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ORIGINAL RESEARCH

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The association of changes in body mass index and metabolic parameters between adults with overweight or obesity and their children in a family-based randomized trial (DiOGenes)

Michelle D. Pang[1] | Hülya Yilmaz[2] | Arne Astrup[3] | Ellen E. Blaak[1] | Marleen A. van Baak[1]

[1]Department of Human Biology, School of Nutrition and Translational Research in Metabolism (NUTRIM), Maastricht University, Maastricht, The Netherlands

[2]Hasan Kalyoncu University, Faculty of Health Sciences, Gaziantep, Turkey

[3]Healthy Wright Centre, Novo Nordisk Foundation, Hellerup, Denmark

Correspondence
Marleen A. van Baak, Department of Human Biology, Maastricht University, PO Box 616, 6200 MD Maastricht, The Netherlands.
Email: m.vanbaak@maastrichtuniversity.nl

Summary

Background: Family-based approaches have been reported to be effective in improving overweight or obesity in children.

Objectives: To investigate the relationship of changes in body mass index and metabolic parameters between adults with overweight or obesity and their children during a weight-maintenance family-based dietary intervention.

Methods: In a multicentre randomized controlled trial, families with at least one parent with overweight or obesity and one healthy child aged between 5 and 18 years, of which the parents completed an 8-week weight-loss phase successfully, were randomized into five different dietary intervention groups to achieve weight maintenance for 6 months. Anthropometric parameters and body composition were measured and blood samples were collected before and after the dietary intervention. Data were analysed using Pearson correlation coefficient analyses and multiple linear regression analysis adjusted for diet group, centre, child's sex and age.

Results: A positive association was found between the change in body mass index (BMI) of the mother and change in BMI-for-age Z-score of first and second child (std $\beta = 0.248$, $p = 0.000$; std $\beta = 0.326$, $p = 0.000$, respectively). The change in BMI of the father was only significantly associated with the change in BMI-for-age Z-score of first child (std $\beta = 0.186$, $p = 0.031$). No consistent pattern of associations between parents and children was found for homeostatic model assessment for insulin resistance, fasting glucose and fasting insulin.

Conclusion: This study supports the inclusion of parents into family-based dietary approaches for weight management of their children regardless of the child's weight status in eight different countries throughout Europe.

Abbreviations: BMI, body mass index; DiOGenes, diet, obesity and genes; RCTs, randomized controlled trials; LCD, low calorie diet; GI, glycaemic index; CID, clinical investigation days; BIA, bioelectrical impedance analysis; DXA, dual-energy X-ray absorptiometry; HOMA-IR, homeostatic model assessment for insulin resistance; SD, standard deviation.

Michelle D. Pang and Hülya Yilmaz contributed equally to this work

KEYWORDS
children, family, lifestyle intervention, metabolic health, obesity, weight loss

1 | INTRODUCTION

Childhood obesity is a growing epidemic and has been recognized as one of the most serious public health challenges.[1] In 2016, the prevalence increased by 9 million since 1990 in infants and young children (aged 0–5 years), whereas the prevalence had increased by 14% since 1975 in children and young adolescents (aged 5–19 years).[2] Childhood obesity is often carried through into adolescence[3] and adulthood[4–7] as shown by longitudinal studies. Besides resulting in obesity in adulthood, childhood obesity increases the risk of many health problems, including dyslipidaemia, hypertension, fatty liver disease, type 2 diabetes mellitus, and psychosocial complications.[8,9] Notably, children of parents with overweight were found to be at greater risk of having overweight themselves.[10] Parental obesity more than doubles the risk of obesity during adulthood among children with or without obesity under 10 years of age.[11,12] Especially maternal obesity was found to be a significant driver for childhood obesity, in which epigenetic transmission from mother to offspring may play a causal role in the predisposition of the child to become overweight or have obesity.[13,14] Parental involvement is the key mediator in driving the obesogenic environment at home. Besides parental obesity, the socioeconomic status, education level, smoking, alcohol consumption, dietary consumption, and physical activity behaviours of the parents also contribute to the obesogenic environment.[13,15] Moreover, the attitude, beliefs, and behaviour of the parents play an important role in the eating behaviour of the child and the decision-making regarding the types of food available at home and its preparation.[16,17] Therefore, parents are often involved and targeted in interventions for child weight management. Nevertheless, parental involvement and treatment components vary across childhood obesity interventions. Systematic reviews have shown greater effectiveness of family-based parent–child interventions and parent-only interventions compared to child-only interventions in improving a child's weight, body mass index (BMI), and body composition.[18] This indicates the importance of parental involvement in weight management and lifestyle interventions targeting childhood obesity. Likewise, greater overall effectiveness was found in studies with greater parental involvement and multiple treatment components, including dietary monitoring, physical activity and behavioural modification, compared to single-component interventions.[18] Nevertheless, parental involvement may be less predictive of successful long-term weight management in children and drop-out rates in family-based long-term weight management programs are often high.

In 2006, a multicentre, randomized, controlled dietary intervention study (Diet, Obesity and Genes [DiOGenes]), in families with at least one parent with overweight or obesity and their children, was conducted, focusing on lifestyle changes for weight maintenance of the whole family.[19] Overweight and obesity were targeted from a dietary perspective and families received dietary and behavioural advice. Although evidence, based on a limited number of randomized controlled trials (RCTs), has shown that BMI changes in children with overweight or obesity and their parents with overweight or obesity were correlated during lifestyle-changing interventions in single-centre trials,[20,21] more research is needed to contribute to the existing evidence and gain more insight in managing children's weight by use of family-based approaches. Furthermore, it remains to be determined whether similar associations between changes in body weight of children and their parents can be found, regardless of their initial weight status, in the context of parental weight-maintenance intervention.

The main aim of this study was to investigate the relationship between children's BMI-for-age Z-score change and parental BMI change during the 6 months family-based dietary intervention focusing on weight maintenance for the whole family (DiOGenes), after the parents had lost weight by means of a dietary intervention. Furthermore, as a secondary aim, the relationship of changes in other metabolic parameters between adults and children was determined.

2 | METHODS

2.1 | Study Design

This is a secondary analysis of the DiOGenes study, a multicentre, randomized, controlled, family-based dietary intervention study, executed between November 2005 and April 2007 at eight European centres: Maastricht (The Netherlands), Copenhagen (Denmark), Cambridge (United Kingdom), Heraklion (Greece), Potsdam (Germany), Pamplona (Spain), Sofia (Bulgaria) and Prague (The Czech Republic). Parents with overweight or obesity followed an 8-week low-calorie diet (LCD) after screening and inclusion in the study. During the LCD period, their children received no instructions with respect to their food intake. Families of which (one of) the parent(s) managed to have a > 8% weight loss were randomized to one of the five diet groups for 6 months. The simple block randomization procedure was used with the centre, the number of parents with overweight or obesity in the family and BMI (greater or less than 34 kg/m^2, screening value) as stratification criteria. A third party performed the randomization, without any knowledge about the subjects, which might affect the outcome of the study. The five intervention diet groups were a high protein/high glycemic index (GI) group, a low protein/high GI group, a high protein/low GI group, a low protein/low GI group and a healthy diet control group. The control group followed the national dietary guidelines in each of the countries, with a medium protein content and without specific instructions on GI. During the 6-month dietary intervention, dietary counselling was arranged every 2–4 weeks. The children were encouraged to be involved in the dietary counselling together with their parents, otherwise, parents were instructed to aid their children with

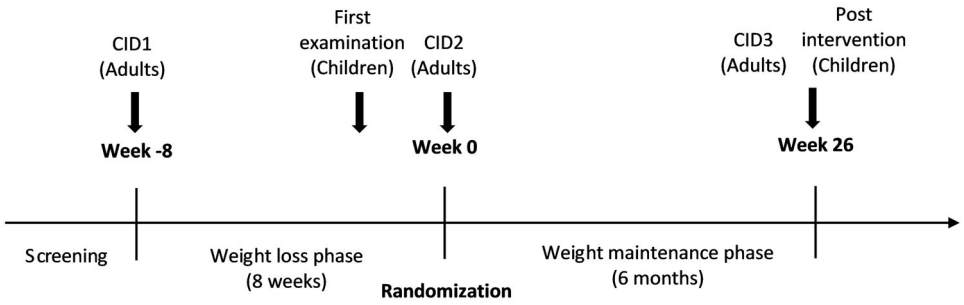

FIGURE 1 The timeline of the study for children and their parents. Weight-loss phase for only parents. Weight-maintenance phase for both parents and children. CID, clinical investigation day

their randomized diets. During the intervention, the families were provided with recipes, together with cooking and behavioural advice. Parents with children below the age of 5 years were advised to feed these children according to local dietary guidelines. The children were requested to attend six counselling sessions, accompanied by their parents, during which intensive guidance was provided. Dietitians advised on weight control and reinforced the diet composition messages through food-choice and behaviour-modification advice. In addition, physical activity and general health issues were also covered by the dieticians. Dietary instructions on the ad libitum diets were provided by trained dietitians, as described previously.[22] Participants were instructed to maintain their habitual physical activity. The adults and children attended clinical examination visits to measure anthropometric parameters and body composition and to collect blood samples at each clinical investigation day (CID). The adults underwent three CIDs, before (CID1) and at the end of the LCD period (CID2), and after the 6-month weight-maintenance phase (CID3). Furthermore, the children attended two CIDs corresponding in time with CIDs 2 and 3 of their parents (Figure 1).

The local ethics committees in the respective countries approved that the study protocol was in accordance with the Declaration of Helsinki and the United Nations Convention on the Rights of the Child. All individuals participating in the study signed an informed consent document according to local legislation after verbal and written instructions. The informed consent document of the children was obtained from their parents or from the child itself from the age of 12 onwards. The study was registered with ClinicalTrials.gov, number NCT00390637. The design of the DiOGenes study has been described extensively elsewhere.[19]

2.2 | Participants

Families of the DiOGenes trial, of which at least one of the parents was overweight or with obesity and completed the 8-week weight-loss phase successfully, were included in the 6-month dietary intervention study. Parents who participated in the study were healthy and below 65 years of age. Furthermore, at least one parent had a BMI between 27 and 45 kg/m^2 at screening and at least one healthy child aged between 5 and 18 years living in their household. The children who were included in this study participated together with their families. Exclusion criteria for parents were BMI >45 kg/m^2, heart or coronary diseases, kidney or liver diseases, psychiatric illnesses, systemic infections or endocrine diseases, weight change >3 kg within 2 months prior to first CID and using prescription medication that might interfere with the outcome of the study. Exclusion criteria for children were systemic infections, chronic disease, or special diets. Further details on recruitment strategies and inclusion and exclusion criteria of the parents and children are provided by Larsen et al..[19]

2.3 | Anthropometric measurements and blood sampling

For all investigations, the same standard operating procedures were used in all centres and all blood samples were analysed in one laboratory to ensure standardization across centres. The children were fasted for 4 h and adults for at least 10 h prior to the investigations. Body weight, height, waist- and hip circumference, and fat percentage (%) were measured and blood samples were collected to determine fasting glucose and insulin as described in a previous paper.[19] Subjects were measured wearing underwear and with an empty bladder. Weight was measured to the nearest 0.1 kg. Height was measured with a wall-mounted stadiometer to the nearest 0.5 cm without shoes. The circumference was measured to the nearest 0.5 cm with a tape, if possible, with the subject standing. Waist circumference was measured between the bottom of the ribs and the top of the hip bone. Hip circumference was measured as the largest circumference in the area around the buttocks. Body composition was assessed by bioelectrical impedance analysis (BIA; Quad-Scan 4000; Bodystat, Douglas, Isle of Man, United Kingdom) at five centres (Maastricht, Pamplona, Heraklion, Sofia and Prague), by dual-energy X-ray absorptiometry (DXA; Lunar Radiation, Madison, WI) at two centres (Copenhagen and Cambridge) and by both DXA and BIA at one centre (Potsdam).

Blood samples were drawn from a Venflon after an overnight fast of at least 10 h and plasma and serum were stored at −80°C until analysis.[19,23] Serum glucose was measured by a colorimetric assay (Ortho-Clinical Diagnostics) for the Vitros 950 analyser and serum insulin was measured by an immunoassay (Siemens Healthcare Diagnostics) for the ADVIA Centaur XP.[23] Homeostatic model assessment (HOMA) was used to estimate insulin resistance (IR) from basal (fasting) glucose and insulin. The homeostatic model assessment for insulin resistance (HOMA-IR) index was calculated as follows: (fasting insulin (mIU/L) × fasting glucose (mmol/L))/22.5.[24] For adults, BMI was calculated as the body weight/height^2 (kg/m^2). For children, gender- and age-specific Z-scores for BMI were calculated using WHO Anthro (0–5 y) and WHO AnthroPlus (5–19 y) software.[25–28]

2.4 | Data analysis

Results are reported as mean ± standard deviation (SD) unless indicated otherwise. The normality of data was assessed with the Shapiro–Wilk test procedure and ln transformation was used if the assumption of normality was not met. Paired *t*-tests were implemented to determine the significance of changes of anthropometric measures, fasting glucose, fasting insulin and HOMA-IR in adults and children over the dietary intervention. Correlations between the changes in BMI-for-age of children and the changes in BMI of father or mother were determined using Pearson correlation coefficient analysis. Linear regression analyses were conducted to examine the relationship between the changes of anthropometric measures, fasting glucose, fasting insulin, and HOMA-IR in mothers, fathers, and children over the diet intervention period. Furthermore, multiple linear regression analyses were conducted with child measures as dependent variables and measures of both parents as independent variables. The analyses were adjusted for diet group, centre, child's sex and age. All data were analysed by using IBM SPSS Statistics 25 and significance was defined as $p < 0.05$.

3 | RESULTS

Only the first and/or second children of families were included in this analysis, resulting in a total of 343 families, comprised of 426 adults and 468 children. Many children had no blood draw or measurement of fat percentage as children did not have to participate in these measurements when they did not want to. For the first children, blood drawing and BIA

TABLE 1 Characteristics of the study population at randomization (at the beginning of the weight-maintenance phase)

		Family (*n* = 343)			
		Adults (*n* = 436)		Children (*n* = 468)	
		Father (*n* = 160)	Mother (*n* = 276)	First child (*n* = 317)	Second child (*n* = 151)
Age (year)		43.4 ± 5.7	41.9 ± 5.8	12.6 ± 3.5	10.7 ± 3.2
Height (cm)		1.78 ± 0.08	1.66 ± 0.07	1.57 ± 0.17	1.48 ± 0.17
Weight (kg)		95.8 ± 15.4	84.6 ± 14.6	58.0 ± 22.2	47.6 ± 18.8
BMI (kg/m^2)		30.0 ± 4.1	30.7 ± 4.6	22.7 ± 5.3	20.7 ± 4.7
BMI (for-age Z-score)				1.2 ± 1.2	1.1 ± 1.3
Waist circumference (cm)		101.9 ± 11.8	94.8 ± 11.7	75.4 ± 13.7	70.0 ± 13.2
Hip circumference (cm)		105.9 ± 9.5	110.9 ± 10.7	88.6 ± 15.8	82.0 ± 15.7
Fat (%)		27.8 ± 6.3	40.2 ± 6.1	28.6 ± 9.0	29.2 ± 9.3
HOMA-IR		2.2 ± 2.9	1.8 ± 1.2	3.3 ± 2.6	2.8 ± 1.9
Fasting glucose (mmol/L)		4.9 ± 0.5	4.7 ± 0.6	4.6 ± 0.5	4.7 ± 0.4
Fasting insulin (mIU/L)		8.6 ± 10.4	7.3 ± 4.5	16.0 ± 13.1	13.2 ± 8.8
Centres (*n* (%))	The Netherlands	39 (24.4)	59 (21.5)	72 (22.7)	41 (27.2)
	Denmark	42 (26.3)	58 (21.2)	60 (18.9)	52 (34.4)
	United Kingdom	12 (7.5)	34 (12.4)	37 (11.7)	19 (12.6)
	Greece	8 (5.0)	17 (6.2)	16 (5.0)	7 (4.6)
	Germany	15 (9.4)	21 (7.7)	28 (8.8)	6 (4.0)
	Spain	20 (12.5)	27 (9.9)	37 (11.7)	23 (15.2)
	Bulgaria	13 (8.1)	34 (12.4)	40 (12.6)	0 (0)
	Czech Republic	11 (6.9)	26 (9.5)	27 (8.5)	3 (2.0)
Diet (*n* (%))	LP/LGI	30 (18.8)	57 (20.8)	65 (20.5)	30 (19.9)
	LP/HGI	29 (18.1)	48 (17.5)	55 (17.4)	27 (17.9)
	HP/LGI	39 (24.4)	59 (21.5)	71 (22.4)	33 (21.9)
	HP/HGI	31 (19.4)	53 (19.3)	59 (18.6)	35 (23.2)
	Healthy diet	31 (19.4)	59 (21.5)	67 (21.1)	26 (17.2)

Note: Values are presented in mean ± SD, unless otherwise specified. Adult's BMI is presented kg/m^2, whereas children's BMI is presented in BMI-for-age Z-score. *Missing values:* Father: waist circumference, hip circumference (*n* = 3); fat percentage (*n* = 22); fasting glucose (*n* = 4); HOMA-IR, fasting insulin (*n* = 15). Mother: waist circumference, hip circumference (*n* = 5); fat percentage (*n* = 44); fasting glucose (*n* = 7); HOMA-IR (*n* = 29); fasting insulin (*n* = 24). First child: waist circumference (*n* = 32); hip circumference (*n* = 33); fat percentage (*n* = 144); fasting glucose (*n* = 111); HOMA-IR, fasting insulin (*n* = 124); Second child: waist circumference (*n* = 2); hip circumference (*n* = 3); fat percentage (*n* = 62); fasting glucose (*n* = 71); HOMA-IR, fasting insulin (*n* = 78).

Abbreviations: BMI, body mass index; HGI, high glycemic index; HOMA-IR, homeostatic model assessment for insulin resistance; HP, high protein; LGI, low glycemic index; LP, low protein.

TABLE 2 Changes in anthropometry and metabolic parameters before and after the weight-maintenance phase in adults and children

	Adults				Children			
	Father		Mother		First child		Second child	
	Change	*p*	Change	*p*	Change	*p*	Change	*p*
BMI (kg/m^2 or Z-score)	0.5 ± 1.6	**0.000**	0.0 ± 2.0	1.000	−0.1 ± 0.4	**0.000**	−0.1 ± 0.6	**0.044**
Waist circumference (cm)	1.2 ± 6.5	**0.032**	0.0 ± 7.0	0.925	−0.5 ± 5.1	0.101	−0.2 ± 4.3	0.568
Hip circumference (cm)	−0.2 ± 4.6	0.656	−0.5 ± 6.7	0.243	0.6 ± 5.3	0.054	0.5 ± 3.9	0.165
Fat (%)	−0.6 ± 3.5	0.069	−1.3 ± 4.6	**0.000**	−1.0 ± 4.9	**0.011**	−1.0 ± 3.6	**0.012**
HOMA-IR	0.5 ± 2.0	**0.008**	0.3 ± 1.1	**0.000**	−0.1 ± 2.0	0.698	0.1 ± 2.2	0.787
Fasting glucose (mmol/L)	0.2 ± 0.5	**0.000**	0.1 ± 0.5	**0.000**	0.0 ± 0.5	0.720	0.0 ± 0.6	0.687
Fasting insulin (mIU/L)	1.5 ± 6.3	**0.007**	0.9 ± 3.7	**0.001**	−0.5 ± 9.5	0.575	0.0 ± 9.0	0.974

Note: Values of change are presented in mean change ± SD. The normality of data was assessed and ln transformation was used for fasting glucose of the second child. Bold values denote significant change $p < 0.05$. Adult BMI is presented in kg/m^2, whereas children's BMI is presented in BMI-for-age Z-score.
Missing values: Father: waist circumference ($n = 4$); hip circumference ($n = 3$); fat percentage ($n = 26$); fasting glucose ($n = 7$); HOMA-IR ($n = 23$); fasting insulin ($n = 22$). Mother: BMI ($n = 2$); waist circumference ($n = 23$); hip circumference ($n = 13$); fat percentage ($n = 60$); fasting glucose ($n = 18$); HOMA-IR ($n = 4$); fasting insulin ($n = 50$). First child: BMI ($n = 1$); waist circumference, hip circumference ($n = 34$); fat percentage ($n = 148$); fasting glucose ($n = 159$); HOMA- IR, fasting insulin ($n = 171$); second child: waist circumference ($n = 2$); hip circumference ($n = 3$); fat percentage ($n = 67$); fasting glucose ($n = 85$); HOMA-IR, fasting insulin ($n = 91$).
Abbreviations: BMI, body mass index; HOMA-IR, homeostatic model assessment for insulin resistance.

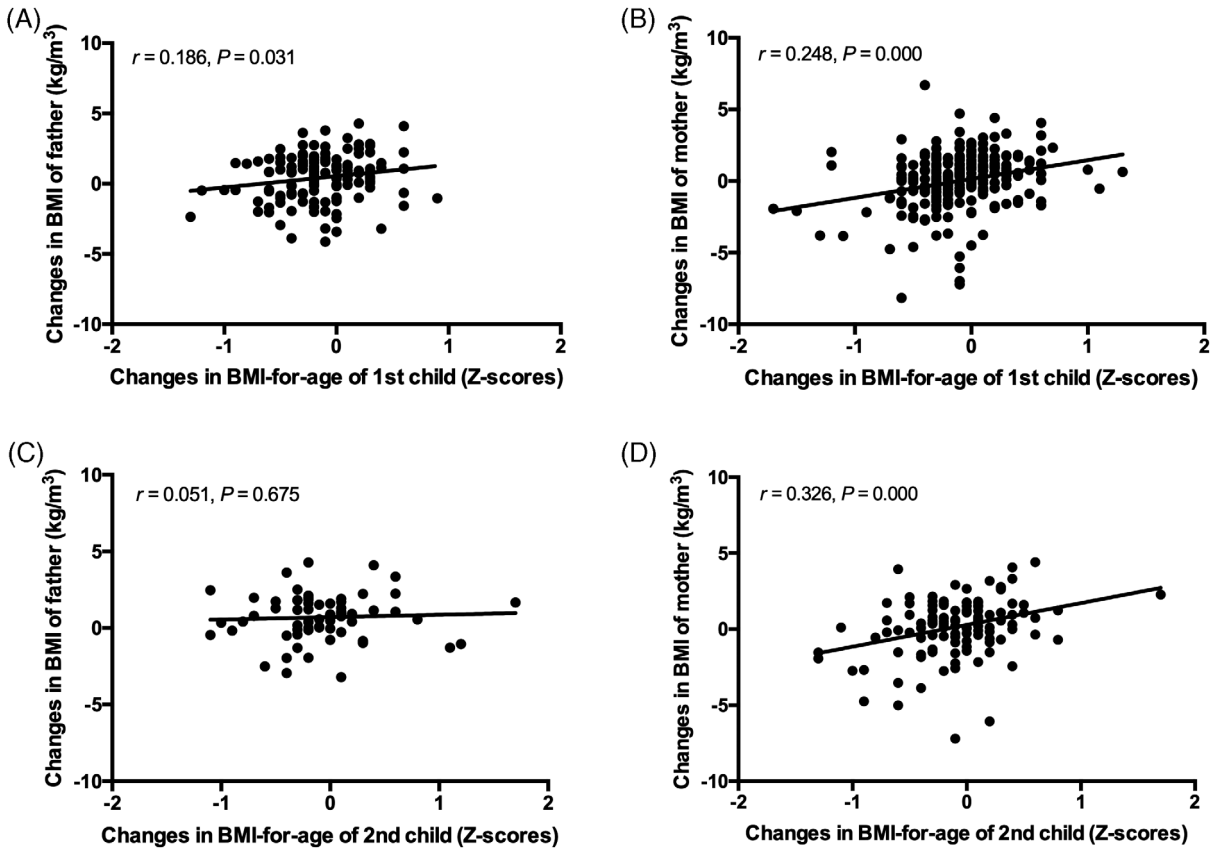

FIGURE 2 Correlation analysis of changes in BMI-for-age of first or second child and changes in BMI of father or mother. (A) Changes in BMI of the father or (B) changes in BMI of the mother were positively associated with the changes in BMI-for-age of the first child. There was no significant association between the (C) changes in BMI of the father or (D) changes in BMI of the mother and changes in BMI-for-age of the second child

TABLE 3 Linear regression analysis of the associations of changes in anthropometry and metabolic parameters over the weight-maintenance phase between father or mother and first or second child

		First child					Second child				
PREDICTORS		***n***	**std β**	***p***	**std β *adj***	***p***	***n***	**std β**	***p***	**std β *adj***	***p***
Father	BMI (kg/m^2 or Z-score)	136	0.186 ± 0.019	**0.031**	0.203 ± 0.021	**0.033**	69	0.051 ± 0.040	0.675	0.134 ± 0.042	0.296
	Waist circumference (cm)	123	−0.008 ± 0.058	0.933	0.019 ± 0.063	0.850	70	0.038 ± 0.060	0.753	0.040 ± 0.065	0.758
	Hip circumference (cm)	124	0.047 ± 0.118	0.606	0.140 ± 0.117	0.121	70	−0.078 ± 0.108	0.522	0.107 ± 0.110	0.385
	Fat (%)	63	0.191 ± 0.163	0.134	0.187 ± 0.171	0.163	33	0.253 ± 0.213	0.163	0.252 ± 0.268	0.271
	HOMA-IR	50	−0.195 ± 0.093	0.174	−0.227 ± 0.112	0.193	31	−0.317 ± 0.160	0.083	−0.527 ± 0.288	**0.020**
	Fasting glucose (mmol/L)	63	−0.012 ± 0.113	0.925	−0.087 ± 0.119	0.523	35	0.204 ± 0.307	0.240	0.076 ± 0.299	0.650
	Fasting insulin (mIU/L)	50	−0.223 ± 0.590	0.120	−0.256 ± 0.684	0.125	33	−0.345 ± 1.855	0.057	−0.528 ± 2.194	**0.020**
Mother	BMI (kg/m^2 or Z-score)	244	0.248 ± 0.012	**0.000**	0.139 ± 0.012	**0.032**	114	0.326 ± 0.020	**0.000**	0.248 ± 0.019	**0.005**
	Waist circumference (cm)	205	0.245 ± 0.055	**0.000**	0.203 ± 0.055	**0.003**	104	0.107 ± 0.063	0.280	0.121 ± 0.056	0.167
	Hip circumference (cm)	212	0.105 ± 0.059	0.128	0.066 ± 0.061	0.350	109	0.038 ± 0.062	0.696	0.063 ± 0.059	0.496
	Fat (%)	104	0.171 ± 0.079	0.083	0.166 ± 0.087	0.126	61	0.188 ± 0.107	0.124	0.201 ± 0.107	0.124
	HOMA-IR	94	0.249 ± 0.176	**0.016**	0.209 ± 0.183	0.050	39	−0.299 ± 0.438	0.065	−0.128 ± 0.521	0.500
	Fasting glucose (mmol/L)	119	−0.098 ± 0.086	0.289	0.006 ± 0.093	0.953	46	−0.148 ± 0.241	0.325	−0.097 ± 0.218	0.478
	Fasting insulin (mIU/L)	95	0.213 ± 1.630	**0.038**	0.122 ± 1.737	0.263	39	−0.228 ± 3.004	0.163	−0.061 ± 3.556	0.750

Note: *n* represents the number of correlations. β coefficients are presented as standardized β coefficient (std β) ± SE; std β adj is adjusted for diet, centre, child's sex and age. Adult BMI is presented in kg/m^2, whereas children's BMI is presented in BMI-for-age Z-score. Bold values denote significant associations ($p < 0.05$).

Abbreviations: BMI, body mass index; HOMA-IR, homeostatic model assessment for insulin resistance.

TABLE 4 Linear regression analysis of the associations of changes in anthropometry and metabolic parameters over the weight-maintenance phase between both parents and first or second child

		First child					Second child				
Predictors		***n***	**std β**	***p***	**std β adj**	***p***	***n***	**std β**	***p***	**std β adj**	***p***
Body mass index (kg/m^2 or Z-score)											
Father	Body mass index	75	0.098 ± 0.027	0.433	0.048 ± 0.028	0.711	41	−0.087 ± 0.061	0.626	−0.086 ± 0.069	0.672
Mother	Body mass index	75	0.322 ± 0.028	**0.011**	0.280 ± 0.028	**0.027**	41	0.480 ± 0.058	**0.010**	0.372 ± 0.063	0.064
Waist circumference (cm)											
Father	Waist circumference	68	0.280 ± 0.061	**0.012**	0.359 ± 0.072	**0.007**	40	0.069 ± 0.078	0.666	−0.009 ± 0.084	0.959
Mother	Waist circumference	68	0.432 ± 0.084	**0.000**	0.452 ± 0.102	**0.001**	40	0.244 ± 0.092	0.134	0.433 ± 0.097	**0.016**
Hip circumference (cm)											
Father	Hip circumference	69	−0.050 ± 0.173	0.701	0.005 ± 0.162	0.970	40	−0.210 ± 0.160	0.215	−0.067 ± 0.176	0.719
Mother	Hip circumference	69	−0.052 ± 0.112	0.693	0.017 ± 0.110	0.894	40	0.074 ± 0.109	0.661	0.171 ± 0.114	0.341
Fat (%)											
Father	Fat percentage	34	0.299 ± 0.169	0.164	0.275 ± 0.219	0.323	24	0.206 ± 0.228	0.405	0.362 ± 0.325	0.244
Mother	Fat percentage	34	0.177 ± 0.221	0.405	0.153 ± 0.289	0.584	24	0.239 ± 0.275	0.334	0.004 ± 0.395	0.992
HOMA-IR											
Father	HOMA-IR	20	−0.393 ± 0.375	0.093	−0.596 ± 0.900	0.304	17	−0.291 ± 0.322	0.261	−0.705 ± 0.434	0.080
Mother	HOMA-IR	20	0.519 ± 0.398	**0.031**	0.823 ± 0.803	0.115	17	−0.341 ± 0.413	0.192	−0.037 ± 0.488	0.904
Glucose (mmol/L)											
Father	Glucose	32	−0.225 ± 0.185	0.318	−0.397 ± 0.232	0.173	21	0.532 ± 0.468	**0.037**	−0.016 ± 1.257	0.981
Mother	Glucose	32	0.196 ± 0.248	0.384	0.261 ± 0.424	0.500	21	−0.504 ± 0.482	**0.047**	−0.284 ± 1.019	0.591
Insulin (mIU/L)											
Father	Insulin	20	−0.435 ± 2.643	0.076	−0.628 ± 5.927	0.271	17	−0.340 ± 2.462	0.186	−0.741 ± 3.111	0.053
Mother	Insulin	20	0.438 ± 3.234	0.075	0.704 ± 6.096	0.157	17	−0.292 ± 3.008	0.253	0.075 ± 3.248	0.786

Note: n represents the number of correlations. β coefficients are presented in standardized β coefficient (std β) ± SE; std β adj is adjusted for diet, centre, child's sex and age. Adult BMI is presented in kg/m^2, whereas children's BMI is presented in BMI-for-age Z-score. Bold values denote significant associations ($p > 0.05$).

Abbreviation: HOMA-IR, homeostatic model assessment for insulin resistance.

or DXA scan were performed in 65% ($n = 206$) and 55% ($n = 174$) of the children, respectively. For the 2nd children, blood drawing and BIA or DXA scan were performed in 53% ($n = 80$) and 59% ($n = 89$) of the children, respectively. Thus, the sample size varies for the different variables. The average age was 12.6 ± 3.5 years for the first (oldest) child and 10.7 ± 3.2 for the second (younger) child. The average age for the parents was 43.4 ± 5.7 for the fathers and 41.9 ± 5.8 for the mothers (Table 1). Before the start of the weight-maintenance phase, the father and/or the mother had successfully lost at least 8% of their initial body weight during the weight-loss phase. At the start of the weight-maintenance phase, the mean BMI-for-age Z-score for the first child was 1.2 ± 1.2 and for the second child, it was 1.1 ± 1.3. Of the first children, overweight or obesity was present in 58.7% of the children, whereas for the second children, this was 53.6%. The mean BMI of the father was 30.4 ± 4.1 kg/m^2, whereas for the mother, the mean BMI was 30.7 ± 4.6 kg/m^2. Furthermore, the study population showed a healthy average fasting glucose value at the start of the weight-maintenance phase in fathers, mothers, first children, and second children (4.9 ± 0.5, 4.7 ± 0.6, 4.6 ± 0.5, and 4.7 ± 0.4 mmol/L, respectively). HOMA-IR for fathers was 2.2 ± 2.9 and for mothers, it was 1.8 ± 1.2. For first children, HOMA-IR was 3.3 ± 2.6 and for second children, it was 2.8 ± 1.9.

3.1 | Changes in anthropometry and metabolic parameters

The BMI and waist circumference of the fathers increased significantly over the 6-month intervention period, whereas no change was found in the mothers (Table 2). The BMI Z-score was decreased significantly in the first child and second child. Moreover, the fat percentage decreased significantly in mothers and first and second children. Furthermore, HOMA-IR, fasting glucose, and fasting insulin were increased significantly in both fathers and mothers, whereas no significant changes were found in children.

3.2 | Association between changes in anthropometry in parents and children

The change in BMI of the mother was found to be a significant predictor for the change in BMI-for-age Z-score of the first and second child (first child unadjusted standardized [std] $\beta = 0.248$, $p = 0.000$, second child unadjusted std $\beta = 0.326$, $p = 0.000$). The change in BMI of the father was only found to be a significant predictor for the change in BMI-for-age Z-score of the first child (unadjusted std $\beta = 0.186$, $p = 0.031$) (Figure 2 and Table 3). Adjustment for the sex and age of the child, diet and centre did not change these results. However, when BMI changes of both parents were included in the model, only the change in BMI of the mother was a significant predictor for the change in BMI-for-age Z-score of the first child and second child, with and without adjustment for child's age and sex, diet and centre (mother unadjusted std $\beta = 0.322$, $p = 0.011$, father unadjusted std $\beta = 0.098$, $p = 0.433$ for first child; mother unadjusted std $\beta = 0.480$, $p = 0.058$, father unadjusted std $\beta = -0.086$, $p = 0.626$ for second child; Table 4). Adjustment for the sex and age of the child, diet and centre did not change these results. Subgroup analyses revealed that 57.1% of the children were overweight or with obesity. Stronger parental associations for changes in BMI-for-age Z-score in first child with overweight or obesity were found compared to lean children. However, in the second child, stronger associations for lean children were found compared to children with overweight or obesity. Partial analysis revealed that the adiposity status, determined using the WHO cut-points for BMI-for-age Z-score, only had a significant impact on the association between the change in BMI Z-score of the first child and the change in BMI of the mother ($r = 0.245$, $p = 0.000$). Furthermore, the change in waist circumference of the mother was found to be a significant predictor for the change in waist circumference of the first child, with or without adjustment for child's age and sex, diet and centre (mother unadjusted std $\beta = 0.245$, $p = 0.000$; father unadjusted std $\beta = -0.008$, $p = 0.933$; Table 3). Associations were not significant for the second child (Table 3). When both parents were included in the regression model, a significant association was found between changes in waist circumference of both father and mother with changes in waist circumference of the first child, with or without adjustment for child's age and sex, diet and centre (father unadjusted std $\beta = 0.280$, $p = 0.012$; mother unadjusted std $\beta = 0.432$, $p = 0.000$; Table 4).

3.3 | Association between changes in metabolic parameters in parents and children

The change in HOMA-IR and fasting insulin of the mother was significantly associated with the change in HOMA-IR and fasting insulin of the first child (HOMA-IR unadjusted std $\beta = 0.249$, $p = 0.016$; fasting insulin unadjusted std $\beta = 0.213$, $p = 0.038$). After adjustment for child's age and sex, diet and centre, the association was not significant anymore (std β adj $= 0.209$, $p = 0.050$; std adj $\beta = 0.122$, $p = 0.263$; (Table 3). Similarly, when both parents were included in the model, only change in HOMA-IR of the mother was found to have a significant association with change in HOMA-IR of first child (std β adj $= 0.519$, $p = 0.031$; Table 4). Again, after adjusting for child's age and sex, diet, and centre, the association was not significant anymore. No significant association in changes of fasting insulin was found when both parents were included in the model. Furthermore, the changes in HOMA-IR and fasting insulin of the father were significantly associated with the changes of the second child after adjustment of child's age and sex, diet and centre (std β adj $= -0.527$, $p = 0.020$; std adj $\beta = -0.528$, $p = 0.020$; Table 3). The significant association was lost when both parents were included in the analysis (Table 4). Although there were no simple correlations between the change in fasting glucose in the second child and father or mother, the associations were significant in the multiple regression analysis (father unadjusted std $\beta = 0.532$, $p = 0.037$; mother unadjusted std $\beta = -0.504$, $p = 0.047$). After adjusting for child's age and sex, diet and centre, the associations were not significant anymore. Further

adjustment of BMI changes did not affect the associations in metabolic parameters.

4 | DISCUSSION

This study investigated the relationship between children's BMI-for-age Z-score change and parental BMI change during a 6 months family-based dietary intervention with the aim to prevent weight (re) gain (DiOGenes). The results showed a positive association between the changes in BMI of the father or mother and the change in BMI-for-age Z-score of the first child, with or without adjustment for child's age and sex, diet and centre. Similar associations were found for the second child. Nevertheless, no significance was reached between the change in BMI of the father and the change in BMI Z-score of the second child, possibly due to the small sample size. Our results are in line with previous RCTs showing similar changes in BMI-for-age and BMI between children with overweight or obesity and their parents with or without overweight or obesity after lifestyle-changing interventions.[20,21,29–31] Although these studies mainly focus on childhood obesity, similar results were expected in the DiOGenes trial, which includes both children with normal weight and children with overweight or obesity, as parents can influence their children's eating behaviour and exposure to factors that foster energy imbalance, regardless of weight status.[32,33] Subgroup analyses revealed stronger parental associations in the first child with overweight or obesity compared to lean. Furthermore, the change in BMI of the mother was found to be the most significant predictor for the changes in BMI-for-age of the first and second child. A longitudinal population-based cohort study also found no association between weight changes of the fathers and the weight changes of their children, whereas an association was found with weight changes of the mothers: weight reduction in the mothers was associated with lower BMI of their offspring.[34] Another population-based cohort study also showed a significant correlation between maternal weight reductions and lower BMI Z-score of their children, regardless of education level, whereas no correlation was found for weight changes of the father.[35] A cluster RCT for child and parent (92% were mothers) weight management study found no significant correlations between changes in BMI of the parent and the BMI percentile of their child, although the changes in adiposity (waist circumference and triceps and subscapular skinfolds) for children and their parent were significantly correlated.[36] These data suggest that mothers, rather than fathers, influence the BMI of their children. This may be due to the fact that in most families, mothers tend to be the primary caregivers and predominantly responsible for managing children's health and food intake of the family.[37] Besides shared environmental factors, common genetic conditions may also be a possible explanation as children can inherit their mother's predisposition to obesity and their ability to control body weight by epigenetic transmission.[38]

No consistent pattern of associations between parents and children was found for the changes in HOMA-IR, fasting glucose and fasting insulin. However, it should be noted that in the parents, these metabolic variables were measured in a hypocaloric state after a significant weight loss before the intervention, which was no longer the case after the weight-maintenance period, whereas children were in an isocaloric state before as well as after the weight-maintenance period. This might have influenced the associations between adults and children with respect to changes in metabolic parameters.

Besides measuring the metabolic variables in a hypocaloric state in parents at the start of the weight-maintenance period, the study had other limitations as well. The number of participating fathers was almost half of that of participating mothers. Additionally, the number of first children enrolled in this study was more than twice as high compared to second children. On top of that, almost half of the participating children had no blood draw or measurement of fat percentage. Thus, the sample size varied for different parameters which may have resulted in a lack of power for some of the outcomes. BMI-for-age Z-score was mainly used as a measure of a child's weight status, whereas fat percentage may have been a better predictor, but this parameter was not available in many children. Although BMI Z-score takes into account the age of the child, the level of body fat may not always be accurately predicted by the BMI Z-score, as it does not take into account a child's secular trend in growth, body build and sexual maturation.[39] Furthermore, other parental influences may have contributed to the child's BMI-for-age Z-score changes that are not included in the current analyses, including physical activity and physiological and socioeconomic factors.

To our knowledge, this study is the first to examine the relationship between the changes in weight and metabolic parameters of first or second child with or without overweight or obesity and the changes in weight and metabolic parameters of the father or mother with overweight or obesity over a 6-month weight-maintenance period after the successful weight loss of the parent(s). Better weight maintenance in parents after a weight-loss intervention is associated with less increase in BMI Z-score in their children, which were included in the dietary weight-maintenance intervention. This study supports the inclusion of parents into family-based dietary approaches for weight management of their children regardless of the child's weight status throughout eight different countries in Europe.

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CONFLICT OF INTEREST

No conflict ofinterest was declared.

ORCID

Michelle D. Pang https://orcid.org/0000-0001-5331-727X
Hülya Yilmaz https://orcid.org/0000-0002-3256-5378
Marleen A. van Baak https://orcid.org/0000-0003-2592-6363

REFERENCES

1. Dehghan M, Akhtar-Danesh N, Merchant A. Childhood obesity, prevalence and prevention. *Nutr J*. 2005;4(24). doi:10.1186/1475-2891-4-24
2. World Health Organisation. *Report of the Commission on Ending Childhood Obesity. Implementation Plan: Executive Summary*. World Health Organization; 2017.
3. Geserick M, Vogel M, Gausche R, et al. Acceleration of BMI in early childhood and risk of sustained obesity. *N Engl J Med*. 2018;379(14):1303-1312. doi:10.1056/NEJMoa1803527
4. Venn A, Thomson R, Schmidt M, et al. Overweight and obesity from childhood to adulthood: a follow-up of participants in the 1985 Australian Schools Health and Fitness Survey. *Med J Aust*. 2007;186(9):458-460. doi:10.5694/j.1326-5377.2007.tb00997.x
5. Stark O, Atkins E, Wolff O, Douglas J. Longitudinal study of obesity in the National Survey of Health and Development. *Br Med J (Clin Res Ed)*. 1981;283(6283):13-17. doi:10.1136/bmj.283.6283.13
6. Clarke W, Lauer R. Does childhood obesity track into adulthood? *Crit Rev Food Sci Nutr*. 1993;33(4–5):423-430. doi:10.1080/10408399309527641
7. Freedman D, Khan L, Serdula M, Dietz W, Srinivasan S, Berenson G. The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. *Pediatrics*. 2005;115(1):22-27. doi:10.1542/peds.2004-0220
8. Biro F, Wien M. Childhood obesity and adult morbidities. *Am J Clin Nutr*. 2019;91(5):1499S-1505S. doi:10.3945/ajcn.2010.28701B
9. Güngör N. Overweight and obesity in children and adolescents. *J Clin Res Pediatr Endocrinol*. 2014;6(3):129-143. doi:10.4274/Jcrpe.1471
10. Bahreynian M, Qorbani M, Khaniabadi B, et al. Association between obesity and parental weight status in children and adolescents. *J Clin Res Pediatr Endocrinol*. 2017;9(2):111-117. doi:10.4274/jcrpe.3790
11. Whitaker R, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med*. 1997;337(13):869-873. doi:10.1056/NEJM199709253371301
12. Santangeli L, Sattar N, Huda S. Impact of maternal obesity on perinatal and childhood outcomes. *Best Pract Res Clin Obstet Gynaecol*. 2015;29(3):438-448.
13. Walsh B, Cullinan J. Decomposing socioeconomic inequalities in childhood obesity: evidence from Ireland. *Econ Hum Biol*. 2015;16:60-72. doi:10.1016/j.ehb.2014.01.003
14. Ling C, Rönn T. Epigenetics in human obesity and type 2 diabetes. *Cell Metab*. 2019;29(5):1028-1044. doi:10.1016/j.cmet.2019.03.009
15. Nobles J, Summerbell C, Brown T, Jago R, Moore T. A secondary analysis of the childhood obesity prevention Cochrane review through a wider determinants of health lens: implications for research funders, researchers, policymakers and practitioners. *Int J Behav Nutr Phys Act*. 2021;18(1):22. doi:10.1186/s12966-021-01082-2
16. Scaglioni S, Salvioni M, Galimberti C. Influence of parental attitudes in the development of children eating behaviour. *Br J Nutr*. 2008;99:S22-S25. doi:10.1017/S0007114508892471
17. Scaglioni S, De Cosmi V, Ciappolino V, Parazzini F, Brambilla P, Agostoni C. Factors influencing children's eating behaviours. *Nutrients*. 2018;10(6):706. doi:10.3390/nu10060706
18. Chai L, Collins C, May C, Brain K, Wong See D, Burrows T. Effectiveness of family-based weight management interventions for children with overweight and obesity: an umbrella review. *JBI Database System Rev Implement Rep*. 2019;17(7):1341-1427. doi:10.11124/JBISRIR-2017-003695
19. Larsen T, Dalskov S, van Baak M, et al. The diet, obesity and genes (Diogenes) dietary study in eight European countries—a Comprehensive design for long-term intervention. *Obes Rev*. 2010;11(1):76-91. doi:10.1111/j.1467-789X.2009.00603.x
20. Watson P, Dugdill L, Pickering K, et al. A whole family approach to childhood obesity management (GOALS): relationship between adult and child BMI change. *Ann Hum Biol*. 2011;38(4):445-452. doi:10.3109/03014460.2011.590531
21. Boutelle K, Cafri G, Crow S. Parent-only treatment for childhood obesity: a randomized controlled trial. *Obesity (Silver Spring)*. 2011;19(3):574-580. doi:10.1038/oby.2010.238
22. Moore CS, Lindroos AK, Kreutzer M, et al. Dietary strategy to manipulate ad libitum macronutrient intake, and glycaemic index, across eight European countries in the Diogenes Study. *Obes Rev*. 2010;11(1):67-75. doi:10.1111/j.1467-789X.2009.00602.x
23. Damsgaard CT, Papadaki A, Jensen SM, et al. Higher protein diets consumed ad libitum improve cardiovascular risk markers in children of overweight parents from eight European countries. *J Nutr*. 2013;143(6):810-817. doi:10.3945/jn.112.173427
24. Wallace TM, Levy JC, Matthews DR. Use and abuse of HOMA modeling. *Diabetes Care*. 2004;27(6):1487-1495.
25. de Onis M, Onyango A, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. *Bull World Health Org*. 2007;85(9):660-667. doi:10.2471/blt.07.043497
26. WHO. The WHO Anthro Survey Analyser.
27. WHO. WHO Anthroplus software.
28. World Health Organization. *WHO Child Growth Standards: Length/Height-for-Age, Weight-for-Age, Weight-for-Length, Weight-for-Height and Body Mass Index-for-Age: Methods and Development*. World Health Organization; 2006.
29. Quattrin T, Roemmich J, Paluch R, Yu J, Epstein L, Ecker M. Efficacy of family-based weight control program for preschool children in primary care. *Pediatrics*. 2012;130(4):660-666. doi:10.1542/peds.2012-0701
30. Wrotniak B, Epstein L, Paluch R, Roemmich J. Parent weight change as a predictor of child weight change in family-based behavioral obesity treatment. *Arch Pediatr Adolesc Med*. 2004;158(4):342-347. doi:10.1001/archpedi.158.4.342
31. Boutelle K, Kang Sim D, Rhee K, Manzano M, Strong D. Family-based treatment program contributors to child weight loss. *Int J Obes (Lond)*. 2020:45(1):77–83. doi:10.1038/s41366-020-0604-9
32. Golan M, Crow S. Parents are key players in the prevention and treatment of weight-related problems. *Nutr Rev*. 2004;62(1):39-50. doi:10.1111/j.1753-4887.2004.tb00005.x
33. Harper L, Sanders K. The effect of adults' eating on young children's acceptance of unfamiliar foods. *Behav Ther*. 1975;15(1):101-108. doi:10.1016/0022-0965(75)90098-3
34. Andriani H, Liao C, Kuo H. Parental weight changes as key predictors of child weight changes. *BMC Public Health*. 2015;15:645. doi:10.1186/s12889-015-2005-x
35. Naess M, Sund E, Lingaas Holmen T, Kvaløy K. Implications of parental lifestyle changes and education level on adolescent offspring weight: a population based cohort study—the HUNT Study, Norway. *BMJ Open*. 2018;8(8):e023406. doi:10.1136/bmjopen-2018-023406
36. Berry D, McMurray R, Schwartz T, Hall E, Neal M. A cluster randomized controlled trial for child and parent weight management: children and parents randomized to the intervention group have correlated changes in adiposity. *BMC Obes*. 2017;4:39. doi:10.1186/s40608-017-0175-z
37. Salganicoff A, Ranji U, Wyn R. Mothers' family health care roles, women ages 18 and older. In: Foundation KF, ed. *Women and Health*

Care: A National Profile Key Findings From the Kaiser Women's Health Survey. CA: Kaiser Family Foundation; 2004.

38. Agarwal P, Morriseau T, Kereliuk S, Doucette C, Wicklow B, Dolinsky V. Maternal obesity, diabetes during pregnancy and epigenetic mechanisms that influence the developmental origins of cardiometabolic disease in the offspring. *Crit Rev Clin Lab Sci*. 2018; 55(2):71-101. doi:10.1080/10408363.2017.1422109
39. Wang Y, Moreno L, Caballero B, Cole T. Limitations of the current World Health Organization growth references for children and adolescents. *Food Nutr Bull*. 2006;27(4 Suppl Growth Standard):S175-S188. doi:10.1177/15648265060274S502

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