Challenges in obesity epidemiology

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Purpose and definitions

The purpose of this review is to summarize current knowledge about the epidemiology of obesity, and to identify the major gaps that a UK partnership of academia, industry and government might address.

Obesity is an excess of body fat leading to ill health. To assess fatness, the body mass index (BMI, kg m$^{-2}$), originating from Quetelet’s ‘average man’ (1), is widely used in clinical settings and population studies. BMI is an imprecise but useful measure of adiposity (2–4). For a given BMI, adiposity varies with age, sex and ethnicity (5); however, BMI correlates reasonably well with body fat mass (2,5,6) and the risks of obesity-related diseases (6–8). Adults with a BMI $>30$ are classified as obese, whereas those with BMI 25–30 are classified as overweight (2). In children, defining overweight and obesity is complicated by the fact that weight varies with height as children grow. But methods to standardize childhood BMI for age and gender are available and give results that have been validated against other measures of adiposity (9). Specious differences in prevalence can occur because of the adoption of different cut-off levels of child BMI standardized for age and gender (10,11). The use of an international standard is a way to avoid this problem and enable comparisons of obesity prevalence between countries (12). Optimal cut-off levels for measuring change over time within countries will, however, vary across the world.

Descriptive epidemiology

Prevalence and secular trends in men and women

Based on a sample representative of England in 2004, 22.7% of men and 23.2% of women were obese and 43.9% of men and 33.9% of women were overweight (13), suggesting that more than half of English adults were either overweight or obese. In 2003, over half of Scottish adults were either overweight or obese (64% of men and 57.3% of women) (14). There was little change in overweight prevalence in England between 1993 and 2004, while obesity prevalence rose by 9.5% in men and 6.8% in women, at the same time the proportion considered lean (BMI 20–25) decreased by 10.6% in men and 8.5% in women (13). Similar trends were observed in Scotland (14). These repeated surveys suggest that the BMI of the UK population shifted to the right. Although the prevalence of obesity is usually higher in women, the prevalence in both sexes in England has been comparable since 2000. In addition, a threefold difference in prevalence of obesity has been observed at age 45 as compared with 16–24 years (13). This difference between age groups has been consistent over time, while the mean BMI and the prevalence of obesity for each age group has been increasing. The rapid rise in adiposity in the UK reflects a global phenomenon – affecting both high- and low-income countries (2,15).
While English obesity prevalence rose three- to fourfold across the two decades from 1980 (16), it doubled in the USA over a similar period, with the USA starting from a higher baseline (17). From studies of US veterans, it has been proposed that the annual growth rate of median BMI was about 0.3% between 1900 and 1976 but almost doubled to 0.5% per year between 1988 and 2000 (18). Recently (1999–2004), obesity prevalence in US women seems to have stopped rising (19).

Prevalence and secular trends in children

The global epidemic of obesity has affected English children substantially. Using International Obesity Taskforce definitions for overweight and obesity (12), among 5–10-year-olds between 1974 and 2002/03 the prevalence of overweight rose from 11.3% to 22.6% in boys and 9.6% to 23.7% in girls, and obesity rose from 1.8% to 6.0% in boys and 1.3% to 6.6% in girls (20). There was a two- to fivefold increase in the prevalence of overweight and obesity in English children over the three decades up to the early 2000s, with the most rapid rise occurring in the 1990s (21,22). In Scotland from 1974 to 1994, the proportion of overweight 4-11-year-olds increased from 5.4% to 10.0% in boys and 8.8% to 15.8% in girls, and the proportion obese increased from 1.7% to 2.1% in boys and 1.9% to 3.2% in girls (23,24).

In the European Union (EU), there are around 22 million overweight children (5 million of these are obese). Assuming a consistent rise in prevalence of overweight and obesity, it is projected, that by 2010, 26 million will be overweight (rising by about 1.3 million per year) and, of these children, 6.4 million will be obese (rising by 350 000 per year) (25).

Rising BMI has been reported in children as young as 3 years old. In England between 1988 and 1998, the proportion 3-year-olds above the 85th centile of the 1990 British Growth Reference (26) rose from 14.7% to 23.6% and those above the 95th centile rose from 5.4% to 9.2% (22). Rising BMI continued in Scottish pre-school children between 1995 and 2001 (23).

Less is known about the trends in adolescents: the sampling has varied too much over time to make valid comparisons.

Three different definitions of childhood overweight and obesity have been used in contemporary studies of UK children — the prevalence figures cannot be compared directly.

Geographical variation

Geographical variation in the prevalence of obesity among middle-aged men has been observed, with the difference in the prevalence of those with higher BMI by up to twofold between different towns (27). London and Yorkshire had slightly higher obesity prevalence in children compared with national average in England in 2001 and 2002, but there was no clear gradient across different regions in the country (28). Although regional differences in mean BMI in England have been observed, there was no clear north–south divide (29). A recent UK study has suggested a temporal/spatial/spread pattern to rising childhood adiposity (30). In developing countries, obesity is observed more in urban than in rural areas (31,32).

Socioeconomic status and ethnicity

In England in 1996, obesity was commoner among those with less education, and in women of a lower occupational status (33). Birth cohort studies suggest that lower socioeconomic status in childhood is associated with higher BMI in adulthood (34,35). An increase over time (1995–2002) in mean BMI was observed among children in both manual and non-manual socioeconomic groups but the percentage change was steeper for those in the manual group (36,37). Although obesity varies with socioeconomic status in children, the gap may have narrowed in the 1990s, at least in some areas (30). In developing countries, higher social status is associated with obesity but the burden of obesity shifts towards the lower socioeconomic groups as the country’s income rises, and affects women at the early stages of economic development (15). Education may be ‘protective’ against obesity in high-income countries but is a risk for obesity in low-income countries, depending on the stage of economic development.

The prevalence of obesity also varies with ethnicity. In England in 2004, the 22.7% of the general population was obese, the prevalence was higher in those of Black Caribbean or Irish ethnicity (both 25.2%), and lower in Bangladeshis (5.8%) or Chinese men (6.0%) (37). The proportion of obese women across ethnic groups ranged from 17.2% (Bangladeshi) to 32.1% (Black Caribbean) and 38.3% (Black Africans), whereas the general population prevalence in women was 23.2%. Chinese women had the lowest proportion of obese women (7.6%). However, secular trends for these ethnic groups are less known. In the USA, the prevalence of obesity was generally higher among non-Hispanic Blacks and Mexican-Americans when compared with non-Hispanic Whites in both men and women (38). Between the survey periods of 1976–1980 and 1999–2002, the highest increase in prevalence was observed among Black females (from 31.0% to 49.6%) and among Hispanic females (from 26.6% to 38.9%) when compared with other ethnic and gender groups (39). BMI increased from 1983 to 1999 among English children (5–11 years) of all ethnic groups (40). Among children (2–20 years) in 1999, Afro-Caribbean girls and Indian and Pakistani boys were more likely to be overweight than girls.
or boys in the general population (41). In the USA, the combined prevalence of overweight and obesity among 6–11-year-olds in 1976–1980 was 6.5% and in 1999–2002 was 15.8% (similar prevalence between boys and girls) (38). During this period, a high prevalence was noted in Black females (from 11.2% to 22.8%) and Hispanic males (from 13.3% to 26.5%) as compared with White males (from 6.1% to 14.0%) and White females (from 5.5% to 13.1%). The largest increases in prevalence were among Black males (from 6.8% to 17%) and Black females (from 11.2% to 22.8%) (39).

Factors influencing the obesity epidemic

Adiposity is approximately the net result of the balance between energy intake and energy expenditure. The approximation is weak in children, where excess energy can be used for growth – it is also weak where adiposity is estimated by BMI in populations with increasing physical activity, particularly in men, when a rising BMI can reflect rising lean not fat mass. Generally, however, excess intake or reduced expenditure of energy would result in a positive energy balance, which, in the long term, could cause weight gain. The rapid rise in excess weight in the population is therefore being driven by increased total energy intake, sedentary lifestyle or both. The rapid and widespread nature of the obesity epidemic across geographical, ethnic, age and gender groups suggests that pervasive environmental and/or behavioural changes underlie the epidemic. Multiple genetic factors are likely to explain the variation of the adiposity phenotype in populations (42).

Roles of dietary factors

Evolution has favoured a preference for energy-dense and fatty foods – the body defends against weight loss but tolerates weight gain (43). Preference for energy-dense foods, weak satiety and strong hunger traits leaves humans susceptible to obesogenic environments. Evidence about the roles of dietary factors in obesity is, however, difficult to produce as diet is difficult to measure – the following findings should be interpreted with caution:

In the UK from 1970 to the mid-1980s, total energy intake decreased (44). In the USA during this period, there was a slight increase in the total energy intake in all age groups (45), although another study suggested that there was no change in children’s energy intake (46). The composition of diet might play a more important role than its total energy content in the obesity epidemic. Excess fat intake can lead to excess weight (47,48). Even when the total energy intake is similar or reduced, a higher proportion of the energy provided by fat is associated with weight gain (49–51). Low-sugar, high-fat diets are a greater risk for obesity than high-sugar, low-fat diets (51). In the UK, the proportion of energy consumed as fat has been increasing while that of carbohydrates has been decreasing since the 1970s (44). In the USA, however, the proportion of energy consumed as fat has decreased (17).

Although the contribution of sugar intake to the obesity epidemic is debatable (50), intake of high-fructose corn syrup, the caloric sweetener used in soft drinks in the USA in the 1970s through to the 1990s, has been linked with obesity (52). Fructose as a percentage of energy intake is high in early infancy but its main source is milk products (53), whereas non-alcoholic beverages including soft drinks become a major source of added fructose later on. Increased intake of carbonated drinks in schools has been related to higher percentages of overweight and obese children (54). In the USA, increased consumption of soft drinks and sugared beverages has been observed since the 1970s (55). The consumption of high-fructose corn syrup increased by >1000% over the three decades up to the early 2000s, while cane sugar intake decreased (52). In 1977, sweetened beverages formed only 3.9% of total energy intake – by 1996 this had risen to 9%.

Patterns of food consumption may also contribute to the obesity epidemic. Fast-food consumption has been associated with more energy-dense food, higher-fat intake and more consumption of sugar-containing drinks (56–58). In a survey in the USA, one in four ate fast food on a normal day (56) and those who ate fast food during the survey day had a higher BMI. The frequency of eating fast food has been prospectively associated with increased weight gain (59). Fast-food expenditure increased from 20% in 1970 to 40% in 1995 in the USA – as a percentage of total energy intake, it increased from 2% in 1970 to 10% in 1995 (60). There was a shift between 1977 and 1996 towards food eaten away from the home (45). Considering that food portion sizes increased in the USA between the survey periods 1977–1978 and 1994–1996 particularly for soft drinks (62%), fruit drinks (48%), French fries (57%) and salty snacks (58%) (61), this trend implies patterns of food consumption could play an important role in the obesity epidemic.

In developing countries, obesity has been associated with higher socioeconomic status but the epidemic spread to those in lower socioeconomic groups when high-fat diets become more affordable (32). Nevertheless, despite the globalization of the obesity epidemic, there remains a wide difference in obesity prevalence between developed and developing countries that may be accounted for by differences in dietary habit. For example, the dietary contribution of fast food and soft drinks remains high among children in the USA but relatively low in Russia, China and the Philippines (62).
Role of physical activity

Reduced energy expenditure has been associated with weight gain (63), and several cohort studies have shown lower weight gain with higher levels of physical activity (64). In children, a dose–response relationship between the number of hours of television viewing and the prevalence of overweight has been observed (65). Sedentary lifestyle and increased mechanization could also be culprits in the obesity epidemic. In England, the number of households with no cars reduced from 48% to 26% between 1971 and 2000 (66), whereas the proportion of those with two or more cars increased steadily from 8% to over 29% in 2004–2005 (66,67). Between 1961 and 2003, the total distance travelled by people by car or van increased from 157 billion to 678 billion passenger kilometres, and between 1994 and 2003, this distance travelled increased by 4% per year. By 2004, 76% of men and 68% of women were travelling to work by car or van.

Between the survey periods 1992–1994 and 2003, walking to and from school decreased from 61% to 53% among 5–10-year-olds and from 44% to 41% among 11–16-year-olds (66). During the same period, travelling by car or van increased from 30% to 39% among 5–10-year-olds and from 16% to 23% among 11–16-year-olds. Perhaps the average distance from home to school, which has increased from 1.9 km to 2.3 km, has influenced the use of the car or van for this purpose.

In 2000–2001, adults in England spent over 2 h on watching television and videos/DVDs but only about half an hour was spent on hobbies, games, or sports as daily leisure activities (66). Between the survey periods of 1987, 1990–1991, 1996–1997 and 2002–2003, adult participation in physical activities (sports, games, walking at least 2 miles) showed no clear decreasing trend (13), although the proportion of those who achieved the physical activity target (at least 5 days a week of 30 min or more of moderate to intense activity) between 1997 and 2004 increased from 32% to 37% of men and 21% to 25% of women (13,68). This increasing trend does not hide the fact that the vast majority during this period remained below the recommended level of physical activity (13,68). During the spread of the US obesity epidemic, the proportion of those who reported involvement in leisure-time physical activity regularly (but not intense) reduced from 33.2% in 1991 to 29.6% in 1998 (69). However, the proportion of inactive adults slightly decreased from 29.7% in 1991 to 28.6% in 1998. Large-scale objective measurement of physical activity in the general population is lacking.

There are also limited data on secular trends of physical activity involvement of children. In the USA, the proportion of those physically active in daily physical education classes decreased between 1991 and 1997 (70). Cardio-respiratory fitness among 9–11-year-olds in England between 1997 and 2003 decreased while the prevalence of obesity increased (71). However, it remains unclear how sedentary lifestyle could be an explanation for increasing obesity prevalence in early childhood, particularly in those below 4 years of age (22).

Roles of early life factors

Factors such as parental BMI, socioeconomic status and birth weight have been shown to predict adult obesity (34). However, the findings are inconsistent between studies, many of which do not take important confounding or mediating factors into account. Breastfeeding has been suggested to protect against obesity but findings are inconsistent (72–74). A meta-analysis concluded a small protective effect of breastfeeding, but there was significant evidence of publication bias (75). Although there are few reports on secular trends of breastfeeding, some studies show an increasing proportion of mothers breastfeeding since the 1970s (76–78). It has also been proposed that maternal smoking during pregnancy might be a risk factor (79). But smoking during pregnancy has been decreasing since the 1970s (77,80). There has been no parallel change in the link between obesity prevalence and breastfeeding practice or maternal smoking during pregnancy in recent years.

Trends in obesity-related diseases

The obesity epidemic is an actual and potential public health crisis. Higher BMI has been associated with premature mortality. In one study, male and female non-smokers who were obese at age 40 died 6–7 years earlier than their non-obese counterparts (81). Obesity is associated with hypertension, dyslipidaemia and insulin resistance (6) – the secular trends in these conditions should indicate the emerging disease impact of the obesity epidemic.

Obesity is an important risk factor for type 2 diabetes. The incidence of type 2 diabetes has been rising since the 1970s (82), and more recently a small but increasing proportion of the diagnoses have been in children (83–85). In contrast, death rates have been decreasing during the past three decades in the UK, with more people surviving to older ages (86). Further, coronary heart disease has been declining since the 1970s in many Western countries (87,88). In the UK, age-standardized death rates for circulatory diseases decreased by 37.8% in men and 34.5% in women between 1995 and 2005 (86). The picture of cardiovascular disease risk factors has also been improving. In the USA, mean cholesterol and the proportion of those with high cholesterol levels declined in the 1980s and 1990s (88). Between 1993 and 2003, mean systolic blood pressure reduced from 137 to 132 mmHg among English adults. There has been a substantial reduction in smoking (13),
which could have a stronger effect on mortality and circulatory disease than obesity (87). This might partly explain the complex picture of rising obesity and falling coronary heart disease – but the full explanation is missing; generally, coronary heart disease is the main cause of excess mortality in obesity, but the contribution of coronary heart disease to overall mortality is dropping – this might speciously suggest that obesity is becoming a weaker determinant of disease. Given that BMI is a relatively weak cardiovascular risk factor, most of the trends in cardiovascular diseases are likely to be due to improved diet and lifestyle and improved treatment and control of risk factors other than obesity (89). A further unknown in relating BMI to disease risk is the equity of improved risk factor control and treatments between obese and non-obese people. If obesity is assumed to be a stable determinant of coronary heart disease, then further gains of about 8% to 10% reduction in coronary heart disease might be achieved if mean BMI was reduced (87,89,90). It remains unclear whether or not increasing obesity prevalence will cancel out further reductions in coronary heart disease incidence (or reverse the decreasing trend). Despite the reduction in mean cholesterol in the 1980s and 1990s in the USA, smaller reductions were observed during the late 1990s even with an increase in the use of cholesterol-lowering drugs during this period (88). The obesity epidemic may have halted the downward trend in mean cholesterol.

Research weaknesses

The high-level weaknesses in the current epidemiology of obesity are first the fragmentation of the understanding of energy balance in populations. There is also a shortage of contemporary evidence about the emergence of obesity-related diseases. In addition, a sparse understanding of the early life determinants of metabolic health and the lack of studies of public health interventions to influence energy balance are areas of significant weakness which have been identified elsewhere in this review. More specific problems are:

Inaccurate and incomplete assessment of energy balance

Capturing dietary habit is difficult and under-reporting of dietary intake is seen more in obese people (91,92). Incomparable methods for assessing diet are used across different studies. Diet evolves more quickly than the instruments used to assess it, such that changes over time in nutritional composition might not be captured in long-running studies. Similar problems are associated with assessment of physical activity. Reporting biases, measurement errors and a lack of validated methods beset physical activity epidemiology (64,93). The determinants of energy balance can vary with age, gender and ethnicity, but so can the sensitivities of the instruments used to assess the determinants – these biases need to be better understood.

The determinants of energy balance interact, but the interactions are seldom reported. This is unsurprising because the interactions are highly complex, requiring larger than usual studies to characterize them. For example, unhealthy diets among adolescents correlate both with television viewing and adverse perception of healthy eating (94). Urbanization in developing countries is associated with higher proportions of obesity (32) and could relate to access to cheap energy-dense foods as well as reduced physical activity. The built environment has been associated with excess weight or body size, probably via influences on both diet and physical activity in the community (95). Fast-food outlets are sometimes clustered in deprived areas (96). The interrelationships of the major determinants of obesity over long periods of time need to be understood as a whole, dynamic system. Larger and more wide-ranging studies are required to reveal the ‘big picture’ of the obesity epidemic.

The need for a ‘big picture’ applies not only to current populations but also across the life course. For example, the combined effect of increasing breastfeeding, decreasing energy and fat intake, and increasing physical activity (68) might have cumulative and multiplicative effects only revealed by a set of longitudinal studies. The search for individual modifiable risk factors might be less fruitful than the search for combinations of factors.

Unclear consequences of long-term excess weight

Obese people are more likely to suffer ill health than those who are lean. The studies that established this, however, involved adult men and women who were children and young adults when obesity prevalence was lower than it is now. As such, they were likely to have had a lower adiposity in early life than today’s young. Given that adiposity tracks from childhood into adulthood (that is, the relative position of a child in the BMI distribution would be maintained later into adulthood) (97,98), a greater proportion of the population now has excess weight for a longer period of time. The health impact of long-term excess fat is poorly understood. One study associated early death with obesity at 18 years of age, but noted that the association was only partly explained by baseline BMI (99). Relevant longitudinal prospective studies are limited.

Obesity-related burden of disease might be underestimated

There is conflicting evidence about the nature and strength of the association between BMI and mortality, as well as cardiovascular disease event rates (100). In a meta-
analysis involving 388 622 individuals and 60 374 deaths, obesity was associated with higher mortality but the pooled risk for the overweight group was not significant (100). In patients with coronary heart disease, lower BMI was even associated with higher mortality (101). Considering that the amount of fat may differ between ethnic groups even with the same BMI (102), it is plausible that the underlying differences in fatness might affect how BMI predicts risks in different populations. One study suggested that fat distribution measures predict myocardial infarction more strongly than BMI does (103). In the highest two-fifths of waist:hip ratio, the population attributable risk was 24.3% while in the top two-fifths of BMI, it was 7.7%. The impact of obesity could have been underestimated.

Childhood obesity: a problem poorly understood

Children are experiencing an obesity epidemic at the same time as adults. But it is difficult to imagine how trends in adult energy balance relate closely to those of young children. There is an association between childhood and parental obesity, but it is unclear whether this is an epigenetic phenomenon (metabolism imprinted on offspring) or simply an indication of a family lifestyle that leads to a positive energy balance.

There is also inconsistency in defining obesity in childhood, as the adiposity associated with BMI varies in strength with the degree of adiposity (104). The definition of childhood overweight and obesity is generally based on statistical rather than biological grounds (10). The classification derived from how BMI relates to mortality in adults (105) could be more meaningful biologically, although the generalizability of such classifications across populations has been questioned (106). The relationship of childhood BMI to adult health outcomes is understudied.

Some patterns of early growth have been linked with later adiposity (107). Studies in this area are limited and inconsistent (108), and it is unclear how these patterns explain the increasing obesity prevalence over time. Cohort and period effects of growth patterns on later obesity need to be investigated. It is plausible that cohorts from different periods may have different susceptibilities to the adverse effects of an obesogenic environment.

The obesity epidemic might have different consequences in children than in adults because children are developing within the obesogenic environment. Child growth and obesity interact (109), but the long-term consequences are unknown. Interventions to prevent excessive weight or to reduce weight in children are understudied (31,110,111). Animal studies suggest that there might be lasting, modifiable risk factors in infancy, but these are unstudied as yet in humans (112).

Population-level preventive measures and interventions are understudied

Trials suggest that moderate weight loss is feasible and improves metabolic health (113–115). However, some observational studies suggest that intentional weight loss is associated with higher mortality in spite of improving metabolic risk factors – these could be confounded by unintentional loss of lean mass in the diseased who intend to lose fat mass (116–118). Other studies show reduced mortality with intentional weight loss (119). The epidemiology is not clear but it is biologically plausible that intentional weight loss with reduced metabolic risk factors could improve health.

Individual approaches to reducing weight might be effective in the short term, and in extreme cases, efficient. However, diet and physical activity are interdependent and are both affected by influences beyond the individual. Among children, prevention of excessive weight gain is paramount but they are clearly not in control of the environment that promotes excessive energy intake and/or insufficient energy expenditure.

There is a lack of research into population measures to help people maintain a healthy weight (120). Although population-based intervention studies on reducing the burden of obesity are needed, more research should be carried out into preventing excessive weight gain and maintaining weight loss.

Expecting the general population to adopt healthy lifestyles is unrealistic if the social and built environment does not promote it. Not enough is known about the role of geographical factors and the built environment on obesity, and the impact of food systems and distribution on food choices and dietary habits. The evidence of how to reverse the obesogenic environment through public health action is lacking. Population-based programmes to reduce obesity are part of many health policies but are grossly understudied (121). The randomized controlled trials that attract most research kudos are largely infeasible in this area – alternative designs, such as quasi-experiments are required (31).

Policy analysis is too narrow

For the health policymaker, obesity epidemiology presents many dimensions and uncertainties. Capturing all dimensions, from biological to social, in policy scenarios with a limited evidence base requires extreme co-ordination. Dividing health policy, for example, into separate ‘task-forces’ for obesity and physical activity, as in the UK (122), could lead to narrow analysis that is less informative than a more complete approach. The opportunity costs are very high because more than half of the population in many countries are classified as overweight or obese. Therefore,
obesity policy analysis needs to be a continuous process—linking public health surveillance with cumulative synthesis of the emerging science. Monitoring the performance of obesity policies on population weight/BMI alone is attractively simple but likely to be misleading: the problem is how to improve health when weights are increasing, not just how to change weights.

**Future research**

The high-level tasks for future epidemiology in obesity are first to piece together extant findings and datasets into a more complete ‘meta-epidemiology’ of energy balance. Prospectively, there is a need to characterize the emergence of obesity-related diseases through continuous, longitudinal studies via routine healthcare data. And, at the same time, to establish cohort studies capable of identifying the early life determinants of metabolic health. In addition, the introduction of public health interventions and new technologies to encourage a healthy energy balance need to be subject to (quasi-)experimental study.

Specific areas of research that need nurturing are described in the following sections.

**Improved characterization of the obesogenic environment**

The factors affecting energy balance should be further explored and characterized at an individual level. These factors are interrelated and the nature of their associations may vary with age or with time. Studies are needed to indicate which factors are relevant in specific contexts (age group, ethnicity, etc.). Future studies should also identify factors that prevent excess weight gain and maintain a healthy weight.

Better and better-integrated assessments of diet, feeding behaviour and physical activity need to be developed. Large numbers of individuals then need to be assessed in this way. In particular, the factors that maintain energy balance need to be identified (123). Information and biotechnologies should be harnessed to increase the feasibility and scope of such studies.

**Rapid translation between biology and population studies**

Knowledge about the biological mechanisms of appetite regulation, energy storage and metabolic health is emerging via a myriad studies into different pathways. The challenge for both biology and epidemiology is to assemble the information in ways that explain the effects measured across populations, and shape hypotheses. Bioinformatics is central to this, but must be introduced into mainstream epidemiology.

**Improved measures of adiposity**

Body mass index is inexpensive and useful for crude assessment of population adiposity. However, indices of fat distribution have been shown to predict metabolic disease risks independently of BMI (103,124). Although measuring fat distribution is advocated (2), the use of these indices is uncommon in clinical or public health practice. In addition, the significance of fat distribution in children is poorly understood. Future studies should explore better metrics of adiposity and examine their usefulness in predicting disease risks. Simple but more predictive measures of adiposity phenotype are needed.

**Life course studies of metabolic health**

The obesity epidemic affects all age groups. Heavier babies and children with higher BMI are more likely to become obese in later life. Infants and pre-school children are getting heavier but what drives this is not known. Some studies suggest that increased post-natal growth may predispose children to become obese later in life, but what influences this accelerated growth is not known. More research should be performed to explore the causes and consequences of obesity throughout the life course. Life course studies of metabolic health, starting at the time of antenatal health care, should be established, recruiting parents continuously and following up their offspring both longitudinally and serially across successive birth (sub-)cohorts.

Research should also be performed on the inter-generational effects of obesity, aiming to disentangle hypotheses on metabolic ‘programming’ through genetic or epigenetic mechanisms, and transmission of dietary behaviours and lifestyle from parents to children.

**Vulnerable groups**

There is a need to understand the determinants and consequences of obesity specific to potentially vulnerable sub-populations. These include ethnic and migrant groups, socially or economically deprived groups, and the elderly.

The percentage of fat increases while muscle mass decreases with age (125). The balance between the adverse effects of excess fat on metabolic health and the ‘protective’ effect of weight on bone health needs to be explored in the elderly. The impact of the obesity epidemic among migrant families needs to be investigated (121).

**Public health surveillance and interventional research**

It has been estimated that in 2002, the direct and indirect ‘obesity cost’ in the EU was €32.8 billion, whereas the cost of subsidizing ‘healthy foods’ was €10 billion, a third of
the obesity cost (126). Translating such estimates into health policy would require a much greater body of public health evidence than exists – for example, the presumption that adopting ‘healthy foods’ would translate into reduced obesity levels is not underpinned by direct evidence.

In order to build the evidence base, there is a need to put current public health initiatives to tackle obesity into (quasi-)experimental frameworks routinely. The UK Public Health Service, research-funding organizations and industry could collaborate over this – combining the public health surveillance of obesity (and more specific measures of adiposity) with the registration and study of all relevant population-based interventions.

Conclusion

It is unusual for governments to acknowledge a global public health problem ahead of comprehensive epidemiology about the problem, as is the case with obesity. There are various reasons for this. First, the condition is highly complex and emerging slowly. It is also clear that the average person thinks that they understand the causes of the problem and know ways to control it. And finally, there is an established industry of ineffective and/or inefficient interventions.

Future studies of obesity need to be more precise and complete in determining the effects of adiposity, which are likely to vary with ethnicity, gender, activity level and fat distribution.

Obesity epidemiology needs a large, internationally co-ordinated research effort without delay.

Conflict of Interest Statement

No conflict of interest was declared.

References


