Obesity 1

The global obesity pandemic: shaped by global drivers and local environments

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This is the first in a Series of four papers about obesity

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The simultaneous increases in obesity in almost all countries seem to be driven mainly by changes in the global food system, which is producing more processed, affordable, and effectively marketed food than ever before. This passive overconsumption of energy leading to obesity is a predictable outcome of market economies predicated on consumption-based growth. The global food system drivers interact with local environmental factors to create a wide variation in obesity prevalence between populations. Within populations, the interactions between environmental and individual factors, including genetic makeup, explain variability in body size between individuals. However, even with this individual variation, the epidemic has predictable patterns in subpopulations. In low-income countries, obesity mostly affects middle-aged adults (especially women) from wealthy, urban environments; whereas in high-income countries it affects both sexes and all ages, but is disproportionately greater in disadvantaged groups. Unlike other major causes of preventable death and disability, such as tobacco use, injuries, and infectious diseases, there are no exemplar populations in which the obesity epidemic has been reversed by public health measures. This absence increases the urgency for evidence-creating policy action, with a priority on reduction of the supply-side drivers.

Introduction

As UN member states prepare to gather in New York in September, 2011, for the first High-Level Meeting of the UN General Assembly on non-communicable diseases (NCDs), the inexorable global rise of obesity will be the toughest challenge that they face. Many countries can serve as excellent exemplars for reduction of infectious diseases, injuries, and some of the risk factors for NCDs, such as smoking, high cholesterol, and hypertension. However, no country can act as a public health exemplar for reduction of obesity and type 2 diabetes. All countries are searching for answers about how to reverse the rising tide of adult and childhood obesity.

The 2004 WHO global strategy on diet, physical activity and health[1] provides an excellent overall guide for societal action. However, with few exceptions, governments have made very slow progress in the implementation of these strategies. The food and media industries have, by contrast, moved rapidly by making various national[2] and international[3] pledges, including self-regulatory codes of practice. Although independent assessment of the true effect of these pledges is needed, governments also need to meet their obligations for policy action and leadership, which are described in several authoritative reports.[1–5]

The aim of *The Lancet*'s Obesity Series is to state the case for action on obesity: what is the size and nature of the problem, what is driving its global increase, what will the future obesity burden be under a business-as-usual scenario, and what action is needed to reverse the epidemic? In this first report in the Series, we describe the obesity epidemic and explain the reasons for its concurrent rise across countries and the wide variation in obesity prevalence between countries. The interaction of these major determinants of obesity has important implications for the action needed to reverse the epidemic.

Key messages

- Changes in the global food system, including reductions in the time-cost of food, seem to be the major drivers of the rise of the global obesity epidemic during the past 3–4 decades, although substantial differences in national and local environments (especially sociocultural, economic, and transport environments) produce the wide variation in obesity prevalence recorded across populations.
- In the first half of the 20th century, increased mechanisation and motorisation were accompanied by corresponding decreases in food energy supply (indicative of consumption), thereby keeping obesity prevalence low. In many high-income countries, an energy balance flipping point seems to have occurred in the 1960s–70s, with an increasing food energy supply now pushing up energy intake and population weight.
- Adult obesity continues to increase almost universally, but in some childhood and adolescent populations the epidemic seems to be flattening or even decreasing.
- Present systems for monitoring population weight and nutrition are inadequate in almost all countries.
- Obesity is the result of people responding normally to the obesogenic environments they find themselves in. Support for individuals to counteract obesogenic environments will continue to be important, but the priority should be for policies to reverse the obesogenic nature of these environments.
- Governments have largely abdicated the responsibility for addressing obesity to individuals, the private sector, and non-governmental organisations, yet the obesity epidemic will not be reversed without government leadership, regulation, and investment in programmes, monitoring, and research.

The global rise in obesity prevalence

The rise of the obesity epidemic seemed to begin almost concurrently in most high-income countries in the 1970s and 1980s;[6] since then, most middle-income and many low-income countries have joined the global surge in obesity prevalence in adults and children.[7–9] By 2008, an estimated 1·46 billion adults globally were overweight (body-mass index [BMI] >25 kg/m²) and 502 million adults were obese (BMI >30 kg/m²).[9] Furthermore, an estimated 170 million children (aged <18 years) globally were classified as overweight or obese.[10] This estimate includes more than 25% of all children in some countries, more than double the proportions from the start of the epidemic (figure 1).

Analysis of the patterns of the obesity epidemic in the past four decades is limited by the absence of representative data from different countries.[11] Nevertheless, the pattern by which obesity prevalence rises in particular populations seems predictable. In low-income and middle-income countries, groups of high socioeconomic status in urban areas tend to be the first to have high obesity prevalence, but the burden of obesity shifts to low socioeconomic status groups and rural areas as a country's gross domestic product (GDP) increases.[14,15] In Brazil, one of the few middle-income countries with repeated cross-sectional surveys of BMI, this pattern was particularly evident for women, with obesity rates increasing rapidly in the lowest income groups.[16] The highest prevalences of overweight and obesity are in middle-age groups (45–59 years) throughout this transition.[12,17]

The global rise of obesity has serious health effects. Raised BMI is an established risk factor for diseases such as type 2 diabetes, cardiovascular diseases, and many cancers.[4,18,19] The disability attributable to obesity and its consequences was calculated in 2004 at more than 36 million disability-adjusted life-years,[18] with obesity accounting for between 2% and 6% of total health-care costs in many countries.[20] NCDs are now the dominant cause of preventable disease burden even in many low-income countries,[18,21] and obesity has overtaken tobacco as the largest preventable cause of disease burden in some regions.[22] Although the reduction in premature mortality and morbidity from cardiovascular diseases in high-income countries during the past 40 years has been substantial, there is serious concern that the rise of obesity and type 2 diabetes will slow or even reverse this trend.[23–25]

The increases in overweight and obesity in adults are widely projected to continue to heighten the burden of obesity-related morbidity and mortality in the coming decades.[1,26] However, encouraging reports are emerging from countries such as Sweden, Switzerland, France, and Australia that overweight and obesity prevalence in some childhood age groups might be flattening or even decreasing.[27] But, overall prevalence is still high. Crucially, very few countries have adequate monitoring systems in place, which is remarkable in view of the importance of this issue. Consequently, the frequency and standard of monitoring urgently needs to improve so that the progress of the global epidemic can be tracked and lessons from the experiences of different countries and population groups can be learnt.

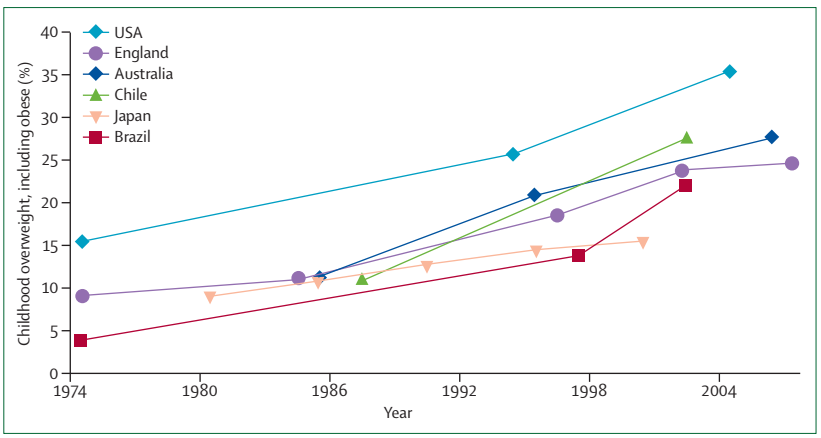

Figure 1: **Estimates of percentage of childhood population overweight, including obese (with use of International Obesity Taskforce cutoffs) in a selection of countries**
Based on data from Wang and Lobstein,[11] International Association for the Study of Obesity,[12] and Matsushita and colleagues.[13]

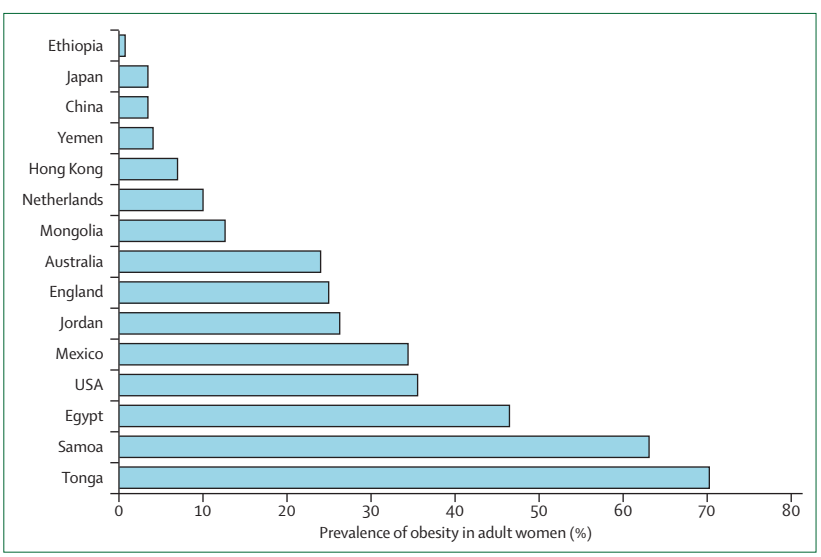

Figure 2: **Prevalence of obesity (body-mass index [BMI] >30 kg/m²) in adult women in a selection of countries in the 2000s**
Data from International Association for the Study of Obesity.[17]

The available data show very wide variations in obesity prevalence globally, particularly for women (figure 2). For some populations (eg, China), small body-frame sizes mean that a BMI cutoff point of 30 kg/m² for obesity will underestimate the amount of over-fatness and comorbidities, compared with other populations with larger frame sizes (eg, Tonga).[28] However, this definitional difficulty does not account for the 100-times differences between the populations (0·7% *vs* 70%). Many of the reasons for the variations across populations are intuitive. For example, Ethiopia does not have sufficient national

wealth for obesity to have manifested itself, and populations in Hong Kong and Jordan have had a greater exposure to obesogenic food environments than do their counterparts in China and Yemen. However, many complexities exist in understanding why some populations and subpopulations are more susceptible to the drivers of obesity than others, and how mediating factors affect different population groups.

Broad economic effects on obesity

The most obvious environmental precondition for a population to develop obesity is sufficient wealth. The relation between GDP and mean BMI is positive and linear up to a GDP of about US$5000 per person per year; at greater GDP, the relation with GDP and BMI is almost flat.[29] A degree of economic prosperity is thus an enabler for obesity, but the level of prosperity does not have to be high for obesity to manifest; in some low-income countries, such as Pacific Island nations, obesity prevalence is very high.[17] A return to national poverty is not a recommended approach to reduce obesity and type 2 diabetes but, as seen in Cuba and Nauru,[30,31] it can have that effect.

The economic transition towards increasing GDP brings with it several other transitions: demographic (younger to older population distribution, rural to urban); epidemiological or health (infectious diseases to NCDs); technological (low to high mechanisation and motorisation); and nutritional (traditional foods and cuisines to more processed energy-dense foods).[32] The pace of change of these transitions has increased substantially in recent decades; so many countries in transition are faced with double burdens of disease. For example, most countries that still have a substantial burden of undernutrition and its related diseases also have a substantial or emerging burden of overnutrition and its related NCDs. Both these conditions need to be addressed together for several important reasons: fetal and infant undernutrition followed by adult overnutrition has a double effect on the later burden of NCDs;[33] the underlying drivers within the food system (eg, food quality and food distribution) are often common to both disorders; and NCDs cannot be ignored even while efforts to reduce undernutrition continue.

In the same way as obesity is the result of people responding normally to the obesogenic environments that they find themselves in, so too do these obesogenic environments arise because businesses and governments are responding normally to the broader economic and political environments that they find themselves in. A central tenet of modern, market-based economies is the benefits of economic growth; and a parallel tenet of business and trade is the benefits of more liberalised, less regulated global markets. Economic growth is especially important for low-income countries to move them from poverty to economic prosperity; however, for high-income countries, higher levels of GDP do not bring greater happiness and wellbeing for their citizens but do bring greater consumption of all products.[34] The technological changes that are creating cheaper and more available food calories and the strong economic forces driving consumption will inevitably lead to overconsumption and obesity.[35–37]

In the broader view, obesity is similar to rising greenhouse gases and environmental degradation as yet another detrimental effect of individual and corporate overconsumption.[34] The pressure for market liberalisation means that regulatory approaches, although feasible, are difficult to achieve—as exemplified in the great reluctance of policymakers to regulate reductions in marketing of obesogenic foods and beverages, such as fast foods and sugar-sweetened drinks, to children. There are many reasons for government intervention to restrict marketing to children, including protection of the rights of children,[38] public demand for regulations,[39] and application of the precautionary principle of preventive action early, even before absolute proof is available.[40] Although obesity has been described as "a sign of commercial success but a market failure"[41] debate exists about whether market failure provides an additional argument for government intervention with respect to prevention of childhood obesity[41–45] (panel 1).

Panel 1: Is the market failing children?

A market fails when prices and the quantities bought and sold are no longer indicative of their costs and benefits to society.[46] Is the market failing children? The first of four reasons for market failure is when vulnerable individuals are not protected.[47] Children are clearly a vulnerable group that warrant societal protection, and this notion represents the strongest argument for government intervention. They are not mature, they do not have nutritional knowledge, are unable to perceive the risks of their behaviour, and their choices are readily affected by marketing.[47–49] The second reason is when consumers do not have the information necessary to make fully informed decisions about their food selection,[47,50] as is also clearly the case with children. However, generally, interventions to rectify information gaps seem to have modest effects.[46] The third reason for market failure is when people prioritise immediate gratification over potential long-term negative results, which is a hallmark of childhood. The final reason relates to spill-over effects (or externalities), when the costs of obesity are borne by society. Although yearly health-care costs to the taxpayer are higher for obese than for non-obese people, reduced life expectancy due to obesity makes it uncertain whether the life-time social costs are actually higher.[42,51] Externalities might arise at the family level through reduced household income or additional carer duties.[46] Thus, there is ample justification for protecting children's health from the predatory effects of markets, yet almost universally, governments are failing in this responsibility. The charge of so-called nannyism almost inevitably arises[42] in relation to regulatory interventions, yet for children, and even for adults, governments have a fundamental role in helping to make healthy choices the easy choices.[52]

Drivers of the obesity epidemic

We define a driver of the global obesity epidemic as an environmental factor that has changed substantially during the past 40 years (coinciding with the upswing of the epidemic), is global in nature (affecting almost all countries with enabling economic conditions), and is rapidly

transmissible (in view of the near simultaneous nature of the epidemic across countries). Some environmental determinants of obesity, such as the built environment, can have important effects on behaviours;[53] however, built environments have not changed simultaneously and universally to become more obesogenic during the past few decades. The built environment is thus unlikely to have been a major driver of the global epidemic, although the way in which people have responded to the built environment (eg, increased traffic congestion) has changed with time and might be important. The obvious possible drivers of the epidemic are in the food system:[35] the increased supply of cheap, palatable, energy-dense foods; improved distribution systems to make food much more accessible and convenient; and more persuasive and pervasive food marketing.[54]

Several studies have tested the hypothesis that increases in the food supply are the dominant drivers of the weight gain in populations.[55–57] Results from these investigations show that the rise in food energy supply was more than sufficient to explain the rise in obesity in the USA from the 1970s,[55,56] and most of the weight increase in the UK since the 1980s.[57] A related hypothesis is that the policies put in place in the USA and other countries to increase the food supply from the 1970s led to a situation in which the abundance of food in these countries began to push up population energy intake—a reversal of the previous situation in which energy intake was pulled down by decreases in physical activity (panel 2, figure 3).

Figure 4 shows the key drivers of the global obesity epidemic and presents an overview framework for understanding of population-level obesity determinants and solutions. Our framework has features in common with other frameworks for obesity determinants[26,65]—eg, the layered levels of determinants that recognise that the physiology of energy balance is proximally determined by behaviours and distally by environments. A further distinction is made between the obesogenic drivers within the food and physical activity environments, which are proximal determinants of behaviours, and more distal systemic drivers. Taxation regimes, regulation of the marketplace, and social and economic policies set the conditions under which businesses and individuals operate, and many of these conditions can have distal effects on obesity.[34,66] For example, independent of the overall wealth of a country or state, the higher the level of income inequality, the higher the prevalence of obesity.[67] These distal effects might convert to higher obesity prevalence through many pathways, such as through psychosocial and behavioural effects.[67]

Our framework (figure 4) recognises the importance of environmental conditions that operate on a population to accentuate or attenuate the effect that the drivers have on the trajectory of changes in obesity prevalence. These moderators or modulators, although important in affecting the slope of the rise of obesity, cannot be deemed drivers of the epidemic if they have not changed sufficiently and coincidentally with the onset of the obesity epidemic. For example, if the availability and promotion of cheap, energy-dense food increases globally throughout several decades, the traditionally high levels of active transport in the Netherlands will help to attenuate this obesogenic effect in the Dutch population; whereas the traditionally high levels of car transport in the USA will accentuate this obesogenic effect in the American population. Similarly, cultural body-size preferences could moderate the drivers such that increased food access will probably have a larger effect in

Panel 2: Energy balance flipping point

In most high-income countries, the energy expenditure needed for daily life has decreased since the beginning of the 20th century because of increasing mechanisation, urbanisation, motorisation, and computerisation. All else being equal, the expected result of an increasingly sedentary lifestyle would have been weight gain. Therefore, why did obesity prevalence not rise substantially until the 1970s?

A parallel reduction of food energy intake coupled with decreasing physical activity is one possible explanation for the low prevalence of obesity during the first three-quarters of the 20th century. Data from the US Department of Agriculture lend support to this explanation since they clearly show a reduction of per-person energy available in the food supply (food production plus imports minus exports and non-human use) from early in the 20th century until the 1960s[58] (figure 3), mainly because of reduced consumption of wheat products.[59,60] A 1948 editorial in the *Journal of the American Medical Association*[61] attributed the fall in US wheat consumption partly to the decreased energy demands resulting from reduction of hard labour. Presciently, the same editorial warned of a potential rise in obesity prevalence if consumption of sugars, sweets, and fats began to increase. Indeed, the 1970s saw a striking rise in the quantity of refined carbohydrates and fats in the US food supply,[62,63] which was paralleled by a sharp increase in the available calories (figure 3) and the onset of the obesity epidemic. As Cutler and colleagues[35] note, there was "a revolution in the mass preparation of food that is roughly comparable to the mass production revolution in manufactured goods that happened a century ago," that "lowered the time price of food consumption". The increased availability and marketing of cheap, readily available food was so great that food waste has progressively increased by about 50% since the 1970s.[55]

We postulate that an energy balance flipping point has occurred in most high-income countries in the past century with two distinct phases: the so-called move less, stay lean phase (1910–60), characterised by decreasing physical activity levels and energy intake, and a population that remained lean; and the subsequent so-called eat more, gain weight phase, characterised by increasing energy intake and a concomitant rise in population weight. In each of these phases, we postulate that energy balance was predominantly achieved by different mechanisms: decreasing energy intake (through appetite mechanisms) to match decreasing expenditure, followed by increasing energy expenditure (through increased weight and, thus, resting metabolic rate) to match increasing intake. Thus, decreases in physical activity in the first phase were probably able to pull down energy intake, because intake was being matched to expenditure. The result was that obesity rates did not increase during this period despite widespread uptake of mechanisation and motorisation. The second phase seems to have been ushered in by an energy balance flipping point, when energy intake rose because of environmental push factors (ie, increasingly available, cheap, tasty, highly promoted obesogenic foods). The concomitant rise in weight was the physiological mechanism for restoration of energy balance.[64] Food supply data from the US lends support to this flipping point hypothesis, but it needs to be tested in other countries and with more diverse datasets.

Tongan women, where large body size is a positive attribute,[68] than in Japanese women, where small body size is deemed ideal.[69]

The effects of the environmental moderators are shown in figure 4 as affecting the rise in obesity but are not depicted as part of the direct driver pathways.

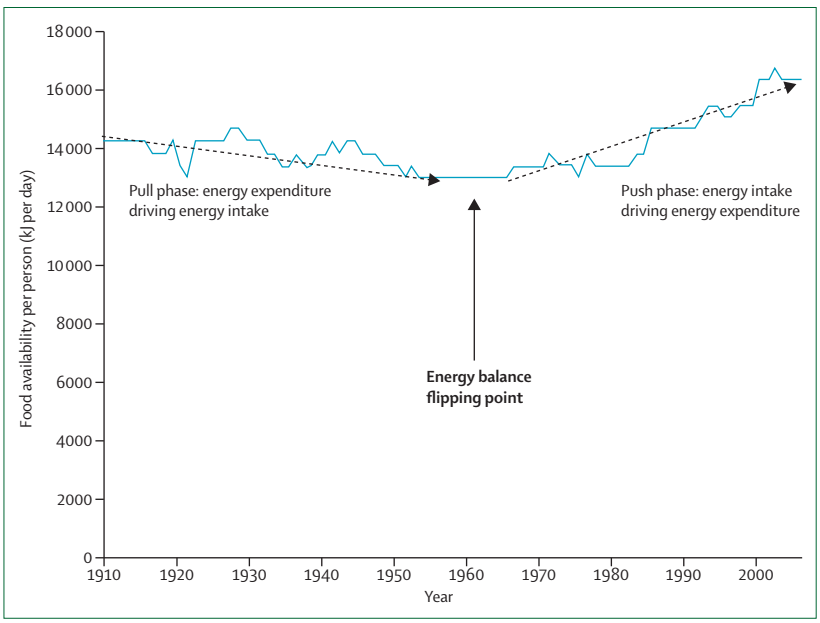

Figure 3: **Food availability for the USA, 1910–2006**[58]
There are two distinct phases: a decrease in food energy supply (postulated to be pulled down by reduced energy expenditure requirements for daily living), followed by an increase in food energy supply (postulated to be pushed up by increasing food access). An energy balance flipping point is proposed, marking the change in how the US population generally achieved energy balance.

However, we recognise that this scheme is simplified and that factors such as active transport environments and body-size preferences could change sufficiently with time to drive changes in obesity prevalence. Indeed, for some countries such as China, rapid urbanisation and motorisation of the population could be judged a major driver of the rise in obesity.[70] Although the interaction between all the environmental factors is undoubtedly complex and multifaceted (panel 3), the strong directional force of the systemic and environmental drivers is evidenced by the continued rise of the epidemic globally.

Other environmental and individual effects

The existing environments within a country (eg, the built environment, transport systems, active recreation opportunities, cuisines and food culture, and culture around body size) can greatly moderate or modulate the effects of the global obesity drivers on population BMI (figure 4). These effects can be powerful and help to explain much of the differences in obesity prevalence between populations. They clearly hold opportunities for interventions to make environments less obesogenic. Interventions such as increasing the price of unhealthy food and beverages[81–83] or decreasing the price of healthy foods[84] have received attention in recent years, but there has been very little research into understanding and changing the powerful sociocultural determinants of food choices, physical activity, and body-size perception.[85] These determinants should be a priority for research if the high prevalence of obesity in specific ethnic groups is to be addressed.[86]

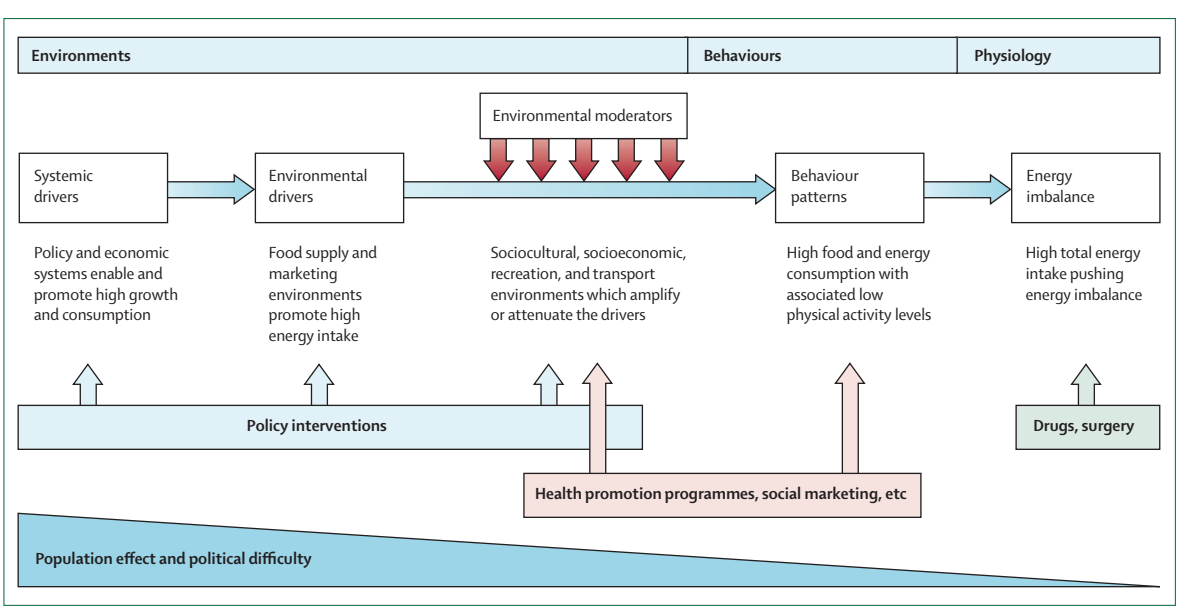

Figure 4: **A framework to categorise obesity determinants and solutions**
The more distal drivers are to the left and the environmental moderators that have an attenuating or accentuating effect are shown, along with some examples. The usual interventions for environmental change are policy based, whereas health promotion programmes can affect environments and behaviours. Drugs and surgery operate at the physiological level. The framework shows that the more upstream interventions that target the systemic drivers might have larger effects, but their political implementation is more difficult than health promotion programmes and medical services.

Within a given environment, BMI differs between individuals, which is indicative of individual moderators or modulators of energy balance. For example, too little sleep and impaired sleep quality have been shown to have important effects on weight gain.[87] Genetic effects are one of the most highly researched determinants of obesity. However, apart from the identification of a few rare, single genetic abnormalities, the quest to identify genes related to common obesity (obesity not caused by an underlying condition) has been disappointing. The potential epigenetic effects of behavioural and environmental factors on genetic expression are now receiving much attention (panel 4).

The role of individual choice in both the causes of and solutions to obesity has great appeal because of its simplicity. However, this notion is in fact highly contentious and is indicative of the tension between the simple and the complex that is ever present in debates about obesity. Undoubtedly the final decision to consume a particular food or beverage, or to exercise or not, is an individual decision. However, to negotiate the complexity of the environment and the choices it poses, many of these decisions are automatic or subconscious. Cohen[98] puts forward a strong argument that "excessive food consumption occurs in ways that defy personal insight or are below individual awareness", and provides plausible neurophysiological mechanisms for many automatic responses to physiological, environmental, and interpersonal cues in relation to eating and physical activity opportunities. Appetite physiologists label this response as "passive overconsumption".[99] Further increase in choice, especially through more processed products on supermarket shelves or more items on fast food menus, is unlikely to reduce overconsumption and could have the opposite effect. Furthermore, cognitive strategies to combat overconsumption, such as weight-loss diets, can be successful for some individuals but are unlikely to be population solutions. Even weight-loss diets that are supported by trial data, such as high protein or low glycaemic index diets,[100] might not be suitable as solutions for global obesity because of their detrimental effect on the environment (eg, high meat protein diets[101]) or staple food production (eg, rice).

Approaches to address the obesity epidemic

Approaches to address the obesity epidemic are broadly categorised within figure 4, with a detailed discussion of solutions presented in the report by Gortmaker and colleagues[75] in this Series.

The effects on population trajectories of obesity prevalence are likely to fundamentally differ between an intervention aimed at motivating behavioural changes (eg, health promotion programmes, social marketing, education) and policy interventions (in this context, meaning enforceable actions such as laws and regulations) that reverse the environmental drivers (eg, reducing the cost of healthy foods and increasing the costs of unhealthy foods). The interventions to motivate behavioural changes could be regarded as counteractions (ie, they counteract drivers of increasingly obesogenic environments by acting on some of their mediators) and they might have important obesity prevention effects, especially in children, if applied to a whole community.[102,103] However, sustainability and affordability are the two major continuing challenges, even for programmes with proven effectiveness.

Panel 3: Modelling for obesity

The Obesity System Map,[71] introduced by the UK Foresight Programme in 2007, is the most comprehensive compilation so far of the determinants of obesity and their inter-relations. The map clearly shows the enormous complexity of the causal relations relevant to obesity by linking physiology, individual behaviours, and environmental variables that stretch from local to global levels. In addition to the interdependence between factors and the importance of feedback loops, there are other characteristics of the obesity problem that are not shown. These characteristics include the heterogeneity of the factors that need to be involved in systems change, the diversity of environments where change needs to take place, the non-linear relations that govern the system's dynamics, and the sometimes random or quantum nature of behaviour change.[72] This complexity creates an apparently intractable or so-called wicked problem,[73] and the imperative to turn around the obesity epidemic clashes with its wicked nature. In aiming to establish and prioritise solutions, the challenge is to reduce the complexity of obesity enough so that it can be understood by researchers, policy makers, and the public without becoming overly simplistic. Some progress in this challenge lies in mathematical modelling applied to complex health and behavioural questions.[64,74,75]

In the past 5 years, several mathematical models have been developed by use of various modelling techniques.[76] As with other models, they can have descriptive, explanatory, or evaluative aims.[77] Figure 5 shows the aims for obesity modelling at the population level. Descriptive studies quantify the present burden and potential future trends of obesity. Explanatory studies analyse the causes of the rise in obesity prevalence with time and the variability across populations. Evaluative models assess the likely effect of interventions to reduce future prevalence.

Mathematical models are "a set of assumptions together with implications drawn from them by mathematical reasoning".[74] Compared with conceptual models, they have the valuable attributes of forcing theoretical precision, making assumptions transparent, promoting data analysis and hypothesis testing, and having many practical applications.[74] Since models match reality in important ways but are far simpler than reality (they draw attention to some aspects of reality but ignore others[78]), they can help us understand complex problems such as obesity and its solutions.[72] Indeed, mathematical modelling has been crucial to understand and to respond to other global threats, such as climate change[79] and infectious diseases.[80]

Irrespective of their goal, all mathematical models need a logical framework linking the pathways between each of the factors in the model, accompanied by tight specifications of the assumptions and quantitative estimates underpinning each of the links. Complex frameworks, such as the Foresight Obesity System Map, attempt to incorporate most individual and population-level determinants. However, substantial simplification of models can arise when only populations are included (determinants causing individual variability drop out), when questions are narrowed (determinants that have not changed with time can be dropped for questions about the rise of obesity), or when solutions are being modelled (factors not included in the intervention can be dropped). These methods can be expected to provide the core techniques to understand and to respond to complex non-communicable disorders such as obesity.[51,64,75]

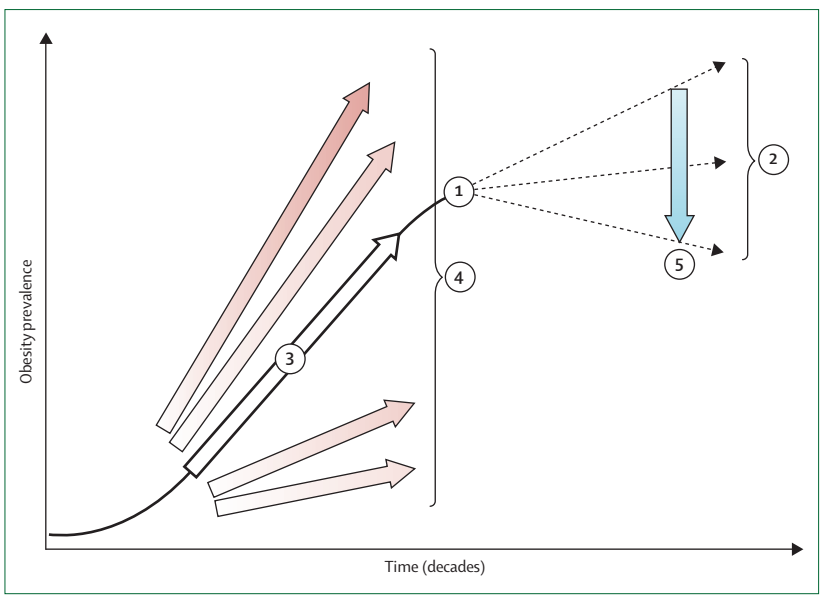

Figure 5: **A schematic diagram of the major uses of modelling of population prevalence of obesity**
The black line is the trajectory of increases in obesity prevalence with time, with the present burden (1) and projected future burdens (2) being descriptive uses of modelling. Explanatory uses of modelling include explaining the rise in obesity with time (3) and the differences in prevalence rates (4). Evaluative uses include assessment of the potential for solutions to reduce the future burden of obesity (5).

Panel 4: How much can genetics explain of the obesity epidemic?

One useful way to think about the relation of genes with obesity was expressed by George Bray when he said, "the genetic background loads the gun, but the environment pulls the trigger".[88] The rise of obesity prevalence throughout the past few decades clearly cannot be accounted for by population genetic changes. The heritability of body-mass index (BMI) is often cited as 40–70%,[89] yet large genome-wide association studies to identify common single nucleotide polymorphisms (SNPs) associated with BMI have been unable to explain more than a small proportion (<2%) of BMI variability.[90,91] Heritability is often misinterpreted as being the proportion of BMI variance caused by genetics; whereas heritability studies, rather than estimating cause, estimate the proportion of genetic variance and non-genetic variance that explains BMI variance in a given study population. The study populations are usually based on twin cohorts which, even if reared apart, live within very narrow environmental variances (rather than the wide variance of environments across countries shown in figure 2), which results in high heritability estimates. These estimates seriously understate the environmental effects and thus provide no indication of the potential to reduce obesity via environmental change.[92] Part of the missing heritability of obesity might be the result of small individual contributions of many SNPs that genome-wide association studies have been underpowered to detect, or some rare variants that are not identified by common SNPs. Much of the variability between individuals in bodyweight might be attributable to gene–environment or gene–behaviour interactions,[93] including interactions in the intrauterine environment.[94] An intriguing possibility mediating the gene–environment interaction is the potential contribution of epigenetic mechanisms that modify the expression of genes. For example, DNA methylation patterns can be affected by maternal diet; these epigenetic modifications can persist for decades,[95,96] and possibly be inherited by future generations.[97] Epigenetic contributions to obesity will need to be addressed through minimisation of the environmental triggers, rather than manipulation of the genetic guns, in the first instance.

Furthermore, such programmes do not address the underlying drivers of the epidemic.

Interventions that aim to reverse obesogenic drivers (and some of the environmental moderators) will almost all be policy-led—mainly government policy (eg, shifting agricultural polices to incorporate health outcomes, banning unhealthy food marketing to children, healthy public sector food service policies) but some could be food industry policies (eg, moving product formulation towards healthier compositions, self-regulation of marketing to children). Policy-led solutions that apply to environments and affect the whole population have several strengths compared with health education and promotion programmes.[104] They tend to be sustainable, affect the whole population (including those who are difficult to reach), become systemic (affect default behaviours), and reverse some of the environmental drivers. The degree of political difficulty for implementation of policy and regulatory interventions is typically much higher than that for programme-based and education-based interventions[105] (figure 4). Reasons for this reluctance to enact affordable and cost-effective policies include the powerful lobby force of the food (and allied) industries against government regulation of the food market[106,107] and public reluctance to change environments to which they have become accustomed (such as car access and cheap parking in cities, and high fat and sugar food choices in canteens). Nevertheless, the experience with trans-fatty acids in Denmark, where legislation was introduced to restrict their use in food production, is an example of a cost-effective government food policy that was successfully enacted for population health benefit.[108]

Policy interventions for obesity can only be realistically directed at the environment (making healthy choices easier) rather than the individual (compelling them to take the healthy choices). Unlike other public health issues for which enforceable policies can directly require specific behaviours (such as wearing a seat belt or not smoking in offices), there are no regulations that will require people to eat, or not eat, certain foods and to exercise,[66] with the possible exception of a few rules operating in school environments. For this reason, obesity prevention policies do not proscribe particular eating and physical activity behaviours and are thus much less intrusive of human liberties than many policies already in place to control other public health problems. The major strategies available to directly affect behaviours aim to increase the motivation to make healthy choices, and include social marketing, health education, and health promotion programmes.

Implications

In this report, we have provided an overview of the global obesity epidemic, describing the size and nature of the problem, discussing its drivers, and mapping out key contributing factors. We draw attention to the food

system, operating through the energy intake side of the energy balance equation, as the dominant driver of the rise in obesity, although many other environmental and individual factors modulate the effect of the drivers on obesity prevalence in populations and obesity presence in individuals. The highly complex global, national, and local food systems, although feeding the world's population, are nevertheless falling short by both promoting overnutrition (overweight or obesity) in an estimated 2 billion adults and children[9,10] and not reaching the further 1 billion people who are undernourished.[109] Both these forms of malnutrition are major contributors to preventable population disease burden and are thus substantial barriers to meeting the Millennium Development Goals.[110] With the global population expected to grow to 9 billion people by 2050,[111] and with increased disruption to food production from climate change—such as increased droughts, floods, hurricanes, and global temperatures[101]—the global food system will be placed under great stress which will probably result in more unequal distribution of calories. Policies that address the food system drivers of obesity will therefore be an important part of the much broader imperative to re-orient food systems towards health and development, particularly in vulnerable populations.

As argued in this report and elsewhere,[34] the economic priorities and policies that promote consumption-based growth, and the regulatory policies that promote market and trade liberalisation have produced many benefits but are now increasingly regarded as contributing to the global crises of overconsumption in general. Obesity is but one of these crises, as the private sector becomes ever more effective in its exploitation of basic human biological drives, desires, and weaknesses. Solutions to obesity and to improve health and development cannot be based on the existing framework (consumption-driven growth creating financially-defined prosperity) because this approach has helped to create the difficulties in the first place. Governments and international organisations such as the UN need to provide global leadership on these issues and not abdicate them to the private sector.[112,113] Actions that reduce the economic incentives for over consumption in general (eg, a price on carbon and subsidies on public transport) can have benefits for both human and environmental health.[34] Moreover, a new framework is needed that is based on broadly defined sustainable economics for prosperity (including economic, social, health, and environmental outcomes).[114]

Four decades after the onset of the global obesity epidemic, the awareness of the threat of obesity to population health and wellbeing can be seen in the plethora of national reports and strategic plans from many countries, yet their conversion to action remains largely unrealised (Australia has had three such unfulfilled national taskforce plans since 1997[115–117]). However, recognition in low-income and middle-income countries that the growing contribution of obesity to its burden of malnutrition is a threat to national development remains low. Some governments are making promising moves to convert the rhetoric into action. For example, in the mid to late 2000s, the Government in England developed a series of initiatives such as the Foresight report,[26] the cross government strategy for obesity,[118] restrictions on unhealthy food marketing to children,[119] and a yearly national child measurement programme.[118] Some other countries, notably Brazil, have made substantial steps in national monitoring programmes, restricting marketing to children, and improving school food; and the US White House Task Force on Childhood Obesity[120] holds much promise to bring about change, because of its high-level political commitment. International leadership from the UN and its agencies, and more national leadership from countries will be essential to solve this great challenge of the global obesity epidemic, which is why the UN High Level Meeting on NCDs in September, 2011, is so central to global population health and development.

Contributors

All authors jointly formulated the major ideas, and read and approved the final version of the report. BAS led the writing of the report. GS assisted in writing the report, and collated and included comments from all authors. KDH co-drafted the sections about genetics and the food supply and commented on the remainder of the paper. MLM co-drafted the section about market failure and commented on the remainder of the report. KM, DTF, and SLG provided comments on the report.

Conflicts of interest

We declare that we have no conflicts of interest.

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