and Healthcare products Regulatory Agency has given approval for azithromycin to be sold without prescription. It will be available to people aged 16 years and over who have tested positive for chlamydia or have had sex with someone who is infected. The reason—to combat soaring rates of chlamydia infection.

Methuselah notes that azithromycin is certainly not an OTC (over-the-counter) medication in New Zealand. Two tablets ($9.90) only can be prescribed for a proven or presumed chlamydia urogenital infection.

This Week in Medicine; Lancet: www.thelancet.com; Vol 327, 16 August 2008.

Microbial forensics and biocrime

This discipline traces the origin of a biological agent using a range of biochemical analyses, including genomic sequencing and protein and carbohydrate fingerprinting. It has been in the news since the anthrax terrorist attacks in the US in 2002. It enabled the FBI to identify that the anthrax in question came from a particular laboratory. It is now proposed that it could be used to pinpoint the source of food or water-borne pathogens such as *Salmonella*.

Another putative use is in the identification of the origin of an hospital-acquired infection—litiginous overtones in the US.

Nature 2008;454:813.

Antihypertensive agents and beneficial add-on effects

Angiotensin-converting enzyme inhibitors (ACEi), angiotension II receptor blocks (ARB), beta blockers, and diuretics in varying combinations have advocates as the best treatment for lowering raised blood pressure (BP).

While there is little doubt that BP reduction is the primary determinant of improved clinical outcomes in hypertensive patients, evidence suggests that not all the antihypertensive drugs have the same effect on other cardiovascular risk factors such as new onset diabetes, or on associated clinical conditions like atrial fibrillation, left ventricular hypertrophy, or congestive heart failure. This paper and editorial point out that ACEi and ARB may well beneficially modify disturbances in the renin-angiotensin-aldosterone system (RAAS) which are usually activated in subjects with raised BP. And CCB have been shown in clinical trials to slow the progression of atherosclerosis.

Very interesting, but as most of the patients with hypertension will probably need two or more antihypertensive medications to achieve BP goals, emphasis on identification of the first-step class of drugs to be used may be futile.

Southern Medical Journal 2008;101;779–80 & 818–23.

A place in the sun for intensive care medicine?

An Australian intensive care physician makes several telling points about hospital medicine in Australasia. For example—"we are faced with an increasingly salvageable but elderly population of patients, generally saddled with multiple comorbidities."

And, the patients are "older and sicker than those we would have encountered in the past. In addition, patients', society's, and our own expectations of medical care; our understanding of the causation and prevention of medical error; and the imperative to show our health care is of high quality, is driving a more defensive and perhaps more interventionist approach to health-care delivery."

These views are strongly endorsed by your scribe. However, in discussing the increasing specialisation of medicine and a decline in general medicine, our intensivist offers the opinion that "it could be argued that intensive care is the only true generalist specialty left in acute hospital adult medical practice." With tongue-in-cheek I trust.

Internal Medicine Journal 2008;38:619–21.

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Measure the quality of healthcare spending

In a UK King's Fund study of spending on healthcare,[1] John Appleby and Anthony Harrison sought to develop a rational, acceptable and evidence-informed process for arriving at sensible limits to healthcare spending, without abandoning two core equity values. These were that (1) the service should be funded in a progressive way (the rich contributing a higher fraction of their income than the poor), and (2) the service should be accessible at time of need, regardless of non-health factors such as income, geography, and so on.

During this government's term, NZ health services have received large increases in funding (10% per year in the past 3 years[2]), but there is an obvious limit to increases that outstrip the growth of the economy to this extent. The election debate provides an opportunity to think more deeply about the spending issue, and assess whether policies are based on evidence or rhetoric.

Appleby and Harrison listed the policy options for future spending as follows:

- Carry on increasing spending at current accelerating rates, postponing the inevitable decision to contain spending; or
- Carry on increased spending at current accelerating rates and improve efficiency and productivity, that is, buy extra time before confronting the inevitable decision to contain spending; or
- Align health spending growth to the long-term growth in the economy, with perhaps some scope for a modest rise in the proportion of GDP devoted to healthcare, as GDP grows.

"The first of these options", their study found, "is likely to be untenable in the medium to long run". The implied health budget would be hard to finance, at a time when other programmes, including social care and pensions, are also likely to impose increased demands on the public purse. Although the demographic changes are of only modest importance for health spending, their impact on these two other areas of spending is likely to grow and both will be hard to shift to the private sector beyond the share it now accounts for".

The second option has general appeal and leads to claims about an "expensive bureaucracy", the identity and costs of which have not been specified. Since many health service managers are clinicians (charge nurses, clinical directors, and so on) and the Health Department costs a fragment of the health budget, how has the postulated release of resource been measured? Major productivity gains from the second option are achievable when systems of hospital clinical work are modernised to help patients get to see the right person at the right time[3] but that requires culture change within the profession, not political direction.

Appleby and Harrison concluded that the third option must be the medium to long-term goal, but that it is not easy to pursue when the forces driving up demand and

costs cannot be contained, and it is the one least likely to be promulgated by politicians in an election year.

Which measurements can determine whether health services use resources wisely and how is the likely effectiveness of proposed new spending evaluated? Pharmaceutical Management Agency of New Zealand (PHARMAC), and The National Institute for Health and Clinical Excellence (NICE) in the UK, have developed some processes for that purpose, but the condemnation[4] heaped upon them reflects the fantasy that there will be no limit to spending on healthcare, and that measures of cost-effectiveness are wanton. However, if public health services are to be sustainable, politicians require more, not less measures of effectiveness, and these should include measures of the effectiveness of government policy changes. The political rhetoric will not change without them.

There is substantial evidence that NZ's health services do well by international standards,[5] and much better than the US, which spends 15% of its GDP on healthcare, but has "the most severe healthcare access problems related to cost, the greatest medical expense burdens, and the most pervasive inequities in care between adults with above-average and below-average income".[3] However there is a naïve, contrary belief in the community, reinforced by political rhetoric and promoted by most, except the Treasury, that more money is all that is required.

The level of public expenditure on the provision of healthcare is ultimately a political matter, but in NZ that decision has been made without enough evidence for effectiveness. As a consequence, political parties' reforms have habitually failed to fulfil their expectations.

If this flawed approach continues in less favourable economic conditions, reduced public expenditure on health will be forced, and society will have to decide what degree of inequality it is prepared to accept.

Appleby and Hansen concluded that,

- "Given the incentives facing individuals, clinical and research professionals, and the private sector, more will never be enough. As long as healthcare is free or nearly so at the point of delivery that will be so, and this will hold true whether or not healthcare is funded from taxation or social insurance. But there is scope for modifying the current pattern of incentives so as to moderate their impact."
- The Department of Health should ensure that the measurements are made to strengthen the knowledge base,
- These measurements should be actively pursued within the benefits-cost matrix they devised.

Whilst detailed information is available about resource inputs to healthcare,[2] the outcomes achieved have not been measured in clinically adequate detail for cost-benefit analyses. This technical agenda should be obligatory, to provide the knowledge required for better expenditure planning.

In this election campaign the parties vie with one another to attract support by proposals that are unsubstantiated by evidence for their effectiveness. There is little acknowledgement (in public) that the rate of inflated expenditure is not sustainable.

At a time when the roles of agencies like PHARMAC and NICE should be expanded to cover the assessment of new treatments and health service reorganisations (to provide the evidence needed for comparative evaluation of treatment options), there is no political will to act.

Financial and other health service incentives should discourage inappropriate treatments and reduce inappropriate activities, but "As long as policies continue to be based on the expansion of facilities and the promotion of demand, more will never be enough"[1].

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Netilmicin withdrawal: impact on neonates

The withdrawal of netilmicin from marketing in New Zealand in 2003 posed a dilemma in neonatal medicine, where this drug had been found to be effective against a wide range of pathogens. At that time, amikacin was proposed as an alternative because of a similar profile of activity against strains of coagulase-negative staphylococci (CoNS) and it became the agent of choice for late onset neonatal sepsis in the Dunedin Neonatal Intensive Care Unit.[1] The other alternative was vancomycin, the excessive use of which is associated with the emergence of multi-resistant organisms.

To assess the impact of this switch we performed a retrospective audit of all neonates treated with netilmicin from 1 February 1999 to 27 July 2003 and amikacin from 1 October 2003 to 31 January 2007. Neonates were diagnosed as septic if they had a positive blood, cerebrospinal fluid, or suprapubic bladder aspirate urine culture; or if they showed persistent clinical signs and a maximum CRP >20 mg/L during the treatment episode.[2] Subsequent treatment failure was defined as: repeated infection caused by the same bacteria within 5 days; or a treatment switch to vancomycin due to no apparent clinical improvement following netilmicin or amikacin treatment.

The dosages of netilmicin and amikacin used for neonates were based on commonly accepted protocols.[3] The data included gestational age, postnatal age, birth weight, sex, results of bacterial cultures, netilmicin and amikacin serum concentrations (Abbott TDx, Abbot Park, Illinois), serum creatinine, and C-reactive protein (CRP). Rates of treatment failure were compared using OR (95%CI) (Stata® Version 8, College Station, TX).

There was a higher than expected rate of treatment failure with amikacin compared with netilmicin. There were 169 treatment episodes with netilmicin in 97 neonates, with 66 episodes of sepsis diagnosed from positive blood cultures in 47 neonates. There were 98 individual amikacin treatment episodes in 80 neonates, with 49 confirmed septic episodes in 33 individuals.

In the netilmicin group, the most common causative organisms were CoNS and group-B streptococci. In the amikacin group, the most common causative organisms were CoNS and there were no infections caused by group B streptococci. There were two deaths in the netilmicin group, with neither attributed to sepsis. Seven neonates in the amikacin group died, four from overwhelming septicaemia. In the netilmicin group, treatment failure occurred in 13 (20%) of the 66 episodes of confirmed sepsis. In those treated with amikacin, treatment failure occurred in 17 (35%) of 49 episodes of sepsis in 12 (15%) individual patients. The odds ratio (OR) (95%CI) for treatment failure, amikacin compared with netilmicin, was 2.92 (1.21–7.04) p=0.02.

When the patients with group B streptococcus in the netilmicin group were excluded from the analysis, the OR (95%CI) for treatment failure was 2.0 (0.87—4.63) p=0.11. A similar proportion of peak drug concentrations were below the therapeutic range for

the two groups: 21 (11.7%) of 170 netilmicin peak concentrations were <5 mg/L and 18 (11.76%) of 153 amikacin peak concentrations were <20 mg/L.

The main limitation of the present study is that the study design was not appropriate to determining efficacy, being an observational study. An additional limitation is the comparison of two drugs with complicated dosing regimens, one of which the clinicians involved had extensive prior experience of, and the second which was new to their practice.

The drug regimens for the two antibiotics were different because an extended dosing regimen was used for amikacin while a frequent dosing regimen was used with netilmicin. It is possible that the increase in the dosing interval is responsible for some treatment failures. The profile of the bacteria populations in the two groups was slightly different with group B streptococcus being more prominent in the netilmicin group, and when these cases were excluded from the analysis there was no difference in response rates. However, the results raise significant concerns that the withdrawal of netilmicin from the New Zealand market may have disadvantaged the neonatal population.

Over the time 1998 to 2002, 64 medicines that had been licensed for paediatric use were withdrawn from the New Zealand market, for which there was no generic alternative available.[4] At the time it appeared that therapeutic alternatives (medicines indicated for the same purpose) were available for all of these medicines and there did not appear to be any consequences of this loss of choice.

The licensing of new medicines for the neonatal population is poor even in countries with initiatives designed to encourage the licensing of medicines for children.[5,6] Hence, the neonatal population is particularly disadvantaged by the replacement of older therapeutic entities with new therapeutic entities.

We conclude that the withdrawal of netilmicin from marketing in New Zealand may have impacted adversely upon the care of the neonatal population.

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A call to reduce harm from tobacco pack marketing and bolster consumer health protection in New Zealand

The New Zealand Commerce Commission recently issued warnings to the major tobacco companies in New Zealand and stated that their use of the descriptors 'light' and 'mild' risked 'breaching the Fair Trading Act'.[1] We suggest that this action indicates a need to consider both the future of tobacco pack marketing in New Zealand, and the function of consumer protection law and agencies charged with protecting public health.

The issue of continued tobacco industry deception—The removal of remaining 'light' and 'mild' descriptors from tobacco packs will probably do little to ameliorate the effects of deceptive marketing to smokers. Such marketing has occurred over decades. Tobacco companies have anticipated and prepared for the greater regulation of all their marketing activities. For instance, in New Zealand and elsewhere, other descriptors and signifiers, such as tobacco pack colours, have been prepared to pre-empt moves to regulate the use of 'light' and 'mild'.[2] Evidence from the UK also reveals that banning the descriptors 'light' and 'mild' may be unlikely to correct mistaken beliefs, which are deeply held and reinforced by other reassuring terms, images and colouring in product marketing.[3]

Some time ago, tobacco marketers began pairing 'blue', 'fine' 'white' and 'silver' with 'light' and 'extra light'; this association has ensured that smokers recognise these words as substitutes for 'light'. Tobacco companies in New Zealand have also established blue and white pack colours as ways to signal the idea of 'light' and 'extra light'.[4] These alternatives to 'light' and 'mild' are likely to ensure that the deception perpetuated on smokers will continue, albeit in a different guise.

These strategies mean that warnings to tobacco companies, such as the one recently issued by the Commerce Commission, will be insufficient to prevent continuing deception. Instead, comprehensive change is necessary.

Tobacco packages are a potent advertisement that makes every smoker a marketer for the brand they smoke. The advertisement is usually visible at the point of sale, each time smokers pull the pack out to light up, and each time they put the pack on a café table. The meticulously researched brand imagery featured on tobacco packages is eye-catching, appealing and likely to increase smoking.[5,6] Increased smoking means increased harm to health, increased healthcare costs, and greater poverty for smokers and their families/whanau.

Research evidence shows that young people see cigarette packs as glamorous and use them as a 'badge' product.[6] Thus the removal of this pernicious marketing will help reduce the risk that children and young adults will experiment with smoking, and become addicted.

The solutions for controlling tobacco pack marketing—To deal appropriately with 'pack marketing', we suggest the following complementary steps. First, increasing the graphic health warning to 100% of the front, top, bottom, and sides of the pack.

This would remove almost all effects of tobacco pack marketing. Second, introduce plain packaging,[7,8] where a brand name could be featured in a standard type font (shape, size, colour, and location) on the 10% of the pack back that is currently not a graphic warning. Except for the plain brand name, tobacco packs would have a uniform regulated colour, shape, size, and texture.[9]

This addition to tobacco control will cost taxpayers nothing; as with graphic warnings, it will be the tobacco companies that properly bear the costs. By contrast, these measures stand to save taxpayers both dollars and heartache.

Upgrading regulatory law and agencies for consumer health protection—The Commerce Commission's recent decision came 19 years after they had first been notified of the deceptive behaviour practised by tobacco companies in New Zealand, and after they had been notified on a number of other occasions.[10] Until the warning last month, neither the Commerce Commission nor the Ministry of Health had used the Fair Trading Act to move against such behaviour. In contrast, the Commission has taken tobacco companies to court to *increase* competition in the New Zealand market.[11] Ironically, the effect of that move was to promote more effective tobacco marketing to the New Zealand public.

The Commerce Commission's decision to not: (i) Act on 'light' and 'mild' in a way that would deter future such behaviour; (ii) Act to control other deceptive aspects of tobacco marketing in New Zealand,[4, 12] and/or (iii) Require remedial action for the deception (or payments to enable such action, as required in Australia)[13], suggests that more effective consumer health protection laws and structures are needed. This is particularly so for tobacco, due to the under-regulation of this extremely dangerous, addictive product.[14]

The systemic problems with New Zealand consumer health protection legislation include: (i) Fragmented government, with insufficiently clear responsibilities (e.g. the Commerce Commission has suggested that the Ministry of Health should cover tobacco consumer protection)[10]; (ii) Legislation that does not sufficiently take health consequences into account; (iii) Insufficient funding for consumer protection;[10] and (iv) Lack of political action to promote greater protection of consumers' health, including penalties for deceptive actions that are harmful to public health. These problems have slowed progress on tobacco control in this country.

The solutions, at least for tobacco consumers, include making the Ministry of Health directly responsible for acting to protect consumers from deception practised by tobacco companies (including Fair Trading Act aspects). This would require sufficient extra funding and staff to deal with tobacco companies (the resources used by PHARMAC to confront pharmaceutical companies would provide an appropriate model).[15,16] However, if the Fair Trading Act is to deal effectively with the general health aspects of consumer protection, it requires amendment. Additional provisions could include incorporating health impact assessment processes,[17] and a precautionary approach, which could require the tobacco industry to prove that its behaviour was not deceptive.

The fundamental cause of all the problems outlined above is the continuing incentive for companies to maximise the profits they make from manufacturing and selling tobacco products. This profit motivation is the underlying barrier to efforts to develop

long-term consumer protection from these hazardous and addictive products. In parallel with the policy initiatives outlined above, the removal of tobacco distribution from the commercial arena would simplify consumer protection, and promote greater public health.[14]

Competing interests: All authors have undertaken work for health sector agencies involved in tobacco control.

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Are practitioners of “alternative” therapies competent to practise medicine? Should the Medical Council take action?

A small number of GPs in New Zealand practice various forms of “alternative” therapy. Some call themselves “holistic” practitioners, “anti-ageing” specialists, “integrative” practitioners, and so on.

This group of doctors tend to request a large number of laboratory tests per patient, and many of the tests requested are unusual in general practice. The unusual tests requested include trace elements (zinc, copper, selenium, mercury, and iodine) as well as IGF-1, IGFBP3, CoQ10, apoE genotyping, salivary cortisol, dihydrotestosterone, and estrone.

The tests listed above should almost never be required in general practice. That these tests are being ordered in large numbers by "alternative" GPs is evidence that these practitioners are operating using different concepts of disease processes from those which are established in orthodox medical practice.

Medicine is distinguished from all the other “alternative” therapies by the fact that it is based on science and evidence. The standard of evidence demanded has been progressively raised to the point that the current standard of evidence on which the value of a therapy or laboratory test is based is the randomised controlled trial.

However, many members of the general public are not scientifically trained and are not in a position to distinguish between science and pseudo-science. The public places its faith in doctors, believing that medicine is a highly regulated profession, and trusting that the doctor will practice scientific medicine. The practice of non-scientific therapies by registered medical practitioners amounts to fraud, and brings disrepute on the medical profession.

It may be argued that fraudulent is not the correct description for some of these doctors, because they may believe in their “alternative” therapies. In that case, they are certainly incompetent to practice medicine, because one of the key competencies of a doctor is the ability to distinguish between science and pseudo-science.

The Medical Council of New Zealand (MCNZ) has the responsibility to ensure that doctors are competent to practice medicine. The evidence of inappropriate laboratory test usage must raise questions about these "alternative" doctors’ competency. The MCNZ should investigate these doctors. They should be asked to explain how they interpret these tests, and how the results affect their therapy. They should be asked to provide a satisfactory level of evidence to support their answers to these questions.

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THE NEW ZEALAND MEDICAL JOURNAL
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The use of deceit in health research

The letter from Shaun Holt "on the responses of alternative practitioners when approached about common childhood illness" published 3 October 2008 in the *NZMJ* (http://www.nzmj.com/journal/121-1283/3282) may have content interest for some readers, but I think it is of great interest to health researchers more generally.

The letter that outlines Shaun Holt's research findings does not mention the ethics committee that approved this research, but given the research design I assume this was the multi-region ethics committee.

The research design involved many levels of deceit—the research participants were provided with fictitious cases without knowing that they were fictitious, it seems that the research participants did not know that they were participating in research, and that the research participants did not know the identity of the researcher.

In my recent teaching of social research methods at a medical school I would have advised students that it would be very difficult to get ethical approval for this type of research. To enlighten the health research community it would be a great service if Shaun Holt shared with us how he managed to negotiate this process and what procedures the ethics committee asked to be followed in order to ensure that the research did not bring the medical profession or the health research community into disrepute.

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Media hype surrounding the 'bullying of junior doctors' article

I am concerned about the article that was published in the 19 September 2008 issue of the *NZMJ* on workplace bullying of junior doctors at Auckland Hospital.[1]

The article received quite a bit of media attention, being quoted in the *New Zealand Herald*[2] and on the *NZ City* website,[3] to name two that I saw.

The results section starts by quoting: "50% (186/373) of these doctors reported at least one episode of bullying behaviour". However, I would like to draw attention to the fact that only 123 of the 373 doctors approached even completed the survey. How so then, that the authors can quote 186 doctors as experiencing at least one episode? This number (186) continues to be used as a denominator regularly throughout the remainder of the results, along with various other denominators, only some of which are explained.

The authors also state their aims as "to explore the frequency, nature, and extent of workplace *bullying*". Although their survey investigated frequency, the authors never answered this part of the aim in detail. Why is this a problem?

It seems that the authors have used the terms 'Bullying' and 'Bullying behaviours' (also known as 'Inappropriate behaviours') interchangeably. A common misconception is that bullying and inappropriate workplace behaviour are the same thing. Inappropriate behaviours may be one-offs or occur sporadically—we are all prone to experiencing these at some time or other. Bullying, on the contrary, is defined as an individual being subjected inappropriate behaviours on an ongoing basis—most commonly specified as an average of one behaviour a week over a period of 6 to 12 months.[4–7]Even the authors of the Auckland study agreed that to be defined as bullying a behaviour must occur consistently over a period of time.

However, the authors of this study do **not** present the prevalence (which is itself in question!) of doctors who were *bullied*, but report the prevalence of those who have experienced at least one episode of inappropriate behaviour. The researchers then declare that the results of their study are comparable to International studies which look at the prevalence of *bullying*.

Unfortunately, the lack of clear definition in these areas and inconsistent reporting of results have enabled the media to create sensational headlines like "Bullying on the increase in hospitals,"[3] which undermines the integrity of our whole health system.

I think, in light of these things, that any attempts to interpret (and generalise) the results of this study should be taken with a grain of salt.

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Response from Scott, Blanschard, and Child

The authors wish to thank the writer for this thoughtful and well researched letter—to which we wholeheartedly agree with most of the comments. Indeed, in the discussion section of our paper, we specifically attempted to highlight the extreme limitations of our study and in particular, the issues of ; a) the definition of bullying b) perception by the victim, and c) prevalence due to responder bias.

Unfortunately, the study survey (and indeed the idea for our study) was derived from the questions asked in the referenced overseas papers. Our survey design was chosen for the express purpose of allowing us to compare our results to these overseas reports—with all of the inherent inbuilt difficulties contained within this survey design.

Finally, we did not seek media attention and did our best at every opportunity to bring sound reason to our limited conclusions and thereby not inflame the inevitable media hype surrounding the topic. While we were aware that the media was likely to be interested, we did not believe that this was a reason to withhold submission for publication but, on the contrary, we believed that it was even more important to publish our findings in a peer-reviewed journal in order to stimulate discussion and such considered responses as that contained within the above letter.

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THE NEW ZEALAND MEDICAL JOURNAL
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Moving beyond early detection of cancer—time to embrace the recommendations of the World Cancer Research Fund

As we approach another general election and political parties jockey for votes, the health system is in danger of becoming something of a political football. The complex issues relating to funding of new, prohibitively expensive cancer drugs such as Herceptin continue to dominate medical debate.

Recent proposals for a colon cancer-screening programme have highlighted the cost and workforce issues associated with screening in general. The Cancer Control Strategy is behind schedule. All agree on one issue and that is the simple fact that we cannot fund the escalating costs associated with early detection and treatment of our current disease burden. It has been suggested that we need to be wealthier as a country to offer our citizens an acceptable level of healthcare and comparisons are made of our poor ranking in health delivery by international standards. Hospitals are overloaded and waiting lists long. An alternative paradigm would require us to be healthier rather than wealthier as a nation.

In stark contrast to the huge publicity and debate generated by the issues listed above, the publication of a crucial report by the World Cancer Research Fund (WCRF) in November 2007 attracted surprisingly little attention from the medical community, including many cancer specialists. The World Cancer Research Fund is a well-established organisation with offices in London and New York. They have systematically reviewed the available published data from across the world on risk factors for all types of cancer. Their first report was produced 10 years ago. Updated findings of a major 5-year follow-up review were published in November 2007. This quote is from the introduction to their detailed report.

"The estimate of the previous WCRF/AICR Report was that cancer is 30 to 40% preventable over time—by appropriate food and nutrition, regular physical activity, and avoidance of obesity. On a global scale this represents over 3 to 4 million cases of cancer that can be prevented in these ways, every year. In many of its forms, cancer is a disease that can cause great suffering and claims many lives. The overall commitment of scientists and other professionals committed to disease prevention, as exemplified by this Report, is to reduce the rates not just of cancer, but of all diseases, so that more people enjoy good health until they eventually die in old age."

This wholehearted enthusiasm for improving quality of life by timely primary prevention should indeed become our declared central health strategy, accepted by all parties and immune from political point scoring and party politics.

The WCRF made the following simple recommendations for cancer prevention, which provide an excellent and practical basis for healthy lifestyle changes.

- **Be as lean as possible without being underweight.**
- **Be physically active for at least 30 minutes a day.**

- **Avoid sugary drinks.** Limit consumption of energy-dense foods (particularly processed foods high in added sugar, or low in fibre, or high in fat.)
- **Eat more of a variety of vegetables, fruits, whole-grains, and pulses such as beans.**
- **Limit consumption of red meats** (such as beef, pork and lamb) and avoid processed meats.
- **If consumed at all, limit alcoholic drinks to two for men and one for women a day.**
- **Limit consumption of salty foods** and food processed with salt (sodium)
- **Don't use supplements to protect against cancer.** Research shows that high-dose nutrient supplements can adversely affect our risk of cancer, so it is best to opt for a balanced diet rather than relying on supplements. (Note: some supplements may be appropriate for specific groups of people.)
- **Do not smoke or chew tobacco.**

Special population recommendations:

- **It's best for mothers to breast-feed exclusively for up to 6 months** and then add other liquids and foods.
- After treatment, cancer survivors should follow the same recommendations for cancer prevention.

Very few people seem to be aware of this report and the strength of evidence that they can make such a positive impact on their health profile by simple lifestyle changes. The WCRF report should be required reading for all health professionals and the recommendations as listed above made widely available to the public. It is crucial that politicians and healthcare professionals at all levels endorse and reinforce this concept at every opportunity.

All of these simple measures can be implemented with immediate effect and at virtually no cost. We must invest wisely in this scientific data and the emphatic confirmation it provides that prevention is indeed better than cure. We cannot afford to contemplate any alternative.

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Reference:

1. World Cancer Research Fund / American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington DC: WCRF/AICR; 2007 November. http://www.dietandcancerreport.org/

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Jenny Margaret Francis

Nelson GP Jenny Francis died on 9 September 2008, leaving her husband Brendon Turner and 2 young children, Lukas aged 5 and Gemma 4.

Jenny's life was one that was packed with living and although it was sadly shortened it was nevertheless one that was filled with happiness and achievement.

Jenny was born in Wellington in 1966. Her very beautiful smile became the hallmark of her personality. She smiled directly into your eyes and you know it came from deep inside her.

She had her primary school years at Raumati School where she quickly surrounded herself with friends. The ability to make and keep friends was to always remain an important part of her life and as a consequence she is now surrounded by them. Her secondary schooling was at Kapiti College with much the same group of friends although there were more of them.

From a young age, she developed a love of outdoor activities and was able to participate in several school trips to exciting places. She was a keen swimmer, not the fastest but was very graceful in the water with a lovely swimming style. She was also a beautiful snow and water skier. She became keenly interested and proficient at knitting, sewing, and embroidery—and piano playing.

She left school in 1984 motivated to go nursing but had to wait a year which she spent training as a laboratory technician. She then entered Wellington Polytechnic for nurse training, graduating with her Diploma of Nursing in 1987. She worked for 2 years as a staff nurse at Wellington Public Hospital. She loved paediatric nursing and was eventually employed in the Neonatal Unit. In 1990 she accompanied a friend to San Francisco where she worked as a nurse at the War Veterans Hospital and later that year at a hospital inland in California.

She returned to New Zealand having made up her mind to become a doctor and did her medical intermediate at Victoria University. She was then a mature student, well motivated and did very well. She was fortunate to associate with another group of great friends and continued with them throughout her years at Otago University, graduating MBChB in 1999. She particularly enjoyed traveling to the Shetland Islands to do her overseas elective in a General Practice there.

She spent 2 years back at Wellington Public Hospital as a house surgeon and in 2001 she moved to Nelson Hospital. This was to prove a very significant change as she met Brendon in the A&E Department. In a very short time they were spending the winter together under canvas on their magic property in the Maitai Valley and soon they had built a lovely home there. Lukas was born 5 years ago followed by Gemma 1½ years later and the arrival of these two was to Jenny the biggest achievement of her life.

Jenny continued to study and increase her medical experience and expertise. She gained a Post Graduate Diploma of Child Health in 2001 and a Post Graduate Diploma of Obstetrics in 2003. She undertook GP training, passed Primex to achieve MRCGP, and was well on her way to becoming a Fellow of that College.

Over the last year Jenny faced up to her illness (ovarian cancer) with amazing courage. She never complained and I am sure that was because she did not want to upset us all. Towards the end she set herself two new goals: one was to achieve a satisfactory home for her family which she was able to move into and enjoy; the second goal was also achieved when Lukas started school at Nelson Central School.

At her funeral, her father read a poem which he felt to be particularly appropriate for Jenny:

Good Friends

At every turning of my life I came across good friends.
Friends who stood by me even when time raced me by.
Farewell, farewell my friends. I smile and bid you goodbye.
No, shed no tears for I need them not. All I need is your smile.
If you feel sad, do think of me. For that is what I'll like.
When you live in the hearts of those you love.
Remember them…You never die.

Dr Tony Eames (GP, Wakefield) wrote this obituary, an abridged version of Jenny's father's speech at her funeral.

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NZMJ Digest cover error

In the October NZMJ Digest (Issue 10) the headline *Inpatients' use, understanding, and attitudes towards traditional, complementary and alternative therapies* appears on the cover (see below).

However the article doesn't appear in the Digest (nor is it mentioned in the Contents list) as it was not chosen as one of the final articles to appear. The cover headline should have been deleted prior to publication.

NZMJ Digest staff apologise to authors Amanda Evans, Bruce Duncan, Patrick McHugh, John Shaw, Craig Wilson as well as its readers for this error.

A summary of the article will be placed in the next NZMJ Digest and a copy of the article can be viewed at http://www.nzmj.com/journal/121-1278/3159/content.pdf