

# Obesity Digest

In this regular section, Matt Capehorn picks out recent key papers published in the area of obesity. To compile the digest, a PubMed search was performed for the 3 months up to 15<sup>th</sup> July using a range of search terms relating to obesity. Articles have been chosen on the basis of their potential interest to healthcare professionals and are rated according to readability, applicability to practice and originality.



## Very-low-calorie diets: An under-used tool

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Why do we not use very-low-calorie diets (VLCDs) more? I appreciate that I may be biased, as I am Medical Director to a commercial provider; however, the evidence for their use is clear. VLCDs, also referred to as VLEDs (very-low-energy diets) and sometimes LELDs (low-energy liquid diets), are based on a diet of less than 800 kcal/day, provided by a range of shakes, soups, low-calorie meals or meal replacement bars, that are formulated to be nutritionally complete when adhered to. They very quickly induce a state of ketosis and calorie deficit and, surprisingly to some, a state of satiety that dispels the myth that people on them will always be “starving” (Gibson et al, 2015).

The NICE (2014) CG189 guidelines on obesity include a review on VLCDs and conclude that we should not routinely use these diets to manage obesity, and should only consider them as part of a multicomponent weight management strategy for people who are obese and have a clinically assessed need to rapidly lose weight. However, we need to question whether every obese person, or those with severe and complex obesity, would benefit from rapid weight loss.

A recent randomised controlled trial, published just too late to be included in the NICE review, clearly demonstrated that gradual weight loss appears to be no better than rapid weight loss for long-term weight control (Purcell et al, 2014). Weight regain is inevitable with any weight loss intervention, for numerous social and homeostatic adaptation reasons, and if the rate of weight regain appears to be pretty consistent, then the rate of weight loss may become more important. If we accept that, in an obese individual, any amount of time spent at a lower weight is beneficial, then rapid weight loss will result in a greater area under the weight-over-time

curve, arguably leading to a greater period of time with associated health benefits. A further systematic review of randomised controlled trials published shortly after the NICE deadline identified a larger list of appropriate studies than the CG189 review, and concluded that VLCDs were well tolerated and effective (Parretti, 2014).

In the article summarised alongside, da Luz and colleagues question whether severe dietary energy restriction increases the risk of binge eating in overweight or obese individuals. They conclude that, when clinically supervised, VLCDs appear safe and beneficial in people with pre-treatment binge eating disorder (BED) and do not necessarily trigger binge eating in those without prior BED.

In the NHS Commissioning Board's (2013) *Clinical Commissioning Policy for Complex and Specialised Obesity Surgery*, it is stated that treatment of obesity should be multicomponent, and that there should be access to more intensive treatments such as VLCDs. This couldn't be clearer, yet commissioners do not fund their use. This can no longer be through lack of evidence and can only be due to reluctance on the parts of commissioners to be seen funding a patient's diet, as this may upset certain sectors of society and/or the Taxpayers' Alliance. Yet we live in the midst of an obesity crisis and here we have a proven safe and effective intervention.

At Addenbrooke's Hospital, Cambridge, they provide their own version of a VLCD; a mix of semi-skimmed milk, Oxo cubes (salt), Fybogel and over-the-counter multivitamins. Whether we all go down the route of our own home-brew or partner with the commercial sector, we should no longer allow any barriers that prevent our patients from having access to all available weight loss interventions, especially prior to bariatric surgery. ■

## Obes Rev

### Do LCDs and VLCDs increase the risk of binge eating?

Readability ////

Applicability to practice ////

Originality ////

1. These authors performed a systematic review of the effects of low-energy diets (LCDs) and very-low-energy diets (VLCDs) on subsequent binge eating behaviours in obese participants.
2. Owing to the high diversity of study designs, a meta-analysis was not possible; therefore, a qualitative analysis was performed. Sample sizes in the 10 studies ranged from 38 to 154 individuals, with a maximum follow-up of around 2 years.
3. Of the four studies that evaluated people with no history of pre-treatment binge eating, three demonstrated an increase in binge eating in response to dieting and one showed no change. Increases in binge eating ranged from one to two isolated incidents to 10–15% of participants developing binge eating disorder (BED).
4. Conversely, all five of the studies with analyses of mixed samples (individuals without prior binge eating and those with subclinical binge eating) demonstrated reductions in binge eating. When LCDs were compared with less severe diets, no excess risk of binge eating was observed.
5. The two studies evaluating only people with prior subclinical binge eating had conflicting results.
6. All of the four studies with analyses of people with prior BED showed improvements in eating behaviour; however, some found that relapses occurred after reintroduction of normal food.
7. The authors conclude that LCDs and VLCDs are safe for people with a history of binge eating, although there is some evidence that they may trigger binge eating in people with no prior history of these behaviours.

da Luz FQ, Hay P, Gibson AA et al (2015) Does severe dietary energy restriction increase binge eating in overweight or obese individuals? A systematic review. *Obes Rev* 16: 652–65

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*“The authors conclude that low-energy and very-low-energy diets are safe for people with a history of binge eating, although there is some evidence that they may trigger binge eating in people with no prior history of these behaviours.”*

## Diabetes

### Effect of *FTO* variants on food intake and BMI in children and adolescents

Readability	✓✓✓✓
Applicability to practice	✓✓✓✓
Originality	✓✓✓✓

1. The fat mass and obesity-associated (*FTO*) gene is the greatest single genetic influence on BMI in both adults and children. This large international study group sought to shed light on the mechanisms behind this in an analysis of data pooled from 14 studies.
2. The analysis involved cross-sectional data from 16 094 children and adolescents, the vast majority of whom were of white European descent.
3. The authors observed a significant association between the minor allele (A-allele) of the *FTO* rs9939609 single-nucleotide polymorphism and higher BMI (effect per allele, 0.07 standard deviations).
4. This allele was also associated with higher total energy intake (effect per allele, 14.6 kcal/day), and this association was unaffected by adjustment for BMI. The direction of the association remained the same after controlling for gender, age, physical activity and geographical location.
5. Interestingly, there was a significant association between *FTO* variants and dietary protein intake on BMI, such that lower protein intake weakened the association between the *FTO* variants and BMI. This suggests that *FTO*'s effects on weight and body composition may be mediated through its effects on protein metabolism.
6. The effects of *FTO* on BMI and energy intake were only significant in white children, although, given that there were only 478 black and 267 Asian participants, this may be due to lack of statistical power. Nonetheless, the authors caution that their results may not be generalisable beyond white populations.

Qi Q, Downer MK, Kilpeläinen TO et al (2015) Dietary intake, *FTO* genetic variants, and adiposity: a combined analysis of over 16,000 children and adolescents. *Diabetes* 64: 2467–76

## Cardiovasc Diabetol

### Normal-weight obesity and risk of subclinical atherosclerosis

Readability	✓✓✓
Applicability to practice	✓✓✓✓
Originality	✓✓✓✓

1. People with normal-weight obesity (NWO), who have high levels of body fat despite a normal BMI, have been shown to have an elevated cardiovascular (CV) risk. These authors sought to elucidate this phenomenon by determining whether people with NWO had a higher incidence of subclinical atherosclerosis, a marker of subsequent CV mortality.
2. From a sample of 3546 South Korean people recruited from a self-referred check-up programme, 283 with NWO (defined as the top tertile of body fat percentage despite a BMI of 18.5–25 kg/m<sup>2</sup>) were compared with 1795 who were normal-weight and lean (middle and lowest tertiles of body fat).
3. People with NWO were more likely to be older, women and smokers. After adjustment for these covariates, they were also found to have a higher mean blood pressure (BP), greater levels of visceral and subcutaneous fat and a higher prevalence of poor metabolic parameters.
4. Regarding markers of subclinical atherosclerosis, the NWO group was found to have a significantly higher pulse wave velocity (PWV; 1474 cm/s vs. 1381 cm/s; *P*=0.006) and a higher prevalence of soft plaques in the coronary artery (21.6% vs. 14.5%; *P*=0.04).
5. After adjustment for other atherosclerosis risk factors, visceral fat levels were independently associated with the number of coronary artery segments with soft plaques.
6. The authors conclude that people with NWO have a greater prevalence of subclinical atherosclerosis, largely driven by the presence of soft plaques in the coronary artery, and that the amount of visceral fat is the major determinant of this.

Kim S, Kyung C, Park JS et al (2015) Normal-weight obesity is associated with increased risk of subclinical atherosclerosis. *Cardiovasc Diabetol* 14: 58

## Int J Obes (Lond)

### Caloric compensation and eating in the absence of hunger in preschool children

Readability	✓✓✓
Applicability to practice	✓✓✓✓
Originality	✓✓✓✓

1. These authors evaluated caloric compensation (compensating for a pre-meal snack by eating less at the main meal) and eating in the absence of hunger (EAH) in 3–6-year-old children in France.
2. In the first of three sessions, each held 1 week apart, 236 children had their typical lunchtime energy intake determined. In the second session, caloric compensation was assessed by comparing the number of calories consumed in the control meal with the number consumed in an identical lunch preceded by an energy-dense snack. In the third session, EAH was assessed by determining the amount of calories consumed in the form of treats offered 10 minutes after lunch.
3. On average, the children compensated for only 52% of the energy preload, and they ate 24% of the lunch's caloric value in the absence of hunger, as treats. This suggests that, even at this young age, children's eating patterns are influenced by external cues and not just by internal hunger cues.
4. Interestingly, children who failed to compensate for the snack were not the same as those who ate when not hungry, suggesting that individuals may differ in response to these cues and/or that the responses are processed at different levels.
5. Maternal use of food as a reward was associated with a higher EAH score but also, surprisingly, a greater compensation score. The authors hypothesise that this may be because children who frequently receive food outside meal times may learn to compensate for this better.
6. Child BMI and adiposity were not significantly related to these behaviours; however, only 6% of the sample were overweight.

Remy E, Issanchou S, Chabanet C et al (2015) Impact of adiposity, age, sex and maternal feeding practices on eating in the absence of hunger and caloric compensation in preschool children. *Int J Obes (Lond)* 39: 925–30

## Int J Obes (Lond)

## Eating speed and obesity: Systematic review

Readability	✓✓✓✓
Applicability to practice	✓✓✓✓
Originality	✓✓✓✓

1. These authors report the results of a systematic review and meta-analysis of 23 studies on the association between eating speed and BMI and obesity.
2. Twelve cross-sectional studies, with a total of 87 878 participants, investigated the association between eating speed and BMI, demonstrating a mean increase in BMI of 1.78 kg/m<sup>2</sup> (95% confidence interval [CI], 1.53–2.04 kg/m<sup>2</sup>) in fast eaters compared with slow eaters.
3. Nine cross-sectional studies, with a total of 24 863 participants, evaluated the effect of eating speed on the risk of obesity, demonstrating an odds ratio (OR) of 2.15 (95% CI, 1.84–2.51) in fast eaters.
4. Two longitudinal studies ( $n=966$ ) showed that fast eating resulted in significantly greater weight gain over a follow-up of 7–8 years. A third ( $n=1314$ , all university students) showed that fast eaters were more likely to develop obesity over the 3-year follow-up (OR, 4.40; 95% CI, 2.22–8.75).
5. There was significant heterogeneity in the magnitude of associations across studies. Notably, however, there was little heterogeneity when comparing studies that adjusted for total energy intake, which suggests that mechanisms other than increased food intake may also have a role in this phenomenon.
6. Study limitations include the fact that the majority of studies were in Japanese people; thus, the findings may not apply to other populations. While eating rate was measured by self report in most studies, previous evidence suggests that self-reported eating speed corresponds to both friend-reported and laboratory-measured speeds.

Ohkuma T, Hirakawa Y, Nakamura U et al (2015) Association between eating rate and obesity: a systematic review and meta-analysis. *Int J Obes (Lond)* 25 May [Epub ahead of print]

## J Clin Endocrinol Metab

## LAGB versus medical management for obesity and type 2 diabetes

Readability	✓✓✓✓
Applicability to practice	✓✓✓✓
Originality	✓✓✓✓

1. In this article, the authors compare the 1-year outcomes of laparoscopic adjustable gastric band (LAGB) surgery and a highly structured medical, diet and lifestyle programme in obese people with type 2 diabetes (T2D).
2. Participants were eligible if they had a BMI of 30–45 kg/m<sup>2</sup> and T2D of  $\geq 1$  year's duration. They were randomised to either LAGB ( $n=18$ ) or intensive treatment ( $n=22$ ).
3. The intensive treatment group were enrolled in the Why WAIT (Weight Achievement and Intensive Treatment) programme, which included weekly, 2-hour group educational sessions, weekly medication adjustment, up to 300 minutes of individualised exercise per week and a structured diet providing 1500–1800 kcal/day.
4. After 12 months, mean HbA<sub>1c</sub> reductions were similar in the two groups, with 33% of people in the LAGB group and 23% in the medical group achieving the glycaemic targets of HbA<sub>1c</sub> <48 mmol/mol (6.5%) and fasting plasma glucose <7.0 mmol/L ( $P$ =non-significant).
5. Mean weight loss was similar between the groups at 3 months but was significantly lower in the LAGB group at 12 months (13.5 kg vs. 8.5 kg;  $P=0.03$ ).
6. Changes in lipid profiles, cardiometabolic risk factors and quality of life scores were similar in the two groups. There were four serious adverse events in the surgery group and one in the medical group.
7. The authors conclude that LAGB and the Why WAIT programme have similar 1-year outcomes. They note that the Why WAIT programme is considerably more intensive and structured than previous interventions used in clinical trials.

Ding SA, Simonson DC, Wewalka M et al (2015) Adjustable gastric band surgery or medical management in patients with type 2 diabetes: a randomized clinical trial. *J Clin Endocrinol Metab* 100: 2546–56

## Int J Obes (Lond)

## Four different growth trajectories in early childhood

Readability	✓✓✓✓
Applicability to practice	✓✓✓✓
Originality	✓✓✓✓

1. Recent studies have shown that children and adolescents typically fall into a small number of categories of growth trajectory, typically characterised as never overweight, always overweight, accelerating growth or declining growth. However, to date, no study has assessed growth trajectories in the very early years of life.
2. Therefore, these authors sought to identify patterns of growth trajectories in a cohort of 557 children from South Australia, using BMI z-scores at birth, 6, 9 and 12 months, and at 2 and 3.5 years.
3. Using latent class growth models, the authors identified four distinct growth trajectories: “low” (slow postnatal growth up to 6 months, then standard growth through to 3.5 years; 15% of children), “intermediate” (a more modest deceleration in growth up to 6 months, then normal growth; 46% of children), “high” (BMI z-score consistently around 1 from birth up to 3.5 years; 35% of children) and “accelerating” (birth BMI z-score around 1, a linear increase in z-score until age 2 years, then a plateau up to 3.5 years; 4% of children).
4. Compared with those in the intermediate category, children in the high and accelerating groups had an increased risk of being overweight or obese at age 9 years (odds ratio [OR], 4.3 and 15.4, respectively).
5. Three antenatal factors – male gender, maternal BMI in early pregnancy and multiparity – were associated with increased odds of being in the high or accelerating groups. Maternal obesity had the greatest effect, nearly quadrupling the chance of being in the accelerating group (OR, 3.72).

Giles LC, Whitrow MJ, Davies MJ et al (2015) Growth trajectories in early childhood, their relationship with antenatal and postnatal factors, and development of obesity by age 9 years: results from an Australian birth cohort study. *Int J Obes (Lond)* 39: 1049–56

*“The authors conclude that people with normal-weight obesity have a greater prevalence of subclinical atherosclerosis, largely driven by the presence of soft plaques in the coronary artery, and that the amount of visceral fat is the major determinant of this.”*

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