Special Article

Primary Prevention of Dementia: Focus on Modifiable Risk Factors

Patsri Srisuwan MD*

** Outpatient Department, Phramongkutklao Hospital, Bangkok, Thailand*

Background: *Dementia will inevitably increase in the aging world. Moreover, there is no cure for dementia. Therefore, primary prevention is very important. There are several factors possibly and/or certainly influencing dementia risk including non-modifiable and modifiable risk factors. There are evidences that the risk of developing dementia may be reduced by modifiable risk factors.*

Lifestyle factors: *The strategies are to encourage regular physical and mental exercise in midlife and in late-life. Those include cognitive activity and higher education, mentally demanding occupations or participation in mentally challenging leisure activities, being more socially active, a diet that is low in saturated fat, a diet with lots of fruits and vegetables, smoking cessation, and prevention of head injury, with loss of consciousness*

Chronic disease factors: *The strategies are to prevent high blood pressure, especially at midlife, diabetes, high serum cholesterol, especially at midlife, and depression or high depressive symptoms.*

Conclusion: *It is important to develop a systematic public-health strategy and research specific to primary prevention of dementia in Thailand with the evidence-based medicine.*

Keywords: *Primary prevention, Dementia*

J Med Assoc Thai 2013; 96 (2): 251-8
Full text. e-Journal: *http://jmat.mat.or.th*

There are currently 18 million people with dementia in Europe, Africa, Asia, and Latin America, and nearly 29 million demented subjects are predicted by 2050[1]. Dementia is one of the most common diseases in the elderly, with crude prevalence rates between 5.9% to 9.4% for subjects aged over 65 in the European Union[2]. In Thailand, the prevalence rate is about 1.8 to 3.3%[3,4]. Dementia drastically affects daily life and personal activities and has numerous clinical complications. Thus, it is not surprising that the cost of care for patients with dementia is more than twice the average cost of care for other chronic diseases[5]. Moreover, there is no systematic public-health strategy for primary prevention of dementia in Thailand.

There are several factors possibly and/or certainly influencing dementia risk such as aging, gender, genetic influence, early-life deleterious conditions, Inborn physical attributes, Illiteracy/lack of early education, environmental stress, vascular risk, lifestyle, and traumas/accidents[6]. Prevention appears to be particularly prominent for antidementia strategies. Those are negatively seen due to the lack of cure of dementia. However, they can be constructively approached because they can be carried out within a multidimensional scheme with the highest chances of success, if adopted in the early adulthood. Primary prevention is directed against dementia prior to its biological onset or against dementia's risk factors. A great deal of attention is being dedicated to the identification and modulation of factors that have a large potential to be modified before the onset. Therefore, the author's focus on modifiable risk factors associated with dementia risk include physical, cognitive, and social activities, diet, smoking, alcohol consumption, head injury, diseases, drugs, supplements, and vitamins. Those are for the primary prevention of dementia. Moreover, the author focuses on prevention in healthy people, excluding mild cognitive impairment.

Correspondence to:
Srisuwan P, Outpatient Department, Phramongkutklao Hospital, Bangkok 10400, Thailand.
Phone: 0-2354-7600-28 ext. 93100
E-mail: patsri2004@yahoo.com

Physical activity

Regular physical exercise is associated with better cognitive function and reduced risk of cognitive decline and dementia. Many studies show that exercise undertaken in midlife and in late-life reduce the risk of dementia. Even simple exercise like walking has been shown beneficial. In a prospective study of 2,509 aged 70 years or older, cognitive performance

measured by a modified Mini-Mental State Examination (MMSE) at baseline, 3, 5, and 8 years was shown to remain substantially stable at follow-up in those subjects performing weekly moderate or vigorous exercise (odd ratio (OR), 1.31; 95% confidence interval (CI), 1.06-1.62)[7]. Moreover, Cassilhas et al conducted randomize controlled trial (RCT) on 62 healthy elderly of average age 69 years. It showed that a 24-weeks program of Moderate- and high-intensity resistance exercise programs provided an improvement in cognition[8]. In a prospective two years study of 18,766 healthy women aged 70 to 81 years, it revealed that higher levels of activity, including walking, were associated with better cognitive performance[9]. In addition, walking is associated with a reduced risk of dementia from a prospective six years study of 2,257 healthy men aged 71 to 93 years. It showed that men who walked the least (<0.25 mile/day) experienced a 1.8-fold excess risk of dementia compared with those who walked more than 2 miles/day (17.8 vs. 10.3/1,000 person-years; relative hazard [RH], 1.77; 95% CI, 1.04-3.01)[10]. More recently, RCT with 120 elder adults showed that aerobic exercise training by fast walking 40 minutes three times per week for one year increased the size of the anterior hippocampus, caudate nucleus, and BDNF (a mediator of neurogenesis in dentate gyrus) comparing with the control group (stretching exercise)[11]. Even dancing was found to be associated with a lower risk of dementia in a prospective cohort of 469 subjects, older than 75 years, following a median follow-up of 5.1 years[12].

Cognitive activity

Higher participation in mentally stimulating activities is associated with better cognitive function and reduced risk of cognitive decline and dementia. Those with a history of higher education, mentally demanding occupations, or participation in mentally challenging leisure activities are consistently found to have a lower risk of developing dementia. More recently, the systematic review from the Cochrane Library analyzed from 24 RCTs of 2,339 healthy elderly average 69.9 years old showed that training program of cognitive domain improved mental capability immediately and delayed verbal recall significantly[13]. In addition, systematic review from seven RCTs found that cognitive training intervention such as computer-based training and paper and pencil training on memory in healthy elderly produced strong and persistent protective effects on longitudinal neuropsychological performance for more than two years[14]. Any type of training such as memory, reasoning, or speed of processing could improve cognition. Moreover, reasoning training resulted in less functional-decline in self-reported instrumental activities of daily living (IADLs). Furthermore, cognitive training improved cognitive abilities specific to the abilities trained for five years after the initiation of the intervention[15]. Level of education can prevent cognitive decline. In a prospective study, cognitive performance measured by MMSE at baseline, 3, 5, and 8 years was shown to remain substantially stable at follow-up in those subjects who had high school education level or greater (OR, 2.75; 95% CI, 1.78-4.26) and ninth grade literacy level or greater (OR, 4.85; 95% CI, 3.00-7.87)[7]. Furthermore, a seven-years prospective study of 1,433 healthy elderly aged 65 years or older, measuring cognitive performance and diagnosis of dementia, showed that the crystallized intelligence reduced incident of dementia and cognitive decline (population attributable fraction, 18.11%; 95% CI, 10.91%-25.42%)[16]. Frequent cognitive activity in old age has been associated with reduced risk of Alzheimer disease (AD) in a prospective study of 700 healthy elderly. The present study showed that more frequent participation in cognitive activity was associated with reduced incidence of AD (hazard ratio (HR), 0.58; 95% CI, 0.44-0.77). On the other hand, a cognitively inactive person was 2.6 times more likely to develop AD than a cognitively active person[17]. Daily mental activities such as work complexity have been associated with a decreased risk of dementia (relative risk (RR), 0.85; 95% CI, 0.75-0.95) in the Kungsholmen Study[18].

Social activity

Being more socially active is associated with reduced risk of cognitive decline and dementia. This has been shown in many studies measuring social engagement in different ways. Combining social activity with mental and/or physical activity may provide even greater benefit in reducing the risk of developing dementia. The risk of dementia was decreased in subjects with high leisure activities (RR, 0.62; 95% CI, 0.46-0.83)[19]. Moreover, Karp et al conducted a three years prospective study. It showed that the risk of dementia was decreased in subjects with higher mental, physical, and social activity by 0.71 (95% CI, 0.49-1.03), 0.61 (95% CI, 0.42-0.87) and 0.68 (95% CI, 0.47-0.99), respectively. In addition, the most beneficial effect was present for subjects with high scores in all or in two of the components of

activities (RR, 0.53; 95% CI, 0.36-0.78)[20]. Wilson et al concluded that the risk of AD was more than doubled in lonely persons compared with persons who were not lonely from a four years prospective study of 823 healthy elderly[21]. Moreover, larger social networks have a protective influence on cognitive function among elderly women from a five years prospective study. Incidence of dementia in women with larger social networks was 0.74 (95% CI, 0.57-0.97), compared with women with smaller social networks[22].

Diet

Diet, low in saturated fat and high in fruits and vegetables have been associated with better cognitive function and reduced risk of dementia. Morris et al found that high vegetable but not fruit consumption may be associated with a slower rate of cognitive decline with older age. The overall mean change in cognitive performance score per year was a decline of 0.04 standardized units in the elderly. In the elderly who had an average of 2.8 servings of vegetable per day, the decline was slower by 0.019 standardized units per year ($p = 0.01$). Moreover, in elderly who had an average of 4.1 vegetable servings per day was slower by 0.018 standardized units per year ($p = 0.02$), but it was not associated with the fruit consumption[23]. On the other hand, Ritchie et al concluded that fruit and vegetable consumption of more than two servings per day can prevent dementia in a population by 6.46%, 95% CI, 0.15%-13.06%)[16]. However, the Doetinchem Cohort Study concluded that total intake of fruits and vegetables was not or was inconsistently associated with cognitive function and cognitive decline. This is because higher reported vegetable intake was associated with lower information processing speed ($p = 0.02$) and worse cognitive flexibility ($p = 0.03$) at baseline. However, with a smaller decline in information processing speed ($p<0.01$) and global cognitive function ($p = 0.02$) at follow-up of 10 years, in middle-aged men and women[24]. For fish and meat consumption, a cross-sectional study in low- and middle-income countries revealed that fish consumption reduced risk of dementia (Poisson regression prevalence ratios (PR), 0.81; 95% CI, 0.72-0.91) in all sites except India. On the other hand, meat consumption increased the risk (PR, 1.19; 95% CI, 1.07-1.31)[25]. Luchsinger et al concluded that higher intake of calories and fats may be associated with higher risk of AD. Subjects who had a high caloric intake (1,870 Kcal per day) increase a risk of AD comparing with low caloric intake (758 Kcal per day) (HR, 1.5; 95% CI, 1.0-2.2)[26]. For Mediterranean diet, higher adherence to the diet was associated with lower risk for AD (OR, 0.76; 95% CI, 0.67-0.87; $p<0.001$)[27].

Smoking

Smoking is associated with an increased risk of dementia. Peter et al concluded from systematic review of 28 studies that the risks of Alzheimer disease, vascular dementia (VaD), dementia, and cognitive decline were 1.59, 1.35, 1.16, and 1.2 time respectively in the current smoking elderly comparing with non-smoking[28]. Moreover, in an eight years prospective study, it was found that not smoking reduced the risk of dementia (OR,1.84; 95% CI, 1.14-2.97)[7]. In addition, heavy smoking in midlife had an increased risk of dementia, AD, and VaD for more than two decades after the prospective study. Furthermore, compared with nonsmokers, those smoking more than two packs a day had an elevated risk of dementia (HR, 2.14; 95% CI, 1.65-2.78), AD (HR, 2.57; 95% CI, 1.63-4.03), and VaD (HR, 2.72; 95% CI, 1.20-6.18) respectively[29].

Alcohol consumption

Consumption of moderate amounts of alcohol has been associated with better cognitive function and reduced risk of dementia. However, excessive amounts over time can increase the risk. In a prospective study, it was found that binge drinking (i.e., alcohol exceeding the amount of 5 bottles of beer or a bottle of wine on 1 occasion at least monthly) in midlife was associated with an increased risk of dementia (RR, 3.2; 95% CI, 1.2-8.6)[30]. On the other hand, in a meta-analyses of 15 prospective studies, light to moderate drinkers RRs of AD, VaD, and dementia compared with nondrinkers were 0.72 (95% CI, 0.61-0.86), 0.75 (95% CI, 0.57-0.98), and 0.74 (95% CI, 0.61-0.91), respectively[31]. Moreover, in systematic review of 23 studies, small amounts of alcohol may be protective against dementia (random effects model, RR, 0.63; 95% CI, 0.53-0.75) and AD (RR, 0.57; 95% CI, 0.44-0.74) but not for vascular dementia (RR, 0.82; 95% CI, 0.50-1.35) or cognitive decline (RR, 0.89; 95% CI, 0.67-1.17)[32].

Head injury

Head injury, with loss of consciousness, is associated with an increased risk of subsequent cognitive decline and dementia. In a population-based

prospective historical cohort design of men who were World War II Navy and Marine veterans found that moderate (HR, 2.32; 95% CI, 1.04-5.17) and severe head injuries (HR, 4.51; 95% CI, 1.77-11.47) in young men may be associated with increased risk of AD and other dementias in late life. However, mild head injuries were inconclusive[33]. In addition, a systemic review of 15 case-control studies revealed that head injury is a significant risk factor for AD (OR, 1.58; 95% CI, 1.21-2.06). However, one from 15 case-control study concluded that risk of head injury in those with AD only found in males (males: OR, 2.29; 95% CI, 1.47-2.06 and females: OR, 0.91; 95% CI, 0.56-1.47)[34].

Diseases

1. Hypertension

High blood pressure, especially at midlife, is consistently associated with an increased risk of developing dementia. Treatment of hypertension has been found to reduce the risk of cognitive decline and dementia. However, the Cochrane Library of four RCTs found that type of antihypertensive medications in the elderly could not prevent cognitive decline. However, most studies ended before completion such as the three trials that reported adverse effects requiring discontinuation of treatment[35]. However, The Honolulu Asia Aging Study on Japanese American revealed in hypertensive men, the duration of the antihypertensive treatment is associated with a reduced risk for dementia and cognitive decline. For each additional year of treatment, there was a reduction in the risk of incident dementia (HR, 0.94; 95% CI, 0.89-0.99)[36]. In addition, an eight years prospective study in hypertensive elderly showed Antihypertensive drug use was associated with 8% (95% CI, 15% to -1%) risk reduction of dementia per year of use for persons 75 years old or younger, but no significant reduction in persons older than 76 years old (95% CI, -11% to 4%)[37].

2. Diabetes

Diabetes is associated with an increased risk of developing dementia. A systematic review of 15 studies revealed that diabetes was associated with a faster decline in cognitive function (RR, 1.47; 95% CI, 1.25-1.73), AD (RR, 1.39; 95% CI, 1.16-1.66) and VaD (RR, 2.38; 95% CI, 1.79-3.18)[38]. In addition, a seven-year prospective study revealed diabetes increased the risk of dementia in the elderly (4.88%; 95%, 1.87%-7.98%)[16]. Moreover, in a 27-year retrospective cohort study among older patients with type 2 diabetes, a history of severe hypoglycemic episodes was associated with a greater risk of dementia. Compared with patients with no hypoglycemia, patients with single or multiple episodes had a graded increase in risk with fully adjusted HRs, for one episode (HR, 1.26; 95% CI, 1.10-1.49), two episodes (HR, 1.80; 95% CI, 1.37-2.36), and three or more episodes (HR, 1.94; 95% CI, 1.42-2.64)[39].

3. Dyslipidemia

High serum cholesterol, especially at midlife, is associated with an increased risk of developing dementia. A meta-analysis of 18 studies found that high midlife total cholesterol increased the risk of AD and any dementia. On the other hand, no association between high late-life total cholesterol and AD or any dementia. In addition, there was no association between high midlife or late-life total cholesterol and VaD[40].

4. Overweight, obesity, and metabolic syndrome

Obesity and high serum cholesterol, especially at midlife, is associated with an increased risk of developing dementia. A meta-analysis of four studies concluded that first, high adiposity increased risk of AD. Second, waist circumference in old age, a measure of central adiposity, may be a better predictor of dementia than body mass index (BMI). Third, lower BMI predicts dementia in elderly people. Finally, weight loss may precede dementia diagnosis by decades[41]. In addition, a case-control study in the elderly confirmed that both overweight and obesity at midlife, independently increase the risk of dementia, AD, and VaD. Compared with normal BMI (20-25), overweight and obesity at midlife were related to dementia with odds ORs (95% CIs) of 1.71 (1.30-2.25) and 3.88 (2.12-7.11), respectively[42]. Moreover, the four years Three City prospective study in the elderly found that metabolic syndrome as a whole and several of its components such as hypertriglyceridemia and low HDL-C had a negative impact on global cognitive from MMSE (HR, 1.22; 95% CI, 1.08-1.37) and specific cognitive function from Benton Visual Retention Test (HR, 1.13, 95% CI, 1.01-1.26)[43].

5. Depression

People with depression or high depressive symptoms have, on average, a higher risk of developing dementia. Analysis from 11 case-control studies showed that history of depression was found to be associated with AD, significantly in episodes of

depression more than 10 years before AD onset. No association was found with anti-depressant treatment and the three major life events considered in the analysis, death of spouse, death of a child, and divorce[44]. In addition, a cross-sectional, family-based, case-control study found that there was a significant association between depression symptoms and AD (OR, 2.13; 95% CI, 1.71-2.67). When the depression symptoms first occur within one year before the onset of AD, the association was higher (OR, 4.57; 95% CI, 2.87-7.31). However, when the depression symptoms first occur more than one year before the onset of AD, the association was lower (OR, 1.38; 95% CI, 1.03-1.85). In families where depression symptoms first occurred more than 25 years before the onset of AD, there was still a modest association (OR, 1.71; 95% CI, 1.03-2.82)[45].

Limitations of studies

There are some limitations in the studies. First, because the delay from initial brain changes to clinical dementia is known to be long, it is possible that an effect of intervention, positive or negative, will take many more years to manifest. Second, the definitions of dementia diagnoses are not standard such as some researches were obtained from medical record. Third, there might be some subjects with undiagnosed dementia in the cohort; some cases might have been missed in participants who had died. Therefore, some selective survival effect cannot be ruled out. Fourth, some lifestyles are highly unlikely making it not possible to conduct an RCT. Therefore, it may never be feasible to answer with absolute certainty. Finally, we cannot exclude the possibility that other unmeasured factors may be influencing the association. Therefore, future research, large longitudinal studies involving several occasions of measurement from midlife to late adulthood are essential. Additionally, meta-analysis studies or systemic reviews also need to answer the questions.

Conclusion

Dementia will inevitably increase in an aging world. There is no cure for dementia. However, there is evidence that the risk of developing dementia may be reduced by modifiable risk factors. It is important to develop systematic public-health strategy and research specific for primary prevention of dementia in Thailand with evidence-based medicine.

Lifestyle factors, regular physical exercise in midlife and in late-life is associated with better cognitive function and reduced risk of cognitive decline and dementia. Cognitive activity is associated with better cognitive function and reduced risk of cognitive decline and dementia. Those with a history of higher education, mentally demanding occupations, or participation in mentally challenging leisure activities are consistently found to have a lower risk of developing dementia. Being more socially active is associated with reduced risk of cognitive decline and dementia. This has been shown in many studies measuring social engagement in different ways. Combining social activity with mental and/or physical activity may provide an even greater benefit in reducing the risk of developing dementia. Diet, low in saturated fat and high in fruits and vegetables have been associated with better cognitive function and reduced risk of dementia. Smoking is associated with an increased risk of dementia. Head injury, with loss of consciousness is associated with an increased risk of subsequent cognitive decline and dementia.

Chronic disease factors such as high blood pressure, especially at midlife, is consistently associated with an increased risk of developing dementia. Treatment of hypertension has been found to reduce the risk of cognitive decline and dementia. Diabetes is associated with an increased risk of developing dementia. High serum cholesterol, especially at midlife, is associated with an increased risk of developing dementia. People with depression or high depressive symptoms have, on average, a higher risk of developing dementia.

Acknowledgement

The author wishes to thank Dr. Sirintorn Chansirikarn, the staffs of Health Intervention and Technology Assessment Program and Outpatient Department, Phramongkutklao Hospital for their assistance.

Potential conflicts of interest

Funding for the review was provided by the Health Intervention and Technology Assessment Program.

References

1. Essink-Bot ML, Pereira J, Packer C, Schwarzinger M, Burstrom K. Cross-national comparability of burden of disease estimates: the European Disability Weights Project. Bull World Health Organ 2002; 80: 644-52.
2. Berr C, Wancata J, Ritchie K. Prevalence of

dementia in the elderly in Europe. Eur Neuropsychopharmacol 2005; 15: 463-71.
3. Phanthumchinda K, Jitapunkul S, Sithi-amorn C, Bunnag SC, Ebrahim S. Prevalence of dementia in an urban slum population in Thiland: validity of screening methods. Int J Geriatr Psychiatr 1991; 6: 639-46.
4. Jitapunkul S, Kunanusont C, Poolcharoen W, Suriyawongpaisal P. Health problems of Thai elderly (A National Survey). Bangkok: National Health Foundation and Ministry of Public Health; 1999.
5. Martin BC, Ricci JF, Kotzan JA, Lang K, Menzin J. The net cost of Alzheimer disease and related dementia: a population-based study of Georgia Medicaid recipients. Alzheimer Dis Assoc Disord 2000; 14: 151-9.
6. Polidori MC, Nelles G, Pientka L. Prevention of dementia: focus on lifestyle. Int J Alzheimers Dis 2010. doi: 10.4061/2010/393579.
7. Yaffe K, Fiocco AJ, Lindquist K, Vittinghoff E, Simonsick EM, Newman AB, et al. Predictors of maintaining cognitive function in older adults: the Health ABC study. Neurology 2009; 72: 2029-35.
8. Cassilhas RC, Viana VA, Grassmann V, Santos RT, Santos RF, Tufik S, et al. The impact of resistance exercise on the cognitive function of the elderly. Med Sci Sports Exerc 2007; 39: 1401-7.
9. Weuve J, Kang JH, Manson JE, Breteler MM, Ware JH, Grodstein F. Physical activity, including walking, and cognitive function in older women. JAMA 2004; 292: 1454-61.
10. Abbott RD, White LR, Ross GW, Masaki KH, Curb JD, Petrovitch H. Walking and dementia in physically capable elderly men. JAMA 2004; 292: 1447-53.
11. Erickson KI, Voss MW, Prakash RS, Basak C, Szabo A, Chaddock L, et al. Exercise training increases size of hippocampus and improves memory. Proc Natl Acad Sci U S A 2011; 108: 3017-22.
12. Verghese J, Lipton RB, Katz MJ, Hall CB, Derby CA, Kuslansky G, et al. Leisure activities and the risk of dementia in the elderly. N Engl J Med 2003; 348: 2508-16.
13. Martin M, Clare L, Altgassen AM, Cameron MH, Zehnder F. Cognition-based interventions for healthy older people and people with mild cognitive impairment. Cochrane Database Syst Rev 2011; (1): CD006220.
14. Valenzuela M, Sachdev P. Can cognitive exercise prevent the onset of dementia? Systematic review of randomized clinical trials with longitudinal follow-up. Am J Geriatr Psychiatry 2009; 17: 179-87.
15. Willis SL, Tennstedt SL, Marsiske M, Ball K, Elias J, Koepke KM, et al. Long-term effects of cognitive training on everyday functional outcomes in older adults. JAMA 2006; 296: 2805-14.
16. Ritchie K, Carriere I, Ritchie CW, Berr C, Artero S, Ancelin ML. Designing prevention programmes to reduce incidence of dementia: prospective cohort study of modifiable risk factors. BMJ 2010; 341: c3885.
17. Wilson RS, Scherr PA, Schneider JA, Tang Y, Bennett DA. Relation of cognitive activity to risk of developing Alzheimer disease. Neurology 2007; 69: 1911-20.
18. Karp A, Andel R, Parker MG, Wang HX, Winblad B, Fratiglioni L. Mentally stimulating activities at work during midlife and dementia risk after age 75: follow-up study from the Kungsholmen Project. Am J Geriatr Psychiatry 2009; 17: 227-36.
19. Scarmeas N, Levy G, Tang MX, Manly J, Stern Y. Influence of leisure activity on the incidence of Alzheimer's disease. Neurology 2001; 57: 2236-42.
20. Karp A, Paillard-Borg S, Wang HX, Silverstein M, Winblad B, Fratiglioni L. Mental, physical and social components in leisure activities equally contribute to decrease dementia risk. Dement Geriatr Cogn Disord 2006; 21: 65-73.
21. Wilson RS, Krueger KR, Arnold SE, Schneider JA, Kelly JF, Barnes LL, et al. Loneliness and risk of Alzheimer disease. Arch Gen Psychiatry 2007; 64: 234-40.
22. Crooks VC, Lubben J, Petitti DB, Little D, Chiu V. Social network, cognitive function, and dementia incidence among elderly women. Am J Public Health 2008; 98: 1221-7.
23. Morris MC, Evans DA, Tangney CC, Bienias JL, Wilson RS. Associations of vegetable and fruit consumption with age-related cognitive change. Neurology 2006; 67: 1370-6.
24. Nooyens AC, Bueno-de-Mesquita HB, van Boxtel MP, van Gelder BM, Verhagen H, Verschuren WM. Fruit and vegetable intake and cognitive decline in middle-aged men and women: the Doetinchem Cohort Study. Br J Nutr 2011; 106: 752-61.
25. Albanese E, Dangour AD, Uauy R, Acosta D, Guerra M, Guerra SS, et al. Dietary fish and

meat intake and dementia in Latin America, China, and India: a 10/66 Dementia Research Group population-based study. Am J Clin Nutr 2009; 90: 392-400.
26. Luchsinger JA, Tang MX, Shea S, Mayeux R. Caloric intake and the risk of Alzheimer disease. Arch Neurol 2002; 59: 1258-63.
27. Scarmeas N, Stern Y, Mayeux R, Luchsinger JA. Mediterranean diet, Alzheimer disease, and vascular mediation. Arch Neurol 2006; 63: 1709-17.
28. Peters R, Poulter R, Warner J, Beckett N, Burch L, Bulpitt C. Smoking, dementia and cognitive decline in the elderly, a systematic review. BMC Geriatr 2008; 8: 36.
29. Rusanen M, Kivipelto M, Quesenberry CP Jr, Zhou J, Whitmer RA. Heavy smoking in midlife and long-term risk of Alzheimer disease and vascular dementia. Arch Intern Med 2011; 171: 333-9.
30. Järvenpää T, Rinne JO, Koskenvuo M, Räihä I, Kaprio J. Binge drinking in midlife and dementia risk. Epidemiology 2005; 16: 766-71.
31. Anstey KJ, Mack HA, Cherbuin N. Alcohol consumption as a risk factor for dementia and cognitive decline: meta-analysis of prospective studies. Am J Geriatr Psychiatry 2009; 17: 542-55.
32. Peters R, Peters J, Warner J, Beckett N, Bulpitt C. Alcohol, dementia and cognitive decline in the elderly: a systematic review. Age Ageing 2008; 37: 505-12.
33. Plassman BL, Havlik RJ, Steffens DC, Helms MJ, Newman TN, Drosdick D, et al. Documented head injury in early adulthood and risk of Alzheimer's disease and other dementias. Neurology 2000; 55: 1158-66.
34. Fleminger S, Oliver DL, Lovestone S, Rabe-Hesketh S, Giora A. Head injury as a risk factor for Alzheimer's disease: the evidence 10 years on; a partial replication. J Neurol Neurosurg Psychiatry 2003; 74: 857-62.
35. McGuinness B, Todd S, Passmore P, Bullock R. Blood pressure lowering in patients without prior cerebrovascular disease for prevention of cognitive impairment and dementia. Cochrane Database Syst Rev 2009; (4): CD004034.
36. Peila R, White LR, Masaki K, Petrovitch H, Launer LJ. Reducing the risk of dementia: efficacy of long-term treatment of hypertension. Stroke 2006; 37: 1165-70.
37. Haag MD, Hofman A, Koudstaal PJ, Breteler MM, Stricker BH. Duration of antihypertensive drug use and risk of dementia: A prospective cohort study. Neurology 2009; 72: 1727-34.
38. Lu FP, Lin KP, Kuo HK. Diabetes and the risk of multi-system aging phenotypes: a systematic review and meta-analysis. PLoS One 2009; 4: e4144.
39. Whitmer RA, Karter AJ, Yaffe K, Quesenberry CP Jr, Selby JV. Hypoglycemic episodes and risk of dementia in older patients with type 2 diabetes mellitus. JAMA 2009; 301: 1565-72.
40. Anstey KJ, Lipnicki DM, Low LF. Cholesterol as a risk factor for dementia and cognitive decline: a systematic review of prospective studies with meta-analysis. Am J Geriatr Psychiatry 2008; 16: 343-54.
41. Luchsinger JA, Gustafson DR. Adiposity and Alzheimer's disease. Curr Opin Clin Nutr Metab Care 2009; 12: 15-21.
42. Xu WL, Atti AR, Gatz M, Pedersen NL, Johansson B, Fratiglioni L. Midlife overweight and obesity increase late-life dementia risk: a population-based twin study. Neurology 2011; 76: 1568-74.
43. Raffaitin C, Feart C, Le Goff M, Amieva H, Helmer C, Akbaraly TN, et al. Metabolic syndrome and cognitive decline in French elders: the Three-City Study. Neurology 2011; 76: 518-25.
44. Jorm AF, van Duijn CM, Chandra V, Fratiglioni L, Graves AB, Heyman A, et al. Psychiatric history and related exposures as risk factors for Alzheimer's disease: a collaborative re-analysis of case-control studies. EURODEM Risk Factors Research Group. Int J Epidemiol 1991; 20 (Suppl 2): S43-7.
45. Green RC, Cupples LA, Kurz A, Auerbach S, Go R, Sadovnick D, et al. Depression as a risk factor for Alzheimer disease: the MIRAGE Study. Arch Neurol 2003; 60: 753-9.

การป้องกันระดับปฐมภูมิในปัจจัยที่ปรับเปลี่ยนได้ของโรคสมองเสื่อม

พัฒน์ศรี ศรีสุวรรณ

ภูมิหลัง: *โรคสมองเสื่อมมีแนวโน้มมากขึ้นเรื่อย ๆเนื่องจากผู้สูงอายุมีจำนวนมากขึ้น ร่วมกับปัจจุบันยังไม่มีทางรักษาโรคสมองเสื่อมให้หายขาด ดังนั้นการป้องกันระดับปฐมภูมิจึงมีความสำคัญอย่างมาก ซึ่งปัจจัยเสี่ยงต่อการเกิดโรคสมองเสื่อมมีทั้งปัจจัยที่ไม่สามารถปรับเปลี่ยนได้ และปัจจัยที่สามารถปรับเปลี่ยนได้ แต่บทนิพนธ์นี้จะนำเสนอในปัจจัยที่สามารถปรับเปลี่ยนได้*

ปัจจัยด้านวิถีการดำรงชีวิต: *รณรงค์ให้มีการออกกำลังกายให้สม่ำเสมอทั้งในวัยกลางคน และวัยสูงอายุ กระตุ้นการใช้ความคิดและส่งเสริมให้มีการศึกษาระดับสูง รวมถึงการมีอาชีพที่ต้องใช้ความคิดหรือมีกิจกรรมยามว่างที่ใช้ความคิดสามารถลดความเสี่ยงต่อโรคสมองเสื่อมได้ ส่งเสริมให้มีการเข้าสังคม รับประทานอาหารที่ดีต่อสุขภาพ คือ ไขมันต่ำ มีผักและผลไม้มาก หยุดสูบบุหรี่ ป้องกันการกระแทกบริเวณศีรษะ โดยเฉพาะการกระแทกรุนแรงจนหมดสติ*

ปัจจัยด้านโรคเรื้อรังต่าง ๆ: *ป้องกันและควบคุมความดันโลหิตสูง ไขมันสูงโดยเฉพาะในวัยกลางคน และวัยสูงอายุ โรคเบาหวานและโรคซึมเศร้าหรืออาการซึมเศร้า*

สรุป: *ควรมีการรณรงค์โดยอาศัยหลักฐานทางวิชาการในการป้องกันระดับปฐมภูมิของโรคสมองเสื่อม และส่งเสริมงานวิจัยในประเทศไทยต่อไป*