

Editorial

Healthy heart, happy life

Cardiovascular diseases have high rates of morbidity, mortality, and disability, and are the leading causes of human death irrespective of age, race, and region. According to the World Heart Federation, one third of adults over the age of 25 suffer from cardiovascular diseases globally¹. Every year over 17.5 million people die from cardiovascular diseases worldwide which contributes to 30 per cent of the annual death². The prognosis of cardiovascular diseases has been greatly improved in recent years with the development of medical technologies leading to a decrease in the morbidity and mortality. However, the morbidity and mortality due to heart diseases in the developing countries is increasing mainly due to environmental factors and unhealthy living habits. A great deal of attention is required to lead a healthy lifestyle and create an unpolluted environment to benefit the life and health of our hearts.

Smoking cessation: Smoking is closely associated with various cardiovascular diseases including coronary heart disease and hypertension³. Compared with non-smokers, the risk of cardiovascular diseases for smokers increases 1.6 fold. A report from the Asia Pacific Cohort Studies Collaboration indicates that smokers are 27 per cent more likely to develop ischaemic heart disease, 9 per cent more likely for haemorrhagic stroke, 4.5 times more likely for hypertension, and 16 times more likely for hyperlipidaemia⁴. Studies suggest that the risk of myocardial infarction of non-smokers who live with smokers increases by 23 per cent⁵. Summary analysis shows that in a person who has no history of cardiovascular disease, smoking cessation reduced the mortality of cardiovascular disease by about 2-35 per cent, which is similar to the effect antihypertensive intervention. Cessation before the age of 40 years

reduces the risk of death associated with continued smoking by about 90 per cent⁶. For those who have heart disease, smoking cessation can reduce the overall mortality by 12-35 per cent. This effect is significantly better than antihypertensive and lipid-lowering treatments⁷.

Diet: Diet is crucial in the development and prevention of cardiovascular disease and is one of the key factors that one can change for a healthy heart. Abnormal blood lipid levels have been shown to have a strong correlation with the risk of coronary heart disease⁸, and the abnormal blood lipid levels are directly related to the diet. A diet rich in saturated fats often causes high serum cholesterol levels. Unsaturated fats, like those found in fish, nuts, seeds and vegetables, are beneficial for heart. These sources of unsaturated fats contain essential fatty acids, including omega-3 and omega-6, which are beneficial to the heart and cannot be produced by the body.

Hypertension is another important risk factor for cardiovascular diseases, which is partly attributed to a high sodium diet. The daily intake of salt should be less than 5g according to the recommendations of the WHO⁹.

The unhealthy diets including fast food increase the risk of hypercholesterolaemia, hypertension, and diabetes, which ultimately damage the heart. To keep a healthy heart, it is necessary to have a diet low in saturated fats and salt, but with plenty of fresh fruit and vegetables.

Exercise: Exercise can influence a variety of cardiovascular regulatory peptides, and can lower C-reactive protein (CRP) levels and delay the development of cardiovascular diseases¹⁰⁻¹². Exercise

can reduce body fat levels and improve insulin sensitivity^{13,14}. Sticking to a long-term exercise regimen is an effective method to reduce CRP and both help prevent cardiovascular disease^{14,15}. Milani and colleagues¹⁶ compared the effects of cardiovascular rehabilitation training for three months in 235 coronary heart disease patients with 42 patients with no rehabilitation therapy. The results showed that the body fat index, motility, and other cardiovascular risk factors were significantly improved in the rehabilitated group¹⁶.

Pollution Control: It is suggested that environmental pollution is a greater cause to congenital heart disease than genetic factors. The development of the foetus is also influenced by a polluted environment during pregnancy and can ultimately lead to congenital heart disease. Although the epidemiological evidence is limited and inconsistencies remain, recent studies have suggested that maternal exposure to air pollution may also play a role in causing congenital anomalies, particularly congenital heart diseases^{17,18}.

The impact of pollution from the environment has been studied on 45 heart disease patients in an area of Helsinki, Finland¹⁹. Volunteers in the trial dramatically reduced the amount of blood flow into the heart after breathing polluted air for just two days. Though there was no pain associated with this change in heart function, it was a hallmark of heart disease progression. Epidemiological studies corroborate the elevated risk for cardiovascular events associated with the exposure to particle pollution (PM)_{2.5}. PM_{2.5} has been associated with the increased risks of myocardial infarction, stroke, arrhythmia, and heart failure exacerbation within hours to days of exposure in susceptible individuals²⁰.

In conclusion, cardiovascular diseases are a devastating set of diseases that are best combated by preventative measures including a healthy diet and a healthy lifestyle. The government agencies and individuals around the world should work together to create societies that promote the pursuit of a healthy heart and happy life.

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References

1. Chockalingam A, Balaguer-Vintro I, Achutti A, de Luna AB, Chalmers J, Farinero E, *et al*. The World Heart Federation's white book: impending global pandemic of cardiovascular diseases: challenges and opportunities for the prevention and control of cardiovascular diseases in developing countries and economies in transition. *Can J Cardiol* 2000;16 : 227-9.
2. WHO. Global atlas on cardiovascular disease prevention and control. Available from: http://www.who.int/cardiovascular_diseases/publications/atlas_cvd/en/2011, accessed on September 27, 2014.
3. Woodward M, Lam TH, Barzi F, Patel A, Gu D, Rodgers A, *et al*. Smoking, quitting, and the risk of cardiovascular disease among women and men in the Asia-Pacific region. *Int J Epidemiol* 2005; 34 : 1036-45.
4. Church TS, Levine BD, McGuire DK, Lamonte MJ, Fitzgerald SJ, Cheng YJ, *et al*. Coronary artery calcium score, risk factors, and incident coronary heart disease events. *Atherosclerosis* 2007; 190 : 224-31.
5. Wells AJ. Passive smoking and coronary heart disease. *N Engl J Med* 1999; 341 : 697-8.
6. Jha P, Ramasundarahettige C, Landsman V, Rostron B, Thun M, Anderson RN, *et al*. 21st-century hazards of smoking and benefits of cessation in the United States. *N Engl J Med* 2013;368:341-50.
7. Turnbull F, Blood Pressure Lowering Treatment Trialists' Collaboration. Effects of different blood-pressure-lowering regimens on major cardiovascular events: results of prospectively-designed overviews of randomised trials. *Lancet* 2003; 362 : 1527-35.
8. Kannel WB, Dawber TR, Friedman GD, Glennon WE, Mcnamara PM. Risk factors in coronary heart disease. An evaluation of several serum lipids as predictors of coronary heart disease; the Framingham Study. *Ann Intern Med* 1964; 61 : 888-99.
9. WHO, Sodium intake for adults and children. Available from: http://www.who.int/nutrition/publications/guidelines/sodium_intake/en/2012, accessed on September 28, 2014.
10. Abramson JL, Vaccarino V. Relationship between physical activity and inflammation among apparently healthy middle-aged and older US adults. *Arch Intern Med* 2002; 162 : 1286-92.
11. Wannamethee SG, Lowe GD, Whincup PH, Rumley A, Walker M, Lennon L. Physical activity and hemostatic and inflammatory variables in elderly men. *Circulation* 2002; 105 : 1785-90.
12. Rahimi K1, Secknus MA, Adam M, Hayerizadeh BF, Fiedler M, Thiery J, *et al*. Correlation of exercise capacity with high-sensitive C-reactive protein in patients with stable coronary artery disease. *Am Heart J* 2005;150:1282-9.
13. Kondo N, Nomura M, Nakaya Y, Ito S, Ohguro T. Association of inflammatory marker and highly sensitive C-reactive protein with aerobic exercise capacity, maximum oxygen uptake and insulin resistance in healthy middle-aged volunteers. *Circ J* 2005; 69 : 452-7.
14. Okita K, Nishijima H, Murakami T, Nagai T, Morita N, Yonezawa K, *et al*. Can exercise training with weight loss lower serum C-reactive protein levels? *Arterioscler Thromb Vasc Biol* 2004; 24 : 1868-73.

15. Tomaszewski M, Charchar FJ, Przybycin M, Crawford L, Wallace AM, Gosek K, *et al*. Strikingly low circulating CRP concentrations in ultramarathon runners independent of markers of adiposity: how low can you go? *Arterioscler Thromb Vasc Biol* 2003; 23 : 1640-4.
16. Milani RV, Lavie CJ, Mehra MR. Reduction in C-reactive protein through cardiac rehabilitation and exercise training. *J Am Coll Cardiol* 2004; 43 : 1056-61.
17. Padula AM, Tager IB, Carmichael SL, Hammond SK, Yang W, Lurmann F, *et al*. Ambient air pollution and traffic exposures and congenital heart defects in the San Joaquin Valley of California. *Paediatr Perinat Epidemiol* 2013; 27 : 329-39.
18. Vrijheid M, Martinez D, Manzanares S, Dadvand P, Schembari A, Rankin J, *et al*. Ambient air pollution and risk of congenital anomalies: a systematic review and meta-analysis. *Environ Health Perspect* 2011; 119 : 598-606.
19. Pekkanen J, Peters A, Hoek G, Tiittanen P, Brunekreef B, de Hartog J, *et al*. Particulate air pollution and risk of ST-segment depression during repeated submaximal exercise tests among subjects with coronary heart disease: the Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air (ULTRA) study. *Circulation* 2002; 106 : 933-8.
20. Simkhovich BZ, Kleinman MT, Kloner RA. Air pollution and cardiovascular injury epidemiology, toxicology, and mechanisms. *J Am Coll Cardiol* 2008; 52 : 719-26.