



Australian and New Zealand Society for Geriatric Medicine
Position Statement No 19
Obesity and the Older Person

Key Points

- 1) Many more Australians classified as overweight and obese are reaching old age.
- 2) Weight loss in the elderly is associated with muscle mass loss.
- 3) Therefore, primary health care measures must target the young and middle-age if the prevalence of obesity is to be reduced in older age.
- 4) Current classification systems for obesity developed for the young should not be directly applied to older people.
- 5) Weight loss should never be recommended in older people purely because of the body mass index or waist circumference measurements.
- 6) Clinician judgment is required in deciding if weight loss is required. Weight loss should only be recommended where there is likely to be benefit to the individual.
- 7) Weight loss in the elderly is associated with muscle mass loss and therefore, any weight loss programs in the elderly must aim to preserve muscle mass.
- 8) Exercise and adequate protein intake are key ingredients of any hypocaloric weight loss program. It is important also to ensure that there is adequate micronutrient intake especially in terms of calcium and Vitamin D.
- 9) There must be supervision of the weight loss program by trained clinicians.
- 10) There is little evidence for use of surgical methods and pharmacological treatment for weight loss in overweight or obese older people.
- 11) Further research is required in terms of:
 - a) Reducing the incidence and prevalence of obesity in young and middle age;
 - b) Managing the morbidly obese older individual allowing for weight loss whilst preserving or strengthening muscle.

This Position Statement represents the views of the Australian and New Zealand Society for Geriatric Medicine. This Statement was approved by the Federal Council of the ANZSGM on 22nd August 2011.

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(Submitted 9th December 2010, Revised 26th April 2010)

Defining older age

Older adults are commonly identified in three different subgroups; those aged 45-64 years (middle-aged), those aged 65-79 years (younger old) and those aged 80 years and over (older old) (1). For the purpose of this position statement, the authors have defined older age as people aged 70 years and older.

Under-nutrition and the Older Person

As people age, there is increased prevalence of under-nutrition and sarcopenia. This must not be overlooked: Australia & New Zealand Society for Geriatric Medicine Position Statement- 'Under-nutrition and the Older Person'.

The Concept Healthy Ageing In Relation To Weight

Generally speaking, Australians are already overweight and obese when reaching older age. In keeping with the concept of 'Healthy Ageing', efforts targeting obesity should be directed at the young and middle-age as this will reduce the number of obese older people. Similarly, efforts should be in place to build lean mass and bone mass across the lifespan ensuring that disease states such as osteoporosis and sarcopenia are less common in old age.

Weight Loss in the Elderly Has Risks

Weight loss in older people occurs commonly and whether unintentional or intentional is associated with poor health outcomes [see (2) for review]. The prospective Cardiovascular

Health Study reported that 17% of the subjects had lost 5% or more of their initial body weight after three years (3). Over the next four years this group had significantly greater total ($2.09 \times \uparrow$ [95% confidence interval 1.67-2.62]) and risk-adjusted mortality ($1.67 \times \uparrow$ [1.29-2.15]) than the stable weight group. This increased mortality risk occurred irrespective of the starting weight and apparently irrespective of whether weight was lost intentionally or not. It is likely that the increased risk of mortality is due to the loss of lean mass or development of **sarcopenia**. When body weight decreases in older people, lean body tissue is lost disproportionately (4, 5). Even when weight is subsequently regained, the gain in lean mass does not make up for the loss of lean mass during the weight loss period (6).

Obesity in the Older Person: Epidemiology

The prevalence of obesity has risen dramatically, from 200 million in 1995 to over 300 million in 2005 (WHO). Approximately 1.1 billion people are currently overweight and it is expected that this total may rise to over 1.5 billion by 2015 (7). The prevalence of obesity increases in successive age groups and is greatest in the 55–64 year age group, with 29% of Australian men and 36% of Australian women in that age group considered obese (8). The prevalence of obesity is lower in Australians aged 75 years and older with 14% of men and 17% of women being classified as obese. Although cross-sectional data show that the average weight is highest among people of late middle age, this does not mean that all individuals lose weight as they age beyond middle age. Longitudinal studies indicate that there is disproportionately high mortality among obese people in middle age

(9-11). In addition, prospective data from the Australian Longitudinal Study on Women's Health indicates that on average, there is a decrease in weight among women in their 70s, with some women gaining and others losing weight at this life stage (12). It is uncertain if weight gain in older age is associated with undesirable health outcomes as it may really be a marker of good health. The prevalence of overweight and obesity is increasing in the older population with an average older Australian now 6–7 kg heavier than 20 years ago (10, 11).

What contributes to overweight and obesity?

Overweight and obesity are caused by an energy imbalance, where energy intake exceeds energy expenditure over a considerable period of time. Hence good nutrition and adequate levels of physical activity play an important role in the prevention of further weight gain throughout the life cycle. Global increases in overweight and obesity are attributable to a number of factors. These include a global shift in diet towards increased intake of energy-dense foods that are high in fat and sugars but low in vitamins, minerals and other micronutrients, and a trend towards decreased physical activity due to the increasingly sedentary nature of many forms of work, changing modes of transportation, and increasing urbanisation (13).

Classifying Overweight and Obesity

There is incontrovertible epidemiological evidence linking excess body weight to adverse health outcomes, however there remains uncertainty regarding the most appropriate way to define excess weight. As

shown in the table 1, weight classification criteria have been specified by the World Health Organisation (WHO) to provide a scheme for classifying health risk according to body mass index (BMI) despite the fact that there is a continuous relationship between body weight and health risk (14).

Table 1: World Health Organization criteria to classify overweight and obesity by body mass index (BMI) and waist circumference (WC).

BMI	Classification	Health risks
<18.5	Underweight	Increased risk
18.5 - 24.9	Normal weight	Least risk
25.0 - 29.9	Overweight	Increased risk
30.0 - 34.9	Obese Class I	High risk
35.0 - 39.9	Obese Class II	Very high risk
≥40.0	Obese Class III	Extremely high risk
Waist circumference		
Men		
94-101 cm		increased
≥ 102 cm		substantially increased
Women		
80-87 cm		increased
≥ 88 cm		substantially increased

While BMI is strongly correlated with fat and lean mass, it does not account for variations in body fat distribution in individuals or population groups (e.g. pathogenic android distribution compared with less a harmful gynoid distribution), and a given BMI may not

correspond to the same percentage fat and lean mass across individuals. Similar limitations apply to WC as different levels of risk associated with a given WC occur within ethnic populations. The WHO defined BMI cut-offs as shown to reflect risks for mortality, type 2 diabetes and cardiovascular diseases without consideration of the effect of age on fat distribution or the occurrence of sarcopenia (discussed below). Weight simplified is a total of fat mass, lean mass, bone mass and water in the body. Therefore, when making a decision as to the need to treat or not and how to treat, clinicians should also take into consideration other factors such as the presence of sarcopenia and the presence of diseases resulting in fluid accumulation. Also, as discussed below, the decisions as to where to place cut-offs were somewhat arbitrary and studies evaluating the associations of the cut-offs with co-morbidity have not necessarily included large numbers of older people, especially those aged 80 years and older and so may not necessarily account for health changes that occur as a result of ageing (e.g. appetite and muscle mass loss).

The Classification System: Is It Applicable For Older People?

The optimal body mass index for life expectancy in older people is said to be in the range of 27-30 kg/m² and is in contrast to weight guidelines for younger individuals stating that the ideal body weight is between 18.5 to 25 kg/m² (15). In a systematic review, Heiat and colleagues concluded that the relationship between BMI and mortality in people over 65 years is a 'U-shaped' curve, with mortality rising only at BMI > 31 kg/m², and perhaps not at any BMI in people over 75

years (15). A combined analysis of the American NHANES I-III (1974-2000) studies revealed no significant mortality increase with any degree of overweight in people aged 70 years and older whilst an increased death rate was noted only in those with a BMI >35 kg/m² among those aged 60-69 years (16).

Historically, ideal weight was defined by insurance company data where overweight was considered as weights exceeding the average for a given height-age-sex group. This concept was advanced to desirable weight, defined as weight associated with lowest mortality, identified from insurance actuarial data. As reviewed by Shah et al, the first weight for height tables were published in 1913 (the Medico-Actuarial Mortality Investigation covered data on height and weight from 1885 to 1909), and then again in 1942-43 by the Metropolitan Life Insurance Company (MLIC) based on longevity (17). The 1959 MLIC tables were based upon data from 26 life insurance companies in the United States and Canada pooled from approximately 4.9 million insured 15-69 year old men and women from 1935 to 1954 and reported as The Build and Blood Pressure Study 1959. In 1983, tables were again produced by the MLIC based upon data from 4.2 million US and Canadian policy holders between 1950 and 1972 (The Build Study 1979), where subjects with significant coronary artery diseases were excluded. In these tables, data were grouped by frame size- light, medium and heavy. The limitations of the MLIC tables have been reported (18, 19).

As reviewed by Flegal et al, weight ranges recommended in the 1980 *Dietary Guidelines for Americans*, were recommended by a 1973

National Institutes of Health conference on obesity that were adapted from the 1959 MLIC table of “desirable” weights for men and women at least 25 y of age (20). In the 1980 guideline, low and high weights for males corresponded to BMI levels of 20.1 and 25.2 respectively and for females, 18.6 and 23.7 respectively. According to Kuczmarski and Flegal, reference values to identify the prevalence of overweight adults aged 25-75 in the United States were first reported in *Health United States* in 1984 (19). These cut-offs of 28.0 kg/m² for men and 35.0 kg/m^{1.5} for women represented the sex-specific 85th percentile for 20-29 year old participants in the second National Health and Nutrition Examination Survey (1976-1980, total persons aged 6 months to 74 years=20,322). Young adults were chosen for the reference population as they were lean and any age related increase in body weight was considered related to fat deposition. These cut off points were subsequently used to report overweight prevalence rates in all annual *Health United States* until 1998 and corresponded reasonably well with the MLIC cut offs identified by a 20% increase over desirable weights.

Few guidelines considered the effect of age in recommendations for BMI cut-off points, however in 1989, as reviewed by Kuczmarski and Flegal, the US Committee on Diet and Health recommended increasingly higher non-sex specific BMI cut offs for each decade of age from 19-24 years (19-24 kg/m²) through 55-64 years (23-38 kg/m²) and 65 years and higher (24-29kg/m²) (19). According to Flegal et al, the 1990 *Dietary Guidelines for Americans* recommended BMI for two age groups (19-34 years, low: 19.1 and high: 25.0;

35+ years, low: 21.0 and high: 27.1) independent of sex, with higher weights in the range more applicable for men, “who tend to have more muscle and bone,” and the lower for women, “who have less muscle and bone” (20). Flegal et al add that these BMI ranges were modified from those recommended in a 1989 report from the National Research Council (NRC) which were based on data suggesting that as age increases, the lowest mortality occurs at progressively increasing body weight (21, 22).

The 1995 *Dietary Guidelines for Americans* defined age independent “healthy” weights for heights corresponding to a BMI range from 19-25 kg/m². As reviewed by Kuczmarski and Flegal, the lower range of 19 was described as the 15th percentile and the Advisory Committee chose the upper range based upon observations of increased morbidity and mortality at BMIs above 25kg/m² and that a cut-off point below 25 would classify over half of the population as obese (19). Levels of moderate (25-28.0 kg/m²) and severe overweight (28- 29 kg/m² or greater) were also identified but an exact BMI value for severe overweight was deliberately not indicated due to a lack consensus on a precise threshold. As remarked upon by Kuczmarski and Flegal, the age neutrality of the 1995 guideline related to evidence from a study that was cited by the 1989 guideline to support age specific cut-offs (19, 21).

The current WHO BMI cut-offs were initially proposed by Garrow in 1981 (based on the 1959 MLIC tables) which were then endorsed by the 1997 WHO Consultation (14, 23). This document was then followed by the Clinical Guideline on the Identification, Evaluation, and

Treatment of Overweight and Obesity in Adults by the National Heart, Lung and Blood Institute. This guideline cited epidemiological studies linking the increased risk of morbidity and mortality to increased BMI (24-33). For example, some of these studies examined diabetes risk in relation to weight or weight gain in large cohort studies including the Nurses Health Study [$>100,000$ women, 30 to 55 years at baseline (1976) with up to 14 years follow-up (24, 25)], the NHANES-1, (1971-1987) Follow-up Study [$>11,000$ participants, 25-70 years, followed for 14 years] (reference) and a cohort of 51,000 male United States health professionals aged 40 to 75 years at baseline in 1986 with five years of follow-up (31)

All of these reported compelling evidence of the increased risks of diabetes associated with high BMI. Compared with subjects with BMIs $<23 \text{ kg/m}^2$, the adjusted relative risk in those with a BMI of at least 35 kg/m^2 was 42.1 (31). Similarly, in the Nurses Health Study, compared to women with a BMI of less than 22, the risks of developing diabetes in those with a BMI of 22, 23, 24, 25-26, 27-28, 29-30, 31-32, 33-34 and 35.0+ were 2.1, 3.5, 2.9, 5.2, 9.6, 19.0, 28.0, 38.5, and 58.2 respectively (25). This evidence clearly points to a very significant increased morbidity risk beyond a BMI of 25 kg/m^2 . Importantly, both analyses by Colditz et al of the Nurses Health Study showed that the increased diabetes risks associated with increasing BMI categories were stable across age strata (24, 25). The NHANES analysis of Lipton et al also showed no interaction of age and BMI in relation to diabetes risk. (reference)

WC is a convenient and valid measure of abdominal fat comprised of subcutaneous and visceral fat. However, in contrast to the rationale for BMI cut-off points, being related to morbidity and mortality outcomes, current recommended waist circumference cut-offs (Table 1) were chosen by deriving the WC levels corresponding to BMI of 25.0 and 30.0 kg/m^2 in two random population samples (34, 35). Lean et al randomly recruited approximately 2000 men and women aged 25-75 from North Glasgow "excluding those chair bound" (35). In this study a waist circumference of $\geq 94 \text{ cm}$ in men and $\geq 80 \text{ cm}$ in women (action level 1) identified individuals with a BMI of at least 25 kg/m^2 or a BMI <25 but a high waist-to-hip ratio (males ≥ 0.95 and females ≥ 0.80) with sensitivity of 96% and specificity of 97.5%. Similarly, a WC of at least 102 cm in men and 88cm in women (action level 2) identified people with a BMI of at least 30.0 kg/m^2 or less than 30 with a high waist-to-hip ratio with sensitivity and specificity of at least 96% and 98% respectively. Han et al used a larger sample ($n=4881$) of albeit younger subjects aged 20-59 year old from the Netherlands (34). The WC action levels applied above in the Lean et al study identified people with BMIs of at least 25.0 (or less than 25.0 with high WHR) and 30.0 kg/m^2 (or less than 30.0 with high WHR) with sensitivities and specificities exceeding 96% (35). This study also determined that these WC action levels were associated with increased risks of demonstrating one or more cardiovascular risk factors. Neither studies determined whether the relationship between WC and BMI was affected by age.

Consequences of overweight and obesity

In the USA, direct costs of obesity have been estimated to be around 9% of the total health care costs and in Europe, between 1% and 5% (36). According to a study of national costs attributed to both overweight (BMI 25–29.9) and obesity (BMI greater than 30), medical expenses accounted for 9.1% of total U.S. medical expenditures in 1998 and may have reached as high as \$78.5 billion (\$92.6 billion in 2002 dollars) (37). It has been estimated that the overall cost of obesity to Australian society and governments was \$58.2 billion in 2008 alone (8).

The health problems and consequences of obesity are many and varied. Many of these are often preventable through a healthy and active lifestyle. There are several large well conducted studies that have shown a clear relationship between excessive body weight and increased mortality and morbidity (38). Mortality and morbidity are also associated with the amount of weight gained in adult life. For example, a weight gain of 10kg or more since young adulthood is associated with increased mortality, coronary heart disease, hypertension, stroke and type 2 diabetes (Table 2) (38). Below, the relationship between obesity and various common age related conditions are discussed.

Functional Impairment and Arthritis

Obesity in older people is associated with increased disability, particularly from lower limb arthritis. In the English Longitudinal Study of Ageing (ELSA) study, both obese men and women had increased relative risks of reporting difficulties with their activities of daily living (men- RR 1.99, 95% CI 1.42-2.78; women – RR 1.66, 95% CI 1.25-2.19) and

having impaired physical function (men RR 1.51, 95% CI 1.05-2.16; women – RR 1.51, 95%CI 1.14-2.00) in comparison to individuals with BMI between 20 and 24.9 kg/m² (reference group) (39). This risk increased between 1.5-2 times in severely obese (BMI \geq 35 kg/m²) individuals. The association of obesity and functional disability is particularly strong in older adults, in whom it is associated with reduced muscle mass and strength and hence with physical frailty (40). A recent review describes that compared to their non obese counterparts, obese older people are less likely to be pain free, have greater limitations of physical function, and are more likely to be homebound (41). Obesity is predictive of a greater rate of future disability, declines in functional status, particularly when associated with loss of skeletal mass, and an increased admission rate to nursing homes (42-44). In the Nurses Health Study, a weight gain of 20 lb (\approx 9 kg) or more over 4 years in women aged 65 years and over was associated with a reduction in reported physical functioning (45).

Obesity and Respiratory and Sleep Disorders

Obesity has a significant impact on respiratory function and it follows that this would impact on overall health. The obesity hypoventilation syndrome (OHS) is a condition where an obese individual with normal lung normal what? Function? chronically hypoventilates (46). At the same time, with increasing body mass index, there is a reduction in forced vital capacity, total lung capacity, residual volume and lung compliance. Respiratory muscle function is also impaired with increasing obesity perhaps due to myopathy or the large pannus imposing on the diaphragm. Most, but

not all patients with OHS have obstructive sleep apnoea and as a result are sleep deprived and lack energy during the daytime.

Obesity and Urinary Incontinence

Cross sectional studies suggest a relationship between increasing BMI and urinary incontinence and the relationship is stronger for stress incontinence than urge incontinence

(47). Prospective cohort studies have also linked being overweight and obese with a subsequent risk of developing new urinary incontinence (47). Weight loss in overweight or obese individuals may not only reduce the risk of developing urinary incontinence but for those suffering from urinary incontinence, weight loss may reduce symptoms(48).

Table 2. Diseases associated with Obesity

Relative risk	Associated with metabolic consequences	Associated with weight
Greatly increased	Type 2 diabetes Gall bladder disease Hypertension Dyslipidaemia Insulin resistance Atherosclerosis	Sleep apnoea Breathlessness Social isolation/depression Daytime sleepiness/fatigue
Moderately increased	Coronary heart disease Stroke Gout/hyperuricaemia	Osteoarthritis Respiratory disease Hernia Psychological problems
Slightly increased	Cancer (breast, endometrial, colon, prostate, uterus) Reproductive abnormalities Impaired infertility Polycystic ovaries Skin complications Cataract	Varicose veins Musculo-skeletal problems Bad back Stress incontinence Oedema/cellulitis

Obesity and Dementia and Depression

Not surprisingly, there is a strong relationship between the presence of mid-life vascular risk factors such as obesity and late life dementia especially for vascular dementia (49). Therefore, achieving ideal weight in youth and middle age is an important strategy to reduce the prevalence of dementia in older age. Interestingly, the association between weight and dementia is less robust and in later life, a low BMI is associated with dementia whilst a

high BMI is not necessarily associated (50). Depressed individuals are more likely to be overweight and obese and this relationship is influenced by factors such as reduced physical and social activity (51). In one study, it was reported that obesity was associated with a 25% increase in odds of mood and anxiety disorders (52)

Emerging Concepts In Relation To Obesity

The Obesity Paradox

It is increasingly reported that obese people with diseases such as hypertension, coronary artery disease, congestive cardiac failure and peripheral arterial disease have better health outcomes than their normal weight counterparts and this is referred to as the obesity paradox (41). The exact mechanism underpinning this observation is uncertain but is likely to be multi-factorial and may include greater metabolic reserve, increased bone mass, hip padding protection by fat, protective effects of certain fat-derived factors such as cytokines and higher blood pressure, reduced activation of the sympathetic nervous system, and lower circulating concentrations of natriuretic peptide (41, 53).

Sarcopenic Obesity

Sarcopenic obesity (SO) combines both the concepts of obesity and sarcopenia in the one individual. In the latest consensus document, it was concluded that sarcopenia refers to both a loss of muscle mass and function (strength and performance) (54). However, past studies of sarcopenic obesity have not accounted for loss of muscle function and therefore, further research is required in this area. Baumgartner et al. has previously defined SO as appendicular skeletal muscle mass/height² less than 2 standard deviation below the sex-specific mean of a younger reference group and percentage of body fat greater than 27% in men and 38% in women (55). With this definition, the prevalence of SO has been reported as 2% in those aged 60-69 years and increasing to 10% in those aged 80 years and over(55). There are other definitions in use. For example, Davison et al. classified

individuals in the upper two quintiles of body fat and in the lower three quintiles of muscle mass as having SO (56). Compared to individuals with just sarcopenia or obesity alone, individuals with SO are said to experience worse health outcomes. For example, subjects with SO at baseline were reported to be two to three times more likely to develop instrumental disability during an eight year follow-up period than lean sarcopenic or non-sarcopenic obese subjects in the New Mexico Aging Process Study (43).

Weight Loss Strategies in The Elderly

Management strategies are discussed below but to summarise, weight loss measures in the elderly should be targeted at patients with a BMI of >30 where the presence of obesity impacts on function and weight reduction is thought to likely benefit the older person. The likely approach is the combination of a hypocaloric diet, aiming at an energy deficit of approximately 500-750kCal/day while providing adequate nutrients, and an exercise program. Dietary measures should be guided by a dietician and behavioural counselling may be required. It is likely that micronutrient supplementation with a multivitamin and attention to Vitamin D and calcium supplementation will be required. Measures to offset lean mass loss include protein intake of 1.2-1.5g/kg/day and exercise. There is no consensus as to the best exercise strategy in the elderly, but a multi-modality program (strength, endurance, balance), guided by an exercise physiologist or physiotherapist, seems appropriate. There is little good quality evidence for LAGB, and this measure should be reserved for patients with a BMI greater than 35-40kg/m² with obesity related co-

morbidities or functional decline who have failed other measures, and who have acceptable operative risk. It is unlikely that surgical treatment will be used in the frail older patient. There is currently no quality evidence to direct the use of very low energy diets or anti-obesity medications in the elderly.

Diet and exercise interventions

As in the younger population, diet and exercise are the cornerstones of obesity management. Much of the research in the area involved an approach combining a hypocaloric diet plus exercise. In some of these studies, in the intervention groups, a combination of hypocaloric diet (kilojoule deficit of 500-750 kCal per day) guided by a dietician, behavioural counselling and multi-modality exercise training (balance, endurance, resistance and flexibility) on 3 days per week was used to achieve a total weight loss of 10% of total body weight, at a rate of no more than 1.5% per week. The results of this approach on various parameters were reported as follows: a) Improvement in insulin sensitivity and fasting glucose (in patients with impaired glucose tolerance)(57); b) Improvement in markers of cardiovascular risk, such as lipids, waist circumference, blood pressure and low-level inflammatory activity (58).; and c) Improvement in V_{O_2} max, physical performance scores and health related quality of life (59).

In The Arthritis, Diet and Activity Promotion (ADAPT) trial (60), older adults (n=252) with knee osteoarthritis were studied for 18 months. The investigators compared diet alone, diet and exercise and exercise alone with a "healthy lifestyle" (control) group. Weight loss was similar in the diet and diet/exercise groups at around 5%. The diet and exercise group

showed the greatest improvement in pain and functional measures – the diet alone group did not differ significantly from the control group in these outcomes.

There are concerns that weight loss may not only accelerate sarcopenia (6) but also contribute to a small (1-2%) decrease in bone mass (61). Therefore, measures to attenuate lean mass loss are important, and the role of protein in achieving this has been studied. One study, enrolling 24 obese postmenopausal women, compared a high protein (1.2-1.5g/kg/day) with a low protein (0.5-0.8g/kg/day) hypocaloric diet (62). It found that the higher protein diet (achieved with the aid of protein supplement drinks) attenuated the loss of lean mass while producing a similar total weight loss to the low protein diet. The other main method of attenuating loss of muscle mass is exercise. All of the trials mentioned here involve multi-modality exercise training, which has been shown to improve general function. There is no consensus about the best exercise strategy in the elderly, however, the best described exercise intervention to offset sarcopenia is progressive resistance training of proximal muscle groups (63). Frailty should not be considered a contra-indication to exercise, indeed, older, more frail, more obese people may show the greatest functional improvements with exercise (64). Exercise physiologists (accessed by referral from a General Practitioner in an Enhanced Primary Care Plan) or physiotherapists can assist with exercise prescription.

Therefore, from these trials, it can be deduced that a hypocaloric diet with high protein content in combination with exercise can not only lead to weight loss but perhaps more

importantly result in improvements in function and disease severity.

Bariatric Surgery

The most common bariatric procedure performed in Australia is Laparoscopic Adjustable Gastric Banding (LAGB), and the procedure is only currently recommended for persons aged 18-65, for whom other weight loss methods have been already been tried (65). LAGB has been shown on meta-analysis to be safer compared with other bariatric procedures such as Laparoscopic Roux en Y Gastric Banding (LRYGB), with similar weight loss efficacy long term (66). A general review of the literature regarding guidelines for bariatric surgery suggested that only the most obese (BMI>40) elderly with severe obesity related complications should be offered bariatric surgery (67-69). More research in the area is needed before LAGB can be recommended for the elderly.

Other Measures: Very Low Calorie Diets and Anti-Obesity Drugs

Very Low Calorie Diets (VLCDs) such as Optifast™ are defined as diets containing energy less than 3.4MJ (800kCal) per day when consumed exclusively (i.e. 3 meals per day). They should also contain 0.8-1.5g/kg/day of high quality protein, sufficient essential fatty acids and micronutrients, and come in the form of shakes or bars. There is currently **no evidence** for their use in the elderly, however, they may be useful in cases where more rapid weight loss is imperative to restore functioning. Close, expert medical monitoring would be essential; medications such as antihypertensives and hypoglycaemics may

need to be decreased or ceased, and changes in warfarin dosage would be common. They are contra-indicated in persons with unstable angina, cardiac failure, severe renal or hepatic dysfunction, malignancies and type 1 diabetes. Potentially serious side effects include cholelithiasis, exacerbation of gout and sequelae of hypovolaemia (70). Anti-obesity drugs can be classified as fat-absorption altering (orlistat), thermogenesis increasing (ephedrine) and anorectic (sibutramine, topiramate, SSRIs such as sertraline or fluoxetine). They have been found to be safe and effective in younger adults in multiple randomised controlled trials. Currently, however, there is **insufficient evidence** to recommend the use of anti-obesity agents in the elderly.

The Future

In terms of weight loss management strategies in the older person, especially those aged 70 and over, larger randomized controlled trials are required to establish methods that allow for weight loss whilst preserving muscle mass loss. Also, to date, the prevalence of obesity in younger and middle-age adults continue to increase and this in turn contributes to increased morbidity in older age. Effective measures including public health strategies are required to ensure healthy nutrition and healthy activity from womb to tomb.

References

1. Prime Minister's Science, Engineering and Innovation Council 2003 (PMSEIC). Promoting healthy ageing in Australia. Canberra.
2. Omran ML, Morley JE. Assessment of protein energy malnutrition in older persons, Part II: Laboratory evaluation. *Nutrition*. 2000; 16: 131-40.
3. Fried LP, Tangen CM, Walston J, et al. Frailty in older adults: evidence for a phenotype. *J Gerontol A Biol Sci Med Sci*. 2001; 56: M146-56.
4. Forbes GB, Reina JC. Adult lean body mass declines with age: some longitudinal observations. *Metabolism*. 1970; 19: 653-63.
5. Chapman IM. Endocrinology of anorexia of ageing. *Best Pract Res Clin Endocrinol Metab*. 2004; 18: 437-52.
6. Newman AB, Lee JS, Visser M, et al. Weight change and the conservation of lean mass in old age: the Health, Aging and Body Composition Study. *Am J Clin Nutr*. 2005; 82: 872-8; quiz 915-6.
7. World Health Organization 2006 - Obesity.
8. Access Economics. The growing cost of obesity in 2008: three years on. Canberra: Diabetes Australia, 2008. Available from: <http://www.accesseconomics.com.au/publicationsreports/showreport.php?id=172>
9. Gill T. Importance of preventing weight gain in adulthood. *Asia Pac J Clin Nutr*. 2002; 11 Suppl 3: S632-6.
10. Australian Institute of Health and Welfare 2004. Australia's health 2004. Canberra: AIHW.
11. Australian Institute of Health and Welfare: Bennett SA, Magnus P & Gibson D 2004. Obesity trends in older Australians. Bulletin no. 12. AIHW cat. no. AUS 42. Canberra: AIHW.
12. Brown WJ, Williams L, Ford JH, et al. Identifying the energy gap: magnitude and determinants of 5-year weight gain in midage women. *Obes Res*. 2005; 13: 1431-41.
13. Department of Health and Ageing. About Overweight and Obesity.
14. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation on obesity. Geneva: World Health Organization, 1997.
15. Heiat A, Vaccarino V, Krumholz HM. An evidence-based assessment of federal guidelines for overweight and obesity as they apply to elderly persons. *Arch Intern Med*. 2001; 161: 1194-203.
16. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *Jama*. 2005; 293: 1861-7.
17. Shah B, Sucher K, Hollenbeck CB. Comparison of Ideal Body Weight Equations and Published Height-Weight Tables With Body Mass Index Tables for Healthy Adults in the United States. *Nutr Clin Pract June* 2006; 21: 312-9.
18. Seltzer C. Limitations of height-weight standards. *NEJM*. 1965; 272: 1132.
19. Kuczmarski R, Flegal K. Criteria for definition of overweight in transition: background and recommendations for the United States. *Am J Clin Nutr*. 2000; 72: 1074-81.
20. Flegal KM, Troiano RP, Ballard-Barbash R. Aim for a Healthy Weight: What Is the Target? *J Nutr*. 2001; 131: 440S-50.
21. Waaler HT. Height, Weight and Mortality - the Norwegian Experience. *Acta Med Scand*. 1984: 1-&.
22. ANDRES R, ELAHI D, TOBIN J, et al. Impact of Age on Weight Goals. *Ann Intern Med*. 1985; 103: 1030-3.
23. Garrow J. *Treat obesity seriously: A Clinical Manual*. . New York: Harcourt Brace/Churchill Livingstone 1981.
24. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight Gain as a Risk Factor for Clinical Diabetes Mellitus in Women. *Ann Intern Med*. 1995; 122: 481-6.
25. Colditz GA, Willett WC, Stampfer MJ, et al. Weight as a Risk Factor for Clinical Diabetes in Women. *Am J Epidemiol*. 1990; 132: 501-13.
26. Hubert HB, Feinleib M, Mcnamara PM, Castelli WP. Obesity as an Independent Risk Factor for Cardiovascular-Disease - a 26-Year Follow-up of Participants in the Framingham Heart-Study. *Circulation*. 1983; 67: 968-77.
27. Lipton RB, Liao YL, Cao GC, et al. Determinants of Incident Non-Insulin-Dependent Diabetes-Mellitus among Blacks and Whites in a National Sample - the Nhanes-I Epidemiologic Follow-up-Study. *Am J Epidemiol*. 1993; 138: 826-39.
28. Hamm P, Shekelle RB, Stamler J. Large Fluctuations in Body-Weight during Young Adulthood and 25-Year Risk of Coronary Death in Men. *Am J Epidemiol*. 1989; 129: 312-8.
29. Lindsted K, Tonstad S, Kuzma JW. Body-Mass Index and Patterns of Mortality among 7th-Day-Adventist Men. *Int J Obesity*. 1991; 15: 397-406.
30. Rabkin SW, Mathewson FAL, Hsu PH. Relation of Body-Weight to Development of Ischemic Heart-Disease in a Cohort of Young North-American Men after a 26-Year

- Observation Period - Manitoba Study. *Am J Cardiol.* 1977; 39: 452-8.
31. Chan JM, Rimm EB, Colditz GA, et al. Obesity, Fat Distribution, and Weight-Gain as Risk-Factors for Clinical Diabetes in Men. *Diabetes Care.* 1994; 17: 961-9.
 32. Gordon T, Doyle JT. Weight and Mortality in Men - the Albany Study. *Int J Epidemiol.* 1988; 17: 77-81.
 33. Higgins M, Kannel W, Garrison R, et al. Hazards of obesity- the Framingham experience. *Acta Med Scand.* 1988; 723: 23-36.
 34. Han TS, Vanleer EM, Seidell JC, Lean MEJ. Waist Circumference Action Levels in the Identification of Cardiovascular Risk-Factors - Prevalence Study in a Random Sample. *Brit Med J.* 1995; 311: 1401-5.
 35. Lean MEJ, Han TS, Morrison CE. Waist Circumference as a Measure for Indicating Need for Weight Management. *Brit Med J.* 1995; 311: 158-61.
 36. National Health and Nutrition Examination Survey 1999-2000.
 37. Finkelstein EA, Fiebelkorn IC, Wang G. National medical spending attributable to overweight and obesity: how much, and who's paying? *Health Aff (Millwood).* 2003; Suppl Web Exclusives: W3-219-26.
 38. Janssen I. Morbidity and mortality risk associated with an overweight BMI in older men and women. *Obesity (Silver Spring).* 2007; 15: 1827-40.
 39. Lang IA, Llewellyn DJ, Alexander K, Melzer D. Obesity, physical function, and mortality in older adults. *J Am Geriatr Soc.* 2008; 56: 1474-8.
 40. Villareal DT, Banks M, Sinacore DR, Klein S. Physical frailty and body composition in obese elderly men and women. *Obes Res.* 2004; 12: 913-20.
 41. Chapman IM. Obesity paradox during aging. *Interdiscip Top Gerontol.* 37: 20-36.
 42. Villareal DT, Apovian CM, Kushner RF, Klein S. Obesity in older adults: technical review and position statement of the American Society for Nutrition and NAASO, The Obesity Society. *Am J Clin Nutr.* 2005; 82: 923-34.
 43. Baumgartner RN, Wayne SJ, Waters DL, et al. Sarcopenic obesity predicts instrumental activities of daily living disability in the elderly. *Obes Res.* 2004; 12: 1995-2004.
 44. Zizza CA, Herring A, Stevens J, Popkin BM. Obesity affects nursing-care facility admission among whites but not blacks. *Obes Res.* 2002; 10: 816-23.
 45. Fine JT, Colditz GA, Coakley EH, et al. A prospective study of weight change and health-related quality of life in women. *JAMA.* 1999; 282: 2136-42.
 46. Powers MA. The obesity hypoventilation syndrome. *Respir Care.* 2008; 53: 1723-30.
 47. Hunskaar S. A systematic review of overweight and obesity as risk factors and targets for clinical intervention for urinary incontinence in women. *Neurourol Urodyn.* 2008; 27: 749-57.
 48. Subak LL, Johnson C, Whitcomb E, et al. Does weight loss improve incontinence in moderately obese women? *Int Urogynecol J Pelvic Floor Dysfunct.* 2002; 13: 40-3.
 49. Richard E, Ligthart SA, Moll van Charante EP, van Gool WA. Vascular risk factors and dementia - towards prevention strategies. *Neth J Med.* 68: 284-90.
 50. Hughes TF, Borenstein AR, Schofield E, et al. Association between late-life body mass index and dementia: The Kame Project. *Neurology.* 2009; 72: 1741-6.
 51. de Wit LM, Fokkema M, van Straten A, et al. Depressive and anxiety disorders and the association with obesity, physical, and social activities. *Depress Anxiety.* 27: 1057-65.
 52. Simon GE, Von Korff M, Saunders K, et al. Association between obesity and psychiatric disorders in the US adult population. *Arch Gen Psychiatry.* 2006; 63: 824-30.
 53. Lavie CJ, Milani RV, Ventura HO. Obesity and cardiovascular disease: risk factor, paradox, and impact of weight loss. *J Am Coll Cardiol.* 2009; 53: 1925-32.
 54. Cruz-Jentoft AJ, Baeyens JP, Bauer JM, et al. Sarcopenia: European consensus on definition and diagnosis: Report of the European Working Group on Sarcopenia in Older People. *Age Ageing.* 39: 412-23.
 55. Baumgartner RN. Body composition in healthy ageing. *Ann N Y Acad Sci.* 2000; 904: 437-48.
 56. Davison KK, Ford ES, Cogswell ME, Dietz WH. Percentage of body fat and body mass index are associated with mobility limitations in people aged 70 and older from NHANES III. *J Am Geriatr Soc.* 2002; 50: 1802-9.
 57. Villareal DT, Banks MR, Patterson BW, et al. Weight loss therapy improves pancreatic endocrine function in obese older adults. *Obesity (Silver Spring).* 2008; 16: 1349-54.
 58. Villareal DT, Miller BV, 3rd, Banks M, et al. Effect of lifestyle intervention on metabolic coronary heart disease risk factors in obese older adults. *Am J Clin Nutr.* 2006; 84: 1317-23.
 59. Villareal DT, Sinacore DM, et al. Effect of weight loss and exercise on frailty in

- obese older adults. *Arch Int Med*. 2004; 166: 860-6.
60. Messier SP, Loeser RF, Miller GD, et al. Exercise and dietary weight loss in overweight and obese older adults with knee osteoarthritis: the Arthritis, Diet, and Activity Promotion Trial. *Arthritis Rheum*. 2004; 50: 1501-10.
61. Villareal DT, Shah K, Banks MR, et al. Effect of weight loss and exercise therapy on bone metabolism and mass in obese older adults: a one-year randomized controlled trial. *J Clin Endocrinol Metab*. 2008; 93: 2181-7.
62. Gordon MM, Bopp MJ, Easter L, et al. Effects of dietary protein on the composition of weight loss in post-menopausal women. *J Nutr Health Aging*. 2008; 12: 505-9.
63. Taaffe D. Sarcopenia: Exercise as a treatment strategy. *Aust Fam Phys*. 2006; 35: 130-33.
64. Sartorio A, Lafortuna CL, Agosti F, et al. Elderly obese women display the greatest improvement in stair climbing performance after a 3-week body mass reduction program. *Int J Obes Relat Metab Disord*. 2004; 28: 1097-104.
65. Victorian Government Department of Human Services. Surgery for Morbid Obesity: Framework for bariatric surgery in Victoria's Public Hospitals. Available at www.health.vic.gov.au. 2009.
66. Chapman AE, Kiroff G, Game P, et al. Laparoscopic adjustable gastric banding in the treatment of obesity: a systematic literature review. *Surgery*. 2004; 135: 326-51.
67. Busetto L, Angrisani L, Basso N, et al. Safety and efficacy of laparoscopic adjustable gastric banding in the elderly. *Obesity (Silver Spring)*. 2008; 16: 334-8.
68. Taylor CJ, Layani L. Laparoscopic adjustable gastric banding in patients > or =60 years old: is it worthwhile? *Obes Surg*. 2006; 16: 1579-83.
69. Yermilov I, McGory ML, Shekelle PW, et al. Appropriateness criteria for bariatric surgery: beyond the NIH guidelines. *Obesity (Silver Spring)*. 2009; 17: 1521-7.
70. Mustajoki P, Pekkarinen T. Very low energy diets in the treatment of obesity. *Obes Rev*. 2001; 2: 61-72.