

IS THE INCIDENCE OF DEMENTIA DECLINING?

A REPORT FOR ALZHEIMER'S AUSTRALIA
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TALKING ABOUT ALZHEIMER'S
ACROSS AUSTRALIA
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Affiliations:

Alzheimer's Australia respectfully acknowledges the Traditional Owners of the land throughout Australia and their continuing connection to country. We pay respect to Elders both past and present and extend that respect to all Aboriginal and Torres Islander people who have made a contribution to our organisation.

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DEFINITIONS

Incidence: The number of new cases of a condition (e.g. dementia) per population at risk in a given period of time.

Prevalence: The proportion of the population found to have a particular condition (e.g. dementia) at a specific point (point prevalence) or over a given period (e.g. 12 month prevalence).

Cohort effect: The effect of being born at about the same time, and therefore likely to have similar experiences and be exposed to the same events and similar risks.

Dementia: A neurological condition in which there is substantial decline in a person's cognitive abilities (memory, executive function, language, etc.) from a previous higher level to the extent that the person is no longer able to independently perform everyday activities. Dementia has many causes.

Alzheimer's disease: The most common cause of dementia due to a degenerative condition of the brain which presents clinically with a gradual and progressive loss of memory and other cognitive functions, usually in late life, and has a distinctive picture on neuropathology, characterised by the presence of amyloid plaques and neurofibrillary tangles.

Vascular dementia: The second most common type of dementia in which there is loss of cognitive function due to the presence of disease of blood vessels in the brain leading to damage to brain tissue from loss of blood flow or, less commonly, bleeding in the brain (generally referred to as stroke or mini-stroke).

FOREWORD

I asked Professor Perminder Sachdev to write this publication because I believe it is important that Australians understand that the prevalence rates of dementia are not set in stone.

Recent research evidence suggests that the prevalence rates of dementia may have declined. The causes of this decline are not well understood but it may be the consequence of a number of environmental factors including better education and health care.

It remains true however that there are significant health factors, such as diabetes and obesity, that may increase dementia prevalence rates in the longer term. As well, in spite of the decline in prevalence rates, the numbers of people with dementia will continue to increase with the ageing of the population.

A much higher priority needs to be given to epidemiological research that provides a basis for understanding the prevalence of dementia in Australia and gives a baseline for monitoring changes over time. It should be a matter of concern for those with a serious commitment to health policy that Australia has to draw on international studies to calculate our country's prevalence rates on the basis of the aged structure of the population.

On a more positive note, Australia was the first country in the world to have a publicly-funded dementia risk reduction program, *Your Brain Matters*, which is administered by Alzheimer's Australia with Commonwealth funding. I hope this new publication will encourage even more Australians to visit our

website www.yourbrainmatters.org.au and follow the recommended action to improve their brain health.

It is essential that health policy makers responsible for preventive health policies make connections between physical and brain health. It is now well established that the risk of dementia is greater for people with diabetes, cardiovascular disease, obesity, HIV/AIDS, and smokers. It follows that health promotion activities relating to physical conditions and lifestyle should make the point that people might be able to doubly benefit from preventive health activities with improved brain health.

I'm grateful to Professor Sachdev, who is now Chief Medical Adviser to Alzheimer's Australia, for preparing this publication. I hope it will serve to make people take a more serious interest in finding out more about the nature of dementia and its prevalence.



Ita Buttrose AO, OBE
National President, Alzheimer's Australia

EXECUTIVE SUMMARY

BACKGROUND

The world-wide projections of the prevalence of dementia in the coming decades have been a source of great concern to health systems and societies around the world. The World Alzheimer Report 2010 (1) estimated that there were 36 million people with dementia in 2010, with an expected doubling every 20 years to nearly 115 million in 2050. These sobering figures are based on assumptions that the age-adjusted prevalence of dementia would remain constant and the population would continue to age at the current rate.

The assumption that the incidence of dementia will remain stable is now being put into question. There is emerging evidence to suggest that the incidence of dementia in older individuals may be declining. It appears that this change may be recent and has possibly occurred only in the last one to two decades. It may also be restricted so far to high income countries, although data from low and middle income countries are lacking.

The reasons for this change are not understood, but education, more stimulating environments and better control of vascular risk factors may have contributed. The data are still preliminary and more studies are needed to establish the extent of this change and understand its causes. It should be noted that the decline is not large enough to offset the increase in prevalence of dementia due to the ageing of the population and therefore investment and efforts to develop better treatments and care for people with dementia need to continue.

The fact that dementia rates are malleable is an encouraging finding but the reduction cannot be taken for granted as gains in population health can easily be lost if societies do not remain vigilant and continually proactive. These preliminary findings provide a strong argument for large scale Government investment in dementia-prevention strategies, which should start from early life.

The world-wide projections of the prevalence of dementia in the coming decades have been a source of great concern to health systems and societies around the world. The World Alzheimer Report 2010 (1) estimated that there were 36 million people with dementia in 2010, with an expected doubling every 20 years to nearly 115 million in 2050. The economic cost of dementia was estimated to exceed 1% of GDP in 2010, and projected to grow (1). Nearly 60% of dementia sufferers now live in low and middle income countries (LMIC) in which health and social care systems are less well developed, and it is estimated that this proportion will rise to >70% by 2050 in what are currently categorised as LMIC (1). These sobering figures are based on assumptions that the age-adjusted prevalence of dementia would remain constant and the population would continue to age at the current rate. Recent data from Europe and North America have challenged the first of these assumptions, i.e. the incidence of dementia will remain constant.

There are a number of reasons why the incidence of dementia could possibly change. The two most common subtypes of dementia are Alzheimer's disease (AD) and vascular dementia (VaD) which together account for nearly three-quarters of all dementia (2), with AD accounting for the majority. There are known risk factors for both disorders, some of which are modifiable and have indeed been modified in recent times. Moreover, the two disorders often overlap in older individuals, and some of their risk and protective factors are shared. If the knowledge about modifiable risk factors is accurate, there is no reason why the incidence of these disorders should remain constant.

MODIFIABLE RISK FACTORS FOR VAD

The current concept of VaD, also referred to as Vascular Cognitive Impairment (VCI) (3) and Vascular Cognitive Disorder (VCD) (4), is broad and encompasses dementia caused by stroke as well as brain injury due to any other vascular disease, in particular small vessel disease which may otherwise go unnoticed clinically (5). Since stroke is an important cause of VaD, any risk factor for stroke is also a risk factor for dementia. In addition, a number of factors increase the risk of small vessel disease and thereby cognitive impairment and dementia.

The modifiable risk factors for stroke have been extensively studied and are identified as hypertension, diabetes mellitus, insulin resistance or the metabolic syndrome, high cholesterol, smoking, excessive alcohol use, obesity, physical inactivity, high homocysteine levels and atrial fibrillation (6). There have been major advances in the identification and management of these risk factors in the last half century and this is evident in the changing epidemiology of stroke. Most studies of stroke incidence, largely conducted in high income countries (HICs), show a secular trend of decline into the mid-1980s, although the trend appears to have plateaued thereafter and even reversed in some cases (7), with only an Australian study showing a decrease in stroke incidence in the mid-1980s (8). Several factors have contributed to this trend, which include better control of risk factors, early diagnosis and intervention and birth-cohort or period-cohort effects, such as foetal or early childhood health which is known to influence cardiovascular health (8). One can speculate that a reducing incidence of stroke will lead to reduced rates of VaD. There are, however, some counterbalancing factors. Firstly, the reduction in stroke has largely occurred in HICs, and the data from LMICs are either not available or suggest an opposite trend in some countries (7). Secondly, the mortality rate from stroke has also gone down (7), suggesting that there is an

increasing number of survivors, with a significant proportion suffering cognitive impairment. Thirdly, stroke is more likely to cause cognitive impairment in older people and those with lower education, previous cognitive deficits, diabetes and atrial fibrillation, and those who have repeat strokes or transient ischaemic attacks (6). Fourthly, early intervention and effective rehabilitation after stroke can reduce the consequent disability, including the effects on cognition. There is a world-wide effort to prevent and effectively treat stroke (9), and prevent the recurrence of strokes (9), and one can anticipate that this will have an impact on the incidence of VaD as well.

Stroke is of course not essential for the development of VaD, and other vascular disorders, in particular arteriosclerotic small vessel disease, are often implicated. A number of modifiable risk factors for these disorders have been identified (table 1), many of which overlap with stroke risk factors, and these can be the targets for primary and secondary prevention at both the individual and population levels. A recent consensus statement (5) made a number of evidence-based recommendations in relation to this: treatment of hypertension, hyperglycaemia, hypercholesterolemia and atrial fibrillation, smoking cessation, moderation of alcohol intake, weight control and increased physical activity are well supported. There are other measures such as treatment of depression and the use of anti-oxidants and anti-inflammatory drugs that currently do not have sufficient supportive evidence for their contribution to reducing the risk of cerebrovascular disease. However, overall trends in public health suggest that vascular disorders are being better managed or prevented and this augurs well for VaD rates, at least in HICs. The only disturbing trend is the rising rates of diabetes, obesity and the metabolic syndrome in both developed and developing countries, which could negate the benefits of other improvements. This is discussed below.

MODIFIABLE RISK FACTORS FOR AD

While late-onset AD, which is by far the most common cause of dementia, has a strong genetic basis, many modifiable risk factors have been identified from case-control and observational studies. They can be categorised as follows:

Vascular risk factors:

Many of the risk factors for stroke and VaD also increase the risk for AD. Most of the evidence comes from observational studies, although intervention studies are beginning to provide evidence that optimal control of these factors may delay if not prevent dementia. The main focus has been on hypertension, diabetes, metabolic syndrome, high cholesterol, mid-life obesity and physical inactivity, although smoking has also been suggested as a risk factor (10). These factors may directly accelerate AD pathology, increase the vascular lesions in the brain that co-exist with AD or have other direct brain effects. Cognitive benefits from the management of these risk factors may therefore emanate through either of these mechanisms, as is suggested for the treatment of hypertension (11). In a recent review, it was concluded that these risk factors accounted for nearly 25% of the population attributable risk for dementia world-wide, and their optimal management could prevent 10-15% cases of dementia (12).

Cognitive and brain reserve:

Repeated observation that high education protects an individual from developing dementia has led to the concept of brain reserve (13). From the conceptualization that a brain with high reserve is bigger and/or has greater morphological and metabolic complexity, there has been an increasing emphasis on cognitive reserve which emphasises functional rather than structural complexity. Proxy measures used to measure cognitive reserve have been education, intellectual abilities and complexity of occupation (14). By one estimate, high cognitive reserve halves the

risk of dementia (15), and the population attributable fraction of education for dementia prevention is estimated at about 20% (12). Importantly, cognitive reserve is not static, and complex cognitive activity through the life span can preserve and enhance it, therefore protecting against cognitive decline and possibly dementia (14). The brain itself is morphologically and functionally plastic, and both brain and cognitive reserve are modifiable factors through complex cognitive activity. Interestingly, physical activity and large social networks have also been suggested to increase brain reserve.

The fact that a high intellectual level, usually but not always reflected in high education, has been found to be protective in most longitudinal studies of cognitive ageing and dementia raises the question whether better educational opportunities and increasing complexity in society has had a beneficial effect through the 20th century. A reference should be made here to what has been called the Flynn or the Flynn-Lynn effect, which refers to the sustained increase in both fluid and crystallised intelligence scores that have been recorded in many parts of the world from 1930 onwards (16). This increase, reflected in the performance of successive cohorts every ten years or so, has been reported on most tests purportedly measuring intelligence, but also attention and memory, and applies to all ages and levels of ability. The rate varies between countries but most industrialized countries show the effect (17). Various explanations have been offered for the effect, with the most important being better schooling, test familiarity, a more stimulating environment during brain development and better nutrition (18). There is recent evidence, however, that the Flynn effect may be ending in developed nations. In Danish male conscripts, the average gain in IQ points was 3 per decade in the 60s and 70s but 2 points in the 1980s and 1.3 points in the 1990s (19). In some countries, it may even have reversed in the last decade or two (19).

Based on this evidence, one would expect that cohorts born later would have an advantage in terms of cognitive reserve and therefore a lower risk for dementia than their older counterparts.

The importance of physical exercise should be particularly emphasised as a protective factor. Exercise has a beneficial effect on a number of other risk factors such as obesity, diabetes and hypertension, and is important for maintaining bone and muscle mass and vitality in general. However, it also has a direct effect on brain function and animal work supports its positive effect on neurogenesis and brain reserve. Many molecular mechanisms underlying this are being explored. For example, aerobic exercise has been shown to stimulate the production of brain-derived neurotrophic factor, a protein that supports existing neurons and promotes the growth of new neurons, and resistance training assists in the production of insulin-like growth factor 1 which is part of a complex system that brain cells employ to interact with their physiologic environment and regulate neural development.

Nutritional factors:

Dietary factors have been reported to influence dementia risk. Of course, the role of diet in obesity and diabetes are self-evident, and these have been mentioned as risk factors in the sections above. The other dietary factor with consistent literature is moderate use of alcohol, which has a protective effect against cognitive decline and dementia (20). Being a teetotaler and drinking excessively are both disadvantageous in comparison. The evidence for a Mediterranean diet, fish consumption, fruit and vegetable consumption and the use of antioxidants is less consistent, as is that for lower levels of homocysteine and the use of folic acid and vitamin B12 (21).

Other risk factors: a history of depression and traumatic brain injury must also be considered as risk factors (12), and both are modifiable at the population level through medical and legislative actions.

Table 1:
Modifiable risk and protective factors for Dementia

VASCULAR DEMENTIA	ALZHEIMER'S DISEASE
1. <i>Brain and cognitive reserve:</i> education, occupation, complex mental activity, physical exercise.	1. <i>Brain and cognitive reserve:</i> education, occupation, complex mental activity, physical exercise.
2. <i>Vascular risk factors:</i> hypertension, diabetes mellitus, insulin resistance or the metabolic syndrome, high cholesterol, smoking, excessive alcohol use, obesity, physical inactivity, high homocysteine levels and atrial fibrillation.	2. <i>Vascular risk factors:</i> hypertension, diabetes mellitus, insulin resistance or the metabolic syndrome, high cholesterol, smoking, excessive alcohol use, obesity, physical inactivity, high homocysteine levels.
3. Stroke and transient ischaemic attack, including optimal treatment and rehabilitation.	3. <i>Nutrition:</i> Mediterranean diet, fish consumption, fruit and vegetable consumption, foods rich in antioxidants, moderate alcohol use, low homocysteine, reduced caloric consumption.
	4. Other: depression, traumatic brain injury

If the risk factors for dementia are indeed modifiable and appear to have changed in frequency over the last century, is this reflected in dementia rates? It is important to distinguish between prevalence and incidence rates in this context. In epidemiology, prevalence refers to the proportion of the population found to have a particular condition, in this case dementia, whereas incidence refers to the number of new cases that develop within a specified period divided by the population at risk. As the number of older people in society increases, the prevalence of dementia rises without necessarily any change in its incidence. Since the ageing of the population influences the prevalence of dementia, the answer to the question whether dementia risk is changing must be sought in age-specific incidence rates. Importantly, this change must be demonstrated in the same geographical region using identical methodology, as population and methodological differences can also lead to unreliable results. Unfortunately, there is no suitable Australian data, but a number of European and North American studies have attempted to address this question, using both incidence and age-specific prevalence rates. Incidence studies are more difficult to conduct as they involve following a large population of people without dementia over a number of years until a significant number develop dementia. Investigators therefore have often relied on prevalence studies at two different time points, after adjusting for any changes in the age structure of the population.

European studies:

One of the earliest studies in this regard was the Lundby study (22) which examined the incidence of 'organic brain syndromes', including multi-infarct and senile dementia, in the whole population of Lundby, a suburb of Gothenburg, Sweden over a 25 year period. This study reported a decreasing incidence between the periods 1947-57 and 1957-72. The data were re-examined by the same authors a few years later (23), who then retracted this claim, stating that there was no statistically significant difference between the two periods.

A Spanish group reported age-specific prevalence rates from the Zaragoza region in two studies about 7 years apart (24). The overall prevalence of dementia was not significantly different in the two studies, but the prevalence in men in the age group 70-84 was lower in the later study. Methodological differences could account for the variation as the time scale was too short to hypothesise cohort effects, although the authors did invoke the environmental hypothesis without suggesting which environmental factors had changed in 7 years.

There have been two other Swedish reports on this issue. Investigators from Gothenburg (25) examined population-based samples comprising several hundred 70-year-old individuals in 1976-77 and 2000-01, and 75-year-olds in 1976-77 and 2005-06, and found no cohort effects on dementia rates. Investigators in Stockholm (26) performed two cross-sectional surveys, the first in 1987-89 and the second in 2001-04, of people aged 75 and over in central Stockholm, and determined DSM-III-R dementia prevalence. They also used survival data to calculate incidence rates. They found that while the prevalence of dementia was stable, the survival of dementia patients had increased, suggesting that the incidence may have decreased.

The Rotterdam study (27) presented more methodologically rigorous data on this topic. It determined dementia incidence over 5 years in two sub-cohorts, the first starting in 1990 and the second in 2000, and noted that the incidence was marginally lower in the second cohort, albeit not statistically significant (rate ratio 0.75, 95% CI 0.56-1.02). Mortality was lower in the second cohort, despite the fact that the prevalence of hypertension and obesity had increased. The incidence of stroke was lower in the 2000 cohort, possibly due to the higher use of antithrombotic and lipid-lowering drugs. The 2000 cohort had more education and also larger brain volumes and less cerebral small vessel disease. A limitation of the study was the relatively small number of dementia cases in the 2000 cohort, limiting statistical power and precluding the examination to dementia subtypes.

A recent UK report was from the Medical Research Council Cognitive Function and Ageing Collaboration (MRC CFAS) (28). They performed their first dementia survey in individuals 65 years or older in 6 areas of England and Wales in 1989 – 94 (CFAS I) and repeated it twenty years later (2008-2011) in three of these areas (CFAS II). The sampling methods were comparable, albeit not the same. If the rates found in CFAS I were projected to CFAS II, after correcting for differences in population structure, the expected prevalence of dementia in 2011 was estimated to be 8.1%. The real prevalence was in fact found to be 6.5%, a decrease of 1.8%. Using these figures, the estimated number of people with dementia in the UK was 664,000 in 1991, and this figure was expected to rise to 884,000 by 2011. The actual number in 2011 was now estimated to be 670,000, a 24% reduction over the predicted figures. Sensitivity analyses suggested that the conclusion that later-born individuals had a lower risk of prevalent dementia was robust. The authors did not explore the possible reasons for this decline, but higher education levels and better control of vascular risk factors were suggested.

A Danish study (29) did not actually measure rates of dementia, but examined the health status of people in the early 90s. They compared the functioning of people in their early 90s who were born either in 1905 or 1915. The 1915 cohort, aged 95 at the time of assessment, performed better in their cognition and ability to function independently than the 1905 cohort, assessed at the age of 93. The physical health of the two cohorts was however very similar. This was despite the fact that those born in 1915 were more likely to survive to the early 90s, and were two years older than the earlier cohort at the time of assessment. The study suggested that more people born later were surviving to older ages and with better mental function and, by inference, lesser risk of dementia. The authors did not find definitive causative factors but argued that a more intellectually stimulating environment for the later cohort was the likely factor.

North American studies:

The incidence of dementia disorders was examined over 25 years (1960-84) through the Rochester

Epidemiology Program Project which has a records linkage system for the Rochester, Minnesota population in the United States (30). The overall age- and sex-adjusted incidence rates of dementia were relatively stable over time, except in individuals >85 years who showed an increase in this period. Dementia attributable to cerebral infarcts reduced from 10% to 5% in this period, arguably due to decline in stroke in this period. The proportion of dementia due to undetermined aetiology also decreased, possibly due to improved diagnostic practices.

Two studies from the United States, while methodologically less rigorous, have suggested a trend toward declining rates of dementia. The first study (31) assessed individuals 65 and over in the National Long Term Care Surveys, drawn from Medicare enrolment, in 1982 and again in 1999. The prevalence of those with severe cognitive impairment had declined from 5.7% to 2.9% in this period, attributed to a decline in mixed (VaD and AD) dementia but not AD dementia itself. The authors attributed the decline to better education and reduced stroke risk. The second study (32) used data from the Health and Retirement Study, a population-based longitudinal survey of US adults, to examine the cognitive status of individuals aged 70 and over in 1993 and 2003 using a 35-point cognitive scale or a proxy report. They found that cognitive impairment was present in 12.2% in 1993 and 8.7% in 2002, but those with impairment had a more rapid decline in the later cohort.

A study of incident AD from Chicago examined individuals ≥ 65 years old every three years from 1997 to 2008 (33). The analyses included 1695 participants, of whom 360 developed AD; there was no change in risk for AD over time (odds ratio = 0.970, 95% CI 0.902 -1.044).

One study (34) compared age-adjusted and overall prevalence of dementia in African Americans aged ≥ 65 years living in Indianapolis, USA in 1992 and 2001. Using a two-stage assessment process, the overall prevalence of dementia was 6.75% and 7.445% respectively, a non-significant difference.

Table 2: Studies examining time trends in dementia prevalence and incidence

AUTHORS	CITY, COUNTRY	MAIN OBJECTIVE	POPULATION STUDIED	MAIN FINDING
Hagnell et al, 1893 (22); Rorsman et al, 1986 (23)	Lundby, Sweden	Incidence of multi-infarct and senile dementia in 1947-57 and 1957-72	Whole population of Lundby	Re-examined data: no sig difference between two periods
Kokmen et al, 1993 (30)	Rochester MN, USA	Crude and age- and sex-adjusted rates of DSM-III dementia from 1960 to 1984	Rochester MN population aged ≥65 years, with record linkage	Dementia rates stable except in >85 who showed an increase
Manton et al, 2005 (31)	USA national survey	Severe cognitive impairment prevalence in people ≥65 from 1982 to 1999	National long-term care interviews through Medicare enrolment	Severe cognitive impairment declined from 5.7% in 1982 to 2.9% in 1999
Lobo et al, 2007 (24)	Zaragoza, Spain	Dementia prevalence 7 years apart in people >65	Two population based studies ZARADEMP-0 (n=1080) and ZARADEMP-1 (n=3715)	Prevalence 5.2% and 3.9% respectively (n.s.); lower prevalence in later study in men aged 70-84
Langa et al, 2008 (32)	USA national	Cognitive impairment in people ≥70 in 1993 and 2003	US Health and Retirement Study	Cognitive impairment rate 12.2% in 1993 and 8.7% in 2002; later group had faster decline
Hall et al, 2009 (34)	Indianapolis, USA	Dementia rates in African Americans in 1992 and 2001	African Americans aged ≥65 living in Indianapolis	No difference in prevalence of dementia despite differences in medical history
Herbert et al, 2010 (33)	Chicago, USA	Incident AD every 3 years from 1997 to 2008	Population based; 1695 individuals ≥65 years old evaluated	AD rates stable over time

Schrijvers et al, 2012 (27)	Rotterdam, Netherlands	Dementia incidence over 5 years in 2 cohorts, starting 1990 and 2000	Population based cohorts 1 (n=5727) and 2 (n=1769)	Incidence marginally lower in 2nd cohort (rate ratio 0.75; 95% CI 0.56-1.02)
Wiberg et al, 2013 (25)	Gothenburg, Sweden	Prevalence of dementia in 70 year olds in 1976-77 and 2000-01	Population based; several hundred in each sample	No differences in rates
Qui et al, 2013 (26)	Stockholm, Sweden	Surveys of people ≥ 75 in 1987-89 and 2001-04	Cross-sectional population based surveys	Prevalence stable, but later cohort had increase survival, suggesting lower incidence
Matthews et al, 2013 (28)	6 and 3 regions in UK, respectively	Dementia surveys of ≥ 65 in 1989-94 (CFAS I) and 2008-11 (CFAS II)	Population based surveys; some differences in methodology	Age-adjusted dementia rates 8.1% in CFAS I and 6.5% in CFAS II; a 24% reduction
Christensen et al, 2013 (29)	Denmark	Cohort effects on cognition and health over 10 years	Birth cohorts, born 1905 and 1915, and assessed at ages 93 and 95 respectively	1915 cohort cognitively & functionally better; similar in physical health

RATES OF NEUROPATHOLOGY

Another approach to answer the question whether the incidence of AD is decreasing with time is to examine if the burden of Alzheimer-type neuropathology is showing a cohort effect. A recent Swedish study (35) compared amyloid deposition in the brains of 1599 individuals who died at ages 65 or greater (mean age 82 ± 8 years) between 1972 and 2006 and were consecutively autopsied at the University Hospitals of Geneva. They found that the amyloid burden in the brain was related to the year of death, and decreased by 24% over the period over the period for the population of people without dementia. The reduction

was particularly marked in the oldest age group; people aged ≥ 85 years in 2006 had less amyloid deposition than those aged 75-84 in 1972. This was despite the fact that the prevalence of dementia, and the other pathology of AD – neurofibrillary tangles – increased in the study population in this period, suggesting that the more recent cohorts coming to autopsy were in fact sicker. The authors concluded that “the brains of older individuals in 2006 were 10 years younger from the point of view of amyloid deposition compared with people who died 30 years earlier.” (p. 330).

CONCLUSIONS

The data, in particular from recent European studies, do suggest that the age-specific prevalence and, by inference, incidence of dementia and severe cognitive impairment may be declining in HICs. The CFAS study (28) suggests that this reduction may be substantial and enough to significantly mitigate, but not totally offset, the effect of ageing of the population on overall prevalence. However, the data thus far should be considered to be preliminary and more evidence must be accumulated. The data, if one takes the Lundby and Rochester studies into consideration, also suggest that this may be a recent phenomenon, although again there are limitations in the evidence available. The phenomenon may, additionally, be as yet restricted to HICs. It would be interesting to acquire similar data from LMICs to see if the pattern of change is related to the demographic cycle. It is not clear whether the reduction is due to changes in VaD and mixed dementia, or rates of AD are also going down.

It is important to understand the underlying reasons for the change. The Rotterdam and Danish data suggest that these cohort effects may be related to education and more stimulating environments in the early period of life. If this is the case, one can expect continuing improvement in rates over the next few decades, as seen in the Flynn effect, but with eventual levelling off or even a decline. There are of course a

number of competing influences, in particular vascular risk factors, occurrence and optimal management of stroke, and physical inactivity that could influence the rates in either direction. As mentioned previously, rates of stroke declined in HICs up to the mid-1980s but there has since then been a levelling off. Rates in some LMICs may in fact be trending upwards. Rates of diabetes, obesity and the metabolic syndrome are increasing in many societies, while that of smoking have an opposite trend (36-39). These changes have not been reflected in cardiovascular health data from Western countries so far (36), but it may well be a matter of time. Ultimately, the trends could also impact on dementia rates.

The situation is therefore complex, but the immediate news from HICs is encouraging that dementia rates are modifiable and may be trending downwards. We need to further examine the reasons for these changes and try to exploit these for public health programs. We must also remain cognisant of the fact that a decline cannot be taken for granted as there are opposing factors in play as well. The overall message is that preventative public health and social strategies have an important role and governments should invest in such strategies as one major part of the fight against dementia.

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