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Unlocking the numerator-denominator bias. II: adjustments to mortality rates by ethnicity and deprivation during 1991-94. The New Zealand Census-Mortality Study

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Abstract

Aims. Maori and Pacific mortality rates are underestimated due to different recording of ethnicity between mortality and census data – the so-called numerator-denominator bias. Ethnicity and deprivation are strongly associated with mortality in New Zealand, but it is unclear what are the independent and overlapping effects of each on health. The objectives of this study were first, to determine the effect of adjusting for numerator-denominator bias on ethnic-specific age-standardised all-cause mortality rates among 0-74 year olds during 1991-94: second, to determine the effect of adjusting for numerator-denominator bias on analyses of the independent associations of ethnic group and small area deprivation with all-cause mortality in New Zealand.

Methods. Direct standardisation methods were used to calculate rates of mortality by ethnic and small area deprivation groupings.

Results. Unadjusted for numerator-denominator bias, Maori had a 70% and 101% higher standardised mortality rate than non-Maori non-Pacific for males and females, respectively. Adjusting for numerator-denominator bias, the excess Maori mortality burden increased to 126% and 158%. For Pacific people, excess mortality increased from -5% and -13% (ie apparently lower mortality rates) to 58% and 54% after adjustment, for males and females respectively. Using data adjusted for numerator-denominator bias, about a third of the Maori to non-Maori non-Pacific disparity in mortality among 0-54 year olds was explained by small area deprivation. Conversely, about a quarter of the mortality gradient by deprivation in New Zealand was explained by ethnic group.

Conclusions. Numerator-denominator bias causes a marked underestimate of the ethnic disparities in mortality in New Zealand for the 1991-4 period, both overall and within strata of deprivation. The distribution of small area deprivation by ethnicity explains some of the ethnic disparities in mortality.

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There are large health inequalities in New Zealand.[1,2] By ethnic group, Maori have approximately twice the mortality rate of non-Maori.[3] Likewise, males from lower occupational classes (eg labourers) have mortality rates twice that of high occupational classes (eg professionals).[4] Both the reductions of ethnic and socio-economic health inequalities are stated public health priorities in New Zealand.[5,6] Reduction of these inequalities requires an understanding of the independent and overlapping effects of the socio-economic and ethnic determinants of health.[7]

The association of ethnicity and socio-economic status (SES) with mortality is shown in Figure 1. The positioning of the variables indicates that one pathway from ethnicity to mortality risk is via socio-economic determinants of health. It is critical to note that the link between ethnicity and SES (ie putting mortality aside) is not some fixed 'law of society'. Rather, SES is distributed unequally by ethnic group in New Zealand because of, among other things, institutional racism and flow-on effects of colonisation. (Reid and colleagues have described this unequal distribution of SES by ethnicity as the 'distribution gap').[8] The arrow directly from ethnicity to mortality risk is not suggesting some immutable biological/genetic variation of health by ethnicity. Rather, it represents all those other possible pathways not including SES (eg interpersonal racism, health behaviours) that are differentially distributed by ethnicity due to other largely structural factors.

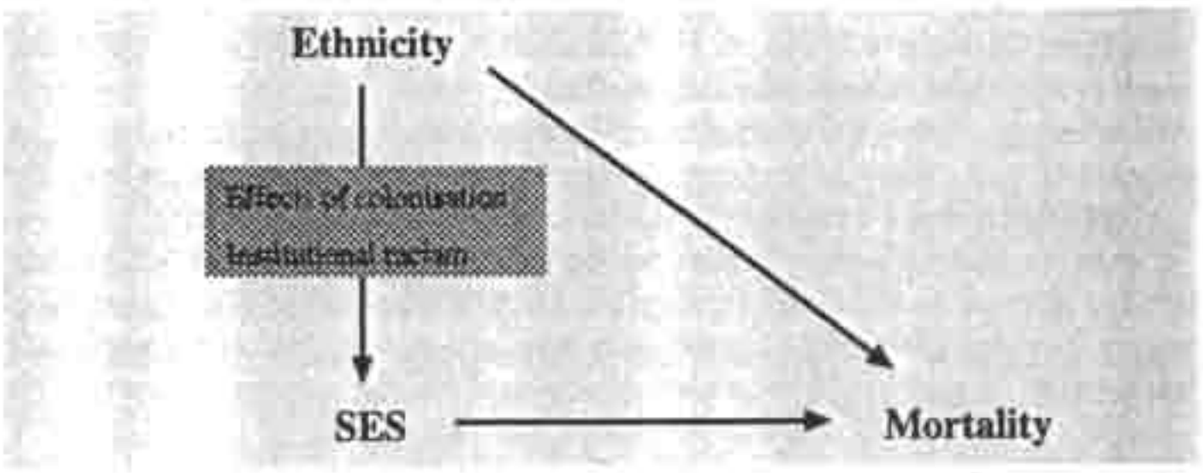

Figure 1. Causal association of ethnicity and socio-economic status (SES).

What does it mean to attribute X% of the difference in mortality between Maori (or Pacific people) and non-Maori non-Pacific to SES? Simply, it means that if the distribution of SES among Maori (and Pacific people) was magically changed to that among non-Maori non-Pacific, then the mortality gap might decrease by the X%. Despite theoretical

limitations and complexities intrinsic in the simultaneous analysis of both ethnic and socio-economic determinants of health,[9-12] such analyses do provide useful information. As argued by Krieger and Davey Smith,[10] it is important to imagine a world where forces such as institutional racism[13] and colonisation had not created the inequitable distribution of SES by ethnic group that we have today.

There is an important potential source of information bias using routine mortality data and census data in New Zealand – the numerator-denominator bias for ethnicity. This bias arises due to ethnicity being recorded differently on mortality data compared to census data, thus biasing ethnic-specific mortality rates. In the preceding paper in this Journal, we quantified this numerator-denominator bias using linked mortality and census records from the New Zealand Census-Mortality Study (NZCMS). Not only were Maori and Pacific deaths severely under-enumerated in the early 1990s compared to the 1991 census, but the magnitude of this numerator-denominator bias varied by age and, possibly, small area deprivation. Therefore, an additional objective of this paper is to demonstrate the effect on ethnic-specific mortality rates and analyses of the overlap between ethnicity and deprivation when correcting for this numerator-denominator bias.

Methods

Mortality (numerator) data. Mortality records for New Zealand residents dying aged 5-74 years from March 6 1991 to March 5 1994, and dying aged 0-4 years from March 6 1991 to March 5 1992, were obtained from New Zealand Health Information Services (NZHIS). (Mortality data for the full three-year period for 0-4 year olds was not required for the larger NZCMS project, and hence were not available for analysis in this paper.) Sex, age and ethnicity data were taken from the National Minimum Data-Set (NMDS), which in turn was sourced from the death registration form completed by the undertaker. The death registration form during the 1991-94 time period only permitted one ethnic group, with three possible values: Maori, Pacific, and non-Maori non-Pacific.

An NZDep91 (1991 New Zealand Index of Deprivation) index score was assigned to the majority of mortality records on the basis of meshblock codes (census administrative regions with a median of 96 individuals) obtained from merging the mortality records with Statistics New Zealand (SNZ) mortality data. The NZDep91 index is a measure of small area deprivation.[14,15] We assigned each mortality record to a decile of deprivation, one being the least deprived and ten the most deprived decile of small areas. For many of the analyses deciles were aggregated; uneven groupings of deciles were used (deciles 1-5, 6-8 and 9-10) due to Maori being more concentrated among the most deprived deciles rendering Maori estimates for a smaller grouping of lesser deprived deciles unstable.

Census (denominator) data. The 1991 census population distribution of 0-74 year olds by sex, age group, sole ethnic group and NZDep91 deciles was used as the denominator in calculations of standardised rates. The use of sole ethnic group has been recommended as the most appropriate denominator for ethnic specific analyses in New Zealand[1] – at least up till 1995 when multiple self-identified ethnic groups were collected on mortality data. To be assigned as sole-Maori meant that the only self-identified ethnicity was Maori, and likewise for sole-Pacific; the remainder were categorised as non-Maori non-Pacific.

Adjustment ratios for numerator-denominator bias of ethnicity. As described in the preceding paper, mortality data dramatically underestimated the number of Maori and Pacific deaths during 1991-4. To correct this underestimation, we calculated adjustment ratios for [sex by age group] and [age group by NZDep91 groupings] (see preceding paper for methods). Due to small numbers, separate ratios by sex were not used for 0-14 and 15-24 year olds Maori and Pacific deaths. Likewise, 0-14 and 15-24 year olds were combined for calculation of the NZDep91 group by age group ratios. The adjustment ratios are available at the NZCMS web-site (http://www.wnmeds.ac.nz/nzcms-info.htm) or directly from the corresponding author.

Standardisation. We used direct age-standardisation[16,17] to calculate the ethnic-specific mortality rates and standardised rate ratios (SRRs; non-Maori non-Pacific as the reference group) for 0-74 year olds during the 1991-94 period. Age-standardisation controls for confounding by the different age-structures of the Maori, Pacific, and non-Maori non-Pacific populations. Five-year age groups were used in the standardisation calculations, and we used the total New Zealand 1991 census population as the external standard. Further, we calculated age and NZDep91 standardised mortality rates and SRRs by ethnic group (Maori and non-Maori non-Pacific only as data for Pacific deaths was too sparse). This allowed an estimation of the percentage of the ethnic inequalities in mortality attributable to deprivation.

Results

38 434 mortality records for New Zealand residents dying aged 5-74 years from March 6 1991 to March 5 1994, and dying aged 0-4 years from March 6 1991 to March 5 1992, were obtained from NZHIS. Of these NZHIS mortality records, 333 mortality records were discarded due to: failing to merge with a mortality record on the SNZ file, or being recorded as a non-New Zealand resident on the SNZ file (the vast majority of the 333), or being linked to two SNZ mortality records. Thus, 38 101 mortality records were available for the age-standardised analyses. A further 4177 of the mortality records were unable to be assigned a NZDep91 score, mainly due to a missing meshblock code on the SNZ mortality file to which they were merged. Thus, a total of 33 924 mortality records (89.0% of all the eligible mortality records) were available for the analyses standardising for both age and NZDep91.

The distribution of deaths by sex by age group by ethnic group is shown in Table 1. The smallest number of deaths was among 0-24 year old Pacific females (n=43). The final column of Table 1 shows the number of deaths that also had a NZDep91 score assigned. Whilst 89.0% of deaths overall had a NZDep91 score assigned, this percentage was notably lower for Maori – particularly 55-74 year old males (73%) and females (77%). These lower percentages are probably due to rural addresses that were difficult to assign a meshblock. Percentages with a NZDep91 score were highest for Pacific people, a predominantly urban population in New Zealand. Conversely, nearly all census records (99%) had an assigned NZDep91 score.

Age standardisation. Age-standardisation mortality rates by ethnic group are shown in Table 2. Unadjusted for numerator-denominator bias, Maori males and females had a 70% and 101% (respectively) higher age-standardised rate of 0-74 year old mortality than non-Maori non-Pacific (SRRs for 'all ages' in Table 2 of 1.70 and 2.01). Following adjustment for numerator-denominator bias, the SRRs increased substantially to 2.26 and 2.58 for males and females, respectively. The SRRs for Pacific people increased markedly following adjustment from 0.95 to 1.58 for males and 0.87 to 1.54 for females. Thus, numerator-denominator bias causes a substantive underestimate of both the Maori and Pacific mortality gap (compared to non-Maori non-Pacific) using routinely published mortality data and census sole ethnicity.

The impact of numerator-denominator bias on the age-standardised mortality rates was most dramatic among 0-24 year olds. Our best estimates were that the Maori SRRs increased from 1.08 to 2.12 form males and from 1.13 to 2.38 for females (Table 2). The impact was similar among 0-24 year old Pacific people.

Age and NZDep91 standardisation. Figure 2 presents the standardised rates of mortality by age group by NZDep91 group, for Maori and non-Maori non-Pacific people, before and after adjustment for numerator-denominator bias. For example, consider the result for 25-54 year old males, unadjusted for numerator-denominator bias. For both Maori and non-Maori non-Pacific, standardised mortality rates are shown for each of three broad NZDep91 groups (deciles 1-5, 6-8, and 9-10) giving a total of six bars. Among both Maori and non-Maori non-Pacific 25-54 year old males, there was a gradient of mortality by deprivation, with mortality increasing with increasing deprivation. Maori males aged 25-54 had a higher mortality than non-Maori non-Pacific within

Table 1. Distribution of deaths for 0-74 year olds (1991-94) by sex, age, NMDS ethnic group and small area deprivation grouping.

		Sex by age (n=38 101)	Sex by age by NZDep91 decile group (n=33 924) Deciles 1-5	Deciles 6-8	Deciles 9-10	All deciles (% of n=38 101)*	
Males							
0-24 yrs	Māori	234	23	54	109	186	(79%)
	Pacific	77	4	26	43	73	(95%)
	non-M non-P	1506	520	412	334	1266	(84%)
25-54 yrs	Māori	692	84	195	296	575	(83%)
	Pacific	129	13	38	75	126	(98%)
	non-M non-P	4233	1599	1164	892	3655	(86%)
55-74 yrs	Māori	1091	117	257	427	801	(73%)
	Pacific	213	24	59	127	210	(99%)
	non-M non-P	15 260	6215	4501	3069	13 785	(90%)
Females							
0-24 yrs	Māori	108	8	21	61	90	(83%)
	Pacific	43	2	14	24	40	(93%)
	non-M non-P	682	233	194	163	590	(87%)
25-54 yrs	Māori	466	57	111	218	386	(83%)
	Pacific	76	10	20	44	74	(97%)
	non-M non-P	2577	1043	680	564	2287	(89%)
55-74 yrs	Māori	845	83	195	375	653	(77%)
	Pacific	124	14	31	77	122	(98%)
	non-M non-P	9745	4018	2988	1999	9005	(92%)

*Percentage of all eligible mortality records that had NZDep91 score.

NZDep91 deciles 1-5 and 6-8, but there was no substantive difference between ethnic groups within the NZDep91 decile 9-10 group (both have standardised rates of about 400 per 100 000). However, this pattern for 25-54 year old males changed dramatically after adjusting for numerator-denominator bias, with a notably increased ethnic disparity within each NZDep91 group. A similar pattern is evident for all other sex and age groups shown in Figure 2.

The adjusted standardised rates shown in Figure 2 are prone to some error and therefore the patterns are more important than the exact rates. Thus, we do not give the exact standardised rates for each sex by age group by ethnic group by NZDep91 group. Shown in Table 3, are the age-standardised mortality rates and SRRs (adjusted for numerator-denominator bias) for those mortality records with a NZDep91 score. (Note that due to fewer Maori deaths being assigned a NZDep91 score than non-Maori non-Pacific deaths (see last column of Table 1), the age-standardised only SRRs given in Table 3 are generally less than those in Table 2 – particularly among 55-74 year olds). Also shown in Table 3 are the age and NZDep91 standardised mortality rates and SRRs. By age group, the percentage reduction in the ethnic disparity due to standardising for NZDep91 was about a third for 0-24 and 25-54 year olds, except among 0-24 year old females where it was 58%. However, this latter finding should be treated with considerable caution due to the small number of Maori 0-24 year old female deaths, particularly in deciles 1-5 (n=8, Table 1). It was not appropriate to calculate an age and NZDep91 standardised mortality rate for 55-74 year old males, as there was a NZDep91 gradient among non-Maori non-Pacific but not among Maori. Among 55-74 year old females, 22% of the ethnic disparity in mortality was estimated to be due to differences in deprivation.

Ethnic group standardisation. We estimated how much of the mortality gradient by deprivation was due to ethnic disparities by standardising further for ethnicity, using data adjusted for numerator-denominator bias. Setting NZDep91 deciles 1-5 as the reference group, standardising by ethnicity reduced the 0-74 year old SRR for the NZDep91 deciles 6-8 group by 23% and 17% and the NZDep91 deciles 9-10 group by 34% and 35%, for males and females respectively.

Discussion

Using routine mortality data for the early 1990s, and 1991 census data (with the recommended 'sole' ethnic groups), and not adjusting for numerator-denominator bias, the excess 0-74 year old mortality among Maori compared to non-Maori non-Pacific was 70% and 101% for males and females, respectively. However, adjusting for numerator-denominator bias, the excess mortality burden among Maori compared to non-Maori non-Pacific increased to 126% and 158%, respectively. Among 0-24 year olds, the impact of adjusting for numerator-denominator bias was even more dramatic, with the Maori to non-Maori non-Pacific mortality gap jumping from (essentially) nil to over 100% excess mortality. Comparing Pacific people to non-Maori non-Pacific, adjusting for numerator-denominator bias reversed a seemingly lower 0-74 year old mortality rate to one over 50% higher among both males and females. Likewise, within strata of deprivation, adjusting for numerator-denominator bias dramatically increased the Maori to non-Maori non-Pacific mortality gap – indeed, often changing an apparently lower mortality rate among Maori to one that was substantially greater.

Adjusted for numerator-denominator bias, 15% (males) and 28% (females) of the Maori to non-Maori non-Pacific 0-74 year old mortality gap was attributable to small area deprivation. This attribution was higher at younger ages, being approximately a third for 0-54 year olds. Failure to adjust for the numerator-denominator bias would have overstated the percentage of the ethnic gap due to deprivation.

How consistent are our findings with previous research in New Zealand? Pearce, Davis and colleagues conducted the major body of comparable research.[18-20] They determined occupational class gradients in mortality for men aged 15-64 years during 1975-77 and 1985-87, and by Maori, non-Maori. They concluded that 19% of the excess rate of mortality between Maori and non-Maori in 1975-77 was

Table 2. Age standardised rates (SR; per 100 000 person years) and standardised rate ratios (SRR; compared to non-Māori non-Pacific) of mortality for 23 435 male and 14 666 female 0-74 year old deaths during 1991-94, by age group and ethnicity. Results are presented both unadjusted and adjusted for numerator-denominator bias.

	Unadjusted for numerator-denominator bias				Adjusted for numerator-denominator bias		
	SR	(95% CI)	SRR	(95% CI)	SR	(% diff SR)*	SRR
Males							
All ages							
Māori	869	(826-912)	1.70	(1.62-1.79)	1104	(27%)	2.26
Pacific	483	(430-537)	0.95	(0.85-1.06)	774	(60%)	1.58
non-M non-P	511	(504-518)	1.00	-	489	(-4%)	
0-24 year olds							
Māori	132	(120-144)	1.08	(0.98-1.19)	226	(71%)	2.12
Pacific	133	(112-153)	1.08	(0.92-1.27)	221	(66%)	2.07
non-M non-P	122	(118-127)	1.00	-	107	(-12%)	
25-44 year olds							
Māori	435	(413-456)	1.84	(1.75-1.94)	588	(35%)	2.68
Pacific	188	(167-210)	0.80	(0.71-0.90)	309	(64%)	1.41
non-M non-P	236	(231-241)	1.00	-	220	(-7%)	
55-74 year olds							
Māori	3994	(3893-4094)	1.75	(1.70-1.79)	4825	(21%)	2.16
Pacific	2211	(2086-2336)	0.97	(0.91-1.02)	3497	(58%)	1.57
non-M non-P	2284	(2270-2299)	1.00	-	2229	(-2%)	
Females							
All ages							
Māori	584	(551-616)	2.01	(1.90-2.13)	715	(22%)	2.58
Pacific	252	(217-286)	0.87	(0.75-1.00)	427	(69%)	1.54
non-M non-P	290	(285-295)	1.00	-	277	(-4%)	1.00
0-24 year olds							
Māori	75	(65-85)	1.13	(0.97-1.30)	135	(80%)	2.38
Pacific	74	(58-90)	1.12	(0.90-1.40)	122	(65%)	2.15
non-M non-P	66	(62-70)	1.00	-	57	(-14%)	1.00
25-44 year olds							
Māori	291	(273-308)	2.06	(1.93-2.20)	374	(29%)	2.87
Pacific	115	(98-132)	0.81	(0.70-0.95)	207	(80%)	1.58
non-M non-P	141	(138-145)	1.00	-	130	(-8%)	1.00
55-74 year olds							
Māori	2722	(2646-2798)	2.12	(2.06-2.18)	3174	(17%)	2.53
Pacific	1091	(1012-1169)	0.85	(0.79-0.91)	1828	(68%)	1.46
non-M non-P	1286	(1276-1296)	1.00	-	1256	(-2%)	1.00

Age standardisation was by 5-year age groups within the stated age range. *Percentage change compared to the unadjusted standardised rate.

Figure 2a. Males

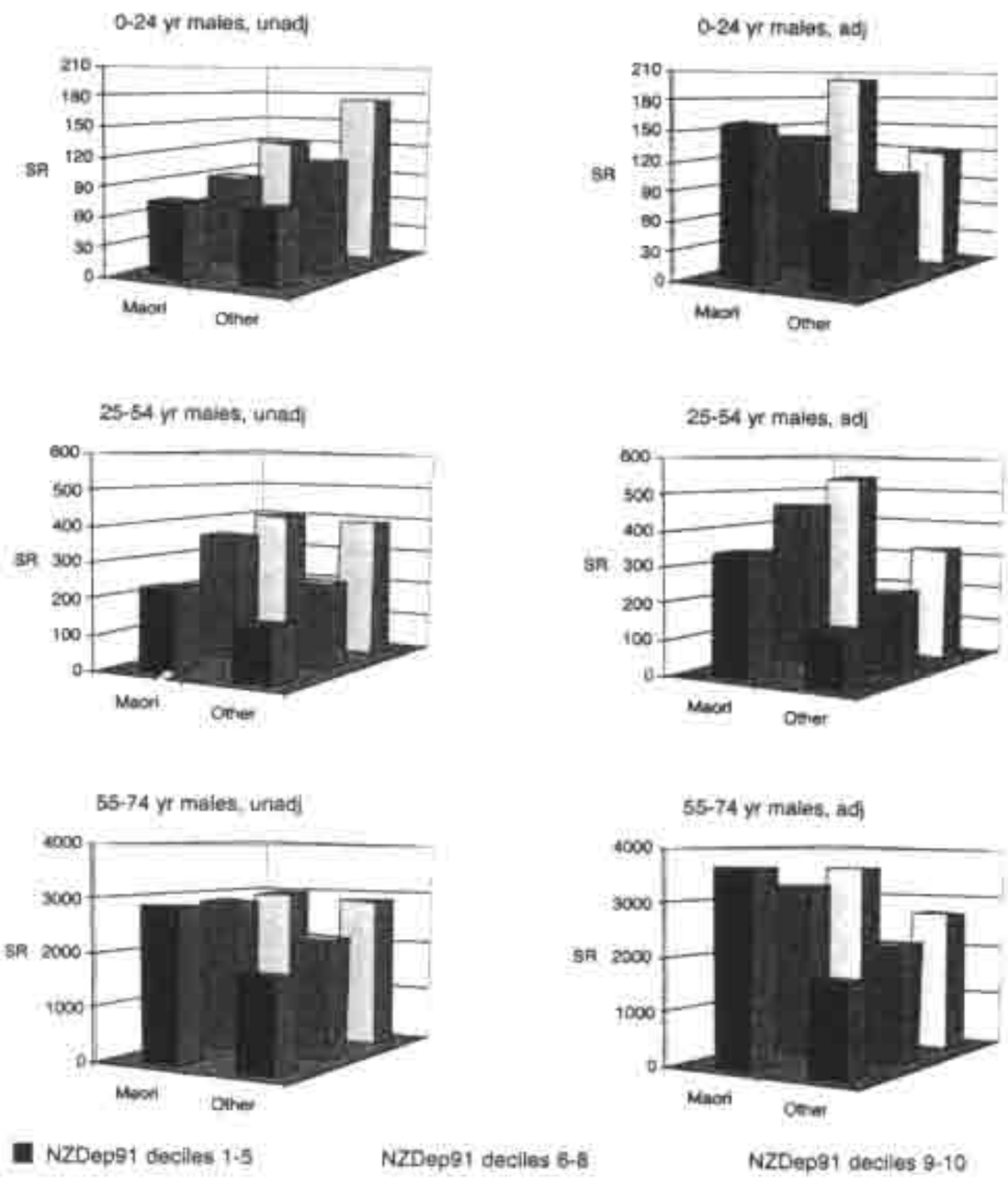

Figure 2b. Females

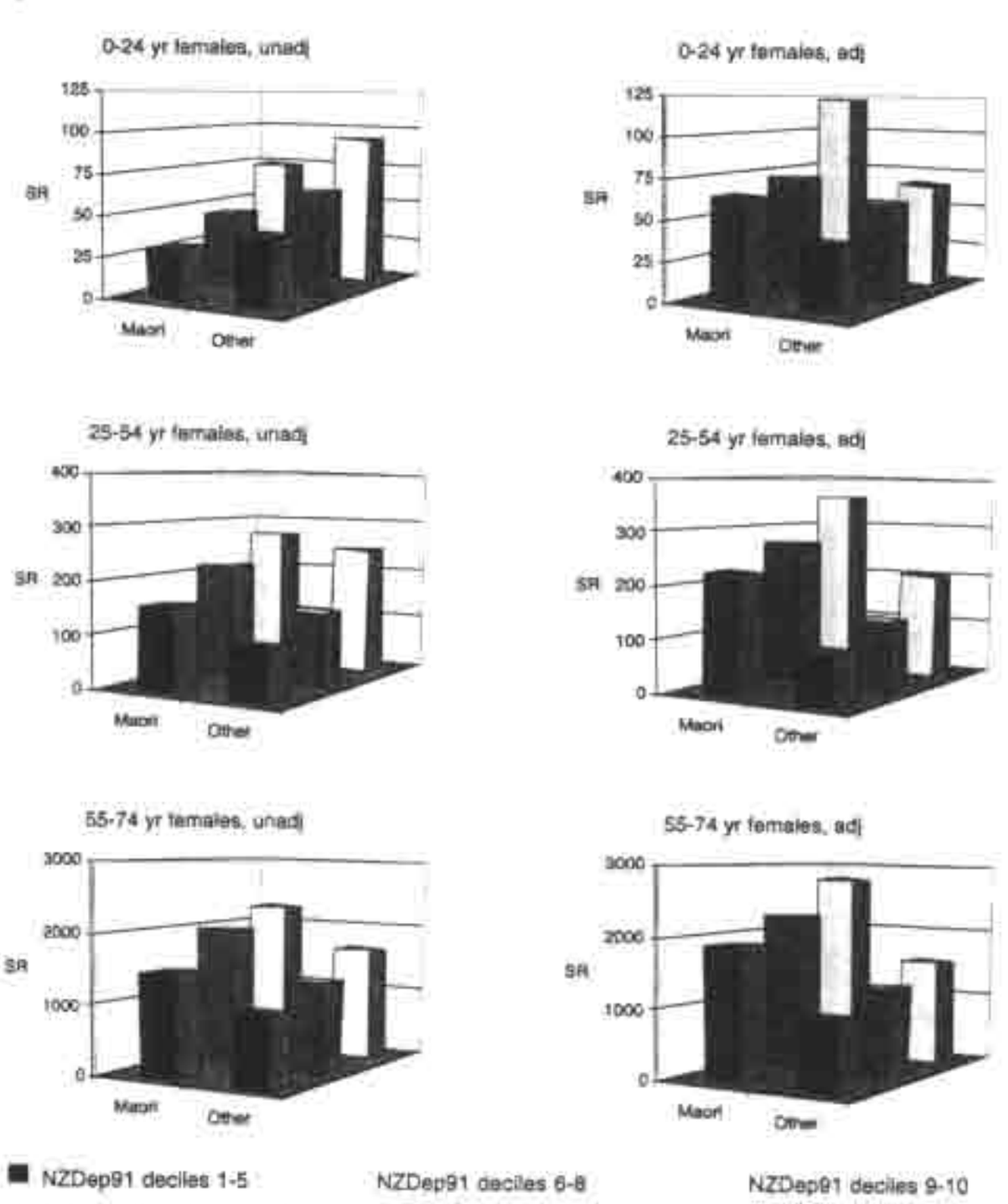

Figure 2. Standardised rates of mortality (per 100 000; deaths during 1991-94) for Māori and Non-Māori non-Pacific by small area deprivation (NZDep91), unadjusted and adjusted for numerator-denominator bias.

Table 3. Standardised rates (per 100 000 person years) and standardised rate ratios (SRR; Mäori compared to non-Mäori non-Pacific) of mortality, within cross-classified strata of sex, age and small area deprivation, for 0-74 year old deaths with a NZDep91 score.

	NMDS Ethnicity	Age-standardised only Standardised rate	SRR	Age and NZDep91 standardised Standardised rate	SRR	% fall SRR*
Males						
All ages	Mäori	838	1.92	808	1.79	15%
	non-M non-P	436	1.00	452	1.00	
0-24 years	Mäori	191	2.11	163	1.74	33%
	non-M non-P	90	1.00	93	1.00	
25-54 years	Mäori	488	2.57	418	2.03	35%
	non-M non-P	190	1.00	206	1.00	
55-74 years	Mäori	3494	1.73	na		
	non-M non-P	2016	1.00	na		
Females						
All ages	Mäori	561	2.22	492	1.88	28%
	non-M non-P	252	1.00	262	1.00	
0-24 years	Mäori	112	2.30	79	1.54	58%
	non-M non-P	49	1.00	51	1.00	
25-54 years	Mäori	310	2.68	268	2.10	34%
	non-M non-P	116	1.00	127	1.00	
55-74 years	Mäori	2429	2.09	2191	1.85	22%
	non-M non-P	1160	1.00	1184	1.00	

All calculations adjust for numerator-denominator bias. Age standardisation was by 5-year age groups within the stated age range. NZDep91 standardisation was by deciles within the stated NZDep91 range*. The percentage decrease between the age-only and age and NZDep91 standardised rate differences for Mäori compared to non-Mäori non-Pacific. For example, for males aged 0-24 years [2.11-1.74] / [2.11-1.0] gives a 33% decrease in the SRR.

attributable to occupational class, and 30% in 1985-97.[18] Despite using a different measure of SES, their results are not inconsistent with ours given the 15-64 year age range – an age range which traverses our three separate age groupings. It is uncertain what effect numerator-denominator bias would have had on the analyses by Pearce and colleagues.

In this paper, we investigated the independent and overlapping associations of ethnicity and small area deprivation with mortality during 1991-94. Thus, we are using one measure of socio-economic status at one point in time – small area deprivation based on the address at time of death (numerator data) and at the 1991 census (denominator data). The advent of measures of small area deprivation in New Zealand has greatly advanced our ability to measure socio-economic inequalities in health.[15,21,22] However, there is a risk that gradients in mortality by deprivation may be mistakenly regarded as the same as gradients by SES more generally. The NZDep91 and 96 indices are measured at the small area-level, and within small areas there will be heterogeneity of individuals' income, education and occupational class – the three 'classic' measures of personal SES.[23-25] Undoubtedly, a greater percentage of the ethnic mortality gap could be attributed to SES if a wider range of socio-economic factors (eg income, education, social class) were controlled for, and for multiple points of the life-course.[7,26] How much, though, is unclear.

Of note in this paper was the tendency for the percentage of the ethnic gap explained by deprivation alone to be less at older ages. This finding is consistent with a range of possible hypotheses, including:

- maybe the non-socio-economic reasons for ethnic inequalities are greater in older cohorts of Maori, compared to younger Maori where inequalities are more a function of socio-economic inequalities. Discrimination or racism that affects individuals directly (eg blood pressure and psychosocial effects) and cumulates over a lifetime is one possible reason;[27]
- maybe deprivation captures much of the socio-economic disparity between Maori and non-Maori in younger age groups, and that other socio-economic factors (eg, income, education) are more important at older ages;
- maybe socio-economic exposures in childhood are more important contributors to ethnic inequalities, and that a much greater proportion of the ethnic inequalities at older age groups would have been explained if deprivation in childhood (not at time of death) had been measured.

The adjustment ratios for numerator-denominator bias in this study will have some inaccuracy. First, as shown in the preceding paper, adjustment ratios were calculated using only a sample of mortality records for the 1991-94 period. Second, random rounding to a near multiple of three required under SNZ confidentiality rules may add further imprecision to the standardised rates. Third, analyses for 0-24 year old Mäori and Pacific people should be treated with caution due to both possible inaccuracies in the adjustment ratios and fewer deaths.

In conclusion, both ethnicity and SES are important determinants of mortality (and more generally health) in New Zealand, and SES 'explains' some (perhaps the majority) of ethnic inequalities. However, it is imperative not to lose sight of why SES is differentially distributed by ethnicity in New Zealand, the most important reasons being power imbalances, institutional racism and the effects of colonisation that have disadvantaged Mäori. Needless to say, these structural inequalities themselves require tackling to reduce ethnic inequalities in health. From another angle, tackling the socio-economic health gradient itself is a priority. Strategies here range from reducing income inequalities to ensuring adequate provision of health care services to socio-economically disadvantaged groups and Mäori.[7,28,29] These tasks are daunting and (often) beyond the reach of the health sector. However, they also present huge opportunities for improving the health status of New Zealanders.

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Evaluation of the BioSign™ PSA membrane test for the identification of semen stains in forensic casework

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Abstract

Aim. To evaluate BioSign™ prostate specific antigen (PSA), a membrane test device used as a clinical aid in the diagnosis of prostate cancer, to determine whether it can be used in forensic laboratories for identifying semen stains.

Methods. Biological fluids were obtained under ethical approval from anonymous consenting donors. BioSign™ PSA was evaluated in terms of its specificity, sensitivity and cost to replace an ELISA (enzyme linked immunosorbent assay) method of PSA detection.

Results. Semen stain extracts and semen diluted 10^5 tested positive with BioSign™ PSA. Animal semen, other human body fluids and commonly encountered household products tested negative. Anomalous results were observed with semen-free condoms containing nonoxynol-9. The cause of these false positive results is not known.

Conclusions. These results and the ease of use of the BioSign™ PSA kit indicate that it is a valuable addition to forensic laboratories and can adequately replace the ELISA method of PSA detection. BioSign™ PSA was not suitable for testing condoms for semen.

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The identification of semen stains is a routine test in forensic laboratories as semen is commonly encountered at crime scenes involving sexual violation. Dried semen stains retain acid phosphatase (ACP) activity for lengthy periods of time, so one screening procedure is to test exhibits for areas of ACP activity.[1] To confirm the presence of semen an extract of the ACP positive area is searched for spermatozoa. If spermatozoa are not detected then another test is required to confirm semen. Human prostate-specific antigen (PSA) has been used in the forensic identification of semen since 1978.[2] One method of detection is an enzyme linked immunosorbent assay (ELISA).[3] Alternative testing systems include commercially available test strips which are designed for the quick and semiquantitative detection of PSA in human serum, and are used as clinical aids in the diagnosis of prostate cancer. One such commercial PSA kit is the BioSign™ PSA kit.[4,5]

The purpose of this study was to determine whether BioSign™ PSA can be used in the routine testing of forensic samples to identify semen stains. The kit was evaluated in terms of its specificity, substrate versatility and cost.

Methods

Biological fluids were obtained under ethical approval from 26 anonymous consenting donors and stored prior to use at -20°C. Stains were made by depositing 10 µl of body fluid onto the substrate and once dried, extracting with 500 µl of sterile distilled water. The testing procedure was performed according to the manufacturer's directions provided in the BioSign™ kit.

Results

Experiments involving dilutions of semen showed that the ELISA method was still the more sensitive of the two methods of PSA, detecting semen diluted 10^7 times. BioSign™ PSA detected semen diluted 10^5 times. Gross inconsistencies were observed with BioSign™ PSA between some neat and small dilutions of semen and the corresponding result line intensities. This anomaly can be explained by the high dose hook effect.[6] These samples were