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Research Article

Diet, Nutrition and The Prevention of Chronic Diseases

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ABSTRACT

To briefly review the current understanding of the etiology and prevention of chronic diseases using a life course approach, demonstrating the lifelong influences on the development of disease. A computer search of the relevant literature was used Medline-'life cycle' and 'nutrition' and reviewing the articles for relevance in addressing the above objective. Articles from references dated before 1990 were followed up separately. A subsequent search using Clio updated the search and extended it by using 'life cycle', 'nutrition' and 'noncommunicable disease' (NCD), and 'life course'. Several published and unpublished WHO reports were key in developing the background and arguments.

Key words: Diet, The Prevention Of Chronic Diseases

INTRODUCTION

A Joint WHO/FAO Expert Consultation on Diet, Nutrition and the Prevention of Chronic Diseases met in Geneva from 28 January to 1 February 2002. The meeting was opened by Dr D. Yach, Executive Director, Noncommunicable Diseases and Mental Health, WHO, on behalf of the Directors-General of the Food and Agriculture Organization of the United Nations and the World Health Organization. The Consultation followed up the work of a WHO Study Group on Diet, Nutrition and Prevention of Noncommunicable Diseases, which had met in 1989 to make recommendations regarding the prevention of chronic diseases and the reduction of their impact (1). The Consultation recognized that the growing epidemic of chronic disease afflicting both developed and developing countries was related to dietary and lifestyle changes and undertook the task of reviewing the considerable scientific progress that has been made in different areas. For example, there is better epidemiological evidence for determining certain risk factors, and the results of a number of new controlled clinical trials are now available. The mechanisms of the chronic disease process are clearer, and interventions have been demonstrated to reduce risk. During the past decade, rapid expansion in a number of relevant scientific fields and, in particular, in the amount of population-based epidemiological evidence has helped to clarify the role of diet in preventing and controlling morbidity and premature mortality resulting from noncommunicable diseases (NCDs). Some of the specific dietary

components that increase the probability of occurrence of these diseases in individuals, and interventions to modify their impact, have also been identified. Furthermore, rapid changes in diets and lifestyles that have occurred with industrialization, urbanization, economic development and market globalization, have accelerated over the past decade. This is having a significant impact on the health and nutritional status of populations, particularly in developing countries and in countries in transition. While standards of living have improved, food availability has expanded and become more diversified, and access to services has increased, there have also been significant negative consequences in terms of inappropriate dietary patterns, decreased physical activities and increased tobacco use, and a corresponding increase in diet-related chronic diseases, especially among poor people. Food and food products have become commodities produced and traded in a market that has expanded from an essentially local base to an increasingly global one. Changes in the world food economy are reflected in shifting dietary patterns, for example, increased consumption of energy-dense diets high in fat, particularly saturated fat, and low in unrefined carbohydrates. These patterns are combined with a decline in energy expenditure that is associated with a sedentary lifestyle --- motorized transport, labour saving devices in the home, the phasing out of physically demanding manual tasks in the workplace, and leisure time that is preponderantly devoted to physically undemanding pastimes. Because of these changes in dietary and lifestyle patterns, chronic NCDs--- including obesity, diabetes mellitus, cardiovascular disease (CVD), hypertension and stroke, and some types of cancer --- are becoming increasingly significant causes of disability and premature death in both developing and newly developed countries, placing additional burdens on already overtaxed national health budgets. The Consultation provided an opportune moment for FAO and WHO to draw on the latest scientific evidence available and to update recommendations for action to governments, international agencies and concerned partners in the public and private sectors. The overall aim of these recommendations is to implement more effective and sustainable policies and strategies to deal with the increasing public health challenges related to diet and health. The Consultation articulated a new platform, not just of dietary and nutrient targets, but of a concept of the human organism's subtle and complex relationship to its environment in relation to chronic diseases. The discussions took into account ecological, societal and behavioural aspects beyond causative mechanisms. The experts looked at diet within the context of the macroeconomic implications of public health recommendations on agriculture, and the global supply and demand for foodstuffs, both fresh and processed. The role of diet in defining the expression of genetic susceptibility to NCDs, the need for responsible and creative partnerships with both traditional and non-traditional partners, And the importance of addressing the whole life course, were all recognized. Nutrition is coming to the fore as a major modifiable determinant of chronic disease, with scientific evidence increasingly supporting the view that alterations in diet have strong effects, both positive and negative, on health throughout life. Most importantly, dietary adjustments may not only influence present health, but may determine whether or not an individual will develop such diseases as cancer, cardiovascular disease and diabetes much later in life. However, these concepts have not led to a change in policies or in practice. In many developing countries, food policies remain focused only on under nutrition and are not addressing the preventation of chronic disease.

Although the primary purpose of the Consultation was to examine and develop recommendations for diet and nutrition in the prevention of chronic diseases, the need for sufficient physical activity was also discussed

and is therefore emphasized in the report. This emphasis is consistent with the trend to consider physical activity alongside the complex of diet, nutrition and health. Some relevant aspects include: Energy expenditure through physical activity is an important part of the energy balance equation that determines body weight. A decrease in energy expenditure through decreased physical activity is likely to be one of the major factors contributing to the global epidemic of overweight and obesity. Physical activity has great influence on body composition --- on the amount of fat, muscle and bone tissue. To a large extent, physical activity and nutrients share the same metabolic pathways and can interact in various ways that influence the risk and pathogenesis of several chronic diseases. Cardiovascular fitness and physical activity have been shown to reduce significantly the effects of overweight and obesity on health.

Physical activity and food intake are both specific and mutually interacting behaviours that are and can be influenced partly by the same measures and policies. Lack of physical activity is already a global health hazard and is a prevalent and rapidly increasing problem in both developed and developing countries, particularly among poor people in large cities. In order to achieve the best results in preventing chronic diseases, the strategies and policies that are applied must fully recognize the essential role of diet, nutrition and physical activity. This report calls for a shift in the conceptual framework for developing strategies for action, placing nutrition --- together with the other principal risk factors for chronic disease, namely, tobacco use and alcohol consumption --- at the forefront of public health policies and programmes.

1. Diet, nutrition and chronic diseases in context

The diets people eat, in all their cultural variety, define to a large extent people's health, growth and development. Risk behaviours, such as tobacco use and physical inactivity, modify the result for better or worse. All this takes place in a social, cultural, political and economic environment that can aggravate the health of populations unless active measures are taken to make the environment a health-promoting one.

Although this report has taken a disease approach for convenience, the Expert Consultation was mindful in all its discussions that diet, nutrition and physical activity do not take place in a vacuum. Since the publication of the earlier report in 1990 (1), there have been great advances in basic research, considerable expansion of knowledge, and much community and international experience in the prevention and control of chronic diseases. At the same time, the human genome has been mapped and must now enter any discussion of chronic disease.

Concurrently there has been a return to the concept of the basic life course, i.e. of the continuity of human lives from fetus to old age. The influences in the womb work differently from later influences, but clearly have a strong effect on the subsequent manifestation of chronic disease. The known risk factors are now recognized as being amenable to alleviation throughout life, even into old age. The continuity of the life course is seen in the way that both undernutrition and overnutrition (as well as a host of other factors) play a role in the development of chronic disease. The effects of man-made and natural environments (and the interaction between the two) on the development of chronic diseases are increasingly recognized. Such factors are also being recognized as happening further and further ''upstream'' in the chain of events predisposing humans to chronic disease. All these broadening perceptions not only give a clearer picture of what is happening in the current epidemic of chronic diseases, but also present many opportunities to address them. The identities of those affected are now better recognized: those most disadvantaged in more affluent countries, and --- in numerical terms far greater --- the populations of the developing and transitional worlds.There is a continuity in the influences contributing to chronic disease

development, and thus also to the opportunities for prevention. These influences include the life course; the microscopic environment of the gene to macroscopic urban and rural environments; the impact of social and political events in one sphere affecting the health and diet of populations far distant; and the way in which already stretched agriculture and oceanic systems will affect the choices available and the recommendations that can be made. For chronic diseases, risks occur at all ages; conversely, all ages are part of the continuum of opportunities for their prevention and control. Both undernutrition and overnutrition are negative influences in terms of disease development, and possibly a combination is even worse; consequently the developing world needs additional targeting. Those with least power need different preventive approaches from the more affluent. Work has to start with the individual risk factors, but, critically, attempts at prevention and health promotion must also take account of the wider social, political and economic environment. Economics, industry, consumer groups and advertising all must be included in the prevention equation.

1.2. Diet, nutrition and the prevention of chronic diseases through the life course

The rapidly increasing burden of chronic diseases is a key determinant of global public health. Already 79% of deaths attributable to chronic diseases are occurring in developing countries, predominantly in middle-aged men(2). There is increasing evidence that chronic disease risks begin in fetal life and continue into old age (3--9). Adult chronic disease, therefore, reflects cumulative differential lifetime exposures to damaging physical and social environments.

For these reasons a life-course approach that captures both the cumulative risk and the many opportunities to intervene that this affords, was adopted by the Expert Consultation. While accepting the imperceptible progression from one life stage to the next, five stages were identified for convenience. These are: fetal development and the maternal environment; infancy; childhood and adolescence; adulthood; and ageing and older people.

2. Fetal development and the maternal environment

The four relevant factors in fetal life are:

(i) intrauterine growth retardation (IUGR);
(ii) premature delivery of a normal growth for gestational age fetus;
(iii) overnutrition in utero; and
(iv) intergenerational factors.

There is considerable evidence, mostly from developed countries, that IUGR is associated with an increased risk of coronary heart disease, stroke, diabetes and raised blood pressure. It may rather be the pattern of growth, i.e. restricted fetal growth followed by very rapid postnatal catch-up growth, that is important in the underlying disease pathways. On the other hand, large size at birth (macrosomia) is also associated with an increased risk of diabetes and cardiovascular disease. Among the adult population in India, an association was found between impaired glucose tolerance and high ponderal index (i.e.fatness) at birth. In Pima Indians, a U-shaped relationship to birthweight was found, whereas no such relationship was found amongst Mexican Americans. Higher birth weight has also been related to an increased risk of breast and other cancers. In sum, the evidence suggests that optimal birth weight and length distribution should be considered, not only in terms of immediate morbidity and mortality but also in regard to long-term outcomes such as susceptibility to diet-related chronic disease later in life.

3. Infancy

Retarded growth in infancy can be a reflected in a failure to gain weight and a failure to gain height. Both retarded growth and excessive weight or height gain (‘‘crossing the centiles’’) can be factors in later incidence of chronic disease. An association between low growth in early infancy (low weight at 1 year) and an increased risk of coronary heart disease (CHD) has been

described, irrespective of size at birth. Blood pressure has been found to be highest in those with retarded fetal growth and greater weight gain in infancy. Short stature, a reflection of socioeconomic deprivation in childhood, is also associated with an increased risk of CHD and stroke, and to some extent, diabetes. The risk of stroke, and also of cancer mortality at several sites, including breast, uterus and colon, is increased if shorter children display an accelerated growth in height.

3.1. Breastfeeding

There is increasing evidence that among term and pre-term infants, breastfeeding is associated with significantly lower blood pressure levels in childhood. Consumption of formula instead of breast milk in infancy has also been shown to increase diastolic and mean arterial blood pressure in later life. Nevertheless, studies with older cohorts and the Dutch study of famine have not identified such associations. There is increasingly strong evidence suggesting that a lower risk of developing obesity may be directly related to length of exclusive breastfeeding although it may not become evident until later in childhood. Some of the discrepancy may be explained by socioeconomic and maternal education factors confounding the findings. Data from most, but not all, observational studies of term infants have generally suggested adverse effects of formula consumption on the other risk factors for cardiovascular disease (as well as blood pressure), but little information to support this finding is available from controlled clinical trials. Nevertheless, the weight of current evidence indicates adverse effects of formula milk on cardiovascular disease risk factors; this is consistent with the observations of increased mortality among older adults who were fed formula as infants. The risk of several chronic diseases of childhood and adolescence (e.g. type 1 diabetes, coeliac disease, some childhood cancers, inflammatory bowel disease) have also been associated with infant feeding on breast-milk substitutes and short-term breastfeeding. There has been great interest in the possible effect of high-cholesterol feeding in early life. Reiser et al. proposed the hypothesis that high-cholesterol feeding in early life may serve to regulate cholesterol and lipoprotein metabolism in later life. Animal data in support of this hypothesis are limited, but the idea of a possible metabolic imprinting served to trigger several retrospective and prospective studies in which cholesterol and lipoprotein metabolism in infants fed human milk were compared with those fed formula. Studies in suckling rats have suggested that the presence of cholesterol in the early diet may serve to define a metabolic pattern for lipoproteins and plasma cholesterol that could be of benefit later in life. The study by Mott, Lewis & McGill on differential diets in infant baboons, however, provided evidence to the contrary in terms of benefit. Nevertheless, the observation of modified responses of adult cholesterol production rates, bile cholesterol saturation indices, and bile acid turnover, depending on whether the baboons were fed breastmilk or formula, served to attract further interest. It was noted that increased atherosclerotic lesions associated with increased levels of plasma total cholesterol were related to increased dietary cholesterol in early life. No long-term human morbidity and mortality data supporting this notion have been reported. Short-term human studies have been in part confounded by diversity in solid food weaning regimens, as well as by the varied composition of fatty acid components of the early diet. The latter are now known to have an impact on circulating lipoprotein cholesterol species. Mean plasma total cholesterol by age 4 months in infants fed breast milk reached 180 mg/dl or greater, while cholesterol values in infants fed formula tended to remain under 150 mg/dl. In a study by Carlson, DeVoe & Barness , infants receiving predominantly a linoleic acid- enriched oil blend exhibited a mean cholesterol concentration of approximately 110 mg/dl. A separate group of infants in that study who

received predominantly oleic acid had a mean cholesterol concentration of 133 mg/dl. Moreover, infants who were fed breast milk and oleic acid-enriched formula had higher high-density lipoprotein (HDL) cholesterol and apoproteins A-I and A-II than the predominantly linoleic acid-enriched oil diet group. The ratio of low- density lipoprotein (LDL) cholesterol plus very low-density lipoprotein (VLDL) cholesterol to HDL cholesterol was lowest for infant receiving the formula in which oleic acid was predominant. Using a similar oleic acid predominant formula, Darmady, Fosbrooke & Lloyd reported. a mean value of 149 mg/dl at age 4 months, compared with 196 mg/dl in a parallel breast-fed group. Most of those infants then received an uncontrolled mixed diet and cow's milk, with no evident differences in plasma cholesterol levels by 12 months, independent of the type of early feeding they had received. A more recent controlled study suggests that the specific fatty acid intake plays a predominant role in determining total and LDL cholesterol. The significance of high dietary cholesterol associated with exclusive human milk feeding during the first 4 months of life has no demonstrated adverse effect. Measurements of serum lipoprotein concentrations and LDL receptor activity in infants suggests that it is the fatty acid content rather than the cholesterol in the diet which regulates cholesterol homeostasis. The regulation of endogenous cholesterol synthesis in infants appears to be regulated in a similar manner to that of adults.

4. Childhood and adolescence

An association between low growth in childhood and an increased risk of CHD has been described, irrespective of size at birth. Although based only on developed country research at this point, this finding gives credence to the importance that is currently attached to the role of

immediate postnatal factors in shaping disease risk. Growth rates in infants in Bangladesh, most of whom had chronic intrauterine under-nourishment and were breastfed, were similar to growth rates of breastfed infants in industrialized countries, but catch-up growth was limited and weight at 12 months was largely a function of weight at birth.

In a study of 11--12 year-old Jamaican children, blood pressure levels were found to be highest in those with retarded fetal growth and greater weight gain between the ages of 7 and 11 years. Similar results were found in India. Low birth weight Indian babies have been described as having a characteristic poor muscle but high fat preservation, so-called ''thin-fat'' babies. This phenotype persists throughout the postnatal period and is associated with an increased central adiposity in childhood that is linked to the highest risk of raised blood pressure and disease. In most studies, the association between low birth weight and high blood pressure has been found to be particularly strong if adjusted to current body size --- body mass index (BMI) --- suggesting the importance of weight gain after birth.

Relative weight in adulthood and weight gain have been found to be associated with increased risk of cancer of the breast, colon, rectum, prostate and other sites. Whether there is an independent effect of childhood weight is difficult to determine, as childhood overweight is usually continued into adulthood. Relative weight in adolescence was significantly associated with colon cancer in one retrospective cohort study. Frankel, Gunnel & Peters, in the follow-up to an earlier survey by Boyd Orr in the late 1930s, found that for both sexes, after accounting for the confounding effects of social class, there was a significant positive relationship between childhood energy intake and adult cancer mortality. The recent review by the International Agency for Research on Cancer (IARC) in Lyon, France, concluded that there was clear evidence of a relationship between onset of obesity (both early and later) and cancer risk.

Short stature (including measures of childhood leg length), a reflection of socioeconomic deprivation in childhood, is associated with an increased risk of CHD and

stroke, and to some extent diabetes. Given that short stature, and specifically short leglength, are particularly
sensitive indicators of early socioeconomic deprivation, their association with later disease very likely reflects an association between early undernutrition and infectious disease load. Height serves partly as an indicator of socioeconomic and nutritional status in childhood. As has been seen, poor fetal development and poor growth during childhood have been associated with increased cardio- vascular disease risk in adulthood, as have indicators of unfavourable social circumstances in childhood. Conversely, a high calorie intake in childhood may be related to an increased risk of cancer in later life. Height is inversely associated with mortality among men and women from all causes, including coronary heart disease, stroke and respiratory disease. Height has also been used as a proxy for usual childhood energy intake, which is particularly related to body mass and the child's level of activity. However, it is clearly an imperfect proxy because when protein intake is adequate (energy appears to be important in this regard only in the first 3 months of life), genetics will define adult height. Protein, particularly animal protein, has been shown to have a selective effect in promoting height growth. It has been suggested that childhood obesity is related to excess protein intake and, of course, overweight or obese children tend to be in the upper percentiles for height. Height has been shown to be related to cancer mortality at several sites, including breast, uterus and colon. The risk of stroke is increased by accelerated growth in height during childhood. As accelerated growth has been
linked to development of hypertension in adult life, this may be the mechanism (plus an association with low socioeconomic status). There is a higher prevalence of raised blood pressure not only in adults of low socioeconomic status, but also in children from low socioeconomic backgrounds, although the latter is not always associated with higher blood pressure later in life. Blood pressure has been found to track from childhood to predict hypertension in adulthood, but with stronger tracking seen in older ages of childhood and in adolescence. Higher blood pressure in childhood (in combination with other risk factors) causes target organ and anatomical changes that are associated with cardiovascular risk, including reduction in artery elasticity, increased ventricular size and mass, haemodynamic increase in cardiac output and peripheral resistance. High blood pressure in children is strongly associated with obesity, in particular central obesity, and clusters and trackswith an adverse serum lipid profile (especially LDL cholesterol) and glucose intolerance. There may be some ethnic differences, although these often seem to be explained by differences in body mass index. A retrospective mortality follow-up of a survey of family diet and health in the United Kingdom (1937--1939) identified significant associations between childhood energy intake and mortality from cancer. The presence and tracking of high blood pressure in children and adolescents occurs against a background of unhealthy lifestyles, including excessive intakes of total and saturated fats, cholesterol and salt, inadequate intakes of potassium, and reduced physical activity, often accompanied by high levels of television viewing. In adolescents, habitual alcohol and tobacco use contributes to raised blood pressure.

There are three critical aspects of adolescence that have an impact on chronic diseases:

(i) the development of risk factors during this period;

(ii) the tracking of risk factors throughout life; and, in terms of prevention,

(iii) the development of healthy or unhealthy habits that tend to stay throughout life, for example physical inactivity because of television viewing. In older children and adolescents, habitual alcohol and tobacco use contribute to raised blood pressure and the development

of other risk factors in early life, most of which track into adulthood.

The clustering of risk factor variables occurs as early as childhood and adolescence, and is associated with atherosclerosis in young adulthood and thus risk of later cardiovascular disease. This clustering has been described as the metabolic --- or ‘‘syndrome X’’ --- clustering of physiological disturbances associated with insulin resistance, including hyperinsulinaemia, impaired glucose tolerance, hypertension, elevated plasma triglyceride and low HDL cholesterol. Raised serum cholesterol both in middle age and in early life are known to be associated with an increased risk of disease later on. The Johns Hopkins Precursor Study showed that serum cholesterol levels in adolescents and young white males were strongly related to subsequent risk of cardiovascular disease mortality and morbidity. Although the risk of obesity does not apparently increase in adults who were overweight at 1 and 3 years old, the risk rises steadily thereafter, regardless of parental weight. Tracking has also been reported in China, where overweight children were 2.8 times as likely to become overweight adolescents; conversely, underweight children were 3.6 times as likely to remain underweight as adolescents. The study found that parental obesity and underweight, and the child’s initial body mass index, dietary fat intake and family income helped predict tracking and changes. However, in a prospective cohort study conducted in the United Kingdom, little tracking from childhood overweight to adulthood obesity was found when using a measure of fatness (percentage body fat for age) that was independent of build. The authors also found that only children obese at 13 years of age had an increased risk of obesity as adults, and that there was no excess adult health risk from childhood or adolescent overweight. Interestingly, they found that in the thinnest children, the more obese they became as adults, the greater was their subsequent risk of developing chronic diseases.

The real concern about these early manifestations of chronic disease, besides the fact that they are occurring earlier and earlier, is that once they have developed they tend to track in that individual throughout life. On the more positive side, there is evidence that they can be corrected. Overweight and obesity are, however, notoriously difficult to correct after becoming established, and there is an established risk of overweight during childhood persisting into adolescence and adulthood. Recent analyses have shown that the later the weight gain in childhood and adolescence, the greater the persistence. More than 60% of overweight children have at least one additional risk factor for cardiovascular disease, such as raised blood pressure, hyperlipidaemia or hyperinsulinaemia, and more than 20% have two or more risk factors.

Habits leading to noncommunicable disease development during adolescence

It seems increasingly likely that there are widespread effects of early diet on later body composition, physiology and cognition. Such observations ‘‘provide strong support for the recent shift away from defining nutritional needs for prevention of acute deficiency symptoms towards long-term prevention of morbidity and mortality’’.

Increased birth weight increases the risk of obesity later, but children with low birth weight tend to remain small into adulthood. In industrialized countries there have been only modest increases in birth weight so the increased levels of obesity described earlier must reflect environmental changes.

5. <u>Adulthood</u>

The three critical questions relating to adulthood were identified as:

(i) to what extent do risk factors continue to be important in the development of chronic diseases;

(ii) to what extent will modifying such risk factors make a difference to the emergence of disease; and

(iii) what is the role of risk factor reduction and modification in secondary prevention and the treatment of those with disease?

Reviewing the evidence within the framework of a life-course approach highlights the importance of the adult phase of life, it being both the period during which most chronic diseases are expressed, as well as a critical time for the preventive reduction of risk factors and for increasing effective treatment.

The most firmly established associations between cardiovascular disease or diabetes and factors in the lifespan are the ones between those diseases and the major known ‘‘adult’’ risk factors, such as tobacco use, obesity, physical inactivity, cholesterol, high blood pressure and alcohol consumption. The factors that have been confirmed to lead to an increased risk of CHD, stroke and diabetes are: high blood pressure for CHD or stroke; high cholesterol (diet) for CHD, and tobacco use for CHD. Other associations are robust and consistent, although they have not necessarily been shown to be reversible: obesity and physical inactivity for CHD, diabetes and stroke; and heavy or binge drinking for CHD and stroke. Most of the studies are from developed countries, but supporting evidence from developing countries is beginning to emerge, for example, from India. In developed countries, low socioeconomic status is associated with higher risk of cardiovascular disease and diabetes. As in the affluent industrialized countries, there appears to be an initial preponderance of cardiovascular disease among the higher socioeconomic groups, for example, as has been found in China. It is presumed that the disease will progressively shift to the more disadvantaged sectors of society. There is some evidence that this is already happening, especially among women in low-income groups, for example in Brazil and South Africa, as well as in countries in economic transition such as Marocco. Other risk factors are continually being recognized or proposed. These include the role of high levels of homocysteine, the related factor of low folate, and the role of iron. From a social sciences perspective, Losier has suggested that socioeconomic level is less important than a certain stability in the physical and social environment. In other words, an individual’s sense of understanding of his or her environment, coupled with control over the course and setting of his or her own life appears to be the most important determinant of health. Marmot, among others, has demonstrated the impact of the wider environment and societal and individual stress on the development of chronic disease.

6. Ageing and older people

There are three critical aspects relating to chronic diseases in the later part of the life-cycle: (i) most chronic diseases will be manifested in this later stage of life;

(ii) there is an absolute benefit for ageing individuals and populations in changing risk factors and adopting health-promoting behaviours such as exercise and healthy diets; and (iii) the need to maximize health by avoiding or delaying preventable disability. Along with the societal and disease transitions, there has been a major demographic shift. Although older people are currently defined as those aged 60 years and above, this definition of older people has a very different meaning from the middle of the last century, when 60 years of age and above often exceeded the average life expectancy, especially in industrialized countries. It is worth remembering, however, that the majority of elderly people will, in fact, be living in the developing world.

Most chronic diseases are present at this period of life --- the result of interactions between multiple disease processes as well as more general losses in physiological functions Cardiovascular disease peaks at this period, as does type 2 diabetes and some cancers. The main burden of chronic diseases is observed at this stage of life and, therefore, needs to be addressed.

6.1. Changing behaviours in older people

In the 1970s, it was thought that risks were not significantly increased after certain late ages and

that there would be no benefit in changing habits, such as dietary habits, after 80 years old as there was no epidemiological evidence that changing habits would affect mortality or even health conditions among older people. There was also a feeling that people ‘‘earned’’ some unhealthy behaviours simply because of reaching ‘‘old age’’. Then there was a more active intervention phase, when older people were encouraged to change their diets in ways that were probably overly rigorous for the expected benefit. More recently, older people have been encouraged to eat a healthy diet --- as large and as varied as possible while maintaining their weight --- and particularly to continue exercise. Liu et al. have reported an observed risk of
atherosclerotic disease among older women that was approximately 30% less in women who ate 5--10 servings of fruits and vegetables per day than in those who ate 2--5 servings per day. It seems that, as elderly patients have a higher cardiovascular risk, they are more likely to gain
from risk factor modification.

Although this age group has received relatively little attention as regards primary prevention, the acceleration in decline caused by external factors is generally believed to be reversible at any age. Interventions aimed at supporting the individual and promoting healthier environments will often lead to increased independence in older age.

Results and Recommendations for preventing cardiovascular disease

The second half of the 20th century has witnessed major shifts in the pattern of disease, in addition to marked improvements in life expectancy, this period is characterized by profound changes in diet and lifestyles which in turn have contributed to an epidemic of noncommunicable diseases. This epidemic is now emerging, and even accelerating, in most
developing countries, while infections and nutritional deficiencies are receding as leading contributors to death and disability.

In developing countries, the effect of the nutrition transition and the concomitant rise in the prevalence of cardiovascular diseases will be to widen the mismatch between health care needs and resources, and already scarce resources will be stretched ever more thinly. Because
unbalanced diets, obesity and physical inactivity all contribute to heart disease, addressing these, along with tobacco use, can help to stem the epidemic. A large measure of success in this area has already been demonstrated in many industrialized countries.

Cardiovascular diseases are the major contributor to the global burden of disease among the noncommunicable diseases. WHO currently attributes one-third of all global deaths (15. million) to CVD, with developing countries, low-income and middle-income countries accounting for 86% of the DALYs lost to CVD worldwide in 1998. In the next two decades the increasing burden of CVD will be borne mostly by developing countries.

The relationship between dietary fats and CVD, especially coronary heart disease, has been extensively investigated, with strong and consistent associations emerging from a wide body of evidence accrued from animal experiments, as well as observational studies, clinical trials and metabolic studies conducted in diverse human populations. Saturated fatty acids raise total and low-density lipoprotein (LDL) cholesterol, but individual fatty acids within this group, have different effects.

Myristic and palmitic acids have the greatest effect and are abundant in diets rich in dairy products and meat. Stearic acid has not been shown to elevate blood cholesterol and is rapidly converted to oleic acid in vivo.

The most effective replacement for saturated fatty acids in terms of coronary heart disease outcome are polyunsaturated fatty acids, especially linoleic acid. This finding is supported by the results of several large randomized clinical trials, in which replacement of saturated and trans fatty acids by polyunsaturated vegetable oils lowered coronary heart disease risk.

CONCLUSION

The intake of food, considered in relation to the body's dietary needs. Good nutrition - an adequate, well balanced diet combined with regular physical activity - is a cornerstone of good health. Poor nutrition can lead to reduced immunity, increased susceptibility to disease, impaired physical and mental development, and reduced productivity. The diet of an organism is what it eats, which is largely determined by the availability, processing and palatability of foods. A healthy diet includes preparation of food and storage methods that preserve nutrients from oxidation, heat or leaching, and that reduce risk of food-born illnesses. But to what extent can contribute a special diet to help alleviate certain diseases, many do not realize. So fresh vegetables play due to its blood-sugar-regulating effect, for example, in diabetes a very important roll. Gout patients should avoid animal products and sugar. Fruit and vegetables and plenty of fiber reduce the risk of cancer and help a tired intestines on the jumps and so on. In different age the approximation is very strictly observed. Wholesome food and drink includes a varied selection, proper amount and combination of nutritious and low-energy food. To choose predominantly fresh plant foods helps the whole metabolism. Too many saturated fatty acids increase the risk of dyslipidemia, with the possible consequence of cardiovascular diseases. These have a health-promoting action and support sustainable diet. In order to we are so well supplied and reduce the risk of food-related diseases with vitamins, minerals and fiber and phytochemical.

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