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# A cohort study comparing cardiovascular risk factors in rural Māori, urban Māori and non-Māori communities in New Zealand

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## ABSTRACT

**Objectives:** To understand health disparities in cardiovascular disease (CVD) in the indigenous Māori of New Zealand, diagnosed and undiagnosed CVD risk factors were compared in rural Māori in an area remote from health services with urban Māori and non-Māori in a city well served with health services.

**Design:** Prospective cohort study.

**Setting:** Hauora Manawa is a cohort study of diagnosed and previously undiagnosed CVD, diabetes and risk factors, based on random selection from electoral rolls of the rural Wairoa District and Christchurch City, New Zealand.

**Participants:** Screening clinics were attended by 252 rural Māori, 243 urban Māori and 256 urban non-Māori, aged 20–64 years.

**Main outcome measures:** The study documented personal and family medical history, blood pressure, anthropometrics, fasting lipids, insulin, glucose, HbA1c and urate to identify risk factors in common and those that differ among the three communities.

**Results:** Mean age (SD) was 45.7 (11.5) versus 42.6 (11.2) versus 43.6 (11.5) years in rural Māori, urban Māori and non-Māori, respectively. Age-adjusted rates of diagnosed cardiac disease were not significantly different across the cohorts (7.5% vs 5.8% vs 2.8%, $p=0.073$). However, rural Māori had significantly higher levels of type-2 diabetes (10.7% vs 3.7% vs 2.4%, $p<0.001$), diagnosed hypertension (25.0% vs 14.9% vs 10.7%, $p<0.001$), treated dyslipidaemia (15.7% vs 7.1% vs 2.8%, $p<0.001$), current smoking (42.8% vs 30.5% vs 15.2%, $p<0.001$) and age-adjusted body mass index (30.7 (7.3) vs 29.1 (6.4) vs 26.1 (4.5) kg/m$^2$, $p<0.001$). Similarly high rates of previously undocumented elevated blood pressure (22.2% vs 23.5% vs 17.6%, $p=0.235$) and high cholesterol (42.1% vs 54.3% vs 42.2%, $p=0.008$) were observed across all cohorts.

**Conclusions:** Supporting integrated rural healthcare to provide screening and management of CVD risk factors would reduce health disparities in this indigenous population.

## ARTICLE SUMMARY

### Article focus

- The indigenous Māori of New Zealand have high rates of CVD mortality and morbidity. Current data on the key risk factors underlying this ethnic disparity mostly come from national surveys relying on self-reported diagnoses or have been collected in New Zealand's largest urban centre, Auckland City.
- This is the first study to screen CVD and associated risk factors concurrently between rural Māori residing in an area remote from health services (Wairoa District), urban Māori in a city well served with secondary and tertiary health services (Christchurch City) and an urban non-Māori cohort.

### Key messages

- We found that rural Māori had higher levels of obesity, smoking, hypertension, dyslipidaemia, diabetes mellitus type 2 and hyperuriaemia than either urban Māori or non-Māori. Thus, national health surveys and data collected in large urban centres may significantly underestimate the burden of CVD risk carried by rural Māori.
- Public health initiatives to reduce rates of smoking among rural Māori, along with enhanced implementation of CVD screening in primary care and more intensive clinical management of hypertension, dyslipidaemia and hypeuricaemia would help reduce cardiovascular health disparities in the New Zealand indigenous population.

### Strengths and limitations of this study

- This study was able to determine current levels of both diagnosed and undiagnosed risk factors within these communities by conducting CVD screening clinics in sectors of the community that are often hard to reach. The findings of this study are limited by the relatively small cohort sizes and lack of a rural non-Māori comparator cohort.

## INTRODUCTION

The indigenous Māori, comprising approximately 15% of the New Zealand (NZ) population, have high rates of cardiovascular disease (CVD) mortality and morbidity.[1 2] Life expectancy for Māori is less than the overall population by 8.6 years for men and 7.9 years for women[3] and CVD is the leading cause of death.[1] For the period 1996–1999, CVD mortality among Māori males was 3.0 times higher than that for non-Māori non-Pacific males and Māori female mortality rates were 4.2 times higher than that for non-Māori non-Pacific females.[2] Key risk factors underlying the ethnic disparity in CVD have recently been reported in Auckland City, New Zealand's largest urban centre (population approximately 1.5 million), using data from general practice clinical databases[4] and from community studies.[5–9] However, three-quarters of Māori live outside of Auckland[10] and nearly 16% reside in rural areas. Recent health statistics indicate that ischaemic heart disease rates are higher among those living in rural areas than in urban New Zealanders.[11] Furthermore, rural Māori have an even shorter life expectancy than urban Māori, with 1.5 years difference for men and 1.2 years difference for women,[11] greater than the urban–rural difference in the general population of 0.8 and 0 years for men and women, respectively.[11] Rural non-Māori do not necessarily share the same levels of deprivation as rural Māori. In areas classified as 'Highly Rural/Remote', almost half (46%) of deprivation quintile five residents are Māori.[11] No studies to date have compared cardiovascular health and risk factors between rural and urban Māori communities, and the only previous study of cardiovascular health in rural Māori was conducted 50 years ago.[12]

We have recently reported high levels of both diagnosed and undiagnosed CVD risk factors, especially hypertension, dyslipidaemia and diabetes in a rural Māori community,[13] with several major risk factors at higher levels than those reported than either national statistics or for Auckland City Māori. To understand how CVD risk factors differ between diverse localities across New Zealand, we have compared the cardiovascular risk profiles of two regionally distinct rural and urban Māori communities and a matched urban comparator cohort of non-Māori to identify risk factors in common and those that differ among these communities. This is the first study to screen CVD and associated risk factors concurrently in rural Māori residing in an area remote from health services and in urban Māori in a metropolitan area well served with secondary and tertiary health services.

## METHODS

### Localities

The Hauora Manawa/Community Heart Study is a cohort study of heart disease in NZ, based on random selection from electoral rolls, sampled to be representative of the age and gender profiles within each participating Māori community. The intention was to select two Māori communities as diverse from each other as possible. The Wairoa District is located in rural Hawkes Bay on the North Island of NZ with a population in 2006 of 8481 of whom 61% identified as Māori ethnicity. It is located 2 h north by road of the nearest, fully serviced hospital. Using the Statistics New Zealand definition of rural that uses workplace area meshblocks to allocate a district to one of four categories,[14] based on employment location as the defining variable, Wairoa District is classified in part 'Rural Area with Low Urban Influence' and part 'Highly Rural//Remote Area.' Wairoa is one of three regions in New Zealand where Māori make up over 60% of the local population.[15] The district has associated socioeconomic deprivation and issues of healthcare access. Māori in Wairoa have a median income of $18 500 (compared with a median of $20 900 for all Māori and $24 400 for all of New Zealand).[15] A matched non-Māori sample was not recruited from the small non-Māori population because of differing demographics between the two ethnic groups, especially the younger population profile of Māori. Operating under the umbrella of the Hawkes Bay District Health Board, which also manages the local hospital situated in the city of Hastings, primary healthcare in Wairoa at the time of the study consisted of four general medical practices, all located within Wairoa township and administered by a single Primary Healthcare Organisation. In addition, there were four Māori health providers across the Wairoa District, providing health support services.

In contrast, Christchurch is the largest city in the South Island with a population in 2006 of 348 435, of whom 7.6% identified as Māori and 75.4% identified as New Zealand European. Christchurch City has a large (600–650 bed) tertiary, teaching and research hospital that provides a full range of emergency, acute, elective and outpatient services. Socioeconomic differences between the Māori and non-Māori urban populations were not so marked, with median income levels for Māori being $22 000 versus $23 400 for the Christchurch population overall.[16] Operating under the umbrella of the Canterbury District Health Board, primary healthcare in Christchurch City is administered by two Primary Healthcare Organisations, with approximately 100 member general practices and 10 additional Māori health providers.

### Participants

Full details of sample selection, recruitment, interviewing processes and kaupapa Māori methodology are published elsewhere.[17] In brief, participants, aged between 20 and 64 years, stratified by age and sex were randomly selected from the combined general and Māori electoral rolls for Wairoa District and for Christchurch City (excluding Banks Peninsula Ward, a largely rural area amalgamated with Christchurch City in 2006). The New Zealand electoral rolls have 93.44% enrolment of the eligible population, are updated every 3 years[18]

and were thought to provide the best sampling frame for this study as it allowed selection of targeted age and gender samples of specific ethnic Māori and non-Māori descent. Participants for the Māori samples were selected from among those who identified as being of Māori descent on the roll and who also self-identified at interview as being of Māori ethnicity. Since 1986, self-identification of ethnicity has become the standard practice in New Zealand government, health and research documentation,[19 20] and participants in this study were asked the ethnicity question as in the New Zealand Census. In the census, people can report multiple ethnicities, and anyone listing Māori is counted as Māori under both prioritised and total response analyses. The Christchurch non-Māori descent selection was frequency matched to the sex- and age-group distribution of the Christchurch Māori descent sample. The overall response rates were 57.6% (95% CI 53.0 to 62.2; 254/441) for the Wairoa Māori descent sample, 48.3% (95% CI 44.1 to 52.4; 267/553) for the Christchurch Māori descent sample and 57.2% (95% CI 52.7 to 61.8; 257/449; 254/340) for the Christchurch non-Māori descent sample. The cooperation rates rate (participation among those with whom contact was established) were 74.7% (95% CI 70.1 to 79.3) for the Wairoa Māori, 66.6% (95% CI 62.0 to 71.2; 267/401) for the Christchurch Māori and 71.4% (95% CI 66.7 to 76.1; 257/360) for the Christchurch non-Māori group.

The current study was unique in that it integrated elements of biomedical CVD research design, such as recruitment by random selection (not consistent with Māori cultural processes in which tribal leaders would select participants) within a kaupapa Māori framework. Kaupapa Maori research provides a structure in which Māori worldview is the norm.[21] The following aspects of kaupapa Māori research were incorporated in the Hauora Manawa Study: the project was Māori led and controlled (lead investigator was Suzanne Pitama, Ngati Kahungunu ki Wairoa); the research objectives were based on Māori values, beliefs and experiences and were validated by the Māori communities who were the focus of the study; the research had to demonstrate benefit to the community; joint ownership of the data/information was negotiated between researchers and participants/communities; the research team was accountable to the communities from which the information was gathered and appropriate analysis was employed to minimise dominant cultural biases.

The study was approved by the multi-region ethics committee (reference MEC/06/03026), and all participants provided written informed consent.

## Screening clinics

Screening clinics were held at Wairoa Hospital (May to December 2007) or at the Māori/Indigenous Health Institute, Christchurch, (January to December 2008), as described previously.[13] In brief, all participants completed an interviewer-administered questionnaire providing demographic, personal and family medical history, current medications, smoking status recorded as current, ex-smoker (quit more than 12 months) or never-smoker, alcohol consumption, physical activity levels (physically active defined as 'at least 30 min of moderate or vigorous physical activity on 5 or more days of the week') and socioeconomic data. Blood pressure was recorded (two measurements, seated, at least 20 min apart), height, weight, waist and hip measurements were taken and body mass index (BMI) was calculated. Body composition analysis was performed by bioimpedance using the Tanita Body Composition Analyser, TBF 310 (Tanita Inc, Tokyo, Japan), and the output automatically classified participants into healthy body fat, overweight body fat and obese body fat ranges according to age and gender. Fasting blood samples were collected and assayed for full blood differential cell counts, plasma lipid profiles, glucose, insulin, creatinine, homocysteine, HbA1c and urate (Wairoa Hospital Laboratory, Hawkes Bay District Health Board or Canterbury Health Laboratories, Christchurch Hospital).

A prior cardiac history was defined as a history of diagnosed myocardial infarction, angina, heart failure, coronary artery bypass graft surgery, percutaneous transluminal coronary angioplasty, pacemaker, other cardiac intervention or ischaemic stroke. Confirmation of prior diagnoses and current management of CVD risk factors were obtained from primary care physicians for all participants. A new diagnosis of hypertension and dyslipidaemia was made at screening in those with levels above the reference ranges with no prior diagnosis and who were not receiving treatment. Hypertension was documented if the average of the two blood pressure readings gave a systolic blood pressure (SBP) >140 mm Hg or diastolic blood pressure >90 mm Hg. Dyslipidaemia was diagnosed if the total cholesterol: high-density lipoprotein cholesterol ratio (HDL:TC) was >4. The Bestpractice electronic decision support software (Bestpractice, 2005–2009, Dunedin, New Zealand) was used to calculate 5-year CVD risk scores according to the New Zealand CVD Risk Guidelines[22] and was also used to classify participants with metabolic syndrome at screening.

## Statistical analysis

All statistical analyses were performed using SPSS V.13.0. Data are expressed as mean (SD) or as percentages. Data collection was complete except for anthropometric data for one rural participant. Logistic regression analyses were used to compare binary outcomes across the three cohorts and univariate analysis for continuous variables, and both unadjusted p values and p values corrected for age and gender are presented. Pairwise comparisons (rural vs urban Māori; urban Māori vs urban non-Māori) were carried out using analysis of variance or $\chi^2$ tests, as appropriate for continuous or discrete variables, respectively. For discrete non-binary variables, overall $\chi^2$ tests on a 3×3 table were used followed by a separate 2×3 $\chi^2$ test for each pairwise comparison. Significance levels were set as $p \leq 0.05$.

## RESULTS

### Baseline characteristics and prior diagnoses

The baseline characteristics of the 751 participants who attended the screening clinics are shown in table 1. Rural Māori participants, at a mean age of 45.7 years, were older than both urban cohorts (42.6 years and 43.6 years). Comparisons of key risk factors contributing to CVD risk across the three cohorts are described below.

Rates of prior cardiac history showed a non-significant trend to be higher in Māori than non-Māori (p=0.073, table 1). Although age of onset of CVD tended to be lower in urban Māori than either of the other groups, the differences among the three cohorts were not significant (51.8±9.8 vs 43.8±5.7 vs 50.2±8.2 years, p=0.539, for rural Māori, urban Māori and urban non-Māori, respectively). A prior diagnosis of rheumatic fever had been made in 2% of Wairoa cohort, 0% of urban Māori and 0.4% of urban non-Māori. Of those with a personal cardiac history, 68% of rural Māori, 100% of urban Māori and 71% of non-Māori reported having first-degree family history of CVD. Overall, including those both with and without a personal cardiac history, the rates of first-degree family history of CVD were 52% rural Māori versus 58% urban Māori versus 55% non-Māori (p=0.401).

A prior diagnosis of hypertension was more common in rural Māori than urban non-Māori (table 1), but not significantly different between the two urban groups, and this was reflected in higher rates of treatment with antihypertensive medications in rural Māori (21.4% vs 11.9% vs 8.6%, p<0.001). Mean SBP and diastolic blood pressure levels at screening were slightly but significantly higher in both Māori groups compared with non-Māori group.

Rates of previously diagnosed treated dyslipidaemia were highest in rural Māori, followed by urban Māori and lowest in non-Māori (table 1, p<0.0001). However, mean values of the key cholesterol variables were above the recommended ranges set by national cardiovascular guidelines in all three cohorts,[22] with urban Māori tending to have the least favourable lipid profiles. Urban Māori had higher low-density lipoprotein (LDL) cholesterol levels than rural Māori although mean levels of TC, HDL cholesterol, triglycerides and TC:HDL ratios did not differ between the two Māori communities. Urban Māori also had higher triglycerides and lower HDL cholesterol levels compared with non-Māori, while mean levels of TC, LDL cholesterol and TC:HDL ratios were similar between the two urban cohorts.

Plasma urate has been reported to be an independent risk factor for CVD.[23] Plasma urate levels were significantly higher in rural Māori than urban Māori, which were also higher than in non-Māori (table 1). Urate levels were above the reference ranges (0.42 mmol/l in men and 0.37 mmol/l in women) in 25% of rural Māori, 10% of urban Māori and 5% of non-Māori. Previously diagnosed gout was reported by 6% of rural and 4% of urban Māori and 0.4% non-Māori. Of those, 56% of

**Table 1** Baseline characteristics and cardiovascular risk factors in rural Māori, urban Māori and urban non-Māori

| | Rural Māori (N=252) (a) | Urban Māori (N=243) (b) | Non-Māori (N=256) (c) | Overall, unadjusted p (adjusted p) | (a) vs (b), unadjusted p (adjusted p) | (b) vs (c), unadjusted p (adjusted p) | (a) vs (c), unadjusted p (adjusted p) |
|---|---|---|---|---|---|---|---|
| Age | 45.7 (11.5) | 42.6 (11.2) | 43.6 (11.5) | 0.009 | 0.003 | 0.339 | 0.038 |
| Gender, female, % (n) | 59.5 (150) | 54.7 (133) | 51.2 (131) | 0.166 | 0.282 | 0.426 | 0.059 |
| Cardiac history*, % (n) | 7.5 (19) | 5.8 (14) | 2.8 (7) | 0.066 (0.073) | 0.443 (0.942) | 0.101 (0.038) | 0.020 (0.034) |
| Hypertension, % (n) | 25.0 (63) | 14.9 (36) | 10.7 (27) | <0.001 (0.001) | 0.006 (0.115) | 0.157 (0.061) | <0.001 (<0.001) |
| Systolic blood pressure, mm Hg, mean (SD) | 130.8 (18.5) | 128.2 (20.6) | 124.5 (14.8) | <0.001 (<0.001) | 0.112 (0.074) | 0.021 (0.010) | <0.001 (<0.001) |
| Diastolic blood pressure, mm Hg, mean (SD) | 85.3 (11.1) | 86.2 (13.6) | 83.0 (12.1) | 0.011 (0.002) | 0.391 (0.358) | 0.004 (0.002) | 0.039 (0.028) |
| Treated dyslipidaemia, % (n) | 15.7 (39) | 7.1 (17) | 2.8 (7) | <0.001 (<0.001) | 0.004 (0.051) | 0.032 (0.007) | <0.001 (<0.001) |
| TC, mmol/l, mean (SD) | 5.1 (1.1) | 5.2 (1.0) | 5.3 (1.1) | 0.057 (0.032) | 0.112 (0.101) | 0.438 (0.423) | 0.018 (0.015) |
| LDL cholesterol, mmol/l, mean (SD) | 3.2 (0.9) | 3.3 (0.8) | 3.4 (0.9) | 0.017 (0.009) | 0.042 (0.036) | 0.462 (0.449) | 0.006 (0.004) |
| HDL cholesterol, mmol/l, mean (SD) | 1.2 (0.3) | 1.2 (0.3) | 1.3 (0.3) | <0.001 (<0.001) | 0.645 (0.629) | <0.001 (<0.001) | <0.001 (<0.001) |
| Triglycerides, mmol/l, mean (SD) | 1.5 (0.8) | 1.4 (1.1) | 1.2 (0.7) | 0.003 (<0.001) | 0.491 (0.474) | 0.009 (0.007) | 0.001 (0.001) |
| TC:HDL ratio, mean (SD) | 4.2 (1.1) | 4.3 (1.1) | 4.1 (1.3) | 0.221 (0.088) | 0.329 (0.305) | 0.083 (0.069) | 0.484 (0.462) |
| Urate, mmol/l, mean (SD) | 0.338 (0.897) | 0.300 (0.093) | 0.276 (0.085) | <0.001 (<0.001) | <0.001 (<0.001) | 0.003 (0.001) | <0.001 (<0.001) |

Continuous variables expressed as means (SD). Adjusted p = p value adjusted for age and gender.
*Defined as a history of myocardial infarction, angina, heart failure, coronary artery bypass graft surgery, percutaneous transluminal coronary angioplasty, pacemaker, other cardiac intervention or ischaemic stroke.
TC, total cholesterol.

rural Māori, 22% urban Māori and 0% non-Māori had plasma urate levels above the gender-specific thresholds at screening.

### Metabolic risk factors

A prior diagnosis of diabetes mellitus type 2 (DM2) (table 2) was significantly more frequent in rural Māori compared with either urban Māori or non-Māori cohorts. In DM2 patients, 48.1% of rural Māori, 66.7% of urban Māori and 33.3% of non-Māori with DM2 had a first-degree family history of DM2. Overall, including those with and without DM2, rates of first-degree family history of DM2 were 43% rural Māori versus 31% urban Māori versus 21% non-Māori ($p<0.001$). Metabolic syndrome, diagnosed at screening, was significantly more prevalent in both rural and urban Māori than non-Māori (table 2).

A gradient in BMI was seen across the three cohorts (table 2), with both the age-adjusted mean BMI and the proportion with a BMI in the obese range being greatest in rural Māori and lowest in non-Māori. A similar gradient was observed in per cent body fat, with more rural Māori classified in the combined overweight and obese per cent body fat categories and more urban Māori than non-Māori in these categories ($p<0.001$). In contrast, there was a significantly higher mean fat-free mass in both Māori communities compared with non-Māori. The waist:hip ratios were slightly higher in rural Māori than either of the two urban groups ($p=0.005$ to $p<0.001$). The mean levels of the diabetes risk markers (fasting glucose, HbA1c and fasting insulin) were significantly higher in rural Māori than urban Māori, remaining significant after adjusting for gender and age ($p<0.001$), with HbA1c and insulin also higher in urban Māori than non-Māori. Inclusion of BMI into the univariate model accounted for cohort differences in fasting insulin levels and fasting glucose. However, HbA1c levels, even when adjusted for age, gender and BMI, remained significantly higher in rural Māori than urban Māori ($p<0.001$) and higher in urban Māori than non-Māori ($p<0.001$).

### Lifestyle risk factors

Rural Māori had the highest rate of current smoking (table 2). In both Māori cohorts, more women were current smokers and more men were ex-smokers; however, there was a trend for men to have a higher calculated number of pack-years than women across all three cohorts. Current smokers were of similar mean age ($42.0\pm10.5$ vs $41.0\pm10.7$ vs $40.7\pm12.5$ years, $p=0.754$).

Alcohol drinking was significantly less common in rural Māori than either urban Māori or non-Māori ($p=0.002$), but the two urban communities were similar in their proportions of alcohol drinkers. The mean age of current drinkers tended to be similar in all three cohorts (rural Māori $44.5\pm11.4$ vs urban Māori $42.4\pm11.2$ years; non-Māori $43.1\pm11.4$ years, $p=0.153$). Recommended sessional drinking limits (no more than six standard drinks per session) were reported to be exceeded daily by 2% rural Māori, 3% urban Māori and 1% non-Māori, weekly by 26% rural Māori, 21% urban Māori and 11% urban non-Māori and monthly or less by 55% rural Māori, 44% urban Māori and 48% non-Māori drinkers.

Physical activity within recommended guidelines was reported by similar proportions of rural Māori, urban Māori and non-Māori (table 2). Physical activity was not independently associated with any other cardiovascular risk factors.

Almost all participants were registered with a general practitioner at the time of screening. Similar proportions of each cohort had visited their doctor within the last 6 months (69% vs 61% vs 66%) or had visited their doctor within the last 12 months (85% vs 74% vs 79%).

### Levels of previously undiagnosed hypertension and dyslipidaemia

At screening, approximately 20% of each community were found to have blood pressures above the recommended range (undiagnosed hypertension, table 3). Overall, the combined rates of diagnosed and undiagnosed hypertension across the three cohorts were not significantly different between the two Māori cohorts when adjusted for age but higher in urban Māori than in non-Māori.

For rural Māori, key risk factors in a logistic regression model for predicting hypertension were gender × age (OR=1.243 (95% CI 1.061 to 1.155), $p<0.0001$), BMI (OR=1.107 (95% CI 1.033 to 1.135), $p<0.001$), current smoker (OR=0.461 (95% CI 0.213 to 0.995), $p=0.049$) and family history of hypertension (OR=2.042 (95% CI 1.178 to 3.540), $p=0.011$). For urban Māori, key risk factors for predicting hypertension were gender × age (OR=1.257 (95% CI 1.106 to 1.430), $p<0.0001$), an obese % body fat, (OR=4.394 (95% CI 2.168 to 8.907), $p<0.0001$) and family history of hypertension (OR=1.972 (95% CI 1.074 to 3.619), $p=0.029$). Among non-Māori, the interaction of gender × fasting triglyceride levels (OR=1.886 (95% CI 1.366 to 2.603), $p<0.0001$) and age (OR=1.512 (95% CI 1.161 to 1.968), $p=0.002$) were the risk factors significantly associated with having hypertension.

New diagnoses of dyslipidaemia made at screening (undiagnosed dyslipidaemia, table 3) were significantly more common in urban Māori after accounting for age than in either rural Māori or non-Māori. Overall, when diagnosed and undiagnosed dyslipidaemia were combined, over half of each Māori cohort and 45% of non-Māori cohort were dyslipidaemic.

For rural Māori, the only independent risk factor identified in a logistic regression model for predicting dyslipidaemia was waist circumference (OR=1.027 (95% CI 1.009 to 1.044), $p=0.003$). In urban Māori, dyslipidaemia was predicted by an interaction of gender and BMI (OR=0.960 (95% CI 0.94 to 0.98, $p<0.0001$, male gender being protective) and an inverse effect of first-degree family history of high cholesterol (OR=0.55 (95% CI 0.32 to 0.96), $p=0.034$). The interaction of

**Table 2** Diabetes, anthropometrics, smoking, alcohol and physical activity in rural Māori, urban Māori and urban non-Māori

| | Rural Māori (N=252) (a) | Urban Māori (N=243) (b) | Non-Māori (N=256) (c) | Overall, unadjusted p (adjusted p) | (a) vs (b), unadjusted p (adjusted p) | (b) vs (c), unadjusted p (adjusted p) | (a) vs (c), unadjusted p (adjusted p) |
|---|---|---|---|---|---|---|---|
| Type 2 diabetes, % (n) | 10.7 (27) | 3.7 (9) | 2.4 (6) | <0.001 (0.001) | 0.004 (0.024) | 0.382 (0.279) | 0.001 (0.001) |
| Metabolic syndrome, % (n) | 35.9 (90) | 27.1 (65) | 14.5 (37) | <0.001 (<0.001) | 0.037 (0.139) | 0.001 (<0.001) | <0.001 (<0.001) |
| BMI, mean (SD) | 30.7 (7.3) | 29.1 (6.4) | 26.1 (4.5) | <0.001 (<0.001) | 0.004 (0.003) | <0.001 (<0.001) | <0.001 (<0.001) |
| BMI categories | | | | | | | |
| Healthy BMI, % (n) | 21.9 (55) | 29.2 (71) | 45.7 (117) | <0.001 | 0.079 | <0.001 | <0.001 |
| Overweight, % (n) | 30.3 (76) | 32.1 (78) | 38.3 (98) | | | | |
| Obese BMI, % (n) | 47.8 (120) | 38.7 (94) | 16.0 (41) | | | | |
| Per cent body fat, mean (SD) | 35.0 (10.7) | 32.2 (9.4) | 28.7 (9.0) | <0.001 (<0.001) | 0.001 (<0.001) | <0.001 (<0.001) | <0.001 (<0.001) |
| Per cent body fat categories | | | | | | | |
| Healthy body fat, % (n) | 20.4 (50) | 30.5 (73) | 41.0 (105) | <0.001 | 0.066 | <0.001 | <0.001 |
| Overweight body fat, % (n) | 25.7 (63) | 23.4 (56) | 31.6 (81) | | | | |
| Obese body fat, % (n) | 51.4 (126) | 44.8 (107) | 25.0 (64) | | | | |
| Fat-free mass, kg, mean (SD) | 55.2 (11.2) | 55.8 (12.3) | 53.8 (10.5) | 0.114 (<0.001) | 0.534 (0.285) | 0.043 (0.001) | 0.159 (0.016) |
| Waist:hip ratio, mean (SD) | 0.91 (0.08) | 0.90 (0.10) | 0.89 (0.08) | 0.012 (<0.001) | 0.034 (0.005) | 0.479 (0.351) | 0.004 (<0.001) |
| Glucose, mmol/l, mean (SD) | 5.7 (1.7) | 5.4 (0.9) | 5.3 (0.5) | <0.001 (<0.001) | 0.003 (0.002) | 0.203 (0.186) | <0.001 (<0.001) |
| HbA1c, %, mean (SD) | 6.3 (1.2) | 5.4 (0.7) | 5.2 (0.4) | <0.001 (<0.001) | <0.001 (<0.001) | 0.002 (0.001) | <0.001 (<0.001) |
| Insulin, pmol/l, mean (SD) | 65.6 (56.9) | 52.7 (54.5) | 42.7 (34.4) | <0.001 (<0.001) | 0.005 (0.005) | 0.025 (0.024) | <0.001 (<0.001) |
| Smoking | | | | | | | |
| Current, % (n) | 42.8 (108) | 30.5 (74) | 15.2 (39) | <0.001 | <0.001 | <0.001 | <0.001 |
| Ex-smoker, % (n) | 38.5 (97) | 33.3 (81) | 29.3 (75) | | | | |
| Never smoked, % (n) | 19.0 (48) | 36.2 (88) | 55.5 (142) | | | | |
| Cigarette pack years, mean (SD) | 5737 (4910) | 5469 (5429) | 4308 (5760) | 0.045 (0.058) | 0.615 (0.588) | 0.060 (0.042) | 0.015 (0.009) |
| Alcohol drinkers, % (n) | 81.3 (205) | 92.2 (224) | 91.4 (234) | <0.001 (0.002) | 0.001 (0.003) | 0.753 (0.808) | 0.001 (0.005) |
| Physically active, % (n) | 68.3 (172) | 70.8 (172) | 64.8 (166) | 0.362 (0.316) | 0.157 (0.380) | 0.542 (0.132) | 0.416 (0.380) |

Continuous variables expressed as means (SD). Adjusted p = p value adjusted for age and gender. Ranges for BMI were healthy range, BMI≥18.5<25.0 kg/m$^2$; overweight, BMI 25.0–29.9 kg/m$^2$; obese ≥30.0 kg/m$^2$. Healthy, overweight and obese body fat ranges calculated using Tanita Body Composition Analyser, TBF 310.
BMI, body mass index.

**Table 3** Prior and new diagnoses of cardiovascular risk factors in rural Māori, urban Māori and urban non-Māori

| | Rural Māori (a) | Urban Māori (b) | Non-Māori (c) | Overall, unadjusted p (adjusted p) | (a) vs (b), unadjusted p (adjusted p) | (b) vs (c), unadjusted p (adjusted p) | (a) vs (c), unadjusted p (adjusted p) |
|---|---|---|---|---|---|---|---|
| Hypertension | | | | | | | |
| Prior diagnosis hypertension, % | 25.0 | 14.8 | 10.5 | <0.001 (0.001) | 0.006 (0.115) | 0.157 (0.061) | <0.001 (<0.001) |
| Undiagnosed hypertension, % | 22.2 | 23.5 | 17.6 | 0.235 (0.124) | 0.744 (0.744) | 0.105 (0.054) | 0.191 (0.109) |
| Total hypertension, % | 47.2 | 38.3 | 28.1 | <0.001 (<0.001) | 0.045 (0.236) | 0.016 (0.002) | <0.001 (<0.001) |
| Dyslipidaemia | | | | | | | |
| Prior diagnosis dyslipidaemia, % | 15.7 | 7.1 | 2.8 | <0.001 (<0.001) | 0.004 (0.051) | 0.032 (0.007) | <0.001 (<0.001) |
| Undiagnosed dyslipidaemia, % | 42.6 | 55.0 | 42.4 | 0.008 (0.004) | 0.005 (0.014) | 0.006 (0.002) | 0.961 (0.570) |
| Total dyslipidaemia, % | 58.2 | 62.2 | 45.6 | 0.001 (<0.001) | 0.373 (0.261) | <0.001 (<0.001) | 0.005 (<0.001) |

Undiagnosed = no prior diagnoses or receiving treatment; total diagnoses = potential + previous diagnosis; adjusted p value = adjusted for age.

gender × BMI was also identified in urban non-Māori, associated with a protective effect in men (OR=0.95 (95% CI 0.93 to 0.97), p<0.0001).

### Five-year CVD risk scores

The calculated 5-year CVD risk scores, as published in the New Zealand CVD Risk Guidelines[22] and stratified by age range and gender, are shown in table 4. These scores estimate the likelihood of having a CVD event (myocardial infarction, new angina, ischaemic stroke, transient ischaemic attack, peripheral vascular disease, congestive heart failure and cardiovascular-related death) within the next 5 years and are based on an algorithm including age, gender, SBP, TC:HDL ratio, current smoker or not and presence or absence of diabetes. In addition, the guidelines recommend that scores for certain high-risk population groups, including Māori be moved up one risk category (5% added). Therefore, the two Māori cohorts have an additional 5% making direct comparisons with the non-Māori group difficult. Nonetheless, table 4 shows the expected trend of increasing risk scores across the three cohorts from rural Māori, to urban Māori, to non-Māori in all age and gender categories except in 60–65-year-old males, where urban Māori males have the highest risk.

## DISCUSSION

CVD is the major factor contributing to the ethnic disparities of life expectancy between Māori and non-Māori in NZ, and yet how key risk factors contributing to this discrepancy vary across different localities has not previously been described. To our knowledge, this is the first study to compare current levels of CVD and associated risk factors between two regionally distinct Māori communities: rural Māori residing in an area remote from health services and urban Māori in a city well served with secondary and tertiary health services, as well as non-Māori residing in the same locality. The salient finding was a gradient of cardiovascular risk with the highest burden of CVD risk in rural Māori, particularly obesity, smoking, hypertension, dyslipidaemia, DM2 and hyperuricaemia. Increased measures of body mass were key risk factors associated with the elevated fasting insulin and glucose levels, diagnosed hypertension and dyslipidaemia in both Māori cohorts, but particularly in rural Māori. However, we also observed that

**Table 4** Mean 5-year CVD risk scores* by age and gender in the rural Maori, urban Maori and urban non-Maori cohorts

| | Rural Māori CVD risk score (n) | | | Urban Māori CVD risk score (n) | | | Urban non-Māori CVD risk score (n) | | |
|---|---|---|---|---|---|---|---|---|---|
| | Males, N=102 | Females, N=150 | All, N=252 | Males, N=110 | Females, N=133 | All, N=243 | Males, N=125 | Females, N=131 | All, N=256 |
| Age range (years), % | | | | | | | | | |
| 20–29 | 5.4 (10) | 5.1 (20) | 5.2 (30) | 5.1 (15) | 5.0 (22) | 5.1 (37) | 1.3 (16) | 0.5 (19) | 0.9 (35) |
| 30–39 | 6.6 (23) | 6.1 (33) | 6.3 (56) | 6.7 (32) | 5.3 (34) | 6.0 (66) | 3.9 (32) | 2.6 (38) | 3.2 (70) |
| 40–49 | 9.9 (22) | 8.0 (41) | 8.6 (63) | 9.0 (33) | 7.2 (42) | 7.9 (75) | 6.0 (36) | 4.5 (37) | 5.2 (73) |
| 50–59 | 15.0 (32) | 10.8 (43) | 12.6 (75) | 14.4 (21) | 10.8 (22) | 12.6 (43) | 10.9 (28) | 6.4 (24) | 8.8 (52) |
| 60–65 | 14.6 (15) | 16.7 (13) | 15.6 (28) | 22.2 (9) | 14.5 (13) | 17.6 (22) | 16.1 (13) | 10.1 (13) | 13.1 (26) |

*Calculated by BestPractice© software according to the New Zealand CVD Risk Guidelines[22]—note that all those of Māori ethnicity have score adjusted upwards by 5% in the NZ CVD Risk Scores.
CVD, cardiovascular disease.

undiagnosed hypertension and dyslipidaemia were common across all three New Zealand population groups, despite the establishment of nationwide CVD screening guidelines.

### Findings in relation to national statistics

The 2006/2007 New Zealand Health Survey documented self-reported health information across the New Zealand population and included analysis by ethnicity but not by locality.[24] National prevalence rates were comparable to levels of CVD risk factors we observed in urban Māori but would substantially underestimate CVD risk in rural Māori. For example, 13.6% of the national population reported having medicated high blood pressure,[24] similar to the 14.9% in our urban Māori but less than the 25% in our rural Māori participants. Nationwide, one in 12 adults (8.4%) had medicated high cholesterol, comparable to the 7.1% in urban Māori in the current study but less than the 15.7% in rural Māori. The national prevalence of diagnosed DM2 was 5% in the general population[24] and 5.8% in Māori (age adjusted).[24] Rural Māori in the current study had double the reported national rates of diabetes (10.7%) compared with only 3.7% in urban Māori and 2.4% in non-Māori. National rates of smoking remain higher in Māori (42.2%) compared with the general New Zealand population (19.9%).[24] In the current study, 42.8% of rural Māori and 30.5% of urban Māori were smokers compared with 15% of urban non-Māori. In our data, the multivariate model predicting hypertension appears to indicate that smoking exerts a protective effect on hypertension in rural Māori. This apparent smoker's paradox, where current smokers have less hypertension or better CVD outcomes than former or never smokers, has been previously reported in the literature.[25 26] It has been suggested that smoking protects against hypertension only in overweight and obese individuals, but not in normal weight individuals,[25] which may explain why it is more evident in the rural cohort. In the New Zealand Health Survey, only 50.5% of adults report being physically active.[24] In the current study, around 70% of each cohort reported being physically active, consistent with national data reporting that Māori are at least as physically active as any other ethnic group in New Zealand.[24] It is apparent that the large BMIs and metabolic risk factors observed among the Māori participants were not associated with a less active lifestyle.

Plasma urate levels, an independent predictor of CVD,[23] were above reference ranges in 25% of rural Māori and 10% of urban Māori. The national prevalence of gout is 1.3%, substantially lower than in the current study, with 6% of rural Māori and 4% of urban Māori having been diagnosed by a doctor with gout. It has been known for some decades that gout is a common problem in Māori and Pacific Islanders[27–29] as a result of a difference in renal urate handling.[30] Very recently, two genetic variants have been shown to have genome-wide significant association with gout.[31] One of these, a variant form of the renal urate transporter gene, SLC2A9, has been shown to be significantly associated with gout in Māori and Pacific Island patients.[32] Serum urate levels may be an important underlying risk factor in Māori and Pacific people.

The estimated 5-year CVD risk scores within each age and gender category showed a trend for increasing risk from urban non-Māori to urban Māori and then to rural Māori (except for men aged 60–64 years). However, these differences were more subtle than might have been expected, considering the differences among the cohorts of each composite risk factor used in the CVD risk score calculation. For example, rural Māori had higher levels of smoking, hypertension and DM2 than either urban Māori or non-Māori and yet the maximum differences between the cohorts in risk scores were less than the 5% adjustment added to those of Māori ethnicity. This suggests that overlapping risk factors (eg, being Māori, having long-term DM2 and having family history of CVD, in which case 5% adjustment is applied only once) may dampen the CVD risk score, possibly underestimating extremes of risk observed in some individuals. Moreover, obesity, a salient risk factor that differed across the cohorts, is not included in the CVD risk score algorithm. Also, small differences in the age distributions between the rural Māori cohort and the other two groups might limit comparisons of CVD risk scores since the scores may not predict well for younger individuals. The performance of the NZ risk assessment equation in predicting 5-year CVD events, particularly for Māori, is currently undergoing scrutiny and reconsideration in the published literature.[33–35] We propose that the individual risk profiles may be more informative when deciding clinical management for a specific patient.

### Findings in relation to other regional studies

The current community cohort design is comparable with an Auckland study based on cross-sectional sampling, the Diabetes Heart and Health Survey 2002/2003.[5–7] In that study, 37% of Māori and 22% of others had raised blood pressure, with 7% of Māori and 2% of others having previously undetected high blood pressure; more diagnosed but less undiagnosed hypertension than in the current study. Mean TC:HDL cholesterol ratios were 4.32 in Māori and 4.04 in others, comparable to our urban Māori and non-Māori cohorts. In addition, approximately 94% of participants had total cholesterol levels above 4 mmol/l, similar to the current study (90.7% across all three cohorts), an alarming proportion of each cohort with lipid profiles above recommended ranges. These findings suggest that elevated cholesterol is a key risk factor that is widespread in the New Zealand population and could be more intensively screened and managed throughout primary care. The Auckland prevalence of DM2 was 12% in Māori,[9] similar rates to the rural Māori, and 3.9% in Europeans similar to both our urban cohorts. Metabolic syndrome was identified in 32% Auckland Māori and 16% of others.[7] This was less than in our

rural Māori sample (35.9%) but similar to our urban cohorts (27.1% and 14.5%). In combination, these data suggest that levels of several risk factors in rural Wairoa Māori exceed those among Māori living in Auckland, especially smoking, undiagnosed hypertension and metabolic syndrome. This contrasts with prior studies from Thailand[36] and Peru[37] that reported higher metabolic and CVD risk factors in urban dwellers than rural residents. However, in Australia, metabolic risk was higher in rural patients with DM2,[38] as in the current study.

The current findings reflect a previous study of metabolic and cardiovascular health in a rural Māori community of the Tuhoe tribe performed 50 years ago.[12 39] Prior *et al* screened 212 Tuhoe adults living in the isolated Ruatahuna Valley in the Urewera region.[12 39] The study highlighted high rates of coronary heart disease, obesity, high cholesterol, hyperuricaemia, clinical gout and diabetes. It is difficult to compare the specific data with the current study, as many of the indices measured are no longer in use, such as 'relative weight' and the use of skin fold measurements to assess body fat. There was a 5% prevalence of rheumatic heart disease, considerably higher than the 2% of rural Māori in the current study. Rates of smoking were 59% among men and 70% among women, also substantially higher than we observed in the rural Wairoa cohort. However, mean blood pressure and rates of DM2 were similar to current Wairoa residents. From those findings, alongside the current study, it would appear that health issues besetting rural Māori have improved only a little over the past half century.

## Strengths and limitations of the study

This study was able to determine current levels of both diagnosed and undiagnosed risk factors within these communities by conducting CVD screening clinics in sectors of the community that are often hard to reach. Prior reports based on areal surveys (usually reliant on self-reported diagnoses) or clinical databases may substantially underestimate disease rates and risk factors in indigenous population groups who may not have equivalent access to healthcare or CVD screening. The findings of this study are limited by the relatively small cohort sizes, the study design being constrained by the costs of the in-depth CVD screening. Also, the lack of a rural non-Māori comparator cohort is a limiting factor, resulting from the difficulty in achieving a matched non-Māori sample because of the small number of non-Māori in Wairoa and differing demographics between Māori and non-Māori in the District. In addition, there were slight differences in age distributions between the rural Māori sample and the other two cohorts. While most age-dependent measures were able to be age adjusted, the algorithm used to calculate the CVD risk scores includes age as a risk factor and hence could not be age adjusted. The authors recognise that use of two blood pressure measurements in a clinic setting is not a definitive diagnosis of hypertension.

In conclusion, this study reports that compared with either urban Māori or non-Māori, there is a greater burden of CVD risk factors, particularly obesity, hypertension, dyslipidaemia, DM2, hyperuricaemia and smoking in rural Māori, who have remained largely unstudied for the past half-century. Moreover, the findings demonstrate that CVD risk profiles ascertained in national health surveys or in New Zealand's largest metropolitan population do not necessarily reflect other localities; Christchurch Māori appear to have lower risk than Auckland Māori, while Māori living in rural Wairoa have substantially higher risk. Despite two-thirds of all three cohorts reporting visiting their primary care physicians in the past 6–12 months, the study identified similarly high levels of undiagnosed hypertension and elevated cholesterol in each of the population samples. Rural Māori participants reported similar attendance of participants with their GPs, indicating good engagement with primary care and suggesting that higher rates of some risk factors among the rural contingent were not necessarily associated with barriers to accessing primary care. However, public health initiatives to reduce rates of smoking among Māori, along with enhanced implementation of CVD screening in primary care and more support for rural general practices to provide intensive clinical management of hypertension, dyslipidaemia and hypeuricaemia would help reduce the CVD health disparities in the New Zealand indigenous population.

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**Contributors** VAC and SGP were responsible for the concept and study design, grant writing, supervising recruitment and screening clinics and VAC drafted the manuscript. They are the guarantors. EJW was responsible for overall statistical design, random selection process and contributed to data interpretation. AFF assisted at screening clinics, performed statistical analysis and assisted in drafting the manuscript. MWG, PJR, TMH, RND, GAW, MAR and RWT provided clinical expertise for cardiovascular disease screening, performed echocardiography (not reported here), interpretation of clinical data and management of referrals for risk factors. KNT-M coordinated the study, especially communication with participants. IGS was involved in study design and edited drafts of the manuscript. All authors had full access to all of the data (including statistical reports and tables) in the study and can take responsibility for the integrity of the data and the accuracy of the data analysis.

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**Competing interests** None.

**Ethics approval** Ethics approval was provided by New Zealand Multi-Region Ethics Committee (Reference MEC/06/03026).

**Provenance and peer review** Not commissioned; externally peer reviewed.

**Data sharing statement** There is no additional data available.

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**STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cohort studies***

| Section/Topic | Item # | Recommendation | Reported on page # |
|---|---|---|---|
| **Title and abstract** | 1 | (*a*) Indicate the study's design with a commonly used term in the title or the abstract | 1 and 2 |
| | | (*b*) Provide in the abstract an informative and balanced summary of what was done and what was found | 2 |
| **Introduction** | | | |
| Background/rationale | 2 | Explain the scientific background and rationale for the investigation being reported | 4 |
| Objectives | 3 | State specific objectives, including any prespecified hypotheses | 4 |
| **Methods** | | | |
| Study design | 4 | Present key elements of study design early in the paper | 5 |
| Setting | 5 | Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection | 5 and 6 |
| Participants | 6 | (*a*) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up | 6 and 7 |
| | | (*b*) For matched studies, give matching criteria and number of exposed and unexposed | 6 |
| Variables | 7 | Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable | 7 to 9 |
| Data sources/ measurement | 8* | For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group | *8 and 9* |
| Bias | 9 | Describe any efforts to address potential sources of bias | 7 and 9 (decision support software used) |
| Study size | 10 | Explain how the study size was arrived at | 6 (previous Methods publication cited) |
| Quantitative variables | 11 | Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why | 9 |
| Statistical methods | 12 | (*a*) Describe all statistical methods, including those used to control for confounding | 9 |
| | | (*b*) Describe any methods used to examine subgroups and interactions | 9 |
| | | (*c*) Explain how missing data were addressed | 9 |

| | | | |
|---|---|---|---|
| | | (*d*) If applicable, explain how loss to follow-up was addressed | NA |
| | | (*e*) Describe any sensitivity analyses | NA |
| **Results** | | | |
| Participants | 13* | (a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed | 6 (previous Methods publication cited) |
| | | (b) Give reasons for non-participation at each stage | - |
| | | (c) Consider use of a flow diagram | NA |
| Descriptive data | 14* | (a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders | 10 |
| | | (b) Indicate number of participants with missing data for each variable of interest | 9 |
| | | (c) Summarise follow-up time (eg, average and total amount) | NA |
| Outcome data | 15* | Report numbers of outcome events or summary measures over time | NA |
| Main results | 16 | (*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included | 9, 11-21 |
| | | (*b*) Report category boundaries when continuous variables were categorized | 17 |
| | | (*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period | NA |
| Other analyses | 17 | Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses | NA |
| **Discussion** | | | |
| Key results | 18 | Summarise key results with reference to study objectives | 23 |
| **Limitations** | | | |
| Interpretation | 20 | Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence | 27 |
| Generalisability | 21 | Discuss the generalisability (external validity) of the study results | 23-26 |
| **Other information** | | | |
| Funding | 22 | Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based | Uploaded to website |

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

**Note:** An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.