Body mass index patterns over 5 y in obese children motivated to participate in a 1-y lifestyle intervention: age as a predictor of long-term success1–3

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ABSTRACT
Background: Long-term outcome after lifestyle interventions in obese children is largely unknown but important to improving intervention.
Objective: The aim was to identify predictors of long-term changes in body mass index (BMI) after lifestyle intervention.
Design: Annual changes in the BMI SD score (BMI-SDS) over 5 y in 663 obese children (aged 4–16 y) motivated to participate in an outpatient lifestyle intervention were analyzed. Child-specific longitudinal curves based on multilevel growth curve models (MLMs) over 5 y were estimated depending on patient characteristics (age and sex).
Results: The mean decrease in BMI-SDS was 0.36 (95% CI: 0.33, 0.39) at the end of the 1-y intervention and 0.46 (95% CI: 0.36, 0.55) 4 y after the intervention. Change in BMI-SDS in the intervention period predicted long-term outcome after 5 y (P < 0.001). MLMs identified age but not sex as a predictor of the outcome: the youngest children (<8 y) at the onset of the intervention had the greatest decrease in BMI-SDS over 5 y, and the oldest children (>13 y) had the least decrease in BMI-SDS (P < 0.05). Whereas there was a larger reduction in BMI-SDS during the intervention in children aged 8–10 y than in children aged 11–12 y, long-term decrease in BMI-SDS was greater in 11–12-y-old children (P < 0.001).
Conclusions: Younger age was associated with the best long-term outcome after participation in the lifestyle intervention, which supports the need for early intervention in childhood obesity. Children aged 8–10 y may need modified intervention, because BMI-SDS increased more in the older children in the long term. However, mean BMI-SDS was significantly lower 4 y after the end of the intervention than at baseline in all age groups. This study was registered at clinicaltrials.gov as NCT00435734. Am J Clin Nutr 2010;91:1165–71.

INTRODUCTION
The increasing prevalence of obesity in childhood and adolescence poses an ever-increasing problem (1). Treatment concepts with long-term efficacy are needed (1). Long-term outpatient training programs consisting of a combination of physical exercise, nutrition education, and behavior therapy have been recommended, although few programs have run and have been evaluated (1, 2).

Our previous studies have shown a reduction in body fat and body mass index (BMI) in motivated obese children participating in the 1-y outpatient obesity intervention “Obeldicks” as compared with untreated control groups, both at the end of intervention and 1 y after the end of intervention (3–9). This BMI reduction was sustained for ≥3 y after the intervention (8, 9).

Predictors for long-term success are potentially beneficial, as is the identification of children particularly profiting in the long-term. Such findings might improve the intervention, even for children having characteristics that are associated with a lower likelihood of intervention success. In a previous study we showed that patient characteristics and behaviors, such as regular participation in exercise groups before intervention, were associated with intervention success (10). However, predictors for long-term success in lifestyle interventions are scarce. Therefore, we tried to provide child-specific longitudinal curves of pediatric obese patients over 5 y depending on patient characteristics (age and sex). These curves were based on multilevel growth curve models and can be used to identify obese patients with a worse or better BMI change than their age- and sex-specific average.

SUBJECTS AND METHODS
We examined all obese children motivated to participate in the outpatient intervention “Obeldicks” in 4 different outpatient centers during the years 1999–2006. Inclusion criteria for lifestyle interventions were an age of 4–16 y, sufficient motivation for a lifestyle change, and no BMI reduction in the past 6 mo.

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2 TR and AMT received grant support from the German “Competence Net Obesity,” which is supported by the German Federal Ministry of Education and Research (grant number 01 GI0839). AMT was partially supported by the Munich Center of Health Sciences (LMUinnovativ) subproject II “Evidence Based Prevention and Modelling of Chronic Diseases.” However, the hypothesis development, analysis, interpretation and conclusions contained in this study are those of the author’s alone. MK received grant support from the University of Witten/Herdecke.
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Motivation was proven by the following criteria: 1) the children had to fill out a questionnaire on their eating and exercise habits; 2) the children’s parents had to fill out a corresponding questionnaire on the eating and exercise habits of their child willing to participate; 3) the children or parents, depending on the child’s age, had to complete a 3-d dietary record; and 4) evidence of participation in local exercise groups for overweight children for ≥8 wk before the start of the intervention. Motivation was assumed when all of these criteria were fulfilled independently of the given answers on the questionnaires or of the results of the dietary records. These criteria were used because they predicted success at the end of our lifestyle intervention in a previous study (10). The local ethics committee of the University of Witten/Herdecke approved this study. Written informed consent was obtained from all subjects and their parents before the study started.

The 1-y intervention “Obeldicks” was described in detail elsewhere (7, 8, 10). Briefly, this intervention was based on physical exercise, nutrition education, and behavior therapy, including the individual psychological care of the child and his or her family. The aim was to decrease BMI in the long-term by lifestyle modification. An interdisciplinary team of pediatricians, diet-assistants, psychologists, and exercise physiologists was responsible for the training. The children were divided into groups according to their sex and age. The 1-y training program was divided into 3 phases (Figure 1). In the intensive phase (3 mo), the children took part in a nutritional course and in an eating-behavior course in 6 group sessions, each lasting 1.5 h. At the same time, parents were invited to attend 6 parents’ evening sessions. In the establishing phase (6 mo), individual psychological care was provided based on systemic and solution-focused theories (30 min/mo). All therapists developed a therapeutic alliance with the children and their families. They involved family members, adopted a nonblaming position, assumed motivation, focused on small changes, identified the resources of the family, and aimed to create a positive climate by reframing (for details see reference 11). The last phase of the program lasted 3 mo. In this phase, further individual care was offered by psychologists, if appropriate. The exercise therapy took place once a week throughout the entire year. It consisted of ball games, jogging, trampoline jumping, and instructions in physical exercise as part of everyday life as well as a reduction in the amount of time spent watching television. The nutritional teaching was based on the prevention concept of the “optimized mixed diet.” Per this concept, the present scientific recommendations were translated into food-based dietary guidelines, which also considered the dietary habits of children and families in Germany (7). In contrast with the present-day diet of children in Germany (ie, 38% of energy as fat, 13% of energy as protein, and 49% of energy as carbohydrates, including 14% of energy as sugar), the optimized mixed diet was reduced in both fat and sugar and contained 30% of energy as fat, 15% of energy as protein, and 55% of energy as carbohydrates, including 5% of energy as sugar. The children followed a “traffic-light system” when selecting their food. With this system, the foods and drinks available in Germany were separated according to their fat and sugar contents into “red = stop,” “orange = consider the amount,” and “green = okay when hungry or thirsty.” In a previous study, we showed via 3-d weighed dietary records a significant (P < 0.01) reduction in the mean (± SD) energy content of 1459 ± 379 kcal/d before intervention to a mean of 1250 ± 299 kcal/d at the end of the 1-y intervention and a significant (P < 0.01) reduction in fat from 36.3 ± 5.0% of energy at the onset of intervention to 30.4 ± 7.1% at the end of the 1-y intervention (7).

Children older than 10 y were separated in sex-specific intervention groups, whereas younger boys and girls received the intervention together. In children younger than 8 y, all interventions apart from exercise classes were applied only to parents (9).

After the 1-y lifestyle intervention, no further interventions were performed. The weight status of the children as BMI was...
assessed at onset and at the end of the 1-y lifestyle intervention and annually in the following 4 y after the end of intervention.

Obesity was defined according to the definition of the International Task Force of Obesity (12). The degree of overweight was quantified by using Cole’s least mean square method, which normalized the BMI skewed distribution and expressed BMI as an SD score (BMI-SDS) (13). Reference data for German children were used (14) because the calculation of BMI-SDS is not possible based on the cutoffs of the International Task Force of Obesity (12). The outcome BMI change was analyzed on the basis of a reduction in BMI-SDS, because BMI is sex- and age-dependent in childhood.

The average BMI-SDS values and SEs for each visit were calculated. Multilevel growth curve models were used to estimate the association of age and sex with individual patient BMI-SDS measurements and how these associations vary over time (15). A multilevel growth curve model predicts the average BMI-SDS curve for patients with a given set of characteristics. The models allow BMI-SDS patterns to differ, even among patients with the same baseline characteristics. In addition, the observed outcomes for each patient will vary about their true BMI-SDS curve because of measurement error and day-to-day variation. Technical aspects of the models are described in detail elsewhere (15). A polynomial time function of degree 3 in the BMI-SDS model significantly increased its prediction ($P < 0.001$). Separate multivariable models were calculated for different age groups. Additionally, a multivariable model a priori considering age, sex, and center included all children and explored possible second-order interactions. Inference was based on iterative generalized least squares (IGLS) (16). The same criterion was used to assess whether the effect of each variable on BMI-SDS changed over time, by assessing interactions between the variable and the functions of time. The model describes the relation between BMI-SDS and time since baseline and thus can be used to predict BMI-SDS at any time point up to 5 y after initial presentation at the outpatient clinic.

An additional analysis examined the relation between age, sex, and baseline BMI-SDS and the reduction in BMI-SDS at different time intervals. These analyses also considered children who dropped out or did not complete the whole intervention course. These children were considered to have not changed their BMI-SDS from baseline. Comparisons of BMI-SDS measurements between specific time points were made by using paired $t$ tests. Multilevel models were fitted by using MLwin (17), and all other statistical analyses were performed with SAS version 9.1.3 (SAS Institute Inc, Cary, NC) and R version 2.7.18.

RESULTS

A total of 663 obese children were included in the study (mean age at baseline: 10.6 y; minimum: 4; maximum: 16 y; 55% female). No age differences between female and male individuals were observed ($t$ test, $P = 0.212$). On the basis of information from their family doctors, none of the children had decreased their BMI-SDS $> 0.05$ in the year before intervention.

A total of 116 (18%) children dropped-out during the intervention, of whom 3 children were again seen at later time points. A reduction in BMI-SDS was observed in 470 (71%) children at the end of intervention (intention-to-treat analysis).

BMI-SDS data were available for 663 children at baseline, for 547 after 1 y, for 390 after 2 y, for 294 after 3 y, for 214 after 4 y, and for 183 after 5 y (Table 1). A total of 2292 measurements of BMI-SDS were available out of $6 \times 663 = 3978$ possible (58%). The highest proportion of missing body weight and height information was observed in year 5 after baseline. There were no differences in missing patterns for females and males (eg, completeness up to 3 y; $P = 0.661$), whereas older children tended to have fewer complete cases (eg, completeness up to 3 y; $P = 0.050$). Patients characteristics stratified by age are shown in Table 2.

Mean BMI-SDS at baseline was 2.46 (95% CI: 2.43, 2.50; minimum: 1.90; maximum: 4.16; Table 1). Female and male patients had similar BMI-SDS values at baseline ($t$ test, $P = 0.921$). Mean BMI-SDS values at baseline were similar across different centers (linear regression with center as dummy variable; $P = 0.930$), whereas age was significantly associated with BMI-SDS at baseline with the lowest values observed at the age of 11–12 y and the highest BMI-SDS in children aged 4–7 y (regression with age as dummy variable; $P < 0.001$; Figure 2, Table 2).

The mean reduction in BMI-SDS of all cases with respective measurements compared with baseline was 0.36 (95% CI: 0.33, 0.39) at the end of the intervention. Annual BMI-SDS decreased during the intervention period (paired $t$ test, $P < 0.001$) but did not change between the end of the intervention and 4 y after the end of the intervention (paired $t$ tests for annual comparisons and comparison between end of intervention and 4 y after end of intervention, $P > 0.05$). Five years after baseline (4 y after the end of the intervention) the BMI-SDS had decreased significantly ($P < 0.001$) by a mean 0.46 (95% CI: 0.36, 0.55).

Multilevel growth curve modeling yielded a model containing a polynomial function of degree 3 as most appropriate for time modeling (Table 3). Age was significantly associated with both the height of the BMI-SDS curve and the course of the BMI-SDS curve (all $P < 0.05$; Figure 2, Table 3) even after

<table>
<thead>
<tr>
<th>Measurement time point</th>
<th>No. of participants considered in the model</th>
<th>Lost to follow-up</th>
<th>Dropout in intervention and without measurements in follow-up</th>
<th>Mean</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>663</td>
<td>—</td>
<td>—</td>
<td>2.46</td>
<td>(2.43, 2.50)</td>
</tr>
<tr>
<td>After 1 y</td>
<td>547</td>
<td>93</td>
<td>66</td>
<td>116</td>
<td>2.10  (2.05, 2.15)</td>
</tr>
<tr>
<td>After 2 y</td>
<td>390</td>
<td>143</td>
<td>111</td>
<td>114</td>
<td>2.09  (2.03, 2.15)</td>
</tr>
<tr>
<td>After 3 y</td>
<td>294</td>
<td>238</td>
<td>96</td>
<td>115</td>
<td>2.07  (2.00, 2.14)</td>
</tr>
<tr>
<td>After 4 y</td>
<td>214</td>
<td>252</td>
<td>114</td>
<td>114</td>
<td>2.03  (1.94, 2.12)</td>
</tr>
<tr>
<td>After 5 y</td>
<td>183</td>
<td>—</td>
<td>—</td>
<td>2.00</td>
<td>(1.89, 2.11)</td>
</tr>
</tbody>
</table>

1. Due to failure to reach the measurement time point during the study period.
2. Includes only children participating in the complete intervention course (1 y).

Table 1

Mean BMI-SD scores (BMI-SDS) over time in the BMI-SDS course model development cohort

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adjustment for sex and baseline BMI-SDS. BMI-SDS curves showed a sharp decline under intervention until up to 2 y after intervention depending on individual’s age (Figure 2). However, this decline was followed by a slight increase. The older the children the lesser the BMI-SDS reduction within 2 y (including the minima in each group; Figure 2) after intervention with a reduction of 0.58 BMI-SDS units for young children aged 4–6 y decreasing to 0.25 BMI-SDS units for adolescents aged 13–16 y. The average BMI-SDS did not get back or did not exceed baseline BMI-SDS values in any age group (Figure 2). The higher BMI-SDS reduction in younger children was also observed when analyzing specific time intervals including the intervention period, such as from baseline to end of the intervention, from baseline to 2 y after intervention, and from baseline to 4 y after intervention (reduction of BMI-SDS loss of 0.4 per additional year in all models; all \( P < 0.05 \) adjusted for sex and baseline BMI-SDS).

When considering age as a dummy variable in sex- and baseline BMI-SDS-adjusted multilevel growth curve models, the inverse relation between age and BMI-SDS reduction during intervention was also observed (data not shown). The BMI-SDS reduction was worse among the oldest children aged 13–16 y at baseline (Table 4 and Figure 2). However, when comparing the BMI-SDS reduction between baseline and 4 y after the end of the 1-y intervention, children aged 8–10 y had a lower BMI-SDS reduction than did the older children aged 11–12 y (Table 4 and Figure 2). The children aged 8–10 y increase their BMI-SDS between the end of the intervention and 4 y later in contrast with any other age group (Table 4). In contrast, there was no univariable relation between age and BMI-SDS reduction from the end of the intervention to 2 y after intervention or between 2 y and 4 y after the intervention in linear models, when considering age as a continuous variable (all \( P > 0.05 \)).

Sex was not significantly associated with BMI-SDS or with the height of the patient characteristic curves, nor with the course of the BMI-SDS curve (Table 3). Additionally, in models considering BMI-SDS differences from baseline to the end of the intervention, from baseline to 2 y after intervention, and from baseline to 4 y after the intervention, sex was not associated with the change in BMI-SDS (all \( P > 0.05 \)).

BMI-SDS at baseline was associated with an unadjusted BMI-SDS reduction during the intervention (\( P < 0.001 \)), whereas no association was observed with other time intervals (all \( P > 0.05 \)). Baseline BMI-SDS was inversely associated with BMI-SDS change during intervention (\( -0.09 \) less of a BMI-SDS reduction per additional BMI-SDS unit at baseline; age- and sex-adjusted \( P < 0.01 \)). Change in BMI-SDS in the intervention period predicted long-term outcome after 5 y (0.66 BMI-SDS reduction after 5 y per additional BMI-SDS unit reduction during intervention; age- and sex-adjusted \( P < 0.001 \)).

The dropout in the intervention period did not differ significantly with respect to age, sex, or BMI-SDS at baseline as compared with children finishing the intervention. The children lost to follow-up after the end of the intervention did not differ significantly with respect to age, sex, or BMI-SDS at baseline as well as a change in BMI-SDS in the intervention as compared with the children with complete follow-up.

**DISCUSSION**

This was the first multicenter study to analyze the outcome of BMI-SDS in the course of 5 y in obese children motivated to participate in a 1-y lifestyle intervention with multilevel growth curve modeling, which allowed the identification of the children with the best and worst outcomes.

First, the change in BMI-SDS in the intervention period of 1 y was a strong predictor of long-term BMI-SDS reduction. This finding agrees with the longest on-going obesity intervention study (10 y) that was published showing the first year to be important for a long-term prognosis (19). Second, the reduction in BMI-SDS was independent of sex. Third, the treatment center was not a predictor, which suggested similar results in other centers with different staff performing the intervention. Finally and most importantly, the main predictor of success in the long-term follow-up period after the end of the intervention was age. The youngest children had the highest decrease in BMI-SDS over

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**TABLE 2**

Study characteristics in the different age groups at baseline

<table>
<thead>
<tr>
<th>Age group</th>
<th>4–7 y</th>
<th>8–10 y</th>
<th>11–12 y</th>
<th>13–16 y</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>110</td>
<td>222</td>
<td>184</td>
<td>147</td>
</tr>
<tr>
<td>Sex (%)</td>
<td>42 (32, 51)</td>
<td>43 (37, 50)</td>
<td>51 (44, 58)</td>
<td>43 (35, 51)</td>
</tr>
<tr>
<td>BMI-SD score</td>
<td>2.69 (2.60, 2.78)</td>
<td>2.40 (2.35, 2.45)</td>
<td>2.39 (2.33, 2.44)</td>
<td>2.49 (2.43, 2.56)</td>
</tr>
</tbody>
</table>

/ Mean; 95% CI in parentheses (all such values).
TABLE 3
Different multilevel growth curve models and the corresponding likelihood (L) of BMI-SDS changes during intervention and follow-up.

<table>
<thead>
<tr>
<th>Model</th>
<th>-2 log(L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \beta_{ij} ) (intercept)</td>
<td>2821</td>
</tr>
<tr>
<td>( \beta_{ij} + \beta_{ij} )</td>
<td>2323</td>
</tr>
<tr>
<td>( \beta_{ij} + \beta_{ij} )</td>
<td>2323</td>
</tr>
<tr>
<td>( \beta_{ij} + \beta_{ij} )</td>
<td>1897</td>
</tr>
<tr>
<td>( \beta_{ij} + \beta_{ij} )</td>
<td>1632</td>
</tr>
<tr>
<td>( \beta_{ij} + \beta_{ij} )</td>
<td>1649</td>
</tr>
<tr>
<td>( \beta_{ij} + \beta_{ij} )</td>
<td>1569</td>
</tr>
<tr>
<td>( \beta_{ij} + \beta_{ij} )</td>
<td>1555</td>
</tr>
<tr>
<td>( \beta_{ij} + \beta_{ij} )</td>
<td>1533</td>
</tr>
<tr>
<td>( \beta_{ij} + \beta_{ij} )</td>
<td>1529</td>
</tr>
</tbody>
</table>

The indexes \( i \) and \( j \) indicate the 2 different levels of the multilevel model.

5 y despite a higher BMI-SDS values at baseline, which suggested that early intervention is most promising. The latter finding supports most guideline recommendations, which suggests an early intervention in childhood obesity (2, 20). The predominating application of the intervention to parents in this age group may partially explain the beneficial outcome (21). Furthermore, parents might additionally influence health behaviors in younger children, and health behavior is better strengthened in younger than in older age (21).

However, children aged 8–10 y had a better reduction in BMI-SDS during the intervention than did older children. However, they gained more BMI-SDS in the long-term follow-up period. Entry into puberty after the end of the intervention with all the associated difficulties and conflicts with parents might in part explain the more unfavorable weight course of these children. In contrast, most of the children older than 10 y were already pubertal during the intervention, which allowed a smoothing of the sometimes difficult interactions between parents and adolescents. An adapted intervention in the pubertal period, even after a successful lifestyle intervention in prepubertal children, might improve the long-term outcome in these children.

The achieved BMI-SDS reduction in the entire study population and its maintenance were more pronounced and most importantly sustained as compared with previous studies (2, 20, 22, 23). Apart from differences in age, race, and degree of overweight, this finding can probably be explained in part by proving the motivation at the beginning of the intervention using success criteria (10). Motivation was not proven in previous published studies (20, 23–26). Furthermore, we can only speculate about which components are crucial for long-term success. In contrast with previous published interventions (23–27), we used a combination of individual care and group sessions. Furthermore, the family therapy focusing on the resources of the family and assuming a motivation of the patients might be an important factor leading to long-term success, as also reported in other studies (11, 28).

In addition to identification of predictors of long-term success, the calculated multilevel growth curve models can possibly be used in other centers treating obese children with different interventions. In addition, supplemental components of the intervention can be evaluated against predicted BMI-SDS curves. The multilevel growth curve models are able to assess individual BMI-SDS courses of obese patients, also allowing its prediction. Such patient-specific curves depending on patient characteristics such as age, sex, and baseline BMI-SDS and possibly BMI-SDS reduction under intervention can be used based on our results.

The strengths of the current study were as follows: 1) the longitudinal analysis over a time period of 4 y after the end of the intervention 2); the considerable sample size of obese children after the intervention 3); the wide age range, which allowed a study of the effect of age on outcome 4); a clear definition of motivation 5); the multicenter study design in 4 cities with 4 different treatment teams; and 6) the multilevel analysis approach, which allowed use of the maximum amount of available data and provided flexible estimations regarding nonlinearity and adjustments.

However, some important potential limitations have to be addressed. First, our findings were derived from motivated participants who were already motivated at baseline; thus, the generalizability of our findings might be limited. Furthermore, the higher follow-up rate (95%) used in our study compared with previous studies (20, 26) might have influenced our findings. To make up for the lack of a control group, the BMI reduction in the present study was compared with BMI reductions of other studies (20, 23–26).

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TABLE 4
Mean change in BMI-SD score (BMI-SDS) during the 1-y intervention and between baseline and the 5-y follow-up by age group.

<table>
<thead>
<tr>
<th>Age group</th>
<th>No. of subjects</th>
<th>Change in BMI-SDS during intervention</th>
<th>Change of BMI-SD between baseline and 5 y follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
<td>95% CI</td>
</tr>
<tr>
<td>4–7 y</td>
<td>110</td>
<td>−0.44</td>
<td>(−0.40, −0.48)</td>
</tr>
<tr>
<td>8–10 y</td>
<td>222</td>
<td>−0.37</td>
<td>(−0.32, −0.41)</td>
</tr>
<tr>
<td>11–12 y</td>
<td>184</td>
<td>−0.26</td>
<td>(−0.21, −0.31)</td>
</tr>
<tr>
<td>13–16 y</td>
<td>147</td>
<td>−0.21</td>
<td>(−0.15, −0.27)</td>
</tr>
</tbody>
</table>

Values were derived from multilevel modeling: \( P < 0.001 \) for effects, age effect, and comparison between BMI-SDS change during intervention and baseline to 5 y of follow-up.
children. Therefore, our results cannot be generalized to a general population of obese children, and it is very likely that un-motivated obese children or children recruited on a population-based level without proving their motivation do not have the same long-term success. However, lifestyle interventions against childhood obesity are only recommended in motivated obese children by guidelines (2, 20). Second, we did not perform a randomized controlled trial or include a control group. Therefore, the efficacy of our lifestyle intervention could not be assessed in the current study, and we could not rule out similar changes in BMI-SDS in motivated children not receiving an intervention. However, in previous studies, we showed a failure of BMI-SDS reduction in motivated children defined in the same way as in this study in contrast with motivated obese children receiving the 1-y lifestyle intervention “Obeldicks” (3, 5). Furthermore, the difference in BMI-SDS reduction between the intervention and control groups in previous studies was also significant 1 y after the end of the intervention (4, 6). Additionally, the motivated children in the current study were not able to reduce their BMI-SDS in the months before intervention (see inclusion criteria), and the long-term outcome after the end of the intervention was related to a reduction of BMI-SDS in the intervention. These findings further support the efficacy of our lifestyle intervention. A randomized control trial preventing obese motivated children from intervention for a period of 5 y might be difficult for ethical as well as practical reasons, particularly if there is reasonable evidence of success of the intervention. Third, dropouts may have influenced our findings. However, in a worst-case scenario we considered children who dropped out as not having had success in the intervention, which did not significantly change the results (data not shown). Additionally, in a sensitivity analysis we carried their last observation forward to the measurement points they missed because of dropping-out of the study. However, this approach also yielded similar results (data not shown). Fourth, our findings need to be evaluated in other studies including children of different ethnic backgrounds as well as children with different degrees of overweight. Fifth, BMI-SDS was used to classify outcome. Although BMI is a good measurement of overweight, it has limitations because it is an indirect measure of fat mass, and some behaviors such as physical exercise can change body composition under stable BMI values (29). Furthermore, we lacked data on cardiovascular disease risk factors or other measurements of body composition 5 y after baseline to prove the clinical relevance of the achieved BMI-SDS reduction. However, changes in BMI-SDS have been reported to be closely associated with changes in cardiovascular disease risk factors (30). In previous studies, we were able to show that the BMI-SDS reduction at the end of our intervention and 1 y after the end of our intervention was associated with an improvement in cardiovascular disease risk factors (4, 6).

In summary, BMI-SDS reduction during the lifestyle intervention was associated with a beneficial long-term outcome. Children younger than 8 y at the beginning of the intervention, at which time the intervention was aimed primarily at the parents, had the best long-term success 4 y after the end of the 1-y “Obeldicks” lifestyle intervention for obese children. Children aged 8–10 y at baseline had worse long-term BMI-SDS outcomes than did children who were 11–12 y of age at baseline, whereas children ≥13 y of age had the lowest BMI-SDS reductions. The reasons for these findings remain unclear and require further study. However, the BMI-SDS was significantly lower 4 y after the end of the lifestyle intervention than before the intervention, independently of age.

We are extremely grateful to all the families who took part in this study. The authors’ responsibilities were as follows—TR: performed the study design and prepared the first version of the manuscript; AMT: performed the statistical analysis; and TR, NL, and MK: performed the measurements. All authors participated in the discussion of the findings. None of the authors declared a conflict of interest.

REFERENCES


