



Small area-level socioeconomic status and all-cause mortality within 10 years in a population-based cohort of women: Data from the Geelong Osteoporosis Study

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ABSTRACT

Background. The social gradient of health and mortality is well-documented. However, data are scarce regarding whether differences in mortality are observed across socio-economic status (SES) measured at the small area-level. We investigated associations between area-level SES and all-cause mortality in Australian women aged ≥ 20 years.

Methods. We examined SES, obesity, hypertension, lifestyle behaviors and all-cause mortality within 10 years post-baseline (1994), for 1494 randomly-selected women. Participants' residential addresses were matched to Australian Bureau of Statistics Census data to identify area-level SES, and deaths were ascertained from the Australian National Deaths Index. Logistic regression models were adjusted for age, and subsequent adjustments made for measures of weight status and lifestyle behaviors.

Results. We observed 243 (16.3%) deaths within 10 years post-baseline. Females in SES quintiles 2–4 (less disadvantaged) had lower odds of mortality (0.49–0.59) compared to SES quintile 1 (most disadvantaged) under the best model, after adjusting for age, smoking status and low mobility.

Conclusions. Compared to the lowest SES quintile (most disadvantaged), females in quintiles 2 to 5 (less disadvantaged) had significantly lower odds ratio of all-cause mortality within 10 years. Associations between extreme social disadvantage and mortality warrant further attention from research, public health and policy arenas.

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Introduction

The social gradient of health is well-documented (WHO, 2006; Wilkinson and Marmot, 1998; Wilkinson and Pickett, 2009); it is observed in relation to most chronic diseases, and mortality, with data suggesting this is also observed in higher income countries (Wilkinson and Pickett, 2009). Although there are few data from Australia pertaining to socio-economic status (SES) and mortality, analyses over a relatively short time period have shown mortality to be higher in socially disadvantaged areas of Tasmania, Australia (Turrell et al., 2006a). A plethora of data from other countries shows such an association to exist, most impressively demonstrated in a 2005 study of education and more than 1 million deaths in European countries, which incorporated 51 million person-years of observation (Huisman

et al., 2005). However, even a study of that size was unable to account for the role played by factors such as weight status or lifestyle-behaviors that may confound or modify any associations between disadvantage and mortality in females. The authors of that study suggested that, compared to men, low levels of wealth, and a range of psychosocial factors and health-related behaviors would be more likely to contribute to inequalities in mortality among women (Davey Smith et al., 1994; Mackenbach et al., 1999).

Obesity, the most commonly observed condition that influences lifestyle-related diseases, is well-documented as a key harbinger of early mortality (Katzmarzyk et al., 2003; Adams et al., 2006); however, a recent systematic review and meta-analysis identified conflicting data regarding associations between levels of obesity and mortality (Flegal et al., 2013), suggesting that more work in this area is needed. Obesity is more often observed in women compared to men (Mackenbach et al., 1999; Sobal and Stunkard, 1989; Pasco et al., 2012a), and is disproportionately observed in lower SES groups (Ball et al., 2002; Brennan et al., 2009a; Turrell et al., 2006b), as are less healthy lifestyle behaviors including smoking, alcohol consumption and physical inactivity; these

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factors may all be associated with obesity and/or non-communicable diseases (Turrell et al., 2006b; WHO, 2004). Hypertension is a key risk factor for cardiovascular disease and related-mortality (Whelton, 1994); furthermore, an increased prevalence of hypertension has consistently been observed in disadvantaged groups (Buys et al., 2015; Diez Roux et al., 2002; Grotto et al., 2008; Minor et al., 2008). In context of the greater rates of obesity and less healthy lifestyles observed in disadvantaged groups in Australia (Turrell et al., 2006b), it would not be surprising for socially disadvantaged women to have a greater likelihood of earlier death than less disadvantaged women. Given that lower SES areas also have a greater prevalence of single parent families (ABS, 1996), which may be more often headed by a female, from a public health perspective, studies that examine health outcomes at the area-level are important, as they may elucidate where targeted attention is required and specific population subgroups that may be most likely to benefit from intervention, specifically women.

In light of the strong argument regarding disparities in mortality between social groups within high-income countries (Wilkinson and Pickett, 2009), and given the limited research on SES disparities in smaller geographic areas and associations with differences in mortality, we aimed to examine associations between weight status, hypertension, lifestyle behaviors, area-level SES and all-cause mortality within 10 years post-baseline recruitment in a randomly selected population-based cohort of women from Australia.

Materials and methods

Study population

Baseline data were derived from an age-stratified random sample of population-based women enrolled in the Geelong Osteoporosis Study (GOS, $n = 1494$); participants aged 20 years and over had been randomly selected and recruited from the Commonwealth electoral rolls for the Barwon Statistical Division (BSD) in south eastern Australia, during 1993–7 (Pasco et al., 2012b). The BSD region is reported as being representative of the broader Australian population (Pasco et al., 2012b; Brennan et al., 2009b). All participants in the GOS female cohort provided written informed consent. Approval for the study was obtained from the Barwon Health Human Research Ethics Committee.

Measurements

Mortality

All-cause mortality was determined by data linkage of the GOS female cohort to the 2006 Australian National Deaths Index, and coded as occurring within 10 years post-baseline recruitment date.

Socioeconomic status

Area-based SES at baseline recruitment was determined by matching the residential address for each subject to the corresponding 1996 Australian Bureau of Statistics (ABS) Census Collection District, the smallest level of division that encompasses approximately 250 households. ABS software was used to determine the Socio-Economic Index for Areas (SEIFA) value based on 1996 census data for each subject (ABS, 1996). SEIFA values summarize the characteristics of subjects within an area, and therefore provide a single measure to rank the level of disadvantage at the area-level. For our analyses, we employed the Index of Relative Socioeconomic Disadvantage (IRSD), from which a summary score is determined based on the SEIFA values; the IRSD incorporates variables that identify areas with (i) low incomes, (ii) little or no occupational or vocational training, and (iii) unskilled occupations.

The IRSD scores for the participants were categorized into quintiles according to cut-points for the study region, whereby SES quintile 1 was the most disadvantaged and SES quintile 5 the least disadvantaged. We have previously reported that no SES differences were observed between GOS participants and non-participants (Pasco et al., 2012b).

Weight status, hypertension, and lifestyle behaviors

At baseline, trained research staff measured body weight using electronic scales to the nearest ± 0.1 kg, and measured height using a wall-mounted stadiometer to ± 0.1 cm. Body mass index (BMI) was calculated as weight/height squared (kg/m^2). BMI was treated as a categorical variable using a three-way split at the cut-points of ≥ 25.0 kg/m^2 indicating overweight and ≥ 30.0 kg/m^2 indicating obesity. Waist circumference was measured to the nearest 0.5 cm, and categorized as a binary variable of waist circumference ≥ 80 cm versus not according to published guidelines regarding cut-points indicative of increased risk for diabetes (AusDiab, 2008). Systolic and diastolic blood pressures were measured in mmHg using an automated upper arm digital blood pressure monitor (UA-767) with subjects seated. Using these blood pressure measures, we identified individuals with Stage 1 hypertension if systolic blood pressure was between 140–159.9 mm Hg and/or diastolic blood pressure was between 90–99.9 mm Hg, and individuals with Stage 2 hypertension if systolic blood pressure was ≥ 160 mm Hg and/or diastolic blood pressure was ≥ 100 mm Hg (AHA, 2014). Self-reported smoking status was categorized as never, current or ever, and included manufactured or hand-rolled cigarettes or cigars. The regularity of alcohol consumption (all types of alcohol combined) was ascertained using a validated food frequency questionnaire (Giles and Ireland, 1996) and categorized as none, less than once per week, several times per week, or every day. Mobility was defined as low if participants self-reported as being sedentary, inactive or bedridden vs. active or very active, using measures as previously reported (Pasco et al., 2012b). All measures of weight status, hypertension, and lifestyle behaviors were ascertained at the baseline recruitment of the GOS cohort.

Statistical analyses

Descriptive characteristics of the study population were tabulated, and the proportions of all-cause mortality that occurred within 10 years were presented according to the population at risk in each SES quintile. To examine differences across SES quintiles, chi-square test was used for categorical data, ANOVA for normally distributed continuous data, and Kruskal–Wallis used where continuous data were non-parametric. Binary logistic regression models assessing the relationship between quintiles of SES and all-cause mortality (holding the most disadvantaged SES quintile 1 as referent) were adjusted for baseline age (continuous variable), and results presented as odds ratio (OR) and 95% confidence intervals (95% CI). Age-adjusted associations between SES and mortality were investigated for the individual role played by each measure of adiposity (BMI, waist or hip circumference), hypertension, and lifestyle behavior (smoking status, alcohol consumption, or mobility). Finally, the best model was investigated, and results for fully-adjusted analyses were presented as OR (95% CI). For each model we present results for goodness of fit using the Hosmer–Lemeshow test. Statistical interactions between SES, age and all variables used in models were tested for effect modification. Statistical significance was set at $p < 0.05$ and analyses were performed using MINITAB (Version 16; Minitab, State College, PA).

Results

Table 1 presents characteristics of the study population ($n = 1494$) across SES quintiles, including all-cause mortality that occurred within 10 years post-baseline recruitment ($n = 243$, 16.3%). Differences across SES quintiles were observed for the baseline measures of categorical BMI ($p = 0.001$), waist and hip circumferences (both $p < 0.001$), and for current or past smoking status (both $p \leq 0.001$). A trend for difference across SES quintiles was observed for alcohol consumption ($p = 0.06$) and mortality within 10 years ($p = 0.08$). No further differences across SES were observed.

Age-adjusted logistic regression models for the association between SES and the odds of mortality within 10 years post-baseline recruitment

Table 1

Baseline (1994) characteristics of Australian women (n = 1494) and all-cause mortality across quintiles of socioeconomic status (SES); data presented as n (%), mean (\pm SD) or median (range).

	SES quintile 1 ^a (n = 288)	SES quintile 2 (n = 310)	SES quintile 3 (n = 323)	SES quintile 4 (n = 252)	SES quintile 5 (n = 319)	p-value
Mortality \leq 10 years	60 (20.8%)	51 (16.4%)	51 (15.8%)	30 (11.8%)	51 (16.0%)	0.08
Baseline age, year	53.5 (\pm 19.8)	55.0 (\pm 20.3)	54.8 (\pm 20.1)	53.4 (\pm 18.8)	53.2 (\pm 19.3)	0.66
BMI, kg/m ² (continuous)	27.0 (15.9–66.6)	25.6 (16.6–43.3)	25.6 (15.5–49.9)	25.4 (17.6–52.1)	24.5 (17.0–44.0)	<0.001
BMI, kg/m ² (categorical)						0.001
\leq 24.9	111 (38.5%)	136 (43.9%)	138 (42.7%)	111 (43.7%)	174 (54.5%)	
25.0–29.9	91 (31.6%)	110 (35.5%)	115 (35.6%)	81 (31.9%)	96 (30.1%)	
\geq 30.0	86 (29.9%)	64 (20.6%)	70 (21.7%)	62 (24.4%)	49 (15.4%)	
Waist \geq 80 cm ^b	191 (68.5%)	180 (59.2%)	190 (60.1%)	147 (60.2%)	154 (50.0%)	<0.001
Hip, cm ^b	105.1 (63.9–156.2)	103.1 (79.4–140.0)	102.7 (82.0–145.4)	103.6 (82.6–158.1)	100.4 (78.4–141.2)	<0.001
Hypertension [†]						0.65
None	206 (74.1%)	200 (67.8%)	210 (69.5%)	169 (71.3%)	222 (73.0%)	
Stage 1	44 (15.8%)	57 (19.3%)	58 (19.2%)	39 (16.5%)	43 (14.1%)	
Stage 2	28 (10.1%)	38 (12.9%)	34 (11.3%)	29 (12.2%)	39 (12.8%)	
Smoking status ^b						<0.001
Current	67 (23.3%)	50 (16.1%)	41 (12.7%)	24 (9.4%)	41 (12.8%)	<0.001
Ever	140 (48.9%)	120 (38.7%)	110 (34.1%)	85 (33.5%)	129 (40.6%)	0.001
Alcohol consumption ^b						0.06
None	79 (27.6%)	84 (27.1%)	71 (22.0%)	52 (20.5%)	71 (22.3%)	
<Once/week	125 (43.7%)	128 (41.3%)	145 (45.0%)	109 (42.9%)	136 (42.8%)	
Several/week	69 (24.1%)	73 (23.5%)	77 (23.9%)	76 (29.9%)	73 (23.0%)	
Every day	13 (4.5%)	25 (8.1%)	29 (9.0%)	17 (6.7%)	38 (12.0%)	
Low mobility ^b	102 (35.5%)	99 (31.9%)	97 (30.1%)	75 (29.5%)	97 (30.5%)	0.54

Significant p-values are bolded.

^a Most disadvantaged SES quintile. BMI = body mass index.

^b Missing data; waist circumference $>$ 80 cm (n = 43), hip circumference (n = 43), hypertension (n = 78), smoking (n = 3), alcohol consumption (n = 4), and mobility (n = 3).

are presented in Fig. 1. Compared to the lowest SES quintile (most disadvantaged), females in quintiles 2 to 5 (less disadvantaged) had significantly lower OR of all-cause mortality within 10 years. Baseline age (continuous) was a predictor for all-cause mortality (OR 1.11, 95% CI 1.10, 1.13) (see also Tables A and B).

Table 2 presents the associations between SES and all-cause mortality after further adjustment was made for measures of weight status and hypertension (Models 1–4 inclusive). Compared to SES quintile 1 (most disadvantaged), we observed the odds of females in SES quintiles 2–4 dying within 10 years to be twice that of less disadvantaged women; associations that were not attenuated by BMI, waist or hip circumference, or hypertension. For SES quintile 5, the OR increased slightly, and the relevant 95% CIs crossed the line of significance for each model. Baseline age remained a significant predictor in each model (all $p \leq 0.001$).

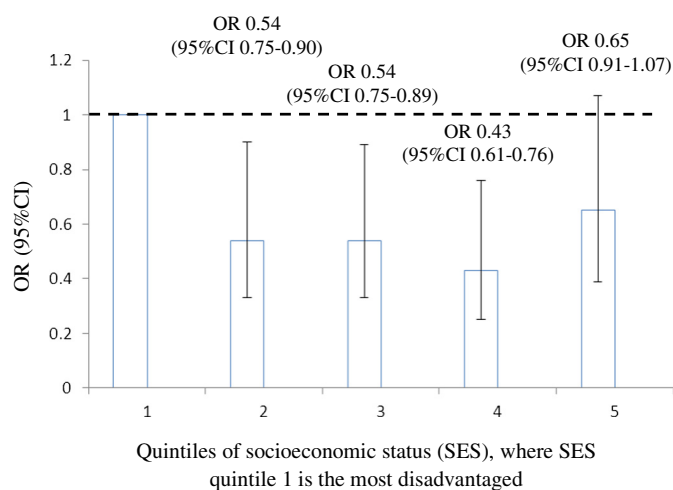


Fig. 1. Age-adjusted logistic regression models showing associations between quintiles of SES (quintile 1 = most disadvantaged) and the odds of mortality within 10 years post-baseline recruitment. Data presented as odds ratios (OR) and 95% confidence intervals (95% CI). Dashed line indicates threshold of significance.

Table 3 presents the associations between SES and all-cause mortality after further adjustment was made for lifestyle behaviors (Models 5–8 inclusive). Associations between SES and mortality were not attenuated by current smoking status or alcohol consumption; however, having ever smoked and low mobility were independent predictors of mortality within 10 years (OR 1.54, 95% CI 1.07, 2.20, and OR 2.18, 95% CI 1.52, 3.14, respectively). As was observed in our models that examined weight status, baseline age remained a predictor in each of the models that accounted for lifestyle behaviors (all $p \leq 0.001$).

Table 4 presents the fully-adjusted best model for associations between SES and mortality within 10 years. Females in SES quintiles 2–4 had a 50–60% reduction in odds of mortality compared to those in SES quintile 1 (most disadvantaged), independent of age (OR 1.10, 95% CI 1.08, 1.12), smoking status (OR 1.49, 95% CI 1.04, 2.15) and low mobility (OR 2.13, 95% CI 1.48, 3.07). Further adjustment for measures of weight status or other lifestyle variables did not attenuate these associations (Table C).

Discussion

Our study reports that in this prospective cohort, the odds of all-cause mortality within 10 years post-baseline for females in less disadvantaged SES quintiles was less than half that of females in the most disadvantaged SES quintile. Associations between small area-based SES and mortality were not explained by measures of weight status or lifestyles, and were independent of baseline age, smoking status and low mobility.

Our findings show that a social gradient of mortality exists for females across SES as measured at the small-area, even in a high income country such as Australia. These data support earlier findings from a multilevel study conducted in a different region of Australia over a shorter time period, which reported that approximately one third of the risk of mortality was explained by area-level SES (Turrell et al., 2006a); our study suggested that about one half of the risk of mortality was explained by area-level SES. We observed current smoking and low mobility were predictors of mortality, independent of age, however, weight status or lifestyle behaviors did not attenuate associations between SES and mortality; an interesting finding given the well-documented social gradient of health and health-related behaviors

Table 2
Age-adjusted logistic regression models for the role played by different measures of adiposity and hypertension ascertained at baseline recruitment (1994), on the associations between quintiles of socioeconomic status (SES) and the odds of all-cause mortality within 10 years in Australian women. Results presented as odds ratios and 95% confidence intervals.

	Model 1: BMI (cut-points)	Model 2: waist circumference	Model 3: hip circumference	Model 4: hypertension
Quintile 1 ^a (referent)	1.00	1.00	1.00	1.00
Quintile 2	0.54 (0.33, 0.90)	0.48 (0.28, 0.81)	0.47 (0.28, 0.80)	0.46 (0.27, 0.79)
Quintile 3	0.54 (0.33, 0.89)	0.48 (0.29, 0.80)	0.47 (0.28, 0.80)	0.52 (0.30, 0.87)
Quintile 4	0.44 (0.25, 0.77)	0.43 (0.24, 0.76)	0.42 (0.24, 0.76)	0.43 (0.24, 0.77)
Quintile 5	0.65 (0.39, 1.08)	0.61 (0.36, 1.03)	0.60 (0.36, 1.02)	0.62 (0.37, 1.05)
Baseline age ^b	1.11 (1.10, 1.13)	1.11 (1.09, 1.13)	1.11 (1.09, 1.12)	1.11 (1.09, 1.13)
BMI, kg/m ² (categories)				
≤24.9 (referent)	1.00	–	–	–
25.0–29.9	0.75 (0.51, 1.09)	–	–	–
≥30.0	0.96 (0.62, 1.49)	–	–	–
Waist ≥80 cm	–	1.13 (0.77, 1.66)	–	–
Hip circumference ^b	–	–	0.99 (0.98, 1.01)	–
Hypertension (categories)				
None (referent)	–	–	–	1.00
Stage 1	–	–	–	0.98 (0.64, 1.50)
Stage 2	–	–	–	1.52 (0.97, 2.36)
Goodness of fit p-value	0.001	0.001	0.001	0.001

Statistically significant associations are bolded.

^a Most disadvantaged SES quintile.

^b Continuous variables.

(Wilkinson and Marmot, 1998; Wilkinson and Pickett, 2006). Nevertheless, a meta-analysis by Flegal et al. showed that, compared to normal weight (defined by BMI), those with grade 1 obesity (BMI of 30 to <35 kg/m²) did not have higher mortality (Flegal et al., 2013). The strength of associations we report between SES and mortality, after accounting for smoking and low mobility, are consistent with a recent US study, which reported that after adjustment for smoking, alcohol consumption, and mobility resulted in a risk ratio for mortality of 1.59 (95% CI 1.03–2.45) for individuals in low SES groups compared with their higher SES counterparts (Nandi et al., 2014). However, there are conflicting data regarding weight (overweight determined by BMI) and mortality. A study from the National Health and Nutrition Examination Survey (NHANES) suggested that overweight subjects do not have an increased risk of mortality above that of normal weight subjects (Flegal et al., 2005). In contrast, a study from the US found a positive association between overweight status and increased mortality for women, and when the authors restricted analyses to non-smokers, those associations strengthened (Adams et al., 2006).

Speculation regarding possible mechanisms for the observed associations between lower SES and a greater likelihood of mortality is

warranted. Socio-environmental factors that are contextually related to lower SES may drive these associations; for instance, areas of lower SES are more likely to have greater rates of unemployment, lower quality or inadequate housing, or limited access to nutritious food. These factors all reduce the ability of an individual to achieve the highest attainable standard of wellbeing and health, being the doctrine of the World Health Organization Commission on the Social Determinants of Health (WHO, 2006). In addition, the notion of social capital suggests that with increased social engagement, networks and community activities, there is likely to be a positive flow on effect for improved health. Lower SES areas are less likely to have high levels of social capital (Turrell et al., 2006a; Subramanian et al., 2003) compared to more advantaged areas. Social capital, as a potential mechanism for reduced mortality in areas that are less disadvantaged, may be explained by considering that shared 'norms' exist within social networks (Putnam, 1993), for instance a greater uptake of preventive health behaviors such as increased physical activity, more healthier food choices, and a network that supports a higher health literacy. Health literacy is the ability of an individual to seek, understand and use health information (Buchbinder et al., 2011). Indeed, up to a quarter of the Australian

Table 3
Age-adjusted logistic regression models showing associations between lifestyle behaviors ascertained at baseline recruitment (1994) and quintiles of socioeconomic status (SES) and the odds of all-cause mortality within 10 years in Australian women. Results presented as odds ratios and 95% confidence intervals.

	Model 5: Smoking (current)	Model 6: Smoking (ever)	Model 7: Alcohol consumption	Model 8: Low mobility
Quintile 1 ^a (referent)	1.00	1.00	1.00	1.00
Quintile 2	0.55 (0.33, 0.92)	0.57 (0.34, 0.94)	0.55 (0.33, 0.91)	0.57 (0.34, 0.95)
Quintile 3	0.56 (0.34, 0.92)	0.58 (0.35, 0.96)	0.54 (0.32, 0.89)	0.55 (0.33, 0.92)
Quintile 4	0.45 (0.26, 0.80)	0.47 (0.27, 0.83)	0.44 (0.25, 0.77)	0.45 (0.26, 0.80)
Quintile 5	0.66 (0.40, 1.10)	0.68 (0.41, 1.14)	0.67 (0.40, 1.12)	0.66 (0.40, 1.11)
Baseline age	1.12 (1.10, 1.13)	1.12 (1.10, 1.13)	1.11 (1.10, 1.13)	1.10 (1.08, 1.11)
Smoking status (categories)				
Never (referent)	1.00	1.00	–	–
Current	1.76 (0.99, 3.12)	–	–	–
Ever	–	1.54 (1.07, 2.20)	–	–
Alcohol consumption (categories)				
None (referent)	–	–	1.00	–
<once/week	–	–	1.21 (0.81, 1.81)	–
Several/week	–	–	1.12 (0.68, 1.85)	–
Every day	–	–	0.93 (0.53, 1.63)	–
Low mobility	–	–	–	2.18 (1.52, 3.14)
Goodness of fit p-value	<0.001	<0.001	0.003	0.008

Statistically significant associations are bolded.

^a Most disadvantaged SES quintile.

Table 4

Logistic regression for the best model showing the associations between lifestyle behaviors ascertained at baseline recruitment (1994) and quintiles of socioeconomic status (SES) and the odds of all-cause mortality within 10 years in Australian women. Results presented as odds ratios and 95% confidence intervals.

<i>Model 10: Smoking (ever) and low mobility</i>	
Quintile 1* (referent)	1.00
Quintile 2	0.59 (0.35, 1.00)
Quintile 3	0.59 (0.35, 0.98)
Quintile 4	0.49 (0.27, 0.86)
Quintile 5	0.69 (0.41, 1.16)
Baseline age	1.10 (1.08, 1.12)
Smoking status (categories)	
Never (referent)	1.00
Ever	1.49 (1.04, 2.15)
Low mobility	2.13 (1.48, 3.07)
Goodness of fit <i>p</i> -value	0.004

Bold values indicate statistical significance ($p < 0.05$).

population has low health literacy (Barber et al., 2009); an issue that is most often seen in older individuals, socially disadvantaged groups, or those of culturally and linguistically diverse populations (Jordan et al., 2010; Weiss, 2007; Adams et al., 2009). It is possible that our findings may be influenced by higher health literacy and increased social capital in females within our less disadvantaged SES quintiles. Should this be the case, those females would have an increased ability to secure health promoting resources (Subramanian et al., 2003) compared to those of lower SES groups. However, despite this speculation, it is important to acknowledge that there are conflicting data regarding the role played by social capital in associations between SES and mortality in Australia (Turrell et al., 2006a).

As expected, increasing baseline age was a consistent predictor of increased likelihood of mortality. Of interest was that in our age-adjusted analysis, we observed a slight peak of increased mortality within 10 years for women resident the least disadvantaged SES quintile; contextual factors within different SES quintiles may partly explain that peak. For instance, that anomaly in the pattern between SES and mortality may be explained by the relocation of some women to a residential aged care facility, nursing home, or a family home, that may be located in an area determined as higher SES compared to the SES of their own area at baseline recruitment. It may be equally plausible that some females may have relocated, for reasons such as frailty or illness, to an area of lower SES relative to their baseline residence, thus confounding associations. Despite these suggestions, it is unlikely that small numbers of our participants residing in an area of different SES would attenuate the strong associations we report.

Our findings support those previously shown at the level of Government administrative districts in Tasmania, Australia (Turrell et al., 2006a), thus providing further evidence that if the responsibility to reduce mortality in disadvantaged groups is picked up by local or state governments, significant benefits may result. This suggestion is in context of the rhetoric concerning health equity as evidenced in the strategic direction statements of many state- and national-level documents; our data highlight the imperative in supporting rhetoric with actions to achieve health equity.

Our study has some limitations. Our *a priori* study aim was to examine SES at baseline recruitment on mortality, thus we did not adjust for residential relocation during the study period. We are unable to exclude the possibility that the variance in the composition of populations within the SES quintiles may explain differences in mortality, and we acknowledge that changes in variables since baseline might play a role in mortality, including pre-existing co-morbidity (Adams et al., 2006). Our *a priori* purpose was to examine all-cause mortality, thus we did not account for co-morbidities. It is possible that our findings may not be generalisable to the broader Australia, especially given that 99% of the GOS female cohort is Caucasian (Pasco et al., 2012b), and that the

BSD does not include areas of geographical remoteness; a factor that may be associated with mortality in other areas of Australia. Our results pertain to females only; whilst the GOS includes cohorts of both sexes, these cohorts were recruited at different baseline time-points and were subsequently followed up at different time-points. This latter issue means that in order to accurately determine area-level SES at both the time of follow-up and at the initial baseline, data-linkage to the Australian Bureau of Statistics would involve different years for each cohort. The ABS has recommended that the socioeconomic indexes from different census years not be compared due to the use of dissimilar variables for diverse census periods, and suggests that it could provide misleading results. Although the point estimate for odds of mortality in SES quintile 5 were similar to those of other SES quintiles, the 95% CI for all models crossed the line of unity and thus no significant difference was observed for this quintile; we suggest that our findings for SES quintile 5 be interpreted as inconclusive, and that further work with larger sample sizes be undertaken. Our measure of mobility reflects lifestyle but also functional ability, which may be related to disability or illness. Our measures of lifestyle behaviors were self-reported, for which the accuracy and reproducibility have not been determined. Finally, we were not able to determine the cause of mortality, nor do we suggest causality among the variables of interest. Our study also has strengths. The study population encompassed a representative sample from the study region, and no SES differences were observed at baseline between participants and non-participants (Pasco et al., 2012b). We present the first data from the BSD region of Australia to examine social disparities in mortality, and suggest that our findings between extreme social disadvantage and mortality warrant further attention from research, public health, health promotion and policy arenas.

Conclusion

In conclusion, we show that, independent of age at baseline, and compared to the lowest SES quintile (most disadvantaged), females in quintiles 2 to 5 (less disadvantaged) had significantly lower OR of all-cause mortality within 10 years. We found little evidence to suggest that measures of weight status or lifestyle behaviors attenuated this association. Larger scale studies should be considered in order to confirm our observations, and to further elucidate the underlying pathway between social disadvantage and mortality in a high income country. Finally, further work is required to examine whether changes in the level of social disadvantage over time may modify the risk of mortality.

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Conflict of interest statement

The authors declare that there are no conflicts of interests.

Contributor roles

All authors participated in the design of the study. SLB-O performed the statistical analysis, and drafted the manuscript. SLB-O, LJW, KLH, SMH, ALS, AGD and JAP guided and reviewed the manuscript. LJW and JAP supervised the statistical analysis. All authors read and approved the final manuscript.

Appendix A

Table A
Age-adjusted logistic regression models for the role played by different measures of adiposity and hypertension ascertained at baseline recruitment (1994), on the associations between quintiles of socioeconomic status (SES) and the odds of all-cause mortality within 10 years in Australian women. Results presented as odds ratios and 95% confidence intervals.

	Quintile 1: BMI (cut-points)	P	Quintile 2: waist circumference	P	Quintile 3: hip circumference	P	Quintile 4: hypertension	p
Quintile 1	1.55 (0.93, 2.57)	0.09	1.63 (0.97, 2.74)	0.07	1.66 (0.98, 2.79)	0.06	1.61 (0.95, 2.73)	0.07
Quintile 2	0.83 (0.50, 1.39)	0.49	0.78 (0.46, 1.32)	0.35	0.78 (0.46, 1.33)	0.36	0.74 (0.44, 1.27)	0.28
Quintile 3	0.83 (0.50, 1.39)	0.48	0.78 (0.46, 1.32)	0.35	0.79 (0.47, 1.32)	0.36	0.83 (0.49, 1.41)	0.49
Quintile 4	0.67 (0.38, 1.19)	0.17	0.69 (0.39, 1.25)	0.23	0.70 (0.39, 1.26)	0.24	0.69 (0.38, 1.24)	0.21
Quintile 5 ^a (referent)	1.00	–	1.00	–	1.00	–	1.00	–
Baseline age ^b	1.11 (1.10, 1.13)	<0.001	1.11 (1.09, 1.13)	<0.001	1.11 (1.09, 1.12)	<0.001	1.11 (1.09, 1.13)	<0.001
BMI, kg/m ² (categories)								
≤24.9 (referent)	1.00	–	–	–	–	–	–	–
25.0–29.9	0.75 (0.51, 1.09)	0.13	–	–	–	–	–	–
≥30.0	0.96 (0.62, 1.49)	0.86	–	–	–	–	–	–
Waist ≥ 80 cm	–	–	1.13 (0.77, 1.66)	0.53	–	–	–	–
Hip circumference ^b	–	–	–	–	0.99 (0.98, 1.01)	0.53	–	–
Hypertension (categories)								
None (referent)	–	–	–	–	–	–	1.00	–
Stage 1	–	–	–	–	–	–	0.98 (0.64, 1.50)	0.92
Stage 2	–	–	–	–	–	–	1.52 (0.97, 2.36)	0.07

Statistically significant associations are bolded, and statistical trends are underlined.

^a Least disadvantaged SES quintile.

^b Continuous variables.

Table B
Age-adjusted logistic regression models showing associations between lifestyle behaviors ascertained at baseline recruitment (1994) and quintiles of socioeconomic status (SES) and the odds of all-cause mortality within 10 years in Australian women. Results presented as odds ratios and 95% confidence intervals.

	Model 5: smoking (current)	P	Model 6: smoking (ever)	P	Model 7: alcohol consumption	P	Model 8: low mobility	p
Quintile 1	1.51 (0.91, 2.51)	0.11	1.46 (0.88, 2.44)	0.14	1.49 (0.89, 2.49)	0.12	1.51 (0.90, 2.52)	0.12
Quintile 2	0.84 (0.50, 1.39)	0.19	0.83 (0.50, 1.38)	0.47	0.82 (0.49, 1.36)	0.43	0.86 (0.51, 1.44)	0.56
Quintile 3	0.84 (0.51, 1.39)	0.50	0.84 (0.51, 1.40)	0.51	0.80 (0.48, 1.33)	0.40	0.83 (0.50, 1.39)	0.49
Quintile 4	0.68 (0.39, 1.21)	0.19	0.68 (0.39, 1.21)	0.19	0.65 (0.37, 1.16)	0.14	0.68 (0.38, 1.21)	0.19
Quintile 5 ^a (referent)	1.00	–	1.00	–	1.00	–	1.00	–
Baseline age	1.12 (1.10, 1.13)	<0.001	1.12 (1.10, 1.13)	<0.001	1.11 (1.10, 1.13)	<0.001	1.10 (1.08, 1.11)	<0.001
Smoking status (categories)								
Never (referent)	1.00	–	1.00	–	–	–	–	–
Current	1.76 (0.99, 3.12)	0.05	–	–	–	–	–	–
Ever	–	–	1.54 (1.07, 2.20)	0.02	–	–	–	–
Alcohol consumption (categories)								
None (referent)	–	–	–	–	1.00	–	–	–
<Once/week	–	–	–	–	1.21 (0.81, 1.81)	0.34	–	–
Several/week	–	–	–	–	1.12 (0.68, 1.85)	0.66	–	–
Every day	–	–	–	–	0.93 (0.53, 1.63)	0.80	–	–
Low mobility	–	–	–	–	–	–	2.18 (1.52, 3.14)	<0.001

Statistically significant associations are bolded, and statistical trends are underlined.

^a Least disadvantaged SES quintile.

Table C
Logistic regression for the best model showing the associations between lifestyle behaviors ascertained at baseline recruitment (1994) and quintiles of socioeconomic status (SES) and the odds of all-cause mortality within 10 years in Australian women. Results presented as odds ratios and 95% confidence intervals.

	Model 10: smoking (ever) and low mobility	p-value
Quintile 1	1.45 (0.86, 2.43)	0.16
Quintile 2	0.86 (0.51, 1.44)	0.57
Quintile 3	0.85 (0.50, 1.42)	0.53
Quintile 4	0.70 (0.39, 1.25)	0.23
Quintile 5 ^a (referent)	1.00	–
Baseline age	1.10 (1.08, 1.12)	<0.001
Smoking status (categories)		
Never (referent)	1.00	–
Ever	1.49 (1.04, 2.15)	0.03
Low mobility	2.13 (1.48, 3.07)	<0.001

Statistically significant associations are bolded.

^a Least disadvantaged SES quintile.

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