Obesity and weight management in the elderly

T. S. Han[†], Abdelouahid Tajar[‡] and **M. E. J. Lean[*]**

[†]*Department of Diabetes and Endocrinology, Ashford and St Peter's NHS Trust, Chertsey, Surrey, UK, and [‡]Arthritis Research UK, Epidemiology Unit, Manchester Academic Health Science Centre, The University of Manchester, Manchester, UK*

Our population is ageing, and obesity is increasing in the elderly bringing massive and rapidly changing burdens of ill health related to increased body weights and fat as well as the main drivers of poor diet and inactivity. Overweight and obesity, and a static body mass index (BMI) commonly conceal sarcopenia (gain in body fat but loss of muscle mass and functional capacity) in older people, aggravated by inactivity. A systematic computerized literature search using an iterative manipulation process of the keywords: obesity, elderly, weight loss. The following databases were accessed on 20 October 2010: Medline, Cochrane Collaboration, Ovid and Scholar Google. A large number of clinical consequences of overweight and obesity are particularly problematic for elderly individuals, including type 2 diabetes mellitus, arthritis, urinary incontinence and depression. The observation that the BMI value associated with the lowest relative mortality is slightly higher in older than in younger adults has often been misinterpreted that obesity is not as harmful in the elderly. BMI may be a less appropriate index in the elderly. All the medical consequences of obesity are multi-factorial but all are alleviated by modest, achievable weight loss (5–10 kg) with an evidence-based maintenance strategy. Since sarcopenic obesity is common in the elderly, a combination of exercise and modest calorie restriction appears to be the optimal method of reducing fat mass and preserving muscle mass. Reduction in polypharmacy is a valuable target for weight management. Age is not an obstacle to weight management interventions using moderate calorie restriction and exercise, and the currently licensed drug orlistat appears to have no age-related hazards. Overall balance of clinical outcomes has not been evaluated. In older people the risks from bariatric surgery outweigh benefits. Obesity, and specifically sarcopenic obesity, should also be prevented not only from younger age, but also during major life transitions including retirement, to improve better health outcomes and quality of life in later years, with a focus on those in 'obese families', where the main increases in obesity are located. Randomized controlled trials to determine health benefits and risks from long-term weight management in obese elderly are necessary.

*Correspondence address. Centre for Population and Health Sciences, Human Nutrition, 4th Floor, Walton Building, Royal Infirmary, Glasgow G4 0SF, UK. E-mail: mike. lean@glasgow.ac.uk

Introduction

Health-care services face a number of emerging challenges at a time of economic austerity and uncertainty. In most countries, there has been rather rapid and continuing increase in life expectancy. Based on mortality rates between 1982 and 2007 in the UK, life expectancy has increased by over 6 years for males to 77.2 years and over 4 years for females to 81.5 years and is expected to continue to rise in the near future (Fig. 1)[1] and this has resulted in an 'ageing population'. Over this 25 year period, the numbers of people over the age of 65 have increased from 8.5 million to 9.8 million and people over the age of 85 years rose from 0.6 million to 1.3 million. These figures are projected to reach 16.1 and 3.1 million by the year 2032.[1] In 2005, there were ~18.8 million European citizens aged over 80 years and is estimated to rise to 34.7 million by 2030, when roughly two active people (15–65) will be caring for one inactive older person (over 65)[2] and by then 20% of the US population will be older than 65 years.[3]

This increase in life expectancy has not necessarily been an increase in healthy, work-capable, life which would justify an increase in pension age. It seems that the main increase has been in extra illness-prone old age. The disease burden of increasing numbers of chronically sick elderly people (defined as >65 years old by the WHO[4]) is a major issue. Most diseases increase in elderly people—particularly type 2 diabetes mellitus (DM2), coronary heart disease (CHD), cancers and mobility problems linked to arthritis and depression. Whilst some major risk factors have declined (e.g. smoking), others have increased. The increase in obesity in the past 40 years represents a truly staggering change in human physique and functional capacity, and possibly the greatest driver to change that public health has ever met.

Historically, obesity was a relatively uncommon problem (6–8% of adults in the 1970s) but its prevalence increased with age, as body fat is acquired, to a peak of ~60 years. Thereafter, body weights changed little, and began to decline in older age.[5] Now together with an ageing population, we see an upward shift in the age at which body fat and body mass index (BMI) stop increasing.[6] The prevalence of obesity (defined as BMI $\geq 30 \, \text{kg/m}^2$) in the over 80-year group is about a half of that in the 50–59-year group.[7] This phenomenon is likely to arise
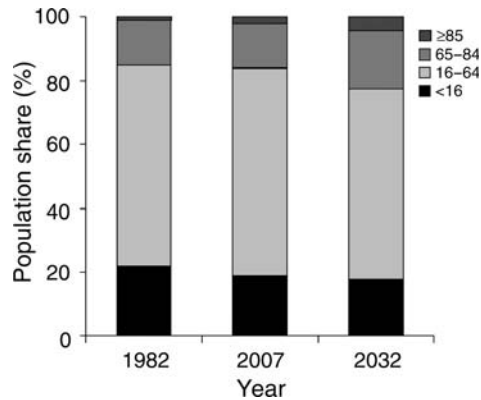
Fig. 1 Population age structure 1982–2032 in the UK (adapted from Dunnell, 2008. *Source*: Office for National Statistics)

from a number of confounding factors such as survival bias, competing mortalities, smoking, weight change and unintentional weight loss in the sick elderly as well as their relatively shortened life expectancy. No change in BMI could be masking a loss in weight if height has fallen, so we are likely overestimating obesity and underestimating undernutrition in this group. However, increasing inactivity and illness in elderly people commonly results in substantial loss of muscle mass while body fat is relatively preserved or increased. A loss of muscle mass may go unnoticed in obese people unless there is clear functional loss of muscle strength. This condition is known as sarcopenic obesity. Current secular trends indicate that the prevalence of obesity and of severe obesity (e.g. BMI $\geq$40 kg/m^2) in the elderly will increase, even if the overall obesity epidemic is declining. The Scottish Health Survey, for example, has most recently shown, in 2008, that while overall prevalence of obesity shows little increase—especially in women, BMI is continuing to rise between age 60 and 70 years.[6] More worryingly, waist circumferences (WC) have shown a 5–10-cm secular increase in the 10 years between 1998 and 2008 in both men and women at all ages between 50 and 70 years (Fig. 2).[8]

There is now good evidence that obesity in the elderly increases cardiometabolic risk, physical disability, impaired quality of life and sexual dysfunction, lower urinary tract symptoms as well as decreased cognitive function and dementia.[9] A number of longitudinal studies have shown that obesity from the middle age years increases the risk of dementia in older ages[10,11] but the association of increased BMI to dementia appears weaker among elderly individuals. Obesity has also been shown to associate with accelerated biological ageing process as indicated by increased shortening of telomere length.[12] These health
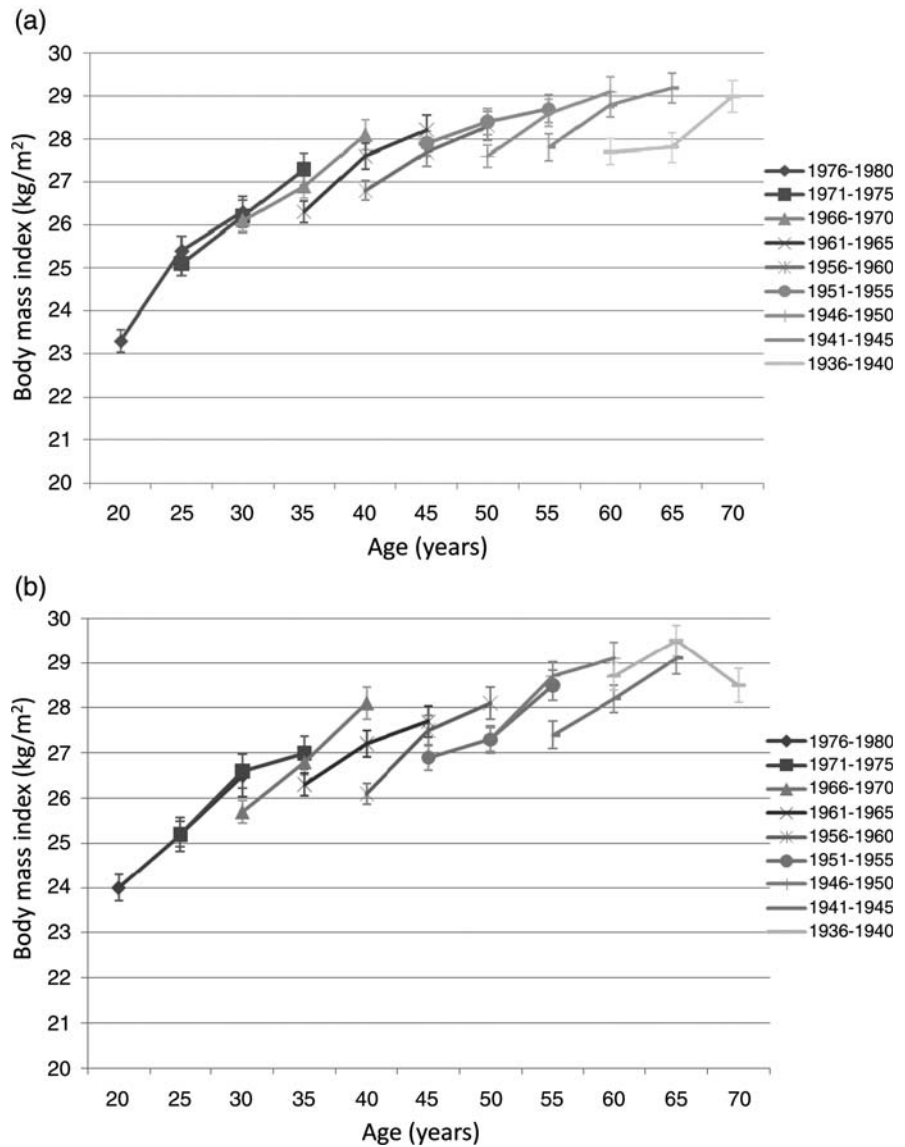
Fig. 2 Data from the cross-sectional Scottish Health Surveys analysed into 'synthetic birth cohorts' of individuals born in the same 5-year bands. Men show rather gradual rises in BMI with age, $\sim$0.5–1.0 kg/m^2 over 10 years up to age 70, and an increase of around 0.3 kg/m^2 between the surveys of 1998 and 2008 (**a**). Women show similar gentle rises in BMI, with little increase in age >65 years (**b**). A pattern comparable to that of BMI is observed for WC in men (**c**) and women (**d**), but subjects from more recent birth cohorts reach a given WC value at a considerably younger age than those who were born earlier. For example those born in 1971 to 1975 reached a mean WC value >80 cm in women, or 94 cm in men, at the age of 35 years, while those who were born 10 or 20 years earlier did not attain the same level until they were 40 or 45 years of age, respectively. C Katsarou and MEJ Lean, unpublished results for ref. [8].
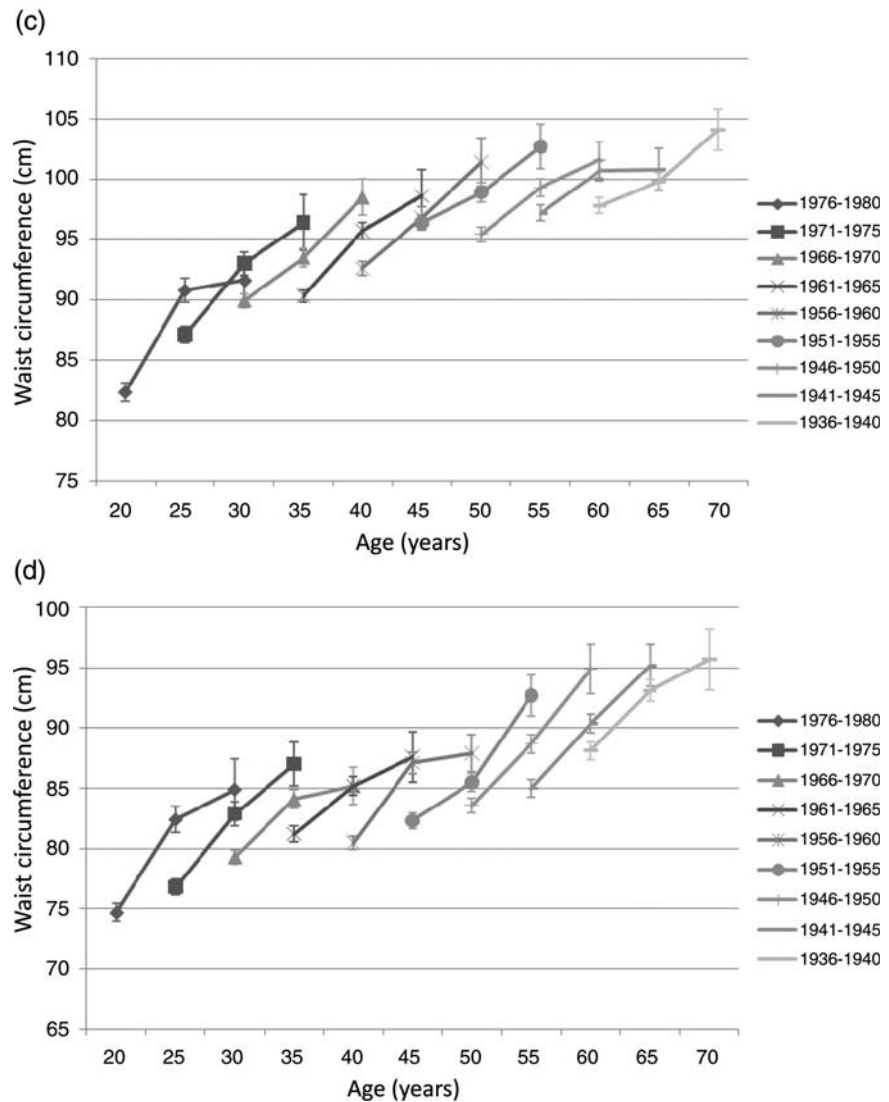
(c)

(d)

Fig. 2 (Continued).

complications may arise directly from obesity or mediated by other associated factors such as chronic low-grade inflammation[13] or even vitamin D insufficiency.[14] Although obesity is generally associated with increased risk of morbidity[15] and mortality,[16,17] there are continuing debates whether obesity is so harmful in the elderly[18] since the relative increase in mortality is less in older than in younger adults and 'ideal' body weight based on mortality is with BMI 25–30 kg/m². The 'obesity paradox' in the elderly may be explained by use of BMI,

which is influenced by muscle mass, rather than waist or other better measure of body fat. There are also mixed reports over the effects of weight loss on mortality in the elderly, although evidence is more clearly positive in the reduction of morbidity from arthritis, DM2 and cardiovascular risk factors. Research has tended to focus excessively on CHD risks, and insufficiently on the multiple effects of obesity on mobility, mood, quality of life and particularly in men, bladder function and sexual health. This review discusses the predicaments of the use of indices of adiposity in relation to morbidity and mortality in the elderly. Evidence from recent literature on the health benefits and risks of weight loss in these individuals will be addressed to provide recommendations for management of obesity in the elderly.

Definition of obesity

Obesity (ICD-9 278)[19] is the disease process characterized by excessive body fat accumulation with complex genetic–environmental aetiology, which results in multiple organ-specific pathological consequences. This definition refers BMI as indicator of adiposity. Although the age limits are not defined, it should be interpreted with caution in the elderly as discussed below.

Indices of adiposity in the elderly

Most surveys employ BMI (Quetelet's index). This is an acceptable index in younger adults for many purposes since it correlates reasonably well with body fat, measured by underwater weighing ($r = 0.7$–0.75) in younger adults. Errors are introduced in the elderly which limit the value of BMI because of a loss in fat-free mass (muscle) while the relative adiposity, and specifically the intra-abdominal fat, continues to increase. The original principle of the Quetelet's index was to eliminate the height factor by dividing weight (kg) by height squared (m^2).[20] However, height may be reduced substantially due to spinal shortening as a consequence of degenerative bone disease or kyphoscoliosis, contributing additional errors to BMI computation in the elderly. Frailty in the elderly also poses difficulty in obtaining weight and height. Increasingly, WC has been used as an index of adiposity in adults. The advantage of WC over BMI is that it correlates highly with both total and intra-abdominal fat. This metabolically active intra-abdominal fat plays a vital role in metabolic disorders including DM2 and Cushing's syndrome and accumulates with age. In adults,

unlike BMI, stature has been shown to have little influence on WC differences.[21] However, in the elderly, WC may underestimate the amounts of adiposity because of decreases in abdominal muscle tone.

There is confusion in the literature over the predictive value of WC. In groups or population with similar BMI, and similar total body fat (particularly thinner individuals) waist will predict the intra-abdominal fat mass which relates particularly to metabolic ill-health. In groups of population with a wide range of BMI and body fat (particularly fatter populations) the WC is dominated by total body fat. In most populations, WC is significantly correlated with both total body fat and intra-abdominal fat, as intra-abdominal fat co-varies with total body fat. Most of this information was derived in younger adults, and should be confounded for the elderly, although there is no *a priori* reason to suggest any difference.

The concept of 'sarcopenic obesity' has been introduced to indicate combination of obesity and muscle atrophy with reduced strength, which is thought to associate most highly with health risks. A consistent body of literature supports the notion that abdominal fat distribution and relative loss of fat-free mass is more important than BMI in determining the health risk associated with obesity in older ages.[22,23] Identification of elderly subjects with sarcopenic obesity is probably clinically relevant, but the definition of sarcopenic obesity, the benefits of its clinical identification as well as its relation to clinical consequences require further study. Our secondary analyses from the Scottish Health Surveys[6] illustrate the disproportionate increase in WC in older people compared with BMI, which indicates gain in fat but loss of lean tissue (Fig. 2).

Pathogenesis

There remains an inescapable truth that obesity is directly caused by a mismatch between energy expenditure and excessive energy intake, and the process is accentuated in the elderly, as sarcopenia and ageing *per se* result in lower basal metabolic rate. Obesity usually develops slowly, e.g. 1 kg/year, through very small (undetectable) energy imbalance, generated by complex rafts of behavioural, psychological, social and metabolic factors. Endocrine disturbances including accelerated decline in endogenous hormones such as testosterone, oestrogens and insulin-like growth factor and changes in neurohumoral modulators of appetite and body composition such as leptin and ghrelin have been implicated through associations mainly in cross-sectional studies but there is seldom a primary endocrine defect or drive to weight gain. It is

estimated that in men a change in BMI from non-obese to obese results in a fall in testosterone equivalent to being 15 years older.[24]

There is often confusion between 'healthy diets' for CHD prevention, and those for weight control. Certain diets will promote CHD, particularly through high saturated fat, salt and low fruit and vegetable consumption and these have not changed greatly in recent decades. The main dietary driver to obesity is a high-fat intake, with sugars also contributing for energy-dense foods and high-energy drinks. These issues tend to come together, since an energy-dense obesogenic diet usually tends to include excess saturated fats, excess salt and insufficient fruits and vegetables, but a 'healthy' diet from the cardiovascular perspective can still cause or maintain obesity. Reduced physical activity in recent decades also aggravates CHD risk as well as promoting further weight gain. The rapid rise in WC of older people probably relates most strongly to lower physical activity at work and in household activities. The current epidemic of obesity is clearly driven by external, environmental and dietary changes, not by new genetic or metabolic factors.[25] On the other hand, inter-generational epidemiology shows that these influences are mainly affecting families with existing obesity[5] so presumably a genetic vulnerability to inactivity and high fat diets. Co-morbidities such as metabolic/endocrine disorders and a number of drugs commonly used in the elderly including steroids, antipsychotics, antidepressants and antileptics and antihyperglycaemic agents, are known to promote weight gain.[26] In most cases, less 'obesogenic' drugs are available. Weight gain is an important reason for low compliance as well as compounding health problems through obesity.

Adverse effects of obesity

Associations between obesity and mortality

The observation that the BMI value associated with the lowest relative mortality is slightly higher in older than in younger adults has often been misinterpreted that obesity is not as harmful in the elderly. In fact, the absolute mortality risk associated with increased BMI increases up to the age of 75 years.[27] Beyond the age of 80 years, the association between BMI and mortality becomes weakened primarily because elderly with low body weight comprises a mix of those who have always been lean and physically active and those who have lost weight through chronic, covert or overt, ill health and cigarette smoking who are often physically inactive. Despite their similar low weight, these two groups have very different body composition and body fat distribution; this is exemplified by a recent analysis of 48 500

men and 56 343 women aged over 50 years at baseline.[28] Compared with individuals with low WC (men <90 cm, women <75 cm), people with large WC (men >110 cm, women >95 cm) were twice more likely to die from all cause over a period between 1997 and 2006. This risk was independent of BMI and persists in groups of men and women over the age of 70 years. In another 6-year perspective study of more than 4000 men aged 60–79 years,[27] all-cause mortality was shown to increase (relative risk: 1.55, 95% CI: 1.01–2.39) in those with WC ≥102 and low mid-upper arm circumference (a marker of sarcopenia).

A small proportion (<10%) of obese individuals have been observed to live into their old age with few health problems. These may represent a selective group of survivors who are less prone to complications of obesity. This notion is supported by a recent analysis of 1325 obese individuals from 8356 subjects, aged between 28 and 75 years, which found that of the 90 (6.8%) individuals with BMI >30 kg/m^2 who were free of metabolic disorders at baseline, only 1 went on to develop cardiovascular event over a median follow-up of 7.5 years, which was not significantly different from their counterparts with BMI <30 kg/m^2.[29] Whether genetic factors play a role in preventing cardiometabolic risk in this group of obese individuals, resulting in increased survival into older age, is currently unknown.

Associations between obesity and morbidity

Metabolic disturbances

By far, the strongest disease associated with overweight and obesity is with metabolic syndrome and DM2. Elderly who are physically active and with small WC have reduced risk of DM2[30] or other features of the metabolic syndrome.[31] The relationship between obesity and high blood pressure persists in advancing age[32,33] with WC being a better predictor than BMI.[34] Dyslipidaemia (reduced HDL-cholesterol and raised triglyceride levels) correlates with WC.[35,36] The prevalence of DM2 in men aged 40–79 years increases with increasing BMI or increasing WC (Fig. 3a). Among obese men (BMI ≥30 kg/m^2 or WC ≥102 cm), DM2 increases linearly with age and the highest prevalence of ∼20% is found in the oldest group (aged 70–79 years) (Fig. 3b). The Table 1 shows that the prevalence of DM2, overall, is lower in men with WC <102 cm (even when BMI ≥30 kg/m^2) and higher in men with WC ≥102 cm and the prevalence is accentuated in men with a combination of WC ≥102 cm and ≥30 kg/m^2. The patterns remain consistent across all age groups including the oldest group of men between 70 and 79 years. This observation suggests that even in elderly men, fat redistribution (as reflected by WC) is more importantly
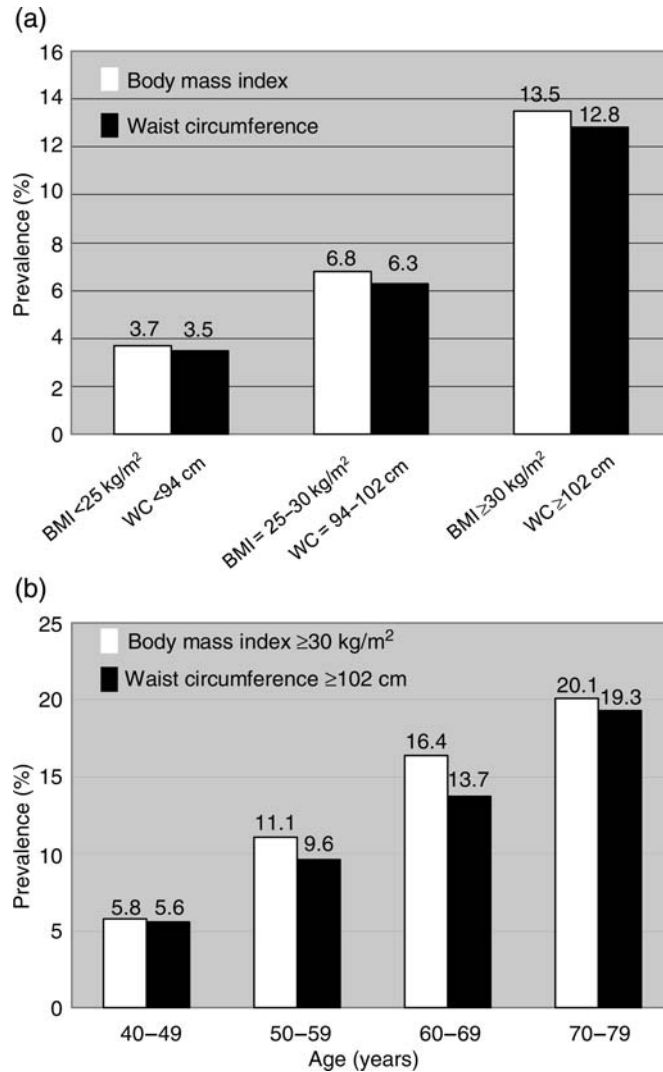
Fig. 3 **(a)** Prevalence of DM2 in men in different categories of BMI or WC in the European Male Ageing Study (courtesy of A. Tajar, F.C.W. Wu *et al*., unpublished data). **(b)** Prevalence of DM2 in obese men (BMI $\geq$30 kg/m^2 or WC $\geq$ 102 cm) in different age categories in the European Male Ageing Study (courtesy of A. Tajar, F.C.W. Wu *et al*., unpublished data)

Table 1 Prevalence of DM2 in men aged 40–79 years categorized by BMI and WC in different age groups.

	Number and proportion (%) of men with DM2				
	Entire sample	40–49 years	50–59 years	60–69 years	70–79 years
BMI <30 kg/m^2 + WC <102 cm	99 (4.9)	4 (0.8)	17 (3.1)	37 (7.6)	41 (8.9)
BMI <30 kg/m^2 + WC $\geq$102 cm	39 (9.4)	2 (3.0)	5 (5.6)	8 (6.9)	24 (16.7)
BMI $\geq$30 kg/m^2 + WC <102 cm	2 (2.7)	0 (0.0)	2 (7.4)	0 (0.0)	0 (0.0)
BMI $\geq$30 kg/m^2 + WC $\geq$102 cm	107 (14.6)	10 (6.8)	23 (11.6)	36 (17.6)	38 (21.0)

associated with DM2 than BMI (a measure of fat and lean mass). At the age of 65, variation in BMI has a very large influence on the remaining lifetime risk of DM2, from ∼3% with BMI <18.5, 29% with BMI 30–35 kg/m^2 and 35% with BMI ≥35 kg/m^2.[37]

While longitudinal studies have consistently shown WC as the strongest predictor of DM2, cross-sectional studies have tended to show slightly stronger relationships with waist-to-hip ratio (WHR). This ratio has no biological meaning and relates only weakly or not at all to body fat, obesity or fat distribution. A given ratio may represent any particular combination of waist and hip circumferences, and a ratio of large waist to large hip circumference is not biologically the same as a ratio of small waist to small hip circumference. It has been postulated that a high WHR is particularly hazardous in cross-sectional studies because it reflects recent reduction in hip circumference, as a result of loss of muscle mass and low physical activity. This is particularly the case for people with DM2.[38,39]

The relationships between obesity, age-related decline in sex hormones and metabolic disorders in older individuals are complex. Late onset hypogonadism in men is more frequent in the obese and has been shown to relate to features of the metabolic syndrome.[40] In women, these relationships are even more complicated. A fall in ovarian-derived oestrogens in premenopausal women increases the risk of cardiometabolic disorders including insulin resistance, but obese postmenopausal women, who are at greater risk of metabolic disorders than their lean counterparts, have higher total concentrations of oestrogens. Oestrogens in postmenopausal women are primarily derived from aromatization of androgens in adipose tissue, which is accentuated by a lack of ovary-derived oestrogens in the postmenopausal state. However, exogenous oestradiol administered to ovariectomized mice has been shown to increase inflammation in adipose tissue but did not cause metabolic disturbances such insulin sensitivity or glucose tolerance.[41] It has been suggested that, in postmenopausal states, androgens derived from adipose tissue progressively predominate over the aromatized oestrogens, leading to metabolic disturbances through an increase in intra-abdominal fat accumulation.[42]

Effects of obesity on cardiovascular disease

It has been suggested, from large studies, that obesity may cease to promote CHD in older people, and even that modest degrees of overweight and obesity could be cardio-protective in the elderly. Not all studies agree with this simplistic interpretation. For example, a longitudinal study of 621 men and 960 women with a mean age of 77 years[43] has shown that BMI of ≥27 kg/m^2 was associated with increased risk of CHD in later life (RR: 1.7, 95% CI: 1.3–2.1). When

those with weight loss of $\geq 10\%$ has been excluded, the risk associated with heavier weight in old age was also observed (RR: 1.4, 95% CI: 1.0–1.9), less frequent in those who are still obese. This study points to the adverse effect of unintentional weight loss. The contribution of weight to risk of CHD in older people may be underestimated if weight history is neglected.[43] In another longitudinal study of 737 men and 860 women aged 70 years,[44] compared with the lowest quintiles, the 15-year relative risk of CHD was 1.36 (1.00–1.85) and 1.42 (1.04–1.92) in men with WC and BMI in the highest quintiles, respectively. These findings were not observed in women.[45] Visscher *et al.*[16] found that, in the Rotterdam Study BMI did not predict CHD in elderly men, but WC ≥ 94 men or ≥ 80 women did. An interpretation of this is that lower BMI in older people can improve loss of muscle mass, whereas high WC, i.e. higher body fat is still hazardous.

Arthritis

Obesity and arthritis tend to go hand in hand. In obese individuals, sustained period of excessive strain results in osteoarthritis of weight-bearing joints,[46,47] but people with severe arthritis who are unable to perform physical activities, resulting in reduced energy expenditure may have greater risk of weight gain. The onset of rheumatoid arthritis, as related conditions often involves a major inflammatory response with malaise, loss of appetite and weight loss. Obesity has also been linked to osteoarthritis of small joints[48] and increases the risk of gout[49] and recently to psoriatic arthritis[50] suggesting that an increased inflammatory response (changes in levels of adipokines such as adiponectin and leptin) may also be involved in the development of arthritic diseases among obese individuals.[51]

Effects of obesity on respiratory function

Shortness of breath is one of the commonest symptoms of the obese, as a result of the increased metabolic rate in obesity, and the increased metabolic demand of minor exertion. This is commonly misdiagnosed as asthma,[52] or as heart failure,[53] particularly if there is peripheral oedema (another purely mechanical complication of obesity). Excessive adipose tissue on the neck, thorax and abdomen results in a number of pulmonary function abnormalities including obesity-hypoventilation syndrome and obstructive sleep apnea.[54,55] These abnormalities are a consequence of obesity-induced reduction in chest wall and lung compliance, small airway dysfunction and expiratory flow limitation, reduction of ventilatory muscle strength and endurance, increased work and oxygen cost of breathing and abnormal ventilation/perfusion relations and arterial oxygen desaturation.[56] Asthma has been reported

to be increased in the obese.[57] This may be partly through aggravated systemic inflammatory processes, with high circulating cytokines.

Urinary incontinence

Whilst obesity is a well-known risk factor for urinary incontinence in women,[58] little information is available in men. In women, the prevalences of all types of urinary incontinence (urge, stress or mixed) increase with increasing BMI,[59] as this problem persists into older age to aggravate an already-common problem.[60,61] In obese individuals, it is often thought that urinary incontinence is due to the effects of excessive weight exerting strain and stretch and eventual weakening of the neuromuscular structures. However, there is more evidence that urinary incontinence in obese individuals is due to raised intra-abdominal and intravesical pressure as the result of increased weight of the abdominal wall.[62,63] The obese diabetic is at particularly high risk of incontinence, if autonomic neuropathy and/or osmotic effects are present.

Cancers

Obesity increases the risks for many cancers tipping the balance for individuals whose other risk factors would not have resulted in cancers. It is estimated that between 7 and 15% of breast cancer cases and 11–14% of bowel cancer cases are attributable to obesity.[64] A pooled analysis of 8 cohort studies of more than 300 000 women has shown that postmenopausal women with BMI ≥ 28 kg/m^2 have 26% increase in risk of breast cancer compared with those with BMI <21 kg/m^2.[65] A more recent analysis from the European Prospective Investigation of Cancer study has shown a 31% excess risk of breast cancer in postmenopausal women with BMI ≥ 30 kg/m^2 compared with those with BMI <25 kg/m^2.[66] The American Cancer Society's Cancer Prevention Study II[67] has shown a risk of colon cancer greater by 75% in men and 25% in women with a BMI ≥ 30 kg/m^2 compared with <25 kg/m^2. Obese postmenopausal women are also at high risk of malignant melanoma and endometrial cancer,[68] and obese elderly adults have increased risk of other cancers, including gallbladder, pancreatic, renal, bladder, uterine, cervical and prostate cancers.[69,70]

The increased risk of cancers in obese individuals is probably due to changes in a number of hormones.[71] Links between sex steroids and cancers and steroid-sensitive tissues or organs such as breast, endometrium, ovary and prostate have been made.[72,73] The higher oestrogen levels in obese postmenopausal women than in their leaner counterparts are thought to promote carcinogenesis. There is evidence that some of the sex hormones have been shown to favour the selective growth of pre-neoplastic and neoplastic cells.[74]

Interest has also focused on the effects of hyperinsulinaemia on cancers since epidemiological evidence has shown significant associations between hyperinsulinaemia and many cancers, with excessive adiposity playing a pivotal role. A prospective study of more than 14 000 New York women has shown that compared with women in the lowest quartile of C-peptide (a marker of endogenous insulin production related to body fat content), those in the top quartile had a 3-fold higher risk of breast cancer and a 4-fold risk of colorectal cancer.[75] Similar observations have been made in men, in the Physicians' Health Study.[76] Compared with men in the lowest quintile of C-peptide, men in the top quintile had a 2.7-fold risk of colorectal cancer. In epidemiological data, it is difficult to pinpoint whether hyperinsulinaemia has a direct effect on carcinogenesis as part of the insulin resistance syndrome since hypertriglyceridaemia and hyperglycaemia have also been shown to associate with increased risk of colon cancer.[77]

Obesity may also cause cancers through other means, for example, oesophageal cancer is increased because of local inflammation gastro-oesophageal reflux disease (GORD). Barrett's oesophagus is a condition of GORD caused by acid reflux resulting in metaplasia of the distal oesophagus, is increased with obesity[78] and may progress to oesophageal cancer.

Impaired quality of life

The most widely used method of assessing quality of life is the well-validated short form 36 health survey questionnaire. This method covers eight health domains ranging from physical function to mental health. There are many studies demonstrating obesity is associated with poor quality of life in older people including impaired physical function, increased bodily pain and lack of vitality.[79–81]

Impaired physical function

The ageing-related progression of sarcopenia and degenerative joint disease is exacerbated by obesity. A vicious cycle is established when such individuals become less physically able, progressing to the inability to perform simple activities of daily living. Consequently, frailty develops when impairment in function and reduction in physiologic reserves are severe enough to cause disability.

Several recent studies have found that obesity in older age and elderly men and women is associated with increased risk for difficulties in performing physical functions. A recent study of Canadian men and women aged 68–82 years, using objective measures of physical

capacity, has shown that obesity was related to reduced capacity in performing 'Timed Up and Go', chair stands, walking speed at normal and fastest pace and one leg stand.[82] In a study of French women aged >75 years, using self-reported questionnaire, obesity was associated with difficulty in performing simple tasks such as walking, climbing stairs, going down stairs, rising from a chair or bed, picking up an object from the floor, lifting heavy objects or reaching an object and moving and these risks were further increased if sarcopenia was present.[83]

Frailty is greatly increased with obesity. A recent data analysis of 3055 community-dwelling men and women aged >65 years who participated in the English Longitudinal Study of Ageing[84] has shown that frailty or frailty index was related to BMI in a U-shaped manner (i.e. increased frailty in people with extremes of low or high BMI). However, in people with large WC (≥88 cm in women and ≥102 cm in men), frailty exists in all BMI categories. Similarly, Visser *et al.*[85] found that mobility–disability in men aged 65–100 years was predicted by high fat mass and not by low lean mass.

Impaired cognitive function

Obesity has been shown to associate with impaired cognitive function. Some mediating factors such vitamin D insufficiency have been implicated but low vitamin D levels are inextricably linked to physical inactivity, so reverse causality may act. Intriguingly, recent studies have shown that brain volume is reduced in obese subjects.[86] Obesity in midlife has been linked to the development of vascular dementia and Alzheimer's disease.[10,11] A recent study of 91 apparently healthy men and women aged between 60 and 80 years has shown that moderate increase in WC (≥94 cm in men and ≥80 cm in women) or hypertriglyceridaemia was associated with subclinical vascular damage.[87] In the elderly, the obesity-related gene *FTO* has also been linked to Alzheimer's disease[88] but an excess of cerebral vascular insufficiency and long-term hypoxia through sleep apnoea probably play major roles. Decline in cognitive function has also been observed to associate with vascular risk factors including dyslipidaemia[89] and hyperglycaemia[90] and the metabolic syndrome in the presence of inflammation.[91]

Weight loss in the elderly

In general, obesity brings a list of medical complications and symptoms, which affect every body system, and even modest intentional

weight loss brings multiple benefits. However, these benefits cannot be assumed to apply in the elderly, and the balance against possible hazards from treatment may be different from those in younger people. Parts of the literature also suggest increased mortality, in elderly lean people, with lowest mortality at higher BMI than in younger people. An 'obesity paradox' is suggested by some evidence that obese subjects have lower mortality than lean.[92] As mentioned earlier, BMI categories derived from younger subjects may be inappropriate in the elderly as reduction in BMI with age frequently involves loss of muscle rather than fat in observation studies. BMI is not a good predictor of CHD or immobility in elderly people. Waist as a better indicator of total body fat is a better predictor.[16,85]

Rumpel *et al.*[93] and Losonczy *et al.*[94] have shown that excess mortality in lean individuals only occurs in those who had lost weight and is not observed among those who have always been lean. Persons who have always been lean are generally healthier than those of similar weight who have lost weight due to conditions that cause unintentional weight loss such as cancers, chronic heart and lung disease. This presents a problem in the interpretation of body weight in the elderly. Furthermore, persons who achieve weight loss intentionally, through lifestyle modification or pharmaceutical intervention, generally acquire a range of clinical benefits and quality of life, whereas unintentional weight loss is most commonly through illness and usually involves loss of lean tissue, which carries a poor prognosis, even if there is subsequent regain which may involve fat tissue or oedema.[95]

Weight loss and mortality

Epidemiological studies have consistently shown that reported weight loss (usually unintentional) leads to increased mortality—even if this is regain (weight cycling). The study of Lean *et al.*[96] showed a convincing increase in life expectancy from intentional weight loss in mainly elderly patients with DM2, using simple dietetic methods. Data from Williamson *et al.*[97] supported this finding in people with DM2, but not for non-diabetic subjects. The older, mostly diabetic, patients in the SCOUT trial with sibutramine to induce weight loss showed ~1% increase in non-fatal MI but no increase in mortality.[98] Weight loss induced by bariatric surgery has been shown to increase survival in extremely obese patients but most elderly obese are excluded by this procedure.

Weight loss and body composition and bone mineral density

Weight loss not only results in reduction of fat mass but also of fat-free mass and possibly bone mineral mass and this may have adverse consequences in the obese elderly. A regimen of diet in conjunction with exercise may prevent these adverse effects. Randomized controlled trials in obese individuals aged over 65 years have shown that fat-free mass could be preserved using this method.[99,100] Regular weight-bearing exercise in addition to diet induced weight loss has been shown to prevent femoral neck bone loss in obese individuals.[101]

Weight loss and morbidity and quality of life: evidence from interventional studies

Although there are few interventional trials of weight loss in obese elderly and health benefits, many studies have included elderly subjects. In obese elderly persons, moderate weight loss of 5–10% has been shown to improve cardiovascular risk factors,[102,103] and if combined with exercise, improve physical function and health-related quality of life.[104] A small 6-month outpatient randomized controlled trial conducted in obese (BMI $\geq$30 kg/m^2) adults aged >65 years who were randomly assigned to diet and exercise therapy (treatment group; $n =$ 17) or no therapy (control group; $n =$ 10) has shown an 8.4% weight loss was accompanied by significant reduction in cardiometabolic risk factors without any adverse effects.[105] In the Diabetes Prevention Program[106] of obese men and women up to the age of 84 years (mean: 50.6 years, BMI: 33.9 kg/m^2), weight loss was achieved by a programme of moderate physical activity of at least 2.5 h a week and reduction in total dietary fat to <25% of calories, coupled with a lifestyle counsellor weekly over a 16-session curriculum and at least bimonthly thereafter. As a result, every kilogram of weight loss through diet and exercise reduced 16% in the incident of DM2 over 3.2 years. When these data were analysed based on age groups, diabetes incidence did not differ by age in the placebo group (11.0, 10.8 and 10.3 cases per 100 person-years in the 25–44, 45–59 and 60–85 years old groups, respectively; $P = 0.71$). Intensive lifestyle intervention was effective in all age groups, DM2 incidence rates fell with increasing age (6.3 vs. 4.9 vs. 3.3 cases per 100 person-years in the 25–44, 45–59 and 60–85 year age groups, respectively; $P_{trend} = 0.007$).[107]

Bales and Buhr[108] examined 16 randomized controlled trials of weight loss interventions in people aged over 60 years with baseline BMI >27 kg/m^2 who lost >3% or 2 kg in weight over 6–12 months. This review has revealed that weight loss interventions led to significant benefits for those with osteoarthritis, CHD and DM2, while having

slightly negative effects on bone mineral density and lean body mass. However, interventions in these trials were relatively short term and by excluding studies showing a reduction of <3% or 2 kg in weight may have resulted in bias. Systematic analysis[109] and meta-analysis [110] of longer term interventions have been performed. In the systematic analysis by McTigue *et al.*,[109] weight loss of 3–4 kg over 1–3.3 years through intensive lifestyle modification led to improved glucose tolerance and physical functioning, reduced incidence of new DM2 and a combined hypertension and cardiovascular endpoint, but also resulted in reduced bone density. A recent meta-analysis by Witham and Avenell[110] of nine trials of obese older adults with mean BMI ≥30 kg/m^2 and age ≥60 years with longer term outcomes (at least 1 year) shows mixed results. On average, there was a reduction of 3 kg of body weight at 1 year. There was no significant change in lipids. One of the studies did demonstrate significant decrease in recurrence of hypertension or cardiovascular events (hazard ratio: 0.65; 95% CI: 0.50–0.85). Improvement in quality of life was observed in one of the two studies.

Weight loss and heart failure

There is currently a scientific paradox, resulting in therapeutic uncertainty and clinical inactivity, over the treatment of obesity in heart failure.[111] High BMI is a very well-established aetiological risk factor for heart failure[112] (by several potential mechanisms importantly including obesity-related hypertension) yet prognosis amongst patients with heart failure has been reported to be worst for thin, cachectic patients and not clearly worse for the obese.

Heart failure is common, affecting 6% of all men at age 50 and 15% aged 51–55.[113] About 10% of all men and women aged >65 are admitted to hospital for heart failure and the number of hospitalizations with heart failure has been reported in many Western countries.[114] It is a serious health hazard, with prognosis similar to some cancers with survival of 89, 79 and 59% at 1, 2 and 5 years.[115]

Obesity increases the risk of congestive heart failure through multiple mechanisms affecting all the major risk factors for CHD, lipids, coagulant and antithrombolytic, and blood pressure. Jordan *et al.*[114] estimate that ~60–70% of all hypertension can be attributed to obesity. There is increased demand for cardiac output by expended lean body mass and metabolic rate, with increased heart rate and blood volume. As a consequence, there is increased cardiac mass and altered cardiac geometry.[116] On the other hand, smoking is a powerful risk factor for heart failure but associated with disproportionately lower peripheral muscle mass and greater central fat distribution than expected for a given BMI.[117]

Confusing counter evidence with heart failure concludes that elevated BMI is not an adverse prognostic factor. Lissin et al.[118] studied 522 US veterans over 6 years and found an immense relationship between BMI and survival, together with exercise capacity, age and aetiology. However, they did not (appear to) adjust for smoking, which is usually less frequent in the obese. Davos et al.[119] studied 589 heart failure patients over 1 year. The results were not significant for a relationship between BMI quintile and survival and were not adjusted for smoking. Their conclusion that thinner patients do worse should be interpreted with caution. Lavie et al.[120] studied 208 patients referred for possible cardiac transplantation for 2 years. They found that survivors had lower subcutaneous skinfold thicknesses (interpreted as lower body fat), but no difference in BMI from those who had clinical events. There was no adjustment for smoking.

There remains a need for good quality research to establish the prognostic importance of obesity in heart failure, particularly in the more common, less severe, hypertension-related disease and the effect of intentional weight loss. It is possible, however that the apparent association between heart failure and obesity is spurious if the diagnosis of heart failure is based only on symptoms of breathlessness and oedema, which may be attributed entirely to obesity. Again the use of BMI may have confused the picture, since less of muscle mass (thus low BMI) is so common, even in the overweight or obese. The WC may avoid this.

Treatment guidelines

Lifestyle intervention

Advice from evidence-based guidelines has been consistent, and does not differ by age.[121–123] In the elderly, the aim of weight loss is to improve physical function and quality of life with less emphasis on cardiovascular risks, but both outcomes apply at all ages.[124] Lifestyle intervention is just as effective in older as in younger individuals.[125] Modest weight loss of 5–10 kg with conventional diet and exercise programme generate a relatively large early reduction in intra-abdominal fat and metabolic improvements. Mood and mobility also improve with quite modest weight loss. Weight loss management programme should be tailored according to the individual needs in order to provide a balanced diet, appropriate level of calorie intake and physical activity as well as duration of therapy. Elderly patients on drugs whose action, or half-life, might be altered by weight loss. These need to be monitored. They include diuretics, antihypertensive, hypoglycaemics, warfarin, digoxin and many others. Relatively modest

weight loss may remove the need for some drugs, which should be withdrawn. These include H$_2$ blockers, analgesics and antidepressants and those listed above. Outcomes on clinical benefits and risk should be monitored routinely and therapeutic adjustments should be applied as required.

Very-low-energy diets (VLED) or low-energy liquid diets (LELD) have conventionally been avoided for elderly patients. There is consequently little or no evidence for, or against, their use in the elderly. The main hazard is the development of gall stones in ~10%, of which 60% are symptomatic.[126] Evidence suggests little difference between VLED 400 kcal/day and a more liberal 800 kcal/day which allows some fat intake, to reduce gallstone problems.[127] Although VLED/LELDs may provide more rapid weight losses in short-term, they are insufficiently better in long-term than moderate calorie diets of 1000–1500 kcal/day to warrant their use under current guidelines.[128] Long-term weight loss maintenance strategies are now becoming available, which may make them more useful. The recent National Obesity Forum statement[129] suggests that there should be no age contraindication for VLED but medical review should be provided for patients over the age of 70 (although there is no evidence for problems in the elderly). Special caution is needed with a range of drugs, especially antidiabetic, antihypertensive and diuretic drugs as with less drastic energy restriction.

Pharmacotherapy

Polypharmacy is common in the elderly, thus benefits and risks of therapy should be considered when introducing additional medications. The only evidence-based and licenced antiobesity medication is now orlistat, a saturated derivative of lipostatin.[130] There is no reason to suppose that its effectiveness or safety would be any different in elderly patients. It is not absorbed by the gastrointestinal tract and binds to intestinal lipases to prevent ~30% of dietary fat absorption. About 8–10% weight loss after 1 year may be achieved by orlistat and older persons have been shown to benefit similar amount of weight loss as younger adults.[131,132] Its adverse profile is restricted to gastrointestinal effects such as steatorrhoea. This only occurs if high-fat meals (>20 g fat) are consumed, but it can be more troublesome in the elderly who may already suffer from faecal incontinence and when dietary adherence is more difficult. Fat soluble vitamins, especially vitamin D, are reduced by orlistat.[133] Values did not fall into the deficient range, but it is advisable to monitor vitamin D levels and to take a fat-soluble multivitamin supplement (2 h before orlistat). It is unlikely that orlistat

would cause sufficient malabsorption of fat soluble vitamin supplements to cause deficiency, but this has not been tested.

Sibutramine is a centrally acting, monoamine reuptake inhibitor, blocking the reuptake of both serotonin and noradrenaline leading to increased satiation.[134] It has recently been withdrawn in most countries (including the USA and Europe) because of a clinical trial in mainly older very high-risk patients with active CHD, who fell outside its licence. Doctors would need to balance the clinical benefits of treatment to individual patients (mean loss of 17 kg, and significant fall in blood pressure) against a possible 1% increase in non-fatal myocardial infarction and stroke, even though that risk was mainly limited to patients already excluded under the licence.

Surgery

There are clear recommendations from the UK National Institute of Clinical Excellence about who should be considered for bariatric surgical procedures.[123] There are various procedures including purely restrictive gastric banding and those which add malabsorption, e.g. gastric bypass, all of which have potential immediate complications, which need to be balanced against long-term benefits. Criteria for bariatric surgery are defined as BMI $\geq$40 or 35–40 kg/m^2 with other significant disease (e.g. sleep apnoea, DM2, high blood pressure) that could be improved by weight loss. The individual is required to have failed with all appropriate non-surgical procedures to achieve clinically beneficial weight loss for at least 6 months, to be generally fit for anaesthesia and surgery, and to commit to the need for long-term follow-up. There are no age guidelines provided but majority of patients undergo bariatric surgery are <60 years.[135] Bariatric procedures in older groups (>60 years) have greater perioperative complications and lower success in achieving reduction of weight and secondary complications mainly because of pre-existing heart disease.[136–138]

Conclusions

Obesity is a common but increasing problem in the elderly. Its prevalence is lower in extreme old age but this is also likely to increase in the future. Decreased physical activity and decreased energy expenditure with ageing predispose to fat accumulation and fat redistribution, but muscle loss, so BMI may not increase with adiposity. WC gives a better indication of adiposity and of sarcopenic obesity. There is

usually some height loss with ageing, making BMI data even more difficult to interpret in the elderly. Reduction in muscle mass is an important determinant of physical function and metabolic rate and leads to the clinical hazards of obesity appearing at a lower BMI in older people. WC may be a better predictor of health risks, particularly CHD, than BMI. Many of the medical consequences of obesity are particularly problematic for elderly people, e.g. DM2, arthritis, urinary incontinence and depression. Chronic inflammation and endocrine changes contribute to the changes in metabolism and body composition that accompany ageing. The focus of treatment should be on reduction of intra-abdominal fat initially with *modest*, conventional diet restriction, and preservation of muscle mass and strength through physical activity. A reduction in polypharmacy is a valuable target for weight management intervention. There is limited information on efficacy and safety from pharmacotherapy in the elderly, but no reason to suspect different results; however, complications of bariatric surgery increase in those>60 years. Obesity, and specifically sarcopenic obesity, in the elderly is potentially preventable, should be tackled from younger ages, and also during major later life transitions such as retirement.

Acknowledgement

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