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Treatment of child obesity

Presented at the 8th International
Workshop of the European Childhood
Obesity Group, 8–10 May 1998,
Wisła-Jawornik, Poland

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Abstract Prevention of obesity should have the highest priority and be started as early in life as possible in high-risk families. This review covers the most promising areas of today's research aimed at finding better ways of treating obesity in the future and an overview of the treatment choices available at present. The cause of obesity is genetic in approximately 40–50% of adults and probably a larger proportion of children. This means that it is possible to remain overweight without a higher calorie intake than that of individuals of normal weight who lack the genetic susceptibility to obesity. Treatment is recommended from 10 years of age with a slightly hypocaloric diet (1500 kcal) and a reduced fat content (30 energy-percent). Exercise is often provided naturally by the child before this age. It is helpful to

try to replace aspects of our sedentary life-style, including televisioning, with moderate physical activities, such as walking. Family therapy has been effective in introducing these changes in life-style. Other strategies, such as behavioral therapy, have also been tried, individually, in groups, or at school. Cognitive therapy has no advantage over behavioral therapy in childhood obesity. No reports on psychodynamic therapy have been published. Surgery is used only in the case of rare inborn metabolic diseases. Thus, further development of obesity therapy is needed, although the treatment of children seems to give better results than treating adults.

Key words Childhood obesity · Treatment · Family therapy · Review

Introduction

The health risk experienced in adult life after childhood obesity has been studied in two long-term follow-ups. Excessive overweight in puberty was associated with higher morbidity and mortality than would have been expected by chance in adult life both in a study conducted in the United States of America, with 55 years of follow-up [85], and in a Swedish study, after 40 years of follow-up of 504 overweight children [84]. In the latter study the obese children developed chronic diseases, such as cardiovascular diseases (precisely twice as frequent as in the reference population), hypertension (1.7 times as fre-

quent), diabetes (3.2 times), locomotor diseases (1.5 times) and digestive diseases (3.8 times as frequent) in later life. The differences were significant for cardiovascular and digestive diseases. Furthermore, the mortality was significantly increased. The most common cause of death was cardiovascular disease. Prevention of obesity should thus have the highest priority and be started as early in life as possible in high-risk families. Childhood obesity is accordingly of major interest. The prevalence of obesity in children differs between countries. It is estimated at 28% in 6- to 11-year-old children and 22% in 12- to 18-year-old children in the United States [48], 16% in boys and 11% in all children aged 4–12 years in Italy [75], and 5% among 4th grade children and 5.2% in boys and

3.5% in girls in the 8th grade in Sweden [39]. The differences may be due to a combination of genetic and environmental factors.

In the following pages I will review the most promising areas of current research aimed at finding better ways of treating obesity in the future. I will also give an overview of the treatment choices available at present. For the diagnosis of obesity in a population-based screening situation, body mass index (BMI) i.e., the body weight in kilograms divided by the height squared expressed in meters, is a convenient measurement. Normal standards for Britain have recently been developed by Cole's group [23, 42]. The younger the child the higher the spontaneous remission rate. This has been used as an argument for not intervening until adolescence. However, the risk of persistence of the obesity beyond the 7th year may be directly proportional to the degree of its severity [13]. Thus, severe obesity in younger children, with the possible exception of the 1st year of life, should be dealt with. In the British BMI chart treatment is recommended for all children above the 99.6th percentile.

The pathophysiology of obesity

Genetics

A distinction between "necessary" genes and "susceptibility" genes has been proposed [51]. A necessary gene is one that causes the disease as a single trait when the gene is dominant or as a double trait when the gene is recessive. A susceptible gene is one that increases susceptibility to or risk of a disease but is not necessary for disease expression. Obesity is one example where heredity is expected to lower the threshold for the development of the disease [11]. The trend seems to be towards a major recessive gene explaining 20–25% of the inheritance (heritability) depending on age plus a multifactorial transmission. It can be estimated that about 5% of the population may be carriers (homozygotes) of the putative recessive gene [12]. The total effect of the genes has been shown to be at least 34%, and probably as much as 40% [11, 102].

It is therefore necessary to view obesity as a genetic and environmental disease. This relieves the patient of the guilt of having caused the disease (as it is genetic) but also excludes the possibility of modifying the inheritance (by using environmental factors such as diet and exercise if one is unfortunate enough to have the genes).

The comparison with allergic diseases such as asthma may be useful. If you have asthma (are obese), it is helpful to eliminate what is provoking your asthma (overweight) i.e., fats (calories). In the case of asthma there are many drugs available, and hopefully in the future that will also be the case for obesity.

The discovery of leptin and the mutation of the β_3 -adrenergic receptor are major achievements made possi-

ble by the intensive genetic research currently being carried out. These discoveries are described below. They emphasize still more the points made above regarding obesity as a disease comparable to other diseases. Leptin (from the Greek leptos meaning thin) is a protein produced by the adipose tissue and controlled by the obese (*ob*) gene found in humans and mice [112]. It has been shown that injection of leptin into mice that have a deficient *ob* gene induces weight reduction [21, 56, 89]. However, in obese humans the leptin level is high instead of low, indicating a different pathophysiology from that in mice [24]. Furthermore, no defective *ob* gene has been found in humans except for some rare exceptions. Nevertheless, leptin is of major interest, as future studies of its effects might give us more clues to a better understanding of the development of obesity. It has been suggested that the leptin receptor might also be associated with obesity, but a relationship has only been found in Pima Indians [31, 41, 50, 58, 101]. Leptin is thought to be a signal for long-acting satiety, i.e., maintained inhibition of further eating after consumption (satiation or inter-meal satiety [9], comparable to the signals that stop feeding behavior (satiety or intra-meal satiety).

Enterostatin, described below, is regarded as a factor leading to early satiety. Enterostatin, a small protein released during the digestion of food, inhibits fat intake [38]. It activates norepinephrine and serotonin. This is of interest as dexfenfluramine, a compound known to release serotonin, has been found to regulate fat intake. However, no significant effects on single-meal eating behavior have been found when humans have been treated with enterostatin [92].

β_3 -Adrenergic receptor

The sympathetic nervous system is thought to activate the β -adrenergic receptors, influencing energy expenditure via catecholamines that mobilize energy-rich lipids by stimulating lipolysis in fat cells in white adipose tissue and thermogenesis in brown adipose tissue and skeletal muscle. The β_3 -adrenergic receptor is thought to regulate the heat production by influencing the brown adipose tissue surrounding vital internal organs [2]. Brown adipose tissue is scattered around the great vessels in the abdomen and the thorax. It has large numbers of mitochondria containing a so-called uncoupling protein that makes heat production and burning of excessive fat possible. The body has a need to react to mild stimulation, such as cold or a meal (thermic effect of food). White adipose tissue serves to store fat, and the breakdown (lipolysis) of fat is mediated by the β_3 -adrenergic receptors. Recently, a mutation of the β_3 -adrenergic receptor gene has been found to be associated with an increased capacity to gain weight, for instance [22, 106, 108]. Clinical treatment studies are now being performed, but only about 10% of the popula-

tion of obese adults studied have proved to have the mutation, indicating that there is a significant number of cases in which it cannot explain adult obesity [108]. In most studies there is no relationship with obesity, and especially when children are investigated, indicating that there might be a subpopulation of adults in whom the β_3 -adrenergic receptor is of importance [43–45, 60, 61, 63, 67, 83, 86, 96, 110]. This might be so when visceral adiposity is concerned, and the conflicting results might be due to different genetics in the different study populations. To sum up, we now know some of the mechanisms regulating food intake but do not yet know the real cause of obesity.

Energy intake

There is a major controversy as to whether obese people eat less than or the same amount as people of normal weight or whether they eat more. This controversy may be solved in the future when we are able to distinguish those who are obese because of their susceptibility genes from those who are obese because they eat too much and lack genetic susceptibility. Of course if we eat vast amounts of food *and* have genetic vulnerability, we become grossly obese, but it is likely that those who eat less than people of normal weight remain moderately obese if they have obesity genes. On the other hand, if these genes are lacking, eating vast amounts of food may produce moderate but not gross obesity. Thus, if we are lean it is certain that we lack obesity genes, and if we are grossly obese, we do have them. If we are moderately obese or of normal weight it is not possible to know whether or not we have the genes. It is therefore not yet possible to know who is at risk of developing severe obesity. I have the clinical impression that most obese Swedish children eat less than their peers during childhood. This might be so, as only 5% of Swedish children are obese, which is the lowest frequency of obesity expected to have a genetic cause (see “Genetics” above). Moreover, it has been demonstrated that they eat faster and do not slow down their eating rate towards the end of a single meal [6]. Adult men eat more than women, normal weight men by eating for a longer time and obese men by eating faster. However, there is no difference between obese and normal-weight adults in the amount ingested [7]. Many studies show that obese people eat less, and usually this is explained by indicating that obese people under-report their eating behavior [32]. However, metabolic adjustments occur to return body fat to the baseline level if energy intake is controlled to hold fat stores constantly above or below the baseline level [70]. Thus, it is difficult to deviate from a set-point for body fat and body weight. This might explain why an obese individual can eat small amounts of food and still remain obese.

Energy expenditure

Few prospective studies have been performed in subjects enrolled before obesity has appeared. In one study children newly born to lean or overweight mothers were studied soon after birth and their total energy expenditure was measured at 3 months using the double-labeled water method. This method makes measurement under free living conditions possible. Weight gain during the 1st year of life was studied. The total energy expenditure at 3 months of age was 21% lower in the infants who became overweight than in the other infants. The reduced energy expenditure was attributed mainly to reduced physical activity [91].

Psychosocial aspects of society

Although there is a major genetic influence in obesity [99, 102], psychosocial factors have also been identified. However, conclusions must be drawn with caution. There might be differences between the general population and obese subpopulations, as shown in a study of self-esteem, locus of control and other psychosocial factors [93]. In this nonclinical population, using prospective data no relation was found between body mass index and adolescent self-esteem and locus of control. In a representative sample of people aged 16–24 years examined in the US in 1981 and followed up 7 years later, the overweight women had fewer years of school and lower incomes, while both men and women who were overweight were less likely than controls to be married. Individuals with other chronic conditions, such as asthma, did not differ from controls. Thus, overweight during adolescence was shown to have important social and economic consequences [49]. Moreover, a Swedish study has shown that the degree of psychosocial stress in a cohort of 971 children followed from birth to 15 years of age was greater in children with rapid weight gain during school years than in controls [76, 77]. However, Danish young men measured at the draft boards since 1943 have shown a steep increase in the prevalence of obesity since 1960, without any change in the psychosocial correlates [98]. In Denmark a prospective study of the psychosocial risk factors of developing obesity was performed. In 1974, 1,258 pupils aged 9–10 years were randomly selected. Most of them (86%) were followed up 10 years later. After controlling for age and body mass index in 1974, sex, and social background, it was found that family structure (biological or other parents and number of siblings) did not affect the risk of adult obesity. However, parental neglect greatly increased the risk (odds ratio 7.1) [72]. Furthermore, the risk of overweight was increased if the mother reported lacking knowledge about her offspring's eating habits and not affected if the mothers expressed acceptance of sweet-eating habits or if more than an average

amount of money was given for sweets [73]. Also, these findings were independent of social background (parents' school education, householder's occupational status and quality of dwellings in area during childhood). Moreover, parental education and occupation was not found to affect the risk of overweight when degree of fatness in childhood and gender were controlled for. However, the risk of overweight was greatly increased for individuals reared in an area with poor quality of dwellings, even when the effect of parental education and occupation was controlled for [71]. The overall conclusion of these conflicting studies may be that it is not only the obesity itself that affects the individual, but also the reactions of society to the condition.

Treatment of obesity

Introduction

Many different treatments for obesity have been investigated, including diet, exercise, surgery and medication. None has been found to be of particular value. It is now clear that treatment needs to be affirmative and long lasting. Single physical treatments are insufficient because of the accompanying psychological factors, and brief treatments fail to take account of the life-long genetic influence. The necessity of chronic treatment is now more widely recognized. This is due to the increasing knowledge of genetics, which has shown that many obese persons have an inherited susceptibility to the development of obesity. Thus, they need treatment throughout life and not merely a short period of training in a good exercise or diet program. After such a period it used to be thought that the problem of obesity was gone and the individual could return to his or her earlier, but somewhat improved, life-style without risking the repeated development of obesity. If they gained weight it was thought that their life-style was more unhealthy than the life-style of normal weight persons. The genetic discoveries now give room for another interpretation. For a person who has inherited the disease of obesity, it is not enough to live as normal weight people do as regards exercise and diet – such a person has to have a more than perfect life-style. A description of what strategies to choose from is given below. Of course, for anyone who has no genetic susceptibility to obesity it is much easier to reduce weight, just by living in the same way as other people of normal weight! However, for anyone who does have the genes, life-style needs special care! This leads to treatment of children at high risk of adult obesity, i.e., the most obese children. Of course, these children might also be helped by health programs aimed at the whole population (see "School-based treatments").

Diets

Moderate caloric restriction is recommended in children and has been evaluated in different programs [34, 40]. This moderate energy restriction does not impair the growth of obese children compared with nonobese children and with correction for the parental contribution to height [35]. For children 10–15 years of age we recommend a diet containing 1500–1700 kcal a day with 30 energy-percent of fat. This gives sufficient calorie restriction while not impeding normal growth during puberty. The calorie content must of course be adjusted for other factors, such as heavy physical activity, when an increase is necessary. It is rare for a diet with a lower calorie content to be needed. Usually, it is more effective to discuss general strategies in changing life-style than technicalities regarding diet (see the section on "Family therapy"). Very low calorie diets (VLCD) are often used in adults. However, they are not recommended in children owing to the risk of impairing the child's growth. Furthermore, the results in adults are not impressive even when VLCDs are combined with behavior therapy. The conclusion is that the weight loss that results from VLCD makes an impression during the first 6–10 months only. Adding behavioral procedures to VLCD increases weight loss for the first year or two, but not beyond 3–5 years [4, 82, 95, 104]. Furthermore, giving VLCD (420 kcal a day) results in a larger weight reduction initially, but after a weight maintenance program the mean weight loss was 10.94 kg, compared with 12.18 kg following a diet of 1,200 kcal a day [105]. Thus, the use of VLCD does not improve the results substantially. It is important to know this, as there is a risk of young teenagers copying adults' use of VLCDs hoping for good results. The VLCD developed from a "liquid protein" diet is associated with at least 60 deaths, but has been regarded as safe when limited to 3 months or less under careful medical supervision [103]. However, we recommend other strategies, not only in children but also in adults, as the initial weight reduction is heavily counteracted by the homeostatic mechanism in the body [70]. In our experience it is much more effective to make small life-style changes over some time. This makes the new life-style a part of daily life and not a cumbersome routine that has to be repeated whenever body weight deviates too much from the desired goal.

Exercise is natural for a child. In our experience it is not necessary to prescribe an exercise program for children under the age of 10 years, for they seem to be active by nature. However, during the teenage period a sedentary lifestyle becomes more evident, making it important to encourage the teenager to exercise. A combined approach encouraging both exercise and a more active life-style has been investigated [36]. The best result was in the group focusing exclusively on life-style, and not in the group targeted at both exercise and improvement of a sedentary life-style. Furthermore, routine or life-style activities.

such as fidgeting, an undetectable type of low-grade activity, may account for more energy expenditure than once thought [28]. This may be used in the context of a mixed program involving diet, exerting a significant impact on childhood obesity. During the family therapy sessions we just encouraged the family to perform physical activity at home [40]. Those receiving family therapy had better physical fitness than those who received the same type of advice regarding physical activity in the control group. This emphasizes that the psychological aspects of changing life-style are more important than the precise techniques of exercise. A comparison might be made with a photographer or a writer. Usually, many people focus on the camera and the techniques used when taking a picture, rather than the photographer's creative sense in recording an object or an event. Writers are not usually asked whether they have used pen, pencil, typewriter or word processor. It is obvious that these tools are less important than a writer's creative process. In treating obesity, too much emphasis has been placed on the techniques instead of on the global approach in changing life-style. Finally, it may be noted that although watching television is part of a sedentary life-style, playing video games is not. This activity causes an increase in metabolic rate and cardiovascular stimulation similar in magnitude to mild-intensity exercise [97].

Pharmacological therapy

The use of drugs in childhood obesity should be reserved for children with such conditions as Prader-Willi syndrome (short, overweight, cognitively impaired, emotionally labile individuals, often with a chromosomal abnormality) [52], in which an uncontrollable appetite may be painful for the patient. This is especially important when the patient is treated with gastroplasty (see below), leading to a reduced gastric volume. In adults the search for an anti-obesity drug is intense. Some major new possibilities are being investigated at the moment and are reviewed briefly below.

Monoamines

There are different mechanisms of action of serotonergic drugs on food intake, such as the release of serotonin by dexfenfluramine, inhibiting food intake. Fluoxetine inhibits serotonin re-uptake, also inhibiting food intake. For other monoamines, such as norepinephrine, some receptors increase and others decrease food intake, while histamine and dopamine decrease food intake by their receptors [14]. However, the serotonergic drugs are currently of major interest. Amphetamine is an addictive drug (α,β -phenethylamine) that is chemically related to dexfenfluramine, and it reduces food intake. The addictive nature is

probably related to its effects on dopaminergic neurotransmission and the anorectic effects to its influence on noradrenergic neurotransmission. However, other β -phenethylamines, such as dexfenfluramine, lack dopaminergic effects. Dexfenfluramine also lacks any effect on norepinephrine, but instead releases serotonin and inhibits serotonin re-uptake [15]. Usually, the chemical association with amphetamine has made it difficult to register these drugs for the treatment of obesity. The anti-obesity drugs are unfortunately thought to go on acting after the treatment has stopped. We do not expect blood pressure to stay low when treatment has stopped, nor do we expect a high cholesterol level to be reduced for ever after treatment. Anti-obesity drugs are expected to keep the obese at a desired weight, and failure is taken as proof of their ineffectiveness. The new discoveries regarding the genetic background of obesity and the different satiation signals may, hopefully, change this attitude. Traditionally, serotonin has been linked to the macro-nutrient carbohydrate via the intermediary step of plasma aminoacid ratios. However, some serotonin drugs can readily reduce the intake of high-fat foods, and there is evidence that serotonin is linked to cholecystokinin and enterostatin [10]. Fluoxetine (serotonin re-uptake inhibitor) is effective in short-term studies, but the long-term results (60 weeks) are less clear, although predictive factors have been studied [47]. Sibutramine, another inhibitor of serotonin re-uptake, has been tried in humans (6 months) and is regarded as a promising drug [94]. Dexfenfluramine gives a small but significant reduction after 1 year of treatment compared with placebo (9.8 ± 0.5 kg vs 7.1 ± 0.5 kg; i.e., 11% and 7% of initial weight, respectively). Long-term treatment is suggested for those who fail to adhere to a diet, with an attempt to withdraw the drug after 1 year [55]. Recently the drug has been withdrawn from the market owing to suspected coronary complications, but further studies are needed to evaluate the risks.

Lipase inhibitors

Lipase inhibitors selectively inhibit intestinal fat absorption by forming inactive intermediates with gastric, pancreatic and pancreatic carboxyl ester lipases. As the last enzyme makes absorption of lipid-soluble vitamins possible, decreased absorption is an expected side effect. Furthermore, steatorrhea is another common adverse effect. Short-term studies have been performed [29]. This and other data have led to the approval of orlistat in Europe, but not yet in the United States.

Combination of drugs

The combination of different drugs has been reviewed elsewhere [5]. Ephedrine stimulates the heart rate and in-

creases blood pressure, but the effects resolve fairly quickly. The effect on thermogenesis (energy consumption) when combined with methylxanthines such as caffeine and/or aspirin persists, however. Ephedrine and caffeine give 3.4 kg greater weight reduction than placebo after 6 months of treatment [3]. The mechanism of potentiation might be due to the effect of caffeine and aspirin on prolongation of norepinephrine activity. There is no additive effect on postprandial thermogenesis with aspirin, although caffeine and ephedrine give a significant effect [62]. Few long-term studies have been performed. The combination of fenfluramine and phentermine (a noradrenergic, centrally active agent) has been tried, but long-term safety has to be investigated in controlled trials. A preliminary trial led to withdrawal of dexfenfluramine from the market owing to suspected coronary complications.

Surgery

Surgery has been used for some time in the treatment of obesity in adults [68]. It started with intestinal bypass surgery during the 1950s and continued with the development of gastric bypass surgery during the 1960s. The gastric surgery was a combination of bypass of the food ingested and restriction of the volume of the ventricle (gastric bypass). However, besides perioperative morbidity and mortality, there are also long-term nutritional side effects. Owing to these problems, intestinal bypass and, to a large extent, gastric bypass have been abandoned. Pure gastric restriction (gastroplasty) was tried during the 1970s, leading to the present major techniques, i.e., vertical banded gastroplasty and gastric banding. These techniques reduce food intake by reducing the gastric volume: a small pouch is created, into which only a limited amount of food can be introduced. Eating more than 100–200 ml elicits vomiting, thus reducing food intake. Laparoscopic techniques are now being developed [87]. Space-occupying intragastric devices such as balloons have been abandoned for lack of effectiveness. Usually 60% of the excess weight has been lost after 5 years of follow-up when gastric restriction is performed. This result seems encouraging. Thus, gastroplasty is most widely used.

The main reason for failure is not technical but behavioral. Probably 30–40% of the patients are not helped by this enforced change of life-style. An individual eats “soft calories” (liquid calories such as mayonnaise), thus maintaining obesity. Why is this behavior not changed? The recent discoveries regarding the genetic background to obesity and the defective satiation power of fat might be an explanation. The satiety induced by restricted gastric volume may not be sufficient in some individuals. Furthermore, the small volume makes an ordinary meal impossible and makes the social side effects unacceptable for some individuals. Both motivational and cognitive factors probably influence outcome, although there are no pub-

lished studies to prove this. Although obesity surgery is effective for some, the side effects indicate the need for better patient selection. Consequently, obesity surgery is an approach that is very rarely used in children. It should only be discussed in the treatment of specific syndromes, such as Prader-Willi syndrome. In these cases early treatment, i.e. perhaps as early as at 10 years of age, might be beneficial.

Liposuction i.e., removal of subcutaneous adipose tissue, does not treat obesity itself. The adverse health effects of obesity are mainly correlated with visceral (intraabdominal) obesity, and the fat surrounding the intestines is not available for liposuction. Indications for its use in children are strictly limited (e.g., severe gynecomastia in boys during puberty).

Psychodynamic therapy

Psychodynamic therapy is probably used less for obesity than for eating disorders. However, Bruch's clinical observations of obesity also included the family [19, 20, 65]. The obese child was described as living in a dysfunctional family, i.e. one with disturbed communication between the parents and child. The child has difficulties in discriminating between emotions and other sensations that come from the body, such as hunger. Eating is then used as a replacement for other emotional needs. This response is founded early in the mother-child relationship, if the child's needs for love, warmth, food etc. are not adequately fulfilled. There are no recent published studies regarding psychodynamic therapy in obesity [90].

Behavioral and cognitive therapies

Behavioral therapy has been used in obesity management since it was first described [100]. The program was based on the belief that obesity was a “learned disease,” so that it would be possible to cure it by “re-learning.” However, successful long-term results have not been achieved [17]. Nonetheless, a 10-year follow-up did show lasting results when booster sessions were given for a period of 4 years. This indicates the difficulties of preserving good results and the need for long-term treatment [8]. Behavioral therapy of obesity is based on the concept of bad eating habits in which insufficient control of stimulus or rewarding behavior results in increased food intake. These habits can be broken down into small sequences, e.g. the frequency of chewing, of meals. The parents are expected to provide reinforcement for the children's improved eating habits. For example, a deposit of money may be paid back to the patient during weight reduction [33]. In 1983, Brownell and coworkers evaluated a program consisting in behavior modification, social support, nutrition, and exercise [18]. They noted that groups in which obese children and their

mothers met the group therapist separately had better results than those in which only the children were seen, or the children were seen together with their mothers. Others have also studied the effects of parent interaction, using three groups [34]. The first group consisted of child-and-parent pairs, where parent and child behavior change and weight loss were reinforced by behavioral techniques; the second group consisted of children only, and the children's behavior change and weight loss were reinforced; and in the nonspecific control group families were reinforced for attendance. The best result was achieved in the parent-and-child group. Recently cognitive therapy has been used in the treatment of obesity, usually combined with behavioral therapy. This combination is based on the assumption that, through practice and reward, changes in key areas of children's cognitive processing will result in behavioral changes. However, the causal connection between the attempt to influence the child's cognitions and the observed behavior changes has not been studied with stringent research designs [66]. There have been few studies evaluating cognitive with behavioral therapy. In one such study, 27 children aged 7–13 years were randomized to either cognitive therapy or behavioral therapy. No differences were found after 3 and 6 months' follow-up, and the therapies were equally effective [30]. The follow-up period was short, however.

In another study behavioral treatment was combined with either cognitive therapy or nutrition education [64]. The different treatments induced different ways of controlling the weight: for instance, in the cognitive group the weight-related cognitions were more adaptive than in the other groups. However, the analysis showed that there were significant differences in the obesity status across time, but not between the different treatments.

Finally, another study with a 3-month follow-up also showed that the addition of cognitive therapy to a behavioral program gave no further improvement beyond that achieved with the behavioral therapy itself [27].

Group therapy

Many different types of therapy can be utilized within the context of a group, and there have been some studies of this approach. For example a peer group behavior codification program of adolescents gave better results than previous individual contacts [111]. However, the development of group cohesion was tenuous and temporary. Girls who were functioning more independently appeared to do better with regard to weight loss. The study was not randomized, and the patient group was small.

In another study individual dietetic counseling, group dietetic counseling and group dietetic counseling with behavior modification were compared [74]. The first and last treatments were equally effective at 1-year follow-up and better than group dietetic counseling alone.

The general impression has been that group therapy has no decisive advantages over individual therapy [1]. Exceptions may include those groups that we select carefully to be strongly homogeneous for gender, age, and social background, for example.

School-based treatments

Behavioral therapy has also been used in a school setting [16]. The program consisted of behavior modification, nutrition education and physical activity. Parents and school personnel were involved. Sixty (95%) of the 63 children (5–12 years) in the 10-week program lost weight, compared with only 3 (21%) of the 14 control children. The program children showed a mean decrease of 15.4% in their percentage overweight, and lost an average of 4.4 kg.

Providing treatment in a wider context at school and furthermore perhaps promoting a good life-style not only to obese children but to all children may be a fruitful approach. However, long-term follow-up is difficult in such studies, with so many individuals being treated and so many variables to be controlled for. School-based treatments have been reviewed, but no single program was significantly better than the others, so that no recommendations could be made [107]. Most of the school-based programs are not directed specifically at obese children, but rather at unselected groups of children. Thus, this area needs more research and should perhaps be seen more as health education provided by society for all individuals.

Early treatment

Early treatment, i.e. treatment started before the major peak incidence of childhood obesity at the age of 10, has shown better results for preschool children than older children [26]. This type of treatment is in one way similar to school-based treatment, as it is common to use groups of children, but differs in being directed specifically at obese children (see above). Our experience is that in older children during puberty, acceptance is very low (10%, unpublished data) when group treatment is offered to children 14 years of age identified after screening as an alternative to individual treatment.

Family therapy

The family is regarded as basic to the child's psychological development and a major factor influencing the child's quality of life. Family therapy has been used for children with behavioral and/or emotional disturbances and for children with chronic diseases. Many studies have been performed, and they have been evaluated in several reviews [–25, 53, 59, 69]. These show family therapy to be

effective in asthma, diabetes, anorexia nervosa, bereavement and adult schizophrenia. It has also been possible to develop family-based diagnostic tests for families in which a child is showing different symptoms [57]. Psycho-educational family therapy has been used in schizophrenia. Orhagen and coworkers studied whether the educational or the therapeutic part of the program was most effective and showed that both were needed [88]. Another Swedish study has shown family therapy to be a cost-effective treatment for childhood asthma [54].

It has also been suggested that family therapy might be helpful in the treatment of obesity [46]: the use of family therapy in treating obese children in a population screened at school has been shown to prevent the progression of obesity in older teenagers if treatment is started at the age of 10 [40]. The families were selected from a population-based sample, and three groups were compared. The first group received conventional treatment, i.e., regular visits to a physician and a dietitian; the second group underwent six sessions of family therapy. In both groups the duration of treatment was 14–18 months. The third group received no treatment. At follow-up 1 year after the end of treatment the body mass index was significantly lower in the family therapy group than in the untreated control group. Furthermore, physical fitness was significantly better in the family therapy group than in the conventionally treated group, and the fat mass (measured by skinfold thickness) was significantly smaller. There was no difference between the family therapy group and the conventionally treated group in body mass index. This might be due to the better physical fitness in combination with the reduced fat mass, leading to a higher muscular mass and thus increasing body mass index.

The effectiveness of the family therapy has led to a primary prevention program in Malmö: screening for obesity, followed by family-based treatment for those at risk. We used a combination of Minuchin's structural model [79–81], and de Shazer's brief therapy model (de Shazer, 1982; de Shazer, 1985; de Shazer, 1988; de Shazer, 1991). The therapist used the structural model as a basis within

which the solution-based model exerted its influence. Usually the situations the families wanted to discuss were those in which the child or parents experienced difficulty in following the prescribed diet or recommended exercise, and not the recommendations per se. During therapy adequate information was essential for success in finding solutions. Usually the family was asked to discuss different solutions at home before the next session. The beliefs and thoughts of the obese child were essential to the process. To summarize, the following directives were found to be useful: (1) Give the family low intensive nonconfrontational contact (2) Identify the resources of the family and acknowledge them. (3) Show respect for the family and use noncondemnatory interventions. (4) Involve important individuals. (5) Try to identify the whole system and relate it to its context. (6) Accept the individual's definition of the problem. (7) Rephrase in a positive context. (8) Emphasize the positive solutions, and start with the small simple solutions. (9) Express appreciation. (10) Discuss an appropriate realistic lowest weight. (11) Give information about the time needed to achieve the goal in the longer term. (12) Convey the message that controlling overweight is hard work.

Conclusions

Prevention of severe obesity in children gives much more hope for the future than treating adults [37, 78, 109]. We also have the impression that learning to follow a healthy life-style, uncluding both diet and exercise, during childhood is more easily incorporated into daily adult life than an attempt to change later. Finally, the new possibilities provided by ongoing research in the field of genetics, combined with indications of better results obtained by preventing obesity in childhood, might give us greater opportunities of controlling the ongoing "weight explosion." It is important to believe what the patient tells us, i.e. dieting is no use. Instead of insisting on diet, we must help our patients to change their life-style.

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