MORTALITY FROM SMOKING IN NEW ZEALAND

The association between cigarette smoking and mortality from all-causes, ischaemic heart disease and stroke in New Zealanders aged 25-74 years, 1981-1984 and 1996-1999

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Abstract

BACKGROUND

Smoking causes death. However, there are two reasons to specifically examine the strength of the smoking-mortality association in New Zealand. First, it is plausible that the strength of association (in epidemiological terms) varies in New Zealand, and may also vary by demographics and over time. Second, and by extension, New Zealand-specific estimates of the smoking-mortality association are required for policy-makers estimating smoking-related burden.

OBJECTIVE

To measure the strength of the association of cigarette smoking with mortality from all-causes, ischaemic heart disease (IHD) and stroke among 25-74 year olds during 1981-84 and 1996-99 in New Zealand.

METHODS

Cohort studies of the New Zealand population, formed by linking information from each of the 1981 and 1996 censuses to mortality data in the following three years, were used to determine mortality incidence rates (deaths per person-years), and subsequently rate ratios and rate differences for current smokers and ex-smokers, compared to never-smokers as the reference group. Age (and for some strata, ethnicity) standardised rate ratios and rate differences were calculated using the direct method. Rate ratios adjusted for age (± ethnicity) and socio-economic position (SEP) were calculated using multivariable analysis (poisson regression).

RESULTS

There were important variations in the association of smoking with mortality by cohort (time) and ethnicity, and to some extent sex and age.

Time

Age and ethnicity standardised rate ratios for all-cause mortality comparing smokers to never smokers (ages 25-74) increased over time, with the excess rate ratio (ie. rate ratio minus one) approximately doubling from 1981-84 to 1996-99, for both males (1.59 (95% CI 1.53-1.66) to 2.05 (1.97-2.14)) and for females (1.49 (1.42-1.56) to 2.01 (1.91-2.12)). Likewise, the excess rate ratios approximately doubled over time for IHD (1.50 (1.40-1.61) to 2.03 (1.87-2.20) for males; 1.86 (1.70-2.04) to 2.67 (2.35-3.03) for females) and for stroke (1.50 (1.29-1.75) to 1.93 (1.59-2.34) for males; 1.65 (1.42-1.92) to 2.51 (2.06-3.05) for females). The standardised rate differences showed some increase over time for all-cause mortality but little change for IHD and stroke.

Ethnicity

There were also marked variations in the standardised rate ratios by ethnic group (Māori, Pacific, and non-Māori non-Pacific), which were determined to be statistically significant for both sexes, both years, and for all measured outcomes. In 1996-99, the male all-cause mortality age-standardised rate ratios for current smokers versus never smokers were 1.51 (1.35-1.69) for Māori, 1.18 (0.94-1.47) for Pacific, and 2.22 (2.12-2.33) for non-Māori non-Pacific. Likewise, among females the rate ratios were 1.45 (1.27-1.66) for Māori, 1.05 (0.75-1.48) for Pacific, and 2.20 (2.09-2.33) for non-Māori non-Pacific. A similar pattern of rate ratio heterogeneity by ethnicity existed in 1981-84, although the strength of the rate ratios was less in all ethnic groups. In contrast to the rate ratio heterogeneity, for 1996-99 Māori and non-Māori non-Pacific standardised rate differences of smokers versus never smokers were reasonably comparable (within sex).

Sex

By sex, the rate ratios were similar between males and females for all-cause mortality. For example, the 1996-99 age and ethnicity standardised estimates for the 25-74 group were 2.05 (1.97-2.14) for males and 2.01 (1.91-2.12) for females. However, the IHD and stroke rate ratios were higher for females than males. Standardised rate differences were higher for males for all-cause and IHD mortality, reflecting the higher underlying mortality rates.

Age

By age, the rate ratios increased with increasing age for all-cause mortality. For example, among females in 1996-99 the age and ethnicity standardised rate ratios for current versus never smokers for the 25-44, 45-64, and 65-74 age groups were 1.20 (1.03-1.40), 1.89 (1.75-2.05), and 2.32 (2.16-2.49) respectively. In contrast, the IHD (and female stroke) rate ratios decreased with increasing age. Thus, the association of smoking with all-cause mortality on a relative scale rose with age, as a greater percentage of deaths at older ages are smoking related. But for the smoking related disease of IHD, the relative risks decreased with age.

Multivariable analysis revealed a moderate degree of confounding by socio-economic position. Adjustment for SEP, as measured by a range of variables, reduced the age and ethnicity adjusted poisson regression estimates for the all-age all-ethnicity group by 21-28% for males and 5-9% for females in 1981-84, and by 33-38% for males and 21-25% for females in 1996-99. Thus, confounding by SEP was more pronounced among males, and increased over time for both males and females. Rate ratios adjusted for SEP still demonstrated heterogeneity by time and ethnicity.

CONCLUSION

The relative strength of the association between smoking and mortality from all-causes, IHD and stroke in the New Zealand population, varies by ethnicity and time. For IHD and stroke, it also varies by sex. Socio-economic position is demonstrated as a moderate confounder of this association, however it does not explain most of the relationship between smoking and mortality, nor the heterogeneity seen. One of the main determinants of the heterogeneity by ethnicity and time is the variation in underlying mortality rates.

The rate ratio estimates determined from this study differ to some degree from those found overseas, and notably so for Māori. Therefore they should be used for any New Zealand-specific research and policy that requires relative risk measures of smoking and mortality.

Statistics NZ security statement

The New Zealand Census-Mortality Study (NZCMS) was initiated by Dr Tony Blakely and his co-researchers from the Wellington School of Medicine, University of Otago. It was approved by the Government Statistician as a Data Laboratory project under the Microdata Access Protocols. This security statement is essentially the same as that provided for the original NZCMS research project.

The NZCMS fully complies with the 1975 Statistics Act.

Requirements of the Statistics Act

Under the Statistics Act 1975 the Government Statistician has legal authority to collect and hold information about people, households and businesses, as well as the responsibility of protecting individual information and limits to the use to which such information can be put. The obligations of the Statistics Act 1975 on data collected under the Act are summarised below.

1. Information collected under the Statistics Act 1975 can be used only for statistical purposes.

2. No information contained in any individual schedule is to be separately published or disclosed to any person who is not an employee of Statistics New Zealand, except as permitted by sections 21(3B), 37A, 37B and 37C of the Act.

3. This project was carried out under section 21(3B). Under Section 21(3B) the Government Statistician requires an independent contractor under contract to Statistics New Zealand, and any employee of the contractor, to make a statutory declaration of secrecy similar to that required of Statistics New Zealand employees where they will have access to information collected under the Act. For the purposes of implementing the confidentiality provisions of the Act, such contractors are deemed to be employees of Statistics New Zealand.

4. Statistical information published by Statistics New Zealand, and its contracted researchers, shall be arranged in such a manner as to prevent any individual information from being identifiable by any person (other than the person who supplied the information), unless the person owning the information has consented to the publication in such manner, or the publication of information in that manner could not reasonably have been foreseen.

5. The Government Statistician is to make office rules to prevent the unauthorised disclosure of individual information in published statistics.

6. Information provided under the Act is privileged. Except for a prosecution under the Act, no information that is provided under the Act can be disclosed or used in any proceedings. Furthermore no person who has completed a statutory declaration of secrecy under section 21 can be compelled in any proceedings to give oral testimony regarding individual information or produce a document with respect to any information obtained in the course of administering the Act, except as provided for in the Act.

Census data

Traditionally, data from the Population Census is published by Statistics New Zealand in aggregated tables and graphs for use throughout schools, business and homes. Recently Statistics New Zealand has sought to increase the benefits that can be obtained from its data by providing access to approved researchers to carry out research projects. Microdata access is provided, at the discretion of the Government Statistician, to allow authoritative statistical research of benefit to the public of New Zealand.

The NZCMS uses anonymous census data and mortality data that are integrated (using a probabilistic linking methodology) as a single dataset for each census year. The NZCMS is the first project for which the census has been linked to an administrative dataset for purposes apart from improving the quality of

Statistics New Zealand surveys. The project has been closely monitored to ensure it complies with Statistics New Zealand's strict confidentiality requirements.

Further information

For further information about confidentiality matters in regard to the NZCMS, please contact either:

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Chapter 1: Introduction

Introduction Summary

Smoking causes a large burden of disease and mortality in New Zealand. Relative risk estimates measuring the strength of the association between smoking and mortality are necessary to calculate this burden. However, as New Zealand-specific estimates are not available relative risk measures have been "borrowed" from overseas studies. It is hypothesised that the relative risk from smoking in New Zealand differs from that observed overseas. A literature review and two cohort studies are conducted to test this hypothesis.

1 Impact of smoking in New Zealand

Tobacco smoking makes the largest contribution of any single risk factor to the burden of disease in New Zealand, accounting for approximately 15% of Disability Adjusted Life Years (DALYs) lost among males, and 9% among females (Tobias and Cheung 2001). It was estimated that the total number of deaths in 1996 caused by smoking was over 4,000 (Ministry of Health 1999). The decline in smoking prevalence has slowed in recent years (around 25% in 2001) (Ministry of Health 2002a); it has become more common among New Zealanders living in more socio-economically deprived areas (Howden-Chapman and Tobias. 2000), and among Māori and Pacific populations (Ministry of Health 2002a). A recent Ministry of Health report shows that tobacco contributes significantly to inequalities in life expectancy, accounting for about one-third of the small area socio-economic gradient and one-quarter of the inequality between Māori and non-Māori (Tobias and Cheung 2001).

Mortality from cardiovascular disease is a particularly important outcome from smoking. Among all ethnic groups cardiovascular disaese causes more deaths, and more "avoidable" deaths, than any other cause in New Zealand (Ministry of Health 1999; Tobias 2001). The number of deaths in 1996 from Ischaemic Heart Disease and Stroke combined that were caused by smoking was estimated to be 1,280 (Ministry of Health 1999). The numbers from lung cancer and COPD were estimated to be 1,083 and 1,160 respectively (Ministry of Health 1999).

Reducing smoking in the population, in order to prevent this toll of morbidity and mortality, is one of the 13 priority objectives of the New Zealand Health Strategy (King 2000).

2 Effect measure data

Calculating the burden of disease and mortality from smoking in New Zealand is particularly important in informing policies and strategies for tobacco control. As part of these calculations it is necessary to know the strength of the association between smoking

exposure and the outcome of interest (eg. mortality), as measured by the relative risk between smokers and non-smokers. To date, accurate measures of effect, such as relative risk estimates, have not been available specifically for the New Zealand population, nor for groups within it. Research and policy within this country has therefore relied on relative risk estimates "borrowed" from overseas studies.

One of the most recent reports on New Zealand tobacco mortality, 'Inhaling Inequality' (Tobias and Cheung 2001), utilises relative risk estimates from the second Cancer Prevention Study (CPS II) to calculate population attributable risk. A 1998 report for Te Puni Kokiri (Laugesen and Clements 1998) and a paper by Laugesen and Swinburn (2000) in Tobacco Control also used CPS II relative risks to respectively calculate deaths attributable to cigarette smoking among Mäori, and deaths averted in New Zealand from smoking cessation.

A number of other New Zealand reports have also used relative risk estimates from overseas studies, including 'The Burden of Disease and Injury in New Zealand' (Tobias 2001), and 'Our Health, Our Future' (Ministry of Health 1999). As a result, the 'Burden of Disease' report (page 26) cautions that:

> "These results should be regarded as approximate only, for the following reasons.
> Associations between the causes considered and the diseases included in the New Zealand Burden of Disease Study have not been fully investigated in all cases.
> The relative risks used to calculate the PARs have mostly been extracted from the international literature and may differ from those pertaining in New Zealand in 1996…."

This report also states "In the absence of data to the contrary, it had to be assumed that relative risks for both Māori and non-Māori ethnic groups were similar."

CPS II relative risks were used in a papers by Peto et al (1992) and Murray and Lopez (1997), and the World Health Report 2002 (WHO 2002) to estimate mortality from

tobacco globally. However Peto et al also caution that such effect measures cannot be extrapolated directly to other populations.

3 Thesis objectives

The rationale for this thesis is effect measure estimates from overseas studies may not accurately reflect the strength of the smoking-mortality association in New Zealand. It is hypothesised that the relative strength of this association in the New Zealand population as a whole, and for populations within it and over time, vary.

Based on these possibilities, the primary objective of this thesis is:

> To measure the strength of the association of cigarette smoking with mortality from all-causes, ischaemic heart disease (IHD) and stroke among 25-74 year olds in the New Zealand population, over time (during 1981-84 and 1996-99), by ethnicity and by sex

A secondary objective is:

> To illustrate that effect measure data, in this case for the association of smoking and mortality, should be determined specifically for the population or populations of interest.

In meeting these objectives, this thesis is comprised of two main parts. The first is a literature review looking at the consistency, or inconsistency, of published mortality effect measures from smoking worldwide, as well as any empirical evidence or theories for any variation.

The second and central part of the thesis consists of new research from two cohort studies measuring the smoking-mortality association in the New Zealand population. Two of the main focus points of this research are examination of this association in different ethnic groups, and possible changes in strength of the association over time. The exposure is

limited to cigarette smoking, and the mortality outcomes of interest are limited to all-cause mortality, ischaemic heart disease, and stroke. This emphasis on cardiovascular disease is justified by its large impact in New Zealand. The relatively short follow-up period of the cohorts, which is fixed at three years (see chapter 3), also allows greater validity for measuring cardiovascular outcomes associated with smoking, as opposed to diseases with a longer latent period such as cancer.

Both the literature review and the new research focus on relative risk estimates, as these are the most commonly reported measure of effect, they provide better comparisons between studies and countries, and they are used for informing policy (eg. through population attributable risk calculations). Nevertheless, rate differences are an important measure of absolute effect that require consideration in parallel with rate differences, and are reported and discussed in this thesis.

In this thesis, the causal relationship between smoking and increased mortality is taken as proven. It does not seek to explain all the biological mechanisms by which smoking causes cardiovascular disease. What this new research adds is demonstration of the size (or strength) of the smoking-mortality association in the entire New Zealand population aged 25-74, and it appears to be the first to do so. In particular, this thesis includes study data for Māori and Pacific as well as non-Māori non-Pacific. As such, the study is statistically powerful.

4 The New Zealand Census-Mortality Study

The main part of this thesis, which estimates New Zealand-specific effect measures of smoking and mortality, is research conducted within a larger ongoing study – the New Zealand Census-Mortality Study (NZCMS). The NZCMS was initiated by Dr Tony Blakely and his co-researchers from the Wellington School of Medicine, University of Otago in the late 1990s. It has so far created datasets containing information from four censuses (1981, 1986, 1991, 1996) linked to mortality records for the three years following each census, thereby creating four separate cohort studies of the entire New

Zealand population over an extended period of time. The NZCMS is described further in chapter 3.

Chapter 2: Consistency of effect measure estimates: literature review

Literature Review Summary

Many studies worldwide have examined the association between cigarette smoking and health outcomes. While these studies report a consistent association between smoking and mortality, the observed *strength* of this association varies for different study populations. Among a number of large cohort studies, the relative risk of all-cause mortality for smokers compared with never-smokers ranges from 1.2 in China to 2.3 in the United States CPS II study. Effect measure estimates for ischaemic heart disease and stroke mortality also vary between different populations.

There are two main explanations for this heterogeneity in the strength of the smoking-mortality association. The first is that it is an artefactual phenomenon, caused by different methodologies between the studies. The second is that the variation in strength is real, and could be due to differences in patterns of tobacco consumption, levels of other health risk factors (that interact with smoking and mortality), and/or the chemical constituents of cigarettes. In particular there are differences between these factors in the New Zealand population and overseas, suggesting that effect measure estimates in this country may also vary.

Variation in the strength of the smoking-mortality association overseas, and differences in factors that may be contributing to this variation, highlight the need for New Zealand-specific data in this area. Very few New Zealand studies have addressed this issue, with only one published study reporting mortality in smokers compared with never-smokers, and none examining this association amongst Māori and Pacific peoples. There are obligations based on both public health need, and tangata whenua and Treaty of Waitangi rights, to obtain ethnicity-specific information such as the health effects of smoking.

A literature review was conducted on the consistency, or inconsistency, of published mortality effect measures from smoking worldwide (ie. the strength of the association between smoking and mortality), as well as any empirical evidence or theories for any variation. This chapter summarises the findings of this review, and illustrates the rationale for and importance of this study.

This review focuses on relative risk estimates, as these tend to be the most common effect measure published, and used for comparison between studies and countries as well as attributable burden calculations. It should be noted however that this is only one measure of association, and some of the findings of this review may not apply to other effect measures (such as rate differences).

This chapter is structured in the following way:

1 A description of the methodology of the literature review.
2 A review of published relative risk estimates, in particular from large cohort studies that have examined smoking and mortality. It should be noted that the precision of the estimates (where available) is discussed separately in section 3.1.2.1 (random error) as a possible explanation for the observed variation in relative risk estimates.
3 A discussion of the possible reasons for the heterogeneity of relative risk estimates seen in section 2, including:
    3.1 Artefactual variation – ie. possible methodological differences between the studies, including study design, random error and systematic error (such as misclassification and confounding)
    3.2 Possible "real" reasons. This section is the major substance of this chapter and focuses on explanations such as effect measure modification (statistical interaction), biological interaction, varying levels of health risk factors, and the chemical constituents of cigarette smoke.
4 This section suggests that all the variations the previous sections highlight the need for New Zealand-specific effect measure estimates, and illustrates that the studies conducted in New Zealand to date are insufficient in this regard.
5 The final section emphasizes the particular importance of ethnicity-specific data in New Zealand.

1 Literature review methodology

The literature review was conducted using the Medline database as well as other sources of information.

1.1 Medline

An initial Medline search was undertaken using a number of MESH headings and keywords.

A search on 'Smoking' or 'Tobacco' MESH headings was done in conjunction with a number of other terms below, but also by itself, with the latter restricted to English language reviews (including evidence based medicine reviews) and meta-analyses since 1980.

Tobacco / Smoking was cross referenced with a number of outcome MESH headings, including 'mortality', 'cause of death', 'fatal outcome', 'hospital mortality', 'survival rate', 'Heart Diseases', 'Angina Pectoris', 'Coronary Disease', 'Risk Factors', 'Myocardial Ischemia', 'Myocardial Infarction', and 'Cerebrovascular Accident'; and also keywords including 'Heart Disease', 'Ischaemic Heart Disease', 'Stroke', 'Cerebrovascular Disease', 'CVD', 'CHD', 'IHD','CVA'. Review articles, and articles on risk factors, for these outcomes were also searched for.

Also cross referenced with Smoking / Tobacco and cardiovascular diseases / outcomes were the MESH terms 'risk' and 'logistic models', and the keywords 'relative risk', 'rate ratio', 'size of effect', 'association', and 'effect size'. The same cross-referencing was done for 'cohort studies' (MESH and keyword).

Articles were also looked for that discussed variability or heterogeneity of results, and reasons for this, using terms such as the MESH headings 'reproducibility of results', 'comparative study', 'observer variation', 'multivariate analysis', 'models, statistical', 'causality', 'epidemiologic methods'; as well as the keywords 'homogeneity', 'heterogeneity', 'variation', 'consistency', 'inconsistency', 'difference', 'variation',

'variability'. A restriction was also placed on just those articles that also matched the MESH headings 'Public Health' or 'Epidemiology'.

Papers were also searched for those that discussed 'effect modification', 'synergy', or 'interaction' as keywords, as well as those discussing 'epidemiologic methods' (keyword and MESH).

The above searches were also modified and combined in various ways to find the most appropriate references. Secondary and Tertiary (and sometimes more) references were also found from citations in the initial journal articles, and from new Medline searches on MESH headings and keywords that became apparent on reading these papers.

1.2 Other Sources

As well as Medline searches, information was obtained from similar searches of the Cochrane Database of Systematic Reviews; looking for relevant articles in recent issues of the 'International Journal of Epidemiology', 'Tobacco Control' and 'Epidemiology; from references suggested by other people, and among those already held by the Department of Public Health and the author.

A number of reports were also obtained from the internet, subsequent to searches using the 'Google' search engine (http://www.google.co.nz) with search terms similar to those used for Medline above, and from searching and browsing specific websites, including:

- Ministry of Health http://www.moh.govt.nz
- National Drug Policy http://www.ndp.govt.nz
- ASH New Zealand http://www.ash.org.nz
- Statistics New Zealand http://www.stats.govt.nz
- Te Puni Kokiri http://www.tpk.govt.nz
- World Health Organisation http://www.who.int
- Centers for Disease Control and Prevention http://www.cdc.gov
- National Cancer Institute (USA): Tobacco Control Research http://cancercontrol.cancer.gov/tcrb/monographs/
- US Surgeon General http://www.surgeongeneral.gov

2 Consistency of published effect measure estimates

A large number of studies worldwide have examined the association between cigarette smoking and health outcomes, and have established a causal relationship for many diseases including cardiovascular disease and lung cancer. A relationship with all-cause mortality is also consistently seen among the large, well-conducted studies. Of the large prospective cohort studies that have measured the effect of smoking on mortality, the two that are probably most widely cited are the British Doctors' Study, and the second Cancer Prevention Study in the United States (CPS II). The former is the longest running cohort study on this issue, and has now being going for more than 40 years (started in 1951) (Doll, Peto et al. 1994). CPS II is probably the largest cohort study in recent years (CPS I was slightly larger), with a cohort of over 700,000 (Thun, Day-Lally et al. 1997a). In some ways these two studies have unofficially taken the role of being the "gold standard" for effect measure estimates of smoking mortality. As mentioned in chapter 1, CPS II data have been used to calculate the global burden of disease from tobacco (Peto, Lopez et al. 1992; Murray and Lopez 1997; WHO 2002), and also for calculating population attributable risk from smoking in New Zealand (Tobias and Cheung 2001).

However both the British Doctors Study and CPS II are not without problems or criticism. For example, the British Doctors' Study is smaller than other studies, and is also on a relatively select subpopulation of the United Kingdom (UK) – ie. medical practitioners – therefore its results may not be generalisable. The CPS II study population may also not be representative of the US population (let alone other countries) as it is comprised of friends, neighbours and acquaintances of American Cancer Society volunteers – these participants were "older, more educated, and more frequently married and part of the middle class than the general US population." (Thun, Day-Lally et al. 1997a).

There is also no real agreement in the literature on whether there is such a thing as "the most accurate" estimate. In fact, a recurring theme appears to be a caution in relying on one study, or on effect measure estimates that have not been specifically measured in the population of interest (Peto, Lopez et al. 1992; Doll, Peto et al. 1994; Prescott, Osler et al. 1997; Beaglehole, Saracci et al. 2001 Oct). This is especially important given that most of

the large studies to date have been conducted in one country – the United States. In 1994, Richard Doll made the point that:

> "whatever its size, no single epidemiological study can provide an adequate basis for assessing the worldwide epidemic of death from tobacco, because the epidemic is at a different stage, and is evolving so differently, in different populations." (Doll, Peto et al. 1994)

Beaglehole et al (2001) also note that for cardiovascular disease "the quantitative relationship between the major risk factors and CVD endpoints vary by population."

Some evidence for this point of view comes from looking at the consistency (or not) of effect measure estimates published in the medical literature. As relevant examples, relative risks (except for Framingham which are Odds Ratios) from a selection of cohort studies looking at (current) smoking and mortality are presented in Table 1 (all-cause mortality), Table 2 (Ischaemic Heart Disease) and Table 3 (Stroke). It should be noted that a selective approach was taken in choosing the studies shown in the tables, rather than presenting a complete systematic review. These studies are some of the largest and/or most recent that are quoted in the literature. The Kaiser Permanente study is also included as it provides the only published data on mortality risk from smoking among African American women (study participants are subscribers of the Kaiser Permanente Medical Care Program in California) (Friedman, Tekawa et al. 1997). The reference group for most of the relative risks is "never-smokers" (except MRFIT – see footnote to tables).

It should be noted that data from MRFIT, which was an intervention study, are from follow-up of the original cohort of men screened for the trial. A total of 361,622 men were screened over a two-year period beginning in 1973, and from this group 12,866 men were randomised into two trial arms (usual care or special intervention). Follow-up of the initial screening group provided a large cohort study examining the effects of smoking.

It should also be noted that Framingham data are possibly less accurate, or less comparable to other studies. It was stated in the 2001 US Surgeon General's report on smoking that the Framingham investigators could not control for the changing background cardiovascular disease rates (for this reason data from Framingham analyses were not

included in the 2001 Surgeon General's report) (USDHHS 2001a). A more detailed explanation was not given. Nevertheless, it is included in the tables for completeness.

Data from only one large prospective cohort study in a non-western population, the Chinese Academy of Preventive Medicine (CAPM) study, are shown in Table 1 (all-cause mortality data available only) (Niu, Yang et al. 1998). There appear to be relatively few large well conducted studies from Asia to date, however continuing analysis from the CAPM study should provide some important information. The Chinese Academy of Preventive Medicine has established 145 nationally representative "disease surveillance points", each with about 100,000 residents in 5-8 groupings (units). All men aged 40 or older in 2-3 units from 45 representative surveillance points were included in this cohort study, starting in 1990-1. Mortality is monitored through official records. Smoker vs non-smoker relative risks were calculated, including that for "vascular" death (not shown in tables), which has a relative risk of 1.13 (95% CI 1.07 – 1.20) (Niu, Yang et al. 1998). Data from a range of other Chinese studies have been examined in a relatively recent review as mentioned later.

For all-cause mortality (Table 1), there is some variation in the relative risks presented. However, when grouped into similar time bands, variation of the point estimates is not great – at least among the "western" studies (note – statistical precision of these estimates is considered later in section 3.1.2.1, "random error", page 23). Among females for example, some of the more comparable recent estimates are 1.9 (CPS II), 1.86 and 1.87 (Nurses Health Study), and 1.9 and 2.1 (Kaiser Permanente). For males, there is slightly more variation among most of the recent data, with estimates from similar studies of 2.06 (2nd half British Doctors), 2.3 (CPS II), 2.2 (MRFIT) and 1.9 and 1.8 (Kaiser Permanente). And importantly, the CAPM study gives a low outlying estimate for males, 1.19 (95% CI 1.13-1.25), giving some indication that relative risk may be different in populations outside the USA and UK. The authors of the CAPM study speculate that the lower relative risk seen in China may be due to older men there not having smoked as persistently in the past – the main increase in tobacco consumption has occurred much later than countries such as the US and Britain – or that people may have smoked different forms of tobacco with a lower risk than cigarettes (Niu, Yang et al. 1998). It is stated that in urban areas of China, where a greater proportion of tobacco use involves cigarettes, the relative risk for

those who began smoking before age 20 is already approaching two (Niu, Yang et al. 1998).

The World Health Report 2002 also states "the relative risk for current tobacco smoking and heart disease appears to be less in the People's Republic of China than in North America and Europe, principally because of a shorter history of smoking among the Chinese." (WHO 2002)

For IHD, there appears to be a similar variation for males that is seen for all-cause mortality, with a range of relative risk point estimates from 1.75 to 2.3 for the more recent studies. There is a wider variation for females, from 1.6 to 4.3, with the Nurses Health Study in particular giving much higher estimates of IHD mortality among women – 4.13 age adjusted (95% CI 3.04-5.63), and 4.3 multivariate (3.0-5.9). As previously noted, statistical precision of the point estimates is discussed later, however it should be highlighted here that even though the 95% confidence intervals for the Nurses Health Study are reasonably wide, the lower limits of the intervals are still higher than the upper limits from the other studies (ie. despite the imprecision of the estimates there still appears to be heterogeneity as the confidence intervals are non-overlapping).

Recent stroke estimates range from 1.7 to 2.5 for males, and 1.8 to 2.58 for females.

It is important to note some particular features of these data that suggest population specific estimates (such as country and time) may be necessary.

Firstly the relative risk estimate for male all-cause mortality in China is considerably lower than the other recent studies. This finding is not corroborated by a review by He and Lam (1999), which examined published data from 13 cross-sectional, 16 case-control, and 13 prospective cohort studies from China and Hong Kong. The Mantel-Haenszel pooled relative risk for IHD from 13 prospective studies was 1.86 (95% CI 1.40 – 2.48) in men and 3.45 (1.78 – 6.67) in women. However, the confidence intervals for the pooled estimates are wide, many of the individual studies had markedly imprecise estimates due to small sizes of the cohorts, and there were other methodological differences between the studies. The authors report that "the results should only be seen as an indication of the

effect of the early stage of the epidemic in China." In contrast, a large retrospective proportional mortality study of one million deaths in China did find similarly low relative risks to the CAPM study (Liu, Peto et al. 1998). Exposure information was obtained on the "participants" – who died during 1986-88 in 98 areas of China – from interviewing surviving family members during 1989-91 (note - possible bias). Outcome data were collected from official health records and interviews with health professionals and families. Age-standardised relative risks (smoker vs non-smoker) for all-cause mortality were 1.23 (Standard Error 0.01) for men aged 35-69 and 1.23 (SE 0.03) for women aged 35-69. The relative risks for IHD were 1.28 (SE 0.03) for men and 1.30 (SE 0.05) for women. For stroke, the values were 1.17 (SE 0.02) and 0.97 (SE 0.03). Even heavy smokers had relatively low relative risks, for example the IHD and stroke estimates for male (aged 35-69) urban smokers of 20 or more cigarettes per day were 1.53 (SE 0.08) and 1.38 (SE 0.05) respectively.

Secondly, the range of IHD mortality relative risk estimates among females in Table 2 is noticeably wide, with values from the Nurses Health Study over four (although as previously mentioned the confidence intervals do not overlap). This increases the uncertainty as to where the "true" IHD relative risk for a population might be for this group.

Thirdly, it appears that time may be an important factor. More recent studies report higher relative risks than CPS I, and the first half of the British Doctors Study. This suggests that older estimates may be less appropriate or relevant to present-day populations.

If a wider range of studies and information is examined, including cardiovascular disease incidence (morbidity) as well as mortality data, the heterogeneity in relative risk estimates becomes even greater. A 1996 review by van de Mheen and Gunning-Schepers on the risks associated with smoking included 83 reports published in the international literature written in English before June 1992. The results showed a range of reported relative risks for a number of outcomes, including CHD and stroke. CHD relative risk ranged from 1.2 to 2.9 for males, and 1.0 to 3.0 for females. Stroke relative risk ranged from 1.1 to 3.7 for males and 1.5 to 5.8 for females. It is also interesting to note the extremely wide variation seen for lung cancer, which will partly contribute to the relative risk of all-cause mortality.

Male lung cancer estimates ranged from 2.5 to 134.5 and for females the range was 1.3 to 46.8. It is hard to know how much this variation is due to imprecision, as confidence intervals are not given. Hankey (1999) also reviewed studies pertaining to smoking and the occurrence of stroke, and found a range of relative risk estimates from two to four. Some studies other than those shown in the tables also show an increase in relative risk over time (USDHHS 2001a).

Table 1: Relative risk estimates of all-cause mortality from cohort studies for smokers compared to never-smokers

Study	Size of Cohort	Years	Length of Follow-up	Age	Size of Sub-group	Method	Male RR	Female RR			
							Current Smoker (95% CI)	Current Smoker (95% CI)	by level of exposure (No. cigs / day)		
									1-14	15-24	25+
Framingham (USA) (Freund at al 1993)	5,209	1948-1982 (approx)	34 years	45-64		Multivariate analysis	1.9 * (1.5-2.3)	1.8 * (1.4-2.3)			
				65-84		Multivariate analysis	1.6 * (1.3-2.0)	1.8 * (1.4-2.2)			
British Doctors Study (UK) † (Doll & Peto 1976; Doll et al 1980; Doll et al 1994)	40,633	1951-1971	20 years	20-85+ in 1951	34,439 male	Age Standardised	1.62				
		1971-1991	20 years	20-85+ in 1951	21,688 male	Age Standardised	2.06				
		1951-1973	22 years	20-85+ in 1951	6,194 female	Age Standardised			0.94	1.55	1.66
CPS I (USA) (Thun et al 1997a)	786,387	1959-1965	6 years	30-85+		Age Standardised	1.7 (1.7-1.8)	1.2 (1.2-1.3)			
CPS II (USA) (Thun et al 1997a; Thun et al 2000)	711,363	1982-1988	6 years	30-85+		Age Standardised	2.3 (2.3-2.4)	1.9 (1.9-2.0)			
MRFIT (USA) † (Kuller et al 1991; Ockene & Shaten 1991)	361,662	1973-1985 (approx)	10 years (average)	35-57		Multivariate analysis	2.2				
Nurses Health Study (USA) (Kawachi et al 1997)	121,700	1976-1988	12 years	30-55		Age Adjusted		1.86 (1.65-2.13)			
						Multivariate analysis †		1.87			
Kaiser Permanente (USA) (Friedman et al 1997)	60,838	1979-1987	6 years (average)	35+ (white)	14,759 male 20,565 female	Age Adjusted ‡	1.9 (1.5-2.3)	1.9 (1.5-2.3)			
				35+ (black)	5,702 male 9,428 female	Age Adjusted ‡	1.8 (1.4-2.5)	2.1 (1.5-2.8)			
CAPM (China) (Niu et al 1998)	224,500	1992 - 1995	4 years (still going)	40+ in 1991			1.19 (1.13-1.25)				

All-Cause RRs World Literature

† Confidence Interval not reported

‡ Mantel-Haenszel method, not standardisation

* Framingham - odds ratios (not relative risk) adjusted for age, systolic blood pressure, total serum cholesterol, glucose intolerance, and left ventricular hypertrophy by electrocardiogram

CPS II - full multivariate adjusted for age, race, education, marital status, occupation, fruit and vegetable consumption, and for CVD also aspirin, alcohol, BMI, physical activity, and fatty food consumption

MRFIT - adjusted for age, diastolic blood pressure, serum cholesterol level, and race

MRFIT - reference group 'nonsmoker' includes ex-smokers at first screen

Nurses Health Study - all-cause multivariate adjusted for age, follow-up period, parental history of MI before age 60, history of hypertension, diabetes, high cholesterol levels, BMI, past use of oral contraceptives, menopausal status, postmenopausal estrogen therapy, and age at starting smoking

Table 2: Relative risk estimates of IHD mortality from cohort studies for smokers compared to never-smokers

Study	Size of Cohort	Years	Length of Follow-up	Age	Size of Sub-group	Method	Male RR	Female RR			
							Current Smoker (95% CI)	Current Smoker (95% CI)	by level of exposure (No. cigs / day)		
									1-14	15-24	25+
British Doctors Study (UK) † (Doll & Peto 1976; Doll et al 1980; Doll et al 1994)	40,633	1951-1971	20 years	20-85+ in 1951	34,439 male	Age Standardised	1.55				
		1971-1991	20 years	20-85+ in 1951	21,688 male	Age Standardised	1.75				
		1951-1973	22 years	20-85+ in 1951	6,194 female	Age Standardised			0.96	2.20	2.12
CPS I (USA) (Thun et al 1997a)	786,387	1959-1965	6 years	30-85+		Age Standardised	1.7 (1.6-1.8)	1.4 (1.3-1.5)			
CPS II (USA) (Thun et al 1997a; Thun et al 2000)	711,363	1982-1988	6 years	30-85+		Age Standardised	1.9 (1.8 - 2.0)	1.8 (1.7-2.0)			
						Multivariate Analysis (age only)	2 (1.9-2.1)	2.1 (1.9-2.2)			
						Multivariate Analysis (full)	1.9 (1.8-2.1)	2.1 (2.0-2.3)			
MRFIT (USA) † (Kuller et al 1991; Ockene & Shaten 1991)	361,662	1973-1985 (approx)	10 years (average)	35-57		Multivariate analysis	2.3				
Nurses Health Study (USA) (Kawachi et al 1997)	121,700	1976-1988	12 years	30-55		Age Adjusted		4.13 (3.04-5.63)			
						Multivariate analysis		4.3 (3.0-5.9)			
Kaiser Permanente (USA) (Friedman et al 1997)	60,838	1979-1987	6 years (average)	35+ (white)	14,759 male 20,565 female	Age Adjusted ‡	2.2 (1.6-3.1)	1.6 (1.05-2.5)			

IHD RRs World Literature

† Confidence Interval not reported

‡ Mantel-Haenszel method, not standardisation

Framingham - odds ratios (not relative risk) adjusted for age, systolic blood pressure, total serum cholesterol, glucose intolerance, and left ventricular hypertrophy by electrocardiogram

CPS II - full multivariate adjusted for age, race, education, marital status, occupation, fruit and vegetable consumption, and for CVD also aspirin, alcohol, BMI, physical activity, and fatty food consumption

MRFIT - adjusted for age, diastolic blood pressure, serum cholesterol level, and race

MRFIT - reference group 'nonsmoker' includes ex-smokers at first screen

Nurses Health Study - IHD multivariate adjusted for age, follow-up period, parental history of MI before age 60, history of hypertension, diabetes, high cholesterol levels, BMI, past use of oral contraceptives, menopusal status, postmenopausal estrogen therapy, and daily number of cigarettes consumed

Table 3: Relative risk estimates of stroke mortality from cohort studies for smokers compared to never-smokers

Study	Size of Cohort	Years	Length of Follow-up	Age	Size of Sub-group	Method	Male RR Current Smoker (95% CI)	Female RR Current Smoker (95% CI)
British Doctors Study (UK) † (Doll & Peto 1976; Doll et al 1980; Doll et al 1994)	40,633	1951-1971	20 years	20-85+ in 1951	34,439 male	Age Standardised	1.29	
		1971-1991	20 years	20-85+ in 1951	21,688 male	Age Standardised	1.80	
CPS I (USA) (Thun et al 1997a)	786,387	1959-1965	6 years	30-85+		Age Standardised	1.3 (1.2-1.4)	1.2 (1.0-1.4)
CPS II (USA) (Thun et al 1997a; Thun et al 2000)	711,363	1982-1988	6 years	30-85+		Age Standardised	1.9 (1.6-2.2)	1.8 (1.6-2.1)
						Multivariate Analysis (age only)	2.1 (1.9-2.4)	2.3 (2.0-2.6)
						Multivariate Analysis (full)	1.7 (1.5-2.0)	2.2 (2.0-2.5)
MRFIT (USA) † (Kuller et al 1991; Ockene & Shaten 1991)	361,662	1973-1985 (approx)	10 years (average)	35-57		Multivariate analysis	2.5	
Nurses Health Study (USA) (Kawachi et al 1997)	121,700	1976-1988	12 years	30-55		Age Adjusted		2.58 (2.08-3.19)

Stroke RRs World Literature

† Confidence Interval not reported

CPS II - full multivariate adjusted for age, race, education, marital status, occupation, fruit and vegetable consumption, and for CVD also aspirin, alcohol, BMI, physical activity, and fatty food consumpt

MRFIT - adjusted for age, diastolic blood pressure, serum cholesterol level, and race

MRFIT - reference group 'nonsmoker' includes ex-smokers at first screen

Nurses Health Study - 'stroke' includes non-fatal stroke as well

Nurses Health Study - stroke multivariate adjusted for age, follow-up period, history of hypertension, diabetes, high cholesterol levels, BMI, past use of oral contrceptives, postmenopausal estrogen therapy, and age at starting smoking

2.1 Evidence for other exposures / diseases

Heterogeneity of relative risk is not only seen for cigarette smoking. For example, another review by Marang-van de Mheen and Gunning-Schepers (1998) found a range of published risk estimates from hypertension for men. The relative risks ranged from 1.45 to 2.77 for CHD, and 1.86 to 5.78 for stroke. The confidence intervals tended to overlap for the CHD estimates, as they also did for many of the stroke estimates, however the lowest stroke estimate 1.86 (95% CI 1.41-2.45) and the highest 5.78 (3.07-10.89), did not. Some of the reasons found for this variation are similar to those for smoking as discussed in the next section.

3 Reasons for heterogeneity of relative risk estimates

Reasons for the some of the differences in relative risk estimates have briefly been mentioned already. This section explores the issue further, looking at the two main reasons why published smoking relative risks could vary. Firstly, variation could be due to artefact, from differences in study methodology or design (therefore factors such as chance and systematic error come into play). Secondly, there may be real differences in relative risk, such that the true strength of the association is different in different populations.

3.1 Artefactual or observed variation

Variation in estimates may be wholly or partially due to properties of the study, rather than real differences in risk.

3.1.1 Basic differences in study design

Some of the heterogeneity in measured risk may be due to basic elements of the study, such as whether it is a cohort or case-control design (although most of the results considered above were from cohort studies), the latter producing odds ratios to indirectly

estimate the relative risk); or whether morbidity or mortality is measured. Case-control studies are prone to influences such as recall bias (may overestimate the association). However, case-control studies may give better estimates of the size of the current exposure-outcome association compared to some of the long-running cohort studies.

Mortality, as opposed to morbidity (disease incidence) captures a range of factors post onset of disease, including access to or compliance with treatment.

3.1.2 Study Methodology

There are also a range of other methodological differences between the studies that could give rise to heterogeneity of estimates, including inaccuracies that may reduce the internal validity of the study (and therefore produce erroneous results).

3.1.2.1 Random error

As illustrated in Table 1 to Table 3, studies vary in size and therefore statistical power or precision. Some of the variation could therefore be due to random error. For example, the female IHD risk given by the Kaiser Permanente study (Table 2) may in fact be closer to 2.5, and the risk from the Nurses Health Study may be closer to 3.0, which is much less difference than 1.6 versus 4.3.

However, while there is a degree of imprecision of some of the estimates, some of the studies, especially CPS II, are extremely precise (narrow confidence intervals). In addition, there are instances where the 95% confidence intervals do not overlap, suggesting statistically significant differences (ie. not merely due to random error). For example, this is seen when comparing the male all-cause mortality relative risk estimate from CPS II, 2.3 (95% CI 2.3-2.4), to the estimate from the CAPM study (China), 1.19 (1.13-1.25). In fact, the upper limit of the CAPM interval is smaller than all of the other lower limits of the male all-cause estimates shown in Table 1. This pattern is also seen for female IHD mortality in the Nurses Health Study (although in the opposite direction) where the lower limits of the two estimates shown in Table 2 (3.04 and 3.0) are both higher than the upper limits of all the other estimates.

3.1.2.2 Length of follow-up

Studies also vary in their length of follow-up, and this may have a bearing on all-cause mortality in particular, which includes diseases with a long latent period. The 1998 results from the CAPM study for example may have underestimated all-cause risk in China as it has only analysed four years worth of data. Short follow-up will be a problem if peoples' smoking status has been changing dramatically before study entry, and diseases with a relatively long latency (e.g. cancer) are the focus of study..

3.1.2.3 Misclassification and confounding

A likely factor contributing to variation in the observed relative risks is the way in which studies deal with measurement of exposure and outcome, and potential biases from this. Outcome measurement is perhaps less of an issue, as for example most of the studies shown in Table 1 to Table 3 used either the ICD9 or ICD8 classifications of IHD and stroke (with the same ICD codes), and all-cause mortality will not be affected by outcome misclassification (assuming comparable completeness of death registration).

Measurement of smoking exposure however is particularly important. There may be different rates of misclassification (between current, ex and never) across studies, including unmeasured differences in smoking cessation and recidivism over time. Some studies also compare current vs non-smokers (eg. MRFIT) so that the reference group actually includes ex-smokers, thereby biasing risk estimates towards 1.0. There may be differences between study populations in the duration of smoking, therefore different accumulated exposures, which are often not accounted for (but has been suggested for the lower relative risk in China (Niu, Yang et al. 1998; WHO 2002)). Similarly, many studies also do not stratify by level of smoking exposure (eg. cigarettes per day), and may in fact be measuring relative risks of different degrees of smoking – for which there is a known dose-response relationship (Doll, Peto et al. 1994). For example the participants in the Nurses Health Study (with a stressful occupation) may in general be heavier smokers than those in the Kaiser Permanente study. Surveys have also shown a range of cigarette consumption between countries. For example, in the 1980s the MONICA study (described later) found among 26 countries that the median number of cigarettes smoker per day (per smoker) ranged between 11 and 25 for males and between 5 and 21 for females (Keil and

Kuulasmaa 1989). The New Zealand part of the study (Auckland) gave values of 20 (males) and 15 (females), while the USA centre (Stanford) was 25 and 20. More recent comparisons of local and overseas data have found that New Zealand in 1995 appeared to rank with the four states in America with the lowest cigarette per day consumption (Laugesen and Swinburn 2000), and had just over half the total USA consumption rate per smoker per day (Laugesen 2000). In the same year New Zealand was second lowest of 21 OECD countries for cigarette consumption per smoker per day (Laugesen 2000; Laugesen and Swinburn 2000).

Studies may or may not have controlled for potential confounders, and differences in the prevalence and distribution of unmeasured confounders (and in any measurement error of confounders) may alter the observed relative risk. An example of this is illustrated in Table 1 and Table 2, where some studies have also undertaken multivariate analysis using a range of variables in addition to adjusting for age, and some have not. In addition, among those multivariate analyses, the number and type of variables differ, including the fact that CPS II is the only study in the table to control for markers of SEP. Nevertheless, from the studies shown that have performed both age-adjusted and multivariate analyses (CPS II and the Nurses Health Study), it does not appear that confounding plays a major role in producing these risk estimates – at least for confounding that has been measured.

3.1.2.4 Effect of age

Effect modification by a major variable such as age will also impact on the observed relative risk where studies differ in the way they are restricted or stratified. Smoking relative risk is known to change with age. Therefore the fact that studies measure risk in different age groups will alter the estimates given. For example, the fact that smoking-mortality relative risk estimates for IHD generally decrease with age (Doll and Peto 1976; Doll, Gray et al. 1980; Thun, Day-Lally et al. 1997a), may (partly) explain why the Nurses Health Study relative risk was higher compared to other studies with older study populations (Nurses Health Study participants were less than 56 years of age).

A very important, and usually overlooked, manifestation of errors by age is relative risk variation arising from the use of standard populations with different age structures across

studies. Many published studies (including those in Table 1 to Table 3) use different standard populations for direct standardisation analyses (although often the standard population is not stated). For example, mortality rates for the female British Doctors were standardised to the age structure of their male British counterparts (Doll, Gray et al. 1980), and the mortality rates for CPS I and II from the analysis by Thun et al (1997a) were standardised to the age structure of the combined CPS I and II population. If disease or mortality rates (and therefore rate ratios) are standardised to a standard population with a younger age structure, this will tend to weight results towards the relative risk for younger people. An example of this is seen in two different published papers, both using data from the CPS II study. As mentioned above, in the paper cited in Table 1 to Table 3 (Thun, Day-Lally et al. 1997a), mortality rates were standardised to the combined CPS I and CPS II study populations, whereas in a paper by Malarcher et al (2000) mortality rates were standardised to the 1986 US population. Although the age structure used in the latter paper was not given, the age structure of the combined CPS I and II groups appears to be older than would be expected of the national population, therefore implying that the Malarcher paper used a younger age structure. It seems probable that for this reason that Malarcher et al found higher age-adjusted relative risks in their analysis of white men – 2.68 (95% CI 2.43 – 2.96) for IHD and 2.97 (2.18 – 4.05) for stroke – compared to 1.9 for all men from Thun (1997a). These differences are more than trivial, given the same underlying data! The age range analysed was also slightly different but actually slightly older for Malarcher (35+ rather than 30+).

3.2 Real variation in relative risk

In addition to artefactual variation, there may be "real" differences in the relative risks between different populations. It should be noted however that there is a great deal of overlap between what could be considered "artefact" and a "real difference", and that a distinction between the two may be somewhat arbitrary. Many of the factors contributing to a "real" difference in relative risk (including those discussed below) can be thought of as just differences between populations which studies have not measured, either because it is currently impossible or it is not feasible or worthwhile to do so. This includes differences in the type of smoking exposure, genetics, social-structural factors (eg. affecting an entire country), and even the combined effects of all the different

permutations of variables that can actually be measured individually (plus the ones that can't).

Examples of the type of smoking exposure that are often not measured, or are too difficult to measure accurately, include the age of initiation of smoking or duration of smoking, past history of cigarette consumption, past and current smoking behaviour (eg. how much of cigarette smoked), and type of tobacco or cigarettes used (discussed later). Differences in these factors will alter the real cumulative or current exposure to cigarette smoke and therefore will contribute to heterogeneity of the observed effect of smoking. Much of the increase in relative risks over time has been attributed to the greater cumulative exposure among smokers in later studies, particularly among women, and the long latency of some health effects such as cancer (USDHHS 1990).

It is worth considering some of these factors from the point of view that studies with exactly the same methodology, taking into account a reasonable range of influences (and measuring all confounding influences), may still demonstrate different relative risks when conducted in different populations. In particular, one of the hypotheses of this thesis is that the relative strength of the association between smoking and mortality in the New Zealand population, both as a whole and for specific groups within it, is different from overseas relative risks. The following discussion outlines an argument for why this might be the case, including explanations based on statistical interaction (which is equivalent to effect measure modification) and biological interaction.

Reiterating the point made earlier, these explanations predominantly focus on reasons for heterogeneity of *relative* risks as a measure of effect.

3.2.1 Effect Measure Modification (Statistical Interaction)

The variation in effect measures seen between countries can be considered a form of effect measure modification, with the country as an effect modifier. Assuming that there is something about a country that has effects on health (other than smoking exposure), then the following "rule" applies: uniformity (absence of modification) with regard to either the difference measure or the ratio measure implies that the other measure must be

heterogeneous (modified) if both the potential effect modifier and the exposure have effects (Rothman and Greenland 1998).

This means that if there was homogeneity in one effect measure – eg. rate differences – between countries or studies, then there must naturally be heterogeneity of the other effect measure – eg. rate ratios; assuming that the underlying mortality rates vary to some degree by country. It would therefore be a brave assumption that we should in fact see homogeneity of relative risks (such as rate ratios) all the time. It should be noted that heterogeneity of both effect measures is likely.

While it is not possible to compare all the studies presented inTable 1 to Table 3 (often only rate ratios are given), the CPS II study shows an example of this "truism". Between CPS I and CPS II, the underlying mortality rates have changed, so that in this case "time" has an effect on mortality (as would "country" – with global variation in underlying mortality rates). However, for male all-cause mortality, the rate differences between current smokers and never smokers are very similar – 1,168 deaths per 100,000 person-years in CPS I, and 1,162 in CPS II (Thun, Day-Lally et al. 1997a). As a result, the rate ratios between CPS I and II for this stratum have increased (from 1.7 to 2.3) over the two "levels" of time.

The relative risk has been therefore been questioned as an appropriate measure of effect, as it will vary simply because baseline mortality rates vary, even if the absolute effect (rate difference) is the same (Prescott, Osler et al. 1998). However, as mentioned previously, this thesis (including the new cohort studies) does focus on relative risks as they are the most commonly reported measure of effect, and they are used for informing policy (eg. through population attributable risk calculations). Nevertheless, rate differences are also considered in the new studies presented later.

3.2.1.1 Risk factors as effect modifiers

Some modification of the size of the smoking-disease (or mortality) relative risk association by the level of other risk factors is described below. These examples show the potential for smoking relative risk to vary depending on the levels of the effect modifiers

in the population of interest. (Many of these examples illustrate sub-multiplicative interaction, whereby the relative risks decrease with a worsening profile of other risk factors).

From the Framingham study, effect modification of the smoker – non-smoker relative risk for cardiovascular disease was seen with the presence of glucose intolerance, high serum cholesterol, and high systolic blood pressure (Castelli and Anderson 1986). The smoking relative risk was less within strata of adverse glucose tolerance, cholesterol and blood pressure, and the presence of all three made an even greater impression. For example, among those participants without left ventricular hypertrophy (LVH), and with low levels of these factors, the relative risk of cardiovascular disease comparing smokers to non-smokers was 1.68. However, among people with all three factors present or at the highest level (but LVH absent), the smoker – non-smoker relative risk was 1.29. That is, the smoking relative risk was modified by the levels of other known cardiovascular disease risk factors.

Among the men screened for the MRFIT study, cardiovascular mortality rate ratios for smokers compared to non-smokers varied depending on the presence or absence of diabetes mellitus. For example, the rate ratio of mortality for heavy smokers (26 or more cigarettes per day) compared to non-smokers among those men without diabetes was 2.65. The same rate ratio among diabetic men was 1.8.

The presence of hypertension, hypercholesterolaemia and diabetes were also shown to be effect modifiers in the Nurses Health Study, with the presence of each lowering the relative risk of current smokers compared to never smokers (for fatal CHD and non-fatal myocardial infarction, MI, combined) (Willett, Green et al. 1987). For example, the relative risk of 'CHD mortality or non-fatal MI' for "light" smokers (1-14 per day) compared to never smokers was 2.8 in women without hypertension, and 1.4 in women with hypertension. For women who smoked 25 or more cigarettes per day the rate ratios were 8.6 (normotensive) compared with 2.8 (hypertensive).

3.2.2 Biological Interaction

Biological interaction is not the same as statistical interaction (although they are often confused). Nevertheless, they are not mutually exclusive, and the biological models can often help explain at a disease mechanism level why we see effect measure modification within the published data (reasons behind the effect modification) (Rothman and Greenland 1998).

Two of the common models for describing biological interaction are the counterfactual model and the sufficient cause model ("causal pies") (Rothman and Greenland 1998).

The "counterfactual model" application to interaction is complex. A description of its application is beyond the scope of this thesis (see Rothman and Greenland (1998) for a description). However, there is an important and intriguing deduction from the counterfactual model. Namely, the absence of biological interactions between two variables implies that the rate difference for the two variables are homogeneous (or the same) by stratum of the other variable. On the other hand, homogeneity of the rate ratios is consistent with biological interaction.

For the purposes of this thesis, though, the sufficient cause model will be presented.

3.2.2.1 Sufficient cause model (causal pies)

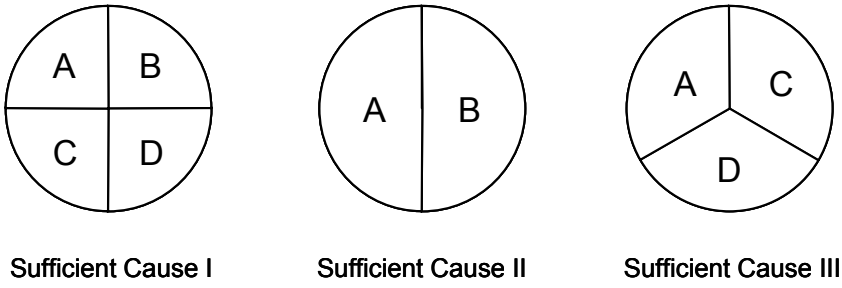
This model also helps to describe possible reasons behind heterogeneity of effects, however it may be difficult to make direct connections from this theory to relative risk or rate ratios.

The essence of this theory is that health outcomes, such as cardiovascular disease, can be produced from combinations of factors (component causes) coming together in different ways, with some combinations leading to disease (a sufficient cause) and some not (Rothman 1976; Rothman and Greenland 1998). This is illustrated in Figure 1, which shows three possible combinations (sufficient causes, or pies) of risk factors (component causes) that will produce a hypothetical disease. In this figure, 'A' represents a necessary cause as it is present in all three pies. For the outcome of "cardiovascular disease", the

letters ‘A’, ‘B’, ‘C’ and ‘D’ could potentially be replaced with ‘genetic susceptibility’, ‘smoking’, ‘poor diet’ and ‘sedentary lifestyle’ respectively.

In this model, biological interaction between two or more component causes means that the causes participate in the same sufficient cause (Rothman and Greenland 1998). For example, if some cases of disease require both component causes (in the absence of either one of the component causes these cases would not occur), this co-participation in a sufficient cause is termed “synergism”. Other cases of disease may require the presence of one component cause and the absence of another in the same “pie” – this is termed “antagonism”.

Figure 1: Rothman’s model of causal pies (adapted from Rothman 1976)



This model demonstrates the importance of other risk factors within a population in determining the “strength” of a component cause such as smoking. It leads Rothman to argue that the terms “strong” or “weak” with regards to a risk factor have no universal basis (Rothman 1976), as the size of an effect is dependent on the distribution of other component causes (within the same sufficient cause) in the population of interest. Considering those sufficient causes that contain the factor of interest, such as smoking; if a large number of these “pies” are completed (thereby leading to disease) because of the abundance of other component causes, then smoking will appear to be a “strong” risk factor.

It was initially suggested by Rothman in 1976 that these mechanisms could lead to a change in observed relative risk in a directly proportional manner – ie. if the strength of

effect increases, this could be observed as an increase in the relative risk estimate (more prevalent component causes leads to an increased relative risk). From more recent discussion on the subject (Rothman and Greenland 1998), it appears that this theory applies more strongly to strength of risk as it might be measured in attributable burden terms – eg. total mortality in the population for which smoking can be attributed as a cause. This makes sense, as with more sufficient causes filled, there are more "pies" with smoking giving rise to cases of disease, therefore a greater part of population mortality overall appears to have smoking as a component cause.

It seems more difficult to translate these causal pies into predictions or explanations of relative risk, even though Rothman states that "strength" could be measured in relative or absolute terms. The effects (on the relative risk) of changing the prevalence of component causes may be inconsistent. For example, increasing two component causes in the population in addition to smoking will increase the number of completed sufficient causes, but generally we do not know the underlying combinations of causes (types of pies) that prevail in that population. It may be that more pies are filled up that do not include smoking compared to the ones that do. In other words mortality rates for never smokers and smokers will both increase (so smoking is having a "stronger" absolute effect), but those for never smokers will increase more than smokers on a relative scale – and the relative risk will decrease. This example tends to fit with the empirical evidence of effect modification as previously discussed.

This model also explains the fact that not every smoker will develop a disease that is known to be associated with smoking, as disease will only develop in those smokers that are exposed to all the component causes required to complete a sufficient cause (Hallqvist, Ahlbom et al. 1996).

Finally, while heuristically useful, the causal pie model has been superceded by the counterfactual model for a complete understanding of interaction. However, one strong implication from the causal pie model, as well as from the empirical evidence of effect measure modification, is that if study populations differ in their levels of risk factors there is every reason to expect effect measures for smoking to vary across those populations.

3.2.3 Risk factor variation

The last two sections, describing the causal pie theory and effect measure modification, illustrate the importance of the prevalence and distribution of risk factors other than smoking in determining the strength of the effect from smoking. The empirical evidence for effect measure modification especially demonstrates the influence this could have directly on relative risk estimates. Differences in the distribution or prevalence of these risk factors between populations, including New Zealand, may therefore contribute significantly to heterogeneity of "real" relative risks from smoking – at least for cardiovascular disease.

In this section, the evidence for substantial variation between populations in risk factors other than smoking is reviewed, further establishing the case for effect measure modification of smoking relative risk.

A review of cardiovascular risk factors in France and Britain gave values for animal fat consumption and alcohol consumption (derived from United Nations data) in 20 countries (Law and Wald 1999) – with a wide range for both. Animal fat consumption (as a percentage of total energy intake) varied from 11.9% to 36.4% among the 20 countries in 1988 – New Zealand was 29.7%, and the United States 22.8%. And in the same year alcohol consumption varied from 3.5 litres ethanol per person to 13.1 – New Zealand was 9.6 and the US was 7.2.

A larger dataset of risk factors worldwide comes from the WHO MONICA project (Multinational Monitoring of Trends and Determinants in Cardiovascular Disease), which has obtained information on cardiovascular and cerebrovascular determinants from cross-sectional surveys in 26 countries, with 39 collaborating centres in total (Keil and Kuulasmaa 1989). From data collected around the period 1982 to 1987 (mostly), a large variation in risk factor levels is seen among the MONICA populations, as described below. Figures from Auckland (the New Zealand centre) and Stanford (the United States centre) are also given for comparison.

Median total cholesterol (mmol/L) in the MONICA populations ranged from 4.1 to 6.4 in men (Auckland 5.7, Stanford 5.3), and from 4.2 to 6.3 in women (Auckland 5.7, Stanford

5.2) (Keil and Kuulasmaa 1989). The percentage of the population with "high cholesterol" (defined as total serum cholesterol 6.5 or greater) ranged from 1% to 50% in men (Auckland 22%, USA not given), and from 2% to 46% in women (Auckland 23%, USA not given) (Anonymous 1994).

The prevalence of hypertension ranged between 8.4% and 45.3% in men (Auckland 20.2%, Stanford 23.5%), and between 12.6% and 40.5% in women (Auckland 18.2%, Stanford 16.7%) (Keil and Kuulasmaa 1989).

Large diversity was also found in the combination of risk factors. The proportions of three risk factors present (hypertension, high cholesterol and smoking) in the MONICA populations varied from 0.3% to 9.1% in men (Auckland 2.3%, Stanford 2.2%), and from 0.1% to 5.4% in women (Auckland 0.8%, Stanford 1.0%) (Keil and Kuulasmaa 1989). The proportions of the populations with no risk factors present varied from 14 to 43% in men, and 22 to 63% in women (Anonymous 1988).

Between population groups within New Zealand, there are also differences in risk factors levels, suggesting that we may see heterogeneity of effect measures in the same country. For example the 1996-97 New Zealand Health Survey found that Māori were more likely to have lower levels of physical activity, have hypertension or diabetes, report a hazardous pattern of drinking, and have a combination of these risk factors (as well as smoking) compared to European/Päkehä people (Sarfati, Scott et al. 1999; Sarfati and Scott 2000). Other New Zealand studies have also shown differences by ethnicity in rates of cardiovascular risk factors, including obesity and fruit and vegetable consumption (Dryson, Metcalf et al. 1992; Bullen, Tipene-Leach et al. 1996; Ministry of Health 2002b).

Cross-sectional comparisons such as these do not take into account temporal factors, such as how long individuals in the populations have been or are exposed to risk, and what trends in risk factor prevalence have occurred over time (including different combinations of "component causes"). This is particularly important given suggestions, and some supporting evidence, that there is a time lag between changes in risk factor levels in a population and changes in cardiovascular disease levels (Williams 1989; Law and Wald 1999). Law and Wald's ecological comparison also looked at past risk factor levels and

found a much stronger correlation with mortality from heart disease from these than was found for more recent levels.

It is therefore important to note that time trends in risk factor levels also show considerable variation between countries. In New Zealand, there has been a reduction in the consumption of saturated animal fats in our diet, with a corresponding increase in vegetable fats, since the late 1960's (Beaglehole, Dobson et al. 1989; Epstein 1989), and reductions are also seen in the US, Australia, and Canada. However, starting levels and the patterns of change over time (including rate of change) are different even amongst these populations (Epstein 1989). For example, Australia and Canada have had steeper declines in animal fat consumption than New Zealand (Epstein 1989), and reductions in this country may have plateaued in the 1980s (Jackson, Beaglehole et al. 1990). Conversely, some countries, such as Japan, Belgium and Finland have had large increases in animal fat consumption over this time. It was also noted by Epstein (1989) that despite dietary changes over time, New Zealand still has a relatively small proportion of its total fat intake from vegetable origins compared to other countries.

There are likely to be more risk factor differences geographically and over time than mentioned here. For example, any significant differences in oral contraceptive use among women smokers – another effect modifier of the smoking-cardiovascular disease association (USDHHS 2001a) – including type of pill and length of use, may have an important impact on relative risk estimates.

3.2.4 Chemical constituents of cigarettes and cigarette smoke

Another factor that may have a significant influence on the heterogeneity of relative risk of mortality from smoking is any variation in the chemical composition of cigarettes and the smoke they produce. The most commonly assessed components of tobacco smoke appear to be tar, carbon monoxide, and nicotine yields as measured by machine smoking, and the levels of each have the potential to increase risk of disease.

Tar is defined as the nicotine-free, dry, particulate mass of tobacco smoke (Fowles and Bates 2000) and contains numerous toxic chemicals, including carcinogens such as

dioxins, metals and nitrosamines (Fowles and Bates 2000). There is good epidemiological evidence for an association between reduced tar yields and the risk of lung cancer, and also a possible link with cardiovascular disease and stroke (Blakely and Bates 1998; Thun and Burns 2001; Sauer, Berlin et al. 2002 Feb 11).

Carbon Monoxide, which is found in the gaseous phase of tobacco smoke and does not necessarily correlate with tar yields (some other gaseous chemicals do, eg. benzene), can reduce the oxygen carrying capacity of blood (by forming carboxyhaemoglobin), thereby increasing the risk of myocardial and cerebral ischaemia (Fowles and Bates 2000).

Levels of nicotine, the main addictive substance in tobacco, also play an important role as they can determine how much tobacco smoke a smoker will endeavour to inhale from each cigarette. This is illustrated by the phenomenon of "compensatory smoking" whereby smokers will inhale more smoke (eg. by blocking ventilation holes, varying frequency and volume of puffs) from cigarettes with reduced nicotine concentrations (Blakely and Bates 1998; Thun and Burns 2001). Intense smoking has been shown to deliver more harmful chemicals than the standard ISO yield tests (Fowles 2003).

Research suggests that levels of these components vary by country and brand, and have changed over time.

An analysis of 32 brands of cigarette in America, 23 brands in Canada and 37 brands in the UK in 1998 shows some differences in smoke yields and nicotine content of tobacco, although the mean values for each country do not appear to be statistically significantly different (Kozlowski, Mehta et al. 1998). The mean tar yields (mg) were 8.8 for America, 9.8 for Canada and 9.1 for the UK. The mean nicotine yields (mg) were 0.67, 0.96 and 0.78 respectively, and the mean total nicotine content (mg) was 10.2, 13.5, and 12.5. The mean carbon monoxide yields (mg) were 9.6, 10.1, and 10.3. Nevertheless, there was marked brand variation in the levels of these components within each country (eg. 1 to 17 mg for tar yield in America), the tar to nicotine ratio, and the maximum tar yields – 17mg in America, 16mg in Canada, and 13 mg in the UK.

The usual indicators have however been described as a crude measure of cigarette toxicity, and may only partially reveal differences in their potential for harm. For example, hydrogen cyanide and arsenic have also been isolated from cigarette smoke at levels that could be hazardous to the cardiovascular system (Fowles and Bates 2000). And the nature of "tar" with regards to its toxic constituents varies widely between different types and sources of tobacco (Fowles and Bates 2000). Some other cigarette components influence the level of absorption of toxic chemicals, such as ammonia (increases smoke ph and facilitates nicotine absorption), and menthol (which increases the tolerability of smoke by numbing sensory nerve endings) (Fowles and Bates 2000).

A recent report published by ESR that includes New Zealand data takes into account some of these factors. Two New Zealand brands of cigarettes were tested (2000 cigarettes in total), one of which – Holiday Extra Mild (HEM), which has the largest market share of the "mild, extra mild, or light" brands – was compared to "mild" cigarettes from Australia (13 brands) and Canada (10 brands) (Fowles 2003). There were a number of differences in the yields of individual components, including tar, nicotine, carbon monoxide, cyanide, and ammonia, as well as differences in composite indexes of toxicity. For example the tar to nicotine ratio in the New Zealand HEM brand (14.08) was significantly higher than the "mild" and "light" brands tested from Australia (10.40) and Canada (9.26). There were also differences in the cardiovascular index (a function of hydrogen cyanide, arsenic and carbon monoxide levels) – 1.6 in HEM, and 1.2 in the Australian and Canadian brands – as well as the cardiovascular index to nicotine ratio – 2.5 for HEM, 1.67 for Australia, and 1.41 for Canada (Fowles 2003). ASH New Zealand has also reported a comparison with 66 mild brands in Canada and the UK, with HEM giving the highest tar to nicotine ratio of the 66 (ASH 2003).

As per the previous discussion on cardiovascular risk factors, a "snapshot" of cigarette toxins at any one moment also does not tell the full story. Different patterns over time of the levels of cigarette constituents will also impact on risk. For example, the tar and nicotine yields of cigarettes, at least those measured by standard machine smoking tests, have markedly decreased over much of the twentieth century in both the USA (USDHHS 1989) and the UK (Jarvis 2001 Dec). The sales-weighted mean tar and nicotine yields (mg/cigarette) of UK manufactured cigarettes decreased from 16.0 and 1.28 respectively

in 1980, to 9.6 and 0.79 in 1999 (Jarvis 2001 Dec). By comparison, the tar and nicotine yields of the five most popular brands in New Zealand have changed little over the same period (Laugesen 2000). The tar yield of these brands was between 14 and 15 in both 1980 and 1999. The nicotine yield was between 1.2 and 1.4 in 1980, and was 1.3 for all brands in 1999. In 1999, the New Zealand sales-weighted average for tar was 12.4mg, and for nicotine 1.1mg (Laugesen 2000).

There are still many other potentially toxic components of cigarette smoke that have not been measured, and could contribute to risk heterogeneity. Fowles and Bates (2000) note that the number of chemical constituents of tobacco smoke as been estimated at over 4000, of which there exists significant data for less than 100. Also, differences in compensatory smoking behaviour between countries and over time will alter the level of toxins delivered to the smoker.

4 New Zealand risk estimates

The discussion in all the previous sections of this chapter strongly leads to the conclusion that it is not possible to be certain of the relative risk of mortality from smoking in the New Zealand population – relative risk is affected by many variables. Therefore, New Zealand-specific estimates ideally need to be calculated rather than "borrowing" data from overseas studies such as CPS II. Four published studies that were found in the literature search have made some measurement of effect in the New Zealand population, although only one of these uses mortality as the outcome of interest (another includes coronary death as a sub-category). All four have some deficiencies, and cannot be relied upon as precise or generalisable.

The first is a case-control study, conducted by the University of Auckland, which examined a 50% random sample of new episodes of stroke (incidence rather than mortality) in Auckland in the year ending 1 March 1982 (Bonita et al. 1986). Analysis was restricted to people aged 35-64, and included 132 cases (from a cardiovascular disease register), and 1586 controls (from an electoral roll-based survey). With regards to smoking exposure, current cigarette smokers were compared with non-smokers (the latter included

ex-smokers). The odds ratios for current smoking and stroke were 3.1 (95% CI 2.0 – 4.9) for men, 2.6 (95% CI 1.4 – 4.6) for women, and 2.9 (95% CI 2.0 – 4.1) for both sexes combined. Ethnicity of the participants was not reported in the paper.

The second study, also conducted by the University of Auckland, followed a cohort of 1,029 "European" Auckland men, aged 35 to 64 at entry, that were part of the Auckland risk factor study in 1982 (Norrish, North et al. 1995). Smoking status was linked to all-cause mortality up to 1991, with 96 deaths recorded. Relative risks were calculated from nine-year incidence rates, and Cox proportional hazards models were used to control for potential confounders. The all-cause mortality current smoker / never smoker relative risk estimate adjusted for age only was 2.01 (95% CI 1.15 – 3.53). Adjusted for age, BMI, socio-economic status (using three levels of the UK Registrar-General classification of social class) and alcohol, the relative risk estimate was 1.89 (95% CI 1.06 – 3.39).

The third study is a population-based case-control study conducted as part of the WHO MONICA project (McElduff, Dobson et al. 1998). It recorded cases of a "major coronary event" during 1986-88 or 1992 among non-Māori non-Pacific people in Auckland aged 35-69, as well as during 1987-94 in Newcastle, Australia. The total number of cases (both cities) was 5,572 and the number of controls was 6,268 (numbers for each city are not given). Multivariate odds ratios (adjusted for age, sex, education, body mass index, and history of coronary heart disease, diabetes and hypertension) for coronary death in Auckland current smokers (compared to never-smokers) were 3.0 (95% CI 2.1 – 4.1) for men and 5.0 (95% CI 2.8 – 8.9) in women.

A case-control study was also conducted in Auckland looking at the relative risk of stroke (incidence again, not mortality) from smoking among non-Māori non-Pacific people (Bonita, Duncan et al. 1999). This was based on the Auckland stroke study, which documented all stroke events in residents of the Auckland population aged 15 years and over during 1991-92. The analysis included 521 cases and 1851 community controls aged 35-74 years. Odds ratios for active smoking, adjusted for age (using the Cochrane-Mantel-Haenszel method), were 4.07 for men (95% CI 2.74 – 6.04) and 4.50 for women (95% CI 3.03 – 6.69).

While all these studies provide useful information, they also have limitations. Firstly, they provide reasonably imprecise estimates, as illustrated by the width of the 95% confidence intervals. Secondly, and more significantly, they either exclude Māori and Pacific people, or in the case of the first study by Bonita et al. (1986) ethnicity is not mentioned. It cannot be presumed that relative risk from smoking for all ethnic groups is the same (for reasons described in the earlier sections). It is important to note that other previous New Zealand studies have also restricted by ethnicity in the same way (although there are also many examples where this is not the case). The Auckland Risk Factor Study as a whole (Jackson, Beaglehole et al. 1990) did not include Māori or Pacific people, and the Auckland University Heart and Health study (a cross-sectional survey of cardiovascular risk factors 1993-94) also excluded Māori and Pacific (Bullen, Simmons et al. 1998).

5 New Zealand Ethnicity Specific Data

It is important that epidemiological studies – such as the one presented in this thesis – in New Zealand provide estimates specific for different ethnic groups. Both a scientific ("needs-based") and a philosophical ("rights-based") argument can be made to support this proposition. While this may not be feasible, or at least accurate, for all ethnic groups, the most useful breakdown for research and policy purposes is for Māori, Pacific and non-Māori non-Pacific.

5.1 Needs based rationale

It is well known now within the New Zealand health sector that Māori and Pacific peoples have poorer health for a wide range of outcomes, and lower life expectancy on average, than non-Māori non-Pacific people. To reduce these health inequalities, there is a need for adequate information, and arguably more information, for Māori and Pacific to help inform research and evidence-based policy, particularly in epidemiology and public health.

In addition, there are significant disparities in health determinants by ethnicity, some of which have already been mentioned. These include "lifestyle" factors (eg. behavioural

factors), socio-economic status, and access to health services (Sarfati, Scott et al. 1999; Howden-Chapman and Tobias 2000; Reid, Robson et al. 2000; Westbrooke, Baxter et al. 2000; Tukuitonga and Bindman 2002; Ministry of Health 2002b; Ministry of Health 2002c). There is also likely to be a degree of racism (personal, institutional, and internalised) that impacts detrimentally on the health of Māori and Pacific (Reid, Robson et al. 2000; Ministry of Health 2002c) subsequent to New Zealand's colonial history. Given the examples previously described of effect measure modification, it can be hypothesised that some of these differences in causal factors may lead to relative risks of smoking mortality that are higher or lower than other ethnic groups. The possibility of this variation increases the importance of calculating ethnicity-specific effect measures.

5.2 Rights based rationale

This argument relates to Māori as tangata whenua and treaty partners. Both the Treaty of Waitangi, and a number of international conventions and covenants on the rights of indigenous people, provide researchers – in particular those that receive crown funding – and government departments (such as the Ministry of Health and Statistics New Zealand) with obligations to meet the statistical needs and rights of Māori.

It has been noted however by Robson and Reid (2001), that official government statistics often seek to meet the statistical needs of the New Zealand population only as a whole, among which Māori are subsumed rather than given at least equal credence. In addition, small studies that actually do give ethnicity specific data may only sample at the same proportions as the total population, which often leads to far less precise measures for Māori (Robson 2002). A lack of the same degree of statistical information for Māori makes it difficult to fully understand all the determinants of health disparities, let alone formulate and implement strategies to reduce them.

Robson and Reid (2001) make the point that "the full expression of tino rangatiratanga positions Māori statistical needs as being equally as valid as those of the total population." Without such an emphasis, not only is article two not fully met, but the crown is also unable to meet both its article one obligation of governance for all peoples, and its article

three obligation of equal rights and privileges. Fully appreciating such obligations would assist in the protection and promotion of hauora Māori.

Chapter 3: Methods

Methods Summary

This thesis is based on two full population cohort studies, conducted as part of the New Zealand Census-Mortality Study (NZCMS). It utilises data from the entire New Zealand census population in 1981-84 and in 1996-99, aged between 25 and 74 years. It calculates mortality incident rates by smoking status (deaths per 100,000 person-years) for all-cause mortality, Ischaemic Heart Disease (IHD), and Cerebrovascular Disease (Stroke) over these two 3-year periods. These rates are also presented stratified by age, sex, and ethnicity. Comparisons of rates between smoking strata gives two measures of association between smoking and mortality – rate ratios and rate differences.

This chapter outlines the methodology used to calculate New Zealand-specific effect measures for cigarette smoking. The results and discussion from this analysis (Chapters four to eight) comprise the main part of this thesis.

This chapter is structured in the following way:

1 & 2	A summary of the cohorts used in this thesis, and the record linkage methodology used in the NZCMS
3	A description of the variables measured for exposure, outcomes and co-variates
4, 5 & 6	A description of the methods used for the part 1 (direct standardisation) analyses, including considerations of random and systematic error
7	A description of the methods used for the part 2 (multivariable) analyses
8	A description of the methodology of the (brief) sensitivity analysis

1 Data source – the NZCMS

The methodology of the NZCMS is described in detail elsewhere (Blakely, Salmond et al. 1999; Blakely, Salmond et al. 2000; Blakely 2002; Hill, Atkinson et al. 2002). Essentially, it is a cohort study that matches New Zealand census records of residents (aged 74 or less) in 1981, 1986, 1991 and 1996 to mortality records for the three years post each census, using anonymous probabilistic record linkage. This creates four linked datasets, with personal information (from the census) and mortality status of each individual in New Zealand over the periods 1981-1984, 1986-1989, 1991-1994 and 1996-1999. This thesis, a 'sub-study' of the NZCMS, uses two of these cohorts.

1.1 Record linkage

The detailed process of linking census records and mortality records is also described in depth elsewhere (Blakely, Salmond et al. 1999; Blakely, Salmond et al. 2000; Blakely and Salmond 2002; Fawcett, Blakely et al. 2002; Hill, Atkinson et al. 2002), and a summary is presented here.

Individual census records (from Statistics New Zealand), and mortality records obtained for the three years post census (from the New Zealand Health Information Service (NZHIS), see section 3.2) were compared using a number of key matching variables, including date of birth, country of birth, sex, ethnicity, and (most importantly) address of usual residence (coded to meshblock or area unit level). This comparison is an iterative, probabilistic record linkage process using anonymous data (so cannot be matched on name), with a commercially available software package, Automatch (Version 4.2, MatchWare Technologies, 1998). When a mortality record was successfully matched to a census record (creating a "link") it was assumed that this individual in the study (census) population did die. The information from each source was combined into a single line listing. Those individuals for whom there was no match with a mortality record (no link) were assumed not to have died.

This linkage process therefore created a dataset of individuals (anonymised) with information on a range of demographic, socio-economic and other variables available from the census (eg. smoking), as well as mortality data for those people who are "linked".

The linkage process can be likened to a diagnostic test, hence can be described in terms such as sensitivity and positive predictive value (Blakely and Salmond 2002). The accuracy of the linkage process is quite high – at least 97% of links found in both the 1981 and 1996 cohorts were estimated to be true links (ie. the positive predictive value of record linkage to detect mortality outcome is greater than 97%).

However, the sensitivity of the anonymous and probabilistic matching process is somewhat lower. For the 1981-84 dataset, 71 % of mortality records were linked, and for the 1996-99 dataset 78 % of mortality records were successfully linked. Consequently, a number of records (ie. study participants) in these datasets would appear not to have died (unlinked) when in fact they have. To adjust for the potential resultant "linkage bias", a weighting was applied to the census cohort records – this is described later in more detail.

2 Study population

2.1 Cohorts used in analyses

The linked datasets, containing anonymous data only, were stored and analysed at Statistics New Zealand (SNZ), Wellington. Permission was granted to the author to use the SNZ datalab for analysis of these data. All the analyses were performed using SAS version 8.2. For the purposes of this thesis, the 1981-84 and 1996-99 datasets were used as they included the two censuses for which smoking information was recorded.

The primary – Part 1 – analysis of the data (mortality rates, rate ratios, and rate differences) was performed by age, sex, ethnicity and smoking status. Therefore it was important that the records used contained data on all these variables. Also the analysis was conducted on those people 25 years old or greater, and less then 75 years of age (ie 25-74 year olds inclusive) throughout the three-year follow-up period. This restriction was

because people under 25 years of age are unlikely to contribute a notable degree of mortality from smoking. For older people, the NZCMS linked datasets already exclude person-time of follow-up over the age of 78.

Therefore the 1981 and 1996 linked cohorts were restricted for the Part 1 analyses, and are referred to as the *first restriction*. Any records that had missing or "not specified" data for age, sex, ethnicity, and smoking status, and were outside the specified age range were excluded from the Part 1 analytic cohorts. Absentee records (those filled out by another person on behalf on someone away from the household) were also excluded, as a census form may also have been filled out by "the absentee" themselves, thereby creating a duplicate record for that person. Table 4 gives a summary description of the study populations for the Part 1 analyses / results.

Table 4: Part 1 Study Populations

1981	1996
Individuals in New Zealand on census night 1981, aged 25-74 years during 1981-1984 *and* Complete data available for age, sex, ethnicity, and smoking status	Individuals in New Zealand on census night 1996, aged 25-74 years during 1996-1999 *and* Complete data available for age, sex, ethnicity, and smoking status

The ethnicity classification used was prioritised ethnicity, taken from self-identified ethnicity at census (see questions in Appendix A), categorised in three groupings: Māori, Pacific, non-Māori non-Pacific. Accordingly, if any self-identified ethnic group was Māori, then prioritised ethnicity was assigned as Māori (even if other ethnic groups were also selected, including Pacific). For those not allocated as Māori, if one of the self-identified ethnic groups was Pacific then the assigned ethnicity was Pacific. The remainder were assigned as non-Māori non-Pacific.

For the purposes of presenting results, age was grouped into 25-44 years (inclusive), 45-64 and 65-74, and also the complete group 25-74 years.

Part 2 analyses involved poisson regression using multiple potential confounding variables (ie. multivariable analysis); therefore it was necessary to ensure that the cohort had complete data for all co-variates. Records were excluded that were missing data for particular variables, in addition to those missing for the first restriction, as listed in Table 5. The new variables were primarily indicators of socio-economic position, and it should be noted that for the purposes of this thesis marital status is included within this term. These datasets for 1981 and 1996 are referred to as the *second restriction*, and are a subset of the first restriction.

Table 5: Part 2 Study Populations

1981	1996
Individuals in New Zealand at usual residence, and at private dwelling, on census night 1981, aged 25-74 years during 1981-1984 *and* Complete data available for sex, ethnicity, smoking status, education, motor vehicle, housing tenure, income, labour force status, marital status, NZ deprivation (NZDep) scale	Individuals in New Zealand at usual residence, and at private dwelling, on census night 1996, aged 25-74 years during 1996-1999 *and* Complete data available for sex, ethnicity, smoking status, education, motor vehicle, housing tenure, income, labour force status, marital status, NZ deprivation (NZDep) scale

As an indication of the number of study participants in each analytic cohort, the size of each cohort at the start of the two study periods – ie. census night 1981 and 1996 – was

calculated, and compared to the original cohort for the same age range. The age structure and prevalence of smoking by age, sex and ethnicity was also calculated for the first restricted cohort at the start of each study period. These findings are presented in Chapter 4.

3 Measurement of exposure, outcome and co-variates

3.1 Exposure – cigarette smoking

The exposure of interest in this study is cigarette smoking. In the linked NZCMS datasets, smoking status was obtained from the smoking questions in each census and measured only at the start of each study period – ie. on census night 1981 and 1996. The census smoking questions are shown in Appendix A. As defined by the nature of the questions, exposure or non-exposure was classified into three categories – current cigarette smokers, ex-smokers, and never-smokers (ie. persons who have never smoked during their lives). People in the first two categories are counted as "exposed" (separately, not combined into a single category), and the third is the non-exposed or reference group.

Two points should be noted. Firstly, the 1981 and 1996 census questions are not exactly the same, however the differences are unlikely to be enough to elicit a different choice of category. Secondly, although the 1981 census also included questions about level of cigarette consumption, this has not been analysed in this study. Such information could potentially be valuable as in reality smoking exposure is a continuous variable with a dose-response effect – in this study all levels have been grouped together. However, primarily due to comparability with the 1996 cohort and time constraints, this analysis for the 1981 cohort was not done.

3.2 Outcomes – all-cause, IHD, stroke mortality

The primary outcome of interest in this study is death. All-cause mortality, as well as cause-specific mortality from IHD and stroke is measured. Outcome information in the

linked datasets was derived from the mortality dataset used in the matching process. Within the mortality dataset or records, both the confirmation of death and cause of death were established by using information from a number of mortality files. In 1981 the files used were the Historical Mortality Data Set (held by NZHIS – this became the National Minimum Dataset after 1988) and the Statistics NZ Vitals File. In 1996 the files used were the National Minimum Dataset, the National Hospital Index Data Set (also held by NZHIS), and the Statistics NZ Vitals File.

For all-cause mortality, a death was defined by a successful match, or "link", between the mortality records (dataset) and census records. A sum of all the linked records gives the total deaths from all-causes.

The specific causes of death were grouped using the ICD-9 coding system. Deaths from IHD were defined as those from 410-414 inclusive, and deaths from stroke were defined as those coded 430-438 inclusive. The latter does include deaths from 'subarachnoid haemorrhage', and 'intracranial haemorrhage other than intracerebral haemorrhage', however both ICD groupings appear to be the standard definitions for IHD (or CHD) and stroke in cohort studies worldwide.

3.3 Co-Variates

Co-variates that were considered potential confounders or effect modifiers were measured or derived from census information (some of which have already been discussed). These were:

- **Age** – initially by five-year age bands
- **Sex** – male and female
- **Ethnicity** – three categories: Māori, Pacific, non-Māori non-Pacific.

and as markers of socio-economic position (see Hill et al 2002 NZCMS technical report for detailed information on these variables):

- **Income** – five levels (quintiles) of Household Equivalised Income derived from census income data using Jensen Index
- **Education** – three levels: no qualification; school qualification; postschool qualification
- **Motor Vehicle Ownership** – three levels: no car; one car; two or more cars
- **Labour Force Status** – three levels: employed; unemployed; not in labour force
- **Housing Tenure** – two levels: owned; rented or other
- **Marital Status** – three levels: never married; previously married; currently married (for 1996 was derived from both definitions available – legal and social)
- **NZDep** – five levels based on New Zealand deprivation 1996 scale (NZDep96)

As noted previously, for the purposes of this thesis marital status is included within "socio-economic position".

4 Part 1 analyses

The part 1 analyses were performed on the first restricted cohort, to produce standardised mortality rates, rate ratios and rate differences.

4.1 Mortality rates

The (weighted) number of deaths from all-causes, IHD and stroke were determined for each three-year period (1981-84 and 1996-99) within the first restricted cohort and stratified by smoking status, giving the number of deaths among smokers, ex-smokers and never smokers (see section 6.1.2 regarding weighting). These data were further stratified by age, sex and ethnicity (ie. within each strata of smoking status), and comprise the *numerators* for calculating mortality incidence rates. All-age and all-ethnicity strata were also used.

The *denominators* used in this study are person-time of follow-up. Each person who filled in the census form in 1981 and 1996 contributes time of observation in the study while they are aged 25 to 74 years (inclusive) over the subsequent three years. In other words, it

is an open cohort of 25-74 year olds. This means that people who were younger than 25 on census night, but turn 25 during the next three years, contribute person-time to the denominator (and deaths to the numerator if they die) after they turn 25. At the other end of the age range, people cease to contribute person-time and mortality data once they turn 75. With regards to the three age bands used – 25-44, 45-64, and 65-74 – the same rules apply. For example, someone who was 43 years of age on census night, will contribute person-time to the 25-44 age group until they turn 45, after which their time and outcome data will belong to the 45-64 age group.

The process of calculating person-time involved splitting the observation time for each person who crossed an age bracket, and creating a duplicate record with the subsequent time of observation and mortality information allocated to the next age bracket. Time of observation for each person ended when they died, turned 75, or reached three years of follow-up. Person-time denominators were determined by adding the time of observation for all records (original and duplicate) in each stratum for which a mortality rate was calculated. Calculations were performed in person-months of observation before later being expressed as person-years. All person-time denominators used for calculating the results presented in this thesis are shown in Appendix C.

Using these numerator (deaths) and denominator (person-time) data, crude (non-standardised) mortality incidence rates were calculated for each strata used in the standardisation process (see next section) as below:

$$\text{Crude Mortality Incidence Rate (deaths per 100,000 person-years)} = \frac{\text{Number of weighted deaths}}{\text{Person-time}}$$

All counts of deaths that are presented in this thesis have been random rounded to base three to preserve confidentiality. However, original analyses were conducted on non-rounded data at Statistics New Zealand.

4.1.1 Age and ethnicity standardisation

The crude mortality rates in five-year age bands were used to calculate age standardised rates; using the 1996 New Zealand population as the external standard. Mortality rates for the all-ethnicity combined strata were also standardised by ethnicity to the same population (labelled "adjusted for ethnicity"). This was done using the direct method as described in Rothman and Greenland (1998), with the age-specific and ethnicity-specific mortality rates weighted by the distribution of person-time in the standard population (NZ 1996).

4.2 Rate ratios and rate differences

The association (or effect) of interest in this study is between smoking status and mortality. The effect measure estimates calculated to demonstrate the strength of this association were mortality rate ratios and mortality rate differences, illustrating the relative risk and excess (absolute) mortality risk from smoking respectively. The term "excess rate ratio" is also sometimes used and is defined as the rate ratio minus one (RR-1).

The rate ratios and rate differences were calculated by comparing the standardised mortality rates of current smokers and ex-smokers, to that of never-smokers (reference group), within the age, sex and ethnicity strata, as illustrated below:

$$\text{Standardised Rate Ratio} = \frac{\text{Standardised Mortality Rate in Current (or Ex) Smokers}}{\text{Standardised Mortality Rate in Never-Smokers}}$$

$$\text{Standardised Rate Difference} = \text{Std Mort Rate in Current (or Ex) Smokers} - \text{Std Mort Rate in Never-Smokers}$$

These effect measures do not represent mortality rate comparisons between the sexes, age groups, or ethnicities.

5 Study precision – random error

To illustrate the precision of the results (ie. how much random error or chance may have contributed to the estimates), 95% confidence intervals were calculated for the standardised mortality rates, standardised rate ratios and standardised rate differences. This was done as per the methodology in Rothman and Greenland (1998).

5.1 Wald testing

Heterogeneity of effect estimates by ethnicity was observed in the results (presented later). To assess whether or not this heterogeneity was statistically significant (ie. not due to random error), Wald testing was conducted (as per Rothman and Greenland 1998, page 275-77) using two degrees of freedom (three ethnicity strata). This tested the standardised rate ratios and rate differences against the null hypothesis of effect measure homogeneity, and where p-values less than 0.05 were obtained the null hypothesis was rejected – ie. there was statistically significant heterogeneity of rate ratios or rate differences by ethnicity.

6 Study validity – reducing systematic errors

6.1 Bias

6.1.1 Selection bias

The part 1 analyses were performed on the largest cohort possible to avoid any selection bias. Only absentees, and those records without full information on age, sex, ethnicity and smoking status were excluded. The amount by which this reduced the sample size was calculated (as presented in Chapter 4).

6.1.2 Linkage bias

As previously mentioned, all deaths in the NZCMS are weighted to account for potential linkage bias Without weighting, linkage bias may introduce a degree of differential

outcome misclassification. The linked cohort records (ie. those who died) are weighted up to represent all eligible mortality records in the three years post-census. The unlinked records are also weighted down to balance the numbers in the cohort (fewer unlinked records will truly be alive).

Fawcett at al (2002) have also reported a lower rate of linkage for certain groups, including:

- Māori, Pacific, and Asian (1996 only) ethnic groups;
- Young adults aged 15-24 years;
- People living in rural areas at the time of death;
- People living in the Northern and Mid-Central Regional Health Authority areas;
- People living in areas with higher NZDep index scores (ie. living in more deprived small areas)

The records in the linked dataset (ie. full NZCMS cohort) are differentially weighted within these strata to account for this additional bias, and give a more accurate representation of the distribution of deaths. For example, if it is shown that among young Māori adults living in rural areas with high NZDep scores, only 2/3 (66%) of deaths are linked, then the linked records in this strata are given are weight of 3/2 (1.5). See Fawcett et al (2002) for details.

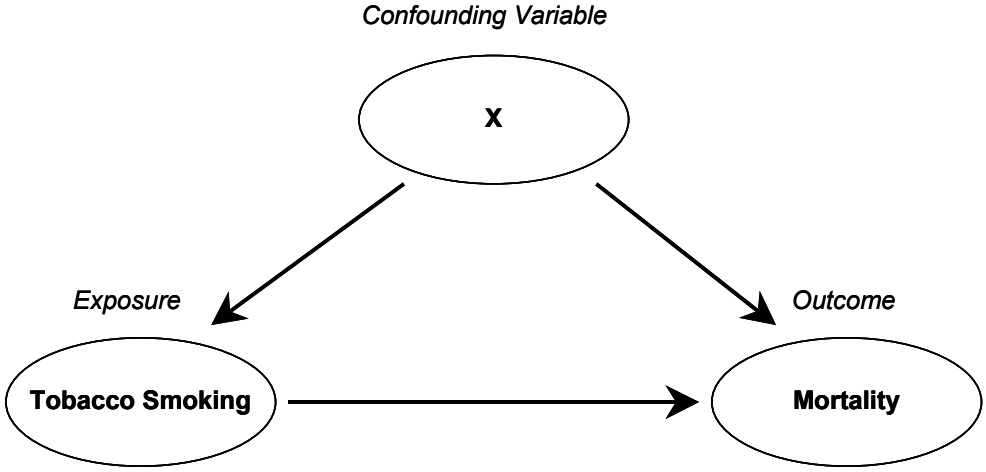
6.2 Confounding

There are a number of potential confounders in this study that may influence the observed association between smoking and mortality. A number of mechanisms in both the study design (eg. all analyses conducted separately by sex) and analysis have been used to remove these confounding effects as much as possible.

The potentially confounding variables have been identified through first principles. Those discussed in this and the next section all have the following properties (also illustrated as a diagrammatic model of confounding in Figure 2):

1. They are associated with current or ex cigarette smoking
2. They are independent risk factors for mortality (or IHD and stroke incidence, which are indicators of higher IHD and stroke mortality) – ie. they are associated with increased mortality in the unexposed (never-smoker) group
3. They are not wholly on the causal chain (from smoking to mortality) – ie. their relationship with mortality among smokers is not solely as an intermediary between smoking and mortality

Figure 2: Basic Model of Confounding



Age, sex and ethnicity are all potential confounders; having the properties above (Ministry of Health 2001; Tobias and Cheung 2001; USDHHS 2001b; Ministry of Health 2002a), at least for cardiovascular mortality.

The confounding effect of age is firstly reduced by restricting the age group under study to 25-74 year olds. By excluding under 25 year-olds, it removes a group of people who have a different mortality risk compared to the average participant (eg. teenagers low; infants high) and are more likely – for under 12 years at least – not to smoke. The effect of age has been further reduced by age standardisation to the 1996 New Zealand population as previously described.

The results are also presented by sex and ethnicity to remove confounding by these variables. For the all-ethnicity combined strata, results have been standardised by ethnicity to control for confounding.

(Stratification by age, sex and ethnicity will also demonstrate any effect measure modification of the smoking-mortality association by these factors.)

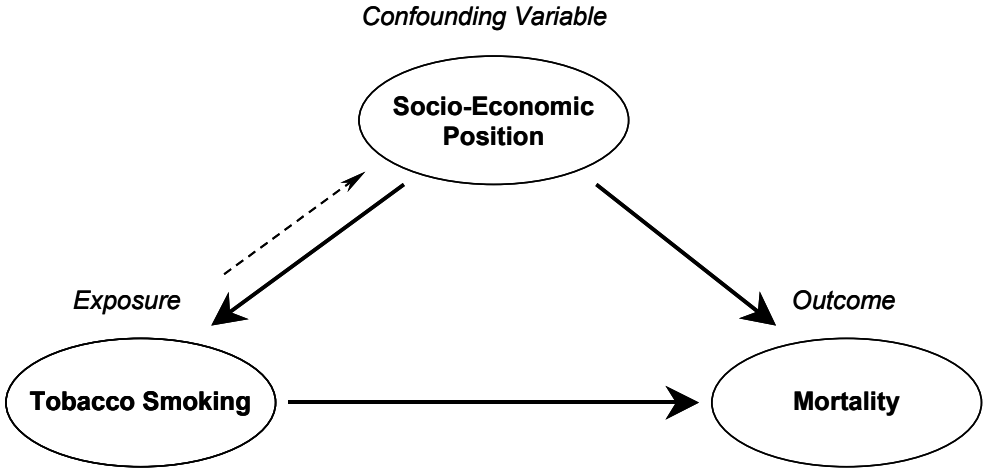
There are numerous other known and potential confounders of the smoking-mortality association. However in this study only those measured by the census questionnaires can be controlled for. These include various markers of socio-economic position (SEP). As discussed in the next section, SEP was controlled for using multivariable analysis (poisson regression), producing adjusted rate ratios. It was not possible to adjust for other variables such as behavioural factors (eg. diet, alcohol, exercise), physiological factors (eg. hypertension, hypercholesterolaemia, obesity) or pharmacological factors (eg. oral contraceptives), which may confound the observed association. For example, the US Surgeon General reported in 1989 that "cigarette smokers have higher rates of alcohol use, are more sedentary, and are less likely to wear seat belts." (USDHHS 1989) However, many of the key confounders appear to be patterned by SEP, which is a proximal or "upstream" determinant (Kaplan and Keil 1993; Sarfati, Scott et al. 1999; Engstrom, Tyden et al. 2000; Howden-Chapman and Tobias 2000). Therefore to some extent, SEP can be used as a proxy for other confounders, and by controlling for SEP there is at least partial control of these "downstream" factors as well.

7 Part 2: Multivariable regression analyses

Socio-economic position (SEP) is a potential confounder of the observed association between smoking and mortality, as it meets all three of the confounding properties (see Figure 3). Firstly, there is a strong association between SEP and smoking (Kaplan and Keil 1993; Sarfati, Scott et al. 1999; Crampton, Salmond et al. 2000; Howden-Chapman, and Tobias 2000; Tobias and Cheung 2001). Secondly, SEP is an independent (of smoking status) risk factor for mortality, both directly (eg. through increased access to pharmaceuticals and private health insurance), and indirectly (eg. through downstream

determinants of health) (Marmot, Rose et al. 1978; Marmot, Smith et al. 1991; Kaplan and Keil 1993; Howden-Chapman and Tobias 2000). Thirdly, the vast majority of the smoking – mortality relationship is not mediated through SEP to any great extent. In other words, the degree to which SEP is a causal determinant of smoking status by far outweighs the degree to which smoking status causes SEP, which in turn may affect mortality risk.

Figure 3: Socio-Economic Position as a confounding variable



In order to establish the degree of confounding from SEP, and remove this from the effect measures of interest, multivariable analyses were performed on the second restricted cohort. This is termed "Part 2" of the analyses and results.

The multivariable analysis was conducted using poisson regression, with smoking as the exposure and mortality (all-cause, IHD, stroke) as the outcome. The regression models included (at different points) age, ethnicity, and markers of SEP as co-variates (see section 3.3, page 50). Sex was not included in the models as a co-variate as results were presented for males and females separately

Ethnicity was included as a co-variate for the 'all-ethnicity, adjusted for ethnicity' group. Note that the results presented for Māori, Pacific, non-Māori non-Pacific, and 'all-ethnicity, not adjusted for ethnicity', have not been controlled for ethnicity.

As all records analysed needed to have complete data on each co-variate, poisson regression was only performed on the second restricted cohort. As previously described, the second restriction is a subset of the first restriction (used in Part 1), which not only excludes people for whom there is incomplete information on age, sex and ethnicity, but also excludes those who have incomplete information on these markers of SEP.

As discussed below, analyses were conducted in a number of steps, using regression models that included different variables. The regression outputs were mortality rate ratios (not rate differences) that are adjusted for these variables. I have termed these poisson effect measures "adjusted rate ratios".

Note: for the 'all-ethnicity adjusted for ethnicity' strata, all models include ethnicity as a co-variate in addition to those listed below.

The first regression model included age as the co-variate (using person-time in five-year age bands). These results are presented as 'rate ratios adjusted for age' or 'Adj RR- Age'.

Secondly, each SEP variable was added to the age model separately, producing rate ratios adjusted for age and income, age and education, age and motor vehicle ownership etc. It was intended to include as many of these variables as possible in the "full" regression model, however the results for each individual factor were analysed at this stage to ensure there were no unexpected or unusual effects (for example very large or very small estimates or confidence intervals due to instability from small cell sizes). No problems with using these variables individually were demonstrated, and each appeared to affect the smoking – mortality rate ratios to some extent.

Thirdly, a "final" or "full" model was run, including age plus all the SEP variables. While each SEP factor can be considered an indicator of SEP in their own right, they are likely to reflect slightly different and limited aspects of SEP (including different stages of the lifecourse), and using a combination will give a more complete measure of SEP (Liberatos, Link et al. 1988; Davey Smith, Shipley et al. 1990; Davey Smith, Hart et al. 1998; Lynch and Kaplan 2000; Blakely and Pearce 2002). For example, individual level variables will give a more accurate measure of "personal SEP" than just using an area-

based index such as NZDep, however NZDep will capture some of the contextual effects of area deprivation that personal SEP will not (Kaplan and Keil 1993; Blakely and Pearce 2002). These "full model" results are presented as 'rate ratios adjusted for age and SEP' or 'Adj RR – Age + SEP'.

7.1 Selection bias in second restricted cohort

The second restricted cohort used in the multivariable analyses is smaller than the first restriction. This may slightly affect the precision of the adjusted estimates, and potentially introduced some selection bias if those excluded (who do not have complete data for SEP) differ in their association between smoking and mortality from those included in the analyses. The size of each restricted cohort at census night in 1981 and 1996 was calculated to estimate the difference in participant numbers (Chapter 4). Other comparisons are given in Chapter 6 where standardised and multivariable results are shown together, and in Appendix C where person-time for both the first and second restriction in the all-age group (25-74 years) is tabulated.

7.2 Rationale for socio-economic variables

The conceptual models for confounding by age, sex, ethnicity and SEP as a whole have already been shown. There also needs to be some prima facie reason for choosing which markers of SEP are used in the regression models. The rationale for including the SEP variables listed above, as potential confounders of the smoking – mortality relationship, is described below. All the co-variates fit into the main SEP model, including the possibility that some are influenced to a small extent by smoking – ie. the small dashed arrow towards SEP in Figure 3 may apply, signifying some degree of mediation (as well as confounding) of the smoking – mortality association.

7.2.1 Income

Smoking prevalence is higher among people and households with lower incomes (USDHHS 1990; Kaplan and Keil 1993; Howden-Chapman and Tobias 2000; Blakely 2002). Smoking cessation also varies with income – higher among higher income groups

(USDHHS 1990) – and there is well demonstrated strong association between income and mortality independent of smoking status (Kaplan and Keil 1993).

Income may also lie partly on the causal chain between smoking and mortality, for example people who smoke may be less inclined to take up high paying jobs if smoking cessation is required, or smoking is difficult in the workplace (eg. due to Smokefree workplace legislation). However the magnitude of this potential effect (dashed arrow) would be far smaller than the influence of income on smoking status.

7.2.2 Education

Smoking prevalence declines (and smoking cessation increases) with increasing number of years of education (USDHHS 1990; USDHHS 2001b). Education is also an independent predictor of mortality (Kaplan and Keil 1993; Howden-Chapman and Tobias 2000; Blakely 2002), and does not lie on the causal chain between smoking and mortality (smoking does not determine educational level).

7.2.3 Marital status

A number of studies have shown that people who are divorced or separated have the highest smoking prevalence and highest overall tobacco use compared to those who are married and never-married (Rosengren, Wedel et al. 1989; USDHHS 1990; Engstrom, Tyden et al. 2000). Non-married people also appear to have a higher risk of mortality (Macintyre 1986; Rosengren, Wedel et al. 1989). Although some of the smoking – mortality relationship here may be mediated through marital status (dashed arrow towards marital status), this is likely to be very small compared to the confounding effect of marital status.

7.2.4 NZDep – small-area deprivation

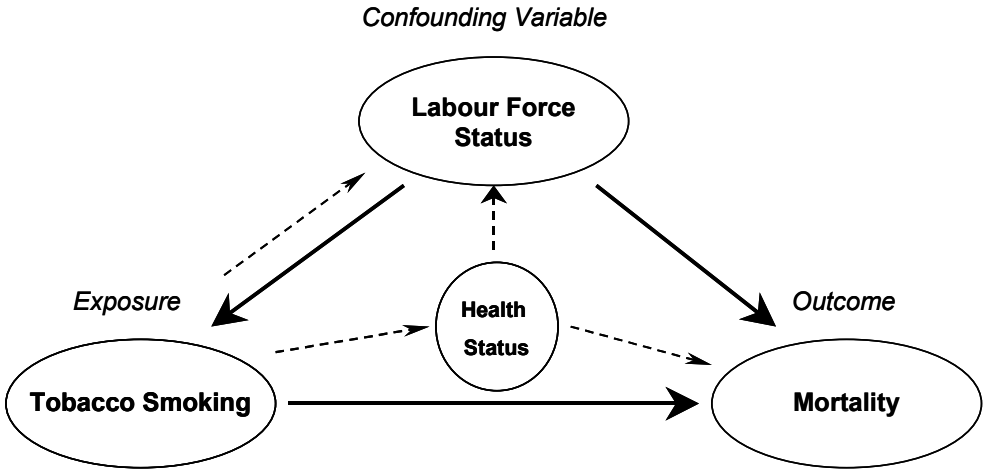
Small-area deprivation as measured by NZDep is associated with both smoking prevalence and mortality (Howden-Chapman and Tobias 2000). Whilst one's smoking habit may have an impact on where one lives (eg. attraction to industries unaffected by smokefree legislation, and therefore certain towns), this association is probably much

smaller than the impact of deprivation on smoking habits. Therefore NZDep is largely a confounder rather than a mediator.

7.2.5 Labour force status

Labour force status is associated with both smoking prevalence and mortality (Kaplan and Keil 1993). This marker is slightly more problematic as a proxy for health status (which is on the causal pathway to mortality), as health status also influences labour force status (two-way association) – see Figure 4. While it is more likely to be a confounder than a mediator, the possibility of some "over-control" here exists.

Figure 4: Labour force status as a confounding and mediating variable



7.2.6 Motor vehicle ownership and housing tenure

Motor vehicle ownership and housing tenure are also associated with both smoking prevalence and mortality (Kaplan and Keil 1993), and do not lie on the causal chain between smoking and mortality (ie. smoking probably does not determine motor vehicle ownership or housing tenure).

8 Part 3: Sensitivity analysis

A limited sensitivity analysis was conducted on a sub-section of data to assess the potential of exposure misclassification, after some significant findings of heterogeneity of the rate ratios by ethnicity were observed within the results.

Using crude data (non-standardised, weighted), the sensitivity of measuring all true current smokers as self-reported current smokers was varied to levels below 100%. This test required initially transforming the three-level smoking status variable (current, ex and never) into a two-level variable (smoker or non-smoker) – ie. ex and never were combined – before later splitting them out again.

For the purposes of this test it was assumed that:

- All people identified as current smokers are current smokers (ie. specificity 100%)
- Of truly current smokers not identifying as current smokers, there is a 50:50 split between self-reporting as ex and never-smokers.

Sensitivity levels of 95%, 90% and 80% were applied, and the resulting impact on observed rate ratios for the data tested is presented in Chapter 7. As these calculations were performed on crude data, a single age bracket was used to avoid confounding as much as possible. The age bracket of 65-74 years for males was chosen to capture a greater number of deaths.

Chapter 4: Study population

This chapter presents the number of participants by sex, age, and ethnicity in the cohorts used for analysis at the start of each cohort period (ie. on census night 1981 and 1996).

Table 6 shows the number of participants in the study population by level of restriction and ethnicity. The "original cohort" is defined as all people age 25-74 years, but excluding absentees. The first restriction for part 1 analyses required complete information on smoking, age, sex and ethnicity, and the second restriction for part 2 analyses additionally required complete information for socio-economic factors. Overall, the first restriction included 98.3% of the original cohort in 1981, and 92.5% in 1996. The second restriction more notably reduced the study size: 73.1% of the original cohort in 1981 and 74.0% in 1996.

Neither the sex nor ethnic distributions vary to a large extent across the different cohorts, however the percentage of the restricted cohorts that were Māori or Pacific slightly decreases with increasing restriction (compared to the full cohort). The male female ratio has an expectedly small female bias.

Table 7 shows the number of participants in the first restricted cohort by age, sex, ethnicity and smoking status. The percentages in brackets are the proportion of people in each age group for the ethnicity-smoking status strata – ie. they show the age structure for the population stratified by ethnicity and smoking status. As expected, the Māori and Pacific participants have overall a younger age structure than non-Māori non-Pacific. The group with the oldest age structure appears to be male non-Māori non-Pacific ex-smokers in both 1981 and 1996.

Table 8 also shows the same number of participants in the first restricted cohort by age, sex, ethnicity and smoking status, however the percentages in brackets are the proportion of people for each smoking status for the ethnicity-age group strata – ie. they show smoking prevalence for the population stratified by ethnicity and age. The group with the highest smoking prevalence in both years was young Māori, particularly young Māori women.

A summary of smoking prevalence changes in the 1981 compared with 1996 study populations is as follows. For non-Māori non-Pacific males the proportion of current smokers has decreased from 34% (1981) to 23% (1996). For non-Māori non-Pacific females there has been a decrease from 27%% to 20%. Māori males have decreased from 51% to 41%, Māori females from 54% to 48%. Pacific males have decreased from 45% to 38% and Pacific females have increased from 24% to 26%. Therefore, most groups have seen a reduction in smoking prevalence, however the size of this decrease has been smallest for Māori and non-Māori non-Pacific women, and prevalence among Pacific women has actually increased.

Note: At a very late stage of the final write-up of this thesis, it was discovered that 10,000 records had been inadvertently (by no fault of the author) left out of the total 1981 linked cohort / dataset (approximately 0.3% of the total dataset). After considerable discussion with the NZCMS team it was decided not to re-run the analyses for this thesis. The 10,000 records were examined, and no differences were found between overall characteristics of these records and the cohort that has been used in this study. In other words, there was no differential loss of data that could lead to selection bias. In addition, the records missing from the smaller 25-74 year age group would be less than 10,000. The 1996 cohort is unaffected.

Table 6: Numbers of participants in study population by level of restriction and ethnicity

		All Ethnicity Combined (% sex)		Maori (% ethnicity)		Pacific (% ethnicity)		Non-Maori Non-Pacific (% ethnicity)	
1981-1984									
Total Number in Original Cohort	Male	793,113	(49 %)	64,020		19,095		709,998	
	Female	811,407	(51 %)	65,694		18,588		727,125	
	Total	1,604,520		129,714	(8 %)	37,683	(2 %)	1,437,120	(90 %)
Total Number in First Restricted Cohort	Male	779,838	(49 %)	62,097		18,363		699,375	
	Female	796,944	(51 %)	63,426		17,733		715,788	
	Total	1,576,782		125,523	(8 %)	36,096	(2 %)	1,415,163	(90 %)
Total Number in Second Restricted Cohort	Male	576,288	(49 %)	38,136		10,245		527,910	
	Female	596,871	(51 %)	39,891		10,440		546,537	
	Total	1,173,159		78,024	(7 %)	20,685	(2 %)	1,074,447	(92 %)
1996-1999									
Total Number in Original Cohort	Male	1,016,388	(49 %)	107,055		37,146		872,187	
	Female	1,059,063	(51 %)	116,607		40,983		901,476	
	Total	2,075,451		223,662	(11 %)	78,126	(4 %)	1,773,663	(85 %)
Total Number in First Restricted Cohort	Male	938,289	(49 %)	101,715		34,572		802,002	
	Female	982,134	(51 %)	110,619		37,854		833,661	
	Total	1,920,423		212,334	(11 %)	72,426	(4 %)	1,635,663	(85 %)
Total Number in Second Restricted Cohort	Male	748,350	(49 %)	70,893		20,748		656,709	
	Female	787,770	(51 %)	77,502		22,680		687,585	
	Total	1,536,126		148,395	(10 %)	43,431	(3 %)	1,344,297	(88 %)

All Counts are random rounded numbers (to base 3). Some totals shown may differ to hand calculations and other tables by an amount of 3 due to random rounding variation.

Table 7: Numbers of participants in First Restricted Cohort by age, sex, ethnicity and smoking status – showing age group percentages

	Age Gp	MALE All Smoking Status (% all age)	MALE Never-Smoked (% all age)	MALE Current Smoker (% all age)	MALE Ex-Smoker (% all age)	FEMALE All Smoking Status (% all age)	FEMALE Never-Smoked (% all age)	FEMALE Current Smoker (% all age)	FEMALE Ex-Smoker (% all age)
1981-1984									
Maori	25-44	41613 (67 %)	11361 (65 %)	22767 (71 %)	7485 (59 %)	42672 (67 %)	10938 (58 %)	25425 (74 %)	6309 (63 %)
	45-64	17472 (28 %)	5202 (30 %)	8058 (25 %)	4212 (33 %)	17655 (28 %)	6492 (34 %)	8115 (24 %)	3045 (30 %)
	65-74	3012 (5 %)	1017 (6 %)	1041 (3 %)	951 (8 %)	3099 (5 %)	1518 (8 %)	870 (3 %)	708 (7 %)
	all age	62097	17580	31866	12648	63426	18948	34410	10062
Pacific	25-44	13911 (76 %)	5736 (77 %)	6342 (76 %)	1836 (70 %)	13467 (76 %)	9099 (77 %)	3243 (76 %)	1125 (70 %)
	45-64	3951 (22 %)	1488 (20 %)	1809 (22 %)	654 (25 %)	3690 (21 %)	2367 (20 %)	924 (22 %)	399 (25 %)
	65-74	501 (3 %)	192 (3 %)	189 (2 %)	120 (5 %)	576 (3 %)	390 (3 %)	108 (3 %)	75 (5 %)
	all age	18363	7416	8340	2610	17733	11856	4275	1599
Non-Maori Non-Pacific	25-44	354861 (51 %)	154128 (61 %)	126261 (52 %)	74475 (36 %)	354798 (50 %)	191682 (48 %)	107133 (55 %)	55986 (48 %)
	45-64	259920 (37 %)	75132 (30 %)	91623 (38 %)	93165 (45 %)	259623 (36 %)	145188 (36 %)	71565 (36 %)	42873 (37 %)
	65-74	84594 (12 %)	23289 (9 %)	22710 (9 %)	38595 (19 %)	101367 (14 %)	66378 (16 %)	17481 (9 %)	17508 (15 %)
	all age	699375	252549	240594	206235	715788	403248	196179	116367
All Ethnicity Combined	25-44	410385 (53 %)	171225 (62 %)	155370 (55 %)	83793 (38 %)	410940 (52 %)	211719 (49 %)	135798 (58 %)	63420 (50 %)
	45-64	281346 (36 %)	81819 (29 %)	101493 (36 %)	98031 (44 %)	280965 (35 %)	154047 (35 %)	80601 (34 %)	46314 (36 %)
	65-74	88107 (11 %)	24501 (9 %)	23940 (9 %)	39666 (18 %)	105042 (13 %)	68286 (16 %)	18462 (8 %)	18294 (14 %)
	all age	779838	277545	280803	221490	796947	434052	234861	128028
1996-1999									
Maori	25-44	67746 (67 %)	24624 (67 %)	30723 (73 %)	12399 (55 %)	75138 (68 %)	20580 (60 %)	40152 (75 %)	14406 (64 %)
	45-64	28992 (29 %)	10251 (28 %)	10335 (25 %)	8406 (37 %)	29784 (27 %)	10929 (32 %)	11991 (22 %)	6861 (30 %)
	65-74	4977 (5 %)	1995 (5 %)	1110 (3 %)	1875 (8 %)	5697 (5 %)	3024 (9 %)	1272 (2 %)	1398 (6 %)
	all age	101715	36870	42168	22680	110619	34533	53415	22665
Pacific	25-44	23319 (67 %)	11475 (67 %)	9297 (70 %)	2550 (60 %)	26010 (69 %)	15942 (65 %)	7611 (78 %)	2457 (71 %)
	45-64	9693 (28 %)	4731 (28 %)	3585 (27 %)	1380 (32 %)	9864 (26 %)	7233 (29 %)	1848 (19 %)	780 (23 %)
	65-74	1557 (5 %)	822 (5 %)	399 (3 %)	336 (8 %)	1983 (5 %)	1515 (6 %)	246 (3 %)	222 (6 %)
	all age	34569	17028	13281	4266	37857	24690	9705	3459
Non-Maori Non-Pacific	25-44	406053 (51 %)	219348 (57 %)	107499 (58 %)	79209 (34 %)	426438 (51 %)	238383 (49 %)	99153 (60 %)	88902 (47 %)
	45-64	294705 (37 %)	128388 (34 %)	62517 (34 %)	103806 (44 %)	296244 (36 %)	173454 (36 %)	52845 (32 %)	69945 (37 %)
	65-74	101241 (13 %)	34641 (9 %)	14235 (8 %)	52365 (22 %)	110979 (13 %)	70155 (15 %)	12369 (8 %)	28458 (15 %)
	all age	801999	382377	184251	235380	833661	481992	164367	187305
All Ethnicity Combined	25-44	497118 (53 %)	255447 (59 %)	147516 (62 %)	94158 (36 %)	527586 (54 %)	274905 (51 %)	146916 (65 %)	105765 (50 %)
	45-64	333396 (36 %)	143370 (33 %)	76434 (32 %)	113592 (43 %)	335892 (34 %)	191619 (35 %)	66687 (29 %)	77589 (36 %)
	65-74	107775 (11 %)	37455 (9 %)	15747 (7 %)	54573 (21 %)	118659 (12 %)	74691 (14 %)	13884 (6 %)	30081 (14 %)
	all age	938289	436272	239697	262323	982137	541215	227487	213435

All Counts are random rounded numbers (to base 3). Some totals shown may differ to hand calculations and other tables by an amount of 3 due to random rounding variation.

Table 8: Numbers of participants in First Restricted Cohort by age, sex, ethnicity and smoking status – showing smoking prevalence

	Age Gp	MALE All Smoking Status	MALE Never-Smoked (% all smok status)		MALE Current Smoker (% all smok status)		MALE Ex-Smoker (% all smok status)		FEMALE All Smoking Status	FEMALE Never-Smoked (% all smok status)		FEMALE Current Smoker (% all smok status)		FEMALE Ex-Smoker (% all smok status)	
1981-1984															
Maori	25-44	41613	11361	(27 %)	22767	(55 %)	7485	(18 %)	42672	10938	(26 %)	25425	(60 %)	6309	(15 %)
	45-64	17472	5202	(30 %)	8058	(46 %)	4212	(24 %)	17655	6492	(37 %)	8115	(46 %)	3045	(17 %)
	65-74	3012	1017	(34 %)	1041	(35 %)	951	(32 %)	3099	1518	(49 %)	870	(28 %)	708	(23 %)
	all age	62097	17580	(28 %)	31866	(51 %)	12648	(20 %)	63426	18948	(30 %)	34410	(54 %)	10062	(16 %)
Pacific	25-44	13911	5736	(41 %)	6342	(46 %)	1836	(13 %)	13467	9099	(68 %)	3243	(24 %)	1125	(8 %)
	45-64	3951	1488	(38 %)	1809	(46 %)	654	(17 %)	3690	2367	(64 %)	924	(25 %)	399	(11 %)
	65-74	501	192	(38 %)	189	(38 %)	120	(24 %)	576	390	(68 %)	108	(19 %)	75	(13 %)
	all age	18363	7416	(40 %)	8340	(45 %)	2610	(14 %)	17733	11856	(67 %)	4275	(24 %)	1599	(9 %)
Non-Maori	25-44	354861	154128	(43 %)	126261	(36 %)	74475	(21 %)	354798	191682	(54 %)	107133	(30 %)	55986	(16 %)
Non-Pacific	45-64	259920	75132	(29 %)	91623	(35 %)	93165	(36 %)	259623	145188	(56 %)	71565	(28 %)	42873	(17 %)
	65-74	84594	23289	(28 %)	22710	(27 %)	38595	(46 %)	101367	66378	(65 %)	17481	(17 %)	17508	(17 %)
	all age	699375	252549	(36 %)	240594	(34 %)	206235	(29 %)	715788	403248	(56 %)	196179	(27 %)	116367	(16 %)
All Ethnicity	25-44	410385	171225	(42 %)	155370	(38 %)	83793	(20 %)	410940	211719	(52 %)	135798	(33 %)	63420	(15 %)
Combined	45-64	281346	81819	(29 %)	101493	(36 %)	98031	(35 %)	280965	154047	(55 %)	80601	(29 %)	46314	(16 %)
	65-74	88107	24501	(28 %)	23940	(27 %)	39666	(45 %)	105042	68286	(65 %)	18462	(18 %)	18294	(17 %)
	all age	779838	277545	(36 %)	280803	(36 %)	221490	(28 %)	796947	434052	(54 %)	234861	(29 %)	128028	(16 %)
1996-1999															
Maori	25-44	67746	24624	(36 %)	30723	(45 %)	12399	(18 %)	75138	20580	(27 %)	40152	(53 %)	14406	(19 %)
	45-64	28992	10251	(35 %)	10335	(36 %)	8406	(29 %)	29784	10929	(37 %)	11991	(40 %)	6861	(23 %)
	65-74	4977	1995	(40 %)	1110	(22 %)	1875	(38 %)	5697	3024	(53 %)	1272	(22 %)	1398	(25 %)
	all age	101715	36870	(36 %)	42168	(41 %)	22680	(22 %)	110619	34533	(31 %)	53415	(48 %)	22665	(20 %)
Pacific	25-44	23319	11475	(49 %)	9297	(40 %)	2550	(11 %)	26010	15942	(61 %)	7611	(29 %)	2457	(9 %)
	45-64	9693	4731	(49 %)	3585	(37 %)	1380	(14 %)	9864	7233	(73 %)	1848	(19 %)	780	(8 %)
	65-74	1557	822	(53 %)	399	(26 %)	336	(22 %)	1983	1515	(76 %)	246	(12 %)	222	(11 %)
	all age	34569	17028	(49 %)	13281	(38 %)	4266	(12 %)	37857	24690	(65 %)	9705	(26 %)	3459	(9 %)
Non-Maori	25-44	406053	219348	(54 %)	107499	(26 %)	79209	(20 %)	426438	238383	(56 %)	99153	(23 %)	88902	(21 %)
Non-Pacific	45-64	294705	128388	(44 %)	62517	(21 %)	103806	(35 %)	296244	173454	(59 %)	52845	(18 %)	69945	(24 %)
	65-74	101241	34641	(34 %)	14235	(14 %)	52365	(52 %)	110979	70155	(63 %)	12369	(11 %)	28458	(26 %)
	all age	801999	382377	(48 %)	184251	(23 %)	235380	(29 %)	833661	481992	(58 %)	164367	(20 %)	187305	(22 %)
All Ethnicity	25-44	497118	255447	(51 %)	147516	(30 %)	94158	(19 %)	527586	274905	(52 %)	146916	(28 %)	105765	(20 %)
Combined	45-64	333396	143370	(43 %)	76434	(23 %)	113592	(34 %)	335892	191619	(57 %)	66687	(20 %)	77589	(23 %)
	65-74	107775	37455	(35 %)	15747	(15 %)	54573	(51 %)	118659	74691	(63 %)	13884	(12 %)	30081	(25 %)
	all age	938289	436272	(46 %)	239697	(26 %)	262323	(28 %)	982137	541215	(55 %)	227487	(23 %)	213435	(22 %)

All Counts are random rounded numbers (to base 3). Some totals shown may differ to hand calculations and other tables by an amount of 3 due to random rounding variation.

Chapter 5: Results - part 1

Part 1 Results Summary

For all-cause mortality and ischaemic heart disease (and possibly stroke), age standardised mortality rates are higher for Māori and Pacific compared with non-Māori non-Pacific. Over time, all-cause mortality rates have dropped markedly for non-Māori non-Pacific, however there is little, if any, downward trend for Māori and Pacific.

For the association of smoking with mortality, there were important variations by cohort (time) and ethnicity, and to some extent sex and age.

Age and ethnicity standardised rate ratios for all-cause mortality, IHD and stroke, comparing smokers to never smokers (ages 25-74) increased over time. The excess rate ratios approximately doubled from 1981-84 to 1996-99, for both males and for females. The standardised rate differences increased over time for all-cause mortality but showed little change for IHD and stroke.

There were also marked variations in the standardised rate ratios by ethnic group (Māori, Pacific, and non-Māori non-Pacific), which were determined to be statistically significant for both sexes, both years, and for all measured outcomes.

By sex, the rate ratios were similar between males and females for all-cause mortality, however the IHD and stroke rate ratios were higher for females than males.

By age, the rate ratios increased with increasing age for all-cause mortality. In contrast, the IHD rate ratios decreased with increasing age (as they also do for stroke in females, and males in 1981).

Results for the Part 1 analyses are presented separately for all-cause mortality, ischaemic heart disease, and stroke, in both tabular and chart form. They include:

- Number of deaths (random rounded)
- Crude (i.e. non-Standardised) Mortality rates
- Standardised Mortality Rates (age-standardised for all strata, plus ethnicity standardised for strata labelled "All Ethnicity Combined adj for eth")
- Standardised Rate Ratios (current and ex-smoker, compared to never smoked)
- Standardised Rate Differences (current and ex-smoker, compared to never smoked)
- 95% Confidence Intervals for each point estimate

These data are broken down by year, age, sex, ethnicity and smoking status. More detailed data – with an age breakdown for each ethnicity – are included in Appendix B (with mortality rates directly corresponding to graphs). Denominator numbers (person-time) used in the rate calculations are also included in the appendices.

All data have been weighted to adjust for linkage bias (as described in Methods section 1.1).

Mortality Rates and Rate Differences are expressed as deaths per 100,000 person-years.

As mentioned in Chapter 3, the Part 1 analyses were performed on the first restricted cohort, in order to include as many people as possible in the resident New Zealand Population, and allow more accurate calculations (i.e. less prone to selection bias, and higher precision).

All-Cause Mortality is presented first due to the greater precision of these results, but many points highlighted in this section are reiterated in the sections on ischaemic heart disease and stroke

1 All-Cause Mortality

1.1 Mortality Rates

Table 9 (male) and Table 10 (female) show the basic data for all-cause mortality.

Comparing the standardised to non-standardised rates, all-cause mortality rates are higher when age-standardised to the 1996 New Zealand population, for most age / sex / ethnicity strata. This indicates that these groupings have a younger age structure than the 1996 New Zealand population. This is particularly so for Māori and Pacific. Those strata that have the reverse pattern are older than the overall 1996 New Zealand population. This is seen for non-Māori non-Pacific ex-smokers (male and female), and female non-Māori non-Pacific never-smokers

Figure 5 and Figure 6 show the standardized all-cause mortality rates in graph form. The figures show 1981-1984 results on the left, 1996-1999 on the right. Graphs for "all-age" (ie. 25-74 years) are at the top of each figure, with the three age bands below. The vertical lines crossing the top of each bar represent the 95% confidence intervals for each point estimate. Estimates are most precise for non-Māori non-Pacific, and least precise for Pacific, with Māori intermediate between the two, as shown by the width of the 95% confidence intervals. This reflects the size of each population, and thereby numbers of deaths in each. Bearing this in mind, the rates for Pacific should be interpreted with caution, but some of the overall trends remain evident.

All-cause mortality rates rise with increasing age, reflected in the fact that the y-axes for the graphs change for each age group. This needs to be kept in mind when making a visual comparison between age groups.

A sex difference is also apparent, with mortality rates higher overall for men than women (therefore y-axes here differ also). On an absolute scale, this difference increases with age (as mortality rates increase).

For most age and sex groupings, Māori and Pacific have higher mortality rates than non-Māori non-Pacific. It is particularly notable that for Māori this pattern is true within all smoking status strata. For example, Māori never-smokers have more than double the mortality rate of non-Māori non-Pacific never-smokers. In 1981, male 25-74 age rates for never-smokers were 1,450 for Māori vs 687 for non-Māori non-Pacific, and in 1996 1,230 vs 442. That is, there are large ethnic differences in mortality rates independent of smoking status.

Over the 15-year period, from 1981 to 1996, standardised rates have dropped markedly for non-Māori non-Pacific in all smoking strata. However for Māori and Pacific an overall time trend is less clear. For Māori, there appears to have been a decrease in mortality for never-smokers and ex-smokers (more clear for Māori female never smokers as the 95% confidence intervals do not overlap), but rates for current smokers have either been static or increased.

1.2 Rate Ratios (demonstrating relative risk)

Rate ratios and rate differences (current and ex-smokers compared to never-smokers) are given in Table 11 and Table 12, and can be conceived visually by comparing the rates shown in Figure 5 and Figure 6.

For the rate ratio estimates there are four main findings.

The first and least surprising, is that overall, current smokers and ex-smokers have a rate ratio greater than 1.0. In other words they are more likely to die than never-smokers (higher mortality rates). Among the overall population (adjusted for ethnicity) there is a gradient in strength of this risk from current smokers at the highest risk, then down to ex-smokers, then to the reference group of never-smokers. This overall gradient is however predominantly the result of the (numerically larger) non-Māori non-Pacific population. Although the confidence intervals are wide (apart from Māori males), there does not seem to be such a consistent gradient within Māori or Pacific groups, particularly in 1981. One particularly notable pattern for Pacific people, is that although most of the 95% confidence intervals tend to overlap, many of the rate ratio estimates are larger for Pacific ex-smokers

than Pacific current smokers. The same can be said for the rate differences among Pacific people.

The second main finding is that there is variation of rate ratios between ethnic groups. In particular, Māori have lower rate ratios for smoking mortality than non-Māori non-Pacific, and the confidence intervals for the all-age estimates are not overlapping. For example, among current smokers in 1996-99 Māori males have a rate ratio of 1.51 (95% CI 1.02-1.39) compared with non-Māori non-Pacific males of 2.22 (2.12-2.33). This variation is consistent by sex, age and crude and standardised rates. A reason for this pattern can be seen from examination of the underlying standardised mortality rates. For example, in Figure 5, the higher mortality rates among Māori males in 1996 naturally gives rise to lower ratios, as a measure of the relative risk, even though the rate difference is not too dissimilar to that for non-Māori non-Pacific. In 1981, the smaller rate difference for Māori also contributes to the lower rate ratios.

A Wald statistical test of heterogeneity was conducted on the rate ratios between the ethnic groups for the all-age strata (25-74 years), which revealed a high degree of statistical significance (ie. the null hypothesis of uniform rate ratios was rejected). For all-cause mortality, the Wald p-values for current smoker rate ratios were less than 0.00001 for males and females for both 1981 and 1996.

The third main finding is an increase in the relative measures of effect of smoking over time, overall and within ethnic groups and age groups. For example, the male all-cause mortality rate ratio in 1981 (all ethnicity combined, ethnicity standardised) is 1.59, so that in 1981 current smokers had a 60% increased risk of dying compared to never-smokers. In 1996, the rate ratio was 2.05 – ie. a 105% increased risk, or double. For females the increase was 1.49 to 2.01. A reason for the increase is that as mortality rates decline for both smokers and never-smokers, the ratio of the two increases if the absolute difference remains about the same. But overall, all-cause mortality rates have declined more sharply amongst never-smokers than current smokers, so that both the rate differences and rate ratios have increased. The pattern for ex-smokers is less clear cut, and the confidence intervals tend to overlap.

Fourth, there does appear to be an increase in rate ratios with age, although this is less so for older males in both years (comparing 45-64 years with 65-74), and females in 1981. Such an increase with age is consistent with a greater percentage of deaths at older ages being smoking-related. For females in 1996 in particular rate ratios increase with increasing age, which is largely driven by the same pattern in non-Māori non-Pacific (see graphs following and tables in Appendix B).

The effect of smoking on all-cause mortality, as reflected in the rate ratios, is similar for males and females overall in both 1981 and 1996. The all-age (25-74) all-ethnicity male rate ratio in 1996 was 2.05, compared to the female rate ratio of 2.01. Within the smaller age strata the 25-44 group shows some sex difference (males higher; eg 1.57 vs 1.20 in 1996-99), with the rate ratios becoming more similar with increasing age (and in 1996-99 the 65-74 year old females had a slightly higher rate ratio 2.32 vs male 2.18). There is less overall similarity (25-74 years) for ex-smokers. For example in 1996-99 the male age and ethnicity adjusted rate ratio was 1.30, and the female age and ethnicity adjusted rate ratio was 1.54 (and the confidence intervals do not overlap).

1.3 Rate Differences (demonstrating absolute risk)

Rate differences for males and females (all ethnicity combined) have increased over time for current smokers compared to never smokers. The 25-74 year old male age and ethnicity standardised rate difference of 444 (95% CI 405 to 482) in 1981-84 increases to 539 (504 to 574) in 1996-99, and the 95% confidence intervals do not overlap. For ex-smokers rate differences tend to decrease over time, however the confidence intervals overlap. Rate differences for females are smaller than males due to their lower mortality rates overall.

There does not appear to be a consistent difference between ethnic groups. Wald testing for heterogeneity by ethnicity demonstrated p-values < 0.05 for both sexes and both years for the current smoker rate differences. However, for a very large study such as the NZCMS, even small variations will often reach statistical significance, and looking at the results it should be noted that for 1996-99 the rate differences for Māori compared to non-Māori non-Pacific are more similar (homogeneous) than the rate ratio comparison.

Table 9: Male All-Cause Mortality Data – No. Deaths, Non-Std Mortality Rates and Std Mortality Rates per 100,000 person-years (First Restrn)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)	
1981-1984													
Maori	all age	564	1,014	1,450	(1,278 - 1,621)	963	935	1,724	(1,556 - 1,892)	495	1,270	1,563	(1,374 - 1,752)
Pacific	all age	93	387	899	(619 - 1,178)	120	444	915	(656 - 1,175)	69	819	1,586	(1,095 - 2,077)
NonM-NonP	all age	4,536	575	687	(663 - 711)	8,169	1,106	1,151	(1,122 - 1,181)	8,190	1,356	886	(862 - 909)
All Ethnicity Combined **not adj for eth**	all age	5,196	598	732	(708 - 756)	9,249	1,065	1,194	(1,164 - 1,223)	8,754	1,344	918	(894 - 941)
All Ethnicity Combined **adj for eth**	all age	5,193	598	749	(724 - 774)	9,249	1,065	1,192	(1,163 - 1,222)	8,754	1,344	948	(922 - 975)
	25-44	744	138	155	(139 - 170)	1,014	210	214	(197 - 231)	369	150	151	(130 - 172)
	45-64	2,064	812	831	(787 - 876)	4,395	1,419	1,351	(1,302 - 1,399)	3,132	1,096	991	(944 - 1,037)
	65-74	2,385	3,221	3,238	(3,085 - 3,392)	3,843	5,143	5,221	(5,029 - 5,414)	5,256	4,417	4,498	(4,347 - 4,648)
1996-1999													
Maori	all age	996	851	1,230	(1,133 - 1,327)	1,284	964	1,857	(1,711 - 2,002)	816	1,198	1,335	(1,223 - 1,446)
Pacific	all age	351	651	974	(837 - 1,111)	279	669	1,144	(944 - 1,345)	144	1,114	1,363	(1,083 - 1,643)
NonM-NonP	all age	4,563	387	442	(427 - 456)	4,479	789	982	(949 - 1,015)	6,753	987	601	(583 - 619)
All Ethnicity Combined **not adj for eth**	all age	5,907	438	512	(497 - 527)	6,042	813	1,094	(1,061 - 1,126)	7,713	1,008	653	(635 - 672)
All Ethnicity Combined **adj for eth**	all age	5,907	438	513	(498 - 528)	6,042	813	1,052	(1,020 - 1,083)	7,713	1,008	668	(649 - 687)
	25-44	1,008	129	136	(124 - 147)	1,026	227	213	(195 - 230)	384	144	141	(121 - 161)
	45-64	2,283	503	527	(502 - 552)	2,622	1,086	1,087	(1,038 - 1,135)	2,334	687	657	(626 - 688)
	65-74	2,613	2,240	2,212	(2,118 - 2,305)	2,391	4,813	4,818	(4,603 - 5,033)	4,995	3,135	3,134	(3,036 - 3,231)

Male All-Cause Mortality Rates by Smoking Status NZCMS

*Random Rounded † Deaths per 100,000 Person-Years

Table 10: Female All-Cause Mortality Data – No. Deaths, Non-Std Mortality Rates and Std Mortality Rates per 100,000 person-years (First Restrn)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)	
1981-1984													
Maori	all age	507	859	1,060	(932 - 1,187)	603	535	1,127	(979 - 1,275)	330	1,035	1,455	(1,237 - 1,674)
Pacific	all age	120	322	579	(433 - 725)	27	203	384	(176 - 592)	36	733	1,242	(708 - 1,777)
NonM-NonP	all age	6,384	523	431	(418 - 443)	3,774	619	685	(659 - 712)	2,631	748	626	(597 - 655)
All Ethnicity Combined **not adj for eth**	all age	7,014	533	455	(442 - 467)	4,407	598	721	(695 - 747)	2,994	771	675	(645 - 704)
All Ethnicity Combined **adj for eth**	all age	7,014	533	480	(465 - 494)	4,407	598	713	(687 - 739)	2,997	771	698	(666 - 730)
	25-44	606	93	106	(94 - 118)	486	114	114	(101 - 128)	207	107	121	(99 - 143)
	45-64	2,382	515	509	(482 - 536)	2,061	834	782	(741 - 822)	1,098	803	739	(683 - 795)
	65-74	4,026	1,966	2,033	(1,954 - 2,111)	1,863	3,033	3,138	(2,967 - 3,309)	1,689	2,977	3,107	(2,924 - 3,291)
1996-1999													
Maori	all age	741	685	821	(749 - 893)	867	508	1,189	(1,068 - 1,310)	591	847	1,216	(1,091 - 1,341)
Pacific	all age	357	462	667	(578 - 756)	93	295	703	(483 - 923)	66	592	867	(591 - 1,143)
NonM-NonP	all age	4,605	316	283	(274 - 292)	2,352	460	623	(595 - 651)	2,931	525	445	(427 - 462)
All Ethnicity Combined **not adj for eth**	all age	5,697	347	322	(313 - 331)	3,309	464	705	(677 - 734)	3,585	561	504	(485 - 522)
All Ethnicity Combined **adj for eth**	all age	5,697	347	330	(320 - 340)	3,309	464	665	(638 - 692)	3,585	561	509	(490 - 528)
	25-44	612	74	79	(71 - 87)	471	103	95	(85 - 106)	240	78	78	(66 - 90)
	45-64	2,124	357	364	(346 - 381)	1,482	692	689	(648 - 730)	1,224	513	517	(483 - 551)
	65-74	2,961	1,337	1,333	(1,280 - 1,387)	1,356	3,131	3,089	(2,904 - 3,274)	2,121	2,372	2,371	(2,258 - 2,484)

Female All-Cause Mortality Rates by Smoking Status NZCMS

*Random Rounded † Deaths per 100,000 Person-Years

Figure 5: Male All-Cause Standardised Mortality Rates per 100,000 person-yrs (First Rst)

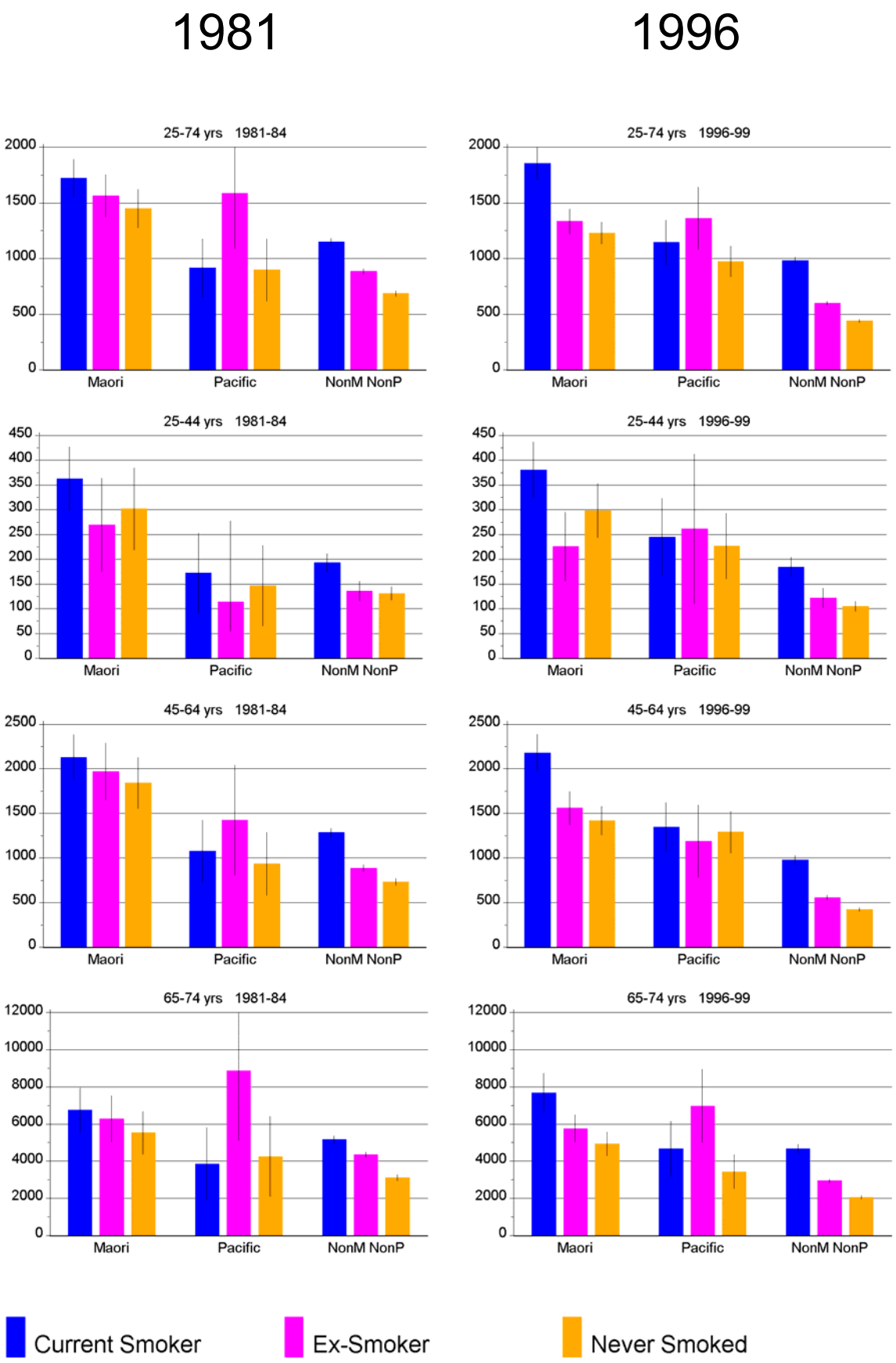


Figure 6: Female All-Cause Standardised Mortality Rates per 100,000 person-yrs (First Rst)

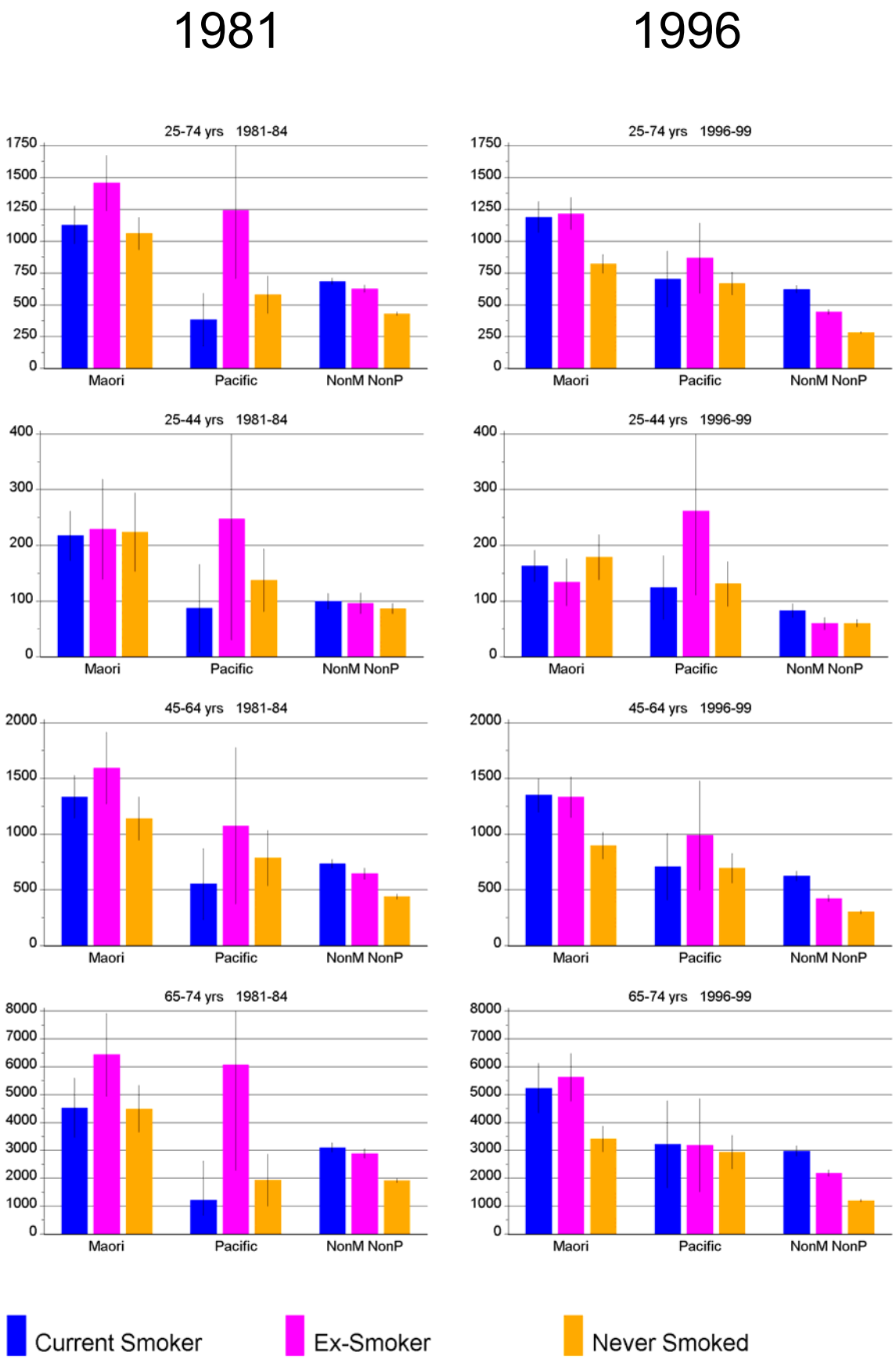


Table 11: Male All-Cause Standardised Rate Ratios and Rate Differences (First Restriction)

	Age Gp	SRR (reference gp never smoked)				SRD (reference gp never smoked)			
		Smoker (95% CI)		Ex-Smoker (95% CI)		Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	all age	1.19	(1.02 - 1.39)	1.08	(0.91 - 1.28)	274	(34 - 514)	113	(-142 - 368)
Pacific	all age	1.02	(0.67 - 1.55)	1.76	(1.14 - 2.74)	17	(-365 - 399)	687	(122 - 1,252)
NonM-NonP	all age	1.68	(1.61 - 1.75)	1.29	(1.23 - 1.35)	464	(427 - 502)	199	(165 - 232)
All Ethnicity Combined **not adj for eth**	all age	1.63	(1.57 - 1.70)	1.25	(1.20 - 1.31)	462	(424 - 500)	186	(152 - 219)
All Ethnicity Combined **adj for eth**	all age	1.59	(1.53 - 1.66)	1.27	(1.21 - 1.32)	444	(405 - 482)	199	(163 - 236)
	25-44	1.38	(1.22 - 1.58)	0.98	(0.82 - 1.16)	59	(36 - 83)	-3	(-30 - 23)
	45-64	1.62	(1.52 - 1.73)	1.19	(1.11 - 1.28)	519	(454 - 585)	159	(95 - 223)
	65-74	1.61	(1.52 - 1.71)	1.39	(1.31 - 1.47)	1983	(1,737 - 2,230)	1259	(1,044 - 1,475)
1996-1999									
Maori	all age	1.51	(1.35 - 1.69)	1.09	(0.97 - 1.22)	627	(452 - 802)	105	(-43 - 253)
Pacific	all age	1.18	(0.94 - 1.47)	1.40	(1.09 - 1.80)	171	(-72 - 413)	389	(78 - 701)
NonM-NonP	all age	2.22	(2.12 - 2.33)	1.36	(1.30 - 1.42)	540	(504 - 576)	159	(136 - 182)
All Ethnicity Combined **not adj for eth**	all age	2.13	(2.05 - 2.23)	1.28	(1.22 - 1.33)	581	(546 - 617)	141	(117 - 165)
All Ethnicity Combined **adj for eth**	all age	2.05	(1.97 - 2.14)	1.30	(1.25 - 1.36)	539	(504 - 574)	155	(131 - 179)
	25-44	1.57	(1.40 - 1.76)	1.04	(0.88 - 1.23)	77	(56 - 98)	6	(-17 - 28)
	45-64	2.06	(1.93 - 2.20)	1.25	(1.16 - 1.33)	559	(505 - 614)	130	(89 - 170)
	65-74	2.18	(2.05 - 2.32)	1.42	(1.34 - 1.49)	2606	(2,372 - 2,841)	922	(787 - 1,057)

Male All-Cause SRR & SRD by Smoking Status NZCMS

Table 12: Female All-Cause Standardised Rate Ratios and Rate Differences (First Restriction)

	Age Gp	SRR (reference gp never smoked)				SRD (reference gp never smoked)			
		Smoker (95% CI)		Ex-Smoker (95% CI)		Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	all age	1.06	(0.89 - 1.27)	1.37	(1.13 - 1.67)	68	(-128 - 263)	396	(142 - 649)
Pacific	all age	0.66	(0.37 - 1.20)	2.15	(1.30 - 3.53)	-195	(-449 - 59)	664	(109 - 1,218)
NonM-NonP	all age	1.59	(1.52 - 1.67)	1.45	(1.38 - 1.53)	254	(225 - 284)	195	(164 - 227)
All Ethnicity Combined **not adj for eth**	all age	1.59	(1.52 - 1.66)	1.48	(1.41 - 1.56)	267	(238 - 296)	220	(188 - 252)
All Ethnicity Combined **adj for eth**	all age	1.49	(1.42 - 1.56)	1.45	(1.38 - 1.54)	233	(203 - 263)	218	(183 - 253)
	25-44	1.08	(0.92 - 1.27)	1.14	(0.92 - 1.41)	8	(-10 - 26)	15	(-10 - 40)
	45-64	1.54	(1.43 - 1.65)	1.45	(1.32 - 1.59)	273	(224 - 321)	230	(168 - 292)
	65-74	1.54	(1.44 - 1.65)	1.53	(1.42 - 1.64)	1106	(917 - 1,294)	1075	(875 - 1,275)
1996-1999									
Maori	all age	1.45	(1.27 - 1.66)	1.48	(1.29 - 1.70)	368	(228 - 509)	395	(251 - 539)
Pacific	all age	1.05	(0.75 - 1.48)	1.30	(0.92 - 1.84)	36	(-201 - 273)	200	(-90 - 490)
NonM-NonP	all age	2.20	(2.09 - 2.33)	1.57	(1.50 - 1.66)	340	(311 - 370)	162	(142 - 182)
All Ethnicity Combined **not adj for eth**	all age	2.19	(2.08 - 2.30)	1.56	(1.49 - 1.64)	383	(354 - 413)	182	(161 - 202)
All Ethnicity Combined **adj for eth**	all age	2.01	(1.91 - 2.12)	1.54	(1.47 - 1.62)	335	(306 - 364)	179	(157 - 200)
	25-44	1.20	(1.03 - 1.40)	0.98	(0.81 - 1.18)	16	(3 - 30)	-2	(-16 - 13)
	45-64	1.89	(1.75 - 2.05)	1.42	(1.31 - 1.54)	325	(281 - 370)	154	(115 - 192)
	65-74	2.32	(2.16 - 2.49)	1.78	(1.67 - 1.89)	1756	(1,563 - 1,948)	1038	(913 - 1,163)

Female All-Cause SRR & SRD by Smoking Status NZCMS

2 Ischaemic Heart Disease

The estimates for Ischaemic Heart Disease (IHD) are less precise than for all-cause mortality, however many of the patterns seen for all-cause mortality are replicated for IHD.

Table 13 (male) and Table 14 (female) show the basic data for IHD mortality.

Table 13, Table 14, Figure 7 and Figure 8 show the IHD standardised mortality rates. Estimates for Pacific and Māori are again less precise, with particularly wide confidence intervals for female IHD rates. Some of the age-specific mortality rates shown in the graphs (and the tables in Appendix B) for Māori and (especially) Pacific are not presented as there were too few deaths to allow any meaningful interpretation.

IHD mortality rates increase with age, and are higher for males and Māori. Pacific peoples may have had lower IHD mortality rates in 1981, but they are clearly intermediate between Māori and non-Māori non-Pacific in 1996. As with all-cause mortality, Māori never-smokers have a higher mortality rate than non-Māori non-Pacific never-smokers.

Standardised mortality rates for IHD have dropped markedly for non-Māori non-Pacific over the 1981-1996 15-year period - regardless of smoking status. Māori also appear to have lower mortality rates for IHD in 1996, however the decline is less, particularly among Māori males, for whom there has been little progress. The pattern for Pacific is unclear.

2.1 Rate Ratios and Rate Differences

IHD rate ratios and rate differences are given in Table 15 and Table 16, and can be conceived visually by comparing the rates shown in Figure 7 and Figure 8.

For IHD, smokers and ex-smokers also have a rate ratio greater than 1.0, and therefore are more likely to die from this cause of death than never-smokers. There is a recognisable gradient (by smoking status) for non-Māori non-Pacific in both years, and for Māori in

1996. However, amongst Pacific for both years and Māori in 1981 – with quite wide confidence intervals – an association of IHD mortality with smoking, and a gradient by smoking status, are harder to discern.

Rate ratios differ between ethnic groups. For all-age IHD estimates, the rate ratios comparing smokers and never smokers are lower among Māori than non-Māori non-Pacific, and the confidence intervals do not overlap. For example, the 1996 rate ratio among 25-74 year old Māori males was 1.34 (1.07 to 1.67), considerably less than 2.21 (2.02 to 2.42) among non-Māori non-Pacific males. On Wald testing, this heterogeneity of the rate ratios by ethnicity was statistically significant at the 95% level (ie. p-values < 0.05) in 1981-84 for both males and females. In 1996-99 the p-values were less than 0.001 for both males and females.

Smoking rate ratios for IHD have increased over time. For all ethnicity combined (ethnicity standardised), the male rate ratio comparing current to never smokers increased from 1.50 to 2.03 - a similar rise to all-cause mortality. The female rate ratio increased from 1.86 to 2.67. Within ethnic groups, this pattern is seen for non-Māori non-Pacific and Māori (although for Māori the confidence intervals overlap). There may be a different pattern (a decrease) for Pacific, although there is greater imprecision here.

A point of contrast between the IHD data and all-cause mortality is that the all-ethnicity smoking rate ratios decrease with age for IHD (they increase for all-cause mortality). There may also be a particularly stronger age gradient for IHD rate ratios for females amongst non-Māori non-Pacific (see appendix B), although the confidence intervals for younger age groups are wide. For example, in 1996-99 the 25-44, 45-64 and 65-74 year non-Māori non-Pacific female rate ratios respectively were 10 (95% CI 2.74-36.51), 3.53 (2.68-4.64) and 2.78 (2.35-3.30).

Also in contrast to all-cause mortality, there does appear to be a substantial difference between male and female rate ratios for IHD overall. For the all-age (25-74 years) all-ethnicity strata, and most ethnic specific strata, females have a higher smoking rate ratio than males for IHD, and for the all-ethnicity estimates the confidence intervals do not

overlap for both 1981 and 1996. Also, for 1996-99, all the age-specific IHD rate ratios (25-44, 45-64, 65-74) within the all-ethnicity stratum were higher for females than males.

The female all-age all-ethnicity smoking rate ratios for IHD (1996 RR = 2.67) are also higher than for all-cause mortality (1996 RR = 2.01), whereas the male all-age rate ratios are roughly comparable (1996 IHD RR of 2.05 versus 1996 all-cause RR of 2.03). It should be noted however, that given the heterogeneity (in different directions) by age for IHD and all-cause mortality, the all-age rate ratios do not tell the whole story, and further patterns emerge when the data are examined by cause, sex and age. For example, in 1996-99 the male all-ethnicity IHD rate ratio for the 25-44 age group was higher than the respective all-cause estimate (2.22 vs 1.57), but among the 65-74 year olds the reverse is seen (1.81 vs 2.18) (the same pattern is seen in 1981-84). For females, almost all the age-specific (all-ethnicity) rate ratios for IHD are higher than those for all-cause mortality.

The IHD rate differences for females are again smaller than males due to their lower mortality rates overall. However, there has been little change over time in all-ethnicity rate differences. The p-values for rate difference heterogeneity by ethnicity were > 0.05, however it should again be noted that the 1996-99 rate differences for Māori compared to non-Māori non-Pacific are reasonably similar (homogeneous).

Table 13: Male IHD Mortality Data – No. Deaths, Non-Std Mortality Rates and Std Mortality Rates per 100,000 person-years (First Restriction)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)	
1981-1984													
Maori	all age	162	288	456	(361 - 550)	240	234	476	(387 - 564)	153	388	489	(388 - 590)
Pacific	all age	12	46	97	(18 - 176)	24	91	222	(89 - 356)	24	279	514	(244 - 783)
NonM-NonP	all age	1,635	207	257	(242 - 271)	2,844	385	400	(383 - 417)	3,045	504	320	(306 - 333)
All Ethnicity Combined **not adj for eth**	all age	1,806	208	266	(252 - 280)	3,111	358	403	(387 - 420)	3,222	494	327	(314 - 341)
All Ethnicity Combined **adj for eth**	all age	1,806	208	268	(253 - 282)	3,111	358	402	(385 - 418)	3,219	494	336	(321 - 351)
	25-44	57	11	14	(9 - 19)	180	37	40	(33 - 48)	54	22	20	(14 - 27)
	45-64	741	291	294	(268 - 319)	1,620	523	496	(468 - 525)	1,239	434	388	(359 - 417)
	65-74	1,011	1,362	1,360	(1,263 - 1,456)	1,311	1,756	1,778	(1,667 - 1,888)	1,923	1,617	1,632	(1,544 - 1,720)
1996-1999													
Maori	all age	240	205	330	(279 - 381)	285	215	441	(370 - 511)	243	356	391	(332 - 450)
Pacific	all age	87	161	263	(192 - 335)	75	185	286	(197 - 374)	30	246	301	(173 - 429)
NonM-NonP	all age	1,167	99	117	(109 - 124)	1,167	205	258	(241 - 274)	1,785	261	149	(141 - 157)
All Ethnicity Combined **not adj for eth**	all age	1,494	111	135	(127 - 143)	1,527	206	282	(266 - 298)	2,058	269	165	(157 - 173)
All Ethnicity Combined **adj for eth**	all age	1,491	111	134	(127 - 142)	1,530	206	273	(257 - 289)	2,058	269	169	(160 - 178)
	25-44	81	10	12	(8 - 15)	120	27	26	(20 - 31)	33	12	11	(6 - 15)
	45-64	591	130	137	(124 - 150)	786	325	325	(299 - 351)	651	192	184	(167 - 201)
	65-74	819	703	694	(641 - 746)	621	1,249	1,255	(1,145 - 1,365)	1,374	862	854	(804 - 904)

Male IHD Mortality Rates by Smoking Status NZCMS

*Random Rounded † Deaths per 100,000 Person-Years

Table 14: Female IHD Mortality Data – No. Deaths, Non-Std Mortality Rates and Std Mortality Rates per 100,000 person-years (First Restriction)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)	
1981-1984													
Maori	all age	120	202	273	(209 - 338)	105	92	267	(187 - 346)	66	206	301	(207 - 396)
Pacific	all age	9	26	47	(8 - 86)	9	69	160	(22 - 299)	6	91	185	(103 - 399)
NonM-NonP	all age	1,590	130	100	(94 - 106)	1,068	175	201	(186 - 215)	627	178	145	(132 - 158)
All Ethnicity Combined **not adj for eth**	all age	1,719	131	105	(99 - 111)	1,182	160	205	(190 - 219)	696	179	153	(139 - 166)
All Ethnicity Combined **adj for eth**	all age	1,719	131	110	(103 - 116)	1,179	160	204	(190 - 218)	696	179	156	(142 - 170)
	25-44	15	3	4	(1 - 6)	33	8	9	(5 - 13)	15	8	8	(3 - 14)
	45-64	375	81	76	(66 - 86)	528	213	203	(182 - 224)	171	126	113	(91 - 134)
	65-74	1,326	649	666	(623 - 710)	621	1,011	1,062	(963 - 1,161)	510	895	922	(827 - 1,018)
1996-1999													
Maori	all age	120	113	145	(115 - 176)	144	84	235	(183 - 288)	90	128	202	(150 - 254)
Pacific	all age	45	61	90	(59 - 121)	12	42	124	(30 - 218)	9	77	118	(20 - 216)
NonM-NonP	all age	606	42	36	(33 - 39)	375	73	107	(95 - 119)	432	77	64	(57 - 70)
All Ethnicity Combined **not adj for eth**	all age	771	47	43	(39 - 46)	531	75	125	(113 - 138)	531	83	73	(66 - 80)
All Ethnicity Combined **adj for eth**	all age	771	47	44	(40 - 47)	531	75	116	(105 - 128)	528	83	74	(67 - 81)
	25-44	12	1	2	(0 - 3)	30	7	6	(3 - 9)	12	4	4	(1 - 6)
	45-64	189	32	32	(27 - 38)	204	95	93	(78 - 108)	135	56	56	(45 - 68)
	65-74	573	258	259	(235 - 282)	297	686	664	(579 - 749)	387	431	431	(384 - 479)

Female IHD Mortality Rates by Smoking Status NZCMS

*Random Rounded † Deaths per 100,000 Person-Years

Figure 7: Male IHD Standardised Mortality Rates per 100,000 person-yrs (First Rst)

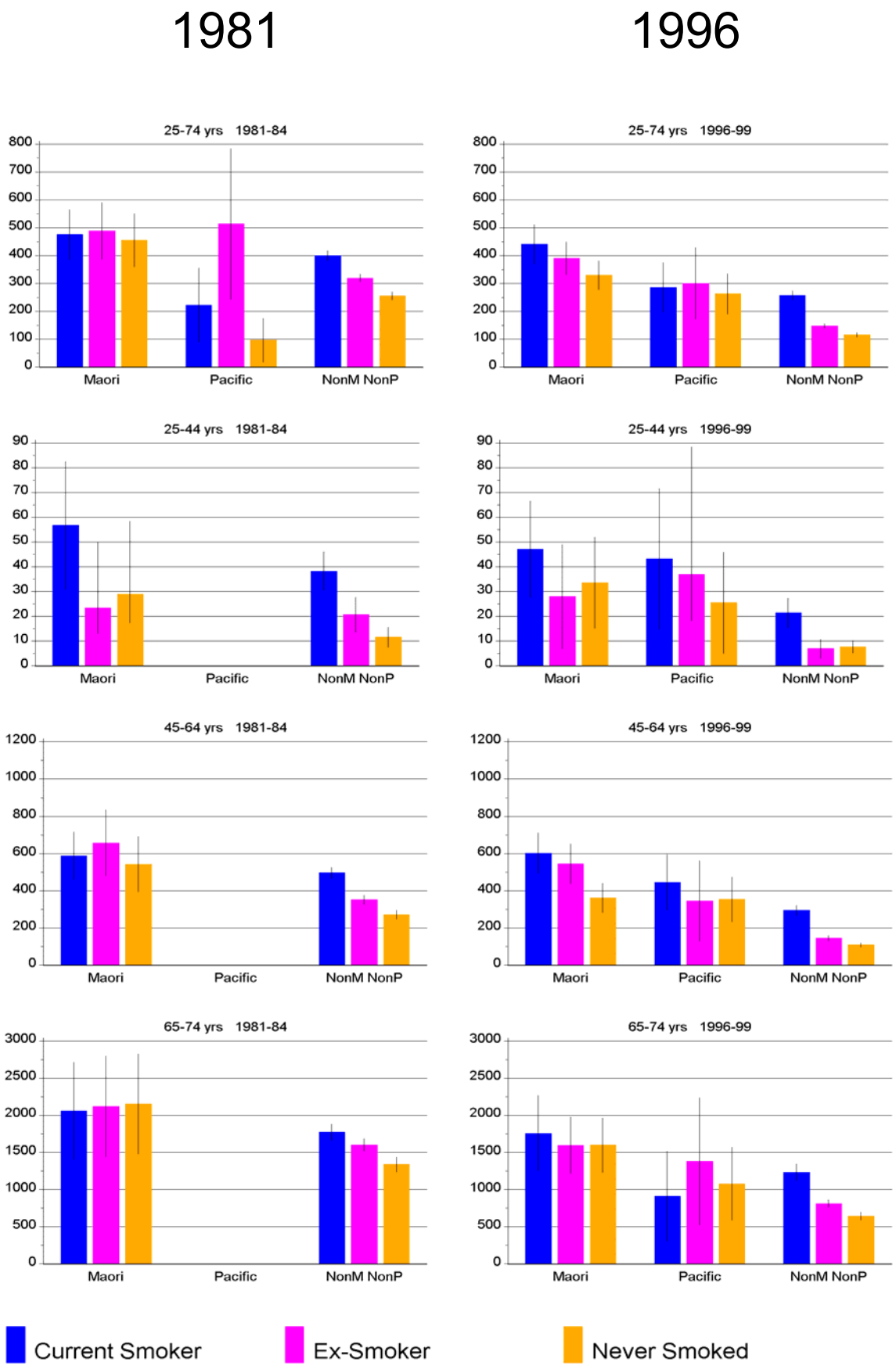


Figure 8: Female IHD Standardised Mortality Rates per 100,000 person-yrs (First Rst)

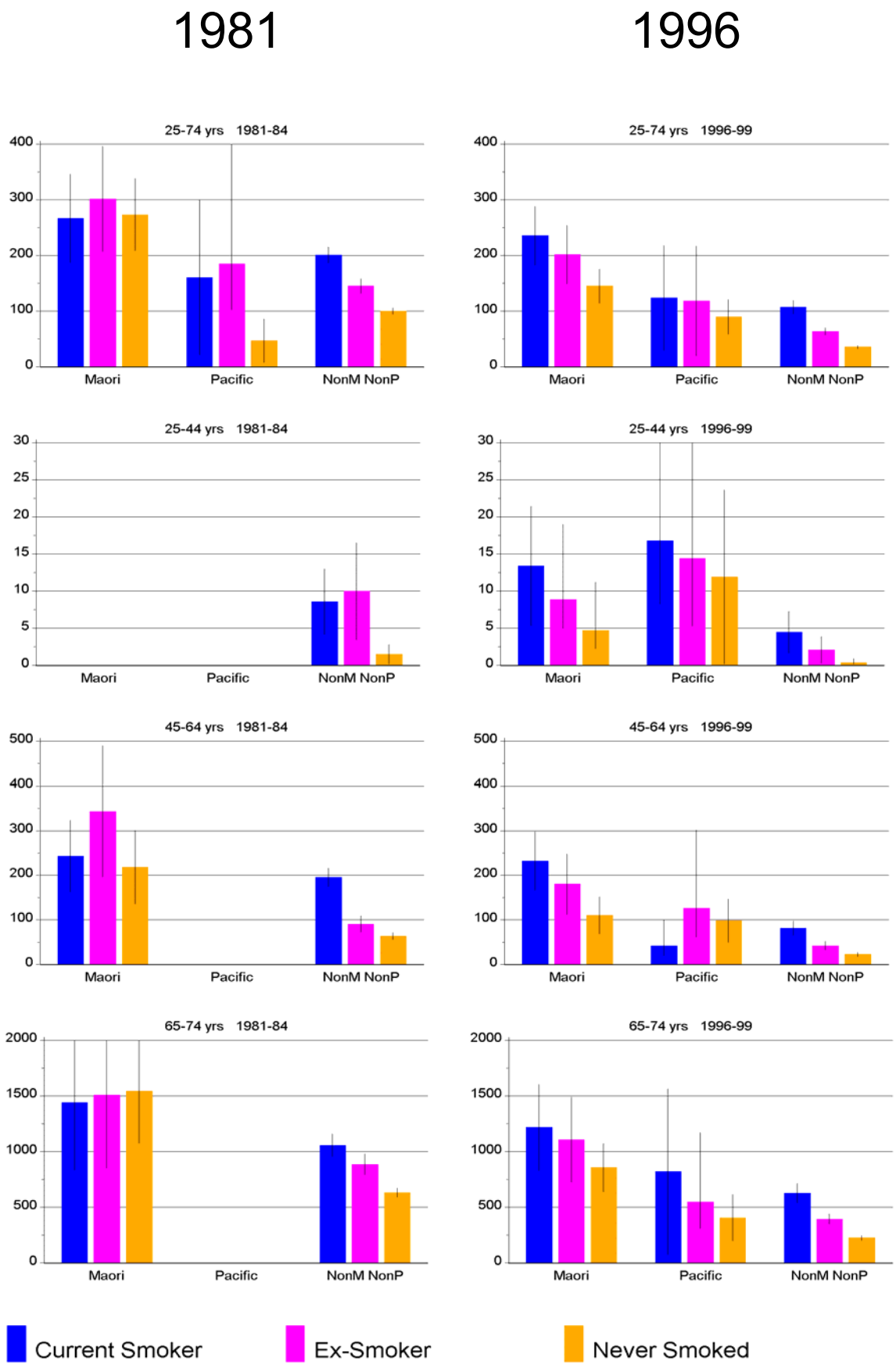


Table 15: Male IHD Standardised Rate Ratios and Rate Differences (First Restriction)

	Age Gp	SRR (reference gp never smoked)				SRD (reference gp never smoked)			
		Smoker (95% CI)		Ex-Smoker (95% CI)		Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	all age	1.04	(0.79 - 1.38)	1.07	(0.80 - 1.44)	20	(-110 - 150)	33	(-105 - 171)
Pacific	all age	2.29	(0.84 - 6.29)	5.29	(2.01 - 13.91)	125	(-30 - 280)	417	(136 - 698)
NonM-NonP	all age	1.56	(1.45 - 1.67)	1.25	(1.16 - 1.34)	144	(121 - 166)	63	(44 - 83)
All Ethnicity Combined **not adj for eth**	all age	1.52	(1.42 - 1.62)	1.23	(1.15 - 1.32)	138	(116 - 160)	62	(42 - 81)
All Ethnicity Combined **adj for eth**	all age	1.50	(1.40 - 1.61)	1.25	(1.17 - 1.35)	134	(112 - 156)	68	(48 - 89)
	25-44	2.93	(1.95 - 4.41)	1.47	(0.90 - 2.41)	27	(18 - 35)	7	(-2 - 15)
	45-64	1.69	(1.53 - 1.88)	1.32	(1.18 - 1.48)	203	(165 - 241)	94	(56 - 133)
	65-74	1.31	(1.19 - 1.44)	1.20	(1.10 - 1.31)	418	(271 - 565)	272	(142 - 403)
1996-1999									
Maori	all age	1.34	(1.07 - 1.67)	1.18	(0.95 - 1.47)	111	(24 - 198)	61	(-17 - 139)
Pacific	all age	1.08	(0.72 - 1.64)	1.14	(0.69 - 1.89)	22	(-92 - 136)	38	(-109 - 184)
NonM-NonP	all age	2.21	(2.02 - 2.42)	1.28	(1.17 - 1.39)	141	(123 - 159)	32	(21 - 43)
All Ethnicity Combined **not adj for eth**	all age	2.09	(1.93 - 2.27)	1.22	(1.13 - 1.32)	147	(129 - 165)	30	(19 - 41)
All Ethnicity Combined **adj for eth**	all age	2.03	(1.87 - 2.20)	1.26	(1.16 - 1.36)	139	(121 - 156)	35	(23 - 46)
	25-44	2.22	(1.56 - 3.16)	0.92	(0.55 - 1.52)	14	(8 - 21)	-1	(-6 - 5)
	45-64	2.37	(2.10 - 2.68)	1.34	(1.18 - 1.53)	188	(159 - 217)	47	(26 - 68)
	65-74	1.81	(1.61 - 2.03)	1.23	(1.12 - 1.36)	562	(440 - 683)	161	(88 - 233)

Male IHD SRR & SRD by Smoking Status NZCMS

Table 16: Female IHD Standardised Rate Ratios and Rate Differences (First Restriction)

	Age Gp	SRR (reference gp never smoked)				SRD (reference gp never smoked)			
		Smoker (95% CI)		Ex-Smoker (95% CI)		Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	all age	0.98	(0.67 - 1.43)	1.10	(0.75 - 1.63)	-7	(-109 - 96)	28	(-86 - 142)
Pacific	all age	3.40	(1.03 - 11.23)	3.92	(0.95 - 16.20)	113	(-31 - 257)	138	(-79 - 355)
NonM-NonP	all age	2.01	(1.83 - 2.20)	1.45	(1.30 - 1.62)	101	(85 - 116)	45	(31 - 60)
All Ethnicity Combined **not adj for eth**	all age	1.95	(1.79 - 2.13)	1.46	(1.31 - 1.62)	100	(85 - 115)	48	(33 - 62)
All Ethnicity Combined **adj for eth**	all age	1.86	(1.70 - 2.04)	1.42	(1.28 - 1.59)	94	(79 - 110)	46	(31 - 62)
	25-44	2.37	(1.03 - 5.44)	2.26	(0.86 - 5.90)	5	(0 - 10)	5	(-1 - 11)
	45-64	2.67	(2.26 - 3.15)	1.48	(1.18 - 1.87)	127	(104 - 150)	37	(13 - 60)
	65-74	1.59	(1.42 - 1.79)	1.38	(1.22 - 1.56)	396	(288 - 504)	256	(151 - 361)
1996-1999									
Maori	all age	1.62	(1.20 - 2.20)	1.39	(1.00 - 1.94)	90	(30 - 151)	57	(-4 - 117)
Pacific	all age	1.38	(0.60 - 3.17)	1.31	(0.53 - 3.23)	34	(-65 - 133)	28	(-75 - 131)
NonM-NonP	all age	3.00	(2.60 - 3.45)	1.79	(1.56 - 2.04)	71	(59 - 84)	28	(21 - 35)
All Ethnicity Combined **not adj for eth**	all age	2.93	(2.59 - 3.33)	1.72	(1.52 - 1.94)	83	(70 - 95)	31	(23 - 38)
All Ethnicity Combined **adj for eth**	all age	2.67	(2.35 - 3.03)	1.70	(1.50 - 1.92)	73	(61 - 85)	30	(23 - 38)
	25-44	3.83	(1.64 - 8.94)	2.29	(0.85 - 6.20)	4	(2 - 7)	2	(-1 - 5)
	45-64	2.87	(2.27 - 3.62)	1.74	(1.34 - 2.26)	60	(44 - 76)	24	(11 - 37)
	65-74	2.57	(2.20 - 3.00)	1.67	(1.45 - 1.92)	406	(318 - 494)	173	(120 - 226)

Female IHD SRR & SRD by Smoking Status NZCMS

3 Stroke

Table 17, Table 18, Figure 9 and Figure 10 show the stroke standardised mortality rates. For many of the strata analysed, the numbers of deaths are small and therefore the confidence intervals wide – in particular for Māori and Pacific (making ethnic comparisons difficult). As for IHD, some of the age-specific mortality rates shown in the graphs (and the tables in Appendix B) for Māori and (especially) Pacific are not presented as there were too few deaths to allow any meaningful interpretation.

Stroke mortality rates increase with age. Although the confidence intervals are quite wide, Māori females may have a higher stroke mortality rate than Māori males. Non-Māori non-Pacific stroke mortality rates for male and female appear similar.

As for IHD, standardised mortality rates for stroke have dropped markedly for non-Māori non-Pacific over the 1981-1996 15-year period, in all smoking strata. This is probably also the case for Māori females, however the trend is less clear for Māori males, and difficult to determine for Pacific.

3.1 Rate Ratios and Rate Differences

Stroke rate ratios and rate differences are given in Table 19 and Table 20, and can be conceived visually by comparing the rates shown in Figure 9 and Figure 10.

For stroke, current smokers and ex-smokers also have a rate ratio greater than 1.0 compared to never smokers. For non-Māori non-Pacific a stepwise gradient over all smoking status groups is less discernable for males, but appears to be present for non-Māori non-Pacific females. There may be a gradient for Māori females in 1996.

There also appears to be variation of rate ratios between ethnic groups, although with frequent overlap of confidence intervals. However, for all-age stroke rate ratio estimates, the confidence intervals for Māori and non-Māori non-Pacific among males do not overlap. The rate ratio point estimates for Māori are lower for stroke when compared to

non-Māori non-Pacific. Reasons for this variation by ethnicity include higher mortality rates, and a smaller or reversed gap (rate difference) in both years.

The Wald p-values for rate ratio heterogeneity by ethnicity (current smokers compared with never smokers, 25-74 years) were less than 0.05 in 1981, and less then 0.01 in 1996

Stroke rate ratios increased over time. For all-ethnicities combined, the male rate ratio increased from 1.50 (in 1981) to 1.93 (in 1996) - a similar rise to all-cause mortality, and IHD. The female rate ratio increased from 1.65 to 2.51 – a similar rise to IHD. Within ethnic groups, this pattern is seen for non-Māori non-Pacific and Māori.

The stroke rate ratio estimates are quite similar to the IHD estimates, and in that regard there is also a sex difference. For the all-age all-ethnicity rate ratios (and most ethnic specific rate ratios), females have a higher rate ratio than males for stroke, although in this case (unlike IHD), the confidence intervals do overlap for the all-ethnicity estimates (and for the 65-74 age group the male and female estimates are similar). The male rate ratios for stroke (1996 RR = 1.93) are similar to both the IHD (2.05) and all-cause (2.03) estimates, while the female estimates (1996 RR = 2.51) align more strongly with IHD (2.67) than all-cause mortality (2.01). It appears that, overall (for the 25-74 age group), smoking has a similar effect on stroke and IHD mortality in relative terms.

The pattern by age for stroke rate ratios is in the same direction (decreasing) as IHD for females in both years, and males in 1981-84. The rate ratios (all-ethnicity) for males in 1996-99 actually increase with age, however it should be taken into account that the number of stroke deaths here in males younger than 65 is relatively small.

There may also be a stronger age gradient for stroke (as with IHD) for females amongst non-Māori non-Pacific (see appendix B), although the confidence intervals for younger age groups are wide. For example, in 1996 the 25-44, 45-64 and 65-74 year old non-Māori non-Pacific female rate ratios respectively were 8.94 (95% CI 3.59-22.31), 5.90 (4.01-8.69) and 2.02 (1.51-2.71).

There appears to be little difference in rate differences for stroke by sex or year. However, the p-values for rate difference heterogeneity by ethnicity (current smokers 25-74 years) were less than 0.05 for males and females in 1981, and for males in 1996.

Table 17: Male Stroke Mortality Data – No. Deaths, Non-Std Mortality Rates and Std Mortality Rates per 100,000 person-years (First Restriction)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)	
1981-1984													
Maori	all age	33	62	104	(55 - 153)	42	40	63	(35 - 90)	27	69	86	(42 - 130)
Pacific	all age	15	55	137	(28 - 245)	9	40	116	(17 - 215)	6	17	25	(9 - 74)
NonM-NonP	all age	339	43	54	(47 - 60)	603	82	88	(79 - 97)	543	90	57	(51 - 63)
All Ethnicity Combined **not adj for eth**	all age	384	44	57	(51 - 64)	654	75	88	(80 - 97)	570	88	59	(53 - 65)
All Ethnicity Combined **adj for eth**	all age	387	44	59	(52 - 66)	654	75	89	(80 - 97)	570	88	59	(53 - 66)
	25-44	18	3	4	(2 - 7)	42	9	9	(6 - 13)	12	5	5	(1 - 8)
	45-64	105	41	43	(33 - 53)	258	83	79	(67 - 91)	138	48	46	(35 - 58)
	65-74	264	356	360	(308 - 412)	354	475	484	(424 - 545)	423	355	352	(311 - 392)
1996-1999													
Maori	all age	54	44	71	(46 - 95)	39	30	72	(42 - 103)	21	28	31	(14 - 47)
Pacific	all age	24	44	77	(37 - 117)	12	24	36	(6 - 66)	6	56	68	(7 - 128)
NonM-NonP	all age	228	19	23	(20 - 27)	219	39	52	(44 - 60)	303	45	25	(22 - 28)
All Ethnicity Combined **not adj for eth**	all age	306	23	28	(24 - 31)	270	36	54	(47 - 62)	333	43	26	(23 - 29)
All Ethnicity Combined **adj for eth**	all age	306	23	28	(24 - 31)	270	36	53	(46 - 61)	330	43	27	(23 - 30)
	25-44	21	3	3	(1 - 5)	18	4	4	(1 - 6)	12	4	3	(1 - 5)
	45-64	114	25	26	(21 - 32)	102	42	43	(34 - 53)	63	18	17	(12 - 23)
	65-74	174	147	145	(121 - 170)	150	302	313	(256 - 370)	258	163	163	(140 - 185)

Male Stroke Mortality Rates by Smoking Status NZCMS

*Random Rounded † Deaths per 100,000 Person-Years

Table 18: Female Stroke Mortality Data – No. Deaths, Non-Std Mortality Rates and Std Mortality Rates per 100,000 person-years (First Restrn)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)	
1981-1984													
Maori	all age	48	84	110	(68 - 152)	63	55	116	(68 - 164)	33	101	158	(83 - 234)
Pacific	all age	9	24	57	(9 - 105)	6	10	13	(5 - 39)	6	96	206	(108 - 464)
NonM-NonP	all age	657	54	42	(38 - 46)	408	67	76	(67 - 85)	240	68	56	(48 - 65)
All Ethnicity Combined **not adj for eth**	all age	714	54	45	(41 - 49)	468	64	79	(70 - 88)	276	71	63	(53 - 72)
All Ethnicity Combined **adj for eth**	all age	714	54	47	(43 - 52)	468	64	78	(69 - 87)	276	71	65	(55 - 76)
	25-44	27	4	5	(2 - 8)	54	13	13	(9 - 18)	15	7	8	(2 - 13)
	45-64	162	35	35	(27 - 42)	207	84	78	(65 - 92)	57	42	39	(24 - 53)
	65-74	525	257	265	(237 - 294)	207	340	358	(299 - 418)	207	362	394	(324 - 463)
1996-1999													
Maori	all age	45	42	50	(32 - 68)	39	25	82	(40 - 123)	33	48	71	(39 - 103)
Pacific	all age	33	41	71	(40 - 103)	9	26	46	(4 - 88)	6	38	61	(34 - 132)
NonM-NonP	all age	267	18	16	(14 - 18)	186	36	48	(40 - 56)	159	28	23	(19 - 27)
All Ethnicity Combined **not adj for eth**	all age	345	21	19	(17 - 21)	237	33	50	(43 - 58)	195	30	27	(23 - 31)
All Ethnicity Combined **adj for eth**	all age	345	21	20	(17 - 22)	237	33	49	(42 - 57)	195	30	28	(23 - 32)
	25-44	12	2	2	(0 - 3)	36	8	9	(5 - 12)	6	1	1	(0 - 2)
	45-64	84	14	15	(12 - 19)	108	51	54	(42 - 66)	54	23	24	(16 - 32)
	65-74	246	112	110	(95 - 125)	93	215	212	(162 - 262)	138	155	154	(125 - 183)

Female Stroke Mortality Rates by Smoking Status NZCMS

*Random Rounded † Deaths per 100,000 Person-Years

Figure 9: Male Stroke Standardised Mortality Rates per 100,000 person-yrs (First Rst)

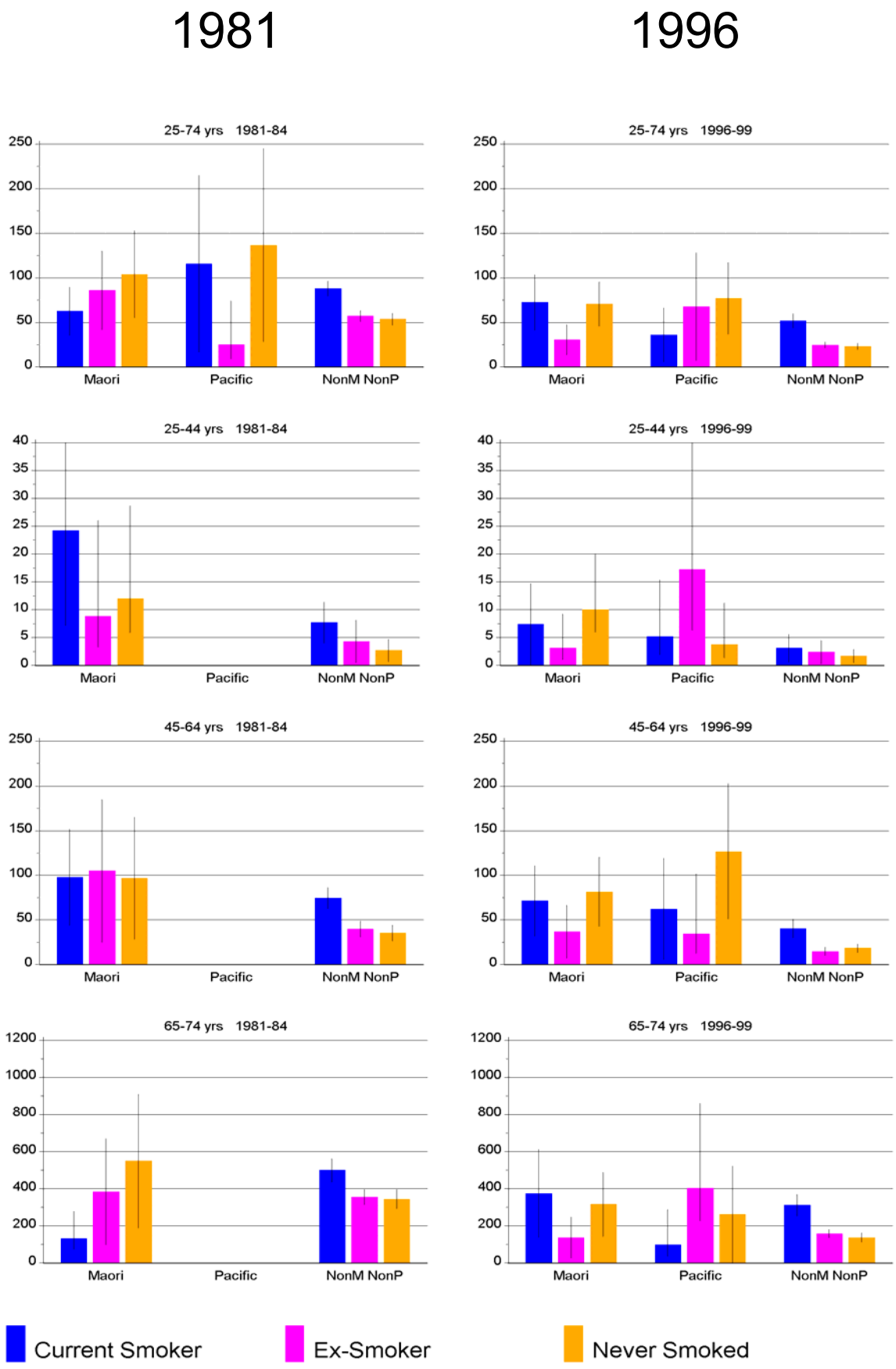


Figure 10: Female Stroke Standardised Mortality Rates per 100,000 person-yrs (First Rst)

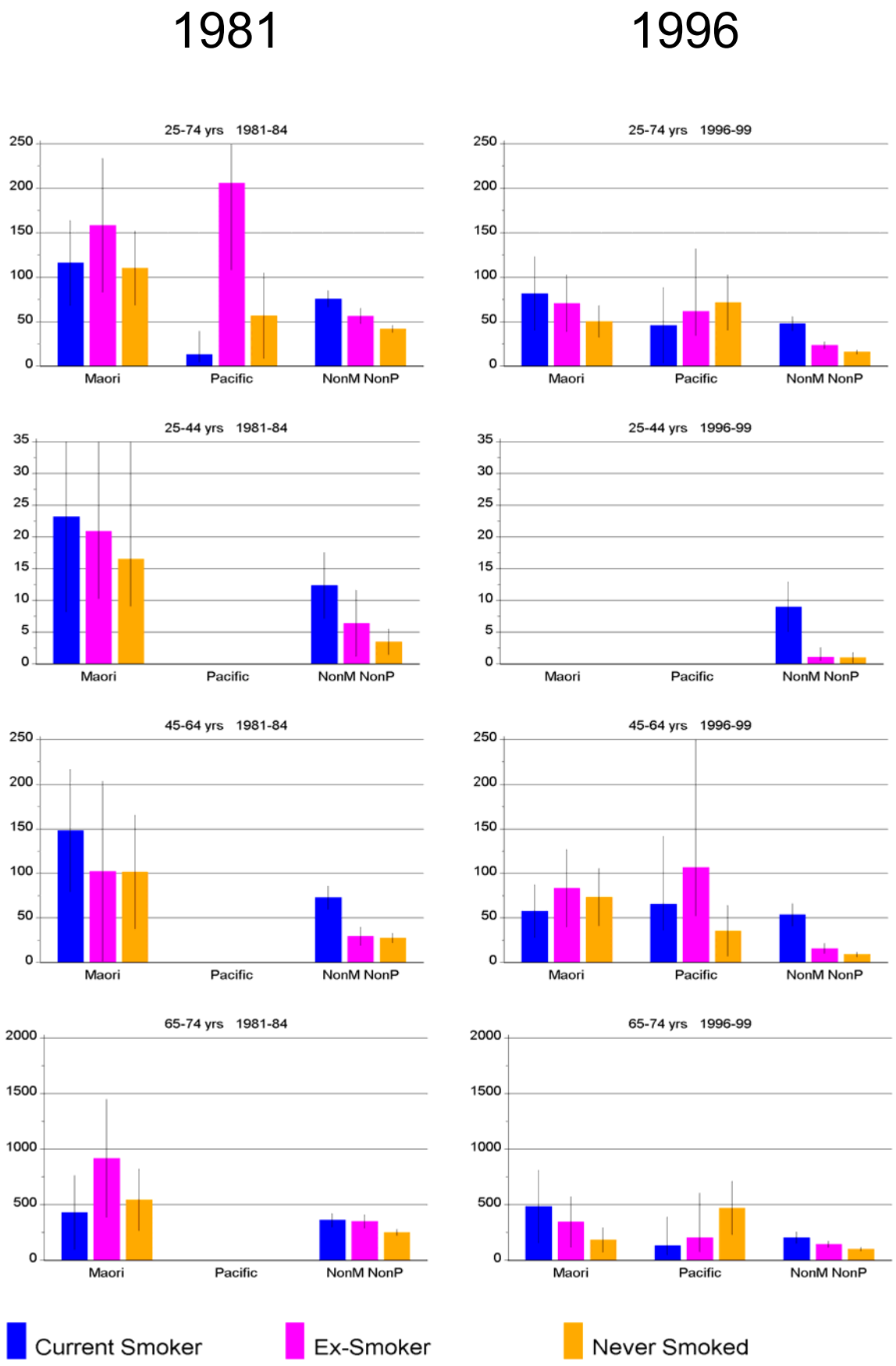


Table 19: Male Stroke Standardised Rate Ratios and Rate Differences (First Restriction)

	Age Gp	SRR (reference gp never smoked)				SRD (reference gp never smoked)			
		Smoker (95% CI)		Ex-Smoker (95% CI)		Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	all age	0.60	(0.32 - 1.14)	0.83	(0.41 - 1.66)	-41	(-97 - 15)	-18	(-84 - 48)
Pacific	all age	0.85	(0.26 - 2.72)	0.18	(0.02 - 1.51)	-21	(-168 - 126)	-112	(-231 - 7)
NonM-NonP	all age	1.64	(1.40 - 1.93)	1.07	(0.90 - 1.26)	34	(23 - 45)	4	(-6 - 13)
All Ethnicity Combined **not adj for eth**	all age	1.54	(1.32 - 1.79)	1.02	(0.87 - 1.19)	31	(20 - 42)	1	(-8 - 10)
All Ethnicity Combined **adj for eth**	all age	1.50	(1.29 - 1.75)	1.01	(0.85 - 1.19)	30	(19 - 41)	0	(-9 - 10)
	25-44	2.15	(0.98 - 4.71)	1.04	(0.36 - 2.99)	5	(0 - 10)	0	(-5 - 5)
	45-64	1.83	(1.37 - 2.44)	1.08	(0.76 - 1.53)	36	(20 - 52)	4	(-12 - 19)
	65-74	1.35	(1.11 - 1.63)	0.98	(0.81 - 1.18)	124	(45 - 204)	-8	(-75 - 58)
1996-1999									
Maori	all age	1.02	(0.59 - 1.78)	0.43	(0.23 - 0.83)	2	(-38 - 41)	-40	(-70 - -10)
Pacific	all age	0.47	(0.17 - 1.26)	0.88	(0.31 - 2.47)	-41	(-91 - 9)	-9	(-82 - 63)
NonM-NonP	all age	2.23	(1.81 - 2.76)	1.07	(0.88 - 1.30)	29	(20 - 37)	2	(-3 - 6)
All Ethnicity Combined **not adj for eth**	all age	1.95	(1.61 - 2.36)	0.93	(0.78 - 1.12)	26	(18 - 35)	-2	(-7 - 3)
All Ethnicity Combined **adj for eth**	all age	1.93	(1.59 - 2.34)	0.96	(0.80 - 1.15)	26	(17 - 34)	-1	(-6 - 4)
	25-44	1.29	(0.55 - 3.00)	1.03	(0.40 - 2.69)	1	(-2 - 4)	0	(-3 - 3)
	45-64	1.65	(1.20 - 2.25)	0.66	(0.46 - 0.95)	17	(6 - 28)	-9	(-17 - -1)
	65-74	2.15	(1.68 - 2.76)	1.12	(0.90 - 1.39)	168	(105 - 230)	17	(-16 - 51)

Male Stroke SRR & SRD by Smoking Status NZCMS

Table 20: Female Stroke Standardised Rate Ratios and Rate Differences (First Restriction)

	Age Gp	SRR (reference gp never smoked)				SRD (reference gp never smoked)			
		Smoker (95% CI)		Ex-Smoker (95% CI)		Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	all age	1.05	(0.60 - 1.84)	1.44	(0.78 - 2.64)	6	(-58 - 69)	48	(-38 - 134)
Pacific	all age	0.23	(0.03 - 1.98)	3.62	(0.80 - 16.49)	-44	(-98 - 11)	149	(-114 - 412)
NonM-NonP	all age	1.80	(1.55 - 2.10)	1.34	(1.12 - 1.61)	34	(24 - 44)	14	(5 - 24)
All Ethnicity Combined **not adj for eth**	all age	1.77	(1.53 - 2.05)	1.41	(1.18 - 1.67)	34	(24 - 44)	18	(8 - 28)
All Ethnicity Combined **adj for eth**	all age	1.65	(1.42 - 1.92)	1.39	(1.15 - 1.67)	31	(21 - 41)	18	(7 - 30)
	25-44	2.48	(1.27 - 4.82)	1.42	(0.58 - 3.50)	8	(2 - 14)	2	(-4 - 8)
	45-64	2.27	(1.72 - 3.00)	1.12	(0.73 - 1.72)	44	(29 - 59)	4	(-12 - 20)
	65-74	1.35	(1.11 - 1.65)	1.48	(1.21 - 1.83)	93	(27 - 159)	129	(53 - 204)
1996-1999									
Maori	all age	1.62	(0.87 - 3.01)	1.41	(0.79 - 2.49)	31	(-14 - 76)	20	(-16 - 57)
Pacific	all age	0.64	(0.23 - 1.78)	0.86	(0.25 - 2.94)	-26	(-78 - 27)	-10	(-87 - 67)
NonM-NonP	all age	3.01	(2.44 - 3.72)	1.47	(1.19 - 1.83)	32	(24 - 40)	8	(3 - 12)
All Ethnicity Combined **not adj for eth**	all age	2.64	(2.17 - 3.20)	1.42	(1.16 - 1.72)	31	(23 - 39)	8	(3 - 13)
All Ethnicity Combined **adj for eth**	all age	2.51	(2.06 - 3.05)	1.40	(1.15 - 1.72)	30	(22 - 38)	8	(3 - 13)
	25-44	5.28	(2.33 - 11.94)	0.52	(0.11 - 2.53)	7	(3 - 11)	-1	(-2 - 1)
	45-64	3.54	(2.54 - 4.91)	1.56	(1.04 - 2.33)	39	(27 - 51)	9	(0 - 17)
	65-74	1.93	(1.47 - 2.54)	1.40	(1.11 - 1.77)	102	(50 - 154)	44	(11 - 77)

Female Stroke SRR & SRD by Smoking Status NZCMS

Chapter 6: Results – part 2 (multivariable analysis)

Part 2 Results Summary

Multivariable analysis revealed a moderate degree of confounding by socio-economic position. Adjustment for SEP, as measured by a range of variables, reduced the age and ethnicity adjusted poisson regression estimates for the all-age all-ethnicity group by 21-28% for males and 5-9% for females in 1981-84, and by 33-38% for males and 21-25% for females in 1996-99 (percentages calculated using the excess rate ratios). Thus, confounding by SEP was more pronounced among males, and increased over time for both males and females. Rate ratios adjusted for SEP still demonstrated heterogeneity by time and ethnicity.

Results for the Part 2 analyses are also presented separately for all-cause mortality, ischaemic heart disease, and stroke, in tabular form. Each table shows the following rate ratios (compared to never smokers) for current and ex-smokers:

- Age-Standardised Rate Ratios (from Part 1 analysis) – for comparison
- Rate Ratios adjusted for confounding by age
- Rate Ratios adjusted for confounding by age and socioeconomic position (SEP)
- 95% Confidence Intervals for each point estimate

These data are broken down by year, age, sex, and ethnicity.

All data have been weighted to adjust for linkage bias.

The age-standardised rate ratios are from analysis of the first restricted cohort (as presented previously), whereas the adjusted rate ratios (using poisson regression) are from analysis of the second restricted cohort (this cohort only include participants from the first restriction that have complete data for the SEP variables).

Socioeconomic variables for which rate ratios have been adjusted (comprising SEP) are: education, car access, household equivalised income, marital status, NZDep, labour force status, and housing tenure.

1 All-Cause Mortality – Adjusted Estimates

The observed strength of the association between smoking and mortality as rate ratios was presented in Part 1. The extent to which this (relative) association is due to confounding by factors such as socio-economic variables, has been investigated by multivariable analysis. The adjusted rate ratios (for all-cause mortality) are shown in Table 21 and Table 22.

There are some key features of these results:

Firstly, the age-adjusted rate ratios are similar to the age standardised rate ratios for most strata, particularly the estimates for current smokers and for all ethnicity combined. Each is produced using a different method, and on cohorts with a different level of restriction. This tends to imply modest selection bias between the two restrictions overall, and helps to validate the standardised (part 1) results. However, there are some notable differences. For example current smoking Pacific females in 1981 have an all-cause standardised rate ratio (SRR) of 0.66 (0.37-1.20) and an age-adjusted RR of 0.44 (0.17-1.10), and in 1996 the respective estimates are 1.05 (0.75-1.48) and 1.46 (0.96-2.22). A notable change is also seen for current smoking Māori males and females in 1981. It should be taken into account however that some of the 95% confidence intervals for these estimates, especially for Pacific, are reasonably wide.

Secondly, the association of current smoking and all-cause mortality in 1981 appears to be only modestly confounded by socio-economic position (SEP). After controlling for SEP, the excess rate ratios for current smokers (all ethnicity, age and ethnicity adjusted) decrease by 23% for males and 9% for females (ie. 'Adj RR – Age' compared with 'Adj RR – Age + SEP'). There is essentially no change for ex-smokers. Accordingly, there was still a rate ratio of 1.44 (1.36 to 1.52) for current smoking males compared to never smoking males in 1981 (adjusting for age, ethnicity and socio-economic position) and a rate ratio of 1.50 (1.40 to 1.60) for females.

Third, the association of current smoking and all-cause mortality in 1996 appears to be more confounded by socio-economic position (SEP) than in 1981. After controlling for

both age and SEP, the excess rate ratios for current smokers (all ethnicity, ethnicity adjusted) decrease by 33% for males and 21% for females (age adjusted vs fully adjusted). Again there is very little change for ex-smokers overall. Despite the greater shift for current smokers in 1996 (compared to 1981) there was still a rate ratio of 1.68 (1.59 to 1.78) for current smoking males (after full adjustment) and a rate ratio of 1.83 (1.70 to 1.95) for females.

The fact that the rate ratios for 1996 decrease more than those for 1981 after controlling for confounding means that the final (age and SEP adjusted) estimates for 1981 and 1996 are closer together – ie. there is less, but still notable, change in relative risk over time than seen in the unadjusted / standardised results. Thus, increasing confounding by SEP over time drives some of the increasing relative risk of mortality by smoking over time.

These results also show that the rate ratios for females are less confounded by socio-economic position than males - the estimates shift less for females. This is particularly apparent in 1981, when the all-cause excess rate ratios for females only changed by 9% (1.55 to 1.50) after full adjustment.

Finally, it is important to note that confounding of the (current) smoking-mortality association appears to occur across all ethnic groups and all age groups to a similar extent (ie. the rate ratios shift by a similar degree). Although some of the rate ratios change more for non-Māori non-Pacific on an absolute scale, if a relative scale is applied the change is greater for Māori. For some of the estimates the opposite pattern is seen. There is also more confounding in 1996 compared to 1981 across all the ethnic groups. The heterogeneity of rate ratios between ethnic groups is still very notable even after fully adjusting for socio-economic position (although some of the ethnic specific estimates are imprecise). In other words, the heterogeneity (or effect modification) by ethnicity seen in the standardised results cannot be attributed to differences between ethnic groups in socio-economic status. It is also important to note that the fully adjusted rate ratios for Māori are still significantly over 1.0 for current smokers in 1996 (male RR=1.25, CI 1.08-1.46, and female RR=1.25, CI 1.04-1.50), and for female ex-smokers in both years (1981 RR=1.40, CI 1.05-1.86, 1996 RR=1.35 CI 1.11-1.64).

The degree of confounding by SEP, and patterns by time and sex, are discussed in more detail in Chapter 8 (Discussion), section 3.5.2, page 141.

Table 21: Male All-Cause Rate Ratios – standardised, and adjusted for confounding (Second Restriction)

	Age Gp	Current Smokers (reference gp never smoked)						Ex-Smokers (reference gp never smoked)					
		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)	
1981-1984													
Maori	all age	1.19	(1.02-1.39)	0.98	(0.79-1.21)	0.91	(0.73-1.12)	1.08	(0.91-1.28)	0.95	(0.75-1.21)	0.97	(0.76-1.23)
Pacific	all age	1.02	(0.67-1.55)	1.08	(0.62-1.89)	1.08	(0.61-1.89)	1.76	(1.14-2.74)	1.73	(0.95-3.17)	1.71	(0.93-3.15)
NonM-NonP	all age	1.68	(1.61-1.75)	1.65	(1.55-1.75)	1.51	(1.42-1.60)	1.29	(1.23-1.35)	1.34	(1.26-1.42)	1.31	(1.23-1.39)
All Ethnicity Combined **not adj for eth**	all age	1.63	(1.57-1.70)	1.59	(1.50-1.68)	1.44	(1.36-1.52)	1.25	(1.20-1.31)	1.30	(1.23-1.37)	1.27	(1.20-1.34)
All Ethnicity Combined **adj for eth**	all age	1.59	(1.53-1.66)	1.57	(1.48-1.66)	1.44	(1.36-1.52)	1.27	(1.21-1.32)	1.31	(1.23-1.38)	1.28	(1.21-1.35)
	25-44	1.38	(1.22-1.58)	1.28	(1.08-1.52)	1.16	(0.98-1.39)	0.98	(0.82-1.16)	1.02	(0.82-1.26)	1.03	(0.83-1.27)
	45-64	1.62	(1.52-1.73)	1.70	(1.56-1.85)	1.57	(1.44-1.71)	1.19	(1.11-1.28)	1.27	(1.16-1.39)	1.26	(1.15-1.37)
	65-74	1.61	(1.52-1.71)	1.61	(1.49-1.74)	1.47	(1.36-1.59)	1.39	(1.31-1.47)	1.41	(1.31-1.52)	1.37	(1.27-1.48)
1996-1999													
Maori	all age	1.51	(1.35-1.69)	1.49	(1.28-1.74)	1.25	(1.08-1.46)	1.09	(0.97-1.22)	1.04	(0.88-1.23)	1.03	(0.88-1.21)
Pacific	all age	1.18	(0.94-1.47)	1.19	(0.86-1.65)	1.08	(0.78-1.50)	1.40	(1.09-1.80)	1.70	(1.20-2.43)	1.87	(1.30-2.67)
NonM-NonP	all age	2.22	(2.12-2.33)	2.16	(2.03-2.30)	1.82	(1.71-1.94)	1.36	(1.30-1.42)	1.43	(1.35-1.51)	1.38	(1.30-1.46)
All Ethnicity Combined **not adj for eth**	all age	2.13	(2.05-2.23)	2.11	(2.00-2.23)	1.70	(1.61-1.80)	1.28	(1.22-1.33)	1.37	(1.30-1.44)	1.32	(1.25-1.39)
All Ethnicity Combined **adj for eth**	all age	2.05	(1.97-2.14)	2.01	(1.90-2.12)	1.68	(1.59-1.78)	1.30	(1.25-1.36)	1.38	(1.31-1.46)	1.33	(1.26-1.40)
	25-44	1.57	(1.40-1.76)	1.62	(1.40-1.88)	1.34	(1.14-1.56)	1.04	(0.88-1.23)	1.07	(0.88-1.30)	1.05	(0.86-1.28)
	45-64	2.06	(1.93-2.20)	2.10	(1.93-2.29)	1.74	(1.59-1.90)	1.25	(1.16-1.33)	1.32	(1.21-1.44)	1.29	(1.18-1.41)
	65-74	2.18	(2.05-2.32)	2.14	(1.98-2.32)	1.84	(1.70-2.00)	1.42	(1.34-1.49)	1.45	(1.36-1.54)	1.40	(1.31-1.49)

Male All-Cause Adj RR NZCMS n

* age-standardised [First Restricted Cohort]

† adjusted for age (5 year bands) [Second Restricted Cohort]

‡ adjusted for age and socio-economic position (SEP) = education, car access, household equivalised income, marital status, NZDep, labour force, housing tenure [Second Restricted Cohort]

Table 22: Female All-Cause Rate Ratios – standardised, and adjusted for confounding (Second Restriction)

	Age Gp	Current Smokers (reference gp never smoked)						Ex-Smokers (reference gp never smoked)					
		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)	
1981-1984													
Maori	all age	1.06	(0.89-1.27)	1.29	(1.01-1.64)	1.22	(0.95-1.55)	1.37	(1.13-1.67)	1.41	(1.06-1.87)	1.40	(1.05-1.86)
Pacific	all age	0.66	(0.37-1.20)	0.44	(0.17-1.10)	0.43	(0.17-1.08)	2.15	(1.30-3.53)	1.53	(0.72-3.23)	1.40	(0.66-3.00)
NonM-NonP	all age	1.59	(1.52-1.67)	1.60	(1.50-1.71)	1.54	(1.44-1.65)	1.45	(1.38-1.53)	1.46	(1.35-1.57)	1.47	(1.36-1.58)
All Ethnicity Combined **not adj for eth**	all age	1.59	(1.52-1.66)	1.62	(1.52-1.73)	1.54	(1.44-1.64)	1.48	(1.41-1.56)	1.48	(1.38-1.59)	1.49	(1.39-1.60)
All Ethnicity Combined **adj for eth**	all age	1.49	(1.42-1.56)	1.55	(1.46-1.66)	1.50	(1.40-1.60)	1.45	(1.38-1.54)	1.46	(1.35-1.56)	1.47	(1.37-1.58)
	25-44	1.08	(0.92-1.27)	1.05	(0.85-1.30)	0.98	(0.79-1.22)	1.14	(0.92-1.41)	0.91	(0.69-1.19)	0.90	(0.69-1.19)
	45-64	1.54	(1.43-1.65)	1.71	(1.56-1.89)	1.63	(1.48-1.80)	1.45	(1.32-1.59)	1.49	(1.33-1.68)	1.49	(1.33-1.68)
	65-74	1.54	(1.44-1.65)	1.59	(1.46-1.73)	1.55	(1.42-1.69)	1.53	(1.42-1.64)	1.54	(1.41-1.68)	1.55	(1.42-1.70)
1996-1999													
Maori	all age	1.45	(1.27-1.66)	1.43	(1.19-1.71)	1.25	(1.04-1.50)	1.48	(1.29-1.70)	1.37	(1.13-1.66)	1.35	(1.11-1.64)
Pacific	all age	1.05	(0.75-1.48)	1.46	(0.96-2.22)	1.51	(0.99-2.31)	1.30	(0.92-1.84)	1.64	(0.99-2.72)	1.71	(1.03-2.84)
NonM-NonP	all age	2.20	(2.09-2.33)	2.23	(2.08-2.40)	1.99	(1.85-2.15)	1.57	(1.50-1.66)	1.66	(1.55-1.77)	1.64	(1.53-1.75)
All Ethnicity Combined **not adj for eth**	all age	2.19	(2.08-2.30)	2.25	(2.11-2.40)	1.92	(1.79-2.05)	1.56	(1.49-1.64)	1.64	(1.54-1.75)	1.62	(1.52-1.73)
All Ethnicity Combined **adj for eth**	all age	2.01	(1.91-2.12)	2.05	(1.92-2.19)	1.83	(1.70-1.95)	1.54	(1.47-1.62)	1.62	(1.53-1.73)	1.61	(1.51-1.71)
	25-44	1.20	(1.03-1.40)	1.23	(1.01-1.50)	1.04	(0.85-1.28)	0.98	(0.81-1.18)	1.06	(0.85-1.33)	1.04	(0.83-1.30)
	45-64	1.89	(1.75-2.05)	2.07	(1.87-2.28)	1.86	(1.68-2.06)	1.42	(1.31-1.54)	1.45	(1.30-1.60)	1.45	(1.30-1.61)
	65-74	2.32	(2.16-2.49)	2.29	(2.09-2.51)	2.07	(1.89-2.27)	1.78	(1.67-1.89)	1.86	(1.72-2.01)	1.83	(1.70-1.98)

Female All-Cause Adj RR NZCMS n

* age-standardised [First Restricted Cohort]

† adjusted for age (5 year bands) [Second Restricted Cohort]

‡ adjusted for age and socio-economic position (SEP) = education, car access, household equivalised income, marital status, NZDep, labour force, housing tenure [Second Restricted Cohort]

2 IHD – Adjusted Estimates

The adjusted rate ratios for IHD mortality are shown in Table 23 and Table 24. Some of the estimates from the regression analysis were invalid (due to small cell numbers), and these are left blank.

The current smoker age-adjusted rate ratios for IHD are somewhat similar to the age standardised rate ratios. There are notable differences for some age strata, for females in 1996, and for Pacific males in 1981, however the confidence intervals are wider.

The association of current smoking and IHD mortality in 1981 also appears to be only modestly confounded by socio-economic position (SEP). There is also less shift in the IHD rate ratios after controlling for confounding in 1981, when compared with 1996, and an especially small shift for females in 1981. For 1981, adjustment for SEP reduced the already age and ethnicity adjusted excess rate ratios by a further 21% for males and 9% for females, giving final adjusted estimates of 1.38 (1.25-1.51) and 1.78 (1.57-2.02) respectively. For 1996 there are larger decreases of 36% for males and 21% for females, giving final adjusted estimates of 1.61 (1.44-1.80) and 2.52 (2.12-2.99).

As with all-cause mortality, the 1981 and 1996 results are closer together when fully adjusted. Therefore, increasing confounding by SEP over time drives some of the increasing relative risk of smoking and IHD mortality.

It can also be noted for IHD that the sex difference, and heterogeneity by ethnicity, in rate ratios persist after adjusting for age and socio-economic position.

Table 23: Male IHD Rate Ratios – standardised, and adjusted for confounding (Second Restriction)

	Age Gp	Current Smokers (reference gp never smoked)						Ex-Smokers (reference gp never smoked)					
		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)	
1981-1984													
Maori	all age	1.04	(0.79-1.38)	0.88	(0.60-1.30)	0.85	(0.58-1.26)	1.07	(0.80-1.44)	0.94	(0.63-1.43)	0.93	(0.61-1.41)
Pacific	all age	2.29	(0.84-6.29)	2.75	(0.79-9.52)	--	--	5.29	(2.01-13.91)	4.77	(1.30-17.52)	--	--
NonM-NonP	all age	1.56	(1.45-1.67)	1.52	(1.38-1.67)	1.41	(1.28-1.55)	1.25	(1.16-1.34)	1.26	(1.14-1.38)	1.22	(1.11-1.34)
All Ethnicity Combined **not adj for eth**	all age	1.52	(1.42-1.62)	1.48	(1.35-1.63)	1.38	(1.25-1.51)	1.23	(1.15-1.32)	1.24	(1.13-1.35)	1.21	(1.10-1.32)
All Ethnicity Combined **adj for eth**	all age	1.50	(1.40-1.61)	1.48	(1.34-1.62)	1.38	(1.25-1.51)	1.25	(1.17-1.35)	1.24	(1.14-1.36)	1.21	(1.11-1.33)
	25-44	2.93	(1.95-4.41)	3.36	(2.04-5.53)	2.92	(1.76-4.84)	1.47	(0.90-2.41)	1.84	(1.01-3.34)	1.77	(0.97-3.23)
	45-64	1.69	(1.53-1.88)	1.75	(1.53-2.00)	1.65	(1.44-1.89)	1.32	(1.18-1.48)	1.35	(1.17-1.55)	1.32	(1.15-1.52)
	65-74	1.31	(1.19-1.44)	1.24	(1.10-1.40)	1.16	(1.02-1.31)	1.20	(1.10-1.31)	1.15	(1.03-1.28)	1.12	(1.00-1.25)
1996-1999													
Maori	all age	1.34	(1.07-1.67)	1.29	(0.95-1.76)	1.13	(0.82-1.54)	1.18	(0.95-1.47)	1.08	(0.79-1.47)	1.06	(0.78-1.45)
Pacific	all age	1.08	(0.72-1.64)	1.07	(0.57-2.02)	0.99	(0.52-1.87)	1.14	(0.69-1.89)	1.32	(0.64-2.73)	1.46	(0.70-3.04)
NonM-NonP	all age	2.21	(2.02-2.42)	2.13	(1.90-2.40)	1.74	(1.54-1.97)	1.28	(1.17-1.39)	1.31	(1.18-1.46)	1.26	(1.13-1.40)
All Ethnicity Combined **not adj for eth**	all age	2.09	(1.93-2.27)	2.04	(1.83-2.27)	1.62	(1.45-1.81)	1.22	(1.13-1.32)	1.27	(1.15-1.40)	1.22	(1.11-1.35)
All Ethnicity Combined **adj for eth**	all age	2.03	(1.87-2.20)	1.95	(1.75-2.18)	1.61	(1.44-1.80)	1.26	(1.16-1.36)	1.28	(1.16-1.41)	1.23	(1.12-1.36)
	25-44	2.22	(1.56-3.16)	2.26	(1.44-3.53)	1.77	(1.12-2.81)	0.92	(0.55-1.52)	0.88	(0.47-1.65)	0.83	(0.44-1.55)
	45-64	2.37	(2.10-2.68)	2.32	(1.98-2.73)	1.90	(1.61-2.24)	1.34	(1.18-1.53)	1.40	(1.19-1.64)	1.36	(1.16-1.60)
	65-74	1.81	(1.61-2.03)	1.72	(1.49-1.99)	1.46	(1.26-1.69)	1.23	(1.12-1.36)	1.19	(1.06-1.34)	1.15	(1.02-1.29)

Male IHD Adj RR NZCMS n

* age-standardised [First Restricted Cohort]

† adjusted for age (5 year bands) [Second Restricted Cohort]

‡ adjusted for age and socio-economic position (SEP) = education, car access, household equivalised income, marital status, NZDep, labour force, housing tenure [Second Restricted Cohort]

Table 24: Female IHD Rate Ratios – standardised, and adjusted for confounding (Second Restriction)

	Age Gp	Current Smokers (reference gp never smoked)						Ex-Smokers (reference gp never smoked)					
		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)	
1981-1984													
Maori	all age	0.98	(0.67-1.43)	1.21	(0.71-2.06)	--	--	1.10	(0.75-1.63)	0.94	(0.48-1.82)	--	--
Pacific	all age	3.40	(1.03-11.23)	--	--	--	--	3.92	(0.95-16.20)	--	--	--	--
NonM-NonP	all age	2.01	(1.83-2.20)	1.92	(1.69-2.18)	1.84	(1.62-2.09)	1.45	(1.30-1.62)	1.40	(1.21-1.62)	1.42	(1.22-1.64)
All Ethnicity Combined **not adj for eth**	all age	1.95	(1.79-2.13)	1.90	(1.68-2.15)	1.81	(1.60-2.05)	1.46	(1.31-1.62)	1.39	(1.20-1.60)	1.40	(1.21-1.62)
All Ethnicity Combined **adj for eth**	all age	1.86	(1.70-2.04)	1.86	(1.64-2.11)	1.78	(1.57-2.02)	1.42	(1.28-1.59)	1.37	(1.19-1.58)	1.39	(1.20-1.60)
	25-44	2.37	(1.03-5.44)	3.93	(1.36-11.31)	--	--	2.26	(0.86-5.90)	4.23	(1.30-13.78)	--	--
	45-64	2.67	(2.26-3.15)	2.74	(2.22-3.40)	2.57	(2.07-3.19)	1.48	(1.18-1.87)	1.33	(0.99-1.80)	1.33	(0.99-1.79)
	65-74	1.59	(1.42-1.79)	1.59	(1.37-1.84)	1.55	(1.33-1.80)	1.38	(1.22-1.56)	1.41	(1.21-1.65)	1.43	(1.23-1.67)
1996-1999													
Maori	all age	1.62	(1.20-2.20)	1.55	(1.00-2.42)	1.33	(0.85-2.09)	1.39	(1.00-1.94)	1.61	(1.02-2.52)	1.59	(1.01-2.50)
Pacific	all age	1.38	(0.60-3.17)	2.46	(0.96-6.29)	--	--	1.31	(0.53-3.23)	1.19	(0.27-5.18)	--	--
NonM-NonP	all age	3.00	(2.60-3.45)	3.36	(2.80-4.03)	2.87	(2.38-3.46)	1.79	(1.56-2.04)	1.91	(1.60-2.27)	1.85	(1.56-2.21)
All Ethnicity Combined **not adj for eth**	all age	2.93	(2.59-3.33)	3.24	(2.74-3.82)	2.65	(2.23-3.13)	1.72	(1.52-1.94)	1.88	(1.60-2.21)	1.82	(1.55-2.14)
All Ethnicity Combined **adj for eth**	all age	2.67	(2.35-3.03)	2.93	(2.48-3.47)	2.52	(2.12-2.99)	1.70	(1.50-1.92)	1.87	(1.59-2.19)	1.82	(1.55-2.14)
	25-44	3.83	(1.64-8.94)	2.54	(0.97-6.66)	--	--	2.29	(0.85-6.20)	1.71	(0.55-5.30)	--	--
	45-64	2.87	(2.27-3.62)	3.35	(2.44-4.59)	2.80	(2.03-3.86)	1.74	(1.34-2.26)	2.15	(1.54-3.01)	2.11	(1.51-2.95)
	65-74	2.57	(2.20-3.00)	2.87	(2.36-3.49)	2.50	(2.06-3.05)	1.67	(1.45-1.92)	1.88	(1.57-2.24)	1.84	(1.54-2.19)

Female IHD Adj RR NZCMS n

* age-standardised [First Restricted Cohort]

† adjusted for age (5 year bands) [Second Restricted Cohort]

‡ adjusted for age and socio-economic position (SEP) = education, car access, household equivalised income, marital status, NZDep, labour force, housing tenure [Second Restricted Cohort]

3 Stroke – Adjusted Estimates

The adjusted rate ratios for stroke mortality are shown in Table 25 and Table 26. Some of the estimates from the regression analysis were invalid (due to small cell numbers), and these are left blank.

For stroke mortality, there are also some strata that have a notable difference between age-standardised and age-adjusted rate ratio estimates. These also tend to have wider confidence intervals.

The association of current smoking and stroke mortality in 1981 also appears to be modestly confounded by socio-economic position (SEP). And, as with all-cause and IHD mortality, there is less shift in the rate ratios after controlling for confounding in 1981, when compared with 1996. For 1981, the all-age all-ethnicity excess rate ratio decreases by 28% for males and 5% for females, giving final adjusted estimates of 1.44 (1.15-1.81) and 1.74 (1.42-2.13) respectively. For 1996 there are larger decreases of 38% for males and 25% for females, giving final adjusted estimates of 1.66 (1.27-2.17) and 2.20 (1.66-2.90). The 1981 and 1996 stroke results are therefore closer together when fully adjusted.

Following the pattern for IHD, female risk of stroke mortality from smoking remains higher than male mortality risk.

For females in 1996, it is possible that there is less of an age gradient (for all-ethnicity) after fully adjusting for confounding (range 5.20 to 1.55 as compared with 7.65 to 1.72), however the confidence intervals are quite wide.

It is impossible to determine whether or not there remains any heterogeneity in the stroke rate ratios by ethnicity after full adjustment. Many of the estimates cannot be determined using the regression model due to small numbers within the cells analysed, consequently producing an invalid result. However, given the persistent heterogeneity seen for all-cause and IHD mortality after full adjustment, the heterogeneity in standardised rate ratios for stroke by ethnicity is unlikely to be due to confounding by socio-economic status and would remain.

Table 25: Male Stroke Rate Ratios – standardised, and adjusted for confounding (Second Restriction)

	Age Gp	Current Smokers (reference gp never smoked)						Ex-Smokers (reference gp never smoked)					
		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)	
1981-1984													
Maori	all age	0.60	(0.32-1.14)	0.54	(0.22-1.34)	--	--	0.83	(0.41-1.66)	0.54	(0.18-1.61)	--	--
Pacific	all age	0.85	(0.26-2.72)	--	--	--	--	0.18	(0.02-1.51)	--	--	--	--
NonM-NonP	all age	1.64	(1.40-1.93)	1.71	(1.35-2.16)	1.52	(1.20-1.93)	1.07	(0.90-1.26)	1.17	(0.93-1.48)	1.14	(0.90-1.44)
All Ethnicity Combined **not adj for eth**	all age	1.54	(1.32-1.79)	1.62	(1.29-2.03)	1.44	(1.15-1.81)	1.02	(0.87-1.19)	1.12	(0.90-1.41)	1.10	(0.88-1.38)
All Ethnicity Combined **adj for eth**	all age	1.50	(1.29-1.75)	1.61	(1.29-2.02)	1.44	(1.15-1.81)	1.01	(0.85-1.19)	1.13	(0.90-1.41)	1.11	(0.88-1.39)
	25-44	2.15	(0.98-4.71)	1.70	(0.66-4.38)	--	--	1.04	(0.36-2.99)	1.26	(0.40-3.99)	--	--
	45-64	1.83	(1.37-2.44)	2.02	(1.34-3.07)	1.81	(1.19-2.75)	1.08	(0.76-1.53)	1.26	(0.81-1.97)	1.26	(0.81-1.97)
	65-74	1.35	(1.11-1.63)	1.54	(1.18-2.01)	1.39	(1.06-1.82)	0.98	(0.81-1.18)	1.10	(0.85-1.41)	1.07	(0.83-1.38)
1996-1999													
Maori	all age	1.02	(0.59-1.78)	0.92	(0.46-1.85)	--	--	0.43	(0.23-0.83)	0.37	(0.15-0.92)	--	--
Pacific	all age	0.47	(0.17-1.26)	1.11	(0.29-4.25)	--	--	0.88	(0.31-2.47)	1.66	(0.41-6.70)	--	--
NonM-NonP	all age	2.23	(1.81-2.76)	2.45	(1.84-3.27)	1.93	(1.43-2.59)	1.07	(0.88-1.30)	1.21	(0.93-1.58)	1.17	(0.90-1.53)
All Ethnicity Combined **not adj for eth**	all age	1.95	(1.61-2.36)	2.16	(1.67-2.80)	1.66	(1.27-2.17)	0.93	(0.78-1.12)	1.05	(0.82-1.34)	1.02	(0.80-1.31)
All Ethnicity Combined **adj for eth**	all age	1.93	(1.59-2.34)	2.06	(1.59-2.67)	1.66	(1.27-2.17)	0.96	(0.80-1.15)	1.07	(0.84-1.37)	1.04	(0.82-1.33)
	25-44	1.29	(0.55-3.00)	1.62	(0.58-4.52)	1.10	(0.38-3.14)	1.03	(0.40-2.69)	1.11	(0.32-3.89)	1.06	(0.30-3.72)
	45-64	1.65	(1.20-2.25)	2.08	(1.37-3.16)	1.54	(1.00-2.37)	0.66	(0.46-0.95)	0.78	(0.49-1.26)	0.76	(0.47-1.23)
	65-74	2.15	(1.68-2.76)	2.02	(1.44-2.84)	1.74	(1.23-2.46)	1.12	(0.90-1.39)	1.20	(0.90-1.60)	1.18	(0.88-1.58)

Male Stroke Adj RR NZCMS n

* age-standardised [First Restricted Cohort]

† adjusted for age (5 year bands) [Second Restricted Cohort]

‡ adjusted for age and socio-economic position (SEP) = education, car access, household equivalised income, marital status, NZDep, labour force, housing tenure [Second Restricted Cohort]

Table 26: Female Stroke Rate Ratios – standardised, and adjusted for confounding (Second Restriction)

	Age Gp	Current Smokers (reference gp never smoked)						Ex-Smokers (reference gp never smoked)					
		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)		SRR * (95% CI)		Adj RR - Age † (95% CI)		Adj RR - Age + SEP ‡ (95% CI)	
1981-1984													
Maori	all age	1.05	(0.60-1.84)	1.33	(0.66-2.68)	--	--	1.44	(0.78-2.64)	0.73	(0.26-2.05)	--	--
Pacific	all age	0.23	(0.03-1.98)	0.92	(0.13-6.65)	--	--	3.62	(0.80-16.49)	1.57	(0.22-11.07)	--	--
NonM-NonP	all age	1.80	(1.55-2.10)	1.84	(1.49-2.27)	1.78	(1.44-2.20)	1.34	(1.12-1.61)	1.05	(0.80-1.37)	1.06	(0.81-1.39)
All Ethnicity Combined **not adj for eth**	all age	1.77	(1.53-2.05)	1.88	(1.54-2.29)	1.80	(1.47-2.20)	1.41	(1.18-1.67)	1.05	(0.81-1.35)	1.06	(0.82-1.37)
All Ethnicity Combined **adj for eth**	all age	1.65	(1.42-1.92)	1.78	(1.46-2.18)	1.74	(1.42-2.13)	1.39	(1.15-1.67)	1.03	(0.79-1.33)	1.04	(0.80-1.34)
	25-44	2.48	(1.27-4.82)	3.45	(1.53-7.79)	3.12	(1.36-7.15)	1.42	(0.58-3.50)	0.63	(0.13-3.01)	0.62	(0.13-2.94)
	45-64	2.27	(1.72-3.00)	2.63	(1.87-3.69)	2.59	(1.84-3.65)	1.12	(0.73-1.72)	0.92	(0.53-1.61)	0.93	(0.53-1.62)
	65-74	1.35	(1.11-1.65)	1.22	(0.93-1.60)	1.19	(0.91-1.56)	1.48	(1.21-1.83)	1.14	(0.86-1.51)	1.15	(0.87-1.52)
1996-1999													
Maori	all age	1.62	(0.87-3.01)	1.06	(0.49-2.29)	--	--	1.41	(0.79-2.49)	1.32	(0.59-2.95)	--	--
Pacific	all age	0.64	(0.23-1.78)	--	--	--	--	0.86	(0.25-2.94)	--	--	--	--
NonM-NonP	all age	3.01	(2.44-3.72)	3.07	(2.30-4.10)	2.66	(1.98-3.57)	1.47	(1.19-1.83)	1.50	(1.12-2.01)	1.50	(1.12-2.01)
All Ethnicity Combined **not adj for eth**	all age	2.64	(2.17-3.20)	2.80	(2.14-3.65)	2.26	(1.72-2.98)	1.42	(1.16-1.72)	1.53	(1.16-2.00)	1.51	(1.15-1.99)
All Ethnicity Combined **adj for eth**	all age	2.51	(2.06-3.05)	2.59	(1.98-3.40)	2.20	(1.66-2.90)	1.40	(1.15-1.72)	1.52	(1.16-1.99)	1.51	(1.15-1.98)
	25-44	5.28	(2.33-11.94)	7.65	(2.60-22.50)	5.20	(1.69-15.96)	0.52	(0.11-2.53)	1.17	(0.20-6.85)	1.09	(0.19-6.42)
	45-64	3.54	(2.54-4.91)	4.01	(2.63-6.13)	3.31	(2.15-5.12)	1.56	(1.04-2.33)	1.42	(0.84-2.39)	1.43	(0.84-2.42)
	65-74	1.93	(1.47-2.54)	1.72	(1.17-2.51)	1.55	(1.05-2.28)	1.40	(1.11-1.77)	1.64	(1.21-2.22)	1.64	(1.21-2.22)

Female Stroke Adj RR NZCMS n

* age-standardised [First Restricted Cohort]

† adjusted for age (5 year bands) [Second Restricted Cohort]

‡ adjusted for age and socio-economic position (SEP) = education, car access, household equivalised income, marital status, NZDep, labour force, housing tenure [Second Restricted Cohort]

Chapter 7: Results – part 3 (sensitivity analysis)

As described in Chapter 3 (page 63) a sensitivity analysis was conducted for Māori males aged 65-74 years in 1996-99, with regards to the accuracy of measuring current smoking status.

Sensitivity levels of 95%, 90%, and 80% (of complete "current smoking" measurement) were applied, which correspond to possible under-measurement or under-reporting by 5%, 10% and 20% respectively. The 20% level of misclassification is an extreme figure – ie. more than would be expected (based on overseas literature there may be around 10% for males in minority ethnicities, see Discussion section 3.3.3).

Table 27 shows that with lower levels of sensitivity, the rate ratios for male Māori current smokers aged 65-74 years in 1996-99 do not change to a great extent, and are still notably lower than those observed for non-Māori non-Pacific for all-cause, IHD and stroke mortality. Therefore, it seems unlikely that misclassification of smoking status that is differential by ethnicity could spuriously give rise to the heterogeneity of relative risk reported above. Note, the stroke rate ratios slightly move up and down depending on the level of sensitivity – this is possible with a trichotomous exposure (Dosemeci, Wacholder et al. 1990; Rothman and Greenland 1998).

Table 27: Sensitivity analysis for male current smokers aged 65-74 years, 1996-99

	Crude Rate Ratios for Current Smokers (reference gp never smoked)				
	Maori				NonM-NonP
	Observed	95% Sensitivity	90% Sensitivity	80% Sensitivity	Observed
1996-1999					
All-Cause	1.54	1.56	1.58	1.62	2.24
IHD	1.12	1.12	1.12	1.13	1.89
Stroke	1.44	1.42	1.43	1.46	2.26

Sensitivity Analysis 65-74yr males NZCMS

Chapter 8: Discussion

Discussion Summary

This study provides effect measure estimates for the smoking-mortality association, including relative risks, specifically for the New Zealand population. Relative risks for 1996-99 appear to vary from those provided by CPS II.

On the whole these estimates are reasonably precise, but may be more prone to some systematic biases, including exposure misclassification and some residual confounding by "lifestyle" factors, as well as selection bias of the multivariable results. Nevertheless, the different sources of error are unlikely to substantially alter the association between smoking and mortality. Most notably, any sources of error are extremely unlikely to explain the important patterns seen by age, sex, and especially ethnicity and time.

These patterns of heterogeneity by strata of ethnicity and time illustrate that the effect of smoking on mortality cannot be fully interpreted by non-stratified and overall effect measure estimates.

Rate ratios increase with age for all-cause mortality, but decrease with age for IHD (and female stroke) mortality. By sex, rate ratios were similar for males and females for all-cause mortality, but for IHD and stroke mortality females have higher rate ratios than males. Over time excess rate ratios have approximately doubled from 1981-84 to 1996-99.

Statistically significant heterogeneity of the rate ratios exists by ethnicity, with Māori and Pacific estimates tending to be lower than non-Māori non-Pacific. Possible explanations for the rate ratio heterogeneity observed include variation in the underlying mortality rates combined with more homogeneous rate differences, and perhaps passive smoking.

These findings show the need for population and ethnicity specific information, and can be used to more accurately inform tobacco control research and policy in New Zealand.

This chapter discusses the important findings of this study, based on the results presented in the previous four chapters. It is structured in the following way:

1 A description of the different measures and comparisons this study can provide
2 A description of the "overall" findings of the study, broken down only by sex and cause of death (ie. a summary of the all-age all-ethnicity results). The difficulty of direct comparisons with overseas studies is mentioned, however some contrasts can be seen against the CPS II data.
3 An examination of the potential sources of error that may have contributed to the observed effect measure estimates, in particular the rate ratios, and the heterogeneity seen. These include chance (random error), and factors that may affect the internal validity of the study such as selection bias, misclassification bias, lag time bias, and confounding. The external validity of the study findings (generalisability) is also considered. Given that the estimates are likely to be reasonably accurate, and in particular that the heterogeneity of rate ratios by demographic strata appears real, some of the more specific patterns within the data are examined in the next four sections. These discuss the influence of:
4 Age;
5 Sex;
6 Ethnicity; and
7 Time

   Each of these four sections also endeavours to make some comparison with overseas findings.
8 Lastly, the implications that the findings of this study will have on health policy and research are discussed, not only for tobacco control but wider afield.

It should be noted that where I have discussed ethnicity, Māori versus non-Māori non-Pacific comparisons predominate due to the greater precision of the Māori estimates compared to the Pacific estimates.

The discussion is mostly limited to the results for current smokers (with ex-smokers on the whole showing lower risk). Where there is a particularly unusual pattern for ex-smokers, this is mentioned.

1 Study effect measures and comparisons

In this thesis, I take the causal relationship between smoking and increased mortality as proven. What this thesis adds is a demonstration of the size (or strength) of this association in the New Zealand population, and it appears to be the first to do so.

Effect measures for smoking have been measured in both relative and absolute terms (rate ratios and rate differences respectively) and both are valuable. This thesis focuses predominantly on analysis of rate ratios, which allow comparison between groups and with other studies, regardless of the underlying mortality rates (eg. higher for men, lower for women). However, rate ratios can also give rise to some inaccurate conclusions and it is not always appropriate to use the rate ratio in isolation. For example, if rates decline over time by the same absolute amount in each group (current and never smoker), the natural mathematical consequence will be that the ratio of the rates increases. A ratio of 20 over 5 would equal 4, and a ratio of 220 over 205 would equal 1.07, even though both are separated by an absolute difference of 15. In these types of circumstances, a measure of the actual gap (deaths per person-years) can be helpful. It also gives an impression of the numbers of people affected by smoking.

The all-age all-ethnicity effect measure estimates (summarised in the next section) give an overall impression of relative and absolute excess risk from smoking in New Zealand. However perhaps one of the most important points to take from the results is that the strength of the association is not fully understood with one overall population estimate. Effect measure estimates show modification by age, sex, ethnicity, and time, and therefore must be assessed with respect to each. In fact it is not so much the individual estimates from this thesis that are most important, but the patterns shown within the results.

It is also important to note that the rate ratios and rate differences in this thesis measure the strength of the smoking-mortality association only within age / sex / ethnicity strata. They do not measure differences in mortality between demographic groups (for example Māori mortality rates compared to non-Māori non-Pacific mortality rates). For example, a lower rate ratio for Māori (current smokers compared to never smokers) does not mean the mortality rate among Māori is less, just that the smoking-mortality association within

Māori is weaker. Looking at the actual mortality rates gives an impression of overall mortality risk (eg. see Figure 5 and Figure 6, pages 80 and 81). Unless otherwise specified, the terms 'effect measure' or 'association' will generally refer to that of the smoking-mortality association within different strata of interest (eg. 'all-age all-ethnicity', 'Māori', 'males').

As this study appears to be the first to analyse in detail mortality rates by smoking status and by demographic strata within New Zealand, it also allows for the first time examination not only between each smoking status (smoking effect measures) but *within* each smoking status. In other words we are able to look at patterns of mortality rates by age, ethnicity, and time separately for smokers and never smokers, and in particular we can look at those rates that are completely unaffected by the influence of smoking – ie. within the never smoked group. We can speculate as to the determinants of these patterns or trends that are not smoking related. This is particularly significant when considering the Māori and Pacific rates, as discussed later.

It is also important to note that this study examines rates of mortality, which is something different to disease incidence. Mortality statistics take into account not only the occurrence of disease, but all those factors that influence post-onset survival as well. For example, health services, personal resources (including insurance), social support, ability to return to work. These factors explain, at least in part, why the results of the association between smoking and IHD / stroke mortality will likely differ from published accounts of the association between smoking and IHD / stroke incidence.

2 Overall findings

As an example of the overall findings from this study, the all-age all-ethnicity (age and ethnicity standardised) rate ratios for current smokers compared to never-smokers, as well as the fully adjusted multivariable estimates, are shown in Table 28. Other aspects of this table (the middle two columns) are discussed in later sections.

Table 28: RR % change from multivariable analysis applied to standardised rate ratios (25-74 years, all ethnicity, ethnicity standardised)

		Current Smokers (reference gp never smoked)			
	Sex	SRR *	% decrease in excess RR from multivariable analysis †	New "adjusted" SRR (applying % change)	Adjusted RR from multivariable analysis (Age + SEP)
1981-1984					
All-Cause	Male	1.59	23 %	1.46	1.44
	Female	1.49	9 %	1.45	1.50
IHD	Male	1.50	21 %	1.40	1.38
	Female	1.86	9 %	1.78	1.78
Stroke	Male	1.50	28 %	1.36	1.44
	Female	1.65	5 %	1.62	1.74
1996-1999					
All-Cause	Male	2.05	33 %	1.71	1.68
	Female	2.01	21 %	1.80	1.83
IHD	Male	2.03	36 %	1.66	1.61
	Female	2.67	21 %	2.32	2.52
Stroke	Male	1.93	38 %	1.58	1.66
	Female	2.51	25 %	2.14	2.20

SRR and Adj RR NZCMS n

* age-standardised [First Restricted Cohort]
† percentage change from excess RR (RR-1) adjusted for age only to excess RR adjusted for age + SEP

By sex, rate ratios were similar for males and females for all-cause mortality, but for IHD and stroke mortality females have higher rate ratios than males. Over time rate ratios have approximately doubled from 1981-84 to 1996-99. These patterns are also discussed further in later sections (section 5, page 149, and section 7, page 161, respectively).

Standardised rate differences overall showed somewhat different patterns to the rate ratios. For example, while they do vary, on balance the all-age all-ethnicity rate differences tend to show more homogeneity over time (less than double).

For all-cause mortality, the all-age all-ethnicity standardised rate differences for males were 444 deaths per 100,000 person-years (405-482) in 1981-84 and 539 (504-574) in 1996-99. For females the rate differences were 233 (203-263) in 1981-84, and 335 (306-364) in 1996-99.

For IHD mortality, the standardised rate differences for males were 134 deaths per 100,000 person-years (112-156) in 1981-84 and 139 (121-156) in 1996-99. For females, the equivalent rate differences were 94 (79-110) and 73 (61-85).

For stroke mortality, the standardised rate differences for males were 30 (19-41) in 1981-84 and 26 (17-34) in 1996-99. For females they were 31 (21-41) and 30 (22-38).

These data also show that for all-cause mortality and IHD, males have larger rate differences between smokers and never smokers than females (in contrast to the rate ratios), reflecting the higher underlying mortality rates. Rate differences for stroke are similar for each sex.

2.1 Comparison with international relative risk estimates

A direct comparison with overall relative risk estimates reported from overseas studies is difficult due to differences in methodology, age range, ethnicity, and measurement of level of exposure (as illustrated in Chapter 2). Summary statistics and measures of association that are given in reports and journal articles from these studies can obscure patterns within the whole dataset and therefore can be misleading. In particular the strong effect modification by age will mean that studies of different age groups may give different rate ratios. This problem is not necessarily solved by, say, direct standardisation *unless* the same standard population is used. For example, for ischaemic heart disease, if the study population is older and/or the rates are weighted to an older population, the resulting summary rate ratio may be lower (as IHD rate ratios decline with age). Presentations of

results by strata can overcome this problem to some degree (eg. age groups – as long as they match – and level of smoking exposure). However, stratified results are not always available, and do not address other issues of non-comparability. For complete comparability with international estimates we would need original data.

Nevertheless, a rough assessment from the figures shown in Table 1, Table 2 and Table 3 (pages 19 to 21), shows that the overall NZCMS results are similar to many of the published data. There is also a similarity between the 1981 NZCMS results and the earlier overseas studies (1950s to 70s), and between the 1996 NZCMS results and the later overseas studies (1970s to 80s), which may be in keeping with the tobacco epidemic arriving in New Zealand slightly later then overseas (lag effect).

A more specific comparison can be made with one of the most widely utilised studies, CPS II, which has been used in attributable burden calculations in NZ (Laugesen and Clements 1998; Laugesen and Swinburn 2000; Tobias and Cheung 2001). CPS II is the largest prospective cohort study that has examined the association of smoking with mortality. For CPS II, data was available for age-specific mortality rates for smokers and never smokers, as well as the size of the standard population used by five-year age bands (Thun, Day-Lally et al. 1997a). It was therefore possible to re-calculate rate ratios for age bands that match the NZCMS 45-64 years and 65-74 years (25-44 was not possible as there was no CPS II age-specific data below age 35), both standardised to CPS and to the 1996 NZ population (the latter used in this study).

Table 29 shows CPS II data standardised to both the CPS population (CPS I and II combined) and the 1996 NZ population, plus the NZCMS findings at the bottom for comparison of all-cause and IHD mortality risk estimates. Even with the CPS II results standardised to the same population (NZ 1996), they are different to the NZCMS results. In addition, the CPS II results are extremely different from those seen for Māori, and cannot be applied to this population. Note, Table 29 does not include 95% confidence intervals as it is merely comparing rate ratio estimates (which would be used for attributable burden calculations) rather than assessing the precision of the results.

Table 29: CPS II mortality rate ratios compared to 1996-99 NZCMS

Study	Age Group	All-Cause Mortality		IHD	
		Male	Female	Male	Female
CPS II age standardised to CPS population	45-64	2.86	2.16	2.93	3.30
	65-74	2.58	2.19	1.80	2.14
CPS II age standardised to 1996 NZ population	45-64	2.85	2.15	3.00	3.37
	65-74	2.57	2.17	1.78	2.09
1996 NZCMS age and ethnicity standardised	45-64	2.06	1.89	2.37	2.87
	65-74	2.18	2.32	1.81	2.57
1996 NZCMS Maori	45-64	1.53	1.50	1.67	2.10
	65-74	1.55	1.53	1.10	1.42

CPS II - NZCMS comparison

Possible reasons for the variation in relative risk (between CPS II and NZCMS) include some of the factors that were discussed in Chapter 2. These include real or possible differences in the amount, duration and behaviour of cigarette consumption, cigarette constituents, and differences in confounding factors and risk factors between the study populations. The effect modification seen by ethnicity in New Zealand may also be a driver, however the non-Māori non-Pacific group still appears different to CPS II (which is predominantly white – 93% (Thun, Day-Lally et al. 1997a)). The differences in rate ratio estimates between CPS II and NZCMS illustrate the point that for further research and policy, population-specific relative risks should be used wherever possible – this means using NZ-specific estimates for attributable disease calculations.

It should be noted that rate difference homogeneity among some strata, combined with varying mortality rates, is consistent with some of the overall rate ratio variation between CPS II and NZCMS. For example, in the 1996-99 NZCMS cohort, among males aged 45-64 the rate difference between current smokers and never smokers is 559 deaths per 100,000 person-years. The corresponding rate difference (standardised to the 1996 NZ population) for CPS II is very similar at 578.5. However the underlying mortality rates for this stratum are lower for CPS II (eg. 898 vs 1087 for current smokers), therefore the NZCMS rate ratios will consequently be lower than the CPS rate ratios. The same pattern

is seen for IHD in the same stratum – rate differences of 188 in the NZCMS versus 185 in CPS II, and lower mortality in CPS II.

As discussed in later sections, comparisons can be made more widely (with other studies) of the patterns seen within the NZCMS results.

3 Potential sources of error

This discussion chapter predominantly assumes that the results in thesis are accurate. Before this is taken as conclusive, and the more detailed patterns in the results are discussed, this assumption must be investigated. The following sections examine the sources of error (both potential and actual) within the study methodology and analysis that may have influenced the observed results, and whether or not they can truly be applied to the New Zealand population.

The sources of error discussed can be classified as random error (chance) and systematic error (bias and confounding).

3.1 Chance

3.1.1 Smoking-mortality association

Precision of the study results implies a lack of random error. It is important to know that we are not observing these data merely due to chance or random variation. Overall, random error is not a large problem for this study. The two "participating" cohorts represent almost the entire New Zealand population on census night 1981 and 1996, with the first restriction (used in Part 1 analyses) being 98.3% of the original cohort in 1981 and 92.5% in 1996. As discussed in chapter 3, the mortality records initially recorded in the linked dataset represent approximately 75% of the actual deaths, and the data are then weighted to approximate 100% of the deaths in New Zealand in the three years post census. Consequently, the high number of outcome events (numerator) and the large amount of person-time (denominator) captured by this study (ie. large study size) gives it a high degree of statistical power and precision overall.

This precision of the results is shown by width of the 95% confidence intervals given for each point estimate (wider intervals indicate less precision). Assuming no systematic bias, in the *hypothetical* situation of repeating this study many times (not strictly possible as the NZCMS does not "sample" part of the study population), these intervals will contain the true population estimate no less than 95% of the time. This gives us a guide as to the

certainty or uncertainty of the point estimates with respect to random variation (or "chance"). The confidence intervals also indicate whether or not the estimate has reached statistical significance. If the confidence crosses or includes 1.0 for the rate ratios, or zero for the rate differences, the estimate is not statistically significant at the 95% level. (However, it is critical to note that it is more important to inspect the central estimate of the ratio or difference first, and use the confidence intervals as indicators of precision of the estimate. Simply treating confidence intervals as test of the null hypothesis loses much information.) In this study we are looking for rate ratios over 1.0 (with reference group never smokers) as this indicates a positive association with the exposure (smoking). If the lower limit of the confidence interval is greater than 1.0, then we can say that there is at least a statistically significant positive association with smoking (with 95% confidence).

The results are most precise for all-cause mortality (narrower confidence intervals), as well as the all-age and all-ethnicity strata, due to larger numbers of deaths. They are less precise for IHD (particularly for females), and least precise for the stroke results. With regards to ethnic groups, the results are most precise for non-Māori non-Pacific and least precise for Pacific, with Māori intermediate. As a result, statistical significance of the rate ratio and rate difference estimates also vary by cause of death and ethnicity, as well as by sex and age. For the multivariable results, the precision and statistical significance of the results follow a similar pattern to the standardised results.

3.1.2 Effect measure heterogeneity by ethnicity

It is also possible that the heterogeneity in risk estimates seen between the different ethnic groups is also due to random error or chance. However, as mentioned in the results chapters, after performing a Wald test on the current smoker all-age data we can be at least 95% confident that the rate ratio heterogeneity is a not a chance finding. In other words, the probability that this study has observed this rate ratio heterogeneity by chance alone is very small.

3.2 Selection Bias

The essence of selection bias is that the relationship between exposure (smoking) and disease is different for those who participate in a study compared with those who do not.

Some selection bias may have been introduced into this study by the exclusion of participants in creating both the first and second restricted cohorts. This is probably more likely to have occurred for the multivariable analyses (second restriction) than the age-standardised calculations (first restriction), as the latter contained quite a high proportion of the original cohort (98.3% in 1981 and 92.5% in 1996). The second restriction was 73.1% of the original cohort in 1981 and 74.0% in 1996. There is no specific reason to believe that those excluded are particularly different than those included, however the significant difference in the number of participants between first and second restrictions raises the issue of selection bias as a possibility. Table 42 (page203) in Appendix C also shows the person-time data for both the first and second restriction for comparison.

Looking at the results presented in Chapter 6, there is likely to be some effect from selection bias on the multivariable results, with some differences seen between the age-standardised and age-adjusted rate ratios – although different methods of analysis (ie. direct standardisation vs poisson regression) will make a modest contribution to differences as well. For example there are some notable differences in smoking effect measures for the second restricted cohort among Pacific female current smokers. However, for the overall estimates (which are more precise) there does not seem to be much variation. For example, for 1996 all-cause mortality within the all-age all-ethnicity grouping (adjusted for ethnicity), the male age-standardised rate ratio is 2.05, compared to the age-adjusted rate ratio of 2.01 (Table 21). For females, the difference is 2.01 compared to 2.05 (Table 22).

Nevertheless, so long as we use the multivariable results from the second restricted cohort to just give an indication of the degree of confounding by SEP (ie. how much the risk estimates change), rather than the actual value of the fully adjusted estimates, we can "side-step" possible selection bias. This is discussed further under section 3.5.2.1 "Degree of confounding by SEP" (page141).

One particularly positive point for this study is that there are no issues with self-selection of participants or "volunteer bias", as can be the case with many prospective cohort

studies, including CPS II (family and friends of American Cancer Society volunteers) (Thun, Day-Lally et al. 1997a).

3.3 Misclassification bias

On the whole the information on which the analyses are performed in this study is likely to be reasonably accurate. There is no issue of recall bias, as there may be in case-control study, the exposure and co-variate information is based on self-reporting using a standardised and well-accepted questionnaire with clear criteria (NZ census), and the outcome data from NZHIS is taken from death certificates based on expert clinical assessment and/or objective testing with or without autopsy. The data handling at Stats NZ is also high quality. Despite this overall high standard there may still have been some misclassification that has affected the results. It is hard to know if any misclassification of confounders would occur, for example if smokers were more likely to give false information about SEP variables, and in which direction this would influence the results (eg. would they over or under report income?). The most important issue in this study is likely to be misclassification of exposure.

3.3.1 Linkage Bias

One type of outcome misclassification that this study could be prone to is linkage bias (as described in chapter 3), as only approximately 75% of deaths were linked back to a census record. This is differential misclassification bias of the mortality outcome. However, as described in chapter 3 (methods), this has been overcome by applying a weighting factor to all records in the linked dataset, and all analyses presented here are on weighted data only. Any residual linkage bias is likely to be small (Blakely, Salmond et al. 2000; Fawcett, Blakely et al. 2002).

It should be noted that it is unlikely that within specific strata (eg. age, ethnicity, geography, deprivation) there will be a significant difference in the smoking-mortality association between those who are linked and unlinked (this probably represents more of a selection bias). Nevertheless, this cannot be proven.

3.3.2 Outcome Misclassification

With regards to the accuracy of cause of death (as written on death certificates), this may be less than ideal, especially without objective investigations or autopsy. One estimate from death certification data in the early 1980s put this error in the vicinity of 10% for coronary heart disease (Jackson, Graham et al. 1988). Any significant inaccuracy in this regard would introduce a degree of misclassification, however as this may just shift deaths from one category to another in all directions this wouldn't necessarily impact on estimates of risk from smoking. Even if some causes of death were "guessed" more commonly than others (ie. differential misclassification), if this occurred for both smokers and never smokers alike this would not change risk sizes within each cause.

Of more concern is any differential misclassification of some causes of death in smokers as compared to never smokers. For example, if clinicians or pathologists are aware that the deceased was a smoker, they may consciously or subconsciously have a greater tendency to diagnose the cause of death as something that is known to be caused by smoking, such as cardiovascular pathology. This differential misclassification bias would tend to falsely increase the deaths of smokers for these causes, and this study would overestimate risk. However, any such misclassification is unlikely to be major given the broad groupings of disease (IHD and stroke) used in this study.

There is obviously no question as to mistakenly classifying death itself, or of all-cause mortality, therefore the all-cause mortality results in this study are unaffected by outcome misclassification.

3.3.3 Exposure Misclassification

Smoking status may also be misclassified to some extent, in three ways. Firstly, the NZCMS follows up participants for three years post census (or death before three years), however it is only their census-night smoking category that is recorded. It is likely that smoking status for some of the participants will have changed over the follow-up period. For example, some of the current smokers on census night will have quit smoking, and some of the ex-smokers will have restarted. Using the fact that current smokers are at greater risk of premature mortality than ex-smokers, the presence of people in the current

smoker group that have actually quit smoking without our knowledge will decrease the number of observed deaths, and mean that we have to some degree underestimated the real mortality risk from current smoking. Likewise, the presence of people that have restarted smoking among the ex-smoker group will increase the number of deaths, and mean than we have to some degree overestimated the real mortality risk in ex-smokers.

Secondly, smoking status recorded on census night may be wrong. A report by Wells et al in 1998 estimates the total misclassification rates for current smokers self-reporting as never-smokers in the US population to be 2.6% for females and 3.4% for males. The rates for current smokers self-reporting as ex-smokers were 2.3% for females and 3.6% for males. If this were also the case in the New Zealand population it would mean that we have slightly underestimated relative risks for current smokers (ie. the results are slightly biased towards the null), and slightly more so for males. However, given the small size of these misclassification rates it would not make a large difference to the results (see sensitivity analysis, chapter 7, as an illustration among Māori males). It is unlikely that there is misclassification in the other direction – ie. never-smokers reporting to be current smokers.

Thirdly, the misclassification of smoking status on census night may vary by ethnicity, and therefore may be a factor in producing the observed heterogeneity in relative risk by ethnicity. In the same report by Wells et al, "an appreciable difference was seen between the misclassification rates for US Blacks and Latinos and the rates for Whites in the United States and majority groups in various other countries". The rate of US minority female smokers (regular and occasional combined) misclassified as never smokers is 4.9%, which is three times the female majority rate (1.6%). For minority males the rate is 5.7%, which is 2.9 times the majority rate (2.0%). The rates for current smokers mis-reporting as ex-smokers were 2.9% for minority females (majority 2.1%) and 4.5% for minority males (majority 3.0%). The total underreporting of current smoking (as never or ex) was therefore 7.8% among minority females and 10.2% among minority males. Such ethnic differences may also apply in New Zealand, and may mean that the smoking-mortality association has been underestimated among Māori and Pacific (if there are higher rates of misclassification) and that the degree of heterogeneity (based on lower effect measures in these groups) has been overestimated.

As the effect measure modification by ethnicity is a particularly important finding of this study, it was endeavoured to quantify the effect that any differential exposure misclassification between the different ethnic groups would have on the results. Using a limited sensitivity analysis (methods in chapter 3, page 63, results in chapter 7), it was demonstrated that even with a high rate – 20% – of under-reporting of current smoking (ie. 80% sensitivity) among older Māori males in 1996-99, the rate ratios changed very little, and were still notably different to those for non-Māori non-Pacific (see Table 27, page 121).

The fact that this study did not measure more precise degrees of exposure can also be thought of as a type of misclassification, although the results as they are presented (for broad categories of smoking) are not actually erroneous as a result. For example, the level of smoking (only available for 1981) was not obtained, and current smokers were grouped together regardless of the amount they smoked (eg. two to forty cigarettes per day in the same category). This again raises the problem with summary statistics. As the health effects of smoking exposure exhibit a dose-response relationship (Doll, Peto et al. 1994), the overall risk estimates presented by this study are likely to underestimate the risk from heavy smoking, and overestimate the risk from light smoking (the degree depends on the prevalence of each). The same issue applies for duration and time of smoking, which is also not measured here. Both the current and ex-smokers will be a heterogenous group, some of whom have smoked for a long time, some only a short duration, and others (among the ex-smokers) who have quit many years before - and who therefore have risk approaching never smokers. The results in this thesis have probably underestimated the risk for long-time current smokers, and overestimated them for those ex-smokers who gave up many years previously.

3.3.4 Misclassification of co-variates

Another form of misclassification that may exist to some degree in this study is that of the variables used to control for confounding, in particular the markers of SEP. For example, people may give a false or at least an inaccurate measure for some co-variates, such as income. In addition, the questions in the census may mismeasure some variables with

regards to reflecting actual SEP. For example, both the 1981 and 1996 census questions on income only pertain to income within the past 12 months, as opposed to "usual" or "previous" income, which may better reflect SEP in some cases. Nevertheless, the extent of misclassification for each co-variate is probably not great. And although measurement errors can potentially add up over a number of variables, they could also occur in different directions for each one (some leading to under-controlling of confounding, some over-controlling), with a possible net zero balance of inaccurately capturing SEP.

3.4 Lag time bias

One final type of measurement error or bias that may have influence the results arises from the fact that some causes of death from smoking have a long lag time from exposure to outcome. In other words the effect takes a long time to become apparent. As the NZCMS only follows up participants for three years post census, there will be mortality outcomes further into the future that have been caused by "current smoking" as it was measured on census night. Many cancers would probably fall into this category. It is therefore likely that all-cause mortality rates (which includes these causes of death) among smokers will have been somewhat underestimated due to this "lag time bias", and as a result all-cause relative risk has been underestimated as well.

3.5 Confounding

3.5.1 Age, Sex, and Ethnicity

As discussed in chapter 3 (methods), confounding by age, sex and ethnicity has been either eliminated or greatly reduced by restriction, stratification, direct standardisation and multivariable analysis. Adjustment for age (standardisation and multivariable) was performed using five year age bands, so it is possible that additional age-related trends within those bands has resulted in a small amount of residual confounding by age, however it is unlikely that any adjustment using one year groupings would change the results in a noticeable way.

3.5.2 Socio-economic position

As shown in chapter 6, smoking rate ratio estimates have also been adjusted for confounding by socio-economic position (SEP) using multivariable analysis. The co-variates used probably captured much of individual SEP, and the inclusion of NZDep also meant that some degree of contextual effect (ie. neighbourhood, small area SEP) was also captured. The significance of SEP as a confounder is discussed below, however it should be first noted that after controlling for SEP there is still an appreciable association between smoking and mortality, and the important patterns remain – ie. heterogeneity by sex, ethnicity and time.

3.5.2.1 Degree of confounding by SEP

The degree to which the "unadjusted" smoking-mortality association is confounded by SEP is described in chapter 6, and summarised in Table 28 (page 127) – column *'% decrease in excess RR from multivariable analysis'*. The all-age all-ethnicity rate ratio estimates shift by a moderate, but not large, amount after adjusting for SEP. In 1981-84 the estimates decreased by 5-9% for females and 21-28% for males, and in 1996-99 by 21-25% for females and 33-38% for males (percentages calculated from the excess rate ratios, RR-1). This confirms the supposition that relative risk estimates that are unadjusted for SEP will be at least partly elevated from this confounding effect.

Looking again at Table 28 (page 127), the amount of risk that has been determined to be due to confounding by SEP (as a percentage) can be applied back to the results from the first restricted cohort, as illustrated in the third column from the left – *'New "adjusted" SRR (applying % change)'*. The figures in this column thereby gives age-standardised results for the first restricted cohort, which are presumably free from selection bias, and that are also adjusted for confounding by SEP (for comparison the fully adjusted multivariable results are shown on the far right).

It is interesting to note that the impact of confounding by SEP seems to be similar across the different outcomes, across all age groups, and across all ethnicities. The latter means that there is still heterogeneity of relative risks by ethnicity. This suggests that it is not

some complex interaction of ethnicity and SEP that gives rise to the heterogeneity of smoking mortality risk by ethnicity.

Another finding is that the relative risks for ex-smokers compared to never-smokers do not seem to change very much at all, suggesting that there is negligible confounding by SEP for this group. This is supported by the research of Hill et al (2003), which suggests that there is little association between SEP and "ex-smoking" (compared to current smoking).

These results also show that there appears to be more confounding by SEP for males, and for the 1996-99 period (therefore there is less increase in effect over time once adjusted for SEP). These patterns are discussed below.

3.5.2.2 SEP confounding between the sexes

The higher observed confounding for males could be due to stronger relationships between smoking and SEP, and/or between SEP and mortality, in males compared to females. For the former, the 2003 report by Hill et al suggests that there was a stronger relationship between SEP and income among males (compared to females) in 1981, however the gradient in 1996 may be similar for both males and females. With regards to a stronger association between SEP and mortality, it is possible that males in lower SEP groups have particularly poor health service access compared to their female counterparts (for example anti-hypertensive or lipid-lowering treatment).

Another explanation for the overall male-female confounding disparity could be that SEP has not been captured as accurately for females. In other words, the variables used do not capture SEP as well for females, for example education and occupation may be less meaningful if they are homemakers – particularly for older women (although it is hard to see if there is any age difference for confounding). Marmot and McDowall (1986) noted that social class as measured by occupation is not a good indicator of differentials in mortality risk for women.

An interesting point to note is that the percentage confounding by SEP for females in 1996-99 is similar to that for males in 1981-84, which could reflect evolution of the tobacco epidemic.

3.5.2.3 SEP confounding over time

The other notable pattern seen is that the relative risk estimates for 1996-99 reduce by a greater degree after controlling for SEP than the 1981-84 results. The 1981 and 1996 relative risks are therefore closer (less change over time) after adjusting for SEP. This suggests that the observed smoking – mortality association has become more strongly confounded by SEP over time. Again such a finding could be explained in two main ways.

The first is that the association of smoking with SEP has increased over time (less in 1981) – ie. smoking has become more strongly aligned with / patterned by SEP over time. Such a trend is perhaps to be expected, as it is consistent with the description of the way in which the smoking epidemic evolves within a country. Lower socio-economic groups tend to take up smoking at a later stage than higher socio-economic groups (Bolego, Poli et al. 2002). An increased patterning of smoking prevalence by SEP in New Zealand from 1981 to 1996 (steepening gradient) has also been demonstrated by Hill et al (2003).

The second, and not mutually exclusive, explanation is that the independent effect of SEP on mortality has increased over time – it is a more significant risk factor than it used to be. This is a trend seen in a UK study (Marmot and McDowall 1986) where the relative disadvantage of manual compared with non-manual workers (as a measure of occupational class) increased between 1970-72 and 1979/83 – ie. the social gradient in mortality risk has widened. In New Zealand, between 1981 and 1996 income inequality has certainly risen (as measured by the GINI coefficient), and median household disposable income has slightly decreased over this time (although the mean level has slightly increased) (Howden-Chapman and Tobias 2000). The degree of inequality between occupational classes has also increased among males since the 1970s (Pearce, Davis et al. 2002).

It is possible to roughly attribute the proportion of the increased (relative) association of smoking and mortality across the two cohorts to the increasing SEP patterning (and hence

confounding) over time. This can be estimated by looking at the part 2 (multivariable) results, comparing the degree of change over time for age and ethnicity adjusted rate ratios, to the "fully adjusted" (age, ethnicity and SEP) rate ratios (using the excess relative risk). For example, for females within the all-age all-ethnicity stratum, from 1981-84 to 1996-99 there was about a 90% increase in age and ethnicity adjusted excess relative risk ({(2.05-1.55)/(1-1.55)}x100), but only a 66% increase for the age, ethnicity and SEP adjusted excess relative risk ({(1.83-1.50)/(1-1.50)}x100). For males there was a 77% increase in age and ethnicity adjusted excess relative risk ({(2.01-1.57)/(1-1.57)}x100), but only a 55% increase for the age, ethnicity and SEP adjusted excess relative risk ({(1.68-1.44)/(1-1.44)}x100). Therefore, about a third of the increase in the association of smoking and mortality, in relative terms, is due to confounding by SEP.

3.5.3 Residual confounding

It is unlikely that there would be a great amount of residual confounding due to SEP within the multivariable results, and certainly not enough to explain all the risk, as a large range of SEP variables were used.

What is of more concern is that there may be residual confounding of the results due to behavioural and physiological risk factors, such as diet, exercise, alcohol, obesity, hypertension and high cholesterol (although many of these overlap anyway). SEP is used as a proxy for these factors as many of them are determined to a great extent by SEP. But as SEP does not entirely determine these variables, part of the smoking-mortality association that has been demonstrated by this study could be explained by unmeasured risk factors. However, some of the large cohort studies that have controlled for these factors have found that they explain very little of the observed risk. For example, in the Nurses Health Study (Kawachi, Colditz et al. 1997), all-cause mortality relative risk (due to smoking) was adjusted for history of hypertension, diabetes, high serum cholesterol, relative weight, parental history of MI before age 60, past use of oral contraceptives, postmenopausal oestrogen therapy, and age at starting smoking, using multivariate models with little change in the smoking-mortality association (see Table 1, chapter 2, page 19). CHD risk estimates were also adjusted for similar variables, again with little change (Table 2). Multivariate analysis was also applied to the CPS II data (Thun, Apicella et al.

2000), and co-variates included both SEP variables as well as total weekly consumption of vegetables and citrus fruit, and for cardiovascular outcomes also aspirin use, alcohol consumption, body mass index, physical activity, and weekly consumption of fatty foods. For IHD and stroke (all-cause not published), the fully adjusted smoking-mortality results differed only slightly from the age-only adjusted results (Table 2 and Table 3). The authors of the CPS II multivariate paper also note that "only four such studies in the United States have, to our knowledge, reported both multivariate and age-adjusted RR [relative risk] estimates associated with active smoking. In none of these did adjustment for factors other than age or sex substantially alter the RR estimates." (Thun, Apicella et al. 2000). Therefore while it is possible that there is some residual confounding in the NZCMS results due to other risk factors for mortality (and that the estimates of relative risk are slightly too high as a consequence) it is likely that this would be small.

3.6 Overall impact of bias and confounding on results

Although (overall) the results of this study are reasonably precise, the NZCMS is prone to systematic error, such as exposure misclassification, and confounding by factors that are not measured in the censuses. It is difficult to determine quantitatively what effect these errors have had on the observed results overall - although sensitivity analyses at least show that exposure misclassification is unlikely to be responsible for the observed heterogeneity by ethnicity. There are certainly a number of biases described above that suggest the results presented have made an overestimation of the strength of the association between smoking and mortality. However, these may be offset largely or completely by biases in the opposite direction, especially underreporting of smoking, smoking cessation in the three years post census, and (for all-cause mortality) undercounting of latent deaths due to short follow-up. Taken as a whole, the estimates in this thesis may be close to the true effect of smoking on mortality (in New Zealand). The notable heterogeneities seen within the results (eg. ethnicity, time) are probably robust findings and represent important new information for research and policy, as discussed later.

Lastly, it is debatable whether or not the multivariable results – free from confounding – or the age-standardised results – free from selection bias – represent the most accurate, and therefore "final", estimates of the study. Given the moderate, and not insignificant

effect of confounding by SEP, the multivariable results are probably closest to the truth. If the numbers presented here are used for further calculations, and both forms of error need to be eliminated, it may be "most correct" to use the type of derived results illustrated in Table 28 – ie. age-standardised estimates but with "confounding percentages" applied.

3.7 External Validity - Generalisability

As this study captures most of the New Zealand population, it is a fair reflection of what was happening for 25-74 year olds in "real life" during 1981-84 and 1996-99.

As a time trend has been noted, it is likely that the 2003 (and beyond) population has a different degree of relative risk from smoking compared to 1996-99. Therefore even the most recent results are less applicable now, and they will continue to become less generalisable over time. Also the results cannot be generalised to those under the age of 25 and over the age of 75. In passing, it is worth noting that the 2006 census will include smoking, enabling updated estimates for the 2006-9 period in the future (assuming record linkage proceeds).

As there is less accuracy for Māori and Pacific results, it is also harder to generalise the findings for these groups compared to those for non-Māori non-Pacific, or the population as a whole. Nevertheless, this appears to be the first time smoking mortality risk in New Zealand has been estimated for ethnicities other than non-Māori non-Pacific, therefore the results are at least far more applicable than previous studies in this country or overseas estimates such as CPS II.

4 Smoking and Age

In this study, the all-cause rate ratios appear to increase slightly with increasing age of participants (as do the rate differences). This implies a greater (relative) increase in mortality with age for smokers compared to never-smokers in New Zealand. This New Zealand pattern is not seen, or is not as strong, in the overseas literature where age specific relative risks have been published. The rate ratios given for CPS II do not show much change with age, although there is possibly some decrease for males (Thun, Day-Lally et al. 1997a). In the 40-year results for male physicians in Britain, there is a slight increase up to age 74, then a decrease (Doll, Peto et al. 1994). For Framingham there is little change (Freund, Belanger et al. 1993) and there is no consistent pattern seen within the Kaiser Permanente results (Friedman, Tekawa et al. 1997).

The pattern for IHD (and stroke in females) is more obvious and more consistent with overseas findings, and *is in the opposite direction to all-cause mortality relative risk*. Relative risk estimates (rate ratios) for IHD, and for stroke among females, in New Zealand decrease with age (rate differences still increase for the same reason as all-cause – ie. increasing mortality rates). The same downward trend in relative risk is seen in the CPS II results for IHD (and stroke) (Thun, Day-Lally et al. 1997a; Thun, Apicella et al. 2000), as it is for IHD in the British Doctors' study (male and female) (Doll and Peto 1976; Doll, Gray et al. 1980; Doll, Peto et al. 1994). IHD relative risks also generally decrease with age among the MRFIT data (although all-cause was not available) (Neaton and Wentworth 1992), and also for the Kaiser Permanente study (Friedman, Tekawa et al. 1997). The reasons for this pattern are not clear, especially as the precise pathogenic mechanisms for smoking and cardiovascular disease are still not conclusive. However, it seems reasonable to suggest that for older age groups, both never-smokers and smokers have undergone many years of atherogenesis and are both at high risk anyway (as shown by mortality rates in Figure 7 to Figure 10), regardless of smoking status. However, at young ages the possibilities of smoking causing accelerated plaque formation and/or acute thrombosis (Freund, Belanger et al. 1993; Tuut and Hense 2001; Bolego, Poli et al. 2002; Talmud, Hawe et al. 2002), and consequently cardiovascular compromise, are marked pathological changes in relation to non-smoking counterparts, thereby giving rise to a large relative

risk. In other words, smoking is a *relatively* strong risk factor when applied to healthier younger arteries.

5 Smoking and Sex

In this study females generally have a similar relative risk to males of all-cause mortality from smoking, however their rate ratios for IHD and stroke are higher. The data also show that for the overall female group (25-74 years), rate ratios for IHD and stroke are higher than for all-cause mortality, while for males the overall rate ratios for IHD and stroke are similar (standardised) or lower (multivariable) than all-cause.

There is not a consistent difference in cardiovascular relative risks by sex in the literature, and in particular for CPS II the relative risks are similar for men and women for IHD and stroke (Thun, Day-Lally et al. 1997a). Nevertheless, other overseas studies have found a sex / gender disparity, although not always in the same direction. It was noted in a commentary on this issue by Prescott, that earlier studies such as Framingham and the British Doctors study found smaller relative risks in women (Prescott 2001), but that "several more recent studies find higher RR in women." This was also noted in a review by Bolego et al in 2002.

For example, higher relative risks in women have been found for myocardial infarction (morbidity) in the Danish population, even after controlling for potential confounders (including education and physiological and behavioural risk factors) (Prescott, Osler et al. 1998; Prescott, Hippe et al. 1998; Prescott, Scharling et al. 2002 Sep). Another study cited by Bolego et al (2002), found a relative risk of coronary death per ten cigarettes per day of 1.8 in women and 1.2 in men (Tverdal, Thelle et al. 1993). Also, the relative risks for IHD found in the Nurses Health Study (Kawachi, Colditz et al. 1997) were well above (over 4) any estimates that have been made for men in large cohort studies (although there is no internal comparison for males). However, findings from a 20-year Scottish cohort study (started in 1971) published in 2001 did not show any significant gender differences in relative risk for vascular causes of death (Marang-van de Mheen, Smith et al. 2001).

Two New Zealand case-control studies (cited in chapter 2, section 4) also found higher relative risks for women for cardiovascular disease (although the confidence intervals are wide). The Auckland stroke study found a higher stroke odds ratio in women (4.50, CI 3.03-6.69) compared to men (4.07, CI 2.74-6.04) (Bonita, Duncan et al. 1999), and

McElduff et al (1998) found higher odds ratios for coronary death in Auckland women 5.0 (95% CI 2.8-8.9) compared to men 3.0 (95% CI 2.1-4.1).

It has been suggested that the conflicting results between studies may be due to several things, such as differences in smoking exposure (smoking habits may differ), uncontrolled confounders or unidentified effect modifiers (eg. oral contraceptive use), different age distribution of women included into the studies, and cessation during follow-up (generally more men than women quit smoking) (USDHHS 2001a; Bolego, Poli et al. 2002). Also, it is possible that older studies may not have observed a sex disparity due to major differences in smoking habits between male and female smokers from older birth cohorts (Prescott, Scharling et al. 2002 Sep).

If the difference in relative risk between men and women is "real", it could be due to biological and/ or behavioural factors. The biological hypothesis is based on the anti-oestrogenic effect of smoking, which would result in younger women smokers effectively getting a "double whammy" from tobacco. Not only do they incur the same pathological effects as men, but they also lose their "natural" oestrogenic protection from IHD compared to non smoking women, thereby gaining an even greater relative mortality risk (Prescott 2001; Bolego, Poli et al. 2002; Prescott, Scharling et al. 2002 Sep). This would tend to explain the particularly high cardiovascular relative risks seen in young women, and this is where the disparity is greatest, but does not explain the disparity that is still seen at older ages. For example, for 1996 the age and ethnicity standardised IHD relative risk (rate ratio) for 25-44 year old women is 3.83 (1.64-8.94), compared to 2.22 (1.56-3.16) for men. For 65-74 year olds the comparative relative risks are 2.57 (2.20-3.00) for women and 1.81 (1.61-2.03) for men.

Behavioural factors may also play a role in the difference between male and female relative risks seen in New Zealand. These could include differences in smoking motivation, for example it has been noted that "women more often use cigarettes as a buffer against negative feelings, whereas men smoke more habitually, or to increase positive feelings" (Jacobson 1981 as cited in Payne 2001). This may in turn alter smoking behaviour. For example, it has been suggested that smoking under time constraints such as taking 'time out' from childcare, may encourage women to smoke harder and faster,

inhaling more, especially when under stress (Payne 2001). There may be post-disease behavioural differences as well (eg accessing health services), although it is not obvious why there would be a greater difference in this behaviour between smoking and non-smoking women, as compared to smoking and non-smoking men.

Given the high prevalence of smoking among young women (Ministry of Health 2002a), it is also plausible that women in this study overall may have an earlier initiation of smoking, and therefore a longer exposure, and this has not been taken into account. It has been noted that in New Zealand, females under the age of 15 are more likely to smoke than their male counterparts, and this may go back as far as the 1970's (Ministry of Health 2002a). In 2001, a national survey reported 15.2% of girls aged 14-15 smoked, compared with 11.6% of boys (Ministry of Health 2002a).

While these results show a greater *relative* risk of cardiovascular disease for women, it is important to keep in mind the fact that overall mortality rates are lower than men, as is the absolute excess risk from smoking (rate difference) for IHD and all-cause mortality (rate differences for stroke are somewhat similar).

6 Smoking and Ethnicity

6.1 Mortality Rates

Overall mortality rate comparisons and time trends for the three ethnic strata analysed in this study are consistent with other published results from the NZCMS that have used linked datasets (thereby overcoming problems with numerator-denominator bias) (Ajwani, Blakely et al. 2003). In these results we see higher mortality rates for Māori and Pacific compared to non-Māori non-Pacific, and these disparities may in fact have increased from 1981 to 1996. Over time, all-cause mortality rates have dropped markedly for non-Māori non-Pacific, however there is little, if any, downward trend for Māori and Pacific. IHD rates have also dropped markedly for non-Māori non-Pacific. Māori have a decline for IHD but this is less, especially among Māori males who have made little progress (Pacific time trend unclear). The small decline in IHD mortality in Māori men was also seen in results from the ARCOS study (Bell, Swinburn et al. 1996). Stroke rates have dropped markedly for non-Māori non-Pacific, probably also for Māori females, however any trends for other groups are difficult to determine.

6.1.1 Never smokers

It is also important to compare mortality rates by ethnicity for never-smokers only, which may be the first time this has been possible in the New Zealand population. Excluding the influence of smoking exposure, we still see a marked disparity in mortality rates by ethnicity (for Māori and Pacific), and on a relative scale this has increased over time. For example, the 1981 all-age, age standardised all-cause mortality rate for Māori never-smokers is 1450 deaths per 100,000 person-years, falling to 1230 in 1996. The non-Māori non-Pacific never-smoker rate was 687 deaths per 100,000 person-years in 1981, falling to 442 in 1996. This gives a never-smoker Māori to non-Māori non-Pacific rate ratio of 2.11 for 1981 (1450 over 687), rising to 2.78 in 1996 (1230 over 442). From the other perspective, mortality rates have fallen by a similar absolute amount in both Māori and non-Māori non-Pacific never-smokers, but with a corresponding increase in relative risk "from ethnicity".

Higher mortality rates for Māori never-smokers (in the all-age strata) can be calculated for females also, and for IHD and stroke, and rate ratios have increased over time. For all-cause mortality, the Māori / non-Māori non-Pacific rate ratios within never smokers are 2.11 (male) and 2.46 (female) for 1981, and 2.78 (male) and 2.90 (female) for 1996. For IHD, the rate ratios are 1.77 (male) and 2.73 (female) for 1981, and 2.82 (male) and 4.03 (female) for 1996. For stroke, the rate ratios are 1.93 (male) and 2.62 (female) for 1981, and 3.09 (male) and 3.13 (female) for 1996. Again, it should be noted that absolute (rate) differences between Māori and non-Māori non-Pacific never-smokers show a different pattern, and are similar for most categories when comparing 1981 and 1996, or have slightly reduced over time.

These ethnic disparities among never-smokers are even more alarming when considering the likelihood that many lifestyle behaviours that affect health tend to cluster together (as per chapter 3 - methods). For example, smokers in New Zealand may be more likely to drink alcohol (in a hazardous way), have a poor diet and exercise less. Conversely, the never-smokers may tend to eat and drink more healthily and exercise more. Assuming this clustering was common by ethnicity, this suggests that ethnic differences in mortality cannot just be attributed to smoking exposure or clustering of other unhealthy individual behaviours, and that other determinants have a direct impact on health inequalities as well, such as socio-economic position (Ministry of Health 2002c). Additionally, there are likely to be ongoing effects of colonisation including or overlapping with institutional, personal, and internalised racism (Reid, Robson et al. 2000; Ministry of Health 2002c). The significant role that such structural factors play in determining health outcomes, in addition to "behaviour pathways", has been emphasized in numerous scientific studies and official reports worldwide (Black, Morris et al. 1992; Whitehead 1992; Acheson, Barker et al. 1998; National Health Committee 1998; Howden-Chapman and Tobias 2000; Ministry of Health 2002c). Examples include material access to preventive measures (eg. safer cars) and better health care (eg. private surgery), and psychosocial factors such as chronic stress (via the HPA axis) (Brunner 1997; Krieger 2001; Ministry of Health 2002c).

6.2 Effect Measure Modification

With regards to the association of smoking with mortality, the heterogeneity of smoking rate ratios between the different ethnicities is an important finding. Essentially, there appears to be a smaller relative difference overall between the mortality rates of current smokers, ex-smokers and never-smokers among Māori and Pacific compared to the pattern seen among non-Māori non-Pacific. This has resulted in lower rate ratios as a measure of the smoking-mortality relationship within Māori and Pacific groups. In epidemiological terms, this can also be thought of as effect measure modification (of the smoking-mortality association) by ethnicity. The presence of this heterogeneity or effect modification is consistent throughout the results, being apparent for crude, age-standardised, and multivariable analyses, for all causes of death examined here, for both sexes and both years. And as previously mentioned, it is statistically significant.

6.2.1 Overseas findings

Unfortunately, not a great deal of comparison can be made between these results and findings from other studies, as the presence of effect modification of the smoking-mortality association by ethnicity appears to be rarely investigated in the mainstream literature. As mentioned in chapter 2, smoking-mortality and smoking-morbidity analyses from the Auckland Risk Factor Study (Norrish, North et al. 1995), the Auckland Stroke Study (Bonita, Duncan et al. 1999) and the case-control study by McElduff et al (1998), did not include Māori or Pacific people. The case-control study by Bonita et al (1986) did not report ethnicity. Overseas, the large cohort studies on smoking and mortality have tended to publish results that either do not give an ethnic breakdown of RRs, including CPS II (93% white) (Thun, Day-Lally et al. 1997a; Thun, Apicella et al. 2000), the British Doctors study (Doll, Peto et al. 1994) and the Nurses Health Study (Kawachi, Colditz et al. 1997); that restrict analyses only to white participants (eg. CPS II (Malarcher, Schulman et al. 2000), MRFIT (Neaton and Wentworth 1992)); or that compare mortality rates only ((Davey Smith, Neaton et al. 1998) MRFIT).

Where there has been some comparison of the smoking-mortality association by race or ethnicity overseas, the results are not conclusive. One paper published from the CPS I study (97% white (Thun, Day-Lally et al. 1997a)) illustrated CHD mortality ratios

(compared to never smoked) by level of cigarette smoking, with little difference between those for black and white males) (Garfinkel 1984). There was more difference in ratios between black and white women, however the number of deaths in each group for black women was small, and the pattern was not consistent (higher ratios for black women smoking 1-9 cigarettes per day, lower ratios for black women smoking 10-19 and 20+ per day) (Garfinkel 1984). From the MRFIT data (men only), regression coefficients for mortality by level of smoking (not rate ratios) have been calculated separately for black (n=23,490; 6.7%) and white (n=325,384; 93.3%) men (Neaton, Kuller et al. 1984). Both groups showed a clear association for all-cause mortality and CHD mortality risk (unclear for stroke among black males) and the coefficients did not differ significantly between the two groups (Neaton, Kuller et al. 1984). One cohort study on a population that was more ethnically diverse (although smaller numbers than MRFIT) is the Kaiser Permanente study – 58% of subjects were white, 25% black and 11% Asian (Friedman, Tekawa et al. 1997). This study did show some heterogeneity of relative risk estimates (current vs never smoker) by race, however there was no clear consistent pattern and where it did occur the 95% confidence intervals tended to be wide and overlapping.

So the important question remains – how are the lower rate ratios for smoking among Māori and Pacific people to be interpreted? And what are the reasons for this heterogeneity? Reiterating a point made earlier, these rate ratios only reflect smoker / never-smoker comparisons within each ethnic group. They do not represent lower overall mortality rates for Māori and Pacific compared to non-Māori non-Pacific. What they do suggest is that *on a relative scale*, cigarette smoking does not appear to increase the risk of mortality as much in the Māori population as it does in the non-Māori non-Pacific population. But the effect of smoking on mortality among Māori is not trivial, illustrated by the *absolute risk difference* that smoking confers among Māori. From the most recent year, 1996, the absolute excess risk from smoking (rate difference) was 627 deaths per 100,000 person-years for Māori males, and 368 deaths per 100,000 person-years for Māori females. This compares to 540 (male) and 340 (female) in this same time period for non-Māori non-Pacific.

6.2.2 Reasons for Effect Measure Modification

There are a number of possible reasons for the effect measure modification of the relative risks by ethnicity.

6.2.2.1 Rate difference homogeneity and variation in mortality rates

The first, and perhaps most obvious explanation for the observed rate ratio heterogeneity is that it is a mathematical effect due to the underlying higher mortality rates among Māori and Pacific, for both smokers and never smokers. This would result in smaller rate ratios even if the rate differences were the same. This seems a logical conclusion for the 1996-99 results, where the rate differences between Māori and non-Māori non-Pacific are reasonably similar. And it could be argued that the absolute difference is a more appropriate measure in this situation. However, in 1981 both the rate ratios *and* the rate differences for Māori are smaller than those for non-Māori non-Pacific (although the confidence intervals are wider).

Reflecting on the apparent rate difference homogeneity in 1996-99 though, it is interesting to note that a lack of "biological interaction" of ethnicity and smoking would give rise to this homogeneity as demonstrated by the counterfactual model (Rothman and Greenland 1998). That is, ethnicity and smoking may have independent effects. Given the distal nature of, particularly, ethnicity, it is difficult to fully explain what this might mean. However, if Māori mortality rates begin to improve again soon, the relative risk of smoking would be expected to increase.

Factors driving up the underlying mortality rates (and therefore producing lower rate ratios) for Māori and Pacific regardless of smoking exposure, and beyond the SEP effect – that we have (in theory) removed – include those discussed previously for Māori never-smokers (section 6.1), such as structurally mediated effects of colonisation and/or racism (including health service effects pre and post onset of disease) as well as differential effects from so called "lifestyle" factors – eg, diet, exercise, obesity (although the effect of these downstream risk factors may also be largely removed by controlling for SEP).

Considering these lifestyle determinants, a clustering of a number of risk factors in high prevalence among Māori and Pacific (Sarfati, Scott et al. 1999; Ministry of Health 2002b) will lead to higher background mortality rates, meaning the rate ratio for smoking among Māori is less. In other words the relative risk of Māori smokers follows the same pattern seen overseas for smokers with higher cholesterol, blood pressure and diabetes, who show a lower relative risk for cardiovascular disease compared to smokers without these risk factors.

Another perspective is that the determinants of high mortality, in particular the proximal or structural causes which drive distal risk factors, are having such an overwhelming and pervasive effect, regardless of smoking status, that there is only a small amount of risk that tobacco smoke can add for the current smokers, at least on a relative scale.

While these explanations for the observed rate ratio heterogeneity by ethnicity are the most likely, additional mechanisms should be considered. In particular given that there are some notable variations in rate differences in 1981 – for example a small absolute gap between Māori smokers and never smokers (this would contribute to lower rate ratios even if underlying mortality rates were similar). Some possibilities are discussed below.

6.2.2.2 Reverse confounding by "lifestyle" factors

One possible reason for the observed heterogeneity of rate ratios is that the mortality rates among never-smokers are inaccurate. For example, the high mortality rates seen among Māori never-smokers may be due to a higher proportion of other risk factors for mortality (not cigarette smoke) in this group compared to Māori current smokers. That is, confounding by lifestyle is causing us to underestimate the smoking rate ratios within Māori. However, this seems most unlikely given that unhealthy lifestyles have been shown to cluster with smoking among Māori (Sarfati, Scott et al. 1999).

6.2.2.3 Exposure misclassification

Smoking mismeasurement should also be considered. As discussed in an earlier section, sensitivity analyses tend to suggest that the rate ratio heterogeneity in 1996-99 cannot be explained by misclassification of smoking status. It is possible however, that some of the

rate difference heterogeneity in 1981-84 could potentially be attributed to this (eg. decreasing the absolute gap between Māori smokers and never smokers), and therefore influencing the rate ratios for this earlier cohort. Other types of exposure misclassification that could be affecting observed mortality rates, and therefore rate ratios, include differences by ethnicity in age at initiation of smoking, duration of smoking, amount smoked, and smoking behaviours (such as compensatory smoking) – factors that were not captured in this study.

What evidence there is on these factors does not tend to suggest that they are contributing to the lower rate ratios seen among Māori and Pacific. For example, cigarette consumption rates appear similar for Māori smokers and non-Māori non-Pacific smokers (number of cigarettes per day per smoker). Using tax-paid consumption data, Māori smokers consumed 23 cigarettes per day in 1981, compared to 24.8 in the total NZ population. In 1996, the respective figures were 17.3 (Māori) and 18 (total) (Laugesen and Clements 1998).

The New Zealand Health Survey (1996-7) found that Māori smokers tended to start smoking earlier than other ethnic groups ($p < 0.0001$) – for example 31.4% of Māori ever smokers reported starting to smoke regularly prior to age 15 years compared to 17.6% of European/Päkehä and 13.8% (7.1–20.5) of Pacific smokers (Sarfati, Scott et al. 1999). This survey also found differences in the duration of smoking by ethnicity, with 45.5% of Māori, 39.5% of European/Päkehä and 41.5% of Pacific people reporting that they had smoked for over 20 years. Both of these findings however would suggest higher mortality rates, and a stronger smoking-mortality association among Māori and Pacific, and therefore do not explain the lower relative risk among these groups.

6.2.2.4 Passive smoking

It is also worth raising the issue of passive smoking, which could be thought of as type of exposure misclassification (of exposure to cigarette smoke) and have the same effect. The mortality rates of many never-smokers may be closer to that of the current smoker group due to their exposure to environmental tobacco smoke, with its consequences being similar (although less strong) to active smoking (Hill 2003). And there is some evidence

that this situation is more common among Māori than Non-Māori, thereby raising never smoker rates for Māori to a greater extent. As reported by Woodward and Laugesen (2001), "a Māori non-smoking adult or child is likely to be surrounded by twice as many smokers per household, on average" (original citation Crampton et al 2000).

6.3 Pacific People

The results from this study suggest that, as a general trend, Pacific ex-smokers have higher mortality rates than Pacific current smokers, consequently giving ex-smokers the highest relative and absolute mortality risk from smoking within this ethnic group. Most of the 95% confidence intervals for these data are wide and tend to overlap (especially so for IHD and stroke), therefore this unusual finding could be due to chance. However as this pattern is seen across many of the analyses, it should at least be considered as a possible real finding.

A possible reason for the high risk observed among Pacific ex-smokers is that smoking behaviour may have changed over time. Pacific ex-smokers may represent a cohort that smoked more heavily and for a longer duration, accumulating more "damage", whereas current smokers around the years 1981 and 1996 were short-term smokers. However, it seems odd that on average, tobacco exposure in the relevant period (eg. pack-years in the five years before census night) was less among current smokers on census night compared with ex-smokers on census night.

It may be that Pacific people are able to give up smoking more easily, or are more inclined to do so. And if smoking cessation is prompted by health concerns (including symptoms of disease), then this could add up to even more Pacific ex-smokers at the start of each cohort that are in poor health (health selection effect).

However, overall the Pacific findings (in particular the tendency to higher relative risks for ex-smokers) should be treated cautiously.

7 Smoking and Time

7.1 Drivers of rate ratio changes over time

Overall, the relative risk of mortality from smoking in New Zealand has increased between the 1981-84 period and the 1996-99 period, for all-causes, IHD and stroke. There appear to be two types of drivers behind this time trend. The first is an overall decline in some of the mortality rates in the all-age all-ethnicity combined group by a similar amount for current smokers and never-smokers alike – ie. a constant rate difference over time. The second is a steeper, or faster decline in some mortality rates for never-smokers compared to current smokers over this time period – ie. increasing rate difference in addition to increasing rate ratio.

IHD and stroke mortality appear to follow the first pattern, where rate differences have been fairly stable (at least for the all-age all-ethnicity grouping). If, as in this case, mortality rates decline for all smoking strata by the same absolute amount, the ratio between the high risk group (current smokers) and the lower reference group (never smokers) will naturally rise (for example 4 over 1 gives a higher ratio than 6 over 3, even though the gap is the same). As explained in chapter 2, in the situation of decreasing mortality rates, there cannot be homogeneity (no change) of both the rate differences and rate ratios.

While all-cause mortality rates have also declined overall, the second rule seems to apply, with increased rate differences over time, adding to the increase in relative risk. In other words, all-cause mortality rates have declined more in absolute terms among never smokers (a steeper drop) than current smokers.

Increases in relative risk of smoking mortality have also been observed overseas, as mentioned in chapter 2. When the results of 40-years of follow-up of male British Doctors was examined by time period (first vs second 20 years), it was found that mortality rates had declined (for all smoking groups) and relative risk estimates had risen for all-cause mortality, IHD and stroke in the second half of the study (1971-1991) (Doll, Peto et al. 1994) – see Table 1, Table 2 and Table 3. Between the CPS I (1959-65) and CPS II

(1982-88) studies (which essentially used the same methodology), there was also a decline in mortality rates and an increase in relative risk from smoking for these causes of death (for both males and females) (Thun, Day-Lally et al. 1997a).

As with the NZCMS results, the British Doctors study found increased rate differences over time for all-cause mortality, as did the CPS studies among females (males unchanged), giving some credibility to the apparently more rapid decline in all-cause mortality among never smokers in New Zealand. For IHD, rate differences actually decreased (ie. smaller absolute risk) over time for the Britsh Doctors study and CPS. For stroke there was an increase seen in the British Doctors study (Doll, Peto et al. 1994), a small increase in CPS females and a small decrease in CPS males (Thun, Day-Lally et al. 1997a).

7.2 Reasons for overall decline in mortality rates

The decline in mortality rates overall that is observed in New Zealand, UK and the United States is likely to be due to a range of determinants, including changes in risk factor levels, and advances in medical care. As noted in chapter 2, in New Zealand much of the change in cardiovascular risk factors appear to have occurred prior to 1981, however it has been suggested that we would expect a lag between such changes and a drop in mortality rates (Williams 1989; Law and Wald 1999).

Medical advances that have probably reduced mortality include better antihypertensive, lipid lowering and antithrombotic treatments. One could expect many of these changes to impact of cardiovascular mortality (rather than other causes) to a greater extent, and for never smokers and smokers alike, which could be why we have seen IHD and stroke rates decline in New Zealand by a similar absolute amount for smokers and never smokers overall. It is concerning that Māori mortality rates have not similarly declined over the same time period across strata of smoking, and provokes the question of whether health care improvements have been accessed equally by all ethnic groups.

7.3 Differential declines in mortality between smokers and never-smokers

What we have not seen in New Zealand (and overseas) is a similar decline in all-cause mortality for both smokers and never smokers. Never smokers have benefited more on an absolute scale. It is important to note that this must also be determined by causes of death other than IHD and stroke, as seen in both the British Doctors and CPS studies. In the British Doctors study mortality rate reductions among non-smokers were accompanied by increases among smokers for some causes of death, especially cancers that are caused by smoking, and respiratory disease other than COPD. During follow-up, lung cancer rates increased 19% in smokers, while they were unchanged in non-smokers (Doll, Peto et al. 1994). From CPS I to II, lung cancer death rates among current cigarette smokers compared with never smokers nearly doubled in men and increased nearly sixfold among women (Thun, Day-Lally et al. 1997a). Increases were still seen after controlling for cigarette consumption (per day) and years of smoking at enrolment (although rates were diminished somewhat) (Thun and Heath 1997b). It is therefore likely that in New Zealand, the decrease in deaths from IHD and stroke among smokers is being partly offset by increased smoking-related deaths from other causes such as lung and other cancers.

So what is behind these patterns? Why have we not seen the same drop in mortality rates among smokers that we have seen in never-smokers? Why have cardiovascular mortality rates perhaps not fallen even more for smokers than non-smokers, so that on a relative scale the risk from smoking remains constant? Why might mortality rates from other causes of death be increasing among smokers? Part of the reason may be that decreases in cardiovascular mortality have partially caused an increase in cancer rates among smokers (later in life) by reducing competing causes of death (Thun, Day-Lally et al. 1995). Another explanation that has been applied to the CPS and British results is that smokers in more recent years have received a more intensive or more toxic exposure to the contents of tobacco smoke, due to differences such as a longer duration of smoking, more harmful smoking behaviour or changes in the chemical constituents of cigarettes.

Current smokers in the 1996 cohort are likely to have smoked for a longer duration than those in 1981, not only because 1996 is at a later stage of the smoking epidemic, but it is also possible that more recent smokers started smoking at a younger age. Recent results

may therefore be revealing the full effect of smoking-related pathology among people who were long-term smokers, especially for cancer deaths. In other words health outcomes that depend on cumulative exposure, or that are latent, are being fully realised (prolonged smoking is more important for lung cancer than current use (Thun and Heath 1997b))

Smoking behaviour may also have changed over time. Although reported cigarette consumption appears to have actually decreased in New Zealand (see ethnicity section above), it has been suggested that later smokers may include more "'hard core' smokers who cannot quit despite health and social concerns" (Thun, Day-Lally et al. 1995). Such smokers may "inhale more deeply, take more puffs per cigarette, or retain the smoke longer in their lungs than did smokers in the past" (Thun, Day-Lally et al. 1995). This behaviour may be partly to compensate for lower nicotine levels in more recent cigarettes. Advertising "lower tar" cigarettes may have also encouraged continued smoking by adults who would otherwise quit (Thun and Heath 1997b).

We may have expected to see greater declines in mortality rates among smokers due to decreases in cigarette tar yields, which has likely occurred in New Zealand pre-1980 even though post-1980 yields appear stable. However, even though tar yields have decreased in the United States since the 1950s (USDHHS 1989; Thun and Heath 1997b) and this has been shown to be associated with lower risk of lung cancer, smoking-related lung cancer mortality from CPS I to CPS II has actually increased. It may be that decreased tar yields have been offset by other changes such as those as mentioned above (and below), as well as compensatory smoking (subsequent to decreased nicotine yields), and that in fact we would have seen even bigger increases in lung cancer mortality if tar yields had not decreased. It may also be that other chemical changes in cigarettes (such as those mentioned in chapter 2 – and unknown changes) have had an impact, and even that cigarettes actually even become more toxic over time for some causes of death.

Even though changes in risk factors other than smoking have been mentioned as factors contributing to an overall decline in mortality rates, it is also possible that there have been differential changes among smokers and never smokers in the distribution of these risk factors over time. These risk factors may therefore differentially confound or modify the effect of smoking on mortality, causing a strengthening of the observed association over

time. This possible confounding has been accounted to some extent in this study (by controlling for SEP). Accordingly, it appears that about a third of the increased relative risk (excess) observed in the NZCMS results was due to increased confounding of the smoking-mortality association over time by SEP. By extension, it is likely that a proportion of the remaining two-thirds of the increase of the smoking-mortality association over time may be due to increasing confounding by the increasingly skewed distribution of other risk factors by SEP over time.

8 Implications for Health Policy and Further Research

The main implications of this study are suggested by the very reason for conducting it – no measures of smoking mortality effect were previously available for the whole New Zealand population. For New Zealand research and policy requiring relative risk estimates for smoking, these new findings can be used in place of those "borrowed" from overseas studies – as already noted, there is some variation between the CPS II relative risk estimates and those found from the NZCMS overall. This study also has wide implications in demonstrating the importance of epidemiological research that is specific to the country and populations of interest, and this is particularly highlighted in three areas: age structure, time, and most importantly, ethnicity.

The first point is to briefly reiterate that made in chapter 2. That is that when calculating age-standardised relative risk, the age structure of the population used as a standard can have a sizeable bearing the results, depending on the relationship between age and the risk of interest. It is therefore important to bear this in mind when comparing results of different studies, and even more so if "borrowing" overseas results to apply to the New Zealand population. If the latter is unavoidable, the age-structure of the standard used should be examined (if possible), and compared to that of the New Zealand population to assess the likelihood of this problem occurring. Even more preferable, if age-specific crude rates are available, the relative risk estimates can be re-standardised to the New Zealand population.

With regards to time, it has been shown in both the NZCMS and overseas results, that relative risk from smoking is changing. Therefore it is important not only to utilise the most recent results available, but also to periodically measure and update risk estimates – including those presented here. This also emphasizes the need for country, sex and ethnicity specific data, as the tobacco epidemic is at a different stage in different populations, therefore even current figures in one population are still not necessarily applicable to another.

Perhaps the most important information to be taken from this research and applied elsewhere is that ethnicity specific data is vital. Summary statistics are known to be often inappropriate, and this usually leads to stratification of risk by age and sex. The effect modification seen here by ethnicity emphasizes that data in New Zealand must also at least be presented for Māori, Pacific and non-Māori non-Pacific separately as well as combined. Data that are not ethnicity specific, or that are based on studies excluding Māori and Pacific, cannot be assumed to be applicable to these populations. And it has now been demonstrated that it is definitely not correct to do so for smoking mortality risk. It is possible that as researchers become more aware of the need for ethnicity specific data, methodologies will change.

Ethnicity specific data is particularly important for informing health policy, including priority-setting and strategies for reducing health inequalities. The new information supplied by this study can be used to re-calculate estimates of the attributable burden of mortality from smoking in New Zealand (which previously used relative risk estimates from overseas, such as CPS II), and produce accurate measures of ethnicity specific attributable risk. For example, the lower smoking rate ratios seen among Māori compared to non-Māori non-Pacific will probably translate into lower attributable risk estimates from this exposure for Māori. However, perhaps one caution that should be attached to using such data is that it must be interpreted correctly. For example, the lower relative risk estimates for Māori could be misconstrued as meaning that cigarette smoking is not an important issue for Māori health. In truth, the absolute effect of smoking (rate differences) is greater among Māori than any other ethnic group for the 1996 population. But even more importantly, the lower smoking rate ratios (and probably lower population attributable risk from smoking), and the higher mortality rates for never-smokers among Māori point to the relatively greater significance of causes of poor health and health inequalities other than smoking (and any associated behaviours) among Māori, reiterating the need to focus not just on individual risk factors but on broader structural determinants as well.

The observed differences by ethnicity, and how they point to other determinants of health, should inform a number of activities within the health sector. These include clinical

guidelines and the use of risk profiling techniques for cardiovascular disease, which, if only based on smoking and other risk factor data will miss the important contribution of ethnicity to predicting mortality risk, over and above the predictive ability of these classic risk factors.

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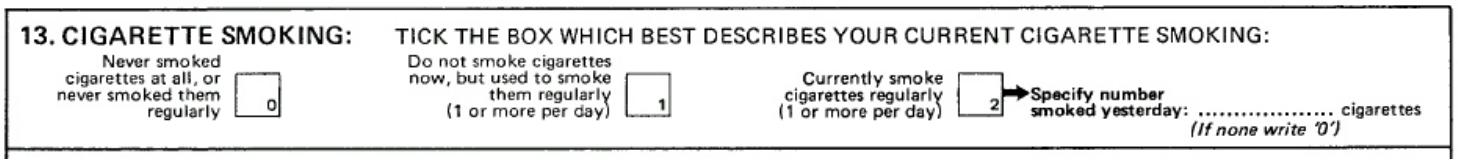
Appendix A: New Zealand census questions

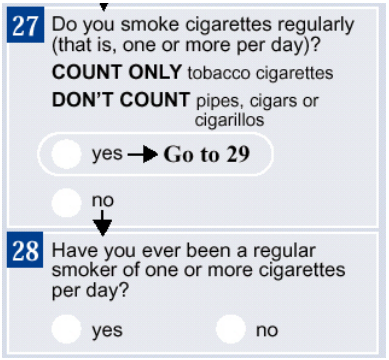
1 Questions pertaining to smoking

In the 1981 and 1996 New Zealand censuses, these questions on smoking were asked of people aged 15 years and older.

1981 Census →

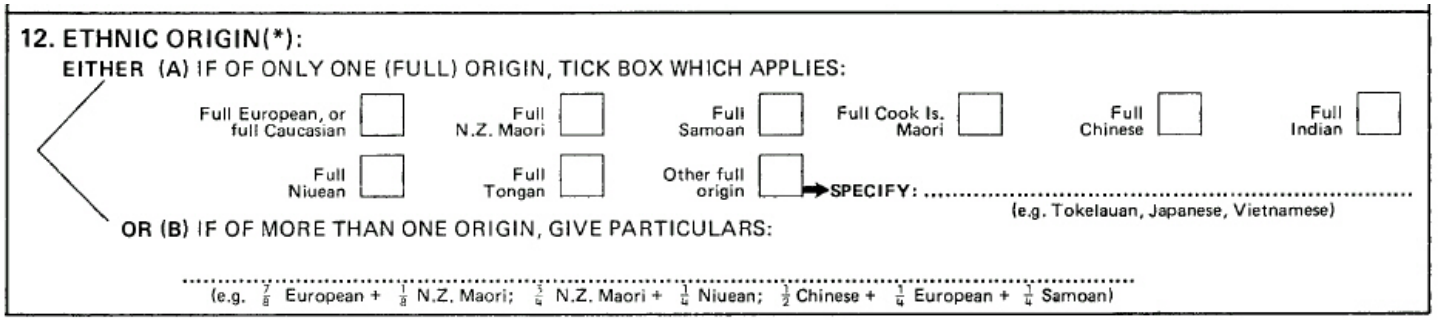


1996 Census →



2 Questions pertaining to ethnicity

1981 Census →


12. ETHNIC ORIGIN(*):

EITHER (A) IF OF ONLY ONE (FULL) ORIGIN, TICK BOX WHICH APPLIES:

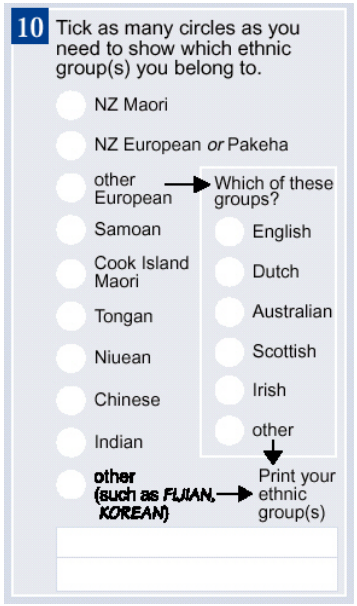
Full European, or full Caucasian ☐ Full N.Z. Maori ☐ Full Samoan ☐ Full Cook Is. Maori ☐ Full Chinese ☐ Full Indian ☐

Full Niuean ☐ Full Tongan ☐ Other full origin ☐ → SPECIFY: ..... (e.g. Tokelauan, Japanese, Vietnamese)

OR (B) IF OF MORE THAN ONE ORIGIN, GIVE PARTICULARS:

..... (e.g. 7/8 European + 1/8 N.Z. Maori; 3/4 N.Z. Maori + 1/4 Niuean; 1/2 Chinese + 1/4 European + 1/4 Samoan)

1996 Census →


10 Tick as many circles as you need to show which ethnic group(s) you belong to.

- NZ Maori
- NZ European *or* Pakeha
- other European → Which of these groups?
  - English
  - Dutch
  - Australian
  - Scottish
  - Irish
  - other →
- Samoan
- Cook Island Maori
- Tongan
- Niuean
- Chinese
- Indian
- other (such as *FIJIAN, KOREAN*) → Print your ethnic group(s)

Appendix B: Additional part 1 data

The following pages in Appendix B contain tables of data that correspond to the part 1 graphs in chapter 5 (they are broken down by sex, ethnicity and age).

All-cause data are given first, then IHD, then stroke.

Within each cause of death, the presentation of tables has the following order:

Male Deaths and Mortality Rates
Female Deaths and Mortality Rates
Male Standardised Rate Ratios and Rate Differences
Female Standardised Rate Ratios and Rate Differences

Table 30: Male All-Cause Mortality Data by Age and Ethnicity (First Restriction)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate†	(95% CI)	No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate†	(95% CI)	No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate†	(95% CI)
1981-1984													
Maori	25-44	105	293	302	(220 - 384)	243	327	362	(298 - 427)	60	256	270	(176 - 364)
	45-64	291	1,762	1,846	(1,560 - 2,132)	504	1,941	2,132	(1,882 - 2,382)	252	1,952	1,971	(1,653 - 2,288)
	65-74	168	5,450	5,529	(4,388 - 6,670)	216	6,712	6,748	(5,543 - 7,953)	186	6,202	6,279	(5,034 - 7,524)
	all age	564	1,014	1,450	(1,278 - 1,621)	963	935	1,724	(1,556 - 1,892)	495	1,270	1,563	(1,374 - 1,752)
Pacific	25-44	24	123	147	(66 - 228)	33	164	173	(92 - 253)	6	96	114	(55 - 278)
	45-64	42	876	935	(583 - 1,288)	60	1,017	1,076	(726 - 1,425)	30	1,385	1,425	(810 - 2,040)
	65-74	27	4,170	4,254	(2,109 - 6,399)	24	3,817	3,850	(1,903 - 5,797)	33	8,372	8,871	(5,123 - 12,620)
	all age	93	387	899	(619 - 1,178)	120	444	915	(656 - 1,175)	69	819	1,586	(1,095 - 2,077)
NonM-	25-44	615	127	131	(118 - 145)	741	190	194	(176 - 212)	306	140	136	(116 - 156)
NonP	45-64	1,734	743	732	(691 - 772)	3,831	1,379	1,285	(1,237 - 1,333)	2,847	1,053	885	(845 - 924)
	65-74	2,190	3,114	3,113	(2,963 - 3,264)	3,600	5,083	5,160	(4,966 - 5,354)	5,037	4,358	4,347	(4,209 - 4,485)
	all age	4,536	575	687	(663 - 711)	8,169	1,106	1,151	(1,122 - 1,181)	8,190	1,356	886	(862 - 909)
1996-1999													
Maori	25-44	225	289	298	(244 - 353)	354	371	381	(324 - 437)	84	229	226	(156 - 295)
	45-64	450	1,403	1,421	(1,263 - 1,580)	648	1,924	2,179	(1,973 - 2,385)	408	1,553	1,561	(1,378 - 1,744)
	65-74	318	4,908	4,939	(4,300 - 5,579)	282	7,511	7,675	(6,613 - 8,737)	327	5,634	5,755	(5,033 - 6,478)
	all age	996	851	1,230	(1,133 - 1,327)	1,284	964	1,857	(1,711 - 2,002)	816	1,198	1,335	(1,223 - 1,446)
Pacific	25-44	81	224	227	(161 - 294)	69	241	245	(167 - 323)	21	266	262	(111 - 412)
	45-64	180	1,189	1,292	(1,058 - 1,525)	150	1,270	1,350	(1,074 - 1,626)	51	1,158	1,190	(788 - 1,593)
	65-74	90	3,358	3,441	(2,541 - 4,341)	60	4,474	4,659	(3,181 - 6,137)	75	6,965	6,976	(5,001 - 8,951)
	all age	351	651	974	(837 - 1,111)	279	669	1,144	(944 - 1,345)	144	1,114	1,363	(1,083 - 1,643)
NonM-	25-44	702	106	105	(95 - 115)	603	184	185	(166 - 204)	282	126	122	(102 - 142)
NonP	45-64	1,653	406	422	(399 - 445)	1,830	931	977	(926 - 1,027)	1,878	608	557	(528 - 585)
	65-74	2,208	2,052	2,056	(1,963 - 2,150)	2,049	4,597	4,678	(4,456 - 4,899)	4,593	3,013	2,948	(2,855 - 3,041)
	all age	4,563	387	442	(427 - 456)	4,479	789	982	(949 - 1,015)	6,753	987	601	(583 - 619)

Male All-Cause Mortality Rates by Smoking Status NZCMS

*Random Rounded

† Deaths per 100,000 Person-Years

Table 31: Female All-Cause Mortality Data by Age and Ethnicity (First Restriction)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate†	(95% CI)	No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate†	(95% CI)	No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate†	(95% CI)
1981-1984													
Maori	25-44	72	209	224	(153 - 294)	159	191	217	(173 - 262)	42	209	229	(139 - 319)
	45-64	228	1,130	1,140	(946 - 1,333)	315	1,182	1,335	(1,143 - 1,527)	150	1,593	1,594	(1,272 - 1,917)
	65-74	207	4,367	4,494	(3,657 - 5,330)	132	4,320	4,526	(3,465 - 5,588)	135	6,167	6,433	(4,939 - 7,927)
	all age	507	859	1,060	(932 - 1,187)	603	535	1,127	(979 - 1,275)	330	1,035	1,455	(1,237 - 1,674)
Pacific	25-44	36	131	137	(81 - 194)	6	77	87	(9 - 166)	6	229	247	(30 - 465)
	45-64	60	753	787	(538 - 1,036)	15	534	553	(234 - 872)	12	1,064	1,075	(372 - 1,778)
	65-74	24	2,032	1,931	(995 - 2,868)	6	1,051	1,212	(672 - 2,613)	18	5,868	6,063	(2,281 - 9,845)
	all age	120	322	579	(433 - 725)	27	203	384	(176 - 592)	36	733	1,242	(708 - 1,777)
NonM-	25-44	495	85	87	(77 - 96)	321	96	100	(86 - 114)	159	93	96	(78 - 115)
NonP	45-64	2,097	482	440	(418 - 462)	1,728	795	736	(695 - 777)	936	741	645	(596 - 695)
	65-74	3,792	1,908	1,911	(1,839 - 1,982)	1,725	2,979	3,104	(2,930 - 3,277)	1,539	2,833	2,889	(2,720 - 3,058)
	all age	6,384	523	431	(418 - 443)	3,774	619	685	(659 - 712)	2,631	748	626	(597 - 655)
1996-1999													
Maori	25-44	114	175	179	(138 - 220)	195	154	163	(135 - 191)	60	134	134	(92 - 177)
	45-64	315	920	898	(779 - 1,016)	459	1,162	1,349	(1,197 - 1,501)	282	1,315	1,332	(1,148 - 1,516)
	65-74	315	3,326	3,416	(2,960 - 3,871)	213	4,914	5,233	(4,350 - 6,116)	249	5,523	5,624	(4,764 - 6,484)
	all age	741	685	821	(749 - 893)	867	508	1,189	(1,068 - 1,310)	591	847	1,216	(1,091 - 1,341)
Pacific	25-44	60	125	131	(91 - 171)	30	117	125	(67 - 182)	18	241	261	(111 - 411)
	45-64	156	675	696	(564 - 828)	39	603	709	(409 - 1,008)	24	958	988	(499 - 1,478)
	65-74	138	2,886	2,930	(2,330 - 3,530)	24	3,232	3,215	(1,645 - 4,785)	21	3,204	3,179	(1,510 - 4,847)
	all age	357	462	667	(578 - 756)	93	295	703	(483 - 923)	66	592	867	(591 - 1,143)
NonM-	25-44	438	62	60	(54 - 67)	249	81	83	(71 - 95)	165	63	60	(49 - 70)
NonP	45-64	1,656	308	302	(286 - 318)	984	585	625	(582 - 668)	915	427	424	(394 - 454)
	65-74	2,508	1,210	1,201	(1,150 - 1,252)	1,119	2,929	2,979	(2,788 - 3,169)	1,851	2,196	2,186	(2,078 - 2,294)
	all age	4,605	316	283	(274 - 292)	2,352	460	623	(595 - 651)	2,931	525	445	(427 - 462)

Female All-Cause Mortality Rates by Smoking Status NZCMS

*Random Rounded

† Deaths per 100,000 Person-Years

Table 32: Male All-Cause Standardised Rate Ratios by Age and Ethnicity (First Restriction)

	Age Gp	SRR (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)		SRD (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	25-44	1.20	(0.87 - 1.66)	0.89	(0.57 - 1.39)	61	(-44 - 165)	-32	(-157 - 93)
	45-64	1.16	(0.95 - 1.40)	1.07	(0.85 - 1.34)	286	(-94 - 666)	125	(-303 - 552)
	65-74	1.22	(0.93 - 1.60)	1.14	(0.85 - 1.51)	1219	(-441 - 2,878)	749	(-939 - 2,438)
	all age	1.19	(1.02 - 1.39)	1.08	(0.91 - 1.28)	274	(34 - 514)	113	(-142 - 368)
Pacific	25-44	1.18	(0.57 - 2.43)	0.78	(0.17 - 3.63)	26	(-89 - 140)	-33	(-216 - 150)
	45-64	1.15	(0.70 - 1.89)	1.52	(0.86 - 2.70)	140	(-356 - 637)	490	(-219 - 1,198)
	65-74	0.91	(0.44 - 1.85)	2.09	(1.08 - 4.03)	-404	(-3,300 - 2,492)	4617	(298 - 8,936)
	all age	1.02	(0.67 - 1.55)	1.76	(1.14 - 2.74)	17	(-365 - 399)	687	(122 - 1,252)
NonM-NonP	25-44	1.47	(1.29 - 1.69)	1.04	(0.87 - 1.24)	62	(40 - 85)	5	(-19 - 29)
	45-64	1.76	(1.64 - 1.88)	1.21	(1.13 - 1.30)	553	(490 - 616)	153	(97 - 210)
	65-74	1.66	(1.56 - 1.76)	1.40	(1.32 - 1.48)	2047	(1,801 - 2,292)	1234	(1,029 - 1,438)
	all age	1.68	(1.61 - 1.75)	1.29	(1.23 - 1.35)	464	(427 - 502)	199	(165 - 232)
1996-1999									
Maori	25-44	1.28	(1.01 - 1.61)	0.76	(0.53 - 1.08)	82	(4 - 161)	-73	(-161 - 15)
	45-64	1.53	(1.32 - 1.77)	1.10	(0.93 - 1.29)	758	(498 - 1,018)	140	(-103 - 382)
	65-74	1.55	(1.29 - 1.88)	1.17	(0.97 - 1.40)	2736	(1,497 - 3,976)	816	(-149 - 1,781)
	all age	1.51	(1.35 - 1.69)	1.09	(0.97 - 1.22)	627	(452 - 802)	105	(-43 - 253)
Pacific	25-44	1.08	(0.70 - 1.66)	1.15	(0.60 - 2.20)	18	(-84 - 121)	35	(-130 - 199)
	45-64	1.05	(0.80 - 1.37)	0.92	(0.63 - 1.35)	59	(-303 - 420)	-101	(-567 - 364)
	65-74	1.35	(0.90 - 2.04)	2.03	(1.38 - 2.98)	1218	(-512 - 2,949)	3535	(1,365 - 5,705)
	all age	1.18	(0.94 - 1.47)	1.40	(1.09 - 1.80)	171	(-72 - 413)	389	(78 - 701)
NonM-NonP	25-44	1.75	(1.53 - 2.02)	1.16	(0.96 - 1.40)	80	(58 - 101)	17	(-6 - 39)
	45-64	2.31	(2.15 - 2.49)	1.32	(1.23 - 1.42)	555	(499 - 610)	135	(98 - 171)
	65-74	2.27	(2.13 - 2.43)	1.43	(1.36 - 1.52)	2621	(2,381 - 2,862)	892	(760 - 1,024)
	all age	2.22	(2.12 - 2.33)	1.36	(1.30 - 1.42)	540	(504 - 576)	159	(136 - 182)

Male All-Cause SRR & SRD by Smoking Status NZCMS

Table 33: Female All-Cause Standardised Rate Ratios by Age and Ethnicity (First Restriction)

	Age Gp	SRR (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)		SRD (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	25-44	0.97	(0.67 - 1.41)	1.02	(0.62 - 1.69)	-6	(-90 - 77)	6	(-109 - 120)
	45-64	1.17	(0.94 - 1.46)	1.40	(1.07 - 1.82)	196	(-77 - 468)	455	(79 - 831)
	65-74	1.01	(0.75 - 1.36)	1.43	(1.06 - 1.93)	33	(-1,319 - 1,384)	1939	(227 - 3,651)
	all age	1.06	(0.89 - 1.27)	1.37	(1.13 - 1.67)	68	(-128 - 263)	396	(142 - 649)
Pacific	25-44	0.64	(0.24 - 1.71)	1.80	(0.68 - 4.76)	-50	(-147 - 47)	110	(-115 - 335)
	45-64	0.70	(0.36 - 1.36)	1.37	(0.66 - 2.83)	-234	(-638 - 171)	288	(-457 - 1,034)
	65-74	0.63	(0.18 - 2.20)	3.14	(1.42 - 6.92)	-720	(-2,405 - 966)	4132	(236 - 8,029)
	all age	0.66	(0.37 - 1.20)	2.15	(1.30 - 3.53)	-195	(-449 - 59)	664	(109 - 1,218)
NonM-NonP	25-44	1.15	(0.97 - 1.37)	1.11	(0.89 - 1.39)	13	(-4 - 30)	10	(-11 - 31)
	45-64	1.67	(1.55 - 1.80)	1.47	(1.34 - 1.61)	296	(250 - 343)	206	(151 - 260)
	65-74	1.62	(1.52 - 1.74)	1.51	(1.41 - 1.62)	1193	(1,005 - 1,381)	978	(795 - 1,162)
	all age	1.59	(1.52 - 1.67)	1.45	(1.38 - 1.53)	254	(225 - 284)	195	(164 - 227)
1996-1999									
Maori	25-44	0.91	(0.68 - 1.21)	0.75	(0.51 - 1.11)	-16	(-66 - 34)	-45	(-104 - 14)
	45-64	1.50	(1.26 - 1.79)	1.48	(1.23 - 1.80)	451	(259 - 644)	434	(216 - 653)
	65-74	1.53	(1.24 - 1.90)	1.65	(1.34 - 2.02)	1818	(824 - 2,811)	2208	(1,236 - 3,181)
	all age	1.45	(1.27 - 1.66)	1.48	(1.29 - 1.70)	368	(228 - 509)	395	(251 - 539)
Pacific	25-44	0.95	(0.55 - 1.65)	2.00	(1.04 - 3.83)	-6	(-76 - 64)	130	(-25 - 286)
	45-64	1.02	(0.64 - 1.62)	1.42	(0.84 - 2.41)	13	(-314 - 340)	293	(-214 - 800)
	65-74	1.10	(0.65 - 1.86)	1.08	(0.62 - 1.91)	285	(-1,395 - 1,966)	249	(-1,524 - 2,022)
	all age	1.05	(0.75 - 1.48)	1.30	(0.92 - 1.84)	36	(-201 - 273)	200	(-90 - 490)
NonM-NonP	25-44	1.37	(1.15 - 1.64)	0.99	(0.80 - 1.22)	23	(9 - 36)	-1	(-13 - 12)
	45-64	2.07	(1.90 - 2.26)	1.41	(1.29 - 1.53)	323	(278 - 369)	122	(89 - 156)
	65-74	2.48	(2.30 - 2.68)	1.82	(1.71 - 1.94)	1777	(1,581 - 1,974)	985	(865 - 1,105)
	all age	2.20	(2.09 - 2.33)	1.57	(1.50 - 1.66)	340	(311 - 370)	162	(142 - 182)

Female All-Cause SRR & SRD by Smoking Status NZCMS

Table 34: Male IHD Mortality Data by Age and Ethnicity (First Restriction)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate†	(95% CI)	No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate†	(95% CI)	No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate†	(95% CI)
1981-1984													
Maori	25-44	9	23	29	(17 - 58)	36	46	57	(31 - 83)	6	21	23	(13 - 50)
	45-64	87	525	544	(396 - 692)	141	546	589	(462 - 716)	84	654	658	(481 - 834)
	65-74	66	2,136	2,154	(1,481 - 2,826)	66	2,008	2,058	(1,402 - 2,714)	60	2,094	2,116	(1,441 - 2,792)
	all age	162	288	456	(361 - 550)	240	234	476	(387 - 564)	153	388	489	(388 - 590)
Pacific	25-44	--	--	--	--	--	--	--	--	--	--	--	--
	45-64	--	--	--	--	--	--	--	--	--	--	--	--
	65-74	--	--	--	--	--	--	--	--	--	--	--	--
	all age	12	46	97	(18 - 176)	24	91	222	(89 - 356)	24	279	514	(244 - 783)
NonM-NonP	25-44	45	10	12	(8 - 16)	144	36	38	(31 - 46)	51	23	21	(14 - 28)
	45-64	648	277	273	(248 - 297)	1,467	528	497	(467 - 527)	1,140	422	352	(328 - 377)
	65-74	942	1,338	1,338	(1,240 - 1,435)	1,236	1,748	1,771	(1,658 - 1,883)	1,851	1,603	1,599	(1,516 - 1,682)
	all age	1,635	207	257	(242 - 271)	2,844	385	400	(383 - 417)	3,045	504	320	(306 - 333)
1996-1999													
Maori	25-44	24	28	34	(15 - 52)	39	43	47	(28 - 67)	9	31	28	(7 - 49)
	45-64	114	358	362	(283 - 441)	180	533	603	(496 - 711)	138	538	544	(438 - 651)
	65-74	102	1,585	1,599	(1,233 - 1,965)	66	1,761	1,757	(1,248 - 2,267)	90	1,554	1,593	(1,212 - 1,973)
	all age	240	205	330	(279 - 381)	285	215	441	(370 - 511)	243	356	391	(332 - 450)
Pacific	25-44	6	23	26	(5 - 46)	12	41	43	(15 - 72)	6	36	37	(18 - 88)
	45-64	48	330	354	(234 - 475)	51	440	445	(296 - 595)	12	339	346	(130 - 563)
	65-74	27	1,054	1,080	(588 - 1,572)	12	1,021	912	(306 - 1,518)	15	1,361	1,380	(524 - 2,235)
	all age	87	161	263	(192 - 335)	75	185	286	(197 - 374)	30	246	301	(173 - 429)
NonM-NonP	25-44	51	8	8	(5 - 10)	69	21	21	(15 - 27)	18	9	7	(3 - 11)
	45-64	426	105	109	(98 - 121)	555	283	295	(267 - 322)	495	161	146	(132 - 160)
	65-74	690	641	642	(590 - 695)	540	1,213	1,232	(1,119 - 1,346)	1,269	832	813	(764 - 862)
	all age	1,167	99	117	(109 - 124)	1,167	205	258	(241 - 274)	1,785	261	149	(141 - 157)

Male IHD Mortality Rates by Smoking Status NZCMS

*Random Rounded

† Deaths per 100,000 Person-Years

Table 35: Female IHD Mortality Data by Age and Ethnicity (First Restriction)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)	
1981-1984													
Maori	25-44	--	--	--	--	--	--	--	--	--	--	--	--
	45-64	42	211	218	(136 - 301)	57	214	243	(163 - 323)	33	341	343	(196 - 489)
	65-74	72	1,489	1,543	(1,077 - 2,009)	39	1,288	1,439	(832 - 2,045)	33	1,496	1,505	(854 - 2,156)
	all age	120	202	273	(209 - 338)	105	92	267	(187 - 346)	66	206	301	(207 - 396)
Pacific	25-44	--	--	--	--	--	--	--	--	--	--	--	--
	45-64	--	--	--	--	--	--	--	--	--	--	--	--
	65-74	--	--	--	--	--	--	--	--	--	--	--	--
	all age	9	26	47	(8 - 86)	9	69	160	(22 - 299)	6	91	185	(103 - 399)
NonM-	25-44	9	1	2	(0 - 3)	27	8	9	(4 - 13)	18	9	10	(4 - 17)
NonP	45-64	330	76	64	(56 - 72)	462	212	195	(175 - 216)	138	110	91	(73 - 109)
	65-74	1,257	631	632	(592 - 673)	582	1,001	1,057	(956 - 1,157)	471	869	887	(794 - 979)
	all age	1,590	130	100	(94 - 106)	1,068	175	201	(186 - 215)	627	178	145	(132 - 158)
1996-1999													
Maori	25-44	6	5	5	(2 - 11)	12	11	13	(5 - 21)	6	9	9	(5 - 19)
	45-64	39	116	111	(69 - 152)	75	187	232	(167 - 297)	39	178	180	(112 - 248)
	65-74	78	837	856	(639 - 1,073)	54	1,274	1,216	(830 - 1,602)	48	1,045	1,107	(728 - 1,485)
	all age	120	113	145	(115 - 176)	144	84	235	(183 - 288)	90	128	202	(150 - 254)
Pacific	25-44	6	11	12	(0 - 24)	6	12	17	(8 - 40)	6	16	14	(5 - 43)
	45-64	24	95	99	(50 - 147)	6	51	42	(21 - 100)	6	127	126	(62 - 302)
	65-74	21	398	407	(199 - 615)	9	881	821	(78 - 1,563)	6	574	550	(309 - 1,172)
	all age	45	61	90	(59 - 121)	12	42	124	(30 - 218)	9	77	118	(20 - 216)
NonM-	25-44	6	1	0.4	(0 - 1)	12	4	5	(2 - 7)	6	2	2	(0 - 4)
NonP	45-64	129	24	23	(19 - 28)	126	75	81	(66 - 97)	93	43	42	(33 - 52)
	65-74	474	229	226	(204 - 248)	237	616	628	(541 - 715)	336	397	394	(348 - 440)
	all age	606	42	36	(33 - 39)	375	73	107	(95 - 119)	432	77	64	(57 - 70)

Female IHD Mortality Rates by Smoking Status NZCMS

*Random Rounded

† Deaths per 100,000 Person-Years

Table 36: Male IHD Standardised Rate Ratios by Age and Ethnicity (First Restriction)

	Age Gp	SRR (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)		SRD (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	25-44	1.96	(0.65 - 5.93)	0.81	(0.18 - 3.69)	28	(-11 - 67)	-6	(-45 - 34)
	45-64	1.08	(0.77 - 1.53)	1.21	(0.83 - 1.77)	45	(-150 - 240)	114	(-117 - 345)
	65-74	0.96	(0.61 - 1.49)	0.98	(0.63 - 1.54)	-95	(-1,035 - 844)	-37	(-990 - 916)
	all age	1.04	(0.79 - 1.38)	1.07	(0.80 - 1.44)	20	(-110 - 150)	33	(-105 - 171)
Pacific	25-44	--	--	--	--	--	--	--	--
	45-64	--	--	--	--	--	--	--	--
	65-74	--	--	--	--	--	--	--	--
	all age	2.29	(0.84 - 6.29)	5.29	(2.01 - 13.91)	125	(-30 - 280)	417	(136 - 698)
NonM-NonP	25-44	3.31	(2.21 - 4.95)	1.79	(1.10 - 2.90)	27	(18 - 36)	9	(1 - 17)
	45-64	1.82	(1.64 - 2.03)	1.29	(1.15 - 1.45)	225	(186 - 263)	80	(45 - 114)
	65-74	1.32	(1.20 - 1.46)	1.20	(1.09 - 1.31)	433	(284 - 581)	261	(134 - 389)
	all age	1.56	(1.45 - 1.67)	1.25	(1.16 - 1.34)	144	(121 - 166)	63	(44 - 83)
1996-1999									
Maori	25-44	1.41	(0.71 - 2.79)	0.83	(0.33 - 2.10)	14	(-13 - 40)	-6	(-34 - 22)
	45-64	1.67	(1.26 - 2.21)	1.50	(1.12 - 2.02)	241	(108 - 375)	182	(50 - 315)
	65-74	1.10	(0.76 - 1.59)	1.00	(0.72 - 1.39)	159	(-468 - 786)	-6	(-534 - 522)
	all age	1.34	(1.07 - 1.67)	1.18	(0.95 - 1.47)	111	(24 - 198)	61	(-17 - 139)
Pacific	25-44	1.70	(0.60 - 4.78)	1.45	(0.29 - 7.20)	18	(-17 - 53)	12	(-44 - 67)
	45-64	1.26	(0.78 - 2.03)	0.98	(0.48 - 1.99)	91	(-102 - 283)	-8	(-256 - 240)
	65-74	0.84	(0.38 - 1.89)	1.28	(0.59 - 2.76)	-168	(-948 - 612)	300	(-687 - 1,286)
	all age	1.08	(0.72 - 1.64)	1.14	(0.69 - 1.89)	22	(-92 - 136)	38	(-109 - 184)
NonM-NonP	25-44	2.79	(1.81 - 4.29)	0.91	(0.48 - 1.70)	14	(7 - 20)	-1	(-5 - 4)
	45-64	2.69	(2.34 - 3.10)	1.33	(1.15 - 1.54)	185	(156 - 215)	37	(18 - 55)
	65-74	1.92	(1.70 - 2.17)	1.26	(1.14 - 1.40)	590	(465 - 715)	170	(99 - 242)
	all age	2.21	(2.02 - 2.42)	1.28	(1.17 - 1.39)	141	(123 - 159)	32	(21 - 43)

Male IHD SRR & SRD by Smoking Status NZCMS

Table 37: Female IHD Standardised Rate Ratios by Age and Ethnicity (First Restriction)

	Age Gp	SRR (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)		SRD (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	25-44	--	--	--	--	--	--	--	--
	45-64	1.11	(0.67 - 1.84)	1.57	(0.89 - 2.78)	25	(-90 - 140)	124	(-44 - 293)
	65-74	0.93	(0.56 - 1.57)	0.98	(0.58 - 1.65)	-104	(-869 - 661)	-38	(-839 - 763)
	all age	0.98	(0.67 - 1.43)	1.10	(0.75 - 1.63)	-7	(-109 - 96)	28	(-86 - 142)
Pacific	25-44	--	--	--	--	--	--	--	--
	45-64	--	--	--	--	--	--	--	--
	65-74	--	--	--	--	--	--	--	--
	all age	3.40	(1.03 - 11.23)	3.92	(0.95 - 16.20)	113	(-31 - 257)	138	(-79 - 355)
NonM-NonP	25-44	5.87	(2.13 - 16.18)	6.81	(2.28 - 20.36)	7	(3 - 12)	9	(2 - 15)
	45-64	3.07	(2.60 - 3.62)	1.43	(1.12 - 1.81)	132	(109 - 154)	27	(7 - 47)
	65-74	1.67	(1.49 - 1.87)	1.40	(1.24 - 1.58)	425	(316 - 533)	254	(154 - 355)
	all age	2.01	(1.83 - 2.20)	1.45	(1.30 - 1.62)	101	(85 - 116)	45	(31 - 60)
1996-1999									
Maori	25-44	2.88	(0.63 - 13.09)	1.92	(0.32 - 11.56)	9	(-2 - 19)	4	(-8 - 16)
	45-64	2.10	(1.31 - 3.36)	1.63	(0.96 - 2.78)	121	(44 - 199)	70	(-10 - 150)
	65-74	1.42	(0.95 - 2.13)	1.29	(0.84 - 1.98)	360	(-83 - 804)	251	(-186 - 688)
	all age	1.62	(1.20 - 2.20)	1.39	(1.00 - 1.94)	90	(30 - 151)	57	(-4 - 117)
Pacific	25-44	1.41	(0.26 - 7.69)	1.20	(0.13 - 10.79)	5	(-21 - 31)	2	(-28 - 33)
	45-64	0.42	(0.10 - 1.85)	1.28	(0.29 - 5.61)	-57	(-132 - 19)	28	(-155 - 210)
	65-74	2.02	(0.71 - 5.71)	1.35	(0.39 - 4.68)	414	(-357 - 1,185)	143	(-513 - 800)
	all age	1.38	(0.60 - 3.17)	1.31	(0.53 - 3.23)	34	(-65 - 133)	28	(-75 - 131)
NonM-NonP	25-44	10.00	(2.74 - 36.51)	4.61	(1.09 - 19.39)	4	(1 - 7)	2	(0 - 4)
	45-64	3.53	(2.68 - 4.64)	1.83	(1.36 - 2.47)	58	(42 - 75)	19	(9 - 30)
	65-74	2.78	(2.35 - 3.30)	1.75	(1.50 - 2.03)	402	(313 - 492)	169	(118 - 219)
	all age	3.00	(2.60 - 3.45)	1.79	(1.56 - 2.04)	71	(59 - 84)	28	(21 - 35)

Female IHD SRR & SRD by Smoking Status NZCMS

Table 38: Male Stroke Mortality Data by Age and Ethnicity (First Restriction)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)	
1981-1984													
Maori	25-44	6	11	12	(6 - 29)	12	19	24	(7 - 41)	6	8	9	(3 - 26)
	45-64	12	84	97	(28 - 165)	21	87	98	(44 - 152)	15	103	105	(25 - 185)
	65-74	18	539	550	(190 - 910)	6	148	131	(74 - 279)	12	392	384	(99 - 668)
	all age	33	62	104	(55 - 153)	42	40	63	(35 - 90)	27	69	86	(42 - 130)
Pacific	25-44	--	--	--	--	--	--	--	--	--	--	--	--
	45-64	--	--	--	--	--	--	--	--	--	--	--	--
	65-74	--	--	--	--	--	--	--	--	--	--	--	--
	all age	15	55	137	(28 - 245)	9	40	116	(17 - 215)	6	17	25	(9 - 74)
NonM-	25-44	12	2	3	(1 - 5)	30	7	8	(4 - 11)	9	4	4	(1 - 8)
NonP	45-64	84	36	35	(26 - 45)	225	82	74	(62 - 86)	123	46	40	(31 - 49)
	65-74	240	344	344	(293 - 395)	345	490	500	(437 - 562)	411	355	354	(314 - 395)
	all age	339	43	54	(47 - 60)	603	82	88	(79 - 97)	543	90	57	(51 - 63)
1996-1999													
Maori	25-44	9	10	10	(6 - 20)	6	7	7	(0 - 15)	6	4	3	(1 - 9)
	45-64	27	81	82	(43 - 120)	21	56	71	(32 - 111)	9	37	37	(7 - 67)
	65-74	18	283	316	(144 - 489)	15	384	375	(139 - 611)	9	143	137	(27 - 248)
	all age	54	44	71	(46 - 95)	39	30	72	(42 - 103)	21	28	31	(14 - 47)
Pacific	25-44	6	3	4	(1 - 11)	6	5	5	(2 - 15)	6	19	17	(6 - 51)
	45-64	18	107	127	(51 - 202)	9	60	62	(6 - 119)	6	34	34	(13 - 102)
	65-74	6	242	262	(4 - 521)	6	116	98	(36 - 289)	6	411	403	(227 - 860)
	all age	24	44	77	(37 - 117)	12	24	36	(6 - 66)	6	56	68	(7 - 128)
NonM-	25-44	9	2	2	(1 - 3)	12	3	3	(1 - 6)	9	3	2	(0 - 5)
NonP	45-64	72	17	18	(14 - 23)	75	39	40	(30 - 51)	51	17	15	(10 - 19)
	65-74	144	136	137	(112 - 162)	135	300	311	(252 - 370)	246	162	158	(136 - 181)
	all age	228	19	23	(20 - 27)	219	39	52	(44 - 60)	303	45	25	(22 - 28)

Male Stroke Mortality Rates by Smoking Status NZCMS

*Random Rounded

† Deaths per 100,000 Person-Years

Table 39: Female Stroke Mortality Data by Age and Ethnicity (First Restriction)

	Age Gp	Never-Smoked				Smoker				Ex-Smoker			
		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)		No. Wgt Deaths*	Non-Std M Rate†	Std Mort Rate† (95% CI)	
1981-1984													
Maori	25-44	6	16	17	(9 - 36)	15	19	23	(8 - 38)	6	17	21	(10 - 50)
	45-64	21	98	102	(38 - 166)	33	126	148	(80 - 217)	12	102	102	(1 - 203)
	65-74	27	511	543	(265 - 821)	12	408	431	(98 - 764)	18	868	916	(385 - 1,447)
	all age	48	84	110	(68 - 152)	63	55	116	(68 - 164)	33	101	158	(83 - 234)
Pacific	25-44	--	--	--	--	--	--	--	--	--	--	--	--
	45-64	--	--	--	--	--	--	--	--	--	--	--	--
	65-74	--	--	--	--	--	--	--	--	--	--	--	--
	all age	9	24	57	(9 - 105)	6	10	13	(5 - 39)	6	96	206	(108 - 464)
NonM-	25-44	21	3	4	(2 - 6)	39	11	12	(7 - 18)	12	6	6	(1 - 12)
NonP	45-64	138	32	28	(22 - 33)	171	79	73	(60 - 86)	48	37	29	(19 - 40)
	65-74	498	250	251	(224 - 277)	198	339	361	(299 - 422)	183	337	350	(289 - 411)
	all age	657	54	42	(38 - 46)	408	67	76	(67 - 85)	240	68	56	(48 - 65)
1996-1999													
Maori	25-44	--	--	--	--	--	--	--	--	--	--	--	--
	45-64	24	74	73	(41 - 106)	21	51	58	(28 - 88)	18	82	83	(40 - 127)
	65-74	18	177	184	(73 - 295)	15	374	484	(157 - 811)	15	346	345	(117 - 573)
	all age	45	42	50	(32 - 68)	39	25	82	(40 - 123)	33	48	71	(39 - 103)
Pacific	25-44	--	--	--	--	--	--	--	--	--	--	--	--
	45-64	6	36	36	(7 - 64)	6	68	66	(36 - 142)	6	110	107	(52 - 255)
	65-74	21	455	470	(230 - 709)	6	158	132	(49 - 390)	6	195	204	(75 - 605)
	all age	33	41	71	(40 - 103)	9	26	46	(4 - 88)	6	38	61	(34 - 132)
NonM-	25-44	9	1	1	(0 - 2)	24	9	9	(5 - 13)	6	1	1	(1 - 3)
NonP	45-64	51	9	9	(6 - 12)	84	50	54	(41 - 66)	33	16	16	(10 - 21)
	65-74	210	101	100	(85 - 115)	75	198	203	(152 - 253)	123	144	143	(115 - 171)
	all age	267	18	16	(14 - 18)	186	36	48	(40 - 56)	159	28	23	(19 - 27)

Female Stroke Mortality Rates by Smoking Status NZCMS

*Random Rounded

† Deaths per 100,000 Person-Years

Table 40: Male Stroke Standardised Rate Ratios by Age and Ethnicity (First Restriction)

	Age Gp	SRR (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)		SRD (reference gp never smoked) Smoker (95% CI)		Ex-Smoker (95% CI)	
1981-1984									
Maori	25-44	2.02	(0.43 - 9.52)	0.73	(0.07 - 8.04)	12	(-12 - 36)	-3	(-27 - 21)
	45-64	1.01	(0.41 - 2.48)	1.09	(0.39 - 3.07)	1	(-86 - 88)	9	(-97 - 114)
	65-74	0.24	(0.06 - 0.88)	0.70	(0.26 - 1.88)	-419	(-808 - -29)	-166	(-625 - 293)
	all age	0.60	(0.32 - 1.14)	0.83	(0.41 - 1.66)	-41	(-97 - 15)	-18	(-84 - 48)
Pacific	25-44	--	--	--	--	--	--	--	--
	45-64	--	--	--	--	--	--	--	--
	65-74	--	--	--	--	--	--	--	--
	all age	0.85	(0.26 - 2.72)	0.18	(0.02 - 1.51)	-21	(-168 - 126)	-112	(-231 - 7)
NonM-NonP	25-44	2.91	(1.20 - 7.03)	1.63	(0.51 - 5.19)	5	(1 - 9)	2	(-3 - 6)
	45-64	2.10	(1.55 - 2.85)	1.12	(0.79 - 1.57)	39	(24 - 54)	4	(-9 - 17)
	65-74	1.45	(1.20 - 1.76)	1.03	(0.85 - 1.24)	155	(75 - 236)	10	(-55 - 75)
	all age	1.64	(1.40 - 1.93)	1.07	(0.90 - 1.26)	34	(23 - 45)	4	(-6 - 13)
1996-1999									
Maori	25-44	0.74	(0.18 - 2.99)	0.31	(0.03 - 2.78)	-3	(-15 - 10)	-7	(-19 - 5)
	45-64	0.87	(0.42 - 1.81)	0.45	(0.18 - 1.15)	-10	(-66 - 45)	-45	(-94 - 4)
	65-74	1.18	(0.52 - 2.73)	0.43	(0.16 - 1.14)	59	(-234 - 351)	-179	(-384 - 26)
	all age	1.02	(0.59 - 1.78)	0.43	(0.23 - 0.83)	2	(-38 - 41)	-40	(-70 - -10)
Pacific	25-44	1.36	(0.09 - 21.73)	4.54	(0.28 - 72.52)	1	(-11 - 14)	13	(-21 - 48)
	45-64	0.49	(0.17 - 1.46)	0.27	(0.03 - 2.10)	-64	(-159 - 30)	-93	(-194 - 9)
	65-74	0.37	(0.04 - 3.33)	1.54	(0.34 - 6.89)	-165	(-486 - 157)	141	(-384 - 666)
	all age	0.47	(0.17 - 1.26)	0.88	(0.31 - 2.47)	-41	(-91 - 9)	-9	(-82 - 63)
NonM-NonP	25-44	1.81	(0.63 - 5.22)	1.37	(0.45 - 4.19)	1	(-1 - 4)	1	(-2 - 3)
	45-64	2.19	(1.52 - 3.16)	0.80	(0.54 - 1.20)	22	(11 - 33)	-4	(-10 - 3)
	65-74	2.28	(1.75 - 2.96)	1.16	(0.92 - 1.46)	175	(111 - 239)	22	(-12 - 55)
	all age	2.23	(1.81 - 2.76)	1.07	(0.88 - 1.30)	29	(20 - 37)	2	(-3 - 6)

Male Stroke SRR & SRD by Smoking Status NZCMS

Table 41: Female Stroke Standardised Rate Ratios by Age and Ethnicity (First Restriction)

	Age Gp	SRR (reference gp never smoked) Smoker (95% CI)		SRR (reference gp never smoked) Ex-Smoker (95% CI)		SRD (reference gp never smoked) Smoker (95% CI)		SRD (reference gp never smoked) Ex-Smoker (95% CI)	
1981-1984									
Maori	25-44	1.41	(0.37 - 5.30)	1.27	(0.21 - 7.70)	7	(-18 - 31)	4	(-30 - 39)
	45-64	1.46	(0.67 - 3.18)	1.01	(0.31 - 3.24)	46	(-47 - 140)	1	(-119 - 120)
	65-74	0.79	(0.31 - 2.00)	1.69	(0.78 - 3.65)	-112	(-546 - 321)	373	(-227 - 972)
	all age	1.05	(0.60 - 1.84)	1.44	(0.78 - 2.64)	6	(-58 - 69)	48	(-38 - 134)
Pacific	25-44	--	--	--	--	--	--	--	--
	45-64	--	--	--	--	--	--	--	--
	65-74	--	--	--	--	--	--	--	--
	all age	0.23	(0.03 - 1.98)	3.62	(0.80 - 16.49)	-44	(-98 - 11)	149	(-114 - 412)
NonM-NonP	25-44	3.54	(1.74 - 7.18)	1.82	(0.68 - 4.90)	9	(3 - 14)	-7	(-21 - 7)
	45-64	2.65	(2.02 - 3.48)	1.07	(0.72 - 1.60)	45	(31 - 60)	62	(-160 - 284)
	65-74	1.44	(1.18 - 1.76)	1.40	(1.14 - 1.71)	110	(43 - 177)	1074	(-982 - 3,130)
	all age	1.80	(1.55 - 2.10)	1.34	(1.12 - 1.61)	34	(24 - 44)	14	(5 - 24)
1996-1999									
Maori	25-44	--	--	--	--	--	--	--	--
	45-64	0.79	(0.40 - 1.55)	1.14	(0.57 - 2.25)	-16	(-59 - 28)	10	(-44 - 64)
	65-74	2.63	(1.07 - 6.51)	1.88	(0.77 - 4.58)	300	(-45 - 646)	161	(-92 - 415)
	all age	1.62	(0.87 - 3.01)	1.41	(0.79 - 2.49)	31	(-14 - 76)	20	(-16 - 57)
Pacific	25-44	--	--	--	--	--	--	--	--
	45-64	1.84	(0.45 - 7.58)	3.00	(0.60 - 14.94)	30	(-51 - 111)	71	(-80 - 222)
	65-74	0.28	(0.04 - 2.13)	0.44	(0.06 - 3.30)	-338	(-690 - 14)	-265	(-732 - 202)
	all age	0.64	(0.23 - 1.78)	0.86	(0.25 - 2.94)	-26	(-78 - 27)	-10	(-87 - 67)
NonM-NonP	25-44	8.94	(3.59 - 22.31)	1.05	(0.21 - 5.36)	8	(4 - 12)	0	(-2 - 2)
	45-64	5.90	(4.01 - 8.69)	1.72	(1.07 - 2.77)	44	(31 - 58)	7	(0 - 13)
	65-74	2.02	(1.51 - 2.71)	1.43	(1.12 - 1.83)	102	(49 - 156)	43	(11 - 75)
	all age	3.01	(2.44 - 3.72)	1.47	(1.19 - 1.83)	32	(24 - 40)	8	(3 - 12)

Female Stroke SRR & SRD by Smoking Status NZCMS

Appendix C: Person-time data

Table 42: Person-time for 25-74 year olds in the first restricted (R1) and second restricted (R2) cohorts

	Age Gp	Person-Time (number of person-years)											
		MALE						FEMALE					
		Never-Smoked		Smoker		Ex-Smoker		Never-Smoked		Smoker		Ex-Smoker	
		Person Time R1	Person Time R2	Person Time R1	Person Time R2	Person Time R1	Person Time R2	Person Time R1	Person Time R2	Person Time R1	Person Time R2	Person Time R1	Person Time R2
1981-1984													
Maori	all age	55,675	35,040	103,168	59,706	39,042	25,254	59,170	38,235	112,784	68,268	31,762	20,745
Pacific	all age	23,781	13,320	26,636	14,355	8,263	4,812	37,715	22,089	14,092	8,193	5,140	3,093
NonM-NonP	all age	789,595	604,863	738,693	536,280	604,127	465,144	1,219,952	942,759	610,128	454,083	351,644	271,185
All Ethnicity	all age	869,050	653,229	868,497	610,344	651,432	495,207	1,316,838	1,003,080	737,004	530,547	388,546	295,026
1996-1999													
Maori	all age	117,139	83,052	133,292	88,050	68,068	49,830	107,909	76,521	170,428	115,848	69,730	51,312
Pacific	all age	53,643	31,515	41,592	24,279	13,004	8,889	76,878	44,283	31,123	19,329	10,801	7,452
NonM-NonP	all age	1,179,378	964,638	568,050	451,689	684,071	569,553	1,458,070	1,196,736	511,620	417,810	558,900	469,047
All Ethnicity	all age	1,350,159	1,079,205	742,933	564,015	765,143	628,272	1,642,858	1,317,540	713,171	552,987	639,431	527,814

r1 vs r2 person-time NZCMS n

Table 43: Person-time for 25-44 year olds, 45-64 year olds, and 65-74 year olds in the first restricted cohort

		Person-Time (number of person-years)					
		MALE			FEMALE		
	Age Gp	Never-Smoked	Current Smoker	Ex-Smoker	Never-Smoked	Current Smoker	Ex-Smoker
1981-1984							
Maori	25-44	36,165	73,941	23,166	34,226	83,155	20,079
	45-64	16,407	25,971	12,894	20,153	26,626	9,482
	65-74	3,103	3,256	2,982	4,792	3,002	2,201
Pacific	25-44	18,227	19,944	5,771	28,644	10,648	3,641
	45-64	4,913	6,066	2,096	7,836	3,060	1,219
	65-74	640	627	396	1,236	385	279
NonMaori	25-44	486,244	390,107	217,940	586,382	334,748	171,240
NonPacific	45-64	233,011	277,762	270,583	434,745	217,428	126,149
	65-74	70,340	70,825	115,604	198,826	57,952	54,255
All Ethnicity	25-44	540,636	483,991	246,877	649,251	428,551	194,961
	45-64	254,331	309,798	285,573	462,733	247,114	136,849
	65-74	74,083	74,708	118,982	204,854	61,339	56,736
1996-1999							
Maori	25-44	78,471	95,861	36,134	64,514	126,519	43,700
	45-64	32,188	33,690	26,141	33,938	39,622	21,521
	65-74	6,479	3,741	5,793	9,457	4,287	4,510
Pacific	25-44	35,878	28,536	7,596	48,925	24,019	7,599
	45-64	15,087	11,696	4,340	23,181	6,310	2,534
	65-74	2,677	1,360	1,069	4,773	794	667
NonMaori	25-44	664,843	327,060	222,516	712,648	305,033	259,958
NonPacific	45-64	406,942	196,426	309,082	538,078	168,376	214,695
	65-74	107,592	44,564	152,472	207,344	38,211	84,248
All Ethnicity	25-44	779,193	451,456	266,246	826,087	455,572	311,257
	45-64	454,218	241,812	339,563	595,197	214,308	238,750
	65-74	116,748	49,665	159,334	221,574	43,292	89,425

r1 person-time NZCMS