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Weight loss in obese adults 65 years and older: A review of the controversy

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ABSTRACT

Obesity in older adults is ubiquitous in many developed countries and is related to various negative health outcomes, making it an important public health target for intervention. However, treatment approaches for obesity in older adults remain controversial due to concerns surrounding the difficulty of behavior change with advancing age, exacerbating the age-related loss of skeletal muscle and bone, and the feasibility of long-term weight maintenance and related health consequences. This review serves to systematically examine the evidence regarding weight loss interventions with a focus on obese (body mass index 30 kg/m^2 and above) older adults (aged 65 years and older) and some proposed mechanisms associated with exercise and caloric restriction (lifestyle intervention). Our findings indicate that healthy weight loss in this age group can be achieved through lifestyle interventions of up to a one-year period. Most interventions reviewed reported a loss of lean body mass and bone mineral density with weight loss. Paradoxically muscle quality and physical function improved. Inflammatory molecules and metabolic markers also improved, although the independent and additive effects of exercise and weight loss on these pathways are poorly understood. Using our review inclusion criteria, only one small pilot study investigating long-term weight maintenance and associated health implications was found in the literature. Future research on lifestyle interventions for obese older adults should address the loss of bone and lean body mass, inflammatory mechanisms, and include sufficient follow-up to assess long-term weight maintenance and health outcomes.

1. Introduction

Obesity in older adults is prevalent in many parts of the world and associated with a sequel of poor health outcomes. The prevalence of obesity has markedly increased in the elderly as more baby boomers become senior citizens (Flegal et al., 2010). During the past 30 years, the proportion of obese older adults has doubled, and their prevalence in 2010 was estimated at 37.45% (Patterson et al., 2004). This reflects both an increase in the total number of older persons and in the percentage of the older population that are obese (Villareal et al., 2005). It also represents a significant increase from the 22.2% obese older adults reported in the 1988–1994 National Health and Examination Survey (NHANES) (Federal Interagency Forum on Aging-Related Statistics, 2010). Currently, the per capita spending on obesity-attributable conditions is greater for Medicare recipients than for younger age groups (Finkelstein et al., 2009). No doubt, the growing number of obese older adults in the population will present public health challenges unless actions are taken to reverse this trend.

Losing weight is difficult, and interventions that work in younger adults cannot be assumed to translate to older populations with co-morbidities, low muscle mass and frailty (Villareal et al., 2004). The appropriate treatment approach for obesity remains highly contentious due to the lack of evidenced-based data demonstrating that long-term weight loss is net beneficial or harmful in this age group. There is evidence that successful weight loss is possible in adults 65 years and older (Armamento-Villareal et al., 2012; Frimel et al., 2008; Kelly et al., 2011; Lambert et al., 2008; Shah et al., 2009, 2011; Villareal et al., 2006a, 2006b, 2008, 2011a). However, weight-loss trials have reported losses of lean body mass and bone mineral density, in addition to fat mass (Armamento-Villareal et al., 2012; Bales and Buhr, 2008; Frimel et al., 2008; Kelly et al., 2011; Lambert et al., 2008; Shah et al., 2009, 2011; Villareal et al., 2006a, 2006b, 2008, 2011a). These negative outcomes discourage many geriatricians from advising weight loss to their obese older patients (Heiat et al., 2001; Morley et al., 2010; Rolland et al., 2006; Rossner, 2001; Sorensen, 2003; Villareal et al., 2005; Zamboni et al., 2005), despite improvements in body composition, physical function, metabolic and cardiovascular parameters that accompany weight loss (Anandacoomarasamy et al., 2009; Cheung and Giangregorio, 2012; Ertek and Cicero, 2012; Forsythe et al., 2008). Given these positive functional and metabolic outcomes, it is somewhat surprising that advising weight loss in obese older adults is still shunned in the medical community

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(Houston et al., 2009; Sommers, 2011). Compounding the confusion surrounding risks versus benefits from intentional weight loss is the lack of human studies to elucidate the mechanisms associated with the loss of muscle and bone. Also lacking are trials with adequate follow-up to assess the behaviors associated with long-term maintenance of weight loss and health outcomes related to sustained weight loss.

In order to address these in a systematic review, we posed the research question: "Is there evidence that weight loss is achievable, safe, and maintainable in obese adults aged 65 years and older?" We hypothesized that weight loss would be achievable and safe despite some loss of lean body mass and bone. We also hypothesized that weight loss could be maintained in the long-term. Our primary aim was to systematically review the evidence on weight loss interventions in obese older adults, with a specific focus on changes in body composition, metabolic markers, and physical function, and also mechanisms associated with intentional weight loss through caloric restriction, exercise or both. We applied rigid criteria for defining older adults (≥65 years) and obesity (BMI≥30 kg/m^2) based on the position statement of the American Society of Nutrition and Obesity Society (Villareal et al., 2005), and only included randomized controlled trials that used direct and precise methods for measuring body composition.

2. Literature search methods

A rigorous inclusion criterion as described above was employed. Only randomized controlled trials with a minimum weight loss intervention of three months, and body composition measured by DXA, MRI, CT, or hydrostatic weighing were included. Studies which targeted specific chronic diseases or conditions (e.g. diabetes mellitus, and osteoarthritis), were excluded.

2.1. Data source

An electronic database search was conducted on MEDLINE and PubMed (both clinical and general) for English language articles, with no cutoff dates. Searches were conducted on 20, 23 and 26–27 January 2012, and again on 18 April 2012, 24 May 2012 and 2 July 2012 to capture newly published material. Two broad search areas were categorized: (1) weight loss through caloric restriction, exercise or both; and (2) long-term maintenance of weight loss, feasibility and safety among older adults. In order to cast the widest net for these two areas of interest, five separate overlapping searches were performed, using the keywords: obese, obesity, older adults, elderly, weight loss, body composition, caloric restriction, lifestyle intervention, diet, exercise, function, long-term feasibility, maintenance, and safety.

2.2. Data synthesis

A total of 2309 prospective articles were initially identified. After removing duplicates and irrelevant studies, 90 articles were retained. Of these 90 articles, 83 were excluded for not meeting the inclusion criteria outlined previously. Three articles were manually added. The selection of articles was agreed upon by two authors (DLW and DTV). The final analysis yielded a total of ten articles meeting all established criteria (Fig. 1). These articles are listed in Table 1. They are not ordered chronologically, but instead grouped by similarities between study design and intervention, for ease of discussion. Only one small pilot study was found under the category *feasibility/maintenance of long-term weight loss in older adults* that satisfied our study selection criteria. This study is not included in Table 1, but is discussed under the subheading Feasibility and long-term maintenance of weight loss, in the Discussion of the systematic review section.

3. Discussion of the systematic review

3.1. Randomized controlled trials

Table 1 summarizes the ten trials that met our inclusion criteria (Armamento-Villareal et al., 2012; Frimel et al., 2008; Kelly et al., 2011; Lambert et al., 2008; Shah et al., 2009, 2011; Villareal et al., 2006a, 2006b, 2008, 2011a). Fig. 2 is a schematic representation of the inter-relationships of the mechanisms discussed in these trials.

Three papers by Villareal et al. (two in 2006 and one in 2008) reported on the same cohort of 27 participants. The participants were sedentary (≤2 exercise sessions per week); with stable body weight (±2 kg) during the preceding year; unchanged medication regimes for at least six months; and mild to moderate frailty as measured by the physical performance test (Brown et al., 2000). The intervention consisted of both diet and exercise (lifestyle intervention). Energy deficit was 500–700 kcal/day supplemented with a daily multivitamin and counseling to consume adequate dietary calcium and vitamin D. The goal was 10% weight loss over the six-month intervention and weight maintenance for an additional six months. Exercise sessions consisted of 90 min of aerobic and resistance exercises, three days per week, at a moderate intensity (~75% peak heart rate) and progressed to 80–90% of peak heart rate. Resistance exercise started at 65% of one repetition maximum (1RM) and progressed to ~80% of 1RM.

As designed, body weight and fat mass (FM) decreased significantly in the intervention group. Fat free mass (FFM) decreased in both groups but the difference was not statistically significant. Physical performance test score, peak oxygen consumption, and functional status all significantly improved in the diet and exercise group. Increases in strength were equal to or greater than reported in earlier trials in non-obese older adults completing a similar exercise program (Binder et al., 2002; Villareal et al., 2003, 2004). The investigators stressed that it was not difficult to change the behavior of these older sedentary adults, showing that it was a feasible intervention, which also provided important social interactions that enhanced compliance.

In the second paper, all CVD risk factors significantly improved in the diet and exercise group (Villareal et al., 2006b). Specific mechanisms were not proposed, but the discussion focused on medical care costs related to metabolic coronary heart disease (CHD) risk factors that were ameliorated by the intervention (Table 1). In the third paper (Villareal et al., 2008), bone turnover was measured by type 1 collagen C-terminal telopeptide (CTX), osteocalcin, and bone-specific alkaline phosphatase. There was a marked increase in serum CTX (~100-fold) and osteocalcin (~60-fold) concentrations in response to weight loss indicating that bone resorption and formation, respectively, were stimulated. Moreover, the increases in both CTX and osteocalcin concentrations correlated with decreases in hip bone mineral density (BMD), suggesting that weight-loss induced bone loss was due to increased bone turnover, with greater stimulation of bone resorption than bone formation. However, the clinical significance of the decrease in BMD was not clear as all participants had high baseline BMD Z-scores, and none had evidence of osteoporosis following weight loss. The investigators argued that BMD was not lost in the spine, which implies that the exercises were more effective in preserving BMD at this site. Exact mechanisms for loss of BMD with weight loss are not currently elucidated, but it was suggested that weight loss decreases the mechanical stress on the hip, without negatively impacting the spine or wrist. Weight loss was also associated with a 25% reduction in serum leptin that was highly correlated with decreased hip BMD. No such relationship was found between decreasing estradiol and changes in BMD. Leptin was discussed in the context of its inhibiting action on the expression of receptor activator of nuclear factor $_{\kappa}$B (NF-$_{\kappa}$B) ligand levels (Burguera et al., 2001) and osteoblast differentiation (Cornish et al., 2002). Levels of insulin-like growth factor 1 (IGF-1), cortisol, and parathyroid hormone (PTH) did not change in response to weight loss, which suggests that these bone-active hormones were not involved