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Risk factors for entry into residential care after a support-needs assessment
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Risk factors for residential care after a support needs assessment were identified from an administrative database in Wellington, New Zealand. The very elderly (ie, persons aged over 80) and those with mobility, continence, and cognitive problems were most at risk. These factors may represent targets for intervention to keep older adults in their own homes.

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F Matthews

An enduring power of attorney can be donated by a competent adult, nominating another person to make welfare and/or financial decisions on his/her behalf in the event of mental incapacity. Attorneys have wide powers, which may be misused. Doctors may be aware that this is happening and may need to turn to the Family Court in order to protect incapacitated patients.

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Assessment prior to institutional care: time to move past the Support Needs Assessment Form (SNAF)

John Campbell

> 'When I use a word' Humpty Dumpty said in rather a scornful tone, 'it means just what I choose it to mean—neither more nor less'
> (*Lewis Carroll, Through the Looking Glass).*

Assessment is certainly a word that means different things to different people. In the assessment of elderly people prior to institutional care there would be advantage to patients and the health system if all the purposes of assessment could be met in a single process and assessment came to mean more, not less.

To clinical staff, medical, nursing, social, and rehabilitation, assessment means the full evaluation of an elderly person with disability to determine what is causing the problems and what can be done to improve the situation.

There are good reasons for doing this at the time the person considers moving to institutional care. The person obviously has problems of sufficient severity that she is proposing giving up her independent lifestyle. Indeed, it is a major social change, and professional advice at this time is a service that a health system should provide.

Although much of the professional advice will come from the person's general practitioner, many of the conditions which affect elderly people are chronic and slowly progressive. They creep up on both patient and practitioner alike. A fresh look might well identify alternative approaches to long-standing difficulties. And there is good evidence that assessment prior to a proposed rest home admission affects the decision. Of 158 elderly people living at home who were assessed in Christchurch prior to rest home or hospital admission, 76 were able to continue in their own homes and 67 were still there 6 months later.[1] This is consistent with overseas findings on the value of clinical assessment prior to moving from home .

Assessment includes more than the evaluation of the elderly person alone. Family carers will often have provided support and their views and wellbeing need careful consideration. Final advice to the patient and family commonly requires a careful evaluation of conflicting desires and open and frank discussion of the options. The health professional has a responsibility to ensure all parties understand the compromises that will have to be made in coming to a decision.

To those responsible for funding institutional care, assessment means ensuring that the people in most need receive the financial support available. Institutional care is expensive, and all developed countries are concerned about the rising costs of providing continuing care for an ageing population. Assessment to ensure that the public expenditure is warranted is an entirely justifiable activity in a health service which must balance competing demands. The Support Needs Assessment Form (SNAF) was an instrument designed 10 or more years ago primarily to meet this assessment objective.

One of the long-standing problems with developments in the New Zealand health system is that we fail to budget for evaluation at the time of planning and introduction. This has been so for SNAF. Despite the many hours that have gone into completing innumerable SNAFs, we do not know whether the process is meeting clearly stated objectives. As Weatherall, Slow, and Wiltshire point out very clearly in the introduction to their paper *Risk factors for entry into residential care after a support needs assessment* (N Z Med J. 2004;117(1202). URL: http://www.nzma.org.nz/journal/117-1202/1075) in this issue of the *Journal,* theirs is the first published review of the effectiveness of the SNAF since its introduction.

The study by Weatherall, Slow, and Wiltshire reports on 2060 SNAF assessments. One-third of people assessed required residential care and the predictors of this need were increasing age, incontinence, mobility problems, and dementia. Unfortunately, the SNAF is such a limited instrument that other possible predictors of residential care admission are not measured. The importance of the predictors that were identified is that their impact can be modified. This is also the situation with other conditions not recorded on the SNAF.

Urinary incontinence in frail, elderly people living in the community is common and associated with potentially reversible conditions.[2] These conditions can be identified by a more comprehensive, clinically focused assessment instrument.[3]

Mobility problems result in falls, which are known to be an independent risk factor for admission to institutional care.[4] Studies, both overseas[5] and in New Zealand,[6] have demonstrated very clearly that around one-third of falls experienced by elderly people can be prevented.

Although people with dementia are likely to require high levels of family and social support, a recent United Kingdom study has shown that they are less likely to use general practice and hospital consultant services than those with preserved cognitive function.[7] People with dementia often have co-morbidities which, if unrecognised and untreated, may contribute to the need for residential home care.

Assessment prior to residential care admission has three main objectives:

- Ensuring that treatable conditions are identified,
- Ensuring that the elderly person and family have all the necessary information to make an informed decision, and
- Ensuring that public money is spent where it is most needed.

Can we meet all these objectives with a single assessment instrument? There has been considerable work done on developing such an instrument and the Minimum Data Set – Resident Assessment Instrument (MDS – RAI) was first used in the United States in 1991. A home care version was developed and tested in the mid-1990s.[3]

These instruments have two important components. They gather a comprehensive database of information, which is important to have available on a person moving to care. This information can be used subsequently to measure change in a person's clinical situation and improve care.[8]

The instrument also contains clinical triggers so that the person assessing is alerted to areas which need further clinical exploration. There has been international interest in

and use of these assessments. The RAI has proved reliable when used in a variety of countries.

In New Zealand we require an instrument that provides both clinical and economic assessment and that enables international comparisons. Such instruments are available and need to be considered and adapted for New Zealand use. Furthermore, they should be used as part of the assessment of an elderly person prior to the provision of publicly funded continuing care. The assessment instrument is, though, simply a screen indicating the possible need for more detailed professional evaluation. This assessment must be 'comprehensive and multidimensional'.[9]

It is critical, therefore, that the assessment process is closely linked with the District Health Board Assessment, Treatment and Rehabilitation service and with primary care.

The SNAF is not adequate as an assessment instrument. It has well and truly had its day. We need to move on and bring our assessment processes up to international standard.

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Responding to partner abuse: understanding its consequences, and recognising the global and historical context

Dr Felicity Goodyear-Smith's article *Recognising and responding to partner abuse: challenging the key facts* (N Z Med J. 2004 Sep 24;117(1202). URL: http://www.nzma.org.nz/journal/117-1202/1074) is presented in this issue of the *Journal* as a critique of the facts in the recent Ministry of Health (MOH) publication *Recognising and responding to partner abuse, a resource for general practitioners*.

Goodyear-Smith's article recommends a more restrictive strategy for the identification and response to partner abuse than is recommended by the MOH document. This more restrictive approach is not consistent with a broader understanding of the literature. As such, it has the potential to skew our interpretation of research findings in a way that will not constructively advance our responses to this critical public health issue.

One of the overall problems in Goodyear-Smith's article is its assertion that the only consequences of partner abuse to be concerned about are physical injuries, and that prevention efforts should primarily be directed at the small proportion of victims who are likely to be at highest risk of re-assault (as this has the greatest potential to reduce the overall number of assaults).

While it is undoubtedly important to provide high levels of assistance to this multiply abused group (assuming we have the skills to identify them in advance, which is doubtful at present), efforts to prevent these individuals from being further assaulted limits us to achieving only a small part of the reduction in violence, and its attendant health burden, that could be possible. Focusing exclusively on this group would rule out the possibility for GPs to play a role in secondary prevention of abuse, by intervening before violence is entrenched. It also runs counter to strong current evidence that relatively less severe physical violence and non-physical abuse have serious health implications that are not restricted to injuries.

A recent comprehensive review drawing on the strongest studies currently available notes that partner abuse can have a wide range of long-term physical health effects (eg, chronic pain, gastrointestinal and cardiac symptoms, sexually transmitted diseases, vaginal bleeding and infection, chronic pelvic pain, and urinary tract infections), and mental health effects (eg, depression, post-traumatic stress disorder, alcohol and drug abuse).[1]

Understanding the wide-ranging consequences of partner violence is important, because unless GPs and other healthcare providers know about (and consider) the possible role of partner abuse in a present illness, they risk compromising the efficacy of any treatment plans they develop. A strategy that focuses only on the more severe instances of physical violence also does nothing to decrease the lifetime prevalence of partner abuse in the general population.

In addition to clouding the link between violence and multiple health effects, Goodyear-Smith seeks to challenge the acceptability of questioning about partner violence for the majority of patients. She does this by stating that a minority of women may not feel comfortable with being asked about abuse, and that GPs risk causing offence by asking about violence. However, by failing to explore why some women report feeling uncomfortable with disclosing abuse, the author does practitioners a disservice.

To undertake adequate healthcare assessments, practitioners often need to ask about topics that some people may feel uncomfortable about. As with many other areas of healthcare inquiry, the method of inquiry (eg, respectful communication, privacy, safety, and a plan for effective response[2]) is central to reducing discomfort. The author's presentation of the information on acceptability without discussing the importance of the context of questioning, obscures the fact that, when questioned appropriately, the majority of women are not offended by being asked about partner abuse.[3] Not presenting the contextual information also misses an important opportunity for education.

Goodyear-Smith also questions the quality of the literature related to the economic cost of partner abuse and the overlap between child and partner abuse. While some of the criticisms raised have merit, they are presented without heed to the historical context. One of the problems that has inhibited evidence-based policy and practice recommendations in this field is that, while activists have been aware that partner abuse is a problem requiring serious attention for over 30 years, practitioners and researchers have been slower to produce the evidence needed to contribute to the solution. A recent comprehensive review of healthcare response to family violence carried out by the Institute of Medicine in the USA concluded that chronic neglect was the best way to describe the current lack of evidence-based knowledge about key questions in this important field.[4]

Goodyear-Smith states that the Snively report overestimates the economic costs of family violence. However, it is more likely that it underestimates costs. When the work was carried out in 1994, it utilised the best available information relating to prevalence and health consequences. Subsequent work has identified health costings associated with partner abuse that were not included (eg, gynaecological problems), that may be underestimated (eg, treatment for mental health effects), and that indicate that the prevalence rate of partner abuse is substantively higher (ie, Snively calculated estimates based on 14% lifetime prevalence, current lifetime estimates from the 2001 NZNCVS are 27%[5]). The World Health Organization (WHO) and the United States' Centers for Disease Control and Prevention (CDC) have also recently released reports on the economic costs of violence that suggest that partner abuse, along with other forms of violence, places a significant drain on the world's economy.[6,7]

Similarly, the author criticises use of the Edleson reference related to the overlap between child and partner abuse, on the basis that it was not a systematic review. What is not said, however, is that one of the factors limiting the Edleson review was the extreme dearth of studies that have explored the co-occurrence of partner abuse and child abuse. Given the ethical and practical difficulties of undertaking research in this area, at present the Edleson review constitutes the best available evidence we have on this topic, and would seem to be a stronger basis for drawing conclusions than assertions based on no evidence.

The author also challenges the notion that women are more likely to be the victims of partner abuse than men, yet much of the data presented in the article actually supports the veracity of this statement, as does data from the NZNCVS (24.6% of women reported lifetime prevalence of partner abuse, compared with 18.2% of men[5]). Practitioners should also be aware that the consequences of men's violence against women is often more severe, with greater likelihood of both physical injury and death, and greater likelihood of engendering fear. Readers wishing to unpack the critique further are referred to a recent comprehensive review.[8]

Understanding the global context of our response to partner abuse is also important. The guidelines presented in the Ministry of Health document are consistent with best practice suggested by major institutions in countries with values similar to New Zealand (eg, the American Medical Association,[9] and the UK Department of Health.[10]), and the importance of partner abuse as a population health issue is recognised by the WHO[11] and the CDC.[12] The more limited approach to assessment and response advocated by this paper is less consistent with international best practice.

If we in New Zealand are to join the rest of the world community in seeking to address this far-too common problem that places a serious drain on the health of the nation, we have to commit to developing a more refined understanding of the depth and breadth of the problem and mount a better response to it. To do this, however, we must work from a framework that recognises that all violence is potentially harmful, and strive to increase our understanding of ways that violence can be prevented. Goodyear-Smith's article, while reminding us not to let emotion cloud our science, may act to impede the development of a serious response to the whole of this serious problem.

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Risk factors for entry into residential care after a support needs assessment

Mark Weatherall, Timothy Slow, Kimmo Wiltshire

Abstract

Aims To establish the influence of risk factors derived from the national 'Support Needs Assessment Form' for entry into residential care in New Zealand.

Methods Using a retrospective cohort design, data was obtained for assessments of people aged over 65 years over a 12-month interval (August 2001 to August 2002) from an administrative database developed by the Needs Assessment and Service Co-ordination Service for Wellington, New Zealand. The risk factors for entry into residential care were examined by logistic regression.

Results 2060 assessments were carried out over this period for people were aged over 65 years; 67.9% were female. The median age was 83 years, inter-quartile range 78 to 88 years. For 33.4% of people, residential care was recommended. For those aged 80 years or older, the relative risk of residential care was 2.95 (95% CI: 2.27 to 3.82) if continence problems were present, and 3.75 (95% CI: 2.99 to 4.73) for those aged 80 years or younger if continence problems were present. For those with mobility problems who had cognitive impairment the relative risk of residential care was 2.95 (95% CI: 2.25 to 3.87), and 1.77 (95% CI: 1.35 to 2.33) if there were no mobility problems.

Conclusions All of older age, continence problems, mobility problems and dementia predicted residential care after assessment. The effect of continence was more prominent for those aged under 80 years of age. The effect of dementia was more prominent for those with mobility problems. Intervention for continence problems and mobility problems has the potential to reduce the use of residential care.

A new model for the provision of support services for people with activity limitation (disability) was introduced into New Zealand in the early 1990s. In this service model, there was an explicit separation of healthcare services pertaining to the assessment of people with activity limitation—together with the co-ordination of support services for needs identified as part of this assessment from services that provided treatment and rehabilitation for people with disease, impairment, and activity limitation.

The motivations for this policy were complex, but included a (not usually stated) assumption that a social model of activity limitation management would lead to a reduction in costs for the provision of residential care for older people, by allowing so-called 'aging in place'. As part of this policy, a nationally consistent instrument, the Support Needs Assessment Form (SNAF), was introduced.[1]

This instrument was to be used throughout New Zealand to provide for consistency of assessment. In passing, it is of interest to note that the instrument was developed by a consensus approach managed by the New Zealand Ministry of Health and that no

proper clinical assessment of the utility of the instrument was ever performed. The 'separation' policy was implemented to variable extent throughout New Zealand and has not been subjected to any serious scientific scrutiny.

However the use of a standard instrument has given rise to the opportunity to use 'SNAF' data collected about people who request support services to examine what factors might predict the use of long-term residential care as a support service.

In New Zealand, residents of long-term care facilities make up about 6.5% of the population aged over 65 years.[2] There is a high prevalence of disease, impairment, and activity limitation in this very frail population.

A 1993 census of all people living in long-term care in Auckland, the largest city in New Zealand with a population of about one million people,[3] found, for example, that 67% of residents had a mobility disorder and 50% had continence problems. A more recent survey of people living in long-term care in another centre in New Zealand found that nearly three-quarters of residents had dementia.[4,5]

Methods

Background—The Support Needs Assessment and Service Coordination agency based in Wellington, New Zealand has developed an administrative database with details about the assessments carried out and services recommended for people requesting support services.

The database includes items from the modified Support Needs Assessment Form used in Wellington and, in particular, whether a person is thought (by the assessor) to have cognitive problems, mobility problems, or continence problems. In addition, the database includes some details of the services recommended and, in particular, whether residential care services were recommended. This study is based on the 12 months of data from this administrative database, August 2001 to August 2002.

Statistical analysis—This was by logistic regression, modelling the probability that residential care was recommended after the support needs assessment. Potential explanatory variables were age, sex— and whether cognitive problems, mobility problems, or continence problems (urinary or faecal) were present. Interactions between these predictors were also examined. A backwards selection using a probability for leaving a term in the model of 0.1 was used to select the final model. SAS version 8.2 (SAS Institute, NC, 1999) was used.[6]

Results

2060 assessments for people aged over 65 years were performed during in the 12 months. A basic description of the subjects is shown in Table 1.

Table 1 Description of subjects

Variable	Description
Sex	67.9% Female
Age (years)	Mean 82.3 (standard deviation: 7.3) Median 83 (Inter-quartile range: 78–88) Range: 65–104
Residential care recommended	33.5%
Continence problems	30.9%
Cognitive problems	23.8%
Mobility problems	73.6%

The univariate analysis for each potential risk factor is shown in Table 2.

Table 2. Univariate odds ratios for the risk of residential care (higher odds ratio means at greater risk)

Variable	Risk: Odds ratio (95% CI)
Age greater than 80 years versus age less than 80 years	1.71 (1.40 to 2.07)
Continence problems	4.73 (3.87 to 5.79)
Cognitive impairment	3.97 (3.21 to 4.91)
Mobility problems	2.19 (1.74 to 2.76)
Sex (Male versus Female)	1.01 (0.83 to 1.23)

For the multivariate model, interaction terms between age and continence status, and mobility and cognitive impairment, were statistically significant. The final model incorporating terms for age, cognitive impairment, continence problems, and mobility problems as well as the stated interaction terms fit the data well with a deviance of 5.80 on 9 degrees of freedom (p=0.76) and a Hosmer and Lemeshow goodness of fit test with a value of 2.06 on 7 degrees of freedom (p=0.95). The multivariate analysis including interaction terms is shown in Table 3.

Table 3. Multivariate odds ratios for the risk of residential care (higher odds ratio means at greater risk)

Variable	Risk: Odds ratio (95% CI)
Age greater than 80: Incontinent versus continent	2.95 (2.27 to 3.82)
Age less than 80: Incontinent versus continent	3.76 (2.99 to 4.73)
Mobility problems: Cognitive impairment versus no cognitive impairment	2.95 (2.25 to 3.87)
No mobility problems: Cognitive impairment versus no cognitive impairment	1.77 (1.35 to 2.33)

Discussion

Since 1996, several published studies have examined the risk factors for residential care utilisation.[7–23] The most common risk factors were worse activity of daily living status (13 studies), living and family arrangements (11 studies), decreased cognitive function (10 studies), older age (7 studies)—and each of a diagnosis of Alzheimer's disease, caregiver characteristics, worse self assessed health status, and ethnicity (2 to 3 studies).

Two studies found increased domestic support services (that protected against moving into residential care), and one study that showed increased domestic support services were associated with an increased risk of moving to residential care.

Consistent with this previous work, the current study found that in all persons of older age, mobility problems and cognitive problems were associated with a higher risk of entry into residential care. Continence problems were usually not identified as a separate risk factor, but subsumed under the rubric of activity of daily living problems, in the previous work. Medically recognised urinary incontinence has been found to be an important risk factor for entry into residential care.[24]

There was interaction between continence problems and age, with the effect of continence problems being more pronounced for people aged less than 80 years.

There was also an interaction between cognitive problems and mobility problems, with the effect of cognitive problems being more marked in those with mobility problems. Interactions between risk factors are not examined consistently in the previous work in this area; this is an important issue for future research as it appears the risk associated with functional factors on moving into residential care (in particular) depends on the particular functional profile.

Weaknesses of the study include the fact that it relies on an accurate assessment on the part of the healthcare workers completing the 'SNAF'. The healthcare workers who complete the assessment tool have a common training; however, the inter-rater reliability of the healthcare workers has not been established. The simple nature of the coding of problems in the particular areas may mitigate this uncertainty.

Some of the recommendations for residential care were assessments from a lesser to a greater level of dependency within institutional care, as in New Zealand there are two 'levels' of residential care; thus this analysis is not one of purely community dwelling older adults.

For purposes of the simple coding, urinary and faecal incontinence were not distinguished. Finally, the analysed data represents the activities of only one assessment and service co-ordination service, and may not be able to be generalised to other similar services in New Zealand or internationally.

A social model of care may not be an ideal way to promote aging in place. Two recent systematic reviews.[25,26] of home-based support for older people reached similar conclusions.

Home support seems to be useful, and in particular may reduce the risk of nursing home admission—if the home support is accompanied by a multi-dimensional geriatric assessment and follow-up; includes multiple follow up home visits; and furthermore if there is a long-term intervention strategy to modify risk factors for deterioration in health and function.

A recent controlled trial where home-support workers also provided a geriatric assessment and rehabilitation style of care[27] was associated with a lower risk of entering residential care.

Analysis of the influence of home support services on the risk of entry to residential care is conflicting. A report of the influence of home-based support services[28] was unable to demonstrate that provision of home support services was associated with decreased utilisation of residential care, while another report[12] found an association between increased home-support service spending and services, and lower risk of residential care.

The conclusions that can be drawn from the study (reported in this paper for health service planning in New Zealand) are that cognitive impairment, mobility disorders, and urinary incontinence occur more commonly in older adults who have a recommendation for long-term residential care. This recommendation occurred in a very large proportion of subjects.

The success of a social model of care, in promoting 'aging in place', has not been convincingly demonstrated in randomised clinical trials—and given the very high proportion of older people who had residential care recommended in this study, this model of care may not reduce the risk of use of long-term care in New Zealand.

A more collaborative approach, between assessment and service co-ordination services, and geriatric and rehabilitation services (that integrates a clinical, diagnostic, therapeutic and rehabilitative approach to older people) may offer better outcomes.

Those persons at most risk for entry into residential care may benefit the most from this model of care—people with mobility problems, continence problems, cognitive problems, and the very elderly (ie, persons aged over 80 years).

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Circumstances and consequences of falls in residential care: the New Zealand story

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Abstract

Aims To describe the circumstances and consequences of falls in long-term residential care homes in New Zealand.

Methods A study followed 14 residential care facilities over 18 months and recorded all falls experienced by 680 residents (97% participation rate). The number and characteristics of falls are described according to the timing, activities, direction, and severity of falls. Predictors of injury are examined using regression techniques.

Results 271 residents sustained 954 falls, 63% of which resulted in injury. The rate of falls increased in the afternoons especially in rest homes. Environmental hazards were involved with 15% of falls. Falling in the dining room was associated with fewer injuries, and falling from a standing height, and to the side, were independently associated with major injury such as hip fracture.

Conclusion The timing and circumstances of observed falls and their subsequent injuries is useful information for healthcare planners, and staff involved in the care of older people living in long-term residential care settings.

Older people living in residential care facilities are frail, have a high prevalence of co-morbidities and age-related impairments, and are prone to falls[1,2]—this combination makes even a simple fall particularly dangerous.[3]

A high proportion of falls result in injuries, with as many as 25% of falls resulting in lacerations, fractures, and the need for hospital care.[3] Non-injurious falls also have a negative impact on residents, with as many as 75% of fallers experiencing loss of confidence and/or a fear of further falls (with a consequent loss of physical activity and function).[4]

The problem of falls in residential care facilities warrants attention because falls are common and distressing for both residents and their carers. Recent research indicates that factors related to the environment,[5] and to nursing or services provision, could place older people at risk of falls. Among less mobile residents, accidental falls have been found to occur around equipment use or during the 'transferring process'.[6] Descriptions of the time and place of fall occurrences, and the consequences of falls among this frail population are rare in the literature.

This study reports falls sustained in a cohort of residential care facilities in the Auckland region. Risk factors related to resident characteristics are reported elsewhere.[7] This paper describes the fall events, circumstances, and timing under which they took place as well as the consequences of the falls in terms of injury and health services utilisation.

Association between characteristics and timing of falls and injury were investigated. An understanding of the nature of falls is an important step in preventing falls among residents in long term residential care.

Methods

Selection of rest homes—In the greater Auckland (New Zealand) region, a random selection was made of rest homes or low-level dependency facilities and private hospitals or high level dependency facilities (as defined by New Zealand Ministry of Health criteria) using random number tables. Rest homes residents who require assistance with most instrumental activities of daily living and at least two basic activities of daily living, can usually ambulate to some degree and feed themselves. In private hospitals or high-level dependency facilities, residents require assistance with most activities of daily living and usually need daily nursing care. Written informed consent was obtained from managers and residents in the selected homes. The Auckland Ethics Committee approved the study.

All the participating homes agreed to undertake falls monitoring within their homes for the study duration (18 months) starting in December 1999. Half of the study homes (n=7) participated in a fall prevention intervention that began on 28 April 2000. As the intervention may have had an impact on fall incidence rates in intervention homes,[8] falls occurring during the intervention period in intervention homes are excluded. This paper reports all falls that occurred during the 5-month period prior to the intervention as well as falls occurring in control group homes for the 12 months of the intervention period (18 months in total).

Study variables—After enrolment, information was collected about each resident's medical diagnoses, medications, and previous falls, from the resident's medical record. Information listed on the summary sheet as being a diagnosis, problem, or disability was considered a 'diagnosis' for the purposes of this study. Functional status was recorded by the registered nurse or lead nursing assistant. This was measured by a validated scale developed in Sheffield,[9] found to be reliable, and then used in the New Zealand Long Term Care Survey.[10,11]

The composite scale comprises three level scales. One of the validated level scales, self care (mobility, dressing, feeding, bathing, and toileting) was used in this study. Composite scales for mobility (transfers, mobility within the home, and ability on stairs) and behaviour (needing night care, social behaviour, memory, wandering, and awareness) were constructed. Each scale is the summed score of its items, rating the resident as independent, able to do self care with a little help, or needing considerable help. Information was also sought concerning staffing levels using a structured interview with the home's management staff, and this was expressed as the ratio of total staff (nursing and nursing assistants)/total residents.

Falls—A one-page standardised falls surveillance form was developed for staff use, and completed for each fall that occurred. All fall forms were faxed or posted to the research centre and read by an independent researcher using a standard definition of a fall: *'unintentionally coming to rest on a lower level'*.[12] Falls not meeting this definition were excluded. Reporting of falls was audited by a visit to the home and by a hand search of the medical record and incident reports to identify unrecorded falls. Falls records were audited for duplicate reports of the same fall event.

Information sought included:

- Time and date of fall,
- Whether the fall was witnessed,
- Location within the facility or outside,
- Whether the resident was wearing hip protectors, and
- Whether environmental hazards or restraints were involved.

Activity at the time of the fall was recorded and included:

- Whether the fall occurred while the resident was on their feet, or from a lower level such as bed chair or toilet,
- Whether walking, turning, or stretching, and
- The direction of the fall.

Space for a written description of the fall was also provided. Fall-related injuries were documented, as was the type of medical attention required.

Injuries were categorised into minor, moderate, and major injuries:

- *Minor injuries* included: bruises, skin tears, haematomas, and the need for a routine doctor visit.
- *Moderate injuries* included: sprains, lacerations including steri-strips and sutures and the need for an urgent doctor visit.
- *Major injuries* included: joint dislocation, fractures, radiological examination, an Accident and Emergency Department visit, and hospital admission.

Injury categories such as mild, moderate, or severe were created for each fall. To make these categories mutually exclusive, the most severe injury sustained was used to decide the most appropriate category. For example, major injuries were not classified as moderate or mild, even if a skin tear was sustained during a fall resulting in fracture.

Analysis—As there was a potential intervention effect, all falls occurring during the intervention period in intervention homes were excluded[8]. Descriptive statistics were used to describe circumstances and frequencies of falls and injuries by level of care using SPSS.[13]

To describe the timing of falls, a rate of falls was calculated. This was the number of falls occurring per hour (averaged over 2-hour periods) per person, using 648 persons as the number enrolled over the study period, for the overall rate. To calculate fall rates for rest homes, the number of falls was divided by the number enrolled from rest homes. This process was repeated for high-level dependency residents in private hospitals. To adjust for the variable amount of time residents were observed (and the falls being accrued over 18 months), rates were then divided by the average number of days residents were enrolled (overall and for each of the dependency levels), multiplied by 3650 and expressed as falls/hour/10 resident years occurring during each 2-hour period. Graphs are presented in the Results showing distribution of fall rates by level of injury severity over 24 hours.

For this analysis, falls occurring while standing, walking, stretching were combined into one variable 'on feet'. Falls occurring at a lower lever such *as 'a fall on/off bed, chair, or toilet'* and *'transferring on/off bed, chair, or toilet'* were combined to create a variable *'fall from a lower level'.* All falls that were described as a slip, trip, trip over object, or having a hazard involved were coded as involving 'a hazard'.

To establish relationships between fall circumstances and level of injury, logistic regression models were constructed with an injurious fall as the dependent variable. Independent variables were: location, activity, direction of fall, presence of restraints, presence of hazards, and time of day (categorised into 2-hourly time units).

As there are many patient characteristics that predict falls, the following variables were controlled for in the models: level of dependency (type of home), gender, presence of a diagnosis of dementia, number of medications, number of diagnoses, and previous falls. As falls were not independent (for example, several falls occurred in many residents), the model was adjusted for clustering by resident using STATA 7.0 software. An exposure term was included in the models—the variable indicating follow-up time in days for each individual in the trial.

Results

Study population—the residential facilities

Of a possible 206 residential care homes in the Auckland region, 27 rest homes (low level dependency homes) and 13 private hospitals (high level dependency homes) were excluded as being too far from the study centre. Of the remaining 121 rest homes and 45 private hospitals, 14 were randomly selected. One declined to participate and another was randomly selected yielding a 93% response rate. Overall these facilities contained 25 units consisting of 9 rest home units, 9 private hospital units, 6 secure units for those with psychogeriatric disorders, and 1 unit for disabled young people.

Study population—the residents

At baseline, 18 residents (out of a possible 648 residents) in the homes refused to participate, thus yielding a response rate of 97%. Before surveillance began, 10 residents died and 3 were transferred from these homes. Data on the functional status and health of these 13 residents were not available.

Fall surveillance began with 617 consented residents in December 1999 and continued until the end of April 2001. Residents admitted into the homes until November 2000 (n=63) were enrolled in the study. During the study period (18 months), 128 residents died (20%), and 83 (12%) residents were transferred to another residential facility or discharged home. Fall surveillance data was collected on a total of 680 residents. Table 1 shows demographic, health and functional status information on the residents.

Table 1. Patient characteristics in a sample of residential care residents

	Secure units	Private hospitals (n %)	Rest homes (n %)	Total (n %)
Residents	95	196	358	649
Gender, female	63 (66%)	137 (69%)	262 (73%)	483 (71%)
Age m (SD)	83.2 (7.7)	81.6 (14.3)	84.2 (8.7)	83.3 (10.5)
Ethnicity, Caucasian	72 (88%)	146 (85%)	242 (84%)	460 (84%)
Diagnosis of dementia	78 (83%)	95 (55%)	131 (38%)	305 (50%)
Previous fall	60 (64%)	86 (49%)	196 (57%)	343 (56%)
Total medications† m (SD)	5.3 (3.0)	5.8 (3.1)	5.7 (3.1)	5.7 (3.1)
Total diagnoses † m (SD)	4.6 (2.1)	5.0 (2.1)	4.8 (2.1)	4.8 (2.1)
Self care score 1-24* m (SD)	6.1 (4.2)	5.3 (3.3)	12.1 (3.3)	9.2 (4.8)
Mobility score 1-12* m (SD)	4.7 (3.9)	3.0 (2.5)	8.2 (2.6)	6.2 (3.7)
Behavioural score 1-20*m (SD)	8.8 (4.4)	11.6 (5.8)	17.3 (3.8)	14.4 (5.7)
Days observed mean (SD)	140 (96)	309 (174)	217 (167)	232 (170)
Died during follow up (18m)	28 (29%)	55 (28%)	43 (12%)	128 (20%)

SD=standard deviation; m=mean; n=demographic and health data available on 652 residents only; *self care, mobility, and behavioural score were calculated from subscales of the dependency questionnaire (higher score means higher level of function); †diagnoses and medications established from the summary sheet in the medical record and medication chart.

The falls

During the 18 months of surveillance, 2,021 falls forms were faxed to the study office. Of these, 32 were excluded as the fall was described as being due to an epileptic seizure—this yielded a total of 1,989 fall forms. Falls occurring in intervention homes during the intervention period were excluded, leaving 954 falls included in this analysis.

Using residents as a denominator, 271 of 680 residents (40% of all residents) sustained one or more falls and 218 (32% of all residents, 80% of fallers) had an injurious fall; 183 (67% of fallers, 27% of all residents) sustained a minor injury; 66 (24% of fallers, 9.7% of all residents) sustained a moderate injury; and 39 (14% of fallers, 5.7% of all residents) sustained a major injury.

Using falls as the denominator (rather then residents), 605 of the 954 falls (63%) resulted in some form of injury. Self-reported falls totalled 271 (28%). Most fall-related injuries were minor (46% of falls), with 12% of falls being of moderate severity and 44 falls resulting in major injury (5% of falls). About one-third of falls were routinely assessed by the GP and 5% resulted in an urgent visit. One-quarter of falls resulted in skin tears and 20% bruises. Of the major injuries, hip fractures were the most common fracture (n=12) with 36 falls resulted in a radiological examination (4%). The details of injuries sustained from falls and treatments provided is described in Table 2 .

Table 2. Falls and injuries in a cohort of 680 residential care residents over an 18-month period

954 falls	Secure units (n %)	Private hospitals (n %)	Rest homes (n %)	Total [n (%) of total falls]
Falls	45	293	614	954 (100%)
Falls with any injury	36 (80)	206 (70)	361 (59)	603 (63)
Minor injuries	27 (60)	161 (55)	255 (42)	443 (46)
Moderate injuries	6 (13)	32 (11)	78 (13)	116 (12)
Major injuries	3 (6)	13 (4)	28 (5)	44 (5)
Details of injuries:				
- Bruises	14 (31)	57 (19)	123 (20)	94 (20)
- Haematomas	3 (7)	16 (5)	11 (2)	30 (3)
- Skin tears	12 (27)	85 (29)	131 (21)	228 (24)
- Abrasions	7 (16)	19 (7)	42 (7)	68 (7)
- GP visit routine	27 (60)	123 (42)	159 (26)	309 (32)
- Steri-strips	6 (13)	28 (15)	72 (12)	106 (11)
- Sutures	0	0	6 (1)	6 (1)
- Sprains	0	1 (0)	5 (1)	6 (1)
- GP visit urgent	4 (9)	12 (4)	29 (5)	45 (5)
- Hip fracture	1 (2)	6 (2)	5 (1)	12 (1)
- Other fracture	1 (2)	0	4 (1)	5 (1)
- Acute A&E assessment	1 (2)	1 (1)	8 (1)	10 (1)
- X-rays taken	2 (5)	11 (4)	23 (4)	36 (4)
- Hospital admission	2 (4)	5 (2)	12 (2)	19 (2)

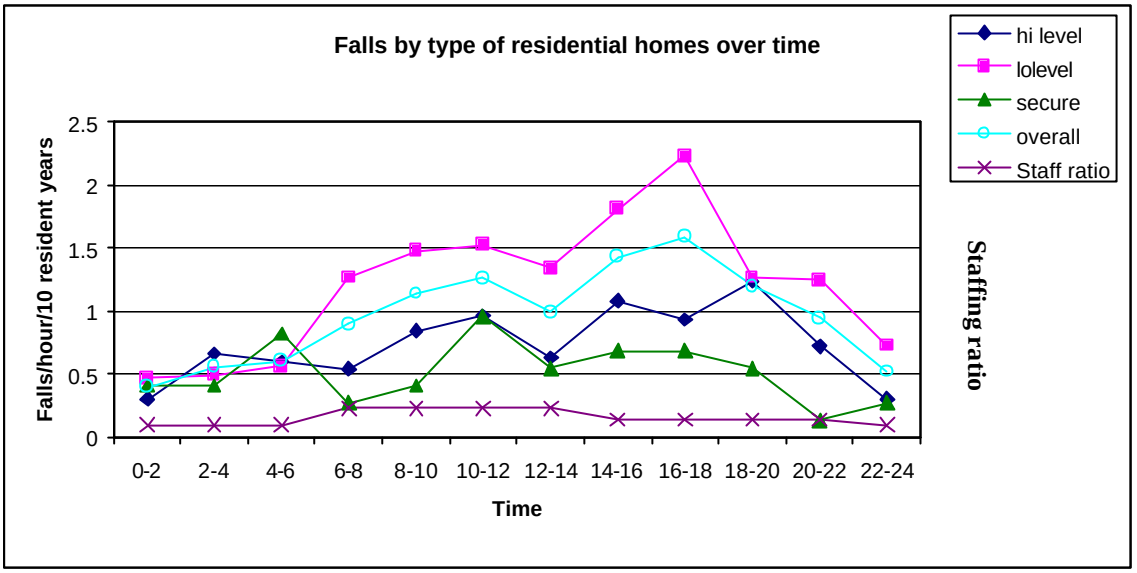
Falls were equally distributed between the days of the week with an average of 136 falls on any particular day of the week. Fall rates tended to be higher during the day when residents were active—with the highest rates observed between 4pm and 6pm (2.2 falls/hour/10 resident years). Falls were mainly contributed by residents in rest homes (Figure 1).

Using logistic regression (adjusted for time in the study, presence of dementia, self-care score, mobility score, behavioural score, age, gender, type of home, and clustering [by resident]), no 2-hour period, compared with the time period 12midnight–2am, was more likely to result in any injury.

Staffing levels in all homes were lowest at night, at intermediate levels in the afternoons, and were highest in the mornings. There was a tendency for falls rates to be higher in the afternoon when staffing ratios were intermediate.

Figure 1. Rate of falls and fall-related injury over 24 hours in a sample of residential care homes in Auckland, New Zealand by (a) type of residence and (b) severity of injury (Using logistic regression, adjusted for time in the study, presence of dementia, self-care score, mobility score, behavioural score, age, gender, type of home, and clustering (by resident); no single 2-hour time period was more likely to result in injury)

(a) Fall rate by type of home



hi level=private hospitals; **lo level**=rest homes; **staff ratio**=staff per shift/total residents.

(b) Fall rate by severity of injury

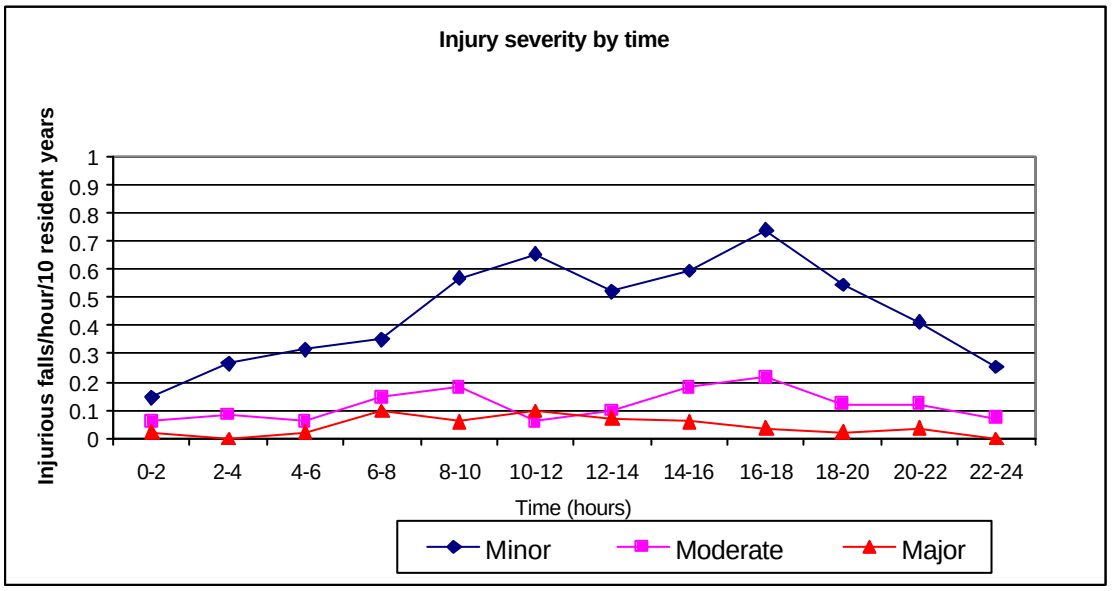
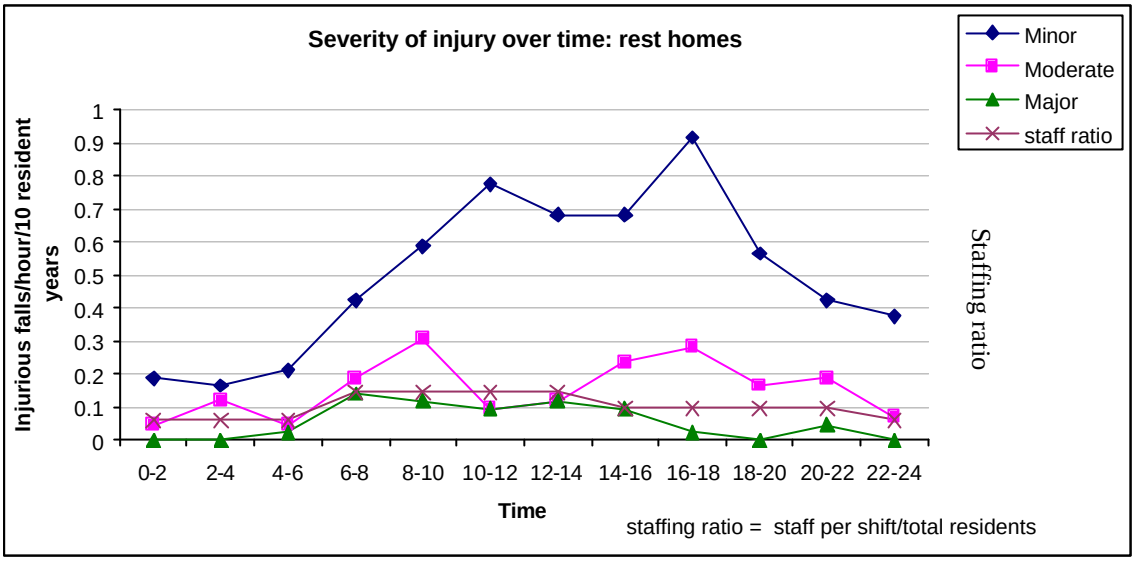
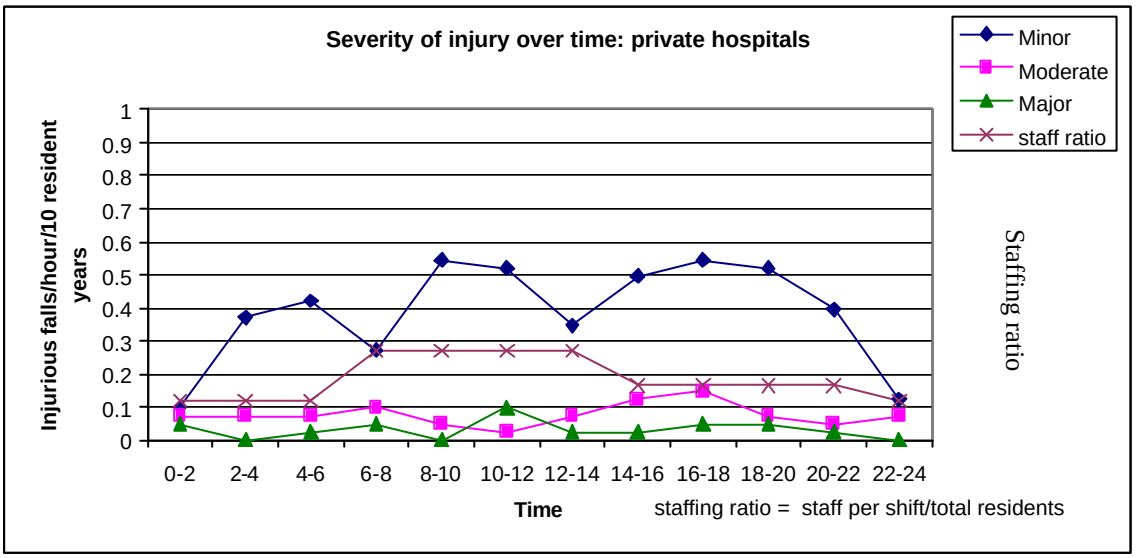


Figure 2. Injury severity over time in (a) rest homes and (b) private hospitals
(Staffing ratio plotted for comparison)

(a) Rest homes



(b) Private hospitals



Injury severity varied throughout the day and differed between the type of home. For example, in rest homes, major injuries from falls were more common in the mornings whereas minor injuries occurred at a higher rate in the late afternoon. Staffing ratios may have been lower when falls with minor injury rates were highest in the afternoons. In the private hospitals and secure units (Figure 2b), falls resulting in moderate and major injuries occurred at similar rates throughout the 24-hour period.

Minor injuries occurred at a lower rate at night compared with during the day. Using logistic regression, adjusted for health and demographic factors, previous falls, time

of day and clustering (by resident) falls occurring in private hospitals OR 2.82 (CI 1.19–6.66) and secure units OR 1.59 (CI 1.02–2.48) were more likely to result in any injury compared with rest homes.

Table 3. Location and activities at the time of falls and fall related injuries in a cohort of residents living in long term residential care facilities

Total falls (n=954)			**Injurious falls**	
	N	**%**	**n**	**%**
*Not seen	729	77%	467	77%
Found on floor	441	46%	296	49%
Indoors	**917**	**96%**	**571**	**95%**
- Residents room	597	63%	378	63%
- Lounge	68	7%	44	7%
- Hallway	79	8%	41	7%
- Toilet	75	8%	51	9%
- Dining room[1]	69	7%	34	6%
- Bathroom[2]	19	2%	17	3%
Outdoors[3]	**35**	**4%**	**31**	**5%**
Activities†				
- Fall while on feet	473	50%	310	51%
- Fall from low level	306	32%	187	31%
Environmental hazard implicated	**142**	**15%**	**95**	**15%**
Fall direction†				
- Sideways[4]	256	27%	182	30%
- Backward	258	27%	151	25%
- Forward	121	13%	85	14%
- Straight down	24	3%	11	2%
- Direction unknown	212	22%	132	22%
- Restraints in use at time of fall	17	2%	13	2%

***Not seen** means the fall was not witnessed, and includes self report falls.

†Percentages do not add to 100 due to missing values.

Logistic regression results (1-4), adjusted for time in the study, presence of dementia, self-care score, mobility score, behavioural score, age, gender, type of home, and clustering (by resident).

[1]OR 0.54 (CI 0.33–0.88) less likely to sustain injury in the dining room

[2]OR 4.67 (CI 1.02–21.36) more likely to sustain injury in the bathroom

[3]OR 4.92 (CI 1.50–16.11) more likely to sustain injury outside

[4]OR 1.70 (CI 1.17–2.46) more likely to sustain injury falling sideways.

The location of falls, and activities noted at the time falls are described in Table 3. Nearly all falls occurred indoors (96%)—the majority of which occurred in the residents room (63%). Adjusting for resident health and functional factors and the type of home, falls sustained in the bathroom were more likely to result in injury OR 4.67 (CI 1.02–21.36). In contrast, falls occurring in the dining room (n=69) were less likely to result in injuries compared with other locations OR .54 (CI 0.33–0.88).

Only 35 falls occurred outdoors, and these were more likely to result in injury OR 4.92 (CI 1.50–6.11). A sideways fall was more likely to result in injury, OR 1.70 (CI 1.17–2.46) whereas other activities and fall circumstances were not associated with

injury. Using the same model with *major injury* as the dependent variable the following circumstances were associated with major injury: fall while on feet OR 2.36 (CI 1.22–4.52) and fall to the side OR 1.69 (CI 1.16–2.47). Falls occurring in between 4pm and 6pm were less likely to be associated with major injury OR .16 (CI 0.03–0.96).

Half of all falls, n=473 (50%), occurred while the residents were on their feet (either standing or walking). A sizeable minority (142, 15%) of falls were recorded as having a hazard involved. These included cords, steps, thresholds, bedroom cluster, mats (n=135), and dim light (n=16). These hazards resulted in falls associated with tripping over an object (n=44) and slipping on wet floors (n=37).

During the study period, 35 falls occurred with hip protectors being worn at the time. No hip fractures occurred as a result of these 35 falls whereas 12 hip fractures resulted from 917 falls occurring without hip protectors in place. Restraints were recorded as in use in 17 falls.

Discussion

Falls in residential care are a major public health concern. The purpose of this study was to describe the circumstances and injuries related to falls sustained by residents living in long-term residential homes in Auckland, New Zealand. Almost half (40%) of the residents sustained a fall during 18 months of surveillance; consistent with other studies.[2]

The majority of these falls resulted in injuries (63%) with 5% of all falls resulting in injuries of major severity, a higher rate for any injury but lower rate for serious falls than other studies.[6,14] Previously, the timing of falls has not been accurately described. When residents were active during the day, they were more likely to sustain injury, and the more active residents fell more but were less likely to injury themselves.

This study identified all falls occurring in the homes, including self-reported falls. We chose to accept self-reported falls and recorded any form of injury. This is justified, as even a minor injury can result in not only decreased mobility and increased morbidity[4] but also increased need for care. Our definition of injury categories has resulted in a higher proportion of falls being categorised as ‘injurious’ (63%) than in other studies which report between 40% and 60% of falls resulting in injury.[12,15]

This paper also uses the fall as the denominator for analysis, rather then the resident, meaning that direct comparison to other studies reporting the proportion of residents injured is less appropriate.

A limitation of this study is the potential for inaccuracy of information regarding self-reported falls that were unwitnessed. These occurred mostly in rest homes, where residents have (in the main) sufficient cognition to report events.

There was no way to validate information about these unwitnessed falls. This was unavoidable as the alternative of running videotapes at all times in all locations within the homes was beyond the scope of this study.

Information about direction and location of the fall was evident to staff when the residents were found and data were recorded. Staff ratios were reported to researchers by the principal nurse manager and averaged over the three usual working-shifts, therefore losing some precision in description.

In addition, injury severity categories were arbitrary, but created after consultation with experts. As the numbers of residents in secure units was small, they were combined with those in private hospitals for the regression analyses. Almost all secure units were placed within private hospitals, thus under similar environmental and staffing conditions, and the fall rates were the most similar between these groups.

It is possible that relationships between circumstances of falls and injury would be better examined in larger samples of secure unit residents separately; however, this was beyond the scope of this study. This study highlights the need for the creation and standardisation of injury severity categories, which would allow for more accurate comparisons between studies.

This paper provides important descriptive information about injuries sustained and treatments provided to a large group of residents which will be useful for health care planners, medical staff and the residential care industry. Most injuries were minor (with bruises and skin tears being most common) as confirmed in other studies.[16] The doctor involved in the routine care of the resident was often involved in the management of fall-related injuries.

This study confirms the finding of peak falls times reported by less rigorous research.[16] The distribution of injurious falls in a 24-hour period is intriguing. The emphasis on higher fall rates in the afternoon in rest homes is important, as this is the time of shift change. It is reasonable to assume that consideration of different ways to deliver care at these times may impact on fall rates, as (notably) the afternoon staffing ratios are lower than the mornings.

In addition, there is a trend for major injuries to occur in the mornings across all homes, a time when maximal staff is available. Mornings are a time of high activity in all homes with toileting, showering, dressing, and breakfasting. Staff often take a morning-tea break, which may make them less available to residents and potentially compounds the risk of major injury at that time. Other studies have shown that non-ambulatory residents are more likely to sustain injury associated with use of equipment and transferring[6] and this may also be a part of the explanation of this finding. Further study is needed to establish the impact of staffing level changes on fall and injury in residential care.

Rest home residents in this study had more falls but were less likely to sustain injury than residents in private hospitals. Furthermore, rest home residents are less frail than those in private hospitals and this will protect them from injury. It must be accepted that active residents are at risk of falls and activity is important for quality of life. The trade off between level of activity and risk of falls deserves more attention in future studies.

The prevalence of falls among residents (40%) in this study is similar to other studies which report 40%–52% of residents sustained a fall.[17] However, our prevalence of injurious falls is higher (23% of residents), perhaps owing to our acceptance of self-reported falls. Thus, a lower rate may have been recorded if a different study design been employed.

While falls in the dining room were less likely to result in injury, falls in the bathroom were more likely to result in injury. This provides additional information useful for planners, managers, and caregivers.

It is reassuring that, in a protected regulated environment, only 15% of falls involved a slipping or tripping hazard. While routine surveillance for such hazards may impact fall rates, it can be postulated that the majority of falls were from intrinsic factors that will require strategies to reduce individual's fall related risk factors.

Protection from fall injuries is very important. The finding that hip fractures were associated with a sideways fall, and that no hip fractures occurred from falls while residents were wearing hip protectors, further supports the efficacy of hip protectors among high-risk individuals in rest homes and private hospitals.[18,19] This is important as an upward trend in the incidence of hip fractures occurring in New Zealand has recently been reported.[20]

The strength of this study is that, in comparison to other studies,[15,16] ascertainment of all falls was achieved and a large sample of residents were prospectively followed. Indeed, the information gathered and analyses techniques used (in investigation of the timing of falls) in this study are unique.

Conclusions

To date, few interventions have been shown to reduce falls among older people living in long-term residential care settings. An understanding of the timing and location of falls within homes provides useful information for healthcare providers concerning staffing levels at differing times of the day—as peaks times of falls and injury may be related to periods of high activity within residential care homes.

It is important for clinical staff to consider intrinsic risk factors for falls, as most falls did not involve environmental hazards. Protection from injury is emphasised by this study—as a sideways fall was related to serious injury and no hip fractures occurred in residents wearing hip protectors.

A variety of strategies involving interventions for individual residents and strategies (about staff, time, and locations in facilities) will be needed to reduce injury. These strategies will, in turn, need to be tested for efficacy.

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Diurnal, weekly, and seasonal variations in stroke occurrence in a population-based study in Auckland, New Zealand

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Abstract

Aims To determine (via a population-based study in Auckland, New Zealand) if there are diurnal, weekly, or seasonal variations in the occurrence of stroke.

Methods All new stroke events in Auckland residents were traced. Time of onset of the stroke was defined as the time when neurological symptoms were first noticed. The day of the week and month of onset were analysed for all strokes.

Results 1711 patients were registered over 1 year. The rate ratios for the onset of stroke in each 6-hourly interval compared with reference interval (1800–2359 hours) were 0.74 (95% CI: 0.61–1.10) for 0000–0559 hours, 2.88 (95% CI: 2.48–3.34) for 0600–1159 hours and 1.74 (95% CI: 1.49–2.05) for 1200–1759 hours. Rate ratios of the seasonal occurrence of stroke compared with spring were 0.75 (95% CI: 0.65–0.86) for summer, 0.83 (95% CI: 0.73–0.95) for autumn and 1.08 (95% CI: 0.96–1.23) for winter. No weekly pattern of stroke occurrence was observed.

Conclusions Strokes were less likely to occur during the summer and autumn than in the winter or spring. There was an increase in the occurrence of stroke in the late morning. The results have implications for the provision of acute stroke services in the community and in hospital.

Temporal variations in the occurrence of stroke may provide insight into the factors that trigger the onset of a stroke. Most studies have identified a circadian variation in the time of onset of stroke with peak occurrence between 0600 and 1200 hours.[1]

There is less information available on variations in the day of the week and season of onset of stroke. Conflicting results in previous studies can be explained by differences in study design (hospital-based versus population-based studies), environmental conditions, risk factor profiles, and relatively small numbers of events available for analysis. There is little information available on the temporal patterns of stroke occurrence in countries with a temperate climate.

Comparison of incidence studies of stroke in various climates may help to identify the mechanisms underlying seasonal variations in stroke onset. The aim of our study was to analyse the diurnal, weekly and seasonal variations in the occurrence of stroke in a large population-based study in Auckland, where there is relatively little variation in temperature throughout the year.[2]

Methods

The methods used in the Auckland Stroke Study have been described previously.[3] All new stroke events in residents of the Auckland region (study population approximately 945,000) in the 12 months

beginning 1 March 1991 were traced through multiple case-finding sources. These included patients who died before they were seen by a doctor, patients managed at home or in long-term care institutions, and Auckland residents who had been admitted with a stroke to a hospital outside the study area.

A stroke was defined as rapidly developing symptoms and/or signs of a focal or, at times, global loss of cerebral function lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin. Subarachnoid haemorrhages were included in the main study, but data on the diurnal, weekly, and seasonal onset of subarachnoid haemorrhage were not included in this report.[4] We report only first-ever in a lifetime events.

The date and time of onset of the stroke was obtained during an interview conducted by a study nurse. For fatal events or severely disabled patients, the interview was held with close relatives or other observers who were familiar with the patient's health, but otherwise the interview was with the patient. Interviews were conducted as soon after the event as possible, except for fatal cases in which the interview was deferred for 6 weeks. The time of onset of the stroke was defined as the time when the patient or an observer first noticed neurological symptoms or signs.

The time of onset was distributed into six hourly intervals: 0000–0559, 0600–1159, 1200–1759, and 1800–2359 hours. When the time of onset was unknown (either because symptoms were first noticed on awakening, or the history was unreliable and the patient was alone at the time of onset), the patient was excluded from analysis of the diurnal variation in the time of onset.

A sensitivity analysis was performed to evaluate the possible effect of excluding patients who first noticed symptoms on awakening. The results were reanalysed by including these patients and assuming that the time of stroke onset was evenly distributed over the first eight hours of the day. The day of the week and month of onset were analysed for all strokes. The month of onset was analysed in seasonal quarters: summer (December–February), autumn (March–May), winter (June–August), and spring (September–November).

Rate ratios (RRs) were computed using Poisson regression allowing for under- and over-dispersion, in which incidence rates of stroke occurrence for a particular time interval were compared with that of a reference interval. Corresponding 95% confidence intervals (CI) were estimated. The population at risk was included in the models as offsets. We evaluated effects of age and sex by means of stratified analyses and adjustment. Age of the patients was conventionally dichotomised into two groups: 15–64 years and 65 years or more. All calculations were performed using SAS version 8.0 software.[5]

Results

A total of 1711 patients (excluding patients with subarachnoid haemorrhage) were registered for the study period of 1 year. The time of day of the onset of stroke was available for 1497 of the stroke events. Reliable information on the time of onset was not available for the other 214 patients (12.5%). The day of the week and the season of onset were known for all events. The age and gender structure of the patient population and the rate of hospitalisation have been reported elsewhere.[3]

Diurnal, weekly and seasonal distribution of the onset of strokes are presented in Tables 1, 2, and 3. The crude rate ratios of the temporal patterns of stroke occurrence are shown in Table 4.

The rate of occurrence of stroke was highest in the late morning (0600–1159 hours) compared with other times of the day, regardless of gender or age group of the patients. The risk of stroke in the afternoon (1200–1759 hours) was approximately two times higher as compared with the evening. If patients who had awakened with symptoms were included, and the time of onset was evenly distributed over the first 8 hours of the day, there was still a significant peak occurrence between 0600 and 1159 hours (Table 5).

Table 1. 24-hour distribution of onset of stroke by 6-hour periods (number of events and rate per 100,000 person-years)

	Men						Women					
Time	<65 years		≥65 years		total		<65 years		≥65 years		total	
Interval (hr)	n	rate	n	rate	n	rate	n	rate	n	rate	n	rate
0000-0559	35	133	57	1673	92	310	19	71	64	1509	83	262
0600-1159	79	301	241	7072	320	1080	44	164	313	7380	357	1127
1200-1759	57	217	122	3580	179	604	36	134	195	4598	231	730
1800-2359	35	133	84	2465	119	401	24	89	92	2169	116	366

Table 2. Distribution of onset of stroke by day of the week (number of events and rate per 100,000 person-years)

	Men						Women					
Day	<65 years		≥65 years		total		<65 years		≥65 years		total	
	n	rate	n	rate	n	rate	n	rate	n	rate	n	rate
Monday	23	50	90	1238	113	208	22	49	110	1883	132	260
Tuesday	38	83	66	908	104	192	20	44	111	1900	131	258
Wednesday	30	65	91	1252	121	223	16	36	132	2260	148	291
Thursday	28	61	80	1100	108	199	13	29	114	1951	127	250
Friday	39	85	99	1362	138	254	18	40	131	2242	149	293
Saturday	38	83	91	1252	129	238	13	29	100	1712	113	222
Sunday	25	54	61	839	86	158	31	69	81	1387	112	220

Table 3. Seasonal distribution of onset of stroke (number of events and rate per 100,000 person-years)

	Men						Women					
Season	<65 years		≥65 years		total		<65 years		≥65 years		total	
	n	rate	n	rate	n	rate	n	rate	n	rate	n	rate
Summer	40	52	115	1141	155	177	20	25	171	1194	191	204
Autumn	43	54	136	1320	179	200	37	46	174	1188	211	220
Winter	65	82	170	1649	235	262	50	62	225	1537	275	287
Spring	73	93	157	1540	230	259	26	32	209	1443	235	254

Table 4. Crude rate ratios of temporal patterns of stroke occurrence (RR and 95% CI)

Variable	Crude RR
Time of 24-hour day	
0000–0559	0.74 (0.61–1.10)
0600–1159	2.88 (2.48–3.34)
1200–1759	1.74 (1.49–2.05)
1800–2359*	reference
Day of the week	
Monday*	reference
Tuesday	0.96 (0.80–1.15)
Wednesday	1.10 (0.92–1.31)
Thursday	0.96 (0.80–1.15)
Friday	1.17 (0.99–1.39)
Saturday	0.99 (0.83–1.18)
Sunday	0.81 (0.67–0.97)
Season	
Summer	0.75 (0.65–0.86)
Autumn	0.83 (0.73–0.95)
Winter	1.08 (0.96–1.23)
Spring*	reference

*Reference variable; RR=rate ratio; CI=confidence interval.

Table 5. Sensitivity analysis: crude rate ratios of stroke occurrence including patients who awakened with symptoms

Time of 24-hour day	Crude RR (95% CI)
0000–0559	1.29 (1.09–1.53)
0600–1159	3.24 (2.80–3.75)
1200–1759	1.74 (1.49–2.05)
1800–2359*	reference

*Reference variable; RR=rate ratio; CI=confidence interval.

The risk of stroke was lowest in the summer, when there was a reduction of about 25% compared with spring. The risk in autumn was about 20% lower than in spring. There was a statistically insignificant trend towards an increased risk of stroke in winter. No particular weekly pattern of stroke occurrence was observed.

Discussion

The major strength of this study was its population-based design with the use of multiple overlapping sources of recruitment that ensured complete case ascertainment. This, together with the uniformly conducted, structured interview of patients and their relatives, minimised selection and information biases. The main drawback of the study was the low rate of imaging. For this reason, it was not possible to obtain reliable information about the temporal occurrence of different subtypes of stroke.

Diurnal patterns—In Auckland, there was a significant increase in occurrence of strokes between 0600 and 1159 hours. Our findings are consistent with a systematic review of 31 studies that reported the time of onset in more than 11,000 strokes.[1] In this review, there was a 49% increase in the occurrence of all types of stroke between

0600 and 1200 hours compared with the number expected if no circadian variation was present. Ischaemic and haemorrhagic strokes each had a significantly higher risk of occurrence between 0600 and 1200 hours, and lowest risk between midnight and 0600 hours.

A peak occurrence of stroke in the late morning appears to refute the concept that a nocturnal reduction in blood pressure and cerebral blood flow is an important trigger for atherothrombotic ischaemic stroke. Diurnal variation in blood pressure parallels the circadian rhythm of stroke, tending to be highest between 0600 and 1200 hours.[6]

An increase in blood pressure may precipitate an ischaemic stroke by triggering haemorrhage into an atherosclerotic plaque, or initiating the coagulation cascade. Therapeutic reduction of blood pressure in the morning may help to prevent stroke. Circadian variations in haemostatic function also may account for a predominance of strokes at the same time.

Circaseptan patterns—A consistent variation in the timing of strokes during the week has not been observed in previous studies.[6–12] The results of our study did not support a particular weekly pattern in the occurrence of strokes. Variations in the occurrence of stroke within the week in different studies may be explained by chance variation.

Seasonal patterns—Most studies on the seasonal variation of stroke have shown a peak occurrence in mortality,[13] and hospital admission rates[14–16] during the winter. The winter peak occurrence of stroke is inversely correlated with environmental temperature.[13] Hospital admission rates and mortality may not mirror the incidence of stroke. Higher hospital admission rates with stroke in the winter may reflect a greater need for hospital admission in cold weather rather than a higher incidence.[17] Pneumonia is a common cause of death in patients who have had an acute stroke. An increase in case fatality during the winter may reflect a higher frequency of respiratory tract infections in the winter.[17]

Conflicting results on the seasonal variation in the occurrence of stroke have been reported in population-based studies. In some of these studies there was a peak in incidence in the winter, or the winter and spring,[18–20] but there were no significant seasonal variations in the overall incidence of stroke in population-based studies in Oxfordshire,[17] Framingham,[7] and Italy.[21] Seasonal variations in the occurrence of different stroke subtypes were seen in Oxfordshire and Framingham.[7,17]

Most studies on seasonal variations in the occurrence of stroke have been conducted in countries that experience extremes in temperature between seasons. No seasonal variation in stroke occurrence, or hospital admissions was observed in subtropical countries.[22,23]

Admissions to hospital with an ischaemic stroke were more common on hot days in some subtropical countries,[22,24] and peaks in stroke mortality may occur during heat waves.[13,25] There may be a U-shaped relation between temperature and mortality from cerebral infarction.[26]

By contrast, mortality from intracerebral haemorrhage decreased with increasing temperature in China,[26] and the occurrence of intracerebral haemorrhage doubled on cold days compared with warm days in Taiwan.[23] In Auckland, there was a significant seasonal variation in the onset of strokes, even though there is relatively little

variation in temperatures during the year. Mean monthly air temperatures in Auckland vary from 10.8°C to 19.8°C.[2]

Seasonal variations in the occurrence of stroke may reflect fluctuations in risk factors. Blood pressure is significantly higher during the winter.[27] Lower environmental temperatures may predispose to development of a stroke by increasing blood pressure through induction of peripheral vasoconstriction.

Seasonal variations in the frequency of infections also may contribute to a higher incidence of ischaemic stroke in the winter.[28] Respiratory infections may increase the risk of arterial thrombosis by increasing plasma fibrinogen and inhibition of fibrinolysis by endotoxins. An alternative explanation for the apparent association between infection and stroke is that patients with cerebrovascular disease may be more vulnerable to develop or die from infections and these deaths are then attributed to the stroke.

Thrombolytic treatment improves the long-term outcome of patients with acute ischaemic stroke, but treatment must be started in the first three hours after the onset of symptoms.[29] Patients who wake up with symptoms can not receive thrombolytic treatment because the time of onset is uncertain.

Identification of the peak time of onset of strokes has implications for the provision of acute stroke services. In Auckland, 45% of strokes occurred between 0600 and 1200 hours. An acute general practice consultation is more difficult to obtain before 0900 than at other times of the day. This may explain why stroke patients who seek general practice consultation take longer to reach hospital than if they call the ambulance service.

Hospital stroke specialists are often committed to ward rounds and outpatient clinics in the morning, and may not be immediately available to assess patients who present at this time with an acute stroke. Recognition of these delaying factors may facilitate changes in management that will lead to earlier treatment of acute ischaemic stroke.

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General Practice care of enduring mental health problems: an evaluation of the Wellington Mental Health Liaison Service

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Abstract

Aims To describe the development and evaluation of a primary care service for a population of mental health consumers who had previously been predominantly cared for by a specialist service.

Methods Consumers were interviewed at regular intervals after entry to the programme. The Health of the Nation Outcomes Scale (HoNOS) and the Life Skills Profile (LSP) outcomes measures were used with consumers at intervals of 0,3,6,12 and 18 months after entry to the programme. A cost comparison was made between services provided to consumers in the year before entry to the programme, and after entry.

Results Consumers reported no deterioration in their clinical condition while under the care of general practitioners, and they were largely satisfied with general practitioner care. Consumers' LSP scores were stable after entry to the programme. General practitioners were initially ambivalent about the programme, but were more supportive after 12 months had elapsed. The education provided to general practitioners, nurses, and receptionists was strongly welcomed.

Conclusions With carefully designed training and support, general practice can provide high-quality community-based mental healthcare for consumers with enduring mental health disorders, and it can support the introduction of integrated mental health care initiatives.

The delivery of mental health services present several important challenges to health systems. In New Zealand, these challenges include the management of an apparently high prevalence of mental disorder[1,2] and disparities in the distribution of illness[3–5] within a context of broader changes in health systems and society.

This paper describes a project, which aimed to provide appropriate and effective mental healthcare for a well-defined consumer group by developing routine primary care services (in conjunction with mental health consumers, specialist clinicians, and general practice staff), and presents results from an evaluation of the project.

Both specific local pressures upon the existing service and a wider international trend to integrate primary and secondary care services contributed to the conception and design of the programme.

Locally, the community mental health service had been under financial pressure and identified significant constraints in effectively managing the volume of consumers under its care. There were long waiting times for access to the service, causing concerns among GPs and the community generally.

It was felt that several consumers (deemed to have relatively low clinical needs) remained within the specialist service because of barriers to discharge.

Perceived barriers included:

- Concerns that GPs were not well trained to manage mental health patients,
- Concerns that primary and secondary care clinicians were not able to share information effectively,
- Concerns that out-of-pocket costs would prevent mental health consumers from accessing GP services, and
- Concerns that general practices did not have the infrastructure to manage flexible appointments and recall patients.

These circumstances caused frustration for consumers, GPs, and specialist services. For patients whose illness had stabilised, there was a possibility that they were not receiving their mental healthcare in the most appropriate setting, and that their recovery was being impeded. There were also problems in addressing their physical health needs.

Nationally, there was some evidence that GPs were prepared to do more mental health work if:

- Barriers of cost and time could be overcome,
- GPs were provided with adequate education and backup from specialised services,[6] and
- A greater alignment of primary and secondary services would fit with overall government health policy.[7]

Internationally, there is a resurgence of interest in using primary care to provide mental health services. In the past decade, a number of researchers have identified roles for general practice in mental health care.[8–11] The development of an effective relationship between primary care and specialist services has been a particularly strong theme—as patients with mental health problems find themselves being cared for in the community,[11–13] and as primary care services have themselves undergone change.[14]

Given the imperatives for change, the Wellington Independent Practice Association (WIPA) and the local government hospital organisation Capital Coast Health (CCH) agreed to work together and with the Wellington Mental Health Consumers Union (Inc) to develop a new programme of general practice based mental healthcare.

Methods

Programme elements—The criteria for entry to the programme were that a patient should have:

- A **Diagnostic and Statistical Manual of Mental Disorders** (DSM) Axis 1 diagnosis,
- A clear need for ongoing mental health support,
- Been reviewed by a psychiatrist within the previous 6 months,
- Been a holder of a community services card (entitling them to partial patient subsidies for GP visits), and
- Consented to join the programme.

Since the beginning of the programme, the entry criteria have been expanded to include people referred from their GP onto the programme. The Wellington Regional Ethics Committee granted ethical approval at the outset of the new programme.

The main structural features of the programme were joint governance arrangements (at both strategic and local level); a new specialist team staff role (the primary care liaison worker); education and support for general practice staff; free GP consultations; and new interface protocols between primary and secondary care.

The joint governance arrangements were developed by convening various committees to oversee the project as a whole, as well as to manage specific aspects such as education. Local steering groups were also convened to manage the implementation in different geographic areas. Each group had representation from mental health consumers, specialist service clinicians, GPs, and primary and secondary care managers. The education group also involved a practice nurse and academics. Involving consumers at all levels of governance and management was an important element in the design and development of the programme.

The primary care liaison worker was a new role established within the specialist community mental health service, which had an exclusive focus upon supporting the consumer in the transfer from specialist care to primary care. The role of the liaison worker involved providing information about the programme to consumers, supporting the consumer with key decisions (such as choosing a GP), helping the GP and consumer to develop a care plan ,and co-ordinating the practical arrangements such as meetings between the parties involved in the process. The liaison workers were available on a consultancy basis to general practices after the transfer had taken place. The liaison workers collected much of the data subsequently used in the evaluation.

The primary care education programme was intended for all practice staff who had contact with mental health consumers—including GPs, practice nurses, and receptionists. The topics were chosen by surveying GPs to find out what needs they had in the area, and by considering the likely needs of consumers who would be on the programme. The GP syllabus involved six modules, which covered Maori mental health, care planning, AIMS testing, acute phase management, schizophrenia management, and the management of bipolar disorder. Nurses and receptionists attended a full day session designed in close collaboration with mental health consumers, which addressed stigmatisation and discrimination issues. In addition there were ongoing clinical education sessions for practice nurses.

As well as the series of seminars, the programme developed a resource manual for general practice (aimed at providing GPs and practice nurses with information to support mental health consumers in primary care). The manual contained a range of decision support tools and guides, as well as practical information about the programme.

Free GP consultations were achieved by using specialist service funding, which was paid to GPs as a flat capitation fee. The capitated amount was paid quarterly in advance to GPs (who agreed not to charge for visits from programme consumers). The amount was based upon an estimated 12 to 15 visits per year for each consumer.

The central aspect of the interface between the specialist service and the general practice was the care plan, which was developed jointly in each case by the primary care liaison worker, the consumer, and the GP (to encourage ownership of the document by all parties). The care plan set the parameters for both the patient and the GP, thus defining the circumstances in which external help and advice might be sought.

The care plan defines the components of treatment and recovery, and also the expectations of all involved in the patient's care. It is individualised to the patient's needs and is consistent with their understanding. Containing details of the patient's clinical condition and treatment, it outlines goals and objectives of management in each case, and identifies safety and other liaison issues to ensure that the patient, GP, and liaison worker had a consistent and clear action plan.

The evaluation sought to establish whether consumers reported changes in satisfaction and health outcome in comparison to their experience before enrolment—and whether the GPs who were directly involved reported greater satisfaction with the new service arrangements. The evaluation also assessed whether the new arrangements were provided at a lower overall cost without any concomitant decrease in quality of care.

Following the introduction of the scheme in 1999, an initial evaluation report was completed at the end of 2000 and data collection has continued since then.

Consumers enrolling on the programme were asked for written consent to be a part of the evaluation. Consumers were able to enrol on the programme without consenting to take part in the evaluation.

Consumer interviews—Programme staff developed structured interviews in consultation with consumers and the Wellington School of Medicine. A consumer fieldworker administered the interviews to consumers at 3, 6, and 12 months after they entered the programme. The interviews took place in a setting of the consumer's choice—usually the consumer's home. The interviews elicited each respondent's views about the information they received prior to transfer, their care plan, the transfer process, the care received in general practice, and their general satisfaction and preference regarding the new arrangements.

Health status—Health outcomes were measured with a range of standard tools, which were variously designed for measuring either general health status or mental health status. The tools were the Health of the Nation Outcomes Scale (HoNos)[15] and the Life Skills Profile.[16] The 'EuroQoL', 'SF-36', and 'Brief Psychiatric Rating' scales were also used at the beginning of the programme, but were later discontinued to reduce the burden of data collection. This wide range of tools was used initially to test the practical implementation of these techniques in the service delivery setting.

The outcomes questionnaires were used at 3, 6, 12, and 18 months after each consumer had entered the programme, and were conducted in an interview with the primary care liaison worker.

Cost analysis—Cost analysis was carried out by comparing the funding of the primary care programme and the cost of any secondary services used by consumers who were on the programme with the cost of secondary care services provided to the participating consumers in the year before they had entered the programme. The analysis did not consider the training, support and administrative costs of either primary or secondary care services.

Results

To date, 370 consumers have entered the programme. 163 consumers agreed to participate in the evaluation, although some consumers declined to provide all of the information requested. In all cases, more recently transferring consumers have been in the programme for a shorter period of time, so measures taken at greater times after transfer cover a small number of consumers.

Diagnoses

Table 1 describes the number of consumers/patients by diagnostic category. The most common diagnosis of programme consumers is for schizophrenia and other psychotic disorders.

Table 1. Diagnosis of programme consumers

Definition	n	%
Depression	89	24%
Mood disorder with psychotic component	99	27%
Others (PTSD, anxiety, dementia)	11	3%
Personality disorder	9	2%
Schizophrenia and other psychotic disorders	162	44%

PTSD=post-traumatic stress disorder.

Consumer interviews

The following data is taken from a sample of 100 people who were interviewed at 3 months post-transfer—2 of those at 12 months, 35 at 24 months, and 13 people at 36 months.

The semi-structured interview primarily addressed the predetermined themes of the transfer process—the care plan, reaction to care provided in general practice, and overall satisfaction or dissatisfaction with the scheme. Summaries of comments from respondents, supported by verbatim quotes, were written during the interview, and collated by the researcher.

Transfer Process

A range of views was expressed about the transfer to general practice. While some consumers were pleased to leave the mental health services,

> 'I didn't mind, I didn't feel pressured, it seemed like the right thing to do. I was given good advice and I followed it'

some expressed ambivalence and others were initially upset about the transfer process.

> 'I was asked but basically I got the impression that they wanted you to go on the scheme. Like the person had a target to meet. The implication was that had to. Obviously the philosophy behind it is to save money'

The process was aided by the work of the Primary Care Liaison Worker, and consumers felt adequately informed (by the Liaison Workers' explanation and information booklet) prior to transfer. Consumers were selected for the transfer process and while many did not feel pressured into accepting or declining transfer there were some who felt they had little choice.

Care plan

> 'I know what is written down rather than it being a mystery going through the system'

Consumers appreciated the apparent transparency of information with having a Care Plan—although the level of consumer involvement in the development of the plans was not always sufficient for consumers to have a sense of 'ownership'. The Care Plan was perceived by some consumers to be equivalent to a formal contract with their GP. As there was no mechanism for updating care plans, consumers felt there was the potential for them to be perceived as a historical rather than a 'living document' with consequent loss of relevance

General practice

The move to a general practice environment was a major transition for many consumers. Some were initially unsure of their GP's knowledge base regarding mental health/illness, and were intimidated by the time constraints of appointments. The physical environment of general practice was unfamiliar, and waiting in noisy waiting rooms was perceived as stressful. There was concern at loss of contact with a psychiatrist, and the perceived difficulty of getting enough support with non-clinical issues.

> ‘My biggest worry is if we become stressed or don't communicate and one of us slip and go down what would happen. Before the nurse would visit or I could call the nurse. But now I don't know what would happen’

For the majority, doctors and nurses in general practice were perceived as supportive—and receptionists played an important role in making people feel comfortable and welcome. ‘Convenience of location’ and ‘not paying for GP appointments’ were major benefits for interviewees, and many commented on the benefit of seeing a GP for physical health as well as mental health. For some consumers, the change to general practice improved their self-esteem and supported them to consider and initiate further personal development.

> ‘He is helping me physically and mentally. I have been able to put on some weight and maintain it. He has been helping me to improve my breathing and I have been trying to give up smoking. That means that I have more money’

Most consumers were able to manage the transition to independence from ongoing community mental health team (CMHT) support. While some missed the specialist knowledge, a nurse or key worker visiting them at home and a range of avenues that provided them more time to talk through issues, others relished the opportunity to be outside of formal mental health service frameworks.

Health status

The outcomes measures that were recorded for the longest period were the LSP and HoNOS scales. The LSP produces an aggregated global score for each consumer on a scale from 39 to 156, which can be averaged across a population of consumers. Table 2 shows the mean LSP score for consumers at the time of entry to the programme, and then at 3, 6, 12, and 18 months after entry. The scores are stable, and do not significantly differ over time at the 95% level of confidence.

Table 2. Mean life skills profile scores

Months from transfer	n	Mean LSP Score	95% CI
0	91	142.5	(140.4–144.6)
3	67	143.5	(141.1–145.9)
6	60	142.0	(136.9–147.1)
12	46	138.5	(131.3–145.7)
18	20	145.8	(142.5–149.1)
Overall weighted average		**142.2**	

LSP=Life Skills Profile.

The HoNOS scale measures the number of consumers who fall into different categories of problem severity, ‘0’ being least severe and ‘4’ being most severe. Severity is measured upon a number of dimensions.

Those patients chosen for analysis in this evaluation were:

- Suffering physical illness or disability problems.
- Problems with depressed mood.
- Problems with activities of daily living.

In each of these dimensions, the proportion of consumers in the least severe category remains high, and does not show fluctuations which are significant at the 95% level. Table 3 shows the proportions of consumers who are in the '0 severity category' on these dimensions. The proportion of consumers who are in the lowest severity category remains stable within 95% confidence limits over an 18-month period.

Table 3. Consumers with low 'Health of the Nation Outcomes Scale' (HoNOS) risk

Dimension	Months from entry	n	Consumers with severity level '0'	%	95% CI
Suffering physical illness or disability problems	0	98	64	65%	(54%–77%)
	3	65	34	52%	(36%–69%)
	6	56	34	61%	(44%–77%)
	12	45	23	51%	(31%–72%)
	18	22	18	82%	(64%–100%)
Problems with depressed mood	0	98	65	66%	(55%–78%)
	3	65	42	65%	(50%–79%)
	6	56	37	66%	(51%–81%)
	12	45	32	71%	(55%–87%)
	18	22	17	77%	(57%–97%)
Problems with activities of daily living	0	98	75	77%	(67%–86%)
	3	65	40	62%	(46%–77%)
	6	56	38	68%	(53%–83%)
	12	45	31	69%	(53%–85%)
	18	22	17	77%	(57%–97%)

Cost analysis

Costs were collected for the pre-transfer utilisation of 60 consumers in the secondary care setting, and for the secondary care used in the first 12 months after transfer. This was added to the cost of funding the Primary Care Liaison Worker position, calculated to manage a caseload of 60 transfers per annum. Therefore we calculated a one-off cost per enrolled consumer by dividing the 1.5 full-time equivalent (FTE) salary by 60. The cost of GP utilisation is calculated as the cost of the annual capitation payment.

The comparison of costs for 12 months prior to transfer, the first 12 months of participation in the programme, and for subsequent annual periods on the programme is shown in Table 4. Even with the additional one-off costs associated with the Liaison Worker, post-transfer service costs in the first year following transfer run at 88% of pre-transfer service costs. In subsequent years, when the Liaison Worker transfer costs no longer apply, the service cost is estimated at less than 20% of pre-transfer costs.

Table 4. Health utilisation costs (New Zealand dollars) before and after transfer

	Units per month	Unit cost	Annual cost	Savings
Pre-discharge service costs				
Psychiatry (monthly rate)	0.366	$62.24	$746.86	
CMHT member (monthly rate)	1.403	$131.24	$1,574.85	
IAU days used	0.08	$32.13	$385.54	
			$2,707.25	
Post-discharge service costs year 1 year				
Psychiatry (monthly rate)	0.014	$2.43	$29.16	
CMHT member (monthly rate)	0.03	$2.81	$33.66	
IAU days used	0	$0.00	$0.00	
CATT contacts used per month	0.1	$10.76	$129.09	
GP capitation payment			$394.00	
PCLW transfer costs			$1,800.00	
			$2,385.91	$321.34
Estimated post-discharge service costs year 2 onwards				
Psychiatry (monthly rate)	0.014	$2.43	$29.16	
CMHT member (monthly rate)	0.03	$2.81	$33.66	
IAU days used	0	$0.00	$0.00	
CATT contacts used per enrolled month	0.026	$2.78	$33.32	
GP capitation payment			$394.00	
PCLW transfer costs			$0.00	
			$490.14	$2,217.11

CMHT=Community Mental Heath Team; IAU=Inpatient Acute Unit; PCLW = Primary Care Liaison Worker; CATT = Crisis Assessment Triage Team.

Discussion

The findings of this evaluation suggest that it is possible for general practice to provide high quality mental health care in the community to a group of consumers with high needs. Several issues regarding the role of the GP in this work have been identified in other studies.[17] These include a whole-person approach to care; challenges in communication and access issues, acknowledging the transition in working styles, and attitudes for both GPs and CMHTs.

The evaluation has attempted to bring together both quantitative and qualitative approaches to highlight ways in which the programme might be regarded as successful. The HoNOS scores indicate no significant changes in acuity providing some reassurance to those concerned about the overall safety issues in GP care with this group of patients.

The results from the qualitative interviews (which noted a number of positive consequences of receiving general practice care) are consistent with the findings of the quantitative outcomes measures (which showed that consumers remained in a good state of both physical and mental health after they had transferred to the programme). Providing good generalist care which addresses both mental and physical health needs is seen as a positive feature of the scheme and is recognised internationally as one of the benefits of GP involvement.[18] Although the cost analyses have used easily obtainable data (rather than a formal economic evaluation), it appears that a general practice-based service is no more expensive than that based within specialist services.

Some other critical elements in the sustainability of the programme are as follows. The transfer process was organised around dedicated primary care liaison workers providing a link between consumers and general practice teams. Involving consumers in the design, implementation, and governance of both the programme and the evaluation provided a sense of ownership and safety for consumers.

Care plans were used as a tool to individualise care, and this has been the subject of ongoing development. The extensive education and support services, which were provided for this programme, resulted in a service that was successful in providing safe patient care for a group of patients with high needs, and with a lower cost of service provision.

These results are subject to a number of limitations arising from the setting for the research, which was a programme evaluation rather than a dedicated research project. The tension between evaluation and immediate clinical needs contributed to sporadic data collection and lower than optimal response rates were also an inherent consequence of the field of the evaluation.

It is not always appropriate (or reasonable) to expect consumers (who are addressing their own mental health issues) to spend time with interviewers or other evaluation workers. The low response rate could have introduced a reporting bias, and we believe it is important that other schemes of this type are subject to formal evaluation. It should also be noted that the data was collected during a period of diminishing GP incomes, and the financial incentives may need to be revised in order for the programme to be sustainable.

Despite these limitations, the consistency of the messages reported from different elements of the evaluation lends strength to the findings. The greater involvement of general practice in the management of enduring mental health disorders is desirable from both clinical and resource perspectives. This evaluation demonstrates that with commitment from consumers and health professionals in both primary and secondary care such involvement can be successful and cost effective.

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Under-reporting of energy intake in the 1997 National Nutrition Survey

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Abstract

Aims To estimate the level of under-reporting of energy intake by gender, age, ethnicity and body size (normal, overweight, obese) in the 1997 National Nutrition Survey (NNS97) in New Zealand.

Methods Data were from 4,258 participants (1,808 men and 2,450 women aged 15 years and over) who completed the 24-hour diet recall; the primary methodology used in the NNS97. Under-reporting was assessed using the ratio of reported energy intake to estimated resting metabolic rate (EI: RMR_{est}). Cut-off limits were used to identify percentages of under-reporters in the various subgroups.

Results Mean EI: RMR_{est} was 1.40 for all participants (1.51 for men, 1.30 for women, $p<0.001$) with older age being associated with lower EI: RMR_{est} ($p<0.001$). There were no significant differences in mean EI: RMR_{est} between ethnic groups for men. Mean EI: RMR_{est} for women were: Maori 1.46, European 1.29, and Pacific 1.37 ($p<0.01$). A larger body size was associated with a significantly lower EI: RMR_{est} especially for women.

Percentages of 'definite' under-reporters (individual EI: RMR_{est} <0.9) were as follows: men 12%, women 21%; Europeans 16%, Maori 23% and Pacific 26%; normal weight (11%), overweight (19%) and obese (27%) participants; and from 10% in the youngest to 23% in the oldest age group ($p<0.001$ for all results).

Conclusion In this study, in agreement with the literature, women, older people and obese people under-reported more than men, younger people and non-obese people. Possible ethnic differences in under-reporting rates need further study. Care is needed in interpreting the energy intake data from the NNS97.

Under-reporting of total energy intake (hereafter referred to as 'under-reporting') is one of several potential sources of measurement error in all types of dietary surveys, and is a common and acknowledged problem. However, the extent of under-reporting in the 1997 National Nutrition Survey (NNS97)[1] and ethnic differences between European, Maori, and Pacific Islanders have not been studied.

The gold standard method for assessing the validity of reported total energy intake is through doubly-labelled water studies which can accurately assess total energy expenditure. However, because of the high cost of these studies, under-reporting of energy intake is most commonly measured by comparing reported energy intake with an individual's estimated basal metabolic rate (BMR) or resting metabolic rate (RMR).

There is a strong positive relationship between BMR, RMR, or total energy expenditure (all of which are very accurately measured) and weight or body mass index (BMI). In other words, the higher the body weight, the more energy is used to

maintain that weight.[2] However, the results of many dietary studies show either no relationship, or a negative relationship, between self-reported energy intake and weight or BMI.[3,4] Since energy intake equals energy expenditure (at weight maintenance), there must be significant under-reporting of energy intake by people in the higher weight and BMI range.

Goldberg et al have suggested that while it may not be possible to improve on the quality of food intake data in dietary studies, what is important is that the possibility of bias (including bias due to under-reporting) is acknowledged and quantified, and that the data are examined and interpreted with this in mind.[3,4] The aims of this study were to estimate the levels of under-reporting by gender, age, ethnicity, and body size in the NNS97 database.

Methods

The NNS97 survey was conducted by the University of Otago, using the primary methodology of multiple-pass 24-hour diet recall (24-HDR).[1,5] For this analysis, data from individual participants were excluded if key variables such as height and weight were missing. Asians were excluded due to their small numbers. After these exclusions, a total of 4,258 participants (1,808 men and 2,450 women) aged 15 years and over remained for the analysis.

Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). A body size variable was created by grouping BMI into three categories: normal weight, overweight, and obese. The BMI ranges used were those suggested for New Zealanders of different ethnicities [6] as follows: for Europeans: normal weight: BMI<25 kg/m^2; overweight: 25 kg/m^2≤BMI<30 kg/m^2; obese: BMI≥30kg/m^2; for Maori and Pacific people: normal weight: BMI<26 kg/m^2; overweight: 26 kg/m^2≤BMI<32 kg/m^2; obese: BMI≥32 kg/m^2. Ethnicity was self identified.[5]

Resting metabolic rate (RMR_{est}) was estimated using several steps. Fat mass (FM, in kg) was calculated from BMI, using equations from Swinburn et al.[7] These equations were different for New Zealand European, Maori, and Samoan males and females (Samoan equations were used for the whole Pacific ethnic group). Fat free mass (FFM, in kg) was calculated by subtracting fat mass from weight. Finally, RMR_{est} was calculated using an equation from Bogardus et al [8] as follows: RMR_{est} (kilocalories per day) = (22.8 x FFM) + 489. RMR_{est} in kilocalories per day was converted to kilojoules per day by multiplying by the standard conversion factor of 4.184.

The ratio between energy intake (EI) and RMR_{est} (EI: RMR_{est}) was calculated by dividing EI by RMR_{est}.[4] Cut-off limits for identifying under-reporting were taken from the work done by Goldberg et al [3] where they used basal metabolic rate (BMR), which is virtually identical to RMR. Cut-off values for evaluating energy intake using the ratio EI:BMR vary according to the sample size and the number of days of diet intake records. The 95th percentile lower cut-off values for EI:BMR based on 1 day of intake (as data were from a 24-HDR) were used to define 'definite' under-reporting in individuals and in population subgroups. Cut-off values for EI: BMR based on 1 day of intake ranged from 0.9 for one person, to 1.53 for a group of 2,000 people.[3] The 0.9 cut-off value was used to classify individuals, so that participants with an EI: RMR_{est} <0.9 were considered 'definite' under-reporters. The group with an EI:RMR_{est} ≥0.9 clearly contains a mixture of adequate reporters, under-reporters and over-reporters.

Because of unequal selection probabilities for participants, all statistical analyses took into account the sampling weights associated with the design of the study. Weighted means and standard errors of the mean (SEM) were calculated either unadjusted or after adjusting for potential confounders, using the statistical package STATA (StatCorp. 2001 Stata Statistical Software: Release 7.0. College Station, TX: Stata Corporation). The percentages of under-reporters calculated also took into account the unequal selection probabilities.

Table 1. Baseline characteristics of participants*

Characteristics		European	Maori	Pacific	All
Number	Male	1451	253	104	1808
	Female	1896	388	166	2450
Age (yrs), mean (SEM)	Male	43.2 (0.61)	34.8 (1.17)	34.2 (1.38)	41.9 (0.54)
	Female	44.6 (0.61)	35.3 (1.16)	34.9 (1.61)	43.1 (0.53)
Height (cm), mean (SEM)	Male	175.4 (0.25)	174.4 (0.47)	174.7 (0.85)	175.3 (0.23)
	Female	162.2 (0.21)	162.1 (0.41)	162.1 (0.61)	162.2 (0.18)
Weight (kg), mean (SEM)	Male	78.9 (0.46)	87.3 (1.66)	95.0 (2.29)	80.4 (0.45)
	Female	67.1 (0.43)	75.2 (1.13)	84.7 (2.45)	68.7 (0.41)
BMI (kg/m^2), mean (SEM)	Male	25.7 (0.13)	28.7 (0.53)	31.0 (0.63)	26.2 (0.13)
	Female	25.6 (0.16)	28.7 (0.43)	32.2 (0.86)	26.2 (0.15)
Body size, § n (%)**					
Normal	Male	607 (45.1)	85 (42.5)	†17 (14.7)	709 (43.7)
	Female	938 (52.2)	147 (39.0)	†43 (24.0)	1128 (49.5)
Overweight	Male	640 (41.9)	98 (29.8)	60 (59.7)	798 (41.1)
	Female	580 (30.3)	141 (33.0)	†50 (28.0)	771 (30.6)
Obese	Male	204 (13.0)	70 (27.7)	†27 (25.6)	30.1 (15.2)
	Female	378 (17.5)	100 (28.0)	73 (48.0)	551 (19.9)

BMI=body mass index, SEM=standard error of the mean.

*The unequal selection probabilities have been taken into account.

**Percentages relate to body size in each ethnic and gender subgroup (ie, males: % normal + % overweight + % obese = 100%).

†Limited sample size within that cell, n <50, and data should be interpreted with caution.

§See methods for definitions.

Table 2. Ratio of reported energy intake to estimated resting metabolic rate ($EI:RMR_{est}$)*

Characteristics		Male		Female		All	
		mean	SEM**	mean	SEM	mean	SEM
All:		1.51	0.018	1.30	0.014	1.40	0.012
Ethnicity:†	European:	1.52	0.019	1.29^{a}	0.016	1.40	0.012
	Maori:	1.50	0.063	1.46^{b}	0.045	1.48	0.038
	Pacific:	1.37	0.074	1.37^{a}	0.075	1.37	0.053
Age (y): ‡	15-29:	1.64^{a}	0.039	1.42^{a}	0.034	1.53^{a}	0.026
	30-39:	1.61^{a}	0.041	1.35^{a}	0.031	1.48^{a}	0.025
	40-49:	1.54^{a}	0.038	1.27^{b}	0.028	1.40^{b}	0.024
	50-59:	1.38^{b}	0.031	1.22^{c}	0.025	1.30^{c}	0.020
	65+:	1.20^{c}	0.028	1.17^{d}	0.026	1.19^{d}	0.019
Body size: §	normal:	1.54^{a}	0.027	1.39^{a}	0.021	1.46^{a}	0.017
	overweight:	1.55^{a}	0.026	1.25^{b}	0.026	1.42^{a}	0.020
	obese:	1.31^{b}	0.036	1.15^{c}	0.025	1.23^{b}	0.022

*The unequal selection probabilities have been taken into account. ** Standard error of mean

†Adjusted for age and body size (and gender in the case of the last column 'all')

‡Adjusted for ethnicity and body size (and gender in the case of the last column 'all')

§Adjusted for ethnicity and age (and gender in the case of the last column 'all'). See methods for definitions

a, b, c, etc: Mean values for each population characteristic in the same column with different superscript letters are significantly different from each other, $p<0.01$.

Results

The baseline characteristics of the participants, 2450 women and 1808 men, are shown in Table 1. European men and women were 8–10 years older than the Maori and Pacific participants. The patterns of higher mean BMI and greater prevalence rates of obesity in Maori and Pacific people compared to European as shown here have previously been reported.[1]

Mean values of EI:RMR_{est} (and SEM) for all participants, and for various subgroups, are presented in Table 2. Mean EI:RMR_{est} for all the participants was 1.40. Overall, females had a significantly lower EI:RMR_{est} than men (1.30 versus 1.51, $p<0.001$, adjusted for age, ethnicity, and body size).

There were no significant differences in mean EI:RMR_{est} between different ethnic groups in men (adjusted for age and body size) but Maori women had a significantly higher mean EI:RMR_{est} compared to European and Pacific women ($p<0.01$). Mean EI:RMR_{est} decreased with age (adjusted for ethnicity and body size). After adjustment for age and ethnicity, obese men and women had significantly lower EI:RMR_{est} compared to overweight and normal weight groups ($p<0.001$), and in women the EI:RMR_{est} of the overweight group was also lower than that of the normal weight group ($p<0.001$).

The mean EI:RMR_{est} values from Table 2 were compared to the cut-off values from Goldberg et al, as discussed in the methods. [3] Cut-off values for one day of intake were used, and varied from a cut-off value of 1.47 for 104 Pacific men, to a cut-off of 1.53 for all 4258 participants. In all subgroups shown in Table 2, the mean EI:RMR_{est} was below the suggested appropriate cut-off value from Goldberg et al, except for the 15–29 year and 30–39 year age groups of men. The prevalence of 'definite' under-reporters, defined as a cut-off value for an individual's EI:RMR_{est} of <0.9[3,9] was 12% in men and 21% in women.

Figure 1 shows the percentage of 'definite' under-reporters in the different ethnic groups. In contrast to the mean EI:RMR_{est} data, the ethnic differences from this analysis were statistically significant for men ($p=0.0004$) but not for women. Figure 2 shows the percentage of 'definite' under-reporters in the different age groups. Differences between age groups in men, women and the total group were statistically significant ($p<0.0005$). The percentage of 'definite' under-reporters in normal weight, overweight and obese groups is presented in Figure 3, with significant differences found across the groups in men and women ($p<0.0001$).

Figure 1. Percentage of 'definite' under-reporters (EI:RMR_{est}<0.9) by *ethnic group*. Significant effects of ethnicity for men (p=0.0004) but not women.
(These percentages take the unequal selection probabilities into account)

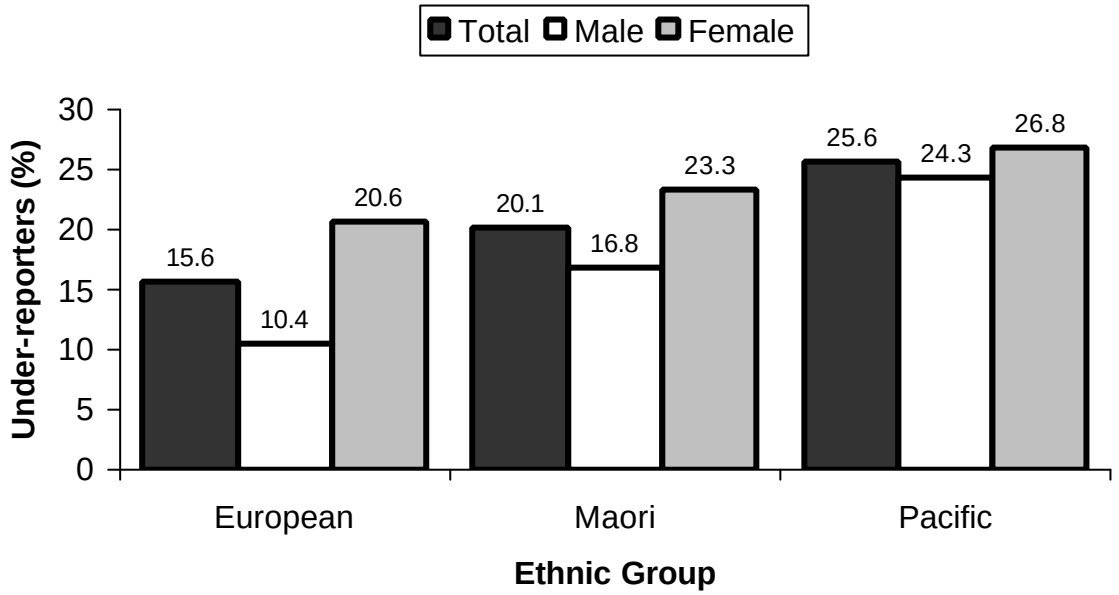


Figure 2. Percentage of 'definite' under-reporters (EI:RMR_{est}<0.9) by *age group*. Significant effects of age (p<0.0005) for both men and women.

(These percentages take the unequal selection probabilities into account)

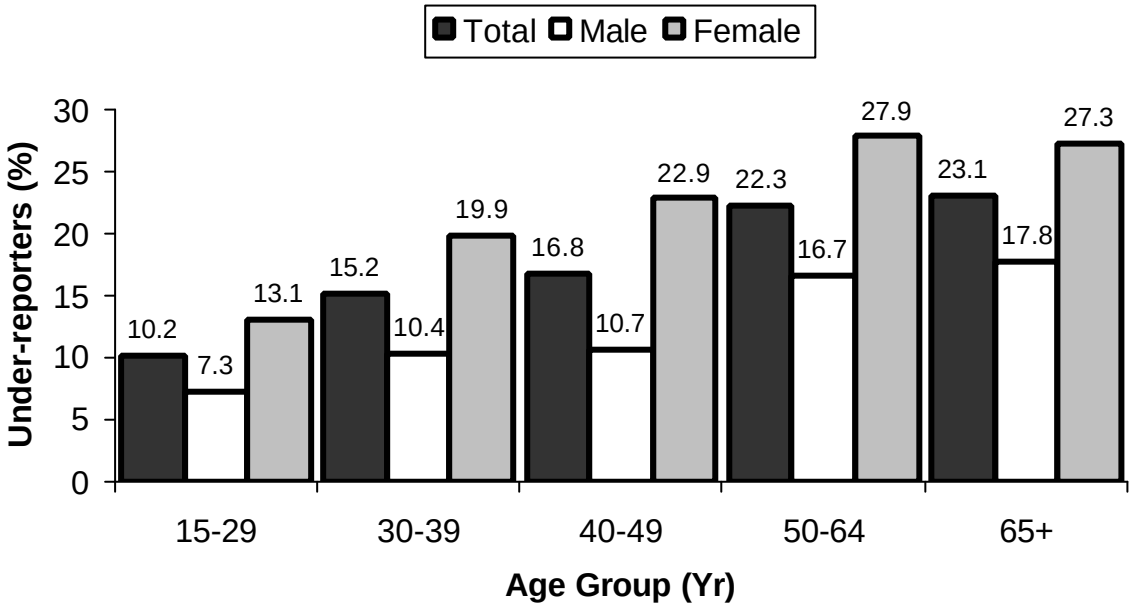
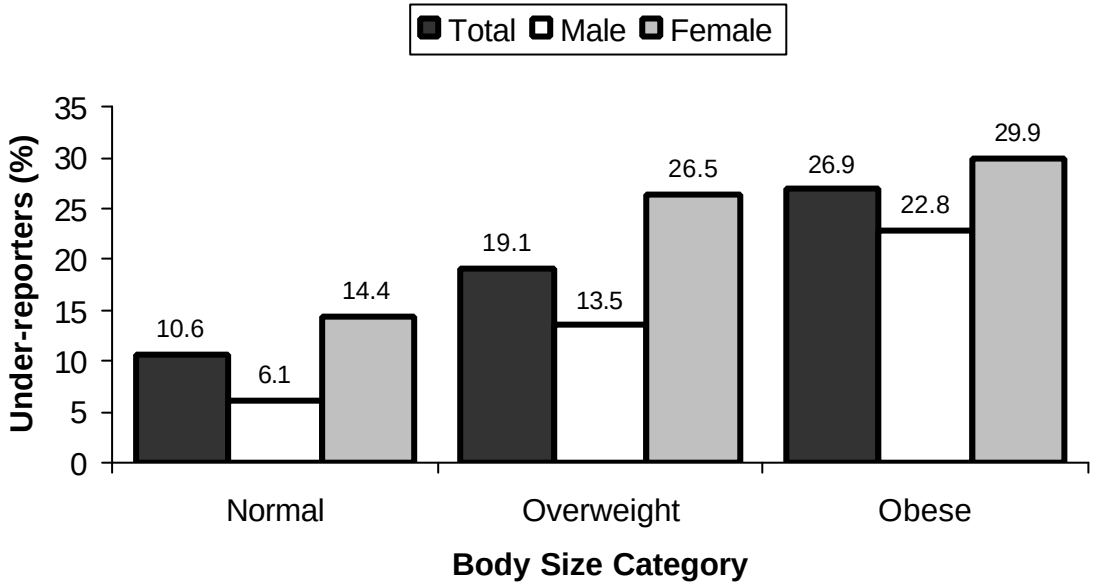


Figure 3: Percentage of 'definite' under-reporters (EI:RMR_{est}<0.9) by *body size category*. Significant effects of body size (p<0.0001) for both men and women.
(These percentages take the unequal selection probabilities into account)



Discussion

This study examined the extent of under-reporting in the 1997 National Nutrition Survey. We found a substantial level of under-reporting across most subgroups analysed. Overall, 12% of men and 21% of women reported energy intakes of less than 90% of their estimated resting metabolic rate (RMR_{est}) and were considered 'definite' under-reporters. In addition, under-reporting was significantly higher in older age groups, and those classified as overweight (women only) or obese. These patterns have been well described in other studies.[9–12]

There is some evidence in the literature suggesting that under-reporting may be more common in members of ethnic minority groups.[9–11] However, in the present study, the results from two different analyses of under-reporting by ethnic group gave mixed results. Using the mean EI:RMR_{est} data, European and Pacific women seemed to have more under-reporting than Maori women. However using EI:RMR_{est} <0.9 cut-off value, under-reporting seemed most prevalent in Pacific, lower in Maori and least prevalent in European women. Low numbers in the Maori and Pacific groups may be contributing to this uncertainty.

A re-examination of the distribution of EI:RMR_{est} by gender and ethnicity showed that there were several very high individual values for EI:RMR_{est} in Maori women, which caused a very positively skewed distribution, and this may explain the higher mean value of EI:RMR_{est}. The range of EI:RMR_{est} values was also wider in Maori women. The median EI:RMR_{est} for all three ethnic groups (in men and women) were slightly lower than the mean EI:RMR_{est} values, but in Maori women the median EI:RMR_{est} was much lower than the mean.

How does the level of under-reporting in the NNS97 compare with other surveys internationally? The NHANES III survey in the US also used 24-hour dietary recall methods. Briefel et al carried out an analysis of under-reporting in that survey.[9] Their data analysis methods were similar to those used here, using the same cut-off value of 0.9 for EI:BMR_{est} (or EI:RMR_{est} as used here), derived from Goldberg et al.[3] Mean values of EI:BMR_{est} in their analysis (1.47 and 1.26 in men and women respectively) were lower than in the NNS97 and the percentages of 'definite' under-reporters were correspondingly higher (18% of men and 28% of women).

Comparisons with other large studies in the literature are more difficult to make, as the methods of assessment of dietary intake vary and include 7-day diet diaries, 3-day diet diaries, and food frequency questionnaires. In a meta-analysis by Black et al, a mean value for EI:BMR of 1.43 (for men and women combined) was calculated for all the studies analysed (all methods), while the mean value for studies using the 24-HDR method was 1.31.[4]

The NNS97 investigators[1] felt that estimating under-reporting using the Schofield equations[13] (which use body weight rather than fat free mass for estimating BMR) might not be appropriate for use in the New Zealand population. As the Schofield equations were developed in a normal weight population (up to 84 kg), but more than 25% of the NNS97 survey population had a weight exceeding 84 kg, the equations could not be assumed to be valid in this group. We have addressed this issue by using fat free mass to calculate RMR. Since New Zealand equations for estimating fat mass were available,[7] fat free mass and then RMR could therefore be estimated.

Fat free mass has been shown to have a much tighter relationship than body weight with RMR, and is the best available determinant of energy expenditure, explaining about 80% of the variance observed between individuals.[14–16] Several authors have strongly supported using prediction equations for BMR which incorporate fat free mass rather than body weight, as these would allow more accurate estimation of BMR, especially in population groups of varying body size and composition.[17,18] As already discussed, RMR and BMR are virtually equivalent and may be substituted for one another. Other studies have used fat free mass in prediction equations for resting metabolic rate (RMR) and for 24-hour energy expenditure.[8,14–16]

The limitations of this analysis can be considered in two broad groups, concerning firstly the methodology and the data collection in the NNS97 survey itself and secondly the methods used here to analyse the data. The NNS97[1] was linked to the concurrent New Zealand Health Survey.[19] Of approximately 9,000 people who participated in the New Zealand Health Survey, and who were invited to participate in the linked NNS97, only 4,636 completed the 24-HDR in the NNS97—an overall response rate of only 50.1%.[1] The possibility of selection bias should therefore be borne in mind when interpreting the data.

An analysis of non-responders (people who took part in the New Zealand Health Survey but not in the NNS97) suggested that the NNS97 sample had similar characteristics to the New Zealand Health Survey sample. [1]

The primary methodology used in the NNS97 was a 24-hour diet recall (24-HDR).[1,5] The data from the 24-HDR have been analysed in this study. Other dietary assessment methods used in dietary studies include retrospective questionnaires of typical diet and prospective diet records (usually for 3 to 7 days, either weighed or quantified in some other way). All dietary assessment methods are subject to bias, usually towards underestimation of habitual energy intake, but the 24-HDR method tends to give lower intakes than other methods.[4] Other issues for the 24-HDR method include intra- and inter-individual variability, day-to-day variability in food intake, weekday and weekend variability, and seasonal variability.

The gold standard method for measuring energy expenditure is to use the doubly-labelled water (DLW) technique.[4,20–22] The rationale for doing a DLW validation study as part of a nutrition survey is enable assessment of the accuracy (or level of inaccuracy) of the dietary intake data with a greater degree of certainty than merely by calculating the ratio of EI:BMR_{est} or EI:RMR_{est}. Energy expenditure can then be compared with self-reported energy intake, and an assessment of the degree of under-reporting can be made. The DLW method has been used in several small New Zealand studies [23,24] but is expensive. No DLW validation study was performed in the NNS97; however the recently completed New Zealand Child Nutrition Survey included a DLW study.

Since the early 1990s, various authors have recommended that the data collected in dietary surveys should include information regarding physical activity level (PAL), dieting and weight-consciousness [4,20,25–29] as well as DLW studies, in order to be able to assess the validity of the survey results and the level of under-reporting. Black has recently concluded that in order to assign the correct Goldberg cut-off values[3] to subjects in dietary surveys, sufficient information on their level of activity is essential.[27, 28]

Conclusions

This study highlights the difficulties of accurately measuring dietary intake through self-reporting methodologies such as the 24-hour diet recall, and the need to acknowledge and to attempt to measure the bias inherent in dietary assessment methods. Analyses such as the present one estimate the level of under-reporting and identify the subgroups with potentially greater levels of under-reporting—namely women, older people, overweight, and obese people. For more accurate estimates of under-reporting, validation studies using doubly-labelled water methodologies[4,20–22] in those subgroups would be needed, as well as collection of information regarding physical activity level and dieting.[25–29]

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Doctors, elder abuse, and enduring powers of attorney

Frances Matthews

Abstract

There is widespread ignorance among doctors and other professionals dealing with incapacitated patients, about the scope and nature of powers granted when an enduring power of attorney is donated. This article discusses some of the shortcomings in the legislation contained in Part IX of the Protection of Personal and Property Rights Act (1988), and the appropriate course of action for those who think that attorneys are misusing their powers and failing to act in the best interests of incapacitated patients.

In New Zealand, the Protection of Personal and Property Rights Act (1988), hereafter termed the PPPR Act, is used to protect vulnerable adults who are unable to make their own decisions about welfare and property matters. Such people lack the capacity to understand the nature, and to foresee the consequences, of decisions in respect of matters relating to personal care and welfare [s.5(a)]; and lack the capacity to communicate decisions in respect of those matters [s.5(b)]. They may wholly or partly lack the competence to manage...their own affairs in relation to...property [s.25(1)(b)]. The Act is administered by the Family Court.

There are two means of protecting people who may be suffering from reversible or irreversible conditions such as brain injury, dementia or intellectual handicap:

(1) By means of court appointed welfare guardians and property managers for those who lack the capacity to make their own decisions.

(2) By means of an attorney appointed by the donor while he or she has the capacity to make decisions.

This article discusses shortcomings in the legislation concerning the appointment and conduct of attorneys contained in Part IX of the PPPR Act, and suggests some steps that doctors can take when dealing with those who have been granted an enduring power of attorney by their patients. There is widespread ignorance about the scope and limitations of enduring powers of attorney, and about the appropriate course of action for those who believe that attorneys are misusing their powers.

Who can donate an enduring power of attorney (EPA)?

Any competent adult can donate an enduring power of attorney (EPA). It may be done at the same time as making a will, and many lawyers encourage their clients to do so as part of sensible estate planning,[1] or it may be done in later life when worries about personal health and the possibility of dementia may be uppermost in a donor's mind.

An EPA is seen as a cheap and accessible way of planning for the future by nominating a person such as family member or close friend to make welfare decisions in the event that the donor becomes incapacitated; for example, by illness or accident (s.98). The same person (s.99), or another person/persons (s.97), or a trustee

corporation may also be given power of attorney in relation to property and financial matters. EPAs in relation to personal welfare only come into force when the donor becomes incapacitated. The power given may be of a very general nature, or may be very specific, depending on the wishes of the donor.

Powers in relation to property can be general or specific according to the wishes of the donor and may come into force at any time regardless of the donor's capacity, or may only come into force in the event of the donor's incapacity. The donor must use an appropriate form specifying their wishes, and the document must be signed and dated by both donor and attorney, and witnessed.

Who can be an attorney?

The Act specifies categories of people who cannot act as attorneys because they would already have been deemed incompetent in some or all areas. An attorney must be not less than 20 years of age, must not be bankrupt, and must not be subject to a personal order or a property order. They must not be special or committed patients under the Mental Health Act 1969. In other words, the attorney must be an adult and must be competent to act for the donor. The enduring power of attorney ceases to have effect if the attorney falls into any of the above categories; dies; or has his or her powers revoked by the courts (s.105), or by the donor; or otherwise becomes incapable of acting (s.106).

There is no mechanism to ensure that an EPA is revoked if the attorney becomes incapable of acting (for whatever reason), including being a special or committed patient. The Act does not further define being 'otherwise incapable of acting'. Suffering an incapacitating mental or physical illness due to dementia, stroke, or serious injury may all qualify.

GPs may find that one of their patients has donated an enduring power of attorney to a spouse or child who then suffers from a physical or mental illness which renders them unable to make decisions on their own, or the donor's behalf. Alternatively GPs, and others, may be unaware of the existence of an EPA.

Rights and responsibilities of attorneys, property managers, and welfare guardians

There are more checks and balances operating on those appointed by the courts than on attorneys, and welfare guardians' and property managers' duties are more closely defined. (see Table 1 and Table 2).

Table 1 Responsibilities of welfare guardians, property managers and attorneys

Variable	Welfare guardian	Property manager	Attorney
Consult person	Yes	Yes	No
Consult others	Yes	Yes	No
File statements	–	Yes	No
Finite time	Yes	Yes	No
Best interests of person?	Yes	Yes	Not specified

Property managers appointed by the courts have a duty to consult the person whose property they manage, and others who are interested in the welfare of the person (s.43), and must prepare and file statements with the court at regular intervals (s.45). Statements filed by individual managers may be reviewed by the Public Trustee or nominated accountants (s.46). Any person, with the court's permission, can inspect the statements that have been filed (s.47). In contrast, attorneys do not have to consult donors or others, and their actions are not subject to automatic scrutiny.

Table 2. Limitations of powers of welfare guardians and attorneys

Decision	Welfare guardian	Attorney
Marry, divorce	No	No
Adoption	No	No
Consent to standard treatment?	Yes	Yes
Consent to ECT?	No	No
Psychosurgery?	No	No
Experiments?	No	No
Refuse lifesaving treatment?	No	No

ECT=electroconvulsive therapy.

Welfare guardians must consult the person for whom they act, and other interested persons (such as family members), as far as is practicable. They must encourage the person to make their own decisions as far as possible, and must also consult with property managers. They cannot make decisions concerning entering into marriage, dissolution of marriage, or adoption of children of the person. They cannot refuse consent to standard lifesaving medical treatment, or treatment that prevents serious damage to the person's health. They cannot consent to electroconvulsive therapy (ECT), psychosurgery, or (in most circumstances) participation in medical experimentation (s.18).

In addition, welfare guardians cannot refuse treatment intended to be lifesaving. This may (in some circumstances) apply to cardiopulmonary resuscitation (CPR); however, many clinicians would view CPR as a futile treatment in elderly patients with serious and multiple medical problems, including cancers, and would advise against attempts at resuscitation. Welfare guardians must promote and protect the welfare and serve the best interests of the person. Their appointment is not indefinite (s.12), as an attorney's is.

Attorneys have the same limitations on their powers as a welfare guardian, but they are not specifically instructed to promote the welfare of the donor, or to consult with the donor, or any other person such as doctors, social workers, or other family members. If they do not act in the donor's best interests, their powers may be revoked by the Family Court (s.105), but someone has to draw the matter to the attention of the court. The court may, if asked by the donor, review an attorney's decisions (s.103).

Any other person (not further defined in the Act) can apply to the court for review of an attorney's decision. Such a person must satisfy the court that there is evidence that the attorney has made the decision while the donor is incapacitated; and if so, the court has discretion to grant permission for a review, depending on a number of

factors including the relationship between the applicant, the attorney and the donor, and the merits of the claim.[2]

In reality, attorneys have wide powers to act (without the obligation to consult others, or account for their actions—unless an order is issued by the Family Court requiring them to do so). This means that if an attorney misuses his or her powers when the donor is incapacitated, others must approach the Court on the donor's behalf. They may not do so because of ignorance of donors' and attorneys' rights and responsibilities, reluctance to interfere in what may be seen as a family matter, or fear of the cost.

Misuse of EPAs

In the UK, when donors become incompetent to manage their own affairs, EPAs must be registered with the Public Guardianship Office (PGO).[3] In addition, certain categories of relations must be informed—including the spouse, parents, children (including adopted children), full and half brothers and sisters of the donor (and their children), and grandchildren of the donor. The PGO does not monitor the way an attorney acts, but it considers complaints and can decide whether the attorney should be removed, or if other arrangements should be made for the donor.

At present, EPAs in the UK deal with property only. It is estimated that about 10,000 new EPAs are registered each year and that 10–15% of EPAs are operated improperly or fraudulently despite the registration requirements.[4]

There is no way of knowing how many EPAs are granted in any year in New Zealand, or how many are misused. In 2001, the Law Commission[1] received evidence of misuse of powers of attorney, including a small study of 130 reported cases of elder abuse in the Auckland area over a 2-year period, of which 40 were attributable to misuse of an EPA.

Misuse of EPAs fell into two broad categories:

(1) Financial impropriety such as embezzlement of donors' funds, sale of donors' property for their own benefit, and theft of donors' possessions.

(2) Failure to provide appropriate care, such as failing to arrange admission to a nursing home in order to save money; and prematurely arranging institutionalisation when the donor could have continued to live in the community.[1]

It is impossible to know how widespread these practices are, but family doctors, geriatricians, and psychiatrists may know of instances where attorneys have not acted in the donor's best interests—for example by refusing assessment or medical treatment for the donor; or refusing services such as Meals on Wheels, Home Help, and various personal carers for the donor; or by withholding money for care, or spending it inappropriately.[1]

The Law Commission identified several problems regarding EPAs:

- Lack of monitoring of the donor's capacity when the EPA is signed, and at the point when the EPA comes into force.
- No requirement for independent legal advice on the implications of EPAs, or on the rights of the competent donor to revoke EPAs.

- No requirement to file accounts and no independent monitoring of the acts of the attorney.
- The powers of the Family Court are largely ineffectual because they are reliant on someone else to take action on the donor's behalf if he or she has become incapacitated.
- Donors are reluctant to start court proceedings against family members.[1]

The Commission advised that (in some circumstances) there should be proof that the donor had received independent legal advice and that the way in which attorneys dealt with property should be monitored. Older donors and those in various institutions should be advised of their rights by a solicitor, and a certificate issued to this effect, and there should be certification by a medical practitioner when the donor becomes incompetent. The Commission also advised the appointment of a Commissioner for the Aged to act on behalf of the elderly.[1] None of these recommendations have yet become law. The Commission rejected the idea of registering EPAs on the grounds of both the cost of maintaining such a register, and for reasons of privacy.[1]

Doctors and EPAs

Both hospital doctors and family doctors may have to deal with attorneys acting on behalf of incompetent donors.

Problems may arise due to:

Issues of validity:

- The donor may have signed an EPA when already incompetent, due to advanced dementia, for example. Discussions of the levels of competence required are outside the scope of this[5,6] and will be the subject of a future paper.
- The attorney may only be authorised to act in specific circumstances and may be exceeding his or her powers by consenting to medical treatment, or refusing it. An attorney with power only to act in relation to property cannot make personal care and welfare decisions, but may be responsible for paying doctors' bills.
- The attorney may be suffering from a physical or mental illness, which may mean that he or she lacks insight into the donor's true conditions and needs, and which precludes his making decisions on behalf of others. An attorney with a problem with substance abuse or gambling addiction may be using the donor's funds inappropriately, and may inappropriately refuse admission to a nursing home in an attempt to retain control of finances.
- The attorney may falsely claim that the donor is incompetent and seek to act on their behalf inappropriately.

Doctors, especially family doctors, with relationships with both donor and attorney, may be aware of these circumstances, or other family members may confide their fears about the circumstances of an elderly relative during the course of a consultation. The donor may confide in the family doctor.

Doctors should be aware of the limits of decision making powers by attorneys in relation to patient welfare, especially if the attorney is proposing to act in a way that is not in the patient's best interests.

It is not sufficient to disapprove of the donor's choice of attorney: the courts uphold the autonomy of competent donors and will not revoke an EPA simply because others disapprove of the attorney, provided he/she is capable of performing the duties required.[7]

It is reasonable for doctors dealing with incompetent patients to ask to see a copy of the power of attorney, and check that the attorney has the power to act on behalf of the patient, that the EPA was signed at a time when the patient was competent to sign it, and that the patient intended the attorney to act in the areas of personal care and welfare. Those dealing with the elderly are aware of vast grey areas where no power of attorney exists but they consult with the patient's spouse or children on personal care issues, reasoning that such people will have the patient's best interests at heart.

The vast majority of attorneys and family members do have the patient's best interests at heart, but a few do not.

Failure to act in the donor's best interests:

- The power of attorney may be a valid one, but the attorney makes decisions or takes actions which are not in the donor's best interests such as failing to seek prompt medical advice when the donor is ill, or refusing to allow the donor to be admitted to a nursing home, or failing to provide appropriate care and supervision. See Table 3.

Table 3. 'Red Flags': Is the attorney acting in the donor's best interests?

1. Attorney does not ensure donor attends scheduled appointments
2. Attorney does not ensure donor fills repeat scripts
3. Recurrent attendances at ED/ clinic with falls/burns
4. Refuses admission to home carers/ District Nurse/Meals on Wheels
5. Refuses hospital or rest home admission

Family doctors may be aware that something is wrong if attorneys do not ensure that donors keep scheduled appointments, or an elderly donor has many emergency room attendances for injuries. A series of attendances for falls or burns may give rise to suspicion that all is not well, either because the patient is not receiving sufficient care and supervision, or that the carer/ attorney is deliberately assaulting the patient. When challenged, attorneys may refuse help and try to intimidate doctors and nurses with statements like 'I've got power of attorney and I know what Dad wants/needs.'

Neighbours, friends, and relatives may become concerned about the care given to elderly people by those with EPAs—and may turn to medical staff, social workers, and charities such as Age Concern, for help.

Medical staff may be reluctant to intervene because of worries about breaching confidentiality if they disclose their suspicions. Medical staff, however, have a duty of care to their patients, and in some circumstances the duty of care overrides

considerations of privacy and confidentiality, particularly in situations where the patient is vulnerable and cannot act for him- or herself.

If a doctor thinks that a patient is in serious and imminent danger,[8] they can disclose their concerns to appropriate people. This may take the form of contact with hospital geriatricians, social workers, or (in extreme circumstances) the police. A GP, after receiving allegations from neighbours and relatives of an elderly person, may visit the patient's home, and find that their circumstances are less than ideal. It may be possible to ask for admission to hospital, or assessment in the community. On a few occasions, it may be clear that the attorney is misusing his or her powers—perhaps making attempts to block hospital admission or access to services. Doctors may then have to turn to the courts for help.

If it is not possible to obtain help from the local hospital, phone or write to the Family Court detailing your concerns and your relationship with the patient. Charities such as Age Concern, or a local Community Law Centre, may be able to help with legal advice. Appropriate forms for the appointment of a welfare guardian or property manager are available from the Family Court, or from their website.[9] The Medical Protection Society (MPS) will be able to advise on issues of disclosure, and it is a good idea to keep them informed of steps you plan to take.

Attorney's powers can be overridden by personal orders from the Family Court (s.100). The Court, a competent donor, or a welfare guardian, can revoke a power of attorney. If the person is incompetent, a welfare guardian (s.10) should, if possible, be appointed in the place of the attorney. Usually the Family Court will appoint counsel to enquire into the circumstances surrounding an application, and under normal circumstances this can take time. In an emergency, such as the need for hospital admission, interim orders can be made, with arrangements for review after a certain period of time.

Appointment of a welfare guardian after a patient has become incompetent can be a time-consuming process. The appointment of an attorney while the donor is still competent to do so is a quick and cheap option for people who want the reassurance that relatives or friends will make decisions for them when the time comes. The EPA, properly used, should act as a shelter for the elderly from the vicissitudes of life, not as a weapon in the hands of the unscrupulous attorney. Doctors, and others caring for incapacitated patients, should be aware of the possibility of misuse, and be prepared to take action on behalf of those who cannot act for themselves. Familiarity with some of the issues involved should ensure they are not deflected from their roles as advocates for their patients by the existence of an enduring power of attorney.

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Erysipelothrix rhusopathiae causing infective endocarditis in a female patient requiring valve replacement

Biju Paul, Wazir Baig

We present a female with infective endocarditis due to *Erysipelothrix rhusopathiae*. Extensive aortic valve damage was present so she was treated surgically by aortic valve replacement. We present the case report and literature review.

E. rhusopathiae is a Gram-positive, non-spore forming catalase-negative bacillus. It affects swine, turkey, ducks, and sheep and is communicable from animals to humans. It causes an occupational disease (Rosenbach's disease in its mild cutaneous form or Klauder's syndrome in its severe systemic form), and is predominantly seen in men.[1] Treatment before 1945 with hyperimmune serum resulted in 100% mortality.[2]

Case report

A 63-year-old female was admitted in December, to the Doncaster Royal Infirmary, UK due to worsening shortness of breath. She was an ex-smoker, abstained from alcohol, and avoided animal contact due to eczema. On examination, she was afebrile, pulse 80/min, respiratory rate 16/min, and blood pressure 117/51 mmHg. Bilateral basal crepitations were present on auscultation of the lung fields. Chest X-ray showed an increased cardiothoracic ratio and prominent vascular markings. Chronic heart failure was diagnosed and anti-failure therapy including diuretics and ACE inhibitors was initiated.

In the ward, she developed a low-grade fever and early diastolic murmur in the aortic area. An echocardiogram revealed severe aortic regurgitation and a large vegetation on the aortic valve. Biventricular systolic function was good. Blood culture grew a Gram-positive bacillus presumed to be *Streptococcus*, and was highly sensitive to penicillin. A diagnosis of infective endocarditis was made, and treatment initiated with penicillin and gentamycin.

Her breathlessness responded to treatment. However, she continued to have a wide pulse pressure and low-grade fever. An oesophageal echocardiogram done 2 weeks later revealed large bulky vegetation on the aortic valve, left ventricular dilatation, and global systolic impairment. She was transferred to the cardiothoracic centre for an aortic valve replacement.

Pathological examination of the aortic valve showed signs of active inflammation. The organism grown from blood culture was sent to the reference laboratory in London and identified as *E. rhusopathiae* and confirmed by examination of 16srRNA.

Discussion

E. rhusopathiae was first isolated in 1880 by Koch and demonstrated to be a pathogen in humans by Rosenbach in 1909. It is a slightly curved, pleomorphic, catalase-negative bacillus.[1]

E. rhusopathiae causes Rosenbach's disease/Klauder's syndrome—an occupational disease affecting butchers, fishermen, farmers, and veterinarians. Alcohol abuse is the most commonly encountered underlying medical condition.[3] It presents during the summer and autumn months', with an incubation period of 1 to 4 days.

It is differentiated from bacillus species by the absence of spores. However, it is commonly misidentified as *Streptococcus viridans* and is often dismissed as a contamination.

The clinical presentation ranges from a mild cutaneous form (erysipeloid) to severe septicaemia. The septic form is usually associated with subacute endocarditis.[3] It causes extensive damage of the native valves with a predilection for the aortic valve. Despite appropriate therapy, the mortality rate for disease caused by *E. rhusopathiae* is 38%.

The organism is extremely sensitive to penicillin, and highly susceptible to cephalosporins, erythromycin and clindamycin[1]—but it is resistant to vancomycin. The recommended therapy for *E. rhusopathiae* endocarditis is 12–20 million units/day of intravenous penicillin in divided doses over 4 to 6 weeks.[5] Penicillin-sensitive patients can be treated with erythromycin or clindamycin.

Valve replacement is required in 36% of cases and relapses can occur. It should be considered in the presence of progressive congestive failure, recurrent emboli, persistent bacteraemia (despite therapy), development of heart block, and presence of large vegetations. Prevention of infection for those in high-risk occupations can be achieved by the use of preventive gear such as gloves. A live attenuated vaccine is available for veterinary use.[6]

Our case is unique because the infection is rarely seen in females or in the winter months. Furthermore, extensive valve destruction requiring surgery is not common and the patient was not an alcoholic and did not have extensive exposure to the organism.

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Recognising and responding to partner abuse: challenging the key facts

Felicity Goodyear-Smith

Abstract

Inter-partner violence is a serious public health problem for a minority of the population. Frequently the problem is hidden and goes undetected. Recognising this violence within the primary healthcare setting and responding appropriately are laudable aims, with significant health gains. However it is important that in raising professional and public awareness of the issue, the case is not over-stated. Too often figures such as a NZ$141 million annual cost of family violence to health are quoted and presented as 'fact' without critical appraisal. Family violence is an emotional topic, and challenge to prevailing viewpoints may be misconstrued as a denial of the problem. These are important issues, and I invite academic debate.

The Ministry of Health has recently released its publication titled *Recognising and responding to partner abuse: a resource for general practice*.[1] This document bears endorsements from the Royal New Zealand College of General Practitioners; Breaking the Cycle of Violence; the New Zealand Medical Association; the New Zealand College of Practice Nurses; Doctors for Sexual Abuse Care; and Accident Compensation Corporation (ACC).

The document follows a 2002 Ministry of Health best practice guideline recommending the annual screening of all female general practice patients sixteen years and over for physical and sexual abuse by their partners.[2] However routine screening for partner abuse currently does not meet recognised screening criteria.[3–5]

The 2002 partner abuse guideline primarily addresses male abuse of female partners. The authors of the current publication are to be commended for recognising that both women and men can be victims of partner abuse. Much of the document is gender neutral, although the advocated 'power/control' model, and recommended actions such as providing a safety plan, address only female victims of male perpetrators.

The need for appropriate detection and intervention in partner abuse is not questioned. However 'partner abuse' has a broad definition and a strong case can be made for GP diagnosis and treatment to focus on the serious end of the spectrum involving significant injury impacting on health.

The Ministry of Health publication in question[1] lists five 'key facts' and promotes the 'power/control' model of family violence. These items raise serious concerns. They are either not evidence-based or are a skewed representation of research findings. The statements suggest an inflated prevalence of partner abuse, and minimise potential objections by patients to routine questioning. This distortion of the scientific evidence reduces the credibility of the publication's recommendations and is a disservice to those requiring active intervention for serious abuse.

The 'key facts' are listed below followed by a critical analysis of their content.

Both women and men experience abuse, however the prevalence is higher for women (Langley et al 1997; Young et al 1997)

The use of the word 'abuse' in this statement is problematic. The publication defines partner abuse as 'the physical, sexual, verbal, and emotional/psychological abuse of current or past intimate partners'. Psychological abuse includes, but is not limited to, 'intimidation harassment, damage to property, threats of physical abuse, sexual abuse, or psychological abuse' and allowing a child to 'see or hear the physical, sexual, or psychological abuse of a person with whom the child has a domestic relationship'.

The claim of a higher prevalence in women can be made for 'physical assault' and for 'sexual abuse', but not for physical, verbal, emotional, or psychological abuse. The relative frequencies of men and women verbally or emotionally/psychologically abusing their partners, or exposing a child to such abuse are unknown.

Evidence indicates that men and women physically abuse their partners in similar percentages.[6] The Dunedin Multidisciplinary Health and Development longitudinal cohort study found that (within partnerships) women actually used more physical violence than men[7]—women reported committing more partner violence than men, and men reported more victimisation than women.[8]

However, far fewer defined this violence as 'assault' causing physical harm, and in those who did, more men than women were named as perpetrators.[9,10] Indeed, these findings suggest that about four men assault women for every one woman who assaults a man.

Partner homicide is a relatively rare event in New Zealand, with more women than men killed by their partners. A study of New Zealand homicides from 1978 to 1987 found that 82 men and 9 women killed their partners during that decade.[11] Another New Zealand study of intentional murders between heterosexual intimates between 1988 to 1995 found 80 male and 22 female offenders.[12] These figures indicate that an average of 11 women and 3 men were murdered by their partners each year.

The majority of women do not object to routine questions about abuse (Ramsay 2002)

While this statement is technically true, over 50% of women have been found to object in some studies. In their systematic review,[5] Ramsay et al report one study where only 43% of women favoured routine inquiry.[13] Their review concludes that 'about one-half to three-quarters of women patients in primary care' found domestic violence screening acceptable. This suggests that between 25–50% of women are not comfortable with screening. GPs will be reluctant to screen women patients if 3 or 4 out of every 10 object to being asked.

Over a lifetime, 15–35% of women experience abuse (Young et al 1997)

It is hard to see how these figures are derived from the reference given. The Young et al report on the New Zealand National Survey of Crime Victims 1996 presents the findings of a random sample of 5000 people aged 15 and over.[14]

The lifetime prevalence of ever experiencing at least one act of physical or sexual abuse from a partner is reported as 15.3% for women and 7.3% for men. The types of violence include use of force (deliberately kicked, pushed, grabbed, shoved you or hit with something in a way that could hurt you); threats (threatened to kick, push, grab, shove you in a way that actually frightened you); deliberately destroyed or threatened to destroy your belongings in a way that frightened you; made you carry out any sexual activity you did not want to (by holding you down, hurting you or threatening you) or used a weapon (such as a knife or gun).

The prevalence rate for Maori (26.9% of women and 11.9% of men) was much higher than for New Zealand European (14.6% women and 6.8% of men). This may not be a racial and cultural difference, but a reflection of the greater representation of Maori in the lower socioeconomic bracket.

What this report highlights most is the extremely uneven distribution of violent victimisation. Only a very small percentage of the population are victims of significant recurrent violence. The vast majority of people have little exposure to violence or threats, but for a small percentage of the population violent events are nearly commonplace. ‘Only 0.5% of the sample (or 6% of those who had been victimised) had been victims of a violent offence 5 or more times, but they accounted for a massive 68% of such offending. Among such victims, the average number of violent and sexual offences was 12.’ The report recommends focusing prevention efforts on those small pockets of the population who are particularly at risk of multiple victimisation.

The co-occurrence of partner abuse with child abuse is 30–60% (Ross 1996; Edelson 1999)

The Ross study referenced involved telephone interviews with 6000 American couples in 1985. Of those husbands who were physically violent towards their wives, 22.8% had engaged in physical child abuse.[15] The percentage of violent wives who had engaged in at least one act of physical child abuse was 23.9%. The relationship between marital violence and child abuse had an odds ratio of 1.12 for violent husbands and 1.04 for violent wives. Male children had a higher predicted probability of physical child abuse than female children from both fathers and mothers.

The Edleson reference[16] is a review paper identifying 35 studies that mention an overlap between child maltreatment and adult domestic violence. The search strategy is not outlined and this is not a formal systematic review. In the identified studies, ‘the co-occurrence of child maltreatment and adult domestic violence ranged from a low of 6.5% overlap to a high of 97%’. Twelve, or almost half the studies, found the overlap to be in the range of 30% to 60% of families with children’. These were generally populations of either battered women or maltreated children, not epidemiological samples. The sampling approaches of these studies were extremely

diverse and ‘probably account for much of the variation in results between studies, making it difficult to draw comparative conclusions’.

In 1994, the annual cost to health was estimated at NZ$141 million (Snively 1994; Young et al 1997)

A critical analysis of Snively’s study indicate severe methodological flaws, including faulty assumptions about prevalence and over-estimates of many of the parameters used in her calculations.[4]

NZ$141 million is based on an assumed prevalence rate of 14%—that every year 1 in 7 women and 1 in 7 children are victims of family violence requiring medical intervention. The costing assumes that annually, each of these women and children (301,700 people) require two visits to a GP; that half of them also require an accident and emergency consultation; that 12.5% (37,711 people) sustain dental injuries requiring an average of $200 dental treatment, and that 5.5% (16,895 people) require hospital admission at an average cost of $17,000 per admission.

These numbers represent huge over-estimates. For example, in the year 2002/2003 there was a total of 6604 ACC dental claims for people suffering dental injuries from being ‘struck by a person or animal’, of whom just under one-third (2,175) were female. Adjusting for population figures in 1994, this equates to about 6200 cases, of whom 2050 would be female.

Some of these cases would be girls not adult women, and due to causes other than domestic violence (for example, injuries from contact during sport; kicks from horses or other animals; assaults from people other than their partner), therefore less than 2000 women are likely to have suffered dental injury from partner assault in 1994. This figure (2000) is much less than the Snively estimate of 37,711 cases of dental treatment resulting from family violence in that year.[17]

Furthermore, it should be noted that the figure of NZ$141 million was an estimate of combined child and partner abuse, but the Ministry of Health document implies that this is the cost to health of partner abuse alone, effectively doubling the estimated cost of partner abuse.

Power and control wheel

The Ministry of Health book also promotes the use of the ‘power and control wheel’ devised by the Duluth Domestic Abuse Intervention Project, Minnesota, USA. This tool is not evidence-based. It addresses only male violence towards females. The Duluth ‘wheel’ is based on the assumption that family violence results from power imbalance between men and women, and that men abuse because they hold the power in our society. However this is not a universally accepted model, and alternatively it can be argued that use of violence is not the act of a powerful man, but rather of one who finds himself relatively powerless.[6]

The desire to do good (reduce and prevent inter-partner violence) does not justify exaggeration or distortion of the evidence to further the cause. The Ministry of Health should disseminate accurate and credible information based on critical appraisal of the research literature.

A particular intervention might seem to be a good idea but if it is based on faulty assumptions, it may be neither effective nor safe. The management of domestic violence requires similar rigorous scientific evaluation as do other areas of clinical intervention.

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Labour obstructed by hydrocephalus

This case report by Dr Howard Slater was published in the New Zealand Medical Journal 1905, Volume 4 (14), p103–4.

Mrs. L. sent for me at midnight, April 11–12, for her fourth confinement. The previous labours had been normal. Labour had begun at 6 p.m. I found the breech presenting in the dorso-anterior position. As the os was fully dilated I ruptured the membranes, when a very considerable quantity of amniotic fluid escaped. The birth of the body soon followed, and with a little manipulation the arms came down. But the head remained at the brim of the pelvis. The cord pulsated for some time, and, the pulsations becoming feebler and feebler, I made traction on the body for a good half-hour, but without further effect than damage to the child. Examining the maternal abdomen I found the uterus as large as at the seventh month of pregnancy, the upper part soft and the lower hard, the latter giving a crackling sensation. Per vaginam the finger could be passed freely as far as the eyes, also up to the occipital region. With one hand on the hard tumour of the uterus and the finger of the other hand on the occipital region, fluctuation between them was found.

The case therefore was apparently one of hydrocephalus; and my colleague, Dr. Claridge, whom I now ca1led in, and for whose kind assistance I am greatly indebted, confirmed these observations and this diagnosis.

The received method of treatment is to use the perforator against which, however, many objections may be urged. It is a clumsy instrument to use on an after-coming head, so I put it aside.

I then, recollecting Dr. Ballantyne's paper in the *British Medical Journal* of the 10th December last, attempted to pass a catheter up the spinal canal. But in making the preliminary incision I had the misfortune to turn the edge of my knife; so this method had to be abandoned.

I then passed a large ascites trochar and cannula to the occipital bone, plunged it forwards towards the centre of the head, and was at once rewarded by a copious flow of fluid. When it ceased, gentle traction brought away the child. It was now 2 a.m.

The head presented a peculiar appearance, due to the overlapping of the parietal bones by the frontal bone, carrying the scalp with it. The wound in the occipital was, of course, small, and presented none of the sharp edges that would have been produced by the perforator.

Recovery was uneventful.

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Domoic acid-induced cardiotoxicity: a mitochondrial approach. J-H Baek, A Clarkson, A Tramoundanas, B Hesp, IA Sammut, DS Kerr. Department of Pharmacology and Toxicology, OSMS, University of Otago, Dunedin.

Domoic acid (DOM) is a potent excitotoxin, structurally related to the excitatory neurotransmitter, glutamate (Glu). DOM has been shown to cause extensive damage in the central nervous system (CNS), however little is known about its effects within the myocardium.

In vitro, DOM has been shown to affect cardiac mitochondrial respiratory enzymes within the electron transport chain and alter mitochondrial-coupled respiration. Mitochondria have been implicated in excitotoxic damage, and alterations in mitochondria are a fundamental feature of aging. Several studies have shown differences in DOM's neurotoxic effects in young and aged rats. An age-linked decline in the activities of mitochondrial enzymes within brain slices following Glu treatment (1mM) was observed. However, the effect of aging in relation to DOM exposure has not been assessed in cardiac mitochondria.

In this preliminary study, hearts from young (3 months) and aged (27 months) rats, treated with *in vivo* DOM (Young: 0.5mg/kg (n=4), 1.0mg/kg (n=2), 2.0mg/kg (n=2); Aged: 0.5mg/kg (n=2), 1.0mg/kg (n=2), 2.0mg/kg (n=2)) were isolated and freeze clamped. Mitochondrial complex enzyme activities were assessed in the prepared cardiac homogenates to assess mitochondrial impairment. Activities of aged Complex I, II/III, IV, V enzymes and citrate synthase were all decreased by DOM in a concentration-dependent fashion, although there was no significance due to small sample size. Similar results were observed in young, however a decrease was not observed for complex V (ATP synthase) activity.

The aged heart has a lower tolerance than the young heart to oxidative stress, due to its decreased anti-oxidant properties within cardiac mitochondria. Following Glu treatment (1mM), aged rats have been shown to have significantly higher formation of reactive oxygen species (ROS). Mitochondrial complex enzymes have been shown to be targets of free radicals and ROS, and this may explain why decreases in complex enzyme activities were observed in this current study.

This study was supported by a Grant from Otago Medical Research Foundation.

Effect of lactation on prolactin signalling by STAT5b in gonadotrophin releasing hormone (GnRH) and tuberoinfundibular dopaminergic neurons. AS Bang, DR Grattan, GM Anderson. Centre for Neuroendocrinology and Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

The hormone prolactin plays important roles in mammary gland development and initiating and maintaining lactation, and therefore is found in high levels during late

pregnancy and lactation. This hyperprolactinemic state is maintained by a reduction in the ability of prolactin to activate inhibitory hypothalamic tuberoinfundibular dopaminergic (TIDA) neurons, and by a stimulatory neuroendocrine reflex evoked by suckling of the young. One of the many other effects of hyperprolactinemia is infertility; however how prolactin acts in the brain to achieve this is not understood. Activation of prolactin receptors in TIDA neurons can be detected by staining for its cytoplasmic transcription factor, STAT5b. The aims of this project were to determine: (1) if prolactin signals via STAT5b in GnRH neurons, which govern reproduction, and (2) whether lactation alters the sensitivity of GnRH and TIDA neurons to prolactin.

Three groups of female rats (n = 9-10 per group) were used: diestrous (normally cycling), lactating with pups removed for 4 h to acutely reduce endogenous prolactin levels, and lactating with pups removed for 24 h. These were each divided into two subgroups, one receiving a single prolactin injection (250 µg) and the other vehicle 45 min before brain collection. Sections containing the arcuate nucleus and preoptic areas were stained by immunohistochemistry to identify prolactin-induced translocation of STAT5b into the nucleus of TIDA or GnRH neurons, respectively.

In diestrous rats, treatment with prolactin induced nuclear STAT5b translocation in TIDA neurons. However these neurons were insensitive to prolactin during lactation. Prolactin did not induce STAT5b signalling in GnRH neurons in either diestrous or lactating rats. These results demonstrate that (1) prolactin does not act directly on GnRH neurons, at least via the STAT5b signalling pathway, and (2) during lactation prolactin signalling is suppressed in TIDA neurons so that hyperprolactinaemia can be maintained.

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A new approach to the delivery of RNA interference to human cells. CY Chan, D Markie. Department of Pathology, Dunedin School of Medicine, University of Otago, Dunedin.

RNA interference (RNAi) is the specific inhibition of gene expression by double-stranded RNAs. One established approach is the *in vivo* expression of short hairpin RNA (shRNA) molecules from plasmid vectors in mammalian cells to induce loss-of-function in various biological systems. The present study describes the development of a novel human RNAi system using the Polymerase Chain Reaction (PCR) that can be controlled as required, making it more efficient than previous RNAi methods.

A template suitable for generating DNA fragments containing both a selectable marker and the shRNA required to knockdown specific genes was constructed. By PCR, we obtained DNA fragments with shRNA targeted against the human *BUB3* gene from this template. We then transfected these fragments into TREX-293 human cells, where they were successfully integrated into the cell chromosomes to create stable cell lines. This result demonstrates that PCR may be useful in generating fragments for this purpose.

In our attempt to knockdown the human *BUB3* gene, the TREX-293 cells still exhibited normal levels of *BUB3* protein, indicating that knockdown was initially

unsuccessful. This may be due to either a failure in the chosen RNA sequence to function effectively as an RNAi template, or a failure of shRNA expression from the fragment, and further experiments using alternative shRNA sequences will be required to answer this question.

However, despite the lack of success with BUB3, we did develop a method that allows us to produce DNA fragments for RNAi by PCR with the potential for carrying out rapid functional analyses of genes. This, therefore, is a first step in the development of assays which can then be used to evaluate novel genes with unknown functions.

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Defining neuropeptide Y interactions with gonadotrophin-releasing hormone neurons in mice. E Cottrell, R Campbell, A Herbison. Department of Physiology, Centre for Neuroendocrinology, Otago School of Medical Sciences, University of Otago, Dunedin.

Reproductive function is governed by a population of cells within the brain, the gonadotrophin-releasing hormone (GnRH) neurons. These cells receive a multitude of signals reflective of the physiological state of the individual. The study of the regulation of networks governing these neurons is of importance in understanding the regulation of fertility, and how this is restricted to appropriate circumstances. Neuropeptide Y (NPY) is one molecule identified as playing a role in regulation of GnRH neuron activity, and proposed as a potential signal in the integration of energy balance and reproductive function. We have been investigating the potential role of the NPY Y1 receptor (Y1R) subtype in the regulation of mouse GnRH neurons, as recent studies have found that the Y1R colocalised with GnRH nerve terminals in the rat.

Mice used in these studies were anaesthetised with sodium pentobarbital and killed by transcardial perfusion with 4% paraformaldehyde fixative solution. Brains were then rapidly dissected out, post-fixed and processed for immunohistochemical (IHC) study. Antibodies used were rabbit anti-NPY Y1R (directed against either the C- or N-terminal regions), sheep anti-GnRH and rabbit anti-galanin. Single-label IHC with Y1R antibodies was done firstly in male animals, to optimise antibody conditions and define Y1R distribution. Following this, double-label IHC with confocal microscopic imaging was employed in female mice to investigate Y1R expression on GnRH neurons. Galanin, a peptide molecule expressed in GnRH cells, was used as a positive control for colocalisation. Of a total of 208 GnRH neurons from five female animals analysed using confocal microscopy for GnRH/Y1R colocalisation, none were found to show convincing Y1R expression. This absence of coexpression was substantiated using a GnRH/galanin control.

Given this negative result, we propose a scenario where NPY may exert indirect effects on the GnRH neuronal system to regulate reproductive function.

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Localisation of prolactin receptor mRNA in identified magnocellular neurons in the female rat brain using dual-label *in situ* hybridisation histochemistry.
R Davis, I Kokay, D Grattan, Centre for Neuroendocrinology and Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

We have previously identified the receptors that mediate the action of the hormone prolactin in several areas of the hypothalamus including the supraoptic and paraventricular nuclei. The aim of this study was to identify the neurochemical phenotype of the magnocellular neurons that express prolactin receptor mRNA in these two nuclei. Furthermore, as the supraoptic and paraventricular nuclei both undergo significant plasticity during lactation, we examined whether prolactin receptor expression changed during this time.

Dual-label *in situ* hybridisation histochemistry was performed on 3-4 sections per brain structure from lactating (n=3-5) and non-pregnant (n=3-5) female rats. Sections were hybridised with a ^{35}S labelled nucleic acid probe that specifically detected the long form of the prolactin receptor together with non-radioactive (digoxygenin-labelled) RNA probes to detect either oxytocin or vasopressin mRNA. Following visualisation of digoxygenin-labelled probes by immunohistochemistry, sections were coated with photographic emulsion and stored at 4°C for 4 weeks before being developed to detect prolactin receptor mRNA. Images were analysed using NIH image software.

In non-pregnant rats, 87 ± 5% (mean ± S.E.M.) of the oxytocin magnocellular neurons in the supraoptic nucleus and 51 ± 8% of neurons in the paraventricular nucleus expressed prolactin receptors. The proportions of neurons showing co-localisation did not change significantly in the lactating group. In contrast, prolactin receptor mRNA was present in less than 25% of vasopressin neurons in both hypothalamic nuclei of non-pregnant animals (24 ± 10% in the paraventricular, and 14 ± 4% in the supraoptic, nucleus) and lactating animals (16 ± 2% in the paraventricular, and 20 ± 5% in the supraoptic, nucleus). The detection of prolactin receptors on oxytocin and to a lesser extent on vasopressin neurons implies prolactin can directly modulate the activity of magnocellular neurons and supports data that suggests prolactin may have important brain actions in addition to its role in the establishment and maintenance of lactation.

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The effects of anti-psychotic drug-induced hyperprolactinaemia on reproductive neuroendocrine function in female rats. DC Kieser, DR Grattan, GM Anderson. Centre for Neuroendocrinology and Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

It is known that hyperprolactinaemia, a common side-effect of many antipsychotic drugs, causes infertility and loss of libido in humans and animals. The underlying mechanisms of this effect are largely unknown. Insight into these mechanisms could lead to better therapies for pathological and antipsychotic drug-induced infertility,

production of improved antipsychotics that avoid this side-effect, or conversely, the generation of new non-steroidal methods for suppressing fertility in both males and females. We investigated whether chronic anti-psychotic drug-induced hyperprolactinaemia inhibited three neuroendocrine parameters necessary for female fertility: the surge of gonadotrophin releasing hormone (GnRH) and luteinizing hormone (LH) that induces ovulation, tonic pulsatile secretion of LH, and the negative feedback of oestradiol on LH pulses.

Ovariectomised rats (n = 5-6) received sulpiride (1.25 mg sc) or vehicle twice-daily for 8-10 days, resulting in marked hyperprolactinaemia. When also treated with oestradiol to mimic the presence of ovarian oestrogens, the frequency of LH pulses was suppressed in hyperprolactinaemic rats ($p < 0.05$). This did not occur in the absence of oestradiol. There was no effect of sulpiride on LH pulse amplitude under either steroidal condition. When rats were acutely treated with doses of oestradiol and progesterone known to induce a preovulatory-like GnRH/LH surge, the peak plasma concentration of LH and the activation of GnRH neurons (as determined by immunocytochemical detection of the neural activity marker Fos in GnRH neurons) were not significantly different between sulpiride- and vehicle-treated rats.

We conclude that the inhibitory effect of hyperprolactinaemia on LH pulse frequency requires the presence of ovarian steroids, and that hyperprolactinaemia does not markedly inhibit the preovulatory surge of GnRH and LH.

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Herbal products: a follow-up survey of opinions, perceptions and behaviours of callers to the New Zealand National Poisons Centre. J Lee, N Smith. Department of Pharmacology & Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.

Health professionals have expressed concerns about consumer misconceptions that herbal products (HP) are “natural, safe and non-toxic”. This study investigated the attitudes, opinions and behaviours towards HPs via a follow-up telephone survey of the 98 general public callers who contacted the National Poison Centre (NPC) regarding HPs, between July 2002 and November 2003. The 60 respondents were 95% female, 73% aged 21-40 years, 32% university educated and 93% of New Zealand European descent.

Two-thirds of respondents recalled no additional advice provided when purchasing HPs, consistent with almost half obtaining HP from sources where professional advice was not available (e.g. supermarket). HPs were used primarily in disease prevention (66.7%). Most respondents (58.3%) did not believe that HPs were more efficacious than conventional medicine (CM), but favoured HPs for their perceived safety. Just over half (55%) also believed that combining HPs with CM result in increased efficacy, compared to when using either independently. Only 15% of all reported products had child safety packaging. Only 43% of HPs were stored with CM but few (13%) of HPs were actually locked away.

Most respondents were willing to tell health professionals about their HP use and adverse reactions. NPC advice was considered by respondents as very useful (70%),

sufficient in quantity (63%) and very clear (62%). Almost all (98%) were satisfied with the NPC service and would recommend it to others.

The study findings emphasise the need for health professionals to discuss safe use and storage of HPs with patients, and caution them against over-estimating HP safety, to minimise problems associated with their use. Child safety packaging for HPs also needs greater promotion. It is anticipated that as HP use becomes more popular there may be a corresponding increase in HP poisonings, and the NPC database should acquire more detailed information to meet this need.

This project was made possible through the generosity of the Dunedin School of Medicine.

Neuroprotective effect of (-)-epigallocatechin gallate in a rat model of hypoxia-ischaemia-induced brain damage. BA Sutherland, O Shaw, AN Clarkson, I Appleton. Department of Pharmacology & Toxicology, Otago School of Medical Sciences, University of Otago, Dunedin.

(-)-Epigallocatechin gallate (EGCG) is a polyphenolic antioxidant that protects cells against free radical damage. It was previously shown that 50 mg/kg EGCG is neuroprotective in a rat model of hypoxia-ischaemia (HI). This study investigated the possible mechanisms underlying the neuroprotective effects of EGCG.

The left common carotid artery was permanently double ligated in 26 day old male Wistar pups (n = 8). Two hours later, the rat was placed in an 8% O_2/92% N_2 atmosphere for 60 minutes. This produced an infarction on the ipsilateral side of the brain. There were three treatment groups: untreated, with no HI (control); HI + 0.9% saline; and HI + 50 mg/kg EGCG. Treatments were administered i.p. daily beginning one day prior to HI for 4 days. 29 day old rats were euthanised in accordance to ethical guidelines.

Western blot analysis found that HI did not alter the protein levels of neuronal nitric oxide synthase (nNOS) or endothelial NOS (eNOS) significantly compared to controls. Inducible NOS (iNOS) was significantly increased after HI (0.22 ± 0.06 optical density (OD)) compared to controls (0.06 ± 0.02 OD; $P<0.05$; unpaired *t*-test) but decreased again with EGCG administration (0.12 ± 0.03 OD). HI + EGCG significantly increased nNOS (0.42 ± 0.06 OD) and eNOS levels (2.18 ± 0.6 OD) compared to HI + saline (nNOS: 0.27 ± 0.05 OD, $P<0.05$, unpaired *t*-test; eNOS: 0.68 ± 0.27 OD, $P<0.05$, unpaired *t*-test).

Previous experiments have identified that nitric oxide (NO) derived from eNOS is neuroprotective, whereas NO from iNOS and nNOS is neurotoxic. Therefore, the neuroprotective effects of EGCG may partly be due to increased eNOS levels and decreased iNOS levels suggesting that EGCG produced its neuroprotection by modulating NOS isoforms. This further substantiates that EGCG is an effective neuroprotectant in neurodegenerative disorders such as HI.

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Molecular characterisation of anginolysin A, a lytic bacteriocin produced by *Streptococcus anginosus*. Y-T Ting, M Dufour, N Heng, J Tagg. Department of Microbiology and Immunology, Otago School of Medical Sciences, University of Otago, Dunedin.

The oral bacterium *Streptococcus anginosus* produces anginolysin A, a cell wall-degrading (lytic) antibacterial protein (bacteriocin) that kills *Streptococcus pyogenes*, the causative agent of streptococcal sore throats. The objectives of this research project were: (i) to use molecular biological techniques to characterise the genetic locus for anginolysin production from two *S. anginosus* strains T-29 and H19, (ii) to compare and contrast the deduced amino acid sequence of anginolysin A with that of zoocin A, the prototype streptococcal lytic bacteriocin, and (iii) to clone the anginolysin gene into an expression vector for protein overexpression in *Escherichia coli* hosts.

Using PCR primers targeting a highly-conserved region within the catalytic domain of lytic enzymes, a PCR product (designated Pep) was obtained. The nucleotide sequence of the Pep product was subsequently used in the design of new inverse PCR primers in order to obtain the rest of the anginolysin A gene (*angA*) as well as the associated bacteriocin immunity gene (*angI*). The function of the *angA* gene was confirmed as gene knockout mutants no longer produced anti-*S. pyogenes* bacteriocin activity.

The anginolysin and zoocin protein sequences are very similar (73%) with the main amino acid differences observed in their substrate-binding (target recognition) domains, which may explain why anginolysin A kills a much narrower range of target bacterial strains. Furthermore, the gene arrangement of the anginolysin A locus was different to that of zoocin A.

Finally, the *angA* genes of strains T-29 and H19 have been cloned into the expression vector pQE-80L to facilitate protein overexpression experiments in *E. coli.* This will allow further biochemical characterisation of purified anginolysin A proteins.

In conclusion, this study has identified a new member of the lytic class of bacteriocins. The information obtained provides the foundation by which future experiments aimed at elucidating structure-function relationships of lytic bacteriocins can be designed.

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Inflammatory cytokine profile during total hip arthroplasty: a pilot study. J Yap, JC Theis. Department of Orthopaedic Surgery, Otago School of Medicine, University of Otago, Dunedin.

Cytokines are molecules in our bodies which are involved in our immune system. Total hip arthroplasty, otherwise known as hip replacement surgery is a common cause of fat embolism, which occurs when pressure in bone cavities displaces fat and bone marrow into the blood circulation. Because of the possible involvement of cytokines in fat embolism, this pilot study was designed to investigate the cytokine profile of subjects who went through total hip replacement surgery.

Blood samples were obtained from a peripheral vein in five subjects at 8 different times during and after a total hip replacement surgery within a period of 72 hours. Three subjects had a routine cemented surgical procedure, one had a partial cemented procedure and one went through an uncemented procedure. Serum levels of three inflammatory cytokines, Interleukin (IL)6, IL10 and IL1β, were measured using an ELISA. Haemodynamic parameters, i.e. blood pressure, oxygen saturation and pulse were measured at similar times when the blood was taken.

The study showed a marked increase in inflammatory cytokines. Very low levels of inflammatory cytokines were detected before and during the operation in all five of the participants. Post-operatively, there was a continuous rise in IL6 concentration, peaking at 6-12 hours followed by a steady decline towards baseline values. There also was a steady and continuous rise in concentration of the anti-inflammatory cytokine IL10, peaking at 12-48 hours after surgery. In the present study, IL1β levels did not change appreciably. The study found no significant correlation between any of haemodynamic parameters and their corresponding cytokine response.

From this pilot study, it can be concluded that total hip replacement surgery causes an increase in inflammatory cytokines. Future studies could evaluate whether a cemented procedure results in a significantly greater inflammatory response.

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Differing hypothalamic responses in pseudopregnant and pregnant rats after central leptin administration. R Augustine, S Bunn, D Grattan. Centre for Neuroendocrinology and Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

During pregnancy in rats, a state of hypothalamic leptin resistance develops that facilitates the hyperphagia characteristic of pregnancy. Pseudopregnant rats are also hyperphagic but have a normal hypothalamic response to leptin. These animals have an identical hormone profile to the first half of pregnancy, but do not form a placenta, allowing the effects of hormones of maternal origin to be distinguished from placental hormones. The aim of this experiment was to examine whether extending pseudopregnancy (PSP), to provide a more prolonged exposure to pregnancy-like maternal hormones, is able to induce a loss of leptin responsiveness in the brain, as occurs during pregnancy.

Sprague-Dawley rats were mated with intact or vasectomised males. Intracerebroventricular (icv) cannulae were implanted 1-3 days later and on day 9 pseudopregnant rats received three blank silastic implants (40 mm long) or implants containing progesterone. Blank-implanted rats resumed estrous cyclicity by approximately day 12, whereas progesterone treatment extended PSP beyond 18 days. On day 13 of pregnancy and extended PSP, rats were fasted for 24 hours and then injected icv with leptin (4 μg in 2 μl) or vehicle. Food intake was measured 24 hours later. Leptin significantly suppressed post-fasting food intake in both sham and progesterone-treated PSP rats compared with vehicle-injected rats (17.0 ± 3.01 vs 24.9 ± 1.51 g ($p < 0.001$, $n = 11$), and 24.7 ± 1.19 vs 29.2 ± 1.17 g ($p < 0.05$, $n = 16$), respectively). There was no significant difference in food intake between pregnant rats given vehicle (27.11 ± 1.82 g, $n = 8$) or leptin (24.1 ± 1.34 g, $n = 8$), demonstrating central leptin resistance in pregnant rats.

During extended PSP there is an increase in food intake, as has previously been observed during pregnancy. These animals responded to central leptin, in contrast to pregnant rats, suggesting that placental-derived hormones may contribute to the changing hypothalamic response to leptin observed during pregnancy.

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The thermal characteristics of bedsharing versus cot-sleeping infants at home. S Baddock, B Galland, M Beckers, B Taylor, D Bolton, A Phillips. Department of Women's and Children's Health, Dunedin School of Medicine, University of Otago, Dunedin.

Routine adult-infant bedsharing remains controversial as some situations increase the risk of Sudden Infant Death Syndrome. Higher rectal temperatures in bedsharing

infants may contribute to the risk. We investigated infants in the natural setting of their own home to compare thermal characteristics of bedsharing and cot-sleeping situations and the potential hazards.

Overnight video and physiological data was collected in family homes from 40 healthy, routine bedsharing infants (5-27 weeks) and 40 routine cot-sleeping infants matched for age and season of study. Physiological recordings included infant rectal, shin and room temperature. Overnight mean rectal, shin and room temperatures were calculated for each 30 minute epoch. Effective thermal insulation for the bedding and clothing of each infant was calculated by using the average measured thickness of typical New Zealand bedding and clothing of similar types. A log of infant sleep positions, the number of episodes of face covering and awakenings, total awake time, number of feeding sessions and infant movements were also recorded.

The mean rectal temperature 2 hours after sleep onset for bedshare infants was 36.79°C and for cot-sleeping infants, 36.75°C [difference 0.05°C (95% CI: -0.03 to 0.14)]. The rate of change thereafter was higher in the bedshare group than the cot group [0.04°C vs 0.03°C/h (difference 0.01, 0.00 to 0.02)]. Bedshare infants had a higher shin temperature at 2 hours [36.41 vs 35.59°C (difference 0.82, 0.15 to 1.48)] and a higher rate of change [0.04 vs -0.01°C/h (difference 0.13, 0.08 to 0.19)]. Bedsharing infants had more bedding. Face covering events were more common and bedshare infants woke and fed more frequently than cot infants (mean wake times/night: 4.6 vs 2.5).

Healthy bedshare infants at home experience warmer thermal conditions than those of cot sleeping infants but they thermoregulate adequately to maintain normal core temperature.

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Adenovirus E1a supplants the tumour suppressor protein, p53. H Campbell, J Royds, A Braithwaite. Department of Pathology, Dunedin School of Medicine, University of Otago, Dunedin.

Attempts have been made to create conditionally replicating adenoviruses (Ads) that selectively replicate in tumour cells. A number of the strategies employed have focused on the finding that > 50% of human tumours lack the p53 protein and utilise p53 deficiency as criteria for selectivity, but this approach has had limited success.

We have demonstrated that in the absence of p53, Ad infection leads to an increase in expression of p53-responsive genes. This indicates an Ad protein can mimic some of p53's roles. We have explored the effects of Ad E1a on p53-target genes in the presence and absence of the Retinoblastoma protein (Rb). E1a expression plasmids were introduced into p53 null cells along with promoter reporter constructs of various p53-target genes, e.g. BAX, and target gene activation was measured.

We found that E1a can induce p53-target genes in the presence of Rb, suggesting that E1a can mimic some of p53's functions. However no induction of these genes was observed in cells that have a mutant Rb. Furthermore, transfecting Rb back into these cells restored activation. This shows that Rb is required for E1a activation of p53-regulated genes. Subsequent bioinformatic analysis revealed that the transcription factor, Sp1, is common to all p53-responsive promoters tested. Additionally Sp1 binds

to E1a, implicating its involvement in transcriptional activation of these promoters. Furthermore, mobility shift assays showed an increase in Sp1 bound to the BAX promoter in the presence of E1a.

Thus, E1a and Rb can co-operate with Sp1 to activate p53-target genes and may fulfill some of p53's roles in p53 mutant or null cancer cells. This evidence may go some of the way to explain the limited success of attempts to target p53 deficiency as criteria for selectivity of conditionally replicating Ads.

Long-term rescue of striatal neurons and motor skills by combined antioxidant-hypothermia treatment after neonatal hypoxic-ischemic brain injury in the rat.
C Hobbs, D Oorschot. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

Neonatal hypoxia-ischemia (HI) is a major cause of cerebral palsy. Neuronal cell death in the striatum of the brain contributes to the motor deficits of cerebral palsy. In our earlier study, combined antioxidant-hypothermia treatment significantly protected striatal neurons one week after neonatal HI in the rat. This study investigated the long-term efficacy of this combination.

To investigate long-term neuroprotection, male Sprague-Dawley rats received six, 12-hourly subcutaneous injections of the antioxidant S-PBN (N-tert-butyl-(2-sulfophenyl)-nitrone, 100 mg/kg, n = 12), or its diluent (n = 12), from postnatal (PN) day 7. HI was induced on PN 8 by right common carotid artery ligation under anaesthesia, followed 2.5 h later by exposure to 8% oxygen for 1.5 h. Diluent-treated pups were then exposed to normothermia (37°C), and S-PBN-treated pups to hypothermia (26°C), for 6 h. Serial 40 µm sections were cut through the right striatum of 12 week old rats and coded. The total number of striatal medium-spiny neurons was stereologically determined using the optical disector/Cavalieri method. S-PBN/hypothermia treatment significantly preserved medium-spiny neurons compared with diluent/normothermia (2,578,000 ± 155,000 versus 1,893,000 ± 192,000, mean ± SEM, p = 0.0083, 2-tailed Mann-Whitney *U* test).

A separate experiment investigated fine motor skills using the staircase test. Eight animals from each HI-exposed treatment group, and 7 normal control animals, were tested daily by a blinded observer, from 9-11 weeks-of-age. S-PBN/hypothermia prevented HI-induced, long-term motor deficits in the forelimb contralateral to the lesion. Specifically, S-PBN/hypothermia improved grasping (p = 0.031; repeated-measures ANOVA) and depth of reach (p = 0.015) compared to diluent/normothermia. HI-exposed, S-PBN/hypothermia-treated animals and normal animals did not differ in either measure (p = 0.503 and p = 0.547, respectively).

This is the first study to identify a treatment that offers persistent striatal neuroprotection and preservation of fine motor skills following neonatal HI in the rat.

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Encapsulation of a model protein from nanoparticles prepared by interfacial polymerisation of different structure types of microemulsions. K Krauel[1], NM Davies[2], T Rades[1]. [1]School of Pharmacy, University of Otago, Dunedin, [2]School of Pharmacy, University of Queensland.

Poly (alkylcyanoacrylate) (PACA) nanoparticles containing protein may be a useful system for the delivery of proteins, especially vaccine delivery. The aim was to investigate entrapment of a model protein, fluorescein isothiocyanate conjugated ovalbumin, (FITC-Ova) in nanoparticles prepared by interfacial polymerisation of microemulsions (water in oil (w/o) droplet and bicontinuous) and to study the effect that the type of microemulsion template and concentration of monomer and protein have on entrapment.

Nanoparticles were prepared by dissolving 100-600 mg ethyl 2-cyanoacrylate monomer in 300-1800 mg chloroform and slowly adding this mixture to a microemulsion. Polymerisation was performed at 4°C and stirring overnight. FITC-Ova (0.1-5 mg) was added to the water component. Particle size and polydispersity index of the resulting nanoparticles were measured by photon correlation spectroscopy and morphology was observed by field-emission cryo-scanning electron microscopy. Determination of the entrapment efficiency was carried out by measuring the fluorescence intensity of the supernatant after spinning down the nanoparticles.

Entrapment of protein was up to 95% and was influenced by the amount of monomer and protein used, whereas entrapment of FITC-Ova increased with increasing monomer concentration from 42% (100 mg monomer) up to 95% (600 mg monomer). Entrapment of FITC-Ova dropped upon increasing the protein and keeping the monomer constant and might be explained by an exceedance of the loading capacity of the nanoparticles between 0.1-0.5 mg FITC-Ova. The type of microemulsion also influenced the entrapment and using equal quantities of protein and monomer led to entrapment of 30% (300 mg monomer) when using a bicontinuous microemulsion compared to 75% with a w/o droplet microemulsion, suggesting that the larger water domains in the bicontinuous microemulsion offered less possibility for interaction of protein with the monomer during polymerisation.

Due to their particulate character and high entrapment efficiency for proteins, PACA nanoparticles render an interesting system for the delivery of vaccines.

Elastic tissue defects: The link between low birthweight and cardiovascular disease? K Pascoe[1], G Jones[1], J Ledingham[2]. [1]Department of Surgery, [2]Department of Pharmacology, Otago School of Medical Sciences, University of Otago, Dunedin.

Atherosclerosis, the pathological entity that lies behind significant morbidity and mortality in the developed world, is often thought of as disease of old age. In fact pathological evidence has determined that the atherosclerotic process begins at a very early age, if not *in utero*. More recently, epidemiological evidence has linked the *in utero* condition with cardiovascular events later in life. The effect of *in utero* growth restriction in the brown norway (BN) rat was examined. The BN rat spontaneously develops elastic tissue defects in the abdominal aorta, which are known to play an integral role in atherosclerotic plaque initiation and progression.

Sixty-three dams were used in the study. The uterine arteries in the ligation group (21 dams) were ligated on day 18 of pregnancy, which decreased pup weight at 72 hours in males and females by 14% when compared to no surgery pups ($P < 0.05$). Dissected aortas were opened longitudinally, pinned flat and stained with Haematoxylin, permitting the visualisation and counting of elastic tissue defects under a light microscope. In both sexes, the abdominal aortas from the ligation group had double the number of elastic tissue defects ($p < 0.05$) compared to the no surgery group at eight weeks of age. At sixteen weeks of age male ligation animals still have 60% ($p = 0.0006$) more defects than the no surgery group, however this difference is no longer evident in females. This increase in defect numbers is not accompanied by any significant change in blood pressure (as assessed by conscious tail-cuff or unconscious intra-arterial methods).

This study supports the hypothesis that links the *in utero* environment to cardiovascular disease later in life. It appears that a even a moderate insult, such as this, can permanently alter blood vessel structure, leading to the formation of increased numbers of elastic tissue defects.

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Basic fibroblast growth factor and small human ovarian follicle initiation.
J Quennell, J-A Stanton, P Hurst. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

Ovarian follicles are nests of cells where female gametes (oocytes) are surrounded by their support cells (the granulosa cells). The molecular mechanisms that underlie the initiation of follicle growth and differentiation are poorly understood. Basic fibroblast growth factor (bFGF) is a molecule that has been implicated in the growth and differentiation of granulosa cells in culture since the 1970s. In this study human ovarian biopsies were used to investigate bFGF gene expression during small follicle development.

Ovarian biopsies were obtained from healthy fertile women, ranging in age between 25 - 34 years, undergoing tubal ligation for fertility control. Small follicles of different developmental sizes were isolated from 1 mm^3 biopsies by laser capture microdissection (LCM). Follicles from 12 µm sections were classified into three different developmental categories (small non-growing follicles, primary follicles, or small secondary follicles) by microscopic criteria. The oocytes and granulosa cells of each follicle category were independently isolated for each patient via LCM. RNA was isolated from each sample and reverse transcribed. Analysis of bFGF gene expression was carried out using real-time PCR. After normalising bFGF gene expression to the housekeeping gene 18S, expression levels for different follicle populations were compared within each patient. All patients showed decreasing expression of bFGF mRNA as the follicles matured ($n = 7$; $p < 0.05$).

This finding suggests that bFGF gene expression is not positively correlated with follicle growth. Instead bFGF could have an inhibitory affect on follicle development. Alternatively the results presented here augment emerging evidence from granulosa cell cultures that bFGF may act as an anti-apoptotic survival factor.

Rad51, a promising new target for Adenoviral tumour therapy. IA Russell[1], JA Royds[1], H-W Stürzbecher[2], AW Braithwaite[1]. [1]Cell Transformation Group, Pathology Department, Dunedin School of Medicine, University of Otago, Dunedin. [2]Institute of Human Genetics, Lübeck Medical University, Ratzeburger Allee 160, 23538 Lübeck, Germany.

Aberrant DNA repair and cancer aetiology are intimately linked. Loss of the ability to repair DNA often results in genomic instability and subsequent tumourigenesis. Likewise, a proportion of high-grade tumours typically over-express DNA repair proteins, such as Rad51. Rad51 is a central player in the repair of double-strand DNA breaks by homologous recombination (HR). Rad51 acts by catalysing the invasion of ssDNA from the break site into a homologous template. Recent evidence indicates that Adenoviral (Ad) proteins can interact with, and manipulate, components of the cell's DNA repair machinery. With this in mind, we have investigated further the interplay between Ads and the DNA repair machinery.

By infecting human fibroblasts with wild-type Ad5, and using protein expression as a marker, we showed that Rad51 levels were markedly up-regulated upon infection. This effect was generalisable to a number of diverse cell types. Rad51 up-regulation has been demonstrated using an array of Ads housing mutations spanning the majority of their early regions. All but one mutant tested led to up-regulation of Rad51. The exceptional mutant, ts125, contains a single amino acid substitution in the DNA binding protein. Flow cytometry data derived from this mutant, coupled with southern and western analyses, directly tied Rad51 up-regulation to Ad DNA replication. Moreover, transient over-expression of Rad51 led to a 45-fold increase in Ad progeny.

We have shown for the first time that Ad specifically up-regulates Rad51 protein expression. Ts125 mutant analysis has coupled this up-regulation directly to Ad DNA replication. Furthermore, over-expression of Rad51 during infection yielded significant increases in the production of daughter virions. Collectively, these data suggest that Rad51 plays a direct role in the Ad replication strategy, increasing the efficiency of the replication process. Importantly, these novel findings may be exploited in the development of therapeutic Ads to target those tumours that over-express Rad51.

Anti-Müllerian hormone may be an autocrine regulator of motoneurons. P-Y Wang, K Koishi, I McLennan. Department of Anatomy and Structural Biology, Otago School of Medical Sciences, University of Otago, Dunedin.

Motoneurons are irreplaceable cells, whose survival is controlled by multiple factors, at least some of which are unknown. Our group has previously used microarray analysis of motoneurons to identify novel regulators. A highly expressed growth factor receptor in the analysis was the type II anti-Müllerian hormone receptor (AMHRII). This receptor mediates the actions of AMH *in vivo*, in concert with the type Ia bone morphogenetic protein receptor (BMPRIa). AMH contributes to sex differentiation and certain functions of adult gonads. The presence of AMHRII in non-sexually-related neurons is without precedence. In this study, we have examined the existence of AMH-related molecules in motoneurons.

Adult spinal motoneurons were isolated from both male (n = 6) and female (n = 5) mice using laser capture microdissection. The copy numbers of AMHRII mRNA and other transforming growth factor (TGF)-beta superfamily mRNAs were quantified in non-amplified total RNA, using real-time PCR. The abundance of AMHRII transcripts (0.41 ± 0.04 % relative to glyceraldehydes-3-phosphate-dehydrogenase, mean ± SEM, n = 11, $p < 0.0001$; *t*-test) was significantly higher than other receptors, including RET (0.09 ± 0.014 %) and TGF-betaRII (0.01 ± 0.005%), which mediate the classical motoneuron survival factors. The ligand for AMHRII (AMH) was also present in the motoneurons (0.003 ± 0.0014%), with an abundance similar to that seen in the testes (0.001 ± 0.0003%). In contrast, other members of the TGF-beta superfamily, such as BMP2, BMP4 and BMP6 were undetectable. The levels of AMH and AMHRII in females were 78 ± 26.3% and 90 ± 4.2% of those in males respectively. AMH and AMHRII proteins were also present: a 63 kDa protein was detected in spinal cord extracts after immunoprecipitation with an antibody specific to AMHRII; AMH and AMHRII were also detected in motoneurons, using immunohistochemistry.

The colocalisation of AMH and AMHRII in motoneurons is consistent with AMH being an autocrine regulator of their functions. This hypothesis is currently being tested.

Supported by a grant from the Royal Society of New Zealand Marsden Fund.

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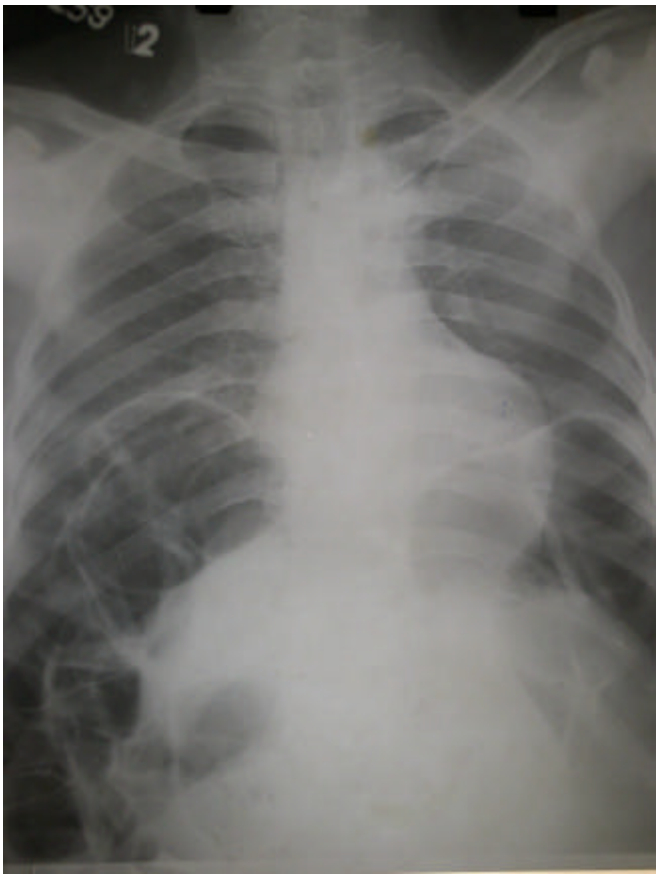
Chilaiditi's Syndrome

Nikhil Kulkarni, Guneesh Dadayal, Sashidhar Yeluri, Amit Kapoor, Ashish Gupta

A 65-year-old male presented with features suggestive of an obstructed right inguinal hernia. An X-ray of the abdomen in the standing position was suggestive of free gas under the right hemidiaphragm (Figure 1). Classical haustral markings could be seen, however, which led to the suspicion of Chilaiditi's anomaly. Normal loops of bowel were seen on inguinal surgery and herniorrhaphy was performed. The patient was counselled about the incidentally discovered anomaly.

Figure 1. X-ray standing film of the abdomen showing the presence of free gas under the right dome of the diaphragm with the presence of classical haustral markings



Discussion

Chilaiditi's Syndrome (CS) is a hepatodiaphragmatic interposition of the colon and rarely the small intestine. It occurs in increased proportions in patients with chronic

lung disease, post necrotic cirrhosis, and ascites. Colonic elongation and laxity of colonic and hepatic suspensory ligaments are the principal predisposing factors.

Although it is usually asymptomatic and an incidental finding in the elderly population, several atypical presentations have been reported in the literature in the form of isolated case reports—and include abdominal discomfort, constipation, partial obstruction, nocturnal vomiting, and recurrent volvulus involving transverse colon and both flexures. In symptomatic cases, suggested treatment options include transverse colectomy, right hemicolectomy, and hepatic extraperitonealisation (hepatopexy).

Although classical haustral markings suggestive of the diagnosis may sometimes be seen, the presence of overlapping features frequently leads to a dilemma in diagnosis. A strong clinical suspicion is advocated to prevent an unnecessary laparotomy. In the elderly especially, this should always be considered in the differential diagnosis for free gas under the dome of the right hemidiaphragm.

Author information: Nikhil V Kulkarni, Ex-Senior Resident; Guneesh Dadayal, PRHO; Sashidhar V Yeluri, Resident; Amit Kapoor, Resident; Ashish Gupta, Ex-Senior Resident, Department of General Surgery, Sir Sayajirao General Hospital and Medical College, Baroda, India.

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On the other hand

The hand you favour as a 10-week-old fetus is the hand you will favour for the rest of your life. The finding comes as a surprise because it had been thought that lifelong hand preferences did not develop until a child was 3 or 4 years old. A team led by Peter Hepper of the Fetal Behavioural Research Centre at Queen's University, Belfast in the UK reached this conclusion after studying ultrasound scans of 1000 fetuses. In one study, nine out of 10 fetuses at 15 weeks' gestation preferred to suck their right thumbs. Hepper's team followed 75 of those fetuses after birth, and found that at 10 to 12 years old all 60 of the right thumb-suckers were right handed, while 10 of the 15 left thumb-suckers were left handed and the rest right handed.

New Scientist, 24 July 2004

Tonsillectomy and postoperative haemorrhage

Apparently diathermy and coblation (a variation of electrosurgery with lower tissue temperatures than diathermy) have overtaken dissection as the favoured tonsillectomy techniques. However, in a recent overview of 252 hospitals in England and N. Ireland it has been found that the overall haemorrhage rate was 3.1 times higher with diathermy tonsillectomy than with cold steel tonsillectomy without any use of diathermy ($p<0.001$). The corresponding relative risk for coblation tonsillectomy was 3.4; $p<0.001$). When cold steel was used for dissection and diathermy only for haemostasis the relative risk was 2.2. ($p=0.002$). Therefore the authors recommend that these newer methods should therefore be used with appropriate caution and only after proper training.

Lancet 2004;364:697–702

Graduate vs conventional entry to medical school?

In a study from the University of Newcastle (New South Wales, Australia) the careers of the first 16 years' graduates have been evaluated. The researchers found no significant differences between graduate and conventional entrants in terms of academic performance (as measured by the award of medical school honours) or research outcomes (as measured by completion of a research degree during or after medical school training, publication of scientific papers or holding career posts in the research sciences).

There were no differences in career positions held by clinicians, choice of general practice or another specialty as a career, practice location (rural or urban) or employment sector (public or private). They found no clear advantage, at least on the outcomes measured in this study, to limiting medical school entry to either group and felt that medical schools could reasonably broaden their selection criteria to include more graduate entry candidates.

Medical Education 2004:38:778–86.

Evidence based bad medicine

The Randomized Aldactone Evaluation Study (RALES) demonstrated that spironolactone significantly improves outcomes in patients with severe heart failure. Use of angiotensin-converting-enzyme (ACE) inhibitors is also indicated in these patients.

That is the good news. The down side is that these patients are susceptible to the development of hyperkalaemia. In a paper from Canada it has been demonstrated that after the publication of RALES there was an increase in the rate of hospitalisation for hyperkalaemia from 2.4/1000 patients to 11/1000 patients ($p<0.001$) and a commensurate increase in deaths in these patients. Unlike the selected trial patients, older patients with renal impairment and those on potassium supplements should not have spironolactone and all should have regular biochemical blood tests.

N Engl J Med 2004;351:543-51

Polio about face in Nigeria

The Nigerian government came under severe pressure last year from radical Muslim leaders opposed to the immunisation campaign on the grounds that the vaccines contained antifertility substances, which they contended may have been targeted mainly at the Muslim population in northern Nigeria.

The bickering led to a suspension in August 2003 of a UN backed campaign to eradicate polio by the end of 2004. This resulted in a marked increase in the number of polio infected and paralysed children and the reinfection of previously polio free states in Nigeria and exportation of the virus in at least six neighbouring countries.

However, in July the Nigerian Health Minister apologised on behalf of the Nigerian government for this development and at the same time pledge to work harder to make polio a history by the end of this year.

BMJ 2004:329:365

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Looking back at the 1987 Cervical Cancer Inquiry

The Auckland Women's Health Council (AWHC) would like to respond to the article by Barbara Heslop entitled *'All about research'—looking back at the 1987 Cervical Cancer Inquiry*, which appeared in the NZMJ on 6 August 2004 (http://www.nzma.org.nz/journal/117-1199/1000/).

The article is of considerable concern to AWHC members as it represents a misguided attempt to rewrite history and casts doubt on a significant event in the history of the medical profession and the development of patient rights. The AWHC has among its members women who sat through the whole Inquiry into the Treatment of Cervical Cancer at National Women's Hospital as well as others who gave evidence or attended some of the sessions. The AWHC was formed at the beginning of 1988 and has always had a special interest in the issues that arose during what has become known as the Cartwright Inquiry.

Over the past 16 years, the Council has been actively involved in the implementation of the recommendations contained in the Report of the Cervical Cancer Inquiry including actively supporting the establishment and ongoing development of the National Cervical Screening Programme, the establishment of the office of the Health and Disability Commissioner, the development of the Code of Rights, and the nationwide patient advocacy system.

It is simply not possible to respond in the form of a letter to the editor to the number of unsubstantiated claims in Barbara Heslop's article. Most of the claims made in the article are in fact refuted in both the Cartwright Report and Sandra Coney's book, *The Unfortunate Experiment*. The AWHC would also draw attention to the fact that the 5000 pages of evidence contained in the transcripts of the Inquiry are also publicly available and prove that the issues Ms Heslop raised were all thoroughly canvassed during the Inquiry.

For example, it was irrefutably demonstrated at the Inquiry that there were medical researchers both here in New Zealand and throughout the world who not only knew about "the unfortunate experiment" that was underway at National Women's Hospital but that during the 1960s and 1970s some of them actually visited the hospital and met with Herbert Green. Many in the medical research community were horrified by their observations during their visits, and word of what was going on spread far and wide among the research community. People like Ralph Richart openly challenged Green at international symposia.

It simply isn't true that Professor Green worked in isolation. The evidence presented during the Inquiry revealed that he was in fact supported in his views by other senior doctors at the hospital—Bonham, Jamieson, and Liggins.

The AWHC would also point out that Bill McIndoe and Jock McLean were not as ignorant as Ms Heslop attempts to make them. It was demonstrated very clearly at the Inquiry that, contrary to Ms Heslop's assertion that neither man "had given much serious thought to scientific hypotheses," both knew exactly what they were doing and had spent decades fighting Green and attempting to protect women whom they

knew to be in serious danger of developing cervical cancer because they were not being treated. The families of these two men believe that the stress they were under contributed to their early deaths. This is why a decade ago the Auckland Women's Health Council and Women's Health Action held a special ceremony during which a pohutukawa tree was planted and a plaque referring to their work placed at the foot of the tree. So their efforts would not be forgotten—or misinterpreted and maligned by those who come after.

We must not forget that over 30 women died as a result of being part of "the unfortunate experiment at National Women's Hospital" and their untimely deaths were entirely avoidable. The 1960s and 1970s were not the Middle Ages of medical research that Ms Heslop's article would make them out to be. There was in fact an international framework arising from the Nazi experiments during World War 2 that set a clear standard for ethics of research that 50 years later meets the test of today.

Lynda Williams
Director
Auckland Women's Health Council

Response

I fail to see why I was "misguided" in recording my perception of medical research over the half century during which I was involved with it. Nor do I delude myself that I, or anybody else, can "re-write history". Because I am not quite sure what it means, I make no comment on the assertion that I "cast doubt on a significant event…". I should perhaps point out that medical scientists, among whom I number myself, are apt to see life as long on doubts and short on certainties.

It is also difficult to comment on the "number of unsubstantiated claims" that I am said to have made, since the single example cited by Ms Williams was not a claim that I had made. I certainly did not maintain that doctors and researchers in NZ during the 1960s and 70s did not know that Herb Green held unorthodox views about cervical pathology. I was well aware of it, as were many of my colleagues. What many of the NWH staff lacked was the scientific "know how" that might have prompted them, when presented with a hypothesis, to ask "Is this hypothesis testable? What sort of evidence will it take to falsify it? Will the evidence be easy to get? How long will it take? Is this the best way of getting the evidence? Are there other ways in which the hypothesis could be falsified? How feasible are they?" and so on. Those with service commitments—clinical or laboratory—usually have neither the time nor the need to ponder on the best ways of dealing with research problems.

There is nothing wrong with holding unorthodox scientific views as long as one can justify them. Much productive research, after all, involves disagreeing with somebody or something. This is the way the scientific world operates—disagreement and questioning are its lifeblood. Trouble arises not because of differences of opinion per se, but when the accuracy of the data on which the opinions are based becomes suspect. There is little doubt that the NWH hierarchy took far too long to take a hard look at the quality of Herb Green's data. Nevertheless the main point of my article, which I reiterate, is that had the scientific "know how" of the main protagonists been more sophisticated than it was, Green's hypothesis could have been disproved

(falsified) in a few months, if not by Green himself, then by McLean. I don't for a moment doubt that McIndoe and McLean knew exactly what they were doing (most of us do). It is unfortunate that they (and especially pathologist McLean) missed seeing that they were almost certainly sitting on archival hospital material that would have allowed them not only to disprove Green's hypothesis quite quickly, but also to publish the relevant findings without reference to Green. My tentative answer to the question "Why didn't they do it?" (because they were not researchers and it did not occur to them) is rather more charitable than Ms Williams' assessment (that they did indeed know, but chose to take a longer and incomparably more stressful route to their destination).

Pointing this out does not entail "misinterpreting or maligning" anybody living or dead, nor does it detract from the significance of what they eventually did. It is no more derogatory than commenting that my parents' generation could have communicated more effectively had they been familiar with today's information technology, or that I could have approached yesterday's immunogenetic problems more effectively had I used today's molecular biological techniques. It merely serves as a reminder that yesterday's research is apt to have obvious shortcomings when viewed from the vantage point of today's knowledge.

Being challenged by the leaders in the field on one's home ground or at conferences does not necessarily mean that one's opinions are wrong, a point well illustrated by Bryan Sykes in *The Seven Daughters of Eve* (Corgi Books 2001, pp 190–193). What it does mean is that one's evidence had better be pretty good, and freely available to anybody who wants to scrutinise it minutely. Those who present material at international conferences—from plenary sessions to posters—expect to be challenged. For heaven's sake, why would anybody go to a conference if this were not going to happen?

Finally, the assertion "The 1960s and 1970s were not the Middle Ages of medical research that Ms Heslop's article would make them out to be". Actually I specified those years as occupying the dawn of the current golden age of biology, and noted the shortcomings of "a lot of medical research and especially clinical research" at that time. Those who are familiar with the allocation of money for medical research will know that for the last 30 years or so, clinical research has experienced difficulty attracting funds in competition with scientifically more sophisticated biomedical research projects. This has been a world-wide phenomenon, and has prompted various solutions, including the establishment of multidisciplinary collaborative research groups whose members have complementary skills. Whatever label one chooses to attach to Green's working years, today's clinical research is hugely different from most of that practised in the 1960s and 1970s. It is not too hard to see why the change had to happen.

Barbara Heslop
Emeritus Professor
Dunedin

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Operative rates for acute intussusception in New Zealand

Acute intussusception (IS) is the most common cause of intestinal obstruction in young children.[1] Its non-operative reduction results in less morbidity, shorter hospital stay, and lower costs.[2] Some overseas centres obtain overall operative rates for IS as low as 22%,[3] with the remainder treated by enema reduction. Accurate information on current management is needed to determine whether similar outcomes are achieved in New Zealand.

Data were obtained from the New Zealand Health Information System (NZHIS) for all public hospital admissions in children aged <15 years with a discharge diagnosis of IS (ICD-10-CM International Code K56.1) from January 1998 to June 2003 inclusive. These were compared with local data from paediatric surgical, radiological and hospital discharge coding databases in Wellington and Christchurch Hospitals. The case notes of each patient from the 2 hospitals were reviewed to confirm that IS had occurred and was correctly coded.

While there was general agreement between NZHIS and local databases for identifying IS, the procedural coding data were difficult to interpret. Of the 325 cases identified by the NZHIS, the overall operation rate was 17.5%, gas enema 26%, barium enema 7%, with 49.5% of patients having no procedure recorded. The NZHIS data showed relatively low operation rates in Christchurch (12%) and Auckland (14%), with higher rates in Otago (20%), Wellington (22%), and regions without paediatric specialist surgical services (23%).

Comparisons between data from local audit in Christchurch and Wellington, and NZHIS, confirmed that a large proportion of the procedural coding for management of IS during the study period, especially gas enemas, was missing from the NZHIS database. Of 34 patients admitted to Christchurch Hospital (1998–2003), NZHIS coding missed 29 gas enemas (in 19 children), 5 surgical manipulations, 1 resection, and 2 barium enemas. For 45 patients admitted to Wellington Hospital during the same period, NZHIS data omitted 15 gas enemas (in 15 children), 10 barium enemas, 4 surgical manipulation, and 4 resections. Local audit found surgical rates of 32% in Christchurch and 40% in Wellington, rates much higher than derived from NZHIS data. Furthermore, some diagnostic coding errors were discovered. NZHIS failed to detect 5 cases found by local audit and 4 patients from NZHIS were not identified by the national paediatric surgical database during the local audit process.

Thus the reliability of current coding and audit systems makes it difficult to determine the quality of IS management with any degree of accuracy and certainty. Referral bias from the transfer of more difficult patients from smaller regional hospitals might explain the higher operative rates encountered in the two audited tertiary centres.[4] To ensure that the optimal management of IS in New Zealand is being achieved, prospective collection of clinical indicator data for IS, along the lines advocated by the Royal Australasian College of Surgeons and Quality New Zealand, is needed. The unexpected association between a tetravalent rhesus-based rotavirus vaccine (RRV-TV; Rotashield; Wyeth-Lederle Vacccines and Pediatrics) and IS further reinforces

the importance of accurate IS data collection, particularly with the imminent licensure of new rotavirus vaccines.[5]

Ellen Chen
Medical Student
Christchurch School of Medicine and Health Sciences

Keith Grimwood
Professor of Paediatrics and Child Health
Wellington School of Medicine and Health Sciences

Spencer Beasley
Professor of Paediatric Surgery
Christchurch School of Medicine and Health Sciences

Ellen Chen received a summer studentship supported by a non-directional grant from GlaxoSmithKline. We thank Rebecca Kay of NZHIS for assistance with case detection.

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Do you remember the introduction of 'The Pill'?

Were you a GP in the 1960s?

Can you help us?

Historians at The University of Otago are researching the revolutionary introduction of the oral contraceptive pill into New Zealand in 1961. We are looking for GPs and Pharmaceuticals Reps who were practicing when the pill was introduced. Is this you? We would like to speak with you about your memories of that time and the impact the pill had on your work.

Interviews can be arranged at a time and place of your convenience over the next couple of months, we'd love to hear from you! Please email or write if you would like to participate.

Nancy de Castro
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Email: nandecastro@yahoo.co.nz

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Lions Clubs New Zealand

The Lloyd Morgan Lions Clubs Charitable Trust Fellowship Award

Fellowship grant to celebrate 50 years of Lions in New Zealand

In recognition of the 50th anniversary of Lions Clubs of New Zealand, the Lloyd Morgan Lions Clubs Charitable Trust Board has announced its intention to award a Trust Fellowship in April 2005.

The Fellowship recipient's research project may involve, but is not limited to, the prevention, diagnosis, treatment and cure of health conditions. The monetary value of the Fellowship will be based on the nature of the application.

Expressions of interest are invited from any organisation who wishes to sponsor a person who could be considered or would qualify for this Fellowship.

David Gault, Chairman of the Trust Evaluation Committee says, "The Fellowship recipient will be a person who has achieved pre-eminence in their particular field of expertise. The awarding of the Fellowship is a significant recognition by Lions Clubs New Zealand of that person's merit, knowledge and qualifications and their potential to advance the welfare of New Zealanders."

This is only the second Fellowship in the 25 year history of the Trust. The first was awarded in April 2000 to Dr. Roderick D MacLeod to celebrate the Trust's 20th Anniversary.

Ron Lawrence, Chief Executive Officer, Lions Clubs New Zealand says "The Fellowship is a significant honour. In monetary terms it provides valuable recognition to an individual who has made a noteworthy contribution in his or her respective field."

Expressions of interest must be submitted by 31 January 2005 (for details on how to apply please see fact sheet). The Fellowship will be awarded in April 2005.

For any further information please contact:

David Gault

Chairman, Trust Evaluation Committee

Phone – (04) 237 7436

Email – david.gault@xtra.co.nz

FACT SHEET

The Lloyd Morgan Lions Clubs Charitable Trust

- The Trust was established in 1979-80 in honour of Lions member Lloyd Morgan, and to commemorate Lloyd's year as President on the International Association of Lions Clubs. Lloyd sadly passed away on 27 August 2001.
- The Trust has become the national resource for the charitable work of Lions Clubs throughout NZ, providing a grant or a loan to Lions Clubs for a range of projects.
- The Trust benefits sick, disabled and distressed people in NZ and the Pacific Islands. It also helps in the field of education.
- The Trust has been able to assist with numerous appeals and projects and already hundreds of thousands of dollars have been given to Lions Clubs by way of grants or loans to assist with a multitude of Lions Club projects.

Fellowship Applications

- Expressions of interest are invited from any organisation that wishes to sponsor a person who would be considered or would qualify for this recognition of excellence.
- In the first place, please send a curriculum vitae in full, addressed to the:

  Trust Chairman
  The Lloyd Morgan Lions Clubs Charitable Trust.
  P.O. Box 1335,
  Palmerston North.

- The closing date for expressions of interest accompanied by a CV is **31 January 2005**. The Fellowship award will be made in April 2005.

Lions Clubs in New Zealand

The first New Zealand Lions Club was formed in 1955 in Auckland. Now there are around 500 Clubs, and 12,700 members. Worldwide, the International Association of Lions Clubs has a membership of over 1.3 million in 192 countries and geographical areas.

The emphasis is on community service in all forms. Lions programmes serve the young and the aged, the disabled and the disadvantaged - anybody who has a need. Programmes are conducted locally, nationally and internationally. They include sight, conservation and work with the blind, citizenship services, hearing and speech action, programmes with the deaf, drug education, and environment, recreational, health and social services.

Lions' contributions to the development and care of New Zealand youth include living skills programmes, drug awareness, an international youth exchange programme, the national Young Speechmaker Contest and International Peace Poster Competition.

In 2005, Lions Clubs New Zealand will celebrate its 50th anniversary of service in New Zealand.

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GRANTS AWARDED JULY 2004

At the July meeting of the Scientific Committee of the National Heart Foundation, a total of 27 grants were awarded. The awards included 10 Project Grants, 11 Fellowships, 2 Small Project Grants, 1 Grant-in-Aid and 3 Travel Grants.

PROJECT GRANTS

Dr John Beca

Paediatric Intensive Care Unit, Starship Hospital

Early prediction of brain damage after heart surgery in infants

$120,000 for 15 months.

Professor Peter George

Canterbury Health Laboratories, Christchurch

Betaine, diet and homocysteine

$200,000 for two years.

Drs Christopher Charles & David Jardine

Christchurch Cardioendocrine Research Group, Christchurch School of Medicine and Health Sciences, University of Otago

Cardiac sympathetic nerve activity: effects of myocardial infarction: sympatholysis and novel hormones

$190,000 for three years.

Dr Beverley Lawton

Department of General Practice, Wellington School of Medicine and Health Sciences, University of Otago

New Zealand Women's Lifestyle Study

$318,073 for three years.

Dr Paget Milsom

Cardiothoracic Surgical Unit, Auckland City Hospital

Dual vent cardiopulmonary bypass to reduce ischemic brain injury in cardiac surgery

$136,424 for two years.

Dr Cliona Ni Mhurchu

Clinical Trials Research Unit, Faculty of Medicine and Health Sciences, University of Auckland

Development of culturally appropriate nutrition education materials for use in a large trial

$97,811for one year.

Professor Russell Scott

Lipid and Diabetes Research Group, Christchurch Hospital

Coenzyme Q10: Potential for improving cardiovascular risk

$107,564 for one year.

Dr Ralph Stewart

Cardiology Department, Auckland City Hospital

Natriuretic peptides for assessment of severe aortic regurgitation

$40,827 for three years.

Dr Rachael Taylor

Department of Human Nutrition, University of Otago

The APPLE project: preventing obesity in children via environmental intervention

$78,242 for three years.

Dr John Lainchbury

Christchurch School of Medicine and Health Sciences, University of Otago

Clinical and neurohormonal predictors of outcome post-DC cardioversion for atrial fibrillation

$111,787 for three years.

FELLOWSHIPS

Ms Gillian Whalley

A National Heart Foundation Senior Fellowship (for three years) was awarded to Ms Gillian Whalley. Dr Whalley will undertake clinical research on 3 projects at the Department of Medicine, University of Auckland.

Dr Nicholas Kang

A *National Heart Foundation Senior Fellowship* (for two years) was awarded to Dr Nicholas Kang. Dr Kang is a Paediatric Cardiac Surgeon who will undertake his Fellowship at the Starship Children's Hospital in Auckland.

Dr James Blake

An *Overseas Training Fellowship* (for one year) was awarded to Dr James Blake. Dr Blake will work as an Interventional Cardiology Fellow at the William Beaumont Hospital in Michigan, USA.

Dr Seif El-Jack

An Overseas Training Fellowship (for one year) was awarded to Dr Seif El-Jack. Dr El-Jack will work as an Interventional Cardiology Fellow at the William Beaumont Hospital in Michigan, USA.

Dr Sanjeevan Pasupati

An *Overseas Training Fellowship* (for one year) was awarded to Dr Sanjeevan Pasupati who will work as an Interventional Cardiology Fellow at St Paul's Hospital, Vancouver, Canada.

Dr Chris Raffel

An *Overseas Training Fellowship (E & W White-Parsons Fellowship)*, for one year, was awarded to Dr Chris Raffel. Dr Raffel will work as an Interventional Cardiology Fellow at the Massachusetts General Hospital and Harvard Medical School, USA.

Dr Cara Wasywich

An *Overseas Training Fellowship* (for one year) was awarded to Dr Cara Wasywich. Dr Wasywich will work as a Clinical and Research Fellow in the Cardiology Department at St Paul's Hospital, Vancouver, Canada.

Dr Judith McCool

A *Research Fellowship* (for three years) was awarded to Dr Judith McCool who is currently employed in the Department of Psychology, Faculty of Medical and Health Sciences, University of Auckland.

Miss Hannah Badland

The *Maori Cardiovascular Research Fellowship* (for three years) was awarded to Ms Hannah Badland, a PhD student in the Division of Sport and Recreation, Faculty of Health, Auckland University of Technology.

Mr Euan Rodger

A *Postgraduate Scholarship* (for three years) was awarded to Mr Euan Rodger who is currently a PhD student in the Department of Biochemistry, University of Otago.

Dr Hamish Jamieson

A *Postgraduate Scholarship* (for two and a half years) was awarded to Dr Hamish Jamieson who will work as a PhD student at the Centre for Education and Research on Ageing, Concord Hospital, Sydney University.

SMALL PROJECT GRANTS

Professor Evan Begg

Department of Medicine, Christchurch School of Medicine and Health Sciences, University of Otago

Effect of CYP2D6 genotype on final tolerated dose, plasma concentrations and outcomes with carvedilol in patients with systolic heart failure

$14,403 for 18 months.

Ms Kathryn Bethune

School of Pharmacy, University of Otago

Assessment of the impact of a pharmacist managed intervention for secondary prevention of cardiovascular disease - pilot study to test methodology

$14,783 for one year.

GRANT-IN-AID

Assoc Professor Robert Doughty

Department of Medicine, Faculty of Medical and Health Sciences, University of Auckland

To purchase refrigerated centrifuge for neurohormone studies

$6,573

TRAVEL GRANTS

Mr James Aoina

Department of Medicine, University of Auckland

Annual Scientific Meeting of CSANZ, Brisbane, Australia

Professor Gregory Kolt

Faculty of Health, Auckland University of Technology

8th International Congress of Behavioural Medicine, Mainz, Germany

Dr Cara Wasywich

Department of Medicine, Faculty of Medical and Health Sciences, University of Auckland

American Heart Association Scientific Sessions, New Orleans, USA

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The Royal Australasian College *of* Physicians

Written Examination 2005

Tuesday 1 March 2005

Please note that applications for the Written Examination 2005 are now available from the Executive Officer, Tania Ireland.

Please email Tania.Ireland@racp.org.nz **or telephone her on (04) 460-8127**

APPLICATIONS ARE DUE BEFORE 22 NOVEMBER 2004

- If you are re-sitting the examination, an application from will automatically be sent to you.
- Any queries regarding your basic training? Please see your Director of Physician Training (DPT)/Director of Paediatric Physician Training (DPPT) first. If they are unable to answer your query, please contact the Executive Officer.