CONTENTS

This Issue in the Journal

A summary of the original articles featured in this issue

Editorials

Regulation of cigarette smoke toxicity
Murray Laugesen, Jefferson Fowles

Chronic obstructive pulmonary disease (COPD): smoking remains the most important cause
Peter Martin, Helen Glasgow, Jane Patterson

Lung cancer in Maori: a neglected priority
Matire Harwood, Sarah Aldington, Richard Beasley

Original Articles

Scope for regulation of cigarette smoke toxicity according to brand differences in published toxicant emissions
Murray Laugesen, Jefferson Fowles

Scope for regulation of cigarette smoke toxicity: the case for including charcoal filters
Murray Laugesen, Jefferson Fowles

Caroline Shaw, Tony Blakely, Diana Sarfati, Jackie Fawcett, Sarah Hill

Smoky homes: a review of the exposure and effects of secondhand smoke in New Zealand homes
George Thomson, Nick Wilson, Philippa Howden-Chapman

Attitudes to, and knowledge of, secondhand smoke in New Zealand homes and cars
George Thomson, Nick Wilson, Philippa Howden-Chapman

Access to tobacco products by New Zealand youth
Helen Darling, Anthony Reeder, Rob McGee, Sheila Williams

Can Quit Practice: a comprehensive smoking cessation programme for the general practice team
Deborah McLeod, Elizabeth Cornford, Susan Pullon, Kawshi de Silva, Corrianne Simpson; for The Can Quit Practice Group
The burden of death, disease, and disability due to alcohol in New Zealand

Jennie Connor, Joanna Broad, Jürgen Rehm, Stephen Vander Hoorn, Rod Jackson

Review Article

Tobacco tax as a health protecting policy: a brief review of the New Zealand evidence

Nick Wilson, George Thomson

Case Reports

Near death episode after exposure to toxic gases from liquid manure

Louise Couch, Laura Martin, Nigel Rankin

Suspicious pulmonary nodules

Simon Janes, Birgit Dijkstra, Carina Miles, Ian Cowan

Viewpoint

Rethinking the regulatory framework for tobacco control in New Zealand

George Thomson, Nick Wilson, Julian Crane

100 Years Ago in the NZMJ

Death under chloroform

Medical History

A tribute: the contribution of the Otago University Medical School and its students in World War 1

Pat Cotter

Methuselah

Selected excerpts from Methuselah

Letters

Bariatric surgery in New Zealand

Paul Samson, Michael Booth

Screening for prostate cancer: a patient’s view

Name withheld by request

Regarding ‘Investigation for iron deficiency anaemia’

John Dunn

CARPA standard treatment manual (New Zealand edition)

Kevin Whitney

Notices

Special offer: CD ROM of the New Zealand Medical Journal
Calling obituary writers
This Issue in the Journal

Scope for regulation of cigarette smoke toxicity according to brand differences in toxicant emissions
M Laugesen, J Fowles

This is the first published report comparing the overall toxicity of cigarette brands across countries using risk assessment and intensive machine smoking, and adjusting for smoke nicotine and the mortality distribution between disease groupings. The method provides a relative toxicity score as a rational basis for regulating cigarette emissions across all brands sold. Holiday Extra-mild’s overall estimated identifiable toxicant emission levels would reduce 39%, and its cancer risk by 37%, if this cigarette was required by regulation to have the same emissions as a certain Canadian regular brand, Export A full flavor. Regulation to reduce brand differences in emissions, without employing charcoal filters, would reduce total cancer risk by 13% for Holiday Extra-mild, based on measurable toxicants. Any toxicity reduction from including a charcoal filter would be additional.

Scope for regulation of cigarette smoke toxicity: the case for including charcoal filters
M Laugesen, J Fowles

This is the first published report comparing what charcoal filters can do in a cigarette company laboratory with what they do in a commercial cigarette. Over the last 40 years cigarette company scientists have reported that charcoal can reduce aldehydes and hydrogen cyanide emissions in cigarette smoke by 75%-80%. In two Mild Seven charcoal filter brand variants sold in New Zealand containing minimal charcoal, no such reduction in these emissions was seen. Emission reductions seen with the charcoal filters as reported 40 years ago, could today lower a brand’s overall identifiable toxicity by over 40%, mainly by reducing gases toxic to lungs, heart and blood vessels. Whether overall total brand toxicity would be reduced by this much is uncertain, as currently unidentified toxicants may not be susceptible to removal by charcoal filters. With more certainty, effective filters could reduce total cigarette cancer risk by at least 5%, or 80 deaths a year.

C Shaw, T Blakely, D Sarfati, J Fawcett, S Hill

Lung cancer is an important cause of mortality in New Zealand. This study examined trends in socioeconomic and ethnic inequalities in lung cancer mortality, between 1981 and 1999. In men, socioeconomic inequalities in mortality were demonstrated in 1981, and persisted over the study period, despite the fall in overall lung cancer mortality in men. For women, with the increase in lung cancer mortality between 1981 and 1999, an increase in socioeconomic inequalities was also seen. This was due
to a disproportionate increase in mortality of lower socioeconomic groups. Divergent mortality trends by ethnic group were seen in both men and women, which led to an increase in ethnic inequalities between 1981 and 1999. Qualitative predictions suggest that these socioeconomic and ethnic inequalities will continue to increase over time unless concerted public health action is taken.

**Smoky homes: a review of the exposure and effects of secondhand smoke in New Zealand homes**

G Thomson, N Wilson, P Howden-Chapman

Almost a fifth of New Zealanders, and over 30% of high school students, are exposed to secondhand smoke in their homes. Maori and those in low-income households are more likely to be exposed than others. New Zealand evidence indicates a significantly increased risk of death for those who live in a household with smokers, with over 250 deaths per year attributed to secondhand smoke in homes. Substantial Government investment in tobacco control is needed to reduce these deaths.

**Attitudes to, and knowledge of, secondhand smoke in New Zealand homes and cars**

G Thomson, N Wilson, P Howden-Chapman

New Zealanders’ knowledge about secondhand smoke (SHS) effects has greatly improved since 1989, but this knowledge may be shallow. Wellington area surveys indicate that many people are not aware that the major consequences of SHS are strokes and heart disease. Increased public support for smokefree homes in the last decade does not necessarily result in smokefree homes. Less than half of 14–15 year olds (with at least one parent who smoked) reported having a smokefree home. Increased investment in mass media campaigns on SHS issues is needed.

**Access to tobacco products by New Zealand youth**

H Darling, A Reeder, R McGee, S Williams

Data from the 2002 Youth Lifestyle Study of secondary school students were analysed to investigate the primary sources of tobacco products, barriers to purchase, and revenue generated from the sale of tobacco to underage youth. Among smokers, more than 40% had purchased tobacco products from commercial sources. The revenue to Government from sales of tobacco to 14–16 year old students, alone, was calculated (conservatively) at $12.5 million in 2002. Greater investment is needed to reduce youth tobacco use, and it would be appropriate to use some of the money generated by under-age sales to help reduce tobacco use among youth.
Can Quit Practice: a comprehensive smoking cessation programme for the general practice team
D McLeod, E Cornford, S Pullon, K de Silva, C Simpson, for The Can Quit Practice Group

The Can Quit Practice Programme was developed for general practices with a focus on the skills of the practice nurse in providing quit support. The quit rates achieved in the Can Quit Practice Programme evaluation confirm that smoking cessation programmes can be successfully implemented and maintained within general practices. Important components of providing quit support in general practices were: an autonomous role for practice nurses; well-managed practice procedures; adequate consultation time; and adequate funding for health promotion.

The burden of death, disease and disability due to alcohol in New Zealand
J Connor, J Broad, J Rehm, S Vander Hoorn, R Jackson

The relationship between alcohol consumption and health is complex. This study summarises harms and benefits of alcohol to the health of Maori and non-Maori populations in New Zealand. Overall, 4% of all deaths in 2000 can be attributed to alcohol; many due to injuries involving young people. The health burden of alcohol is dependent on drinking patterns as well as volumes, and so varies markedly by age, sex, and ethnicity.

Tobacco tax as a health protecting policy: a brief review of the New Zealand evidence
N Wilson, G Thomson

This review found that there is good evidence that tobacco taxation is associated with reduced tobacco consumption in the New Zealand setting. There was also some limited evidence for equity benefits from taxation increases. Two New Zealand studies suggest the potential for higher tobacco prices contributing to the control of youth smoking rates. There is a need for considerable improvement in tobacco taxation policy in this country, to better protect public health and to improve equity.
Regulation of cigarette smoke toxicity

The science of regulating cigarette smoke is the subject of two papers in this issue of the Journal.1,2

Ten points set out the strands of the scientific argument for regulation:

- **Smoking kills—through smoke.** Inhaled cigarette smoke kills 4400 New Zealanders annually.3 All tobacco deaths in New Zealand are deaths due to smoking, and almost all are due to smoking manufactured cigarettes or cigarette tobacco.

- **The leading toxicants have been identified.** Of the 4000 or more smoke compounds, three gases—acrolein, butadiene, and hydrogen cyanide—account for 65% of the identifiable toxicity, and the next 10 account for most of the rest.1

- **The leading toxicants are VOCs** (volatile organic compounds), in the invisible gas phase of smoke. VOCs account for over 80% of total smoke toxicity.1

- **These toxicants have different effects.** Butadiene, for example, causes cancer; hydrogen cyanide is toxic to the cardiovascular system; and acrolein is toxic to the respiratory system.1

- **The regulator can estimate toxicity.** A method is described that enables any health ministry to test the leading toxicant emissions of all brands sold, and allot an overall relative toxicity score for every brand.1

- **Regulation can reduce toxicity.**1,2 Leading toxicants would be tested regularly as a condition of continued sale, and highly toxic emitters excluded. Each year the permitted maximum levels can be reduced.

- **Emission differences highlight the need.** Substantial differences in emissions and in overall toxicity across brands argue the case for regulation.

- **Regulation is overdue.** Charcoal filters have been optional or token in cigarettes for the past 40 years, despite their known protective effects.2

- **Regulation will be worthwhile.** Smoke carcinogens identified to date account for over one-third of observed cancer risk.1 Reduction in carcinogenic emissions thus translates into considerable potential for cancer risk reduction. Regulation can reduce cancer deaths by at least 80 a year2 by employing charcoal filters.2 Further deaths may be preventable by regulating to stop the sale of high-emission brands.

- **Fire deaths can also be reduced.** It makes sense to regulate for fire-safer cigarettes as has been done in the state of New York, at the same time as regulating to reduce smoke emissions. Cigarettes are designed to burn full length, and so when left unattended they do so, and can cause fires, killing several people annually, often nonsmokers.4

Beyond these new perspectives on smoke science, regulation of tobacco products involves other issues—of power and control, public safety, consumer rights, and
Government’s duty of care towards smokers. Despite regulation, new brands will still be launched, and smoking will remain a dangerous activity.

Cigarette regulation is about power—who should control the toxicity of cigarette smoke—the Ministry of Health, or the cigarette firms? Leaving the responsibility with the smoker, by publishing toxicity ratings on the packet, assumes smokers will read the fine print and be able to source a less dangerous brand. The unregulated cigarette market, however, provides little choice. Cigarette firms have ignored the reports of their own scientists for 40 years on the safety advantages of charcoal filters. As the firms have not protected their own customers, the time is right for Government to use its latent powers and regulate cigarette smoke toxicity in the public interest—of smokers.

Cigarette regulation is about the public safety of ¾ million citizens inhaling unduly dangerous cigarette smoke an average of 200 times a day. These people populate doctors’ waiting rooms, and die at twice the rate of nonsmokers of the same age. The case for regulation is self-evident. For example, the carcinogenic tobacco-specific nitrosamines (TSNA) varied by a factor of 66 across the top 10 New Zealand cigarette cigarette brands when tested unburnt. And New Zealand’s favourite ‘mild’ cigarette, Holiday Extra-mild, emerged as the most toxic of 37 international brands for which comparable data were obtainable.

Cigarette regulation is about the consumer rights of smokers. No regulation, absolutely none, yet governs the inherent risk of cigarettes and their smoke, though Parliament in 1990 gave the Ministry of Health the necessary powers in the Smoke-free Environments Act. Smokers, who pay Treasury over $5.40 in tax per packet, are entitled to some toxicity controls. For some mental health staff in secure units, work exposure to second-hand smoke is now recorded on each occasion; cigarette smoke is an official workplace hazard.

In contrast, smokers’ rights are not protected. The cigarette firms’ tests of their own brands by their own laboratories have seldom been audited independently by Government, and they only test nicotine, carbon monoxide, and tar for printing on the packet. This information is virtually useless. The nicotine yield accounts for less than 1% of the variance in nicotine absorption carbon monoxide accounts for less than 1% of total cigarette smoke toxicity. Tar is a proxy measure for the 18% of total cigarette smoke toxicity associated with smoke solids, but low-tar cigarettes are found to emit more toxicants overall, not less. The Ministry of Health now has the opportunity to regulate to ban misleading labels, and reduce the offending toxicants in the smoke.

In reviewing the Smoke-free Environments Regulations, the Ministry of Health may decide to regulate for graphic health warnings on tobacco packets, to ban certain misleading descriptors, fully test tobacco product emissions, and publish the results. These are desirable aims, but insufficient by themselves. Disclosure alone has its limits—all brands contain the same leading toxicants in their smoke. Cigarette smoke is dangerously and defectively toxic. Only regulated limits on harmful smoke constituents can reliably decrease smoke toxicity.

Regulation of smoke is part of Government’s duty of care towards its addicted smoker- taxpayers—who contribute over $850 million annually in excise. Smokers, whether unwilling, unable, unready to quit, or recent relapers, face either giving up
Smokers face this ‘quit or die’ dilemma in increasing numbers. In the age group 35 years and over, in which nearly all smoking deaths occur, smoking prevalence decreased slightly (20.4% to 19.2%) between 1996 and 2002, but the numbers smoking increased by 33,000 (10%) to 377,000, due to ageing of the population. Among Maori, the numbers at risk are also increasing, as smoking prevalence has stayed at around 50% since 1990.

Regulation of smoke toxicity provides a third way of sorts out of this dilemma. Strong regulation could save an estimated 80 smokers a year from fatal cancers, out of 1700 cigarette cancer deaths a year. Cardio-respiratory toxicity can also be reduced, but the health gain cannot be estimated.

Finally, despite regulation:

- **New brands will still be launched.** In regulating to reduce cigarette smoke toxicity, a major concern is that cigarette makers will make false claims and use safety as a selling point, as with king-size filters in the 1960s. Today, however, new cigarette brands face penalties for misleading claims and tobacco brand-name advertising.

As long as cigarettes remain legal to sell, Government will not stop cigarette firms introducing low emission brands—unless they make false product claims. New brands with apparently lower toxicity are being test-marketed and one is to be released in Australia within 12 months. Brands of this type when introduced, could halt the recent decline in adolescent smoking. The total smoking control programme may need to be activated further to meet this challenge.

Old or new, all brands need regulated limits on their toxicity. Health groups can lobby for firm and expeditious regulation. Regulations control the carcinogens permitted in manufactured food and beverages, and manufactured tobacco products should be no exception.

- **Smoking will remain a dangerous activity.** Any smoke mixed with nicotine is to a large extent irreducibly toxic, because the smoker needs to inhale more nicotine (and the toxic gases) on an hourly basis. Even if regulation could halve smoke toxicity, the risks of early death from smoking would be still 1 in 4—more dangerous than 12 continuous climbing seasons on the high peaks of Mt Cook National Park—and without the views.

New Zealand is in a position to become the first country to effectively regulate cigarette smoke toxicity. Continuation of the present state of affairs—unregulated, unduly-toxic cigarette sales—is disastrous. ‘Failure to act in these circumstances is negligence’. Regulation is long overdue.

**Author information:** Murray Laugesen, Public Health Physician, Health New Zealand Ltd, Devonport, Auckland; Jefferson Fowles, Senior Scientist, ESR (Institute of Environmental Science and Research) Ltd, Porirua

**Potential conflict of interest:** The authors are co-inventors for a patent *Apparatus and methods for testing toxicity of cigarette smoke*, NZP 537968, 28 January 2005.

**Correspondence:** Dr Murray Laugesen, Health New Zealand Ltd, PO Box 32 099, Devonport, Auckland. Fax: (09) 446 0647; email: laugesen@healthnz.co.nz
References:


Chronic obstructive pulmonary disease (COPD): smoking remains the most important cause

Peter Martin, Helen Glasgow, Jane Patterson

Chronic obstructive pulmonary disease (COPD) affects 15% (200,000) of the adult population of New Zealand. It is the fourth-most-common cause of death after cancer, heart disease, and stroke—and accounted for 5.1% of all deaths in 1999.\textsuperscript{1} This figure is likely to be an underestimate of the true mortality, as COPD is often a contributing factor to deaths recorded as due to other causes.

The disease is estimated to cost more than $13 million\textsuperscript{2} in hospital costs annually, and accounted for more than 9000 hospital discharges per year between 1997–1999. The total economic impact to the community is much greater, and includes healthcare costs incurred outside of hospital and intangible costs such as lost productivity from illness and premature death.

Smoking has long been recognised as the most important cause of COPD.\textsuperscript{3} Recently, however, there has been increasing interest in other factors which result in similar patterns of lung damage. Chronic asthma (especially if poorly controlled) can lead to changes in the lungs comparable to those caused by smoking.\textsuperscript{4} Asthmatics who smoke also have impaired response to treatment.\textsuperscript{5,6} This may lead to less satisfactory asthma control and an accelerated decline in lung function—an effect which is additional to that of the smoking itself.

Some workers are exposed to allergens (as well as vapours, gas, dust, and fumes) which may be significant in the development of COPD.\textsuperscript{7} In some countries, development of COPD has been shown to be independent of the effects of smoking, and it is estimated that one in five cases of COPD may be attributable to occupational exposure.\textsuperscript{8} However at-risk occupations tend to have high rates of smoking, and so the influence of combined factors is difficult to determine.

There have also been studies on the effects of air pollution on the development of chronic lung disease.\textsuperscript{9} For non-smokers, the most important form of air pollution is often exposure to environmental tobacco smoke, and this is an important factor in the development of many diseases, including COPD.\textsuperscript{10} Genetic factors such as alpha \( \text{I} \)-antitrypsin deficiency are significant, and these effects are greatly accelerated if the person smokes.

Increasing understanding about these anatomical changes, which lead to COPD, has provided evidence which can support measures to modify the risk factors for the disease, including techniques to optimise management of asthma. This will reduce airway damage and remodelling, leading to chronic impairment of lung function. This evidence also provides strong support for effective industrial measures to reduce the effects of workplace hazards, and it adds to the evidence about the need to reduce air pollution. However New Zealand experiences lower levels of pollution compared to heavily industrialised countries and does not have many of the industries which lead to severe workplace exposures.
Therefore it must be emphasised that, in New Zealand, most cases of COPD result from chronic smoking. Recent estimates suggest that smoking accounts for 69% of the global burden of COPD.\textsuperscript{11} Low levels of air pollution and lack of heavy industry in New Zealand suggest that the contribution of smoking is likely to be higher. This effect is completely preventable, and provides many opportunities for intervention; smoking control remains the most effective measure to reduce the burden of COPD.

However many patients have a low awareness of the cause-and-effect link between smoking and COPD, and certainly a poor understanding of the magnitude of the risk. Perhaps there may be a well-intentioned attempt to shield patients from feelings of guilt and this can lead to a conspiracy of silence. Whilst poorly controlled asthma can certainly result in COPD, a more frequently encountered problem is the misdiagnosis of COPD as asthma.\textsuperscript{4} This leads to the inappropriate application of asthma treatments, and a failure to apply appropriate interventions; in particular, the failure to highlight the link between smoking and COPD and the need for smoking cessation.

Thousands of New Zealand smokers with COPD continue with their hazardous smoking behaviour without a true understanding of the risk. It is interesting to note that the same argument of protection of patients from guilt is rarely applied to lung cancer, where the link is widely acknowledged.

We are fortunate in New Zealand to have a wide range of smoking cessation support available throughout the country, and evaluations of these services show good success rates.\textsuperscript{12,13} Economic evaluations of smoking control interventions have demonstrated their cost-effectiveness and highlighted the value of investing in smoking cessation activities.

For example, a recent Ministry of Health study analysed the cost-effectiveness of subsidised Nicotine Replacement Therapy (NRT) made available through the Quitline service and through the Quit Cards programme, which provides subsidised NRT to primary healthcare providers.\textsuperscript{14}

The study included an analysis comparing the estimated costs associated with these programmes and the health gains from long-term smoking cessation measured in Quality-adjusted Life Years (QALYs) with an estimate of NZ$2,942 per QALY.\textsuperscript{14,15} This compares favourably with cost per QALY estimates for a range of other health concerns such as NZ$3,478–$7,434 for green prescriptions, $25,000 for kidney transplantations, and $35–$50,000 for dialysis.\textsuperscript{14,15} Smoking cessation, which combines support with heavily subsidised NRT, is highly cost-effective.

We need to be vigilant in correctly diagnosing COPD. Most of these patients will have developed the disease after a long period of smoking. Medical practitioners need to be aware of the diagnostic problems especially in relation to asthma. Smoking cessation is the only effective measure which will alter the natural history of this disease, and medical practitioners have a major role in initiating the process of assisting a smoker to quit.

The combination of simple advice and heavily subsidised NRT is very cost-effective and needs to be easily available.

**Author information:** Peter Martin, Medical Advisor; Helen Glasgow, Director, The Quit Group; Jane Patterson, Director, Asthma and Respiratory Foundation of New Zealand, Wellington
Correspondence: Peter Martin, Medical Advisor, The Quit Group; PO Box 12 605, Wellington. Fax: (04) 470 7632; email: peter.martin@quit.org.nz

References:


2. Personal communication from the Quit group; data sourced from the NZ Ministry of Health Public Health Intelligence.


Lung cancer in Maori: a neglected priority

Matire Harwood, Sarah Aldington, Richard Beasley

Cancer is the leading cause of death in New Zealand and lung cancer dominates as the most common cause of death from cancer.\(^1,2\) Its high incidence and poor prognosis make it an important public health issue.

Despite its importance, there has been little research in New Zealand into the causes, prevention, or screening programmes for lung cancer—or its investigation and management. Therefore, it is timely that, in this issue of the Journal, Shaw et al have reported trends in the incidence and mortality rates of lung cancer by ethnicity and socioeconomic status for people living in New Zealand.\(^3\)

Disturbingly, the study shows that (despite a reduction in overall rates) lung cancer inequalities by ethnicity and socioeconomic position have remained static or increased in New Zealand from 1981 to 1999.\(^3\) The lung cancer rates for Maori are particularly concerning. The death rate from lung cancer in Maori is three times higher than in non-Maori, and the average age of death from lung cancer in Maori is lower (63 years compared to 70 years) than non-Maori. Furthermore, the incidence of lung cancer in New Zealand Maori is, without exception, the highest in the world.\(^1,2\) The reasons for this ‘unenviable distinction’ need to be explored and addressed.

The association between lung cancer and smoking tobacco is well documented,\(^4\) and the high rates of tobacco smoking in Maori are likely to contribute to the high incidence of lung cancer observed. However, many communities in Asia and Europe have similar rates of smoking, yet lower lung cancer rates. This suggests that other factors (acting independently of or together with tobacco smoking) make Maori more susceptible to developing lung cancer.

Importantly, the proportion of cases of lung cancer in Maori that are due to tobacco smoking has never been ascertained, and remains unknown. While it is assumed that almost all cases of lung cancer in Maori are due to smoking, this is unlikely to be the case. Environmental tobacco smoke (passive smoking), smoking marijuana, occupational exposures, diet, socioeconomic status, or level of deprivation are also likely to play a role in the pathogenesis of lung cancer in Maori.

The prevalence of asthma is also high in Maori adults and there is evidence that this chronic inflammatory disorder of the airways is also a risk factor for lung cancer.\(^5\) The role of these, and other potential risk factors, requires further exploration, as does the efficacy of related prevention programmes.

Currently lung cancer risk reduction programmes tend to focus on reducing tobacco smoking, however, these have made little impact on smoking rates in Maori. Despite widespread public health programmes and other initiatives, the rate of smoking in Maori has remained around 50% over the last 20 years, during a period when the smoking rates in non-Maori have fallen substantially.

The commitment to fund nicotine replacement therapy and ‘Quit Smoking’ programmes in Maori over recent years has been impressive, although the decision by
PHARMAC not to fund bupropion, a proven smoking cessation treatment in Maori, is indefensible and contrary to the Government’s tobacco control plan. The quality of the assessment and management of lung cancer along the care pathway is a related issue. There is circumstantial evidence of inequalities in the care of Maori with lung cancer, and that this results in worse outcomes. For example, the ratio of Maori to non-Maori mortality for lung cancer is higher than that for lung cancer incidence (3.5 for mortality compared with 2.8 for incidence). In other words, case fatality rates for lung cancer are higher for Maori compared to non Maori.

Possible explanations include a delay in presentation (for whatever reason) or delays in the investigation, diagnosis, staging, or treatment of lung cancer. Maori are less likely than non Maori to have their cancer staged at diagnosis and the reasons for this are not clear.

While there is no New Zealand data on whether treatment rates may also differ, ethnic differences certainly exist in the treatment of early stage lung cancer in the United States. Indeed, the lower survival rates for black patients with lung cancer compared to white patients is largely explained by the lower rate of surgery. Screening programmes for lung cancer are not available in New Zealand, and therefore early diagnosis and treatment is important. Late diagnosis of lung cancer has devastating consequences because of the limited treatment options.

The other unrecognised ethnic disparity is the high rates of lung cancer in the Pacific people, which is twice as high as in non Maori non Pacific people. It would be important that any initiatives developed to reduce the incidence of lung cancer and improve the outcomes in Maori are also implemented in the Pacific community.

In conclusion, the disparities in lung cancer rates across ethnicity and socioeconomic status in New Zealand are disturbing, and (as predicted by Shaw and colleagues) these disparities are likely to increase over time. This raises a number of issues, including whether the lack of research and public health emphasis on lung cancer may be due to a lack of concern for the Maori, Pacific, and disadvantaged populations most at risk, or whether it may be in part due to the ‘stigma, shame and blame’ related to lung cancer.

If the current trends continue, the future burden of lung cancer will fall most heavily on Maori, Pacific, and disadvantaged socioeconomic groups. Can we assure them that we are doing all we can to find solutions?

Author information: Matire Harwood, Medical Research Fellow; Sarah Aldington, Medical Research Fellow; Richard Beasley, Director, Medical Research Institute of New Zealand, Wellington

Correspondence: Professor Richard Beasley, Medical Research Institute of New Zealand, PO Box 10055, Wellington. Fax: (04) 472 9224; email: richard.beasley@mrinz.ac.nz

References:


Scope for regulation of cigarette smoke toxicity according to brand differences in toxicant emissions

Murray Laugesen, Jefferson Fowles

**Abstract**

**Aims** To explore the scope for regulating to reduce the toxicity of manufactured cigarettes sold in New Zealand (NZ), based on published toxicant emissions by brand.

**Methods** Internet searches of published cigarette smoke emissions of 13 toxicants chosen on risk assessment principles, for 20 British Columbian, 15 Australian brands, and one NZ brand, Holiday Extra-mild (HEM), tested by Health Canada intensive smoke machine method at Labstat Inc, Kitchener, Ontario, as a ratio of toxicant to nicotine yield. We estimated relative overall smoke toxicity per disease group and per brand, after adjusting for the published cigarette-attributed mortality fractions for cancer, cardiovascular, and respiratory disease.

**Results** After allowing for nicotine yield, filter ventilation, and compensatory over-smoking, there were significant differences between brands, with the NZ brand estimated to be the most toxic. Low-yield cigarettes (<0.9 mg nicotine ISO) were estimated to be on average 19% more potent overall than medium-yield cigarettes (p<0.01). Of toxicants identified and measurable in smoke; 1,3-butadiene accounted for 45% of cancer potency; hydrogen cyanide for 89% of cardiovascular; and acrolein for 97% of respiratory potency—these three toxicants accounting for 65% of identified brand potency. Individual toxicant emissions varied across brands by a factor of 1.5 for carbon monoxide, to 32 for lead. Compared with HEM, one Canadian brand, ‘Export A full flavor’, carried a 37% lower cancer risk. This lower risk was largely due to differences in nicotine yield, lowering the toxicant/nicotine ratio.

**Conclusions** Cigarettes, unregulated, are unduly dangerous. Though many smoke toxicants cannot yet be quantified, risk assessment based on current data suggests that regulation could partly reduce identifiable cancer risk, and possibly eliminate the excess cardiovascular and respiratory toxicity of HEM, when compared with regular Canadian brands. The first goal should be to reduce emissions of the leading three toxicants, in addition to more effective charcoal filters. Tobacco smoke, unlike unburnt or non-smoking tobacco, contains toxic gases and trillions of reactive oxygen species molecules per puff, and will remain inherently harmful. Regulation could usefully part-reduce smoke toxicity exposure for continuing smokers, while not relenting on efforts to assist smokers and society to be quit of smoking.

Since 1990 in New Zealand, the Smoke-free Environments Act has provided powers at Section 31 to regulate harmful constituents ‘contained in or generated in the smoke’ of tobacco products, once the harmful constituents are named in regulations. The Ministry of Health’s regulatory review of the regulations in 2004 included consultation for regulations needed to give effect to Section 31, which also gives powers to remove fire accelerants from paper, and to reduce the nicotine content of cigarettes. Improved nicotine delivery devices are needed, and more effective...
smoking cessation methods. This paper, however, confines itself to the regulated reduction of cigarette smoke toxicity.

Until now, the New Zealand comprehensive tobacco control programme\(^2\) has focused on reducing prevalence and consumption. A national quit campaign began in 1999. A law change in 2003 banned smoking in all workplaces and hospitality venues from December 2004. Smoke toxicity per cigarette, has however, escaped policy attention.

This paper explores the feasibility and scope for reducing the toxicity per cigarette with respect to cancer, cardiovascular, and respiratory diseases. Scope for reducing toxicity seemed considerable—by the ISO machine test, tar varied 100-fold among New Zealand brands;\(^3\) and carcinogen NNK nine-fold among Marlboro brands from 30 countries.\(^4\) What was lacking was a method of assessing the combined risk from the various toxicants, and of scoring the overall toxicity of each brand. Smoke constituents include toxicants, such as carbon monoxide (CO), other vapour phase gases, particulates (tar); and the main addictive constituent, nicotine.

**Cancer risk**—Ethylene oxide, a known human carcinogen, has been found in the vapour phase of cigarette smoke of fumigated and unfumigated tobacco\(^5\) and in cigarette smoke,\(^6\) but has not been tested alongside other smoke carcinogens, even in British Columbia. Also un-quantified is the contribution of tumour promoting agents (such as free radicals) in the smoke stream, which almost certainly increases the cancer potency of the mixture in relation to the sum of its individual carcinogens.

**Respiratory disease risk**—Toxicants, such as aldehydes and acrolein, paralyse respiratory cilia, and work with reactive oxygen species (ROS), estimated at 10\(^{14}\) molecules per puff, to kill alveolar and bronchial epithelial cells.\(^7\) ROS in the vapour phase exist so briefly that their measurement is not possible, and may account for considerable unmeasured toxicity.

**Cardiovascular disease risk**—Nicotine on its own increases heart rate but not cardiovascular risk—in a randomised controlled trial of 3900 smokers chewing nicotine gum over 5 years, neither its use nor its dose was associated with cardiovascular hospitalisation or mortality rates.\(^8\) Hydrogen cyanide (HCN) inhibits cytochrome-c oxidase, blocking the cell from using its oxygen; the nervous system and the heart are particularly sensitive. For this reason, the Californian Environmental Protection Agency website assesses HCN as the most potent cardiovascular toxicant in smoke.\(^9\) Sublethal doses can result in vascular lesions and myocardial toxicity in animals.\(^10\) In fire victims, HCN may potentiate the hypoxic effect of CO. HCN in plasma has a half-life of only 14 minutes\(^10\)—so that smoking should seldom lead to rising HCN levels during the day. Much less is known of the effects of small repeated exposures.

Cigarette smoking carries a 10-fold excess sudden-death risk, unmatched by any other coronary risk factor, and a 3.6 fold excess risk of myocardial infarction.\(^11\) This risk is not due to nicotine as such\(^8\) or to oral tobacco, which has a disputed effect if any on cardiovascular risk.\(^12\) Carbon monoxide, hydrogen cyanide, and nicotine, by increasing demands on the heart via the sympathetic nervous system, all tend to create a deficit of usable oxygen in the myocardium. Cigarette smoking and oral snuff both increase plasma cotinine—but only smoking facilitates the formation of thromboxane \(A_2\) which enhances platelet stickiness and aggregation.\(^13\)
Free radicals in smoke, through increasing platelet aggregation, platelet activation, and inflammation, also play a critical role in cardiovascular disease. Cigarette smoking also inhibits the formation of endothelial nitric oxide, which normally protects the endothelium from platelet aggregation; fresh cigarette smoke contains 100–600 mcg of nitric oxide per cigarette, which oxidises to nitrogen dioxide over a few minutes. Nitric oxide smoke emission is proportional to the nitrate level in unburnt tobacco, but was not ranked as a smoke toxicant.

Identification of leading toxicants—The leading smoke toxicants identified are largely products of combustion, and in the gas phase, rather than in the particulates. Most of the estimated toxicity was found to have come from a limited number of constituents in the smoke. The usual cellulose acetate filter traps particulates, but not gases.

Method of testing—Regulation of cigarette smoke emissions for harm reduction purposes requires a method of inter-brand comparison which reflects the inhaled volume of mainstream smoke and the resultant toxicant exposure of the smoker. Since 1990, New Zealand regulations have prescribed the ISO smoking machine method for measuring emissions as the basis of tar ratings on the side of the cigarette pack. This method tends to underestimate the average amount of mainstream smoke inhaled per cigarette from all cigarettes, and particularly from low yield cigarettes. The smoker’s toxicant exposure is correspondingly underestimated:

- Across 32 studies, American smokers took an average 43-ml puff on average every 34 seconds. No puffing data on New Zealand smokers were available.
- In low yield (nicotine and tar both lowered) brands, smokers inhale more per puff (compensatory smoking) than with medium yield or ‘full flavour’ brands, thus increasing toxicant exposure above that indicated by machine-measured emissions.
- Ventilated filters used in low tar brands since the 1990s, contain tiny perforations in the filter, through which air is drawn, diluting the mainstream smoke. Smoking machines take standard-sized puffs, so ventilated filters reduce machine-measured tar, and CO per puff, a marketing advantage.
- In contrast, smokers tend to respond to ventilated filters by taking larger puffs as above, and/or in the case of very low yield cigarettes, by blocking the vents with their fingers or lips, inhaling more toxicant than the machine readings indicate.

Method

Selection of emission measurement methods that approximate human smoke exposure—To obtain a machine reading to better approximate human smoking, and in the absence of puffing measurements for New Zealand smokers, the machine smoking formula used in this paper to compare brands, is: toxicant exposure equals toxicant yield (as tested) divided by nicotine yield (as tested). The machine smoke test used was the Health Canada intensive method.

Estimation of toxicity of the emissions—The state of California has recognised over 800 compounds as causing cancer, of which the 158 found in cigarette smoke were reviewed. Of these, cancer potency units (the greater the CPU the greater the cancer risk at a given dose) were available for 40 known or suspected human carcinogens. CPUs were multiplied by per cigarette smoke emissions and divided by 20m³, the daily breathing rate, to estimate the cancer risk index (CRI) for each carcinogen. For other diseases, 17 toxicants with published reference exposure levels (RELS) were found: the lower the REL the greater the risk at a
given dose. Yield levels divided by RELs, and by 20m³, measured the non-cancer risk index (NCRI) for each non-cancer toxicant.

**Selection of brands**—We included all commercially available filter-tipped manufactured cigarette brands with published emissions data from intensive machine smoking, tested at the same independent laboratory—Labstat Inc, Kitchener, Ontario—19 medium nicotine brands (15 British Columbian, four Australian brands), and 17 low nicotine brands (five British Columbian, 11 Australian, and New Zealand’s top-selling low nicotine brand, Holiday Extra mild[20]).

The overseas data were from overseas websites.[21][22] The Canadian brand ‘Players Premiere king size’ was not included, as that brand is no longer sold; its nicotine yield was 1.87 mg—apparently unacceptable to Canadian smokers.[23] Eclipse, a brand that ‘heats rather than burns’ tobacco, Omni, a cigarette including palladium in its tobacco, and many other brands were excluded due to the lack of full published toxicant emission data based on intensive smoking machine methods.[24]

**Selection of toxicants**—To compare brand toxicity, we selected the top 10 carcinogens with the highest cancer risk index per cigarette per day[11] (Table 1) after eliminating acetamide, thought to act through a non-genotoxic mechanism of carcinogenesis. For cardiovascular and respiratory risk, we included the known toxicants[9] as listed in Table 2.

For cardiovascular toxicity, hydrogen cyanide, carbon monoxide, and arsenic[9] were included, but cresols (a separate and therefore expensive test accounting for 7% of cardiovascular toxicity) were omitted, as was phenol (0.5% of cardiovascular toxicity). Some toxicants had multiple toxic effects, so that 13 toxicants, as listed in Table 1 and 2, were sufficient to compare brands: carbon monoxide, hydrogen cyanide, arsenic, lead, cadmium, and chromium; acetaldehyde, acrolein, and formaldehyde; and butadiene, acrylonitrile, benzene, ethylene oxide, and NNN. Cancer potency estimates were not available for NNK. Ammonia and pH were measured, but not used to estimate the free base form of the nicotine. Nicotine was noted (not as a toxicant) to permit estimation of toxicant to nicotine emission ratios, as a measure of toxicant exposures.

**Estimation of toxicity by cause-of-death grouping**—The Californian Environmental Protection Agency database lists each toxicant’s target disease groupings.[10] Based on the toxicant emissions in each brand’s smoke, the cancer, cardiovascular, and respiratory risks were calculated for different brands, separately for ISO and for intensive machine testing. For example, the cardiovascular risk index was estimated as in Table 2 as the sum of NCRIs for hydrogen cyanide, arsenic, benzene, and carbon monoxide in the mainstream smoke of that brand, with cresols and phenol not measured on this occasion.

**Estimation of overall brand toxicity**—We then weighted the relative toxicity estimate for each disease group according to each group’s relative contributions to New Zealand cigarette mortality in 2000, whereby 39% of cigarette deaths were attributed to cancer, 26% to cardiovascular, and 25% to respiratory mortality. Another 10% were due to other medical causes of death, which have not been attributed to specified toxicants.[25]

For each brand and disease group, we standardised the toxicity against the average toxicity of the 15 British Columbian medium nicotine brands under intensive machine smoking, scored as 1.00—based on the fact that British Columbia was the only jurisdiction which had published tests on all brands sold. To estimate the scope for toxicity reduction for smokers of the popular New Zealand HEM brand, we compared its toxicity with that of the least toxic of the other 36 brands reviewed.
### Table 1. Carcinogenic toxicants in the smoke of New Zealand Holiday Extra-mild cigarettes, 2002

<table>
<thead>
<tr>
<th>Toxicant (V=mainly in vapour phase; P=mainly in particulate phase)</th>
<th>Cancer potency factor (mcg/m³)-¹</th>
<th>Yield of toxicant in mainstream smoke mcg per cigarette, intense method</th>
<th>Toxicant fraction of total cancer toxicity % *</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,3 Butadiene (V)</td>
<td>0.00017</td>
<td>94.6</td>
<td>44.6</td>
</tr>
<tr>
<td>Acrylonitrile (V)</td>
<td>0.00029</td>
<td>18.7</td>
<td>15.1</td>
</tr>
<tr>
<td>Acetaldehyde (V)</td>
<td>0.0000027</td>
<td>1198</td>
<td>9.0</td>
</tr>
<tr>
<td>Benzene (V)</td>
<td>0.000029</td>
<td>80.7</td>
<td>6.5</td>
</tr>
<tr>
<td>Formaldehyde (V)</td>
<td>0.000006</td>
<td>150</td>
<td>2.5</td>
</tr>
<tr>
<td>Ethylene oxide (V)</td>
<td>0.000089</td>
<td>9</td>
<td>2.2</td>
</tr>
<tr>
<td>Cadmium (P)</td>
<td>0.0042</td>
<td>.0557</td>
<td>0.65</td>
</tr>
<tr>
<td>NNN (P)</td>
<td>0.0004</td>
<td>.0187</td>
<td>0.02</td>
</tr>
<tr>
<td>Lead (P)</td>
<td>0.000012</td>
<td>.0397</td>
<td>0.01</td>
</tr>
<tr>
<td>Arsenic (P)</td>
<td>0.0033</td>
<td>0.00163*</td>
<td>0.01</td>
</tr>
<tr>
<td>Others identified, not measured for this brand**</td>
<td></td>
<td></td>
<td>19</td>
</tr>
</tbody>
</table>

* Fraction based on ISO readings, Fowles and Dybing 2003. **Arsenic was present but not quantifiable. The value given is half of the lowest detected amount in the smoke of Canadian brands. **Estimated risk includes approximately 19% from carcinogens of known potency not measured for this cigarette (Based on reported ISO yields for all carcinogens **Table 1). Of the top 10 carcinogens, acetamide and N-nitrosopyrrolidine were not measured. Chromium was present but not quantifiable.

### Table 2. Cardiovascular and respiratory toxicants in the smoke of New Zealand Holiday Extra mild cigarettes, 2002

<table>
<thead>
<tr>
<th>Toxicant (V=mainly in vapour phase; P=mainly in particulate phase)</th>
<th>Reference exposure level for minimum toxic effect (mcg/m³)</th>
<th>Yield of toxicant in mainstream smoke mcg per cigarette, intense method</th>
<th>Toxicant as percentage of total group toxicity %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydrogen cyanide (V,P)</td>
<td>3</td>
<td>282</td>
<td>89.1</td>
</tr>
<tr>
<td>Arsenic (P)</td>
<td>0.03</td>
<td>0.00163</td>
<td>0.05</td>
</tr>
<tr>
<td>Carbon monoxide (V)</td>
<td>10,000</td>
<td>26400</td>
<td>2.5</td>
</tr>
<tr>
<td>Benzene (V)</td>
<td>60</td>
<td>80.7</td>
<td>1.3</td>
</tr>
<tr>
<td>Cresols (V)</td>
<td>4</td>
<td>NM (19.7#)</td>
<td>7.0**</td>
</tr>
<tr>
<td>Phenol (V)</td>
<td>600</td>
<td>NM (26.1#)</td>
<td>0.05**</td>
</tr>
<tr>
<td>Respiratory:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acrolein (V)</td>
<td>0.02</td>
<td>148</td>
<td>97.1</td>
</tr>
<tr>
<td>Acetaldehyde (V)</td>
<td>9</td>
<td>1198</td>
<td>1.8</td>
</tr>
<tr>
<td>Formaldehyde (V)</td>
<td>2</td>
<td>150</td>
<td>1.0</td>
</tr>
<tr>
<td>Acrylonitrile (V)</td>
<td>2</td>
<td>18.7</td>
<td>0.12</td>
</tr>
<tr>
<td>Cadmium (P)</td>
<td>0.01</td>
<td>0.0557</td>
<td>0.07</td>
</tr>
<tr>
<td>Chromium (P)</td>
<td>0.0008</td>
<td>NQ</td>
<td>0</td>
</tr>
<tr>
<td>Others (dioxins, nickel)</td>
<td></td>
<td></td>
<td>.0003</td>
</tr>
</tbody>
</table>

NQ= Not quantifiable; NM=Not measured; *Arsenic was detected, but was not quantifiable. The value given is half of that of the lowest level detected in the smoke of Canadian brands; ** Machine smoke measured under ISO conditions. Fowles and Dybing 2003. **Fraction based on ISO readings, from Fowles and Dybing 2003. www.tobaccocontrol.com
Table 3. Top three ranking smoke toxicants of manufactured cigarette brands, tested by the intensive method; estimated identifiable cancer, and overall toxicity by brand.

<table>
<thead>
<tr>
<th>Brands by country (number tested)</th>
<th>Nicotine yield mcg/cigarette, mean, SD=standard deviation, (range)</th>
<th>Acrolein mcg/cigarette, mean SD, (range)</th>
<th>Hydrogen cyanide mcg/cigarette mean, SD (range)</th>
<th>1:3, Butadiene mcg/cigarette, mean, SD (range)</th>
<th>Identifiable cancer toxicity relative to Canadian medium-nicotine brand average mean, SD (range)</th>
<th>Identifiable overall brand toxicity relative to Canadian medium-nicotine brand average=1.00 mean, SD (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Low nicotine brands</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ (1) HEM</td>
<td>1.8</td>
<td>81</td>
<td>155</td>
<td>52</td>
<td>0.55</td>
<td>1.39</td>
</tr>
<tr>
<td>Australia (11)</td>
<td>1.9 SD 0.26 (1.5-2.2)</td>
<td>62</td>
<td>124</td>
<td>48</td>
<td>0.50 SD 0.05 (0.42-0.57)</td>
<td>1.18 SD 0.10 (1.00-1.33)</td>
</tr>
<tr>
<td>British Columbia (5)</td>
<td>2.3 SD 0.51 (1.7-2.9)</td>
<td>64</td>
<td>123</td>
<td>30</td>
<td>0.42 SD 0.05 (0.37-0.48)</td>
<td>1.10 SD 0.16 (0.90-1.27)</td>
</tr>
<tr>
<td><strong>Medium nicotine brands</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australia (4)</td>
<td>2.4 SD 0.11 (2.3-2.5)</td>
<td>40</td>
<td>98</td>
<td>38</td>
<td>0.40 SD 0.03 (0.36-0.43)</td>
<td>0.92 SD 0.06 (0.88-1.00)</td>
</tr>
<tr>
<td>British Columbia (15)</td>
<td>2.8 SD 0.32 (2.2-3.2)</td>
<td>57.2</td>
<td>104.0</td>
<td>34.1</td>
<td>0.39 SD 0.06 (0.32-0.51)</td>
<td>1.00 SD 0.11 (0.85-1.24)</td>
</tr>
<tr>
<td>Export A FF (least toxic)</td>
<td>2.9 % change HEM to Export A FF</td>
<td>-42</td>
<td>-49</td>
<td>-37</td>
<td>-37</td>
<td>-39</td>
</tr>
</tbody>
</table>

HEM = Holiday Extra-mild brand, 0.6 mg nicotine yield on ISO testing; Export A full flavor, a Canadian cigarette, 1.3 mg nicotine yield on ISO testing; Low nicotine ≤0.9 mg yield on ISO smoke machine test condition; Medium = 0.9 to 1.3 mg nicotine, ISO; Except for nicotine values, all values are nicotine-adjusted (toxicant value divided by nicotine value, both measured under intense smoking condition); Note: The small number of Australian and New Zealand brands tested may not be representative of their markets: the New Zealand brand was higher, and the Australian brands lower, than the average for Canadian brands = 1.00. In British Columbia all main brands on sale were tested.
Results

- The mainstream smoke of all cigarette brands studied contained the same leading carcinogenic, cardiovascular, and respiratory toxicants in their smoke.

- Of the measurable, identifiable toxicants, the three most significant in all brands studied were acrolein, butadiene, and hydrogen cyanide. These three accounted for 65% of overall identifiable toxicity.

- Toxicants in the vapour phase accounted for over 80% of the HEM cigarette’s toxicity. Overall, toxicants in the particulate phase (for which tar is a proxy measure) accounted for no more than 18% of overall cigarette smoke toxicity.

- The 13 toxicants tested accounted for 81% of identifiable cancer risk, 99.5% of identifiable cardiovascular toxicity, 99.9% of the identifiable respiratory toxicity, and 83% of the overall identifiable cigarette smoke toxicity.

- For Canadian regular brands, the ratio of intensive to ISO emissions approximately doubled, with less increases for some heavy metals. Arsenic was detected in measurable quantity in one brand’s smoke only, from Canada.

- The toxicant/nicotine ratio was virtually the same by either ISO or intensive method for medium and low nicotine cigarettes considered as a combined group. Under intense smoking conditions, HEM tar was 33 mg per cigarette, HEM nicotine 1.8 mg; and the tar/nicotine ratio 18, the highest for any brand. Samples of HEM purchased 6 months later, under intense machine smoking conditions, tested tar at 29 mg, nicotine 1.7 mg; tar/nicotine ratio 17. (ISO results for both samples were 9 mg tar, 0.6 mg; ratio 15).

- The tobacco-specific nitrosamine NNN for HEM in Table 1, and its ratio to nicotine was the lowest among all 37 brands.

Table 1

Four carcinogens accounted for 76% of the identifiable cancer risk of HEM: 1,3-butadiene (45%); and acrylonitrile, acetaldehyde, and benzene, a further 31%. Ethylene oxide, a known carcinogen, is included here—using the reported value of 9 ug/cigarette. To allow for identifiable carcinogens not measured, we left 19% of the carcinogenic risk unattributed to any toxicant, on the basis of published CPUs and ISO emissions. Using published tables based on ISO smoking conditions, Table 1 87% of the identifiable carcinogenic risk was in the vapour phase, and 13% in the particulate phase.

Cancer risk based on CRIs versus cigarette cancer mortality—We updated the previous estimate of the percentage of cigarette cancer deaths accounted for by CRIs, using lifetime lung cancer risks from the American Cancer Society’s Cancer Prevention Study II in the 1980s, against CRIs estimated using intensive smoking machine testing results from Table 1, otherwise using yield data from Table 1 scaled upwards to mimic intensive smoking, using the intense to ISO ratio (2.15) for summary CRIs for emissions as reported for Canadian regular cigarettes. On this basis, approximately 35% of lifetime cigarette cancers (lung and other sites) were explained by CRIs (27% of lung cancer in men, and 76% of lung cancer in women).
Table 2

Hydrogen cyanide was found in both vapour and particulate phases. Its very low REL indicated that even one cigarette smoked was sufficient to exceed this threshold and produce a toxic effect. Hydrogen cyanide made up 89% of the identifiable and measurable cardiovascular toxicity of HEM mainstream cigarette smoke, and carbon monoxide only 2.5%. For respiratory toxicity, acrolein, and cadmium were the most toxic per unit weight but when the quantity in smoke was considered, acrolein made up 97% of the estimated respiratory toxicity of HEM smoke. Vapour-phase toxicants contributed over half of cardiovascular toxicity and virtually all of respiratory toxicity.

Table 3

For the three most powerful toxicants, HEM had the highest toxicant/nicotine ratios among the 37 low and medium nicotine brands. On toxicant emissions alone, it was not the highest. The overall toxicity per brand in the far right column attributes 10% of relative toxicity to those toxicants (unknown, so not reducible by any known means) responsible for ‘other medical’ causes of death. This accounts for the somewhat lower reductions obtained in the last column, compared with the second to last column for cancer.

The lead level in HEM smoke was second highest of 37 brands at 40 nanograms per cigarette, and the highest for lead/nicotine. In contrast, HEM gave the lowest NNN/nicotine ratio. The NNN/nicotine ratio varied by a factor of 6.4 between brands. Nicotine varied among medium nicotine brands from 0.9 mg to 1.3 mg per cigarette when tested under ISO smoking conditions (not shown in Table 3), and varied more, from 1.5 to 3.2 mg per cigarette, under intense machine smoking.

Among low nicotine brands, nicotine varied from 0.4 mg to 0.89 mg under ISO smoking conditions, and from 1.5 to 2.9 mg under intense smoking conditions. Thus under intensive smoking conditions, ‘low’ and ‘medium’ nicotine cigarettes gave similar values and ranges for nicotine yields. Across brands, the toxicant to nicotine ratios varied from 1.8 for tar/nicotine by either method, to 3.3 to 3.4 for acrolein/nicotine, for ISO and intensive methods respectively. Under ISO test conditions, HCN/nicotine and butadiene/nicotine varied four fold between brands, but under intensive test conditions both varied less—HCN/nicotine varied 2-fold and butadiene/nicotine 2.4-fold.

Comparing relative toxicity, low yield brands (relative average toxicity 1.17, SD 0.16) were 19% more potent overall than medium yield brands (relative average toxicity 0.98, SD 0.11), based on the toxicant to nicotine ratio (p<0.01), standardised against Canadian medium-nicotine brands (relative toxicity 1.00).

Spreadsheet estimates show that if HEM nitrosamine (NNN) emissions were increased five-fold to the levels seen in the US Marlboro brands, HEM’s overall toxicity would have increased by (only) 0.1%.
Discussion

The main findings

• Holiday Extra-mild, New Zealand’s most popular mild cigarette, was the most toxic of the 37 brands for which published toxicities were available, based on toxicant/nicotine ratios.

• The method described shows scope for down-regulating individual toxicant emissions, and total brand toxicity, to that of lower emission brands.

• Export A full flavor, the least toxic cigarette, was estimated at 39% less toxic than HEM. Export A’s reduced toxicity was mostly due to its higher nicotine emission, and the correspondingly lower toxicant/nicotine ratio.

• Focusing on the 13 leading toxicants among the 4000 chemicals in tobacco smoke makes regulation feasible. The top three accounted for 65% of the cigarette’s toxicity. The dominant toxicant for cancer was 1,3-butadiene for cancer; for cardiovascular disease, HCN; for respiratory disease, acrolein. The emissions of these toxicants varied considerably between brands, suggesting that much of the toxicity of a cigarette can be influenced by cigarette engineering or the tobacco blend used.

• Under intensive smoking conditions low nicotine brands tended to have higher toxicant emissions than medium nicotine brands, as judged by toxicant/nicotine ratios.

Strengths and weaknesses of this study

Strengths—For the first time, this study shows how cigarette brands can be scored by a summary measure of overall brand toxicity, based on leading toxicants and their target organs, assessed by toxicological risk assessment methods.

Test results includes all brands with published test results from the same independent laboratory, Labstat Inc, using Health Canada intensive smoking conditions; others were not considered.

Weaknesses and limitations—Reproductive and developmental toxicity effects were not considered. The two leading toxicants in this category, arsenic and 1,3-butadiene, were already given weight in Table 1, and so brand rankings would not greatly change if this extra toxicity effects category was included. Only British Columbia displayed chemical emissions under intense conditions for all or most brands sold. New Zealand, thus far, has tested only one New Zealand brand (HEM) under intense smoking conditions. Holiday regular, the highest volume-selling New Zealand cigarette, was tested in 2002 by ISO method only.

The toxicant to nicotine ratio adjusts for compensatory over-smoking in low yield brands, and its usefulness for medium-nicotine brands may need reassessment once puff volume data is known for New Zealand smokers and brands. A lack of cigarette engineering information on filter ventilation, filter efficiency, or paper porosity precluded further elucidation of the reasons for brand to brand differences found in smoke emissions. The toxicity estimates are not absolute, but relative, and compare only the identifiable, measurable toxicants in mainstream smoke.
This study compares cigarettes and their machine-generated smoke, but does not allow for how smokers smoke their cigarette, and how much they inhale.

Table 1 gives the estimated cancer risk of smoking one cigarette daily over a lifetime. As estimated, the identified carcinogenic emissions account for 35% of cigarette cancer mortality. For non-cancer risk there is no method to link toxicity level to absolute levels of disease risk; we cannot estimate how much of the total cardiovascular and respiratory risk has been identified, or how much the estimated reduction in emissions will translate into reduced toxicant absorption and decreases in mortality.

The percentage differences between the toxicity of HEM and Export A full flavor in Table 3 may not reflect reductions in toxicity obtainable for the total market. HEM is the only brand fully tested to date, and its relative toxicity may be higher or lower than the New Zealand all-brand average.

**Comparison of results with current knowledge**

In a toxicity-regulated cigarette, unidentified or unmeasured toxicants in smoke, such as free radicals, may or may not reduce in parallel to the reductions anticipated for known toxicant emissions, may not do so immediately, and the time required for effective switching to reduced-toxicant brands is uncertain.

The risk assessment approach, using toxicological data largely from animal studies for toxicity, emphasises the vapour phase rather than tar or particulates (Table 1), and emphasises the vapour phase carcinogens 1,3-butadiene and benzene (Table 1) rather than the tar constituents nitrosamines, and benzo(alpha)pyrene. For cardiovascular toxicity, risk assessment emphasises HCN rather than CO (Table 2).

The test results for HEM only apply to the cigarettes sampled for testing. For tar and nicotine at least, the results were confirmed by almost identical results from cigarettes purchased 6 months later. The manufacturer (BAT) reported the HEM brand yielded 0.8 mg nicotine on ISO testing in 2002, but manufacturers’ reports had not previously been checked by an independent laboratory.

**What this study means**

Overall smoke cancer risk indices can be estimated, and non-cancer indices also, but with less certainty. The overall toxicity of brands can be compared, based on each toxicant’s potency per microgram and the amount of each in that brand’s smoke. The relative toxicity scores for each brand allow comparison of brands across countries and time periods, if identical methods and, as in this study, the same laboratory is used. Potency factors may be revised by expert groups from time to time, and emissions will change also. Thus relative toxicity scores may need revision at least annually.

**The toxicant to nicotine ratio**—This ratio could only be used because all the cigarettes were of commercial design, so that tar and nicotine varied within narrow limits. Research cigarettes if low in nicotine yield at 0.05 mg, and with tar at 10 mg,—have a tar/nicotine ratio of 200:1.

**Cancer risk** (Table 3)—As addicted smokers may tend to seize on any excuse to keep smoking, undue claims of lessened toxicity or disease prevention are unhelpful.
Conversely, unduly conservative estimates may discourage regulators from removing excess toxicants. This paper suggests that regulation based on exploiting existing brand differences (without using charcoal filters) could lower the identifiable cancer risk of HEM by 37%. As identifiable risk represents about 35% of total cigarette cancer risk, the overall total cigarette cancer risk reduction achievable (if, and only if, this brand’s toxicity was representative of all brands) would be (0.35 x 37%) =13% or 224 of the 1732 cigarette cancer deaths in 2000.25

**Non-cancer toxicity.** (Table 2)—With respiratory disease, and particularly with cardiovascular disease, we lacked sufficient clinical or toxicological data to determine the total non-cancer fraction of total toxicity represented by the toxicants in Table 2.

**Overall toxicity** (Table 3)—If the cigarette with the highest overall toxicological risk estimate (HEM in this case) was re-engineered to achieve the toxicant/nicotine emission of the average of 15 Canadian medium nicotine brands, its identifiable toxicity would reduce 28%. If re-engineered to achieve those of the least toxic brand, Export A full flavor, HEM’s identifiable toxicity could be reduced by 39%.

**Nitrosamines**—We confirm great variation in nitrosamine levels. Marlboro cigarettes sold in New Zealand were imported from the United States. HEM’s NNN emission in Table 1 on intensive testing is eight times lower than for US Marlboro cigarettes machine smoked less intensively.24 Regulation can force highly toxic brands off the market, but if regulation is confined to nitrosamines, it will do little for New Zealand smokers. Less than 0.1% of cigarette cancer deaths would be prevented (Table 1), and none of the 60% of cigarette deaths due to non-cancer causes.25 Comprehensive regulation of all leading cigarette toxicants is required.

**Lag times**—After implementation of the regulations, the interval before death rates decreased would vary with the disease. Half of the achievable reduction in cardiovascular risk can be expected within a year of implementation, achieving full effect on cigarette mortality within 10 to 15 years, based on the known effects of stopping smoking.29

Regulations to require regular monitoring, brand by brand disclosures of tobacco constituents and emissions, and reductions in leading emissions across all cigarettes, are now overdue. Though many toxicants remain unidentified or unmeasured this paper provides a framework for comparing and substantially reducing the identifiable toxicity of both manufactured and hand-rolled cigarettes.

**Author information:** Murray Laugesen, Public Health Physician, Health New Zealand Ltd, Devonport, Auckland; Jefferson Fowles, Senior Scientist, ESR (Institute of Environmental Science and Research), Porirua.

**Potential conflict of interest:** The authors are co-inventors for a patent *Apparatus and methods for testing toxicity of cigarette smoke*, NZP 537968, 28 January 2005.

**Acknowledgements:** ASH New Zealand Inc. (Action on Smoking and Health) Auckland (www.ash.org.nz) funded this study.

**Correspondence:** Dr Murray Laugesen, Health New Zealand Ltd, PO Box 32 099, Devonport, Auckland. (0274) 884 375; email: laugesen@healthnz.co.nz
What this paper adds

This is the first published report comparing the overall toxicity of cigarette brands across countries using risk assessment and intensive machine smoking, and adjusting for smoke nicotine and the mortality distribution between disease groupings. The method provides a relative toxicity score as a rational basis for regulating cigarette emissions across all brands sold.

Holiday Extra-mild’s overall estimated identifiable toxicant emission levels would reduce 39%, and its cancer risk by 37%, if this cigarette was required by regulation to have the same emissions as a certain Canadian regular brand, Export A full flavor. Regulation to reduce brand differences in emissions, without employing charcoal filters, would reduce total cancer risk by 13% for Holiday Extra-mild, based on measurable toxicants. Any toxicity reduction from including a charcoal filter would be additional.

Glossary

Yield or emission (mainstream smoke), or potential exposure The weight of toxicant or harmful constituent collected from the mouth end by smoking the cigarette by machine.

Low nicotine or low yield Nicotine less than 0.9 mg per cigarette on ISO test.

Medium nicotine or medium yield Nicotine 0.9 mg or more per cigarette on ISO test.

NNN N-Nitrosonornicotine, a tobacco-specific nitrosamine.

NNK 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone, a tobacco-specific nitrosamine.

Standard ISO or FTC machine smoking Machine takes a puff of 35 ml, puff lasts 2 seconds, 60 seconds between puffs. No taping of the filter.

Intensive machine smoking (Health Canada) Machine takes 55 ml puff, puff lasts 2 seconds, 30 seconds between puffs. Tape covers all of filter and tipping paper.

References:


18. Kozlowski LT, O’Connor RJ. Cigarette filter ventilation is a defective design because of misleading taste, bigger puffs, and blocked vents. Tobacco Control. 2002;11:Suppl i40–i50.


Scope for regulation of cigarette smoke toxicity: the case for including charcoal filters

Murray Laugesen, Jefferson Fowles

Abstract

Aim To compare the emissions toxicity of two manufactured cigarette brands, one with and one without a charcoal filter, in the light of manufacturers’ laboratory research findings on the properties of charcoal filters.

Method Emissions of Mild Seven charcoal filter brands, regular (labelled ‘12 mg tar’) and Light (labelled ‘9 mg tar’) purchased in 2004, were compared with those of Holiday Extra-mild brand (9 mg tar, acetate filter), purchased in 2002. All emissions were tested under intensive machine smoking conditions by Labstat International Inc., Kitchener, Ontario.

Results The Mild Seven brands contained a small amount of charcoal, its black granules visible against the white acetate filter. The charcoal filter in the brands tested did not reduce toxicity to the extent expected, though they gave significantly lower emissions for the respiratory—totoxicants acrolein (14%–17% lower, \( p \leq 0.01 \)) and formaldehyde (26–37% lower, \( p \leq 0.01 \)). Reductions were not significant for acetaldehyde, and actually higher for hydrogen cyanide. Overall, estimated cardiovascular-respiratory toxicity was not reduced, whether based on toxicant emissions or the toxicant to nicotine ratios.

Of the packet labels, neither tar yield (mg) nor the descriptors ‘mild’, ‘light’, or ‘extra-mild’, or ‘charcoal filter’ for these three brands was associated with any reduction of the combined respiratory—and cardiovascular toxicity of mainstream smoke, as measured by leading toxicants tested by the intensive method.

Previously secret documents from cigarette companies, including British American Tobacco, reported reductions of 75%–80% in hydrogen cyanide, acetaldehyde, acrolein, and formaldehyde in mainstream smoke from addition of charcoal to the filter. We estimated that an effective charcoal filter could reduce a brand’s overall relative toxicity score for identifiable toxicant by over 40%.

Conclusion Since 1965, major cigarette firms have known from their chemists that many smoke toxicants, including hydrogen cyanide and acrolein, were removable by manufacturing the cigarette with a charcoal filter. To this day, few brands have charcoal filters. The best known, Mild Seven, contained a token charcoal filter only. In neither Japan nor New Zealand did this brand lower cardiovascular toxicant emissions in smoke. In the Smoke-free Environments Act, Government has the power to lower smoke emissions by regulation, but no regulations are in place. The Act does not give power to add filters to cigarettes, but does give power to lower smoke emissions to the level attainable by using a charcoal filter, which could reduce smoke emission toxicity to a large extent. Regulation to require effective charcoal filters is now long overdue.
A committed but concerned smoker might ask—‘Which cigarette brand has the least toxic emissions in its smoke?’ If a certain brand had significantly lower emissions, we reasoned that smokers should be informed of the fact. If there was such a brand, no cigarette manufacturer was making any such claim. In 2003, the Ministry of Health had tested only two brands for emission toxicity. By 2004, the Ministry was reviewing the Smoke-free Environments regulations, including the possibility of regulating toxicity.

We looked first at Japan, where two-thirds of cigarettes sold in the preceding 20 years had charcoal filter tips, thought to possibly explain the lower rate of lung cancer in male smokers in Japan versus the United States. The most popular Japanese brand, Mild Seven, probably the world’s most popular charcoal brand, was sold in small quantities in New Zealand. Data from the Ministry of Health in Tokyo, however, showed that the Mild Seven charcoal filter cigarette, and other regular cigarettes with charcoal filters did not lower emissions below the Canadian regular brands average as published by the Government of British Columbia. As charcoal is widely used in gas masks and in chemical laboratories to adsorb gases, the reason for these findings was unclear.

Method

Toxicant selection—Toxicants for comparing brands were selected by toxicological risk assessment.

Cigarette manufacturers’ reports and charcoal filter references—Document collections at www.tobaccodocuments.org (previously secret internal industry documents accessed by legal discovery) and www.pubmed.org (US Institutes of Medicine) were searched on acrolein, cyanide, and charcoal. In addition, Swedish Match supplied a short summary of past experiments with charcoal filters. (Wahlberg I. personal communication, 2004).

Smoke tests—Emissions from two charcoal filter brands, Mild Seven regular, labelled 13 mg tar, and Mild Seven Lights, labelled 8 mg tar, were compared with Holiday Extra Mild (9 mg tar), an acetate filter brand. (The labels for tar were based on the ISO method—puff volume 35 ml, puff interval 60 seconds, puff duration 2 seconds, no holes covered).

The Mild Seven charcoal filter brands were purchased in Newmarket, Auckland in 2004, and analysed by Labstat International Inc, Kitchener, Ontario, Canada, using the Health Canada intensive machine smoking method (55-ml puff volume, puff duration of 2 seconds, 30 seconds between puffs, and all ventilation holes covered). For comparison, we used data from Holiday Extra-mild cigarettes, purchased in Wellington in 2002, and also tested at that time by Labstat by the same intensive machine smoking method. For both studies, we tested a short list of toxicants known from industry documents to be reduced by charcoal filters. Brands were tested for tar and nicotine; for the respiratory toxicants acrolein, acetaldehyde, formaldehyde; and for the cardiovascular toxicants carbon monoxide and hydrogen cyanide.

Spreadsheet scenarios—We created emissions spreadsheets containing the observed emissions for each brand. Then we changed certain emissions to simulate the effects of regulation for charcoal filters and/or switching to brands with lower emission toxicity. We ran separate estimations of each brand’s observed toxicity (with an acetate filter), then with a combined acetate-charcoal filter, assuming the acetate functioned as before, and the charcoal removed 75% of acetaldehyde, acrolein, benzene, hydrogen cyanide, and formaldehyde, as found on emission testing of mainstream smoke from that brand of (acetate filter) cigarette.

Results

Cigarette company documents—In 1964-5, RJ Reynolds, and British American Tobacco (BAT) scientists showed that charcoal filters lowered hydrogen cyanide in smoke by 74% to 78%, and aldehydes by 75% to 95%. In the 1990s, Swedish Tobacco, before it sold its cigarette business and became Swedish Match, compared...
two 80% ventilated cigarette brands, one with a charcoal filter, the other with a
cellulose acetate filter. Charcoal reduced the gas phase components by 80% compared
with the acetate filter cigarette. The greatest reductions were found for aromatic
hydrocarbons, nitriles, and ketones. (Wahlberg I. personal communication, 2004).

**Observed effects in smoke test results**—Labstat Inc reported that in both variants of
Mild Seven charcoal filter cigarettes, the charcoal granules were mixed and
inseparable from the cellulose acetate portion of the ‘dual filter’, so that the charcoal
could not be weighed. From our own visual examination, it appeared that the charcoal
was a very minor component of the filter.

Table 1 shows that on testing by intensive machine smoking, emissions for tar,
nicotine, and carbon monoxide for all three brands were much higher than the levels
given on the packet label (which were based on the less intense ISO method).

**Table 1. Per cigarette emissions in mainstream smoke of three brands sold in
New Zealand, with and without charcoal filters, tested by intensive smoking
machine method at Labstat Inc Canada**

<table>
<thead>
<tr>
<th>Mean (standard deviation)</th>
<th>Mild Seven Regular</th>
<th>Mild Seven Lights</th>
<th>Holiday Extra Mild</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year purchased</td>
<td>2004 NZ</td>
<td>2004 NZ</td>
<td>2002 NZ</td>
</tr>
<tr>
<td>Packet label#</td>
<td>12 mg tar</td>
<td>8 mg tar</td>
<td>9 mg tar</td>
</tr>
<tr>
<td>Filter</td>
<td>Charcoal, acetate</td>
<td>Charcoal, acetate</td>
<td>Acetate</td>
</tr>
<tr>
<td>Tobacco weight mg</td>
<td>1004 (10)</td>
<td>972 (12)</td>
<td>888 (na)</td>
</tr>
<tr>
<td>“Tar” mg</td>
<td>29.4 (1.6)</td>
<td>28.4 (0.9)</td>
<td>32.6 (1.6)</td>
</tr>
<tr>
<td>Nicotine mg</td>
<td>1.91 (0.06)</td>
<td>1.71 (0.04)</td>
<td>1.82 (0.08)</td>
</tr>
<tr>
<td>Carbon monoxide mg</td>
<td>25.1 (0.7)</td>
<td>25.7 (1.5)</td>
<td>26.4 (1.0)</td>
</tr>
<tr>
<td>Formaldehyde mcg</td>
<td>111** (12)</td>
<td>94.8** (6.4)</td>
<td>150 (12)</td>
</tr>
<tr>
<td>Acetaldehyde mcg</td>
<td>1152 (47)</td>
<td>1172 (55)</td>
<td>1198 (61)</td>
</tr>
<tr>
<td>Acrolein mcg</td>
<td>123** (8)</td>
<td>128** (6)</td>
<td>148 (6)</td>
</tr>
<tr>
<td>Hydrogen cyanide mcg</td>
<td>310 (11)</td>
<td>339* (27)</td>
<td>282 (18)</td>
</tr>
</tbody>
</table>

#Value on packet label was determined by ISO method; na = not available; *p ≤ 0.05 and, **p ≤ 0.01, with respect to
the value for Holiday Extra Mild.

Compared with Holiday Extra-Mild, the Mild Seven charcoal filter brands (both
regular and lights) gave significantly lower emissions for the respiratory toxicants
acrolein (p < 0.01), and formaldehyde (p < 0.01). For acetaldehyde, a respiratory
toxicant, and carcinogen, levels were not significantly different in the charcoal brands.
Emissions of hydrogen cyanide, a cardiovascular toxicant, tended to be higher in the
smoke of the Mild Seven brands, than for Holiday Extra-Mild. On spreadsheet
simulation, assuming untested analytes were the same in all three brands, overall
relative brand toxicity for Holiday Extra Mild was intermediate between the two Mild
Seven brand variants.

**Estimated effects on overall identifiable toxicity**—Estimating from the emissions
spreadsheet, we scored the relative toxicity of the average of British Columbian
regular brands under intensive conditions as the standard, as 1.00. Adding a charcoal
filter reduced overall estimated identifiable toxicity by 43% to 0.57. The estimated
overall relative emission toxicity of Holiday Extra Mild under intensive smoking
conditions with an estimated score of 1.39, was higher than for Canadian brands.
Adding a charcoal filter similarly reduced HEM’s overall identifiable toxicity by 44% to 0.78. The emission toxicity of ‘Export A full flavor’ was among the lowest of Canadian brands at 0.85: but was estimated to be lower still at 0.49 with an efficient charcoal filter—a 42% reduction.

**Non-cancer risk**—Substituting in the emissions spreadsheet, for Canadian regular brands on average, a charcoal filter (see Method) reduced the estimated cardiovascular and respiratory identifiable toxicity by 72% and 74% respectively. For Holiday Extra-mild, the reduction was 72% and 75% respectively. No method was available for assessing what fraction of these reductions might be applicable to total non-cancer smoking risk. Thus no non-cancer mortality reduction estimate can be made.

**Cancer risk**—The Canadian regular brands (overall total relative toxicity score 1.00) provided the standard score for cancer toxicity of 0.39, based on an estimated 39% of cigarette-attributed deaths in New Zealand in 2000 being due to cancers. With a charcoal filter, cancer toxicity reduced 13%, from 0.39 to 0.34, as we assumed no reduction of 1,3-butadiene, the main carcinogen. As identifiable cancer risk represented 35% of total cancer risk, total cancer risk from smoking reduced by (0.35*13) = 4.6%. This is a smaller but more firmly based estimate than is available for cardiorespiratory risk, where the main benefits of charcoal filters can be achieved.

If regulation utilised both brand and filter differences in emission toxicity, (HEM without a charcoal filter, compared with ‘Export A full flavor’ fitted with a charcoal filter), the difference was from 1.39 to 0.49, a 65% reduction in identifiable toxicity exposure overall, and a 47% reduction in identifiable cancer toxicity exposure. These relative toxicities are based on toxicant to nicotine ratios. If identifiable cancer risk reduced 47% from 0.55 to 0.29, then total cancer risk from smoking can be lowered 47%* 0.35= 16.5% that is, by 16.5% if charcoal filters and other differences in brand toxicity are utilised. Were Holiday Extra-mild’s emissions found to be typical of New Zealand cigarettes (it is the only brand fully tested so far), then in 2000, cigarette cancer deaths could have been 280 fewer.

**Discussion**

This study shows that:

- The intensive smoking machine method gave emission toxicity levels approximately double that from the ISO method.
- Low tar ratings, ‘mild’, ‘light’, or ‘extra-mild’ labels on cigarette packets do not equate with reduced toxicant emissions.
- The ineffectiveness of the charcoal filter in Mild Seven cigarettes noted in analyses by the Ministry of Health in Tokyo is confirmed for the same brand sold in New Zealand.
- Charcoal filters are unlikely to explain differences in lung cancer rates between Japan and the United States.
- Since 1965, major cigarette companies have knowingly exposed smokers to high levels of these major smoke toxicants, which, their chemists had reported, could be greatly reduced by using charcoal filters.
Descriptors on cigarette packets—In view of current interest in banning of misleading descriptors as a possible outcome of the Ministry of Health’s current review of packet labelling, we note that of the three brands studied, whether considering the tar ratings on the side of the packet, the descriptors ‘light’, ‘extra-mild,’ or the filter description ‘charcoal filter’, none was matched by reduced toxicant emissions. Our findings illustrate the need for Government to revise its regulations and ban misleading labels on cigarette packets unless the claims can be substantiated.

Limitations of the study—Findings are limited to the brands we studied, and to the time of purchase. Manufacturers may have since changed a brand’s design or tobacco blend and consequent emissions, as most emissions are not monitored by the Ministry of Health. In the current state of science we are unable to quantitatively describe the relationship between non-cancer toxicants in smoke and future disease risk. Other smoke hazards including reactive oxygen species remain unmeasured. Some toxicants are known to interact. Also smoking machines measure the toxicant exposure of the machine, whereas smokers may inhale more intensively, and also exhale some of the gases, thus retaining and absorbing some gases more than others. Further, actual toxicant exposure implies measurement of the inhaled smoke. Combining effects from charcoal filters and other brand differences assumes independent effects, which in practice may not be fully realisable, as some interaction may be difficult to avoid.

The charcoal filter—The light dusting of charcoal found in Mild Seven filters may satisfy consumer taste and conform nominally to its label. It does not exploit charcoal’s toxicant-reducing potential. Smokers may take time to become accustomed to the taste of charcoal filter cigarettes, but if these filters are mandated across all brands, smokers would buy and smoke them. Besides Mild Seven, BAT’s Kent brand had a charcoal filter, but it also appeared to contain little charcoal. Charcoal filter cigarettes need to contain active charcoal (with greater surface areas and pores, up to 180 mg per cigarette) if they are to maximally reduce toxicant gases. Regulations will need to tilt manufacture towards maximally effective filters, against the manufacturer’s wish not to upset the smoker.

This reports of cigarette chemists over the past 40 years are in line with the large apparent reductions in emissions recently reported by manufacturers; in 2004 for the Advance brand’s trionic (charcoal, cellulose acetate, resin) filter, and in 2005 for Philip Morris’ carbon filters technology. These charcoal filter cigarettes are currently being test-marketed in the United States, but there is no sign of them being launched in New Zealand as yet.

Regulation for effective filters should extend to filters sold loose, as in New Zealand loose cigarette tobacco accounts for one-quarter of all tobacco sold for smoking, and for over half of tobacco smoked by Maori.

As the excess cumulative premature death risk of continuing to smoke is 50%, it is obvious that even if regulation reduces cigarette smoke toxicity substantially, smoking will remain a highly dangerous activity. Quitting is still the best advice.

The introduction of acetate filters in cigarettes in the 1960s led to soaring sales, encouraged by tobacco advertising. A new filter could not easily repeat this now, in the face of advertising and smoking bans, anti-smoking media campaigns, and
tobacco taxation policies. As cigarette toxicity is regulated, however, these support measures should be increased.

**Author information:** Murray Laugesen Public Health Physician, Health New Zealand Ltd, Devonport, Auckland; Jefferson Fowles, Senior Scientist, ESR (Institute of Environmental Science and Research), Porirua

**Acknowledgement** Heart Foundation of New Zealand funded this study.

**Correspondence:** Dr Murray Laugesen, Health New Zealand Ltd, PO Box 32 099, Devonport, Auckland. Email: laugesen@healthnz.co.nz

---

**What this paper adds**

This is the first published report comparing what charcoal filters can do in a cigarette company laboratory with what they do in a commercial cigarette.

Over the last 40 years cigarette company scientists have reported that charcoal can reduce aldehydes and hydrogen cyanide emissions in cigarette smoke by 75%-80%. In two Mild Seven charcoal filter brand variants sold in New Zealand containing minimal charcoal, no such reduction in these emissions was seen.

Emission reductions seen with the charcoal filters as reported 40 years ago, could today lower a brand’s overall identifiable toxicity by over 40%, mainly by reducing gases toxic to lungs, heart and blood vessels. Whether overall total brand toxicity would be reduced by this much is uncertain, as currently unidentified toxicants may not be susceptible to removal by charcoal filters. With more certainty, effective filters could reduce total cigarette cancer risk by at least 5%, or 80 deaths a year.

---

**References**


Caroline Shaw, Tony Blakely, Diana Sarfati, Jackie Fawcett, Sarah Hill

Abstract

Aim Tobacco use and resultant health effects have been described as an epidemic that progresses through the population. This paper aims to describe and explain trends in lung cancer mortality by ethnicity and socioeconomic position in New Zealand between 1981–1999.


Results Lung cancer mortality decreased in males and increased in females over the time period studied. In males, socioeconomic inequality persisted despite a decline in mortality in all socioeconomic groups. In females, a disproportionate increase in the mortality of lower socioeconomic groups compared to higher socioeconomic groups resulted in an increase in inequality. Divergent trends by ethnic group resulted in an increase in ethnic inequalities between 1981 and 1996 in both males and females.

Conclusions There are significant and growing ethnic and socioeconomic inequalities in lung cancer mortality in New Zealand. In the current absence of concerted public health action these inequalities will probably widen in future decades.

Background

New Zealand has relatively high lung cancer incidence and mortality, particularly among Maori.\(^1\) Lung cancer incidence and mortality trends largely reflect historical cigarette use and tobacco control efforts—although the role of occupational exposures is probably underestimated.\(^3\)

In 1994, Lopez, using historical data from a number of developed countries, proposed a model describing trends in tobacco-use and the resultant health effects. He showed that tobacco-use tends to progress through the population in a predictable way, like an ‘epidemic’, which differs by sex and, possibly, socioeconomic group. He noted that males tend to take up smoking initially and suffer the health consequences first, while females take up smoking later, and at a lower rate than males, resulting in fewer health consequences.\(^4\)

The Lopez model also describes the transition in population distribution of tobacco use from initially being equally distributed among socioeconomic groups (or possibly concentrated in the higher socioeconomic groups), to being concentrated among the lower socioeconomic groups as the higher socioeconomic groups abandon smoking.\(^4\)
Temporal trends in lung cancer incidence and mortality in New Zealand demonstrate this epidemic pattern by sex (see Figure 1). These patterns by sex are similar to trends seen in Australia, the UK, Ireland, and the USA.\textsuperscript{5-7}

**Figure 1 Age-standardised lung cancer incidence and mortality New Zealand (all ages; 1948–1999)**

According to the Lopez model, one would anticipate that lung cancer mortality would evolve from being an ‘egalitarian’ cause of death to one that is progressively confined to the lower socioeconomic groups over time (i.e. an increase in both relative and absolute inequalities).\textsuperscript{8}

Data on lung cancer mortality by socioeconomic position (SEP) is not available in New Zealand prior to the 1970s. Data available for males show higher rates of lung cancer mortality in lower socioeconomic groups in 1974–78, 1984–87, and 1996–97; and for females in 1996–97.\textsuperscript{9-11} Unfortunately, these studies do not allow us to evaluate whether inequalities are increasing or decreasing over time, as different measures of socioeconomic position were used in each study.

Ethnic disparities in lung cancer are not described in the Lopez model. Given the importance of ethnicity on health, and the diverse ethnic makeup of New Zealand’s population, it is important to explore the effects of the lung cancer epidemic on the different ethnic groups in New Zealand. Lung cancer incidence was higher for Maori compared with non-Maori in 1996–97,\textsuperscript{11} and incidence was higher in Pacific people compared to non-Maori/non-Pacific in the 1980s.\textsuperscript{12}

Systematic undercounting of Maori and Pacific people in routine cancer incidence and mortality statistics means that these data are likely to have underestimated the excess burden of lung cancer among Maori and Pacific people.\textsuperscript{13,14}
The New Zealand Census Mortality Study, through the anonymous record linkage of census and mortality records, allows an accurate description of ethnic and socioeconomic trends in lung cancer mortality in New Zealand. To inform tobacco policy in New Zealand, these trends (particularly the resultant future projections) need to be described. We hypothesise that, given the current epidemic staging; socioeconomic inequalities in lung cancer will have increased for females between 1981 and 1999. The tobacco epidemic peaked earlier in men than in women, hence trends for males are less easy to predict. We also hypothesise that there are likely to be divergent trends in lung cancer mortality between ethnic groups.

Methods

Background—The methodology of the NZCMS is discussed in detail in other publications. A brief summary of methods relevant to this paper is included. We used direct analyses on NZCMS data to determine socioeconomic trends. For ethnic trends, we applied adjustment ratios (derived from the NZCMS) to routine mortality and census data in order to compensate for undercounting of deaths in some ethnic groups in historical mortality data.


The percentage of records linked ranged from 71–78%, with positive predictive value of the linkage in excess of 96%. Linkage varied by age, rurality, ethnicity, and small-area deprivation so linkage weights were applied to overcome any potential misclassification bias of the mortality outcome caused by differential success of linkage. Deaths from lung cancer (ICD 162) were identified from the ICD code for underlying cause of death from the mortality data.

Socioeconomic trends—All individuals aged 25–77 at follow-up (either 3 years after census or at death within those 3 years) with valid income or education information were included in analyses. Information regarding education was obtained from individual census forms, however this data was missing for between 2–11% of census respondents.

An intercensal classification of educational qualifications was used to harmonise educational categories across censuses. Individuals were then divided into three groups: those with no qualifications, school qualifications, and post-school qualifications. Income was collated at a household level for individuals aged 25–77 and equivalised for household size using the Jensen equivalisation index.

Incomes were consumer price index adjusted for inflation to 1996 dollars, then divided into three income groups with cut points of low (<$26 010), medium ($26 010 to <$43 020), and high ($43 020). The household income variable was unable to be calculated for between 15–21% of individuals due to one or more adults in the household being absent on census night or declining to report an income.

Mortality rates (and 95% confidence intervals [CI]) were calculated with direct standardisation to the age and ethnic structure of the 1991 cohort. To overcome the problem of changing group size over time, the relative and slope index of inequality (RII and SII, respectively) were used to calculate population inequality in relative and absolute terms, respectively, in each cohort.

The RII is a regression based equivalent to a relative risk measure for the poorest compared to the richest (or people with lowest compared to highest educational qualification), but utilises mortality rates across all levels of income (and education). The SII is the absolute difference in mortality rates between the two extreme ends of the socioeconomic continuum.

Ethnic trends—Mortality data was provided by the New Zealand Health Information Services (NZHIS) for the years 1980–1999 by year of registration of death. Years were grouped into four periods: 1980–84, 1985–89, 1990–1995, and 1996–99. For each of the four periods, 1981, 1986, 1991, and 1996 census data by strata of sex, age, and ethnicity were used as denominator data in the calculation of mortality rates.

To adjust for the undercounting of Maori and Pacific deaths on mortality records, adjustment factors were used to estimate correct mortality counts. The method used to estimate the adjustment factors is described elsewhere. These corrected mortality counts and the census population counts were then
used to calculate direct age-standardised mortality rates (and 95% confidence intervals),\textsuperscript{20} using the WHO standard population as the standard population.\textsuperscript{22}

This paper uses the prioritised concept of ethnicity. In the ‘prioritised’ concept, ethnicity was assigned as Maori if one of the up to three possible self-identified ethnicity responses on the 1986, 1991, or 1996 censuses was Maori or, in 1981, those who recorded any degree of Maori ethnic origin.

For those not allocated as Maori, the prioritised ethnic group was assigned as Pacific if one of the self-identified ethnic groups was Pacific or, in 1981, any degree of Pacific ethnic origin was noted. The remaining records were assigned as non-Maori non-Pacific, of whom the majority were of NZ European ethnicity.

**Results**

**Socioeconomic trends**

**Males**—Between 1981 and 1999, a decline in lung cancer mortality in all education and income groups was observed among males. The rate of decline differed by socioeconomic group, with the greatest decline being seen in the high-income group (52%, $p$ for trend 0.04) and the least in the post-school education group (12%, $p$ for trend 0.23) (See Table 1 and Figure 2). Male mortality remained higher than female mortality despite these substantial declines.

At all points in time there was a socioeconomic gradient in male lung cancer mortality. That is, males in lower education and income groups had higher mortality than those in higher income and education groups. For example, the RII relative risk-type measure for education in 1981–84 was 2.30 (95% CI: 1.50–3.53), and the absolute difference counterpart (the SII) was 66 per 100 000 (95% CI: 38–94). There was some evidence of increasing relative inequality by income (i.e. RII increased from 1.80 in 1981–84 to 4.54 in 1996–99; $p$ for trend 0.15), but little evidence of changing relative inequality by education. There was no consistent trend towards increasing or decreasing absolute inequalities by either income or education (see Table 2).

**Females**—Lung cancer mortality trends diverged between the different socioeconomic groups from 1981 to 1999. The low-income group had a 70% increase in mortality from 27/100 000 to 46/100 000 ($p$ for trend 0.01); and similarly, mortality in the no qualifications group increased from 29/100 000 in 1981 to 48/100 000 in 1999, a 66% increase ($p$ for trend 0.02). The medium and high SEP groups showed no significant change in mortality rates, although there was some indication that mortality in high income groups may be falling over time (see Table 1 and Figure 2).
Table 1 Lung cancer mortality rates, per 100,000 population, by socioeconomic position (and 95% confidence intervals)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Income, 25–77 year olds</strong> (using analyses directly on linked census-mortality data)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>27 (23–32)</td>
<td>36 (32–40)</td>
<td>40 (36–45)</td>
<td>46 (42–50)</td>
<td>0.01</td>
</tr>
<tr>
<td>Medium</td>
<td>29 (23–35)</td>
<td>31 (26–36)</td>
<td>32 (26–37)</td>
<td>31 (26–36)</td>
<td>0.25</td>
</tr>
<tr>
<td>High</td>
<td>27 (20–34)</td>
<td>24 (18–30)</td>
<td>25 (19–31)</td>
<td>21 (16–26)</td>
<td>0.10</td>
</tr>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>102 (93–112)</td>
<td>102 (93–110)</td>
<td>87 (81–94)</td>
<td>79 (73–86)</td>
<td>0.03</td>
</tr>
<tr>
<td>Medium</td>
<td>82 (73–92)</td>
<td>75 (68–82)</td>
<td>65 (58–73)</td>
<td>59 (52–66)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>High</td>
<td>71 (61–81)</td>
<td>50 (43–58)</td>
<td>49 (41–58)</td>
<td>34 (28–40)</td>
<td>0.04</td>
</tr>
<tr>
<td><strong>Education, 25–77 year olds</strong> (using analyses directly on linked census-mortality data)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nil</td>
<td>29 (26–33)</td>
<td>34 (31–38)</td>
<td>44 (40–49)</td>
<td>48 (44–52)</td>
<td>0.02</td>
</tr>
<tr>
<td>School</td>
<td>23 (15–31)</td>
<td>37 (30–44)</td>
<td>30 (25–35)</td>
<td>33 (28–38)</td>
<td>0.52</td>
</tr>
<tr>
<td>Post-School</td>
<td>16 (10–23)</td>
<td>21 (15–26)</td>
<td>23 (18–29)</td>
<td>22 (18–26)</td>
<td>0.19</td>
</tr>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nil</td>
<td>98 (92–104)</td>
<td>95 (88–101)</td>
<td>84 (77–90)</td>
<td>78 (73–84)</td>
<td>0.01</td>
</tr>
<tr>
<td>School</td>
<td>77 (82–91)</td>
<td>67 (58–77)</td>
<td>70 (62–78)</td>
<td>54 (47–61)</td>
<td>0.14</td>
</tr>
<tr>
<td>Post-School</td>
<td>57 (46–68)</td>
<td>67 (59–74)</td>
<td>61 (54–67)</td>
<td>50 (45–55)</td>
<td>0.23</td>
</tr>
</tbody>
</table>
### Table 2 Relative and absolute inequality measures by socioeconomic position (25–77 year olds; 1981–1999)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Income, 25–77 year olds (using analyses directly on linked census-mortality data)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RII</td>
<td>1.14 (0.74–1.76)</td>
<td>1.83 (1.25–2.68)</td>
<td>2.09 (1.37–3.19)</td>
<td>4.09 (2.51–6.67)</td>
<td>0.03</td>
</tr>
<tr>
<td>SII</td>
<td>4 (-9–16)</td>
<td>18 (9–26)</td>
<td>23 (20–26)</td>
<td>40 (20–60)</td>
<td>0.06</td>
</tr>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RII</td>
<td>1.80 (1.39–2.33)</td>
<td>3.36 (2.44–4.61)</td>
<td>2.72 (1.97–3.74)</td>
<td>4.54 (2.99–6.89)</td>
<td>0.15</td>
</tr>
<tr>
<td>SII</td>
<td>47 (30–64)</td>
<td>80 (68–91)</td>
<td>60 (47–74)</td>
<td>69 (53–85)</td>
<td>0.82</td>
</tr>
<tr>
<td><strong>Education 25–77 year olds (using analyses directly on linked census-mortality data)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RII</td>
<td>2.53 (1.42–4.52)</td>
<td>2.03 (1.35–3.05)</td>
<td>3.18 (2.08–4.86)</td>
<td>4.15 (2.53–6.80)</td>
<td>0.18</td>
</tr>
<tr>
<td>SII</td>
<td>22 (10–34)</td>
<td>21 (1–40)</td>
<td>34 (24–44)</td>
<td>42 (34–49)</td>
<td>0.03</td>
</tr>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RII</td>
<td>2.30 (1.50–3.53)</td>
<td>1.97 (1.55–2.50)</td>
<td>1.99 (1.55–2.56)</td>
<td>2.72 (2.11–3.52)</td>
<td>0.42</td>
</tr>
<tr>
<td>SII</td>
<td>66 (38–94)</td>
<td>51 (20–82)</td>
<td>45 (28–62)</td>
<td>54 (21–87)</td>
<td>0.29</td>
</tr>
</tbody>
</table>

### Table 3 Lung cancer mortality rates, per 100,000 population, by ethnic group (and 95% confidence intervals)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ethnic group, 1–74 year olds (using NZCMS adjusters applied to routine data)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maori</td>
<td>49 (43–55)</td>
<td>54 (48–60)</td>
<td>60 (54–65)</td>
<td>69 (63–75)</td>
<td>0.01</td>
</tr>
<tr>
<td>Pacific</td>
<td>5 (1–9)</td>
<td>18 (11–25)</td>
<td>14 (10–19)</td>
<td>20 (15–26)</td>
<td>0.10</td>
</tr>
<tr>
<td>Non-Maori/non-Pacific</td>
<td>12 (11–13)</td>
<td>14 (13–15)</td>
<td>15 (14–16)</td>
<td>14 (13–15)</td>
<td>0.22</td>
</tr>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maori</td>
<td>84 (76–92)</td>
<td>76 (69–84)</td>
<td>87 (80–94)</td>
<td>86 (79–93)</td>
<td>0.54</td>
</tr>
<tr>
<td>Pacific</td>
<td>55 (38–72)</td>
<td>56 (43–69)</td>
<td>59 (49–69)</td>
<td>64 (53–75)</td>
<td>0.05</td>
</tr>
<tr>
<td>Non-Maori/non-Pacific</td>
<td>41 (39–42)</td>
<td>36 (35–38)</td>
<td>32 (31–33)</td>
<td>25 (24–26)</td>
<td>0.01</td>
</tr>
</tbody>
</table>
Figure 2 Age standardised lung cancer mortality rates among 25–77 year olds 1981–1999 (per 100 000) (using analyses directly on linked census-mortality data).

The divergent trends in mortality rates in the socioeconomic groups in females are reflected in the population measures of inequality (see Table 2). By income, there was little relative inequality in the 1981 cohort, RII 1.14 (95% CI: 0.74–1.76); but by 1999, the RII had increased to 4.09 (95% CI: 2.51–6.67) (p for trend=0.03). Absolute inequality increased by income from 4 per 100,000 (95% CI: -9–16) to 40 per 100 000 (95% CI: 20–60) (p for trend=0.06). Relative and absolute inequality also increased by education.

**Ethnic trends**

**Males**—Maori males had no decline in lung cancer mortality between 1980 and 1999, but there was a 16% increase in Pacific male mortality and, by contrast, a 24% decline in mortality for non-Maori/non-Pacific males (see Figure 3 and Table 3).

As a result of these different trends, ethnic inequalities in lung cancer increased over the time period studied with Maori men being 3.50 times (95% CI: 3.19–3.84) more likely to die of lung cancer in 1999 compared to 2.07 times (95% CI: 1.87–2.29) in 1981. Pacific men were 1.35 times (95% CI: 0.99–1.83) more likely to die in 1981; but by 1999, this had increased to 2.61 times (95% CI: 2.19–3.10).
Figure 3 Lung cancer mortality rates (per 100 000) by prioritised ethnic group among 1–74 year olds (using NZCMS adjusters applied to routine data)

**Discussion**

This study shows that socioeconomic and ethnic *inequalities* in lung cancer mortality remained static or increased in New Zealand from 1981 to 1999. While lung cancer mortality declined in males, there was no decrease in socioeconomic inequalities, and there was a substantial increase in ethnic inequalities. For females, there was not only an increase in overall mortality, but also an increase in both ethnic and socioeconomic inequalities.

Overall trends in lung cancer incidence and mortality suggest that New Zealand is in Stage 4 of the tobacco epidemic, which along with Ireland and UK is among the most advanced in the world. During Stage 4, smoking prevalence in both sexes declines, as does male lung cancer mortality, while female lung cancer rapidly rises to a peak and then starts to wane. Findings around inequalities are of interest both domestically (in order to plan local services) and internationally as a guide to what may be anticipated if action to avoid inequalities is not taken.
Socioeconomic Trends

This study shows that, despite the peak in the male lung cancer epidemic 20 years ago and decreases in mortality rates in all socioeconomic groups, inequality has not reduced. Similar findings have been noted in the USA, UK, and Australia where relative and absolute inequality do not appear to have decreased. In contrast, findings in Canada show that relative and absolute inequality have decreased among males since the epidemic peaked.

Interestingly, there was no evidence of the epidemic in Canada being staged by socioeconomic position as lung cancer mortality in all SEP groups (measured by area average income) peaked at the same time. This is in contrast to findings in the UK and US.

In contrast, findings in Canada show that relative and absolute inequality have decreased since the epidemic peaked.

In females, relative and absolute inequality has increased despite a slowing of the rate of increase in the lung cancer mortality since the 1990s (rates are forecast to increase slightly during the next decade). The increase in inequality is due to the disproportionate increase in mortality among lower SEP groups. Comparable patterns have been seen in the US, UK, and Australia, but not Canada where only absolute inequality between the SEP groups increased as the epidemic increased to a peak.

Changes in lung cancer mortality reflect the historical patterns in cigarette use by socioeconomic position and the staged nature of smoking through the population (although other causes of lung cancer, such as life course deprivation and occupational exposures, are important among lower SEP groups).

In New Zealand, population tobacco use has been monitored only since 1976, when socioeconomic patterning of tobacco use was already present in males and females. Subsequent monitoring of these differences shows that, despite an overall decline in smoking prevalence, socioeconomic inequalities in tobacco use have increased in relative terms.

Ethnic trends

The ethnic trends in mortality in New Zealand are extremely concerning. These findings suggest that lung cancer is an increasing source of health inequality between ethnic groups in New Zealand. There is evidence of differential survival from lung cancer by ethnic group, related to stage and, possibly, healthcare differences.

Nevertheless, because of the high fatality of lung cancer, changes in ethnic inequalities are largely due to changes in underlying incidence.

The increase in Pacific lung cancer mortality is probably due in part to the increasing prevalence of tobacco use in this group following migration to New Zealand in the 1970s (although the lag period suggests that tobacco use may have increased prior to migration). The mortality pattern in Pacific males is particularly interesting given that it is in the opposite direction to other ethnic groups. It suggests that migrant groups may not have the same overall trends in lung cancer as other ethnic groups in the population.

The different lung cancer mortality trends among Maori compared to non-Maori/non-Pacific may reflect Maori being in an earlier stage of the tobacco epidemic. Alternatively, the influence of other factors may make the Lopez model inadequate to describe the effects of tobacco use in Maori.
Exposure to tobacco commenced by the late 19th century among Maori, although we have no data on how it dispersed through the population. However there is some evidence to suggest that smoking prevalence in Maori women has historically been more akin to Maori male smoking prevalence, rather than non-Maori/non-Pacific female tobacco use.

Contemporary data showing the extremely high rates of tobacco use and lung cancer among Maori females, and the persistence of these patterns over time provide further evidence that the Lopez model is insufficient to describe the tobacco epidemic in this group.

The legacy of colonisation on indigenous people (which has included social marginalisation, cultural alienation, and the disproportionate representation of the colonised population in lower socioeconomic groups) needs to be considered as an explanation for the differing tobacco epidemic in Maori.

Current inequities of tobacco impact on Maori are likely to be exacerbated in the future since ethnic inequalities in tobacco use widened between 1981 and 1999, reflecting the failure of tobacco control efforts at that time to engage sufficiently with Maori. Postulated reasons for this failure include the monocultural nature of anti-smoking messages, financial barriers to smoking cessation, and the uneven impact of tobacco control legislation during this period (for example, the 1990 smokefree environments legislation resulted in differential exposure to second hand smoke in workplaces by ethnicity).

On the other hand, recent tobacco-control initiatives (such as the Quit Programme) have been increasingly designed for Maori and low socioeconomic groups, and the recent Smokefree Environment amendments prohibiting smoking in all workplaces and bars and cafes may help to reduce smoking inequalities in the near future.

Where to from here? What can we expect in lung cancer mortality inequalities in the future? Expected patterns of lung cancer mortality and inequalities in the next 20–30 years are described in Table 4. These are based on the epidemic patterns, the known time lag between population tobacco use and changes in lung cancer mortality (20–30 years between population changes in tobacco prevalence and lung cancer mortality changes, and a 30–40 year lag between maximal tobacco prevalence and the peak of the lung cancer epidemic) and available time trend data on tobacco prevalence by ethnicity and SEP in New Zealand.

This study suggests that lung cancer inequalities seen in New Zealand by ethnic group and SEP are likely to persist, or increase over time. However these are not inevitable, as mortality risk both at an individual and a population level can be averted or diminished by tobacco cessation, even at a late age. While New Zealand has historically had a relatively comprehensive tobacco control programme (compared to other countries), innovative interventions (such as the Aukati Kai Paipa programme) that are focused on groups with the highest need are now required to reduce inequalities in lung cancer mortality.
### Table 4 Predictions for mortality rates and inequalities in lung cancer mortality for the next 20–30 years in New Zealand

<table>
<thead>
<tr>
<th>Socioeconomic position</th>
<th>Mortality rates</th>
<th>Inequality</th>
<th>Ethnic group</th>
<th>Mortality rates</th>
<th>Inequality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td>Maori</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• All income and education groups will continue to decline.</td>
<td>• Absolute inequality will remain stable</td>
<td>• Maori should start to decline within the next 5–10 years.</td>
<td>• Absolute and relative inequality will increase until Maori mortality starts to decline</td>
<td>• Absolute and relative inequality will increase until Maori mortality starts to decline</td>
</tr>
<tr>
<td></td>
<td>• Absolute rates differences between the socioeconomic groups will be preserved.</td>
<td>• Relative inequality will increase</td>
<td>• Pacific men will peak within next 10–20 years</td>
<td>• Then absolute inequality will decrease, but relative inequality may continue to increase.</td>
<td>• Pacific men will peak within next 10–20 years</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>nMnP</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Lung cancer will decline in high-income group and rate of decline will increase.</td>
<td>• An increase in both absolute and relative inequalities by income will be seen for the next 10–15 years.</td>
<td>• Maori will peak within next 15 years and start to decline.</td>
<td>• Ethnic inequalities will increase in absolute and relative terms until Maori and Pacific women reach lung cancer epidemic peaks.</td>
<td>• Ethnic inequalities will increase in absolute and relative terms until Maori and Pacific women reach lung cancer epidemic peaks.</td>
</tr>
<tr>
<td></td>
<td>• Medium income group decline will become more apparent.</td>
<td>• By education we expect a sustained increase in relative and absolute inequality as there has been no decrease in smoking prevalence in the no education group between 1981 and 1996.</td>
<td>• BUT low income Maori group will remain high for a prolonged period.</td>
<td>• Following peaks absolute inequalities will decline but relative in inequalities may continue to increase or perhaps to decrease.</td>
<td>• Following peaks absolute inequalities will decline but relative in inequalities may continue to increase or perhaps to decrease.</td>
</tr>
<tr>
<td></td>
<td>• Low-income peak will occur and decline will start within next 20 years.</td>
<td>• The no education group will have a sustained peak of lung cancer mortality.</td>
<td>• Pacific will continue to increase to a lower peak than Maori – peak in lung cancer may occur in about 30 years.</td>
<td>• nMnP will continue to decline.</td>
<td>• nMnP will continue to decline.</td>
</tr>
<tr>
<td></td>
<td>• The no education group will have a sustained peak of lung cancer mortality.</td>
<td></td>
<td>• nMnP will continue to decline.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

nMnP = non-Maori/non-Pacific
**Summary Statistics New Zealand Security Statement**—The New Zealand Census Mortality Study (NZCMS) is a study of the relationship between socioeconomic factors and mortality in New Zealand, based on the integration of anonymised population census data from Statistics New Zealand and mortality data from the New Zealand Health Information Service. The project was approved by Statistics New Zealand as a Data Laboratory project under the Microdata Access Protocols in 1997. The datasets created by the integration process are covered by the Statistics Act and can be used for statistical purposes only. Only approved researchers who have signed Statistics New Zealand’s declaration of secrecy can access the integrated data in the Data Laboratory. (A full security statement is in a technical report at [http://www.wnmeds.ac.nz/nzcms-info.htm](http://www.wnmeds.ac.nz/nzcms-info.htm) For further information about confidentiality matters in regard to this study please contact Statistics New Zealand.

**Ethical Statement:** The programme of work of the New Zealand Census Mortality Study has approval from the Wellington Ethics Committee (Reference number 98/7).

**Author information:** Caroline Shaw, Research Fellow, Tony Blakely, Associate Professor; Diana Sarfati, Senior Research Fellow; Jackie Fawcett, Research Fellow, Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, Wellington South; Sarah Hill, Public Health Medicine Registrar, Wellington

**Acknowledgements:** The NZCMS is conducted in collaboration with Statistics New Zealand and within the confines of the Statistics Act 1975. The NZCMS was funded by the Health Research Council of New Zealand, and is now funded by the Ministry of Health. Dr Shaw acknowledges salary support from the Australasian Faculty of Public Health Medicine and the University of Otago. In addition, we gratefully acknowledge comments on earlier drafts by Darren Hunt, Ricci Harris, Martin Tobias, George Thomson, and Nick Wilson.

**Correspondence:** Associate Professor Tony Blakely, Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, PO Box 7343, Wellington South. Fax: (04) 389 5319; email: tblakely@wnmeds.ac.nz

**References:**


Smoky homes: a review of the exposure and effects of secondhand smoke in New Zealand homes

George Thomson, Nick Wilson, Philippa Howden-Chapman

**Abstract**

**Aims** To review the evidence of exposure to secondhand smoke (SHS) in New Zealand homes and its effects on health.

**Methods** A search for relevant literature was made in April–May 2004, using Medline and other databases, and via inquiries to official and other agencies. Data on the types of households with smoking members were obtained by an analysis of 1996 Census data.

**Results** National survey data indicate that at least 18% of all New Zealanders and 30% of Maori are exposed to SHS in the home. Surveys of high school students indicate home SHS exposure levels of 30% or more. The exposure appears to have decreased during 1996–2003 for Maori and the general population (p<0.001 for trend for both), with low-income households more likely to be exposed than others. There is an absence of exposure data for many specific population groups including pregnant women and infants.

New Zealand evidence from two large cohort studies indicates an increased risk of death of at least 15% for never smokers, aged 45–74, if they live in a household with smokers. Over 250 deaths per year are estimated to be attributable to SHS exposure in New Zealand homes; over double the mortality from SHS exposure at work.

**Conclusions** Improved information on SHS exposure in the New Zealand setting is needed. The levels of home SHS exposure and estimated mortality burden justify a substantial Government and health-agency investment to reduce this exposure, particularly for children, Maori, and those in low-income households.

This article reviews the evidence of the exposure to secondhand smoke (SHS) in New Zealand homes, and the evidence of the effects of that exposure. Work on this topic is part of research by the Housing and Health Research Programme/He Kainga Oranga of the University of Otago, on the health risks in the domestic indoor environment. SHS exposure in New Zealand workplaces has previously been reviewed.¹

Secondhand smoke is a major public health problem in New Zealand,² with no known safe level of exposure. SHS is associated with cardiovascular disease, cancers, respiratory and reproductive problems, and the damage of genetic material, potentially affecting the health of future generations. It has been described as the most dangerous common environmental air pollutant in developed countries.³,⁴

The health impact from SHS can be immediate, with reductions in arterial elasticity in healthy young adult non-smokers after 30 minutes exposure.⁵

Infants and children have characteristics that make them even more likely to be affected by SHS. They have smaller airways, higher respiratory rates, and immature immune systems. Infants inhale double the quantity of household dust compared to adults, and so inhale more dust containing SHS particulates (perhaps 40 more times more per body weight than adults). Infants also have greater hand/object/mouth...
contact, and so absorb proportionately more SHS through ingestion, as well as through inhalation.\(^6\)

SHS exposure for children increases the risk of: asthma exacerbations, lower respiratory illness, lung damage, middle ear disease, behavioural and learning problems, and Sudden Infant Death Syndrome (SIDS).\(^7\) In addition to the direct effect of SHS exposure on infants, the exposure of pregnant women to SHS adversely affects the health of their children.\(^8\)

**Methods**

A search was made for literature on SHS in the home setting during April–May 2004, through Medline, EBSCO, and Proquest electronic databases, using combinations of the search terms: Zealand, Maori, environmental, tobacco, secondhand, smok*, home*, infant*, child*, and parent*. The references within the material found enabled further publications to be accessed. In addition, reports were obtained by inquiries to official and other agencies. Data on the types of households with smoking members were obtained by an analysis of the 1996 Census results.\(^9\) Additional trend analyses were conducted on some of the survey data using the software package Epi Info 2000.

**Results**

**The prevalence of exposure to SHS**—A 2003 survey of those aged 15 years and over indicates that 18% of the general population are exposed to SHS in their own home, with 20% also reporting exposure to SHS in other people’s homes.\(^10\) Twenty-two percent of children were potentially exposed.\(^11\) These findings are compatible with the high proportion of respondents who reported smoking bans in their homes (75% of Maori, 80% overall).\(^10\) However, a survey of Year 10 students (aged 14–15 years) reported 30% exposed to SHS at home,\(^12\) and a 2002 survey of Year 10 and 12 students reported 44% of their homes as smoky.\(^13\)

There are little data on the amount of time per day that people are exposed to SHS in New Zealand homes. In 1996, survey respondents, who were exposed to SHS ‘away from work’ (not necessarily at home), reported an average of 3.4 hours exposure on weekdays (4.4 hours for Maori). At weekends, those exposed to SHS reported an average of 4.4 hours exposure ‘in their homes’ per day (5.1 hours for Maori).\(^14\)

The average reported time spent smoking in the 1999 *New Zealand Time Use Survey* was 1.6 hours per day for those who smoked, with women aged 12–29 years reporting smoking an average 1.9 hours/day.\(^15\) No data were found on the time spent smoking in homes.

In the 1996 Census, 38% of households with children (aged 17 and under), included smokers. Because over 7% of the adults living with children did not specify their smoking status, and non-reporting of smoking by those aged under 15 is probable, the proportion of households with smokers and children could have been significantly larger. Overall, households with children were more likely to contain a reported smoker than all households (38% compared to 33% respectively).\(^9\)

The existence of smokers living in a household does not necessarily lead to direct SHS exposure inside the home, nor does the absence of smokers living in a household prevent direct exposure in the home. Non-reported smokers under 15 may not smoke inside their homes. A 1996 survey reported that 30% of all smokers, and 37% of Maori smokers, restricted their smoking to outside their houses.\(^14\) The overall
proportion was even higher (40%) among smokers in houses with children under 5 years.

On the other hand, in a 2001 survey of Year 10 students, where neither parent smoked, 11% of students still reported that they were exposed to SHS in the home. Thus some children appear to be still exposed to SHS from visitors or non-parent household members.

**Time trends in SHS exposure**—Reported exposure to SHS in homes appears to have been decreasing over the last 15 years (Table 1). Between 1996 and 2003, the reduction for Maori and the total population was highly statistically significant (p<0.001 for trend for both). The ASH surveys of Year 10 students also indicate a steady reduction over time (p<0.00001 for trend) (Figure 1).

### Table 1: The proportion of New Zealanders aged over 14 years reporting regular SHS exposure at home (National surveys)

<table>
<thead>
<tr>
<th>Year</th>
<th>Number surveyed</th>
<th>Maori</th>
<th>Total population</th>
</tr>
</thead>
<tbody>
<tr>
<td>1989†</td>
<td>2300</td>
<td>54%</td>
<td>26%</td>
</tr>
<tr>
<td>1991†</td>
<td>2000</td>
<td>39%</td>
<td>25%</td>
</tr>
<tr>
<td>1996‡</td>
<td>2020</td>
<td>48%</td>
<td>28%</td>
</tr>
<tr>
<td>2003*</td>
<td>1502</td>
<td>30%</td>
<td>18%</td>
</tr>
<tr>
<td>2003*</td>
<td>500</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

† Only non-smokers asked; ‡ Smokers and non-smokers aged 15 plus; *Aged 15 plus, exposed to SHS in their home in one of the last seven days.

### Figure 1: SHS exposure at home for Year 10 students, 1992–2003*

* Year 10 students, asked if people smoked in their home. In 2003, the question changed to one asking students if people smoked in their home in one of the last 7 days.

**The unequal SHS exposure by ethnicity, income, socioeconomic status, and location**—Different population groups are exposed to very different levels of SHS. Maori are almost twice as likely to report SHS exposure compared to the whole population (Table 1). In 2001, 59% of 14–15 year old students in Year 10 attending schools classified by the Ministry of Education as being of socioeconomic decile 1
and 2 (i.e. the most deprived) had a parent who smoked. This compared with 27% of students in decile nine and ten schools. In 2003, the rate of SHS exposure reported by adults was 37% in households with an income under $10,000, compared to 7% for households with incomes between $70,000–$100,000.

Two local studies give further insights on the likelihood of differential SHS exposure in populations. A 1993–94 survey of rural or small-town Bay of Plenty children found smokers in homes of 57% of the children (41% of Pakeha [New Zealand European], 71% of Maori). A 1993 survey of Christchurch children compared a random group of 6–7 year olds from across Christchurch, with all 5–8 year olds in an industrial suburb of Christchurch (Hornby). Twenty-nine percent of the Christchurch-wide children were reported to have a smoker in their home, compared to 44% in Hornby. In comparison, national Census data from 1996 showed that 38% of households with children reported smokers in the household.

The health effects from home-related SHS exposure—There have been two recent New Zealand studies that estimate the effect of SHS exposure at home on mortality. The first conservatively estimated over 250 deaths in New Zealand per year resulting from current home SHS exposure; over double the mortality from workplace exposure. This estimate did not include the effects of SHS on current smokers.

The second study of two large cohorts utilised 1981 and 1996 Census data for never-smoking adults aged 45–74 and linked mortality data. It found an increased risk of death of 15% (per person/year), for those who lived in a household with smokers, compared to those that did not. This may be an underestimate, as the increased risk of death from home exposure may be even greater if the confounding effect of SHS exposure outside homes on New Zealanders could be allowed for. These two studies have been compared, and found to be broadly consistent in their findings.

Data from the Dunedin longitudinal study indicated that parental smoking significantly impaired the lung function of children as they developed between ages 9 to 15. A study of children aged 3–27 months, who were hospitalised with lower respiratory illness, showed that the more severely ill children had higher hair nicotine levels, indicating greater exposure to SHS.

In a study of Canterbury infants born during 1992, there was a statistically significant (52%) increased risk of hospitalisation within 10 months for infants of smoking mothers (allowing for ethnicity and educational level). The study estimated that 14% of all infant hospitalisations for children aged 6–10 months were attributable to maternal smoking (before and after birth). Furthermore, the number of respiratory illness hospitalisations (attributable to SHS) of New Zealand children aged 2 years has been estimated as over 500 per annum.

Based on 1991–1993 data, the risk of SIDS was increased by maternal smoking and a combination of maternal smoking and bed sharing. The latter combination increased the risk by five times, compared to children with non-smoking mothers. The total New Zealand deaths from SIDS that were attributable to SHS have been estimated at about 50 per year. In addition, two New Zealand studies have found a significantly increased risk of carriage of Neisseria meningitidis among those exposed to SHS.

Other likely consequences of SHS exposure in the home include an estimated 15,000 episodes of childhood asthma annually, more than 27,000 medical consultations for...
child respiratory problems, and 1500 operations in hospital to treat glue ear. In addition, surveys in 2001 and 2002 of Year 10 and 12 students indicate that smoking inside the home increases the normalcy of smoking for children, and thus the likelihood of children becoming smokers.

**Discussion**

**The exposure of children**— This review suggests that while SHS exposure in New Zealand homes appears to have been reduced over time, older children are consistently more likely to report being exposed than adults. This finding contrasts with research from Ontario, Australia, and California, which indicates that households with children are more likely to be reported as smokefree, compared to all households. However, these overseas surveys questioned adults. The New Zealand high school students may have been more candid than adults, or they may have been more or less accurate in their perceptions and memory of exposure.

Both student surveys reported a much higher level of SHS exposure at home (30% or more) than the level reported by the 2003 survey of those aged 15 and over (18%). As well as possible differences in accuracy and honesty, New Zealand households with teenage children may be more likely to have smoking inside, compared to all New Zealand households. While households without children are less likely to contain smokers, this does not appear to explain the differences between the exposures reported by the surveys. The validation of self-reporting surveys by objective monitoring is an ongoing need, if only because of possible changes in the accuracy of self-reports over time.

Biomarkers and other objective monitors for SHS exposure include levels of cotinine, and hair nicotine. The cotinine levels measured in non-smokers is a substance produced by the metabolism of nicotine, and thus serves as a proxy for all the many elements of SHS. Cotinine levels can be found in blood, saliva, or urine. Other monitors include fixed gauges such as monitor badges that can be placed inside houses or on clothes, and air sampling (nicotine concentration and particulate levels as a proxy for SHS). The contamination by SHS of interior surfaces could also be measured, and the nature of that contamination analysed.

Using the same survey question to establish the SHS exposure status of homes, the 2002 survey of Year 10 and 12 students reported 44% of homes as smoky, compared to the 30% reported by the 2003 survey of Year 10 students. While both were national surveys, there is some difference in their samples. The survey conducted in 2002 generally used two classes of Year 10 students and one class of Year 12 students, randomly selected in each participating school, resulting in a total of 914 Year 12 students and 2,520 Year 10 students, with a mean age 15.0 years (Personal communication, H Darling, 2004). The 2003 survey of Year 10 students was limited to those aged 14 or 15 years, with a sample of around 30,000. Such sample differences may therefore explain some part of the discrepancy in these results, along with a downward trend in SHS exposure. Nevertheless, the reason for such a large difference is still not readily explainable.

**Differing trends for smoking prevalence, tobacco consumption and home SHS exposure**—The reported fall (by over one-third) in home SHS exposure during 1996–
2003, for both Maori and the whole population, contrasts with the static rate of adult smoking prevalence during the period (from 26% to 25%). Apart from a movement to smoking outside, other possible reasons for this contrast are that a constant proportion of smokers were tending to concentrate in a smaller proportion of households, and/or that the size of households with smokers was decreasing relative to other households. This pattern, of SHS exposure declining faster than smoking prevalence, is repeated in data from California, the USA, and Australia.

Tobacco consumption in New Zealand fell from 1511 cigarette equivalents per smoker in 1996, to 1187 per smoker in 2002. This 21% reduction suggests that the duration and intensity of home SHS exposure will have declined on average. However, as the dose response effect of SHS does not appear to be linear, a decrease in duration and intensity may not result in an equivalent reduction in harm to health among those exposed to SHS.

**The exposure of particular populations and possible trends**—The New Zealand pattern of greater SHS exposure at home for those in low-income households is consistent with American evidence. It is also consistent with the New Zealand evidence for total SHS exposure at work and home. The higher SHS exposure means that the existing financial disadvantage of low-income households is compounded by the likelihood of increased illness and premature death. Therefore, improved control of the SHS problem has potential to reduce health inequalities in the New Zealand setting.

The proportion of the New Zealand population that is Maori, Pacific, and Asian (particularly those under 18 years) is growing. By 2016, just over half of all children are projected to be in these three ethnic groups. Thus, if current differentials in SHS exposure levels persist, the population effect of higher SHS exposure on Maori children may become relatively more important.

**Exposure from re-emission from deposited SHS particulates**—The reported exposure to SHS does not take into account the re-emission of material from the tobacco smoke deposited on household surfaces, clothes, and skin. There may also be SHS exposure due to direct hand or mouth contact with household surfaces, clothes, and skin. A 1996 study of child inpatients (aged 3 months to 10 years) in Wellington found that reported smoking outside by others in the household did not reduce hair nicotine in the children. This may have been due to misreporting, to the child’s exposure outside their own house, to smoke brought inside on clothes or other objects, and to previous smoking in the house (due to the long life of smoke residues).

**Improving surveillance and research**—No national data have been published on the SHS exposure of Pacific Peoples or Asian groups, or of pregnant women and infants. Therefore, information from the routine national Health Surveys of the Ministry of Health is needed to fill this gap. Furthermore, regional data from national surveys of SHS exposure need to be analysed, to help focus District Health Board policymakers on areas and groups at particular risk.

As shown above, some local rates of SHS exposure may be double the national rates. National and local data are also needed on the duration of exposure, and some of the direct and indirect effects of SHS exposure at home—including primary care visits, school and work absenteeism, and unintentional injury rates. Evidence on some of
these outcomes will be available in the future from the *Housing, Insulation and Health Study* of the Housing and Health Research Programme.53

Research on the financial costs of SHS in homes is desirable to determine the resulting health care spending, lost pay, lost and lower production, and the costs of work and other injuries. Other related spending that could be isolated are those of higher cleaning and maintenance costs, lower home resale prices, and higher insurance costs.54 Apart from the direct health care costs, other indirect and intangible costs from childrens’ sickness (resulting from SHS) that could potentially be measured include: time off work for parents to care for sick children, healthcare-related transport, and the downstream financial and other costs of the psychological stress on parents.55

At present, there is a lack of a standardised classification system of SHS exposure levels that can be recognised by New Zealand policymakers, health professionals, and others as requiring action to protect the general population or particular vulnerable groups (e.g. those with established respiratory conditions). This lack is echoed in other jurisdictions.56 In contrast to the official national target developed for smokefree workplaces in New Zealand,57 there is no such target for increasing the prevalence of smokefree homes.

**Policy implications for SHS control**—The recent decrease in home SHS exposure is a public health success, but the evidence still indicates a significant danger to health within homes for at least a fifth of the New Zealand population. The consequent mortality is likely to be at least double that from workplace SHS exposure before 2004, and is likely to become relatively greater as workplace SHS exposure is sharply reduced.

An investment and policy focus by Government and other agencies is needed to reduce SHS exposure for all New Zealanders, with the priority on improving the protection of those groups most at risk—children, Maori, and those in low-income households. Child exposure is a particular concern, as children may have no one to negotiate smokefree homes on their behalf.

The recent increase in smokefree workplaces, due to the *Smoke-free Environments Amendment Act 2003*, is likely to support the trend towards smokefree homes. This is because the existence of smokefree workplaces changes social norms,58 with some resulting association between working in smokefree places and living in smokefree homes.59,60

Apart from improving health and reducing a range of costs, smokefree homes have a protective effect for the risk of child smoking uptake, and also help smokers reduce and quit smoking.33 There is evidence that comprehensive tobacco control programs are associated with increased smokefree homes.61,62 Possible options for central Government include strengthening tobacco control programmes, especially for groups most at need, and strengthening mass media campaigns that specifically promote smokefree homes. Indeed, a Government target for the verifiable reduction of home SHS exposure is essential.

**Author information:** George W Thomson, Research Fellow; Nick A Wilson, Senior Lecturer; Philippa Howden-Chapman, Associate Professor, Department of Public
Health, Wellington School of Medicine and Health Sciences, University of Otago, Wellington

Acknowledgements: Comments from Dr Murray Laugesen and an anonymous reviewer on drafts were much appreciated. In addition, we thank all the agencies and individuals who helped us access data, particularly the Health Sponsorship Council and Helen Darling. (The Health Research Council of New Zealand funds the Housing and Health Research Programme.)

Correspondence: Dr George Thomson, Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, PO Box 7343, Wellington South. Fax: (04) 389 5319; email: gthomson@wnmeds.ac.nz

References:


42. Centers for Disease Control. Percentage of adults who were current, former, or never smokers. Atlanta: Centers for Disease Control; 2003.


Attitudes to, and knowledge of, secondhand smoke in New Zealand homes and cars

George Thomson, Nick Wilson, Philippa Howden-Chapman

Abstract

Aims To review the evidence on knowledge and attitudes among the New Zealand public concerning secondhand smoke (SHS) and smoking in homes and cars.

Methods A literature search for published and unpublished material relevant to New Zealand.

Results While New Zealanders’ knowledge about SHS effects has improved since 1989, with 90% or more of the adult population aware of a risk to health, this knowledge may be shallow. Wellington area surveys indicate that significant proportions of the population are not aware of both the major consequences of SHS, that is, strokes and heart disease.

Survey data indicates increasing public support for smokefree homes during 1999–2003, particularly among Maori who showed a 68% increase in support during that period. In 2003, over 80% of New Zealand smokers indicated that people have a right to smokefree homes. However, these attitudes do not necessarily result in smokefree homes. Of those 14–15 year olds with at least one parent who smoked, less than 45% reported having a smokefree home.

Conclusions Improved tobacco control and increased investment in mass media campaigns on SHS issues are needed to strengthen healthy norms around smokefree homes and cars.

This article reviews the evidence on knowledge and attitudes among the New Zealand public concerning secondhand smoke (SHS) and smoking in homes and cars. Work on this topic is part of work by the Housing and Health Research Programme/He Kainga Oranga of the University of Otago, to identify health risks in the home setting and the methods of reducing them.

The adoption and implementation of public and private policies to control SHS depends partly on the related knowledge and attitudes of the population. As New Zealand legislation now controls smoking in nearly all interior work and public places, homes and cars are now the areas with the most potential for further protecting public health from the SHS hazard.

The context for knowledge about the effects of exposure to SHS includes the information found in the media (including paid advertising), formal education in schools and elsewhere, information to patients and their families from health professionals, and the experience of individuals in observing the effects of SHS. In addition, the tobacco industry and other commercial groups in New Zealand still deny the substantial health effects of SHS.¹
Worldwide, the tobacco industry has been concerned to maintain the idea that the scientific evidence about SHS harm is debated and controversial. For instance, a study found that reviews of the health effects of SHS were over 80 times more likely to find no health effects if the authors were tobacco industry funded. In New Zealand, a representative of British American Tobacco (BAT) gave evidence to the Health Select Committee of Parliament in November 2002. He was reported as saying that:

‘...in our view, it has not been established that ETS [environmental tobacco smoke] exposure genuinely increases the risk of nonsmokers developing lung cancer or heart disease’

Currently, the BAT New Zealand website states:

‘...we think that many of the claims against environmental tobacco smoke have been overstated. Specifically, we don’t believe that it has been shown to cause chronic disease, such as lung cancer, cardiovascular disease or chronic obstructive pulmonary disease, in adult non-smokers’

‘.... the studies on lung cancer to date suggest that if there is a risk, it is too small to measure with any certainty. …. There is evidence for example that exposure to it is related to acute illnesses, like respiratory and ear infections, in children who live in smoking households’

Much of the context for public knowledge about SHS is determined by the coverage of the subject by mass media. In the USA, there has been a persistent gap between the scientific consensus about SHS harm and the media coverage of that consensus, with media continuing to report that the science was ‘controversial’.

Attitudes to SHS and restrictions on smoking arise within a context of beliefs about rights and obligations. For instance, depending on their views and the way in which the topic is framed, people will support the rights of children to health, or will advocate a smoker’s right to do what they want in their own home (including smoking in a house with children). The New Zealand Human Rights Commission does not consider that smoking is a right under the Human Rights Act.

The normalcy of smoking restrictions within a society affects attitudes about SHS restrictions in private places. Workplace bans appear to create spillover effects, with Australian evidence indicating that those working in places with smoking restrictions are more likely to discourage visitors from smoking in their homes. Other predictors of positive attitudes to SHS restrictions in the home include the presence of children, some or all the adults being non-smokers, and believing that SHS can harm people.

Some of the elements that may affect smokers or non-smokers attitudes about SHS include perceptions about the amount of SHS around themselves or their children. If the amount is perceived as small, and the risk of that SHS to health is perceived as trivial, action to change the situation can be seen as unnecessary. This tendency can be exacerbated by the extent to which people have unfounded optimistic views about risks to themselves. Optimism tends to be greatest for risks thought to be personally controllable, and where the evidence of harm is delayed.

Methods

A search was made in April–May 2004, through Medline and other electronic search engines, using combinations of the following search words: Zealand, Maori, environmental, secondhand, tobacco, smok*, home*, infant*, child*, and parent*. The references within the material found enabled further publications to be identified. In addition, official and other reports were obtained by inquiries to official and other agencies. Additional trend analyses were conducted on some of the data obtained using the software package Epi Info 2000.
Results

Public knowledge of SHS hazards—In 1989, there were considerable differences in the reported knowledge of harm to health from SHS, by ethnicity (Table 1). However, by 1999 there was little difference between the responses of Maori respondents and respondents from the total population, with at least 90% agreeing in 2003 that there was harm from SHS (Table 1).

Table 1: Proportion of the adult population agreeing to statements that SHS causes harm (various national surveys)

<table>
<thead>
<tr>
<th>Year</th>
<th>Adult population</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Over 55 years</td>
</tr>
<tr>
<td>1989*</td>
<td>74%</td>
</tr>
<tr>
<td>1991*</td>
<td>86%</td>
</tr>
<tr>
<td>1999‡</td>
<td>92%</td>
</tr>
<tr>
<td>2001‡</td>
<td>94%</td>
</tr>
<tr>
<td>2001#</td>
<td>87%</td>
</tr>
<tr>
<td>2003†</td>
<td>91%</td>
</tr>
</tbody>
</table>

*Agree to the statement ‘The health of non-smokers can be damaged by other people’s tobacco smoke’. 11,12
#Agree to the statement ‘Smoke from other people’s cigarettes is harmful to you’ – by saying ‘probably’ or ‘definitely’. 13,‡Agree to the statement ‘People’s health can be damaged by other people’s tobacco smoke’. 14
†Agree to the statement ‘People’s health can be damaged by other people’s tobacco smoke’ (‘slightly’ or ‘strongly’). 13

One of the first extensive surveys in New Zealand, on knowledge about SHS effects, was done in 1988 for the Tobacco Institute of New Zealand (TINZ) by the Heylen Research Centre. The survey was of 1000 people aged 15 and over. When asked if the statement ‘Science has not established that other peoples’ cigarette smoke is a health hazard to non-smokers’ was true or false, 26% said true, and 69% (52% of smokers) said false. 10 The survey report gives data from earlier New Zealand surveys, with reactions to the statement ‘Cigarette smoking is not harmful to non-smokers’. Agreement to this statement in 1982 and 1985 was 16% and 12% respectively. 10

A 1989 survey for the Department of Health asked if the statement: ‘The health of non-smokers can be damaged by other people’s tobacco smoke’ was true. A large majority (84%) agreed, 6% disagreed, and 10% said neither, or didn’t know. 11

Populations whose agreement was lower than average included those over 55 years of age (74%), Maori (65%), Pacific (69%), and smokers (60%). By 1991, the same statement was agreed to by significantly more of those groups who previously had lower than average agreement. 12

The depth of the knowledge about SHS effects—The depth of knowledge about harm from SHS has been investigated in two Wellington area surveys: in 1997 and 1999–2000. These indicated that only half or less of the groups surveyed were aware that SHS contributed to all of five specific health conditions (Table 2). 16,17 In one survey, of Wellington bar and restaurant staff and owners, less than a third of interviewees knew of the risk for strokes from SHS. 17 In an Auckland survey, 1376 Pacific mothers of 6-week-old infants were ‘given a short description of sudden infant
death syndrome (SIDS)’ and asked if they had heard of ‘the ways parents could help prevent SIDS or cot death’. Only 32% reported maternal smoking as a risk factor.

Table 2: Knowledge about particular SHS effects on health identified in local surveys

<table>
<thead>
<tr>
<th>Question</th>
<th>Survey of Wellington bar and eating place workers 1999–200017</th>
<th>Public survey in Wellington, 199716</th>
</tr>
</thead>
<tbody>
<tr>
<td>Does breathing other people’s smoke increase the risk of:</td>
<td>Answering ‘Yes’</td>
<td>Answering ‘Yes’</td>
</tr>
<tr>
<td>Asthma</td>
<td>80%</td>
<td>60%</td>
</tr>
<tr>
<td>Cancer</td>
<td>69%</td>
<td>76%</td>
</tr>
<tr>
<td>Heart disease</td>
<td>61%</td>
<td>57%</td>
</tr>
<tr>
<td>Breathing/respiratory problems*</td>
<td>92%</td>
<td>58%</td>
</tr>
<tr>
<td>Cot death</td>
<td>53%</td>
<td>69%</td>
</tr>
</tbody>
</table>

* The 1997 survey used the word ‘respiratory’ instead of ‘breathing’.

Public attitudes to SHS in homes and cars—Apart from the 1988 survey for TINZ, most of the New Zealand data on attitudes to SHS in homes and cars has been gathered since 1999. The questions used have varied from asking directly if ‘people should be able to smoke’ in homes and cars, to questions which frame smokefree homes as a right. In addition, there have been questions about smoking when children are around or when there are car passengers. Data about reported smokefree policies for homes can also be interpreted as evidence about attitudes.

The 1988 survey for TINZ gave seven options for preferred smoking policies in homes and cars. Even so, 41% of non-smokers and 4% of smokers wanted no smoking at all in their own homes. For other peoples’ homes, 35% of non-smokers and 18% of smokers wanted no smoking at all. For private cars, 58% of non-smokers and 18% of smokers wanted no smoking at all.10

A 1997 Wellington area survey asked for reactions to the statement ‘it should be made illegal for people to smoke in cars when there are passengers.’ Over 50% of interviewees agreed, including 43% of smokers. Over 85% of interviewees (78% of smokers) agreed that homes should be smokefree ‘when there are children around,’ and 94% agreed that cars with children in them should be smokefree (86% of smokers).16 Surveys between 1999 and 2003 indicate that support for smoking at home has declined significantly (Table 3).15

If questions were framed in terms of children, or rights for smokefree homes, very different answers were given. During 1999–2003, over 90% of both Maori and the general population disagreed with the statement that it was ‘OK to smoke around children.’15 Over 80% of both Maori and the general population indicated that people have a right to live in an environment free of smoke. Most smokers (81% overall, 76% of Maori smokers) agreed to this principle.13,15
Table 3: Attitudes to smoking in homes and cars

<table>
<thead>
<tr>
<th>Year</th>
<th>Proportion answering “not at all” to the statement:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>“People should be able to smoke at home”</td>
</tr>
<tr>
<td></td>
<td>Maori</td>
</tr>
<tr>
<td>1999</td>
<td>22%</td>
</tr>
<tr>
<td>2001</td>
<td>25%</td>
</tr>
<tr>
<td>2003</td>
<td>37%</td>
</tr>
</tbody>
</table>

P value for trend: p<0.00001  p<0.00001  p=0.52  p<0.00001

Finally, the depth of information about the effects of SHS may affect attitudes to smoking. In a survey during 1999–2000 of Wellington bar and restaurant staff and owners, interviewees were asked about the risk of seven health conditions from SHS. Those aware of all seven risks were twice as likely compared to all other interviewees to want no smoking in bars (14% compared to 7%, p=0.009), and over twice as likely to want stronger restrictions on smoking in bars (21% compared to 9%, p=0.012).

Attitudes about SHS relative to behaviour—There are large gaps between people’s general views on SHS and their self-reported smoking behaviour. In the 1997 Wellington area survey, only 50% of smokers ‘reported not smoking in the company of children’, despite 78% agreeing that homes should be smokefree ‘when there are children around’. There are also gaps between behaviour and the general acceptance of rights for smokefree homes. Of 14–15 year old students with at least one parent who smoked, less than 45% reported having a smokefree home, despite over 80% acceptance by smokers of the right for a smokefree home.

Discussion

Limitations of the data—The data on the depth of knowledge about SHS effects are from regional surveys, and may not be generalisable to the rest of the country. Because of this, and the changes in knowledge and attitudes over time, there is an ongoing need for national data on these issues. There is also a need for surveys that seek unprompted responses to questions such as ‘what are the health effects of other peoples’ smoke’. These questions may be more effective in determining the depth of knowledge about SHS, compared to questions that prompt about particular health effects.

There is also a lack of New Zealand data on the perceptions of SHS harm compared to other causes of harm, the perceived immediacy or distance of SHS harm, the frequency of any prompts about SHS harm, and their effectiveness. Furthermore, because of the wide variance between the answers for different questions about attitudes to SHS in private places, there appears to be a need for much more detailed data on the way New Zealanders balance support for ‘rights’ to smokefree homes and their preferences for permitted smoking.

Key findings—While New Zealand adults’ awareness that SHS is harmful appears to have been high since 1988 or before, with generally less than 10% of the population...
unaware of the harm since 1999, this impression may obscure considerable variance in the depth of knowledge about the harm.

The New Zealand data illustrate that when survey questions about attitudes to SHS restrictions include the context of rights to live in a smokefree setting, interviewees are much more likely to favour the smokefree approach. This result echoes New Zealand survey data about attitudes to smokefree workplaces, where survey questions that included the context of rights to be smokefree also produced higher levels of support. However, declared attitudes on the need for smokefree homes are not necessarily reflected in actions by smokers to reduce SHS exposure to others.

The depth of knowledge—The evidence of a varied depth of knowledge of SHS effects (depending on the groups asked and the questions used) is repeated elsewhere. In an Australian survey in 2000, there were large differences between the knowledge of SHS health effects—ranging from over 80% agreeing that there was an increased risk of lung cancer, child asthma, and child respiratory problems, to only 31% agreeing that there was an increased risk of child ear problems. The quality of knowledge about SHS effects is important, as behaviour about SHS depends on the depth of information available, and the emotional value of the information to the recipient, amongst other things.

The effect of knowledge and attitudes about SHS on actions—Elements which may effect smokers or non-smokers actions include their knowledge about SHS, their interest in SHS effects, their ability to make plans to act, and their ability to carry out any plans. For instance, they may know of some risks from SHS to children, but the risk is either outweighed by other immediate needs, or there is an indifference to that level of risk. The possible and future health effects of SHS often appear distant compared to the immediate need to end nicotine craving, or welcome a (smoking) guest. Smokers can use ‘self-exempting’ beliefs (varied forms of denial) to help reduce the contradictions between their knowledge of SHS risks, and their behaviour.

Smokers or non-smokers may be interested in SHS risks, but may be unable to make plans to act. They may have limited experience of others near them succeeding, have little experience of being able to persuade others to change behaviour, or be unable to break down the tasks into practical stages. The ‘costs’ of acting may be too great at particular times or overall, compared to the perceived benefits (the costs may be immediate and concrete, and benefits diffuse and in the future).

Factors in deciding actions include the severity and likelihood of perceived effects of SHS; the perceived benefits and costs of acting; the proximity of the threat or benefit; and the frequency of effective prompts about threats, benefits, and their proximity. Many of these factors are heavily dependent on context, such as the opinion of friends, experience, cues (smokefree notices, bans elsewhere), and emotions (e.g. due to experience of illness of children).

Policy implications—Changing the behaviour of others in one’s household, especially smokers, is more difficult when there are higher numbers of smokers per household. Once there is some form of local majority in favour of smokefree homes (e.g. the majority of a personal circle), changing household behaviour is likely to become easier however. Thus a ‘critical mass’ is needed for the adoption of the idea. In turn, these local norms are generally influenced by societal norms.
The need for local and societal-wide change suggests the importance of further Government investment in mass media campaigns on SHS issues (to supplement the low intensity campaigns that have occurred in recent years in New Zealand). Evidence elsewhere indicates that exposure to such campaigns, within strong and comprehensive national tobacco control programs, can increase the likelihood of smokefree homes.\textsuperscript{4,26,27} This is supported by the strength of the evidence showing that, compared to many tobacco control interventions,\textsuperscript{28} mass media campaigns (as part of comprehensive tobacco control programs) can decrease smoking prevalence cost-effectively.

However, the very low levels of resources invested in New Zealand tobacco control health promotion campaigns (which are largely mass media) may be below an adequate intensity threshold to be sufficiently effective. In the 2003–2004 year, for example, less than $7 million was so invested by Government through the two main national agencies: the Health Sponsorship Council and the Quit Group (personal communication, J Muschamp, 2004).\textsuperscript{29} This compares with, for instance, over $31 million per year spend on road safety information and promotion.\textsuperscript{30}

Furthermore, annual tobacco-related deaths in New Zealand are over 10 times the level of road deaths.\textsuperscript{30,31} Thus, national-level health promotion spending to prevent deaths is under $1400/death for tobacco control, compared to $69,900/death for road safety. Indeed, in terms of the prevention of premature deaths, national tobacco control health promotion campaigns appears to be funded at a fiftieth or less of the rate for road safety.

Further actions needed to increase knowledge and change attitudes include the enforcement of existing New Zealand statute law by Government, so as to prevent the dissemination of misleading information on smoking and SHS by the tobacco industry.

**Conclusions**

While New Zealanders’ knowledge about SHS effects has improved since 1989, this knowledge appears to remain shallow in quality. Survey data indicates that public support for smokefree homes has increased, but varies markedly with the type of question asked. Further reduction in SHS exposure in homes requires further changes in societal norms on such exposure. A greater investment to create supportive environments for smokefree homes and cars would help these changes, and facilitate a reduction in the substantial morbidity and mortality burden from SHS.

**Author information:** George W Thomson, Research Fellow; Nick A Wilson, Senior Lecturer; Philippa Howden-Chapman, Associate Professor, Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, Wellington

**Acknowledgements:** The Health Research Council of New Zealand funds the Housing and Health Research Programme/He Kainga Oranga of the University of Otago, and The Health Sponsorship Council was very helpful in supplying reports and information to us. We also very much appreciate the advice given by an anonymous reviewer.
Correspondence: Dr George Thomson. Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, PO Box 7343, Wellington South. Fax: (04) 389 5319; email: gthomson@wnmeds.ac.nz

References:


Access to tobacco products by New Zealand youth

Helen Darling, Anthony Reeder, Rob McGee, Sheila Williams

Abstract

**Aims** To describe the sources of cigarettes for under-age youth who had smoked in the previous month, the frequency of their purchases and the revenue generated.

**Methods** A self-report questionnaire was administered to 3434 secondary school students from 82 schools, randomly selected using multi-stage cluster sampling.

**Results** Over one-third of the students who smoked had purchased tobacco products from commercial sources in the month before the survey; most frequently from dairies and service stations. For more than one-third of smokers (35.7%), being younger than 18 years was not a barrier to purchasing tobacco products. During 2002, the retail value of tobacco sales to those 14–16 years, alone, was estimated to be in excess of $18 million, with around $12.5 million of this going to the Government as taxes.

**Conclusions** Policies that restrict youth access to tobacco products can only be effective if they are rigorously enforced. Many young New Zealanders have no difficulty in purchasing tobacco products, thereby generating significant revenue. Total sales to all smokers under 18 years would be likely to exceed of $24 million, with around $17 million in taxes. Current legislation and enforcement is not a sufficient deterrent to ensure retailer compliance with age restrictions. It would be appropriate to use at least some of the revenue from under-age sales to fund health promotion programmes to reduce tobacco smoking and other health-compromising behaviours among youth. Nationally collated data on monitoring visits, prosecutions, and fines for under-age sales are currently not readily available, thereby limiting opportunities for evaluation.

In recent months, the World Health Organization has confirmed that the health burden attributable to addictive substances is greatest for tobacco. Dependence most often begins during adolescence, but the most serious consequences usually occur in later life. It is, therefore, logical to prevent dependence in the first place. Direct prevention programmes for youth have traditionally taken place in school settings, through mass media interventions, and through restricted access to tobacco products. Encouraging parents to stop smoking may (indirectly) reduce youth smoking through decreasing exposure to secondhand smoke and access to tobacco products.

A study, based on data from the US national Youth Risk Behaviour Survey, reported that nearly one-quarter of smokers purchased tobacco products from a store, despite legislation to prevent this. Difficulties in achieving high levels of compliance appear common, and this may be due, in part, to obstacles to enforcement, such as retailer opposition, and the cost of compliance monitoring.

In 1996, New Zealand (NZ) legislation was amended to make it illegal for persons under the age of 18 years to purchase tobacco products after July 1997 (previously the lower age limit was 16 years). Restricting access to tobacco products is a relatively
controversial preventive measure. It has been argued that restricting the sale of tobacco to those under 18 years of age reinforces the perception of smoking as an ‘adult’ behaviour, thereby making it more desirable among youth who aspire to adult status. Similarly, in the absence of commercial sources of tobacco, young people may seek and develop other sources, thereby negating access restrictions as an intervention.

In NZ, Laugesen and Scragg (1999) reported changes in youth purchasing behaviour between 1992 and 1997. There was a substantial decrease in the proportion of under-age youth purchasing cigarettes from commercial outlets. In spite of the decrease, it was estimated that the retail value for these cigarette purchases was $8.7 million per year, and the risk of retailer prosecution was considered minimal at that time. Purchasing from retail outlets continued to be the main source of cigarettes for NZ youth. A recent study of purchasing behaviour found that commercial outlets were the usual source of cigarettes for 61.8% of those reporting recent smoking. The estimated retail value, including taxes, of cigarette purchases by underage youth in 2000 was in excess of $25.8 million.

The aims of this present study are to address the following questions:

- What are the primary sources of tobacco products for under-age youth in NZ?
- Are under-age youth asked for identification to prove their age, and, are they refused sales?
- What revenue was generated from under-age sales of tobacco in NZ during 2002?

**Methods**

**Sample**—Data for this study came from the Health Sponsorship Council’s 2002 Youth Lifestyle Study (YLS), a biennial survey of tobacco-related attitudes and behaviours. The procedures for this study have been described in detail elsewhere. In summary, using a multi-stage cluster sampling approach, secondary schools and school classes were randomly selected from within six geographical regions. Probability weights were assigned at the individual student level.

**Procedures**—The YLS was piloted by HD and the study proper conducted in two waves during May and November 2002. As a consequence of high student absence rates in some schools in May (due, in part, to industrial action by students and teachers, snow, and flooding), the survey was completed in November using the same sampling method.

The survey was administered by trained interviewers. Participants were advised that their answers were confidential and anonymous, but that their survey form would be checked for completeness by the interviewer. On average, the questionnaire took 40 minutes to complete.

**Measures**—The YLS questionnaire contained six sections: demographic data; interests; use of the media; tobacco smoking beliefs, attitudes, and behaviours; exposure to health promotion messages; and, a measure of self-concept.

Smoking status was determined by response to the question—*How often do you smoke now?* Participants who reported smoking *at least once a day... week... or month* were categorised as current smokers for the purpose of these analyses. Data were collected on purchasing tobacco products. Participants were asked—*In the last 30 days (1 month) how did you usually get your own cigarettes?* The response categories provided are presented in Table 1. To identify specific commercial sources of cigarettes, participants were asked—*which places did you buy cigarettes from in the last 30 days (one month)?* The response categories (never, once, 2-3 times, 4 times or more) were provided for each of the places listed in Table 2. Participants were also asked—*When you bought, or tried to buy cigarettes, in a store during the last 30 days (one month), were you ever asked to show proof of age?* Response categories provided were: I did not try to buy cigarettes in a store during the last 30 day; Yes, I was asked to show proof of age (ID); No, I was not asked to show proof of age (ID).
Responses to the question - During the past 30 days (1 month) what brand of cigarettes did you usually smoke? - were used to identify the most popular brand of cigarette. Prices for all brands included in the responses to this question were obtained from a selection of Dunedin retail outlets during May 2002.

Table 1. Usual sources of cigarettes for all smokers and daily smokers

<table>
<thead>
<tr>
<th>Source</th>
<th>All smokers 2000 YLS</th>
<th>All smokers 2002 YLS (95% CI)</th>
<th>Daily smokers 2002 YLS (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I bought them from a shop</td>
<td>44.3</td>
<td>35.3 (30.7–40.3)</td>
<td>47.5 (41.3–53.9)</td>
</tr>
<tr>
<td>I got them from friends</td>
<td>29.2</td>
<td>24.7 (21.3–28.4)</td>
<td>13.1 (9.5–17.8)</td>
</tr>
<tr>
<td>Someone else bought them for me</td>
<td>10.4</td>
<td>12.1 (10.0–14.6)</td>
<td>14.3 (11.2–18.1)</td>
</tr>
<tr>
<td>I got them from my parents</td>
<td>3.2</td>
<td>6.7 (4.9–9.0)</td>
<td>10.0 (7.4–13.4)</td>
</tr>
<tr>
<td>I stole them</td>
<td>3.3</td>
<td>5.7 (3.8–8.5)</td>
<td>3.4 (2.0–5.9)</td>
</tr>
<tr>
<td>I bought them from another student</td>
<td>7.1</td>
<td>4.2 (2.2–7.8)</td>
<td>3.6 (1.6–8.1)</td>
</tr>
<tr>
<td>I got them from my brother / sister</td>
<td>2.4</td>
<td>2.4 (1.5–3.8)</td>
<td>2.8 (1.4–5.3)</td>
</tr>
<tr>
<td>Other</td>
<td>–</td>
<td>9.0 (7.0–11.5)</td>
<td>5.2 (3.1–8.6)</td>
</tr>
</tbody>
</table>

YLS= Youth Lifestyle Study; CI=confidence interval.

Table 2. YLS 2002 proportions (95% CI) purchasing from selected outlets, all smokers, 14–16 years

<table>
<thead>
<tr>
<th>Retail outlet</th>
<th>Never (95% CI)</th>
<th>Once (95% CI)</th>
<th>2–3 times (95% CI)</th>
<th>4 or more times (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dairy</td>
<td>37.3 (32.2–42.6)</td>
<td>21.8 (18.5–25.5)</td>
<td>17.3 (14.3–20.8)</td>
<td>23.6 (18.9–29.1)</td>
</tr>
<tr>
<td>Liquor store / hotel</td>
<td>80.5 (75.9–84.5)</td>
<td>9.0 (6.9–11.7)</td>
<td>5.8 (4.1–8.2)</td>
<td>4.7 (2.6–8.1)</td>
</tr>
<tr>
<td>Service station</td>
<td>58.1 (52.8–63.2)</td>
<td>15.4 (12.5–18.8)</td>
<td>14.7 (11.9–18.0)</td>
<td>11.8 (8.5–16.1)</td>
</tr>
<tr>
<td>Supermarket</td>
<td>77.3 (72.4–81.5)</td>
<td>8.8 (6.2–12.4)</td>
<td>6.5 (4.8–8.8)</td>
<td>7.4 (5.0–10.8)</td>
</tr>
<tr>
<td>Takeaway shop</td>
<td>85.1 (80.9–88.4)</td>
<td>6.3 (4.6–8.6)</td>
<td>3.0 (2.0–4.5)</td>
<td>5.6 (3.7–8.2)</td>
</tr>
<tr>
<td>Vending machine</td>
<td>82.4 (79.4–85.0)</td>
<td>7.8 (5.9–10.2)</td>
<td>3.6 (2.3–5.6)</td>
<td>6.3 (4.5–8.8)</td>
</tr>
<tr>
<td>Other shop</td>
<td>87.8 (84.4–90.5)</td>
<td>4.1 (2.4–7.1)</td>
<td>2.8 (1.8–4.4)</td>
<td>5.3 (3.3–8.4)</td>
</tr>
</tbody>
</table>

Results

Eligible schools (n=141) were invited to participate in the 2002 YLS; 82 schools agreed to participate (response rate 58.2%). Higher socioeconomic decile schools were slightly over-represented: 43.1% of participants attended schools of decile 7 or higher. The remainder of participants attended decile 1–3 (24.3%) or decile 4–6 (32.5%) schools. The absentee rate in participating school classes was relatively low (median 13.3% absent). Overall, 3434 young people completed the YLS; 51.7% were boys, 59.6% were from Year 10, and the mean age was 15.0 years. Maori students were under-represented in the sample: 15.4% self-identified as NZ Maori compared with 20% in the general population of equivalent age. 17

Nearly one-quarter of participants smoked at least monthly (22.7%). Smoking increased incrementally with increasing age from 21.1% that smoked at least monthly at 14 years to 22.4% (15 year olds); and, 24.9% (16 years). Commercial outlets were the most common sources of cigarettes for all smokers (35.3%), and more so for daily smokers (47.5%). Sources of cigarettes for all smokers and daily smokers are presented in Table 1 along with the sources of cigarettes reported for all smokers in
the 2000 YLS. The 2000 YLS used comparable measures to the 2002 YLS and was administered in 53 NZ secondary schools.14

More than one-third of the smokers (38.0%) had not been asked to show proof of age when purchasing cigarettes in the month preceding the survey. Similarly, more than one-third of participants had not been refused cigarette purchases on the basis of their age (35.7%). Logistic regression was used to investigate the association between smokers who were refused cigarettes (binary variable) and age. There was a significant positive association between increasing age and not being refused sales of tobacco products (OR 1.33; 95% CI 1.17–1.52); that is, older students were more likely to be able to buy cigarettes.

The total number of packs of cigarettes purchased by NZ youth in a 12-month period was estimated using data about purchasing from selected outlets (see Table 2). Participants’ responses were totalled conservatively, so that for the response category 2-3 times the mid-point was used; and for 4 times or more, 4 times was assumed. It was assumed that a single pack was purchased on each occasion: the total number of occasions was divided by 751 (the number of students 14–16 years who had smoked in the previous month), and then multiplied by 12 (months).

Based on this calculation, student smokers smoked approximately 54.7 packets per year, or 1.05 packets per week. Data were obtained from Statistics New Zealand for the 2001 Census usual resident population, 14–16 years.18 Extrapolating from the YLS monthly smoking prevalence rates for 14, 15, and 16-year-olds to the usual resident population of the same age, 37,067 young people 14–16 years were likely to be monthly smokers, at least.

Based upon this calculation, it was estimated that approximately 2,029,048 packets of cigarettes were consumed by those less than 18 years of age. This estimate is based on three assumptions:

- The mid-point of categories for purchasing cigarettes was used;
- Only one packet of cigarettes was purchased on each occasion; and
- The smoking prevalence within the population of NZ youth 14–16 years was extrapolated from sample prevalence rates for each year of age.

The average cost per pack of the brand most commonly used by youth in 2002, was $8.95. The total estimated value of purchases by youth 14–16 years, therefore, amounted to $18,159,976 of which approximately $2,272,533 was GST; $10,449,595 tobacco tax; and $5,437,848 was retained by retailers, but from which wholesale costs were deducted.19

**Discussion**

Data were obtained from a school-based cross-sectional survey; lower-decile schools and young people who self-identified as NZ Maori were under-represented in the sample.

Low socioeconomic status has been widely identified as a risk factor for cigarette smoking;20 for this reason, it is likely that this study presents a conservative estimate of the purchasing of tobacco products by under-age youth (14–16 years) in NZ. Furthermore, the assumption (that underpinned the estimation of cigarette packages
purchased) errs on the side of caution, as it is possible that more than one pack of cigarettes was purchased on each occasion.

Among the students surveyed, more than two-thirds of all smokers and nearly one-half of the daily smokers usually purchased their cigarettes from commercial sources. This finding is consistent with the model proposed by DiFranza (2001)\(^2\)\(^1\) that daily smokers require regular, reliable sources of cigarettes. Further investigation, using longitudinal data, might usefully examine the relations between smoking stages and sources of cigarettes.

When compared to the results of the 2000 YLS,\(^1\)\(^4\) greater proportions of young people reported getting cigarettes from parents, getting someone else to purchase cigarettes for them, and stealing cigarettes. A smaller proportion of students purchased cigarettes from commercial sources in 2002 (35.3%) compared with students purchasing in 2000 (44.3%). Although the difference between the surveys was statistically significant, this should be interpreted cautiously because the survey procedures were not identical.

Compliance with youth access laws is measured in NZ using controlled purchasing operations; however collated national information is not available to allow comparison between levels of compliance and the proportions of under-age youth purchasing cigarettes. Nevertheless, over one-third of youth who purchased cigarettes in the month prior to the study did so on at least one occasion without being asked for proof of age.

The apparent ease with which under-age youth are able to purchase tobacco products would suggest a need for a review of compliance enforcement. The review by DiFranza (2001)\(^2\)\(^1\) clearly identified a high level of compliance was required before youth access laws would decrease youth prevalence. It is clear that to achieve the intention of NZ legislation, greater resources are required to ensure its successful implementation. Youth access programmes, alone, are unlikely to achieve a decrease in youth smoking prevalence; however, they are critical in maintaining the consistent message that tobacco smoking is a serious health issue.

The apparent lack of consensus among tobacco control researchers on the effects of youth access restrictions should be interpreted cautiously. It is possible that poorly enforced legislation may be at least as harmful as having no legislation. Indeed, youth tobacco use is a complex problem that requires many levels of intervention, including youth access restrictions, with adequate enforcement, as part of a comprehensive prevention programme to further denormalise tobacco use.

The value of revenue generated from under-age tobacco sales was estimated by extrapolating from the smoking prevalence of students 14–16 years among a nationally representative sample of NZ adolescents, and from Census 2001 data. In the absence of data on actual retailer revenue obtained from youth, this is considered a valid measure of revenue. It is similar to the method of revenue calculation used by Laugesen and Scragg (1999),\(^1\)\(^3\) but differs in that we were able to estimate the number of packs of cigarettes bought per week from responses to the question—*which places did you buy cigarettes from in the last 30 days?*, which elicited the frequency of purchases from each category of commercial outlet.
We found that tobacco products were most often obtained from commercial sources. Extrapolation from the estimated revenue generated from under-age sales of tobacco suggests that considerable retailer and government revenue is obtained from illegal sales to youth. Furthermore, the estimate of revenue is conservative as it is based on under-age purchases by youth 14 -16 years, alone, assumes purchase of a single pack per occasion and excludes purchases made on their behalf.

The amended *Smoke-free Environments Act 1990*\(^{7}\) prohibits the sale or supply of tobacco products to youth less than 18 years. It is likely that the revenue generated from under-age sales to aged 17 years and less than 14 years is also considerable. Crude extrapolation from the survey data (to obtain estimates of total sales to all those under 18 years) suggests that total sales greatly exceed the conservative level reported and would probably exceed $35 million per year, of which $24 million would be in taxes.

This raises several issues, perhaps the most serious of which concerns the ethical implications of Government and retailer revenue generated from the development of addiction during adolescence. We have estimated that the NZ Government receives over $12.5 million in taxes from youth 14–16 years and considerably more that this for all under-age smokers.

Therefore, it would be appropriate for at least some of this money to be used to support a comprehensive youth tobacco control programme that included (but was not limited to) increased resources to ensure improved retailer compliance. In addition, there is a need for regular, timely, collated national data on visits, prosecutions, and fines so that the access programme can be properly evaluated.

**Author information:** Helen Darling, PhD Candidate; Anthony I Reeder, Senior Research Fellow; Rob McGee, Associate Professor; Social and Behavioural Research in Cancer Group; Sheila Williams, Senior Research Fellow (Biostatistician), Department of Preventive and Social Medicine, University of Otago, Dunedin.

**Acknowledgements:** The Health Sponsorship Council was the primary contributor to the 2002 YLS, with additional support from the Ministry of Health, Cancer Society of New Zealand, The Quit Group, and the Social and Behavioural Research in Cancer Group at the University of Otago. Dr Reeder and the Social and Behavioural Research in Cancer Group receive support from the Cancer Society of New Zealand and the University of Otago. The research was completed while Helen Darling was the recipient of post-graduate scholarships from the Health Sponsorship Council and the University of Otago.

**Correspondence:** Ms Helen Darling, Social and Behavioural Research in Cancer Group, Department of Preventive and Social Medicine, University of Otago, PO Box 913, Dunedin. Fax: (03) 479 7298; helen.darling@stonebow.otago.ac.nz

**References:**


Can Quit Practice: a comprehensive smoking cessation programme for the general practice team

Deborah McLeod, Elizabeth Cornford, Susan Pullon, Kawshi de Silva, Corrianne Simpson; for The Can Quit Practice Group

Abstract

Aims To develop, implement, and evaluate a programme of training and support for smoking cessation provision in general practice.

Methods The Can Quit Practice Programme was developed for delivery in general practices with a particular focus on the skills of the practice nurse (PN) in providing quit support. The Programme utilises the principles of brief intervention by the GP or PN followed by a systematic quit support programme delivered by trained practice nurses (quit advisors). Alternative implementation strategies and the provision of ongoing support and problem solving sessions were integral parts of the Programme. The evaluation used qualitative and quantitative methods to establish quit rates for participants enrolled in the Programme and explore the efficacy of programme delivery.

Results The quit rates achieved by 85 smokers (from 14 general practices) enrolled in the Can Quit Practice Programme evaluation were; 25.9% at 3 months; 22.4% at 6 months; and 20.0% at 9 months. Important components of successful implementation were: an autonomous role for PNs; well-managed practice procedures; adequate consultation time; and adequate funding for health promotion.

Conclusion Smoking cessation programmes can be successfully implemented and maintained within general practices as an integrated part of primary healthcare.

Smoking continues to be a significant cause of morbidity and premature mortality. Since 1985, tobacco control measures introduced in New Zealand have included banning tobacco advertising, increased taxation, discouraging the sale of tobacco to young people, smokefree workplaces and public areas, and smoking cessation services.

The Quitline, a national smoking cessation telephone counselling service, was established in 1999. Subsidised nicotine replacement therapy (NRT) is available to smokers through an exchange card system from the Quitline or from healthcare providers who have received recognised training, and maintain adequate recording and reporting systems. Smoking cessation guidelines based on a frequent brief intervention model have been disseminated to all primary care providers in New Zealand. The guidelines stress that there is ‘good evidence that even brief advice from health professionals has a significant effect on smoking cessation rates.’ (pg3). Free training programmes for health professionals have been made available nationally.

However, while GPs appear to be in an excellent position to deliver a focused brief intervention, the smoking cessation activity provided as part of ‘usual’ care is
variable. While many GPs ask about smoking, less record smoking status in patient notes and further questioning or follow-up of smokers is inadequate. Barriers to providing smoking cessation advice and support have been reported as lack of GP time, patients’ resistance and remuneration issues.

Extending the role of the PN has been recommended as a way of overcoming some of the barriers facing GPs who undertake health promotion work and helping to realise the health promotion potential of general practice. This recommendation is based upon evidence that individual smoking cessation advice and counselling given by nurses to their patients is an effective intervention, and that nurses gain satisfaction from the counselling role.

In response to the evidence and the above recommendation, the Can Quit Practice Programme was established. The specific objectives were to increase the frequency and effectiveness of brief intervention for smoking cessation within general practice; to provide PNs with appropriate knowledge, skills and support so that they could advise smokers and support them through quit attempts; and to develop an effective infrastructure to implement smoking cessation initiatives.

This paper describes the Can Quit Practice Programme and an evaluation of the Programme.

Methods

Setting

The general practice team is the most widely used health professional group, and four out of five New Zealanders visit their general practice at least once each year. At the time of the study, funding for GP and PN care was predominantly fee-for-services. Patients were able to choose which general practice they attended and were usually able to see the practitioner of choice within 48 hours. GP consultations were typically 12 to 15 minutes in length. PN typically had shorter consultations, often in open-plan treatment areas.

The Can Quit Practice Programme

Programme structure—The Can Quit Practice Programme encompasses two main activities: brief intervention; and the provision of more time-consuming smoking cessation advice and support including assessment, quit advice and follow-up. The practice quit advisor(s), typically a trained PN(s), provides the smoking cessation advice and support. The Programme is consistent with evidence-based guidelines, and was developed after extensive consultation with GPs, PNs, Maori smoking cessation providers, members of the Quit Group (a national smoking policy group), and a nurse experienced in providing smoking cessation support. Specialised training, clinical guides, and resources were developed for the Programme. The training is provided at two levels with each level taking one day (Table 1). CS provides training through a mix of formal teaching, video clips and practical sessions, including role-plays and case studies. Training began in May 2001.

Programme delivery—The GP or PN utilises brief intervention in the context of a consultation, records smoking status on the patient’s notes, and refers patients who are thinking about changing their smoking (or who need to quit for health reasons) to a PN who has been trained as the practice quit advisor.

The brief intervention strategy is based on the '5As' of smoking cessation:

- Asking about smoking and recording smoking status;
- Assessing readiness to quit, and
- Advising, Assisting, and Arranging appropriate follow-up for smokers.
Table 1. Specialised training, clinical guides, and resources developed for the Can Quit Practice Programme

<table>
<thead>
<tr>
<th>Training</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1 (Level 1)</td>
<td>Provides a general understanding of smoking cessation including brief intervention strategies, patient assessment, referral situations, pharmacotherapy, the exchange card programme, relapse prevention and follow-ups.</td>
</tr>
<tr>
<td>Day 2 (Level 2)</td>
<td>Suggests ways of implementing smoking cessation in a general practice setting.</td>
</tr>
<tr>
<td></td>
<td>Extends participants’ understanding and skills in motivational interviewing. Topics addressed include reflective listening, building readiness for change, guiding decisions and avoiding arguments, negotiating a plan and following up effectively.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ongoing support</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Implementation assistance</td>
<td>All practices implementing the Programme are visited at least once for staff training in the Programme and implementation assistance.</td>
</tr>
<tr>
<td></td>
<td>Practices were given a written guide to implementation, including suggested ways of recording smoking status and forms for recording patient follow-ups, provided during the training and again at practice visits.</td>
</tr>
<tr>
<td></td>
<td>Practices were encouraged to register as providers of nicotine exchange cards†.</td>
</tr>
<tr>
<td></td>
<td>On-call smoking cessation, motivational interviewing and implementation support provided.</td>
</tr>
<tr>
<td>Follow-up sessions</td>
<td>Held at regular intervals so that those providing smoking cessation advice and support can enhance their skills and network with others involved in this work.</td>
</tr>
<tr>
<td>Programme accreditation</td>
<td>Practices receive Can Quit Practice accreditation, including an accreditation certificate, if the GP offers smoking cessation support; one or more PNs undergo smoking cessation training; the practice has a system for follow-up monitoring of smokers.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Resources developed for the Can Quit Practice Programme*</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Training and reference manual12</td>
<td>Includes patient assessment guides and an implementation guide.</td>
</tr>
<tr>
<td>A starter pack of pamphlets</td>
<td>Suitable for patients at different stages in the change cycle.</td>
</tr>
<tr>
<td>Brief intervention flowchart20</td>
<td>Encompasses the ‘5As’ of smoking cessation and which can be used as a prompt by GPs and PNs during routine consultations. The flowchart is available as an A4 laminated sheet for use on the desktop and as an A3 wall poster.</td>
</tr>
<tr>
<td>Wheel of change13</td>
<td>A physical representation of the transtheoretical model that can be used by the GP or PN to determine where smokers are in the change cycle, that is, their readiness to quit.</td>
</tr>
</tbody>
</table>

*Samples are available on the website http://intranet.wnmeds.ac.nz/academic/gp/research/index.html; †Nicotine exchange cards enable patients to receive subsidised nicotine replacement therapy.

The provision of smoking cessation advice and support by the practice quit advisor incorporates the Prochaska and DiClemente transtheoretical model of change and motivational interviewing principles. The transtheoretical model of change, describes smoking cessation as a dynamic process with change occurring through a series of stages.9 Motivational interviewing is ‘a directive, client-centred style that helps clients to explore and resolve their ambivalences about quitting.’10 Practitioners working within the ‘spirit’ of motivational interviewing, aim to increase a smoker’s self-efficacy, and to avoid or de-escalate patient resistance through strategies such as refraining from judging or contradicting patients, or pushing for change prematurely.11

Programme implementation—The Can Quit Practice Programme was implemented in the greater Wellington area (Wellington, Porirua, and Hutt cities and the Kapiti Coast) in the lower North Island of New Zealand. Smoking cessation co-ordinators at GP organisations in the locality assisted in planning the Programme. All 114 practices in the locality were posted an invitation to participate and were offered free training. The postal invitation was supplemented with telephone calls and practice visits. After the training sessions, practices were offered extensive implementation support, which included one or more visits to the practice. The visits were scheduled at times when as many of the general practice team as possible were available. Different strategies to implement the Programme were
discussed to find the most suitable approach for each general practice. Assistance was provided to set up recording and recall systems.

**Programme evaluation**

The evaluation utilised both quantitative and qualitative methods.

**Quantitative evaluation**—The first 14 practices fully implementing the Programme were asked to participate in a quantitative evaluation to assess changes in smoking cessation activity by participating practices, and quit rates for patients registering with the Programme for quit support. Smoking cessation activity was evaluated by surveying all patients who had attended each practice on a randomly selected day before, and after, the Can Quit Practice Programme had been implemented (the pre- and post-implementation surveys).

Patients were mailed a questionnaire about whether they recalled being asked about their smoking, alcohol consumption, and physical activity during their practice visit. Patients, who described themselves as current smokers, were asked about their readiness to quit. Pre-paid addressed envelopes were included and one reminder letter sent to non-responding patients. In addition to the questionnaire, the notes of sampled patients were audited to determine if their smoking status was recorded.

Quit rates were audited by comparing patients from the pre- and post-implementation survey who ‘thought they should quit’ or were ‘actively doing things to quit’, with consecutive patients registering in the Can Quit Practice Programme by completing a registration form. Consent was sought from patients to forward a copy of this registration form to the researchers. Patients were followed up and interviewed by telephone, and their smoking status ascertained at 3, 6, and 9 months after registration. Two reminder calls were made, and follow-up by fax or mail attempted, for patients who could not be reached by phone.

**Qualitative evaluation**—The aim of the qualitative evaluation was to explore the relevance of the training, resources, and ongoing support from the perspective of clinical staff providing smoking cessation advice and support. The qualitative evaluation consisted of interviews with personnel from a wider range of practices than the quantitative evaluation, including those who had declined to participate in the training. A sample of 22 individuals was purposively chosen to provide a mix of PNs and GPs with varying levels of prior training and practice commitment to smoking cessation activities and who belonged to different general practice organisations, and worked in practices of different sizes and with different ethnic and socioeconomic patient profiles. Interviews were conducted between June and September 2002.

Three interview schedules were developed to accommodate: participants who had received Can Quit Practice training and worked in an accredited practice; those who had received Can Quit Practice training but their practice had not implemented the Programme; and those who had declined to participate in the training.

Interviews with Programme participants were most commonly face-to-face and were carried out by EC. Topics covered included smoking cessation activity within the practice, perceptions of the Programme, and barriers and facilitators to participation. Barriers to participation were explored in telephone interviews with those not participating in the Programme. All interviews were recorded and transcribed verbatim. Transcriptions were checked for accuracy by the original interviewer.

Data from interviews were supplemented with field notes kept by EC, which included observations, details of key events, and data gathered from contact with PNs and GPs.

**Data analysis**—Questionnaire responses and audit data were entered into a Microsoft Access 97 database then exported into EpiInfo for analyses.

Significant and recurrent themes were identified from qualitative data (interview transcripts and field notes). Data were then categorised and coded according to those themes. Validation of the themes, categories and coding was done through a process of independent analysis and subsequent agreement of results.

The Wellington Ethics Committee approved the research.
Results

Participation in the quantitative evaluation

Ten of the 14 (71%) practices invited to do so participated in both the pre- and post-implementation baseline surveys, although post-implementation data from one practice went missing and was unable to be replaced. Six of the 10 practices participated in both the pre- and post-implementation notes audits. Practices not participating in the evaluation claimed to be too busy to do so. Responses were received from 169/347 patients (49%) to pre-implementation questionnaires, and from 167/328 (51%) to post-implementation questionnaires. All 27 patients in the pre- and post-implementation surveys who identified as current smokers (including 7 who were thinking about quitting, and 11 who were actively quitting) were followed up to assess their progress.

Participation in the qualitative evaluation

In total 16 PNs and three GPs were interviewed from 16 practices. Interviews could not be arranged with the other three GPs from the original sample, although none explicitly declined to participate.

Participation in the Can Quit Practice Programme

Between May 2001 and October 2002, 48 PNs and 9 GPs from 39 of 114 (34%) general practices received training in the Can Quit Practice Programme. Forty-six of the 114 practices in the locality already offered a structured brief intervention programme.

While personnel from 15 (33%) of these 46 practices participated in the training, they only partially implemented the Programme. Personnel from 24 (35%) of the remaining 68 practices participated in the training. Seventeen (71%) of these 24 practices (with a total of 65,300 registered patients) went on to fully implement the Programme and receive Can Quit Practice accreditation.

Accredited practices included a broad spectrum of practice types, including those providing care to populations with a high percentage of patients with health subsidy cards. The acceptability of the Programme to this group was confirmed by the demographic profile of 85 consecutive patients registering for quit support from the 14 practices participating in the evaluation; 21 (60%) of the 85 patients held health subsidy cards; and 21 (25%) were Maori, 9 (11%) were Pacific, and a further 11 (13%) identified with ethnic groups other than New Zealand European.

Recruitment to training was time intensive. Direct approaches (such as invitations to individuals and faxes to practices) were particularly effective. Participation in the training allowed practices to apply to be registered providers of subsidised NRT, and this was an important aid to recruitment. Free training and continuing education credits for attending were not remarked upon by the attendees as recruitment strategies, possibly because these have become standard practice in New Zealand. As the Programme progressed, the general level of awareness of the training increased and recruitment became easier, with contact being initiated by the practices in some cases.
A key motivation for participation in the training was to improve the smoking cessation advice and support that could be offered to patients. It was not necessary to promote the value of smoking cessation with health professionals, as they were familiar with the health benefits of quitting. Barriers to recruitment for training included perceptions that the majority of patients making quit attempts would continue or return to smoking, and lack of time to attend the training.

Those persons who participated in the training confirmed its relevance and value, and commented on the usefulness of the training manual and assessment guides\textsuperscript{12} as ongoing references. The practice quit advisors, in the main, reported enjoying the counselling aspect of the work; and smokers participating in the Programme valued the support that they had received. Both PNs and GPs reported extending their use of motivational interviewing techniques to other aspects of their clinical practice. A common theme was the usefulness of the transtheoretical change model as a framework for health professionals to use to understand their patients, and the value of the concept for patient education and encouragement:

\begin{center}
I think people, if they know there is a process and they’re working through a process, they’re much more likely to keep on keeping on [PN]
\end{center}

Despite the perceived usefulness of the change model, only two of the participants reported using the wheel or ‘cycle of change’ resource\textsuperscript{13} regularly for assessment and for patient education.

Many practices found the implementation visits useful, although the Can Quit Practice team almost always initiated the visits. The visits provided the opportunity for the practice team to talk among themselves, receive information about the Programme and how it had been implemented elsewhere. The internal organisation of the practice, including the role of the PN, was a significant factor impacting on successful implementation. Without some autonomy, freedom from reception duties, and sufficient uninterrupted time to do the work, PNs working as practice quit advisors could not provide adequate quit support and the Programme could not be implemented.

Implementing the Can Quit Practice Programme in a range of general practices confirmed that all aspects of the Programme needed to be flexible enough to be adapted to the different characteristics of each general practice in which it was being implemented.

**The impact of the Can Quit Practice Programme**

**Asking and recording**—In the interviews, participants reported asking about smoking more often. A patient presenting with a smoking-related health problem was reported as the strongest trigger to asking about smoking within a consultation. Other triggers were the smell of cigarette smoke on the patient, consultations related to pregnancy, and the first consultation with a new patient. Being busy, or dealing with urgent medical problems or upset patients, were also times when patients may not be asked about their smoking. There were no significant differences pre- and post-implementation (chi-squared=0.13, p=0.719) between patient reports of being asked about their smoking by their GP (Table 2). However, although numbers were small, there appeared to be an increase in the extent to which nurses asked about smoking and about alcohol consumption.
Table 2. Patient reports of being asked about their smoking, alcohol consumption, and physical activity

<table>
<thead>
<tr>
<th>Variable</th>
<th>Smoking</th>
<th>Alcohol consumption</th>
<th>Physical activity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Post-implementation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP only</td>
<td>19/109</td>
<td>(17.4)</td>
<td>11/107</td>
</tr>
<tr>
<td>Nurse only</td>
<td>1/16</td>
<td>(6.3)</td>
<td>1/16</td>
</tr>
<tr>
<td>Total*</td>
<td>30/145</td>
<td>(20.7)</td>
<td>19/142</td>
</tr>
<tr>
<td>Post-implementation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP only</td>
<td>21/109</td>
<td>(19.3)</td>
<td>17/106</td>
</tr>
<tr>
<td>Nurse only</td>
<td>2/16</td>
<td>(12.5)</td>
<td>3/16</td>
</tr>
<tr>
<td>Total†</td>
<td>26/142</td>
<td>(18.3)</td>
<td>22/139</td>
</tr>
</tbody>
</table>

*Total includes 10 patients who were seen by both a GP and a nurse; †Total includes 3 patients who were seen by both a GP and a nurse.

PNs with more professional autonomy, strong views about smoking, and established relationships with patients, reported asking about smoking:

I basically do a lot more talking [than I used to]. Sometimes I might catch the patient before they go into the [Doctor's] room, so I'm talking to them about their smoking and whether they want to give up [PN]

Participants in the qualitative evaluation also reported an improved level and visibility of smoking status recording in the patient’s notes. Practice-wide and appropriate recording systems were considered to be an essential adjunct to asking. Several practices with computerised clinical notes had begun to use READ codes to classify smokers, so that smoking would appear on a patient's problem list. Computerised alerts that could be set to remind doctors and nurses to ask patients about smoking were considered to be too intrusive. However, the pre-and post-implementation audits did not reflect this perceived increase in recording smoking status in patient notes (Table 3)—but in those notes where smoking status was recorded, it was usually highly visible during the consultation.

Table 3. The extent to which smoking status was recorded in patient notes

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-implementation</th>
<th>Post-implementation</th>
<th>Total recorded</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Smokers‡</td>
<td>15/22</td>
<td>(68.2)</td>
<td>13/17</td>
</tr>
<tr>
<td>Ex-smokers‡</td>
<td>28/46</td>
<td>(60.9)</td>
<td>16/45</td>
</tr>
<tr>
<td>Non-smokers‡</td>
<td>34/71</td>
<td>(47.9)</td>
<td>24/49</td>
</tr>
<tr>
<td>Total smoking status recorded</td>
<td>137/220*</td>
<td>(62.3)</td>
<td>121/212*</td>
</tr>
</tbody>
</table>

*Results from 6 practices participating in both audits; †Results from all 9 participating practices; ‡Audit matched against the smoking status of those responding to the surveys.

Most (over 60%) patients in the pre-and post-implementation survey accepted the involvement of PNs and GPs in smoking cessation (Table 4).
Table 4. Patients’ attitudes to the involvement of the general practice team in providing quit support

<table>
<thead>
<tr>
<th>Patient’s opinion about the extent to which health professionals should be involved in providing quit support</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>not at all</td>
<td>extremely involved</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pre-implementation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-GP</td>
<td>10 (6%)</td>
<td>3 (2%)</td>
<td>16 (10%)</td>
<td>23 (15%)</td>
<td>33 (21%)</td>
<td>24 (15%)</td>
<td>47 (30%)</td>
</tr>
<tr>
<td>-Nurse</td>
<td>18 (12%)</td>
<td>7 (5%)</td>
<td>13 (8%)</td>
<td>24 (15%)</td>
<td>35 (22%)</td>
<td>18 (12%)</td>
<td>41 (26%)</td>
</tr>
<tr>
<td><strong>Post-implementation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-GP</td>
<td>7 (5%)</td>
<td>10 (7%)</td>
<td>16 (10%)</td>
<td>27 (17%)</td>
<td>35 (23%)</td>
<td>20 (13%)</td>
<td>40 (26%)</td>
</tr>
<tr>
<td>-Nurse</td>
<td>13 (9%)</td>
<td>9 (6%)</td>
<td>18 (12%)</td>
<td>22 (14%)</td>
<td>32 (21%)</td>
<td>17 (11%)</td>
<td>42 (28%)</td>
</tr>
</tbody>
</table>

**Assessing**—When questioned, PNs and GPs stated that they found it relatively easy to assess their patients’ readiness to quit. Most asked simple questions about smoking and then interpreted how patients responded, as well as considering their actual response:

I’ll just ask them. Have they ever thought about talking to someone about stopping smoking, about how ready they might be, whether they feel ready now, or have they tried in the past. And usually they’ll say ‘Oh I’ve tried three or four times’ [in a depressed tone of voice] or something like that. There’s not many who have never tried. Not many. And if I can, I pursue it then. There are different ways. Would you like to come back and speak with me? Or have you got the time now? Or—depends on the situation. [PN]

**Advising, assisting, and arranging follow-up**—In practices where the Can Quit Practice Programme had been implemented patients were referred to the practice quit advisor for smoking cessation advice. Consultation with the practice quit advisor might be immediate if the advisor was available, or an appointment would be made. Participants generally regarded immediate referrals as an opportunity to provide information to patients, build rapport, and encourage a return visit, rather than an opportunity to begin a quit attempt. Subsequent follow-up strategies varied, but were most commonly opportunistic at the patient’s next appointment.

**Delivering quit support**—There were strong indications that participating in the training and the Programme had led to real changes in the way patients were advised; with advice being provided earlier in the change cycle and the benefits of quitting personalised:

If you make it a personal benefit that’s related to their situation, I think that helps make it more important to them, more significant. [GP]

Providing smoking cessation support was time consuming for the practice quit advisor, and funding this time was a challenge, especially for fee-for-service practices. Self-referral and patient-initiated follow-ups were important strategies for keeping the amount of time spent on quit support within reasonable bounds. Equally importantly, they were also regarded by many practice quit advisors as a sign of patient motivation and a sharing of responsibility with the patient for their quit attempt.
Table 5. Quit rates for smokers who participated in the pre- and post-implementation surveys versus smokers who participated in the Can Quit Practice Programme

<table>
<thead>
<tr>
<th>Follow up</th>
<th>Not in Programme* (n=18)</th>
<th>In Can Quit Practice Programme (n=85)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Quit†  n %</td>
<td>Smoking†  n %</td>
</tr>
<tr>
<td>3 months</td>
<td>1 (5.6) 17 (94.4)</td>
<td>5 22 (25.9) 63 (74.1)</td>
</tr>
<tr>
<td>6 months</td>
<td>1 (5.6) 17 (94.4)</td>
<td>6 19 (22.4) 66 (77.6)</td>
</tr>
<tr>
<td>9 months</td>
<td>1 (5.6) 17 (94.4)</td>
<td>10 17 (20.0) 68 (80.0)</td>
</tr>
<tr>
<td>Continuously quit</td>
<td>1 (5.6) 17 (94.4)</td>
<td>16 (18.8%)</td>
</tr>
</tbody>
</table>

*Results from a sample of surveyed patients who reported thinking about quitting or actively quitting; †Point prevalence quit rate with the denominator including patients lost to follow-up.
Providing NRT was a significant issue for many practice quit advisors unfamiliar with prescribing. Many were concerned with the inflexibility of the exchange card programme and patient perception of NRT as a ‘magic bullet’. Over time, as PNs gained experience in providing patient education and assessing and managing different patient requirements, the problems associated with prescribing NRT decreased.

Quit rates—Can Quit Practice registration data were provided for 85 consecutive patients from 14 practices fully implementing the Programme. Quit rates were good: 25.9% at 3 months; 22.4% at 6 months; and 20.0% at 9 months. Quit rates were lower (5.6%) for the 18 smokers from the pre- and post-implementation survey sample who had indicated a readiness to quit but were not registered with the Programme for quit support (Table 5).

Discussion

Smoking cessation is a health promotion activity that general practices can effectively engage in. Programmes ranging from frequent brief intervention to more extensive counselling have been shown to have an impact on quit rates with multiple interventions and individualised advice on multiple occasions producing the best results. Evaluation of the Can Quit Practice Programme and an evaluation of the Pegasus Health programme have demonstrated that smoking cessation programmes based in New Zealand general practice, utilising the skill mix of GPs and practice nurses, are effective.

Programmes implemented in general practices have the capacity to reach smokers with few resources or who are unwilling or unable to access other agencies but who attend general practices for their health care. The challenge is to encourage and enable health professionals to engage in smoking cessation activities. In the Can Quit Practice evaluation, practices reporting they had high proportion of patients who smoked were most enthusiastic about participating.

The Can Quit Practice Programme was developed to overcome previously identified barriers to general practice participation in smoking cessation. Previous initiatives have identified the difficulty of increasing the rate with which the GP enquires about smoking status with asking being more likely to occur with adult patients and with patients with smoking related health conditions. The audit of the Can Quit Practice Programme also failed to provide quantitative evidence for any increase in asking about smoking and the recording of smoking status in the clinical notes of patients by GPs.

While it is disappointing that no increase in asking about or recording smoking status was noted the post-implementation audit occurred soon after the implementation of the Can Quit Practice Programme. Also far more practice nurses than GPs attended the Can Quit Practice training but the quantitative evaluation was based on a sample of patients selected from GP consultations. An increased rate of smoking status recording may have occurred over a longer time frame or may have been evident in nurse consultations.

The Can Quit Practice Programme was developed to focus on the potential of the PN as a health educator and it was this aspect of the Programme that appeared to be most effective. The evaluation confirmed that PNs can be effective quit advisors. PNs
reported gaining satisfaction from providing quit advice and support, and patients valued their involvement. With PNs taking on this role, a significant barrier to providing quit support in general practice, the lack of GP time, was also overcome. The ongoing availability and funding of PN time has become the new challenge, albeit one which can be met, if practices perceive sufficient need within their practice population. This extension of the role of the PN is also consistent with The New Zealand Primary Health Care Strategy, which promotes both workforce development and a multi-disciplinary approach within primary care.\textsuperscript{17,18}

Training in motivational interviewing was described almost universally by participants as a useful way of minimising patient ‘resistance’ and of maintaining the clinician’s own motivation to work with smokers. It was also described by many of the participants as a valuable approach in other clinical situations. The use of subsidised NRT was also part of the Programme. While clinicians and patients have regarded subsidised NRT as a valuable aid to successful smoking cessation, many of the practice quit advisors stressed the importance of also educating patients about the limitations of NRT.

A range of implementation strategies and the provision of ongoing support and problem solving sessions were integral parts of the Can Quit Practice Programme. Dissemination of clinical guidelines and the provision of training, either on their own or together, are not enough to bring about changes in clinical practice.\textsuperscript{19} The evaluation confirmed that the implementation visit(s) and a flexible approach to integrating the Programme into practice activities, was a requirement for effective implementation. Participating practices employed a range of strategies, charging regimes, and organisational arrangements for the delivery of quit support.

The ongoing cost of providing the Can Quit Practice Programme to general practices would include the costs of a smoking cessation trainer(s) for the training days, implementation and follow up sessions. However, adequate administrative support for the trainer in contacting practices and arranging bookings for training and follow-up sessions is essential. There is also clearly both a real cost and an opportunity cost with respect to practice nurse time for practices deciding to implement the Programme. In this evaluation practices utilised a range of strategies to address this cost including charges to patients, obtaining external funding and absorbing the cost. In New Zealand, the introduction of primary health organisations has the potential to provide a suitable funding structure if the time commitments are realistically funded.

The Can Quit Practice Programme has met the initial aim of developing and implementing a smoking cessation programme able to be effectively delivered within New Zealand general practice. This aim has been achieved by providing PNs and GPs with specialised training, on-call support and practice-based implementation assistance, and by actively encouraging practices to participate. Practice wide commitment, well-organised practice administration, communication and internal audit systems, and adequate consultation time for PNs and GPs are essential prerequisites for effective implementation. Adequate commitment to, and funding for, health promotion and disease prevention work, not only within practices but also at regional and national level are essential to facilitate practice commitment to smoking cessation.
Author information: Deborah McLeod, Research Director; Elizabeth Cornford, Assistant Research Fellow; Susan Pullon, Senior Lecturer, Department of General Practice, Wellington School of Medicine and Health Sciences, University of Otago, Wellington; Kawshi de Silva, Health Promotion Manager; Corrianne Simpson, Quit Advisor, Cancer Society of New Zealand, Wellington Division (Inc), Wellington

Acknowledgements: The Wellington Division of the Cancer Society funded the Can Quit Practice Programme and the evaluation—we thank the Society for continuing to fund training and support for health professionals. We also thank the practice nurses, GPs, and patients who participated in the Programme and its evaluation (and who taught us a great deal about the satisfaction and challenges of smoking cessation).

Correspondence: Dr Deborah McLeod, General Practice Department, Wellington School of Medicine and Health Sciences, PO Box 7343, Wellington South. Fax: (04) 385 5995; email: dmcleod@wnmeds.ac.nz

References:


The burden of death, disease, and disability due to alcohol in New Zealand

Jennie Connor, Joanna Broad, Jürgen Rehm, Stephen Vander Hoorn, Rod Jackson

Abstract

Aim To estimate the burden of death, disease, and disability attributable to alcohol consumption in New Zealand

Methods We applied the World Health Organization’s comparative risk assessment methodology at country level; separately for Maori and non-Maori where possible. We combined the best estimates of alcohol consumption in the populations, with best estimates of alcohol-disease relationships from the international and, where available, national epidemiological literature, to calculate the proportions of alcohol-related conditions attributable to alcohol.

Results We estimated that 3.9% of deaths in New Zealand in 2000 were attributable to alcohol consumption (approximately 1037 deaths), and approximately 981 deaths were prevented by alcohol, resulting in a net loss of about 56 lives. As a consequence, 17,200 years of life were lost, but only 5,300 years of life gained; a net loss of almost 12,000 years of life. The burden was substantially higher for younger age groups, for men compared with women, and for Maori compared with non-Maori. Injury was the biggest contributing cause of death and years of life lost, while positive effects were largely due to reduced coronary disease mortality in elderly people. The impact of alcohol on these conditions depended on the pattern as well as volume of drinking.

In a separate analysis that included estimates of morbidity, we calculated a net loss of 26,000 disability-adjusted life years (DALYs) due to alcohol in 2002, with 76% lost by men. Alcohol use disorders accounted for about half of all DALYs lost.

Conclusions Five main messages emerged from the analysis that can inform policy to reduce the health burden of alcohol: there are no health benefits of drinking alcohol before middle age; pattern of drinking is an important determinant of health effects; injuries are a major component of the alcohol burden; alcohol use disorders underlie many adverse effects; and the health impact of alcohol falls inequitably on Maori.

The relationship between alcohol consumption and health is complex, and a better understanding of the determinants of this relationship is essential for effective strategies to reduce harm from alcohol.

Biological and social effects of alcohol-use result from three main intermediaries or pathways: intoxication, dependence, and direct biochemical effects. These effects relate to both the average volume of alcohol consumed and the pattern of drinking. Direct biochemical effects include both harmful and beneficial effects; for example, chronic pancreatic and liver damage on one hand, and improvements in blood lipid and coagulation profiles on the other.

Intoxication is a powerful mediator of acute adverse outcomes, and risk of injury is increased at even moderate levels of consumption when there may be little subjective
experience of intoxication. Alcohol dependence, a disorder in itself, mediates the impact of alcohol on all classes of health outcome.

Average volume of drinking, or total alcohol consumption in a population, has been used to measure relationships between alcohol and disease. However, average consumption is not a good predictor of intoxication and consequent injury—or of health benefits derived from small frequent doses of alcohol, such as reduction in coronary heart disease. Such effects are better predicted by including measures of pattern of drinking. ‘Pattern of drinking’ refers to the way in which most alcohol is consumed (such as in irregular heavy drinking occasions, or binge drinking) compared with light-to-moderate drinking on a daily basis, and also ‘where and with whom’ drinking occurs.

Previous quantitative studies of the overall health impact of alcohol drinking on the New Zealand population have attempted the complicated task of estimating the net effect of the impact of alcohol on many aspects of health. The comparative risk assessment (CRA) methodology developed for the World Health Report 2002 has provided an opportunity to update and refine these estimates.

Methods

Details of the methodology used in this study are published elsewhere, and will only be briefly outlined here.

Comparative risk assessment (CRA)—The CRA methodology was developed by the World Health Organization (WHO) as a systematic approach to measuring the burden of disease attributable to a range of important global risk factors, and ranking them. CRA aims to combine best estimates of the risk factor distribution in the population, with best estimates of risk factor-disease relationships from the international epidemiological literature to measure the impact of each major risk factor. In the World Health Report 2002, global and regional burden of disease due to a range of risk factors was estimated. This included alcohol, but no country level estimates were calculated.

The WHO CRA for alcohol drew heavily on existing reports on the quantification of drug-caused mortality and morbidity in Australia and Canada, as well as reviewing new epidemiological evidence about the association of alcohol with health outcomes. New methods were developed for incorporating the effect of pattern of drinking for some conditions, and for modelling estimates where reliable data on the individual level were lacking.

This report employs the CRA approach at a country level, and for Maori and non-Maori separately where this has been possible.

Prevalence and patterns of exposure to alcohol—We calculated average daily consumption and pattern of drinking from the 2000 National Alcohol and Te Ao Waipiro surveys for New Zealanders aged between 15 and 65 years, weighted to represent consumption for the whole adult NZ population. Data from five other New Zealand (NZ) studies were used to estimate the alcohol consumption patterns for those older than 65 years, and consumption during pregnancy was estimated from survey data in order to include the health effects of drinking in pregnancy.

We used four levels of average daily consumption, with different cut points for men and women, corresponding to those used by the WHO Global Burden of Disease Study (based originally on English et al).

To capture the effect of heavy drinking episodes, we used the pattern of drinking classification developed by WHO, based on evidence about average harmful and beneficial effects of different alcohol consumption patterns in populations worldwide. The WHO pattern of drinking category for a country is determined by scoring men and women according to the proportion that drinks daily, the frequency of getting drunk, usual drinking quantity per session, fiesta binge drinking, drinking with meals, and drinking in public places.

Alcohol consumption in New Zealand had been classified by WHO as Pattern 2 of four possible patterns, where Pattern 1 is the most beneficial and Pattern 4 is the most detrimental. Based on the best available NZ survey data, we allocated non-Maori to Pattern 2 and Maori to a Pattern 3. Although
non-Maori were more likely to be alcohol drinkers and drink more often, they drank less on a typical drinking occasion, when compared with Maori. The differences were such that average alcohol consumption per day amongst Maori and non-Maori was similar, but the health implications were different.

**Alcohol-related conditions included in the study**—The selection of the conditions attributable to alcohol was based on evidence of established epidemiological relationships, assessed by the CRA group, using meta-analyses, new research, and biological evidence (for details see Rehm et al). Three groups of conditions were considered: wholly alcohol-attributable conditions, with an alcohol-attributable fraction (AAF) of 100%; chronic conditions where alcohol is a contributing cause (detrimental or beneficial); and acute conditions where alcohol is a contributing cause.

Although some conditions were omitted or combined into broader groups where detailed epidemiological evidence was lacking, most of the alcohol-related burden is in fact due to a relatively few major disease categories. The conditions included in this study are listed in Table 1. Social outcomes of alcohol consumption (such as family problems, public disorder, or workplace problems) have not been included unless they are coded in ICD-10 (International Classification of Diseases Version 10), although it is recognised that they also contribute to population health.

**Estimating alcohol-disease relationships and alcohol-attributable fractions**—A few conditions are, by definition, wholly attributable to alcohol. For all others, the proportion of the burden that was attributable to alcohol (AAF) was established from the available epidemiological evidence—by sex, age group, and Maori/non-Maori status.

For most chronic conditions where pattern of drinking had not been demonstrated to be important, the AAF was derived from combining prevalence data and relative risk estimates from meta-analyses, using standard methods for estimating attributable risk. This group included cancers, hypertensive disease, epilepsy, cardiac arrhythmias, oesophageal varices, pancreatitis, and low birth weight. The beneficial effects of alcohol on diabetes incidence, stroke, and cholelithiasis were calculated in the same way, yielding negative AArs. The AArs for unipolar depression were estimated indirectly from the prevalence of alcohol abuse and dependence in the NZ population, in the absence of better data.

Although the effect of pattern of drinking may be underestimated in some conditions (such as stroke) because pattern information has not been routinely collected in epidemiological studies, the risks of coronary heart disease, unintentional injuries, and intentional injuries are known to be associated with pattern independently of average volume. Multilevel-modelling was used by the WHO CRA group to assess the effect of pattern on coronary heart disease (CHD) outcomes, and we have applied the relative risk estimates for Pattern 2 drinkers in our analysis. As Maori have a different drinking pattern on average, a sensitivity analysis investigated the scenario where there was no CHD benefit for Maori.

Injuries make up most of the acute adverse effects of alcohol, and as the risk is associated with episodes of intoxication, it is highly pattern dependent. Car crash injuries are the single biggest cause group and are the best studied. We used a population-based case-control study from Auckland to estimate the AArs for car crash injury by age, sex, and Maori/non-Maori status. We then used the ratio of Maori AAF: non-Maori AAF (1.5) to scale the AArs for other injuries for Maori.

**Calculation of alcohol attributable burden of disease and injury: mortality, YLLs, and DALYs**—The health burden of each of the alcohol-related conditions was measured using routinely collected mortality data from the New Zealand Health Information Service (NZHIS) mortality database for 2000—and the estimated burden for each condition in disability-adjusted life years (DALYs) lost in 2002 was obtained from the Global Burden of Disease (GBD) 2000 Study.

The DALY is a summary health gap measure that integrates fatal and nonfatal outcomes (measured by years of life lost and years of life lived with disability). Mortality data were available for Maori and non-Maori separately, as well as by sex and age group, but DALY data were not. The analysis was restricted to people over 15 years of age, apart from the secondary effects of drinking by an adult.

Alcohol-attributable mortality was calculated by multiplying the mortality from each alcohol-related condition in 2000 by the alcohol attributable fraction for that condition, for each age, sex, and ethnicity subgroup. Years of life lost (YLL), a measure of the burden of premature mortality, were derived from mortality data by the ‘remaining life expectancy method.’ This method measures the difference between age at death and life expectancy remaining at that age.

As life expectancies for Maori and non-Maori differ, a model life table (Coale and Demeny model life table West level26) was used for both populations, to estimate ‘standard expected years of life lost’ (SEYLL) as in the GBD Study, and discounted at 3% per annum.
Alcohol attributable DALYs have been calculated by multiplying the DALY count for each alcohol-related condition in New Zealand in 2002 (provided by WHO) by the alcohol attributable fraction for that condition, for each age and sex subgroup. DALY counts are age-weighted and also discounted at 3% per annum. Mortality, YLL, and DALY rates have been derived from the counts (using 2000 and 2002 mid-year population estimates provided by Statistics New Zealand). Rates were age standardised by the direct method using the WHO World population as the standard population.

Table 1. Alcohol-related conditions included in the study

<table>
<thead>
<tr>
<th>Cancers</th>
<th>Digestive disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Mouth and oropharyngeal cancers</td>
<td>- Cholelithias</td>
</tr>
<tr>
<td>- Oesophagus cancer</td>
<td>- Pancreatitis</td>
</tr>
<tr>
<td>- Liver cancer</td>
<td>- Alcoholic liver cirrhosis</td>
</tr>
<tr>
<td>- Laryngeal cancer</td>
<td></td>
</tr>
<tr>
<td>- Breast cancer</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td>- Diabetes mellitus</td>
<td></td>
</tr>
<tr>
<td>Neuro-psychiatric disorders</td>
<td></td>
</tr>
<tr>
<td>- Alcohol use disorders</td>
<td></td>
</tr>
<tr>
<td>- Unipolar depressive disorders</td>
<td></td>
</tr>
<tr>
<td>- Epilepsy</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular disorders</td>
<td></td>
</tr>
<tr>
<td>- Hypertensive heart disease</td>
<td></td>
</tr>
<tr>
<td>- Ischaemic heart disease</td>
<td></td>
</tr>
<tr>
<td>- Cardiac arrhythmias</td>
<td></td>
</tr>
<tr>
<td>- Oesophageal varices</td>
<td></td>
</tr>
<tr>
<td>- Stroke, ischaemic, or haemorrhagic</td>
<td></td>
</tr>
<tr>
<td>Digestive disorders</td>
<td></td>
</tr>
<tr>
<td>- Cholelithias</td>
<td></td>
</tr>
<tr>
<td>- Pancreatitis</td>
<td></td>
</tr>
<tr>
<td>- Alcoholic liver cirrhosis</td>
<td></td>
</tr>
<tr>
<td>Conditions arising during pregnancy</td>
<td></td>
</tr>
<tr>
<td>- Spontaneous abortion</td>
<td></td>
</tr>
<tr>
<td>- Low birth weight</td>
<td></td>
</tr>
<tr>
<td>- Fetal alcohol syndrome</td>
<td></td>
</tr>
<tr>
<td>Injuries</td>
<td></td>
</tr>
<tr>
<td>- Road traffic injuries</td>
<td></td>
</tr>
<tr>
<td>- Alcohol poisoning</td>
<td></td>
</tr>
<tr>
<td>- Other poisonings</td>
<td></td>
</tr>
<tr>
<td>- Falls</td>
<td></td>
</tr>
<tr>
<td>- Drownings</td>
<td></td>
</tr>
<tr>
<td>- Other unintentional injuries</td>
<td></td>
</tr>
<tr>
<td>- Self-inflicted injuries</td>
<td></td>
</tr>
<tr>
<td>- Violence</td>
<td></td>
</tr>
<tr>
<td>- Other intentional injuries</td>
<td></td>
</tr>
</tbody>
</table>

Results

Mortality—We estimated that 1037 deaths in New Zealand in 2000 were attributable to alcohol consumption; representing 3.9% of all deaths. Alcohol consumption was also estimated to prevent 981 deaths in the same year, resulting in a net loss of 56 lives.

The mortality burden was not evenly distributed by sex or ethnicity (Table 2). In non-Maori women, deaths prevented by alcohol consumption outweighed deaths caused, but in all men, and in Maori women, more deaths were caused than prevented. The standardised alcohol-related death rate for men was considerably higher than for women in both Maori and non-Maori. The alcohol-related death rate for Maori overall was 4.2 times the rate for non-Maori, after standardisation to the WHO world population to eliminate the effect of differences in the age structure of the two populations. More lives were lost due to alcohol as well as fewer deaths prevented by alcohol in Maori compared with non-Maori, relative to the size of their populations.

More than half (51%) of alcohol-related deaths were due to injuries, 24% were due to cancer, and 25% to other chronic diseases. Most of the deaths prevented were from reduction in coronary heart disease (78%) and stroke (18%).

The predominance of injury as a cause of death in children and young adults, and of ischaemic heart disease and stroke in older adults, means that the balance of risks and benefits of alcohol consumption varied with age. Figures 1 and 2 show the balance.
between detrimental and preventive effects of alcohol for Maori and non-Maori, at
different ages. The effects of different average drinking patterns in Maori and non-
Maori, and the small proportion of Maori in the oldest age groups, are reflected in
these Figures.

Figure 1. Number of deaths caused and prevented by alcohol consumption in
2000 among Maori (by age group)

Figure 2. Number of deaths caused and prevented by alcohol consumption in
2000 among non-Maori (by age group)
Table 2. Mortality and years of life lost (YLL) attributable to alcohol (by ethnicity and sex) in 2000

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Deaths caused</th>
<th>% of all deaths</th>
<th>Deaths prevented</th>
<th>Net deaths (count)</th>
<th>Net deaths (rate)*</th>
<th>Net YLL (count)</th>
<th>Net YLL (rate) *</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maori</td>
<td>161</td>
<td>11.3</td>
<td>47</td>
<td>114</td>
<td>37.8</td>
<td>3143</td>
<td>110</td>
</tr>
<tr>
<td>Non-Maori</td>
<td>557</td>
<td>4.5</td>
<td>476</td>
<td>81</td>
<td>9.7</td>
<td>6533</td>
<td>442</td>
</tr>
<tr>
<td>Total</td>
<td>718</td>
<td>5.2</td>
<td>523</td>
<td>195</td>
<td>13.6</td>
<td>9676</td>
<td>548</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maori</td>
<td>45</td>
<td>3.9</td>
<td>26</td>
<td>19</td>
<td>1.9</td>
<td>769</td>
<td>240</td>
</tr>
<tr>
<td>Non-Maori</td>
<td>273</td>
<td>2.3</td>
<td>431</td>
<td>-158</td>
<td>-0.8</td>
<td>1468</td>
<td>112</td>
</tr>
<tr>
<td>Total</td>
<td>319</td>
<td>2.5</td>
<td>457</td>
<td>-139</td>
<td>-0.1</td>
<td>2237</td>
<td>136</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maori</td>
<td>206</td>
<td>8.0</td>
<td>73</td>
<td>133</td>
<td>19.0</td>
<td>3912</td>
<td>656</td>
</tr>
<tr>
<td>Non-Maori</td>
<td>831</td>
<td>3.4</td>
<td>907</td>
<td>-77</td>
<td>4.5</td>
<td>8001</td>
<td>276</td>
</tr>
<tr>
<td>Total</td>
<td>1037</td>
<td>3.9</td>
<td>981</td>
<td>56</td>
<td>6.7</td>
<td>11913</td>
<td>339</td>
</tr>
</tbody>
</table>

*Rate per 100,000 age-standardised to WHO world population.

Years of life lost—Years of life lost (YLL) incorporate the impact of deaths at different ages. The net effects of alcohol on YLL for the 2000 year are also summarised in Table 2. As with mortality, the burden is not evenly spread in the population—it is higher in men than women, and higher in Maori than non-Maori.

Table 3 shows the numbers and proportions of alcohol-attributable YLLs by condition. Injury is the leading cause of alcohol-related YLLs and as many injury deaths occur in younger age groups, injury is responsible for an even larger proportion of alcohol-attributable YLLs (72%) than deaths (51%).

Years of life gained by alcohol consumption are summarised by condition in Table 4. Over 80% of the life years gained were from reduction in IHD in the elderly. The balance of gains and losses due to alcohol for Maori and non-Maori in different age groups is shown in Figure 3.

Table 3. Proportions of alcohol-attributable years of life lost (by condition) in 2000

<table>
<thead>
<tr>
<th>Condition</th>
<th>Men</th>
<th>Women</th>
<th>Percent of all alcohol attributable YLLs (n=17,207)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancers</td>
<td>1189</td>
<td>1186</td>
<td>13.8%</td>
</tr>
<tr>
<td>Other chronic diseases</td>
<td>1509</td>
<td>827</td>
<td>13.6%</td>
</tr>
<tr>
<td>Injuries</td>
<td>10234</td>
<td>2200</td>
<td>72.3%</td>
</tr>
</tbody>
</table>
Table 4. Years of life gained by alcohol consumption (by condition) in 2000

<table>
<thead>
<tr>
<th>Condition</th>
<th>Men</th>
<th>Women</th>
<th>Percentage of all years of life gained due to alcohol (n=5291)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischaemic heart disease</td>
<td>3033</td>
<td>1298</td>
<td>81.8%</td>
</tr>
<tr>
<td>Stroke</td>
<td>142</td>
<td>538</td>
<td>12.8%</td>
</tr>
<tr>
<td>Diabetes</td>
<td>123</td>
<td>129</td>
<td>4.8%</td>
</tr>
<tr>
<td>Cholelithiasis</td>
<td>11</td>
<td>17</td>
<td>&lt;1%</td>
</tr>
</tbody>
</table>

Figure 3. Age-specific rates of net years of life lost (YLL) due to alcohol in 2000 (by ethnicity and gender)

Coronary heart disease mortality in Maori—The effect of alcohol consumption on CHD in Maori in New Zealand is less well understood than in populations where it has been directly studied. In the calculations above, we have assumed Maori accrue the same benefit in the prevention of CHD from drinking the same average volumes of alcohol as non-Maori, even though the pattern of drinking is different.

Recalculating the impact of alcohol on Maori assuming no preventive effect on CHD results in the total net deaths due to alcohol in Maori rising from 133 to 195, and for the whole population from 56 to 118. Due to small numbers of Maori in the affected age groups, this increases the age-standardised mortality rate from 38 to 72 per 100,000 in Maori men, and from less than 2 to almost 16 per 100,000 in Maori women. Therefore, under this assumption, there are virtually no health benefits for Maori from drinking at any age.
Disability-adjusted life years (DALYs)—Alcohol-attributable DALYs were calculated using our estimates of AAFs combined with WHO estimates of DALY burden in New Zealand for alcohol-related conditions. Due to combined outcome data, it was not possible to analyse Maori and non-Maori separately.

Table 5 summarises DALYs lost due to alcohol in 2002. The burden in men was three times that in women, accounting for 76% of all alcohol-attributable DALYs lost. Alcohol use disorders comprised the largest cause group, accounting for 49% of DALYs lost, and this proportion was the same for men and women. Approximately 11% of alcohol-attributable DALYs lost in women was due to the increased risk of breast cancer.

Table 5. Alcohol attributable DALYs; total NZ population 2002

<table>
<thead>
<tr>
<th>Sex</th>
<th>DALYs lost</th>
<th>Percentage of all DALYs lost</th>
<th>DALYs gained</th>
<th>Net DALYs lost</th>
<th>DALY rate*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>23,540</td>
<td>10.4%</td>
<td>3,910</td>
<td>19,630</td>
<td>1,075</td>
</tr>
<tr>
<td>Females</td>
<td>10,003</td>
<td>4.4%</td>
<td>3,662</td>
<td>6,341</td>
<td>386</td>
</tr>
<tr>
<td>Total</td>
<td>33,543</td>
<td>7.4%</td>
<td>7,572</td>
<td>25,971</td>
<td>726</td>
</tr>
</tbody>
</table>

*Rate per 100,000 age-standardised to WHO world population

The balance of DALYs lost and gained varied with age, and the net DALY burden of alcohol at different ages is summarised in Figure 4.

Figure 4. Net number of disability-adjusted life years (DALYs) caused or prevented by alcohol consumption in 2002
Discussion

In 2000, about 1037 deaths were caused (and 981 deaths prevented) by alcohol, resulting in a net loss of about 56 lives. Since lives lost were younger on average than lives saved, this resulted in a net loss of almost 12,000 years of life. The burden was substantially higher for men compared with women, and for Maori compared with non-Maori. Injury was the biggest contributing cause, while positive effects were largely due to reduced coronary disease mortality in elderly people. The impact of alcohol on these two conditions depended on pattern of drinking as well as volume.

A net loss of 26,000 disability-adjusted life years (DALYs) due to alcohol was calculated for 2002, with 76% lost by men. Alcohol-use disorders accounted for about half of all DALYs lost.

There are several important limitations that should be considered when interpreting the results of this study. The scope of the analysis is limited to conditions captured by the ICD-10 coding system and therefore excludes social outcomes, and many mental health conditions. Estimates from other countries have indicated that the costs of social consequences of alcohol exceed the cost of direct health consequences. There is uncertainty in the estimates arising from measurements of exposure, determination of risk relationships, and from outcome assessment, especially for non-fatal outcomes.

The methodology does not account for the lag time between exposure to alcohol and development of each condition, with current exposure used as a proxy for the relevant exposure period. Knowledge of risk-relationships is still evolving and some have been better characterised than others. Reliable prevalence and risk information is particularly lacking amongst the elderly, who are seldom participants in epidemiological research or health surveys.

This study has endeavoured to take an approach consistent with the Treaty of Waitangi. That is, analyses of the alcohol attributable burden of disease for Maori and non-Maori have been conducted separately where possible. However, the evidence base for estimating the burden of alcohol for Maori is very small, with little specific information on risk relationships and non-fatal outcomes for Maori. The extrapolation from available data sources to Maori may be less appropriate than for non-Maori, and further research needs to be undertaken to address these issues.

The mortality rates calculated from data collected in 2000 are unlikely to be affected by numerator-denominator bias, since the classification of ethnicity for mortality and census data was similar by then.

Alcohol is responsible for a considerable burden of ill-health, and further public health intervention is warranted. Moreover, most of the benefits of alcohol are based on specific patterns of drinking, which are associated with small risk for other disease endpoints, so the burden of alcohol use could be substantially reduced while retaining the benefits.

Five main messages emerged from the analysis that should inform the public health response: there are no health benefits of drinking alcohol before middle age; pattern of drinking is an important determinant of the health effects of alcohol; injury is a major component of the alcohol burden; alcohol use disorders underlie many of the adverse effects of alcohol; and the health burden of alcohol falls inequitably on Maori.
Evidence-based policy measures exist\(^2,3,5,6\) that could be used to reduce alcohol-related harm. Based on our findings, the focus of these interventions in New Zealand should be reduction of alcohol-related injury, and reduction of the disproportionate burden of alcohol for Maori. However, both of these objectives involve modifying drinking patterns, which are largely socially and culturally determined. Therefore, as well as targeted strategies, policy measures aimed at the modification of the wider drinking culture of New Zealand will be required.

Author information: Jennie Connor, Senior Lecturer (Epidemiology); Joanna Broad, Senior Research Fellow; Rod T Jackson, Professor and Head of Section, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland; Stephen Vander Hoorn, Biostatistician, Clinical Trials Research Unit, University of Auckland; Jürgen Rehm, Director, Centre for Addiction and Mental Health, Toronto, Canada

Acknowledgments: This study was based on work by the World Health Organization (WHO) for the World Health Report 2002, and the Comparative Risk Assessment Project. We are grateful to WHO for making available methods and spreadsheets, and to Colin Mathers (Coordinator, Epidemiology and Burden of Disease, WHO) for facilitating our use of Global Burden of Disease Study data.

The assistance of the following people is also gratefully acknowledged: Sally Casswell and Megan Pledger (Centre for Social and Health Outcomes Research and Evaluation) and Helen Moewaka Barnes (Te Ropu Wariki), Massey University; and John Bushnell (Wellington School of Medicine and Health Sciences), University of Otago. Datasets were also provided by Robert Scragg and Patricia Metcalf (Workforce Diabetes Survey); Stephen MacMahon, Robyn Norton and Shanthi Ameratunga (New Zealand Blood Donors Health Study); Rod Jackson, Stephen MacMahon and Robyn Norton (Fletcher-Challenge University of Auckland Heart and Health Study); Ricci Harris, Paparangi Reid and Phillipa Gander (Sleep Survey); the Ministry of Health (New Zealand Health Survey and National Nutrition Survey); and Robyn Norton, Jennie Connor, and Rod Jackson (Auckland Car Crash Injury Study).

We also thank the other members of the Alcohol Burden of Disease and Disability Group: Dale Bramley, Shanthi Ameratunga, Sue Wells, Robert Scragg, and Patricia Metcalf for their contributions.

This study was funded by a project grant from the Alcohol Advisory Council of New Zealand (ALAC). Jürgen Rehm was supported by an ALAC Visiting Fellowship in 2002/2003.

Correspondence: Dr Jennie Connor, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Private Bag 92019, Auckland. Fax: (09) 373 7494; email: j.connor@auckland.ac.nz

References:


Tobacco tax as a health protecting policy: a brief review of the New Zealand evidence

Nick Wilson, George Thomson

Abstract

Aim To review the evidence relating to tobacco taxation as a health and equity protecting policy for New Zealand.

Methods Searches of Medline, EconLit, ECONbase, Index NZ, and library databases for literature on tobacco taxation.

Results The New Zealand evidence indicates that increases in tobacco prices are associated with decreases in tobacco consumption in the general population over the long term. This finding comes from multiple studies relating to: tobacco supplies released from bond, supermarket tobacco sales, household tobacco expenditure data, trends in smoking prevalence data, and from data on calls to the Quitline service.

For the 1988–1998 period, the overall price elasticity of demand for all smoking households was estimated to be such that a 10% price increase would lower demand by 5% to 8%. Two studies are suggestive that increased tobacco affordability is also a risk factor for higher youth smoking rates. There is evidence from two studies that tobacco price increases reduce tobacco consumption in some low-income groups and one other study indicates that tobacco taxation is likely to be providing overall health benefit to low-income New Zealanders. These findings are broadly consistent with the very large body of scientific evidence from other developed countries.

Conclusions There is good evidence that tobacco taxation is associated with reduced tobacco consumption in the New Zealand setting, and some limited evidence for equity benefits from taxation increases. Substantial scope exists for improving tobacco taxation policy in New Zealand to better protect public health and to improve equity.

Customs and excise taxes on tobacco have long been used by the New Zealand Government to collect revenue. In the previous 25 years, such taxes have been justified by the Ministry of Health and some political leaders on the grounds of protecting public health. Since 1990, there have been four tobacco tax rises above the rate of inflation, in 1991, 1995, 1998, and 2000.

In 2001, 70% of the cost of New Zealand tobacco was made up by the excise and other taxes imposed by government regulation. In comparison with other developed countries, New Zealand had the 16th highest proportion of tobacco price as taxation, and a packet of cigarettes was ranked seventh in terms of affordability (as measured by minutes of labour to buy a pack of cigarettes). Although there is a significant body of data and literature relating to tobacco tax in the New Zealand context, this has only been reviewed once before, in 1995, and never in the peer-reviewed journal literature.
Methods

Medline searches were conducted for the period 1966 to July 2004, with text word search terms including ‘Zealand’ and (‘tobacco’ or ‘smoking’) and (‘tax’ or ‘price’ or ‘sales’). These terms were also used in a search of EconLit, ECONbase, and Index New Zealand. Unpublished reports were identified from the bibliographies of review articles on tobacco control in New Zealand, and by searches of the databases of the New Zealand Ministry of Health library, the Treasury library and the electronic newsletter NZ Smokefree e-News. This review has focused on health and equity related issues, with less emphasis on the numerous other aspects of tobacco taxation (such as revenue generation).

Results

In addition to the 12 New Zealand-specific articles identified in the previous review, this review identified a further 10 publications in Medline and EconLit relating to New Zealand and tobacco tax (or tobacco price or sales). Searches based on bibliographies, library databases, and the electronic NZ Smokefree e-News identified an additional 30 other relevant documents.

The impact of tobacco tax or price changes on consumption—During 1981–1991, there were eight economic studies of tobacco demand that used monthly, quarterly, or annual data on cigarettes released from factories (to proxy retail sales). All of these studies (and the one meta-analysis) indicated that price rises reduced demand (short-term price elasticities ranged from -0.08 to -0.15, and long-run elasticities from -0.07 to -0.52). For a 10% price increase, the demand would fall in the long term by 0.7% to 5.2%. The two studies that examined price reductions found that these were related to significantly increased demand. Analysis of a different data source (weekly cigarette sales from supermarkets) also found that tobacco tax increases reduced sales (at least in the short term). More recently, a Cancer Society report has detailed supermarket cigarette sales data before and after the 1991, 1998, and 2000 budgets (which included tobacco tax increases). For a period of 4 to 6 weeks after the tax increases in these respective years, it found sales reductions of 11%, 10%, and 16% for price increases of 21%, 15%, and 23% respectively.

A Ministry of Health report also describes declines in total tobacco consumption at the national level associated with the tax increases in 1998 and 2000, and for the excise adjustment on loose tobacco in 1995 (where a 38% increase reduced loose tobacco consumption by 17% in the short term). This pattern is consistent with data from the national Quitline, which indicates that the tax increase in May 2000 was associated with a marked increase in the telephone calls to the Quitline (16,000 calls in May 2000 compared with around 6000 per month in the month before and with no other tobacco control intervention introduced). Another analysis found that although tobacco consumption fell during 2000 after the tax rise in May (by 16%), the percentage of the population who smoked only declined by 3% for a 4-month period (April to July) before returning to the pre-tax level. Longer-term data indicate that the May 2000 tax rise reduced tobacco consumption between 2000 and 2001 by 10%. An analysis of household economic survey data and census data has also indicated that tobacco taxation increases reduce consumption. This work estimated that the overall price elasticity of demand during the 1988–1998 period for all households
with tobacco purchases to be in the range of -0.5 to -0.8. That is, for a 10% price increase, the number of cigarettes purchased by the average smoking household fell by between 5% and 8%.

Price changes appear to have also influenced the type of product smoked. Trends in tobacco released indicate that the tobacco tax increases between 1988 and 1995 induced a shift towards roll-your-own tobacco. Increased taxes on roll-your-own tobacco in 1995 reduced those purchases. Supermarket sales after the 1998 tax rise indicate a renewed move to budget brands and roll-your-own tobacco.

There appears to have been no published New Zealand studies on the impact of tobacco tax increases on youth smoking rates. Nevertheless, recent national survey data suggest that affordability of tobacco is relevant for youth since cigarette smoking was found to be positively related to the amount of pocket money provided to adolescents. A multi-regional survey also found that buying cigarettes by secondary school students was associated with them having ‘more money to spend’.

**Impacts of tobacco tax/price on low-income groups**—A 1985 New Zealand study considered the regressive nature of tobacco taxation. These authors reported that, for the third-to-bottom decile (by disposable income) of families with two adults and three children, over 4.5% of aggregate household disposable income went on tobacco. This compared with less than 1.5% for the top two disposable income quintiles. Similarly, a Tobacco Institute publication reported that tobacco taxation in New Zealand was regressive.

More recently, one study has reported that for some low-income households 14% of the non-housing budget was spent on tobacco—of which around 70% can be attributed to taxation. In terms of the impact of tobacco taxation increases, one analysis examined individual Household Economic Survey (HES) data. It reported that Maori and Pacific had significantly higher price elasticities than Pakeha (New Zealand European), resulting in smaller increases in spending after any price rise. Another analysis using HES data for the 1988 to 1998 period reported that “average ‘sole adult and children’ households and Maori ‘sole adult and children’ households reduced the number of cigarettes purchased after a price rise to a greater extent than other types of households”.

Other recent work has considered the adverse impact of the deprivation associated with financial hardship attributable to tobacco taxation. For all people living in the most deprived 30% of neighbourhoods, the estimated loss of life expectancy attributable to tobacco tax ranged from 0.009 to 0.044 years (i.e. from around 3 to 16 days of lost life expectancy). For this relatively deprived population, the loss of life expectancy attributable to tobacco tax was 37 to 273 times less than that attributable to smoking.

**Public attitudes to tobacco tax**—One study published in 2002 reported that only 40% of those surveyed considered that higher tobacco taxes would help people to quit smoking. In contrast, a survey in 2003 reported that 71% supported tobacco tax ‘to encourage healthier lifestyles’.
Discussion

The New Zealand evidence

There are various limitations with the quality of the New Zealand studies relating to tobacco taxation, one being the crudeness of the tobacco consumption data. This data source has often been influenced by tobacco industry practices of releasing products before and after the tax-mediated price rise. Household Economic Survey (HES) data also suffers from under-reporting of tobacco expenditure. There is also a lack of reliable data on the impact of tobacco tax on specific population groups—such as low-income New Zealanders. Nevertheless, much of the work has been published in peer reviewed journals and is of adequate quality to inform policy making. Indeed, one of the studies\textsuperscript{14} was included in a recent major systematic review.\textsuperscript{26}

To improve the information available for evidence-based policy making, further studies using HES data are needed. Similarly, the New Zealand work on tobacco affordability and youth smoking needs to be expanded—given that preventing smoking uptake is a strong argument for tobacco taxation.\textsuperscript{26,27}

Overall, it appears that the New Zealand studies consistently indicate that tobacco tax and price increases are related to reduced tobacco consumption. The findings for studies since 1995 are also consistent with those of an earlier review of New Zealand data published in 1995.\textsuperscript{4} Furthermore, the opposition by the tobacco industry based in New Zealand to tobacco taxation\textsuperscript{28} also suggests that this intervention threatens sales and industry profits.

The available New Zealand evidence indicates that tobacco tax is a regressive tax in terms of the proportion of income spent. Nevertheless, the evidence from two studies is also suggestive that at a population level tobacco taxation increases might actually be progressive (i.e. there is a greater reduction of consumption in low-income groups and therefore an enhancement of health equity). There is also evidence\textsuperscript{12} which indicates that tobacco taxation in New Zealand is likely to benefit health among the most deprived populations (because the harm from tax is small compared to the harm from smoking).

Comparisons with international data

The New Zealand data are consistent with the international evidence, with one recent systematic review reporting `strong scientific evidence` that increasing the unit price for tobacco products is effective in increasing smoking cessation and in reducing consumption.\textsuperscript{26} Similarly, a review published by the World Bank found that international data on the effectiveness of tobacco taxation to reduce tobacco consumption is convincing.\textsuperscript{29} An analysis undertaken for the World Health Report 2002 found that tobacco taxation is the most cost effective tobacco control option in all regions of the world.\textsuperscript{30}

As with many consumption taxes, tobacco taxation is generally considered in the literature to be regressive, with low-income groups being taxed disproportionately, relative to those on higher incomes.\textsuperscript{29,31} In contrast, however, some economists consider that tobacco taxation is probably progressive overall, when the benefits of taxation as a `self-control device` are appropriately accounted for.\textsuperscript{32}
This view is based on survey data from both the United States and Canada on how tobacco excise tax at a state or provincial level is associated with higher subjective well-being or ‘happiness’ among those with a propensity to smoke. However, there remains the problem of the effect on households when smokers do not quit or reduce smoking sufficiently to cut their tobacco spending.

In terms of the possible progressivity of tobacco tax increases, the limited New Zealand data is consistent with some international evidence that low-income populations and those with poorer education are more price responsive (i.e. from the United States, United Kingdom, and possibly Italy). For these reasons, tobacco taxation increases have been described by some as being ‘a progressive public health policy.’

Implications for policy makers and the health sector

The major implication of these data is that policy makers and the health sector should continue to support the use of tobacco tax as a key aspect of the country’s multi-component tobacco control strategy.

Nevertheless, tobacco taxation could be refined in several ways:

- **Clearly defining tobacco tax as a justifiable health-protecting tax**—A clear and coherent approach to tobacco taxation is needed by governments, with this tax being primarily defined as a public health intervention. This approach fits with the World Bank report on tobacco taxation that indicated that a valid rationale for the taxes is to use these as a policy instrument to achieve a desired public health target (e.g. to meet a government’s goals of reducing tobacco consumption and the prevention of smoking uptake by youth). Using this rationale, tobacco tax could be regularly increased up to the point at which government considers its public health goals are achieved. This approach makes good economic sense, in that tobacco taxation increases are one of the most cost-effective tobacco control interventions available. Thus, appropriate tobacco taxation policy will mean that Government will ultimately need to spend less on other tobacco control interventions, while achieving its goal of a lower level of smoking in society.

The World Bank report also notes that modern economic theory holds that consumers are usually the best judges of how to spend their money (the principle of ‘consumer sovereignty’). However, in the case of expenditure on tobacco, this report and other research suggests that smokers and those starting smoking do not appear to make rational and informed choices; they appear to misjudge the risks involved, and have imperfect knowledge on which to base decisions. This is partly because the public have a very poor understanding of the nature of nicotine addiction and its severity, which means that starting smokers underestimate the dangers and costs. Smokers may not accurately perceive their own risks because of over-optimism, an illusion of control, lack of high impact information, and/or an inability to evaluate the available information.

- **Adopting a long-term strategy of regular price increases combined with smoking cessation support**—Having a routine system of annual tobacco taxation increases has been recommended in a ‘World’s best practice’ review of tobacco taxation and for the New Zealand setting. This approach would also signal to smokers in a transparent manner that smoking will continually become
more expensive over time (i.e. that it is an unsustainable addiction in economic terms). Such an approach would also allow for maximum synergy between the price rises and smoking cessation publicity, and would also give smokers time to prepare for a quit attempt. The UK Government has emphasised the importance of the health basis for tobacco tax and has raised the tax on tobacco by 5% per year for a number of years over and above the inflation rate.\textsuperscript{50}

New Zealand successfully followed this strategy in 1988–89, when the Government announced the raising of tobacco tax by three steps over 10 months (each tax rise was by 50 cents).\textsuperscript{51} Accompanying these increases were a range of paid and unpaid health promotional strategies to encourage quitting.

- **Protecting low-income New Zealanders**—Tobacco taxation increases may particularly benefit low-income groups at a population level, given the available New Zealand (and some international) evidence for tax increases being progressive. Nevertheless, for those low-income smokers exposed to tax increases, it is desirable to maximise the quit rate and minimise the potential harm from financial hardship for those in the households of smokers who do not quit. Maximising the quit rate could be achieved by further strengthening support for smoking cessation (i.e. enhanced promotion of the free national Quitline service; use of subsidised nicotine replacement therapy; and use of culturally-appropriate services such as the Aukati Kaipaipa programmes).

- **Adopting a dedicated tobacco tax**—Unlike alcohol and gambling tax revenue (parts of which are used for specific purposes), no part of tobacco tax revenue is formally allocated to tobacco control, or any other purpose. Dedicated taxes can be necessary where the policy process otherwise chronically underfunds a critical public service. Tobacco control appears to have one of the strongest arguments for such a revenue stream, as otherwise, the Government uses the revenue from the sale of a lethal, addictive drug for the benefit of the general population.

New Zealand smokers and their households (who are more likely to be poorer, Maori or Pacific Peoples) are used as a revenue source with a relatively small effort to help free them of nicotine addiction. Government spending on tobacco control is equivalent to less than 3% of the tax revenue from tobacco.\textsuperscript{52} Far more funding for tobacco control is needed, given the scale of the smoking problem.\textsuperscript{53} A number of other jurisdictions have dedicated tobacco taxes\textsuperscript{29} and there is evidence that these have greatly assisted tobacco control efforts (e.g. Massachusetts in the United States\textsuperscript{54}).

- **A special inquiry on tobacco taxation**—Several issues about New Zealand tobacco taxation policy require an explicit, expert and public consideration—including ethical issues\textsuperscript{55} and the optimum basis for policy, tax rates, revenue use, and sources of official advice. Since 1988,\textsuperscript{56} there has been no specific inquiry on tobacco taxation at Ministerial or Royal Commission level. Such an investigation could allow a well-resourced, expert, and public deliberation. Considering that Government obtains over NZ$900 million dollars a year from taxes on tobacco, and the sub-optimal policies described above, a high level inquiry appears therefore to be a minimal requirement to ensure that the policy basis, ethics, and effectiveness is optimal.
Conclusions

There is good evidence that tobacco taxation is associated with reduced tobacco consumption in the New Zealand setting, and some limited evidence for equity benefits from taxation increases. The impact of taxation on consumption is consistent with a very large body of strong scientific evidence from other developed countries.

Major options for further improving tobacco taxation policy include:

- Clearly defining tobacco tax as a justifiable health-protecting tax;
- Adopting a long-term strategy of regular price increases combined with improved smoking cessation programmes; and
- Dedicating a proportion of the tobacco tax revenue for tobacco control.

Author information: Nick A Wilson, Public Health Physician; George W Thomson, Research Fellow, Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, Wellington South

Acknowledgements: George Thomson received funding from the NZ Heart Foundation, the Auckland and Wellington Divisions of the Cancer Society of New Zealand, and the University of Otago Research Committee.

Correspondence: Dr Nick Wilson, Department of Public Health, Wellington School of Medicine and Health Sciences, PO Box 7343, Wellington South. Fax: (04) 476 3646; email: nwilson@actrix.gen.nz

References:


49. Laugesen M. What is needed to reduce smoking prevalence – tobacco taxation policy should be re-designed to aid long term quitting around a national annual quit day [Editorial]. NZ Smokefree e-News. 2003;7(23), 29 September.

54. CDC. Cigarette smoking before and after an excise tax increase and an anti-smoking campaign – Massachusetts, 1990-1996. MMWR. 1996;45:966–70.


Near death episode after exposure to toxic gases from liquid manure
Louise Couch, Laura Martin, Nigel Rankin

Liquid manure is used in New Zealand as a nitrogen source in mushroom production. During its formation, toxic gases including hydrogen sulphide (H₂S), methane, and carbon monoxide are generated. In a confined space, potentially lethal concentrations can arise. This paper illustrates an under-recognised hazard in New Zealand.

Case report
A 26-year-old mushroom farm worker (Patient A) collapsed after stirring chicken manure slurry contained inside a water tank. A 49-year-old coworker (Patient B) attempted a rescue but was also overcome. Both men were pulled free through a hole cut in the side of the tank (Figure 1).

Figure 1. The tank in which the victims were overwhelmed

Bystanders performed cardiopulmonary resuscitation (CPR) on Patient A for 2 minutes. On ambulance arrival, he had a Glasgow Coma Score (GCS) of ‘4’ with decerebrated posturing. He was intubated at the scene. Patient B was unresponsive
when rescued but quickly roused to a GCS of ‘14’. Both men were transported to hospital by helicopter.

Patient A filled the emergency department with a ‘rotten eggs’ smell. Coarse bilateral crepitations were auscultated, and his oxygen saturation was 91% on 10 L. Copious amounts of foul smelling material were suctioned from the endotracheal tube.

Sodium Nitrite 3% at 0.3 ml/kg was administered for presumed H\textsubscript{2}S poisoning. Hyperbaric oxygen therapy was considered, but the patient was too unstable to transfer. In the ICU, Patient A initially required 100% oxygen. He was given imipenem for aspiration pneumonia and topical chloramphenicol for bilateral corneal burns. On day 2, his GCS was E1 VT M5. A small right-sided pneumothorax with surgical emphysema was noted. This was treated conservatively.

Patient A was orientated and extubated on day 3, and discharged home on day 6. He required ENT follow-up for otitis externa and sinusitis.

Patient B’s GCS had deteriorated from 15 to 3 during transfer, however he improved rapidly when given high-flow oxygen. He also was given sodium nitrite and treated for aspiration pneumonia and painful corneal burns. In addition, he suffered a foul post-nasal drip and nasal discharge. This was managed with oral antibiotics and nasal douches. One year later, both patients had returned to work.

Occupational Safety and Health Service (OSH) conducted studies of the gases emitted from the tank. (Table 1)

<table>
<thead>
<tr>
<th>Table 1. Gases in the tank</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Effluent tank gas</strong></td>
</tr>
<tr>
<td>Carbon monoxide</td>
</tr>
<tr>
<td>Methane</td>
</tr>
<tr>
<td>Hydrogen sulphide</td>
</tr>
<tr>
<td>Oxygen</td>
</tr>
</tbody>
</table>

*ppm=parts per million.

**Discussion**

This report illustrates how dangerous H\textsubscript{2}S exposure can be. It also demonstrates that a good outcome can occur despite an initially poor presentation. In the previous 5 years, there have been two deaths reported in New Zealand’s agricultural industry that were associated with ‘toxic fumes’.\textsuperscript{1} The ‘near miss’ rate is unknown.

H\textsubscript{2}S is a colourless, odiferous gas formed during anaerobic degradation of protein.\textsuperscript{2} It can be found naturally occurring in sewers, septic tanks, slurry stores, and mines,\textsuperscript{3} and is also a byproduct of processes involving sulphur-containing compounds. Exposures typically occur at sites some distance from emergency services.\textsuperscript{4}

At a concentration of 20–30 ppm, H\textsubscript{2}S has a characteristic malodorous scent of ‘rotten eggs’. At a higher concentration, olfactory paralysis can occur and so both victims and rescuers maybe unaware of their exposure.\textsuperscript{5,6} The toxic effects occur rapidly. H\textsubscript{2}S binds to cytochrome oxidase inhibiting oxidative phosphorylation. H\textsubscript{2}S can cause
bronchospasm\textsuperscript{6,7} and secondary hypoxia may develop due to respiratory paralysis and pulmonary oedema. Other effects are listed in Table 2.

**Table 2. Effects of H\textsubscript{2}S (from references 3 and 6)**

<table>
<thead>
<tr>
<th>Concentration H\textsubscript{2}S (ppm)</th>
<th>Symptoms and signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0004–0.02</td>
<td>Odour threshold</td>
</tr>
<tr>
<td>20–30</td>
<td>Rotten eggs’ smell</td>
</tr>
<tr>
<td>70–200</td>
<td>Irritation of respiratory tract and conjunctiva</td>
</tr>
<tr>
<td>100–150</td>
<td>Olfactory paralysis</td>
</tr>
<tr>
<td>250–500</td>
<td>Pulmonary oedema</td>
</tr>
<tr>
<td>400–700</td>
<td>Headache, dizziness, ataxia, unconsciousness</td>
</tr>
<tr>
<td>&gt;500</td>
<td>Respiratory paralysis, death (no warning)</td>
</tr>
</tbody>
</table>

\*ppm=parts per million.

Management includes rapid removal from the source, decontamination, high-flow oxygen, nitrite therapy, and consideration of hyperbaric oxygen.\textsuperscript{3,7,8} Nitrite therapy produces methaemoglobin to which the H\textsubscript{2}S preferentially binds releasing haemoglobin.\textsuperscript{5}

Liquid manure when agitated in a confined space is a potential hazard.\textsuperscript{9} The testing done by OSH (Table 1) found H\textsubscript{2}S concentrations were above the maximum allowed for short (15 ppm) and prolonged exposure (10 ppm). This was a simulation and probably underestimates the true levels of H\textsubscript{2}S to which the patients were exposed.

Information about the risks of exposure to toxic gases is briefly covered in the OSH handbook (under the *Working in Confined Spaces* section).\textsuperscript{10} It is vital that rescuers recognise this danger, and use respiratory apparatus and protective clothing to avoid further casualties.

**Author information:** Louise Couch, Emergency Medicine Registrar; Laura Martin, Emergency Medicine Registrar; Nigel Rankin, Intensivist, Middlemore Hospital, Counties Manukau District Health Board, Auckland

**Correspondence:** Dr Nigel Rankin, Department of Intensive Care Medicine, Middlemore Hospital, Private Bag 93311, Otahuhu, Auckland. Email: nrankin@middlemore.co.nz

**References:**


Suspicious pulmonary nodules

Simon Janes, Birgit Dijkstra, Carina Miles, Ian Cowan

Case report—A 55-year-old woman with breast carcinoma was admitted for a left mastectomy. A routine preoperative chest radiograph identified multiple bilateral pulmonary nodules (Figure 1). A high-resolution computed tomography (CT) scan confirmed the nodules were predominantly in the upper zones, with borderline mediastinal lymphadenopathy (Figure 2). The features were suggestive of miliary pulmonary metastases so surgery was postponed until a diagnosis was confirmed. She had no respiratory symptoms but kept parrots.

Bronchioalveolar lavage, infectious serology, serum calcium, and angiotensin-converting enzyme levels were all normal. A transbronchial biopsy showed multiple non-caseating epithelioid granulomas, containing multinucleated giant cells (Figure 3). There were no acid fast bacilli, fungi, or metastatic carcinoma cells. The granulomatous inflammation was consistent with sarcoid.
Discussion—Sarcoidosis is a chronic multisystem disorder of unknown cause that most frequently affects the lungs. Most patients are symptomatic, experiencing dyspnea or a dry cough. Haemoptysis or sputum production is rare. Pulmonary sarcoidosis has three distinct radiological patterns: type I-bilateral hilar adenopathy with no parenchymal abnormalities; type II-bilateral hilar adenopathy with diffuse parenchymal changes; and type III-diffuse parenchymal changes without hilar adenopathy. Large metastatic-like nodules are unusual but can occur.

Author information: Simon Janes, House Surgeon, Department of General Surgery; Birgit Dijkstra, Consultant Surgeon, Department of General Surgery; Carina Miles, Consultant Pathologist, Department of Anatomical Pathology; Ian Cowan, Consultant Radiologist, Department of Radiology, Christchurch Public Hospital, Christchurch

Correspondence: Simon Janes, Surgical Senior House Officer, Department of Surgery, New Cross Hospital, Wolverhampton WV10 0QP, England. Fax: +44 1753 634825; email: simonjanes@doctors.org.uk
Rethinking the regulatory framework for tobacco control in New Zealand

George Thomson, Nick Wilson, Julian Crane

Abstract

Tobacco is a particularly unusual consumer product in that it is highly addictive, kills over half its long-term users, and is a major cause of premature death and health inequalities in New Zealand. We therefore examined the place of regulatory frameworks in advancing tobacco control, and suggest the formation of a Government Tobacco Authority. Such an authority could enable the Government to specify the design of tobacco products (to maximise harm reduction), to eliminate the marketing for profit of branded products, and to appropriately control the introduction of alternative nicotine delivery devices or less hazardous alternative tobacco products. As the authority could be funded through levies on the tobacco industry, it has the potential advantage of delivering major population health gains while costing the taxpayer nothing.

The Ministry of Health has recently started a process for enhancing tobacco product regulation, and has invited public submissions to this Review. While this is a very positive development, it is part of a pattern of narrowly focusing on particular tobacco control interventions—smokefree policies, marketing restrictions, tobacco taxation, media campaigns, and cessation. Little thought has been given to the overall policy, regulatory, and organisational framework in which these interventions might occur. This article suggests a new approach to the regulation of tobacco in New Zealand.

The current Review provides us with an opportunity to consider the setting in which new labelling, disclosure, and product control regulations might be most effective. Given that reducing tobacco-related harm is one of the most important means of improving health status and reducing health inequalities in New Zealand, the suitability of the regulatory framework and organisations for tobacco control is a significant health sector issue. There is the potential to prevent thousands of cases per year of unnecessary premature death and disability amongst New Zealanders. A disproportionate number of these cases are amongst Maori, Pacific peoples, and low-income New Zealanders.

Tobacco control in New Zealand has had some effect in reducing smoking prevalence and raising awareness of the associated health issues. However, since 1991, there has been little change in overall smoking prevalence, and little improvement for Maori or the most disadvantaged.

The problem of an insufficiently regulated industry

The importance of the current Review can be seen by the present situation, where neither the government nor the public know what is in particular tobacco products, how the products are constructed, or what the consequences are of any design change. Nor do they have any mechanism for finding much of this information. Government
also does not have the power to make tobacco companies disclose their marketing tactics, or the companies' research and planning for evading regulations on packaging, warning labels, branding, product descriptors, and product measurement. This situation is for companies making products that kill at least half the people that use them long-term, when used as the makers intend.\(^5\)

A particularly strong, independent and effective regulatory framework is necessary for tobacco, given the size and nature of the tobacco companies, the effectiveness of their marketing, the addictiveness of the product and the danger of tobacco smoke to smokers and others. Of the companies operating in New Zealand, in 2002, British American Tobacco was credited with over $US 25 billion of assets worldwide, and Philip Morris with almost $US 85 billion of assets.\(^6\)

Furthermore, a recent Cochrane systematic review found that ‘tobacco advertising and promotion increases the likelihood that adolescents will start to smoke’.\(^7\) Marketing occurs wherever tobacco companies are able to make branded products available to customers, or when the companies can interact with distributors and retailers.

Tobacco companies have a history of combining to contest health measures in New Zealand and elsewhere.\(^8,9\) They have used undisclosed front groups,\(^10\) the destruction of documents\(^11,12\) and the evasion of legislation.\(^13\) The tobacco industry has demonstrated great skill in many countries and in international organisations in resisting regulation, by exerting pressure on national and international political processes.\(^14–16\)

In 2001, the World Health Assembly of the World Health Organization (WHO) passed a resolution that recognised the subversive nature of the tobacco industry. The Assembly expressed ‘great concern [about] the findings of the Committee of Experts on Tobacco Industry Documents, namely, that the tobacco industry has operated for years with the expressed intention of subverting the role of governments and of WHO in implementing public health policies to combat the tobacco epidemic’.\(^17\) The Committee of Experts on Tobacco Industry Documents reported in July 2000 that the attempted subversion had been ‘elaborate, well financed, sophisticated, and usually invisible’.\(^18\)

**A possible model: marketing control**

We suggest that the regulatory frameworks suggested by Borland\(^19\) and Liberman\(^20\) are required for the effective regulation of tobacco. These frameworks would enable the Government to make tobacco companies only sell products in New Zealand to a Tobacco Authority, would give government the ability to specify the constituents in the products, and could remove the ability of companies to sell branded products to retailers or customers. Essentially, such frameworks place a non-profit organisation between tobacco manufacturers and retailers, removing the profit motive from the marketing of tobacco to consumers (but not from retailing). We suggest that, to cover any costs to Government, the costs of such an Authority be fully recovered by a levy on the suppliers of tobacco to the Authority.

Such an authority would have at least five ways of reducing or removing tobacco-related harm. They are: modifying tobacco products; changing the way that the products are marketed; offering substitutes (e.g. nicotine replacement therapy);
controlling tobacco prices; and changing the political, social, and economic arena in which the tobacco industry operates.

An example of such a change to that arena would be ensuring greater transparency for tobacco industry activities. New Zealand is relatively suitable for the running of such non-profit marketing system, due to its well-organised border control organisations and its geographic isolation, which minimise the smuggling that could reduce the effect of controls on tobacco products. Elsewhere, involvement in smuggling is a standard tobacco company strategy to lower prices and increase sales.21,22 Another favourable factor for the success of bold tobacco control innovations in New Zealand is the Government’s commitment to reducing smoking prevalence and reducing health inequalities,23,24 demonstrated by the Smoke-free Environments Amendment Act 2003.

If tobacco manufacturing companies were required to sell only to an official or non-profit agency, that Authority would be able to control both the nature of the product, and the nature of the distribution and retail structure for tobacco. The Authority could supply current retailers with unbranded products, could restrict the number of retailing outlets, and could require conditions for retailing that were more appropriate to the sale of an addictive, dangerous drug. One eventual option would be the supply of tobacco in relatively clinical settings, where health promotion and the treatment of nicotine dependence could be the dominant imperative.

A Tobacco Authority, such as suggested by Borland and Liberman, could also appropriately control nicotine delivery devices or alternative tobacco products posing less hazard than tobacco. Suitable regulatory frameworks for such devices are urgently needed.25 The control of modified ‘safer’ cigarettes is also an urgent matter,26 with very large financial incentives pushing their introduction.27

**Legislative foundations for a tobacco authority**

The model would require legislative arrangements to ensure that before the tobacco companies exited the New Zealand market in any way, they would be required to be bonded to cover their liabilities. Otherwise, the companies could escape their liabilities by disolvement or bankruptcy proceedings. In New Zealand, a bond has been required for mining operations, to cover the possibility of bankrupt companies escaping their environmental and social responsibilities.28

In the previous 2 years, there have been at least two threats by very large tobacco companies to either exit a market, or to go bankrupt. Currently, Japan Tobacco’s Canadian branch has protection through federal bankruptcy laws to avoid a court order to pay $C 1.36 billion in fraudulently unpaid taxes.29 In September 2003, Philip Morris used the threat of bankruptcy in asking for a smaller court bond in a USA court.30

**A further regulatory framework option: possible disclosure powers**

A further regulatory approach would be to minimise the industry’s planning and executive abilities. One avenue towards this strategy would be to increase the ability of government and the public to monitor the records and planning of tobacco companies.

This approach has precedents, in that the New Zealand Government has provided powers to the Commerce Commission, under the Commerce Act, to call for
documents and to obtain evidence from witnesses, so as to be able to better control the financial markets. The Commission can also apply to a court under the *Fair Trading Act* (s.42), in circumstances where the Act had been transgressed, to get information from those contravening the Act.

If our society is willing to institute controls over financial markets, should we not equally consider similar control over market processes that seriously affect our health? Comparable powers could be given to the Tobacco Authority, or to the Ministry of Health. Powers similar to that given to the Commerce Commission under the Commerce Act (ss.98-100) would more likely be simpler and more effective to use, compared to a court-ordered process as under the *Fair Trading Act*. The practical effect of such powers is that tobacco companies could be required to disclose all of the research and planning that they, their parent and associated companies do on marketing and product design.

Policies to increase the Government and public’s knowledge about the industry could have a number of benefits. Greater knowledge about the industry’s behaviour could provide an increased public and political willingness to control or prevent any such behaviour adverse to health.

Such increased powers of inquiry for government would need to be accompanied by an increased government ability to analyse material about the industry. The New Zealand public health sector has little apparent capacity to use even the available court-released tobacco industry documents that are relevant to this country, and no government reports have been published that use them. Any Government programme to understand and control the industry would need a greater investment in making accessible the presently available industry document material. It would also need to ensure a better use of the present documentary material, for instance in getting analyses of the material published and publicised.

Such a policy direction—increasing the information about and analysis of the industry—might help change the reactive nature of tobacco control in New Zealand. For instance, at present, there appears to be no active efforts by Government agencies to establish the extent of, let alone counter, sophisticated new marketing techniques such as Internet sales, Internet-accessed video games, and the use of mobile phones and dance parties. This is in contrast to the monitoring of the industry efforts in such jurisdictions as California and the development of such programmes by the Victoria State Government-funded VicHealth Centre for Tobacco Control. The tobacco industry plans well ahead, often for decades or more. Effective tobacco control would benefit from this strategic attitude, of ‘looking upstream’, both in terms of the causes of tobacco harm, and in terms of looking ahead in time.

However, these disclosure powers would be less necessary if the Tobacco Authority marketing structure was adopted, as marketing and product design could be controlled by the Authority. Manufacturers would have to fully disclose product designs when tendering for supply to the Authority. The disclosure powers would be only necessary to establish the tobacco companies’ accountability for previous activity.

**Creating a sustainable and effective tobacco authority**

Any effective tobacco control agency will be attacked by the tobacco industry and its allies, with skilful and well resourced attempts at eroding its powers and
effectiveness. The structure of any Tobacco Authority would need to be able to withstand such attacks, and would need independence from political interference, a stable funding flow, and the ability to focus on achieving health and equity gains. PHARMAC, as an agency which has successfully and consistently taken on another large and aggressive industry (pharmaceuticals), may serve as a model for aspects of a Tobacco Authority structure. Some of the credit for PHARMAC’s success could be ascribed to the clear focus of the agency, and its relatively independent decision making structure.

Conclusions

To further protect the health of New Zealanders, the Government’s tobacco control efforts need a comprehensive regulatory framework in order to acquire much greater control over the tobacco industry. Because it is structural, laying down the long-term foundations for the removal of a major cause of disease, such a step would arguably be one of the single most important contributions to the health of New Zealanders that any government could make. And it could cost the taxpayer nothing.

Author information: George Thomson, Research Fellow, Department of Public Health; Nick Wilson, Senior Lecturer; Department of Public Health; Julian Crane, Professor, Department of Medicine, Wellington School of Medicine and Health Sciences, University of Otago, Wellington South

Acknowledgements: The authors thank an anonymous reviewer for helpful comments.

Correspondence: George Thomson, Wellington School of Medicine and Health Sciences, University of Otago, PO Box 7343, Wellington South. Fax: (04) 389 5319; email: gthomson@wnmeds.ac.nz

References:


Death under chloroform

This extract comes from the New Zealand Medical Journal 1905, Volume 4 (14), p139.

At an inquest on Margaret Wickham, who died at the Christchurch Hospital on 13th April whilst an anaesthetic was being administered, it was stated that deceased had suffered from pleurisy. On 20th March she was operated upon for the removal of part of a rib, and during the present week a second operation was decided on in order to remove one or two more ribs.

Chloroform was administered, as ether could not be used owing to the deceased’s chest trouble. The house surgeon had only commenced to administer the anaesthetic when deceased began to struggle violently. He stopped administering, and reassured the patient. On resuming the administration of the anaesthetic she again struggled and commenced to cough. Her pulse was very feeble, and stopped almost immediately, together with the breathing. Every effort was made for nearly an hour in induce respiration. Deceased took the anaesthetic very badly on the first occasion, but the only chance lay in performing the second operation. The post-mortem examination had revealed that the heart was in a healthy condition.

A verdict was returned in accordance with evidence, and the jury expressed the opinion that every precaution had been taken.
A tribute: the contribution of the Otago University Medical School and its students in World War 1

Pat Cotter

The recent return to New Zealand of the “unknown warrior” (now entombed in front of the National War Memorial in Wellington) has stimulated interest in the history of our fighting forces.

The Gallipoli campaign has long been the centre of our national acts of remembrance, although the losses of our troops in Europe far exceeded those on the Gallipoli Peninsula, Turkey. The forthcoming 90th Anniversary of the Gallipoli landing (on ANZAC Day, April 25) is an appropriate occasion to record and pay tribute to a group of our soldiers, who, to the best of our knowledge, have never before been documented. The men documented below, when war broke out, were medical students in Dunedin.

Many years later, one of the participants, DS Milne, wrote of his response at the outbreak of war:

“The Otago Daily Times, which announced the war, also declared that the war could not last longer than three months. There was not enough money in the world to run a modern war longer than that. Believing this na""ıve statement, we petitioned the authorities to let us sit our exams now [in August] instead of January when the war would be over”

In the euphoria of the occasion, there was a mixture of patriotism and a sense of adventure not to be missed.

With quite remarkable speed, the students responded. War was declared by the New Zealand Government on August 4, 1914. The following letter dated August 5, 1914 was delivered to Major Falconer, Senior Territorial Officer (who was also on the medical staff of the Dunedin Hospital).

For many years, this remarkable (framed) letter hung on the corridor wall of the old Dunedin Hospital:

```
To Major Falconer Dunedin Hospital
Dunedin: August 5th, 1914.

The following Final Year Medical Students are willing, if qualified, to place their services at the disposal of the New Zealand Government as Medical Officers for the Expeditionary Force.

[21 signatures appear]

This makes a total of 21 who have signed this letter
```
The 21 final-year students were:

<table>
<thead>
<tr>
<th>Name</th>
<th>Full Name</th>
<th>Qualification and Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>AITKEN</td>
<td>William (Peter)</td>
<td>MB ChB. MD. FRACP. MC. ChCh</td>
</tr>
<tr>
<td>BLAUBAUM</td>
<td>Ivan</td>
<td>MB ChB. Melbourne</td>
</tr>
<tr>
<td>CONNOR</td>
<td>John</td>
<td>MB ChB. MC. SBStJ. Mayor Ashburton</td>
</tr>
<tr>
<td>HASLETT</td>
<td>Selwyn Langstaff</td>
<td>MB ChB. Tauranga</td>
</tr>
<tr>
<td>JORY</td>
<td>Phillip John</td>
<td>MB ChB. FRCS. DSO London</td>
</tr>
<tr>
<td>MacCORMICK</td>
<td>Kenneth</td>
<td>MB ChB. FRCS. FRACS. DSO. CBE Pres. NZ Branch BMA. Pres. Red Cross Auckland</td>
</tr>
<tr>
<td>MacKAY</td>
<td>Donald</td>
<td>MB ChB. Kaipara</td>
</tr>
<tr>
<td>MARSHALL</td>
<td>Angus McPhee</td>
<td>MB ChB. Died before 1925</td>
</tr>
<tr>
<td>MILNE</td>
<td>Donald Stuart</td>
<td>MB ChB. Hutt Valley</td>
</tr>
<tr>
<td>REDPATH</td>
<td>George</td>
<td>MB ChB. Remuera, Chatham Islands</td>
</tr>
<tr>
<td>REID</td>
<td>Oswald James</td>
<td>MB ChB. Takapuna</td>
</tr>
<tr>
<td>REID</td>
<td>William Jameson</td>
<td>MB ChB. New Plymouth</td>
</tr>
<tr>
<td>RITCHIE</td>
<td>Thomas Russell</td>
<td>MB ChB. DPH. MOH. Dunedin. DGH Wellington.</td>
</tr>
<tr>
<td>SCANNELL</td>
<td>William Gladstone</td>
<td>MB ChB. ENT specialist Christchurch</td>
</tr>
<tr>
<td>SHARP</td>
<td>George Stanley</td>
<td>MB ChB. Featherston</td>
</tr>
<tr>
<td>SHORT</td>
<td>Aubrey Vincent</td>
<td>MB ChB. MC. Died due to 1918 flu in Christchurch</td>
</tr>
<tr>
<td>WALLIS</td>
<td>Wilfred Stanley</td>
<td>MB ChB. OBE. Orthopaedic surgeon. Rotorua OStJ Hon. Fellow NZ Orthopaedic Soc.</td>
</tr>
<tr>
<td>WEBB</td>
<td>Ernest John Herbert</td>
<td>MB ChB. BE (MINING). Died in accident – active service</td>
</tr>
<tr>
<td>WHITTON</td>
<td>Noel Stewart</td>
<td>MB ChB. Christchurch</td>
</tr>
<tr>
<td>WILL</td>
<td>William Hunter</td>
<td>MB ChB. Palmerston North</td>
</tr>
<tr>
<td>WITHERS</td>
<td>Robert Lanktree</td>
<td>MB ChB. Kaikoura</td>
</tr>
</tbody>
</table>

The University authorities agreed to the request although it is easy to think of their reluctance to see students break their training.

The fifth-year students sat their exams, were passed, commissioned, and joined the medical corps. They were not, however, registered. It is probably because the obstetrics and gynaecology training was incomplete. This was done when they returned to New Zealand in due course. All of this group, except EJH Webb (who died), returned to New Zealand.

The next big group were the third-year students whose First Professional Exam was advanced to August. Many passed and volunteered as stretcher bearers in the expeditionary force. There were various other students in their first, second, and fourth years who also enlisted.

It is interesting that they all sailed in the expeditionary force—and on the way, met the survivors from the German cruiser *Emden* which had been destroyed by the Australian cruiser *Sydney* at Cocos Island off Western Australia. This provided them with a sudden introduction to the violence of war.
Other students at Otago who are believed to have served in World War I include:

<table>
<thead>
<tr>
<th>YEAR STUDENTS</th>
<th>1914: 3RD YEAR STUDENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>BOOTH</td>
<td>Leonard Hugh MB ChB. UK</td>
</tr>
<tr>
<td>CHRISTIE</td>
<td>Robert Lyall MB ChB. Then rejoined NZMC Porirua. Died in a motor vehicle accident in 1932</td>
</tr>
<tr>
<td>CURRIE</td>
<td>Donald Eric MB ChB. Christchurch</td>
</tr>
<tr>
<td>DENNISTON</td>
<td>TH Died at Gallipoli</td>
</tr>
<tr>
<td>FISHER</td>
<td>Geoffrey Jasper St.Clair MB ChB. FRCOG. OBE. Auckland</td>
</tr>
<tr>
<td>FITZGERALD</td>
<td>Gerald Patrick MB ChB. FRCOG. Dunedin</td>
</tr>
<tr>
<td>GLASGOW</td>
<td>Wilfred Thompson MB ChB. Christchurch</td>
</tr>
<tr>
<td>KELLY</td>
<td>GA</td>
</tr>
<tr>
<td>O'SULLIVAN</td>
<td>Aeneas William Tolster MB ChB. Died due to the 1918 flu</td>
</tr>
<tr>
<td>PARKER</td>
<td>Spencer Tauria MB ChB. MRCS. LRCP. FRCS. UK</td>
</tr>
<tr>
<td>WILL</td>
<td>James Leslie Allan MB ChB. FRCSE. FRACS. CBE. Rejoined NZMC. Chairman NZ Red Cross. Pres. Orthopaedic Assn. ChCh.</td>
</tr>
<tr>
<td>2ND YEAR STUDENTS</td>
<td>BARNETT Geoffrey Michael Fulton MB ChB. MRCS. LRCP. FRCS. FRACS. Dunedin</td>
</tr>
<tr>
<td>BEGG</td>
<td>Arama Thomas MB ChB. Pukekohe</td>
</tr>
<tr>
<td>BEGG</td>
<td>Andrew Muir MB ChB. Dunedin</td>
</tr>
<tr>
<td>FRASER</td>
<td>Charles Stanley Frederick MB ChB. Timaru</td>
</tr>
<tr>
<td>SEFTON</td>
<td>WF</td>
</tr>
<tr>
<td>THOMSON</td>
<td>George Herbert MB ChB. WW2 POW. New Plymouth</td>
</tr>
<tr>
<td>1ST YEAR STUDENTS</td>
<td>CHURCH Robert Alan Henry MB ChB. MM Marton</td>
</tr>
<tr>
<td>LAIDLAW</td>
<td>RR</td>
</tr>
<tr>
<td>KITCHEN</td>
<td>Raymond Leopold Albert LRCPED. LRCS. ED. LRFPS. MRCPEd. Hastings</td>
</tr>
<tr>
<td>PATERSON</td>
<td>James Aitkenhead MB ChB. Auckland</td>
</tr>
<tr>
<td>SALMOND</td>
<td>Kenneth Guthrie MB ChB. Feilding</td>
</tr>
<tr>
<td>FISHER</td>
<td>J</td>
</tr>
<tr>
<td>CAMERON</td>
<td>N</td>
</tr>
<tr>
<td>OTHERS</td>
<td>CHRISTIE Alan Leslie MB ChB. Died due to the 1918 flu</td>
</tr>
<tr>
<td>BELL</td>
<td>Leslie George MB ChB. Lumsden</td>
</tr>
<tr>
<td>CHILTON</td>
<td>Charles Died at Gallipoli</td>
</tr>
<tr>
<td>JAMESON</td>
<td>Alfred Barrett MB ChB. FRCGP Chairman Council. MSc. Orakei</td>
</tr>
<tr>
<td>JOSEPH</td>
<td>Edward Gordon MB ChB. Edin. MRCS. LRCP. MS.Minnesota. Died in Israel</td>
</tr>
</tbody>
</table>
After the end of the Gallipoli campaign, all the medical students were recalled to Dunedin to complete their training. The imminent shortage of doctors was appreciated. At that time, the course was 5 years. Such was the calibre of these men that several (notably JL Will, RL Christie, and FM Spencer) qualified and rejoined the Army to serve in Europe. Some served in World War 2.

Photograph 1. This is probably a photograph of all the unqualified students taken on their return from Gallipoli to Dunedin (late 1915–early 1916)

*Back Row*—JL Will, CS Fraser, GP Fitzgerald, RL Christie, N Cameron  
*Middle Row*—GH Thomson, RA Church, DE Currie, GJS Fisher, FM Spencer, KG Salmond.  
*Front Row*—WF Sefton, RLA Kitchen, GA Kelly, ST Parker, LH Booth, AM Begg, AW O’Sullivan  
*Sitting (on floor)*—AT Begg, LG Bell, WT Glasgow  
*Absent*—GMF Barnett, RR Laidlaw, JG Stewart, EG Joseph, J Fisher
Odd snippets of information emerge from the growing biographical record. Dr DS Milne recalled (in a letter written 50 years later) that his first case was a man whose leg was badly cut by a kick from a horse on Wellington wharves before the troopships sailed. The same doctor noted that his first case on reaching the NZ Army Camp in Egypt was a similar cut from a horse’s hoof.

This extract from the Otago University Review of 1915—an article entitled *ANZAC Anecdotes*—helps to understand the culture shock of the students when they reached the battlefront in Turkey:

“Some of us were fortunate enough to land on Gallipoli on the first day of landing. It seems still that we were in a dream. But a little previously, in the perfect morning hours, we had glided out of Lemnos Harbour, and now we were standing in this awful inferno, helplessly holding a stretcher in a dazed manner with shrapnel spitting all round us. But a few trenchant remarks from our officer wakened us up, and we speedily assisted to look after the enormous number of wounded who covered the beach. Barge after barge was filled up and taken away to the transports and Hospital Ships”

Two of the first 21 died tragically. EJH Webb, who had completed a degree in mining engineering before turning to medicine, died at Colombo in Ceylon (Sri Lanka) from a shipboard accident while on the voyage out from New Zealand in 1914.

Another, AV Short, returned to New Zealand shortly before the end of the war. He became a senior house surgeon at Christchurch Hospital and died on November 15, 1918 (4 days after the Armistice was signed) from pneumonia contracted while battling the terrible influenza epidemic which swept the world that year.
Geoffrey Barnett with his brother Ralph and father Louis (later Sir Louis) all served together at Gallipoli (we have a photograph of them).

Others enjoyed long and distinguished careers in New Zealand and abroad. Kenneth MacCormick, for example, served in Egypt, Gallipoli, and France. He won the DSO in 1917 and returned to become a surgeon and urologist in Auckland. During World War 2, he served as Director of Medical Service for the 2NZEF from 1940 to 1943 with the rank of Brigadier. In addition, he was President of the NZ branch of the BMA in 1953-54 and President of the NZ Red Cross.

PJ Jory remained in Britain after World War 1 as an eminent ENT specialist. In World War 2, he commanded a military general hospital and went on to a spell as a ship’s surgeon. When he died in 1973, the *Lancet* recalled him as “Wise and good, humorous and kind, and in all his many interests eager and generous.” Ivan Blaubaum later served in the Australian Army Medical Corps and then at Melbourne Children’s Hospital.

TR Ritchie, a tailor’s son who was born in Gore, was for a time Medical Officer of Health in Samoa and later (from 1947 to 1950) New Zealand’s Director General of Health. WS Wallis, born at Opawa in Christchurch, became one of New Zealand’s pioneer orthopaedic surgeons working in hospitals at Rotorua.

Others, of the original 21, went on to serve communities in many parts of New Zealand. RL Withers, for instance, practiced in Kaikoura from 1919 to 1949. WG Scannell was an EENT specialist in Christchurch. John Connor served in Ashburton from 1922 to 1965, and was Mayor of the town from 1940 to 1944. William (Peter) Aitken was a physician in Christchurch up to his death in 1958. George Redpath practiced for a time in the Chatham Islands.

After all these years, the information available is limited. In piecing this together, I have been helped by TS Weston, LJH Davies, and NW Fitzgerald. There is considerable information in the biography of FM Spencer *A Doctor at War* by his daughter Christine Daniel. There will be errors and omissions. Any corrections and additional material will be welcomed.

The record of medical practitioners in New Zealand 1840–1930, *Historia Nunc Vivat*, by Rex Wright St Clair (who died recently), has been invaluable in tracing the later careers of these men. It is remarkable how many returned and picked up the threads of their lives. There were some very notable ones among this elite group.
Photograph 3. Photograph of the model of the Gallipoli Peninsula made by PC Fenwick on his return to Egypt. The model is on display in the Auckland War Memorial Museum

Note: This work has been done using the facilities of the Cotter Medical History Trust. For several years, the Cotter Medical History Trust in Christchurch has been recording the lives and careers of our earlier doctors, and now has an archive of many hundreds of often quite comprehensive records. All the information in this article is from the Trust Archive. Corrections and further information are actively sought and encouraged.

Author information: Pat Cotter, Retired Surgeon (involved in the Medical History Trust), Christchurch

Correspondence: Pat Cotter, 63 Rossall Street, Fendalton, Christchurch. Email: naylorna@xtra.co.nz
Chronic obstructive pulmonary disease (COPD) and evidence-based guidelines of management

There are several high-level guidelines available for the treatment of acute exacerbations of COPD. The recommendations include nebulized salbutamol ± ipratropium, oral corticosteroid, and pulmonary education and rehabilitation.

Antibiotic therapy is not routine, but should be considered with increased sputum volume, and non-invasive positive pressure ventilation (NIPPV) should be considered in all patients with pH <7.35 and respiratory rate >30.

In selected cases, the use of long-term oxygen therapy improves survival. On the other hand, most guidelines advise against the use of intravenous aminophylline. A recent paper from Melbourne reports on an audit on the management of 84 such patients. They report that recommendations for steroid initiation and avoidance of aminophylline were well used but concordance rates for other recommendations were generally less than 60%.

Malignancy and venous thrombosis

It has long been recognised that there is an association between cancer and venous thrombosis—in fact it was first documented by Trousseau in 1868. A recent study from the Netherlands tries to quantify and qualify this important subject.

In this large case-control study of venous thrombosis, they found that there was a seven-fold increased risk for venous thrombosis in patients with a malignancy. Gastrointestinal cancer, lung cancer, and hematological cancer were the malignancies associated with the greatest relative risk and, unsurprisingly, the more advanced the disease, the greater the risk. The risk is approximately 12- 17-fold increased for patients with cancer who have the factor V Leiden or the prothrombin 20210A mutation.

The authors conclude that rather than screening for these factors “it may be more cost-effective to consider prophylactic anticoagulant therapy for patients with cancer who have an increased risk to develop venous thrombosis.”

Drug-eluting coronary stents?

Coronary artery stenting has revolutionised the management of ischaemic heart disease. However restenosis is a major limitation to long-term success, and it is estimated that 14% of patients who undergo stent implantation require a second intervention within a year to manage restenosis. Could this be overcome by the use of drug-eluting stents?
Sirolimus, a macrolide antibiotic with immunosuppressive and antimitotic properties, was found to be potent in reducing the restenosis incidence from 35.4% to 3.2%. So why not use sirolimus-eluting stents routinely?

You know the answer—too expensive. Three papers in the Canadian Medical Journal tackle this problem. Their conclusions—a split vote.

CMAJ 2005;172:323–5, 345–51, and 361–2

Medical malpractice—USA

We are familiar with the litigation problem faced by doctors in the US. I recall the American neurosurgeon who told me that it took his earnings for the first 5 months of each year to pay his malpractice insurance!

Now, however, George Bush has announced that he is to ask Congress to impose strict limits on medical malpractice lawsuits, saying that doctors “should be focused on fighting illnesses, not on fighting lawsuits.” He has proposed that Congress should set a limit of $US250,000 for non-economic damages, such as “pain and suffering.” As the Republicans have a Senate majority it may even happen.

But, the immediate past president of the American Medical Association has pointed out that most medical liability claims—almost 70%—do not result in any payments but still cost an average of US$90,000 to defend sucessfully.

BMJ 2005;330:164

Bacterial biofilm

Why don’t antibiotics eradicate all bacterial infections? Because they can’t get into the biofilm. The what? A biofilm is a community of microorganisms that are associated with a surface and typically enveloped in an extracellular matrix. Apparently they’re everywhere, e.g. the plaque on human teeth—that’s why the dentist likes to scrape it off.

More importantly, biofilm-associated infections are related to biomaterials and implants, such as infection associated with intravascular catheters and prosthetic-valve endocarditis. And we all know how the orthopaedic surgeons fear infection in their joint implants. So bacterial biofilm is a major problem. At the frontline, we can all help by minimising the risks of dubiously needed intravenous and bladder catheters.

Bariatric surgery in New Zealand

We commend the authors (Martin I. Bariatric surgery: folly or the future? [editorial]. URL: http://www.nzma.org.nz/journal/117-1207/1209 and He M, Stubbs R. Gastric bypass surgery for severe obesity: what can be achieved? [original article] URL: http://www.nzma.org.nz/journal/117-1207/1207) on their bariatric surgery papers in the 17 December 2004 issue of the Journal. The papers were timely, and contained some important messages. However, some issues raised require further discussion.

As mentioned previously, bariatric surgery is not new. Gastric bypass surgery has been around for over 30 years. Improved surgical technique and perioperative care over this time now result in clear health benefits from gastric bypass surgery.¹ For instance, bariatric surgeons at MacGill University Health Centre (MUHC), Montreal, Canada, in an observational 2-cohort prospective study, have shown that gastric bypass surgery decreases long-term mortality (0.68 % versus 6.17%), morbidity, and the use of healthcare resources in morbidly obese patients.²

When compared to non-operative management in severely obese patients, bariatric surgery is increasingly shown to have better outcomes and cost-effectiveness,³ even in the absence of evidence from well-constructed randomised clinical trials. Many operations are currently considered to be “gold standard” treatments despite a lack of randomised clinical trials to support their use. Such examples include laparoscopic cholecystectomy, total mesorectal resection of rectal tumours, and liver resection.

Surgeons trained in gastric bypass surgery, with appropriate workloads to maintain skill level, achieve excellent results.⁴ The volume-outcome relationship is, however, a complex issue. Preoperative assessment and treatment of comorbidities; perioperative care via anaesthetists, intensivists and ward staff; plus input from nutritionists, physiotherapists, and psychologists are all important components in achieving good outcomes. Intuitively these variables are likely to be of higher quality in a large-volume unit. It is imperative that low-volume units (<25 procedures/year) strive to maximise such supportive care if they are to achieve similar outcomes.

In the USA, an emerging bariatric operation amongst the American Society for Bariatric Surgery is laparoscopic gastric banding. In Australia, it is the primary operation.⁵ There is no randomised, clinical trial comparing laparoscopic banding with gastric bypass surgery. Part of the reason for this is there is no universally accepted gastric bypass procedure.

One area of ongoing debate is the role of laparoscopic adjustable banding and laparoscopic gastric bypass surgery. Gastric bypass is associated with greater resolution of comorbidities and less mortality than laparoscopic banding.⁶ To date, this has not been demonstrated with adjustable banding; however, the attraction of laparoscopic banding, its safety, adjustability, reversibility and relative ease of surgical input are attractive features for the patient and surgeon alike.
Hopefully, in time, randomised clinical trials will be performed to compare these procedures in units with large volume and experience in both techniques; however, it is very unlikely such a trial would be able to get off the ground in New Zealand.

When comparing outcomes of operative techniques, it should be noted that, since the introduction of laparoscopic surgery, the length of hospital stay has fallen significantly in most instances. As surgeons gain more confidence in discharging postoperative patients sooner after laparoscopic procedures, it is expected that length of stay will also be reduced in patients after open procedures. We are already seeing this trend at North Shore Hospital. Our median stay for laparoscopic bypass is 3 days. As a result of our experience with laparoscopic early discharge, we have now been able to discharge many of our open gastric bypass patients on postoperative days 3 or 4.

We agree that bariatric surgery should be performed in the public system in New Zealand. However it must be emphasised that good outcomes from surgery are achieved in patients who are committed to weight-reduction lifestyles through sensible diet and exercise regimes. In the absence of severe obesity comorbidities, patients should only be offered surgery in the Public Sector after demonstrating compliance to some simple advice regarding exercise and lifestyle.

There are currently limited resources in New Zealand to offer bariatric surgery to all those who would benefit from it. Aside from funding issues, there is also a paucity of surgeons working in the public system trained in bariatric techniques. Hence the upper gastrointestinal surgical trainee is currently likely to receive minimal exposure to bariatric surgery.

We echo Professor Martin’s comment on the need for a nationwide database on outcomes from bariatric surgery in both the public and private systems. To add weight to the above statements, it is imperative that results from bariatric surgery continue to be published.

North Shore Hospital, Auckland, has been running a bariatric surgery programme since October 2001. We have been collecting data prospectively on patient outcomes following weight reduction surgery in the public system. These results will be from a population of carefully selected patients with proven compliance to lifestyle advice, or patients with severe obesity-related comorbidities. This represents a somewhat different population to those from the Wakefield Hospital Study and will hopefully add further evidence that modern bariatric surgery is safe and produces good outcomes for both the patient and society at large.

Drs Paul Samson and Michael Booth
North Shore Hospital
Auckland

References:


Screening for prostate cancer: a patient’s view

I’m 44 years old and part way through a combined EBRT and brachytherapy treatment programme for prostate cancer. A routine work medical detected a slightly elevated PSA (4.2). After being retested some months later, and a biopsy, the diagnosis was made. I have no family history, so the inclusion of the PSA test in the medical was random and, in my mind, fortuitous. My own GP told me he never includes PSA tests in a routine medical for an under 50 year old, unless there are special circumstances.

The views of Associate Professor Richardson (in the February 11 and February 25 issues of the Journal) on prostate cancer screening, supported (although Nazi references to criticise another’s position are better described as unfortunate and intemperate) by Dr Corwin, seem flawed to me.

Screening programmes, like any form of population sampling, has a risk of bias. Politicians, marketers, credit card companies, researchers, and other users of sample data know this. But that doesn’t stop them gathering sample data and then making adjustments to ameliorate bias risks, or applying standard statistical techniques so as to not overstate the confidence that can be had in the analysis.

We’d live in much less well-informed world if use were only ever made of data that are free from any bias or analysis of which we are 100% confident.

The real issues, from a patient perspective, are:

• Is early diagnosis better than late? Surely, the answer must be yes. If only because diagnosis gives choice.

• What should a patient do when given the diagnosis? The answer, obviously enough, is to choose a course of treatment. This may be “watchful waiting”.

My own experience is that the options and risks were well explained, there is plenty of readily accessible literature, and the final choice was mine. Even the possibility of histological error was explored.

Associate Professor Richardson may be right in her contention that treatment of early prostate cancer has uncertain benefits, and in a year or two I might wish not to have the side effects of treatment.

But far more certainly she is wrong to suppose, as she implicitly does, that for my own sake I shouldn’t have had the chance to make that choice, or that I had insufficient information to make it rationally.

Name withheld by request
Regarding ‘Investigation for iron deficiency anaemia’


Firstly, they have shown that faecal occult blood testing is probably worse than useless with so many false negatives and positives. Secondly, a quarter of these anaemic patients will have bowel cancer. Colonoscopy is therefore mandatory. However a disappointing feature is that fewer than half of their patients underwent duodenal biopsy. I know that I have missed the diagnosis of coeliac disease in the past. This condition is surprisingly common, being present in 1–2% of our population. The symptoms are legion and variable, but iron deficiency anaemia is certainly a presentation.

I now routinely biopsy the antrum and duodenum in every gastroscopy, and consequently make this diagnosis in four or five adult patients a year. Coeliac disease may well have been missed somewhere in this study’s cohort of 85.

John P Dunn
Director
Endoscopy Auckland
CARPA standard treatment manual (New Zealand edition)

A workshop was held in Dunedin on 26\textsuperscript{th} and 27\textsuperscript{th} of January, 2005 with the intention of investigating the possibility of producing a Standard Treatment Manual for New Zealand rural and remote practice. Sabina Knight, the Chair of the Editorial Boards of CARPA and CRANA, attended the workshop along with 34 Nurses and Doctors from throughout New Zealand. Following 2 days of discussion and debate, strong support from the participants of the workshop was articulated for this project.

The aim of this initiative is to provide clinicians in primary care, particularly those in remote practice, with evidence-based standard treatment guidelines from which to base their clinical decision-making. From the Dunedin meeting, an Editorial Committee was formed with the aim of publishing a New Zealand publication of evidence-based standard treatment guidelines for use in primary care and remote/rural practice, based on the Australian CARPA Standard Treatment Guidelines 4\textsuperscript{th} edition. Once a draft document has been produced, it is planned to have this reviewed by a group of expert clinicians before the final version is published.

The Editorial Committee would like to invite interested clinicians to participate in the KIWI CARPA initiative either as a member of the Editorial Committee or as a member of the Review Group. The Editorial Committee meets via teleconference each fortnight, the reviewers have yet to be convened. Membership of both groups will require a certain commitment of time and effort, and a willingness to see the project completed.

If you are interested in this initiative, please contact:

- **Mathew Stokes – interim Chairman**  
  Lumsden Health Care  
  58 Garden St, PO Box 20  
  Lumsden, Southland  
  (pn.stokes@xtra.co.nz)

- **Kevin Whitney – interim Vice-Chairman**  
  Tokanui Medical Centre  
  Tokanui, Southland  
  (whitneykm@xtra.co.nz)

- **Kate Baldwin**  
  Private Bag 1921  
  Dunedin  
  (Kate.Baldwin@otagodhb.govt.nz)

Kevin Whitney  
Vice-Chairman (interim)  
Kiwi CARPA project
Special offer: CD ROM of the New Zealand Medical Journal

The first 2 years of the online New Zealand Medical Journal (June 2002–June 2004) are now available in CD ROM format. This means you can own your own copy of the NZMJ for this 2-year period.

Cost

Within New Zealand: NZ$21 (includes GST, postage, and packaging)
Outside New Zealand: NZ$25 (includes $5 postage and packaging)

If you wish to purchase the NZMJ CD ROM, please email Debbie@nzma.org.nz to arrange payment.
Calling obituary writers

Obituaries do not write themselves, oddly enough. They require organisation, time, and respect for our departed colleagues.

Many decades ago, our medical practitioners were Otago graduates with a sprinkling of Brits. Things have changed. With the Auckland Medical School and our overseas-trained colleagues coming from a wide variety of medical schools, it is no longer a tight little club where everyone knows everyone else.

Thus we need a network to tell us who has died and who amongst family and colleagues can best give the rest of us some overview of the life, both professional and social, of our dead colleague.

For several years I have tried to rejuvenate this part of the Journal. I have been greatly helped in this task by colleagues in Auckland, Wellington, and Dunedin who scan the local newspapers. Anyone whose death notice does not appear in one of the four main dailies is liable to miss out.

Who is to write the obituary? Someone who cares. It need not be a literary masterpiece but should give something of the texture of the person’s life. Most funerals have a eulogy and the eulogist is often in the best position to help with an obituary.

About 400 words is usual but, with the electronic journal, space is no longer the problem it was in the days of hard copy.

The next time a colleague dies, ask yourself: “Who is going to do the obituary?” It could be you or someone whose arm may need only a gentle twist.

Most of the Journal belongs to the younger and brighter of us but the obituaries belong to us all. Even the old and cranky.

Roy Holmes
Coordinator of Obituaries
NZMJ