Neonatal Anthropometrics and Obesity Treatment Response in Children and Adolescents

Ulrik Lausten-Thomsen, PhD[1], Morten Asp Vonsild Lund, MD[2,3], Sara Elizabeth Stinson, MSc[4], Christine Frithioff-Bøjsøe, MD[2,4], Louise Aas Holm, MD[2,4], Jennifer Lyn Baker, PhD[4,5], Cilius Esmann Fonvig, PhD[2,4,6], Michael Christiansen, MD[3,7,8], Lars Ängquist, PhD[4], Torben Hansen, PhD[4], and Jens-Christian Holm, PhD[2,4,8]

Objective To investigate the relationship between in utero growth conditions, as indicated by neonatal anthropometric measures, and childhood obesity treatment response, to examine the potential usefulness of neonatal anthropometrics as a potential childhood obesity treatment stratification tool.

Study design The study included 2474 children and adolescents with obesity (mean age, 11.2 years; range, 5.0-18.9 years) treated at the Children's Obesity Clinic in Holbæk, Denmark. Treatment response was registered prospectively, and neonatal data were collected from national electronic registers.

Results Birth weight, birth length, birth weight for gestational age, and large for gestational age status were positively associated with the degree of obesity at treatment initiation. After a mean (SD) of 1.27 (0.69) years of enrollment in obesity treatment, the children exhibited a mean reduction of −0.32 (0.50) in body mass index SD score. No significant associations between neonatal anthropometric measures and childhood obesity treatment response were detected.

Conclusions Neonatal anthropometric measures were positively associated with the degree of obesity at treatment initiation but not with response to multidisciplinary treatment of childhood obesity. Individualization of obesity treatment based on neonatal anthropometry does not seem warranted. *(J Pediatr 2021; ■:1-5)*.

The etiology of childhood obesity is both complex and multifactorial and has been linked to prenatal factors[1] along with genetic,[2] metabolic,[3] nutritional,[3] physical activity,[4] socioeconomic,[5] and psychological[6] factors. During prenatal organogenesis, rapid growth and functional maturation of organ systems occurs, with deposition of fat mass and establishment of homeostatic mechanisms. These processes are believed to be sensitive to disturbances of the intrauterine milieu, especially stress and the availability of nutrients.[7] Birth weight has been suggested to be an indicator of the quality of the intrauterine environment.[8] Studies have demonstrated an independent relationship between birth weight and body mass index (BMI) in both childhood and adulthood,[9-11] and, accordingly, more evidence of the prenatal origins of childhood obesity has emerged.[12]

It is possible that the association between an increased risk of childhood obesity and preceding neonatal anthropometric measures reflects prenatally occurring metabolic derangement or perturbed homeostatic mechanisms, through some as-yet to be fully determined mechanisms. If so, it follows logically that the intrauterine milieu also might influence subsequent treatment response in children and adolescents with obesity.

The present study aimed to explore the relationship between in utero growth conditions, as indicated by neonatal anthropometric measures and subsequent childhood obesity treatment response, to investigate whether neonatal anthropometrics could be used as an independent prognostic marker for treatment response.

Methods

The study population consisted of children and adolescents treated according to the Holbæk Obesity Treatment Protocol.[13] The inclusion criteria for this study

BMI	Body mass index
BW-GA	Birth weight for gestational age
LGA	Large for gestational age
PI	Ponderal index
SGA	Small for gestational age

From the [1]Department of Neonatology, Copenhagen University Hospital Rigshospitalet, Copenhagen, Denmark; [2]The Children's Obesity Clinic, Department of Pediatrics, Copenhagen University Hospital Holbæk, Holbæk, Denmark; [3]Department of Biomedical Sciences, [4]Novo Nordisk Foundation Center for Basic Metabolic Research, Faculty of Health and Medical Sciences, and [8]Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark; [5]Center for Clinical Research and Prevention, Bispebjerg and Frederiksberg Hospital, Copenhagen, Denmark; [6]Department of Pediatrics, Kolding Hospital, Kolding, Denmark; and [7]Department for Congenital Disorders, Danish National Biobank and Biomarkers, Statens Serum Institut, Copenhagen, Denmark

U.L.-T. and J.-C.H. contributed equally to this work.

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were childhood obesity, defined as a BMI >95th percentile for age and sex[14]; age at treatment initiation between 5 and 19 years; availability of follow-up information within 6-48 months from later visits at the clinic; and available birth weight. We excluded very preterm children (ie, those born before completed 32nd week of gestation; n = 14) and very postterm children (ie, those born after the 43rd week of gestation; n = 3), twins (n = 7), and children with extreme (potentially unrealistic) ponderal index (PI) values (ie, <17 [n = 11] or >35 [n = 13]). No other exclusion criteria were applied.

All participants and/or their parents provided signed informed consent. The study was approved by the Danish Data Protection Agency and the Regional Scientific Ethics Committee (SJ-104) and is registered at ClinicalTrials.gov (identifier NCT00928473).

The children and adolescents were treated using the Holbæk Obesity Treatment Protocol, a family-based individualized treatment regimen described in detail elsewhere.[13,15] Each patient was seen by a nurse and a pediatrician who performed a general investigation and evaluated the patient to identify obesity and its complications, as well as differential diagnoses. An individual treatment plan comprising 10-20 items aimed at controlling the environment around the patient with obesity was then prescribed. The patient was subsequently seen by a pediatrician, a dietician, and/or a nurse at individual intervals depending on the condition, treatment response, and practical requirements.[13]

Doctors or nurses examined all patients at entry in the program and at follow-up visits. Height and weight, while wearing light indoor clothing and without shoes, were measured using a stadiometer to the nearest millimeter and a digital weight scale (model BC418; Tanita) to the nearest 100 g, respectively. In addition, at baseline and follow-up, BMI SDSs were calculated according to Danish BMI charts[14] to account for age- and sex-related variation in reference intervals. Notably, the 95th percentile (obesity) used for inclusion at baseline is equivalent to BMI SDS >1.64, and the 99th percentile (severe obesity) is equal to BMI SDS >2.33.

Additional information on age, sex, socioeconomic status, maternal smoking during pregnancy (yes/no), and duration of breastfeeding (reported in whole months) was obtained through a structured family interview. Socioeconomic status was categorized into 5 groups (with grade 1 representing the highest incomes) depending on parental occupation, based on a national classification system.[16] Treatment response was measured as the change in BMI SDS during the follow-up period (follow-up measure subtracted by the baseline measure). The follow-up information when multiple eligible observations were available was selected using an algorithm, with the aim of making as much information as possible available while being as close as possible to 1 year after baseline. Here the information (data availability) relates to various types of variables, such as dual-energy x-ray absorptiometry and magnetic resonance measures, as well as blood biochemistry variables. The actual selection was made prior to this project for a general setting (where possibly all types of variables would be used), explaining the slight complexity of the algorithm even though not all variable types are used for this specific project.

Information about birth weight, birth length, and duration of pregnancy was obtained by linking government-issued personal identification numbers for each child to data from the nationwide Danish Neonatal Screening Biobank.[17] Birth weight relative to gestational age (BW-GA) in SDs was calculated as the standardized difference between the observed and expected birth weight for a given gestational age using a Scandinavian reference.[18] Small for gestational age (SGA) was defined as BW-GA <10th standard normal percentile; large for gestational age (LGA), as BW-GA >90th standard normal percentile. The neonatal PI was calculated as PI = birth weight/birth length^3 (kg/m^3).

The association between neonatal anthropometrics and degree of obesity at treatment initiation, expressed as BMI SDS (ie, baseline BMI SDS), as well as the association between neonatal anthropometrics and subsequent treatment response at follow-up (ΔBMI SDS), were estimated using multiple linear regression models. The analyses were run based on all eligible data (the full sample) and also separately for boys and girls, for which these basic models were adjusted for age and sex (when applicable). Moreover, interaction analyses were conducted with respect to baseline age (<11 years at entry), using a binary indicator variable to underlie respective interaction variables for our set of neonatal anthropometric variables considered. The cutoff of 11 years allowed for analysis of 2 groups of approximately equal size. Further interaction analyses with respect to baseline age were conducted using limits of 9 years for girls and 10 years for boys, age intervals that allow for approximation of prepubertal cohorts.[19] Additionally, in a subgroup of patients with available data (ie, the restricted sample), all the models were further adjusted for socioeconomic status, duration of breastfeeding, and maternal pregnancy smoking. An additional subgroup analysis was further adjusted for the duration of posttreatment follow-up. All neonatal anthropometric variables (exposures) were used separately and individually (ie, one by one) for the fitted models. Statistical analyses were performed using Stata 15.1 (StataCorp). Statistical significance was set at $P < .05$.

Results

A total of 2474 eligible children with obesity (1321 girls) were included in the study between June 2008 and February 2020. At baseline, the participants had a mean age of 11.2 years (range, 5.0-18.9 years) and a mean BMI SDS of 2.93 (range, 1.64-7.66) corresponding to a mean BMI >99th percentile for age and sex. Neonatal anthropometric measures and clinical obesity measures at treatment initiation are detailed in **Table I**. For the restricted sample (n = 1401) underlying the more heavily adjusted analyses, similar descriptive information is provided in **Table II** (available at www.jpeds.com).

In multiple regression models adjusted for age and sex (or only age for the sex-specific analyses), a positive association

Table I. Baseline characteristics and treatment response for all included patients

Characteristics/treatment responses	All patients (n = 2474)	Girls (n = 1321)	Boys (n = 1153)
At birth			
Gestational age, wk, mean ± SD	39.6 ± 1.8 [32-43]	39.6 ± 1.8 [32-43]	39.6 ± 1.9 [32-43]
Birth weight, kg, mean ± SD	3.57 ± 0.59 [1.18-5.34]	3.55 ± 0.58 [1.18-5.20]	3.60 ± 0.61 [1.40-5.34]
Birth length, cm, mean ± SD	52.1 ± 2.6 [38-60]	51.8 ± 2.5 [38-59]	52.4 ± 2.7 [40-60]
PI, kg/m^3, mean ± SD	25.2 ± 2.7 [17.1-34.6]	25.4 ± 2.7 [17.1-34.6]	25.0 ± 2.6 [17.3-34.4]
BW-GA (SDS), mean ± SD	0.03 ± 1.24 [−4.22 to 6.16]	0.11 ± 1.27 [−4.22 to 6.16]	−0.05 ± 1.21 [−3.85 to 5.61]
SGA, %	12.3	12.2	13.0
LGA, %	15.9	16.6	13.6
At treatment start			
Age, y, mean ± SD	11.2 ± 3.0 [5.0-18.9]	11.1 ± 3.2 [5.0-18.9]	11.3 ± 2.9 [5.0-18.9]
BMI SDS, mean ± SD	2.93 ± 0.69 [1.64-7.66]	2.74 ± 0.56 [1.64-5.46]	3.15 ± 0.76 [1.65-7.66]
Height, cm, mean ± SD	150.9 ± 16.2 [106.5-199.5]	149.5 ± 15.8 [106.5-186.8]	152.5 ± 16.5 [110.7-199.5]
Weight, kg, mean ± SD	63.0 ± 23.4 [22.1-185.0]	61.8 ± 23.1 [22.1-185.0]	64.3 ± 23.8 [22.8-158.7]
Treatment response			
Treatment duration, y, mean ± SD	1.27 ± 0.69 [0.50-3.98]	1.31 ± 0.73 [0.50-3.98]	1.22 ± 0.64 [0.50-3.96]
ΔBMI SDS, mean ± SD	−0.32 ± 0.50 [−2.96 to 1.44]	−0.24 ± 0.47 [−2.96 to 1.44]	−0.40 ± 0.52 [−2.76 to 1.44]

between several neonatal anthropometric measurement and BMI SDS at treatment initiation was demonstrated. A birth weight increase of 1 kg was associated with a 0.049-unit higher BMI SDS at treatment initiation (95% CI, 0.005- 0.092; $P = .028$), and a birth length increase of 1 cm was associated with a 0.014-unit higher BMI SDS (95% CI, 0.004-0.024; $P = .008$). A 1-unit increase in BW-GA was associated with a 0.036-unit higher BMI SDS (95% CI, 0.015-0.056; $P = .001$) and being born LGA was associated with a 0.111-unit higher BMI SDS (95% CI, 0.039-0.182; $P = .002$) at treatment initiation (Table III). Associations between BMI SDS at treatment initiation and gestational age, PI, and SGA status were not statistically significant (Table III).

Similar patterns and tendencies for these associations were found when the sexes were examined individually. However, only LGA remained statistically significant for girls, whereas birth length, BW-GA (SDS), and LGA remained statistically significant for boys (Table III). Generally, the interaction analyses with respect to age at treatment initiation—both overall and sex-specific—indicated that the associations tended to be slightly attenuated for older age (although the estimated interactions were not significant; results not shown).

Overall, the more heavily adjusted analyses based on the restricted sample size showed some indication for stronger effects on the basic and sex-specific associations, and the general pattern remained similar. Only birth weight for boys changed significance status (from nonsignificance though borderline to significance), and the P value itself changed only modestly (Table IV; available at www.jpeds.com).

The participants exhibited a mean (SD) obesity reduction through ΔBMI SDS of −0.32 (0.50) unit (range, −2.96 to 1.44), with the majority reducing their degree of overweight (median, −0.23; IQR, −0.56 to −0.00). For girls and boys, the mean reductions were −0.24 (0.47) and −0.40 (0.52), respectively (Table I). The mean (SD) treatment time was 1.27 (0.69) years (range, 0.50-3.98 years), although the majority of follow-ups were close to 1 year (median, 1.07 years; IQR, 0.89-1.38 years).

Association analyses between neonatal anthropometric measures and the subsequent treatment response were performed in the same way as for the baseline BMI SDS outcome. Although no statistically significant associations were demonstrated, the estimated associations generally indicated smaller ΔBMI SDS for higher levels of the neonatal measures (Table V).

The same overall observations were made when examining the sexes individually, with no statistically significant findings and the same general pattern as for the overall associations (Table V). Overall interaction analyses with respect to

Table III. Association between neonatal anthropometric measures and degree of obesity at the start of treatment for all included patients

	All patients				Girls				Boys			
Anthropometric measures	n	β	95% CI	P	n	β	95% CI	P	n	β	95% CI	P
Gestational age, wk	2417	−0.005	−0.019 to 0.009	.49	1284	−0.001	−0.018 to 0.017	.95	1133	−0.006	−0.029 to 0.017	.60
Birth weight, kg	2474	0.049	0.005 to 0.092	**.028**	1321	0.040	−0.012 to 0.092	.13	1153	0.067	0.004 to 0.138	.066
Birth length, cm	2400	0.014	0.004 to 0.024	**.008**	1283	0.012	−0.000 to 0.024	.053	1117	0.017	0.001 to 0.033	**.039**
PI, kg/m^3	2400	0.002	−0.008 to 0.012	.66	1283	−0.001	−0.013 to 0.010	.82	1117	0.007	−0.009 to 0.024	.40
BW-GA (SDS)	2417	0.036	0.015 to 0.056	**.001**	1284	0.023	−0.001 to 0.047	.064	1133	0.052	0.017 to 0.087	**.004**
SGA	2417	−0.051	−0.131 to 0.028	.20	1284	0.008	−0.089 to 0.104	.88	1133	−0.118	−0.246 to 0.009	.069
LGA	2417	0.111	0.039 to 0.182	**.002**	1284	0.093	0.013 to 0.172	**.023**	1133	0.146	0.021 to 0.271	**.022**

β indicates estimated association effects. Analyses were adjusted for age and sex (when applicable; see text for details). Significant values are in bold type.

Table V. Association between neonatal anthropometric measures and change in degree of obesity during treatment for all included patients

	All patients				Girls				Boys			
Anthropometric measures	n	β	95% CI	P	n	β	95% CI	P	n	β	95% CI	P
Gestational age, wk	2417	−0.003	−0.014 to 0.008	.57	1284	−0.010	−0.024 to 0.004	.16	1133	0.004	−0.012 to 0.021	.59
Birth weight, kg	2474	−0.030	−0.062 to 0.003	.074	1321	−0.039	−0.082 to 0.004	.078	1153	−0.020	−0.070 to 0.030	.43
Birth length, cm	2400	−0.006	−0.014 to 0.002	.12	1283	−0.007	−0.017 to 0.003	.17	1117	−0.005	−0.016 to 0.007	.41
PI, kg/m^3	2400	−0.003	−0.010 to 0.004	.41	1283	−0.005	−0.015 to 0.004	.26	1117	−0.000	−0.012 to 0.011	.95
BW-GA (SDS)	2417	−0.014	−0.030 to 0.002	.088	1284	−0.010	−0.030 to 0.010	.32	1133	−0.018	−0.043 to 0.007	.16
SGA	2417	0.004	−0.056 to 0.063	.90	1284	0.015	−0.065 to 0.094	.72	1133	−0.008	−0.098 to 0.083	.87
LGA	2417	−0.030	−0.083 to 0.024	.28	1284	−0.011	−0.077 to 0.054	.73	1133	−0.055	−0.143 to 0.034	.23

Analyses were adjusted for age and sex (when applicable; see text for details).

age at treatment initiation (<11 or ≥11 years) revealed no significant findings ($P > .10$ for all), with no clear apparent pattern among the estimated effect directions (results not shown). No significant findings were found when using age <9 or ≥9 years for girls and <10 or ≥10 years for boys ($P > .10$ for all; results not shown). In the additionally adjusted analyses, there remained no significant associations with treatment response (ΔBMI SDS), and the analyses did not show any obvious regular pattern of change (**Table VI**; available at www.jpeds.com). No significant changes in the results were observed when further adjusting for treatment duration (data not shown).

Discussion

In this study of Danish children and adolescents with obesity, we demonstrated some positive associations between the overall effect of prenatal conditions, as assessed by neonatal anthropometric measures, and the degree of obesity at initiation of a multidisciplinary childhood obesity treatment program. However, after a mean of 1.27 years of treatment, no neonatal anthropometric measure was found to be associated with the obesity management response. Our findings add to the increasing evidence for in utero programming of childhood obesity and are in concordance with the literature[10,20,21] as well as with previous studies from our research group.[22,23] Based on the findings linking neonatal anthropometric measures to subsequent childhood obesity, it seems possible that neonatal anthropometric measures similarly could predict the response to obesity management. This idea is not supported by the present study, however, as we detected no effects of neonatal anthropometric measures as indicators of prenatal growth conditions on the outcome of obesity treatment.

In this study, we did not observe consistent differences by sex between neonatal anthropometry and BMI at treatment initiation or with treatment response. Even though there were statistically significant associations between birth length and BW-GA with BMI SDS at treatment initiation in the boys, the effects were minimally different from those in the girls. Our analyses did not identify any associations between neonatal anthropometry and response to treatment as assessed by a change in BMI SDS. Although alternate approaches to examining BMI changes, such as using the percent of the 95th percentile, have been recommended when using the US Centers for Disease Control BMI reference,[24,25] such recommendations do not exist for the Danish BMI reference.

There are several possible explanations for our results in this study. Overall, the lack of association between neonatal anthropometric measures and treatment response is unlikely to be related to a lack of prenatal exposure, as the data showed an association between neonatal anthropometric measures and childhood obesity, in concordance with the literature. Moreover, given that >75% of the children reduced their degree of obesity during treatment, it is equally unlikely that a clinically relevant prenatal influence on treatment response has eluded detection owing to a lack of treatment effect. It is possible that an association between neonatal anthropometric measures and obesity management results manifest only after several years of treatment, something this study was not designed to test.

Our data do not refute the existence of an association between neonatal anthropometric measures and response to obesity treatment. It is also possible that this association is more pronounced in a subset of pregnancies, such as those affected by maternal diabetes. However, because the effect of in utero conditions (represented here by, eg, birth weight) on the degree of obesity at treatment initiation was measurable, its effect on treatment response must be of a much smaller—possibly clinically insignificant—magnitude, because it eluded detection. If there is an influence of birth weight on the obesity treatment response, then our data from this large cohort of children and adolescents with obesity suggest that the effect is likely to be of modest relevance in a clinical treatment setting.

Adjusting for duration of posttreatment follow-up did not alter the association, and using age defined as prepubertal age also did not seemingly interact with the association. However, the lack of available detailed information on pubertal development is a limitation of this study, given that puberty-associated hormonal changes potentially influence the response to obesity treatment in adolescents.

In the clinical treatment of pediatric obesity, outcomes other than change in BMI, such as improvements in cardiometabolic health or dyslipidemia,[26] are also important.

Examining these factors in relation to neonatal anthropometry was beyond the scope of this study, however.

Our study indicates that children and adolescents with obesity are likely to obtain a decrease in the degree of obesity following a multidisciplinary childhood obesity treatment protocol regardless of their prenatal growth conditions. Consequently, our findings do not support the individualization of obesity treatment based on neonatal anthropometric measures. ■

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Address for reprints: Ulrik Lausten-Thomsen, PhD, Department of Neonatology, Copenhagen University Hospital Rigshospitalet, Blegdamsvej 9, 2100 Copenhagen, Denmark. E-mail: ulrik.lausten-thomsen@regionh.dk

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Table II. Baseline characteristics and treatment response for the restricted sample (used for additionally adjusted analyses)

Characteristics/responses	All patients (n = 1401)	Girls (n = 754)	Boys (n = 647)
At birth			
Gestational age, wk, mean ± SD [range]	39.5 ± 1.8 [32-43]	39.5 ± 1.8 [32-43]	39.5 ± 1.9 [32-43]
Birth weight, kg, mean ± SD [range]	3.59 ± 0.59 [1.50-5.34]	3.56 ± 0.58 [1.50-5.15]	3.62 ± 0.59 [1.80-5.34]
Birth length, cm, mean ± SD [range]	52.2 ± 2.6 [38-59]	51.8 ± 2.6 [38-59]	52.5 ± 2.6 [41-59]
PI, kg/m^3, mean ± SD [range]	25.2 ± 2.6 [17.4-34.3]	25.4 ± 2.6 [18.1-34.3]	24.9 ± 2.6 [17.4-34.0]
BW-GA (SDS), mean ± SD [range]	0.10 ± 1.25 [−4.22 to 6.16]	0.15 ± 1.28 [−4.22 to 6.16]	0.04 ± 1.21 [−3.85 to 5.61]
SGA, %	12.3	12.2	13.0
LGA, %	15.9	16.6	13.6
Maternal smoking, yes/no, n (%)	408 (29.1)/993 (70.9)	222 (29.4)/532 (70.6)	186 (28.8)/461 (71.2)
Breastfeeding, n (%)			
0-3 mo	621 (44.3)	319 (42.3)	302 (46.7)
4-5 mo	421 (30.1)	240 (31.8)	181 (28.0)
6+ mo	359 (25.6)	195 (25.9)	164 (25.3)
At treatment start			
Age, y, mean ± SD [range]	11.0 ± 2.9 [5.0-18.9]	10.9 ± 3.1 [5.0-18.5]	11.0 ± 2.7 [5.0-18.9]
BMI SDS, mean ± SD [range]	2.88 ± 0.67 [1.64-6.12]	2.69 ± 0.54 [1.64-4.57]	3.09 ± 0.74 [1.65-6.12]
Height, cm, mean ± SD [range]	150.1 ± 15.9 [106.5-199.5]	148.9 ± 15.6 [106.5-182.4]	151.4 ± 16.1 [112.7-199.5]
Weight, kg, mean ± SD [range]	60.8 ± 21.8 [22.1-158.7]	60.1 ± 21.7 [22.1-131.9]	61.6 ± 21.9 [23.8-158.7]
Socioeconomic status, n (%)			
Grade 1	126 (9.0)	59 (7.8)	67 (10.4)
Grade 2	346 (24.7)	197 (26.1)	149 (23.0)
Grade 3	459 (32.8)	248 (32.9)	211 (34.6)
Grade 4	329 (23.5)	176 (23.3)	153 (23.7)
Grade 5	141 (10.1)	74 (9.8)	67 (10.4)
Treatment response			
Treatment duration, yr, mean ± SD [range]	1.23 ± 0.65 [0.50-3.96]	1.26 ± 0.54 [0.50-3.96]	1.19 ± 0.59 [0.50-3.84]
ΔBMI SDS, mean ± SD [range]	−0.32 ± 0.49 [−2.54 to 1.44]	−0.26 ± 0.47 [−2.54 to 1.44]	−0.39 ± 0.51 [−2.51 to 1.44]

Table IV. Association between neonatal anthropometric measures and degree of obesity at the start of treatment for the restricted sample (used for additionally adjusted analyses)

	All patients				Girls				Boys			
Anthropometric measures	n	β	95% CI	P	n	β	95% CI	P	n	β	95% CI	P
Gestational age, wk	1383	−0.015	−0.033 to 0.003	.10	742	−0.004	−0.025 to 0.018	.74	641	−0.023	−0.052 to 0.007	.13
Birth weight, kg	1401	0.079	0.022 to 0.136	**.006**	754	0.048	−0.019 to 0.114	.16	647	0.124	0.029 to 0.219	**.011**
Birth length, cm	1374	0.016	0.003 to 0.030	**.014**	739	0.011	−0.004 to 0.026	.14	635	0.025	0.002 to 0.047	**.033**
PI, kg/m^3	1374	0.004	−0.009 to 0.017	.52	739	−0.003	−0.018 to 0.011	.67	635	0.015	−0.006 to 0.037	.17
BW-GA (SDS)	1383	0.060	0.033 to 0.087	**.000**	742	0.029	−0.002 to 0.059	.066	641	0.101	0.055 to 0.148	**.000**
SGA	1383	−0.021	−0.128 to 0.084	.69	742	0.112	−0.013 to 0.236	.079	641	−0.176	−0.353 to 0.000	.051
LGA	1383	0.178	0.090 to 0.266	**.000**	742	0.151	0.054 to 0.247	**.002**	641	0.219	0.061 to 0.377	**.006**

Analyses were adjusted for age, sex, socioeconomic status, duration of breastfeeding, and maternal pregnancy smoking (see text for details). Significant values are in bold type.

Table VI. Association between neonatal anthropometric measures and change in degree of obesity during treatment for the restricted sample (used for additionally adjusted analyses)

	All patients				Girls				Boys			
Anthropometric measures	**n**	**β**	**95% CI**	***P***	**n**	**β**	**95% CI**	***P***	**n**	**β**	**95% CI**	***P***
Gestational age, wk	1383	0.003	−0.011 to 0.017	.65	742	−0.005	−0.024 to 0.014	.59	641	0.013	−0.008 to 0.034	.23
Birth weight, kg	1401	−0.026	−0.071 to 0.019	.25	754	−0.044	−0.000 to 0.015	.15	647	−0.004	−0.073 to 0.065	.91
Birth length, cm	1374	−0.001	−0.011 to 0.010	.89	739	−0.004	−0.018 to 0.009	.55	635	0.004	−0.012 to 0.020	.60
PI, kg/m^3	1374	−0.010	−0.020 to 0.000	.053	739	−0.010	−0.023 to 0.003	.13	635	−0.010	−0.026 to 0.005	.20
BW-GA (SDS)	1383	−0.018	−0.039 to 0.003	.10	742	−0.015	−0.042 to 0.011	.26	641	0.017	−0.055 to −0.013	.23
SGA	1383	−0.018	−0.100 to 0.065	.68	742	0.001	−0.108 to 0.110	.98	641	−0.037	−0.164 to 0.090	.56
LGA	1383	−0.065	−0.133 to 0.004	.064	742	−0.040	−0.125 to 0.045	.36	641	−0.102	−0.215 to 0.012	.079

Analyses were adjusted for age, sex, socioeconomic status, duration of breastfeeding, and maternal pregnancy smoking (see text for details).