Inhaling Inequality
Tobacco’s contribution to health inequality in New Zealand

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Foreword

Tobacco consumption makes a major contribution to both socioeconomic and ethnic inequalities in health in New Zealand. Indeed, tobacco has been widely recognised as the leading preventable cause of premature mortality in developed countries. New Zealand is internationally recognised for the quality and comprehensiveness of its tobacco control programme. Nevertheless, there is still some way to go, as this report makes clear.

Building on an earlier report in the Occasional Bulletin series, which analysed the socioeconomic gradient in life expectancy, this report attempts to quantify the contribution of tobacco to the loss in life expectancy experienced by males and females, different ethnic groups, and neighbourhoods varying in their degree of material deprivation.

Overall it concludes that the loss of health to tobacco accounts for about one-fifth of the gender difference in life expectancy at birth, one-quarter of the inequality between Māori and non-Māori, and one-third of the deprivation gradient.

The report highlights the importance of tackling the root causes of social inequality, as well as treating the ‘symptoms’, such as smoking. The estimates of the contribution of tobacco to social inequalities in health presented in this report will provide a baseline for future monitoring, and will be of value in the formulation and evaluation of tobacco control policies and programmes. The report should be of interest to policy makers and their advisors, health professionals concerned with tobacco control, and a wide range of community groups concerned with public health.

Comments on this report are welcomed, and should be sent to Public Health Intelligence, Ministry of Health, PO Box 5013, Wellington.

Don Matheson
Deputy Director-General
Public Health Directorate
Acknowledgements

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Disclaimer

Opinions expressed are those of the authors and do not necessarily reflect the views of the Ministry of Health.
Introduction

In New Zealand, as in other developed countries, socioeconomic and ethnic inequalities in health are of great concern (Howden-Chapman and Tobias 2000; Ministry of Health 2001d). Neighbourhood of residence, classified at the small area level according to degree of deprivation, is widely used in New Zealand as a marker of social circumstances (Salmond et al 1998). A steep and more-or-less monotonic gradient exists between life expectancy at birth (and other ages) and deprivation decile, for both genders and all major ethnic groups (Ministry of Health 2001c). Ethnic differences in life expectancy are well established, with the European ethnic group having the highest, Māori the lowest, and Pacific peoples an intermediate level (Ministry of Health 1999). Socioeconomic factors contribute to this ethnic disparity in survival chances, because the Māori, Pacific and European populations are unequally distributed across the deprivation deciles, with the former being over-represented and the latter under-represented in the more deprived deciles (Te Ropu Rangahau Hauora a Eru Pomare 2001).

The prevalence of tobacco smoking is also socially patterned. A steep and approximately linear gradient exists between smoking prevalence and deprivation decile, for both genders and at most ages (Crampton et al 2000). Smoking is also much more prevalent among Māori (both genders) and Pacific people (particularly males) than European or Asian New Zealanders – especially at younger ages (Ministry of Health 2001e).

Smoking is a major risk factor for chronic diseases, including cardiovascular and respiratory diseases and many types of cancer, which collectively account for a substantial proportion of premature mortality (Ministry of Health 1999). It is therefore of interest to quantify the magnitude of the ‘tobacco pathway’ linking socioeconomic position (or deprivation), gender and ethnicity to survival chances (Figure 1). That is, to what extent would the disparity in mortality between social groups (defined by deprivation level, ethnicity or gender) be narrowed were tobacco smoking to be eliminated?

Figure 1: A conceptual model linking deprivation, tobacco and life expectancy

![Figure 1](image-url)
To answer this question we use life expectancy (differentiated by deprivation, ethnicity and gender) as the indicator of mortality, in view of its intuitive appeal and independence from population age structure (Ministry of Health 2001c). Population-attributable risk methods are used to construct smoking-deleted life tables for each deprivation decile and ethnic group by gender (that is, life tables with deaths attributable to smoking removed). The analysis is future oriented – it estimates how much the current inequalities could be reduced in the future, were differences in smoking between the social strata to be eliminated (as opposed to estimating the current tobacco burden). For this reason, the analysis uses current smoking prevalence estimates instead of past (lagged) exposures to calculate the population-attributable risks. For the same reason, we prefer to calculate smoking-attributable fractions directly, instead of using the indirect method of Peto et al (1994).

In interpreting the results presented here it should be recognised that tobacco consumption and socioeconomic circumstances are by no means independent of each other. Rather, smoking prevalence is itself partly determined by the social context and cultural setting. What this analysis attempts to do is to quantify the magnitude of this ‘tobacco pathway’ linking deprivation and discrimination to (premature) death.

The cause-deleted life table model can also be used to provide an estimate of the burden of tobacco on the population as a whole. This measure of burden is also of policy interest.
Data and Methods

Data sources

Data on smoking prevalence was obtained from the 1996 Census of Population and Dwellings (Statistics New Zealand). The proportion of current smokers in each age, gender, ethnic and deprivation decile cell was extracted from this data set. Missing responses to the smoking items were excluded, so the prevalence estimates are conservative.

For the relative risk (RR) of mortality conditional on smoking, estimates from the American Cancer Society CPS II study, a large cohort study of over one million US adults conducted in the 1980s, were used (Thun and Heath 1997; Thun et al 2000). The RR estimates were differentiated by age and gender, but not by socioeconomic status or ethnicity. In our study the same sets of age–gender RR estimates were used and assumed to be invariant across deprivation and ethnic groups. This represents a conservative assumption, as the intensity (consumption of tobacco per smoker) and duration of smoking (and hence pack-years of cumulative exposure) are likely to be higher in more deprived neighbourhoods and among more marginalised ethnic groups.

The NZDep96 Index of Deprivation (Salmond et al 1998) was used as the deprivation measure. The index combines nine variables from the 1996 Census reflecting eight domains of deprivation (Table 1). Each variable was calculated as the proportion of people with the specified characteristic in each small area comprising one or more meshblocks. NZDep96 is the score on the first component of a principal component analysis of these nine adjusted proportions. The NZDep96 index is a decile scale created from these scores, with a value of 10 indicating that the meshblock is in the most deprived 10 percent of small areas in New Zealand, in turn corresponding almost exactly to the most deprived 10 percent of the total New Zealand population.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Domain of deprivation</th>
<th>Statistic (proportion of the small area population with characteristic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Communication</td>
<td>People with no access to a telephone</td>
</tr>
<tr>
<td>2</td>
<td>Income</td>
<td>People aged 18–59 receiving a means-tested benefit</td>
</tr>
<tr>
<td>3</td>
<td>Income</td>
<td>People living in households with equivalised income below an income threshold</td>
</tr>
<tr>
<td>4</td>
<td>Employment</td>
<td>People aged 15–59 unemployed</td>
</tr>
<tr>
<td>5</td>
<td>Transport</td>
<td>People with no access to a car</td>
</tr>
<tr>
<td>6</td>
<td>Support</td>
<td>People aged &lt; 60 living in a single-parent family</td>
</tr>
<tr>
<td>7</td>
<td>Qualifications</td>
<td>People aged 18–59 without any qualifications</td>
</tr>
<tr>
<td>8</td>
<td>Housing tenure</td>
<td>People not living in own home</td>
</tr>
<tr>
<td>9</td>
<td>Living space</td>
<td>People living in households below equivalised bedroom occupancy threshold</td>
</tr>
</tbody>
</table>

1 Meshblocks are the smallest geographic units defined by Statistics New Zealand, containing a median of 90 people.
Mortality data for 1995–97 by meshblock was obtained from Statistics New Zealand, stratified by age, gender, ethnicity and deprivation decile. For ethnic analyses, only 1996–97 data were used, to reduce numerator–denominator bias (Blakely et al, in preparation).

Methods

Life tables for the total population, and three ethnic groups – Europeans, Māori and Pacific peoples – were obtained from Statistics New Zealand. Abridged life tables for NZDep96 deciles were constructed for the period 1995–97 using standard demographic methods (SNZ 1999).

Due to small population bases, Māori and Pacific life tables could be constructed only for aggregates of deprivation deciles: deciles 1–7, 8–9 and 10 for Māori; deciles 1–8 and 9–10 for Pacific peoples. Life tables for Europeans were re-calculated using the same decile groups to make direct ethnic comparisons possible.

Smoking-deleted life tables were constructed for each deprivation and ethnicity category. This was done by removing the fraction of mortality attributable to smoking from the central mortality rate within each age–sex cell. The attributable fractions for each subgroup were derived from the smoking prevalence and RR data using the formula (Rothman and Greenland 1998):

$$AF = p(RR-1) / [p(RR-1) + 1]$$

where p is the prevalence of smoking and RR the relative risk of (all-cause) mortality conditional on smoking (age, gender, ethnic and deprivation decile indexes suppressed).

The set of smoking-deleted central mortality rates were smoothed using techniques conventionally employed in life table construction, and the smoking-deleted life tables calculated in the usual way.

Note that the indirect method for calculating smoking-attributable fractions developed by Peto et al (1994), which attempts to compensate for the long time lag between exposure to tobacco smoke and some health outcomes (such as lung cancer), is not used here. However, comparisons undertaken both internationally (Valkonen and Poppel 1997) and in New Zealand (Ministry of Health 1999a) find little difference in practice between estimates obtained using the indirect and the direct methods.

Analyses

Having constructed smoking-deleted life tables, the impact of smoking within each population subgroup was estimated by comparing each smoking-deleted life expectancy with its corresponding ordinary life expectancy at birth and other selected ages. This analysis was done separately for the deprivation-specific and ethnic-specific life tables (by gender).

Analysis of the three-way impact of smoking, deprivation and ethnicity on life expectancy was based on comparison of deprivation-specific or ethnic-specific ordinary and smoking-deleted life expectancies, with the European decile 1 smoking-deleted life expectancy (male or female as appropriate) as the ‘reference’ (Figure 2).
Selection of the life expectancy ‘reference’

The choice of an external reference for comparative analysis runs the risk of being interpreted as favouring a ‘deficit’ model of ethnic or socioeconomic disparity. A ‘development’ or rights-based model of disparity would be more consistent with the use of an internal rather than an external reference. An external reference is used here to allow a direct comparison of all three ethnic groups. This is appropriate for the purposes of this study, and is also compatible with the Crown’s obligations to monitor Māori and non-Māori comparative health status under the Treaty of Waitangi.

The European decile 1 smoking-deleted life expectancy (at birth or any other selected age, for males or females as appropriate), which is the highest of any subgroup, is used as the reference in order to provide a realistically achievable standard for the whole population. Its selection does not imply that decile 1 European non-smokers cannot further improve their longevity (that is, it is not intended to represent some ideal or absolute standard of health). However, the fact that one subgroup of the population has already achieved this level of survivorship implies that all remaining subgroups can also realistically aspire to it. The reference life expectancies for both genders for selected ages are shown later in the document (Table 4).
Inhaling Inequality: Tobacco’s contribution to health inequality in New Zealand

Results

Deprivation, ethnicity and life expectancy

The three major ethnic groups included in this study were unevenly distributed across deprivation deciles, with Māori and Pacific peoples being over-represented and Europeans under-represented in the more deprived deciles (Figure 3). In 1996 75 percent of all Māori and 83 percent of all Pacific people were more deprived than the national average (resided in deciles 6–10). Indeed, the distributions for these two ethnic groups tended to be concentrated at the most deprived deciles: over a quarter of Māori and over one-third of Pacific people lived in decile 10 areas, compared to less than 5 percent of Europeans.

Figure 3: Percentage distribution of 1996 Census population, by NZDep96 deprivation decile and ethnic group

Life expectancy shows a steep gradient with deprivation (Figure 4). In 1995–97 the difference in life expectancy at birth between deprivation decile 1 and decile 10 was 9.2 years for males and 6.7 years for females. These differentials are similar in magnitude to those between Māori and European ethnic groups (Table 2) – 8.4 years for males and 9.3 years for females in 1996–97 (Ministry of Health 1999a). The Pacific–European disparity is relatively smaller, at 5.8 years for males and 5.3 years for females.
Figure 4: Life expectancy at birth, by NZDep96 deprivation decile and gender, 1995–97

![Life expectancy graph](image)

Source of base data: Statistics New Zealand

Table 2: Life expectancy, by ethnic group, selected ages, 1995–97

<table>
<thead>
<tr>
<th>Age</th>
<th>Male</th>
<th></th>
<th></th>
<th>Female</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>European</td>
<td>Māori</td>
<td>Pacific</td>
<td>European</td>
<td>Māori</td>
<td>Pacific</td>
</tr>
<tr>
<td>0</td>
<td>75.6</td>
<td>67.2</td>
<td>69.8</td>
<td>80.9</td>
<td>71.6</td>
<td>75.6</td>
</tr>
<tr>
<td>15</td>
<td>61.3</td>
<td>53.4</td>
<td>55.8</td>
<td>66.5</td>
<td>57.7</td>
<td>61.3</td>
</tr>
<tr>
<td>45</td>
<td>33.0</td>
<td>26.2</td>
<td>28.0</td>
<td>37.4</td>
<td>29.4</td>
<td>32.8</td>
</tr>
<tr>
<td>65</td>
<td>15.9</td>
<td>12.2</td>
<td>13.4</td>
<td>19.6</td>
<td>14.5</td>
<td>16.6</td>
</tr>
</tbody>
</table>

Source of base data: Statistics New Zealand

Deprivation gradients in life expectancy vary considerably between ethnic groups (Figure 5). Because of the small Māori population base, life tables by deprivation for Māori could only be calculated for deciles 1–7 combined, deciles 8 and 9 combined and decile 10 as stand-alone. Life tables for Europeans were re-calculated using the same decile groupings (1–7, 8–9 and 10) to enable comparison between these two ethnic groups. The contrasting distributions of ethnic populations across deprivation deciles (as noted in Figure 3) mean that the population-weighted mid-points of deciles 1–7 differ between the two ethnic groups, at around 4.8 for Māori and 3.8 for Europeans. A smaller difference in weighted mid-points was found for the decile 8–9 aggregate as well. Life expectancy at birth for Pacific peoples had to be more highly aggregated in view of the even smaller and more skewed population distribution of this ethnic group. Results for Pacific peoples for deciles 1–8 combined and deciles 9 and 10 combined are included in Figure 5 for comparison.
While having the lowest life expectancy at birth compared to their European and Pacific counterparts, Māori also display the steepest gradient in life expectancy with deprivation. However, this may be at least partly an artefact of differential numerator–denominator bias across the deprivation deciles (Blakely et al, in preparation).

**Pattern of smoking across sociodemographic groups**

Smoking was also highly socially patterned, being more prevalent in more deprived areas (Figure 6). With the possible exception of Pacific females, the prevalence of smoking increased steadily over the deprivation scale and tended to accelerate in the most deprived deciles. For the population as a whole, the proportion of people who were current regular smokers increased from less than 15 percent in decile 1 to 38 percent in decile 10.

The prevalence of smoking among Māori was significantly higher compared to that in other ethnic groups. At the 1996 Census over 40 percent of all Māori aged 15 and above identified themselves as current smokers, a level double that for Europeans. The higher prevalence of smoking among Māori females compared to Māori males is particularly noteworthy (Figure 6).
Figure 6: Age-standardised proportion of current smokers, by NZDep96 deprivation decile, ethnic group and gender, 1996 Census

Source of base data: Statistics New Zealand
Note: age-standardised to Segi’s world population.
The high prevalence of smoking among Māori and, to a lesser extent, Pacific males may largely be explained by the interaction between smoking and two sociodemographic factors: concentration of these populations in more deprived areas (which are associated with higher smoking prevalence), and their younger population age structure (smoking prevalence peaks at young adult ages). In contrast, Pacific females demonstrated a relatively smaller excess prevalence of smoking in relation to European females, and in fact a lower prevalence among those living in the more deprived areas. Whether this reflects cultural norms, and may attenuate with further acculturation, is unclear at present.

**Smoking and life expectancy**

To calculate the fraction of mortality attributable to tobacco smoking, we used the direct method, based on current smoking prevalence (from the 1996 Census) and estimates of the relative risk (RR) of all-cause mortality of current smokers (from a large US cohort study, the CPSII study). The same age- and gender-specific RR schedule was used for all ethnic and deprivation groups, smoothed by cubic spline interpolation (Figure 7).

The RR for current smokers increases rapidly at young adult and middle ages. For male smokers the RR peaked in the 50–59 age group, where the risk of mortality is nearly three times higher than for non-smokers. RRs for female smokers are lower than those of male smokers, and peaked at slightly older ages, at over twice the level of that for female non-smokers. The decline in RR at older ages reflects a mortality selection effect for smokers, together with rising competing mortality from other (non-tobacco-related) causes.

The RR estimates used here are conservative in that they are based on comparison with never smokers, some of whom were in fact exposed to second-hand smoke. The estimates are particularly conservative for females, as they are based on historically lesser intensities and shorter durations of female than male smoking – trends that may not continue in the future.
Source of base data: modified from CPS II

Smoking-deleted life tables were constructed using attributable-mortality fractions calculated from these prevalence and RR data. Comparing the smoking-deleted with the ordinary life tables for the total New Zealand population during 1995–97, an additional 1.8 years for males and 0.8 years for females would be added to life expectancy at birth were deaths caused by smoking to be eliminated (after allowing sufficient time for the residual elevated risk of ex-smokers in the population to return to baseline). That is, life expectancy at birth would increase from 74.4 years to 76.2 years for males and from 79.6 years to 80.4 years for females (Table 3), corresponding to increases of 2.4 percent and 1.0 percent in life expectancy respectively.

Table 3: Smoking-deleted life expectancy at selected ages, by gender, total population, 1995–97

<table>
<thead>
<tr>
<th>Age</th>
<th>Male</th>
<th>Tobacco burden</th>
<th>Female</th>
<th>Tobacco burden</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LE</td>
<td>LE (smoking-deleted)</td>
<td>(years)</td>
<td>(% of LE)</td>
</tr>
<tr>
<td>0</td>
<td>74.4</td>
<td>76.2</td>
<td>1.8</td>
<td>2.4</td>
</tr>
<tr>
<td>15</td>
<td>60.2</td>
<td>62.0</td>
<td>1.8</td>
<td>3.0</td>
</tr>
<tr>
<td>45</td>
<td>32.2</td>
<td>33.8</td>
<td>1.6</td>
<td>5.0</td>
</tr>
<tr>
<td>65</td>
<td>15.6</td>
<td>16.4</td>
<td>0.8</td>
<td>5.1</td>
</tr>
</tbody>
</table>

* Equivalent to the contribution of the tobacco pathway to the total health loss.

LE = life expectancy
In our model, the impact of smoking on survival was assumed to start at adult ages and any fatal effects of second-hand smoking were not explicitly accounted for, especially among children. It is therefore useful to also look at the impact of smoking on life expectancy at other selected adult ages (Table 3). As expected given our model, the entire life expectancy gain from eliminating smoking is realised at ages 15 and above. Indeed, almost all the gain is realised after age 45. The increase in life expectancy at age 65 accounts for approximately half of the total gain from smoking elimination, with life expectancy at that age increasing from the current level of 15.6 years for males and 19.0 years for females, to 16.4 years and 19.4 years respectively.

**Smoking, deprivation and life expectancy**

To analyse the contribution of both tobacco-related and non-tobacco-related pathways to socioeconomic and ethnic disparities in life expectancy, it was necessary to select a reference against which the life expectancy (at any age) of each subgroup could be compared. The reference selected was the life expectancy (for any age and gender) of the subgroup with the least exposure to tobacco and to deprivation – that is, the European decile 1 smoking-deleted life expectancy (Table 4).

<table>
<thead>
<tr>
<th>Age</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>79.4</td>
<td>82.7</td>
</tr>
<tr>
<td>15</td>
<td>64.8</td>
<td>68.1</td>
</tr>
<tr>
<td>45</td>
<td>36.1</td>
<td>38.8</td>
</tr>
<tr>
<td>65</td>
<td>17.8</td>
<td>20.3</td>
</tr>
</tbody>
</table>

*Note: reference = European decile 1 smoking-deleted life expectancy.*

The total health loss (or more accurately, survival loss) sustained by any population subgroup could then be calculated as the difference between the reference life expectancy and that of the subgroup (at any age and for either gender). Furthermore, the contribution of tobacco consumption to this total health loss is the difference between the smoking-deleted and ordinary life expectancies for that population group (at any age and for either gender). Finally, the non-tobacco contribution to the total health loss is then approximated by the difference between the total health loss and the tobacco contribution (Figure 2).

Since the majority of gains in life expectancy at birth from eliminating smoking occur at ages 45 and above (Table 3), only the estimates for smoking-deleted life expectancy at birth and at ages 45 and 65 for each deprivation decile are shown in Tables 5a–5c, and summarised in Figure 8. To facilitate comparisons across deciles, a regression curve is fitted to each of the four life expectancy series in Figure 8.
Table 5a: Impact of tobacco consumption on the deprivation gradient in life expectancy at birth, total population, 1995–97

<table>
<thead>
<tr>
<th>NZDep96 decile</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LEₐ</td>
<td>78.1</td>
<td>77.6</td>
<td>76.5</td>
<td>75.7</td>
<td>75.1</td>
<td>74.5</td>
<td>73.9</td>
<td>72.7</td>
<td>71.4</td>
<td>68.9</td>
<td>74.4</td>
</tr>
<tr>
<td>LEₐ (smoking-del)</td>
<td>79.0</td>
<td>78.7</td>
<td>77.8</td>
<td>77.2</td>
<td>76.9</td>
<td>76.5</td>
<td>76.1</td>
<td>75.3</td>
<td>74.3</td>
<td>72.6</td>
<td>76.2</td>
</tr>
<tr>
<td>Total health loss (TBL)</td>
<td>1.3</td>
<td>1.8</td>
<td>2.9</td>
<td>3.7</td>
<td>4.2</td>
<td>4.8</td>
<td>5.5</td>
<td>6.6</td>
<td>8.0</td>
<td>10.4</td>
<td>5.0</td>
</tr>
<tr>
<td>Tobacco contribution (Tob)</td>
<td>1.0</td>
<td>1.1</td>
<td>1.3</td>
<td>1.5</td>
<td>1.7</td>
<td>2.0</td>
<td>2.3</td>
<td>2.5</td>
<td>2.9</td>
<td>3.7</td>
<td>1.8</td>
</tr>
<tr>
<td>Non-tobacco contribution (Nontob)</td>
<td>0.3</td>
<td>0.7</td>
<td>1.6</td>
<td>2.2</td>
<td>2.5</td>
<td>2.9</td>
<td>3.2</td>
<td>4.1</td>
<td>5.1</td>
<td>6.7</td>
<td>3.2</td>
</tr>
<tr>
<td>Tob/THL (%)</td>
<td>36</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female**</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>LEₐ</td>
<td>82.1</td>
<td>81.5</td>
<td>81.2</td>
<td>81.1</td>
<td>80.6</td>
<td>79.9</td>
<td>79.6</td>
<td>78.5</td>
<td>77.8</td>
<td>75.4</td>
<td>79.6</td>
</tr>
<tr>
<td>LEₐ (smoking-del)</td>
<td>82.5</td>
<td>81.9</td>
<td>81.8</td>
<td>81.7</td>
<td>81.2</td>
<td>80.7</td>
<td>80.5</td>
<td>79.5</td>
<td>79.0</td>
<td>77.1</td>
<td>80.4</td>
</tr>
<tr>
<td>Total health loss (TBL)</td>
<td>0.6</td>
<td>1.2</td>
<td>1.5</td>
<td>1.6</td>
<td>2.1</td>
<td>2.8</td>
<td>3.1</td>
<td>4.2</td>
<td>4.9</td>
<td>7.3</td>
<td>3.1</td>
</tr>
<tr>
<td>Tobacco contribution (Tob)</td>
<td>0.4</td>
<td>0.4</td>
<td>0.5</td>
<td>0.6</td>
<td>0.6</td>
<td>0.7</td>
<td>0.9</td>
<td>1.0</td>
<td>1.2</td>
<td>1.7</td>
<td>0.8</td>
</tr>
<tr>
<td>Non-tobacco contribution (Nontob)</td>
<td>0.2</td>
<td>0.8</td>
<td>1.0</td>
<td>1.0</td>
<td>1.5</td>
<td>2.0</td>
<td>2.2</td>
<td>3.2</td>
<td>3.7</td>
<td>5.6</td>
<td>2.3</td>
</tr>
<tr>
<td>Tob/THL (%)</td>
<td>27</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Reference = 79.4  
** Reference = 82.7

Table 5a shows that males in deprivation decile 10 lose over nine years of life expectancy at birth more than do their counterparts in decile 1; for females, the loss is slightly smaller at under seven years. In each decile, males lose about twice as many life years to tobacco as females do. Across the deprivation range the total health loss increases dramatically from least to most deprived areas – as does the tobacco loss.

The contribution of tobacco to the total health loss appears to decrease in relative terms (although increasing in absolute years) from least to most deprived deciles, but the relative decrease merely reflects the selection of decile 1 as the standard. With respect to relative effects, only the tobacco contribution to the health loss sustained by the (sub)group as a whole, pooling over the deprivation deciles, is meaningful (36 percent for males and 27 percent for females when measured from birth).

Table 5b: Impact of tobacco consumption on the deprivation gradient in life expectancy at age 45, total population, 1995–97

<table>
<thead>
<tr>
<th>NZDep96 decile</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LEₐ</td>
<td>34.9</td>
<td>34.5</td>
<td>33.9</td>
<td>33.0</td>
<td>32.7</td>
<td>32.2</td>
<td>31.5</td>
<td>30.8</td>
<td>30.0</td>
<td>28.3</td>
<td>32.2</td>
</tr>
<tr>
<td>LEₐ (smoking-del)</td>
<td>35.8</td>
<td>35.5</td>
<td>35.2</td>
<td>34.4</td>
<td>34.2</td>
<td>34.0</td>
<td>33.5</td>
<td>33.1</td>
<td>32.6</td>
<td>31.6</td>
<td>33.8</td>
</tr>
<tr>
<td>Total health loss (TBL)</td>
<td>1.2</td>
<td>1.7</td>
<td>2.2</td>
<td>3.1</td>
<td>3.5</td>
<td>3.9</td>
<td>4.6</td>
<td>5.3</td>
<td>6.2</td>
<td>7.9</td>
<td>3.9</td>
</tr>
<tr>
<td>Tobacco contribution (Tob)</td>
<td>0.9</td>
<td>1.0</td>
<td>1.2</td>
<td>1.4</td>
<td>1.6</td>
<td>1.8</td>
<td>2.1</td>
<td>2.3</td>
<td>2.7</td>
<td>3.4</td>
<td>1.6</td>
</tr>
</tbody>
</table>
Table 5b shows that much of the total health loss sustained by all sociodemographic subgroups occurs at ages 45 and above. This is even more so for tobacco than non-tobacco losses.

Table 5c: Impact of tobacco consumption on the deprivation gradient in life expectancy at age 65, total population, 1995–97

Table 5c shows that, overall, about half the total health loss occurs in old age. The proportion is lower for more deprived areas, and higher for socioeconomically advantaged neighbourhoods. Tobacco loss (depressing life expectancy at birth by 1.8 years for males and 0.8 years for females
when averaged across the whole population) contributes about a third to the total loss whether the whole life cycle, or just ages 45 onwards or 65 onwards, are considered.

The impact of tobacco smoking on survival was greater in absolute terms in the more deprived areas, driven by their higher prevalence of smoking and higher overall risk of mortality. By eliminating smoking, life expectancy at birth for decile 10 would increase by 3.7 years for males and 1.7 years for females. In contrast, the corresponding increase for decile 1 is only 1.0 years for males and 0.4 years for females. The increasing impact of smoking on survival across the deprivation scale is reflected by the widening gap between the fitted ordinary life expectancy and smoking-deleted life expectancy curves for each gender (Figure 8).

Also shown in Figure 8 is the considerably steeper deprivation gradient in life expectancy among the more deprived deciles. Eliminating smoking would go some way to alleviating this inequality (for example, inspection of Figure 8 shows that eliminating tobacco would have a particularly beneficial effect on the life expectancy of males in deciles 7–10).

**Figure 8:** Life expectancy at birth and smoking-deleted life expectancy at birth, with fitted gradient curves, by NZDep96 deprivation decile and gender, total population, 1995–97

Estimates of the total health loss (THL) sustained by the different deprivation deciles, including the impact of tobacco and the contribution of the non-tobacco pathway, are also summarised in Tables 5a–5c and illustrated graphically in Figure 9.
Figure 9: Estimated impact of tobacco consumption on deprivation gradient in life expectancy, 1995–97

Reference = 79.4 years (European male decile 1 smoking-deleted LE)

Reference = 82.7 years (European female decile 1 smoking-deleted LE)
Smoking, deprivation, gender and life expectancy

Also of interest are changes in the gender gap in survival chances with tobacco elimination. The gender gap in life expectancy at birth as shown in Figure 8 widens appreciably over the deprivation scale, but narrows once smoking-related deaths are removed. Gender differentials in life expectancy at birth and smoking-deleted life expectancy at birth for each decile are summarised in Table 6.

Table 6: Gender differentials in life expectancy at birth and smoking-deleted life expectancy at birth, by NZDep96 deprivation decile, total population, 1995–97

<table>
<thead>
<tr>
<th>NZDep96 decile</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender difference in LE₀</td>
<td>4.0</td>
<td>4.0</td>
<td>4.8</td>
<td>5.5</td>
<td>5.4</td>
<td>5.4</td>
<td>5.8</td>
<td>5.7</td>
<td>6.4</td>
<td>6.5</td>
<td>5.2</td>
</tr>
<tr>
<td>Gender difference in smoking-del LE₀</td>
<td>3.4</td>
<td>3.2</td>
<td>4.0</td>
<td>4.5</td>
<td>4.3</td>
<td>4.2</td>
<td>4.4</td>
<td>4.2</td>
<td>4.7</td>
<td>4.5</td>
<td>4.3</td>
</tr>
<tr>
<td>% reduction in gender differential</td>
<td>15</td>
<td>18</td>
<td>17</td>
<td>17</td>
<td>20</td>
<td>22</td>
<td>24</td>
<td>26</td>
<td>27</td>
<td>31</td>
<td>18</td>
</tr>
</tbody>
</table>

LE₀ = life expectancy at birth

On average, females outlive males by 5.2 years. The female advantage in life expectancy at birth ranges from 4.0 years in the least deprived areas to around 6.5 years in the most deprived areas. By eliminating tobacco deaths, however, the female advantage would reduce by 18 percent, to 4.3 years (pooling deciles).

Gender differentials in smoking-deleted life expectancy in the least deprived areas would reduce to just over three years, but average approximately 4.5 years in deciles 4–10.

Smoking, deprivation, ethnicity and life expectancy

The estimates for total health loss (THL) at birth and at ages 45 and 65 for each ethnic group (by gender) and the corresponding tobacco and non-tobacco impact estimates (based on comparison with the European decile 1 smoking-deleted life expectancy ‘reference’) are shown in Table 7 and summarised graphically in Figure 10.
Table 7: Impact of smoking on the ethnic disparity in life expectancy at selected ages, 1995–97

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ref.</td>
<td>European</td>
</tr>
<tr>
<td>At birth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LE₀</td>
<td>–</td>
<td>75.6</td>
</tr>
<tr>
<td>LE₀ (smoking-del)</td>
<td>79.4</td>
<td>77.2</td>
</tr>
<tr>
<td>Total health lost (THL)</td>
<td>–</td>
<td>3.8</td>
</tr>
<tr>
<td>Tobacco contribution (Tob)</td>
<td>–</td>
<td>1.6</td>
</tr>
<tr>
<td>Non-tobacco contribution (Nontob)</td>
<td>–</td>
<td>2.2</td>
</tr>
<tr>
<td>Tob/THL (%)</td>
<td>–</td>
<td>42</td>
</tr>
<tr>
<td>At age 45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LE₀</td>
<td>–</td>
<td>33.0</td>
</tr>
<tr>
<td>LE₀ (smoking-del)</td>
<td>36.1</td>
<td>34.5</td>
</tr>
<tr>
<td>Total health lost (THL)</td>
<td>–</td>
<td>3.1</td>
</tr>
<tr>
<td>Tobacco contribution (Tob)</td>
<td>–</td>
<td>1.4</td>
</tr>
<tr>
<td>Non-tobacco contribution (Nontob)</td>
<td>–</td>
<td>1.7</td>
</tr>
<tr>
<td>Tob/THL (%)</td>
<td>–</td>
<td>46</td>
</tr>
<tr>
<td>At age 65</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LE₀</td>
<td>–</td>
<td>15.9</td>
</tr>
<tr>
<td>LE₀ (smoking-del)</td>
<td>17.8</td>
<td>16.7</td>
</tr>
<tr>
<td>Total health lost (THL)</td>
<td>–</td>
<td>1.9</td>
</tr>
<tr>
<td>Tobacco contribution (Tob)</td>
<td>–</td>
<td>0.8</td>
</tr>
<tr>
<td>Non-tobacco contribution (Nontob)</td>
<td>–</td>
<td>1.1</td>
</tr>
<tr>
<td>Tob/THL (%)</td>
<td>–</td>
<td>42</td>
</tr>
</tbody>
</table>

Reference = European decile 1 smoking-deleted life expectancy
Figure 10: Impact of tobacco consumption on ethnic disparity in life expectancy at birth, 1995–97

Reference = 79.4 years (European male decile 1 smoking-deleted LE)

Reference = 82.7 years (European female decile 1 smoking-deleted LE)
For Europeans, the total health loss (THL) measured at birth (for the whole population group) was 3.8 years for males and 1.8 years for females. The tobacco contribution to this THL was relatively small in absolute terms (1.6 years for males and 0.7 years for females), but represents around 40 percent of THL for both genders.

While for European males half of the THL from birth occurs in old age (1.9 of 3.8 years), less than 40 percent of the European female THL (0.7 of 1.8 years) occurs at ages 65 and above, reflecting the flattening of the deprivation gradient in mortality among European females at older ages.

By contrast, if all Māori enjoyed the mortality experience of European decile 1 non-smokers, over 12 years and 11 years would be added to life expectancy at birth for Māori males and females respectively. These potential gains represent more than 15 percent improvement from current (1996–97) life expectancy. The majority of the health gain for Māori would come from non-tobacco-related pathways, with tobacco contributing 29 percent to male and 22 percent to female THL. That is, the contribution of tobacco to Māori health loss is greater in absolute, but smaller in relative, terms than is its contribution to the health loss sustained by Europeans.

For Pacific peoples the THL at birth was estimated to be 9.5 years for males and 7.1 years for females. The tobacco share of this health loss was estimated to be 37 percent for males, similar to the other ethnic groups, but only 13 percent for females – significantly lower than for the other ethnic groups. The low tobacco contribution to Pacific female health loss, especially at older ages, reflects the contrasting patterns of high deprivation but relatively lower prevalence of tobacco smoking experienced by this group (Figures 3 and 6).

Ethnic variation in the contribution of tobacco to the gender gap in life expectancy is summarised in Table 8. By eliminating tobacco consumption, the gender differential in life expectancy for Europeans would reduce by 17 percent, from 5.3 years to 4.4 years. The corresponding reduction for Māori would be 24 percent, from 4.4 years to 3.3 years. The largest reduction, however, would occur among Pacific peoples because of the relatively low tobacco contribution to THL among Pacific females: the gender difference in life expectancy among Pacific peoples would almost halve, from 5.8 years to 3.2 years.

Table 8: Gender differentials in life expectancy at birth and smoking-deleted life expectancy at birth, by ethnicity, 1995–97

<table>
<thead>
<tr>
<th></th>
<th>European</th>
<th>Māori</th>
<th>Pacific peoples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender difference in LE0</td>
<td>5.3</td>
<td>4.4</td>
<td>5.8</td>
</tr>
<tr>
<td>Gender difference in smoking-del LE0</td>
<td>4.4</td>
<td>3.3</td>
<td>3.2</td>
</tr>
<tr>
<td>% reduction in gender differentials</td>
<td>17</td>
<td>24</td>
<td>46</td>
</tr>
</tbody>
</table>
Discussion

We have used data on the prevalence and excess mortality risk of smoking to construct smoking-deleted life tables for both genders, three major ethnic groups, and (aggregated) deprivation deciles in New Zealand. Comparison of the corresponding cause-deleted and ordinary life tables allows forecasting of how social inequalities in life expectancy in New Zealand would change were smoking to be eliminated from all subgroups of the population.

Limitations of the model

In interpreting these estimates, the limitations of our data and methods need to be understood. The smoking prevalence data is derived from the 1996 New Zealand Census and is therefore quite robust (although self-reported smoking generally slightly underestimates true prevalence [Carabello et al 2001]), but some misclassification is likely due to missing responses (about 8 percent of the total) and social desirability bias. The rates used here are therefore likely to be underestimates, and the estimates of tobacco burden are therefore conservative. If desired, sensitivity analysis could be done to quantify the uncertainty introduced into the estimates through misclassification of smoking status in the Census.

The RR estimates for all-cause mortality conditional on current smoking were derived from a US study carried out in the 1980s, and – even if internally valid – may not fully apply to the New Zealand population in the early part of the present century, whose duration and intensity of smoking, and exposure to other risk factors, may be different. In particular, generalisability of the RR estimates to the Māori and Pacific ethnic groups may be less satisfactory. Similarly, using the same RR estimates for all deprivation groups may not fully capture the differences between them. Again, sensitivity analysis around the RR estimates may help to quantify the uncertainty introduced through this variable. The estimates used here are considered to be conservative, and in particular are likely to underestimate the impact of smoking on deprived and marginalised social groups.

It is also likely that the impact of tobacco consumption on females (of all ethnic groups) has been underestimated in this study. This is because the prevalence and RR estimates used for females reflect historically lower rates, durations and intensities of smoking than experienced by males – differences that are rapidly disappearing (or may have already disappeared). So it would be reasonable to predict that the gender difference estimated here in the burden of tobacco, and in the contribution of tobacco to social inequalities in life expectancy, will diminish in the future.

While the mortality data is robust, the assignment of ethnicity on death certificates did not fully match that on the Census questionnaire in 1996, suggesting that Māori and Pacific mortality rates – when measured using the total ethnic group concept – may still be underestimated (Blakely et al in preparation).

Limitations of the NZDep96 deprivation index include failure to capture all relevant dimensions of socioeconomic status, failure to fully remove variation within social categories included in the index, restriction to a single point of time (as opposed to measuring cumulative disadvantage across the lifecycle), and location at the small area rather than the individual level (Ministry of Health 2001a).
Finally, our model makes two simplifying assumptions with respect to smoking itself. Firstly, we exclude the health impact of second-hand smoke, which means we may have underestimated the impact of smoking on the survivorship gradient and disparities by 8 percent or more (Woodward and Laugesen 2001). Secondly, our model neglects the lag period between quitting smoking and reverting to baseline risk of dying (about five to ten years [Doll et al 1994]); that is, we do not explicitly include ex-smokers in our model. The rationale for this is that we are more concerned with modelling the medium to long term rather than the short term. However, if desired, ex-smoking could be included in the model as the prevalence of ex-smoking is known from the 1996 Census, and estimates of the RR for ex-smokers are also available from the CPSII study (and other research).

Also, our model is limited to the use of aggregated data and static modelling. The New Zealand Census Mortality Study may allow some of the research questions considered here to be analysed at the individual level, by linking individual death certificates to Census questionnaires (using anonymous probabilistic matching) (Blakely in preparation). Useful information may also be extractable from a dynamic model of the smoking population constructed by Clements (in preparation).

Despite these limitations, the cause-deleted life table model reported here does provide a reasonable estimate of the impact of smoking on both socioeconomic gradients and ethnic and gender disparities in mortality.

**Summary of key findings**

For the total population, the elimination of tobacco would increase life expectancy at birth by 1.8 years for males and 0.8 years for females, once the residual risk of ex-smokers washed out. This corresponds to a 2.4 percent increase in life expectancy at birth for males and a 1.0 percent increase for females.

The impact of tobacco elimination increases progressively with degree of deprivation, from 1.2 percent of life expectancy (males) and 0.5 percent (females) in decile 1, to 5.3 percent and 2.2 percent respectively in decile 10.

By comparison with the European decile 1 smoking-deleted reference life table, 36 percent (males) and 27 percent (females) of the observed loss in life expectancy at birth would be avoided were smoking to be eliminated from the population. That is, the ‘tobacco pathway’ mediates about one-third of the socioeconomic gradient in mortality (genders and ethnic groups pooled).

Using the same reference value, the elimination of tobacco would increase European, Māori and Pacific life expectancy at birth by 1.6 years (2.1 percent), 3.5 years (5.2 percent) and 3.6 years (5.1 percent) for males and 0.7 years (0.8 percent), 2.4 years (3.3 percent) and 0.9 years (1.2 percent) for females respectively (deprivation deciles pooled).

Approximately 42 percent of the health loss sustained by European males and 38 percent of that sustained by European females across all deprivation deciles (using European decile 1 smoking-deleted life expectancy as the reference) would be regained were tobacco consumption to be eliminated from this ethnic group. The corresponding proportions for Māori are 29 percent and 22 percent respectively. Tobacco smoking makes a similar contribution to the total health loss of Pacific males (37 percent), but a smaller contribution to that experienced by Pacific females (13 percent), reflecting the currently lower prevalence of smoking among the latter group.
Because of the gender variation in relative risk of mortality conditional on smoking (and to a lesser extent, the current gender differential in smoking prevalence), the elimination of tobacco consumption would reduce the gender gap in life expectancy at birth for the total population by 18 percent, from 5.2 years to 4.3 years. The reduction in the gender differential would be greater in more deprived areas and for Māori, and particularly for Pacific peoples.

The burden of tobacco

As well as quantifying the contribution of tobacco to the deprivation gradient and ethnic or gender gap in life expectancy, our model also provides a novel way of measuring the tobacco burden. Previously, the burden of tobacco on New Zealanders has been quantified in terms of the number of deaths attributable to tobacco (using the Peto et al method) (Ministry of Health 1999b), or in terms of disability-adjusted life years (Ministry of Health 2001a). Cause-deleted life table modelling provides another view of this burden.

Cause-deleted life tables for major groups of causes (related diseases or injuries) have previously been calculated for 1995–97 (Ministry of Health 1999). Key results from these life tables are compared with those reported here for smoking (Table 9).

Table 9: Changes in life expectancy at birth by eliminating deaths due to different causes, by gender, total population, 1995–97

<table>
<thead>
<tr>
<th>Cause-of-death eliminated</th>
<th>Male</th>
<th></th>
<th>Female</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Increase in LE</td>
<td>% increase</td>
<td>Increase in LE</td>
<td>% increase</td>
</tr>
<tr>
<td>Smoking</td>
<td>1.8</td>
<td>2.4</td>
<td>0.8</td>
<td>1.0</td>
</tr>
<tr>
<td>Infectious diseases</td>
<td>0.5</td>
<td>0.6</td>
<td>0.6</td>
<td>0.8</td>
</tr>
<tr>
<td>Maternal and infant conditions</td>
<td>0.6</td>
<td>0.8</td>
<td>0.6</td>
<td>0.7</td>
</tr>
<tr>
<td>Unintentional injury</td>
<td>1.1</td>
<td>1.4</td>
<td>0.5</td>
<td>0.6</td>
</tr>
<tr>
<td>Intentional injury</td>
<td>0.7</td>
<td>1.0</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>Neuropsychiatric conditions</td>
<td>0.4</td>
<td>0.5</td>
<td>0.5</td>
<td>0.6</td>
</tr>
<tr>
<td>Cancers</td>
<td>3.7</td>
<td>4.9</td>
<td>3.9</td>
<td>4.9</td>
</tr>
<tr>
<td>Cardiovascular diseases</td>
<td>4.4</td>
<td>6.0</td>
<td>4.0</td>
<td>5.0</td>
</tr>
<tr>
<td>Other chronic diseases</td>
<td>2.8</td>
<td>3.8</td>
<td>3.0</td>
<td>3.8</td>
</tr>
</tbody>
</table>

Source: Data on smoking elimination see above; data on all other causes see Ministry of Health 1999.

The estimates of increases in life expectancy from tobacco elimination are considered to be conservative, principally because the effects of second-hand smoke are not included in the model. Nevertheless, the impact on population health of eliminating tobacco consumption is estimated to be greater than that of infectious diseases and maternal and infant conditions combined, or similar to that of injuries (intentional and unintentional combined).

While the impact of tobacco elimination appears to be smaller than that of cancer or cardiovascular disease, smoking of course contributes to these outcomes. Such overlaps should be borne in mind when inspecting Table 9, which combines different levels of causation (in this case, diseases or injuries and risk factors) in the same table.
From a policy perspective, modifiability (responsiveness to intervention) and equity (distributional) considerations are also relevant, in addition to the size of the burdens attributable to different causes (Ministry of Health 2001b). Such considerations would further enhance the relative importance of tobacco as a cause of health loss.

**Population versus individual benefit**

How does our estimate of the tobacco burden (1.8 years loss in life expectancy at birth for males, 0.8 years for females) relate to the well-known estimate of Peto that smokers can expect to lose, on average, 14 years of life (Peto et al 1994)? Our model calculates the gain in life expectancy from eliminating smoking for the total population. At the individual level, this gain of course accrues specifically to smokers (about a quarter of the total population) – and is further restricted to the subset (about half\(^2\)) of smokers who in fact die prematurely from a smoking-related cause.

If instead of diffusing the gain across the total population, we consider only those who personally benefit (individuals who would otherwise have smoked and died prematurely as a result of their smoking), our estimate would be approximately eight times higher – about 14 years (1.8 x 4 x 2) for males and half this (0.8 x 4 x 2) for females, in reasonable agreement with that of Peto.

It may however be more realistic to consider the impact of tobacco consumption as falling on all smokers, whether they die of smoking-related or unrelated causes. In which case we estimate that the *average* smoker loses about 5.2 years of life (1.8 x 4 years for males and 0.8 x 4 years for females).

From a policy perspective, the gain in health for the population as a whole may be more salient, while from a health promotion perspective it may be more relevant to focus on the potential gain to individuals (for example, in encouraging and assisting smokers to quit).

**Conclusion**

The cause-deleted life expectancy model reported here enables monitoring of the absolute and relative magnitudes of the tobacco and non-tobacco pathways leading to health inequality (relative to any selected reference, such as the European decile 1 smoking-deleted life table used here). Such monitoring provides insight into the contribution of tobacco to inequalities in health outcomes, and may assist both in evaluating the success of the tobacco control programme, and in placing this programme in the broader context of social equity.

In summary, the model suggests that, overall, at least one-third of the deprivation gradient in life expectancy at birth or older ages, one-quarter of the corresponding ethnic disparity, and one-fifth of the corresponding gender gap, is accounted for by tobacco consumption, allowing for the conservative assumptions used in constructing the model.

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\(^2\) Since RR averages about 2 across adulthood, it follows that about half of persistent smokers who began smoking during adolescence or young adulthood will die from their smoking.
References


Inhaling Inequality: Tobacco’s contribution to health inequality in New Zealand


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