Intergenerational Cardiovascular Disease Risk Factors Involve Both Maternal and Paternal BMI

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OBJECTIVE — To examine the association between parental BMI and offspring cardiovascular disease (CVD) risk factors.

RESEARCH DESIGN AND METHODS — The study comprised 940 children (9.5 ± 0.4 years) and 873 adolescents (15.5 ± 0.5 years). Parental weight and height were reported by the mother and the father, and BMI was calculated. CVD risk factors included total (sum of five skinfolds) and central (waist circumference) body fat, blood pressure, cardiorespiratory fitness, insulin sensitivity, total cholesterol, HDL cholesterol, triglycerides, and fibrinogen.

RESULTS — Maternal and paternal BMI were positively associated with total and central fatness in offspring ($P < 0.001$). BMIs of both parents were significantly related to fibrinogen levels ($P < 0.02$), but these associations disappeared when controlling for fatness. There was a positive relationship between maternal and paternal BMI and waist circumference in the offspring regardless of total adiposity and height ($P < 0.001$). Maternal BMI was negatively associated with offspring cardiorespiratory fitness independently of fatness ($P < 0.02$). These relationships persisted when overweight descendants were excluded from the analysis. There were no significant associations between parental BMI and the other CVD risk factors.

CONCLUSIONS — Both maternal and paternal BMI increase CVD risk factors of their offspring, characterized by total and central body fat, and higher maternal BMI was associated with poorer cardiorespiratory fitness. Our findings give further support to the concept that adiposity in parents transmits susceptibility to CVD risk to descendants, which is detectable even in the absence of overweight in offspring.

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Parental obesity substantially increases the risk of obesity in offspring through genetic, biological, or environmental influences (1). The fetal overnutrition hypothesis suggests that maternal obesity and/or gestational diabetes may predispose offspring to increased adiposity in adulthood (2). Human studies showed a greater influence of maternal than paternal BMI on offspring adiposity (3,4). In contrast, others suggested that the contribution of the mother and the father on both prenatal and postnatal programming of intergenerational obesity may be similar according to the genomic imprinting (5).

Most of the studies focused on the relationships of maternal and paternal BMI with their offspring BMI provided contradictory results (3,6,7), and only one study compared the association of maternal and paternal BMI with total body fat in the offspring (8). Whether the parental BMI-offspring body fat relationship applies to other established cardiovascular disease (CVD) risk factors remains to be elucidated.

Excess adiposity leads to increased CVD risk factors and biological pathway alterations as insulin resistance, dyslipemia, hypertension, systemic inflammation, and low cardiorespiratory fitness (9). Therefore, the parental BMI-offspring CVD risk factor relationship may be influenced by the offspring body composition.

The European Youth Heart Study (EYHS) provides an opportunity to better understand the parental-descendant aggregation of CVD factors by controlling for other potential confounding factors that could mediate in this relationship. Therefore, the aim of this study was to determine the association between both maternal and paternal BMI and the offspring CVD risk factors including total and central body fat, cardiorespiratory fitness, insulin sensitivity, blood pressure, blood lipids, and fibrinogen. We also examined the role of offspring adiposity in this relationship.

RESEARCH DESIGN AND METHODS

— The parent and offspring were participants in the Estonian ($n = 975$) and Swedish ($n = 838$) parts of the EYHS, a multicenter study of children (9.5 ± 0.4 years) and adolescents (15.5 ± 0.5 years). The EYHS is a school-based cross-sectional study designed to examine the interactions between personal, environmental, and lifestyle influences on risk factors for future CVD. Study design, selection criteria, and sample calculations have been reported elsewhere (10). Data collection took place from September 1998 to May 1999. In Estonia, the sampling area was the city of Tartu and its surrounding rural area. In Sweden, eight municipalities were chosen for data collection (Botkyrka, Haninge, Huddinge, Nynäshamn, Salem, Södertälje, Tireso, and Örebro). The study protocol was performed in accordance with the ethical standards laid down in the 1961 Declaration of Helsinki (as revised in 2000) and was approved by the Research Ethics

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Committees of the University of Tartu (no. 49/30-199), Örebro County Council (no. 690/98), and Huddinge University Hospital (no. 474/98). Children and adolescents gave verbal assent after procedures were explained, and one parent or legal guardian provided written informed consent. A total of 940 children and 873 adolescents with complete data on body fatness and parental BMI were included in the present study.

Parental BMI

Parents reported their current weight and height, and we calculated their BMI. Parents with a BMI ≥25 kg/m^2 were categorized as being overweight/obese (hereafter called overweight), whereas those with a BMI <25 kg/m^2 were classified as nonoverweight. We also obtained parental age through questionnaire.

Offspring CVD risk factors

Total and central body fat. Height and weight were measured by standardized procedures, and BMI was calculated. BMI *Z* score by sex was also calculated. Overweight and obesity status in children and adolescents was defined following the International Obesity Task Force that proposed sex- and age-adjusted BMI cutoff points (11). Skinfold thickness was measured at the biceps, triceps, subscapular, suprailiac, and triceps surae areas. Waist circumference was taken between the lower rib margin and the iliac crest, at the end of gentle expiration. The sum of five skinfolds and waist circumference were used to estimate total and central body fat, respectively.

Cardiorespiratory fitness. Cardiovascular fitness was determined by a maximum cycle-ergometer test and is expressed as the maximal power output per kilogram of body mass (milliliters per kilogram per minute). This test has been validated previously in children and adolescents (12).

Blood parameters. We assessed total cholesterol, HDL cholesterol, and triglycerides, glucose, and insulin levels as reported elsewhere (13). The homeostasis model assessment was calculated as fasting insulin (milliunits per liter) × fasting glucose (millimoles per liter)/22.5. Fibrinogen was measured using kits from DakoCytomation (Glostrup, Denmark). C-reactive protein and complement factors C3 and C4 were measured in a subsample of 280 Swedish children (n = 164) and adolescents (n = 116) as described elsewhere (14).

Blood pressure. Systolic and diastolic blood pressure was measured with an automatic oscillometric method (Dinamap model XL; Critikon, Tampa, FL). Subjects were in a seated, relaxed position, and recordings were made every 2nd min for 10 min to obtain a systolic recording not varying by > 5 mmHg. The mean value of the last three recordings was used as the resting systolic and diastolic blood pressure in millimeters of mercury. The mean arterial pressure was calculated as follows: diastolic pressure + [0.333 × (systolic blood pressure − diastolic pressure)].

Confounders. Several variables potentially related to BMI or body composition of parents or to the CVD risk factors of their offspring were taken into account (15–17). Socioeconomic status (SES) was assessed via questionnaire and defined by the maternal and paternal educational status, coded as 0 (below university education) and 1 (university education). Birth weight data were collected from parental recall.

SES data were available in 99.3% of children (99.3% in girls and 99.4% in boys) and in 99.5% of adolescents (99.5% of females and 99.5% of males). Birth weight was obtained in 95.0% of children (94.8% of girls and 95.2% of boys) and 96.8% of adolescents (97.1% of females and 96.3% of males).

Puberty stage was assessed by a trained researcher according to Tanner and Whitehouse (18). It was obtained in 98.8% of children (99.4% of girls and 98.1% of boys) and 93.7% of adolescents (93.8% of females and 93.6% of males).

Statistical analysis

Statistical analyses were performed using SPSS software (version 16.0; SPSS, Chicago, IL), and the level of significance was set at 0.05. Data are presented as means ± SD, unless otherwise stated. Variables with skewed distribution, i.e., sum of five skinfolds, waist circumference, insulin, and triglycerides, were transformed to the natural logarithm to obtain a more symmetrical distribution. We compared offspring CVD risk factors differences between parental weight status (none of them overweight, only one overweight, and both overweight) by one-way ANCOVA after adjustment for study location and mother and father age.

Regression analysis was used to examine the association of both maternal and paternal BMI with offspring CVD risk factors. These relationships were analyzed in two separate regression models: model 1 examined the association of maternal or paternal BMI with offspring CVD risk factors after controlling for study location, parent and descendant age and sex, and puberty stage and model 2 was additionally controlled for total body fat (sum of five skinfolds). Each body composition variable and CVD risk factor was examined in a different regression model.

Likewise, those CVD risk factors significantly associated with parental BMI in the regression analysis were examined across maternal and paternal weight status (none of them overweight, only father overweight, only mother overweight, and both parents overweight) by ANCOVA, after adjustment for study location, parent and descendant age, and puberty stage. Waist circumference was additionally adjusted with offspring height to control for current body size.

RESULTS

— Maternal BMI and age were 24.1 ± 6.0 kg/m^2 and 39.3 ± 6.0 years, respectively, and paternal BMI and age were 25.8 ± 3.4 kg/m^2 and 41.8 ± 6.9 years. There were no statistically significant differences in maternal (P = 0.661) and paternal (P = 0.626) BMI according to study location. The clinical and anthropometric characteristics of our sample according to the number of parents with overweight are shown in Table 1.

Maternal and paternal BMI and offspring CVD risk factors

Both maternal and paternal BMI (Table 2) were positively and significantly associated with offspring BMI and skinfold thickness, independently of confounders (P for all <0.001). Both maternal (β = 0.588 [95% CI 0.416–0.760], P < 0.001) and paternal (β = 0.607 [0.386–0.827], P < 0.001) BMIs remained positively related to offspring total body fat after further controlling for cardiorespiratory fitness.

BMI of both parents was associated (P < 0.001 for all) with waist circumference (model 1). These associations remained significant after further adjustment for total body fat (P < 0.001 for both maternal and paternal BMI), and persisted when fitness was added to the model for either maternal BMI (β = 0.068 [95% CI 0.024–0.112], P = 0.002) or paternal BMI (β = 0.114 [0.059–0.169], P < 0.001). The exclusion of overweight descendants (n = 240) did not substantially change the outcome (β = 0.042

Table 1—*Characteristics of the study sample stratified by parental overweight status*

	None overweight*	Only one overweight*	Both overweight*	*P*
n	592	852	344	
Age (years)	12.2 ± 3.0	12.4 ± 3.0	12.8 ± 3.0	0.368
Puberty stage (%)				<0.001
Stage I	48.9	39.8	33.8	
Stage II	7.9	12.7	12.6	
Stage III	5.4	4.5	4.8	
Stage IV	15.4	17.4	18.8	
Stage V	22.4	25.5	30.2	
Female sex (%)	46.4	45.2	47.8	0.706
BMI (kg/m^2)	17.8 ± 2.7	18.9 ± 3.2	20.0 ± 3.4	<0.001
BMI *Z* score	−0.291 ± 0.80	0.079 ± 1.04	0.427 ± 0.9	<0.001
Sum of 5 skinfold thickness (mm)†	41.5 ± 16.8	47.4 ± 21.2	53.2 ± 24.5	<0.001
Waist circumference (cm)†	62.0 ± 7.2	64.3 ± 8.8	66.9 ± 9.3	<0.001
Glucose (mmol/l)	5.00 ± 0.42	5.03 ± 0.55	5.01 ± 0.42	0.370
Insulin (pmol/l)†	54.6 ± 41.5	56.8 ± 35.0	58.3 ± 33.8	0.036
HOMA-IR	1.80 ± 1.54	1.85 ± 1.19	1.93 ± 1.13	0.862
Total cholesterol (mmol/l)	4.31 ± 0.77	4.35 ± 0.79	4.27 ± 0.73	0.401
HDL cholesterol (mmol/l)	1.39 ± 0.30	1.39 ± 0.30	1.36 ± 0.30	0.540
Triglycerides (mmol/l)†	0.77 ± 0.32	0.79 ± 0.33	0.81 ± 0.56	0.461
Fibrinogen (g/l)	2.67 ± 0.50	2.70 ± 0.58	2.78 ± 0.59	0.026
Systolic blood pressure (mmHg)	106.8 ± 10.1	108.0 ± 10.6	109.6 ± 11.7	0.007
Diastolic blood pressure (mmHg)	62.2 ± 7.1	62.4 ± 6.7	62.8 ± 6.4	0.588
Mean arterial pressure‡	77.0 ± 7.3	77.6 ± 7.1	78.4 ± 7.2	0.091
Fitness (ml · kg^{-1} · min^{-1})	42.3 ± 7.3	41.0 ± 7.9	40.3 ± 8.0	<0.001

Data are means ± SD. *Adjusted with maternal and paternal age. †Analysis was performed on log-transformed data, but nontransformed data are presented in the table. ‡Mean arterial pressure = diastolic pressure + [0.333 × (systolic blood pressure − diastolic pressure)].

[0.000–0.084], $P = 0.043$ for maternal BMI and $\beta = 0.097$ [0.045–0.112], $P < 0.001$ for paternal BMI).

Both maternal and paternal BMIs were significantly related to fibrinogen levels ($P < 0.02$), but these associations disappeared when fatness was added to the model. Similarly, BMIs of parents were significantly associated with their offspring C-reactive protein and complements C3 and C4 (all $P < 0.05$), but these associations became nonsignificant after further controlling for fatness (all $P > 0.1$). Systolic blood pressure and insulin increased linearly with the number of parents who were overweight (Table 1), but there were no statistically significant associations with maternal and paternal BMI (Table 2). The other CVD risk factors (glucose, homeostasis model assessment, diastolic blood pressure, total and HDL cholesterol, and triglycerides) were not associated with parental BMI.

Maternal BMI was negatively associated with cardiorespiratory fitness of the offspring in model 1 ($P < 0.001$), and this relationship remained significant after further controlling for fatness ($P < 0.02$, model 2). This relationship persisted when overweight descendants were excluded from the analysis ($\beta = -0.071$ [95% CI −0.140 to −0.002], $P = 0.043$). There was a significant relationship between paternal BMI and offspring fitness ($P < 0.001$), which was abolished when the analysis was controlled for fatness.

We also examined whether the associations of maternal and paternal BMI with CVD risk factors were modified after further controlling for other confounders such as SES and birth weight. The outcome did not change substantially (data not shown).

Finally, we examined the possible transgenerational association of parental BMI on CVD risks factors in a sex-specific trait (father-son and mother-daughter). The effect of either paternal or maternal BMI on CVD risk factors was similar for both their sons and daughters (data not shown).

Maternal and paternal weight status and offspring CVD risk factors

Figure 1 shows the combined effect of weight status of both parents on total and central body fat and on cardiorespiratory fitness of descendants by age-groups and sex groups. Offspring with two overweight parents had higher BMI, fatness, and waist circumference values than descendants with a single overweight parent ($P < 0.001$ for all). Total and central fatness markers were higher in descendants with a single overweight parent than in the referent (none overweight) group ($P < 0.001$ for all). There were no significant differences in BMI, sum of five skinfolds, and waist circumference between offspring with only the mother or only the father overweight. The results were consistent in children and adolescents and in boys and girls.

Descendants with two parents overweight had lower cardiorespiratory fitness than either their peers whose mother and father were both nonoverweight ($P < 0.001$) or their peers whose father was overweight ($P = 0.001$). There were no differences between offspring with two parents overweight or only mother overweight ($P = 0.880$). Descendants whose mother was overweight had lower fitness than those whose father was overweight ($P = 0.004$). These relationships were not altered substantially when examined separately by age-group or sex.

CONCLUSIONS

In the present study we examined the association between both maternal and paternal BMI and common CVD risk factors in 1,813 Estonian and Swedish youth participating in the EYHS. The results indicated that some of the relationships between parental BMI and offspring CVD factors, i.e., fibrinogen or systolic blood pressure, are mediated by familial aggregation of adiposity. Likewise, our findings suggest that both maternal and paternal BMI are associated with total and central adiposity in the offspring, whereas increased maternal BMI was related to poorer cardiorespiratory fitness regardless of fatness, even in absence of overweight. To the best of our knowledge, there are no previous studies examining the association between either parental weight status or BMI and CVD risk factors in the offspring.

Maternal and paternal BMI and fibrinogen and blood pressure in the offspring

Fibrinogen and systolic blood pressure levels were higher in descendants with one or two overweight parents than in the referent group (none of parents overweight). Likewise, descendants' fibrinogen levels, as well as C-reactive protein and complements C3 and C4, increase

Table 2—*Multiple regression coefficients (β) and SEM examining the association of maternal and paternal BMI with cardiovascular disease risk factors in offspring*

	Maternal BMI								Paternal BMI							
	Model 1				Model 2				Model 1				Model 2			
	β	SEM	*P*	*n*	β	SEM	*P*	*n*	β	SEM	*P*	*n*	β	SEM	*P*	*n*
BMI (kg/m^2)	0.143	0.013	<0.001	1,956					0.152	0.017	<0.001	1,734				
Sum of 5 skinfolds (mm)*	0.957	0.100	<0.001	1,956					0.900	0.132	<0.001	1,691				
Waist circumference (cm)*	0.280	0.031	<0.001	1,956	0.059	0.022	0.007	1,910	0.309	0.040	<0.001	1,734	0.104	0.028	<0.001	1,691
Systolic blood pressure (mmHg)	0.079	0.052	0.129	1,955	0.027	0.054	0.625	1,909	0.114	0.066	0.086	1,733	0.062	0.068	0.365	1,690
Diastolic blood pressure (mmHg)	0.000	0.037	0.994	1,955	−0.015	0.038	0.702	1,909	0.068	0.046	0.146	1,733	0.059	0.049	0.257	1,690
Blood pressure (mmHg)	0.026	0.038	0.484	1,955	0.003	0.039	0.944	1,955	0.083	0.048	0.082	1,733	0.057	0.050	0.248	1,690
Insulin (pmol/l)*	−0.049	0.219	0.746	1,277	−0.405	0.222	0.146	1,267	−0.315	0.269	0.927	1,088	−0.359	0.260	0.927	1,160
Glucose (mmol/l)	−0.002	0.003	0.502	1,790	−0.004	0.003	0.201	1,754	−0.004	0.004	0.292	1,576	−0.005	0.004	0.143	1,543
HOMA-IR	−0.004	0.008	0.636	1,275	−0.015	0.008	0.065	1,266	−0.007	0.010	0.658	1,096	−0.015	0.010	0.129	1,087
Total cholesterol (mmol/l)	−0.002	0.004	0.576	1,791	−0.007	0.004	0.114	1,755	0.009	0.006	0.108	1,577	0.006	0.006	0.263	1,544
Triglycerides (mmol/l)*	0.001	0.002	0.573	1,790	−0.001	0.002	0.170	1,754	0.000	0.003	0.950	1,576	−0.002	0.003	0.446	1,543
HDL cholesterol (mmol/l)	−0.002	0.002	0.364	1,790	0.001	0.002	0.671	1,754	0.000	0.002	0.647	1,576	0.000	0.002	0.886	1,543
Fibrinogen (g/l)	0.008	0.004	0.039	1,262	0.002	0.004	0.664	1,252	0.012	0.005	0.011	1,089	0.007	0.005	0.112	1,079
Fitness (ml · kg^{-1} · min^{-1})	−0.262	0.036	<0.001	1,846	−0.077	0.032	0.015	1,801	−0.176	0.046	<0.001	1,638	−0.008	0.040	0.845	1,596

Model 1: maternal or paternal BMI adjusted for country, offspring sex, age, and puberty stage, and mother or father age. Model 2: model 1 + fatness of the offspring. *Analysis was performed on log-transformed data, but nontransformed data are presented in the table.

with maternal and paternal BMI, but these associations were no longer significant when controlling for fatness of the offspring. A high concentration of fibrinogen and elevated systolic blood pressure are considered major CVD risk factors, which are linked to adiposity in children and adolescents (9). A previous report that showed higher levels of C-reactive protein in nonobese offspring of obese parents (19) concluded that the inflammation share could be transmitted from obese parents to offspring, because this proinflammatory state is a potential precursor of obesity. However, in this study, analysis was not adjusted with total or central body fat, which are independently associated with low-grade inflammation (9).

Maternal and paternal BMI and total and central adiposity in the offspring

The present study also showed that total and central fatness were significantly higher in youth with one or two overweight parents than in those with nonoverweight parents but that that the influence of maternal weight status on offspring fatness was not meaningfully greater than the paternal weight status influence. Stratified analysis by age-groups or sex groups indicates that the associations are consistent for children and adolescents and for boys and girls. Moreover, the effect of both parental BMIs on offspring waist circumference was independent of total body fat and height, and the outcome persisted when overweight children were excluded from the analysis.

Four large studies using BMI as an indicator of obesity reported contradictory results regarding maternal and paternal associations with offspring adiposity. In a large Finnish cohort (2), offspring BMI after 3 years was associated equally strongly with paternal and maternal BMI. In an Australian birth cohort (3), maternal BMI was more strongly associated with offspring BMI than was paternal BMI. However, in the Avon Longitudinal Study of Parents and Children (ALSPAC) study (6), associations were similar in mothers and fathers. Lawlor et al. (8) showed that the maternal-paternal difference in their associations with offspring fat mass, measured by dual-energy X-ray absorptiometry, was small. Finally, Li et al. (7) reported that excessive BMI gain of parents during childhood and adulthood was associated with higher BMI and obesity risk in the offspring.

Findings from longitudinal studies indicated that although there is evidence that patterns of lifestyle factors related to obesity often co-occur within families, these parents-descendants fatness associations may reflect a combined influence of both genetic and lifestyle factors. Indeed, early establishment of lifestyle patterns could be maintained into adulthood and transmitted to offspring (7).

Kivimäki et al. (2) reported that associations of maternal BMI similar to that of paternal BMI with offspring BMI would probably support similar mechanisms for mothers and fathers. Thus, they argued that genetic inheritance and shared familial environment are mechanisms likely to act similarly with respect to mothers and fathers, whereas fetal overnutrition or undernutrition

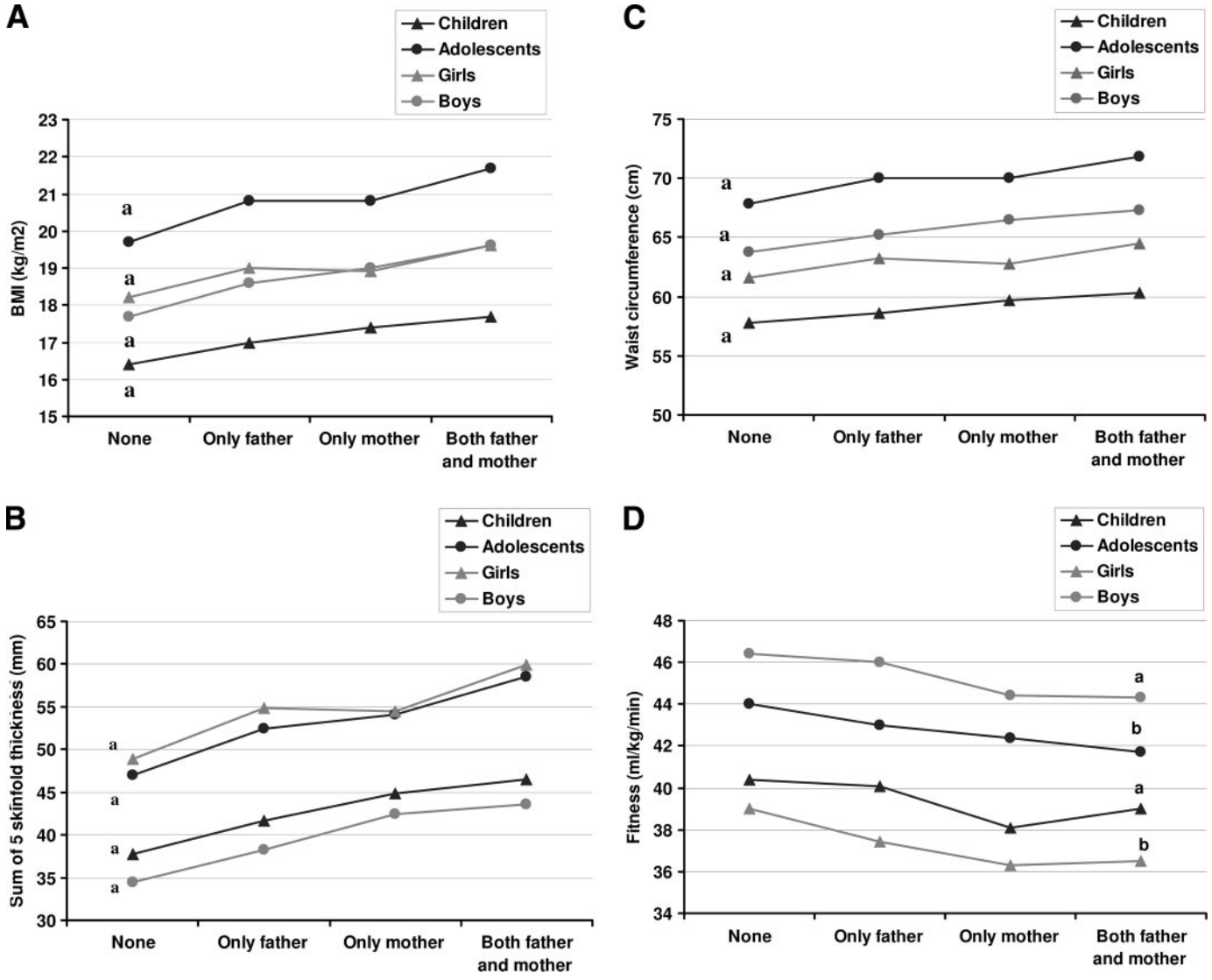

Figure 1—*BMI* (A)*, sum of five skinfold thickness* (B)*, waist circumference* (C)*, and cardiorespiratory fitness* (D) *in offspring (children and adolescents and males and females) according to their maternal and paternal weight status and adjusted by study location, sex when examining two sexes together, age, puberty stage, and height.* $^{a}P_{trend} < 0.01$ *in all;* $^{b}P_{trend} < 0.001$*. Both father and mother, both parents overweight; None, none of parents overweight; Only father, only father overweight; Only mother, only mother overweight.*

could be a mother-specific effect. Earlier, another possibility, the transgenerational response, had been considered in the gene-environment interplay underlying common diseases. This requires a mechanism for transmitting environmental exposure information that then alters gene expression in the next generation(s). The epigenetic inheritance has to be considered as a possible mechanism, particularly when transmission is down the male line (20). Our results do not concur with previous studies, suggesting a sex-specific maleline transgenerational effect in humans (20,21). Thus, the effect of both paternal and maternal BMI was similar in either their daughters or sons.

Maternal and paternal BMI and cardiorespiratory fitness in the offspring

We observed that cardiorespiratory fitness in offspring was associated with maternal BMI regardless of the descendant's fatness. Interestingly, the results persisted after exclusion of overweight youth. One unexpected finding was that there was a higher influence of maternal than paternal BMI or weight status on offspring fitness. Whereas the association of maternal BMI on offspring fitness was independent of total body fat, the effect of paternal BMI disappeared when controlling for fatness. This difference in the effect of maternal and paternal BMI on offspring fitness was observed when either parental BMI was treated as a dichotomous or continuous variable. Therefore, descendants with only a mother overweight had lower cardiorespiratory fitness than those with only a father overweight, whereas no differences were found for the group with two parents overweight. As far as we are aware, this is the first study to examine the relationship between parental BMI and cardiorespiratory fitness in the offspring. One possible mechanism explaining the stronger effect of maternal BMI on descendant fitness could be related to the mitochondrial background influencing V_{O_2}max (22). Some proteins of the oxidative phosphorylation system are encoded by mi-

tochondrial DNA (mtDNA). Interestingly, mtDNA shows regional genetic variations that are considered to be one of the important factors in adaptation to environmental conditions at a much higher rate than nuclear DNA (23). Thus, we could hypothesize that maternal lifestyle and adiposity could affect this mtDNA variability, decreasing cardiorespiratory fitness in the offspring.

Study limitations and strengths

One limitation of the present study is that it relies on the use of reported measures of both maternal and paternal weight and height when their offspring was recruited. Nevertheless, studies relating reported to measured weight and height suggest that reporting, especially in young adults (<60 years) (24) is generally accurate, with no evidence of substantial sex-related differences (25). Further, we cannot draw any conclusion regarding how much of the relationship between parental BMI and CVD risk is due to genetic or environmental factors or to an interaction between them. Our study has as an advantage the use of a relatively large sample and the inclusion of several potential confounders of lifestyle and behavior in the analysis.

In summary, central and total adiposity, as well as cardiorespiratory fitness, are important risk factors of CVD already in children and adolescents and are strong predictors of the presence of the metabolic syndrome early in childhood. Further, the effect of higher parental BMI on offspring increased central body fat and poor fitness regardless of total adiposity may provide insight into the etiology of CVD.

Our findings support the idea that much CVD risk (increased total and central adiposity and poor fitness) in childhood and adolescence is predictable at or soon after birth. Thus, babies at high risk could be defined, even at birth, from the maternal and paternal history of adiposity. Because childhood adiposity prevalence is substantially increasing across generations, these results could be interpreted as an intergenerational increase of obesity and CVD risk through successive generations independent of further genetic and environmental factors. Because parental overweight is one of the strongest predictors of childhood adiposity and maternal overweight of fitness, policies and interventions should focus on parents to provide them with an environment to support healthy behaviors for themselves and their offspring.

Finally, both maternal and paternal BMI, as continuous form and weight status, increase CVD risk factors of their offspring characterized by total and central fatness, whereas higher maternal BMI was additionally associated with poor cardiorespiratory fitness regardless of youth fatness. Our findings lend further support to the concept that adiposity in parents transmits susceptibility to a higher CVD risk to their descendants, which is detectable even in the absence of overweight in offspring.

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