Prevention of obesity – is it possible?

M. J. Müller, M. Mast, I. Asbeck, K. Langnäse and A. Grund

Institut für Humanernährung und Lebensmittelkunde, Christian-Albrechts-Universität zu Kiel, Kiel, Germany.

Received 10 July 2000; revised 10 July 2000; accepted 11 July 2000

Address reprint requests to: Prof. Dr M. J. Müller, Institut für Humanernährung und Lebensmittelkunde,Christian-Albrechts-Universität zu Kiel, Düsternbrooker Weg 17, D-24105 Kiel, Germany.
E-mail: mmueller@nutrfoodsc.uni-kiel.de

Summary

Obesity prevention is necessary to address the steady rise in the prevalence of obesity. Although all experts agree that obesity prevention has high priority there is almost no research in this area. There is also no structured framework for obesity prevention. The effectiveness of different intervention strategies is not well documented. Regarding *universal prevention* little rigorous evaluation has been carried out in larger populations. Obesity prevention has been integrated into community-wide programmes preventing coronary heart disease. Although effective with respect to reduction in cardiovascular risk factors these programmes did not affect mean body mass index (BMI) of the target populations. *Selective prevention* directed at high risk individuals (e.g. at children with obese parents) exhibited various degrees of effectiveness. However, at present, definitive statements cannot be made because of the limited number of studies as well as limits in study design. Finally, *targeted prevention* produced promising results in obese children when compared to no treatment. However, there are only very few long-term follow-up data. There is no clear idea about comprehensive interventions studying combinations of different strategies. It is tempting to speculate that predictors of treatment outcome (e.g. psychological and sociodemographic factors) may also serve as barriers to preventive strategies, but this has not yet been investigated. Taken together, obesity prevention should become a high priority research goal. First results of obesity prevention programmes are promising. As well as health promotion and counselling, better school education and social support appear to be promising strategies for future interventions.

Keywords: health promotion, nutrition education, Obesity, prevention.

obesity reviews (2001) **2**, 15–28

Introduction

The prevalence of overweight tendencies, moderate and severe obesity has been steadily increasing over the last 50 years. Obesity is the most common nutrition disorder in developed countries. Current estimates indicate that the prevalence of obesity is still rising, thus, overweight tendencies and obesity now represent a major public health concern. Concomitantly, the prevalence of obesity in developing countries is also increasing at an alarming rate. Obesity, once established, is difficult to treat. Therefore, prevention of obesity and its comorbidities are high priority research goals. However, at present, there is no structured framework for obesity prevention but numerous proposals for research and future directions have been suggested (1–4). Despite the national importance of obesity and costs to health services there are no nationally co-ordinated strategies for research and development in this area.

Until now only a few human studies have investigated the prevention of obesity. This may be explained by (1) lack of effective prevention strategies; (2) under-funding of public health and obesity research; (3) problems identifying risk individuals and groups; (4) non-understanding of obesity as a chronic, lifelong metabolic condition; (5) limited knowledge about the pathophysiology of obesity; (6) the assumption that obesity is metabolically or genetically explained and, thus, molecular biologists will offer the solution of the problem; and (7) by a lack of practical pre-

ventive skills and public health training as regular parts of medical education. The latter point is, in part, due to public health professionals of whom many regard their subject as population based and outside the clinical domain. Vice versa, clinicians sometimes feel that epidemiology and public health are inferior to clinical research. Public health itself has a dilemma because describing a problem (i.e. the obesity epidemic) without providing evidence that intervention strategies can prevent it is unsatisfactory.

Preventive strategies

Obesity is a public health problem and needs to be addressed from a population or community perspective, as well as with respect to the individual subject (2). Preventive strategies must tackle the individual, community and political level. In practice, different levels of prevention strategies aimed at conditions with many factors, such as obesity, have been used. First, intervention strategies are directed at everyone in a community with the aim to stabilize or to reduce the mean BMI in a population (i.e. *universal prevention*) (1,2). Second, *selective prevention* is directed at high risk individuals (e.g. children of obese parents or a family history of type II diabetes mellitus) (1,2). It is concerned with improving the knowledge and skills of people to increase competence and personal autonomy and, thus, to prevent excessive weight gain. Third, *targeted or secondary prevention*, is directed at overweight and obese individuals to prevent further weight gain and/or to reduce body weight (1,2). When compared with the latter approach *universal* and *selective prevention* are considered as primary prevention, i.e. these strategies are targeted at non-obese subjects.

Obesity prevention – first experiences in adults and children

At present there are no comprehensive population-wide strategies to specifically tackle the problem of obesity. Obesity prevention strategies have been integrated into community-wide programmes preventing coronary heart disease in adult populations (e.g. The North Carelia Project or the Deutsche Herzkreislauf-Präventionsstudie (DHP)) (5,6). These interventions consisted of mass media education, worksite and school-based programmes to integrate prevention strategies into community structures. Although capable of significantly reducing coronary heart disease risks and mortality, universal prevention was without effect on BMI in the The North Carelia Project (5). This finding is in line with the results of the DHP (6). In the DHP a number of health promoting initiatives have been offered to a representative group of more than one million people. Health promotion was brought about by a network of physicians, health insurances, communities and local authorities. As a result blood pressure, smoking habits and plasma cholesterol concentration all decreased but no net effect on BMI was observed (6). The Stanford Five-City Project is another community-wide health education programme geared toward prevention of strokes and coronary heart disease (CHD) (7). This programme was organized in collaboration with existing community organizations. Two treatment cities received continual exposure to five risk factor education campaigns per year. After 6 years, BMI increased rather than decreased within the intervention group (+0.42 kg/m^2); and an increase in BMI was also seen in the control group (change in BMI by +0.51 kg/m^2) (7). These findings are in line with the result of another universal prevention programme on the reduction of cardiovascular disease risks, the Minnesota Heart Health Programme (8). In this study three communities received a social and behavioural management programme. Over a follow-up period of 10 years mean BMI increased in both the intervention and the control communities (8).

Many authors consider obesity prevention as prevention of weight gain in children and adults in schools as well as in the wider community (9–11), and various degrees of effectiveness were reported. For example, the authors of the Minnesota Heart Health Programme specifically tackled weight gain in a larger community and reported recent results from a distinct activity within the programme, the so called Pound of Prevention (POP) study (10). POP addressed an adult overweight population (mean BMI varied from 26.1 to 28.2 $kg\,m^{-2}$). The study was conducted in collaboration with four local health Departments. Education on nutrition and exercise was brought about through newsletters, semiannual classes on nutrition and exercise, plus a lottery incentive for participation. Despite increased weight monitoring no changes in other targeted behaviours, as well as increases in body weight, were observed in all groups of participants during a follow-up of 1 year (10). However, the authors observed considerable differences between high and low income participants (9). They concluded that educational messages about diet and exercise may produce negative effects on weight in low income women. The further follow-up of POP will document whether low-intensity educational strategies will be effective in reducing rate of weight gain and if specific strategies for different socio-economic subgroups are necessary. The project SPARK (Sports, Play, and Active Recreation for Kids) studied the effects of physical education on adiposity in children (12). In a strict sense this was not an obesity prevention programme, because the authors only encouraged physical activity without addressing dietary modification. SPARK was a school-wide intervention aimed at fourth grade, 9-year-old children from seven suburban elementary schools in southern California. In 1990 a school-based physical activity promotion programme was offered by certified physical education spe-

cialists or classroom teachers within four schools. Data were compared with three control schools in which usual physical education featured. The children were followed for 2–3 years and total data sets were obtained from 305 boys and 244 girls. By spring 1992 there were no group differences in BMI as well as skin-fold thickness (12). Thus, the authors concluded that physical activity alone is not sufficient to reduce adiposity in children.

The 'Know Your Body' project (KYB) was initiated in 1975 (13). The aim of the programme was to modify the population distribution of risk factors for chronic diseases. It was evaluated in a 5 year field trial in the New York city area. The programme was school based, and teachers delivered. Parental participation in curriculum activities and attendance at seminars were intended. As a result favourable effects on health knowledge, dietary habits, blood cholesterol and rate of initiation or cigarette smoking were reported (13–15). However, KYB appeared to have no effect on body mass (15). These data were in line with two school-based studies, conducted in Finland and Norway, over 2 year periods of intervention (16,17). These community-based programmes targeted seventh to eighth grade (16) as well as fifth grade to seventh grade students (17). Both, the Finish and the Norwegian studies found favourable changes in food consumption and blood levels of cholesterol, but no favourable effect on obesity was observed (16,17). The authors speculated that tackling obesity requires more intensive and individualized interventions. Manios and co-workers adapted the KYB school health promotion programme on the island of Crete (18–20). The objective of the study programme was to promote healthy dietary practices and other health habits in children with the ultimate aim to reduce the cardiovascular risk in adult life. A school-based intervention directed at children, together with seminars organized for parents, was implemented. After 3 years, follow-up measurements obtained in a subgroup of 288 (intervention group) and 183 (controls) children, respectively, revealed positive changes in health knowledge and behaviour, as well as in plasma lipids in the intervention group (18). When compared with control children the age dependant increase in BMI was smaller in children of the intervention group (+0.7 vs. 1.8 $kg\,m^{-2}$; $P < 0.001$; 18). However, the concomitant measurements of fat mass by anthropometric methods showed only small and mainly non-significant group differences (18,19). Using a similar programme for 1274 third and fourth grades boys in North Carolina, the authors of the Cardiovascular Health in Children (CHIC) Study reported an immediate increase in health knowledge and self-reported physical activity in response to an 8 week exercise programme and an 8 week programme on nutrition and smoking (21). In the intervention group there was a non-significant decrease in blood cholesterol and body fat (21).

This is in line with the results of a primary prevention programme of cardiovascular disease in Moscow (22). The authors of that study followed 477 12-year-old boys over a period of 1 and 3 years. Methods used for health promotion included group counselling lectures within schools. As a result plasma lipids and blood pressure improved with the prevalence of overweight children decreasing within 1 year but then increasing again to reach the values of the reference population after 3 years (22). The Child and Adolescent Trial for Cardiovascular Health (CATCH) followed a total of 5106 third grade students (23). A third grade to fifth grade intervention included school food service modifications, enhanced physical education and classroom health curricula. During three school years the authors observed improvements in energy (fat) intake and physical activity but body mass, blood pressure and blood cholesterol remained unaffected (23).

In addition to the above mentioned studies a number of authors have studied the effect of obesity prevention or treatment in ethnic minorities (24). For example, data have been published on interventions directed at African-American mothers and daughters (25) as well as at Navajo and Pueblo Indians (26) aimed at obesity prevention by dietary changes (25) or diet and exercise (26). Psychological sessions and group discussions (25) or implementation of a school education curriculum (26) aimed at increasing knowledge and changing health behaviours were performed. Although weight data were not reported in all of the above mentioned studies interventions resulted in differences in percentage calories from fat within an 1 year follow-up (25). This is in line with another school-based intervention directed at Caucasian children (27). After 2 years these authors found that nutritional intake (less energy, fat and sodium, more fibre) as well as physical activity improved in children of the intervention schools (17). These prevention strategies can be considered as *selected prevention*, whereas, e.g. the Crete experience (18–20) and CATCH (23) are a *universal prevention*.

Few researchers (28–30) have shown effective management of overweight and obese children (i.e. *targeted prevention*) suggesting that this is a possible measure of prevention of adult obesity. Flodmark and co-workers suggest that family therapy was effective to prevent the progression of severe obesity in children of 10–11 years (28). These children were followed for 1 year after intervention was completed. Mean group BMI was 24.7 (family therapy), 25.5 (dietary counselling) and 25.1 $kg\,m^{-2}$ (control group) and reached 25.8, 27.1 and 27.9 $kg\,m^{-2}$, respectively, after 1 year ($P < 0046$ for family therapy vs. controls, 28). The increase in BMI was 5% in the group receiving family therapy vs. 12% in the control group ($P < 0{,}02$; 28). However, the groups differed with respect to the number of children with severe obesity (i.e. BMI > 30 $kg\,m^{-2}$; 1/20, 5/19, 14/48 in the three groups,

respectively; 28). Epstein and co-workers completed four randomized controlled studies (30–33). Three of these studies tackled obese children with obese parents who are at a greater risk of becoming obese. These authors were most successful when children were treated together with their parents. The follow-up period lasted up to 10 years (30). The effectiveness of these family based and lifestyle interventions was well documented by weight changes, a reduced prevalence of obesity as well as lifestyle changes (30–33). The 10 year decreases in overweight percentages were –7.5% vs. +14.3% in the control group (30).

There were only two controlled studies on obesity prevention in non-obese children (34). First, a group of 80 children of 3–4 months old were given a prudent diet by educating their mothers in a private partnership practice (34). The children were then followed for up to 3 years. Out of 80 children from the intervention group one was overweight. In the control group 12 out of 50 children were overweight (34). These children showed a remarkable difference as they grew. By the age of three the diet group showed a prevalence of overweight tendencies at 1.28% compared with the 'average American diet' group with a prevalence of 25.5% (34), suggesting the value of early interventions. In the second study, a group of 106 (intervention school) and 100 (control school) children of approximately 9 years of age underwent a school intervention programme consisting of 18 lessons with a duration of 30–50 min (35). The intervention was based on social cognitive theory and intended a reduction in TV-watching, videotape and video game use. A 6 month follow-up showed a significant decrease in children's television viewing as well as in the consumption of high-fat foods. There was an age-dependent increase in various indices of fatness in both groups. However when compared with controls the relative increase in BMI and fat mass were reduced in the intervention group (35). These data suggest that decreasing physical inactivity decreases body fatness (35). This is in line with the results of a previous study on overweight children (33). The results of the two studies are promising but long-term follow-up was not monitored.

Suitable periods for obesity prevention

Some authors assume that early intervention in a paediatric population is superior to intervention strategies directed at adults (1–4,36). Specific periods for the development of overweight problems and obesity have been identified in children. These include the prenatal period, the period between the ages of 5 and 7 (so called adiposity rebound) and adolescence (36). Although most people become obese as adults, there is a significant association between BMI in childhood or adolescence and in adults (37–40). The persistence of obesity into adulthood appeared to rise linearly throughout childhood. In addition, overweight tendencies in childhood were shown to be predictive for adult morbidity and mortality (40,41). Antecedents of adult disease (hypertension, hyperlipidaemia, abnormal glucose tolerance) occur with increased frequencies in obese children and adolescents (42–46). However, there are only a few long-term follow-up data spanning from childhood to adulthood. These data suggest life-long persistence and health consequences of overweight tendencies and obesity in many children (42). Preventive strategies should, therefore, consider critical periods of early onset of obesity. It has been proposed, that prevention of paediatric obesity may be the only effective treatment of adult obesity (1). Taken into account the long-term health risks of child and adolescent fatness obesity prevention in children may also add to prevent obesity-related diseases in adulthood.

Suitable interventions to prevent weight gain

Prevention strategies aimed at childhood and adult obesity are based on the knowledge of risk factors and beliefs about the aetiology of the disease. Risk factors of childhood obesity include parental fatness, social factors, birth weight, timing or rate of maturation, physical activity/inactivity, dietary factors (including early infant feeding practices), and other behavioural and psychological factors (47). These risk factors are related but their relationship is unknown at individual, as well as at population levels. Although most risk factors for obesity seem to be self evident, their confounding or cumulative effects on the development of obesity, as well as their clustering and their effects over time on the causal pathway of the development of obesity, remain unclear with respect to a given individual as well as with respect to a greater population of obese subjects. In children, as well as in adults, a quite comfortable lifestyle and an almost unlimited individual consumption are associated with the marked increase in obesity prevalence (48). There is evidence that simple strategies to reverse this trend include eating regular meals, avoiding snacking, drinking water instead of calorie containing beverages, reducing dietary fat below 30% of energy intake, cutting down TV-time ($<1\,h\,d^{-1}$) and/or being more active (49). In addition to the environmental and behavioural determinants of body weight the importance of genes for body weight and obesity has been demonstrated in animals and humans. From a public health point of view it is important to consider that during the last 50 years the prevalence of obesity increased within a genetically stable population suggesting the importance of obesity promoting and/or inhibitory characteristics of the macro-environment (48). The enormous secular change in obesity is evidence for the strong impact of environmental factors on large numbers without any individual being identified. This idea is also supported by data from large family studies showing that in a heterogeneous population the heritability of different

measures of body fat content varied between only 5 and 40% (maximum) of the age- and gender-adjusted phenotypic variance (50). Although the concept that some people may have inherited a greater susceptibility to store excess energy than others is attractive, most prospective studies on the impact of different metabolic phenotypes (e.g. subjects with a reduced metabolic rate and/or high fat oxidation) on weight changes were done in selected populations (e.g. Pima Indians 51) and could not be reproduced in other populations (52,53,54). These data indicate that in genetically heterogeneous populations metabolic factors are, at least, weak predictors of weight gain and in most people the propensity to become obese is not due to measurable and inherent metabolic abnormalities, thus, questioning the concept of increased energy efficiency in overweight and obese subjects. Although detailed knowledge of interaction between individuals and the environment may add to future intervention programmes to prevent obesity, present public health strategies consider critical periods (which are irreversible, e.g. prenatal, childhood, adolescents) and high risk periods (which are reversible, e.g. pregnancy, smoking cessation, etc.) (47). From a public health point of view some risk factors are modifiable (e.g. dietary intake) but others (e.g. genetic factors) cannot be changed and, thus, are not a target of intervention strategies. Most authors agree that action is primarily required at societal level to counter the environmental influences on physical activity and dietary intake. Emphasis is now more on physical activity rather than on dietary changes. Interventions to prevent weight gain include group sessions, school programmes, correspondence programmes, individual counselling, behaviour change methods and/or individual/group exercise programmes. At present, it is unclear whether more complex techniques add to effectiveness of the provision of information alone or if face-to-face interventions are more effective than those delivered by post, for example. When starting a prevention programme the likelihood of implementation, cost and geographical scope have to be considered too.

Outcome measures of obesity prevention programmes

Suitable outcome measures of obesity prevention are objective means or self-reported measures of the nutritional state (e.g. BMI), comorbidities (e.g. a decrease in blood pressure), behaviour changes (e.g. in diet or physical activity) and/or increases in competence and health knowledge. To be sure about the effects long-term follow-up data are necessary. At present there is no scientific consensus about suitable outcome measures as well as follow-up periods in studies on obesity prevention (1,2). Taking into account the criteria for evaluating the outcome of approaches to treat obesity published by the Food and Nutrition Board of the Institution of Medicine (1), 'long-term' has been considered for follow-up periods of at least 1 year, but clearly more prolonged periods are preferable in obesity prevention studies. Prevention of weight gain and weight loss are suitable outcome measures in studies on individuals. From a public health point of view an improvement in health-related behaviour within a population and/or a decrease in health risks are preferable outcomes. The prevalence of comorbidities and side effects have to be regularly monitored in prevention programmes. Today obesity prevention strategies frequently resemble those of obesity treatment. Some authorities feel that obesity prevention is just preponing the principles of obesity treatment. This idea is misleading, since health promotion and prevention include non-specific (i.e. non-body weight and non-diet related) measures. By contrast, treatment specifically tackles body weight, e.g. by diet and exercise. The public health idea suggests that in a given population the prevalence of obesity is related to the mean body mass index of that population. Prevention strategies are targeted at non-obese as well as obese subjects in order to shift the median BMI of a given population to the left and, thus, reduce the prevalence of overweight tendencies and obesity. By contrast, treatment strategies tackle 'only' overweight and obese individuals in order to reduce their body weight and/or to prevent further weight gain. These two approaches are not in competition. However, it is worthwhile to formulate outcome measures according to different types of prevention (1).

Outcome measures for *universal prevention* are:

(1) reduction in the prevalence of obesity in the general population;
(2) an overall reduction in average weight of the population;
(3) improvements in nutritional intake, eating habits, exercise and other health-related activities;
(4) improved knowledge, attitudes and norms regarding nutrition, weight, eating habits and exercise;
(5) decreased rates of comorbidities;
(6) public policy and environmental change indices.

Outcome measures for *selective prevention* are:

(1) prevention of weight gain in high risk individuals;
(2) decreased excessive dieting among dieters;
(3) improved lifestyle patterns (e.g. better nutrition).

Outcome measure for *targeted prevention* are:

(1) reduction in the number of obese-related comorbidities;
(2) increase in the number of obese subjects who are successful in attaining and maintaining a relatively small

weight loss (e.g. 10% of initial body weight) and a decrease in the number who gain a small amount of weight (e.g. +2 kg).

It is evident from this catalogue that it is difficult and time consuming to evaluate the impact of an obesity prevention programme in a greater population. Obviously there is a further need for consensus, e.g. regarding the definitions of success (e.g. what is a successful reduction in the prevalence of obesity in a population?) or suitable methods used to document changes in nutrient intake and physical activity in greater populations. Outcome measures can be more exactly defined in studies on high-risk individuals with interventions that are matched or targeted to specific risk factors. Since there have been and, as far as we are aware, are going to be only very few studies in the area of obesity prevention, a need for and also the chance of standardization and evaluation of the studies is obvious.

Prevention of obesity: concept and first results of the Kiel Obesity Prevention Study (KOPS)

Concept

Based on our current and limited knowledge and the idea that prevention of childhood obesity is an effective treatment of adult obesity, the Kiel Obesity Prevention Study (KOPS) was started in 1996 (55). KOPS is an ongoing 8 years follow-up study. We first enrolled a large scale cohort of children of 5–7 years, who were/are characterized with respect to bio-behavioural determinants of body weight. The cross-sectional data will be further extended by longitudinal data sets allowing analyses of changes over time and intraindividual comparisons of the parameters measured. All subjects are going to be re-investigated after 4 (at 9–11 years), and after 8 years (at 13–15 years) using an identical study protocol. In addition to the assessments interventions, education curriculum, counselling and sport programmes, are organized at three schools for all children, parents and teachers as well as within families of overweight and obese children and their parents. The intervention programme of KOPS is based on the assumption that attitudes of behaviour or other factors (e.g. familar disposition for becoming overweight and obese, low social state) are predictive for weight changes. We believe that prevention of becoming overweight is possible by choosing a healthy lifestyle. This message is important for all people but it is of particular interest for those carrying a risk (e.g. a child with a familiar disposition for becoming overweight and obese, low social state). Lifestyle changes are accomplished by increased knowledge, self-monitoring, self-esteem and personal autonomy. A healthy lifestyle can be supported by counselling, education and social support. Within KOPS the same behavioural and educational messages are given to all children and their parents. These are (1) eat fruit and vegetables each day; (2) reduce the intake of high fat foods; (3) keep active at least 1 h a day and (4) decrease TV consumption to less than 1 h a day. These messages were delivered to all primary school children within their first year in three representative schools in Kiel. An 8 h course of nutrition education including so called 'active breaks' was offered by a skilled nutritionist together with a teacher. The messages were also addressed to their parents during a school meeting. In addition, the teachers were trained regularly by a structured nutrition education programme within a continuation class. In addition to 'school interventions' for all children, parents and teachers, families with overweight and obese children and/or obese parents (BMI > 30 $kg\,m^{-2}$) were offered a face-to-face counselling and support programme within the family environment. We propose that interventions within the family context has a greater impact on the child than individual counselling. Facilitating family functioning, irrespectively of whether it is impaired or not, improves outcome (28). The family intervention consisted of 3–5 home visits organized by a nutritionist. Family counselling considered personal preferences of individual family members and was based on the structure, organization and lifestyle (diet, activity) of children and parents. Reinforcement and corrections were done on the basis of the individual behaviours. Counselling and organization within families (e.g. 'shopping', 'cooking', 'reducing sedentary behaviours' 'resetting the family table') were performed at 3–5 occasions within a period of 3 months. Special attention was paid to healthy food choices for between meal eating. In addition, a 6 month structured sport programme (2 times a week) was offered to overweight and obese children. KOPS allows further analyses of the role of individual risk factors as well as of long-term effectiveness of different intervention strategies. The primary hypothesis being evaluated is that participants of the intervention groups and schools will gain less body weight and fat mass over the course of the study than those in the control groups. We hypothesize that the prevalence of being overweight increases spontaneously from 22% (5–7 year-old children) to 27% (9–11 year-old children) and 35% (13–15 year-old children). The cumulative incidence of being overweight in a risk group (children of obese parents) was assumed to be 12.5% per year. We also assume that about 10% of normal weight 5–7-year-old children without risk become overweight at the age of 9–11 and 13–15 years, respectively. Other questions of interests are whether the effects of the interventions differ between individuals with different baseline characteristics and attitudes of behaviour or other factors (e.g. familiar disposition for overweight and obesity) are predictive for weight changes, individually or in combination with intervention assignment.

Risk factors for obesity in 5–7-year-old children

The cross-sectional data obtained on 2460 children between 1996 and 1999 show a marked increase in the prevalence of being overweight in 5–7-year-old children (Fig. 1a,b) (55,56). Our data did not provide evidence for the idea that attitudes of food intake are predictive of becoming overweight (55,57). The frequency of individual food choices, as well as an overall diet quality index, did not differ between overweight, normal weight and underweight children (57). By contrast, mean BMI, as well as the prevalence of being overweight, were increased in children with low activity as well as in children with a high TV consumption (>1 h when compared to <1 h per day; 58). Children with a high TV consumption more frequently had unhealthy food choices suggesting an accumulation of unhealthy behaviours (55,57,58). A high level of physical inactivity together with unhealthy food choices were more frequently seen in children from families with a low social state (58,59). In 5–7-year-old children there is a linear social gradient in unhealthy behaviours (55,59,60). (See Fig. 2 for social gradients in food intake and Fig. 3 for the association between physical activity/inactivity and social state.) It is obvious from our first analysis of data that besides health-related behaviours parental obesity and a low social state are associated with an increased mean BMI and a higher prevalence of being overweight in children (55). Both factors add to each other and the highest mean BMI is seen in children with two obese parents and a low social state (K. Langnäse, M. Mast, M.J. Müller, unpublished data). The differences between children differing with respect to parental fatness could not be fully explained by differences in health-related behaviours. A similar linear social gradient was seen in children differing with respect to parental fatness. This is in accordance with an inverse social gradient for BMI and the prevalence of obesity: the highest prevalence of being overweight as well as the highest mean BMI is observed for low social class children (Fig. 4). The social gradient is more pronounced in children of obese parents (K. Langnäse, M. Mast, M.J. Müller, unpublished data).

Interventions

Intervention strategies of KOPS are shown in Fig. 5 (55,61). Between 1996 and 2000 school interventions targeted a total of 414 children and their parents, as well as 30 teachers. In addition, 92 out of 368 eligible families completed the family intervention programme. The acceptance of school intervention reached 87%, 75% and 100%

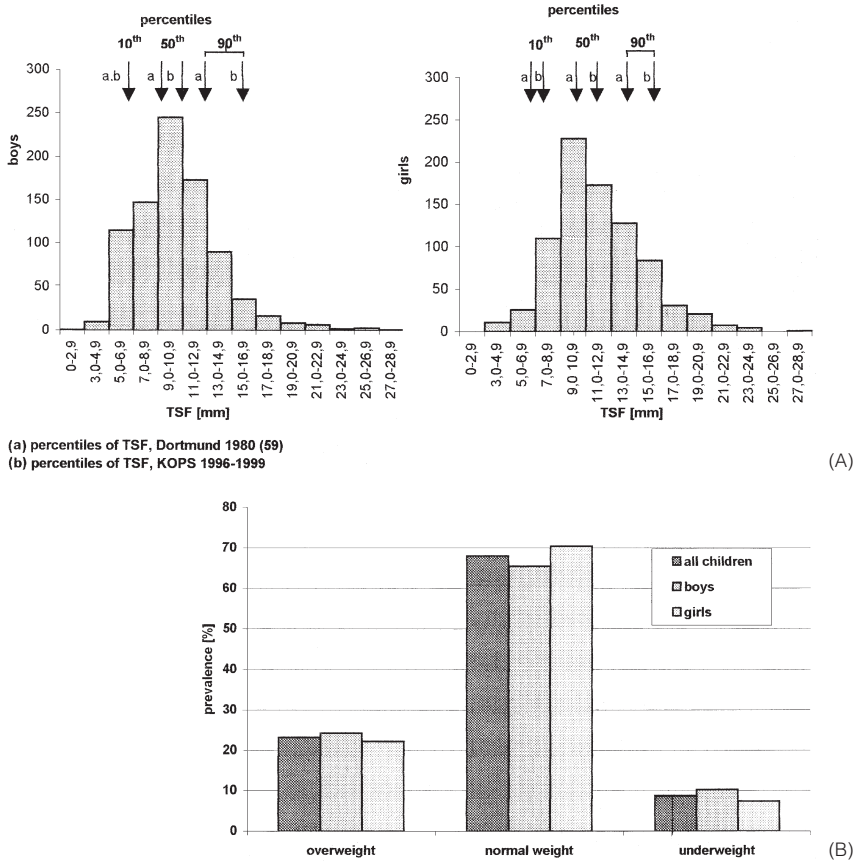


Figure 1 Distribution of triceps skinfold (A) and prevalence of being overweight, normal weight and underweight (B) in 5–7 year-old children investigated between 1996 and 1999 in Kiel. (A) (a) Percentiles of TSF, Reincken *et al.* 1980 (64): 214 boys aged 6 years: TSF (triceps skinfold thickness): 9,3 mm (4, 4–20,2), 213 girls aged 6 years: TSF: 10,7 mm (5,3–22,0); (b) Percentiles of TSF, KOPS 1996–1999: 765 boys aged 6 years: TSF: 10,3 mm (3, 6–26,0), 819 girls aged 6 years: TSF: 11,3 mm (3,3–24,7). (B): a: 739 boys aged 6,3 years(5,0–7,8): BMI (body mass index): 16,1 kg m^{-2} (11,1–32,1); b: 758 girls aged 6,3 years (5,0–7,8): BMI: 15,8 kg m^{-2} (9,3–25,1); overweight is defined as TSF ≥ 90th percentile from Reincken and co-workers (64), normal weight as TSF between 10th and 90th percentile and underweight as TSF ≤ 10th percentile.

foods social class[a]	whole wheat bread	white bread	fruits	potatos	yogurt for children	sweets	crisps	limo-nade
low middle high	▲	▼	▲	▼	▼	▲	▼	▼
p[b]	<0,001	<0,0001	<0,0001	<0,05	<0,0001	<0,05	<0,0001	<0,0001

▲ daily consumption **increases** from children of low social class to children of high social class

▼ daily consumption **decreases** from children of low social class to children of high social class

[a] chi²-tests testing the following consumption frequencies: daily ↔ several times per week + once a week + less/never

Figure 2 Social gradients in food intake. Chi²-tests testing the following consumption frequencies: daily ⇔ several times per week + once a week + less/never. Boys: n = 673, girls: n = 677; mean age: 6,2 years, mean BMI (boys): 15,9 kg m^{-2}, mean BMI (girls): 15,7 kg m^{-2}.

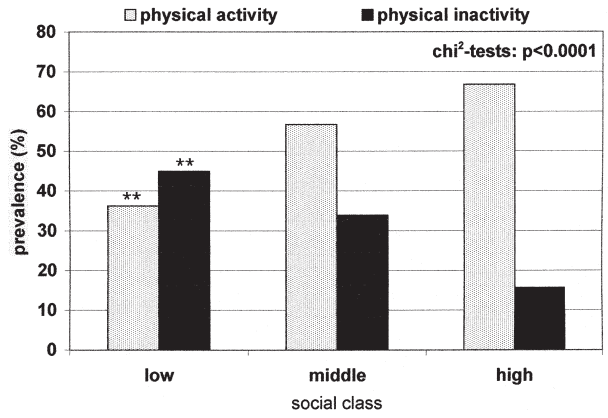


Figure 3 Association between socioeconomic state (as charaterized by parental school education) and TV-consumption and regular sports activities in a group of 1690 5–7 year-old children investigated between 1996 and 1999 in Kiel. Boys: n = 673, girls: n = 677; mean age: 6,2 years, mean BMI (boys): 15,9 kg m^{-2}, mean BMI (girls): 15,7 kg m^{-2}.

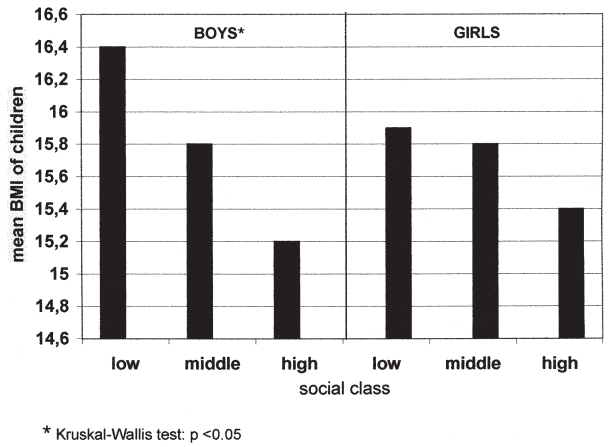


Figure 4 Association between socioeconomic state (as charaterized by parental school education) and mean BMI of 1690 5–7 year-old children investigated 1996–1999 in Kiel. Boys: n = 673, girls: n = 677; mean age: 6,2 years, mean BMI (boys): 15,9 kg m^{-2}, mean BMI (girls): 15,7 kg m^{-2}.

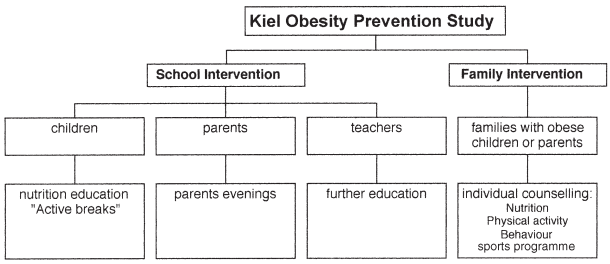


Figure 5 Intervention strategies of KOPS.

of children, parents and teachers. 48% of eligible families were visited for one session, at least 25% of the families completed the programme. The short-term effects were studied for all children within 3 months after the end of the interventions. As a result of school interventions good nutrition knowledge (as assessed by a questionnaire) was observed in 60% of the children, compared to the previous 48% (i.e. an increase in 25%). In addition, daily physical activities were reported by 65% of children, i.e. an increase of 12% from the previous 58%. Concomitantly, a further 28% of the children became members of a sports club. Regarding family intervention, daily fruit and vegetable consumption increased by 50% (from 40 to 60% of the children). Concomitantly the frequency of daily intake of low-fat food increased from 20% to 50%. Daily physical activity also increased and was observed in 68% of the children compared to the previous 50% . This increase was associated with a decrease in TV-watching from a mean value of 1.9 h to 1.6 h day^{-1}. All changes became significant at $P < 0.05$. The results of a 1 year follow-up of a group of overweight children after a 6 month structured exercise programme show improvements in physiological variables (Fig. 6). The nutritional state of children in the so called 'intervention schools' was reassessed after a period of 1 year. Data were compared to the nutritional state of children in matched 'control schools'. Figure 7 shows the changes after 1 year in anthropometrically

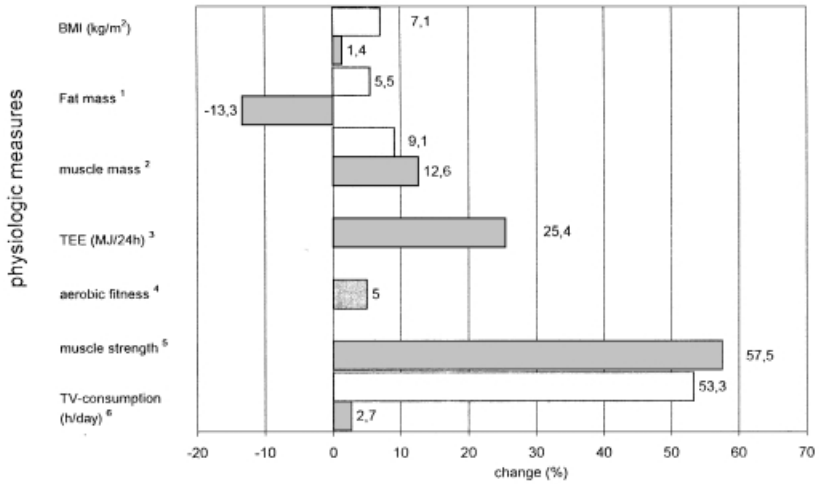


Figure 6 Changes in physiological outcomes after a 6 month structured sports programme in 17 pre-pubertal overweight children who were followed for 1 year (■). Data were compared with 15 control children (without intervention □). BMI: body mass index, [1] fat mass measured by anthropometry and bioelectrical analysis as described previously (60), [2] upper arm muscle area, [3] TEE: total energy expenditure measured by indirect calorimetry and 24 h HR-monitoring, [4] submaximal respiratory exchange ratio at a fixed heart rate from 170 bqm, [5] muscle strength of legs measured by computer tensiometry, [6] assessed by questionnaire.

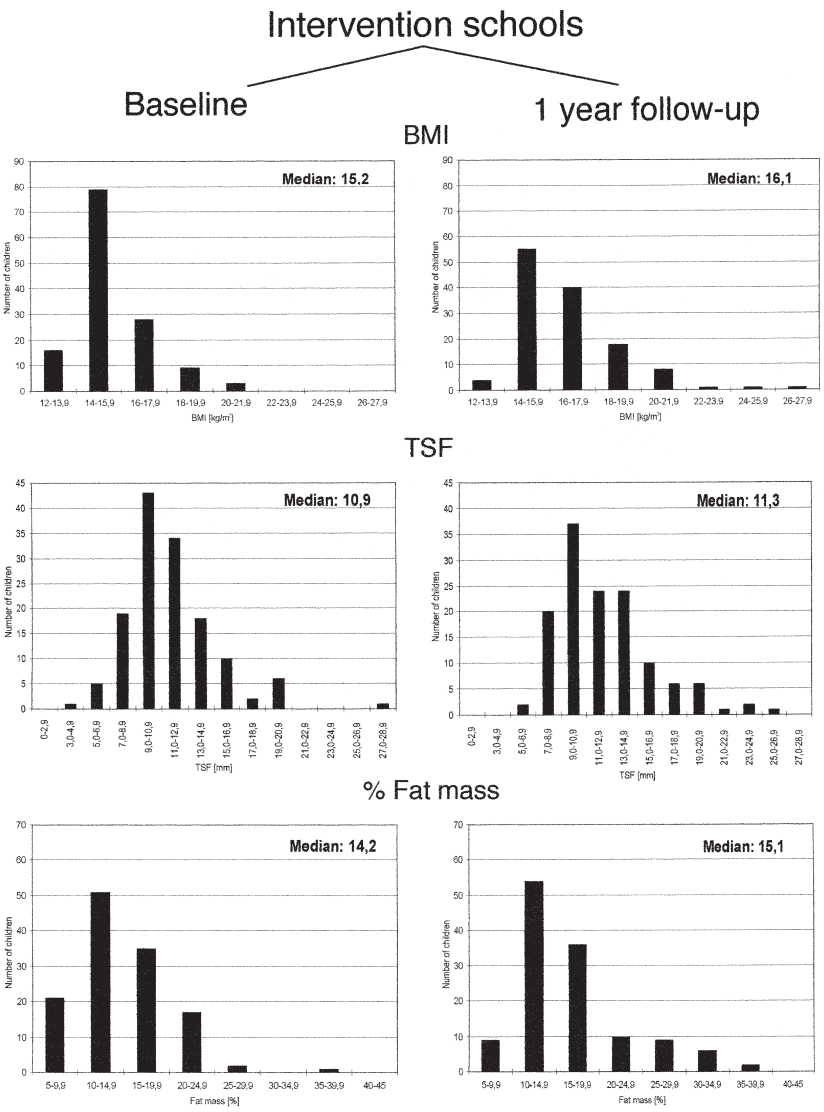
derived fat mass in all children of 'intervention' and 'control schools'. During 1 year there was a significant change in measures of fat mass in both groups. When compared with children of 'intervention schools' children of 'control schools' showed disproportionate increases in the median TSF, as well as percentage fat mass (Fig. 7).

Discussion

There are only a few studies on obesity prevention in children and adults. *Universal prevention* directed at reducing cardiovascular risk factors in greater populations of adults did not affect the nutritional state in adults. Although different with respect to study populations, study design and intervention strategies, prevention studies addressed at children showed some improved outcomes. Studies using *selective* or *targeted prevention* strategies also showed some improvement in health-related behaviours, as well as nutritional state in overweight and obese children. *Targeted prevention* in obese children was most successful when children were treated together with their parents. However, short-term follow-up periods, as well as the inconsistent use of outcome measures, limit the value of published data. There is only one long-term (i.e 10 year) follow-up study on *targeted prevention* in a small population of obese children aged 6–12 years (30). The effectiveness of this family based and lifestyle interventions was well documented by decreases in percent overweight (–7.5% vs. +14.3%; 30). These are promising results. However, the data also reflect the long-term limits of prevention and treatment strategies. The mean values of changes in nutritional state and health habits may camouflage the fact that preventive strategies are more effective in some but are without, or even have negative effects, in other subjects. At present we do not have measures to identify suitable responders for intervention strategies. It should be mentioned that comprehensive interventions studying combinations of different strategies have not been performed. Although some positive effects were reported, simple interventions in a single area (like a school health education programme) are unlikely to work on their own. The influences of the parents, peer group pressure, advertising, self image, etc. have to be taken into account to develop tactics to match the complexity of the causes (14). At present there is no concerted action but many strategies in health promotion are followed in isolation.

When compared to the studies mentioned above, KOPS combines different prevention strategies directed at a group of children between the ages of 5 and 7 years. Since Kiel is not a healthy city, KOPS is also done in isolation. First results of a 1-year follow-up study are promising. They show some improvement of health-related behaviours as well as the nutritional state of children. Familial disposition and social state are considered as covariates of body weight and, possibly, the effects of the interventions. Our data suggest that in 5–7-year-old children there is already an inverse social gradient in unhealthy behaviours. The manifestation of health risks in terms of being overweight is strongly affected by parental fatness. Thus, simple messages (e.g. eat fruit and vegetables each day, reduce the intake of high fat foods, keep active at least 1 h a day and decrease TV consumption to less than 1 h a day) will not counter the whole problem. It is tempting to speculate that a simplifying approach (which is necessary in preventive medicine) will weaken the success of the intervention.

At present we feel that school curricula on health promotion have to be established and implemented for all children; this is a *sine qua non* for a future decrease in obesity prevalence. In addition, *targeted interventions* directed at children with obese parents and a low social class should be encouraged. It is well documented that middle and high class, as well as intact families, were able to benefit better from treatment than families sharing other characteristics. Future interventions should, therefore, consider that besides traditional counselling and information we should



(A)

Figure 7 Distribution of BMI triceps skinfold (TSF) and % fat mass in 297 children (136 in the intervention group and 161 in a sociodemographically matched control group) at baseline and after 1 year with or without school-based intervention. When compared with the control group there is a significant shift in the distribution of TSF 1 year after intervention ($P < 0.01$), whereas no group differences in the distribution of TSF were observed at baseline. Intervention schools: 61 girls aged 6,3 years (5,0–7,8), BMI: 15,5 kg m^{-2} (12,5–21,2) at baseline, 16,2 kg m^{-2} (12, 5–26,2) at follow-up; 75 boys aged 6,3 years (5,0–7,8), BMI: 15,6 kg m^{-2} (13,2–20,5) at baseline, 16,6 kg m^{-2} (13,8–24,3) at follow-up. Control schools: 82 girls aged 6,1 years (5,0–7,1), BMI: 15,7 kg m^{-2} (11, 5–25,1) at baseline, 16,7 kg m^{-2} (11,7–25,9) at follow-up; 79 boys aged 6,2 years(5,0-7,3), BMI: 15,6 kg m^{-2} (12, 1–20,5) at baseline, 16,6 kg m^{-2} (13,1–22,6) at follow-up.

also use other indirect strategies directed at social support of individual families (e.g. let us try to understand why some families have lost the social context of eating, and how we can help them to 'reset the family table') as well as better school education for all children. In the long-term these strategies will help to improve the knowledge and skills of individuals and hopefully reduce the steady rise in the prevalence of obesity.

It has been suggested that the pandemic of obesity requires an ecologic approach characterized by a collaborative strategy with multiple sectors which impact on the problem (62,63). Thus, strategies aimed at obesity prevention should also tackle the physical, economic and sociocultural environment by e.g. policy, legislation or food technology. This idea is a wider public health approach resembling epidemiological triads used to control other epidemics. At present we do not have much experience tackling the obesogenic and creating a supportive environment. Swinburn and co-workers have applied a framework for identifying and prioritizing environmental interventions for obesity (i.e. the ANGELO framework, 63). At a population level the authors used it with stakeholders from the Torre Strait and the Pacific islands. In addition, at the setting level the programme was applied to fast food outlets. These are first and important steps to tackle the obesity epidemic at a population level. However, the value of these strategies has not been confirmed by follow-up.

Conclusion

Obesity prevention is necessary. It is an ideal, but yet unproven, measure to address the steady rise in the prevalence of obesity in developed countries. It is tempting to speculate that successful obesity prevention will lead to

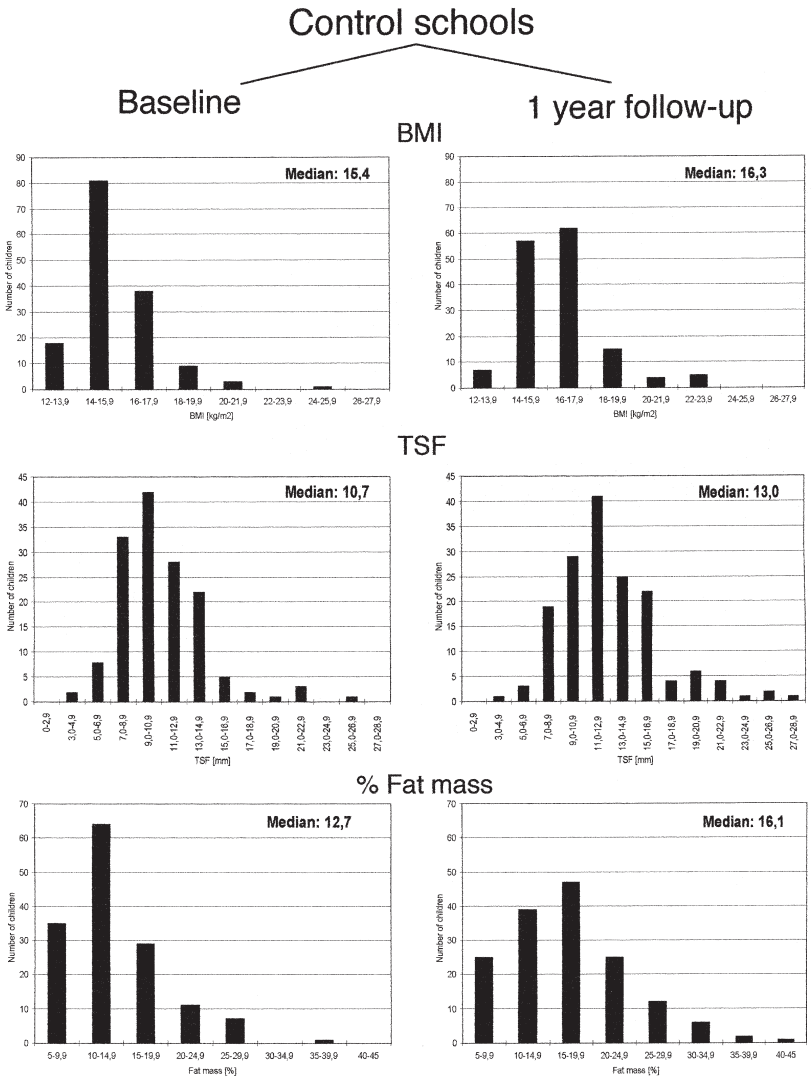


Figure 7 *Continued* (B)

a decreased prevalence of metabolic syndrome, diabetes mellitus, hypertension and cardiovascular disease and, thus, will have beneficial public health consequences. These strategies should be extended by an 'ecological' approach creating a supportive environment. Treatment and prevention strategies have to be complemented by community interventions and healthy public policy (Fig. 8). However, some authors feel that we presently do not have the experience and tools to prevent obesity (1). In fact, there are only a few controlled studies in this area. The results of these studies are promising, although they show only limited effects of different prevention strategies on various outcome measures. Thus, optimistically, we may conclude that obesity prevention is possible and beneficial. But simple messages and naive enthusiasm will not solve the problem. Interventions in a single area are unlikely to work on their own. Further inputs (e.g. from local authorities, implementation of national programmes of child health promotion within schools or the communities; involving food industry) are necessary. Besides health promotion and counselling, better school education and social support seem to be the most effective strategies for future interventions. Taken into account the public health dimension of the obesity epidemic there is a need for further controlled studies using comprehensive interventions for obesity prevention.

A note added

The need for public health strategies to prevent obesity has been frequently stated. But no one has been doing the job yet. It is not only a matter for political authorities but also a matter for ourselves as scientists. Obesity research is still not the number one research task. For example, in Germany there are numerous Departments in the field of human nutrition but only two or three groups are working

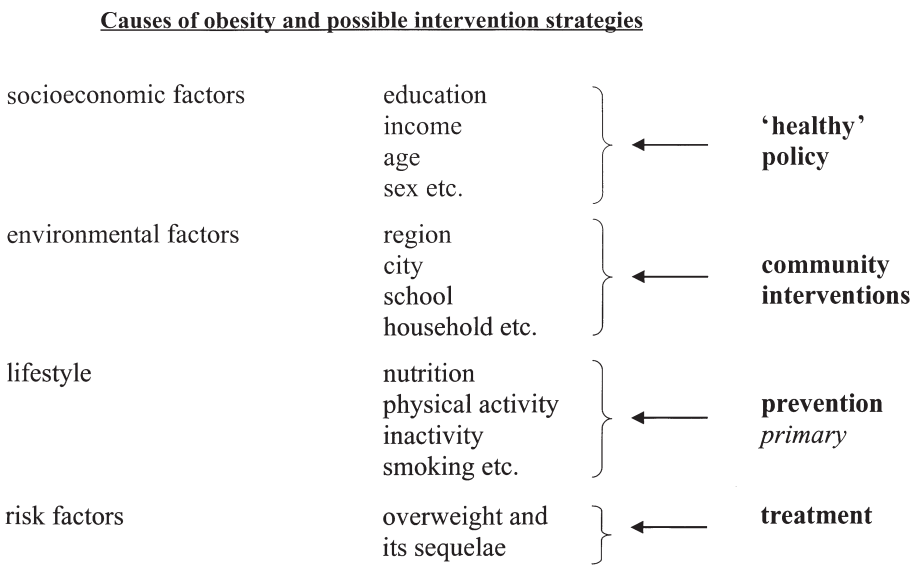


Figure 8 Levels of causation of overweight and obesity and corresponding types of intervention. Modified according to McKinlay and Marceau, 1999 (65).

in the area of obesity research. In addition, nutrition comes only second in the curricula of postgraduate university courses of public health, and obesity is not even a topic of public health research in Germany. Obesity research itself needs a further comment: After having followed about 30 years of obesity research the first author of this article cannot ignore the fact that besides focusing more and more on the molecular basis of obesity the prevalence of obesity has still been rising. Faced with this discrepancy we have to ask ourselves: Why do most scientists still prefer to address the pathophysiology of obesity rather than entering the area of obesity prevention? The members of the scientific community should make up their minds: If obesity really is the challenge of this millennium we have to work hard in the area of obesity prevention. Clearly, speaking about obesity prevention is easier than doing the job. But the job has to be done.

Acknowledgements

The authors wish to thank all co-workers, teachers, parents and children as well as the school physicians of the city of Kiel. KOPS is supported by Deutsche Forschungsgemeinschaft (DFG Mü 714 5–1), Schwartauer Werke AG, Bad Schwartau, Team Success AG, Selent, Else Kröner Stiftung, Bad Homburg, Hansa Tiefkühlmenü, Hilter, Data input, Frankfurt and Wirtschaftliche Vereinigung Zucker, Bonn, Deutsche Gesellschaft für Gesundes Leben, Bickenbach.

References

1. Thomas PR (ed). *Weighing the Options – Criteria for Weight-Management Programs*. National Academic Press: Washington, 1995.
2. Report of a WHO Consultation on Obesity, Genf, 3–5 June 1996. *Obesity, Preventing and managing a global epidemic*. WHO/NUT/NCD/ 98.1, 1996
3. Jeffery RW. Prevention of obesity. In: Bray G, Bouchard C, James WPT (eds). *Handbook of Obesity*. M. Decker: New York, 1997, pp 819–830.
4. The National Task Force on Prevention and Treatment of Obesity: Research Direction. *Obesity Res* 1994; **2**: 571–584.
5. Puska P, Nissinen A, Tuomilekto I, Salona IT, Koskula K, McAlister A, Kottke TE, Meccoby N, Farquhar IW. The community – based strategy to prevent coronary heart disease: Conclusions from the Ten years of North Carelia Project. *Ann Rev Public Health* 1985; **6**: 147–193.
6. Krauter H, Klaas C, Hoffmeister H, Laaser U. *Prävention von Herzkreislauf Krankheiten*. Gesundheitsforschung JUVENTA: München, 1995.
7. Taylor CB, Fortman SP, Flora J, Kayman S, Barrett DC, Jatulis D, Farghar JW. Effect of long term community health education on body mass index. *Am J Epidemiol* 1991; **134**: 235–249.
8. Jeffrey RW, Fray CW, French SA, Hellerstedt WL, Marunay D, Cuepker RV, Blackburn H. Evaluation of weight reduction in a community intervention for cardiovascular disease risks: change in body mass index in the Minnesota Heart Health Program. *Int J Obes* 1995; **19**: 30–39.
9. Jeffery RW, French SA. Preventing weight gain in adults: design, methods and one year results from the Pound of Prevention Study. *Int J Obes* 1997; **21**: 457–464.
10. Jeffery RW, French JA. Preventing weight gain in adults: design methods and one year results from the pound of prevention study. *Int J Obes* 1997; **21**: 457–464.
11. Hardemann W, Griffin S, Johnston M, Kinmoth AL, Wareham NJ. Interventions to prevent weight gain: a systematic review of psychological models and behaviour change methods. *Int J Obes* 2000; **24**: 131–143.
12. Sallis JE, McKencie TL, Alcarez JE, Kolody B, Hovell MF, Nader PR. Project Spark-Effects of Physical exercise on adiposity in children. In: Williams L, Kimm YS (eds). *Prevention and Treatment of Childhood Obesity* Ann New York Acad Sci: New York, 1993; **299**: 127–136.

13. Walter HJ, Wynder EL. The development, implementation, evaluation and future directions of a chronic disease prevention programme for children: the 'know your body program'. *Prev Med* 1989; **18**: 59–71.
14. MacFarfalone A. Health promotion and children and teenagers. *Br Med J* 1993; **306**: 81.
15. Walter HJ, Hofmann A, Vanghan RD, Wynder E. Modification of risk factors for Coronary Heart Disease. Five year result of a school-based intervention trial. *New Engl J Med* 1988; **318**: 1093–1100.
16. Puska P, Vartiainan E, Pallonan V. The North Carolia Youth Project: Evaluation of two years of intervention on Health Behavior and CVD risk factors among 13–15-years-old children. *Prev Med* 1982; **11**: 550–570.
17. Tell GS, Vellar OD. Non-communicable disease risk factor intervention in Norwegian adolescents: The Oslo youth study. In: Hetzel BS, Berenson. GS (eds). *Cardiovascular Risk Factors in Childhood: Epidemiology and Prevention*. Elsevier: Amsterdam, 1987, pp 203–217.
18. Mannios Y, Moschandreas J, Hatzis C, Kafatos A. Evaluation of a health and nutrition education program in primary school children of Crete over a three-year period. *Prev Med* 1999; **28**: 149–159.
19. Manios Y, Kafatos A, Mamalaks G. The effects of a health education intervention initiated at first grade over a 3 year period: physical activity and fitness indices. *Health Education Res* 1999; **13**: 593–606.
20. Mamalakis G, Kafatos A, Manios Y, Anagnostopoulow T, Apostolaki I. Obesity indices in a cohort of primary school children in Crete: a six year prospective study. *Int J Obes* 2000; **24**: 765–771.
21. Harrell JS, McMurray RG, Bangdicrala SI, Frauman AC, Gansky SA, Bradley CB. Effects of a school-based intervention to reduce cardiovascular risk factors in elemental school children. The Cardiovascular Health Children (CHIC) Study. *J Pediatr* 1996; **128**: 797–805
22. Alexandrov AA, Maslennikova G, Kulikov SM, Propirnji GA, Perrova NV. Primary Prevention of Cardiovascular Disease: 3-Year-Intervention Results in Boys of 12 Years of Age. *Prev Med* 1992; **21**: 53–62.
23. Luepker RV, Perry CL, Mckinlay SM, Nader PR, Parcel GS, Stone EJ, Webber LS, Elder JP, Feldman HA, Johnson CC, Kelder SH, Wu M. Outcomes of a Field Trial to Improve Children's Dietary Patterns and Physical Activity. *JAMA* 1996; **275**: 768–776.
24. Jackson MY, Proulx JM, Pelican S. Obesity prevention. *Am J Clin Nutr* 1991; **53**: 1625S–1630S.
25. Stolley MJ, Fitsgibbon MC. Effects of an obesity prevention program on eating behaviour of African-American mothers and daughters. *Health Education Behav* 1997; **24**: 152–164.
26. Davids S, Gomer Y, Lambert C, Skipper B. Primary prevention of obesity in American Indian children. In: Williams CL, Kimm SYS (eds). *Prevention and Treatment of Childhood Obesity*. New York Academy of Sciences: New York, 1993, pp 167–180.
27. Douelly JE, Jacobsen DJ, Whatley JE, Hill JO, Swift CL, Cherrington A, Polk B, Tran ZV, Reed G. Nutrition and physical activity program to alternate obesity and to promote physical and metabolic fitness in elementary school children. *Obes Res* 1996; **4**: 229–243.
28. Flodmark CE, Ohlsson T, Ryden O, Sveger T. Prevention of progression to severe obesity in a group of obese Schoolchildren treated with family therapy. *Pediatrics* 1993; **91**: 880–884.
29. Davis K, Christoffel KK. Obesity in preschool and school-aged children. treatment early and often may be the best. *Arch Pediatrics Adolescent Medicine* 1994; **148**: 1257–1261.
30. Epstein L, Valoski A, Wing RR, Mccurley J. Ten-year follow-up of behavioral family-based treatment of obese children. *JAMA* 1990; **264**: 2519–2523.
31. Epstein LH, Myers MD, Raynor HA, Saelens BE. Treatment of pediatric Obesity. *Pediatrics* 1998; **101**: 554–570.
32. Epstein LH, Valoski A, Wing RR, McCurley I. Ten year outcomes of behavioral family-based treatment for childhood obesity. *Health Psychol* 1994; **13**: 373–383.
33. Epstein LH, Valoski AM, Vara LS, McCurley I, Wisniewski L, Kalarchin MA, Klein UR, Shraper LR. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children. *Health Psychol* 1995; **14**: 109-115.
34. Pisacano JC, Lichter H, Ritter J, Siegal AP. An attempt at prevention of obesity in infancy. *Pediatrics* 1978; **61**: 360–364.
35. Robinson TN. Reducing children's television viewing to prevent obesity. A randomized controlled trial. *JAMA* 1999; **282**: 1561–1567.
36. Dietz WH. Early influences on body weight regulation. In: Bouchard C, Bray GA (eds). *Regulation of Body Weight-Biological, behavioral mechanisms*. J. Wiley & Sons: Chichester, 1996, pp 149–158.
37. Guo SS, Roche AF, Cameron Chumlea W, Gardner JD, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35y. *Am J Clin Nutr* 1994; **59**: 810–819.
38. Must A, Jacques PF, Dallal GE. Bajema CJ,.Dietz WH. Long-term morbidity and mortality of overweight adolescents. *N Engl J Med* 1992; **327**: 1350–1355.
39. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parenteral obesity. *N Engl J Med* 1997; **337**: 869–873.
40. Parsons TJ, Power C, Logan S, Summerbell CD. Chidhood predictors of adult obesity: a systematic review. *Int J Obes* 1999; **23**(**8**): 1–107.
41. Dietz WH. Health consequences of obesity in youth: Childhood predictors of adult disease. *Pediatrics* 1998; **101**: 518–525.
42. Power C, Lake IU, Cole TJ. Measurement and long-term health risks of child and adolescent fatness. *Int J Obes* 1997; **21**: 507–526.
43. Berenson GS, Srinivasan SR, Bao LN, Newman WP, Tracy RE, Wattigney WA, for the Bogalusa Heart Study. Association between multiple Cardiovascular risk factors and atherosklerosis in children and young adults. *N Engl J Med* 1998; **338**: 1650–1656.
44. Berenson GS, Wattigney WA, Bao W, Srinivasan SR, Radhakrishuamurrthy B. Rationale to study the early natural history of heart disease: The Bogalusa Heart Study. *Am J Med Sci* 1995; **310**(**1**): 22–28.
45. Mahoney LT, Burns TL, Stanford W, Thompson BH, Witt ID, Rost CA, Lauer RM. Coronary risk factors measured in childhood and young adults life are associated with coronary artery calcification in young adults: The Muscatine Study. *J Am Coll Cardiol* 1996; **27**: 277–284.
46. Vanhala M, Vanhala P, Kumpusalo E, Halonon P, Tallala I. Relation between obesity from childhood to adulthood and the metabolic syndrom: population-based study. *BMJ* 1998; **317**: 319–321.
47. Barker DJP, Blundell JE, Dietz WH, Epstein LH, Jeffry RW, Remschmidt H, Rolls BJ, Rössner S, Saris WHM. Group

Report: What Are the Bio-Behavioral Determinants of Body Weight Regulation? In: Bouchasel C, Bray GA (eds). *Regulation of body weight*. J. Wiley & Sons: Chichester, 1996, pp 159–177.
48. Hill IO, Peters IC. Environmental contribution to the obesity epidemic. *Science* 1998; **280**: 1371–1374.
49. Glenny AM, Meara SO, Melville A, Sheldon TA, Wilson C. The treatment and prevention of obesity. a systematic review of the literature. *Int J Obes* 1997; **21**: 715–737.
50. Bouchard C, Perusse L. Genetics of obesity: Family studies. In: Bouchard C (ed). *The Genetics of* Obesity. CRCPress: Boca Raton, 1994, pp 79–92.
51. Ravussin E, Swinburn BA. Metabolic predictors of obesity: cross-sectional versus longitudinal data. *Int J Obes* 1993; **17**(**3**): 28–31.
52. Seidell JC, Muller DC, Sorkin ID, Andres R. Fasting resting exchange ratio and resting metabolic rate as predictors of weight gain: the Baltimore Longitudinal study on aging. *Int J Obes* 1992; **16**: 667–674.
53. Armatruda IM, Statt MC, Welle SC. Total and resting energy expenditure in obese women reduced to ideal body weight. *J Clin Investig* 1993; **92**: 1236–1242.
53a. Katzmartyk PT, Perusse L, Tremblay A, Bouchard C. No association between resting metabolic rate or respiratory exchange ratio and subsequent weight changes in body mass and fatness: $5^1/_2$ year follow up of the Quebec Family Study. *Eur J Clin Nutr* 2000; **54**: 610–614.
54. Müller MJ, Körtzinger I, Mast M, König E. Prävention der Adipositas. *Deutsches Ärzteblatt* 1998; **95**: A2027–A2030.
55. Mast M, Körtzinger I, König E, Müller M. Gender differences in fat mass of 5–7 year old children. *Int J Obes* 1998; **22**: 878–884.
56. Mast M, Körtzinger I, Müller MJ. Nutrition behaviour and status of children in Kiel aged 5–7 years. *Akt Ernährungsmedizin* 1998; **23**: 1–7.
57. Müller MJ, Mast M, Körtzinger I, Grund A, Langnäse K. Physical activity and diet in 5–7 years old children. *Pub Health Nutr* 1999; **2, 3**(**A**): 443–444.
58. Langnäse K, Mast M, Müller MJ. Social status, nutrition and health. *Akt Ernährungsmedizin* 2000; **25**: 16–19.
59. Grund A, Vollbrecht H, Frandsen M, Krause W, Siewers M, Rieckert H, Müller MJ. No effect of gender on different components of daily energy expenditure in free living prepubertal children. *Int J Obes* 2000; **24**: 299–305.
60. Asbeck I, Langnäse K, Mast M, Grund A, Müller MJ. Education in nutrition and family counseling on a local level. *Akt Ernährungsmedizin* 2000; **25**: 33–37.
61. Egger G, Swinburn B. An ecologic approach to one obesity pandemic. *Br Med J* 1997; **315**: 477–480.
62. Swinburn B, Egger G, Razza F. Dissecting obesogenic environments: The development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Prev Med* 1999; **29**: 563–570.
63. Reincken L, Stolley H, Droese W, van Oost G. Longitudinale Körperentwicklung gesunder Kinder II. Größe, Gewicht, Hautfalten von Kindern im Alter von 1. 5 Bis 16 Jahren. *Klin Pädiatr* 1980; **192**: 25–33.
64. Mckinlay JB, Marceau. A tale of 3 tails. *Am J Public Health* 1999; **89**: 295–298.