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ORIGINAL CONTRIBUTION

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Impact of parental BMI on the manifestation of overweight 5–7 year old children

Summary *Background* There is an increase in the prevalence of overweight and obese children. Genetic and environmental factors are contributing factors but the influence of parental nutritional state on early manifestation of overweight is not well characterised.

Aim of the study To systematically investigate the impact of parental BMI on the manifestation of overweight in 5 to 7 year old children. *Methods* Cross-sectional study (as a part of the *K*iel *O*besity *P*revention *S*tudy [KOPS]) of 3306 children aged 5–7 years and their parents. The nutritional state of the children (BMI, triceps skinfold, fat mass, prevalence of overweight) was investigated in subgroups differing with respect to parental BMI. *Results* BMI of the children was significantly correlated with parental BMI ($r = 0.272$, $p < 0.01$). Children's BMI showed closer associations with maternal than with paternal BMI ($r = 0.254$ vs. 0.159, $p < 0.01$). A multivariate regression analysis showed that parental BMI explained 7.6 % of the variance in children's BMI. OR for overweight was elevated in children with at least one overweight parent (overweight mother: OR 2.9 (boys)/3.1 (girls); overweight father: OR 1.8 (boys)/2.4 (girls). OR was highest for children with two obese parents (OR 7.6 (boys)/6.3 (girls). Children with one obese parent were more frequently overweight than children with one overweight parent. *Conclusions* Parental BMI showed only a weak correlation with the BMI of their children. However, children's risk of becoming overweight increased with parental overweight and obesity. Thus, familial disposition has to be taken into account to identify risk groups for preventive measures.

Key words Childhood obesity – parent-infant relationship

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Background

The prevalence of overweight and obesity has increased during the last few years. This was shown in adults [1, 2] as well as in children [3, 4]. At the moment, the rise in the prevalence of overweight and obesity cannot be explained definitely. Some authors attribute the increase to high-fat diets associated with a low level of physical activity [5, 6]. However, many studies failed to detect differences in life style habits between obese and non-obese subjects [7–9]. It is therefore assumed that genetics and gene-environment interactions may play a greater role in the manifestation of overweight [10]. Obesity, once established, is difficult to treat. Therefore, prevention measures are necessary [2, 11]. It has been proposed that obesity prevention should start as early as possible and that prevention in a pediatric population may be the only effective treatment of adult obesity [12]. However specific target groups for early intervention measures have not been identified.

Little is known regarding the determinants of early manifestation of overweight. Vogler et al. [13] proposed that most of the familial risk for childhood obesity is likely to be explained by genetic factors and only a minor effect can be due to shared environment. We have previously shown a positive relation between parental weight and children's BMI [14]. The present study sets

out to systematically investigate the association between parental BMI and children's BMI in a large population of prepubertal children. Our main questions are: 1) How does the nutritional state of the children correlate with increasing BMI of the parents? 2) Is there a specific effect of paternal or maternal overweight on the nutritional state of the children? 3) Is there an impact of the parental BMI within the respective groups of children's nutritional state?

The study was planned to characterize subgroups of children with a high risk of becoming obese in order to specifically tackle them with preventive measures.

Subjects and methods

Study design

The *K*iel *O*besity *P*revention *S*tudy (KOPS) was started in 1996 to examine the prevalence and incidence of overweight and obesity in prepubertal children. Up to the year 2000, 4137 5–7 year old children participated in the study. The enrollment was combined with the medical school examination which is obligatory for all children before entering their first year in primary school. About 37 % of all first graders in Kiel/Northwest-Germany in this period took part in KOPS. This first part of the study has a cross-sectional design. Aims and methods of the total study have been described previously [15]. The local ethical committee approved the study. Parents and children voluntarily participated at KOPS. Written consent was obtained from all parents.

Subjects

In this paper data from 3306 children (1687 boys and 1619 girls) and their parents with complete data for anthropometric measurements (79.9 % of the total KOPS population) were analyzed. Reasons for excluding data were incomplete details on size and weight of the parents or incomplete anthropometric data of the children because of their refusing some measurements. The mean ages of boys and girls were 6.3 and 6.2 years, respectively. The mean ages of mothers and fathers were 34.3 and 37.5 years, respectively.

The nutritional state of the children was classified according to BMI percentiles for German children [16]. Obesity was defined as BMI equal or above age- and gender-specific 97^{th} percentile; overweight was defined as BMI between age- and gender-specific 90^{th} and 97^{th} percentile. Normal weight children had a BMI between age- and gender-specific 10^{th} and 90^{th} percentile and underweight was defined as BMI equal or below age- and gender-specific 10^{th} percentile.

The BMI of the parents was calculated from self-reported heights and weights. In adults obesity was defined as BMI ≥ 30 kg/m^2, overweight as BMI between 25 and 30 kg/m^2. The cut off for normal weight was set at BMI < 25 kg/m^2 and underweight was defined as BMI $<$ 18.5 kg/m^2 according to WHO [17]. The parents were divided into eight groups: 1) underweight mother and normal weight father, 2) both parents were normal weight, 3) normal weight mother and overweight father, 4) vice versa, 5) both parents were overweight, 6) normal weight mother and obese father, 7) vice versa, 8) both parents were obese. There was no corresponding group to group 1 because of the low prevalence of underweight fathers (n = 20; 0.6 %).

Measurements

Weight was measured to the nearest 0.1 kg on a calibrated balance-beam scale with subjects wearing underclothes. Height was assessed to the nearest 0.5 cm. Body fat was assessed by anthropometric measurements as well as bioelectrical impedance analysis (BIA). BIA measurements were done at 50 kHz, using the Multi Frequency Analyzer, BIA 2000 M® (Data Input GmbH, Frankfurt, Germany). The coefficients of variation (cv) for repeated (n = 3) estimations of R (resistance) and Xc (reactance) in 10 children aged 5–7 years were 1.0 and 2.1 %, respectively, resulting in a cv of 1.5 % in percent fat mass. Triceps (TSF) and supscapular (SSF) skinfold thickness were measured to the nearest 0.2 mm with a Lafayette caliper (Model 01127, Lafayette Instrument Company, Indiana 47903, USA), calibrated to exert a constant pressure of 10 g/mm^2. Skinfold thickness was determined using standard techniques [18]. The cv for repeated measurements (n = 3) of TSF and SSF in 150 children aged 5–7 years were 4.2 % and 5.1 %, respectively.

Fat free mass (kg) was calculated according to Goran et al. [19]. Fat mass (kg) was then determined as the difference between body weight and fat free mass and expressed as percent body fat.

Statistical methods

SPSS procedures (version 10; SPSS Inc., Chicago, IL, USA) were used for data analysis. The Mann-Whitney U test controlled for significant differences in parameters of the nutritional state between gender. The chi-square test tested for a significantly different distribution of the nutritional state between genders. Significant differences between the parental BMI groups were checked with the Kruskal-Wallis test. The Bonferroni test was used for post hoc analysis. Odds ratios (OR) were calculated as a measure of risk factor for being overweight or obese. For all OR, group 2 (normal weight parents) served as a reference group. Pearson correlation coeffi-

cients were calculated between child's and parental BMI. A linear regression analysis was run with BMI of the children as dependent variable and BMI of mothers and fathers as independent variables. Level of significance was set at $p < 0.05$.

Results

Characteristics of the study population are presented in Table 1. Boys were older, taller and heavier but had lower triceps skinfold and percent fat mass than girls. There were no sex-differences in BMI. Mothers were younger, smaller and also had a lower BMI than the fathers. In contrast to children, there were sex-differences in parents with respect to the prevalence of overweight, normal weight and underweight. Mothers were more frequently underweight and normal weight than fathers, but fathers were more frequently overweight and obese.

BMI of the children was significantly correlated with parental BMI, i. e., $(BMI_{mother} + BMI_{father})/2$ ($r = 0.272$, $p < 0.01$). The correlation was stronger in mothers ($r = 0.249$, $p < 0.01$) than in fathers ($r = 0.159$, $p < 0.01$). Boys showed marginally stronger correlation to parental BMI than girls (to maternal BMI: boys: $r = 0.254$, $p < 0.01$, girls: $r = 0.242$, $p < 0.01$; to paternal BMI: boys: $r = 0.160$, $p < 0.01$, girls: $r = 0.159$, $p < 0.01$). The correlation coefficients were controlled for age- and gender-dependent BMI distribution.

Using a multivariate linear regression analysis parental BMI explained 7.6 % of the variance in children's BMI.

Of the parents, 43.6 % were normal weight. In 46.3 % and 10.1 %, at least one parent was overweight and obese, respectively. Table 2 shows the impact of parental BMI on BMI, TSF, fat mass and prevalence of overweight of their children. Significant sex-differences were seen between the groups. Significant differences between groups were seen with respect to weight, BMI, TSF and percent fat mass as well as in the prevalence of over-

Table 1 Characteristics (median, interquartile range) of children and their parents

	Boys (n = 1687)	Girls (n = 1619)	Fathers (n = 3306)	Mothers (n = 3306)
Age [years]	6.3 (0.5)*	6.2 (0.5)*	37 (8)*	34 (6)*
Height [m]	1.20 (0.07)*	1.19 (0.07)*	1.80 (0.1)*	1.68 (0.1)*
Weight [kg]	22.5 (4.5)*	22.0 (4.5)*	80.0 (14)*	63.0 (13)*
BMI [kg/m^2]	15.5 (1.8)	15.5 (2.1)	24.7 (3.8)*	22.5 (4.4)*
TSF[a] [mm]	10.0 (4.0)*	11.3 (4.4)*	–	–
Fat mass[b] [%]	15.1 (4.4)*	18.8 (5.2)*	–	–
Obese [%]	5.2	4.1	6.7**	5.7**
Overweight [%]	5.7	7.0	37.4**	19.4**
Normal weight [%]	81.4	81.0	55.3**	69.7**
Underweight [%]	7.7	7.8	0.6**	5.1**

[a] triceps skinfold; [b] according to Goran et al. [19]
* Mann-Whitney U-test: significant differences between boys and girls and mothers and fathers ($p < 0.05$); ** Chi-square test: significantly different distribution of the nutritional state of the parents ($p < 0.05$)

Table 2 Nutritional state (median/interquartile range) of 3306 5–7-year-old children (1687 boys, 1619 girls) and prevalences of overweight (%/absolute number) stratified by BMI of their parents (key variables are marked)

group number	1	2	3	4	5	6	7	8
BMI_{Father} [kg/m^2]	< 25	< 25	≥ 25–< 30	< 25	≥ 25–< 30	≥ 30	< 25	≥ 30
BMI_{Mother} [kg/m^2]	< 18.5	≥ 18.5–< 25	< 25	≥ 25–< 30	≥ 25–< 30	< 25	≥ 30	≥ 30
boys (n)	51	703	437	157	164	71	63	41
age [years]	6.3 (0.6)[f]	6.2 (0.5)	6.3 (0.6)	6.3 (0.5)	6.3 (0.5)	6.2 (0.6)	6.4 (0.6)	6.2 (0.5)
height [cm]	1.21 (0.1)[f]	1.2 (0.1)[f]	1.2 (0.1)[f]	1.21 (0.1)[f]	1.21 (0.1)	1.21 (0.1)[f]	1.2 (0.1)	1.22 (0.1)
weight [kg][e]	22.0 (3.7)[f]	22.0 (3.7)[f]	22.1 (4.0)	22.5 (5.2)	23.0 (5.1)	22.7 (5.8)	23.2 (5.5)	25.0 (9.0)
BMI [kg/m^2][e]	**15.2 (1.7)[f]**	**15.3 (1.7)**	**15.4 (1.8)**	**15.6 (2.3)**	**16.0 (2.4)**	**15.6 (3.0)**	**16.1 (2.3)**	**16.8 (3.8)**
TSF[a] [mm][e]	9.6 (3.3)[f]	10.0 (4.0)[f]	10.0 (3.7)[f]	10.3 (4.4)[f]	10.6 (5.3)[f]	10.0 (5.7)[f]	11.0 (5.7)	12.0 (7.4)
FM[b] [%][e]	14.5 (4.0)[f]	14.7 (3.9)[f]	15.1 (4.4)[f]	15.4 (4.4)[f]	16.1 (5.3)[f]	15.1 (6.4)[f]	16.0 (6.7)[f]	18.0 (8.2)[f]
overweight [% (n)][c, e]	2 (1)	5.8 (41)	9.4 (41)	14.6 (23)	21.3 (35)	15.5 (11)	28.6 (18)	34.1 (14)
OR (95 % CI)[d]	**0.3** (0.1–2.4)	**1**	**1.8**[g] (1.1–2.7)	**2.9**[g] (1.6–5.0)	**4.6**[g] (2.8–7.5)	**3.1**[g] (1.5–6.3)	**6.8**[g] (3.6–12.7)	**7.6**[g] (4.4–13.0)
girls (n)	51	635	451	136	186	75	50	35
age [years]	6.2 (0.4)[f]	6.2 (0.4)	6.2 (0.5)	6.3 (0.5)	6.2 (0.6)	6.2 (0.5)	6.2 (0.5)	6.2 (0.5)
height [cm]	1.17 (0.1)[f]	1.2 (0.1)[f]	1.19 (0.1)[f]	1.19 (0.1)[f]	1.21 (0.1)	1.18 (0.1)[f]	1.22 (0.1)	1.19 (0.1)
weight [kg][e]	20.0 (4.4)[f]	21.7 (4.0)[f]	22.0 (4.3)	22.2 (5.0)	23.2 (5.1)	22.0 (6.0)	23.5 (4.6)	23.0 (6.1)
BMI [kg/m^2][e]	**14.5 (1.8)[f]**	**15.3 (1.8)**	**15.5 (2.2)**	**15.7 (2.6)**	**16.1 (2.5)**	**15.7 (3.2)**	**16.1 (3.0)**	**16.0 (3.5)**
TSF[a] [mm][e]	10.3 (4.1)[f]	11.0 (3.9)[f]	11.0 (4.4)[f]	11.6 (4.6)[f]	12.3 (5.0)[f]	11.7 (4.7)[f]	12.0 (5.2)	13.3 (4.7)
FM[b] [%][e]	17.2 (5.0)[f]	18.3 (4.6)[f]	18.7 (5.3)[f]	19.3 (5.2)[f]	20.4 (5.5)[f]	19.9 (6.5)[f]	19.5 (6.8)[f]	20.9 (6.4)[f]
overweight [% (n)][c, e]	2 (1)	5.5 (35)	11.5 (52)	14.7 (20)	19.9 (37)	21.3 (16)	22.0 (11)	25.7 (9)
OR (95 % CI)[d]	**0.3** (0.1–2.6)	**1**	**2.4**[g] (1.5–3.7)	**3.1**[g] (1.7–5.6)	**4.5**[g] (2.7–7.3)	**4.9**[g] (2.6–9.3)	**5.1**[g] (2.4–10.8)	**6.3**[g] (2.7–14.3)

[a] triceps-skinfold; [b] fat mass according to Goran et al. [19]; [c] according to German reference population (Kromeyer-Hauschild et al. [16]); [d] Odds Ratio (95 % confidence interval); [e] Kruskal-Wallis test for significant differences between parents' BMI groups ($p < 0.05$); [f] Mann-Whitney test for significant differences between gender ($p < 0.05$); [g] OR significantly different from the reference group (group 2)

weight (Table 2). Results of the post hoc test are presented in Table 3: in group 8, boys' weight and BMI were significantly different from weights and BMI seen in all other groups. Girls of underweight mothers and normal weight fathers (group 1) had significantly lower weights and BMI when compared to all other groups except group 2 for BMI (Table 3).

Odds ratios for being overweight (OR, are given in Table 2) were significantly elevated for boys and girls with at least one overweight parent. OR were even higher for children with at least one obese parent. The highest OR was seen in children whose parents were both obese. Overweight or obesity in mothers resulted in higher ORs than overweight or obesity in fathers. Boys with an obese father but a normal weight mother had a lower OR than boys with two overweight parents. In girls, OR were higher in children with an obese father and a normal weight mother than in children with two overweight parents.

OR in children with an underweight mother and a normal weight father (group 1) was decreased (OR = 0.3) compared to the reference group. The difference was not significant.

The impact of parental BMI on the respective nutritional states of the children is demonstrated in Table 4. Significant differences between the parent groups were seen in normal weight children: BMI in both sexes as well as TSF in girls rose with increasing parental BMI. Post hoc analysis did not find a significant group effect.

Discussion

In this cross-sectional study we investigated the impact of parental BMI on the manifestation of overweight in 5–7 year old children. Overweight of the parents is a risk factor of overweight in 5–7 year old children even if only one parent is affected (either the mother or the father). We only found a weak correlation between BMI of parents and their children. This is in line with previous data. Whitaker et al. [20] also showed that the relationship between the parental and offspring's BMI becomes more important in children who were at least 7 years old. Most of the studies with young children could not find an association [21]. By contrast, correlation coefficients were higher in other studies investigating older children [22–24]. However, the association between the parental and children's BMI is also influenced by parental sex. Consistent with Cutting et al. [24] we found a stronger correlation between children's and maternal BMI when compared to the paternal association. Fogelholm et al. [22] could also show a stronger correlation between the BMI of sons and maternal BMI. Concomitantly there was no association between maternal BMI and the BMI of their daughters. We also saw that maternal BMI had a greater influence on the nutritional state of boys than on girls (group 6 vs. 7).

Consistent with the results of the correlation coefficients our OR showed a stronger influence of maternal BMI than of paternal BMI on the prevalence of overweight in children (Table 2: parent group 3 versus 4 as well as 6 versus 7). Sørensen et al. [25] and Kaplowitz et

Table 3 Significant differences (Bonferroni, $p < 0.05$) between the parent groups in regard to weight, BMI, TSF and percent fat mass of the children (post hoc test for table 2)

boys:								
significantly different to:	1	2	3	4	5	6	7	8
1					a, b, d		a, b, c, d	a, b, c, d
2				b, c, d	a, b, c, d	d	a, b, c, d	a, b, c, d
3					a, b, d		a, b, d	a, b, c, d
4								a, b, c, d
5								a, b, c, d
6								a, b, c, d
7								a, b
8								
girls:								
significantly different to:	1	2	3	4	5	6	7	8
1		a	a, b	a, b	a, b, d	a, b, d	a, b	a, b, c, d
2				b	a, b, c, d	b, c, d	a, b	b, c, d
3					a, b, c, d		a, b	
4					d		a	
5								
6								
7								
8								

[a] weight; [b] BMI; [c] TSF; [d] percent fat mass according to Goran et al. [19]
1 = father normal weight, mother underweight; 2 = both parents normal weight; 3 = father overweight, mother normal weight; 4 = father normal weight, mother overweight; 5 = both parents overweight; 6 = father obese, mother normal weight; 7 = father normal weight, mother obese; 8 = both parents obese

Table 4 Impact of parental BMI on the respective nutritional states (median/interquartile range) of the children (n = 3306)

	boys				girls			
nutritional state of the parents	at least one parent underweight	both normal weight	at least one parent overweight	at least one parent obese	at least one parent underweight	both normal weight	at least one parent overweight	at least one parent obese
underweight children								
$n_{(mothers/fathers)}$[a]	6 (5/1)	62 (62/62)	58 (17/47)	4 (3/2)	16 (14/2)	53 (53/53)	45 (17/35)	12 (5/8)
age [years]	6.3 (0.9)	6.3 (0.6)	6.3 (0.4)	6.3 (0.4)	6.1 (0.6)	6.3 (0.6)	6.2 (0.5)	6.2 (0.5)
height [cm]	1.23 (0.05)	1.19 (0.08)	1.20 (0.08)	1.21 (0.09)	1.16 (0.06)	1.18 (0.05)	1.19 (0.1)	1.16 (0.07)
weight [kg]	19.5 (1.3)	18.8 (3.1)	19.5 (2.8)	19.5 (3.4)	18.0 (2.6)	18.0 (2.0)	18.0 (3.0)	17.8 (2.2)
BMI [kg/m²]	**13.2 (0.7)**	**13.4 (0.6)**	**13.4 (0.7)**	**13.3 (0.4)**	**13.1 (1.1)**	**13.2 (0.4)**	**13.1 (0.8)**	**13.2 (0.7)**
TSF[b] [mm]	8.1 (3.3)	8.6 (3.3)	8.0 (3.2)	8.5 (3.4)	10.3 (3.0)	8.7 (2.5)	9.6 (3.7)	8.5 (5.2)
FM[c] [%]	11.4 (5.8)	11.4 (5.1)	11.7 (2.7)	11.9 (2.4)	16.6 (4.3)	15.6 (2.5)	15.4 (3.7)	14.6 (3.7)
normal weight children								
$n_{(mothers/fathers)}$[a]	51 (45/6)	593 (593/593)	601 (246/478)	128 (69/85)	41 (36/5)	540 (540/540)	619 (248/513)	112 (60/77)
age [years]	6.3 (0.6)	6.2 (0.5)	6.3 (0.5)	6.2 (0.6)	6.2 (0.5)	6.2 (0.5)	6.3 (0.6)	6.2 (0.5)
height [cm]	1.19 (0.08)	1.20 (0.07)	1.20 (0.07)	1.20 (0.08)	1.18 (0.07)	1.20 (0.07)	1.19 (0.07)	1.19 (0.0.6)
weight [kg]	22.0 (3.2)	22.0 (3.5)	22.5 (3.1)	22.5 (3.2)	21.0 (3.9)	22.0 (3.4)	22.0 (3.7)	22.0 (4.0)
BMI [kg/m²]	**15.2 (1.6)[d]**	**15.3 (1.4)[d]**	**15.5 (1.5)[d]**	**15.6 (1.5)[d]**	**14.8 (1.7)[d]**	**15.4 (1.5)[d]**	**15.5 (1.7)[d]**	**15.6 (1.6)[d]**
TSF[b] [mm]	9.7 (4.0)	10.0 (3.4)	10.0 (3.4)	10.0 (4.0)	10.3 (5.7)[d]	11.0 (3.4)[d, e]	11.0 (4.0)[d, e]	11.6 (3.0)[d]
FM[c] [%]	15.0 (3.9)	14.7 (3.5)	15.2 (3.6)	14.9 (4.2)	17.8 (6.0)	18.3 (4.1)	18.7 (4.3)	19.2 (4.2)
overweight children								
$n_{(mothers/fathers)}$[a]	1 (1/0)	26 (26/26)	56 (27/44)	14 (10/7)	0	26 (26/26)	72 (36/61)	16 (11/10)
age [years]	6.8	6.3 (0.4)	6.2 (0.5)	6.2 (0.6)		6.1 (0.4)	6.2 (0.6)	6.2 (0.5)
height [cm]	1.22	1.24 (0.08)	1.23 (0.07)	1.25 (0.1)		1.22 (0.06)	1.23 (0.08)	1.20 (0.11)
weight [kg]	27.0	27.2 (5.0)	28.0 (4.3)	29.0 (5.7)		27.8 (2.5)	27.6 (5.5)	26.4 (6.4)
BMI [kg/m²]	**18.1**	**18.6 (0.7)**	**18.4 (0.8)**	**18.4 (1.1)**		**18.7 (0.9)**	**18.5 (0.7)**	**18.4 (1.1)**
TSF[b] [mm]	10.0	13.5 (4.3)	14.3 (5.2)	15.8 (6.2)		15.3 (5.0)	15.7 (3.0)	16.8 (3.4)
FM[c] [%]	16.2	20.4 (3.2)	20.9 (5.4)	21.6 (5.4)		23.6 (4.3)	24.0 (4.7)	24.3 (4.1)
obese children								
$n_{(mothers/fathers)}$[a]	0	15 (15/15)	43 (31/32)	29 (22/18)	1 (1/0)	9 (9/9)	37 (21/28)	20 (9/15)
age [years]		6.2 (0.3)	6.4 (0.4)	6.2 (0.4)	6.7	6.2 (0.7)	6.1 (0.4)	6.3 (0.5)
height [cm]		1.24 (0.08)	1.25 (0.08)	1.24 (0.04)	1.28	1.23 (0.05)	1.21 (0.1)	1.24 (0.09)
weight [kg]		32.5 (5.2)	32.1 (7.7)	32.7 (7.2)	33.0	31.3 (5.2)	31.0 (6.0)	32.7 (7.3)
BMI [kg/m²]		**20.8 (2.6)**	**21.2 (4.0)**	**20.7 (3.4)**	**20.1**	**21.6 (1.8)**	**21.5 (1.7)**	**21.6 (3.6)**
TSF[b] [mm]		18.3 (6.7)	17.7 (8.2)	19.3 (7.9)	16.0	20.3 (6.0)	17.0 (6.5)	19.5 (10.0)
FM[c] [%]		25.3 (7.8)	26.5 (6.3)	25.8 (8.0)	23.7	29.2 (4.9)	27.7 (5.1)	30.4 (7.7)

[a] number of mothers/fathers with the respective nutritional state; [b] triceps-skinfold; [c] fat mass according to Goran et al. [19]; [d] Kruskal-Wallis test for significant differences between parents' BMI groups ($p < 0.05$); [e] Bonferroni as post hoc test ($p < 0.05$)

al. [26] also reported a stronger relationship between mothers and their children than for fathers and their children. This observation may be due to the fact that children's food environment is usually shaped more by mothers than fathers [27].

Table 2 also showed the greatest influence of parental BMI on the nutritional state of children with two overweight or obese parents. This result is in line with Whitaker et al. [28]. Hebebrand et al. [29] assumed that the phenomenon of 'assortative mating' (i. e., that two obese parents are passing on additive and non-additive genetic factors underlying obesity to their children) may explain the increased rates of extreme obesity in this subgroup of children.

Although the classification of the nutritional state of the children followed BMI percentiles, differences between the different parental BMI groups were greater regarding the prevalence of overweight and OR than the medians of the BMI of the children (Table 2). Thus, children's BMI values were close to the cut offs for overweight. There has been a long-term discussion about the definition of overweight and obesity in childhood, which is not finished yet [30, 31]. It is still unclear whether BMI is the best marker of overweight in children. Triceps-skinfold and fat mass (as derived from bioelectrical impedance analysis) are other parameters to define overweight. All methods have limitations. Depending on the algorithm applied, fat mass differs intra-individually [32]. BMI seems to have a low sensitivity to screen overweight children [33]. Nevertheless, we found the same tendencies for all three parameters (BMI, TSF and percent fat mass) with regard to the impact of parental nutritional state. We have seen the most significant differences between parental groups for BMI (Table 3). We found that for normal weight children the BMI increased with rising BMI of the parents (Table 4).

The absence of a group effect may be due to smaller statistical power of the post hoc test. In overweight children we saw the same trend for TSF and fat mass but not for BMI. There was no consistent trend in underweight and obese children. This may be due to the smaller number of children in these groups. To our knowledge, not many published studies have investigated differences in parameters of nutritional state in children of the same weight state in respect to familial predisposition of obesity. Only Francis et al. [34] stated a significantly higher percentage of abdominal body fat and lower fat free mass in non-obese 6–10 year old children of obese mothers, compared to normal weight children of non-obese mothers. This effect leads to the assumption that normal weight children with at least one overweight or obese parent are at risk of becoming overweight or obese [34].

The self-reported heights and weights of the parents may be a limitation of our study. In the German National Health Interview and Examination Survey 1998, in which heights and weights were measured, 15 % of the men and 13 % of the women (aged 30–39 years) were classified as obese (BMI ≥ 30) [35]. In our study only 7 % and 6 % were obese, respectively. Self-reports are known to underestimate BMI; i. e., heights are generally overestimated, while weights are underestimated [36]. To estimate the difference between measured and reported height and weight, Wardle et al. [37] found that the measured BMI was (on average) 1.62 units higher than the reported BMI. Correcting the self-reported BMI of our parents in our study around +1.62 BMI units, we found a similar prevalence of obesity as in the German National Health Interview and Examination Survey 1998 (14 % obese fathers and 10 % obese mothers). The still lower prevalence may depend on a selection of the study population. Because the participation rate in our study was voluntary the non-response analysis showed a marginal bias to lean families (data not shown). After correction of BMI we found the same differences and tendencies as before. Nevertheless, some differences between the groups can be concealed because we cannot assure that all the parents are correctly classified into the groups. Wardle et al. [37] showed that the underestimation of the BMI is greater for obese parents than for normal weight subjects. We could not consider this. So, some of the true obese parents may be classified as overweight and some of the true overweight parents are possibly in the group of normal weight parents, respectively.

This paper shows that the nutritional state of prepubertal children is influenced by parental BMI. The "biological" effect seems small (i. e., r^2 of about 7.6 in a multivariate regression), but a familial disposition of overweight is associated with a higher risk to develop overweight (see high OR). Parental overweight and obesity are risk factors of childhood overweight. To prevent a further increase of obesity in adulthood the familial disposition of overweight (BMI of the parents) has to identify the risk group for preventive measures.

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