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RESEARCH
TRANSLATION
FACULTY

CASE FOR ACTION- PROPOSAL TO NHMRC

Proposal for national project to reduce or delay cognitive decline and dementia

Authors:

Professor Henry Brodaty
Professor Kaarin J. Anstey
Emeritus Professor Maree Gleeson
Professor Colin Masters
Professor John McGrath
Professor Christopher Rowe

Submitted by the Research Translation Faculty Dementia Steering Group (May 2015)

DEMENTIA

The National Health and Medical Research Council (NHMRC) Research Translation Faculty (the Faculty) was established as a key advisory forum in 2012. The primary work of the Faculty for the 2013-15 Triennium has been to help NHMRC accelerate the translation of research by identifying the most significant gaps between research evidence and health policy and practice in each of the major health areas in the NHMRC Strategic Plan, and to propose to NHMRC possible action it could consider taking to address that gap – these are called Cases for Action. In April and May 2013, fourteen Faculty steering groups were established as NHMRC working committees to each oversee the development of a Case for Action.

The Faculty's Dementia Steering Group is comprised of a range of experts and includes primary (1˚) and secondary (2˚) representatives of NHMRC Health Care Committee (HCC), Prevention and Community Health Committee (PCHC) and Research Committee (RC). Further information is available at: www.nhmrc.gov.au/research/research-translation/research-translation-faculty/research-translation-faculty-steering-groups.

NHMRC Research Translation Faculty Dementia Steering Group - Membership

Professor Henry Brodaty (Chair)
Professor Kaarin J. Anstey
Professor Colin Masters
Professor Christopher Rowe
Professor John McGrath (RC)
Professor Debra Rickwood (HCC 1˚)
Emeritus Professor Maree Gleeson (PCHC 1˚)
Professor Helen Zorbas (HCC 2˚)
Ms Meagan Lawson (PCHC 2˚)

Research Translation Faculty Steering Group Secretariat

faculty@nhmrc.gov.au

Declaration of interests

The declarations of interests of Steering Group members, authors and contributors are available at Appendix 1.

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NHMRC Research Translation Faculty

Dementia Steering Group Case for Action

Title: Proposal for national project to reduce or delay cognitive decline and dementia

Submitted to NHMRC for consideration: May 2015

Steering Group Members:

Members	Position and affiliation
Prof. Henry Brodaty (Chair)	Scientia Professor of Ageing and Mental Health Director, Dementia Collaborative Research Centre The University of New South Wales
Prof. Kaarin J. Anstey	Director, Centre for Research on Ageing, Health and Wellbeing
Prof. Colin Masters	Senior Deputy Director, Florey Institute of Neuroscience and Mental Health Laureate Professor, The University of Melbourne
Prof. Christopher Rowe	Director, Department of Nuclear Medicine and Centre for PET, Austin Health
Prof. John McGrath (RC contact)	Director, Queensland Centre for Mental Health Learning Queensland Brain Institute, The University of Queensland
Prof. Debra Rickwood (HCC primary contact)	Professor of Psychology, The University of Canberra
Emeritus Prof. Maree Gleeson (PCHC primary contact)	Faculty of Health, The University of Newcastle
Prof. Helen Zorbas (HCC secondary contact)	Chief Executive Officer, Cancer Australia
Ms Meagan Lawson (PCHC secondary contact)	General Manager, Communications and Public Relations, Fitness Australia

HCC – Health Care Committee; PCHC – Prevention and Community Health Committee; RC – Research Committee

Authors (position and affiliation listed above):
Professor Henry Brodaty, Professor Kaarin J. Anstey, Emeritus Professor Maree Gleeson, Professor Colin Masters, Professor John McGrath and Professor Christopher Rowe.

NHMRC KT in Dementia Proposal

Proposal for national project to reduce or delay cognitive decline and dementia

NHMRC Research Translation Faculty Dementia Steering Group

Executive Summary

Evidence is accumulating that healthy lifestyle and medical care are associated with risk of Alzheimer's disease and dementia. Barnes and Yaffe (2011) calculated that 50% of the population attributable risk (PAR) of Alzheimer's could be accounted for by seven potentially modifiable risk factors and that reducing their rates by 25% would result in 3 million fewer cases of Alzheimer's in the world. Norton et al (2014) recalculated these rates, with particular focus on Europe, and came to similar conclusions except that they allowed for interactions and mutuality between risk factors and found that the seven risk factors accounted for 30% of the PAR.

In their comprehensive and rigorous review, Prince et al (2014) concluded that the evidence was robust that less education in early life, hypertension in mid-life, and depression, smoking and diabetes in late-life increased the risk of dementia.

Robust evidence is not yet available that attending to other identified risk factors such as physical and cognitive inactivity in mid- or late-life, hyperlipidaemia, obesity or depression in mid-life can delay cognitive decline and dementia.

The NHMRC Research Translation Faculty Dementia Steering Group (the Steering Group) recognises that attending to risk factors to prevent or delay cognitive decline in later life holds promise and still requires more research to determine effectiveness, and that combining with other initiatives to reduce cardiovascular risk factors is likely to lead to synergies. The Steering Group makes the following recommendations:

1. that Clinical Practice Guidelines about physical activity, diet, obesity, alcohol, health checks for hyperlipidaemia and hypertension, diabetes mellitus, smoking, social engagement, cognitive stimulation and depression include statements about possible benefits on cognition including delay in onset of dementia generally and Alzheimer's and vascular dementia in particular;
2. that education be provided to primary care practitioners, general practitioners and practice nurses, about cognitive benefits of attending to risk factors;
3. that future longitudinal cohort studies be strongly encouraged to include cognitive outcomes in their designs; and
4. that the NHMRC convene a round table with other Translation groups focussing on diabetes, obesity, smoking, hypertension and primary care and early childhood education with the aim of combining prevention strategies.

Summary

There are many potentially modifiable risk factors for cognitive decline and dementia generally and Alzheimer's disease specifically which overlap considerably with risk factors for cardiovascular disease and diabetes. There is potential for large scale population based interventions which would potentially have major benefits on delaying cognitive decline and dementia, preventing heart disease, hypertension and stroke, and reducing gait disorders, falls and sarcopenia in the older population. Such a combined intervention would need to be conducted in tandem with relevant interest groups and primary health care as well as with support of governments at federal and state levels underpinned by a successful media campaign (e.g. "Life Be In It").

Authors

Henry Brodaty

Kaarin J. Anstey

Maree Gleeson

Colin Masters

John McGrath

Christopher Rowe

Executive summary references

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Supporting submission

1. Dementia in Australia

- Dementia is an umbrella term for cognitive and functional decline interfering with day to day function. There are over 100 causes of dementia with Alzheimer's disease accounting for over 50% of cases. Other common causes are vascular dementia, Lewy body dementia and fronto-temporal dementia. While about 10% of dementias have their onset before 65 years, mostly people develop dementia in later life when mixed causes are more common.

- AIHW (2012) estimated that over 330,000 Australians have dementia now and projections are that this will exceed 900,000 by 2050 (1).

- Access Economics (2003) estimated that the cost to Australia of dementia exceeds $6b (2). The Productivity Commission (2013) estimated that aged care costs 0.8% of GDP now and would reach 1.8% by 2050 (3); Access Economics estimated that dementia costs would exceed 3% of GDP by 2050(2).

- Unless there is a cure or a way of delaying onset of dementia, and assuming the projected tripling of the dementia prevalence, the current 180,000 residential care beds will need to triple by 2050. This means that Australia will require an extra 360,000 beds by 2050 or more than 750 new beds every month for 40 years of which 500 beds will be for dementia. In parallel with this, strategies will be needed to address the extensive shortfalls in the aged care workforce both in community and residential care.

- The most recent Intergenerational Report from the Australian Government (4) highlights the rationale for greater investment in dementia-related research and services. Apart from the aging population and the associated increased burden on the health sector, we need to encourage mature age participation to optimize future economic growth prospects. Dementia has a disproportionate impact on Australian's economic growth, living standards and government finances.

- Despite 30 years of drug trials, none of the major causes of dementia has a cure or even a drug treatment to modify the course of the disease. While many trials are underway, it is doubtful that even if a successful drug for Alzheimer's disease were to be developed that this would be available within a decade and even then it would not be helpful for the non-Alzheimer's dementias and may have little benefit for established cases.

2. Prevention and delaying onset of dementia

As dementia is largely a disease of late life, delaying the onset by only a few years can have major impact on the prevalence and ultimately reduce incidence. For instance, a 2 year delay would result in a 20% reduction in prevalence i.e. 60,000 fewer cases today (5, 6) and a 5 year delay would provide a 50% reduction in prevalence i.e. 150,000 fewer cases today (5, 7). There is emerging evidence of reduced prevalence or incidence of dementia or cognitive decline in UK, Sweden and Denmark which has been attributed to improvement and early life education, diet and lifestyle (8-10).

3. Review of risk factors

3.1 Physical exercise

There is consistent evidence that physical exercise is associated with reduced dementia risk, with higher levels of activity associated with the lowest risk (11, 12). The effect of physical activity on brain ageing and neurodegeneration is also corroborated by neuroimaging studies and intervention studies (13, 14).

Many of the risk factors for cognitive decline and dementia are relevant to the provision of an exercise program. Foremost among these risk factors is the low level of physical activity and physical fitness in the middle aged and older Australian population. Only ≈ 30% of Australians aged 55-75 report participation in optimal levels of physical activity (150 min/week of moderate intensity aerobic activities) (15).

In those with chronic conditions related to cognitive decline such as type 2 diabetes and obesity, the proportion drops to 10% or less. The situation is even worse for resistance training, with data from the 2011 US Behavioral Risk Factor Surveillance Survey showing that only 24% of middle-aged and older adults engaged in

resistance training ≥2x/week, less if older age, obese or low educational levels. The very individuals who are at highest risk of diseases related to physical inactivity (including dementia), are the ones most likely to be insufficiently active. Many of risk factors for dementia are the same as the risk factors for low physical activity highlighting the need for this lifestyle modification.

3.2 Diet

A dietary pattern that includes high fish consumption, high fruit and vegetable intake and low levels of red meat consumption is associated with reduced dementia risk (16). The dietary component with the strongest link to dementia risk reduction is fish, with three or more servings a week being associated with lower risk (12, 17, 18).

Reviews have questioned the evidence of a relationship between the micro and macronutrients (vitamin B6, vitamin B12, folate, vitamin C, vitamin E, flavonoids, omega-3, Mediterranean diet) and cognitive function (19, 20). While some studies have shown positive results, particularly those using cross-sectional designs, the findings have not been consistently supported in prospective cohort studies, and preventive interventions have generally failed the critical test of randomised controlled trials.

3.3 Obesity

Obesity and being overweight during midlife has been consistently associated with increased late-life dementia risk (21). The relationship between late-life obesity and dementia risk is not clear (21-23) and if anything, it appears that weight loss, regardless of initial weight, is associated with dementia risk (24).

The evidence is more compelling of the link between obesity and vascular pathology and AD. Obesity-associated biological mechanisms that increase Alzheimer's pathology are hyperinsulinaemia, advanced glycosylation end products (AGEs), and production of cytokines and adipokines. Prince et al (2014) concluded that evidence to confirm an association between midlife adiposity and incident dementia is inadequate, possibly confounded by the decline in body mass that accompanies dementia and may precede clinical onset by up to a decade (19). Other confounders are the associations between obesity and vascular risk factors such as diabetes and hypertension, which if controlled for, reduce the association between obesity and

dementia. Central obesity in midlife (waist circumference) may be better measure than total obesity (BMI).

Dietary interventions should be tailored to the cognitive risk factor profile of individuals, based on current evidence-based practice. Diabetes/diabetes risk, hypertension/pre-hypertension, hyperlipidaemia, and overweight/obesity *all* require dietary education, counselling and support to ensure sustained behavioural change around the specific targeted goals of: lowered intake of saturated fat, higher protein, lower glycaemic index (GI), higher whole grains and salt restriction.

3.4 Alcohol

The evidence for alcohol as a risk or protective factor is mixed and weak (20). Studies have shown a relationship between low levels of alcohol intake (rather than abstinence) and reduced risk of dementia and cognitive decline (25, 26). A confound has been whether abstainers are life-long abstainers or recent abstainers who would be more likely to have ceased drinking alcohol because of health conditions which may themselves be risk factors for dementia (i.e. reverse causality).

Males drinking more than 3 standard drinks per day and females drinking two or more should receive advice about effects of excess alcohol on the brain and recommend they cut down or seek assistance if they find this difficult.

3.5 Health checks

Persons aged over 40 years should seek an annual check-up with their GP including blood pressure readings, weight, height and waist circumference for all and blood tests including lipid profiles for many.

3.6 Hypertension

The link between abnormally high or low blood pressure in late life and dementia risk is inconsistent (27), however, high blood pressure in midlife may represent a risk for dementia in later life (28). It appears that the trajectory of declining blood pressure from mid to late adulthood may be related to risk of dementia (29) and that low blood pressure in late life may increase the risk of dementia(30). Hence messages about

the link between blood pressure and dementia need to be carefully communicated and evidence-based.

The Australian Heart Foundation (31) defines normal to high blood pressure (which US defines as prehypertension) as systolic of 120-139mm Hg or diastolic of 80-89 mm Hg (in upper arm). Persons with prehypertension should be advised to complete relevant lifestyle modules and have repeat blood pressure recordings and those with high blood pressure will be *additionally* advised to seek medical treatment.

3.7 Hyperlipidaemia

High level of serum cholesterol during midlife is associated with elevated dementia risk (32), however, this relationship is not consistently evident for high cholesterol in late life (33).

The National Vascular Disease Prevention Alliance (34). has defined borderline-high risk hypercholesterolaemia as 5.2 – 6.2mol/L and very high risk as >6.2 mmol/L. Those with borderline hypercholesterolaemia could be encouraged to follow dietary and exercise programs and then be re-evaluated. Those at very high risk could be *additionally* be recommended to seek treatment from their GPs. These decisions will need to be considered in context of other cardiovascular risk factors and triglyceride levels (i.e. those with TG levels > 5.18 mmol/L).

3.8 Diabetes

Type 2 diabetes is associated with cognitive dysfunction and structural brain abnormalities, including atrophy. In a recent systematic review of 86 studies (35), hyperglycaemia, was modestly negatively associated with cognitive function in people with type 2 diabetes without dementia. Although robust animal data suggest that exercise improves cognition and brain structure and metabolism, such trials are rare in humans with type 2 diabetes. A systematic review of weight loss interventions reported significant small effects on memory and executive function in obese adults (36) but these trials did not target diabetes specifically. The Look AHEAD trial of lifestyle modification in type 2 diabetes (37) included a cross-sectional ancillary study on cognitive function (N = 978; 53-84 years) 8 years after enrolment. There was no overall difference in cognition between the intensive lifestyle intervention or control groups at that time point, although lack of longitudinal

data limit interpretation of these results. However, there was evidence for better processing speed and composite score among overweight (but not obese) participants.

Recommendations for diabetes prevention include aerobic and resistance exercise (38) and nutritional recommendations (similar to obesity module) advice such as restriction of saturated fat and high GI carbohydrates, a higher protein intake (~25% energy) with emphasis on increased intake fruits, vegetables and wholegrains.

3.9 Smoking

Smoking in late-life is a well-known risk factor for developing dementia (39). Furthermore, smoking cessation is associated with less late-life cognitive decline and brain atrophy than continued smoking (40). While former smokers have not been shown to be at increased risk of dementia in meta-analyses (41), there is a lack of long term follow-up data on this group, and it is unknown whether the findings is due to adults who smoked briefly in their youth, or those who smoked heavily into adulthood. Hence caution is required in making statements about former smokers and dementia risk and it is still advisable for multiple health reasons for no adult to smoke. Additionally, smoking is associated with significantly lower levels of fitness as well as higher levels of obesity, hypertension, hyperlipidaemia, diabetes risk and depression, and dementia itself. Smokers can receive advice about undertaking an online course, e.g. Quit Now (42), given the number of the Quitline (131 848), seek GP-based assistance and can be provided an exercise program, which increases the likelihood of success in some trials (43). This most recent Cochrane review stresses the need for large robust trials in this domain to define the efficacy of this approach. Currently available programs are recommended but there is little evidence for effectiveness and an enhanced module with strong behavioural theory/internet content and exercise prescription is hypothesised to be superior to current approaches.

3.10 Social engagement

There is consistent evidence that higher levels of social engagement are associated with reduced risk of dementia (44-46). Social engagement measures include different types of relationships, living arrangements, size and quantity of social

networks and amount of social activities. Social engagement has been linked to lower rates of dementia through plausible biological mechanisms (47), although the mechanism is not well understood and the association may result from reverse causality even with follow-up periods over several years.

3.11 Cognitive Stimulation/training

On a world-wide basis, low education is the single risk factor contributing *greatest potentially modifiable* disease burden (6, 28). Engaging in cognitively stimulating activities in late life (e.g., reading, playing puzzles and attending museums and concerts) is associated with a lower risk of dementia (48, 49). Late life cognitive activity may delay the impact of neurodegeneration in a similar manner to the role of education level on dementia risk and onset. Nevertheless, the mechanisms and effective dosage are not known, and to date, and while there is no reliable evidence for an effect of cognitive training programs on delaying dementia there are many trials demonstrating improvement in cognition in healthy older persons and in those with Mild Cognitive Impairment.

A meta-analysis of 22 cohort studies found that high-level cognitive activity, at any stage of the lifespan, was associated with a 46% reduction in risk for incident all-cause dementia compared to low (OR=0.54, 95%CI 0.49-0.59) (50). Cognitive lifestyles are “set-up” in childhood and adolescent through education (19, 50), which has a profound effect on dementia risk in later life (51, 52). In later life, engagement in cognitively demanding leisure activities are linked to lower dementia incidence, independent of either education or occupational complexity (50), and in cohort studies that have excluded ascertainment bias cannot be easily explained by reverse causality (53).

3.12 Depression

Depression and depressive symptoms, either in midlife or latelife, are consistently associated with elevated risk of cognitive decline and dementia (54, 55). In late-life, late-onset depression may represent a prodrome of Alzheimer’s dementia itself (54). Treatment of depression – there is now good evidence that internet based therapy (NNT = 2) is more powerful than drug treatment (NNT = 10) for non-psychotic unipolar depression (56).

4. Summary

There are many potentially modifiable risk factors for cognitive decline and dementia generally and Alzheimer's disease specifically which overlap considerably with risk factors for cardiovascular disease and diabetes. There is potential for large scale population based interventions which would potentially have major benefits on delaying cognitive decline and dementia, preventing heart disease, hypertension and stroke, and reducing gait disorders, falls and sarcopenia in the older population. Such a combined intervention would need to be conducted in tandem with relevant interest groups and primary care as well as with support of governments at federal and state levels underpinned by a successful media campaign (e.g. "Life Be In It"). Further support for this was the publication in March 2015 in the Lancet of the first RCT demonstrating that attention to risk factors could reduce the cognitive decline in 60-77 year old Finns at risk for Alzheimer's disease (57).

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Dementia Case for Action - Declarations of Interests

The declarations of interests of Steering Group members, authors and contributors to this Case for Action are listed below.

Name and Role(s)	Interests declared
Prof Henry Brodaty • Steering Group Chair • Author	**Relationships** • Alzheimer's Australia, Alzheimer's NSW, Alzheimer's Australia Dementia Research Foundation, International Psychogeriatric Association, Alzheimer's Association (USA) International Conference Organising Committee. **Employment** • UNSW employee, Dementia Collaborative Research Centre, and South East Sydney Local Health District. **Board Membership** • Multiple pharmaceutical companies in past. Current (in last two years): Nutricia, Merck, Lilly. **Consultancy fees/honorarium** • Multiple pharmaceutical companies. Current (in last two years): Nutricia, Merck, Lilly. **Grants** • NHMRC and Department of Health and Ageing. Intends to apply for NHMRC grants in future • Industry sponsored grants o Current: Tau Therapeutics o Previous: Eisai, Janssen, Lilly, Lundbeck, Medivation, Merck, Novartis, Parke-Davis, Pfizer, Roche, Sanofi, Servier, Voyager, Wyeth. **Support for travel or accommodation** • Pharmaceutical Company Board meetings • Conference presentations by conference organisers – Alzheimer's Disease International, Alzheimer's Association International Conference (AAIC). **Speeches/lectures** • Multiple speeches/lectures delivered in relation to Alzheimer's disease and dementia. **Other** (eg: unpaid advisory roles) • Aged Care Reform Implementation Council, Minister's Dementia Advisory Group.
Prof Kaarin J. Anstey • Steering Group member • Author	**Other** • Authored report on research funding for Dementia that analysed NHMRC grant and funding data. Anstey, K.J., Kiely, K.M., (2012). Evaluation of NHMRC Data on the Funding of Dementia Research in Australia. A Report for Alzheimer's Australia Paper 26. **Relationships** • Board Member, Alzheimer's Australia Dementia Research Foundation. **Grants** • Receives NHMRC Grant support and will be applying for NHMRC grant support in future. **Consultancy fees/honorarium** • Receives funding as invited speaker eg. 2015 AAIC conference • Invited speaker on Diabetes and cognitive decline. Honorarium and expenses paid by Eli Lilly, 2013.
Prof Colin Masters • Steering Group member • Author	**Ownership interests** • Has shares and options in Prana Biotechnology which is developing drugs for dementia. **Consultancy fees/honorarium** • Received as member of the Global Advisory Committee for Eli Lilly which is developing drugs for Alzheimer's disease • Consultant to Eli Lilly, Prana Biotechnology and Actinogen.

Name and Role(s)	Interests declared
Prof Colin Masters *…continued*	**Grants** • Receives NHMRC Grant support and will be applying for NHMRC grant support in future. **Speeches/Lectures** • Eli Lilly, Prana Biotechnology.
Prof Christopher Rowe • Steering Group member • Author	**Relationships** • Alzheimer's Association (USA) Imaging subgroup Chair. **Consultancy fees/honorarium** • On an intermittent basis, provides advice to companies on the development of imaging agents for dementia and on the planning and conduct of therapeutic trials for dementia. Has also, and is likely to continue to be, asked to give lectures at national and international meetings on imaging in dementia. For these activities a standard rate of reimbursement may be received. • Within the last 12 months, has received payments for the following: o Stockholm-Springfield Symposium on Alzheimer's Disease - invited speaker standard reimbursement from the conference organiser. o Alzheimer's Association International Conference (AAIC) 2012- speaker fee received for satellite meeting sponsored by GE Healthcare o European Association of Nuclear Medicine (EANM) 2012 - invited speaker; standard reimbursement received from the conference organiser o Human Amyloid Imaging (HAI) 2013- airfare from Navidea (see below) o The International Conference on Alzheimer's and Parkinson's Disease AD/PD 2013- invited speaker standard reimbursement from the conference organiser. **Grants** • Funds to support both commercially sponsored and Investigator initiated studies from companies are paid to Austin Health. Research funding is currently received from Bayer/Piramal, Avid Radiopharmaceuticals/Lilly, and GE Healthcare • Grants from academic sources are paid to University of Melbourne, Austin Health or Austin Medical Research Foundation. Current sources include, NHMRC, Science Industry Endowment Fund, Co-operative Research Centre for Mental Health and the Dementia Collaborative Research Centres. **Support for travel** • Reimbursement for a Premium economy airfare to the 2013 HAI meeting in Miami was received from Navidea. Data were presented from a study relevant to the development of an imaging agent for Alzheimer's Disease that is owned by Navidea and also met to discuss the Phase II and III clinical trials for this product. **Meals/beverages** • Usually meets with research collaborators to discuss projects over a meal (breakfast, lunch or dinner) at international meetings if we mutually attend. If the collaborator is from a commercial company, they usually pay for the meal. Meal cost would range from $25-$100 and comply with US regulations. **Speeches/lectures** • Numerous national and international lectures on imaging in dementia. **Other** • Taskforce member (international) for appropriate use of imaging in dementia.
Prof John McGrath • Steering Group member • Research Committee contact • Author	**Employment** • Director, Queensland Centre for Mental Health Learning - employee of Queensland Health Department. **Board membership** • Board Member, Research Australia. Research Australia advocates on behalf of Health and Medical Research. It is an unpaid position.

Name and Role(s)	Interests declared
Prof John McGrath *…continued*	**Grants** • Grants and Fellowship from NHMRC and ARC (2010 onwards). Summary: o National Health and Medical Research Council. T Burne, A Mackay-Sim, J McGrath. Social behaviour in rats developmentally deficient in Vitamin D: Modelling the negative symptoms of schizophrenia. 2008- 2010 o Australian Research Council Discovery Grant. E Whitelaw, S Chong, JJ McGrath. The role of epigenetics in the early gestational programming of adult phenotype by ethanol. 2008-2010 o National Health and Medical Research Council. J McGrath, D Eyles, T Burne, E Whitelaw. Advanced paternal age: behavioural, neuroanatomical and genomic correlates in the offspring of older fathers. 2009-2011 o Australian Government Department of Health and Ageing. V Morgan, A Jablensky, Anna Waterreus, Robert Bush, John McGrath, Carol Harvey, Pat McGorry, David Castle, Martin Cohen, Helen Stain, Cherrie Galletly, Andrew MacKinnon. National survey of high impact psychosis (SHIP) – Phase 1-3. 2009-2011 o National Health and Medical Research Council. J McGrath, D Eyles, D St Clair, PB Mortensen, Neonatal vitamin D status and risk of schizophrenia: a replication in two independent samples. 2011-2013 o National Health and Medical Research Council. T Burne, A Mackay-Sim, D Eyles, J McGrath. Attentional processing in developmentally vitamin D deficient rats: Modelling the cognitive symptoms of schizophrenia. 2011- 2013 o National Health and Medical Research Council. D Eyles, T Burne, J McGrath. Developmental vitamin D deficiency and prefrontal cortical dysfunction. 2012 -2014 o National Health and Medical Research Council. D Eyles, J McGrath, T Burne. Early pharmacological intervention in an animal model of schizophrenia. 2013 -2015 o National Health and Medical Research Council. N Martin, J McGrath, M Wright. Exploring modifiable risk factors for mental illness in young adults: infection, vitamin D and stress. 2013 -2015 o National Health and Medical Research Council. J Scott, J McGrath, J Najman, R Alati, A Mamun, A Clavario. The outcomes of adolescents and young adults who experience hallucinations: A birth cohort study. 2013- 2015 o National Health and Medical Research Council. John Cade Fellowship. Modifiable risk factors for Serious Mental Illness - an integrated program 2013- 2017 o National Health and Medical Research Council J. McGrath, D Eyles. Is developmental vitamin D deficiency associated with autism-related phenotypes: a birth cohort study. 2014- 2017 o National Health and Medical Research Council D. Eyles, T. Burne (AI. McGrath). The developmental vitamin D deficiency animal model of schizophrenia: Critical window for intervention and optimal dose. 2014 - 2016. o National Health and Medical Research Council T. Burne (AI J McGrath, D Eyles). Low adult vitamin D levels and cognitive dysfunction. 2014- 2016. **Relationships** • University appointments (unpaid) - Conjoint Professor at the Queensland Brain Institute, the University of Queensland, and an Adjunct Professor at Griffith University.
Prof Debra Rickwood • Steering Group member • Health Care Committee (HCC) primary contact	**Employment** • Professor of Psychology, Faculty of Health, University of Canberra • Chief Scientific Advisor, headspace National Youth Mental Health Foundation Inc. **Activities** • Member, Scientific Leadership Council for the Young and Well Cooperative Research Centre • Member, Australian Institute of Criminology Research Ethics Committee.

Name and Role(s)	Interests declared
Prof Debra Rickwood *…continued*	**Board membership** • Director, Australian Psychological Society • Director, Richmond Fellowship ACT • Editorial Board, Advances in Mental Health. **Relationships** • Fellow, Australian Psychological Society • Member, Australian Psychological Society, College of Community Psychologists • Member, Australian Institute of Company Directors. **Grants** • Australian Rotary Health Research Project Grant • NHMRC Project Grant • NHMRC Partnership Grant • Cooperative Research Centre for Young People, Technology and Wellbeing Grant • beyondblue National Priority Driven Research Program Grant. **Consultancy fees – honorarium** • Mental Illness Fellowship Victoria.
Prof Helen Zorbas • Steering Group member • HCC secondary contact	• Nil interests to declare
Emer Prof Maree Gleeson • Steering Group member • Prevention and Community Health Committee (PCHC) primary Contact • Author	**Board membership** • Non Executive Director, Central Coast Local Health District – Chair, Research Committee • Non Executive Director Hunter Water Corporation – Interest-organisation is affected by that Australian Water Quality Guidelines • Non Executive Director Hunter Valley Research Foundation - Organisation undertakes economic assessments of medical research and health surveys • Non Executive Director Nationwide Superannuation Fund – no potential conflicts identified.
Ms Meagan Lawson • Steering Group member • PCHC secondary contact	• Nil interests to declare.