Epidemiology of Obesity

1

W. P. T. James

Contents

Abstract

Obesity, although recognised millennia ago as an unusual feature and a societal handicap, only since the 1980s has it become a major clinical and public health problem. Originally a disease of affluence it became evident in poorer countries in the 1990s with children then showing increasing evidence of their excess weight gain with all its propensities to premature disease and death. Obesity rates are

W. P. T. James (✉)
London School of Hygiene and Tropical Medicine, London, UK
e-mail: Jeanhjames@aol.com

P. Sbraccia, N. Finer (eds.), *Obesity*, Endocrinology,
https://doi.org/10.1007/978-3-319-46933-1_1

rising rapidly in poor countries with clear evidence that many societies are more prone to obesity's amplification of diabetes and hypertension rates than in Western Europe and North American. These differences probably relate to the impact of poor fetal and early nutrition as well as infections on development and the epigenetic control of metabolism. The epidemic was precipitated by dramatic rises in the mechanisation and computerisation of labour, household work and home entertainment combined with a huge drive to market readily prepared high energy dense fatty, sugary and salty foods and drinks. Now dietary factors dominate global health burdens and obesity overwhelms health services with the global societal cost estimated as $2trillion a year, approximately the same as the cost of all warfare and conflicts. Only coherent government initiatives can reverse these burdens with little evidence so far of any appropriate national or international response.

Keywords
Anthropometric indices · Obesity · Morbidity · Mortality · Prevalence · Burden of disease · Economic cost · Prevention

Introduction

In this chapter there will be a focus on the overall societal patterns of obesity and how we are seeing a shift in the spectrum of the human body's structure linked to both immediate environmental factors and those long-standing effects which have led to both genetic selection and involved epigenetic programming as well as generational structural changes in the composition of the body with associated morbidity and epidemiological effects. The societal health burdens and their economic implications will be outlined before finally considering the implications for the key potential components of prevention stemming from this epidemiological understanding.

History

This has been exceptionally well covered by Bray (1997) who pointed out that there were images of obesity perhaps as deities in Europe from prehistoric times about 23,000–25,000 years ago with further images in the early agricultural period 5000–6000 years BC in Mesopotamia and later in Egypt by which time obesity was already seen particularly in the ruling classes and was considered objectionable rather than representing a wonderful god-like status. Chinese and Indian medicine also dealt with obesity as a problem condition before the Roman Galen distinguished between "moderate" and "immoderate" obesity. So for centuries physicians have sought to engage with the problem of obesity and its causes with attention paid to genetic factors by assessing the familial propensity to obesity and then twin studies. But it was Quetelet in 1835 who assessed man's size on a population basis by

developing the idea of standardizing people of different sizes by deriving the index W/H^2 in metric units, now termed the body mass index, whereas Livi later that century suggested the cube rather than the square of height should be used since weight reflected a three-dimensional being. This ponderal index was simply converted to a corpulence measure (not index) W/H^3 by Rohrer and is now often called the ponderal index.

BMI and Mortality Risk

The health implications of being too heavy were first made coherent with analyses of the mortality risk from the insurance industry in the USA published in 1915. Large tables were developed with weights and heights given in imperial units where weights corresponding to the appropriate low mortality range quoted for the individuals' height (with the men and women measured lightly dressed and wearing shoes). Repeated analyses by the Metropolitan Life Insurance Company followed with the analyses being divided at each height into adults of small, medium, and large frame sizes. By the 1960s population analyses were being assessed for the relative benefits of different indices, but by 1972 Ancel Keys, using some of his and colleagues' Seven Country data and other small surveys, concluded that the W/H^2 measure was suitable for general use. At the same time the BMI measure was being assigned to the USA insurance mortality data and checked in relation to the very large Build Study from the USA by a UK Government group (James 1976) which assigned BMI "normal" weights as between 19.1 and 24.6 for women and 19.7–24.9 for men, these BMIs corresponding to the lowest mortality values with obesity conventionally taken as 20% above the normal weight. These figures were then simplified by Garrow using BMI 20–25 as a normal BMI for clinical use as was then proposed by the US Fogarty Conference in 1973 (Bray 1976).

By 1983 the importance of a distinction being made between smokers and nonsmokers was evident in the analyses of the London Royal College of Physicians working party (Black 1983; Fig. 1) which showed that smoking men of normal weight had a mortality rate which was equivalent to that of nonsmoking obese men. It was also recognized that smokers were usually thinner than nonsmokers because smoking induces an increase in metabolic rate as well as reducing appetite (Dallosso and James 1984). Therefore ignoring the different relationship between BMI and mortality in smokers leads to the common finding that the lowest mortality rate seems to be when BMIs are about 27–30, because the thinner smokers in the lower BMI ranges have an increased mortality. Since then large detailed integrated multinational studies taking account of smoking and involving almost a million individuals from 54 international studies have confirmed that a BMI of 25 is an appropriate crude upper limit of normal or what should more accurately be termed "acceptable" body weights (Prospective Studies Collaboration 2009).

This approach to the health impact of excess weight assessed as BMI has for many decades been recognized as crude, and clinicians and body composition experts have always highlighted the crude nature of the correlation between BMI

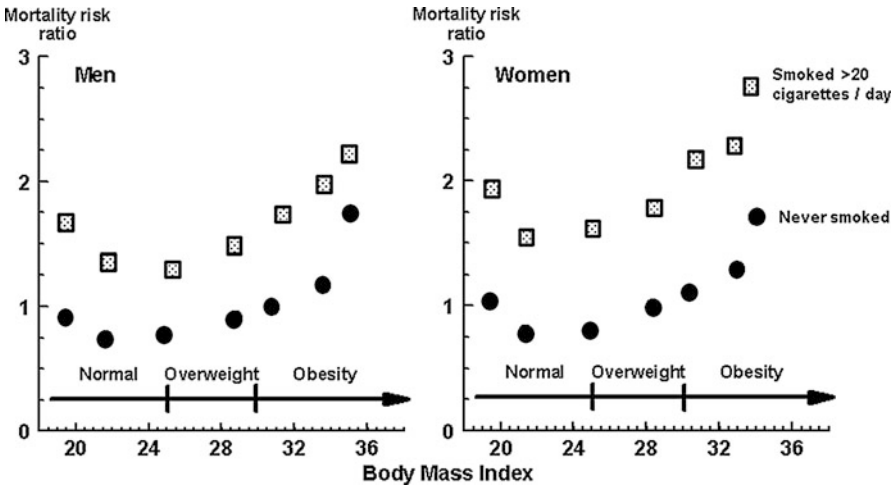

Fig. 1 The need to differentiate the effect of smoking and body weight on mortality. The mortality risk of those smoking >20 cigarettes/d (in red) is compared with those who have never smoked (blue) in relation to their respective body weights (Data taken from the London Royal College of Physicians Report on Obesity (see Black 1983))

and body fat. Ancel Keys and his colleagues were highlighting 60 or so years ago how the presence of blood pressure, smoking, and high blood cholesterol levels were far better predictors of deaths from coronary artery disease with the contribution of BMI making little extra difference (Keys et al. 1972). More recently the stronger association with other risk factors than with the crude measure of BMI together with assessments of any progressive organ damage has been shown to produce a far better classification of mortality (Sharma and Kushner 2009; Padwal et al. 2011).

BMI and Morbidity

The relationship between BMI and the health impact of excess weight has often been confused with the original BMI criteria relating to mortality risk. The "normal" range of BMIs, e.g., 20–25, does not give a suitable range for the lowest risk, for example, for hypertension, diabetes, coronary artery disease, or cancer, as these conditions not only depend on other environmental factors, e.g., salt in relation to hypertension or excess intakes of specific saturated fatty acids for coronary heart disease and a multiplicity of environmental factors leading to cancer, but also is roughly linearly related to BMI levels down to about 20. This implies that there are environmental factors which often combine to promote both weight increase and the concomitant disease, and indeed weight loss can often ameliorate the disease but whether this is the loss of body fat per se with all its hormonal and metabolic consequences or in part the parallel effects of the dietary changes needed to reduce the severity or impact of the concomitant disease is often not clear.

Nevertheless some of the conditions do seem to depend to a substantial extent on clear factors linked to body weight, e.g., the propensity to develop diabetes seems to relate to the duration of being overweight/obese as well as the magnitude of excess weight, (Abdullah et al. 2016) and reducing body weight has been found to be a critical factor in limiting the development of diabetes in those overweight/obese individuals at high risk of diabetes (He et al. 2015). Furthermore hypertension can be ameliorated by weight loss, and this may not just reflect the reduction in food intake and therefore sodium ingestion but also the change of diet with more potassium-rich fruit and vegetables. Nevertheless there are clear hormonal factors involving the angiotensin-renin system that also play a part.

Refining the Anthropometric Indices of Excess Weight and Their Relationship to Disease: The Value of Waist Measurements

For decades the importance of body shape as well as size as a predictor of disease risk has been highlighted with an original emphasis on the selective increase in the dimensions of the waist or hips. Then attempts were made to simplify clinical approaches by focusing on the waist measurements with the Scottish clinical management committee stimulating Han and colleagues to develop a set of waist measurements corresponding epidemiologically with the BMI 25 and 30 measurements in a population of young Dutch adults (Han et al. 1995). These values were incorporated tentatively into the first comprehensive World Health Organisation (WHO) report on obesity (WHO 2000) and have been widely used and incorporated, e.g., by the US National Institutes of Health (NIH) into appropriate body weight and shape standards relating to risk (National Institutes of Health 1998). Later the INTERHEART international study revealed that waist and waist/hip (W/H) ratios were a better index of the risk of coronary heart disease than BMI (Yusuf et al. 2005) with marginally better statistical if not practical predictability with the use of W/H values as increased hip values seem to be a protective of heart disease, perhaps relating to the body's ability to store fat safely. This has been repeatedly confirmed with some suggestion that waist for height in metric units with a simple ratio cutoff of 0.5 rather than hip circumference is a better predictor of disease risk factors, e.g., dyslipidemia, increased blood glucose levels, or higher blood pressures (Ashwell and Gibson 2016).

Broadening the Acceptable BMI Limits and the Greater Sensitivities to the Morbidity Impact of Weight Gain in Non-Caucasian Communities

By 1995 it had been accepted by WHO that the normal lower limit of adult BMI should be reduced to 18.5 in the non-Western world, i.e. the majority of the global population adults were much thinner but seemed healthy and able to sustain beneficial manual work, e.g., in agriculture at BMIs of 17–18.5, but evidence from South

American, African, and Asian analyses of morbidity showed an increased susceptibility to infections and time off work when BMIs were below 17.0. Mortality rates were observed to be increased when BMIs were below 16.0 so these became the BMI cut-off measures for undernutrition in adults (James et al. 1988; Ferro-Luzzi et al. 1992). The cutoff of 18.5 was chosen because it was shown that this seemed reasonable because populations with a median BMI of 20 had only about 10% of adults (but in practice with slightly more women than men) with BMIs of less than 18.5 and about 10% of adults with BMIs more than 25 (this time with more men than women). Yet concerns relating to obesity were still dominated by assessments in Western, i.e. European and North American, communities so when the international technical expert group met in WHO in 1997 to consider the problem of obesity (WHO 2000) the Japanese and other Asian experts' proposal to have the upper acceptable BMI limit reduced from 25 to 23, on the grounds that Asian communities were much more prone to the comorbidities associated with weight gain at much lower BMIs, was rejected. So, in the absence of coherent evidence to support the Asian proposition, the "acceptable" BMI range was maintained at 18.5–25.0. WHO then did hold an expert discussion in Singapore (WHO 2004) where attempts were made to see if one could define different societies by their relative body fat content in the usual BMI range of 18.5–24.9 as it seemed that many Asian communities had a smaller skeletal but a larger fat mass at the same BMI (Deurenberg et al. 2002; Deurenberg et al. 2003). However, it was recognized that not only were there few nationally representative data on the body composition of different ethnic groups but in addition Chinese children in Beijing and in Singapore had different body fat contents as did the rural and urban Thailand adults. This suggested that the differing body composition in similar ethnic groups was not an intrinsic ethnic feature but in some way reflected the response to some environmental factors. Nevertheless the WHO group in Singapore suggested that an upper normal BMI limit of 23 rather than 25 might be adopted by at least some Asian governments as the operational norm, and the Japanese and Indian governments now use these criteria. However, the newly formed Chinese obesity collaborative group led by the Prime Minister's advisor, Chen Chung Ming, concluded, after their own health analyses, that a BMI of 24 was most suitable in China (Chen 2008).

The basis for the concern that Asians in general were more sensitive to the comorbidities of weight gain already had been demonstrated in an earlier small UK study showing that the selected South East Asians were more prone to diabetes at lower degrees of obesity than British Caucasian adults (McKeigue et al. 1991). Then a major analysis of about 263,000 adults across Asia including the Chinese, Koreans, Japanese, Indonesians, Thais, and Indians showed that Asians, when considered as a group, were more prone to diabetes and indeed their waist or waist/hip ratios or waist/height ratios were better predictors of diabetes and hypertension than BMI with the Asians more prone to abdominal obesity at the same BMI (Huxley et al. 2008). This same phenomenon was then observed in Mexicans when compared with USA non–Hispanic whites (Sanchez-Castillo et al. 2005; see Fig. 2) and in the African diaspora. African Americans not only have higher BMIs than whites or Hispanics but their diabetes rates are even higher than one would expect for

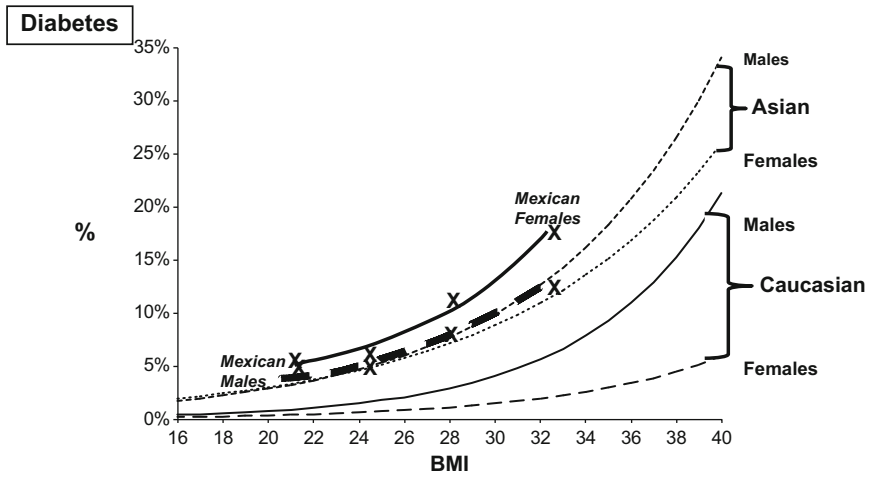

Fig. 2 The relationship between the BMI of Asians and Mexicans when compared with Caucasians. The comparison of the Asians with Caucasians (mostly Australasians) was set out by Huxley et al. (2008) and the representative Mexican data are derived from the 2000 national survey and compared with representative measured data from the age-matched US non-Hispanic white population (Sanchez-Castillo et al. 2005). The US data for non-Hispanic whites, used for the comparison with Mexican analyses, are almost identical to the Caucasian data shown and therefore have not been included in the graph

their greater size (Shai et al. 2006). Attempts to identify a genetic basis for this have so far been unsuccessful (Yako et al. 2016) with studies of the African diaspora also showing marked differences in glucose metabolism in different communities eating different diets and with objectively measured differences in physical activity (Atiase et al. 2015). However, studies even of the seemingly genetically obesity prone PIMA Indians from Mexico and Arizona in the USA show that with similar genetic profiles their dramatic national differences in BMI and diabetes prevalences were largely environmentally determined (Schulz and Chaudhari 2015) with very low obesity and diabetes rates in the hard working, home farming Pima Mexicans consuming a 25% fat, high fiber diet with a negligible sugar content (Chaudhari et al. 2013).

Secular Trends and Sex Differences in Obesity's Prevalence

Although, as noted earlier, adult obesity had been recognized as a clinical problem for centuries it did not emerge as a substantial health issue until the second half of the twentieth century when an appreciable number of middle-aged adults (usually women) started complaining about their inability to lose weight with doctors noting that they had a number of disabilities including back ache, arthritis, and breathlessness, i.e., comorbidities understandably linked to their excess weight. National or employee surveys in the UK suggested that the average BMI of men and women started to rise first in late middle age, i.e., the 50–65 year group from about the early

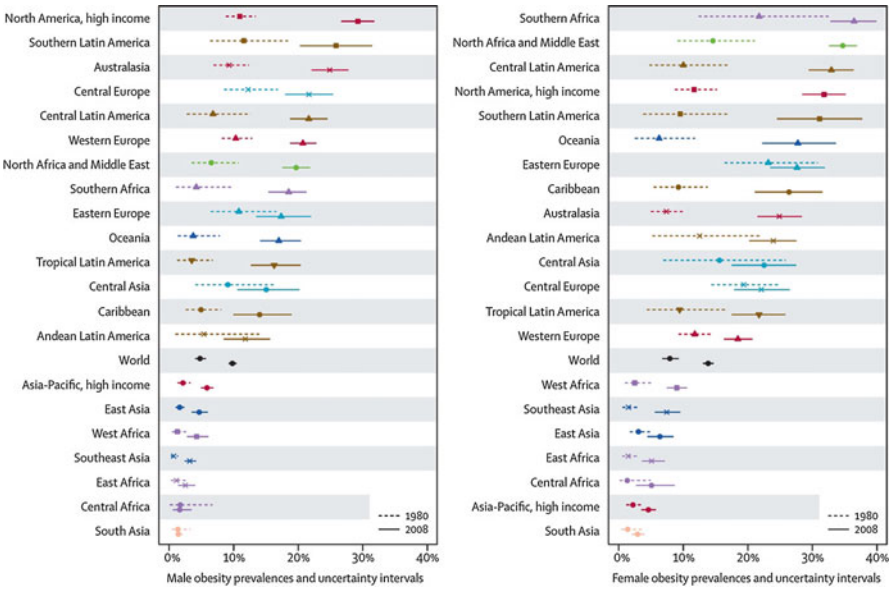

Fig. 3 The global epidemic of obesity in adults) Figure taken with permission from Finucane et al. (2011) showing the regional age standardized prevalences of obesity in men and women measured first in 1980 and again in 2008

1960s, but in the USA postwar data from the 1959 Build and Blood Pressure Study of the Society of Actuaries was already showing evidence of obesity although the terms for their definition at that time included adults with BMIs over about 27.5 and therefore would have included some more muscular males involved in the manual work common at that time. Internationally it was also clear that some societies, e.g., the Polynesian women and African women in South Africa and the Caribbean, already had high obesity rates in the 1960s by the time they were middle aged (Christakis 1975). By the 1980s, however, obesity as a public health problem was becoming very evident and since then there have been numerous studies and analyses across the globe showing the escalation of obesity globally (Finucane et al. 2011; Fig. 3). Note the sex differences in the responses to the new "obesogenic" environment and that lower-income regions are now often showing much higher obesity rates than Western Europe, for example, particularly in women.

Although there was widespread concern about the prevalence of obesity in the year 2000 recent analyses clearly show that the greatest increases in the prevalence of obesity are in proportion to the previous prevalences, i.e. countries with the highest prevalences have been showing the greatest increases (Dobbs et al. 2014; see Fig. 4). So clearly there is a need to understand the underlying forces for such a marked relationship. When national data are now considered in detail it also becomes clear that the populations in lower-income countries and particularly in Asia are currently seeing explosive increases in obesity with women's obesity rates usually exceeding those for men (Stevens et al. 2012). Although these sex

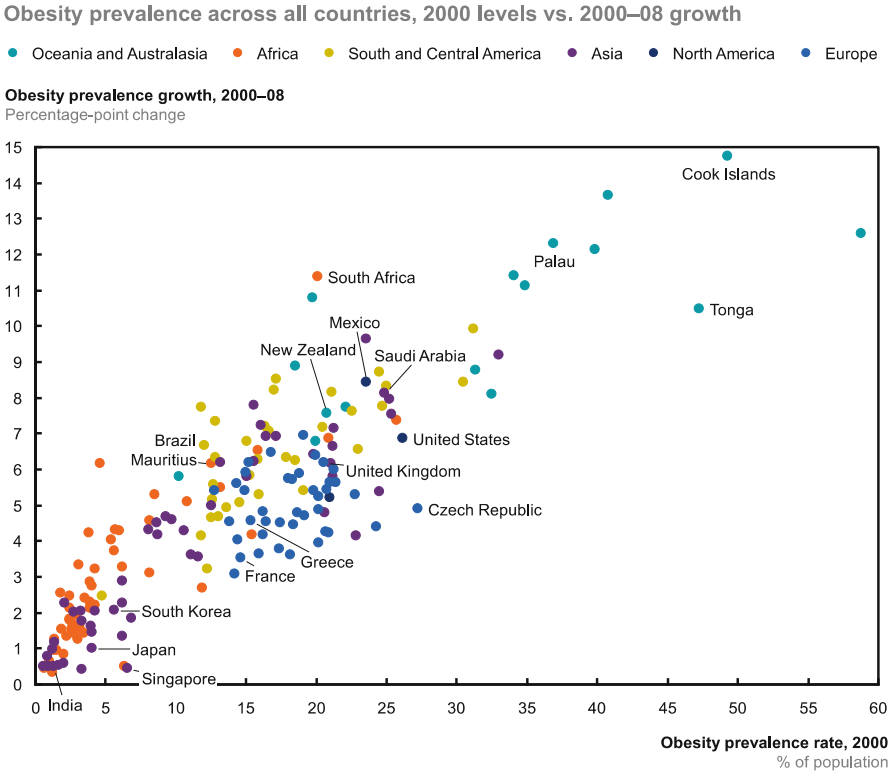

Fig. 4 Obesity prevalence growth has momentum: countries with the highest prevalence in 2000 experienced the most subsequent growth in prevalence. (Taken from the McKinsey Institute report on obesity (Dobbs et al. 2014)

differences in the propensity to obesity are usually ascribed to environmental factors, it is also evident that women are more prone biologically to develop obesity when energy imbalance occurs because their capacity to accrete lean tissue, e.g. muscle with its increased 24 h resting energy demands, means that sustained excess energy has to be accumulated as more fat in women (James and Reeds 1997).

Children's Criteria for Normal Weight Gain and Obesity

Childhood obesity was recognized early on in seemingly genetically distinct and often unusual cases but children in society as a whole only began to display marked weight gain from about the early 1980s. When the WHO group met in 1997 to deal with obesity in general it had to focus on adults as at that time there seemed no clear readily accepted definitions of obesity in children although an earlier WHO group, set up originally to deal with anthropometric issues relating to childhood malnutrition, had arbitrarily used the conventional WHO 2 SD cutoff points for designating

abnormal findings (WHO 1995). Therefore the International Obesity Task Force (IOTF) established a group which assessed the options and recognized that the use of the BMI as a measure of appropriate body proportions in children was crude. The choice of 2 for the power of height in the BMI calculations was shown not to be really appropriate except at about the age of 6 and ideally should have been different at younger and older ages (Franklin 1999). Nevertheless the BMI was agreed as the best simplified option, and the IOTF developed criteria by linking the percentile curves of BMIs of children from age 2 to 18 years to adult BMI cutoffs at the age of 18 with BMIs of 25 and 30 and then finding the corresponding percentiles for boys and girls at each age in an integrated set of nationally representative data for children from six countries where there were meticulous measurements of children at a time when obesity was not considered a problem, i.e., in early USA and British data, plus survey data from the Netherlands, Hong Kong, Brazil, and in addition in Singapore where there was some concern and where ideally these data should perhaps not have been included in the reference percentile curves (Cole et al. 2000). This set of age- and sex-specific reference points then made the analyses of population obesity rates coherent and multiple analysts have used these criteria for several years.

Assessments of childhood overweight and obesity prevalences, using the IOTF cut-points on representative or community surveys in Australia going back over a century[iii](38), clearly show that obesity suddenly emerged in the early 1980s in Australia and also in lower income countries after a short interval. The childhood epidemic is now evident in all 5 continents (see Fig. 5; Lobstein et al. 2015) and has continued to escalate particularly in poorer countries where there is little or no effort as yet to combat the problem.

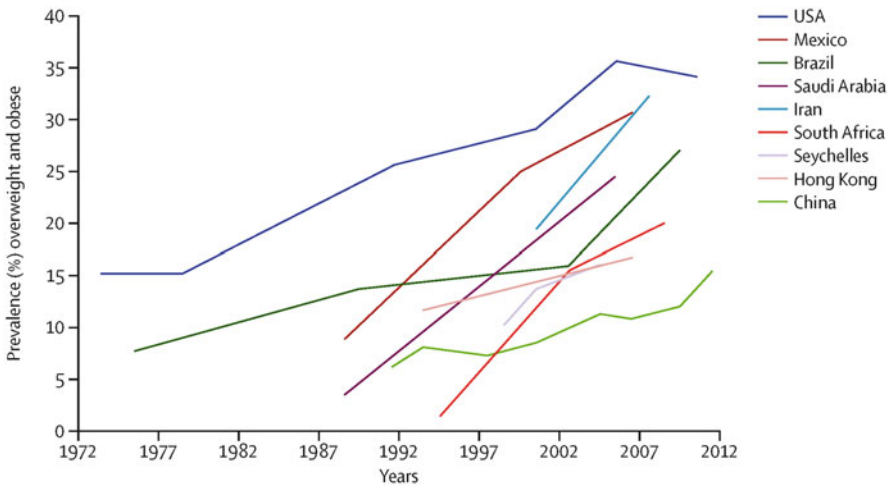

Fig. 5 The childhood obesity epidemic. New analyses of the emergence of obesity in children in lower-income countries compared with the USA (Taken from Lobstein et al. 2015)

More recently WHO established their own criteria for childhood overweight and obesity and used the birth to 5-year-old data from a meticulously organized six-nation international study of normal weight babes at birth who were then exclusively breast fed for the WHO specified optimum time of 6 months before being weaned onto appropriate diets and then followed up. The astonishing finding was the babes born in Norway, India, Oman, Ghana, Brazil, or the USA had amazingly similar growth rates with no discernable national differences at all (WHO 2006; WHO and Multicentre Growth Reference Study Group 2006). So these could now provide not just arbitrary reference cutoffs but a standard specifying how children anywhere in the world should grow optimally. Then WHO staff took adjusted old 1977 USA BMI values with some selection for 5–19-year-old children as the reference values so that the one standard deviation (ISD) of the USA data reference corresponded to BMIs of 25 at 19 years and the 2SD BMI value was about 30. Unfortunately WHO then specified as "overweight": only those children below 5 years with BMIs >2SD above the median whereas this "overweight" designation from 6–19-year-old was so designated when the BMI was above >1SD not 2 SD, with the latter limit now being designated as "obese" and therefore roughly corresponding to a BMI of 30 when adult. The differences of the new WHO BMI reference points and those of the IOTF and CDC seemed small, but the WHO approach has been heavily criticized by Cole and Lobstein who also developed a complete profile of percentiles corresponding to all the degrees of overweight and obesity as well as underweight designated for adults by WHO but based on their six nationally representative global data sets (Cole and Lobstein 2012). On this basis the different degrees of childhood obesity and underweight can be calculated for all societies.

Intergenerational Amplification of Obesity and Cohort Effects on Childhood Obesity

The probability that an overweight or obese child remains in the same category when adult rises markedly the longer they remain too heavy as adolescents. This then amplifies their future risk of both diabetes and cardiovascular disease (Baker et al. 2007). More recently it has become evident that mothers entering pregnancy when overweight and then putting on substantial amounts of weight in pregnancy are more likely to produce larger babies who then more readily become overweight in early childhood with further tracking of the excess weight into early adult life (Institute of Medicine 2009). This then means that cohorts of bigger babies and more overweight children are now emerging and one can now distinguish between the cohort effects and the impact of an adverse "obesogenic" environment in each age group in a population study – see Fig. 6 (Allman-Farinelli et al. 2008). However, when these overweight children/adolescents mature and themselves conceive there is then developing an intergenerational cycle of increasing childhood obesity within a population and this problem is going to be very difficult to reverse. These analyses are now being applied in several countries, e.g. the USA, (Reither et al. 2009) and are emphasizing the impact of early fetal changes reflecting the epigenetic and perhaps physiological

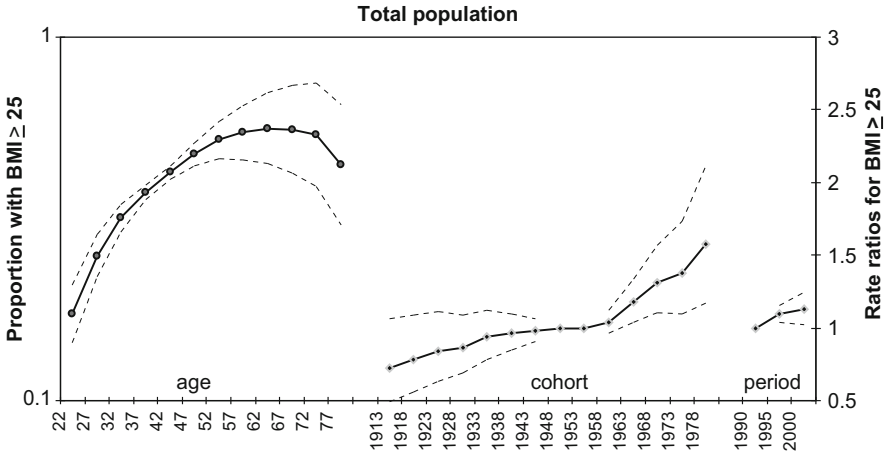

Fig. 6 An analysis of repeated surveys of children in Australia analyzed to distinguish between the age relationship (in months), the cohort dependent, and the secular (period) effects on the degree of overweight (Taken from Allmann-Farinelli et al.) On the *left* is the age (in months)-related increase followed by the cohort effect which is set out in terms of the cohorts born in different years (between 1913 and 1978) and on the *right* is the period effect showing the change in impact of the environment between 1990 and the year 2000

programming of generations at the early phases of fetal development, particularly during the setting of the trajectory of fetal growth. The basis for these epigenetic changes is now under intense investigation but these changes imply that the physical as well as the metabolic and nutritional state of young women is also very important at the time when they conceive. These findings also imply that the future epidemic may only be prevented long term if we focus on the well-being and BMI status of young women as a whole. Given the prevalence of unplanned pregnancies the general well-being of young women within the whole population becomes important.

This intergenerational effect not only relates to a successive amplification of the maternal overweight problem but studies from lower-income countries show that maternal malnutrition also has profound effects on the fetus with, in the Indian subcontinent, a reduction in the growth of lean tissues within the fetus with an excess body fat content even if the baby is born small (D'Angelo et al. 2015). There is also a marked tendency to abdominal obesity with its amplified risks of glucose intolerance and hypertension (Yoo 2016). This maternal malnutrition therefore may in part explain the ethnic differences in the propensity to diabetes on weight gain in different communities.

The Burden of Disease Associated with Obesity

The handicaps associated with obesity are many with several more continuing to be added as careful studies document the extent of the obesity handicap. So, for example, it is becoming clear that obese individuals are more susceptible to

infections sometimes for mechanical reasons, e.g., their greater difficulty with breathing, increased gastric reflux leading to lung infections, and their greater skin infection problems. In addition there seem to be changes in immune function with a greater intrinsic susceptibility to the acquisition of an infection and to the development of greater complications when infected with a greater resulting need for more frequent hospitalization. So there is an increased risk of urinary tract infections, of gastric helicobacter pylori infection, and pancreatitis as well as a greater risk of severe infections in obese subjects when suffering from trauma. Objectively the failure to mount an appropriate immune response is also shown by the far poorer antibody responses to vaccination with most vaccines although the response to influenza vaccine is not necessarily poorer in the obese (Tagliabue et al. 2016).

In addition to a greater susceptibility to infections in obesity there is also the well-documented increased risk of developing diabetes, hypercholesterolemia, and high blood pressure with therefore an increased propensity to coronary heart disease and strokes (see ▶ Chap. 11, "Obesity, Hypertension, and Dyslipidemia"). These increased risks are amplified by the usual finding that overweight/obese individuals also have a poor diet which also influences their tendency to increased blood uric acid levels and the risk of gout. There is also an increased likelihood of nonalcoholic fatty liver (see ▶ Chap. 4, "Roles of Gut Hormones in the Regulation of Food Intake and Body Weight" Van Gaal) and when sugary diets are consumed of more dental caries.

Weight Gain, Obesity, and the Risk of Several Cancers

It has also long been recognized that excess weight gain increases the likelihood of developing several cancers. These have been carefully characterized by the World Cancer Research Fund (WCRF) in exhaustive and systematically updated epidemiological analyses (World Cancer Research Fund/American Institute of Cancer Research 2007) backed usually by an understanding of a clear plausible mechanism. Some cancers are considered "convincingly" linked to excess weight gain and others are "probably" linked (see Table 1). The degree to which one can be confident that these relationships apply biologically was in practice based on the consistency of multiple cohort studies. This approach is, however, fraught with problems of interpretation, because they depend on the accuracy of dietary measures such as food frequency questionnaires often made many years beforehand and even if repeated are very subject to systematic as well as random measurement errors as well as secular changes in eating habits. Few long-term studies are available with biomarkers of dietary intake. There are the additional problems that when comparing two groups with different diets there may be several other characteristics that in part explain their different outcomes, and it is not always possible to identify these adequately. There is also the issue of how to cope with the intrinsic biological differences in the way in which individuals respond to the same intake or change in diet. This has long been recognized as exceptionally important when considering, for example, the blood low density lipoprotein (LDL) cholesterol responses to a

Table 1 The World Cancer Research Fund/International Cancer Research Fund analyses of the relationship between excess weight gain and its effect in inducing cancers

Convincing evidence of weight gain inducing cancers of:
Esophagus (the adenocarcinoma type associated with gastric reflux)
Pancreas
Colorectum
Postmenopausal breast
Endometrium
Kidney
Liver
Advanced prostate cancer
Evidence of a probable induction of cancers of:
Gallbladder
Ovaries
Cardia part of the stomach
Probable decreased risk
Premenopausal breast
Cancer survival:
Increased risk of mortality in premenopausal and postmenopausal women when overweight or obese once breast cancer is diagnosed

Data taken from the original WCRF/AICR 1997 (Yoo 2016) analyses but updated from their continuing analyses see: http://www.wcrf.org/int/research-we-fund/continuous-update-project-findings-reports

defined intake of saturated fats where some individuals will show a fivefold greater increase in LDL cholesterol levels than others. Therefore a cohort study with perhaps at most a two to threefold range in diets within a community usually is unable to show a relationship between saturated fatty acid intakes and coronary heart disease even when it is clearly established that an increase in LDL cholesterol is causally linked to the development of coronary heart disease. This is but one example of the range of individual metabolic responses to the same intake of many different nutrients, these responses being determined by both genetic factors and the magnitude of enzyme systems which may in part be determined by the mass of that organ conditioned by physical activity in the case of muscle or by a sustained change in intake inducing a substrate amplification of the relevant pathway. So the problem with the analysis of cancer risks is that we do not often have a really good understanding of the causal mechanisms so we can see which environmental factors interact with this mechanism and either amplify it or inhibit it.

The magnitude of the potential environmental impact on the development of cancers can be seen when one compares the 10-fold differences in the age- and sex-matched differences in the incidence of, for example, breast and colorectal cancer in Japan versus the USA when first measured 50 years ago by cancer registries. Furthermore studies within Japan showed a fourfold increase in colon cancer over a period of 30 years, and migrant studies also clearly show the increasing propensity to both breast and colon cancer when Japanese migrate to the USA. Subsequent

Table 2 World Cancer Research Fund updated estimates of preventability (PAF%) of cancers of which body fatness is a cause in the UK, USA, China, and Brazil

Cancer type	USA		UK		Brazil		China	
	Male	Female	Male	Female	Male	Female	Male	Female
Esophagus (adenocarcinoma)	37	30	35	20	26	14	19	7
Stomach (cardia)	18	27	18	20	13	14	10	8
Pancreas	17	20	14	16	8	13	5	10
Gallbladder	11	28	8	21	3	15	2	10
Liver	27	28	22	19	11	13	6	7
Colorectum	17	15	15	13	10	11	8	9
Breast (postmenopausal)	–	17	–	16	–	14	–	12
Ovary	–	5	–	4	–	3	–	1
Endometrium	–	50	–	38	–	5	–	4
Prostate (advanced)	11	–	9	–	5	–	4	–
Kidney	20	28	17	21	10	16	6	10
Total of these cancers	21	21	16	17	12	14	12	10

Using numbers of new cases of cancer diagnosed annually from GLOBOCAN 2012 for both men and women combined this translates to about 117,000 cases of cancer in the USA, about 23,000 for the UK, about 17,000 for Brazil, and about 99,000 for China being preventable if everyone had a healthy weight

Based on the WCRF 2009 approach but updated see: http://www.wcrf.org/int/cancer-facts-figures/preventability-estimates/cancer-preventability-estimates-body-fatness

generations display cancer rates which increasingly converge with those in USA Caucasians. Yet in cohort studies we only seem to be able to discern a 50% increased propensity to breast or colorectal cancer with particular diets. This probably means that not only are the dietary studies flawed but also that we have not begun to take account of the differences in the propensity of different individuals to have a fivefold or more differences in those reactive metabolic processing pathways which lead to cancer for reasons both genetic and epigenetic relating to both paternal and maternal environmental factors. So we probably have grossly underestimated the dietary and perhaps the BMI-related effects on cancer propensity. Table 2 illustrates the results of an approach to distinguishing the contributions of an increase in BMI to the risk of the cancers in different affluent and middle-income countries based on the approach set out by WCRF in 2009 (World Cancer Research Fund/American Society for Cancer Research 2009) but now updated on the basis of new analyses.

Functional Impairments

Although it has long been recognized that diabetes, hypertension, and some cancers are much commoner in the overweight and obese, it is the constraints on people's

mobility that they first notice with their very high prevalence of backache and the greater extent and degree of arthritis especially of the knees, hips, and ankles induced at least in part by their excessive weight gain. However, there is also evidence that arthritis of the hands is more common – perhaps another sign of the impaired immune response in the obese. The pain on movement and patient's breathlessness explains a substantial part of their everyday immobility and distress.

However, there is the additional mental burden often induced by their sense of failure to reduce weight accompanied by the widely recognized public disapproval in affluent societies. Candidates for a job appointment or for promotion within almost any field are likely to fare worse if they are overweight or obese. Obese individuals have therefore, perhaps not surprisingly, been documented to be less productive when at work and to have more time off work. They also often feel depressed and suffer from a greater sense of isolation from society. To add to their personal burden they are likely to retire early, to remain isolated from society, and to suffer earlier the first stages of brain aging with earlier signs of cognitive decline with later dementia in part seemingly related to the brain's considerable sensitivity to insulin (Kullmann et al. 2016) and the increasing brain insulin resistance as weight gain occurs. This brain insulin resistance brings functional handicaps which are also evident in those overweight/obese individuals who have progressed to type 2 diabetes. Patients with type 2 diabetes display impaired mental performance in almost all neuropsychological tests with the greatest impairments being found in memory, information-processing speed, and executive function. These problems are in part reversed rapidly with bariatric surgery (Handley et al. 2016) implying that the insulin resistance effect may be important. However, there is also more progressive brain atrophy with aging with obesity, and the accompanying impact of atherosclerotic changes in the cerebrovascular circulation contribute substantially to the progressive cognitive decline in obesity.

Calculating the Burden of Disability and Premature Mortality

The standard approaches used by WHO and others to estimate the overall burden of disease in a society involves calculating the number of years of life lost (YLL) by premature deaths. This was originally taken by WHO as the number of years lost before the age of 75 years, but more recently Murray and colleagues in Seattle (GBD 2015 DALYs and HALE Collaborators 2016) have simply taken the longest life expectancy of any group of more than 5 million within a particular geography. To these YLL lost can then be added the years during which individuals were handicapped by disabilities to give the total number of years of disability and yours of premature death. This sum is called the Disability Adjusted Life Years (DALYs) lost. Then the proportion of the total DALYs that are accounted for by different diseases is estimated. In practice, the DALY calculations of the impact of, for example, diabetes or coronary heart disease usually deal with each risk factor separately without accounting for how these risk factors might interact in a synergistic or inhibitory manner with amplification or a reduction in each factor's impact

on the total burden of disability. So the total of the factions of DALYs accounted for by diabetes, heart disease, and cancers, etc., usually adds up to over 100% of the observed total DALYs but allows one to rank the importance of different conditions in any one society.

This estimation of DALYs attributed to different diseases does not take into account the risk factors such as weight gain, high blood cholesterol levels, and high blood pressure which are simply seen as risk factors and so are not displayed in the DALY calculations which in any case do not include the whole range of functional disabilities noted above. However, separate analyses can be made for risk factors where one assigns a proportion of a disease which is attributable to a particular risk factor with the estimates usually based not on clear clinical trial data but on inferences from cohort studies.

When these BMI analyses were first included in a risk analysis by WHO for the Millennium analyses of risk on the basis of a need to estimate the extent to which the disease burden was preventable, it was necessary to identify the optimum levels of each risk factor. Thus for blood pressure the optimum systolic blood pressure was not the clinical cutoff of 140 mmHg but 115 mmHg, for total cholesterol levels it was 3.8 mmol/L (later rounded up to 4.0 mmol/L), and for smoking it was to have never smoked. Similarly for the BMI the optimum weight status on a global level was a BMI of about 21 (which was later changed by US investigators to a BMI of 23 perhaps because they could not cope with the implications for the USA of a global standard set on the basis of mortality, morbidity, and functional criteria). A high blood glucose and diabetes were not set as risk factors at that time because the global evidence on its prevalence was inadequate, but with the accumulation of data blood glucose was added with minimum optimum values for health being set at 5.3 mmol/L (Afshin et al. 2015) whereas WHO in a more cautious mode has taken a higher glucose value of 7.0 mm/L (World Health Organisation 2014). Originally WHO using these optimum values showed that the top risk factors for the DALY burden of what they then termed "developed" countries in descending order were smoking, high blood pressure, alcohol consumption, and a high blood cholesterol with overweight coming in as the fifth biggest risk factor for the whole disease burden in these relatively rich countries (World Health Organisation 2002). The point that cholesterol levels and blood pressure are magnified by obesity and smoking reduces the obesity rate was not explicitly considered. Nevertheless in the so-called low mortality developing countries, i.e., representing a variety of countries including, for example, Mexico, South America, and the Caribbean as well as many other countries, e.g., China, overweight was still the fifth biggest risk factor. So this was the first time that WHO and national governments really recognized the magnitude of ill-health stemming from the problem of overweight and obesity.

Since then the Gates funded Seattle/Boston/London group, often collaborating with WHO, has assessed both the disease burden and the accompanying risk factors in different parts of the world with updates being made on a continuing basis. Analyses of the disease burden usually still consider obesity as a risk factor not a disease outcome, but more recently the risk factor analyses have been extended to include dietary factors and physical inactivity based primarily on the authors'

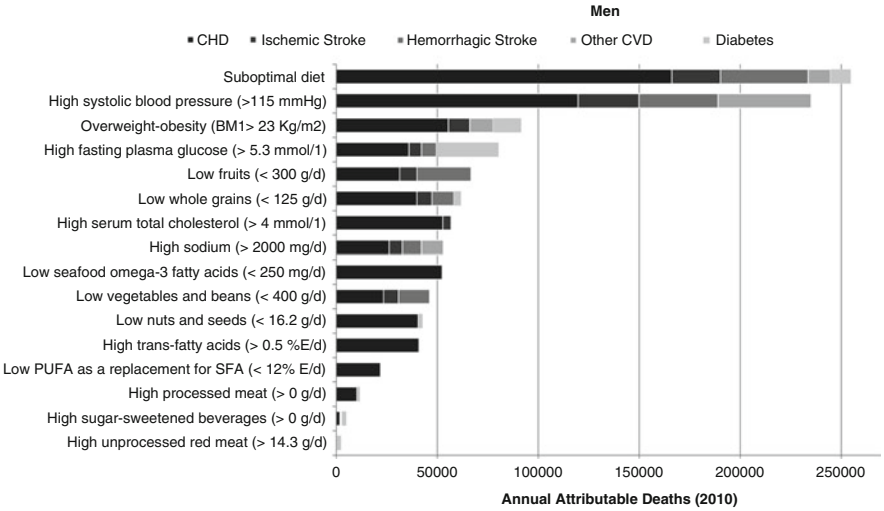

Fig. 7 The impact of different risk factors on the mortality rates from diabetes and cardiovascular disease in the Middle East and North Africa (Taken from Afshin et al. (2015))

interpretation of the proportion of risk factors accounting for particular diseases. These involve meta-analyses of cohort studies on diet, physical inactivity, and disease combined with a huge collation of dietary studies and then complex mathematical analyses of the proportions of risk attributed to the different risk factors. The estimates also often involve a crude extrapolation to countries where dietary measures and disease data are either hopeless or nonexistent. With these major caveats in mind Fig. 7 shows more recent analyses relating in this example to the Middle East where excess weight gain is the third most important risk factor after a suboptimum diet and high blood pressure.

Economic Impact of Obesity

These functional and societal handicaps impose a burden that can now be quantitated by economists in financial terms and these so-called indirect costs add to the widely recognized increased direct costs of medical services through the cost of medical consultations in the community, hospitalization, and the cost of any pharmaceutical treatments provided at home. Doctors, familiar with the immediate costs of really heavy patients, usually do not realize that the incremental costs of obesity are evident even in the overweight group, i.e., in those with BMIs of 25–29.9. So when analyses of the direct medical service costs are linked to the proportion of adults in a society with different degrees of overweight/obesity then the total direct medical costs of the overweight in a country are appreciable (Withrow and Alter 2011) accounting for about 1–3% of a country's total healthcare expenditures but with obese individuals costing about 30% more than their normal weight peers. Figure 8 shows, however,

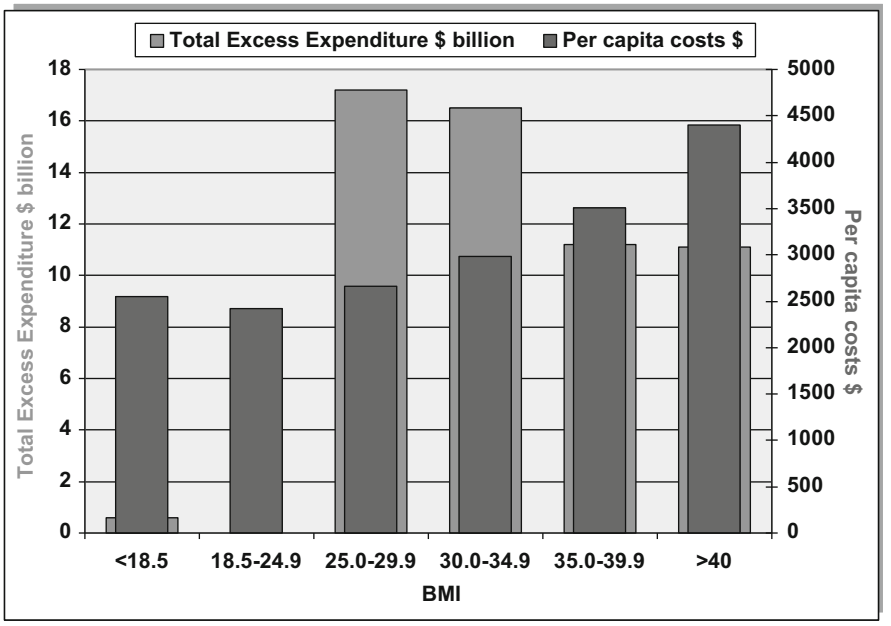

Fig. 8 The cost of different degrees of excess weight in the USA. The data on the average annual medical costs of adults with different BMIs is taken from Arterburn et al. (2005) but then the prevalence of the distribution of BMIs was found from NHANES statistics corresponding to the same time. The prevalence multiplied by the individual cost of each BMI group gives the total national burden in $billions per year for each BMI group

that if we take some crude USA estimates of the direct medical costs of individuals of different weights then there is a progressive increase in costs from a normal weight status to extreme obesity (Arterburn et al. 2005). Then if one considers the prevalence of these different degrees of overweight on a national basis at the time of the cost analyses then the total costs for a country from adults just being overweight with BMIs 25–<30 then the small incremental personal cost for the overweight individual translates on a national basis to a substantial health care cost. These costs are either paid personally by the large numbers of individuals or by the state if there is a national health service. This means that if the state is directly or indirectly responsible for the health costs then clearly a focus only on the most obese cases is inappropriate and the actual costs of being overweight should not be neglected.

Most analyses of the costs associated with obesity reveal, however, that the indirect economic costs of disability, absenteeism, and early retirement are even greater than the direct medical costs and amount to about 60% of all costs in advanced economies (Dee et al. 2014). Fewer estimates of the economic costs of obesity have been made in lower-income countries, but in many countries most individuals cannot afford medical consultations, tests, or drug therapy so their condition is neglected until they incur serious illness with its major costs. In poor countries ill health then induces poverty not only because people are unable to work

and have no welfare benefits but also in addition they usually have to pay for the costs of their treatment (World Bank 2014). In India and several other Asian countries these costs have long been known to induce catastrophic debts with households effectively locked into intergenerational repayments of debt in a manner akin to slavery. The amplification of risks from diabetes exacerbated by even modest weight gains therefore becomes important in the economic analyses of different health systems in Asia, Mexico, and probably in many other countries where the propensity to weight gain with additional sensitivities to diabetes, hypertension, and other noncommunicable diseases is rising rapidly.

The burden of disease in more affluent countries is also usually greater in the lower socioeconomic groups and the healthy life expectancies differ between the rich and poor by up to 20 years (Marmot 2010). The McKinsey Institute (Dobbs et al. 2014) also estimated using OECD statistics that the societal burden of obesity usually ranked as one of the top five social burdens in both rich and middle-income countries. Using the disease burden analyses on a global basis and recent analyses of obesity's economic costs based on World Bank data, the McKinsey Institute estimated the total global economic costs of obesity as $2trillion per year – only just below the $2.1 trillion costs of smoking and all armed conflicts and terrorism in the world.

The Drivers of Obesity: Epidemiological Implications for Population Prevention and Economic Benefits

Analyses of the health costs of obesity illustrate the dimensions of the challenge because although the focus of the public and of policy makers is on how to prevent obesity in children it becomes clear that a reduction in health costs becomes evident in a society within months if the number of overweight and obese in the adult, not children, population could lose weight whereas combatting childhood obesity brings economic benefits only about 40 years later if one considers the major costs, e.g., of diabetes as one of the great medical expenses relating to excess weight gain. This is shown in Fig. 9 taken from the UK Chief Scientists Foresight obesity analysis with its microsimulation studies involving actual health costs of diabetes in England (Foresight 2007). To prevent any further increases in total health care costs relating to obesity would require the average BMI of the adult population in England to fall by 8 BMI units i.e. bringing it back to an average of BMI 20-21, a fiigure which matches the original analysis of the optimum BMI for a population. So given this perspective and the well-recognized continuing escalation on obesity rates globally in adults, one can consider which major risk factors are or were responsible for the epidemic from an epidemiological point of view and then use a variety of analytical methods to quantify the potential impact of different measures.

Clearly a marked reduction in physical activity has occurred over the decades, and this automatically means that we need far less food to maintain energy balance. This secular change in demand may have amounted to an average reduction of 500–1000 kcal or more per day, and if we consider the old data from the Baltimore

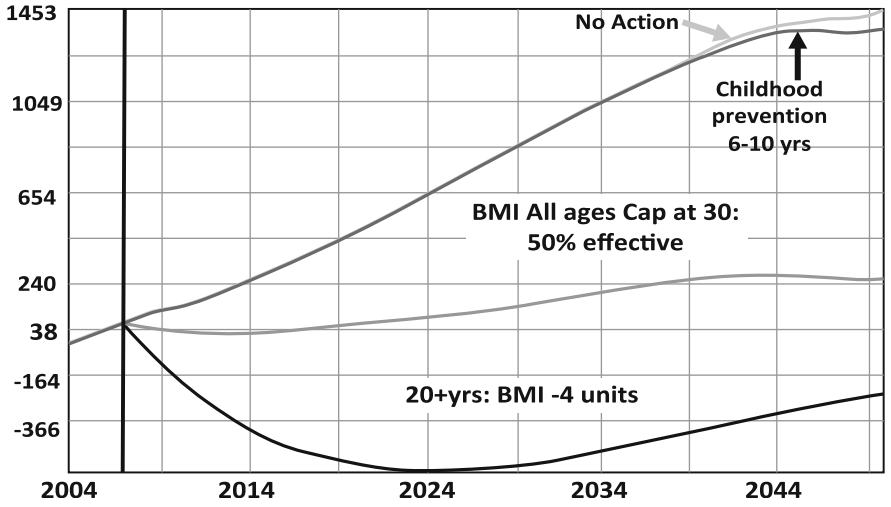

Fig. 9 The predicted future health care costs of diabetes in £millions from 2004 to 2050 in England in relation to the potential changes in the prevalence of obesity induced by different strategies either in children or by reducing the obesity rate by 50%, i.e., with a cap on the number of those with BMIs30+ or by having all adults reduce their BMI by 4 units (Taken from the UK Foresight report on obesity 2007)

aging study where they monitored men at the age of 25 and 70 years and then use current updated analyses of different ranges of physical activity (see Fig. 10) the fall in energy expenditure with age is dominated by the reduction in activity in sport and general activity (James et al. 1989). There were some early secular changes at that time in the USA as well as the very small intrinsic aging effect, but the overall cause of the decline in energy needs is the age-related fall in general and sports activities. So adults needing to maintain their energy balance will have to either subconsciously or deliberately reduce their food intake by anything from 500 kcal–1800 kcal/day. Most men are now by the age of 25 years only undertaking moderate activity so the average man of 70 kg would still need to reduce their intake by about 1200 kcal/d over their life time with similarly active average women needing to reduce their intake by perhaps 800 kcal/d.

This implies the need to rethink the whole strategy for maintaining physical activity throughout life. But the mechanization of work is unlikely to be reversed as it brings huge economic benefits and the idea of removing all the household aids that minimize work in the home would mean many more hours of housework and would be totally unacceptable in most if not all societies. Urban design to amplify walking, cycling, and minimize the public's use of cars for everyday activity is now seen to be valuable as is the provision of parks and other spaces and facilities for leisure time sports (Sallis et al. 2016). Nevertheless the major focus needs to be on factors that promote unnecessary food intake.

Detailed analyses of the factors promoting weight gain were set out by WHO in 2003 (WHO 2003) and are set out in Table 3 with additional updates by WHO on the

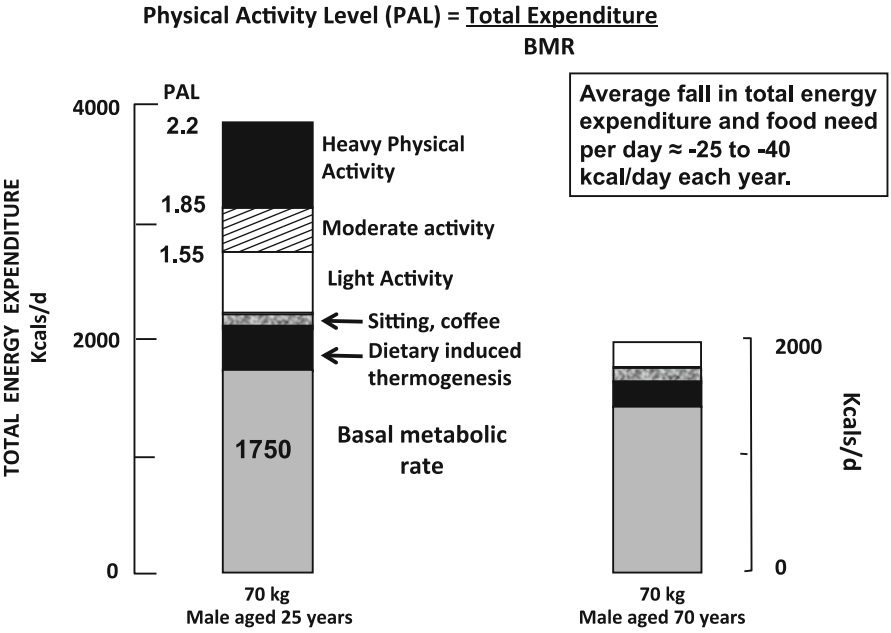

Fig. 10 The fall in energy needs with age as shown by repeated measures of body weight, body composition, and basal metabolic rate in the same men when 25 years and 70 years of age. (Data adapted from the USA Baltimore Ageing Study but preserving the body weight as constant to illustrate the aging effects). The different degrees of potential energy needs were taken from the original FAO/WHO/UNU 1981 analyses of energy requirements at different activities and the impact of reducing physical activity as observed with aging: so a 1000 kcal–1800 kcal fall in daily energy expenditure occurs from youth to old age depending on the degree of physical activity so if the young men were moderately active the fall in energy needs is equivalent to the need to reduce energy intake by about 30 kcals/day each year on average, but this may vary from about a 25 kcal to 40 kcal/day decline each year from the age of 25 years to 70 years (Adapted and redrawn from James, Ralph and Ferro-Luzzi (1989))

need to restrict sugar intakes (WHO 2015) and on the basis of their recent updated systematic analyses to reduce total fat intake (Hooper et al. 2015). These careful analyses relate to the original WHO proposition that the foods' energy density was the key to promoting inadvertent, i.e. "passive overconsumption" of kcalories. This emphasis on energy density fits with the recent UK government's scientific advisory committee on nutrition highlighted the need to substantially increase the intake of dietary fiber (SACN 2015). WHO highlighted the effects of food marketing on energy intake and more recently in the UK Public Health England (PHE) has found that 40% of all foods purchased are in response to special marketing promotions which almost always involve the promotion of high fat, high sugar, and salty foods as well as sugary drinks (Public Health England 2015). The traditional approaches of trying to induce behavior change in a population by health education has been repeatedly tried for over 30 years and has clearly failed even if backed by subtle techniques such as the "nudge" manipulation of purchasing circumstances

Table 3 The causes of excess weight gain and obesity as assessed by the World Health Organisation based on its original 2003 analyses but updated by their new expert analyses of the impact of total fat and free sugars on the propensity to weight gain

Strength of evidence	Decreases risk	No relationship observed	Increases risk
Convincing	(a) regular physical exercise (b) high NSP (dietary fiber) intake		(a) High intake of energy dense, nutrient poor foods. (New confirmatory analyses[a]: high total fat and sugar intakes) (b) Sedentary lifestyles (c) Heavy marketing of energy dense foods[b] and fast food outlets. (d) Adverse socioeconomic conditions in developed countries (especially for women) (e) Sugar sweetened soft drinks and fruit juices
Probable	(a) Home and school environments that support healthy food choices for children[b] (b) Promoting linear growth (c) Breastfeeding		(a) Large portion sizes[c] (b) high proportion of food prepared outside the home (western countries)[a]
Possible	(a) Low glycaemic index foods	Protein content of the diet	(a) "Rigid restraint, periodic disinhibition" eating patterns
Insufficient	(a) Increasing eating frequency		(a) Alcohol

Note:
[a]This table is set out as in the original WHO 916 report (2003) (World Health Organization 2003) except that new analyses by Hooper et al. (2015) for WHO have now confirmed the importance of a high fat diet and separate WHO (2015) and (SACN 2015) analyses, with systematic reviews of total free sugar intakes, have also highlighted their role in promoting weight gain but still probably through an effect on the energy density of foods.
[b]This designates what the experts for WHO in 2003 considered was a reasonable set of judgments based on associated evidence and expert opinion. Since then further analyses usually support these propositions
[c]This signifies that portion sizes and the proportion of prepared foods outside the household has been moved up from a "possible" cause to a "probable" cause on the basis of more recent analyses

(House of Lords 2011). This in part is because no government can match the sophistication, intensity, pervasive, and endlessly repeated effects of marketing by food companies and supermarkets (Cohen et al. 2015). These approaches involve a variety of remarkably subtle and well-researched methods including the development of methods that help to evade normal conscious decision-making. Such techniques also involve the constant siting of food outlets and vending machines to stimulate impulse buying, the manipulation of subconscious registered eye catching labels, the length and position of each item's display in supermarkets, and the

unrecognized increasingly routine use of synthetic flavors chosen on the basis of molecular responsive laboratory plates incorporating huge numbers of distinct olfactory receptors. These distinct flavors have been shown experimentally to appeal separately to men and to women including those taste preferences in young women at different physiological phases of their menstrual cycle. This as well as brain imaging techniques to identify those flavors which trigger the brain's pleasure sensors in effect amplify the pleasurable experiences of the foods and therefore their chances of being repurchased. These often-unrecognized marketing techniques are very difficult to combat by any policy process.

The other major factors impacting on food choice and the magnitude of food intake involve marketing by offering lower priced products. Although in the medical field this has not received much analysis, economists for decades have understood and estimated mathematically the impact of price changes on purchasing habits. This price elasticity was used to vary the subsidy or tax on specific foods and therefore the consumers' choices. This then systematically changed the consumption and reinforced the costs of eating more or less fruit and vegetables as well as meat and fish. These items have proved to be very price responsive whereas the intake of fats and oils and sugary products are less readily affected by small price changes.

The actual ranking of the price of these different foods has changed substantially over the decades in large part because subsidies induced major changes in the primary cost of commodities. Thus farm prices have traditionally been dramatically affected by multibillion-dollar farm subsidies in most parts of the world but particularly in the USA and Europe where subsidies have differentially favored meat, fats, oils, and sugar production with horticultural products receiving much less favored treatment. This has led to major changes in commodity prices with new calculations demonstrating that to purchase a healthy diet costs a household about three times the cost of buying cheaper fat and sugar rich foods with few vegetables and fruit (Wiggins et al. 2015).

As the production of fats, oils, and sugar has risen markedly and with it the total food energy being produced the farmers then have to do everything possible to sell their products to food manufacturers who in turn do their utmost to increase their volume as well as their price turnover in complex negotiations with supermarkets undertaking the same exercises. So the whole food chain in Western societies and increasingly in lower-income countries is locked into an intense effort to encourage people to buy more food when in practice they need to consume less. This relationship between the drive for profits and health relates to the tobacco and alcohol industries as well as the fast food industry (Moodie et al. 2013). It is little wonder therefore that there is a relationship between the total food kcalories available in a country and the development of obesity. So now in practice in the UK 30–40% of all household food purchased is discarded as food waste compared with about 2% in the straightened times of the 1950s.

To combat all these factors means that policies need to be developed right across different branches of government with the need for multiple steps rather than

assuming crudely that a single "magic bullet" will suffice. This has been emphasized repeatedly by many government sponsored analyses as well as by independent analysts such as the OECD (Sassi 2010) and the McKinsey Institute which estimated the strength of evidence and cost effectiveness of 60 different measures (Dobbs et al. 2014). If the United Kingdom were to deploy all 60 interventions, the analyses suggest that these multiple but modest measures could reverse the rising obesity rates and bring about 20% of overweight and obese individuals back into the normal weight category within 5–10 years with an estimated total economic saving on health, employment, and social costs of $25billion a year (including a $1.5 billion saving for the UK NHS). These analyses combined with a variety of systematic reviews of cost-effective measures and national experience relating to both food and physical activity allowed the Eastern Mediterranean Region of the WHO to set out priority actions for combatting obesity in the region (see Table 4) where obesity and diabetes rates are among the highest in the world (WHO (EMRO) 2017). Regulatory backed progressive food reformulation will be a higher priority in Western societies where a greater proportion of food is already sold as food products or prepared meals.

Table 4 Policy strategies for obesity prevention based on numerous systematic analyses of cost-effectiveness and national experience

1. Reformulation:
National progressive mandatory reductions in fat sugar and salt every 3 years
Apply to total fat, saturated fat, free sugars, and salt
Audit, publicize
Include street traders and fast food outlets
Include reduced portion sizing
2. Fiscal measures:
That is, taxes and subsidies of food (but also relate to socioeconomic policies to reduce inequality). Taxes best used as a commodity tax on fat, sugar, sugary drink and not a product-based VAT measure
3. Public procurement:
Introduce mandatory nutrition standards in all publicly funded institutions (and progressively involving private providers with nutrition standards for types of food served)
Aim for progressive reductions in dietary fat to 25%, free sugar to 5 g%, and salt to 2.0 g/1000 kcal
Provide training to catering companies on appropriate catering methods in public institutions to reduce the use of frying and sweetening of foods and help/training with menu redesign
4. Physical activity interventions with wide variety of policies throughout life with (a) media, (b) multiple school actions, worksite, transport, civic recreation opportunities, and urban redesign; transport changes crucial
5. Food supply and trade
Establish mandatory national food standards thereby overcoming free world trade regulations by affecting local production as well as imports

(*continued*)

Table 4 (continued)

Take Finnish canteen experience of "free" salad bar/vegetables; city planning, e.g. controlling public adverts, density/location of fast food outlets
Sign up to/implement Milan urban food pact and sustainable food plan
6. Marketing
Children <18 years focus for 1st phase only with application of WHO-agreed ban on marketing. Then:
Apply restrictions to all marketing of high fat, sugary, salty (HFSS) foods to population by all means
Abolish food promotions of HFSS foods in its many forms by legal means
Establish a national and then regional legal process with potential global agreement on liability of food companies for their advertising effects in other countries – thereby setting internet/cable TV standards
7. Labeling with traffic light labeling shown to be most effective. Need standard display as a mandatory requirement on all packaged foods/menu displays; consider related supermarket layouts
8. Breast feeding. Many national practices very poor so implement:
Mandatory baby-friendly hospitals and clinic facilities
Implement WHO bans on breast milk substitutes anywhere associated with pregnancy
Provide and promote facilities for breastfeeding at work/in public/mandatory maternal leave for 6 months
9. Mass media campaigns: Their main purpose is to build support for the other policies and actions; a few of the more receptive public will change their living patterns as a result
10. Health sector:
Prepregnancy counselling and management crucial
Community-based/GP screening for high-risk groups with early interventions
Integrated focus on dietary improvements, tobacco use cessation, exercise, and their life-long benefits

Policies adapted for general use in countries with very developed industrialized food system from the WHO EMRO analyses of policy needs for obesity and diabetes prevention (2017) (WHO (EMRO) 2017)

Unless there is a coherent approach to government-led regulatory measures then most experts find it difficult to foresee any reduction in the epidemic of obesity because of the modest improvements attained in some countries in the children's prevalences of obesity. Small changes can be induced by tackling individuals within the community at risk of diabetes and then instituting substantial changes by specific advice and monitoring over a prolonged time with a 5% reduction in weight and falls in fat intake to 25%, with increases in fruit and vegetable and fiber intake together, of course, with little or no sugar added to the diet, and with some increase in physical activity. If this is coordinated on a state or national bases then the distribution of BMIs within a community can be changed a little as well as helping to prevent diabetes, but the impact on obesity as such is very modest and few countries are yet able to undertake the major interventions on a national individual basis that Finland is engaged in (Salopuro et al. 2011).

References

Abdullah A, Amin FA, Hanum F, Stoelwinder J, Tanamas S, Wolf R, Wong E, Peeters A. Estimating the risk of type-2 diabetes using obese-years in a contemporary population of the Framingham study. Glob Health Action. 2016;9(1):30421.

Afshin A, Micha R, Khatibzadeh S, Fahimi S, Shi P, Powles J, Singh G, Yakoob MY, Abdollahi M, Al-Hooti S, Farzadfar F, Houshiar-Rad A, Hwalla N, Koksal E, Musaiger A, Pekcan G, Sibai AM, Zaghloul S, Danaei G, Ezzati M, Mozaffarian D, 2010 Global Burden of Diseases, Injuries, Risk Factors Study: NUTRItrition, ChrOnic Diseases Expert Group (NUTRICODE), Metabolic Risk Factors of ChrOnic Diseases Collaborating Group. The impact of dietary habits and metabolic risk factors on cardiovascular and diabetes mortality in countries of the Middle East and North Africa in 2010: a comparative risk assessment analysis. BMJ Open. 2015;5(5):e006385.

Allman-Farinelli MA, Chey T, Bauman AE, Gill T, James WP. Age, period and birth cohort effects on prevalence of overweight and obesity in Australian adults from 1990 to 2000. Eur J Clin Nutr. 2008;62(7):898–907.

Arterburn DE, Maciejewski ML, Tsevat J. Impact of morbid obesity on medical expenditures in adults. Int J Obes. 2005;29(3):334–9.

Ashwell M, Gibson S. Waist-to-height ratio as an indicator of 'early health risk': simpler and more predictive than using a 'matrix' based on BMI and waist circumference. BMJ Open. 2016;6(3): e010159.

Atiase Y, Farni K, Plange-Rhule J, Luke A, Bovet P, Forrester TG, Lambert V, Levitt NS, Kliethermes S, Cao G, Durazo-Arvizu RA, Cooper RS, Dugas LR. A comparison of indices of glucose metabolism in five black populations: data from modeling the epidemiologic transition study (METS). BMC Public Health. 2015;15:895.

Baker JL, Olsen LW, Sørensen TI. Childhood body-mass index and the risk of coronary heart disease in adulthood. N Engl J Med. 2007;357(23):2329–37.

Black D. Obesity – a report of the royal college of physicians. J R Coll Physicians Lond. 1983;17(1):1–58.

Bray GA (Ed) Obesity in perspective. In: Proceedings of the 1973 John E Fogarty international conference Centre on obesity, vol 2. Washington, DC: DHEW Publication; 1976.

Bray GA. Historical framework for the development of the ideas about obesity. In: Bray GA, Bouchard C, WPT J, editors. Handbook of obesity. New York: Marcel Dekker, Inc; 1997. p. 1–29.

Chaudhari LS, Begay RC, Schulz LO. Fifteen years of change in the food environment in a rural Mexican community: the Maycoba project. Rural Remote Health. 2013;13:2404.

Chen CM. Overview of obesity in mainland China. Obes Rev. 2008;9(Suppl1):14–21.

Christakis G. The prevalence of adult obesity. In: Bray GA, Bouchard C, WPT J, editors. Handbook of obesity. New York: Marcel Dekker, Inc; 1975. p. 209–13.

Cohen DA, Collins R, Hunter G, Ghosh-Dastidar B, Dubowitz T. Store impulse marketing strategies and body mass index. Am J Public Health. 2015;105(7):1446–52.

Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. Pediatr Obes. 2012;7(4):284–94.

Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ. 2000;320:1240–3.

D'Angelo S, Yajnik CS, Kumaran K, Joglekar C, Lubree H, Crozier SR, Godfrey KM, Robinson SM, Fall CH, Inskip HM, SWS Study Group and the PMNS Study Group. Body size and body composition: a comparison of children in India and the UK through infancy and early childhood. J Epidemiol Community Health. 2015;69(12):1147–53.

Dallosso HM, James WP. The role of smoking in the regulation of energy balance. Int J Obes. 1984;8(4):365–75.

Dee A, Kearns K, O'Neill C, Sharp L, Staines A, O'Dwyer V, Fitzgerald S, Perry IJ. The direct and indirect costs of both overweight and obesity: a systematic review. BMC Res Notes. 2014;7:242.

Deurenberg P, Deurenberg-Yap M, Guricci S. Asians are different from Caucasians and from each other in their body mass index/body fat per cent relationship. Obes Rev. 2002;3(3):141–6.
Deurenberg P, Deurenberg-Yap M, Foo LL, Schmidt G, Wang J. Differences in body composition between Singapore Chinese, Beijing Chinese and Dutch children. Eur J Clin Nutr. 2003;57:405–9.
Dobbs R, Sawers C, Thompson F, Manyika J, Woetzel J, Child P, McKenna S, Spatharou A. Overcoming obesity: an initial economic assessment. A discussion paper by the McKinsey Global Institute Nov 2014. See: http://www.mckinsey.com/industries/healthcare-systems-and-services/our-insights/how-the-world-could-better-fight-obesity
Ferro-Luzzi A, Sette S, Franklin M, James WP. A simplified approach of assessing adult chronic energy deficiency. Eur J Clin Nutr. 1992;46(3):173–86.
Finucane MM, Stevens GA, Cowan MJ, Danaei G, Lin JK, Paciorek CJ, Singh GM, Gutierrez HR, Lu Y, Bahalim AN, Farzadfar F, Riley LM, Ezzati M. Global burden of metabolic risk factors of chronic diseases collaborating group (Body mass index). National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9·1 million participants. Lancet. 2011;377(9765):557–67.
Foresight. Foresight, Tackling Obesities: Future Choices. London: Government Office of Science., See: www.foresight.gov.uk; 2007.
Franklin MF. Comparison of weight and height relations in boys from 4 countries. Am J Clin Nutr. 1999;70(S1):157S–62S.
GBD 2015 DALYs and HALE Collaborators. Global, regional, and national disability-adjusted life-years (DALYs) for 315 diseases and injuries and healthy life expectancy (HALE), 1990–2015: a systematic analysis for the global burden of disease study 2015. Lancet. 2016;388:1603–58.
Han TS, van Leer EM, Seidell JC, Lean ME. Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. BMJ. 1995;311(7017):1401–5.
Handley JD, Williams DM, Caplin S, Stephens JW, Barry J. Changes in cognitive function following bariatric surgery: a systematic review. Obes Surg. 2016;26:2530–7.
He L, Tuomilehto J, Qiao Q, Söderberg S, Daimon M, Chambers J, Pitkäniemi J, DECODA study group. Impact of classical risk factors of type 2 diabetes among Asian Indian, Chinese and Japanese populations. Diabetes Metab. 2015;41(5):401–9.
Hooper L, Abdelhamid A, Bunn D, Brown T, Summerbell CD, Skeaff CM. Effects of total fat intake on body weight (review). Cochrane Libr. 2015;8:CD011834.
House of Lords. Behaviour Change. 2011. Paper HL179. See: http://www.parliament.uk/business/committees/committees-a-z/lords-select/science-and-technology-committee/inquiries/parliament-2010/behaviour
Huxley R, James WP, Barzi F, Patel JV, Lear SA, Suriyawongpaisal P, Janus E, Caterson I, Zimmet P, Prabhakaran D, Reddy S, Woodward M, Obesity in Asia Collaboration. Ethnic comparisons of the cross-sectional relationships between measures of body size with diabetes and hypertension. Obes Rev. 2008;9(Suppl 1):53–61.
Institute of Medicine. National Research Council Weight gain during pregnancy: reexamining the guidelines. In: Rasmussen KM, Yaktine AL, editors. Committee to reexamine the guidelines. Washington, DC: The National Academies Press; 2009. ISBN 0-309-13114-6.
James WPT. Research on obesity. A report of the DHSS/MRC Group. London: H.M.S.O; 1976.
James WPT, Reeds PJ. Nutrient partitioning. In: Bray GA, Bouchard C, James WPT, editors. Handbook on obesity. New York: Marcel Dekker Inc; 1997. p. 555–71.
James WPT, Ferro-Luzzi A, Waterlow JC. Definition of chronic energy deficiency in adults. Report of a working party of the International Dietary Energy Consultative Group. Eur J Clin Nutr. 1988;42(12):969–98.
James WPT, Ralph A, Ferro-Luzzi A. Energy needs of the elderly: a new approach. In: Munro HN, Danford DE, editors. Nutrition, ageing and the elderly. New York/London: Plenum Press; 1989. p. 129–51.
Keys A, Fidanza F, Karvonen MJ, Kimuru N, Taylor HL. Indices of relative weight and obesity. J Chronic Dis. 1972;25:329–43. and International Journal of Epidemiology, 2014, 655–66

Kullmann S, Heni M, Hallschmid M, Fritsche A, Preissl H, Häring HU. Brain insulin resistance at the crossroads of metabolic and cognitive disorders in humans. Physiol Rev. 2016;96(4):1169–209.
Lobstein T, Jackson-Leach R, Moodie ML, Hall KD, Gortmaker SL, Swinburn BA, James WP, Wang Y, McPherson K. Child and adolescent obesity: part of a bigger picture. Lancet. 2015;385(9986):2510–20.
Marmot M, Society F. Healthy lives. Strategic review of health inequalities in England post 2010. London: The Marmot Review; 2010.
McKeigue PM, Shah B, Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in south Asians. Lancet. 1991;337:382–6.
Moodie R, Stuckler D, Monteiro C, Sheron N, Neal B, Thamarangsi T, Lincoln P, Casswell S, Lancet NCD Action Group. Profits and pandemics: prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries. Lancet. 2013;381 (9867):670–9.
National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. Washington, DC: US Department of Health and Human Services; 1998.
Norton K, Dollman J, Martin M, Harten N. Descriptive epidemiology of childhood overweight and obesity in Australia: 1901–2003. Int J Pediatr Obes. 2006;1(4):232–8.
Padwal RS, Pajewski NM, Allison DB, Sharma AM. Using the Edmonton obesity staging system to predict mortality in a population-representative cohort of people with overweight and obesity. CMAJ. 2011;183(14):E1059–66. https://doi.org/10.1503/cmaj.110387. Epub 2011 Aug 15. PubMed PMID: 21844111.
Prospective Studies Collaboration. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. Lancet. 2009;373:1083–96.
Public Health England. Sugar reduction. The evidence for action. 2015. see: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/470179/Sugar_reduction_The_evidence_for_action.pdf
Reither EN, Hauser RM, Yang Y. Do birth cohorts matter? Age-period-cohort analyses of the obesity epidemic in the United States. Soc Sci Med. 2009;69(10):1439–48.
SACN (Scientific Advisory Committee on Nutrition). Carbohydrates and health. 2015. HMSO. See: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/445503/SACN_Carbohydrates_and_Health.pdf
Sallis JF, Bull F, Burdett R, Frank LD, Griffiths P, Giles-Corti B, Stevenson M. Use of science to guide city planning policy and practice: how to achieve healthy and sustainable future cities. Lancet. 2016;388(10062):2936–47.
Salopuro TM, Saaristo T, Oksa H, Puolijoki H, Vanhala M, Ebeling T, Niskanen L, Tuomilehto J, Uusitupa M, Peltonen M. Population-level effects of the national diabetes prevention programme (FIN-D2D) on the body weight, the waist circumference, and the prevalence of obesity. BMC Public Health. 2011;11:350.
Sanchez-Castillo CP, Velasquez-Monroy O, Lara-Esqueda A, Berber A, Sepulveda J, Tapia-Conyer R, James WP. Diabetes and hypertension increases in a society with abdominal obesity: results of the Mexican National Health Survey 2000. Public Health Nutr. 2005;8:53–60.
Sassi F. Obesity and the economics of prevention. Fit not fat. Organisation for economic co-operation and Development. 2010. See http://www.oecd.org/els/health-systems/obesity-and-the-economics-of-prevention-9789264084865-en.htm
Schulz LO, Chaudhari LS. High-risk populations: the Pimas of Arizona and Mexico. Curr Obes Rep. 2015;4(1):92–8.
Shai I, Jiang R, Manson JE, Stampfer MJ, Willett WC, Colditz GA, Hu FB. Ethnicity, obesity, and risk of type 2 diabetes in women: a 20-year follow-up study. Diabetes Care. 2006;29:1585–90.
Sharma AM, Kushner RF. A proposed clinical staging system for obesity. Int J Obes (Lond). 2009;33(3):289–95. https://doi.org/10.1038/ijo.2009.2. PMID:19188927.
Stevens GA, Singh GM, Lu Y, Danaei G, Lin JK, Finucane MM, Bahalim AN, RK MI, Gutierrez HR, Cowan M, Paciorek CJ, Farzadfar F, Riley L, Ezzati M. Global burden of metabolic risk factors of chronic diseases collaborating group (Body mass index). National,

regional, and global trends in adult overweight and obesity prevalences. Popul Health Metrics. 2012;10(1):22.
Tagliabue C, Principi N, Giavoli C, Esposito S. Obesity: impact of infections and response to vaccines. Eur J Clin Microbiol Infect Dis. 2016;35(3):325–31.
WHO. Physical status: the use and interpretation of anthropometry. Report of a WHO expert committee, World Health Organisation technical report series. Geneva: World Health Organization; 1995.
WHO. WHO child growth standards: methods and development. Geneva: World Health Organization; 2006.
WHO. Guideline on sugars intake for adults and children. Geneva: WHO; 2015. see: http://www.who.int/nutrition/publications/guidelines/sugars_intake/en/
WHO (EMRO). Proposed policy priorities for preventing obesity and diabetes in the Eastern Mediterranean Region. Report for the WHO Regional Office for the Eastern Mediterranean. 2017 (in press).
WHO expert consultation (held in Singapore). Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004;363:157–63.
WHO, Multicentre Growth Reference Study Group. Assessment of differences in linear growth among populations in the WHO Multicentre Growth Reference Study. Acta Paediatr Suppl. 2006;450:56–65.
Wiggins S, Keats S, Han E, Shimokawa S, Alberto J, Hernández V, Clara RM. The rising cost of a healthy diet. Changing relative prices of foods in high-income and emerging economies. Overseas Development Institute. 2015. See: https://www.odi.org/publications/8877-rising-cost-healthy-diet-changing-relative-prices-foods-high-income-emergingeconomies#downloads.
Withrow D, Alter DA. The economic burden of obesity worldwide: a systematic review of the direct costs of obesity. Obes Rev. 2011;12(2):131–41.
World Bank. Poverty and health. Briefing paper. 25 Aug 2014. See: http://www.worldbank.org/en/topic/health/brief/poverty-health
World Cancer Research Fund/American Institute of Cancer Research. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Washington, DC: AICR; 2007.
World Cancer Research Fund/American Society for Cancer Research. Policy and action for cancer prevention. Food, nutrition and physical activity: a global perspective. Washington, DC: AICR; 2009.
World Health Organisation. The world health report. Reducing risks, promoting healthy life. Geneva: World Health Organisation; 2002.
World Health Organisation. Global status report on noncommunicable diseases. Geneva: World Health Organisation; 2014.
World Health Organization. Diet, nutrition and the prevention of chronic diseases. Report of a joint WHO/FAO expert consultation, WHO technical report series, vol. 916. Geneva: World Health Organization; 2003.
World Health Organization (WHO). Obesity: preventing and managing the global epidemic. Report of a WHO consultation: WHO technical report series, vol. 894. Geneva: World Health Organization; 2000.
Yako YY, Guewo-Fokeng M, Balti EV, Bouatia-Naji N, Matsha TE, Sobngwi E, Erasmus RT, Echouffo-Tcheugui JB, Kengne AP. Genetic risk of type 2 diabetes in populations of the African continent: a systematic review and meta-analyses. Diabetes Res Clin Pract. 2016;114:136–50.
Yoo EG. Waist-to-height ratio as a screening tool for obesity and cardiometabolic risk. Korean J Pediatr. 2016;59(11):425–31.
Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL, Lisheng L, Tanomsup S, Wangai P Jr, Razak F, Sharma AM, Anand SS, INTERHEART Study Investigators. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. Lancet. 2005;366(9497):1640–9.