The New Zealand Longitudinal Study of Ageing

Summary Report

- Cognitive Functioning -

Lauren Callow & Fiona Alpass

2014

A research collaboration between

The Health and Ageing Research Team, School of Psychology, Massey University	The Family Centre Social Policy Research Unit	The Foundation for Research, Science and Technology

CONTENTS

LIST OF TABLES

LIST OF FIGURES

Introduction

The majority of New Zealanders aged over 65 (93%) live in private dwellings (Statistics New Zealand, 2007) and many have a strong desire to remain ageing in place (Davey, 2006). Admission to residential or nursing care facilities is generally regarded as undesirable, and is not only a financial cost to the individual and the state but is also associated with negative outcomes such as increased mortality (Wolinsky et al., 1992). Therefore, there is desire to prevent or delay admission to such facilities and maintain the independence of older people. One of the leading causes of institutionalisation is cognitive impairment (Luppa, et al., 2010).

Decline in Cognitive Function

Complaints of memory problems are common among older adults and can be a great source of concern (Hurt, Burns, Brown, & Barrowclough, 2012). Cognitive decline refers to a range of changes that may vary from mild (those associated with normal ageing) to severe where the ability to perform activities of daily life (e.g., paying bills, medical regimes, planning activities, dressing, bathing) is greatly impacted. With normal cognitive ageing a number of mental functions such as verbal ability, some numerical abilities and general knowledge show minimal decline (Deary et al., 2009). Other functions can decline from middle age on including memory, processing speed and executive functions. These changes may manifest themselves as increased forgetfulness, word finding difficulty, slowed reaction time and difficulty learning new tasks. When changes are greater than would be expected due to normal ageing and the individual's education level, but less than required for a diagnosis of dementia, and result in only mild functional impairment, they are categorised as mild cognitive impairment (MCI).

Prevalence

Determining population prevalence of cognitive impairment is difficult. The Health and Retirement study in the United States undertook an assessment of rates of cognitive impairment in a nationally representative sample (the AHEAD study) and found an estimated 6% of people aged 70 years and older in the community had moderate to severe cognitive impairment (Suthers, Jung, & Crimmins, 2003). Studies in the United Kingdom have estimates of impairment ranging from 2.3% (65-74 years old), 7.2% (75-84 years old) and 21.9% (85+), (Melzer, Ely, & Brayne, 1997). An early New Zealand study suggested that for people aged over 65 the prevalence of dementia was 7.7% and 30% for those over 85 (Campbell, McCosh & Reinken et al., 1983). Alzheimers New Zealand (2012) predicts that by 2050, 147,359 New Zealanders will have dementia – over 2.6% of the population, and more than triple current numbers, however prevalence rates for cognitive impairment in New Zealand are not yet determined.

Assessing Cognitive Function

Assessing cognitive function in older adults reliably and accurately provides the opportunity for early intervention and support. Often impaired cognitive function goes undetected or is not recognised. For instance, it has been estimated that only a third of those with dementia in New Zealand actually receive a diagnosis (National Audit Office, 2007). International studies have also found a significant proportion of dementia cases go undiagnosed in the community (Boustani et al., 2003; Sternberg, Wolfson, & Baumgarten,

2000) and undocumented in primary practice (Bradford, Kunik, Schulz, Williams & Singh, 2009; Cerhan et al., 2007). Given that measures of cognitive performance can be significant predictors of a dementia diagnosis (Powell et al., 2006), the need for early and accurate screening is apparent. As noted earlier cognitive functioning is thought to exist on a continuum, in which some individuals may experience normal cognitive ageing, then MCI which then transitions into dementia. This progression highlights the need for assessment over time as studies have shown that subtle changes can be measured even over short periods, especially in the oldest age groups (Albert et al., 2007).

Predictors

The APOE4 allele of apolipoprotein E (APOE) is the major genetic risk factor for Alzheimer's disease (Kim, Basak & Holtzman, 2009). Those with this allele of the gene are more likely to suffer early death, cardiovascular disease, stroke and Alzheimer's dementia (Anstey & Christensen, 2000). It occurs in approximately 25% of the population and has been shown to be a reliable predictor of MCI, with a frequency of 32% in MCI subjects versus 20% in healthy control subjects (Brainerd et al., 2011).

Age is the strongest predictor for cognitive decline (Salthouse, 2009). One prospective study found that cognitive scores declined significantly over 10 years in those aged 45 to 70 at baseline and decline was faster in older people (Singh-Manoux et al., 2012). Rates of dementia also increase with age as can be seen from Figure 1. Berra, Wancata and Ritchie (2005) found prevalence rates for dementia from studies in the European Union ranging from 1.2% to 4.7% for those aged 65 to 74 years, 4.5% and 18.3% for those aged 75 to 74 years and 11.5% to 39% for those 85+.

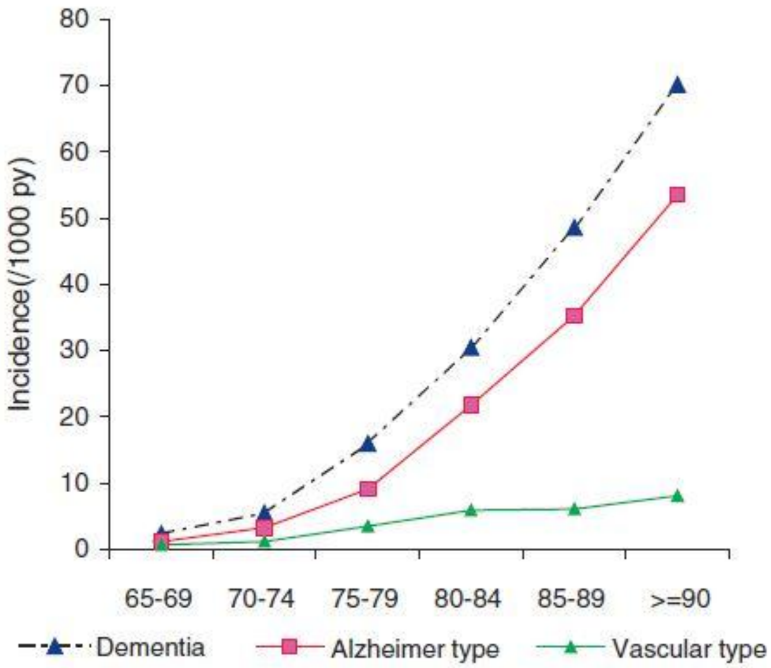

Figure 1. Incidence of all dementias, Alzheimer and vascular type of dementia (per 1000 person-years): pooled analyses of 8 studies from the EURODEM group (Fratiglioni et al., 2000, reproduced from Berra, Wancata & Ritchie 2005).

Cognitive functioning

Although these studies are strongly suggestive of cognitive decline with advancing age, it should be remembered that there is considerable heterogeneity within older aged cohorts in the rate and type of cognitive decline. In addition, there are of course those elderly who do not develop cognitive impairment as a result of ageing. Other age related variables such as health and social engagement may also partially account for cognitive decline with age.

There is considerable evidence that physical activity has a protective effect against cognitive decline and dementia. In one large prospective study, higher levels of physical activity were associated with a reduction in cognitive decline, with more active and regular exercise having greater protective effects (Albert et al., 1995). In a recent meta-analysis of 16 prospective epidemiological studies of physical activity and dementia, Hamer and Chida (2009) found that physical activity reduced the risk of dementia and Alzheimer's disease by 28% and 45% respectively.

Numerous studies have linked educational attainment to reduced cognitive decline with age (e.g. Albert et al., 1995; Lee et al., 2003). The risk of dementias in old age is also reduced in those with higher education levels compared to those with lower levels (Wilson et al., 2009). One mechanism proposed to account for this effect is cognitive reserve. That is, those with higher educational attainment may still decline but from a higher point before meeting an impairment threshold compared to those with lower educational attainment. Another possibility is that education may be a proxy for environmental experiences that impact on cognition, (e.g., health behaviours, SES). Additionally, more highly educated people may perform better on tests as they may be more used to tests and testing situations.

A number of studies show that loneliness is a risk factor for cognitive decline (e.g., Hawkley & Cacioppo, 2010; Wilson et al., 2007). Seeman et al. (2001) found levels of emotional support were related to better cognitive functioning and were protective of cognitive ageing longitudinally. Krueger et al. (2009), using multiple measures of social engagement found that more frequent participation in social activities and a higher level of perceived social support were associated with higher levels of cognitive functioning in older adults.

Physical health also plays a role in maintaining cognitive health. A number of age-related conditions have been shown to be related to cognitive decline such as cardiovascular disease, obesity, hypertension and diabetes (Kivipelto et al, 2001; Kumari & Marmot, 2005). In addition, the use of some medications to treat these conditions is being investigated for their potential to prevent or delay cognitive decline (e.g. statins). Health behaviours (in addition to physical health as noted above) have also been linked to cognitive health with smoking and alcohol consumption receiving attention (Anstey, Mack, & Cherbuin, 2009; Anstey, von Sanden, Salim & O'Kearney, 2007).

There is increasing evidence that depressive symptoms are associated with poorer cognitive functioning performance (Bunce, Batterham, Mackinnon, & Christensen, 2012; Elderkin-Thompson et al., 2003; van den Kommer et al., 2012), dementia (Lyketsos et al., 2000) and higher risk of cognitive decline (Modrego & Ferrandez, 2004; Rosenberg et al., 2010). It may be that depression is an early symptom of cognitive decline (Rosenberg et al., 2010) or lapses in cognitive abilities may lead to despondency and subsequent depression. In addition, the perceived inevitability of cognitive decline may result in less attention being paid to mental health (Bassuk, Berkman, & Wypij, 1998).

Anxiety may also lead to cognitive difficulties. Higher levels of anxiety are associated with difficulties with concentration, working memory and processing information, (Hertzog, Van Alstine, Usala, Hultsch, & Dixon, 1990). There is possibly a curvilinear relationship between anxiety and cognitive performance such that mild levels of anxiety are beneficial when performing cognitive tasks but high levels of anxiety interfere with performance (Bierman et al., 2005b).

The Current Study

In the two NZLSA waves a subsample of participants completed Addenbrooke’s Cognitive Examination Revised (ACE-R, Mioshi et al., 2006) as part of a battery of scales and items used in face-to-face interviews. Other measures included questions relating to demographics, income and assets, depression symptoms and anxiety symptoms. Interviews took approximately one hour to complete.

The ACE-R has been modified for use with New Zealanders, (the 'Kiwi' ACE-R; Taylor, 2008) and permission was obtained from the developers to use the modified version in the NZLSA face-to-face interviews. In accordance with suggestions from the developers, more site specific anterograde, retrograde and delayed recall memory components were modified to make the ACE-R more culturally acceptable. For example, using a New Zealand address in memory tasks and recalling the current New Zealand Prime Minister rather than the President of the United States of America. Other countries have also followed these guideline changes and have found little change to the psychometric properties of the measure (Alexopoulos et al., 2007; Garcia-Caballero et al., 2006; Konstantinopoulou et al., 2010).

The face-to-face sample was recruited through the 2010 postal survey participants who volunteered to be interviewed. The 2010 face-to-face sample study is comprised of 1,001 participants with 44.2% male. Age ranged from 49-84 years with a mean age of 64.44 (sd=7.77). the majority were European New Zealanders (68.5%) with a large proportion of Māori (25.8%) largely due to oversampling for the postal survey. Just over 70% were married or partnered and nearly a quarter had tertiary qualifications (22.8%). See Table 1 for a comparison with the 2006 census information.

Compared to the general population aged 45-84, the current sample that volunteered were more highly educated, under-sampled in the 45-54 age group and 75+ age group and had a greater proportion of people in the 60-64, 65-79 and 70-74 age groups. Pacific Peoples and Asian ethnic groups were under represented.

Table 1.
Demographic description of the NZLSA 2010 Face-To-Face sample and the general population aged 45 to 84 years from the 2006 Census.

	% NZLSA sample aged 49-84, N=1001	% General population (2006) aged 45-84, N=1,453,194,
Sex		
Male	44.2	47.6
Female	55.8	52.3
Age		
45-54	12.3	38.5
55-59	14.9	16.8
60-64	22.9	13.2
65-69	20.6	10.9
70-74	17.4	8.4
75-79	6.3	7.2
80-84	3.2	5.0
Ethnicity		
European	68.5	67.7
Māori	25.8	7.5
Pacific Island	0.7	3.1
Asian	0.6	5.4
Other	4.4	12.4
Missing	-	3.7
Marital Status		
Married	63.5	61.4
Civil Union/De facto	7.1	-
Divorced/Separated	10.8	15.4
Widow or Widower	11.3	9.7
Single	7.3	7.3
Missing	-	6.1
Highest Qualification		
No Qualifications	17.1	30.2
Secondary School	22.4	26.9
Post-Secondary /trade	38.7	16.8
University Degree	21.8	7.9
Missing	-	18.2

ACE-R Scores

Total ACE-R scores and subscale scores are presented in Table 2. Based on the suggested ACE-R cut-off scores for cognitive impairment in the original development paper, (<82: sensitivity = 0.84, specificity = 1.0), 36 people (3.6%) would be classified as cognitively impaired. Rates of impairment increased in the older age groups. Percentages of participants that scored below the cut-off for each age group were : <55 (2.4%), 55-64

(2.0%), 65-74 (4.4%), 75+ (10.5%). Total ACE-R score correlated highly with all of the sub-domains; Pearson correlations are shown in Table 3.

Table 2.
ACE-R total and sub-domain scores (N=1,001).

Domain (points available)	Minimum	Maximum	Mean (SD)
ACE-R total (100)	56	100	93.07 (5.47)
Attention/Orientation (18)	12	18	17.82 (0.60)
Memory (26)	5	26	23.71 (2.72)
Verbal Fluency (14)	0	14	11.33 (2.16)
Language (26)	13	26	24.91 (1.58)
Visual-spatial (16)	10	16	15.30 (1.10)

Table 3.
Pearson's correlations of the ACE-R total and sub-domain scores.

	ACE-R	Attention/ Orientation	Memory	Verbal Fluency	Language	Visual-spatial
ACE-R	1	0.37	0.78	0.72	0.67	0.46
Attention/ Orientation		1	0.20	0.18	0.27	0.07*
Memory			1	0.33	0.33	0.19
Verbal Fluency				1	0.35	0.21
Language					1	0.25
Visual-spatial						1

All correlations at p<.001 except * at p<.05

Demographics[1]

Total ACE-R scores differed significantly by age as expected, with scores declining with age, p<.001, Figure 2. Māori had slightly lower scores (m=91.80, sd=6.39) compared to non-Māori (m=93.4, sd=5.11), p<.05, however, when controlling for economic living standards this finding became non-significant indicating that differences were due to economic inequalities. Women (m=93.89, sd=5.10) had slightly higher scores than men (m=92.66, sd=5.21), p<.001. There was a clear educational gradient for scores on the ACE-R with higher educational attainment related to better cognitive functioning, p<.001, see

[1] A weighting variable was applied to the following analyses to account for oversampling of Māori.

Figure 3. ACE-R scores differed significantly by economic living standards but only between the "Fairly Comfortable" group and the "Good" and "Very Good" groups, p<.01, see Figure 4.

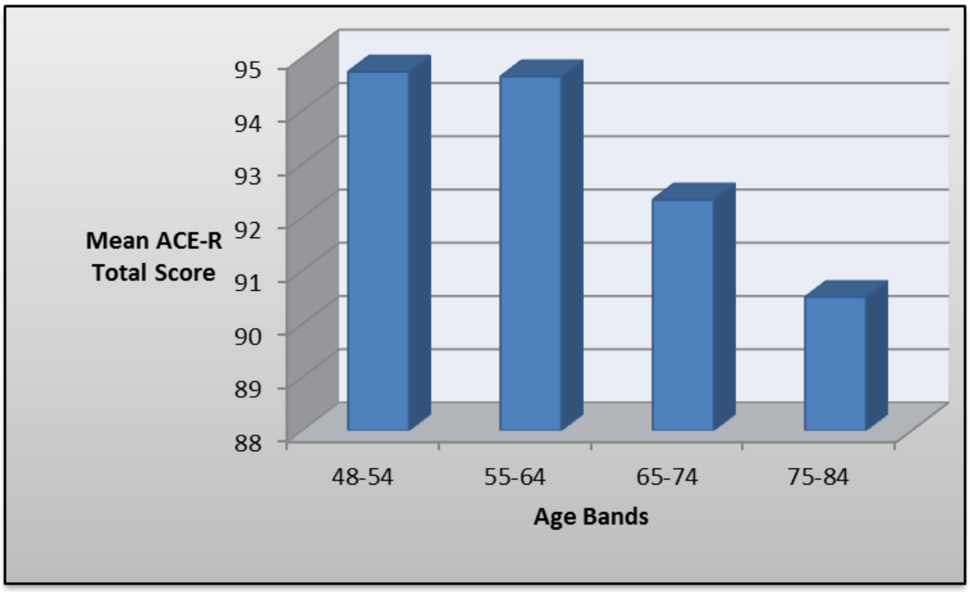

Figure 2. Mean ACE-R total scores by age bands.

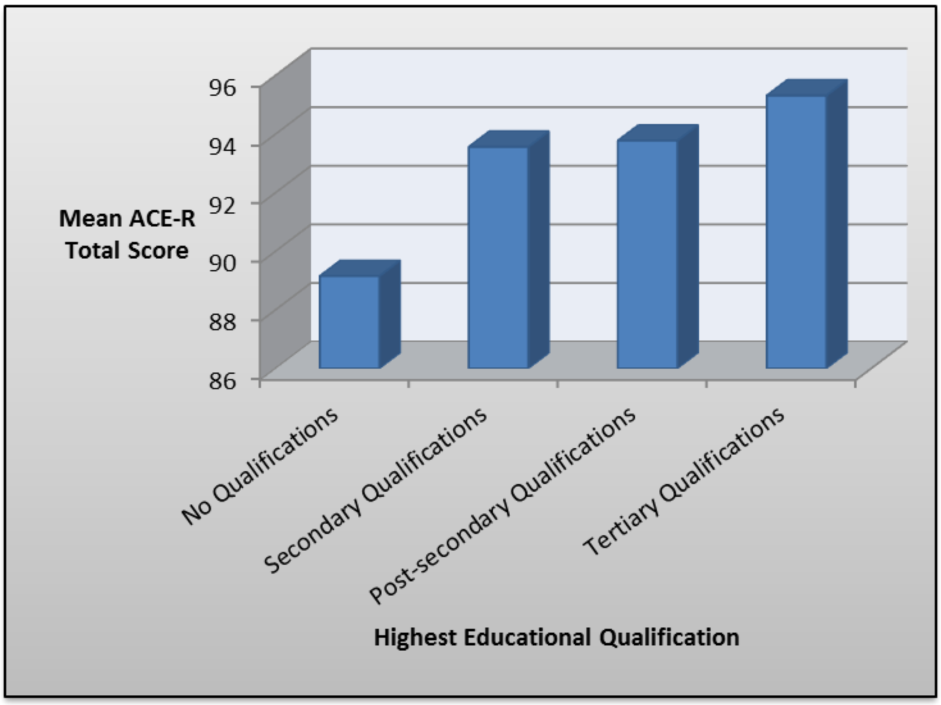

Figure 3. Mean ACE-R total scores by highest educational qualification.

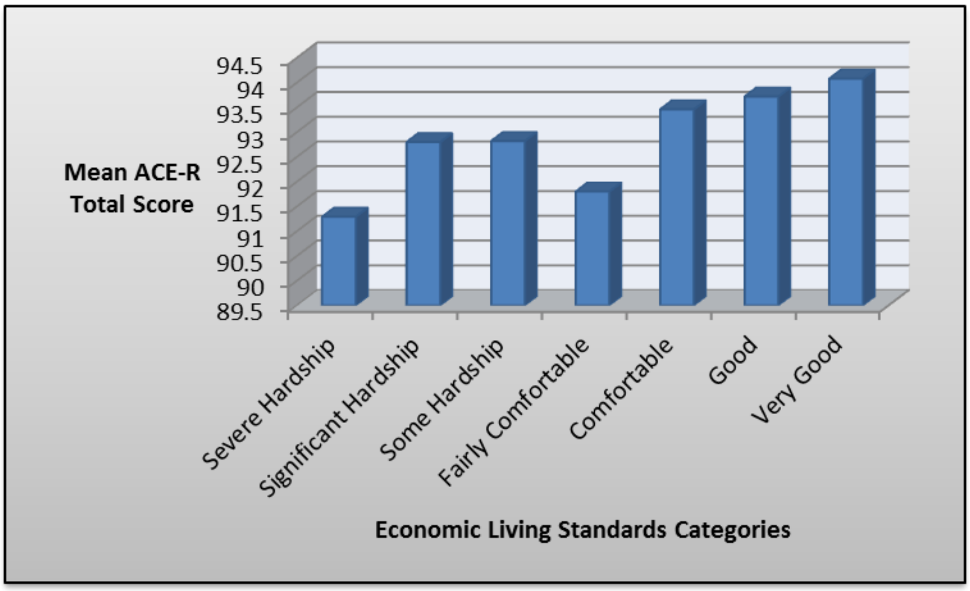

Figure 4. Mean ACE-R total scores by economic living standards categories.

Health, Health Behaviours and Wellbeing

ACE-R scores differed significantly between those diagnosed with four chronic health conditions compared to those who were not: diabetes, p<.001; heart disease, p<.05; hypertension, p<.01; and stroke, p<.01 (see Figure 5). Comparing hazardous drinkers (n=472) to non-hazardous drinkers (n=441) there were no differences in cognitive status. Current smokers (n=185) scored significantly lower on the ACE-R compared to lifetime non-smokers (n=522) and previous smokers (n=282), p<.001 (see Figure 6). Engaging in more frequent moderate physical activity (e.g., gradening, brisk walking) was related to higher ACE-R scores but the significant difference was only between those engaged in activity more than once a week compareed to those engaged once a week, p<.01 (see Figure 7). Those that met the criteria for clinical depression on the CES-D10 (scores >9) scored signficantly lower on the ACE-R compared to non-depressed participants, p<.001 (see Figure 8). Using a cut-off score of 8/9 for the Geriatric Anxiety Scale (sensitivity of 73% and specificity of 80%; Cheung, 2007), 105 (10.5%) identified as having an anxiety disorder. This group scored lower on the ACE-R compared to the “not anxious” goup (Figure 8). Finally, Ace-R scores were significantly positively related to SF12 physical health component scores, r=.245, p<.001, but were unrelated to SF12 mental health component scores.

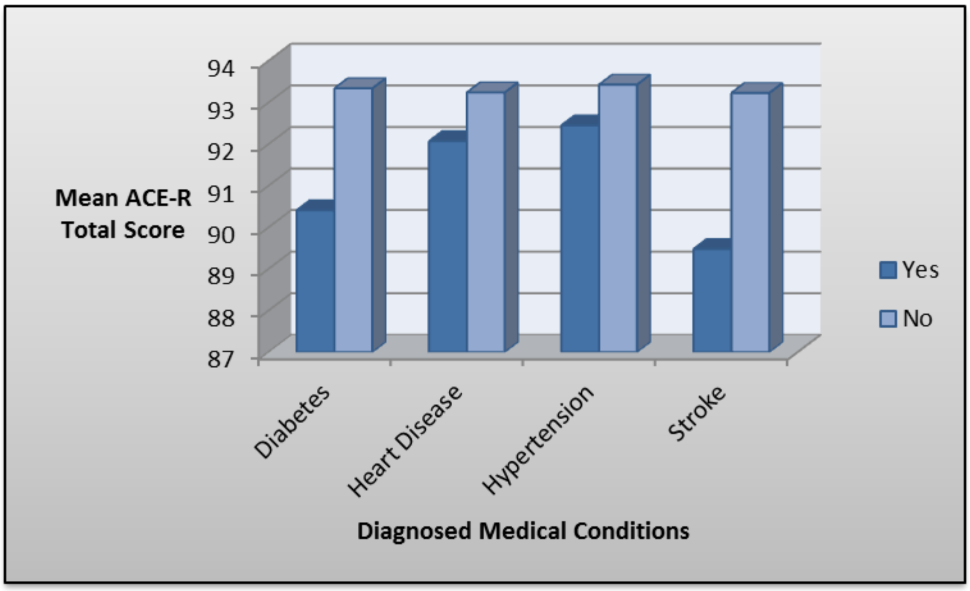

Figure 5. Mean ACE-R total scores by diagnosed medical conditions.

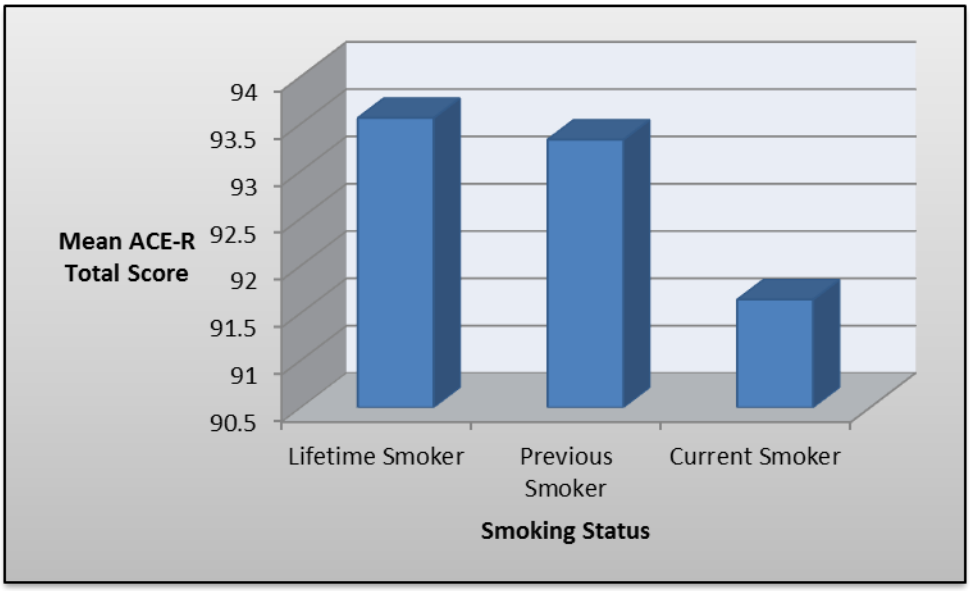

Figure 6. Mean ACE-R total scores by smoking status.

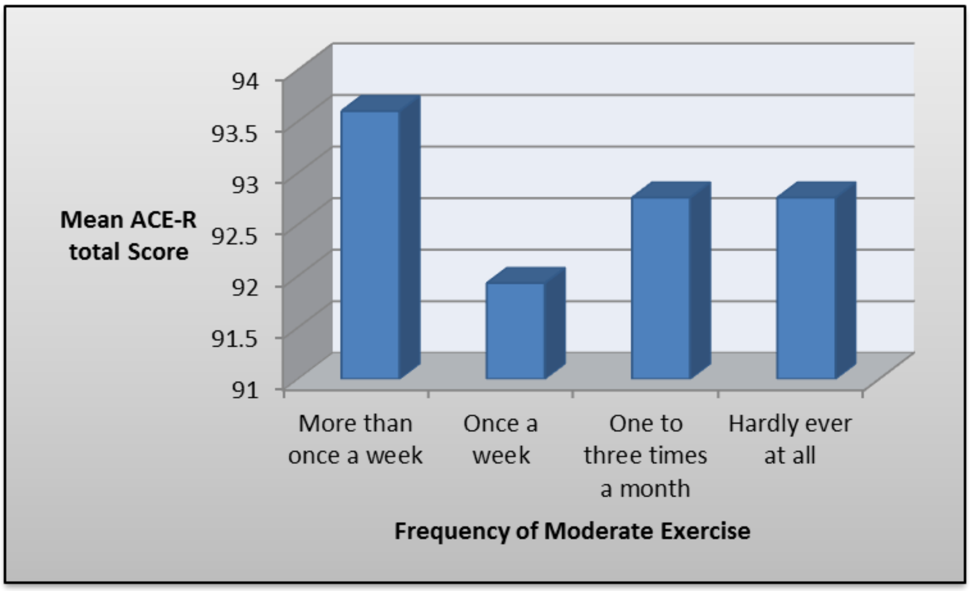

Figure 7. Mean ACE-R score by frequency of moderate exercise.

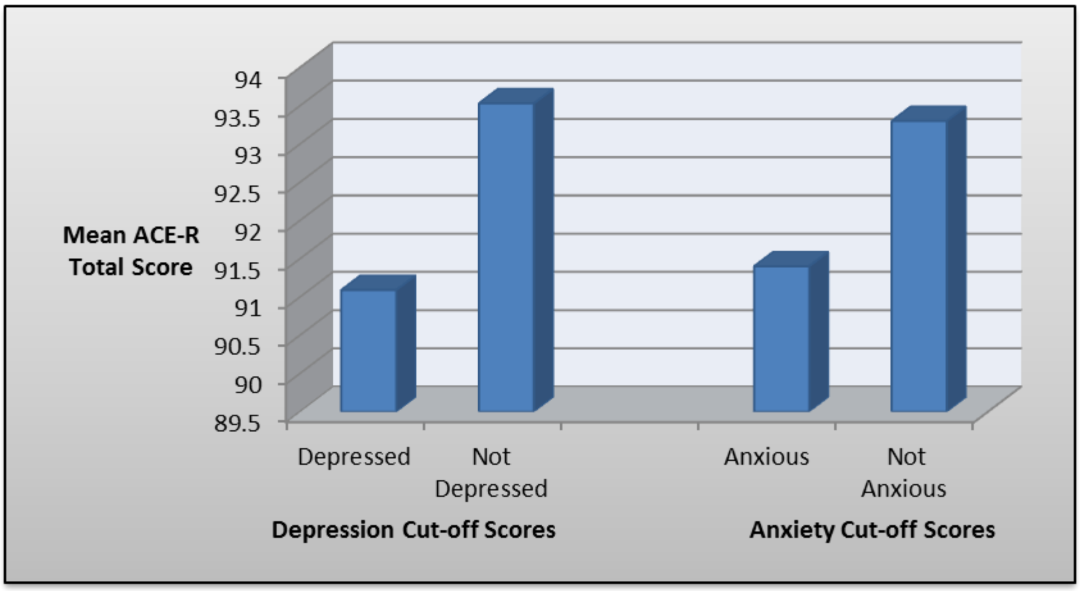

Figure 8 . Mean ACE-R total scores by depression and anxeity cut-off scores.

Social Engagement

Loneliness, social support and types of social networks have been shown to be reliably related to mental and physical health (Noone & Stephens, 2013). Similarly, ACE-R scores were related to reported levels of loneliness. Participants were asked 11 questions about their social and emotional loneliness including their experiences of general emptiness, the people they feel close to, and how often they feel rejected (de Jong Gierveld, van Groenou, Hoogendoorn, & Smit, 2009). Categorising participants into those that reported they were not lonely at all and those that reported they were either moderately, severely or very severely lonely, we see a slightly lower score for those that reported some measure of loneliness, $p<.01$ (Figure 9). ACE-R scores were also positively related to perceptions of social support (r=.149) which was measured with the Social Provisions Scale (Cutrona & Russell, 1987). There were no differences in scores across different types of social networks.

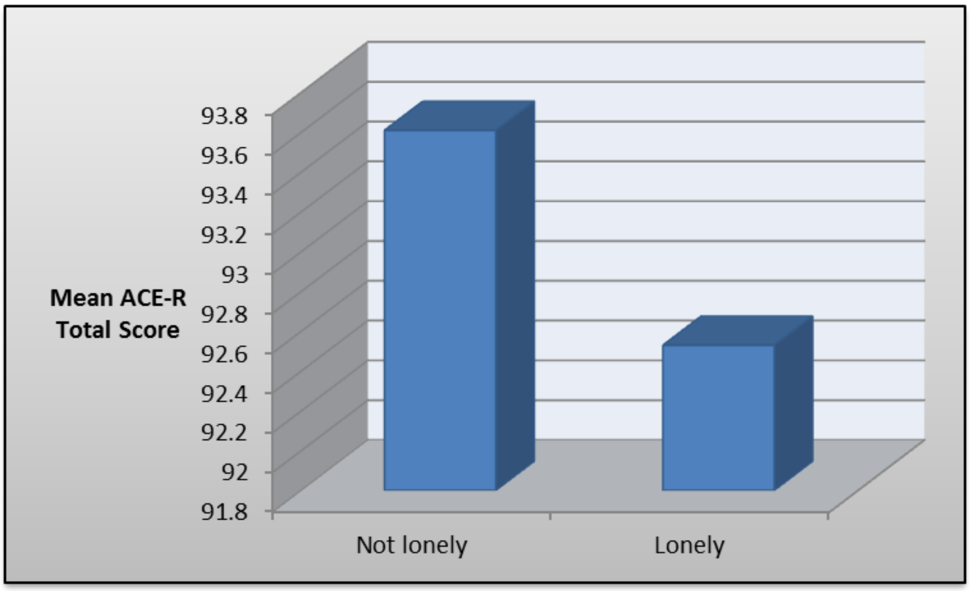

Figure 9. Mean ACE-R scores across loneliness categories.

A hierarchical regression was undertaken to assess the contribution of all demographic, health, well-being, health behaviour and social engagement (loneliness and social support) variables that were significantly related bivariately to ACE-R scores. Education (no school qualifications, secondary school qualifications, post-secondary qualifications, tertiary qualifications) and smoking status (current smoker, previous smoker, never smoked) were dummy coded. Table 4 shows five models: (1) age; (2) age + demographics; (3) age + demographics + physical and mental health; (4) age + demographics + physical and mental health + health behaviours; and (5) age + demographics + physical and mental health + health behaviours + social engagement. Age maintained a significant relationship with cognitive functioning in all models with lower function associated with older age. Demographic variables contributed the most to explained variance in ACE-R scores with a pattern of higher education related to better cognitive function. Of the health diagnoses, only diabetes (1=yes, 0=no) was related to ACE-R scores. Physical health (SF12) and anxiety levels were related in the expected direction, however depression was not significant in the final model. Looking at health behaviours, current smokers reported lower scores than previous smokers and non-smokers, however physical activity was not significant when controlling for other variables. Neither of the social engagement variables contributed to the final model. Age alone explained 7% of variance in ACE-R scores with 22% explained with all variables in the model.

Table 4.
Hierachical regression models for cognitive functioning scores on the ACE-R.

	Model 1	Model 2	Model 3	Model 4	Model 5
	B	*B*	*B*	*B*	*B*
Age	-.270***	-.217***	-.192***	-.191***	-.186***
Gender (Ref. male)		.112*	.106**	.103**	.098**
Educ1[a]		-.224***	-.186***	-.176***	-.182***
Educ2[b]		-.034	-.032	-.035	-.035
Educ3[c]		-.104**	-.106**	-.104**	-.104**
Ethnicity (Ref. Māori)		.062	.062	.060	.062
Economic Living Standards		.133**	.003	-.007	-.018
Diabetes			-.071*	-.073*	-.071*
Heart Problems			.019	.023	.020
Hypertension			.013	.005	.004
Stroke			-.055	-.054	-.053
Physical Health			.146***	.150***	.146***
Mental Health			-.010	-.012	-.028
Depression			-.082	-.079**	-.067
Anxiety			-.084*	-.090*	-.087*
Physical Activity				.003	.010
Smoke1[d]				-.080*	-.078*
Smoke2[e]				.010	.009
Loneliness					-.061
Perceived Social Support					.013
R^2	.072	.182	.219	.221	.223
F	57.32***	24.12***	14.64***	12.52***	11.45***
R^2 change		.117	.046	.005	.003
F R^2 change		17.30***	5.34***	1.69	1.64

Note. *p<.05, **p<.001, **p<.001
[a] Educ1 = no educational qualifications versus secondary school, post-secondary and tertiary qualifications (1,0)
[b] Educ2 = no educational qualifications and secondary school qualifications versus post-secondary and tertiary qualifications (1,0)
[c] Educ3 = no educational qualifications, secondary school qualifications, post-secondary qualifications versus tertiary qualifications (1,0)
[d] Smoke1 = Current smokers versus previous smokers and never smokers (1,0)
[e] Smoke2 = Current smokers and previous smokers versus never smokers (1,0)

Cognitive Functioning Over Time

Looking at participant (n=881) scores on the ACE-R in 2012 and based on the suggested ACE-R cut-off scores for cognitive impairment in the original development paper, (<82: sensitivity = 0.84, specificity = 1.0), 50 people (5.7%) would be classified as cognitively impaired. ACE-R scores in 2010 and 2012 were highly correlated, r=.71, p<.001. There was a slight drop in mean ACE-R total scores between wave 1 (m=93.40, sd=5.25) and wave 2 (m=92.32, sd=6.01) and a paired sample t-test showed this change was significant, p<.001. Just over half those who were retested had a decrease in ACE-R score between waves (482, 54.5%), while 34.5% improved. Around a quarter of those whose scores declined (112) did so by only one point (23.2%). A further 252 (52.3%) declined between 2 and 5 points, 97 (20.1%) between 6 and 10 pints, and 21 participants (4.4%) declined by over 11 points. Comparing those who declined to those who stayed the same or improved, decliners were older (mean difference 1.13 years, p<.05) (see Figure 10), had lower educational attainment (see Figure 11), were in poorer health (p<.05), and were more likely to rate their memory as poorer now (2012) than it had been at wave 1 (p<.001). None of the other wave 1 variables of interest were related to change in ACE-R scores between waves.

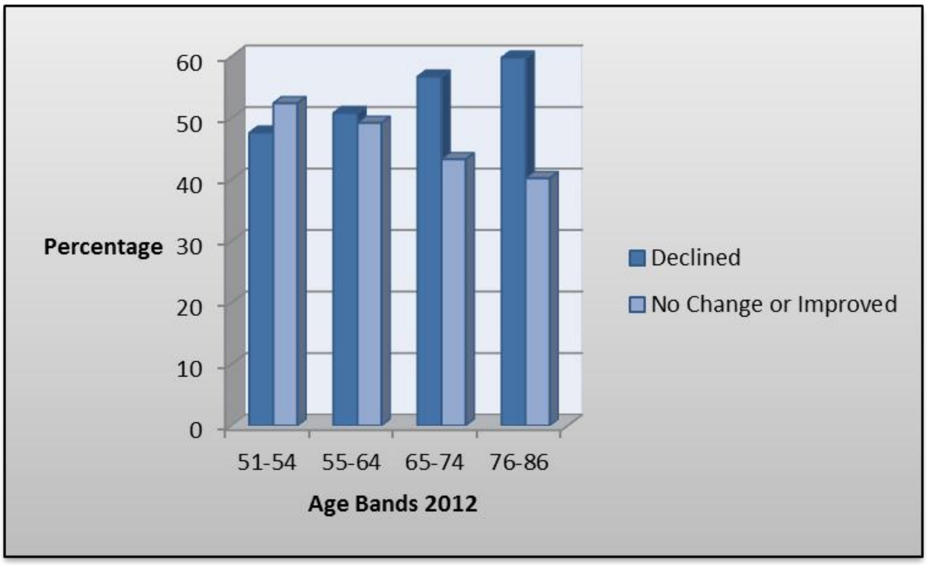

Figure 10. Change in ACE-R scores by age bands 2012.

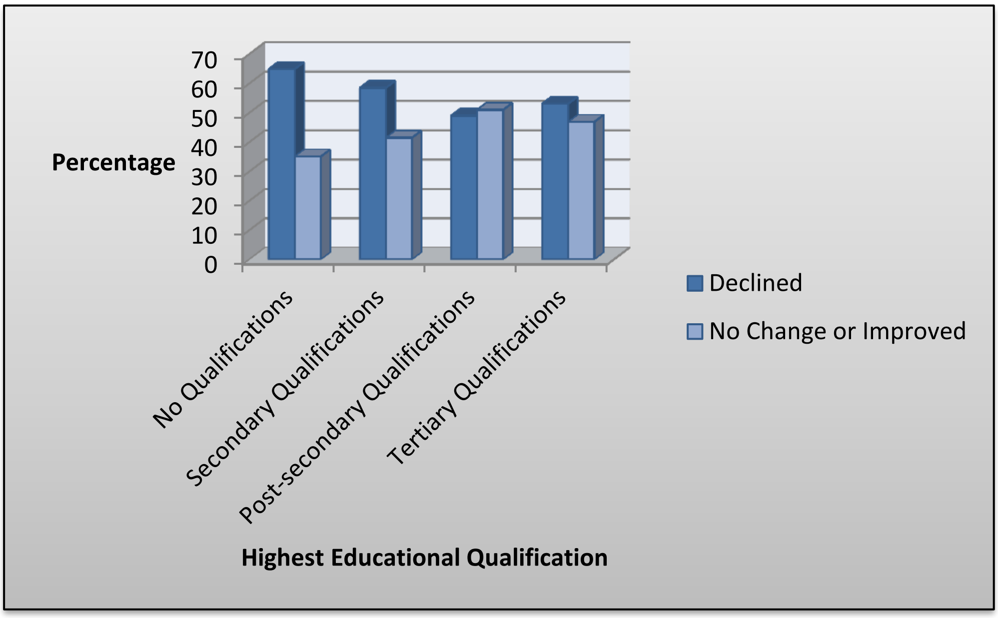

Figure 11. Change in ACE-R scores by highest educational qualification.

Summary and Concluding Comments

As noted earlier, prevalence rates for cognitive impairment vary depending on sampling and definitions of impairment. In our study using the original ACE-R cut-off scores we found an overall rate of 3.6% in wave 1 and 5.7% in wave 2. Looking at age range rates we found 2.4% of those under 55 years of age scored less than the cut-off score in 2010, with 2.0% of 55 to 64 years-olds, 4.4% of 65 to 74 year-olds and a rate of 10.5% for those 75 years and older. These rates, although representing slightly different age groupings are similar to those reported by international and New Zealand research (Campbell et al., 1983; Meltzer et al., 1997; Suthers et al.,2003).

As expected cognitive functioning decreased with age. Age continued to be significantly related to cognitive functioning even when controlling for age related variables such as health, health behaviours, wellbeing and social engagement. There was also a clear educational gradient for scores on the ACE-R with higher educational attainment related to better cognitive functioning as has been found in previous studies (Albert et al., 1995; Lee et al., 2003). The relationship between education and ACE-R scores was particularly apparent at the lowest and highest educational levels and was maintained when controlling for other environmental experiences related to cognition that may be a consequence of education (e.g. health behaviours and economic living standards). This provides support for the cognitive reserve hypothesis i.e. more highly educated individuals may still experience cognitive decline with age but they begin at a higher point above an impairment threshold.
Of the four health conditions examined that have been previously associated with cognitive decline, only a diagnosis of diabetes remained significant when controlling for other variables. Cukierman, Gerstein & Williamson (2005) in a review of cognitive decline and dementias in diabetes found that individuals with diabetes had a 1.2- to 1.5-fold greater change over time in measures of cognitive function than those without diabetes. The authors suggest cognitive decline should therefore be added to the list of chronic complications of

diabetes. Given the alarming increase in rates of diabetes in our ageing population, the impact on future rates of dementia is of concern.

Of the health behaviours, alcohol consumption and physical activity were unrelated to ACE-R scores. As moderate alcohol use has been shown to be related to improved health (Di Castelnuovo, 2006), perhaps more detailed analysis of levels of alcohol use and cognitive functioning need to be conducted. We assessed physical activity using three items asking participants to report how often they took part in sports or activities that were vigorous (e.g. running), moderately energetic (e.g. gardening) or mildly energetic (e.g. vacuuming). It is likely that a more rigorous assessment of daily physical activity would provide better insights into the link between activity and cognitive health.

Current smokers reported lower scores than non-smokers (including former smokers) on the ACE-R when controlling for other important variables. Recently reported findings from a 30 year prospective study of middle aged men in Wales highlights the importance for cognitive health of maintaining a range of healthy behaviours over time (e.g. non-smoking, an acceptable BMI, a high fruit and vegetable intake, regular physical activity, and low/moderate alcohol intake) (Elwood et al., 2013). Odds ratios for men following four or five of these healthy behaviours were 0.36 (95% CI: 0.12, 1.09; $p <0.001$) for cognitive impairment, and 0.36 (95% CI: 0.07, 1.99; $p <0.02$) for dementia. The importance of physical health for cognitive functioning is also reflected in the strong relationship between the SF12 physical health component score and ACE-R scores in multivariate analysis.

Although depression scores were related to cognitive functioning at bivariate level, this relationship was non-significant in the final multivariate model. However, anxiety maintained a significant (but small) relationship with ACE-R scores when controlling for other variables. Anxiety is associated with cognitive difficulties such as concentration, working memory and processing information (Hertzog et al., 1990) and is one of the most common neuropsychiatric symptoms associated with mild cognitive impairment (Apostolova & Cummings, 2008). The role of anxiety in the development of cognitive impairment is not clear (Beaudreau & O'Hara, 2008). Anxiety may be a response to early cognitive symptoms of impairment and its manifestation may change over time, with higher levels at the onset of MCI lessening as cognitive decline progresses (Diefenbach, Bragdon & Blank, 2013).

As noted in another NZLSA report (Noone & Stephens, 2014) “poor social connections, fewer social activities and social disengagement in people over the age of 65 have been shown to predict greater risk of cognitive decline..”. In the NZLSA sample, loneliness and perceived social support were associated with lower ACE-R scores in bivariate analyses, however these relationships became non-significant in multivariate analysis.

Decline in ACE-R scores was seen between the two waves for just over half the participants and this was more pronounced for those in older age groups, consistent with age related declines in cognitive functioning. However, a significant proportion (34.5%) improved scores between the two waves highlighting the considerable heterogeneity within older aged cohorts in the occurrence, rate and type of cognitive decline with age. Educational attainment was once again a significant factor in change in cognitive functioning over time as was physical health supporting the extensive evidence for the importance of these factors in maintaining cognitive health in later years.

References

Albert, M., Blackler, D., Moss, M., Tanzi, R., & McArdle, J. (2007). Longitudinal change in cognitive performance among individuals with mild cognitive impairment. *Neuropsychology, 21*(2), 158-169.

Albert, M., Jones, K., Savage, C., Berkman, L., Seeman, T., Blazer, D., & Rowe, J. (1995). Predictors of cognitive change in older persons: MacArthur studies of successful aging. Psychology and Aging, 10(4), 578-589.

Alexopoulos, P., Mioshi, E., Greim, B., & Kurz, A. (2007). Brief assessment of cognitive mental status in German: The Addenbrooke's Cognitive Examination (ACE) and the Addenbrooke's Cognitive Examination Revised (ACE-R). *European Journal of Geriatrics, 9*(4), 175-181.

Alzheimers New Zealand Inc. (2012). *Updated dementia economic impact report, 2011, New Zealand.* http://www.alzheimers.org.nz/information/reports-statistics.

Anstey, K., & Christensen, H. (2000). Education, activity, health, blood pressure and Apolipoprotein E as Predictors of cognitive change in old age: A review. *Gerontology, 46*(3), 163-177. doi: 10.1159/000022153

Anstey, K. J., Mack, H. A., & Cherbuin, N. (2009). Alcohol consumption as a risk factor for dementia and cognitive decline. Meta-analysis of prospective studies. The American *Journal of Geriatric Psychiatry,17*(7), 542-555.

Anstey, K. J, von Sanden, C., Salim, A., & O'Kearney, R. (2007). Smoking as a risk factor for dementia and cognitive decline: a meta-analysis of prospective studies. *American Journal of Epidemiology,166*(4), 367-378.

Apostolova, L. G., & Cummings, J. L. (2008). Neuropsychiatric manifestations in mild cognitive impairment: a systematic review of the literature. *Dementia and Geriatric Cognitive Disorders, 25*, 115–126.

Bassuk, S., Berkman, L., & Wypij, D. (1998). Depressive symptamology and incident cognitive decline in an elderly community sample. *Archives of General Psychiatry, 55*, 1073-1081.

Berkman, L. F., Glass, T., Brissette, I., & Seeman, T. E. (2000). From social integration to health: Durkheim in the new millennium. *Social Science & Medicine, 51*(6), 843-857.

Berra, C., Wancatab, J. & Ritchie, K. (2005). Prevalence of dementia in the elderly in Europe. *European Neuropsychopharmacology, 15*, 463 – 471.

Bierman, E. J. M., Comijs, H. C., Jonker, C., & Beekman, A. T. F. (2005). Effects of anxiety versus depression on cognition in later life. *American Journal of Geriatric Psychiatry, 13*(8), 686-693.

Bradford, A., Kunik, M. E., Schulz, P., Williams, S. P., & Singh, H. (2009). Missed and delayed diagnosis of dementia in primary care: Prevalence and contributing factors. *Alzheimer Disease and Associated Disorders, 23*(4): 306–314.

Brainerd, C. J., Reyna, V. F., Petersen,R. C., Smith, G. E., & Taub, E. S. (2011). Is the apolipoprotein e genotype a biomarker for mild cognitive impairment? Findings from a nationally representative study. *Neuropsychology, 25*(6), 679-689.

Beaudreau, S. A. &. O'Hara, R. (2008). Late-life anxiety and cognitive impairment: a review. *American Journal of Geriatric Psychiatry, 16*, 790–803

Boustani, M., Peterson, B., Hanson, L., Harris, R., & Lohr, K. N. (2003). Screening for dementia in primary care: A summary of the evidence for the U.S. Preventive Services Task Force. *Annals of Internal Medicine, 138*(11), 927.

Bunce, D., Batterham, P., Mackinnon, A., & Christensen, H. (2012). Depression, anxiety and cognition in community-dwelling adults aged 70 years and over. *Journal of*

Psychiatric Research, 46(12), 1662-1666. doi: http://dx.doi.org/10.1016/j.jpsychires.2012.08.023

Campbell, A., McCosh, L., & Reinken, J. (1983). Dementia in old age and the need for services. *Age and Ageing, 12*, 11-16.

Cerhan , J. H., Ivnik , R. J., Smith , G. E., Machulda , M. M., Boeve , B. F., Knopman , D. S., Petersen, R. C., & Tangalos, E. G. (2007). Alzheimer's disease patients' cognitive status and course years prior to symptom recognition. *Aging, Neuropsychology, and Cognition: A Journal on Normal and Dysfunctional Development, 14*(3), 227-235, DOI: 10.1080/13825580500320707

Cheung, G. (2007). Concurrent validity of the Geriatric Anxiety Inventory in late-life depression. *International Psychogeriatrics, 19*(02), 333-335. doi: 10.1017/S1041610206004340.

Cukierman, T., Gerstein, H. C., & Williamson, J. D. (2005). Cognitive decline and dementia in diabetes—systematic overview of prospective observational studies. *Diabetologia, 48,* 2460–2469. DOI 10.1007/s00125-005-0023-4.

Cutrona, C. E., & Russell, D. W. (1987). The provisions of social relationships and adaptation to stress. In W. H. Jones & D. Perlman (Eds.*), Advances in personal relationships.* Greenwich: JAI Press.

Davey, J. (2006). "Ageing in place": The views of older homeowners on maintenance, renovation and adaptation. Social Policy Journal of New Zealand, 27, 128-141.

Deary, I. J., Corley, J., Gow, A. J., Harris, S. E., Houlihan, L. M., Marioni, R. E., Penke, L., Rafnsson, S. B., & Starr, J. M. (2009). Age-associated cognitive decline. *British Medical Bulletin, 92,* 135–152.

de Jong Gierveld, J., van Groenou, M. B., Hoogendoorn, A. W., & Smit, J. H. (2009). Quality of marriages in later life and emotional and social loneliness. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 64*(4), 497-506.

Di Castelnuovo, A., Costanzo, S., Bagnardi, V., Benedetta Donati, M., Iacoviello, L., & de Gaetano, G. (2006). Alcohol dosing and total mortality in men and women: an updated meta-analysis of 34 prospective studies. *Archives of Internal Medicine, 166,* 2437–2445.

Diefenbach, G. J., Bragdon, L. B., & Blank, K. (2013). Geriatric Anxiety Inventory: Factor structure and associations with cognitive status. *The American Journal of Geriatric Psychiatry,* Available online http://dx.doi.org/10.1016/j.jagp.2013.04.014.

Elderkin-Thompson, V., Kumar, A., Bilkerb, W., Dunkin, J., Mintz, J., Moberg, P., Mesholam, R. I., & Gur, R. E. (2003). Neuropsychological deficits among patients with late-onset minor and major depression. *Archives of Clinical Neuropsychology, 18,* 529-549.

Elwood P, Galante J, Pickering J, Palmer S, Bayer A, et al. (2013) Healthy lifestyles reduce the incidence of chronic diseases and dementia: Evidence from the Caerphilly Cohort Study. *PLoS ONE, 8*(12): e81877. doi:10.1371/journal.pone.0081877.

Garcia-Caballero, A., Garcia-Lado, I., Gonzalez-Hermida, J., Recimil, M., Area, R., Manes, F., . . . Berrios, G. (2006). Validation of the Spanish version of the Addenbrooke's Cognitive Examination in a rural community in Spain. *International Journal of Geriatric Psychiatry, 21*, 239-245.

Hamer, M., & Chida, Y. (2009). Physical activity and risk of neurodegenerative disease: a systematic review of prospective evidence. *Psychological Medicine, 39,* 3-11.

Hawkley, L. C. & Cacioppo, J. T. (2010). Loneliness matters: A theoretical and empirical review of consequences and mechanisms. *Annals of Behavioral Medicine, 40,* 218–227. DOI 10.1007/s12160-010-9210-8.

Hertzog, C., Van Alstine, J., Usala, P., Hultsch, D., & Dixon, R. (1990). Measurement properties of the Center for Epidemiological Studies Depression Scale (CES-D) in older populations. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 2*(1), 64-72. doi: doi: 10.1037/1040-3590.2.1.64

Hurt, C. S., Burns, A., Brown, R. G., & Barrowclough, C. (2012). Why don't older adults with subjective memory complaints seek help*? International Journal of Geriatric Psychiatry, 27*, 394–400.

Kim, J., Basak, J. M., & Holtzman, D. M. (2009). The role of Apolipoprotein E in Alzheimer's disease. *Neuron, 63*, 287-303.

Kivipelto, M., Helkala, E. L., Laakso, M. P., Hänninen, T., Hallikainen, M., Alhainen, K., Soininen, H., Tuomilehto, J., & Nissien, A. (2001). Midlife vascular risk factors and Alzheimer's disease in later life: longitudinal, population based study. *British Medical Journal, 322*, 1447-51.

Konstantinopoulou, E., Kosmidis, M., Kiosseoglou, G., Karacostas, D., & Taskos, N. (2010). Adaptation of Addenbrooke's cognitive-examination revised for the greek population. *European Journal of Neurology,18*(3), 442–447.

Krueger, K. R., Wilson, R. S., Kamenetsky, J. M., Barnes, L. L., Bienias, J. L. & Bennett, D. A. (2009) Social engagement and cognitive function in old age. *Experimental Aging Research: An International Journal Devoted to the Scientific Study of the Aging Process, 35*(1), 45-60, DOI:10.1080/03610730802545028.

Kumari, M., & Marmot, M, (2005). Diabetes and cognitive function in a middle-aged cohort: findings from the Whitehall II study. *Neurology, 65*,1597-603.

Lee, S., Kawachi, I., Berkman, L. F., & Grodstein, F. (2003). Education, other socioeconomic indicators, and cognitive function. American Journal of Epidemiology,157, 712–720.

Lyketsos, C. G., Steinberg, M., Tschanz, J. T., Norton, M. C., Steffens, D. C., Breitner, J. C. (2000). Mental and behavioural disturbances in dementia: Findings from the cache county study on memory in aging. *American Journal of Psychiatry,157*, 708–714.

Luppa, M., Luck, T., Weyerer, S., König, H., Brähler, E., & Riedel-Heller, S. G. (2010). Predication of institutionalization in the elderly: A systematic review. *Age and Ageing, 39*, 31-38.

Melzer, D., Ely, M., & Brayne, C. (1997). Cognitive impairment in elderly people: population based estimate of the future in England, Scotland, and Wales. *British Medical Journal, 315*(7106), 462. doi: 10.1136/bmj.315.7106.462.

Mioshi, E., Dawson, K., Mitchell, J., Arnold, R., Hodges, J., & Turvey, C. (2006). The Addenbrooke's Cognitive Examination Revised (ACE-R): a brief cognitive test battery for dementia screening. *International Journal of Geriatric Psychiatry, 21*, 1078-1085.

Modrego, P. J., & Ferrandez, J. (2004). Depression in patients with mild cognitive impairment increases the risk of developing dementia of Alzheimer type: A prospective cohort study. *Archives of Neurology, 61*,1290–1293.

National Audit Office. (2007). Improving services and support for people with dementia. Retrieved 01/07, 2012, from http://www.nao.org.uk/publications/0607/support_for_people_with_dementia.aspx.

Noone, J. H., & Stephens, C. V. (2013). *Social support. Summary report for the New Zealand Longitudinal Study of Ageing.* Palmerston North: Massey University.

Powell, M., Smith, G., Knopman, D., Parisi, J., Boeve, B., & Petersen, R. (2006). Cognitive measures predict pathologic Alzheimer disease. *Archives of Neurology, 63*, 865-868.

Rosenberg, P. B., Mielke, M. M.,Xue, Q., & Carlson, M. C. (2010). Depressive symptoms predict incident cognitive impairment in cognitive healthy older women. *American*

Journal of Geriatric Psychiatry, 18(3), 204–211. doi:10.1097/JGP.0b013e3181c53487

Salthouse, T. A. (2009). When does age-related cognitive decline begin? *Neurobiology of Aging, 30*(4): 507–514. doi:10.1016/j.neurobiolaging.2008.09.023.

Seeman, T. E., Lusignolo, T. M., Albert, M., & Berkman, L. (2001). Social relationships, social support, and patterns of cognitive aging in healthy, high-functioning older adults: MacArthur Studies of Successful Aging. *Health Psychology, 20*(4), 243-255. DOI: 10.1037//0278-6133.20.4.243

Singh-Manoux, A., Kivimaki, M., Glymour, M. M., Elbaz, A., Berr, C., Ebmeier, K. P., Ferrie, J. E., & Dugravot, A. (2012). Timing and onset of cognitive decline: Results from Whitehall II prospective cohort study. *British Medical Journal, 344,* 2-8. doi: 10.1136/bmj.d7622.

Statistics New Zealand (2007). *New Zealand's 65+ Population: A statistical volume.* Wellington: Statistics New Zealand.

Sternberg, S., Wolfson, C., & Baumgarten, M. (2000). Undetected dementia in community-dwelling older people: the Canadian Study of Health and Aging. *Journal of the American Geriatrics Society, 58,* 1430–1434.

Suthers, K., Jung, K., & Crimmins, E. (2003). Life Expectancy with cognitive impairment in the older population of the United States. *The Journals of Gerontology: Social Sciences, 58*(3), 179-186.

Taylor, D. (2008). A guide to the 'kiwi' version of the ACE-R. powerpoint presentation Auckland psychologists for older peoples services Auckland, New Zealand.

van den Kommer, T., Comijs, H., Aartsen, M., Huisman, M., Deeg, D., & Beekman, A. (2012). Depression and cognition: How do they interrelate in old age? *American Journal of Geriatric Psychiatry,* 10.1097/JGP.1090b1013e31824878d31824873.

Wilson, R. S., Krueger, K. R., Arnold, S. E., Schneider, J. A., Kelly, J. F., Barnes, L. L., Tang, Y., & Bennett, D. A. (2007). Loneliness and risk of alzheimer disease. *Archives of General Psychiatry, 64,* 234–240.

Wilson, R. S., Hebert, L. E., Scherr, P. A., Barnes, L. L., Mendes de Leon, C. F., & Evans, D. A. (2009). Educational attainment and cognitive decline in old age. *Neurology, 72,* 460-465.

Wolinsky, F. D., Callahan, C. M., Fitzgerald, J. F., & Johnson, R. J. (1992). The risk of nursing home placement and subsequent death among older adults. *The Journal of Gerontology, 47*, 173–82.