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Quantifying the role of modifiable risk factors in the differences in cardiovascular disease mortality rates between metropolitan and rural populations in Australia: a macrosimulation modelling study

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ABSTRACT

Objectives The study aimed (1) to quantify differences in modifiable risk factors between urban and rural populations, and (2) to determine the number of rural cardiovascular disease (CVD) and ischaemic heart disease (IHD) deaths that could be averted or delayed if risk factor levels in rural areas were equivalent to metropolitan areas.

Setting National population estimates, risk factor prevalence, CVD and IHD deaths data were analysed by rurality using a macrosimulation Preventable Risk Integrated Model for chronic disease risk. Uncertainty analysis was conducted using a Monte Carlo simulation of 10 000 iterations to calculate 95% credible intervals (CIs).

Participants National data sets of men and women over the age of 18 years living in urban and rural Australia.

Results If people living in rural Australia had the same levels of risk factors as those in metropolitan areas, approximately 1461 (95% CI 1107 to 1791) deaths could be delayed from CVD annually. Of these CVD deaths, 793 (95% CI 506 to 1065) would be from IHD. The IHD mortality gap between metropolitan and rural populations would be reduced by 38.2% (95% CI 24.4% to 50.6%).

Conclusions A significant portion of deaths from CVD and IHD could be averted with improvements in risk factors; more than one-third of the excess IHD deaths in rural Australia were attributed to differences in risk factors. As much as two-thirds of the increased IHD mortality rate in rural areas could not be accounted for by modifiable risk factors, however, and this requires further investigation.

Strengths and limitations of this study

- This is the first study to attempt to quantify the role of modifiable factors in the increased burden of cardiovascular diseases in rural areas when compared with urban Australia.
- The Preventable Risk Integrated Model (PRIME) has been previously published and is internationally recognised.
- This study used three representative national data sets, namely the Census, Australian Institute of Health and Welfare National Mortality Database, and the Australian Health Survey, as inputs for the PRIME model.
- Data used in the model were self-reported and could lead to underestimations of the level of behavioural risk factors, and subsequently mortality gap reductions, although it is unlikely that these biases would differentially affect rural versus metro populations.
- Very remote populations of Australia (0.9% of the total population) were excluded.

INTRODUCTION

Despite high-quality, universal healthcare systems and standards in Australia, there are still disparities in the burden of chronic disease experienced by people with lower socioeconomic status, Aboriginal and Torres Strait Islanders, and rural residents.[1] Australians living outside major cities experience a substantially increased burden of cardiovascular diseases (CVDs),[2] which is consistent with findings for rural populations in other developed countries such as the USA, Canada and the UK.[3–5] CVD in its most common form ischaemic heart disease (IHD) is the leading cause of death in Australia,[6] and individuals residing in regional or remote areas are estimated to be between 1.2 and 1.5 times more likely to die from IHD than those residing in metropolitan areas.[1]

The reasons for observed health inequalities between metropolitan and rural populations appear to be multifactorial and highly complex.[2 7–9] It has been hypothesised that rural populations experience greater CVD and IHD burden due to variation in access

to health services, individual socioeconomic status, rates of modifiable risk factors, as well as potentially reduced access to specialised evidence-based treatments, such as prompt surgical intervention in rural hospitals.[7 10–13] The National Rural Health Alliance of Australia states that if rural Australians are to achieve the same health outcomes as their metropolitan counterparts by the year 2020, it is not just access to health services that need to be improved,[14] but that more focus needs to be placed on improving the socioeconomic determinants of health (such as lower education, incomes and employment) for rural communities, and reducing risk factors for chronic diseases such as smoking and physical inactivity.[14]

CVDs, particularly IHD, are largely preventable, and the modifiable behavioural risk factors are well known.[15] Clinical risk factors such as high cholesterol, high blood pressure and obesity interact with behavioural factors such as poor diet, smoking, risky alcohol consumption and physical inactivity to increase the risk of IHD and CVD.[13] Based on population health surveys, these risk factors differ by rurality, with higher smoking rates, increased high-risk alcohol consumption and lower physical activity levels being reported by non-city-dwelling Australians.[2]

Currently, there is minimal evidence quantifying the extent to which modifiable individual risk factors contribute to the increased burden of CVD and IHD in rural areas, and how much of this burden could be reduced if behavioural risk factors were comparable among rural Australians as those among their counterparts in cities. Increased knowledge of how differential risk factor rates contribute to the increased rural CVD burden could support the identification of policy priorities and prevention programmes for rural areas, as previous public health prevention efforts have been shown to be less effective for rural populations compared with those living in metropolitan areas.[16]

Aims

This study aimed to (1) quantify differences in the prevalence of major CVD risk factors between rural and metropolitan populations, and (2) determine the number and proportion of rural deaths from CVD, and specifically from IHD, that could be averted or delayed in rural areas if the levels of risk factors were equivalent to those in metropolitan Australia.

METHODS

The Preventable Risk Integrated Model

This study used a macrosimulation model, the Preventable Risk Integrated Model (PRIME),[17] to estimate age-specific and sex-specific changes in CVD and IHD mortality that would result from changes to the population prevalence of risk factors in Australia. PRIME has been used in the UK, New Zealand and Canada to model a range of risk factor scenarios.[17–20] The model can be used to examine the likely changes in mortality rates of many chronic diseases, including IHD, under different counterfactual scenarios of population behavioural risk factors.[17]

The PRIME model is built on a framework of linkage between (1) modifiable behaviours, (2) clinical risk factors and (3) mortality outcomes. This framework is parameterised using the best available evidence from meta-analyses of published studies, and is described in detail elsewhere.[17] PRIME requires data on prevalence of modifiable risk factors, mortality rates and population estimates by 5-year age groups and sex, for both baseline and counterfactual scenarios. For this study, PRIME was used to estimate the number of CVD and IHD deaths that would be delayed or averted if those living in rural Australia had the same modifiable risk factor levels as those living in major cities. The baseline scenario used the current levels of modifiable risk factors in those living in rural areas, and the counterfactual scenario involved applying the level of risk factors currently observed in the metropolitan population. An additional 'best case' scenario was also modelled, by only changing risk factor levels that were more favourable among those living in metropolitan areas, when compared with the rural population.

Population data

Population size estimates by age, sex and rurality were derived from the 2011 Australian Census. The 'rural' population was defined as all residents not classified by the Australian Bureau of Statistics (ABS) as living in major cities.[21] These data were accessed through subscription to the online ABS data program TableBuilder.[22]

Risk factor data

The individual modifiable risk factors included in the PRIME model (table 1) were mean dietary intakes, alcohol consumption, prevalence of smoking, physical activity levels and mean body mass index (BMI) scores. All risk factor estimates were obtained from 9973 individuals aged 15 years and over surveyed for the 2011–2012 Australian Health Survey (AHS), which surveyed people from all areas of Australia, excluding very remote areas.[23] Mean values were calculated by age, sex and remoteness for each risk factor parameter, through the ABS program TableBuilder.[22]

Dietary intake data were collected using a 24-hour recall of all foods and fluids consumed in the day prior to the interview. Participants provided 24-hour recall data on two separate occasions, at least 8 days apart. Implausible intakes were excluded if the day of intake data had an energy intake (EI) to basal metabolic rate ratio of less than 0.9, as per recommendation from the ABS.[23] Mean daily dietary intakes from the 2 days of data collection were calculated for each participant for energy (kcal/day), fruit (g/day), vegetables (g/day), fibre (g/day), dietary cholesterol (mg/day), salt (g/day), total fat (% EI), saturated fat (% EI), monounsaturated fat (% EI) and polyunsaturated fat (% EI). The percentage of people who consumed less than one serve of fruit and

Table 1 Summary of risk factor data entered into the Preventable Risk Integrated Model

Risk factor	Parameter	Unit
Diet	Total energy intake	Kcal/day
	Proportion of low/non-consumers of fruit (<1 serve/day)	% of population
	Proportion of low/non-consumers of vegetables (<1 serve/day)	% of population
	Mean vegetable consumption of the remaining population	g/day
	Mean fruit consumption of the remaining population	g/day
	Fibre consumption	g/day
	Dietary cholesterol consumption	mg/day
	Salt consumption	g/day
	Total fat intake	% of total energy intake
	Saturated fat intake	% of total energy intake
	Monounsaturated fat intake	% of total energy intake
	Polyunsaturated fat intake	% of total energy intake
Alcohol	Proportion of low consumers (<1 g/day)	% of population
	Mean consumption among the remaining population	g/day of pure alcohol
Smoking	Current smokers	% of population
	Ex-smokers	% of population
	Never smokers	% of population
Physical activity	Proportion of population who are sedentary	% of population
	Amount of moderate-vigorous activity among the remaining population	MET hours per week
Anthropometry	Body mass index	kg/m^2
	Height	m

MET, metabolic equivalent of task.

less than one serve of vegetables was also calculated as required by the model. Mean BMI for the rural population, by 5-year age group and sex, was required by the model, and the effect of obesity on mortality rates was modelled using the differences in EI and physical activity levels between the two populations. Participants reported if they were current smokers, ex-smokers or if they had never smoked, and the age-specific, sex-specific and location-specific prevalences of each of these categories were derived.

The PRIME model requires the proportion of the population classified as 'sedentary', and physical activity levels in the form of mean metabolic equivalent of task (MET) hours per week, in the remaining (active) population. The sedentary proportion of the population was calculated as the sum of those classified as insufficiently active or sedentary by the ABS. Participants were classified as insufficiently active or sedentary if they did not meet the physical activity recommendations of 150 min per week, over five separate sessions.[24] Mean minutes of moderate and vigorous activity over the last week were used to calculate average daily minutes of activity. This duration was then multiplied by metabolic equivalents as provided by the ABS to convert the estimates to MET hours of activity per week.

The model requires the percentage of non-drinkers within each population by age and sex, which was calculated as those who reported either no alcohol consumption, or <1 g per day on average, over the surveyed period. Mean daily intake of pure alcohol in millilitres, by age group and sex, was calculated as the weighted daily mean (mL) within TableBuilder, then converted to grams of pure alcohol.

Mortality data

Deaths due to CVD (International Classification of Diseases- Tenth revision (ICD)-10 codes I00-I99) and IHD (ICD-10 code I20-I25) by state or territory and remoteness area of usual residence, by sex and 5-year age group, among people aged 15 years and over were provided by the Australian Institute of Health and Welfare (AIHW) for the year 2011 from the AIHW National Mortality Database (unpublished). These data were provided after an application for a specific request to the AIHW and are not publicly available.

Mortality gap

The mortality gap between rural and metropolitan areas was calculated by applying the metropolitan death rate for those aged 15 years and over to the rural population, to determine the number of rural deaths that would

Table 2 Differences in individual risk factors, rural compared with metropolitan areas, in women and men in 2011–2012, with 95% CI

	Women				Men			
Dietary component	**Mean intake (95% CI): rural**	**Mean intake (95% CI): metropolitan**	**Mean difference**	**p Value (t-test)**	**Mean intake (95% CI): rural**	**Mean intake (95% CI): metropolitan**	**Mean difference**	**p Value (t-test)**
Fruit (g) Serves*	257 (250 to 264) 1.7 serves	264 (258 to 269) 1.9 serves	7	p=NS	222 (214 to 231) 1.5 serves	231 (225 to 237) 1.5 serves	9	p=NS
Vegetables (g) Serves*	201 (196 to 205) 2.7 serves	184 (180 to 187) 2.45 serves*	17	p<0.0001	180 (176 to 186) 2.4 serves	158 (155 to 176) 2.1 serves	23	p<0.0001
Energy intake (kJ)	8496 (8372 to 8620)	8332 (8241 to 8424)	164	p=0.03	10 987 (10 818 to 11 157)	10 760 (10 634 to 10 887)	227	p=0.03
Fibre (g)	23.10 (23 to 24)	23 (23 to 24)	0.1	p=NS	27.0 (26.3 to 27.7)	26.6 (26.1 to 27.0)	0.4	p=NS
Sodium (mg)	2339 (2282 to 2396)	2279 (2236 to 2320)	61	p=NS	3046 (2969 to 3121)	2973 (2914 to 3032)	72	p=NS
Dietary cholesterol (mg)	299 (289 to 309)	284 (277 to 292)	15	p=0.02	380 (366 to 393)	375 (363 to 385)	5	p=NS
% Energy from fat	32 (31.7 to 32.5)	32 (31.5 to 32)	0.3	p=NS	31.5 (31 to 32)	31 (30.5 to 31.1)	0.5	p=0.01
% Energy from saturated fat	13.1 (12.8 to 13.3)	12.4 (12.2 to 12.5)	0.7	p<0.0001	12.9 (12.7 to 13.2)	12.1 (11.9 to 12.3)	0.8	p<0.0001
% Energy from monounsaturated fat	12.0 (11.8 to 12.1)	12.1 (11.9 to 12.3)	0.1	p=NS	12.0 (11.6 to 12.0)	12.0 (11.8 to 12.1)	0.1	p=NS
% Energy from polyunsaturated fat	4.8 (4.7 to 4.9)	5.0 (5.0 to 5.14)	0.2	p=0.0016	4.6 (4.5 to 4.7)	4.7 (4.6 to 4.8)	0.1	p=NS
Alcohol intake (g)	21.6	17.5	4.1	p=0.02	23.0	19.5	3.5	p=NS
% Current smokers	17.4	12.5	4.9	p=0.04	22.8	16	6.8	p=0.002
Physical activity (MET hours)	13.3	14.2	0.9	p=NS	14.0	14.5	0.5	p=NS

*One serve of fruit is 150 g and 1 serve of vegetables is 75 g according to the Australian dietary guidelines.
MET, metabolic equivalent of task; NS, not significant.

Table 3 Deaths averted or delayed from chronic diseases in rural Australia, under the counterfactual scenario, 95% credible intervals, by sex

	Deaths	Averted or delayed deaths (% of total deaths in the category)
CVD all ages		
All	13 600	1461 (10.7)
Male	6846	629 (9.2)
Female	6754	828 (12.3)
CVD <75 years		
All	3137	420 (13.4)
Male	2200	343 (15.6)
Female	915	78 (8.5)
IHD all ages		
All	7560	793 (10.5)
Male	4367	418 (9.6)
Female	3193	374 (11.7)
IHD <75 years		
All	2089	304 (14.6)
Male	1636	267 (16.3)
Female	452	37 (8.2)

CVD, cardiovascular disease; IHD, ischaemic heart disease.

be expected if cause-specific mortality rates were equal between metropolitan and rural areas. This figure was subtracted from the actual number of deaths in rural areas to calculate the gap in mortality between the two populations.

Uncertainty analysis

The PRIME model has a built-in Monte Carlo analysis function to generate 95% credible intervals (CIs) around the outputs, which allows for the different distribution of epidemiological parameters (eg, the level of relative risk for a disease outcome for a particular risk factor level) within the model that have been derived from the literature.[17] The intervals produced are based only on the uncertainty in the model parameters, and not on the variability of the original data used in the baseline and counterfactual scenarios. Monte Carlo simulation of 10 000 iterations was used to generate 95% CIs for each of the estimates of deaths delayed or averted under the counterfactual scenario.

RESULTS

Differences in individual risk factors between rural and metropolitan areas

There was a mixture of differences in dietary intake, between rural and metropolitan (table 2), that were statistically significant for many dietary components, although many were relatively small in absolute magnitude. Two of the dietary intake levels were more favourable in rural areas, and the rest were more favourable in metropolitan areas. Vegetable intakes were significantly higher for rural populations (p<0.001), also resulting in a slightly higher fibre intake, while intake of fruit was not significantly different between the two populations.

EI and the percentage of energy from saturated fat were significantly higher among rural populations (p<0.05). Rural women reported a significantly higher dietary cholesterol intake (p<0.05) and lower percentage energy from polyunsaturated fat (p<0.01) than their metropolitan counterparts. There were no other significant differences in intake across the other dietary components analysed.

The mean alcohol intake (g/day) in those who reported drinking alcohol was significantly higher in rural areas for women (p<0.05); however, there were no differences for men. There were significantly more current smokers in rural areas compared with major cities (women 19.5% vs

Table 4 Preventable deaths from CVD and IHD attributable to individual risk factors if rural populations had the same risk factor levels as populations in major cities

Risk factor	Total CVD deaths averted/delayed under counterfactual scenario (95% CI)	Total IHD deaths averted/delayed under counterfactual scenario (95% CI)
Overall diet	−199 (−389 to 10)	−190 (−380 to 4)
Fruit and vegetables	−290 (−475 to 103)	−298 (−486 to 111)
Fibre	−70 (−103 to 39)	−47 (−75 to 19)
Fats	121 (98 to 145)	133 (113 to 155)
Salt	40 (17 to 62)	19 (8 to 31)
Physical activity	84 (66 to 103)	53 (38 to 69)
Smoking	337 (290 to 388)	225 (192 to 260)
Alcohol	−55 (−101 to 18)	−28 (−52 to 5)
Obesity	1309 (100 to 1608)	740 (50 to 955)

Positive numbers represent deaths delayed or averted under the counterfactual scenario, while negative numbers represent an increase in deaths under the counterfactual scenario.
CVD, cardiovascular disease; IHD, ischaemic heart disease.

Table 5 Deaths averted or delayed from chronic diseases in rural Australia, under the 'best case' scenario, 95% credible intervals, by sex

	Averted or delayed deaths (% of total deaths in the category) best case
CVD all ages	
All	1669 (12.2)
Male	771 (11.2)
Female	898 (13.3)
CVD <75 years	
All	445 (14.2)
Male	371 (16.8)
Female	74 (8.0)
IHD all ages	
All	1161 (15.3)
Male	707 (16.2)
Female	456 (14.2)
IHD <75 years	
All	352 (16.9)
Male	309 (18.9)
Female	44 (9.7)

CVD, cardiovascular disease; IHD, ischaemic heart disease.

14.0%; men 24.2% vs 18.7%). The proportion of people who had never smoked was higher in metropolitan areas.

Fewer rural people were meeting recommendations for physical activity; 37.1% of men in rural areas were meeting recommendations compared with 44.0% in major cities. This was also true for women (36.5% in rural areas, 46.3% in cities). There were no significant differences between the two populations in the mean MET hours per week of moderate-vigorous activity performed by the active population.

Smoking rates were significantly higher in rural areas for both women and men. The mean BMI for women was significantly higher (p<0.001) in rural areas compared with major cities (women 26.8 vs 27.6 kg/m^2; men 27.3 vs 27.8 kg/m^2).

Deaths from CVD and IHD averted or delayed by risk factor changes

In 2011, 13 600 people aged 15 years and over died from CVD in rural areas, with 6846 and 6754 deaths occurring in men and women, respectively. In terms of premature deaths, 3137 of these deaths occurred in those aged under 75 years, 2200 in men and 915 in women. IHD was the cause of 7560 deaths, with 2089 deaths occurring in those under 75 years (1636 men, and 452 women).

In total, 1461 (1107 to 1791) or 11% of all rural CVD deaths would be delayed or averted, if rural populations were to have the same levels of individual risk factors as their metropolitan counterparts (table 3). Of the deaths prevented from CVD, 793 (510 to 1065) would be from IHD, slightly more of these among men (418) than women (374), a 10.5% reduction in rural IHD deaths. Premature IHD deaths (75 years and under) would account for 38.3% of the IHD deaths that would be delayed or averted, or 4% of all rural IHD deaths. Most of these premature deaths would be averted for men, 266 (232 to 301), compared with 37 (28 to 46) in women.

The rural to metropolitan mortality gap was equivalent to approximately 2075 additional IHD deaths in the rural population when compared with the metropolitan population in 2011 (data not shown). In the counterfactual scenario, risk factor differences accounted for 38.2% of the gap, leaving 61.8% due to other, unmeasured factors. There were substantial differences by sex, with 28.1% of the gap attributable to risk factor differences for men, compared with 66.7% for women.

Deaths attributable to individual risk factors

Table 4 shows the number of deaths that would be delayed or averted under the counterfactual scenario for each individual risk factor for both CVD and IHD. Obesity and smoking, as individual risk factors, accounted for the largest numbers of CVD and IHD deaths that could be prevented in rural areas, contributing 1309 (1000 to 1608) and 337 (290 to 388) deaths delayed or averted, respectively, for CVD, and 740 (504 to 955) and 225 (192 to 260) for IHD. The adjustment of fruit and vegetable intakes showed that more people would die from CVD (−290 (05% CI −475 to −103)) and IHD (−298 (95% CI −486 to −111)) if intakes in rural areas were to match those of metropolitan areas, since in this case vegetable intakes would be worse under the counterfactual scenario.

An additional 'best case' scenario was explored in which only risk factor changes that resulted in improvements in cardiovascular mortality were modelled. As shown in table 4, changes to rural vegetable, fibre and alcohol intakes to match those in major cities resulted in an increase in deaths from CVD; therefore, these three risk factors were held unchanged in the 'best case' scenario. Table 5 shows that in this scenario, a total of 1669 (1380 to 1950) deaths could be delayed or averted in rural areas. Of this total, 1161 (943 to 1365) deaths would be averted from IHD, which would lead to a mortality gap reduction of 56% (45.4% to 65.7%) in rural Australia.

DISCUSSION

This analysis showed that more than one-third of the IHD mortality inequality between metro and rural areas can be attributed to differences in individual risk factors, an important consideration for health policy and intervention planning. Importantly, however, over 60% of the increased mortality burden in rural areas was unexplained by risk factor differences.

The majority of individual risk factors were found to differ between rural and metropolitan areas, with significantly higher BMI, EIs and proportion of EI from fats observed in rural populations, along with higher levels of

high-risk alcohol consumption and sedentary behaviour. Rural people did, however, report significantly higher vegetable intakes, which equated to consumption of approximately 10% more vegetables than their metropolitan counterparts, consistent with previous health survey data.[2]

The origins of the differences in individual modifiable IHD risk factors between rural and metropolitan areas[2] are likely to be complex, arising from the interaction of a range of factors at the individual and community levels, including socioeconomic disadvantage, education levels, access to primary health services and health literacy.[25] Living outside of metropolitan areas has been associated with more risk factors for chronic diseases in adults,[26] with some evidence that geographical location should be assessed as a social determinant of health, above and beyond socioeconomic status and area-level disadvantage.[27] There is evidence for this in that self-reported prevalence of IHD, diabetes and cerebrovascular disease was found to be similar in rural and metropolitan areas, despite much higher measured mortality rates from these diseases in rural areas, potentially revealing reduced health awareness in rural populations.[28] This may be an important contributor to differences in outcomes for rural populations, as lower health literacy has been linked to poorer outcomes in patients with heart disease.[25]

In terms of individual risk factors, differences in obesity and tobacco smoking appeared to have the biggest impact on the increased burden of IHD in the rural population, when compared with metropolitan areas. Obesity is a well-known risk factor for IHD[15] and has been consistently shown to be higher in rural populations of Australia for the past few decades.[28 29] Differences in EI and physical activity energy expenditure between rural and metropolitan populations led to substantial reductions in obesity-mediated mortality in the counterfactual scenario. These reductions explained the largest proportion of the mortality gap between the two populations of any of the behaviours studied. Higher EIs in the rural sample, which increase the likelihood of a higher BMI,[30] could be reflective of the consumption of more low-cost, energy-dense foods, possibly in part due to reduced access to healthier foods, which has been well documented in rural and regional areas.[31]

The cardioprotective effects of alcohol consumption are often debated.[32] Our results showed that current levels of alcohol intake do not appear to contribute to increased cardiovascular risk for rural populations, with 55 fewer CVD deaths averted (an increase in deaths overall) as a result of the difference in consumption between the two populations. This was mainly due to the fact that although overall intakes were higher in rural areas, intakes among men over 75 years of age were lower in rural areas than in metro areas, therefore leading to a modelled increase in consumption and therefore associated deaths in this group under the counterfactual scenario.

Tobacco smoking is recognised as the largest single preventable cause of mortality and morbidity in Australia,[33] and higher smoking rates in rural areas have been apparent since at least 1993.[16 26] Despite numerous public health initiatives and interventions over this period, the rural smoking rate has remained comparatively high while the metropolitan rate has continued to decline,[16 33] possibly indicating that such preventative efforts have not adequately reached rural Australians.[16]

The results of this study suggest that substantial gains could be made in reducing the CVD mortality gap between metropolitan and rural populations if modifiable risk factors could be improved in rural areas. Improving risk factor profiles in rural populations to at least match that of their metropolitan counterparts could be assumed to be a reasonable target, and is certainly a relatively modest goal, given that even metropolitan risk factor levels are far from ideal.[34 35] Under the 'best case scenario', if only unfavourable risk factors in rural areas were changed (eg, vegetable intakes left the same), approximately 200 additional CVD deaths to the counterfactual scenario could be prevented every year. Targeting the unfavourable risk factors, such as smoking and obesity in rural people, could be modest and achievable targets for health policy and/or community interventions in order to reduce the rural death rate to be closer to the levels observed in metropolitan areas. Importantly, even if the counterfactual risk factor reduction scenario were to be achieved, a significant proportion (almost two-thirds) of the excess deaths would remain, a finding that should prompt major reflection on the role of socio-economic disadvantage, healthcare provision and other, less prominent risk factors in the perpetuation of rural health inequalities.

Strengths

This study used three routinely collected, representative national data sets, namely the Census, AIHW National Mortality Database and the AHS, as inputs for the PRIME model. These data sets represent the highest quality and comprehensive population data available currently for Australia. The use of three robust data sets is required for PRIME to provide accurate disease outputs. The PRIME model also has many strengths, including that it is able to model the effect of changes in multiple risk factors simultaneously on mortality rates from different chronic diseases, by age and sex. The model has been designed using the strongest available scientific evidence on the links between chronic disease mortality and dietary intake, alcohol consumption, smoking, physical activity and obesity levels.[16]

Limitations

Data used in the model were self-reported, which carries multiple limitations and could lead to underestimations of the level of behavioural risk factors, and subsequently mortality gap reductions,[36] although it is less likely that these biases would differentially affect rural versus metro populations. Very remote areas of Australia were not sampled in the AHS; therefore, the risk factor levels for these populations were not included in the analysis.[23]

This may lead to an underestimation of the prevalence of some risk factors, as residents of very remote areas are known to experience even higher burden from IHD than the broader rural population[2]; however, they also represent a very small percentage of the total Australian population (0.9%).[26] The uncertainty analysis conducted on the number of deaths averted only accounts for uncertainty within the model parameters, but not the uncertainty of estimates from the Australian Heath Survey. Lastly, we were unable to account for the diversity in characteristics between differing levels of remoteness (eg, inner regional areas compared with outer regional or remote areas) due to small population numbers. Instead, it was necessary to make a single comparison between those living within and outside major cities. Differences in population characteristics, access to health services and environmental factors can vary significantly by differing levels of remoteness, and this is an important consideration for policy and planning when trying to improve health in non-metropolitan areas.

CONCLUSIONS

There is potential for improvements in the level of CVD burden observed in rural Australians, if the prevalence of modifiable risk factors such as obesity and smoking were to be reduced to match those of metropolitan areas. If such a scenario was to be achieved, 38% of the mortality gap between rural and metropolitan areas could be reduced; however, investigations into factors responsible for the remaining 62% of this inequality are needed in order to work towards achieving health equality for rural populations.

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Contributors LA organised the collection and conversion of data, completed all data entry into the PRIME model, analysis of the results, and writing of the manuscript. MN contributed to defining the original research idea, data collection, use of the PRIME model and analysis. KLP and JPJ assisted with conversion of the data to be compatible with the PRIME model. SA contributed to defining the original research idea. All authors reviewed and intellectually contributed to the manuscript and all stages of compilation.

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Competing interests None declared.

Ethics approval An exemption from ethics review was obtained from Deakin University's Human Research Ethics Unit.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement The PRIME model is available upon request from Dr Peter Scarborough contactable at peter.scarborough@dph.ox.ac.uk. Population and health survey data were accessed through the Australian Bureau of Statistics TableBuilder. The mortality data (ICD deaths by remoteness) were provided by the AIHW under a specific data request.

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