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Effect of parental overweight and serum leptin levels on the manifestation of overweight in 7-year-old Korean children

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Abstract

Objective: To explore the relationship between children and their parents in terms of various anthropometric parameters and obesity-related hormone levels and to identify early indicators for child obesity.
Design: Cross-sectional observational study.
Setting: Urban areas of Korea in 2005.
Subjects: A total 124 families with 7-year-old children participated. Anthropometric and blood biochemistry data and information concerning the children's lifestyles, dietary habits and parental and grandparental weight status were obtained.
Results: The mean values for all anthropometric parameters were greater in overweight children than in children of normal weight. Very close relationships existed between the anthropometric parameters of children and their parents. Children with two overweight parents showed the highest odds for being overweight (OR 7·62). The strong relationship between overweight children and grandparental and parental overweight, especially on the maternal side, suggests gender differences in the intergenerational transmission of body weight. We also noted a greater risk of being overweight in children with a parent with high serum leptin level.
Conclusions: Grandparental and parental weight status and parental serum leptin levels enable us to identify childhood obesity at an early age and may help to counter the current epidemic of adult obesity.

Keywords
Childhood overweight
Parental overweight
Serum leptin

Childhood overweight is increasing worldwide[1,2]. Korean children have also become heavier; the ratio of children who exceed Korean BMI standards for 85 percentile cut-off increased from 5·4% in 1998 to 11·3% in 2001[3].

Childhood overweight and obesity are associated with insulin resistance and components of metabolic syndrome, such as increased blood pressure and abnormal glucose and lipid metabolism[4,5]. Thus, early detection of individuals at risk of being overweight may have long-term benefits for the prevention of childhood obesity.

Although several cross-sectional and longitudinal studies in Western and Asian countries have shown close relationships between parent and child obesity[6–8], and many studies have reported genetic and environmental factors in childhood obesity[9–11], the influence of parental characteristics such as food intake and biometric parameters on childhood obesity are not well characterised. The association between parental obesity and overweight in their offspring has been characterised to a lesser extent for young children than for adolescents. One reasons for this could be difficulty defining childhood overweight and obesity. Although BMI is used as a surrogate indicator of overweight in adults, many studies used sex- and age-specific BMI percentile charts to define overweight in children, based on the International Overweight Task Force (IOTF) standards or local reference data[1,3,12,13]. In the current study, we defined overweight using the twenty age- and sex-specific cut-off points described by Cole *et al.*[1].

Anthropometric variables are strongly related to the amount of body fat, as are the levels of several hormones such as leptin, adiponectin and insulin. Leptin concentration is positively correlated with many indices of obesity and insulin resistance[14]. Adiponectin is an adipocytokine that is exclusively synthesised by adipose tissue, and its concentration is inversely related to the degree of adiposity[15]. Adiponectin has been suggested as a candidate mediator of the development of obesity-related insulin resistance. Obese individuals tend to be insulin resistant; they become more insulin sensitive with weight loss[16].

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Although the degree of insulin resistance and levels of leptin and adiponectin are thought to be highly related to weight status, few data are available regarding the relationship between hormone levels in children and their parents. Elucidation of the relationships between the biometric parameters of parents and their children may permit early detection of children at risk for overweight and obesity. Therefore, our objective was to examine the relationships between children and their parents in various anthropometric parameters and obesity-related hormone levels.

Methods

Subjects

We recruited 124 7-year-old children (74 boys and 50 girls) and their parents from volunteers in Gwacheon city and Junggu, Seoul, between October and November 2005. Convenient sampling was used in the current study. The sole inclusion criterion was enrolment in the first grade; no exclusion criteria were applied. The study was approved by the institutional review board of the Seoul-Paik Hospital, Inje University, and the Korea Center for Disease Control and Prevention. Informed consent was obtained from the parents of the children.

Anthropometric measurements

Height was measured with an automatic stadiometer (model DS 102; Jenix, Seoul, Korea). Weight and percentage body fat were measured by bioimpedence analysis using a body composition analyser (model BC418; Tanita, Tokyo, Japan). BMI was calculated by dividing weight in kilograms by the square of height in metres. The definition of overweight in parents was BMI $\geq 25\,kg/m^2$, as defined by the WHO guidelines for adult obesity[17]. For children, overweight was defined as a BMI equal to or greater than a cut-off point corresponding to $25\,kg/m^2$ at 18 years of age for each age and sex using the IOTF standard[1].

Diet intakes and questionnaire analysis

Typical dietary intake for each child was estimated from the modified 3 d food records, which included the food records of two weekdays and one weekend day. The 3 d records were filled by children with the help of parents. The dietitian checked the records by interviewing with child and his/her parents on the physical examination day.

A total of 123 children (99·1 %) completed the dietary records. Total nutrient intake was determined for each child using the values estimated from the Computer-Aided Nutritional Analysis Program (CAN Pro) 2·0, a nutrient database developed by the Korean Nutrition Society[18].

A total of 120 children (96·8 %) completed the questionnaire developed for the current study. The questionnaire was used to determine the frequency of fruit and vegetable consumption, the frequency of physical activity per week and the weight status of the grandparents. On the basis of the levels of physical activity reported on the modified standardised questionnaires[19], the children were divided into two physical activity groups: low exercise (less than four times/week) and high exercise (more than four times/week). Parents were subjectively asked to report the obesity status (yes or no) in the child's maternal and paternal grandparents. The questionnaires were completed at home by the children and their parent.

Biochemical analyses

Blood samples were taken after fasting 12 h overnight. TAG, total cholesterol (TC), HDL-cholesterol (HDL-C) and LDL-cholesterol (LDL-C) levels were measured using enzymatic procedures and an autoanalyser (model 7180; Hitachi, Tokyo, Japan). Fasting serum glucose was measured using a hexokinase method. Fasting serum insulin was measured using a radioimmunoassay kit (Diagnostic Products Corporation, Los Angeles, CA, USA), and fasting leptin was determined using a kit from Linco Research (St Charles, MO, USA). Fasting adiponectin concentration was measured using an enzyme immunoassay (Bio-Vender, Modrice, Czech Republic). Insulin resistance was measured using homeostasis model assessment for insulin resistance (HOMA-IR) as the product of fasting insulin and glucose concentrations divided by 22·5[20].

Statistical analyses

Statistical analyses were conducted using the SAS statistical software package version 9·1 (SAS Institute Inc., Cary, NC, USA). Data were expressed as mean values with SD for continuous variables. The mean values for the groups were compared using Student's t test. Associations among variables in children and between variables in children and their parents are expressed as Pearson's correlation coefficients. Logistic regression analysis was performed to examine the risk of overweight in children based on the weight status of parents and to determine the odds of overweight in children associated with the leptin levels of both parents. Receiver operating characteristic (ROC) curve analyses of parental overweight and leptin levels were obtained to identify overweight children. Sensitivity and specificity were calculated for detecting child overweight.

Results

Characteristics of children and Pearson's correlation coefficients between BMI and other variables in children

The subjects' anthropometric values and blood profiles are presented in Table 1. All variables regarding anthropometric and biochemical parameters, except for body fat

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Table 1 Basic characteristics of family members

					Sons (*n* 74)						Daughters (*n* 50)					
	Fathers (*n* 124)		Mothers (*n* 124)		Total		Normal (*n* 47)		Overweight (*n* 27)		Total		Normal (*n* 37)		Overweight (*n* 13)	
Variable	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age (years)	40·3*	4·2	37·7	3·8	7·3	0·3	7·25	0·3	7·32	0·3	7·2	0·3	7·16	0·3	7·3	0·3
Height (cm)	171·7*	5·8	159·8	4·7	126·3*	4·9	125·1†	4·3	128·4	5·3	123·7	5·6	121·7†	4·4	129·1	5·2
Weight (kg)	73·0*	10·3	58·4	8·3	28·8*	6·0	25·3†	2·7	35·0	5·2	25·4	5·3	22·9†	2·4	32·7	4·4
BMI (kg/m^2)	24·8*	3·0	22·9	3·2	18·0*	3·0	16·1†	1·2	21·2	2·3	16·5	2·2	15·4†	1·2	19·6	1·3
Body fat (%)	25·3*	5·4	33·6	5·6	21·6	8·5	16·6†	4·2	30·3	6·7	21·1	6·4	18·2†	3·9	29·2	4·8
Waist (cm)	86·5*	7·6	77·5	8·2	59·5*	7·6	55·2†	3·7	67·0	6·7	55·6	6·5	52·4†	3·2	64·5	4·7
Hip (cm)	97·6*	5·5	94·0	6·2	69·9*	6·6	66·0†	3·6	76·6	5·0	66·4	5·7	63·6†	3·3	74·2	3·2
WHR	0·89*	0·04	0·82	0·06	0·85	0·05	0·84†	0·05	0·87	0·04	0·84	0·04	0·82†	0·04	0·87	0·04
TAG (mmol/l)‡	3·7*	2·0	2·2	1·4	1·7	1·2	1·3†	0·6	2·2	1·7	2·0	1·0	1·8†	0·8	2·6	1·2
TC (mmol/l)‡	5·1*	0·9	4·5	0·7	4·5	0·7	4·5	0·8	4·5	0·7	4·5	0·8	4·5	0·8	4·5	0·7
HDL-C (mmol/l)‡	1·3*	0·3	1·5	0·3	1·6*	0·3	1·7†	0·3	1·4	0·3	1·5	0·2	1·5	0·3	1·4	0·2
LDL-C (mmol/l)‡	3·1*	0·9	2·5	0·6	2·6	0·7	2·5	0·7	2·6	0·6	2·6	0·7	2·7	0·7	2·5	0·6
Glucose (mmol/l)‡	5·2*	0·9	4·9	0·5	5·0	0·4	5·0	0·5	4·9	0·3	4·9	0·3	4·9	0·3	4·8	0·4
Insulin (μg/ml)‡	12·8*	7·4	4·5	3·7	5·7	2·5	4·8†	1·9	7·4	2·6	6·0	4·1	5·2	2·3	8·7	6·8
HOMA-IR‡	3·04*	2·0	1·01	0·9	1·3	0·6	1·1†	0·5	1·6	0·6	1·3	0·9	1·1	0·5	1·9	1·4
Leptin (ng/ml)‡	4·0*	2·2	8·2	4·6	4·7	3·4	2·9†	2·0	7·9	3·1	4·5	2·7	3·3†	1·4	8·0	2·5
Adiponectin (μg/ml)‡	6·9*	2·9	10·8	5·0	12·6	4·5	13·2	4·8	11·6	3·9	13·8	5·2	14·8†	5·2	10·8	3·9
Birth weight (kg)					3·37	0·46	3·30	0·44	3·49	0·48	3·22	0·41	3·19	0·37	3·32	0·48
Energy intake (kJ/d)‡	7318·4*	1711·2	5829·3	1405·6	6428·4*	1485·2	5999·6†	1254·2	7206·4	1577·4	5408·0	1557·0	5409·8	1773·5	5403·0	723·5
Exercise ≥ 4 d/week, *n* (%)	17	14·2	41	34·5	30	41·7	17	37·0	13	50·0	11	23·4	9	26·5	2	15·4
Overweight ratio, *n* (%)	56	45·2	24	19·4	27	36·5					13	26·0				

WHR, waist-to-hip ratio; TC, total cholesterol; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance = [fasting serum insulin level (μg/ml) × fasting plasma glucose level (mmol/l)] ÷ 22·5.

*$P < 0·05$ significant difference between genders.

†Significantly different between normal weight children and overweight children among sons and daughters ($P < 0·05$).

‡On these variables, *P* value was determined after logarithmic transformation; however, mean and SD values are presented on untransformed variables.

and adiponectin levels, were significantly higher in fathers than mothers. The prevalence of overweight was 45·2% for fathers and 19·4% for mothers. Mean energy intake for fathers was greater than mothers. No difference was noted in frequency of physical activity more than four times/week between the two parents.

At 7 years of age, children showed a gender difference in weight status and metabolic parameters. All anthropometric values except body fat percentage and waist-to-hip ratio (WHR) were significantly higher in boys than in girls. The prevalence of obesity was 36·5% in boys and 26·0% in girls. All variables were further presented according to the weight status of children (Table 1). Overweight children had significantly greater height, weight, BMI, body fat, WHR, TAG and leptin concentrations than children of normal weight regardless of sex. Insulin and HOMA-IR levels tended to be greater in overweight than in normal weight boys. Mean energy intake in overweight boys was significantly higher than in normal weight boys. The percentage of low birth weight for children was 0·03. Just three children had low birth weight (<2·5 kg), all of them were boys. Because the number of low birth weight children was small, there was no association between low birth weight children and the others.

BMI values of the children were closely correlated with anthropometric variables when adjusted by the gender of the children. The correlation coefficients of BMI with height, weight, body fat, waist and WHR are 0·46, 0·93, 0·92, 0·92 and 0·49, respectively, at $P<0·001$. Although BMI was positively correlated with TAG, insulin, HOMA-IR and leptin levels ($r=0·27$, 0·30, 0·29 and 0·79, respectively; $P<0·05$), BMI was inversely related to levels of HDL-C and adiponectin ($r=-0·24$ and $-0·27$, respectively; $P<0·05$). BMI was positively correlated with total energy intake ($r=0·31$; $P=0·003$).

Relationship between children and their parents in anthropometric parameters and obesity-related hormone levels and energy intake

There were statistically significant differences in BMI, percentage of body fat, WHR and TAG levels among fathers when classified according to their sons' weight status (Table 2). No significant differences were noted in any variable between fathers with overweight daughters *v.* fathers with normal weight daughters.

Mothers with overweight daughters had greater body weight, BMI, percentage of body fat and WHR than those with normal weight daughters. The only statistically significant difference between mothers with overweight sons *v.* mothers with normal weight sons was in hip circumference. Fathers with overweight sons had greater fasting insulin and HOMA-IR levels than fathers with normal weight sons. However, no statistically significant differences were observed in fasting insulin or HOMA-IR levels among mothers classified according to their child's weight status. Fathers of overweight sons and mothers of overweight daughters had significantly greater leptin levels and lower adiponectin levels, respectively, than the fathers and mothers of normal weight children. Energy intake by parents with overweight children was not significantly different compared to that of parents with normal weight children. We analysed all variables of father and mother using a two-way ANOVA. The effect of the interaction between overweight status (normal/overweight) and gender of children (son/daughter) was not detected in father. In the contrary, the body weight, BMI, body fat, waist circumference, WHR and serum adiponectin level of mother were statistically significant by the interaction between overweight status and gender of children.

Pearson's correlation coefficients between anthropometric values, lipid profiles and hormone levels in parents and children are shown in Table 3. Children's anthropometric values, such as BMI and body fat, which are indicative of obesity, showed significant relationships with those of each parent. Leptin and adiponectin were significantly correlated with parents and children.

Odds for overweight in children according to parental and grandparental weight status

Table 4 shows OR for overweight in children based on the weight status of their parents and grandparents. The OR for overweight in children both of whose parents were overweight was significantly higher than in children with at least one normal weight parent (OR 7·62, 95% CI 2·01, 28·80). The OR for overweight in daughters with overweight mothers (OR 10·5, 95% CI 1·03, 107·15) was seven times greater than that of daughters with overweight fathers (OR 1·11, 95% CI 0·16, 7·52; data not shown).

On the basis of the questionnaire, we explored further associations between overweight in children *v.* grandparents. Although children with more than one overweight grandparent did not show a higher significantly chance of being overweight, children with two overweight parents and more than one overweight grandparent showed a higher chance of being overweight (Table 4). In particular, the OR for being overweight was greater in children with an overweight mother and overweight maternal grandparents.

Receiver operating characteristic curve analyses for identifying overweight children

Parental overweight as a criterion for identifying overweight children had a sensitivity of 0·23 and a specificity of 0·95 (Fig. 1, Table 5). In children, the leptin cut-off value having the best sensitivity and specificity for identifying overweight was 4·4 ng/ml (sensitivity, 0·93; specificity, 0·86). In parents, the best leptin cut-off values for

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Table 2 Variables of parents according to the weight status of their children

	Total		Sons				Daughters			
			Normal (*n* 47)		Overweight (*n* 27)		Normal (*n* 37)		Overweight (*n* 13)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Fathers										
Age (years)	40·3	4·2	40·4	4·2	40·1	3·9	40·4	4·3	40·6	5
Height (cm)	171·7	5·8	172·3	6·4	171·5	5·6	171·1	4·7	171·5	7·3
Weight (kg)	73	10·3	70·7	9·2	74·4	9·8	73·7	12	76·7	8·9
BMI (kg/m^2)	24·7	3	23·8*	2·7	25·2	2·6	25·1	3·5	26·1	2·4
Body fat (%)	25·3	5·4	23·8*	6·0	27	4·9	25·6	5·2	26·2	3·7
Waist (cm)	86·5	7·6	84·3*	7·4	7·4	74	87	8	89·5	7·2
Hip (cm)	97·6	5·5	96·9	5·7	98·5	4·5	97·5	5·9	99	5·1
WHR	0·89	0·04	0·87*	0·04	0·9	0·04	0·89	0·04	0·9	0·04
TAG (mmol/l)	3·7	2	2·9*	1·7	4·3	2·1	3·8	1·8	4·9	2·6
TC (mmol/l)	5·1	0·9	5	0·9	5·2	0·9	5·2	1·1	5	0·8
HDL-C (mmol/l)	1·3	0·3	1·4	0·3	1·3	0·3	1·3	0·3	1·2	0·3
LDL-C (mmol/l)	3·1	0·9	3	0·9	3·1	0·8	3·2	1	2·9	0·9
Glucose (mmol/l)	5·2	0·9	5·1	0·9	5·4	1·3	5·3	0·8	5·3	0·4
Insulin (μg/ml)	12·8	7·4	10·3*	6·01	14·7	9·29	14	6·87	14·9	7·33
HOMA-IR	3·04	2	2·42*	1·65	3·61	2·71	3·29	1·69	3·57	1·84
Leptin (ng/ml)	4	2·2	3·2*	1·8	4·5	2·1	4·3	2·2	5	2·4
Adiponectin (μg/ml)	6·9	2·9	7·5*	3·6	5·8	2·1	7	2·4	6·7	2·3
Energy intake (kJ/d)	7318·4	1711·2	6946·6	1658	7682·1	1953·1	7571·6	1543·2	7253·6	1740·7
Exercise ≥ 4 d/week, *n* (%)	17	14·16	8	17·39	3	11·11	6	17·65	0	
Overweight ratio, *n* (%)	56	45·16	13	27·66	14	51·85	21	56·76	8	61·54
Mothers										
Age (years)	37·7	3·8	38·2	4	37·4	4·1	37·6	3·3	36·9	4·3
Height (cm)	159·8	4·7	159·6	4·8	159·9	5·3	160·4	4·7	158·8	3·1
Weight (kg)	58·4†	8·3	56·5	7·1	59·3	8·1	57·2*	7·0	67·1	11·2
BMI (kg/m^2)	22·9†	3·2	22·2	2·6	23·2	2·6	22·2*	2·3	26·7	5·3
Body fat (%)	33·6†	5·6	32·4	5·5	33·9	5·4	32·9*	4·4	39·2	6·3
Waist (cm)	77·5†	8·2	76·2	7·6	77·7	7·6	75·8*	6·5	86·2	10·7
Hip (cm)	94	6·2	92·4*	5·5	95·2	5·7	93·2*	4·9	99·6	9
WHR	0·82†	0·06	0·82	0·06	0·82	0·05	0·81*	0·05	0·87	0·05
TAG (mmol/l)	2·2	1·4	2	1·3	2·1	1·1	2·1	1·4	3·3	2·4
TC (mmol/l)	4·5	0·7	4·5	0·7	4·4	0·8	4·4	0·8	4·7	0·7
HDL-C (mmol/l)	1·5	0·3	1·5	0·4	1·5	0·3	1·5	0·2	1·5	0·3
LDL-C (mmol/l)	2·5	0·6	2·5	0·6	2·6	0·7	2·5	0·6	2·6	0·4
Glucose (mmol/l)	4·9	0·5	4·9	0·4	4·8	0·5	4·9	0·4	5·2	1
Insulin (μg/ml)	4·5	3·65	3·96	3·21	4·45	3·09	4·9	4·24	5·49	4·43
HOMA-IR	1·01	0·88	0·87	0·73	0·95	0·67	1·1	1	1·35	1·29
Leptin (ng/ml)	8·2	4·6	7·6	5·1	8·9	4	7·2*	3·7	11·9	4·9
Adiponectin (μg/ml)	10·8†	5·0	10·8	4·8	10·7	6·3	11·9*	4·3	7·7	2·8
Energy intake (kJ/d)	5829·3	1405·6	5871·2	1378·4	6385·6	1722·7	5507·3	1280·6	5457	731·4
Exercise ≥ 4 d/week, *n* (%)	41	34·45	15	33·33	10	37·03	12	35·29	4	30·77
Overweight ratio, *n* (%)	24	19·35	6	12·77	7	25·93	4	10·81	7	53·85

WHR, waist-to-hip ratio; TC, total cholesterol; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance = [fasting serum insulin level (μg/ml) × fasting plasma glucose level (mmol/l)] ÷ 22·5.
*$P < 0·05$ by Student's *t* test.
†$P < 0·05$ by a two-way ANOVA for interaction between overweight status and gender of children.

identifying overweight children were 3·2 ng/ml (sensitivity, 0·80; specificity, 0·54) for fathers and 8·2 ng/ml (sensitivity, 0·65; specificity, 0·70) for mothers.

Discussion

Recent studies have shown that childhood obesity precedes adult obesity[21,22]. Thus, methods for detecting overweight in early childhood and avoiding accelerated weight gain in childhood should be explored as a means of preventing adult obesity. The risk of children becoming overweight might be greater if either one or both parents are obese[13,19,22]. Although many studies describing the relationships between parental and child obesity have identified familial factors that affect obesity in children, the effect of the weight of the parents on the propensity for overweight in their children has not been characterised to any great extent, especially for young children. Most studies have focused on either very young children or adolescents[13,23,24]. Together with the fetal period and adolescence, 6–7 years is thought to be the best time to start preventive strategies against excessive weight gain, as this age comes immediately after the adiposity rebound phase. This early adiposity rebound was reported to be associated with parental obesity[25].

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Table 3 Age-adjusted correlation coefficients of variables between children and their parents

Variable	Father–son (*n* 74)	Father–daughter (*n* 50)	Mother–son (*n* 74)	Mother–daughter (*n* 50)	Father–mother (*n* 124)	Father–child (*n* 124)	Mother–child (*n* 124)
Height	0·44*	0·42*	0·44*	0·07	0·13	0·43*	0·28*
Weight	0·36*	0·16	0·39*	0·58*	0·15	0·23*	0·40*
BMI	0·36*	0·14	0·37*	0·57*	0·09	0·21*	0·38*
Body fat	0·30*	0·22	0·24	0·49*	0·07	0·27*	0·32*
Waist	0·28*	0·13	0·24*	0·52*	0·09	0·18*	0·31*
Hip	0·23	0·14	0·37*	0·46*	−0·0007	0·17	0·35*
WHR	0·10	0·03	0·14	0·35*	0·05	0·05	0·21*
TAG†	0·17	0·34*	0·23	0·63*	0·15	0·26*	0·40*
TC†	0·26*	0·28	0·26*	0·44*	0·04	0·27*	0·34*
HDL-C†	0·37*	0·27	0·46*	0·27	0·08	0·36*	0·39*
LDL-C†	0·45*	0·30*	0·24*	0·38*	0·06	0·39*	0·29*
Glucose†	0·18	0·27	0·18	0·05	0·15	0·21*	0·11
Insulin†	0·08	0·11	0·26*	0·24	−0·07	0·09	0·24
HOMA-IR†	0·08	0·14	0·25*	0·22	−0·04	0·11	0·23
Leptin‡	0·43*	0·26	0·48*	0·52*	0·30*	0·35*	0·48*
Adiponectin†	0·35*	0·33*	0·34*	0·58*	−0·002	0·35*	0·38*
Energy intake†	0·33*	0·13	0·28*	0·19	0·14	0·20*	0·29*

WHR, waist-to-hip ratio; TC, total cholesterol; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance.
*$P < 0·05$ by partial correlation adjusted for age.
†On these variables, *P* value was determined after logarithmic transformation; however, mean and SD values are presented on untransformed variables.

Table 4 Associations between family overweight and child overweight

Family characteristics	Weight status	Prevalence of child overweight	OR	95% CI
Parental overweight		Total (*n* 124)		
	Both normal weight	22·8 (13/57)	1·00	
	One parent overweight	33·3 (18/54)	1·69	0·73, 3·91
	Both parents overweight	69·2 (9/13)	7·62	2·01, 28·8
Grandparental overweight		Total (*n* 114)		
	All normal weight	43·9 (50/114)	1·00	
	More than one grandparent overweight	56·1 (64/114)	1·82	0·81, 4·09
Parental and grandparental overweight (both paternal and maternal side)		Total (*n* 109)		
	Both parents and grandparents at normal weight	19·2 (5/26)	1·00	
	Either parent and more than one grandparent overweight	29·8 (17/57)	1·79	0·58, 5·52
	Both parents and more than one grandparent overweight	53·9 (14/26)	4·9	1·41, 16·99
Paternal and grandparental overweight (paternal side)		Total (*n* 116)		
	Both normal weight	26·9 (14/52)	1·00	
	Either parent and more than one grandparent overweight	36·4 (16/44)	1·55	0·65, 3·69
	Both parents and more than one grandparent overweight	40·0 (8/20)	1·81	0·61, 5·35
Maternal and grandparental overweight (maternal side)		Total (*n* 113)		
	Both normal	27·1 (16/59)	1·00	
	Either parent and more than one grandparent overweight	31·0 (13/42)	1·21	0·51, 2·88
	Both parents and more than one grandparent overweight	66·7 (8/12)	5·38	1·42, 20·33

Values are percentages (*n* = overweight/*N* = total).

Therefore, we aimed to determine the influence of family risk factors for overweight, parental weight and biochemical parameters on the distribution of overweight and obesity status in children commencing primary school.

Our study shows close associations between parents and their offspring in terms of body weight and obesity-related phenotypes, even at a pre-adolescent stage (7 years of age). Our data suggest that having both parents overweight vastly increases the risk of their offspring being overweight. The risk for overweight in children with one overweight parent tended to be greater than that in children with two normal weight parents. These results suggest that parental overweight status is an important predictor of overweight in early childhood. At an early age, children have a closer relationship with the same-sex parent in terms of their obesity-related phenotypes and hormonal levels, consistent with the results of earlier studies[22,26,27]. We also found close associations between grandparental overweight and overweight in the children. Because the data concerning grandparental weight status were acquired by questionnaire, our results need to be confirmed. However, the strong relationships between

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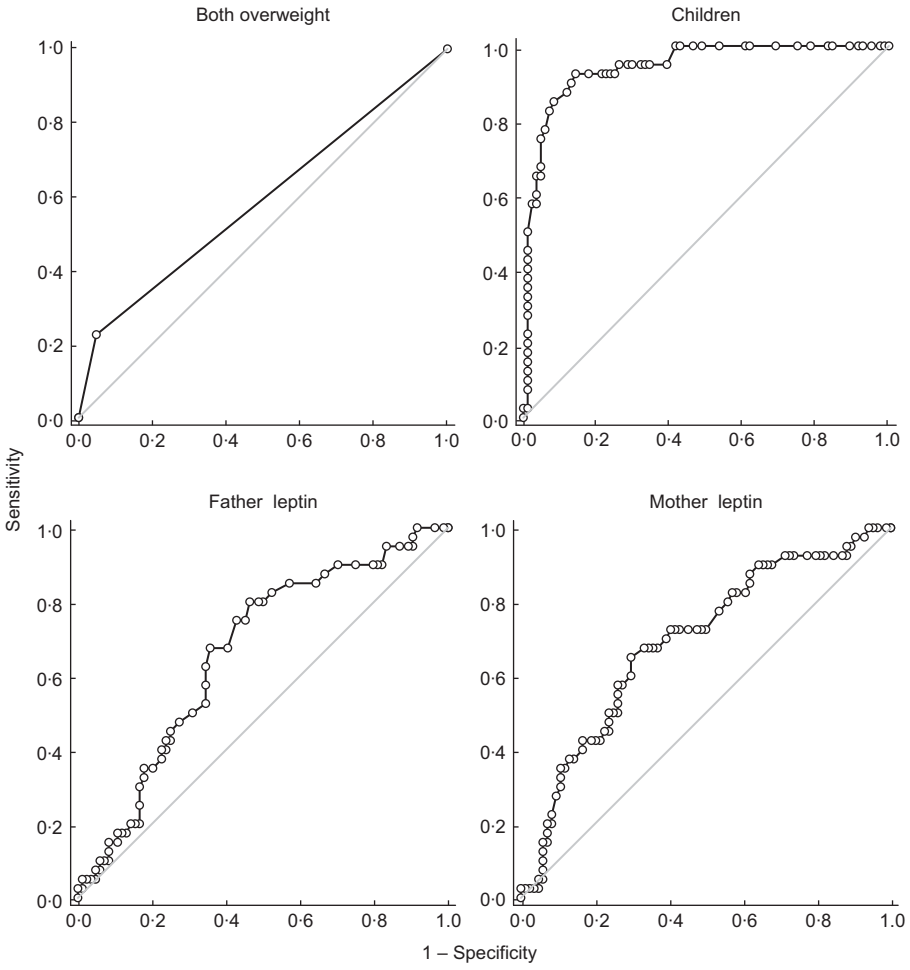

Fig. 1 Receiver operating characteristic curve for identification of overweight children by parental overweight or serum leptin levels in children and parents (AUC = 0·59, 0·94, 0·66 and 0·69 for both overweight, children, father and mother leptin, respectively). AUC, area under the curve

children and their parents' and grandparents' weight status, especially on the maternal side, suggest gender differences in the intergenerational transmission of body weight.

Because familial transmission is a risk factor for obesity, it is important to evaluate familial resemblance in lifestyle and biochemical parameters to identify management strategies for preventing childhood obesity and its related metabolic disorders. We observed a closer relationship in energy intake level, one of the lifestyle parameters, between parents and their children. We also observed a closer relationship in biochemical parameters, such as TAG, HDL-C and LDL-C, between parents and their children. Although we found no relationship between children and parent in insulin levels, we noted a close relationship between children and parent in levels of leptin and adiponectin. Leptin is produced by adipocytes and regulates food intake at the hypothalamic level. Leptin concentrations in the blood are correlated with the amount of body fat and BMI[28,29]. We showed that leptin levels in overweight children are greater than in normal weight children, which is consistent with the previously reported study that showed close relationships among leptin concentrations, the amount of body fat and BMI[29]. Furthermore, the risk of overweight in children increased when their fathers or mothers had elevated leptin levels. Leptin level was associated with body weight and predicted weight gain in obese children, suggesting that leptin concentrations in children and their parents could be used to identify children at risk of obesity. Our ROC analysis supports this hypothesis. High leptin levels in children identified 92·5 % of overweight children, whereas parental overweight identified only 23–55 % of such children. A parental leptin concentration greater than the cut-off value identified 65–80 % of overweight children, suggesting that parental leptin concentrations are more sensitive for identifying the risk of childhood obesity than parental overweight.

In our study, maternal leptin level detected overweight children with high specificity, and they could be easily detected at lower level of paternal leptin with higher sensitivity. Women had leptin concentrations two times

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Table 5 Receiver operating characteristic curve analysis for identification of overweight children by parental overweight or serum leptin levels in children and parents

		Children leptin	Father leptin	Mother leptin
Cut-off value	Both overweight	>4·4 (ng/ml)	>3·2 (ng/ml)	>8·2 (ng/ml)
AUC	0·59	0·94	0·66	0·69
95 % CI	0·50, 0·68	0·89, 0·98	0·57, 0·75	0·60, 0·77
Sensitivity	0·23	0·93	0·80	0·65
Specificity	0·95	0·86	0·54	0·70

AUC, area under the curve.

greater than those of men, reflecting gender differences in body fat distribution and contribution to weight gain.

Our study showed that adiponectin level was negatively correlated with BMI and its concentration was lower in overweight children. The inverse association between fatness and adiponectin level is consistent with earlier reports[15,30]. In our study, the risk for overweight in children tended to decrease as adiponectin levels of both parents increased. This suggests that high parental adiponectin levels have a protective effect against overweight in children. However, this was not statistically significant, perhaps because of the small number of subjects in each group.

Conclusion

In conclusion, the risk of overweight in young children was greater when both parents were overweight. Therefore, parental BMI is a good indicator of overweight children or elevated BMI at young ages. The strong relationship between children and grandparental and parental overweight, especially on the maternal side, suggests gender differences in the intergenerational transmission of body weight. Leptin levels in children and their parents may also identify the risk of childhood overweight. As a greater risk of overweight was observed in children with overweight parents or parents with high leptin levels, children in these categories should be considered targets for the intervention and management of obesity.

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