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THE NEW ZEALAND MEDICAL JOURNAL

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EDITORIALS

And now Auckland

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On December 19th 2001, a letter signed by over 150 specialist doctors was sent to the Chairman of the Auckland District Health Board (ADHB). It expressed a loss of confidence in the Board's Chief Executive Officer (CEO), and his executive. For many of the signatories this letter was an expression of real anger. For years, specialist doctors in New Zealand have struggled in their efforts to provide high quality healthcare to patients in an environment where, increasingly, the frustrations of constrained resources have been exacerbated by conflict with the corporate culture of management. Since the imposition of general management into healthcare, the traditional focus on providing the best possible treatment to patients has been blurred by imposed theories from profit-driven commercial organisations. These theories fit uncomfortably into the culture of public hospitals, whose purpose is to care for the ill. Current deficits suggest they have not even proved efficacious in containing costs.

This outbreak of anger is not an isolated event but, as must be patently obvious to any reader of the Journal,[1-5] it is symptomatic of a widespread and serious malady. The editorial of 27th April 2001, which deals with the report of the Health and Disability Commissioner on Gisborne Hospital (a report described as echoing his predecessor's findings in respect of Christchurch Hospital) could be applied unchanged to the present difficulties faced by ADHB.[2] Unfortunately it seems that the message is not getting through.

At the heart of the problem lies a fundamental premise that is simply incorrect. This holds that the running of a complex organisation requires no specific expertise in its core business. Furthermore, no need is seen for staying in one position long enough to gain a real understanding of the issues, or to see the downstream consequences of decisions. In a matter as complex as healthcare, this premise is nonsense.

In healthcare management (whether in a funding organisation or in a hospital), five years is a long time in one job. In contrast, for most hospital specialists it is less than the time spent in postgraduate training. Continuous change has meant that enormous effort expended by doctors, nurses and other operational staff in documenting the case for particular services or facilities, and in developing relationships of trust with managers, simply goes to waste. Time and time again clinicians have to start from scratch with explanations of how their units work to new managers.

ADHB's important Hospital Services Development Plan (HSDP) will guide the building of the new hospital on the Grafton site, and the subsequent relocation and reorganisation of clinical services. Many aspects of this complex and challenging project have been the subject of extensive and time-consuming consultation, and clinical staff at all levels have engaged in the process in addition to their arduous normal duties. All too frequently, as the unfortunate J Alfred Prufrock observed. "There is time for decisions and revisions which a minute will reverse."[6] The relocating of the Paediatric Cardiac Surgical Unit to within the Children's Hospital is a good example. This is a matter on which clinical advice has been entirely consistent, and in line with the recommendations of the Bristol Inquiry[7] following a spate of excess paediatric deaths: very sick children should be treated in a specialist paediatric hsopital. For years, clinicians involved with successive plans to achieve this end have had to contend with repeated requests to revisit old ground. It seems that no decision is worth the paper on which it has been signed off. Even at this late stage clinicians have again been asked to accept substantial compromises in this aspect of HSDP, including the possibility of leaving the paediatric cardiac service within the adult facility. If a proper solution cannot be afforded this situation should be explained, explicitly, to the public. Endless persistence in seeking clinician's endorsement of second-best is not consultation.

The specific event that precipitated the letter was a decision to 'restructure' the entire management tree. The first step involved the sudden simultaneous disestablishment of the positions of five general managers with whom clinicians had, to varying degrees, established working relationships, and mutual trust. The next round proposes that many important positions involving clinicians with managerial responsibility (eg nurse managers) will also be disestablished. The draft proposal for this stage has been widely circulated, and several hundred submissions have been prepared. Many staff are understandably sceptical that this consultative process is genuine. Whatever the outcome, the proposals themselves have created insecurity, and key personnel, whose jobs *may* be on the line, are seeking alternative employment. If these people go, the valuable asset of their institutional memory will be lost to the organisation.

To remove simultaneously many people intimately familiar with the functioning of the organisation seems a risk in the run up to a major clinical services (under HSDP). In addition, this whole business seems a shabby way of dealing with a group of loyal employees, and particularly ironic in an organisation whose alleged raison d'etre is to care for New Zealanders. How the organisation expects to foster trust in the light of such behaviour is a complete mystery.

The core business of public hospitals is the treatment of sick patients, and this is unertaken primarily by doctors, nurses, and allied health professionals. Healthcare is not a simple business, and in the case of each of these groups,

substantial specific training is required. In each case there is also an element of vocation – a fundamental commitment to healthcare which defines an entire career. Clinicians are there for the long term – they, and their patients, have to live with the consequenes of the decisions made by politicians, elected board members, health funders and senior hospital managers long after most of these individuals have come and gone.

Hospitals need good managers; this is quite a different matter from saying that hospitals should be run by managers. The concept of partnership between managers and clinicians is laudable, but dominance by managers is not partnership. We strongly disagree with the model of an executive, which, with few constraints on autocratic behaviour, arbitrarily imposes policy on its clinical staff regardless of their viewpoint. Rather, we suggest that clinicians, as stable, uniquely qualified, key members of a publicly funded organisation should be supported by management to provide healthcare for patients – a very different model indeed.

The latter view can be strongly supported on the basis of one simple fact. However fickle the changing ideology of government, however constrained the resource, however challenging the need to maximise the return on funds, senior clinicians, collectively, are likely to do a better job for New Zealand's patients than generic managers. In particular, a key element in obtaining the best return on limited funds clearly lies with doctors, who, on account of their training and qualifications, direct the clinical management of patients. The clinical decisions made by doctors have a huge impact on expenditure.

There are those who would question whether New Zealand can any longer afford excellent healthcare. We disagree. On the contrary, we think New Zealand can't afford to have anything less than excellent healthcare, because the cheapest option is to treat patients properly, and in time. It is demonstrably foolish to save money in a hospital budget by denying treatment to patients who, being left untreated, will predictably return in worse condition, requiring even more expensive treatment.[8] Equally, poor quality treatment will simply lead to financial and social costs of complications and inadequate patient outcomes, which will outweigh any savings made by not providing excellent treatment in the first place.[9] Claims of limited resources ring hollow in the aftermath of the Government's rescue package for Air New Zealand, but, in the end, it simply makes bad business sense to under fund healthcare.

A major factor in achieving good results is the development of well-established clinical units with stable, highly trained, long-term staff at every level. The importance of teamwork was stressed in the report from the Bristol Inquiry.[7] Good results require ongoing commitment to the continual improvement of individual skills, knowledge and ability, and to the functioning of a unit as a whole. It takes years of training and experience to produce a team able to achieve really outstanding results – but in the end this doesn't cost more than doing the same thing badly, in an environment of never-ending instability. Repeated changes to the structure and personnel (including managers) of a clinical unit are particularly destructive. Results depend in no small part on the willingness of staff to go the extra distance. This willingness is promoted by a sense of identity with a highly functional team, built around the needs of patients. Morale is strongly linked to the sense of self-worth and efficacy that arises from belonging to a clinical unit which provides good healthcare for patients. Commitment to fiscal responsibility will also be found at this level, rather than from any concept of corporate loyalty.

We think it is questionable whether any single individual, manager, doctor, or anyone else, should be given substantial autonomy to run a large hospiral, or group of hospitals. The task is too complex, and the conflicts too unmanageable for one person. A more sensible plan (put forward in the letter) would be to entrust decision making to a clinical board. Most, but not all, will be doctors. These unit heads are typically very knowledgeable about healthcare in general, and about their particular organisation. Unlike generic managers, their training and careers have been focussed from the outset on treating paitents. There is no need for anxiety that they would not understand the fiscal constraints of our health service in New Zealand – no clinician who has reached a position of responsibility could fail to be fully cognisant of this problem today. Importantly, they tend to be in touch with their colleagues and to have the support of their staff – including their non-clinical colleagues. And it is this support from ground up that really determines the success of any initiative within a large organisation. It seems to us that in ADHB at least, senior management is losing that support.

Obviously such a board should include senior nurses and allied health professionals, and equally obviously it should include a senior manager. Importantly, it should answer to its elected District Health Board rather than to a CEO. Management should implement agreed strategy, rather than impose policy from above. Unlike the current restructuring, the change we propose should be approached with care. There may be a case for developing a career path in hospital administration as a medical specialty, as has been done in Australia. Time should be taken to get the details right. Nevertheless, change is needed – real change, not simply a change in appearances.

ADHB's clinical staff have established a reputation for the provision of high quality healthcare. There is a heavy responsibility on decision makers not to reduce an excellent service to a mediocre one. In particular, Auckland's new hospital should improve standards, and not just cut costs. Clinicians can be relied on to do their best in this regard – managers, explicitly charged with cutting costs, might be less committed to this ideal.

We doubt that the public of New Zealand fully realise the extent to which clinicians in general and doctors in particular have been disenfranchised within the public health system. It is time the country re-considers the way it wants it's hospitals run.

1. The Editors. Patients, professionalism and politics: a new beginning. NZ Med J 2000; 113: 23.
2. The Editors. Now Gisborne Hospital. NZ Med J 2001; 114: 173-4.
3. The Editors. Big business and our public health. Creeping privatisation? NZ Med J 2001; 114: 483.
4. The Editors. Health services at breaking point. ... don't panic? NZ Med J 2002; 115: 1.
5. The Editors. Which way forward? NZ Med J 2002; 115: 31.
6. Elliot TS. The love song of J Alfred Prufrock. Selected Poems. Harmondsworth: Faber and Faber, 1952.
7. Inquiry TBRI. The inquiry into the management of care of children receiving complex heart surgery at the Bristol Royal Infirmary. Bristol: 2001.
8. Neutze J, Haydock D. Prioritisation and cardiac events while waiting for coronary bypass surgery in New Zealand. NZ Med J 2000; 113: 69-70.
9. Institute of Medicine. To err is human: Building a safer health system. Washington DC: National Academy Press; 2000.

Socio-economic factors and death rates

Charlotte Paul, ***Associate Professor, Department of Preventive and Social Medicine, University of Otago Medical School, Dunedin.***

Socio-economic deprivation has been associated with higher death rates ever since reliable data were available in England and Wales in the 1840s.[1] Such findings have been replicated for men in New Zealand, for total mortality and for specific causes of death, in the 1970s and 1980s. These analyses examined the effects of occupational class, using mortality data for the numerator and census populations for the denominator.[2,3] What can another study add? The census-mortality study in this issue of the Journal is the first cohort study in New Zealand to examine these issues.[4] With this design the problems of numerator-denominator bias – where occupational class is differently attributed in mortality and census data – should be avoided, and there should be an opportunity to sort out the direction of causality. That is, it should be possible to distinguish, for different causes of death, the role of premature illness in leading to deprivation (so called 'social selection') from the role of deprivation in leading to premature illness and death (so called 'social causation'). This will be more meaningful when the period of follow-up is longer in subsequent analyses. The study also includes a wider range of socio-economic measures, data on specific causes of death, and on women as well as on men.

The potential for high quality information is somewhat limited by the study methods. The methods involved anonymous and probabilistic linking of mortality and census data. Linking was based on date of birth, sex, ethnic group, country of birth and domicile code. Overall 77% of eligible mortality records were linked but this was much lower for certain groups such as young adult deaths and deaths from injury.[5] Clearly those who had not moved domicile between census and death were much more likely to be linked. The resulting bias has been carefully investigated and the authors imply there may be some underestimation of socio-economic differences.[5] Linking using personal identifiers, with a much higher proportion of links, has been common practice in other countries.[6] In the future it would be preferable if this superior method could be used in New Zealand as well.

Nevertheless, as well as pioneering new methods, the authors have provided some valuable insights. Particularly important is the confirmation that death rates for women show similar, though less striking, associations with socio-economic circumstances. The earlier mortality studies were confined to men because at that time an occupation was more likely to be recorded on male death certificates. In comparing a range of socio-economic measures, the investigators found that income showed the strongest associations, followed by small area deprivation and education. Whether this represents a larger 'health selection' effect of income than of the other measures or represents a stronger causal association is unclear.

The most important finding, and one which the authors consider is unlikely to be due to health selection, is the relationship between unemployment and death by suicide. Yet in a study such as this it is not possible to take account of family and psychological factors which may have influenced both becoming unemployed and risk of death by suicide. A New Zealand study of serious suicide attempts found that the association with unemployment became non-significant after adjusting for current psychiatric disorder.[7] Without such adjustment the relative risk estimates were of the same order as found in this study. There is already a consensus that it is not simply unemployment per se which precipitates suicidal behaviour, but that unemployment increases the likelihood of other adverse life events and lessens the psychological and social resources needed to cope.[8] Hence adjusting for psychiatric disorder, which may have followed the unemployment, may be over adjustment. Neither study proves the direction of causality one way or the other. The relationship between unemployment, psychological disorder and suicide is likely to differ from country to country depending on, among other matters, the level of financial support and retraining.[8] These matters require further research and action in New Zealand. Whatever the direction of causality, the unemployed are clearly a vulnerable group for suicide.

The results which are familiar also need to be emphasized, because the persistence of disparities should be a call to action. There were strong associations between socio-economic deprivation and excess deaths from traditional diseases of poverty - accidents and respiratory diseases - though not infections (which is surprising). Coronary heart disease and cancer mortality were also higher in lower socio-economic groups, though part of this was explained by social selection. Consistently strong associations were seen with smoking related diseases - chronic obstructive pulmonary disease and lung cancer. This is a reminder of how socially patterned smoking has become and what a large part smoking plays in health inequalities.[9]

What the census-mortality study leaves unanswered is whether these inequalities are getting better or worse. For that we need to turn to the analysis by Pearce and colleagues of occupational class and mortality in working age men in the 1990s.[10] These results have been awaited with considerable interest because they were expected to show that the market-led reforms which started in the 1980s had widened inequalities.[11] The overall results suggest that mortality differences between occupational classes may have increased, but methodological difficulties prevent a precise determination. More striking, and consistent with the way in which economic hardship has fallen disproportionately on certain groups, is the evidence for widening differences in mortality rates by occupational class within Maori.[12]

Writing in the 1980s,[2] Pearce and colleagues gave two reasons for studying socio-economic differences in mortality: to seek for clues to causes of disease, and to identify groups in the community who have an excess mortality which is potentially preventable. It is for the latter that these results are particularly important. Over the last years various government agencies have made proposals to address social and economic determinants of health and health inequalities.[13,14] The greatest poverty is probably amongst Maori and Pacific sole parents and families with children.[15] Although the effects on health should not be the only, or even the main, reason to reduce poverty, persistent advocacy by health agencies may help to shift social values back towards a concern for solidarity across all socio-economic groups. One of the benefits of increased solidarity may be a common determination to demand, and pay for through taxes, an adequately funded health system.

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1. Whitehead M. William Farr's legacy to the study of inequalities in health. Bull World Health Organ 2000; 78: 86-7.
2. Pearce NE, Davis PB, Smith AH, Foster FH. Mortality and social class in New Zealand. 1: Overall male mortality. NZ Med J 1983; 96: 281-5.
3. Pearce NE, Marshall S, Borman B. Undiminished social class differences in New Zealand men. NZ Med J 1991; 104: 153-6.
4. Blakely T, Woodward A, Pearce N et al. Socio-economic factors and mortality among 25-64 year olds followed from 1991 to 1994. NZ Med J 2002; 115: 93-7.
5. Blakely T, Salmond C, Woodward A. Anonymous record linkage of 1991 census records and 1991-94 mortality records: The New Zealand Census - Mortality Study. (Also at http://www.wnmeds.ac.nz/nzcms-info.htm). Wellington: Department of Public Heatlh, Wellington School of Medicine, University of Otago; 1999.
6. Harding S, Brown J, Rosato M, Hattersley L. Socio-economic differentials in health: illustrations from the Office for National Statistics Longitudinal Study. Health Stat Quarterly 1999; 1: 5-15.
7. Beautrais AL, Joyce PR, Mulder RT. Unemployment and serious suicide attempts. Psychol Med 1998; 28: 209-18.
8. Bartley M. Unemployment and ill heath: understanding the relationship. J Epidemiol Community Health 1994; 48: 333-7.
9. Tobias M, Cheung J. Inhaling inequality. Tobacco's contribution to health inequality in New Zealand. Public Health Intelligence Occasional Bulletin No. 7. Wellington: Ministry of Health; 2001.
10. Pearce N, Davis P, Sporle A. Persistent social class mortality differences in New Zealand men aged 15-64: an analysis of mortality during 1995-1997. Aust NZ J Public Heath. In Press.
11. Paul C. Inequalities in health and redefining values. NZ Med J 1993; 106: 121-2.
12. Sporle A, Pearce N, Davis P. Social class mortality differences in Maori and non-Maori men aged 15-64 during the last two decades. NZ Med J. In Press.
13. National Health Committee. The social, cultural and economic determinants of health in New Zealand: action to improve health. Wellington: National Advisory Committee on Health and Disability; 1998.
14. King A. Implementing the New Zealand health strategy 2001. Wellington: Ministry of Health; 2001.
15. Stephens R. The social impact of reforms: poverty in Aotearoa/New Zealand. Soc Pol Admin 2000; 34: 64-86.

Planet Earth, getting too hot for health?

Most climatologists now believe that the Earth's atmosphere is warming, but no one knows how high, or how fast, temperatures may rise. And even though several national and international studies this year predicted that tropical diseases such as malaria and dengue may extend their ranges as the world warms – and that disrupted storm and rainfall patterns may raise threats of everything from crop failures to cholera - no scientific consensus exists on precisely what ecological upsets will hit which countries, where, in the coming decades. Climate computer models cannot fine-tune their projections to regional levels that could tell local officials, for example, whether to prepare for droughts, or floods, or both.

But several major conclusions are clear. "What needs to be recognized is that there is very little doubt among leading scientists [who have taken part in recent studies] that climate change is a reality," says WHO environmental health expert Dr Carlos Corvalan. "We don't yet know how severe the impacts are going to be or how accurate the predictions of environmental change are, but the evidence is accumulating, and ecological and human health impacts are expected. We are also concerned that the health impacts of global warming will strike hardest at developing nations, particularly the poorest."

Bull WHO 2001; 79: 1090.

Books as carriers of disease

Given the current concern about transmission of anthrax spores via the mail, it may be instructive to revisit early research on whether books can transmit other infectious diseases. As the theory of spontaneous generation gave way to ground-breaking discoveries in the new science of bacteriology by Pasteur, Lister, Koch and others, it was perhaps not surprising that this question was posed at an 1879 meeting of librarians in Chicago. Although there was no evidence that bibliophiles had ever expressed a "fear of books as vehicles of pestilence", the question seemed to exercise greatly the minds of librarians, microbiologists and public health physicians, and numerous articles were published on the subject in medical and library journals over the succeeding 60 years. These articles sought to determine whether books could transmit infectious diseases and how library books could best be sterilised without damage.

An early survey of United States boards of health elicited some notable and bizarre cases of infectious diseases acquired from books. These included scarlet fever transmitted by a book in which a young sufferer had inserted strips of his peeling skin as bookmarks, diphtheria in two children acquired through handling school books from a farmhouse where six cases of the disease had occurred 42 years previously, and smallpox in a man who borrowed books from a circulating library in a neighbouring town affected by a smallpox epidemic.

An 1896 issue of *The Lancet* drew attention to a French study demonstrating isolation of streptococci, pneumococci and *Corynebacterium diphtheriae*, but not *Salmonella typhi* or *Mycobacterium tuberculosis*, from books soiled with the secretions of infected patients. However, the obviously cynical author felt that fear of contagion would be insufficient to drive readers to buy rather than borrow books. A later study found that washings from library books which had been borrowed by people with tuberculosis failed to transmit infection when inoculated into the peritoneal cavity of guinea pigs.

MJ Ferson. MJ Aust 2001; 175: 663-4.

ORIGINAL ARTICLES

Socio-economic factors and mortality among 25-64 year olds followed from 1991 to 1994: the New Zealand Census-Mortality Study

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Abstract

Aim. To measure the association of income, education, occupational class, small area socio-economic deprivation, car access and labour force status with mortality among 25-64 year old males and females using the 1991 census-cohort of the New Zealand Census-Mortality Study.

Methods. Mortality records for 1991-94 were anonymously and probabilistically linked to 1991 census records, thereby creating a cohort study of all New Zealand census respondents. Odds ratios of mortality comparing categories of each socio-economic factor were calculated using logistic regression. For income, education and deprivation (NZDep91) a modified relative index of inequality ($RII_{10:90}$) was calculated. The $RII_{10:90}$ estimates the relative risk of mortality for low socio-economic people (10^{th} percentile rank) compared to high socio-economic people (90^{th} percentile rank) allowing direct comparisons across socio-economic factors.

Results. The relative risk of all-cause mortality for 25-64 year old males with an equivalised household income less than $20 000, compared to greater than $50 000, was 2.16 (95% confidence interval 1.99 to 2.34). For females, this relative risk was 1.68 (1.52 to 1.86). Using the $RII_{10:90}$ all-cause mortality was 2.22, 1.94 and 1.58 times greater among low compared to high socio-economic males for income, NZDep91 and education, respectively. For females, these $RII_{10:90}$ estimates were 1.77, 1.69 and 1.57, respectively. By cause of death, the strongest gradients were observed for respiratory diseases, followed by lung cancer, cardiovascular disease and unintentional injury. For suicide deaths, unemployed males and females had 2.70 (1.84 to 3.95) and 2.86 (1.19 to 6.85) greater rates than the employed.

Conclusions. There are strong socio-economic gradients for all-cause mortality and most specific causes of mortality among both males and female adults in New Zealand, regardless of the choice of socio-economic factor. The gradients were strongest for income, followed by small area deprivation and education, and strongest for 'preventable' causes of death.

NZ Med J 2002; 115: 93-7

Two major bodies of research are available in New Zealand on the association of socio-economic factors with mortality. First, Pearce and colleagues documented the association of occupational class with 15-64 year old male mortality for 1974-78 and 1985-87 (1995-97 forthcoming).[1,2] This body of work found, among other things, an approximately two-fold difference in mortality for the lower occupational classes compared to the higher occupational classes and steeper gradients for deaths amenable to medical intervention. Second, the development of a small area measure of deprivation by Salmond, Crampton and Sutton (NZDep91 and NZDep96)[3] has allowed the measurement of socio-economic mortality gradients by age, sex and ethnic groups.[4,5]

The New Zealand Census-Mortality Study aims to measure socio-economic mortality gradients by linking census (1981, 1986, 1991 and 1996) and mortality records, thereby creating cohort studies of the entire New Zealand census population. The aim of this paper is to present the associations of all-cause and cause-specific mortality during 1991-94 with education, income, car access, small area deprivation, occupational class and labour force status, for premature mortality among males and females aged 25 to 64 years on census night 1991.

Methods

Socio-economic factors from census data. Highest qualification was specified as a four-level variable: tertiary qualification, trade and other qualifications, school qualification only, and no formal qualifications. Household income was equivalised for the number of adults and children in a household using the Jensen Index (Jensen J, Department of Social Welfare. Unpublished), thereby allowing for economies of scale and varying consumption requirements between adults and children. Car access (a marker of asset wealth and access to community resources)[6] was simply the number (nil, one, and two or more) of cars in each household. Labour force status was categorised as the employed, the unemployed (but seeking and available for work), and the non-labour force (eg students, permanently ill, retired). Small area deprivation was assigned on the basis of usual residence using NZDep91 (a New Zealand measure of socio-economic deprivation)[3] and was categorised in quintiles from the least to most deprived of small areas. Occupational class was assigned into six categories based on the New Zealand Socio-Economic Index,[7,8] with a modified cut-point between classes 1 and 2.[9]

Mortality outcome. In addition to all causes combined, deaths were categorised as: cancer (with sub-classifications for colorectal, lung, prostate and breast), cardiovascular (with ischaemic heart disease and cerebrovascular disease), infection and pneumonia, respiratory (with chronic obstructive pulmonary disease), unintentional injury (with whether the death was due to a road traffic crash), suicide, homicide, and remaining causes of death.[9]

Mortality records were anonymously and probabilistically linked to 1991 census records.[10,11] Briefly, of 19 128 eligible decedents who were aged 25-64 years on 1991-census night and died in the six month to three year period following census night, 14 322 (74.9%) were successfully linked to a census record. This linkage success varied by age and ethnicity. However, within age by ethnicity strata there was relatively little difference by occupational class and small area deprivation in linkage success.[11]

Analyses. The 1991-census cohort was restricted to 25-64 year olds with complete data for highest qualification, household income, car access and labour force status. Additionally, deaths in the first six months after census night were excluded due to possible bias from health selection effects. The association of each socio-economic factor with all-cause and cause-specific mortality was determined by logistic regression, with age and ethnicity controlled as covariates. Detailed tabular results of the logistic regression

analyses (similar to that presented in Table 1 for income but with 95% confidence intervals) are available elsewhere,[9] from the NZCMS website,[12] or from the authors.

$RII_{10:90}$. To summarise the association of income, education and NZDep91 with all-cause and cause-specific mortality, we used a variant of the relative index of inequality (RII).[13] Briefly, the RII is a form of relative risk comparing the mortality risk for the most socio-economically *disadvantaged* person compared to the most socio-economically *advantaged*. The RII assumes a linear association of mortality risk with *rank* of the socio-economic factor. A linear regression line was fitted to the odds ratios by midpoint on the cumulative proportion distribution for each ranked socio-economic factor. For example, the intercept of a regression equation for income is the estimated mortality risk at the zero percentile of income (in this case the 'hypothetical' poorest person), the slope is the difference in mortality between the zero and 100th percentile (richest person), and the RII is equal to [intercept] / [intercept + slope]. (As the mortality risk usually decreases with increasing income, the slope is usually negative, and thus the RII is greater than 1.0). The RII becomes unstable if the mortality risk among the most socio-economically advantaged is much less than among the disadvantaged, and is inappropriate if the relationship is non-linear (particularly at the extremes). For these reasons, and also for greater policy relevance, in this paper we present the RII for the 10th compared to 90th percentile based on the same regression line (the $RII_{10:90}$), but both the RII and $RII_{10:90}$ are presented in the Archive Tables on the NZCMS web-site.[12]

Occupational class. Analyses by occupational class were problematic, and are reported on in detail elsewhere.[9,12] For the purposes of this paper, we present only summary results for all males aged 25-64 years with a current occupation on the 1991 census, discarding deaths in the first year of follow-up.

Results

Household income. There was a strong monotonic association of household income with all-cause mortality among both males and females, and for both 25-44 and 45-64 year olds (Figure 1). Comparing the lowest (<$10 000) and highest (>$70 000) household income categories, the relative risks were 2.25 and 2.05 for 25-44 and 45-64 year old males, respectively, and 1.59 and 1.58 for 25-44 and 45-64 year old females, respectively.

Table 1 shows the odds ratios of all-cause and cause-specific mortality for 25-64 year olds by aggregated income categories. The odds ratios of all-cause mortality for 25-64 year old males with an equivalised household income less than $20 000, compared to greater than $50 000, was 2.16 (95% confidence interval 1.99 to 2.34). For females, this odds ratio was 1.68 (1.52 to 1.86). The majority of causes of death were strongly associated with income, with higher mortality among those with lower income. The exceptions to this generalisation were colorectal cancer, infection and pneumonia, homicide, female suicide and female non-road traffic crash injury deaths. However, the lack of association for these latter four causes of death may be due to the small number of deaths. Particularly strong gradients were apparent for respiratory causes of death (including lung cancer), with approximately five-fold greater respiratory mortality among the lowest income groups compared to the highest income group and two and a half times the mortality risk for lung cancer.

Highest qualification and small area deprivation. For those living in the most socio-economically deprived quintile of small areas compared to the least deprived the odds ratios were 2.05 (95% confidence interval 1.88 to 2.23) and 1.73 (1.56 to 1.92) for males and females, respectively. Similarly, the all-cause mortality odds ratios for those with nil

Table 1. Numbers of deaths during 1991-94, odds ratios of death by category of household income, and $RII_{10:90}$ estimates (for household income, highest qualification (education) and small area socio-economic deprivation (NZDep91)) among 1.5 million 25-64 year olds.*

	Deaths	OR by cat of income ($000s)				$RII_{10:90}$ by:		
		≥$50	$30-$49	$20-$29	<$20	Income	Education	NZDep91
Males								
Cancer	2019	1.00	1.33	1.46	1.65	1.69	1.37	1.45
Colorectal	378	1.00	1.22	1.18	1.18	1.19	0.98	1.10
Lung†	456	1.00	1.53	1.80	2.60	2.64	2.46	2.78
Prostate†	108	1.00	1.56	1.47	2.32	2.19	2.12	1.09
Cardiovascular disease	2319	1.00	1.45	1.80	2.31	2.40	1.65	2.13
IHD	1728	1.00	1.45	1.81	2.26	2.36	1.71	2.10
Cerebrovascular	240	1.00	1.77	1.93	2.76	2.77	1.88	2.60
Infection and pneumonia	111	1.00	1.10	0.89	1.11	1.01	0.78	2.66
Respiratory†	198	1.00	1.50	2.58	4.38	4.60	2.05	4.32
COPD†	153	1.00	2.08	3.94	5.59	7.45	2.23	4.96
Unintentional injury	357	1.00	1.65	2.10	2.30	2.52	1.95	1.72
Road traffic crash	183	1.00	1.61	1.89	1.99	2.15	1.64	1.77
Other unintentional	180	1.00	1.69	2.35	2.67	3.00	2.37	1.70
Suicide	282	1.00	1.29	1.50	2.25	2.22	1.74	1.85
Homicide, intentional injury	27	1.00	1.08	0.84	3.21	(na)	(na)	(na)
Other	438	1.00	1.69	2.76	4.06	4.62	1.89	3.28
All causes	**5766**	**1.00**	**1.40**	**1.70**	**2.16**	**2.22**	**1.58**	**1.94**
Females								
Cancer	1947	1.00	1.06	1.16	1.17	1.20	1.24	1.29
Colorectal	303	1.00	0.66	0.80	0.82	0.87	0.86	0.87
Lung†	255	1.00	1.60	1.92	2.47	2.59	4.61	3.53
Breast	477	1.00	0.93	1.22	0.93	1.00	1.34	0.97
Cardiovascular disease	915	1.00	1.45	2.14	2.65	2.92	2.27	2.43
IHD	498	1.00	1.60	2.56	3.66	4.15	3.36	2.59
Cerebrovascular	228	1.00	1.20	1.35	1.40	1.44	1.46	2.13
Infection and pneumonia	54	1.00	1.79	1.57	1.41	1.48	0.84	1.10
Respiratory†	180	1.00	1.51	3.00	4.89	5.88	2.92	3.24
COPD†	135	1.00	1.71	3.14	5.61	6.86	3.60	2.88
Unintentional injury	129	1.00	1.45	1.74	2.60	2.71	1.46	1.69
Road traffic crash	84	1.00	0.87	1.49	1.85	2.22	1.84	2.05
Other unintentional	42	1.00	5.76	3.53	8.22	5.40	0.94	1.21
Suicide	90	1.00	1.26	0.98	2.00	1.83	0.91	1.95
Homicide, intentional injury	15	1.00	2.52	2.61	8.43	(na)	(na)`	(na)
Other	369	1.00	1.31	2.17	2.31	2.66	3.11	2.13
All causes	**3702**	**1.00**	**1.18**	**1.46**	**1.68**	**1.77**	**1.57**	**1.69**

*The odds ratios are from a logistic regression model with age in ten-year age groups and ethnicity dichotomised as Maori and Pacific Island, and non-Maori non-Pacific. Numbers of deaths are random rounded to the nearest multiple of three as per SNZ protocol, but odds ratios are calculated with exact data. Odds ratios and 95% confidence intervals for household income (although with the <$20 000 group as the reference group as it contained the most deaths), highest qualification and small area deprivation may be found in Archive Table 1, Archive Table 2 and Archive Table 3 on the NZCMS web-site (http://www.wnmeds.ac.nz/nzcms-info.htm) or directly from the authors. † Only age-group 45-64 included in analysis.

qualifications compared to those with tertiary qualifications were 1.64 (1.52 to 1.77) and 1.49 (1.37 to 1.62) for males and females, respectively (Detailed results available elsewhere).[9,12]

Income, education and deprivation compared by $RII_{10:90}$. The $RII_{10:90}$ estimates of the all-cause and cause-specific mortality gradients by household income, highest qualification and small area socio-economic deprivation (NZDep91) are shown in Table 1, and plotted in Figure 2. For all-cause mortality by income, the $RII_{10:90}$ were 2.22 and 1.77 for males and females, respectively. That is, using the odds ratios in Table 1 and the proportions of the 25 to 64 year-old cohort in each income-group, we estimated that a woman with a low household income (10th percentile rank) had 1.77 times the mortality risk of a woman with a high income (90th percentile rank). For both males and females, the $RII_{10:90}$ estimates of the all-cause mortality gradients were strongest for income, intermediary for NZDep91, and weakest for education.

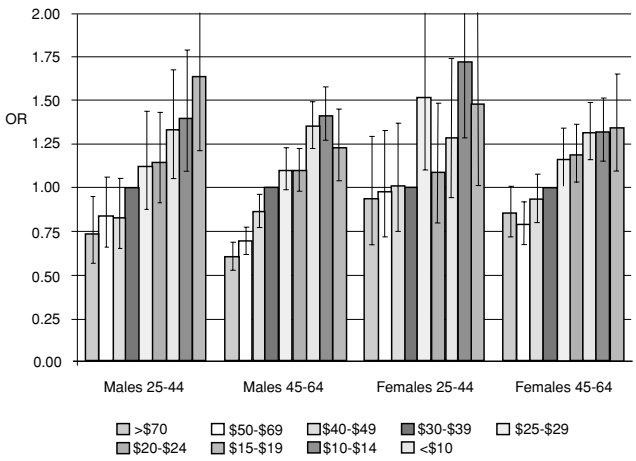

Figure 1. Age and ethnicity adjusted odds ratios of all-cause mortality by equivalised household income (in $1000s) for 25-44 and 45-64 year olds, males and females. Bars are 95% confidence intervals. The reference category for the odds ratios is $30-39 999.

For cause-specific mortality, several patterns are evident from Figure 2. First, as with all-cause mortality, there was tendency for income gradients to be strongest, followed by NZDep91 then education. Second, the gradients (regardless of socio-economic factor) were strongest for respiratory diseases, followed by lung cancer, cardiovascular diseases, and unintentional injury. Third, the patterns were similar between males and females. For example, among both sexes all three socio-economic factors were strongly associated with respiratory mortality but not colorectal cancer mortality. (Note that for more uncommon causes of death among 25-64 year olds eg prostate cancer, and those with large socio-economic mortality gradients eg, chronic obstructive pulmonary disease, the $RII_{10:90}$ may be unstable as represented by the scatter in Figure 2).

Occupational class. The $RII_{10:90}$ for male all-cause mortality was 1.48, and by cause of death was 1.36 for cancer, 1.56 for cardiovascular disease, 1.60 for injury and 1.75 for suicide.

Car access (measure of asset wealth and access to community resources). Compared to males living in households with access to two or more cars, males without car access had an odds ratio of all-cause mortality of 2.23 (2.03 to 2.46) and males with access to one car had an odds ratio of 1.35 (1.28 to 1.43). For females, the odds ratios were 1.67 (1.49 to 1.87) and 1.30 (1.21 to 1.39), respectively.

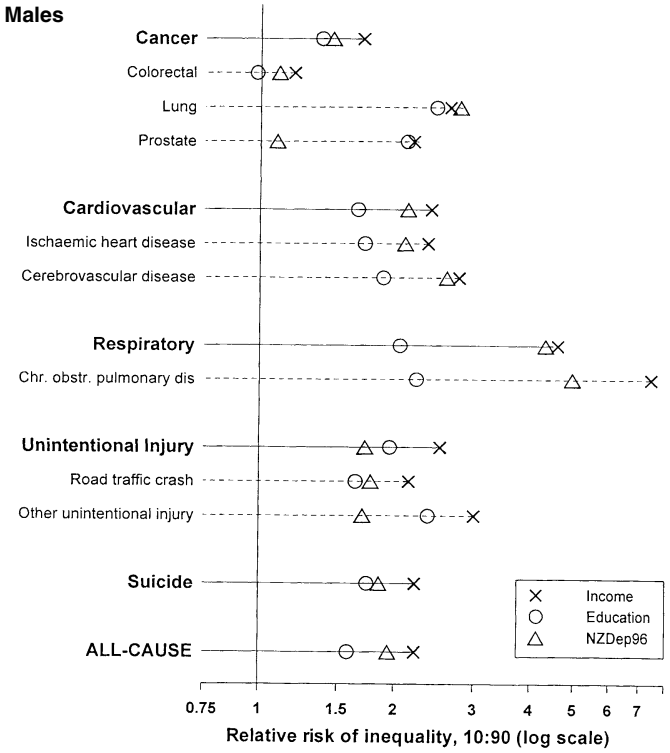

Figure 2a. Cause-specific relative indices of inequality (10:90) by income, education and small area socio-economic deprivation in the 1991 NZCMS cohort among 25-64 year olds. Lines are not error bars. $R11_{10:90}$ values are presented in Table 1.

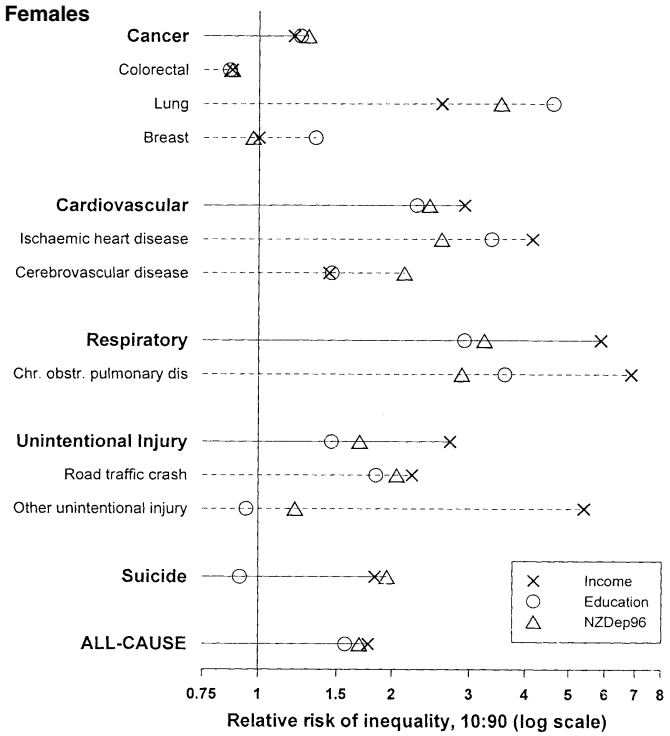

Figure 2b. Cause-specific relative indices of inequality (10:90) by income, education and small area socio-economic deprivation in the 1991 NZCMS cohort among 25-64 year olds. Lines are not error bars. $R11_{10:90}$ values are presented in Table 1.

Labour force status. Those in the *non-active labour* force had the most elevated risks of mortality compared to the employed (Table 2). This was expected for cancer and cardiovascular disease (and possibly suicide) as people in poor health are more likely to be in the non-active labour force. The association of

Table 2. All- and cause-specific mortality odds ratios (95% CI) by labour force status in the 1991 NZCMS cohort among 25-64 year olds, adjusted for age and ethnicity.*

	Employed		Unemployed		Non-active labour force	
Cohort size (males; females)	(519,195; 406,107)		(39,312; 29,760)		(90,243; 231,315)	
	OR	Death	OR (95% CI)	Deaths	OR (95% CI)	Deaths
Males						
Cancer	1.00	1059	1.24 (0.99-1.56)	81	2.01 (1.82-2.23)	882
CVD	1.00	1092	1.42 (1.15-1.74)	99	2.45 (2.23-2.68)	1125
Unintent. Injury	1.00	264	1.25 (0.84-1.85)	27	1.42 (1.06-1.91)	66
Suicide	1.00	168	2.70 (1.84-3.95)	33	3.29 (2.45-4.43)	81
All causes	**1.00**	**2850**	**1.40 (1.24-1.59)**	**270**	**2.56 (2.41-2.72)**	**2646**
Females						
Cancer	1.00	789	0.88 (0.63-1.22)	36	1.52 (1.37-1.68)	1119
CVD	1.00	228	1.16 (0.70-1.93)	15	2.68 (2.28-3.15)	675
Unintent. Injury	1.00	60	0.62 (0.19-2.00)	6	1.73 (1.19-2.53)	66
Suicide	1.00	33	2.86 (1.19-6.85)	6	2.77 (1.74-4.43)	51
All causes	**1.00**	**1227**	**1.15 (0.92-1.43)**	**84**	**2.07 (1.92-2.23)**	**2388**

• The odds ratios are from a logistic regression model with age in ten-year age groups and ethnicity dichotomised as Maori and Pacific Island, and non-Maori non-Pacific. Numbers of deaths are random rounded to the nearest multiple of three as per SNZ protocol, but odds ratios are calculated with exact data.

suicide death with *unemployment* was particularly strong, with odds ratios compared to the employed of 2.70 (1.84 to 3.95) and 2.86 (1.19 to 6.85) for males and females, respectively. (There was also a notable excess risk of suicide death among the non-active labour force). Thus, much of the elevated all-cause mortality among the unemployed (odds ratios of 1.40 (1.24 to 1.59) and 1.15 (0.92 to 1.43) for males and females, respectively) was due to suicide.

Discussion

Our results demonstrate strong socio-economic mortality gradients among adults for a range of socio-economic factors and a range of causes of death. Gradients were steepest for more preventable causes of death (Figure 2, eg lung cancer). There was little evidence of a threshold effect, rather each step up the socio-economic ladder was associated with an incremental reduction in mortality risk. Extensive sensitivity analyses reported elsewhere suggest that the results in this paper may be modestly underestimated due to bias in the record linkage but little affected by selection bias.[9] The census variables are crude measures of socio-economic factors.[9] For example, the household income variable is calculated from tick-box categories on the census, will be prone to errors based on incorrect recall of the previous years income, and was not a measure of 'usual' income. Accordingly, we would expect *all* the associations of the individual-level socio-economic factors with mortality presented in this paper to be underestimated due to misclassification bias. Thus, there is an indisputably strong *crude* association of socio-economic factors with mortality in New Zealand. However, causal inference based on the analyses presented in this paper requires a careful consideration of confounding, causal pathways and health selection – issues discussed below.

In addition to the generalised and strong socio-economic mortality gradient there was a particularly strong association of unemployment with suicide (Table 2). Similar elevated risks of suicide among the unemployed have been shown internationally[14-16] and in New Zealand for self inflicted harm.[17] Given that to be classified as unemployed on the 1991 census required being both available for work and seeking work, we do not believe that this elevated suicide rate for the unemployed is a function of people in poor mental health (and at higher risk of suicide) drifting out of employment into unemployment, since such people would tend to move completely out of the workforce into the non-labour force group. Further, multivariate analyses (reported elsewhere) suggest little confounding of the association of unemployment with suicide by other socio-economic factors – a quite different pattern to that observed for cancer, cardiovascular and injury deaths.[9] Thus, our results are consistent with a causal association of unemployment with suicide death. However, Beautrais et al found that after controlling for childhood, family and educational factors there was no statistically significant association of unemployment with suicide *attempt* in the Canterbury Suicide Project, a case-control study of 302 individuals who made a serious suicide attempt and 1028 randomly selected controls.[18] It is possible that the association of unemployment with suicide attempt is different from that with suicide death, or that this study was insufficiently powerful to detect a statistically significant association.

The associations of household income with all-cause mortality were stronger among males than females, and this difference was due mainly to a stronger association of household income with cancer death among males (Table 1). Such a finding suggests the possibility of health selection whereby poor health prior to death causes one's income to decrease (as is common with cancer) thereby spuriously strengthening the association of income with cancer death. As males among this cohort can be assumed to be the major contributors (on average) to household income, the pattern of a stronger association of income with cancer death among males is further suggestive of such health selection bias. Extensive sensitivity analyses to test this possibility in the NZCMS were inconclusive.[9] On balance, some of the observed association of income with cancer (and probably cardiovascular deaths) in the NZCMS was due to health selection, but not all of it.

What is the policy importance of the findings in this paper? Would it really lower someone's mortality risk if his or her income suddenly increased by a large amount (or another socio-economic factor was substantially altered) and nothing else changed? For some people in difficult circumstances it may make a big difference (being able to move to safer, less crowded, housing for instance). But overall, changing individual incomes without making any other changes is likely to have only a modest impact on population health in the short term.[19] First, it takes time for social influences to affect health. An increase in income might improve diet and as a result, over years, lower the risk of ischaemic heart disease. (However, counter-examples are possible too. For respiratory disease improved housing

conditions may have an immediate beneficial effect.) Second, socio-economic resources of individuals are not randomly distributed. Having a high income is a function of many factors including one's education and previous life circumstances. These correlated factors will also be predictors of health and therefore confounders of the income-mortality association. A life-course perspective suggests that health is a function of cumulative events experienced throughout life, and even inter-generationally.[20] Third, the socio-economic factors used in this study may be viewed as a mixture of 'markers' of underlying socio-economic conditions (eg car ownership), component items that reflect one's structural position on the 'macro' socio-economic ladder (eg income and education), as well as mediating variables at the 'micro' level that are proximate to health (eg sufficient income to afford a healthy diet). Thus, net health gains from changing socio-economic resources of individuals (particularly 'marker' variables like car access), but not changing the associated structure of society, are likely to be modest. Indeed, without changing the structure of society (eg narrowing income distributions) lifting one person up the socio-economic ladder may be (necessarily) balanced by someone else falling down the ladder.

Reliance on 'downstream' public health interventions (eg health education) have historically improved health more rapidly for higher socio-economic groups, thereby *increasing* relative health inequalities. In addition to targeting these downstream interventions more effectively and equitably (eg smoking quit programmes in poorer communities), and 'upstream' structural change to society suggested above, health inequalities would also probably be reduced by 'mid-stream' population-based interventions that preferentially improve health among lower socio-economic groups. Examples of such mid-stream interventions include reduction in tobacco availability, injury prevention programmes in the workplace, increased access to primary health care services, and access to safe and affordable housing.

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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 web-site (http://www.wnmeds.ac.NewZealand/nzcms-info.htm). For further information about confidentiality matters in regard to this study please contact Statistics New Zealand.

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1. Pearce N, Marshall S, Borman B. Undiminished social class mortality differences in New Zealand men. NZ Med J 1991; 104: 153-6.
2. Marshall S, Kawachi I, Pearce N, Borman B. Social class differences in mortality from diseases amenable to medical intervention in New Zealand. Int J Epidemiol 1993; 22: 255-61.
3. Salmond C, Crampton P, Sutton F. NZDep 91: A new Zealand index of deprivation. Aust NZ J Public Health 1998; 22: 835-7.
4. Salmond C, Crampton P. Deprivation and health. In: Howden-Chapman P, editor. Social inequalities in health: New Zealand 1999. Wellington: Ministry of Health; 2000. p9-64.
5. Blakely T, Kiro C, Woodward A. Unlocking the numerator-denominator bias. II: Adjustments to mortality rates by ethnicity and deprivation during 1991-94. NZ Med J in press.
6. Macintyre S, Ellaway A, Der G et al. Do housing tenure and car access predict health because they are simply markers of income or self-esteem? A Scottish study. J Epidemiol Community Health 1998; 52: 657-64.
7. Davis P, McLeod K, Ransom M, Ongley P. The New Zealand Socioeconomic Index of Occupational Status (NZSEI). Wellington: Statistics New Zealand; 1997.
8. Davis P, McLeod K, Ransom M et al. The New Zealand Socioeconomic Index: developing and validating an occupationally-derived indicator of socioeconomic status. Aust NZ J Public Health 1999; 23: 27-33.
9. Blakely T. Socio-economic factors and mortality among 25-64 year olds: The New Zealand Census-Mortality Study. [Doctorate] Dunedin: University of Otago; 2001. Available at http://www.wnmeds.ac.nz/nzcms-info.htm
10. Blakely T, Salmond C, Woodward A. Anonymous record linkage of 1991 census records and 1991-94 mortality records: The New Zealand Census-Mortality Study. Wellington: Department of Public Health, Wellington School of Medicine, University of Otago; 1999. Available at http://www.wnmeds.ac.nz/nzcms-info.htm
11. Blakely T, Salmond C, Woodward A. Anonymous linkage of New Zealand mortality and Census data. Aust NZ J Public Health 2000; 24: 92-5.
12. NZCMS Web-Site. http://www.wnmeds.ac.nz/nzcms-info.htm
13. Mackenbach J, Kunst A. Measuring the magnitude of socio-economic inequalities in health: an overview of available measures illustrated with two examples from Europe. Soc Sci Med 1997; 44: 757-71.
14. Martikainen P. Unemployment and mortality among Finnish men, 1981-5. BMJ 1990; 301: 407-11.
15. Bartley M. Unemployment and ill health: understanding the relationship. J Epidemiol Community Health 1994; 48: 333-7.
16. Moser KA, Fox AJ, Jones DR, Goldblatt PO. Unemployment and mortality: further evidence from the OPCS longitudinal study 1971-81. Lancet 1986; I: 365-7.
17. Ngati Kahungunu Iwi Incorporated, Te Ropu Rangahau Hauora a Eru Pomare. Mauri Mahi: Does unemployment affect health? The closures of Whatatu and Tomoana. Hastings: Ngati Kahununu Iwi Incorporated; Wellington: Te Ropu Rangahu Hauora a Eru Pomare; 2001.
18. Beautrais A, Joyce P, Mulder R. Unemployment and serious suicide attempts. Psychol Med 1998; 28: 209-18.
19. Blakely T, Woodward A. Counterfactual challenges to social epidemiology. Australas Epidemiologist 2000; 7: 28-31.
20. Kuh D, Ben-Shlomo Y editors. A life course approach to chronic disease epidemiology. Oxford: Oxford University Press; 1997.

Asthma linked with high paracetamol use

The high use of paracetamol in Britain and other English-speaking countries may be directly responsible for the surge in asthma cases and other respiratory illnesses compared with the Mediterranean countries and in eastern Europe.

In the biggest study of asthma undertaken involving 140 000 people in 22 countries the link between the drug's sale and prevalence of asthma startled researchers because it showed that in some countries people were up to eight times more likely to contract allergic respiratory diseases than in other apparently similar places.

Christer Janson, associate professor at the department of medical science at Uppsala University in Sweden, said paracetamol sales were high in English-speaking countries, and were positively associated with asthma symptoms, eczema and allergic eye problems in children, and wheeze, asthma, eye and bronchial problems in adults.

A high prevalence was found in Australia, New Zealand, Britain and the United States, with low scores in Iceland, Greece, Norway, Italy and Spain.

The extraordinary finding after the 10-year research programme was one of a number of new leads for researchers into asthma, which has reached epidemic proportions in Britain, where it kills 1600 people a year and affects one of seven children.

The European Community respiratory health survey involving 14 experts is published in the European Respiratory Journal this month.

Professor Janson said the main conclusion was that it was not geographical differences, for example more sunshine or living in the country, that made people more likely to get asthma but environmental reasons, like dust mites, pet cats, cooking with gas, or dust at work. Factors such as different testing methods and skin types were studied to make sure the results were not distorted, and were discounted.

Paul Brown. Guardian Weekly 13-19 September, 2001.

The comparability of community outcomes for European and non-European survivors of stroke in New Zealand

Harry McNaughton, ***Senior Lecturer;*** **Mark Weatherall,** ***Senior Lecturer;*** **Kathryn McPherson,** ***Senior Lecturer;*** **William Taylor,** ***Senior Lecturer;*** **Matire Harwood,** ***Research Fellow, Rehabilitation Teaching and Research Unit, Wellington School of Medicine, Wellington.***

Abstract

Aims. To measure community outcomes for stroke comparing European and non-European survivors.

Methods. This was a prospective, hospital-based study of consecutive patients admitted to three general hospitals in Wellington with acute stroke. Patients were assessed using a range of instruments for prestroke function, function while in hospital, and then followed for twelve months post-hospital discharge. Ethnicity was decided by self-report and Maori, Pacific people and Asians were grouped together as "non-Europeans" for analysis.

Results. 181 people with stroke were enrolled of whom 171 (94.5%) were followed up to death or twelve months post hospital discharge. 33 (18%) were non-European with 13 (7%) Maori, 14 (8%) Pacific people and 6 (3%) Asians. Non-European survivors at twelve months post hospital discharge were more likely to be dependent (corrected OR 21.0, 95% CI 3.1, 141), have significantly lower Functional Independence Measure scores, lower London Handicap Scores and lower scores on the Short Form 36 domains of physical functioning and vitality and Physical Component Summary score.

Conclusions. Community outcomes for survivors of stroke may be worse for non-Europeans although this should be confirmed in a larger study.

NZ Med J 2002; 115: 98-100

No information is currently available that compares community outcomes for European and non-European survivors of stroke in New Zealand. A higher age-matched 28 day case fatality rate for Maori and Pacific people, especially men, compared to Europeans, was described in the 1991 Auckland Stroke Study.[1] Maori and Pacific people tend to have worse stroke risk factor profiles than Europeans with higher rates of smoking, higher mean blood pressure, higher mean weights and higher relative risks of impaired glucose tolerance.[2] Perhaps more importantly with regard to longer term outcomes, socio-economic indicators are worse for Maori and Pacific people with higher rates of unemployment, higher proportions of the workforce on low incomes, poorer educational achievement, poorer housing and overcrowding of housing compared to Europeans.[3]

As part of a prospective study of stroke patients admitted to hospital in the Wellington region during 1997, we measured community outcomes for survivors of stroke, comparing European and non-European groups.

Methods

Participants. Participants were patients consecutively admitted to any of the three general hospitals in the Wellington region meeting the WHO definition for stroke[4] (but not including subarachnoid haemorrhage) during 1 February to end November 1997. To be included, the stroke symptoms had to occur within seven days of presentation to hospital and the person's normal residence had to be within the greater Wellington region. Those with acute stroke that were not admitted to hospital (around 25% of cases in New Zealand[5]) were not included.

Setting. The three hospitals serve a population of around 350 000 in the greater Wellington area. Each centre includes emergency medical services, acute medical care, inpatient rehabilitation, and a variety of community rehabilitation services. Two hospitals have an intensive care unit.

Ascertainment of stroke events. Multiple sources were used to identify all possible stroke events. A majority of cases were identified through daily searches of hospital admission lists, and a wide range of admission diagnoses (such as collapse, left or right side weakness, transient ischaemic attack) were investigated to ensure that all strokes were included. In addition, all wards were visited daily to detect missed cases, and to identify new cases of stroke in patients initially admitted with another diagnosis. Regular contact was also maintained with clinical nurse managers, ward clerks, doctors and nurses of each ward to ensure that every case was included. Finally, discharge lists of all patients with the International Classification of Diseases (ICD) (9th Revision) codes of 431-434 and 436-438 were reviewed to estimate the completeness of case ascertainment.

Procedure. At the first assessment, generally within the first three days of the admission, demographic details including ethnicity, and details of the stroke itself were collected. Ethnicity was determined by self-report and participants were grouped into "Maori", "Pacific people", "Asian" or "European". Strokes were classified, using the Oxfordshire Community Stroke Project classification.[6] At the first assessment, disability was measured using the Functional Independence Measure (FIM).[7] In addition, data on prestroke function was estimated using the FIM, Short Form 36 (SF36),[8] London Handicap Scale (LHS)[9] and Modified Rankin Scale (MRS).[10] The interviewer asked the patient or the person who knew them best, to answer questions about their health "as if it was two weeks ago". Further information relating to the person's living and work situation and income was also collected at the first assessment. Patients were then seen every two weeks while in hospital and FIM score recorded. After discharge, from hospital, patients were assessed at three months, six months and finally at 12 months post-discharge. On these occasions, disability was measured (using the FIM) along with handicap, independence and health status measures (LHS, MRS, SF36). Information concerning the living situation, benefit and other income, contact with health service providers and volunteer groups was also collected at each time point. Information was collected by telephone interview with the patient, or a face-to-face visit when there was a communication disorder, memory difficulty or no telephone.

Major efforts were made to ensure follow-up. Multiple attempts (up to five) at telephone contact, letters to the last known address, contact with the GP surgery, review of hospital notes for further admissions or death were all tried before a case was labelled as 'uncertain whereabouts'.

Measures. The FIM is an 18-item, 7-point scale (minimum 18, maximum 126) measuring disability with increasing values indicative of greater levels of independence. The LHS provides a measure on each of the six WHO dimensions of handicap combined into a weighted score ranging from 100 (no disadvantage) to 0 (maximum possible disadvantage). The SF-36 is a 36-item measure of functional status related to health. Scores are computed for each of eight domains with scores ranging from zero to 100 where higher scores indicate less impact of health on functioning. In addition, a Physical Component Score (PCS) and Mental Component Score (MCS) were computed. The MRS is a 6-point rating scale grading patients on their overall level of independence. A score of five demonstrates a high level of dependence, zero indicates complete independence with no symptoms and scores of two or less cover the range of 'independence from others'. Recalled prestroke assessment using any of these measures has not been adequately validated, although an attempt has been made to do so as part of this study and the results will be published separately. Those results show that for the group as a whole, the prestroke assessments of people with stroke match expected values for age-matched community living controls. The study was approved by the local Ethics Committee and all participants gave written, informed consent.

Statistical analysis. Non-parametric statistics were used for data not meeting assumptions for normality. Categorical variables were analysed using χ^2 tests. Multiple logistic regression models were used to calculate odds ratios. Each of the baseline and stroke variables were entered into a

univariate analysis with significant variables ($p < 0.1$) retained and entered into the multivariate analysis. Subsequent refinement of the model occurred to achieve the best fit with fewest variables. SPSS 10.0 software was used for the analyses.

Results

181 patients were identified with stroke and agreed to participate. Systematic case note review covering the study period suggested that around 75% of all stroke patients admitted to hospital and eligible to participate were enrolled. The main reason for non-enrolment of eligible cases was failure to give written, informed consent. Of those enrolled in the study, 33 (18%) were non-European with thirteen (7%) Maori, fourteen (8%) Pacific people and six (3%) Asians. This compares with 12% Maori, 7% Pacific people and 3% Asians in the Wellington region in the 1996 census[11] and 5% Maori, 6% Pacific people and 2% Asians in the 1991 Auckland Stroke Study (ASS).[1] The Maori, Pacific and Asian subjects were combined in a single "non-European" group. In the ASS, Asian people were grouped with Europeans. However, as many Indians living in New Zealand are originally from Fiji, we chose to include Asians with other non-Europeans. The values of the pre-stroke variables for Asian participants tend to fall somewhere between those of Europeans and Maori/Pacific people (data not shown). However, the values for age, pre-stroke independence and disability are closer to those of non-Europeans and, we feel, justifies their inclusion in the non-European group.

Prior to the stroke, the non-Europeans were younger (median difference around ten years), more likely to be living with another adult, with higher (better) disability scores and higher (better) physical function scores than the Europeans (Table 1). There was a significant difference in the distribution of stroke types between European and non-Europeans in the study: the proportion of haemorrhages was higher in the non-Europeans (27% vs 7%), with more lacunar infarcts in the European group.

Only one out of 148 Europeans was unable to be followed up to death or at least one community assessment, but nine of 33 (27%) non-Europeans could not be followed up. Searching of hospital records, the 1999 Electoral Roll and the National Death Register allowed confirmation that the one European not followed up was alive more than twelve months after hospital discharge. Of the nine non-Europeans not followed up, five were alive in New Zealand at least 12 months after hospital discharge, one was known to be alive in Samoa and none of the remaining three were recorded as having died in New Zealand.

53% of the European group who survived to hospital discharge were independent of help from others (MRS < 3) at twelve months whereas only 14% of non-Europeans were independent (Table 2). After correcting for age and initial FIM score using multiple logistic regression, the corrected odds ratio was 20.1 (95% CI 3.1, 141). Other measures confirmed the picture of poorer outcomes for non-European survivors. The non Europeans scored a median 10 points lower (around 10% of the total score range) on the FIM at twelve months post discharge compared to the Europeans ($p = 0.04$). There were significant differences in handicap (LHS median difference 8.2, $p = 0.005$), physical functioning (median difference 22.5, $p = 0.01$) and vitality (median difference 15, $p = 0.02$), all favouring European survivors. The mean PCS score of the SF 36 was significantly lower for non-Europeans (mean difference 5.3, $p = 0.02$) although this was not the case for the MCS (mean difference zero).

Discussion

These results suggest that health outcomes in non-European survivors of stroke were worse than for Europeans twelve months following hospital discharge. However, some caution is required. Firstly, the study was relatively small. Secondly, there was incomplete follow-up of the non-Europeans. The subjects not followed up were not significantly different in age or initial disability scores but had significantly higher median FIM scores at hospital discharge (110 vs 83, $p = 0.03$). If these subjects were assigned twelve month outcomes based on their discharge FIM scores, the corrected odds ratio for independence is 3.5 (95% CI 1.4, 8.8), ie remaining significantly in favour of Europeans. Thirdly, there was an unexpectedly good survival rate to hospital discharge in the non-Europeans (corrected odds ratio of not dying 3.9, 95% CI 0.7, 22.6) which persisted to twelve months post discharge (corrected odds ratio of not dying 9.1, 95% CI 1.6, 52.7). As almost all of the deaths were in people with severe strokes, the median FIM score at initial assessment of survivors to

Table 1. Baseline characteristics and features of stroke.

Variable	European (n=148)	Non-European (n=33)	p value
Age: median (IQR)	77.6 (71.2, 84.0)	66.9 (57.9, 75.9)	<0.0001
Female: n (%)	79 (53)	17 (52)	0.85
Previous stroke: n (%)	43 (29)	11 (33)	0.63
Current smoker: n (%)	29 (20)	10 (30)	0.18
Lives with at least 1 other adult: n (%)	77 (52)	25 (76)	0.013
Pre-stroke health status:			
Independence (MRS <3): n (%)	124 (86)	31 (94)	0.22
Disability (FIM): median (IQR)	124 (118.8, 126)	126 (121, 126)	0.006
Handicap (LHS): median (IQR)	83.8 (72.5, 95.1)	84.7 (74.6, 94.8)	0.93
Physical function: median (IQR)	75 (55, 95)	85 (71, 99)	0.04
General Health: median (IQR)	72 (54.5, 89.5)	72 (59.5, 84.5)	0.66
Vitality: median (IQR)	60 (45, 75)	65 (52.5, 77.5)	0.32
Stroke variables:			
Initial FIM: median (IQR)	63.5 (23, 101)	57 (28, 82)	0.75
Systolic BP on admission: mean (sd)	170.5 (35.4)	176.7 (35.6)	0.37
Stroke type: n (%)			
LACI	49 (34)	5 (17)	p = 0.016
POCI	14 (10)	4 (13)	
PACI	43 (30)	8 (27)	
TACI	27 (19)	5 (17)	
PICH	10 (7)	8 (27)	

IQR = interquartile range, MRS = Modified Rankin Scale, FIM = Functional Independence Measure, LHS = London Handicap Scale, Physical Function, General Health and Vitality are domains of the Short Form 36. LACI = lacunar infarct, POCI = posterior circulation infarct, PACI = partial anterior circulation infarct, TACI= total anterior circulation infarct, PICH = primary intracerebral haemorrhage.

Table 2. Results of selected health outcomes at 12 month follow-up comparing Europeans and non-Europeans.

Variable	European (n = 146)	Non-European (n = 24)	p value
FIM: median (IQR)	111 (88, 121)	101 (71, 111)	0.04
MRS <3: n (%)	51 (53)	3 (14)	0.001
LHS: median (IQR)	65.3 (58.1, 83.9)	57.1 (43.5, 65.5)	0.005
Physical Function (SF36): median (IQR)	35 (10, 67.5)	12.5 (0, 30)	0.01
General Health (SF36): median (IQR)	58.5 (45, 72)	53.5 (35, 65)	0.09
Vitality (SF36): median (IQR)	55 (40, 67.5)	40 (35, 55)	0.02
PCS: mean (95% CI)	38.7 (36.7, 40.7)	33.4 (29.8, 36.9)	0.02
MCS: mean (95% CI)	54.8 (52.9, 56.6)	54.8 (51.2, 58.3)	1.0

IQR = interquartile range, MRS = Modified Rankin Scale, FIM = Functional Independence Measure, LHS = London Handicap Scale. PCS = Physical Component Summary score and MCS = Mental Component Summay score of the Short Form 36 (SF-36). Physical Function, General Health and Vitality are individual domains of the SF-36.

twelve months was significantly lower in the non-European group (non-Europeans median 51 vs Europeans median 83, Mann Whitney U test p = 0.002). This means that the surviving non-Europeans had had more severe strokes than the surviving Europeans. Nevertheless, correcting for initial disability level in the multiple logistic regression equation for independence at twelve months still provides significantly higher odds favouring European stroke patients.

If, despite these caveats, worse health outcomes at twelve months for non-European survivors of stroke are real, what might be the explanation? One possibility for the increased dependence might be cultural, ie in Maori and Pacific cultures, independence might not be seen as a goal for either the patient or their family. This could lead to a higher (worse) score on the MRS despite a level of disability that would normally translate into a lower score. However an analysis of the mean FIM score for each grade of the MRS for European and non-European groups suggests that similar levels of disability translate into the same level of dependence ie the non-European people scoring 3 on the MRS (dependence on others) are no less disabled than their European counterparts (data available from authors). It is possible however, that cultural mores affected the outcome - if independence from others is not something that a person is aiming for, it is possible that the level of improvement in both disability and independence achieved will be limited. A community survey of Asian elders in Leicester, UK suggested that they were more dependent, compared to elderly Europeans.[12] Ebrahim suggested that this difference was culture-specific and that cultural differences exist for simple activities of daily living such as dressing, bathing and eating.[13] This may also be the case in New Zealand but data to support this hypothesis are currently lacking.

Another possibility is that the 'standard' measures of outcome are not appropriate for some ethnic groups, or that the interpretation of results from these measures should allow for differences in ethnicity. This argument can be partly countered by our use of pre-stroke estimation of disability, independence, health status and handicap where the non-Europeans scored at least as well as the Europeans on these measures. We can not, however, rule out the possibility that 'health' status, as measured in the SF36 or LHS, may be substantially different for Maori, Pacific people and Europeans following a significant health event such as a stroke.[14] In particular, aspects such as spirituality and whanaunatanga (family support) are not captured by these measures.

Finally we must consider those factors shown to be associated with poor functional outcome. Co-morbid conditions such as diabetes and heart disease, a delay in seeking medical care and a delay in receiving rehabilitation are more prevalent in ethnic minority groups[15-18] and may explain the disparity in stroke outcomes seen. Other barriers to quality stroke care may exist such as poor access or inappropriate treatment. Patient preference and provider bias may play important roles in access and treatment.

Demographic projections for New Zealand reveal an ageing population and a higher proportion of non-Europeans,[11] which suggests that stroke in non-Europeans will get proportionately more common in the future. Further investigation of outcomes for people of different ethnic groups after stroke is required to confirm, or refute, the suggestion made here that health outcomes may be worse for non-European survivors following stroke in New Zealand. Disparities in health outcomes suggest a problem with the performance of a health system.[19] It is important to identify the disparities and work towards eliminating them. We are currently working to identify barriers to stroke recovery in Maori, prior to developing and testing interventions to maximise community outcomes following stroke.

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1. Bonita R, Broad JB, Beaglehole R. Ethnic differences in stroke incidence and case fatality in Auckland, New Zealand. Stroke 1997; 28: 758-61.
2. Our health, our future. Hauora pakari, koiora roa. The state of the public health in New Zealand 1994. Wellington: Public Health Commission; 1994.
3. Durie M. Whaiora: Maori health development. 2nd ed. Auckland: Oxford University Press; 1998.
4. Hatano S. Experience from a multicentre stroke register: a preliminary report. Bull World Health Organ 1976; 54: 541-53.
5. Bonita R, Broad JB, Beaglehole R. Changes in stroke incidence and case-fatality in Auckland, New Zealand, 1981-91. Lancet 1993; 342: 1470-3.
6. Bamford J, Sandercock PA, Dennis M et al. Classification and natural history of clinically identifiable subtypes of cerebral infarction. Lancet 1991; 337: 1521-6.
7. Granger CV, Cotter AC, Hamilton BB, Fiedler RC. Functional assessment scales: a study of persons after stroke. Arch Phys Med Rehabil 1993; 74: 133-8.
8. Ware JE, Sherbourne CD. The MOS 36-item Short-Form Health Survey (SF-36). Conceptual framework and item selection. Med Care 1992; 30: 473-83.
9. Harwood RH, Gompertz P, Ebrahim S. Handicap one year after a stroke: validity of a new scale. J Neurol Neurosurg Psychiatry 1994; 57: 825-9.
10. Wade DT. Measurement in neurological rehabilitation. Oxford: Oxford University Press; 1992.
11. Statistics New Zealand. New Zealand Official Yearbook 1998. Wellington: GP Publications; 1998.
12. Donaldson LJ. Health and social status of elderly Asians: a community survey. BMJ 1986; 293: 1079-82.
13. Ebrahim S. Ethnic elders. BMJ 1996; 313: 610-3.
14. Scott KM, Tobias MI, Sarfati D, Haslett SJ. SF-36 health survey reliability, validity and norms for New Zealand. Aust NZ J Public Health 1999; 23: 401-6.
15. Flick CL. Stroke rehabilitatio. 4. Stroke outcome and psychosocial consequences. Arch Phys Med Rehabil 1999; 80: S21-6.
16. Bell C, Swinburn B, Stewart A et al. Ethnic differences and recent trends in coronary heart disease incidence in New Zealand. NZ Med J 1996; 109: 66-8.
17. Smaje C, Grand JL. Ethnicity, equity and the use of health services in the British NHS. Soc Sci Med 1997; 45: 485-96.
18. Wolinsky FD, Aguirre BE, Fann LJ et al. Ethnic differences in the demand for physician and hospital utilization among older adults in major American cities: conspicuous evidence of considerable inequalities. Milbank Q 1989; 67: 412-49.
19. World Health Organisation. The World Health Report 2000. Health Systems: improving performance. Geneva: World Health Organisation; 2000.

The comparability of resource utilisation for Europeans and non-Europeans following stroke in New Zealand

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Abstract

Aims. We sought to explore resource use both in hospital and the community twelve months after hospital discharge for patients of different ethnicity admitted to hospital with acute stroke.

Methods. Resource utilisation data were collected for consecutive patients admitted to each of three general hospitals in the Wellington region over a nine month period. Patients were interviewed, where possible, at three, six and twelve months after hospital discharge. Ethnicity was determined by self report.

Results. Non-Europeans had longer hospital stays than Europeans (median 36 days vs 18 days, p = 0.01). Contact with rehabilitation professionals in the community was low for all groups with no significant differences between Europeans and non-Europeans. For the entire cohort, spending on institutional care was around ten times higher than spending on community rehabilitation in the first twelve months following stroke.

Conclusions. Differences in hospital stay after stroke may reflect problems of access to inpatient rehabilitation services for younger people and not relate directly to the ethnicity of the patient. With the projected increasing proportion of Maori and Pacific people in the population, combined with the aging of that population, health policy-makers need to consider the implications of differences in resource utilisation for different ethnic groups in New Zealand. How to manage all the available resources for people with stroke to maximise outcome remains an important issue for health funders.

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Little reliable information about resource utilisation for different ethnic groups in New Zealand, either in hospital or the community following stroke, is available. Based on national hospital discharge statistics for 1992,[1] the mean length of hospital stay was shorter for Maori than non Maori aged 65 years and over (all causes 12.6 days vs 14.2 days respectively) and specifically for cerebrovascular disease was shorter for Maori than non Maori (19.3 days vs 29.5 days respectively). There are problems using hospital discharge statistics however. As Bonita and colleagues have pointed out, interhospital or intrahospital transfers (to a rehabilitation ward for example) are usually counted as a new admission[2] and the discharge coding for stroke is unreliable.[2,3] In the Auckland Stroke Study in 1991, where strokes were identified prospectively, meeting standard definitions, length of stay was longest for the Maori patients (mean 43 days vs 28 days for Europeans) although the median length of stay (15 days) was the same.[4]

As part of a prospective study of stroke patients admitted to hospital in the Wellington region during 1997, we sought to investigate the comparability of resource utilisation for people from different ethnic groups both in hospital and in the twelve months following hospital discharge.

Methods

The ascertainment of stroke events and recruitment of participants to the study are described in the preceeding paper. Ethnicity was determined by self-report.

Details of hospital stay in different locations were collected prospectively and checked against hospital records. Details of contact with health professionals in the community in the year following stroke were collected by interview at three, six and twelve months following stroke. Participants were asked how many hours of face-to-face contact they had had in the previous two weeks with various health professionals which included physiotherapists, occupational therapists, social workers, psychologists, rehabilitation nurses, district nurses and practice nurses. The hours of contact in the last two weeks was used to calculate an average number of hours for the preceding time period. These data were used to calculate a figure for hours of rehabilitation professional contact over the twelve months following hospital discharge. Figures used in the computation of costs were provided by Capital Coast Health or agencies dealing with the provision of institutional and home care services. Hospital costs are based on per bed day costs of $350 for medical beds and $330 for rehabilitation beds. Community costs are based on a mean rate of $70/hour for therapist time, $13.50 per hour for home help, $14.30 per hour of personal care, $590 per week for rest home care and $942 per week for private hospital care. All costs are in New Zealand dollars. The study was approved by the local Ethics Committee and all participants gave written, informed consent.

Statistical analysis. Non-parametric statistics were used for data not meeting assumptions for normality. Categorical variables were analysed using χ^2 tests. Multiple logistic regression models were used to calculate odds ratios. Each of the baseline and stroke variables were entered into a univariate analysis with significant variables ($p < 0.1$) retained and entered into the multivariate analysis. Subsequent refinement of the model occurred to achieve the best fit with fewest variables. SPSS 10.0 software was used for the analyses.

Results

Details of enrolment, follow-up and outcome are presented in the preceeding paper.

Hospital care. Non-European patients stayed significantly longer in hospital than European patients: median 36 days (IQR [interquartile range] 15, 58) vs 18 days (IQR 7, 44), p = 0.01 (Table 1). Length of stay on the medical wards was also significantly longer for non-Europeans (median 14 vs 8 days for Europeans, p = 0.008). When only hospital survivors are included, differences in median total length of stay were smaller (26 days for Europeans, 36 days for non-Europeans, p = 0.04). Of the non-Europeans, Maori had the longest length of stay (median 53 days). More of the Maori patients that survived (77%) than European survivors (47%) were transferred to inpatient rehabilitation.

Discharge destination (Table 2). There was a trend to more Europeans than non-Europeans being discharged to institutional care (uncorrected odds ratio [OR] 2.6 [95% CI 0.84, 8.0]). With correction for age and initial Functional Independence Measure (FIM) score the odds ratio for discharge to institutional care was similar (OR 3.0 [95% CI 0.80, 11.2]).

Rehabilitation in the community. The amount of health professional contact once discharged from hospital was small

Table 1. Details of hospital stay.

		European	non-European	Maori	Pacific people	Asian	p value*
Number		148	33	13	14	6	
Total LOS	median (IQR)	18 (7, 44)	36 (15, 58)	53 (35, 61)	29 (14,44)	23.5 (7, 75)	0.01
Medical LOS	days	8 (5, 16.5)	14 (8, 25)	14 (11, 27)	15 (7, 18)	8 (5, 35)	0.008
Rehab LOS		28 (14, 52)	36 (15, 47)	41.5 (25, 51)	33 (11, 44.5)	18 (15, 40)	NS
Transferred to rehab: n (%)		69 (47)	21 (64)	10 (77)	8 (57)	3 (50)	NS

LOS = length of stay, IQR = interquartile range, rehab = inpatient rehabilitation, NS = not significant. *comparing European and non-European groups.

but there was no difference between European and non-Europeans (Table 3). The majority of people by three months post-discharge received no input from therapists or nurses. Maori people received more hours of health professional contact once in the community than the other groups (median 13.7 hours vs zero hours in the other groups, p = 0.015). Mean hours of contact for the groups were Maori (32 hours), European (13.1 hours), Pacific people (4.2 hours) and Asians (zero hours). However, the numbers in these subgroups were small and the data could be readily skewed by a small number of aberrant cases.

Table 2. Hospital discharge destination for survivors from different ethnic groups.

	Private residence	Institutional care
Maori	10 (83.3)	2 (16.7)
Pacific people	12 (92.3)	1 (7.7)
Asian	5 (83.3)	1 (16.7)
All non-European	27 (87.1)	4 (12.9)
European	83 (72.2)	32 (27.8)

$\chi^2 = 2.9$, p = 0.09 (non-European vs European).

Costs (Table 4). The observed difference in hospital costs (mean $9600 for Europeans vs $13 600 for non-Europeans, p = 0.01) mirrors length of hospital stay as this is how hospital costs were computed. There were no significant differences in community costs between non-Europeans and Europeans.

Discussion

The only significant difference in resource use between Europeans and non-Europeans was with hospital length of stay, also reflected in hospital costs. Non-Europeans, particularly Maori, had a longer stay. However, interpretation of this and other results requires caution.

The numbers were small and follow-up of non-European groups was limited (73% compared to 98% for Europeans) with a tendency for less disabled non-Europeans not to be followed up successfully, potentially overestimating community resource use for non-Europeans. As described in the preceeding paper, there was an unexpectedly higher survival for non-Europeans compared to Europeans. Surviving non-Europeans had more severe strokes (lower Functional Independence Measure scores in the first week) than surviving Europeans, again perhaps overestimating the relative community resource use of non-Europeans. A significant weakness of the study design in collecting information regarding community resource use was that the first assessment was at three months after discharge. Many people may have received some input from rehabilitation professionals between hospital discharge and three months but if this contact ceased by about ten weeks it would not have been recorded. A subsequent (unpublished) investigation of a further cohort of similar patients with stroke discharged from Wellington Hospital in 2000 indicates that many patients receive little formal rehabilitation post hospital discharge with a range of zero to ten hours (median three hours) in the first ten weeks. Effectively, what we have described here are longer term efforts at rehabilitation extending from ten weeks to twelve months after discharge which will have underestimated total rehabilitation contact in the community by a few hours per patient.

It is difficult to compare directly our results for community rehabilitation with those of the 1981 Auckland Stroke Study.[5] In that study, around 25% of stroke patients referred for outpatient physiotherapy were still receiving input from a physiotherapist six months after their stroke and 25% of patients with a speech disturbance were still receiving speech language therapy six months after their stroke. In our study, 16% of people alive and assessed at six months had had face-to-face contact with a physiotherapist in the previous two

Table 3. Resource utilisation in the community in the twelve months following stroke.

	European	non-European	Maori	Pacific people	Asian	p value*
n	96	22	8	10	4	
Total hours:						NS
mean	13.1	13.5	32	4.2	0	
median (IQR)	0 (0, 13)	0 (0, 6.5)	13.7 (2.6, 55.9)	0 (0, 0)	0 (0, 0)	
Nurshing hours:						NS
mean	4.0	4.1	8.1	2.6	0	
median (IQR)	0 (0, 0)	0 (0, 0)	0 (0, 13)	0 (0, 0)	0 (0, 0)	
Therapist hours:						NS
mean	9.1	9.4	23.9	1.6	0	
median (IQR)	0 (0, 5.2)	0 (0, 6.5)	7.15 (2.6, 36.4)	0 (0, 0)	0 (0, 0)	

IQR = interquartile range, NS = not significant. *comparing European and non-European groups.

weeks with the corresponding values for other professionals being: occupational therapy (10%), social work (2%) and speech language therapy (1%). These differences may reflect geographical differences in service provision or, more likely, changes to service provision over the last 20 years as community rehabilitation budgets have become increasingly constrained. Given the much higher proportion of community spending for stroke on institutional care rather than rehabilitation (Table 4), reducing spending on community rehabilitation may represent false economy.

Table 4. Comparison of costs in hospital and community.

Cost: Mean (95% CI) $NZ 000's	European	non-European	p value
Hospital	9.59 (8.14, 11.05)	13.56 (10.32, 16.80)	0.01
Community:			
Rehabilitation	0.62 (0.35, 0.90)	0.86 (0.28, 1.44)	NS
Institutional care	6.81 (4.42, 9.19)	7.65 (3.12, 12.17)	NS
Home help/personal care	1.32 (0.34, 2.30)	1.29 (0.47, 2.10)	NS
Total community cost	8.75 (6.17, 11.32)	9.79 (5.27, 14.31)	NS
Total 1 year cost	18.34 (14.89, 21.79)	23.35 (17.24, 29.47)	0.01

The longer stays in medical wards for non-Europeans could reflect a problem of access into rehabilitation wards for those aged less than 65 years. These patients were admitted to one of two specific rehabilitation wards for younger people. The median medical length of stay for patients admitted to these wards was substantially longer than for those admitted to rehabilitation wards for people aged 65 years and over (18 days vs 9 days, p = 0.02). As 45% of the non-European patients were less than 65 years old compared to 16% of the European patients, and a higher proportion of non-Europeans received inpatient rehabilitation, this could explain much of the difference in medical length of stay. Modelling total length of hospital stay in hospital survivors with multiple linear regression, adjusting for age and transfer to inpatient rehabilitation, the difference between Europeans and non-Europeans was non-significant (β = 5.8, 95% CI -3.8, 15.4).

Accepting that these data are far from definitive, they still raise important questions for health planners. Both Maori and Pacific populations are increasing relative to the European population in New Zealand.[6] Improving survival for elders of these communities suggest that stroke and its consequences will become an increasing problem. Predicting hospital bed use for people with stroke in the future is a priority given the increasing trend to reduce total numbers of hospital beds. Any systematic differences in bed usage and resource use in the community related to ethnicity need to be fully explored to allow accurate predictions to be made in the future and possible interventions designed to ensure equity of access and equity of outcome for all ethnic groups in New Zealand.

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1. Pomare EW. Hauora: Maori standards of health III: a study of the years 1970 - 1991. Wellington: Te Ropu Rangahau Hauora a Eru Pomare; 1995.
2. Bonita R, Beaglehole R. Hospital bed use by stroke patients: the Auckland stroke study. N Z Med J 1988; 101: 497-9.
3. Davenport RJ, Dennis MS, Warlow CP. The accuracy of Scottish Morbidity Record (SMR1) data for identifying hospitalised stroke patients. Health Bull 1996; 54: 402-5.
4. Bonita R, Broad JB, Beaglehole R. Ethnic differences in stroke incidence and case fatality in Auckland, New Zealand. Stroke 1997; 28: 758-61.
5. Bonita R. Rehabilitation services received by stroke patients: the Auckland stroke study. N Z Med J 1988; 101: 595-7.
6. Statistics New Zealand. New Zealand Official Yearbook 1998. Wellington: GP Publications, 1998.

Observations of summer sun protection among children in New Zealand: 1998-2000

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Abstract

Aims. To examine sun protection among New Zealand children (ten years and under) at beaches and playgrounds.
Methods. In the summers of 1998, 1999 and 2000, observations were made of 753 children at selected beaches and playgrounds in Dunedin and Hawkes Bay to determine the extent of sun protection. Parents/carers were also interviewed about sun protection.
Results. Across most body sites, levels of protection were high. Clothing and SPF15+ sunscreen were used most often, while the use of shade was relatively low. About four in every ten parents/caregivers were aware of the UV index or burn-time. Level of sun protection among children at beaches was best predicted by parent/carer's own level of protection, the child's perceived susceptibility to burning, and being a preschooler.
Conclusions. Observed levels of sun protection among the children were high and most likely reflected an increased awareness among parents and carers of the social acceptability of protective behaviours. Nevertheless, opportunities to seek shade were limited, and the provision of shade at beaches and playgrounds represents an important next step in a community wide approach to sun protection.

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The prevention of sunburn, especially among children, is a prime focus of community efforts to reduce the incidence of melanoma in New Zealand. For young children, sun exposure and sunburn are the key modifiable risk factors for melanocytic naevi, which represent the most important risk factor for melanoma.[1]

In collaboration with the Cancer Society of New Zealand, the Social and Behavioural Research in Cancer Group (SBG) initiated surveys of sun exposure and protection among young children, particularly those ten years and under.[2-5] This research suggests that nine out of ten children are outdoors on any summer saturday and/or sunday for about two hours,

and one in fourteen children experience sunburn causing blistering of the skin or prolonged pain.[2] Over summer, about one in three children experience sunburn at least once, mostly on the face, nose, ears and neck.[5] About half of the children wear some sunscreen, while about six in ten wear a hat or a cap. Many parents, however, continue to hold attitudes such as their child "looks healthier with a tan," which may promote harmful exposure to the sun. Furthermore, low levels of sun protection and sunburn in children were significantly associated with lower parental use of protection and parental sunburn.[2,4]

Overall, our research indicates a relatively high degree of sun protection and low extent of sunburn for young children, in comparison with adolescents and adults.[6-9] One shortcoming of this research is its reliance on parental report. Direct observations of children might address concerns about recall accuracy and possible adult reluctance to report behaviours which may reflect a lack of child sun protection.[10] This paper examines patterns and correlates of sun protection among young children at beaches and playgrounds in Dunedin and beaches in Hawkes Bay, using a combination of direct observation and parent/carer interview.

Methods

Dunedin surveys 1998 and 2000. In Dunedin (population 120,000), the sample consisted of children aged ten years and under present at selected beaches and parks/playgrounds between 14th and 31st January 1998, and 7th and 17th January 2000. The same three beaches and three playgrounds were chosen each summer. A fourth playground near one beach was included in 2000. Observations were carried out by a trained interviewer between 12.00 noon to 2.30 pm to capture the peak period for UV exposure. Each summer, a different person carried out the observations. Days were selected according to the prevailing weather (at least 15°C at 10.00am and absence of heavy cloud).

All adults accompanied by young children were approached to take part. If they agreed, sociodemographic information (age, ethnicity and place of residence) was obtained, together with time spent at that location; knowledge of burn-time and/or the UV Index (UVI) for the day; and each child's age. The parent/carer was asked to imagine the effect on the child's skin of 30 minutes unprotected sun exposure for the first time in summer. Susceptibility to burning was defined by either "get severe sunburn with blistering" or "have painful sunburn." Frequency and severity of any sunburn (skin red, sore or blistered) in the current summer was assessed. These questions, based upon earlier surveys,[5] were designed to be answered in a few minutes.

Sun protection (hat/cap, clothing, SPF 15+ sunscreen, and shade) of each child was noted for different body areas (Table 3). Protection of hands, back of knees and feet was added in 2000. If sunscreen was used, SPF rating, time of first application and any re-application(s) were noted. The procedures were similar to those of Hill and colleagues in Victoria, Australia (personal communication). A separate form was completed for each child. Air temperature was measured at the time of each observation and cloud cover (rated as none, thin, thick, overcast thin and overcast thick), and wind (none, weak and strong) were noted.

Hawkes Bay survey 1999. A similar study was carried out at four Hawkes Bay beaches, in collaboration with the Public Health Unit, Health Care Hawkes Bay. The sample consisted of children aged 10 years and under present at the beaches between 27th December 1998 and 14th February 1999. Observations were carried out by three trained observers between 11.00 am and 4.00 pm. Days were selected with temperature > 18°C at 10.00am and no heavy cloud. Child behavioural observations were made as in the Dunedin surveys, however, sun protection observations were also coded for the parents/carers.

Results

Observational occasions. Table 1 shows characteristics of the three observation occasions. Overall, 372 parents/carers participated in the surveys and 753 children were observed. There were very few refusals; e.g. only two adults in the 1999 survey and five in the 2000 survey. There were some differences among the surveys in terms of prevailing weather, (and there is evidence that sun protection varies with weather conditions).[11] Thus, statistical comparisons across the surveys are problematic, and were not made for this reason.

Table 1. Characteristics of observation occasions in 1998, 1999 and 2000*.

	1998	1999	2000
Observation occasions (N)	14	12	13
Parents/carers approached (N)	116	115	141
Children observed (N)	217	237	299
Parents/carers approached at beaches	47.4%	100%	47.5%
Average temperature	23.8°C	24.6°C	17.9°C
Cloudless sky	21.6%	71.7%	38.1%
No wind	33.6%	10.5%	15.2%

*1998 and 2000 Dunedin; 1999 Hawkes Bay.

Characteristics of participants. Table 2 shows parent/carer and child characteristics. Most children were accompanied by their mother or a female carer. Most parent/carers were under 40 years of age; in the Hawkes Bay sample, about one-quarter self-identified as Maori. There were equal numbers of boys and girls. About two-thirds of the children had skin susceptible to burning, and many had already been sunburnt that summer. In the Dunedin samples, two-thirds of the children had been in the sun for less than one hour. In Hawkes Bay nearly 60% had been in the sun for more than one hour, which may reflect the longer observation period there. In the two more recent surveys 36% and 42% of parents/carers reported having taken note of the UVI or burn-time reported in the media.

Table 2. Characteristics of participants in the 1998, 1999 and 2000 surveys.

		1998	1999	2000
Parent/carer				
Sex:	Female	--	82.6%	73.8%
Age:	< 30 yr	19.8%	30.7%	32.1%
	30-39 yr	61.2%	53.5%	46.4%
	> 40 yr	19.0%	15.8%	21.4%
Ethnicity:	NZ European	91.4%	73.0%	90.0%
	NZ Maori	2.6%	23.5%	8.6%
	Other	6.0%	3.5%	1.4%
Noted UV index/burn-time		13.9%	35.7%	41.8%
Children				
Sex:	Female	52.1%	51.1%	48.8%
Age (average yrs)		4.3	5.7	5.2
Exposure:	< 1 hr	63.8%	42.6%	67.4.%
	1-2 hr	25.9%	32.1%	24.1%
	> 2 hr	10.3%	25.3%	8.5%
Susceptible to sunburn (severe/painful burn)		66.8%	59.5%	63.5%
Summer sunburn		23.9%	39.7%	16.3%
Sore/blistered sunburn		8.0%	18.4%	5.4%
Sunburned > once		4.7%	16.2%	2.4%

Sun protection. Table 3 shows the extent of adequate sun protection by use of shade, hat, clothing or SPF15+ sunscreen at each body site. Protection of the trunk (shoulders, back, chest and stomach) and the upper arm and upper leg was very high in all three surveys. Protection was less for the scalp, ears, neck, lower arms, hands and lower legs than the other sites. Protection of the face and nose was also low in the Dunedin surveys.

Sunscreen was the most popular form of protection. In 1998, 50.9% of the children were wearing sunscreen compared with 62.2% in 2000. In Hawkes Bay, 87.3% of children were wearing sunscreen. This was almost universally SPF15 or higher and about two-thirds of parents/carers reported that it had been applied before the children went outside in the sun. Among children outside for longer than two hours, most had sunscreen reapplied. On the other hand, there was little use of shade. For example, the Dunedin

findings indicated that in 1998, only twelve children (5.5% of the total sample) were in total shade; in 2000, this had increased to 24 (8.1%). Some of these children were infants in covered prams, strollers or bassinets.

Table 3. Sun protection across body sites in 1998-2000.

	Adequate protection*		
Body site	1998	1999	2000
Scalp	27.6%	65.4%	36.1%
Face	59.9%	90.3%	69.7%
Nose	59.4%	89.8%	69.7%
Ears	49.8%	77.4%	69.4%
Neck	60.8%	85.5%	69.6%
Shoulder	95.9%	93.2%	98.7%
Back	98.2%	92.8%	99.0%
Upper arm	98.2%	83.7%	98.3%
Lower arm	74.7%	74.0%	86.9%
Chest	98.6%	93.6%	99.0%
Stomach	98.2%	93.5%	98.7%
Upper leg	99.1%	88.5%	99.3%
Lower leg	58.1%	66.5%	83.7%
Hands		60.2%	65.0%
Back of knees		64.8%	89.6%
Feet		49.8%	88.8%

*One or more of shade, clothing SPF15+ sunscreen.

Correlates of sun protection. Following Foot et al,[10] each of nine body regions was assigned points for protection (hat/clothing, SP15+ sunscreen and/or shade protection) reflecting comparative risks of melanoma at particular body sites. Protection of the face, back and legs received a score of three each; the shoulders and upper arms a score of two each; while the neck, lower arms, chest and stomach received one each. This provided a "sun protection" score from 0-17 with maximum protection at 17, a high level of protection from 13-16, and low protection from 0-13.

The Hawkes Bay data were used to model child sun protection because they allowed the inclusion of the parent/carer's sun protection score in the regression models.[12] 64.6% children had maximum protection; 12.7% had a high level of protection; and 22.7% had low protection. Three sets of predictors were examined. The "environmental variables" included temperature (< 24ºC vs 24ºC or above), cloud, wind, and length of time outside (< 1 hr, 1-2 hr or > 2 hr). The 'parent/carer variables' included age (< 29 years vs 30 years or above), sex, ethnicity (Maori/Pacific Islander vs NZ European); own sun protection; and awareness of the UVI or burn-time. Finally 'child variables' included age, sex, sensitivity to sunburn, and prior experience of sunburn. In the final model the effect of each variable was adjusted for the other variables, and for data 'clustering' in that one parent/carer may have been responsible for more than one child.

The model suggested that higher sun protection among children was associated with the parent/carer having a sun protection score of 17 (adjusted OR = 5.16 with 95% Confidence Interval 1.94 – 13.43); the child being susceptible to sunburn (adjusted OR = 4.53; 95% CI 2.11 – 9.71); the child being younger than five years (adjusted OR = 3.95; 95% CI 2.10 – 7.41); a non-Maori parent/carer (adjusted OR = 3.07; 95% CI 1.39 – 6.78); and the child being female (adjusted OR = 2.01; 95% CI 1.07 – 3.78). These odds ratios are proportional odds and estimate the increased odds of protection as the child moved from a lower to the next higher level of sun protection. Awareness of the UVI or burn-time was not significantly associated with the child's sun protection, but was associated with the parent/carer level of protection.

Discussion

As far as we are aware, these are the first observational studies of sun protection among New Zealand children. Participation by parents/carers was very high with only a handful refusing to take part, as was the case in the Australian study.[10] We believe parents and carers were willing to participate because of the importance they attach to child sun protection. Given our concerns about differences between the surveys in weather and participant characteristics, it is more appropriate to regard the surveys as three snapshots of sun protection in situations where unprotected children were at risk of sunburn.

So what do the results suggest? First, levels of sun protection were relatively high. In the two most recent surveys, about two-thirds of the children had maximum protection scores. Protection was also highest among preschool children, those susceptible to sunburn, and those whose parent/carer had maximum sun protection. In Hawkes Bay, the latter two variables showed the strongest associations with the child's protection score. Across different body sites, the trunk, as well as upper arms and legs all had adequate protection in over 90% children observed. The lower areas of limbs and the neck, nose, ears and face were less adequately protected. The scalp, however, was the least protected in all three surveys, suggesting the wearing of hats by young children needs continuing emphasis.

Sunscreen use was high with 60% children wearing SP15+ sunscreen in the 2000 survey and 90% in the 1999 survey. The sunscreen had been applied usually before going outside and re-applied where the children had been in the sun for more than two hours. The extent of sunscreen use is comparable to that reported by parents, retrospectively.[2] While use of sunscreen is controversial in New Zealand,[13] appropriate re-application probably offers some protection against burning.[8] In the present study, parents/carers appeared to be using sunscreen appropriately. Few children were protected by shade and this was not unexpected, given that there were no artificial shade structures at any of the Dunedin playgrounds, and trees provided only partial shade at two. At the various beaches there were no convenient sources of shade. So, parents and carers at most locations had to rely on clothing and sunscreen to protect their children.

The Cancer Society of New Zealand has been promoting sun protection for over 20 years, most recently the protection of young children by their parents.[14] The high levels of protection we observed should be heartening in terms of efforts to raise parental awareness. They are also consistent with reports by parents of the social acceptability of sun protection behaviours, the high recall of sun protection messages, and the better availability of more protective clothing.[15] Awareness of UVI or burn-time was also relatively high at about 40% of parent/carers, a finding consistent with a national survey showing high awareness of sun protection messages in the media, especially among sun-sensitive individuals.[16]

While sun protection among children at playgrounds and beaches is relatively high, the wearing of hats needs to be further encouraged. Continuation of current programmes is needed to maintain these high levels. At the same time, however, many parents hold attitudes that favour their children having a tan.[4] Such attitudes need to be countered, especially as about 10% children had already experienced a sore blistering sunburn over the summer. To a very large degree, the sun protection levels observed among the children represented the efforts of adults and children themselves to adopt protective behaviours. This comes at some cost in terms of time, money and effort.[15] There is a clear need for the promotion and provision of shade at beaches and playgrounds, especially in the light of increases over recent summers in sunburning ultraviolet radiation in New Zealand.[17]

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1. Harrison SL, MacLennan R, Speare R et al. Sun exposure and melanocytic naevi in young Australian children. Lancet 1994; 344: 1529-32.
2. McGee R, Williams S, Glasgow H. Sunburn and sun-protection among young children. J Paediatr Child Health 1997; 33: 234-8.
3. Morris J, Bandaranayake M, McGee, R. Children's understanding of sun protection behaviours: a comparative analysis. J Paediatr Child Health 1998; 34: 254-9.
4. Morris J, McGee R, Bandaranayake M. Sun protection and the predictors of sunburn among young children. J Paediatr Child Health 1998; 34: 557-62.
5. McGee, R. Sun exposure and sun protection among New Zealand children. In: UV radiation and its effects - an update. Wellington: Royal Society of New Zealand (Miscellaneous Series no. 49); 1998.
6. McGee R, Williams S. Adolescence and sun protection. N Z Med J 1992; 105: 401-3.
7. Richards R, McGee R, Knight RG. Sun protection practices, knowledge and attitudes to tans among New Zealand adolescents, 1991 – 1997. NZ Med J 2001; 114: 229-31.
8. Richards R, McGee R, Knight RG. Sunburn and sun protection amongst New Zealand adolescents over a summer weekend. Aust NZ J Public Health 2001; 25: 352-4.
9. McGee R, Williams S, Cox B et al. A community survey of sun exposure, sunburn and sun protection. N Z Med J 1995; 108: 508-10.
10. Foot G, Girgis A, Boyle CA et al. Solar protection behaviours: a study of beachgoers. Aust J Pub Health 1993; 17: 209-14.
11. Dunn J, Lynch B. Climatic conditions and the reporting of sun-protective behavior survey data: a brief report. Am J Health Promot 2001; 15: 241-3.
12. Hosmer DW, Lemeshow S. Applied logistic regression. New York: John Wiley and Sons; 1989.
13. Bulliard JR. Determinants of sunburn and measures of sun exposure in New Zealand. In: UV radiation and its effects - an update. Wellington: Royal Society of New Zealand (Miscellaneous Series no. 49); 1998.
14. Kill B. Review of melanoma prevention programme: a report for the Cancer Society of New Zealand. Wellington: Invicta Associates; 1995.
15. Reeder AI, McAllister S, Bulliard JL. Child sun protection in New Zealand: parental views and societal responsibilities. Health Promot J Aust 2000; 10: 217-23.
16. Bulliard JL, Reeder AI. Getting the message across: sun protection information in media weather reports in New Zealand. N Z Med J 2001; 114: 67-70.
17. McKenzie R, Connor B, Bodeker G. Increased summertime UV radiation in New Zealand in response to ozone loss. Science 1999; 285: 1709-11.

Awareness, knowledge and attitudes of lead maternity carers towards early-onset neonatal group B streptococcal disease

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Abstract

Aim. To determine awareness, knowledge and attitudes of lead maternity carers (LMCs) towards early-onset neonatal group B *streptococcus* (GBS) infection and its prevention.

Methods. An anonymous, self-administered questionnaire was sent to the 155 practising LMCs in Wellington and Hutt Valley.

Results. Completed questionnaires were returned by 84 (54%) LMCs (59 midwives). 66 (79%) believed perinatal GBS infections were important, 70 (85%) supported antenatal screening, while 68 (81%) were confident of determining risk factors for GBS infection and counselling women. However, less than one-third nominated major risk factors, none identified all five high-risk criteria and only 22 (26%) regularly discussed GBS with clients. When asked to name high-risk criteria, midwives were more likely than doctors to disclose they had incomplete knowledge or not to answer this item (53% vs 20%; $p < 0.006$). Of the 48 (57%) LMCs routinely employing GBS prevention strategies, 34 (71%) used culture-based screening, relying mainly upon high-vaginal swabs from the first-trimester.

Conclusions. Despite widespread awareness of perinatal GBS disease, only 57% of surveyed LMCs practised prevention strategies and none completely followed published recommendations. A New Zealand consensus or improved dissemination of local guidelines is required to achieve further reductions in neonatal GBS sepsis.

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Group B *streptococcus* (GBS) is the leading cause of early-onset neonatal sepsis.[1] It is usually acquired during labour from the maternal genital tract. As intrapartum antibiotics interrupt vertical transmission, early-onset GBS infection is now believed to be a preventable disease.[2] Women at high-risk of delivering affected infants can be identified by either detecting GBS carriers or by recognition of certain obstetric factors. North American consensus guidelines provide the standard of care, advocating intrapartum antibiotics for women at high risk of delivering an infant with GBS disease.[3] Although national guidelines have not been developed in New Zealand, several obstetric units have formulated their own prevention strategies based upon this consensus model of care. There are limited data from selected Australian and New Zealand centres to show that with introduction of prevention strategies between 1991-1997 the incidence of early-onset-GBS disease fell from 2.0 to 0.5 cases per 1000 live births.[4] A more recent New Zealand-based study confirmed the rate of early-onset GBS sepsis as 0.5 per 1000 and that 14 of 19 (74%) obstetric centres associated with level II/III neonatal units had formal GBS prevention policies.[5] Nevertheless, further improvements are possible as this study also showed that nearly 60% of infants with GBS sepsis had risk factors but did not receive intrapartum antibiotics. We therefore surveyed lead maternity carers (LMCs) in the Wellington and Hutt regions to learn of their awareness, knowledge and attitudes towards early-onset neonatal GBS infection and its prevention.

Methods

The Health Funding Authority identified all practising LMCs, including obstetric/maternity educators and home birth midwives in the Wellington/Hutt regions. During August 1999 an anonymous, self-administered seven-page postal questionnaire with reply-paid addressed envelopes was mailed to 155 LMCs, followed by a reminder letter two-weeks later.

The questionnaire consisted of three main sections: demographic, management and antenatal screening. Five-point Likert scales assessed attitudes, while closed and open-ended items determined respondent knowledge and awareness of early-onset neonatal GBS infection. Free-text fields encouraged feedback and comment. Several midwives at Wellington Women's Hospital piloted the questionnaire prior to its distribution. Items in the questionnaire that also assessed awareness of human immunodeficiency virus infections in pregnancy have been reported previously.[6] Statistical analyses were performed using the Statistical Package for Social Sciences (Version 9, SPSS Inc. Chicago, USA). Group comparisons were made by χ^2 tests. Significance was set at $p < 0.05$. The Wellington Ethics Committee approved the survey.

Results

Completed questionnaires were returned by 84 (54%) of 155 LMCs surveyed. Of 84 respondents, 59 (70%) were midwives, 18 (22%) were general practitioners and 7 (8%) were obstetricians. Overall, there were 67 (80%) females, 51 (61%) were aged > 40 years, 43 (51%) had overseas training and 52 (62%) had practised antenatal care for ≤ 10 years.

66 respondents (79%) believed perinatal GBS infections were an important public health issue and 70 (85%) indicated antenatal testing for GBS colonisation was desirable. While 25 (30%) thought no barriers existed for routine antenatal GBS screening, consent, counselling and time constraints were each raised by nine (11%) respondents, and another eight (10%) each mentioned antibiotic resistance and creating anxiety. Although 68 (81%) felt comfortable assessing risk factors, discussing screening tests and counselling clients, just 29 (35%) had educational material available and only 22 (26%) regularly broached the subject of GBS during the third trimester. There were no significant differences between respondents according to age, profession, time spent in antenatal care or country of training.

Table 1 outlines the major risk factors identified by LMCs. Overall, 29% reported a previously affected infant as being at high-risk for perinatal GBS infection. However <25% recognised other obstetric factors in GBS prevention strategies. None identified all five high-risk criteria. Midwives were significantly more likely than doctors to acknowledge they were unaware of risk factors (11 vs 1) or not to answer this item (20 vs 4; p <0.006).

Table 1. Risk factors for early-onset neonatal GBS infection identified by 84 Wellington and Hutt lead maternity carers.

Major risk factor during labour[3]	Midwives (n = 59) n	%	Doctors (n= 25) n	%	P-Value
Prior GBS affected infant	15	25	9	36	0.33
GBS bacteruria this pregnancy	0	0	1	4	0.30
Preterm delivery	7	12	6	24	0.16
Prolonged membrane rupture	6	10	6	24	0.10
Intrapartum fever	2	3	3	12	0.15
Other *	12	20	5	20	0.97
Unsure or no response	31	53	5	20	0.006

* Included vaginal discharge, sexually transmitted infections, multiple sexual partners, socio-economic status and ethnicity.

48 of 84 (57%) LMCs surveyed, routinely employed prevention strategies during the previous twelve-months. Of these, 34 (71%) performed antenatal screening, eleven (23%) used risk factor assessment and three (6%) administered intramuscular penicillin to the newborn infant. However, at least two-thirds nominating a culture-based screening approach used high vaginal cultures from the first trimester to determine if intrapartum antibiotics were indicated. When asked of their preferred prevention strategy if all proved cost-effective, 51 (61%) LMCs indicated antenatal screening, 23 (27%) identified risk-based assessment and ten (12%) remained unsure or believed it was the women's choice. In addition, two midwives and one doctor wished to continue postnatal penicillin prophylaxis. Responses by doctors and midwives were similar and did not differ by age, experience or country of training.

Discussion

Despite awareness of the public health importance of early-onset neonatal GBS infection, fewer than 60% of LMCs surveyed in the Wellington/Hutt region routinely practised a prevention strategy or shared this information with their clients. There was also incomplete knowledge of GBS prevention strategies[3] and considerable variation in practice. Although understandable from the limited robust studies comparing specific strategies,[7,8] this meant that few LMCs maximised the prevention of early-onset GBS disease.

The current study compares with views of Australian obstetricians where 56% support universal antenatal screening and almost all believe in risk-based assessment.[9] Surveys from the United States also show widespread support for GBS prevention strategies with more than 90% of obstetric providers having an individual policy.[10] In contrast to our findings, approximately 80% of those surveyed in the United States who used the screening-based strategy reported obtaining cultures within one week of the recommended 35-37 week's gestation. When employing a risk-based approach a similar proportion indicated that they would administer intrapartum antibiotics for all five of the high-risk criteria.

With only partial implementation of prevention strategies in New Zealand and almost 60% of affected infants representing missed opportunities for prevention, additional reduction in the incidence of early-onset neonatal GBS sepsis is possible.[5,11] Both screening and risk-based strategies rely upon accurate specimen collection and risk-recognition. Of concern is that most surveyed LMCs used high vaginal swabs in the first trimester when anogenital cultures between 35-37 weeks gestation best predict GBS colonisation of women delivering at term.[12] Indeed, few were aware of the major risk factors for early-onset neonatal GBS infection and some nominated factors believed to be unimportant. The practice of postnatal penicillin prophylaxis favoured by a minority of LMCs is no longer recommended, as this is associated with increased overall mortality from penicillin-resistant organisms.[13] These observations highlight the value of developing national consensus guidelines and widely disseminating educational programmes.

Our findings are subject to at least three limitations. First, because the survey was conducted in one region the results may not apply to the rest of New Zealand. Additionally, while the response rate of 54% is comparable with other studies,[9] different characteristics between responders and non-responders of motivation and interest may introduce bias. Improved response rates may have been achieved by a shorter questionnaire, focussing solely upon GBS infection and removing anonymity to allow follow-up communication with non-respondents. Second, at the time of the survey the two main obstetric centres within the region relied upon individual best practice and have only recently introduced GBS prevention policies. This contrasts with some other centres where guidelines have been disseminated to local LMCs. Nonetheless, hospital policy is not necessarily a good guide to individual practice.[14] Moreover, a recent survey of nineteen obstetric units responsible for delivering almost three-quarters of the nation's annual birth cohort found that local guidelines often did not follow overseas recommendations (manuscript in preparation). Finally, the questionnaire measured only reported practices of LMCs and not the services provided.

The optimal GBS prevention strategy for New Zealand is yet to be determined. Consensus practice guidelines developed in Australia, supported by health economic analyses,[15,16] recommend intrapartum antibiotics be used only for women with defined GBS risk factors and not to rely upon culture-based screening methods.[17] It is important that either a New Zealand consensus is developed soon or local guidelines are better defined and disseminated.

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1. Isaacs D, Barfield CP, Grimwood K et al. Systemic bacterial and fungal infections in Australian neonatal units. Med J Aust 1995; 162: 198-201.
2. Boyer KM, Gotoff SP. Prevention of early-onset neonatal group B streptococcal disease with selective intrapartum chemoprophylaxis. N Engl J Med 1986; 314: 1665-9.
3. Centers for Disease Control. Prevention of perinatal group B streptococcal disease: a public health perspective. Morb Mortal Wkly Rep 1996; 45(RR-7): 1-24.
4. Isaacs D, Royle JA for the Australasian Study Group for Neonatal Infections. Intrapartum antibiotics and early-onset neonatal sepsis caused by group B *streptococcus* and by other organisms in Australia. Pediatr Infect Dis J 1999; 18: 524-8.
5. Grimwood K, Darlow BA, Gosling IA et al. Early onset neonatal group B streptococcal infections in New Zealand 1998-1999. J Paediatr Child Health 2002; 38: (in press).
6. Gosling I, Grimwood K, Dickson N. HIV awareness during pregnancy. NZ Med J 2000; 113: 321-2.
7. Rosenstein NE, Schuchat A, Neonatal GBS Study Group. Opportunities for prevention of perinatal group B streptococcal disease: a multistate surveillance analysis. Obstet Gynecol 1997; 90: 901-6.
8. Main EK, Slagle T. Prevention of early-onset invasive neonatal group B streptococcal disease in a private hospital setting: The superiority of culture-based protocols. Am J Obstet Gynecol 2000; 182: 1344-54.
9. McLaughlin K, Crowther C. Universal antenatal group B *streptococcus* screening? The opinions of obstetricians and neonatologists within Australia. Aust NZ J Obstet Gynaecol 2000; 40: 338-40.
10. Lynfield R, White K, Danila R et al. Adoption of perinatal group B streptococcal disease prevention recommendations by prenatal-care providers – Connecticut and Minnesota, 1998. Morb Mortal Wkly Rep 2000; 49: 228-32.
11. Schrag SJ, Zywicki S, Farley MM et al. Group B streptococcal disease in the era of intrapartum antibiotic prophylaxis. N Engl J Med. 2000; 342: 15-20.
12. Yancey MK, Schuchat A, Brown LK et al. The accuracy of late antenatal screening cultures in predicting genital group B streptococcal colonization at delivery. Obstet Gynecol 1996; 88: 811-5.
13. Benitz WE, Gould JB, Druzin ML et al. Antimicrobial prevention of early-onset group B streptococcal sepsis: estimates of risk reduction based on a critical literature review. Pediatrics 1999; 103/6/e78.
14. Ansell P, Roman E, Fear NT, Renfrew MJ. Vitamin K policies and midwifery practice: questionnaire survey. BMJ 2001; 322: 1148-52.
15. Gilbert GL, Isaacs D, Burgess MA et al. Prevention of neonatal group B streptococcal sepsis: is routine antenatal screening appropriate? Aust NZ J Obstet Gynaecol 1995; 35: 120-6.
16. Garland SM, Kelly N. Early-onset neonatal group B streptococcal sepsis: economics of various prevention strategies. Med J Aust 1995; 162: 413-7.
17. Oats JJN. Routine antenatal screening: a need to evaluate Australian practice. Med J Aust 2000; 172: 311-2.

CASE REPORT

Vibrio vulnificus necrotising fasciitis and septicaemia

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A 79 year old man presented to Middlemore Hospital in February 2001 having been found confused and febrile. The previous day he had sustained a superficial laceration to his left foot while swimming in the Hauraki Gulf off Waiheke Island. Past medical history included diabetes mellitus, stable angina and mild asthma.

On examination he was tachycardic, hypotensive, febrile (39°C) and had a decreased level of consciousness. There was a small laceration on the dorsum of the left foot, and cellulitis of the left lower leg. Necrotising fasciitis was not apparent clinically.

Abnormal laboratory results included a mild thrombocytopenia, a neutrophilia (13.2 x10^9/L), mild renal impairment and hypoxia.

He received intravenous (IV) fluid resuscitation, and IV ceftriaxone 2g, penicillin 1.2g and metronidazole 500mg. Over the next few hours the area of cellulitis enlarged and his condition deteriorated. He went to the operating theatre for surgical debridement of necrotic fat and fascia, and compartment fasciotomies. Post operatively in the Intensive Care Unit, he received noradrenaline, further IV fluid resuscitation, IV flucloxacillin 2g 6-hourly, clindamycin 600mg 6-hourly, and ciprofloxacin 400mg 12-hourly for 24 hours then 200mg 12-hourly.

Twelve hours after admission a gram negative bacillus was isolated from blood cultures and tissue specimens. It was identified as *Vibrio vulnificus*, susceptible to trimethoprim/sulphamethoxazole, amoxycillin, ciprofloxacin and cefuroxime, and intermediate to gentamicin. Specific therapy with ciprofloxacin was continued for a total of fourteen days.

Further surgical debridement was performed on the second day of admission; tissue specimens were sterile. The initial site of infection healed after skin grafting. His course in the Intensive Care Unit was complicated by renal failure requiring haemodialysis, arrhythmias, and nosocomial infections. Three months into his admission, he suffered a cardiac arrest and died.

Discussion

V. vulnificus is found in estuarine and coastal waters worldwide, and is part of the normal flora of molluscan shellfish in the summer months. Optimal growth occurs between 20ºC and 40ºC, and no growth occurs at temperatures less than 10ºC.[1-4]

It is an uncommon human pathogen, even in the geographical locations where it is most often reported (Japan, southern USA and Taiwan). There are no previous published reports of *V. vulnificus* infection from New Zealand.

Human disease is typically acquired from the gastrointestinal tract through consumption of raw seafood (particularly oysters), or at the skin, either from a wound caused by a colonised marine organism or exposure of a pre-existing wound to seawater. At either site the disease may remain localised or progress to bacteraemia and severe sepsis. Patients presenting with bacteraemia commonly have an underlying illness, and mortality (with treatment) is 50-90%. Conversely, patients presenting with wound infection typically have less comorbidity, and mortality is 7-25%.[2,3,5] Risk factors for the development of *V. vulnificus* infection include elevated serum iron levels, liver disease and an immunocompromised state. Infection is more common in males (ratio >2:1) and in older age (median age in reported series ranges from 50 to 70 years).[2,3,5,6]

Management includes aggressive resuscitation, surgical resection of necrotising cellulitis/fasciitis, and intravenous antibiotics. The antibiotic/s of choice have not been determined. Penicillins, cephalosporins, aminoglycosides, chloramphenicol, tetracyclines and quinolones have been used.[2,3,5,6]

The mean sea surface water temperatures in the Hauraki Gulf range from 13.5ºC in August to 21.1ºC in February (NIWA: Sea Surface Temperature Archives 1999). The National Institute of Water and Atmospheric Research (NIWA) predict possible temperature increases over the next 100 years of 2 - 6ºC (personal communication: NIWA). *V. vulnificus* may thus become an increasingly important pathogen in New Zealand.

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1. Levine WC, Griffin PM. Vibrio infections on the Gulf Coast: results of first year of regional surveillance. Gulf Coast Vibrio Working Group. J Infect Dis 1993; 167: 479-83.
2. Park SD, Shon JN. *Vibrio vulnificus* septicemia in Korea: clinical and epidemiological findings in seventy patients. J Am Acad Dermatol 1991; 24: 397-403.
3. Chuang YC, Liu CY, Lan CK et al. *Vibrio vulnificus* infection in Taiwan: report of 28 cases and review of clinical manifestations and treatment. Clin Infect Dis 1992;15: 271-6.
4. Kelly MT. Effect of temperature and salinity on *Vibrio (Bececkea) vulnificus* occurrence in a Gulf Coast environment. Appl Environ Microbiol 1982; 44: 985-98.
5. Klontz KC, LS, Schreiber M, et al. *Syndromes of Vibrio vulnificus* infections: Clinical and epidemiologic features in Florida cases, 1981-1987. Ann Int Med 1988; 109: 318-23.
6. Hlandy WG, Klontz KC. The epidemiology of Vibrio infections in Florida, 1981-93. J Infect Dis 1996; 173: 1176-83.

VIEWPOINTS

Socio-economic position is more than just NZDep

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The development of small area measures of socio-economic deprivation using 1991 (NZDep91) and 1996 (NZDep96) census data has been a huge advance for research and policy in New Zealand. The components of the NZDep index include the proportions at a small area-level (involving approximately 100 individuals) of census variables such as individuals with/in: no telephone access; no car access; receipt of a means-tested benefit; unemployment; low-household income; single-parent families; nil qualifications; non-tenured homes; and household crowding.[1,2] NZDep is now routinely used as a variable by which to report health status,[3,4] in population-based funding formulae, and in research.[5-11] It has several advantages. First, it can be assigned retrospectively and inexpensively to many data-sets using just an address. Second, unlike variables such as occupational class for which the relevant information is often not available in routine health records, the vast majority of routine health records have an address. Third, the variable is a robust measure of socio-economic position based on the average values of census variables. (The term 'socio-economic position' is an umbrella term for a range of socio-economic factors including income, education, material and social deprivation, absolute and relative poverty, occupational class, asset wealth, and so on).[12,13] Consequently, the ranking of individuals by the NZDep score/decile assigned to their neighbourhood is, on average, strongly and linearly related to health and other social outcomes.

As with any good tool, the widespread use of NZDep also makes it prone to widespread misuse. Our aim in this paper is to address these potential misuses and, hopefully, prevent or reduce their occurrence. Our views, although explored in more depth, are consistent with those expressed elsewhere by the originators of NZDep.[2]

Broadly speaking, there are four potential uses of NZDep:

- funding formulae and resource allocation
- community-based advocacy
- targeting of policy/interventions
- research and analysis.

Perhaps the main reason for developing NZDep was for resource allocation.[14] For example, NZDep can be used as a component in funding formulae for primary health care. As long as its use in funding is for large aggregates of people then NZDep is robust – misclassification of rich people who live in deprived areas and poor people who live in non-deprived should all balance out on average at the aggregate-level. However, NZDep is prone to misuse when used to target interventions and for research and analysis. We consider these two situations in the next two sections.

Limitations for targeting

There is no threshold above which socio-economic position causes poor health, rather poor health is distributed as a *gradient* by socio-economic position.[4,15,16] Thus, targeting policies at high(er) risk groups not only has a surprisingly small return even if the interventions are effective, but by focusing on the high risk groups misses the point that the occurrence of high risk groups is a function of how the whole population is structured. As eloquently articulated by Rose,[17,18] this paradox points to the need for population-based strategies rather than just targeting. In terms of the maldistribution of health by 'deprivation', therefore, we not only require policies targeted at deprived people/places, but also policies that prevent deprivation in the first place.

Given that some targeting is required as one component of a multi-pronged strategy, NZDep is an obvious candidate to use. However, just because a factor (such as NZDep) is the easiest to measure, does not mean that it is the most important socio-economic factor in aetiologic terms (see below) or the most appropriate basis for targeting interventions.[12,13] For example, one might argue that targeting of subsidies for GP visits should be done on the basis of income.

Not all 'deprived people' live in deprived small areas.[12,13] Shown in Table 1 are the number of 25-64 year olds in the 1991 census (n= 1.65 million) who were Maori, Pacific, unemployed, living in a household with an equivalised household income less than $20 000, nil educational qualifications, belonging to occupational class 6 (i.e. lowest class) and living in a household with no car access. In addition, Table 1 shows the percentage of these people living in each quintile of small areas by NZDep91, where quintile 1 comprises the least deprived areas and quintile 5 the most deprived areas. Of note, only 25% to 30% of the poor, those with no formal educational qualifications and belonging to occupational class 6 also live in the most deprived quintiles of small areas (as measured by NZDep). Figure 1 makes the same point, but for children by household income by NZDep91. Whilst income-based and area-based measures of socio-economic status are highly correlated, children living in low-income households also reside in non-deprived small areas and vice-versa. Salmond and Crampton have also reported only a weak correlation between the individual-level measure of deprivation and NZDep.[2] Generalising, and conversely, the (vast) majority of 'deprived' individuals according to the three classic measures of socio-economic position (income, education and occupational class) would miss out on any interventions targeted to the most deprived

quintiles of small areas by deprivation. Thus, if targeting of interventions by individual socio-economic position is needed, more than just targeting by socio-economic deprivation of the neighbourhood is required.

Table 1. Percentage distribution of 1991 census respondents age 25-64 years by demographic or socio-economic factors by NZDep91 quintiles (1 = least deprived; 5 = most deprived).

Variable	Census count	Percentage distribution by NZDep91 quintile 1	2	3	4	5
Ethnicity						
Maori	165 300	6%	10%	15%	24%	45%
Pacific	62 589	4%	7%	12%	23%	54%
Non-Maori non-Pacific	1 411 941	25%	23%	21%	18%	13%
Socio-economic factors						
Unemployed	89 823	11%	15%	18%	24%	33%
Income < $20,000	326 523	11%	16%	20%	23%	30%
Nil Education	557 028	14%	18%	20%	22%	26%
NZSEI Occupational class 6	110 643	13%	18%	20%	24%	25%
Nil car access	96 375	5%	9%	14%	24%	48%

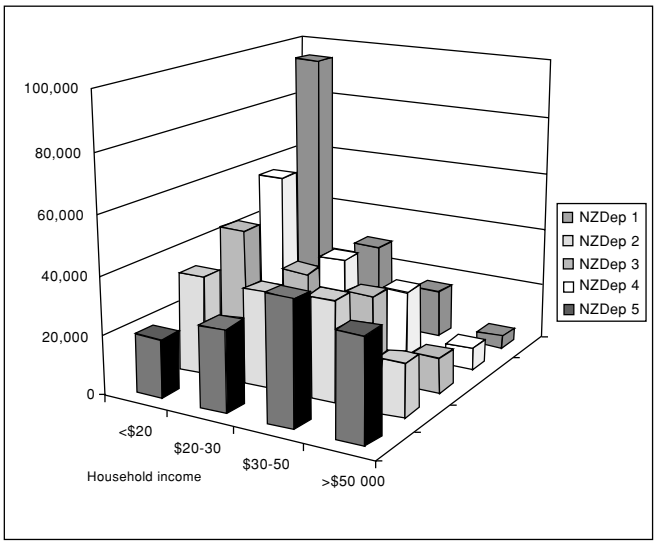

Figure 1. Number of 0-14 year old children at 1991 census by cross-classification of equivalised household income and quintile of small area socio-economic deprivation.

We are also concerned about debates among policy-makers regarding whether, having included NZDep in funding formula, it is necessary to further include ethnicity. The answer is clearly yes: any area-based interventions will only reach a minority of Maori; residual differences in health status by ethnic group remain after incorporating just one socio-economic factor[10,19] (see below); ethnicity is not included in NZDep; and addressing Maori health issues also requires consideration of cultural issues of access to health care (eg cultural safety) in addition to the more general socio-economic issues that we are considering here.

Limitations for research/analysis

NZDep is an area-based measure. Compared to other small area deprivation indices internationally it is calculated at a low-level of aggregation of approximately 100 people. Use of these particularly small areas to calculate deprivation NZDep is, in large part at least, capturing personal socio-economic position.[2,20] It also probably captures some contextual effects on health of the neighbourhood's socio-economic profile, but it is difficult to tease apart the contextual from the individual-level effects on health. For example, we have found that about half of the association of NZDep with mortality in the New Zealand Census-Mortality Study is 'explained' by controlling for personal socio-economic factors (work in progress). But the interpretation of this finding is problematic. Due to inevitable measurement error (and the failure to measure socio-economic factors across the life-course) much of this observed residual association of small area deprivation with mortality probably still reflects the effects of personal socio-economic position.

The fundamental misuse of NZDep in research and analysis is the implicit assumption that small area deprivation and socio-economic position are synonymous. For example, we have seen many analyses of 'health inequalities by deprivation' conceptualised as fully capturing 'health inequalities by socio-economic position'. This assumption is incorrect. Indeed, to assume that any *single* measure of socio-economic position is synonymous with socio-economic position is incorrect. Small area deprivation (or any other single socio-economic factors) is only one measure of the broader construct of socio-economic position. Our point may seem overly pedantic, but if not understood has serious implications when extended to analyses that attempt to answer the question "How much of the association of X with health status is 'due' to socio-economic position?" For example, consider the issue of how much of the association of ethnicity with mortality is attributable to socio-economic position.[10,18,19] Demonstrating that *only* a third of the gap between Maori and non-Maori mortality rates is due to NZDep (or any other separate measure of socio-economic position) *does not mean* that only a third of the gap is due to socio-economic position.[10] Without doubt, controlling for a range of socio-economic factors and at a number of points during the life-course would see much more of the Maori non-Maori gap 'explained'. (What it actually means to control for socio-economic position when considering ethnic differences in health is a complex issue beyond the scope of this paper).[21,22] This problem also applies to other research questions. For example, finding a statistically significant association of (lack of) fruit and vegetable consumption with ischaemic heart disease having controlled for small area deprivation (or any other separate measure of socio-economic position) at the time of diagnosis does not mean that there is still not further residual confounding by socio-economic position.[23,24]

Conclusion

NZDep is a powerful tool, and is easier to use in practice than most other measures of socio-economic position. However, there are also problems with using it alone as a measure of socio-economic position. In particular:

- the use of some threshold of NZDep (or any other measure of socio-economic position) for 'targeting' interventions at a high-risk populations may divert attention from population-based strategies; health inequalities span right across society as a gradient and are not just confined to the 'deprived'
- however, if high risk groups or individuals are to be 'targeted' then it is unlikely that an area-based strategy alone is most appropriate as:
  - not all deprived people live in deprived areas
  - area-level socio-economic effects on health are important, but probably not as important as personal socio-economic effects
- other measures of socio-economic position, and ethnicity, are required in addition to just NZDep for any targeting of interventions

It should be stressed that the issues described here are not merely academic. Taking a targeted and area-based approach to health policy and public health interventions has major implications for the future health of New Zealanders, and the future role of the public health services. If such an approach is to be followed, it should be evidence-based, and carefully justified and planned, and should only occur after considerable debate. The danger is that such an approach may occur by default, rather than by design. In particular, it would be easy for future policy in New Zealand to be targeted by NZDep alone just because it is easy to measure. If targeting of public health interventions by socio-economic position is required, a careful case-by-case consideration is required of which and how many measures of socio-economic position to use rather than just defaulting to NZDep.

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1. Salmond C, Crampton P, Sutton F. NZDep91: A New Zealand index of deprivation. Aust NZ J Public Health 1998; 22: 835-7.
2. Salmond C, Crampton P. NZDep96 - what does it measure? Soc Pol J NZ 2001; 17: 82-100.
3. Ministry of Health. Our health, our future. Wellington: Ministry of Health; 1999.
4. Howden-Chapman P, Tobias M, editors. Social inequalities in health: New Zealand 1999. Wellington: Ministry of Health; 2000.
5. Tobias M, Jackson G. Avoidable mortality in New Zealand, 1981-97. Aust NZ J Public Health 2001; 25: 15-20.
6. Jackson G, Tobias M. Potentially avoidable hospitalisations in New Zealand, 1989-98. Aust NZ J Public Health 2001; 25: 212-21.
7. Barnett J. Does place of residence matter? Contextual effects and smoking in Christchurch. NZ Med J 2000; 113: 433-5.
8. Crampton P, Salmond C, Woodward A, Reid P. Socioeconomic deprivation and ethnicity are both important for anti-tobacco health promotion. Health Educ Behav 2000; 27: 317-27.
9. Salmond C, Crampton P, Hales S et al. Asthma prevalence and deprivation: a small area analysis. J Epidemiol Community Health 1999; 53: 476-80.
10. Blakely T, Kiro C, Woodward A. Unlocking the numerator-denominator bias. II: Adjustments to mortality rates by ethnicity and deprivation during 1991-94. NZ Med J 2002; 115: 43-8.
11. Blakely T, Robson B, Atkinson J et al. Unlocking the numerator-denominator bias. I: Adjustment ratios by ethnicity for 1991-94 mortality data. NZ Med J 2002; 115: 39-43.
12. Lynch J, Kaplan G. Socioeconomic position. In: Berkman L, Kawachi I, editors. Social epidemiology. New York: Oxford University Press; 2000: p13-35.
13. Krieger N. A glossary for social epidemiology. J Epidemiol Community Health 2001; 56: 693-700.
14. Crampton P, Laugesen M. The use of indices of need in resource allocation formulae for primary health care. Wellington: Health Services Research Centre, 1995.
15. Blakely T. Socio-economic factors and mortality among 25-64 year olds: The New Zealand Census-Mortality Study. (Also at http://www.wnmeds.ac.nz/nzcms-info.htm) [doctorate]. University of Otago; 2001.
16. Howden-Chapman P, Blakely T, Blaiklock A, Kiro C. Closing the health gap. NZ Med J 2000; 113: 301-2.
17. Rose G, Day S. The population mean predicts the number of deviant individuals. BMJ 1990; 301: 1031-4.
18. Rose G. The strategy of preventive medicine. Oxford: Oxford University Press; 1992.
19. Pearce N, Pomare E, Marshall S, Borman B. Mortality and social class in Maori and non Maori New Zealand men: changes between 1975-7 and 1985-7. NZ Med J 1993; 106: 193-6.
20. Blakely T, Woodward A. Ecological effects in multi-level studies. J Epidemiol Community Health 2000; 54: 367-74.
21. Davey Smith G. Learning to live with complexity: ethnicity, socioeconomic position, and health in Britain and the United States. Am J Public Health 2000;90:1694-8.
22. Kaufman J, Cooper R, McGee D. Socioeconomic status and health in Blacks and Whites: the problem of residual confounding and the resiliency of race. Epidemiol 1997; 8: 621-8.
23. Davey Smith G, Phillips A. Confounding in epidemiological studies: why "independent" effects may not be all they seem. BMJ 1992; 305: 757-9.
24. Rothman K, Greenland S. Modern Epidemiology. 2nd ed. Philadelphia: Lippincott-Raven; 1998.

How much excercise is enough? Are we sending the right message?

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For decades, health professionals have encouraged people to become more active, thereby reducing the likelihood of developing coronary heart disease (CHD), hypertension, diabetes and other conditions linked to sedentary living. Through the 1970s and 80s, consensus recommendations stated that vigorous activity lasting 20 – 60 minutes should be performed 3 – 5 times per week[1] for health benefit. Large cohort studies have repeatedly shown that the health benefit of regular exercise is dose-dependent with regard to total caloric expenditure[2] and intensity,[3] and thus vigorous activities were the focus of recommendations. Unfortunately, such recommendations have not resulted in a greater percentage of the population becoming more active.[4]

In 1995, an expert committee on exercise and health convened by the Center for Disease Control (CDC) and the American College of Sports Medicine reviewed the available data with the goal of establishing a new consensus guideline designed to encourage the ever-growing sedentary population to begin some form of regular exercise. Close examination of data from prospective studies revealed that a caloric expenditure of approximately 1500 kcal/week was the minimum threshold of activity providing a health/survival benefit.[4,5] More importantly, it was determined that daily or near daily performance of moderate intensity activities could achieve this goal. This prompted a major change in the exercise message, which now stated "Every US adult should accumulate 30 minutes or more of moderate intensity physical activity on most, preferably all, days of the week".[4] The scientific merit of this recommendation for the general population has been debated,[6] and opponents argue that there is much stronger evidence supporting a health benefit from vigorous exercise for non-sedentary individuals.

The authors of the CDC recommendation admit that the new guideline focuses on the least fit individuals who are unlikely to become involved in more vigorous levels of activity, and they acknowledge a strong likelihood that vigorous exercise and greater weekly energy expenditures provide greater benefit.[4] From a public health perspective, this approach is justifiable, as the most sedentary have the highest mortality burden.[7] However, for a general practitioner (GP) or practice nurse assisting an individual patient, close adherence to these guidelines may inhibit capable individuals from performing higher intensity activity, which provides greater health benefit.

In New Zealand, GPs appear to be less likely to recommend more vigorous exercise (unpublished findings from the Hillary Commission). Thus, the trend for exercise recommendations appears to be that as westernised society becomes less and less fit, recommendations are simply decreased, as exemplified by the National Health Committee of New Zealand, which recently stated that even 1000 kcal/week will provide a health benefit.[8]

Is less than 1500 kcal/week beneficial?

While there is some evidence to support the National Health Committee's claims, there is very little evidence that refutes them. The 'cut-offs' used for minimum energy expenditure in previous studies have seemingly been randomly assigned and thus may not accurately determine the minimum energy expenditure required for benefit. The MRFIT trial[5] stratified

subjects into tertiles of estimated leisure-time physical activity and found that the middle tertile, who averaged 224 kcal/day (approximately 1500 kcal/week) in leisure-time physical activity, provided the maximum survival benefit (no additional benefit in the highest tertile). The 1978 assessment of the Harvard Alumni data[9] selected 2000 kcal/week as the dividing line for their comparison, and, not surprisingly, found a significantly better outcome for those above 2000 kcal/week. A more thorough assessment of the same data set in 1986 showed that mortality rates decreased steadily from 500 through 3500 kcal/week suggesting that a 'threshold' for health benefit may occur at some energy expenditure lower than 2000 or even 1500 kcal/week.[2] These findings suggest that very low levels of activity will improve the health of sedentary individuals. The fact remains however, that stronger evidence supports an increased benefit at higher caloric expenditures, which may include 'vigorous' activity.

Is vigorous exercise more beneficial?

The majority of literature suggests that the health benefits of exercise for cardiovascular disease (CVD) are dose-dependent. However, only recently has direct evidence supported the claim that vigorous activity is more beneficial than moderate or light activity. Recent reports of the Harvard Alumni Trial indicate that survival benefit increases as self-reported involvement in 'vigorous sport activity' increases. In one report which separated the individual effects of self-reported blocks walked, stairs climbed, sports or recreational activities (moderate and vigorous), only total sports and recreational activity ($p = 0.042$) and vigorous activities ($p = 0.02$) were inversely associated with CVD.[10] Another report of the Harvard data[11] shows that light activity (< 4 metabolic equivalents or METs) provided no benefit ($p = 0.72$), moderate activity (4-6 METs) provided some benefit ($p = 0.07$) and vigorous activity (> 6 METs) predicted clear mortality benefit ($p < 0.001$).

Earlier studies estimated weekly caloric expenditure from leisure time activity,[2,5,9] without carefully accounting for the intensity of activities performed. From these studies it could only be assumed that higher self-reported weekly caloric expenditures are more likely to be achieved with vigorous activity. For example, using CDC calculations, 3000-3500kcal/week, which has been reported as the level of activity providing the greatest benefit[2] would require a 75 kg individual to walk at 6.4 km/hr for 68-80 minutes every day of the week (8.0 - 9.3 hours/week). Alternatively, the same individual could achieve this expenditure by jogging 27-32 minutes per day at 12 km/hr.[12] Indirectly, this suggests that the benefit of 30 minutes of physical activity per day increases with increasing intensity levels.

Exercise prescription in New Zealand

Physician-advised exercise prescription has been shown to involve greater numbers of patients in regular exercise.[13] In New Zealand, the Green Prescription initiative was started by the Hillary Commission in 1997 in an attempt to involve GPs in getting more New Zealanders active. The prescription, which is written by physicians and practice nurses, adheres closely to the CDC recommendations, outlining the frequency and duration of walking or 'moderate activity' the patient should perform. The Hillary Commission has also produced physical activity guidelines for health professionals stating that vigorous exercise adds additional benefit and should be conducted three days a week for 20 minutes or more for extra health and fitness.

Statistics gathered in a randomised clinical trial showed that NZ GPs successfully increased participants' recreational physical activity and felt that written prescriptions helped them formalise exercise goals for participants. They also felt that written prescriptions were more effective than verbal advice alone.[14] In the most recent survey, responding GPs (50%) wrote an average of 3.7 scripts/month, or 57 720 prescriptions annually (unpublished data from the Hillary Commission). These data clearly identify the potential for GPs to influence the behavioural patterns of New Zealanders. However, the answers to two new questions in the survey may demonstrate a misinterpretation of the CDC recommendations. The first asked if the GPs believed that 30 minutes of moderate physical activity most days improves health and the second asked if vigorous exercise for 30 minutes three days of the week helped. Despite evidence that the health benefit of exercise is dose-dependent, 96% of GPs agreed with the moderate exercise message, but only 47% agreed with the benefits of vigorous exercise (unpublished results). Furthermore, misinterpretation of 'moderate' activity by physicians and patients may result in well-intending individuals undertaking an exercise regime which fails to meet the requirements for a health benefit, or certainly fails to maximise any benefit.

What is moderate activity?

The intensity of any activity is relative to the individual. The commonly accepted definition of moderate intensity exercise is 45-59% of an individual's maximum aerobic capacity.[15] In healthy, normally active individuals, age and gender are the major determinants of maximal aerobic capacity (MAC) and the following two regression equations can be used to estimate an individual's MAC in metabolic equivalents or METs:[16]

Males MAC = [60 - (0.55 x age)]/3.5
Females MAC = [48 – (0.37 x age)]/3.5

Using 45-59% of the MAC so determined can be prescribed as a starting 'intensity' of exercise. The weekly 'dose' of exercise is then determined by the duration and frequency of each bout of this intensity ('dose' = intensity x duration x frequency). To calculate this weekly 'dose' of energy expenditure the intensity in METs (Table 1) is converted to the caloric expenditure for the particular individual by multiplying METs x 1 kcal/kg/hour. Thus, the energy expenditure of an activity of 5 METs intensity, (very brisk walking) undertaken by a 75 kg person is:

5 x 1 kcal/kg/hour (1 MET) x 75 kg = 375 kcal/hour.

Table 1. Examples of energy expenditure from leisure activities of intensity between 3-6 METs.

Specific leisure activity	MET value	Energy expenditure (kcal/kg/hour)
Bicycling, < 16 kph	4.0	4.0
Bicycling, 16 – 19 kph	6.0	6.0
Swimming, leisurely, not lap swimming	6.0	6.0
Walking, 4.8 kph (3 mph, moderate pace)	3.3	3.3
Walking, 6.4 kph (4 mph, very brisk pace)	5.0	5.0
Walking, 5.6 kph uphill (3.5 mph)	6.0	6.0

MET data from reference [12].

If this activity has a duration of 30 min (0.5 hour) and is repeated every day of the week the 'dose' of exercise is:

375 kcal/hr x 0.5 hr/day x 7 days = 1312.5 kcal/week

Calculating the absolute amount of energy expenditure (dose) on a daily or weekly basis is useful because health benefits have usually been expressed in relation to absolute energy expenditure. Note that the weekly expenditure calculated in the example above is less than the CDC

minimum threshold of 1500 kcal/ week despite meeting the recommendation of moderate activity for 30 min of most days of the week. Even if a minimum energy expenditure threshold is conclusively established, it should not obscure the fact that increasing expenditure above this level will further increase health benefits.

In an attempt to apply a relative measure to a large population, the CDC recommendation defines moderate activity as 3-6 METs for all individuals regardless of age, gender or fitness level. Figure 1 shows that some part of this range falls within 45 – 59% of predicted MAC for any age/gender stratification. Nonetheless, most of this range would be classified as light activity for males up to age 40 or vigorous activity for people 60 or older. For these reasons, and individual differences in fitness and exercise risk, exercise prescription should be individualised when possible.

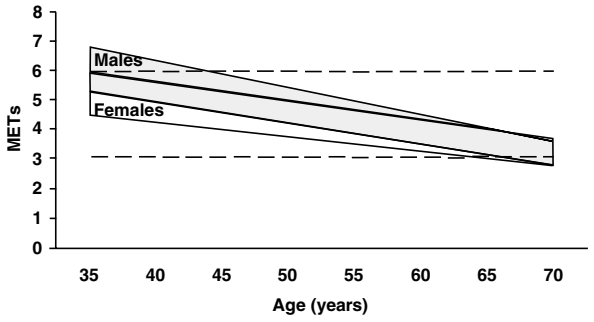

Figure 1. Population versus individual estimates of moderate physical activity. Age and gender-based predictions of moderate activity level (shaded bars) as compared to the CDC recommendatoins of 3 – 6 METS.[4,16]

Is there an acute risk associated with vigorous exercise?

The risk of cardiovascular complication is transiently higher during or immediately after vigorous exercise, particularly in those who do not perform vigorous activity regularly. The relative risk of acute myocardial infarction increases by up to six-fold during vigorous physical activity.[17] The risk of cardiac arrest during exercise has been estimated to be 56 times greater in sedentary men, but only 5 times greater in those with high levels of habitual physical activity.[18] Not surprisingly, habitual vigorous physical activity reduces the rate of sudden death[18] particularly during vigorous activity.[19] However, the rate of cardiac arrest during vigorous physical activity may be up to 100-fold higher in those with coronary artery disease.[20] For these individuals, or those with 'high risk' for coronary artery disease, moderate intensity physical activity may be more appropriate.

A revised message

It is clear that formalised exercise prescription by GPs and practice nurses has the potential to alter the lifestyle, and thus improve the health of New Zealanders. Furthermore, this method of intervention (Green Prescription) seems to effectively involve more individuals in physical activity than other, non-medically prescribed methods. Based on a paucity of well-quantified exercise data, low levels of physical activity (< 1000 kcal/week) may provide some health benefit to those who are older or currently sedentary. However, there is stronger evidence to suggest that greater doses of physical activity, which may include vigorous activity, provide additional benefit for non-sedentary, healthy individuals. For these reasons, GPs and practice nurses should individualise their exercise prescription based on age, gender and fitness level, and encourage vigorous (> 6 METs) activity in those who are younger, healthy and capable of performing it.

A GP's exercise prescription 'ready reckoner', which provides tables and instructions for appropriate exercise prescription, is available from:

Sport and Recreation NZ (formerly the Hillary Commission)
PO Box 2251,
Wellington.

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1. American College of Sports Medicine. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. Med Sci Sport Exec 1990; 22: 265-74.
2. Paffenbarger RS, Hyde RT, Wing AL et al. Physical activity, all-cause mortality, and longevity of college alumni. N Engl J Med 1986; 314: 605-13.
3. Morris JN, Clayton DG, Everitt MG et al. Exercise in leisure time: coronary attack and death rates. Br Heart J 1990; 63: 325-34.
4. Pate RR, Pratt M, Blair SN et al. Physical activity and public health: A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. JAMA 1995; 273: 402-7.
5. Leon AS, Connett J, Jacobs DR et al. Leisure-time physical activity levels and risk of coronary heart disease and death. JAMA 1987; 258: 2388-95.
6. Williams PT. Physical activity and public health. JAMA 1995; 274: 534.
7. Powell KE, Blair SN. The public health burdens of sedentary living habits: theoretical but realistic estimates. Med Sci Sport Exerc 1994; 26: 851-6.
8. National Health Committee. Active for life: A call for action. Wellington: Ministry of Health; 1998.
9. Paffenbarger RS, Wing AL, Hyde RT. Physical activity as an index of heart attack risk in college alumni. Am J Epidemiol 1978; 108: 161-75.
10. Sesso HD, Paffenbarger RS, Lee IM. Physical activity and coronary heart disease in men: the Harvard Alumni Health Study. Circulation 2000; 102: 975-80.
11. Lee IM, Paffenbarger RS. Associations of light, moderate and vigorous intensity physical activity with longevity. Am J Epidemiol 2000; 151: 293-9.
12. Ainsworth BE, Haskell WL, Whitt MC et al. Compendium of physical activities: an update of activity codes and MET intensities. Med Science Sport Exerc 2000; 32 (Suppl 9): S498-S516.
13. Lewis BS, Lynch WD. The effects of physician advice on exercise behaviour. Prev Med 1993; 22: 110-21.
14. Swinburn BA, Walter LG, Arroll B et al. The Green Prescription Study: a randomised controlled trial of written exercise advice provided by general practitioners. Am J Public Health 1998; 88: 288-91.
15. United States Department of Health and Human services. Physical activity and health: a report of the Surgeon General. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 1996.
16. Center for Disease Control and Prevention. Physical activity trends - United States 1990-1998. MMWR Morb Mortal Wkly Rep 2000; 50: 166-9.
17. Mittleman MA, Maclure M, Tofler GH et al. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion. N Engl J Med 1993; 329: 1677-83.
18. Siscovick DS, Weiss NS, Fletcher RH et al. The incidence of primary cardiac arrest during vigorous exercise. N Engl J Med 1984; 311: 874-7.
19. Albert CM, Mittleman MA, Chae CU et al. Triggering of sudden death from cardiac causes by vigorous exertion. N Engl J Med 2000; 343: 1355-61.
20. Cobb LA, Weaver WD. Exercise: a risk for sudden death in patients with coronary heart disease. J Am Coll Cardiol 1986; 7: 215-9.

PROCEEDINGS

159th Scientific Meeting of the Otago Medical School Research Society, October 18th 2001.

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Opioid receptor regulation of prolactin secretion at the end of pregnancy. Zane B Andrews, David R Grattan. Department of Anatomy and Structural Biology, School of Medical Sciences, University of Otago.

Prolactin secretion is tonically inhibited by tuberoinfundibular dopamine (TIDA) neurons located in the hypothalamus. We have shown that naloxone, a broad-spectrum opioid receptor antagonist, suppresses prolactin secretion during late pregnancy by activating TIDA neurons. To investigate the specific opioid receptor subtypes involved in facilitating prolactin secretion at this time, we measured opioid receptor gene expression throughout pregnancy and examined the effect of specific opioid receptor antagonists on prolactin secretion and TIDA neuronal activity.

Relative kappa, mu and delta opioid receptor mRNA expression was measured in the microdissected arcuate nucleus (location of TIDA neuronal cell bodies) on day 7, 14, or 21 of pregnancy and day 7 of lactation using sequence specific primers and fluorogenic probes complementary to the cDNA of each receptor. cDNA was amplified by quantitative PCR using the ABI 7700 Taqman system. Selective opioid receptor antagonists nor-binaltrophimine (kappa), ß funaltrexamine (mu) and naltrindole (delta) or saline were administered intracerebroventricularly. Two hours after antagonist administration brains were collected and processed to measure TIDA neuronal activity. Trunk blood was collected and serum prolactin measured by radioimmunoassay.

Kappa opioid receptor mRNA expression in the arcuate nucleus was significantly increased on day 21 of pregnancy when compared to other time points. Expression Mu opioid receptor mRNA did not change during pregnancy and no delta receptor mRNA was detected. Nor-binaltrophimine and ß funaltrexamine significantly increased TIDA neuronal activity compared to saline treated controls. Increases in TIDA activity were associated with significant reductions in serum prolactin levels. Naltrindole had no effect on TIDA neuronal activity or serum prolactin.

These data indicate that kappa and mu opioid receptors are important in the regulation of prolactin secretion at the end of pregnancy. The increase in kappa receptor mRNA on day 21 of pregnancy, however, suggests that endogenous opioid peptides may preferentially act at kappa receptors during late pregnancy.

Sites of interaction of the bacterial and mitochondrial protein synthesis release factors with the RNA of the ribosome. Marjan Askarian-Amiri, Warren P Tate. Department of Biochemistry and Centre for Gene Research, Otago School of Medical Sciences, University of Otago.

Termination of protein synthesis is a multi-step process involving a highly specific interaction between ribosomes and presumably rRNA, mRNA and protein decoding factors. A protein release factor (RF) binds to the ribosomes and decodes the stop signal. It has been shown that RFs bind to ribosomes at the active centre during this decoding event.

An approach to detect the putative regions of the ribosome in contact with bacterial RFs, has been carried out following in principle the SERF (SElection of Random RNA Fragment) technology. Some conserved regions of the *E. coli* rRNA in contact with RFs have been identified. In the small subunit of ribosome (16S rRNA) the identified elements were part of helix 21 (H21), H24 (790 loop) and H26. In the large subunit (23S rRNA), H78 and H80 in domain V, and H42-44 in domain II have been identified to interact with RFs. Loop D of 5S rRNA was the other region of the 50S small subunit that interacts with both RFs. This loop is structurally important and acts as a bridge between domain II and V of 23S rRNA.

The regions of the 50S small subunit of ribosome identified here to be in contact with RFs are located in the platform of the central region of the 16S rRNA. The front and back of the platform may exhibit some flexibility and make this region optimal for factor binding sites. The selected regions of 23S rRNA were H42-44, H78 and H80. Part of H42-44 is the L11 binding site and it's interaction with bacterial RF1 has previously been shown by hydroxyl radical probing, and H42-44 also forms part of the ribosomal GTPase centre. H78 and 80 of 23S rRNA are located close to the exit tunnel of the ribosome that with L1 make part of the tunnel.

The effect of St John's wort on hepatic drug metabolising enzymes. Belinda Bray,[1] NB Perry,[2] DB Menkes,[3] RJ Rosengren.[1] Department of Pharmacology and Toxicology,[1] Otago School of Medical Sciences, Department of Chemistry,[2] Department of Psychological Medicine,[3] Dunedin School of Medicine, University of Otago.

This investigation was designed to determine the ability of St. John's wort (SJW), a readily available antidepressant, to induce various hepatic cytochrome P450 (CYP450) or conjugation enzymes. SJW (140 or 280 mg/kg/day) was administered to male Swiss Webster mice (25g) for 1, 2 or 3 weeks. Enzymatic activity was analysed in hepatic microsomes for all of the following drug metabolising enzymes: CYP3A, CYP1A, CYP2E1, UDP-glucuronosyltransferase (UDPGT) and glutathione S-transferase (GST). The catalytic activity of CYP1A, as determined by ethoxyresorufin O-deethylation, was unchanged from control following any dose or duration of SJW. In contrast the catalytic activity of CYP3A (determined by erythromycin N-demethylation) and CYP2E1 (determined by p-nitrophenol hydroxylation) were induced by SJW at both doses. For each of these isoforms, three weeks of SJW administration resulted in a two-fold increase in catalytic activity, no induction was evident prior to three weeks. Results from western immunoblotting studies supported the changes in catalytic activity, as CYP2E1 and CYP3A polypeptide levels were increased at the three week time point. Studies investigating changes in conjugation enzymes demonstrated that GST activity, as determined by Chloro dinitrobenzene conjugation, and UDPGT activity, as determined by p-nitrophenol conjugation, were unchanged from control following all SJW treatments. These results indicate that drug interactions with SJW are not likely to occur through an increase in the activities of CYP1A, GST or UDPGT. However, long term SJW usage may have clinically relevant effects due to the induction of CYP2E1 and CYP3A. Specifically, patients taking medications that either have a narrow therapeutic index or are bioactivated by these enzymes may be at risk from SJW-related complications. Additionally, it appears that increased metabolism of drugs and chemicals is unlikely to occur following acute SJW but instead is linked to prolonged daily ingestion.

Androgen and progesterone receptor expression in ovarian surface epithelium of aged mice. Olivia L Clow, Peter R Hurst, Jean S Fleming. Department of Anatomy & Structural Biology, Otago School of Medical Sciences, University of Otago.

One hypothesis of ovarian surface epithelial (OSE) carcinogenesis is that high levels of progesterone are protective, whereas high levels of androgen increase the risk of ovarian cancer. Epithelial lined inclusion cysts are common in all women and are thought to be precursors of ovarian cancer. This study aimed to investigate spatial and temporal differences in expression of androgen receptor (AR) and progesterone receptor (PR) in the OSE and inclusion cysts of ovulating, aged mice.

Ovaries were collected from 17, 12-month Swiss Webster mice at 5h intervals post ovulation and two anoestrous animals. AR and PR immunohistochemistry was performed on 4 µm sections, cut from paraffin-embedded tissue. OSE and inclusion cysts were scored for nuclear staining density and cellular morphology.

PR staining density was low in OSE from anoestrous ovaries compared with ovulating ovaries, with no differences between surface cuboidal or squamous cells. AR expression however, was low in OSE from all animals, with very few squamous cells staining. The majority of cells in inclusion cysts from all ovaries were stained for AR and PR, except for squamous cells which showed little AR expression. In contrast invaginated OSE showed no staining for either receptor. No variation in AR or PR expression was observed with time after ovulation in OSE or inclusion cysts. The strong upregulation of AR expression in cyst epithelial cells suggests that androgen responsiveness may play a role in the aetiology of ovarian cancer.

Development of Iscoms and other colloidal particles as vaccine delivery systems. Patrick H Demana, Nigel M Davies, Thomas Rades. School of Pharmacy, University of Otago.

Vaccination is one of the major achievements of modern medicine. Diseases such as smallpox have been eradicated while polio and measles are now controlled. Despite these successes, there are many microbial diseases which to date cannot be treated or prevented through the use of vaccines. One of the major drawbacks on the efficacy and the use of

vaccines has been the lack of effective delivery systems for vaccine antigens. Particulate vaccine delivery systems have been shown to be more immunogenic than solutions of subunit vaccines. Parental administration of vaccines through the use of injections is not ideal in most vaccination programmes. Based on these two major problems, the purpose of this study was to investigate a colloidal delivery system for delivery of vaccine antigens across the skin.

We have prepared Immunostimulating complexes (Iscoms) as a particulate colloidal vaccine delivery system using a novel method recently developed in our laboratory (hydration technique). Using this technique, we observed various other colloidal structures such as Iscoms-like structures, liposomes, liposomes containing micelles and/or Iscoms as well as lamellae, helical ring-like and worm-like micellar structures in addition to Iscoms depending on the ratio of the components used to form Iscoms (saponin Quil A, cholesterol and phospholipid). The resulting colloidal structures were characterized by transmission electron microscopy, photon correlation spectroscopy, fluorescence measurements and analytical sucrose gradient ultracentrifugation. Depending on the ratio of the components, the predominance of some structures over others could be established in different regions of a pseudo-ternary phase diagram. However, most mixtures contained a variety of structures, some which may be important as colloidal vaccine delivery systems.

Evaluating the effectiveness of 'Cue Exposure Therapy' by measuring its ability to diminish cue strength of personal smoking triggers. Stuart G Ferguson, Louis S Leland, Jr. Department of Psychology, University of Otago.

This study was designed to test a method of evaluating behavioural smoking cessation programs that was free of confounds associated with current methods. We used this evaluation to assess a popular behavioural smoking cessation technique - Cue Exposure Therapy (CET).

The evaluation method developed in this study measures how effective a behavioural technique is at breaking the association between smoking triggers and craving. This evaluation allows researchers to isolate the behavioural component of the addiction.

Measures of cue strength (the level of craving stimulated by exposure to a smoking trigger) were obtained for four personal smoking triggers for each of ten participants. To do this, participants used guided imagery to 'experience' their personal triggers and then rated the level of craving generated by each trigger. Two of the triggers (randomly chosen) were then treated using a four-week course of CET while the remaining two triggers remained untreated (as a control). At the end of the study, a second measure of cue strength was obtained for all four triggers. T-tests were used to compare the pre- and post-cue strength scores for both the treatment and control triggers. Triggers treated with CET showed a significant decrease in cue strength (11.2 to 6.6, $t(9) = 2.74$, $p = 0.011$, one-tailed) over the course of the study while the untreated triggers did not (10.1 to 9.4, $t(9) = 0.57$, $p = 0.292$, one-tailed).

It was concluded that CET is an effective treatment for the behavioural component of the smoking addiction so long as the principal personal triggers are treated. The evaluation technique successfully isolated the behavioural component of smoking addiction and in doing so removed confounds associated with traditional assessment methods.

Calcium and brain proteomics: a glimpse into the enigmatic role of calbindin$_{28kDa}$. JC Kon, MJ Hubbard. Department of Biochemistry, Otago School of Medical Sciences, University of Otago.

The physiological role of calbindin$_{28kDa}$ (CB28) remains unclear despite concerted investigations spanning three decades. Biomedical interest emanates from associations of this calmodulin-like calcium-binding protein with neurodegenerative disorders (e.g. Alzheimer's), apoptosis and epithelial calcium transport. Its wide expression and high evolutionary conservation suggest that CB28 has an important intracellular role. A role in calcium regulation seems likely since altered CB28 expression has been associated with calcium cytotoxicity. CB28 is generally regarded as a mobile calcium buffer in tissues where it is most abundant (>1% of soluble protein in brain, dental enamel cells, kidney). However, the calcium buffer proposal was seriously challenged by revelations that calcium regulation appears largely normal in CB28-nullmutant mice.

Our previous investigations pointed to an alternative role for CB28 that involves interactions with target proteins in the particulate fraction (cytoskeleton, endoplasmic reticulum). Here we addressed an ensuing hypothesis that CB28, like calmodulin, has a calcium-signalling role through modulation of protein phosphorylation.

A 2-dimensional gel approach was used to assay protein phosphorylation in soluble and particulate fractions of murine brain. In extracts physically depleted of calmodulin, supplementation with exogenous CB28 failed to restore calmodulin-dependent phosphorylation. In extracts lacking CB28 (from nullmutant mice), addition of CB28 also failed to invoke phosphorylation changes. Surprisingly however, we found that CB28 was itself phosphorylated when added to particulate fractions.

These results give new insights to CB28 function by providing the first strong evidence that, (a) CB28 is a phosphorylation target, and (b) CB28 does not have a broad phosphoregulatory role like calmodulin. The particulate association of phospho-CB28 supports our postulated role for CB28-target protein interactions. The proteomic approach was beneficial since it revealed a novel post-translational modification of CB28 and enabled numerous phosphorylation events to be monitored simultaneously.

The role of L-arginine metabolism in hypoxia-ischaemic-induced brain injury. Hanzhong Liu, David Jackson, Ian Appleton. Department of Pharmacology and Toxicology, Otago School of Medical Sciences, University of Otago.

L-arginine can be metabolized by either nitric oxide synthase (NOS) with the formation of L-citrulline and nitric oxide (NO), or arginase with the production of L-ornithine and urea. L-ornithine can subsequently be converted to proline (the building block for collagen synthesis) and the polyamines, putrescine, spermidine and spermine, which are essential for cell growth and differentiation. It has previously been demonstrated that NO has a key role in the pathological response of the brain to hypoxia-ischaemia (HI). However, the role of arginase and the interaction of arginase with NOS has not been determined. Therefore, in this study we investigated the induction of NOS and arginase during HI-induced brain damage. HI-induced brain damage was produced in 26-day old rats and the brains were removed after three days. The activity and protein levels of arginase and the three isoforms of NOS were then determined in the left and right hemisphere and the cerebellum. HI caused a significant increase in total NOS (control, 1337.6 ± 59.6 pmol/mg protein vs HI, 2130.3 ± 286.0; $p<0.05$) and iNOS (control, 46.3 ± 4.6 pmol/mg protein vs HI, 324.0 ± 91.8; $p<0.01$) activity in the ischaemic hemisphere. This effect was mirrored by a significant induction of nNOS ($p<0.01$) and iNOS ($p<0.001$) protein levels. In addition there was a significant increase in arginase activity (control, 27.2 ± 0.8 µg urea/mg protein vs HI, 34.8 ± 1.4; $p<0.001$) and protein levels ($p<0.01$) in the ischaemic hemisphere. The increased NOS activity observed in the ischaemic hemisphere is consistent with a role for NO in the pathology of HI. However, we have demonstrated for the first time that there is also a significant upregulation of the enzyme arginase during HI. We speculate that this increase in arginase activity may be an attempt at repair following HI-induced brain damage.

ERp29: Investigating the distribution and function of a recently-discovered protein in the rat brain. Jennifer Macleod,[1] Mike Hubbard,[2] Rod Sayer.[1] Departments of Physiology[1] and Biochemistry,[2] Otago School of Medical Sciences, University of Otago.

ERp29 is a recently-discovered protein that has been localised to the endoplasmic reticulum (ER). It has been dissociated from a direct role in calcium handling, but implicated in the processing of secretory proteins. Growing evidence for the involvement of the ER in human protein-misfolding diseases highlights the need to understand the function of this and other reticuloplasmins.

To gain insights into the presently unknown function of ERp29, we determined its distribution in the rat brain, which expresses ERp29 highly relative to other reticuloplasmins. Tissue was fixed in 4% paraformaldehyde and wax-embedded. Sections were incubated in rabbit polyclonal anti-ERp29 and processed using the avidin-biotin complex (ABC) method to give a diaminobenzidine (DAB) colour product. Some sections were fluorescently double-labelled for the detection of nuclei or oligodendrocytes.

ERp29 was detected throughout the rat brain and was found in both neuronal and non-neuronal cells. Only a two-fold range was observed between the most abundant (cerebellum) and least abundant (cerebral cortex) regions. At the cellular level, considerable variation in ERp29 expression was observed. Some cells, such as cerebellar Purkinje neurons and cortical pyramidal neurons, had strong ERp29 labelling, whereas others, such as thalamic neurons, did not express ERp29 highly. We conclude that ERp29 is a reticuloplasmin of major importance throughout the brain, but that some cells have a greater functional requirement for it.

Reduced motor cortical activation in a rat model of Parkinsonian bradykinesia. LC Parr-Brownlie, BI Hyland. Department of Physiology and Neuroscience Research Centre, Otago School of Medical Sciences, University of Otago.

Bradykinesia is a prominent symptom of Parkinson's disease but little is known of its physiological basis. We investigated changes in motor cortex activity and movement speed, secondary to loss of dopaminergic function, following dopamine receptor blockade (haloperidol 0.12 mg/kg i.p.). Extracellular recordings were made from 497 motor cortex cells in 4 rats during control and haloperidol experiments (n=110 and 387, respectively). To ensure successful drug delivery, cell recordings (n=90) were selected from experiments in which haloperidol increased the duration of the terminal phase of forelimb reaching movements (movement time, MT) by more than 50%. In these recordings, haloperidol significantly increased MT by 206 ± 172% (mean ± SD, $p=0.001$, one factor ANOVA) and

significantly decreased mean resting firing rate by 35 ± 56% (p=0.005). There was a trend for movement-related excitations to decrease by 40 ± 37% when haloperidol was administered, which did not reach significance with the numbers available (n=14).

These results indicate that acute reduction of dopaminergic activity sufficient to induce bradykinesia decreases both resting and movement-related firing of most neurons in the motor cortex of the rat. This confirms one previous report in monkeys chronically lesioned by the neurotoxin, 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), and extends it by showing that the acute and chronic cortical effects of reduced dopamine activity may be similar. It is proposed that the reduction in resting and movement-related firing rates account for changes in cerebral blood flow and the bradykinesia of Parkinsonism.

Early microvascular events in acute rejection of liver allografts in the rat. Michael N Tawadrous, Ayako Mabuchi, Xingyi Zhang, Antony M Wheatley. Department of Physiology, Otago School of Medical Sciences, University of Otago.

Little is known about the *in vivo* microcirculatory alterations in acute rejection of liver allografts, with only one study performed using a non-arterialised transplant model. We investigated the hepatic microcirculation during allograft rejection in the untreated Dark Agouti (DA) to Lewis rat combination using intravital fluorescence microscopy (IVFM) and laser Doppler flowmetry. Findings were compared with Lewis isografts and sham-operated controls. We also performed non-arterialised syngeneic transplants to investigate the impact of intact arterial blood supply on the hepatic microcirculation in the first week post-transplantation.

Rat orthotopic liver transplantation (OLT) with or without arterialisation was performed under ether anaesthesia with total graft ischaemia duration of <2h. IVFM liver examination was performed on days 2 (n=7), 4 (n=5) and 7 (n=7) post-transplantation in the allograft groups and on day 7 in the syngeneic groups. In the first set of experiments, 3 groups of animals were investigated: Control (n=8), AOLT (arterialised OLT; n=8) and NOLT (non-arterialised OLT; n=7).

Microvascular perfusion was significantly higher in AOLT compared with NOLT animals at 7 days post-operatively [Lobular Perfusion Index: 0.91 vs 0.63 respectively, p<0.001; density of perfused sinusoids (n/400,000μm^2): 8.1 vs 5.8 respectively, p<0.05]. In addition, large diameter-slow velocity vessels surrounded by a ring of intense vitamin A autofluorescence (proliferated hepatic stellate cells) were occasionally encountered in NOLT livers. Serum levels of conjugated bilirubin and markers of hepatocyte injury were elevated in the NOLT but not the AOLT group, thus indicating the value of arterialisation in rat OLT. In the arterialised DA to Lewis combination, lobular perfusion and laser Doppler signal dropped progressively from day 4. Pronounced peri-portal Rhodamine 6G fluorescence was found in the 4- and 7-day groups, which correlated with hyper-fluorescence of Hoescht H33342-stained hepatocyte nuclei (presumed to be apoptotic). Hepatocyte hyper-fluorescence was observed as early as two days after allogeneic transplantation despite the intact microvascular perfusion. Our data indicate the significance of early microvascular events in the progression of a rejection episode and provide a platform for further studies on microcirculatory alterations in acute rejection.

Evidence supporting a role for GABA in mediating the effects of leptin on the reproductive axis. Brigitte J Todd, David R Grattan. Department of Anatomy and Structural Biology and Neuroscience Research Centre, Otago School of Medical Sciences, University of Otago.

A significant increase in metabolic demand or reduction in supply of nutrients is associated with suppressed fertility. The adipose-derived hormone leptin may mediate this link. The obese Zucker rat has a non-functional leptin receptor. We have shown that a leptin signal is required for generation of pulsatile luteinising hormone (LH) secretion, integral to an animal's reproductive capacity. GABAergic neurons in the rostral hypothalamus, an area of major inhibitory regulation of LH secretion, express leptin receptors. We examined γ-aminobutyric acid (GABA) activity in specific brain regions of Zucker rats to investigate the hypothesis that leptin stimulates the reproductive axis by inhibiting a GABAergic neuronal pathway.

Obese (fa/fa) and lean (Fa/?) female Zucker rats were ovariectomised. One week later, half the animals in each group were injected with aminooxyacetic acid (AOAA), a drug which prevents GABA degradation. One hour later, rats were decapitated and their brains rapidly removed and frozen. GABA concentrations in a variety of micro-dissected brain areas were measured by HPLC. The rate of accumulation of GABA in the tissue following AOAA was taken as an estimate of GABA turnover. Differences in mean GABA accumulation following AOAA were measured by group factorial analysis of variance. Obese rats showed significantly higher GABA activity than lean rats (p<0.05) in the diagonal band of Broca (39.98 ± 11.29 ng GABA/μg protein *cf* 11.69 ± 1.44), ventrolateral preoptic area (11.40 ± 0.43 *cf* 4.78 ± 0.59), and paraventricular nucleus (15.05 ± 1.63 *cf* 4.84 ± 0.89), while there were no differences between phenotypes in the medial preoptic area, median eminence, or lateral hypothalamus. Lean rats showed significantly higher GABA activity in the cingulate cortex (9.14 ng ± 1.63 ng GABA/μg protein) than obese rats (4.96 ± 0.61 ng GABA/μg protein, p<0.05).

This study showed that obese Zucker rats have increased GABA activity in some hypothalamic regions involved in the control of LH secretion suggesting that the lack of a leptin signal leads to activation of GABA neurons, thus contributing to LH suppression. Inhibition of the GABAergic pathway may be one mechanism by which leptin acts to facilitate LH secretion.