

# Trends in Ethnic Disparities in Stroke Incidence in Auckland, New Zealand, During 1981 to 2003

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**Background and Purpose**—Although geographical variations in stroke rates are well documented, limited data exist on temporal trends in ethnic-specific stroke incidence.

**Methods**—We assessed trends in ethnic-specific stroke rates using standard diagnostic criteria and community-wide surveillance procedures in Auckland, New Zealand (NZ) in 1981 to 1982, 1991 to 1992, and 2002 to 2003. Indirect and direct methods were used to adjust first-ever (incident) and total (attack) rates for changes in the structure of the population and reported with 95% CIs. Ethnicity was self-defined and categorized as “NZ/European,” “Maori,” “Pacific peoples,” and “Asian and other.”

**Results**—Stroke attack (19%; 95% CI, 11% to 26%) and incidence rates (19%; 95% CI, 12% to 24%) declined significantly in NZ/Europeans from 1981 to 1982 to 2002 to 2003. These rates remained high or increased in other ethnic groups, particularly for Pacific peoples in whom stroke attack rates increased by 66% (95% CI; 11% to 225%) over the periods. Some favorable downward trends in vascular risk factors, such as cigarette smoking, were counterbalanced by increasing age, body mass index, and diabetes in certain ethnic groups.

**Conclusions**—Divergent trends in ethnic-specific stroke incidence and attack rates, and of associated risk factors, have occurred in Auckland over recent decades. The findings provide mixed views as to the future burden of stroke in populations undergoing similar lifestyle and structural changes. (*Stroke*. 2006;37:56-62.)

**Key Words:** stroke ■ epidemiology ■ incidence

Stroke is a major noncommunicable disease of increasing global importance. Although much of the disease burden has been described in economically rich populations including certain ethnic minority groups, limited information exists on temporal trends in ethnic disparities in stroke risk and outcome.<sup>1</sup> Stroke mortality, for example, shows similar downwards trends in black and white Americans,<sup>1,2</sup> but such data are complicated by inaccuracies in death certification and population enumeration,<sup>3,4</sup> whereas hospital-based series are prone to referral bias.<sup>5</sup> Population-based surveillance provides the most reliable assessment of the stroke burden and feedback on the success of strategies to modify risk factors, such as reduction of blood pressure, cigarette smoking, and serum cholesterol. In this article, we extend analyses from a series of methodological “ideal” stroke incidence studies<sup>6</sup> in Auckland, New Zealand (NZ),<sup>7-9</sup> which have shown that Maori and Pacific peoples have considerably higher stroke rates than NZ/Europeans.<sup>10</sup> By including data

from a third study conducted in 2002 to 2003, we aimed to determine ethnic-specific trends in stroke incidence and associated risk factors over 20 years.

## Subjects and Methods

The study methods have been described previously.<sup>11</sup> Briefly, 3 Auckland Regional Community Stroke (ARCOS) studies were conducted using prospective population-based registers to ascertain all cases of first-ever (incident) and recurrent stroke events occurring among adults in the “usually resident” population ( $\geq 15$  years of age) of Auckland over the 12-month periods, 1981 to 1982, 1991 to 1992, and 2002 to 2003. Sampling procedures were used in the first 2 studies. The 1981 to 1982 study used a cluster sample of 50% of all registered primary care general practitioners (GPs) in Auckland to identify a representative sample of half of all stroke events in the population (a total of 703 strokes were registered in 680 patients);<sup>7</sup> the 1991 to 1992 study used a register of all stroke events managed in hospital, and a cluster sample of 25% of all GPs to estimate the total number of “nonhospitalized, nonfatal” stroke events (a total of 1803 strokes were registered in 1761 patients (after adjustment for

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**TABLE 1. Characteristics of Stroke Patients in Each Study Period**

Study Period	1981–1982 (n=1360)		1991–1992 (n=1761)		2002–2003 (n=1938)		Test for Trend P Value*
	n	%	n	%	n	%	
Age, y							<0.001
15–64	332	24.4	425	24.1	472	24.4	
65–74	312	22.9	499	28.3	445	23.0	
75–84	556	40.9	592	33.6	637	32.9	
85+	160	11.8	245	13.9	384	19.8	
Sex							0.15
Male	662	48.7	817	46.4	892	46.0	
Ethnicity†							<0.001
NZ/European	1248	91.8	1532	87.0	1431	73.8	
Maori	60	4.4	82	4.7	102	5.3	
Pacific peoples	32	2.4	111	6.3	197	10.2	
Asian and other	20	1.5	36	2.0	162	8.4	

\*P value calculated using Cochran-Armitage trend test; †there are 46 cases with missing ethnicity in the 2002–2003 study.

sampling).<sup>9</sup> The most recent study used case ascertainment procedures involving all GPs in Auckland.

The population of Auckland (≈940 000 people ≥15 years of age; 2001 Census) is one of the most ethnically diverse in the region, comprising NZ/European (in 66%), Maori (9%), Pacific peoples (11%), and “Asian and other” ethnic groups (14%; of which >90% are Asian). Since 1981, an increase in older people (43% increase in people ≥75 years of age) and non-NZ/Europeans has occurred, including a doubling in the proportion of Pacific people and a 10-fold increase in Asian and other groups attributable mainly to migration. The population has been served consistently by 4 large public acute care hospitals, 2 public medical specialist hospitals, 2 main private medical acute care hospitals, and a large number of long-stay residential care facilities over all of the study periods.

In each study period, experienced study nurses undertook face-to-face interviews with patients or, when the patient was deceased or disabled, the partner (or other reliable proxy) as soon as possible after notification of a stroke. A questionnaire, with item definitions consistent across the 3 studies, was used to obtain information regarding demographics, clinical features, management, and risk factors. Ethnicity was defined by self-identification, and grouped according to “NZ/European” (New Zealand Europeans, British, American, Australian, etc), “Maori” (indigenous New Zealanders), “Pacific peoples” (Tongan, Samoan, Niuean, Cook Island Maori, etc), and “Asian and other” (mainly Chinese and Indian with other ethnic groups such as Pakistani, Iraqi, etc). In the 1981 Census, the ethnicity question reflected “ethnic origin” and referred to the blood mixture of races within a person, whereas in the 1991 and 2001 Census, ethnicity was defined as a measure of cultural affiliation as opposed to race or ancestry.

### Statistical Analysis

All original data sets from the 1981 to 1982 and 1991 to 1992 studies were reviewed, reanalyzed, and rates calculated using standardized methods and published Census data.<sup>12–14</sup> All results are weighted according to the sampling design of the studies to represent all strokes in the Auckland population (crude numbers were multiplied by 2 for the 1981 to 1982 study, and nonhospitalized nonfatal cases were weighted by 4 for the 1991 to 1992 study). Descriptive statistics were used to assess trends in the characteristics of stroke patients in ethnic group across the study periods: significance in the distribution of categorical variables was tested using the Cochran-Armitage method and for continuous variables with Kruskal-Wallis nonparametric ANOVA. Crude annual incidence (“first-ever-in-a-lifetime” event) and attack (all events) rates per 100 000, together with 95%

CI, were calculated using Poisson distribution. Indirect standardization was used to determine ethnic-specific temporal trends, rates in 1981 to 1982 and 1991 to 1992 adjusted to the age and sex structure of each ethnic group in the population of Auckland in 2001.<sup>14</sup> Standardized event ratios (SERs) quantified changes in the standardized rates within each ethnic group between the 2 previous studies and the current study.<sup>15</sup> Direct standardization, adjusted rates to the age structure of World Health Organization (WHO) world population,<sup>16</sup> was used to compare rates between ethnic groups.

### Results

Table 1 shows significant changes in the age and ethnic structure of the 3 studies. In particular, a decline in the proportion of NZ/Europeans was associated with nearly 4- and 5-fold increases in patients identified as Pacific and Asian and Other peoples, respectively, over the study periods. Table 2 shows that the average age of stroke patients increased in all ethnic groups except Asian and Other, although this later group was complicated by small numbers. Strokes occurred at younger ages in Maori and Pacific people (on average, 10 to 15 years earlier) compared with NZ/Europeans, and the consistent low proportions of these groups born in NZ reflects the high levels of migration into the country from South Pacific countries. Although the proportion of current smokers declined in all ethnic groups, the frequency remained high in Maori, while increasing body mass index (BMI), and diabetes was seen in all ethnic groups. A history of heart disease was generally stable with an increase in the history of high blood pressure across the study periods in NZ/Europeans.

Significant changes in the patterns of stroke management were evident, with the proportions managed in hospital increasing across the 3 studies for all ethnic groups, with NZ/Europeans having the lowest and Maori the highest levels of hospital admission, in each of the 3 studies. The proportion of patients receiving neuroimaging (computed tomography [CT] or MRI) also increased for all ethnic groups.

Table 3 and Figure 1 show trends in ethnic-specific indirect standardized rates and corresponding SERs. Compared with

**TABLE 2. Demographic Characteristics, Medical History, and Management, by Ethnic Group in Each Study Period**

Study Period	1981–1982		1991–1992		2002–2003		P Value*
	n	%	n	%	n	%	
European	(n=1248)		(n=1532)		(n=1431)‡		
Demographics							
Born in New Zealand	872	68.8	1095	70.5	1022	66.4	0.14
Age, mean ( $\pm$ SD)	72.8	(12.8)	73.5	(12.1)	75.6	(12.5)	<0.01†
Male	608	48.7	692	45.2	652	45.6	0.11
Past medical history							
High blood pressure	632	50.6	802	52.7	783	57.7	<0.01
Myocardial infarction	146	11.7	273	17.9	190	13.5	0.24
Stroke	314	25.2	404	26.4	361	25.2	0.99
Diabetes mellitus	98	7.9	193	12.7	179	12.7	<0.01
Current smoker	330	26.7	330	21.7	162	12.6	<0.01
BMI, mean ( $\pm$ SD)	23.3	(4.1)	23.7	(4.5)	24.7	(5.1)	<0.01†
Management							
Admission to hospital	790	63.3	1088	71.0	1284	89.7	<0.01
Neuroimaging	134	18.9	851	69.5	1236	86.4	<0.01
Maori	(n=60)		(n=82)		(n=102)		
Demographics							
Born in New Zealand	60	100.0	82	100.0	102	100.0	0.86
Age, mean ( $\pm$ SD)	56.7	(14.2)	55.0	(16.1)	60.7	(14.3)	0.09†
Male	28	46.7	40	48.8	43	42.2	0.51
Past medical history							
High blood pressure	38	63.3	41	52.6	63	62.4	0.92
Myocardial infarction	8	13.3	9	11.4	12	11.9	0.81
Stroke	14	23.3	21	25.6	12	11.8	0.04
Diabetes mellitus	20	33.3	19	24.7	35	34.7	0.69
Current smoker	32	53.3	41	50.6	35	38.9	0.07
BMI, mean ( $\pm$ SD)	28.6	(8.9)	28.3	(6.1)	29.8	(6.9)	0.47†
Management							
Admission to hospital	48	80.0	73	89.0	99	97.1	<0.01
Neuroimaging	18	50.0	65	83.3	99	97.1	<0.01
Pacific peoples	(n=32)		(n=111)		(n=197)		
Demographics							
Born in New Zealand	0	0.0	2	1.8	4	2.1	0.48
Age, mean ( $\pm$ SD)	55.8	(9.0)	59.7	(15.0)	64.5	(13.6)	<0.01†
Male	22	68.8	67	60.4	88	44.7	<0.01
Past medical history							
High blood pressure	22	68.8	49	44.9	124	65.6	0.12
Myocardial infarction	2	6.3	3	2.8	15	7.9	0.24
Stroke	2	6.3	25	22.5	54	27.4	0.01
Diabetes mellitus	12	46.2	16	15.1	69	36.1	0.16
Current smoker	12	37.5	31	28.7	23	13.1	<0.01
BMI, mean ( $\pm$ SD)	30.2	(5.5)	29.1	(7.3)	30.8	(7.7)	0.78†
Management							
Admission to hospital	26	81.3	87	78.4	189	95.9	<0.01
Neuroimaging	6	42.9	74	77.9	180	91.4	<0.01

(Continues)

**TABLE 2. (Continued)**

Study Period	1981–1982		1991–1992		2002–2003		P Value*
	n	%	n	%	n	%	
Asian and other	(n=20)		(n=36)		(n=162)		
Demographics							
Born in New Zealand	4	20.0	0	0.0	4	2.6	<0.01
Age, mean (±SD)	72.9	(12.2)	65.6	(13.2)	65.9	(13.9)	0.27†
Male	4	20.0	18	50.0	87	53.7	0.01
Past medical history							
High blood pressure	8	40.0	18	50.0	88	58.7	0.08
Myocardial infarction	0	0.0	3	8.3	17	11.0	0.12
Stroke	0	0.0	6	16.7	36	22.2	0.02
Diabetes mellitus	4	20.0	8	22.9	40	26.1	0.50
Current smoker	0	0.0	9	25.0	15	10.3	0.98
BMI, mean (±SD)	22.9	(2.7)	22.5	(4.5)	25.6	(6.9)	0.04‡
Management							
Admission to hospital	14	70.0	28	77.8	157	97.5	<0.01
Neuroimaging	4	40.0	18	64.3	153	95.0	<0.01

\*P value calculated using Cochran-Armitage trend test; †P value calculated using ANOVA; ‡there are 46 cases with missing ethnicity in the 2002–2003 study.

1981 to 1982, rates declined among NZ/Europeans (incidence 19%; 95% CI, 11% to 26%; attack rates 19%; 95% CI, 12% to 24%) in 2002 to 2003. However, attack rates increased by 66% (95% CI, 11% to 225%) in Pacific peoples over the study period. Although the rates for Asian and Other people are complicated by small numbers, the trend was for a modest decline in high rates over time. Direct standardized rates, shown in Figure 2, also show declines in NZ/Europeans and increasing rates for Maori and Pacific peoples, who have higher age-adjusted rates than NZ/Europeans. Figure 2 also shows increasing divergence in standardized rates between ethnic groups over time, so that the ratio of attack rates in Pacific peoples was almost double that in NZ Europeans (1.93; 95% CI, 1.65 to 2.26) in 2002 to 2003.

Finally, Table 4 shows there were consistent declines in 28-day case fatality rates across all 4 ethnic groups, although

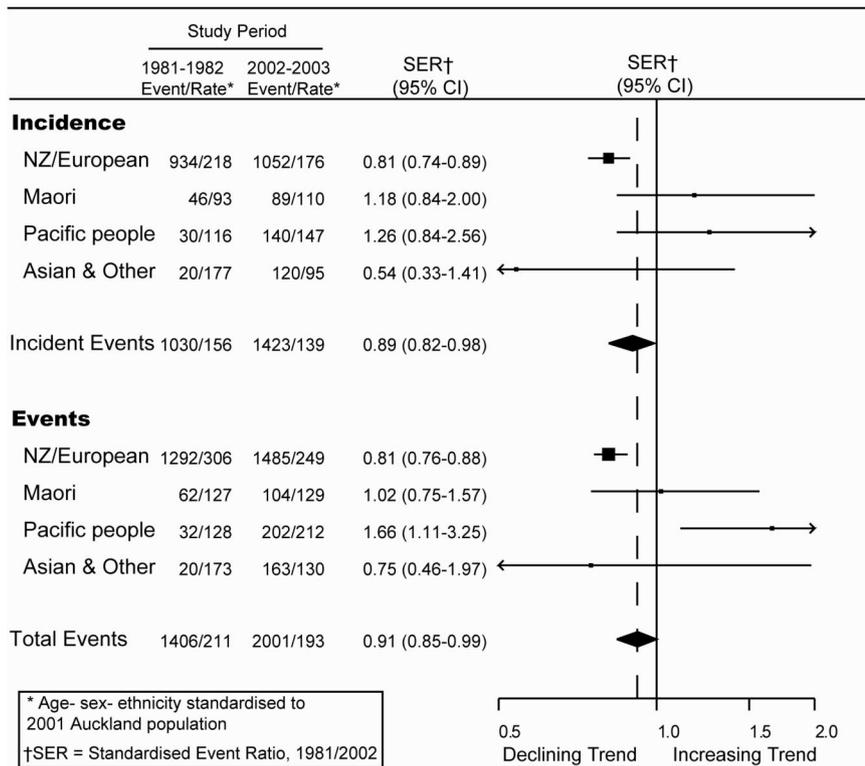
this trend was not significant for Maori, with the largest occurring in Pacific people (55% decline) and Asian and other (70% decline) groups from 1981 to 1982 and 2002 to 2003.

### Discussion

Our study shows encouraging declines in stroke event rates and incidence and in some associated risk exposures among NZ/Europeans in Auckland over 20 years. However, the doubling in event rates in Pacific peoples and ongoing high stroke rates in Maori, together with high frequencies of diabetes and obesity, indicate ongoing and even increasing ethnic disparities in the stroke burden in this population. Because incidence directly reflects levels of absolute disease risk and provides feedback in relation to recent primary and

**TABLE 3. Ethnic-Specific Annual Stroke Rates (per 100 000) Standardized to the 2001 Auckland Population**

	1981–1982			1991–1992			2002–2003		
	n	Rate	(95% CI)	n	Rate	(95% CI)	n	Rate	(95% CI)
NZ/European									
Incidence	934	218	(198–238)	1128	213	(197–229)	1052	176	(166–187)
Total events	1292	306	(282–329)	1572	298	(278–318)	1485	249	(237–262)
Maori									
Incidence	46	93	(55–131)	61	103	(73–134)	89	110	(89–136)
Total events	62	127	(82–171)	82	140	(105–174)	104	129	(106–156)
Pacific peoples									
Incidence	30	116	(57–175)	86	151	(113–189)	140	147	(125–174)
Total events	32	128	(65–191)	113	202	(157–246)	202	212	(185–244)
Asian and other									
Incidence	20	177	(67–287)	30	91	(47–135)	120	95	(80–114)
Total events	20	173	(66–280)	36	110	(63–156)	163	130	(111–151)

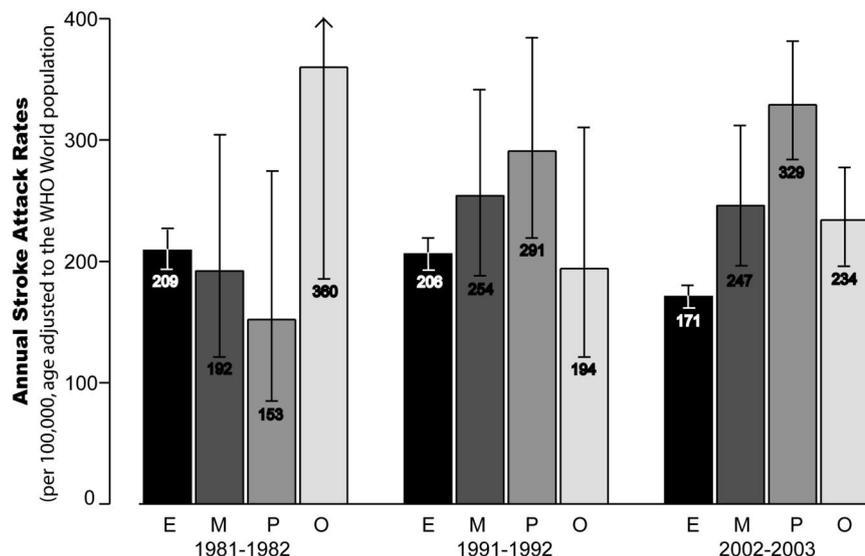


**Figure 1.** Ethnic-specific stroke incidence and attack rates and SERs age-standardized to the 2001 Auckland population.

secondary stroke prevention strategies on a background of structural and lifestyle changes, these complex and divergent trends in ethnic-specific rates and exposures provide mixed views regarding the future burden of stroke, both locally and regionally.

The decline in rates among NZ/Europeans is consistent with data from the more homogeneous but older populations of northern Europe<sup>17-19</sup> and consistent with estimates of a decline in stroke incidence in a predominately white population in Oxfordshire, England, over 20 years.<sup>17</sup> Similarly, the previously recognized higher<sup>10</sup> and now noted to be increasing rates among Maori and Pacific peoples in our population support other data of ethnic disparities in stroke risk, includ-

ing high rates in Hispanics and blacks in the United States<sup>20-22</sup> and black Caribbean people in the United Kingdom.<sup>23</sup> In our study, the near doubling in the difference in direct standardized stroke rates between Maori and Pacific people and NZ/Europeans over the study periods mirrors that seen for stroke incidence in blacks compared with whites in the United States<sup>21,22</sup> and the United Kingdom.<sup>23</sup> Although genetic factors may be one explanation for these disparities, differences in socioeconomic circumstances and exposure to risk factors<sup>24</sup> and access to services that are important to the management of risk factors<sup>25</sup> may also be important. Our findings of higher relative proportions of hospitalization and neuroimaging in Maori and Pacific people is similar to



**Figure 2.** Trends in ethnic-specific stroke attack rates by study period. Rates were age-standardized to the WHO world population and shown with 95% CI. Ethnic groups defined as E = NZ/European, M = Maori, P = Pacific peoples, and O = Asian and other.

**TABLE 4. Ethnic-Specific 28-Day Case Fatality After Stroke by Study Period**

	1981–1982			1991–1992			2002–2003			<i>P</i> Value*
	n	%	95% CI	n	%	95% CI	n	%	95% CI	
NZ/European	408	33	(30–35)	362	24	(22–26)	304	21	(19–23)	<0.001
Maori	18	30	(18–42)	20	24	(15–34)	24	24	(15–32)	0.39
Pacific peoples	14	44	(27–61)	32	29	(21–37)	39	20	(14–25)	0.002
Asian and Other	10	50	(28–72)	7	19	(7–32)	24	15	(9–20)	0.001
Total	450	33	(31–36)	421	24	(22–26)	391	21	(19–23)	<0.001

\**P* value calculated using Cochran-Armitage trend test.

studies including blacks in London.<sup>26</sup> This may reflect less access to diagnostic investigations or management of the illness outside the hospital with more severe strokes in these groups. High rates of stroke in NZ/European and lower rates of hospitalization may reflect less severe strokes in this population. However, 28-day case fatality rates were similar between ethnic groups for the 3 study periods. Increasing stroke rates and no change in case fatality in Maori is contrary to trends in national stroke deaths from 1980 to 1999,<sup>27</sup> thus emphasizing how mortality data may disguise incidence trends. However, this study also found diverging trends in mortality because rates in Europeans declined at a much steeper rate than Maori and Pacific.<sup>27</sup>

The finding of stable proportional frequencies of previous heart disease across ethnic groups suggests that any potential rise in the prevalence with ischemic heart disease is not impacting on stroke rates. The uniform declines seen in the proportion of current smokers may be attributed to success from antismoking campaigns beginning in 1985,<sup>28</sup> although this is less apparent in Maori, in which the proportion remains disproportionately high compared with other ethnic groups. The other notable trend of increasing BMI and history of diabetes in NZ/Europeans, together with the ongoing high frequencies of these exposures in the other ethnic groups, reinforces the growing impact of these risks on cardiovascular disease and other health outcomes. However, history of diabetes was based on self-report and may be subject to bias with underestimates in the rates of diabetes. However, increases in the proportions of NZ/Europeans with a history of high blood pressure are confounded by changes in diagnostic criteria for hypertension and a lowering in the threshold to commence treatment over time, although control of blood pressure in high-risk individuals explains only a small fraction of trends in stroke rates.<sup>29,30</sup>

Monitoring stroke in a population-wide context is challenging,<sup>6,31</sup> not least to ensure that trends are not distorted by changes in diagnostic coding practices and referral patterns. In particular, consideration should be given to addressing confounding because of demographic changes, in particular age, for comparisons of rates between ethnic groups and over time. Our quality control procedures suggest an improvement in the completeness of case ascertainment across the 3 studies, perhaps because of the increased availability of CT and higher hospitalization for stroke.<sup>11</sup> However, we feel that any potential bias in the estimation of rates is likely to be small because of the size of the study population and near consistency of definitions and data acquisition methods

across ethnic groups. Unfortunately, we were unable to look at trends in stroke subtypes because of the low use of neuroimaging in the first study.

Indirect standardization, which adjusted rates from the 1981 to 1982 and 1991 to 1992 studies to the age and sex structure of each ethnic group in the 2001 Auckland population, was used to assess temporal trends in stroke rates within each ethnic group. These rates mirrored the crude rates, high rates in NZ/Europeans, which may appear contrary to other data showing higher stroke rates in blacks and non-Europeans.<sup>2,10</sup> However, these other studies have used the direct method of standardization, adjusting rates to an external standard population. When we used direct standardization, using the WHO standard world population,<sup>16</sup> which is weighted toward younger ages, higher rates in Maori and Pacific peoples compared with NZ/Europeans were seen. By presenting the data using both methods, we demonstrate how ethnic-specific rates (and their interpretation) depend greatly on the method of standardization and choice of reference population.<sup>32</sup>

Another issue to consider is that error in the estimation of rates may have occurred because of misclassification of ethnicity across the studies because of changes in the self-perception of ethnicity, the census definition of ethnicity, or mode of collection of these data.<sup>33</sup> Thus, miscalculation in the numerator and denominator groupings for ethnicity may have arisen in the studies leading to underestimation of rates in Maori and Pacific peoples. However, all Census used a method of prioritization so that people could be categorized into single ethnic groups to allow numerically small groups to be identified from larger groups, and that special priority was given to Maori and Pacific people when multiple responses were given.<sup>27,33</sup> Moreover, because our studies were all undertaken in the same (or next in the most recent) year of each Census, using similar questions of self-defined ethnicity as in the Census, any misclassification of ethnicity is likely to be nondifferential.

In summary, a decline in stroke rates in NZ/Europeans has been offset by markedly increased rates in Maori and Pacific peoples to produce only modest overall declines in stroke in Auckland.<sup>11</sup> Some positive changes in the profile of risk factors, such as declines in the proportions of current smokers with stroke, was counterbalanced by increasing, or ongoing high, frequencies of diabetes and obesity in all ethnic groups. The divergent trends and ongoing ethnic disparities in stroke call attention for urgent policy development and the imple-

mentation of prevention strategies appropriate for all ethnic groups in New Zealand.

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