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Sociology, Community

Report Epidemiology Cardiovascular diseases and dietary fats

- Introduction

- Cardiovascular Diseases

- What is Cardiovascular Diseases (CVD)?

CVDs include diseases of the heart, vascular diseases of the brain and diseases of blood vessels such as coronary heart disease, atherosclerosis, hypertension, stroke, peripheral vascular disease and heart failure. Coronary heart disease (CHD) is the most deadly CVD that due to lacking blood flow to the network of blood vessels surrounding the heart, also named coronary artery disease (CAD) or ischemic heart disease (IHD) (Nabel and Braunwald, 2012).

- The pathology of heart attack and stroke

Atherosclerosis is the major cause of CVD. The underlying disease process in the blood vessels that results in coronary heart disease (heart attack) and cerebrovascular disease (stroke) is known as atherosclerosis. Atherosclerosis is a complex pathological process in the walls of blood vessels that develop over a long period. In atherosclerosis, fatty material and cholesterol are deposited inside the lumen of blood vessels (arteries), which cause the fibrosis plaque in the vessels. The plaque making it harder for blood to flow through and the plaque can rupture, which triggers the blood clot to develop into a potential heart attack (in coronary artery) or stroke (in the brain).

- Factors that influencing the CVD

There are several dimensions of factors that promote the process of atherosclerosis. Such dimensions include behavioural, metabolic and other

risk factors. The behavioural risk factors include tobacco use, physical inactivity, harmful use of alcohol and unhealthy diet (high intake of salt, fat and calories) (“ WHO | Global status report on non-communicable diseases”, 2010). Metabolic risk factors such as raised blood pressure (e. g. hypertension), raised blood sugar (e. g. diabetes), raised blood lipids (e. g. cholesterol) and obesity. It is indicated that elevated intakes of total, saturated fat, and dietary cholesterol serum cholesterol, which in turn increases the risk of developing coronary heart disease (CHD) (Esrey et al., 1996).

- Trends in Cardiovascular diseases

- Global Trends in Cardiovascular diseases

CVDs are responsible for over 17. 3 million deaths (mainly coronary heart disease, stroke and rheumatic heart disease) per year and are the leading causes of death in the world (World Health Organization, 2008).

- Demography

Driver et al. (2008) made the figure (figure 1), which shows age specific crude incidence of confirmed major cardiovascular disease by the first event (non-fatal myocardial infarction, non-fatal stroke, and death from cardiovascular disease).

Yusuf et al. (2001) introduced a graph (figure 2), showed the variations in rates of CHD between ethnic groups in Canada and categorized it by gender.

Figure 2. Variations in the rates of cardiovascular disease (CHD) between ethnic groups in Canada. 25 Note the high rates of CHD among Canadians of European and South Asian descent compared with the markedly low rates

among Chinese. The rates of cerebrovascular disease are low and similar in all three ethnic groups. By contrast, cancer death rates are lowest among South Asians and highest among Europeans, with Chinese exhibiting intermediate rates. This apparent dissociation of CHD and cancer rates is surprising because several of the common causes of heart disease and common cancers tend to be the same (e. g. tobacco use or obesity).

- Prevalence of Cardiovascular diseases in England

- The Projected Trends of CVD Mortality

The World Health Organization estimates there will be about 20 million CVD deaths in 2015, accounting for 30 percent of all deaths worldwide. By 2030, it is estimated that global cardiovascular deaths from 16. 7 million in 2002 will rise to 23. 3 million in 2030 (Mathers and Loncar, 2006).

- Dietary Suggestion for CVD Patients

Advice to substitute vegetable oils rich in polyunsaturated fatty acids (PUFAs) for animal fats rich in saturated fatty acids (SFAs) has been a cornerstone of worldwide dietary guidelines for reduction risk of coronary heart disease. Since that time, there has been increased recognition that the general category of PUFAs comprises multiple species of omega 3 (n-3) and n-6 PUFAs with unique biochemical properties.

Clinical cardiovascular benefits of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) have been extensively reported in several but no all randomized controlled trials (“ Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction,” 1999). However, there is currently no clinical trial evidence indicating that replacing SFA with n-6 linoleic acid (n-6 LA), without a concurrent increase in

n-3 PUFAs can lower the risk of cardiovascular disease or death.

In the study of evaluating the effects of replacing dietary intake of saturated fat with linoleic acid (n-6 PUFA), done by Ramsden et al. (2013), the author uses the Sydney Diet Heart Study (SDHS), which is a randomized controlled dietary trail conducted from 1966 to 1973. The SDHS provides a unique opportunity to evaluate the cardiovascular effects of replacing saturated fatty acid (SFA) with n-6 LA from safflower oil, which is especially rich in linoleic acid without other reported PUFAs. Eligible patients participated in this study were aged 30-59 years admitted to academic teaching hospitals in Australia, for an episode of acute myocardial infarction (86%), or acute coronary insufficiency or angina (14%). 237 participants were assigned to dietary intervention group which received instruction to increase their PUFA intake to about 15% of food energy, and to reduce their intake of saturated fatty acid (SFA) and dietary cholesterol to less than 10% of food energy and 300mg per day, respectively. 221 participants were assigned to the control group that received no specific dietary instruction. However, some participants began substituting polyunsaturated margarine for butter after their coronary event. Participants were both tested at baseline and 12-month follow-up for their blood cholesterol, triglycerides level and blood pressure, and also CVD and CHD mortality rates.

The table (table 1) represents the baseline characteristics of participants. From this table, we can tell that there were no significant differences between two groups. Both control and intervention groups have similar age (49. 1 vs. 48. 7), body mass index (BMI) (25. 4 vs. 25. 1), systolic blood pressure (136. 9 vs. 136. 6), total cholesterol (282. 0 vs. 281. 3),

triglycerides (TG) (185. 9 vs. 189) levels and glucose level (82. 2 vs. 84). But with slightly differences showed in smoking status (68. 8% vs. 71. 5% of smokers at admission) and alcohol consumptions (moderate: 44% vs. 37%; heavy 18. 1% vs. 20. 8%) between two groups.

This table (table 2) represents the baseline and follow-up dietary data from SDHS. With p value less than 0. 001, it shows a significant difference between groups in their PUFA, SFA, PUFA/SFA ratio, MUFA intake level and dietary cholesterol from a single seven-day food record (data showed the median number of the group that is categorized into interquartile range). In the intervention group, the participants increased their PUFA intake from 6. 1% of total daily calorie intake to 15. 4% with a significant 9. 3% change as compared to the baseline. As for SFA, the intervention group reduced their SFA intake from 16. 2% to 9. 3% total daily calories intake with a significant 6. 9% change as compared to the baseline. Total cholesterol also shows a significant difference between baseline and follow-up in the intervention group, which drops from 477 to 238 mg/day.

This table (table 3) shows the changes of risk factors such as total cholesterol level and TG level between baseline and 12-month follow-up. A reported P value of <0. 001 for the total cholesterol levels gives reasonable evidence to support the alternative hypothesis of a difference between the total cholesterol level in control and the intervention groups in the baseline and the 12-month follow-up.

In this figure (figure 4) shows the five years cumulative death rates of control and intervention groups. The three figures from the top to the bottom show the cumulative death rate of all cause, cardiovascular disease (classified by

International Classification of Diseases (ICD)-10) and coronary heart disease respectively. For all cause cumulative death rate, the intervention group has 17. 6 percent of the death rate while the control group has 11. 8 percent of the death rate from all cause. The hazard ratio is 1. 62 indicating that the intervention group has 1. 62 times increased all cause mortality than the control group (with 1. 00 as the lower bound of 95% confidence interval and 2. 64 as the higher bound of 95% confidence interval; p-value 0. 051). Compared to the control group (11. 0% CVD mortality rate), the intervention group has 17. 2% cardiovascular disease mortality rate. The intervention group also has HR of 1. 7, which means it is 1. 7 times higher CVD mortality risk than the control group. With 1. 03 as the lower bound of 95% confidence interval and 2. 80 as the higher bound of 95% confidence interval (p value= 0. 037), this means that it is unlikely to have arisen by chance. The probability that this difference is due to chance is less than 0. 037. So does the coronary heart disease (CHD) mortality, in compared with the control group with 10. 1% CHD mortality rate, the intervention group has 16. 3% of CHD mortality rate and 1. 74 times higher risk of death from CHD than the control group (p value= 0. 036). The hazard ratio has a lower bound of 95% confidence interval, 1. 04, and a higher bound of 95% confidence interval as 2. 92.

The table (table 4) shows the mortality outcomes (all cause, cardiovascular disease and coronary heart disease) according to the longitude changes in dietary fatty acid intake. The model 2 is after adjustment of age, dietary cholesterol, baseline BMI, smoking, alcohol use and marital status. When categorize the linoleic acid (LA) PUFA intake, the intervention group has the

hazard ratio of 1. 29 when compare the follow-up data with the baseline data. The p value of 0. 05 shows a significant difference and the difference is unlikely to have arisen by chance. The control group shows a hazard ratio of 0. 76 in saturated fatty acid (SFA) intake, which indicates that this factor might be a protective factor of the exposure. However, with the p value of 0. 25, there is no significant difference between groups, and it is likely that the difference is arisen by chance.

This table (table 5) shows the risk of cardiovascular death according to longitude changes in dietary PUFA, stratified by the source of oxidative stress. The hazard ratio of 1. 7 in moderate/heavy alcohol use, in the intervention group indicates that the individual who has moderate/heavy alcohol intake (> 200 kcal/day) has 1. 7 higher risks of suffering from cardiovascular death in a longitudinal term. With the p value <0. 01, indicates the there is a significant difference between the groups and it is unlikely to arise by chance. In the whole sample, all the participants who are moderate/heavy alcohol consumption has the hazard ratio (HR) of 1. 66 which means a 1. 66 times of higher risk in suffering from cardiovascular death than from the baseline. With the presence of the smoking habit in the whole sample, the smoker has HR of 1. 46, which indicates a 1. 46 times higher risk of suffering cardiovascular disease in the long term. This finding is aligns with the general acknowledgement that smoking and alcohol consumption are oxidative stress that could increase the mortality of the disease.

- Discussion

The dietary guidelines of substituting PUFA oil for saturated fatty acid (SFA)

intake have been suggested for the patients with cardiovascular disease (CVD) and coronary heart disease (CHD). When this dietary advice was first originated in the 1960s, PUFAs were regarded as an uniform molecular category with relevant biological mechanism, which is the reduction in blood cholesterol (Keys et al., 1965). Omega 6 (n-6) linoleic acid (LA) was the best-known dietary PUFA at that time. Therefore, the terms “ PUFA” and “ LA” were often used interchangeably when delivering dietary advice to the patient. With no clarified classification between n-3 and n-6 PUFA, the consumer might lack ability to choose and distinguish from daily dietary oil between n-3 and n-6 PUFA. There was research found that higher intake of n-3 fatty acid is associated with reduced cardiovascular disease mortality (Aarsetøy et al., 2008). However, currently, there is no clinical clarification that replacing SFA with n-6 PUFA and a concurrent increase in n-3 PUFA intake can reduce the cardiovascular disease or death. Therefore, benefits attributed to PUFAs as a general category might be due to n-3 PUFAs specifically, such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), which are mostly found in fish oil. Such benefits are not necessarily generalized to n-6 LA or other PUFA species. Since n-6 linoleic acid is the most abundant dietary PUFA that can be found in safflower oil, sunflower oil and cottonseed oil. Edible oil sources with markedly different contents of fatty acids are commercially available. It is important to ascertain the benefits and the risks specific to n-6 LA.

- References

Aarsetøy, H., Pönitz, V., Nilsen, O. B., Grundt, H., Harris, W. S., Nilsen, D. W. T., 2008. Low levels of cellular omega-3 increase the risk of ventricular

fibrillation during the acute ischaemic phase of a myocardial infarction. Resuscitation 78, 258–264. doi: 10. 1016/j. resuscitation. 2008. 04. 007

Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial. Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico, 1999. . Lancet 354, 447–455.

Driver, J. A., Djousse, L., Logroscino, G., Gaziano, J. M., Kurth, T., 2008. Incidence of cardiovascular disease and cancer in advanced age: prospective cohort study. BMJ 337, a2467–a2467. doi: 10. 1136/bmj. a2467

Esrey, K. L., Joseph, L., Grover, S. A., 1996. Relationship between dietary intake and coronary heart disease mortality: lipid research clinics prevalence follow-up study. J. Clin. Epidemiol. 49, 211–216.

Keys, A., Anderson, J. T., Grande, F., 1965. Serum cholesterol response to changes in the diet: I. Iodine value of dietary fat versus 2S-P. Metabolism 14, 747–758. doi: 10. 1016/0026-0495(65)90001-6

Mathers, C. D., Loncar, D., 2006. Projections of global mortality and burden of disease from 2002 to 2030. PLoS Med. 3, e442.

Nabel, E. G., Braunwald, E., 2012. A Tale of Coronary Artery Disease and Myocardial Infarction. N. Engl. J. Med. 366, 54–63. doi: 10. 1056/NEJMra1112570

Ramsden, C. E., Zamora, D., Leelarthaepin, B., Majchrzak-Hong, S. F., Faurot, K. R., Suchindran, C. M., Ringel, A., Davis, J. M., Hibbeln, J. R., 2013. Use of dietary linoleic acid for secondary prevention of coronary heart disease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. BMJ 346, e8707–e8707. doi: 10. 1136/bmj. e8707

WHO | Global status report on noncommunicable diseases 2010 [WWW Document], n. d. WHO. URL http://www. who. int/nmh/publications/ncd_report2010/en/ (accessed 4. 6. 14).

World Health Organization, 2008. World health statistics 2008. World Health Organization.

Yusuf, S., Reddy, S., Ôunpuu, S., Anand, S., 2001. Global Burden of Cardiovascular Diseases Part II: Variations in Cardiovascular Disease by Specific Ethnic Groups and Geographic Regions and Prevention Strategies. Circulation 104, 2855–2864. doi: 10. 1161/hc4701. 099488