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Research Article

Application of the modified PGW method for determining the smoking-attributable fraction of deaths in New Zealand Maori, Pacific, and non-Maori non-Pacific populations

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Table of Contents

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Abstract

BACKGROUND
Preston, Glei, and Wilmoth recently proposed a new method for estimating smoking-attributable mortality in high-income countries, and an improvement to the method was proposed by Rostron. The method greatly simplifies estimation of smoking-attributable fractions, but additional testing has been recommended to validate the approach.

OBJECTIVE
We apply the Rostron (PGW-R) method to ethnic groups in New Zealand and compare the results with published estimates from other sources, with the purpose of determining their consistency and exploring possible reasons for any divergence.

METHODS
Four different sources were identified with ethnic-specific estimates of smoking-attributable mortality fractions (SAMF) for Maori, Pacific Island, and European/Other ethnic groups in New Zealand for periods between 1995 and 1999. These employed a variety of direct and indirect estimation techniques. The results were compared with PGW-R method estimates for the same period and ethnic groups.

RESULTS
Although the PGW-R method produced SAMF estimates that were within 5% of those derived using the Peto-Lopez method for the European/Other and total populations (in males and females), there were significant discrepancies between them in the Maori and Pacific SAMF estimates. Results using direct methods from a census linkage study were inconsistent with both the Peto-Lopez and the PGW-R method. Seven possible

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explanations for these discrepancies were considered and discussed, but none could fully account for the differences.

CONCLUSIONS
The results of this work raise questions not only about the validity of the PGW-R method, but also about the accuracy of the estimates derived from the Peto-Lopez and direct methods, at least in these populations. Further research should examine the applicability of the key assumptions of the PGW method. Other work to determine the effects of possible misclassification bias in the direct method estimates would also aid interpretation of these findings.

COMMENTS
Accurate methods for determining the population health impact of smoking are vital for policymakers to ensure that tobacco control is awarded the appropriate emphasis and resourcing.

1. Introduction

Preston, Glei, and Wilmoth (2010) recently proposed a new method for estimating smoking-attributable mortality in high-income countries and applied the method to the 21 high-income countries from which it was developed. The Preston, Glei, and Wilmoth (PGW) method has some similarity to the widely used Peto-Lopez method (Peto et al. 1992) in that it estimates a population's accumulated smoking harm from the excess lung cancer mortality in that population (the mortality above that which would be expected in a population that had never smoked). However, the PGW method has the important advantage of not relying on the assumption that relative risks for causes of death other than lung cancer can be applied to other countries. Instead, it determines the macro-level statistical association between excess lung cancer mortality and death rates from all other causes combined. In addition, the PGW method is simpler than the standard Peto-Lopez method because it does not require accurate cause of death coding other than for lung cancer. Since the precision of cause of death coding can vary by ethnicity for a number of reasons (Joshy et al. 2010) this could represent a significant practical advantage.

Following publication of the PGW method, Rostron (2010) proposed a modification to it (referred here as the PGW-R method) that improved its consistency with estimates of smoking-attributable mortality fractions (SAMF) derived from the Peto-Lopez method, particularly for females. It did this by introducing an age-year

interaction term in the negative binomial regression model. The PGW-R model can therefore be expressed as follows:

$$\ln M_0 = \beta_a X_a + \beta_t X_t + \beta_c X_c + \beta(t \times X_a) + \beta_{ct}(t \times X_c) + \beta_L M_L + \beta_{Lt}(M_L \times t) + \beta_{La}(M_L \times X_a)$$

where M_0 is the death rate from causes other than lung cancer by age, sex, year, and country; X_a is a set of dummy variables for each age group, X_t is a set of dummy variables for each year, X_c is a set of dummy variables for each country, (t x X_c) is an interaction term between year as a linear variable and country, M_L represents the lung cancer death rate, (M_L x t) is an interaction term between the lung cancer death rate and year as a linear variable, (M_L x X_a) is an interaction term between the lung cancer death rate and age group, and (t x X_a) is an interaction term between age group and year as a linear variable.

Rostron called for additional research to evaluate how well the PGW-R method performs for countries other than the United States, and how it compares with results from methods used to produce national estimates of SAMF in those countries. Also, the original PGW paper pointed out that the method may not work well in populations where factors other than smoking have a major impact on lung cancer mortality (Preston, Glei, and Wilmoth 2010).

In this paper we apply the PGW-R method to ethnic groups in New Zealand and compare the results with published estimates from other sources with the purpose of determining their consistency and exploring possible reasons for any divergence. Applying the PGW-R method to the examination of ethnic differences in smoking-attributable mortality offers a test of its validity and robustness beyond that achieved with international comparisons because it faces the potential complexities arising from differences in lung cancer incidence in life-time non-smokers, possible differences in the association between smoking and non-lung cancer mortality, and ethnic differences in data quality (although the latter in New Zealand is generally high).

The sources of SAMF estimates used here for comparison purposes were obtained using a variety of methods. Laugesen and Swinburn (2000) applied the Peto-Lopez method to the New Zealand population in 1996 using 1995 mortality rates to estimate SAMF in men and women aged 35-69. The Laugesen and Clements (1998) study of smoking-attributable mortality among Maori also used the Peto-Lopez method but applied this to the Maori population of 1989-1993. These two studies are jointly referred to as the Laugesen studies in this paper.

A New Zealand Ministry of Health publication (Ministry of Health 1999) applied the methods of English et al. (1995), using their same relative risks for smoking-related mortality along with ethnicity-specific estimates of exposure based on responses to the

smoking question in the 1996 census, to calculate SAMF for Maori, Pacific, and European/Other ethnicities.

In the Tobias and Cheung (2001) analysis smoking-deleted life tables for 1995-7 were created using current smoking prevalence from the 1996 population census by calculating age-sex specific SAMF based on the relative risk of all-cause mortality for current smokers compared with never-smokers derived from the Cancer Prevention Study II (CPS II) study. Their results are presented as the proportion of 'total health loss' attributable to smoking, where the total health loss is the difference in life expectancy between the population of interest and the smoking-deleted life expectancy of Europeans in the lowest deprivation decile (79.4 years at birth for males and 82.7 for females).

The fourth source of comparison estimates come from the New Zealand Census Mortality Study (Wilson, Blakely, and Tobias 2006), which is a large cohort of the New Zealand 1996 census population aged 45-74 years linked anonymously and probabilistically to three years of subsequent mortality data, weighted to adjust for linkage bias. In this study the estimates of SAMF were calculated directly from age-smoking standardised mortality rates within this cohort.

2. Methods

Lung cancer and total deaths by age, sex, and ethnicity were obtained from Statistics New Zealand's Mortality Database. Population numbers were obtained from the Wang (2011) projections based on Statistics New Zealand estimates. Abridged life tables for the period 1996-1999 were created for Maori, Pacific, and non-Maori non-Pacific New Zealand resident populations using the Chiang II method (Toson and Baker 2003). As in the second PGW study (Preston, Glei, and Wilmoth 2011), all-cause death counts and rates were taken from the Human Mortality Database (University of California Berkeley and Max Planck Institute for Demographic Research 2011), while cause-specific distributions of deaths were taken from the WHO World Mortality Database (World Health Organisation 2011).

In New Zealand ethnicity is self-declared and multiple ethnicities are recorded. When mutually exclusive categories are required, such as in regression analyses, data is often reported using 'prioritised ethnicity', which assigns a single ethnicity to individuals based on a ranking specified in the national ethnicity standards (Maori then Pacific then Asian then Other then European) (Ministry of Health 2004).

The PGW-R method was used for determining the SAMF. The method is described in detail in Rostron's paper (Rostron 2010). As with the original PGW method it involves firstly calculating the fraction of lung cancer deaths attributable to

smoking (A_L) in each sex-age-ethnicity stratum. This is done by subtracting stratum-specific lung cancer death rates among people who have never smoked (λ_L^N) from the stratum-specific lung cancer mortality rate found in the population of interest (M_L) and dividing the difference by this same rate.

$$A_L = \frac{M_L - \lambda_L^N}{M_L} \qquad (1)$$

The values for λ_L^N are obtained from the CPS II study (Thun et al. 1982) and are presented in Table 1. Where M_L - λ_L^N is negative (i.e., where observed mortality from lung cancer is lower than the expected rate among CPS II lifetime non-smokers) the value for A_L is set at 0. This occurs infrequently and usually only in cases where small population sizes give rise to large variances in the estimates. Although it could be argued that negative expected rates should have been used to avoid the problem of preferentially truncating underestimate errors but not overestimate errors, this was the method described by Preston, Glei, and Wilmoth (2010), and hence it has been followed here.

The second step is to estimate the mortality attributable to smoking from other causes of death (A_0) using the following formula:

$$A_0 = 1 - e^{-\beta_L'\left(M_L - \lambda_L^N\right)} \qquad (2)$$

where β_L' is the combined coefficient of lung cancer mortality in a regression model predicting non-lung cancer mortality using negative binomial models, including (in the PGW-R version of the method) all two-way interactions between year, age, and lung cancer mortality (see the supplementary material in Preston, Glei, and Wilmoth (2010) for a full description of the regression model from which the β_L' coefficients are derived). Typically both β_L' and M_L - λ_L^N are positive, resulting in positive mortality attributable to smoking from causes of death other than lung cancer, but if either β_L' or M_L - λ_L^N is negative, then their value is set to zero before calculating A_0. A negative value of β_L' would imply that lung cancer mortality rates are inversely associated with mortality from all other causes, suggesting, implausibly, that smoking has a protective effect in some age-sex strata.

The final step is to combine smoking-attributable lung cancer mortality with smoking attributable to deaths from other causes to produce the overall smoking-attributable fraction of deaths (A):

$$A = \frac{A_L D_L + A_0 D_0}{D} \qquad (3)$$

where D_L, D_0, and D are the observed numbers of deaths from lung cancer, other causes, and all causes combined respectively.

The β'_L parameters were calculated from the most up-to-date data available (from 1950 up until 2009 where possible) in the World Mortality Database (World Health Organisation 2011) and Human Mortality Database (University of California Berkeley and Max Planck Institute for Demographic Research 2011) using an identical negative binomial regression model to that used by Rostron (2010), with three exceptions: (i) Ireland was included and the United Kingdom was modelled as three separate nations (England and Wales, Scotland, and Northern Ireland); (ii) in the interaction terms the 'year' variable was modelled as categorical (in five-year bands) rather than continuous (linear); (iii) parameters were estimated for 80-85 years and 85+ rather than just 80+ years.[3] The values of β'_L obtained from this analysis are shown in Table 2, where they are compared with those used in the other studies.

A Montecarlo simulation with 1000 replications was used to generate confidence limits on the SAMF estimates. To do so the β'_L parameters were treated as normally distributed random variables using Wald standard errors, while the D_o and D_L numbers were simulated as Poisson random variables.

SAMF estimates for Maori, Pacific, and non-Maori non-Pacific male and female populations were calculated for the period 1996-1999 and these were compared with the estimates published in the studies listed above. The sensitivity of the PGW-R method to assumed rates of cancer among lifetime non-smokers was assessed by changing these values in the calculation. Smoking-deleted life tables were created to compare the impact of smoking on life expectancy in each sex-ethnicity group with the estimates from the Tobias and Cheung analysis.

3. Results

Table 3 shows the SAMF estimates obtained by the PGW-R method for each sex-ethnicity category and compares these with those obtained in the NZCMS (cohort) analysis, from the Laugesen (Peto-Lopez method) analysis, and those from the Ministry of Health analysis. There are marked differences between some of the SAMF estimates

[3] Although Preston, Glei, and Wilmoth's second paper provided a coefficient estimate for the age group 85+, this was estimated as the average of the coefficients for 70-74, 75-79, and 80-84. The age category 85+ was excluded from their regression model.

obtained from the various different sources. These are described in detail in the discussion section.

In Table 4 estimates of life expectancy at birth for each sex-ethnicity population group are presented, along with the life expectancies at birth derived from the smoking-deleted life table, and the difference between these two estimates for both the PGW-R method and the published figures from the Tobias and Cheung study. The time period covered by the latter is slightly different, which may explain its slightly higher overall life expectancy figures compared with the PGW-R method. The results of the PGW-R method are generally similar to those obtained by Tobias and Cheung for non-Maori, Maori males, and Pacific females. However, for Maori females the PGW-R method give a much higher loss of life expectancy attributable to smoking than the Tobias and Cheung estimates, while for Pacific males the PGW-R attributes a lower loss of life expectancy to smoking than Tobias and Cheung. However, it should be noted that the non-Maori non-Pacific group in the Ministry of Health analysis only includes those of European ethnicity (who nevertheless comprise the vast majority of this group).

The PGW-R method relies on the assumption that lung cancer mortality rates among people who have never smoked are approximately constant across the world (and equal to those from CPS II). This assumption has been challenged, as discussed below. Lung cancer incidence rates (which approximate to mortality rates given the high case-fatality rate of this disease) are given in Table 5 (census-cancer registry-linked data provided by Tony Blakely in a personal communication). They show large differences between these ethnic groups for both men and women, with about four times the lung cancer incidence rate in Maori compared with European/Other ethnicities.

The sensitivity of PGW-R estimates of smoking-attributable mortality to ethnic variation in lung cancer mortality among never smokers is explored in Table 6 by replacing the CPS II values used in the model with the true lung cancer mortality rates among Maori and European/Other lifetime non-smokers, estimated from the incidence rates in Table 5 and published relative survival rates for Maori and non-Maori (Ministry of Health 2010). For both groups there is a substantial reduction in the SAMF, to the point that the Maori male SAMF becomes comparable with the NZCMS value, although the female Maori SAMF remains significantly higher. For non-Maori non-Pacific estimates, on the other hand, the reduction in SAMF widens the disparity with the NZCMS estimates.

4. Discussion

Comparison of the SAMF estimates

The main purpose of this study was to determine whether the PGW-R SAMF estimates are consistent with the other SAMF estimates in New Zealand's population and in its various ethnic subpopulations. If the PGW-R method obtained similar results to other methods it would support the validity of this and the other methods. On the other hand, if the PGW-R results are different from those obtained by other methods then at least one of these methods must be in error.

For the total population the SAMFs obtained from the PGW-R method are 2-4% lower than those obtained in the Ministry of Health estimates and around 5% lower than the Laugesen estimates, but they are about 12% less than the NZCMS estimates. However, the most striking discrepancies between the studies are in the estimates for Maori. PGW-R and Laugesen both found higher SAMF estimates for Maori than European/Other, but the NZCMS study found the opposite. In the Ministry of Health study there were only small differences between Maori, Pacific, and European/Other ethnicities.

The life expectancy reductions attributable to smoking produced by the PGW-R method were reasonably compatible with those of Tobias and Cheung, except for Maori women and Pacific men. The PGW-R method estimated that, for women, almost half of the ethnic difference in life expectancy between Maori and European/Other was attributable to smoking, and for men it was over a third of the difference. In contrast, for Pacific men PGW-R attributed only 16% of the life expectancy difference to smoking compared with 34% by Tobias and Cheung.

This is not the first time that apparently contradictory results have been reported for SAMF in Maori compared to non-Maori. Easton (1995), comparing survivorship for the 1975-1977 period between Maori and non-Maori, found that almost all of the Maori/non-Maori difference in life expectancy at 25 years of age could be explained by smoking. In contrast, Smith and Pearce (1984) found that only 15% of the mortality difference between Maori and non-Maori between the ages of 15 and 64 could be explained by smoking (16% for females).

The differences in estimated SAMF are rather surprising, given the wide acceptance of the Peto-Lopez method and the high degree of consistency reported between the Peto-Lopez and PGW methods (Rostron 2010; Preston, Glei, and Wilmoth 2011). To try and explain them we consider seven possibilities:

1. Differences in the study populations
2. Passive smoking
3. Exposure to environmental carcinogens
4. Misclassification bias

5. Cannabis smoking
6. Ethnic differences in lung cancer among never smokers
7. Ethnic differences in the relationship between smoking and non-lung cancer mortality

Differences in the study populations

There are some differences between the studies in the populations used to calculate the SAMF estimates. The PGW-R method calculates the proportion of smoking-attributable deaths in the population aged 50 and over, whereas the Ministry of Health study (ages 15 and over) and Laugesen (ages 35+) studies use a wider age range in which the proportion of smoking-related deaths might be expected to be smaller. Hence, the finding that PGW-R estimates were lower than Ministry of Health estimates for all ethnicities except Pacific males cannot be explained by the difference in age ranges.

The PGW-R estimates, like those from the NZCMS, are derived from 1996-1999 data while the Tobias and Cheung ethnic-specific estimates cover the period 1995-1997. With falling rates of smoking-attributable death the 1996-1999 SAMF estimates would be expected to be lower than the 1995-1997 estimates, but not to the extent seen in the PGW-R and Ministry of Health results. In the Ministry of Health analysis the non-Maori non-Pacific life expectancies are restricted to European ethnicity, but the main discrepancies with the PGW-R results were in the Maori and Pacific ethnicities.

Passive smoking

When the excess lung cancer mortality rate is used as a proxy for smoking-attributable harm this rate includes the deaths attributable to passive smoking as well as those caused by active smoking, and therefore the mortality from non-lung cancer causes also incorporates the effects of passive smoking. However, the effect of passive smoking is not fully reflected in the excess lung cancer mortality rate because a proportion of deaths among the CPS II population of lifetime non-smokers would have been due to passive smoking.[4] In CPS II at baseline, 59% of the lifetime non-smoking women and 11% of the men were married to current or former smokers (Cardenas et al. 1997). Hence the PGW-R SAMF estimates do not entirely account for the effects of passive smoking on lung cancer.

Even if they did, would this be sufficient to explain the differences between PGW-R and NZCMS estimates? For non-Maori non-Pacific, allowing for the effects of passive smoking would exacerbate the discrepancies, since the NZCMS SAMF estimates (which do not incorporate the effects of passive smoking) are already higher

[4] This is because the purpose of subtracting the CPS II lung cancer rate in never smokers is to remove any expected lung cancer deaths that are not attributable to smoking. If these rates include deaths from passive smoking then the true smoking-attributable lung cancer mortality fraction will be underestimated.

than the PGW-R estimates in this group. For Maori men the PGW-R SAMF estimates are more than double the NZCMS estimates, which, to be explicable by passive smoking, would require that more deaths were attributable to passive smoking than active smoking. Clearly this is implausible.

Exposure to environmental carcinogens

Fine particulate matter and sulphur dioxide are associated with significantly increased risks of lung cancer and cardiopulmonary disease (Pope et al. 2002). Indoor coal fire smoke and cooking oil fumes are also recognised risk factors for lung cancer (Thun et al. 2008). However it is unlikely that ethnic differences in exposure to these carcinogens would bias the PGW-R SAMF estimates for two reasons: the number of deaths attributable to outdoor air pollution in New Zealand is relatively small (around 268 deaths per year for 1996-1999 in Auckland where about one third of the population live) (Scoggins et al. 2004); and Maori and Pacific are not more likely to live in areas with worse particulate air pollution in New Zealand (Pearce and Kingham 2008). Furthermore, the incidence rate of lung cancer among lifetime non-smokers in the relatively affluent CPS-II cohort was similar to that in the Swedish Construction Worker cohort, suggesting that differences in exposure to occupational and environmental pollutants were either small or carried relatively low risk (Thun et al. 2008).

Misclassification bias

The finding by the NZCMS of higher smoking-attributable mortality in European/Other males, who smoke less than Maori males, seems incongruous, especially since European/Other have lower lung cancer mortality rates. This section explores misclassification bias as a possible explanation; other possible explanations are discussed in the sections below. The NZCMS method depends upon census data for determining smoking exposure. Non-differential misclassification of exposure will raise or lower SAMF estimates depending upon the direction in which misclassification occurs: a tendency to over-report never smoking (plausibly the most likely misclassification) will tend to lower the observed SAMF below its true level. However, if the misclassification is non-differential by ethnicity it will raise NZCMS SAMF estimates for both Maori and European Other, which would cause the former to come closer to those of the PGW-R method, while the latter diverge even more. Only a tendency among Maori to over-report never smoking and a tendency among non-Maori to under-report it could increase the consistency of PGW-R and NZCMS estimates for both ethnicities.

Nevertheless, consideration should also be given to the fact that Maori start smoking earlier and continue longer (Laugesen and Swinburn 2000), and these

differences in exposure are not reflected in census classifications of current smoker, ex-smoker, and never-smoker. Among year ten students in 1997, for example, smoking prevalence in Maori females was 2.8 times that in non-Maori non-Pacific females, and in 1992 it was 3.1 times greater (Laugesen and Scragg 1999). Although there is no data on ethnic differences in tobacco consumption amongst smokers, if Maori were to have a higher intensity of exposure then this might explain some of the difference between PGW-R and NZCMS in Maori SAMF estimates. It would not, however, account for the higher SAMF in European males estimated from the NZCMS compared with the PGW-R method.

Disease misclassification could also bias SAMF estimates upward for Maori and Pacific if these ethnicities were more likely to be misclassified with lung cancer as the cause of death (e.g., when the lung tumour was metastatic rather than primary). However, lung cancer as a cause of death is generally considered to have a high degree of accuracy (Percy, Stanek, and Gloeckler 1981).

Ethnic differences in lung cancer among never smokers

Wilson, Blakely and Tobias (2006) questioned the reliability of the original Ministry of Health SAMF estimates on the grounds that Maori lung cancer mortality rates are higher than would be expected on the basis of tobacco smoking alone. This contention is supported by the data in Table 5, which shows mortality rates among Maori ranging from 1.74 times higher than European (in male current smokers) to 4.19 times higher in women who have never smoked. Furthermore, the rates among lifetime non-smokers are much higher than those found in the CPS II cohort, even for European/Other (Table 6). If the difference in lung cancer mortality is due to factors other than higher intensity, earlier onset, and longer duration of smoking in Maori (as already mentioned), then it will lead to an overestimate of smoking-attributable mortality from both lung cancer and other causes.

In a comprehensive review, Thun et al. (2008) noted significant ethnic differences in cancer mortality among lifetime non-smokers. Lung cancer incidence and death rates were found to be 34% higher among African American women than European women and 33% higher for men. Mortality rate ratios were even higher among Asians but the difference was not significant for those residing outside of Asia. The high mortality rate among European/Other non-smokers is perhaps more surprising, since Thun et al. (2008) found little evidence that CPS-II rates were underestimated or that lung cancer rates have changed among lifelong non-smokers in countries of a similar level of development.

The original Peto-Lopez method (the version used by Laugesen and Clements) made no adjustment for variation in non-smoker lung cancer mortality rates, but subsequent applications of the method have (Ezzati and Lopez 2003). This was justified

on the grounds that a large retrospective study in China found the relative risk of smoking for lung cancer to be virtually constant (at around three for smokers versus non-smokers) among cities with up to a ten-fold variation in their non-smoker lung cancer mortality rates (Liu et al. 1998). The modified smoking impact factors which are used to determine smoking-attributable deaths from causes other than lung cancer were therefore reduced by the ratio of lung cancer mortality in non-smokers to that in the reference population.

Variation in non-smoker lung cancer mortality brings into question the PGW method's assumption (also assumed in the Peto-Lopez method) that "smoking is the only source of variation in lung cancer death rates in the populations under consideration" (Preston, Glei, and Wilmoth 2011: 108). If the PGW-R SAMF estimates are adjusted by replacing the CPS-II λ_L^N rates with the actual ones found in the NZCMS study, then Maori estimates become more similar to those from NZCMS, but the European/Other estimates also diverge further from those of the NZCMS. Moreover, in contrast to the data from China, the relative risks of smoking are not constant across ethnic groups as they are across cities in China (Blakely and Wilson 2005). Excess mortality is two to three times lower in Maori (as shown in Table 5), and there is no obvious risk factor, such as indoor coal smoke, that explains the much higher lung cancer mortality rates in all New Zealand ethnicities.

Cannabis smoking

Cannabis is a risk factor that might explain some ethnic variation in lung cancer mortality among lifelong non-smokers and the high level of lung cancer mortality in this group generally. A recent NZ case-control study of lung cancer in adults under 55 years of age found that the risk of lung cancer increased by 8% for each joint-year of cannabis smoking (compared with 7% for each pack-year of cigarette smoking) (Aldington et al. 2008). Furthermore, those who started smoking cannabis under the age of 16 had a relative risk of 10.3 compared with those starting aged 21 or older. As with tobacco, Maori men and women smoke cannabis at a higher intensity than European and also begin at a younger age. However, the link between cannabis smoking and lung cancer remains uncertain (Hall and Degenhardt 2009).

The effect of higher cannabis use in Maori could therefore be to raise the lung cancer excess mortality (M_L - λ_L^N), which would also cause an increase in the number of deaths attributable to smoking from causes other than cancer (D_0). The effect on SAMF estimates would be to bias these upwards. However, since this would be true for non-Maori as well as Maori (in proportion to their respective cannabis exposure), a carcinogenic effect of cannabis would not explain the higher SAMF estimates for non-Maori non-Pacific reported from the NZCMS compared with the PGW-R estimates.

Ethnic differences in the relationship between smoking and non-lung cancer mortality

Apart from assuming that smoking is the only source of variation in lung cancer death rates and that in non-smokers the CPS-II rates are valid for all populations, the PGW-R also relies upon the assumption that the relationship between smoking-related harm and non-lung cancer mortality is constant across populations for a given age-sex group in any given year. Preston, Glei, and Wilmoth (2011) cite several studies (including Liu et al. (1998), discussed above) in support of this constancy assumption, but Hunt et al. (2005) pointed out that this assumption does not appear to hold across New Zealand's ethnic groups. As with the lung cancer mortality-relative risks for smoking shown in Table 5, Hunt et al found relative risks were much higher among non-Maori non-Pacific compared with Maori (and Pacific) for ischaemic heart disease and all-cause mortality.

There are at least two possibilities that would call into question the validity of this assumption. One is that for any given smoking-related disease there may be ethnic differences in the effect of exposure on mortality. These could be mediated by factors that affect disease survival, including access to health care, comorbidities, and nutrition, as well as factors that affect susceptibility to acquiring the disease, such as genetic predispositions. Thus Maori and Pacific estimates of smoking-attributable mortality fractions could be biased upwards if, within these ethnic groups, a given exposure of smoking is relatively more likely to lead to acquiring and/or dying of lung cancer than other smoking-related causes of death such as myocardial infarction. This might occur if smoking has a weaker relative effect on cardiovascular disease for Maori and Pacific than for other ethnicities, or, conversely, if it confers a higher risk of developing lung cancer. Although Maori and Pacific (and South Asian ethnicities) are actually known to be more susceptible to cardiovascular events than other ethnic groups for a given Framingham risk profile (Riddell et al. 2010), they may have even greater relative susceptibilities to lung cancer.

The other possibility is that the mix of disease varies by ethnicity because of exposures unrelated to smoking (e.g., diet). If cardiovascular disease in Maori is driven more by diabetes and lifestyle factors other than smoking, the strength of the association between smoking and non-lung cancer mortality would be less and SAMF estimates would then be overestimated. Again this would not explain the difference in SAMF estimates for European/Other ethnicities between the NZCMS and PGW-R methods.

5. Conclusions

The new PGW-R method offers the potential to simplify the estimation of smoking-attributable mortality fractions and has certain theoretical advantages over the well established Peto-Lopez method. However, its validity has yet to be determined, particularly when applied to ethnic or other subpopulations, and this analysis has revealed several issues that call this into question:

1. Lung cancer mortality rates among lifelong non-smokers are much higher than those derived from CPS II for all ethnicities in New Zealand, but particularly so for Maori.
2. Relative risks of smoking for lung cancer, ischaemic heart disease, and all-cause mortality are not constant across ethnic groups in New Zealand.
3. Ethnic differences in exposure to risk factors other than tobacco smoke lead to ethnic differences in the mix of diseases that are not necessarily consistent with the PGW-R method's assumed relationship between lung cancer and non-lung cancer mortality rates.
4. The PGW-R ethnic-specific estimates of SAMF are not consistent with direct estimates from the NZCMS.

On the other hand, for the NZCMS the significantly higher SAMF in non-Maori non-Pacific men compared with Maori men also raises questions about the accuracy of these estimates, unless the combined mortality effects of all other risk factors outweigh the effects on diseases other than lung cancer of greater smoke exposure in Maori. No single explanation has been found that adequately accounts for all of these issues. We suggest that they could be due to a combination of imprecision in the measurement of true exposure in studies using simple current and ex-smoker categories, the effects of cannabis smoking or some other as yet unidentified non-tobacco causes of lung cancer, and methodological weaknesses with the PGW-R method. Until we better understand the reasons for these discrepancies it would be prudent to use both NZCMS estimates and the PGW-R method cautiously when examining ethnic differences in SAMF.

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Appendices

Table 1: Lung cancer death rates among non-smokers

Age group (years)	CPS-II study mortality rate (per 1000 population)	
	Males	Females
50-54	0.06	0.06
55-59	0.05	0.07
60-64	0.12	0.12
65-69	0.22	0.17
70-74	0.35	0.31
75-79	0.52	0.33
80-84	0.89	0.58
85+	0.87	0.61

Table 2: Model coefficients for lung cancer death rates derived using the PGW and PGW-R methods

Age group	Model coefficients for lung cancer death rates (per 1000)					
	Preston *et al* (Preston, Glei, and Wilmoth 2011) (PGW method) for 2003		Rostron (Rostron 2010) (PGW-R method) for 2003		Updated PGW-R model for 1995-1999*	
	Male	Female	Male	Female	Male	Female
50-54	0.320	0.745	0.348	0.707	0.302	0.734
55-59	0.170	0.482	0.174	0.510	0.163	0.499
60-64	0.104	0.297	0.113	0.382	0.102	0.340
65-69	0.069	0.162	0.079	0.218	0.069	0.200
70-74	0.048	0.087	0.060	0.137	0.047	0.111
75-79	0.038	0.057	0.046	0.061	0.031	0.040
80-84	0.040	0.094	0.028	0.013	0.021	-0.010
85+	0.042	0.080			0.012	-0.069

*Note:** Since 1995-1999 is the period being analysed here the coefficients for this period are presented.

Table 3: Smoking-attributable fraction of all deaths by PGW-R method compared with Ministry of Health, NZCMS and Laugesen estimates

	Maori		Pacific		European/other		Total population	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
Current study (PGW-R) method[a] (95% C.I.)	36% (33%-39%)	48% (44%-51%)	27% (22%-31%)	15% (10%-20%)	14% (13%-15%)	6.7% (6.2%-7.2%)	15% (14%-16%)	8.8% (8.3%-9.4%)
NZCMS cohort study[b]	17%	25%	-	-	28%	25%	25%	24%
MoH (English *et al* method)[c]	22%	21%	19%	8%	19%	10%	19%	11%
Laugesen et al (Peto-Lopez method)[d]	29%	34%	-	-	-	-	17%	

Note:

a. Aged 50 plus in years 1996-1999
b. Aged 45-74 in years 1996-1999
c. Age 15 and over in 1996
d. Ages 35 and over, 1995 for total population, 1989-93 for Maori.

Table 4: Life expectancy at birth in years by ethnicity and sex, including and excluding smoking-attributable deaths: PGW-R method compared with Tobias and Cheung's estimates

	Maori		Pacific		Non-Maori non-Pacific		Total population	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
Including smoking-attributable deaths								
PGW-R method[a]	67.1	72.1	69.4	75.8	76.3	81.4	75.1	80.3
Tobias & Cheung[b]	67.2	71.6	69.8	75.6	75.6	80.9	74.4	79.6
Excluding smoking-attributable deaths								
PGW-R method	70.9	77.6	71.9	77.3	77.7	82.3	76.6	81.5
Tobias & Cheung	70.7	74.0	73.4	76.5	77.2	81.6	76.2	80.4
Life expectancy difference								
PGW-R method	3.8	5.5	2.5	1.5	1.4	0.9	1.5	1.2
Tobias & Cheung	3.5	2.4	3.6	0.9	1.6	0.7	1.8	1.0
Proportion of ethnic difference in life expectancy attributable to smoking								
PGW-R method	35%	49%	16%	11%	-	-	-	-
Tobias & Cheung	23%	18%	34%	4%	-	-	-	-

Note:

[a]1996-1999 based on attributable fraction over the age of 50.
[b]1995-1997 based on attributable fraction over the age of 15.

Table 5: Lung cancer incidence rates from 1996-1999 in Maori and European/Other 55-74 year-olds who have never smoked (cases per 100,000 with 95% confidence limits)

	Lung cancer incidence rates				Rate ratios for		Rate ratios for smoking status			
	Maori		Euro/other		Maori vs Euro/Other		Maori		Euro/Other	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Never smoked	181 (122-23)	126 (86-166)	45 (38-53)	30 (26-35)	4.00	4.19	1.0	1.0	1.0	1.0
Ex-smoker	286 (215-358)	292 (207-378)	158 (146-170)	126 (112-140)	1.81	2.32	1.6	2.3	3.5	4.2
Current smoker	813 (684-941)	722 (601-844)	467 (433-500)	333 (302-364)	1.74	2.17	4.5	5.7	10.4	11.1

Source: Tony Blakely, NZCMS data, personal communication.

Table 6: Sensitivity analysis of the effect of assumed lung cancer mortality in never smokers (λ_L^N) on estimated smoking-attributable mortality fractions in ages 55-74

Assumed λ_L^N	Smoking-attributable mortality fraction (95% C.I.)			
	Maori		European/Other	
	Male	Female	Male	Female
CPS II	40.5% (36.7%-44.2%)	62.1% (57.3%-66.9%)	18.6% (17.4%-19.8%)	19.5% (17.9%-21.0%)
Actual [a] λ_L^N	25.6% (21.3%-29.8%)	33.6% (29.2%-38.0%)	15.0% (13.9%-16.2%)	15.7% (14.1%-17.2%)
RR[b]	12.2	8.5	2.4	1.7

Note:

[a]/ Estimated from incidence rate with case-fatality rates based on published five year relative survival from 1994-2007.(Ministry of Health 2010).

[b]/ Relative rate of actual lung cancer mortality in lifetime non-smokers compared with the CPS II rate, assuming case fatality rates of 94%, 92%, 89% and 88% for Maori male/female and European/Other male/female respectively.