Review article

Nutrition and cardiovascular disease: an Asia Pacific perspective

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Changes in the dietary intake patterns of countries in the Asia Pacific region are considered in relation to trends of cardiovascular disease mortality. Cardiovascular disease now constitutes the major cause of mortality in many of the countries of the region. The mortality rate for coronary heart disease (CHD) has been on the decline since the mid-1960s in countries such as Australia, New Zealand and Japan, while a decline in other countries, including Singapore and Hong Kong, appears to be occurring about two decades later after a delayed increase. In contrast, countries like Malaysia and China have had and continue an upward trend for CHD mortality. Nonetheless, the mortality rates due to CHD in New Zealand, Australia, Singapore followed by Hong Kong rank among the highest in the region. In China, Taiwan and Japan, death due to cerebrovascular disease remains a major cause of death, although the latter two countries have undergone a significant decline in stroke death rates since 1970.

The intakes of fat from land animal products, fish and vegetable oils, depending on fatty acid patterns and, possibly other constituents, are candidate contributors to the different atherogenic and thrombotic effects. Countries which have a higher mortality from CHD tend to have a higher intake of energy from fat and proportion of fat from animal products. These fat intakes may operate to increase hypercholesterolemia and overweight in various countries. Again, intakes of other food items and constituents used in the region such as soybeans, dietary antioxidants in fruits, vegetables, seeds, cereals, nuts and tea and alcohol consumption are candidate cardio-protectants.

The wide dietary scope of Asia Pacific populations, from diverse socio-cultural backgrounds, and at different levels of economic and technological development poses several analytic challenges and opportunities. Future research should improve the datasets and think laterally about pathogenesis and intervention.

Key words: Diet, cardiovascular disease, Asia Pacific, epidemiology, risk factors, coronary heart disease, antioxidants, lipids, hypertension, soy beans, obesity, diabetes

Introduction

International comparisons of the patterns and determinants of cardiovascular disease (CVD) go back more than 60 years ago. Raab in 1932, as quoted by Stamler[1], noted "the relative rarity of atherosclerosis and hypertension among the chiefly vegetable-consuming inhabitants of China, Africa, Dutch East India, and the enormous frequency of arteriosclerosis and hypertension among the peoples of Europe and North America who consume large quantities of eggs, butter....". Stamler[1] also quoted Rosenthal who had observed in 1934 that "in no race for which a high cholesterol intake (in the form of eggs, butter and milk) and fat intake are recorded is atherosclerosis absent....".

The epidemiologic relationship between diet and CVD in Western countries has been extensively researched, following the Framingham Study which began in the 1940s[2]. Several cross-cultural comparisons have also been undertaken in these countries such as the Seven Countries Study[3]; the International Atherosclerosis Project[4]; the Ni-Hon-San Study[5]; the Boston-Ireland Diet-Heart Study[6], and the WHO MONICA Project[7]. These studies have contributed a wealth of information towards the understanding of the roles of various risk factors of CVD, which include cigarette smoking, sedentariness, obesity, hypertension, diabetes and dyslipidaemia besides dietary factors.

Compared to Western countries, there are not as many studies pertaining to CVD and its risk factors in the developing countries. There is also a paucity of reliable data on deaths due to CVD in these countries due to a lack of complete vital registration and medical certification of deaths. For example, among countries of the WHO Western Pacific region, cause-of-death statistics are available only for 23% of the population, as compared to 94% for Europe[8]. In comparison, in the industrialised countries which include Japan, Australia and New Zealand, cause-specific mortality data by age and sex are available in the WHO mortality data base, some cases extending back to 1950.

In industrialised countries, deaths due to CVD account for almost half of all deaths[8]. Within this category, coronary heart disease (CHD) or ischaemic heart disease is the single most important cause of mortality. In 1990, out of 10.9 million deaths in industrialised countries, just over 5.3 million were due to CVD, primarily CHD (2.7 million or 25% of total deaths) and cerebrovascular disease (1.4 million or 13% of total deaths). Since the mid 1960s, a number of these countries including Japan, United States and Australia have experienced considerable decline in CVD mortality[9]. In the case of developing countries, however, Bulato, Lopez and Stephens[10] estimated that by the year 2015, the proportion of deaths caused by CVD will be almost twice the level in 1985 (Table 1). This is not unexpected given that the majority of the people in the developing countries are under 35 years of age and CVD risk factors like obesity and hypercholesterolemia are already prevalent among the young.

The Asia Pacific region represents an important area in terms of its economic, technological, and demographic resources. The population in this region accounts for about one-third of the total

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global population (Table 2). These countries have a multitude of ethnic groups with widely diverse dietary habits. The health risks faced by these countries range from traditional environmental exposures (such as poor sanitation), and modern agricultural hazards (such as pesticide contamination of water and food), to those associated with urbanisation and industrialisation (such as chronic non-communicable diseases). Countries in this region also differ widely with respect to their nutritional status, ranging from countries still grappling with problems of undernutrition (Papua New Guinea, Indonesia, the Philippines), to those who are beset more with health problems associated with overnutrition (Singapore, Hong Kong, Australia, New Zealand). In between are countries undergoing nutrition transition faced with both the "old" problems of nutrient deficiencies and the new problems of overnutrition (China, Malaysia, Thailand, Taiwan).

Table 1. Estimated and projected distribution of the major causes of death.

Disease category	Developed countries		Developing countries	
	1985 %	2015 %	1985 %	2015 %
Infectious & parasitic diseases	9	7	36	19
Neoplasms	18	18	7	14
Diseases of circulatory system &certain degenerative diseases	50	53	19	35
Complications of pregnancy	0	0	1	1
Perinatal conditions	1	1	8	5
Injury and poisoning	6	5	8	7
All other and unknown causes	15	16	21	19
All causes	100	100	100	100

Source: Bulato, Lopez & Stephens, 1989 (Ref10)

Table 2. Demographic indicators of some Asia Pacific countries

Country	Population (million)	Life expectancy at birth Men 1993	Women 1993
China	1,205.2	69.0	73.0
Indonesia	194.6	62.0	65.0
Japan	125.0	76.3	82.5
Vietnam	71.0	62.0	67.0
Philippines	66.5	63.0	67.0
Thailand	56.9	65.7	70.4
		(1992)	(1992)
Republic of Korea	44.5	69.0	76.0
Malaysia	19.2	69.0	73.0
Australia	17.8	73.7	80
		(1992)	(1992)
Pacific Islands Total	6.7		
Papua New Guinea	4.2	55.0	57.0
Fiji	0.8	70.0	74
Solomon Islands	0.4	69.0	73
Hong Kong	5.9	75.0	81.0
New Zealand	3.5	72.0	78.0
Singapore	2.8	73.7	78.3
		(1992)	(1992)

Source: Statistical Yearbook for Asia and the Pacific United Nations ESCAP, Bangkok, 1994

This article compares the CVD situation among the countries in the Asia Pacific region, focusing on the patterns of mortality due to coronary heart disease (CHD) and cerebrovascular disease. The prevalence of dietary risk factors of CVD in the various countries in the region is discussed at length. Included are fat intake from animal products, fish and vegetable oils. Also presented in the discussion are other dietary items with CVD implications such as vitamin antioxidants namely, vitamin E, β-carotene and vitamin C, soybean and alcohol consumption. Cardiovascular disease risk factors related to diet as exemplified by obesity and diabetes mellitus are also discussed.

Limitations of review

A comparison of the CVD situation in such a widely diverse array of backgrounds poses several challenges. A major constraint lies in the limited intercountry comparability of mortality data. Death attributed to CVD in one country might be attributed to a different form of heart disease in another country . This variation can be due to differences in diagnosis and terminology on death certificates, or in the coding methodology across nations as applied to the International Classification of Diseases (ICD)[11]. The lack of medical personnel to certify the underlying cause of death and the unavailability of diagnostic aids especially in rural areas can also affect the quality and hence the comparability of mortality data internationally. Low figures reported may not necessarily reflect low incidence or mortality levels but may be due to inaccuracies in data collection[12].

In this article, two major types of CVD, coronary heart disease and cerebrovascular disease, are highlighted. For the purpose of intercountry comparisons here, mortality due to CHD refers to codes 410-414 in both the Eighth Revision (introduced in 1968/70) and the Ninth Revision (introduced in 1979/80) of the ICD. The ICD codes for cerebrovascular disease are 430-438.

The trends in CHD mortality have been well documented for some countries in the region including Australia, New Zealand, Japan and Singapore. However, inter-report comparison faces the constraint of reports covering varying age groups and time periods. For example, while some reports showed CHD mortality trends for subjects aged 40-69, others described the trends for ages 45-64[11,13]. Different reports described different age intervals (ages 30-34, or 30-39 or 30-44), thereby rendering direct comparisons difficult. Some reports present mortality rates as age-adjusted to the world population standard[14], while individual country reports often are not presented in this manner.

A substantial amount of data was drawn from the publications of the World Health Organisation and the Food and Agriculture Organisation. A constraint arises for Taiwan which is not included in these reports as it is not a member of the United Nations. The availability of data for Taiwan and other countries was dependent upon reports based on MEDLINE search output.

In light of the lack of reliable primary dietary data from surveys for several countries in the region, food availability data from FAO Food Balance Sheets are used to compare the dietary patterns and changes of Asia Pacific countries. The shortcomings of food balance sheet data or "food disappearance" data are well recognised, especially with regards to the data not reflecting distribution and accessibility by different socio-economic groups. The data may over-estimate actual consumption by as much as 25%[15]. Nonetheless, food balance sheets represent a useful source of information from which inter-country comparisons of dietary trends and patterns can be made.

Cardiovascular disease mortality

Given the diverse socio-economic background of Asia Pacific countries, it is not surprising that the region has a wide range in CVD mortality rates and trends. In New Zealand and Australia, the proportion of mortality attributed to CVD for men accounts for over 40% of total mortality (Table 3). This level is in line with that for industrialised countries (49%)[16]. However, Japan is an exceptional case among industrialised countries in that its mortality due CVD accounts for less than 30% of total death cases. This level in Japan places it among less industrialised countries such as Malaysia and the Philippines. In comparison, Singapore and the urban parts of China have intermediate mortality levels for CVD which lie between 30-40% of total deaths.

The CVD proportionate mortality for women in the Asia Pacific countries shows a similar pattern as that for men, with

New Zealand topping the countries followed by Australia and Singapore (Table 3). Women in Japan, the Philippines and Thailand rank among the lowest in terms of the proportion of death cases due to CVD.

Taylor, Lewis and Levy[17] in reporting the mortality patterns in the Pacific Islands, revealed that the proportionate mortality for CVD is relatively high in the more developed islands associated with the United States, New Zealand and among the New Caledonian Europeans. In comparison, the less developed Melanesian countries manifest higher proportional mortality from infectious disease.

Table 3. Proportionate mortality from cardiovascular diseases.

	Male			Female		
	Cardio-vascular disease	Coronary heart disease	Cerebro vascular disease	Cardio-vascular disease	Coronary heart disease	Cerebro vascular disease
Industrialised countries 1990*	48.6	21.4	10.5	59.0	21.9	16.8
New Zealand	45.8	34.7	4.8	30.3	16.7	7.4
Australia	41.7	31.1	5.1	28.8	16.6	7.1
China (Beijing)	40.6	10.4	20.8	45.8	7.3	24.3
Singapore**	35.1	19.7	9.8	39.0	17.9	14.5
Malaysia***	29.1	12.7	7.5	30.1	9.4	10.3
Japan	27.7	5.5	12.3	26.8	3.5	13.6
Philippines**	25.6	6.8	5.5	28.1	6.4	5.9
Thailand**	17.3	0.4	2.7	15.3	0.4	2.2

Sources: World Health Statistics Annual, 1989 (ref 18); *Lopez, 1990 (ref 16); **SEAMIC Health Statistics, 1993 (ref 19); ***Vital Statistics Peninsular Malaysia, 1991 (ref 20).

The low proportionate mortality for CVD in some countries like Thailand, Malaysia and Indonesia could be due in part to the fact that these countries have a low proportion of death cases that are medically inspected and certified. For example, in Malaysia, only about 41% of the annual total deaths are medically certified[21], compared to 65-70% in Singapore[22] and 100% in Australia, New Zealand and Japan[18]. For countries with a relatively low rate of medical certification, the cause-of-death statistics refer largely to only the urban population or selected sub-population groups. Hence, the interpretation of such data should be circumspect.

Mortality rates for coronary heart disease

One may arbitrarily categorise the Asia Pacific countries into three categories according to their mortality rates for CHD for men. The first category is comprised of New Zealand, Australia and Singapore with CHD mortality rates exceeding 100 per 100,000 population for all ages. As shown in Table 4, New Zealand is first with a mortality rate of 228 per 100,000 population for men and 173 for women followed by Australia with 191 and 161 for men and women, respectively. Singapore lies third with values of 104 for men and 77 for women, respectively. When compared on the basis of age-standardised to world standard population[14], however, the CHD mortality in Singapore is similar to Australia, that is, 132 (men) and 78 (women) for Singapore and 140 (men) and 73 (women) for Australia (Table 5). The respective figures for New Zealand with the highest mortality rates are 173 and 84 per 100,000.

Hong Kong belongs to the next highest category of CHD mortality rates with a value of between 50-100 per 100,000 population (Table 4). Heart diseases with CHD as the major category have become the second leading cause of mortality during the past 20 years[23]. Socio-economic class is known to influence CHD mortality patterns. It has been reported in Hong Kong that the more affluent population groups, which include professional, administrative and managerial workers, have higher standardised mortality rates for CHD than do manual and agricultural workers[24,25].

The third category, with the lowest range of CHD mortality rates of below 50 per 100,000, includes Japan, China, the Philippines, Malaysia, Thailand (Table 4) and Indonesia[26]. Japan stands out as an industrialised country influenced by a Western lifestyle but at the same time, it shows a low CHD mortality rate. When compared on the basis of age-standardised to world standard population, the CHD mortality of Japan (at 28 per 100,000) is about one-sixth that of New Zealand (at 173) and one-fifth that of Australia (140) for both sexes[14]. Regional differences exist in that age-adjusted CHD mortality is higher in urban prefectures such as Tokyo and Osaka than in the rural counterparts[27].

Table 4. Cardiovascular disease mortality rates in Asia Pacific countries (for all ages, per 100,000 population).

	Male			Female		
	Coronary heart disease	Cerebro vacular disease	Circulatory system diseases	Coronary heart disease	Cerebro vacular disease	Circulatory system diseases
New Zealand 1991	228	62	335	173	95	336
Australia 1992	191	55	304	161	80	312
Singapore 1991	104	55	190	77	69	177
Hong Kong 1991	56	49	140	37	56	142
Japan 1992	45	92	247	38	100	256
China 1990						
urban	49	126	222	46	117	222
rural	24	104	177	21	104	182
Philippines 1991*	38	30	142	24	22	107
Malaysia 1990**	31	15	69	19	16	45
Thailand 1992*	24	15	98	2	9	60

Sources: WHO Health Statistics Annual, 1993 (ref 14)
*SEAMIC Health Statistics, 1993 (SEAMIC, 1995) (ref 19)
** Vital Statistics Peninsular Malaysia, 1991 (ref 20).

Table 5. Cardiovascular disease mortality rates in Asia Pacific countries. (age-standardised rates, per 100,000 population)*

	Male			Female		
	cardio-vascular disease	coronary heart disease	cerebro vascular disease	cardio-vascular disease	coronary heart disease	cerebro vascular disease
New Zealand 1992	268	173	46	159	84	43
Singapore 1991	241	132	72	178	78	68
Australia 1992	222	140	39	139	73	35
Japan 1992	157	28	58	101	15	40

* age-standardised to world standard population
Source: World Health Statistics Annual, 1993 (ref 14)

CHD in China was once a disease of little significance, but has become increasingly important. In 1958, Tung (as quoted in Tao *et al*[28]) described "the uncommon occurrence in China of atherosclerotic heart disease which comprised of only 7% to 12% of adult cardiac cases seen in hospitals and clinics". The mortality rate attributed to CVD has increased from 86.2 per 100,000 in 1957 (12.1% of total deaths) to 214.3 per 100,000 in 1990 (35.8% of all deaths)[29]. It is to be expected that a vast country like China might have a very wide range of values for the incidence of and mortality from CHD[30]. In the urban areas, CHD is the leading cause of death and its mortality rate is twice as high as in rural areas for both men and women. According to the Sino-MONICA Project which commenced in 1983 to monitor the trends in

morbidity and mortality from CVD in China, CHD incidence is higher in the northern provinces than in the southern areas[29]. Qingdao in the northern province of Shandong had the highest incidence of CHD at 203 and 96 per 100,000 for men and women aged 35-74, respectively, between 1985-89, compared to Chuxian in the south with one-tenth the levels in Quingdao.

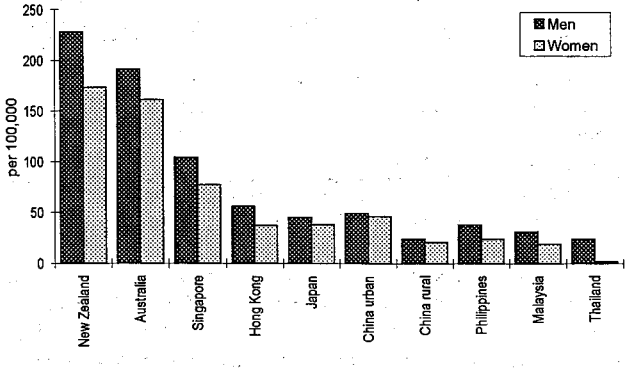
Mortality due to CVD has emerged as a major cause of death in the last two decades in the Philippines, Malaysia, Indonesia and China. In the Philippines, CVD was not among the ten leading causes of death before 1962, but since 1964, it has become the second most frequent cause of death after respiratory diseases[31,32]. In Indonesia, CHD has replaced rheumatic heart disease as the most prevalent diagnosis among patients admitted with CVD[33].

In Peninsular Malaysia, CVD has become the leading cause of death since 1970[20]. In Malaysia and Singapore, Indians are consistently reported to have the highest CHD mortality rate among the other ethnic groups in those two countries namely, Chinese and Malays. In 1990, the mortality rate for Indians in Peninsular Malaysia was 51.8 per 100,000 (for all ages) compared to 25.5 and 14.4 for Chinese and Malays respectively[34]. In Singapore, the age-adjusted death rate for CHD for Indian men is three times higher than that for Chinese men and one and a half times that for Malay men[22]. South Asians in other countries (for example, England, Fiji, West Indies, South Africa and United States) have also been shown to have a higher incidence of CHD compared with other ethnic groups as reviewed by Rao and White[35].

Among less socio-economically advanced countries in the region less influenced by Western diet, CVD does not yet rank as the leading cause of death. For example, in Papua New Guinea, the major forms of heart disease are more typical of developing countries (rheumatic heart disease, congenital heart disease and cardiomyopathy)[36]. Lindeberg and Lundh[37] also reported that CHD and stroke appear to be absent among the traditional Melanesian horticulturalists in Kitava, Trobriand Island and Papua New Guinea.

Figure 1 illustrates the CHD mortality rates of men and women in the Asia Pacific countries. In general, men have a higher CHD mortality rate than women when considered for all ages. In older age groups, however, the sex mortality differential narrows as exemplified by the case in Malaysia in 1990. On an age-standardised basis, the sex mortality ratio for ages 35-49 was 4.7 and it decreased to 1.9 for ages 50 and above[34].

Figure 1. Coronary heart disease mortality in Asia Pacific countries.



Trends in coronary heart disease mortality

The trend in CHD mortality in industrialised countries was generally upward until the late 1960s[38]. CHD became the leading cause of mortality for middle-aged and older people. Nonetheless, the situation has reversed since the early 1970s with the CHD mortality trend on the decline. For example in the Asia Pacific region, Thom[11] reported that between 1969-78, Australia showed a decline of 24% for men aged 45-64 years of age, and New Zealand decreased by 22%. Further declines were shown between 1979-85 for men of this age group, with decreases of 25% and 20% respectively for Australia, and New Zealand. Women aged 45-64 in these three countries also experienced substantial declines in CHD mortality during these two periods.

The decline in Australia has occurred in all age groups but to a greater extent in women and younger age groups[39]. The CHD death rates continue to decline at 3-5% per annum without sign of tapering off (Table 6). Cardiovascular disease is no longer the major cause of death among young and middle-aged Australian men and women[40]. Most deaths in this group are due to cancer. Nonetheless, CVD remains the major cause of death among the elderly of both sexes.

In New Zealand, as in Australia, the greatest decline was observed for younger age groups in males and females[41]. However, it is noted that despite the significant magnitudes involved in the decline of CHD mortality in both Australia and New Zealand in the past two decades, CHD is still the leading cause of death in these countries. By world standards, CHD death rates in Australia and New Zealand remain high[42].

Table 6. Changes in mortality rates of coronary heart disease for ages 25-64 in Asia Pacific countries.

	Male (%)		Female (%)	
	1970/74 - 1980/84	1980/84 - 1985/89	1970/74 - 1980/84	1980/84 - 1985/89
Australia	-39.1	-25.4	-42.0	-28.5
New Zealand	-27.9	-17.6	-29.2	-22.4
Japan	-13.2	-10.9	-33.6	-20.8
Singapore	+14.2	-21.2	+29.0	-2.5
Malaysia*	+25.5	+8.2	+63.5	+19.4

Sources: WHO Health Statistics Annual, 1993 (ref 230)
*Vital Statistics Peninsular Malaysia, 1974-91 (ref 21)

In comparison, while the CHD mortality of Japan increased in the 1950s until about 1967, and thereafter declined for both men and women, the CHD mortality rate in Japan has been at a low level since the 1950s[43]. The current CHD mortality rate of Japan is less than one quarter that of Australia and one-fifth that of New Zealand. The declining trend in Japan shows women and younger age groups making earlier starts and clearer declines[27,44].

Hong Kong and Singapore are examples of affluent Asian countries with distinct Western influences on their dietary habits and lifestyles. Both countries experienced an increase in CHD mortality in the 1970s. The secular trend in Hong Kong between 1970-79 showed a significant increase of 1.7% each year for men and 1.9% for women for all ages[23]. The increase for Singapore men was 14.2% and 29% for women between 1970/74-1980/84 (Table 6). Thereafter, both countries have shown a downward trend in their CHD mortality trends. Hong Kong showed small declines of 0.5% and 0.3% per year for men and women respectively, while Singapore experienced larger rates of decline, especially for the men, that is, by 21.2% and 2.5% for women. The sex mortality ratio for CHD in Singapore for ages 30-69 shifted from 3.5 in 1959/63 to 2.8 in 1979/83, reflecting a greater decline for men during that period.

These two countries appear to be undergoing a decline in CHD mortality about two decades later than in the United States, Australia, New Zealand and Japan. Hughes[45] pointed out that the decline in Singapore seems to begin with a younger age group for a successively earlier period, (1974-78 in ages 45-49 years; 1969-73 in ages 40-44 years, and 1964-68 in ages 35-39). This could be due more to a cohort effect rather than due to a particular year of death.

While industrialised countries (Australia, New Zealand and Japan) and the other affluent countries in the region (Singapore and Hong Kong) are on the downward trend in CHD mortality, there are other countries which appear to be maintaining an upward trend. Malaysia and China are two such countries. The CHD mortality in Malaysia was 8.0 per 100,00 population for all

ages in 1965 and this rate has been on the rise, reaching 23 in 1991[34]. The rise has been more rapid for women, whose CHD mortality more than doubled between 1975-90 (from 6.1 to 13.6), compared to about 30% increase for men during the same period.

The CHD trends for China are extrapolated from the situations in the cities of Beijing, Shanghai and Guangzhou between 1976-86 as reported by Tao *et al*[28]. During this period, the CHD death rates for ages 35-74 showed a steady increase for Guangzhou and Shanghai, whereas there was no discernible change for Beijing. Nonetheless, the level in Beijing in 1986 was three times that of Shanghai and Guangzhou. When compared on an age-standardised basis for men and women (aged 35-74) between urban Beijing and other countries, Beijing has twice the rate of Japan but one-third that of Australia.

Mortality rates for cerebrovascular disease

Cerebrovascular disease or stroke remains the principal cause of morbidity and mortality among adults in several Asian and Pacific Island populations. China, Taiwan and Japan rank highest in terms of stroke mortality in the region. The mortality rates for cerebrovascular disease in these three countries stand close to 100 or higher per 100,000 population for men and women for all ages[46] (Table 4). In China, stroke is the leading cause of death, while in Taiwan, it currently ranks second, next to cancer, after being the main cause of death for almost 20 years from 1963-1982[46]. The major type of stroke in Taiwan is cerebral infarction while for Japan and China, it is cerebral haemorrhage[47,48].

In Japan, twice as many men (2.1) and women (2.6) die from stroke than from CHD[28] (Table 4). In China, the stroke to CHD mortality ratio ranges from about 2.5 in urban areas to almost 5.0 in rural areas. Chonghua, Zhaosu and Yingkai[29] reported that the incidence of stroke is four times that of acute myocardial infarction in some areas in China. There are about five million surviving stroke patients and some 1.3 million new cases occur each year. In comparison, New Zealand and Australia have a stroke to CHD mortality ratio of only 0.3 for men and about 0.6 for women.

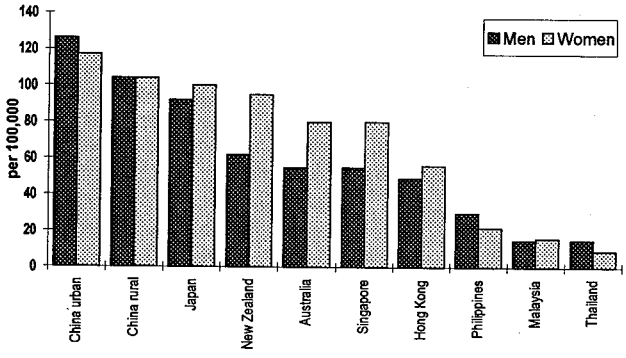
There are geographic and socio-economic differentials in stroke mortality just as for CHD mortality. In Taiwan, the mortality rates are higher in the northern regions and in urban areas[46]. Similarly, the northern provinces of China have a higher incidence of and mortality rate from stroke then the south. There is a three to fivefold difference between the mortality rates for the north and the south for both men and women[29]. In Japan, workers in agriculture, sales, transportation and service industries have higher rates of cerebrovascular disease than those in managerial and administrative positions[27].

Countries in the region with a lower mortality rate for stroke of between 50-100 per 100,000 population include New Zealand, Australia, Singapore and Hong Kong. Their stroke mortality rates for men in 1991/92 ranged from 39 per 100,000 population in Australia and 72 for Singapore, and among women, the rates ranged from 34 per 100,000 population for Australia to 68 for Singapore[14].

The next group of countries with the lowest level of stroke mortality rate in the region are the Philippines, Malaysia and Thailand[49]. Their stroke mortality rate is below 50 per 100,000 population. The question that arises is, as previously mentioned with CHD mortality, whether the low stroke mortality rate actually reflects the real situation, or is it due to under-reporting of cases owing to a low level of medically certified death cases in these countries.

Figure 2 shows the cerebrovascular mortality rates of men and women in the region. Slightly more men than women die from cerebrovascular disease when the stroke mortality rate between men and women is compared on the basis of age-standardised to world population. The stroke mortality female:male ratio in the region ranges from 0.7 for Japan and China, to 0.8 for Taiwan and Hong Kong and to 0.9 for Australia, New Zealand and Singapore[14,30]. On an age-specific basis, in 1991/92 for ages 25-64, the female:male ratio for stroke mortality ranged from 0.6 for Japan to 0.7 for Singapore and 0.8 for both Australia and New Zealand. The gap narrows in the older age category of 64 years and above, where the female:male mortality ratio approached 0.9 for Japan and 1.2 for Singapore, Australia and New Zealand. This indicates that women become more prone to stroke at older ages than men. An epidemiologic study in Taiwan further demonstrated this trend[46]. The sex ratio for the prevalence of stroke was found higher for men for the younger ages from 36-64, but the ratio reached 1.0 for ages 65-74. However, for ages 75 and above, the prevalence for stroke was again higher for men.

Figure 2. Cerebrovascular disease mortality in Asia Pacific countries.



Trends in cerebrovascular disease mortality

Some Asia Pacific countries with high mortality rates for cerebrovascular disease have experienced a substantial quantum of decline since 1970. Among the countries in the region with the highest tertile of mortality for cerebrovascular disease (China, Taiwan and Japan), Japan has undergone the most substantial decline since 1970 for both men and women (Table 7). The decline in Japan was 20.1% for men and 22% for women in the 1980s. Taiwan has also experienced a reduction in its stroke death rate, albeit less rapidly than Japan during about the same period. Its decrease was 17.5% for men and 18.5% for women between 1972 and 1983[50], as compared to 43.6% and 42.4% for men and women respectively for Japan between 1970/74 and 1980/84.

Table 7. Changes in mortality rates of cerebrovascular disease for ages 25-64 in Asia Pacific countries.

	Male (%)		Female (%)	
	1970/74-1980/84	1980/84-1985/89	1970/74-1980/84	1980/84-1985/89
Australia	-43.7	-32.5	-51.9	-32.7
Japan	-43.6	-20.1	-42.4	-22.0
New Zealand	-38.7	-19.5	-42.8	-30.6
Singapore	-35.6	-22.7	-35.2	-26.5
Malaysia*	-15.6	-10.3	+3.1	-5.7

Sources: WHO Health Statistics Annual, 1993 (ref 14)
*Vital Statistics Peninsular Malaysia, 1974-92 (ref 21)

In Singapore, decline in cerebrovascular disease commenced in early 1970 for males and mid-1970s for females[51]. Between 1969 and 1983, the decline was more marked in males than females for ages above 40 years. This differential appears to have narrowed as indicated in Table 6, which shows the declines in males and females between 1980/84 and 1985/89 as quite close (22.7% and 26.5% respectively).

Australia and New Zealand, which have the second highest tertile of stroke mortality rate in the region, have also undergone declines as rapidly as Japan since 1970. The magnitude of the decline in the former countries in the 1970s ranged between 39-44% in males and between 43-52% in females (Table 6). It is noteworthy that higher female rates of decline were found in

Australia and New Zealand, and the same pattern prevails between 1980/84 and 1985/89 (Table 7). This pattern is the reverse of the situation in Singapore and also in the United States and the United Kingdom, where higher male rates of decline were encountered as observed by Hughes[51].

The stroke mortality decline for Malaysia has not been as rapid as the above mentioned countries. Between 1980/84 and 1985/89, it declined by 10.3% for men and 5.7% for women. The present mortality rate for stroke in Malaysia stands among the lowest level in the region, but this is based on only 41% of total deaths which are medically certified.

Based on limited data, China appears not to have experienced the marked decline in stroke mortality as other Asia Pacific countries. Monitoring of stroke mortality in Beijing from 1985 to 1989 showed no change in the incidence of stroke, though mortality from stroke was observed to decline slightly in both sexes[29].

Dietary risk factors

The substantial decline in CHD mortality since the mid-1960s in the United States and other industrialised countries has led to numerous studies on the determinants of CHD mortality decline. The main contributions towards the decline have been attributed to medical interventions, improved control of hypertension, decline in cigarette smoking and reduction in plasma cholesterol[52]. Meta-analysis of several randomised controlled trials of cholesterol reduction suggests that a 10% decrease corresponds on average to a reduction of about 25% in CHD incidence[53]. The development of atherosclerosis goes beyond raised levels of serum total cholesterol and LDL-cholesterol. Evidence is accumulating on the crucial role played by the oxidised form of LDL-cholesterol[54]. Oxidised LDL has several atherogenic properties including that it is chemotatic, it increases platelet aggregation, and it promotes smooth muscle proliferation[55]. Oxidised LDL-cholesterol is taken up by macrophages forming foam cells. These cells accumulate as fatty streaks in the intima of the arteries leading to irregular narrowing of the blood vessels (stenosis)[56].

The other process which contributes to the development of CHD is thrombosis. In a normal physiological state, once a thrombus has completed its task of wound repairing on the endothelial surfaces of blood vessels, there is a mechanism for dissolving the thrombus so that it can cause no obstruction. A defect in any stage of the mechanism can result in a thrombus detaching itself and it may then block a coronary vessel precipitating a myocardial infarction[57]. The type of dietary fat consumed may contribute to both atherosclerosis and thrombosis.

Dietary fat

Implications of dietary fat and hypercholesterolemia

Many epidemiological and experimental studies have demonstrated the association between high intakes of fat with high levels of serum cholesterol, although conflicting evidence still prevails[15]. Plasma cholesterol concentration is known to increase when dietary carbohydrates are replaced by certain saturated fatty acids (SFA) and to decrease when carbohydrates are replaced by polyunsaturated fatty acids (PUFA). This quantitative responsiveness of plasma lipids was first summarised in the predictive equations established by Keys[58] and Hegsted[59]. Subsequently, Mensinck and Katan[60] and Hegsted *et al*[61] developed formulas to predict dietary responsiveness of not only total cholesterol, but also of low density lipoprotein (LDL) and high density lipoprotein (HDL) cholesterol in both men and women.

It is recognised that some SFA are more atherogenic or hypercholesterolaemic, while others have a greater role in thrombogenesis. Fatty acids up to 10 carbon atoms do not influence plasma cholesterol as they are absorbed directly into the blood and rapidly metabolised in the liver, unlike the longer chain acids which are absorbed as chylomicrons. Stearic acid (C18:0) is confirmed to be neutral in its effect on plasma total cholesterol and lipoprotein cholesterol concentrations in men and women[62].

In contrast, lauric acid (C12:0), myristic acid (C14:0) and palmitic acid (C16:0) collectively contribute to 70% of the raising of plasma cholesterol concentration by total SFA[60]. The regression analyses of Yu *et al*[62] suggest that myristic acid is 5-6 times more hypercholesterolaemic than either lauric acid or palmitic acid.

The hypercholesterolaemic effect of palmitic acid appears to vary depending on the experimental design used. While studies reporting on the ability of palmitic acid to elevate plasma total cholesterol and LDL-cholesterol concentrations persist[63-65], others have found palmitic acid to elevate plasma cholesterol only in hypercholesterolaemic subjects (serum cholesterol >5.8 mmol/L) and especially those consuming diets with >400 mg cholesterol/day, but not in normocholesterolaemic subjects consuming diets containing <300 mg cholesterol/day[66-68]. Dietary fats rich in SFA include coconut oil, palm kernel oil, fat from beef, pork and mutton, and milk fat and its products such as butter, cream and cheese.

Cis-monounsaturated fatty acids (cis-MUFA) have been demonstrated to be hypocholesterolaemic but less so than PUFA. In contrast to PUFA, cis-MUFA do not lower HDL-cholesterol[69]. In fact, cis-MUFA as represented by oleic acid (C18:1 n-9) not only decreases plasma total cholesterol and LDL cholesterol concentrations, but also increases HDL cholesterol in both men and women[62]. Olive oil containing more than 75% oleic acid is an important part of the Mediterranean diet, and is believed to provide protection against CHD among the Southern Europeans. For example, in the Seven Countries Study, Keys *et al*[58] noted that CHD death rates were lowest in cohorts with olive oil as the main dietary fat. Other sources of dietary fat high in cis-MUFA are rapeseed (canola) oil, and high oleic forms of safflower and sunflower oils. Rapeseed oil is the main type of vegetable oil consumed in China and Hong Kong, whilst soybean oil is the main type used in New Zealand, Fiji, Japan, the Republic of Korea and Thailand[70].

In contrast, dietary trans-MUFA are directly related to total and LDL-cholesterol levels and inversely related to HDL-cholesterol[71]. Trans-MUFA are reported to raise LDL-cholesterol when consumed at concentrations exceeding 6% of total energy (which is about the mean level in USA and twice that in Australia)[72]. Another important observation with the consumption of trans fatty acids (mainly elaidic acid) is that they significantly elevate the level of plasma Lp(a), an independent risk factor of CHD[64]. Intake of trans-MUFA range from 2 g/day in Japan to about 10 g/day in the United States and Canada. The dietary significance of these findings is in the increased consumption of trans fatty acids in recent years with increased use of hydrogenated or partially hydrogenated vegetable oils in a wide variety of foods such as margarines, shortenings, spreads, gravies, dressings, icings, pastries, breakfast cereals and baby foods.

In a review of the role of diet in changing CHD in ten countries, Truswell[73] reported that increasing PUFA intake was the most consistent factor related to the decline in CHD mortality. The hypocholesterolaemic effect of PUFA of the n-6 series on total cholesterol and LDL-cholesterol have been consistently established since the 1950s when corn oil (rich in n-6 fatty acid) was found to lower blood cholesterol, particularly when it replaced butter or lard in the diet[74]. However, diets high in PUFA tend to depress HDL-cholesterol[75]. This effect does not benefit women in whom a low HDL concentration poses a greater CHD risk than a high LDL concentration[76]. The parent member of n-6 PUFA is linoleic acid (C18:2) which is commonly found in vegetable seed oils. Dietary sources of n-6 PUFA include soybean oil, sunflower oil and safflower oil besides corn oil.

Alpha-linolenic acid (LNA; C18:3) is the principal PUFA of the n-3 series that is found in the green tissue of plants. A significant content of LNA is found in canola oil from low-erucic rapeseed (11% LNA), soybean oil and wheat germ oil (each

7%)[77]. LNA is present in minimal amounts in animal fats. However, through the use of fish meal, fish oil, flax and canola oil as animal feed, the concentrations of LNA, eicosapentaenoic acid (EPA; C20:5) and docosahexaenoic acid (DHA; 22:6n3) can be increased in poultry, eggs and pork[78]. Photosynthesis in most algae and phytoplankton brings about the production of n-3 PUFA which is eventually passed through the food chain and incorporated into fish lipids[79]. Fatty fish (mackerel, herring, salmon) are a rich source of EPA and DHA. Fish and fish dishes provide 14% of the average daily intake of n-3 fatty acids in the United Kingdom, while the major sources are vegetables (22%), meat (19%) and cereals (17%)[80].

N-3 PUFA are known to lower total cholesterol, LDL and VLDL-cholesterol, triglycerides and to increase HDL-cholesterol[81]. The ingestion of EPA and DHA from fish or fish oils also leads to an antithrombotic effect which includes (1) a decrease in thromboxane A2, a potent platelet aggregator and vasoconstrictor and (2) an increase in prostacyclin PGI3, which together with PGI2, are active vasodilators and inhibitors of platelet aggregation, thereby producing an antithrombotic state[82].

The ratio of PUFA to SFA (P/S) is often considered as a measure of whether a diet is atherogenic, but this indicator is recognised to have some limitations. The major limitation is that it does not take into account the fact that different types of SFA and PUFA have widely different effects on plasma cholesterol. Ulbricht and Southgate[15] formulated new indices of atherogenicity and thrombogenicity which incorporate n-6 and n-3 PUFA and MUFA as an alternative indicator of the P/S ratio. However, Fehily *et al*[83] have provided evidence showing that these indices of atherogenicity and thrombogenicity are weak predictors of CHD risk.

Epidemiology of fat intake

Countries such as the United States, New Zealand and Australia, which have experienced substantial decline in CHD mortality since the mid-1960s, have also undergone a parallel decline in the intake of foods high in saturated fats and cholesterol. Fat intake in the United States declined from a peak of 40-42% of total energy in the late 1950s till mid-1960s to a level of 34% in 1984[84]. Intake of whole milk dropped by 48% between 1963/67 to 1973/85 while the intake of low fat milk rose by 167% during the same period. Similarly, the intake of eggs declined by 18% while that of fish and shellfish increased by 28% over the same period. These changes have brought about an increase in the proportion of polyunsaturated fatty acids consumed.

It is noted that variations exist in the dietary secular trends for different population groups in United States. For example, in the Framingham population for which much dietary data have been generated, a recent study showed that, while their dietary levels of cholesterol declined considerably between 1957/60 and 1984/88, (from 535 mg per day in 1957/60 to 376 mg in 1984/88 for men, and from 491 mg to 254 mg for women), their intakes of macronutrients and fat appeared to have changed only slightly[85]. Fat intake remained at about 38-39% of total energy over the three decades for both men and women in Framingham.

The dietary guidelines advocated by the American Heart Association and the National Cholesterol Education Program prescribes a Step 1 diet that limits total dietary fat to 30% and less by decreasing intake of saturated fatty acids to less than 10% of energy, and dietary cholesterol is restricted to less than 300 mg per day[86].

Based on food availability data from FAO Food Balance Sheets for Asia-Pacific countries, countries with higher CHD mortality rates tend to have higher proportions of dietary energy from fat (Table 8). For example, in New Zealand and Australia, more than 35% of total energy is derived from fat compared to countries like the Philippines and Thailand with levels below 15%. The latter group receives more than three-quarters of their total dietary energy from carbohydrates.

Table 8: proportions of daily dietary energy from fat, carbohydrates and protein in 1986-1988.

	Fat (%)	Carbohydrate (%)	Protein (%)	Total energy (kcals)
New Zealand	37.2	50.2	12.6	3,475
Australia	36.8	51.3	11.9	3,339
Hong Kong	35.5	52.2	12.3	2,883
Japan	25.6	61.7	12.7	2,822
Malaysia	25.6	65.7	8.7	2,665
Singapore	25.3	63.4	11.3	2,882
Fiji	24.5	65.9	9.6	2,783
Brunei Darussalam	23.6	63.3	11.1	2,843
Solomon Islands	22.6	67.4	10.0	2,142
Republic of Korea	17.1	72.0	10.9	2,867
China	15.0	75.3	9.7	2,637
Indonesia	14.5	76.8	8.7	2,631
Philippines	13.3	77.5	9.2	2,235
Thailand	13.1	78.3	8.6	2,288

Food and Agriculture Org. Food Balance Sheets, Rome, 1991. (ref 70)

It is noteworthy that the levels of fat from animal products in New Zealand (114.8 g/day per capita) and Australia (87.8 g/day) rank among the highest in the region, despite having experienced substantial decreases in recent decades (Table 9). The levels of fat from animal products in 1986/88 in these two countries amounted to about 90% of the level in 1972/74. During this period, the level of available fat from animal products increased in most of the Asia Pacific countries. Hong Kong and Singapore have increased to levels exceeding 50-70 g/day per capita of fat from animal products.

The group with the next highest available fat from animal products comprises Japan, Fiji and Brunei Darulssalam with levels between 25-50 g/day. In some Pacific islands including Fiji, their traditional diet, sufficient in fish and micronutrients from a variety of plant sources, has been replaced with a diet with increased animal fat and sugar content[87]. The other countries in the region including Malaysia, Republic of Korea and China have also undergone increases, but their levels of fat from animal products remain at below 25 g/day. Indonesia shows the lowest amount of available fat from animal products at 4.3 g/day.

Table 9. Per capita available fat from animal products among Asia Pacific countries in 1986/88.

	Available fat from animal products (g/day)	Available fat as % of 1972/74 level	Main sources of animal fat (% of fat from animal products)
New Zealand	114.8	90	meat (mutton) (45%) butter (33%)
Australia	87.8	88	meat (bovine) (57%)
Hong Kong	64.7	117	meat (pork) (82%)
Singapore	53.4	120	meat (pork) (74%)
Japan	37.4	126	meat (pork) (32%) fish & seafood (21%)
Fiji	36.9	113	meat (mutton) (30%) butter (45%)
Brunei Darussalam	35.1	186	meat (poultry) (59%)
Malaysia	24.8	120	meat (poultry) (59%)
Rep of Korea	23.3	224	meat (pork) (47%)
China	22.3	197	meat (pork) (80%)
Philippines	15.4	99	meat (pork) (65%)
Thailand	13.3	122	meat (pork) (69%)
Solomon Islands	12.3	93	meat (pork, bovine) (43%) fish & seafood (37%)
Indonesia	4.3	157	meat (poultry) (54%)

Food and Agriculture Org. Food Balance Sheets, Rome, FAO, 1991

Meat is the principal source of animal fat in the region. Although consumption of butter has declined with preference for margarine in several countries[39,84], it remains a major source of animal fat New Zealand, Australia, and Fiji (Table 9). Intake of meat and eggs appears not to have declined in several Asia Pacific countries. Table 10 shows increased availability for meat and eggs per capita per year in the 1970s and 1980s for most of the countries. Despite having experienced decreases in meat consumption from the late 1960s into the 1980s, New Zealand and Australia still rank among those with the highest levels of available meat (above 100 kg per capita per year) in the region. This is followed by countries including Hong Kong, Singapore and Brunei Darussalam with the next highest level of available meat (exceeding 50 kg per capita per year). These countries have shown substantial increases in meat availability in recent decades. The other Asia Pacific countries with lower levels of meat availability (less than 40 kg per year) have also experienced increases in meat availability with the exception of the Philippines and Solomon Islands.

The level of available eggs in most of the Asia Pacific countries has increased from the 1972/74 level (Table 10). Eggs remain an important dietary item for Japan, Hong Kong, Singapore and Brunei Darussalam, where egg availability is among the highest in the region at 10-20 kg per capita per year. Egg availability has declined in New Zealand and Australia most likely due to greater awareness of the cholesterol content.

Table 10. Per capita available meat and eggs among Asia Pacific countries in 1986/88.

	*Meat (kg/ year)	Available meat as % of 1972/74 level	Eggs (kg/ year)	Available eggs as % of 1972/74 level
New Zealand	107.4	92	13.2	78
Australia	104.3	103	10.7	85
Hong Kong	87.8	134	13.3	123
Singapore	75.1	149	11.3	141
Brunei Darussalam	54.8	210	11.8	112
Japan	38.8	164	18.2	113
Malaysia	30.9	159	8.9	129
Fiji	24.1	144	2.8	156
China	23.0	205	5.2	236
Thailand	19.3	126	3.5	109
Rep. of Korea	17.9	289	7.9	184
Philippines	16.1	95	4.3	119
Solomon Islands	10.4	85	0.9	64
Indonesia	6.0	146	2.3	383

* includes beef, mutton, pork and poultry. (Food and Agriculture Organisation Food Balance Sheets, FAO, Rome, 1991)

Countries in the region with a higher prevalence of CHD mortality tend to show higher mean population plasma cholesterol values (Table 11). New Zealand (6.0 mmol/L) shows the highest mean value followed by Thailand, Singapore and Australia. Thailand appears as an exceptional case in showing an unexpectedly high mean plasma cholesterol level (5.9 mmol/L), and a relatively high prevalence of hypercholesterolemia (22%) for both males and females, considering that it is reported to be amongst those with the lowest mortality rate for both CHD and cerebrovascular disease in the region. As pointed out previously, the low mortality figures for some countries may be an artefact due to under-reporting of medically certified death cases. A word of caution is emphasised here in comparing the cholesterol values in Table 11 as they were from different sources which covered different time periods and with varying sample sizes. It is seen that the average population plasma cholesterol concentrations in Japan, Indonesia, Malaysia, Taiwan and urban China do not differ widely (5.0-5.2 mmol/L). Rural China and the Philippines have the lowest average of about 4.5 mmol/L.

The patterns of fat intake and the prevalence of hypercholesterolaemia in some Asia Pacific countries are discussed below in greater detail.

Table 11. Mean serum cholesterol values and prevalence of hypercholesterolemia in Asia Pacific countries.

	Mean serum cholesterol (mmol/L)		Hypercholesterolaemia (%)	
	Male	Female	Male	Female
New Zealand Mann *et al* 1991[88]	6.0	6.1	33.0	30.0
Thailand INCLEN 1992[89]	5.9	-	22	-
Australia NHF-REPS, 1990	5.4	5.3	-	-
NHFA-AIH, 1989[90]	-	-	16.0	14.2
Singapore Hughes *et al* 1990[91]	5.8	5.8	-	-
Japan Okayama *et al* 1993[92]	5.2	5.2	29.4	30.4
Indonesia MONICA-Jakarta,	5.2	5.3	13.4	-
Boedhi-Darmojo, 1993[26]			male &	female
Malaysia UPM/IMR 1992/93[93]	5.0	5.3	12.3	19.8
Taiwan Lyu *et al* 1994[94]	5.0	4.8	-	-
China Folsom *et al* 1994[95]				
Urban	5.0	5.2	-	-
Rural	4.5	4.4	-	-
INCLEN, 1992[89] Shanghai	3.8	-	2.0	-
Philippines INCLEN 1992[89]	4.6	-	7.0	-

Hypercholesterolemia: total serum cholesterol = 6.5 mmol/L or higher

(a) New Zealand

In New Zealand, Jackson and Beaglehole[41] attributed 38-51% of the decline in CHD mortality in men aged 35-64 between 1968-80 to reductions in per capita consumption of saturated fats and dietary cholesterol and to decline in cigarette smoking. Per capita consumption of cholesterol rose from about 650 mg per day in 1955 to 740 mg in the early 1970s, after which it dropped to 615 mg in 1981. This drop is reflects a reduction in the consumption of eggs, beef, mutton, butter and whole milk. Reduction in the consumption of these foods also has led to a similar trend in the intake of saturated fat in New Zealand. Intake of saturated fat peaked in 1963 and then declined by 12% between 1968 and 1980 to a level of about 25 g per head per day. Concomitant with this decline in saturated fat intake has been a rise in polyunsaturated fat consumption derived mainly from vegetable oils which increased by 73% between 1967 and 1978. This has brought about an increase in the polyunsaturated: saturated fat (P/S) ratio from 0.11 in the 1960s to 0.22 in 1980. This value is still low, nonetheless, as compared with the ratios in other Asian countries, and the value recommended by WHO for both industrialised and developing countries (P/S >1.0) (WHO, 1990).

New Zealanders aged 18-24 have a mean cholesterol level which is about 0.5 mmol/L higher than in Britain[88]. Those aged 35-64 years have levels comparable with the North Karelia province in Finland which has one of the highest CHD mortality rates in the world. The high level of mean serum cholesterol persists in New Zealand despite having experienced decreases in the 1970s and 1980s. For example, average serum cholesterol concentration declined by 2.9-4.4% for men aged 35-64 years between 1968-1980[41]. The decline appears to have slowed down as in a more recent study of men and women aged 40-64 years in Auckland, Jackson *et al*[96] reported their mean serum cholesterol level declined by only about 1% between 1982-1987. In terms of the prevalence of hypercholesterolemia (where serum cholesterol is 6.5 mmol/L or higher), New Zealand ranks highest with 33% among males and 30% for females (Table 11).

(b) Australia

Thompson, Hobbs and Martin[39] estimated that dietary changes in fat since 1950 could have contributed to about 30% of Australia's CHD mortality decline. Dietary cholesterol intakes have also decreased by 18% between 1950 and 1985. This was attributed to

the decline in the consumption of meat, eggs and butter by 14%, 38% and 60%, respectively between 1968/69 to 1984/85. During the same period, the consumption of poultry, fish and table margarine increased by 175%, 40% and 347% respectively.

The prevalence of hypercholesterolemia in Australia has been reported for various migrant populations, which show a wide differential in CHD mortality rate. For example, migrants from Western and Eastern Europe have about twice the mortality rates of those from Southern Europe and Southeast Asia[97]. The diets of Southern European migrants remain significantly different with consumption of less dairy fat other than cheese, and more cereals, vegetables and wine[98]. In contrast, Bennett[99] reported that blood lipids, including total cholesterol, HLD and LDL-cholesterol of various immigrant population groups were comparable with the levels of native-born Australians, who were assessed by the 1989 Australian National Heart Foundation (ANF) Survey[90]. This finding suggests that blood lipids play little part in explaining CVD mortality difference among Australian migrant groups. A similar finding was reported by Hsu-Hage and Wahlqvist[100] for Melbourne Chinese and by Wilson *et al*[101] for Greek-Australians, in that the prevalence levels of hypercholesterolemia (serum cholesterol 6.5 mmol/L or higher) in these migrant populations were found to approximate the mean level for the NHF sample aged 25-64 years. Whilst it is generally accepted that CVD is related to a number of risk factors, the relationship is exceedingly complex and is made more so by the additional factors associated with immigration.

(c) Singapore

The trend for available fat in Singapore is reflected in its CHD mortality pattern. As pointed out previously, Singapore ranks closer to Australia than the other countries in South East Asia with regards to CHD mortality rates, but unlike Australia and New Zealand which have experienced substantial declines in CHD mortality commencing in the mid-1960s, Singapore, resembling Hong Kong, commenced its decline about a decade later in the 1970s. Between 1961 to 1980, per capita availability for meat, eggs, milk, and animal oils and fats increased by 135.1%, 79.3%, 35.4% and 72.9% respectively[102]. Given the increased intake of saturated fat in the 1980s, the P/S ratio for adult Singapore Chinese is rather low ranging from 0.3 to 0.5. However, while the P/S ratio of Singapore approaches that of the Framingham adults[85], the dietary cholesterol intake for Singapore remains relatively lower at 131-222 mg per capita per day compared to 415 mg for males and 260 mg for females in Framingham.

The three main ethnic groups in Singapore namely, Chinese, Malays and Indians show differences in risk factor levels. Malays show the highest rate for mean systolic blood pressure and cigarette smoking, while Indians have the lowest mean HDL-cholesterol concentration[91], there was no ethnic difference in mean total cholesterol, LDL-cholesterol as total triglyceride concentration. Chinese have the highest ratio of apo A-I/apo B[103]. It is suggested that the higher level of apo B and lower levels of HDL-cholesterol, apo A-I and apo A-II in Indians contribute towards this group having the highest incidence of CHD among the ethnic groups in Singapore.

(d) Hong Kong

The current relatively low rate of CHD mortality in Hong Kong ranks it in the middle tertile among Asia Pacific countries. However, its dietary pattern points towards future increased CHD incidence as reflected by an upward trend in the apparent consumption of animal products and the proportion of energy from fat (35.5%) approaching the levels in Australia and New Zealand. Moreover, the dietary habits of young children and adolescents may place these children at future risk of diet-related chronic diseases including CHD unless curbed by interventions. Prominent dietary preferences of the younger generation include Westernised fast foods (burgers, pizzas, French fries), soft drinks, bakery goods and dairy products. Given such dietary preferences, Lee *et al*[104] showed that the diet of 12-year old adolescents in Hong Kong is high in fat (28.8% of total energy), saturated fat (above 10% of energy), and cholesterol (mean 481 mg/day) whereas the P/S ratio is low (0.43). A similar finding was reported by Leung *et al*[105] in that, compared with seven-year old children in Southern China, Hong Kong children of the same age consumed 37% more fat daily and showed a higher mean plasma cholesterol level (5.18 mmol/L versus 3.89). Hong Kong children consumed more milk, eggs, French fries, and fast foods than their counterparts in Southern China.

(e) Japan

In Japan too, there are indications that the fat intake is considerably higher among young children. For example, girls aged ten to eleven years were found to be consuming fat amounting to 34% of total energy[106]. This amount approaches the level (37%) which is typical of the intake of the general American population.

The traditional Japanese diet is high in carbohydrates, low in fats and high in seafood contributing to a relatively high P/S ratio. It typically is comprised of boiled rice, fermented soybean paste ("miso") used in soup, fish, salty pickles ("tsukemono"), and non-green-yellow vegetables[107]. However, the Japanese dietary habits have become more Westernised and per capita consumption of meat, eggs, milk and diary products have been on the rise since 1955, albeit more slowly since 1972[43]. The annual National Nutrition Surveys of Japan indicate that carbohydrate intake has been reduced remarkably from 81% of total energy in 1949 to 50% in 1988. Animal protein intake increased by 13.3% between 1960 and 1985, while fat consumption increased by 130% during the same period[108]. Consequently, since the second World War, fat intake in Japan has increased from about 7% of total energy to currently about 25%[92]. The mean MUFA intake in Japan was 20.3 g/day or 39% of total fatty acids, which is less than half the level in United States.

Dietary cholesterol has increased over the years to a level of 363 mg per capita per day in 1989. This level is double that of China and almost three times that of Singapore. Nonetheless, as Japan ranks lowest among the industrialised countries in mortality rates for CHD, it is suggested that this situation could be that "the Japanese people continue to eat rice as the main dish while balancing animal and vegetable foods, reducing salt consumption, eating large amounts of fish and incorporating meat and milk into their meal planning"[109]. They also have a uniquely high intake of green tea, with its anti-oxidant properties[110]

Salt intake in Japan declined steadily from a level of 14.5g per day in 1972 to 12g in 1987[108]. This factor together with the substantial decline in systolic and diastolic blood pressure levels for every age-sex group between the 1960s and 1980s are believed to have contributed to the significant decline in cerebrovascular disease during this period[47]. In comparison, in China, potassium rather than sodium was found to relate more directly with blood pressure[111]. Hatano[27] provided evidence for the decrease in hypertension as a more important risk factor than hyperlipidaemia for CHD decline.

It is noteworthy that Japan, although it has one-fifth the level of CHD mortality rate of New Zealand, has the next highest prevalence of hypercholesterolemia in the region with levels that approach those in New Zealand namely, 29.4% and 30.4% for males and females respectively (Table 12). It is reported that in the past decade or so, serum cholesterol has increased by 1 mg/dL each year[106]. Autopsy data for Japanese males and females under 40 years show evidence of fatty streaks in more than 20% of those aged 10-19 years, and increasingly more complicated lesions were found with increasing age. Goto[106] provided evidence of the association of early development of fatty streaks and fibrous plaques with an increased in myocardial infarction occurring in younger persons.

(f) China

The dietary pattern of China in general is less Westernised compared with several Asia Pacific countries. The Chinese diet is low in total fat, saturated fat and cholesterol with a high P/S ratio, despite recent increases in the consumption of energy. The first Chinese Total Diet Study undertaken in 1990, showed the mean fat intake accounted for 21.2% of total energy which had increased from 15.9% in 1982[112]. This proportion of fat energy is relatively low compared with nearly 40% for countries like New Zealand, Australia and Hong Kong (Table 8). Dietary total fat, SFA, PUFA and cholesterol are higher in the urban than the rural areas[113]. Lee *et al*[104] showed that the intake of MUFA in China in both men (29.1g/day) and women (22.4 g/day) was less than two-thirds that (45.7 and 38.5 g/day respectively) in the United States. The Chinese Total Diet Study[112] reported a similar mean level of MUFA intake level of MUFA at 22.2 g/day which amounted to 44% of total fatty acids.

In terms of per capita total available animal fat, China shows a relatively low level among the Asia Pacific countries (22.3g per day) (Table 9). Urban-rural dietary differences are marked especially in fat intake. The P/S ratio of the Chinese diet remains high at 1.0, which is double that of United States and four times that of New Zealand. The dietary cholesterol contribution also remains low at 173 mg/day, being derived principally from eggs (54.4%) and meat (28.9%). The per capita availability of both eggs and meat, is predominantly pork, and relatively low in the region (Table 9). However, as the Chinese Total Diet indicates, fat intake from animal food is on the increase, having risen from 36.3% or total fat in 1982 to 53% in 1990. This trend may be expected to persist, especially in urban areas, given the current high level of economic development, including the spread of Western dietary influences.

China has a similar level of CHD mortality rate to Japan, but it shows a much lower prevalence of hypercholesterolaemic males and females (1.0% for each case in Table 11), coupled with a low average plasma cholesterol level. Mean values for plasma total cholesterol and LDL-cholesterol for all counties combined were 3.28 and 1.66 mmol/L respectively[114]. Liu *et al*[113] reported mean plasma cholesterol, triglycerides, and LDL-cholesterol concentrations were higher in the urban population compared to the corresponding rural population. The mean total cholesterol levels in both sectors were low (4.7 mmol/L and 4.2 mmol/L in urban and rural males, respectively).

It was suggested in the Multiple Risk Factor Intervention Trial (MRFIT)[115] that low cholesterol may cause intracranial haemorrhage leading to stroke mortality. However, this has been refuted by Chen *et al*[114,116] who reported that there is no strong association between low cholesterol levels and haemorrhagic stroke mortality rates. Additional evidence is provided by Wexun *et al*[117]. The blood lipids of subjects in 65 rural counties who consumed a low-fat and low-protein diet throughout their entire life were presented. It is of interest that no significant correlation between the various cholesterol fractions (LDL, HDL and total cholesterol) and mortality from CHD, cerebrovascular disease and hypertensive heart disease was found. Rather, a significant inverse association between erythrocyte oleate (18:1; n-9) and CVD mortality, especially CHD, was found. Also, rice intake was positively correlated with erythrocyte oleate. The authors suggested a protective role for oleate through the reduction in the synthesis of proaggregatory thromboxanes derived from arachidonate, in a similar manner to the mechanism for the protective effect of n-3 fatty acid-rich oils on platelet aggregation. Thus, CVD mortality in China may be more related to thrombotic tendency than to arterial cholesterol deposition[117].

(g) Taiwan

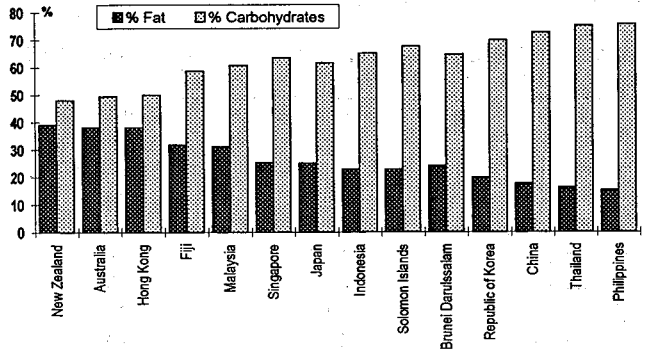
A dietary assessment of Taiwanese subjects showed fat intake by men was 34% of total energy and 37% for women[94]. These levels are comparable with the result obtained from the 1986-88 Taiwan Dietary Survey, which reported an average intake of 36% fat mainly from soybean oil and pork. The mean MUFA intake in Taiwan was 29.9 g/day in men and 23.9 g/day in women or about 36% of total fatty acids for both sexes. This level is about the same as in Japan but lower than that in China.

The daily per capita cholesterol intake for Taiwan of 338 mg for men and 258 mg for women[118] is also higher than the average intake of 179 mg for China. Nonetheless, in Taiwan, the intake of polyunsaturated fat (26 g for men and 21 g for women) is higher than the intake of saturated fat (21 g for men and 17 g for women). This renders the P/S ratio for Taiwan (1.4) even higher than that for Japan and China (1.0).

(h) Malaysia

The dietary pattern in Malaysia is available from food balance sheets for the period between 1961/63 until 1992. Food consumption data are limited to cross-sectional studies involving a small number of subjects. As expected of a rapidly developing country with substantial Western influence on dietary habits, the pattern of energy has shifted towards increasingly more energy from fat sources and concomitantly less from carbohydrates. Percentage of energy from fat increased from 17.9% in 1961/63 to 23.1% in 1982/84 and to 31.1% in 1992[70,119]. Meanwhile, energy from carbohydrates decreased from 74% to 60.6% over the last three decades. Throughout this period, the principal type of fat consumed was from vegetable products, constituting about two-thirds of total fat, while animal products contributed the remaining proportion. The bulk of vegetable fat came from vegetable oils which rose 2.5 times from the 1960s level to 56.9 g per capita per day in 1992. As Malaysia is the leading producer of palm oil in the world, it is not surprising that palm oil is the principal type of vegetable oil used. Nonetheless, since the mid-1980s, the proportion of soybean oil has increased from a mere 5% of total vegetable oils to 22% in 1992.

Figure 3. Proportions of daily dietary available calories from fat and carbohydrates.



As for fat from animal products, the main source is meat, of which the major type is poultry (54%). Whilst total meat available has increased from 14.4 kg per capita per year in the 1960s to 36 kg in 1992, the increase has emerged faster for red meat (beef and pork) with the demand on a per capita basis for poultry about constant over the last decade.

The ethnic differentials in CHD risk factors in Malaysia are similar in many respects to the situation in Singapore as described previously. Indians are consistently reported to show the highest mean prevalence of hypercholesterolemia compared to Chinese and Malays[120,121]. The prevalence of hypertension tends to be higher among Malays[122,123].

In general, Asia Pacific countries with a higher level of energy from fat sources tend to show a concomitant lower level of energy from carbohydrates (Figure 3).

Fish and fish oils

The low death rate from CHD among the Eskimos (Inuit) in Greenland, despite their high fat diet, has been attributed to their high intake of fish[124]. The average per capita fish consumption of the Eskimos is estimated to be about 400 g per day[125]. An inverse relation between consumption of fish and CHD mortality has been found in some prospective studies in the Netherlands[126] and the United States[127], but not in other studies in Norway[128] and the United States[129,130]. The latter study from 1986-1992 included more than 51,000 male health professionals aged 40-75 years. This landmark study found that, after controlling for age and other coronary risk factors, there was no significant associations between dietary intake of n-3 fatty acids or fish intake and the risk of CHD. One explanation suggested for the negative finding in the latter three studies is that the subjects were already taking high levels of fish, and that any beneficial effect is obtained with one or two servings of fish per week and higher levels may not be better[131].

Fish oil supplements have been found to lower blood pressure in normotensive or hypertensive subjects. Subjects eating a fish meal a day (4.8g n-3 fatty acids) in a 30% energy fat diet were found to be most likely to show a fall in blood pressure as well as improving blood lipid profile and platelet function[132]. However, one would have to consume a large amount of fatty fish (almost 200g per day) in order to obtain that high level of n-3 fatty acids.

The low incidence of myocardial infarction in Japan has been attributed to its high intake of fish and other seafood, which amount to about 100 g per day per capita[92]. This amount of fish consumed provides one-third of the total daily intake of n-3 PUFA. Based on the thrombogenicity and atherogenicity indices of Ulbright and Southgate[15], which take into account the ratio of n-3 PUFA, n-6 PUFA and MUFA content, the Japanese diet is computed to have a thrombogenicity index of 0.4 and atherogenicity index of 0.4. These levels resemble closely the indices for Greenland Eskimo which are 0.39 and 0.28 respectively. In contrast, the United Kingdom, whose CHD mortality rate in 1985 was about eight times higher than that for Japan[14], has thrombogenicity and atherogenicity indices which are relatively higher at 0.93 and 1.21 respectively.

Table 12. Per capita available fish in among Asia Pacific countries in 1986/88.

	Fish & seafood (kg/year)	Available fish & seafood as % of 1972/74 level
Japan	73.2	98
Republic of Korea	70.3	182
Solomon Islands	57.8	105
Hong Kong	47.9	101
Fiji	44.8	184
New Zealand	41.1	259
Brunei Darussalam	40.8	155
Singapore	39.1	81
Malaysia	33.3	120
Philippines	32.5	96
Thailand	22.0	101
Australia	15.8	112
Indonesia	13.9	146
China	8.2	155

Food and Agriculture Org Food Balance Sheets, FAO, Rome, 1991

Besides Japan, the Republic of Korea also has a high fish intake. According to availability data, the Republic of Korea had 70.3kg of fish and seafood per person per year in 1986/88 compared to 73.2kg in Japan. Intake of fresh and processed fish was 44g per capita per day in 1984, having increased five-fold from the level in 1969[133]. Countries with the next highest level (40-70 kg per year) of available fish and seafood include the Solomon Islands, Hong Kong, Fiji New Zealand and Brunei Darussalam (Table 12). China, Indonesia and Australia rank among those with the lowest available fish on a per capita basis.

Japan is the largest single producer of fish oils and fats, averaging 29% of total world production from 1988 to 1990[134]. As a whole, Asia's production averaged 32% and South America was about 30% over the same period. Extensive variations in the EPA and DHA content in fish oils prevail among interspecies and intraspecies. For example, the EPA content of fish oils may range from 20-100 mg/g fatty acids in herring oil to 170-250 mg/g fatty acids for anchovy[79]. Besides that, geographical and seasonal variations also lead to wide variations in fatty acid composition in fish oils. Seasonal variations may lead to a 75% decrease in the EPA content.

Increasingly, fish and plant oil mixtures are being sold as health foods in the form of encapsulated products. For example, such products containing a combined EPA and DHA concentration of 300 mg/g are commonly available. Fish oils, notably fish liver oils, are also sources of vitamins A, D and E but there are substantial inter- and intra-species variations.

Vitamin antioxidants

Three important naturally occurring vitamin antioxidants discussed here are vitamin E, β-carotene and ascorbic acid.

(i) Vitamin E

The beneficial cardiovascular effects of fish and fish oils are mediated through their EPA and DHA to control aggregatory and inflammatory responses. High levels of long-chain PUFA incorporated into cellular membranes potentiate their peroxidisability and increase the requirement for antioxidants[135]. Vitamin E is a major dietary antioxidant which minimises free radical tissue damage either by stopping the initial chain reaction or by reacting with lipid peroxyl radicals. In this manner, membrane PUFA is protected from lipid peroxidation. Some epidemiologic studies have shown an inverse association between vitamin E levels and incidence of CHD[136-138].

It is recognised that a high intake of PUFA increases the requirement for vitamin E. Thus, increasing the intake of fish or fish oil supplement without adequate antioxidant protection may result in *in vivo* peroxidation of n-3 PUFA, thereby reducing its cardiovascular benefits. It has been proposed that a minimal amount of 0.6 mg of vitamin E in the form of d-α-tocopherol for every gram of PUFA is required to prevent vitamin E depletion; but further studies are called for to determine vitamin E adequacy when n-3 PUFA intake is increased[139].

The dietary patterns reported for some Asia Pacific countries with a high intake of PUFA appear to attain the recommended minimal vitamin E/PUFA ratio of 0.6. In Japan, where fish intake is high, Okayama *et al*[92] estimated the total PUFA consumed amounted to 15.3g per day per capita and the vitamin E intake was 8.3mg, thus giving a ratio of approximately 0.5 of vitamin E to PUFA. In China, fish intake is lower but total PUFA is about the same as in Japan (16.8g in males and 14.3g in females), being derived mainly from soybean products[104], with a daily intake of vitamin E estimated at 8.9mg giving a vitamin E/PUFA ratio of 0.624. Likewise, Lee *et al*[140] reported that the diet in Singapore provides a ratio of 0.7 mg vitamin E per gram of PUFA. The main sources of vitamin E in these countries are likely to include nuts, peanut butter, oil, vinegar, margarines, sweet potatoes, cabbage, tuna and eggs.

The role of antioxidants in atherosclerosis remains to be elucidated, as some studies have reported no association between antioxidant levels and vascular mortality[141,142]. Hense *et al*[143] also reported a lack of any substantial association between serum vitamin E and acute myocardial infarction during three years of follow up of the MONICA Augsburg cohort. The authors postulated that the high mean vitamin E levels in the study subjects could be providing close to maximum antioxidant protection, and hence no positive association.

(ii) β-carotene

Several epidemiological investigations have reported findings of an inverse relationship between β-carotene intake and CVD[144]. The mechanism for a protective effect of β-carotene is not completely understood. As an antioxidant, it may reduce free radical-induced oxidation of LDL-cholesterol[145]. β-carotene may increase HDL through its conversion to retinoids, which can stimulate synthesis of apolipoprotein A-I, a major component of HDL[146]. Some believe that a protective effect of β-carotene is evident only in subjects with low concentrations of both HDL-cholesterol and β-carotene, as in the case of cigarette smokers[147]. In a study on the effect of β-carotene supplementation (50 mg/day for one year) on lipoproteins in smokers, van Poppel *et al*[148] reported a lack of effect of β-carotene on HDL-cholesterol, total cholesterol, apo A-I and apo B-100 concentrations. They concluded that a protective effect of β-carotene would not appear to be mediated through changes in the concentrations of plasma lipoproteins in healthy male smokers.

The principal dietary sources of β-carotene are vegetables such as carrots, sweet potatoes, yellow squash, spinach, cantaloupe and broccoli. The consumption of fresh fruits and vegetables was inversely related to the risk of CHD in several ecological studies as reported by Graziano and Hennekens[149]. The intake of vegetables and fruits is high in many Asia Pacific countries. According to the availability data in 1986/88, the Republic of Korea, New Zealand, Singapore, Australia and Hong Kong rank amongst the highest with apparent intakes of over 400g of vegetables and fruits per capita per day. They are followed by Thailand, Malaysia, China and Brunei Darulssalam (200-400g). Countries with less 200g intake include Solomon Islands, Fiji, the Philippines and Indonesia. Consistent with this, the study of Lee *et al*[140] showed intake of β-carotene in Singapore to be 5-9 times greater than that in China[112]. Consumption of β-carotene in the United States is also higher than in China by about 1.5 times[104].

(iii) Ascorbic acid

Ascorbic acid acts as an important biological reducing agent in several situations. It is involved in collagen synthesis and increased capillary fragility is one sign of ascorbic acid deficiency. Ascorbic acid is also known to decrease platelet aggregation and increase fibrinolytic activity.

Ascorbic acid is believed to be the major antioxidant for regenerating vitamin E from tocopheryl chromanyl radicals. As vitamin E occurs in very small amounts in membranes, it must be recycled with efficiency in order to meet the demands of the potential lipid peroxidation turnover[55]. Ascorbic acid protects vitamin E against oxidation. The vitamin C: vitamin E ratio may be more important biologically than absolute ascorbate levels. Therefore, fruits and vegetables rich in ascorbic acid should be encouraged when a diet containing large quantities of fatty fish is consumed.

In the NHANES-I Study, cardiovascular mortality rate was 34% lower than expected among participants with the highest ascorbic acid intake. However, other studies which controlled for other vitamin supplement use, reveal no significant correlation between ascorbic acid and CHD incidence. There appears to be a need for extra ascorbic acid by four population groups namely, smokers, diabetics, hypertensives and the elderly[150].

Excessive intake of certain vitamins such as A, K and C may be associated with oxidative stress. For example, in vitro experiments reveal that high vitamin C may promote oxidative reactions by enhancing the release of transition metals from protein complexes, thus promoting the generation of hydroxyl radicals[147]. However, *in vivo* evidence of the pro-oxidative role of vitamin C is lacking, unless through increased iron storage.

4.4. Dietary fibre

Higher intake of vegetables especially legumes, leads to a higher intake of fibre. Several studies by Anderson and his coworkers[151] indicate that dietary fibre can significantly reduce serum total cholesterol and LDL-cholesterol and improve the ratio of HDL-cholesterol/LDL-cholesterol. Fibre-rich oat bran and bean products have been the focus of studies on the hypo-cholesterolaemic effects of fibres[152]. Oat bran is rich in oat gum which is believed to be the major cholesterol-lowering component in oat products. There are some hypotheses to explain the hypocholesterolaemic effects of soluble fibres such as oat and bean products. Soluble fibre may exert its effect through changes in bile acid absorption, hepatic production of lipoproteins or peripheral clearance of lipoproteins[153].

In addition to its hypocholesterolaemic effects, high fibre diets also promote weight loss, improve glycaemic control, reduce fasting serum triglycerides and may lower blood pressure[154]. The consumption of legumes, which are characterised by a high content of water-soluble fibre, has been shown to reduce glucose intolerance[155].

Intake of total fibre in China may be high, about three times that in United States that is, 33 g/day compared to 11 g/day respectively[156]. Higher intakes of plant products in China are also reflected in mean ascorbic acid and retinol equivalent intakes twice those in the United States.

4.5. Soybeans

The soybean has been used for centuries as food in East Asia and Southeast Asia. It is consumed in various forms (as different types of bean curd, soy sauce, soy milk, and fermented soy products). The protein content in soybeans is well recognised as an important source of good quality protein in meeting the essential amino acid and protein needs of both children and adults[157]. In addition, the role of soy foods in protecting against chronic diseases such as cancer, heart disease, kidney disease and osteoporosis is the focus of many studies.

Much research has been undertaken on the reduction of plasma cholesterol with soy protein[158]. Diets with soy protein substituted for animal protein have been found to have the ability to lower total plasma cholesterol, LDL-cholesterol, VLDL-cholesterol and total triglycerides levels in hypercholesterolaemic children, men and women[159,160]. The intakes of isolated soy protein needed to produce an hypercholesterolaemic effect is estimated at 25-50g daily. The mechanism for the cholesterol-lowering effect of soy remains to be fully elucidated. The resultant cholesterol reduction brought about by soy occurs in the LDL fraction, without any adverse effect on HDL[158]. The component of soybean/protein responsible also remains to be established. Investigations centre on the amino acid content, rate of protein digestibility, interactions between dietary protein, minerals and associated PUFA and hormonal factors like soy isoflavones[161].

Among Asia Pacific countries, Japan ranks highest in intake of soybean according to food availability data[70] with 9.6 kg/year per capita in 1984/86 followed by Indonesia (7.2 kg/year), the Republic of Korea (6.4 kg/year), China (3.1 kg/year) and Thailand (2.0 kg/year). Based on a quantitative food frequency questionnaire, Lee *et al*[140] estimated the median daily consumption of total soy products in Singapore as 36g. In this study, the group with breast cancer was found to consume a significantly lower mean level of soy products than the control group. Another health implication of soybeans was reported by Guo *et al*[30] in that intake of legumes, particularly soy, was negatively correlated with cerebrovascular disease mortality rates in men and women in China.

4.6. Alcohol consumption

In an extensive review of the association between alcohol consumption and CHD risk, Moore and Pearson[162] provided evidence of an inverse relationship between moderate alcohol consumption and CHD mortality. The relationship is U shaped indicating increased risk for both abstainers and heavy drinkers. Two drinks a day was believed to be protective against CHD[163].

The data from MRFIT Study indicated that in men, the protective effect may be mediated through an increase in the HDL-cholesterol[164]. Result from the Bogalusa Heart Study, which was monitored from 1981 to 1991, showed an association between alcohol consumption and HDL-cholesterol in the latest survey, but the authors suggested that this finding could reflect the aging of the cohort[165]. Other studies have attributed the beneficial effects of moderate consumption of alcohol to an antioxidant role of other constituents of the beverage as in red wine[166].

Inter-country comparison of intakes of wine, beer and spirits (absolute alcohol) and how theses relate to CHD mortality decline in industrialised countries, by Epstein[167] revealed no relationship of alcohol consumption with change in CHD mortality. However, different kinds of alcoholic beverages may have different effects on drinkers, depending on personality, gender or pharmaco-kinetics.

In Japan, several epidemiological studies have reported on alcohol consumption as a risk factor for hypertension and stroke[168]. Alcohol intake is related to blood pressure levels of both rural and urban populations, independent of relative weight, serum lipid levels, smoking rate, uric acid, and haemoglobin concentrations[169]. Most alcohol is consumed by men, mostly in the 40-49 years age group, and the rates of those who drink every day are 47% for men and 3% for women[170]. Alcohol consumption has been on an upward trend, for example, it increased from less than 4 litres/adult/year in mid-1950 to about 9 litres/adult/year in mid-1980. Japanese men in Japan consume more alcohol (28g/day) than Japanese men living in Hawaii (13g/day) and in California (9g/day)[171]. Ueshima[108] attributed the low CHD mortality in Japan in part to the drinking habit of the Japanese. In comparing alcohol intakes in men and women from Taiwan and Framingham, Lyu *et al*[94], the higher alcohol intake by Framingham men (4.8% versus 2.1% of total energy for Taiwan men) showed strong positive associations with HDL-cholesterol and apo A-I, and a negative association with apo B concentrations.

Chinese men and women in rural areas of China tend to consume more alcohol than their counterparts in urban areas[95]. Compared to Chinese in United States, Chinese in China reported higher consumption of alcoholic beverages (combined beer, wine and hard liquor), but lower than that consumed by Whites[104]. The average number of drinks per week among Chinese men and women in China was 5.5. and 1.3 respectively, whereas those of US white men and women were 7.3 and 2.3 respectively.

In Australia, alcohol consumption at the rate of 1 to 17 drinks per week was found more commonly among male immigrants from the United Kingdom, Italy, Western and Eastern Europe[99], than those from Asia. Female immigrants from the United Kingdom showed the highest drinking frequency. The National Health and Medical Research Council of Australia recommends fewer than 28 drinks per week for men, and fewer than 14 drinks per week for women as responsible drinking behaviour[172]. It is estimated that the deaths from heart attack in Australia that are prevented by alcohol are offset to a significant extent by the deaths caused by liver cirrhosis and other diseases arising from long term alcohol drinking[87], and will also be, in part, from stroke.

Despite increasing the risk of hypertension, alcohol consumption of up to 3-4 drinks a day may reduce the risk of CHD, but at high levels of consumption, the incidence of haemorrhagic stroke and heart failure increases[173]. Any recommendation regarding increasing alcohol consumption is an unlikely public health activity because of important social and medical consequences of alcohol intoxication and chronic drinking[98].

4.7 Iron overload

Sullivan[174] was among the earliest to suggest that iron depletion is able to provide protection against CHD. Knottnerus *et al*[175] in the Netherlands and others elsewhere have shown that the incidence of myocardial infarction increased directly with serum ferritin level. A ferritin level of 200μg/L or more compared to normal levels of 100-150μg/L, doubled the risk of CHD. It is postulated that high levels of iron increase the oxidation of LDL-cholesterol leading to atherosclerosis, as well as initiating cancer. However, several studies have failed to establish such a clear relationship. Oshaug *et al*[176] observed a consistent association between serum ferritin and recognised risk factors of CHD, the strongest being indicators which are related to body fat distribution (BMI and WHR). They suggest that the observed association between serum ferritin and CHD may be an effect of obesity.

Rao and White[35] commented on the resemblance of the knobby structure on the red cell membrane made by the mature malarial parasite to one of the platelet receptors. This may favour platelet interaction with such red cells leading to clot formation in microvessels or facilitate the attachment of these cells on the endothelial surface. The authors observed that the lysis of the red cells may serve as a source of iron containing proteins needed for oxidation of lipoproteins in the vicinity of the vessel wall.

The issue of iron overload being deleterious to health has important policy implications for developing countries, in relations to their iron fortification efforts as a means of ameliorating the "old" problem of iron deficiency. Iron deficiency anaemia persists as a major public health problem in many developing countries in the region including Malaysia, Indonesia, Thailand and the Micronesian Islands.

5. CVD risk factors related to diet

5.1. Obesity

Obesity is associated with several diseases and metabolic disorders such as heart disease, diabetes mellitus, hypertension, gall bladder disease and some types of cancer. Obese subjects have increased VLDL-triglyceride synthesis, increased production and clearance rates of LDL apo B and LDL-cholesterol, and decreased plasma apo A-I and HDL-cholesterol concentrations. Excess energy intake coupled with lack of physical exercise depresses HDL concentrations. Knuiman *et al*[177] reported an average decrease of 0.020 mmol/L of HDL-cholesterol for each unit increase in BMI.

Fat distribution rather than overall fatness has become an important focus of obesity research. An accumulation of intra-peritoneal fat, that is, fat stored in the abdominal cavity as mesenteric and omental fat, is believed to be a stronger indicator of CVD and other metabolic complications of obesity than overall fatness as measured by BMI. A predominance of abdominal visceral fat or upper body fat (android obesity) is related to the development of non-insulin diabetes (NIDDM), hypertension and CHD[178]. Men are more susceptible to upper fat accumulation whereas women tend to have greater accumulation of fat in the gluteo-femoral (hip-thigh) region (gynoid obesity).

There are direct and indirect methods for assessment of abdominal visceral fat, ranging from computed tomography to anthropometric measurements, as reviewed by van der Kooy and Seidell[179]. Anthropometric measurements, being easy to perform and relatively inexpensive, lend themselves readily to epidemiological studies and clinical screening purposes. In more recent studies, the waist-to-hip ratio (WHR) has been used as a measure of fat distribution, as it has been found to correlate highly with the intra-abdominal visceral fat mass, after adjusting for the effects of age and degree of overweight[180]. Increasing WHR is also accompanied by increasing fasting plasma glucose and insulin levels[181]. In addition, increasing WHR leads to an increase in fasting plasma triglyceride and a decrease in HDL-cholesterol.

Increasingly, more studies have reported on the importance of abdominal fat as a significant cardiovascular risk. The visceral fat mass correlates more significantly with fasting and postglucose challenge plasma insulin levels, fasting plasma triglycerides, and blood pressure than total body fat mass. In Melbourne Chinese, the relatively high WHR (0.90 for men and 0.83 for women) better

predicts the seemingly high prevalence of hypercholesterolemia than does the low overall prevalence of overweight[100].

A comparison of BMI among the Asia Pacific countries shows that New Zealand and Australia have the highest mean BMI values for both men and women (Table 13). The mean BMI for men in both countries lies between 25-26 kg/m^2 whilst that for women is between 24-25 kg/m^2. Taiwan has the next highest mean value for BMI at 24.2 kg/m^2 for men and 22.6 kg/m^2 for women. Other countries in the region have BMI mean values between 20-24 kg/m^2. Meanwhile the prevalence of overweight (BMI > 25.0 kg/m^2) is highest in New Zealand (55% for men and 38% for women) and in Australia (50% for men and 34.9% for women). The prevalence of overweight in the other countries, with the exception of China, ranged from 19% in Thailand to 24% in the Philippines.

China has a relatively low prevalence of overweight ranging from 7.5% in rural areas to 12% in urban areas; albeit a slight increase in the prevalence of overweight in recent years has been recorded[182]. A low mean BMI among Chinese in China is confirmed by data from the PRC-USA Cooperative Research Programme on the epidemiology of cardiovascular and cardiopulmonary diseases and their risk factors. The mean BMI of the cohorts examined in 1987/88 ranged from 20.1 to 21.9 kg/m^2 in men and women from rural and urban areas in China, while their mean WHR was 0.84 and 0.80 respectively[95]. Controlling for age and BMI, the WHR showed a significant negative correlation with fasting HDL-cholesterol (both sexes), and a positive correlation with serum triglycerides (both sexes), and total and LDL-cholesterol (men). In another major study on CVD risk factors in China, He *et al*[183] reported that even in a lean population with a low mean blood pressure, BMI correlated positively with both systolic and diastolic pressure, after adjustment for age, smoking, alcohol intake and physical activity.

In the International Clinical Epidemiology Network (INCLEN) study involving men aged 35-65 years in 12 centres in seven countries (China, Thailand, the Philippines, Indonesia, Chile, Colombia and Brazil), BMI was found to be strongly correlated with plasma cholesterol and blood pressure levels in almost all population groups[89]. In a study of a multicultural workforce in New Zealand, BMI and smoking were found to correlate positively with triglycerides, total and LDL-cholesterol levels, and inversely with HDL-cholesterol[184].

In several Asia Pacific countries with a relatively low prevalence of overweight among adults, there appears to be a trend towards increasing prevalence of overweight and obesity amongst the children (Table 14). In Thailand, the prevalence of obesity (>120% weight for height of Bangkok reference) in urban children aged 6-12 years rose from 12.2% in 1991 to 13.5% in 1992 and to 15.6% in 1993[185]. Yap and Tan[186] revealed that the prevalence of obesity (≥ 120% median weight for height of Singapore Ministry of Health reference) for school age boys in Singapore increased from 9% in 1984 to 14.5% in 1989, whilst in girls, it rose from 8% to 10.4% over the same period. The authors found no significant difference in the total daily intake of energy, nor in the level of mean energy expenditure between the obese and control groups for both boys and girls. Obesity tends to run in the families of the obese cases in this study. Singaporean Chinese youths, on average, spent more time in sedentary activities than American Chinese counterparts[187]. In Taiwan, the prevalence of overweight among boys and girls aged ten years increased from 8.4% and 4.6% respectively in 1980/82 to 14.4% and 14.1% in 1986/88 according to BMI which exceeds 120% the mean weight for age of Taiwan reference[188]. Yamashiro[189] estimated the prevalence of obese school children in Japan at 5-8%.

Table 13. Mean body mass index and prevalence of overweight and hypertension in Asia Pacific countries.

	Mean BMI (kg/m^2)		Overweight (%)		Hypertension (%)	
	Male	Female	Male	Female	Male	Female
New Zealand Mann *et al* 1991[88]	25.8	24.8	55.0	38.0	23.2	14.6
Australia NHF-RFPS, 1989	25.4	24.1	50.0	34.9	19.8	15.6
Taiwan Lyu *et al*, 1994[94,118]	24.2	22.6	-	-	22 male& female	
Malaysia UPM/IMR 1992/93[93]	23.1	24.4	22.3	30.8	6.3	9.0
Japan Okayama *et al*, 1993[92] (*Kikkawa *et al*, 1992)[200]	23.0	22.6	23.0	22.6	27.7*	22.5*
Philippines INCLEN, 1992[89]	23.0	-	24.0	-	-	-
Indonesia MONICA-Jakarta Boedhi-Darmojo, 1993	22.6	23.6	22.6	23.6	13.6	16.0
Singapore Hughes *et al* 1990[91]	22.5	23.5	22-23	22-25	15-23	11-18
Thailand INCLEN, 1992[89]	22.2	-	19.0	-	21.0	-
China Folsom *et al.*, 1994[95] urban	20.7	21.9	-	-	9.0	11.0
Rural	20.3	20.2	-	-	12.0	6.0
Ge *et al*, 1994[190] urban	-	-	12.0 male & female		-	-
Rural	-	-	7.5 male & female		-	-

Hypertension: Systolic blood pressure ≥ 160 mm Hg or diastolic bp ≥ 95 and/or receiving treatment for hypertension; Overweight: BMI ≥ 25.0

Table 14. Prevalence of obesity in children and adolescents in Asia Pacific countries

Country	Age (years)	Percent obese Boys	Girls	Criteria	Reference
Australia		5-15	8-19		Darnton-Hill, de Boer & Nair, 1995
Singapore	6-16	14.5	10.4	>120% wt/ht S'pore ref	Yap & Tan, 1994
Thailand	6-12	15.6 (boys & girls)		>120% wt ht Bangkok ref	Mo-suwan, Junjana & Puetpaiboon, 1993
Taiwan	10.0	14.4	14.1	>120% wt/age Taiwan ref	Kao *et al.*, 1991
Philippines	3-6	7.1 (boys & girls)		Philippines ref	Darnton-Hill, de Boer & Nair, 1995
Japan	6.0	2.9	3.3	>120 wt/ht Japan ref	Darnton-Hill, de Boer & Nair, 1995
	14.0	7.2	6.9		
Malaysia	>1-6	1.3	0.7	>+2sd wt/ht NCHS ref	UPM/IMR, 1992/93
	>6-12	2.4	3.0		
Pacific Islands					Darnton-Hill, de Boer & Nair, 1995
Kiribati	school children	11.1 (boys & girls)		>+2sd wt/ht NCHs ref	
Tonga	3-6	23.4 (boys & girls)		>120% wt/ht NCHS ref	

In Tonga, 23% of children under five years were reported to be obese in the 1986 nutrition survey, while in Australia, about 30% of children are overweight, with 5-15% of boys and 8-19% of girls considered obese[191]. These authors also reported an increase in the prevalence of obese children in the Philippines which still has a marked degree of undernutrition. The prevalence of overweight preschool children was below 5% in the 1980s but by 1992, it had increased to 7.1%.

Obesity in children is a matter of public health concern as it is known to be associated with "adult degenerative diseases", in themselves usually CHD risk factors, including hypertension, hypercholesterolemia, fatty liver, and NIDDM[192]. Obesity in children predisposes to these chronic disorders later in life, with the risk of premature death and excess morbidity.

5.2 Diabetes mellitus

Diabetes may be classified clinically into four types namely, insulin dependent (IDDM or Type I) diabetes, non-insulin dependent (NIDDM or Type II) diabetes, gestational diabetes (GDM), and other types of diabetes associated with other diseases that damage the pancreas or produce insulin resistance. NIDDM diabetes is by far the most common form of diabetes accounting for 90% of cases worldwide[193]. NIDDM contributes to cardiac, cerebral and peripheral vascular disease, besides blindness (retinopathy) and end stage renal failure (nephropathy)[194].

The prevalence of NIDDM varies from a low of 0.7% in rural Papua New Guinea[195] to a high of 41% in the Nauruans, while the highest in the world is probably among the Pima/Papago Indians in USA (50%)[196]. The prevalence of diabetes in China is low ranging from 0.15% in Guizhou to 1.15% in Ningxia as reported by Cheah, Wang and Sum[197]. The prevalence of NIDDM in China is estimated as 1.6%103. Diabetes is reported to be more common in urban than rural areas.

The urban-rural differential in diabetic prevalence is evident in many countries. For example, among Malays in Malaysia, the prevalence of NIDDM in a remote village was reported as 2.8%, and it is higher in a rural village (6.7%), while a village in an urban location had the highest prevalence of 8.2%[198]. Similarly, the prevalence of NIDDM differs in rural Fiji (1.1%) and in the urban areas (5.3%)[199].

Epidemiologic studies undertaken in Japan in the past decade show an increase in the proportion of the population with NIDDM[200]. Approximately 10% of the population aged 40 years and above is estimated to have NIDDM. Hypertension is closely linked with NIDDM in Japan and it leads to complications such as cardiac failure, nephropathy and stroke. The prevalence of hypertension remains high in Japan despite a decline in both systolic and diastolic pressure since the 1950s[170]. However, CHD is not a common complication in Japanese NIDDM patients, unlike other ethnic populations with similar manifestations of hypertension, and lipid abnormalities[200].

The prevalence of diabetes in adults in New Zealand was reported as 2.8% in Europeans, 4.6% in Pacific Islanders and 6.95% in Maori[201]. Ethnic differences also prevail for risk factors such as hypertension and body mass index[202].

Migrant Indian populations show a relatively higher prevalence of NIDDM than Indians in India. Indians in India have a NIDDM prevalence of 2.4% in rural areas and a level of 8.2% in urban areas[203]. In comparison, the prevalence for Indians in Singapore and Malaysia are 12.8%[194] and 16% respectively[123]. Indians elsewhere also have high prevalences of diabetes, (10-37% in Cape Town, 5.7% in Fiji and 16.4% in Surinam[197]).

In comparing the mortality rates for diabetes among countries in Southeast Asia, Singapore shows the highest levels for both men and women at 6.6 and 9.8 per 100,000 respectively[19]. The prevalence of NIDDM among Chinese in Singapore aged 18-69 years is high at 8%[194]. Brunei Darulssalam and the Philippines appear to have low mortality rates for diabetes, under 5.5 per 100,000 for men and under about 6.5 for women.

While NIDDM is characterised by a genetic predisposition, environmental and lifestyle factors are also strongly linked to its development. Factors implicated in the increase of NIDDM with urbanisation include decreased physical activity, increased stress and increased intake of sugar and fat, leading to increased prevalence of overweight and obesity.

Both body fat mass and abdominal fat distribution are major risk factors for NIDDM in prospective epidemiological studies. In a study on Malaysian Malays, the prevalence of diabetes was 4.5 times higher in obese men than non-obese, and in women, it was 6 times higher in obese as in non-obese[198]. Among Japanese Americans, the intra-abdominal fat area from a computed tomography scan was found to be a main predictor of diabetes incidence[204]. It is postulated that increased insulin resistance in NIDDM may be associated with elevated levels of androgens (in the case of women) or cortisols, or with increased concentrations of free fatty acids[205]. Both types of hormones may directly deposit fat in the abdominal regions through mechanisms that prevent storage in the periphery, or through direct actions on abdominal adipose tissue. Androgens and free fatty acids may inhibit hepatic uptake of insulin leading to peripheral hyperinsulinaemia. In a study on Japanese women, WHR was shown to correlate with fasting insulin and androgenicity (sex hormone binding globulin)[206].

Diet plays a central role in the control of diabetes. Carbohydrates are well known to exert a strong influence on glucose tolerance, and epidemiological surveys have attested to the inverse relationship between carbohydrate consumption and prevalence of diabetes[207]. However, too high a carbohydrate level in the diet (such as 65% of total energy intake) has been shown to have deleterious effects on glycaemic control in NIDDM patients compared with higher-fat low-carbohydrate diets[208]. Evidence seems conflicting with regard to the detrimental effect of simple sugars on diabetes, and more studies are needed to clarify the role of sugar as a risk factor of diabetes[209]. However, small additions of sugar to meals or snacks have no detectable effect on glycaemic response in people with diabetes. The glycaemic response of starchy foods is known to depend on its fibre content, especially the water-soluble fibre[210]. The mechanisms for the beneficial effect of soluble fibres on glucose metabolism suggested include the delay of gastric emptying and effects on gastrointestinal hormone secretion.

Besides carbohydrates and fibre, dietary fat is also recognised for its ability to affect tissue insulin sensitivity and hepatic glucose output. Saturated fatty acids and dietary cholesterol were shown to relate positively to fasting and postprandial glucose levels in normoglycaemic men, independent of energy intake and body fatness[211]. PUFA have been found to have beneficial effects on glucose metabolism. N-3 fatty acids are believed to exert their influence through their known potential to decrease VLDL triglyceride synthesis[212]. In addition, it has been suggested that the incorporation of PUFA in cellular membranes affects the fluidity of the membranes, which leads to an enhancement in the activity of insulin receptors and glucose transport, and increase in tissue insulin sensitivity.

Apart from dietary factors, physical activity has a favourable effect on glycaemic response in NIDDM. Two major prospective studies involving large numbers of subjects in Boston showed that the incidence of NIDDM was inversely related to the frequency of vigorous exercise ("enough to work up a sweat") in both men[213] and women[214]. The inverse relation of exercise to risk of NIDDM was particularly patent among overweight subjects. Helmrich *et al*[215] computed that the occurrence of NIDDM in middle-aged men was reduced by 6% for every increment of 500 kcal per week in leisure-time physical activity. Regular exercise may protect against the development of NIDDM by maintaining a preferred lean-to-fat ratio with respect to body mass. Adipose tissue is the major site for insulin insensitivity which is reduced by physical activity.

6. Conclusions

In reviewing the trends in CVD mortality and its dietary implications as prevailing in the Asia Pacific region, several salient aspects have emerged. Some dietary factors such as saturated fatty acids and dietary cholesterol have been consistently shown to exert detrimental atherogenic and/or thrombotic effects on CHD development. Gurr[57] succinctly summarised the state of art with respect to the influence of dietary lipids on plasma cholesterol as follows:

(i) there is a positive association between plasma total or LDL-cholesterol and risk of CHD in younger men;
(ii) for the population in general, there is no similar association between plasma total cholesterol and total CHD mortality;
(iii) there is an inverse association between HDL-cholesterol and CHD risk;
(iv) there is no relationship between dietary lipids and either plasma lipids or CHD mortality;
(v) although permissive plasma cholesterol may not be the predominant known risk factor when compared with smoking, hypertension and aspects of energy balance. Increasing prevalence of NIDDM may alter this risk hierarchy further function

Each step in the pathway from coronary risk factor to morbid or lethal event may have its own nutritional determinants[110]. The prevalence of smoking is high, especially among men, in several Asia Pacific countries. It is estimated that the percent of smokers among men exceed 55% in Indonesia[89], the Philippines[32], and Japan[92], while in China[95] and Thailand[89], the proportion of men who smokes exceeds 65%. An ethnic differential in smoking is seen in Singapore where cigarette smoking was highest among Malays (53.3%) followed by Indians (44.5%) and Chinese (37.4%)80. Smoking was the single most prevalent risk factor among CVD patients in Malaysia, with over 70% of the patients being smokers[216].

Cigarette smoking is well established as a major CHD risk factor through its increase in LDL-cholesterol and VLDL-cholesterol, and decrease in HDL-cholesterol[217], amongst other effects. The biochemical mechanisms by which smoking influences lipoprotein metabolism are not completely elucidated. One postulation is that nicotine increases catecholamine levels leading to a mobilisation of free fatty acids from adipocytes and resulting in increased hepatic triglyceride synthesis and VLDL secretion. Even modest cigarette smoking adversely alters serum lipid and lipoprotein levels in the young.

Among the various CVD risk factors, cigarette smoking is probably the most amenable to amelioration. For example in Australia, the prevalence of smoking has dropped significantly from over 40% in 1980 in males to under 25% in 1990[90]. Likewise for females, smoking prevalence has decreased from about 30% to 20% over the same period. This has come about through a concerted effort by the mass media, public, politicians and professionals including doctors[124].

While some epidemiological studies such as the Honolulu Heart Program revealed clear relations between risk factors, including reductions in systolic blood pressure, cigarette smoking, intake of cholesterol and animal protein, and increase in vegetable protein, with trends of declining rates of CHD[218], *apparent discrepancies* in these relationships remain as exemplified by Blackburn[219]:

(1) a sustained decline in CHD mortality in the face of increased body mass and obesity in Australia[13] and in United States[52];
(2) in Japan, deaths from CHD and stroke are decreasing paralleled by decline in mean blood pressure; however, there remains an extensive prevalence of hypertension and tobacco smoking, while the mean plasma total cholesterol (which presently is below 5.0 mmol/L) tends to rise;
(3) in Finland, the levelling off of the fall in CHD mortality rate appears to correlate with a levelling off of risk factor reduction, There are no further declines in dietary fat intake or smoking[220];
(4) in the Framingham cohort, no change in CHD incidence in men despite progressively lower levels of smoking, blood pressure and total cholesterol found in that cohort.
(5) a number of studies have noted that conventional risk factors such cholesterol and/or smoking do not significantly explain risk gradients for CHD for Chinese, either in China[221], or as migrants in London[222] or for the Japanese[30,223].
(6) coronary deaths have declined in US women despite an increase in cigarette smoking.

Thus, better *models of risk factors/ disease relationships* are needed, based on reliable data. The above examples also serve to reiterate the limitations of risk factor research in population health. The risk factor research tradition has its roots in infectious disease epidemiology and tends to focus on specific risks associated with specific diseases[224]. Over the years, there have been a proliferation of risk factors of CVD identified. Many of these risk factors are signs or symptoms of the disease processes rather than causal agents, while others are likely to be spurious correlations or proxy measures of complex interactions that may or may not have a contributory influence[225]. Epidemiological association in general relates risk factors to the end-points of CVD As pointed out by Gurr[57], since these end-points have a multifactorial basis, one to one relationships between epidemiologically identified factors and outcomes cannot be expected to be a universal finding.

Another conflicting area is fat distribution measured anthropometrically. Fat distribution and body shape appear to vary among ethnic groups. Wang *et al*[226] showed that, although Asians have a lower BMI, they are fatter with a higher upper-body subcutaneous fat than whites, especially in women. In a study on migrant groups in Australia. Bennet[99] reported that women from Asia tend to have a lower BMI but higher WHR compared to women from Western Europe who have higher WHR but not higher BMI. The WHR of the Melbourne Chinese better predicts other CVD risk factors present such as hypertension and hypercholesterolemia than does their mean BMI[100]. The Melbourne Chinese have a bigger mean waist circumference and WHR but smaller hip circumference than Chinese in Southern China. In comparison, Malay women in Malaysia show a lower prevalence of overweight according to WHR than by BMI. This is attributed to their apparently bigger hip circumference and thus smaller WHR, especially in women aged 30-50 years[93]. While many studies report the WHR to be a close proxy indicator for upper body fat , others such as Wing *et al*[227] and van der Kooy *et al*[179] find that WHR may not be independently related to changes in CVD risk factors, including changes in visceral fat.

In relation to *food composition* data, there is a need for more detailed analyses to be undertaken on the nutrient content of local/indigenous foods like products of locally bred animals, indigenous plant foods, foods prepared by indigenous methods, and ready-to-eat foods of different cultures. In light of the importance of certain nutrients with respect to CVD for example, PUFA especially n-3 and n-6 fatty acids, MUFA, SFA, cholesterol, fibre including water-soluble fibre, α-linoleic acid and vitamin E, it is imperative for such nutrients to be included in every country's food composition database. There is also a major need for the establishment of reliable surveillance systems of eating habits in representative samples of the populations.

In this review of populations of widely diverse socio-economic background, gaps in the availability of reliable data render comparisons difficult. Statistics related to incidences, prevalences and mortality rates for CHD and stroke are not uniformly available for all Asia Pacific countries. The *data* may be in country or institution reports or journals published in the vernacular language only. The office of the World Health Organization for the Western Pacific based in Manila is

recommended to serve as the focal point or depository for all health-related publications on the Asia Pacific region. Likewise, the Food and Agriculture Organisation Regional Office for Asia and the Pacific based in Bangkok could serve as the resource centre for all food and nutrition-related publications written on the region.

In conclusion, as CVD remains the leading cause of death in Asia Pacific countries, there is a need for a more coordinated effort to reduce the incidence of the major risk factors of CVD, provided we understand and model them well. In the past, such efforts appear to have brought about more benefits to those with higher income, education and occupation[228]. The onset of decline of CHD in the United States was 2-10 times more likely to occur in communities with the highest socio-economic attributes. More advantaged communities have greater resources for the promotion of more healthy lifestyles, and they are better able to make choices and take advantage of improvements in medical care. Behavioural modifications take time but concerted efforts by all, the government, mass media, food manufacturers, health professionals and the public, are needed for the health benefits to reach all, regardless of socio-economic and cultural background.

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营养与心血管疾病: 亚太地区的展望

摘要

亞太地區國家膳食模式的改變 被認爲與心血管疾病死亡率有關. 目前該地區的許多國家, 心血管疾病已是死亡率的主要原因. 1960年代中期, 澳大利亞, 紐西蘭和日本等國家冠心病(CHD) 的死亡率已開始下降, 但是新加坡及香港卻遲了將近20年. 相反, 馬來西亞和中國冠心病的死亡率還繼續上升. 雖然自1970年后臺灣和日本的中風死亡率已明顯下降, 但是在中國, 臺灣和日本, 腦血管病仍然是死亡的主因.

陸地動物油, 魚油和植物油, 根據其脂肪酸含量和其它成分的不同, 對動脈硬化和血栓形成有不同的影響. 冠心病死亡率較高的國家, 大都攝取較高脂肪和部分動物脂肪爲能量的來源. 在許多國家, 進食脂肪過多也許會增加高膽固醇血症和超重. 再者, 該地區內進食其它食物如黃豆, 在水果, 青菜, 種子, 谷類, 干果和茶葉中的抗氧化劑和少量酒對心臟有保護作用.

亞太地區人群膳食廣泛多樣, 從不同社會文化背景, 不同經濟和技術水平, 可提出 各種的研究方式. 未來的研究應考慮發病機理和干預試驗的研究.

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