Socioeconomic disparities in stroke rates and outcome: pooled analysis of stroke incidence studies in Australia and New Zealand

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ABSTRACT

Objective: To assess the influence of area-level socioeconomic status (SES) on incidence and case-fatality rates for stroke.

Design, setting and participants: Analysis of pooled data for 3077 patients with incident stroke from three population-based studies in Perth, Melbourne, and Auckland between 1995 and 2003.

Main outcome measures: Incidence and 12-month case-fatality rates for stroke.

Results: Annual age-standardised stroke incidence rates ranged from 77 per 100 000 person-years (95% CI, 72–83) in the least deprived areas to 131 per 100 000 person-years (95% CI, 120–141) in the most deprived areas (rate ratio, 1.70; 95% CI, 1.47–1.95; $P < 0.001$). The population attributable risk of stroke was 19% (95% CI, 12%–27%) for those living in the most deprived areas compared with the least deprived areas. Compared with people in the least deprived areas, those in the most deprived areas tended to be younger (mean age, 68 v 77 years; $P < 0.001$), had more comorbidities such as hypertension (58% v 51%; $P < 0.001$) and diabetes (22% v 12%; $P < 0.001$), and were more likely to smoke (23% v 8%; $P < 0.001$). After adjustment for age, area-level SES was not associated with 12-month case-fatality rate.

Conclusions: Our analysis provides evidence that people living in areas that are relatively more deprived in socioeconomic terms experience higher rates of stroke. This may be explained by a higher prevalence of risk factors among these populations, such as hypertension, diabetes and cigarette smoking. Effective preventive measures in the more deprived areas of the community could substantially reduce rates of stroke.

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Stroke is a major non-communicable disease of increasing global importance, with an estimated 5.7 million deaths from stroke occurring each year, and many more survivors living with residual disability.[1] Given the limited availability of effective acute treatment options, prevention is the most realistic approach to averting the growing burden of stroke worldwide. Although several modifiable risk factors for stroke have been well established, uncertainty persists regarding the influence of others. In particular, the strength and mechanisms of association of socioeconomic status (SES) are controversial, although data are accumulating[2-4] from different parts of the world, including Australia and New Zealand.[5-6] Stroke is a challenging disease to study in a population-wide context because of complexities in case ascertainment, diagnosis, measurement, and the inclusion of an adequately large sample to assess multiple exposures with modest effect. The aim of this study was to pool comparable population-based incidence studies to determine the influence of SES on both the incidence rates and case-fatality rates for stroke in Australia and New Zealand.

METHODS

Study population

We used individual patient data from three population-based "ideal" stroke incidence studies[7] conducted in Australia and New Zealand within a decade of each other. Comparable methods had been used across the studies, as described elsewhere.[5,8-10] The included studies were the Perth Community Stroke Study (PCSS; two separate 12-month study periods beginning in 1995 and 2000);[9] the Northeast Melbourne Stroke Incidence Study (NEMESIS; 36 months during 1996–1999);[5,10] and the Auckland Regional Community Stroke (ARCOS) Study (12 months, 2002–2003).[8]

Measures of socioeconomic deprivation

We used similar area-level measures of relative socioeconomic "deprivation" for the populations of Australia (Index of Relative Socio-Economic Disadvantage [IRSD][11]) and New Zealand (New Zealand index of deprivation [NZDep][12]). For the Australian studies, we matched postcodes of registered patients to the IRSD; and for the New Zealand study, we matched registered patients to the NZDep 2001 score by meshblock (an area unit of about 90 people). Corresponding quintiles of area deprivation were derived. We used the IRSD constructed from 1996 census data for the 1995 PCSS and NEMESIS, and the index constructed from 2001 census data for the 2000 PCSS.

Statistical analyses

We pooled individual-level data for all patients aged > 15 years with a first-ever (incident) stroke (numerator) and for whom quintile of deprivation could be determined, and took population data for people aged > 15 years (denominator) from the census that was relevant to each study (1996 Australian census for the 1995 PCSS and NEMESIS; 2001 Australian census for the 2000 PCSS; and 2001 New Zealand census for ARCOS). To assess consistency, given the use of different deprivation measures, we conducted a sensitivity analysis investigating associations between stroke incidence across quintile of deprivation, by country.

Direct standardisation of rates to the World Health Organization "world" population[13] was performed to allow comparisons across quintiles. Relative differences in stroke rates were calculated by $(\text{rate}_{Q5} - \text{rate}_{Q1})/\text{rate}_{Q1}$, where Q = quintile. We used Poisson regression analysis to determine the statistical significance of differences in crude incidence between quintiles 1 and 5. For age-standardised incidence, we assessed statistical significance using the mid-*P* exact test.[14] The population-attributable risk fraction was calculated from age-standardised rates, and exact mid-*P* 95% confidence intervals were derived.[14]

We used logistic regression analysis to (i) determine whether patient demographics and outcome varied by quintile of deprivation, and (ii) examine the effect of area-level SES on 12-month case-fatality rates. Models

were developed in a step-wise process, akin to mediation analysis, to determine whether other variables influenced the relationship between 12-month case-fatality rate and area-level SES. In the final model, which included all covariates, collinearity was assessed using mean and individual variance inflation factors and condition numbers, and all plausible first-order interactions were assessed and only included in the model if they were significant to the $P < 0.001$ level due to multiple testing. Otherwise, the level of significance was considered to be $P < 0.05$.

All analyses were conducted using Stata, version 11.1 (StataCorp, College Station, Tex, USA).

RESULTS

Overall, of 3133 patients with incident stroke, 3077 (98%) were matched to deprivation data: 1367 of 1423 (96%) from the ARCOS study, 1314 (100%) from NEMESIS, 213 (100%) from the 1995 PCSS and 183 (100%) from the 2000 PCSS. These cases of stroke were identified from a total population of 1 741 765 person-years. Fifty-six cases from the ARCOS study were excluded from analyses due to missing data on area of residence. With these cases excluded, the crude incidence rate was reduced from 160 to 153 per 100 000 person-years in New Zealand. However, the excluded cases shared similar patient characteristics to the full study population in terms of distribution of ethnicity, employment status, history of comorbidities (hypertension, diabetes, atrial fibrillation), stroke type, and stroke severity (as measured by loss of consciousness).

Age and deprivation were inversely related, with patients in the most deprived areas having strokes at an average age of 68 years, compared with 77 years in the least deprived areas (Box 1). Patients from the most deprived areas who had strokes were more likely to be of non-European ethnicity, blue collar workers or current smokers, and to have a history of hypertension and diabetes. Conversely, a U-shaped relationship was found for alcohol consumption, with higher proportions of current alcohol consumption among patients in both the least and most deprived quintiles, although this is likely to be confounded by age and socioeconomic factors. The effects of deprivation by sex were also examined. Although similar associations to the overall results were found for females, there were no significant differences between area-level deprivation and pre-stroke institutional care, history of hypertension and diabetes among males, after adjustment for age (data available on request).

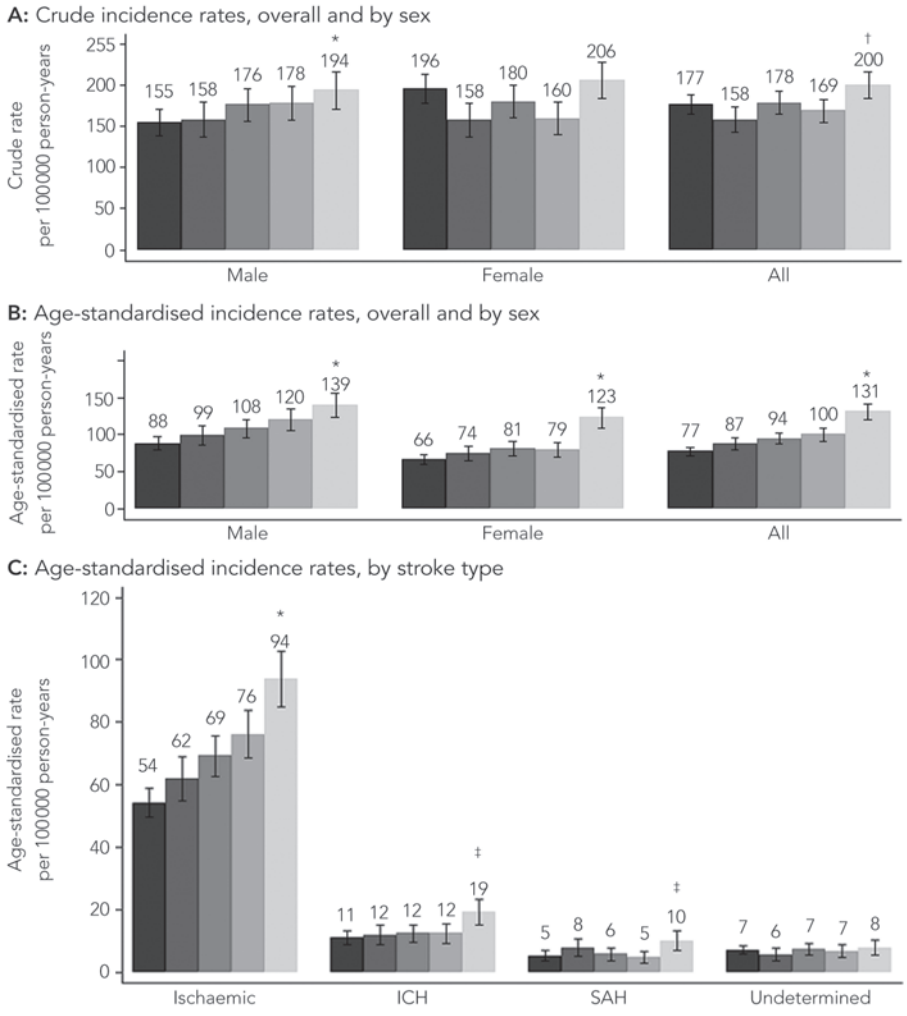
The crude annual stroke incidence rate was higher among patients from the most deprived areas (quintile 5; 200 per 100 000 person-years [95% CI, 184–216]) compared with those from the least deprived areas (quintile 1; 177 per 100 000 person-

1 Patient demographics and outcome, overall and by quintile of area-level socioeconomic status*

	Total ($n = 3077$)	Q1 ($n = 871$)	Q2 ($n = 435$)	Q3 ($n = 628$)	Q4 ($n = 533$)	Q5 ($n = 610$)	$P^{\dagger}$ (crude)	$P^{\dagger}$ (age-adjusted)
Age, mean (SD)	73 (14)	77 (13)	73 (14)	74 (14)	73 (14)	68 (15)	<0.001	—
Male	1422 (46)	359 (41)	208 (48)	298 (47)	274 (51)	283 (46)	0.008	0.46
Australia	1710 (56)	615 (71)	183 (42)	344 (55)	254 (48)	314 (51)	<0.001	<0.001
Ethnicity: European‡	2565 (85)	807 (95)	379 (89)	538 (88)	448 (85)	393 (65)	<0.001	<0.001
Employment status								
Not working§	278 (9)	58 (7)	36 (8)	40 (6)	50 (9)	94 (15)	<0.001	0.002
Full/part-time	437 (14)	120 (14)	66 (15)	78 (12)	77 (14)	96 (16)	0.42	<0.001
Retired	2090 (68)	604 (69)	299 (69)	461 (73)	367 (69)	359 (59)	0.001	0.004
Unknown	272 (9)	89 (10)	34 (8)	49 (8)	39 (7)	61 (10)	0.56	0.53
Social class								
Professional¶	993 (32)	432 (50)	143 (33)	144 (23)	136 (26)	138 (23)	<0.001	<0.001
White collar**	491 (16)	142 (16)	65 (15)	99 (16)	79 (15)	106 (17)	0.75	0.97
Blue collar††	850 (28)	136 (16)	100 (23)	189 (30)	188 (35)	237 (39)	<0.001	<0.001
Unknown	743 (24)	161 (18)	127 (29)	196 (31)	130 (24)	129 (21)	0.24	0.03
Pre-stroke institutional care								
Yes	516 (17)	192 (22)	70 (16)	111 (18)	65 (12)	78 (13)	<0.001	0.02
Unknown	246 (8)	35 (4)	46 (11)	57 (9)	51 (10)	57 (9)	<0.001	0.002
History of								
Hypertension	1670 (55)	440 (51)	232 (54)	352 (57)	297 (56)	349 (58)	0.006	<0.001
Diabetes	500 (16)	107 (12)	74 (17)	91 (15)	93 (17)	135 (22)	<0.001	<0.001
MI	423 (14)	113 (13)	65 (15)	105 (17)	66 (12)	74 (12)	0.49	0.70
Atrial fibrillation	714 (24)	216 (25)	102 (24)	148 (24)	114 (22)	134 (22)	0.11	0.38
Smoking behaviour‡‡								
Current smoker	432 (14)	67 (8)	54 (12)	80 (13)	91 (17)	140 (23)	<0.001	<0.001
Unknown	423 (14)	141 (16)	64 (15)	98 (16)	64 (12)	56 (9)	<0.001	0.007
Drinking behaviour‡‡								
Current drinker	1270 (41)	409 (47)	170 (39)	228 (36)	213 (40)	250 (41)	0.01	<0.001
Unknown	559 (18)	165 (19)	83 (19)	135 (21)	89 (17)	87 (14)	0.02	0.23
Stroke subtype								
Ischaemic stroke	2219 (72)	600 (69)	310 (71)	463 (74)	405 (76)	441 (72)	0.03	0.01
ICH	399 (13)	117 (13)	56 (13)	77 (12)	62 (12)	87 (14)	0.97	0.44
SAH	166 (5)	43 (5)	33 (8)	29 (5)	22 (4)	39 (6)	0.84	0.05
Unknown	293 (10)	111 (13)	36 (8)	59 (9)	44 (8)	43 (7)	<0.001	0.51
Lost consciousness§§	972 (32)	293 (34)	139 (32)	186 (30)	156 (30)	198 (33)	0.33	0.46
Dead at 7 days	413 (13)	131 (15)	50 (11)	93 (15)	63 (12)	76 (12)	0.16	0.67
Dead at 28 days	631 (21)	202 (23)	84 (19)	137 (22)	97 (18)	111 (18)	0.01	0.60
Dead at 12 months	1015 (33)	314 (36)	144 (33)	209 (33)	168 (32)	180 (30)	0.007	0.53

ICH = intracerebral haemorrhage. MI = myocardial infarction. Q = quintile (quintile 1, least deprived; quintile 5, most deprived). SAH = subarachnoid haemorrhage. * Values reported as mean (SD) or no. (%) of patients; percentages are based on non-missing values. † P for difference between quintiles. ‡ Defined according to self-reported ethnic origin of being New Zealander/Australian or European. § Home duties or other (unemployed, redundant, disability pensioner, student). ¶ For example, doctor, lawyer, accountant, manager, executive, teacher. ** Skilled non-manual vocations (eg, salesman, shop owner). †† Skilled/semi-skilled/unskilled labour (eg, craftsman, driver, labourer). ‡‡ Immediately before stroke. §§ Unconscious or semi-conscious within 24 h of onset. ◆

2 Stroke incidence rates by quintile of area-level socioeconomic status



Error bars show 95% CIs. *P* values were calculated for rate ratios between Q1 and Q5. ICH = intracerebral haemorrhage. Q = quintile. SAH = subarachnoid haemorrhage. * $P < 0.001$. † $P < 0.05$. ‡ $P < 0.01$. ◆

years [95% CI, 165–188]) (Box 2, A). Overall, the age-standardised incidence rate per 100 000 person-years was 70% higher (95% CI, 47%–95%; $P < 0.001$) among the most deprived group compared with the least deprived group (131 [95% CI, 120–141] v 77 [95% CI, 72–83], respectively) (Box 2, B). The linearity of this increase was more marked among males than females, but the absolute difference in age-standardised rates was greater among females (57) than males (51). The relative difference in age-standardised rates for the most deprived group compared with the least deprived was 58% in males (95% CI, 30%–93%; $P < 0.001$), and 87% (95% CI, 51%–130%; $P < 0.001$) in females. Interestingly, the age-adjusted incidence rate was higher among males than females in each category of deprivation; the difference was statistically significant in all deprivation categories except quintile 5.

Significant increases in age-standardised rates were observed between the lowest and highest deprivation quintiles for the major subtypes of stroke, but no pattern was observed for strokes of undetermined type (Box 2, C).

The sensitivity analysis showed a trend of increasing age-standardised incidence rates per 100 000 person-years with increasing deprivation in both countries, from 80 (95% CI, 73–87) in quintile 1 to 127 (95% CI, 112–142) in quintile 5 in Australia, and from 72 (95% CI, 63–81) to 133 (95% CI, 118–148) in New Zealand.

The proportion of stroke in the population attributable to living in the most deprived areas compared with the least deprived areas was 19% (95% CI, 12%–27%), after adjustment for age. Area-level deprivation was not found to be associated with 12-month case-fatality rate, except in the unadjusted model where there was an observed difference between the most and least deprived areas (Box 3). Age was associated with 12-month case-fatality rate (Box 3), while in the largest model (Model 5), pre-stroke institutional care, history of diabetes or atrial fibrillation, prior myocardial infarction, and pre-stroke smoking were all associated with an increased risk of death within 1 year (Box 4). Although social class, as an indicator of individual SES, was not associated with death, active employment status was associated with a reduced risk of death (Box 5). No significant variable interactions requiring inclusion were detected, and no collinearity was detected in the final model.

DISCUSSION

In this unique pooled analysis of three population-based stroke studies, we have shown disparities in the incidence rates of stroke between patients classified according to area-level relative deprivation, with people in the most disadvantaged areas having the highest rates of stroke. These data are supported by the observation that patients living in less disadvantaged areas tend to be older and healthier, with fewer risk factors such as hypertension, diabetes, and smoking. This is consistent with the 2001 National Health Survey finding among about 26 900 people from all states and territories in Australia that higher proportions of people living in the more deprived areas smoke or have hypertension.[15] Similar trends were found in the 2006–07 New Zealand Health Survey.[16] Our findings are also consistent with a study that showed a 10-fold difference in stroke mortality and disability-adjusted life-years lost between countries with the highest SES and those with the lowest SES.[17]

Further, our study indicates that improving the SES profile of those living in the most deprived areas could prevent up to one-fifth of strokes in Australia and New Zealand.

Interestingly, males had significantly higher rates of stroke than females in each category of deprivation except in the "most deprived", although statistical power may have been compromised in this category because of small numbers. This suggests differing levels of prevention and self-care between males and females. We noted a trend for greater levels of hypertension and

3 Odds ratios (95% CI) for the association of area-level SES and demographics with case-fatality rates for stroke at 1 year

	Model 1 (*n* = 3077)	Model 2 (*n* = 3077)	Model 3 (*n* = 3016*)	Model 4 (*n* = 2912*)	Model 5 (*n* = 2912*)
Quintile 2 v quintile 1	0.88 (0.69–1.12)	1.06 (0.82–1.37)	1.10 (0.85–1.44)	1.14 (0.83–1.58)	1.12 (0.80–1.56)
Quintile 3 v quintile 1	0.88 (0.71–1.10)	1.01 (0.81–1.27)	1.02 (0.81–1.29)	1.11 (0.83–1.48)	1.10 (0.82–1.49)
Quintile 4 v quintile 1	0.82 (0.65–1.03)	1.00 (0.78–1.27)	1.05 (0.82–1.34)	1.20 (0.89–1.62)	1.19 (0.87–1.62)
Quintile 5 v quintile 1	0.74 (0.59–0.93)	1.12 (0.89–1.42)	1.12 (0.87–1.43)	1.27 (0.95–1.72)	1.25 (0.92–1.70)
Age: per year	—	1.06 (1.05–1.06)	1.06 (1.05–1.06)	1.06 (1.05–1.07)	1.06 (1.05–1.07)
Male	—	0.90 (0.77–1.06)	0.90 (0.77–1.07)	1.18 (0.96–1.46)	1.27 (1.02–1.58)
Australia	—	—	1.23 (1.04–1.46)	1.72 (1.37–2.15)	1.61 (1.27–2.03)
Ethnicity: European	—	—	0.83 (0.64–1.08)	1.24 (0.90–1.70)	1.27 (0.92–1.76)

SES = socioeconomic status. Quintile 1 – least deprived. Quintile 5 – most deprived. * Excluding missing patient data for ethnicity (61). ◆

diabetes as the deprivation level increased among females but not males (data available on request).

We did not find an association between area-level SES and 12-month case-fatality rates. Rather, the risk of death was found to be principally driven by age, premorbid functioning, and severity of illness. Although it has been highlighted that females may receive substandard levels of care compared with males,[18,19] our finding of males being 27% more likely to die than females in the year after stroke does not provide strong support for this possibility. This finding of a poorer stroke survival in males is mirrored in publications from Asia, Europe, and North America.[20-22]

With regard to surrogate markers for individual SES, social class (professional versus white collar, blue collar, or other) was not found to have a significant impact on survival, but being actively employed before having a stroke was found to be protective. This may be an artefact reflecting the younger age of patients who work, but it may also be attributable to increased levels of social support or networks, whether real or perceived, in a working environment. Social network measures, especially of the number of different fields in which a person has social contacts, have been shown to be strongly associated with improved survival up to 15 years after incident stroke.[23]

Our study has several limitations. Firstly, the use of Australian postcodes, which may cover hundreds of households, is a crude measure of deprivation when compared with New Zealand meshblocks, which contain, on average, 90 people. The Australian and New Zealand data may therefore not be directly comparable, but they are aggregated up to quintiles, so any measurement error is likely to be minimised. Secondly, although the NZDep and IRSD indexes were developed using similar factors from New Zealand and Australian census data, and thus share comparable indices of disadvantage, variations in casemix and measurement error could still have been a source of bias that could have driven our results towards finding no relationship between case-fatality rate and SES. As our main SES index was area-level deprivation, the results are also subject to "ecological fallacy", where associations at the area level may not necessarily hold true at the individual level. Nonetheless, the finding that employment status, a surrogate marker of individual SES, was also associated with outcome, provides support for the reliability of our data. Moreover, because of data limitations, we were unable to consider the impact of other variables, such as education, home or goods ownership, and early-life influences, which have all been shown

4 Odds ratios (95% CI) for the association of clinical characteristics, smoking and drinking with case-fatality rates for stroke at 1 year

	Model 4 (*n* = 2912*)	Model 5 (*n* = 2912*)
Pre-stroke institutional care		
Yes	2.06 (1.56–2.71)	2.06 (1.56–2.71)
Unknown	2.17 (1.46–3.21)	1.93 (1.28–2.90)
History of hypertension	0.83 (0.68–1.01)	0.83 (0.68–1.01)
History of diabetes	1.48 (1.14–1.92)	1.46 (1.12–1.89)
History of myocardial infarction	1.76 (1.35–2.29)	1.79 (1.37–2.34)
History of atrial fibrillation	1.64 (1.31–2.05)	1.63 (1.30–2.04)
Smoking behaviour (reference: prestroke, non-smoker)		
Current smoker	2.08 (1.54–2.82)	2.03 (1.50–2.76)
Unknown	1.80 (1.20–2.69)	1.70 (1.13–2.56)
Drinking behaviour (reference: prestroke, ex/non-drinker)		
Current drinker	0.71 (0.57–0.90)	0.74 (0.59–0.93)
Unknown	1.73 (1.20–2.50)	1.59 (1.09–2.30)
Stroke subtype (reference: ischaemic stroke)		
Intracerebral haemorrhage	2.38 (1.78–3.17)	2.28 (1.71–3.05)
Subarachnoid haemorrhage	2.31 (1.45–3.67)	2.38 (1.48–3.83)
Unknown	2.03 (1.41–2.93)	2.03 (1.40–2.93)
Un- or semiconscious within 24 h of onset	6.32 (5.09–7.85)	6.35 (5.11–7.89)

* Excluding missing patient data for: previous diagnoses of hypertension (37), diabetes (16), myocardial infarction (24) and atrial fibrillation (16), and unconscious or semiconscious within 24 h of onset (11). ◆

5 Odds ratios (95% CI) for the association of individual socioeconomic factors with case-fatality rates for stroke at 1 year

	Model 5 (*n* = 2912)
Employment status*	
Full or part-time work	0.56 (0.33–0.94)
Retired	0.83 (0.57–1.22)
Unknown	1.33 (0.81–2.18)
Social class†	
White collar	1.00 (0.73–1.36)
Blue collar	0.96 (0.73–1.26)
Unknown	1.23 (0.92–1.65)

* Reference: not working. † Reference: professional. ◆

previously to play a role in the occurrence of disease.[24,25] Further, our sample size, although large, may still have been underpowered to detect any influence of area-level SES on case-fatality rate. Finally, without any matched control data or population-based data on potential confounding variables, our study only provides evidence that area-level SES influences the rates of stroke, and does not provide any explanations for the reasons for this disparity. Whether such disparity is due to differential risk-factor profiles, in terms of prevalence, severity or susceptibility to disease, or access to care and intensity of management, requires elaboration in future research.

In summary, this study provides evidence of an association between area-level SES and stroke incidence rates in Australia and New Zealand, but of no clear relationship between SES and survival at 12 months. Although the latter finding provides reassurance that the systems of health care in the two countries do not appear to discriminate against people in more disadvantaged areas, the former reaffirms the importance of targeted prevention directed at people living in disadvantaged areas where there are disproportionately high rates of disease.

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COMPETING INTERESTS

None identified.

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