A comparison of Australian rural and metropolitan cardiovascular risk and mortality: the Greater Green Triangle and North West Adelaide Population Surveys.

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ARTICLE SUMMARY

ARTICLE FOCUS

- The study aim was to more objectively understand causes of geographical cardiovascular disease (CVD) mortality disparities in Australia by; (a) comparing measures of CVD risk (objective and self-reported data) between a rural population (Greater Green Triangle, GGT) and urban population (North West Adelaide, NWA)
- (b) comparing CVD mortality rates between GGT and NWA and other areas Australia-wide and
- (c) describing the relationship between socioeconomic status (SES) and CVD mortality rates.

KEY MESSAGES

- This study supports existing evidence of a social gradient in cardiovascular health.
- This study provides evidence to reject the assertion that location of residence in Australia necessarily results in poorer cardiovascular health.

STRENGTHS AND LIMITATIONS OF THIS STUDY

- This is the first comparison of both self-report and biomedical data from a wholly rural/regional Australian population study with a metropolitan population study.
- Determinants of cardiovascular health are contextual, and the study populations will not necessarily represent rural and urban populations more generally in Australia.
- Direct analysis of associations between risk factors, SES and CVD mortality in the sample data sets was not possible due to the cross-sectional rather than longitudinal design of the two population-based risk factor studies and other methodological differences in sampling and data collection.

ABSTRACT

Background: Cardiovascular (CVD) mortality disparities between rural/regional and urban-dwelling residents of Australia are persistent. Unavailability of biomedical CVD risk factor data has, until now, limited efforts to understand the causes of the disparity.

Methods: This study investigated rural/regional-urban CVD mortality disparities by comparing (a) CVD risk measures between a regional population (Greater Green Triangle Risk Factor (cross-sectional) Study 2004-2006 (GGT RFS, n =1563)), and an urban population (North West Adelaide Health (longitudinal cohort) Study 2004-2006) (NWAHS Stage 2, n=3036)) (b) Australian Bureau of Statistics (ABS) CVD mortality rates between these and other Australian regions and (c) ABS CVD mortality rates by an area-level indicator of socioeconomic status, the Index of Relative Socioeconomic Disadvantage (IRSD).

Results: Few significant differences in CVD risk between the study regions, with absolute CVD risk ranging from approximately 5% to 30% in the 35-39 and 70-74 age groups respectively. Similar mean 2003-2007 mortality rates in the Greater Green Triangle (GGT) region (98), the North West Adelaide (NWA) region (103) and regional Australia (92). NWA mortality rates exceeded that of other city areas (70). Lower measures of SES were associated with worse CVD outcomes regardless of geographic location.

Conclusions: Metropolitan areas do not always have better CVD risk factor profiles and outcomes than rural/regional areas. Needs assessments are required for different settings to elucidate relative contributions of the multiple determinants of risk and the appropriate cardiac health care strategies to improve outcomes.

MAIN TEXT

INTRODUCTION

Place of residence is an important determinant of health. In many settings worldwide, there is an underinvestment in health-promoting infrastructure and opportunities in rural communities leading to urban migration and geographical health inequalities [1]. Australia is a highly urbanised country with approximately two-thirds of the population living in major cities [2]. Well-documented health inequalities exist between regional and remote versus urban settings. In the former, life expectancy is 1-7 years lower and decreases with increasing remoteness [3]. An approximate 10% difference in all-cause mortality rates has been consistently documented between major cities and the rest of Australia [4].

As in many other countries, cardiovascular disease (CVD) - principally ischaemic heart disease (IHD) and cerebrovascular disease - is the largest contributor to overall mortality in Australia [5]. Coronary heart disease and 'other' circulatory diseases are the two largest contributors to the excess mortality observed outside major city areas (20% and 17% of the excess mortality between 2002 and 2004) [4]. Measuring contributions of biological and behavioural risk factors, social and economic determinants, access to quality care and broader politico-structural influences on CVD health outcomes in Australia has proved difficult, especially in rural areas.

A recent Australian Institute of Health and Welfare report found that prevalence of key CVD risk factors increases with increasing remoteness from major city areas [6]. Such self-report data, however, has limitations. Despite the obvious need for more objectively measured population data, very little risk factor data in the form of biomedical measurements is available for comparative studies between remote, regional and urban areas. Better evidence is required to develop strategies to address inequalities.

This paper reports on absolute CVD risk from two population biomedical surveys covering a regional area (Greater Green Triangle, GGT) and metropolitan area (North-west Adelaide, NWA), along with CVD mortality rates from corresponding regions drawn from national data records. To our knowledge, it is the

only comparative study of measured biomedical risk factors and mortality data between specifically regional and urban populations in Australia to date.

The study aim was to more objectively understand causes of geographical CVD mortality disparities by; (a) comparing measures of CVD risk (objective and self-reported data) between GGT and NWA; (b) comparing CVD mortality rates between GGT and NWA and other areas Australia-wide and (c) describing the relationship between socioeconomic status (SES) and CVD mortality rates.

We hypothesised that 1) higher mortality rates would be observed in GGT than NWA and that 2) these would be influenced by worse CVD risk factor profiles in the former.

METHODS

Study design

This study compared CVD risk factor data (individual as well as absolute 5-year CVD risk) from two studies - a regional cross-sectional population survey and an urban longitudinal cohort - conducted over a similar time period. In addition, Australian Bureau of Statistics (ABS) CVD mortality rates in different geographical locations were compared and the relationship between mortality and SES explored.

Population and sample

Comparing measures of CVD risk

Details of the methodology of both studies have been published elsewhere [7-11]. Below is a brief summary of the setting, population and sample.

Greater Green Triangle Risk Factor Study

GGT encompasses a population of 225,000 in south-east South Australia and south-west Victoria. The Greater Green Triangle Risk Factor Study (GGT RFS) comprised three cross-sectional population surveys (Limestone Coast, Corangamite and Wimmera Shire Risk Factor Surveys) conducted between 2004 and 2006. In total, 1563 randomly selected persons aged 25-74 provided some information (self-administered questionnaire +/- attendance at survey site for anthropometric and biomedical measurements including fasting venous blood specimens for lipids and glucose). Socioeconomic indicators of GGT RFS participants compared with available population statistics indicated that the survey population closely represented the overall GGT population [7].

North West Adelaide Health Study

Adelaide, the capital of South Australia, has a population of 1.18 million [12]. The northern and western suburbs, stretching from Glenelg to Gawler, encompass approximately half of Adelaide's population and one-third of the South Australian population. The North West Adelaide Health Study (NWAHS) is a largely

representative cohort of over 4000 randomly selected adults aged 18 and over recruited from NWA between 2000 and 2003 (Stage 1) returning between 2004 and 2006 (Stage 2). Each stage included a telephone survey, self-administered questionnaire and anthropometric and bio-medical examination. NWAHS Stage 1 participants had some demographic differences but no health risk behaviour differences compared with ABS 2006 census data and South Australian Surveillance and Monitoring System data [13].

In this study, participants aged 25-74 were used in order to make the age range of both populations comparable. From NWAHS, only Stage 2 participants were used and 3036 provided information.

Sources and measures

Comparing measures of CVD risk

Demographic characteristics have been reported previously and are presented in Table 1 [14]. A comprehensive examination of methodologies and questionnaire wordings of both studies had been undertaken in order to ensure that variables were comparable. Some aspects could not be compared due to differences in questions used such a household income, levels of alcohol consumption, physical activity and quality of life. GGT RFS participant age was calculated from the survey date after assuming each individual was born on June 30 in their given year of birth. NWAHS participant age was calculated from their date of birth and clinic appointment date and truncated.

Five-year absolute CVD risk, defined as IHD and stroke collectively, was calculated using the Framingham equation which is used to make Australian cardiovascular event risk charts [15]. Calculation of CVD risk was restricted to participants aged 35-74 who reported no history of heart attack or stroke. Biomedical measurements required for use of the equation were available from both studies. Smoking status was determined by self-report. Diabetes was defined as having a survey fasting plasma glucose level of 7.0mmol/L or above and/or having self-reported diabetes. As the questionnaire used in GGT RFS asked whether a participant had ever been diagnosed with impaired glucose tolerance, participants who responded positively were considered to have diabetes. As no electrocardiogram information was available for any participants, the left ventricular hypertrophy variable was excluded from the risk calculation.

Comparing CVD mortality outcomes

Mortality rates were obtained using 2003-2007 ABS mortality (numerator) and Estimated Residential Population (ERP, denominator) data according to relevant 2006 Statistical Local Area (SLA) codes [16-17]. ABS Australian Standard Geographical Classification System (ASGC) for Remoteness Areas uses categories major cities, inner regional, outer regional, remote and very remote [17]. Thirty-one SLA codes representing GGT (n=13) and NWA (n=18) were used. According to ASGC all GGT SLAs were classified as inner or outer regional and all NWA SLAs as major city areas. In this comparative study 'inner and outer regional' areas consisted of all areas in this ASGC category combined, but excluded GGT SLAs. 'Remote and very remote' areas represented all such ASGC areas combined. 'Major cities' included all Australian cities classified as such by the ASGC, excluding NWA SLAs. Mortality information was extracted according to predefined International Classification of Diseases (ICD) 10 codes [5]. ICD 10 codes I20-I25 and I61-I64 were used to make up the category IHD and Stroke.

Relationship between SES and CVD mortality rates

SES was measured using IRSD (Index of Relative Socio-economic Disadvantage). IRSD is one of four ABS Socio-Economic Indexes For Areas (SEIFA), which are area-based summary measures of relative socio-economic disadvantage [18]. IRSD takes into account a range of variables including education, employment and financial well-being. Although area and individual-level SES may have independent effects on health outcomes, only area-level SES was taken into account.

The distribution of IRSD scores between GGT and NWA SLAs were compared and the relationship between IRSD and CVD mortality rates explored.

Analyses

Statistical analyses were undertaken using Stata V12 and IBM SPSS Statistics V19. CVD risk factor data for participants are reported as mean values with standard errors for continuous variables and proportions with 95% confidence intervals (using the Agresti-Coull technique) for discrete variables. Independent sample t-tests were used to assess differences between means (α=0.05), with the Welsch method applied when the assumption of homogenous variance was not met. Chi-Square ($\chi 2$) tests were used to assess differences between proportions (α=0.05). The relationship between mortality rates and IRSD scores was examined using linear regression.

Ethics

Ethics approval for GGT RFS was received from the Flinders Clinical Research Ethics Committee, Adelaide, approval number 207/034. Ethics approval for NWAHS Stage 2 was received from The Queen Elizabeth Hospital (TQEH) Human Research Ethics Committee (HREC), Adelaide, approval number 2004030. HREC approval for comparison analysis was given by the University of South Australia, Adelaide, and the Queensland University of Technology, Brisbane, approval numbers P136/09 and 1000000988.

RESULTS

Demographic characteristics of participants

NWAHS participants were younger, more diverse in their country of origin, more likely to be single, separated or divorced and less likely to be in part time or casual employment than GGT RFS participants (Table 1). A slightly lower proportion of NWAHS participants identified as being of Aboriginal/Torres Strait Islander origin.

Table 1: Demographic characteristics of participants by location

	NWAHS			GGT RFS			
	n	%	95% CI	n	%	95% CI	
Demographics							
Sex							
Male	1437	50.2	(48.0 - 52.4)	714	50.2	(46.9 - 53.5)	
Female	1426	49.8	(47.6 - 52.0)	708	49.8	(46.5 - 53.1)	
Age							
25 to 44 years	1412	49.3	(47.1 -51.6)	599	42.1	(38.6 - 45.7)	*
45 to 54 years	620	21.6	(20.1 - 23.3)	350	24.6	(22.3 - 27.1)	*
55 to 64 years	477	16.7	(15.4 - 18.0)	277	19.5	(17.6 - 21.5)	*
65 to 74 years	355	12.4	(11.3 - 13.6)	196	13.8	(12.4 - 15.3)	
Aboriginal or Torres Strait Islander							
No	2785	97.3	(96.5 - 97.8)	1405	98.8	(98.1 - 99.3)	*
Yes	13	0.4	(0.2 - 0.8)	8	0.6	(0.3 - 1.1)	
Country of birth							
Australia or New Zealand	2064	72.1	(70.2 - 73.9)	1339	94.1	(92.8 - 95.2)	*
UK or Ireland	451	15.8	(14.4 - 17.3)	27	1.9	(1.4 - 2.6)	*
Europe	223	7.8	(6.8 - 8.9)	26	1.8	(1.4 - 2.5)	*
Other	116	4.0	(3.2 - 5.1)	28	2.0	(1.3 - 3.0)	*
Highest level of education obtained							
Secondary school or lower	1568	57.9	(55.5 - 60.3)	920	64.7	(61.4 - 67.9)	*
Trade / Apprenticeship / Certificate / Diploma / Vocational training (TAFE/VET)	651	24.1	(22.0 - 26.3)	254	17.9	(15.3 - 20.8)	*
Bachelor degree or higher	460	17.0	(15.1 - 19.1)	229	16.1	(13.7 - 18.8)	
Marital Status							
Married or living with a partner	1988	73.5	(71.2 - 75.6)	1198	84.2	(81.8 - 86.4)	*
Separated or divorced	252	9.3	(8.3 - 10.5)	86	6.0	(4.8 - 7.6)	*
Widowed	77	2.9	(2.4 - 3.4)	46	3.2	(2.6 - 4.0)	
Never married (single)	381	14.1	(12.1 - 16.3)	91	6.4	(4.8 - 8.5)	*
Work Status							
Full time employed	1352	50.0	(47.5 - 52.4)	680	47.8	(44.5 - 51.1)	
Part time / Casual employment	514	19.0	(17.2 - 20.9)	327	23.0	(20.2 - 26.0)	*
Unemployed	58	2.2	(1.6 - 2.9)	43	3.0	(2.2 - 4.3)	
Home duties	304	11.2	(9.9 - 12.7)	126	8.8	(7.1 - 11.0)	*
Retired	378	14.0	(12.8 - 15.2)	209	14.7	(13.1 - 16.4)	
Student	27	1.0	(0.6 - 1.8)	3	0.2	(0.06 - 0.7)	#
Other	64	2.4	(1.8 - 3.0)	11	0.8	(0.4 - 1.6)	*
TOTAL	2864	100%		1422	100%		

Data source: North West Adelaide Health Study Stage 2, 2004-2006, 25 to 74 years. Greater Green Triangle Risk Factor Study, 2004-2006, 25 to 74 years.
Note: The weighting of the data can result in rounding discrepancies or totals not adding.
#Insufficient numbers for a statistical test.
**Statistically significantly different (χ^2 test, $p < 0.05$) GGT RFS compared with NWAHS.*
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Comparing measures of CVD risk

Framingham 5-year absolute CVD risk scores were not significantly different between GGT RFS and NWAHS participants (age-specific groups and overall, Figure 1(a)).

[Insert Figures 1(a) and 1(b) here]

There were some differences in individual CVD risk factors after standardising to the 2006 Australian population but the magnitude of differences were small (Table 2). NWAHS participants had a lower mean systolic blood pressure and higher mean diastolic blood pressure than GGT RFS participants. HDL cholesterol was lower in NWAHS (men and overall). Total triglycerides were higher in NWAHS overall (though not quite reaching statistical significance) yet lower in NWA women. NWA men had higher BMI and waist circumference. NWAHS participants (women and overall) were more likely to be smokers. Prevalence of diabetes/IGT was higher in NWAHS (men and overall).

Table 2: Individual CVD risk factor data by location

		NWAHS Mean (SE, N)	GGT RFS Mean (SE, N)	*p*-value
Mean systolic blood pressure (mmHg)		123.37 (0.31, 2639)	126.00 (0.48, 1419)	**<0.001**
	Men	126.71 (0.43, 1302)	128.60 (0.64, 700)	**0.014**
	Women	120.12 (0.44, 1337)	123.47 (0.69, 719)	**<0.001**
Mean diastolic blood pressure (mmHg)		80.55 (0.20, 2639)	76.06 (0.29, 1418)	**<0.001**
	Men	83.80 (0.26, 1302)	79.27 (0.40, 700)	**<0.001**
	Women	77.39 (0.26, 1337)	72.93(0.39, 718)	**<0.001**
Total cholesterol (mmol/L)		5.37 (0.02, 2647)	5.37 (0.03, 1377)	0.903
	Men	5.38 (0.03, 1299)	5.39 (0.04, 680)	0.887
	Women	5.36 (0.03, 1348)	5.34 (0.04, 697)	0.742
LDL cholesterol (mmol/L)		3.26 (0.02, 2554)	3.22 (0.03, 1353)	0.171
	Men	3.31 (0.03, 1221)	3.30 (0.04, 658)	0.852
	Women	3.22 (0.02, 1333)	3.14 (0.04, 694)	0.071
HDL cholesterol (mmol/L)		1.43 (0.01, 2647)	1.46 (0.01, 1377)	**0.003**
	Men	1.28 (0.01, 1299)	1.33 (0.01, 680)	**0.001**
	Women	1.56 (0.01, 1348)	1.59 (0.01, 697)	0.148
Total-C/HDL-C ratio		3.97 (0.02, 2647)	3.93 (0.04, 1377)	0.328
	Men	4.38 (0.03, 1299)	4.31 (0.06, 680)	0.298
	Women	3.58 (0.03, 1348)	3.56 (0.04, 697)	0.657
LDL-C/HDL-C ratio		2.40 (0.02, 2554)	2.37 (0.03, 1353)	0.388

	Men	2.66 (0.02, 1221)		2.63 (0.04, 658)		0.585
	Women	2.16 (0.02, 1333)		2.13 (0.03, 694)		0.351
Total triglycerides (mmol/L)		1.55 (0.03, 2647)		1.48 (0.03, 1322)		0.065†
	Men	1.83 (0.05, 1299)		1.64 (0.04, 650)		0.262†
	Women	1.28 (0.03, 1348)		1.33 (0.03, 673)		**<0.001†**
BMI (kg/m2)		28.30 (0.11, 2658)		28.00 (0.15, 1413)		0.089
	Men	28.51 (0.14, 1308)		28.05 (0.18, 699)		**0.043**
	Women	28.09 (0.17, 1349)		27.92 (0.23, 714)		0.545
Waist circumference (cm)						
	Men	99.77 (0.38, 1302)		97.85 (0.48, 695)		**0.002**
	Women	87.71 (0.39, 1337)		88.07 (0.55, 714)		0.587
		NWAHS		GGT RFS		
		n (%)	**95% CI**	**n (%)**	**95% CI**	***p*-value**
Current smokers		2642 (21.35)	19.83-22.95	1405 (17.79)	15.88-19.88	**0.028**
	Men	1301 (22.75)	20.55-25.11	696 (20.26)	17.44-23.41	0.302
	Women	1341 (19.99)	17.93-22.21	709 (15.37)	12.90-18.22	**0.032**
Known diabetes or Impaired Glucose Tolerance (IGT)		2656 (7.72)	6.76-8.80	1422 (5.84)	4.73-7.18	**0.037**
	Men	1307 (8.34)	6.96-9.97	700 (5.14)	3.72-7.06	**0.014**
	Women	1350 (7.19)	5.92-8.69	721 (6.38)	4.80-8.42	0.520

Data source: North West Adelaide Health Study Stage 2, 2004-2006, 25 to 74 years. Greater Green Triangle Risk Factor Study, 2004-2006, 25 to 74 years.
Note: The weighting of the data can result in rounding discrepancies or totals not adding.
†p-values based on log of the variable in order to address right skewedness of data.

Comparing CVD mortality outcomes

Figure 1(b) shows the relationship between IHD and Stroke mortality and age for GGT and NWA. Table 3 compares IHD and Stroke mortality rates between different regions of interest. IHD and Stroke mortality in inner and outer regional areas was generally worse than in major cities (p<0.001). Remote and very remote areas had significantly higher mortality rates than all other categories (p<0.001).

Table 3: Comparison of ischaemic heart disease (IHD) and stroke mortality rates by age group

Age Group (years)	**35-39**	**40-44**	**45-49**	**50-54**	**55-59**	**60-64**	**65-69**	**70-74**	**35-74: Crude**
NWA	9 (5-16)	19 (13-28)	34 (26-46)	62 (49-78)	99 (81-120)	157 (132-186)	281 (246-322)	419 (373-470)	103 (96-110)
GGT	5 (0-20)	14 (6-31)	28 (16-50)	44 (27-71)	99 (70-139)	130 (94-180)	224 (171-292)	488 (402-592)	98 (87-111)
NWA vs. GGT	*p=0.427*	*p=0.511*	*p=0.562*	*p=0.208*	*p=0.987*	*p=0.319*	*p=0.130*	*p=0.182*	*p=0.489*
Major Cities (Ex NWA)	6 (5-6)	12 (11-13)	23 (22-24)	38 (37-40)	63 (61-66)	110 (107-114)	199 (193-205)	369 (361-377)	70 (69-71)
NWA vs. Major Cities	*p=0.061*	*p=0.010**	*p=0.006**	*p=0.000**	*p=0.000**	*p=0.000**	*p=0.000**	*p= 0.028**	

(Ex NWA)									*p<0.001**
Inner and Outer Regional (Ex GGT)	8 (7-10)	16 (15-18)	30 (28-32)	47 (44-50)	75 (71-79)	131 (125-137)	230 (222-238)	440 (428-453)	92 (91-94)
GGT vs. Inner and Outer Regional (Ex GGT)	*p=0.492*	*p=0.714*	*p=0.872*	*p=0.768*	*p=0.097*	*p=0.991*	*p=0.840*	*p=0.295*	*p=0.341*
Remote and Very Remote	36 (28-47)	48 (39-60)	76 (64-91)	82 (68-99)	138 (118-162)	210 (181-243)	345 (300-395)	593 (524-671)	125 (118-132)

Age specific IHD and Stroke Mortality Rates per 100,000 population (95% Confidence Intervals) by age group. Mean of all deaths from 2003-2007
**Statistically significantly different (χ^2 test, $p < 0.05$).*

In all age groups GGT mortality rates were representative of those of inner and outer regional areas (crude mortality rates for 35-74 years: inner and outer regional versus GGT 92 versus 98, p=0.341). NWA mortality was generally higher than in other major Australian cities (crude mortality rates for 35-74 years: major cities versus NWA 70 versus 103, p=0.028). GGT and NWA mortality rates did not differ significantly despite NWA being a major city location (crude mortality rates for 35-74 years: GGT versus NWA p=0.489).

Relationship between SES and CVD mortality rates

A comparison of IRSD scores using an independent samples median test indicated no significant difference between the two study areas (p=0.108). However there was a significant difference in the distribution of IRSD scores (p=0.022), with scores in NWA skewed towards the lower end of the scale (Figure 2(a)).

Increasing mortality was consistently associated with lower IRSD scores. When age-specific mortality rates for age class 35-74 were plotted against IRSD (Figure 2(b)), both study areas were most closely aligned with inner and outer regional areas. Closer inspection of study areas at the SLA level indicated that the trend remained. In NWA (Figure 2(c)) IRSD explained around 46% (n=18, β=-0.389) of the variation in mortality. In GGT (Figure 2(d)) IRSD explained approximately 19% (n=13, β=-0.477) of the variation in mortality, although the relationship was not statistically significant.

[Insert Figures 2(a), 2(b), 2(c) and 2(d) here]

DISCUSSION

Geographic and socioeconomic disparities in CVD mortality were first described in Australia in the late 1990s, [19] and initiated debate about likely explanations. Socioeconomic and cultural diversity between regions, differential prevalence of CVD risk factors, and variations in patterns of medical care were postulated as potential causative factors. This work started a debate about the most appropriate actions both within and outside the healthcare system to address these disparities [20-21]. Progress since has been slow in advancing our understanding of these issues, impeded by the lack of comprehensive, high quality data on CVD risk factor prevalence across the Australian population.

Based on AIHW published data,[3, 4, 6] and the only previous Australian study to analyse the contribution of CVD risk factor prevalence differences to the rural/regional–urban CVD mortality gap [22], our original hypothesis in this study was that GGT CVD risk factor profiles, and CVD mortality, would be worse than in NWA. Unexpectedly, GGT and NWA were similar in terms of absolute CVD risk scores, individual CVD risk factors and mortality rates. Furthermore, mortality rates in the regional GGT population are consistent with those observed in most regional areas of Australia, but lower than in remote areas, and higher than in the overall Australian metropolitan population. CVD mortality rates in the metropolitan NWA population are significantly higher than in the overall Australian metropolitan population.

Social gradients in health – 'caused by unequal distribution of power, income, goods and services' lead to inequitable health outcomes within and between populations [1]. Poorer Australians have worse CVD outcomes [23]. This was demonstrated in our study by the strong relationship between IRSD and CVD mortality at a national level (Figure 2(b)) as well as within NWA (Figure 2(c)). The trend was present within GGT (Figure 2(d)) although statistically non-significant. This can likely be explained by limited sample size coupled with a relatively narrow range of IRSD scores compared with NWA. These findings are consistent with other evidence in the Australian literature and from other developed countries regarding the association between low SES and increased levels of CVD risk factors, morbidity and mortality [21].

The influence of a broad range of social determinants (for example, quality of housing, employment, income

level, education etc.) on biological determinants of CVD, as well as differential access to health-promoting services may explain a significant part of the rural/regional-urban divide in CVD mortality in Australia. There is also growing evidence that variation in implementation of evidence based CVD care across geographic, institutional and even subspecialty boundaries may be an important determinant [24-25]. Implementation of evidence-based practice may provide an opportunity to reduce disparities in CVD outcomes, including geographically determined disparities, at relatively low cost and in shorter time frames than those required to address socioeconomic disparities across large populations.

All of the aforementioned variables and their relationship with CVD health outcomes are complex, yet all should be taken into account when formulating strategies to address inequalities.

Our study has limitations. Firstly, there are difficulties in extrapolating results from single rural and urban populations. This regional study population is relatively culturally and socioeconomically homogenous and probably representative of many (but not all) regional areas in Australia. The urban population is more culturally and socioeconomically diverse with over-representation of the socioeconomically disadvantaged compared with the overall Australian urban population. Secondly, we were unable to directly analyse associations between risk factors, SES and CVD mortality in the sample data sets due to the cross sectional rather than longitudinal design of the two population-based risk factor studies and other methodological differences in sampling and data collection. Time frames influencing some cross-sectional measured risk factor variables, compared with those operating over whole lifetimes to determine clinical outcomes such as CVD mortality, are different and we cannot be sure that they are stable or changing at the same rate in two geographically distinct populations.

Strategies for comprehensive, high quality CVD risk factor surveillance should cover all population groups, regardless of geography or SES. Preferably, there should be longitudinal follow up, combined with appropriate epidemiological and health services research to investigate which interventions are most cost effectively able to reduce disparities in CVD outcomes in the specific context of each of our social and health care systems.

Legend of Figures

Figure 1: Framingham absolute CVD risk and IHD and stroke mortality rates by age

Figure 2: Relationship between IHD and stroke mortality and IRSD

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COMPETING INTERESTS

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CONTRIBUTORSHIP STATEMENT

PT conceived and designed the study. AT is Epidemiological Principal Investigator of the North West Adelaide Health Study. JAD was the chief investigator for the GGT DPP.

AT, RC, AM, JG and JAD were involved in acquisition of data. BP, EJ, EP and VV analysed and interpreted the data. EP and EJ drafted the manuscript and were responsible for its revisions. VV and RC helped to draft the manuscript. All authors contributed to specific sections in the manuscript. All authors read and approved the final manuscript.

DATA SHARING STATEMENT

There is no additional data publicly available. For further enquiries on accessing data please contact Prof. James Dunbar at director@greaterhealth.org and Janet Grant at janet.grant@adelaide.edu.au

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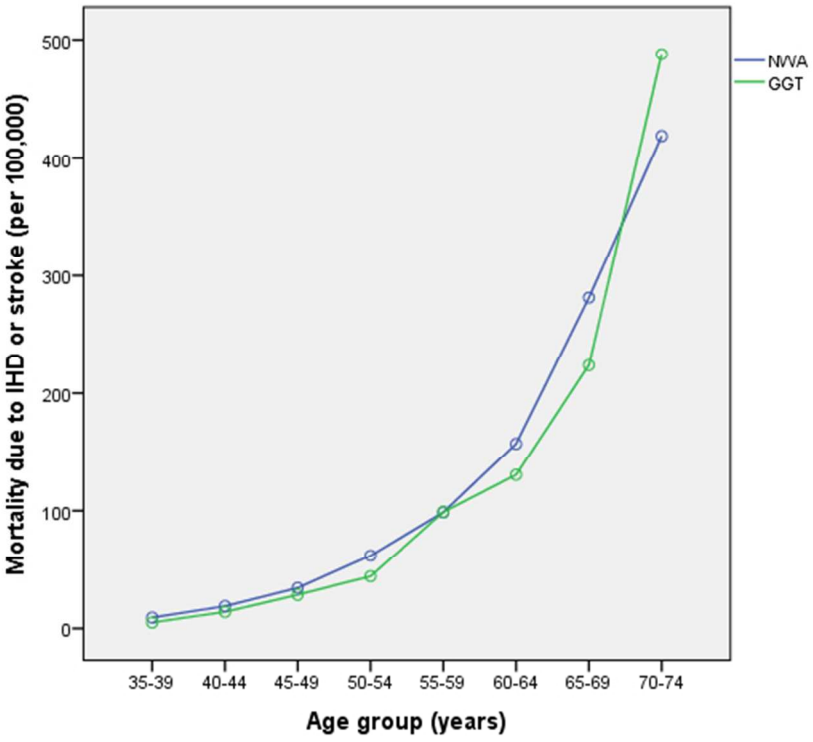

Figure 1: (a) Framingham absolute CVD risk and (b) IHD and stroke mortality rates by age
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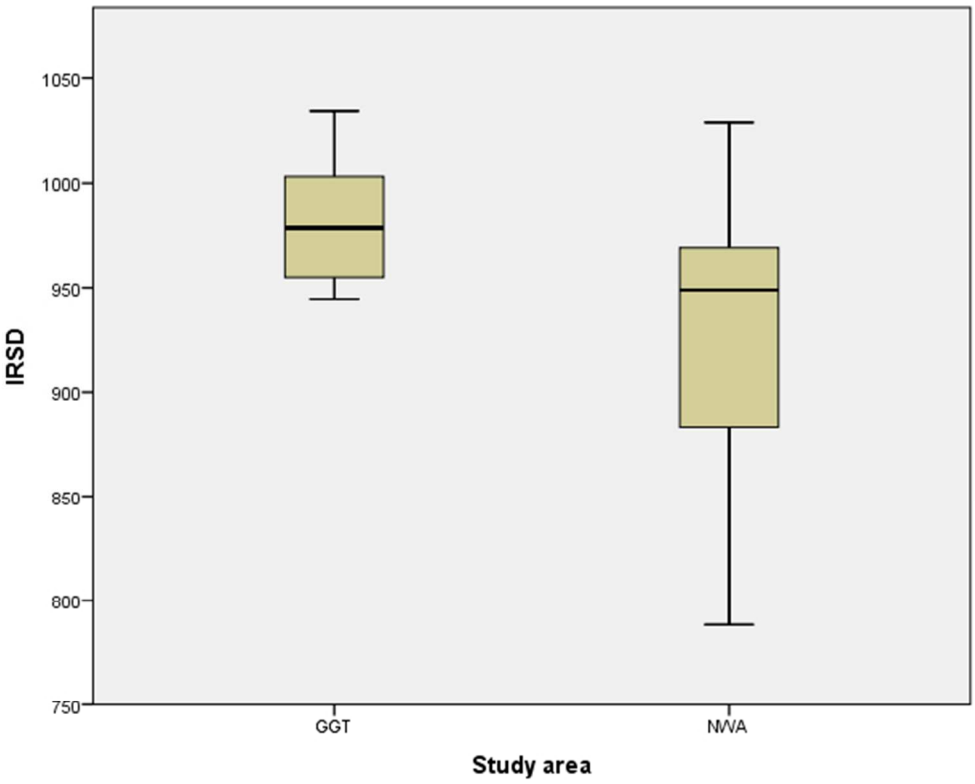

Figure 2: Relationship between IHD and stroke mortality and IRSD. (a) Distribution of IRSD scores between GGT and NWA; (b) IHD and stroke mortality rates by median IRSD for relevant geographical areas; (c) IHD and stroke mortality rates by IRSD for NWA SLAS; (d) IHD and stroke mortality rates by IRSD for GGT SLAs
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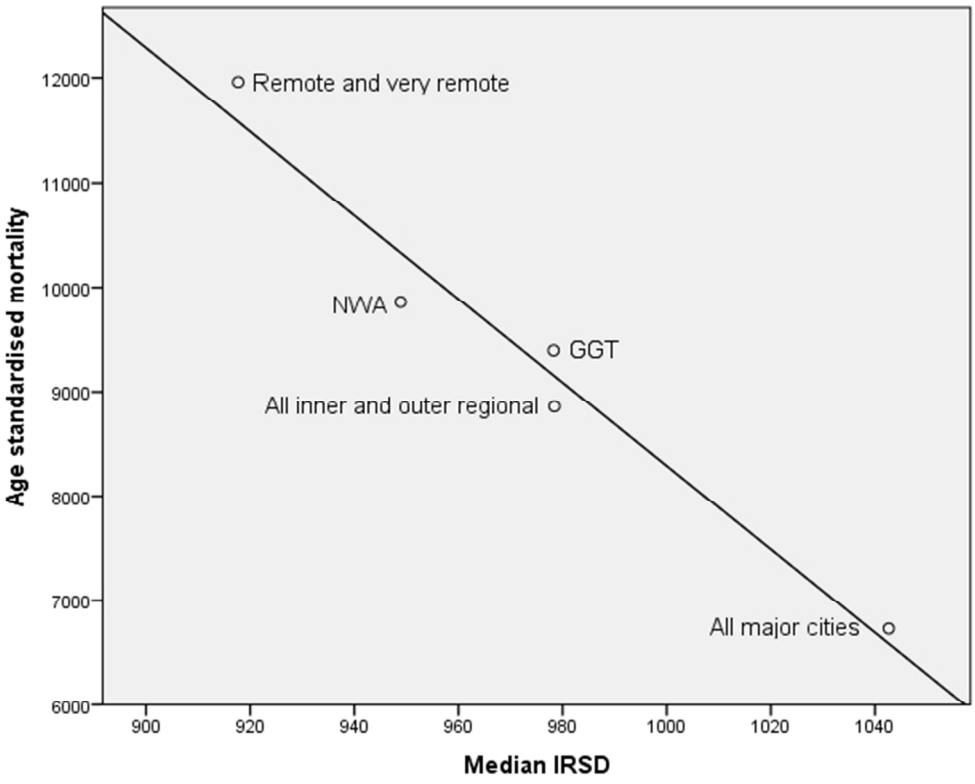

Figure 2: Relationship between IHD and stroke mortality and IRSD. (a) Distribution of IRSD scores between GGT and NWA; (b) IHD and stroke mortality rates by median IRSD for relevant geographical areas standardised to the Australian 2006 ERP; (c) IHD and stroke mortality rates by IRSD for NWA SLAS; (d) IHD and stroke mortality rates by IRSD for GGT SLAs
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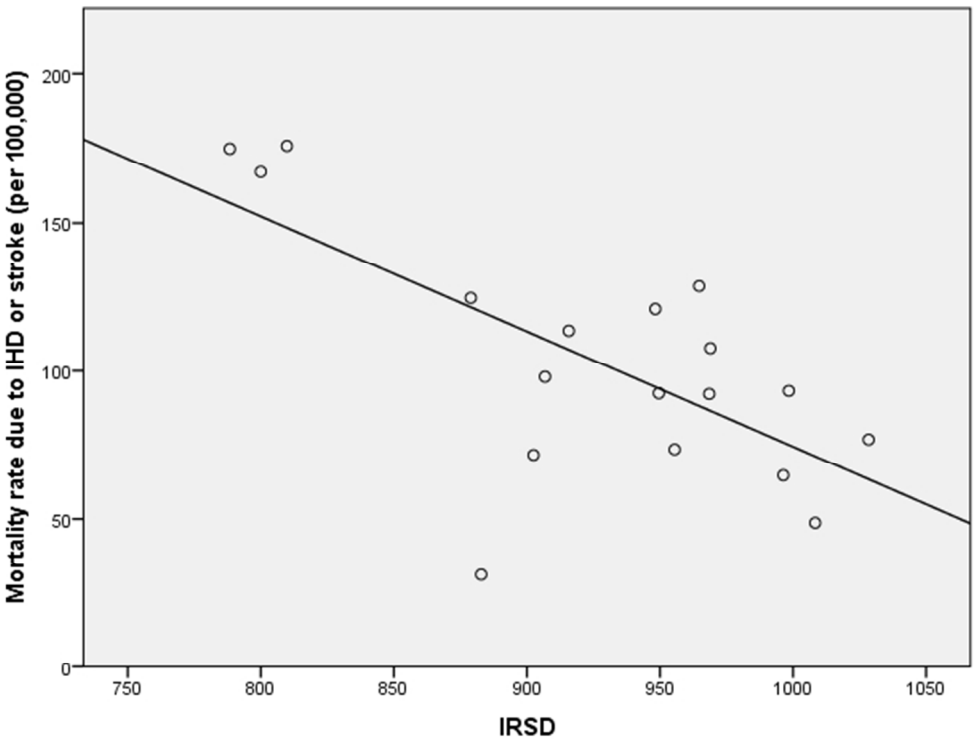

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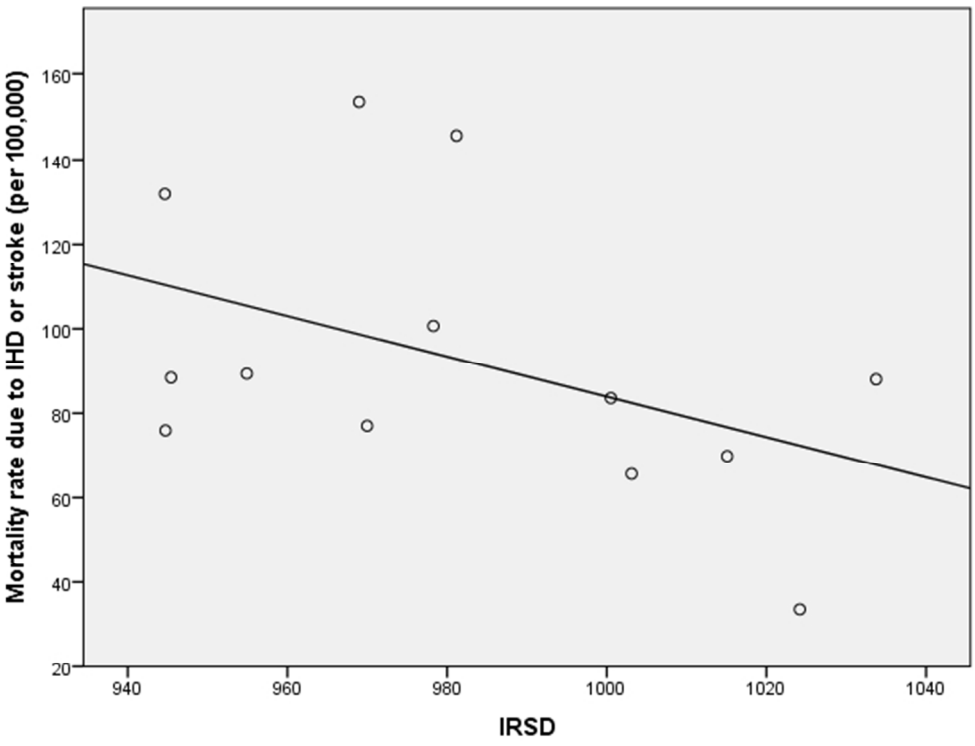

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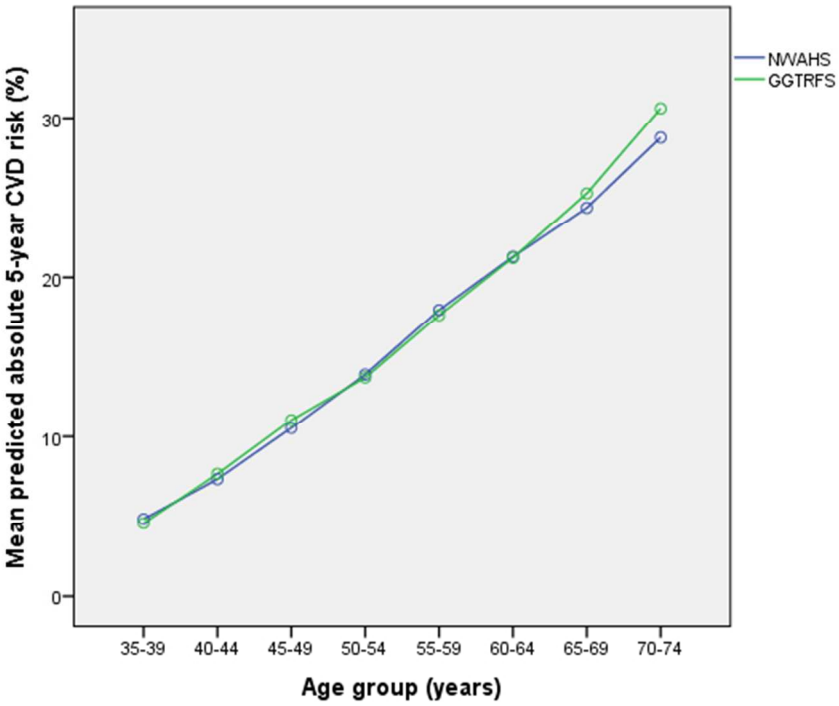

Figure 1: (a) Framingham absolute CVD risk and (b) IHD and stroke mortality rates by age
220x176mm (72 x 72 DPI)

STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation	Addressed on page number:
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	2
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	3
Objectives	3	State specific objectives, including any prespecified hypotheses	3
Methods			
Study design	4	Present key elements of study design early in the paper	4
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants	5,6
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case	
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	6,7
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	6,7
Bias	9	Describe any efforts to address potential sources of bias	6,7
Study size	10	Explain how the study size was arrived at	5,6
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	8
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	8
		(*b*) Describe any methods used to examine subgroups and interactions	7, 8, Fig 1(a), Fig 1(b)

(*c*) Explain how missing data were addressed	
(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy	5 (references 7-11)
(*e*) Describe any sensitivity analyses	NA

Continued on next page

Results			**Addressed on page number:**
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	5,6
		(b) Give reasons for non-participation at each stage	NA
		(c) Consider use of a flow diagram	
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	15
		(b) Indicate number of participants with missing data for each variable of interest	9-11 (Tables 1 and 2)
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)	
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time	
		Case-control study—Report numbers in each exposure category, or summary measures of exposure	
		Cross-sectional study—Report numbers of outcome events or summary measures	10-12
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	5 (reference 14) 11-12 (Table 3)
		(*b*) Report category boundaries when continuous variables were categorized	NA
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	NA
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	8
Discussion			
Key results	18	Summarise key results with reference to study objectives	12
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	14,15
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	14,15
Generalisability	21	Discuss the generalisability (external validity) of the study results	15
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	16

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

A comparison of Australian rural and metropolitan cardiovascular risk and mortality: the Greater Green Triangle and North West Adelaide Population Surveys.

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Keywords:	Coronary heart disease < CARDIOLOGY, EPIDEMIOLOGY, PUBLIC HEALTH

TITLE	*A comparison of Australian rural and metropolitan cardiovascular risk and mortality: the Greater Green Triangle and North West Adelaide Population Surveys.*
TYPE OF ARTICLE	Research

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KEYWORDS	
Rural Health, Cardiovascular Diseases, Health Status Disparities, Delivery of Health Care, Socioeconomic Factors	
WORD COUNT	2615

ARTICLE SUMMARY

ARTICLE FOCUS

- The study aim was to more objectively understand causes of geographical cardiovascular disease (CVD) mortality disparities in Australia by; (a) comparing measures of CVD risk (objective and self-reported data) between a rural population (Greater Green Triangle, GGT) and urban population (North West Adelaide, NWA)
- (b) comparing CVD mortality rates between GGT and NWA and other areas Australia-wide and
- (c) describing the relationship between socioeconomic status (SES) and CVD mortality rates.

KEY MESSAGES

- This study supports existing evidence of a social gradient in cardiovascular health.
- This study provides evidence to reject the assertion that location of residence in Australia necessarily results in poorer cardiovascular health.

STRENGTHS AND LIMITATIONS OF THIS STUDY

- This is the first comparison of both self-report and biomedical data from a wholly rural/regional Australian population study with a metropolitan population study.
- Determinants of cardiovascular health are contextual, and the study populations will not necessarily represent rural and urban populations more generally in Australia.
- Direct analysis of associations between risk factors, SES and CVD mortality in the sample data sets was not possible due to the cross-sectional rather than longitudinal design of the two population-based risk factor studies and other methodological differences in sampling and data collection.

ABSTRACT

Background: Cardiovascular (CVD) mortality disparities between rural/regional and urban-dwelling residents of Australia are persistent. Unavailability of biomedical CVD risk factor data has, until now, limited efforts to understand the causes of the disparity.

Methods: This study investigated rural/regional-urban CVD mortality disparities by comparing (a) CVD risk measures between a regional population (Greater Green Triangle Risk Factor (cross-sectional) Study 2004-2006 (GGT RFS, n =1563)), and an urban population (North West Adelaide Health (longitudinal cohort) Study 2004-2006) (NWAHS Stage 2, n=3036)) (b) Australian Bureau of Statistics (ABS) CVD mortality rates between these and other Australian regions and (c) ABS CVD mortality rates by an area-level indicator of socioeconomic status, the Index of Relative Socioeconomic Disadvantage (IRSD).

Results: Few significant differences in CVD risk between the study regions, with absolute CVD risk ranging from approximately 5% to 30% in the 35-39 and 70-74 age groups respectively. Similar mean 2003-2007 mortality rates in the Greater Green Triangle (GGT) region (98), the North West Adelaide (NWA) region (103) and regional Australia (92). NWA mortality rates exceeded that of other city areas (70). Lower measures of SES were associated with worse CVD outcomes regardless of geographic location.

Conclusions: Metropolitan areas do not always have better CVD risk factor profiles and outcomes than rural/regional areas. Needs assessments are required for different settings to elucidate relative contributions of the multiple determinants of risk and the appropriate cardiac health care strategies to improve outcomes.

MAIN TEXT

INTRODUCTION

Place of residence is an important determinant of health. In many settings worldwide, there is an underinvestment in health-promoting infrastructure and opportunities in rural communities leading to urban migration and geographical health inequalities [1]. Australia is a highly urbanised country with approximately two-thirds of the population living in major cities [2]. Well-documented health inequalities exist between regional and remote versus urban settings. In the former, life expectancy is 1-7 years lower and decreases with increasing remoteness [3]. An approximate 10% difference in all-cause mortality rates has been consistently documented between major cities and the rest of Australia [4].

As in many other countries, cardiovascular disease (CVD) - principally ischaemic heart disease (IHD) and cerebrovascular disease - is the largest contributor to overall mortality in Australia [5]. Coronary heart disease and 'other' circulatory diseases are the two largest contributors to the excess mortality observed outside major city areas (20% and 17% of the excess mortality between 2002 and 2004) [4]. Measuring contributions of biological and behavioural risk factors, social and economic determinants, access to quality care and broader politico-structural influences on CVD health outcomes in Australia has proved difficult, especially in rural areas.

A recent Australian Institute of Health and Welfare report found that prevalence of key CVD risk factors increases with increasing remoteness from major city areas [6]. Such self-report data, however, has limitations. Despite the obvious need for more objectively measured population data, very little risk factor data in the form of biomedical measurements is available for comparative studies between remote, regional and urban areas. Better evidence is required to develop strategies to address inequalities.

This paper reports on absolute CVD risk from two population biomedical surveys covering a regional area (Greater Green Triangle, GGT) and metropolitan area (North-west Adelaide, NWA), along with CVD mortality rates from corresponding regions drawn from national data records. To our knowledge, it is the

only comparative study of measured biomedical risk factors and mortality data between specifically regional and urban populations in Australia to date.

The study aim was to more objectively understand causes of geographical CVD mortality disparities by; (a) comparing measures of CVD risk (objective and self-reported data) between GGT and NWA; (b) comparing CVD mortality rates between GGT and NWA and other areas Australia-wide and (c) describing the relationship between socioeconomic status (SES) and CVD mortality rates.

We hypothesised that 1) higher mortality rates would be observed in GGT than NWA and that 2) these would be influenced by worse CVD risk factor profiles in the former.

METHODS

Study design

This study compared CVD risk factor data (individual as well as absolute 5-year CVD risk) from two studies - a regional cross-sectional population survey and an urban longitudinal cohort - conducted over a similar time period. In addition, Australian Bureau of Statistics (ABS) CVD mortality rates in different geographical locations were compared and the relationship between mortality and SES explored.

Population and sample

Comparing measures of CVD risk

Details of the methodology of both studies have been published elsewhere [7-11]. Below is a brief summary of the setting, population and sample.

Greater Green Triangle Risk Factor Study

GGT encompasses a population of 225,000 in south-east South Australia and south-west Victoria. The Greater Green Triangle Risk Factor Study (GGT RFS) comprised three cross-sectional population surveys (Limestone Coast, Corangamite and Wimmera Shire Risk Factor Surveys) conducted between 2004 and 2006. In total, 1563 randomly selected persons aged 25-74 provided some information (self-administered questionnaire +/- attendance at survey site for anthropometric and biomedical measurements including fasting venous blood specimens for lipids and glucose). Socioeconomic indicators of GGT RFS participants compared with available population statistics indicated that the survey population closely represented the overall GGT population [7].

North West Adelaide Health Study

Adelaide, the capital of South Australia, has a population of 1.18 million [12]. The northern and western suburbs, stretching from Glenelg to Gawler, encompass approximately half of Adelaide's population and one-third of the South Australian population. The North West Adelaide Health Study (NWAHS) is a largely

representative cohort of over 4000 randomly selected adults aged 18 and over recruited from NWA between 2000 and 2003 (Stage 1) returning between 2004 and 2006 (Stage 2). Each stage included a telephone survey, self-administered questionnaire and anthropometric and bio-medical examination. NWAHS Stage 1 participants had some demographic differences but no health risk behaviour differences compared with ABS 2006 census data and South Australian Surveillance and Monitoring System data [13].

In this study, participants aged 25-74 were used in order to make the age range of both populations comparable. From NWAHS, only Stage 2 participants were used and 3036 provided information.

Sources and measures

Comparing measures of CVD risk

Demographic characteristics have been reported previously and are presented in Table 1 [14]. A comprehensive examination of methodologies and questionnaire wordings of both studies had been undertaken in order to ensure that variables were comparable. Some aspects could not be compared due to differences in questions used such a household income, levels of alcohol consumption, physical activity and quality of life. GGT RFS participant age was calculated from the survey date after assuming each individual was born on June 30 in their given year of birth. NWAHS participant age was calculated from their date of birth and clinic appointment date and truncated.

Five-year absolute CVD risk, defined as IHD and stroke collectively, was calculated using the Framingham equation which is used to make Australian cardiovascular event risk charts [15]. Calculation of CVD risk was restricted to participants aged 35-74 who reported no history of heart attack or stroke. Biomedical measurements required for use of the equation were available from both studies. Smoking status was determined by self-report. Diabetes was defined as having a survey fasting plasma glucose level of 7.0mmol/L or above and/or having self-reported diabetes. As the questionnaire used in GGT RFS asked whether a participant had ever been diagnosed with impaired glucose tolerance, participants who responded positively were considered to have diabetes. As no electrocardiogram information was available for any participants, the left ventricular hypertrophy variable was excluded from the risk calculation.

Comparing CVD mortality outcomes

Mortality rates were obtained using 2003-2007 ABS mortality (numerator) and Estimated Residential Population (ERP, denominator) data according to relevant 2006 Statistical Local Area (SLA) codes [16-17]. ABS Australian Standard Geographical Classification System (ASGC) for Remoteness Areas uses categories major cities, inner regional, outer regional, remote and very remote [17]. Thirty-one SLA codes representing GGT (n=13) and NWA (n=18) were used. According to ASGC all GGT SLAs were classified as inner or outer regional and all NWA SLAs as major city areas. In this comparative study 'inner and outer regional' areas consisted of all areas in this ASGC category combined, but excluded GGT SLAs. 'Remote and very remote' areas represented all such ASGC areas combined. 'Major cities' included all Australian cities classified as such by the ASGC, excluding NWA SLAs. Mortality information was extracted according to predefined International Classification of Diseases (ICD) 10 codes [5]. ICD 10 codes I20-I25 and I61-I64 were used to make up the category IHD and Stroke.

Relationship between SES and CVD mortality rates

SES was measured using IRSD (Index of Relative Socio-economic Disadvantage). IRSD is one of four ABS Socio-Economic Indexes For Areas (SEIFA), which are area-based summary measures of relative socio-economic disadvantage [18]. IRSD takes into account a range of variables including education, employment and financial well-being. Although area and individual-level SES may have independent effects on health outcomes, only area-level SES was taken into account.

The distribution of IRSD scores between GGT and NWA SLAs were compared and the relationship between IRSD and CVD mortality rates explored.

Analyses

Statistical analyses were undertaken using Stata V12 and IBM SPSS Statistics V19. CVD risk factor data for participants are reported as mean values with standard errors for continuous variables and proportions with 95% confidence intervals (using the Agresti-Coull technique) for discrete variables. Independent sample t-tests were used to assess differences between means (α=0.05), with the Welsch method applied when the assumption of homogenous variance was not met. Chi-Square ($\chi 2$) tests were used to assess differences between proportions (α=0.05). The relationship between mortality rates and IRSD scores was examined using linear regression.

Ethics

Ethics approval for GGT RFS was received from the Flinders Clinical Research Ethics Committee, Adelaide, approval number 207/034. Ethics approval for NWAHS Stage 2 was received from The Queen Elizabeth Hospital (TQEH) Human Research Ethics Committee (HREC), Adelaide, approval number 2004030. HREC approval for comparison analysis was given by the University of South Australia, Adelaide, and the Queensland University of Technology, Brisbane, approval numbers P136/09 and 1000000988.

RESULTS

Demographic characteristics of participants

NWAHS participants were younger, more diverse in their country of origin, more likely to be single, separated or divorced and less likely to be in part time or casual employment than GGT RFS participants (Table 1).

Table 1: Demographic characteristics of participants by location

	NWAHS			GGT RFS			
	n	**%**	**95% CI**	**n**	**%**	**95% CI**	
Demographics							
Sex							
Male	1437	50.2	(48.0 - 52.4)	714	50.2	(46.9 - 53.5)	
Female	1426	49.8	(47.6 - 52.0)	708	49.8	(46.5 - 53.1)	
Age							
25 to 44 years	1412	49.3	(47.1 -51.6)	599	42.1	(38.6 - 45.7)	*
45 to 54 years	620	21.6	(20.1 - 23.3)	350	24.6	(22.3 - 27.1)	*
55 to 64 years	477	16.7	(15.4 - 18.0)	277	19.5	(17.6 - 21.5)	*
65 to 74 years	355	12.4	(11.3 - 13.6)	196	13.8	(12.4 - 15.3)	
Aboriginal or Torres Strait Islander							
No	2785	97.3	(96.5 - 97.8)	1405	98.8	(98.1 - 99.3)	*
Yes	13	0.4	(0.2 - 0.8)	8	0.6	(0.3 - 1.1)	
Country of birth							
Australia or New Zealand	2064	72.1	(70.2 - 73.9)	1339	94.1	(92.8 - 95.2)	*
UK or Ireland	451	15.8	(14.4 - 17.3)	27	1.9	(1.4 - 2.6)	*
Europe	223	7.8	(6.8 - 8.9)	26	1.8	(1.4 - 2.5)	*
Other	116	4.0	(3.2 - 5.1)	28	2.0	(1.3 - 3.0)	*
Highest level of education obtained							
Secondary school or lower	1568	57.9	(55.5 - 60.3)	920	64.7	(61.4 - 67.9)	*
Trade / Apprenticeship / Certificate / Diploma / Vocational training (TAFE/VET)	651	24.1	(22.0 - 26.3)	254	17.9	(15.3 - 20.8)	*
Bachelor degree or higher	460	17.0	(15.1 - 19.1)	229	16.1	(13.7 - 18.8)	
Marital Status							
Married or living with a partner	1988	73.5	(71.2 - 75.6)	1198	84.2	(81.8 - 86.4)	*
Separated or divorced	252	9.3	(8.3 - 10.5)	86	6.0	(4.8 - 7.6)	*
Widowed	77	2.9	(2.4 - 3.4)	46	3.2	(2.6 - 4.0)	
Never married (single)	381	14.1	(12.1 - 16.3)	91	6.4	(4.8 - 8.5)	*
Work Status							
Full time employed	1352	50.0	(47.5 - 52.4)	680	47.8	(44.5 - 51.1)	
Part time / Casual employment	514	19.0	(17.2 - 20.9)	327	23.0	(20.2 - 26.0)	*
Unemployed	58	2.2	(1.6 - 2.9)	43	3.0	(2.2 - 4.3)	
Home duties	304	11.2	(9.9 - 12.7)	126	8.8	(7.1 - 11.0)	*
Retired	378	14.0	(12.8 - 15.2)	209	14.7	(13.1 - 16.4)	
Student	27	1.0	(0.6 - 1.8)	3	0.2	(0.06 - 0.7)	#
Other	64	2.4	(1.8 - 3.0)	11	0.8	(0.4 - 1.6)	*
TOTAL	2864	100%		1422	100%		

Note: The weighting of the data can result in rounding discrepancies or totals not adding.

#Insufficient numbers for a statistical test.
**Statistically significantly different (χ^2 test, $p < 0.05$) GGT RFS compared with NWAHS.*
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Comparing measures of CVD risk

Framingham 5-year absolute CVD risk scores were not significantly different between GGT RFS and NWAHS participants (age-specific groups and overall, Figure 1(a)).

[Insert Figures 1(a) and 1(b) here]

There were some differences in individual CVD risk factors after standardising to the 2006 Australian population but the magnitude of differences were small (Table 2). NWAHS participants had a lower mean systolic blood pressure and higher mean diastolic blood pressure than GGT RFS participants. HDL cholesterol was lower in NWAHS (men and overall). Total triglycerides were higher in NWAHS overall (though not quite reaching statistical significance) yet lower in NWA women. NWA men had higher BMI and waist circumference. NWAHS participants (women and overall) were more likely to be smokers. Prevalence of diabetes/IGT was higher in NWAHS (men and overall).

Table 2: Individual CVD risk factor data by location

		NWAHS Mean (SE, N)	GGT RFS Mean (SE, N)	*p*-value
Mean systolic blood pressure (mmHg)		123.37 (0.31, 2639)	126.00 (0.48, 1419)	**<0.001**
	Men	126.71 (0.43, 1302)	128.60 (0.64, 700)	**0.014**
	Women	120.12 (0.44, 1337)	123.47 (0.69, 719)	**<0.001**
Mean diastolic blood pressure (mmHg)		80.55 (0.20, 2639)	76.06 (0.29, 1418)	**<0.001**
	Men	83.80 (0.26, 1302)	79.27 (0.40, 700)	**<0.001**
	Women	77.39 (0.26, 1337)	72.93(0.39, 718)	**<0.001**
Total cholesterol (mmol/L)		5.37 (0.02, 2647)	5.37 (0.03, 1377)	0.903
	Men	5.38 (0.03, 1299)	5.39 (0.04, 680)	0.887
	Women	5.36 (0.03, 1348)	5.34 (0.04, 697)	0.742
LDL cholesterol (mmol/L)		3.26 (0.02, 2554)	3.22 (0.03, 1353)	0.171
	Men	3.31 (0.03, 1221)	3.30 (0.04, 658)	0.852
	Women	3.22 (0.02, 1333)	3.14 (0.04, 694)	0.071
HDL cholesterol (mmol/L)		1.43 (0.01, 2647)	1.46 (0.01, 1377)	**0.003**
	Men	1.28 (0.01, 1299)	1.33 (0.01, 680)	**0.001**
	Women	1.56 (0.01, 1348)	1.59 (0.01, 697)	0.148
Total-C/HDL-C ratio		3.97 (0.02, 2647)	3.93 (0.04, 1377)	0.328
	Men	4.38 (0.03, 1299)	4.31 (0.06, 680)	0.298
	Women	3.58 (0.03, 1348)	3.56 (0.04, 697)	0.657
LDL-C/HDL-C ratio		2.40 (0.02, 2554)	2.37 (0.03, 1353)	0.388
	Men	2.66 (0.02, 1221)	2.63 (0.04, 658)	0.585
	Women	2.16 (0.02, 1333)	2.13 (0.03, 694)	0.351
Total triglycerides (mmol/L)		1.55 (0.03, 2647)	1.48 (0.03, 1322)	0.065†

	Men	1.83 (0.05, 1299)	1.64 (0.04, 650)	0.262†
	Women	1.28 (0.03, 1348)	1.33 (0.03, 673)	**<0.001†**
BMI (kg/m2)		28.30 (0.11, 2658)	28.00 (0.15, 1413)	0.089
	Men	28.51 (0.14, 1308)	28.05 (0.18, 699)	**0.043**
	Women	28.09 (0.17, 1349)	27.92 (0.23, 714)	0.545
Waist circumference (cm)				
	Men	99.77 (0.38, 1302)	97.85 (0.48, 695)	**0.002**
	Women	87.71 (0.39, 1337)	88.07 (0.55, 714)	0.587
		NWAHS	GGT RFS	
		%, 95% CI (n)	**%, 95% CI (n)**	***p*-value**
Current smokers		21.35, 19.83-22.95 (2642)	17.79, 15.88-19.88 (1405)	**0.028**
	Men	22.75, 20.55-25.11 (1301)	20.26, 17.44-23.41(696)	0.302
	Women	19.99, 17.93-22.21 (1341)	15.37, 12.90-18.22 (709)	**0.032**
Known diabetes or Impaired Glucose Tolerance (IGT)		7.72, 6.76-8.80 (2656)	5.84, 4.73-7.18 (1422)	**0.037**
	Men	8.34, 6.96-9.97 (1307)	5.14, 3.72-7.06 (700)	**0.014**
	Women	7.19, 5.92-8.69 (1350)	6.38, 4.80-8.42 (721)	0.520

Note: The weighting of the data can result in rounding discrepancies or totals not adding.
†p-values based on log of the variable in order to address right skewedness of data.

Comparing CVD mortality outcomes

Figure 1(b) shows the relationship between IHD and Stroke mortality and age for GGT and NWA. Table 3 compares IHD and Stroke mortality rates between different regions of interest. IHD and Stroke mortality in inner and outer regional areas was generally worse than in major cities (p<0.001). Remote and very remote areas had significantly higher mortality rates than all other categories (p<0.001).

Table 3: Comparison of ischaemic heart disease (IHD) and stroke mortality rates by age group (Source: 2003-2007 Australian Bureau of Statistics)

Age Group (years)	**35-39**	**40-44**	**45-49**	**50-54**	**55-59**	**60-64**	**65-69**	**70-74**	**35-74: Crude**
NWA	9 (5-16)	19 (13-28)	34 (26-46)	62 (49-78)	99 (81-120)	157 (132-186)	281 (246-322)	419 (373-470)	103 (96-110)
GGT	5 (0-20)	14 (6-31)	28 (16-50)	44 (27-71)	99 (70-139)	130 (94-180)	224 (171-292)	488 (402-592)	98 (87-111)
NWA vs. GGT	*p=0.427*	*p=0.511*	*p=0.562*	*p=0.208*	*p=0.987*	*p=0.319*	*p=0.130*	*p=0.182*	*p=0.489*
Major Cities (Ex NWA)	6 (5-6)	12 (11-13)	23 (22-24)	38 (37-40)	63 (61-66)	110 (107-114)	199 (193-205)	369 (361-377)	70 (69-71)
NWA vs.	*p=0.061*	*p=0.010**	*p=0.006**	*p=0.000**	*p=0.000**	*p=0.000**	*p=0.000**	*p= 0.028**	

Major Cities (Ex NWA)									*p<0.001**
Inner and Outer Regional (Ex GGT)	8 (7-10)	16 (15-18)	30 (28-32)	47 (44-50)	75 (71-79)	131 (125-137)	230 (222-238)	440 (428-453)	92 (91-94)
GGT vs. Inner and Outer Regional (Ex GGT)	*p=0.492*	*p=0.714*	*p=0.872*	*p=0.768*	*p=0.097*	*p=0.991*	*p=0.840*	*p=0.295*	*p=0.341*
Remote and Very Remote	36 (28-47)	48 (39-60)	76 (64-91)	82 (68-99)	138 (118-162)	210 (181-243)	345 (300-395)	593 (524-671)	125 (118-132)

Age specific IHD and Stroke Mortality Rates per 100,000 population (95% Confidence Intervals) by age group. Mean of all deaths from 2003-2007
**Statistically significantly different (χ^2 test, $p < 0.05$).*

In all age groups GGT mortality rates were representative of those of inner and outer regional areas (crude mortality rates for 35-74 years: inner and outer regional versus GGT 92 versus 98, p=0.341). NWA mortality was generally higher than in other major Australian cities (crude mortality rates for 35-74 years: major cities versus NWA 70 versus 103, p=0.028). GGT and NWA mortality rates did not differ significantly despite NWA being a major city location (crude mortality rates for 35-74 years: GGT versus NWA p=0.489).

Relationship between SES and CVD mortality rates

A comparison of IRSD scores using an independent samples median test indicated no significant difference between the two study areas (p=0.108). However there was a significant difference in the distribution of IRSD scores (p=0.022), with scores in NWA skewed towards the lower end of the scale (Figure 2(a)).

Increasing mortality was consistently associated with lower IRSD scores. When age-specific mortality rates for age class 35-74 were plotted against IRSD (Figure 2(b)), both study areas were most closely aligned with inner and outer regional areas. Closer inspection of study areas at the SLA level indicated that the trend remained. In NWA (Figure 2(c)) IRSD explained around 46% (n=18, β=-0.389) of the variation in mortality. In GGT (Figure 2(d)) IRSD explained approximately 19% (n=13, β=-0.477) of the variation in mortality, although the relationship was not statistically significant.

[Insert Figures 2(a), 2(b), 2(c) and 2(d) here]

DISCUSSION

Geographic and socioeconomic disparities in CVD mortality were first described in Australia in the late 1990s, [19] and initiated debate about likely explanations. Socioeconomic and cultural diversity between regions, differential prevalence of CVD risk factors, and variations in patterns of medical care were postulated as potential causative factors. This work started a debate about the most appropriate actions both within and outside the healthcare system to address these disparities [20-21]. Progress since has been slow in advancing our understanding of these issues, impeded by the lack of comprehensive, high quality data on CVD risk factor prevalence across the Australian population.

Based on AIHW published data,[3, 4, 6] and the only previous Australian study to analyse the contribution of CVD risk factor prevalence differences to the rural/regional–urban CVD mortality gap [22], our original hypothesis in this study was that GGT CVD risk factor profiles, and CVD mortality, would be worse than in NWA. Unexpectedly, GGT and NWA were similar in terms of absolute CVD risk scores, individual CVD risk factors and mortality rates. Furthermore, mortality rates in the regional GGT population are consistent with those observed in most regional areas of Australia, but lower than in remote areas, and higher than in the overall Australian metropolitan population. CVD mortality rates in the metropolitan NWA population are significantly higher than in the overall Australian metropolitan population.

Social gradients in health – 'caused by unequal distribution of power, income, goods and services' lead to inequitable health outcomes within and between populations [1]. Poorer Australians have worse CVD outcomes [23]. This was demonstrated in our study by the strong relationship between IRSD and CVD mortality at a national level (Figure 2(b)) as well as within NWA (Figure 2(c)). The trend was present within GGT (Figure 2(d)) although statistically non-significant. This can likely be explained by limited sample size coupled with a relatively narrow range of IRSD scores compared with NWA. These findings are consistent with other evidence in the Australian literature and from other developed countries regarding the association between low SES and increased levels of CVD risk factors, morbidity and mortality [21].

The influence of a broad range of social determinants (for example, quality of housing, employment, income

level, education etc.) on biological determinants of CVD, as well as differential access to health-promoting services may explain a significant part of the rural/regional-urban divide in CVD mortality in Australia. There is also growing evidence that variation in implementation of evidence based CVD care across geographic, institutional and even subspecialty boundaries may be an important determinant [24-25]. Implementation of evidence-based practice may provide an opportunity to reduce disparities in CVD outcomes, including geographically determined disparities, at relatively low cost and in shorter time frames than those required to address socioeconomic disparities across large populations.

All of the aforementioned variables and their relationship with CVD health outcomes are complex, yet all should be taken into account when formulating strategies to address inequalities.

Our study has limitations. Firstly, there are difficulties in extrapolating results from single rural and urban populations. This regional study population is relatively culturally and socioeconomically homogenous and probably representative of many (but not all) regional areas in Australia. The urban population is more culturally and socioeconomically diverse with over-representation of the socioeconomically disadvantaged compared with the overall Australian urban population. Secondly, we were unable to directly analyse associations between risk factors, SES and CVD mortality in the sample data sets due to the cross sectional rather than longitudinal design of the two population-based risk factor studies and other methodological differences in sampling and data collection. Time frames influencing some cross-sectional measured risk factor variables, compared with those operating over whole lifetimes to determine clinical outcomes such as CVD mortality, are different and we cannot be sure that they are stable or changing at the same rate in two geographically distinct populations. Some such variables which were not measured in our study, such as population levels of salt intake, may have resulted in the difference in systolic and diastolic blood pressures in our two study groups. However, we think that the most likely explanation for this observation is inter-observer variation in the measurement of blood pressure.

Strategies for comprehensive, high quality CVD risk factor surveillance should cover all population groups, regardless of geography or SES. Preferably, there should be longitudinal follow up, combined with appropriate epidemiological and health services research to investigate which interventions are most cost

effectively able to reduce disparities in CVD outcomes in the specific context of each of our social and health care systems.

Legend of Figures

Figure 1: Framingham absolute CVD risk and IHD and stroke mortality rates by age

Figure 2: Relationship between IHD and stroke mortality and IRSD

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out the Wimmera Shire part of GGT risk factor prevalence study.

CONTRIBUTORSHIP STATEMENT

PT conceived and designed the study. AT is Epidemiological Principal Investigator of the North West Adelaide Health Study. JAD was the chief investigator for the GGT DPP.

AT, RC, AM, JG and JAD were involved in acquisition of data. BP, EJ, EP and VV analysed and interpreted the data. EP and EJ drafted the manuscript and were responsible for its revisions. VV and RC helped to draft the manuscript. All authors contributed to specific sections in the manuscript. All authors read and approved the final manuscript.

DATA SHARING STATEMENT

There is no additional data publicly available. For further enquiries on accessing data please contact Prof. James Dunbar at director@greaterhealth.org and Janet Grant at janet.grant@adelaide.edu.au

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TITLE	*A comparison of Australian rural and metropolitan cardiovascular risk and mortality: the Greater Green Triangle and North West Adelaide Population Surveys.*
TYPE OF ARTICLE	Research

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ARTICLE SUMMARY

ARTICLE FOCUS

- The study aim was to more objectively understand causes of geographical cardiovascular disease (CVD) mortality disparities in Australia by; (a) comparing measures of CVD risk (objective and self-reported data) between a rural population (Greater Green Triangle, GGT) and urban population (North West Adelaide, NWA)
- (b) comparing CVD mortality rates between GGT and NWA and other areas Australia-wide and
- (c) describing the relationship between socioeconomic status (SES) and CVD mortality rates.

KEY MESSAGES

- This study supports existing evidence of a social gradient in cardiovascular health.
- This study provides evidence to reject the assertion that location of residence in Australia necessarily results in poorer cardiovascular health.

STRENGTHS AND LIMITATIONS OF THIS STUDY

- This is the first comparison of both self-report and biomedical data from a wholly rural/regional Australian population study with a metropolitan population study.
- Determinants of cardiovascular health are contextual, and the study populations will not necessarily represent rural and urban populations more generally in Australia.
- Direct analysis of associations between risk factors, SES and CVD mortality in the sample data sets was not possible due to the cross-sectional rather than longitudinal design of the two population-based risk factor studies and other methodological differences in sampling and data collection.

ABSTRACT

Background: Cardiovascular (CVD) mortality disparities between rural/regional and urban-dwelling residents of Australia are persistent. Unavailability of biomedical CVD risk factor data has, until now, limited efforts to understand the causes of the disparity.

Methods: This study investigated rural/regional-urban CVD mortality disparities by comparing (a) CVD risk measures between a regional population (Greater Green Triangle Risk Factor (cross-sectional) Study 2004-2006 (GGT RFS, n =1563)), and an urban population (North West Adelaide Health (longitudinal cohort) Study 2004-2006) (NWAHS Stage 2, n=3036)) (b) Australian Bureau of Statistics (ABS) CVD mortality rates between these and other Australian regions and (c) ABS CVD mortality rates by an area-level indicator of socioeconomic status, the Index of Relative Socioeconomic Disadvantage (IRSD).

Results: Few significant differences in CVD risk between the study regions, with absolute CVD risk ranging from approximately 5% to 30% in the 35-39 and 70-74 age groups respectively. Similar mean 2003-2007 mortality rates in the Greater Green Triangle (GGT) region (98), the North West Adelaide (NWA) region (103) and regional Australia (92). NWA mortality rates exceeded that of other city areas (70). Lower measures of SES were associated with worse CVD outcomes regardless of geographic location.

Conclusions: Metropolitan areas do not always have better CVD risk factor profiles and outcomes than rural/regional areas. Needs assessments are required for different settings to elucidate relative contributions of the multiple determinants of risk and the appropriate cardiac health care strategies to improve outcomes.

MAIN TEXT

INTRODUCTION

Place of residence is an important determinant of health. In many settings worldwide, there is an underinvestment in health-promoting infrastructure and opportunities in rural communities leading to urban migration and geographical health inequalities [1]. Australia is a highly urbanised country with approximately two-thirds of the population living in major cities [2]. Well-documented health inequalities exist between regional and remote versus urban settings. In the former, life expectancy is 1-7 years lower and decreases with increasing remoteness [3]. An approximate 10% difference in all-cause mortality rates has been consistently documented between major cities and the rest of Australia [4].

As in many other countries, cardiovascular disease (CVD) - principally ischaemic heart disease (IHD) and cerebrovascular disease - is the largest contributor to overall mortality in Australia [5]. Coronary heart disease and 'other' circulatory diseases are the two largest contributors to the excess mortality observed outside major city areas (20% and 17% of the excess mortality between 2002 and 2004) [4]. Measuring contributions of biological and behavioural risk factors, social and economic determinants, access to quality care and broader politico-structural influences on CVD health outcomes in Australia has proved difficult, especially in rural areas.

A recent Australian Institute of Health and Welfare report found that prevalence of key CVD risk factors increases with increasing remoteness from major city areas [6]. Such self-report data, however, has limitations. Despite the obvious need for more objectively measured population data, very little risk factor data in the form of biomedical measurements is available for comparative studies between remote, regional and urban areas. Better evidence is required to develop strategies to address inequalities.

This paper reports on absolute CVD risk from two population biomedical surveys covering a regional area (Greater Green Triangle, GGT) and metropolitan area (North-west Adelaide, NWA), along with CVD mortality rates from corresponding regions drawn from national data records. To our knowledge, it is the

only comparative study of measured biomedical risk factors and mortality data between specifically regional and urban populations in Australia to date.

The study aim was to more objectively understand causes of geographical CVD mortality disparities by; (a) comparing measures of CVD risk (objective and self-reported data) between GGT and NWA; (b) comparing CVD mortality rates between GGT and NWA and other areas Australia-wide and (c) describing the relationship between socioeconomic status (SES) and CVD mortality rates.

We hypothesised that 1) higher mortality rates would be observed in GGT than NWA and that 2) these would be influenced by worse CVD risk factor profiles in the former.

METHODS

Study design

This study compared CVD risk factor data (individual as well as absolute 5-year CVD risk) from two studies - a regional cross-sectional population survey and an urban longitudinal cohort - conducted over a similar time period. In addition, Australian Bureau of Statistics (ABS) CVD mortality rates in different geographical locations were compared and the relationship between mortality and SES explored.

Population and sample

Comparing measures of CVD risk

Details of the methodology of both studies have been published elsewhere [7-11]. Below is a brief summary of the setting, population and sample.

Greater Green Triangle Risk Factor Study

GGT encompasses a population of 225,000 in south-east South Australia and south-west Victoria. The Greater Green Triangle Risk Factor Study (GGT RFS) comprised three cross-sectional population surveys (Limestone Coast, Corangamite and Wimmera Shire Risk Factor Surveys) conducted between 2004 and 2006. In total, 1563 randomly selected persons aged 25-74 provided some information (self-administered questionnaire +/- attendance at survey site for anthropometric and biomedical measurements including fasting venous blood specimens for lipids and glucose). Socioeconomic indicators of GGT RFS participants compared with available population statistics indicated that the survey population closely represented the overall GGT population [7].

North West Adelaide Health Study

Adelaide, the capital of South Australia, has a population of 1.18 million [12]. The northern and western suburbs, stretching from Glenelg to Gawler, encompass approximately half of Adelaide's population and one-third of the South Australian population. The North West Adelaide Health Study (NWAHS) is a largely

representative cohort of over 4000 randomly selected adults aged 18 and over recruited from NWA between 2000 and 2003 (Stage 1) returning between 2004 and 2006 (Stage 2). Each stage included a telephone survey, self-administered questionnaire and anthropometric and bio-medical examination. NWAHS Stage 1 participants had some demographic differences but no health risk behaviour differences compared with ABS 2006 census data and South Australian Surveillance and Monitoring System data [13].

In this study, participants aged 25-74 were used in order to make the age range of both populations comparable. From NWAHS, only Stage 2 participants were used and 3036 provided information.

Sources and measures

Comparing measures of CVD risk

Demographic characteristics have been reported previously and are presented in Table 1 [14]. A comprehensive examination of methodologies and questionnaire wordings of both studies had been undertaken in order to ensure that variables were comparable. Some aspects could not be compared due to differences in questions used such a household income, levels of alcohol consumption, physical activity and quality of life. GGT RFS participant age was calculated from the survey date after assuming each individual was born on June 30 in their given year of birth. NWAHS participant age was calculated from their date of birth and clinic appointment date and truncated.

Five-year absolute CVD risk, defined as IHD and stroke collectively, was calculated using the Framingham equation which is used to make Australian cardiovascular event risk charts [15]. Calculation of CVD risk was restricted to participants aged 35-74 who reported no history of heart attack or stroke. Biomedical measurements required for use of the equation were available from both studies. Smoking status was determined by self-report. Diabetes was defined as having a survey fasting plasma glucose level of 7.0mmol/L or above and/or having self-reported diabetes. As the questionnaire used in GGT RFS asked whether a participant had ever been diagnosed with impaired glucose tolerance, participants who responded positively were considered to have diabetes. As no electrocardiogram information was available for any participants, the left ventricular hypertrophy variable was excluded from the risk calculation.

Comparing CVD mortality outcomes

Mortality rates were obtained using 2003-2007 ABS mortality (numerator) and Estimated Residential Population (ERP, denominator) data according to relevant 2006 Statistical Local Area (SLA) codes [16-17]. ABS Australian Standard Geographical Classification System (ASGC) for Remoteness Areas uses categories major cities, inner regional, outer regional, remote and very remote [17]. Thirty-one SLA codes representing GGT (n=13) and NWA (n=18) were used. According to ASGC all GGT SLAs were classified as inner or outer regional and all NWA SLAs as major city areas. In this comparative study 'inner and outer regional' areas consisted of all areas in this ASGC category combined, but excluded GGT SLAs. 'Remote and very remote' areas represented all such ASGC areas combined. 'Major cities' included all Australian cities classified as such by the ASGC, excluding NWA SLAs. Mortality information was extracted according to predefined International Classification of Diseases (ICD) 10 codes [5]. ICD 10 codes I20-I25 and I61-I64 were used to make up the category IHD and Stroke.

Relationship between SES and CVD mortality rates

SES was measured using IRSD (Index of Relative Socio-economic Disadvantage). IRSD is one of four ABS Socio-Economic Indexes For Areas (SEIFA), which are area-based summary measures of relative socio-economic disadvantage [18]. IRSD takes into account a range of variables including education, employment and financial well-being. Although area and individual-level SES may have independent effects on health outcomes, only area-level SES was taken into account.

The distribution of IRSD scores between GGT and NWA SLAs were compared and the relationship between IRSD and CVD mortality rates explored.

Analyses

Statistical analyses were undertaken using Stata V12 and IBM SPSS Statistics V19. CVD risk factor data for participants are reported as mean values with standard errors for continuous variables and proportions with 95% confidence intervals (using the Agresti-Coull technique) for discrete variables. Independent sample t-tests were used to assess differences between means (α=0.05), with the Welsch method applied when the assumption of homogenous variance was not met. Chi-Square ($\chi 2$) tests were used to assess differences between proportions (α=0.05). The relationship between mortality rates and IRSD scores was examined using linear regression.

Ethics

Ethics approval for GGT RFS was received from the Flinders Clinical Research Ethics Committee, Adelaide, approval number 207/034. Ethics approval for NWAHS Stage 2 was received from The Queen Elizabeth Hospital (TQEH) Human Research Ethics Committee (HREC), Adelaide, approval number 2004030. HREC approval for comparison analysis was given by the University of South Australia, Adelaide, and the Queensland University of Technology, Brisbane, approval numbers P136/09 and 1000000988.

RESULTS

Demographic characteristics of participants

NWAHS participants were younger, more diverse in their country of origin, more likely to be single, separated or divorced and less likely to be in part time or casual employment than GGT RFS participants (Table 1). ~~A slightly lower proportion of NWAHS participants identified as being of Aboriginal/Torres Strait Islander origin.~~

Table 1: Demographic characteristics of participants by location

	NWAHS			GGT RFS			
	n	%	95% CI	n	%	95% CI	
Demographics							
Sex							
Male	1437	50.2	(48.0 - 52.4)	714	50.2	(46.9 - 53.5)	
Female	1426	49.8	(47.6 - 52.0)	708	49.8	(46.5 - 53.1)	
Age							
25 to 44 years	1412	49.3	(47.1 -51.6)	599	42.1	(38.6 - 45.7)	*
45 to 54 years	620	21.6	(20.1 - 23.3)	350	24.6	(22.3 - 27.1)	*
55 to 64 years	477	16.7	(15.4 - 18.0)	277	19.5	(17.6 - 21.5)	*
65 to 74 years	355	12.4	(11.3 - 13.6)	196	13.8	(12.4 - 15.3)	
Aboriginal or Torres Strait Islander							
No	2785	97.3	(96.5 - 97.8)	1405	98.8	(98.1 - 99.3)	*
Yes	13	0.4	(0.2 - 0.8)	8	0.6	(0.3 - 1.1)	
Country of birth							
Australia or New Zealand	2064	72.1	(70.2 - 73.9)	1339	94.1	(92.8 - 95.2)	*
UK or Ireland	451	15.8	(14.4 - 17.3)	27	1.9	(1.4 - 2.6)	*
Europe	223	7.8	(6.8 - 8.9)	26	1.8	(1.4 - 2.5)	*
Other	116	4.0	(3.2 - 5.1)	28	2.0	(1.3 - 3.0)	*
Highest level of education obtained							
Secondary school or lower	1568	57.9	(55.5 - 60.3)	920	64.7	(61.4 - 67.9)	*
Trade / Apprenticeship / Certificate / Diploma / Vocational training (TAFE/VET)	651	24.1	(22.0 - 26.3)	254	17.9	(15.3 - 20.8)	*
Bachelor degree or higher	460	17.0	(15.1 - 19.1)	229	16.1	(13.7 - 18.8)	
Marital Status							
Married or living with a partner	1988	73.5	(71.2 - 75.6)	1198	84.2	(81.8 - 86.4)	*
Separated or divorced	252	9.3	(8.3 - 10.5)	86	6.0	(4.8 - 7.6)	*
Widowed	77	2.9	(2.4 - 3.4)	46	3.2	(2.6 - 4.0)	
Never married (single)	381	14.1	(12.1 - 16.3)	91	6.4	(4.8 - 8.5)	*
Work Status							
Full time employed	1352	50.0	(47.5 - 52.4)	680	47.8	(44.5 - 51.1)	
Part time / Casual employment	514	19.0	(17.2 - 20.9)	327	23.0	(20.2 - 26.0)	*
Unemployed	58	2.2	(1.6 - 2.9)	43	3.0	(2.2 - 4.3)	
Home duties	304	11.2	(9.9 - 12.7)	126	8.8	(7.1 - 11.0)	*
Retired	378	14.0	(12.8 - 15.2)	209	14.7	(13.1 - 16.4)	
Student	27	1.0	(0.6 - 1.8)	3	0.2	(0.06 - 0.7)	#
Other	64	2.4	(1.8 - 3.0)	11	0.8	(0.4 - 1.6)	*
TOTAL	2864	100%		1422	100%		

~~*Data source: North West Adelaide Health Study Stage 2, 2004-2006, 25 to 74 years. Greater Green Triangle Risk Factor Study, 2004-2006, 25 to 74 years.*~~

Note: The weighting of the data can result in rounding discrepancies or totals not adding.
#Insufficient numbers for a statistical test.
**Statistically significantly different (χ^2 test, $p < 0.05$) GGT RFS compared with NWAHS.*
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Comparing measures of CVD risk

Framingham 5-year absolute CVD risk scores were not significantly different between GGT RFS and NWAHS participants (age-specific groups and overall, Figure 1(a)).

[Insert Figures 1(a) and 1(b) here]

There were some differences in individual CVD risk factors after standardising to the 2006 Australian population but the magnitude of differences were small (Table 2). NWAHS participants had a lower mean systolic blood pressure and higher mean diastolic blood pressure than GGT RFS participants. HDL cholesterol was lower in NWAHS (men and overall). Total triglycerides were higher in NWAHS overall (though not quite reaching statistical significance) yet lower in NWA women. NWA men had higher BMI and waist circumference. NWAHS participants (women and overall) were more likely to be smokers. Prevalence of diabetes/IGT was higher in NWAHS (men and overall).

Table 2: Individual CVD risk factor data by location

		NWAHS Mean (SE, N)	GGT RFS Mean (SE, N)	*p*-value
Mean systolic blood pressure (mmHg)		123.37 (0.31, 2639)	126.00 (0.48, 1419)	**<0.001**
	Men	126.71 (0.43, 1302)	128.60 (0.64, 700)	**0.014**
	Women	120.12 (0.44, 1337)	123.47 (0.69, 719)	**<0.001**
Mean diastolic blood pressure (mmHg)		80.55 (0.20, 2639)	76.06 (0.29, 1418)	**<0.001**
	Men	83.80 (0.26, 1302)	79.27 (0.40, 700)	**<0.001**
	Women	77.39 (0.26, 1337)	72.93(0.39, 718)	**<0.001**
Total cholesterol (mmol/L)		5.37 (0.02, 2647)	5.37 (0.03, 1377)	0.903
	Men	5.38 (0.03, 1299)	5.39 (0.04, 680)	0.887
	Women	5.36 (0.03, 1348)	5.34 (0.04, 697)	0.742
LDL cholesterol (mmol/L)		3.26 (0.02, 2554)	3.22 (0.03, 1353)	0.171
	Men	3.31 (0.03, 1221)	3.30 (0.04, 658)	0.852
	Women	3.22 (0.02, 1333)	3.14 (0.04, 694)	0.071
HDL cholesterol (mmol/L)		1.43 (0.01, 2647)	1.46 (0.01, 1377)	**0.003**
	Men	1.28 (0.01, 1299)	1.33 (0.01, 680)	**0.001**
	Women	1.56 (0.01, 1348)	1.59 (0.01, 697)	0.148
Total-C/HDL-C ratio		3.97 (0.02, 2647)	3.93 (0.04, 1377)	0.328
	Men	4.38 (0.03, 1299)	4.31 (0.06, 680)	0.298
	Women	3.58 (0.03, 1348)	3.56 (0.04, 697)	0.657
LDL-C/HDL-C ratio		2.40 (0.02, 2554)	2.37 (0.03, 1353)	0.388

	Men	2.66 (0.02, 1221)	2.63 (0.04, 658)	0.585
	Women	2.16 (0.02, 1333)	2.13 (0.03, 694)	0.351
Total triglycerides (mmol/L)		1.55 (0.03, 2647)	1.48 (0.03, 1322)	0.065†
	Men	1.83 (0.05, 1299)	1.64 (0.04, 650)	0.262†
	Women	1.28 (0.03, 1348)	1.33 (0.03, 673)	**<0.001†**
BMI (kg/m2)		28.30 (0.11, 2658)	28.00 (0.15, 1413)	0.089
	Men	28.51 (0.14, 1308)	28.05 (0.18, 699)	**0.043**
	Women	28.09 (0.17, 1349)	27.92 (0.23, 714)	0.545
Waist circumference (cm)				
	Men	99.77 (0.38, 1302)	97.85 (0.48, 695)	**0.002**
	Women	87.71 (0.39, 1337)	88.07 (0.55, 714)	0.587
		NWAHS	GGT RFS	
		~~%, 95% CI (n)n (%)~~ ~~95% CI~~	~~%, 95% CI (n)n (%)~~ ~~95% CI~~	***p*-value**
Current smokers		~~2642~~ (21.35~~)~~, 19.83-22.95 (2642) ~~19.83-22.95~~	~~1405~~ (17.79,) 15.88-19.88 (1405) ~~15.88-19.88~~	**0.028**
	Men	~~1301~~ (22.75,~~)~~ 20.55-25.11 (1301) ~~20.55-25.11~~	~~696~~ (20.26,~~)~~ 17.44-23.41(696) ~~17.44-23.41~~	0.302
	Women	~~1341~~ (19.99,~~)~~ 17.93-22.21 (1341) ~~17.93-22.21~~	~~709~~ (15.37,~~)~~ 12.90-18.22 (709) ~~12.90-18.22~~	**0.032**
Known diabetes or Impaired Glucose Tolerance (IGT)		~~2656~~ (7.72,~~)~~ 6.76-8.80 (2656) ~~6.76-8.80~~	~~1422~~ (5.84,~~)~~ 4.73-7.18 (1422) ~~4.73-7.18~~	**0.037**
	Men	~~1307~~ (8.34,~~)~~ 6.96-9.97 (1307) ~~6.96-9.97~~	~~700~~ (5.14,~~)~~ 3.72-7.06 (700) ~~3.72-7.06~~	**0.014**
	Women	~~1350~~ (7.19,~~)~~ 5.92-8.69 (1350) ~~5.92-8.69~~	~~721~~ (6.38,~~)~~ 4.80-8.42 (721) ~~4.80-8.42~~	0.520

~~*Data source: North West Adelaide Health Study Stage 2, 2004-2006, 25 to 74 years. Greater Green Triangle Risk Factor Study, 2004-2006, 25 to 74 years.*~~

Note: The weighting of the data can result in rounding discrepancies or totals not adding.

†p-values based on log of the variable in order to address right skewedness of data.

Comparing CVD mortality outcomes

Figure 1(b) shows the relationship between IHD and Stroke mortality and age for GGT and NWA. Table 3 compares IHD and Stroke mortality rates between different regions of interest. IHD and Stroke mortality in inner and outer regional areas was generally worse than in major cities (p<0.001). Remote and very remote areas had significantly higher mortality rates than all other categories (p<0.001).

Table 3: Comparison of ischaemic heart disease (IHD) and stroke mortality rates by age group (Source: 2003-2007 Australian Bureau of Statistics)

Age Group (years)	**35-39**	**40-44**	**45-49**	**50-54**	**55-59**	**60-64**	**65-69**	**70-74**	**35-74: Crude**
NWA	9 (5-16)	19 (13-28)	34 (26-46)	62 (49-78)	99 (81-120)	157 (132-186)	281 (246-322)	419 (373-470)	103 (96-110)

GGT	5 (0-20)	14 (6-31)	28 (16-50)	44 (27-71)	99 (70-139)	130 (94-180)	224 (171-292)	488 (402-592)	98 (87-111)
NWA vs. GGT	*p=0.427*	*p=0.511*	*p=0.562*	*p=0.208*	*p=0.987*	*p=0.319*	*p=0.130*	*p=0.182*	*p=0.489*
Major Cities (Ex NWA)	6 (5-6)	12 (11-13)	23 (22-24)	38 (37-40)	63 (61-66)	110 (107-114)	199 (193-205)	369 (361-377)	70 (69-71)
NWA vs. Major Cities (Ex NWA)	*p=0.061*	*p=0.010**	*p=0.006**	*p=0.000**	*p=0.000**	*p=0.000**	*p=0.000**	*p= 0.028**	*p<0.001**
Inner and Outer Regional (Ex GGT)	8 (7-10)	16 (15-18)	30 (28-32)	47 (44-50)	75 (71-79)	131 (125-137)	230 (222-238)	440 (428-453)	92 (91-94)
GGT vs. Inner and Outer Regional (Ex GGT)	*p=0.492*	*p=0.714*	*p=0.872*	*p=0.768*	*p=0.097*	*p=0.991*	*p=0.840*	*p=0.295*	*p=0.341*
Remote and Very Remote	36 (28-47)	48 (39-60)	76 (64-91)	82 (68-99)	138 (118-162)	210 (181-243)	345 (300-395)	593 (524-671)	125 (118-132)

Age specific IHD and Stroke Mortality Rates per 100,000 population (95% Confidence Intervals) by age group. Mean of all deaths from 2003-2007
**Statistically significantly different (χ^2 test, $p < 0.05$).*

In all age groups GGT mortality rates were representative of those of inner and outer regional areas (crude mortality rates for 35-74 years: inner and outer regional versus GGT 92 versus 98, p=0.341). NWA mortality was generally higher than in other major Australian cities (crude mortality rates for 35-74 years: major cities versus NWA 70 versus 103, p=0.028). GGT and NWA mortality rates did not differ significantly despite NWA being a major city location (crude mortality rates for 35-74 years: GGT versus NWA p=0.489).

Relationship between SES and CVD mortality rates

A comparison of IRSD scores using an independent samples median test indicated no significant difference between the two study areas (p=0.108). However there was a significant difference in the distribution of IRSD scores (p=0.022), with scores in NWA skewed towards the lower end of the scale (Figure 2(a)).

Increasing mortality was consistently associated with lower IRSD scores. When age-specific mortality rates for age class 35-74 were plotted against IRSD (Figure 2(b)), both study areas were most closely aligned with

inner and outer regional areas. Closer inspection of study areas at the SLA level indicated that the trend remained. In NWA (Figure 2(c)) IRSD explained around 46% (n=18, β=-0.389) of the variation in mortality. In GGT (Figure 2(d)) IRSD explained approximately 19% (n=13, β=-0.477) of the variation in mortality, although the relationship was not statistically significant.

[Insert Figures 2(a), 2(b), 2(c) and 2(d) here]

DISCUSSION

Geographic and socioeconomic disparities in CVD mortality were first described in Australia in the late 1990s, [19] and initiated debate about likely explanations. Socioeconomic and cultural diversity between regions, differential prevalence of CVD risk factors, and variations in patterns of medical care were postulated as potential causative factors. This work started a debate about the most appropriate actions both within and outside the healthcare system to address these disparities [20-21]. Progress since has been slow in advancing our understanding of these issues, impeded by the lack of comprehensive, high quality data on CVD risk factor prevalence across the Australian population.

Based on AIHW published data,[3, 4, 6] and the only previous Australian study to analyse the contribution of CVD risk factor prevalence differences to the rural/regional–urban CVD mortality gap [22], our original hypothesis in this study was that GGT CVD risk factor profiles, and CVD mortality, would be worse than in NWA. Unexpectedly, GGT and NWA were similar in terms of absolute CVD risk scores, individual CVD risk factors and mortality rates. Furthermore, mortality rates in the regional GGT population are consistent with those observed in most regional areas of Australia, but lower than in remote areas, and higher than in the overall Australian metropolitan population. CVD mortality rates in the metropolitan NWA population are significantly higher than in the overall Australian metropolitan population.

Social gradients in health – 'caused by unequal distribution of power, income, goods and services' lead to inequitable health outcomes within and between populations [1]. Poorer Australians have worse CVD outcomes [23]. This was demonstrated in our study by the strong relationship between IRSD and CVD mortality at a national level (Figure 2(b)) as well as within NWA (Figure 2(c)). The trend was present within GGT (Figure 2(d)) although statistically non-significant. This can likely be explained by limited sample size coupled with a relatively narrow range of IRSD scores compared with NWA. These findings are consistent with other evidence in the Australian literature and from other developed countries regarding the association between low SES and increased levels of CVD risk factors, morbidity and mortality [21].

The influence of a broad range of social determinants (for example, quality of housing, employment, income

level, education etc.) on biological determinants of CVD, as well as differential access to health-promoting services may explain a significant part of the rural/regional-urban divide in CVD mortality in Australia. There is also growing evidence that variation in implementation of evidence based CVD care across geographic, institutional and even subspecialty boundaries may be an important determinant [24-25]. Implementation of evidence-based practice may provide an opportunity to reduce disparities in CVD outcomes, including geographically determined disparities, at relatively low cost and in shorter time frames than those required to address socioeconomic disparities across large populations.

All of the aforementioned variables and their relationship with CVD health outcomes are complex, yet all should be taken into account when formulating strategies to address inequalities.

Our study has limitations. Firstly, there are difficulties in extrapolating results from single rural and urban populations. This regional study population is relatively culturally and socioeconomically homogenous and probably representative of many (but not all) regional areas in Australia. The urban population is more culturally and socioeconomically diverse with over-representation of the socioeconomically disadvantaged compared with the overall Australian urban population. Secondly, we were unable to directly analyse associations between risk factors, SES and CVD mortality in the sample data sets due to the cross sectional rather than longitudinal design of the two population-based risk factor studies and other methodological differences in sampling and data collection. Time frames influencing some cross-sectional measured risk factor variables, compared with those operating over whole lifetimes to determine clinical outcomes such as CVD mortality, are different and we cannot be sure that they are stable or changing at the same rate in two geographically distinct populations. Some such variables which were not measured in our study, such as population levels of salt intake, may have resulted in the difference in systolic and diastolic blood pressures in our two study groups. However, we think that the most likely explanation for this observation is inter-observer variation in the measurement of blood pressure.

Strategies for comprehensive, high quality CVD risk factor surveillance should cover all population groups, regardless of geography or SES. Preferably, there should be longitudinal follow up, combined with appropriate epidemiological and health services research to investigate which interventions are most cost

effectively able to reduce disparities in CVD outcomes in the specific context of each of our social and health care systems.

Legend of Figures

Figure 1: Framingham absolute CVD risk and IHD and stroke mortality rates by age

Figure 2: Relationship between IHD and stroke mortality and IRSD

ACKNOWLEDGEMENTS

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COMPETING INTERESTS

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FUNDING

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The views expressed in this manuscript are those of the authors and do not necessarily represent, or should be attributed to, the views of the Australian Government Department of Health and Ageing or other funders.

The funders of the GGT RFS and NWAHS studies had no role in the collection, analysis and interpretation data of the data, nor in the writing of the report or the decision to submit the paper for publication.

No specific funding was received for the comparison study on which this manuscript is based.

out the Wimmera Shire part of GGT risk factor prevalence study.

CONTRIBUTORSHIP STATEMENT

PT conceived and designed the study. AT is Epidemiological Principal Investigator of the North West Adelaide Health Study. JAD was the chief investigator for the GGT DPP.

AT, RC, AM, JG and JAD were involved in acquisition of data. BP, EJ, EP and VV analysed and interpreted the data. EP and EJ drafted the manuscript and were responsible for its revisions. VV and RC helped to draft the manuscript. All authors contributed to specific sections in the manuscript. All authors read and approved the final manuscript.

DATA SHARING STATEMENT

There is no additional data publicly available. For further enquiries on accessing data please contact Prof. James Dunbar at director@greaterhealth.org and Janet Grant at janet.grant@adelaide.edu.au

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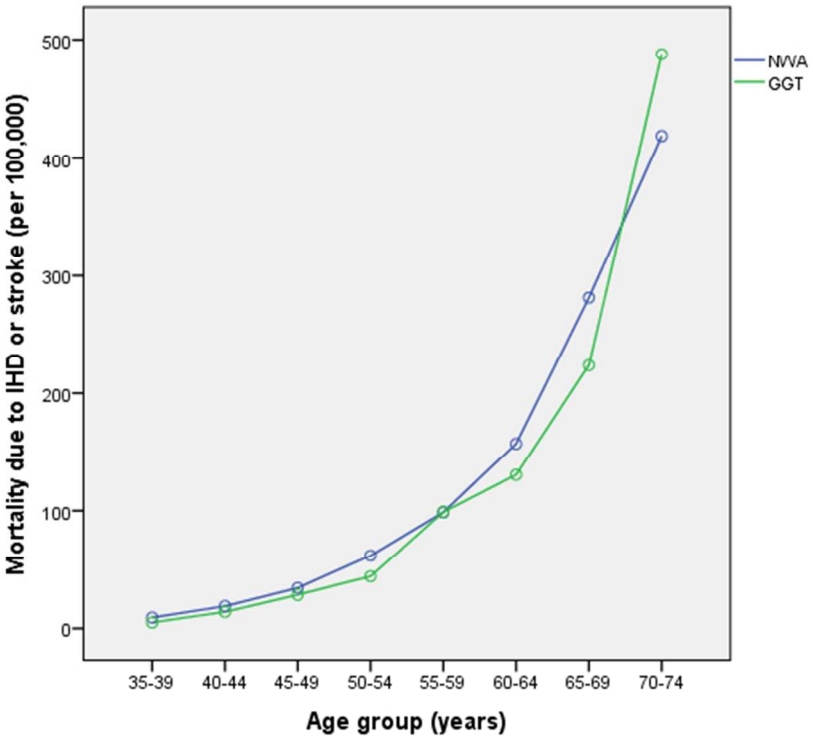

Figure 1: (a) Framingham absolute CVD risk and (b) IHD and stroke mortality rates by age
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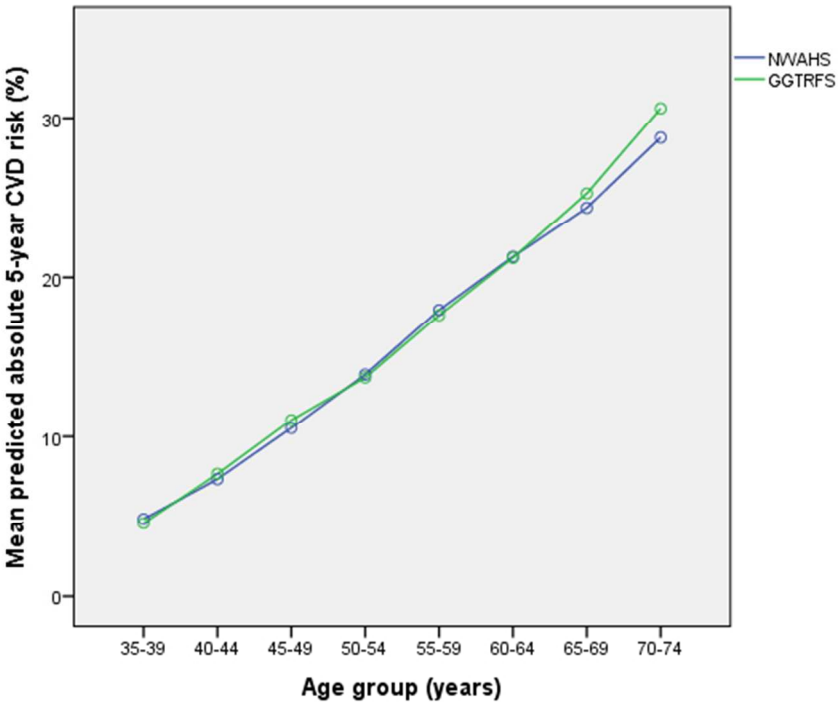

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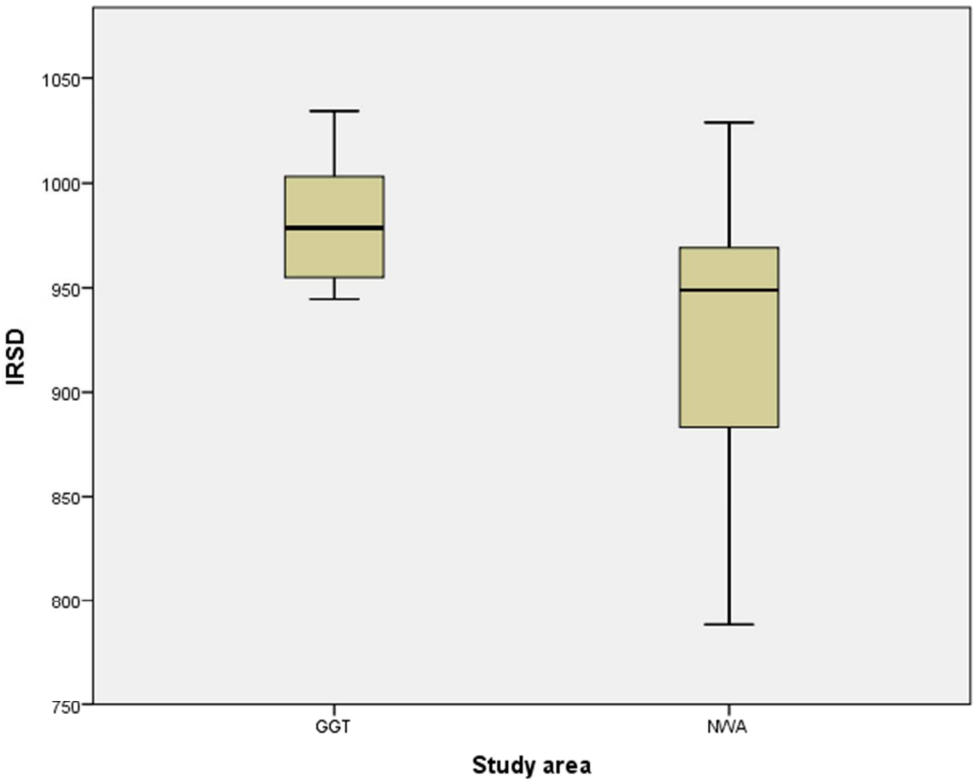

Figure 2: Relationship between IHD and stroke mortality and IRSD. (a) Distribution of IRSD scores between GGT and NWA; (b) IHD and stroke mortality rates by median IRSD for relevant geographical areas; (c) IHD and stroke mortality rates by IRSD for NWA SLAS; (d) IHD and stroke mortality rates by IRSD for GGT SLAs
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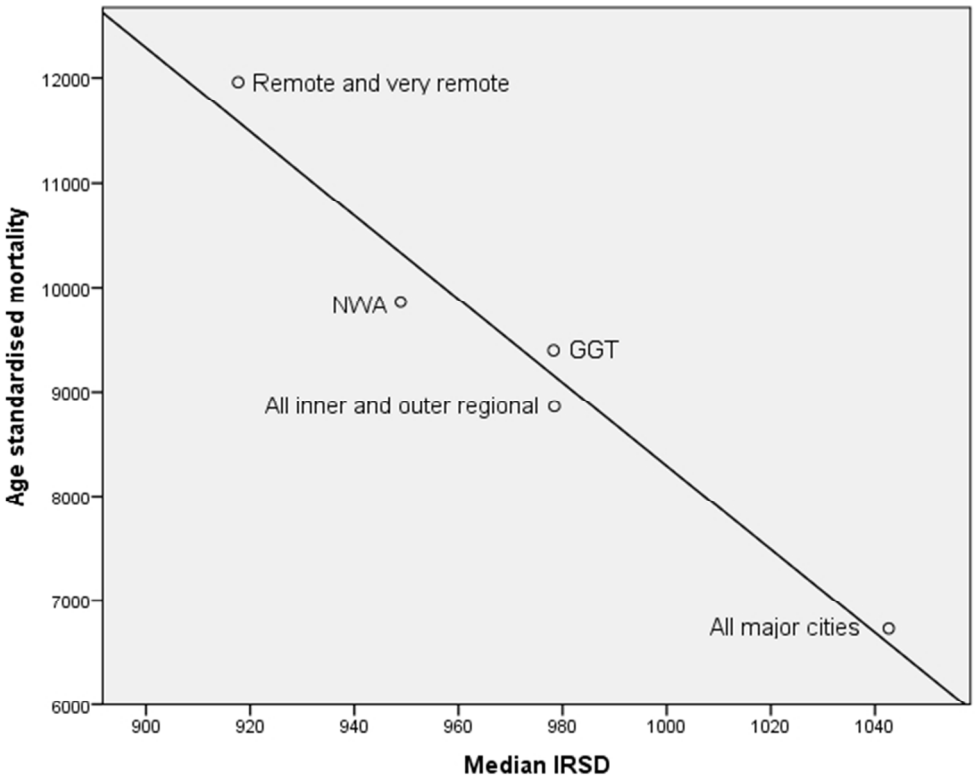

Figure 2: Relationship between IHD and stroke mortality and IRSD. (a) Distribution of IRSD scores between GGT and NWA; (b) IHD and stroke mortality rates by median IRSD for relevant geographical areas standardised to the Australian 2006 ERP; (c) IHD and stroke mortality rates by IRSD for NWA SLAS; (d) IHD and stroke mortality rates by IRSD for GGT SLAs
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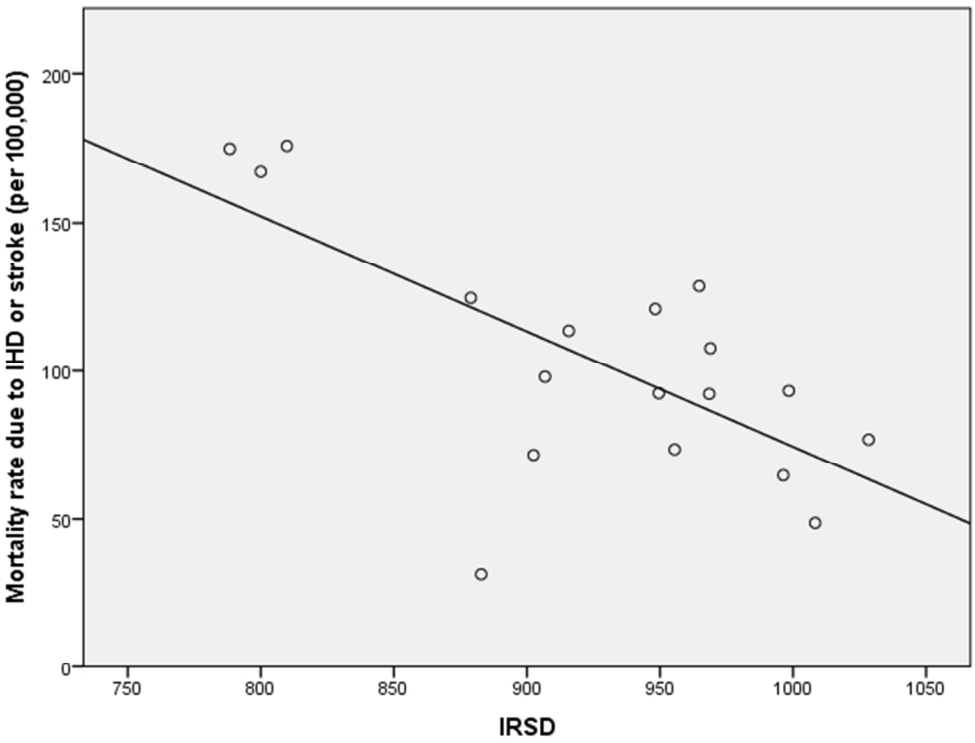

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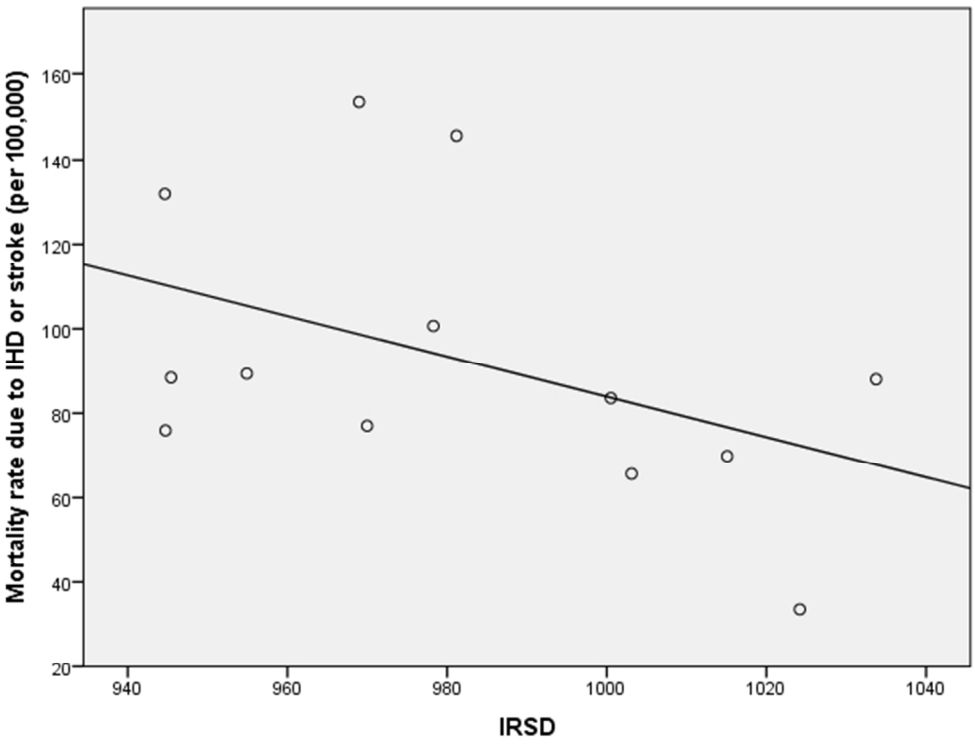

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STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation	Addressed on page number:
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	2
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	3
Objectives	3	State specific objectives, including any prespecified hypotheses	3
Methods			
Study design	4	Present key elements of study design early in the paper	4
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants	5,6
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case	
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	6,7
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	6,7
Bias	9	Describe any efforts to address potential sources of bias	6,7
Study size	10	Explain how the study size was arrived at	5,6
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	8
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	8
		(*b*) Describe any methods used to examine subgroups and interactions	7, 8, Fig 1(a), Fig 1(b)

(*c*) Explain how missing data were addressed	
(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy	5 (references 7-11)
(*e*) Describe any sensitivity analyses	NA

Continued on next page

Results			Addressed on page number:
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	5,6
		(b) Give reasons for non-participation at each stage	NA
		(c) Consider use of a flow diagram	
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	15
		(b) Indicate number of participants with missing data for each variable of interest	9-11 (Tables 1 and 2)
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)	
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time	
		Case-control study—Report numbers in each exposure category, or summary measures of exposure	
		Cross-sectional study—Report numbers of outcome events or summary measures	10-12
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	5 (reference 14) 11-12 (Table 3)
		(*b*) Report category boundaries when continuous variables were categorized	NA
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	NA
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	8
Discussion			
Key results	18	Summarise key results with reference to study objectives	12
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	14,15
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	14,15
Generalisability	21	Discuss the generalisability (external validity) of the study results	15
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	16

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

Correction

Tideman P, Taylor A, Janus E, *et al*. A comparison of Australian rural and metropolitan cardiovascular risk and mortality: the Greater Green Triangle and North West Adelaide Population Surveys. *BMJ Open* 2013;3:e003203. An error in the coding of one of the categorical variables used to calculate the Framingham five-year risk was detected following publication. The error does not affect the overall conclusions drawn in this paper but has changed figure 1A. The corrected figure 1A is below.

In addition, the first sentence of the Results section of the Abstract should now read: 'Few significant differences in CVD risk between the study regions, with mean absolute CVD risk ranging from approximately 1% in the age group 35–39 years to 14% in the age group 70–74 years.'

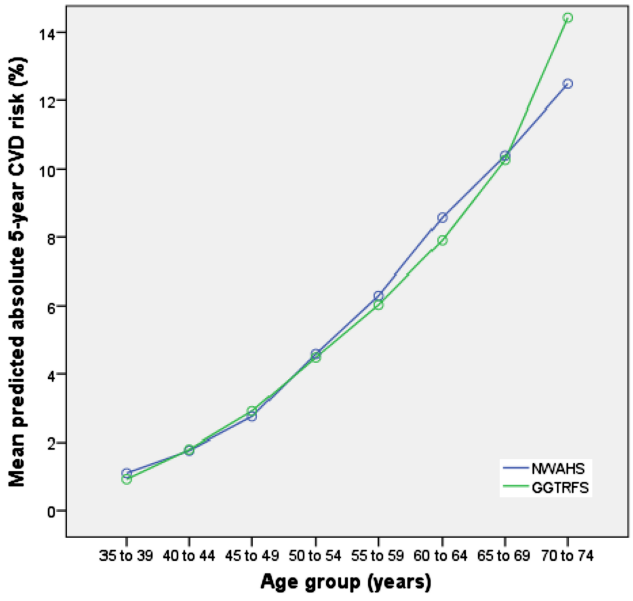

Figure 1A Framingham absolute cardiovascular disease risk by age.

BMJ Open 2014;**4**:e003203corr1. doi:10.1136/bmjopen-2013-003203corr1