

Chair Professors

Public Lecture Series

Persons with Alzheimer's Disease and Their Caregivers

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Chair Professor of Psychology and Gerontology

The Hong Kong Institute of Education

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About the Author

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Sheung-Tak Cheng is Chair Professor of Psychology and Gerontology at the Hong Kong Institute of Education, where he also serves as the founding Head of the Department of Psychological Studies, and founding Co-Director of the Center for Psychosocial Health and Aging. He has published almost 100 articles in psychology and gerontology, and is an internationally recognized authority in gerontology, especially Chinese gerontology. He serves on the editorial board of *Psychology and Aging*, *Aging & Mental Health*, *GeroPsych*, *Journal of Applied Gerontology*, and *Research on Aging*. He was invited to write editorials for *International Psychogeriatrics* and to review Hong Kong's field of gerontology in *The Gerontologist*. He is chief editor of the book *Successful Aging in East Asia*, coming out later in the year by Springer, as well as special sections on 'Dementia and Dementia in Asia' and 'Asian Families and Well-being in Later Life' in *Aging & Mental Health*.



He is elected Fellow of the American Psychological Association (APA) and the Association for Psychological Science (APS). He was awarded the Outstanding International Psychologist Award by APA's Division 52 in 2007, and was honored in 2008 with the Certificate of Appreciation for Contributions as a Humanitarian Worker that was jointly presented by APA's Division 48 and Psychologists for Social Responsibility, both for his work in aging.

Due to personal reasons, Professor Cheng was unable to finish secondary school and took part in the public examinations through self-study. Following undergraduate education at the Chinese University of Hong Kong, he was educated at the State University of New York at Buffalo, where he received a Ph.D. specializing in Clinical and Community Psychology. There, he was heavily influenced by his mentor, Murray Levine, who told him that academics should retire as long as they ceased to be intellectually productive. In this lecture, we will learn that being intellectually active is one of the best ways to prevent dementia.

Professor Cheng's research has been supported primarily by the Research Grants Council, which has provided funding for several landmark projects, including a clinical trial on the effects of mahjong and Tai Chi on the cognition of people with dementia (he does not encourage playing mahjong for long hours or gambling), a longitudinal study of the social networks of community-dwelling older adults, and two long-term studies (one of which being a clinical trial) on Alzheimer caregivers. Recently, capitalizing on financial support from the Hong Kong Institute of Education, he has started neuroimaging studies in persons with mild cognitive impairment, with the eventual aim of identifying the neural basis of interventions that can lower the risk of dementia. Cutting across these investigations is a focus on support to older persons, especially family support and caregiving, as well as the limits of interventions to improve the conditions of persons with dementia as well as their caregivers. He believes that a research agenda with a broad focus (as opposed to a single-minded focus) has the best potential to address the complex issues faced by an aging society, because helping the patient means helping family caregivers, and vice versa. Recently, he has also received support from the Tai Hung Fai Charity Foundation to launch a project on pain management of older persons, for which he is deeply appreciative.

He is a member of the Working Group on Active Ageing, Elderly Commission, HKSAR Government. He also serves in various other consultancy roles to the Research Grants Council and non-government organizations. He was also a former consultant to the United Nations Programme on Ageing.

Persons with Alzheimer's Disease and Their Caregivers

Sheung-Tak Cheng

Abstract

The projected sharp rise in the prevalence of dementia has become one of the most significant public health issues around the world, and Alzheimer's disease is the most common form of dementia. Recent research suggests that the symptoms of Alzheimer's disease are preceded by pathological changes in the brain for as long as two decades. This period of symptom-free pathological changes has opened up a window of early diagnosis and treatment. Nevertheless, current research suggests that the greatest promise lies in lifestyle changes to tackle cardiovascular and metabolic risk factors and to promote cognitive reserve. Apart from finding cures, a more realistic goal is to compress morbidity through consistent application of healthy lifestyles. This lecture will highlight these issues and discuss relevant findings. Because Alzheimer's disease will unlikely be cured in the foreseeable future, our society cannot continue to ignore the needs of families and caregivers as we have done before. This lecture will also highlight the challenges for caregivers and families, and will describe an innovative intervention to promote positive mindset in family caregivers in Hong Kong. Prior knowledge of Alzheimer's disease is not required for this lecture.

Introduction

The phenomenon of global aging has been called both a “triumph” and a “challenge.” As much as it is a triumph of medical advances and public health over diseases, injuries, and malnutrition in many parts of the world, it also presents enormous economic, social, and health-care challenges to societies to sustain support to older persons. Accelerated aging of the population is often seen during periods of high rates of socioeconomic development which give rise to improvements in health care, hygiene, nutrition, living and working environment, and so on. As Asia has enjoyed rapid progress in socioeconomic development in recent decades, it is no surprise that Asia is a rapidly aging continent (Cheng, Chan, & Phillips, 2008; Cheng & Heller, 2009; Cheng, in press).

With global aging comes more and more people living into very old ages. At advanced ages, the risk of cognitive impairment and dementia increases significantly. Approximately one-third of persons aged 85 years or over have dementia and the majority are women. The World Health Organization (2012) has called dementia a “public health priority.” The number of persons suffering from dementia worldwide is estimated to increase from 36 million in 2010 to 66 million in 2030, and further to 115 million in 2050, representing an increase of approximately 80% every 20 years! The sharp rise of dementia cases in the next few decades has been referred figuratively to by some as the “dementia tsunami” (Cheng et al., 2011). The increase is much more accelerated in developing than in developed countries. For instance, China is estimated to more than double its dementia population from approximately 5 to 11 million between 2010 and 2030 (Cheng, 2012).

Alzheimer’s Disease

The most common form of dementia is Alzheimer’s disease (AD), accounting for about two-thirds of all the dementia cases (Fratiglioni, De Ronchi, & Agüero-Torres, 1999; Lobo et al., 2000). AD was described by Alois Alzheimer, a German psychiatrist and neurologist, more than a century ago. In the year 1901, Alzheimer came to work with a female patient by the name Auguste Deter who was 51 years old and presented with progressive memory loss and paranoid delusions, and later auditory hallucinations, aphasia (language impairments), apraxia (inability to execute purposeful actions), and agnosia (inability to recognize objects, smell, etc.). Mrs. Deter died in 1906. At autopsy,

her brain revealed massive atrophies; almost one-third of the cortical neurons were missing. In addition, there were two histopathological hallmarks: (i) patchy deposits of amyloid peptides called *plaques* found in the spaces between neurons (i.e., nerve cells) and (ii) aggregates of paired helical filaments within neurons called *neurofibrillary tangles*. Amyloid plaques and neurofibrillary tangles have since been recognized as the hallmarks of AD. The term “Alzheimer’s disease” was formally introduced by Emile Kraepelin in 1910 (Beach, 1987).

More specifically, plaques consist of compact sheets of beta-amyloid (in particular an isoform called $A\beta_{42}$ resulting from abnormal processing of the amyloid precursor protein) in its insoluble form and are believed to be neurotoxic, whereas neurofibrillary tangles are the result of hyperphosphorylation of the tau protein. Optimally phosphorylated tau is responsible for holding up the structure of microtubules which are part of the skeleton of the neuron as well as channels for intracellular transport (i.e., transporting substances such as nutrients along the neuron). Tau proteins which are hyperphosphorylated become unstable and the microtubules fall apart. As a result, neurons cannot function properly and eventually die.

As mentioned, Auguste Deter became symptomatic in her early 50s. Subsequently, professionals distinguish between late-onset (symptom onset after the age of 60 or 65) and early-onset cases; the latter account for just a few percentage of all AD cases and are usually due to gene mutations that may be inherited. Over 90% of AD cases are sporadic (i.e., not running in families), with onset in old age. The average life expectancy after symptom onset is about 6 years for people with AD (Brookmeyer, Corrada, Curriero, & Kawas, 2002; Fitzpatrick, Kuller, Lopez, Kawas, & Jagust, 2005; Knopman, Rocca, Cha, Edland, & Kokmen, 2003), although there are considerable variations across individuals.

It should be noted that finding plaques and tangles at autopsy does not necessarily mean they are the causes, or etiological factors, of the disease in AD patients. For instance, plaque accumulation may be a by-product rather than the actual toxic form (Kayed & Lasagna-Reeves, 2013; Klein, 2013). Moreover, abnormal amyloid processing, tau tangles, and synaptic loss may all be products of some underlying process such as oxidative stress and glial (the brain’s immune system) inflammation (Ingelsson et al., 2004). Nevertheless, current views on the development of sporadic AD are heavily influenced by the amyloid cascade hypothesis which postulates beta-amyloid deposition to be the distal etiological factor. This is followed by other biomarkers, such as hyperphosphorylated tau in cerebrospinal fluid, bilateral temporal-parietal hypometabolism on fluorodeoxyglucose positron emission tomography, and

hippocampal atrophy on magnetic resonance imaging much further downstream at times closer to symptom onset. Recent neuroimaging findings suggest that beta-amyloid depositions begin as early as two decades prior to symptom onset (Jack et al., 2010; Villemagne et al., 2013). Nevertheless, the amyloid cascade hypothesis remains a conceptual model of the time series of events leading to AD but does not necessarily identify the etiology until it is proven. One reason I say this is that the locations of amyloid deposition do not align well with the regional distributions of tangle formation, brain atrophy, and other pathological biomarkers (Ingelsson et al., 2004; La Joie et al., 2012; Villain et al., 2012).

The long latency between detectable amyloid deposition and symptom onset raises the question of whether early diagnosis and early treatment are possible (Chiu & Brodaty, 2013; Dubois, Gauthier, & Cummings, 2013; Dubois et al., 2010). Treatments are guided by theory and so researchers are aiming to use medications to stimulate antibodies against beta-amyloid, an approach called A β immunotherapy (A β being the short form for beta-amyloid). Preliminary findings suggest that such a form of therapy is successful in inducing the body's immune system to produce A β antibodies (Winblad et al., 2012). The idea is that if we can eliminate or significantly reduce beta-amyloid accumulation in the brain, we can prevent its toxicity from triggering the cascade of pathologies leading eventually to AD.

As much as I hope pharmaceutical interventions would work in treating AD, it will be a long way before we know whether immunotherapy can deliver the job for several reasons. I am not a biomedical specialist but I can share some of my thoughts which may or may not be agreed upon by researchers in the biomedical sciences. First, as with any medication, there are side effects and the risks may turn out to be difficult to manage. Second, it is unclear whether a safe dose of the vaccine can generate sufficient immune responses to fight the build up of beta-amyloid over many years. Third, if beta-amyloid build up is simply a by-product of an underlying disease process that is still unknown, the treatment will be ineffective in preventing the occurrence of AD. Finally, suppose we start a randomized controlled trial today, yet given the chronic course of the disease and the uncertain timing of the unfolding of different events along the cascade, it will probably be many years or even decades before we know whether the drug works and to what extent, not to mention the long process of investigations before a drug is considered safe to be used in humans. It is therefore possible that when the "dementia tsunami" hits, we will still be without an effective drug. This has led some researchers to consider nonpharmaceutical interventions as an alternative. Before I discuss plausible nonpharmaceutical interventions, let me briefly discuss the concept of cognitive reserve.

Cognitive Reserve

Cognitive reserve refers to the brain's ability to tolerate age-related and pathological changes without developing cognitive impairment (Fratiglioni & Wang, 2007; Stern, 2012). Cognitive reserve has been measured in terms of education years, occupational complexity, premorbid intelligence, and linguistic ability, all of which have been shown to lower the incidence of dementia or cognitive decline in longitudinal studies, even after controlling for neuropathology and genetic and biological risk factors (Karp et al., 2009; Shadlen et al., 2005; Snowdon et al., 1996). It is an important concept because it suggests that one way to tackle the risk of dementia is to build up cognitive reserve. The concept of reserve is not new to medicine at all. Many of our bodily functions, such as cardiopulmonary functions and muscle strength, all have reserve capacity. Such reserves enable us to maintain our functioning despite age-related declines.

Cognitive reserve has been put forth as an explanation for the modest correlation between pathological measures and cognitive performance. One way to investigate such associations would be to examine the extent of amyloid burden, tau tangles, brain atrophy, and so on in the postmortem brain, and correlate these with the patients' cognitive scores close to death. One famous study of this sort was the Nun Study in which many Catholic nuns donated their brains for research. Researchers found that even with significant amount of neurofibrillary tangles in the hippocampus and surrounding regions (responsible for memory and first affected in AD), more than 30% were not symptomatic – a condition we may call *Alzheimer's disease without dementia*. Even for nuns with tangles spread to the temporo-parietal and frontal neocortex (the parts of the brain controlling for advanced mental functions), 8% were asymptomatic (Snowdon, 2003). Another study of people with mild cognitive impairment (MCI; a condition less severe than AD but is expected to progress to AD) showed that plaque burden correlated moderately with measures of executive function but not with global cognition (Price et al., 2009). Other studies showed that cognition had only moderate correlations with the number of neurons and even the number of synapses in the hippocampus CA1 region (Scheff, Price, Schmitt, DeKosky, & Mufson, 2007; von Gunten et al., 2006). (A synapse is an intercellular space connecting two neurons and allow them to communicate with each other.)

Older adults with higher cognitive reserve are hypothesized to be able to cope with neuropathology through reserve capacity at the cellular, neuronal, synaptic, and circuit levels. As said, better reserve allows the brain to tolerate

pathological changes. What does it mean? First, given the same degree of pathology such as amyloid and tau loads, those with better reserve will remain asymptomatic for longer periods. An alternative way of saying this is: for people with similar degree of cognitive impairment, those with better reserve have had more neuropathology (Bennett et al., 2003; Roe, Xiong, Miller, & Morris, 2007; Roe et al., 2008; Roe et al., 2011; Rolstad et al., 2009). Second, once significant cognitive impairment becomes evident, decline is faster in those with higher education or occupational complexity (Andel, Vigen, Mack, Clark, & Gatz, 2006; Hall et al., 2007; Roselli et al., 2009), but education does not reduce the risk of mortality from AD (Bruandet et al., 2008; Qiu, Bäckman, Winblad, Agüero-Torres, & Fratiglioni, 2001). In other words, once disease processes take over the brain and reserve can no longer cope with the degree of pathology, the patients will show a rapid downhill course. Together, these two processes delay symptom onset and shorten disease duration (i.e., compression of morbidity).

The simplest model, called the passive or the static model, is one of brain volumetric reserve in terms of the number of neurons and synaptic connections. For instance, education was found to be associated with higher cortical thickness in specific brain regions in older adults with normal cognition, but *lower* cortical thickness in those with AD (Liu et al., 2012), especially in the temporo-parietal and frontal cortices, regions that are most affected in AD (Seo et al., 2011).

The reserve model based on microcellular processes is rather speculative at the moment and there are not a lot of data to support it. Some studies have found hypertrophy (i.e., increase in the size of the neuron, including the soma) in hippocampal and anterior cingulate neurons in asymptomatic AD, but hypotrophy in symptomatic AD, and MCI somewhere in between (Iacono et al., 2008; Riudavets et al., 2007). Hypotrophy is believed to be compensation at the cellular level and an attempt to forestall degeneration in response to AD pathology such as amyloid burden and tau tangles.

Finally, the most interesting aspect of cognitive reserve is the utilization of compensatory neural circuits. In this scenario, called the active or the dynamic model, alternative neural circuits are utilized to compensate for the disruption in preexisting networks. In fact, compensation exists in normal aging but is intensified with AD pathology. With age, there is a tendency to shift the load of cognitive processing from posterior to anterior regions in the brain (called posterior-anterior shift in aging [PASA]; Davis, Dennis, Daselaar, Fleck, & Cabeza, 2008). The anterior regions involved in compensation correspond to the prefrontal cortex that controls the most advanced mental processes broadly termed executive function (e.g., divided attention, planning, reasoning,

decision-making, inhibition, monitoring). The general idea, as articulated by the compensation-related utilization of neural circuits hypothesis (CRUNCH) and later the scaffolding theory of aging and cognition (STAC), is that with age-related declines in brain structure and function, a “crunch” due to cognitive load will occur in preexisting circuits and the brain would re-route circuits to the prefrontal cortex to “help” (Park & Reuter-Lorenz, 2009; Reuter-Lorenz & Cappell, 2008). Similar prefrontal compensatory activations have been reported in persons with MCI or early AD (Clément & Belleville, 2012; Grady et al., 2003; Woodard et al., 1998).

Using a technique called resting-state functional magnetic resonance imaging, our research group has recently demonstrated similar patterns in the default mode network in persons with MCI. The default mode network is deactivated when the brain attends to external stimuli but is activated when the brain is “at rest” or is engaged in task-independent activities such as self-referential thought. Without going into further details, the default mode network provides another way of looking into the brain functioning of persons with incipient AD. Compared with normal controls, person with MCI demonstrated decreased activity in the posterior regions but increased activity in the prefrontal cortex (Shi et al., 2013). We believe that these findings are related to the compensatory function of neural networks.

Lifestyle Interventions

The foregoing review begs the question: How do we build up our cognitive reserve? As a matter of fact, most cognitive skills started to decline around the age of 30 (Park et al., 2002), by which time our educational attainment has reached the peak. It turns out that the best way to develop cognitive reserve is to engage consistently in stimulating leisure activities, in particular physical and intellectual activities including aerobic exercise, mind-body exercise such as Tai Chi and yoga, reading and writing, playing chess, singing or playing instruments, solving puzzles, calligraphy, painting, and certain computer activities. Longitudinal studies have consistently shown that, even after controlling for neuropathology and genetic and biological risk factors, physical and cognitive activities could reduce the risk of AD or dementia by as much as 50% (Akbaraly et al., 2009; Buchman et al., 2012; Scarmeas et al., 2009; Lam & Chan, in press; Verghese et al., 2003; Wilson, Scherr, Schneider, Tang, & Bennett, 2007; Wilson et al., 2002). Passive cognitive activities, such as watching TV, had no effect or even an adverse effect (Rundek & Bennett, 2006), probably due to a sedentary lifestyle.

A few remarks are warranted concerning the effects of physical and intellectual activities on AD incidence. In epidemiological studies, physical activity tends to become statistically redundant (i.e., nonsignificant) when considered together with intellectual activity (Akbaraly et al., 2009; Leung et al., 2011; Sturman et al., 2005; Verghese et al., 2003; Wilson et al., 2007; Wilson et al., 2002), although there are exceptions (Buchman et al., 2012; Geda et al., 2012; Scarmeas et al., 2009). Moreover, intellectual activity, but not physical activity, is associated with the time-course pattern in cognitive decline consistent with the cognitive reserve hypothesis (i.e., delay of symptom onset and rapidity of decline afterwards; (Hall et al., 2009; Helzner, Scarmeas, Cosentino, Portet, & Stern, 2007; Wilson et al., 2010)). And although physical activity has been found to be associated with reduced amyloid load (Brown et al., 2012; Liang et al., 2010), the association is not consistently reported and seems to be, again, redundant when considered together with intellectual activity (Landau et al., 2012). Finally, some studies found that education was no longer a significant predictor of AD incidence or cognitive decline after controlling for cognitive activities (Hall et al., 2009; Lopes, Ferrioli, Nakano, Litvoc, & Bottino, 2012; Wilson et al., 2002). Taken together, the present evidence suggests that intellectual activity engagement is closer to the model of cognitive reserve than physical activity. The fact that people who are physically active also tend to be cognitively active may explain the lack of independent effect of physical activity in multivariate models (Cheng et al., in press).

Nevertheless, it would be a mistake to think that physical activity is not important. There are several reasons why physical activity cannot be ignored. First, physical activity (Cheng, Leung, & Chan, 2013; Miller, Balady, & Fletcher, 1997; Pattyn, Cornelissen, Eshghi, & Vanhees, 2013; Reddigan, Ardern, Riddell, & Kuk, 2011; Woo, Yau, & Yu, in press; Xu et al., 2012), but not intellectual activity, reduces other major risk factors for AD, namely hypercholesterolemia, hypertension, atherosclerosis, stroke, Type 2 diabetes, and impaired lung function (Guo et al., 2007; Martins et al., 2006). Levels of these risk factors at midlife predict the incidence of AD or dementia decades later (Guo et al., 2007; Kivipelto et al., 2005; Whitmer, Sidney, Selby, Johnston, & Yaffe, 2005) and so it is important to start physical activity as early as possible and maintain it over time. Second, physical exercise improves cerebral blood flow and vascular health, which benefits brain functioning (Churchill et al., 2002; Rogers, Meyer, & Mortel, 1990). Third, optimal physical activity is associated with the stimulation of growth factors such as the brain-derived neurotrophic factor (BDNF) which is neuroprotective and conducive to neuroplasticity (Cotman, Berchtold, & Christie, 2007; Foster, Rosenblatt, & Kuljiš, 2011). Third, like intellectual activity, physical exercise has been found to play a role in neuroplasticity (e.g., synapse proliferation and neurogenesis),

especially in the hippocampal formation, and in maintaining prefrontal/executive functions (Colcombe et al., 2006; Erickson et al., 2011; Foster et al., 2011; Kempermann, Gast, & Gage, 2002). Many physical activities, like yoga and tennis, are also cognitively stimulating and hence there may be common pathways between physical and intellectual activities in terms of protecting brain health (Kempermann et al., 2010).

While there have been plenty of randomized clinical trials on the effects of physical exercise on cognition (Baker et al., 2010; Colcombe & Kramer, 2003; Heyn, Abreu, & Ottenbacher, 2004; Lautenschlager et al., 2008; Muscari et al., 2010), whether exercise can actually lower the risk of AD has been uncertain until recently. Linda Lam has conducted an inspiring trial comparing the effects of a 24-form Tai Chi and a toning and stretching exercise (control) in community-dwelling older adults with amnesic MCI (mean age = 78 years). The exercises were learned and practiced in community centers for 4-6 weeks, and then the participants were expected to continue practicing at least three times a week of no less than 30 minutes' duration per session. Adherence to the program was monitored by center staff. The primary outcome measure was conversion to dementia after one year. Among the completers, 4% of the Tai Chi group, compared with 17% of the toning-and-stretching group, deteriorated to at least mild dementia, representing an 80% reduction in risk over the course of one year in multivariate models (Lam et al., 2011; Lam et al., 2012).

I have also made a contribution by testing the effects of Tai Chi and mahjong in nursing home residents (mean age = 82 years). This was, to the best of my knowledge, the first randomized trial involving cognitive leisure activities. Almost half of the participants had cognitive functioning at a level similar to Lam's study, which we labeled as very mild dementia; the rest had mild to moderate dementia. Mahjong and Tai Chi were practiced 3 times a week for an hour per session over a 3-month duration; they were compared with a simple handicraft group running on the same schedule as control. The Tai Chi was a 12-form Yang style practiced in a sitting position that was developed by Dr. William Tsang for frail individuals. At 9 months, both mahjong and Tai Chi participants maintained their global cognition while control participants deteriorated expectedly. Similar patterns were observed for other neuropsychological measures (Cheng et al., in press). Results did not differ by degrees of cognitive impairment. In addition, although Tai Chi and mahjong had similar effects on cognition, the latter appeared to be better in improving mood and lowering depression (Cheng, Chow, Yu, & Chan, 2012) which is also an identified risk factor for cognitive decline (Jeste, Depp, & Vahia, 2010; van den Kommer et al., 2012). (It is important to point out that we did not involve competition or gambling in the mahjong games in this trial.)

These two trials are important for several reasons. First, as randomized controlled trials, they provided definitive evidence about the effectiveness of cognitive and physical leisure activities. Second, they demonstrate that it is not too late to start engaging in stimulating activities even if you have not been active before. Third, we have now evidence that engaging in stimulating activities can actually lower the risk of developing dementia. Although Cheng's trial had half of the participants having clinical dementia, in light of the fact that intervention effects would be more muted in persons with more compromised neuronal integrity and network functioning, the results are revealing. Cheng's and Lam's teams are collaborating to run more trials to discover activities most likely to benefit prefrontal functioning as this is the brain region most responsible for compensatory cognitive reserve.

Taken together, research has suggested a lifetime accumulation of risk as well as protection in terms of the development of dementia in old age. First, early education, which may be partly affected by parental social class, leads to differences in occupational complexity (such as managerial versus unskilled jobs; Richards & Sacker, 2003) and enables higher levels of intellectual activity over the course of a lifetime. Education may act on AD/dementia incidence primarily through engagement in intellectually stimulating activities; a few epidemiological studies have found that education is not an independent predictor once the effect of intellectual activity is accounted for (Hall et al., 2009; Lopes et al., 2012; Wilson et al., 2002). Education also improves health literacy and a higher likelihood of adherence to a healthy lifestyle including diet low in saturated fat and physical activities. (Diet is also a predictor of dementia incidence but will not be covered in this paper.) A healthy lifestyle offers protection both physically and cognitively, against the development of dementia.

The message is clear: As people are living longer, the risk of dementia is increased. However, there is optimism in terms of compression of morbidity, if not prevention of dementia. Nevertheless, *we can only be as optimistic as the extent to which we adhere to a healthy and active lifestyle*. By opting for a healthier lifestyle, we not only take care of our own brain and body, but we also bless our family members as AD or dementia, once strikes, affects not only the patient, but also the family. It is to family caregiving that I now turn.

Family Caregiving

Caring for a family member with AD is a chronically stressful experience. The

round-the-clock nature of caregiving often leads to physical and emotional exhaustion, health problems, depression, cardiovascular diseases, sleep disturbances, and even early mortality (Aneshensel, Pearlin, Mullan, Zarit, & Whitlatch, 1995; Mausbach, Patterson, Rabinowitz, Grant, & Schulz, 2007; Pinquart & Sörensen, 2003b; Vitaliano, Zhang, & Scanlan, 2003). They are often called an invisible group because they are mostly outside of our formal healthcare and social service system. They carry the major burden of day-to-day care of relatives with AD with little assistance from the service sector.

Demographic Characteristics

Between 2009 and 2011, we recruited a total of 142 primary caregivers of relatives with AD from clinics, social service agencies and a community household sample to participate in a longitudinal survey. The caregivers were interviewed every six months over an 18-month period (four waves). Inclusion criteria were (a) a primary caregiver, (b) care-recipient meeting National Institute of Neurological and Communicative Disorders and Stroke–Alzheimer’s Disease and Related Disorders Association criteria for possible Alzheimer’s disease (McKhann et al., 1984), and (c) care-recipient aged 60 years or over. Exclusion criteria were the caregiver having <14 caregiving hours a week or cognitive impairment. Of the participants, 6% were caring for someone with very mild AD, 30% mild AD, 54% moderate, and 11% severe. Two-thirds of the caregivers were children, daughters-in-law, or granddaughter. (It is noteworthy that only 10 of the caregivers, or 7%, were daughters-in-law, whereas 16% were sons, further suggesting changing gender roles and family structure in our society.) The majority of the caregivers, disregarding generation, was women. Seventy percent of the child/child-in-law/grandchild caregivers lived with the care-recipient. They had been taking care of the relatives for an average of four years, and provided an average of 76 hours of direct and supervised care per week, with spouses doing more. A quarter had domestic helpers to assist them (Cheng, Lam, & Kwok, in press; Cheng, Lam, Kwok, Ng, & Fung, in press).

Factors Predicting Caregiver Burden and Depression

Burden refers to the perceived negative impacts due to caregiving, including exhaustion, distress, not having enough time for oneself, financial impacts, and the feeling of being “stuck” in the role indefinitely (i.e., entrapped). Across culture, behavioral and psychological symptoms of dementia (BPSD) are more predictive of caregiver burden than functional and cognitive impairment of the care-recipient (Pinquart & Sörensen, 2003a; Torti, Gwyther, Reed, Friedman, & Schulman, 2004), although cultural values may moderate the way they respond

to BPSD (Knight & Sayegh, 2010; Sayegh & Knight, 2011). BPSD refers to a range of noncognitive symptoms including wandering, hoarding, repetitive vocalizations, aggression, delusion, hallucination, anxiety, dysphoria, apathy, and so on. BPSD results from a combination of changes in the brain and environmental factors. For example, a noisy or unfamiliar environment can aggravate BPSD. Some patients may also get particularly confused and manifest more BPSD in the evening (i.e., sundowning).

Through confirmatory factor analysis using weighted least squares with mean and variance adjustment (WLSMV) estimator in MPlus (Cheng, Kwok, & Lam, 2012), we found that BPSD can be grouped into three major symptom clusters (i.e., syndromes) that reflect neurodegenerative changes and psychological reactions to these changes, namely, mood disturbance (apathy, depression, anxiety, sleep and appetite disturbance), psychosis (delusions and hallucinations), and overt behavioral problems (aggression, aberrant motor behaviors, disinhibition, and irritability). Almost 90% of persons with AD have at least one BPSD over the course of the disease (Gauthier et al., 2010; Robert et al., 2005).

Compared with U.S. caregivers (Chow et al., 2002), Hong Kong Chinese caregivers reported more overt behavioral problems and less apathy in the care-recipient. 42% of the care-recipients were reported to have aggression, and 49% had aberrant motor behaviors (e.g., pacing, wandering, hoarding). However, only 33% had apathy and 38% had depression. (In the West, apathy is the most common symptom.) Aberrant motor behaviors, delusions, hallucinations, and sleep disturbances were reported to be more common among those with moderate than with mild AD. Additionally, because overt behavioral problems were highly correlated with the other syndromes, we suggested that patient behavioral problems are primarily secondary to the other symptoms (i.e., arise as a reaction to other symptoms), and for this reason, are more prevalent throughout the course of the disease (Cheng et al., 2012).

We then examined the major factors accounting for caregiver burden and depression. Here, we were the first to examine the differential effects of the different BPSD syndromes, as previous studies typically examined all BPSDs as a whole. Besides the BPSD syndromes, we also studied the simultaneous effects of care-recipient variables, namely MMSE (a global measure of cognitive impairment), activities of daily living (i.e., functional impairment), and use of day care and psychiatric services; and caregiver variables, namely age, gender, employment, relationship to care-recipient, whether living together with care-recipient, caregiving duration, caregiving hours per week, assistance from domestic helper, and subjective health (Cheng et al., in press).

Our results confirmed the international literature that BPSD were the most important determinant of caregiver burden. But among BPSD, overt behavioral problems and mood disturbances were most predictive of burden. The care-recipients' memory-related problems (e.g., repeating the same questions again and again) had some effect on one of the burden measures, whereas their activities of daily living had no independent effects after the other factors were controlled. After controlling for burden and role overload as well, only behavioral problems accounted for caregiver depression. Together with the fact that behavioral problems were most often reported by the caregivers, it appeared that Chinese caregivers were particularly concerned with overt behavioral disturbances in their family members. This appears to correspond well to the Chinese's general tendency toward behavioral control. On the other hand, as suggested above, Chinese caregivers may be less sensitive to covert disturbances (e.g., apathy) than U.S. caregivers. Finally, as expected, the longer one spent in providing care (i.e., number of caregiving hours per week), the more burdened and depressed one was, suggesting that respite (i.e., taking a break from caregiving) should be an effective measure to reduce caregiver stress. Subjective health was also inversely related to burden and depression (Cheng et al., in press).

Positive Gains in Caregiving

Caregivers often report positive gains, such as becoming a stronger person, insights about hardship, shifts in priorities, a sense of purpose, a closer bonding with the care-recipient, and so on. Burden and positive gains appear to be two independent aspects of the caregiving experience. Positive gain is not simply the absence of burden (Kramer, 1997); in fact, it is hardly likely that a caregiver would achieve high positive gain without being placed in a challenging situation in the long term (Folkman, 1997). In other words, we believe that positive gains arise from the same situation that provokes burden of caring for relatives with dementia.

However, whereas burden has been well-researched and relatively well-understood, little is known about the determinants of positive gains in caregiving. For example, whereas BPSD are the primary determinants of burden in dementia caregivers, they are weakly or not related to positive gains (Gilliam & Steffen, 2006; Liew et al., 2010; Rapp & Chao, 2000). Kramer (1997) suggested that positive gains might be more related to caregiver than to care-recipient variables. In our study, we proposed self-efficacy to be a factor in positive gains in caregiving.

Caregiver Self-Efficacy, Positive Gains, and Burden

Self-efficacy refers to the belief that one is able to execute courses of actions to achieve desired goals in specific situations (Bandura, 1997). Caregivers with a higher sense of efficacy are likely to view caregiving tasks as challenges to be mastered, to recover quickly from setbacks, and to persist in finding ways to cope, whereas those low in self-efficacy may dwell on personal deficiencies and the consequences of failures (Bandura, 1997; Steffen, McKibbin, Zeiss, Gallagher-Thompson, & Bandura, 2002). We examined three aspects of caregiving self-efficacy, namely obtaining respite, responding to disruptive behaviors, and controlling upsetting thoughts (Steffen et al., 2002). Our theoretical model (Cheng, Lam, Kwok, Ng, & Fung, 2013) assumes that caregiver self-efficacy has moderating effects on the relationships between care-recipient conditions on the one hand, and both negative (i.e., subjective burden) and positive (e.g., uplifts, satisfaction) appraisals on the other. In other words, we postulated that high positive gain and low burden are most likely when caregivers high in self-efficacy confront *more* difficult challenges from BPSD. Our assumption is consistent with the notion that persons with high self-efficacy tend to see difficulties as manageable and hence would be less likely to view the situation as burdensome. The moderating role of self-efficacy on positive gains requires more explanation, as it has never been examined in the literature.

Some of the most commonly reported positive gains, such as becoming a stronger person and a sense of purpose, are clearly related to self-efficacy. However, such gains do not happen simply because one is given the role of a caregiver; someone with high self-efficacy still need to meet the challenges on a day-in, day-out basis for the insights to emerge (Folkman, 1997). Hence positive gains are arguably most likely when caregivers with high self-efficacy are confronted with high care-recipient demands such as difficult behaviors. Therefore, we hypothesized a moderating effect of self-efficacy on the relationship between BPSD and positive gains. Our model, depicting how self-efficacy lowers depression through two pathways involving both positive and negative appraisals, is consistent with the 2-factor model of caregiver appraisal by Lawton, Moss, Kleban, Glicksman and Rovine (1991). Our model also suggests that the lack of a consistent relationship between BPSD and positive gains may be due to the fact that the nature of the relationship varies by levels of self-efficacy such that the overall relationship is weak. In a series of carefully constructed regression models, our model was confirmed.

First, only self-efficacy in controlling upsetting thoughts had moderating effects on positive gains, burden, and role overload: When caregivers were efficacious in controlling upsetting thoughts, they experienced more positive gains and less

burden and overload when there were *more* BPSD to manage. Second, on top of burden and overload, only self-efficacy in obtaining respite from relatives and friends had a direct effect on depression. Third, Chinese caregivers were much less efficacious in obtaining respite than in controlling upsetting thoughts or managing disruptive behaviors (Cheng et al., 2013), and this aspect was similar to U.S. caregivers (Steffen et al., 2002). The difficulty of caregivers in obtaining respite from relatives may have implications for our family systems. Furthermore, the moderating effects of self-efficacy in controlling upsetting thoughts suggest the importance of designing interventions to equip caregivers with cognitive techniques to reduce negative thoughts and promote positive thinking (Cheng et al., 2012).

Who are Helping the Caregivers?

We are the first group to use a network mapping procedure to study the social networks of Alzheimer caregivers in detail (Cheng et al., in press). Participants were given a set of three concentric circles with a smaller circle in the center labeled “me.” Participants were asked to place (a) people so close that it was hard to imagine life without them into the inner circle, (b) people not as close but still very important to them into the middle circle, and (c) people not yet mentioned but nonetheless close and important enough to be included in the personal network into the outer circle. Additionally, we asked them whether there were people who were not placed in the three circles but who had been special to them because of helping them with caregiving tasks. This last question was added to ensure that people who were not traditionally considered as part of social network (e.g., a maid or social worker) but with whom one had developed emotionally meaningful ties were not missed. Only one individual was identified by this last question in the whole sample, who was grouped into the outer circle. For each network member, participants provided the following information: age, gender, relationship, and frequency of face-to-face and other methods (e.g., phone, email) of contact, emotional support provided, instrumental support (inc. respite) provided, frequency of negative interactions, and satisfaction with social support obtained. In the interest of space, I will only present the most striking findings. Unless otherwise mentioned, the data below represent network members aggregated across all levels of closeness (i.e., circle placement).

The caregivers reported an averaged 4.4 network members, much lower than the average network size (~11) for Hong Kong adults, regardless of age (Cheng, Lee, Chan, Leung, & Lee, 2009; Cheng, Li, Leung, & Chan, 2011; Fung, Stoeber, Yeung, & Lang, 2008). Seven spouse, 1 sibling, and 6 child (not in-law) caregivers were without any network member at all (i.e., not a single person important to them)! As expected, the most common network members of the

older (spouse and sibling) caregivers were children and daughters-in-law, whereas those of the younger caregivers were siblings, followed by children and parents. However, whereas the older caregivers had 78 sons and 78 daughters in total, only 37 sons (47%) and 55 daughters (71%) were included in their networks. Even for the younger caregivers whose children were small, only 45 (70%) of 64 sons and 40 (61%) of 66 daughters were listed as network members! They had severed emotional ties with even their own biological children, not to mention siblings, extended relatives in-laws, etc. Likewise, the number of friends and neighbors averaged less than 1 per caregiver!

The participants reported little instrumental support from the network members. However, caregivers generally felt that it was not as easy to get a network member to listen to their concerns as to receive the other forms of emotional support. Moreover, negative interactions were generally very low. Despite the relative lack of instrumental assistance, caregivers reported a high level of satisfaction with the support obtained. In view of their relatively small networks, it was possible that the caregivers had detached themselves from people with whom relationships were not that positive, and the networks might be further trimmed with age due to socioemotional selectivity (Carstensen, 2006).

Regression analyses were conducted to see to what extent social network characteristics predicted satisfaction with social support as well as positive gains and burden in caregivers. Controlling for other factors, those with more network members were more satisfied with their social support obtained, and reported more positive gains and lower role overload. In addition, consistent with the 2-factor model of caregiver appraisal and well-being, positive support from network members were associated with positive gains, whereas negative interactions were associated with burden and overload, beyond the effects of network size.

Comments. Dementia caregivers are chronically stressed. In traditional Chinese families, caregivers may start to shoulder the initial responsibilities in order to protect the collective well-being of the family, but nonetheless expect help from other family members when they need it. When the care-recipient's condition deteriorates and displays, for example, more BPSD, the caregiver may start to feel overwhelmed and expect other family members to share the responsibility. However, it may be here that problems often occur, as when the support was not forthcoming. Family members may not have a good grasp of the disease and the care-recipient's conditions as well as the caregiver's needs, or they may simply be unwilling to help for one reason or another, leading to consistent disappointment and a gradual detachment from each other, if not an abrupt severance of ties.

Intervention for Caregivers

In view of the above findings, we designed a new, multi-component intervention intended to help caregivers reduce stress and acquire more positive gains through cognitive restructuring (i.e., looking at the caregiving demands with different angles). We called this a *benefit-finding intervention*. It contrasts sharply with caregiver interventions in the extant literature which focus on education and helping caregivers problem-solve (such as managing behavioral problems) but not thought changing. In this intervention, psychoeducation is combined with cognitive reappraisal exercises in order to promote positive perspectives in caregivers (Cheng et al., 2012). The trial is ongoing but I will nonetheless describe the main features of this study.

We planned two parallel trials in order to find out how modes of delivery affect intervention effectiveness. Trial 1 was conducted in group settings (e.g., social centers, clinics), whereas Trial 2 was conducted in the caregivers' own homes where they received one-on-one instructions from our trainers. In each of these trials, the benefit-finding intervention was compared to two control groups representing variants of the traditional psychoeducation approach. The first control was a "pure" psychoeducation program (i.e., no cognitive restructuring). However, this program was, strictly speaking, not a treatment-as-usual control because it was far more in-depth and comprehensive than the typical psychoeducation programs offered locally. Programs offered by local service agencies tend to be brief (e.g., consisting of a few sessions) and to focus on information provision only. Thus, another, so-called "simplified" psychoeducation program aimed at providing general information but without addressing actual applications was included. Thus, there were three treatment arms in each trial.

Each treatment condition consisted of 12-14 hours of training/education over a period of eight consecutive weeks. In the group mode, this was accomplished by 8 weekly group interventions lasting 1.5-2 hours each. To facilitate interaction during group sessions, each group had a maximum of 10 caregivers. In the individual mode, there were four biweekly sessions of approximately 3 hours each conducted at the caregiver's home. The number of face-to-face contact hours between trainer and caregiver is held to be constant across the three intervention groups to control for possible dosage effects. Further to the baseline interventions, boosters aimed to refresh and extend learning will be provided for all three groups at roughly 14 months after the conclusion of the initial treatment. The specific contents of the training programs are available in Cheng et al. (2012).

For both trials, the primary outcomes were caregiver burden, cortisol, perceived benefits, subjective health, psychological well-being, and depression. The secondary outcomes were caregiver coping, and care-recipient's BPSD and functional impairment. All outcome measures, except cortisol, were obtained at baseline (0 month; T1), post-treatment (2 months; T2), and 6 (T3), 12 (T4), 18 (T5) and 30 months (T6). Salivary cortisol, collected using the Salivette 5 times during the day (immediately after awakening, 30 minutes post-awakening, 45 minutes post-awakening, and at 11:00 and 21:00) for two consecutive days, was obtained at T1-T4 only. This design allowed an assessment of the long-term effects of the interventions. The trial will not be completed until the end of 2014 but initial, qualitative findings will be presented at the lecture.

Overall Comments

In this paper, I have attempted to cover a lot of ground, from etiology of AD to disease compression/prevention to caregiving. Space does not permit thorough treatments of each of these topics but I hope that the talk can raise awareness of issues around dementia, our risk of getting AD, and improve, in general, 'dementia literacy.' We have seen that AD is a chronic disease that can span several decades. The disease process occurs long before symptom onset. The risk of developing cognitive impairment is higher as we get older and among those 85 years or above, 1 in 3 will suffer from cognitive impairment. However, not all of those with evident pathology will develop symptoms. I have argued that curing AD may not be a realistic goal in the foreseeable future; rather, compressing morbidity through engaging in a healthy and active lifestyle is a more pertinent public health goal. *By compressing morbidity, we will also compress the burden of caregiving.*

For this reason, much needs to be done to invest resources into research and services for dementia, especially in the pre-clinical phase. Services and protocols to assess older people with memory complaints for MCI are needed. It is not easy to change entrenched lifestyles, especially to do so in order to avoid a distant and probabilistic disease (Lam & Cheng, 2013). Hence much research is needed to identify the most cost-effective interventions to rehabilitate cognitive reserve, that is, things that are not only effective but are also acceptable to the general public and represent the least amount of changes in people's lives. Family support may be crucial to behavioral initiation as well as long-term adherence to lifestyle interventions (Lam & Cheng, 2013).

Because the prevalence of dementia doubles every five years until the age of 90

(Fratiglioni et al., 1999; Lobo et al., 2000), we can reduce the number of cases by half if we can delay onset by 5 years. There is optimism, but this optimism relies on collective effort as well as prompt actions by policymakers, who tend to have a near-term horizon, to be willing to bring dementia onto their agendas.

Another urgent issue concerns supporting family caregivers. In Hong Kong, public policies have been guided by the assumption of family self-reliance (Cheng & Mak, 2007; Cheng, Lum, Lam, & Fung, in press). The argument is that family functions would be undermined if there is too much government intervention (Cheng et al., in press). However, family functions are not as robust as before (Cheng, in press) and filial values with regard to sick care have declined (Cheng & Chan, 2006; Cheng & Chan, in press). We have seen that Alzheimer caregivers are often isolated and receive little support from their family and friends. Is it time to reexamine our deep-rooted assumptions about family functions and support for older persons?

In this connection, it is important to recognize that the mental health of family caregivers is a significant, yet unrecognized, public health issue. Allow me to be a bit provocative here: As a society, we have abandoned the family caregivers who shoulder the bulk of care for people with dementia, unpaid and with little support. More should be done to explore how social service agencies can empower caregivers. Aside from providing direct services, such as respite and caregiver training, more should be done to facilitate mutual emotional support among caregivers. One potential obstacle, and not a small one, is how to get Chinese people to open up themselves to strangers and talk not just about the burden of caregiving, but the loss of support from (immediate) family members. Undoing the myth of family robustness may not be something we want to do, but may be crucial to facilitate help-seeking. Having a relative with dementia and unconcerned family members may constitute a double stigma (Cheng et al., 2011) that deters seeking help (Corrigan, 2004). Thus, tackling stigma at the societal level is also necessary.

In addition, the potential impact of this pandemic on the family is worrisome. *Whether we can preserve our traditional family values will depend very much on whether this pandemic will divide families, or unite them.* Family counseling cannot be delivered too late; relationship mending is very difficult when family members have broken apart. Often, family members do not even see the need for family counseling or do not want outside intervention into private, family matters. Therefore, family work needs to be built into interventions for caregivers at the early stage. Communication among family members is facilitated when they share the same awareness of the needs of the patient as well as the physical and psychological demands of caregiving. Effective communication is the prerequisite for enhanced family support for the

caregiver.

If I may end this paper with yet another provocative comment: Dementia may be a problem, but it is our attitude toward it that is the bigger issue. We can avoid confronting our risk and hope someone else will get it, or we can start reexamining and changing our lifestyles as well as the lifestyles of our family members, colleagues, and friends. We can also look at caregiving not so much as burden, but as an opportunity to grow and learn and to spend more time with the ones we love. Tackling this dementia tsunami **starts with you.**

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As Co-Investigator

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An Interplay of Age, Contextual Factors, Team Process and Organizational Creativity in Hong Kong Professionals (2011-13), \$697,675, funded by Research Grants Council General Research Fund No. CityU143810. (PI: A Hui)

The Making of a Creative Hong Kong: Creativity for All Ages, and Age Integration in Creative Industries (2010-12), \$870,000, funded by Research Grants Council Strategic Public Policy Research Grant CityU1001-SPPR-09. (PI: A Hui)

Age Differences in Response to Negative, Positive, and Overly Positive Images of Aging (2009-11), \$575,281, funded by Research Grants Council General Research Fund No. CUHK4430/08H. (PI: H Fung)

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Guest Editorials

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Research excellence is characterised by systematic inquiry, scholarship, and knowledge transfer that supports innovation and development. With this philosophy, The Hong Kong Institute of Education endeavors to create space for dialogue and exchange to promote research in key areas relating to education and human development. This Lecture Series aims to share with the community together research issues that are relevant for the future. It will also identify areas where further effort is needed to strengthen the evidence base that informs policy and practice.