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Differences in cardiovascular mortality between Australia and New Zealand according to socioeconomic status: findings from the Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study

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Abstract

Background Cardiovascular mortality is higher in New Zealand compared to Australia, but reasons for this difference are uncertain. This study describes differences in cardiovascular risk factors and cardiovascular mortality in Australians and New Zealanders with stable coronary artery disease stratified by socioeconomic status.

Methods Socioeconomic status was estimated from the residential area of 5949 Australians and 2784 New Zealanders with a history of myocardial infarction or unstable angina who participated in the Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study. Socioeconomic and international differences in cardiovascular risk factors, medical treatments, and cardiovascular mortality during a median follow-up period of 7.8 years were evaluated.

Results Cardiovascular mortality increased as the median residential-area income decreased in both Australia (hazard ratio [HR]/income tertile 1.20, 95% confidence interval [CI] 1.08–1.32) and New Zealand (HR 1.16, 95%CI 1.02–1.31), but was higher in New Zealand across all socioeconomic groups (HR 1.42, 95%CI 1.25–1.61). Obesity, smoking, and a high white blood cell count at baseline were associated with higher cardiovascular mortality and were more common in lower-income areas in both countries.

The total:HDL cholesterol ratio was higher in New Zealand, but similar across all socioeconomic groups. In both countries there were socioeconomic gradients in open-label usage of cholesterol-lowering medication, percutaneous coronary intervention, and coronary artery bypass surgery. However, Australians in all socioeconomic groups were more likely than New Zealanders to receive these treatments.

Conclusions Although there is an important socioeconomic gradient in cardiovascular mortality in both Australia and New Zealand, cardiovascular mortality is higher in New Zealanders than Australians with stable coronary disease from all socioeconomic groups.

International differences in cardiovascular morbidity and mortality appear to be strongly influenced by social, economic, and political factors.[1]

Cardiovascular mortality is falling in the established market economies of North America, Western Europe, Australia, and New Zealand[2,3]—but differs substantially

between countries and between different socioeconomic groups within these countries.[4] It has been estimated that approximately half of the decline in mortality is due to improvements in the treatment and secondary prevention of coronary artery disease.[5]

Besides differences in medical care, differences in diet, lifestyle, psychosocial stress, and other socioeconomic factors may contribute to mortality differences between populations.[6–8]

It is possible that international differences in cardiovascular disease among the established market economies are driven by the same mechanisms that are responsible for socioeconomic differences in cardiovascular disease within these countries.[2] Further, it has been proposed that strategies to reduce socioeconomic gradients within a country may also reduce overall mortality.[9]

Australia and New Zealand have broadly similar political systems, cultures, and socioeconomic gradients in wealth, but mortality from ischaemic heart disease is about 25% higher in New Zealand than in Australia.[10] The Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study was a cholesterol-lowering clinical trial comparing the 3-hydroxy-3-methylglutaryl coenzyme-A (HMGCoA) reductase inhibitor, pravastatin, with placebo treatment in 9014 stable patients with a history of myocardial infarction or hospitalisation for unstable angina, randomised throughout Australia and New Zealand between 1990 and 1992.[11]

The aim of this analysis was to determine whether differences in cardiovascular mortality between Australian and New Zealand LIPID participants were mediated by the same mechanisms that influenced socioeconomic mortality differences within each country, or by different mechanisms.

Methods

Study population—The LIPID Study enrolled Australian and New Zealand men and women aged 31 to 75 years with a history of acute myocardial infarction or hospitalisation for unstable angina within the previous 3 months to 3 years. The exclusion criteria included significant illness during the preceding 3 months, unavailability for long-term follow-up, significant cardiac failure (New York Heart Association [NYHA] class III or IV), and treatment with cholesterol-lowering medication.

After a run-in phase, participants with a fasting total serum cholesterol level in the range of 4.0 to 7.0 mmol/L and a serum triglyceride level of ≤5.0 mmol/L were randomly assigned to receive either pravastatin 40 mg/daily or a matching placebo. All participants received dietary and general lifestyle advice. The patients' care was otherwise under the direction of their usual doctors, including the option for commencement of open-label cholesterol-lowering medication if deemed necessary.

Baseline assessment—The following cardiovascular risk factors were documented at the baseline assessment: current smoking, height, body mass index, measured systolic and diastolic blood pressure, reported diagnosis of hypertension, fasting blood glucose level, reported diagnosis of diabetes, urine protein level, urine sugar level, white blood cell count, serum total and high-density lipoprotein (HDL) cholesterol levels, and the triglyceride level.

The following clinical measures of disease severity were documented: history of myocardial infarction, myocardial infarction prior to the qualifying event, angina duration, Canadian Cardiovascular Society (CCS) angina classification, and NYHA symptom classification. Usage of drug treatments including aspirin, beta-blockers, angiotensin-converting-enzyme (ACE) inhibitors, and calcium antagonists was also documented, as was percutaneous coronary intervention (PCI) or coronary artery bypass surgery (CABG) before the qualifying event or between the qualifying event and randomisation.

Follow-up—Randomisation for the LIPID Study took place between June 1990 and December 1992. Routine visits were scheduled every 6 months thereafter. Drop-in to active cholesterol-lowering

treatment during the study was documented. Discontinuation of the study medication for longer than 2 weeks was classified as temporary discontinuation. All acute cardiovascular events and hospital admissions were documented, including admissions for PCI or CABG.

Information on deaths was obtained from hospital records, death certificates, autopsy reports, and physicians' notes—and an Outcome Assessment Committee reviewed the documentation on all deaths from coronary heart disease, including fatal myocardial infarction, sudden death, death in hospital after possible myocardial infarction, and death related to heart failure.

At the final study follow-up visit in 1997, all patients were offered open-label pravastatin treatment and invited to participate in the LIPID Cohort Study.[12] Patients unable or unwilling to attend further clinic visits were asked to give consent for follow-up of their health status through their usual doctor and/or medical records. The vital status of all but two individuals in the LIPID Study and LIPID Cohort Study was documented to December 1999.

Measurement of socioeconomic status—A residential-area-based measure of socioeconomic status was obtained by linking the street address at randomisation to the corresponding census collection districts of 5949 (99.8%) Australian and 2784 (91.2%) New Zealand LIPID participants. The population census used for the analysis were undertaken on 5 March 1991 in New Zealand and 6 August 1991 in Australia. The median income of the general population aged >15 years in each census collection district (averaging approximately 200 households) was then obtained from the Australian Bureau of Statistics or Statistics New Zealand.

For comparison of the LIPID Study participants with the general population of each country, subjects were classified into tertiles based on the median income of the general population in their residential area, matched for age (in 5-year bands) and gender in each country.

Statistical analysis—The dataset used for analysis included all LIPID participants to whom we were able to assign a residential-area income value. Income was examined separately for each country as a continuous variable and also as a categorical variable stratified into deciles and tertiles. Other variables were also examined as both continuous and categorical variables. For binary versions, the median value of the LIPID population was used where an accepted cut-off level had not been prespecified (height, baseline white blood cell count, serum lipids). For ease of presentation, univariate results are shown for income tertiles and the binary versions of other variables.

Chi-squared (χ^2) tests were used to compare proportions, and the Chi-squared test for trends was used to evaluate linear trends in proportions. For age, the Student's t-test was used to compare means, and linear regression was used to evaluate linear trends. Standard survival analysis methods were used to compare cardiovascular mortality by country and by income, including Kaplan-Meier survival curves and Cox regression methods. PCI and CABG during follow-up were treated as time-dependent covariates. A modified backwards-stepwise procedure was used to select the models.

Variables were included in the final model if they were significantly associated with a risk of cardiovascular mortality, or if they confounded the associations between cardiovascular mortality and country or residential-area income. Residential-area income was included in the final model as a linear term, centred at decile 6. This enhanced the comparability of income classifications between Australia and New Zealand, and there was no evidence of any departure from a linear decrease in the log hazard with a decrease in the income decile. The proportion of the country and income effects explained by measured prognostic variables was estimated using the landmark method:[13]

Bootstrap confidence intervals were constructed with 500 replications using Stata Statistical Software (Release 7.0, Stata Corporation, College Station, Texas, USA).

Results

Residential-area income by country

The median residential-area incomes of LIPID participants were broadly representative of the general population in both Australia and New Zealand. Australian participants had a median residential-area income of AU$13,538 (interquartile range [IQR] AU$11,408 to $16,049), while New Zealand participants had a median residential-area income of NZ$14,487 (IQR NZ$12,501 to $17,502).

The median residential area income of Australian participants in $NZ was $17,813 (IQR $15,013 to $21,120) using an estimated exchange rate of AU$0.76=NZ$1.00 for 1991. Thirty-three percent of Australian participants lived in the lowest, 35% in the middle, and 31% in the highest residential-area income tertile of the general population matched for age and gender, while 29% of New Zealand participants lived in the lowest, 36% in the middle, and 35% in the highest income tertile.

Both countries had a similar distribution of income. In Australia, the median income of the middle tertile was 1.3 times that of the lowest tertile, while the median income of the highest tertile was 1.6 times that of the lowest tertile. The corresponding figures in New Zealand were 1.2 and 1.6 times higher, respectively.

Cardiovascular risk factors

The distributions of age and sex were similar in both countries. Participants who lived in higher-income areas were more likely to be younger and male, but this trend was small (Table 1). A higher percentage of Australians than New Zealanders had a reported diagnosis of hypertension, although the percentages of participants with a measured blood pressure of ≥140/90 mmHg at baseline were similar in both countries.

Australians were also more likely to have a diagnosis of diabetes, although blood glucose levels were similar in both countries. New Zealanders had a higher total:HDL cholesterol ratio than Australians. Similar percentages of the participants in both countries were current smokers, obese (body mass index >30 kg/m^2), of shorter stature, had a high baseline white blood cell count, or had a high serum triglyceride level.

Socioeconomic gradients in risk factors differed from those between countries. Neither country had a clear socioeconomic gradient in hypertension, measured blood pressure ≥140/90 mmHg, elevated blood glucose, or serum total:HDL cholesterol ratio. Lower-income participants in both countries were more likely to be current smokers, to be obese, and to be of shorter stature than higher-income participants. In Australia, lower-income participants were more likely to have a high white blood cell count than higher-income participants.

Treatments

At baseline, Australians were more likely to be taking a calcium antagonist, and New Zealanders were more likely to be taking a beta-blocker or ACE inhibitor. The usage of aspirin was similar in both countries. Australians were more likely to be treated with PCI or CABG than New Zealanders. During follow-up in the double-blind phase of the trial, New Zealanders were less likely to discontinue the study medication, while Australians were more likely to receive off-study open-label cholesterol-lowering medication.

In both countries, lower-income participants were less likely to be taking aspirin, but more likely to be taking calcium antagonists. The percentages of participants who discontinued the study medication were similar across all socioeconomic groups. Lower-income participants were less likely to be treated with off-study cholesterol-lowering medication, PCI, or CABG than higher-income participants.

Table 1. Age, gender, and cardiovascular risk factors stratified by country and by socioeconomic group in Australia and New Zealand

Cardiovascular risk factors at baseline	Country of residence	All (%)*	P value	Residential-area income tertile (%)*			P value for trend
				Lower	Middle	Higher	
Mean age (years)*	Australia	61		61	61	60	<0.0001
	New Zealand	61	0.9	62	61	60	<0.0001
Male gender	Australia	84		81	84	86	<0.0001
	New Zealand	82	0.01	79	81	85	0.0002
History of hypertension	Australia	44		44	45	44	0.9
	New Zealand	37	<0.0001	38	38	35	0.3
Blood pressure ≥140/90 mmHg	Australia	48		49	49	46	0.07
	New Zealand	47	0.6	49	46	46	0.2
Diagnosed diabetes	Australia	9		10	10	8	0.2
	New Zealand	7	0.003	8	8	6	0.02
Fasting glucose >7 mmol/L	Australia	9		9	9	8	0.5
	New Zealand	8	0.3	8	7	9	0.2
Urine protein present	Australia	5		6	5	5	0.2
	New Zealand	5	0.5	4	5	6	0.1
Total:HDL cholesterol ratio >6.1	Australia	48		50	48	48	0.2
	New Zealand	53	<0.0001	52	55	52	0.9
HDL cholesterol <1.0 mmol/L	Australia	62		62	62	63	0.5
	New Zealand	64	0.01	62	68	63	0.7
Triglycerides ≥1.6 mmol/L	Australia	51		52	50	50	0.2
	New Zealand	49	0.07	47	52	48	0.7
White blood cell count ≥8.2 × 10^9/L	Australia	26		30	26	23	<0.0001
	New Zealand	27	0.4	28	28	26	0.4
Current smoker	Australia	9		11	9	9	0.02
	New Zealand	10	0.4	12	11	7	0.001
Body mass index >30	Australia	18		20	18	15	<0.0001
	New Zealand	18	0.6	21	19	15	0.0005
Height <166 cm	Australia	24		27	25	21	<0.0001
	New Zealand	25	0.5	30	24	21	<0.0001

*Table shows the percentage of participants (%) with each risk factor, except for age, where the mean age is shown.
HDL = high-density lipoprotein.

Table 2. Measures of cardiovascular disease severity, cardiovascular symptoms, and treatments stratified by country and by socioeconomic group in Australia and New Zealand

	Country of residence			Residential-area income tertile (%)			P value for trend
		%	P value	Lower	Middle	Higher	
Cardiovascular disease at baseline							
History of myocardial infarction	Australia	71		72	70	73	0.4
	New Zealand	74	0.02	72	73	73	0.04
Angina duration >5 years	Australia	13		16	14	10	<0.0001
	New Zealand	14	0.5	16	13	11	0.0007
Dyspnoea, NYHA class ≥2	Australia	50		53	50	46	<0.0001
	New Zealand	49	0.4	52	50	44	0.0003
Angina, CCS class ≥2	Australia	35		39	34	31	<0.0001
	New Zealand	41	<0.0001	43	45	35	0.0009
Treatments at baseline assessment							
Aspirin	Australia	82		80	82	84	0.0001
	New Zealand	83	0.3	80	83	85	0.006
ACE inhibitor	Australia	15		15	16	14	0.1
	New Zealand	17	0.01	20	16	16	0.03
Beta-blocker	Australia	45		45	45	46	0.6
	New Zealand	50	0.001	49	51	48	0.7
Calcium antagonist	Australia	37		40	36	35	0.002
	New Zealand	29	<0.0001	30	34	24	0.003
Previous PCI*	Australia	16		14	17	17	0.02
	New Zealand	11	<0.0001	10	11	11	0.4
Previous CABG*	Australia	33		30	32	26	<0.0001
	New Zealand	25	<0.0001	24	25	27	0.16
Treatments during follow-up							
Off-study cholesterol treatment	Australia	17		15	15	20	<0.0001
	New Zealand	11	<0.0001	10	12	12	0.1
Discontinued study treatment†	Australia	33		33	33	32	0.5
	New Zealand	25	<0.0001	26	25	24	0.3
PCI during follow up	Australia	8		6	7	9	0.0001
	New Zealand	6	0.02	5	6	7	0.07
CABG during follow-up	Australia	14		13	13	15	0.03
	New Zealand	11	0.004	9	13	13	0.005

*Includes PCI or CABG before or after the qualifying event; †Includes temporary and permanent discontinuation; ACE = angiotensin-converting-enzyme; CABG=coronary artery bypass surgery; CCS=Canadian Cardiovascular Society; NYHA = New York Heart Association class; PCI = percutaneous coronary intervention.

Symptoms of cardiovascular disease

Exertional dyspnoea was more common among New Zealanders than among Australians, although dyspnoea classifications were similar in both countries (Table 2). Lower-income groups in both countries were more likely to report angina and dyspnoea, and to have a longer history of angina. There was a small but statistically significant difference between countries in the proportion of subjects with a history of myocardial infarction, but no such difference between socioeconomic groups.

Figure 1. (A) Cardiovascular mortality and (B) non-cardiovascular mortality of Australian and New Zealand LIPID participants in the lowest, middle, and highest tertiles of residential area-based income

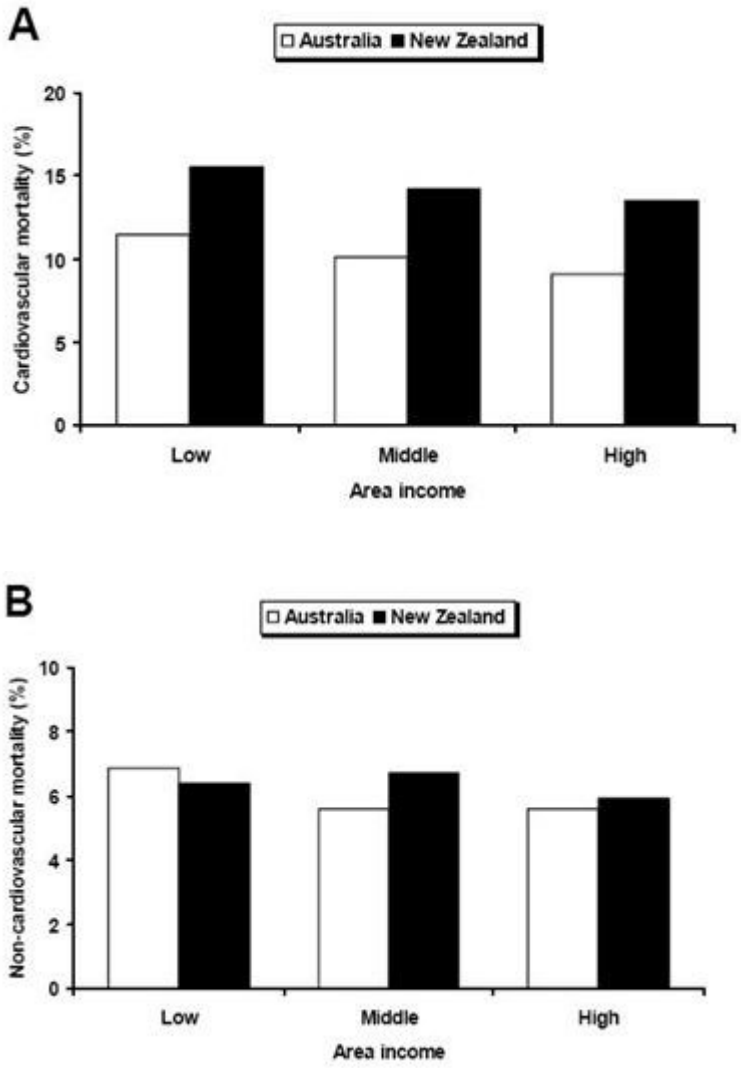

Table 3. Multivariate risk factor model for cardiovascular death in all study participants

Risk predictor	Hazard ratio	95% CI	P value
Cardiovascular risk factors at baseline			
History of hypertension	1.14	(1.00–1.30)	0.047
Fasting glucose >7.0 mmol/L	1.59	(1.33–1.90)	<0.0005
Total:HDL cholesterol ratio*	1.04	(1.02–1.06)	<0.0005
HDL cholesterol <1.0 mmol/L	1.17	(1.01–1.36)	0.03
Triglycerides ≥1.6 mmol/L	0.79	(0.69–0.91)	0.001
Urine protein present	1.50	(1.20–1.87)	<0.0005
White blood cell count†	1.04	(1.03–1.06)	<0.0005
Current smoker	1.71	(1.35–2.17)	<0.0005
Body mass index >30	1.17	(0.99–1.38)	0.06
Cardiovascular disease severity			
History of myocardial infarction	1.67	(1.40–2.00)	<0.0005
Dyspnoea, NYHA class ≥2	1.24	(1.01–2.02)	0.04
Angina, CCS class 1	1.25	(1.08–1.44)	0.002
Angina, CCS class ≥2	1.35	(1.08–1.67)	0.007
Treatments			
Aspirin	0.85	(0.73–0.99)	0.04
ACE inhibitor	1.78	(1.54–2.06)	<0.0005
Beta–blocker	0.84	(0.73–0.96)	0.01
Randomisation to pravastatin	0.72	(0.63–0.81)	<0.0005
Off-study cholesterol-lowering treatment	0.44	(0.34–0.57)	<0.0005
Temporarily discontinued study treatment	0.71	(0.56–0.92)	0.008
Permanently discontinued study treatment	1.06	(0.91–1.25)	0.4
PCI before qualifying event	0.72	(0.49–1.05)	0.09
PCI between qualifying event and randomisation	0.61	(0.45–0.83)	0.002
PCI during follow-up	1.66	(1.23–2.23)	0.001
CABG before qualifying event	1.53	(1.28–1.83)	<0.0005
CABG between qualifying event and randomisation	0.77	(0.64–0.94)	0.01
CABG during follow-up	1.40	(1.13–1.72)	0.002
Country and residential-area income			
Country	1.34	(1.17–1.53)	<0.0005
Residential-area income decile	1.04	(1.01–1.08)	0.003
Country-income decile interaction	0.99	(0.94–1.03)	0.5

The model also included terms for age (as a quadratic) and gender. Height, blood pressure, low-density-lipoprotein cholesterol and calcium antagonist treatment at baseline were not significant (P>0.05) in the multivariate model; *The hazard ratio and CI are for a 1-unit increase in the total:HDL cholesterol ratio; †The hazard ratio and CI are for a 1 x 10^9/L increase in the white blood cell count; ACE = angiotensin-converting-enzyme; CABG=coronary artery bypass surgery; CCS Canadian Cardiovascular Society; HDL=high-density-lipoprotein; NYHA = New York Heart Association; PCI = percutaneous coronary intervention.

Cardiovascular mortality

Cardiovascular mortality during the LIPID Study was higher in New Zealand than in Australia (age- and sex-adjusted hazard ratio [HR] 1.42, 95%CI 1.25–1.61). The difference in non-cardiovascular mortality between the two countries was smaller and not statistically significant (New Zealand versus Australia HR 1.08, 95%CI 0.90–1.29). There was a definite gradient in cardiovascular mortality according to socioeconomic status in both countries, with mortality being higher in participants from lower-income areas (Figure 1A).

The increase in cardiovascular mortality with a decrease in residential-area income of one tertile was similar in both countries (Australian HR 1.20, 95%CI 1.08–1.32; New Zealand HR 1.16, 95%CI 1.02–1.31). There was also a graded increase in non-cardiovascular mortality with a decrease in residential-area income of one tertile in Australia (HR 1.18, 95%CI 1.04–1.35), but this gradient was not statistically significant in New Zealand (HR 1.04, 95%CI 0.87–1.24; Figure 1B).

There was no evidence that the benefits of pravastatin differed between countries or between socioeconomic groups.

Multivariate model

Multivariate predictors of cardiovascular mortality are presented in Table 3. After adjustment for all prognostic variables, the excess risk of cardiovascular death in New Zealand compared with Australia was reduced from 42% (with the analysis adjusted only for age and sex) to 35%. The difference in cardiovascular mortality between the two countries was present at all income levels, with a slightly greater difference in the higher-income deciles.

In Australia, the risk of cardiovascular death increased by 58% from the highest to the lowest income decile after adjustment for age and sex, and by 46% after adjustment for all baseline prognostic factors. In New Zealand, the risk of cardiovascular death increased by 45% from the highest to the lowest income decile after adjustment for age and sex, and by 35% after adjustment for all baseline prognostic factors.

Discussion

In this study, which recruited representative coronary heart disease patients, mortality in both Australia and New Zealand increased progressively as the median residential-area income decreased. However, while non-cardiovascular mortality was similar in both countries, cardiovascular mortality was about 40% higher in New Zealand across all socioeconomic groups. This excess was not explained by coronary heart disease risk factors associated with socioeconomic status, or by greater socioeconomic disparities in measures of disease severity, treatments or income in New Zealand. There were treatment differences between countries across all socioeconomic groups, but the extent of their influence upon the differences in cardiovascular mortality observed in the study is uncertain.

Off-study open-label cholesterol-lowering drug treatments, which were relatively expensive during the 1990s, were more widely used by higher-income participants than lower-income participants during the double-blind phase of the trial, and more widely prescribed in Australia than in New Zealand. Government policy to restrict access to statins in New Zealand at this time may have contributed to these differences.[14]

The rates of PCI and CABG were lower in New Zealand than in Australia, and lower in lower socioeconomic groups within both countries. This was despite a greater prevalence of angina in New Zealand and in lower-income participants. However the impact of different rates of revascularization on outcomes is difficult to evaluate in this study. In addition accurate information on indications for PCI/CABG and LV ejection fraction is not available.

Clinical trials suggest coronary revascularisation does not decrease mortality for most patients with stable coronary artery disease,[15] but PCI early after an acute coronary syndrome decreases the risk of recurrent myocardial infarction.[16]

The average serum total:HDL cholesterol ratio was higher in New Zealand than in Australia. The explanation for this is unclear, but may reflect differences in the national diet, genetic factors or selection of participants for the trial. There was no socioeconomic gradient in serum lipid levels.

Although Australians were more likely to have received a diagnosis of hypertension or diabetes, measured blood pressure and blood glucose levels were similar in both countries. This may suggest that the diagnoses of hypertension and diabetes were made earlier in Australia. There were no clear international differences in other cardiovascular risk factors.

In contrast to international differences, there was no socioeconomic gradient in serum lipid levels or hypertension, but obesity and smoking were more prevalent in lower socioeconomic groups. These lifestyle risk factors probably reflect health behaviours that are established by young adulthood and continue to influence disease rates for many years.

The socioeconomic gradient in the white blood cell count, which was more evident in Australia, suggests that inflammation may be more prevalent in individuals from lower-income areas. The association between the white blood cell count and cardiovascular mortality in this study[17] is consistent with previous studies in which non-specific markers of systemic inflammation were associated with an increased risk of first or recurrent cardiovascular events.[18]

Previous analyses suggest that multiple factors have contributed to the decline in coronary heart disease mortality both in New Zealand[19] and internationally during the 1980s and 1990s.[20] In a recent analysis from the US, about half the decline in coronary heart disease mortality, was attributable to changes in risk factors and about half to medical treatments.[20]

In contrast, in the current study, measured variables appear to explain only a small proportion of the international and socioeconomic differences in cardiovascular event rates in the multivariate analysis.[21] There are several possible explanations. A number of important risk factors were not measured including diet, exercise, psychosocial variables and many aspects of medical care. Categorisation of risk factors as “present” or “absent” will under estimate the graded association between most risk factors and events. Finally, multivariate analysis may not appropriately adjust for effects of treatments which are known from randomized clinical trails to improve outcomes.

The study population was selected by agreement to participate in a clinical trial. The socioeconomic distribution of study participants was similar to that of the general population in both Australia and New Zealand. However, certain groups may have been under-represented, and we have no data on ethnicity. It is also possible there were differences in selections of patients invited to participate in the study between countries. For example New Zealand participants were more likely on average to have a history of angina, suggesting they may have more severe coronary disease at baseline.

Participation in the LIPID Study may also have influenced standards of care, and it is possible that the magnitude of treatment differences was greater or smaller than in routine medical practice. The proportion of current smokers was lower than in the general population, but the proportion of ex-smokers was higher, reflecting the impact of the diagnosis of coronary artery disease on smoking behaviour.

Census-based measures of socioeconomic status have been consistently associated with health outcomes in many studies.[4,22,23] However, because income is heterogeneous within areas, an aggregate measure is a crude estimate of individual income, which would underestimate any socioeconomic gradient related to personal income.

Residential-area-based measures do, however, include information on other health effects associated with the residential area.[24,25] They also have the advantage of being determined by long-term socioeconomic status, and may therefore be more reliable than current income when assessing health over many years in a study involving participants who may be working, retired or unemployed at the time of assessment. It is difficult to adequately account for international differences in buying power. For this reason, relative differences between residential areas and between countries were used for the analysis.

In conclusion, this study suggests that there are differences in the risk factors that contribute to the international and socioeconomic gradients in cardiovascular mortality in Australia and New Zealand. This suggests that specific strategies are needed to reduce socioeconomic gradients in cardiovascular mortality in Australia and New Zealand, and these may differ from those designed to reduce national cardiovascular mortality rates.

Competing interests: None of the authors has any conflict of interest in connection with this work.

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