Increased Body Mass Index in Parent-Child Dyads Predicts the Offspring Risk of Meeting Bariatric Surgery Criteria

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Context: Obesity in children is a major public health concern.

Objective: This study examined the value of using parent-child dyads' adiposity status for predicting the individual's later eligibility for bariatric surgery (EBS).

Design, Setting, and Participants: The cohort consisted of 2647 individuals from the longitudinal Cardiovascular Risk in Young Finns Study. Baseline information included own and parental body mass index (BMI) in 1980 (children aged 3–18 years), whereas adult follow-up assessment examined EBS 21–31 years later.

Main Outcome Measure: EBS in adulthood was defined as: 1) BMI greater than 40 kg/m^2 or 2) BMI greater than 35 kg/m^2 with at least one of the following metabolic complications: type 2 diabetes, hypertension, or dyslipidemia.

Results: Addition of parents' BMI improved the prediction of adulthood EBS compared to the model including child's BMI, age, and sex (area under the curve values [95% confidence interval] (0.80 [0.74–0.85] vs 0.74 [0.68–0.81], $P = .003$). Obese children with an obese parent had a 21.2% chance of being EBS in adulthood. Compared to nonobese families, the risk ratio for EBS was 14.2 (95% confidence interval 8.0–25.2, $P < .001$) in obese children with an obese parent. The absolute risk of EBS was 30.9% if both child and parent were obese on more than one childhood assessment compared to 15.2% if they were obese only once, or 2.1% if they were never obese ($P < .05$).

Conclusions: These longitudinal data show that a combination of the child's and parents' BMI at baseline assessment is a useful predictive tool for assessing later EBS, and highlights the importance of accounting for parental BMI in the assessment of child obesity. (***J Clin Endocrinol Metab*** **100: 4257–4263, 2015)**

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Abbreviations: BMI, body mass index; BP, blood pressure; CI, confidence interval; CRP, C-reactive protein; EBS, eligibility for bariatric surgery; GHbA1c, glycated hemoglobin.

Approximately 2.1 billion people in the world are overweight or obese, and consequently at increased risk of many health problems, including heart disease, type 2 diabetes, and cancer (1). These conditions account for 70% of deaths in the United States, and 75% of all health care costs (2). Compounding this problem is the increasing burden of childhood obesity; one-third of children and adolescents are currently overweight or obese in the United States, compared with approximately 15% in the 1970s (3).

The management of obesity is complex, and traditional clinical care delivery systems are inadequately designed to successfully tackle the problem (2). Problems exist across the entire spectrum of health care, from policy provision to the clinical management of severe obesity, in which pharmacotherapy or surgery is often needed (4). Although severe obesity (grade 2; body mass index [BMI] >35 kg/m^2) affects a smaller proportion of individuals, it accounts for a disproportionate amount of health care costs, and bariatric surgery is often promoted as being cost-effective (5, 6), despite a lack of robust long-term data (7). Recent meta-analyses using follow-up data have shown that all-cause mortality is increased, especially among individuals with grade 2 obesity or obese individuals with metabolic risk factors (8, 9).

Modifiable early-life risk factors have been shown to predict later childhood obesity (10, 11). We have observed that cardiometabolic risk in adulthood associated with obesity can be reversed among those overweight/obese children who become nonobese adults, suggesting that nonsurgical interventions may have long-term benefit (12). However, there are few data on childhood and family predictors of persistent and severe adult obesity, hampering development of targeted effective interventions earlier in the life course.

We investigated the use of parent-child dyads' adiposity status during the offspring's childhood for predicting the individual's later eligibility for bariatric surgery (EBS). Our primary hypothesis was that data parental BMI improves the predictive ability of EBS risk. The analyses are based on the longitudinal Cardiovascular Risk in Young Finns Study, which includes data on 2647 individuals with baseline information on the child's own and parental adiposity status, and adulthood follow-up assessment performed 21–31 years later.

Materials and Methods

Participants

The Cardiovascular Risk in Young Finns Study is an ongoing prospective study of cardiovascular risk factors from childhood onwards. All of the field studies have been conducted in Helsinki, Kuopio, Oulu, Tampere, and Turku, the five Finnish cities with medical faculties. The study commenced in 1980 when 4320 children were randomly selected based a national registry at age groups of 3, 6, 9, 12, 15, and 18 years within the surroundings of the five study sites were invited. Of these, 3596 individuals participated. In adulthood, comprehensive follow-ups have been performed in 2001, 2007, and 2011 (13). The current sample included those 2647 individuals with baseline data on their own and parental adiposity status and adulthood follow-up assessment of adiposity status and cardiovascular measures performed in 2001, 2007, or 2011. Baseline risk factors of those retained in follow-up are largely comparable to nonparticipants (14). The study complies with the Declaration of Helsinki and has institutional ethics approval. Written informed consent was obtained from all participants.

Parental adiposity status

In childhood, questionnaires completed by the parents of the participant were used to obtain data on mother's and father's height and weight, from which BMI at enrollment was calculated as weight (kg)/height (m)^2.

Anthropometric and clinical assessment

Height and weight, rounded to the nearest 1 cm and 0.1 kg, respectively, were measured at all time-points using a similar protocol and BMI was calculated. Baseline blood pressure (BP) in those aged 3 years was measured by ultrasound; other childhood ages were measure by a mercury sphygmomanometer. Blood samples were obtained following a 12-hour fast. Standard enzymatic methods were used for serum total cholesterol, triglycerides, high-density lipoprotein cholesterol, and plasma glucose, and glycated hemoglobin (GHbA1c). Low-density lipoprotein cholesterol was calculated using the Friedewald formula (15). High-sensitivity C-reactive protein (CRP) was measured by an automated analyzer using a latex turbidimetric immunoassay. Questionnaires were used to obtain data on birth weight, socioeconomic status (parental study years), physical activity, and diet (16). The subjects were asked to fill in a questionnaire on habitual dietary choices including a short food frequency questionnaire with six response categories: 1. daily, 2. almost every day, 3. a couple of times per week, 4. once a week, 5. a couple of times per month, 6. more seldom or never. The response categories were converted into times of consumption per week (1 = 9.5; 2 = 4.3; 3 = 1.7; 4 = 0.7; 5 = 0.1; 6 = 0). Based on our earlier reports, we used vegetables and fruit (including juices) as dietary indicators (13). Physical activity was assessed with questions concerning the frequency and intensity of physical activity, and a physical activity index was calculated based on the variables as previously described (17). There were two different kinds of physical activity questionnaires for the younger (3- to 6-year-old children, a parent-completed questionnaire) and older children (9- to 18-year-old children, self-completed questionnaire). The calculated physical activity indices were age-standardized to allow comparison across age groups.

Classification of adulthood outcomes

Data on medications for hypertension, dyslipidemia, and type 2 diabetes were gathered from a registry of the Social Insurance Institution of Finland, including data on all medication purchases between 1993 and 2012. These data also include information on reimbursement decisions for hypertension (systolic BP

≥160 mm Hg and/or diastolic BP >95 mm Hg persistently during a 6-month follow-up period with nonmedical treatment) and type 2 diabetes (fasting plasma glucose ≥7 mmol/liter, nonfasting plasma glucose ≥11.1 mmol/liter, and/or GHbA1c ≥6.5% in two different measurements) made by the Social Insurance Institution of Finland based on applications from medical practitioners. Participants were classified as having type 2 diabetes if they had: (1 a fasting plasma glucose of 7.0 mmol/liter or higher, (2 GHbA1C at least 6.5%, 3) reported receiving oral hypoglycemic agents and/or insulin injections and did not have type 1 diabetes, (4 reported a history of physician diagnosed type 2 diabetes, or (5 had a medication reimbursement decision for type 2 diabetes (18). Hypertension was defined by reimbursement decision data and dyslipidemia by purchases of statins or ezetimibe. EBS in adult life was considered if 1) the BMI was greater than 40 kg/m^2 or 2) the BMI was greater than 35 kg/m^2 and there was at least one of the following metabolic complications of obesity: type 2 diabetes, hypertension, or dyslipidemia.

Statistical analyses

All statistical analyses were performed with SAS 9.3 software. Baseline and follow-up characteristics of the study sample are provided as mean (SD) for continuous variables or proportions for categorical variables.

Baseline adiposity measures and adult outcomes

We used Poisson regression to estimate the relative risk and their 95% confidence intervals (CI) 95% of continuous measures of children's and parent's BMI levels at baseline with adult morbid obesity. The incremental value of adding risk variables to predict morbid obesity was examined using multivariable logistic regression models. The ability of different models to predict obesity risk was estimated using C statistics by calculating the area under the receiver operating characteristic curve (19) with comparisons made using the DeLong algorithm.

Baseline obesity combinations and adult outcomes

To investigate how different combinations of the child's own and parental obesity at baseline predict later risk of obesity-related outcomes, we constructed dichotomized BMI variables. Among children, obesity was defined as BMI higher than the age- and sex-specific 85th percentile within the cohort. These cut-points and their corresponding US Centers for Disease Control and Prevention percentile points are shown in Supplemental Table 1. For parents, an obesity cut-point of 30 kg/m^2 was used. The associations between different child-parent adiposity combinations and adult outcomes were estimated using Poisson regression providing age- and sex-adjusted relative risks and 95% CIs. Because there were no interactions by child age and sex for the bariatric surgery criteria outcome variable, all analyses combined sex and age groups.

Results

The clinical characteristics of 2647 participants having both childhood and adulthood data (73.6% of the 3596 individuals examined at baseline study) are shown in Table 1. Mean age at baseline was 10.5 years and mean follow-up period was 29.3 years. At follow-up, 3.5% met EBS criteria, 7.1% had medication for dyslipidemia, 3.7% had reimbursed medication for hypertension, and 3.7% had type 2 diabetes.

Table 1. Characteristics of the Study Cohort

Variable	Statistic
N	2647
Males (%)	46.7
Baseline	
Offspring age (years)	10.5 (5.0)
BMI (kg/m^2)	17.8 (3.1)
Mother's BMI (kg/m^2)	23.9 (3.8)
Father's BMI (kg/m^2) (n = 2324)	25.4 (3.1)
Systolic blood pressure (mm Hg)	113 (12)
LDL-cholesterol (mmol/liter)	3.48 (0.82)
HDL-cholesterol (mmol/liter)	1.56 (0.31)
Birth weight (kg) (n = 2304)	0.66 (0.30)
CRP (mg/liter) (n = 2166)	1.0 (3.0)
Parental school years	10.1 (3.2)
Vegetable consumption (servings/week)	6.9 (2.8)
Fruit consumption (servings/week)	6.3 (2.9)
Physical activity (z-score) (n = 2453)	0.1 (1.0)
Adulthood	
Offspring age (years)	39.8 (6.3)
BMI (kg/m^2)	26.4 (5.1)
EBS (%)	3.5
BMI >35 kg/m^2 (%)	7.1
Hypertension medication (%)	3.7
Lipid-lowering medication (%)	7.1
Type 2 diabetes (%)	3.7

Abbreviations: LDL, low-density lipoprotein; HDL, high-density lipoprotein.

n = 2647, unless stated otherwise. Statistics are mean ± (SD) for continuous variables or percent for categorical variables.

Baseline adiposity measures and adult outcomes

Child's own BMI, mother's BMI, and father's BMI were all independently associated with the risk of later EBS in adulthood (Table 2). These effects remained significant in a model adjusted with data on age, sex, BP, lipids, CRP, birth weight, physical activity, socioeconomic status, and diet data included.

Addition of mother's BMI improved the predictive ability compared to the model initially including child's own BMI, age and sex (area under the curve values [95% CI] 0.79 [0.73–0.85) vs. 0.74 [0.68–0.81], $P = .01$). Father's BMI did not provide significant improvement (0.76 [0.70–0.83] vs 0.74 [0.68–0.81], $P = .10$). Inclusion of both mother's and father's BMI (0.80 [0.74–0.85] vs 0.74 [0.68–0.81], $P = .003$) improved the predictive ability when comparing to the model with child's BMI, whereas it did not improve the prediction provided by the model including child's and mother's BMI [95% CI] 0.80 [0.74–0.85] vs 0.79 [0.73–0.85], $P = .16$).

Baseline obesity combinations and adult outcomes

The risks of all adult outcomes were significantly increased among individuals with childhood obesity and

Table 2. Multivariable RR and 95% CI Between Childhood BMI (in 1980), Mother's BMI in 1980, and Father's BMI in 1980, With Adulthood EBS (BMI >40 kg/m^2 or BMI >35 kg/m^2 and at Least One of the Following Comorbidities: Type 2 Diabetes or Medication for Hypertension or Lipid-Lowering Medication

	Adjusted With Age and Sex		Fully Adjusted	
Variable	**RR (95% CI)**	***P* Value**	**RR (95% CI)**	***P* Value**
Own BMI	2.04 (1.69–2.46)	<.001	1.80 (1.28–2.52)	<.001
Mother's BMI	1.35 (1.11–1.64)	.003	1.72 (1.35–2.19)	<.001
Father's BMI	1.32 (1.09–1.60)	.006	1.39 (1.07–1.79)	.01

Abbreviation: RR, relative risks.

Results are from Poisson regression models. Fully adjusted model included data on age, sex, blood pressure, lipids, CRP, birth weight, physical activity, socioeconomic status, and diet.

RR-values are for 1-SD increase in BMI levels.

whose mother or father were obese (Table 3). Compared to the reference group (nonobese child-parent dyad), the relative risk of meeting bariatric surgery criteria was increased more than 14-fold among child-parent dyads with obese baseline BMI. Absolute prevalences were 23.6% for those with obese child-obese mother pairings and 25.5% for obese child-obese father pairings. As expected, all comparisons with the reference group were also significant among obese children who did not have either an obese mother or father. Those who were nonobese as a child but had an obese mother showed increased risk for all adult outcomes apart from hypertension, whereas, conversely, those who were nonobese as children with an obese father showed no increased risk for any of the adult outcomes. Supplemental Table 2 shows an estimate concerning US population based on present data and recent US statistics

Table 3. Prevalences, RRs, and Their 95% CI of Cardiometabolic Outcomes in Adulthood According to Childhood Own BMI and Mother's or Father's BMI

	Own BMI and Mother's BMI			Own BMI and Father's BMI		
	%	**RR (95% CI)**	***P* Value**	**%**	**RR (95% CI)**	***P* Value**
EBS criteria met						
Group I	1.5	Reference		1.8		
Group II	6.9	4.2 (2.1–8.6)	<.001	1.3	0.7 (0.2–2.9)	.62
Group III	11.5	8.0 (5.0–12.5)	<.001	8.4	5.0 (3.0–8.3)	<.001
Group IV	23.6	14.4 (8.0–26.3)	<.001	25.5	14.4 (8.1–25.6)	<.001
BMI >35 kg/m^2						
Group I	2.9	Reference		2.9		
Group II	7.0	2.3 (1.3–4.1)	.003	4.5	1.5 (0.8–3.0)	.22
Group III	16.0	5.6 (4.1–7.6)	<.001	14.1	4.9 (3.5–6.8)	<.001
Group IV	34.3	11.6 (7.8–17.2)	<.001	37.3	12.6 (8.5–18.6)	<.001
Hypertension medication						
Group I	2.9	Reference		3.1		
Group II	6.0	1.7 (0.9–3.0)	.08	3.5	1.0 (0.5–2.2)	.90
Group III	6.6	2.4 (1.6–3.6)	<.001	5.6	1.9 (1.2–3.1)	.006
Group IV	12.5	3.6 (1.8–6.9)	<.001	14.3	4.4 (2.4–8.2)	<.001
Lipid-lowering medication						
Group I	5.9	Reference		6.3		
Group II	14.6	2.1 (1.5–2.9)	<.001	5.5	0.8 (0.5–1.5)	.50
Group III	10.7	1.9 (1.4–2.6)	<.001	10.7	1.8 (1.3–2.5)	<.001
Group IV	13.9	2.0 (1.1–3.5)	.02	7.5	1.2 (0.5–3.0)	.63
Type 2 diabetes						
Group I	2.2	Reference		2.2		
Group II	9.1	3.5 (2.1–5.7)	<.001	2.5	0.9 (0.4–2.1)	.78
Group III	5.9	2.7 (1.8–4.3)	<.001	5.8	2.2 (1.4–3.6)	<.001
Group IV	13.7	5.2 (2.8–9.8)	<.001	10.5	3.9 (1.9–8.3)	<.001

Abbreviation: RR, relative risks.

Relative risks calculated with Poisson regression adjusted with age and sex.

Group I: own BMI ≤ age- and sex-specific 85th percentile and mother's (n = 2125)/father's (n = 1889) BMI ≤30 kg/m^2; group II: own BMI ≤ age- and sex-specific 85th percentile and mother's (n = 144)/father's (n = 150) BMI >30 kg/m^2; group III: own BMI > age- and sex-specific 85th percentile and mother's (n = 323)/father's (n = 286) BMI ≤30 kg/m^2; group IV: own BMI >age- and sex-specific 85th percentile and mother's (n = 55)/father's (n = 55) BMI >30 kg/m^2.

Table 4. Prevalences, RRs, and 95% CI of Cardiometabolic Outcomes in Adulthood According to Childhood Own BMI, Mother's BMI, and Father's BMI

	Own BMI and Both Parents' BMI		
	%	RR (95%CI)	*P* Value
EBS criteria met			
Group I	1.4	Reference	
Group II	4.5	3.0 (1.5–6.2)	.003
Group III	7.8	5.9 (3.3–10.7)	<.001
Group IV	21.2	14.2 (8.0–25.2)	<.001
Group V	25.0	18.6 (4.6–74.6)	<.001
BMI >35 kg/m^2			
Group I	2.7	Reference	
Group II	6.2	2.3 (1.4–3.7)	.003
Group III	12.0	4.6 (3.1–6.7)	<.001
Group IV	32.1	11.6 (8.0–16.6)	<.001
Group V	50.0	18.4 (9.2–36.6)	<.001
Hypertension medication			
Group I	2.9	Reference	
Group II	5.3	1.6 (0.9–2.7)	.08
Group III	4.8	1.9 (1.1–3.2)	.02
Group IV	11.5	3.6 (2.0–6.6)	<.001
Group V	20.0	6.4 (1.8–23.1)	.004
Lipid-lowering medication			
Group I	5.6	Reference	
Group II	10.9	1.7 (1.2–2.4)	.003
Group III	10.9	2.1 (1.5–3.0)	<.001
Group IV	9.5	1.5 (0.9–2.8)	.13
Group V	10.0	1.7 (0.2–12.1)	.61
Type 2 diabetes			
Group I	2.2	Reference	
Group II	5.3	2.1 (1.2–3.6)	.007
Group III	5.1	2.5 (1.4–4.2)	.001
Group IV	9.4	3.8 (2.0–7.3)	<.001
Group V	30.0	12.6 (4.4–35.8)	<.001

Abbreviation: RR, relative risks.

Relative risks calculated with Poisson regression adjusted with age and sex.

Group I: own BMI ≤ age- and sex-specific 85th percentile and mother's (n = 2125)/father's (n = 1889) BMI ≤30 kg/m^2; group II: own BMI ≤ age- and sex-specific 85th percentile and mother's (n = 144)/father's (n = 150) BMI >30 kg/m^2; group III: own BMI > age- and sex-specific 85th percentile and mother's (n = 323)/father's (n = 286) BMI ≤30 kg/m^2; group IV: own BMI >age- and sex-specific 85th percentile and mother's (n = 55)/father's (n = 55) BMI >30 kg/m^2.

(20). This estimation suggests that approximately 2.2 million of present US children would meet EBS criteria in adulthood.

Table 4 shows results from analyses combining data on child's own and both parents' BMI status. In these analyses, all comparisons with the reference group showed significant differences for EBS criteria. Obese children with an obese parent had a 2.4 times (95% CI 1.3–4.4, P = .006) higher prevalence of EBS than obese children with nonobese parents (absolute values 7.8% and 21.2%). Instead, there was no risk difference between obese children with two obese parents when compared to obese children with one obese parent (prevalences 21.2% and 25.0%, P = .78).

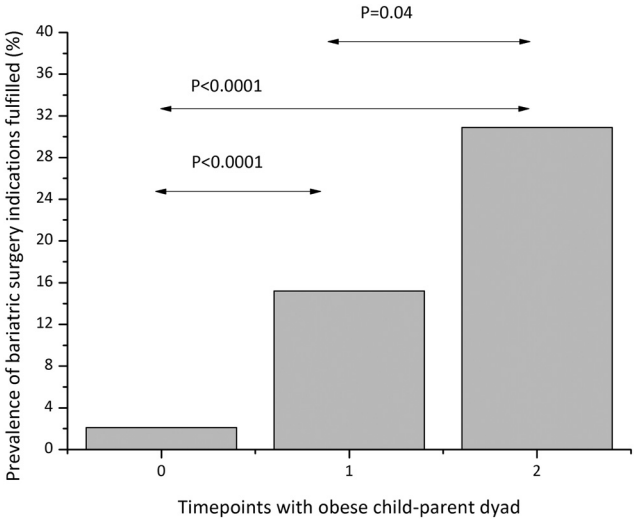

Figure 1. Prevalence of fulfilling EBS criteria according to number of time-points in childhood (1980, 1983) when the child's BMI is higher than age- and sex-specific 85th percentile and either mother's or father's BMI is greater than 30 kg/m^2. *P* values from Poisson regression adjusted with age and sex. N = 1859 because 788 individuals had missing data on own, mother's, or father's BMI in 1983 follow-up.

When the child and one parent were obese at two consecutive two time-points (1980 and 1983), 30.9% of offspring were morbidly obese in adulthood (Figure 1). The risk was significantly increased compared with 15.2% among those whose child-parent dyad was obese at a single time-point and 2.1% among those whose child-parent dyad was not obese at both time-points.

Discussion

The main finding of this study is that among families with both an obese child and an obese parent, the offspring's risk for later EBS in adulthood is markedly increased. Within a large cohort of children followed for over 20 years, we observed that compared to nonobese child-parent dyad, the risk of fulfilling EBS criteria was increased 14-fold in high-risk families. The absolute risk in obese child-normal weight dyads was 21%. Moreover, the risk was doubled (30.9% vs 15.2%) if both members of the dyad were obese at two time-points in childhood, when compared to those with obesity at only one time point.

We and others have previously shown that both own and parental BMI in childhood are independent predictors of later obesity as well as obesity-related disease, such as type 2 diabetes, hypertension, and dyslipidemia. Within the International Childhood Cardiovascular Cohort consortium combining data from the Young Finns Study, Childhood Determinants of Adult Health study (Australia), the Bogalusa Heart Study (Louisiana, United States),

and the Muscatine Study (Iowa, United States), childhood overweight/obesity has been associated with 2.4-fold increased risk of type 2 diabetes, 1.8-fold risk of hypertension, and 1.5-fold risk of dyslipidemia (12). In the Young Finns Study, we have shown that maternal obesity is predictive of 4-fold risk of later type 2 diabetes among offspring (21). In keeping with this, Fall et al (22) have reported in a retrospective analysis that adults with type 2 diabetes had heavier mothers during pregnancy. In the Generation R study (Rotterdam, The Netherlands), parental prepregnancy BMI levels were predictive of higher systolic BP and lower high-density lipoprotein cholesterol levels at age 6 years (23). In a recent retrospective report, Hochner et al (24) reported that maternal BMI before pregnancy and weight gain during pregnancy are associated with offspring BMI, waist circumference, BP, insulin, and dyslipidemia at 32 years of age. The present study extends these findings and indicates that the risk of severe obesity and comorbidities in adult life is substantially increased if both members of the parent-child dyad are obese. A concerning finding is that approximately one-quarter of these children will meet EBS criteria in adulthood. In the context of the US population, this would mean that of the present 65.6 million US children aged 3–18 years, more than 2 million would meet criteria for bariatric surgery (Supplemental Table 1).

The present data provide important information for preventive work in family-based settings. In contrast to genetic- or laboratory-based testing, assessment of parental BMI could be easily and immediately assessed upon visits in child-care units. Importantly, it provides data on both genetic and environmental or lifestyle-derived factors associated with obesity (10, 11). In the case of obesity, the clinical benefit of early recognition of high-risk individuals has been highlighted by the data from this and three other studies within the International Childhood Cardiovascular Cohort consortium showing that even though childhood overweight or obesity is predictive of adult cardiometabolic outcomes, the risks were normalized among those individuals who become nonobese adults (12). Based on our data, the prediction of later risk among children can be significantly improved by adding data on parental adiposity status. It seems that both mother's and father's BMI levels provide useful information, and repeated family measures can provide a more accurate estimate than a single evaluation. Instead, it is not essential to have BMI information available from both parents. These findings would increase the attention of clinicians to the usefulness of measuring parental obesity and thereby facilitate the identification of those who would benefit most from targeted interventions in childhood to prevent and treat obesity-related disorders (25). However, the actual implementation of present findings requires further derivative and validation research.

The main strength of this study is that the data are from a large, randomly selected cohort of adults prospectively followed-up for up to 31 years since childhood. Limitations include the loss of original participants during the long-term follow-up. However, we have previously shown that the follow-up cohort is representative of the original sample (14). Data on parental BMI were gathered using questionnaires rather than actual measurements and further prospective studies, which allow measurement of parental anthropometry (including BMI and generalized and regional adiposity); subsequent longitudinal follow-up of the offspring are warranted. Notwithstanding, the correlation between self-reported BMI and measured values in adults is high ($r = 0.93–0.96$) (26, 27). In addition, when misclassification occurs, it is more likely to occur among those with higher BMI (28), suggesting that a possible bias in the present article may underestimate the true effect size of parental obesity. In particular, some child-mother-father triads had a small number of observations; therefore, these findings should be interpreted cautiously. Finally, because our study cohort was racially homogenous, the generalizability of our results may be limited to white Caucasians and studies in other populations are warranted.

Our data from the longitudinal Cardiovascular Risk in Young Finns Study shows that information on parental BMI in the assessment of childhood obesity improves the prediction of whether that child may become eligible for bariatric surgery in adult life. Parent-child dyads with obesity evident in both generations at two time-points were linked to more than a 30% absolute risk of the child developing later severe obesity. Therefore, these data suggest that family-based evaluations in childhood obesity are critical in recognizing those children most at risk of long-term weight-related health problems.

Acknowledgments

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