COUNTIES MANUKAU DISTRICT HEALTH BOARD
A Community Partnership

Life expectancy in 2005: Contribution to life expectancy gaps of major disease areas in CMDHB

December 2008

James Smith
Gary Jackson
Siniva Sinclair
Counties Manukau District Health Board

This report is available in PDF format on
www.cmdhb.org.nz

Published in December 2008
By Counties Manukau District Health Board
Private Bag 94052
South Auckland Mail Centre
Manukau City
New Zealand

ISBN 978-0-9582961-3-7

Key points

- Differences in life expectancy between Maaori, Pacific peoples and those of non-Maaori, non-Pacific ethnicities have been consistent and longstanding in Counties Manukau. Each year over the last decade, Maaori life expectancy at birth has been around ten years less than non-Maaori, non-Pacific, while Pacific life expectancy has been around 5-7 years less than non-Maaori, non-Pacific

- Life expectancy at birth is a useful way to compare populations as it is an easily understandable summary measure, is reflective of current mortality across different age groups and allows comparison of groups with different population structures. In this study, abridged life tables were created for Maaori, Pacific and non-Maaori, non-Pacific living in Counties Manukau in 2005. Mortality data for 2005 were disassociated and ranked according to magnitude of absolute mortality numbers and according to inequity in mortality by ethnicity. Mortality data were then aggregated into five 'disease areas'. 'Expected' mortality numbers for each disease area were substituted into life tables for Maaori and Pacific populations to estimate the contribution of each of these areas to gaps in life expectancy at birth between Maaori, Pacific and non-Maaori, non-Pacific

- In 2005, a gap of 10.3 years was identified between Maaori and non-Maaori, non-Pacific, while a gap of 5.6 years was identified between Pacific and non-Maaori, non-Pacific. The major disease areas contributing to these gaps and their proportional contribution to the total gaps are summarised in the table below. Lung diseases related to smoking, cardiovascular diseases, (non-lung) cancer, diabetes and infant mortality were found to be the main causes of death that contributed to inequity in life expectancy between Maaori, Pacific and non-Maaori, non-Pacific

Table: Major disease areas contributing to life expectancy gaps in CMDHB in 2005 and proportional contribution of each area

Cause of death (disease area)	Maaori	Pacific
Infant mortality	5%	10%
Cardiovascular disease	16%	35%
Smoking-related lung disease	20%	9%
Cancer (non-lung)	17%	6%
Diabetes	7%	13%
Remainder	37%	27%

- Smoking was found to be the 'big ticket item', contributing not only to smoking-related lung diseases such as lung cancer, but to all five disease areas identified. The association between smoking prevalence and disparity in life expectancy was therefore examined in greater detail in models which used counterfactual smoking prevalence estimates to predict life expectancy gaps:
    - Differences in smoking frequency accounted for at least 10-20% of the life expectancy gap between Maaori and non-Maaori, non-Pacific in CMDHB

    - Differences in smoking frequency between Maaori and non-Maaori, non-Pacific in 2005 were responsible for at least 2-15% of the life expectancy gap for Pacific

- In order to reduce life expectancy gaps in Counties Manukau, this report gives high priority to interventions aimed at reducing the prevalence of tobacco smoking in Maaori and Pacific populations. Such interventions must recognise that:

    - Considerable reductions in smoking prevalence are necessary to demonstrate significant changes in life expectancy. For example, reducing smoking prevalence by seven percentage points in Maaori (from the current prevalence of 46.8% to 40.0% - a 15% shift in smoking prevalence) would translate to about a 3-5 month narrowing in the life expectancy gap
    - Initiatives directed at reducing the burden of smoking will not affect life expectancy immediately. It is unlikely that any effect of such interventions on life expectancy would be seen before five years

- This report supports the implementation and continuation of other programmes aimed at primary (before cardiovascular disease/diabetes has been diagnosed) and secondary (reducing the impact of cardiovascular disease/diabetes after diagnosis) prevention of cardiovascular disease and diabetes in Counties Manukau

- This report recommends further work examining the drivers behind cancer mortality differences by ethnicity observed in Counties Manukau

- This report recommends additional research to tease out the key drivers of infant mortality in Maaori and Pacific communities in CMDHB. However, initiatives to reduce mortality from SIDS in Maaori and Pacific populations appear likely to have an important influence on reducing infant mortality disparities

- Life expectancy appears to be a useful measure for target setting and strategic monitoring in CMDHB

Table of contents

List of tables

List of figures

List of abbreviations

BDM	Births, Deaths and Marriages
CCM	Chronic Care Management (Programme)
CMDHB	Counties Manukau District Health Board
COPD	Chronic obstructive pulmonary disease
CVD	Cardiovascular disease
DHB	District health board
GBD	Global Burden of Disease
ICD-10-AM	International Classification of Diseases, 10th edition, Australian Modification
IHD	Ischaemic heart disease
LE	Life expectancy
MELAA	Middle Eastern, Latin American and African ethnicities
MOH ID	Ministry of Health Information Directorate
NHI	National Health Index
NMDS	National Minimum Data Set
NZCMS	New Zealand Census-Mortality Study
NZHS	New Zealand Health Survey
PAR%	Population attributable risk percent
PVD	Peripheral vascular disease
SAMMEC	Smoking-attributable mortality, morbidity and economic costs
SIDS	Sudden infant death syndrome
SIR	Smoking impact ratio
SRD	Standardised rate difference
SUDI	Sudden unexpected death in infancy
US	United States
WHO	World Health Organization

Background

This report describes the gap in life expectancy between those of Maaori and Pacific ethnicities and non-Maaori, non-Pacific people in Counties Manukau District Health Board (CMDHB). The category non-Maaori, non-Pacific includes all people who do not identify with Maaori or Pacific ethnicities, such as those of NZ European ethnicity.

The report used mortality data from 2005 to create life tables for those of Maaori, Pacific and non-Maaori, non-Pacific ethnicities in CMDHB. These life tables were then incorporated into two models, which described gaps in life expectancy in 2005. By stratifying the mortality data by cause of death, the contribution of various disease groupings to the overall gap in life expectancy could then be examined. The specific disease groupings examined in the analysis were:

- Mortality in the first year of life
- Cardiovascular disease (CVD), including ischaemic heart disease (IHD), peripheral vascular disease (PVD) and cerebrovascular disease
- Smoking-related lung disease (a broad category that included lung cancer and chronic obstructive pulmonary disease [COPD])
- Malignancy (all cancers except lung cancer, which was addressed under smoking-related lung disease)
- Diabetes

Aims and objectives

The aim of this project was to create interactive, evidence-based models to estimate the impact of specific disease areas on the life expectancy gap between those of Maaori and Pacific ethnicities and non-Maaori, non-Pacific people within CMDHB and identify areas with the greatest potential for population-level interventions.

This aim was addressed through the following objectives:

- To create a tool that assists with planning of health promotion and specific population-focused disease interventions in CMDHB
- To create a tool that contributes towards the reduction of inequity in health outcomes in CMDHB
- To identify and prioritise areas of health need that are most amenable to population health-based intervention
- Identify and highlight potential areas for future research
- Determine feasibility of including specific life expectancy targets in CMDHB strategic plan monitoring

Life expectancy

Life expectancy is often used as a summary measure of health status within populations. It can be estimated at any age, but is usually used in the context of life expectancy at birth. Life expectancy is defined as the average number of years that an individual of a given age is expected to live, if current mortality rates remain unchanged [1-3]. It is therefore a hypothetical measure, which does not indicate how long an individual is actually likely to live, as it does not account for future changes in the incidence and treatment of diseases [1, 3]. Rather, it is a reflection of current mortality across all age groups.

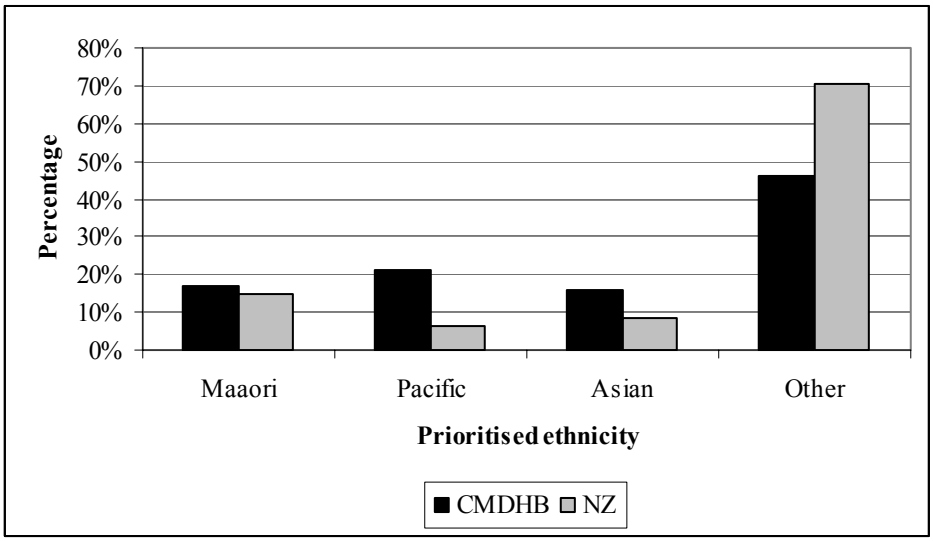
An important advantage of life expectancy is its utility in comparing populations with different age structures, across different time periods [4, 5]. As Griffiths and Fitzpatrick note, "…it is a summary measure of mortality at every age that allows comparisons to be made between areas and time periods without the need to assume a particular standard population" [4]. These features make life expectancy a useful tool in the examination of health inequities between ethnicities in CMDHB, as the three groups have differing population structures. A further reason to use life expectancy at birth to analyse health inequity in CMDHB is that it is easily recognised and understood by those without backgrounds in epidemiology and health care.

Life expectancy is currently used in CMDHB to measure progress in reducing health disparities between those of Maaori and Pacific ethnicities and those of Other (non-Maaori, non-Pacific) ethnicity in important organisational documents such as the Statement of Intent 2007/08 and District Strategic Plan 2006-2011 [6].

The CMDHB population

CMDHB contains a diverse population with a complex range of health needs and service requirements [7]. The population is the fastest-growing of any district health board (DHB) in New Zealand, and is characterised by: A high proportion of Maaori, a high proportion of Pacific peoples and a high proportion of Asian peoples compared with other DHB's [7]. CMDHB is also notable for the relative youthfulness and for the relatively high level of socioeconomic deprivation experienced by its population.

Figure 1: Proportion of people of each (prioritised) ethnicity in CMDHB and New Zealand in 2006



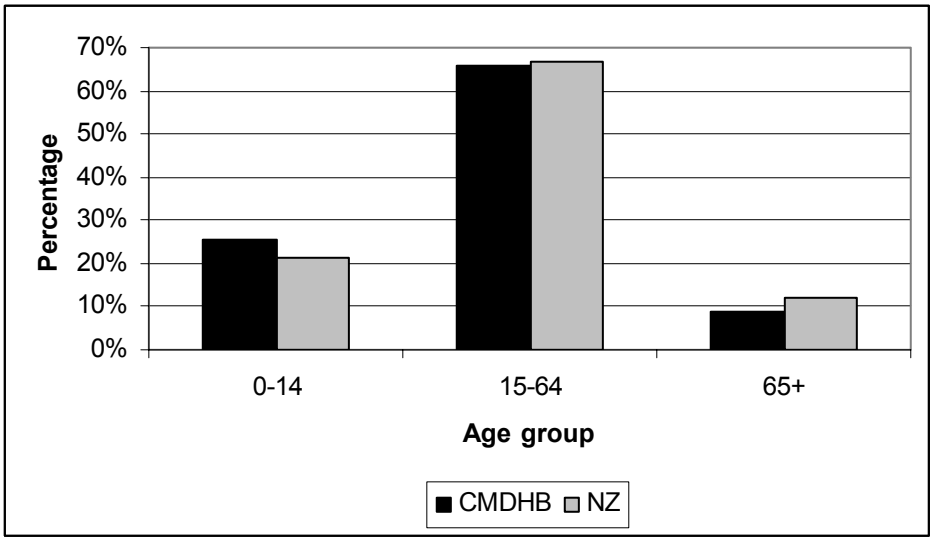
Data source: NZ Census (2006)

CMDHB covers an area of close to 3,000 sq km, which includes the territorial authorities of Manukau City, Papakura District and Franklin District. In 2007, the population of CMDHB was estimated to be 464,000, around 11% of the total New Zealand population of 4.23 million people [7].

Figure 1 describes the proportion of people of each (prioritised) ethnicity in CMDHB compared with the New Zealand population in 2006, while Figure 2 describes the

proportion of people aged less than 15 years or 65 years or more in CMDHB compared with the New Zealand population overall in 2006.

Figure 2: Proportion of population aged <15 and ≥65 years in CMDHB and New Zealand in 2006

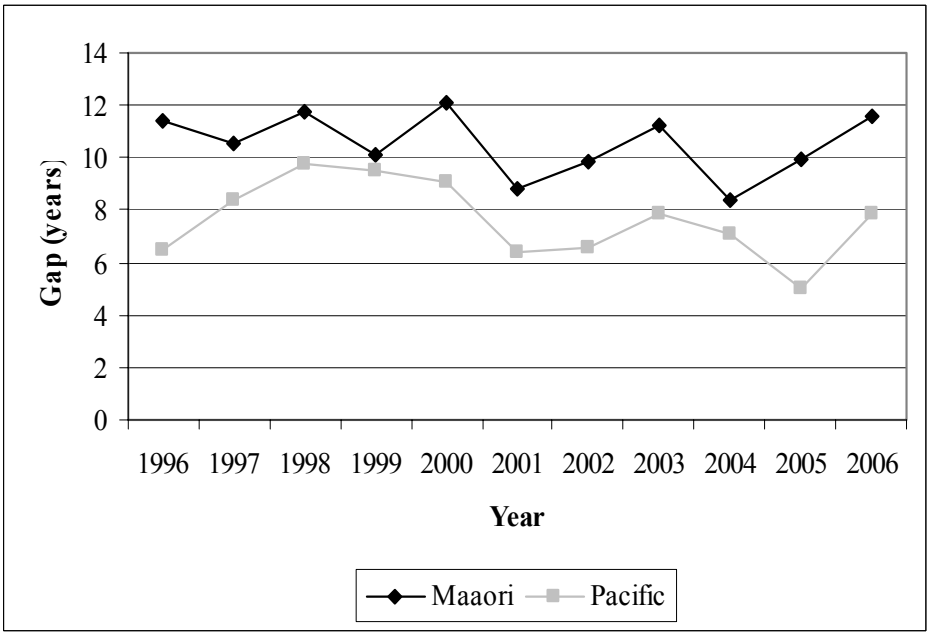


Data source: NZ Census (2006)

Life expectancy in CMDHB

Inequities in health, often referred to as health inequalities, are "differences in health that are unnecessary, avoidable, unfair and unjust" [8]. Inequity in health between Maaori and non-Maaori has been a dominant feature of the New Zealand health landscape for many years [9-11].

Figure 3: Gap in life expectancy at birth (in years) between those of Maaori and Pacific ethnicities and those of non-Maaori, non-Pacific ethnicities in CMDHB, 1996-2006



Data source: Ministry of Health Mortality Collection

Differences in life expectancy were used to illustrate health inequity between Maaori and non-Maaori at a national level, in the recent report Hauora IV [12]. For the period 2000-2002, life expectancy at birth for Maaori males was 69.0 years, while for non-Maaori males it was 77.2 years, a difference of more than eight years. Similarly, life expectancy at birth during the 2000-2002 time period was 73.2 years for Maaori females and 81.9 years for non-Maaori females, a difference of almost nine years. For Pacific peoples, the life expectancy nationally was 67.9 years for men and 73.9 years for women between 1996 and 1999, while at the same time it was 75.7 years and 80.8 years respectively for those of non-Maaori, non-Pacific ethnicities [9]. This represented a difference of almost eight years for men and almost seven years for women.

Life expectancy at birth in CMDHB has generally improved for Maaori and Pacific peoples over the last ten to fifteen years [13, 14]. However, despite these improvements, a gap in life expectancy at birth between Maaori and non-Maaori, non-Pacific of around ten years and between Pacific and non-Maaori, non-Pacific of around seven years has been relatively consistent in CMDHB over the past decade (Figure 3).

Comment on ethnicity

Ethnicity is a social construct which is centred around cultural affiliation and shared identity [15]. It is self-perceived, may change over time and individuals can belong to more than one ethnicity. Ethnicity is distinct from concepts such as race, citizenship, nationality and ancestry. Statistics New Zealand defines an ethnic group as having members who identify with some or all of the following characteristics [16]:

- A common proper name
- One or more elements of common culture which need not be specified, but may include religion, customs, or language
- Unique community of interests, feelings and actions
- A shared sense of common origins or ancestry, and
- A common geographic origin

Consistent with the Ministry of Health document *Ethnicity Data Protocols for the Health and Disability Sector[17]*, ethnicity in the mortality data used for this report was recorded at Statistics New Zealand Level 2 [18]. Three ethnicity fields are available in the mortality database, although only the first field is mandatory [19].

The models developed for the analyses in this report use prioritised ethnicity. In prioritised ethnicity, people are assigned to only one ethnic group, according to a specific prioritisation schedule. The rationale behind prioritisation is that there are situations, for example the creation of life tables by ethnicity, where individuals need to be allocated to only one ethnic group. Groups of policy importance, such as Maaori and Pacific peoples, must be identified so that findings for these groups are not lost in amongst those of the dominant NZ European group [17].

The downside of using prioritised ethnicity is that it is contrary to the idea of individual self-identification with more than one ethnicity, mentioned above. Furthermore, the prioritisation process means that certain groups become over-represented at the expense of others. These drawbacks must be considered when appraising the findings of this report.

The group non-Maaori, non-Pacific in this report refers to a heterogenous group of people who did not identify with Maaori or Pacific ethnicities in Counties Manukau in 2005. This mixed group was composed mainly of those of NZ European ethnicity, although various Asian ethnicities comprised around a quarter of the group [7]. The non-Maaori, non-Pacific group in Counties Manukau is relatively affluent compared with the rest of New Zealand and has a slightly higher life expectancy [7, 20]. The current report uses the slightly higher CMDHB benchmark in its appraisal of life expectancy gaps.

Methods

Life tables

Life tables "…are used to display the survival pattern of a community when we do not know the exact survival time of each individual, but we do know the number of individuals who survive at a succession of time points" [21]. This study uses 'current life tables', meaning that current age-specific death rates are applied to a hypothetical population to determine expected survival [21]. This hypothetical population is a closed cohort, meaning that no new members are added over time and members are only lost due to death [3].

The Chiang II method of life table calculation was used in this report. The two Chiang methods of constructing life tables are widely used internationally, and have been validated for use with smaller population groups [5, 22]. These methods use abridged life tables, which aggregate deaths and population level data into age groups (in contrast with complete, national-level life tables which analyse mortality by year of age). Age-specific mortality rates are calculated for each age interval, and are then used to calculate the probability of dying in each age interval. The calculated probabilities are subsequently applied to the hypothetical birth cohort. In this instance, a hypothetical cohort of 100,000 people was used (100,000 is a fairly standard initial cohort in life tables). Five-year age intervals were used to group data, for all age groups between five years and 84 years. Infants (aged < 1 year) were considered separately, leaving a four-year age band between ages one and four years (inclusive). Mortality in those aged ≥ 85 years was aggregated.

The Chiang methods use specific values in life table calculations that estimate the fraction of the age interval lived by the group that die within the interval. This value is labelled 'a_x' in the life tables. In keeping with current convention, this report assumed that deaths within particular age intervals were evenly spread [5]. Therefore, the average years lived in each age band (by those who died in that age group) was half way through the age interval. The life tables used in this study applied a_x values of 0.5 for all age bands except infants, where a value of 0.1 was used (in recognition of the fact that most infant deaths occur within the first few weeks of life). The Chiang II method of life table calculation used in this analysis is very similar to the original Chiang method. It differs only subtly, in the calculation of variance in age groups which have zero deaths. The Chiang II method has been validated in small populations, down to 5,000 people. Further detail on Chiang and Chiang II methods can be found in the epidemiology literature [5, 22].

Time period of mortality data

The calendar year 2005 was chosen for this study, for the following reasons:

- 2005 is the most recent year for which there is a complete set of ICD-10-AM coded mortality data for CMDHB
- There were sufficient numbers of deaths in each age category to undertake the analyses using the life table models
- Life expectancy estimates by ethnicity for 2005 are generally representative of the trend in life expectancy over the past ten years, although the gap between Pacific and non-Maaori, non-Pacific in 2005 was smaller than in previous years

Ministry of Health Mortality Collection

Detail on the Mortality Collection can be found on the Ministry of Health Information Directorate (MOH ID) website - http://www.ID.govt.nz/. This data collection covers all deaths registered in New Zealand since 1948. It includes all registered foetal deaths (stillbirths).

By law, all deaths must be registered within three working days after the cremation or burial of a body. Each time a death occurs, the undertaker or funeral director completes the notification of death registration form. Relevant documentation, including the medical certificate of causes of death completed by the medical practitioner, is forwarded to Births, Deaths and Marriages (BDM). BDM then forward electronic death registration data to MOH ID monthly. The data is assigned an NHI number, ethnicity and domicile codes by MOH ID. Clinical coders then code for underlying cause of death. The coding of cause of death data is considered to be of a high standard, as MOH ID coders undertake a thorough process of verification which involves cross-referencing with other MOH ID databases and original documents in cases of missing data or ambiguity [23, 24]. Sources of data for clinical coding include: Hospital discharge data from NMDS[1], post-mortem reports and coroners findings, NZCR[2], Police, Land Transport Safety Authority and Water Safety New Zealand; together with letters to medical practitioners, coroners and hospital medical records departments. Cause of death is then coded using ICD-10-AM[3].

Ethnicity is initially entered onto the notification of death registration form by the funeral director, in consultation with the family. Later, ethnicity is coded by MOH ID clinical coders to Statistics New Zealand Level 2, consistent with ethnicity recording requirements for the New Zealand health and disability sector [17]. Up to three ethnicity codes can be stored in the system. Ethnicity codes are prioritised in this study using the standard prioritisation protocol recommended by the Ministry of Health [17].

Disease groupings

Mortality data from 2005 in CMDHB were separated into six separate disease 'areas' or 'groupings' in this report, according to the principal ICD-10-AM cause of death code. The following six areas were used:

- Area one: Infant mortality (i.e. all mortality in children aged under one year)
- Area two: Cardiovascular disease. This area included ischaemic heart disease (IHD), peripheral vascular disease (PVD) and cerebrovascular disease (stroke)
- Area three: Smoking-related lung disease. This group included the main lung diseases commonly associated with smoking, such as COPD and lung cancer (diseases mainly, but not always caused by smoking)
- Area four: Cancer. This disease grouping looked at all malignant cancers except lung cancer (which was covered in area two - smoking-related lung diseases)
- Area five: Diabetes

[1] National Minimum Data Set
[2] New Zealand Cancer Registry
[3] International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification

- Remainder: This group included all deaths in 2005 in CMDHB which did not have ICD-10-AM principal cause of death codes that fitted any of the five areas above

The specific ICD-10-AM codes for each of the five major disease areas are given in Table 1. Any death which did not have a principal ICD-10-AM cause of death diagnosis code listed in Table 1 was included in the remainder group.

Table 1: ICD-10-AM cause of death codes used to categorise individual deaths into specific disease groupings

Disease area	ICD-10-AM code	Description of ICD-10-AM code
Area one: Infant mortality	No specific ICD-10-AM codes are assigned to this area. All deaths in those aged < 1 year are included	
Area two: Cardiovascular disease	I10 - I15	Hypertensive diseases
	I20 - I25	Ischaemic heart diseases
	I50	Heart failure
	I63	Cerebral infarction
	I64	Stroke - not classified as haemorrhage or infarction
	I69	Sequelae of cerebrovascular disease
	I70	Atherosclerosis
	I71	Aortic aneurysm and dissection
	I72	Other aneurysm
	I74	Arterial embolism and throbosis
Area three: Smoking-related lung disease	J42	Unspecified chronic bronchitis
	J43	Emphysema
	J44	Other chronic obstructive pulmonary disease
	C34	Malignant neoplasm of bronchus and lung
Area four: Cancer	This disease grouping looks at all malignant cancers except lung cancer (which is covered in area two - smoking-related lung disease)	
	C00 - C26	Malignant neoplasms of lip, oral cavity, pharynx and digestive organs
	C40 - C96	Remaining group of malignancies except lung cancer
Area five: Diabetes	E10 - E14	Diabetes mellitus

Although the process of aggregating mortality data into disease areas was systematic (based on both absolute contribution of causes of death to mortality numbers and on the contribution of these causes of death to inequity), the rationale behind the grouping of these five cause of death areas was somewhat arbitrary. The Global Burden of Disease Projects undertaken by WHO between 1990 and 2002 looked at the contribution of three distinct disease groups to mortality and life expectancy as part of the overall examination of worldwide disease burden: Group I consisted mainly of communicable diseases (but also included maternal diseases, perinatal conditions and nutritional deficiencies), Group II consisted of chronic diseases and Group III

comprised intentional and unintentional injuries [25, 26]. While these categories may be suited to the international environment, they are not necessarily appropriate for evaluation of the aetiology of life expectancy gaps in CMDHB or New Zealand more generally. Local research indicates that groupings around causes of death such as CVD, diabetes, cancer and respiratory diseases may have more relevance to this analysis [9].

'League tables' were created of cause of death by ethnicity for 2001 to 2005 (Appendix one). Where a biological/anatomical relationship existed between common causes of death, these causes were grouped together under one disease area. The five areas which emerged from this analysis were then critically considered in the context of local health needs assessments [13, 27]. Finally, the five areas were discussed with Maaori and Pacific Health teams at respective team meetings, with feedback sought on the suitability of the five separate areas and on the alignment of these areas with the strategic priorities of these teams in Counties Manukau.

Contribution of disease groups to mortality

The contribution of each of the five disease areas to the life expectancy gap between those of Maaori and Pacific ethnicities and those of non-Maaori, non-Pacific ethnicities was assessed in the models by substituting age-specific mortality data for Maaori and Pacific prioritised groups with adjusted age-specific mortality data for non-Maaori, non-Pacific people in CMDHB. For example, in examining the role of Area two: Cardiovascular disease, in the life expectancy gap between Maaori and non-Maaori, non-Pacific; absolute numbers of deaths attributable to cardiovascular disease in each age stratum for Maaori (i.e. deaths with principal ICD-10-AM cause of death codes found in Area two in Table 1) were substituted with derived 'expected' numbers. The 'expected' number for each age stratum was the absolute number of deaths for non-Maaori, non-Pacific in that age category, adjusted to fit differences in population size between the two populations. Therefore, a simple substitution of Maaori and Pacific mortality with (adjusted) non-Maaori, non-Pacific mortality in CMDHB was used to estimate the effect on the life expectancy gap of each of the five areas.

Adjustment

The sum of percentage contributions of each of the five disease areas individually did not exactly equal the total percentage contribution of all disease areas (in aggregate) to the life expectancy gaps. It is likely that these differences were due to rounding errors, whereby 'expected' numbers of deaths (which were originally calculated using decimal places and then entered the life tables as whole numbers) were 'smoothed' out in the aggregation process, leading to minor variations when compared to the sum of the individual contributions. A minor rescaling was undertaken, whereby proportional disease area contributions were adjusted so that their sum equalled the aggregate contribution of all five areas combined.

Secondary analysis of smoking impact

Disparities in mortality from diseases related to smoking were identified in the main life expectancy models as providing a key contribution to life expectancy gaps. Further simplified analysis of the relationship between smoking prevalence and life expectancy was therefore undertaken, using the following methods.

Simplified models of life expectancy by ethnicity in CMDHB used all-cause mortality rate ratios in current smokers (compared with never-smokers) to examine the effect of changing the prevalence of smoking in Maaori and Pacific populations on the life expectancy gap between Maaori, Pacific and non-Maaori, non-Pacific. These were uncomplicated models, intended to provide insight into the effect of changing smoking prevalence on life expectancy trends in general terms. They were not intended to provide a detailed forecast of changes in life expectancy gaps. A description of assumptions used in these models is provided in Appendix Four.

Population attributable risk percent (PAR%) is defined as "the reduction in incidence that would be achieved if the population had been entirely unexposed, compared with its current (actual) exposure pattern" (p.67) [3]. PAR% describes the amount of disease (or mortality) in a population that can be attributed to an exposure [1, 3]. In this case, we are interested in how much mortality in CMDHB could have been explained by smoking in 2005. Box 1 provides the equation used to estimate PAR% in this report. Confidence intervals for PAR% were estimated using the method for attributable risk described by Altman et al [28].

Box 1. Population attributable risk equation

$$PAR\% = \frac{F(RR - 1)}{1 + F(RR - 1)} \times 100$$

Where: F is the prevalence of regular smoking in the population

The models in this report compared mortality from smoking given the current prevalence of regular smoking in CMDHB, with various counterfactual prevalence scenarios for regular smoking in CMDHB. In counterfactual approaches to the attribution of an outcome such as mortality to a risk factor like smoking, hypothetical scenarios are generated in which the effect of different risk factor exposure distributions on outcomes is estimated [3, 29]. For the models in this report, prevalence of regular smoking in CMDHB was altered in various hypothetical scenarios to understand the impact of such changes on mortality and on the life expectancy gap between Maaori, Pacific peoples and those of non-Maaori, non-Pacific ethnicities. Counterfactual mortality numbers were substituted into each age category in the abridged life tables for Maaori and Pacific peoples in 2005, based on the PAR% for each of the hypothetical prevalence estimates.

Estimates of mortality attributable to smoking in New Zealand and internationally have applied mortality risk estimates from the American Cancer Society, Cancer Prevention Study (CPS) II to various study populations [29-34]. CPS II was a prospective study of over one million smokers and non-smokers aged ≥ 30 years in the US between 1982 and 1988 [35, 36]. Rate ratios for all-cause mortality (current smokers vs. non-smokers) for six years of follow up in CPS II have been described by Thun et al [37]. The rate ratios for all-cause mortality (current smokers vs. non-smokers) in CPS II were 2.3 (95% CI 2.3 to 2.4) for males and 1.9 (95% CI 1.9 to 2.0) for females. However, data for CPS II were collected in the early 1980's and 93% of the study population were white Americans, leaving questions as to whether the rate ratios found in this study were suitable for use in the CMDHB population. Hunt et al

found that the relative effect of smoking on mortality differs over time and by ethnicity [38]. They used linked census and mortality (NZCMS) data to compare mortality between current smokers and never smokers in New Zealand between 1981-84 and 1996-99. They described the rate ratios presented in Table 2 in the 1996-99 New Zealand cohort. These rate ratios were used in the models in this report. Models which used rate ratios from CPS II have also been included in the report for comparative purposes. Note that the rate ratios for mortality in smokers are much lower in Maaori and Pacific peoples than for non-Maaori, non-Pacific in this study. It is thought that absolute differences in mortality rates explain in part this heterogeneity in the association between smoking and mortality by ethnicity. Maaori and Pacific peoples have higher mortality rates than non-Maaori, non-Pacific. Although risk differences between smokers and non-smokers were similar in each ethnic group, the higher mortality among Maaori and Pacific peoples meant that the rate ratios were comparatively lower. A second possible explanation for the effect measure modification seen was that Maaori and Pacific peoples experienced higher mortality due to a range of different (and often interacting) factors such as access to health care and socioeconomic position. In the context of these other factors, cigarette smoking may have been relatively less prominent as a risk factor for mortality.

Table 2: Standardised rate ratios for all-cause mortality – current vs. never smokers aged 25-74 years – in New Zealand, by ethnicity, 1996-1999

	Males		Females	
	Rate ratio	(95% CI)	Rate ratio	(95% CI)
Maaori	1.51	(1.35 to 1.69)	1.45	(1.27 to 1.66)
Pacific	1.18	(0.94 to 1.47)	1.05	(0.75 to 1.48)
non-Maaori, non-Pacific	2.22	(2.12 to 2.33)	2.20	(2.09 to 2.33)
All ethnicities	2.05	(1.97 to 2.14)	2.01	(1.91 to 2.12)

Adapted from: Hunt D, et al (2005). Int J Epi; 34:1020-1028

Box 2. Key model assumptions

- Mortality attributable to smoking differed by sex
- Proportion of total age-specific mortality attributable to smoking was consistent across all adult age groups (either ≥ 25 or ≥ 30 years)
- Smoking prevalence was a reasonable indicator of accumulated smoking risk (including factors like induction time for smoking-related conditions) and any affect of a change in prevalence on mortality was immediate
- Mortality risk in ex-smokers was the same as that in non-smokers
- Smoking prevalence and life expectancy at birth in non-Maaori, non-Pacific remained constant
- Second-hand smoke exposure did not contribute to mortality and life expectancy gaps
- Estimates of attribution did not require adjustment with a smoking impact ratio (SIR)

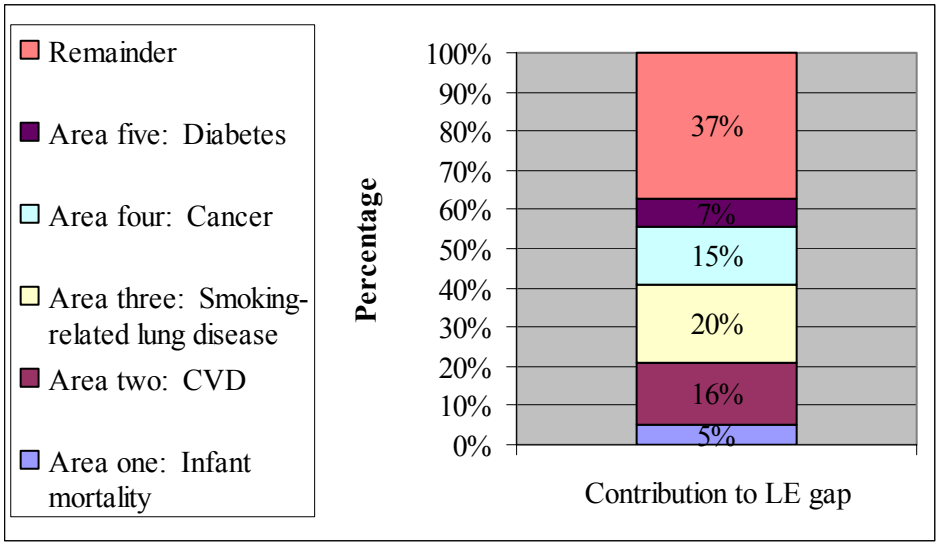
The models in this report provide simplified estimates of the impact of changes in smoking prevalence on PAR% and on life expectancy gaps between Maaori, Pacific peoples and non-Maaori, non-Pacific. Significant areas where smoking contributes to mortality have been omitted from the models, as the contribution of such areas could not be adequately accounted for within the models. Examples of such areas include the impact of second-hand smoking on mortality (and in particular on infant mortality) and the increased risk of mortality among ex-smokers. Some information on assumptions used in these models is given in Box 2. Further detail on the models can be found in Appendix Four.

Results and interpretation

Maaori

Using the models developed for those of Maaori ethnicity and those of non-Maaori, non-Pacific ethnicities, life expectancy at birth in 2005 was estimated for each group. For Maaori (both males and females), life expectancy at birth in 2005 was 72.1 years (95% CI 70.6 to 73.6 years). For non-Maaori, non-Pacific, life expectancy at birth in 2005 was 82.4 years (95% CI 81.9 to 82.9 years). This was a difference of over ten years (10.3 years). The proportional contribution of each disease area to this disparity is presented in Figure 4.

Figure 4: Contribution of each main cause of death area to LE gap between Maaori and non-Maaori, non-Pacific in CMDHB, 2005



Area three: Smoking-related lung disease was the main contributor to the disparity in life expectancy observed. Smoking-related lung disease, which included lung cancer and chronic bronchitis (COPD), contributed to over 20% of the difference in life expectancy. In 2005, 47 Maaori died of lung cancer or COPD, five times the number that would be expected if Maaori mortality rates from smoking-related lung disease were the same as those of non-Maaori, non-Pacific ethnicities. If mortality rates from smoking-related lung disease were the same in each group, the life expectancy gap would have been narrowed by over two years (Figure 5). Note that this estimate relates only to lung disease caused by smoking. It does not take into account the effect of smoking on each of the other four disease areas, especially cardiovascular disease and cancers other than lung cancer.

In 2005, 56 Maaori deaths were attributed to cardiovascular disease. Only 20 cardiovascular deaths would have been expected if the cardiovascular mortality rate in Maaori had been the same as in non-Maaori, non-Pacific. Furthermore, Maaori deaths from cardiovascular disease tended to be in younger age groups (Figure 6). The combination of these two factors meant that cardiovascular disease made a considerable contribution to the life expectancy gap, of around 20 months.

Figure 5: Contribution of each main cause of death area to LE gap in years between Maaori and non-Maaori, non-Pacific in CMDHB, 2005

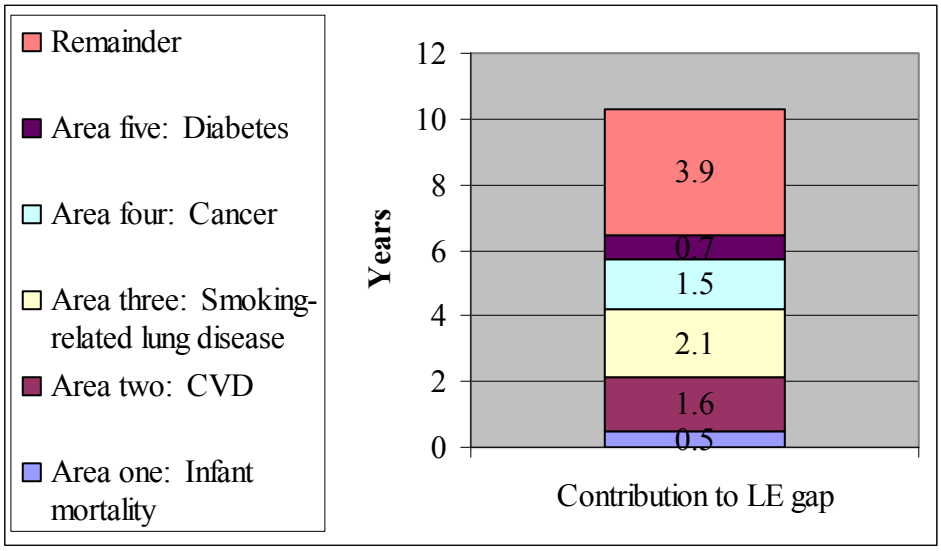
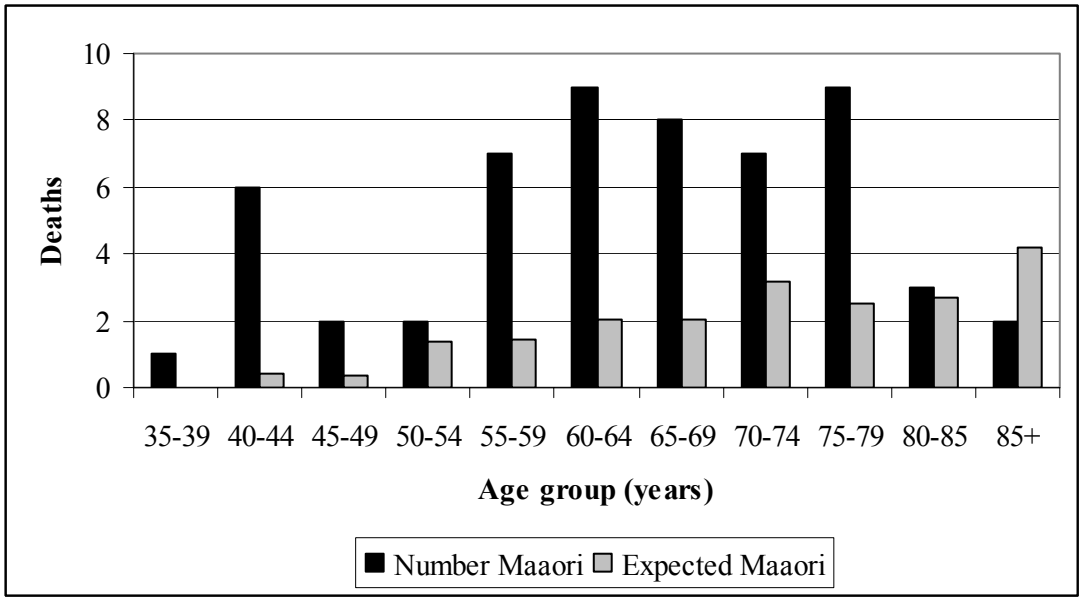


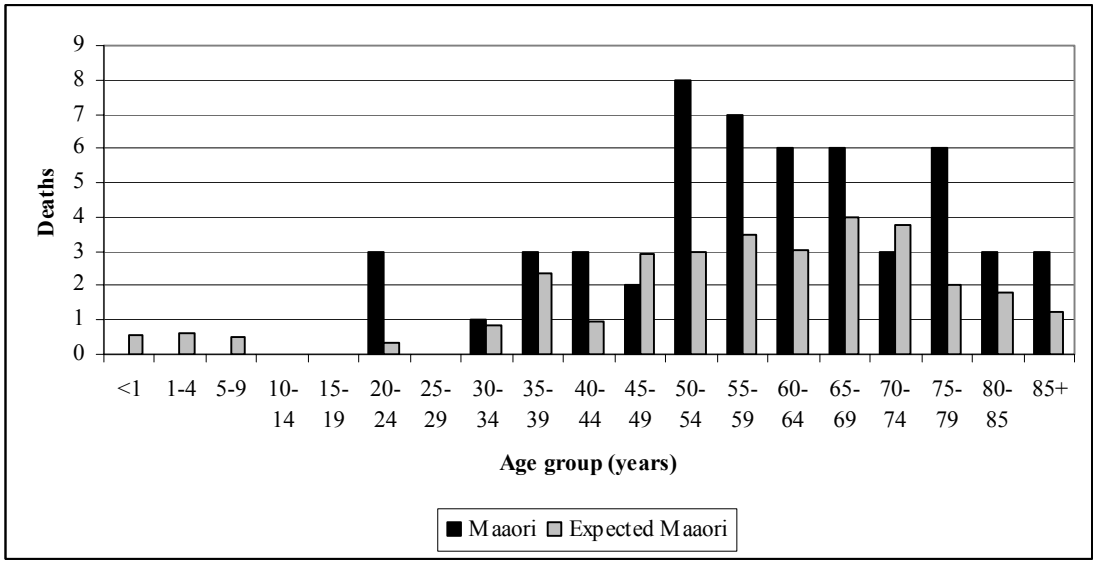
Figure 6: Deaths from CVD amongst Maaori in CMDHB by age group in 2005, and expected deaths (based on non-Maaori, non-Pacific CVD mortality)



Aside from cancer deaths amongst the very young, Maaori cancer deaths (excluding lung cancer which is dealt with in area three) outstripped those expected, based on non-Maaori, non-Pacific rates, in almost every age group (Figure 7). Had Maaori cancer mortality rates been the same as for non-Maaori, non-Pacific in 2005, the life expectancy gap would have closed by 15%, or around 18 months.

In 2005, 19 deaths amongst Maaori in CMDHB were attributed to diabetes, six times as many as would have been expected if Maaori had the same mortality from diabetes as non-Maaori, non-Pacific. Again, Maaori deaths from diabetes were greater than those expected in all groups aged ≥ 30 years. Overall, mortality due to diabetes accounted for seven percent of the gap in life expectancy between Maaori and non-Maaori, non-Pacific (around 18 months).

Figure 7: Deaths from cancer (excluding lung cancer) amongst Maaori in CMDHB by age group in 2005, and expected deaths (based on non-Maaori, non-Pacific cancer mortality)



The contribution of infant mortality to the life expectancy gap is the smallest of any of the five disease areas, at around six months. During 2005, 24 Maaori infants (aged under one year) died, whereas around ten infants would have been expected to die if the Maaori infant mortality rate had been the same as for non-Maaori, non-Pacific (i.e. the Maaori infant mortality rate was almost 2.5 times the expected rate).

The remaining 37% of the life expectancy gap between Maaori and non-Maaori, non-Pacific consisted of a wide range of ICD-10-AM codes. Each of these codes contributed only very small numbers of deaths to the model and none of the codes could be grouped into another meaningful disease area for further analysis in the model.

Pacific peoples

Life expectancy at birth in 2005 for Pacific peoples was compared with life expectancy for those of non-Maaori, non-Pacific ethnicities. Life expectancy at birth for Pacific peoples in 2005 was 76.8 years (95% CI 75.5 to 78.1 years). This was more than 5.6 years less than the 82.4 years (95% CI 81.9 to 82.9 years) enjoyed by those of non-Maaori, non-Pacific ethnicities. The proportional contribution of each disease area to this disparity is presented in Figure 8.

Area two: Cardiovascular disease had the greatest contribution to the life expectancy gap between Pacific peoples and non-Maaori, non-Pacific. Area two contributed to 35% of the total gap, or almost two years (Figure 9). Amongst Pacific peoples, 80 deaths in 2005 were attributed to cardiovascular disease, whereas only 40 deaths would have been expected if Pacific peoples had the same cardiovascular mortality rates as non-Maaori, non-Pacific. Pacific cardiovascular deaths exceeded expected mortality in all age groups (aged $\geq$ 30 years) (Figure 10).

Figure 8: Contribution of each main cause of death area to LE gap between Pacific and non-Maaori, non-Pacific in CMDHB, 2005

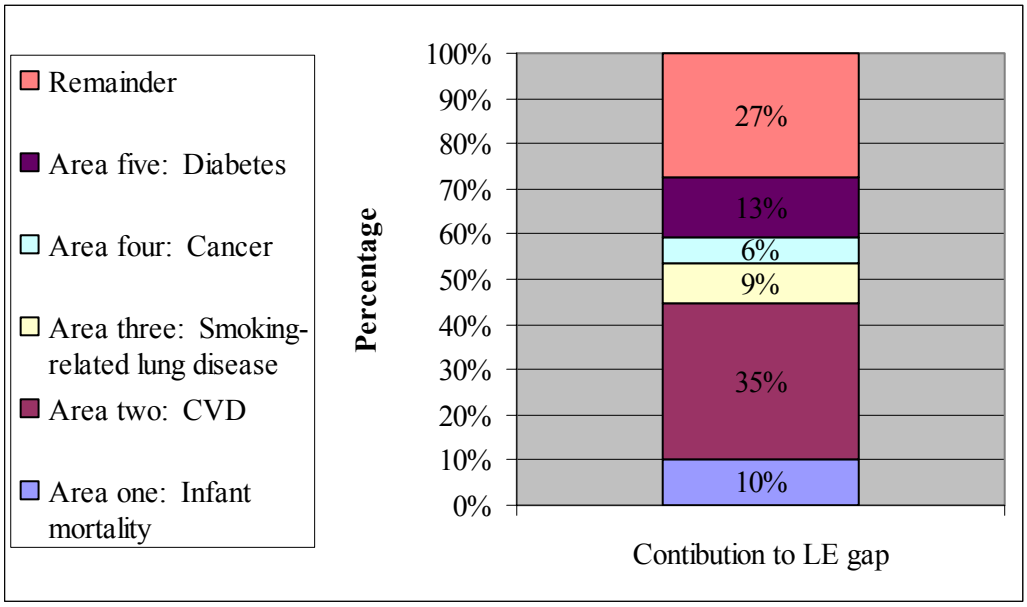
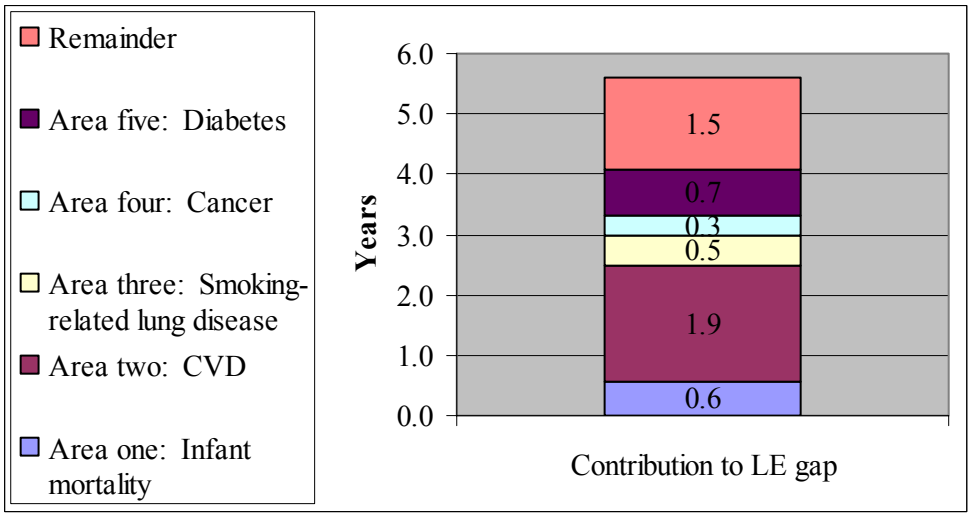


Figure 9: Contribution of each main cause of death area to LE gap in years between Pacific and non-Maaori, non-Pacific in CMDHB, 2005

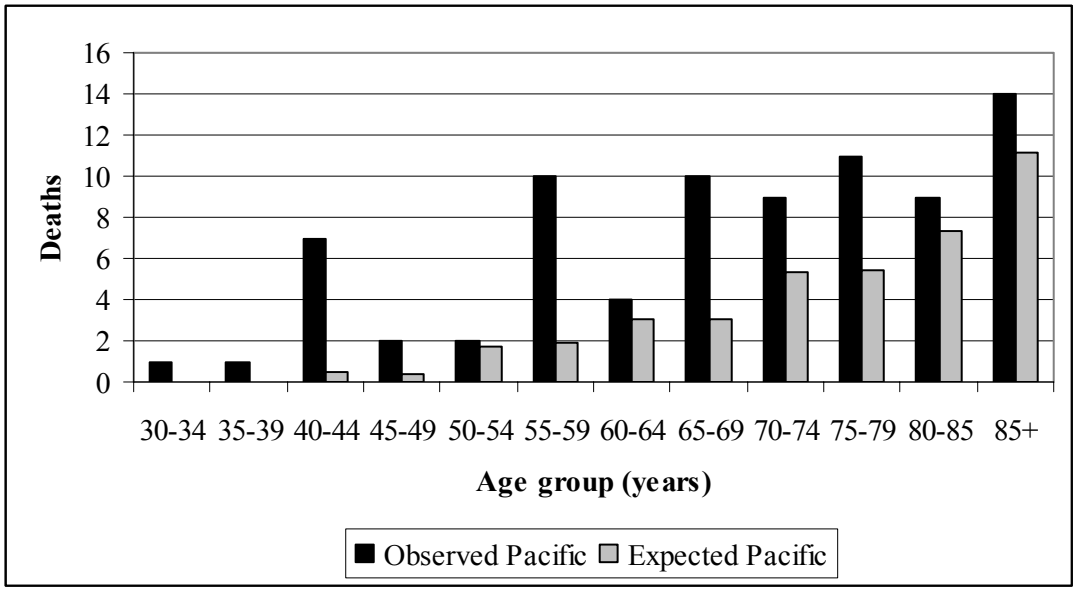


Area five: Diabetes was responsible for 13% of the life expectancy gap between Pacific and non-Maaori, non-Pacific in 2005, or around nine months (Figure 9). There were 22 deaths amongst Pacific peoples in 2005 which were attributed to diabetes, whereas only five would have been expected if Pacific peoples had the same diabetes mortality rate as non-Maaori, non-Pacific. Taken together, diabetes and cardiovascular disease were responsible for almost half of the life expectancy gap between Pacific and non-Maaori, non-Pacific, or two years and eight months.

Smoking-related lung disease had a much smaller impact on the life expectancy gap for Pacific peoples in 2005 than for the Maaori life expectancy gap. While death from smoking-related lung disease was responsible for over 20% of the Maaori life expectancy gap, it was responsible for 9% of the gap between Pacific and non-Maaori, non-Pacific. This was equivalent to about six months of the gap. As discussed earlier, the contribution of smoking-related lung disease to the life expectancy gap

does not take into account the numerous other lethal effects of tobacco on life expectancy, such as effects on cardiovascular disease and other types of cancer.

Figure 10: Deaths from CVD amongst Pacific peoples in CMDHB by age group in 2005, and expected deaths (based on non-Maaori, non-Pacific CVD mortality)



Infant mortality was responsible for about ten percent of the difference in life expectancy between Pacific and non-Maaori, non-Pacific in 2005, or about seven months. Twenty two Pacific infants died in 2005, while only six would have been expected to die if infant mortality rates had been the same for Pacific as for non-Maaori, non-Pacific - an almost fourfold difference.

Mortality due to non-lung cancer provided the smallest contribution to the difference in life expectancy between Pacific and non-Maaori, non-Pacific in 2005, at around four months or six percent of the total. Sixty Pacific people died of cancers (other than lung cancer, which is dealt with in smoking-related lung diseases). If Pacific cancer mortality rates had been the same as non-Maaori, non-Pacific in 2005, 49 such deaths would have been expected.

About one quarter (27%) of the gap between Pacific and non-Maaori, non-Pacific life expectancy was due to the wide range of ICD-10-AM codes that made up the remainder group. As was the case with analysis of the gap between Maaori and non-Maaori, non-Pacific, each of the codes contributed only small numbers of deaths to the model and none of the codes could be grouped into another meaningful disease area for analysis in the model.

Results of smoking analysis

Simplified analysis of the impact of smoking on life expectancy in Counties Manukau was undertaken. This analysis looked at the relationship between smoking prevalence and mortality from all causes among Maaori and Pacific smokers.

Maaori

When rate ratios for all-cause mortality for smokers versus never-smokers of 1.51 (95% CI 1.35 to 1.69) for Maaori men and 1.45 (95% CI 1.27 to 1.66) for Maaori

women [38] were applied to the standard PAR% equation given in Box 1 for Maaori adults aged ≥ 25 years in 2005, hypothetical smoking-attributable mortality percentages using different counterfactual smoking prevalence estimates were obtained (Table 3).

At the 2006 smoking prevalence of 46.8% for Maaori adults (aged ≥ 15 years) in CMDHB (50.3% for women and 42.5% for men), around 45-50 deaths out of a total of 300 in 2005 were considered attributable to smoking. If the prevalence of smoking were reduced to 20%, which was the smoking prevalence for those of NZ European ethnicity in CMDHB in 2006, this number would have halved to about 20-25 deaths due to smoking. The mortality estimates seen in Table 3 are somewhat less than would have been expected, based on actual ICD-10-AM mortality codes for Maaori in CMDHB in 2005. There were around 45-50 Maaori deaths for smoking-related lung disease alone in 2005, the majority of which would have been caused by smoking. Furthermore, there were around 110 additional deaths due to cardiovascular disease and other cancers, at least some of which would have been attributable to smoking. For comparative purposes, the ethnicity-standardised rate ratios for all-cause mortality in men and women in New Zealand (2.05 and 2.01 respectively) [38] were used to estimate PAR% (Table 4). These mortality rate ratios are closer to those historically used to estimate smoking attributable mortality burden [33, 37, 39, 40] and also relate directly to the New Zealand context.

Table 3: Expected PAR% and mortality estimates for smoking-attributable mortality in CMDHB in 2005 (Maaori adults aged ≥ 25 years), using counterfactual smoking prevalence estimates

Female				
		95% CI for PAR (%)		Estimated smoking
Prevalence	PAR (%)	Lower	Upper	attributable deaths in 2005
50.3%	18.5%	12.0%	24.9%	23
50.0%	18.4%	11.9%	24.8%	23
45.0%	16.8%	10.8%	22.9%	21
40.0%	15.3%	9.7%	20.9%	19
30.0%	11.9%	7.5%	16.5%	15
20.0%	8.3%	5.1%	11.7%	10
10.0%	4.3%	2.6%	6.2%	5
Male				
		95% CI for PAR (%)		Estimated smoking
Prevalence	PAR (%)	Lower	Upper	attributable deaths in 2005
42.5%	17.8%	12.9%	22.7%	24
40.0%	16.9%	12.3%	21.6%	23
35.0%	15.1%	10.9%	19.5%	20
30.0%	13.3%	9.5%	17.1%	18
20.0%	9.3%	6.5%	12.1%	13
10.0%	4.9%	3.4%	6.5%	7

When the counterfactual scenarios described in Table 3 are applied to hypothetical life tables for Maaori in CMDHB in 2005, a distribution of life expectancy (and life expectancy gap) according to smoking prevalence can be obtained, as described by

the blue curve 'Hunt 1' in Figure 11. At the current adult smoking prevalence for Maaori of 46.8%, life expectancy at birth for Maaori is 72.1 years compared with 82.4 years for non-Maaori, non-Pacific, a difference of 10.3 years. When smoking prevalence in Maaori is reduced to that of NZ European adults (20.0%), the counterfactual life expectancy rises by 1.2 years to 73.3 years (and the life expectancy gap correspondingly narrows by about 12%). If the prevalence of smoking is lowered to zero (i.e. in a hypothetical scenario where nobody of Maaori ethnicity smoked), life expectancy at birth would rise by 2.6 years to 74.7 years and the gap between Maaori and non-Maaori, non-Pacific would narrow to 7.7 years (a 25% narrowing), as can be seen by the intersect with the 'y' axis in Figure 11. The pink curve 'Hunt 2' in Figure 11 uses the male and female mortality rate ratios for all ethnicities described in the paper by Hunt et al [38]. The greater risk of smoking mortality in this scenario is reflected in the steeper curve. In this scenario, if the smoking prevalence of NZ European adults in CMDHB remained fixed, 5.2 years (more than half of the life expectancy gap) would have been removed if Maaori smoking prevalence had been zero. In the counterfactual situation where Maaori smoking prevalence was the same as that for NZ European adults, the life expectancy gap narrowed by over two years, or around 20%. This is closer to what would be expected based on the findings of the original models which looked at the contribution of particular disease areas to mortality and to the life expectancy gap between Maaori and non-Maaori, non-Pacific in CMDHB in 2005.

Table 4: Expected PAR% and mortality estimates for smoking-attributable mortality in CMDHB in 2005 (Maaori adults aged ≥ 25 years), using counterfactual smoking prevalence estimates and aggregated all-cause mortality rate ratios for ALL ethnicities in New Zealand

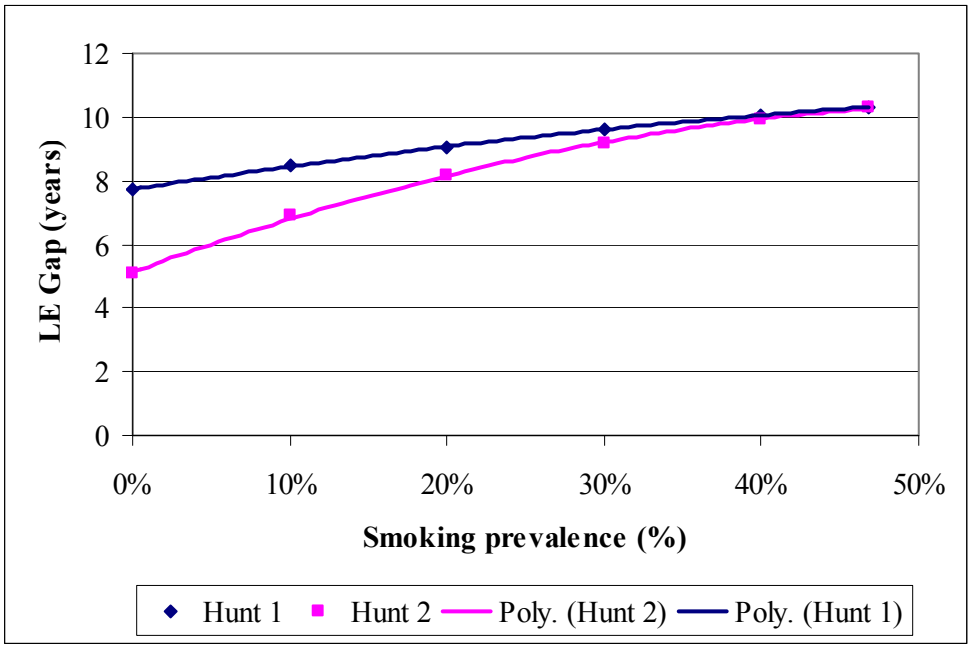
Female

		95% CI for PAR (%)		Estimated smoking
Prevalence	PAR (%)	Lower	Upper	attributable deaths in 2005
50.3%	33.7%	31.4%	36.0%	43
50.0%	33.6%	31.3%	35.9%	43
45.0%	31.2%	29.1%	33.5%	40
40.0%	28.8%	26.7%	30.9%	37
30.0%	23.3%	21.4%	25.1%	30
20.0%	16.8%	15.4%	18.3%	21
10.0%	9.2%	8.3%	10.1%	12

Male

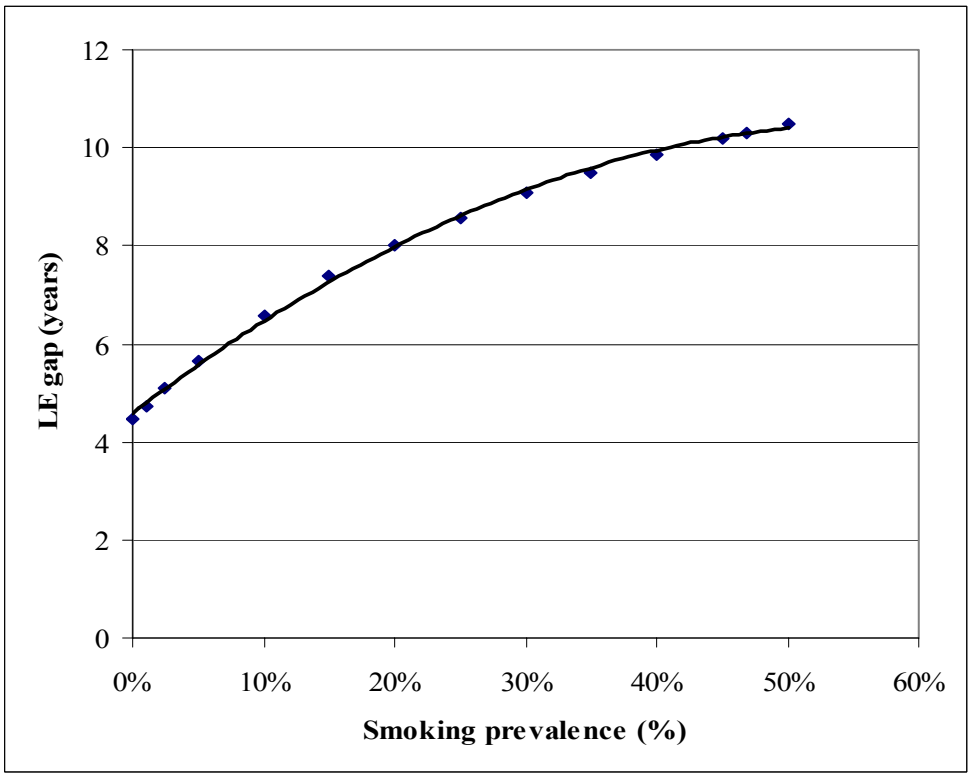
		95% CI for PAR (%)		Estimated smoking
Prevalence	PAR (%)	Lower	Upper	attributable deaths in 2005
42.5%	30.9%	29.2%	32.6%	40
40.0%	29.6%	28.0%	31.3%	39
35.0%	26.9%	25.3%	28.5%	35
30.0%	24.0%	22.5%	25.5%	31
20.0%	17.4%	16.2%	18.6%	23
10.0%	9.5%	8.8%	10.2%	12

Figure 11: Life expectancy gap between Maaori and non-Maaori, non-Pacific in CMDHB in 2005 in different counterfactual scenarios of adult smoking prevalence



By way of comparison, the rate ratio for all-cause mortality from CPS II (2.17) was used to generate similar scenarios of life expectancy with varying smoking prevalence [37]. The rationale for using this risk ratio is that CPS II findings have been used for much of the estimation of smoking mortality burden internationally over the past 20 years.

Figure 12: Life expectancy gap between Maaori and non-Maaori, non-Pacific in CMDHB in 2005, using different counterfactual adult smoking prevalence figures and all-cause mortality risk ratio from the US CPS II study



However, estimates generated using the CPS II rate ratio must be treated with caution, as the CPS II rate ratio is likely to be obsolete in the New Zealand context [38] and estimates have not been adjusted for relative lung cancer mortality (see Appendix Four). However, these estimates do give some insight into life expectancy gaps in scenarios where the risk of mortality among smokers is taken to be greater than that used in the ethnicity-specific model above. In the counterfactual scenario where smoking prevalence in adults dropped to 20.0% (the same prevalence as NZ European adults in CMDHB in 2006), the life expectancy gap between Maaori and non-Maaori, non-Pacific narrowed by about 20% to eight years, and the new Maaori life expectancy at birth became 74.4 years (Figure 12). As can be seen in Figure 12, the total contribution of smoking to the life expectancy gap for Maaori was about six years. In the hypothetical scenario where prevalence of tobacco smoking in Maaori was zero, Maaori life expectancy rose to almost 78 years.

Pacific

Rate ratios for all-cause mortality for smokers versus never-smokers of 1.18 (95% CI 0.94 to 1.47) for Pacific men and 1.05 (95% CI 0.75 to 1.48) for Pacific women [38] were applied to the standard PAR% equation given in Box 1 for Pacific adults aged ≥ 25 years in 2005. Hypothetical smoking-attributable mortality using different counterfactual smoking prevalence estimates was then obtained (Table 5).

Table 5: Expected PAR% and mortality estimates for smoking-attributable mortality in CMDHB in 2005 (Pacific adults aged ≥ 25 years), using counterfactual smoking prevalence estimates

Female				
		95% CI for PAR (%)		Estimated smoking
Prevalence	PAR (%)	Lower	Upper	attributable deaths in 2005
26.7%	1.3%	0.0%	11.4%	3
25.0%	1.2%	0.0%	10.7%	3
20.0%	1.0%	0.0%	8.8%	2
15.0%	0.7%	0.0%	6.7%	2
10.0%	0.5%	0.0%	4.6%	2
5.0%	0.2%	0.0%	2.3%	1
Male				
		95% CI for PAR (%)		Estimated smoking
Prevalence	PAR (%)	Lower	Upper	attributable deaths in 2005
34.3%	5.8%	0.0%	13.9%	8
30.0%	5.1%	0.0%	12.4%	7
25.0%	4.3%	0.0%	10.5%	6
20.0%	3.5%	0.0%	8.6%	5
15.0%	2.6%	0.0%	6.6%	4
10.0%	1.8%	0.0%	4.5%	3

Because the mortality rate ratios calculated by Hunt for Pacific male and female smokers versus non-smokers were so low (compared with those for all ethnicities), the PAR% estimates for all-cause mortality were also very low. The estimated number of deaths attributable to smoking in the CMDHB Pacific population in 2005 was only 11

people using these risk ratios. If the smoking prevalence were to reduce to 20.0% (the prevalence for NZ European adults in CMDHB in 2006), the number of smoking deaths would drop to only seven.

Figure 13: Life expectancy gap between Pacific and non-Maaori, non-Pacific in CMDHB in 2005 in different counterfactual scenarios of adult smoking prevalence, using rate ratios for all-cause mortality in Pacific smokers estimated by Hunt et al

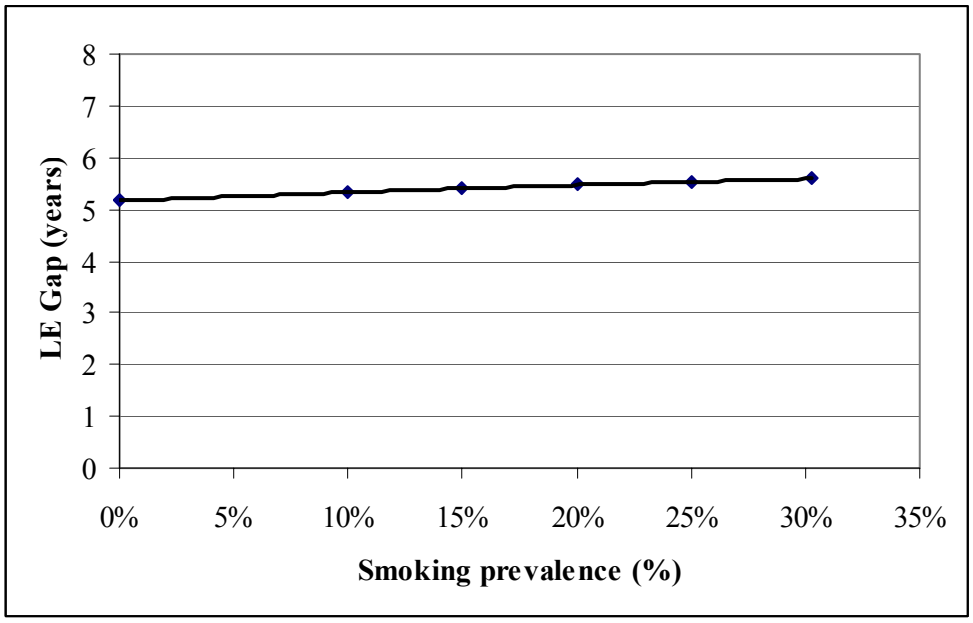


Table 6: Estimated PAR% and mortality due to smoking among Pacific adults aged ≥ 25 years in CMDHB in 2005, using all-cause mortality rate ratios for ALL ethnicities (from Hunt et al) and various counterfactual smoking prevalence scenarios

Female

		95% CI for PAR (%)		Estimated smoking
Prevalence	PAR (%)	Lower	Upper	attributable deaths in 2005
26.7%	21.2%	19.5%	23.0%	28
25.0%	20.2%	18.5%	21.9%	26
20.0%	16.8%	15.4%	18.3%	22
15.0%	13.2%	12.0%	14.4%	18
10.0%	9.2%	8.3%	10.1%	13
5.0%	4.8%	4.4%	5.3%	7

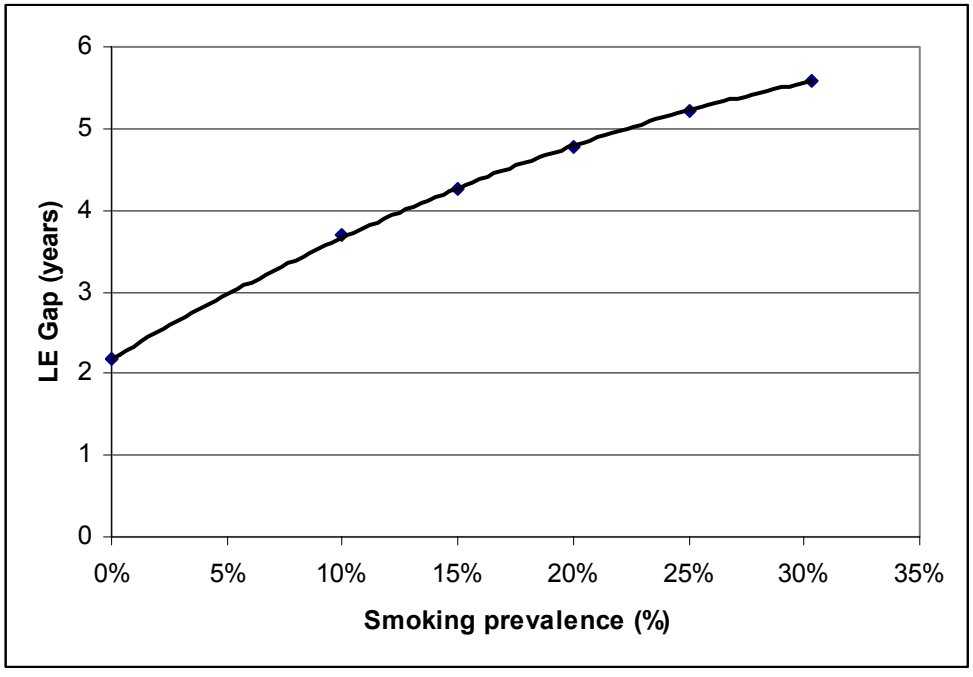
Male

		95% CI for PAR (%)		Estimated smoking
Prevalence	PAR (%)	Lower	Upper	attributable deaths in 2005
34.3%	26.5%	25.0%	28.1%	38
30.0%	24.0%	22.5%	25.5%	34
25.0%	20.8%	19.5%	22.2%	30
20.0%	17.4%	16.2%	18.6%	25
15.0%	13.6%	12.7%	14.6%	19
10.0%	9.5%	8.8%	10.2%	14

The small numbers of deaths attributable to smoking (using the rate ratios presented by Hunt in the 2005 Pacific population) translate to only a very modest shift in life expectancy gap over different counterfactual smoking prevalence estimates (Figure 13). A drop in Pacific smoking from the current prevalence of 30.3% to the NZ European prevalence of 20.0% was only associated with a reduction in the life expectancy gap of about one month, while a complete hypothetical absence of smoking (i.e. a scenario where Pacific smoking prevalence was historically zero while that for non-Maaori, non-Pacific remained constant) was associated only with a narrowing of the life expectancy gap of about five months.

The mortality estimates found using the rate ratios developed by Hunt et al are likely to have underestimated the total mortality attributable to smoking in the Pacific population in 2005. To start with, there were 31 deaths due to smoking-related lung disease in the Pacific population of CMDHB in 2005 and the vast majority of these were likely to have been due to smoking. It is also likely that at least some of the 80 Pacific deaths due to cardiovascular disease and some of the 60 deaths due to cancers (other than lung cancer) would have been attributable to smoking. A second set of analyses were therefore undertaken using the mortality rate ratios for all ethnicities (by sex) presented in the paper by Hunt et al [38]. The rate ratios for all-cause mortality in this model were 2.05 for males and 2.01 for females. In this second analysis, around 65-70 Pacific deaths in CMDHB were attributed to smoking, given that the smoking prevalence in Pacific peoples in 2006 was 30.3% (Table 6). In the counterfactual situation where the Pacific smoking prevalence dropped to 20.0%, smoking attributable mortality dropped by around 30% to 45-50 Pacific people in 2005.

Figure 14: Life expectancy gap between Pacific and non-Maaori, non-Pacific in CMDHB in 2005 in different counterfactual scenarios of adult smoking prevalence, using rate ratios for all-cause mortality in ALL ethnicities estimated by Hunt et al

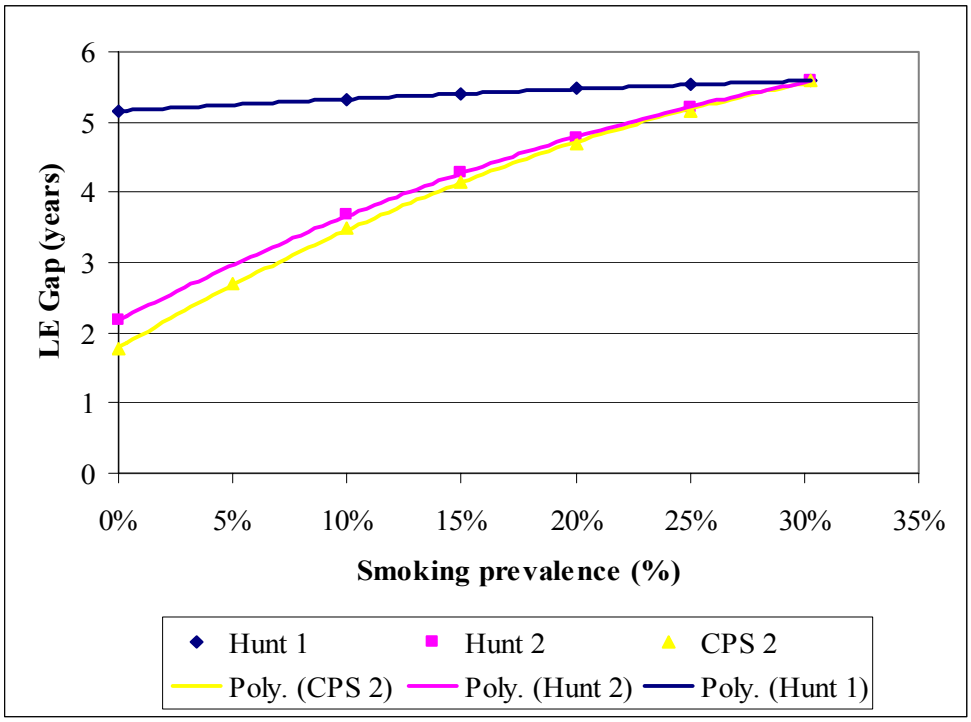


Using the all-cause mortality rate ratio estimates for all ethnicities in this second model, smoking prevalence had a much greater impact on the life expectancy gap

between Pacific and non-Maaori, non-Pacific than in the first model. A reduction in Pacific smoking prevalence from the 2006 estimate of 30.3% to a 20.0% prevalence scenario was associated with an almost ten month reduction in the life expectancy gap (Figure 14). In the counterfactual situation where the prevalence of smoking was zero in the Pacific population, the life expectancy gap narrowed by about 4.5 years to just over two years.

For comparison, all-cause mortality rate ratios for those aged ≥ 30 years in CPS II [37] were also used in a third scenario to examine the impact on life expectancy gap between Pacific and non-Maaori, non-Pacific of changing smoking prevalence. Results from this analysis can be seen in Figure 15. As expected, the analyses which used all-cause mortality rate ratios of about two (CPS II and the all ethnicities rate ratio from Hunt et al) generated similar life expectancy gap estimates, whereas the third curve which used a mortality risk estimate much closer to one is considerably more flat.

Figure 15: Predicted gap in LE at birth between Pacific and non-Maaori, non-Pacific in CMDHB in 2005 by counterfactual smoking prevalence for three distinct smoking risk scenarios



Discussion

This discussion examines the main findings of the analyses conducted in this report, including the strengths and limitations of this type of life table analysis. Since smoking was identified as a critical factor contributing to disparities in life expectancy in Counties Manukau, the impact of smoking on life expectancy is then reviewed in detail. Finally, recommendations for future population-level intervention are presented.

Equity in health is a notion that is routed in concepts of social justice and fairness [8, 41]. Returning to the quote by Whitehead in the introduction to this report [8], inequity between groups in number of expected years lived is a difference in health outcome that is unnecessary, avoidable, unfair and unjust. Explanations for health inequity are complex and typically include discussion of broader health determinants such as income and social status, health literacy and education, structure of social and physical environments, discrimination and access to health services. This report is somewhat superficial in that it does not attempt to explore the 'upstream' reasons behind the differences seen in life expectancy at birth between Maaori, Pacific and non-Maaori, non-Pacific peoples in Counties Manukau. Rather, it has examined the 'downstream' contributions of specific disease areas to life expectancy gaps. Such 'downstream' analysis is, however, significant in the DHB setting as it provides important planning information for the provision of health services and programmes aimed at reducing inequity.

While not a complete measure of the health status of populations, life expectancy at birth is never-the-less useful as it is readily understood by those without health training, the mortality input - a 'hard outcome' - is not easily open to question and life table analyses allow comparison between different populations without the need for an external standard. Substantial gaps in life expectancy at birth between Maaori, Pacific and non-Maaori, non-Pacific have been a consistent feature of the health landscape of both Counties Manukau and of the nation for a long time. This was the first study to separate out the absolute and proportional contributions of different causes of death to the overall disparities in life expectancy at birth between Maaori, Pacific and non-Maaori, non-Pacific in Counties Manukau. The year 2005 was chosen for this analysis, as this year was representative of trends in life expectancy in CMDHB in recent years and was also the year with the most recent coded mortality data.

In this report, simple 'league tables' of mortality were examined to identify ICD-10-AM principal cause of death codes with the greatest absolute contributions to mortality overall and to inequity. Codes were then grouped according to biological association into five disease areas. Abridged life tables were created for Maaori, Pacific peoples and those of non-Maaori, non-Pacific ethnicities in CMDHB in 2005. The total gap in life expectancy at birth was found to be about ten years for Maaori and 5.5 years for Pacific. The contribution of each disease area to life expectancy gaps was then analysed by substituting actual age-specific mortality with 'expected' counterfactual mortality based on rates for the non-Maaori, non-Pacific group. The proportional contribution of each of the five identified disease areas and of the remainder mortality to the ten- and 5.5-year gaps was then found.

The report *Tracking Disparity* used linked census and mortality data (NZCMS, the New Zealand Census-Mortality Study) to look at absolute and relative ethnic and socioeconomic inequity in mortality in those aged one to 74 years for five separate periods between 1981 and 2004 [10]. This report provides the most comprehensive and most recent published analysis of the contribution of various disease areas to mortality gaps by ethnicity nationally, for comparison with our findings. The authors found that Maaori had the highest mortality rates by age group of any ethnicity for the entire 1981 to 2004 period in New Zealand. Mortality rates for Maaori were followed in turn by Pacific peoples, then European/Other ethnicities and finally those of Asian ethnicities. However, gaps in mortality (both relative and absolute) between those of Maaori and Pacific ethnicities and those of European/Other ethnicities narrowed somewhat in the late 1990s and early 2000s nationally.

In *Tracking Disparity*, the contribution of different disease areas to absolute inequities in mortality by ethnicity was examined by looking at the percentage contribution of each cause-specific standardised rate difference (SRD) to the overall SRD [10]. As was the case in our Counties Manukau study, some minor adjustment/rescaling was necessary to account for differences between the sum of contributing causes and the all-cause SRD. Also, neither deaths in infants nor deaths in those aged ≥ 75 years were reported. Findings for those aged one to 74 years in *Tracking Disparity* have been recast along the lines of the specific disease areas used in this report, so that the percentage contribution of different causes of death to the absolute gap in mortality rates (SRD) between Maaori and European/Other in New Zealand in 2001-04 can be compared to the results for life expectancy disparity presented in the results section of this CMDHB report (Table 7).

Table 7: Percentage contribution of different causes of death to absolute gap in mortality rates (SRD) between Maaori and European/Other in New Zealand, 2001-04

Cause of death	Males	Females
Cardiovascular disease	41%	36%
Lung cancer and chronic lung disease	15%	26%
Non-lung cancer	14%	14%
Unintentional injury and suicide	9%	4%
Remainder causes	21%	20%
Total	100%	100%

Adapted from: Blakely T, et al (2007)

Aside from the obvious difference between the outcome measured in *Tracking Disparity* (mortality rate) and the outcome of life expectancy at birth used in this report, there were a number of other differences (such as exclusion of infant mortality) that made it difficult to compare the two studies. However, areas like cardiovascular disease, lung diseases such as those related to smoking and other (non-lung) cancers stood out as the major contributors to mortality disparity, as they did in the CMDHB study. Cardiovascular disease had the greatest contribution to absolute mortality differences in *Tracking Disparity*, accounting for around 40% of disparity in both sexes across the 1981 to 2004 period. In the CMDHB study, cardiovascular disease accounted for only 16% of the difference in life expectancy at birth between Maaori and non-Maaori, non-Pacific, but 35% of the gap for Pacific in 2005. The contribution of lung diseases associated with smoking to observed mortality and life

expectancy differentials appeared similar between the two studies. This report found a 20% contribution of smoking-related lung diseases to the life expectancy gap for Maaori and this contribution appears similar to that described by the aggregate category 'Lung cancer and chronic lung disease' in Table 7.

Contributing causes to the mortality gap between Pacific people aged one to 74 years and Europeans/Others in *Tracking Disparity* have also been recast in Table 8. Again, cardiovascular disease stood out as the major contributing factor to the mortality gap between Pacific and European/Other across the entire 1981 to 2004 period. In 2001-04, cardiovascular disease accounted for around half of the mortality gap in males and around 40% of the gap in females. In comparison, cardiovascular disease accounted for about 35% of the life expectancy gap between Pacific and non-Maaori, non-Pacific in the 2005 model used in this report. After cardiovascular disease, diabetes was the next largest contributor to the life expectancy gap in the CMDHB report (13%). Diabetes was included in the remainder group 'Other causes' in *Tracking Disparity*. 'Other causes' played a greater role in the mortality gap for Pacific than it did for Maaori (around 40% versus around 20%) and it is possible that diabetes mortality was an important component of 'Other causes' in the analysis for Pacific peoples in *Tracking Disparity*. Smoking-related lung disease was responsible for about nine percent of the CMDHB life expectancy gap in 2005, whereas a similar constellation of lung diseases was found to be responsible for three percent of the mortality gap in Pacific males and nine percent in Pacific females in *Tracking Disparity*.

Table 8: Percentage contribution of different causes of death to absolute gap in mortality rates (SRD) between Pacific and European/Other in New Zealand, 2001-04

Cause of death	Males	Females
Cardiovascular disease	48%	38%
Lung cancer and chronic lung disease	3%	9%
Non-lung cancer	10%	16%
Unintentional injury and suicide	3%	-3%
Remainder causes	37%	40%
Total	101%	100%

Adapted from: Blakely T, et al (2007)

In terms of absolute contribution to the life expectancy gap, smoking-related lung disease in Maaori in CMDHB contributed the greatest amount of time of any cause of death in either the Maaori or Pacific models in this study – 2.1 years. Five times as many Maaori died from such causes in 2005 as would have been expected based on mortality for non-Maaori, non-Pacific. Furthermore, smoking probably contributed to all of the other major disease areas to some extent. The prevalence of smoking among Maaori in CMDHB in 2006 was the highest of any ethnic group, and the prevalence of smoking in Maaori women was over 50% [42]. The contribution of smoking-related lung disease towards inequity in life expectancy was comparatively less among Pacific peoples in 2005, although lung diseases still accounted for around nine percent of the life expectancy gap and smoking was responsible for an unknown but potentially large share of deaths in other disease areas, like cardiovascular disease. Because of the magnitude of the contribution of diseases related to smoking to

inequity in life expectancy, smoking has been explored in much greater detail later in this discussion.

Deaths attributed to cardiovascular disease had the largest contribution to inequity in Pacific life expectancy and ranked second in contribution to the life expectancy gap in Maaori. This finding was consistent with national evidence that both cardiovascular mortality and the prevalence of cardiovascular disease are considerably higher in Maaori and Pacific populations than in non-Maaori, non-Pacific groups [10, 43]. A recent study of the burden of modifiable cardiovascular risk factors among 973 patients with acute cardiovascular events presenting to Middlemore Hospital in Counties Manukau found that Maaori and Pacific patients (and in particular younger Maaori and Pacific patients) were more likely to have a greater burden of modifiable risk factors for cardiovascular disease such as smoking and obesity than non-Maaori, non-Pacific patients [44]. These research findings and the findings of this report lend support to the implementation of robust population-level interventions targeting Maaori and Pacific and directed at primary and secondary prevention of modifiable cardiovascular disease risk factors. The findings suggest that successful implementation of such interventions is likely to have a positive influence on current ethnicity-based disparities in life expectancy.

Cancers other than lung cancer were found to be responsible for about 15% of the life expectancy gap for Maaori and six percent of the gap for Pacific. Maaori experience an unequal burden of cancer morbidity and mortality nationally. In the period 2000-04, the age- and sex-standardised cancer incidence rate (including lung cancer) was nine percent higher for Maaori than for non-Maaori in New Zealand, while the mortality rate was 77% higher for Maaori than for non-Maaori [12]. Maaori with cancer were less likely to be diagnosed at an early stage than non-Maaori for many cancers, a possible (partial) explanation for differences in mortality after diagnosis between Maaori and non-Maaori [12]. Blakely et al found that the standardised mortality rate ratios for non-lung cancer in Maaori (compared with European/Other) were 1.62 in males and 1.58 in females in New Zealand in 2001-04 [10]. In Pacific, national mortality rate ratios for non-lung cancer in 2001-04 were 1.26 and 1.32 for males and females respectively [10]. Non-lung cancers (and lung cancer) have been recognised as important components of potentially-avoidable mortality among Pacific peoples in Counties Manukau [27]. As reviewed later in this discussion, smoking is a risk factor for many cancers other than lung cancer. It is likely that differences in smoking prevalence in Counties Manukau between Maaori, Pacific and non-Maaori, non-Pacific populations have an important role in the contribution of non-lung cancers to inequity in life expectancy at birth. Further work needs to be undertaken to understand the role of smoking as a driver of non-lung cancer disparities in Counties Manukau and also to understand the other key drivers behind these disparities. Such analysis at a regional level is planned to commence in early 2009.

Deaths due to diabetes caused 13% of the life expectancy gap between Pacific and non-Maaori, non-Pacific and seven percent of the life expectancy gap between Maaori and non-Maaori, non-Pacific in 2005. Around 27,000 people in Counties Manukau were thought to have diabetes in 2008 and the prevalence of diabetes in Maaori and Pacific communities was much greater than that found among non-Maaori, non-Pacific populations [45]. Data from the 2006/07 NZHS paint an alarming picture of obesity in Maaori and Pacific populations in Counties Manukau, with around half of

Maaori adults and 80% of Pacific adults considered obese (BMI ≥ 30) in this survey [46, 47]. *Let's Beat Diabetes* (LBD) is a long-term, intersectoral strategy which draws on wide-ranging activities such as community-based programmes, social marketing, support for primary care and collaboration with the food industry, to prevent or delay the onset of type 2 diabetes, limit disease progression and improve quality of life for those with diabetes in Counties Manukau [48]. Extensive work has already been undertaken looking at the relationship between diabetes and health inequity in Counties Manukau through the LBD programme. This report does not seek to explore this relationship further.

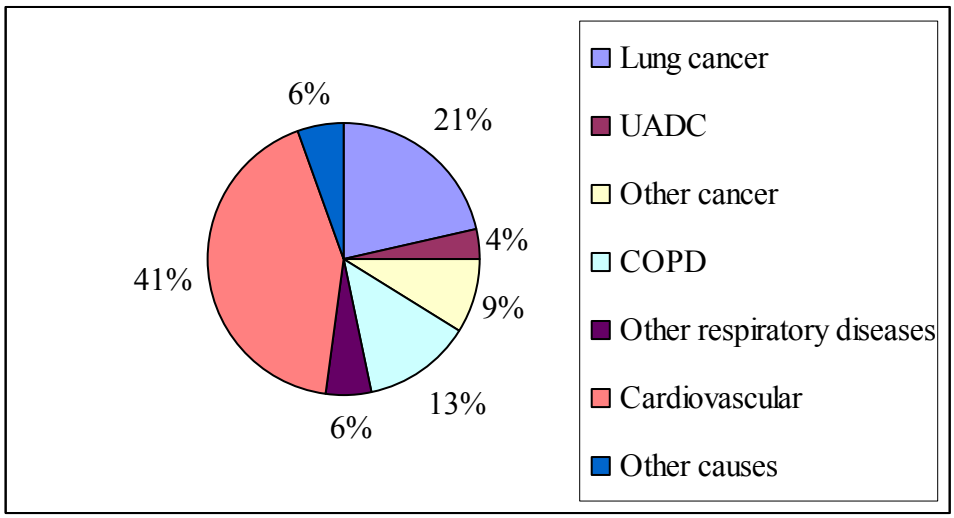
Infant mortality was responsible for around seven months of the life expectancy gap for Pacific and around six months of the life expectancy gap for Maaori. Infant mortality among Pacific peoples in Counties Manukau in 2003-05 was 9.7 per 1,000 for females and 8.5 per 1,000 for males, compared with 4.0 per 1,000 and 7.3 per 1,000 for non-Maaori, non-Pacific females and males [27]. At a national level, Maaori infant deaths in 2000-04 were 64% higher than those for non-Maaori [12]. The most common cause of death in neonates (i.e. less than one month old) in CMDHB in 2000-04 was extreme prematurity, while for those aged between one month and one year the most common cause of death was sudden infant death syndrome (SIDS or SUDI) [49]. Infant death numbers in 2005 were too small to undertake any meaningful analysis on the drivers of mortality. However, six of 24 deaths in Maaori infants were attributed to SIDS, compared with one of 22 for Pacific infants and none of 18 for non-Maaori, non-Pacific infants in CMDHB. These numbers are consistent with national data, which suggest that SIDS and conditions such as premature birth are more important to Maaori infant mortality than congenital abnormalities [12, 13, 50]. Maaori and Pacific ethnicities, deprivation and male sex were all noted to be risk factors for neonatal mortality resulting from extreme prematurity and other perinatal conditions nationally in 2000-04 [49]. Maaori and Pacific ethnicities and deprivation were found to be risk factors for infant mortality due to SIDS in this period, with the relative risk for infants of Maaori ethnicity being 6.3 compared with European infants [49]. Further work needs to be done to understand the drivers of infant mortality in Maaori and Pacific communities in Counties Manukau. However, based on local and national findings, initiatives to reduce mortality from SIDS in Maaori and Pacific populations appear likely to have an important influence on reducing infant mortality disparities.

Smoking and mortality

Smoking is an important cause of mortality. It has been ranked as the second leading cause of death behind dietary causes in New Zealand, accounting for around 18% of deaths in this country [51]. Smoking is also recognised as an important driver of health inequity in New Zealand [10, 52]. As presented in greater detail in the following sections, significant disparity exists in smoking prevalence by ethnicity in Counties Manukau and Maaori adults have a considerably higher smoking prevalence than other groups. Lung diseases related to smoking were identified as the biggest driver of the life expectancy gap between Maaori and non-Maaori, non-Pacific and as an important driver of the gap between Pacific peoples and non-Maaori, non-Pacific. However, the effects of smoking are not limited to lung disease. The contribution of different causes of death to overall mortality due to smoking in industrialised countries is shown in Figure 16, which uses estimates from the Global Burden of

Disease study [29]. Differences in smoking by ethnicity are likely to have an impact on all five disease areas used in the life expectancy models.

Figure 16: Distribution of mortality due to smoking by cause in industrialised countries, 2000



UADC Upper aerodigestive cancer
Adapted from: Ezzati et al (2004)

Around 20% (two years) of the life expectancy gap between Maaori and non-Maaori, non-Pacific was attributed to respiratory diseases caused by smoking – lung cancer and chronic pulmonary diseases such as emphysema and COPD. About nine percent (six months) of the difference in life expectancy between Pacific peoples and non-Maaori, non-Pacific was attributed to these lung diseases. Smokers have around a 16-fold increased risk of lung cancer compared with non-smokers and about a 12-fold increased risk of developing COPD [37]. In countries such as New Zealand where smoking is commonplace, smoking is responsible for around 90% of lung cancers of all types [36, 53, 54]. In New Zealand and Australia, the hazard ratio for mortality due to lung cancer in current smokers versus non-smokers is about ten [55]. Close to three quarters of all COPD deaths in the United States have been attributed to smoking [40] and the mortality ratio for COPD among heavy smokers (≥ 25 cigarettes per day) compared with non-smokers has been found to be as high as 32 [56]. It therefore appears reasonable to attribute a substantial amount of the smoking-related lung disease mortality difference between Maaori and Pacific peoples and those of other ethnicities to smoking.

There is a causal relationship between household smoking and the risk of SIDS (SUDI), which is independent of the effects of maternal smoking during pregnancy [40, 57]. Dose-response relationships have been noted between risk of SIDS and both the number of cigarettes smoked and number of smokers in a household, together with the overall duration of exposure to second-hand smoke [40]. The higher prevalence of smoking in Maaori and Pacific communities, together with greater housing density among Maaori and Pacific families, means that exposure to second-hand smoke is likely to be more common among Maaori and Pacific infants than those of other ethnicities in CMDHB. This difference in smoke exposure is likely to contribute to the greater mortality seen among Maaori and Pacific infants, thereby widening disparities in life expectancy.

Smoking is widely acknowledged as an important contributor to the burden of cardiovascular disease. The British Doctors' Study reported that male (heavy) smokers had around twice the risk of death from coronary heart disease compared with non-smokers after 40 years of follow up [58]. The Nurses Health Study found a relative risk for death from IHD among current smokers of over four, and risk increased with the number of cigarettes smoked each day [59, 60]. Smoking is also an important risk factor for other forms of cardiovascular disease, such as peripheral vascular disease and cerebrovascular disease (stroke) [40]. Maaori have greater exposure to second-hand smoke [61, 62] and exposure to second-hand smoke is associated with an increased risk of cardiovascular disease among non-smokers. A meta-analysis of 18 studies showed that non-smokers who had been exposed to second-hand smoke had around a 25% increased risk of IHD compared with non-smokers with no passive smoking exposure [63]. The implication is that differences in smoking prevalence between Maaori, Pacific and non-Maaori, non-Pacific have an important influence on the differences seen in cardiovascular mortality between these groups and therefore on the life expectancy disparities seen.

In addition to the strong causal relationship between smoking and lung cancer, cigarette smoking is associated with death from a wide range of other malignancies. Table 9 shows the relative risk of death from several different cancer types attributed to smoking in the SAMMEC models used by the US Centers for Disease Control and Prevention [33]. Exposure to second-hand smoke in non-smokers is also associated with malignancies such as sinus cancer [40]. All of the cancer types listed in Table 9 (except lung cancer) feed into mortality data for Area four in the life expectancy gap models. The differences in smoking prevalence in CMDHB between Maaori, Pacific and non-Maaori, non-Pacific are therefore likely to contribute to the life expectancy gap of 1.5 years for Maaori and four months seen for Pacific in 2005.

Table 9: Relative risk of death from various cancer types in smokers vs. non-smokers

Cancer type	ICD-10 code	Male		Female	
		Current smoker	Former smoker	Current smoker	Former smoker
Lip, oral cavity, pharynx	C00-C14	10.89	3.40	5.08	2.29
Oesophagus	C15	6.76	4.46	7.75	2.79
Stomach	C16	1.96	1.47	1.36	1.32
Pancreas	C25	2.31	1.15	2.25	1.55
Larynx	C32	14.60	6.34	13.02	5.16
Trachea, lung, bronchus	C33-C34	23.26	8.70	12.69	4.53
Cervix uteri	C53	0.00	0.00	1.59	1.14
Kidney, renal pelvis	C64-C65	2.72	1.73	1.29	1.05
Urinary bladder	C67	3.27	2.09	2.22	1.89
Acute myeloid leukaemia	C92.0	1.86	1.33	1.13	1.38

Adapted from: US Centers for Disease Control and Prevention (2008)

There is a less direct link between smoking and death from diabetes (Area five). The combination of smoking and diabetes mellitus is known to substantially enhance risk of micro- and macrovascular disease, and smoking is known to be a significant independent risk factor for all-cause mortality in those with diabetes [64, 65]. As well

as an acknowledged effect on cardiovascular disease in diabetes, smoking is associated with worse outcomes for renal disease and neuropathy (nerve damage) [66-68]. Data relating ethnicity to smoking in those with diabetes is not currently available for CMDHB. However, it is possible that existing disparities in smoking prevalence by ethnicity may carry through to the diabetes population in CMDHB, and this would then have the effect of further widening the mortality gap for diabetes.

Smoking prevalence in CMDHB

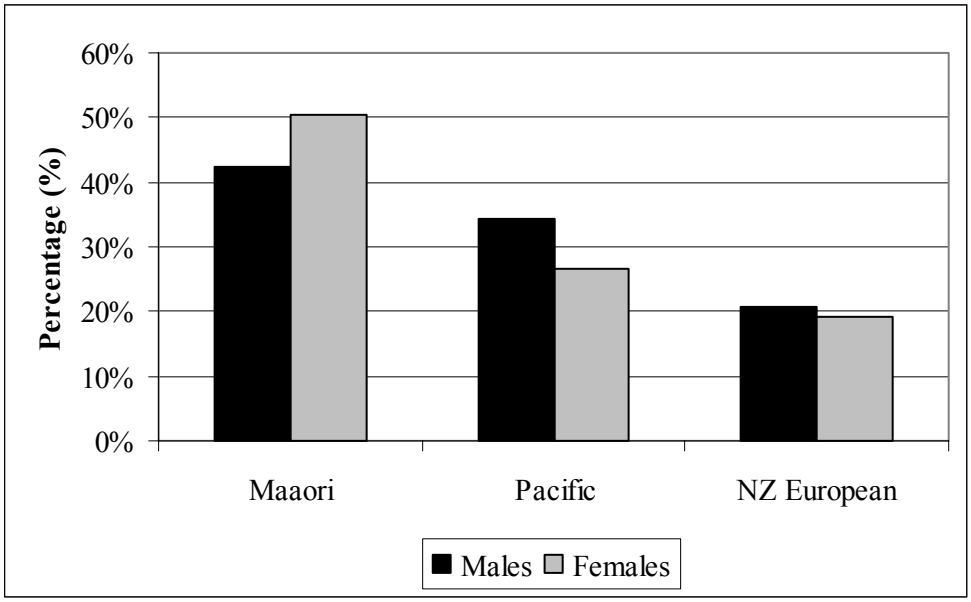
The 2006/07 New Zealand Health Survey (NZHS) estimated that 21.0% of adults (aged ≥ 15 years) in CMDHB were current smokers, or around 67,700 people [47]. This estimate was similar to that found in the 2006 national census, where 22.1% of adults in CMDHB were found to be regular smokers [42].

Table 10: Crude prevalence of regular smoking among adults (age ≥ 15 years) in CMDHB, by ethnicity in 2006

	Males	Females	Total	NZ Total
Maaori	42.5%	50.3%	46.8%	42.2%
Pacific	34.3%	26.7%	30.3%	30.3%
NZ European	20.8%	19.3%	20.0%	19.4%
Asian	16.3%	3.4%	9.6%	11.1%
MELAA	22.0%	8.9%	15.5%	15.1%
Other ethnicity	16.6%	15.4%	16.0%	16.6%
Total	24.0%	20.4%	22.1%	20.7%

Data source: Ministry of Health (2008)

Figure 17: Prevalence of regular smoking among Maaori, Pacific and NZ European adults (≥ 15 years) in CMDHB, 2006 (unadjusted)



Data source: Ministry of Health (2008)

Considerable inequity is noted in smoking prevalence by ethnicity (Table 10 and Figure 17). Maaori women had the highest regular smoking prevalence of any group, with over half of Maaori women smoking regularly. This was considerably higher

than women in any other ethnic group and was almost twice as high as the next highest prevalence of regular smoking (Pacific women). Overall, smoking prevalence among Maaori and Pacific peoples was 2.3 and 1.5 times that of NZ European adults respectively.

Contribution of smoking to mortality and inequity nationally

Ezzati et al used data from the American Cancer Society's Cancer Prevention Study (CPS) II, the WHO Global Burden of Disease Database and a retrospective study of one million deaths in China to estimate the proportion of mortality attributable to smoking as part of the Global Burden of Disease (GBD) study [39]. Mortality attributable to smoking in industrialised countries like New Zealand is presented in Table 11.

Table 11: Attributable total mortality (%) in industrialised countries due to smoking, 2000

	Male	Female
Age 30-69 years	33%	12%
Age ≥ 70 years	24%	9%

In the 2001 Ministry of Health report, *Inhaling Inequality*, around one third of all Maaori deaths were attributable to tobacco smoking [69]. This report estimated that about a quarter of the gap in life expectancy between Maaori and non-Maaori, non-Pacific in 1995-97 was due to smoking. However, the results of the report have since been questioned [70, 71]. It is thought that the contribution of smoking to the life expectancy gap was overestimated in *Inhaling Inequality*, as relative risks for smoking mortality have subsequently been revised to take account of ethnicity [38]. Also, Maaori lung cancer mortality rates are higher than those expected from smoking alone, indicating that the method used in the report (which used lung cancer mortality as an indicator of accumulated smoking hazard in population comparisons – see Appendix Four, point number seven) may have further overestimated smoking-related mortality burden in Maaori [71].

Tobias looked at disability adjusted life years (DALYs) lost which were attributable to smoking in New Zealand, in the report *The Burden of Disease and Injury in New Zealand* [31]. This report found that smoking accounted for about 15% of DALYs lost in males and around 9% of DALYs lost in females. Tobacco had the greatest contribution to DALYs lost of any of the eight major risk factors analysed in both males and females. However, as with *Inhaling Inequality,* Tobias applied risk ratios from CPS II to the New Zealand context and more suitable local risk estimates for the association between smoking and mortality have subsequently been developed [38].

Blakely et al used New Zealand Census-Mortality Study (NZCMS) data to examine the contribution of tobacco smoking and socioeconomic position to ethnic inequalities in mortality in New Zealand [70]. They found that smoking had only a small apparent contribution to mortality differences between Maaori and non-Maaori, non-Pacific in 1981-84 and 1996-99. They concluded that smoking accounted for somewhere between a negligible contribution and around ten percent of the mortality gap between Maaori and non-Maaori, non-Pacific (eight percent for women and five percent for men). The contribution of smoking to the mortality gap in women was greater for each of the two cohorts studied. The authors noted that the overall contribution of

smoking to the mortality gap was likely to be rising, in line with diverging trends in smoking prevalence by ethnicity [72]. They also noted that the 0-10% contribution seemed surprisingly low. However, the discussion reviewed the various factors that may have affected the estimates (e.g. differential misclassification of smoking status) and concluded that the findings represented reasonable estimates of the contribution of smoking to ethnic mortality gaps.

Wilson et al synthesised epidemiological data (such as that used in the NZCMS study by Blakely et al, above) related to smoking and health inequalities to look at socioeconomic- and ethnicity-based mortality gaps in New Zealand [70, 71, 73, 74]. Using NZCMS data for 45-74 year olds in 1996-99, they estimated that around one quarter of mortality in the population was attributable to smoking (i.e. would have been eliminated if all current and ex-smokers had been never smokers in a counterfactual scenario). For Maaori males, the population attributable risk percent for smoking in this analysis was 17%, while for Maaori females it was 25% (vs. 28% and 25% for non-Maaori, non-Pacific males and females respectively). They found that although the proportional contribution of smoking to the mortality gap between Maaori and non-Maaori, non-Pacific was quite low in the study by Blakely et al (above), the *absolute* mortality attributable to smoking in Maaori was substantially greater than that in non-Maaori, non-Pacific people.

In a further analysis of the contribution of smoking to inequalities in mortality by education using NZCMS data for those aged 45-74 years in 1996-99, Blakely and Wilson conclude that if all current smokers stopped (and became ex-smokers), the overall mortality rate for men would be expected to fall by around 11%, while for women it would drop by about 5% [74]. In the counterfactual situation where smoking had historically been absent from New Zealand society, mortality rates would drop by about 26% for men and 25% for women (as described in Wilson et al [above]).

Blakely et al summarise the contribution of tobacco smoking to inequalities in ethnic mortality in the report *Tracking Disparity* [10]. They estimate that tobacco contributes to around ten percent to 20% of ethnic inequalities in mortality. This estimate is based on the various analyses that have used NZCMS data to examine the relationship between smoking and mortality by ethnicity in New Zealand [70, 71, 73, 74].

Strengths and limitations

The key strength of this study was that it was able to synthesise complex mortality data into outputs – life expectancy estimates – easily understood by those without training in medical and health sciences. It was able to show the main contributors to life expectancy inequity in terms of simple percentage contributions to the total gaps and simple year/month time measures. Major strengths and limitations of this study are presented in Table 12.

ICD-10-AM codes for principal cause of death were used to group absolute mortality numbers (by age and ethnicity) in 2005 into the five major disease groupings and the remainder groups. Clinical coders undertake a rigorous process of assigning principal cause of death codes to each death in New Zealand [23, 24]. This means that data related to cause of death were likely to be accurate. It also means that classification of ethnicity was likely to be very accurate in comparison to other sources of data such as

NMDS. However, although classification of cause of death was likely to be quite accurate, in many cases that there would have been several contributing causes (not only the cause given in the principal cause of death code). The complexity and multiplicity of causes of death has not been captured in these analyses and this may have lead to over-simplification of the analyses and of the contribution of each disease area to mortality.

Absolute mortality figures are the only input into the life tables used in this report and are therefore the only determinants of the estimates used to explore inequity between Maaori, Pacific and non-Maaori, non-Pacific. Mortality differences by ethnicity only tell part of the inequity story and a more complete picture could be gained by including a morbidity component in the analyses. However, mortality by age group is still a useful measure of health status of populations and can provide helpful narratives describing health inequity. It is a 'hard' outcome, meaning that there is little debate about accuracy of measurement or health importance of the endpoint [75]. Furthermore, there is strong correlation between mortality and morbidity, meaning that a description of mortality can give some insight into morbidity [76].

A systematic approach was used to aggregate specific ICD-10-AM cause of death codes into the five broad disease areas. This approach involved review of mortality data for 2005 and ranking of individual codes by absolute magnitude of mortality and by the extent of mortality inequity by ethnicity for these codes. Codes which had the greatest contribution to mortality and inequity were grouped according to biological association. These disease groupings were then discussed with Maaori and Pacific teams to ensure consistency with current priorities for these teams. This means that disease areas were grouped in a way that had local significance and relevance for Counties Manukau (rather than simply using generic disease groupings such as those developed by WHO).

Table 12: Strengths and weaknesses of study

Strengths	Weaknesses
• Life expectancy is an easy concept for people without health sciences training to understand • Mortality is a 'hard' endpoint • Ethnicity classification in Mortality Collection is likely to be accurate • All age groups included in analyses • Contributions to life expectancy gaps could be dissected into simple percentages and time measures • Aggregation of disease areas into classifications relevant to local needs	• Use of mortality alone does not account for differences in morbidity and provides an incomplete picture of inequities in health • Only data for single year (2005) used in analyses • Only principal cause of death ICD-10-AM codes used in analyses • Number of deaths by ethnicity, age and cause small • Numbers too small to break down by gender and specific Pacific ethnicity

The Chiang II abridged life table has been validated in small populations, down to about 5,000 people [5, 22]. The life tables used in this analysis had 75,000 people for Maaori, 92,000 people for Pacific and 276,000 people for non-Maaori, non-Pacific. However, death is still a relatively uncommon outcome. When broken down into groups of causes, some of the numbers of deaths became fairly small (the smallest

group was deaths due to diabetes in Maaori – 19 people). Sensitivity analyses on the models showed that changes in small numbers in individual age groups did not have a particularly large impact on life expectancy at birth in the models. However, it should be remembered that the models were intended to be used as a guide to the major drivers of mortality and therefore life expectancy in Maaori and Pacific communities, rather than to provide exact estimates of these measures. Numbers of deaths were also too small to disassociate death numbers further by sex and by specific Pacific ethnicity.

The rationale behind the choice of the year 2005 for this study has been given elsewhere. A potential concern is that the contributions of specific disease areas in 2005 may not have been representative of the magnitude of these disease areas in the longer term. Life expectancy estimates in 2005 were generally representative of trends in life expectancy at birth in Counties Manukau in previous years. Also, trends in absolute mortality and mortality rates by age group within specific disease areas were observed for the years leading up to 2005, to ensure that death numbers used in the models were broadly similar to those for 2005. However, as noted earlier, the life expectancy gap for Pacific peoples in 2005 was the least of any year in the previous ten years.

There are a number of limitations and assumptions sitting behind the simplified models for smoking prevalence and life expectancy. These are documented in Appendix Four. Of particular importance in interpretation of the models is that they are static, meaning they do not include a time component. They assume that any impact of changing smoking prevalence is reflected in an immediate reduction in the life expectancy gap. This assumption is not supported by evidence from medical and health sciences literature, but for reasons of pragmatism is necessary in this case. Any impact of a reduction in smoking prevalence on mortality is likely to be slow and probably occur over several years. An analysis of the predicted impact of achieving smoking reduction goals in the US on mortality found substantial delay in benefit [34]. When looking at smoking initiation, a benefit in mortality was predicted to be seen after 35 years, while only one quarter of the benefit in mortality reduction would be seen in the following 70 years. Increasing cessation showed benefits to life expectancy sooner than those of initiation, but there was still a delay of many years. Other authors report more immediate effects on mortality of reducing smoking prevalence, especially in relation to cessation [40, 77]. Russell et al reported that complete cessation of smoking among US adults in the early 1990s would result in a 15% mortality reduction after five years and an 11% reduction after 20 years [77]. In developing the UK Health Inequalities Intervention Tool, the London Health Observatory estimates that any interventions to increase smoking cessation would take at least five years to have any effect on life expectancy [78, 79]. It therefore seems reasonable to assume that the lead time before any benefits of population-level smoking interventions in Counties Manukau become apparent would be around five years.

Conclusions and recommendations

The following conclusions and recommendations have been drawn from this analysis:

- Substantial inequity in life expectancy at birth exists in CMDHB between Maaori, Pacific peoples and those of non-Maaori, non-Pacific origins – around ten years for Maaori and 5.5 years for Pacific peoples

- Mortality data using ICD-10-AM cause of death codes can be separated out and the main causes of death can be analysed to look at absolute and proportional contributions to life expectancy

- Lung diseases related to smoking, cardiovascular diseases, (non-lung) cancer, diabetes and infant mortality were found to be the main causes of death that contributed to inequity in life expectancy between Maaori, Pacific and non-Maaori, non-Pacific

- Smoking was found to be the 'big ticket item', contributing not only to smoking-related lung diseases such as lung cancer and COPD, but to all five disease areas identified. The association between smoking prevalence and disparity in life expectancy was therefore examined in greater detail in models which used counterfactual smoking prevalence estimates to predict life expectancy gaps:
    - Differences in smoking frequency accounted for at least 10-20% of the life expectancy gap between Maaori and non-Maaori, non-Pacific in CMDHB
    - Differences in smoking frequency between Maaori and non-Maaori, non-Pacific in 2005 were responsible for at least 2-15% of the life expectancy gap for Pacific

- This report recommends that population-level interventions aimed at reducing the high frequency of smoking in Maaori and Pacific populations in Counties Manukau are a high priority, if inequity in life expectancy is to be addressed. Any population-level interventions to reduce gaps in smoking-associated mortality by ethnicity must recognise that:

    - Reasonably large reductions in smoking prevalence will need to occur before significant changes are seen in life expectancy (and life expectancy gaps). For example, a reduction in smoking prevalence of about seven percentage points in Maaori (from the current prevalence of 46.8% to 40.0% - a 15% shift in smoking prevalence) would be associated with about a 3-5 month narrowing in the life expectancy gap. The models in this report can be used to predict the approximate life expectancy changes expected at given adult smoking prevalence levels
    - Initiatives directed at reducing the burden of smoking will not affect life expectancy immediately. This is particularly relevant for strategies designed to reduce initiation (which will not affect life expectancy until the current teenage cohort reaches middle life). For smoking cessation, effects will be seen sooner but will still not become apparent for several years
    - The smoking models in this report assume that smoking prevalence in non-Maaori, non-Pacific will remain constant. This is unlikely, especially if

increased population-level effort is directed at reducing smoking prevalence. In fact, such interventions may have a greater affect on non-Maaori, non-Pacific smoking prevalence. The result will be a dilution of the effect of these efforts on life expectancy gaps

- Cardiovascular disease and diabetes both had important contributions to life expectancy disparity in Counties Manukau in 2005. Smoking is an important risk factor for cardiovascular disease and is known to worsen outcomes in those with diabetes. Efforts to reduce smoking should therefore have some effect on these areas. This report also supports the implementation and continuation of other programmes aimed at primary (before cardiovascular disease has been diagnosed) and secondary (reducing the impact of cardiovascular disease after diagnosis) prevention of cardiovascular disease and diabetes in Counties Manukau

- Mortality differences due to non-lung cancer were important components of life expectancy disparities in 2005. Again, smoking is likely to have a role in the causal pathway of some of these cancers. However, many other factors (such as social, environmental and health service access factors) probably contribute to the differences observed. This report recommends further work examining the drivers behind cancer mortality differences by ethnicity observed in Counties Manukau

- Infant mortality is another driver of life expectancy gaps in Counties Manukau. Additional analysis is required to tease out the main influences on infant mortality in Maaori and Pacific communities in CMDHB. However, initiatives to reduce mortality from SIDS in Maaori and Pacific populations appear likely to have an important influence on reducing infant mortality disparities

- Life expectancy appears to be a useful measure for target setting and strategic monitoring in CMDHB

Appendix one: Mortality league tables

Table: Causes of child and youth (0-14 years) mortality 2001-2005

Cause of death	Mortality rate per 100,000			Difference in rate	
	Maaori	Pacific	Other (reference)	Maaori vs. Other	Pacific vs. Other
P07 Disorders related to short gestation and low birth weight, not elsewhere classified	15.4	13.3	3.5	**11.9**	**9.8**
R95 Sudden infant death syndrome	22.1	4.0	1.2	**20.9**	2.8
W75 Accidental suffocation and strangulation in bed	8.1	2.7	0.4	**7.7**	2.3
P21 Birth asphyxia	1.5	4.0	1.5	0.0	2.5
V03 Pedestrian injured in collision with car, pick-up truck or van	5.1	2.7	0.4	4.7	2.3
P29 Cardiovascular disorders originating in the perinatal period	2.2	2.7	0.8	1.4	1.9
P28 Other respiratory conditions originating in the perinatal period	2.9	2.7	0.0	2.9	2.7
P91 Other disturbances of cerebral status of newborn	1.5	1.3	1.5	0.0	-0.2
Q04 Other congenital malformations of brain	0.7	2.0	1.2	-0.5	0.8
Q79 Congenital malformations of the musculoskeletal system, not elsewhere classified	0.7	2.0	1.2	-0.5	0.8
R99 Other ill-defined and unspecified causes of mortality	2.9	2.0	0.0	2.9	2.0
J18 Pneumonia, organism unspecified	1.5	2.0	0.4	1.1	1.6
P27 Chronic respiratory disease originating in the perinatal period	0.7	2.0	0.4	0.3	1.6
P36 Bacterial sepsis of newborn	0.0	2.0	0.8	-0.8	1.2
Q21 Congenital malformations of cardiac septa	0.7	1.3	0.8	-0.1	0.5
Q87 Other specified congenital malformation syndromes affecting multiple systems	0.0	3.3	0.0	0.0	3.3
Q91 Edwards' syndrome and Patau's syndrome	0.7	2.0	0.4	0.3	1.6
Total	113.3	86.7	40.6	**72.7**	**46.1**

Table: Causes of mortality in adults aged 15-64 years, 2001-2005

Cause of death	Mortality rate per 100,000			Difference in rate	
	Maaori	Pacific	Other (reference)	Maaori vs. Other	Pacific vs. Other
I25 Chronic ischaemic heart disease	37.8	32.7	13.2	**24.6**	**19.5**
C34 Malignant neoplasm of bronchus and lung	38.7	13.9	11.9	**26.8**	2.0
I21 Acute myocardial infarction	19.6	13.5	8.1	**11.5**	**5.4**
X70 Intentional self-harm by hanging, strangulation and suffocation	24.7	10.0	6.3	**18.4**	3.7
C50 Malignant neoplasm of breast	13.5	10.4	8.5	5.0	1.9
E11 Non-insulin-dependent diabetes mellitus	22.8	22.7	2.6	**20.2**	**20.1**
I42 Cardiomyopathy	11.2	6.9	3.8	**7.4**	3.1
C18 Malignant neoplasm of colon	0.9	2.7	5.9	-5.0	-3.2
J44 Other chronic obstructive pulmonary disease	12.6	4.2	2.5	**10.1**	1.7
C16 Malignant neoplasm of stomach	8.9	5.0	3.0	**5.9**	2.0
C71 Malignant neoplasm of brain	1.4	3.1	4.7	-3.3	-1.6
I61 Intracerebral haemorrhage	4.7	8.1	2.0	2.7	**6.1**
V43 Car occupant injured in collision with car, pick-up truck or van	6.1	3.5	3.0	3.1	0.5
C80 Malignant neoplasm without specification of site	3.3	1.9	3.6	-0.3	-1.7
E66 Obesity	4.7	10.0	0.9	3.8	**9.1**
I60 Subarachnoid haemorrhage	6.5	2.7	2.5	4.0	0.2
C43 Malignant melanoma of skin	0.5	0.8	4.2	-3.7	-3.4
X67 Intentional self-poisoning by and exposure to other gases and vapours	1.9	0.0	3.9	-2.0	-3.9
C22 Malignant neoplasm of liver and intrahepatic bile ducts	5.6	4.6	1.6	4.0	3.0
C25 Malignant neoplasm of pancreas	4.2	1.5	2.8	1.4	-1.3
C56 Malignant neoplasm of ovary	3.3	2.7	2.4	0.9	0.3
C20 Malignant neoplasm of rectum	1.9	4.6	1.9	0.0	2.7
I71 Aortic aneurysm and dissection	4.7	3.5	1.5	3.2	2.0
I08 Multiple valve diseases	5.6	4.2	0.6	5.0	3.6
C53 Malignant neoplasm of cervix uteri	3.7	3.1	1.2	2.5	1.9
Total	383.3	276.0	169.8	**213.5**	**106.2**

Table: Causes of mortality in adults aged 65-74 years, 2001-2005

Cause of death	Mortality rate per 100,000			Difference in rate	
	Maaori	Pacific	Other (reference)	Maaori vs. Other	Pacific vs. Other
I25 Chronic ischaemic heart disease	289.7	244.8	158.4	**131.3**	**86.4**
C34 Malignant neoplasm of bronchus and lung	524.1	288.5	131.8	**392.3**	**156.7**
I21 Acute myocardial infarction	289.7	253.5	146.8	**142.9**	**106.7**
J44 Other chronic obstructive pulmonary disease	427.6	192.3	97.1	**330.5**	**95.2**
E11 Non-insulin-dependent diabetes mellitus	496.6	332.2	33.5	**463.1**	**298.7**
C18 Malignant neoplasm of colon	82.8	62.2	76.3	6.5	-14.1
C61 Malignant neoplasm of prostate	69.0	52.4	53.2	**15.8**	-0.8
C80 Malignant neoplasm without specification of site	55.2	78.7	32.4	**22.8**	**46.3**
I71 Aortic aneurysm and dissection	69.0	87.4	28.9	**40.1**	**58.5**
C25 Malignant neoplasm of pancreas	41.4	26.2	38.2	3.2	-12.0
I61 Intracerebral haemorrhage	41.4	78.7	27.7	13.7	**51.0**
C16 Malignant neoplasm of stomach	82.8	69.9	24.3	**58.5**	**45.6**
C50 Malignant neoplasm of breast	41.4	35.0	32.4	9.0	2.6
C20 Malignant neoplasm of rectum	41.4	17.5	31.2	10.2	-13.7
C43 Malignant melanoma of skin	13.8	0.0	34.7	-20.9	-34.7
I64 Stroke, not specified as haemorrhage or infarction	27.6	61.2	23.1	4.5	**38.1**
C71 Malignant neoplasm of brain	13.8	8.7	27.7	-13.9	-19.0
C22 Malignant neoplasm of liver and intrahepatic bile ducts	0.0	104.9	12.7	-12.7	**92.2**
I69 Sequelae of cerebrovascular disease	69.0	78.7	9.2	**59.8**	**69.5**
C64 Malignant neoplasm of kidney, except renal pelvis	69.0	26.2	15.0	**54.0**	11.2
Total	3806.9	3208.0	1575.9	**2231.0**	**1632.1**

Table: Causes of mortality in adults aged ≥ 74 years, 2001-2005

Cause of death	Mortality rate per 100,000			Difference in rate	
	Maaori	Pacific	Other (reference)	Maaori vs. Other	Pacific vs. Other
I25 Chronic ischaemic heart disease	751.2	817.5	937.5	-186.3	-120.0
I21 Acute myocardial infarction	704.2	931.6	905.4	-201.2	**26.2**
J44 Other chronic obstructive pulmonary disease	845.1	589.4	484.8	**360.3**	**104.6**
I64 Stroke, not specified as haemorrhage or infarction	140.8	323.2	443.9	-303.1	-120.7
C34 Malignant neoplasm of bronchus and lung	845.1	399.2	249.7	**595.4**	**149.5**
E11 Non-insulin-dependent diabetes mellitus	610.3	665.4	150.4	**459.9**	**515.0**
G30 Alzheimer's disease	234.7	38.0	163.6	**71.1**	-125.6
C18 Malignant neoplasm of colon	140.8	95.1	160.6	-19.8	-65.5
C61 Malignant neoplasm of prostate	140.8	266.2	147.5	-6.7	**118.7**
I63 Cerebral infarction	328.6	152.1	143.1	**185.5**	9.0
F03 Unspecified dementia	93.9	133.1	141.6	-47.7	-8.5
I69 Sequelae of cerebrovascular disease	140.8	399.2	113.9	**26.9**	**285.3**
I50 Heart failure	140.8	228.1	118.3	**22.5**	**109.8**
J18 Pneumonia, organism unspecified	46.9	114.1	124.1	-77.2	-10.0
I61 Intracerebral haemorrhage	0.0	209.1	108.1	-108.1	**101.0**
I71 Aortic aneurysm and dissection	187.8	171.1	103.7	**84.1**	**67.4**
I35 Nonrheumatic aortic valve disorders	0.0	38.0	116.8	-116.8	-78.8
C80 Malignant neoplasm without specification of site	234.7	76.0	86.2	**148.5**	-10.2
C16 Malignant neoplasm of stomach	140.8	171.1	75.9	**64.9**	**95.2**
Total	8591.5	8270.0	6961.2	**1630.3**	**1308.8**

Appendix two: Sample life table for Maaori, CMDHB 2005 (Chiang II method)

	x	n	a_x	*pop*	*death*	M_x	q_x	p_x	l_x	d_x	L_x	T_x	e_x	$var(q_x)$	$[(1-a_x)n_1+e_{x+1}]^2 var(q_x(1-a_x)n_1+e_{x+1}]^2$	$Var(e_x)$	*SE*	Lower 95% confidence interval	Upper 95% confidence interval
<1	0	1	0.1	2148	**24**	0.011173	0.0110619	0.988938	100000	1106	99004	7208076	72.08	0.000005042	267125663	5715436213	0.57154	0.7560	70.60	73.56
1-4	1	4	0.5	7730	**1**	0.000129	0.0005173	0.999483	98894	51	395473	7109071	71.89	0.000000267	12790233	5448310551	0.55709	0.7464	70.42	73.35
5-9	5	5	0.5	9196	**3**	0.000326	0.0016298	0.998370	98843	161	493810	6713598	67.92	0.000000884	37085238	5435520318	0.55636	0.7459	66.46	69.38
10-14	10	5	0.5	8648	**1**	0.000116	0.0005780	0.999422	98682	57	493265	6219788	63.03	0.000000334	11926305	5398435080	0.55437	0.7446	61.57	64.49
15-19	15	5	0.5	7672	**4**	0.000521	0.0026035	0.997397	98625	257	492481	5726523	58.06	0.000001690	51019775	5386508775	0.55378	0.7442	56.61	59.52
20-24	20	5	0.5	6034	**9**	0.001492	0.0074300	0.992570	98368	731	490012	5234042	53.21	0.000006088	153763121	5335489000	0.55140	0.7426	51.75	54.66
25-29	25	5	0.5	5368	**4**	0.000745	0.0037189	0.996281	97637	363	487277	4744030	48.59	0.000003445	70273180	5181725879	0.54356	0.7373	47.14	50.03
30-34	30	5	0.5	5352	**10**	0.001868	0.0092989	0.990701	97274	905	484108	4256754	43.76	0.000008566	140597900	5111452699	0.54020	0.7350	42.32	45.20
35-39	35	5	0.5	5274	**14**	0.002655	0.0131852	0.986815	96369	1271	478670	3772646	39.15	0.000012254	156956841	4970854799	0.53525	0.7316	37.71	40.58
40-44	40	5	0.5	4640	**24**	0.005172	0.0255319	0.974468	95099	2428	469423	3293977	34.64	0.000026468	260351490	4813897958	0.53229	0.7296	33.21	36.07
45-49	45	5	0.5	3770	**17**	0.004509	0.0222951	0.977705	92671	2066	458187	2824554	30.48	0.000028588	201059895	4553546468	0.53023	0.7282	29.05	31.91
50-54	50	5	0.5	2948	**23**	0.007802	0.0382632	0.961737	90604	3467	444355	2366366	26.12	0.000061220	303072810	4352486573	0.53020	0.7281	24.69	27.54
55-59	55	5	0.5	2260	**24**	0.010619	0.0517241	0.948276	87138	4507	424420	1922011	22.06	0.000105709	341401161	4049413763	0.53331	0.7303	20.63	23.49
60-64	60	5	0.5	1506	**41**	0.027224	0.1274479	0.872552	82631	10531	386825	1497591	18.12	0.000345679	756750515	3708012602	0.54308	0.7369	16.68	19.57
65-69	65	5	0.5	1048	**32**	0.030534	0.1418440	0.858156	72099	10227	334930	1110766	15.41	0.000539558	634387551	2951262087	0.56773	0.7535	13.93	16.88
70-74	70	5	0.5	594	**23**	0.038721	0.1765157	0.823484	61873	10921	282059	775836	12.54	0.001115564	634721679	2316874537	0.60521	0.7780	11.01	14.06
75-79	75	5	0.5	298	**28**	0.093960	0.3804348	0.619565	50951	19384	206296	493777	9.69	0.003202502	1120017572	1682152858	0.64798	0.8050	8.11	11.27
80-85	80	5	0.5	126	**8**	0.063492	0.2739726	0.726027	31568	8649	136216	287481	9.11	0.006812042	562135286	562135286	0.56411	0.7511	7.63	10.58
85+	85	13	0.5	66	**10**	0.151515	1.0000000	0.000000	22919	22919	151265	151265	6.60	0.000000000	0	0	0.00000	0.0000	6.60	6.60
				74678	300															

Appendix three: Sample life table for Pacific, CMDHB 2005 (Chiang II method)

	x	n	a_x	*pop*	*death*	M_x	q_x	p_x	l_x	d_x	L_x	T_x	e_x	$var(q_x)$	$(1-a_x)n_1+e_{x+1}]^2 var(c'[(1-a_x)n_1+e_{x+1}]^2 v_i$	$Var(e_x)$	*SE*	Lower 95% confidence interval	Upper 95% confidence interval
<1	0	1	0.1	2227	**22**	0.009879	0.0097917	0.990208	100000	979	99119	7679500	76.80	0.000004315	258881754	4417003310	0.44170	0.6646	75.49	78.10
1-4	1	4	0.5	8597	**5**	0.000582	0.0023237	0.997676	99021	230	395623	7580382	76.55	0.000001077	58990871	4158121555	0.42408	0.6512	75.28	77.83
5-9	5	5	0.5	10752	**2**	0.000186	0.0009296	0.999070	98791	92	493724	7184759	72.73	0.000000432	20817705	4099130684	0.42001	0.6481	71.46	74.00
10-14	10	5	0.5	10118	**3**	0.000297	0.0014814	0.998519	98699	146	493129	6691034	67.79	0.000000730	30424394	4078312979	0.41865	0.6470	66.52	69.06
15-19	15	5	0.5	9218	**4**	0.000434	0.0021673	0.997833	98553	214	492229	6197906	62.89	0.000001172	41685350	4047888585	0.41677	0.6456	61.62	64.15
20-24	20	5	0.5	7544	**7**	0.000928	0.0046287	0.995371	98339	455	490557	5705676	58.02	0.000003047	91663199	4006203235	0.41427	0.6436	56.76	59.28
25-29	25	5	0.5	6866	**5**	0.000728	0.0036345	0.996365	97884	356	488530	5215119	53.28	0.000002632	65507056	3914540036	0.40856	0.6392	52.03	54.53
30-34	30	5	0.5	6860	**11**	0.001603	0.0079855	0.992015	97528	779	485694	4726588	48.46	0.000005751	117431333	3849032980	0.40466	0.6361	47.22	49.71
35-39	35	5	0.5	6806	**13**	0.001910	0.0095050	0.990495	96749	920	481448	4240895	43.83	0.000006884	112206196	3731601648	0.39866	0.6314	42.60	45.07
40-44	40	5	0.5	5854	**19**	0.003246	0.0160976	0.983902	95830	1543	475292	3759447	39.23	0.000013419	171739699	3619395452	0.39413	0.6278	38.00	40.46
45-49	45	5	0.5	4662	**7**	0.001502	0.0074794	0.992521	94287	705	469672	3284155	34.83	0.000007932	74826279	3447655752	0.38781	0.6227	33.61	36.05
50-54	50	5	0.5	3652	**15**	0.004107	0.0203280	0.979672	93582	1902	463154	2814483	30.08	0.000026988	187254648	3372829474	0.38513	0.6206	28.86	31.29
55-59	55	5	0.5	2912	**37**	0.012706	0.0615743	0.938426	91680	5645	444285	2351329	25.65	0.000096161	491746628	3185574826	0.37900	0.6156	24.44	26.85
60-64	60	5	0.5	2238	**25**	0.011171	0.0543360	0.945664	86034	4675	418485	1907044	22.17	0.000111679	357501360	2693828198	0.36394	0.6033	20.98	23.35
65-69	65	5	0.5	1578	**33**	0.020913	0.0993677	0.900632	81360	8085	386587	1488558	18.30	0.000269478	548709601	2336326838	0.35295	0.5941	17.13	19.46
70-74	70	5	0.5	998	**30**	0.030060	0.1397950	0.860205	73275	10243	340767	1101971	15.04	0.000560356	639272710	1787617237	0.33294	0.5770	13.91	16.17
75-79	75	5	0.5	642	**27**	0.042056	0.1902748	0.809725	63032	11993	285175	761204	12.08	0.001085767	603386998	1148344526	0.28904	0.5376	11.02	13.13
80-85	80	5	0.5	346	**21**	0.060694	0.2634881	0.736512	51038	13448	221572	476029	9.33	0.002434907	544957528	544957528	0.20920	0.4574	8.43	10.22
85+	85	14	0.5	176	**26**	0.147727	1.0000000	0.000000	37590	37590	254458	254458	6.77	0.000000000	0	0	0.00000	0.0000	6.77	6.77
				92046	**312**															

Appendix four: Notes on CMDHB attributable mortality models

This appendix describes the key assumptions used in the models looking at the effect of changing smoking prevalence on the life expectancy gap. The assumptions are listed numerically below.

1. Static models

For simplicity, an assumption was made that the models were static. In other words, the models assume that any reduction in smoking prevalence will be reflected in an immediate drop in smoking-attributable mortality. In reality, any impact of a reduction in smoking prevalence on mortality is likely to be slow and probably occur over several years. An analysis of the predicted impact of achieving smoking reduction goals in the US on mortality found substantial delay in benefit [34]. When looking at smoking initiation, a benefit in mortality was predicted to be seen after 35 years, while only one quarter of the benefit in mortality reduction would be seen in the following 70 years. Increasing cessation showed benefits to life expectancy sooner than those of initiation, but there was still a delay of many years. Other authors report more immediate effects on mortality of reducing smoking prevalence, especially in relation to cessation [40, 77]. Russell et al reported that complete cessation of smoking would result in a 15% mortality reduction after five years and an 11% reduction after 20 years [77]. In developing the UK Health Inequalities Intervention Tool, the London Health Observatory estimates that any interventions to increase smoking cessation would take at least five years to have any effect on life expectancy [78, 79].

2. Mortality by gender

Mortality rate ratios were available for Maaori, Pacific and non-Maaori, non-Pacific by sex [38]. Because of the heterogeneity of these ratios, separate ethnicity- and gender-specific rate ratios were applied to Maaori and Pacific male and female deaths for 2005. The expected deaths for each sex in each counterfactual scenario were then aggregated to give total hypothetical deaths for each age group in 2005 for Maaori and Pacific. The total hypothetical deaths for each age group were then substituted into life tables to give an aggregated life expectancy estimate by ethnicity.

The comparison models for Maaori and Pacific used adjusted mortality rate ratios taken from the CPS II study [37]. The all-cause mortality rate ratio for male smokers in this study was 2.3, while for female smokers it was 1.9. As these comparison models were intended to provide simple alternatives to the main models, age-adjusted deaths in males and females were aggregated and averaged to provide a simple total rate ratio which was applied to all ethnicity categories, of 2.17.

3. Mortality by age group

Given that there is heterogeneity in mortality rate ratios by ethnicity and by sex, it is also likely that there is heterogeneity by age group. However, an assumption was made for the models in this report that changes in attributable mortality by ethnicity and sex for each counterfactual scenario were consistent across all age groups of ≥ 25 years (≥ 30 years for the comparison models which used the CPS II mortality rate ratio). The age ranges were chosen as these were (roughly) the age ranges of the

study populations for the two studies used to guide the risk ratio estimates used in the models [37, 38]. The rationale for using consistent attributable mortality across all adult (≥ 25 or ≥ 30 years) was as follows:

- Mortality rate ratios were not available by age group and there was subsequently no way to reasonably distribute mortality differentially by age
- The models are intended only as 'back-of-the-envelope' guides to changes in life expectancy gaps as smoking prevalence changes. Using single risk ratios for mortality attributable to smoking across adult age ranges was the most simple way to estimate these changes
- Greater mortality in older age groups meant that proportional contribution of these groups to mortality was greater than in younger age groups

4. SIDS/SUDI

The impact of smoking on SIDS/SUDI was not included in the models, as it was not possible to find reliable data which allowed calculation of attributable risk percent for smoking in SIDS. In 2005 in CMDHB, there were six deaths in Maaori infants attributed to SIDS, compared with one for Pacific infants and none for non-Maaori, non-Pacific infants. The rate of SIDS death in Maaori was consistently higher than that for other groups over the 2001-2005 period (inclusive). During that five-year period, the average annual rate of SIDS mortality for Maaori was 8.4 per 100,000, while for Pacific it was 1.4 per 100,000 and for non-Maaori, non-Pacific it was 0.2 per 100,000. Note that numbers of infant deaths were small across this period and all of the mortality rates presented should be treated with caution.

Simple sensitivity analysis was carried out to understand the impact on life expectancy if none of the six Maaori SIDS deaths had occurred in 2005, i.e. the Maaori SIDS rate had been consistent with non-Maaori, non-Pacific. In this counterfactual case, Maaori life expectancy at birth increased from 72.08 years to 72.28 years – a difference of about two months. When the same sensitivity analysis was applied to Pacific life expectancy at birth, only a minimal difference of less than one month change in life expectancy was observed.

5. Ex-smokers and mortality

Ex-smokers are known to have an increased risk of mortality compared with never-smokers and this increased risk is believed to reduce over time after quitting. Some initial efforts were made to account for increased risk of mortality among ex-smokers in CMDHB, i.e. by substituting changes in prevalence of current smoking with changes in prevalence of former smoking and applying mortality rate ratios by ethnicity for ex-smokers derived from the study by Hunt et al [38], to obtain PAR% estimates for ex-smokers. However, it was thought that the mortality risk ratio estimates for ex-smokers by ethnicity derived from Hunt et al were probably not accurate enough for these purposes, as there were substantial overlaps in confidence intervals for the standardised mortality rates from which these estimates were derived. For simplicity, an assumption was made that risk for ex-smokers in the models was the same as that for never-smokers.

6. Second-hand smoke

It is not possible to account for mortality from second-hand smoke exposure in the models, as no local data are available on such deaths. However, second-hand smoke exposure is known to increase mortality by about 15% [80]. Each year in New

Zealand, about 350 people are killed by exposure to second-hand smoke [81]. If Counties Manukau has about 11% of New Zealand's smokers (67,700 out of 619,000 adult smokers in 2006/07) [47], it is reasonable to assume that between 35 and 40 people will be killed each year in CMDHB by exposure to second-hand smoke. Given the inequity in the distribution of smoking prevalence by ethnicity and that Maaori are more likely to be exposed to environmental tobacco smoke [61], it is likely that Maaori and Pacific peoples will share an unequal burden of this mortality. Second-hand smoke exposure is therefore likely to have a material impact on life expectancy gaps in CMDHB.

7. SIR/Peto method

In general, the amount of lung cancer in smokers versus never-smokers in a population indicates the accumulated hazard due to tobacco exposure and the 'maturity' of the smoking epidemic in that population [35, 39]. This observation was used to develop the smoking impact ratio (SIR). SIR is defined as the "population lung cancer mortality in excess of never-smokers, relative to excess lung cancer mortality for a known reference group of smokers" (p.888) [39]. SIR has been used in international estimates of disease burden attributable to smoking such as GBD [29] and in national estimates [69]. However, Maaori lung cancer rates are higher than expected due to smoking on its own. This means that the SIR method developed by Peto is likely to overestimate the mortality attributable to smoking in Maaori [71, 73]. Also, all-caused mortality rate ratios for Maaori, Pacific and non-Maaori, non-Pacific smokers (vs. never-smokers) in New Zealand are available, negating the need to adjust mortality in these groups to fit with a reference population (such as CPS II). The models in this report have therefore not used adjustment with SIR.

8. Static deaths for non-Maaori, non-Pacific

An assumption is made in the models presented in this report that mortality for non-Maaori, non-Pacific remains unchanged, i.e. that the life expectancy remains fixed at 82.4 years. It is recognised that this assumption is unlikely; however, the aim of these models was to look at changes in Maaori and Pacific life expectancy with changes in smoking prevalence. The current non-Maaori, non-Pacific life expectancy was therefore a useful benchmark against which to make comparisons.

9. Prevalence as an indicator of accumulated smoking risk

According to Ezzati and Lopez, "[c]urrent prevalence of smoking alone is… an insufficient indicator of accumulated risk from smoking" (p.885) [39]. This is because the accumulated hazards of smoking depend on a variety of different factors, such as age of smoking initiation and duration of smoking, number of cigarettes smoked per day (and degree of inhalation), and type of cigarettes smoked (for example, Maaori smokers are more likely to smoke hand-rolled rather than factory-made cigarettes [82]). The use of smoking prevalence as an indicator of accumulated smoking hazard in this study is therefore recognised as a limitation of the methodology.

References

[1] Webb P, Bain C, Pirozzo S. Essential epidemiology: An introduction for students and health professionals. Cambridge: Cambridge University Press 2005.

[2] Beaglehole R, Bonita R, Kjellstrom T. Basic epidemiology. Geneva: World Health Organization 1993.

[3] Rothman KJ, Greenland S, Lash TL. Modern epidemiology. 3rd ed. Philadelphia, PA: Lippencott Williams and Wilkins 2008.

[4] Griffiths C, Fitzpatrick J. Geographic inequalities in life expectancy in the United Kingdom, 1997-1997. Health Statistics Quarterly. 2001;9:16-28.

[5] Toson B, Baker A. Life expectancy at birth: Methodological options for small populations. National statistics methodological series number 33. Norwich: Her Majesty's Stationary Office; 2003.

[6] Counties Manukau District Health Board. About the DHB: Planning documents. 2008 [cited 2008 04 July]; Available from: http://www.cmdhb.org.nz/counties/about_cmdhb/planning/planning-documents.htm#DSP

[7] Wang K, Jackson G. The changing demography of Counties Manukau District Health Board. Manukau City: Counties Manukau District Health Board; 2008.

[8] Whitehead M. The concepts and principles of equity and health. International Journal of Health Services. 1992;22(3):429-45.

[9] Ajwani S, Blakely T, Robson B, et al. Decades of disparity: Ethnic mortality trends in New Zealand 1980-1999. Wellington: Ministry of Health and University of Otago; 2003.

[10] Blakely T, Tobias M, Atkinson J, et al. Tracking disparity: Trends in ethnic and socioeconomic inequalities in mortality, 1981-2004. Wellington: Ministry of Health; 2007.

[11] Ministry of Health. Tatau Kahukura: Maori health chart book. Public Health Intelligence monitoring report number 5. Wellington: Ministry of Health 2006.

[12] Robson B, Harris R, eds. Hauora: Maori standards of health IV. A study of the years 2000-2005. Wellington: Te Ropu Rangahau Hauora a Eru Pomare 2007.

[13] Winnard D. Maaori health needs analysis 2006/07. Manukau City: Counties Manukau District Health Board; 2007.

[14] Papa D. Life expectancy data for CMDHB. 2008.

[15] Smith AD. The ethnic revival. Cambridge: Cambridge University Press 1981.

[16] Statistics New Zealand. Statistical standard for ethnicity 2005. 2005 [cited 2008 20 March]; Available from: http://www.stats.govt.nz/NR/rdonlyres/DE0A3946-655C-4F82-BA4F-A38859C5E83D/0/StatisticalStandardforEthnicity2005.pdf

[17] Ministry of Health. Ethnicity data protocols for the health and disability sector. Wellington: Ministry of Health; 2004.

[18] New Zealand Health Information Service. Mortality collection data dictionary, version 1.2. Wellington: Ministry of Health; 2004.

[19] Ministry of Health. National Health Index training presentation. [cited 2008 15 July]; Available from: http://www.moh.govt.nz/moh.nsf/pagesns/271/$File/nhi-presentation.ppt#412,25,Slide

[20] Bascand G. New Zealand period life tables: 2005-2007. 2008 [cited 2008 08 December]; Available from: http://www.stats.govt.nz/NR/rdonlyres/82731AC4-71BD-434F-9EC8-05030DD4194F/0/newzealandperiodlifetables200507hotp.pdf

[21] Kirkwood BR, Sterne JAC. Essential medical statistics. 2nd ed. Oxford: Blackwell Science Ltd 2003.
[22] Chiang CL. Life table and mortality analysis. Geneva: World Health Organization; 1978.
[23] Pearce N, Mannetje A. Occupation in routinely collected national health data. Report to the New Zealand Health Information Service. Wellington: Centre for Public Health Research, Massey University; 2004.
[24] Health Outcomes International Pty Ltd. Methods and systems used to measure and monitor occupational disease and injury in New Zealand. NOHSAC technical report 2. Wellington: National Occupational Health and Safety Advisory Committee; 2005.
[25] Salomon JA, Mathers CD, Murray CJL, Ferguson B. Methods for life expectancy and healthy life expectancy uncertainty analysis. Global Programme on Evidence for Health Policy working paper number 10. Geneva: World Health Organization; 2001.
[26] Salomon JA, Murray CJL. Compositional models for mortality by age, sex and cause. GPE discussion paper series number 11. Geneva: World Health Organization; 2002.
[27] Novak B. Ethnic-specific health needs assessment for Pacific people in Counties Manukau. Manukau City: Counties Manukau District Health Board; 2007.
[28] Altman DG, Machin D, Bryant TN, Gardener MJ. Statistics with confidence. London: British Medical Journal 2000.
[29] Ezzati M, Lopez AD, Rodgers A, Murray CJL. Comparative quantification of health risks: Global and regional burden of disease attributable to selected major risk factors. Geneva: World Health Organization 2004.
[30] Ministry of Health. Our health, our future - Hauora pakari, koiora roa. The health of New Zealanders 1999. Wellington: Ministry of Health; 1999.
[31] Tobias M. The burden of disease and injury in New Zealand. Wellington: Ministry of Health; 2001.
[32] Ezzati M, Henley J, Thun MJ, Lopez AD. Role of smoking in global and regional cardiovascular mortality. Circulation. 2005;112:489-97.
[33] Centers for Disease Control and Prevention. Smoking-attributable mortality, morbidity and economic costs (SAMMEC). 2008 [cited 2008 23 October]; Available from: http://apps.nccd.cdc.gov/sammec/
[34] Silverstein MD, Nietert PJ, Zoller JS, et al. Predicted impact of attaining smoking reduction goals on mortality. Southern Medical Journal. 2001;94(2):176-80.
[35] Peto R, Lopez AD, Boreham J, et al. Mortality from tobacco in developed countries: Indirect estimation from national vital statistics. Lancet. 1992;339:1268-78.
[36] Peto R, Lopez AD, Boreham J, et al. Mortality from smoking in developed countries 1950-2000: Indirect estimates from national vital statistics. Oxford: Oxford University Press 1994.
[37] Thun MJ, Day-Lally CA, Calle EE, et al. Excess mortality among cigarette smokers: Changes in a 20-year interval. American Journal of Public Health. 1995;85(9):1223-30.
[38] Hunt D, Blakely T, Woodward A, Wilson N. The smoking-mortality association varies over time and by ethnicity in New Zealand. International Journal of Epidemiology. 2005;34:1020-8.
[39] Ezzati M, Lopez AD. Chapter 11: Smoking and oral tobacco use. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative quantification of health*

risks: Global and regional burden of disease attributable to selected major risk factors. Geneva: World Health Organization 2004.
[40] Husten CG, Thorne SL. Tobacco: Health effects and control. In: Wallace RB, ed. *Public health and preventive medicine*. 15th ed. New York: McGraw-Hill Companies 2008.
[41] Reid P, Robson B. The state of Maori health. In: Mulholland M, ed. *State of the Maori nation: Twenty-first-century issues in Aotearoa*. Auckland: Reed 2006.
[42] Ministry of Health. Smoking prevalence rates: Census 2006. District health board report: Counties Manukau. Wellington: Ministry of Health; 2008.
[43] Chan WC, Wright C, Riddell T, et al. Ethnic and socioeconomic disparities in the prevalence of cardiovascular disease in New Zealand. New Zealand Medical Journal. 2008;121(1285).
[44] Kerr AJ, MacLachlan A, Furness S, et al. The burden of modifiable cardiovascular risk factors in the coronary care unit by age, ethnicity, and socioeconomic status - PREDICT CVD-9. New Zealand Medical Journal. 2008;121(1285).
[45] Smith J, Papa D, Jackson G. Diabetes in CMDHB and northern region: Estimation using routinely collected data. Manukau City: Counties Manukau District Health Board; 2008.
[46] Jackson G. Morbid obesity in CMDHB adults – time trends 2003 to 2007. Internal memorandum, Counties Manukau District Health Board 2008.
[47] Ministry of Health. A portrait of health: Key results of the 2006/07 New Zealand Health Survey. Wellington: Ministry of Health; 2008.
[48] Counties Manukau District Health Board. Let's beat diabetes: A five year plan to prevent and manage type 2 diabetes in Counties Manukau. Manukau City: Counties Manukau District Health Board; 2005.
[49] Craig E, Jackson C, Yeo Han D. The health of children and young people in Counties Manukau. Auckland: New Zealand Child and Youth Epidemiology Service; 2007.
[50] Paediatric Society of New Zealand. The health status of children and young people in Counties Manukau. Wellington: Paediatric Society of New Zealand; 2005.
[51] Ministry of Health. Looking upstream: Causes of death cross-classified by risk and condition, New Zealand 1997. Wellington: Ministry of Health; 2004.
[52] Tobias M, Cheung J. Inhaling inequity: Tobacco's contribution to health inequity in New Zealand. Wellington: Ministry of Health; 2001.
[53] Alberts WM. Lung cancer guidelines: Introduction. Chest. 2003;123(1 Suppl 1):S1-S2.
[54] Alberg AJ, Samet JM. Epidemiology of lung cancer. Chest. 2003;123(1 Suppl 1):S21-S49.
[55] Huxley R, Jamrozik K, Lam TH, et al. Impact of smoking and smoking cessation on lung cancer mortality in the Asia-Pacific region. American Journal of Public Health. 2007;165(11):1280-6.
[56] US Department of Health and Human Services. The health benefits of smoking cessation: A report of the surgeon general. Number 90-8416. Atlanta: Centers for Disease Control and Prevention, Office on Smoking and Health; 1990.
[57] U.S. Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke: A report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2006.

[58] Doll R, Peto R, Wheatley K, et al. Mortality in relation to smoking: 40 years' observations on male British doctors. British Medical Journal. 1994;309:901-11.
[59] Stampfer MJ, Hu FB, Manson JE, et al. Primary prevention of coronary heart disease in women through diet and lifestyle. New England Journal of Medicine. 2000;343(1):16-22.
[60] Kawachi I, Colditz GA, Stampfer MJ, al e. Smoking cessation and time course of decreased risks of coronary heart disease in middle-aged women. Archives of Internal Medicine. 1994;154(2):169-75.
[61] Whitlock G, MacMahon S, Vander Hoorn S, et al. Association of environmental tobacco smoke exposure with socioeconomic status in a population of 7725 New Zealanders. Tobacco Control. 1998;7:276-80.
[62] Gillespie J, Milne K, Wilson N. Second-hand smoke in New Zealand homes and cars: Exposure, attitudes and behaviours in 2004. New Zealand Medical Journal. 2005;118(U1782).
[63] He J, Vupputuri S, Allen K, et al. Passive smoking and the risk of coronary heart disease: A meta-analysis of epidemiologic studies. New England Journal of Medicine. 1999;340(12):920-6.
[64] Haire-Joshu D, Glasgow RE, Tibbs TL. Smoking and diabetes. Diabetes Care. 1999;22(11):1887-98.
[65] Moy CS, LaPorte RE, Dorman JS, et al. Insulin-dependent diabetes mellitus mortality. The risk of cigarette smoking. Circulation. 1990;82(1):37-43.
[66] Mitchell BD, Hawthorne VM, Vinik AI. Cigarette smoking and neuropathy in diabetic patients. Diabetes Care. 1990;13(4):434-7.
[67] Stegmayr BG. A study of patients with diabetes mellitus (type 1) and end-stage renal failure: Tobacco usage may increase risk of nephropathy and death. Journal of Internal Medicine. 1990;228(2):121-4.
[68] Biesenbach G, Zazgornik J. Influence of smoking on the survival rate of diabetic patients requiring hemodialysis. Diabetes Care. 1996;19(6):625-8.
[69] Ministry of Health. Inhaling inequality: Tobacco's contribution to health inequality in New Zealand. Wellington: Ministry of Health; 2001.
[70] Blakely T, Fawcett J, Hunt D, Wilson N. What is the contribution of smoking and socioeconomic position to ethnic inequalities in mortality in New Zealand. Lancet. 2006;368:44-52.
[71] Wilson N, Blakely T, Tobias M. What potential has tobacco control for reducing health inequalities? The New Zealand situation. International Journal for Equity in Health. 2006;5:14.
[72] Hill S, Blakely T, Fawcett J, Howden-Chapman P. Could mainstream anti-smoking programmes increase inequalities in tobacco use? New Zealand data from 1981-1996. Australian and New Zealand Journal of Public Health. 2005;29:279-84.
[73] Wilson N, Blakely T, Tobias M. Correction: What potential has tobacco control for reducing health inequalities? The New Zealand situation. International Journal for Equity in Health. 2006;5:16.
[74] Blakely T, Wilson N. The contribution of smoking to inequalities in mortality by education varies over time and by sex: Two national cohort studies, 1981-84 and 1996-99. International Journal of Epidemiology. 2005;34:1054-62.
[75] Dawes M, Davies P, Gray A, et al. Evidence-based practice: A primer for health care professionals. 2nd ed. Philadelphia: Elsevier Limited 2005.
[76] Murray CJL, Salomon JA, Mathers CD. A critical examination of summary measures of population health. Bulletin of the World Health Organization. 2000;78(8):981-94.

[77] Russell LB, Carson JL, Taylor WC, et al. Modelling all-cause mortality: Projections of the impact of smoking cessation based on the NHEFS-NHANES I Epidemiologic Follow Up Study. American Journal of Public Health. 1998;88(4):630-6.
[78] London Health Observatory. Health inequalities intervention tool: Technical document. London: Department of Health and Association of Public Health Observatories; 2007.
[79] London Health Observatory. Health inequalities intervention tool: Frequently asked questions. London: Department of Health and Association of Public Health Observatories; 2007.
[80] Hill S, Blakely T, Kawachi I, Woodward A. Mortality among 'never smokers' living with smokers: Two cohort studies, 1081-84 and 1996-99. British Medical Journal. 2004;328:988-9.
[81] Woodward A, Laugesen M. How many deaths are caused by second-hand cigarette smoke? Tobacco Control. 2001;10:383-8.
[82] Ministry of Health. Tobacco trends 2006: Monitoring tobacco use in New Zealand. Wellington: Ministry of Health; 2006.