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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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ABSTRACT

Objectives: 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. This study aimed to further investigate such disparities.

Design: Comparison of (1) CVD risk measures between a regional (Greater Green Triangle Risk Factor Study (GGT RFS, cross-sectional study, 2004–2006) and an urban population (North West Adelaide Health Study (NWAHS, longitudinal cohort study, 2004–2006); (2) Australian Bureau of Statistics (ABS) CVD mortality rates between these and other Australian regions; and (3) ABS CVD mortality rates by an area-level indicator of socioeconomic status, the Index of Relative Socioeconomic Disadvantage (IRSD).

Setting: Greater Green Triangle (GGT, Limestone Coast, Wimmera and Corangamite Shires) of South-Western Victoria and North-West Adelaide (NWA).

Participants: 1563 GGT RFS and 3036 NWAHS stage 2 participants (aged 25–74) provided some information (self-administered questionnaire +/- anthropometric and biomedical measurements).

Primary and secondary outcome measures: Age-group specific measures of absolute CVD risk, ABS CVD mortality rates by study group and Australian Standard Geographical Classification (ASGC) region.

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 (crude) mortality rates in GGT (98, 95% CI 87 to 111), NWA (103, 95% CI 96 to 110) and regional Australia (92, 95% CI 91 to 94). NWA mortality rates exceeded that of other city areas (70, 95% CI 69 to 71). Lower measures of socioeconomic status 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

ARTICLE SUMMARY

Article focus

- The study aim was to more objectively understand causes of geographical cardiovascular disease (CVD) mortality disparities in Australia by; (1) comparing measures of CVD risk (objective and self-reported data) between a rural population (Greater Green Triangle, GGT) and an urban population (North West Adelaide, (NWA)).
- (2) Comparing CVD mortality rates among GGT and NWA and other areas Australia-wide.
- (3) 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.

for different settings to elucidate relative contributions of the multiple determinants of risk and appropriate cardiac healthcare strategies to improve outcomes.

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 politicostructural 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 are available for comparative studies among remote, regional and urban areas. Better evidence is required to develop strategies to address inequalities.

This article 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 aim of the study was to more objectively understand causes of geographical CVD mortality disparities by (1) comparing measures of CVD risk (objective and self-reported data) between GGT and NWA, (2) comparing CVD mortality rates between GGT and NWA and other areas Australia-wide and (3) 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} Discussed 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 more than 18 years 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 biomedical 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 selected to make the age range of both populations comparable. From NWAHS, only stage 2 participants were selected 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 to ensure that variables were comparable. Some aspects could not be compared due to differences in questions used such as 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 ischaemic heart disease (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 years 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 mmol/L or above and/or having self-reported diabetes. As the questionnaire

Table 1 Demographic characteristics of participants by location

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

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

*Statistically significantly different (χ^2 test, $p < 0.05$) GGT RFS compared with NWAHS.

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†Insufficient numbers for a statistical test.

NWAHS, North West Adelaide Health Study; GGT RFS, Greater Green Triangle Risk Factor Study.

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 index of relative socioeconomic disadvantage (IRSD). IRSD is one of the four ABS socioeconomic indexes for areas (SEIFA), which are area-based summary measures of relative socioeconomic 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 V.12 and IBM SPSS Statistics V.19. CVD risk factor data for participants are reported as mean values with SEs for continuous variables and proportions with 95% CIs (using the Agresti-Coull technique) for discrete variables. Independent sample t tests were used to assess differences between means ($\alpha=0.05$), with the Welch method applied when the assumption of homogenous variance was not met. The χ^2 tests were used to assess differences between proportions ($\alpha=0.05$). The relationship between mortality rates and IRSD scores was examined using linear regression.

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).

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 1A).

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

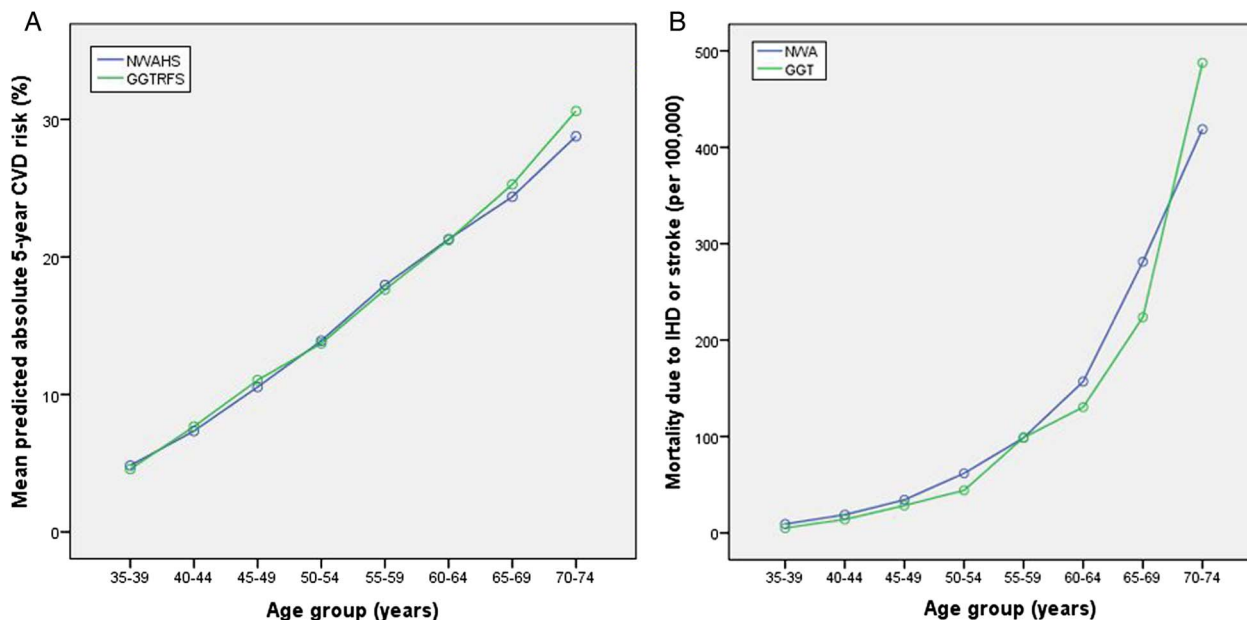


Figure 1 (A) Framingham absolute cardiovascular disease risk and (B) ischaemic heart disease and stroke mortality rates by age.

pressure and higher mean diastolic blood pressure than GGT RFS participants. High-density lipoprotein (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 body mass index (BMI) and waist circumference. NWAHS participants (women and overall) were more likely to be smokers. Prevalence of diabetes/impaired glucose tolerance (IGT) was higher in NWAHS (men and overall).

Comparing CVD mortality outcomes

Figure 1B shows the relationship between IHD and stroke mortality and age for GGT and NWA. Table 3 compares IHD and stroke mortality rates among

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 ($p<0.001$).

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 vs GGT 92 vs 98, $p=0.341$). NWA mortality was generally higher than in other major Australian cities (crude mortality rates for 35–74 years: major cities vs NWA 70 vs 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 vs NWA $p=0.489$).

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 (mm Hg)	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/m ²)	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
	%	95% CI	(n)	%	95% CI	(n)	
Current smokers	21.35	19.83 to 22.95	(2642)	17.79	15.88 to 19.88	(1405)	0.028
Men	22.75	20.55 to 25.11	(1301)	20.26	17.44 to 23.41	(696)	0.302
Women	19.99	17.93 to 22.21	(1341)	15.37	12.90 to 18.22	(709)	0.032
Known diabetes or Impaired Glucose Tolerance	7.72	6.76 to 8.80	(2656)	5.84	4.73 to 7.18	(1422)	0.037
Men	8.34	6.96 to 9.97	(1307)	5.14	3.72 to 7.06	(700)	0.014
Women	7.19	5.92 to 8.69	(1350)	6.38	4.80 to 8.42	(721)	0.520

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.

Bold indicates significant p values.

BMI, body mass index; CVD, cardiovascular disease; GGT RFS, Greater Green Triangle Risk Factor Study; HDL, high-density lipoprotein; IGT, impaired glucose tolerance; LDL, low-density lipoprotein; NWAHS, North West Adelaide Health Study.

Table 3 Comparison of ischaemic heart disease 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 value)	0.427	0.511	0.562	0.208	0.987	0.319	0.130	0.182	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 value)	0.061	0.010*	0.006*	0.000*	0.000*	0.000*	0.000*	0.028*	<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 value)	0.492	0.714	0.872	0.768	0.097	0.991	0.840	0.295	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% CI) by age group. Mean of all deaths from 2003 to 2007.

*Statistically significantly different (χ^2 test, $p < 0.05$).

GGT, Greater Green Triangle; IHD, ischaemic heart disease; NWA, North West Adelaide.

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 2A).

Increasing mortality was consistently associated with lower IRSD scores. When age-specific mortality rates for age class 35–74 were plotted against IRSD (figure 2B), 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 2C) IRSD explained around 46% ($n=18$, $\beta=-0.389$) of the variation in mortality. In GGT (figure 2D) IRSD explained approximately 19% ($n=13$, $\beta=-0.477$) of the variation in mortality, although the relationship was not statistically significant.

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 among regions, differential prevalence of CVD risk factors and variations in patterns of medical care were postulated as potential causative factors. This work initiated a debate about the most appropriate actions to be undertaken 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.

On the basis of 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 2B) as well as within NWA (figure 2C). The trend was present within GGT (figure 2D), although

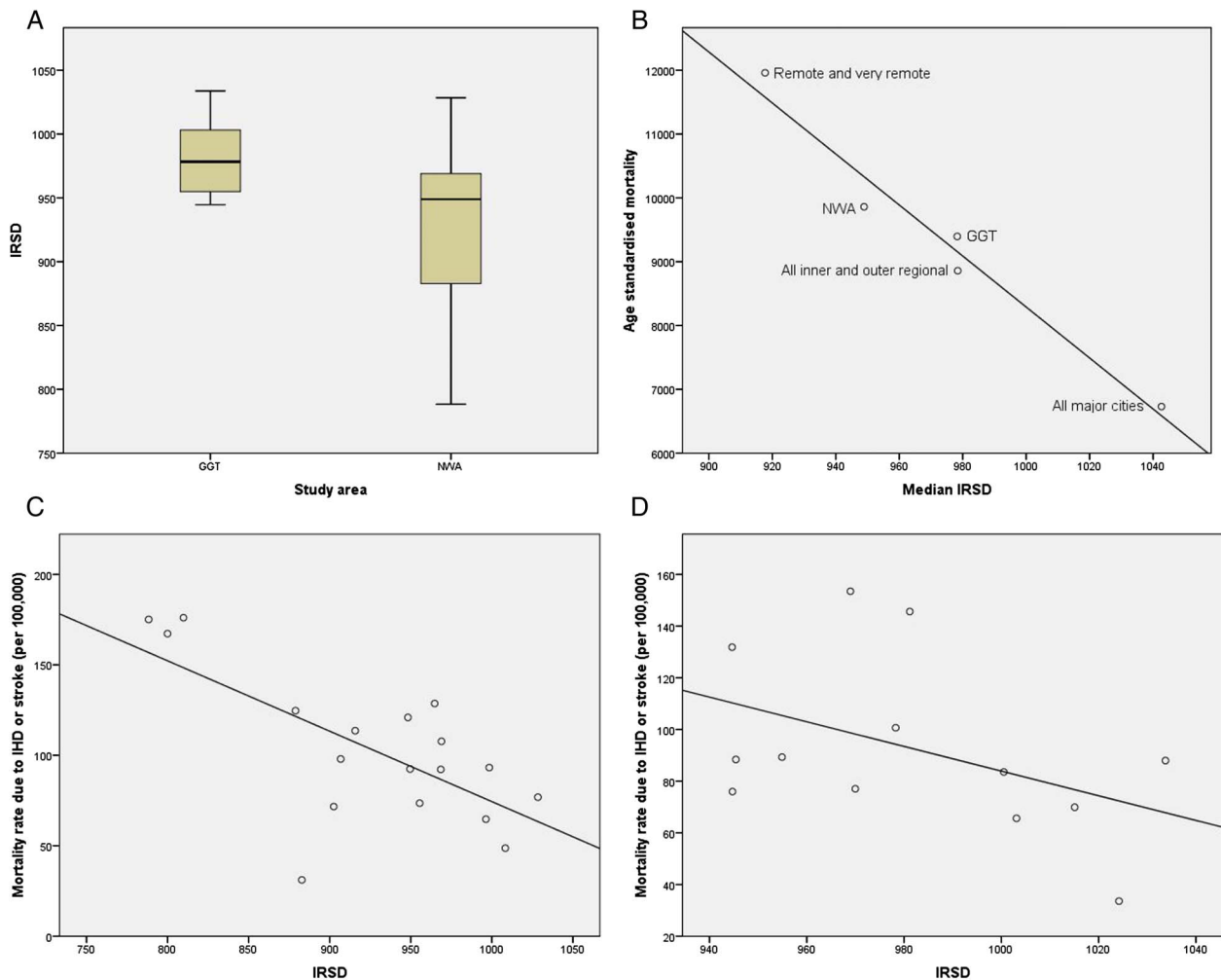


Figure 2 Relationship between ischaemic heart disease (IHD) and stroke mortality and IRSD. (A) Distribution of Index of Relative Socioeconomic Disadvantage (IRSD) scores between greater green triangle (GGT) and north west Adelaide (NWA); (B) IHD and stroke mortality rates by median IRSD for relevant geographical areas; (C) IHD and stroke mortality rates by IRSD for NWA statistical local area (SLAs); (D) IHD and stroke mortality rates by IRSD for GGT SLAs.

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 among low SES and increased levels of CVD risk factors, morbidity and mortality.²¹

The influence of a broad range of social determinants (eg, 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 socio-economic 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. First, 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 overrepresentation of the socioeconomically disadvantaged compared with the overall Australian urban population. Second, we were unable to directly analyse associations among 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 whether 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 interobserver 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 able to cost-effectively reduce disparities in CVD outcomes in the specific context of each of our social and healthcare systems.

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Contributors PT conceived and designed the study. 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.

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