

PAPER

Parental overweight, socioeconomic status and high birth weight are the major determinants of overweight and obesity in 5–7 y-old children: baseline data of the Kiel Obesity Prevention Study (KOPS)

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OBJECTIVES: To identify the major risk factors of overweight and obesity in prepubertal children.

DESIGN: Cross-sectional study.

SETTING: In all, 32 primary schools in Kiel (248 000 inhabitants), northwest Germany.

SUBJECTS: A total of 2631 5–7-y-old German children and their parents.

MAIN OUTCOME MEASURES: Weight status, socio-economic status (SES), parental overweight, dietary intake, activity, inactivity and further determinants (birth weight, breast feeding, nutritional status of siblings) of the children.

RESULTS: The prevalence of overweight (≥ 90 th BMI percentile of reference) was 9.2% in boys and 11.2% in girls, respectively. Considered univariately, family-, environment- and development-related determinants showed some relations to overweight and obesity. In multivariate analyses parental overweight, a low SES as well as a high birth weight were the strongest independent risk factors of overweight and obesity in children. Additionally, there were sex-specific risk factors: parental smoking and single households were risk factors in boys, whereas a low activity was associated with obesity in girls. Birth weight was associated with obesity, but not with overweight. The prevalence of obesity reached 29.2% in boys and 33.4% in girls with all the three main risk factors.

CONCLUSIONS: Overweight families of low SES have the highest risk of overweight and obese children. Future prevention programmes must also take into account sex-specific risk factors.

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Introduction

The prevalence of overweight and obesity in children is rising in Europe.^{1,2} Specific periods for the development of overweight and obesity have been identified in children. These include the prenatal period, the period between the ages 5 and 7 (so called adiposity rebound) and adolescence.³ Although most people become obese as adults, there is a significant association between BMI in childhood or adolescence and in adults,^{4–6} (for a recent review, see Parsons *et al*⁷). Persistence of obesity into adulthood appeared to rise

linearly throughout childhood. In addition, childhood overweight was shown to be predictive for adult morbidity and mortality.⁸ Antecedents of adult disease (hypertension, hyperlipidemia, abnormal glucose tolerance) occur with increased frequencies in obese children and adolescents.^{9–13} However, there are only few long-term follow-up data spanning the childhood to adulthood period. These data suggest life-long persistence and health consequences of overweight and obesity in many children,⁸ suggesting a strong need for prevention.

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.³ These risk factors are related but their relationship is unknown at the individual as well as at the population level. Although most risk factors for early-onset

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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 to the development of childhood obesity, remain unclear with respect to a given individual as well as with respect to a greater populations. Despite a tremendous amount of research, the respective contributions of dietary intake, patterns of activity, sedentary behaviours and familiar factors present a 'confused' picture.²

Obesity is a complex multifactorial trait. Environmental factors favouring overweight and obesity include a high fat and energy intake, a low level of habitual physical activity as well as frequent inactive behaviours. Today, less than 50% of the children engage in routine physical activities.^{2,14,15} Concomitantly, sedentary activities (eg TV viewing) have markedly increased.^{2,14,16,17} As many as 45% of the children snack caloric-dense foods at least twice a day.^{2,14,18} These data provide some evidence for the contributions of environmental factors to overweight and obesity. However, the within-subject associations of health-related behaviours as well as the ranking of determinants of childhood overweight are unclear.

The Kiel Obesity Prevention Study (KOPS) was started in 1996 and is planned to run until the year 2009.¹⁹ The aims of KOPS are three-fold. First, to describe the prevalence and incidence of overweight and obesity in children and adolescents. Second, to characterise the determinants and risk factors of childhood overweight. Third, to assess the long-term effect of a low-level school- and family-based intervention. Faced with the problems mentioned above, we have used cross-sectional data of the first KOPS cohort T0 to analyse the determinants of overweight in prepubertal children (ie second aim).

Methods

Subjects

The KOPS was started in 1996. The study design and the nomenclature of the KOPS cohorts and recruitment of the children are described in another paper.¹⁹ Between 1996 and 2001, 4997 5–7-y-old children (2503 boys, 2494 girls) were recruited (KOPS cohort T0). These are 41% of all children who entered the school-entry examinations in this time period. A questionnaire about family-, environment- and development-related determinants was offered to all parents of KOPS cohort T0. In all, 2631 parents (of 1301 boys and 1330 girls, ie 53%) completed the questionnaire (KOPS subcohort T0). Cohort T0 as well as the subcohort T0 were representative when compared with the total population of 5–7-y-old children ($n = 12\,254$) investigated by the school physicians during this time period.²⁰ This paper analyses the data of KOPS subcohort T0.

Nutritional status of the children

Measurements of the weight status are described in detail previously.¹⁹ Briefly, the body mass index (BMI) was

calculated from measured height and weight (weight (kg)/height² (m²)). The weight status of the children was classified according to German BMI percentiles:²¹ ≤ 10 th percentile = underweight, > 10 th– < 90 th percentile = normal weight, ≥ 90 th–97th percentile = overweight, > 97 th percentile = obese.

Triceps (TSF), biceps (BSF), subscapular (SSF) and supra-iliac (SIF) skinfolds were measured and a bioelectric impedance analysis was accomplished. To assess the fat mass of the children, the sum of four skinfolds was used in the one hand; on the other hand, the fat mass was calculated with an algorithm derived from BIA, which was developed in Kiel.²²

Determinants related to family, environment and development of the children

Possible risk factors for childhood obesity were collected using a questionnaire addressing the following determinants:

Family data:

- Nutritional status of mothers and fathers: self-reported height and weight; classified in categories according to BMI:²³ 18.5–24.9 kg/m² = normal weight, 25–29.9 kg/m² = overweight, > 30 kg/m² = obese.
- Nutritional status of siblings: reported weight and height by parents; classified in categories according to German BMI percentiles:²¹ ≤ 10 th percentile = underweight, > 10 th– < 90 th percentile = normal weight, ≥ 90 th–97th percentile = overweight, > 97 th percentile = obese.
- Smoking habits of parents: classified in categories: 0, 1–15, > 15 cigarettes/day.

Social data:

- Socio-economic status (SES) according to parental education—highest level attained by either parent: 'low' = 9 school years, 'middle' = 10 school years, 'high' = 12 school years or more.
- Single parenthood: dichotomous (yes/no).
- Nationality: dichotomous (German vs non-German).

Infancy:

- Birth weight: continuous in kilograms from well-baby-check-up book measurements; classified in categories using cutoff points from German percentiles (10th and 90th, Danielzik²⁰) taking sex and duration of pregnancy into account.
- Breast feeding: classified in categories: 0– < 1 , 1– < 3 months, 3– < 6 , > 6 months (self-reported by parents).

Physical activity/inactivity:

- Regular sport activities in a club: classified in categories: 0, 0– < 1 , 1–2, > 2 h/week.
- Media time: watching TV or playing computer games; classified in categories: 0, 0– < 1 , 1–2, > 2 h/day.

Nutrition:

- Food frequency with 26 items: we chose six items (three 'healthy' and three 'risk-related' foods) to specify the consumption in children with different nutritional status: fruits, vegetables, wholemeal bread, soft drinks, chips and sweets, classified in categories: never, 1–2, 3–5 times/week, daily healthy eating index (HEI):¹⁸ calculated from all items of the food frequency questionnaire (FFQ); each item was evaluated by a three-stage scale according to German recommendations for nutrition in children.²⁴ HEI was classified in categories according to tertiles: ≥ 34 points = 3rd tertile, 30–33 points = 2nd tertile, ≤ 29 points = 1st tertile (for the multivariate analyses). Based on scientific recommendations, the following classification was used: > 46 –52 points = very good, > 40 –46 points = good, > 30 –40 points = satisfactory, < 30 points = poor.
- The FFQ was validated against a 7-day diet record in 24 children.²⁵ Additionally, differences in HEI were analysed when either parents or children completed the FFQ. There were nonsystematic differences in several food items, that is, healthy as well as unhealthy foods were over- and underestimated by children. We took parental data as more plausible when compared to data of 5–7-y-old children.

Statistical analysis

The statistical analysis was performed with SPSS 10.0 for Windows (SPSS Inc., Chicago, IL, USA). Results were presented as the median and interquartile range (IQR). The nonparametric Mann–Whitney *U*-test was used to determine gender differences. The nonparametric Kruskal–Wallis test was performed to analyse differences in weight status of the children with respect to continuous variables. The χ^2 test compared the prevalences of categorical variables.

Multivariate logistic regression analyses were performed to identify independent risk factors of overweight or obesity. Separate analyses were performed with regard to sex, overweight and obesity. The weight status of the children (normal weight–overweight and normal weight–obese, respectively) was used as dependent variable, all family-, environment- and development-related determinants were independent variables. Categorical variables were converted in dichotomous dummy variables. The 'best' category was used as reference in each variable.

Level of significance was set at $P < 0.05$.

Results**Family-, environment- and development-related determinants**

Table 1 shows the characteristics of the children of KOPS. Boys were heavier and taller than girls, but girls had a higher fat mass than boys. Table 2 demonstrates family-, environ-

Table 1 Characterisation of the KOPS study population (median/IQR) of 5–7-y-old children

	KOPS population		
	All	Boys	Girls
<i>n</i>	2631	1301	1330
Age (years)	6.2 (5.9–6.5)	6.2* (6.0–6.5)	6.2* (5.9–6.5)
Weight (kg)	22.1 (20.4–24.6)	22.5* (20.7–24.7)	22.0* (20.0–24.5)
Height (m)	1.20 (1.16–1.24)	1.21* (1.17–1.24)	1.19* (1.16–1.23)
BMI (kg/m ²)	15.5 (14.6–16.5)	15.5 (14.7–16.4)	15.5 (14.5–16.6)
$\Sigma 4$ SF (mm)	28.3 (23.0–35.3)	26.3* (22.0–32.3)	30.2* (24.7–38.3)
FM (%)	28.4 (25.2–31.9)	27.2* (24.1–30.1)	29.7* (26.3–33.2)
Obese (%)	4.0	3.7	4.4
Overweight (%)	6.1	5.5	6.8
Normal weight (%)	82.8	83.5	82.1
Under weight (%)	7.1	7.4	6.8

$\Sigma 4$ SF = sum of 4 skinfolds (TSF+BSF+SSF+SIF), FM = fat mass. *Significant differences between boys and girls (Mann–Whitney *U*-test, $P < 0.05$).

ment- and development-related factors of children of KOPS with different weight status. Overweight and obesity of the children were associated with overweight and obesity in mothers, fathers and siblings. Parents of obese children smoked more cigarettes per day than parents of leaner children. There was an inverse gradient in SES: when compared with high SES, children from low SES families were more frequently overweight. In addition, overweight and obese children were more rarely breast fed than normal weight children. When compared to normal weight children, overweight and obese children frequently had a higher birth weight. There were some sex differences: 24% of overweight girls had an obese mother compared to 10% of overweight boys. The SES distribution was different between overweight and obese boys and girls: 42% of overweight girls belonged to low SES when compared to 24% of overweight boys. The corresponding numbers for obese children were 34% in girls and 55% in boys, respectively. When compared with normal weight boys, normal weight girls were more often breast fed for 1–3 months. There were also sex differences in the association between low birth weight and normal weight. The prevalence of normal weight girls with low birth weight was doubled compared to normal weight boys with low birth weight (5.1 and 2.6%, respectively).

Table 3 shows the physical activity and inactivity habits of the children. While normal- and overweight children showed comparable activities, obese boys and girls were less active. Regarding media times, obese and overweight children had a similar behaviour but in both groups media times were higher when compared with normal weight children. Food intake was different between normal weight and obese children (Table 4): fruit and vegetables were consumed less frequently in obese children than in non-obese pupils. By contrast, obese children drank more frequently lemonades and ate more frequently chips than normal weight children. The intake of sweets was lower in

Table 2 Family-, environment- and development-related determinants of children in the KOPS study population of 5–7-y-old children stratified by weight status^a

	All	Underweight	Normal weight	Overweight	Obese
<i>n</i>	2638	186	2178	161	106
Mother					
Underweight (%)	4.2	8.2 [#]	4.3 [#]	0 [#]	2.0 [#]
Normal weight (%)	66.5	74.8 [#]	69.4 [#]	48.9 [#]	35.0 [#]
Overweight (%)	20.2	14.1 [#]	18.8 [#]	33.6 [#]	30.7 [#]
Obese (%)	9.1	0 [#]	7.4 [#]	17.6 [#]	32.4 [#]
Father					
Underweight (%)	0.6	1.2 [#]	0.6 [#]	0 [#]	0 [#]
Normal weight (%)	52.0	59.9 [#]	54.0 [#]	34.6 [#]	33.9 [#]
Overweight (%)	39.3	36.4 [#]	38.4 [#]	54.4 [#]	36.9 [#]
Obese (%)	8.1	2.5 [#]	7.0 [#]	11.0 [#]	29.1 [#]
Siblings					
No (%)	33.8	34.3 [#]	33.8 [#]	33.0 [#]	34.2 [#]
At least one underweight (%)	19.6	29.8 [#]	20.1 [#]	14.2 [#]	6.1 [#]
All normal weight (%)	17.9	7.3 [#]	16.4 [#]	27.5 [#]	40.8 [#]
At least one overweight/obese (%)	62.4	62.1 [#]	63.4 [#]	58.3 [#]	53.1 [#]
Smoker (parents)					
No (%)	43.6	44.0	44.4	40.5	33.5
1–15 cig./day (%)	18.5	16.5	18.8	20.2	13.3
> 15 cig./day (%)	37.9	39.5	36.8	39.3	53.2
SES					
Low (%)	23.9	22.0 [#]	21.8 [#]	33.5 [#]	45.2 [#]
Middle (%)	30.0	33.9 [#]	29.4 [#]	36.4 [#]	26.6 [#]
High (%)	46.1	44.0 [#]	48.8 [#]	30.1 [#]	28.2 [#]
Single parenthood					
No (%)	79.1	85.2	79.3	76.2	71.2
Yes (%)	20.8	14.8	20.6	23.8	28.8
Breast feeding					
0 month (%)	14.5	15.9 [#]	13.5 [#]	18.9 [#]	22.8 [#]
1–3 month (%)	25.2	26.9 [#]	24.8 [#]	25.1 [#]	28.9 [#]
3–6 month (%)	29.6	28.1 [#]	30.0 [#]	25.1 [#]	31.7 [#]
> 6 month (%)	30.6	29.1 [#]	31.7 [#]	30.9 [#]	16.7 [#]
Birth weight					
Low (%)	4.5	12.8 [#]	3.9 [#]	3.3 [#]	4.3 [#]
Middle (%)	79.8	83.9 [#]	80.8 [#]	77.1 [#]	65.0 [#]
High (%)	15.7	3.2 [#]	15.3 [#]	19.6 [#]	30.7 [#]

^aAccording to BMI percentiles of Kromeyer-Hauschild *et al* (2001), [#]Significant differences between weight status of the children (χ^2 test, $P < 0.05$).

obese pupils. Nevertheless, diet quality as measured by the HEI did not differ between children with different weight status. Sex differences were seen in normal weight children: when compared with boys girls had a higher intake of vegetable but had a lower consumption of soft drinks. The consumption of sweets was greater in obese boys than in obese girls. There was a sex effect on the distribution of the HEI in obese children: obese girls were more often in the third tertile than obese boys.

Figure 1 shows the distribution of the HEI in the total population of children. No child reached a very good HEI

Table 3 Physical activity and inactivity of children in the KOPS study population stratified by weight status^a

	All	Underweight	Normal weight	Overweight	Obese
<i>n</i>	2638	186	2178	161	106
Activity					
0 h/week (%)	43.7	46.5 [#]	42.0 [#]	43.9 [#]	64.2 [#]
0–<1 h/week (%)	22.7	19.6 [#]	23.5 [#]	19.9 [#]	18.0 [#]
1–<2 h/week (%)	21.1	23.9 [#]	21.7 [#]	19.3 [#]	12.7 [#]
> 2 h/week (%)	12.4	10.0 [#]	12.8 [#]	16.9 [#]	5.0 [#]
Inactivity					
0 h/day (%)	4.4	5.2 [#]	4.7 [#]	2.6 [#]	1.2 [#]
0–<1 h/day (%)	54.8	61.4 [#]	56.8 [#]	41.4 [#]	33.3 [#]
1–< 2 h/day (%)	29.8	20.1 [#]	29.3 [#]	36.8 [#]	40.7 [#]
> 2 h/day (%)	11.0	13.2 [#]	9.2 [#]	19.2 [#]	24.9 [#]

^aAccording to BMI percentiles of Kromeyer-Hauschild *et al*,²¹ [#]Significant differences between weight status of the children (χ^2 test, $P < 0.05$).

and only 3% were characterised as 'good'. In all, 62% of the children had a satisfactory eating behaviour and 35% had a poor nutrition. Comparing the distribution of BMI with that of the HEI, it becomes evident that the HEI did not differ between children with different weight status. Children from each BMI group could reach a high HEI as well as a low HEI (Figure 1). Regarding groups of children with (i) different HEI, (ii) different physical activity level and (iii) different media times, the median of BMI only differed significantly with respect to the degree of inactivity (Figure 2). A high degree of inactivity was associated with a higher BMI. There was no association between BMI and either HEI or the degree of activity.

Identification of independent risk factors (multivariate analysis)

To analyse which determinants have the strongest influence on the manifestation of overweight and obesity in prepubertal children, multivariate logistic regression analyses were accomplished considering sex as well as overweight and obesity. Table 5 reports significant variables of the logistic regression analyses. In all the four analyses, parental obesity was a strong predictor for developing overweight and obesity in childhood. A low SES was also found to be a strong risk factor in the three analyses. Birth weight was associated with obesity but not with overweight. There were sex-specific differences: in boys the risk of overweight and obesity was increased in groups of children from smokers and single households. In girls a low activity was also a risk factor. While in boys a low birth weight was associated with obesity, in girls a high birth weight increased the risk of obesity.

Children with combinations of risk factors ('high-risk children')

Table 6 shows the weight status as well as the prevalence of overweight and obesity in children, with a cluster of main

Table 4 Nutrition habits of children in the KOPS study population stratified by weight status^a

	All	Underweight	Normal weight	Overweight	Obese
<i>n</i>	2638	186	2178	161	106
Fruit					
Daily (%)	67.9	70.3 [#]	68.0 [#]	67.0 [#]	64.8 [#]
3–5 × /week (%)	26.6	21.9 [#]	26.9 [#]	31.1 [#]	24.0 [#]
1–2 × /week (%)	4.4	4.6 [#]	4.1 [#]	1.9 [#]	11.2 [#]
Never (%)	1.1	3.2 [#]	1.1 [#]	0 [#]	0 [#]
Vegetable					
Daily (%)	45.8	39.8 [#]	46.7 [#]	48.5 [#]	36.3 [#]
3–5 × /week (%)	42.7	45.6 [#]	42.6 [#]	43.2 [#]	39.9 [#]
1–2 × /week (%)	8.2	9.6 [#]	7.9 [#]	3.8 [#]	16.2 [#]
Never (%)	3.3	5.0 [#]	2.8 [#]	4.4 [#]	7.5 [#]
White bread					
Daily (%)	41.7	40.5	42.1	42.8	33.0
3–5 × /week (%)	34.4	30.1	34.6	35.5	36.0
1–2 × /week (%)	12.4	14.5	12.2	11.8	14.0
Never (%)	11.5	15.0	11.1	9.9	17.0
Soft drinks					
Daily (%)	4.1	3.4 [#]	3.8 [#]	4.6 [#]	11.8 [#]
3–5 × /week (%)	11.0	18.4 [#]	9.9 [#]	11.8 [#]	18.6 [#]
1–2 × /week (%)	17.7	15.6 [#]	17.6 [#]	22.9 [#]	15.7 [#]
Never (%)	67.2	62.6 [#]	68.6 [#]	60.8 [#]	53.9 [#]
Chips					
Daily (%)	2.0	3.9 [#]	1.8 [#]	1.9 [#]	2.9 [#]
3–5 × /week (%)	14.6	19.6 [#]	13.6 [#]	17.9 [#]	21.6 [#]
1–2 × /week (%)	36.2	33.5 [#]	37.1 [#]	34.0 [#]	25.5 [#]
Never (%)	47.2	43.0 [#]	47.5 [#]	46.2 [#]	50.0 [#]
Sweets					
Daily (%)	24.1	23.5 [#]	24.8 [#]	19.2 [#]	18.6 [#]
3–5 × /week (%)	55.5	56.4 [#]	55.5 [#]	52.6 [#]	58.8 [#]
1–2 × /week (%)	16.1	17.3 [#]	15.7 [#]	23.1 [#]	12.7 [#]
Never (%)	4.2	2.8 [#]	4.0 [#]	5.1 [#]	9.8
HEI					
Median	31 (28–34)	30 (27–33)	31 (28–34)	31 (28–34)	32 (28–34)
3rd tertile (%)	30.7	24.1	31.5	27.9	28.4
2nd tertile (%)	33.9	30.9	33.5	40.1	37.4
1st tertile (%)	35.4	45.0	35.0	32.0	34.2

^aAccording to BMI percentiles of Kromeyer-Hauschild *et al.*²¹ wm: whole meal.

[#]Significant differences between weight status of the children (χ^2 test, $P < 0.05$).

risk factors identified in the multivariate analysis (parental overweight, low SES and high birth weight) and their combinations. Children with two risk factors had a higher BMI, higher skinfold thickness and higher fat mass when compared with children with one risk factor. Combining all the three risk factors resulted in the highest prevalence of overweight and obesity (29.2% in boys and 33.4% in girls).

Discussion

The major determinants of overweight and obesity of 5–7-year-old children are parental overweight, a low SES and a high

birth weight (Table 5). There are very few studies which analyse the impact of multiple risk factors on the prevalence of obesity in childhood. Many studies focussed on one risk factor which was controlled for confounder variables. For example, von Kries *et al.*²⁶ analysed the impact of maternal smoking during pregnancy on the prevalence of overweight in 5–7-year-old German children. In accordance to our study, von Kries *et al.* identified obesity of the parents, a high birth weight and a low SES as significant risk factors; additionally, TV watching and smoking during pregnancy influenced the risk of childhood overweight. In a multivariate analysis Strauss and Knight,²⁷ Fogelholm *et al.*,²⁸ Maffei *et al.*,²⁹ as well as Hediger *et al.*,³⁰ also identified parental overweight and obesity as the strongest determinants of childhood overweight.

In our study, the multivariate analysis was stratified according to gender as well as overweight and obesity. In fact different determinants were identified as significant risk factors of childhood overweight and obesity, respectively, and between gender (Table 5): parental smoking and single households were risk factors for boys, whereas a low activity was associated with obesity in girls. In addition, birth weight was associated with obesity, but not with overweight. These between-group differences were neglected in most of the other studies. Our data may have an impact for future programmes on prevention of childhood overweight and obesity. We recommend that prevention programmes besides the major determinants have also to focus on the degree of overweight as well as on sex-specific risk factors.

Parental overweight is a strong risk factor for childhood obesity. This aspect includes genetic as well as behavioural factors which are difficult to separate in a field study. Data from twin, adoption and family studies suggest that genetic factors may account for 25–90% of inter-individual differences in fat mass.³¹ Familial patterns of obesity may also be explained by familial similarities in eating patterns, dietary composition and physical activity.^{32,33} Vogler *et al.*³⁴ proposed that most of the familial risk is likely to be explained by genetic factors, whereas only a minor effect is due to shared environment. In addition, gene–environment interactions are likely to account for the dramatic increase in the prevalence of childhood overweight in the last few years.³⁵

As already shown in other studies,⁷ we found a higher prevalence of overweight in children from low social class when compared with children from high SES^{36,37} (Table 2). The inverse social gradient was still significant after controlling for weight status of the parents. In our study, we defined the SES according to the education of the parents. Education is only one social determinant of health. Comparing different variables used to characterise SES (eg employment, family size, income, marital status, social circumstances), Langnäse showed that the different categories overlap but that parental education is the best marker to characterise the association between SES and overweight in children.³⁸ The impact of social inequalities in health must be a goal for future research.

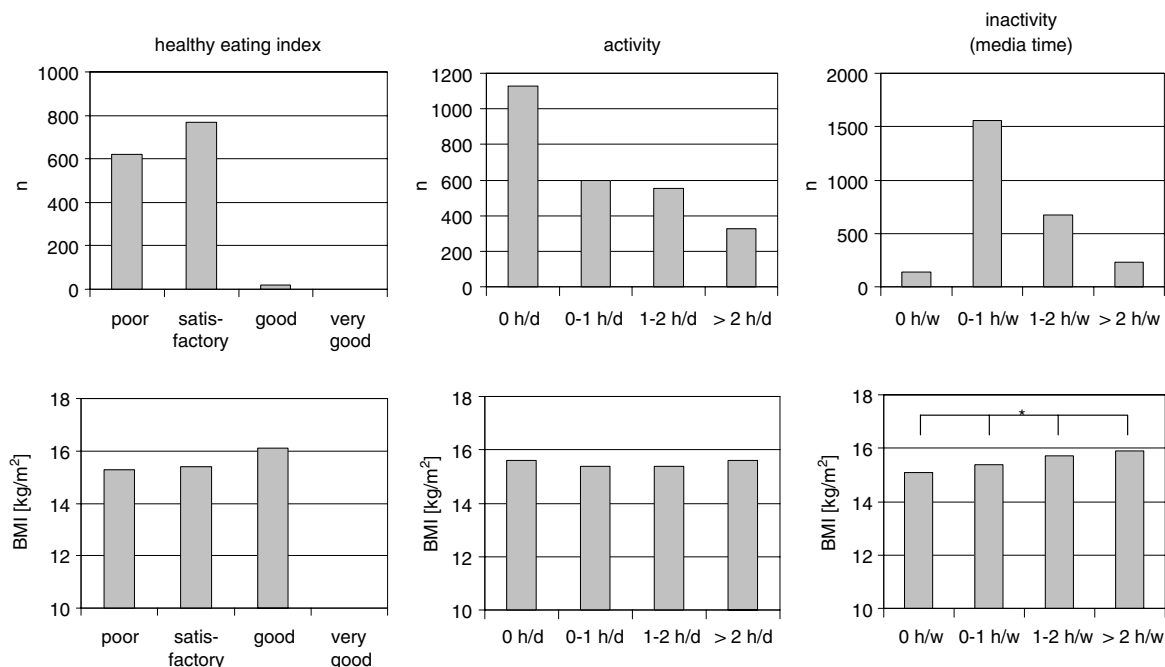


Figure 1 Different levels of HEI, activity and inactivity and corresponding median of BMI within the KOPS study population. *Significant differences in median of BMI (Kruskal–Wallis test, $P < 0.05$).

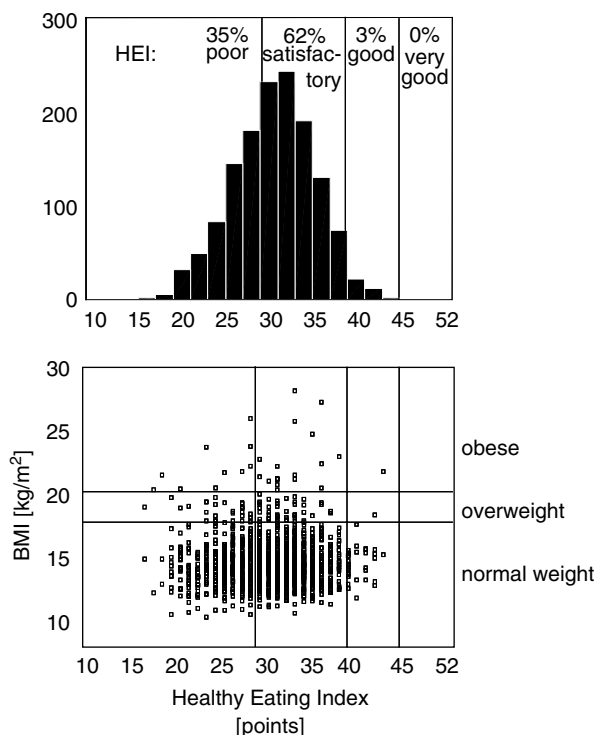


Figure 2 Distribution of HEI and corresponding distribution of BMI within the KOPS study population. HEI: 46–52 points=very good, 40–45 points=good, 30–39 points=satisfactory, < 30 points=poor (distribution in percent is marked in the upper figure).

Table 5 Results of logistic regression analyses

Dependent variable	Independent variables	OR	95% CI	P	Explained variance (%) ^a	
Boys	Overweight	Single parenthood	3.4	1.7–7.1	0.0009	33
		Obese parents	2.9	1.4–6.0	0.0039	
		Smoker	2.6	1.1–6.3	0.0371	
		Overweight parents	2.5	1.4–4.5	0.0024	
		SES	9.8	1.8–53.1	0.0080	
	Obesity	Parental smoking	16.2	2.1–121.5	0.0069	
	Low birth weight	13.7	1.8–105.7	0.0121		
	Low SES	9.3	1.6–51.9	0.0114		
	Obese parents	8.8	2.5–30.6	0.0006		
	Single parenthood	7.5	1.8–31.3	0.0054		
Girls	Overweight	Obese parents	4.5	2.3–9.0	0.0000	28
		Low SES	2.1	1.1–4.2	0.0381	
		Overweight parents	2.1	1.2–3.8	0.0084	
		1–3 m breastfed	0.4	0.2–0.9	0.0242	
Obesity	Low activity	8.9	1.7–46.6	0.0095	31	
	Obese parents	6.2	2.2–18.0	0.0007		
	High birth weight	3.2	1.3–7.8	0.0110		

^aNagelkerke R^2 .

Table 6 Nutritional status (median/IQR) and prevalence of overweight (ow) of children with different risk factors of overweight

	n	BMI (kg/m ²)	4 SF (mm)	FM (%)	Prevalence of	
					Overweight (%)	Obesity (%)
<i>Boys</i>						
Parental ow	699	15.7 (14.9–16.8)	27.7 (22.9–34.7)	27.5 (24.2–30.7)	7.9	5.6
Low SES	286	15.7 (14.8–16.6)	27.9 (23.0–34.0)	27.3 (23.8–30.8)	5.6	8.4
High birth weight	183	16.0 (15.0–17.2)	28.3 (24.0–36.3)	28.5 (25.0–32.0)	7.1	8.7
Parental ow+low SES	183	15.8 (14.9–17.2)	28.7 (23.1–36.3)	27.6 (24.1–31.4)	7.7	11.5
Low SES+high birth weight	32	16.0 (14.9–18.9)	28.5 (24.1–49.3)	29.6 (25.0–33.8)	6.3	21.9
Parental ow+high birth weight	123	16.0 (15.0–17.2)	28.7 (24.0–37.3)	28.7 (25.1–32.3)	7.3	9.8
Parental ow+low SES+high birth weight	24	16.3 (14.9–19.3)	28.2 (24.4–49.3)	28.9 (25.0–33.8)	4.2	25.0
<i>Girls</i>						
Parental ow	739	15.8 (14.6–17.1)	31.7 (25.7–40.8)	29.9 (26.7–34.0)	10.0	6.5
Low SES	313	15.9 (14.6–17.2)	31.9 (25.4–43.4)	30.0 (26.9–34.2)	11.8	6.1
High birth weight	178	16.1 (15.2–17.1)	30.5 (25.9–38.8)	31.0 (27.4–33.9)	9.0	6.7
Parental ow+low SES	213	16.1 (14.7–17.5)	32.3 (26.3–45.3)	30.2 (27.0–34.9)	14.6	7.5
Low SES+high birth weight	40	16.3 (15.5–18.5)	35.8 (25.8–50.6)	31.7 (26.9–37.2)	15.0	12.5
Parental ow+high birth weight	110	16.3 (16.3–17.7)	32.3 (27.4–43.4)	31.8 (28.1–35.1)	12.7	10.0
Parental ow+low SES+high birth weight	30	16.4 (15.7–19.2)	35.8 (26.2–54.4)	31.2 (25.9–38.3)	16.7	16.7

Σ4 SF = sum of 4 skinfolds (TSF+BSF+SSF+SIF), FM = fat mass, ow = overweight.

Birth weight has an association with the weight status of prepubertal children. Children who were overweight at the age of 5–7y often had had already a high birth weight (Table 2). This idea was supported by the authors of a recent review.⁷ However, our multivariate analyses showed that a low birth weight was also associated with obesity in boys. This is contrary to previous data published in the literature. Some authors found the same association,³⁸ whereas others could show that a low birth weight is only a risk factor in children who had a so called 'catch-up growth' (ie a high weight gain within the first 2y of life).^{39,40} These data suggest that birth weight can serve as an early characterisation of a risk group for the development of overweight and obesity in childhood.

Parental overweight, a low SES and a high birth weight were independent risk factors for childhood overweight (Table 5). The combination of these risk factors resulted in the highest risk of overweight in 5–7-y-old children (Table 6). We therefore recommend to address preventive measures to 'high-risk children' and families, respectively. It is likely that universal prevention must be combined with selective prevention to effectively tackle overweight as well as obese children. That is why KOPS follows two independent intervention strategies: universal prevention at schools plus selective prevention within overweight families.¹⁹

When compared with parental overweight and a low SES, our cross-sectional data suggest that lifestyle variables like nutrition, activity and inactivity seem to play a minor role for the development of childhood overweight (Figure 2). Although there were some between-group differences in the intake of several foods, the HEI (ie an index of health-related nutrition quality) did not differ between children with different weight status (Table 4, Figure 1). This agrees with

other studies that showed no differences in nutritional habits between overweight and normal weight children.⁴¹ Our results are comparable with the results of studies using a semiquantitative food frequency or a 3-day diet diary and therefore could analyse the macronutrients in longitudinal studies.^{42,43} These authors also found no association between the intake of single macronutrients and body fat mass.^{42,43} By contrast, other studies found a significant but weak association between macronutrient intake and weight status in children, explaining only 5% of the variation in body fatness by fat intake.⁴⁴

Contrary to food intake, differences in physical activity and inactivity were observed between overweight and normal weight children (Table 2). Based on our cross-sectional data, the influence of inactivity seemed to be stronger than that of activity (Figure 2). This result is in line with the results of other authors.^{45,46} Both Epstein *et al*⁴⁷ and Robinson *et al*⁴⁸ could show that a reduction of inactivity had an effect on the decrease of prevalence of childhood overweight. It should be mentioned that the parameter of inactivity used in this study (ie media time) may not reflect reduced energy expenditure. Grund *et al*⁴⁹ compared media time with 24-h heart rate monitoring (as a measure of 24-h energy expenditure) but did not find any association. This and our present data suggested that physical activity and inactivity differently contributed to childhood obesity. Anyhow, all these data provide strong evidence for the idea that reducing television viewing and promoting outdoor playing is a strategy to tackle the obesity epidemic in young children.⁵⁰

Some limitations of our study should be discussed. Self-reported height and weight were used to calculate BMI of parents and siblings. This may result in some inaccuracies.

However, we have tried to correct the BMI by adding 1.62 units of BMI (recommended by Wardle *et al*⁵¹). Using this approach does not affect the between-group differences observed in our study.⁵²

Owing to the cross-sectional nature of our study design, it is difficult to find out whether the differences in determinants precede or follow changes in fatness. Therefore, prospective studies (with long-term follow-up) have to analyse data of 'incident' children, for example, children who become overweight or obese during the study period. Within the next few years, we will have these data because KOPS has a cross-sectional as well as a longitudinal design.¹⁹ Regarding the data of the multivariate analyses, the high percentage of unexplained variance of BMI (41–72%) is a further limitation of our study. It is obvious that the determinants measured in our study could not fully explain the variability of BMI. This may be due to the cross-sectional study design as well as the inaccuracy of the methods applied (eg our questionnaire covered some qualitative instead of quantitative data). It is also tempting to speculate that genetic factors can add to the remaining variance. To address at least some of these questions, we also have to wait for analyses of longitudinal data of KOPS. In addition, the genetic contribution is currently under investigation in a subpopulation of 200 KOPS families where grandparents, parents and children are investigated together.

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