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Adult obesity complications: challenges and clinical impact

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***Abstract*:** The complications associated with adult obesity are overwhelming national healthcare systems. No country has yet implemented a successful population-level strategy to reverse the rising trends of obesity. This article presents epidemiological data on the complications of adult obesity and discusses some of the challenges associated with managing this disease at a population and individual level.

Keywords: complications, epidemiology, morbidity, obesity

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Introduction

Adult obesity [body mass index (BMI) >30 kg/m^2] was estimated to affect 10.8% of men (266 million) and 14.9% of women (375 million) worldwide in 2014. This has more than doubled when compared with worldwide figures in 1975 where 3.2% of men and 6.4% of women were obese. If this trend persists, by 2025, 18% of men and 21% of women will be obese.[1] Since 2006, the rise in adult obesity has remained stable in many developed countries except for morbid obesity (BMI > 40 kg/m^2), which continues to rise[2]; in developing countries obesity prevalence is rising towards levels seen in the Western world.[3] Indeed, the World Health Organisation (WHO) has set governments across the world the challenge of preventing further rises in obesity by 2025 to meet the overarching aim of preventing premature death from the four most common non-communicable diseases – cardiovascular disease (CVD), diabetes, cancer and chronic respiratory disease.[4]

The current review presents epidemiological data pertaining to the complications of adult obesity and some of the challenges associated with managing this disease at a population and individual level.

Obesity, mortality and BMI

Obesity, as defined by BMI (Table 1), is associated with an increased risk of all-cause mortality, with CVD and malignancy being the most common causes of death.[5–8] A meta-analysis of 239 prospective studies involving 10.6 million individuals from Asia, Australia, New Zealand, Europe and North America found that all-cause mortality was lowest between a BMI of 20–25 kg/m^2 but increased significantly just below this range and throughout the overweight/obese categories,[8] which suggests a J-shaped relationship between BMI and mortality. Ethnic differences for BMI ranges defining overweight and obesity exist, especially between Caucasian and Asian populations, reflecting the higher risk of cardiometabolic complications at a lower BMI in the latter population (Table 1).[9] Although BMI is the simplest and most common anthropometric method for diagnosing obesity, waist circumference (WC) or waist-to-hip ratio (WHR) may better predict cardiometabolic disease because they are better measures of abdominal obesity.[10,11] Combining BMI and WC or WHR will capture total body fat distribution better than BMI alone and may help identify individuals with metabolic syndrome (Table 2) at an earlier stage. Given that individuals frequently know their waist size, this may be a more practical measure to self-report compared with height and weight, which can often be misreported.[12]

Mechanisms by which obesity causes complications

The excess adiposity that characterises obesity can cause complications through anatomical and metabolic effects.

Table 1. Adult BMI classification.[13]

Classification	BMI (kg/m²)	
	Caucasian	South Asian* and Chinese
Healthy or 'normal' weight	18.5–24.9	18.5–23
Overweight or pre-obesity	25–29.9	23–27.5
Obesity I	30–34.9	⩾27.5
Obesity II	35–39.9	
Obesity III	⩾40	

*South Asian people are immigrants and descendants from Bangladesh, Bhutan, India, Indian-Caribbean (immigrants of South Asian family origin), Maldives, Nepal, Pakistan and Sri Lanka.[14]
BMI, body mass index.

Table 2. Risk factors used in the clinical diagnosis of the metabolic syndrome.[15]

Measure	Cut-off values
Elevated WC*	Caucasian: >80 cm in females; >94 cm in males.
Reduced HDL cholesterol	<1.3 mmol/l in females; <1 mmol/l in males or on drug therapy to increase HDL
Elevated triglycerdies	>1.7 mmol/l or on drug therapy to reduce triglycerdies
Elevated blood pressure	Systolic ⩾130 and/or diastolic ⩾85 mm Hg or on anti-hypertensive therapy
Elevated fasting plasma glucose	>5.6 mmol/l or on drug therapy for hyperglycaemia

Three risk factors from Table 2 are required for a diagnosis of the metabolic syndrome.
*WC cut-off values for Europeans are included but ethnic-specific values according to the IDF criteria should be used to individualise risk.
HDL, high-density lipoprotein; IDF, International Diabetes Federation; WC, waist circumference.

Anatomical effects

Increased adipose tissue can place strain at various body sites leading to obstructive sleep apnoea (OSA), obesity hypoventilation syndrome (OHS) and osteoarthritis, especially of weight bearing joints.[16–18] Also, increased intra-abdominal pressure is associated with oesophageal disorders such as gastro-oesophageal reflux disease (GORD) and Barrett's oesphagus.[19]

Subcutaneous adipose tissue is a 'metabolic sink' that stores excess calories as triglycerdies through adipocyte hyperplasia and hypertrophy, which protects lean visceral organs such as the heart, kidney, liver and pancreas. However, if subcutaneous adipose tissue capacity is exceeded, hypertrophied adipocytes rupture, triggering inflammation, and triglycerdies are deposited within visceral adipose tissue[20]; indeed obesity is associated with diastolic heart failure, chronic kidney disease (CKD), non-alcoholic fatty liver disease (NAFLD) and type 2 diabetes mellitus (T2DM).[21]

Metabolic effects

Visceral adipose tissue is a potent source of proinflammatory cytokines [tumour necrosis factor alpha (TNF-α), interleukin (IL)-1 and IL-6], which are implicated in cardiometabolic diseases, malignancy and infectious diseases among patients with obesity.[20] Lipid-induced cellular insults (lipotoxicity) due to elevated free fatty acids and lipid intermediates such as ceramides are also implicated in cardiometabolic disorders (e.g. insulin resistance, NAFLD, CVD) that are associated with the metabolic syndrome.[22] Chronic inflammation and endothelial dysfunction are also key mediators linking obesity with CVD.[23]

Type 2 diabetes mellitus

Diabetes mellitus affected 8.5% of the adult European population in 2013, which equates to 56.3 million people.[24] Latest figures suggest that 4.7 million people in the United Kingdom (UK) are affected by diabetes (6% of the UK population), of which 90% have T2DM. UK diabetes prevalence is expected to reach 5 million by 2025.[25] 'Diabesity' describes the concurrent obesity and T2DM epidemic over the past few decades because the risk of T2DM increases with BMI. A recent population study involving 2.8 million UK adults between 2000 and 2018 showed that a BMI of 30–35 kg/m² was associated with a five times increased risk of T2DM, which increased to a 12 times higher risk in those with a BMI of 40–45 km/m².[26] One mechanism linking obesity to T2DM is related to an increase in liver and pancreatic visceral fat,[27] which is better measured by WC or WHR than BMI. Excess hepatic triglycerdies are transported in very low-density lipoproteins to all tissues, including the beta-cells of the pancreas, and over many years this results in progressive pancreatic beta-cell dedifferentiation with a subsequent relatively sudden onset of clinical diabetes.[27] Data from the Counterpoint, Counterbalance and DIRECT

studies have demonstrated that remission of T2DM and improvements in liver and pancreatic fat using magnetic resonance imaging were achieved with a very low-calorie diet (600–853 kcal/day) for 8 weeks to achieve weight loss of 15 kg.[28–30] These studies demonstrate that remission of T2DM depended primarily on weight loss through reductions in liver and pancreatic visceral fat.[27]

Cardiovascular disease

Approximately 17.9 million people die from CVD annually, which accounts for 31% of all deaths worldwide. Ischaemic heart disease and stroke are the two most common causes of mortality worldwide.[31]

Coronary heart disease. A case-control study involving 27,000 participants from 52 countries demonstrated that WHR was the strongest predictor of myocardial infarction (MI), independent of age, gender, ethnicity, smoking status or CVD risk factors (hypertension, diabetes, dyslipidaemia). The relationship between BMI and MI was weaker and less consistent across ethnic groups.[32] The EPIC-Norfolk prospective cohort study involving 24,508 UK men and women followed over 9.1 years also found that WHR was more consistently and strongly predictive of coronary heart disease (CHD) after adjusting for BMI, smoking, hypertension and hypercholesterolaemia.[33] Clearly, CHD is strongly associated with obesity but indices of abdominal obesity are better predictors than BMI.[34] The distribution of fat independently mediates the risk between obesity and CHD and this is likely to be due to ectopic visceral fat promoting chronic inflammation, which participates in all stages of atherosclerosis, including acute thrombosis.[35] Indeed, abdominal obesity is the hallmark of the metabolic syndrome (Table 2) which increases cardiometabolic risk.[15]

Stroke. Obesity is associated with an increased risk of stroke but this relationship is stronger and more consistent for ischaemic stroke. A meta-analysis of 25 studies involving 2,247,961 participants from Western and Eastern countries showed that obese individuals (BMI $>$ 30 kg/m^2) had a 64% increased risk of ischaemic stroke [relative risk (RR) 1.64, 95% confidence interval (CI) 1.36–1.99] and 24% increased risk of haemorrhagic stroke, which was not significant (RR 1.24, 95% CI 0.99–1.54).[36] The association between obesity and ischaemic stroke is mediated by conventional modifiable CVD risk factors and independent mechanisms related to proinflammatory cytokines, reduced levels of adiponectin and a prothrombotic state (hyperfibrinogenaemia, hyperviscosity), which contribute to endothelial cell dysfunction and atherosclerosis.[37,38] The relationship between obesity and haemorrhagic stroke is less consistent.[39]

Gastrointestinal complications

There are several gastrointestinal and hepatobiliary complications of obesity (Table 3), many of which are common and present sooner than cardiometabolic disorders.[19] Therefore screening for obesity in patients with gastrointestinal and hepatobiliary disease should be common practice for early weight loss intervention.

Non-alcoholic fatty liver disease. NAFLD has an estimated prevalence of 25.2% worldwide and 23.7% in Europe,[40] but the true incidence is difficult to characterise due to different diagnostic criteria between studies. The prevalence of NAFLD has increased over the past four decades alongside the increase in obesity.[40] A meta-analysis of 20 studies (12,065 cases, 33,693 controls), 17 from Asian countries and 3 from Western countries, demonstrated that the odds of NAFLD increased by 3–10% per 1 cm increase in WC and 13–38% per 1-unit increase in BMI.[41] Although both BMI and WC were independently associated with NAFLD, markers of abdominal obesity were stronger predictors and remained associated with NAFLD after adjusting for BMI. This may explain why some patients with a normal BMI can develop NAFLD, which is more commonly seen in rural areas of some Asian countries (25–30%) compared with the United States (US) and Europe (10–20%).[41] Therefore, both BMI and WC or WHR should be used to assess NAFLD risk. NAFLD is considered the hepatic manifestation of the metabolic syndrome,[42] whereas longitudinal studies suggest that NAFLD precedes the metabolic syndrome and T2DM.[43] NAFLD increases the risk of T2DM, hypertension, dyslipidaemia and CKD, and it is no surprise that CVD is the leading cause of mortality among this patient group.[10,11] Up to one-third of NAFLD patients are at risk of developing non-alcoholic steatohepatitis (NASH),[44] which can progress to liver cirrhosis, hepatocellular carcinoma (HCC), decompensated liver cirrhosis and death.[45] Therefore, individuals with NAFLD

Table 3. Quantified risk ratios and physiological mechanism of selected gastrointestinal diseases associated with obesity. Taken and adapted from Camilleri et al.[19]

Gastrointestinal disease	Obesity as a risk factor		Physiological mechanism by which obesity is associated with gastrointestinal disease
	Risk expressed as OR or RR	95% CI	
Oesophagus			
GORD	OR, 1.94	1.46–2.57	↑ intra-abdominal pressure, ↓ Oesophageal pressure. ↑ Oestrogen
Erosive oesophagitis	OR, 1.87	1.51–2.31	Abdominal adiposity
Barrett's oesophagus	OR, 4.0	1.4–11.1	Abdominal adiposity, ↓ Adiponectin, ↑Leptin
Oesophageal adenocarcinoma	Men: OR, 2.4 Women: OR, 2.1 RR, 4.8	1.9–3.2 1.4–3.2 3.0–7.7	Abdominal adiposity, ↓ Adiponectin, ↑ Leptin, Insulin-like growth factor –1 and –2
Stomach			
Gastritis	OR, 2.23	1.59–3.11	↓ Adiponectin
Gastric cancer	OR, 1.55 RR (Cardia), 1.8	1.31–1.84 1.3–2.5	Proinflammatory, adipokines, Insulin-like growth factor –1
Hepatobiliary			
NAFLD	RR, 4.6	2.5–11.0	Abdominal obesity, ↑ serum free fatty acids, ↑ hepatic triglycerides, hepatic de novo lipogenesis
Liver cirrhosis	RR, 4.1	1.4–11.4	Non-alcoholic fatty liver disease, non-alcoholic steatohepatitis, proinflammatory
Hepatocellular carcinoma	RR, 1.8	1.6–2.1	Non-alcoholic fatty liver disease, non-alcoholic steatohepatitis, proinflammatory
Gallstone disease (gallstones, cholecystitis)	Men: RR 2.51 Women: RR, 2.32	2.16–2.91 1.17–4.57	Abdominal obesity, ↑ Insulin, ↑ leptin, ↑ lipids, insulin resistance, dysmotility
Gallbladder cancer	RR, 1.3	1.2–1.4	↑ risk of gallstones, chronic inflammation
Pancreas			
Acute pancreatitis	RR, 2.20	1.82–2.66	Hyperlipidaemia, chronic inflammation
Pancreatic cancer	Men: RR, 1.10 Women: RR, 1.13 RR, 1.5	1.04–1.22 1.05–1.18 1.2–1.8	Insulin-like growth factor binding protein 1
Intestinal			
Diarrhoea	OR, 2.7	1.10–6.8	↑ Bile acids, accelerated colonic transit
Diverticular disease	RR, 1.78	1.08–2.94	Chronic inflammation, alteration in gut microbiota
Colonic polyps	OR, 1.44	1.23–1.70	Chronic inflammation
Colorectal cancer	Men: RR, 1.95 Women: RR, 1.15 RR, 1.3	1.59–2.39 1.06–1.24 1.3–1.4	Chronic inflammation, ↑ adipokines, bile acids, insulin resistance, gut microbiota

CI, confidence interval; GORD, gastro-oesophageal reflux disease; NAFLD, non-alcoholic fatty liver disease; OR, odds ratio; RR, relative risk.

require early weight loss intervention to prevent both cardiovascular- and liver-related morbidity and mortality.

Biliary complications. Obesity increases the risk of gallbladder disease. A systematic review of 17 prospective studies involving 1,921,103 participants found a RR of 1.63 for a 5-unit increase in BMI and a RR of 1.46 for a 10 cm increase in WC.[46] There was an almost two-fold increased risk of gallbladder disease from the lower to the upper limit of the normal BMI range (18.5–24.9 kg/m^2), which suggests that even moderate increases in adiposity increase risk.[46] Hormone changes and gallbladder dysmotility are suggested mechanisms to explain the association between obesity and gallbladder disease (Table 3).[19]

Oesphageal complications. Obesity is also associated with oesophageal disorders (Table 3). The prevalence of GORD increases with obesity and meta-analyses report a positive association between BMI and GORD.[47,48] Central obesity is an independent predictor of the consequences of GORD (oesphagitis, Barrett's oesphagus, adenocarcinoma).[49]

Respiratory

Obstructive sleep apnoea. Obesity is the most common risk factor for the development of OSA. Observational data from 2.8 million UK adults found that class I and III obesity were associated with a 5-times and 22-times increased risk of OSA, respectively,[26] which suggests that the risk of OSA increases considerably at a higher BMI. Untreated OSA can cause excessive daytime somnolence, negatively affect work performance, increase the risk of CVD and threaten vehicle licence if driving is affected.[50,51] Proposed mechanisms linking obesity to OSA include adipokines, upper airway adiposity and increased neck circumference causing pharyngeal collapse.[50]

Obesity hypoventilation syndrome. OHS is defined as a combination of obesity (BMI > 30 kg/m^2), daytime hypercapnia (pCO^2 > 6 kpa) and sleep disordered breathing that are not due to other conditions associated with alveolar hypoventilation.[17] OHS has an estimated prevalence of 8.5% in patients with OSA and 19–31% among obese patients.[55,56] The pathophysiology of OHS may be related to leptin resistance causing central hypoventilation, impaired compensatory response to hypercapnia and impaired respiratory mechanics due to obesity.[57] The morbidity and mortality of OHS is greater than OSA. The chronic daytime hypoxia and hypercapnia increase the risk of pulmonary hypertension, right-sided heart failure and cor pulmonale.[17] Weight loss is recommended for both OSA and OHS, but adherence to lifestyle interventions can be difficult for this cohort because their exercise capacity is limited due to daytime somnolence, fatigue and chronic hypoxia, whereas poor sleep is associated with increased appetite.[16] Pharmacological therapy has not been proven to be effective in OSA and OHS.[16] Bariatric surgery is an effective treatment for OSA and parameters of sleep quality,[58] but data on OHS is limited due to the associated pulmonary and cardiac complications and therefore weight loss in this group of patients with chronic cardiorespiratory disease can be challenging.[17] Presently, no randomised control data exist to support bariatric surgery as an intervention to treat OHS.[59]

Asthma. Obesity increases the risk of asthma in children and adults. Over the past 40 years, there have been parallel increases in childhood obesity and asthma, with asthma prevalence doubling between 1980 and 1994.[52] A meta-analysis of seven prospective epidemiological studies involving 333,102 adult participants found that the prevalence of asthma was 38% in overweight individuals and 92% in obese individuals.[53] Two distinct asthma phenotypes have been described in obese patients; the early-onset allergic form and the late-onset non-allergic form,[54] and weight loss has been associated with improvements in lung function and asthma symptoms among obese patients.[16] The mechanism by which obesity increases asthma risk is unclear but may be related to mechanical, inflammatory and hormonal factors.[52]

Cancer

After smoking, obesity is the second biggest preventable cause of cancer in the UK and maintaining a normal weight could prevent 22,800 annual UK cases.[60] In 2001, the International Agency for Research on Cancer concluded that obesity accounted for 10% of post-menopausal breast cancers and 11% of colon cancers. For kidney, lower oesophageal adenocarcinoma and endometrial cancer, the risks attributed to BMI alone were 25%, 37% and 39%, respectively.[61]

A population-based prospective cohort study using data from 5.24 million UK adults concluded that BMI was associated with 17 cancers.[62] Each 5 kg/m^2 increase in BMI was approximately linearly associated with cancer of the uterus, gallbladder, kidney, cervix, thyroid and leukaemia. There was a non-linear but positive association between BMI and liver, colon, ovarian and post-menopausal breast cancer.[62] The authors concluded that the heterogeneity in the effects of BMI on cancer risk suggests that there may be different mechanisms based malignancy type and patient subgroup.[62] Frequently cited mechanisms linking obesity to malignancy include systemic alterations in endogenous hormone metabolism (e.g. insulin, insulin-like growth factor, sex steroids) and chronic inflammation mediated by adipokines.[61]

Obesity also impacts cancer prognosis. A meta-analysis of 82 studies involving 213,075 breast cancer patients showed that obesity (BMI $>$ 30 kg/m^2) was associated with increased cancer-related mortality.[63] Similarly, the Nurses' Health Study, which included 5204 patients with non-metastatic breast cancer, showed that weight gain after diagnosis was associated with increased risk of recurrence and breast-cancer specific mortality.[64] Weight loss by diet and physical activity has been shown to reduce the risk of postmenopausal breast cancer; however, evidence for other cancers is less robust.[65]

Obesity and cognition

Cardiovascular risk factors such as T2DM, dyslipidaemia and hypertension are well-established complications of obesity that increase the risk of dementia and Alzheimer's disease.[21] An independent relationship between mid-life obesity and dementia also exists. A meta-analysis of 39 prospective cohort studies analysing data from 1.3 million adults across the US, Europe and Asia found that a high BMI (overweight or obese range) was associated with an increased risk of dementia when BMI was measured 20 years prior to dementia diagnosis, but this relationship was reversed when BMI was measured closer to dementia diagnosis ($<$10 years).[66] The latter finding could be interpreted as obesity being protective; however, it is likely to be explained by reverse causation and the former finding can be explained by the fact that clinical dementia is preceded by a long (20–30 years) preclinical phase where weight loss is common.[67,68]

Genitourinary

Obesity is an important preventable risk factor for the development CKD because it is associated with major CKD risk factors: diabetes mellitus and hypertension.[69] A large cohort study accruing over 8 million person-years found that a BMI $>$ 25 was an independent predictor for end-stage renal disease. When compared with normal-weight controls (BMI 18.5–24.9 kg/m^2) the RR of end-stage renal disease for overweight individuals was 1.87 (95% CI; 1.64–2.14) and 7.07 (95% CI; 5.37–9.31) for those with class III obesity after adjusting for other CKD risk factors.[69] One proposed independent mechanism linking obesity to CKD is hyperfiltration due to the increased metabolic demands of excess body weight.[70]

Between 1986 and 2000, there was a 10-fold increase in obesity-related glomerulopathy, which is characterised by proteinuria, glomerulomegaly, progressive glomerulosclerosis and renal function decline.[71] Short-term improvement is achieved with renin-angiotensin-aldosterone blockade, whereas weight loss through low-calorie dieting or bariatric surgery is associated with improvements in proteinuria and kidney function.[72] A prospective randomised control trial observed that 3 months of endurance and endurance-strength exercise among obese women (BMI 35 kg/m^2) was associated with an 10 ml/min/1.73 m^2 improvements in estimated glomerular filtration rate.[73]

Obesity can increase the risk of kidney stones,[74] and roux-en-y gastric bypass, an operation used to treat obesity, can also increase the risk of hyperoxaluric kidney stones due to increased enteral oxalate absorption.[75] General and central obesity are both associated with urinary incontinence in men and women, overactive bladder syndrome in women and benign prostatic hyperplasia in men.[76,77]

Musculoskeletal

Obesity is a well-recognised risk factor for the development and progression of osteoarthritis in weight-bearing joints, especially the knee.[18] There is a 36% increased risk of knee osteoarthritis with every 2 unit increase in BMI and patients with obesity suffer more severe joint degeneration.[78] Both obesity and osteoarthritis can reduce mobility, which can increase the risk of weight gain. In patients with osteoarthritis, weight loss of 10% has been associated with an improvement in joint

symptoms, physical function and health related quality of life.[18]

Osteoarthritis. Obesity is also associated with osteoarthritis in non-weight bearing joints such as the hands, which is linked to increased levels of adipokines.[79] Similarly, inflammatory markers observed in obesity are also associated with preclinical rheumatoid arthritis.[80] Prospective cohort data from the Nurses' Health Study accruing more than 4,500,000 person-years of follow up showed that excess body weight (BMI > 24.9 kg/m^2) was associated with a 40–70% increased risk of rheumatoid arthritis in women, with the highest risk observed in overweight or obese women aged 18 years old.[80] Therefore, interventions that combat childhood obesity may reduce the incidence of adult rheumatoid arthritis.

Gout. Obesity has been independently associated with gout. A longitudinal community-based cohort study involving 15,533 men and women demonstrated that the relative risk of gout was almost doubled in those with a BMI > 30 kg/m^2, and that obesity was associated with earlier onset of the disease.[81] Both gout and obesity are associated with elevated levels of serum uric acid and weight loss has been associated with reduced incidence of hyperuricaemia and gout attacks.[82]

Psychosocial

Individuals with obesity are often stigmatised in education, health and employment settings. This results in obesity discrimination,[83] which has increased by 66% over the past decade with prevalence rates comparable with those of race-based discrimination.[84] Discrimination can result in low self-esteem and poor body image, which can negatively impact engagement in physical activity.[85] Obesity is also associated with psychiatric comorbidity. A cross-sectional US epidemiological survey showed that obesity (BMI > 30 kg/m^2) was associated with an approximately 25% increased odds of mood and anxiety disorders.[86] Similarly, another US epidemiological study involving 41,654 respondents in the National Epidemiologic Survey on Alcohol and Related Conditions showed that obesity was associated with an increased odds of alcohol use and mood, anxiety, and personality disorders, with odds ratio ranging from 1.28 to 2.08.[87] Increased BMI is also associated with an increased risk of suicidal ideation in women but not in men.[88,89]

Challenges

Obesity has a complex aetiology that requires a multifaceted strategy for prevention and treatment at a population and individual level.[90] The social ecological model can provide a framework to help identify the personal and environmental determinants of obesity which can facilitate the development of interventions.[91] Indeed, primary and secondary prevention of obesity requires input and collaboration from multiple bodies, such as the government, policy makers, legislative powers and healthcare system. Figure 1 provides an overview of selected interventions, superimposed onto a modified social ecological model, that have been implemented in different countries. No country has yet implemented a successful population-level strategy to reverse the rising trends of obesity.[1]

The environment is obesogenic. Healthful messages from policy makers are often undermined by advertisements that promote large portions of highly palatable energy-dense processed foods and sugar-sweetened beverages,[92] which are key drivers of obesity.[93] The availability of fast-food outlets around schools may be associated with an increased risk of unhealthy eating patterns and childhood obesity, especially in deprived areas.[94,95] This could be curtailed by governments granting local authorities' the power to restrict take-away outlets, especially close to schools. Furthermore, fast-foods are more readily available to both children and adults at any time of day through ordering via mobile phone applications; however, the implications of this on eating behaviour and childhood obesity remain to be elucidated. Policy makers should strongly consider implementing legislation regarding the age at which take-away foods can be purchased, and responsibility must be shared by local providers of fast-foods to enforce this legislation.[96,97] Clearly, labelling the calorie content of takeaway foods may also help consumers opt for more sensible food choices.[93]

Obesity impacts the poorest in society. A UK study of 119,669 individuals aged 37–73 found a strong association between higher BMI and lower socioeconomic status, especially in women.[98] Similarly, a US study reported that overweight women are more likely to work in lower paying-jobs than non-overweight women and all men.[99] This health inequality is further compounded by the fact that fast-food availability is greater in areas of higher deprivation.[100] Taxation of

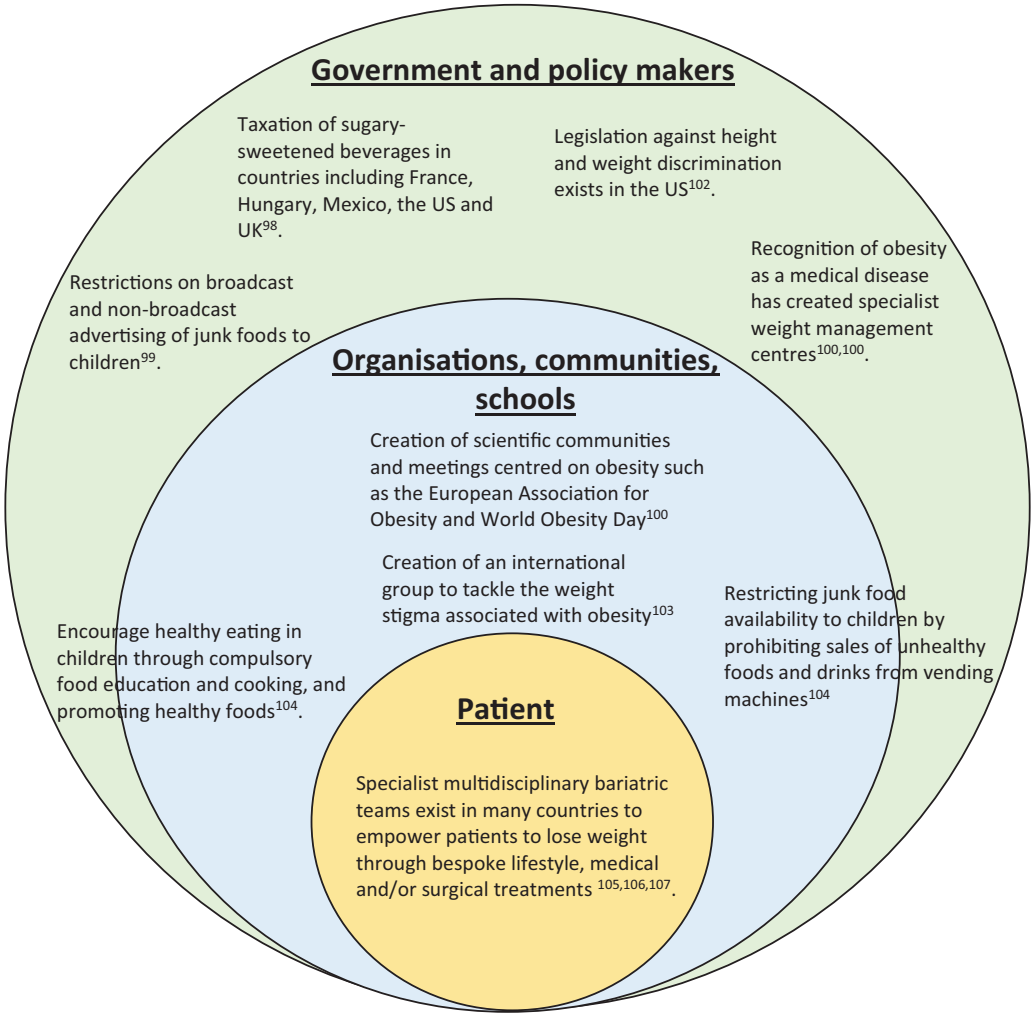

Figure 1. Examples of selected interventions used by different countries to prevent and treat obesity displayed on a modified social ecological model.
UK, United Kingdom; US, United States.

unhealthy foods may be one strategy to limit the availability of fast-foods. Indeed, a tax on sugary-sweetened drinks in Mexico led to an average reduction of 7.6% in purchases of these beverages,[101] whilst a 21% reduction in consumption was observed amongst low-income neighbourhoods in California.[102] In the UK, the soft drinks levy has raised money from taxation to invest into physical activity and healthy eating in UK schools,[103] but whether any of these changes will prevent obesity remains to be seen.

Individuals with obesity face a pervasive form of social stigma due to their weight that subjects them to discrimination in employment, education and healthcare. In the workplace, there is a lack of legislation that protects the vast majority of individuals with obesity who experience discrimination. The UK Equality Act (2010) does not specifically prohibit discrimination against obesity[104] and in 2014, the European Court of Justice ruled that being severely overweight could be considered a disability yet obesity *per se* is not specified as a disabling condition in European Union (EU) employment law.[105] However, some US states have recently introduced legislation that protects against height and weight discrimination,[106] and legislation is a key step to tackling the stigma associated with obesity.

Recognising obesity as a disease rather than a lifestyle choice will address the fallacy that obesity is the fault of the individual due to laziness or gluttony and replace it with scientific knowledge that

body weight is maintained within a relatively narrow individualised range by a precise subconscious homeostatic mechanism.[105,107] Changing this narrative is fundamental so that patients with obesity receive appropriate treatment because there is evidence that patients with obesity are not receiving appropriate referral to specialist services. Worldwide, 0.1–2% of eligible obese patients undergo bariatric or metabolic surgery.[108] In the UK, access to specialist weight management centres is variable in some areas and absent in others. Only 1% of patients who fulfil the National Institute of Clinical Excellence (NICE) eligibility for bariatric surgery are able to access this service in the UK.[109] Greater awareness of the efficacy and cost-effectiveness of surgical interventions for obesity and morbid obesity as well as pathways to access this service should be easily available for local clinicians so that their patients can receive appropriate treatment.[110,111]

In 2012, the US Preventative Services Task Force recommended that all adults be screened for obesity and those with a BMI $> 30\,kg/m^2$ should be offered referral for an intensive multicomponent behavioural intervention.[112] Screening may be one way to increase referral to specialist weight management centres and there is good evidence that treating patients with obesity early in their disease course, especially those with T2DM, can prevent or delay complications.[93]

Patients with obesity can be challenging to manage because the causes and complications of the disease are patient specific and this requires bespoke management at a specialist multidisciplinary weight management centre. Behavioural interventions are fundamental to lifelong weight management, and unique strategies are required for weight loss, maintenance of weight loss and avoiding weight regain, all of which require motivation and commitment from patients.[93] This can be challenging because patients with obesity often have psychological, psychiatric and medical comorbidities that can negatively impact long-term adherence to behavioural interventions.[93] Data from two large randomised control trials of lifestyle interventions, the Diabetes Prevention Programme and the Look AHEAD trial,[113,114] suggest that frequency of patient contact, individualising patient care and face-to-face interventions were important predictors of weight loss. In a separate study, patients who attended group sessions every other week for 1 year after weight loss maintained 13 kg of their initial 13.2 kg weight loss,[115] which suggests that regular group sessions may prevent weight regain. However, implementing behavioural interventions can be difficult due to a lack of resources and time. Remotely delivered behavioural programmes via telephone or the internet are alternative approaches that may be more easily accessible and affordable. Patients who received 20 weight loss intervention phone calls over 6 months lost an average of 4.9 kg; those who received 10 calls lost 3.2 kg and those who were self-directed lost 2.3 kg.[116] In another study, patients who received 24 weekly bespoke weight loss sessions *via* email in addition to internet resources lost 4.4 kg after 1 year when compared with a group receiving internet resources only who lost 2.0 kg.[117] Despite their popularity, little is known about the effectiveness of smart-phone applications for weight management and therefore more research is needed.

Conclusion

Obesity is a multisystem disease that increases the risk of the most common non-communicable chronic diseases of the 21st century.[21,57] The population is developing obesity at a younger age and it is likely that these individuals will suffer morbidity for longer.[2,118] This will be challenging for clinicians because the symptom and disease burden from multi-organ impairment can become irreversible without timely intervention. Early identification of individuals with obesity through simple anthropometric measurements should be a priority for prompt interventions to prevent morbidity and the associated healthcare and economic costs.[119]

Tackling obesity requires a whole systems approach. Governments and policy makers, rather than individuals, have the ability to change the food environment through regulation, taxation and restricting the availability of high-calorie processed foods to adults and children. Patients with obesity who face weight-based discrimination deserve policies and legislation that aim to prevent weight-based inequality. This will help change the current narrative that patients with obesity are to blame for their disease, which fuels a pervasive form of social stigma. Replacing this fallacy with scientific knowledge can prevent discrimination and facilitate referral to specialist weight management centres where a multidisciplinary team can provide bespoke patient care.

Author contribution(s)

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Nadim Haboubi: Conceptualization; Formal analysis; Supervision; Visualization; Writing-review & editing.

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