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Review Article
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Type 2 diabetes as an 'infectious' disease: is this the Black Death of the 21st century?

D. R. Matthews*†‡ and P. C. Matthews§¶

*Oxford Centre for Diabetes Endocrinology and Metabolism, Oxford, UK, †National Institute for Health Research, Oxford Biomedical Research Centre, Oxford, UK, ‡Harris Manchester College, Oxford, UK, §Peter Medawar Building for Pathogen Research, University of Oxford, UK and ¶Department of Infectious Diseases and Microbiology, Oxford Radcliffe Hospitals, Oxford, UK

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Abstract

We are currently facing a global pandemic of obesity and Type 2 diabetes. In some settings, the population prevalence of Type 2 diabetes is 50%, and half of those affected will die from diabetes-related complications. Eight centuries ago, an epidemic of bubonic plague swept across Europe, killing at least half of its victims. We here draw comparisons between these two pandemics, proposing close analogies between the 'Black Death' of the 14th century and the modern-day equivalent of Type 2 diabetes. Both diseases can be considered in terms of an aetiological agent, a reservoir, a vector and a predisposing toxic environment; populations can be considered as highly susceptible to the transmissable agents of Type 2 diabetes in the setting of calorie excess, inadequate food labelling, poorly regulated advertising and sedentary lifestyles. As for tackling a pandemic of a contagious microbial pathogen, we believe that breaking the cycle of transmission in the diabetes epidemic must be underpinned by political will and prompt, decisive legislation backed by the medical community. Far from fearing that such measures edge us towards a 'nanny state', we believe individuals should expect a responsible government to safeguard them from the toxic milieu that puts them at risk of obesity and its complications, and that communities and populations have the right to have their health protected.

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Keywords Black Death, bubonic plague, epidemic, obesity, Type 2 diabetes

Introduction

Type 2 diabetes is pandemic. This was not the case in the 1950s but it is now. Sixty years ago, Type 2 diabetes had a prevalence of less than 1% in almost all countries of the world (and indeed in some communities was scarcely present at all). The Pima Indian population of Arizona [1] attracted attention in the 1970s because diabetes was found in a large proportion of the population, but this (at the time remarkable) example of change was replicated in many other communities over the subsequent decades. The low prevalence increased in the developed world; it is now 4% in the UK [2] and well in excess of 8% in most states of the USA [3]. In the emergent economies, prevalence ranges up to 50% (urban elderly) of the population [4]. In stark terms, this means that in the UK one in 25 adults has diabetes, while in the USA the figure is one in 12. The problem is even greater in the developing economies, with diabetes being found in up to one in five adults.

One important aspect of any epidemic is the recognition that there must be environmental as well as genetic aetiology, and new triggers to explain the increase in caseload. Although genes undoubtedly alter population susceptibilities, and individual risks may have a genetic component, epidemic causation will always be based on exposure to transmittable, toxic or environmental aetiology. A second important feature relates to prevention. If the problem did not exist 60 years ago and now it does, it also follows that we could potentially move back to the pre-existing prevalence if the pathophysiology can be identified, and risk factors eliminated or minimized.

In an *Oxford Health Alliance* international meeting in 2004, Professor Sir John Bell drew a parallel between diabetes and the Black Death in the 14th century both in terms of extent and in their economic consequences [5]. The increasing occurrence of Type 2 diabetes and obesity now fulfil the criteria for an epidemic, in being an unexpectedly large increase in a disease

Correspondence to: Professor D. R. Matthews, Harris Manchester College, Oxford OX1 3TD, UK. E-mail: david.matthews@ocdem.ox.ac.uk

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state and, indeed, for a pandemic in being worldwide. The Type 2 diabetes pandemic also shares many characteristics with infectious disease. Both have swept through societies with a temporal and geographical succession, and can be related to a toxic environment, a reservoir, a vector and an aetiological agent.

Specific analogies between bubonic plague and Type 2 diabetes

This paper, based on the Banting Lecture given in Liverpool in March 2010, propounds the thesis that Type 2 diabetes behaves like an infectious disease, drawing specific parallels with bubonic plague (Table 1). 'Infectious' here means 'a pestilence liable to be transmitted, apt to spread and quickly affecting others' [6].

The earliest likely account of bubonic plague is ca. 1000 BC, where a terrible affliction is described as being expiated by golden models of tumours (buboes) and of rats [7]. In the UK we have descriptions dating from 450 of a 'grievous plague…which soon destroyed such numbers of them that the living scarcely availed to bury the dead'; in 664 'a sudden pestilence first depopulated the southern parts of Britain and afterwards attacked the Kingdom of Northumbria, raging far and wide with cruel devastation and laying low a vast number of people' [8]. There was another 'very great pestilence' in 961 in London. But the worst and most catastrophic of the plagues was that which swept across Europe between 1346 and 1353 (Fig. 1). Some centuries later, this was coined the 'Black Death' [Latin: *atra* (terrible or black) *mors* (death)] [9].

By analogy, between 1980 and 2020 a huge plague of diabetes has wrought havoc throughout the world. We could speculate that in years to come there may be a debate as to whether pandemics caused by the Black Death or diabetes should most aptly be called 'the greatest ever demographic disaster' [9]. In the last decade, diabetes has rampaged throughout the USA, reaching a population prevalence of over 10% in some states (Fig. 2). Type 2 diabetes spreads as an epidemic disease, killing as many people in the city of London in 4 years as the Black Death did in the same time period (although, of course, the denominator has changed), and both epidemics have a preventable pathogenesis: in the case of the Black Death, *Yersinia pestis* and in the case of Type 2 diabetes, calorie excess in the setting of a toxic environment (Table 2).

Aetiological agents

The Black Death is most likely to have been bubonic plague caused by *Y. pestis*, which causes a spectrum of disease ranging from infected lymph glands (buboes) to pneumonia and septicaemia [11]. Once the illness becomes bacteraemic, plague is almost invariably fatal. The pathological agents in obesity and Type 2 diabetes (by analogy with *Y. pestis*) are high-fat and high-calorie diets. As with many contagious microbial infections, exposure to the aetiological agents required to cause diabetes and obesity also depends on 'inoculation' above a certain critical threshold.

Vectors

Transmission of plague is by fleas from infected rats, which leave the animal when it dies and move on to a human host. In the case of diabetes and obesity, the 'vectors' of disease (by analogy with the fleas) are advertisements and inducements to poor lifestyle. Plague, before its aetiology was understood, was often thought to be divine punishment [12]. Similarly, popular – and indeed medical and governmental – attitudes have attributed the obesity epidemic to a sinning population ('it's your own fault if you are overweight'), with catastrophic consequences [13]. What individuals eat and do may, at a micro level, be a matter of personal choice, but at a population and national level are clearly matters of food availability, cost, education, availability of information and environmental influences on physical activity.

Table 1 Comparisons between characteristics of epidemics of bubonic plague (14th century) and Type 2 diabetes (21st century)

	Bubonic plague	Type 2 diabetes	Notes
Epidemic spread	✔	✔	7 vs. 40 years
Lethal disease	✔	✔	50% mortality in both. Black death, acute; Type 2 diabetes, 20 years
London deaths	ca. 40 000 in 4 years	ca. 40 000 in 4 years	Population denominator: 100 000 in 14th century vs. ca.15 million in 2010
Environmental pathogenesis	✔	✔	See Table 2
Population affected	Up to 50%	Up to 50%	
Demographic catastrophe	✔	✔	Worldwide spread
Economic catastrophe	✔	✔	Type 2 diabetes, >15% of many health budgets. More if complication care is included
Personal catastrophe	✔	✔	
Preventable	✔	✔	

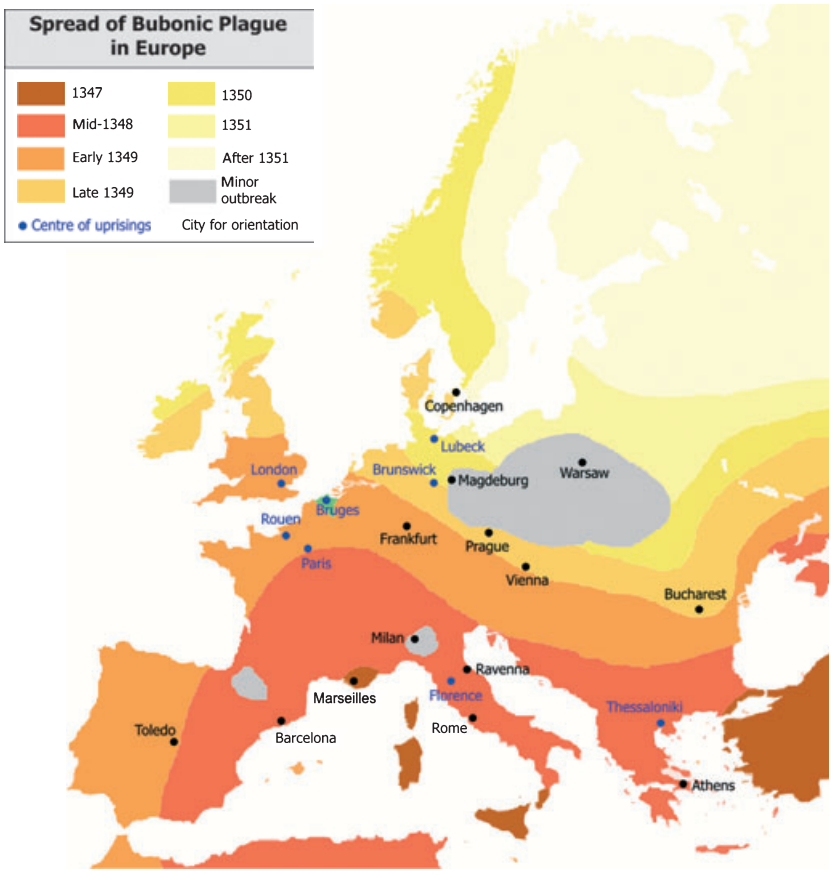

FIGURE 1 Spread of the bubonic plague ('Black Death') epidemic through Europe in the 14th century. Figure reproduced from commons.wikimedia.org under the terms of the GNU Free Documentation Licence.

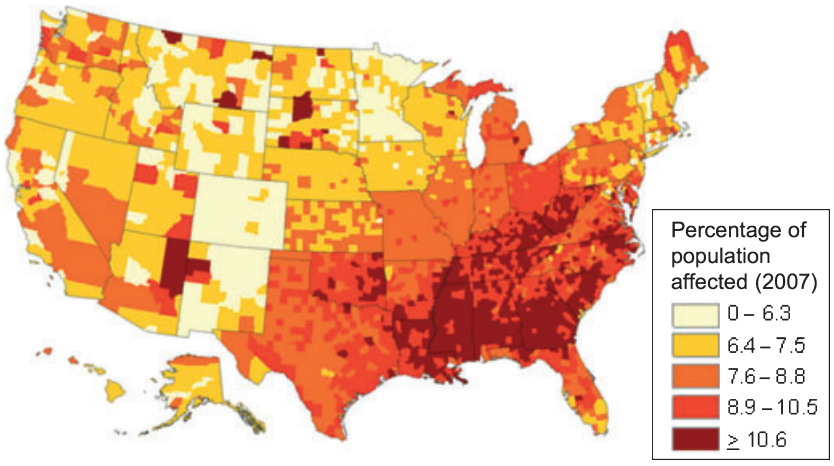

FIGURE 2 Prevalence of Type 2 diabetes in the USA (2007) [3].

Reservoirs

The epizootic reservoirs of bubonic plague are the non-human repositories of *Y. pestis*. There are about 350 mammals known to be susceptible to plague, and over 80 species of flea are known to carry the organism [14]. Rats are the most likely reservoirs for man because of physical proximity. By analogy, the 'reservoir' for obesity/Type 2 diabetes is the food mountain available to us. In the same way that rats are the most likely plague reservoir because of proximity, it is also true that fast-

Table 2 Analogies between bubonic plague and Type 2 diabetes

	Bubonic plague	Obesity and Type 2 diabetes
Aetiological agent	*Yersinia pestis*	Calorie excess
Vector	Flea (*Xenopsylla cheopis*)	Advertising/branding Promotion Low-cost incentives Food availability Inadequate education
(Epizootic) reservoir	Black rat (*Rattus rattus*)	Food mountain
Toxic environment	Overcrowding Poor sanitation	Lifestyle

food outlets and supermarkets are often close to areas of dense population and are open for long hours, providing an accessible reservoir of available and cheap calories. This problem is worse in areas of poverty.

Bubonic plague cannot become widely disseminated if only small numbers of organisms are present; there has to be a sufficient inoculating dose for infection of an epizootic host, a vector and ultimately a human subject. The same is true of creeping weight gain, leading to huge increases in risks for diabetes [15]; it not the quality of food that is important in this case, but the quantity.

Toxic environment

Type 2 diabetes cannot change its prevalence by an order of magnitude – as it has done in some communities in a generation – on the basis of genetic susceptibility, although genes may eventually explain differing racial propensities. The enormous burden of disease that now exists in some populations is exemplified in Sri Lanka, where over 25% of adults in the 60–70 year age group have Type 2 diabetes (Fig. 3A). The epidemic must be related to a change in environment and/or behaviour that has supervened in the last half-century. This predisposing milieu has been termed the 'toxic environment' [16,17]. The environment is as polluted as it was in the 14th century for unsanitary conditions and rats, and in later years for respiratory disease at the time of the London smogs or for cholera at the time of sewage-contaminated well-water in the 19th century [18]; the pollution may even be visible, but the toxicity and danger are ignored.

We know from the mediaeval records that plague pandemics sprang out of urban environments [10]. Here could be found the most prevalent reservoirs and vectors. Similarly, for Type 2 diabetes we know from well-conducted studies that the urban environment is much more toxic in terms of diabetes prevalence than the rural setting (Fig. 3B) [4,19,20]. Urban environments have the biggest exposure to the reservoir of food availability and advertising and lower opportunities for physical exercise.

The progressive increase in the size of portions dispensed by fast-food chains over time was highlighted by *The Independent* newspaper, as was the food industry's abandonment of the calorie count scheme (Table 3) [21]. This is an illustration of the toxic environment; a high consumption of calories leads to weight gain, obesity and huge risks of diabetes. Of course risk does not equate to cause, but the risk is certain and the environmental cause simultaneously obvious yet unaddressed. The wide availability of highly palatable cheap calories is an incentive for overconsumption. In such an environment, the exercise of personal choice becomes extremely difficult or, regrettably, in the case of poor members of society, nearly impossible. On low incomes, eating healthily is a huge problem, and fast-food outlets provide hot food, prepared to consistent standards, for a relatively small cash outlay. The hidden problem is that of fat and calorie consumption, which transforms into a repeated inoculation, leading susceptible individuals towards obesity and Type 2 diabetes [22,23].

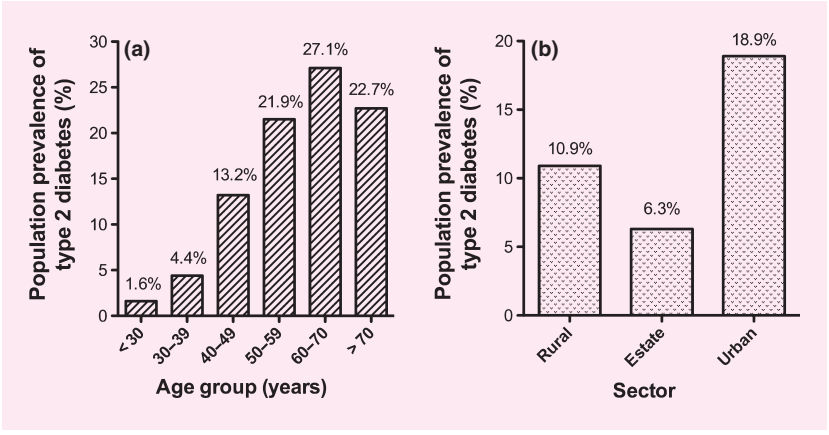

FIGURE 3 Type 2 diabetes in Sri Lanka. (a) Population prevalence in adults according to age group. (b) Population prevalence according to sector (location). 'Estate' refers to manual workers on tea plantations. Adapted from Katulanda *et al*. [4].

Table 3 Content and calorie count of typical meals available from common fast food outlets

Restaurant	Content	Total calories	Percentage of adult male calorie RDA
Pizza Hut	Half large pepperoni feast pizza Two slices of garlic bread Pepsi (375 ml)	1738	69.5
Burger King	Double whopper Regular fries Regular chocolate milkshake	1643	65.7
KFC	Quarter of a family feast bucket: Three pieces of chicken Regular fries Quarter portion of regular coleslaw Quarter portion of barbecue beans Pepsi (375 ml)	1132	45.3
Subway	Foot-long meatball marinara sub Chocolate chip cookie Coca-cola (455 ml)	1421	56.8
McDonalds	Big Mac Medium fries Medium chocolate milkshake	1235	49.4

RDA, recommended daily allowance.
adapted from *The Independent* newspaper [21]. Data for individual restaurants are available on-line at http://www.pizzahut.co.uk/restaurants/menus–deals/dietary-information.aspx, http://www.burgerking.co.uk/nutrition, http://www.kfc.co.uk/nutrition/, http://www.subway.co.uk/menu/nutritional.aspx, http://www.mcdonalds.co.uk/food/nutrition/nutrition-counter.mcd. Interestingly, these organizations all make dietary and nutritional information available on-line, but do not provide calorie counts at point of sale.

Resonances between the Black Death and Type 2 diabetes

Death rates

Pandemics of both diabetes and bubonic plague have killed very large numbers of people. The case fatality rate for plague is estimated to be between 30 and 100% if left untreated [11]. In London in 1348, between 36 and 50% of the population and 36% of the clergy died [9]. In York, plague began in 1349, killing about 40% of the population and 40% of the clergy. We know the data for clergy rather accurately, since parish records recorded their names especially carefully.

The case fatality rate for Type 2 diabetes may not be dissimilar to that of bubonic plague; death attributable to diabetes or its complications occurs in over 50% of those affected, including cardiac death, sudden death and stroke [24,25]. Mortality differences can be seen in terms of the suddenness and imminence of death in the case of the Black Death, but Type 2 diabetes is similarly dangerous, with a time period of years rather than days. The chronicity of diabetes, with its associated morbidity, means that the cost per case to the health service is higher than that of an acute infectious disease.

Consequences of regarding Type 2 diabetes as a pandemic 'infectious' disease

Drawing an analogy between the Black Death and Type 2 diabetes is not simply an academic conceit. If we take seriously the idea that Type 2 diabetes and obesity share characteristics with epidemics of contagious disease, we can direct our actions and interventions appropriately, starting with an analysis of source points, infectious environments, carrier vectors, reservoirs and at risk groups. To choose a recent example, we have considered differences in approach for the recent H1N1 influenza ('swine flu') pandemic and Type 2 diabetes in terms of government action. Table 4 delineates how one would normally address a serious outbreak, measures that were fully implemented in the case of influenza. With Type 2 diabetes, despite the fact that we know that interventions in public health can reap huge benefits, there is a reluctance, verging on anathema, to address the question of excessive consumption in our society. This is not simply a matter of personal choice, because the population is systematically and, perhaps malignly, underinformed about the calorie content of most foods [21].

Tackling the epidemic of Type 2 diabetes and obesity

Simple interventions are possible. We need a consistent and robust system of food labelling, a concerted campaign of education, regulation of advertising and a national consensus on portion sizing. Self-regulation is not sufficient. The current 'traffic-light' food labelling system naively classifies items into

Table 4 Measures taken to address a serious outbreak

Action point	H1N1 Influenza	Type 2 diabetes
Collect the data	✔	×
Assemble an emergency (government) team	✔	±
Establish the basics (data collection)	✔	×
Focus on initial goals:		
Rapid gathering of laboratory samples	✔	×
Assessment of primary prevention measures	✔	±
Identification of possible therapies	✔	✔
Break the infectious cycle	✔	×
Immunize the population	✔	×

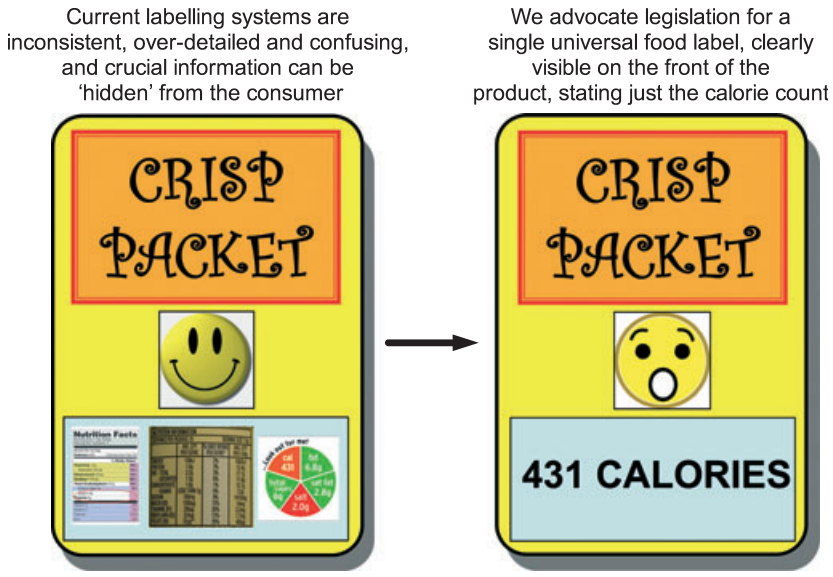

FIGURE 4 Illustration of the merits of a single compulsory 'calorie label' compared with a multitude of dietary information that cannot be clearly interpreted by the consumer.

'good, medium and bad', with confusingly displayed multiple traffic-lights for fats, salt and calories. A recent study found that this had no discernable effect on the relative healthiness of consumer purchases [26]. Industry has responded to the need for clear labelling by introducing a multiplicity of categories; fat, trans-fat, sugars, protein, fibre, sodium, salt, calories, kilojoules and per portion/per weight breakdown may all appear on the label (Fig. 4). This militates against any of the information being used routinely. It is clearly a scandal that information is so badly presented on our foodstuffs that the calorie content is often obscured. For Type 2 diabetes, the main issue is that of fat consumption and calories; since fat contains more calories than carbohydrates, the single number of the calorie count is the best single numerical indicator of a food's contribution to weight gain. In New York, there is a welcome move towards insisting that foods have calorie labelling (exemplified in Fig. 4) and some evidence that this affects consumer choice [27,28].

Ever-escalating portion sizes have doubtless contributed to the epidemic. The food industry has used super-sizing as a way of enticing consumers (Fig. 5). A typical 'deal' given to the customer

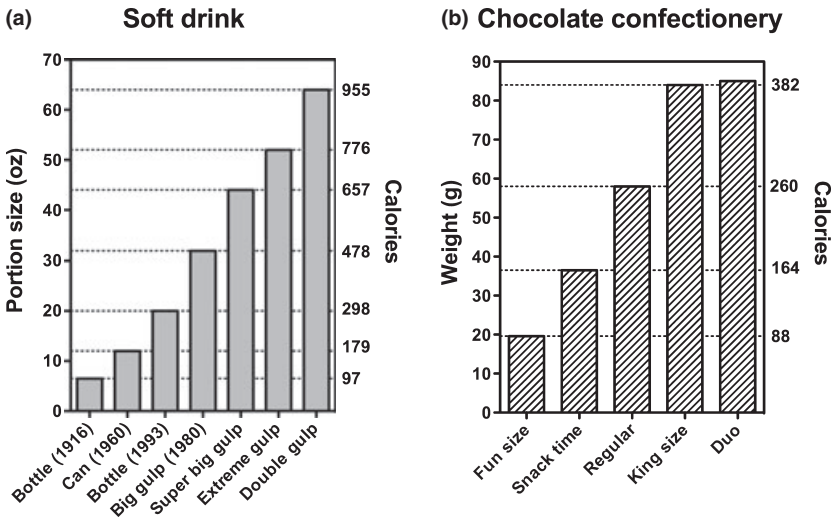

FIGURE 5 Portion sizes and calorie counts of common snack items. (a) Soft drink volumes and calorie content, based on portion sizes currently marketed (calculated without ice). Date of release of the item is included in parentheses where available. (b) Weight and calorie content of a typical chocolate confectionery item. Data available on-line at http://www.thecoca-colacompany.com, http://www.marsnutrition.co.uk/ and http://www.weightlossresources.co.uk/calories/calorie_counter/chocolate_sweets.htm.

is double the portion size for a small increment in price. The extra calories sold generate more profit for the manufacturer and vendor, while the consumer loses the capacity to regulate food intake and recognize what constitutes an appropriate portion. The profit is at the expense of population health. This phenomenon was exemplified in *The Times* newspaper, which pictured, on its front page, a child consuming popcorn at the cinema with the tagline 'Horror at the movies – the 1800 calorie bag of popcorn' [29].

Medical vs. social and political models

Within the medical world, there is a tendency to focus on disease precursors which are the signs of impending problems. These include reference to syndromes such as insulin resistance, dyslipidaemia and obesity. The medical model does not encourage an involvement in health promotion that embraces urban design, education, advertising, architecture and legislation. Politicians do not like the concept of a 'nanny state' interfering with free choice, so the toxic environment persists unchallenged [13]. The medical fraternity do not regard it as their domain, and government shies away from confronting big business. But opportunities do exist for the food industry to down-size. There is a huge and growing market for lower calorie foods. It amounts to legislative cowardice if authorities neglect to impose visible, universal and unencumbered calorie labelling and fail to negotiate a concordat on portion control.

The problem is compounded by a failure to face the problem as it is. Bubonic plague could not be stopped by wishing that the population were less degenerate. Nor can the diabetes epidemic be stopped by the World Health Organization (WHO), politicians and governments discussing 'non-communicable disease' or, as they prefer, 'NCD'. Which government is going to put the problem anywhere near the top of its agenda if the medical profession publishes papers such as 'Feedback on WHO global report on diet, nutrition and prevention of chronic diseases (NCD)'? [30]. The NCD terminology simply characterizes this vast problem as 'not something else'. Moreover, it is ludicrous to classify a global pandemic of diabetes and obesity as being 'not a communicable disease', when in fact the evidence is strongly in the opposite direction. Diabetes, like bubonic plague, *is* communicable. We must grasp this simple fact; the alternative is the supine view that it's everybody's fault and therefore no-one's responsibility. The food industry handle is still on the calorie pump of our worldwide Broad Street. The aetiological agent of the obesity/Type 2 diabetes epidemic is calorie excess, transmitted by the vectors of advertising and low-cost incentives, from a reservoir of accessible and cheap food outlets in an unconstrained toxic environment that predisposes to a sedentary lifestyle. The resulting overconsumption fuels the inevitable pandemic of obesity and Type 2 diabetes.

This is a problem which could be researched and addressed [31]. The necessity for clean air and unpolluted water are now routinely addressed by legislation and by meticulous surveillance. There is no public outrage that the government seeks to protect its population through legislation in other contexts; for instance, seatbelts in cars are now compulsory, public buildings must provide fire escapes, we have crash barriers on motorways and smoking in public buildings is not allowed. However, startlingly few public health or governmental measures are in place to protect or warn us about excess calorie consumption. Simple legislation could transform the spread of the epidemic, by insisting on judicious and explicit labelling of calorie content of food, enforcing a minimum size for such labels, addressing the issue of portion control and increasing public awareness.

Conclusions

Ultimately, the analogies between the Black Death and Type 2 diabetes are close. Both are pandemic diseases caused by agents and mechanisms which are now well understood. Preventative measures and judicious removal of reservoirs and vectors could halt the epidemic of Type 2 diabetes and transform the health of the world. Failure to act now can only result in a perpetuation of a tragic and unnecessary pandemic.

Competing interests

Nothing to declare.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Slides S1. Lecture slides from the Banting Memorial Lecture given in Liverpool in March 2010.

Video Clip S2. Video clip of Professor Sir John Bell, Regius Professor of Medicine, University of Oxford discussing the epidemic of diabetes in 2008.

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