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Obesity treatment: An overview with a dietary perspective

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ABSTRACT

We are facing a rapid increase in the prevalence of obesity that is mainly related to behavioural changes in modern society. Life style changes and above all dietary changes are thus essential components in all obesity treatment programmes and in the present article an outline is given of different dietary treatment strategies. The role played by specific macronutrients, especially fat and carbohydrates, is also discussed. Many studies have been undertaken to evaluate the effects of different therapeutic approaches. In spite of this, knowledge about the design and implementation of dietary treatment programmes is rather limited, mainly due to methodological shortcomings. However, some features seem common to successful interventions. A multicomponent strategy including a low energy diet, increased physical activity and behaviour therapy seems more efficient than treatment programmes using just one or two of these modalities. Furthermore, obesity is a chronic disorder and long-term treatment improves weight loss and maintenance.

Key words: Dietary treatment, GI, obesity, pharmacological treatment, very-low-calorie-diets, weight loss

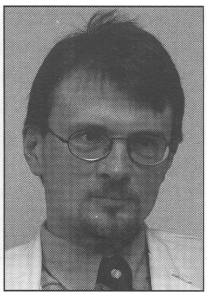
Introduction

The prevalence of obesity (BMI≥30.0 kg/m^2) is rapidly increasing world-wide and WHO describes the situation as an ongoing epidemic (1). The picture is similar in the Nordic countries (2-4). In Sweden for instance the obesity prevalence was 6.6% among men and 8.8% among women in 1980/81, while the corresponding figures in 1996/97 were 10.0% and 11.9%, respectively (2).

Obesity can be defined as an excessive accumulation of adipose tissue to the extent that health may be impaired. A positive energy balance (energy intake > energy expenditure) is a prerequisite for the development of obesity. Ultimately, body weight is the result of interactions between genetic and environmental factors, acting through various physiological mediators of energy intake and expenditure. Although the genetic component of weight control is important, the escalating obesity prevalence is obviously much more related to behavioural changes in modern high-tech society than to genetic changes in the population (5). Reduced physical activity and/or increased energy intake (e.g. availability of highly palatable foods) seem to contribute to the present situation (6).

Adipose tissue constitutes the largest energy depot of the body and since the storage capacity for carbohydrates and protein is limited and *de-novo* lipogenesis is an energetically expensive process energy balance is to a large extent equal to fat balance (7). A positive association between the increasing obesity prevalence and increased energy intake has been difficult to find. One reason for this could be the notorious problem of accurately reporting food intake in epidemiological surveys (5).

Obesity is a chronic disorder and there is thus a need for long-term intervention strategies. With the exception of surgery, it has however proven difficult to achieve large and long-standing weight losses. Intuitively, diet and physical activity are cornerstones in all obesity treatment programmes and in the following sections an overview is given of non-surgical options for treating adult obesity. Specific dietary components will be accounted for in more detail. Pharmacological treatment will mainly be discussed with reference to the concomitant dietary intervention.



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Dietary management

Life style change, resulting in a negative energy balance, is fundamental in all obesity treatment programmes. Theoretically, a negative energy balance can be achieved by reducing energy intake, increasing physical activity or both. Without a conscious restriction of food intake weight loss is usually small (8). The most important part of obesity treatment is thus to reduce the total energy intake. In earlier studies hypocaloric diets of 5.0-6.3 MJ/d were usually prescribed to all individuals regardless of their actual energy requirements. Presently, it is more common to estimate the individual 24-hour energy expenditure and to subtract 2.1-4.2 MJ from that value. An energy reduction of this size will result in a weight loss of 0.5-1 kg per week (9,10). The calculated energy level serves as a theoretical background in dietary counselling, where emphasis for practical reasons has to be put on food choices and behaviour rather than on calories *per se*.

The energy restricted diets should be healthy and in line with the general recommendations for the entire population (11). It is also important that dietary changes are long-term rather than temporary restrictions of specific foods (9). Since the intake of total fat and saturated fat is high in the Nordic countries (12), emphasis should be given on total fat intake and fat quality. However, in order to reduce the total energy intake, carbohydrates and protein need to be restricted as well. Dietary recommendations should also aim at a higher intake of fruits and vegetables and a shift to more whole grain cereals (11). Clinical experience suggests that many obese patients have irregular eating patterns and a more planned meal structure may help controlling energy intake.

Education is important in dietary treatment, since patients need to know what a healthy low energy diet consists of, how to

Table 1. Some elements in behaviour treatment programmes. Adapted from (1,9,10).

Self-monitoring of eating and exercise behaviour To objectify behaviour through observation and recording is a key step in behaviour therapy. Food intake and the circumstances in which it occurs are recorded in detail. Similarly, the frequency, intensity and type of physical activity are recorded.
Setting behavioural goals Specific goals are set for changes in eating and activity patterns over the treatment period.
Stimulus control Limiting exposure to cues that prompt overeating e.g. to avoid buying highly palatable foods.
Cognitive restructuring A method to identify and modify dysfunctional thoughts and attitudes about weight regulation
Relapse prevention A continuing process designed to promote maintenance of treatment induced weight loss.

choose food items, how to judge nutrient content from food labels and how to cook (10). However, there are many reasons why people, in spite of knowledge, do not change to a healthier diet. Commonly perceived barriers are: not wanting to give up certain foods, finding time, prices, preferences of others, eating out and unappealing foods (13). Dietary programmes should therefore include techniques for behavioural change. Common elements in behaviour modification strategies are outlined in Table 1 (1,9,10). No single behavioural method, or combination of methods, has proven clearly superior and various strategies can be used (10). In the literature it is sometimes difficult to draw a distinct line between dietary treatment and behaviour modification, since behavioural change is an important part of most dietary treatment programmes and most structured behaviour modification programmes include education on nutrition and physical activity. Furthermore, behaviour modification is often an ill-defined component of many treatment programmes (14).

Group therapy is commonly used but little is known about the effectiveness of group versus individual therapy (9). It has been suggested that group therapy is more cost effective than individual treatment (15), but this has not been evaluated in relation to weight loss or drop-out rates. It is not known whether some individuals benefit more from group therapy than others. Neither is the optimal group size nor the frequency or length of appointments known (15).

Results of dietary treatment

Weight loss in dietary treatment programmes is in the order of 5-10% (16). After six months weight loss usually declines and a plateau is reached (10). Figure 1 can serve as an illustration of weight reduction during two years of dietary intervention. In this randomised study, one group of obese patients received a very low calorie diet for 12 weeks followed by dietary treatment up to two years (VLCD group), whereas the other group received the same dietary treatment programme alone for two years (non VLCD group) (17). Based on results from studies with dietary treatment it has been suggested that an individual weight loss goal of 10% is realistic, since the observed average of 8% also includes subjects who do not lose any weight at all (10). This weight loss may seem modest, but it has been suggested that it is better to maintain a moderate weight loss over a long period of time than to rapidly regain weight after a more pronounced initial weight loss (10). Furthermore a number of studies have shown that a moderate weight loss (less than 10% of the initial weight) improves both glycemic control and lipid profile and reduces blood pressure (1).

Obtaining the weight loss goal has often been considered as the end of treatment. Unfortunately, there is a gradual return to pre-treatment body weights over time (18). Since obesity is a chronic disease it is important that monitoring and encouragement continue on a long-term basis (10). However, studies with long-term follow-up are rare. Ayyad and Andersen have recently published a review of studies with a follow-up time longer than three years. Based on their criteria for success (maintenance of all weight initially lost or maintenance of at least 9 to 11 kg of the initial weight loss), 15% of the study patients could be defined as successful. Active follow-up was generally associated with a greater weight loss than a more passive approach (14).

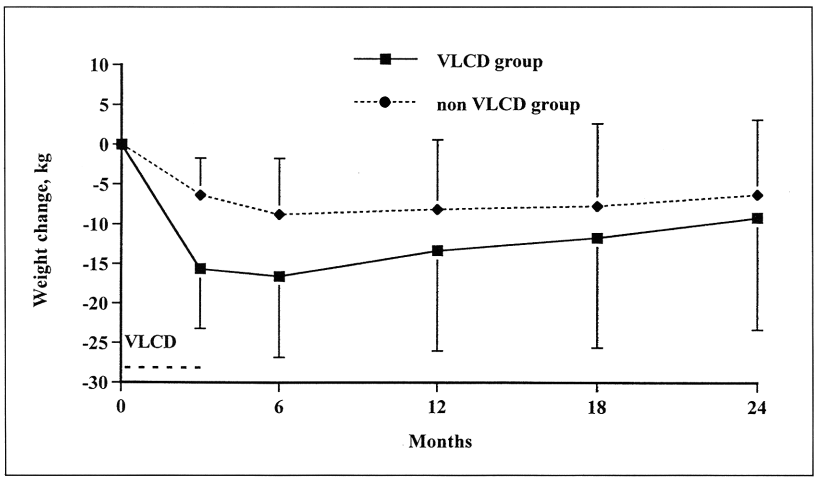

Figure 1. Weight changes during 24 months of active treatment. Mean values and one standard deviation are shown. Weight losses after 24 months were 9.2 ± 14.2 kg (7.4 ± 11.3%) in the VLCD group and 6.3 ± 9.4 kg (5.4 ± 7.9%) in the non VLCD group. The VLCD group received a very low calorie diet (VLCD) for 12 weeks followed by hypocaloric diet and behavioural support, while the non VLCD group received the diet and support programme alone. (From Torgerson JS et al: Int J Obes 1997;21:987-94, with permission).

Dietary fat

The role of fat intake in obesity has been intensely discussed (19-21). There are several reasons why high fat diets may promote obesity. Compared to carbohydrates fat is more readily stored as an energy reserve in the adipose tissue (22). Short-term studies have suggested that fat is less satiating than carbohydrates and protein joule for joule (23). Furthermore, high fat diets are usually more energy dense than low fat diets due to the higher energy content per gram of fat compared to carbohydrates and protein. High fat energy dense foods can therefore result in passive overconsumption (24).

Intervention studies with the primary aim to reduce dietary fat but with otherwise unrestricted total energy intake usually show weight loss. A recent meta-analysis suggests that a reduction in the energy intake derived from fat is associated with a weight loss that is about 3 kg larger than among control subjects (25). The weight loss observed in many low fat interventions has lead to the suggestion that concentrating on reducing fat intake would result in a reduced total energy intake. Consequently, obese subjects given such instructions would also lose weight, especially since low fat diets would allow for larger portion sizes of non-fat foods. In spite of this, randomised trials primarily aiming at weight reduction have not shown larger weight loss on low fat diets compared to energy restricted diets (26,27). In a study by Shah et al the low fat diet produced a weight loss of 4.4 kg and the low energy diet a weight loss of 3.8 kg after six months (26). Jeffery et al obtained weight losses that after six months were 4.6 kg on a low fat diet and 3.7 on a low energy diet. After 12 months, weight regain was similar in both groups (27). However, the palatability of the fat restricted diet was rated higher in both studies (26,27).

Low fat diets with *ad libitum* intake of carbohydrates and protein may be more effective in maintaining weight loss. Toubro and Astrup assigned patients having lost on average 13 kg to a one-year maintenance programme of either ad libitum low fat high carbohydrate diet or a fixed energy diet (28). Two years after initiation of the maintenance programme the *ad libitum* low fat high carbohydrate diet group had regained significantly less weight than the fixed energy diet group (5.4 vs. 11.3 kg).

A crucial point when discussing the role of low fat diets in obesity treatment is dietary adherence. It is not known whether it is easier to follow a low fat diet long-term than to continuously restrict energy intake. Adherence to low fat diets may depend on the level of fat restriction. Fat restriction differs between studies and when fat contributes to approximately 30% of the total energy intake it is doubtful whether the diet still can be viewed as a low fat considering the fat recommendations for the general population (11). Differences in adherence may also depend on the dietary education given as well the cultural context. Doubts have also been raised about very-low-fat-diets, since they may increase triglycerides and reduce HDL-cholesterol (29). Furthermore, many processed low fat products on the market may be energy dense and thus not helpful when trying to lose weight (10,29).

Dietary carbohydrates

When reducing fat intake it is important to know what type of carbohydrates that are consumed. If carbohydrates are specified in study reports they are often described only as simple or complex. Furthermore, educational emphasis seems to be given to sources of fat, fat quality, low fat cooking etcetera, while the counselling on carbohydrates is usually less clearly stated.

The metabolic response to starchy foods may differ according to a number of factors such as particle size, cellular structure, properties of the starch and processing steps (30). The glycemic index (GI) indicates the effect of carbohydrate-containing foods on blood glucose levels (31). The GI is usually defined as the area under the glucose response curve during a 2-hour period after consumption of 50 g carbohydrates from a test food and values are expressed relative to the effect of a standard (usually white bread or glucose), (30). It is possible to construct diets with similar amounts of energy from macronutrients as well as similar fiber and simple carbohydrate content but with different GI (32). Diets with a low GI have been shown to improve lipid profile and normalise fibrinolytic activity in subjects with type 2 diabetes (32). The glycemic response of foods may also influence hunger and energy regulation. Short-term studies indicate that consumption of high GI carbohydrates may increase hunger and overeating (33). Among obese male teenagers, a low GI meal was associated with a lower ad libitum intake 5-10 hours after a test meal than after meals with higher GI (34). The long-term effect of low GI foods on energy regulation is not clear. A 12-week cross-over study on 16 obese hyperinsulinemic women showed larger weight losses after a low GI energy restricted diet than after a high GI energy restricted diet (7.4 vs. 4.5 kg) (35).

Protein and alcohol

Little is known about the role of protein intake in relation to weight loss and weight maintenance. In one Danish study obese subjects were randomly assigned either to a low fat high carbohydrate diet, a low fat high protein diet or to a control group instructed not to change diet. Subjects in the two intervention groups selected their food from a shop at the research unit. After six months subjects in the low fat high protein group had lost more weight than the low fat high carbohydrate group (8.9 vs. 5.1 kg), (36). However, protein intake in the industrialised world is already high and there is some uncertainty concerning possible adverse effects of high protein diets e.g. renal damage and reduction of bone density (29).

Alcohol is at the top of the oxidation hierarchy. It suppresses oxidation of other macronutrients, especially fat, and it is not stored (37). Alcohol provides more energy per gram than carbohydrates and protein and is thus more energy dense. Epidemiological studies have suggested a positive association between alcohol intake and BMI among men, while the association among women is negative in many studies (9).

Meal patterns

A negative association between meal frequency and body weight has been observed in some epidemiological studies (38-41), while other investigators have found no such association (42,43). Methodological problems and lack of standardised definitions of meals and snacks have complicated interpretation of the results.

Using a method where subjects describe times and frequencies of consumption of main meals, light meals, snacks and drinks across an ordinary day it has been suggested that obese women eat significantly more meals per day than non-obese women (44). The greatest difference was seen in the number of snacks and the reported intake of snacks was also positively associated with energy intake (Bertéus Forslund H, *personal communication 2001*). This is in agreement with a French study on obese women where snackers had a higher energy intake than non-snackers (45). Since snack meals seem to be associated with total energy intake it is doubtful whether the current Swedish recommendation to eat two to three snack meals per day is optimal for obese subjects wanting to lose weight (11).

Physical activity

Weight loss is modest if energy expenditure is increased without dietary restriction (8,10). A meta-analysis by Garrow and Summerbell has shown that aerobic exercise alone results in a weight loss of 3 kg in men and 1.4 kg in women as compared to

sedentary controls (8). However, physical activity has numerous other beneficial effects regardless of BMI. Subjects who engage in moderate or vigorous exercise at least once a week are less likely to have type 2 diabetes, cardiovascular disease, hip fractures or mental illness and have lower mortality rates than those who are less active (46).

Based on a number of observational studies it has been suggested that subjects that are physically active do not gain/regain weight to the same extent as people who are not physically active (47). Fogelholm and Kukkunen-Harjula have recently published a systematic review of physical activity and weight maintenance and they conclude that a high physical activity level of approximately 6.3-8.4 MJ/week is associated with improved weight maintenance (47). However, the actual effect of recommended exercise programmes on weight maintenance may be small (47, 48). This is partly explained by low adherence to prescribed exercise programmes. Increasing physical activity enough to maintain body weight may in the long run be difficult for subjects who live in a sedentary environment.

Very Low Calorie Diets

A very low calorie diet (VLCD) is defined as a diet of less than 3.3 MJ/d. VLCDs are relatively enriched in protein, have a low carbohydrate content and fulfil recommended daily allowances for vitamins and minerals. They are mainly provided as powder in measured portions and mixed with water before serving.

VLCDs are indicated in obese patients (BMI≥30.0 kg/m^2) and in subjects with substantial overweight (BMI>27 kg/m^2) associated with disorders or risk factors that could be ameliorated by rapid weight loss e.g. diabetes type 2 (49,50). Preoperative VLCD treatment of obese patients can also be used in order to reduce the operative risk (51). Contraindications for VLCD treatment vary in the literature and the most relevant are summarised in Table 2 (49,52,53). Diabetes type 2 is not a contraindication to VLCD treatment but patients must be well informed so that adjustments of oral hypoglycemic agents and/or insulin doses can be rapidly undertaken.

A VLCD is usually given for 8-16 weeks and followed by a refeeding phase when a hypocaloric diet is gradually introduced. The low energy content results in an initial depletion of glycogen stores. Since glycogen has a large water-binding capacity and since the rapid decrease in insulin levels results in renal losses of sodium and water body weight is reduced in the order of 2-5 kg during the first week (54). Once glycogen stores are depleted a ketosis develops that might result in a beneficial anorexia and analysis of ketones in urine can be used to assess compliance during VLCD treatment (50). A VLCD for 8-16 weeks results in weight losses in the order of 20-25 kg and this is significantly greater than what can be achieved with hypocaloric diets (10, 50).

There is an increased risk of gallstone formation during VLCD treatment and the incidence of clinical cholelithiasis has been reported to be 4-5% (55). Patients with gout face a slightly elevated risk of acute exacerbations, since uric acid levels are transiently increased (56, 57). Among minor side effects are fatigue, dizziness, postural hypotension, constipation, dry skin, hair loss and cold intolerance (49). Approximately 25% of the weight loss during VLCD treatment is lean tissue, which is similar to what is observed during treatment with low calorie diets (3.3-6.3 MJ/d) (49,58).

The long-term weight loss with VLCDs is not better than with low calorie diets (10). However, a recent review of Swedish trials of VLCD combined with comprehensive support programmes indicate that weight loss after one to two years of treatment is approximately 8-10% (50), which is also exemplified in Figure 1 (17). Weight loss of this magnitude is similar to current results with pharmacological treatment. There is an ongoing discussion about the safety and nutritional adequacy of VLCD. There are data indicating that weight loss is similar irrespective of how much the energy intake is reduced below 3.8 MJ/d (59,60). It might increase safety even further to use diets with energy levels above 3.3 MJ/d, especially if patients are treated without adequate medical supervision.

Table 2. Contraindications to VLCD treatment. Adapted from (49,52,53).

- BMI≤25.0 kg/m^2
- Pregnancy and lactation
- Diabetes type 1
- Severe catabolic disease
- Severe renal or hepatic disease
- Unstable angina pectoris or recent myocardial infarction
- Malignant cardiac arrhythmias or prolonged QT-interval
- Recent stroke or transient ischemic attack
- Severe psychiatric disorder including bulimia nervosa
- Drug abuse
- Children and adolescents

Pharmacological treatment

In clinical trials of new anti-obesity drugs dietary intervention is basic treatment for all patients, while the study drug, according to randomisation, is used as add on therapy for some subjects. The weight change achieved in the non-placebo group thus represents a combination of dietary and pharmacological effects. However, the ambition (e.g. energy deficit) and intensity (e.g. number of visits) of the dietary interventions vary, which complicates direct comparison of studies.

Orlistat inhibits gastrointestinal lipase activity and reduces the uptake of dietary fat with about 30%. This promotes a negative energy balance and weight loss (61). Since fecal fat is increased there is a risk of gastrointestinal side effects if dietary fat is not reduced. Patients treated with orlistat are thus recommended a diet with 30% of the energy as fat to reduce this potential side effect. Data from placebo-controlled clinical trials show that gastrointestinal side effects are more common among patients treated with orlistat. They are generally of mild to moderate intensity and occur early in treatment (62-66).

Orlistat 120 mg three times a day combined with hypocaloric diet (approximately minus 2.5 MJ/d), reduces body weight about 10% after one year of treatment, which is significantly better than the 6% achieved with diet alone. During a second treatment year all patients were prescribed an eucaloric diet and the corresponding weight reductions after two years were about 8% and 4%, respectively (62-64). Half of the final weight loss in the orlistat group could thus be attributed to the dietary intervention. The reason for changing from hypocaloric to eucaloric diet was to examine the effect of orlistat also on weight maintenance. In the clinical setting however, patients still obese after one year of treatment would not have been recommended to increase the daily energy intake and it might be that the combined effect of orlistat and diet became less optimal due to the study design.

A two-year study with orlistat in a primary health care setting was designed as above, with hypocaloric diet year one followed by eucaloric diet year two. However, physicians not trained in nutrition or weight management techniques gave all dietary prescriptions and patient visits were also less frequent. Weight reduction after one year was 7.9% in the orlistat group and 4.2% among patients that received hypocaloric diet. After two years the corresponding weight losses were 5.0% and 1.7%, respectively (65). As shown also in other primary health care trials, weight loss becomes smaller than in academic centres (66).

Sibutramine is a reuptake inhibitor of serotonin and norepinephrine that has weight reducing properties by increasing both satiety and energy expenditure (67). In a recently published two-year study all patients were prescribed a hypocaloric diet (minus 2.5 MJ/d) with less than 30% of the energy intake from fat, together with sibutramine 10 mg/d, for six months. Patients that lost more than 5% in weight were then randomised to sibutramine or placebo, plus the hypocaloric diet, for further 18 months. Unfortunately, weight changes were only reported in kg but since the average baseline weight was 102 kg a 1-kg weight change is close to 1%. Weight loss after six months was about 12 kg. Patients that subsequently received the hypocaloric diet regained weight and ended up with a two-year weight loss of about 4.5 kg. The corresponding weigh loss in the sibutramine group was 10 kg (68). As with orlistat, half of the final weight reduction could thus be attributed to the diet.

VLCD followed by pharmacotherapy is theoretically interesting, since it offers a possibility to increase both weight loss and maintenance. This was illustrated in a study where VLCD for one month was followed by 12 months on hypocaloric diet plus sibutramine 10 mg/d or placebo. The average weight loss during the VLCD phase was about 7.5 kg. Subjects in the placebo group regained 0.5 kg the following year, while patients on sibutramine lost still another 5 kg (69).

Some concluding remarks

Extensive reviews focusing on obesity treatment have been published recently (10,14,70). It is striking that knowledge about the design and implementation of treatment programmes is rather limited, in spite of the large number of studies performed. One reason is that too many trials do not fulfil acceptable methodological criteria with respect to randomisation, sample size and length of treatment (14,70). However, some features seem common to successful interventions. A multicomponent strategy combining a low energy diet, increased physical activity and behaviour therapy seems more efficient than programmes using just one or two of these modalities (10,70). Furthermore, frequent patient visits and continued long-term contacts improve weight loss and maintenance (10,14,70). However, the relative efficacy of different visit frequencies to physicians, dieticians or nurses is not known.

Pharmacotherapy becomes sub-optimal when the concomitant dietary counselling is too rudimentary or infrequent (65,66,71). This is not an argument against drug treatment of obesity. Instead it emphasises the need for comprehensive treatment programmes. An initial VLCD period followed by pharmacological intervention to counteract weight regain and/or induce further weight loss seems to be a promising multimodality treatment approach deserving further attention.

The optimal contribution to the total energy intake from different macronutrients is not known. A low fat diet may be helpful in reducing weight and maintaining an achieved weight loss (1,9). It also seems that some individuals will benefit from a low fat high carbohydrate diet. Furthermore, both animal data and epidemiological studies have suggested a varying susceptibility to high fat diets (72,73). If low fat diets are recommended attention should also be paid to the carbohydrates. It is important that fat is not simply replaced by sweet foods with a high energy density. Furthermore, the type of complex carbohydrates in a diet needs to be specified in more detail. The metabolic response to foods with high or low glycemic index (GI) differs and it is possible that foods with different GI effect appetite and energy regulation differently. Further research regarding possible different long-term effects of foods with different GI is warranted.

Obese individuals wanting to lose weight are confronted with strong counteracting biological, psycho-social and environmental forces. The health care system or the affected individuals can not manage the epidemic on their own and a joint action from governments, food industry, international agencies, media and consumer organisations is necessary to modify modern environment so that it becomes less obesity-promoting (1).

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