

Contribution of Nutrition to the Health Transition in Developing Countries: A Framework for Research and Intervention

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The major focus of public health programs in developing populations is alleviating undernutrition. In South Africa, however, as in many other developing countries, the African population is experiencing rapid urbanization characterized by a double burden of disease in which noncommunicable diseases (NCD) become more prevalent and infectious diseases remain undefeated. The possible mechanisms through which nutrition contributes to the additional vulnerability to NCD experienced by developing populations are explored, and research priorities in this area are identified.

The global health transition,¹ also referred to as the epidemiologic transition,² can be defined as the complex changes in patterns of health, disease, and mortality that result from demographic and associated economic and societal changes in a world population that is getting older.^{1,2} In this transition, noncommunicable diseases (NCD) replace infectious diseases, perhaps with the exception of HIV/AIDS, as primary causes of morbidity and mortality.^{1,2} But, by contrast with the health transition experienced during industrialization and economic growth in developed countries, many developing countries that are in a rapid process of urbanization are experiencing a health transition characterized by a double burden of disease in which NCD becomes more prevalent and infectious diseases remain undefeated.^{1,2} Coronary heart disease (CHD), stroke, and other NCDs now cause 39% of all deaths in developing countries.¹ The alarming trend is that NCDs affect younger people in developing countries more often than in developed countries.¹ Of the estimated 12 million global deaths

per year caused by CHD and stroke, an increasing number occur in developing countries, whereas dramatic reductions in mortality from circulatory diseases are experienced in developed countries such as Australia, Canada, Finland, New Zealand, and the United States.¹

The 1998 World Health Report¹ rightly points out that the war against ill-health in the 21st century will have to be fought simultaneously on the two main fronts of infectious and noncommunicable diseases and that many developing countries will come under greater attack from both. The major dilemma facing these countries is how to best promote economic growth, reduce infectious diseases, and at the same time delay or prevent the onset of NCD.³ According to Walker and Segal,⁴ a major problem facing developing countries today is the increasing dichotomy in socioeconomic status: the rich are getting richer and the poor are getting poorer. The inequality in income is reflected in inequalities in health. This challenge will call for difficult decisions about the allocation of sparse resources in the fragile economies and limited health budgets of developing countries.

Nutrition underlies many of the public health problems and societal challenges in the world today. A better understanding of the determinants and changes in dietary patterns and nutrient intakes during the demographic transition in developing countries and how these changes or the nutrition transition influences health outcomes could lead to more appropriate and relevant policies, strategies, and intervention programs to protect and promote health and to prevent and control disease.

For a better understanding of why people in developing countries who are experiencing this demographic transition seem to be at increased risk for NCD when compared with the present situation in developed countries, it is probably necessary to move beyond the epidemiologic associations between nutrition and health to an analysis of the possible mechanisms through which nutrition influences the development of NCD. The objectives of this paper are to briefly review the nutrition transition, explore the possible mechanisms through which and to what ex-

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tent nutrition contributes to the development of NCD in developing countries, identify research priorities in this area, and suggest how knowledge of the possible mechanisms should be incorporated into direct intervention programs.

The Nutrition Transition

The nutrition transition is a sequence of characteristic changes in dietary patterns and nutrient intakes associated with social, cultural, and economic changes during the demographic transition.²⁻⁴ Traditionally, the nutrition transition, although not named as such, was described as a change from a traditional, indigenous, rural, high-fiber, low-fat diet that is eaten by poorer people to a more affluent, Western type of diet, rich in animal fats and low in fiber, which is eaten by those who are better-off economically.⁵ At present, the changes are more complicated than this basic description. In an analysis of global food availability data for 1962–1994, Drewnowski and Popkin³ showed an uncoupling of the classic relationship between income and fat intake resulting from the global availability of cheap vegetable fats and oils. This has resulted in increased fat intakes in low-income countries. They concluded that the nutrition transition now occurs at lower levels of the gross national product than it had previously, and that it is accelerated by high urbanization rates.

It is important to realize that the nutrition transition, as illustrated in Figure 1, is characterized by a stepwise progression that depends on the balance of contributing factors. For example, whereas urbanization in more industrialized countries was associated with economic growth, in many developing countries it results in urban poverty.⁶ This will influence food affordability and choices and may lead to diets that are inadequate in certain micronutrients.

Despite the monotony of a small variety of foods, the low-fat, high-fiber, rural diet can be adequate in both macro- and micronutrients and is associated with low risk of both infectious and chronic diseases.⁷ More often, however, this diet has been associated with undernutrition and increased risk of infectious diseases,⁸ especially in children and when other determinants of malnutrition (e.g., lack of care, household food insecurity, disease, poverty, and lack of health services) are present. It is therefore conceivable that in changing to a more affluent, Western type of diet (which is more diverse and varied and contains more foods from animal products, more fats, and less staple foods and fiber), a stage will be reached where the rural diet becomes more adequate but can still be prudent. This point, where both adequacy and prudence are reached, is the “optimal” diet to which many informed people in developed countries now aspire or have returned to decrease their risk of NCD. In rich, developed countries such as the United States, meat intake is declining, and fat intake has an inverse relationship with socioeconomic status.³ In contrast, in many developing, low-income countries, so-

cioeconomic status has a positive relationship with fat intake and risk of NCD.³ Based on the contribution of total fat and especially saturated fat to risk of NCD,⁹ these differences in fat intake could partly explain the “social drift”⁶ of chronic diseases. In the past, for example, CHD was more prevalent in the higher social classes. Today, this is still true for less developed countries; however, in more developed countries, those in the lower socioeconomic class have higher rates of NCD.¹⁰

Figure 1 illustrates the consequences of the nutrition transition and the different health patterns associated with the various “stages” of the transition. The figure clearly indicates that nutrition is but one of many factors that influence the risk of both infectious and chronic diseases. The different patterns in the emergence of NCD associated with very similar dietary changes in different populations and countries may provide a clue to the importance of other contributing factors.

Comparison of the Nutrition Transition and Emergence of NCD in Different Areas of the World

In South Africa, it is the African (black) population who is presently experiencing a rapid process of urbanization. The white, European-African-Malays, and Asian-Indian populations are following diets in which more than 35% of total energy is contributed by fat.¹¹ The diet of rural Africans is still very low in fat (less than 20% in some instances),^{7,11} but urban Africans are now consuming approximately 30% of their energy as fat.¹¹⁻¹³ The contribution of total protein to energy intake does not vary much between these groups and seems not to change much with urbanization. However, the ratio of plant to animal protein intake has changed dramatically in the diets of urbanizing Africans. MacIntyre¹² showed that this ratio was 1.13:1.0 in rural African women and 0.58:1.0 in urban women. The corresponding figures for men were 1.0:1.0 (rural) and 0.72:1.0 (urban). The same study showed that consumption of fruit and vegetables increased with urbanization, leading to higher dietary fiber and micronutrient intakes in the urban African groups.

At this time, it seems that the diet of most urban Africans may be more micronutrient-dense than that of rural Africans, but more prudent than the typical Western diet eaten by other population groups in South Africa.¹¹⁻¹³ Low calcium intake is a feature of the diet of both rural and urban Africans.¹¹ The emergence of NCD in South African blacks is characterized by high rates of female obesity,¹⁴ hypertension in men and women,¹⁵ and stroke¹⁶ but low rates of CHD¹⁷ and large bowel cancer.¹⁸ The prevalence of obesity in black women is now double that of white women.¹⁴ The high prevalence of noninsulin dependent diabetes mellitus (NIDDM) in South African Indians has been documented,¹⁹ but the situation in blacks is less clear.²⁰ Levitt and coworkers²⁰ found an age-adjusted world

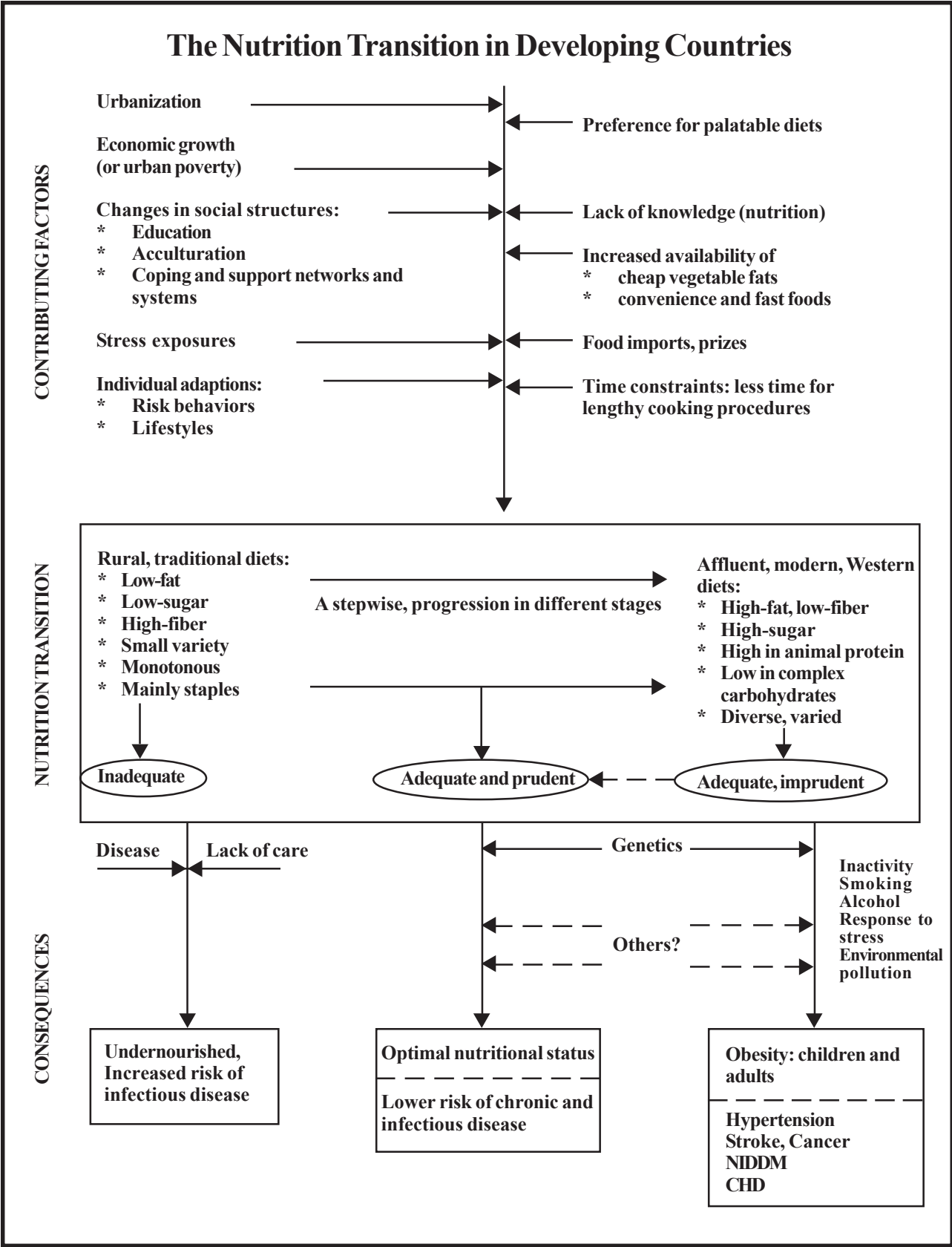


Figure 1. Schematic representation of the contributing factors to and consequences of the nutrition transition.

population prevalence of 8.0% (confidence interval = 5.8–10.3%) in urban Africans in the Cape Town area. Mollentze et al.²¹ found an age- and sex-adjusted prevalence of 6% in Africans in Mangaung, an urban area in the Free State, and 4.8% in the more rural area of QwaQwa. These studies indicate that the degree of obesity and level of urbanization are factors that possibly contribute to a rise in NIDDM prevalence with urbanization of Africans.

The nutrition transition in China^{22,23} has resulted in attaining food security and meeting energy and protein needs on a national and household level, although undernutrition still exists in certain segments of Chinese society. The transition has been characterized by increased intakes of meat, plant oils, and alcohol. Twenty-five percent of the Chinese population of 1.2 billion live in urban centers, and 75% of these households consume more than 30% of their energy as fat.²² Milk consumption remains low (11 kg/person per year) with resultant low mean calcium intake (400–500 g/day). Salt intakes vary geographically from 13–17 g/person per day in the north to 11–15 g/person per day in the south. Salt intake showed a positive correlation with blood pressure in Chinese populations.²² It seems that the nutrition transition in China is associated with increases in obesity and hypertension.^{22,23}

Gopalan²⁴ reviewed the nutrition transition in Southeast Asia. Increased intakes of total fat, hydrogenated fat in place of vegetable oils, ghee, foods from animal origin, and sugars are commonly seen with urbanization. Whereas urbanization and migration of Africans and Chinese people are associated with an early emergence of obesity, hypertension, and stroke, South Asians show high mortality from CHD and accompanied NIDDM. Insulin resistance associated with central (android) obesity seems to be a feature of Indian patients with NIDDM, even when they are not overweight or obese by conventional criteria.²⁵

In Latin America and the Caribbean, economic development and food imports have led to increased intake of foods of animal origin and of total fat, whereas intake of fruits, vegetables, roots, and tubers are low.²⁶ High prevalence of obesity among women and teenage girls is seen. Countries with the highest per capita energy availability have significantly higher mortality rates owing to diabetes mellitus, whereas countries with the highest fat intakes have the highest CHD mortality.²⁶

The above illustrations suggest that worldwide the nutrition transition is characterized by excessive intakes of energy, fat (especially from animal products), and sugars (in some countries), probably because when enough food is available and affordable the sense of taste acts as the main gatekeeper or determinant of food consumption, and the preference for sweet and fatty foods is an innate human characteristic.³ The data also suggest, however, that the pattern of emergence of NCD differs in various areas of the world. Reddy²⁷ pointed out that part of this variance might be related to the different stages of epide-

miologic transition, but that genetic–environment interactions probably contribute most to this variability. This notion is supported by the observations that migrant populations often display higher prevalence of NCD than their host populations.²⁵ In addition to a possible genetic vulnerability, however, other mechanisms such as early metabolic programming also should be considered.

Mechanisms Through Which the Nutrition Transition May Contribute to Development of NCD

The “direct” mechanisms through which the imprudent, typical Western diet and overnutrition influence the development of NCD have been the subject of intensive research on an epidemiologic, clinical, and molecular level during the past 50 years. All NCD are multifactorial, and several lifestyle factors such as smoking, inactivity, adverse response to stress, lack of sleep, and alcohol abuse will interact with nutritional factors to increase the risk of these diseases in genetically susceptible individuals. The nutritional factors that are thought to contribute directly to risk of NCD are summarized in Table 1, which compiles information from recent reviews on the subject.^{28–36}

But the higher prevalence of NCD in migrants than in host populations,²⁵ the very rapid increases seen with urbanization and westernization of traditional indigenous populations,^{19–24} and the fact that in these populations people are affected at younger ages than in developed populations¹ suggest an “extra” vulnerability of people in transition. The fact that all known adult risk factors of lifestyle do not fully explain incidence, prevalence, geographic variations, trends over time, and individual variations of CHD has led to the formulation of hypotheses that fetal malnutrition^{37,38} and infant nutrition³⁹ determine adult responses to environmental changes, including diet, thereby influencing the risk of NCD. A number of publications in recent years have provided evidence for these relationships between early malnutrition and later susceptibility to hypertension, abnormal lipid metabolism, insulin resistance, altered endocrine and immune functions, and consequent increased risk for obesity, CHD, and NIDDM. David Barker’s group in Southampton^{37,38} and others^{40–43} have shown that undernutrition in utero could affect cells in organs during critical periods of development, thereby influencing the distribution of cell types, hormonal feedback mechanisms, and metabolic activity and organ structure, resulting in babies with small size or disproportionate size and compromised organ functions at birth. Epidemiologic studies comparing risk of NCD in adults based on information about birth size and anthropometry confirmed that maternal and fetal undernutrition may influence adult responses to the environment.^{37–43} As James⁴⁴ pointed out, however, the additional contribution of fetal malnutrition to adult risk of NCD is not yet established.

Table 1. Possible Mechanisms Through Which Nutrients and Dietary Factors Influence Development of Noncommunicable Diseases (NCD)

Dietary Factor	Physiologic, Biochemical Effect (Mechanism; Increased Risk)	Health Outcomes (Increased Risk)	Reference
Energy excess	* Fat storage * Overweight * Obesity	All NCD	28
↑ Total fat	↑ TC, LDLC, TG, ↓ HDLC	Atherosclerosis, CHD	29
↑ Saturated fat	↑ IR	Hypertension, NIDDM	30
↑ <i>Trans</i> fatty acids	↑ LDLC, ↓ HDLC		30
↑ Sodium	Abnormal renal function curve: disturbed water and electrolyte balance	Hypertension, stroke	31
↓ Calcium	Hypertension? ↓ Bone mass	Hypertension, stroke, osteoporosis	32
↓ Antioxidants	↑ Oxidation of LDLC and other molecules, changing their function	Cancer, CHD	33
↓ Dietary fiber	↑ TC, LDLC, TG, ↓ HDLC ↑ IR ↑ Fibrinogen? Clotting? ↓ Fibrinolysis	Atherosclerosis Hypertension, NIDDM Thrombosis, stroke, MI Thrombosis, stroke, MI	34
Undernutrition	↑ Fibrinogen	Thrombosis, stroke, MI	35
↓ Phytochemicals	↓ Modulation of hormone functions	↑ Cancers in different sites	36

Note: TC = total serum cholesterol; LDLC = low-density lipoprotein cholesterol; TG = triglycerides; HDLC = high-density lipoprotein cholesterol; CHD = coronary heart disease; IR = insulin resistance; NIDDM = noninsulin dependent diabetes mellitus; MI = myocardial infarction.

The extensive work of McGill and coworkers³⁹ with the baboon model over a period of more than 20 years showed that infant feeding and weaning practices may program metabolism further to determine metabolic responses to dietary challenges in later life. Reporting on the effect of intrauterine and infant malnutrition during the siege of Leningrad on adult risk of disease more than 50 years later, however, Stanner and coworkers⁴⁵ found no effect of malnutrition in subjects who were exposed to undernutrition as infants during the siege. Clearly, much more evidence is needed before firm conclusions regarding metabolic programming in humans can be drawn.

Nevertheless, if maternal, intrauterine, and infant malnutrition confer an additional risk for the development of NCD in adulthood, the developing world population will be even worse off than the population in developed countries when there are alterations in dietary and physical activity patterns. Developing countries already carry a heavy burden of low birth weight and stunting. This possible additional risk is summarized in Figure 2.

The black box at the top of the figure indicates that, regardless of fetal and childhood circumstances, when people are exposed to the typical Western diet, the risk of NCD will be increased through the mechanisms shown in Table 1. But there are indications that undernutrition in adult life—especially in African men³⁵—also is one of the

determinants of increased plasma fibrinogen, a major risk factor for hypertension, stroke, and myocardial infarction. The second box shows the possibility of a thrifty genotype, which may confer additional risk. It has been hypothesized⁴⁶ that this genotype, strengthened by natural selection, may allow some individuals or populations to better withstand starvation, but that this genotype also makes them more susceptible to obesity in environments characterized by adequate or abundant amounts of food and low levels of physical activity. The third box shows how fetal malnutrition may lead to increased risk of NCD in adult life, regardless of the adult diet. The fourth box indicates how metabolic programming could contribute to risk. Therefore, available evidence suggests that if children grow up in poor, disadvantaged conditions and then become prosperous, the metabolic system will not be able to cope with the new environment;^{37,39} this may lead to the epidemics of NCD seen in populations of developing countries undergoing the nutrition transition.

The above hypotheses seem to explain the sharp increases in NCD in developing populations in transition. It should be emphasized that most of the evidence, as mentioned above, comes from developed populations where circumstances during childhood could be traced. Much more research in developing populations, however, is needed.

Possible Mechanisms Creating Additional Risk of NCD

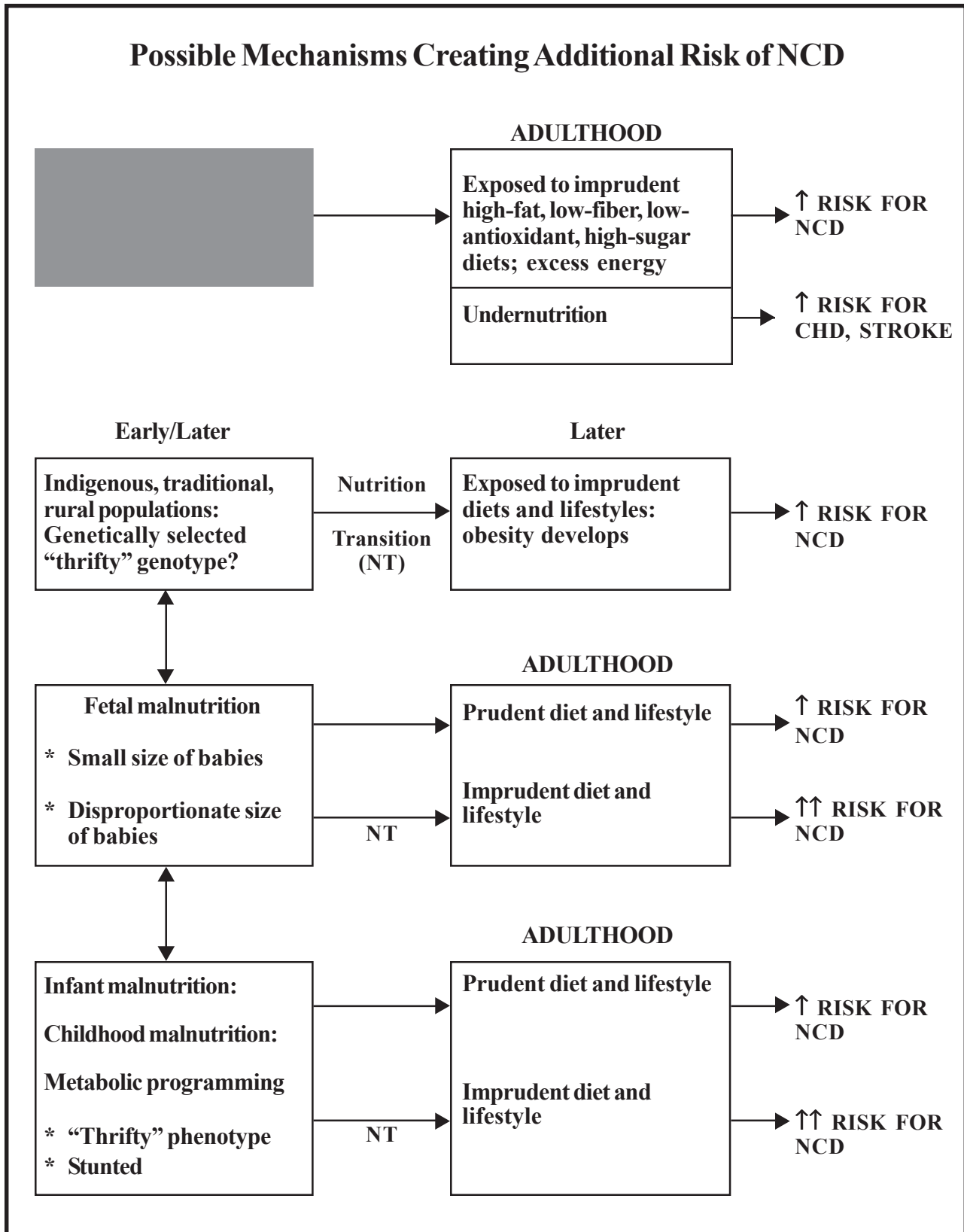


Figure 2. Schematic representation of ways in which fetal, infant, and childhood circumstances may influence risk of NCD in adulthood.

Research Needed to Confirm the Early Life Hypotheses

Barker^{37,38} mentions that to better understand the effects of fetal malnutrition, more should be known about the underlying cellular and molecular mechanisms, of factors limiting nutrients and oxygen to the fetus, how the fetus adapts to the limited supply of nutrients and oxygen, and how these adaptations program organ structure and function. Other issues also need further clarification. Especially in developing populations, the relative contributions of genetic susceptibility, fetal malnutrition, and adverse influences during childhood development on later risk for NCD should be dissected. The major question that should be answered is whether optimal diets and lifestyles during adulthood would cancel out or prevent the possible long-term effects of early malnutrition and genetic predisposition to NCD. Data from developed countries⁴⁷ suggest that appropriate interventions and healthful lifestyles during adult life can prevent or delay the onset of NCD and also may reverse the progression of these diseases.

In many developing countries such as South Africa, people who grew up under adverse and disadvantaged circumstances have now adopted typical Western lifestyles and eating patterns. Interethnic comparisons of people who have different childhood circumstances regarding lifestyle and/or nutrition risk factors for NCD could provide useful information to test the early malnutrition hypotheses. Measurements of the effects of birth circumstances and childhood malnutrition (stunting) on adult risk of people in different stages of the nutrition transition should now be possible. Moreover, aggressive lifestyle and dietary interventions in these adults should be started because they will provide information on the possibility of preventing the long-term effects of fetal and childhood malnutrition.

Possible Health Consequences in Developing Countries

Starting dietary and lifestyle interventions in developing populations in which the major focus of public health programs is alleviating undernutrition, especially in women and children, will be difficult and politically sensitive. However, the existing increases of NCD and their future burden on health services should be sufficient motivation to institute such interventions.

Programs could be designed that address both under- and overnutrition simultaneously, with emphasis on optimal nutrition. Whereas programs in different countries to address undernutrition could be very similar to one another because of similar immediate, basic, and underlying causes of undernutrition, programs aimed to address the causes and consequences of overnutrition in different countries will probably have different aims. For

example, in South Africa and China, emphasis could be on preventing obesity and hypertension (i.e., concentrating on energy, sodium, and calcium intakes), whereas in Southeast Asia, emphasis could be on preventing NIDDM and CHD (i.e., concentrating on energy, type of fat, and fiber intakes).

The putative “extra” or additional vulnerability for NCD experienced by developing populations also could be addressed by rethinking optimal nutritional needs.⁴⁴ For example, the amount of vitamin C in the diet that is necessary to prevent a deficiency (scurvy) is set at 60 mg/day. To prevent oxidized DNA in sperm cells, at least 100 mg/day is needed,⁴⁴ and to prevent NCD, 120 mg/day has been suggested.⁴⁸ It could be that if daily requirements of micronutrients are set at a level that will prevent deficiency diseases and at the same time prevent chronic diseases, this “extra” vulnerability could be alleviated. This paradigm shift in setting micronutrient requirements is not new. Russell⁴⁹ recently pointed out that scientists today should use chronic disease prevention rather than deficiency states to set daily requirements for the elderly. Instead of deciding how much of a particular nutrient is needed to maintain “normal” blood levels and prevent a deficiency state, the emphasis should be on how much of the nutrient is needed to prevent chronic disease such as osteoporosis or influence a marker of a chronic disease such as homocysteine level.⁵⁰

The challenge for developing countries will be to design appropriate, multifaceted, and multisectorial programs that address under- and overnutrition jointly and that provide adequate, affordable, and quality foods to make this possible. If health policies are based on nutritional concerns, and if emphasis is on promotion and prevention rather than on curative services, this should be possible, even in countries with limited health budgets.

Conclusions and Summary

Several authors in recent years have remarked on the increased vulnerability of developing populations experiencing a demographic and nutrition transition to increased risk of NCD at a time when the battle against infectious diseases, including HIV/AIDS, is ongoing. There is accumulating evidence that, in addition to a possible genetic contribution, fetal and childhood malnutrition contribute to this vulnerability. Fetal and childhood malnutrition will therefore bring about a higher susceptibility to the consequences of overnutrition when people are exposed to affluent conditions during adult life. It seems, therefore, that to prevent NCD or at least to decrease the risk of development of these diseases, it would be necessary to prevent undernutrition in pregnant mothers and children and overnutrition at all stages of the life cycle. Because dietary behaviors are developed early in life, the principles of a prudent diet should be promoted among children in developing countries. The protective factors in the diet, such

as antioxidants and phytochemicals, should guide recommendations and formulation of nutrient requirements.

The prevention of undernutrition (e.g., by increasing household food security, increasing health services, and improving socioeconomic status) is often the major focus of health and nutrition policies and programs in the developing world. These programs, however, also should include strategies to prevent overnutrition and promote healthy lifestyles. Nutrition messages to the population should promote an "optimal" diet and create awareness of the consequences of overnutrition. To do this on limited health budgets, concerted efforts from various public and private sectors, including the food industry in developing countries, will be needed. The food industry can play a very important role in promoting health in developing countries. More foods and food products reflecting traditional dietary patterns and "designed" for optimal nutrition principles should become available at affordable prices in these countries. To overcome undernutrition without "creating" overnutrition is a challenge that developing countries cannot ignore if the ultimate goal is to invest in the long-term health of their people.

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