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Association between paternal physique and obesity in children at the age of 3 years: the Japan Environment and Children's Study

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Abstract

Obesity during development has been reported to be a determinant factor in the future development of non-communicable diseases (NCDs). Parental obesity is suggested to be a predictor of children's obesity, and it is important to consider parental factors to prevent NCDs in the progeny. Previously, we showed that paternal height had a stronger association with infant birth weight than paternal body mass index (BMI) in the Japanese population. However, only a few studies have examined the association between paternal physique and postnatal obesity. This study aimed to investigate the association between parental physique and obesity in children at the age of 3. This study used fixed data on 33,291 parent–child pairs from the Japan Environment and Children's Study, an ongoing national birth cohort study. The association between paternal physique (BMI and height) and children's obesity at the age of 3 was examined using multivariate logistic regression analysis. The higher the paternal BMI quartiles, the higher the odds ratio for obesity in male and female children at 3 years of age ($P < 0.0001$). However, paternal height quartiles were not associated with male or female obesity. These results differ from the association between paternal physique and infant birth weight, and it is possible that prenatal epigenetic and environmental factors of paternal origin were responsible for the differences between these two studies. The association between paternal BMI and obesity in children at the age of 3 suggests that paternal factors may be involved in the development of NCDs in future progeny.

Introduction

The developmental origins of health and disease (DOHaD) hypothesis is the theory that prenatal, genetic, and postnatal environmental factors play a role in developing non-communicable diseases (NCDs) in progeny, primarily through epigenetic changes[1]. Obesity among children has increased dramatically worldwide since 1990[2], and obesity during development and physiological plasticity are known to be determining factors in the future development of NCDs[3]. The incidence of NCDs is rapidly increasing, and NCD-related deaths have been predicted to account for more than 75% of global deaths by 2030[4].

The importance of focusing on obesity in children can be explained in terms of early adiposity rebound (AR). AR refers to an increase in body mass index (BMI) after a nadir, and AR occurring before the age of 5 is called early AR[5]. Early AR is a useful marker for predicting the risk of obesity and NCDs in childhood and adulthood[6,7]. A longitudinal study in Japan showed that children with early AR are predisposed to developing metabolic syndrome in the future[8].

Various factors contribute to obesity in young children, including dietary habits, physical inactivity, genetic predisposition, socioeconomic status, and environmental influences[9]. It has also been shown that parental obesity is one of the indicators associated with childhood obesity[10]. While the association between maternal physique and childhood obesity has been extensively studied, reports on the relationship between paternal physique and childhood obesity remain limited[11,12]. Contrarily, the significance of paternal factors in influencing the health and development of progeny, known as Paternal Origins of Health and Disease (POHAD), is gaining attention[13].

Since the 1980s, BMI averages have been rising worldwide[14], and in recent years, annual BMI trends for Japanese men have also continued to increase[15,16]. Therefore, it is important to gain insight into the paternal factors that may influence children's obesity.

Our previous study demonstrated that paternal height correlates with infant birth weight more strongly than paternal BMI in the general Japanese population[17]. However, the literature lacks evidence regarding how this association may evolve during the child's growth, particularly in early childhood. Considering the points, we conducted this study to elucidate the association between obesity in children at the age of 3, corresponding to the period of early AR, and paternal physique, specifically height and BMI.

Materials and methods

Study participants and data collection

The Japan Environment and Children's Study (JECS) is an ongoing nationwide birth cohort study. It is a large-scale epidemiological research project that is being funded. The main aim of the JECS is to investigate the environmental factors that influence the health and development of children. Pregnant women were recruited from 15 regional centers nationwide for the JECS between January 2011 and March 2014.

The inclusion criteria were (1) residing in the Study Area at the time of recruitment and planning to deliver at a cooperating healthcare provider; (2) expected delivery date after August 1, 2011; and (3) ability to comprehend Japanese language and completing the self-administered questionnaire.

Cooperating healthcare providers and local government offices, which issued Maternal and Child Health Handbooks, were instructed to contact pregnant women and register those willing to participate. The pregnant women's partners (children's fathers) were encouraged to participate whenever possible. Self-administered questionnaires, which were completed in the first trimester (MT1) and second/third trimester (MT2), collected information regarding demographic factors, weight and height, medical and obstetric history, physical and mental health, lifestyle, occupation, environmental exposures at home and in the workplace, housing conditions, and socioeconomic status. Between the mother's early pregnancy (MT1) and 1 month after delivery, their partners (children's fathers) were asked to complete a questionnaire (FT1). The father's questionnaire (FT1) collected information on demographic factors, weight and height, medical history, physical and mental health, lifestyle, occupation, and environmental exposure at home and in the workplace. After birth, self-administered questionnaires regarding children's physical measurements and nutritional status (including breastfeeding) were collected every 6 months until the children reached 13. Information transcribed from the medical records such as parity, conception method, delivery mode, obstetric history, and birth weight was transcribed by physicians, midwives/nurses, and/or research coordinators by standard operating procedures. This study used dataset jecs-ta-20190930, released in October 2019 and revised in November 2022. Serial measurements such as paternal and maternal weight (kg), height (cm), and BMI (kg/m^2) were obtained from the first trimester questionnaire (MT1 or FT1). Obesity in children at the age of 3 was defined as BMI $\geq$ 95th percentile concerning the BMI curve given by the Japanese Society for Pediatric Endocrinology[18].

The JECS protocol was reviewed and approved by the Ministry of the Environment's Epidemiological Studies Review Board and the Ethics Committees of all participating institutions. It was conducted in accordance with the principles of the Declaration of Helsinki and other national regulations and guidelines. Written informed consent was obtained from all the participants.

Statistical analysis

We divided paternal BMI into quartiles and analyzed the differences in paternal, maternal, and children's characteristics according to the paternal BMI. Continuous variables were analyzed using the Kruskal–Wallis test and categorical variables using X^2 tests. The association between paternal BMI and obesity in children at the age of 3 was examined using multivariate logistic regression analysis. Odds ratios (ORs) and 95% confidence intervals (95% CIs) for obesity in children at the age of 3 according to paternal BMI were calculated, with the lowest group (<21.2 kg/m^2) in the paternal BMI quartile as the reference. Multivariate logistic regression analyses were adjusted for paternal and maternal age at the time of answering MT1 and FT1, paternal and maternal smoking status, paternal and maternal alcohol consumption, paternal and maternal highest level of education, maternal BMI at the time of answering MT1, gestational age at delivery, parity, conception method, delivery mode, gestational weight gain, marital status, family income, gestational age at the time of answering MT1 and FT1, age in months at the time of physical measurements of the 3 years old, and lactation status at 6 months of age. Next, the linear associations between paternal BMI and the proportion of children with obesity at the age of 3 were analyzed using the Cochran–Armitage test.

Next, we divided paternal height into quartiles and analyzed the differences in paternal, maternal, and children's characteristics according to paternal height. The association between paternal height and obesity in children at the age of 3 was examined using multivariate logistic regression analysis. ORs and 95% CIs for obesity in children at the age of 3 according to paternal height were calculated, with the lowest group (<168 cm) in the paternal height quartile as the reference. Multivariate logistic regression analyses were adjusted for paternal and maternal age at the time of answering MT1 and FT1, paternal and maternal smoking status, paternal and maternal alcohol consumption, paternal and maternal highest level of education, maternal BMI at the time of answering MT1, gestational age at delivery, parity, conception method, delivery mode, gestational weight gain, marital status, family income, gestational age at the time of answering MT1 and FT1, age in months at the time of physical measurements of 3 years old, and lactation status at 6 months of age. Next, the linear associations between paternal height and obesity in children at the age of 3 were analyzed using the Cochran-Armitage test for trend. Statistical significance was set at $P < 0.05$. All statistical analyses were performed using the SAS software, version 9.4 (SAS Institute Inc., Cary, NC, USA).

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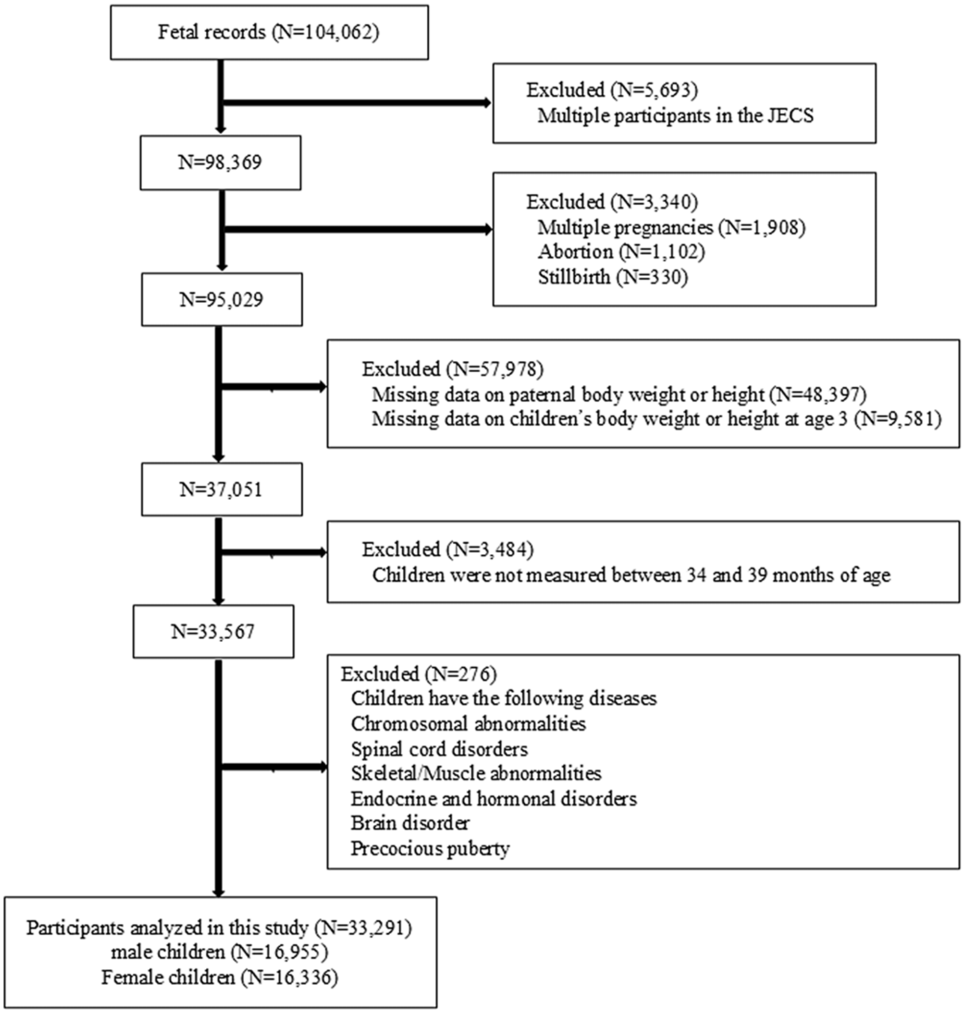

Figure 1. Flow chart of the selection process for participants in this study.

Results

Characteristics of the parents and children

Of the 104,062 fetal records, 70,771 (68.0%) were excluded due to multiple participation, multiple pregnancies, abortion, stillbirth, missing paternal and 3 years old children's weight or height data, or other reasons (Fig. 1). Serial measurements of the children aged 3 years, such as weight (kg), height (cm), and BMI (kg/m^2) were obtained from the dataset only for those in whom measurements were made between 34 and 38 week and 6 days of gestation. The total number of parent–child pairs included in the study was 33,291. The numbers of male and female children were 16,955 and 16,336, respectively.

First, to examine the characteristics of parents and their children, the participants were divided into four groups according to the paternal BMI of male and female children, respectively. Paternal BMI of male children was as follows: Quartile 1 (<21.2 kg/m^2), Quartile 2 (21.2–22.8 kg/m^2), Quartile 3 (22.9–25.1 kg/m^2), and Quartile 4 (≥25.2 kg/m^2). Paternal BMI of female children were Quartile 1 (<21.1 kg/m^2), Quartile 2 (21.1–22.9 kg/m^2), Quartile 3 (23.0–25.2 kg/m^2), and Quartile 4 (≥25.3 kg/m^2). According to the paternal BMI of male children, the number of participants in Quartiles 1–4 was 4,269, 4,133, 4,312, and 4,241, respectively. According to the paternal BMI of female children, the number of participants in Quartiles 1–4 was 3,989, 4,168, 4,127, and 4,052, respectively. Supplementary Tables S1 and S2 demonstrate the paternal characteristics of male and female children according to the paternal BMI categories, respectively. In the context of paternal characteristics of male and female children, the higher the paternal BMI, the higher the paternal age at the time of answering FT1. Moreover, the higher the paternal BMI, the higher the percentage of smokers and drinkers (Supplementary Tables S1 and S2). Supplementary Tables S3 and S4 demonstrate the maternal characteristics of male and female children according to paternal BMI categories, respectively. Regarding maternal characteristics of male and female children, the higher the paternal BMI, the higher the maternal age and BMI at the time of answering MT1 (Supplementary Tables S3 and S4). Supplementary Tables S3 and S4 also show that the higher the paternal BMI, the higher the rates of prolificacy, cesarean section, formula milk feeding, gestational diabetes mellitus, and hypertensive disorders of pregnancy.

Tables 1 and 2 show the characteristics of male and female children according to paternal BMI categories, respectively. Among the characteristics of male children at the age of 3, higher paternal BMI was associated with higher body weight, height, BMI, and birth weight of the children (Table 1). Table 2 shows results similar to those in Table 1. The median BMI and proportion of male children at the age of 3 were 16.0% and 10.4%, respectively. The proportion of obesity in male children at the age of 3 according to paternal BMI categories was Quartile 1, 7.0%; Quartile 2, 9.7%; Quartile 3, 11.4%; and Quartile 4, 13.5% (Table 1). The median BMI and proportion of female children at the age of 3 were 15.9%

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Table 1. Male children's characteristics by paternal BMI

	Total	Paternal BMI (kg/m^2) Quartile 1 (<21.2)	Quartile 2 (21.2–22.8)	Quartile 3 (23.9–25.1)	Quartile 4 (≥25.2)	*p*-value
Number of subjects	16,955	4,269	4,133	4,312	4,241	
Gestational age at birth, weeks	39 (38–40)	39 (38–40)	39 (38–40)	39 (38–40)	39 (38–40)	0.74
Birth weight, g	3,074 (2,834–3,330)	3,042 (2,810–3,294)	3,060 (2,824–3,306)	3,083.5 (2,842–3,334)	3,116 (2,864–3,381)	<0.0001
LBW, *n* (%)	1,129 (6.7)	298 (7.0)	312 (7.6)	261 (6.1)	258 (6.1)	0.01
Macrosomia, *n* (%)	170 (1.0)	42 (1.0)	34 (0.8)	50 (1.2)	44 (1.0)	0.5
Preterm birth at less than 37 weeks of gestation	816 (4.8)	201 (4.7)	202 (4.9)	209 (4.9)	204 (4.8)	0.98
at less than 34 weeks of gestation	153 (0.9)	33 (0.8)	41 (1.0)	32 (0.7)	47 (1.1)	0.22
at less than 28 weeks of gestation	48 (0.3)	10 (0.2)	14 (0.3)	13 (0.3)	11 (0.3)	0.81
Lactation status at 6 months old, *n* (%)						<0.0001
Breast milk only	9,315 (54.9)	2,407 (59.3)	2,351 (59.3)	2,392 (58.4)	2,165 (53.7)	
Breast milk and formula milk	4,121 (24.3)	1,000 (24,7)	1,015 (25.6)	1,061 (25.9)	1,045 (25.9)	
Formula milk only	2,711 (16,0)	650 (16,0)	597 (15.1)	645 (15.7)	819 (20.3)	
Age in months at the time of the survey at age 3, months	35 (35–36)	35 (35–36)	35 (35–36)	35 (35–36)	35 (35–36)	0.42
Body weight at age 3, kg	13.7 (12.8–14.6)	13.5 (12.5–14.3)	13.6 (12.8–14.5)	13.8 (12.9–14.7)	14.0 (13.0–15.0)	<0.0001
Height at age 3, cm	92.3 (90.0–94.8)	92.0 (90.0–94.3)	92.2 (90.0–94.7)	92.4 (90.2–94.8)	92.8 (90.4–95.0)	<0.0001
BMI at age 3, kg/m^2	16.0 (15.3–16.8)	15.8 (15.1–16.6)	16.0 (15.2–16.8)	16.0 (15.4–16.9)	16.2 (15.4–17.0)	<0.0001
Obesity at age 3, *n* (%)	1,763 (10.4)	299 (7.0)	399 (9.7)	491 (11.4)	574 (13.5)	<0.0001

Data are expressed as median (IQR) or number (percent).
BMI, body mass index; LBW, low birth weight.

Table 2. Female children's characteristics by paternal BMI

	Total	Paternal BMI (kg/m^2) Quartile 1 (<21.1)	Quartile 2 (21.1–22.9)	Quartile 3 (23.0–25.2)	Quartile 4 (≥25.3)	*p*-value
Number of subjects	16,336	3,989	4,168	4,127	4,052	
Gestational age at birth, weeks	39 (38–40)	39 (38–40)	39 (38–40)	39 (38–40)	39 (38–40)	0.15
Birth weight, g	2,984 (2,750–3,230)	2,960 (2,740–3,200)	2,978 (2,738–3,214)	2,988 (2,758–3,230)	3,013 (2,766–3,275)	<0.0001
LBW, *n* (%)	1,398 (8.6)	334 (8.4)	386 (9.3)	353 (8.6)	325 (8.0)	0.23
Macrosomia, *n* (%)	92 (0.6)	10 (0.3)	23 (0.6)	31 (0.8)	28 (0.7)	0.5
Preterm birth at less than 37 weeks of gestation	569 (3.5)	113 (24.4)	149 (3.6)	160 (3.9)	147 (3.6)	0.064
at less than 34 weeks of gestation	121 (0.7)	19 (0.5)	31 (0.7)	41 (1.0)	30 (0.7)	0.061
at less than 28 weeks of gestation	40 (0.2)	7 (0.2)	7 (0.2)	16 (0.4)	10 (0.3)	0.15
Lactation status at 6 months old, *n* (%)						0.00025
Breast milk only	9,232	2,322 (60.7)	2.421 (60.3)	2,318 (59.2)	2,171 (56.5)	

(*Continued*)

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Table 2. (*Continued*)

	Total	Paternal BMI (kg/m^2) Quartile 1 (<21.1)	Quartile 2 (21.1–22.9)	Quartile 3 (23.0–25.2)	Quartile 4 (≥25.3)	*p*-value
Breast milk and formula milk	3,754	875 (22.9)	965 (24.0)	950 (24.2)	964 (25.1)	
Formula milk only	2,614	629 (16.4)	628 (15.7)	651 (16.6)	706 (18.4)	
Age in months at the time of the survey at age 3, months	35 (35–36)	35 (35–36)	35 (35–36)	35 (35–36)	35 (35–36)	0.0013
Body weight at age 3, kg	13.2 (12.3–14.2)	13.0 (12.1–14.0)	13.1 (12.3–14.0)	13.3 (12.4–14.2)	13.5 (12.6–14.5)	<0.0001
Height at age 3, cm	91.2 (89.0–93.5)	90.9 (88.9–93.0)	91.0 (89.0–93.5)	91.4 (89.2–93.8)	91.7 (89.5–94.0)	<0.0001
BMI at age 3, kg/m^2	15.9 (15.1–16.7)	15.7 (14.9–16.5)	15.8 (15.1–16.6)	15.9 (15.1–16.7)	16.1 (15.3–16.9)	<0.0001
Obesity at age 3, *n* (%)	1,544 (9.5)	284 (7.1)	356 (8.5)	395 (9.6)	509 (12.6)	<0.0001

Data are expressed as median (IQR) or number (percent).
BMI, body mass index; LBW, low birth weight.

and 9.5%, respectively. The proportion of obesity in female children at the age of 3 according to paternal BMI categories was Quartile 1, 7.1%; Quartile 2, 8.5%; Quartile 3, 9.6%; and Quartile 4, 12.6% (Table 2). The higher the paternal BMI, the higher the percentage of obesity at the age of 3 among male and female children.

Next, the participants were divided into four groups according to the paternal height of male and female children. The paternal height of male children was classified as follows: Quartile 1 (<168.0 cm), Quartile 2 (168.0–171.9 cm), Quartile 3 (172.0–175.4 cm), and Quartile 4 (≥175.5 cm). The paternal height of female children was categorized as follows: Quartile 1 (<168.0 cm), Quartile 2 (168.0–171.9 cm), Quartile 3 (172.0–175.4 cm), and Quartile 4 (≥175.5 cm). The number of participants in Quartiles 1–4 according to paternal height of male children were 3,671, 4,717, 4,310, and 4,257, respectively. The number of participants in Quartiles 1–4, according to the paternal height of female children, was 3,584, 4,450, 4,138, and 4,164, respectively. Supplementary Tables S5 and S6 show the paternal characteristics of male and female children according to paternal height categories, respectively. In the context of paternal characteristics of male and female children, the higher the paternal height, the higher the paternal body weight when answering FT. However, no such features were found in the paternal BMI (Supplementary Tables S5 and S6). Supplementary Tables S5 and S6 also show that the higher the paternal height, the higher the percentage of drinkers, and the higher the level of education. Supplementary Tables S7 and S8 demonstrate the maternal characteristics of male and female children according to paternal height categories, respectively. Regarding maternal characteristics of male and female children, the higher the paternal height, the higher the maternal height and BMI at the time of answering MT1 (Supplementary Tables S7 and S8). Supplementary Tables S7 and S8 also show that the higher the paternal height, the higher the percentage of the highest level of education and annual income. Tables 3 and 4 show the characteristics of male and female children according to paternal height categories, respectively. In male children, the higher the paternal height, the higher the body weight and height at the age of 3, along with the birth weight (Table 3). Table 4 shows results similar to those in Table 3. There were no significant differences among the four paternal height categories in the distribution of obesity in male and female children at the age of 3 years.

Association between paternal physique and obesity in children at the age of 3 years

Table 5 shows crude and adjusted ORs and 95% CI for obesity in children at the age of 3 about paternal BMI categories. In the context of male children, compared with the reference group (Quartile 1: paternal BMI < 21.2 kg/m^2), adjusted ORs of obesity in children at the age of 3 was 1.31 (95% CI 1.10–1.56) for Quartile 2 (paternal BMI 21.2–22.8 kg/m^2), 1.61 (95% CI 1.36–1.90) for Quartile 3 (paternal BMI 22.9–25.1 kg/m^2), and 1.77 (95% CI 1.50–2.09) for Quartile 4 (paternal BMI ≥ 25.2 kg/m^2). In terms of female children, compared with the reference group (Quartile 1: paternal BMI < 21.1 kg/m^2), adjusted ORs of obesity in children at the age of 3 was 1.26 (95% CI 1.05–1.50) for Quartile 2 (paternal BMI 21.1–22.9 kg/m^2), 1.39 (95% CI 1.17–1.66) for Quartile 3 (paternal BMI 23.0–25.2 kg/m^2), and 1.66 (95% CI 1.40–1.97) for Quartile 4 (paternal BMI ≥ 25.3 kg/m^2). Higher paternal BMI was associated with higher odds of obesity in children at the age of 3. This trend was significant among males and females ($P < 0.0001$).

Table 6 shows crude and adjusted ORs and 95% CI for obesity in children at the age of 3 according to paternal height categories. Paternal height showed no association with the ORs of obesity in male and female children at the age of 3.

Discussion

The present study showed that paternal BMI is associated independently with obesity in children at the age of 3 years. In contrast, paternal height showed no association with obesity in children at the age of 3 years. Although the association between paternal BMI and obesity in children at the age of 3 years was consistent with previous studies from different populations and ethnicities[19], there have been no previous studies on the association between paternal height and obesity in children at the age of 3 years. This study was the first to explore this association.

Several studies have shown an association between paternal BMI and obesity among children. Moreover, a stronger association

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Table 3. Male children's characteristics by paternal height

	Total	Paternal height (cm) Quartile 1 (<168.0)	Quartile 2 (168.0–171.9)	Quartile 3 (172.0–175.4)	Quartile 4 (≥175.5)	*p*-value
Number of subjects	16,955	3,671	4,717	4,310	4,257	
Gestational age at birth, weeks	39 (38–40)	39 (38–40)	39 (38–40)	39 (38–40)	39 (38–40)	0.55
Birth weight, g	3,074 (2,834–3,330)	3,030 (2,800–3,276)	3,068 (2,826–3,314)	3,080 (2,840 –3,334)	3,113 (2,872–3,388)	<0.0001
LBW, *n* (%)	1,129 (6.7)	287 (7.8)	345 (7.3)	270 (6.3)	227 (5.3)	<0.0001
Macrosomia, *n* (%)	170 (1.0)	33 (0.9)	49 (1.0)	39 (0.9)	49 (1.2)	0.61
Preterm birth at less than 37 weeks of gestation	816 (4.8)	184 (5.0)	225 (4.8)	218 (5.1)	189 (4.4)	0.53
at less than 34 weeks of gestation	153 (0.9)	34 (0.9)	53 (1.1)	36 (0.8)	30 (0.7)	0.2
at less than 28 weeks of gestation	48 (0.3)	9 (0.3)	16 (0.3)	9 (0.2)	14 (0.3)	0.6
Lactation status at 6 months old, *n* (%)						<0.0001
Breast milk only	9,315 (54.9)	1,951 (55.6)	2,540 (56.6)	2,400 (58.8)	2,424 (59.7)	
Breast milk and formula milk	4,121 (24.3)	901 (25.7)	1,154 (25.7)	1,043 (25.5)	1,023 (25.2)	
Formula milk only	2,711 (16.0)	659 (18.8)	797 (17.8)	640 (15.7)	615 (15.1)	
Age in months at the time of the survey at age 3, months	35 (35–36)	35 (35–36)	35 (35–36)	35 (35–36)	35 (35–36)	0.0028
Body weight at age 3, kg	13.7 (12.8–14.6)	13.2 (12.4–14.2)	13.6 (12.7–14.5)	13.8 (12.9–14.7)	14.0 (13.1–15.0)	<0.0001
Height at age 3, cm	92.3 (90.0–94.8)	91.0 (89.0–93.0)	92.0 (90.0–94.2)	92.7 (90.5–95.0)	93.7 (91.5–96.0)	<0.0001
BMI at age 3, kg/m^2	16.0 (15.3–16.8)	16.0 (15.3–16.8)	16.0 (15.3–16.8)	16.0 (15.3–16.8)	16.0 (15.3–16.8)	0.35
Obesity at age 3, *n* (%)	1,763 (10.4)	380 (10.4)	532 (11.3)	435 (10.1)	416 (9.8)	0.1

Data are expressed as median (IQR) or number (percent).
BMI, body mass index; LBW, low birth weight.

Table 4. Female children's characteristics by paternal height

	Total	Paternal height (cm) Quartile 1 (<168.0)	Quartile 2 (168.0–171.9)	Quartile 3 (172.0–175.4)	Quartile 4 (≥175.5)	*p*-value
Number of subjects	16,336	3,584	4,450	4,138	4,164	
Gestational age at birth, weeks	39 (38–40)	39 (38–40)	39 (38–40)	39 (38–40)	39 (38–40)	0.28
Birth weight, g	2,984 (2,750–3,230)	2,940 (2,710–3,178)	2,980 (2,744–3,234)	2,992 (2,764–3,238)	3,018 (2,782–3,260)	<0.0001
LBW, *n* (%)	1,398 (8.6)	371 (10.4)	377 (8.5)	344 (8.3)	306 (7.4)	<0.0001
Macrosomia, *n* (%)	92 (0.6)	14 (0.4)	24 (0.5)	22 (0.5)	32 (0.8)	0.16
Preterm birth at less than 37 weeks of gestation	569 (3.5)	141 (3.9)	131 (2.9)	156 (3.8)	141 (3.4)	0.067
at less than 34 weeks of gestation	121 (0.7)	38 (1.1)	25 (0.6)	30 (0.7)	28 (0.7)	0.066
at less than 28 weeks of gestation	40 (0.2)	10 (0.3)	11 (0.3)	10 (0.2)	9 (0.2)	0.96

(*Continued*)

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Table 4. (*Continued*)

	Total	Paternal height (cm) Quartile 1 (<168.0)	Quartile 2 (168.0–171.9)	Quartile 3 (172.0–175.4)	Quartile 4 (≥175.5)	*p*-value
Lactation status at 6 months old, *n* (%)						0.0001
Breast milk only	9,232 (56.5)	1,924 (56.1)	2,505 (59.1)	2,334 (59.0)	2,469 (62.1)	
Breast milk and formula milk	3,754 (23.0)	872 (25.4)	1,009 (23.8)	959 (24.2)	914 (23.0)	
Formula milk only	2,614 (16.0)	632 (18.4)	726 (17.1)	665 (16.8)	591 (14.9)	
Age in months at the time of the survey at age 3, months	35 (35–36)	35 (35–36)	35 (35–36)	35 (35–36)	35 (35–36)	0.12
Body weight at age 3, kg	13.2 (12.3–14.2)	12.9 (12.0–13.8)	13.0 (12.2–14.0)	13.3 (12.5–14.2)	13.6 (12.7–14.5)	<0.0001
Height at age 3, cm	91.2 (89.0–93.5)	91.0 (89.0–93.0)	92.0 (90.0–94.2)	92.7 (90.5–95.0)	93.7 (91.5–96.0)	<0.0001
BMI at age 3, kg/m^2	15.9 (15.5–16.7)	16.0 (15.3–16.8)	16.0 (15.3–16.8)	16.0 (15.3–16.8)	16.0 (15.3–16.8)	0.06
Obesity at age 3, *n* (%)	1,544 (9.5)	342 (9.5)	439 (9.9)	412 (10.0)	351 (8.4)	0.065

Data are expressed as median (IQR) or number (percent).
BMI, body mass index; LBW, low birth weight.

Table 5. Association between paternal BMI and children's obesity at age 3

	Paternal BMI (kg/m^2) Quartile 1 (<21.2)	Quartile 2 (21.2–22.8)	*p*-value	Quartile 3 (22.9–25.1)	*p*-value	Quartile 4 (≥25.2)	*p*-value	*p* for trend
Male children								
Crude OR (95% CI)	Reference	1.42 (1.21–1.66)	<0.0001	1.71 (1.47–1.99)	<0.0001	2.08 (1.80–2.41)	<0.0001	<0.0001
Adjusted OR (95% CI)	Reference	1.31 (1.10–1.56)	0.0022	1.61 (1.36–1.90)	<0.0001	1.77 (1.50–2.09)	<0.0001	
	Paternal BMI (kg/m^2) Quartile 1 (<21.1)	**Quartile 2 (21.2–22.9)**	***p*-value**	**Quartile 3 (23.0–25.1)**	***p*-value**	**Quartile 4 (≥25.2)**	***p*-value**	***p* for trend**
Female children								
Crude OR (95% CI)	Reference	1.22 (1.04–1.43)	0.017	1.38 (1.18–1.62)	<0.0001	1.87 (1.61–2.19)	<0.0001	<0.0001
Adjusted OR (95% CI)	Reference	1.26 (1.05–1.50)	0.014	1.39 (1.17–1.66)	0.0003	1.66 (1.40–1.97)	<0.0001	

OR, odds ratio; CI, confidence interval; BMI, body mass index; MT1, the first trimester questionnaire for mother; FT1, the first trimester questionnaire for father.
Adjusted for paternal age at FT1 questionnaire, maternal age at MT1 questionnaire, maternal BMI at MT1 questionnaire, gestational age at delivery, paternal and maternal smoking status, paternal and maternal alcohol consumption, paternal and maternal highest level of education, parity, conception method, delivery mode, gestational weight gain, marital status, family income, gestational age at MT1 and FT1 questionnaire, age in months at the time of the survey at age 3, lactation status at 6 months old.

has been reported between paternal BMI and obesity in children than between maternal BMI and children's obesity[20–22]. Contrastingly, some studies have reported that the association between maternal BMI and obesity in children is stronger than that between paternal BMI and obesity in children[23,24]. The ages of children in the aforementioned studies varied from infancy to childhood, which differed from the age taken into consideration in our study. A previous study examining children of the same age as the present study reported the association between paternal BMI and the BMI of children at the age of 3[19]. However, our findings differ from those of this previous study on obesity in children of the same age. Thus, the association between maternal BMI and obesity in children at the age of 3 was stronger than that between paternal BMI and obesity in children at the age of 3 (Table 5 and Supplementary Table S9). In the present study, there were no differences in the effect of paternal and maternal BMI on the proportion of children with obesity at the age of 3 according to sex. Meanwhile, previous reports showed that paternal and maternal obesity were associated with an increased risk of obesity in female and male children, respectively[25].

According to the National Nutrition Survey by the Ministry of Health, Labour and Welfare, the proportion of men and women with BMI ≥ 30 kg/m^2 is less than 5% in Japan, significantly lower than the 35% proportion in the United States[15,26]. On the other hand, the proportion of men in Japan with a BMI ≥ 25 kg/m^2 is approximately 30%, of which 23.1% belong to the age group of

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Table 6. Association between paternal height and children's obesity at age 3

	Paternal height (cm)							
	Quartile 1 (<168.0)	Quartile 2 (168.0–171.9)	*p*-value	Quartile 3 (172.0–175.4)	*p*-value	Quartile 4 (≥175.5)	*p*-value	*p* for trend
Male children								
Crude OR (95% CI)	Reference	1.10 (0.96–1.27)	0.18	0.97 (0.84–1.12)	0.7	0.94 (0.81–1.09)	0.39	0.13
Adjusted OR (95% CI)	Reference	1.09 (0.93–1.27)	0.29	0.96 (0.81–1.12)	0.58	0.94 (0.80–1.11)	0.45	
	Paternal height (cm)							
	Quartile 1 (<168.0)	Quartile 2 (168.0–171.9)	*p*-value	Quartile 3 (172.0–175.4)	*p*-value	Quartile 4 (≥175.5)	*p*-value	*p* for trend
Female children								
Crude OR (95% CI)	Reference	1.04 (0.89–1.20)	0.63	1.05 (0.90–1.22)	0.54	0.87 (0.75–1.02)	0.087	0.1
Adjusted OR (95% CI)	Reference	1.02 (0.86–1.20)	0.83	1.06 (0.9–1.26)	0.47	0.92 (0.77–1.09)	0.31	

OR, odds ratio; CI, confidence interval; MT1, the first trimester questionnaire for mother; FT1, the first trimester questionnaire for father.
Adjusted for paternal age at FT1 questionnaire, maternal age at MT1 questionnaire, maternal BMI at MT1 questionnaire, gestational age at delivery, paternal and maternal smoking status, paternal and maternal alcohol consumption, paternal and maternal highest level of education, parity, conception method, delivery mode, gestational weight gain, marital status, family income, gestational age at MT1 and FT1 questionnaire, age in months at the time of the survey at age 3, and lactation status at 6 months old.

20–29 years and 29.4% belong to the 30–39 age group[26,27]. The distribution of paternal BMI in this study was almost similar to that of the general Japanese men's population.

We previously reported that paternal height had a stronger effect on infant birth weight than paternal BMI in the Japanese population[17]. In contrast, this study found no significant association between paternal height and obesity in children at the age of 3 years. In addition, we also reported an association between paternal BMI and birth weight in male infants. However, there was only a weak association between paternal BMI and birth weight in female infants[17]. However, this study showed a strong association between paternal BMI and obesity in children at the age of 3 years, regardless of the sex of the children. No clear sexual dimorphism was observed in the association between paternal anthropometric factors and obesity at the age of 3.

According to the DOHaD hypothesis, the health and disease predisposition of the next generation is determined by the fertilized egg environment, intrauterine environment, and infant environment. It is thought to be primarily influenced by the mother[1, 12,28]. The present study also analyzed the association between maternal physique (BMI and height) and obesity in children at the age of 3 years using multivariate logistic regression analysis (Supplementary Table S9 and Table S10). As shown in Supplementary Table S9, a strong association was found between maternal BMI and obesity in children at the age of 3 years. Recently, several reports have been published on paternal factors that influence children's health. For instance, paternal exposure to environmental factors such as diet may affect the health of the offspring, and these paternal factors control offspring development through direct genetic, epigenetic, and indirect effects, including maternal prenatal environment[13]. However, few studies have supported this mechanism in the context of epigenetic effects passed from fathers to their children.

Epigenetics are heritable molecular changes that affect gene expression but do not involve changes in the underlying DNA sequence[29]. An animal study showed that a high-fat diet in paternal rats initiates pancreatic β-cell dysfunction in female offspring through epigenetic mechanisms[30]. The most commonly assumed epigenetic mechanism transmits epigenetic factors in sperm such as DNA methylation, chromatin modifications, and non-coding RNAs[31]. For example, a previous study showed that continuous feeding of a high-fat diet to male rats caused gene expression changes via histone modifications in the liver of their offspring[32]. Therefore, aside from DNA methylation, there is growing evidence that sperm RNAs and histone modifications are possible candidates for epigenetic inheritance; however, these mechanisms need to be studied extensively[33].

In addition, obesity in children at the age of 3 corresponds to early AR and may be an indicator of future obesity and NCDs[5]. From the perspective of early AR, the height-weight and BMI curves should be included in the follow-up from the assessment from childhood as part of the ancillary evaluation[34]. Early intervention for obesity in infants, for example, if the BMI of a child at the age of 3 is more than that of a 1.5-year-old child, the child should be considered at risk for future metabolic syndrome, and adequate measures should be taken[8,35]. Furthermore, it has been suggested that the preconception diet and nutritional status of the father and mother may influence the obesity phenotype of their offspring through direct or indirect effects[10,12,13]. This implies that a healthy lifestyle in fathers could positively affect prenatal epigenetic outcomes and postnatal environmental factors, thereby contributing to obesity prevention. However, few reports have addressed the impact of paternal factors on early AR. Future research should investigate the long-term relationship between paternal physique and obesity in children aged 3 and older.

The strengths of this study are that it is a large prospective cohort study of 33,291 pregnant women and that it was adjusted for various confounding factors of parents and their children that may influence obesity in children at the age of 3. Moreover, this is the first study to have examined the association between paternal physique (BMI and height) and obesity in children at the age of 3.

This study had several limitations that need consideration. First, we did not obtain information on lifestyle factors, such as dietary intake and physical activity, which are considered to have a strong influence on the development of childhood adiposity, and thus could not adjust for them. A recent global meta-analysis of 457 studies showed that six food and environmental factors (access to convenience stores, supermarkets, grocery stores, full-service restaurants, fast food restaurants, and fruit and vegetable markets) contribute to childhood obesity[36]. In addition, incorporating

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exercises and activities 3 days a week in preschool may inhibit the increase in the BMI of children[37]. The environmental factors that we were unable to adjust for were likely to be more relevant to children's obesity than the paternal physique, and this suggests that epigenetics alone is not enough to explain it. In contrast, epidemiological studies in four megacities across China showed an independent association between maternal and child obesity, even after adjusting for fast food intake by children[38]. The influence of postnatal environmental factors on childhood obesity is likely significant. However, it is also possible that factors such as the prenatal environment and epigenetics might play a role in its development. A multinational cross-sectional study in 12 countries reported that breastfeeding was a protective factor against obesity and high body fat content in children, independent of maternal BMI[39].

Furthermore, a breastfeeding duration of less than 4 months was associated with the greatest risk of being overweight[40]. In this study, we incorporated breastfeeding status at 6 months of age as one of the adjustment factors to reflect postnatal nutrition status. Second, the physical measurements of parents and children were self-reported. The magnitude to which self-reported data overestimates or underestimates the real values is considered negligible in clinical and research use[41]. Third, 67% of the participants were excluded based on the study criteria, making the finding susceptible to selection bias. The study compared the analyzed and excluded groups and found no significant differences in maternal BMI and obesity in children at the age of 3; however, most of the other variables were significantly different. The exclusion group had missing essential data, such as the father's weight and height, which represents a significant limitation of this study, as comparisons between the analyzed and exclusion groups regarding paternal variables could not be made. It is likely that the analyzed group contains a higher proportion of cooperative participants with health awareness, making it impossible to extrapolate the findings of this study to all fathers in the Japanese population in relation to their 3-year-old children.

In conclusion, the association between paternal BMI and obesity in children at the age of 3 suggests that paternal factors may be as involved as maternal factors in the development of NCDs in future progeny. Continuing this study for a longer period of time is warranted to clarify the relationship between the paternal physique and obesity in children at the age of ≥3 years, which would contribute to the prevention of NCDs.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S2040174424000473

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References

1. Hanson MA, Gluckman PD. Early developmental conditioning of later health and disease: physiology or pathophysiology? *Physiol Rev*. 2014; 94(4), 1027–1076.
2. De Onis M, Blössner M, Borghi E. Global prevalence and trends of overweight and obesity among preschool children. *Am J Clin Nutr*. 2010; 92(5), 1257–1264.
3. Moreno-Mendez E, Quintero-Fabian S, Fernandez-Mejia C, Lazo-de-la-Vega-Monroy ML. Early-life programming of adipose tissue. *Nutr Res Rev*. 2020; 33(2), 244–259.
4. Wang Y, Wang J. Modelling and prediction of global non-communicable diseases. *BMC Public Health*. 2020; 20(1), 822.
5. Rolland-Cachera MF, Deheeger M, Bellisle F, *et al.* Adiposity rebound in children: a simple indicator for predicting obesity. *Am J Clin Nutr*. 1984; 39(1), 129–135.
6. Cissé AH, Lioret S, de Lauzon-Guillain B, *et al.* Association between perinatal factors, genetic susceptibility to obesity and age at adiposity rebound in children of the EDEN mother-child cohort. *Int J Obes (Lond)*. 2021; 45(8), 1802–1810.
7. Lin D, Chen D, Huang J, *et al.* Pre-Birth and early-life factors associated with the timing of adiposity peak and rebound: a large population-based longitudinal study. *Front Pediatr*. 2021; 22(9), 742551.
8. Koyama S, Ichikawa G, Kojima M, *et al.* Adiposity rebound and the development of metabolic syndrome. *Pediatrics*. 2014; 133(1), e114–e119.
9. Aris IM, Rifas-Shiman SL, Li LJ, *et al.* Parental obesity and offspring pubertal development: project viva. *J Pediatr*. 2019; 215, 123–131.
10. Voerman E, Santos S, Patro Golab B, *et al.* Maternal body mass index, gestational weight gain, and the risk of overweight and obesity across childhood: An individual participant data meta-analysis. *PLoS Med*. 2019; 16(2), e1002744.
11. Catalano PM, Ehrenberg HM. The short- and long-term implications of maternal obesity on the mother and her offspring. *BJOG*. 2006; 113(10), 1126–1133.
12. Tarrade A, Panchenko P, Junien C, *et al.* Placental contribution to nutritional programming of health and diseases: epigenetics and sexual dimorphism. *J Exp Biol*. 2015; 218(1), 50–58.
13. Shi Q, Qi K. Developmental origins of health and disease: impact of paternal nutrition and lifestyle. *Pediatr Investig*. 2023; 7(2), 111–131.
14. Finucane MM, Stevens GA, Cowan MJ, *et al.* National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9·1 million participants. *Lancet*. 2011; 377(9765), 557–567.
15. National Health and Nutrition Survey 2019. 2019. Ministry of Health, Labour and Welfare.
16. Tatsumi Y, Ohno Y, Morimoto A, *et al.* BMI percentile curves for Japanese men and women aged 20-79 years who underwent a health check-up in 1980 and 2005. *Obes Res Clin Pract*. 2013; 7(5), e401–e406.
17. Takagi K, Iwama N, Metoki H, *et al.* Paternal height has an impact on birth weight of their offspring in a Japanese population: the Japan environment and children's study. *J Dev Orig Health Dis*. 2019; 10(5), 542–554.
18. Kato N, Takimoto H, Sudo N. The cubic functions for spline smoothed L, S and M values for BMI reference data of Japanese children. *Clin Pediatr Endocr*. 2011; 20(2), 47–49.
19. Fleten C, Nystad W, Stigum H, *et al.* Parent-offspring body mass index associations in the Norwegian mother and child cohort study: a family-

based approach to studying the role of the intrauterine environment in childhood adiposity. *Am J Epidemiol*. 2012; 176(2), 83–92.
20. Öhlund I, Hernell O, Hörnell A, Stenlund H, Lind T. BMI at 4 years of age is associated with previous and current protein intake and with paternal BMI. *Eur J Clin Nutr*. 2010; 64(2), 138–145.
21. Bralić I, Vrdoljak J, Kovacić V. Associations between parental and child overweight and obesity. *Coll Antropol*. 2005; 29(2), 481–486.
22. Banjari I, Martinović M, Belojević G, *et al.* Poverty and other correlates of obesity and underweight among 7-year-olds from Croatia and Montenegro. *Public Health*. 2020; 182, 64–69.
23. Danielzik S, Langnäse K, Mast M, Spethmann C, Müller MJ. Impact of parental BMI on the manifestation of overweight 5-7 years old children. *Eur J Nutr*. 2002; 41(3), 132–138.
24. Sørensen TIA, Ajslev TA, Ängquist L, *et al.* Comparison of associations of maternal peri-pregnancy and paternal anthropometrics with child anthropometrics from birth through age 7 y assessed in the Danish national birth cohort. *Am J Clin Nutr*. 2016; 104(2), 389–396.
25. Jiang MH, Yang Y, Guo XF, Sun YX. Association between child and adolescent obesity and parental weight status: a cross-sectional study from rural North China. *J Intl Med Res*. 2013; 41(4), 1326–1332.
26. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA*. 2014; 311(8), 806–814.
27. Ogawa W, Miyazaki S. Diagnosis criteria for obesity and obesity disease. *Health Evaluation and Promotion*. 2015; 40, 301–306. (In Japanese).
28. Parrettini S, Caroli A, Torlone E. Nutrition and metabolic adaptations in physiological and complicated pregnancy: focus on obesity and gestational diabetes. *Front Endocrinol (Lausanne)*. 2020; 11, 611929.
29. Ptashne M. On the use of the word epigenetic. *Curr Biol*. 2007; 17(7), 233–236.
30. Ng SF, Lin RCY, Laybutt DR, *et al.* Chronic high-fat diet in father's programs β 2-cell dysfunction in female rat offspring. *Nature*. 2010; 467(7318), 963–966.
31. Day J, Savani S, Krempley BD, Nguyen M, Kitlinska JB. Influence of paternal preconception exposures on their offspring: through epigenetics to phenotype *Am J Stem Cells*. 2016; 5, 11–18.
32. Terashima M, Barbour S, Ren J, Yu W, Han Y, Muegge K. Effect of high fat diet on paternal sperm histone distribution and male offspring liver gene expression. *Epigenetics*. 2015; 10(9), 861–871.
33. Rando OJ. Intergenerational transfer of epigenetic information in sperm. *Cold Spring Harb Perspect Med*. 2016; 6(5), a022988.
34. Pomi A, PePe G, Aversa T, *et al.* Early adiposity rebound: predictors and outcomes. *Ital J Pediatr*. 2024; 50(1), 98.
35. Arisaka O, Sairenchi T, Ichikawa G, Koyama S. Increase of body mass index (BMI) from 1.5 to 3 years of age augments the degree of insulin resistance corresponding to BMI at 12 years of age. *J Pediatr Endocrinol Metab*. 2017; 30(4), 455–457.
36. Jia P, Shi Y, Jiang Q, *et al.* Environmental determinants of childhood obesity: a meta-analysis. *Lancet Glob Health*. 2023; 11, S7.
37. Whooten RC, Perkins ME, Gerber MW, Taveras EM. Effects of before-school physical activity on obesity prevention and wellness. *Am J Prev Med*. 2018; 54(4), 510–518.
38. Zhao Y, Wang L, Xue H, Wang H, Wang Y. Fast food consumption and its associations with obesity and hypertension among children: results from the baseline data of the childhood obesity study in China mega-cities. *BMC Public Health*. 2017; 17(1), 933.
39. Ma J, Qiao Y, Zhao P, *et al.* Breastfeeding and childhood obesity: a 12-country study. *Matern Child Nutr*. 2020; 16(3), e12984.
40. Burke V, Beilin LJ, Simmer K, *et al.* Breastfeeding and overweight: longitudinal analysis in an Australian birth cohort. *J Pediatr*. 2005; 147(1), 56–61.
41. Seijo M, Minckas N, Cormick G, *et al.* Comparison of self-reported and directly measured weight and height among women of reproductive age: a systematic review and meta-analysis. *Acta Obstet Gynecol Scand*. 2018; 97(4), 429–439.

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