Dietary Interventions in the Treatment of Paediatric Obesity

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Introduction

Paediatric obesity is a major worldwide health issue. Prevention of obesity in childhood and adolescence is important. However, effective treatments for those already affected are needed to reduce the impacts of obesity on their physical, psychological and social development. Obesity management requires a coordinated

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University of Sydney, Discipline of Paediatrics and Adolescent Health, The Children's Hospital at Westmead Clinical School, Westmead, NSW, Australia e-mail: Sarah.garnett@health.nsw.gov.au model of care to deliver a sustained result. Table 16.1 outlines the key principles of obesity management in children and adolescents. The underlying principle of obesity treatment is to focus on changes in behaviours, including diet and physical activity, which influence body weight and adiposity. In this chapter, we will focus on the role of dietary intervention in the treatment of paediatric obesity.

Efficacy of Dietary Interventions

The aim of dietary intervention in weight management is to contribute to an energy deficit. Numerous randomised controlled trials (RCTs) and meta-analyses have demonstrated that interventions that included a dietary component were efficacious in weight loss at least in the short to medium term in children and adolescents [1-3]. The latest systematic review and meta-analyses by Ho and colleagues [1], which included 38 studies, estimated that the effect size for lifestyle interventions with a dietary component was a decrease in body mass index (BMI) of 1.30 kg/m² [95% CI, 1.03–1.58] and a decrease in total body fat of 3.2% [95% CI, 1.39-5.01] at the end of active intervention (range 3-12 months) compared to usual care or minimal intervention. This review also demonstrated that the weight loss effect was sustained after programme completion and that studies with an intervention period

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- Management of obesity-associated co-morbidities
- · Family involvement
- A developmentally appropriate approach
- · Long-term behaviour modification
- Dietary change
- Increased physical activity
- Decreased sedentary behaviours
- Consideration of the use of pharmacotherapy and other forms of nonconventional therapy

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longer than 6 months had a greater weight loss than shorter-term interventions [1]. Inclusion of an exercise programme did not improve weight loss, but those who received an exercise intervention in addition to a dietary component had a greater increase in lean body mass and a greater reduction in total fat compared to those who received a dietary component alone [4].

Structured lifestyle intervention programmes incorporating a dietary component have also been associated with improved cardio-metabolic outcomes. A meta-analysis of five RCTs including 440 participants aged between 8 and 16 years showed that structured lifestyle interventions incorporating a dietary component resulted in significant improvements in total cholesterol mean difference 0.28 weighed mmol/L (10.81 mg/dL); 95% CI, 0.23-0.34] and triglycerides [0.15 mmol/L (13.29 mg/dL); 95% CI, 0.01–0.24] up to 2 years from baseline, as well as improvements in fasting insulin [55.1pmol/L (7.93 uU/mL); 95% CI, 39.1-71.2] and homeostatic model assessment of insulin resistance (HOMA-IR, 2.32; 95% CI, 1.39-3.25) up to 1 year from baseline [4]. However, the impact of lifestyle interventions on blood pressure is less certain, and long-term data are limited. Importantly, evidence suggests that lifestyle interventions that included a dietary component lead to improvement in cardio-metabolic outcomes in children and adolescents with obesity even in the absence of weight loss or body composition change [1, 2]. Including an exercise component to the intervention can lead to greater improvements in high-density lipoprotein, fasting glucose and fasting insulin levels, but not total cholesterol or triglycerides (Table 16.2) [4]. Nevertheless, partial weight regain and subsequent regression in cardio-metabolic profiles to baseline levels are common in lifestyle intervention programmes at follow-up [4]. Longer-term studies are warranted to examine if the improvements in cardio-metabolic profile that resulted from obesity treatment in childhood will prevent the development of obesity-associated cardiovascular co-morbidities in adulthood.

Dietary Interventions

For many children and adolescents who are overweight, dietary interventions are frequently aimed at weight maintenance that is to slow or prevent weight gain rather than weight loss. However, given the high prevalence of obesity, the increasing prevalence of severe obesity and co-morbidities and the high number of youth who are heavier than their ideal adult body weight, dietary interventions for weight loss are frequently indicated. While the appropriateness of weight loss can be contentious due to proposed physiological and psychological impacts, there is no evidence to suggest that adiposity loss in children and adolescents with obesity causes harm.

The optimal diet for achieving an energy deficit in children and adolescents with obesity is unknown. Intervention trials have examined a number of different dietary strategies with the general agreement that adherence to energy restriction remains the most effective for weight loss [5]. However, achieving long-term adherence to energy restriction is challenging, and it is unlikely that one dietary strategy will fit all. In addition, there is potential for the specific application of certain dietary strategies to lead to greater short-term weight loss and target particular cardio-metabolic risk factors [5]. It is also essential that treatment options are acceptable to both the child/adolescent and their parents/caregivers. Below we explore some conventional and novel dietary approaches.

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| Table 1 |

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|------------------|---|-----------------------------|----------------------|-----------------------|----------------------|----------------------------|----------------------------|----------------------|---|---|-------------------|
| Study | Study arms (follow-up time point) | Sample size at follow-up | BMI change, kg/m² | Body fat change, % | TC change, mmol/L | HDL-C change, mmol/L | LDL-C change, mmol/L | TG change, mmol/L | Fasting glucose change, mmol/L | Fasting insulin change, pmol/L | HOMA-IR change |
| Diet-only (D) | compared with | diet-plus-exerci | se $(D + E)$ inter | ventions | | | | | | | |
| Becque | D (20 wk) | 11 | N/A | -3.5 | -0.24 | 0.10 | N/A | -0.20 | N/A | N/A | N/A |
| 1988 [50] | D + E (20 wk) | 11 | | -3.0 | -0.55 | 0.21*# | | -0.50 | | | |
| Rocchini | D (20 wk) | 15 | N/A | -6.0* | N/A | N/A | N/A | N/A | -0.11 | -34.7* | N/A |
| 1988 [51, 52] | D + E (20 wk) | 18 | | -4.0*# | | | | | -0.11 | -48.6* | |
| Sung 2002 | D (6 wk) | 41 | -0.5 | -0.2 | -0.3* | -0.1^{*} | -0.2 | 0.1 | N/A | N/A | N/A |
| [53] | D + E (6 wk) | 41 | -0.2 | -0.7* | -0.3* | -0.1 | -0.4* | 0.3 | | | |
| Woo 2004, | D (6 wk) | 41 | -0.6* | -0.2 | -0.3* | -0.1^{*} | -0.2 | 0.0 | N/A | N/A | N/A |
| Yu 2004, Yu | D + E (6 wk) | 41 | -0.2 | -0.7* | -0.3* | -0.1 | -0.3* | 0.3 | | | |
| 2005 154 561 | D (1 y) | 41 | -0.2 | -1.3 | -0.2* | 0.2 | -0.4* | 0.2 | | | |
| [0C-+C] | D + E (1 y) | 22 | 0.1 | -4.9* | 0.0 | 0.2* | -0.3* | 0.1 | | | |
| Ribeiro | D (4 mo) | 18 | -3.0* | N/A | -0.18* | 0.00 | -0.03 | -0.32* | -0.28* | -83.34* | -2.0* |
| 2005 [57] | D + E (4 mo) | 21 | -3.0* | | -0.39* | 0.13*# | -0.36 | -0.08*#† | -0.28* | -34.73*#† | -1.4^{*+} |
| Davis 2009 | D (16 wk) | 10 | 0.3 | N/A | N/A | N/A | N/A | N/A | 0.14 | 31.25 | N/A |
| [58] | D + E (ST,16 wk) | 6 | 1.1 | | | | | | -0.20 | -5.56 | |
| | D + E (CAST,16 wk) | 15 | -0.5 | | | | | | -0.24 | -3.47 | |
| Shalitin | D (12 wk) | 55 | -2.06* | -4.48* | -0.31^{*} | -0.07* | -0.11 | -0.28* | 0.02 | 4.44 | 0.17 |
| 2009 [59] | D + E (12 wk) | 55 | -2.02* | -3.48* | -0.28* | -0.02 | -0.19*# | -0.16^{*} | -0.08 | -12.92 | -0.42 |
| | D (1 y) | 55 | 0.36 | -3.63 | -0.42* | -0.02 | -0.29* | -0.22* | 0.06 | 23.13 | 0.74 |
| | D + E (1 y) | 55 | -0.06 | -4.56* | -0.18 | 0.004 | -0.15 | -0.08 | 0.12 | 28.68* | 0.93* |
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| Table 16.2(| (continued) | | | | | | | | | | |
|--|--|---|---|---|--|--|--|---|---|---|-------------------------------|
| Study | Study arms (follow-up time point) | Sample size at follow-up | BMI change, kg/m ² | Body fat change, % | TC change, mmol/L | HDL-C change, mmol/L | LDL-C change, mmol/L | TG change, mmol/L | Fasting glucose change, mmol/L | Fasting insulin change, pmol/L | HOMA-IR change |
| Okely 2010, | D (6 mo) | 42 | -0.8 | N/A | | | | | | | N/A |
| Burrows | D + E (6 mo) | 60 | -0.9 | | | | | | | | |
| 2008, F | D (1 y) | 42 | -0.5 | | | | | | | | |
| 5010 | D + E (1 y) | 60 | -0.2 | | | | | | | | |
| [60-62] | | | | | -0.07 | 0.04 | -0.09 | -0.03 | 0.07 | 0.69 | |
| | | | | | 0.07 | 0.03 | 0.03 | 0.00 | -0.11 | -20.14 | |
| | | | | | -0.03 | -0.01 | -0.01 | -0.02 | 0.11 | -31.25 | |
| | | | | | 0.17 | 0.06 | 0.06 | 0.00 | -0.03 | -23.61 | |
| Diet-only (D |) compared with | exercise-only (E | E) interventions | | | | | | | | |
| Kelishadi | D (6 mo) | 47 | -1.1* | N/A | -0.24* | 0.03 | -0.13 | -0.17* | -0.17 | -8.1 | -0.9 |
| 2008 [63] | E (6 mo) | 45 | -1.04^{*} | | -0.2* | 0.04 | -0.19 | -0.14 | -0.16 | -7.4 | -0.7 |
| | D (1 y) | 45 | 0.7 | | -0.03 | 0.02 | -0.04 | 0.05 | -0.06 | 2.7 | 0.3 |
| | E (1 y) | 42 | 0.5 | | 0.06 | 0.02 | 0.03 | 0.04 | 0.04 | 2.5 | 0.4 |
| Shalitin | D (12 wk) | 55 | -2.06* | -4.48* | -0.31^{*} | -0.07* | -0.11 | -0.28* | 0.02 | 4.44 | 0.17 |
| 2009 [59] | E (12 wk) | 52 | -1.01*# | -1.33* | -0.03 | -0.06 | 0.10# | -0.15* | -0.11 | -10.83 | -0.39 |
| | D (1 y) | 55 | 0.36 | -3.63 | -0.42* | -0.02 | -0.29* | -0.22* | 0.06 | 23.13 | 0.74 |
| | E (1 y) | 52 | 0.42 | -1.16 | -0.16 | -0.04 | -0.16 | 0.06 | -0.09 | 12.92 | 0.36 |
| Okely 2010, | D (12 wk) | 42 | -0.8 | N/A | -0.07 | 0.04 | -0.09 | -0.03 | 0.07 | 0.69 | N/A |
| Burrows | E (12 wk) | 63 | -0.3 | | 0.07 | 0.06 | 0.10 | 0.13 | -0.08 | -14.58 | |
| 2008, P | D (1 y) | 42 | -0.5 | | -0.03 | -0.01 | -0.01 | -0.02 | 0.11 | -31.25 | |
| burrows 2010 [60–62] | E (1 y) | 63 | 0.4 | | 0.18 | -0.02 | 0.20 | 0.28 | 0.04 | -16.67 | |
| <i>BMI</i> , body main of insulin resiand strengther | ass index; <i>HDL-C</i> stance; <i>D</i> , dietary n training; *, post | C, high-density l y intervention; <i>E</i> t-treatment sign | lipoprotein cholo 3, exercise interv ificantly differed | esterol; <i>LDL-C</i> vention; <i>wk</i> , we nt from baselin | ', low-density sek(s); y, year le $(p < 0.05)$; | lipoprotein cho ; <i>mo</i> , month(s) #, post-treatme | olesterol; <i>TGs</i> , ; <i>N/A</i> , not appl ent significantl | triglycerides; icable; ST, stre y different | <i>HOMA-IR</i> , ho some trainin | meostasis mod g; CAST, com | el assessmen bined aerobio |
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Conventional Approaches

Currently, the most frequently reported dietary interventions for weight management in dietary intervention trials are healthy eating advice, which is low in fat, based on dietary guidelines or the Traffic Light/Stop Light diet [1, 4, 6].

Healthy Eating Advice Based on Dietary Guidelines

Food-based dietary guidelines provide advice on foods and food groups to provide the required nutrients to promote overall health and prevent chronic diseases. In many countries, including Australia, the USA and the UK, current guidelines recommend a diet that is high in carbohydrate (45–65% of daily energy) and low in fat (less than 35% of daily energy), with protein contributing approximately 15% of daily energy. This diet is often supported by food guides, in the form of food pyramids and food plates (e.g. Fig. 16.1), which are used for consumer education. It is recommended that these guidelines be adopted at a population level [7, 8], including for the treatment of child and adolescent obesity.

A diet based on the dietary guidelines may also be energy restricted. Determining the appropriate energy targets for weight loss can be challenging, needs to be assessed on an individual basis and will depend on a number of factors including age, sex, physical activity levels, co-morbidities and the speed of weight loss required. Dietary intervention trials involving an energy-restricted diet are often based on either a standardised daily kilocalorie (kcal) deficit of 300-500 kcal, 30% less than the reported energy intake or 15% less than the estimated energy requirement [1]. Some studies impose energy restrictions on snacks and beverages only. However, if energy restriction is indicated, a diet based on dietary guidelines can present challenges in achieving micronutrient adequacy, particularly essential fatty acids, and careful selection of foods is required [9]. This diet is generally successful in achieving weight loss in the short term in children and adolescents [1]. In adults, a 2015 systematic review and meta-analysis suggested that these diets are equally as effective as other dietary interventions, including low carbohydrate and higher fat, of similar intensity in the long term [10].



Fig. 16.1 Choose MyPlate. MyPlate was developed by the United States Department of Agriculture to assist individuals create healthier eating styles (Source: https://

www.choosemyplate.gov. US Department of Agriculture. Public Domain)

Traffic Light/Stop Light

The Traffic Light/Stop Light diet is an energycontrolled approach. Foods in each category are colour coded according to their caloric densities per average serving: green for low-calorie foods that can be eaten freely, yellow for moderate-calorie foods that can be eaten occasionally and red for high-calorie foods that should be eaten rarely. This diet was one of the original diets to treat childhood obesity as well as used in intervention trials [6]. Similar to a diet based on dietary guidelines, this diet is successful in achieving modest weight loss in the short term in children and adolescents [1].

Novel Approaches

While there is evidence to support conventional dietary interventions short term, their effectiveness in sustaining long-term changes remains uncertain. Hence, there has been recent interest in exploring different dietary strategies that optimise weight loss as well as improve cardiometabolic outcomes. These strategies include modifying the macronutrient content and/or the quality of carbohydrate, as well as novel dietary approaches to restrict energy intake.

Increased-Protein Diet

An increased-protein diet typically aims for approximately 26–44% of daily energy as carbohydrate, <35% as fat and 20–40% as protein [11]. These diets are hypothesised to lead to greater weight loss by evoking (a) sustained satiety despite reduced energy intake, (b) sustained energy expenditure despite loss in body mass by sparing loss of fat-free mass and (c) increased dietary-induced thermogenesis, because the thermal effect of protein is greater than that of carbohydrate or fat [12]. In children and adolescents, there have been seven studies, ranging in duration from 1 to 12 months, comparing isocaloric increased-protein (mean $24.4 \pm 3.9\%$ of energy) and standard-protein (mean $16.3 \pm 2.1\%$ of energy) diets [5, 13]. The studies included boys and girls, aged 7-18 years, with a mean sample size of 110 participants [5]. All studies found improved weight, fasting glucose, fasting insulin, insulin sensitivity, blood lipids and blood pressure irrespective of diet group and irrespective of whether participants were freeliving or in highly controlled environments (boarding school or camp). Figure 16.2d shows the 12-month change in glycaemic status and body composition measures from one of the included RCTs undertaken by Garnett and colleagues [14]. This study specifically targeted adolescents with insulin resistance and/or prediabetes and randomised 111 participants to a hypocaloric diet, which was either increased protein (25-30% of energy) or standard protein (15% protein). The authors reported no significant differences between diet groups at any time point. The authors concluded, as did the systematic review and a later similar study by Truby et al. [13], that the protein content of the diet appears to have little effect when given isocalorically. The results emphasise the importance of total energy intake for improved weight status in children and adolescents with obesity.

In adults, results from several trials have indicated that increased-protein diets increased fat loss, attenuated reductions in fat-free mass and improved an array of cardio-metabolic factors, including glucose homeostasis and the blood lipid profile compared to standard-protein diets [15]. However, the effects are not consistently reported. A recent systematic review that compared energy-restricted, isocaloric, high-protein (mean $30.5 \pm 2.4\%$ of energy) with a standardprotein (mean $17.5 \pm 1.5\%$ of energy) diet concluded that the high-protein diet provided modest benefits for reductions in body weight (0.79 kg; 95% CI, 1.50-0.08 kg), fat mass (0.87 kg; 95% CI, 1.26-0.48 kg) and triglycerides [0.23 mmol/L (8.9 mg/dL); 95% CI, 0.33-0.12 mmol/L], with better preservation of lean body mass. Changes in fasting plasma glucose, fasting insulin, blood pressure, total cholesterol and lowand high-density cholesterol were similar across dietary treatments [15].





Fig. 16.2 (**a**–**d**) Glycaemic status and body composition measures by dietary group over the 12-month intervention. Estimated marginal means (SE) are presented from linear mixed models for the increased-protein diet group (*downward triangle*) and the high-carbohydrate diet group (*upward triangle*). Differences between diet groups were not significant at any time period. (**a**) Insulin sensitivity index. ¹Significance between baseline and 3 months and 12 months as indicated. ²Significance between 3 and 12 months. (**b**) Total body fat percent (Fat % DXA). ¹Significance between baseline and 3 months and 12 months as indicated. ²Significance between 3 and 12 months (**c**) Fat-free mass index. ¹Significance between

It is not clear why the protein effect is not seen in children and adolescents. Achieving dietary protein targets in studies of free-living children and adolescents is difficult, and in part the lack of difference may be due to the reported lower protein intake in youth [5, 16]. In addition, some will contend that RCTs are not the ideal study design to determine the effect of dietary intervention. The isocaloric nature of experimental diets may blunt the satiating effect of protein thought to contribute to a lower ad libitum total energy

baseline and 3 months and 12 months as indicated. ²Significance between 3 and 12 months. (**d**) BMI%95th centile. ¹Significance between baseline and 3 months, 6 months and 12 months as indicated. ³Significance between 3 and 6 months.⁴Significance between 6 and 12 months (Source: Garnett SP, Gow ML, Ho M, Baur LA, Noakes M, Woodhead HJ, Broderick CR, Chisholm K, Briody J, De S, et al. Improved insulin sensitivity and body composition, irrespective of macronutrient intake, after a 12 month intervention in adolescents with prediabetes; RESIST a randomised control trial. BMC Pediatrics. 2014;14:289)

intake and consequent improved weight loss as reported in some adult studies [12, 17].

Very Low-Carbohydrate Diet

A popular alternative to the low-fat diet is a very low-carbohydrate diet, typically aiming for <50 g carbohydrate per day with high or ad libitum fat and/or protein intake (e.g. the Atkins diet) (Table 16.3). A recent systematic review

| Carbohydrate diet classification | Amount of carbohydrate | Example of dietary pattern |
|--|---|---|
| Typical/high-carbohydrate diets | 45- > 65% of total calories | Low-fat diet, Traffic Light/Stop Light diet, standard-protein diet, lower-GI diet |
| Moderately restricted carbohydrate diets | 26-44% of total calories | Intermittent fasting diet, increased- protein diet |
| Low-carbohydrate diets | 51–130 g/day (or approximately 16–26% of calories of a 2000 calorie diet) | Low-carbohydrate diet |
| Very low-carbohydrate diets | Typically 20–50 g/day or 5–15% of total calories | Very low-carbohydrate diet, very low-energy diet |

 Table 16.3
 Classification of diets based on carbohydrate content

Source: Gow ML, Garnett SP, Baur LA, and Lister NB. The Effectiveness of Different Diet Strategies to Reduce Type 2 Diabetes Risk in Youth. Nutrients. 2016;8(8):486

suggested that a very low-carbohydrate diet may result in greater weight loss (a mean decrease in BMI of 1.46; 95% CI, 0.44–2.48 and a mean decrease in BMI z-score 0.25; 95% CI, 0.06–0.44) immediately following active treatment (10–26 weeks) compared with a lowfat diet [5]. However, the difference between diet groups was not maintained at the 2-year follow-up, nor was the difference observed in the larger, higher methodological quality studies [18, 19]. The current evidence suggests that a very low-carbohydrate diet may lead to greater short-term weight loss and may be useful when indicated, for example, in severe obesity prior to surgery.

In terms of cardio-metabolic outcomes, very low-carbohydrate diets reportedly improve insulin levels and/or insulin resistance compared with a low-fat diet immediately following active treatment [18, 20] and at follow-up [5]. These findings suggest that a very lowcarbohydrate diet as part of obesity treatment may facilitate improvements in hyperinsulinemia compared with a traditional low-fat approach in children and adolescents. Furthermore, a very low-carbohydrate diet may also facilitate improved body composition [21] and/or triglycerides but may increase LDL cholesterol levels [18, 22]. In adults with type 2 diabetes, a very low-carbohydrate diet may improve glucose status and lipid profile when compared to a high-carbohydrate diet (>200 g/ day). To date, there is insufficient evidence for

use of very low-carbohydrate diets in children and adolescents with diabetes [23].

There have been no reported adverse effects on cardio-metabolic profile in association with following a very low-carbohydrate diet in children and adolescents, suggesting the short-term safety of the diet. The long-term safety of very low-carbohydrate diets in children and adolescents is unknown. One concern is that restricting carbohydrate in the diet, without a sufficient increase in vegetable consumption, reduces the intake of nutrients obtained from high-quality carbohydrates, particularly fibre and phytochemicals [24]. An increased feeling of fatigue has also been a reported side effect of following a very low-carbohydrate diet in adults [25]. This could result in a reduced desire to complete physical activity in youth with overweight or obesity and should be considered in an individual who is, or plans to be, very active as part of their weight loss regimen.

Low Glycaemic Index/Glycaemic Load

A diet that has a lower glycaemic index (GI) generally refers to a balanced diet that incorporates carbohydrate foods that have reduced glycaemic load (GL), i.e. foods/meals that produce a slower rise in blood glucose levels and have lower overall carbohydrate content [26]. However, consumption of lower-GI foods does not necessarily translate to a "healthy" diet,

with some discretionary foods such as ice cream, cakes and potato crisps having a low GI. In children and adolescents, a 2015 systematic review that included nine RCTs with a duration between 10 and 96 weeks and a total of 1065 children and adolescents found that lower-GI/GL diets produced greater improvements in insulin resistance (HOMA-IR mean difference 0.70; 95% CI, 0.04–1.37) and triglyceride levels (mean difference 0.17 mmol/L; 95% CI, 0.05–0.30) compared to higher-GI/GL diets [27]. However, there was no beneficial effect for weight loss or cholesterol levels (total, HDL or LDL). Similar findings have been reported in young adults with obesity [28].

The DiOGenes study is the largest study conducted to date which examined the effect of varying the GI and protein content of the diet on weight and cardio-metabolic outcomes in children, recruiting families from eight European countries. Eligible parents were randomised as a family unit to one of five ad libitum diets: lowprotein and low-GI, low-protein and high-GI, high-protein and low-GI, high-protein and high-GI and control diet (national dietary guidelines, medium protein content and no instructions on GI) [29]. A difference of 15 GI units between the higher-GI and lower-GI diets was targeted. The results of this study showed that neither GI nor protein had an isolated effect on body composition among children following an ad libitum diet. However, the low-protein, high-GI combination increased body fat, whereas the highprotein, low-GI combination was protective against obesity [30]. It is important to recognise that the children in the DiOGenes study were not necessarily overweight; they were the children of parents who were overweight or obese. The children were not given advice on weight loss but were educated on the diet's ability to regulate appetite [30].

Very Low-Energy Diet

A very low-energy diet (VLED) is a dietary approach that has gained popularity due to its association with rapid weight loss. It is a very strict diet aiming for <800 kcal/day. VLEDs are largely protein based and contain essential fatty acids, vitamins and minerals but very little carbohydrates (typically <50 g) and are aimed at inducing ketosis [31]. They reduce portion size and, consequently, energy intake. Because a VLED is difficult to follow, it is usually implemented short term, aiming for rapid weight loss, and comprised of meal replacement products (e.g. shakes, bars, soups, desserts) to achieve nutritional adequacy.

Studies in adolescents with obesity have demonstrated that a VLED can safely induce rapid weight loss in the short term (4–15 kg over 3–12 weeks) while preserving lean body mass [21, 32-34]. Studies have also demonstrated improvements in blood pressure, total cholesterol, LDL and HDL cholesterol, triglycerides, fasting glucose, fasting insulin, HbA1c and insulin sensitivity [21, 32, 33]. One of these studies found that a short-term (10 weeks) daily VLED (600-800 kcal/day) compared with a hypocaloric low-fat diet produced significantly greater reductions in percentage body fat while maintaining lean body mass [21]. In another study comparing a VLED with a hypocaloric low-fat diet, weight loss was greater in the VLED group at 4 months, but this was not sustained at 12 months [32]. A recent pilot study investigated the effects of an 8-week VLED in adolescents with type 2 diabetes [34]. Rapid weight loss was achieved, and 6 months after the VLED intervention, four of the five participants who adhered to the diet had reversal of type 2 diabetes. These results highlight the potential benefit of following a VLED beyond weight loss [34].

Overall VLEDs are tolerated by adolescents and result in rapid weight loss, improvements in body composition and improved metabolic risk profile short term, but long-term outcomes are not clear. In adults the metabolic benefits of bariatric surgery have been reproduced by VLED [35]. The diet, although strict, may be an alternative to pharmacological therapies or surgical interventions to treat adolescents with severe obesity. VLEDs require intensive monitoring by a team of health professionals.

Intermittent Fasting

Intermittent fasting is also known as "intermittent energy restriction" and "alternative day fasting". This diet has been popularised as the 5:2 diet and has gained much media interest and celebrity endorsement. Intermittent fasting includes 1-4 "fasting" (or VLED) days per week, where energy intake is drastically limited (typically less than 600 kcal), and 3-6 "feeding" days per week, where food is either consumed ad libitum or a diet based on healthy eating guidelines. It is speculated that intermittent fasting comprising of shorter periods of energy restriction coupled with longer periods of habitual energy intake may be more sustainable and promote better adherence than continuous daily energy restriction [36]. To date, studies examining the effectiveness of intermittent fasting have not been conducted in children or adolescents.

In 2016 there were two systematic reviews of adult trials comparing intermittent fasting with daily energy restrictions [37, 38]. While there were conflicting results from individual studies on which group achieved the greatest weight loss, overall both reviews indicated that intermittent fasting is as equally effective as daily energy restriction in the short term (5 weeks to 6 months) and long term (12-18 months) to help individuals with obesity decrease weight (4-8%) and body fat and reduce cardio-metabolic risk [37, 38]. Measures of compliance were limited, but one study indicated that short-term compliance for the intermittent fasting group was effective but not in the long term [39]. A greater number of adverse effects were also experienced in the intermittent fasting group which included headache, lack of energy and difficulty fitting the diet into their daily routine. Trials are ongoing in children and adolescents.

Potential Risks of Dietary Interventions

Disordered Eating and/or Eating Disorders

A concern about using dietary energy restrictions in obesity treatment is the potential risk of developing or exacerbating disordered eating and/or eating disorders. Overweight children are more likely to have dysfunctional eating behaviours, including emotional eating and restrained eating, compared to normal-weight children [40, 41], and overweight adolescents are more likely to engage in disordered eating, such as binge eating [42]. The RESIST study examined the impact of using a prescriptive hypocaloric meal plan (500 kcal less than the recommended daily energy intake) on the psychological dimensions of eating behaviours in over 100 adolescents with obesity [43]. The results of the RESIST study showed that a prescriptive dietary approach led to significant reductions in dysfunctional eating behaviours, particularly external and emotional eating, and the intervention did not elicit any adverse effects on dietary restraint in adolescents with obesity. Evidence from an earlier systematic review also supports the view that professionally administered paediatric weight loss interventions do not increase the risk of eating disorders and may, indeed, improve psychological wellbeing in adolescents with obesity [44]. Furthermore, there is evidence that adolescents seeking weight management have a preference for prescriptive dietary advice, as opposed to unstructured advice [43, 45]. In adults, a recent systematic review of the safety of severe dietary energy restriction (430 to 1200 kcal/day) in overweight and obese adults reported that clinically supervised programmes do not necessarily trigger binge eating in overweight individuals without pre-existing binge eating disorder [46]. On the contrary, severe dietary energy restriction does not exacerbate and may even reduce binge eating in overweight individuals with

pre-existing subclinical binge eating or binge eating disorder. These findings indicate that dietary interventions are generally safe when they are run by professional teams. In view of the association between obesity and disordered eating, it is recommended that eating behaviour should be monitored during, and preferably also after, obesity treatment.

Nutritional Adequacy of Reduced Energy Diets

Children and adolescents have unique and differing nutrition requirements, including calcium, iron and zinc, and energy-restricted diets reduce the opportunity to meet micronutrient requirements by limiting overall food intake. There is a paucity of data on the effect of energy restriction on nutritional adequacy. There is one recent paper which has examined the nutritional adequacy of three energy-restricted diets which are utilised in clinical practice for adolescents with obesity: a diet based on dietary guidelines, a modified-carbohydrate diet and an intermittent fasting diet [9]. In this paper the authors undertook dietary modelling and demonstrated that these eating patterns can be adapted to achieve nutritional adequacy and energy restriction; however, they did need careful consideration to meet nutritional requirements of adolescents.

Reduced Resting Metabolic Rate and Lean Body Mass

Concerns have been expressed that dietary restrictions used in obesity treatment in children and adolescents may adversely decrease resting metabolic rate and lean body mass [47]. Results from a systematic review which included five RCTs comparing diet-only interventions with a diet-plus-exercise interventions reported that an energy-restricted diet (900 to 1400 kcal/day) along with a moderate protein content (20-30%) did not result in a loss of lean body mass in 6- to 18-year-olds over 4 months [4]. Loss of lean body mass was reported in one small study (n = 38) in which children with obesity (8-12 years old) were prescribed an 1800 kcal high-carbohydrate low-fat diet (65% total energy from carbohydrate,15% from protein and 20% from fat) and participated in supervised exercise training three times/week (60 min/session) [48]. Participants lost 1.3 kg [95% CI, -2.01-0.59] in lean body mass over 4 months. Overall these findings suggest that for lifestyle interventions with an exercise component, diets with an increased protein content may protect against the loss of lean body mass.

Conclusion

The current evidence indicates that an improvement in weight status can be achieved in children and adolescents with obesity, irrespective of the dietary macronutrient profile or dietary pattern, provided the diet is energy reduced. This suggests that the primary objective of dietary interventions should be to reduce total energy intake. However, there is some inconsistent evidence from child and adolescent obesity treatment intervention studies to suggest that very low-carbohydrate diets, VLEDs and lower-GI diets may achieve greater weight loss compared with a more traditional low-fat diet, at least in the short term. These diets also appear to have advantages over a low-fat diet for improving cardiometabolic risk profile. A common feature of these diets is that they ultimately reduce glycaemic load by modifying diet carbohydrate quality or quantity compared with the traditional low-fat approach. These dietary options give clinicians multiple diet strategies to offer children and adolescents, which may assist in achieving greater weight loss compared with a low-fat approach. Hence, diets may be personalised depending on patient preference and suitability.

Editor's Comments and Question

Social media and mainline newspapers are awash with testimonials "demonstrating" that obesity and its complications are caused by discrete macronutrients such as sugar, fructose or saturated fat. However, as discussed in Chap. 1, it is the *patterns* of dietary intake, rather than the intake of single macronutrients, that are the principal determinants of childhood weight gain. Diets that are high in energy density, fat and sugar and low in fibre, fruits and vegetables are associated with higher percent body fat and excess adiposity in childhood and adolescence. Total caloric intake, as you note, is critically important.

It is not surprising, then, that reductions in any *single* macronutrient have had little or no effect on body mass index in obese children^{a,b,c} and that long-term weight loss in obese adults is determined by total energy intake, rather than the specific macronutrient content of the diet^{d,e,f}. Moreover, the effect of any *single* macronutrient on metabolic risk (e.g. type 2 diabetes mellitus) pales in comparison to the effect of obesity itself^g.

Common-sense approaches to diet can reduce weight in obese children and prevent weight gain in children at risk: in my experience, nearly anyone can lose weight by eliminating sugar drinks and reducing intake of fried and fast foods and highdensity starches. The trick is actually doing it.

Where do you place your focus in promoting weight loss in obese children?

Authors' Response

The focus of our approach to weight loss in children is dependent upon a number of factors including the age of the child, the amount of weight loss required and the presence or absence of co-morbidities.

For the younger child, the focus of treatment is the family, and generally we aim at simple changes as you have indicated above. We encourage drinking water and limit the intake of fruit juice, cordial and soft drink. We encourage the use of low-fat dairy food types for children over the age of 2 years instead of full cream and encourage the consumption of fruits and vegetables. We also consider it important to eat breakfast and to sit down and enjoy meals together as a family with the television switched off.

In our adolescent clinic, where many of the adolescents present greater than their ideal adult weight with several co-morbidities, we aim for weight loss. We offer a number of the weight loss strategies as discussed in this chapter. Diets are personalised depending on patient preference and suitability. Adolescents are allowed to change strategy; we are acutely aware that if there is no weight loss in the first 3 months of treatment, a different strategy is indicated^h.

Our primary focus for adolescents with obesity and type 2 diabetes is rapid weight loss. We recognise the severity of the condition including increased morbidity and mortality. The traditional approach has been the management of blood sugar levels, but results from our recent study have indicated that it is possible to reverse the pathology of type 2 diabetes with a very low-energy diet in adolescents as seen in adult studiesⁱ. We see this as a first-line treatment option in newly diagnosed youth.

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