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Connecting the Dots in Childhood Obesity Disparities: A Review of Growth Patterns from Birth to Pre-Adolescence

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Abstract

In this review, we considered how disparities in obesity emerge between birth, when socially disadvantaged infants tend to be small, and later in childhood, when socially disadvantaged groups have high risk of obesity. We reviewed epidemiologic evidence of socioeconomic and racial/ethnic differences in growth from infancy to pre-adolescence. Minority race/ethnicity and lower socioeconomic status was associated with rapid weight gain in infancy but not in older age groups, and social differences in linear growth and relative weight were unclear. Infant feeding practices was the most consistent mediator of social disparities in growth, but mediation analysis was uncommon and other factors have only begun to be explored. Complex life course processes challenge the field of social epidemiology to develop innovative study designs and analytic techniques with which to pose and test challenging yet impactful research questions about how obesity disparities evolve throughout childhood.

Keywords

Obesity; Socioeconomic factors; Ethnic groups; infant; child; longitudinal

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Compliance with Ethics Guidelines

Conflict of Interest

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Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

INTRODUCTION

Health disparities, patterned by social strata, are among the most consistently observed and persistent epidemiologic finding across the life course and across disease outcomes [1, 2]. Disparities often manifest as excess risk of low birth weight at the start of life [3, 4], excess obesity in childhood [5, 6], and excess diabetes and cardiovascular disease risk by adulthood [7]. Thus, disparities are not static, but follow a developmental trajectory [8]. In this paper, we consider how disparities in obesity evolve throughout childhood. We consider three aspects of this research, summarized in the theoretical framework (Figure 1) and described in the following sections.

Social differences in longitudinal childhood growth patterns

Adverse fetal development – indicated by neonatal health metrics – exhibits clear social patterning (Figure 1; Pathway A). Compared to white women in the U.S., African American women carry a persistent excess burden of preterm and low birth weight deliveries [3, 4], while Hispanic women have higher risk of macrosomic deliveries [9]. In adolescence (Pathway B), African American and Hispanic children have higher obesity risk [10, 11]. Socioeconomic disparities are similar: children in low-income families have elevated risk of small size at birth (Pathway A) and obesity in childhood (Pathway B) [12].

These patterns imply meaningful subgroup differences in growth between birth and adolescence, in which body size may increase rapidly in early childhood or remain elevated from birth. However, evidence of how disparities in body size change with increasing age is derived primarily from cross-sectional studies compared across age groups. In this paper, we review evidence of socioeconomic and racial/ethnic differences in longitudinal growth patterns from birth to pre-adolescence (Figure 1, Pathway C). We focus on this period of growth because it lies between two life stages in which disparities are extensively documented but is itself underexplored.

Modifiable factors underlying social differences in growth

Next, we evaluate the state of knowledge about factors that drive social differences in growth patterns. We draw from Vanderweele and Robinson's mediation framework for epidemiologic analysis that examines race/ethnicity as an exposure, in which covariates are included in order to investigate factors that underlie racial/ethnic differences in health [13]. We theorize that social differences in childhood growth are mediated largely by inequitable distribution of environmental, behavioral, and health drivers of growth at each life stage (Figure 1, dotted lines). For example, African American women are more likely to experience greater psychosocial stress due to racial discrimination [14], face barriers to healthy diet and physical activity [15, 16], and develop obesity and related disease [17]. Each of these factors is associated with adverse fetal growth, often indicated by low or high birth weight. Therefore, social patterning of these maternal risk factors may mediate social disparities in birth outcomes (Pathway A). Likewise, social differences in obesity in adolescence or beyond (Pathway B) are theorized to arise from the inequitable distribution of healthy neighborhood, school, and family resources needed for healthy lifestyles in childhood [18].

Factors that contribute to social differences in early life growth (Pathway C; birth to pre-adolescence) are less studied, but likely involve adverse prenatal exposures and inadequate resources needed for healthy breastfeeding and infant feeding [19]. In this review, we explore evidence of the role of maternal and infant factors in social differences in early life growth.

Interdependence of growth across critical life stages

Lastly, in light of what we now understand about early life programming of later life health and disease [20, 21], we review the literature with an eye to the complex set of biological processes that link prenatal growth, early life growth, and later growth and obesity. Fetal development involves myriad processes that can “program” the offspring with elevated susceptibility to obesity and disease later in childhood and in adulthood (Figure 1, Pathway D1) [22]. Programmed characteristics, particularly elevated appetite, can promote accelerated growth in early life (Pathway D2) [23]. Beyond its contribution to accumulating lean and fat mass, early life growth interacts with fetal programming characteristics in driving obesity and disease risk (Pathway D3) [24].

Understanding disparities in later-childhood and adult health requires understanding the impact of social differences across these early developmental periods. In this review, we consider how prenatal, early life, and later childhood factors are addressed in epidemiologic study design.

Objectives

The objectives of this paper were threefold: (1) review the evidence of socioeconomic and racial/ethnic differences in growth from infancy to pre-adolescence in Western countries, (2) assess the state of knowledge about modifiable factors that drive sociodemographic differences in growth patterns, and (3) discuss how epidemiologic studies address interdependence among prenatal, early life, and later obesity. Broadly, we sought to identify knowledge gaps, characterize methodological challenges, and provide recommendations for understanding the key drivers of social differences in childhood growth patterns.

Literature review

We conducted a PubMed search for published studies that reported race/ethnic or socioeconomic (income or education) differences in growth in weight, stature, or relative weight (e.g., Body Mass Index [BMI]). Details about our literature search strategy are described in Appendix A. Briefly, we included studies that examined at least two weight, stature, or relative weight measures from birth to 12 years, were conducted in Western study populations, and were published or available ahead of print between January 1, 2000 and September 15, 2015. This article does not contain any studies with human or animal subjects performed by any of the authors.

Thirty five papers met inclusion criteria. Twenty one studies were conducted in Europe, representing 15 distinct cohorts (Appendix Table S1A). Twelve were conducted in North America and 2 in Australia (Appendix Table S1B).

1. SOCIAL DIFFERENCES IN CHILDHOOD GROWTH: RESULTS AND GAPS

Methods used to model growth over time

The reviewed studies examined growth in weight, length, or relative weight using three broad types of analytical strategies.

Difference variables—Thirteen studies examined growth by modeling the difference in weight, length, or relative weight between two time points as a function of SES, race/ethnicity, or other predictors of growth [25-36]. These measures are indicated as *outcome (age range)* [e.g., *weight z-score (birth to 12m)*] in Tables 1-2 and Appendix Tables S2-3.

Several studies categorized the difference variable into an indicator of rapid growth using Ong and Loos's definition [37], either dichotomizing (>0.67 vs. 0.67 SD) [27, 35] or trichotomizing (<-0.67 , -0.67 to 0.67 , >0.67 SD) [28, 30]; or top quartile of weight gain [26, 33]. These definitions were applied to periods of varying length (range: 3 months to 2 years) and ages (range: birth-3 months, 3-5 years).

Hierarchical longitudinal modeling—Twelve studies used longitudinal models to study weight, length, or relative weight over three or more time points [38-49]. These studies typically used mixed effects models, where baseline level and growth velocity may vary across children. In the absence of interactions between time and social division, these studies [38, 40, 42, 45-47, 49] provide information about social differences in BMI, weight, or length averaged over discrete time periods. Social differences in growth velocity can be inferred by comparing social differences in body size across age group; for example, SES differentials in shorter height observed in infancy but diminishing with age suggest that lower SES children experienced faster linear growth after infancy. These results are indicated as *outcome (age range)* in Tables 1-2 and Appendix Tables S2-3.

If present [39, 41, 44, 48], the age*social division interaction coefficient indicates the degree to which growth over time varies by social division. Results from these studies distinguish between social differences in body size that results from maintenance of, for example, excess growth attained at birth or in early life, versus accelerated or decelerated growth. We represent these findings as *Rate of change (outcome, age range)* (Table 2; Appendix Tables S2-3).

Two studies used mixed effects models to characterize adiposity transitions [50, 51] that are strongly predictive of obesity later in life [52, 53]: timing, velocity, and BMI at infancy peak (typically at 7 months of age) or adiposity rebound (typically at 5-7 years) for each child. Other studies investigated time to overweight onset using survival analysis [54] or constructed growth reference curves using the LMST method [55].

Latent growth classes—Six studies used latent growth curve classification methods to examine discrete patterns of change in BMI or overweight/obesity throughout childhood. This approach identifies groups of children with similar baseline body size and growth over time. We present these findings in the "Relative weight" subsection below.

Results: social differences in childhood growth

In this section, we report the most minimally adjusted associations available because we were interested in the total estimate effects of SES or race/ethnicity on growth, not adjusted for mediators such as breastfeeding. We compare results with different sets of covariates within a mediation framework in Section 2.

Weight—From birth to 12 months, minority race/ethnicity and lower SES was typically associated with faster weight gain (Table 1; Appendix Table S2) indicated by more positive weight differences [25, 30, 34–36], higher risk of rapid growth [28, 33], or diminishment of lower weight in lower SES or minorities with increasing age [45]. However, results were mixed [29, 36, 39, 42, 49], especially with regard to specific race/ethnic groups [28, 30, 45]. Race/ethnicity and SES were less consistently associated with categorical definitions of rapid infant weight gain [28, 30, 33, 35]. After one year of age, social differences in faster weight gain were mixed [26–28, 39, 55] (Appendix Table S2). Seed et al found black race differentials in faster weight gain from birth to 2–3 years only in children born preterm [31].

Length and height—With regard to stature, racial/ethnic minority and lower SES infants tended to be shorter at birth [39, 41, 47]. From birth to 12 months, minority race/ethnicity was associated with faster linear growth [25, 39, 42, 44, 45], with heterogeneity across specific race/ethnic group [45]. Evidence was equivocal for low SES, which was associated with faster [47] or similar [29, 41] linear growth in the first year (Appendix Table S3). Associations between low SES or minority race/ethnicity and faster linear growth were more common after the first three months of life [41, 45] and in periods spanning beyond one year [47, 48, 55], in some cases greatly reducing or overcoming social differences in stature by the end of the study period [46, 47, 49]. However, in other studies, minority ethnicity [39] or lower SES [40, 41, 44] was associated with slower linear growth later in childhood.

Relative weight (weight-for-length, BMI, overweight or obesity status)—In the first year of life, relative weight change was unrelated to lower SES (Table 2) [29] and inconsistently associated with race/ethnicity [25, 29, 42]. Beyond 12 months, lower SES [34, 38, 48] but not race [38] was associated with faster relative weight change beyond 12 months though with variation in timing [49] and by sex and ethnic subgroup [48, 55]. Notably, Semmler and colleagues provide evidence that lower SES is associated with faster increase in BMI z-score from 27 months to 14 years of age, but only if the parent was obese [32]. The authors posit that SES is a stronger driver of obesity among children with genetic predisposition to obesity.

Studies characterizing growth throughout childhood suggest that social disparities in overweight occurs by 2–4 years of age and worsens with increasing age (data not shown). Children with lower SES [56–59] or black race [59] tended to exhibit “persistent overweight” from 2–4 years to adolescence. Pryor et al did not identify a persistent overweight trajectory among children followed from 6 months, but children from families with low SES were at higher risk of following a “high rising” BMI trajectory, which diverged at around 3 years [60]. Children overweight in infancy were less likely to resolve their overweight status as they aged into pre-adolescents if they were from lower SES or

ethnic minority families [61]. Lower SES [57] and black [59] children were more likely to exhibit late onset overweight or increasing overweight severity [56] than stable normal weight trajectories.

Evidence from Jones-Smith et al suggests heterogeneity in SES disparities in overweight/obesity prevalence, which emerged at approximately 9 months of age and magnified over time, but only among whites, Hispanics, and Asians [43] (data not shown). No SES-related differences in overweight/obesity prevalence were observed in African Americans and American Indians. In a smaller study population followed from 5 to 18 years of age, obesity onset occurred at younger ages in blacks than whites, with important sex differences [54]: in whites, obesity onset was earlier in girls than in boys; in blacks, the reverse was found.

Racial differences were also observed with regard to infancy BMI peak and adiposity rebound. Roy et al showed that African American infants reached infancy BMI peak 0.4 months earlier than white infants, at which time they were 0.5 BMI units heavier [50]. In contrast, Wen et al observed similar infancy BMI peak characteristics in African American and white children [51]. However, African American children experienced adiposity rebound at younger ages; they were not heavier at adiposity rebound, but they gained 0.9 greater BMI units by 18 years of age [51].

Future research directions for understanding social differences in childhood growth

Research in diverse U.S. populations—Over half of studies were conducted in Europe (Appendix Tables S1A-B). In the U.S., the absence of universal health coverage [62], inequitable social supports, and maternity and paternity leave policies [63] likely exacerbate socioeconomic disparities occurring in the perinatal period and early childhood. Relevant U.S. studies draw from nationally representative cohorts [43, 58, 59], but often rely on health care system data [38, 51, 56] or research-based birth cohorts [33, 50], which often have limited sociodemographic diversity. More research in representative and low-income U.S. populations is needed to understand and resolve social disparities in early childhood growth within a U.S. context.

Catch-down growth—In general, the reviewed studies focused on social predictors of accelerated growth in infancy and early childhood, typically following small size at birth. However, large size at birth (macrosomia) is also a strong predictor of later obesity [64]. Crume et al described a distinct growth pattern in offspring prenatally exposed to GDM: these children were born heavier, grew slower in the first 10 months, and aligned with non-GDM growth curves from 10-26 months of age [38], but subsequently exhibited accelerated growth from 27 months to 14 years of age. May et al found that low income children with high birth weight ($z\text{-score} > 1$) experienced slower weight gain and slower linear growth in the first three months than children with normal birth weight [29]. More investigation of optimal growth among infants born large is needed in diverse study populations.

Growth measures—Categorical definitions of weight gain were less consistently associated with social predictors, suggesting that non-rapid weight gain may comprise heterogeneous patterns that include catch-down growth. Further, future studies that delineate

gains in lean versus fat mass, which varies from the time of birth [65, 66] and across race/ethnicity [67], may help clarify inconsistent findings with regard to relative weight.

Summary

Existing research provides strong evidence that lower SES and racial minority groups tend to gain weight more rapidly in the first year of life. In contrast, social differences in relative weight and linear growth are best characterized as vastly heterogeneous with respect to sex, age groups, specific race/ethnic groups, and interactions between race/ethnicity and SES. The challenge for social epidemiologists is to leverage increasingly available longitudinal data and analysis methods to understand the vast array of growth patterns observed in diverse study populations and specific subpopulations.

2. MODIFIABLE FACTORS UNDERLYING SOCIAL DIFFERENCES IN GROWTH: RESULTS AND GAPS

To reduce disparities in childhood obesity, we need greater understanding of the causal processes through which social differences in growth are constructed and, by extension, can be deconstructed. Here, we consider studies that examined potential drivers of social disparities in growth.

Methods used to examine mediators of social differences in growth

Studies that examined maternal or early life factors theorized to underlie social differences in growth patterns used a wide range of approaches (Table 3; Appendix Table S4). Two studies used path mediation analysis [34, 58], a structural equation modeling approach in which multiple pathways are estimated simultaneously [68, 69]. The models simultaneously estimated effects of (a) SES or race/ethnicity on growth (Figure 1, Pathway C), (b) of SES or race/ethnicity on the mediator (e.g., infant feeding), and (c) estimated effects of the mediator on growth.

Seven studies tested mediation by fitting successive models with different sets of hypothesized mediators [25, 28, 35, 40, 47, 55, 57]; two studies also quantified attenuation after adjusting for mediators [28, 35] (Table 3). These studies inform the extent to which social differences in growth were observed after controlling for maternal or early life factors. Three studies fit similar successive models but did not conceptualize the adjustment variables as mediators of the association between social division and growth [33, 36, 39]. One study tested hypotheses related to interactive mechanisms underlying social differences in growth [32].

Most studies reported estimates from models simultaneously adjusted for numerous risk factors [26, 27, 30, 31, 43, 44, 46, 50, 51, 56, 59-61, 70], or conducted minimally adjusted analysis [41, 42, 48, 49]. In three studies, the primary study aim did not align with our research question [29, 38, 45], but provided findings that informed our research question of social differences of growth.

Results: mediators of social differences in childhood growth

The most consistent finding was that breastfeeding attenuated social differences in growth in weight or weight for length [25, 28, 34, 35, 47], but not in linear growth [25, 40, 47]. Layte et al found that infant feeding practices attenuated class differences in weight gain from 9 months to 3 years, but not from birth to 9 months [28]. No other studies examined breastfeeding mediation within specific time periods.

Evidence for parental characteristics underlying disparities in growth was mixed. In their path mediation model, Van den Berg et al found partial mediation of social differences in weight or weight-for-length gain by maternal BMI, prenatal smoking, and maternal age [34]. Other studies found no mediation by maternal pre-pregnancy weight status [25, 28, 35, 40, 55], prenatal smoking [28, 35, 40, 55], or maternal age [40, 55]. Maternal and paternal height, a marker for genetic potential or nutrition status, attenuated social differences in growth in two studies [40, 47], though inconsistently by age and in other studies [55]. With the exception of Van den Berg et al's path analysis, conclusions about mediation of social differences in growth did not vary by mediation analysis approach.

Other factors examined as mediators included daycare attendance [47], parenting practices [58], maternal depression [58] and prenatal alcohol consumption [28], and paternal smoking and weight status [40]. While worthy of study, we do not summarize findings due to sparse data.

Future directions for understanding factors underlying social differences in childhood growth

Mediation framework and analysis—Only two of the reviewed studies conducted systematic evaluation of associations between social factors and growth, between social factors and mediators, and between mediators and growth. Originating in psychology [71], with recent developments in epidemiology [13, 72], greater application of mediation frameworks and analysis are needed to identify factors that are both socially patterned and important for infant childhood growth.

Improved measurement of mediators—Infant feeding was the most promising factor mediating social disparities in growth. However, infant feeding measures do not typically capture critical details such as quantity or type of formula or complimentary foods and quality of breastmilk [25]. Thus, improved infant feeding measures are needed to fully understand how infant feeding contributes to social differences in infant growth.

Several studies examine parental characteristics as gross proxies for environmental or biological processes. For example, maternal or paternal BMI has been used to indicate genetic predisposition [32, 38] or environmental factors [57]; it could also be a proxy for fetal programming [73]. Incorporation of measures that directly measure the factor of interest – genetic markers [74], family environment characteristics [75], epigenetic markers of fetal programming [76] – are needed to identify factors that should be targeted in order to reduce obesity disparities.

Consideration of multi-level mediators—Contextual factors that drive maternal and infant behaviors are often theorized, but their contribution to social differences in infant growth has only begun to be explored, largely with regard to neighborhood socioeconomic indicators [57]. Between- and within-country variation in workplace supports for breastfeeding and breastmilk expression may also contribute to social variation in infant feeding practices, but their impacts on infant growth has not been studied. Further study of other environmental drivers of adverse growth, such as chemical pollutants [77], food and physical activity environments [78, 79], and social influences [75, 80] would advance knowledge about modifiable factors that contribute to social differences in childhood growth.

Summary

Strategies to reduce social disparities in childhood growth remain unclear. Healthy infant feeding is a promising approach, but there is much to be learned about specific aspects of infant feeding that can reduce disparities. Research on other modifiable factors has just begun. Success in defining actionable steps needed to reduce disparities requires further articulation of causal mediation frameworks representing processes that produce social disparities in growth, designing social epidemiologic studies to align with and test those frameworks, and further investigation of environmental and policy drivers of mediators.

3. INTERDEPENDENCE OF GROWTH ACROSS CRITICAL LIFE STAGES

To understand the extent to which reducing social disparities in early life growth will reduce disparities later in life, we must understand how pathways from prenatal, infant, and childhood growth work together, and over what periods of time (Figure 1). In this section, we consider the methodological challenge of appropriately recognizing interdependence of processes across life stages in social epidemiologic life course research. Empirical evidence about this topic is scant, so we focus on methodological issues and future research needs for understanding processes that yield disparities in childhood obesity.

Controlling for birth weight

To recognize the independent effects of fetal and early life growth on childhood obesity (Figure 1; Pathways D2 and D3), most studies adjusted for birth weight or gestational age at birth (Table 3; Appendix Table S4). Other studies exclude children born low birth weight, small for gestational age, preterm, or with serious health conditions because these subgroups exhibit distinct growth patterns.

However, the interpretation becomes complex when studying prenatal social or other predictors of early life growth. Such factors are often measured during pregnancy, or unlikely to differ from conditions during pregnancy. Fetal development (indicated by birth weight) occurs after these exposures and is thus conceptualized as a partial mediator of the association between social division and early life growth. Therefore, if an association between social division and early childhood growth is adjusted for fetal development, it reflects social differences in early childhood growth that are attributable to factors that did not impact fetal development. Social factors that impact early childhood growth but not

prenatal development are likely to be rare, perhaps including factors related to infant feeding but not maternal health.

Yet the “total” social disparity in early life growth patterns includes the portion that occurs through fetal development processes. Adjusting for birth weight can thus comprise over adjustment, in which one estimates the effect of a social factor on postnatal growth while controlling for the effect of the same factor on birth weight [81, 82]. As discussed in the next section, innovative methodology is needed to improve understanding of the full pathway from social disadvantage to childhood and adolescent obesity.

Growth trajectories and the next phase of hypotheses

Most longitudinal models compare growth across individuals, *within* specified age ranges. In contrast, studies that use latent growth curves [56-61] or characterize adiposity transitions [50, 51] model sequencing and timing of childhood growth for each individual as a continuous series of events. These methods are useful for hypothesis generation about factors driving different timing or magnitude of growth. Yet testing hypotheses about drivers of growth patterns require even more sophisticated analytic methods that can model not only mediating and interactive relationships among exposures and growth during discrete time periods, but also the sequential pathways among time periods. Path analysis, as described in Section 2, can accommodate these types of complex pathways [83]. Other possible approaches include dynamic systems models [84] or simulation methods [85].

Impacts on later life health

Returning to our ultimate goal of understanding how to reduce disparities that manifest throughout the life course, quantification of the contribution of social divisions and socially patterned risk factors to later life health is needed. Large, diverse study populations followed from birth through adulthood would inform these research questions but pose practical challenges in terms of cost and duration. These complex life course issues challenge the field of social epidemiology to develop innovative study designs and analytic techniques, such as synthetic cohorts and other simulation methods [85].

Summary

This area of inquiry is rich and offers tremendous potential for revealing not only how social disparities evolve through childhood and beyond, but how to mitigate development of disparities. Realizing that potential requires methodological advancements in social epidemiology that overcome challenges posed by interactive, long-term processes, heterogeneous responses, and data constraints. Moreover, rather than constraining our research questions to what is possible with traditional methods, social epidemiology must seek and create novel methods to answer complex yet impactful research questions.

CONCLUSION

Racial and ethnic minorities and children in families with lower SES exhibit different growth patterns in infancy and early childhood. Rapid weight gain in infancy was the most consistent disparity. Drivers of these disparities may include adverse infant feeding

practices, but other potential drivers have only begun to be explored. Future research needs to be explicitly tethered to a conceptual framework for the hypothesized underlying processes, align study design with these frameworks, and test alternative frameworks. Any hope for effective public health interventions designed to slow the trends of increasing obesity disparity will require a broader perspective as to the causes and consequences of social inequalities on health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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APPENDIX A: Literature search methods

We searched for publications in PubMed that reported race/ethnic or socioeconomic (income or education) differences in growth in weight, length or height, relative weight (Body Mass Index [BMI] or weight-for-length z-scores), or adiposity. We applied search terms capturing concepts of (a) growth ("weight gain" or [("body mass index" or "weight" or "height" or "adiposity") and (growth or trajectory)]), (b) early life (Infant or infancy or "early life" or neonatal or newborn) and (c) social divisions (Income or race or ethnicity or ethnic or socioeconomic or "social class"). We conducted our search in September 2015, limiting to 3,646 papers published or published ahead of print as of September 15, 2015, dating back to January 1, 2000. In addition to keyword searches, we examined references from relevant papers.

We include peer reviewed, published manuscripts that report social (race/ethnicity, income, education, or SES index or proxy measure) predictors of growth in weight, height or length, or BMI or other relative weight measure (at least two time points from birth and pre-adolescence [approximated as 12 years of age]). In order to focus on social disparities in early life growth that occurs within obesogenic environments and that are generalizable to Western societies, we exclude studies conducted in developing countries or Asia, or that focus on stunting or special clinical populations (e.g., HIV, congenital abnormalities). While obesity and obesogenic environments are becoming more prevalent in the developing world, obesity etiology is distinct in countries in which Western diet has recently become prevalent. We also exclude papers that focus on a research question other than social predictors of early life growth and only report our associations of interest in descriptive tables. While the papers presented do not represent the universe of relevant studies, we believe they are representative of the literature on social differences in early life growth as of September 2015.

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Papers of importance

- Van Den Berg 2013: Example mediation analysis using path analysis
- Layte 2014: Example mediation analysis using successive models and difference variable outcomes.
- Roy 2015: Example characterization of BMI infancy peak and adiposity rebound
- Fairly 2013: Example use of mixed effects models with time interactions
- Jansen 2013: Example of latent growth curve analysis

Figure 1.
Conceptual framework for the progression of social disparities in growth from birth to childhood

Table 1Studies reporting social differences in weight gain from birth-12 months of age¹

	Weight gain or weight ²	Rapid weight gain ²
May 2013 (US)	<i>weight z-score (0-3m)</i>	-NA-
Hispanic (Ref: non-Hispanic)	○	
Income (continuous)	○	
Wijlaars 2011 (UK)	<i>SDS weight (0-3m)</i>	<i>Rapid growth (0-3m)</i>
Higher SES	↓	↓
Bulk-Bunschoten 2002 (Netherlands)	<i>weight z-score (0-4m)</i>	-NA-
Education (Ref: university)		
Primary plus 5 years	○	
Primary plus 4 years	↑	
Primary education or less	↑	
Dutch language (ref: other)	↓	
Reeske, 2013 (Germany)	<i>weight z-score (0-6m)</i>	<i>Rapid growth (0-6m)</i>
Migrant background (Ref: German)		
Turkey	↑	○
Eastern European	↑	○
Other	↑	○
SES-Migrant background (Ref: High SES German)		
Low SES	○ (all backgrounds)	○ (all backgrounds)
Middle SES	↑ (Turkish, Other only)	○ (all backgrounds) ³
High SES	↑ (Eastern European)	
Taveras 2010 (US)	-NA-	<i>Rapid growth (0-6m)</i>
Race (Ref: non-Hispanic white)		
African American		↑
Hispanic		○
De Hoog 2011 (Netherlands)	<i>SDS weight (4w-6m)</i>	-NA-
Ethnicity (Ref: Dutch)		
African	↑	
Turkish	↑	
Moroccan	↑	
Others	○	
Leffelaar, 2010	<i>Weight SDS (4w, 13w, 26w, 39w, 52w)</i>	-NA-
Ethnicity (Ref: Dutch)		
Surinamese	↓, ↓, ○, ○, ○	
Turkish	↑, ↑, ↑, ↑, ↑	

	Weight gain or weight ²	Rapid weight gain ²
Moroccan	○, ○, ↑, ↑, ↑	
Other non-Western	○, ○, ○, ○, ○	
Other Western	○, ○, ○, ○, ○	
<hr/>		
Van Den Berg 2013 (Netherlands)	weight z-score (birth-12m)	
Lower education (Ref: Higher)	↑	
<hr/>		
Wright 2004 (UK)	Weight SDS (birth, 6m, 12m)	
Decreasing paternal social class	○, ○, ○	

Abbreviations: NA, Not applicable, study did not examine this outcome; Ref, Referent category; SDS, Standard Deviation Score; Units of time: m, months; y, years

↑, ○, ↓: social division exhibited greater, similar, or lesser (respectively) growth or larger body size than referent

¹Studies that also report weight gain after 12 months are presented in Appendix Table S2. Italics indicate the growth metric and age range examined [*weight* is a difference variable, $weight_t - weight_{(t-1)}$]. Papers are listed from youngest to oldest study populations.

²Weight gain represents continuous change in weight; Rapid weight gain is a categorized variable

³Middle and High SES were combined in analysis of Rapid Growth outcome

Table 2

Social differences in growth between discrete time points (ages): BMI or weight-for-length (WFL)[†]

	Approximate age range				
	Birth-6mos	6-12mos	12-24mos	2-4 yr	5+yr
May 2013 (US)	<i>WFL z-score (0-3m)</i>				
Hispanic (Ref: non-Hispanic)	↑				
Higher income	○				
De Hoog 2011 (Netherlands)	<i>WFL SDS (4w-6m)</i>				
Ethnicity (Ref: Dutch)					
African	○				
Turkish	○				
Moroccan	○				
Others	○				
Johnson 2012 (UK)	<i>BMI z-score (10d-15m)</i>				
Pakistani (Ref: white)	↓ ² (at each month)				
Hof 2011 (Netherlands)	<i>BMI for age reference values (0-3y)</i>				
Race/ethnicity (Ref: Dutch)					
Surinamese	↓ (males, <40w; females, <50w)				
Turkish	↓ (males, diverge at ≈40w; females, diverge at ≈100w)				
Moroccan	↑ (all ages, strongest in females)				
	↑ (all ages, strongest in females)				
Murasko 2014 (US)	<i>Rate of change in BMI (birth-5y)</i>				
Increasing income	↑ (strongest in black males, all females)				
Van Den Berg 2013 (Netherlands)	<i>WFL z-score (1-5y)</i>				
Lower education (Ref: Higher)	↑				
Crume 2011 (US)	<i>BMI (0-26m, spline at 11m)</i>				
Race/ethnicity (Ref: White)					<i>BMI (27m-14y)</i>

	Approximate age range				
	Birth-6mos	6-12mos	12-24mos	2-4 yr	5+yr
Hispanic		○			○
African American		○			○
Lower income ³		↑			○
Education (Ref: >high school)		○			○
Semmler 2009 (UK)					
<i>BMI z-score (4-11y)</i>					
Lower SES (Ref: Higher)				○ if lean parent, ↑ if obese parent	
Wright 2004 (UK)					
<i>BMI SDS (9y, 13y, adult)</i>					
Decreasing paternal social class					○, ○, ↑

Abbreviations: Ref, Referent category; SDS, Standard Deviation Score; Units of time: m, months; y, years

↑, ○, ↓: social division exhibited greater, similar, or lesser (respectively) growth or larger body size than referent

¹Italics indicate the growth metric and age range examined. *outcome is a difference variable, weight_t – weight_(t-1)* Outcome indicates social differences in the anthropometric measure over the age range. *Rate of change* indicates comparison of velocity of growth during the age range. Papers are listed from youngest to oldest study populations.

²Sex-stratified analysis; results similar for males and females

³For comparability, converted published result to represent growth associated with lower SES

Approaches used to test mediators of association between SES or race/ethnicity and growth⁷

Table 3

Reference	Primary exposure(s)	Maternal, paternal RFs	Birth	Infant/Childhood	Mediation approach	Mediation results
Lane 2013	Income	+Maternal depression ~Prenatal smoking	Exclude: >singleton, serious illness	+Positive parenting +Parenting style	Path analysis mediation model	Partial mediation by permissive parenting style
Van Den Berg 2013	Maternal education	+Prenatal smoking +Maternal pre-preg BMI +Maternal age Exclude: non-Dutch	+BW ~GA ~Sex	+Breastfeeding duration +Solids (age at introduction)	Path analysis mediation model	Weight gain: partially mediated by prenatal smoking, breastfeeding, maternal age Weight-for-length gain: partially mediated by maternal BMI
De Hoog 2011	Ethnicity	~Maternal age, parity ~Maternal, paternal height ~Maternal BMI, hypertension, diabetes ~Prenatal smoking ~Maternal education ~Living with partner	~BW, GA Exclude: >singleton, preterm, SGA	+Breastfeeding duration +Formula feeding +Complementary food (age at initiation)	Successive models w/mediators	Similar associations in models containing infant feeding practices Slight attenuation in African descent-weight, length SDS association by infant feeding
Galobardes 2012	Maternal education	+Maternal age +Maternal height, BMI +Number of children +Prenatal smoking +Maternal diet +Paternal height, BMI +Paternal smoking	~sex +GA Exclude: >singleton, fetal loss (<27 days post birth); preterm	~age +Breastfeeding	Successive models w/mediators	Attenuation in models containing maternal height, paternal height, mid-parental height. Other potential explanatory characteristics did not impact education-height association.
Hof 2011	Immigrant status	+Maternal height, weight +Maternal age +Maternal smoking	Stratified by gender Exclude: >singleton, preterm		Successive models w/mediators	Sex-ethnic differences in growth curves were similar after adjustment for mediators
Jansen 2013	SES	+Parental BMI	+BW ~Indigenous status	~non-English speaking background	Successive models w/mediators	Slight attenuation in models containing mediators

Reference	Primary exposure(s)	Maternal, paternal RFs	Birth	Infant/Childhood	Mediation approach	Mediation results
Layte 2014	Social class	~Maternal age ~Gestational weight gain +Prenatal smoking, alcohol +Maternal weight status	~Sex ~GA, BW ~Birth order ~Multiple birth	+Breastfeeding, weaning + Dietary quality + Television watching	Successive models w/mediators ²	Rapid growth (birth-9m): no attenuation Rapid growth (9m-3y): attenuation by infant nutrition
Silva 2012	Maternal education	+Prenatal smoking +Maternal, paternal height Exclude: non-Dutch	+BW, GA Exclude: >singleton, >1 st birth	+Breastfeeding	Successive models w/mediators	Mixed: little attenuation by breastfeeding, strengthened association with maternal, Paternal RFs; varied by child age
Wijlaars 2011	SES	+Parental BMI +Prenatal smoking	~Gender ~GA Exclude: non-twins	~3 month age +Breastfeeding	Successive models w/mediators ²	Strong attenuation by breastfeeding

Abbreviations: BMI, Body Mass Index; BW, birth weight; GA, gestational age at birth; GDM, Gestational Diabetes

¹ Among 35 studies in literature review. Studies in review that did not conduct mediation analysis are reported in Appendix Table S4. Papers are grouped by mediation analysis approach, then alphabetically by author.

² Quantified attenuation of SES coefficient after including mediators in the model.

+Explicitly conceptualized as mediators ~Control variables