

MENTAL HEALTH IN NEW ZEALAND FROM A PUBLIC HEALTH PERSPECTIVE

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CHAPTER 22: DEMENTIA

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The dementias are a group of disorders of multiple aetiologies that are characterised by deficits in cognition, of sufficient severity to cause a decline in social, occupational and personal functioning. The dementias are devastating illnesses and responsible for considerable morbidity and mortality. These disorders most commonly affect the elderly population, but some, such as AIDS dementia complex, Huntington's disease, Pick's disease, alcohol-related dementia and hereditary presenile dementia, affect younger people. All the dementias have complications that reach beyond the sufferer. Expensive facilities and services at substantial social and financial cost are needed for their management. While many of the dementias are incurable, to some extent their complications may be preventable.

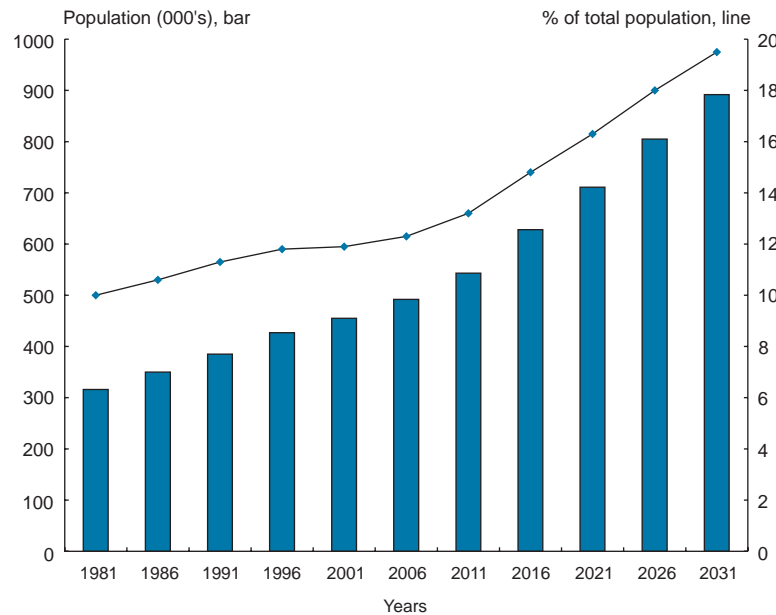
Dementia affects people in three domains, the cognitive, the functional and the behavioural. Cognitive impairment, memory loss and failure to attend to their personal care always occur in dementia, which has a deteriorating, progressive course. These problems are responsible for most of the need for residential services. Problems in the third, behavioural domain, although very common (up to 80 percent of patients), tend to vary in problematic intensity. The behavioural and psychiatric complications of dementia are many. They include features of psychosis such as hallucinations and delusions, depression, anxiety, aggression (both physical and verbal), agitation, wandering, challenging vocalisation and diurnal rhythm disturbances (Burns et al 1990a, 1990b, 1990c, 1990d; Becker et al 1994; Lykestos and Rabins 1994). Some behaviours are simply the result of misinterpretation of environmental stimuli because of lowered cognitive functioning, some are the result of disinhibited personality traits, and some are the result of psychotic stimuli such as hallucinations or delusions. Assisting caregivers to acquire management skills can reduce these behaviours without resort to potentially harmful medications.

By 2031, 19.5 percent of the New Zealand population will be over the age of 65, and 5.4 percent will be over 80 (see Figure 22.1). The ageing of the population applies to all racial groups in New Zealand, including Māori and Pacific people whose elderly populations are expected to increase by 9 and 8 percent respectively by 2031. The growing absolute number and increasing proportion of elderly people in the population mean that disorders affecting older people will have a greater impact on society.

AGEING, COGNITION AND DEMENTIA

Many older people notice as they age that their memory and their ability to learn new information decline. For many this subclinical cognitive impairment is not progressive and does not lead to the

Figure 22.1: New Zealand population projections for the population 65 years and over, 1981–2031



Source: Statistics New Zealand

significant deterioration in functioning that is characteristic of dementia. It is not clear whether this cognitive decline is a 'normal' consequence of ageing or an impairment continuum with dementia, or whether it predicts development of dementia.

Longitudinal studies are needed but few exist and none of these examines New Zealand elderly populations. Most studies published thus far are too short in duration (3–4 year follow-up periods) to clarify the issue, but do provide some indicators.

The current literature appears to be slightly in favour of there being an increased risk for dementia developing in those with subclinical cognitive impairment with increasing age. Despite variability of opinion on the status of cognitive impairment with no dementia (CIND) as an aspect of 'normality', a single discrete entity of minimal dementia, a range of subtypes or a prodromal phase of senile dementia, all studies agree that increasing age is the major risk factor for developing both cognitive impairment and dementia.

Although substantial numbers of persons with CIND do not progress to senile dementia, the border between the two is very unclear. Currently this is clinical judgement, set at the point when cognitive impairments significantly interfere with ability to function. There needs to be better agreement on this point as functioning criteria can be very arbitrary. Social factors such as the amount of support given, social stimulation or isolation, complicating anxiety or depression and non-dementing comorbid conditions such as visual or hearing difficulties can all influence the point at which functioning becomes impaired. A long-term longitudinal study is required to investigate cognitive aspects of ageing in New Zealand. A model for such a project is that initiated by the UK Medical Research Council and Department of Health to investigate the social and medical implications of being 65 and over in an ageing society. This project includes cognitive decline as a major area for study.

Despite the importance of recognising dementia, primary care physicians often miss cases (Ineichen 1994). Identified cases in primary care and those presenting to services are fewer than would be expected for population survey data (Pilkington 1992). A survey of New Zealand general practitioners found that few routinely used bedside cognitive screening tests with their elderly patients (Pilkington 1992). Of those that did, most used the 10-question Mental Status Questionnaire (Kahn et al 1960). This instrument is quick and easy to use but will only identify the more obvious 'cases'. Of those that are identified as having dementia, fewer than 10–20 percent reach specialist assessment services, and these are likely to be those with behavioural or severe functional difficulties. There may also be cultural variations in willingness to access services: racial minorities have been found to be less likely to access services (Braun et al 1995).

EPIDEMIOLOGY OF DEMENTIA

AGE-RELATED DEMENTIAS

The most common disorder is dementia of the Alzheimer type (DAT). Alzheimer's disease may not be a single disease but several related conditions (Blennow et al 1993). In Western countries, DAT represents about 60 percent of all diagnosed dementias and is more common in women. It is characterised by a progressive decline in memory, new learning, language skills, recognition and ability to function. Variants such as dementia of the Lewy body type (DLBT) are becoming increasingly recognised as qualitatively different from DAT and represent about 10–12 percent of all dementias. DLBT presents with similar cognitive deficits to DAT but patients with DLBT are more likely to have visual hallucinations, delusions and falls, and have a marked sensitivity to neuroleptic medication. Patients with DLBT may develop parkinsonian symptoms as part of the dementing disorder and DLBT is often (but not exclusively) the type of dementia that occurs in 20 percent of patients with Parkinson's disease (McKeith 1995). It is more common in men (1.5:1).

VASCULAR DEMENTIAS

The next most important dementias to consider are the vascular dementias. These are also a group of several disorders that range from the subcortical Binswanger type dementia to the cortical multi-infarct picture. Vascular disorders represent about 10–15 percent of dementias and are slightly more common in men. The low incidence of vascular dementia in some countries (eg, China and Japan) may provide some clues to specific risk factors for these disorders in the future (Graves et al 1994).

OTHER DEMENTIAS

Alcohol is responsible for about a further 10 percent of all dementias. All other causes of dementia, including Pick's disease, Huntington's disease, neurodegenerative disorders and AIDS-related dementia, make up the small proportion remaining.

PREVALENCE

The prevalence of dementia in overseas studies varies from 5–8 percent of the total elderly population (Saunders 1993). The figures vary in different studies as different case-finding instruments are often used. Virtually all published studies agree that the incidence of dementia increases with age. At age 65 years about 3 percent of the elderly population will have an age-related dementia but this figure rises steeply to over 20 percent at age 80 (Copeland et al 1987; Saunders 1993; Ebly et al 1994). For New Zealand, in the absence of local data, assuming the same incidence of dementia as in the overseas studies, the present growth in population bands to continue and a stable total net population of 3.5 million, we should expect 20 000 to 30 000 people with dementia by the year 2000. As the greatest rise in the population will be in those aged 80 or over, prevalence could double to 50 000–60 000 by 2031, an annual rise in incidence of 1.6 percent. According to the large Swedish Lundby epidemiological study, in a European population similar to our own (Hagnell et al 1993), the cumulative lifetime risk for developing DAT is 25.5 percent for men and 31.9 percent for women. For the most severely impaired cases, the figures are still significantly high at 15 percent for men and 22.2 percent for women. For vascular dementia, the cumulative risk is 29.8 percent for men and 25.1 percent for women (all cases) and for the more severely impaired, 16.6 percent for men and 15.2 percent for women.

RISK FACTORS FOR THE DEMENTIAS

GENETIC

Recent work on Alzheimer's disease has demonstrated genetic links associated with the hallmark pathological lesions in some patients (Kuusisto et al 1994; Lovestone 1994; Brietner and Welsh 1995). One such gene on chromosome 21 is in proximity to the gene controlling the expression of Down's syndrome, possibly accounting for the association between Down's syndrome and DAT. Two genes implicated in familial early onset disease (1–2 percent of cases of DAT) are presenilin 1 on chromosome 14 and presenilin 2 on chromosome 1. Situated on chromosome 19 is a gene that controls production of apolipoprotein E (ApoE), a lipid transporter protein essential for the integrity of neuronal cell walls. There are several forms of this protein. Current thinking is that the ApoEε4 allele for this protein may make the brain more vulnerable to the events that cause DAT or that possession of the allele may bring forward the age at which these events might occur. Recent work also suggests that possession of the ApoEε4 phenotype may also be a risk factor for dementia of the Lewy body type (McKeith 1995). It may be that possession of the allele increases vulnerability to other brain disease or compromise recovery from brain injuries (National Institute on Aging/Alzheimer's Association Working Group Consensus Statement 1996). Having a genetic vulnerability does not inevitably lead to dementia, nor does it explain every case of Alzheimer's disease (Rebeck et al 1994). Some ApoEε4 carriers survive to very old ages and remain cognitively intact. Factors other than genotypes almost certainly influence expression of the disease.

NON-GENETIC RISK FACTORS

Head Trauma

Several environmental factors have been postulated as potential risks for DAT. The evidence of a link between head injury and DAT is weak despite several studies looking at this question. Further development of the genetic studies outlined above may make the position clearer. If apolipoprotein E is found to influence recovery of the brain from injury by its effect on the deposition of amyloid, then carriers of the ApoEε4 allele may also have an increased risk to both the negative consequences of head injury and dementia. Thus the risk for developing dementia after head injury may depend not so much on the nature of their injury but the genetic potential for repair. Such a study has yet to be published. If this hypothesis is substantiated then it could have implications for sports or activities where the risk of head injury is high, such as motor racing and boxing, as those participants with a high risk could be identified.

Toxins

Various toxins have been suggested as potential causes of Alzheimer's dementia. Aluminium, for example, received considerable publicity in this respect a few years ago. This has not been substantiated, despite several studies, including one case-control study of moderate statistical power in the UK (Forster et al 1995; Taylor et al 1995) and one of autopsies (Mann DMA 1993). Dietary deficiency of selenium and calcium have been also been suggested as risk factors but again not found to be statistically significant (Emard et al 1995). Of perhaps more interest is the finding of the Canadian Study of Health and Aging (1994) that exposure to solvents, glues, pesticides and organic fertilisers increases risk for Alzheimer's disease. Another case control study in North America confirmed this (Kukull et al 1995) and concluded that previous solvent exposure increased the risk for DAT by 2.3 for all and 6.0 for males, most of whom were occupationally exposed.

Cigarette smoking is a major risk factor for many health problems but not for DAT. On the contrary, the current evidence is that nicotine may have a protective effect on early onset DAT. While nicotine may have some protective effect for DAT, cigarette smoking remains a major risk factor for atherosclerosis, hypertension, cardiac disease and stroke, known risk factors for the vascular dementias.

Cerebrovascular Disease and Dementia

Vascular causes for dementia are probably more common than is currently appreciated. Risk factors associated with cardiovascular disease, that is, hypercholesterolaemia, diabetes mellitus, cigarette smoking, alcohol overuse and untreated hypertension, also apply to the vascular dementias. As these risk factors for vascular dementia are well known and are amenable to intervention and treatment, there is considerable potential for prevention of the vascular dementias (Jorm 1994). While there is clear evidence that hypertension increases the risk of cardiac disease or stroke, evidence that untreated hypertension *per se* in the elderly leads to insidious cognitive impairment is mixed. A cross-sectional,

multi-centre, single blind, placebo-controlled trial of treatment for mild to moderate hypertension in older people showed no significant differences in the untreated or treated group on psychometric tests of cognitive function (Mann AH 1993). In contrast, a recently reported 15-year comprehensive longitudinal trial of 70-year-old people in Sweden demonstrated a significant relationship between elevated blood pressure at age 70 and the development of not just vascular but also Alzheimer-type dementia 10–15 years later (Skoog et al 1996). It is suggested that raised blood pressure predisposes to cerebrovascular ischaemia leading to demyelination that causes dementia by disconnection of the subcortical–cortical association pathways. This finding that hypertension may be an additional risk factor for Alzheimer's disease has been replicated in other studies both published (Martyn 1996), and yet to be published, such as the UK Medical Research Council multi-centre study on cognitive function and ageing (McCracken 1996; Brayne et al 1996).

Alcohol

Heavy alcohol use can cause cognitive impairment as the substance is neurotoxic in large amounts, both directly and through an associated nutritional deficiency of thiamine. Approximately 10 percent of alcohol abusers develop a dementia as a result of drinking (Allen 1994) and this represents about 10 percent of all dementias. The Wernicke-Korsakoff syndrome is caused by thiamine deficiency influenced by a genetic predisposition to abnormal thiamine-dependent transketolase (Victor et al 1989). It is usually, although not invariably, associated with alcohol abuse. Other causes of thiamine deficiency such as persistent vomiting or malnutrition may also cause the syndrome. Diet is often marginal in older people living alone and cases of malnutrition do occur. Most of the research has concentrated on mortality from cardiovascular disease. An association between alcohol and haemorrhagic stroke showed an increasing incidence of stroke with increasing alcohol intake (Beaglehole and Jackson 1992). However, as heavy drinkers are often also smokers it is difficult to separate the effect of alcohol from related cigarette smoking that would also increase the risk for stroke. Even when alcohol is not implicated as a cause of dementia, heavy alcohol use decreases survival in older people with or without dementia (Jagger et al 1995).

The effect of chronic low doses of alcohol on cognition has not been adequately reported to date. Two studies currently in progress in France (Epidemiology of Vascular Aging, Paris (Dufouil et al 1996)) and Australia (Centre for Education and Research on Aging, Sydney (Dent et al 1996)) have so far not shown any deleterious effect from mild to moderate alcohol intake on cognition. So far, some results show the opposite. The French group has observed a positive effect with mild alcohol intake and cognition in women and the Australian study on aged war veteran males showed no significant effect on cognition even after lifelong consumption at the hazardous level.

Benzodiazepines

Prolonged benzodiazepine use can cause cognitive impairments indistinguishable from those caused by alcohol and often resembling DAT. This is a particular problem for older people who have sleeping problems and start on benzodiazepine hypnotics to correct this. Yet despite this, prescribing of benzodiazepines may be increasing (Sullivan 1988).

PROTECTIVE FACTORS

Some factors appear to be preventive for dementia. An interesting finding from the Canadian Study of Health and Aging (1994) identified low education as a risk factor for Alzheimer's disease. This finding has been confirmed (Stern et al 1994). It may be that increased educational and occupational attainment reduces the risk of incipient DAT, either by decreasing ease of clinical detection of DAT or by imparting a cognitive reserve that delays the onset of the clinical manifestations. Education and occupation have remained significant predictors after controlling for age, site, sex, stroke, and baseline mental status scores in several other studies (White et al 1994; Mortel et al 1995) including twin studies (Reed et al 1994). However, the research fails to differentiate between whether the protective factor is the pre-morbid IQ or the level of education, nor does it predict what outcomes might be expected when there is a mixture of risk and protective factors such as in the high-IQ, highly educated, homo- or heterozygote carriers of ApoEε4.

DISABILITY AND DEMENTIA

Dementia is one of the major causes of disability in the elderly, affecting personal care, everyday cognitive activities, and social behaviour (Jorm 1994). Prevention of dementia would make a major contribution to the postponement of disability in old age. Irrespective of the cause of the dementia, there are substantial risks for complications that cause greater disability and increase the need for care. These are often treatable and preventable.

DEPRESSION

Depression, if sufficiently severe, has marked but reversible effects on memory and cognition. Patients with dementia complicated by depression have a decrease in function and cognition due to the depressive component alone. Several studies have indicated that depression is a common complication of dementia (Vida et al 1994; Migliorelli et al 1995; Reichman and Coyne 1995) and give prevalence figures of 23–40 percent for dysthymia, and 23–29 percent for *DSM-III-R* major depressive episodes. However, most of these studies did not differentiate demented patients with a previous psychiatric history from those without. A review of two Alzheimer databases that excluded patients with a previous history of depression found a very low prevalence of depression over a four-year follow-up period (Weiner et al 1994). The study concluded that, longitudinally, Alzheimer's disease itself does not predispose to depression. However, pre-morbid depression may herald the onset of the dementia and a previous history of depression increases the likelihood of depression as a complication of the dementia. Depression and dementia are two separate diseases and coexistent depression can significantly reduce functioning and increase disability.

MALNUTRITION

Problems with food intake can result from several causes in cognitively impaired individuals, such as declining taste and olfactory thresholds, depression, failing memory and inability to perform the steps necessary to prepare food. Community care workers frequently find refrigerators containing rotting food and their clients living on biscuits and tea. While there is no doubt that malnutrition can be a frequent consequence of cognitive impairment and can increase disability, evidence that either malnutrition or micronutrient deficiency can cause dementia is not substantiated in the literature.

FALLS

The risk of falls and injuries as a complication of dementia is substantial. Cognitive impairment and inability to perform activities of daily living are substantial risk factors for falls. The rate of all injuries in a sample of 283 patients with dementia living in the community was substantial at 58.4 per 100 people per year (Oleske et al 1995).

POLYPHARMACY AND PSYCHOTROPIC DRUGS

Another major cause of morbidity in older people with dementia is drug interactions, polypharmacy and overuse of psychotropic medication for behaviour disorders. This last-mentioned problem became so acute in the US that in 1987 the US Government, presented with overwhelming evidence on the overuse and misuse of psychotropic medication, legislated for regulatory measures for neuroleptic drug administration in nursing homes (Samuels and Katz 1994). The US experience is not unique and neuroleptics have been widely used in New Zealand as means of social control and restraint for challenging behaviours in demented people. Antipsychotic medications are useful when behaviours result from psychotic phenomena, but there is no evidence that they are anything other than a chemical restraint for the majority of behaviours such as wandering or aggression. Unfortunately this is a major reason for prescribing these drugs for older people with dementia (Nasman et al 1993). Behavioural techniques are labour- and training-intensive and while such interventions seem preferable on humanitarian grounds, their efficacy has not yet been shown to be superior to medication. There are no studies comparing medication either singly or in combination with behavioural and environmental interventions in the treatment of challenging behaviours in demented older people.

However, the consequences of misuse of medication are serious. The ageing brain is very sensitive to the effects of neuroleptics. The effect of overuse of these drugs is to cause over-sedation, tardive disorders, falls, incontinence and loss of quality of life. In addition, these drugs are common causes of delirium in dementia. Add delirium to behavioural complications of a dementing disorder and the result is an unmanageable patient for the average rest home or private hospital. Acute hospitalisation is usually the result. In one Auckland unit, the commonest cause for delirium in demented patients is overuse of psychotropic drugs or drug interactions. The unit's average hospital length of stay required to reverse and treat delirium is 14–21 days.

CAREGIVER STRESS

The care needed for people with dementia is extensive. Many patients are unmanageable at home because of disability, difficult behaviours, risk of accidents or inability of frail elderly spouses to contend with difficulties. Not only are nursing and rest home places needed but there is also a need for specialised units to deal with the behaviourally disturbed. As a result of the increasing absolute numbers and the capping of expenditure on hospital or health service based services, there has been an increasing trend worldwide as well as in New Zealand for more reliance on informal caregiving. Cost shifting from the health to the community sector is not without impact on the community in terms of caregiver morbidity.

Dementia can also cause morbidity in others and there is a considerable literature on carer burden, carer stress and elder abuse (see Chapter 6 for more detailed discussion on this topic). Caregivers of elderly demented people commonly report negative effects on their functioning. The considerable psychological morbidity in caregivers of both the cognitively impaired and intact groups is largely

accounted for by physical disability (LoGiudice et al 1995). This is most burdensome for caregivers who are unable or unwilling to access adequate services. Caregivers of older people tend to be women, usually adult daughters or spouses.

In a study of 103 American lower socioeconomic status caregivers, care burden had an inverse relationship with perceived family support for the caregiver (Biegel et al 1994). This study also showed that the most stressful problem for caregivers of the demented was the frequency of problem or unpredictable behaviours. Thirty-seven percent reported detriment to their health and 41 percent did not feel they got enough support from health agencies. Other studies report similar findings. A direct relationship between the severity of the patient's symptoms and deleterious effects on the caregiver is a common finding in these studies. Daughters seem to suffer more care burden than spouses (Jones and Peters 1992) and depression and anxiety are the commonest symptoms. Unfortunately this may not always reverse if the index patient is hospitalised as caregivers sometimes feel immense guilt and sadness that such action was necessary, despite the damage to their own health. On the other hand, not all burden is negative (Macera et al 1993) and it should be noted that many caregivers gain self-esteem through caring for an aged spouse or parent.

SUMMARY OF KEY POINTS

- 20 000 to 32 000 New Zealanders can be expected to be suffering from dementia by the year 2000.
- The majority of these will have dementia of the Alzheimer type (DAT).
- Possession of the ApoEε4 phenotype indicates a higher probability, but not inevitability, of developing DAT.
- The ApoEε4 phenotype possibly increases vulnerability to brain disease and injury in general, thus lowering the age of onset of dementia.
- Other risk factors are important for expression of the disease in addition to genetic vulnerability.
- Risk factors for Alzheimer type dementia:
 - *Definite:* age, ApoEε4 genotype, Down's syndrome, family history
 - *Possible:* female sex, cerebrovascular disease, previous hypertension, poor education, head injury, solvent and pesticide exposure
 - *More evidence needed:* aluminium toxicity, selenium deficiency.
- Higher levels of education appear to be either protective against DAT or, by providing a cognitive reserve, delay the age of onset. The role of IQ is not known.
- Risk factors for vascular dementias are stroke, hypertension, diabetes mellitus, alcohol abuse, and cigarette smoking.
- Increased disability in dementia can be caused by depression, falls, malnutrition, delirium and overuse of psychotropic medications.
- Caregiver stress is a major problem with considerable morbidity and is a major reason for institutionalisation.

PRIMARY PREVENTION AND SCREENING PROGRAMMES

Environmental risk factors can be targeted to reduce problems. There is some evidence about the effect of solvents, their effects on the brain and an increased risk for DAT. This risk factor is highly preventable through education and modification of work practices. Other risk factors can be reduced through health promotion. Particular emphasis needs to be put on moderation of alcohol intake, dietary management, control of hypertension, diabetes mellitus and increasing exercise. All these risk factors also have implications in other health problems. Avoidance of smoking is desirable to reduce risk of vascular dementia.

There is an important role for education of the general public on risk factors that influence dementia. Channels for this include the media as well as specific health promotion programmes. There are several community groups and agencies whose main interest is the quality of life of the older population (eg, Alzheimer's Society and Age Concern), which could have a role in disseminating health information.

EARLY IDENTIFICATION AND SECONDARY PREVENTION

As dementia is common but underdiagnosed, more teaching emphasis needs to be included in medical school curricula on these disorders. Ongoing education of doctors particularly at primary care levels on dementia, risk factors and complications would also be useful. A review of several overseas and Australian studies found knowledge about dementia and depression at primary care level to be inadequate (Bowers et al 1992).

Some disorders of cognition are reversible and a major advantage of early detection of dementia in the community is to detect these reversible impairments caused by such disorders as depression, hydrocephalus, metabolic disorders or brain tumours. Other disorders coexist with or increase risk for dementia such as hypertension or stroke and early identification and appropriate treatment of these could modify subsequent morbidity or disability from dementia.

General practitioners could be encouraged to think more about routinely screening older people for cognitive impairment. Although the 10-question Mental Status Questionnaire (Kahn et al 1960) is considered woefully inadequate by most secondary services, even this used routinely would be better than nothing. A better screening test is the Mini-Mental Status Examination of Folstein and Folstein (Folstein et al 1975) as, with training, this is a reliable screening instrument, is easy to administer, takes less than five minutes and is much more sensitive than the Mental Status Questionnaire. However, this test will only identify about 75 percent of 'cases' using a high cut-off of 27/30. Addition of further simple tests such as drawing a clock-face can improve sensitivity by another 10 percent. Patients thus identified could be directed to appropriate services, modifiable risk factors addressed, problem medication avoided, complications anticipated or prevented and families at risk for caregiver stress identified. However, it should be noted that screening for early or mild dementia may lead to premature rather than delayed residential care as caregivers accept the inevitable decline and place their relatives before the burden of care becomes too great (O'Connor et al 1991).

Currently, Alzheimer's disease is irreversible and the basic pathology untreatable. ApoE gene testing is becoming more common in Europe and the US as a routine test for determining risk for Alzheimer's disease in non-demented people. Such action is not without considerable ethical and economic implications. The presence of ApoEε4 does not necessarily predicate inevitable dementia, only an increased risk. Without treatments to reverse or significantly modify the condition, knowledge of such risk could cause negative anticipatory stress for individuals and families. At the present time, ApoE screening for non-demented people as a general screening test has not been recommended by the Consensus working party on ApoE genotyping (National Institute on Aging/Alzheimer's Association Working Group Consensus Statement 1996) as its usefulness in predicting risk has not been established.

Some early cases of Alzheimer's disease may respond to the cholinergic drugs, tacrine or other nootropics (medication that improves cognitive function). If a patient can be identified early, this could benefit subsequent management by helping caregivers recognise problems early before they become intractable or complicated. In symptomatic patients, finding one or more ApoEε4 alleles is highly suggestive of Alzheimer's disease and the test has probably more utility in symptomatic or very high-risk individuals. Clear guidelines would need to be established for the test to be more available. For each individual, these should include a mandatory counselling component, construction of an adequate pedigree, disclosure of risks, disclosure of possible implications of the disease, benefits and burdens of receiving test results, alternatives to testing and the conditions under which disclosure is made to family members and third parties (National Institute on Aging/Alzheimer's Association Working Group Consensus Statement 1996).

TERTIARY PREVENTION

As the absolute number of people with dementia increases, services to support both sufferers and their caregivers will be necessary. The literature on caregiver stress indicates that services for the patients alone are insufficient and that services need to be designed with caregiver needs in mind as well. More respite care is needed to give caregivers a break and to enable them to continue caring. Caregivers at risk need to be identified and given support before problems arise. The patient's depression can be treated with antidepressants and even electroconvulsive therapy in mildly demented cases with reversal of the depressive cognitive component and improvement in functioning. Many of the complications of inappropriate medication and environmentally induced behavioural problems could be avoided if there were sufficient specialist units with well-trained staff for the behaviourally disturbed.

CONCLUSIONS

Dementia is a problem that affects a substantial number of people, causes great distress and has major economic implications. Scientific study of the biological, psychological and social effects of the dementias is a major priority area for research in the health sciences worldwide and almost every day there is new ground-breaking work somewhere in the world. However, a national longitudinal study is needed on the impact of the social, medical and cognitive implications of being over 65 years of age in New Zealand. A longitudinal study on the effect on cognition of treating hypertension could demonstrate a significant public health impact. Little is known of national patterns of caregiver stress, especially in Māori and Pacific people. Although we can profit greatly from the accumulated experience and knowledge from overseas, New Zealand needs to develop its own national strategy for dealing with the major public health issues of the dementias that are becoming evident with the ageing of our population.

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