Non-industrialised countries and affluence

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The prevalence of type 2 diabetes is rising rapidly in all non-industrialised populations. By 2025, three-quarters of the world’s 300 million adults with diabetes will be in non-industrialised countries, and almost a third in India and China alone. There is strong evidence that this epidemic has been triggered by social and economic development and urbanisation, which are associated with general improvements in nutrition and longevity, but also with obesity, reduced physical exercise and other diabetogenic factors. There is evidence too that fetal growth retardation and growth failure in infancy, both still widespread in non-industrialised populations, increase susceptibility to diabetes. An additional factor may be intergenerational effects of gestational diabetes occurring in mothers who grew poorly in early life and become obese as adults. Prevention of type 2 diabetes will require measures to promote exercise and reduce obesity in adults and children, alongside programmes to achieve healthy fetal and infant growth.

The rising prevalence of type 2 diabetes in non-industrialised populations

Standardised prevalence data for type 2 diabetes are available with a world-wide coverage probably unequalled for any other disease, allowing confident comparisons between people of similar ethnicity living in different settings, and giving a clear picture of secular trends in many populations over the last 20 years. The epidemiological story is remarkably consistent. An epidemic of diabetes is unfolding in countries undergoing rapid economic development and modernisation. Non-industrialised countries are exchanging their high morbidity from infectious disease for morbidity from ‘diseases of affluence’ including type 2 diabetes and cardiovascular disease.

The prevalence of type 2 diabetes is lowest among people who still have a ‘traditional’ or ‘primitive’ lifestyle as either hunter-gatherers or subsistence farmers. Examples are the Mapuche Indians in Chile, rural Bantu in Tanzania, and rural communities in the Pacific islands and South Asia. Even in these populations, it cannot be described as a ‘rare’ disease, affecting 1–3% of people aged 30–64 years. The prevalence is higher in people who have moved away from the traditional way of life,
either to live in towns and cities or through migration to another country. This has been described in all major ethnic groups (Fig. 1). Among South Asians, it is less than 5% in rural South India, around 12% in urban South India, and 15–20% in migrants to Mauritius, Fiji, Singapore, Tanzania, The Netherlands and the UK. Among Chinese, it ranges from less than 3% in rural China to 15–20% in urban Taiwan and Mauritius, and among people of African origin, from less than 3%
in Cameroon, to around 10% among people of West African descent living in Jamaica, and 15% in Jamaicans living in the UK.

The evidence is that high rates of disease in urban centres have arisen within a single generation. The largest increases are described in populations which have undergone the most rapid and extreme change, such as Ethiopian Jews who migrated to Israel, moving from severe malnutrition and a traditional way of life to a modern urban setting. They have a prevalence of 9% compared with 1–2% in Ethiopia itself\textsuperscript{13}. Another example is that of the Micronesian Nauruan islanders. Nauru suffered severe deprivation during Japanese occupation in World War II, but became wealthy from phosphate mining post-war\textsuperscript{14}. The Nauruans now have one of the highest rates of type 2 diabetes in the world (37% in the 25–74-year-old age group). Examples which may be more typical for large populations in non-industrialised countries, are Mauritius, where the prevalence has increased 30% in Asians, Creoles and Chinese between 1987 and 1998\textsuperscript{8}, Madras, South India, where it rose by 40% between 1988 and 1994\textsuperscript{15}, and China, where it is estimated to have increased 3-fold in the last 10 years\textsuperscript{16}.

Predictions for the future are worse. In 1998, King used the World Health Organization’s diabetes database to predict global rates of diabetes for the years 2000 and 2025, based on trends in population size, age structure and urbanisation\textsuperscript{17}. According to this analysis, the prevalence will continue to rise, by 30% world-wide, from 4.0% to 5.4%. The number of adults with diabetes will increase from 135 million in 1995 to 300 million in 2025. Although the prevalence will remain higher in industrialised countries, the proportional rise will be greater in non-industrialised countries (48%), and greatest in China (68%) and India (59%). Because of the large populations involved, 75% of the world’s adult diabetics will be in non-industrialised countries. India will have more people with diabetes (57 million) than any other country, followed by China (38 million). Unlike industrialised countries, where the highest number of people with diabetes will be in the oldest age groups, in non-industrialised countries this will be in the 45–64 year age group. These predictions agree closely with estimates made by others\textsuperscript{18,19}.

These estimates apply to type 2 diabetes in adults. An emerging problem, so far reported mainly in native American, African and Hispanic communities in the US, and in Japan, is type 2 diabetes in children and adolescents\textsuperscript{20}. Among blacks in Charleston, South Carolina and Hispanics in Ventura, California, 45% of new cases of diabetes in children are type 2. Among Japanese schoolchildren, type 2 diabetes is 7 times more common than type 1, and its incidence has increased 30-fold in the last 20 years. The likelihood is that type 2 diabetes in children will start to emerge in non-industrialised countries too.
The human and economic cost of diabetes

For many people in non-industrialised countries, the cost of even basic treatment for diabetes is crippling\textsuperscript{21,22}. Chale estimated annual treatment costs in 1992 at US$287 for those requiring insulin and US$103 for those on oral treatment. Home blood glucose monitoring once a week, which improves diabetic control, costs US$160 per year in Bangladesh\textsuperscript{23}. These costs represent 6–12 months’ wages for a labourer in the poorest non-industrialised countries. Chale’s paper ended with the words: ‘if African patients with diabetes have to pay for their treatment most will be unable to do so and will die’. Certainly many patients in poor countries will receive care well below ideal. In a nurse-led community programme in South Africa, home blood glucose monitoring was not available, and not even part of the equation. The target, woefully sub-optimal in modern terms, was simply freedom from symptoms of hyper- or hypo-glycaemia\textsuperscript{24}.

Good glycaemic control delays the onset of diabetic complications but demands that, in addition to early diagnosis and the availability of drugs and specialist medical care, the patient understands the disease. Poverty and lower levels of education in non-industrialised countries will almost certainly translate into worse disease\textsuperscript{25,26}. Many of the large number of people becoming diabetic in middle age will experience its chronic complications during their working lives. Access to treatment for diabetic retinopathy and renal disease, and prosthetic rehabilitation after amputation, are limited. Data from Africa and India show a high prevalence of micro- and macro-albuminuria\textsuperscript{27} and a more rapid progression to end-stage renal failure than in Western patients\textsuperscript{28}. In the Caribbean countries, a high proportion of surgical cases are patients with diabetic foot problems, and many lower-limb amputees remain permanently bed-ridden because they are not rehabilitated\textsuperscript{25}. There are few data from non-industrialised countries on mortality from diabetes, but a report from a tertiary referral centre in Kashmir, India suggested a 10 year reduction in life-span\textsuperscript{29}. The commonest causes of death were infection and chronic renal failure, unlike coronary heart disease and stroke, the leading causes of death among people with diabetes in industrialised countries.

Aetiology

Affluence and obesity

The epidemic of type 2 diabetes has been attributed to the ‘epidemiological transition’, a global trend away from traditional lifestyles and towards
urbanisation. In general, urbanisation (and migration) are associated with increased affluence or economic well-being and type 2 diabetes is thought to be a price to pay. This is a complex issue, however. Although early studies in white US and UK populations showed that people with greater affluence, education and social standing had a higher risk of diabetes, recent studies show the highest rates of disease in the most deprived sections of the community. The same sequence may be repeating itself in non-industrialised countries. Currently, the well-off get more disease. Type 2 diabetes appears to be a disease of newly affluent more than ‘established’ affluent populations. Consistent with this, while white populations of European origin have moderately high rates of type 2 diabetes (5–10%), which are also rising, their epidemic has been slower and less extreme.

An important feature of the epidemiological transition is obesity. There is a clear association between obesity, which increases insulin resistance, and type 2 diabetes. In all populations, the prevalence of diabetes increases with increasing body mass index. Research from industrialised countries shows that the greater the duration of obesity the higher the risk of diabetes, and that obesity starting in childhood is a risk factor. Therefore, the worldwide trend towards obesity, in both adults and children, is a cause for concern. Rates of childhood obesity remain low in some non-industrialised countries such as India (< 1%), but with increasing prosperity have risen to more than 4% in Mauritius, Bolivia and Iran, and more than 10% in Chile and Jamaica. In many non-industrialised populations, this is not perceived as a health problem, indeed quite the opposite. Obesity is often seen as a symbol of health, beauty and status, reflecting a man’s ability to provide for his family and a woman’s skill as a mother and cook.

Despite its powerful effect on risk, obesity does not fully account for the increase in type 2 diabetes in non-industrialised communities, where diabetes occurs commonly at levels of body mass index considered healthy in industrialised countries. This may be partly explained by the distribution of body fat. Central (abdominal) obesity, a characteristic of South Asian populations, is more closely linked with type 2 diabetes than generalised or peripheral obesity.

**Reduced physical activity**

Clinical studies show that exercise increases insulin sensitivity and glucose tolerance. The prevalence of type 2 diabetes is higher in more sedentary people, and individuals with the disease are less active than those without. Populations with low rates of type 2 diabetes are characterised by the high levels of physical activity associated with hunting and gathering or farming. Urbanisation and migration
frequently lead to a more sedentary way of life: food and fuel come from shops just down the road, water is on tap and does not have to be carried from the well, there is more labour-saving technology in the home, bicycles are replaced by motorbikes and cars, and work is in offices and factories. Physical activity declines among children too, because of access to television and computer games, and ‘hot-house’ studying in countries where entry to limited places in higher education is subject to intense competition. Exercise as a healthy leisure activity is a recent Western concept; there are often strong climatic, economic and cultural factors discouraging exercise among urban populations in non-industrialised countries as well as migrants from these countries44,53.

Poor nutrition: caloric excess and micronutrient deficiency

Himsworth showed reduced mortality and hospital admissions for type 2 diabetes in the UK during periods of wartime food rationing when calorie intakes decreased54. Differences in fat intakes correspond well with population differences in the prevalence of type 2 diabetes31. On the other hand, prospective studies looking for dietary determinants of diabetes (so far confined to industrialised countries) have failed to show a clear link between carbohydrate or fat intakes and incidence31,55,56. Micronutrient deficiency may play a role in susceptibility to disease. Boucher has described an association between vitamin D deficiency and impaired insulin secretion and type 2 diabetes57. Although more data are required, there is some evidence too that omega-3 fatty acids, found in green leafy vegetables, nuts, vegetable oils and fish, may protect against a number of cardiovascular risk factors including diabetes31,58. Both calorie excess and poor dietary quality are features of urbanisation and migration, especially among the poor, who buy highly refined, energy-dense food, while the better-off can afford a healthier mixed diet59,60.

Socio-economic inequality and stress

Workers seeking a better life in foreign places often work in menial jobs, have low incomes, live in poor housing, have difficulty communicating, are exposed to crime and aggression, have reduced access to health care and lack traditional family support mechanisms61. Associations between social inequality and poor health are well-recognised62, although poorly understood, and social inequality has been linked to type 2 diabetes and the metabolic syndrome35. Possible mechanisms are increased inflammatory cytokines or stimulation of the hypothalamo–pituitary–cortical axis62.
Environmental toxins

Cassava and other cyanide-containing foods were once thought to be a cause of diabetes in tropical countries. This is not supported by recent evidence, although it remains possible that micronutrient undernutrition or diets low in anti-oxidants, enhance the effects of such environmental toxins. Cities in many non-industrialised countries are chemically polluted, and this has been cited as a possible risk factor for diabetes, acting through the production of inflammatory cytokines.

Genes

Finally, the susceptibility to diabetes of populations in non-industrialised countries has been attributed to genes. According to Neel’s hypothesis, a ‘thrifty genotype’ may have enhanced survival in subsistence conditions in the past, but becomes detrimental in a modern urban setting of plentiful food and reduced physical work. Zimmet has proposed that thrifty genes promote fat storage, perhaps mediated by leptin resistance, providing a survival advantage during periods of starvation. It has also been suggested that the tendency to store fat centrally, a feature of South Asian Indian populations, may have a genetic basis. Central body fat, more metabolically active than peripheral fat and less likely to impede locomotion, may have evolved as a site for quick storage and mobilisation in time of need. Though their existence is plausible, these genes have not so far been identified.

Early-life origins of type 2 diabetes

Epidemiological studies in the UK, Europe and the US have shown that men and women who were small at birth, with a low birth weight, are at increased risk of developing cardiovascular disease, hypertension, type 2 diabetes and the metabolic syndrome in adult life. Their findings have led to the ‘thrifty phenotype’ hypothesis (see elsewhere in this issue) which proposes that fetal undernutrition, occurring at critical periods in the development of pancreatic islet cells and insulin-sensitive tissues such as muscle and liver, has permanent metabolic consequences, including life-long insulin resistance. The epidemiological data suggest that post-natal events modify the risk associated with low birth weight. For example, growth failure in infancy, and the later development of obesity, add to the risk.

Intra-uterine growth retardation and failure to thrive in infancy are common in non-industrialised countries, probably largely due to
maternal stunting and undernutrition. In India, the mean full-term birth weight is 2.6–2.7 kg, almost 1 kg lower than in Western Europe, and 25% of full-term babies are born low birth weight (<2.5 kg). Most studies linking size at birth with later disease come from industrialised countries, but some data are available from India and China. In India, among children born in the KEM Hospital, Pune, those of lower birth weight were more insulin resistant at the age of 8 years. They showed other features of the metabolic syndrome: higher blood pressure and subscapular/triceps skinfold ratios and lower HDL-cholesterol concentrations. The highest levels of insulin resistance were in children who were small at birth but had a high fat mass at 8 years (Fig. 2). There was a statistically significant interaction between birth weight and 8-year fat mass; the effect of low birth weight was greatest in children with the highest fat mass at 8 years, and the effect of increased 8-year fat mass greatest in children of low birth weight (Fig. 2). Low birth weight was not associated with impaired glucose tolerance at this age, and it is not yet known if insulin resistance in childhood persists into adult life or predicts future diabetes. However, these data suggest that low birth weight leads to insulin resistance when combined with later obesity, and that better intra-uterine growth may protect against the adverse effects of obesity.

Table 1 shows data from studies of young adults in China and India. Among men and women aged 41–47 years born in Beijing, lower birth weight was associated with higher fasting and 2-h plasma glucose and insulin concentrations in an oral glucose tolerance test, and features of the metabolic syndrome including raised systolic blood pressure, higher serum triglyceride concentrations and lower serum HDL-cholesterol.
Glucose and insulin concentrations were also inversely related to the mother’s body mass index in pregnancy; offspring of thinner mothers were more glucose intolerant and insulin resistant. In a study of young adults (mean age 29 years) born in the Holdsworth Memorial Hospital, Mysore, South India, lower birth weight was associated with higher 2-h glucose concentrations, higher rates of type 2 diabetes and impaired glucose tolerance, and higher serum triglyceride concentrations (Table 1). These findings are similar to those in Western populations and support the ‘thrifty phenotype’ hypothesis.

A study of older Indian adults (aged 45–65 years) born in Mysore showed somewhat different results. Although lower birth weight subjects had higher fasting insulin concentrations, they did not have higher rates of type 2 diabetes. The prevalence of diabetes was increased in men and women who were short but relatively heavy at birth, with a high ponderal index (birth weight/birth length; Fig. 3A). This group of men and women were characterised by a low 30-min insulin increment, a marker of reduced first phase insulin secretion, even when those with diabetes were excluded (Fig. 3B). There were also surprising associations with maternal weight and pelvimetry; the prevalence of diabetes was increased in offspring of mothers with a higher body weight and larger pelvic intercristal and interspinous external diameters.

<table>
<thead>
<tr>
<th>Birth weight (kg)</th>
<th>n</th>
<th>2-h glucose (mmol/l)</th>
<th>Systolic blood pressure (mmHg)</th>
<th>Serum triglycerides (mmol/l)</th>
<th>Fasting insulin (pmol/l)</th>
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<td>≤ 2.5</td>
<td>44</td>
<td>7.9</td>
<td>128</td>
<td>1.77</td>
<td>46</td>
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<td>115</td>
<td>6.1</td>
<td>122</td>
<td>1.02</td>
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<tr>
<td>All</td>
<td>627</td>
<td>6.6</td>
<td>125</td>
<td>1.26</td>
<td>43</td>
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Table 1 2-h plasma glucose concentrations and other components of the insulin resistance syndrome according to birth weight

P value adjusted for age, sex, and adult body mass index.

Fall CHD & Veena SR, unpublished data
These findings are clearly different from studies in the West and in the younger Pune, Mysore and Beijing cohorts. The link with higher maternal weight and higher ponderal index at birth suggested to us that another factor causing type 2 diabetes in this population may be gestational diabetes, which is associated with maternal obesity and leads to fatter (‘macrosomic’) babies. In poor communities in India, mothers are often stunted and undernourished, and their babies are born small and thin. According to the ‘thrifty phenotype’ hypothesis, these babies are at risk of developing insulin resistance in childhood and adult life, especially if their circumstances in later life allow them to become obese. If they are female, their insulin resistance will be further exacerbated in pregnancy, and may lead to gestational diabetes. There is a growing evidence that women who experienced deprivation in early life, indicated by low birth weight\textsuperscript{72–75} and/or short stature\textsuperscript{76–78} have an increased risk of developing this complication of pregnancy.

**Effects of maternal gestational diabetes**

It is well-established that the offspring of mothers with diabetes in pregnancy are at increased risk of developing adult type 2 diabetes. This has been most clearly shown among the US Pima Indians, who have high rates of gestational diabetes (Fig. 4A)\textsuperscript{79}, and recently confirmed in white Caucasians in the US Nurse’s Study\textsuperscript{80}. Among the Pima Indians, 70% of people with prenatal exposure to a diabetic environment were diabetic at 25–34 years of age (Fig. 4A)\textsuperscript{79}. This phenomenon is probably environmentally rather than genetically mediated; mothers who were prediabetic, and developed diabetes at some time after their pregnancy,
did not transmit this high risk to their offspring (Fig. 4A). This is supported by animal studies in which induction of diabetes or hyperglycaemia in mother rats using streptozotocin or glucose infusion leads to insulin resistance, deficits in insulin secretion, and diabetes in the offspring\textsuperscript{81,82}.

The importance of gestational diabetes as a factor in the epidemic of type 2 diabetes in non-industrialised countries is not known, and there are few recent data on the incidence of gestational diabetes. In her world-wide review, King cites low rates of 3.5\% in Karachi, Pakistan, 0.6\% in Madras, India and 0.6\% in Taipei, China\textsuperscript{83}. A recent prospective study in Mysore, India, however, showed a much higher incidence of 6\%\textsuperscript{78}. The babies born in this study are being followed up to determine the effects on their glucose/insulin metabolism. Rates of gestational diabetes are known to be high among women from non-industrialised countries who have migrated to more affluent countries. In Melbourne, Australia, the prevalence was 15\% among Asian women, 14\% among Chinese and 10\% among women of African origin, compared with only 5\% among white Caucasian women. Similarly, in London, UK, 6\% of Asian mothers and 3\% of Afro-Caribbean mothers developed gestational diabetes compared with 1\% of white Caucasian women\textsuperscript{83}. It seems likely that gestational diabetes is playing a role in fuelling the rise in type 2 diabetes that occurs with the epidemiological transition in non-industrialised countries. Figure 5 presents a model to explain how increasing prosperity and urbanisation, in populations made insulin resistant by generations of fetal malnutrition, leads to the appearance of gestational diabetes and a rising epidemic of type 2 diabetes.

Both intra-uterine undernutrition and maternal diabetes may lead to obesity and hence increased risk of type 2 diabetes. Boys exposed \textit{in utero} to the Dutch famine were at increased risk of obesity in early adult life\textsuperscript{84}. Low birth weight has been shown to be associated with central

![Graph A](image1)

**Fig. 4** Prevalence of (A) type 2 diabetes and (B) severe obesity in offspring of non-diabetic, prediabetic and diabetic women: data from the Pima Indians\textsuperscript{79}.
obesity in studies of adults and children in the UK and India. At birth, Indian babies are lighter than UK babies, but have similar amounts of truncal body fat, measured by subscapular skinfold thickness, suggesting fat preservation despite growth retardation. It is not yet known whether this persists post-natally. The Pima Indian studies have shown that maternal gestational diabetes is also associated with a high risk of obesity in the next generation, especially in childhood and young adult life (Fig. 4B).

Prevention of type 2 diabetes

Despite clearly identified modifiable environmental risk factors for type 2 diabetes, surprisingly little is done about its prevention in Western countries compared, for example, with coronary heart disease. This may be because relatively few people develop diabetes, those who do can generally afford to be treated, and major complications tend to develop only late in life. If the predictions hold true, however, and prevalence rates of 10–20% become typical in urban communities in non-industrialised countries, the situation will be very different and impossible to ignore. Preventive measures are urgently needed.
Among US Hispanics and Japanese, the high prevalence of type 2 diabetes compared with white Caucasians is most marked in people of low education.32,33 Despite its association with higher income and socio-economic status, studies from non-industrialised countries have also identified lack of education as a risk factor for diabetes.16,88 Stern has predicted a ‘descending limb’ to epidemics of the disease, as people become better educated, learn to eat better, avoid obesity and lead more active lives.33 In an important and so far unique test of this, Pan conducted a randomised, controlled trial of diet and/or exercise in men and women with impaired glucose tolerance living in Da Qing, China.89 Subjects were identified by screening 110,660 people, and randomised by centre to receive advice on diet, exercise, both or neither (control group). Dietary advice included detailed recommendations about intakes of carbohydrate, fat, protein, vegetables and alcohol and achieving a target body mass index. People in the exercise groups were advised to increase their activity by at least one ‘unit’ per day (30 min of slow walking, 20 min of fast walking, 10 min of strenuous exercise (e.g. running) or 5 min of very strenuous exercise (e.g. swimming or skipping). Advice was given one-to-one by a physician, and later in groups, and continued regularly for 6 years of follow-up.

During 6 years, the incidence of diabetes was 68% in the control group, but significantly lower (40–50%) in all three intervention groups (Fig. 6A), despite no effect on mean body mass index (Fig. 6B). Dietary advice was as
effective as exercise, and there was no apparent benefit of combining the two. The subjects, most of whom migrated to Da Qing from all over China to work in the oil industry are probably representative of many populations undergoing the epidemiological transition in the non-industrialised world. This study is important in showing that intervention is possible and effective, though potentially expensive on a large scale. It needs to be replicated, and further studies are needed to determine whether primary intervention, when glucose tolerance is still normal, or in childhood, is effective. Enough is known, however, to recommend strongly the promotion of exercise and avoidance of obesity in non-industrialised populations.

The ‘thrifty phenotype’ hypothesis would predict that diabetes will recede naturally when nutrition improves sufficiently and for enough time (probably at least one generation) to lead to improvements in fetal nutrition and growth. That this could happen is supported by animal experiments. Sand rats transferred from a wild to a caged environment become obese and diabetic. The effects are worst in the generation directly transferred and become milder, then disappear, in subsequent generations. So far, there has been only one report of a fall in the incidence of diabetes in a human population, the Nauruan islanders. Their age-specific prevalence fell from 28% in 1975/1976 to 24% in 1987, despite no reduction in obesity or increase in physical activity. The authors of this report suggested that the decline was due to reduced fertility in women with a strong genetic tendency for and, therefore early onset of, type 2 diabetes. An alternative explanation is that prosperity has led to improved fetal nutrition and thus to individuals who are less susceptible to diabetes.

The potential to prevent type 2 diabetes can been added to a long list of reasons to recommend the promotion of healthy fetal and infant growth in non-industrialised countries. There are large gaps in scientific knowledge as to the best ways of achieving this. Enough is known, however, to recommend improvements in the nutrition and growth of (female) infants, children and adolescents, encouraging the delaying of childbearing until the mother’s own growth is complete, and promoting adequate maternal intakes of energy, protein and micronutrients during pregnancy itself. There is sufficient evidence of long-term adverse effects of gestational diabetes on the offspring to recommend that in populations with adequate energy intakes mothers, especially those who are stunted and at increased risk of gestational diabetes, should avoid becoming obese. Finally, screening for and intensive management of gestational diabetes should probably become more rigorous.

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