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Does the association between early life growth and later obesity differ by race/ethnicity or socioeconomic status? A systematic review

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Abstract

Purpose: Rapid growth during infancy predicts higher risk of obesity later in childhood. The association between patterns of early life growth and later obesity may differ by race/ethnicity or socioeconomic status (SES), but prior evidence syntheses do not consider vulnerable subpopulations.

Methods: We systemically reviewed published studies that explored patterns of early life growth (0-24 months of age) as predictors of later obesity (>24 months) that were either conducted in racial/ethnic minority or low-SES study populations or assessed effect modification of this association by race/ethnicity or SES. Literature searches were conducted in PubMed and SocINDEX.

Results: Ten studies met inclusion criteria. Faster growth during the first 2 years of life was consistently associated with later obesity irrespective of definition and timing of exposure and outcome measures. Associations were strongest in populations composed of greater proportions of racial/ethnic minority and/or low-SES children. For example, ORs ranged from 1.17 (95% CI: 1.11, 1.24) in a heterogeneous population to 9.24 (95% CI: 3.73, 22.9) in an entirely low-SES non-white population.

Conclusions: The impact of rapid growth in infancy on later obesity may differ by social stratification factors such as race/ethnicity and family income. More robust and inclusive studies examining these associations are needed.

Keywords

Overweight; Review [Publication Type]; Infant; Weight Gain; Growth and Development; Ethnic Groups; Continental Population Groups; Socioeconomic Factors; Social Environment

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INTRODUCTION

In the United States, 33.4% of children and adolescents are overweight and 26.1% are obese [1]. Disparities in obesity persist, with obesity prevalence as high as 33.4% and 31.5% in Black girls and Hispanic boys, respectively [1]. Further, obesity prevalence has increased by as much as 33% for children in low-education, low-income, and higher unemployment households [2]. Children with obesity are at greater risk of developing adverse health outcomes such as type 2 diabetes and metabolic syndrome [3]. As such, disparities in childhood obesity based on race, ethnicity, and socioeconomic status (SES) likely further contribute to health disparities in adulthood.

Systematic reviews of over 25 studies suggest that faster growth in weight, length, relative weight, and adiposity during infancy are associated with elevated risk for obesity in childhood and beyond [4–9]. The study populations in these reviews are, naturally, those represented in most empirical studies on the topic: predominantly white race, and moderate- to high-SES subgroups or national SES profiles [4–9]. Social gradients are observed for obesity and obesity-related infant growth patterns [10], but few studies consider the degree to which infant growth patterns predict later obesity differentially by race/ethnicity or SES.

Differential associations by race/ethnicity or SES may result from interactions between prenatal and postnatal drivers of infant growth patterns. Maternal exposures – such as poor nutrition and environmental toxins – have been shown to modify fetal growth and gestational age at birth [11], both of which shape growth patterns in infancy and beyond [12]. Moreover, developmental alterations resulting from prenatal conditions may intensify responses to infant growth [10]. Minority race/ethnicity or lower SES mothers are not only disproportionately exposed to critical determinants of prenatal development -- poor nutrition [13], environmental toxins [14], and elevated stress levels [15] – but also face inadequate supports for healthy postnatal practices such as breastfeeding [19] due to inadequate work place breastfeeding policies [16].

We hypothesized that, as a result of differences in prenatal development and postnatal conditions, minority race/ethnicity or lower SES infants are more sensitive to variations in early life growth. The objective of this paper is to systematically review the epidemiologic literature regarding the association between patterns of early life growth and later obesity in racial/ethnic minority or low-SES subgroups in Western countries.

MATERIALS and METHODS

Literature search strategy

We conducted literature searches within PubMed and SocINDEX for published studies that examined early life growth as a predictor of later obesity in racial/ethnic minority or low-SES children. We applied search terms for concepts of (a) growth (e.g. weight gain, body mass index (BMI) trajectory), (b) early life (e.g. infant, newborn), (c) social divisions (e.g. race, ethnicity, SES), and (d) obesity or overweight. We also employed use of medical subject headings (MeSH) and text terms (Appendix A). Our search, conducted in April 2016, was restricted to papers published after January 1, 2000. This date was selected

because general population studies on the topic first appeared around 2000, and papers featuring social subgroups followed. Further, our review of reference lists (described below) identified no relevant papers published prior to 2000.

Inclusion criteria

We included published English language papers of original observational studies that examined anthropometric measurements (e.g. weight, height, skin-fold thickness) at two or more time points between birth and 24 months, which were subsequently used to predict later obesity (>24 months). Our objective was to determine if the association between early life growth and later obesity differed by race/ethnicity or SES. While within-study comparisons were ideal for addressing this research question, the sparse state of the current literature in the field necessitated broadening our scope to include studies that focused on subgroups. As such, we included studies that (a) examined associations between early life growth and later obesity within racial/ethnic minority (any race/ethnicity other than non-Hispanic white) or low-SES (any indicator or proxy of low-SES e.g. low-income, low educational attainment, or unskilled occupation) study populations or (b) assessed effect modification of this association by social division (race/ethnicity, income, education, or a SES index or proxy measure) in broader populations. Due to our interest in growth within the context of the Western food environment, we only included studies examining early life growth that occurred in Western countries.

Review of candidate papers

Each paper found in the database search was independently examined by two members of the study team. Each study team member reviewed paper titles, and papers that were clearly outside the review topic were excluded. If the study title did not provide sufficient information to determine inclusion status, the abstract and then the full text were read. Discrepant determinations were resolved by a third reviewer. We used the same process to review studies listed in the reference lists of included papers.

Extraction and study characteristics

For each included paper, two reviewers independently extracted information pertaining to the study design and results. Extracted study characteristics included exposure and outcome definition and ascertainment, racial/ethnic and SES composition of the study population, covariates and modifying variables such as maternal BMI and feeding mode, and degree and management of study attrition (Appendix B).

Quality assessment

To assess each included study's resiliency from bias, we employed a modified version of the NIH Quality Assessment Tool for Observational Cohort and Cross-sectional Studies (Appendix B; [17]). Two independent reviewers appraised each study for resiliency from bias and all studies met more than 60% of the quality parameters. Because our goal was to report the quality and make recommendations for future research, rather than synthesizing the data in a meta-analysis we included all studies. This study inclusion approach was consistent with the emerging nature of this research topic. The number of quality parameters

met by each study can be found in Appendix C and the implications of unmet quality parameters are examined in the discussion.

RESULTS

Study selection

PubMed and SocINDEX searches identified 1677 unique articles (Figure 1). Of these, 1564 articles were excluded based on title or abstract, leaving 113 articles for full text review; nine were included. Review of reference lists yielded one article, for a total of 10 articles. The primary reasons for exclusion of abstracts were the absence of a measure of early life growth or the examination of early life growth as an outcome. The primary reason for exclusion of full text articles was the use of a population without restriction to, or examination of effect modification by, race/ethnicity or SES.

Study characteristics

With the exception of one German study [18], the studies examined US populations primarily sourced from urban Northeastern hospitals and clinics (Table 1). Studies examined eight unique cohorts of infants born from 1959 through 2010, though most cohorts were born between 1990 and 1999. The majority of studies focused on race/ethnicity or SES subgroups; only four studies involved within-study comparisons [18–21].

Relationship between early life growth and later obesity

Overall, nine of the ten studies found that more rapid early life growth velocity was associated with a greater risk of later obesity (Table 2) or higher BMI (Table 3) (ranging from 25 months to 23 years). Study populations for eight studies were either low-SES [21,22] and/or completely [19,23] or mostly (69% to 93%; [22,24–27]) racial and/or ethnic minorities. These items were ascertained using electronic health record (EHR) data in four studies [22,23,26,27], and from maternal self-report surveys [18,21] or interviews [19,20,24,25] in six studies. Items were limited to standard classifications of race/ethnicity, income, and education. Additional facets of race/ethnicity such as language, migration status, and rurality were not reported. Associations between early life growth and later obesity ranged from moderate (OR: 1.17 [95% CI: 1.11, 1.24]; [20]) to strong (OR: 9.24 [95% CI: 3.73, 22.9]; [22]). Stronger associations tended to be in study populations with higher proportions of low-SES and nonwhite children; however, direct comparison was difficult due to differences in exposure definitions. In the four studies that performed tests for effect modification, no evidence of interaction was observed between early life growth and Black race [20], early life growth and Hispanic ethnicity [21], or early life growth and maternal education [18,19]. Interaction terms were not included in final models for these reports.

Study design considerations

Timing.—The majority of studies (70%) focused on a single period of early life growth, capturing growth from birth to, by our definition, as late as 24 months. All studies defined early life growth as growth starting at birth (8 studies) or between 2 and 7 days (2 studies [23,26], with a median growth interval of 6 months (70% with periods of 12 months or

shorter). Two studies examined a second period of growth between 4 and 12 months [23,27]. Subsequent obesity was ascertained as early as 25 months and as late as 23 years of age (Median: 4.75 years; 3 to 11 years in 90% of studies).

In our qualitative comparison of findings between studies, the strength and magnitude of the association between early life growth and later obesity did not vary based on period of early life growth. However, for the two studies that examined more than one period of growth, intervals that began at ages 4 to 12 months generated associations of greater strength and magnitude [23,27]. Similarly, the strength and magnitude of the association did not vary based on the age at which subsequent obesity was ascertained.

Classification.—Early life growth was typically characterized as the difference in weight or relative weight between two time points ([18–25,27]; Appendix C). Obesity was typically characterized by standardizing BMI and/or skinfold thickness relative to age- and sex-specific 2001 or 2005 CDC growth chart data, and subsequently categorized [19–23,25,26]. Associations were strongest in the three studies examining a binary indicator of rapid growth as a predictor of categorically-defined obesity [19,22,23]. However, comparison of magnitudes of association among exposure definitions was challenging due to vast differences in scaling of early life growth.

Covariates.—All studies met a minimum of 7 of the 12 quality criteria (Appendix C) and all but one [27] included minimal adjustment for sex, birth size, and SES. Four studies adjusted for breastfeeding during infancy [18,21] or fruit, vegetable, cereal and grain consumption during childhood (>24 months) [24,25] and one study adjusted for maternal substance use (e.g. tobacco; [24]). The effect measures for the relationship between early life growth and later obesity in these studies were attenuated compared to studies that did not adjust for these dietary and maternal substance use factors.

Other effect modifiers.—Associations between rapid infant growth and later obesity were stronger in infants born to mothers who smoked during pregnancy [18], were overweight prior to pregnancy [18], and who gave birth preterm [24].

Exclusion criteria.—Many studies excluded preterm births; associations between rapid infant growth and later obesity were particularly strong in studies that included births irrespective of gestational age [21–23,27] or restricted their sample to preterm births [24,25]. Similarly, infant growth-obesity associations were weaker in studies that excluded multiple births [18–20,24,25], non-biological mother-child pairs [21], and non-English speakers [24,25].

Missing data.—Missing data was the most frequent study quality issue in this review, with all 10 studies reporting greater than 20% missing data. Missingness was attributed to both attrition and incomplete data collection at a single time point. For example, of the 577 eligible infants in Gaskins et al.'s (2010) study, 11-year follow up data was available for 68% (n=393), and 79% (n=312) had complete data for the relevant covariates [25]. In our review, the study composed of the greatest concentration of minority race/ethnicity or lower SES individuals (32% Black, 54% Hispanic, 3% other race; 85% on Medicaid, 100% low-

income; [17]) reported the highest amount of missing data (62%; Table 2) and the study with the lowest concentration of minority race/ethnicity or lower SES individuals (exact numbers undisclosed but largely ethnically German; [13]) reported the lowest amount of missing data (28%; Table 2).

DISCUSSION

In this systematic review of published studies on early life growth patterns and later obesity, we found that studies in populations with the highest concentrations of minority race/ethnicity or lower SES showed the strongest, most consistently positive associations. However, the available evidence is predominantly based on subgroup-specific populations with few studies that formally tested effect modification. This summary is based on 10 studies and approximately 15,000 individuals observed since birth.

This study expanded on previous reviews [4–6] by assessing the relationship between early life growth and later obesity within and across racial/ethnic and SES subgroups. As noted in most previous systematic reviews [4,5], meta-analyses was not possible due to the heterogeneity of exposure and outcome definitions. In one exception, Ong and Loos standardized the results of 15 studies, yielding a pooled adjusted odds ratio of 1.84 [6]. In our review, we observed consistency in the direction of the association between early life growth and later obesity irrespective of great inter-study variability, supporting the robustness of these associations.

Potential mechanisms

Reasons that associations between early life growth and later obesity are stronger in minority race/ethnicity or lower SES children are unknown, but could reflect SES or race/ethnic differences in prenatal development, differences in postnatal drivers of growth, or both.

Prenatal development.—Prenatal exposures – indicated by body size at birth, maternal tobacco use and social stature, or other metrics – by way of adverse developmental alterations, may influence associations between early life growth and later obesity in two ways. First, prenatal exposures such as maternal smoking may increase propensity for both rapid infant growth [28] and later obesity [29] through alterations to the fetal brain that lead to appetite and impulse control dysfunction [30,31]. That is, associations between early life growth and later obesity may reflect effects of, for example, smoking-induced prenatal perturbations, rather than early life growth *per se*. Given that key prenatal exposures such as smoking and maternal obesity are most common in the lowest SES stratum [32], stronger associations between early life growth and later obesity in lower SES groups could reflect differential confounding by prenatal exposures. In our review, the association between early life growth and later obesity was stronger in studies that did not adjust for maternal pre-pregnancy weight or tobacco use [22,23].

Second, prenatal alterations may increase sensitivity to any causal effects of abnormal early life growth patterns [32]. For example, modified glucose and lipid metabolism [33] resulting from adverse prenatal conditions could intensify the impacts of rapid growth on long-term

appetite and metabolic regulation. This mechanism is supported by findings that rapid infant growth is more strongly associated with obesity among infants with indicators of adverse prenatal conditions – preterm infants [24] and infants born to mothers who were overweight and/or smoked tobacco during pregnancy [18]. Gestational age at birth can be indicative of maternal exposure to elevated stress levels, poor nutrition, and exposure to environmental toxins [14], all more common in low-income women [13,15,34]. Preterm birth is strongly associated with maternal race and SES [35–37] and is a known risk factor for both rapid infant growth and later obesity [38].

Contributions of maternal exposures to race/ethnicity and/or SES-based differences in the association between early life growth and later obesity require further empirical testing. Further, parsing out the causal role of early life growth from the roles of prenatal drivers of postnatal growth requires expanding multi-disciplinary longitudinal intergenerational cohort study designs – that capture detailed high-quality data on the children, their parents, and potentially their grandparents [39] – to currently underrepresented minority race/ethnicity and lower SES populations [40].

Postnatal drivers of growth.—In our review, the association between early life growth and later obesity was attenuated in studies that adjusted for breastfeeding [18,21]. Infant feeding practices (e.g. breastfeeding, complementary feeding) are perhaps the most established determinants of early life growth [41]. Systemic barriers to meeting recommended breastfeeding practices - such as inadequate paid leave policies and inequitable wages - disproportionately affect the children of racial and ethnic minorities and individuals in lower socioeconomic classes [16]. As a result, infant feeding practices, or other socially patterned drivers of early life growth, may contribute to race/ethnicity and/or SES-based differences in the association between early life growth and later obesity.

Future research needs.—Understanding the mechanisms underlying the strong associations observed in socially disadvantaged populations is critical for designing early intervention strategies that target the most important drivers of obesity in our most disadvantaged children. In particular, further research is needed to disentangle and identify the specific prenatal and postnatal drivers and deterrents of obesity among racial/ethnic minority and low-SES children with special attention to maternal exposures, infant feeding practices, and the role of social policy.

Methodological challenges and implications for future research

Existing research on childhood obesity underrepresents the children most likely to be affected. Yet research in low-SES and racial/ethnic minority children poses several important challenges. These challenges may contribute to subgroup differences in associations between early life growth and later obesity, and limit the field's ability to advance knowledge in early life determinants of obesity in minority race/ethnicity or lower SES children.

Population inclusivity.—The majority of existing studies of the association between early life growth and later obesity have been conducted in more affluent non-Hispanic white children. We report here evidence from the ten studies that consider the role of race/ethnicity

and/or SES, only four of which included within study comparisons. Recruitment and enrollment is challenging in minority race/ethnicity or lower SES subgroups due to factors such as mistrust, language barriers, barriers due to low educational attainment, and competing demands between work and caretaking [42]. Two studies employing data from the Maternal Lifestyle Study had a remarkably low Latina inclusion rate relative to the target populations (e.g. <7% in the study population vs. up to 53.6% among residents of participating cities) [24,25,43], possibly reflecting language or other barriers to study participation. Participatory research approaches, ensuring key supports (e.g., provision of transport vouchers, waiver of clinic fees) [32,33], and provision of study materials in multiple languages [44] are critical for promoting participation in diverse populations. These costly but essential expenses require flexibility and accommodation by research funders.

Characterizing race, ethnicity, and SES.—Race, ethnicity, and SES were characterized broadly and unidimensionally. Additional facets of race/ethnicity, such as country of origin and language, were not reported in any of the eligible studies. This approach to characterization of these factors, while standard, can distort within-group differences that may be important to early life growth and the associations of interest. Different social forces may be operating in different ethnic minority groups, but these cannot be studied without collection of more detailed social factors. Similarly, with the exception of one study that accounted for marital status, education, employment status and occupational prestige in their measure of SES [25], income or education were used as imperfect proxies for SES. Moreover, the current body of literature on racial/ethnic minorities is largely limited to Black and Hispanic women and their children; Asians were underrepresented in eligible literature. Further, incomplete data collection contributed to a large proportion of missingness observed in this review; minority race/ethnicity and lower SES status are disproportionately more likely to be missing [49]. Importantly, EHR is increasingly used and valuable for providing rich longitudinal data on large cohorts, but race, ethnicity, and SES data are routinely captured unidimensionally and less completely in this data source. As such, initiatives that incentivize hospitals to improve their services and provide ample training and infrastructure to staff to accurately collect additional demographic information may be needed to provide critical support for both immediate patient care and future research [46].

Study duration.—Studying the relationship between early life growth and later obesity requires a cohort design of longer than two years in duration. Attrition is expected for any study of that length, but minority race/ethnicity or lower SES populations have higher loss to follow-up [23] than predominantly white and more affluent populations in both prospective and retrospective cohorts. Restriction of analyses to complete cases, as observed in most of the studies in this review, could lead to bias in outcome ascertainment or sample selection. In our review, associations between early life growth and later obesity were generally consistent irrespective of age at outcome ascertainment, suggesting that attrition bias was not a powerful driver of the associations in our review. However, advancing methods to improve study retention are critical in order to investigate later life outcomes, such as prediabetes and diabetes.

Exclusion of preterm births.—Forty percent of included studies restricted their analytic sample to term births [18–20,26]. Given the many common causes of preterm birth, low birth weight, and subsequent growth patterns, exclusion of preterm births could lead to selection bias, attenuating the association between early life growth and later obesity in race/ethnicity and/or SES subgroups. The statistically non-significant race/ethnicity or SES interaction results found in three studies that excluded preterm births [18–20] could reflect this bias. Our findings suggest that future studies on this topic consider including preterm and low birth weight infants as well as analytically testing for interaction by gestational age and birth weight.

Adjustment for potential mechanisms.—Four studies adjusted for breastfeeding status and childhood diet [18,21,24,25] and one study adjusted for maternal tobacco use [24], factors we postulate contribute to race/ethnicity and SES-based differences in the early life growth-later obesity association. The attenuated relationship in these four studies compared to studies that did not adjust for infant feeding or childhood diet is consistent with this hypothesis. The decision to adjust for these factors in future studies comparing race/ethnicity and SES subgroups should explicitly consider the question of interest. Early life-later obesity associations adjusted for these potential mechanisms reflect the estimated effect of early life growth on obesity, independent of the examined mechanisms. Associations adjusted for all hypothesized mechanisms, for example, reflect the biologic effects of rapid growth during infancy. Associations adjusted for one or more selected mechanisms reflect the residual association not explained by the included mechanisms, perhaps to identify additional mechanisms. In contrast, associations not adjusted for primal drivers of growth provides the overall estimated effect of early life growth and its proximal drivers; this estimate is useful for estimating health benefits of promoting healthy growth through breastfeeding and other supports.

Conclusion

The relationship between more rapid early life growth and later obesity is strongest among racial/ethnic minority and/or low-SES individuals. Yet, clinical recommendations and public health guidelines for infant growth are largely informed by research findings from predominantly white and moderate-to-high SES populations. Further research to clarify subgroup differences in associations between early life growth and obesity and to understand the mechanisms driving these differences is necessary to inform policy for obesity prevention and the reduction of health disparities.

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Appendix A. Search terms

Concept	Terms for Pubmed	Terms for SocINDEX
Growth	Search (“catch-up growth” OR “compensatory growth” OR “growth trajectory” OR “rapid growth” OR “Weight Gain”[Mesh] OR “Body Weight”[Mesh] OR “Adiposity”[Mesh] OR “Growth and Development”[Mesh] OR “weight gain” OR “body weight” OR “adiposity” OR “growth and development”)	Search (“catch-up growth” OR “compensatory growth” OR “growth trajectory” OR “rapid growth” OR “weight gain” OR “body weight” OR “adiposity” OR “growth and development”)
Early life	Search “Infant”[Mesh] or “early life” or “infan*” or “neonat*” or “newborn” or “postnatal” OR “early weight gain”	Search “early life” or “infan*” or “neonat*” or “newborn” or “postnatal” OR “early weight gain”
Social divisions	Search (“Ethnic Groups”[Mesh] OR “Continental Population Groups”[Mesh] OR “Socioeconomic Factors”[Mesh] OR “Health Status Disparities”[Mesh] OR “Residence Characteristics”[Mesh] OR “Social Environment”[Mesh] OR “Social Welfare”[Mesh] OR “inner-city” or “socioeconomic” or “race” or “ethnic*” or “income” or “social class” or “education” or “occupation”)	Search (“inner-city” or “socioeconomic” or “race” or “ethnic*” or “income” or “social class” or “education” or “occupation”)
Obesity/ overweight	Search (“Overweight”[Mesh] or “obes*” or “overweight” or “body mass index”)	Search (“obes*” or “overweight” or “body mass index”)
Excluding Locations	Search (“Africa”[Mesh] OR “Asia”[Mesh] OR “Indian Ocean Island”[Mesh] OR “Indonesia”[Mesh] OR “Japan”[Mesh] OR “Macau”[Mesh] OR “Mediterranean Islands”[Mesh] OR “Philippines”[Mesh] OR “Taiwan”[Mesh] OR “Caribbean Region”[Mesh] OR “Central America”[Mesh] OR “Gulf of Mexico”[Mesh] OR “Latin America”[Mesh] OR “South America”[Mesh])	
Restrictions	Filters: Publication date from 2000/01/01	

Appendix B. Study extraction and quality assessment tool

Reviewer	Click here to enter text.
First Author	Click here to enter text.
Year	Click here to enter text.
Journal	Click here to enter text.
Title	Click here to enter text.

Extraction Component		Details
Study characteristics		
	Aim/objectives of study	Click here to enter text.
	Name (if established cohort)	Click here to enter text.
	Study inclusion criteria (e.g. singleton, AGA, healthy)	Click here to enter text.
	Recruitment procedures used	Click here to enter text.
	Geographic location (Be as specific as possible)	Click here to enter text.
Participant characteristics		
	Overall Size (N=?) Analytic sample (N=?)	Click here to enter text.

Extraction Component		Details
	Baseline (N=?)	
	Birth year(s)	Click here to enter text.
	Social division composition (e.g. % black, % below FPL, WIC)	Click here to enter text.
	Setting (e.g. clinic, population)	Click here to enter text.
	Exposure and covariates	
	Exposure defined	Click here to enter text.
	Exposure ascertainment (e.g. linkage to EMR)	Click here to enter text.
	Exposure time points	Click here to enter text.
	(e.g. Birth to 6 months)	
	Adjusting covariates	Click here to enter text.
	Modifying variables (e.g., sex, race/ethnicity)	Click here to enter text.
	Outcome	
	Defined (e.g. Age/sex-specific BMI of 85 th percentile – 2000 CDC growth charts)	Click here to enter text.
	Age(s) at outcome	Click here to enter text.
	Analytic method	Click here to enter text.
	Missing Data (e.g. how did they deal with missing data, what proportion missing, etc)	Click here to enter text.
	Results (e.g. ORs, RRs, beta coefficients) <i>If subgroup analysis presented, reports for each subgroup</i> <i>If available, present crude and adjusted.</i>	Click here to enter text.
	Notes	Click here to enter text.

Criteria	Yes	No	Other*	Notes
1. Research question or objective clearly stated	☐	☐	Choose an item.	Click here to enter text.
2. Study Population clearly defined	☐	☐	Choose an item.	Click here to enter text.
3. Participation rate of eligible persons at least 50%	☐	☐	Choose an item.	Click here to enter text.
4. All subjects selected from the same or similar populations (including the same time period)	☐	☐	Choose an item.	Click here to enter text.
5. Inclusion and exclusion criteria for being in the study pre-specified and applied uniformly to all participants	☐	☐	Choose an item.	Click here to enter text.
6. Exposures of interest were measured prior to the outcome being measured	☐	☐	Choose an item.	Click here to enter text.
7. For exposures that can vary in amount or level, the study examined different levels of the exposure as related to the outcome (e.g., categories of exposure, or exposure measured as continuous variable)	☐	☐	Choose an item.	Click here to enter text.
8. Exposure measures (independent variables) were clearly defined, valid, reliable, and implemented consistently across all study participants	☐	☐	Choose an item.	Click here to enter text.
9. Outcome measures (dependent variables) were clearly defined, valid, reliable, and implemented consistently across all study participants	☐	☐	Choose an item.	Click here to enter text.

Criteria	Yes	No	Other [*]	Notes
10. Loss to follow-up after baseline was 20% or less	☐	☐	Choose an item.	Click here to enter text.
11. Key potential confounding variables were measured and adjusted statistically for their impact on the relationship between exposure(s) and outcome(s)	☐	☐	Choose an item.	Click here to enter text.

^{*} CD, cannot determine; NA, not applicable; NR, not reported

Appendix C. Supplemental Table

Table 4.

Study design details

First author, year	Exposure Definition Period	Outcome Definition Age at Ascertainment	Exclusions	Control Variables	N ¹	Proportion of Baseline Included in Analysis, %	Result	No. of Quality Criteria Met ²
Dennison, 2006	Rate of weight gain (100g/mo) Birth to 6 months	BMI 85th (at-risk for overweight) & BMI 95th (overweight) age and sex-specific percentiles 4 years	(1) birth weight < 1800 grams (2) Breast fed > 1 day but < 1 month (3) < 2 follow-up measurements	Model 1: Ethnicity, birth weight, sex, breast-feeding history Model 2: Model 1 + maternal BMI	616 276	44.7% 20.0%	OR (95% CI) At-risk for overweight: 1.43 (1.27,1.60) Overweight: 1.44 (1.26,1.65) OR (95% CI) At-risk for overweight: 1.41 (1.18,1.68) Overweight: 1.44 (1.35,2.21)	8
Gaskins, 2010	Weight change per month (g/mo) Birth to 12 months	BMI 85th (overweight) & BMI 95th (obese) age and sex-specific percentiles 11 years	(1) age < 18yrs (2) history psychosis or institutionalization for retardation or emotional problems (3) language barriers (4) multiple gestation (5) birthweight < 501g (6) gestational age < 37wks (7) low infant survivability (8) maternal intent to move out of catchment area (9) chromosomal abnormality (10) toxoplasmosis, other agents, rubella, cytomegalovirus, or herpes infection	Model 1: Site, prepregnancy BMI, pregestational diabetic history, birth weight, sex, race/ethnicity, size for gestational age, exercise, television viewing, fruits and vegetable consumption, grain and cereal consumption.	312	54.1%	OR (95% CI) Overweight: 1.61 (1.07,2.43) Obese: 2.69 (1.80,4.00)	9
Goedell, 2009	Rapid weight gain Birth to 4 months & Birth to 12 months	85th BMI 95th (overweight), 95th BMI < 97th (obese), and BMI 97th (extremely obese) age and sex-specific percentiles 24-38 months	Records missing: (1) sex (2) birth weight (3) height and/or weight from same visit at 4 months, 1 years or between 24 and 38 months	Model 1: Unadjusted Model 2: Sex and birth weight	203 203	38.4% 38.4%	OR (95% CI) Rapid weight gain birth to 4 months - Overweight: 2.49 (0.97,6.38) - Obese: 1.94 (0.89,4.21) Rapid weight gain birth to 12 months - Overweight: 2.06 (0.86,4.92) - Obese: 9.24 (3.73,22.91) OR (95% CI) Rapid weight gain birth to 12 months - Extremely obese: 11.7 (4.5,30.0)	7

First author, year	Exposure Definition Period	Outcome Definition Age at Ascertainment	Exclusions	Control Variables	N ^J	Proportion of Baseline Included in Analysis, %	Result	No. of Quality Criteria Met
Karaolis-Danckert, 2008	Rapid weight gain Birth to 24 months	Body fat % & BMI SDS Trajectories 2-6 years	(1) multiple gestation (2) gestational age <37 weeks or >42 weeks (3) birth weight 2500 g (4) birth weight and length inappropriate-for-gestational age (5) systemic disease, growth disorders (eg, growth hormone deficiency), chromosomal or congenital abnormalities, or abnormalities of the heart, gastrointestinal tract, or urogenital tract; (6) incomplete information on breastfeeding status, maternal anthropometric measures, and in utero tobacco exposure	Model 1: Interaction between rapid weight gain and time Model 2: Model 1 + BMI SD score at birth, sex, gestational age group, in utero tobacco exposure, maternal overweight, interactions (in utero tobacco exposure*maternal overweight, rapid weight gain*in utero tobacco exposure, rapid weight gain*maternal overweight; time interactions with sex, in utero tobacco exposure, maternal overweight, bottle-feeding, season of birth, and rapid weight gain; 3-way interactions with time and rapid weight gain with: in utero tobacco exposure and maternal overweight)	370	72.0%	$\beta \pm SE$ Body fat % -Rapid weight gain: 1.83±0.35 -Time*rapid weight gain: 0.10±0.13 BMI SDS Trajectories -Rapid weight gain: 0.91±0.10 -Time*rapid weight gain: -0.06±0.02 $\beta \pm SE$ Body fat % -Rapid weight gain: 1.71±0.41 -Rapid weight gain*in utero tobacco exposure: 0.12±0.72 -Rapid weight gain*maternal overweight: -0.34±0.81 -Time*rapid weight gain: -0.42 ± 0.15 -Time*rapid weight gain*in utero tobacco exposure: 0.78±0.28 -Time*rapid weight gain*maternal overweight: 1.01±0.30 BMI SDS Trajectories -Rapid weight gain: 1.07±0.13 -Time*rapid weight gain: -0.12±0.03 -Time*rapid weight gain*in utero tobacco exposure: 0.09±0.06 -Time*rapid weight gain*maternal overweight: 0.13±0.06	8
Karp, 2012	Weight change, quartiles Birth to 3 months & 4 to 6 months	Mean Log-transformed BMI 4-6 years	(1) Records missing birth data	Model 1: Sex, age, and birth weight	154	Unknown	$\beta \pm SE$ Weight gain birth to 3 months - Unknown, not significant Weight gain 4 months to 6 months - <1.5 kg: 1.200 ± 0.09 - 1.5 to 1.9kg: 1.201 ± 0.08 - 1.9 to 2.5kg: 1.229 ± 0.09	7

First author, year	Exposure Definition Period	Outcome Definition Age at Ascertainment	Exclusions	Control Variables	N ^J	Proportion of Baseline Included in Analysis, %	Result	No. of Quality Criteria Met
Okilero, 2012	Moderate or severe rapid growth 2 days-5 months; 6-11 months; 2 days-11 months; 12-23 months	BMI 95th (obese) age and sex-specific percentile 4-5 years	(1) Lived outside CHC zip code (2) Did not attend CHC for well-child care (3) Did not have pre-K assessment at CHC	Model 1: Ethnicity, cohort, and sex	389	67.3%	- >2.5kg: 1.232 ± 0.08 OR (95% CI) Obese at 4-5 yrs Rapid weight gain 2 days-5months Moderate: 1.12 (0.61,2.10) Severe: 1.17 (0.62,2.21) Rapid weight gain 6-11 months Moderate: 1.03 (0.38,2.78) Severe: 1.33 (0.39,4.54) Rapid weight gain 2 days-11 months Moderate: 1.35 (0.76,2.38) Severe: 1.44 (0.79,2.62) Rapid weight gain 12-23 months Moderate: 2.20 (1.11,4.37) Severe: 4.36 (1.85,10.27) OR (95% CI)Obese: 0.85 (0.10,7.20)	8
Roy, 2015	Velocity to peak BMI 7 to 408 days	BMI 95th (Obese) age and sex-specific percentile 4 years	(1) <6 biologically plausible length and weight measurements in first 408 days of life (2) <2 visits at any of the following age ranges (days): 0-88, 89-224, or 225-408 (3) <37 weeks gestation	Model 1: Sex, race, birth weight, Medicaid status, poverty prevalence, maternal gestational diabetes, smoking, 90th percentile of weight checks, magnitude of peak BMI, timing of peak BMI	635	59.1%	For each g/mo increase in weight from birth to 4 months Full term babies BMI β ± SE: 0.07 ± 0.03 Pre-term babies BMI β ± SE: 0.11 ± 0.03	10
Shankaran, 2010	Weight change per month (g/mo) Birth to 4 months	BMI ³ 9 years	(1) age <18yrs (2) history psychosis or institutionalization for retardation or emotional problems (3) language barriers (4) born at another hospital (5) multiple gestation (6) birthweight <501g (7) gestational age >42wks (8) attending physician judgment of low infant survivability (9) maternal intent to move out of catchment area (10) chromosomal abnormality (11) toxoplasmosis, other agents, rubella, cytomegalovirus, or herpes infection	Model 1: Maternal race, education, and weight at delivery, prenatal exposures (cocaine, alcohol, tobacco, opiate, marijuana), clinic site, sex, birth weight, size for gestational age, factors at 9 years of age (exercise, hours watching TV on school days, calories consumed)	880	69.9%		9
Sietler, 2002	Rate of weight gain (100g/mo) Birth to 6 months	BMI 95th (overweight) age and sex-specific percentile	(1) <37 or >42 weeks gestation (2) Multiple gestation	Model 1: Site, age of child at assessment, birth weight (100g), gestational age (wks), sex, race (Black, white), first-	122 77	63.3%	OR (95% CI) Overweight: 1.38 (1.32,1.44)	8

First author, year	Exposure Definition Period	Outcome Definition Age at Ascertainment	Exclusions	Control Variables	N ¹	Proportion of Baseline Included in Analysis, %	Result	No. of Quality Criteria Met
		7 years		born status, maternal BMI and education				
Stettler, 2003	Rapid weight gain Birth to 4 months	BMI > 30 (obese) & BMI 25 & skin-fold thickness 85th percentile (overweight-overfat) 18.0-22.9 years	(1) <37 or >42 weeks gestation (2) Multiple gestation (3) Follow up between age 18 and 23 years	Model 1: Sex, birth weight, gestational age, firstborn status, birth year, maternal factors (BMI, age, smoking, education) Model 2: Model 1 + Overweight status at age 7 years	115 95	59.8%	OR (95% CI) Overweight: 1.17 (1.11,1.24) OR (95% CI) Obese: 5.22 (1.55,17.6) Overweight-overfat: 6.72 (1.93,23.4)	8
					213	71.0%	OR (95% CI) Obese: 3.39 (0.94,12.2) Overweight-overfat: 4.92 (1.32,18.3)	8

Abbreviations: BMI, Body Mass Index; SD, Standard Deviation; SDS, Sex- and age-independent SD scores; CHC, community health center; SES, Socioeconomic Status; WIC, Women, Infants, and Children;

¹ N analytic sample ;

² Modified version of the NIH Quality Assessment Tool for Observational Cohort and Cross-sectional Studies ([1]; Appendix B);

³ 0.67 increase in weight-for-age z-scores SDs;

⁴ 2000 CDC growth charts;

⁵ 2005 CDC growth charts;

⁶ 1 increase in weight-for-age z-scores SDs;

⁷ Waist for length Z score, moderate defined as 0.67 and <1.0 SD while severe is defined as 1.0 SD

* Does not adjust for SES but sample restricted to only low SES

List of Abbreviations/Acronyms:

SES	Socioeconomic Status
BMI	Body Mass Index

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Figure 1.

Selection of studies for inclusion in a systematic review of early life growth and later obesity by social divisions, 2000-2016

characteristics and social divisions examined in the reviewed studies

Table 1.

First author, year [Reference]	Study name Location	Setting	Sociodemographic Composition	N ¹	Birth Years	Early Life Growth Interval	Obesity-related Outcome Ascertainment
Stettler, 2002 [20]	National Collaborative Perinatal Project <i>Boston, MA; Buffalo, NY; New Orleans, LA; Baltimore, MD; Richmond, VA; Minneapolis, MN; New York, NY; Portland, OR; Philadelphia, PA; Providence, RI; Memphis, TN</i>	University Hospitals	Race/Ethnicity • 49% Black Median Maternal Education (range) • 11 years (6-16 years)	12277	1959-1965	2 d-5 mo 6-11 mo 2 d-11 mo 12-23 mo	4-5 y
Stettler, 2003 [19]	National Collaborative Perinatal Project, Philadelphia Blood Pressure Project <i>Philadelphia, PA</i>	University Hospitals	Race/Ethnicity • 100% Black Median Maternal Education (range) • 11 years (7-12 years)	213	1962-1966	0-4 mo	18.0-22.9 y
Okhiro, 2012 [23]	<i>Rural Hawaii</i>	Community Health Clinic	Race/Ethnicity • 66% Native Hawaiian • 22% Samoan • 12% Filipino 76% of clinic families 200% FPL	389	1981-1983 1986-1988 1991-1993 1996-1998	0-6 mo	7 y
Karaolis-Danckert, 2008 [18]	Multicenter Allergy Study (MAS-90) <i>Germany (Berlin, Munich, Mainz, Dusseldorf, Freiburg)</i>	University Hospitals	Race/Ethnicity ² • Unknown, predominantly Caucasian Parental Education ² • 45.3% <high school • 54.7% High school/GED	370	1990	0-24 mo	2-6 y
Shankaran, 2010 [24]	Maternal Lifestyle Study <i>Providence, RI; Coral Gables, FL; Memphis, TN; Detroit, MI</i>	University Hospitals	Race/Ethnicity • 81% Black • 7% Other Mean Maternal Education (SD) • 11.7 years (2 years)	880	1993-1995	0-4 mo	9 y
Gaskins, 2010 [25]	Maternal Lifestyle Study <i>Providence, RI; Coral Gables, FL; Memphis, TN; Detroit, MI</i>	University Hospitals	Race/Ethnicity • 80% Black • 3% Hispanic Mean SES, Hollingshead ³ (SD) 29.8 (10.1)	312	1993-1995	0-12 mo	11 y
Dennison, 2006 [21]	<i>New York, NY</i>	WIC Clinics	Race/Ethnicity • 31% Hispanic • 19% Non-Hispanic Black Education • 32% <high school • 42% High school/GED 100% low-income	616	1994-1999	0-6 mo	4 y

First author, year [Reference]	Study name Location	Setting	Sociodemographic Composition	N ¹	Birth Years	Early Life Growth Interval	Obesity-related Outcome Ascertainment
Goodell, 2009 [22]	Hartford, CT	Pediatric Primary Care Clinics & Major Hospitals	Race/Ethnicity <ul style="list-style-type: none">• 32% Black• 54% Hispanic• 4% other 85% Medicaid; 100% low-income	203	1995-1995 and 1998-1999	0-4 mo 0-12 mo	24-38 mo
Roy, 2015 [26]	A Study of the Genetic Causes of Complex Pediatric Disorders Philadelphia, PA	University Hospital & Satellite Primary Care Clinic	Race/Ethnicity <ul style="list-style-type: none">• 61% Black• 8% other 45% Medicaid	635	2000-2010	7- 408 d	4 y
Karp, 2012 [27]	Brooklyn, NY	Resident Continuity Clinic	Racial distribution of clinic families <ul style="list-style-type: none">• 81% Black (primarily Afro-Caribbean)• 10% Hispanic• 2% Asian• 2% other 31% of clinic families <100% FPL	154	Not Defined	0-3 mo 4-6 mo	4-6 y

Abbreviations: WIC, Women, Infants, and Children; mo, months; d, days; y, years; SES, Socioeconomic status; FPL, federal poverty level.

¹ N analytic sample

² Information retrieved from an alternative source. Education percentages based on infants in MAS-90 followed up at 1 year [47].

³ Four-factor index of SES, average score of 29 synonymous to social strata of semiskilled workers based [48].

Summary of studies examining early life growth as a predictor of categorical overweight/obesity status, ranked by OR magnitude & direction

Table 2.

First Author, Year [Reference]	Exposure ¹		Outcome Life Stage ²	OR (95% CI) ³	Social divisions			Notable Exclusions	Strengths	Limitations
	Definition	Period			Racial/Ethnic Minorities	Low Income	Low Parental Education			
Goodell, 2009 [22]	Rapid Weight Gain	Birth to 12 mo	Toddler	Overweight: 2.06 (0.86, 4.92) Obesity: 9.24 (3.73, 22.91)	90%	100%	ND		• Examined two periods of early life growth	• Minimal adjustment for confounders • 61.6% missing data • Binary exposure • Two different non-consecutive birth cohorts
Stettler, 2003 [19]	Rapid Weight Gain	Birth to 4 mo	Young Adult	Obesity: 3.39 (0.94, 12.2) Overweight-overfat: 4.92 (1.32, 18.3)	100%	ND	~50%	• Preterm • Post term • Multiple Births	• Analytically examined interaction with education • Additional adjustment for important maternal factors • Only 29% missing data	• Binary exposure
Okhiro, 2012 [23]	Severe Rapid Growth	12 to 23 mo	Preschooler	Obese: 4.36 (1.85, 10.27)	100%	ND	ND		• Enrolled at mandatory pre-k assessment • Examined multiple periods of early life growth • Examined two tiers of rapid growth (moderate and severe)	• Minimal adjustment for confounders • 32.7% missing data • Multiple non-consecutive birth cohorts; 17 year span
Gaskins, 2010 [25]	Weight change per month (g/mo)	Birth to 12 mo	Middle childhood	Overweight: 1.61 (1.07, 2.43) Obesity: 2.69 (1.80, 4.00)	88%	ND	ND	• Mothers that were: • Previously institutionalized • <18 years • Not fluent in English	• Additional adjustment for important maternal factors • Recruited from multiple US regions • Special focus on preterm birth	• 45.9% missing data
Dennison, 2006 [21]	Rate of weight gain (100g/mo)	Birth to 6 mo	Preschooler	At-risk for overweight: 1.41 (1.01, 1.98) Overweight: 1.44 (1.35, 2.21)	88%	ND	ND	• Adopted and foster care children • Very low birth weight (<1800 g)	• Did not exclude preterm, post term or multiple births • Analytically examined interaction with ethnicity	• 55.3% missing data
Stettler, 2002 [20]	Rate of weight gain (100g/mo)	Birth to 6 mo	Middle childhood	Overweight: 1.17 (1.11, 1.24)	49%	ND	~50%	• Preterm • Post term • Multiple Births	• Analytically examined interaction with race • Additional adjustment for important maternal factors • Recruited from multiple US regions	• 40.2% missing data
Roy, 2015 [26]	Velocity to peak (kg m ² /mo)	7 to 408 d	Preschooler	Obesity: 0.85 (0.10, 7.20)	69%	45%	ND	• Preterm • Those with <6 weight measurements in 1 st 408 days of life	• Additional adjustment for important maternal factors • Allowed for early life growth durations unique to each individual • Examined additional components of early life growth: Infancy peak BMI and age at Infancy peak BMI	• 40.9% missing data

OR, Odds Ratio; BMI, Body Mass Index; mo, months; y, years; d, days;; ND, not defined; Ideal adjustment included studies that adjusted for at least sex, birth size, and socioeconomic status

¹ All studies have at least one period of early life growth defined as starting between birth and 7 days of age;

² Outcome childhood stage: Toddlers, 2-3y; Preschoolers, 3-5y; middle childhood, 6-11y; young teens, 12-14y; teenagers, 15-17y; young adult, 18-35y;[49]

³ OR corresponds to the most adjusted model presented. For those studies with multiple examined associations correspond to the greatest OR

⁴ Duration of early-life growth is based on each individuals age at peak BMI with mean±SD at 8.6 ± 1.4 months

Summary of 3 studies with continuous outcomes, ranked by effect size of β

Table 3.

First Author, Year [Reference]	Exposure ¹		Outcome		β (95% CI) ³	Social divisions			Notable Exclusions	Strengths	Limitations
	Definition	Period	Definition	Life Stage ²		Racial/Ethnic Minorities	Low Income	Low Parental Education			
Karp, 2012 [27]	Weight Change, 4th quartile <2.5 kg	4 to 6 mo	$Log(\widehat{BMI})$	Preschooler	1.23 (1.07,1.39)	95%	ND	ND		• Clinic serves a largely inner city immigrant population	• Minimal adjustment for confounders • BMI outcome not standardized to sex and age • Missing data unknown
Karaolis-Danckert, 2008 [18]	Rapid Weight Gain	Birth to 24 mo	BMI SDS trajectory	Toddler - Preschooler	0.95 ⁴	ND, low	ND	45-30%	• Preterm • Post term • Multiple Births • Low birth weight	• Analytically examined interactions with education factors • Additional examination of effect modification and adjustment for important maternal factors • Only 28% missing data	• Birth cohort and sociodemographics of target population undefined • Binary exposure
			<i>Mother not overweight, not smoker</i>								
			<i>Mother not overweight, smoker</i>								
			<i>Mother overweight, not smoker</i>								
Shankaran, 2010 [24]	Weight change per month (g/mo)	Birth to 4 BMI Middle	BMI	Mother overweight, smoker	1.16 ⁴	88%	ND	ND	Mothers that were: • Institutionalized • Pre-term • <18 years • Recruited from multiple US regions • Not fluent in English	• Additional adjustment for important maternal factors • Did not adjust for important confounders • Recruited from multiple US regions • Large prospective sample	• 31% missing data
				Born preterm	0.11 (0.05,0.17)						
				Born at term	0.07 (0.01, 0.13)						

BMI, Body Mass Index; SDS: Standard Deviation Score; mo, months; y, years; d, days; ND, not defined; Ideal adjustment included studies that adjusted for at least sex, birth size, and socioeconomic status

¹ All studies have at least one period of early life growth defined as starting between birth and 7 days of age;

² Outcome childhood stage: Toddlers, 2-3y; Preschoolers, 3-5y; middle childhood, 6-11y; young teens, 12-14y; teenagers, 15-17y; young adult, 18-35y;[49]

³ β corresponds to the most adjusted model presented. For those studies with multiple examined associations correspond to the greatest β

⁴ Conditional estimates calculated using adjusted coefficients in Karaolis-Danckert (2008), Table 4[