

*The Second Africa Nutritional Epidemiology Conference was held at Gimpa, Legon, Ghana on 15–18 August 2006*

## **The epidemiological and nutrition transition in developing countries: evolving trends and their impact in public health and human development**

### **Epidemiological and nutrition transition in developing countries: impact on human health and development**

Paul Amuna\* and Francis B. Zotor

*School of Science, University of Greenwich, Medway Campus, Chatham Maritime, Kent ME4 4TB, UK*

Whereas common infectious and parasitic diseases such as malaria and the HIV/AIDS pandemic remain major unresolved health problems in many developing countries, emerging non-communicable diseases relating to diet and lifestyle have been increasing over the last two decades, thus creating a double burden of disease and impacting negatively on already over-stretched health services in these countries. Prevalence rates for type 2 diabetes mellitus and CVD in sub-Saharan Africa have seen a 10-fold increase in the last 20 years. In the Arab Gulf current prevalence rates are between 25 and 35% for the adult population, whilst evidence of the metabolic syndrome is emerging in children and adolescents. The present review focuses on the concept of the epidemiological and nutritional transition. It looks at historical trends in socio-economic status and lifestyle and trends in nutrition-related non-communicable diseases over the last two decades, particularly in developing countries with rising income levels, as well as the other extreme of poverty, chronic hunger and coping strategies and metabolic adaptations in fetal life that predispose to non-communicable disease risk in later life. The role of preventable environmental risk factors for obesity and the metabolic syndrome in developing countries is emphasized and also these challenges are related to meeting the millennium development goals. The possible implications of these changing trends for human and economic development in poorly-resourced healthcare settings and the implications for nutrition training are also discussed.

#### **Epidemiological and nutritional transition: Double burden of disease: Millennium development goals: HIV/AIDS: Sub-Saharan Africa**

The demographic transition, described as an upward shift in population dynamics associated with socio-economic development (i.e. rising incomes, education, employment, improvements in health status and life expectancy and changes in lifestyles), has been said to be accompanied by an epidemiological transition<sup>(1)</sup>. The latter transition was described by Omran in the 1970s as a characteristic shift in the disease pattern of a population as mortality falls during the demographic transition<sup>(2)</sup>. Thus, as acute infectious diseases are reduced, chronic degenerative diseases increase in prominence, causing a gradual shift in the age pattern of mortality from younger to older ages. It is believed that the epidemiological transition in industrialized

countries emerged towards the early 1900s, with rising levels of non-communicable diseases (NCD) that probably peaked by the mid 1950s, accompanied by a marked fall in infectious-disease morbidity and mortality<sup>(3)</sup>. Evidence for the emergence of the epidemiological transition has often been associated with epidemics of diseases of the heart and blood vessels (including hypertension, IHD and cerebrovascular disease), cancers, type 2 diabetes mellitus, osteoporosis, neuropsychiatric disorders and other chronic diseases<sup>(4)</sup>, which are now major contributors to the burden of disease in both developed and developing countries.

As the understanding of the links between demographic changes and their impact on the nutritional status of

---

**Abbreviations:** MDG, millennium development goal; NCD, non-communicable diseases.

**\*Corresponding author:** Dr Paul Amuna, fax +44 208 331 9805, email P.amuna@gre.ac.uk

individuals and populations has increased, the term nutrition transition has been used to characterize the shift in disease patterns towards diet- or nutrition-related NCD, including type 2 diabetes mellitus, CVD, stroke, high blood pressure, gout and certain cancers. This shift in disease patterns is associated with changes in behaviours, lifestyles, diets, physical inactivity, smoking and alcohol consumption.

Accumulating evidence points to strong phenotypic and genotypic factors that define individual risk to stressful environmental-exposure variables during fetal life<sup>(5,6)</sup>, the early infant environment and later life. Adverse exposure and the accumulation of nutritional and health risk has a greater impact in developing countries, in which a very strong link between poor maternal nutritional status and low birth weight and increased infant and childhood morbidity and mortality has been established. Compared with developed countries, which have low intrauterine growth restriction rates, in developing countries the incidence of intrauterine growth restriction and congenital anomalies varies between 15 and 50%<sup>(7,8)</sup>.

Of greater importance and concern to nutritional scientists, health workers and governments in developing countries is the double burden of disease following the epidemiological and nutritional transition. Whereas in developed countries there has been a fall in perinatal, infant and childhood mortality rates from communicable diseases and a drop in maternal mortality rates in real terms, in most developing countries diseases related to poverty and food insecurity continue to contribute to rising mortality rates. At the same time, in those developing countries that have rising incomes the overall NCD burden is relatively higher than that seen in developed countries<sup>(9)</sup>. Furthermore, diseases such as malaria, and over the last three decades the HIV/AIDS pandemic, have added to the overall burden of disease in countries with poor health services and weaker economies. A matter of great concern and requiring urgent action is how these various factors have combined to undermine human and economic development in these countries.

### Veterans of the early malnutrition wars

The 'early malnutrition wars' begin *in utero* during early embryonic and fetal development when the developing fetus depends entirely on its own genotype and the materno-fetal environment. During these critical periods of human development there is increased demand for energy to meet the metabolic demands of pregnancy, i.e. to support maternal weight gain and ensure the growth of the fetus, placenta and associated maternal tissues<sup>(10-12)</sup>. Reduced energy intake or food restriction, particularly in an undernourished mother, will predispose to nutritional stresses resulting in intrauterine growth restriction as a coping mechanism, with varying outcomes that include the birth of babies who are small for their gestational age or of low birth weight, which has early-life and/or later-life health consequences<sup>(13-15)</sup>. The pattern of growth during the critical periods of fetal life is a strong predictor of later susceptibility to type 2 diabetes mellitus, hypertension and hyperlipidaemias<sup>(16-20)</sup>.

There is considerable evidence from animal studies and clinical assessments in human subjects to support the view that the prenatal environment triggers developmental changes in the endocrine, organ and physiological characteristics of the fetus that may persist after birth. The list of postnatal traits shown to be influenced by the prenatal environment has expanded to include such important functions as immunity, reproductive function, growth rates and muscle mass<sup>(21,22)</sup>.

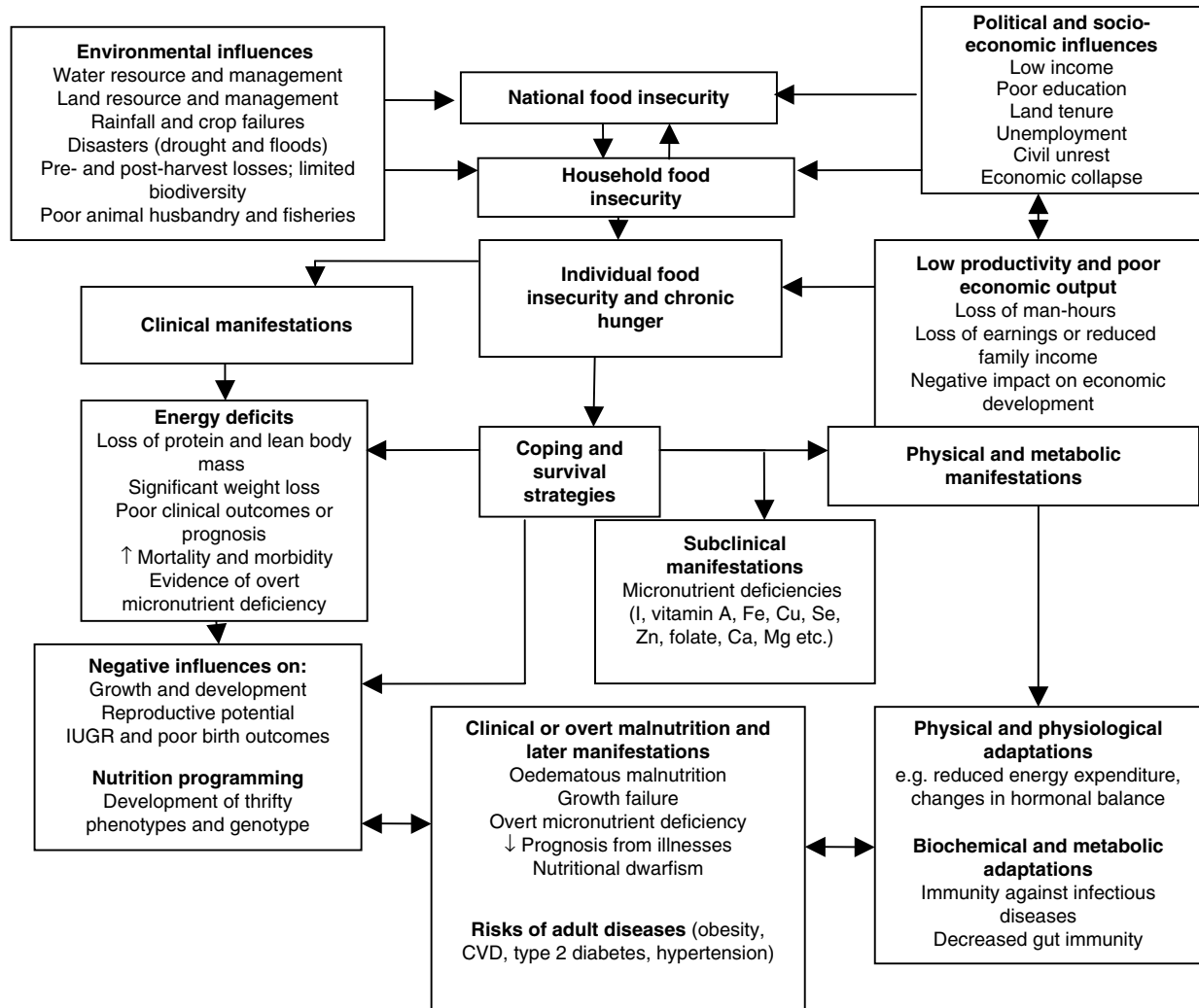
Chronic hunger and continuous maternal malnutrition during pregnancy thus triggers fetal adaptations that are aimed at survival, probably with 'energy economy' at the centre of these survival mechanisms. This notion is supported by the fact that the largest component of tissue deposition, even in the mother during pregnancy, is that of fat deposition, accounting for 72% of tissue deposition<sup>(10,12,23)</sup>. It has previously been demonstrated that undernourished pregnant women in developing countries may show a pronounced suppression of metabolism that persists into the third trimester and offsets later increases in BMR, resulting in net energy saving over the entire gestational period<sup>(24)</sup>. This avid desire to economize and preserve energy at all costs under extreme conditions most probably occurs at the expense of other metabolic needs such as the synthesis of protective proteins of the immune system. Such 'compromises' may in turn increase the risk of infectious and communicable diseases in the neonate and infant and thus contribute markedly to the high rates of perinatal and infant mortality seen in many poor countries. Fig. 1 shows a proposed model of interactions that may have a bearing on health outcomes in food-insecure environments.

It has been suggested that inter-individual variations in metabolic responses to pregnancy represent a biologically-significant plasticity that has true adaptive value in enabling women to carry pregnancy to term under a wide range of nutritional conditions<sup>(10)</sup>, the full significance of the long-term outcomes of which are now becoming clearer<sup>(25)</sup>. The inter-individual differences have been interpreted as evidence of a functional plasticity that offers survival value to the fetus under conditions of suboptimal maternal nutritional status<sup>(21)</sup>. These authors have suggested that there is a mechanism that can monitor the mother's pre-pregnancy energy status and adjust the homeorrhetic changes in maternal metabolism accordingly, and evidence suggests that these metabolic adjustments, on average, assure a fetal weight that is proportional to maternal size<sup>(21)</sup>.

However, the costs of such metabolic adjustments to the offspring should be considered in the short and long term. It is known that women from poorer countries have much lower percentage pregnancy weight gains despite having low initial body weights<sup>(26)</sup>, and this factor appears to help reduce the rate of fetal growth restriction and weight reduction in the newborn<sup>(27)</sup>.

Links between the 'early malnutrition wars' or nutritional stresses in pregnancy and chronic adult disease through programming have been widely studied and reported since Hale's original hypothesis in 1962<sup>(5,17-20,22,28,29)</sup>.

In the context of mothers in poor developing countries, the thrifty phenotype and thrifty genotype hypotheses<sup>(30)</sup>



**Fig. 1.** A proposed model of interactions between food insecurity, human adaptations and nutritional and health risk in situations of poverty and chronic hunger. IUGR, intrauterine growth restriction; ↓, decreased; ↑, increased.

are of particular significance. In the former it is proposed that fetuses subjected to early nutrient restriction become metabolically attuned to these conditions and sustain aspects of a thrifty metabolism for the rest of their life<sup>(31,32)</sup>. The implications are that if the conditions of poverty and chronic hunger are replaced by affluence or nutritional abundance in later life, then thrifty phenotypes may be especially vulnerable to diseases of affluence, including type 2 diabetes mellitus and CVD<sup>(33)</sup>. Furthermore, the ‘veterans’ of the ‘early malnutrition wars’, who are born alive and may be of low birth weight, are more susceptible to infectious illnesses developed through the effects of intrauterine malnutrition on the developing immune system<sup>(21)</sup>. The thrifty phenotype model suggests that the organism adapts to poor nutrition in early life by programming its insulin metabolism to expect a similarly-depleted environment subsequently, and this adaptation appears to operate through insulin resistance rather than insulin secretion<sup>(34)</sup>.

The thrifty genotype hypothesis<sup>(30)</sup>, on the other hand, proposes that insulin resistance may have been beneficial

during periods of starvation because of its ability to maintain nutrition of the brain without degrading body protein stores, thus leading to natural selection of the underlying ‘diabetes genes’<sup>(35)</sup>. The notion that genes reducing insulin secretion or increasing insulin resistance will predispose to diabetes even in the young has been supported by a number of studies, including one relating to birth weight<sup>(36)</sup> and mutations of the glucokinase gene that influence both insulin secretion and birth weight<sup>(37)</sup>.

Indeed, the thrifty phenotype and thrifty genotype hypotheses are best viewed as complementary. The thrifty genotype hypothesis can account for selection over many generations, and hence explain the population differences in susceptibility to type 2 diabetes mellitus, whereas the thrifty phenotype hypothesis lends itself to individual adaptations during a single lifespan<sup>(38)</sup>.

Although veterans of the ‘early malnutrition wars’ bear many scars, these scars may not be readily visible but may be subclinical, subtle and present in a number of different ways (Fig. 1). It is the hidden scars that should be of particular concern, as they occur in the silent majority and

may deprive them of their full genetic potential. Such scars include micronutrient deficiencies and other biochemical disorders affecting metabolism, child development and cognitive and immune functions (Fig. 1).

The offspring may remain well protected as long as the environment is poor, but the programmed trait becomes inappropriate and maladaptive when the environment improves, e.g. with affluence or improved nutrition.

### A model of interacting factors predisposing to the double burden of disease in developing countries in relation to attainment of the millennium development goals

In 2000 diet-related diseases were by far the leading causes of death globally, accounting for  $>23.5 \times 10^6$  deaths<sup>(39)</sup>. These deaths undoubtedly include deaths attributable to under- and overnutrition, the co-existence of which, particularly in developing countries, is a major cause for concern. As is well known, high perinatal and infant mortality rates are strong markers of economic development, and in turn are an expression of the extent of poverty and the state of health service provision.

The clinical and physical manifestations of poverty and malnutrition must never be considered in isolation, as they have a direct impact on both present and future generations based on the thrifty phenotype and genotype hypotheses. An attempt has been made to put the model of interacting factors influencing the double burden of disease (Fig. 1) in the context of the millennium development goals (MDG) set out by the UN to be achieved by 2015<sup>(40)</sup>.

The first MDG is to eradicate extreme poverty and hunger. To achieve this ambitious goal the underlying causes of poverty and chronic hunger need to be addressed. Thus, the environmental, political and socio-economic influences giving rise to low productivity and poor economic output need to be resolved through national efforts towards self-emancipation and meaningful, honest and targeted global partnerships (MDG8). Such efforts should form part of mainstream national economic planning and its multi-faceted multi-sectoral nature recognized as the basis for national multi-agency policy planning and implementation through targeted national development plans. Failure to address these underlying causes has remained the major cause of national, household and individual food insecurity, the manifestations of which include subclinical micronutrient deficiencies and overt clinical presentations of growth failure and other forms of malnutrition.

Current global trends of disease burden further support the need for urgent sustained national and global action, particularly in developing countries. The global burden of NCD and the contribution of low- to middle-income countries is presented in Table 1.

Furthermore, recent statistics of the global leading causes of death emphasize the impact of the double burden of disease in developing countries. Of the ten leading causes of death in 2000 IHD and cerebrovascular disease were at the top in both developed (36.3% leading ten causes) and developing (17.1% leading ten causes) countries<sup>(9)</sup>. Except for respiratory infections (3.5%

**Table 1.** Contribution of low- and middle-income countries to the global burden of disease (from Reddy<sup>(52)</sup>)

	World	High-income countries	Low- and middle-income countries
Total deaths			
<i>n</i> ( $\times 10^3$ )	53 929	8033	45 897
%		14.9	85.1
Non communicable diseases (NCD)			
<i>n</i> ( $\times 10^3$ )	31 717	7024	24 693
%		22.1	77.9
Total disability life years (DALY) lost			
<i>n</i> ( $\times 10^3$ )	1 382 564	108 305	1 274 259
%		7.8	92.2
DALY loss as a result of NCD			
<i>n</i> ( $\times 10^3$ )	595 363	87 732	507 631
%		14.7	85.3

leading ten causes), all ten leading causes of death in developed countries were NCD<sup>(9)</sup>. In contrast, the majority of the ten leading causes of death in developing countries were largely preventable, and included (% leading ten causes): respiratory infections, 7.7; HIV/AIDS, 6.9; perinatal conditions, 5.6; chronic obstructive pulmonary disease, 5; diarrhoeal disease, 4.9; tuberculosis, 3.7; malaria, 2.6; road-traffic accidents, 2.5<sup>(9)</sup>.

Perinatal conditions, diarrhoeal diseases and tuberculosis are related to poverty, overcrowding and poor obstetric services. Diarrhoeal diseases are the direct result of poor water resource availability and management as well as food-safety issues. The high burden of disease from chronic obstructive pulmonary disease is largely related to tobacco smoking, which has been increasing in developing countries at the time when government legislation in developed countries has led to restrictions on smoking in public places. Reducing the extent of diseases of poverty and perinatal mortality would require the attainment of MDG2 and 3, i.e. achieving universal primary education and promoting gender equality and empowerment of women respectively.

Other MDG include: reduction in child mortality (MDG4); improving maternal health (MDG5); combating HIV/AIDS, malaria and other diseases (MDG6). Reducing childhood morbidity and mortality would require improved mother and child health services for early identification of nutritional and health risk. Skills training for health workers in the detection and treatment of malnutrition has also proved to markedly reduce child mortality, as a number of case studies in developing countries have shown<sup>(41)</sup>, and it is the present authors' view that this approach must be actively promoted and pursued globally as part of the strategy to achieving the MDG. Such an approach alongside improvements in maternal health services and, in particular, obstetric services would improve maternal well-being and, hopefully, result in better pregnancy outcomes. Improving maternal nutrition may help to reduce

**Table 2.** Projected global IHD mortality rates based on 1990 levels (from Bellizzi *et al.*<sup>(45)</sup>)

Region	Women			Men		
	1990 ( $\times 10^3$ )	2020 ( $\times 10^3$ )	% Increase	1990 ( $\times 10^3$ )	2020 ( $\times 10^3$ )	% Increase
Developed countries	1397	1809	29	1297	1921	48
Developing countries	1737	3825	120	1828	4337	137
China	377	684	81	386	811	110
Latin America	169	412	144	179	444	148

intrauterine nutritional stress, thereby reducing the levels of intrauterine growth restriction and its associated fetal adaptations, which may have health implications in early and/or later life.

Addressing the HIV/AIDS burden as a major goal has wide-ranging benefits, and failure to do so not only increases the health burden and decreases the lifespan, but has direct economic consequences, as most of those affected comprise the work force in developing countries. The relationship between HIV/AIDS and attainment of the MDG has been discussed elsewhere<sup>(42)</sup>.

Malaria control remains a major unresolved issue despite decades of apparent attempts to control the disease. Vector control to break the life cycle of the plasmodium parasite, although the best strategy for dealing with the disease, continues to elude health policy makers in many malaria-endemic developing countries. The use of mosquito nets impregnated with pyrethroids such as carbofuran and permethrin only deal with exposure to the parasite indoors and at certain times. Furthermore, following their apparent initial success in reducing malaria incidence, morbidity and mortality, resistance to these chemical agents is increasingly being reported<sup>(43)</sup>, thus suggesting that their limited impact may be short-lived and thus less cost effective in the long term.

Ultimately, the lynchpin between socio-economic status, food security and nutritional well-being lies in the ability of the individual, family and community to obtain good-quality food at affordable prices all year round and to have good water resources and food preservation methods that ensure food and nutritional safety. Approaches to increased food production in developing countries must be seriously addressed, using approaches similar to those of the Green Revolution of the 1970s, and will be a strong element in ensuring environmental sustainability (MDG7). Sustainable agriculture and appropriate applications of biodiversity need to be at the centre of the food security and nutrition agenda. The merits and disadvantages of food biotechnology need to be weighed very carefully and with great sensitivity, but objectively and with strong ethical standards. There is no doubt that biotechnology has a place in meeting the ultimate goal of eradicating chronic hunger. The way in which this process of increasing food production is executed, and the long-term benefits and sustainability, however, remain rather contentious issues.

Meeting national targets for poverty reduction and eradication of hunger would also require global partnerships for development (MDG8). Recently, the G8 group of economically-powerful nations, the UN and EU countries as

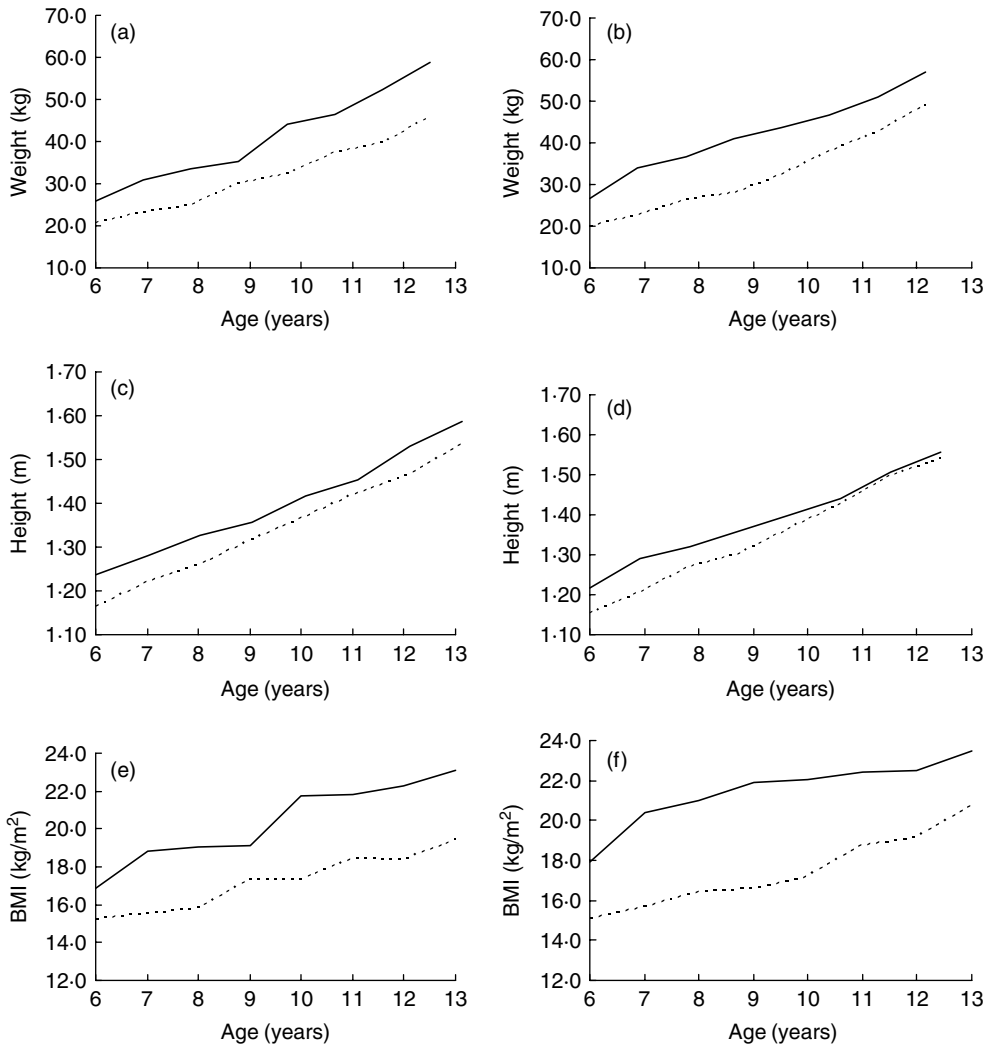
well as the USA, Canada, Australia and some Arab Gulf States, have pledged their commitment to support poor countries in getting out of poverty. Individuals and organisations have also contributed in no small measure to the new drive to 'make poverty history', although the fruits of these endeavours are yet to be fully realized.

The burden of NCD is projected to increase in developing countries if present trends continue. As shown in the model (Fig. 1), the adaptive or coping strategies of the individual lead to a number of physical, physiological and metabolic manifestations ranging from subtle subclinical outcomes to more overt and severe outcomes. Micro-nutrient deficiencies including Fe deficiency remain one of the major global nutritional challenges associated with poverty. Over the last six decades the global prevalence of Fe deficiency has been increasing, with levels rising from approximately 32% in 1960, to 42% in 1990 and approximately 60% in 2000<sup>(39)</sup>.

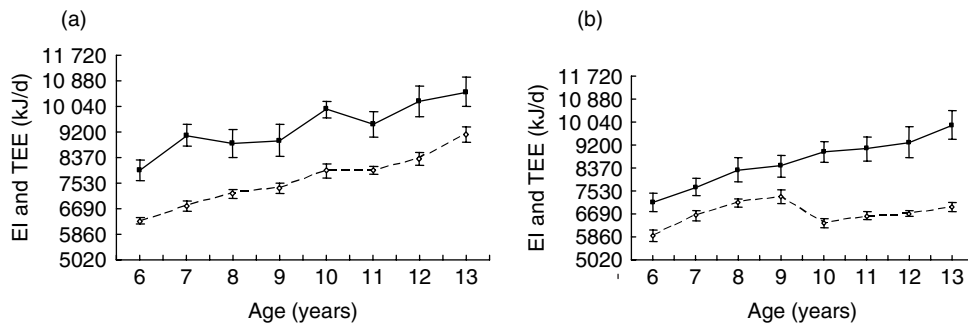
Projections of global IHD prevalence rates by the year 2020, based on 1990 levels (Table 2) and on current epidemiological trends, have recently been presented<sup>(44)</sup>. These projections show clearly that there is a skewing of the global burden of IHD, with the heaviest burden being borne by developing countries among women (120% overall) and Latin America having the highest increase in prevalence (144%) from 1990 levels. Over the same period the increase in IHD prevalence in developed countries is expected to be 29% among women. A similar pattern is expected in the male population, with increases by 2020 of 137% and 48% in developing and developed countries respectively. A number of countries in the EU have recently passed legislation to limit smoking in public places. If these policies on smoking prove effective, it is possible that levels of IHD in these richer countries could be even lower than predicted.

#### **Impact of the epidemiological and nutritional transition on childhood obesity and nutritional risk in a developing country in which incomes are rising**

Trends in overweight and obesity among children and adolescents have been reported for Europe and high-income Asian countries<sup>(45)</sup>, as well as for developing countries<sup>(7)</sup> in which income levels are rising, including China<sup>(46)</sup>, Chile<sup>(47)</sup> and Kuwait<sup>(48)</sup>. The evidence suggests that as income levels rise obesity and its co-morbidities become more prevalent among children and young adults in developing countries<sup>(49)</sup>. The obesity phenomenon has been investigated in a genetically-homogenous sample of



**Fig. 2.** Comparison of mean weight (a,b), height (c,d) and BMI (e,f) of age- and gender-matched 6–13-year-old Kuwaiti schoolchildren (males: a,c,e; females: b,d,f) in 2004 (—)<sup>(50)</sup> and 1984 (- - -)<sup>(53)</sup>.



**Fig. 3.** Differences between mean energy intake (EI; ■) and total daily energy expenditure (TEE; ◇) among male (a) and female (b) Kuwaiti schoolchildren. Values are means and standard deviations represented by vertical bars. Differences were significant for both males and females ( $P < 0.005$ ). (From Al-Shammari *et al.*<sup>(51)</sup>.)

1536 6–13-year-old Kuwaiti schoolchildren and an attempt has been made to study the trends over the last two decades. Some of the findings have recently been

reported<sup>(50,51)</sup> and they show that present-day age- and gender-matched cohorts of 6–13-year-old Kuwaiti children are taller and heavier than their counterparts were 20 years

ago, with a significant upward shift in BMI in both boys and girls ( $P = 0.02$ ; Fig. 2). Similarly, a significant excess of energy intake above estimated total daily energy expenditure was found for both boys and girls ( $P < 0.005$ ; Fig. 3). Rising income levels in Kuwait have led to a more sedentary lifestyle, over-reliance on motor vehicles and fast foods and poor food choices among school-age children. If these present trends are allowed to continue, the impact on the health service would be enormous. These significant shifts in an otherwise homogenous population within the last two decades seem to confirm an impact of the demographic and epidemiological transition on nutritional risk.

### Implications for training of nutrition and health professionals

The increasing burden of communicable diseases and NCD in developing countries has extensive economic ramifications, requiring ever-increasing contributions of the national income for health services. Developing countries will continue to bear an enormous burden of NCD, and the economic costs of the total disability-adjusted life years lost are extremely high. Furthermore, with persistently high levels of poverty and malnutrition the MDG set by the UN will not be met by 2015. The need for urgent, targeted and meaningful action is thus overdue and needs to happen at different levels, including building capacity in the field of nutrition and public health to help to address the issues. Currently, well-trained nutritionists and other relevant health professionals are in short supply in many developing countries, particularly Africa. To help to address the growing gap, the following proposals are put forward:

1. there is a clear and urgent need in developing countries, especially Africa, for modifications in the curricula for the health sciences (medicine, nursing, pharmacy, dentistry, public health and other subjects allied to medicine) to reflect these new realities;
2. the key focus of nutrition education of health professionals should be to enable them to: appreciate the relevance and importance of nutrition to the promotion of good health and the prevention and treatment of disease; identify nutrition-related problems of individuals and of the community; give consistent and sound advice to individuals in an appropriate manner and to work in close collaboration with dietitians and clinical nutritionists for more specific advice; know and promote dietary guidelines and nutrition requirements; provide appropriate and safe clinical nutritional support, working closely with registered dietitians in a multi-disciplinary team;
3. there must be an understanding of the costs and benefits of nutritional approaches in addition to pharmacological and other approaches to therapeutic and preventive health care, e.g. in the treatment of malnutrition and HIV/AIDS;
4. evidence-based nutritional knowledge should be assessed, updated and applied in healthcare practice;
5. the important role of nutrition in clinical-care audit and its importance as part of public health promotion and preventive programmes should be recognized.

### Summary

Joint training in nutrition for health professionals from all disciplines is desirable and should be considered. The input on nutrition should also be planned on a multi-disciplinary basis wherever possible. There is a need for global South–South and North–South collaboration between healthcare institutions to undertake joint research, nutrition interventions and other projects of benefit to populations in developing countries. Finally, nutrition needs to be at the centre of the health policy, and nutritionists and healthcare professionals need to work together to influence change in current approaches to healthcare provision in the fight against the double burden of disease in Africa and other developing regions.

### References

1. Lee R (2003) The demographic transition: three centuries of fundamental change. *J Econ Perspect* **17**, 167–190.
2. Omran AR (1971) The epidemiological transition: a theory of epidemiology of population change. *Milbank Q* **49**, 509–538.
3. Detels R & Breslow L (1997) Current scope and concerns in public health. In *Oxford Textbook of Public Health*, vol. 1, 3rd ed., pp. 3–17 [R Detels, WW Holland, J McEwen and GS Omenn, editors]. Oxford: Oxford University Press.
4. Patel MS, Srinivasan M & Laycock SG (2004) Nutrient-induced maternal hyperinsulinemia and metabolic programming in pregnancy. In *The Impact of Maternal Nutrition on the Offspring*. vol. 55, *Nestle Nutrition Workshop Series Pediatric Program*, pp. 137–151 [G Hornstra, R Uauy and X Yang, editors]. Cambridge: Woodhead Publishing Ltd.
5. Barker D (1992) *Fetal and Infant Origins of Adult Disease*. London: BMJ Publishing Group Ltd.
6. Lucas A (1994) Role of nutritional programming in determining adult morbidity. *Arch Dis Child* **71**, 288–290.
7. de Onis M & Blossner M (2000) Prevalence and trends of overweight among preschool children in developing countries. *Am J Clin Nutr* **72**, 1032–1039.
8. Ramakrishnan U, Manjrekar R, Rivera J, Gonzales-Cossio T & Martorell R (1999) Micronutrients and pregnancy outcome: a review of the literature. *Nutr Res* **19**, 103–159.
9. Beaglehole R & Yach D (2003) Globalisation and the prevention and control of non-communicable disease: the neglected chronic diseases of adults. *Lancet* **362**, 903–908.
10. Prentice AM, Spaaij CJK, Goldberg GR, Poppitt SD, van Raaij MAJ, Totto M, Swann D & Black AE (1994) Energy requirements of pregnant and lactating women. In *Energy and Protein Requirements. Proceedings of an International Dietary Energy Consultative Group Workshop* [NS Scrimshaw, JC Waterlow and B Schürch, editors]. <http://www.unu.edu/Unupress/food2/UID01E/uid01e14.htm#energy%20requirements%20of%20pregnant%20and%20lactating%20women>
11. Shetty PS & James WPT (1994) Defining chronic energy deficiency. *Body Mass Index – A Measure of Chronic Energy Deficiency in Adults* *FAO Food and Nutrition Paper no. 56*. Rome: FAO; available at [http://www.fao.org/docrep/t1970e/t1970e02.htm#P45\\_10041](http://www.fao.org/docrep/t1970e/t1970e02.htm#P45_10041)

12. Food and Agriculture Organization (2004) *Human Energy Requirements: Report of a Joint FAO/WHO/UNU Expert Consultation. Food and Nutrition Technical Report Series* no. 1. Rome: FAO.
13. Barker DJP (1997a) Fetal nutrition and cardiovascular disease in later life. *Br Med Bull* **53**, 96–108.
14. Barker DJP (1997b) Maternal nutrition, fetal nutrition and disease in later life. *Nutrition* **13**, 807–813.
15. Godfrey KM & Robinson S (1998) Maternal nutrition, placental growth and fetal programming. *Proc Nutr Soc* **57**, 105–111.
16. Barker DJP, Gluckman PD, Godfrey KM, Harding JE, Owens JA & Robinson JS (1993) Fetal nutrition and cardiovascular disease in adult life. *Lancet* **341**, 938–941.
17. Goldberg GR & Prentice AM (1994) Maternal and fetal determinants of adult diseases. *Nutr Rev* **52**, 191–200.
18. Lucas A, Fewtrell MS & Cole TJ (1999) Fetal origins of adult disease – the hypothesis revisited. *Br Med J* **319**, 245–249.
19. Robinson R (2001) The fetal origins of adult disease. *Br Med J* **322**, 375–376.
20. Ericksson JD (2005) The fetal origins hypothesis – 10 years on. *Br Med J* **330**, 1096–1097.
21. Prentice AM & Goldberg GR (2000) Energy adaptations in human pregnancy: limits and long-term consequences. *Am J Clin Nutr* **71**, 1226S–1232S.
22. Kuzawa CW (2005) Fetal origins of developmental plasticity: are fetal cues reliable predictors of future nutritional environment? *Am J Hum Biol* **17**, 5–21.
23. Hytten FE & Chamberlain G (1991) Weight gain in pregnancy. In *Clinical Physiology in Obstetrics*, pp. 173–203 [FE Hytten and G Chamberlain, editors]. Oxford: Blackwell Scientific Publications.
24. Lawrence M, Lawrence F, Coward WA, Cole TJ & Whitehead RG (1987) Energy requirements of pregnancy in the Gambia. *Lancet* **ii**, 1072–1076.
25. Godfrey KM & Barker DJB (2000) Fetal nutrition and adult disease. *Am J Clin Nutr* **71**, 1344S–1352S.
26. Neuman C, Bwibo N & Sigman M (1993) *Diet Quantity and Quality: Functional Effects on Rural Kenyan Families. Kenya Project Final Report, Phase II – 1989–1992*. Washington, DC: USAID.
27. King JC (2003) The risk of maternal nutritional depletion and poor outcomes increases in early or closely spaced pregnancies. *J Nutr* **133**, 1732S–1736S.
28. Lucas A (1991) Programming by early nutrition in man. In *The Childhood Environment and Adult Disease. CIBA Foundation Symposium* no. 156, pp. 38–55 [GR Bock and J Whelan, editors]. Chichester, West Sussex: John Wiley and Sons.
29. Barker DJP, Osmond C, Simmonds SJ & Wield GA (1993) The relation of small head circumference and thinness at birth to death from cardiovascular disease in adult life. *Br Med J* **306**, 422–426.
30. Hales CN & Barker DJ (1992). Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia* **35**, 595–601.
31. Wells JCK (2002) Thermal environment and human birth weight. *J Theor Biol* **214**, 413–425.
32. Wells JCK & Cole TJ (2002) Birth weight and environmental heat load: a between-population analysis. *Am J Phys Anthropol* **119**, 276–282.
33. Moore SE (1998) Nutrition, immunity and the fetal and infant origins of disease hypothesis in developing countries. *Proc Nutr Soc* **57**, 241–247.
34. Fowden AL & Hill DJ (2001) Intra-uterine programming of the endocrine pancreas. *Br Med Bull* **60**, 123–142.
35. Reaven GM (1998) Hypothesis: muscle insulin resistance in the ('not-so') thrifty genotype. *Diabetologia* **41**, 482–484.
36. Lindsay RS, Dabellea D, Roumain J, Hanson RL, Bennett PH & Knowler WC (2000) Type 2 diabetes and low birth weight: the role of paternal inheritance in the association of low birth weight and diabetes. *Diabetes* **49**, 445–449.
37. Hattersley AT, Beards F, Ballantyne E, Appleton M, Harvey R & Ellard S (1998) Mutations in the glucokinase gene of the fetus result in reduced birth weight. *Nat Genet* **19**, 268–270.
38. Lindsay RS & Bennett PH (2001) Type 2 diabetes, the thrifty phenotype – an overview. *Br Med Bull* **60**, 21–32.
39. World Health Organization (2002) *Vaccines and Biologicals: WHO Vaccine-Preventable Diseases: Monitoring System. 2002 Global Summary*. Geneva: WHO.
40. United Nations (2001) Road map towards the implementation of the United Nations Millennium Declaration. Report of the Secretary-General A/56/326. <http://www.un.org/documents/ga/docs/56/a56326.pdf>
41. Jackson AA, Ashworth A & Khanum S (2006) Improving child survival: Malnutrition Taskforce and the paediatrician's responsibility. *Arch Dis Child* **91**, 706–710.
42. Colecraft E (2008) HIV/AIDS: nutritional implications and impact on human development. *Proc Nutr Soc* **67**, 109–113.
43. Fanello C, Santomalazza F & della Torre A (2003) Simultaneous identification of species and molecular forms of the *Anopheles gambiae* complex by PCR-RFLP. *Med Vet Entomol* **16**, 461–464.
44. Yusuf S, Reddy S, Ôunpuu S & Anand S (2001) Global burden of cardiovascular diseases. Part II: Variations in cardiovascular disease by specific ethnic groups and geographic regions and prevention strategies. *Circulation* **104**, 2855–2864.
45. Bellizzi MC, Horgan GW, Guillaume M & Dietz W (2002). Prevalence of childhood and adolescent overweight and obesity in Asian and European countries. In *Obesity in Childhood and Adolescence. Nestle Nutrition Workshop Series* no. 49, pp. 23–35 [C Chen and WH Dietz, editors]. Philadelphia, PA: Lippincott Williams & Wilkins.
46. Chen C (2002) The growth pattern of Chinese children. In *Obesity in Childhood and Adolescence. Nestle Nutrition Workshop Series* no. 49, pp. 37–44 [C Chen and WH Dietz, editors]. Philadelphia, PA: Lippincott Williams & Wilkins.
47. Kain J, Burrows R & Uauy R (2002) Obesity trends in Chilean children and adolescents: Basic determinants. In *Obesity in Childhood and Adolescence. Nestle Nutrition Workshop Series* no. 49, pp. 45–61 [C Chen and WH Dietz, editors]. Philadelphia, PA: Lippincott Williams & Wilkins.
48. Al-Moussa MA, Shaltout AA, Nkansa-Dwamena D, Mourad M, AlSheikh N, Agha N & Galal DO (1999) Factors associated with obesity in Kuwaiti children. *Eur J Epidemiol* **15**, 41–49.
49. Freedman DS, Serdula MK & Khan LK (2002) The adult health consequences of childhood obesity. In *Obesity in Childhood and Adolescence. Nestle Nutrition Workshop Series* no. 49, pp. 63–82 [C Chen and WH Dietz, editors]. Philadelphia, PA: Lippincott Williams & Wilkins.
50. Al-Shammari H, Amuna P, Tewfik I, Bumejjad A & Zotor F (2006) Changing trends in physical characteristics and obesity risk in 6–13 year old Kuwaiti school children: evidence of a nutritional and epidemiological transition? *Proc Nutr Soc* **65**, 95A.
51. Al-Shammari H, Bumejjad A, Amuna P *et al.* (2005) Health habits and risk of obesity among 6–13 year old



- school children and adolescents. In *Nutrition and Health Current Topics* 4, pp. 207–208 [T Carr and K Descheemaeker, editors]. Antwerp-Apeldoorn, Garant.
52. Reddy KS (2002) Cardiovascular diseases in the developing countries: dimensions, determinants, dynamics and directions for public health action. *Public Health Nutr* **5**, 231–237.
53. Eid N, Al-Hooti S, Boorisly N & Khalafawi M (1986) Nutritional anthropometry of school children in Kuwait. *Nutr Rep Int* **33**, 253–260.