Dementia: Facing the Epidemic

Alzheimer's Australia
Paper 18
September 2009

Presentation by Professor Constantine Lyketsos
The National Press Club, Canberra
ACKNOWLEDGMENTS

Alzheimer’s Australia would like to thank Professor Constantine Lyketsos for his presentation as part of Dementia Awareness Week.

Alzheimer’s Australia would like to acknowledge the support of the Australian Government for their continuing funding of Dementia Awareness Week.

The printing and distribution of this document has been made possible by support from the Australian Government Department of Health and Ageing. The opinions expressed in the publication are those of the authors and not necessarily those of the Australian Government.
Foreword

In recent years an important part of Dementia Awareness Week has been to invite an eminent overseas speaker as the guest of Alzheimer’s Australia to promote awareness of dementia in a series of public lectures across Australia. This has been made possible by funding from the Australian Government through the National Dementia Support Program and unconditional grants from Pfizer Australia.

This year, Alzheimer’s Australia was very fortunate to have Professor Constantine Lyketsos as our guest speaker. For the first time, we arranged for our guest speaker to address the Press Club with a view to ensuring that even greater numbers of people have the opportunity to hear from one of the leading international authorities on dementia.

The speech is wide ranging in explaining the nature of dementia, what is known about the pathology of dementia, the therapeutic strategies being investigated by the researchers, the importance of early intervention and the delivery of quality dementia care.

After 25 years of dementia research, a lot more is now known about dementia. There are exciting possibilities in dementia research in identifying those people most at risk of dementia and the therapeutic strategies that would make early intervention and prevention possible.

The enduring message of the wide ranging presentation of Professor Lyketsos is for increased investment in dementia research to reduce the number of people with dementia.

Glenn Rees  
Chief Executive Officer  
September 2009
Professor Lykestos is an active clinician, teacher, and researcher. Dr. Lyketsos is The Elizabeth Plank Althouse Professor in Alzheimer’s disease research, Vice Chair of Psychiatry at Johns Hopkins University, and Chair of Psychiatry, at Johns Hopkins Bayview. He also directs the Johns Hopkins Memory and Alzheimer's Treatment Center, which provides cutting edge care to large numbers of patients while facilitating the translational research mission for the field.

An expert in the care and treatment of patients with Alzheimer’s and related dementias, he has carried out pioneering work regarding the epidemiology and treatment of neuropsychiatric features of Alzheimer disease. His team is developing biomarker methods to accelerate treatment development for Alzheimer’s and designing and implementing innovative clinical trials for the treatment of Alzheimer’s. He also leads efforts to ensure the provision of state of the art Dementia Care to people with dementia in their community or assisted living homes.

His clinical expertise has been recognized by citation as one of America’s Top Doctors for several years and was recently cited as a Best Doctor In America. He has been honoured by election to membership of the American College of Psychiatrists, the American College of Neuropsychopharmacology, and as Distinguished Fellow of the American Psychiatric Association. Dr Lyketsos has authored over 250 publications, book chapters, and commentaries, and Guest Edited several Journal special issues. He is the co-author of Practical Dementia Care (with Peter Rabins and Cynthia Steele) and of Psychiatric Aspects of Neurologic Diseases: Practical Approaches to Patient Care (with Peter Rabins, John Lipsey, and Philip Slavney).
Thank you very much Ken (Randall, President of the National Press Club). It’s my great pleasure to be here with you today.

I thank Alzheimer's Australia for their invitation and the Australian Government for the funding that makes it possible for me to visit Australia during Dementia Awareness Week.

I should acknowledge too, the unconditional grant that Pfizer provide every year to Alzheimer's Australia for their awareness activities.

Governments in the last century tackled polio, cancer, heart disease and HIV/AIDS with passion and commitment.

My message is simple. We need the same passion and commitment to resolve the enormous challenges that dementia poses for our health care systems and for our society.

Let’s be clear. The dementia epidemic is upon us. The facts speak for themselves.

Earlier this week, Alzheimer's Disease International published the *World Alzheimer's Report*.

It was estimated in that report that there are 35.6 million people with dementia today. The numbers will nearly double every 20 years to 65.7 million in 2030 and 115.4 million in 2050.

In other words, by the middle of this century the number of people with dementia worldwide will be five times the population of Australia today.

In Australia, we know that by the middle of the century there will be over 1 million Australians with dementia. There are over 250,000 Australians with dementia today.

The social and economic consequences of this increase will be extraordinary.

This is not simply because of the sheer numbers of people who will have dementia, but because dementia is one of the most disabling of all chronic non communicable diseases.

The brain diseases that cause dementia, such as Alzheimer’s disease, develop decades before diagnosis. There will be millions of others with milder memory disorders or who are concerned about losing their memories.
Australia became the first country in the world in 2005 to make dementia a National Health Priority. It was an inspirational decision for the rest of us. But few others have since followed Australia’s lead.

Why is it that dementia has not been the focus of greater attention at the international and national levels?

Perhaps it has seemed like tomorrow’s problem. In fact, dementia is very much today’s problem. It is a problem not only for our parents, but for us, for anyone who hopes to live a long life. If you live beyond 85 you will have a 1/3 chance of getting dementia, and another 1/3 chance of caring for someone with dementia.

Maybe it is because dementia does not seem to kill as quickly as cancer or heart. But of course, dementia is terminal.

Maybe it is because dementia predominantly affects the elderly, and if we are honest, perhaps ageism is an important factor in the neglect of dementia.

Maybe it is because dementia is regarded as an inevitable part of ageing. Well of course it is not.

Maybe researchers like myself have failed to get across the important advances in research and the prospects for new therapeutic interventions. Or, about the effectiveness of currently existing therapies in helping patients and their carers: we cannot yet cure, but we can treat.

I am not a social commentator, but I can tell you about the positives of how we can tackle the dementia epidemic based on the evidence.

Before I get into the detail of what I have to say to you, let me emphasize my main messages.

First, we are within reach – maybe 5-20 years – of therapeutic interventions that will modify or slow the progression and onset of dementia. With greater investment in dementia research, those interventions might be available even sooner.

Second, we need a greater effort in applying what we now know from our research about diagnosis and management of dementia.

Third, researchers have demonstrated the benefits of good dementia care, but knowledge translation to those delivering care has been all too slow.

Fourth, we know how to reduce the risk of dementia. How can we better inform the wider community about what they can do to reduce their risk of dementia?

In short, I have no doubt that with determination we can reduce the prevalence of dementia and improve the quality of dementia care.

Let me give you the evidence for my well grounded optimism. In doing so, I will
First, define what we mean by dementia.

Second, explain what we know about the causes of dementia.

Third, outline strategies for therapeutic interventions that may delay the onset of dementia or modify its progression.

Fourth, talk about the elements of good quality dementia care.

Lastly, emphasise the importance of dementia research.

**So What is Dementia?**

Dementia is a clinical syndrome used to describe the symptoms of a large group of conditions that result in a progressive decline cognition. People associate dementia with loss of memory, but there are many other consequences, including decline in reasoning, communication skills and the capacity to organise daily life.

Dementia is associated with what clinicians call Behavioural and Psychological Symptoms of Dementia (BPSD). These symptoms vary greatly with the individual, but at some time every individual with dementia will experience depression, psychosis, aggression, apathy, or wandering.

Dementia can be caused by over 100 different diseases that affect the brain. These include neurodegenerative diseases such as Alzheimer's, Parkinson's or untreated hypertension, which slowly erodes brain tissue.

In fact, most cases of dementia are the result of a mix of different brain diseases, each contributing to the patient’s symptoms. We can say with confidence that Alzheimer's, together with vascular disease, account for well over 75% of all dementias.

While the main risk factor is age, dementia can affect those under 65. In Australia it is estimated that there are 15,000 people with younger onset dementia. The social and economic consequences for these young people, and their families, are particularly devastating.

Dementia can be distinguished from normal cognitive ageing through careful diagnostic evaluation by trained physicians. Dementia is usually preceded, sometimes over years, by mild memory loss or confusion but without affecting daily life in a major way. These precursor symptoms are known as mild cognitive impairment or MCI.

Dementia generally progresses over 5-8 years, sometimes as long as 20. Eventually patients with dementia become severely debilitated with little ability to communicate with their environment, verbally or otherwise. They typically become incontinent, unable to walk and require considerable effort to feed.

In the terminal stage which can last months, sometimes years, the patient is bed bound, non-verbal and totally dependent on others. Dementia accelerates death
through debilitation, by making its victim vulnerable to infection, aspiration and damaging falls, or through wasting away. In the United States as in Australia, dementia is estimated to be the fourth largest cause of death in older people.

The carers of people with dementia become isolated from their personal and social networks because they spend almost all their time supervising or caring, with precious few moments alone. Research has repeatedly shown that caring for someone with dementia is unlike any other carer experience.

Dementia care is more physically and emotionally overwhelming and as a consequence, it is more damaging to the carer’s health. In some cases lethal for carers as well. We must never forget, therefore, that any serious effort to care for people with dementia must include care for the carers as a central component.

What Do We Know About The Pathology of Dementia?

While we do not fully understand the details, research over the past 25 years has given us a wealth of knowledge about the changes that take place in the brain with dementia. We now understand that dementia is characterised by changes to the brain that precede the clinical picture by years if not decades. Put another way, brain-damaging processes unfold in the brains of large numbers of people without symptoms for years. Treating these asymptomatic people is the eventual goal of dementia prevention.

There are interesting parallels with heart disease. We know that years of high cholesterol, smoking and related factors damage blood vessels. Introducing cholesterol or other therapies at a late stage to those who already have loss of heart muscle tissue and other problems is likely to be ineffective.

So too with dementia. Once an individual has started to lose significant numbers of brain cells, therapeutic interventions are less likely to be effective. The message is that we need to intervene early before the damage is too great.

Let me give you a more medical description. Two thirds of people with dementia have what we call the “Alzheimer’s pathology” in their brains. This is a characteristic physical pattern in the brain consisting of amyloid plaques and neurofibrillary tangles around or within the nerve cells of the brain. In addition, tiny immune cells, known as the microglia, become highly active. These cells are normally responsible for “cleaning up” damaged tissue, but in Alzheimer’s disease they seem to be associated with substantial neuronal loss. These hallmarks of Alzheimer’s in the brain likely develop, as I have said, over a very long time.

Under the prevailing hypothesis – the amyloid hypothesis – researchers believe that this long process begins with the misprocessing of a protein. We all have the amyloid precursor protein or APP in our brains. But in Alzheimer’s disease, the protein is misprocessed to produce a toxic protein fragment known as Abeta1-42.

Over time, through steady production, Abeta1-42 accumulates in plaques outside nerve cells. These are the amyloid plaques we see under the microscope. Over time Abeta1-42 erodes the synaptic connections between the nerve cells and ultimately
kills them. How this happens is not clear. Possibly our immune system activates the microglia which, in their frustrated efforts to clear the plaques, produce large amounts of inflammatory chemicals near synapses that are also toxic.

As the synapses that connect different nerve cells slowly disconnect, chemical communication between nerve cells in the brain falters, leading to symptoms. The function of nerve cells is to communicate. A disconnected nerve cell activates a “self-destruct” signal, leading to neuronal death, or apoptosis. In the process of dying, neurofibrillary tangles appear. Although poorly understood, it is likely that very important, secondary mechanisms become activated and the process spreads through the brain.

This spread through the brain follows a predictable pattern. The cortex, or rational thinking part of the brain, is involved at first, eventually affecting cells that project from cortex into the deeper sub cortical areas. The most vulnerable part of cortex, the area which dies first, is the hippocampus which is central for memory. The dying process involves several neuro-transmitter systems, such as those that make serotonin, dopamine, and norepinephrine. The loss on these latter systems has significant consequences for the behavioural and psychological symptoms of dementia.

The particular symptoms a patient suffers depends upon the location of the brain damage. Patients with different causes of dementia may have similar symptoms because different diseases affect the same parts of the brain. Patients with Alzheimer’s and Pick’s Disease both have memory loss, disinhibited behaviours, sleep disturbance, etc., if their particular disease happens to hit the relevant brain areas and systems.

What Are The Possible Therapeutic Strategies?

The excitement of dementia research is that the evidence base generated in the last 25 years has enabled us to identify a number of strategies that offer hope for the treatment of dementia. Broadly speaking, these strategies fall into three categories:

1. Prevention or removal of amyloid formation (particular to the treatment of Alzheimer’s disease) using new compounds such as beta and gamma secretase inhibitors, and vaccination.
2. Modulation of known risk factors including lowering blood pressure, reducing oxidative stress or inflammation, and stroke prevention.
3. Increasing growth of connections between brain cells or growing new brain cells to replace those lost, using nerve growth factors, stem cells and drugs that increase neurogenesis.

In the case of Alzheimer’s disease, much of the recent focus has been on the biology of amyloid as a treatment target.

Since the toxic amyloid form of Abeta1-42 is made through misprocessing of APP by beta and gamma secretases, several pharmaceutical companies have developed beta and gamma secretase inhibitor medications that have progressed to human therapeutic trials.
Another line of treatments has emerged from the understanding antibodies can clear the amyloid within the plaques from the brain. This has been shown repeatedly in mouse models of Alzheimer's amyloid.

Medications for all of these strategies are in clinical trials. For all these approaches significant caution is necessary because of unknown safety concerns. For example, inhibiting beta and gamma secretases may run severe, yet unknown, risks since these enzymes have functions beyond the processing of APP. An early study of active immunotherapy years ago led to evidence that amyloid was cleared from the brain, but also to serious untoward effects that caused encephalitis, a brain inflammation, and death in about a dozen research participants with Alzheimer's.

While these anti-amyloid therapies appear exciting, it is very important that we remain cautious. We will be testing the amyloid hypothesis over the next decade. But we will be testing one aspect of it, namely that clearing amyloid in people with dementia can reverse symptoms. As with the heart disease example this is not too different from reducing cholesterol in somebody with heart failure. In the case of dementia the brain damage may simply be too much to be reversed. A sobering study followed people from the first vaccination study and reported that those who died did not have much amyloid but died with advanced Alzheimer like dementia.

The second strategy I referred to involves modulating factors such as high blood pressure, high cholesterol, oxidative stress, and inflammation, which we know are associated with the progression of dementia in the brain. Many medications involving these mechanisms have not been found helpful in people with dementia. Nevertheless, these treatments may help delay onset of dementia. Consequently, efforts are underway, involving medications, dietary change, exercise, and mental stimulation to examine the preventive value of these strategies.

In the meantime, while we cannot do much about ageing or our genetic makeup, we can do something about our lifestyle. There is good evidence that physical, mental and social activity together with good nutrition will assist some to reduce their risk of dementia. While there is no guarantee that if you do all the right things you will avoid dementia, lifestyle changes will undoubtedly benefit some. And it will help physical health as well as brain health, and is a course of action that can do no harm.

Until new therapeutic interventions are available, those concerned with public health should be encouraging people of all ages, and particularly those in their forties and fifties to reduce their risk of dementia through public education activities such as the Mind Your Mind ® Program developed by Alzheimer's Australia. It is cheap at any price compared with the cost of dementia both economic and social.

Lastly, I mentioned strategies to keep injured nerve cells from dying or to use stem cells. Research in this area is in its infancy and considered by many as “high risk—high pay off” as it may alter more fundamental disease mechanism. Substances have been identified that are natural nerve growth factors or otherwise “neuroprotective.” Delivering these to the slowly dying dementia brain, at the right time, will prove to be an enormous challenge but may produce huge payoffs. Substances which are neuroprotective in the test tube, however, have not been helpful if delivered in pill form to patients with dementia. Perhaps delivery into the brain by neurosurgical
means will be more effective—a trial is already starting in the US of a nerve growth factor injected directly into the hippocampus of people with Alzheimer’s dementia.

Regarding stem cells, many researchers are sceptical of these technologies. But the job of the researcher and clinician is to be interested in all possibilities. There is some evidence in mouse studies that stem cells may hold out promise. The hope is that we may be able to use stem cells to replace multiple cell types thus patching broken connections over parts of damaged brains. Scientists in Australia at the University of New South Wales, the Queensland Brain Institute and at the Monash Immunology and Stem Cell Laboratory are in the forefront of brain stem cell research, both in the development of new types of stem cells and also in testing them in brain disease.

The Importance of Early Intervention

The great challenge for us is how are we going to target and treat people in the early phases of the brain disease when there are no symptoms? Imagine a cascade that begins with amyloid and its processing, followed by deposition of plaques, synaptic disconnection, injury to neurons, tangle formation, death of neurons and death of neuronal systems leading to symptoms. We need to develop measures for each part of this cascade in living people.

There is recognition of the importance of these issues in the research effort being made to identify significant biomarkers that measure each part of the cascade. For example, we now have the ability to measure the deposition of amyloid in the brain using PET with two radio labelled molecules.

We are close to using PET radio labelled molecules to image activation of the brain’s immune system. Similarly we can measure accurately levels of amyloid and other characteristic features like tau protein in cerebrospinal fluid obtained through a spinal tap – a routine procedure that is increasingly becoming part of the research and care of people with dementia.

MRI brain imaging methods are being used to quantify the loss of structure and function in key parts of the brain, and may be directly able to image amyloid plaques.

Blood tests are also being developed. At Johns Hopkins we developing blood measures of lipids that might spill out of the brain as nerve cell membranes die off.

Very soon, we will be able to place a person in a single scanner for 2 hours to get an MRI and two PET scans measuring amyloid or microglia activation. This coupled with cerebrospinal fluid and blood tests will allows us to stage the process in anybody at risk, with signs of early mild cognitive impairment, or with dementia. By repeating these procedures we will be able to estimate the speed at which the process is evolving in any individual.

The HIV-AIDS field was revolutionized by the ability to measure viral load and count the CD4 receptor cells. Very soon we will have similar advances in the Alzheimer’s field allowing us to quantify the biological signature of the disease in individuals. Through this we will be better able to target treatments at stages when they are most
likely to be effective. For example, we will be able to target these designer anti-
amyloid medications early on in the amyloid period of the disease.

**Delivering Quality Dementia Care**

Let me now shift to discussion of dementia care. In 2005-2006 I had the privilege of
leading a task force of the American Association for Geriatric Psychiatry that
produced a position statement regarding principles of care for people with dementia.

The basic premise of this report is that there currently exists a systematic, evidence-
based approach for the delivery of cutting-edge care for people with dementia. One
of the major reasons for the report was our concern with the ongoing under-
recognition, under-diagnosis and under-treatment of dementia.

Discouraging studies showed that over 20 years, despite advances in knowledge and
public awareness, there has been little improvement in the USA in the detection and
treatment of dementia. In fact, primary care doctors in the US in the early part of this
century are no better at detecting and treating dementia than they were 20 years
ago, even though more effective therapies are available.

I understand that you have much the same problems in Australia.

The benefit of dementia care has been repeatedly demonstrated by significant
evidence. Callahan and collaborators in Indianapolis compared guideline-based
dementia care delivered in the primary-care environment to care-as-usual. They
found that patients with dementia and their caregivers did better and tended to live in
the community longer.

Mary Mittleman and her collaborators have shown that a simple carer-targeted
intervention for people living at home, one that makes available skills training, basic
education, and access to experts during a crisis was associated with a delay of
nursing home placement by about 18 months.

In the Maryland assisted-living study we showed that Dementia Care was associated
with staying 200 days longer in an assisted living facility. At about $150 per day that
is a $30,000/year difference. 2-3 million people in the USA live in these facilities,
about 2/3 of whom have dementia.

Most recently in the Netherlands a clinic-based Dementia Care intervention
demonstrated solid cost-effectiveness at a value of €1267 for each quality adjusted
life year (QALY).

The main message is that while we cannot cure dementia we can clearly care for it
effectively. It is essential that these effective treatments be delivered early, at home,
and comprehensively. The key elements of dementia care include treating aspects of
the disease as appropriate, introducing currently available medication treatments,
effective management of behavioural and psychological symptoms and, most
importantly, providing systematic support for carers and patients.

People with dementia need comfort and emotional support. Their safety must be
ensured through discontinuing driving when it's necessary, providing support for those who live alone, supervising medication administration and other safety measures. Daily structure is critical, as is supervisory care to assist them with functional losses in daily activities, such as dressing and grooming. Activity and stimulation is needed to avoid de-conditioning—social engagement, physical activity, and mental activity must be actively encouraged and maintained.

Medical co-morbidity, which is common in the elderly, with or without dementia, must be managed aggressively. Preventive primary care through vaccination, personal hygiene, restful sleep, hydration, nutrition, dental care, and management of co-morbid conditions such as diabetes, hypertension, etc, is critical.

Dementia carers require systematic support. While they need comfort, they especially need education about dementia and its progression. Most importantly they need professional support in the difficult journey that they face. None of us are born or trained to be carers.

Caring for a person with dementia requires specific skills in providing activities, overseeing medication, managing crises, and handling problem behaviours, all of which require availability and input from dementia care professionals. Dementia carers require respite that is tailored to their individual needs. Finally it's critical that dementia carers maintain their networks and personal lives as well as their own health and mental health maintenance.

In Baltimore we are engaged in the maintaining independence at home (MIND@HOME) project, in many ways modelled after work done in Australia. The aim of MIND@HOME is to engage whole communities in finding people with dementia in their homes, assessing their needs, and delivering needs-based dementia care to them and their carers so as to maximize their ability to stay home.

**Investing in Research**

The research agenda for the field is progressing along three fronts. One front is basic science using modern techniques of molecular biology, animal modelling and genetics, to produce a better understanding of the disease and candidate therapies. At the same time the clinical science of quantifying Alzheimer's disease in living people at different stages will allow us to introduce the appropriate therapies that the basic science delivers. Finally, while we wait for these major advances we must continue to push the envelope and maximize therapies that we already have, by making them available to as many as possible.

In the United States we currently spend close to US$1 billion a year in Alzheimer's related research. This may not be enough. If we are to find a cure for Alzheimer's, a major priority given the huge societal, economic and health costs, we must invest 10 to 20 times more over the next decade to rapidly accelerate the effort to find a cure.

In the last century governments responded to the challenges presented by polio, cancer, heart disease and HIV AIDS. We need the same passion and commitment to face the chronic disease challenges in this century presented by neurological diseases – of which dementia is the most important.
Australia has started to invest in dementia research from a very low base in recent times. Even so in your researchers have made seminal contributions to dementia research in key areas – Professor Colin Masters was among the first in the world to identify and isolate amyloid; Professor Brodaty has made important contributions to identifying the benefits of well designed carer programs, and Professor Jorm laid the basis for identifying risk factors. How much more could Australia contribute if dementia research was better funded!

A major advantage that Australian scientists have is a focused research infrastructure with little waste, which is able to produce cutting-edge research at a much lower cost.

Conclusion

In speaking to you today I deliver a message of hope and optimism. The scourge of dementia will be with us throughout our lifetimes. We are making great strides in our ability to understand its basic biology. If things work out right in the next 20-30 years we will have effective, tailored therapies for people with dementia that prevent the disease before symptoms.

We are, however, in a race against time. We are not making the sort of investment necessary to find the cure in time to alleviate the epidemic. Current realities are such that our ability to deliver the necessary investments is not supported by the will of the decision-makers or by the reality of the stressed economics times in which we live.

So I conclude as I started:

1. With greater investment in research we are within reach of therapeutic interventions that will delay the onset of dementia or modify its progression

2. We know a great deal about good quality dementia care but have been slow to translate it into practice

3. We have a way to go in early intervention because, quite frankly, diagnosis is poor with the consequence that dementia is grossly under recognized.

My hope is that Australia will continue to lead the way in giving dementia the priority it needs as the chronic disease of the 21st century.
Alzheimer’s Australia Publications

Quality Dementia Care Series
1. Practice in Residential Aged Care Facilities, for all Staff
2. Practice for Managers in Residential Aged Care Facilities
3. Nurturing the Heart: creativity, art therapy and dementia
4. Understanding Younger Onset Dementia
5. Younger Onset Dementia, a practical guide

Papers
1. Dementia: A Major Health Problem for Australia. September 2001
2. Quality Dementia Care, February 2003
3. Dementia Care and the Built Environment, June 2004
5. Legal Planning and Dementia. April 2005
6. Dementia: Can It Be Prevented? August 2005 (superceded by paper 13)
7. Palliative Care and Dementia. February 2006
9. 100 Years of Alzheimer’s: Towards a World without Dementia. August 2006
15. Dementia, Lesbians and Gay Men (in production)
17. Respite Care for People Living with Dementia. May 2009
18. Facing the Epidemic, Professor Lyketsos speech at National Press Club September 2009

Reports commissioned from Access Economics
The Dementia Epidemic: Economic Impact and Positive Solutions for Australia, March 2003
Delaying the Onset of Alzheimer’s Disease: Projections and Issues, August 2004
Dementia Estimates and Projections: Australian States and Territories, February 2005
Dementia in the Asia Pacific Region: The Epidemic is Here, September 2006
Dementia Prevalence and Incidence Among Australian’s Who Do Not Speak English at Home, November 2006
Making Choices, future dementia care: projections, problems and preferences, April 2009
Keeping Dementia Front of mind: Prevalence and Incidence 2009-2050

Other Papers
Dementia Research: A Vision for Australia September 2004
National Consumer Summit on Dementia Communique, October 2005
Mind Your Mind: A Users Guide to Dementia Risk Reduction 2006
Beginning the Conversation: Addressing Dementia in Aboriginal and Torres Strait Islander Communities, November 2006
National Dementia Manifesto 2007-2010
Dementia: A Major Health Problem for Indigenous People August 2007
In Our Own Words, Younger Onset Dementia, February 2009
National Consumer Summit Younger Onset Dementia Communique, February 2009
Dementia: Facing the Epidemic, a vision for a world class dementia care system, September 2009

These documents and others available on www.alzheimers.org.au
Visit the Alzheimer’s Australia website at www.alzheimers.org.au for comprehensive information about
• dementia and care
• information, education and training
• other services offered by member organisations

Or for information and advice contact the National Dementia Helpline on 1800 100 500
(National Dementia Helpline is an Australian Government funded initiative)