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The burden of modifiable cardiovascular risk factors in the coronary care unit by age, ethnicity, and socioeconomic status—PREDICT CVD-9

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Abstract

Aims To investigate the burden of modifiable cardiovascular disease (CVD) risk factors in patients admitted to coronary care by age, ethnicity, and socioeconomic status.

Design and setting Cross-sectional study of patients presenting to the Middlemore Hospital Coronary Care Unit with an acute CVD event from July 2004 to June 2006.

Methods CVD risk factor data was electronically collected using Acute PREDICT. Socioeconomic status was estimated using the NZ Deprivation 2001 index (NZDep01).

Results Of 973 patients 34% were <55 years and 10% were <45 years, 24.8% were women, and 44.6% lived in areas classified as most deprived. 61.5% were European/other, 13.0% NZ Māori, 15.2% Pacific, and 10.3% South Asian. Younger patients, regardless of ethnicity, were much more likely to be smokers, be obese, have elevated LDL and trigyceride, and low HDL levels. Māori and Pacific patients were more likely than European/other patients to smoke, have diabetes, obesity, elevated triglycerides, and low HDL. These ethnic differences persisted across the age range. Increasing deprivation was associated with more smoking, obesity, hypertriglyceridaemia and diabetes, with the excess of smoking and obesity being most pronounced in younger patients.

Conclusions In patients presenting to coronary care, there is a high burden of adverse modifiable CVD risk factors, particularly in younger patients and among Māori and Pacific people from areas of high deprivation. These risk factors are a major and reversible contributor to future CVD risk in these groups, and an important target for secondary prevention programs.

Cardiovascular disease (CVD) is the leading cause of death and hospitalisations for New Zealanders. Those who suffer a CVD event such as a myocardial infarction are at very high risk for further events. However this risk could be reduced by as much as 80% by targeting modifiable risk factors which include poor diet, obesity, physical inactivity, high blood pressure, hyperlipidaemia, and smoking using appropriate combinations of lifestyle modification and effective preventive medications.[1]

CVD increases with age and occurs earlier in men than in women but there are also disparities in cardiovascular health outcomes by ethnicity and by socioeconomic deprivation. Age-specific coronary disease mortality rates for Māori and Pacific people are 2–3 times those of non-Māori non-Pacific.[2]

A similar magnitude of increased mortality occurs for those from the most deprived compared to the least deprived areas.[3] The prevalence of poorly controlled modifiable CVD risk factors is known to be high in these groups from population studies.[4,5]

The aim of this study was to describe the burden of modifiable cardiovascular risk factors in patients presenting to a Coronary Care Unit (CCU) in South Auckland by age, ethnicity, and socioeconomic status.

Methods

Acute PREDICT—From 2004 the PREDICT-CVD electronic decision support program for CVD risk assessment and management[6] was implemented in the CCU of Middlemore Hospital as part of the 'Acute PREDICT' program.

Nursing and junior medical staff were encouraged to use PREDICT to assess and manage cardiovascular risk in individual patients admitted with an acute cardiovascular event when the patient was in a stable condition (usually at Day 2 of admission). Patients who died early, were too clinically unstable, or were discharged before an assessment could be completed, were not entered into PREDICT.

The program was accessed on all the CCU computers via the hospital intranet and communicated with a decision support server within CMDHB Information Technology services. All data items (demographic, CVD risk factors, and management variables) were manually entered. The ward clerk entered the demographic details from the hospital patient information system on admission to CCU whilst the clinical details were subsequently entered by nursing staff and verified by medical staff.

Once all the required data was entered, it was sent to the central server and within seconds the clinician received evidence-based risk management recommendations derived from New Zealand CVD guidelines.[7] Each time PREDICT was used, an electronic CVD profile was stored. For the purposes of this study, the first completed assessment for each patient from 1 July 2004 to 30 June 2006 was extracted and made available to the research team with only the National Health Index (NHI) number retained as an unique identifier.

Data and definitions – Detailed data definitions have been published previously.[6] In brief, demographic data collected included age, gender, ethnicity, and the National Health Index (NHI) number. For these analyses ethnicity was categorised in four groups: New Zealand Māori, Pacific, South Asian, and European/other. Pacific peoples were defined according to New Zealand Health Information Service ethnicity data protocol[8] as having Level 2 codes 31 to 37 and South Asian defined as Level 2 codes 43 and 44 excluding Japanese and Korean—i.e. being Indian, Fijian Indian, Pakistani, Sri Lankan, Bangladeshi, Nepali, Afghani, or Tibetan.

As a measure of socioeconomic status (SES) the domicile code (where available and valid) for each patient was obtained from the hospital information system. This was linked to New Zealand Deprivation 2001 (NZDep01) index and reported as decile of deprivation from 1 (least deprived) to 10 (most deprived). The NZDep2001 is a small area index of deprivation that provides a score for each meshblock in New Zealand based on nine variables (material and social domains of deprivation) from the 2001 Census.[9,10]

CVD risk factor data included family history of premature ischaemic cardiovascular disease, diagnosis of Type 2 diabetes, smoking status (smoker, non-smoker, or past smoker who quit more than 12 months ago), systolic and diastolic blood pressure (mmHg) and lipids (total cholesterol/HDL ratio, LDL, HDL and triglycerides), and body mass index (BMI).

The blood pressure was the mean of two consecutive readings when the patient was clinically stable on day two or three in hospital. Lipid data entered was the lipid profile drawn at hospital admission in the emergency department.

Details regarding prior CVD admissions, including ischaemic heart disease (IHD), stroke or transient ischaemic attack (TIA), peripheral vascular disease (PVD), percutaneous coronary intervention (PCI), and/or coronary artery bypass graft (CABG), for patients in the cohort were obtained from Counties Manukau District Health Board case-mix data. Clinical targets for individual CVD risk factors are drawn from the New Zealand CVD guidelines.[7]

To compare the clustering of risk factors in individual patients each patient was scored out of 7 according to the number of guidleline risk factor targets not achieved. Patients received one point for each of the following—BMI ≥30 kg/m^2, smoker, Type 2 diabetes, systolic BP >130 mmHg, LDL cholesterol >2.5 mmol/L, HDL cholesterol <1.0 mmol/L, and serum triglyceride level ≥1.7 mmol/L.

The study was approved as an audit by Northern X Regional Ethics Committee (AKY/03/12/314).

Analyses—All analyses were conducted using SAS version 9.1 software, and plots drawn using MS Excel software. Difference in mean age between ethnic groups was tested using a generalised linear model adjusted for ethnicity. Differences in demographics and treatment targets by age group and ethnicity were assessed using the Chi-squared statistic of general association. Differences in demographics and treatment targets by deprivation quintile were assessed using Cochran-Mantel-Haenszel test for non-zero correlation, stratified by age group.

To investigate the association of ethnicity on modifiable risk factors, generalised linear models with adjustment for age, gender, and NZDep01 decile were used.

Results

Between 1 July 2004 to 30 June 2006, 1813 patients were admitted to the Middlemore Hospital Coronary Care Unit with an acute cardiovascular event and 973 (54%) had a PREDICT assessment completed. Patients not assessed with PREDICT were similar in their distribution of ethnicity and socioeconomic status to those assessed. However they were slightly older (61.7±13, 59.9±12y, respectively) and more likely to have had 3 or more previous CVD admissions.[11]

Age and gender (Table 1, and Figures 1–2)—Thirty-four percent of acute CVD presentations to the Middlemore CCU occurred in patients younger than 55 years of age; 10% were in those under 45 years. Younger patients were much more likely to be smokers, be obese, and have elevated LDL and trigyceride levels, and low HDL. However, the number failing to meet New Zealand CVD Guideline target risk factor levels was high in all age groups. The cohort included 75.2% men. Women were on an average of 3 years older than men (62 years and 59 years, respectively). Compared with men they had higher HDL levels (1.5 mmol/L vs 1.2 mmol/L, p<0.0001), lower triglyceride levels (1.9 vs 2.1 mmol/L, p=0.003) but identical mean LDL levels (2.9 mmol/L). Women were more likely to be obese defined as BMI ≥30 kg/m^2 (47% vs 40%, p=0.047), and had higher mean blood pressures (129.5 vs 126.4 mmHg, p=0.03). Rates of smoking and Type 2 diabetes were not significantly different.

Ethnicity (Table 2, Figures 1-2)—There were 61.5% classified as European or other ethnicity, 13.0% NZ Māori, 15.2% Pacific, and 10.3% South Asian. The Māori, Pacific, and South Asian patients were younger than the European/other group by 10.7, 9.3, and 7.3 years respectively, and more likely to live in areas of greater deprivation (65%, 83% and 49% in NZDep01 9 or 10, respectively, compared with 30% of European/other patients).

Compared with the European/other patients, the Māori and Pacific patients were more likely to smoke and have higher levels of risk factors associated with the metabolic syndrome,[7] including Type 2 diabetes, obesity, elevated triglycerides, and low HDL. The burden of these risk factors was higher in younger Māori and Pacific compared with the European/other groups, but persisted across the age range. The South Asian patients also had higher rates of Type 2 diabetes, smoking, and elevated triglycerides relative to the European/other group, but rates of obesity were similar. Mean systolic BP and LDL cholesterol did not vary significantly across the ethnic groups.

Table 1. Demographics and failure to meet clinical targets, by age group

	All		Age group					P value
			<45	45–54	55–64	65–74	75+	
	N=973		n=102	n=230	n=274	n=249	n=118	
	100%		(10.5%)	(23.6%)	(28.1%)	(25.6%)	(12.1%)	
	N	%	%	%	%	%	%	
Demographic								
Mean (± SD) age, years	[59.9 ± 11.9]		[39.3]	[50.1]	[59.2]	[69.4]	[78.1]	-
Women	241	24.8	22.5	18.3	21.5	31.7	32.2	**0.0006**
European & other*	598	61.5	36.3	44.3	57.3	75.9	95.8	
NZ Māori	127	13.1	27.5	19.1	15.0	4.4	2.5	**<0.0001**
Pacific†	148	15.2	22.5	22.6	16.8	10.4	0.8	
South Asian	100	10.3	13.7	13.9	10.9	9.2	0.8	
Deprivation**								
NZ Dep 1/2	129	13.3	6.9	12.6	12.8	14.5	18.6	
NZ Dep 3/4	122	12.5	7.8	9.1	13.1	14.9	16.9	
NZ Dep 5/6	102	10.5	8.8	6.5	11.7	12.4	12.7	**0.0003**
NZ Dep 7/8	175	18.0	15.7	18.7	14.2	20.5	22.0	
NZ Dep 9/10	434	44.6	59.8	52.6	47.4	34.9	29.7	
History and clinical targets								
First admission	644	66.2	69.6	65.2	71.5	62.2	61.0	0.12
Type 2 diabetes	239	24.6	19.6	21.7	29.2	29.3	13.6	**0.0027**
Current smoker	262	26.9	60.8	37.4	27.7	11.6	7.6	**<0.0001**
Systolic BP >130 mmHg	332	34.1	18.6	33.0	30.7	40.2	44.9	**0.0002**
HDL <1.0 mmol/L	178	18.3	23.5	19.1	18.6	16.1	16.1	0.52
LDL >2.5 mmol/L	531	54.6	64.7	61.7	60.6	43.4	41.5	**<0.0001**
Triglycerides ≥1.7 mmol/L	507	52.1	69.6	59.1	53.3	42.2	41.5	**<0.0001**
BMI ≥30	407	41.8	58.8	51.7	43.4	34.9	18.6	**<0.0001**
Waist >100 cm(M) or >90 cm(F)	683	70.2	71.6	72.2	72.6	70.3	59.3	0.09

* Includes European and all other specific ethnicity groups other than Māori , Pacific and South Asian, and also those with missing information
**not available for 11 patients
† Mostly of Samoan, Tongan, Niuean, or Cook Islands origin

Figure 1. Percentage of current smokers (top panel), mean BMI (kg/m^2) (middle) and percentage of patients with Type 2 diabetes (lowest panel), shown as a function of age for both ethnicity (left) and SES estimated by decile of deprivation (right)

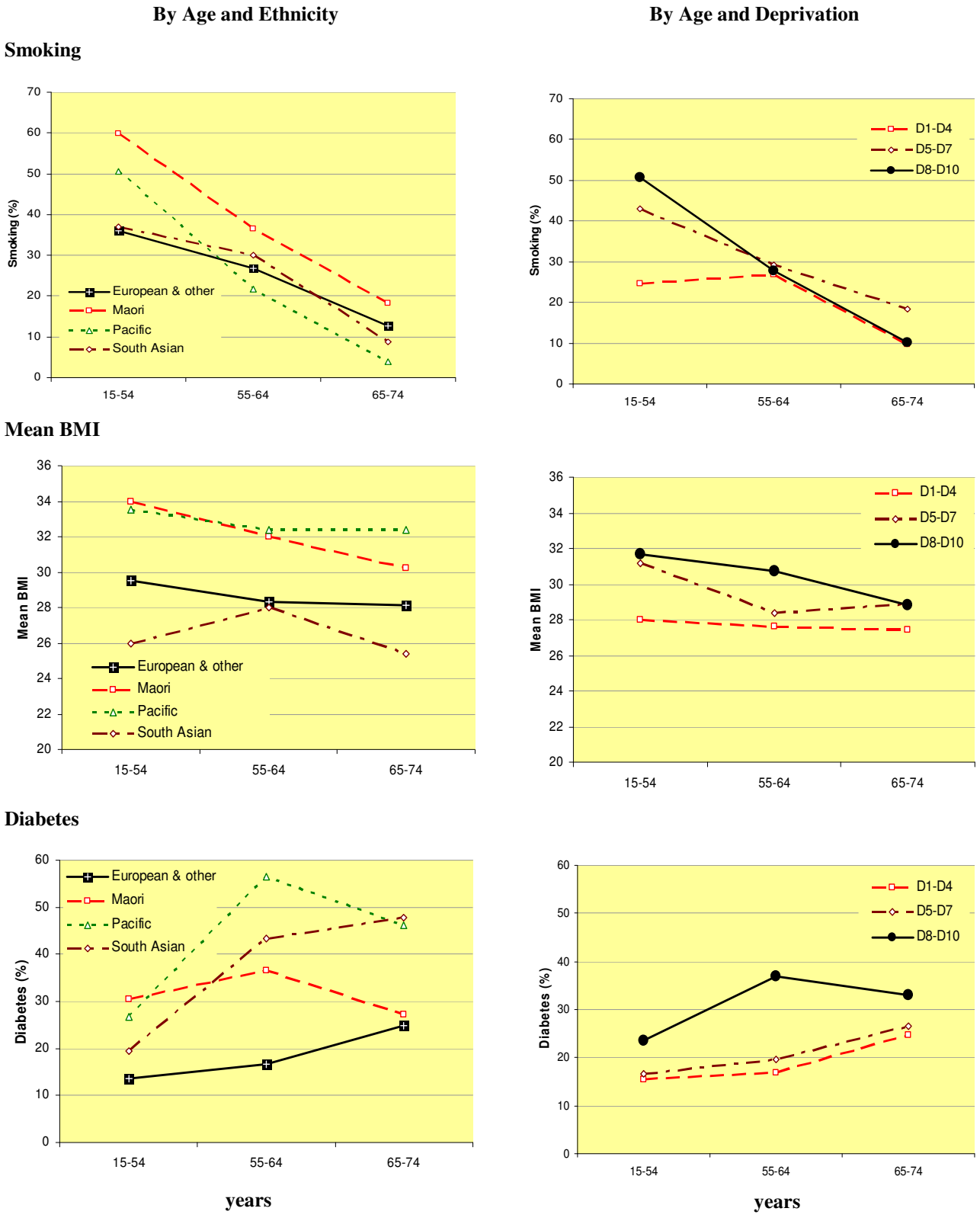


Figure 2. Mean LDL (top panel), HDL (middle) and triglyceride levels (below) in mmol/L are shown as a function of age group for both ethnicity (left) and SES estimated by decile of deprivation (right)

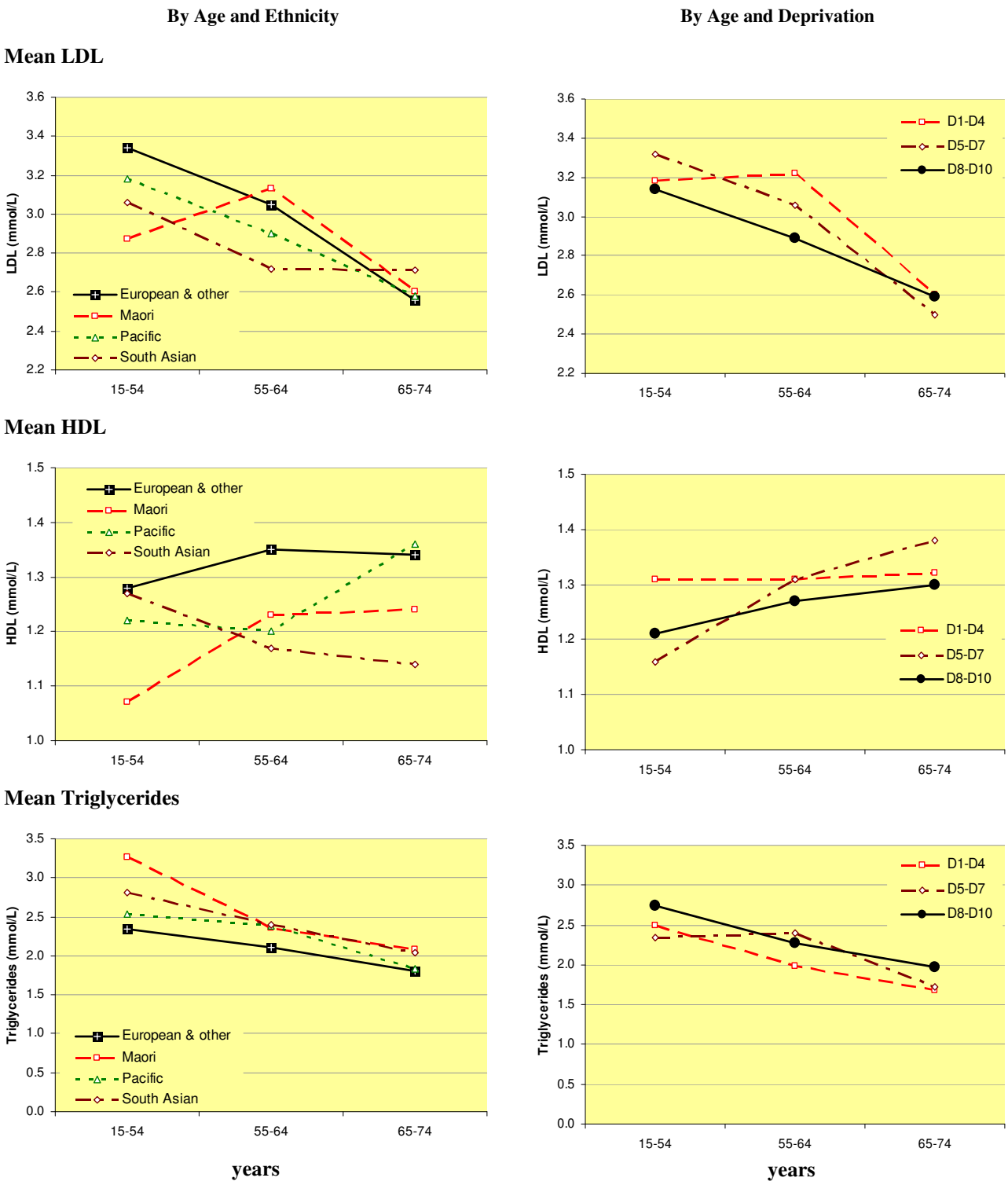


Table 2. Demographics and failure to meet clinical targets, by ethnicity

	All		Ethnicity group				P value
			European*	NZ Māori	Pacific	South Asian	
	N=973 100%		n=598 (61.5%)	n=127 (13.1%)	n=148 (15.2%)	n=100 (10.3%)	
	N	%	%	%	%	%	
Demographic							
Mean (± SD) age, years	[59.9 ± 11.9]		[63.4]	[52.7]	[54.1]	[56.1]	**< 0.0001**
Women	241	24.8	25.8	32.3	23.0	12.0	**0.0043**
European & other*	598	61.5	100.0				-
NZ Māori	127	13.1		100.0			-
Pacific	148	15.2			100.0		-
Indian	100	10.3				100.0	-
Deprivation**							
NZ Dep 1/2	129	13.3	18.9	3.1	0.7	11.0	
NZ Dep 3/4	122	12.5	16.9	3.9	2.7	12.0	
NZ Dep 5/6	102	10.5	14.9	7.1	.	4.0	**<0.0001**
NZ Dep 7/8	175	18.0	18.7	19.7	12.2	20.0	
NZ Dep 9/10	434	44.6	29.9	65.4	83.1	49.0	
History and clinical targets							
First admission	644	66.2	66.7	62.2	66.2	68.0	0.77
Type 2 diabetes	239	24.6	18.1	31.5	39.2	33.0	**<0.0001**
Current smoker	262	26.9	20.9	47.2	33.1	28.0	**<0.0001**
Systolic BP >130mmHg	332	34.1	34.3	36.2	35.1	29.0	0.68
HDL <1.0mmol/L	178	18.3	15.7	24.4	19.6	24.0	**0.0425**
LDL >2.5 mmol/L	531	54.6	53.2	54.3	59.5	56.0	0.58
Triglycerides ≥1.7mmol/L	507	52.1	46.2	68.5	55.4	62.0	**<0.0001**
BMI ≥30	407	41.8	32.9	66.9	68.9	23.0	**<0.0001**
Waist >100cm(M) or >90cm(F)	683	70.2	65.4	84.3	85.8	58.0	**<0.0001**

* Includes European and all other specific ethnicity groups other than Māori , Pacific, and South Asian, and also those with missing information

**Not available for 11 patients

Table 3. Demographics and failure to meet clinical targets by NZ Deprivation Index

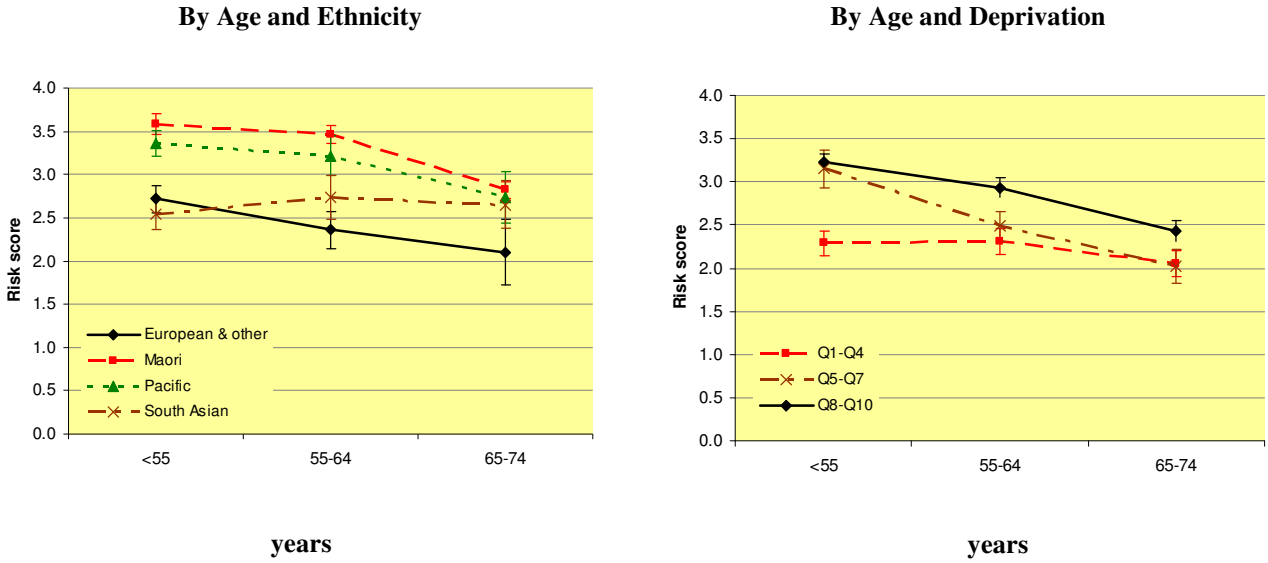
		NZ Deprivation Index decile					
	All	**D 1/2**	**D 3/4**	**D 5/6**	**D 7/8**	**D 9/10**	**P value**
	N=962*	n=129	n=122	n=102	n=175	n=434	
	%	*%*	*%*	*%*	*%*	*%*	
Demographic							
Mean (SD) age, years	[59.9 ± 11.9]	[62.5]	[62.7]	[61.9]	[61.0]	[57.2]	**< 0.0001**
Women	24.8	14.7	27.9	25.5	26.9	26.0	**0.0122**
European & other	61.5	87.6	82.8	87.3	64.0	41.2	
NZ Māori	13.1	3.1	4.1	8.8	14.3	19.1	0.14
Pacific	15.2	0.8	3.3	-	10.3	28.3	
South Asian	10.3	8.5	9.8	3.9	11.4	11.3	
History and clinical targets							
First admission	66.2	70.5	69.7	65.7	58.9	66.4	0.14
Diabetes mellitus	24.6	13.2	24.6	18.6	22.9	29.7	**<0.0001**
Current smoker	26.9	17.1	20.5	23.5	26.3	33.2	**0.0117**
Systolic BP >130mmHg	34.1	39.5	28.7	35.3	33.7	33.6	0.85
HDL <1.0mmol/L	25.8	14.7	25.4	22.6	27.4	29.5	0.23
LDL >2.5 mmol/L	54.6	61.2	51.6	54.9	51.4	54.6	0.08
Triglycerides ≥1.7mmol/L	52.1	36.4	47.5	50.0	51.4	58.5	**0.0003**
BMI ≥30	41.8	22.5	27.0	45.1	40.6	51.8	**<0.0001**
Waist >100cm(M) or >90cm(F)	70.2	58.9	56.6	70.6	69.1	77.9	**<0.0001**

* 11 patients with missing NZ Dep not included

Socioeconomic status according to NZ Deprivation Index (Table 3, Figure 1–2)— A large proportion (44.6%) of the patients lived in areas classified as most deprived (NZDep01 deciles 9 and 10). Patients from the most deprived areas were on average 4 to 5 years younger than those from less deprived deciles. Increasing level of deprivation was associated with smoking, obesity, higher triglyceride levels, and diabetes. Rates of smoking and obesity with deprivation are most pronounced in younger patients. Blood pressure, LDL, and HDL levels did not vary with NZDep01 deciles. Patients from NZDep 1 to 3 were predominently European/other whereas those from the more deprived areas were more ethnically diverse.

Clustering of risk according to age, ethnicity and NZ Deprivation index (Figure 3)—The mean number of poorly controlled CVD risk factors is highest in young Māori and Pacific patients and the young from areas of greater deprivation. Māori and Pacific patients have on average approximately one more poorly controlled risk factor compared with European/other patients across the age range.

Figure 3. The mean risk score for patients by age ethnicity and NZ Deprivation 01. The risk score counts up to 7 poorly controlled CVD risk factors* for each patient



*BMI ≥30 kg/m^2, smoker, Type 2 diabetes, systolic BP >130 mmHg, LDL cholesterol >2.5 mmol/L, HDL cholesterol <1.0 mmol/L and serum triglyceride level ≥1.7 mmol/L

Association of ethnicity with modifiable risk factors in patients under 65 years living in more deprived areas—To investigate the association between ethnicity and risk factors adjusting for deprivation a subgroup analysis was performed. There were insufficient Māori and Pacific patients to compare risk factors across ethnic groups in patients over 65 years or who lived in less deprived areas. This subgroup analysis therefore included only those patients aged less than 65 years and from NZDep01 deciles 9 or 10.

For this analysis patients were grouped into one of three ethnicity groups - Māori, Pacific, and non- Māori /non-Pacific. After adjusting for age, gender, and NZDep Māori were more likely than non- Māori /non-Pacific patients to be current smokers (OR 2.27, 95%CI 1.22–4.20), and there was a trend towards higher mean BMI (mean difference 2.72 kg/m^2, 95%CI 0.90–4.53), more frequent Type 2 diabetes (OR 1.66, 95%CI 0.84–3.25) and higher mean triglyceride levels (mean difference 0.49 mmol/L, 95%CI -0.09–1.07 mmol/L).

Compared with non-Māori/non-Pacific patients the Pacific patients had higher BMI (mean difference 3.60 kg/m^2, 95%CI 1.93–5.27) and more frequent Type 2 diabetes (OR 2.28, 95%CI 1.23–4.21) but similar smoking rates (OR 1.15, 95%CI 0.64–2.04). They were less likely to report a family history of premature CVD (OR=0.44, 95%CI 0.25–0.75).

Prior CVD admissions—For 644 (66.2%) patients the index admission was their first CVD-related admission. Patients with prior admissions had lower mean LDL cholesterol (2.5 vs 3.1 mmol/L, p<0.0001) probably reflecting initiation of lipid lowering therapy in those patients. They were also less likely to be smokers (19.8% vs 30.6%, p=0.001). However, they were just as likely to be obese, and have elevated triglycerides and were more likely to have Type 2 diabetes mellitus (30% vs 21.3%, p=0.0005).

In these patients with previous CVD events, failure to achieve recommended treatment targets was common: 40.4% had LDL levels above 2.5 mmol/L, 19.5% HDL below 1.0 mmol/L (men) or 1.3 mmol/L (women) , 47% had triglycerides above 1.7 mmol/L, 43.2% were classified as obese (defined as BMI≥30 kg/m^2, and 20% smokers.

Discussion

In a cohort of patients presenting to the Coronary Care Unit there are important and systematic differences in the levels of modifiable CVD risk factors according to age, ethnicity, socioeconomic status, and history of prior CVD events. The most important observation was the greater burden of adverse modifiable risk factors in younger patients and particularly among Māori and Pacific people from areas of greater deprivation. These risk factors are a major and reversible contributor to future events in these groups and an important target for secondary prevention programmes.

Age effects—Age shows a strong association with modifiable risk factor incidence, with better levels at increasing age except for blood pressure and diabetes. This inverse association of risk factors with age is to be expected as people with lower exposure to risk factors will take longer to accumulate sufficient exposure to precipitate a coronary event , but some studies suggest a possible cohort effect, with poorer risk factor levels in younger patients than previously.[12]

Some studies suggest that patients presenting prematurely with CVD have a greater genetic predispostion than older patients.[13,14] However, unlike genetic factors, many of CVD risk factors identified particularly in younger people in this cohort can be modified using existing lifestyle and pharmacological approaches.

Despite the improved data in older patients the burden of modifiable risk is still high. Compared with younger patients, older patients were more likely to have hypertension and Type 2 diabetes than other modifiable risks.

Ethnicity and CVD risk factors—The finding that Māori and Pacific people have worse CVD risk profiles than other ethnic groups is concerning given their known excess age adjusted coronary and all-cause mortality rates.[15] Key differences in smoking rates, obesity, and diabetes remained when age, gender, and level of deprivation were controlled. In particular the combination of high smoking rates, obesity and Type 2 diabetes in Māori patients, and of obesity and diabetes in Pacific patients are of concern.

In this study we do not have comparative data regarding the incidence of CVD risk factors in the overall population and the risk distributions observed reflect in part the underlying risk distributions in the population. Nevertheless, these findings are consistent with prior New Zealand primary care data[16] which reported higher rates of smoking, elevated blood pressure levels, lipid levels and prevalence of diabetes in Māori compared with non-Māori patients. Taken together with the known benefits of CVD risk factor modification, these data suggest that aggressive secondary CVD risk factor management within Māori and Pacific groups may have major benefits for those populations.

SES and CVD risk—Socioeconomic status has long been recognised as an important risk factor for cardiovascular diease.[17] This is partly due to documented associations between SES and adverse levels of modifiable cardiovascular risk factors in our study and others.[4] However, two recent studies have found that area based measures of SES had predictive value independent of the traditional Framingham equation risk variables.[18,19] These SES measures may be a proxy for variables such as psychosocial stress, access to health care, and adherence with therapy which are only partly captured by traditional risk factors.

SES, ethnicity, and modifiable risk—Prior New Zealand data has reported that at least half of the ethnic disparity in mortality between Māori and non- Māori is accounted by SES.[15] However, in this cohort using NZDep01 as a measure of SES, there remained very significant differences between Māori and Pacific compared with non-Māori /non-Pacific in modifiable risk factor levels in the the patients resident in more deprived areas.

While this will in part be due to lack of specificity of the deprivation measure (which is based on where an individual lives and not any specific personal measures of SES), other factors not captured by SES measures may vary by ethnicity and may therefore influence modifiable risk factor levels. These could include cultural beliefs and lifestyle behaviors, psychosocial stress, health system access and performance, and medication compliance.

CVD risk factors in patients with known CVD—Of particular concern is the observed gap between observed and recommended risk factor levels in those presenting with a known history of CVD. In these patients CVD risk factors should already be aggressively managed.[7] Whilst it is encouraging that these patients have lower LDL level and smoking rates compared with first presenters, 40% still failed to meet the conservative NZGG LDL target of 2.5 mmol/L. This and the high levels of

obesity and elevated triglyceride levels indicate important gaps in lifestyle and pharmacological management in secondary prevention in our community.

Limitations—As discussed, the measure of socioeconomic status used in this study is an area based measure and does not take into account determinants of socioeconomic status at an individual level. As a result it is possible that the effects of socioeconomic status may have been underestimated in this study.[20]

Misclassification of ethnicity within this study must be considered. A recent report that compared self-identified ethnicity data with hospital record data found concordance for about 90% of New Zealand Europeans, but only 70% of Māori, Pacific, and South Asian people.[21]

The New Zealand Guideline Group (NZGG) definition[7] of metabolic syndrome requires at least three out of five risk factors. In our data, fasting glucose was not available and triglyceride levels were not consistently fasting so we are unable to report prevalence of metabolic syndrome according to NZGG criteria.

Acute PREDICT blood pressure data was collected when patients were lying quietly in hospital, typically on medication started at admission. The relatively good target blood pressure data we report under these conditions probably underestimates the problem of blood pressure control in the community. LDL and triglyceride data were from admission samples and not necessarily fasting. As a result the failure to meet targets for these fractions may have been overestimated. However, this is a random effect and unlikely to bias the ethnic and socioeconomic differences observed.

Just over half of the CCU patients in the study period were entered into Acute PREDICT. The most common reasons for non-inclusion were random factors, particularly time and staff constraints. Although approximately 1 to 2% of CCU admissions die after admission to CCU and are not entered into PREDICT, most other very sick patients although not assessed when acutely unwell were eligible when they had recovered. There were only minor differences between those included and not included for key determinants of risk which included age, gender, ethnicity, socioeconomic status and history of CVD. It is therefore unlikely that the risk factor distribution in the PREDICT group is unrepresentative of the overall CCU group.

Population perspective—Two-thirds of patients in our cohort presented with their first CVD event. Many of these patients had a cluster of potentially modifiable risk factors, emphasising that improved population health initiatives and individualised CVD risk screening and management programmes are needed. But, whilst programs specifically targeted to improve lifestyle risk factors in high risk groups will help to improve cardiovascular risk, ongoing efforts to reduce inequalities and poverty in New Zealand society are also critical.[22]

Several general issues for the primary prevention and management of CVD among Māori and Pacific people also need addressing. These include: systematic differences in the distribution of income, education, employment, and housing that shape Māor/Pacific peoples' exposure to health risks and their access to (and utilisation of) health services,[5] the negative and cumulative effect of cardiovascular risk factors over a lifetime, differential treatment and referral patterns*)*, and the impact of racial discrimination.[23]

Conclusion

In patients presenting to the Coronary Care Unit there is a high burden of modifiable CVD risk factors, smoking, obesity, diabetes mellitus, raised triglycerides, and low HDL particularly in younger patients, and particularly among Māori and Pacific people from areas of greater deprivation. This data can be used to inform the targeting of CVD secondary prevention programmes.

Competing interests: None known.

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