

Thematic Article

Lifecycle nutrition and cardiovascular health: the aged

Kazuo Kondo¹ MD, PhD, Widjaja Lukito² MD, PhD and Gayle S Savige³ PhD, GradDipDietetics

¹*Institute of Environmental Science for Human Life, Ochanomizu University, Otsuka, Bunkyo-ku, Tokyo, Japan*

²*SEAMEO TROPMED, Regional Center for Community Nutrition, University of Indonesia, Jakarta, Indonesia*

³*FAO Centre of Excellence, Faculty of Medicine, Nursing and Health Sciences, Monash University, Melbourne, Australia*

As the world's population ages, cardiovascular health becomes increasingly important. The ageing process gradually leads to a decline in the structure and function of the cardiovascular system. Other factors associated with ageing can hasten this decline, for instance, lifestyles that have become more sedentary. Additionally, the prevalence of hypertension, dyslipidaemia and diabetes, major risk factors for cardiovascular disease increase with age. Nutrition throughout the lifecycle can help prevent the development of these conditions and appropriate food habits instigated later in life can improve the management of these conditions and their impact on cardiovascular health.

Key words: cardiovascular, coronary heart disease, lifestyle, nutrition, oxidation, physical activity, tea.

Introduction

The proportion of the world's population over the age of 60 is growing in both developing and developed countries (Table 1). This can be attributed, at least in part, to increases in life expectancy and decreases in birth rates. The proportion of the 'oldest old' (those over 80 years) is also increasing rapidly. Cardiovascular disease (CVD), a major cause of death and disability among the aged, can be delayed with healthy lifestyles, especially physical activity and nutrition.¹ For these reasons promoting cardiovascular health in the elderly remains important and effective.

The duration and exposure to CVD risk factors over time and/or certain periods in an individual's lifecycle may also influence cardiovascular health.² For example, healthy ageing begins during gestation, as fetal undernutrition appears to be a risk factor for the subsequent development of cardiovascular disease.³ In a prospective study of nearly 30 000 US men aged 40–75 years, body mass index (BMI), waist-to-hip ratio and weight gain since 21 years old were associated with an increased risk of coronary heart disease (CHD).⁴

Factors influencing cardiovascular health

Cardiovascular structure and function

There are a number of age-related changes that occur to the cardiovascular system. They include increases in cardiac mass, left ventricular (LV) wall thickness and collagen deposition. Parts of the valvular structures thicken and increase in circumference, the arterial wall contains more collagen and there is an increase in intimal thickness. These structural changes result in functional changes, such as a decrease in heart rate at rest, a decrease in maximal heart rate during exercise, a decrease in LV compliance and a decrease in vascular compliance.⁵ While these structural and functional changes are attributed to the ageing process, they are further accentuated with disease and disuse, factors that are potentially modifiable with appropriate lifestyle measures.

Social networks

Ageing adults are often more vulnerable to losing social networks as their children leave home, they retire from the work place and they become less physically active. These changes can adversely impact on health as social networks appear to play a fundamental role in health and well-being.^{6,7} Japanese men living in California have higher rates of heart disease compared to Japanese men living in Japan. These differences can be attributed to some extent on the differences in diet and other lifestyle factors that exist between the two populations. However, differences in rates of heart disease still occur within the population of Japanese men living in California. Differences in these rates of heart disease appear to relate, in part, to differences in social networks and not just diet and lifestyle. Higher rates of heart disease were found among those men who had given up or lost the tight, social network of their traditional culture after migrating.⁸

Frailty

Functional decline or frailty is a condition in which a person's reserve capacity in a number of physiological systems has deteriorated to the point that the risk of disability and death is increased with minor external stressors such as infections.⁹ Ageing adults can ward off many aspects of functional decline with regular physical activity, especially strength training¹⁰ and by ensuring nutritional status is not compromised.¹¹

Correspondence address: Dr Gayle S Savige, FAO Centre of Excellence, C/-Department of Epidemiology and Preventive Medicine, Faculty of Medicine, Nursing and Health Sciences, Monash Medical School, Alfred Hospital, Prahran, Victoria 3181, Australia.

Tel: +61 3 99030891; Fax: +61 3 9903 0584

Email: gayle.savige@med.monash.edu.au

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Table 1. Projected proportion of the population aged 65 years and over, 1990–2025

Region	Year	Percentage of the population		
		≥ 65 years	≥ 75 years	≥ 80 years
Oceania	1990	9.3	3.6	1.8
	2010	11.0	4.8	2.8
	2025	15.0	6.6	3.6
Asia*	1990	4.8	1.5	0.6
	2010	6.8	2.5	1.2
	2025	10.0	3.6	1.8

*Data excludes countries of the former USSR. Table 1 adapted from World Technical Report Series 853.

Immune function

Immune function declines with age and this appears to be associated with increased inflammatory activity in blood.¹² In a cross-sectional study, the plasma concentrations of tumour necrosis factor- α (TNF- α), a pro-inflammatory cytokine, was higher in a cohort of elderly adults compared with a group of young, healthy controls.¹² Furthermore, within the group of elderly subjects, those with the highest concentrations of TNF- α were more likely to have had a clinical diagnosis of atherosclerosis compared with those who had low to intermediate plasma concentrations of TNF- α , even after controlling for confounding factors such as severe health disorders and medications. In addition, TNF- α was also weakly associated with triglycerides, low levels of high-density lipoprotein/total cholesterol ratios, C-reactive protein and leucocyte counts, which are all risk factors of atherogenesis and thromboembolic complications. Studies examining the effect of enhanced nutrition and regular physical activity in the aged, have shown these lifestyle factors can impact favourably on immune function.^{13,14}

Oxidation

Of current interest in the aetiology of heart disease is the accumulation of oxidation products in the body, and the role of anti-oxidants in minimising this oxidative damage. As it is modified low-density lipoprotein (LDL) and not unmodified LDL, that is atherogenic, interest has focused on anti-oxidants which inhibit LDL oxidation. There are many anti-oxidants in addition to vitamin E, vitamin C and carotenoids such as polyphenols. Tea, red wine and many vegetables are rich in polyphenols. These polyphenols and the polyphenols found in red wine may inhibit LDL oxidation. Diets enriched in polyphenols, including the red colour components of certain foods, such as anthocyanidin, may inhibit LDL oxidation.

A study examining the association between LDL oxidation and the consumption of red wine was carried out in 10 male volunteers. The volunteers were asked to drink vodka for 14 days and then red wine for 14 days. All subjects received a standard diet. Oxidation of LDL was measured by the lag time method. A 10% longer lag time was observed after consumption of red wine, but there was no difference in lag time after the consumption of vodka. This and other studies of ours suggest that red wine intake rather than alcohol per se inhibits LDL oxidation and may reduce atherosclerosis.¹⁵

Perhaps dietary components, including Japanese green tea, also rich in polyphenols, especially catechins, may protect

against LDL oxidation. To test this hypothesis, a group of volunteers were asked to consume green tea. The LDL oxidation lag time was measured before consuming the tea and at 1, 2, 4 and 6 h intervals after consuming the tea. Lag time was significantly prolonged at 1 and 2 h after consuming green tea. Serum polyphenol (epigallocatechin gallate and epicatechin gallate) concentrations also significantly increased during this time, suggesting that catechins in green tea are absorbed in the intestine and enter the blood stream and inhibit LDL oxidation.¹⁶

Only a few epidemiological studies exist that address these issues. One example, the Zutphen elderly study, found that when flavonoid intake was more than 19 mg/day, CHD risk decreased by one-third compared to those with a lower flavonoid consumption.¹⁷ It is also known that the consumption of black tea, onions and apples contributed to the outcome in this study.

Risk factors influencing CVD

Hypertension

Hypertension is more prevalent in older adults and is a major risk factor in the development of CVD.¹⁸ This so-called age-related rise in blood pressure is due, at least in part, to the accumulating effects of certain lifestyle factors.¹⁹ Dietary interventions have been successful in lowering blood pressure,^{20,21} as have interventions involving exercise training.^{22,23}

Lipids

In most western populations, adverse changes occur in lipid profiles with advancing years.²⁴ Dyslipidaemia has been shown to be an effective predictor of CHD not only in the middle-aged, but also in the elderly.²⁴ The rate of CHD mortality is generally lower among the Japanese compared with many other industrialised nations with ageing populations.²⁵ Environmental factors appear likely to explain some of this difference, such as the differences in the intake of saturated fat and its impact on serum cholesterol concentrations.²⁶ In a longitudinal study (conducted in Japan), spanning a 28-year period between 1958 and 1986, total serum cholesterol concentrations were found to increase as the subjects aged, but the magnitude of these increases were similar in all generations during the same time period. Furthermore, at any given age, total cholesterol concentrations were lower in subjects born in the previous decade.²⁷ The investigators suggest changes in dietary patterns, especially in relation to the intake of fat, have probably contributed to this upward trend in total serum cholesterol concentrations.

Other lifestyle factors may help to negate the unfavourable changes that occur in relation to lipid concentrations with age. One study examining the difference in lipid concentrations between older, habitually active women (distance runners) and their sedentary controls found two important differences. First, the elevation in total cholesterol associated with age was smaller in the runners compared with the sedentary women. Second, the concentration of cholesterol subfractions, LDL and high-density lipoprotein (HDL), contributing to this age-related rise in total cholesterol differed between the two groups. Sedentary women had a higher LDL cholesterol concentration, whereas the physically active women had a higher concentration of the *protective* HDL cholesterol.²⁸

Diabetes

The prevalence of type II diabetes rises sharply with age and is a major risk factor in CVD.²⁹ Lifestyle interventions involving diet and physical activity conducted in more than 110 000 men and women recruited from health care clinics in China have shown such interventions are associated with a significant reduction in the risk of developing diabetes.³⁰

Smoking

Smoking is associated with both CHD and stroke, but this habit usually declines in ageing adults.²⁴ However, the prevalence of smoking differs among populations. In Japan for instance, the rate of smoking is high (although it is now declining).²⁵ Despite the high rates of smoking among Japanese, the incidence of CHD remains relatively low.²⁵ This 'Japanese paradox' may be influenced by other protective factors including dietary components such as Japanese green tea, which is rich in polyphenols, especially catechins. In China, cigarette smoking increased threefold between the 1950s and 1987 and the first national smoking survey conducted in 1984 found the percentage of males over the age of 15 who smoked was similar to that in the USA during the 1950s.³¹ This trend will have an adverse affect on cardiovascular health and such effects will be more severe if the protective aspects of traditional lifestyles (diets low in saturated fat and physical activity) give way to more western habits.

Sex

Cardiovascular disease tends to affect women at an older age.³² Some of this sex-related difference is probably because of the potential benefits of oestrogen on lipid profiles, fibrinogen concentrations, blood pressure, arterial tone, blood glucose and insulin resistance.³³ However, lifestyle factors are also likely to contribute to this sex difference such as smoking.³⁴ As women tend to outlive men, the problem of CVD is going to grow in importance, and appropriate public health programs encouraging healthy lifestyles need to be actively promoted in this segment of the population.

Body composition

Obesity and abdominal fatness have both been shown to be predictors of CHD.³⁵ In older adults, obesity may be less of a risk factor for CHD than in younger adults, at least in men.⁴ However, the distribution of bodyfat may be more important; for example, in the Health Professionals Follow-up study, abdominal fatness in older men was more strongly associated

with coronary disease than BMI. A similar relationship was also found among a cohort of elderly men and women living in Verona, Italy.³⁶

Poor diet (reduced nutrient density, increased energy density)

Energy intake usually declines with advancing years and this has been largely attributed to a reduction in physical activity.³⁷ Reductions in energy expenditure and energy intake can impact adversely on CVD risk factors. Lower intakes of energy are also associated with an increased risk of nutrient deficiencies. Nutrient deficiencies in folate and vitamins B6 and B12 can lead to elevated levels of homocysteine. The intake of anti-oxidants may also be reduced at a time when anti-oxidant consumption may be of increasing importance. The age-related decline in taste and smell may also lead to less desirable food habits with the addition of salt and its possible consequences on blood pressure.

Alcohol

Alcohol can have both a positive and negative effect on cardiovascular health. It can elevate blood pressure increasing the risk of vascular damage and stroke, and it can contribute to hypertriglyceridaemia.³³ The protective effects of alcohol consumption relate to its potentially favourable impact on HDL cholesterol, plasma fibrinogen and platelet aggregation.³³

Homocysteine

Hyperhomocysteinemia is an independent risk factor for vascular disease.^{38,39} In the Framingham Study, nearly 30% of the original cohort aged between 67 and 96 years had high concentrations of homocysteine in their blood and in almost two-thirds of these cases the nutrient status of folate and one or more B vitamins was low.⁴⁰

Fibrinogen

Plasma fibrinogen is a risk factor for CHD and stroke,^{41,42} and its concentration increases with age.⁴³ The regular intake of fish may attenuate the risk of fibrinogen in relation to CVD, although the consumption of white fish has been shown to be positively associated (albeit weakly) with elevated fibrinogen concentrations.⁴³

Conclusion

Many lifestyle factors impact on cardiovascular health. Promoting desirable food habits, fostering social networks and encouraging physical activity can help to maintain cardiovascular health and prevent the development of risk factors important in CVD.

References

1. Wahlqvist ML, Savige GS. Interventions aimed at dietary and lifestyle changes to promote healthy aging. *Eur J Clin Nutr* 2000; 54: S148-S156.
2. Kaplan GA, Haan MN, Wallace RB. Understanding changing risk factor associations with increasing age in adults. *Annu Rev Public Health* 1999; 20: 89-108.
3. Barker DJP. Maternal nutrition, fetal nutrition, and disease in later life. *Nutrition* 1997; 13: 807-813.
4. Rimm EB, Stampfer MJ, Giovannucci E, Ascherio A, Spiegelman D, Colditz GA, Willet WC. Body size and fat distribution as predictors of coronary heart disease among middle-aged and older US men. *Am J Epidemiol* 1995; 141: 1117-1127.

5. Duncan AK, Vittone J, Fleming KC, Smith HC. Cardiovascular disease in elderly patients. *Mayo Clin Proc* 1996; 71: 184–196.
6. Glass TA, Leon CM, Marottoli RA, Berkman LF. Population based study of social and productive activities as predictors of survival among elderly Americans. *BMJ* 1999; 319: 478–483.
7. Andrews GR, Esterman AJ, Braunack-Mayer AJ, Rungie CM. *Aging in the Western Pacific*. Manila: World Health Organization, Regional Office for the Western Pacific, 1986.
8. Marmot MG, Syme SL. Acculturation and coronary heart disease in Japanese Americans. *Am J Epidemiol* 1976; 104: 225–247.
9. Campbell AJ, Buchner DM. Unstable disability and the fluctuations of frailty. *Age Ageing* 1997; 26: 315–318.
10. Fiatarone MA, O'Neill EF, Ryan ND, Clements KM, Solares GR, Nelson ME, Roberts SB, Kehayias JJ, Lipsitz LA, Evans WJ. Exercise training and nutritional supplementation for physical frailty in very elderly people. *N Engl J Med* 1994; 330: 1769–1775.
11. Winograd CH, Gerety MB, Chung M, Goldstein MK, Dominguez F Jr, Vallone R. Screening for frailty: criteria and predictors of outcomes. *J Am Geriatric Soc* 1991; 39: 778–784.
12. Bruunsgaard H, Skinhoj P, Pedersen AN, Schroll M, Pedersen BK. Ageing, tumour necrosis factor-alpha (TNF-alpha) and atherosclerosis. *Clin Exp Immunol* 2000; 121: 255–260.
13. Chandra RK. Effect of vitamin and trace-element supplementation on immune response and infection in elderly subjects. *Lancet* 1992; 340: 1124–1127.
14. Nieman DC, Henson DA, Gusewitch G, Warren BJ, Dotson RC, Butterworth DE, Nehlsen-Cannarella SL. Physical activity and immune function in elderly women. *Med Sci Sports Exerc* 1993; 25: 823–831.
15. Kondo K, Matsumoto A, Kurata H, Tanahashi H, Koda H, Amachi T, Itakura H. Inhibition of oxidation of low-density lipoprotein with red wine. *Lancet* 1994; 344: 1152.
16. Unno T, Kondo K, Itakura H, Takeo T. Analysis of (-)-epigallocatechin gallate in human serum obtained after ingesting green tea. *Biosci Biotechnol Biochem* 1996; 60: 2066–2068.
17. Hertog MGL, Feskens EJM, Hollman PCH, Katan MB, Kromhout D. Dietary antioxidant flavonoids and risk of coronary heart disease – the Zutphen elderly study. *Lancet* 1993; 342: 1007–1011.
18. Report of a WHO Expert Committee. Hypertension control. Technical Report Series no. 862. Geneva: WHO, 1996.
19. Fletcher A, Bulpitt C. Epidemiology of hypertension in the elderly. *J Hypertens* 1994; 12: S3–S5.
20. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, Bray GA, Vogt TM, Cutler JA, Windhauser MM, Lin PH, Karanja N. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med* 1997; 336: 1117–1124.
21. Bao DQ, Mori TA, Burke V, Puddey IB, Beilin LJ. Effects of dietary fish and weight reduction on ambulatory blood pressure in overweight hypertensives. *Hypertension* 1998; 32: 710–717.
22. Hagberg JM, Montain SJ, Martin WH, Ehsani AA. Effect of exercise training in 60- to 69-year-old persons with essential hypertension. *Am J Cardiol* 1989; 64: 348–353.
23. Seals DR, Reiling MJ. Effect of regular exercise on 24-hour arterial pressure in older hypertensive humans. *Hypertension* 1991; 18: 583–592.
24. Uusitupa MIJ, Sarkkinen ES. Risk factors of cardiovascular disease in the elderly. *Nutr Metab Cardiovasc Dis* 1998; 8: 341–348.
25. Iso H, Shimamoto T, Kitamura A, Iida M, Komachi Y. Trends of cardiovascular risk factors and diseases in Japan: Implications for primordial prevention. *Prev Med* 1999; 29: S102–S105.
26. WHO Study Group. Diet nutrition and the prevention of chronic disease. Technical Report Series no. 797. Geneva: WHO, 1990.
27. Yamada M, Wong FL, Kodama K, Sasaki H, Shimaoka K, Yamakido M. Longitudinal trends in total serum cholesterol levels in a Japanese cohort, 1958–86. *J Clin Epidemiol* 1997; 50: 425–434.
28. Stevenson ET, DeSouza CA, Jones PP, Vanpelt RE, Seals DR. Physically active women demonstrate less adverse age-related changes in plasma lipids and lipoproteins. *Am J Cardiol* 1997; 80: 1360–1364.
29. King H, Rewers M. Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. WHO Ad Hoc Diabetes Reporting Group. *Diabetes Care* 1993; 16: 157–177.
30. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang YY, Wang JP, Zheng H, Zhang H, Bennett PH, Howard BV. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance – The Da Qing IGT and Diabetes Study. *Diabetes Care* 1997; 20: 537–544.
31. Yu JJ, Mattson ME, Boyd GM, Mueller MD, Shopland DR, Pechacek TF, Cullen JW. A comparison of smoking patterns in the People's Republic of China with the United States: an impending health catastrophe in the middle kingdom. *JAMA* 1990; 264: 1575–1579.
32. Tsang TSM, Barnes ME, Gersh BJ, Hayes SN. Risks of coronary heart disease in women: Current understanding and evolving concepts. *Mayo Clin Proc* 2000; 75: 1289–1303.
33. WHO Scientific Group. Cardiovascular disease risk factors. New areas for research. WHO Technical Report Series no. 84. Geneva: WHO, 1994.
34. Jousilahti P, Vartiainen E, Tuomilehto J, Puska P. Sex, age, cardiovascular risk factors and coronary heart disease. A prospective follow-up study of 14,786 middle-aged men and women in Finland. *Circulation* 1999; 99: 1165–1172.
35. Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Bjorntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *Br Med J Clin Res Education* 1984; 288: 1401–1404.
36. Turcato E, Bosello O, Di Francesco V, Harris TB, Zoico E, Bissoli L, Fracassi E, Zamboni M. Waist circumference and abdominal sagittal diameter as surrogates of body fat distribution in the elderly: their relation with cardiovascular risk factors. *Int J Obesity* 2000; 24: 1005–1010.
37. James WPT. Energy. In: Horwitz A, Macfadyen DM, Munro H, Scrimshaw NS, Steen B, Williams TF, eds. *Nutrition in the elderly*. The World Health Organization. New York: Oxford University Press, 1989.
38. Clarke R, Daly L, Robinson K, Naughten E, Cahalane S, Fowler B, Graham I. Hyperhomocysteinemia: an independent risk factor for vascular disease. *N Engl J Med* 1991; 324: 1149–1155.
39. Chambers JC, McGregor A, Jean-Marie J, Obeid OA, Kooner JS. Demonstration of rapid onset vascular endothelial dysfunction after hyperhomocysteinemia – An effect reversible with vitamin C therapy. *Circulation* 1999; 99: 1156–1160.
40. Selhub J, Jacques PF, Wilson PWF, Rush D, Rosenberg IH. Vitamin status and intake as primary determinants of homocysteinemia in an elderly population. *JAMA* 1993; 270: 2693–2698.
41. Wilhelmsen L, Svardsudd K, Korsan-Bengtson K, Larsson B, Welin L, Tibblin G. Fibrinogen as a risk factor for stroke and myocardial infarction. *N Engl J Med* 1984; 311: 501–505.
42. Kannel WB, Wolf PA, Castelli WP, D'Agostino RB. Fibrinogen and risk of cardiovascular disease. The Framingham Study. *JAMA* 1987; 258: 1183–1186.
43. Lee AJ, Smith WC, Lowe GD, Tunstall-Pedoe H. Plasma fibrinogen and coronary risk factors: the Scottish Heart Health Study. *J Clin Epidemiol* 1990; 43: 913–919.