Global and societal implications of the diabetes epidemic

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Changes in human behaviour and lifestyle over the last century have resulted in a dramatic increase in the incidence of diabetes worldwide. The epidemic is chiefly of type 2 diabetes and also the associated conditions known as 'diabesity' and 'metabolic syndrome'. In conjunction with genetic susceptibility, particularly in certain ethnic groups, type 2 diabetes is brought on by environmental and behavioural factors such as a sedentary lifestyle, overly rich nutrition and obesity. The prevention of diabetes and control of its micro- and macrovascular complications will require an integrated, international approach if we are to see significant reduction in the huge premature morbidity and mortality it causes.

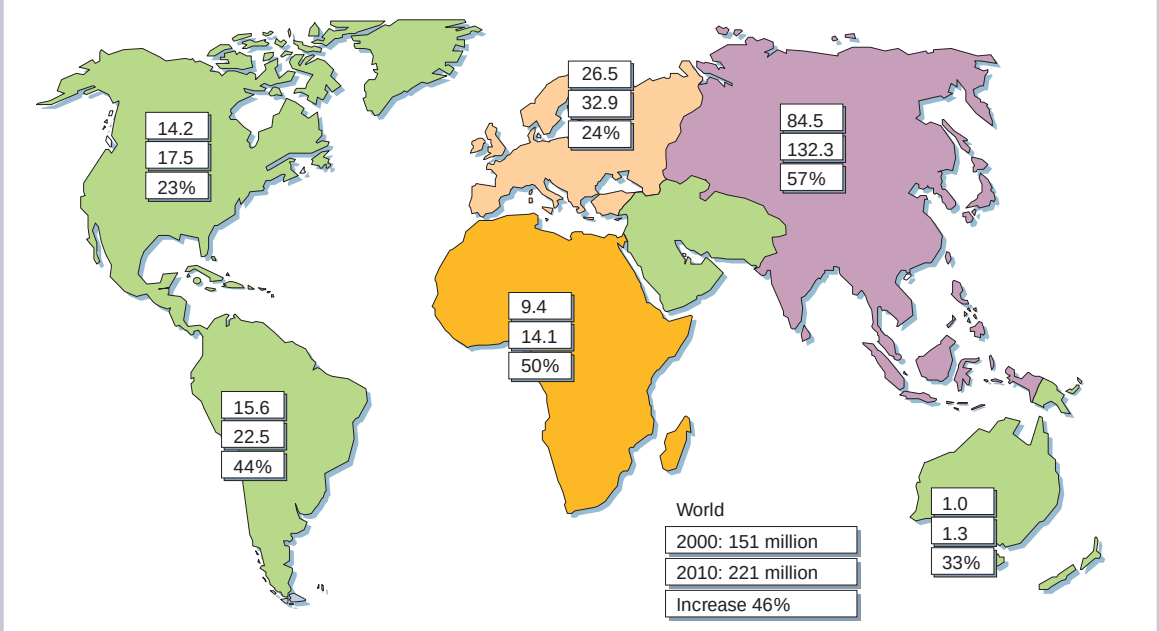
"Man may be the captain of his fate, but he is also the victim of his blood sugar"
Wilfrid Oakley [*Trans. Med. Soc. Lond.* **78,** 16 (1962)]

Diabetes mellitus, long considered a disease of minor significance to world health, is now taking its place as one of the main threats to human health in the 21st century[1]. The past two decades have seen an explosive increase in the number of people diagnosed with diabetes worldwide[2,3]. Pronounced changes in the human environment, and in human behaviour and lifestyle, have accompanied globalization, and these have resulted in escalating rates of both obesity and diabetes. Hence the recent adoption of the term 'diabesity'[4], first suggested by Shafrir several decades ago[5].

There are two main forms of diabetes[6]. Type 1 diabetes is due primarily to autoimmune-mediated destruction of pancreatic β-cell islets, resulting in absolute insulin deficiency. People with type 1 diabetes must take exogenous insulin for survival to prevent the development of ketoacidosis. Its frequency is low relative to type 2 diabetes, which accounts for over 90% of cases globally. Type 2 diabetes is characterized by insulin resistance and/or abnormal insulin secretion, either of which may predominate. People with type 2 diabetes are not dependent on exogenous insulin, but may require it for control of blood glucose levels if this is not achieved with diet alone or with oral hypoglycaemic agents.

The diabetes epidemic relates particularly to type 2 diabetes, and is taking place both in developed and developing nations[7]. Paradoxically, part of the problem relates to the achievements in public health during the 20th century, with

Figure 1 Numbers of people with diabetes (in millions) for 2000 and 2010 (top and middle values, respectively), and the percentage increase. Data adapted from ref. 2.



people living longer owing to elimination of many of the communicable diseases[8]. Non-communicable diseases (NCD) such as diabetes and cardiovascular disease (CVD) have now become the main public health challenge for the 21st century, as a result of their impact on personal and national health and the premature morbidity and mortality associated with the NCDs[1].

After taking so long to gain recognition, interest in diabetes is now mounting rapidly[7] and it is an exciting time for researchers and clinicians involved in the study and treatment of the disease. The problem has crept up on an unsuspecting public health community. The global figure of people with diabetes is set to rise from the current estimate of 150 million to 220 million in 2010, and 300 million in 2025 (Fig. 1)[2,3]. Most cases will be of type 2 diabetes, which is strongly associated with a sedentary lifestyle and obesity[7]. This trend of increasing prevalence of diabetes and obesity has already imposed a huge burden on health-care systems and this will continue to increase in the future[1,9].

Although type 2 diabetes is numerically more prevalent in the general population, type 1 diabetes is the most common chronic disease of children. But with the increasing prevalence of type 2 diabetes in children and adolescents, the order may be reversed within one to two decades[10,11].

Prevention of complications

Prevention of complications is a key issue because of the huge premature morbidity and mortality associated with the disease[1,12]. In the past decade, several major studies have focused attention on the need for strict control of glycaemia to prevent and/or reduce the risk of both the specific microvascular and the less specific macrovascular complications[7].

The Diabetes Control and Complications Trial[13] was a landmark study and the flagship for a number of studies that established the value of intensive control of blood glucose to prevent the retinal, renal and neuropathic complications of diabetes. The United Kingdom Prospective Diabetes Study (UKPDS)[14] fulfilled the same role for type 2 diabetes. Subsequently, there were other important studies that underline the importance of active medical intervention (including control of blood pressure and lipids as well as glucose) for the reduction of the risk of diabetes complications. This applies to micro vascular complications, as shown, for example, by the Stockholm[15], MICRO-HOPE[16] and Kumamoto studies[17], and to macrovascular disease from the 4S[18], CARE[19] and MICRO-HOPE[16] studies.

The concealed burden of impaired glucose tolerance

Type 2 diabetes is increasingly common, indeed epidemic, primarily because of increases in the prevalence of a sedentary lifestyle and obesity[20]. The possibility of preventing type 2 diabetes by interventions that affect the lifestyles of subjects at high risk for the disease is now the subject of a number of studies; these have focused on people with impaired glucose tolerance (IGT)[21–23]. IGT is defined as hyperglycaemia (with glucose values intermediate between normal and diabetes) following a glucose load (ref. 6 and Table 1), and affects at least 200 million people worldwide. It represents a key stage in the natural history of type 2 diabetes as these people are at much higher future risk than the general population for developing diabetes[24,25]. Approximately 40% of subjects progress to diabetes over 5–10 years, but some revert to normal or remain IGT.

Subjects with IGT also have a heightened risk of macrovascular disease[25–29]. Because of this, and the association with other known CVD risk factors including hypertension, dyslipidaemia and central obesity[6,30], the diagnosis of IGT, particularly in apparently healthy and ambulatory individuals, has important prognostic implications[29,31].

Impaired fasting glucose (IFG) was introduced recently as another category of abnormal glucose metabolism[6,32]. It is defined on the basis of fasting glucose concentration (Table 1) and, like IGT, it is associated with risk of CVD and future diabetes. The American Diabetes Association had hoped that their recommendation of the adoption of IFG[32] would simplify the screening for at-risk individuals, with the need for an oral glucose-tolerance test being circumvented. However, the desired outcome has yet to be achieved as IGT is a better predictor than IFG of risk of future diabetes and of mortality[22,27,28,33].

Table 1 Values for diagnosis* of diabetes and other types of hyperglycaemia

	Glucose concentration (mmol l^{-1})			
	Plasma		Whole blood	
	Venous	Capillary	Venous	Capillary
Diabetes mellitus				
Fasting	⩾7.0	⩾7.0	⩾6.1	⩾6.1
2-h post-glucose load	⩾11.1	⩾12.2	⩾10.0	⩾11.1
Impaired glucose tolerance				
Fasting concentration	<7.0	<7.0	<6.1	<6.1
2-h post-glucose load	7.8–11.0	8.9–12.1	6.7–9.9	7.8–11.0
Impaired fasting glucose				
Fasting	6.1–6.9	6.1–6.9	5.6–6.0	5.6–6.0
2-h post-glucose load	<7.8	<8.9	<6.7	<7.8

*Note that diabetes can be diagnosed in an individual only when these diagnostic values are confirmed on another day. Data from ref. 24.
Ranges of values are inclusive (that is, 6.1–6.9 means ⩾6.1 and <7.0).

Type 2 diabetes and the metabolic syndrome

Type 2 diabetes is a multifactorial disease that shows heterogeneity in many respects[34]. Our understanding of the disease and related disorders such as IGT and IFG is undergoing a radical change, particularly as data suggest that the risk of complications commences many years before the onset of clinical diabetes[30,35]. Previously, it was regarded as a relatively distinct disease entity, but in reality, type 2 diabetes (and its associated hyperglycaemia or dysglycaemia) is often a manifestation of a much broader underlying disorder[7,36]. This includes the metabolic syndrome (sometimes called syndrome X; Fig. 2) — a cluster of CVD risk factors that, in addition to glucose intolerance (that is, IGT or diabetes), includes hyperinsulinaemia, dyslipidaemia, hypertension, visceral obesity, hypercoagulability and microalbuminuria. This new paradigm relating to type 2 diabetes also influences contemporary therapy for the disease[7]. Evidence now exists for a far more aggressive approach to treating not just the hyperglycaemia, but also other CVD risk factors such as hypertension, dyslipidaemia and central obesity in type 2 diabetic

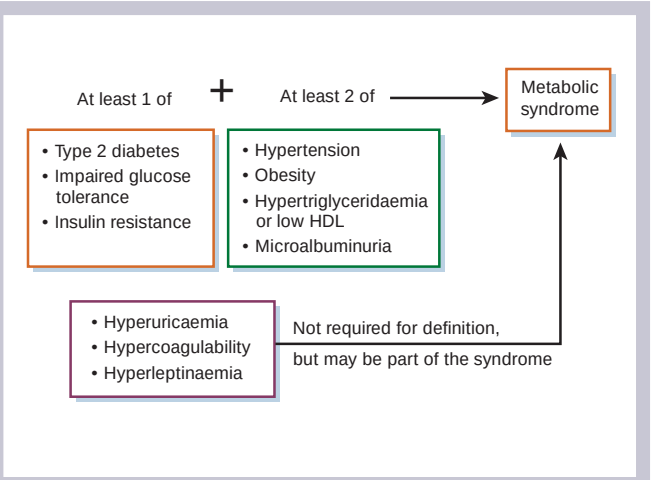


Figure 2 Metabolic syndrome as defined by the World Health Organization[24]. Insulin resistance is defined as being within the highest quartile for the relevant population. Hypertension is defined as blood pressure ⩾140/90. Obesity is defined as a body-mass index ⩾30 kg m^{-2}, or a waist–hip ratio (WHR) > 0.90 for males and WHR > 0.85 for females. Hypertriglyceridaemia is defined as ⩾1.7 mmol triglycerides l^{-1}. Low high-density lipoprotein (HDL) is defined as < 0.9 mmol l^{-1} for men; < 1.0 mmol l^{-1} for women. Microalbuminuria is a urinary albumin excretion rate ⩾20 μg min^{-1} or albumin creatinine ratio ⩾30 mg min^{-1}.

patients, with the hope of significantly reducing cardiovascular morbidity and mortality.

There is continuing debate as to the primary aetiological factor for the syndrome. Genetic factors, visceral obesity, insulin resistance and endothelial dysfunction may all contribute either solely or jointly[36,37]. Irrespective of the aetiologic debate, a recent report from the WHO[6] has highlighted both the need and the importance of a consistent definition of the metabolic syndrome and has suggested parameters. Although the criteria may change as new prospective data become available, this initiative provides a basis for the development of a standardized definition that will allow international comparisons of prevalence, incidence and natural history, as previously there was no internationally agreed definition.

The reliability of this definition in terms of predicting the prevalence of, and the CVD risk associated with, the metabolic syndrome has been reported recently[38]. Cardiovascular mortality was assessed in 3,606 subjects from the Botnia study (a large-scale study of type 2 diabetes begun in Finland in 1990) with a median follow-up of 6.9 years. In women and men, respectively, the metabolic syndrome was recorded in 10 and 15% of subjects with normal glucose tolerance, 42 and 64% of those with IFG/IGT, and 78 and 84% of those with type 2 diabetes. The risk for coronary heart disease and stroke was increased threefold in subjects with the syndrome, and cardiovascular mortality was markedly increased (12.0% in subjects with the syndrome versus 2.2% in those without; $P < 0.001$). This study confirms the hope of the WHO report[6] that the WHO definition of the metabolic syndrome will identify subjects with increased CVD morbidity and mortality. It offers a tool for comparison of results from different studies.

Diabetes in children and youth

Type 2 diabetes in children, teenagers and adolescents is a serious new aspect to the epidemic and an emerging public health problem of significant proportions[10,11,39]. Although type 1 diabetes remains the main form of the disease in children worldwide, it seems possible that type 2 diabetes will be the predominant form within ten years in many ethnic groups and potentially in Europid groups (that is, of European descent). Type 2 diabetes has already been reported in children from Japan, the United States, Pacific Islands, Hong Kong, Australia and the United Kingdom[10,39–41]. Among children in Japan, it is already more common than type 1 diabetes, accounting for 80% of childhood diabetes; the incidence almost doubled between 1976–80 and 1991–5 (ref. 42).

The rising prevalence of obesity and type 2 diabetes in children is symptomatic of the effects of globalization and industrialization affecting all societies[1], with sedentary lifestyle and obesity[11] the predominant factors involved. As a result of this new and alarming scenario, a joint consensus statement has been issued recently by the American Diabetes Association and the American Academy of Pediatrics[40]. We now urgently need epidemiological data to outline the extent of the problem, as most of the reports so far are clinic based[11,39].

Although type 2 diabetes in Europeans is usually characterized by onset (often asymptomatic) after 50 years of age, in Pacific islanders and other high-prevalence groups such as southern Asians, onset in the 20–30-year age group is now increasingly common[7,39]. The socioeconomic and public health impact of this downward shift in disease onset on society is much greater through effects on the work force and premature morbidity and mortality, as well as through the negative impact on fertility and reproduction[43].

The appearance of type 2 diabetes in a younger age group also raises new issues in classification of diabetes. For example, how do we differentiate type 2 diabetes in children from type 1? Certainly, the presence of C-peptide and absence of markers of autoimmunity such as antibodies to glutamic acid decarboxylase may help to diagnose type 2 diabetes[44]. What therapies are safe in this age group? Most pharmacological therapies for diabetes and its associated conditions (apart from insulin) are not approved for use in children and the same applies for those for blood pressure and dyslipidaemia[11,40]. How will we tackle the problem of diabetes complications at an earlier age? All of these issues need to be addressed urgently, and information on behavioural and environmental factors will be required to plan intervention strategies.

Table 2 Aetiological determinants and risk factors of type 2 diabetes

Genetic factors
Genetic markers, family history, 'thrifty gene(s)'
Demographic characteristics
Sex, age, ethnicity
Behavioural- and lifestyle-related risk factors
Obesity (including distribution of obesity and duration)
Physical inactivity
Diet
Stress
'Westernization, urbanization, modernization'
Metabolic determinants and intermediate risk categories of type 2 diabetes
Impaired glucose tolerance
Insulin resistance
Pregnancy-related determinants (parity, gestational diabetes, diabetes in offspring of women with diabetes during pregnancy, intra-uterine mal - or overnutrition)

The main factors in the type 2 diabetes epidemic

The diabetes epidemic, although apparent right around the world, has been most pronounced in non-Europid populations, as evidenced by studies from Native American and Canadian communities, Pacific and Indian Ocean island populations[45], groups in India[46] and Australian Aboriginal communities[47]. In the Pacific island of Nauru, where diabetes was virtually unknown 50 years ago, it is now present in approximately 40% of adults[48]. The potential for increases in the number of cases of diabetes is greatest in Asia[2]. Data from Mauritius show the highest yet reported prevalence in people of Chinese extraction, in addition to demonstrating a high diabetes prevalence and a notable secular increase between 1987 and 1998 in Asian Indians and Creoles[7]. Together with evidence that prevalence of type 2 diabetes doubled between 1984 and 1992 in Singaporean Chinese[49], and with the high prevalence in Taiwan[50], these data provide alarming indicators of the size of the future epidemic in the People's Republic of China. Here, the overall prevalence of type 2 diabetes was, until recently, less than 1%. Recent studies show a threefold increase in prevalence in certain areas of China within the past two decades[51]. The scenario is highlighted in Fig. 3 where the prevalence

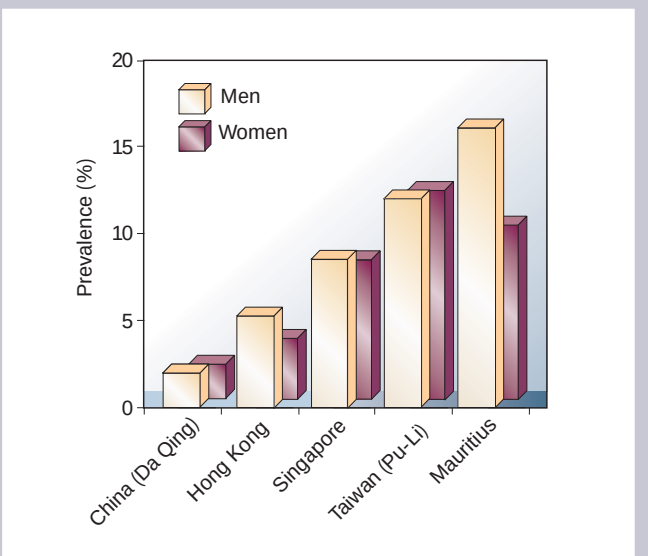


Figure 3 The prevalence of type 2 diabetes mellitus among Chinese in Hong Kong, Singapore, Taiwan and Mauritius, compared with that in the People's Republic of China[6].

of diabetes in Chinese populations in China, Singapore, Taiwan and Mauritius is compared. If China were to experience just one-half of the current rate of diabetes in Taiwan, the number of individuals with diabetes will increase from 8 million in 1996 to over 32 million by 2010. The data from the Indian subcontinent are equally disconcerting[7,46].

What are the reasons for this epidemic? Apart from the heightened genetic susceptibility of certain ethnic groups, environmental and behavioural factors such as sedentary lifestyle, nutrition and obesity (Table 2) are clearly important[52]. One of the major debates in diabetes and NCD aetiology is the issue of thrifty genotype versus thrifty phenotype[7]. In one corner are those who postulate that humans are genetically programmed for the hunter–gatherer era. The 'thrifty genotype' hypothesis[53] provides an explanation of the very high prevalence of obesity and type 2 diabetes in the American Pima Indians, Australian Aborigines and Pacific Islanders — the basis for the susceptibility to diabesity could be the result of an evolutionary advantageous thrifty genotype that promoted fat deposition and storage of calories in times of plenty. This mechanism would have conferred a survival advantage during the regular famines and natural disasters that were interspersed with feast periods. With Westernization, these populations now have a plentiful supply of a diet with an excess of energy, simple carbohydrates and saturated fats. This has been accompanied by a reduction in both occupational and leisure-based physical activity. Both factors may therefore cause the previously favourable metabolic profile seen in 'survivors' to become a handicap, which results in obesity and type 2 diabetes[54].

There exists an excellent model of this phenomenon — the Israeli sand rat (*Psammomys obesus*)[7]. When this animal is removed from the sparse diet of its natural environment and given an abundant, high-calorie diet, it develops all of the components of the metabolic syndrome, including diabetes and obesity. Recently, several new obesity- and diabetes-related genes have been isolated and sequenced from *P. obesus*, including the beacon gene[55]. Intracerebroventricular infusion of beacon protein results in a dose-dependent increase in food intake and body weight and an increase in hypothalamic expression of neuropeptide Y, a brain peptide that stimulates food intake.

In the other corner sit the supporters of the thrifty phenotype hypothesis, which is based on the epidemiological observations linking low birth weight with the risk of adult disease (obesity, diabetes and hypertension). Hales and Barker have hypothesized[56] that intrauterine malnutrition leads to reduced birth size and to permanent changes in structure and function, which predispose to disease in adult life. Although accepting the association between low birth weight and subsequent disease, a number of authors have questioned the 'environmental' interpretation, pointing out that, paradoxically, the data could be consistent with the thrifty genotype scenario[7]. Given the strong evidence for a role for genes in type 2 diabetes, it is also possible that the surviving low-birth-weight babies are actually an example of the thrifty gene operating — fetuses with low birth weight that survive are the example of a 'survival of the fittest' gene in an environment of intrauterine malnutrition[57]. Because they have the thrifty genotype, these survivors would also be at risk of disease in later life.

More detailed reviews on this debate and a number of related issues are available elsewhere[57,58]. The reviewers point out that claiming causal inference in this association must be seriously challenged. Programming studies are unique in that potential causes are temporally separated from effects by a span of some five decades. Joseph and Kramer[59] list various direct and indirect evidence that suggests the reported association of low birth weight and the later development of type 2 diabetes may be biased rather than causal. They state: "…selection bias, failure to define, measure, and adequately control for the confounding health consequences of social deprivation and inconsistencies in the hypotheses tested and in methods of data analysis and reporting are among the factors that weigh against a causal explanation for the associations observed". Even if the relationship is shown ultimately to be causal, low birth weight explains only a small proportion of diabetes, and does not exclude the importance of other hypotheses (such as the thrifty genotype).

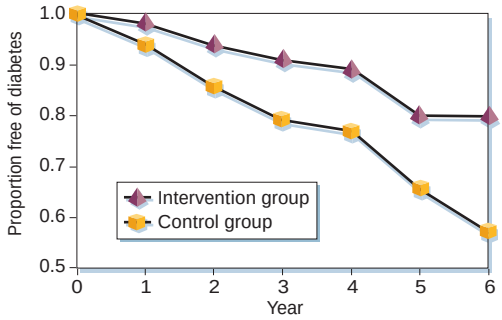


Figure 4 Reduction in risk of progressing from IGT to diabetes as a result of changes in intensive lifestyle. Data adapted with permission from ref. 22.

Approaches to prevention of type 2 diabetes

Although focusing and directing funds at treating diabetes and its complications is important, the rapid escalation of numbers of people with diabetes demands urgent action on prevention[60,61]. If not, the economic costs of premature morbidity and mortality from diabetes could absorb much of the health care budgets of both developing and affluent nations[1,60]. Imagine the potential crisis in Asia, which will probably be home to 61% of the total global projected number of people with diabetes by 2010.

Recent studies have highlighted the potential for intervention in IGT subjects to reduce progression to type 2 diabetes. One such study is the recently completed Diabetes Prevention Program in the United States[62]. This showed that over three years, lifestyle intervention (targeting diet and exercise) reduced the risk of progressing from IGT to diabetes by 58%, whereas the oral hypoglycaemic drug, metformin, reduced risk by 31% (ref. 63). Two other large-scale studies — from Da Qing, China[21], and from Finland[22] — have also demonstrated the efficacy of lifestyle interventions. In the Finnish study, the cumulative incidence of diabetes after four years was 11% in the intervention group and 23% in the control group. During the trial, the risk of diabetes was reduced by 58% ($P < 0.001$) in the intervention group (Fig. 4), and was directly associated with changes in lifestyle. The same strategy is appropriate for a number of non-communicable diseases, but it is quite clear that for these strategies to work, there is a need for both a population approach and political commitment.

Several studies provide evidence that the approach aimed at high-risk individuals (for example, those with IGT) may not be enough to prevent all cases of type 2 diabetes. Data from the UKPDS indicate that pancreatic β-cell function is already substantially reduced at the time of clinical diagnosis of type 2 diabetes[14,64]. Even at the earlier stage of IGT, β-cell function is already impaired[65] and intervention at this stage may be too late to prevent many cases of diabetes. In addition, intensive intervention programmes using well-developed behaviour-modification approaches often show high relapse rates[66], with weight gain and an increase in blood glucose occurring 1 to 2 years after an initially encouraging response. As a result, a number of studies are either underway or are being planned to examine whether therapeutic intervention with drugs such as metformin, α-glucosidase inhibitors, angiotensin-converting enzyme inhibitors and thiazolidinediones might be used for prevention as well.

What is not yet known is whether treatment of IGT and IFG specifically can delay or prevent the appearance of macrovascular disease, the main cause of morbidity and mortality in type 2 diabetes[30]. However, delaying the onset of diabetes in such high-risk

subjects will itself provide benefit in terms of morbidity and mortality. It may therefore be prudent to treat such individuals with, at the least, lifestyle advice or with glucose-lowering agents of proven long-term safety while more data are accumulated.

Sociocultural perspectives of type 2 diabetes prevention

One of the myths of the modern world is that health is determined largely by individual choice[67]. The myth is particularly exemplified in the area of NCDs such as type 2 diabetes. Changes in work patterns from heavy labour to sedentary, the increase in computerization and mechanization, and improved transport are just a few of the changes that have had an impact on human health[1]. These should be considered in any approaches to improve health worldwide, along with decreasing easy access to fast foods and empty calories. Thus prevention requires a public health approach accompanied by major structural changes in society. Can we turn the clock back?

The strategy used by government officials in Philadelphia may not be the answer, but it certainly underlines the point. Philadelphia was voted fattest city in the United States and officials patrol the streets with weighing machines in an effort to persuade locals to lose weight[68]. There is an urgent need to address the social and political issues in the public health approach to prevention of type 2 diabetes, some of which have been addressed recently by McKinlay and Marceau[69]. Major shifts in public policy are necessary to create an environment for the whole community or nation in which individual behavioural initiatives can succeed. This may require changes in taxation and reimbursement for health promotion, provision of safe conditions (for example, from assault or traffic) for the elderly and younger sectors of the community, as well as community and workplace access to facilities for exercise.

Aetiology of type 1 diabetes

Type 1 diabetes is a discrete disorder and its pathogenesis involves environmental triggers that may activate autoimmune mechanisms in genetically susceptible individuals, leading to progressive loss of pancreatic islet β-cells[70]. In many respects, attempts to solve the puzzle of the aetiology of type 1 diabetes have been disappointing despite decades of research[70,71]. Predisposition is mediated by a number of genes that interact in a complex manner with each other and the environment[70,71].

Very low rates of type 1 diabetes have been found in Asian populations[72], whereas Finland shows the world's highest incidence of 35 cases per 100,000 (ref. 73). This differs from other Baltic States[74], notably Estonia[75] whose population is linguistically and ethnically very similar to that of Finland, but who suffer only a third the incidence. This indicates that environmental factors have a particularly powerful influence on the appearance of type 1 diabetes.

Despite these compelling epidemiological findings, the environmental factor/s that precipitate type 1 diabetes in genetically susceptible individuals have remained speculative, although viruses — including *in utero* exposure, which is remote from clinical onset — have been suggested, as have dairy products and early weaning onto cow's milk, and increased dietary nitrate and nitrite (refs 70,76 and Table 3). The environmental agents that cause this injury are difficult to identify owing to the long period between exposure and the onset of hyperglycaemia, the complex genetics of the disease, and the likely multiple insults needed to initiate insulitis.

We believe that the triggering agent is likely to be environmental and ubiquitous and have recently suggested a new possible candidate[77]. We reported that minute quantities of the macrolide antibiotic bafilomycin A1 (bafA1) caused reduced glucose tolerance and disruption of the pancreatic islets in mice. BafA1 and related macrolides are produced by *Streptomyces* species. A *Streptomyces* species is also the source of streptozotocin, an agent used to produce experimental diabetes in rodents, either by a single administration or by multiple low-dose administration. The latter is taken to be a reliable model of induced autoimmune diabetes in rodents[78].

Table 3 Possible aetiological factors in type 1 diabetes

Non-genetic*	Genetic†
Viral infections (for example, coxsackie, cytomegalovirus)	Human leukocyte antigen (HLA) associated
Early infant diet (early cessation of breast feeding/early introduction of cow's milk)	Non-HLA associated
Perinatal infections	
Toxins (for example, dietary nitrosamines, bafilomycin, concanamycin A)	
Vaccine administration	

*No clear evidence for the role of any of these agents has been established.
†Consistent evidence for both HLA- and non-HLA-associated genes has been established.

Streptomyces species are ubiquitously present in soil and some can infest tuberous vegetables such as potatoes and sugar beet[77]. Hence dietary exposure to a *Streptomyces* toxin could possibly cause repetitive pancreatic islet β-cell damage, and so be diabetogenic in humans genetically susceptible to autoimmune insulitis.

Future perspectives

It will take a much more integrated and international approach to have a significant impact on the diabetes epidemic. We must accept that type 2 diabetes is not just a disease, but a symptom of a much larger global problem — the effect on human health of environmental and lifestyle changes[1].

It may not be too late to develop highly integrated policies for education and intervention[69,76]. A large proportion of cases of type 2 diabetes is preventable. Initiatives for consumer education that promote a healthy diet could be reinforced by legislative changes such as increased taxation of certain 'unhealthy' foods. This is obviously a complex area, fraught with potential and political hazards.

Diabetes is likely to remain a huge threat to public health in the years to come. In the absence of effective and affordable (particularly for developing nations) interventions for both types of diabetes, the frequency will escalate worldwide, with the main impact being seen in developing nations and the disadvantaged minorities in developed nations[2,3,45]. Thus prevention of diabetes and its micro- and macrovascular complications should be an essential component of future public health strategies for all nations. An urgent priority is the establishment of a multidisciplinary international task force representing all parties that can help reverse the underlying socioeconomic causes of the problem and address the issues that have led to the diabesity and NCD epidemic. □

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