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“Public health is the art of applying science in the context of politics so as to reduce inequalities in health while ensuring the best health for the greatest number”.

After more than a decade of increasing social and economic inequalities in New Zealand, a Prime Ministerial Committee has been set up to close the ‘gaps’. This article emphasises that health status does not just vary across the gap between rich and poor, but is graded across the whole population, and that universal, rather than targeted policies are likely to have more impact on health.

Why do some groups of people consistently enjoy better health and live longer than others? Just as an individual's health is determined in part by the gene pool, the characteristics of the communities in which people live and work – the social equivalents of the gene pool – explain differences in health between groups. The causal pathways are not clearly established, but the link between social organisation and health has been shown in many developed countries. For instance, although many children who live in adverse social and economic circumstances develop into healthy and competent adults, on average, socio-economic disadvantage is embodied in children’s height and cognitive development.

Inequalities in the distribution of material resources, income, education, employment and housing, generate health inequalities. All sectors of society are affected: there are no neat ‘cut-off’ points. As a result, health status is distributed as a gradient up the social hierarchy and applies to almost all causes of death - from cancer, cardiovascular disease and Alzheimer’s dementia, to injuries. Individual health-related behaviours such as smoking only partially explain this strong graduated relationship, and such behaviours are themselves socio-economically patterned.

Some economists and conservative politicians maintain that economic inequality is good for incentives, growth and wealth creation. According to this view, equity is a necessary casualty in the pursuit of an efficient economy. The counter argument is that inequality inhibits economic growth by undermining social cohesion, ignoring investment opportunities, lowering levels of education for the poor, reducing spending power and increasing social, economic and political instability. Several cross-national studies have found a negative correlation between the average rates of economic growth and measures of inequality. This appears to happen because countries, like New Zealand, which have large inequalities, invest less in education and other forms of human and social capital compared with countries like Finland and the Netherlands, which have more egalitarian societies.

As Kawachi et al state, “health is one of the most extraordinarily sensitive indicators of the social costs of inequality”. Countries that minimise economic inequalities are societies where children and young people are more likely to be able to develop to their full potential. These factors are essential prerequisites for greater prosperity for the country as a whole.

The New Zealand 1996 Census and related surveys show that social and economic inequalities are widespread and have significant effects on health. These health inequalities do not occur just among individuals. People living in more deprived areas are more likely to have poor health and live shorter lives. At a regional level in New Zealand, income inequality (over and above household income) is adversely associated with both mortality and hospitalisation rates.

Woven in with social and economic determinants of health is an additional factor: ethnicity. Maori and Pacific peoples at all educational, occupational and income levels, have poorer health than non-Maori. This suggests there are other, pervasive characteristics of New Zealand society that cause poor Maori and Pacific peoples’ health.

The most pronounced indicator of social inequality in New Zealand over the last two decades is the growth in income inequality. Can the negative impact of social inequality on health be reduced by material redistribution? Probably. Governments have the power to redistribute resources through taxes, and in New Zealand, where total tax revenues as a percentage of GDP are below average for OECD countries, there is clear support from the majority of adults for income distribution as a key role of central government. However, increasing the progressivity of income tax will only have an impact on reducing inequality to the degree that wealthy people do not avoid taxes.

Research suggests that redistributive policies probably have an overall positive effect on health and might even benefit those on the highest incomes. But income redistribution is unlikely to be sufficient to eliminate population health differences, when the primary source of inequality lies in the distribution of social and institutional opportunities that affect health. For example, educational and housing investments are critical, as is improving workplace safety.

Herein lies one of the difficulties. The economic incentives inherent in different institutional arrangements may still perpetuate inequalities even as income or occupational gaps close. For example, Maori professionals with tertiary education, in common with other minority groups, still earn significantly less than non-Maori professionals.
Population-based policies directed to everybody, rather than targeted social and health measures, are (paradoxically) likely to have most benefit for the poor and those with higher risk factors. Policies designed for ‘not only the poor’ are likely to be more effective and more politically sustainable than policies targeted on class or ethnic categories. The durability of national superannuation is an example. A universal approach may improve overall health status, more especially for the poor, than a targeted approach.

While reducing inequalities is likely to have the greatest impact on population health, health care policy itself remains important. Cross-country comparisons show that if we had universal primary health care services, funded by taxes or social insurance (as do all other countries in the OECD except the USA), access to immunisations and other preventive health care would be greatly improved. The present fragmented system of targeted funding of primary health care services required large out-of-pocket payments for most people, which acts as a deterrent and hinders continuity of care.

A universal policy approach does not mean homogeneity of services. There are obligations under the Treaty of Waitangi to address the degree to which policies can differentially affect Maori. Mainstream institutions must have strong incentives to ensure Maori have equitable access to resources, including health resources. The approach taken in the 1990s was to provide start-up funds for numerous Maori providers, but it is clear that resources available to many of these providers have been insufficient. Maori providers usually service small groups of Maori, which limits their effectiveness for a population overall with high health needs. The focus on targeted services, often locally-based with limited national links, can create crucial service and information gaps.

Although provision of culturally specific health and education services indicates that the Crown may be responding to its Article II Treaty obligations, attempts to transfer resources to Maori lead to Pakeha cries of separatism. A universal provision of services under Article III would have the advantage of stressing common citizenship.

When targeted health services are promoted, care must be taken to ensure they are not isolated and under-resourced.

In conclusion, differences between social, occupational and ethnic groups are the most pressing health problem facing New Zealand. Inequality is unfair and makes us all worse off. But targeting ‘the poor’ will not minimise the overall social variations in health, may stigmatise poor people and even accentuate health inequalities. Available evidence suggests that a range of mutually reinforcing redistributive policies are needed affecting income, education, employment, housing and health services across the population. This is likely to have a major impact, both to improve New Zealand’s health and, over time, increase the country’s prosperity.

Acknowledgements. We thank Ralph Chapman, Alistair Woodward, Martin Tolhurst and Colleen Ivory for their comments.

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Regionalisation of intensive care has become commonplace. A rapid transportation system, with competent medical and nursing intensive care staff, is integral to the effective management of critically ill patients. Given New Zealand's geography, helicopters play a vital role in transportation of the critically ill. With reduction of rural health resources, an efficient system of transit care is indispensable. The Whangarei model of transit care for the critically ill exemplifies a multidisciplinary team approach. It encompasses the doctors and nurses in the ICU at Whangarei Hospital, the paramedics at the St John Ambulance and the helicopter crew who fly the Northland Emergency Services Trust (NEST) helicopter.

Methods

Northland is New Zealand's northernmost province with a population of 137,000. This swells to nearly 300,000 in summer. Northland's density of population is only fourteen per km², compared to 21 per km² elsewhere in the North Island. Whangarei Hospital is a 225 bed regional base hospital that provides specialist medical, surgical, orthopaedic, obstetric, emergency and ICU services. Kaitaia, in the far-north, is a secondary care hospital with 30-35 beds. Northland also has four primary care hospitals (a 20-bed Bay of Islands Hospital at Kawakawa, Dargaville Hospital with fifteen acute beds and hospitals at Rawene and Kaeo). Apart from the towns with primary care hospitals, Kaikohe, Kerikeri, Paihia and Russell are townships with medical centres staffed by general practitioners. 60% of Northland’s population live outside Whangarei city limits.

Whangarei Hospital’s ICU is a six-bed Level II unit providing general intensive care. It has about 425 admissions per year and 1200 patient days. 75% of ICU admissions are adults. Patients requiring Neurosurgical care and some patients requiring tertiary level critical care are transferred to the Department of Critical Care Medicine (DCCM) at Auckland Hospital. Those requiring cardio-thoracic critical care are transferred to Green Lane Hospital and patients requiring advanced care for burns are transferred to Middlemore Hospital, Auckland. Children requiring tertiary level intensive care are transferred to the Paediatric Intensive Care Unit at Auckland's Starship hospital.

All adult patients (≥ fifteen years) and most children requiring transit critical care are transported by the Whangarei transit care team. The threshold for transferring critically ill children to Auckland is lower than it is for adults. The Starship team retrieves some critically ill children from Whangarei.

Heliocopter in transit care. The ICU at Whangarei Hospital is a one-stop coordinating centre for organisation of inter-hospital transit care of the critically ill in the region (Figure 1). Inter-hospital missions are classified as Medevacs. The pre-hospital care of road trauma victims and other accidents is provided by para-medics (Advanced Care Officers) from St John Ambulance. These missions are classified as Casevacs.

Abstract

Aims. To describe our experience in transit care of the critically ill in Northland and to highlight the multidisciplinary co-operation which renders this an efficient model of transit care in suburban and rural areas.

Methods. Since its inception in 1988, Northland's wholly community owned rescue helicopter has played an integral part in transit Intensive Care in Northland. This aids in transportation of medical and nursing intensive care staff to outlying primary hospitals for patient stabilisation, subsequent transfer of the patient to the intensive care unit (ICU) in Whangarei and, occasionally, for transfer to specialised critical care services in Auckland.

Results. As of August 1999, over 3900 helicopter missions have been accomplished. A doctor, vetting each request for Medevac, minimises over- triage, thus ensuring adequate levels of transit care and effective utilisation of expensive resources. More than 90% of non-obstetric adult patients were ventilated and the number of Medevac missions increased over the years. The Accident Compensation Corporation (ACC) funds 40% of all flights, another 40% of flights are funded by Northland Health and the Order of St John funds the remainder.

Conclusions. With a widespread geography, efficient transit care of the critically ill is imperative to quality hospital care and ensures equity of access to the rural populace. The Whangarei system of transit intensive care is an ideal template for suburban and rural areas.
transferring. Rarely, the ICU doctor is requisitioned by the St John Ambulance to the scene of major accidents in the region.

Results
From 1988 until the end of August 1999, the Northland Helicopter completed over 3900 missions. Until 1997 we used a BK-117 back-loading helicopter. Presently we use a Sikorsky S-76, a two-pilot, twin-engine machine, capable of accommodating two ventilated patients and a cabin capacity for four transit care staff. Operationally the S-76 is IFR (instrument flight rules) capable at all times. A global positioning system (GPS) approach system is in place to all major health-care facilities in Northland and Auckland. Safe flights by day or night are the aim and necessity. Visual flight rules (VFR) operation alone would not suffice, given the climatic conditions in the region. The aircraft is operated to Class-I performance standards.

An indigenously designed mobile ICU, “Transit Care Bridge”, served us in the early 1990s. The Transit Care Bridge has since been developed into a modern contraption made of Kevlar and e-glass, replete with all the basic monitoring and therapeutic accoutrements of an intensive care bed (including invasive and non-invasive measurement of blood pressure, oximetry, end-tidal CO₂, ECG and temperature). This bridge clips onto the ambulance stretcher and is well suited to transportation by hand (in remote areas and hilly terrain), by surface and by helicopter.

The number of Medevac transit care missions was 120 in 1996, 160 in 1997 and 200 in 1998. The number of medical patients requiring transit care equalled the number of trauma patients in 1998 (Figure 2). Obstetric patients were over represented in 1998, reflecting the reduction of maternity services in Kaitaia Hospital. An ICU doctor and an ICU nurse staffed all Medevac missions, except obstetric ones. Midwives care for most obstetric patients, while seriously ill obstetric patients have an obstetric doctor in attendance.

Provision of a helicopter emergency medical service (HEMS) and transit care of the critically ill is expensive. The ACC bore the expenses of two transportations (surface and air) required in the first 24 hours after an accident. All transportations for trauma patients beyond 24 hours, and all transit care of non-trauma patients were funded by Northland Health. The expenses for logistics-driven missions (inclement weather precluding deployment of a surface ambulance, lack of surface ambulance due to prior commitment and other Ambulance exigencies) were borne by the St John Ambulance.

In the nearly 4000 missions (Figure 3) flown by the Northland Helicopter since its inception in 1988, 39% were funded by the ACC, Northland Health has funded another 40% and St John Ambulance has borne the expenses for 14%. Police use of the helicopter amounted to 4% of missions and others (marine rescue etc) have logged 3% of the helicopter missions.

Figure 2. Number and type of Medevac transit care missions 1996-8.

Figure 3. Number and type of Medevac transit care missions 1996-8.

Discussion
The first aeromedical evacuation occurred during the 1870 Franco-Prussian war when hot air balloons carried injured Frenchmen to safety. Air evacuation of the injured gained credibility in World War I and World War II, and reached new heights during the Vietnam conflict. The Association of Air Medical Services has provided guidelines for air medical transport of trauma patients. The first recorded civilian use of a helicopter for medical transport in New Zealand was in 1969 when John Reid of Nelson helicoptered a young girl after a caving accident. By 1971 the Auckland Surf Life Saving organisation had acquired a Hillier 12E helicopter. The first organised civilian medical use of a helicopter in USA was in 1972 in Denver, Colorado. In the same year Sydney acquired Australia’s first HEMS.

There is considerable evidence supporting the use of HEMS in the management of trauma patients and HEMS has been shown to improve survival in traumatised patients. While many studies have demonstrated the value of a rapid transportation system in the initial care of the trauma patient, a study from the London HEMS showed that a helicopter made little impact on survival, except perhaps in the most severely injured patients. Even when the methodological flaw of this study is overlooked, the study was specific to the system studied and probably has little validity for use of HEMS elsewhere. There are no valid studies yet comparing the outcome of helicopter and surface transportation in non-trauma patients who are critically ill from medical and surgical causes. In most centres, trauma accounts for only about a third of those patients as unstable; a third of his patients had life threatening complications during transfer. The need for physician intervention in patients air-transferred after myocardial infarction was emphasised in a study of 100 patients. The Australian experience highlighted the need for a coordinated approach to major incidents, including the early deployment of
Changes in work practice after a respiratory health survey among welders in New Zealand

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Abstract

Aims. To assess changes in work practice among eight New Zealand engineering sites, following a study of occupational respiratory symptoms in welders two years previously.

Methods. In 1996, we found that an acute decrease in lung function was more common in welders working without local exhaust ventilation. Findings were reported back to management, study participants and the media. Two years later we re-visited the eight welding sites.

Results. Of the eight engineering sites, two had local exhaust ventilation present in both Study 1 and Study 2, one site had installed local exhaust ventilation in part of the site since Study 1 and one site had moved to larger premises. The remaining four sites had no exhaust ventilation or change to the workplace between studies. Five welders (12.8%) used respiratory protection in both Study 1 and Study 2, seven welders (18%) used respiratory protection in Study 1 but not Study 2, four welders (10.3%) did not have respiratory protection in Study 1 but did in Study 2 and nine welders (23.1%) did not use respiratory protection at all in Study 1 or Study 2.

Conclusion. Further effort is required to ensure that such studies lead to significant improvements in the work environment.

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Welding involves joining pieces of metal by using heat and/or pressure. The process produces a complex emission (or plume) containing metal fume, and gases such as ozone, and oxides of nitrogen and carbon.1 When inhaled, this may lead to occupational respiratory symptoms in welders.2 Work as a welder has been linked with occupational respiratory effects such as asthma,3 airway irritation, acute bronchitis and metal fume fever.4 Some epidemiological studies have shown a decline in the lung function of welders throughout the working day,1,5-9 although this has not been seen in all studies.1,6 There have also been a small number of cohort studies conducted on the longterm respiratory effects of welding.1,1-13 Beckett et al did not find chronic irreversible effects on spirometry or airway reactivity in his three year study close to the welding plume.5 The use of local exhaust ventilation is the most practical way to minimise exposure.6 Respiratory protective devices are a less effective form of protection. The use of a welding helmet alone does not protect welders from inhaling welding plume.20

Several reports of cross sectional or longitudinal studies of welders’ occupational respiratory symptoms have discussed the presence or absence of local exhaust ventilation.1,2,7,9,11-19 Because the nature of welding requires welders to work with their faces close to the welding process, they have an acute exposure to the welding plume. The use of local exhaust ventilation is the most practical way to minimise exposure.2 Respiratory protective devices are a less effective form of protection. The use of a welding helmet alone does not protect welders from inhaling welding plume.20

In 1996, we visited eight engineering companies throughout New Zealand whose primary industrial process was welding (Study 1). The sites used mild steel and stainless steel as a base metal, and either metal inert gas (MIG) or tungsten inert gas (TIG) gas-shielded arc welding techniques. These produce similar emissions of metal fume, containing ozone, nitrogen dioxide and carbon monoxide, although all forms of welding produce emissions.21,22 The 1996 study found that an acute decrease in FEV₁ was more prevalent among welders than the comparison group of non-welders, and was more common among welders without local exhaust ventilation.69 Results from the study were reported back to management and study participants at each engineering site. Study results were also reported to the NZ Engineering Printing and Manufacturing Union, and in the media. We re-visited the eight engineering sites in 1998 (Study 2). Our aim was to monitor pulmonary function measurements and respiratory symptoms in as many workers as possible, two years after our first visit. The results for lung function and respiratory symptoms are reported elsewhere.23 However, we were also interested in any changes in the presence and use of local exhaust ventilation and other forms of respiratory protection, and whether these had an effect on reducing any annual decline in pulmonary function. The findings of this part of the study are reported here.

Methods

The methods for the two year follow up study have been discussed in detail elsewhere.24 In brief, we re-visited the eight New Zealand engineering sites that were included in our 1996 study (Study 1).69 All welders and comparison group participants from Study 1 were invited to take part in the two year follow up study (Study 2), which involved a questionnaire and pulmonary function testing. Workers who had left their 1996 job were invited to take part in their new place of employment or at home. The inclusion of these workers was important in order to minimise the ‘healthy worker effect’, whereby workers who suffer from work related symptoms leave their job and thus leave behind a relatively healthy active workforce.24,25 A questionnaire was administered to document demographic data, work related respiratory symptoms, respiratory protection (including local exhaust ventilation and personal respiratory protection) and smoking habits. We used questions from the Medical Research Council questionnaire26 to define chronic bronchitis (regular phlegm production at any time during the day or night for at least three months of the year and for at least two years). Smokers were defined as participants who were currently smoking at least one cigarette per day, one cigarette per week or one ounce of tobacco per month at the time of Study 1. Four smokers who quit between 1996 and our 1998 follow up study were still classified as smokers. Long term ex-smokers and participants who had never smoked according to the above definition were classified as non-smokers.

For respiratory symptoms, welding was defined as a welder at the time of Study 1 was used to differentiate between welders and non-welders in Study 2, since this was considered to be most relevant to changes in pulmonary function. For example, seven welders who stopped welding between Study 1 and Study 2 were still classified as welders. Welders were defined as study participants who spent more than 5% of their usual workday welding at the time of Study 1. Non-welders were defined as participants who had never worked as a welder, were ex-welders at the time of Study 1, or did less than 5% of welding throughout their usual work day.

For the analyses of respiratory protection, however, welding status at the time of Study 2 was used, since this was considered to be most relevant to the use of protective equipment. Thus, welders were defined as study participants who spent more than 5% of their usual workday welding at the time of Study 2.

A separate questionnaire was administered to the managers of each of the engineering sites, recording the base metal worked with, the welding technique in use and whether there was local exhaust ventilation in use. This was compared to the same observations from Study 1. The presence and use of personal respiratory protection by welders over the last two years was recorded by comparing participant responses at Study 1 and Study 2. If a welder from Study 1 had stopped welding, we asked if this was as a result of respiratory or other health problems.

Pulmonary function tests used a calibrated portable spirometer (Alpha Spirometer, Vitalograph) which was used in Study 1. Pulmonary function measurements were taken before the work shift (prior to any exposure to welding fumes and fourteen hours since any previous welding), fifteen minutes after welding exposure (or fifteen minutes into the work shift for the comparison group of non-welders), and lastly, seven hours into the work shift. Forced expiratory volume (FEV₁), forced vital capacity (FVC), forced expiratory flow at 25-75% of the FVC (FEF₂₅₋₇₅), and peak expiratory flow (PEF) were measured with each subject standing upright. The best of three forced expiratory manoeuvres were used in accordance with guidelines by Quinlan.27

All data were double entered and verified using Epi Info,28 and SAS29 was used for data analysis. All subjects gave written informed consent and the Wellington Ethics Committee approved the study.

Results

In Study 1 we collected data from 137 participants, comprising 62 welders and 75 non-welders. 96 (70%) of the Study 1 participants (54 welders and 38 non-welders by the pulmonary function analysis definition and 45 welders and 51 non-welders for the respiratory protection analysis) took part in Study 2. Eleven of the Study 2 participants had left the job they held at the time of Study 1. They participated in Study 2 from their homes or in their new workplace. Of these eleven participants, two were on sick leave, one was unemployed, two were retired and six had a new job. Only one participant refused to take part in Study 2, and we were unable to contact this study participant. Of the 51 non-welding women were excluded from the Study 2 sample as there were no female welders in Study 2. In total, there were 41 workers whose data were collected in Study 1 but not Study 2. The mean age and pulmonary function values of these 41 did not differ from the mean age and pulmonary function of the 96 Study 2 participants.

All eight welding sites continued to use mild steel and stainless steel as a base metal, and also continued their use of MIG or TIG welding techniques. Of the eight engineering sites, two had local exhaust ventilation present in both Study 1 and Study 2, one of the engineering sites had no local exhaust present in Study 1 but had installed local exhaust ventilation in part of the site before Study 2, and one site had moved to larger premises where welding was performed in a greater
area. The remaining four sites had no exhaust ventilation or change to the workplace between Study 1 and Study 2.

The personal respiratory protection analysis involved 39 welders, of whom 25 did not have local exhaust ventilation available at either study. Of these, five (12.8%) used respiratory protection in both Study 1 and Study 2, seven (18%) did not have respiratory protection in Study 1 but did in Study 2, and nine (23.1%) did not use respiratory protection at all in Study 1 or Study 2 (Table 2). Thus, local exhaust ventilation had been installed in one site following Study 1, however, the reported use of respiratory protection decreased. None of the 45 welders reported that they had stopped welding because of respiratory symptoms, but three welders (7%) and one non-welder (2%) had stopped welding because of other health problems.

Regarding self-reported respiratory symptoms for both studies, welders had more chronic bronchitis and reported more phlegm production and wheezing compared to non-welders (Table 1). Smoking welders, and welders who worked without local exhaust ventilation or respiratory protection, had an increased risk for accelerated decline in FEV₁ and FVC.

Discussion

This study highlights the problems of achieving improved preventive measures to protect welders exposed to welding plume. The limited use of personal respiratory protection may be explained by the effect of temperature and humidity levels in a respirator on user acceptability during exercise.30 The study of Neilson et al found that respiratory protective devices are used in only 20-30% of the work situations where they are needed, and this was attributed to environmental conditions, physical work demands, psychological and social factors and the individual characteristics of the wearer.30 Such evidence reinforces the importance of local exhaust ventilation as the best protection against the inhalation of welding plume. However, both MIG and TIG welding techniques produce a gas shield around the point of the weld to stop oxidation,21 and this shield is sensitive to air movement and therefore local exhaust ventilation.

The Australian Standard (AS 1763 PT3) provides guidelines to employers of appropriate ventilation for different welding processes and the fumes generated from various metals. The findings of our two year follow up study are consistent with the recommendations of the Australian Standard, Beckett et al2 and Chinn et al,12,13 in that we found that welders without either respiratory protection or local exhaust ventilation in use while welding had a greater annual decline both in FVC and FEV₁ than welders with protection.21

In hindsight, our method of distributing the results of the original study could be improved upon. It could be argued that further workplace feedback might have been more successful in reinforcing the message from Study 1 to workers. The provision of local exhaust ventilation and use of personal respiratory protection in the workplace might have improved if we had reported back to each worker in person. We could have also conducted informal, interactive presentations on the study results with small groups of workers and management.

<table>
<thead>
<tr>
<th>Table 1. Respiratory symptoms reported by welders and non-welders in Study 1 and Study 2.</th>
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<tbody>
<tr>
<td><strong>Study 1</strong></td>
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<tr>
<td><strong>Welders n=62</strong></td>
</tr>
<tr>
<td>Chronic bronchitis</td>
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<tr>
<td>Persistent cough</td>
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<tr>
<td>Work related cough</td>
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<tr>
<td>Chest tightness only with a cold</td>
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<tr>
<td>Work-related chest tightness</td>
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<tr>
<td>Wheezing or whistling in the chest in the last 12 months</td>
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<tr>
<td>Wheeze only with a cold</td>
</tr>
<tr>
<td>Work related wheeze</td>
</tr>
<tr>
<td>Shortness of breath grade 1</td>
</tr>
<tr>
<td>Work-related shortness of breath</td>
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<tr>
<td>Any work-related symptom</td>
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<tr>
<th>Table 2. Presence of local exhaust ventilation and respiratory protection for welders during Study 1 and Study 2.*</th>
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<tr>
<td><strong>STUDY 2</strong></td>
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<tr>
<td><strong>STUDY 1</strong></td>
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<tr>
<td>Local exhaust ventilation</td>
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<tr>
<td>No local exhaust ventilation but respiratory protection</td>
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<td>Respiratory protection</td>
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<tr>
<td>No local exhaust ventilation and no respiratory protection</td>
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<td>Totals</td>
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</table>

*Six welders were excluded from analysis
In conclusion, our two year follow up study found that providing results of the study to the employers and study participants led to little change in behaviour and work environment between Study 1 and Study 2. Distribution of study findings in this case was not a definitive vehicle for change, and further effort is required to ensure that such studies lead to significant changes in work practice and the provision of protective equipment. In the absence of strong external incentives for employers from regulatory bodies, information alone is insufficient to prevent injury. We suggest it might be more effective to spend time with workers and management individually and in small groups, and ask the workers themselves what evidence they need in order to change their work practices.

Acknowledgements. The Wellington Asthma Research Group is funded by a Programme Grant and this study was funded in part from a Limited Budget Grant from the Health Research Council of New Zealand. Dr Riitta Erkinjuntti-Pekkanen also received support from the Yrjö Jahnsson foundation, The Finnish Anti-Tuberculosis Association Foundation and Finnish Cultural Foundation. Dr David Fishwick was part funded by the Finnish Anti-Tuberculosis Association Foundation and Erkinjuntti-Pekkanen also received support from the Yrjö Jahnsson Budget Grant from the Health Research Council of New Zealand. Dr Riitta by a Programme Grant and this study was funded in part from a Limited

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Body Mass Index reference curves derived from a New Zealand Birth Cohort

Sheila Williams, Senior Research Fellow, Department of Preventive and Social Medicine, Dunedin School of Medicine, University of Otago, Dunedin.

Abstract

Aim. To construct reference curves for body mass index (BMI) and estimate the prevalence of obesity or being overweight at ages eighteen and 21 years.

Methods. Smoothed BMI curves were derived from data from a birth cohort born in Dunedin in 1972-73, followed up at two yearly intervals from age three to fifteen years, and then at ages eighteen, 21 and 26 years.

Results. Nine curves 0.67 SD apart, corresponding to the 0.4, 2, 9, 25, 50, 75, 91, 98, 99.6 centiles are provided. The prevalence of obesity, a BMI of 30 kg/m² or more, was 2.7% for men and 2.6% for women at age eighteen years, and 5.8% for men and 5% for women at age 21 years. At age eighteen years, 14.9% men and 16.8% of women were overweight, with a BMI between 25 kg/m² and 30 kg/m². The prevalence increased to 24.7% for men and 24.2% for women at age 21 years.

Conclusion. At age eighteen years, the value of the 98th centile was close to WHO criteria defining obesity in adults. This suggests that it could be used to describe obese children and adolescents. The 75th centile could be used in a similar way to delineate those regarded as overweight.

Obesity is of public concern because of its association with cardiovascular disease, hypertension and diabetes in adults. Obesity in childhood and adolescence is linked with childhood and adolescent disease as well as adult obesity. The most easily obtained measure of adiposity is body mass index (BMI), which has been shown to correlate with total body fat and percent body fat in children and adolescents. In the past, growth charts have been used to assess weight and length in infants and weight and height in children and adolescents as a way of monitoring their growth. One disadvantage of using a weight chart is that no adjustment is made for height, so it is possible for children with the same weight to be too fat or too thin. As BMI, unlike weight, is not strongly correlated with height, reference curves for BMI are useful for appraising adiposity in children and adolescents.
This report uses data from the Dunedin Multidisciplinary Health and Development Study to construct reference charts for BMI for children from the age of three years to adults aged 21 years. It also presents the prevalence of being overweight or obese in 18 and 21 year olds using the WHO guidelines for adults.

**Methods**

The sample was a birth cohort born in Queen Mary Hospital, Dunedin between 1st April 1972 and 31st March 1973. Their mothers were resident in the Dunedin metropolitan area at the time of their birth. Consent to participate in the study was given for 1037 of the 1139 children who were traced, and continued to be resident in the Otago area, at the time of their third birthday. The cohort was seen again at two yearly intervals to age fifteen, and then at ages eighteen, 21 and 26 years, with a systematic sub-sample being seen at ages four and six years. Compared with all New Zealand, the cohort is under representative of Maori and other Polynesians. The study is described in detail elsewhere.

Stature was measured to the nearest millimetre using a portable Harpenden Stadiometer. Weight was recorded to the nearest 0.1 kilogram using a Lindell Beam Balance, the participants being weighed in light clothing. No adjustment has been made for this as it is customary to weigh children in light clothing in clinical practice. Body mass index (BMI) was calculated as weight/height², the units being kg/m². Observations for women known to be pregnant at the time of their age eighteen or 21 year assessment were excluded. As this was one of a number of procedures carried out at the study centre at the time of each assessment, a small amount of data is missing. Weight and height measurements were not always recorded for children assessed outside Dunedin.

The reference or distance curves were derived using the LMS method. This uses a transformation to normalise the data at each age then fits and smooths curves for this (L) for the median (M), and the coefficient of variation (standard deviation/mean) (S) to characterize the changing distribution of BMI. Centile curves two-thirds or 0.67 of a standard deviation apart and correspond to the following centiles: 0.4, 2, 9, 25, 50, 75, 91, 98. As no data were available between birth and age three years, BMI distance curves were constructed for children from age three years onwards. The ages used to construct the growth curves using the LMS method were rounded to one month.

**Results**

The data for males and females from age three to 21 years are shown in Table 1 and 2. These show that BMI declined between the ages three and seven years, before increasing until at least early adulthood. Also shown are the median values for the centiles of interest.

The available data were used to construct distance centile curves (Figures 1 and 2). The curves were 0.67 or two-thirds of a standard deviation apart and correspond to the following centiles: 0.4, 2, 9, 25, 50, 75, 91, 98 and 99.6.

The sample was divided into four groups at ages eighteen and 21 years. The underweight group (BMI less than 18 kg/m²) included ten (2.2%) men and 20 (4.8%) women at age eighteen years and six (1.2%) men and eleven (2.5%) women at age 21 years. The group with a BMI in the normal range (between 18.5 kg/m² and 25 kg/m²) consisted of 361 (80.2%) men and 316 (75.8%) women at age eighteen years and 119 (24.7%) men and 107 (24.2%) women at age 21 years. The obese group (BMI of 30 kg/m² or more) included twelve

---

**Table 1. Means and standard deviations overall, together with medians for specified centiles, for BMI in males.**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Number</th>
<th>Mean</th>
<th>SD</th>
<th>Centiles</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>467</td>
<td>16.3</td>
<td>1.27</td>
<td>14.2</td>
</tr>
<tr>
<td>5</td>
<td>457</td>
<td>16.0</td>
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<td>13.9</td>
</tr>
<tr>
<td>7</td>
<td>450</td>
<td>15.9</td>
<td>1.33</td>
<td>13.7</td>
</tr>
<tr>
<td>9</td>
<td>427</td>
<td>16.1</td>
<td>1.37</td>
<td>13.9</td>
</tr>
<tr>
<td>11</td>
<td>372</td>
<td>17.5</td>
<td>1.25</td>
<td>14.5</td>
</tr>
<tr>
<td>13</td>
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<td>2.14</td>
<td>15.4</td>
</tr>
<tr>
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<td>18</td>
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<td>22.7</td>
<td>3.19</td>
<td>17.9</td>
</tr>
<tr>
<td>21</td>
<td>482</td>
<td>23.9</td>
<td>3.31</td>
<td>19.1</td>
</tr>
</tbody>
</table>

**Table 2. Means and standard deviations overall, together with medians for specified centiles, for BMI in females.**

<table>
<thead>
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<th>Age (years)</th>
<th>Number</th>
<th>Mean</th>
<th>SD</th>
<th>Centiles</th>
</tr>
</thead>
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<td>3</td>
<td>458</td>
<td>16.2</td>
<td>1.31</td>
<td>13.6</td>
</tr>
<tr>
<td>5</td>
<td>455</td>
<td>16.8</td>
<td>1.25</td>
<td>14.1</td>
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<tr>
<td>7</td>
<td>418</td>
<td>15.8</td>
<td>1.39</td>
<td>13.3</td>
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<tr>
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<td>16.4</td>
<td>1.76</td>
<td>13.5</td>
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<td>11</td>
<td>339</td>
<td>17.6</td>
<td>2.22</td>
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<tr>
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<td>355</td>
<td>20.0</td>
<td>2.64</td>
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<tr>
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<td>465</td>
<td>21.0</td>
<td>2.86</td>
<td>16.6</td>
</tr>
<tr>
<td>18</td>
<td>417</td>
<td>22.9</td>
<td>3.18</td>
<td>17.9</td>
</tr>
<tr>
<td>21</td>
<td>442</td>
<td>23.8</td>
<td>3.60</td>
<td>18.5</td>
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</table>
followed by an increase to adulthood, is consistent with the decrease in BMI between age three and seven years, reflecting secular changes.

Discussion

The decrease in BMI between age three and seven years, followed by an increase to adulthood, is consistent with patterns reported in other studies. The nadir, or the age of adiposity rebound, occurred at 6.0 years for boys and 5.6 years for girls. In a comprehensive national survey of the New Zealand population carried out in 1989-1990, the mean BMI for fifteen to eighteen year old males and females was 21.8 kg/m² and 22.3 kg/m² respectively. Those for nineteen to 24 year olds were 22.3 kg/m² and 23.5 kg/m². These were similar to the means for this cohort at the ages of fifteen and eighteen years. By the time the cohort reached the age of eighteen years, it was possible to classify some of them as pre-obese or obese using the recognised WHO cutoffs of 25 kg/m² and 30 kg/m². The prevalence of obesity was similar to the 3% for both age groups for men and the 2% for fifteen to eighteen year old women reported in the national survey, but lower than the 13% reported for nineteen to 24 year old women in the national survey. Those data were collected at the same time as the data for the eighteen year old assessment in this study.

The differences between the mean birth weights in this cohort born in 1972-73 and that of the controls for the New Zealand Cot Death Study born between 1987 and 1990 were small, 73 g for males 29 g for females, those in the birth cohort being smaller. These may be related to secular changes.

These charts, derived from longitudinal data, provide a reference range against which individuals can be compared. It has been argued that cross-sectional data are more suitable for the derivation of growth charts, because reference ranges are cross-sectional in nature. Others have argued that growth curves derived from cross-sectional data provide a snapshot of a reference population and the results do not reflect birth cohort or secular trends which may alter growth trajectories. Comparisons with data from other sources suggest, that at birth and in early adulthood, those in this study were comparable with other New Zealanders. It must be recognised, however, that these centiles may not be suitable for Maori and Pacific people.

Some sort of screening test is implicit in any comparison of an individual with reference ranges such as these. Which centile should be used to delineate those who are too fat? Many studies have used the 85th centile to define the overweight and the 95th to define the obese. The disadvantage of such definitions is that their magnitude depends upon the sample from which they were derived, and secular changes mean that despite increasing levels of obesity the proportion described as overweight or obese remains the same. A recent British study used the 98th centile to define children as obese, and certainly identifying such a small percentage of children in this cohort corresponds quite closely with the prevalence of obesity in the sample, using the WHO guidelines, at ages eighteen and 21 years. The 2nd centile could be used in a similar way to define those who are too thin.

A recent suggestion is that the centile which corresponds to a BMI of 25 kg/m² in early adulthood could be identified and extrapolated backwards into childhood to identify those at risk of being overweight. This corresponded to the 69th centile at age 21 years and the 85th centile at age eighteen years. Elsewhere, it has been shown that using the 75th centile as a threshold in this sample has a positive predictive value of between 50 and 70% for being overweight at age 21 years for seven, eleven and fifteen year olds. Its sensitivity, however, was around 50% (unpublished data). As results from a number of studies show that obesity in adults is related to obesity in childhood, it is tempting to think that by identifying obese or overweight children, measures could be introduced which would prevent them from becoming obese or overweight as adults. However, it is known that childhood obesity is difficult to treat or prevent for more than a few children, and that preoccupation with body size may even be harmful. The BMI growth charts do however provide a standard against which to measure a child or adolescent, which may provide reassurance for many.

Acknowledgements. The Dunedin Multidisciplinary Research and Development Unit and the author are supported by the Health Research Council of New Zealand. The support of the study’s director Dr Phil Silva, the participants and those who collected the data is acknowledged.

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Cardiovascular diseases are the most important of the nutrition-related diseases in affluent countries, and in New Zealand, coronary heart disease (CHD) alone accounts for a quarter of all deaths annually. The national food supply affects the national diet, which in turn affects national health status, and so food supply merits regular review. New Zealand’s food supply has traditionally been high in meat and dairy products, but continues to evolve in response to the increasing influence of ethnic cuisines and health concerns. A high intake of saturated fatty acids (SFA) is associated with high rates of CHD through its impact on cholesterol metabolism, predisposing to CHD, and the fat content of the food supply remains high, predisposing to obesity. Continued efforts are needed to improve the diet of New Zealanders and to maintain food supply data collection for long term monitoring of these changes.

The New Zealand food supply and diet - trends 1961-95 and comparison with other OECD countries

Murray Laugesen, Public Health Physician, Health New Zealand, Boyd Swinburn, Medical Director, National Heart Foundation, Auckland.

Abstract

Aims. To compare the New Zealand food supply and trends from 1961 to 1995 with other Organisation for Economic Co-operation and Development (OECD) countries, with an emphasis on foods linked with coronary heart disease (CHD).

Method. Food and Agricultural Organization per capita food supply statistics for 24 OECD countries were converted to nutritional supply values and adjusted for edible portion.

Results. In 1995, New Zealand had the highest supply per capita of butter and meat fats among OECD countries, ranking its food supply highest for thrombogenicity and third for atherogenicity. Seafood and alcohol supply were average and vitamin E supply was high compared with other OECD countries. Beneficial trends have occurred with increases in fruit consumption, vegetable consumption and fibre intake between 1961 and 1995. While total fat intake has not changed appreciably, the fatty acid profile has shifted and is now less likely to promote CHD.

Conclusions. The New Zealand diet’s tendency to promote CHD has decreased, particularly since 1985. The diet’s fatty acid profile, however, remains highly atherogenic and thrombogenic, predisposing to CHD, and the fat content of the food supply remains high, predisposing to obesity. Continued efforts are needed to improve the diet of New Zealanders and to maintain food supply data collection for long term monitoring of these changes.

Cardiovascular diseases are the most important of the nutrition-related diseases in affluent countries, and in New Zealand, coronary heart disease (CHD) alone accounts for a quarter of all deaths annually. The national food supply affects the national diet, which in turn affects national health status, and so food supply merits regular review. New Zealand’s food supply has traditionally been high in meat and dairy products, but continues to evolve in response to the increasing influence of ethnic cuisines and health concerns. A high intake of saturated fatty acids (SFA) is associated with high rates of CHD through its impact on cholesterol metabolism, predisposing to CHD, and the fat content of the food supply remains high, predisposing to obesity. Continued efforts are needed to improve the diet of New Zealanders and to maintain food supply data collection for long term monitoring of these changes.

The methods of analysis were similar to those of a previous study, except that the present study used FAO FBS data rather than OECD data. The FBS data, from the FAO website, as of November 1998, represented the food available per capita at the consumer level for 77 food groups and five alcoholic beverages in kilograms per person per year for the years 1961, 1965, 1970, 1975, 1980, 1985, 1990 and 1995. FAO values were used to calculate energy, fats and protein composition of foods. Other nutritive values and dressed meat carcass composition were from UK data.

FAO food balance sheets are based on the food balance principle that for each food group, food supply (production, including an allowance for home production, imports minus exports + stock changes) equals food utilisation (feed + seed + food manufacturing + waste + non-food uses + home production + imports minus exports + stock changes). Food utilisation data can be obtained from the FAO website.
and oils were rated as 100% edible, tallow as 80%. Energy from alcoholic beverages (alcohol and sugar) was then added to the food data. Alcohol content was rated as: 11% by volume for wine, 40% for spirits, 3.5% for beer, 5% for fruit-based beverages and 15% for sake.14 A typical food was selected to characterise each of the food groups. To calculate daily per capita nutrient supplied, the weight in grams of each daily per capita food group was multiplied by its edible fraction and the nutrient content per gram of the food typifying that food group, for each of 30 nutrients. Fat content for all foods was analysed by type (SFA, MUFA, PUFA) and, in the case of SFA, by carbon chain length.12 Overall 110 nutritional descriptors for the per capita food supply of each country by year were calculated, for example, myristic acid in dairy products. FAO nutritional descriptors for the per capita food supply of each country by OECD countries in 1995. Significant declines in both indices were not evident until 1985-1995 when they fell by 17% (AI) and 11% (TI).16 In general, SFAs increase the propensity for fatty acids to increase or decrease serum cholesterol, and for those not listed, the average composition of a wide range of such vegetables or fruits was used to calculate nutrient composition of the unspecified fruit or vegetable. Meat supply in the FAO FBS was based on dressed carcass weight, published edibility factors13 and the FAO energy, fat and protein values for beef-beef, pork, mutton and poultry meat. The propensity for fatty acids to increase or decrease serum cholesterol has been incorporated into an Atherogenicity Index (AI) by Ullrich and Southgate.13 Similarly, the propensity to increase or decrease thrombosis has been incorporated into the Thrombogenicity Index (TI).16 Higher values for AI and TI infer a greater risk of the diet promoting CHD.

The nutritional composition of each edible food was assumed to be uniform across regions and years. Due to lack of data, it could be made for leaner carcasses in recent decades due to changes in breeding and farming practices. Cheese was assumed to be from cow’s milk, comprising 31% fat except for Greece, where cheese comes mainly from sheep and goat’s milk and the fat content was estimated at 19.6%.12 No allowance was made for vitamins or minerals lost in storage or cooking, or better preserved in recent years with refrigeration or microwave cooking, or added artificially, or as self-medication with dietary supplements. The FBS did not provide data on dietary salt. Since food supply means the amount of food made available to consumers, no allowance was made for waste beyond this point, whether due to deterioration in storage, waste in preparation, or food discarded or used as pet food.

The New Zealand food supply remained high in fat, varying between 36.7 and 39.5% of energy with no discernable trends over time. New Zealand ranked near the middle of OECD countries, with Belgium having the highest fat content (43%) and Japan (26%) and Mexico (25%) the lowest. Carbohydrate contributed 48% to total energy, for which New Zealand was also ranked near the middle. Apart from Mexico, only in the high fish-supply countries of Japan, Norway and Iceland did carbohydrate provide more than half the total energy.

The contribution of SFAs to the fat supply declined little from 1965 (53%) to 50% in 1985, then declined steeply to 45% in 1995. Despite this decline, New Zealand still ranked fourth for SFA supply in 1995. The contribution of MUFAs to the energy supply increased only slightly from 1961 to 1995, despite an eight-fold increase in oil supply. This was largely because 81% of New Zealand’s MUFA supply came from animal sources, with vegetable oils contributing only 7g of the 57 g/capita/day of MUFA in the 1995 food supply. Also, increases in vegetable MUFA supply have been offset by decreases in the supply from dairy fat. The contribution of PUFAs to the energy supply increased between 1975 and 1995, but in 1995 accounted for only 6% of energy (24 g/capita/day), ranking New Zealand 21st out of 24 OECD countries. In 1995, seafood fats and oils contributed only 0.06% of food energy in the New Zealand food supply, whereas in Japan, seafood fats and oils contributed ten times this percentage.

Atherogenic and thrombogenic indices. The fatty acid pattern of the diet can be summarised as two indices, indicating the propensity of the diet to promote atherosclerosis (Atherogenic Index or AI) and thrombosis (Thrombogenic Index or TI).16 In general, SFAs increase the indices and PUFAs and MUFAs decrease them, so the pattern is similar to the trends described above (Table 4). Significant declines in both indices were not evident until 1985-1995 when they fell by 17% (AI) and 11% (TI). However, in 1995, the New Zealand food supply still remained extremely highly ranked for AI (3rd) and TI (1st). In contrast, Australia ranked eighth and seventh respectively. In 1995, the New Zealand food supply had the highest content of stearic acid (mainly from dairy fats, beef fat, and...
sheep fat), which is believed to increase thrombogenesis, and was third highest for myristic acid (mainly from dairy fat), which raises serum cholesterol.

Vitamin E, dietary fibre, and alcohol. The New Zealand food supply ranked sixth highest among OECD countries for Vitamin E supply, while for dietary fibre, it ranked thirteenth (Table 4). Vitamin E supply has changed little over the years. In 1995, the main sources of Vitamin E were vegetable oils (34%), vegetables and fruit (21%) and wheat (20%). Dietary fibre increased somewhat from 1970 onwards. In 1995, the main sources were cereals (60%), and vegetables and fruit (28%).

Trends in alcohol supply (Tables 1, 3, 4) showed a large increase from 1961, peaking in the 1975-1985 decade and followed by a moderate decrease to 1995. Beer provided 64% of the 1995 New Zealand alcohol supply. Alcohol at 14.5 g/capita/day ranked New Zealand twelfth out of 24 OECD countries, and fifteenth as a fraction of dietary energy.

Discussion
These FAO food balance data from 1961-1995 show two overall patterns. First, the New Zealand food supply has

<table>
<thead>
<tr>
<th>Source</th>
<th>Saturated fat (g/capita/day)</th>
<th>Saturated fat (% of total)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total in dairy products</td>
<td>24.2</td>
<td>36</td>
</tr>
<tr>
<td>in butterfat</td>
<td>12.4</td>
<td>18</td>
</tr>
<tr>
<td>in milkfat, cream</td>
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<td>11</td>
</tr>
<tr>
<td>in cheese</td>
<td>4.5</td>
<td>7</td>
</tr>
<tr>
<td>Total in meat</td>
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<td>48</td>
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<tr>
<td>in beef</td>
<td>7.1</td>
<td>11</td>
</tr>
<tr>
<td>in sheep meat</td>
<td>9.8</td>
<td>15</td>
</tr>
<tr>
<td>in pig meat</td>
<td>3.5</td>
<td>5</td>
</tr>
<tr>
<td>in poultry</td>
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<td>5</td>
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<td>in separated fats</td>
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<td>11</td>
</tr>
<tr>
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</tr>
<tr>
<td>Eggs</td>
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<td>2</td>
</tr>
<tr>
<td>Cereals</td>
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</tr>
<tr>
<td>Fruit</td>
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<td>0.5</td>
</tr>
<tr>
<td>Vegetables</td>
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<td>0.2</td>
</tr>
<tr>
<td>Seafood</td>
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<tr>
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<td>0.3</td>
</tr>
<tr>
<td>Total</td>
<td>65.3</td>
<td>100</td>
</tr>
</tbody>
</table>

*Separate fat includes slaughter fats, butcheted fats, offal, and fat in other meats such as game. †Energy from all animal products including dairy products and butter, meat, offal, separated fats, seafood, eggs, as % of total diet energy.

Table 3. Comparison of the supply of selected foods by various sources.

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<thead>
<tr>
<th></th>
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<th></th>
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<tbody>
<tr>
<td>Butterfat (kg/capita/year)</td>
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<tr>
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<td>10.3</td>
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<td>9.9</td>
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<td>Beef (kg/capita/year)§</td>
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<td>Sheep meat (kg/capita/year)§</td>
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<td>31.2</td>
<td>26.2</td>
<td>22.7</td>
<td>25.2</td>
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<td>33.0</td>
<td>25.5</td>
<td>25.8</td>
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<tr>
<td>Alcohol (L/capita/year)</td>
<td></td>
<td></td>
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<td>FAO</td>
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<td>5.8</td>
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</tbody>
</table>

*Food and Agriculture Organisation. †1993 data, 1995 not published. ‡International Dairy Federation. §Beef and sheep meat represent edible meat (carcass-derived weight x edibility factor of 0.82 and 0.83 respectively) excluding separable fats and offal. NA=not available.
improved significantly with respect to risk for CHD, particularly over the 1985-1995 period. Second, despite these improvements, the food supply still remains highly atherogenic and thrombogenic by OECD standards because of the continuing high contribution of dairy and meat fats.

The New Zealand fat supply generally improved more between 1985 and 1993 than in previous decades, with decreases in butterfat and increases in vegetable oil improving the fatty acid profile of the diet. These changes paralleled changes in mean serum cholesterol observed in the Auckland Heart and Health Surveys, where the decrease from between 1982 and 1987 was 0.07 mmol/L, the Auckland Heart and Health Surveys, where the decline in CHD mortality.

Health promotion programmes may have stimulated some of these improvements. Between 1985 and 1995, the Heart Foundation began its Heartbeat programme in schools, workplaces and communities and its “Pick the Tick” food labelling programme. The Cancer Society’s Fit Food campaigns and the 5+ a day campaigns highlighted the benefits of a diet high in fruit and vegetables. Also, manufacturers and supermarkets have provided an increasing range of healthy food choices, while ethnic foods and dishes have diversified the diet. Freer trade with other countries also.

The 1995 OECD rankings indicate that, despite these positives changes, the New Zealand food supply has a long way to go before it could be considered ‘heart healthy’. Having the most thrombogenic and third most atherogenic food supply shows the magnitude of the change needed to achieve a national diet even as heart healthy as Australia, the US or UK, let alone comparable to Japan or the Mediterranean countries. Meat and dairy products dominate plant-based products in the New Zealand food supply and New Zealand appears to be amongst the least vegetarian in the OECD (second ranked for energy from animal sources) and possibly the world. If the 1985-95 rate of change in the food supply continues to accelerate, New Zealand may gain ground on its fellow OECD countries, but similar improvements are occurring in many of these countries also.

The lack of decreases (or even possible increases) in the total energy and fat supply do not bode well for the current epidemic of obesity. Over the last 30 years, energy expenditure has continued to decline, due mainly to the marked increase in labour-saving machines for home, work and transport. In future, these trends are likely to continue, and will need to be matched by increased recreational activity and decreased fat and energy consumption, if the rising prevalence of obesity is to be reversed.

The data in this paper have certain limitations, in addition to those outlined in the methods section. First, the 33% decrease in dairy fat supply for 1985-95 requires cautious interpretation, due to the increased food products trade between Australia and New Zealand. If butterfat or cream is manufactured into exported products (such as biscuits or ice cream), they count as locally consumed. Conversely, butterfat in imported biscuits is not counted as locally consumed. Second, the increase in meat fat supply between 1990 and 1995 is likely to be due to a change in estimation method. Prior to 1990, separated fats were calculated as a percentage of beef carcass weight, but since 1990, it has come from surveys of abattoirs, with higher values reported. When separated fats in 1995 were calculated from the same percentage of beef carcass weight, but since 1990, it has come from surveys of abattoirs, with higher values reported. When separated fats in 1995 were calculated from the same ratio to beef as in 1980 and 1985, the Table 1 1990-95 increase in total meat fat disappeared. Nevertheless, by either method, New Zealand’s meat fat supply ranked first or first equal among OECD countries. Thirdly, more fat was probably cut off meat by butchers and consumers in the 1990s than in previous decades. Although some of this trimmed fat may re-enter the food chain (as in processed meats or frying fats), an increase in fat discarded is not detected by food balances. Further research is needed to quantify the leanness of meat sold to consumers, the fate of fat trimmed commercially and household wastage.

This paper raises the need to have reliable national data on food supply for monitoring and policy development. Statistics New Zealand discontinued its collection of food balance sheets in 1997. This severely jeopardises the ability of the Ministry of Health, the food industry and public health groups to monitor trends in food supply and comparisons with other countries. The collection of FBS data needs to be re-instituted and backed up with additional research on its underlying assumptions.


<table>
<thead>
<tr>
<th>Year</th>
<th>Total energy (kcal/day)</th>
<th>Fats (% diet energy)</th>
<th>Diets cholesterol (mg/1000 kcal)</th>
<th>Atherogenicity index</th>
<th>Thrombogenicity index</th>
<th>Vitamin E (mg/1000 kcal)</th>
<th>Total fibre (g/day)</th>
<th>Alcohol (% diet energy)</th>
</tr>
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<tbody>
<tr>
<td>1961</td>
<td>3190</td>
<td>35.8</td>
<td>180</td>
<td>1.2</td>
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<td>2.5</td>
</tr>
<tr>
<td>1965</td>
<td>3300</td>
<td>36.0</td>
<td>184</td>
<td>1.2</td>
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<td>2.5</td>
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<tr>
<td>1970</td>
<td>3255</td>
<td>35.9</td>
<td>187</td>
<td>1.2</td>
<td>1.9</td>
<td>2.5</td>
<td>27</td>
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<tr>
<td>1975</td>
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<td>36.9</td>
<td>187</td>
<td>1.1</td>
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<td>2.3</td>
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<td>0.9</td>
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<td>2.3</td>
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<td>1990</td>
<td>3478</td>
<td>37.3</td>
<td>161</td>
<td>0.9</td>
<td>1.5</td>
<td>2.3</td>
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<td>1995</td>
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<td>147</td>
<td>0.9</td>
<td>1.5</td>
<td>2.3</td>
<td>34</td>
<td>2.8</td>
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<tr>
<td>1985-95 change</td>
<td>-1%</td>
<td>-11%</td>
<td>-16%</td>
<td>-17%</td>
<td>-11%</td>
<td>5%</td>
<td>24%</td>
<td>-20%</td>
</tr>
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</table>
In conclusion, the data presented here suggest that the New Zealand diet has improved significantly over the last fifteen years, but despite this, it remains one of the least heart healthy of all OECD countries. A continued multi-sectoral effort is needed to reduce the country’s burden of CHD and obesity through improvements in the national diet.

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IN PRACTICE

“Herbal Ecstasy”: a case series of adverse reactions

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Abstract

Aims. To report five cases of adverse effects associated with the ingestion of “Herbal Ecstasy” tablets and discuss possible mechanisms of toxicity.
Method. Composition of the “Herbal Ecstasy” tablets was determined by discussions with distributors and with Medsafe. Reference to relevant texts and an internet and Medline literature search was used to identify articles of interest.
Results. Three patients complained of minor symptoms such as perceptual disturbances, anorexia, inability to sleep, dizziness, palpitations and paresthesia. A fourth patient presented with palpitations and ventricular bigeminy, and a fifth patient presented with headache, vomiting, and a hypertensive crisis. Different brands of “Herbal Ecstasy” have different compositions, some containing caffeine and kava, and one contained ephedrine.

Conclusion. These five patients presented with adverse reactions to “Herbal Ecstasy” ranging from minor to major. Knowledge of the possible mechanisms of toxicity will help plan therapy in cases of major toxicity.

“Herbal Ecstasy” is a general term for a number of different herbal preparations. In New Zealand, some of the names for these include “The Bomb”, “Reds” and “Sublime”. The preparations are advertised as stimulants and have been legally available in selected stores and over the internet. “The Bomb” was recently taken off the market in New Zealand after investigation by the Ministry of Health showed that it contained substantial amounts of ephedrine.1

Four cases of toxicity from “Herbal Ecstasy” which occurred in conjunction with a music festival in January 1999, and one case from June 1999 are reported. The composition of preparations available in New Zealand, and possible mechanisms of toxicity are discussed.

Case reports
Case 1. An 18 year old male presented complaining of itchy arms and alterations to smell and taste. He had ingested one “Herbal Ecstasy” tablet and two bottles of “Red Bull” energy drink nine hours previously. He was afebrile with a pulse rate of 70/min and blood pressure of 120/70 mmHg. He was discharged with no treatment.
Case 2. A 17 year old male presented with a headache, loss of appetite and inability to sleep. He had ingested four tablets of “Herbal Ecstasy” 24 hours prior to presentation. He was alert and orientated with a pulse rate of 72/min and blood pressure of 130/85 mmHg. He was given paracetamol, observed and discharged.
Case 3. A 17 year old female presented with dizziness, palpitations, inability to sleep and tingling in the fingertips. She had ingested three “Herbal Ecstasy” tablets the previous night and two “V” drinks the morning of her presentation. She had no significant past history and was on no prescribed medications. She was afebrile, anxious and tachycardic with a pulse rate of 120/min and blood pressure of 147/87 mmHg. There was a soft systolic murmur at the left sternal edge. The ECG showed a sinus arrhythmia with the heart rate...
of autonomic hyper-reflexia can include hypertension, pyelonephritis, uterine contractions and surgery. Manifestations bowel or intestinal distention, hot or cold skin stimulation, previously reported as causing the response have been bladder, influence on the thoracolumbar sympathetic outflow.2 Stimuli seen in 65-85% of patients with a spinal cord injury above T7, autonomic hyper-reflexia response. Autonomic hyper-reflexia is associated with a hypertensive crisis and encephalopathy, 5, and a description of what was probably reflex bradycardia, within half an hour of ingestion of “The Bomb” tablets in Case 4.

Case 4. A 16 year old male presented to the Emergency Department with palpitations and agitation. His symptoms had started three hours after ingesting alcohol and “Herbal Ecstasy” tablets. He had no significant past history and took no medications. He was alert and orientated with a pulse rate of 110/min and blood pressure of 150/60 mmHg. He was afibrile and not dyspnoeic. The initial ECG showed ventricular bigeminy at 118/min, but no ischaemic changes. A chest x-ray and blood tests including a full blood count, urea, electrolytes, arterial blood gas, clotting screen, paracetamol/ salicylate screen and troponin T were normal. An ethanol level was 4 mmol/L. Creatinine kinase was elevated at 511 U/L (normal range 60-220). The patient was commenced on 40% oxygen by mask and given one litre of normal saline intravenously. His agitation settled within two hours and a repeat ECG showed ventricular trigeminy at a rate of 93/min. The patient was discharged four hours after presentation.

Case 5. A 28 year old man presented to the Emergency Department complaining of severe headache associated with vomiting and photophobia. He had ingested four tablets called “The Bomb” three hours prior to presentation. Half an hour after ingesting the tablets he felt unwell, developed a mild headache and noticed his pulse slowing. Past history included a T6 paraplegia following a motorcycle crash eight years previously. He opened his eyes to voice and was disorientated, with an initial pulse rate of 50/min and a blood pressure of 210/120 mmHg. He was afibrile but mildly dyspnoeic. Neurological examination revealed terminal neck stiffness and T6 paraplegia, but normal power in both arms. An ECG was normal. The patient was given intravenous metoclopramide and morphine followed by intravenous labetalol 50 mg. His blood pressure decreased to 154/111, then to 130/68 mmHg. Blood tests, including a full blood count, urea, electrolytes, glucose and amylase were normal. Computerised tomography of his head showed no subarachnoid or intracranial hemorrhage. He was observed overnight and remained normotensive. When reviewed in the morning, he had a mild headache, but the terminal neck stiffness had resolved and he was discharged.

Discussion
The symptoms experienced by Cases 1, 2 and 3 were mild and resolved with simple or no treatment. There are many possible causes of the symptoms, including sleep deprivation, which many people at the music festival experienced, so it is difficult to attribute the symptoms solely to “Herbal Ecstasy”. The palpitations and agitation experienced by Case 4 appeared soon after the ingestion of “Herbal Ecstasy” tablets, which almost certainly caused the ventricular bigeminy. As the troponin T was normal, it is unlikely that the raised creatinine kinase was of cardiac origin, but was most likely due to increased muscle activity associated with his agitation. The onset of headache within half an hour of ingestion of “The Bomb” tablets in Case 5, and a description of what was probably reflex bradycardia, associated with a hypertensive crisis and encephalopathy, suggests a strong alpha adrenergic effect, or the triggering of an autonomic hyper-reflexia response. Autonomic hyper-reflexia is seen in 65-85% of patients with a spinal cord injury above T7, and is associated with the absence of supraspinal inhibitory influence on the thoracolumbar sympathetic outflow.2 Stimuli previously reported as causing the response have been bladder, bowel or intestinal distention, hot or cold skin stimulation, pyelonephritis, uterine contractions and surgery. Manifestations of autonomic hyper-reflexia can include hypertension, bradycardia, dysrhythmias, headache, nasal congestion, blurred vision, sweating, vasoconstriction, convulsions, cerebral haemorrhage and death.2 It is difficult to know which component of “The Bomb” triggered this response, although ephedrine containing herbs have been associated with cardiovascular toxicity in the past (see below). The patient responded well to therapy and sustained no permanent adverse effects.

“The Bomb” contained Bringham tea (12.7 mg ephedrine per capsule), Fo-ti, Astralgus, Rehmannia, licorice root and Lycii berries. It has been withdrawn from the market and is being reformulated to exclude the herb containing ephedrine. “Reds” contains amino acids, kava, caffeine (100 mg), inositol, and vitamins C, B6 and B1. “Sublime” contains kava, sasparilla root, gotu kola root, amino acids, caffeine (40 mg), inositol (15 mg), and vitamins B6 & B1. Information on some of the components of “Herbal Ecstasy” tablets is not readily available.

Cardiovascular toxicity has been reported after ingestion of herbal preparations containing caffeine and ephedrine.1 In New Zealand, substances containing ephedrine are deemed medicines and thus are subject to the relevant controls, but may be freely available in other countries. The Chinese herb Ma Huang, or Ephedra, is known to contain ephedrine, norephedrine, pseudoephedrine and similar substances, and mania, psychosis and hepatitis have been reported following its use.1 Stroke and intracranial haemorrhage have also been reported following ephedrine ingestion.1 Ephedrine has both alpha- and beta-adrenergic effects,2 and toxicity is the result of over-stimulation of the adrenergic receptors. Major toxic effects include: hypertension, tachyarrhythmias, myocardial ischaemia, intracerebral haemorrhage, seizures, manic conditions and psychosis. Less serious effects include: headache, dilated pupils, nausea, vomiting, anxiety, restlessness, giddiness, sweating, muscular weakness and tremors.1,2 Management of toxicity includes supportive cares and decontamination with activated charcoal in severe poisonings, but caution with gastric lavage in the presence of hypertension, as this can increase intracranial pressure.4 For severe hypertension, titrated intravenous sodium nitroprusside is the quickest and safest means of reducing blood pressure, but intravenous labetalol can be used, however additional agents may be required.4 Patients with agitation and psychosis should be watched for hyperthermia, and are best treated with benzodiazepines, as antispsychotics can worsen cardiovascular toxicity.4

Many preparations include caffeine or caffeine sources such as guarana and kola nut. One cup of coffee contains 30 to 200 mg of caffeine, and caffeine toxicity can occur after a dose of as little as 50 mg, but significant toxicity usually occurs at doses more than 15mg/kg, with the lethal dose being more than 100 mg/kg.7 Toxic effects can include: anxiety, agitation, confusion, psychosis, vomiting, myoclonus, seizures, pulmonary oedema, rhabdomyolysis, metabolic disturbances and cardiac arrhythmias such as sinus tachycardia, paroxysmal supra-ventricular tachyarrhythmias or ventricular dysrhythmias.1,7 Management is mostly supportive: decontamination with activated charcoal in an early presentation, rehydration, correction of electrolyte abnormalities, antiemetics, benzodiazepines for seizures, and standard therapy for arrhythmias.1

One preparation contained licorice root (Glycyrrhiza glabra) which has mineralocorticoid activity, so excessive ingestion can cause symptoms of sodium retention (hypertension, dyspnoea, peripheral oedema) and hypokalaemia (lethargy, muscle cramps, paraesthesia and tetany).8 Kava (Piper methysticum), which has sedative properties, is in some preparations and inebriation is the main potential toxicity.9

In summary, five patients presented with adverse reactions to “Herbal Ecstasy” ranging from minor to major. Different brands of “Herbal Ecstasy” have different compositions. Most
patients responded to general and supportive therapy, and the patient with the hypertensive crisis responded to intravenous labetalol. Knowledge of the possible mechanisms of toxicity will help plan therapy in cases of major toxicity. It is important to ask about herbal alternative medicines when taking a drug history, and any adverse reactions should be reported to the Centre for Adverse Reactions Monitoring.

Acknowledgements. Thanks to Peter Pratt of the Medsafe Compliance Team for information on the analysis of “The Bomb”, and thanks to Chris Fowlie from the Hemp Store and to Mareeol from Beds for information about product components.

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VOCATIONAL DRIVING, DIABETES AND INSULIN USE

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NZ Med J 2000; 113: 313.5

Medical conditions can impact on the ability to drive and the risk of drivers having crashes, but it is difficult to obtain precise estimates of the risks. Failure to declare medical conditions, the difficulties in ascertaining their role in crashes, together with a lack of any registry for such data means that reliable evidence is hard to come by. This is not a problem unique to New Zealand, so overseas data cannot fill this gap. Here, as elsewhere, the regulations regarding driving and medical conditions are essentially pragmatic. In 1999, a new system of licensing for drivers was introduced in New Zealand. As a result of the changes to the section of the regulations, the most obvious driving risk for hypoglycaemic drivers tends to underestimate the degree to which their driving is impaired.

One of the important changes in the new regulations concerns vocational drivers with diabetes who use insulin. For the first time, a differentiation is made between drivers of commercial vehicles with type 2 (non-insulin dependent) diabetes who use insulin to control their diabetes and those with type 1 (insulin dependent) diabetes. Drivers of commercial vehicles with type 1 diabetes are permitted to drive Class 1 vehicles (up to 4500 kg gross laden weight), although they will not be granted the endorsements to carry passengers, but insulin using drivers of commercial vehicles who have type 2 diabetes can drive vehicles of any category, providing they do not have a history of severe hypoglycaemia, and providing certain medical and work conditions are adhered to. The conditions of work include limited driving hours and regular meal breaks. Medical conditions include: three monthly certification of continued compliance with treatment, adequate glycaemic control and the absence of severe hypoglycaemia and significant diabetes complications. These conditions are more stringent than those imposed on type 2 diabetes subjects who are on sulphonylurea drugs in whom six monthly certification is required.

Apart from visual impairment due to retinopathy (or its treatment) and cataract, which are covered in a separate section of the regulations, the most obvious driving risk for people with diabetes is hypoglycaemia. There is absolutely no doubt that hypoglycaemia impairs driving performance. Experiments with driving simulators in subjects with type 1 diabetes have demonstrated that subjective impairment of driving performance may be perceived when the blood glucose is as high as 3.6 mmol/L. Between 3.6 and 2.6 mmol/L, objective assessments of driving performance decline markedly. Importantly, this decline in objective driving performance is much greater in magnitude than the subjective - so people may believe that they are driving better than they really are. Below 2.6 mmol/L, driving ability is severely impaired. As with alcohol, hypoglycaemic drivers tend to underestimate the degree to which their driving is impaired.

Hypoglycaemia, whilst it can occur in patients taking sulphonylurea drugs, is most likely to occur in subjects taking insulin. Hence, in most countries where regulations are imposed, it is the insulin users who are subject to restrictions. The particular question of whether vocational driving should be permitted by insulin users has generated the greatest debate - and different countries have come to widely differing conclusions. Vocational drivers inevitably have a greater risk of a crash - they are on the road for more hours - and the nature of their vehicles (larger size) and sometimes their cargo (passengers or dangerous goods), means that there is the potential for more damage when they do crash.

Exactly how big is the risk, and how does it compare to other risks that society is prepared to tolerate on the roads in order to maintain mobility and convenience? This question has been addressed by Lave, Songer and La Porte, who calculated from the data available to them the risk of severe hypoglycaemia in insulin users, and compared this in magnitude to other risks. They estimated that the relative risk of a crash for commercial motor vehicle drivers who used insulin was increased 3.7 fold for type 1 diabetes and 2.7 fold for type 2 (these estimates excluded those who had a history of severe hypoglycaemia in whom the relative risk of a crash was increased nearly twenty fold). They demonstrated that this level of risk is well within that currently tolerated by society (for example, the risk of sixteen year old males having a crash is 42 times greater than that of 40 year old women).
In these exercises, Lave, Songer and La Porte had fairly limited data available to assess the risk of severe hypoglycaemia in subjects with type 2 diabetes who were treated with insulin. They concluded that it was probably half the rate of that in subjects with type 1 diabetes. However, data published more recently suggest that the risk is substantially lower in subjects with type 2 diabetes. In the UK Prospective Diabetes Study Group (UKPDS) study, subjects with type 2 diabetes having insulin therapy and maintaining an HbA1c level between 6.0 and 7.8% had one twentieth the rate of severe hypoglycaemia of subjects with type 1 diabetes in the intensively treated arm of the Diabetes Control and Complications Trial Research Group (DCCT) trial who maintained a mean HbA1c of 7.1%. The differentiation between insulin using subjects with type 2 diabetes and those with type 1 diabetes in the 1999 New Zealand regulations, reflects a welcome acknowledgement that the former are at much lower risk of severe hypoglycaemia.

In deciding these matters, governments have to balance public safety with personal choice and fairness. The Land Transport Safety Authority is to be congratulated on its flexible and fair minded approach in taking this liberalising step, which contrasts with developments in the United Kingdom, where recent regulatory changes seem to have been driven by administrative simplicity. There, new regulations are now distinctly illiberal, with a blanket ban on any insulin user driving a vehicle of over 3500kg gross laden weight. Although there are still anomalies in the New Zealand regulations, they do represent a significant step forward for people with diabetes, particularly for those vocational drivers with type 2 diabetes who need to use insulin, who no longer have to choose between preserving their health and their job. Exactly how many people will benefit immediately from the new regulations is unknown, but with the prevalence of type 2 diabetes ever increasing, and 5-7% of subjects each year needing to go on to insulin, the numbers will certainly increase.

Regulations about driving and diabetes will continue to evolve, it is to be hoped in the light of evidence and reliable statistics. With regard to the latter, the Land Transport Safety Authority does require that all drivers of classes 2-5 vehicles provide information about their diabetes, so it should be possible to determine whether the frequency of crashes in insulin using vocational drivers exceeds that of non-diabetic drivers. In the future, it is to be hoped that an evidence based system can be developed whereby individual cases can be considered on their merits. Whilst this is the ideal, it has the potential for abuse if patients are able to ‘shop around’ for a favourable medical report. Any such system mandates a high degree of consistency in its application.

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**Managed care in America: lessons for New Zealand**

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The 1990s have seen a fundamental shift in the way that health care is organised, financed, and delivered in the USA. Managed care in its many forms has become the dominant system for providing personal health care. An imprecise term ‘managed care’ encompasses a range of strategies, which aim primarily to control the cost of health care, but also impact on its quality. Most developed countries are struggling with this tension between cost and quality, and countries like New Zealand are looking increasingly to America and managed care. Managed care has potential for both benefit and harm. The purpose of this article is to tease out the strengths and weaknesses in the way that managed care is practised in the USA, and look at the lessons for New Zealand.

**Managed care**

**What it is and what it isn’t.** Managed care is a system that takes responsibility for the health of circumscribed enrolled populations, and attempts to modify the behaviour of doctors, and patients (Table 1). It is not a single homogeneous entity, in fact there are so many variants in the US, that it has been described as an “unintelligible alphabet soup of three letter health plans.” Some commentators use Health Maintenance Organisation (HMO) and managed care interchangeably, though HMOs are but one form of managed care. Likewise, ‘integrated care’ has been used but integration may, or may not be a feature. A taxonomy of Managed Care Organisations (MCOs) is presented in Table 2. A further development has been the rise in plans that operate on a ‘for-profit’ basis. Membership in such plans increased by 91.6% between 1984 and 1994, meaning that in 1994 close to 60% of HMO members were in ‘for-profit’ plans.

<table>
<thead>
<tr>
<th>Table 1. Fundamental features of an archetypal managed care organisation.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Organised system responsible for financing and delivery of care.</td>
</tr>
<tr>
<td>2. Enrolled population.</td>
</tr>
<tr>
<td>3. Focus on prevention/population perspective.</td>
</tr>
<tr>
<td>4. Pre-paid fixed premium per member, regardless of actual services used.</td>
</tr>
<tr>
<td>5. Varying degrees of financial risk for providers, possibly through capitation, withholds (withholding of payment if provider utilisation patterns are outside the organisations’ norm), and bonuses.</td>
</tr>
<tr>
<td>7. Defined services/benefit package.</td>
</tr>
<tr>
<td>8. Minimal financial barriers to access.</td>
</tr>
<tr>
<td>10. Quality management systems.</td>
</tr>
</tbody>
</table>

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2. JF Howard, Land Transport Safety Authority. (personal communication).
Managed care techniques. The MCO attempts to ‘manage care’ by influencing provider patient behaviour using an array of methods (Table 3). The financial incentives to encourage doctors to change their practice, and for patients to restrict their choice of providers are the most controversial. Provider behaviour can be influenced by the use of bonuses, paid if certain quality parameters are met (for example, immunisation and screening rates), or conversely, income can be withheld if a provider is an outlier compared to the average, with respect to his or her utilisation patterns (provider profiling).

Disadvantages of the managed care approach
These can be divided into those inherent in managed care, and those that are the product of the American medical system and societal values.

Inherent disadvantages. The first concern is the effect of managed care on the doctor/patient relationship. This relationship is fundamentally about trust. In the managed care situation, the doctor may be seen as a double agent serving not only the individual patient, but also the MCO and enrollees. There is some evidence that patients’ trust in their doctor is eroded if they believe that the doctor is paid under a capitated arrangement. However, the relationship between method of payment and trust, appears to be mitigated by the interpersonal skills of the doctor. Furthermore potential conflicts of interest are inherent in all payment systems. The financial interests of the doctor was a factor with fee-for-service, encouraging over-servicing, sometimes to the detriment of individual patients, and often to the wider community, through wide practice variation and inappropriate care.

The second concern is that cost and not quality has been managed. It is easier to keep costs down by excluding chronically ill or expensive to treat patients (adverse risk selection), than to improve the quality of care. However again, adverse selection is not unique to managed care, being a concern in any private competitive insurance scheme.

Disadvantages in managed care as it is practised in the USA.
A number of disadvantages are exacerbated by the structure of the American health financing system. It is competitive, often designed to maximise profits, and reduce costs for employers. Since World War II, employers have offered health insurance to their employees, and since the 1980’s they have been a powerful lobby group favouring managed care as a way to decrease their costs. For-profit MCOs have been the most aggressive in using financial incentives to increase profits for shareholders, and not necessarily to improve patient care.

Adverse risk selection is more likely in a price competitive health model where there are perverse incentives to avoid developing good practices to care for sick people. Plans deliberately market their services to relatively healthy people, and avoid practices (such as becoming experts in treating AIDS) that would attract high cost patients.

The problem of competing on financial and not quality parameters is compounded by the problem of disseminating information on the relative quality standards of different health plans. Patients are price sensitive, but there is no good evidence that they are able to distinguish between plans on the basis of quality. Where information on quality does exist, it is by no means comprehensive or necessarily comprehensible. Any competition in the US therefore has essentially been on the basis of costs, or more correctly health premiums.
The social solidarity, which is seen in countries like the UK and New Zealand, is not a strong feature of the individualistic, market-orientated American society. There is a deeply ingrained mistrust of government, which pervades many aspects of American life. "...the United States continues to resist a universal system, because adopting one would mean giving too large a role to government". Without a strong government role in health care, much of the responsibility for providing and regulating health care has been transferred to the private sector. MCOs set priorities, ration health care, control costs, accredit hospitals, monitor quality, and determine payment rates for doctors.

The impact of managed care on health care costs and quality
The American health care system is the most expensive in the world, spending US$4000 (1997 dollars) per person, compared with New Zealand which spends approximately US$1300 per person. This increased spending is not reflected in better population health, as measured by two proxies - life expectancy and infant mortality - in which New Zealand outperforms the USA. The cost of health care, and the rise in health care premiums, led to the expansion of managed care. So how has managed care fared in this regard?

Health insurance premiums in USA, which were rising rapidly in the late 1980s and early 1990s (for example, 18% between 1988 and 1989), rose only 0.2% in 1997. Likewise, the rise in overall health care costs slowed, increasing by only 4.8% in 1997, the slowest annual rate increase since 1962. This has been achieved by restricting the range of services, doctor payments and hospital stays. Hospital stays for all diagnoses are among the shortest in the developed world at 7.8 days, compared with 9.8 days in the UK, though still longer than in New Zealand (6.5 days). Much of the savings in premiums are savings to employers, but at the expense of patients and their families, who experience higher out of pocket expenses, and greater responsibility for recuperative and psychiatric care.

The impact on costs and insurance premiums may be ‘one-off’ savings. Whether managed care has fundamentally altered the reasons for increased medical costs, such as the introduction of new technologies and the demand for health care, is much less clear. Evidence of the transient nature of the cost savings can be seen in a recent survey. It revealed that drug prices rose 13.8% in 1998, and for the first time in this regard?

Financial incentives can jeopardise the doctor/patient relationship. To avoid this, payment arrangements can be structured so as to decrease the conflict of interest for the doctor and bolster trust. The intensity of financial incentives is important. First, the entity paid by capitation must have the power to redesign the process of care, but should not be at the level of individual practitioners, as this can distort the medical decision-making process. Second, the size of bonuses or deductions should be small and not determined annually, as this may act as an incentive to change practice towards the end of the financial year. Finally, ‘stop-loss’ provisions can ensure that doctors are not exposed to undue financial risk if they are caring for a patient who requires an exceptionally expensive treatment. Adverse risk selection can be mitigated by regulation or risk adjustment to reflect the expected health costs of caring for patients with differing health status. Most plans in the USA adjust payments to doctors, using just two factors - age and sex - however, these factors are poor predictors of variation in medical costs. Work is ongoing in improving these methods. Two other features of managed care as practised in America should be avoided: the reliance on industry/employer concerns to shape policy direction, and the dominance of for-profit organisations, if such profits are not directed to improvements in patient care.

Lessons for New Zealand
As New Zealand looks at further health reform, it is important to see what can be learned from the managed care experiment in the United States, to acknowledge that it is indeed an experiment, of uncertain effectiveness. Importing the American concept of managed care wholesale, is neither feasible nor desirable. There are aspects of managed care which are positive, and pitfalls to be aware of. The first positive is that the process of care can be managed with the potential to improve quality of care for patients. Strengthening the population perspective and disease prevention is a core activity of this approach. It is also clear that one can and should measure the quality of care provided, and those measures should have relevance to patients, be reproducible, and case-mix adjusted. Sophisticated information systems are vital. Managed care can provide the impetus for rationalising care and the development of innovative practices such as enhancing the scope of activities for practice nurses and other allied health professionals, or increasing communication and integration between health care sectors.

The pitfalls centre on how financial incentives are used, and their impact on the doctor-patient relationship. The use of market competition to improve cost and quality evolved out of historical, political, and economic imperatives in the USA that are not relevant in New Zealand. The 1993 reforms in New Zealand failed to show an advantage of this competitive model, and international recognition is growing that competition in medicine has not been successful, and can be detrimental to the co-operative ethos that should characterise health care delivery.

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Conclusions
The American managed care ‘experiment’ has highlighted positive features, but also inherent and culture-bound flaws. It has succeeded in temporarily slowing the growth in health care costs, and the feared reduction in quality has not been seen. However, if there is a problem with quality it appears to have affected the most vulnerable populations.

It is not realistic for New Zealand to import this process unchanged, though there are aspects of managed care that could improve patient care, such as the emphasis on quality measurement, innovative care practises, and accountability for the health of a given population. If managed care or managed care techniques are introduced, the process should be co-ordinated and planned, and the objectives should be explicitly outlined and debated. Adequate controls and a
commitment to measuring the effect of such changes must support any change. Such reform will only be successful if both the public and health professionals are involved.

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