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EDITORIALS

Which way forward?

Much medical practice is based on teamwork. In practice teamwork depends on trust between colleagues. This is best attained in an environment where good communication and cooperation are facilitated by management. Replacing trust with contracts and accountability, the 'health reforms' of the 1990's emphasised financial management and competition which increased bureaucratic documentation and administrative staff numbers. As a result spending on administration in hospitals increased from 9% to 25% or more, although accurate figures are not available (personal communication, Ministry of Health). As Vote Health is finite this decreased the funds available for direct patient care aggravating the pressure on clinical services.

Whilst trust and goodwill are essential it is equally important to ensure standards of care through appropriate quality control processes. The financial incentives and directives of the reforms did not foster this, but encouraged autocratic decisions aimed to control spending. Plsek and Wilson[1] suggest that the biggest barrier to improvement in complex systems such as health are the incumbent leaders who have risen within the hierarchy based on these command and control methods. In some hospitals a few senior medical staff decided to support the focus of management on financial targets and became "trusted" advisors. Collaboration with those priorities of management appears not to have achieved quality improvement but, rather, led to tragedies brought to light by the Stent, Gisborne Hospital and Cervical Screening Inquiries. These have all demonstrated the need for better attention to independent health professional advice.

The selective health professional advice employed by the Ministry of Health and the Boards, did not prevent these failures. The health professional advisory group appointed by CCMAU did not prevent the imposition of a business plan on Canterbury Health in 1994 that ultimately led to unnecessary deaths. "The drive for efficiency within an unrealistic time-frame, with minimal patient focus … contributed to the under-resourcing of Canterbury Health and a breakdown in the relationships between clinicians and management."[2] At Tairawhiti Healthcare Limited "the key driver for the changes was the financial imperative for THL to live within its budget". This led to "a traumatised community within Gisborne Hospital … senior doctors and nurses felt disenfranchised … suspicion and distrust was endemic."[3] These instances illustrate how independent health professional advice was marginalised and compliant health advisors used by a management which did not act "in an orderly and accountable way".[4] Consultative mechanisms must be developed to ensure that this authoritarian type of relationship between CCMAU, the Ministry of Health and Boards and between Boards and health professional staff does not continue.

Traditionally the person or persons having authority must accept accountability. Over the last ten years accountability has been lost in the system or transferred to the health professional employee. Unless we develop effective working relationships between clinical staff, management and Boards, and discard the command and control approach, shared responsibility cannot develop properly. The creative solutions needed to coordinate primary, secondary and tertiary care services can be found if excessive prescription is removed and professionals can act with commonsense.

How can we put the past behind us?

- Mistakes need to be acknowledged in each organisation so that reconciliation can occur among health professionals and between health professionals and management. This will be difficult – but is necessary.
- The damage to relationships between management and health professionals by directives from CCMAU and the Ministry of Health needs to be acknowledged. Again, difficult: again necessary.
- Mechanisms for consultation between staff and management of District Health Boards should be developed and strengthened. Clinicians should be ELECTED by their colleagues to management roles for specified limited periods with the possibility of RE-ELECTION for one term. There should then be a period of stand-down for at least one term. This will ensure that there are valid clinical representatives.
- A change to more interactive administration needs to occur. Whilst health professionals employed as advisors, and chief executives will be a source of immediate advice, they should not be the only source. For trust and co-operation to be restored, honesty and openness need to replace the cosmetic consultation which occurred following the 'health reforms'.
- Unnecessary bureaucracy including paperwork needs to be removed so health professionals can give priority to direct patient care. The systems of financial control and accountability should be reviewed and those that are not cost-effective, or interfere with shared responsibility, should be withdrawn.

The legacy of the 'health reforms' has been disengagement by many health professionals from their previous commitment to the health service. Many remain disenfranchised and find enormous barriers to their hopes to bring about positive change. We cannot retrieve these ten lost years, but we now need to insist on effective change to the processes of management. We believe the answer lies in effective cooperation between management and health professional staff. The above points outline a possible way forward. What do you think?

The Editors

1. Plsek P, Wilson T. Complexity, leadership and management in health care organisation. BMJ 2001; 323:746-9.
2. A report by the Health and Disability Commissioner. Canterbury Health Limited. Auckland: 1998 April.
3. A report by the Health and Disability Commissioner. Gisborne Hospital 1999-2000. Auckland: 2001 March.
4. Satyanand A. But it's mine – isn't it? NZ Med J 2002; 115: 20-2.

Reporting news in health – good news, bad news and spin

Readers of our editorial articles might note that most have been critical of changes imposed on our health system over the past decade. The command-and-control approach to management, the dominance of financial directives in planning and the exclusion of professional input had deleterious effects on the delivery of health care whilst demoralising health professionals and wasting scarce resources. These editorials have been accused of lacking balance[1,2] and stretching the truth.[3]

This raises the issue of balance in the reporting of health matters. We have gained the impression that reports of health issues derived from "official" sources generally show a "good news" perspective. For example PHARMAC regularly sends out information on new deals it has made with pharmaceutical companies which will enhance patient access to medicines whilst saving precious funds. There are few reports on the broader effects of PHARMAC's actions on, for example, the paper-workload of health professionals, the ability of doctors to prescribe evidence-based treatments, the hospitalisation or adverse event rate[4,5] for patients forced to switch medications within or beyond a drug class, or the loss of support for research from industry. The Health Funding Authority was revealed to have "battled for positive spin" over the Gisborne cervical cancer crisis.[6]

Funding the good news machine is not necessarily cheap for the taxpayer. It is noteworthy in this regard that the Corporate Affairs Intelligence Unit of the National Health Service Executive (otherwise known as the 'good news unit') in the UK, established in 1993, was wound up in 1997 to save costs.[7] In New Zealand, some Crown Health Enterprises (CHEs) printed expensive, glossy brochures, ran programmes on local television stations and payed sizeable salaries to information officers to project a generally positive image at a time when, in retrospect, things were going wrong. At the same time, access to CHE information was neither free nor fulsome. One daily newspaper health reporter, clearly frustrated by how news was being managed by the local CHE, noted in 1999: "Before 1993, journalists reported on the monthly management meetings of local hospital boards. Pointedly, they were not invited into the boardrooms of post-reform managers. An important supply of objective public information dried up. Six years into the reforms, hospital managers still meet behind closed doors. Journalists, and thus the public they represent, remain excluded. Only what "information managers" decide is appropriate for public airing is released and controversial issues, if not kept under wraps, are given a carefully managed release".[8]

Times have moved on and we are in the era of District Health Boards (DHBs). One stated aim is that there should now be more openness, itself a recognition of the previous secretive approach. We hope that openness will indeed prevail. DHBs are currently struggling with insufficient funding and find themselves in industrial conflict with their nurses and allied health professionals. This is a result of the years of competitive, commercial management which has led to unacceptable disparities in pay and conditions throughout the country. It would be a positive step under these difficult fiscal circumstances for the DHBs to stop spending precious taxpayer dollars on activities that are more in the nature of advertising. We contend that DHBs should be open with the public and leave reporting of health news to independent investigative journalists. Until balance is restored to reporting on matters of health, we will continue to record, to the best of our ability, what is left out by the good news machinery. There seems no reason to apologise for filling in blank spaces.

The Editors

1. Roake J. The Booking System for surgery: evaluate or abandon. NZ Med J 2001; 114: 503.
2. Beasley S, MacMillam S. Reaffirming professionalism in medicine – yet again. NZ Med J 2001; 114: 504.
3. Davidson JRM. Creeping privatisation? NZ Med J 2002; 115: 26.
4. Thomas MC, Mann J, Williams S. The impact of reference pricing on clinical lipid control. NZ Med J 1998; 111: 292-4.
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7. Jones J. NHS "good news unit" to be wound up. BMJ 1997; 314: 769.
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University accused of violating academic freedom to safeguard funding from drug companies

An international group of renowned scientists has accused Canada's largest university of violating academic freedom for fear of losing research funds from drug companies when it revoked a job offer to an outspoken British psychiatrist.

A letter to the University of Toronto signed by 27 leading scientists, including two Nobel laureates of medicine, said the decision to rescind a professorship offered to Dr David Healy, who currently works at the University of Wales at Bangor, has "besmirched" the name of the University of Toronto and "poisoned the reputation" of its Centre for Addiction and Mental Health.

Dr Arvid Carlsson, this year's winner of the Nobel prize in medicine, and Dr Julius Axelrod, the 1970 winner, were among those who branded the affair "an affront to the standards of free speech and academic freedom."

Dr Healy was offered the post of director of the mood and anxiety disorders clinic at the centre after he was chosen by a search committee. He went to Toronto in November 2000 to discuss moving arrangements and to speak at a psychopharmacology seminar before some of his future colleagues. According to the executive director of the centre, Dr Paul Garfinkel, two of the points he made upset several of those colleagues: that selective serotonin reuptake inhibitors such as fluoxetine (Prozac) could lead to anxiety and suicidal thoughts; and that psychiatry, spurred on by the drugs industry, was overtreating people.

"Several of the people he would have been working with were deeply shocked by the extreme nature of his views, and by his poor methodology and lack of supporting evidence," Dr Garfinkel told the BMJ. "It was felt that, in a clinical setting, it would be difficult for him to effectively lead a programme where he could not rely on the respect of his colleagues."

Owen Dyer. BMJ 2001; 323: 591.

ORIGINAL ARTICLES

Awareness of Sudden Infant Death Syndrome risk factors among mothers of Pacific infants in New Zealand

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Abstract

Aim. To describe the awareness of Sudden Infant Death Syndrome (SIDS) risk factors among mothers of Pacific infants in New Zealand.

Methods. The data were gathered as part of the Pacific Islands Families Study in which 1376 mothers were interviewed when their infants were six weeks old. Included in this interview were questions designed to examine the mothers' awareness of SIDS risk factors.

Results. Over one third (38.8%) of mothers were unable to accurately report a SIDS risk factor, 53.4% reported the risk associated with putting the baby to sleep in a prone position, 31.5% maternal smoking, and 19.5% correctly reported other SIDS risk factors. Lack of awareness of SIDS risk factors was significantly associated with Samoan and Cook Islands Maori ethnicity, being Pacific Islands born, having no post school qualifications, lower household income, not being fluent in English, having more than five children, and not attending antenatal classes.

Conclusions. Despite SIDS prevention efforts, a considerable number of mothers in this cohort reported no awareness of SIDS risk factors. More effective methods are needed to provide consistent SIDS prevention information across Pacific ethnic groups.

NZ Med J 2002; 115: 33-5

Sudden Infant Death Syndrome (SIDS) is a leading cause of infant mortality in the postnatal period in New Zealand.[1] National and international prevention campaigns have been successful in reducing the SIDS mortality rate dramatically.[2,3] Between 1985 and 1994, the SIDS rate halved from 4.2 to 2.1 deaths per 1000 live births. The number of SIDS deaths for Pacific infants has been reported as similar to the rate for the total population.[4] However, there is concern that the real picture may be masked by ethnic misclassification and under reporting of SIDS and that Pacific SIDS mortality rates may either be remaining constant or increasing.[5-7]

The three main modifiable risk factors are prone sleeping position of the infant, maternal smoking, and lack of breastfeeding.[8,9] Sharing a bed with another person has also been recognised as a potential risk factor under some circumstances, particularly if the mother is a smoker.[10-14] In addition, there are a number of possible risk factors discussed by the medical profession and the media. One example is cot mattress wrapping[15] based on the toxic gas theory[16] and the disagreements that a number of researchers have with this theory.[17]

In view of concern expressed about the Pacific SIDS mortality rate[5-7] the main Pacific Islands Families project included questions designed to examine awareness that mothers of six-week old Pacific infants had of SIDS risk factors.

Methods

Data were collected as part of the Pacific Islands Families: First Two Years of Life (PIF) Study. The PIF Study is a longitudinal investigation of 1398 infants born at Middlemore Hospital, South Auckland during the year 2000. Middlemore Hospital was chosen as it has the largest number of Pacific births in New Zealand and is representative of the major Pacific ethnicities. All potential participants were selected from births at Middlemore Hospital where the child had at least one parent who identified as being of a Pacific Island ethnicity and also a New Zealand permanent resident. Recruitment occurred through the Birthing Unit in conjunction with the Pacific Islands Cultural Resource Unit that provided a daily list of Pacific admissions.

Approximately six-weeks after the birth of their child, Pacific interviewers, fluent in both English and a Pacific language, visited mothers in their homes. Once eligibility criteria were established and informed consent was gained, mothers participated in one-hour interviews concerning the health and development of the child and family functioning. This interview was carried out in the preferred language of the mother. Detailed information about the cohort and procedures is described elsewhere.[18]

To examine awareness of SIDS risk factors mothers were given a short description of SIDS and asked if they had heard advice about the ways parents could help prevent SIDS or cot death. If the mothers indicated that they had heard advice they were then asked to describe what parents were advised to do. No prompts were given. Socio-demographic and pre-natal factors that might be expected to influence awareness of SIDS risk factors were assessed by univariate and multivariate procedures.

Results

The cohort was made up of 87.8% of all eligible Pacific births that occurred from 15 March to 17 December 2000. Of the 1376 mothers of the cohort (1.7% gave birth to twins), 47.2% self identified their major ethnic group as Samoan, 21% as Tongan, 16.9% as Cook Islands Maori, 4.3% as Niuean, 3.4% as Other Pacific (includes mothers identifying equally with two or more Pacific groups, equally with Pacific and non-Pacific groups, or with Pacific groups other than Samoan, Tongan, Cook Island Maori or Niuean), and 7.2% as Non- Pacific. For mothers, mean (SD) age was 27 (6.2) years, 80.5% were married or in defacto partnerships, 33% were New Zealand born and 27.4% had post-school qualifications.

38.8% of mothers were unable to accurately report at least one SIDS risk factor, 53.4% reported the risk associated with putting the baby to sleep in a prone position and 31.5% maternal smoking. A small percentage reported the possible risk associated with co-sleeping (5.8%) or with not breastfeeding (1.7%). Some mothers (12.5%) reported infant

sleeping practices such as keeping the baby's face clear when they were sleeping and keeping the mattress clean and dry. A small number reported hearing about the need for special mattress wrapping[15] (0.4%) to reduce the risk of SIDS, and 5.8% gave responses that were also coded inaccurate. These responses were concerned with maternal alcohol use, general infant feeding and keeping the house tidy.

Table 1 lists the variables examined for potential association with lack of awareness of SIDS risk factors. For the categories within each variable the numbers and percentages of mothers who did not report at least one SIDS risk factor are given, along with the associated odds ratios. Ethnicity, household income, maternal education, English fluency, parity, maternal birthplace and attendance at antenatal classes were significant ($p< 0.05$). Maternal age and social marital status did not reach significance. When controlling for the effects of all variables in Table 1 in a multiple regression model, factors that remained significantly associated ($p<0.05$) with lack of awareness of SIDS risk factors were the mother's ethnic identity, being Pacific Island born, having no post school qualifications, not being fluent in English and not having attended antenatal classes.

Discussion

These findings show that despite efforts to prevent SIDS, a considerable percentage (38.8%) of mothers in this cohort had no awareness of SIDS risk factors. Only 53.4% of mothers reported the risk associated with putting the infant to sleep in a prone position and 31.5% about maternal smoking. Smaller percentages correctly reported other major SIDS risk factors. These findings are in line with results reported in New Zealand[9] and Australian[19] studies. The 1993 New Zealand study in which Pacific mothers (18.5%) were part of the sample, found that without prompting 60% of the sample identified prone sleeping position, 55% maternal smoking, 25% lack of breastfeeding and 14% co-sleeping as SIDS risk factors.[9] Our findings suggest that little progress has been made in raising awareness of mothers of Pacific infants about SIDS risk factors.

In the present study, 0.4% of mothers reported hearing about the need for special mattress wrapping[15] and some gave inaccurate responses about what parents can do to reduce the risk of SIDS. It is likely that the number of possible risk factors and conflicting messages may be responsible for some confusion among mothers about what are established, modifiable SIDS risk factors. Furthermore, information about parental care practices that may pose a risk to the infant may have been given but not linked overtly to SIDS.

There were significant ethnic differences in mother's awareness of SIDS risk factors. Of particular concern is the finding that Samoan and Cook Islands Maori mothers were significantly more likely than Tongan mothers to lack awareness about SIDS risk factors. Although most SIDS risk factors are described in brochures, printed in all these Pacific languages, it is possible that more emphasis is placed on SIDS by some Pacific community groups than others.

Compared to New Zealand- born mothers, their Pacific-born counterparts were significantly more likely to lack awareness about SIDS risk factors. As there are no specific

Table 1. Numbers (percentages) and univariate odds ratios of lack of awareness of SIDS risk factors by selected maternal variables.

Variable	Category	No SIDS risk factors reported		Univariate odds ratio (95% CI)	
Age (yr)	<20	48	(43.2)	1.00	
	20-29	258	(35.8)	0.73	(0.49, 1.10)
	30-39	207	(41.4)	0.93	(0.61, 1.41)
	40+	20	(45.5)	1.09	(0.54, 2.21)
Ethnicity	Tongan	79	(27.3)	1.00	
	Samoan	301	(46.3)	2.29	(1.70, 3.10)‡
	Cook Islands Maori	103	(44.4)	2.12	(1.47, 3.06)‡
	Niuean	18	(30.5)	1.17	(0.63, 2.15)
	Other Pacific§	14	(29.8)	1.13	(0.57, 2.22)
	Non Pacific	19	(19.2)	0.63	(0.36, 1.11)
Social Marital Status	Non partnered	101	(37.7)	1.00	
	Partnered	433	(39.1)	1.10	(0.81, 1.40)
Education	Post school qualification	85	(22.5)	1.00	
	Secondary school qualification	154	(33.2)	1.71	(1.25, 2.33)†
	No formal qualifications	295	(55.1)	4.22	(3.14, 5.68)‡
English Fluency	Yes	246	(28.9)	1.00	
	No	288	(54.9)	2.99	(2.38, 3.75)‡
Household Income	<$10 000	42	(47.2)	2.62	(1.30, 5.29)†
	$10 000-$20 000	149	(40.5)	2.00	(1.09, 3.65)*
	$20 001-$30 000	185	(40.9)	2.03	(1.12, 3.69)*
	$30 001-$40 000	83	(32.2)	1.39	(0.75, 2.60)
	$40 001-$50 000	35	(35.7)	1.63	(0.81, 3.29)
	>$50 000	16	(25.4)	1.00	
	Unknown	24	(50.0)	2.93	(1.32, 6.63)†
Parity	1	133	(35.6)	1.00	
	2-4	294	(38.4)	1.13	(0.87, 1.46)
	5+	96	(44.7)	1.46	(1.04, 2.06)*
	Unknown	11	(52.4)	1.99	(0.83, 4.82)
Born in NZ	Yes	117	(25.8)	1.00	
	No	417	(45.2)	2.38	(1.86, 3.05)‡
Attended Antenatal Classes	Yes	18	(15.9)	1.00	
	No	508	(40.5)	3.59	(2.14, 6.02)‡

*p<0.05; †p<0.01; ‡p<0.001. §Includes mothers identifying equally with two or more Pacific groups, equally with Pacific and non Pacific groups, or with Pacific groups other than Tongan, Samoan, Cook Islands Maori or Niuean.

SIDS prevention programmes in the Pacific Islands nations, Pacific-born mothers may be unaware that SIDS is a health issue in New Zealand and may be less likely to pay attention to prevention programmes. Indeed, mothers not fluent in English were significantly more likely to lack awareness about SIDS risk factors. These findings are in line with earlier work[9] which found that mothers reporting no knowledge of SIDS factors also had limited English. Even though brochures are printed in the major Pacific languages, it is clear that the SIDS prevention message is not reaching many mothers in the present cohort.

The association of socioeconomic factors with SIDS[10,20,21] demonstrates a need to address poverty and unemployment in prevention programmes.[21-24] The mothers in this cohort were primarily resident in South Auckland where economic issues are of concern for many families. Our findings revealed significant associations between low awareness of SIDS and no post school qualifications, a household income below $30 000, and having more than five children. Absence of post school qualifications had earlier been shown to be associated with signifcantly reduced awareness of SIDS.[19]

These findings support the suggestion[21] that information about SIDS risk factors is not reaching mothers in some communities. Issues that may affect awareness of SIDS risk factors include access to written information that is expensive, limited attendance at antenatal classes, short hospital stays, reliance on emergency services, and the highly mobile nature of Pacific families. We demonstrate that mothers who did not attend antenatal classes (8.2%) were significantly more likely to lack awareness of SIDS risk factors than mothers who had attended classes. One reason for lack of awareness of risk factors may therefore be difficulties in reaching mothers who do not attend antenatal classes or maternity groups. More resources are needed to strengthen community networks and create opportunities to provide information to mothers who may be picking up confusing messages from other sources. SIDS programmes on Pacific radio stations, television and video may be effective ways to reach Pacific mothers.

Delivery of information about SIDS risk factors needs to be tailored to specific Pacific groups and to be available to mothers (and the extended family) in the months after the birth of the infant. Information is likely to reach Pacific communities most effectively when there is an integrated approach among health professionals, community leaders and Pacific agencies. In particular, uniformity of messages about SIDS, provided in a consistent way, may decrease confusion and raise awareness of SIDS risk factors among mothers of Pacific infants.

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Antimicrobial use in animal feed – time to stop

Antimicrobials have been used in food animals in North America and Europe for nearly half a century. Among the most common are drugs that are either identical to or related to those administered to humans, including penicillins, tetracyclines, cephalosporins (including ceftiofur, a third-generation cephalosporin), fluoroquinolones, avoparcin (a glycopeptide that is related to vancomycin), and virginiamycin (a streptogramin that is related to quinupristin-dalfopristin). These antimicrobial agents are given to food animals as therapy for an infection or, in the absence of disease, for subtherapeutic purposes with the goals of growth promotion and enhanced feed efficiency (improved nutritional benefits of the animal feed). There is considerable controversy about the amounts of antimicrobials that are given to food animals, relative to the amounts given to humans, since manufacturers are not required to provide precise production figures. One estimate is that 50 percent of all antimicrobials produced in the United States are administered to animals, mostly for subtherapeutic uses. The union of Concerned Scientists recently estimated that, each year, 24.6 million lb (11.2 million kg) of antimicrobials are given to animals for nontherapeutic purposes and 2 million lb (900,000 kg) are given for therapy; in contrast, 3 million lb (1.3 million kg) are given to humans. Whichever figures are accepted, it is fair to state that substantial amounts of antimicrobials are administered to food animals for growth promotion and feed efficiency in the absence of known disease.

On the basis of discussions by an expert committee of the Alliance for the Prudent Use of Antibiotics, several recommendations can be made. Antimicrobials should be used only when indicated in individual infected animals for a targeted pathogen and prescribed by a veterinarian. The use of certain drugs that have important uses in humans, such as fluoroquinolones and third-generation cephalosporins, should be prohibited in animals. Finally, the subtherapeutic use of these agents to promote growth and feeding efficiency should be banned – a move that would decrease the burden of antimicrobial resistance in the environment and provide health-related benefits to both humans and animals.

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Associations between ethnicity and obstetric intervention in New Zealand

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Abstract

Aims. To determine whether the lower rates of obstetric interventions in Maori and Pacific Island women from the New Zealand Ministry of Health obstetric procedures report in 1999 existed also in National Women's Hospital (NWH), Auckland data and if so whether they persisted after controlling for parity and obstetric risk.

Methods. The study population included 43 367 singleton, cephalic deliveries, not preceded by caesarean section at NWH from 1992-1999. Ethnicity was Maori, Pacific Island, or other. Obstetric interventions were explored at two time points: (1) at the initiation of the delivery process: induction of labour, prelabour caesarean section, or spontaneous onset of labour; and (2) at the point of delivery: either caesarean section, operative vaginal delivery, or spontaneous vaginal birth. Independent associations were found by fitting polytomous logistic regression models.

Results. 10% of the study population were Maori, 19% Pacific Islanders, and 71% other. Unadjusted analyses showed lower rates of all obstetric interventions for Maori and Pacific Island women. Adjusted analyses showed that rates of induction of labour, prelabour caesarean, and operative vaginal delivery were lower for Maori and Pacific women than for all other ethnicities grouped together. However, caesarean delivery rates overall were not different for Maori or Pacific Island women.

Conclusions. The adjusted analysis did not confirm the association seen in the New Zealand Ministry data between ethnicity and caesarean section. However, induction, prelabour caesarean section, and operative vaginal delivery were less common in Maori and Pacific Island women.

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In October 1999, the New Zealand Ministry of Health released an obstetric procedures report[1] which identified a relationship between lower rates of obstetric procedures, including induction of labour, epidural anaesthesia, episiotomy, operative vaginal delivery, and caesarean section, and Maori or Pacific Island ethnicity. Maori and Pacific Island women have higher risk pregnancies and more health problems[1] and so higher rates of obstetric intervention might be expected. The opposite finding in the Ministry report was proposed as evidence that "factors to do with preferences and expectations are playing a more significant part than clinical need".[1] The report concluded that the data "raise questions that should be addressed by clinicians in individual units".[1]

The above findings raise concern. Firstly, if maternal preferences and expectations are playing an important role in obstetric interventions, then this is of relevance to the ongoing debate and concern related to the aetiology and management of rising obstetric intervention rates. Secondly, these conclusions are based on simple statistical analyses that are corrected only for maternal age, while the associations may be confounded by other factors, such as parity and clinical risk which are not accounted for.

The aim of this study was to explore whether the associations reported between ethnicity and obstetric intervention in the Ministry document were present in the National Women's Hospital (NWH) data and if so whether they persisted after controlling for potential confounding factors such as parity and obstetric risk.

Methods

The study population included the first singleton, cephalic delivery, not preceded by previous caesarean section, of women delivered at NWH, Auckland, between January 1992 and December 1999. All data were from the obstetric database at NWH. Ethnicity was categorised as Maori, Pacific Island, and 'other' for comparability with the Ministry document. Ethnicity was obtained from the booking form sent to the hospital by a private caregiver (obstetrician, general practitioner, or midwife) or filled in by the hospital midwife at the first hospital antenatal visit. Only one ethnic group was recorded per woman. All Pacific Island groups were included together. Other ethnicities include European, Asian, and 'Other'. Obstetric interventions were explored at two time points: (1) at the initiation of the delivery process: either induction of labour (including failed induction), prelabour caesarean section (including elective caesarean before labour and emergency caesarean before onset of contractions), or spontaneous onset of labour; and (2) at the point of delivery: either caesarean section, operative vaginal delivery, or spontaneous vaginal birth.

Statistical Methods. Univariate associations between ethnicity and obstetric interventions are presented as odds ratios (with 95% confidence intervals). Univariate associations between maternal characteristics, obstetric risk factors and ethnicity have been tested using Chi-squared tests for frequency data. Independent associations between ethnicity and obstetric interventions were explored at the two time points by fitting polytomous logistic regression models using the catmod procedure of SAS Version 6.12 (Cary, North Carolina). Polytomous logistic regression allows a logistic regression model to be fitted in a situation whether there are more than two possible outcomes, as seen here. Three models are presented for each of the outcomes; the first adjusting only for maternal age (as in the Ministry document), the second adjusting for age and parity, and the final model adjusting for a larger selection of confounders (as listed in the Results). Variables were included in the final models for a priori reasons, and then removed by manual backward selection if they were insignificant ($p>0.05$) and were not important confounders (ie, did not alter the parameter estimates for ethnicity by more than 10%). The variables included in the models were defined as follows: parity was either nulliparous or multiparous; smoking included any smoking before or during pregnancy; gestation at hospital booking for a public patient was the gestation the patient first visited the hospital and for a private patient was either the gestation at the first visit or the gestation at which details of booking were entered onto the hospital database; caregiver was public, private general practitioner, private midwife, private obstetrician, and some women were unbooked; transfer was transfer of care from booking at another facility to NWH prior to birth; hypertension included women with a history of essential hypertension or diastolic blood pressure > 90mmHg in pregnancy; diabetes included pre-existing and gestational diabetes; antepartum haemorrhage was any bleeding after 20 weeks gestation; small for gestational age was birthweight less than the 3rd percentile for gestational age;[2] preterm was birth <37 completed weeks gestation; postterm delivery was delivery at or after 41 completed weeks gestation.

Results

There were 66 952 deliveries at NWH between January 1992 and December 1999. The study sample includes the first eligible pregnancy of 43 367 women who had singleton,

cephalic deliveries, without previous caesarean section. Of these pregnancies, 4361 (10%) mothers were Maori, 8197 (19%) were Pacific Islanders, and 30 809 (71%) were of other ethnicities. The unadjusted associations between ethnicity and obstetric interventions are presented in Table 1 as rates and odds ratios, showing the significantly lower intervention rates in Maori and Pacific Island women compared to all other ethnicities together.

Table 1 presents unadjusted and adjusted associations between ethnicity and each intervention. The odds ratios for induction of labour for Maori and Pacific Island women compared to all others increase towards one (or no effect) with adjustment for age and parity, highlighting that Maori and Pacific Island women have their babies younger and have more babies. However, adjusting for other confounders, including obstetric risk factors, reduces these odds ratios again and results in significantly lower rates in Maori and Pacific Island women compared to 'other'.

The associations between ethnicity and prelabour caesarean show similar patterns to the associations with induction, with attenuation of the association after adjusting for the increased age and increased rate of nulliparity of 'other' ethnicities, but persistence of reduced odds (approximately 40%) of prelabour caesarean after controlling for other factors.

The attenuation, after controlling for age and parity, of the large odds of operative vaginal delivery for Maori and Pacific Island women compared to other ethnicities shows again that older age and nulliparity (as seen in the 'other' ethnicities group) are associated with increased intervention rates. However, the marked reduction in odds (30 and 50%) for Maori and Pacific Island women persists after controlling for other obvious risk factors.

The association between ethnicity and caesareans (including prelabour and emergency) is attenuated by age and parity to one, and although there is some reduction in the odds ratio with adjusting for other risk factors, there is no significant difference in caesarean section rate for Maori or Pacific Island women compared to other ethnicities.

Table 2 shows unadjusted associations between maternal characteristics, obstetric risk factors, perinatal outcomes and ethnicity. There were significant associations between ethnicity and: age, parity, smoking, gestation at booking, booking caregiver, transfer of care, small for gestational age birth, hypertensive disease, diabetes, antepartum haemorrhage, gestation at delivery and birthweight. Maori and Pacific Island women were less likely to have epidural analgesia.

Discussion

This analysis was prompted by concern at the findings of the Ministry of Health document (1999) which revealed lower rates of obstetric intervention in women of Maori or Pacific Island ethnicities.[1] In the current study, unlike that of the Ministry, it was possible to control for age and parity and for a number of potential confounders in the complex relationship between ethnicity and obstetric intervention. Comprehensive prospective national perinatal data collection would allow this type of analysis to be performed centrally resulting in more informative presentation of national data.

Our data show that rates of induction of labour, prelabour caesarean, and operative vaginal delivery are much lower for Maori and Pacific women than for all other ethnicities grouped together, even after controlling for measurable differences between the groups. However, caesarean delivery rates overall are not different for Maori or Pacific Island women compared with 'other' women. The observed unadjusted differences in caesarean section rate associated with ethnicity are attributable to age and parity differences. Maori and Pacific Island women have lower rates of prelabour caesarean which are compensated for by higher rates of emergency caesarean.

The findings of this analysis are limited by the data available within the NWH database. It was not possible to look at the potential confounding effect of maternal weight because of missing data. Nor was it possible to look at the temporal relationship between epidural analgesia and obstetric intervention as the data regarding dilatation and time at insertion of an epidural are not currently collected.

Previous reports of the associations between ethnicity and caesarean section rate in New Zealand have been unadjusted for the important confounders controlled for here (especially age and parity) and their findings have therefore been similar to those of the Ministry report.[3-5]

Table 1. Associations [OR (95% CI)] between ethnicity and induction of labour, prelabour caesarean section, operative vaginal delivery, and caesarean section.

Outcome	Ethnicity	N/total n	Rate (%)	Unadjusted model OR (95% CI)	Age adjusted model OR (95% CI)	Age and parity adjusted model OR (95% CI)	Final model* OR (95% CI)
Induction of labour†							
	Other	8749/30 809	28.4	Referent	Referent	Referent	Referent
	Maori	1091/4361	25.0	0.83 (0.77-0.89)	0.87 (0.80-0.94)	0.96 (0.89-1.04)	0.85 (0.78-0.93)
	Pacific Islander	1791/8197	21.9	0.69 (0.65-0.73)	0.71 (0.59-0.85)	0.78 (0.73-0.83)	0.69 (0.64-0.74)
Prelabour caesarean†							
	Other	960/30 809	3.1	Referent	Referent	Referent	Referent
	Maori	103/4361	2.4	0.71 (0.58-0.88)	0.86 (0.69-1.07)	0.97 (0.78-1.20)	0.57 (0.43-0.75)
	Pacific Islander	150/8197	1.8	0.53 (0.44-0.63)	0.59 (0.49-0.70)	0.65 (0.54-0.78)	0.58 (0.46-0.72)
Operative vaginal delivery‡							
	Other	5971/30 809	19.4	Referent	Referent	Referent	Referent
	Maori	406/4361	9.3	0.39 (0.35-0.44)	0.42 (0.38-0.47)	0.69 (0.62-0.78)	0.71 (0.63-0.81)
	Pacific Islander	598/8197	7.3	0.31 (0.28-0.33)	0.32 (0.29-0.35)	0.49 (0.45-0.54)	0.50 (0.45-0.56)
Caesarean section‡							
	Other	4825/30 809	15.7	Referent	Referent	Referent	Referent
	Maori	485/4361	11.1	0.58 (0.52-0.64)	0.67 (0.60-0.74)	1.04 (0.93-1.16)	0.93 (0.82-1.06)
	Pacific Islander	1033/8197	12.6	0.65 (0.61-0.70)	0.71 (0.66-0.76)	1.03 (0.95-1.12)	0.94 (0.85-1.03)

*Induction of labour and prelabour caesarean final models adjusted for age, parity, smoking, hospital booking before 24 weeks, booking caregiver, transfer, obstetric risk factors (hypertension, diabetes, SGA, APH), postterm delivery (41+ weeks), and birthweight. Operative vaginal delivery and caesarean section final models adjusted for age, parity, smoking, hospital booking before 24 weeks, booking caregiver, transfer, obstetric risk factors (hypertension, diabetes, SGA, APH), preterm delivery (<37 weeks), postterm delivery (41+ weeks), and birthweight. †Odds ratios present odds of induction of labour or prelabour caesarean versus spontaneous onset of labour for Maori or Pacific Island women compared to the odds for all other ethnicities. ‡Odds ratios present odds of operative vaginal delivery or caesarean section versus spontaneous vaginal delivery for Maori or Pacific Island women compared to the odds for all other ethnicities.

Table 2. Univariate associations between maternal characteristics, obstetric risk factors, perinatal outcomes, and ethnicity.

	Maori n=4361		Pacific Island n=8197		Other n=30 808		p
Age (yrs)							
<20	946	22%	817	10%	1191	4%	
20-24	1402	32%	2320	28%	4176	14%	
25-29	1025	23%	2514	31%	9259	30%	
30-34	626	14%	1616	20%	10 678	35%	
35-39	287	7%	730	9%	4648	15%	
≥40	75	2%	200	2%	856	3%	≤0.001
Nulliparous	2260	52%	4046	49%	20 514	67%	≤0.001
Smoker*	2393	62%	2080	28%	5237	19%	≤0.001
Booked before 24 wks	2734	67%	4601	56%	23 741	77%	≤0.001
Booking caregiver†							
Public	2582	59%	5506	67%	9516	31%	
Private obstetrician	121	3%	149	2%	7626	25%	
General practitioner	911	21%	1628	20%	9051	29%	
Independent midwife	626	14%	742	9%	4348	14%	
Unbooked	98	2%	143	2%	115	0.4%	≤0.001
Transfer	621	14%	579	7%	2505	8%	≤0.001
Small for gestational age	175	4%	151	2%	1034	3%	≤0.001
Hypertensive disease	446	10%	933	11%	2682	9%	≤0.001
Diabetes (any type)	101	2%	314	4%	688	2%	≤0.001
Antepartum haemorrhage	244	6%	328	4%	1425	5%	≤0.001
Gestation at delivery							
Preterm (<37 wks)	504	12%	498	6%	2626	9%	≤0.001
Postterm (≥41 wks)	811	19%	1790	22%	6918	22%	≤0.001
Birthweight							
<1000g	57	1.3%	51	0.6%	263	0.9%	
1000-1999g	167	3.8%	121	1.5%	697	2.3%	
2000-2999g	1043	24%	1014	12%	6303	20%	
3000-3999g	2634	60%	5426	66%	20 028	65%	
4000-4999g	454	10%	1537	19%	3479	11%	
≥5000g	6	0.1%	48	0.6%	38	0.1%	≤0.001
Epidural analgesia	1445	33%	2460	30%	15 574	51%	<0.001

*Smoking status unknown = 4679. †Private unknown caregiver = 205.

Similarly, a recent paper from New South Wales showed indigenous women had lower unadjusted rates of obstetric interventions (induction of labour, planned caesarean section, epidural, caesarean after labour, instrumental delivery, and episiotomy).[6] More interesting analyses have adjusted for confounding factors. A recent analysis from Adelaide reported an association between non-Caucasian ethnicity and lower rates of induction or elective caesarean section, but not with caesarean section after labour.[7] A Californian study showed lower rates of caesarean among Blacks compared to Whites in unadjusted analyses, but higher rates among Blacks in the adjusted analyses.[8] A further North American publication, from Alabama, showed lower rates of caesarean section for Blacks in unadjusted analyses, but no difference after adjusting for sociodemographic and obstetric/medical risk factors.[9] White non-Hispanics had a higher rate of induction of labour than all other ethnic groups after controlling for clinical factors (including age, parity, and medical/obstetric risk factors) in a study from Arizona.[10] There is a lack of published adjusted data on the association between ethnicity and obstetric interventions other than caesarean section.

If we assume that this multivariate analysis controls for most clinical and other differences that might affect the association between ethnicity and obstetric intervention, then we expect that the odds ratios for obstetric intervention in the final models would be one for all ethnic groups. That this is true for total caesarean section shows that ethnicity is not associated with caesarean section rates which suggests that caesarean section is performed for perceived 'clinical need'. Conversely, that the odds ratios for Maori and Pacific Island women for induction of labour, prelabour caesarean, and operative vaginal delivery are significantly less than one suggests that other factors may play a role. Lower rates of epidural analgesia for Maori and Pacific Island women may partly explain the lower rate of operative vaginal delivery, as randomised trials have shown that epidural analgesia increases operative vaginal delivery rate.[11] Access to care in this urban population probably does not explain the differences in these analyses as gestation at booking (defined as before or after 24 weeks) was included as a variable in the multivariate models.

Further elucidation of the reasons for disparity in obstetric interventions by ethnicity might involve qualitative methodology such as focus groups. If it is found that women are making the decisions which are leading to increases in interventions without proven benefits, they deserve education to allow them to make well informed decisions.

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Unlocking the numerator-denominator bias. I: adjustments ratios by ethnicity for 1991-94 mortality data. The New Zealand Census-Mortality Study

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Abstract

Aim. To determine the extent of the under-reporting of Mäori and Pacific mortality among 0-74 year olds for the period 1991-94.

Methods. A subset (n=22 578) of highly probable linked 1991 census and 1991-94 mortality records were selected from the 31 635 census-mortality links in the New Zealand Census-Mortality Study. The numbers of decedents assigned as Mäori, Pacific, and non-Mäori non-Pacific were compared between mortality and census data.

Results. Compared to the death registration form, 29% more 0-74 year old decedents during 1991-94 had self-identified as sole-Mäori on the 1991 census (46% for prioritised-Mäori). This numerator-denominator bias was greater among the young and those living in central and southern New Zealand. Among 0-14, 15-24, 25-44, 45-64, and 65-74 year old decedents, respectively, 91%, 50%, 41%, 26% and 15% more decedents had self-identified as sole-Mäori on the 1991 census. For Northern, Midland, Central and Southern regional health authority areas, respectively, 14%, 17%, 81% and 102% more decedents had self-identified as sole-Mäori. Among Pacific decedents 68% more 0-74 year old decedents had self-identified as sole-Pacific on the 1991 census (78% for prioritised-Pacific group). This bias for Pacific decedents did not notably vary by age and region.

Conclusions. This study confirms substantial underestimation of Mäori and Pacific mortality rates for the period 1991-94, even using the recommended sole-ethnic group denominator. The results from this study should be used to adjust ethnic-specific mortality rates for the early 1990s. Population-based funding formulas that included region-specific Mäori mortality rates would have particularly disadvantaged central and southern regions.

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Mortality and census data routinely collected by government are used for measuring trends over time, planning and evaluating health services and policies, and for deciding where health funding gets spent. Inaccuracies in the recording of ethnicity can bias the health profiles of population groups and resultant resource allocation decisions.

Mäori and Pacific mortality rates in New Zealand are known to be higher than non-Mäori non-Pacific mortality rates.[1] Routinely published mortality rates use census data for the denominator, and mortality data (sourced from death registration forms usually completed by the funeral director) for the numerator. There is a major problem affecting these routinely calculated and published rates (especially for the period prior to 1996) – namely the use of different ethnicity questions and data collection methods between the death registration form and the census, causing a 'numerator-denominator bias'. The change in how ethnicity was collected on mortality data from September 1995 on, making comparisons over time difficult, is another problem,[2-4] but is beyond the scope of this paper.

Prior to 1995 the 'degree of Mäori (Pacific) blood greater than half' was recorded on the death registration form (RG28). If this section of the death registration form was not completed then the deceased was classified as non-Mäori non-Pacific. It appears that funeral directors often assumed the extent of ancestry of the deceased, or did not complete it.[5] In contrast, the 1986 and 1991 censuses elicited self-identified ethnicity, and allowed for the recording of multiple ethnic groups. The combination of these factors has lead to significant undercounting of Mäori and Pacific deaths.[6] The extent of this under-counting varied with both age[3] and locality.[2,7] In the calculation of ethnic specific mortality rates the numerator-denominator bias was minimised, but not removed, by the use of sole-Mäori (or Pacific) classification for the denominator data.[5] Previously, Graham et al estimated that Mäori coronary heart disease deaths in Auckland during 1983-84 (using routine mortality data) were underestimated by nearly half compared to a self-identified ethnic group.[8]

The objective of our study was to quantify the numerator-denominator bias for national ethnic-specific mortality rates during 1991-94, both for calculations using the sole and prioritised ethnic group denominators (Mäori, Pacific, and non-Mäori non-Pacific). We used mortality records from 1991-94 that had been linked to the 1991 census in the New

Zealand Census-Mortality Study (NZCMS).[9] The primary aim of the NZCMS is to investigate socio-economic determinants of mortality. However, the linking of census and mortality records allows a direct comparison of the ethnic group on mortality data and the preceding census for what is highly likely to be the *same* individual. As such, the NZCMS enables quantification of the numerator-denominator bias affecting mortality rates for the total population, and by sex, age, region and deprivation.

Methods

Generating a weighted data set of linked census and mortality records. 41310 mortality records were obtained from New Zealand Health Information Service for all New Zealand residents aged 0-74 years on census night 1991 (5 March), and who died in the subsequent three years. Variables available included geocodes (meshblock and census area units), sex, age, ethnicity (half or more Māori or Pacific 'blood'), country of birth, regional health authority (RHA), and small area deprivation. The deprivation variable was the NZDep91 index, which measures relative socio-economic deprivation of the small area in which the person lived.[10] NZDep91 scores were available for 36 927 mortality records (89.4%).

The mortality records were anonymously and probabilistically linked to 1991 census records by Statistics New Zealand as part of the NZCMS.[9,11,12] For this particular study strict criteria were set for inclusion: of the 41 310 mortality records 22 578 were successfully linked to a census record with the same meshblock, sex, date of birth and country of birth, and had a NZDep91 score.[12] Inclusion in the linked data-set for this study varied by demographic factors, most notably only 34.5% of the Māori and 35.7% of the Pacific mortality records (based on the death registration form) were linked, compared to 56.9% of non-Māori non-Pacific mortality records. Inclusion largely depended on two factors: first having a meshblock assigned to the death record, and second each decedent having the same meshblock on their mortality record and their previous census record. Approximately 20% of Māori did not have a meshblock assigned to their death record compared to 10% of non-Māori. Other factors affecting the chance of being linked included younger age, rurality, mobility and socioeconomic status. Because of this varying inclusion in the linked data-set by ethnic group (and other demographic variables) we weighted the number of deaths in each demographic strata to be representative of the 41 310 mortality records.[9,11,12] As not all mortality records had a NZDep91 score, two weights were estimated then combined into an overall weight.

Step 1: Calculating a weight to make the linked data-set (n=22 578) representative of the eligible mortality records with NZDep91 scores (n=36 927). For those deaths with an NZDep91 score, 243 strata were formed by cross-classifying sex, age, RHA and NZDep91 groupings. For Māori and Pacific decedents NZDep91 and RHA categories were aggregated to ensure that no strata had less than three decedents on the final data set, and in most instances more than ten. The within-stratum weights were calculated by dividing the number of eligible mortality records with non-missing NZDep91 scores (n=36 927) by the number of mortality records in the linked data set (n=22 578) within each of the 243 strata.[12]

Step 2: Calculating a weight to make the mortality data set with NZDep91 scores (n=36 927) representative of all eligible mortality records (n=41 310). A total of 90 strata were formed, 40 each for Māori and non-Māori non-Pacific (sex by age-group by RHA). For Pacific decedents, RHA was simply disregarded making ten strata. The within-stratum weights were calculated by dividing the number of all eligible mortality records by the number of eligible mortality records with non-missing NZDep91 scores within each of the 90 strata.

Step 3: Calculate the final weight. The product of the above two weights (the final weight) was then assigned to each mortality record in the final data set.

Determining the numerator-denominator bias. The numerator-denominator bias was calculated as a 'correction factor' by using weighted cross-classifications of the linked data-set, for death registration form ethnicity by the census 'sole' or 'prioritised' ethnicity. (*Sole* ethnic group was assigned as Māori on the 1991 census if only one ethnic group was self-identified, and that was Māori. The sole group was assigned as Pacific if the census form stated only one self-identified ethnic group, and that was Pacific. The remainder were assigned as non-Māori non-Pacific. *Prioritised* ethnic group was assigned as Māori if one of the three possible self-identified ethnicity responses on the 1991 census was Māori. For those not allocated as Māori, the prioritised ethnic group was assigned as Pacific if one of the self-identified ethnic groups was Pacific. The remainder were assigned as non-Māori non-Pacific.) Further, this cross-classification (for sole census ethnicity only) was conducted by strata of sex, age, RHA, and small area deprivation to determine heterogeneity of the numerator-denominator bias.

Results

Table 1 shows the weighted cross-classification of 0-74 year olds deaths during 1991-94 by the death registration form and census sole ethnicity. 4482 of the deaths were estimated as having self-identified as sole-Māori on the 1991 census, compared to 3471 that were recorded as Māori on the death registration form. Thus, the number of Māori deaths according to mortality data for the 1991-94 period needed multiplying by 1.29 to be comparable to the 1991 census sole-Māori ethnic group as a denominator. For Pacific people (sole census ethnicity), the 'correction factor' was substantially greater at 1.68. For non-Māori non-Pacific the ratio was 0.96. The prioritised Māori, Pacific, and non-Māori non-Pacific correction factors were further again from 1.0, being 1.46, 1.78 and 0.94, respectively (Table 1).

For Māori, the adjustment ratios by age-group were 1.9, 1.50, 1.41, 1.26, and 1.15 for the 0-14, 15-24, 25-44, 45-64, and 65-74 year olds, respectively (Table 2). A trend by age was not evident for Pacific people, although the ratios estimates were imprecise (due to small numbers) for 0-14 and 15-24 year olds. As the Māori (and Pacific) adjustment ratios were so high for 0-14 year olds, and Māori and Pacific people make up a higher proportion of 0-14 year olds than of the total population, the non-Māori non-Pacific ratio among 0-14 year olds was substantially less than 1.0, being 0.83.

There was also marked regional variation in the numerator-denominator bias by RHA for Māori, with the

Table 1. Death registration form ethnicity by census sole and prioritised ethnicity, for 1991-94 mortality records linked to a 1991 census record.

Census ethnicity	Death registration form ethnicity: Māori	Pacific	non-M non-P	Total	Census to mortality ratio*
Sole					
Māori	3162	6	1314	4482	1.29
Pacific People	12	618	474	1101	1.68
non-M non-P	300	30	35 397	35 727	0.96
Total	3471	654	37 182	41 310	
Prioritised					
Māori	3342	12	1719	5076	1.46
Pacific People	12	639	513	1164	1.78
non-M non-P	120	6	34 947	35 070	0.94
Total	3471	654	37 182	41 310	

All numbers in table are weighted, and then random rounded to a multiple of three as per Statistics New Zealand protocol. Minimum cell size is 6. *The census to mortality ratio is the census total divided by the death registration form total eg, for Māori sole ethnicity 1.29 = 4482/3471. As such, 1.29 is the correction factor to apply to 1991-1994 ethnic specific mortality rates calculated using sole ethnicity as the denominator. The true ratio (calculated without random rounding due to Statistics New Zealand protocol) was within plus or minus 0.05 of the given ratio.

Table 2. Number of deaths stratified by age by: death registration form ethnicity, census sole ethnicity, and census prioritised ethnicity.

Age group	Ethnic group	Death reg form	Census sole	Census prioritised	Census sole to death reg ratio*
0-14 years	Mäori	96	183	243	1.91†
	Pacific People	45	72	81	1.60‡
	non-M non-P	687	573	507	0.83
	Total	831	831	831	
15-24 years	Mäori	210	315	417	1.50
	Pacific People	45	84	93	1.87‡
	non-M non-P	1512	1368	1251	0.90
	Total	1764	1764	1764	
25-44 years	Mäori	582	819	912	1.41
	Pacific People	117	219	225	1.87
	non-M non-P	3510	3171	3066	0.90
	Total	4206	4206	4206	
45-64 years	Mäori	1713	2160	2373	1.26
	Pacific People	273	450	468	1.65
	non-M non-P	12 933	12 309	12 078	0.95
	Total	14 922	14 922	14 922	
65-74 years	Mäori	873	1005	1128	1.15
	Pacific People	171	279	291	1.63
	non-M non-P	18 537	18 303	18 165	0.99
	Total	19 584	19 584	19 584	

All numbers in table are weighted, and then random rounded to a multiple of three as per Statistics New Zealand protocol. *The census sole ethnicity to death registration form ratio, eg, for Mäori 0-14 years 1.91 = 183/96. As such, it is the correction factor to apply to 1991-1994 ethnic and age-specific mortality rates calculated using sole census ethnicity as the denominator. Unless indicated with an asterix(es), the true ratio (calculated without random rounding due to Statistics New Zealand protocol) was within plus or minus 0.05 of the given ratio. †Due to Statistics New Zealand random rounding protocol, the true ratio may be as much as 0.1 above or below the given ratio. ‡The true ratio may be as much as 0.2 above or below the given ratio.

census sole to mortality ratio being 1.14 and 1.17 for the Northern and Midland RHAs, but notably greater at 1.81 and 2.02 for the Central and Southern RHAs (Table 3). There was no regional variation among Pacific decedents.

The numerator-denominator bias possibly varied by small area socio-economic deprivation for Mäori, with the ratio being 1.48 among the four least deprived deciles, 1.19 for deciles 5 and 6, 1.25 for deciles 7 and 8, and 1.31 for the two most deprived deciles (see Table 5 of reference[12]). However, this suggestion of a greater numerator-denominator bias among the least deprived small areas must be treated cautiously; few Mäori deaths occurred in the least deprived small areas, and the ratio for deciles 1 to 6 combined was 1.30 – little different from the two remaining deprivation groups. There was no apparent trend for Pacific decedents.

The numerator-denominator bias did not vary much by sex, being 1.31 and 1.27 for sole-Mäori males and females respectively, and 1.70 and 1.66 for sole-Pacific. Not presented in this paper are the results for numerator-denominator bias at two-levels of stratification (eg, age by RHA), and using the National Health Index file ethnicity (rather than the NMDS death registration form ethnicity). These more detailed results are published in a technical report.[12] Of note, though, the differential numerator-denominator bias by age persisted within regions, and vice versa.

Discussion

This paper has confirmed a substantial numerator-denominator bias between mortality and census data in the early 1990s, even using the recommended[5] census sole ethnic group as the denominator. Overall in the three years following 1991 census night 29% more decedents had self-identified as sole-Mäori on the census compared to those recorded as Mäori on the mortality data. Using the census-prioritised ethnicity, 46% more decedents had self-identified as Mäori on the census. For Pacific people the bias was worse again, being 68% and 78% for sole and prioritised ethnicity, respectively. Thus, using routine mortality and census data for the 1991-94 period, Mäori and Pacific mortality rates are substantially underestimated.

Importantly, the numerator-denominator bias was particularly severe for young Mäori and for Mäori living in central and southern regions. This is consistent with an earlier comparison of ethnic group between death registrations and the National Health Index (NHI) during 1992 to June 1995. (The NHI better approximated self-identified ethnicity.) This study found that overall 35% of the deaths classified as Mäori on the NHI were coded as non-Mäori on death registrations. This undercount was higher among the young being 44% in those under 50 years compared to 33% in those over 50. The Northern and Midland RHAs had an undercount of 28% and 26%, respectively, compared to 57% in the Central RHA and 60% in the Southern RHA.[7]

Child mortality illustrates the impact of this numerator-denominator bias. If the observed relative risk of mortality comparing Mäori to non-Mäori non-Pacific 0-14 year olds was 1.2 using routine data for the 1991-94 period, our results suggest the relative risk was actually 2.8. (Calculated from the adjustment ratios of 1.91 and 0.83 for Mäori and non-Mäori Non-Pacific 0-14 year olds, respectively shown in Table 2, where 2.8 = 1.2 x [1.91/0.83].) This large numerator-denominator bias among children explains why the relative risk of mortality among Mäori children compared to non-Mäori using routine data has been curiously (and unexpectedly) low in the early 1990s.[5]

We believe our results provide a reasonably accurate quantification of the numerator-denominator bias for the 1991-94 time period. However, there may still be some inaccuracies. First, as the record linkage of census and mortality records was anonymous and probabilistic, some of the links would not have been for the same person. For the *total* set of 31 635 linked census and mortality records in the NZCMS, the percentage of links that were false links has been estimated at 2-3%.[11] However, for the subset of 22 578 links used in this study the percentage of false links was considerably lower, resulting in a negligible source of inaccuracy.

Table 3. Number of deaths stratified by regional health authority by: death registration form ethnicity, census sole ethnicity, and census prioritised ethnicity.

RHA	Ethnic group	Death reg form	Census sole	Census prioritised	Census sole to death reg ratio
Northern	Mäori	1284	1464	1647	1.14
	non-M non-P	10 635	10 134	9915	0.95
	Total	12 390	12 390	12 390	
Midland	Mäori	1512	1767	1929	1.17
	non-M non-P	7218	6945	6777	0.96
	Total	8775	8775	8775	
Central	Mäori	543	984	1137	1.81
	non-M non-P	9834	9336	9168	0.95
	Total	10 518	10 518	10 518	
Southern	Mäori	132	267	360	2.02
	non-M non-P	9495	9309	9213	0.98
	Total	9630	9630	9630	

All numbers in table are weighted, and then random rounded to a multiple of three as per Statistics New Zealand protocol. The true ratio (calculated without random rounding due to Statistics New Zealand protocol) was within plus or minus 0.05 of the given ratio. RHA = regional health authority.

Second, there was potential for selection bias in our estimates of numerator-denominator bias, particularly with the lower percentage of Mäori and Pacific mortality records included in the linked data-set. To mitigate against this we conducted *weighted* analyses. Any remaining selection bias would require residual systematic differences in census self-identified ethnicity between those linked and those not linked *within the weighting strata* (ie, within [NMDS ethnicity] by [sex] by [age] by [NZDep91] by [RHA] strata). For example, just as more Mäori (based on NMDS ethnicity) were excluded from the linked data-set than non-Mäori non-Pacific, it is possible that even within strata of NMDS ethnicity (and age by sex by NZDep91 by RHA) that decedents who were actually Mäori on census data were under-represented. As the numerator-denominator bias mainly arises due to decedents coded as non-Mäori non-Pacific on NMDS data actually being Mäori (or Pacific) on census data, the magnitude of any residual selection bias is a function of the number of non-Mäori non-Pacific (based on NMDS ethnic group) excluded from the linked data-set. Two different sensitivity analyses about this possibility (published elsewhere[12]) suggest a modest (if any) residual selection bias. For example, the 1.29 overall ratio for sole-Mäori (Table 1) might actually have been as high as 1.31 but almost certainly no higher than 1.35, and the 1.46 overall ratio for prioritised Mäori (Table 1) might actually have been as high as 1.51 but almost certainly no higher than 1.57. Regarding prioritised ethnicity, we note the 80% increase in the total number of Mäori deaths from 1994 to 1996.[1] This also suggests our adjustment ratios for *prioritised* ethnicity might be underestimates, but more definitive comment on this must await repetition of the analyses described in this paper for the 1996 census.

The results from this study are directly applicable to the early 1990s, and possibly provide an approximate picture of the numerator-denominator bias for the 1980s. (The following paper in this issue of the Journal, demonstrates the effect of using the adjustment ratios presented in this paper on mortality rates). However, our findings are not directly generalisable to the late 1990s due to the introduction of compulsory recording of ethnicity on mortality data, the introduction of multiple ethnic groups on mortality data, and the change in the census ethnicity question between 1991 and 1996 census.[2-4,13] Of note, only about 9% of total Mäori deaths in the late 1990s have more than one ethnic group, compared to 48% of the total Mäori ethnic group on the 1996 census – a large difference that is not fully attributable to age. Together, these changes will have altered the amount of numerator-denominator bias. Thus, using 'sole' ethnic group (for both numerator and denominator now) to calculate Mäori mortality rates in the late 1990s undoubtedly *overestimates* the Mäori and Pacific mortality rates. For mortality rates based on 'prioritised' ethnic group, one might expect that they were still somewhat underestimated during the late 1990s.[3] As part of the NZCMS, we are currently linking deaths to the 1981, 1986 and 1996 censuses. When these linkages are complete, we will provide a set of adjustment ratios for the full 20-year period, permitting a more robust comparison over time of Mäori, Pacific and non-Mäori non-Pacific mortality rates.

It is clear that during the early 1990s there were large numerator-denominator biases between census and mortality data that caused both Mäori and Pacific mortality rates to be severely underestimated – even using census sole ethnicity as the denominator. The extent of this underestimation disguised the true extent of the ethnic disparities in mortality. These underestimates were particularly severe for Pacific people, and young Mäori and Mäori living in central and Southern regions. Population-based funding formulas that included region-specific Mäori mortality rates would have particularly disadvantaged central and southern regions. The results from this study should be used to adjust ethnic-specific mortality rates for the early 1990s.

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Summary Statistics New Zealand Security Statement. The (New Zealand Census-Mortality Study) NZCMS is a study of the relationship between socio-economic factors and mortality in New Zealand, based on the integration of anonymised population census data from Statistics New Zealand and mortality data from the New Zealand Health Information Service. The project was approved by Statistics New Zealand as a Data Laboratory project under the Microdata Access Protocols in 1997. The data sets created by the integration process are covered by the Statistics Act and can be used for statistical purposes only. Only approved researchers who have signed Statistics New Zealand's declaration of secrecy can access the integrated data in the Data Laboratory. A full security statement is published at the NZCMS website (http://www.wnmeds.ac.New Zealand/nzcms-info.htm). For further information about confidentiality matters in regard to this study please contact Statistics New Zealand.

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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.

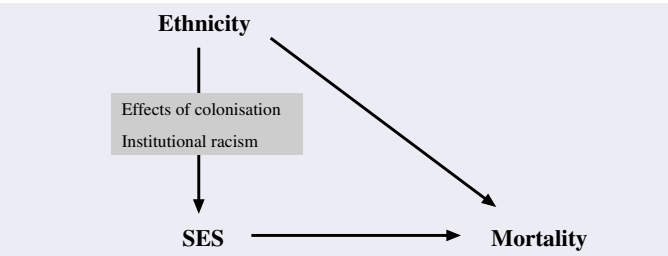

Figure 1. Causal association of ethnicity and socio-economic status (SES).

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[3] – 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	Mäori	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	Mäori	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	Mäori	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	Mäori	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	Mäori	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	Mäori	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-Mäori 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							
Mäori	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							
Mäori	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							
Mäori	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							
Mäori	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							
Mäori	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							
Mäori	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							
Mäori	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							
Mäori	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

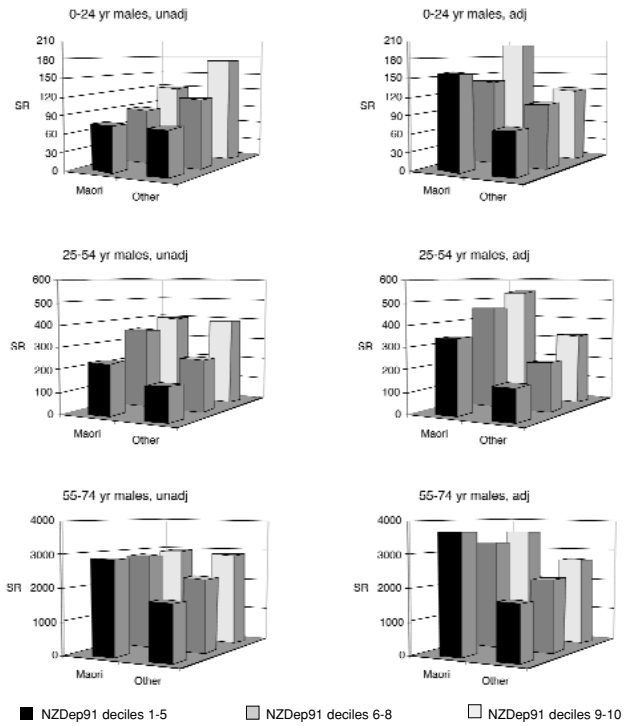

Figure 2b. Females

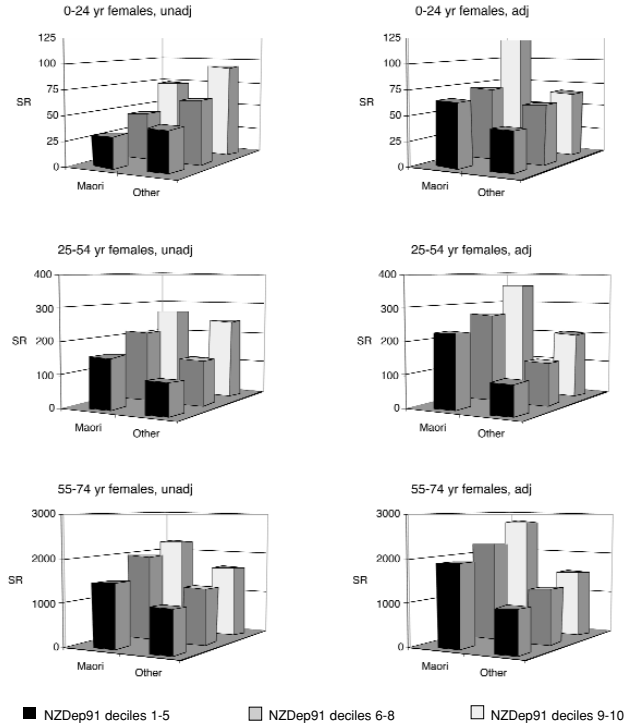

Figure 2. Standardised rates of mortality (per 100 000; deaths during 1991-94) for Mäori and Non-Mäori non-Pacific by small area deprivation (NZDep91), unadjusted and adjusted for numerator-denominator bias.

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		Age and NZDep91 standardised		
		Standardised rate	SRR	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.[2,28,29] 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 -20ºC. 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

Table 1. PSA results from different samples.

Sample Type	Description	No. Tested	PSA Results	Sample Type	Description	No. Tested	PSA
Semen	'normal'	14	+	Breast Milk		8	-
	'vasectomized'	3	+	Rheumatoid factor		3	-
	'oligospermic'	6	+	Vaginal fluid		5	-
	'azoospermic'	1	+	Body fluid mixtures	Pooled semen and blood	5	+
	'antisperm antibody'	2	+		Pooled semen and urine	5	+
Semen Stains	Various storage conditions, 30 days exposure	9	+		Pooled semen and vaginal fluid	3	+
Blood	Male	5	-		Pooled semen and mentrual blood	3	+
	Female	5	-	Condoms containing semen, 7 day incubation	With and without nonoxynol-9	2 types	+
	Menstrual	5	-	Condoms containing no semen, 7 day incubation	Without nonoxynol-9	2	-
Urine	Male	5	-		With nonoxonyl-9	2	+
	Female	5	-	Spermicides	Octoxynol-9	1	-
Saliva	Male	5	-		Nonoxynol-9	1	-
	Female	5	-	Sanitary Products	Pads	10 types	-
Perspiration	Male	5	-				
	Female	5	-		Tampons	2 types	-
Animal Semen	Dog, cat, goat, deer, cattle and horse	6	-	Domestic Contaminants	Various household products	8	-

easily identified as they gave strong positive results to screening tests for semen, but uncharacteristically faint PSA results or negative PSA results. Reporting false negatives was avoided by diluting these samples and re-testing. The PSA results obtained from a range of samples tested with BioSign™ PSA are presented in Table 1.

Low levels of PSA have been detected in male and female blood,[7,8] urine,[9] breast milk,[10] saliva[11] and blood rheumatoid factor.[3] All such samples tested negative with BioSign™ PSA indicating that the extraction protocol and the sensitivity of BioSign™ PSA ensures that low levels of PSA are not detected.

BioSign™ PSA produced expected results in all but one area of testing. The exception was false positive results obtained from semen-free condoms containing the spermicide nonoxynol-9. This anomaly may be due to an interaction between nonoxynol-9 and another component within the condom.

An evaluation of BioSign™ PSA showed that although the ELISA reagents were cheaper than BioSign™ PSA (NZ$4.38 and $15.28 respectively), when labour costs were considered, BioSign™ PSA became the more economical method for laboratories, like forensic laboratories, that have a low throughput of samples. It was at least ten times cheaper to test a single sample for PSA using BioSign™ PSA than by ELISA (NZ$36.95 and $329.38 respectively).

Discussion

The results in this report are part of a more detailed study,[12] and show that BioSign™ PSA is a valuable addition to the forensic laboratory. It is a robust and rapid alternative to the ELISA method of PSA detection for semen identification and also offers sufficient sensitivity in the identification of semen stains. Its relative lack of sensitivity does have a practical benefit in forensic casework as it minimises false positive results from non-semen samples. The cause of the anomalous results obtained from semen-free condoms containing nonoxynol-9 could not be identified. It is advised that BioSign™ PSA is not used to test condoms for semen.

BioSign™ PSA is one of several rapid membrane devices designed to test elevated PSA levels in human serum, as an aid in the diagnosis of prostate cancer. Other PSA membrane test devices have also been investigated for their suitability in forensic laboratories.[13,14] These investigators reported similar findings and also recommended the inclusion of these commercial kits in the forensic testing regime for semen.[13,14] One limitation of BioSign™ PSA is that it can not be used as a sole test for semen. ACP screening and searching for spermatozoa are still necessary steps in the identification of semen stains. Regardless, the inclusion of this clinical test in forensic investigations illustrates how advances in one scientific field can be successfully applied to another.

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Independent Practice Associations in New Zealand: a study of governance structures and process

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Abstract

Aims. To describe the governance structures and processes of four primary care organisations (PCOs) and to evaluate member general practitioners' (GPs) perceptions of the effectiveness of these structures and processes.

Methods. A sample of four PCOs was chosen in 1999, including three independent practitioner associations (IPAs), and a member organisation of CareNet. The chief executive officer of each PCO was interviewed, and the 245 member practitioners were surveyed with a written questionnaire.

Results. The response rate to the GP survey was 78.4%. A two tier governance structure was identifiable for all four PCOs: policy board or steering committee, and working committees. In addition each utilised a peer group review process to provide input to some administrative functions. The CareNet PCO emphasised reduced bureaucracy and a loose administrative structure and process. There was a high level of respondent satisfaction (>90%) with their PCO governance processes.

Conclusions. Governance structures and processes have developed within the four PCOs that reflect the needs of member practitioners and according to the requirements of each district and PCO membership. A high level of member satisfaction with their PCO, its governance and processes, was evident.

NZ med J 2002; 115: 50-2

Over the last decade general practice in New Zealand has experienced major change. Many general practitioners (GPs), who previously worked in relatively isolated practices and partnerships, now belong to independent practice associations (IPAs).[1] IPAs are responsible for a wide range of quality programs and managing an increasing amount of public funding.[2] A diverse range of organisations has evolved providing primary medical care and a range of other related services. These have been termed primary care organisations (PCOs).[2] Judging from recent statements from government regarding policy for primary care,[3] it is likely that PCOs will play an increasingly important role in the organisation and provision of primary care services.

This paper focuses on one aspect of four PCOs—governance. Governance may be thought of as "the function which holds management and the organisation accountable for its actions and which helps provide management with overall strategic direction in guiding the organisation's activities".[4] The related concept of clinical governance has gained currency over the past two years.[5-8] Although definitions of clinical governance are still contested (especially around issues of resource management), the concept has been defined by Malcolm[6] as "the exercise of corporate accountability, both external and internal, for the management of clinical performance throughout a health service organisation". Essentially, clinical governance captures the notion of assurance of, and accountability for, high standards of health care.

Given recent policy statements related to development of primary care infrastructure and governance of PCOs—especially with respect to involvement of community and consumer representatives and different types of health professionals in PCO governance,[3] we thought it timely to study governance arrangements in PCOs, and consider the implications from primary care and policy perspectives.

Methods

The study used a combination of quantitative and qualitative methods. In 1999 a sample of four PCOs was selected to provide a range of sizes, urban and rural settings, and different organisational frameworks. The sample was not intended to be representative of PCOs, but rather to illustrate the types of organisational arrangements that are developing. The sample included two distinct types of PCO—CareNet and IPAs. Two IPAs were urban —Wellington and Manawatu, and the third was a smaller rural IPA—Tararua. They represented 245 GPs providing primary care services to over 360 000 patients.

First, following pre-testing of an interview questionnaire, telephone interviews were carried out with the chief executive officer (CEO) of each PCO. Each interview took from 30-60 minutes; notes were taken during the interview, and immediately written up. Analysis of the four interviews employed cross-case analysis, with a search for patterns or themes within each setting and across cases, and with the grouping together of answers from the different interviewees to common questions.

Second, a survey questionnaire was mailed to the 245 GPs. Re-mailing of the survey instrument with a covering letter was repeated three times in order to obtain an acceptable final response rate. The postal questionnaires were returned by mail, and data were entered into a Microsoft Access database. Data were then exported into EpiInfo version 6, and analysed. Simple descriptive statistics are reported here. A coding and transcription process was used to analyse the survey open questions.

Results

Membership of the organisations was as follows: Tararua IPA, seven GPs; Wellington IPA, 104 GPs; Manawatu IPA, 73 GPs; Nelson CareNet, 61 GPs.

Governance structures and processes. Each organisation contained two tiers of governance structure, a policy tier and a committee tier. Each utilised a peer group review process to provide input to some administrative functions.

Policy tier. All three IPAs were governed by an elected board of GP directors. Elections occurred annually and all IPA members were eligible to vote. Policy was defined by board members and implemented by the CEO. The Wellington and Manawatu IPA boards met monthly; the Tararua IPA board six-weekly. The number of board members varied: six, Wellington IPA; seven, Manawatu IPA; four, Tararua IPA. Nelson CareNet was structured differently and was directed by a GP co-ordinator (who carried out the role of CEO) and steering committee of seven GPs.

Four characteristic features existed for policy-making within Wellington and Manawatu IPAs. The boards had the month by month policy function of determining IPA policy. Working committees, each with a board representative, produced reports and recommendations to the IPA board. Special general or regional meetings formed a basis for strategic planning and policy development on IPA matters of special concern. Wellington and Manawatu members

were frequently canvassed for their views, comment or signature vote on policy recommendations. Policy-making in the smaller rural Tararua IPA was the responsibility of the board of four directors. However, it was characterised by an ongoing dynamic of accountability between the directors and the peer review group, until consensus could be achieved.

Nelson CareNet was led by a GP co-ordinator, who shouldered day to day management and worked in consultation with a steering group of seven GPs. Members of the steering group were self-appointed, on the basis of professional interest, energy and commitment. The co-ordinator was funded by the HFA and worked part-time from a home office. Communication with members was via a two-weekly newsletter produced by the co-ordinator, and regular e-mail communication with 25 members. Nelson CareNet policy development was born out of the steering committee and co-ordinator. The Nelson CareNet steering committee met three to four monthly. Special general meetings could be called to formulate policy when necessary.

Committee tier. A large tier of working committees was a feature of Wellington IPA governance processes, and to a lesser degree in the Manawatu IPA. These working committees supported and informed the governance function of the IPA boards. Wellington IPA had four committees of 4-8 members: an information technology committee; a quality assurance and consumers' committee; a pharmacy committee; and a laboratory committee. Manawatu IPA had two working committees with membership from 6-8 GPs: a pharmacy committee and a laboratory committee. These committees were serviced by IPA administrative staff and met monthly or two-monthly.

Both Wellington and Manawatu IPAs had representation on six or more multi-disciplinary Hospital and Health Service committees to address service delivery and clinical guidelines, for example for diabetes and asthma.

The work of the Tararua IPA board was supported by a committee including all IPA members. The group served three functions: a forum for the board of directors to discuss IPA issues; a peer review group; and a forum for the development of clinical guidelines.

Nelson CareNet had one working committee consisting of two GPs and a practice nurse, whose function was to improve immunisation rates within Nelson CareNet. None of the four PCOs had a committee that dealt with radiology and echocardiography.

Peer review group tier. All four PCOs had active peer review groups, originally founded by the Royal New Zealand College of General Practitioners (RNZCGP). Three functions were evident across all four PCOs: peer review, education and accreditation; colleagial and social interaction; and PCO administrative purposes e.g. the development or pre-testing of clinical guidelines.

Allocation of savings. Some PCOs had achieved financial 'savings' from year to year, by for example more efficient use of laboratory tests. Savings achieved by the three IPAs were allocated to a variety of initiatives including the technical development of services, for example tympanometry and audiology screening, and new health care projects such as diabetic retinopathy screening.

GP survey questionnaire. The initial response rate was 50.6% (124/245). Re-mailing three times, resulted in a response rate for all four PCOs of 78.4% (Tararua 85.7% (6/7); Manawatu 76.7% (56/73); Wellington 81.7% (85/104); Nelson 73.8% (45/61)).

Process issues. GPs had joined their IPAs for a number of reasons. Respondents were asked to select from six options reasons for their joining their PCO Table 1.

Table 1. Reasons for joining PCO.

Reason for joining IPA	Number
Negotiation with HFA	111
Secure HFA funding	76
Keep with colleagues	104
Improved quality of practice	81
No better alternative	50
Other	36

There was regular communication between the PCOs and member GPs on PCO matters. Over 55% (106/192) of respondents communicated with their PCO at a monthly frequency and 14% (28/192) communicated weekly.

Evaluation of PCOs. Members were asked how satisfied they were with the overall performance of their PCO. 82.3% (158/192) had no dissatisfactions. Concerns of the 7.3% (20/192) dissatisfied members were centred around the tendency of PCOs to develop their own bureaucracy, and compliance problems.

Respondents were asked if they had benefited financially from membership of their PCO. 25% (48/192) replied that their practice had benefited from PCO membership. Ways in which the practice might have benefited were: "managed care frees me for clinical work"; "negotiation as a group is cost saving"; through funding of new clinical services contracts (eg, sexual health, maternity, and mental health contracts Wellington IPA); IPA related professional payments (eg, meetings); through sustained HFA funding of general medical services subsidy; funding of technical developments (e.g. information technology, communication technology, tympanometers, spirometer); funding of professional development of nurses.

Discussion

This study describes the governance structures and operational processes of new organisations that have developed in primary care over the last few years. They represent a significant departure from the previous 'cottage industry' approach of general practice. It is clear that they have already developed an infrastructure and governance that enables them to carry out a range of complex coordination and purchasing procedures. With large memberships, and urban location, numerous working committees had developed to carry out these functions in the larger IPAs. These committees addressed various health service issues. They were usually serviced by PCO administrative staff, and had active links with the PCO board. A new feature in these urban IPAs was the development of multidisciplinary working committees collaborating with secondary care health professionals. The development of committee structures in organisations that have grown rapidly could help to explain the concern of some of the doctors that the IPA's were themselves becoming bureaucratic in nature.

Although the initial reasons for joining a PCO were economic, the IPAs had achieved high satisfaction levels from their members for the activities they undertook.

The creation of the PCOs has led to significant changes in working style and practice, with, for example, increased communication with outside organisations by GPs (eg, with other PCOs, hospital committees and funding authorities). Over 55% of practitioner respondents communicated with their PCO at least monthly and nearly 15% communicated at least weekly. The repayment for this time investment can be seen in an accountability structure that can be successfully used for contracting and the subsequent increase in new services for patients.

Collaboration between doctors has been identified as a hallmark feature of PCO process.[1,9,10] Collaboration was observable on a variety of levels. The sustained function of PCO boards and working committees required collaboration from their members. Representation from board to working committees and back again required an ethic of collaboration. Multidisciplinary committees were pioneering the way to integrated care with secondary care services, especially within the two larger urban IPAs. These were examples of collaboration operating across health disciplines and across primary and secondary health sectors. Peer review groups were examples of collaboration in action—where practitioners sought to learn from their peers and to improve their professional skills.

We revealed a variety of fora and processes common to all four PCOs. The key policy group was the PCO board or steering group. The PCOs at the time of this survey remained GP run and led organisations, with little official consumer or community involvement. Since the publication and directive of the primary care strategy[3] many IPAs have made efforts to increase their degree of consumer representation at board and committee level.

The development of IPAs has occurred in parallel with the growth of third sector organisations (non-profit non-government organisations such as union clinics) in New Zealand, and infrastructural developments in other countries such as the UK and Australia. In most New Zealand third sector organisations, patients and community representatives sit on the board of management, with community representation from trade unions, iwi or hapu groups, community organisations or other primary care providers.[11]

In most New Zealand PCOs, activities for clinical governance such as audit and peer groups have largely become the responsibility of the PCO, together with the RNZCGP. These organisational and governance structures have a parallel in the emergence of Divisions of General Practice in Australia[12] and Primary Care Commissioning groups (PCCG) in the UK.[13] While Malcolm has heralded New Zealand's IPAs as a working model for clinical governance in United Kingdom and elsewhere,[14] it remains unclear how they can most effectively contribute to health care. Moreover morale is low in general practice and by international comparison primary health care in New Zealand has relatively low levels of funding.[15]

In conclusion, governance structures and processes have developed within the four PCOs that reflect the needs of member practitioners and according to the requirements of each district and PCO membership. A high level of member satisfaction with their PCO, its governance and processes, was evident.

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CASE REPORT

Push enteroscopy: a new service for New Zealand

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Introduction

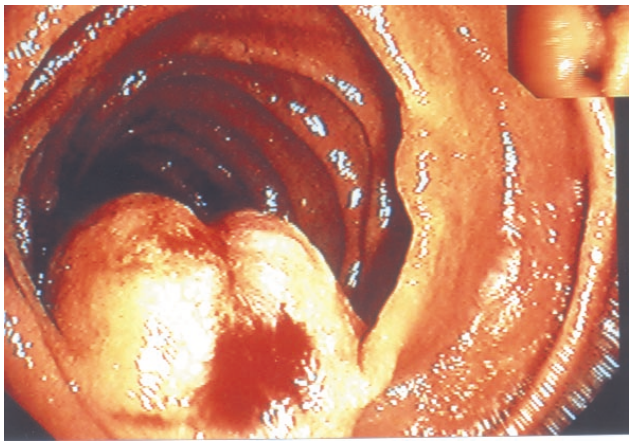
A 61 year old man with an eighteen year history of recurrent gastrointestinal (GI) bleeding was referred for push enteroscopy, a service recently introduced into New Zealand. This case highlights the clinical usefulness of the procedure.

Case report

The patient presented initially in 1982 with melaena (Hb 56g/L). Upper GI endoscopy showed only a hiatus hernia. Subsequently he had a normal barium meal and barium enema. He next presented in 1991 with further melaena, but reported three episodes of transient black stools since 1982. Isotope red cell study or angiography were suggested if he bled again. In 2000 he consulted a private gastroenterologist for investigation of iron-deficient anaemia (Hb 101g/L), with strongly positive faecal occult blood. Repeat endoscopy confirmed the hiatus hernia. Ileo-colonoscopy showed no evidence of blood. Ten days later he was admitted to hospital with symptomatic anaemia, and was referred for push enteroscopy. In the proximal jejunum a 4cm submucosal tumour was demonstrated with surface ulceration (Figure 1) and contact bleeding. Biopsies showed only normal mucosa. He underwent elective laparotomy and resection of the lesion. The remainder of the small bowel was normal. Histological and immunohistochemical features were consistent with a benign gastrointestinal stromal tumour. Follow-up fourteen months later revealed the patient to be completely well. Haemoglobin and iron studies are stable.

Discussion

Push enteroscopy, introduced to New Zealand at Auckland Hospital last year, allows direct visualisation of the small bowel mucosa as far as the mid-jejunum. In addition, this procedure offers therapeutic options such as coagulation of vascular lesions, tissue biopsy, and mucosal 'tattooing' to aid surgical localisation of lesions.



GI bleeding with no apparent cause, even after extensive investigation, is termed "GI bleeding of obscure origin". The commonest cause is small bowel angiodysplasia, accounting for 80% of cases.[1] Small bowel tumours are the second most common cause in all patients, and the commonest cause in those younger than 50 years.[2] The type of blood loss (frank bleeding or occult) is not an effective means of differentiating between angiodysplasia and small bowel tumour.[2] Causes of chronic bleeding from the small intestine can reasonably be classified by age. In patients under 25 years, think Meckel's diverticulum. Patients between 30-50 years, suspect a small bowel tumour.[2] In patients over 50 years, angiodysplasia is the commonest cause.[2]

Push enteroscopy can be performed on any routine endoscopy list, although fluoroscopy is often used to assess depth of intubation. Push enteroscopy takes considerably longer than standard gastroscopy (a typical procedure lasts 30-50 minutes), but is performed using similar intravenous sedation (typically an opioid/midazolam combination). A separate, flexible overtube can be advanced through the pylorus to reduce looping within the stomach. Although use of an overtube has been shown to increase depth of enteroscope insertion, this has not been shown to increase pathological findings.[3] Risks of push enteroscopy are similar to standard endoscopic procedures, with therapeutic procedures carrying a slightly higher risk of complications compared to diagnostic procedures. Potential complications are rare but, similar to other intraluminal endoscopic procedures, include bleeding and perforation.

At the time of writing, wireless capsule endoscopy has become available in New Zealand. This new technology allows painless imaging of the entire small bowel[4] but has no therapeutic capabilities. Although a potentially exciting diagnostic tool, it remains to be seen where it will fit into GI investigation strategies, and the comparative cost is likely to limit its use to tertiary referral centres. Because of its proven performance, therapeutic capabilities, and relative cost-effectiveness, push enteroscopy remains the preferred initial choice for investigation of GI bleeding of obscure origin.

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VIEWPOINTS

Post-market stress syndrome

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Soon after the election of the Labour-Alliance government in November 1999 the first indication of a significant change in the direction of health policy occurred. The then largest United States based Health Maintenance Organisation Aetna concluded, on the basis of what was known about Labour-Alliance health policies, that New Zealand was no longer a market likely to produce the profit returns they desired so they withdrew.

Lessons of the 1990s market experiment

In 1993 our publicly-provided health system was based on area health boards which were replaced by crown health enterprises (CHEs) and subsequently by hospital and health services. These were more narrowly based, focussing on secondary and tertiary care, and governed by state-owned companies operating, for the first time, under the Commerce and Companies Acts. Commercial competition was now the core of the system that was to drive its future direction. CHEs were to compete rather than cooperate with each other and also to compete with the private sector. Owing to its relative small size, the private sector required a boost in order to establish a 'level playing field' for business competition. This included the ill-fated cumbersome and inefficient attempt at user charges for public hospitals.

Services were put up for contestable bidding with strong suggestions of ideological favouritism towards the private sector. Although it did not eventuate, general practitioners (GPs) were encouraged to fund-hold in order to purchase secondary services. Privatisation encroached upon the public system at least around the margins. Clinical coordination and integration between primary and secondary care became entangled with privatisation masked under the language of first 'managed care' and then 'integrated care'.

If a country wants to ensure universal and comprehensive health services of good quality, it requires, in order of priority:

1. A single public based funding system. The more fragmented the funding sources the more fragmented and

less integrated the delivery and organisation of health services.
2. Structural capacity for universal public provision.

The market experiment attempted to introduce competition and consequently fragmentation at the second, rather than first (and more important), pillar of a relatively integrated public health system. The closest it got was the short-lived formation of four regional funding authorities and the unbundling of ACC funding for public hospital. The original design for alternative health care plans that would have helped introduce competition to funding were put on hold because of complexity. Consequently the inroads it was hoped competition would make were more limited and, despite all the difficulties and disruption, the integrity of the publicly provided health system largely remained intact.

It was inevitable that this ideological experiment would fail. Markets thrive on short-term unpredictability whereas public good provision thrives on longer-term predictability. Competition in providing was limited to the margins and even more so in funding. It also came into conflict with the cooperative ethos of health professionals and the wider public. Further, it was compounded by under-funding that, in effect, assisted the creation of a 'level playing field' for the private sector and privatisation capacity. In the first half of the decade, public hospitals were seriously under-funded in relation to the costs of running them and maintaining services. In real per capita terms, funding fell by around 13% according to the Health Ministry's Income and Expenditure Trends. Coupled with the unpopular ideology, out-of-control CHE deficits and the embarrassment of increasing waiting list statistics, public reaction was strong. Threats to access and the viability of valued services led to public outcry inclusive of large uncoordinated demonstrations in the middle of the decade.

The result was inevitably political. The language of competition was dropped and was replaced with that of cooperation, although the underpinning legislation, including Commerce Act coverage that promoted competition and condemned so-called 'anti-competitive' practices, was fundamentally unchanged. Funding increases in the rest of the decade compensated largely in dollar terms for the preceding under-funding. But there were two important constraints:

1. Public hospitals cannot run like hirepool companies. Damage to infrastructure, resource, intellectual capital and morale cannot be restored overnight.
2. The additional funding was disproportionately time-limited and linked to waiting times initiatives. In mental health, the new funding was largely linked to the expansion of new services rather than the maintenance of existing services.

Thus by the end of the last decade and up until the last general election we had a public health system that was betwixt and between. It was an inefficient, direction-less hybrid governed by legislation promoting competition on the one hand and managed by policy statements promoting cooperation on the other.

Need for change

Change was needed to address the following:

1. An underpinning legislative framework aligned with the ethos and values of health professionals and the public.
2. A capacity of workforce development and planning that had been neglected during a decade of lost opportunity because of the false ideological belief that markets would sort things out.
3. In a country of less than four million people how could we justify two central government agencies, the Ministry of Health and Health Funding Authority (HFA), competing against each other for the ear of the Health Minister?
4. Effectiveness was undermined by a high level of distrust and at times disrespect between those responsible for policy advice and implementation and those responsible for providing health services.
5. The straitjacket of privatisation constrained the capacity for greater primary-secondary care coordination and integration.
6. The preoccupation of 'working to contract' conflicted with the professional approach of 'going the extra mile'.
7. Insufficient effective long-term planning over funding needs for both primary and secondary care.

Not to put too fine a point on it, change was needed because, no matter how hard one tries to buff it, one can never get a turd to shine.

Immediate problems and challenges

Certainly much has changed since the election of the Labour-Alliance government. With removal of coverage of the Commerce and Companies Acts, our legislative framework is now better aligned with public and health professional values. There is now only one central government health bureaucracy. Workforce development and planning is no longer ideologically frowned upon with positive moves being made in this direction, and primary-secondary coordination can now proceed without the straitjacket of privatisation.

But health remains in the media headlines. Partly this is because of legacies of the past decade and chickens coming home to roost. It is not easy to turn around the damage of a misplaced decade within two years. But some of it also had to do with the performance of a government that has grasped some issues well but others no so well. The actual doing of policy implementation is both much more exciting but also much harder than its advocacy, at least in part due to a series of connected factors including:

1. Lack of rigorous involvement and engagement between government and representative bodies of health professionals well placed to assess the pulse of the health sector. The government is certainly consultative and open but needs to advance further to greater inter-active engagement at a level comparable to that of government-Ministry interaction.
2. Lack of confidence, whether justified or not, by district health boards (DHBs) in the performance of the Ministry in negotiating with them and in advising the Minister.
3. At times less than rigorous, and even poor advice to government on particular issues including adequate funding levels and privatisation proposals.
4. Excessive expectations over the ability of boards to provide positive leadership, confusing practical reality with lines on paper. Boards are not the apex of the system and effective change and advancement will not be delivered by top-down hierarchical structures.

Two key issues among many

Arising out of this situation the government is facing serious problems, all of which are resolvable but require will and direction. The new direction has yet to address the disruptive deficiencies of the past decade. The longer these remain unresolved the more they compound. Two key issues, among several others, are funding and bureaucracy.

Funding. Our public health system has been systemically under-funded and under-capitalised for at least a decade. The Budget (2001-02) had some positives such as additional monies for new mental health services and converting time-

limited funding for electives into permanent baseline funding. But apart from a large component for services that were demand-driven and demand-paid anyway (mainly primary care and to a lesser extent pharmaceuticals), this was as good as it got. For the maintenance of existing public hospital services, once the mirrors were removed, the actual funding increase was a mere $900 000. In other words, an increase of around 0.03% to maintain existing public hospital services when their running costs are expected to increase by around 3.2%. Now that DHBs are also responsible for primary care the scope for cost shifting is minimised.

This leaves two alternatives – reduction of services or increasing deficits. The latter is the most likely and, despite the poor economics that underpin it, the lesser of two evils. This is exactly the situation that the former government faced towards the end of the last decade. With deficits nationally well over $220m they got round it through creative accounting known as the 'deficit switch' in which the debt was transferred centrally to the HFA. This government will have to consider a similar approach if it wants to avoid reductions to the range and quality of patient services. Either another 'deficit switch' or an injection of equity funding can do this.

The government has repeated a fundamental error of its predecessors. It has restructured and under-funded at the same time; exactly what happened when first area health boards and then CHEs were introduced.

Bureaucracy and transition costs. Excessive bureaucracy and transaction costs were a major criticism of the market experiment of the 1990s including by the current government coalition parties when in opposition – and for good reason. One of the initial criticisms of the new DHB system was that it would lead to 21 different health services. This view lacked precision and was too sweeping, particularly given that DHBs would be governed by a binding national health strategy and ministerial-approved annual plans.

There is, however, a problem of a different nature largely due to the fact that so many of those that advise government and manage the system were trained in the ideology of the 1990s. This included the belief that there was a fundamental tension and conflict between funding and providing. Consequently we had to have what was called the 'funder-provider split'. This view continues to prevail and now many DHBs are replicating the funder-provider split that used to exist on a national level up to 21 different times. While there may be some tension between funding and providing, it is more of role demarcation and operational good 'house-keeping' than conflict of interest. Its significance is exaggerated and is less important than, for example, relationships between different health professional groups, between health professionals and managers, and between DHBs and central government. The effect of over-stating its significance is to increase bureaucracy and transaction costs by creating provider and funding arms with artificially generated walls between them.

If the government is going to achieve its highly laudable objectives, it needs to ensure that the part of the health sector that is most in tune with and aligned to its objectives (the health professionals) are actively engaged at a much higher level than is currently the case. The gearbox of involvement must go well beyond formal and accessible consultation to a new higher gear of active and regular engagement on both macro and micro issues.

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This article is derived from a much longer address, The Direction of the New Zealand Public Health System or How Many Surrealists does it take to Change a Light Bulb, given to the New Zealand Society of Hospital and Community Dentistry Conference, Wellington, 27 July, 2001 <www.asms.org.nz>.

Patients' rights in the United States: from 'down-under' the situation seems upside-down

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In June-July 2001, the US Senate debated and passed a 'patients' bill of rights'. While deemed 'bipartisan' legislation, this largely reflected the demands of the Democratic majority in the upper house. In August 2001, Congress then endorsed its own patients' rights legislation, but this more closely reflected the desires of the White House and Republican party. Attempts to establish a US patients' bill of rights have been laboured. For around five years, different proposals for patients' rights protections have moved between Senate and Congress.[1] Each time their passage has been thwarted. This has been due to the peculiar nature of the US political system, designed to prohibit legislative enactment without considerable struggle and compromise, and the differing perceptions of the politicians and legislative coalitions whose holds on power have varied. Although both houses and the White House now concur that a patients' rights bill is needed, whether all three can agree to a common piece of legislation remains to be seen. Whatever the outcome, for someone from 'down-under', the situation in the US seems 'upside-down'.

Despite the differing opinions over what a patients' rights bill should embody, the basic premise remains that the majority of Americans have few protections or sources of redress when it comes to health care. Today, in the world's most complicated and unfathomable health system, patients - and doctors - have come to take second place to financial decisionmakers. The root of the problem is a system where health care has become dominated by insurance plans. Generally, plans are operated by private, profit-driven companies functioning on a capitation basis providing circumscribed packages of benefits to subscribing customers. The 'customers' are largely employers who offer health insurance as a salary component. Around 190 million Americans have their health care provided via some sort of health plan.

There are pointed differences between much of the developed world and the US in terms of how health services are developed and delivered. Elsewhere, 'gatekeeping' is the realm of doctors, frequently working alongside governments and health managers to develop mechanisms for patient and

service prioritisation.[2] In the US, health plans tend to set the 'limits' to service provision: before treatment commences funder approval must be obtained. This has spawned numerous problems. First, many patients do not receive medically necessary care due to plans denying funding. Second, doctors have become insurers' agents, often responsible for withholding services or failing to give patients complete information about their conditions or other (more costly) diagnostic alternatives. Third, patients are undoubtedly the losers in the system, whether it is the inability to request an independent review of a service-denial decision, or the prospect of a tremendous personal expense if a non-plan provider provides unapproved services (eg, at the scene of an emergency).

The patients' rights bill is intended to put some 'balance' into the US health system and give health plans their comeuppance. A recent poll showed that only 15% of Americans had any faith in their plans and, by implication, the health care system.[3] Other research shows similarly low levels of confidence.[4] There is political agreement that patients' rights legislation should provide rights to external review of health plan decisions, guarantee access to specialists, doctors of choice and emergency care at the nearest facility, and remove 'gag clauses' from insurers' standing orders which restrict communications between doctors and patients. Senate and Congress remain divided over the limits to damages awardable for pain and suffering which result from denial of treatment, whether lawsuits should be heard in state or federal courts, and the liability of employers who facilitate plan decisions.

Opponents of patients' rights legislation argue that it will inevitably drive up costs through litigation and increased health care delivery which, presumably, will be delivered based on fairer assessments of genuine need. Rising costs, they say, will increase the numbers of uninsured Americans as insurers and employers pull out of health care. While there is little evidence of increasing litigation or costs in states, such as Texas, that already have patients' rights legislation, the levels of compensation seem excessive. The limit will be at least $US500 000 and could be as high as $US5m. There are arguments, also, that the 'freedom' of Americans will be further eroded through the introduction of yet another piece of federal law and the restrictions on the activities of 'the market' this will provoke. Yet, in terms of access to health care, only those with the capacity to pay and an intensive knowledge of this highly complex health system have freedom to choose.

There remain fundamental issues with the US health system that a patients' bill of rights cannot address. Guidelines and standards for service delivery are lacking, as are measures for calculating optimal funding levels. Consequently, the health 'market' overserves many, while a vast number of US citizens are underserved.[5] Following developments in the UK and New Zealand, with careful organisation, primary care doctors could play a vital role in the US, in managing, monitoring and integrating the care of patients, if given the political and financial backing to do so.[6,7] The notion of trust in medicine could also be nurtured so that patients grow to be comfortable in the care and advice they are being offered.[8] A patients' bill of rights unfortunately reinforces existing institutions by building on the 'us and them' mindset characteristic of markets and American culture, and moves further away from what someone from 'down-under' might see as a system designed to deliver appropriate and cost-effective health care.

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Applications to US medical schools fall for fourth year

Applications to medical schools in the United States have declined for the fourth consecutive year, according to a survey sponsored by the Liaison Committee on Medical Education. The committee's questionnaire was designed to assess the state of medical school education and was completed last year by all 125 medical schools in the United States.

Applications have plummeted since reaching a high of 47 000 in 1996. However, they are still well above the low reached in 1988, when only 27 000 students applied for admission. Nevertheless, many schools are worried that a trend in falling applications has been established. In the year 2000 there were 37 092 applications for the first year class and 17 538 acceptances.

The number of applicants shrank by 3.7% from 1999. Of these applicants, 17 274 were women, a 0.9% drop from 1999. But the percentage of women entering the first year of medical school has remained essentially the same, at 46%.

In 2000 the ratio of overall applications to acceptances was slightly greater than 2 to 1, and the grade point averages and scores in the admission tests were virtually identical to those of entering students in 1999 (JAMA 2001; 286: 1049-55).

Among the reasons cited for the decline are the drop in doctors' income, reduced autonomy spawned by managed care, a perceived loss of prestige in the profession, and the long period of training, coupled with the large debt incurred by many students.

Medical education in the United States is long and expensive: four years of undergraduate education followed by four years of medical school and then 3-7 years of postgraduate residency training, depending on the specialty.

Medical students who finance their education through student loans incur an average debt on the loan of $80 000-120 000 (£55 000-82 000). Undergraduates considering medical school may instead choose more lucrative careers, such as in business or the technology sector.

Deborah Josefson. BMJ 2001; 323: 592.