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Longitudinal changes in infant body composition: association with childhood obesity

M. B. Koontz¹, D. D. Gunzler², L. Presley³, and P. M. Catalano³

¹Department of Pediatrics, Rainbow Babies & Children's Hospital, Case Western Reserve University, Cleveland, OH, USA

²Center for Health Care Research & Policy, MetroHealth Medical Center, Case Western Reserve University, Cleveland, OH, USA

³Department of Reproductive Biology, Center for Reproductive Health, MetroHealth Medical Center, Case Western Reserve University, Cleveland, OH, USA

Summary

Background—Rapid weight gain in infancy has been established as a risk factor for the development of later obesity.

Objective—We aimed to investigate the role of changes in infant body composition (assessed via total body electrical conductivity) on the development of overweight/obesity in mid-childhood.

Methods—Fifty-three term infants were evaluated at birth, three times during infancy and in mid-childhood. Logistic regression was used to determine associations between rates of total weight gain, fat mass gain and lean mass gain during infancy and later overweight/obesity (defined as body mass index [BMI] 85th percentile), adjusted for birth weight and parent education.

Results—At follow-up (age 9.0 ± 1.8 years), 30% were overweight/obese. More rapid total weight gain from 0 to 4 months was associated with twofold odds (odds ratio [OR] 1.98, 95% confidence interval [CI] 1.05–3.74, $P = 0.04$) of overweight/obesity in mid-childhood. From 0 to 8 months, more rapid weight gain was associated with nearly fivefold odds (OR 4.76, 95% CI 1.05–21.5, $P = 0.04$), and more rapid fat mass gain was associated with eightfold odds (OR 8.03, 95% CI 1.11–58.2, $P = 0.04$) of later overweight/obesity.

Address for correspondence: Dr MB Koontz, Pediatric Endocrinology & Metabolism, Rainbow Babies & Children's Hospital, 11100 Euclid Avenue, Suite 737, Cleveland, OH 44106, USA. michaela.koontz@case.edu.

Conflict of Interest Statement

The authors received funding from the National Institutes of Health to conduct this research. Otherwise, the authors have no conflicts of interest to disclose.

Author contributions

The authors' responsibilities were as follows – MBK and PMC conceptualized and designed the study; LP collected data; MBK and DDG performed data analyses; MBK, DDG and PMC interpreted the data; MBK wrote the first draft of the manuscript. All authors were involved in writing the manuscript and had final approval of the submitted version.

Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's web-site:

Table S1. (Alternate model). Associations between weight gain in infancy and childhood overweight/obesity status.

Conclusion—This exploratory study suggests that rapid weight gain, especially fat mass gain, in earlier infancy predisposes to mid-childhood overweight/obesity.

Keywords

Infant fat mass; infant weight gain; perinatal programming

Rapid weight gain in infancy has been established as a risk factor for the development of obesity in later childhood and adulthood (1–4). However, previous studies have been limited by lack of measures of changes in infant adiposity and body composition, and these factors may be better predictors of future obesity than simple weight gain (4). In addition, there has been conflicting evidence regarding critical periods of rapid early weight gain (5): some reports implicate the first 2 years of life (6,7) and others only early infancy (8–11), while other authors found no specific critical window (12). Our objective was to assess the relationship between changes in infant body composition during different infancy time periods and the development of overweight/obesity in mid-childhood.

The protocol was approved by the Institutional Review Board. Pregnant women were recruited from a clinic for a prospective study of infant growth through the first year of life. Mothers were contacted 6–11 years after birth to invite them to participate in follow-up studies of the children. Infants who were preterm, from multifetal gestations and those with congenital anomalies were excluded. At follow-up, exclusion criteria included medical conditions and medications affecting weight. Parents provided written informed consent; at follow-up, children gave assent.

Infants were evaluated at birth, 4 months (± 3 weeks), 8 months (± 3 weeks) and 12 months (± 3 weeks). Infant body composition was assessed via total body electrical conductivity (TOBEC; pediatric model HP-2, EM-SCAN, Inc., Springfield, IL, USA) at each time point. For four infants without TOBEC measurements at birth, body composition was estimated via skin-fold measurement using a validated formula (13). Maternal educational attainment, race and smoking status were obtained by history and review of the antenatal record at birth. Gestational diabetes was defined using National Diabetes Data Group criteria (14) and women with diabetes were managed according to our previously published protocol (15). At follow-up at 6–11 years of age, child's height and weight were measured and body mass index (BMI) calculated as weight in kilograms divided by the square of height in metres. Age- and sex-specific BMI percentiles were calculated using Centers for Disease Control and Prevention (CDC) growth charts (16), and children with BMI ≥ 85 th percentile were classified as overweight/obese (17).

Rates of weight gain, fat mass gain and lean mass gain for each infancy time period (0–4 months, 0–8 months and 0–12 months) were calculated as change in weight (or mass) between each time point and birth, divided by exact age in months at that visit, and expressed as 100 g per month. Separate logistic regression models were used to determine associations between rates of total weight gain, fat mass gain and lean mass gain for each infancy time period (main exposure variables) and later overweight/obesity status (main outcome). Regression models included demographic and perinatal variables that have been shown in prior research to be associated with childhood obesity (17–19). Models were thus

adjusted for birth weight and maternal education (categorical); age and gender were not included as covariates because these are already incorporated into the BMI percentile-based definition of overweight/obesity. The small sample size limited the number of covariates that could be included in models. Alternate models were adjusted for maternal smoking status and gestational diabetes exposure.

Results of regression models are presented as odds of being overweight/obese vs. non-overweight/obese in mid-childhood per 100 g per month increase in weight/mass during infancy. In other words, an odds ratio (OR) of 2 corresponds to a twofold odds of being overweight/obese in mid-childhood per 100 g increase in weight/mass during that infancy time period. P -value < 0.05 was considered significant. Statistical analyses were performed using SPSS (IBM SPSS, version 20, Chicago, IL, USA) or Statistical Analysis Software version 9.2 (SAS Institute, Cary, NC, USA).

Demographic characteristics are presented in Table 1. Results of logistic regression models are presented in Table 2. For each 100 g month⁻¹ increase in weight gain from 0 to 4 months, there was a twofold odds of being overweight/obese in mid-childhood (95% confidence interval [CI] 1.05–3.74, $P = 0.04$). From 0 to 8 months, each 100 g month⁻¹ increase in weight gain was associated with nearly fivefold odds (95% CI 1.05–21.5, $P = 0.04$) of later overweight/obesity, and each 100 g month⁻¹ increase in fat mass gain was associated with eightfold odds (95% CI 1.11–58.2, $P = 0.04$) of later overweight/obesity. No significant associations were found for rates of weight gain or fat mass gain from 0 to 12 months, or for rates of lean mass gain at any time interval. Similar results were obtained when maternal smoking status and gestational diabetes exposure were substituted for birth weight and maternal education in the analytic models (Table S1).

Our results demonstrate for the first time a relationship between fat mass accrual during infancy and later overweight/obesity. We also show an association between rapid weight gain in early infancy and childhood overweight/obesity, a finding that is in agreement with some (8–11) but not with other (7,12) prior studies. We found that mid-childhood overweight/obesity was more strongly associated with rapid fat mass gain than rapid total weight gain, suggesting that excessive deposition of adipose tissue (but not lean mass) during infancy is a key factor in the future development of obesity. The conflicting findings from previous studies regarding the relationship between weight gain in infancy and later obesity may be due to the lack of body composition data in those studies.

The mechanisms underlying the relationship between weight gain in infancy and subsequent development of obesity are not entirely clear. Adipogenesis occurs primarily during prenatal and early postnatal development, therefore excessive deposition of adipose tissue during early infancy may induce a developmental response (i.e. ‘perinatal programming’) within adipocytes (20). Recent studies suggest that the programming response involves epigenetic and transcriptional changes, endoplasmic reticulum stress and/or reactive oxygen species (21,22).

A major strength of our study is inclusion of measures of body composition during multiple time points in infancy, which is a unique contribution to the literature. Other strengths

include the prospective design and long-term follow-up. Our study was limited by lack of data on infant nutrition (feeding mode, weaning and maternal diet), which have been shown to be important influences on adiposity and/or weight trajectories during infancy (23–27). Other limitations include the small sample size, lack of racial/ethnic diversity and lack of data on pubertal status. Given the limited sample size, these results should be considered exploratory and larger samples are needed to verify these findings. Additional studies of infant body composition, particularly longitudinal assessments, could inform efforts to improve nutritional management during this critical time of growth (20,28).

In conclusion, our results suggest that rapid weight gain in earlier infancy predicts mid-childhood overweight/obesity. Fat mass accrual seems to contribute to this association more than lean mass accrual. Preventing childhood obesity requires further research to establish optimal infant growth patterns and determinants of growth, which likely include infant nutritional practices as well as maternal diet/weight gain (29).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Characteristics of the participants

	Number of observations	Mean or proportion	SD
Gender (% female)	53	64	
Race (% Caucasian)	53	98	
Gestational age (weeks)	53	39.2	1.3
Maternal smoking status (% smokers)	53	13	
Gestational diabetes status (% born to mothers with gestational diabetes)	53	38	
Birth weight (g)	53	3360	492
Birth fat mass (g)	53	378	179
Birth lean mass (g)	53	2974	387
Birth to 4 months			
Rate of weight gain (g month ⁻¹)	39	769	171
Rate of fat mass gain (g month ⁻¹)	38	350	116
Rate of lean mass gain (g month ⁻¹)	38	412	87
Birth to 8 months			
Rate of weight gain (g month ⁻¹)	31	616	112
Rate of fat mass gain (g month ⁻¹)	31	258	68
Rate of lean mass gain (g month ⁻¹)	31	359	60
Birth to 12 months			
Rate of weight gain (g month ⁻¹)	32	499	89
Rate of fat mass gain (g month ⁻¹)	31	180	58
Rate of lean mass gain (g month ⁻¹)	31	320	47
Child age at follow-up (years)	53	9.0	1.8
Child BMI at follow-up (kg m ⁻²)	53	18.0	3.0
% overweight/obese at follow-up	53	30	

BMI, body mass index; SD, standard deviation.

Table 2Associations between weight gain in infancy and childhood overweight/obesity status[†]

Variables	OR	95% CI	P-value
0–4 months			
Total weight gain (100 g month ⁻¹)	1.98	1.05–3.74	0.04
Fat mass gain (100 g month ⁻¹)	1.91	0.78–4.68	0.16
Lean mass gain (100 g month ⁻¹)	2.42	0.78–7.58	0.13
0–8 months			
Total weight gain (100 g month ⁻¹)	4.76	1.05–21.5	0.04
Fat mass gain (100 g month ⁻¹)	8.03	1.11–58.2	0.04
Lean mass gain (100 g month ⁻¹)	5.06	0.71–36.2	0.11
0–12 months			
Total weight gain (100 g month ⁻¹)	1.55	0.59–4.07	0.37
Fat mass gain (100 g month ⁻¹)	1.67	0.34–8.20	0.53
Lean mass gain (100 g month ⁻¹)	2.55	0.41–15.7	0.31

[†] Adjusted for birth weight and maternal education (categorical). Age and gender were not included because they are already incorporated into the definition of the outcome variable (body mass index percentile-based overweight/obesity status). CI, confidence interval; OR, odds ratio.