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The prevalence of rapid weight gain in infancy differs by the growth reference and age interval used for evaluation

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Abstract

Infant rapid weight gain (RWG) may predict subsequent obesity, but there are inconsistencies in the growth references and age intervals used for assessment. This study evaluated whether the prevalence of RWG (an increase of > 0.67 in weight-for-age z-score) differed by growth reference (2006 WHO standards vs. 2000 CDC references) and age interval of assessment (0–3, 0–6, 6–12 and 0–12 months). Pooled data from singleton term infants from two observational studies on maternal mood disorders during pregnancy were used (n=161). Differences in RWG prevalence by growth reference and age interval were tested using Cochran's Q and McNemar's tests. The CDC reference produced a higher RWG prevalence (14% of infants additionally categorized as RWG, p<0.0001) within the 0–3 month age interval compared to the WHO standards; this pattern was reversed for the 6–12 and 0–12 month intervals. RWG prevalence did not differ across age interval within the WHO standards, but did differ with the CDC references (range: 22% for 0–3 months, to 4.2% for 6–12 months, p<0.0001). Caution is advised when comparing studies with different criteria for RWG. Future studies should use the 2006 WHO standards and a consistent age interval of evaluation.

Keywords

Rapid weight gain; growth references; infant growth

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INTRODUCTION

Childhood obesity may be caused in large part by risk factors experienced in utero and in infancy, resulting in early infant growth patterns associated with future obesity and related chronic disease (Gillman, 2005; Sinclair et al., 2007; Hanson & Gluckman, 2008). Potential pathways include the influences of maternal nutrition and hormones on child metabolism, gene expression, and related growth and development (IOM, 2009; Gillman et al., 2006; Gluckman & Hanson, 2008; Gluckman et al., 2011; Sullivan & Grove, 2010; Sibley et al., 2010). These findings, combined with strong evidence for the tracking of obesity from childhood into adulthood (Singh et al., 2008) provide a powerful argument for early intervention.

Infant rapid weight gain (RWG) is a growth pattern of concern that could be used for the early identification of children at increased risk for later obesity. Multiple studies have concluded that infant RWG is associated with obesity in later childhood and adulthood (Hui et al., 2008; Lamb et al., 2010; Shehadeh et al., 2008; Stettler et al., 2003; Stettler et al., 2002; Stettler, 2007; Baird et al., 2005; Dennison et al., 2006; Ong & Loos, 2006b; Monteiro & Victora, 2005; Goodell et al., 2009; Karaolis-Danckert et al., 2008). This relationship holds even when birth size is accounted for, indicating an association regardless of whether the RWG is “catch-up growth” in babies born small-for-gestational-age, or whether it occurs in larger neonates (Karaolis-Danckert et al., 2008; Ong & Loos, 2006a; Taveras et al., 2009). Although there is variation in how infant RWG is measured (Druet et al., 2012; Bekkers et al., 2011; Karp et al., 2012; Taveras et al., 2011; Moss & Yeaton, 2012; Stettler & Iotova, 2010), some consensus has been reached. The most common and recommended definition of RWG is an increase of > 0.67 in sex-specific weight-for-age z-score (WAZ) within a specific time period, representing biologically significant weight gain coincident with crossing one major percentile line on a growth chart (Ong et al., 2000; Monteiro & Victora, 2005; Druet & Ong, 2008).

However, two specific inconsistencies in the measurement of RWG remain. First, much of the research on infant RWG is based on z-scores calculated using the 2000 US Centers for Disease Control and Prevention (CDC) growth references (Stettler et al., 2003; Stettler et al., 2002; Dennison et al., 2006; Goodell et al., 2009; Taveras et al., 2009), rather than the 2006 World Health Organization (WHO) growth standards. The WHO standards are considered the best model of normative growth and have been widely adopted worldwide (de Onis et al., 2012), including in the US for children < 24 months (Grummer-Strawn et al., 2010). The WHO standards are based on longitudinal data from an international cohort of healthy breastfed children, while the CDC references are based on a mix of cross-sectional and longitudinal data from formula and mixed-fed US infants (Kuczmarski et al., 2002; Dewey et al., 1995; Hediger et al., 2000); the growth trajectories between the references differ most during the first 24 months of life, when RWG is measured (de Onis et al., 2007a). With respect to WAZ, the measure used to evaluate RWG, the WHO standards have higher mean values before 6 months of age compared to the CDC references, then lower mean values through 24 months of age. Several studies have evaluated the impact of using the WHO standards versus the CDC references on the prevalence of various anthropometric indicators, such as stunting, wasting and overweight, and have found meaningful differences (de Onis et al., 2007a; de Onis et al., 2011; Mei & Grummer-Strawn, 2011; Mei et al., 2008; Padula et al., 2012; Mushtaq et al., 2012; Rifas-Shiman et al., 2011; Twells & Newhook, 2011; Maalouf-Manasseh et al., 2011). However, we are unaware of any study exploring how RWG prevalence differs by growth reference.

Second, there is a lack of consistency regarding the age interval used to evaluate infant RWG. The age intervals used in the literature vary from approximately 3 to 24 months in length, and differ in timing as well; some studies measure RWG immediately following birth, others focus on the period following the introduction of solid foods, while others assess RWG over the entirety of the first 1–2 years of life (Hui et al., 2008; Lamb et al., 2010; Shehadeh et al., 2008; Stettler et al., 2003; Stettler et al., 2002; Stettler, 2007; Baird et al., 2005; Dennison et al., 2006; Ong & Loos, 2006b; Monteiro & Victora, 2005; Goodell et al., 2009; Karaolis-Danckert et al., 2008). Early life encompasses a dynamic window of developmental plasticity, and research indicates that nutrition and growth during this period have the ability to impact obesity and disease risk over the entire life course (Dyer & Rosenfeld, 2011; Corvalan et al., 2009; Wells et al., 2007; Barker, 2004). A consistent age interval for evaluating RWG should be defined, both to establish a reliable measure and to recognize that the variable nature of early growth may be relevant for reflecting causes and predicting outcomes.

This study used pooled data from two observational studies on mood disorders and medication use during pregnancy to contribute towards filling these gaps in the literature. We evaluated whether infant RWG prevalence differs by growth reference used for evaluation (WHO standards vs. CDC references) and age interval of assessment (0–3 months, 0–6 months, 6–12 months and 0–12 months). This work complements existing studies by incorporating RWG into the body of anthropometric indicators compared between the WHO and CDC growth references, and will contribute towards efforts to formulate reliable methods for defining infant RWG—a potentially useful early predictor of subsequent obesity risk.

METHODS

Study Design and Population

We used secondary pooled data from two prospective observational studies focused on maternal and infant outcomes associated with maternal mood disorders with and without prescribed medication use during pregnancy; both studies were carried out by the same principal investigator with similar protocols. One study focused on major depressive disorder with and without selective serotonin reuptake inhibitor antidepressant (SSRI) medication use (Wisner et al., 2009); the other focused on maternal bipolar disorder with and without antimanic/other psychotropic medication exposure. The study on depression enrolled women aged 15–44 years at ≤ 20 weeks gestation in two US cities (Cleveland, OH and Pittsburgh, PA) with exclusions for psychosis, bipolar disorder or chronic diseases. The study on bipolar disorder enrolled women aged 18–45 years at ≤ 20 weeks gestation at one site (Pittsburgh, PA) with exclusions for substance use excluding marijuana. In both cases, women with diagnoses (DSM-IV major depression disorder or bipolar disorder of any subtype), both medicated and unmedicated, were enrolled along with controls. Study evaluations occurred throughout pregnancy, with follow-up visits at targets of 2 weeks, 12 weeks, 26 weeks and 52 weeks postpartum. In the study on depression, follow-up data was additionally collected at 78 and 104 weeks postpartum. The joint data set is called the Antidepressant and Antimanic Medication Use during Pregnancy (ADMUP) dataset.

While mood disorders with and without medication use were the primary exposures in these studies, these exposures do not impact the validity and conclusions of the present work. The objective of the present study was to evaluate whether different criteria for defining RWG alter the prevalence of this measure within a population, with paired comparisons of the same measures within individuals that together represent a distribution of sizes and growth patterns. Growth trajectories within any population are influenced by a variety of exposures, however these exposures do not influence how one growth reference or other criterion performs versus another. Furthermore, since mood disorders are associated with obesity in women (Roberts et al., 2003; Onyike et al., 2003) and maternal obesity and depression are associated with child obesity (Yu et al., 2013; Cnattingius et al., 2012; Gross et al., 2013; Wang et al., 2013; Morrissey & Dagher, 2014), this population may serve as an at-risk population of interest.

Among the 474 eligible women recruited across the pooled studies, delivery data were available for 323 singleton, term (≥ 37 weeks gestation) infants. With attrition and the availability of follow-up data, the analytic sample for the present study was n=161.

Measurement and Definition of RWG

Birth weight and length were obtained by review of hospital delivery records. Anthropometric measures at follow-up points were assessed by trained study personnel blinded to exposure. Weight was measured on standard digital scales to the nearest 1 gram. Length in centimeters was measured from crown to heel on pediatric examination tables with built-in rulers. Weight measures were converted to exact age- and sex-specific WAZ using both the WHO growth standards and the CDC growth references for measures at birth, 3 months of age, 6 months of age and 12 months of age. For each measurement after birth, we accepted measures from ± 6 weeks of the target age to allow for variation in actual timing of follow-up visits, with age-specific z-scores calculated using exact age at measurement. For each set of growth reference-specific z-scores, children were categorized with RWG using the accepted definition of an increase of > 0.67 in WAZ (Monteiro & Victora, 2005) within each of four age intervals: 0–3 months, 0–6 months, 6–12 months and 0–12 months of life. These age intervals correspond to those commonly seen in the RWG literature, and reflect different developmental stages; the 0–3 and 0–6 month intervals occur prior to the recommended introduction of solid foods (WHO, 2003), the 6–12 month interval follows the recommended age for the initiation of solid foods, and the 0–12 month interval encompasses the first year of life.

Infant and Maternal Characteristics

Infant characteristics evaluated included sex, birth weight, and ever breastfed status (yes/no) from clinical interview. Maternal characteristics included self-identified race (white, African-American, or other), study exposure status from chart review (categorized as control or exposed (either MDD or bipolar with or without medication use)), and pre-pregnancy body mass index (BMI, weight(kg)/height(m)^2) categorized as underweight (BMI < 18.5), normal (18.5 ≤ BMI < 25), overweight (25 ≤ BMI < 30), or obese (BMI ≥ 30)(WHO, 2000) based on self-reported weight and measured height.

Analytic Sample Sizes for Designated Age Intervals

Delivery data were available for 323 singleton term infants. With attrition and the limits placed on acceptable ages for anthropometric measurements, the mean length ± standard deviation and analytic sample sizes for the designated age intervals were 14.4 ± 1.7 weeks for the 0–3 month interval (n=161), 29.1 ± 1.6 weeks for the 0–6 month interval (n=129), 24.6 ± 2.7 weeks for the 6–12 month interval (n=97), and 53.6 ± 2.0 weeks for the 0–12 month interval (n=134).

Statistical Analysis

Within each age interval, McNemar's test for the difference between proportions in paired data was used to determine whether infant RWG prevalence differed by growth reference used for assessment (McNemar, 1947; Lachenbruch & Lynch, 1998; Sheskin, 2004). When the number of discordant pairs was < 10, the exact form of McNemar's test was used (Siegel, 1988). As a sensitivity analysis, we repeated these analyses for the smaller sample (n=72) of infants with available measures at all follow-up points, to determine whether our results were affected by variation in the individuals included in each age interval.

Within each growth reference, for the subset of the population with data across all four age intervals of interest (n=72), Cochran's Q was used to determine whether infant RWG prevalence differed among the four age intervals of interest (Cochran, 1950). When Cochran's Q was significant, we used pairwise McNemar's tests to determine which intervals differed from each other (Sheskin, 2004; Lachenbruch & Lynch, 1998; McNemar, 1947).

The calculation of growth-related z-scores is sex-specific, which provides some inherent control for variation by sex, and none of our results differed by sex. Thus our results are pooled across sex.

RESULTS

Descriptive statistics were calculated for the n=161 infants with RWG data for the 0–3 month age interval, with some missing values, primarily for the maternal pre-pregnancy BMI data (21 missing values). The majority of mothers were white (84%), with the remaining mothers primarily African-American (14%). One quarter of the women were married or cohabiting, while the rest were single (including women that were separated, widowed or divorced). The majority of mothers had either completed their GED or graduated from high school (30%), or had completed further education, such as college, graduate work, or vocational training (67%). Approximately half of mothers (49%) were categorized as exposed (major depressive disorder or bipolar, with or without medication use). Pre-pregnancy BMI data indicated that 16% of mothers were overweight and 29% were obese. Mean maternal age was 31 ± 5 years.

With respect to infant characteristics, 57% were male, and 77% were ever breastfed. Mean birth weight was 3551 ± 504 g, producing a mean WAZ at birth of 0.48 ± 0.97 using the WHO growth standards, and 0.17 ± 0.97 using the CDC growth references.

The proportion of infants categorized with RWG was significantly different by the growth reference used for WAZ calculation for each age interval tested, except for the interval from 0–6 months; the CDC reference produced a higher prevalence of RWG in early infancy, and the WHO standards produced a higher prevalence in later infancy and across the first year of life as a whole (Table I). For the 0–3 month age interval, 9.9% of infants were categorized as RWG by both references, with an additional 14.3% of infants categorized as RWG using the CDC references but not the WHO standards ($p<0.0001$). For the 6–12 and the 0–12 month intervals, the pattern was reversed; a proportion of infants was categorized as RWG using both references, with an additional proportion categorized as RWG using the WHO standards but not the CDC references (e.g., 5.2% of infants exhibited RWG regardless of which reference was used from 6–12 months, with an additional 9.3% categorized as RWG using the WHO standards only ($p=0.0039$)).

We compared the proportion of infants categorized with RWG across the four age intervals of interest within each growth reference for the subsample of infants with data for all age intervals considered ($n=72$). The prevalence of RWG differed by age interval when the CDC references were used for evaluation (Table II); McNemar's paired chi-square tests indicated that each of the four age intervals differed significantly from each other with respect to the proportion of infants with RWG ($p<0.05$), *except* for the 0–3 month (22.2%) vs. 0–6 month comparison (19.4%), and the 6–12 month (4.2%) vs. 0–12 month (11.1%) comparison. There were no differences in the proportion of infants with RWG across age intervals when the WHO growth standards were used for evaluation.

DISCUSSION

This study showed that the proportion of infants categorized with RWG differed by the criteria used for its assessment (growth reference and age interval). Compared to the CDC growth references, the WHO growth standards resulted in a significantly lower prevalence of RWG in early infancy (0–3 months of age), and a higher prevalence of RWG for the 6–12 month and 0–12 month age intervals, reflecting the different shapes of the growth trajectories between the two references. Furthermore, RWG prevalence did not differ across various age intervals with the WHO standards but did with the CDC reference, indicating that the age interval of evaluation has less influence on the reliability of the RWG measure when using the WHO standards.

A number of studies have compared growth and body composition measures (e.g., stunting, wasting and overweight) between the WHO standards and CDC references and have found meaningful differences (de Onis et al., 2007a; de Onis et al., 2011; Mei & Grummer-Strawn, 2011; Mei et al., 2008; Padula et al., 2012; Mushtaq et al., 2012; Rifas-Shiman et al., 2011; Twells & Newhook, 2011; Maalouf-Manasseh et al., 2011). However, we are unaware of any study comparing RWG prevalence, using the definition of an increase of > 0.67 WAZ, between the two growth references. As previously stated, the magnitude of > 0.67

corresponds to the crossing *one* major percentile line on a growth chart (Monteiro & Victora, 2005; Ong et al., 2000). We found one related paper (Mei & Grummer-Strawn, 2011) that examined the percentage of children that crossed at least *two* major percentile lines at any point during the first 24 months of age for a variety of anthropometric measures including WAZ. Similar to our findings regarding RWG, this study found that the prevalence of children that crossed two or more growth chart percentile lines for WAZ was higher in the first six months of life when using the CDC vs. the WHO references, with the opposite pattern seen from 6–12 months; however no statistical testing was presented (Mei & Grummer-Strawn, 2011).

A primary limitation of this research is sample size and attrition, which are common issues with any longitudinal study, and particularly in samples with mood disorders. In order to maximize our sample sizes, we used all available data for each age interval considered, with diminished sample sizes in the later age intervals. To confirm that the differences in RWG we observed across age intervals were not related to differences in the population analyzed, we repeated our analyses with the subsample of children with compete data (n=72) as a sensitivity analyses, with very similar results. Ultimately, we would have liked to have had enough children at a follow-up point beyond one year of age to determine, controlling for key covariates, whether RWG for each age interval/growth reference combination was associated with subsequent childhood obesity. Although follow-up data were available for n=113 children at 2 years of age, no child was obese (BMI z-score > 3) and only one child was overweight (BMI z-score > 2) by the WHO definition (de Onis et al., 2007b), thus the sample sizes were too low to meaningfully calculate the odds of obesity or overweight/obesity as a function of infant RWG categorization. Simple t-tests did indicate that mean BMI percentiles were higher at 2 years among children that exhibited RWG in infancy, regardless of which growth reference was used, for all the age intervals of evaluation except the 6–12 month interval (data not shown). We look forward to further research using a larger study population with follow-up anthropometric data to clarify these associations and the ability of a consistently defined RWG measure in infancy to serve as a valid predictor of subsequent obesity. No study we are aware of has looked at this issue.

Another limitation was the need to allow for a window of time (± 6 weeks) around each target age for including infant anthropometric data in our analyses due to variations in timing of follow-up visits. Despite these windows, the defined age intervals still framed meaningful differences in interval length and timing during infancy. Furthermore, when we reduced the window to ± 4 weeks, our results were very similar (data not shown).

Lastly, the potential for measurement error is always a limitation with anthropometric measurement data. Although some error is unavoidable, it is likely to be random in nature. Furthermore, the anthropometric measure most prone to error, length in children < 24 months (Rifas-Shiman et al., 2005), is not used for evaluating RWG. Additionally, since the comparisons among different criteria for defining RWG depend on the same anthropometric measurements for each individual, our results are still informative.

This paper contributes towards addressing the lack of reliability across the literature in the measurement of infant RWG. It should be noted that the inconsistencies highlighted in this research are not errors, but rather the natural consequences of shifting to improved reference data for monitoring infant growth (WHO standards vs. CDC references), and the general paucity and variation in available longitudinal infant growth data (resulting in variation in age intervals assessed). The literature shows that RWG measures, despite inconsistent methods of assessment, tend to predict subsequent obesity risk in various populations overall. This literature is important and highlights the potential for infant RWG to be used as an effective screening tool for the early identification of infants at risk for subsequent obesity. That being said, comparing results across studies using varied criteria for assessment is discouraged. It is our intent to draw attention to these issues as a call for a reliable and valid definition for infant RWG based consistently on the recommended WHO growth standards and on a defined age interval selected to serve as a valid predictor of subsequent obesity risk.

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References

Baird J, Fisher D, Lucas P, Kleijnen J, Roberts H, Law C. Being big or growing fast: systematic review of size and growth in infancy and later obesity. BMJ. 2005; 331:929. [PubMed: 16227306]

Barker DJ. Developmental origins of adult health and disease. J Epidemiol Community Health. 2004; 58:114–115. [PubMed: 14729887]

Bekkers MB, Brunekreef B, Smit HA, Kerkhof M, Koppelman GH, Oldenwening M, Wijga AH. Early-life determinants of total and HDL cholesterol concentrations in 8-year-old children; the PIAMA birth cohort study. PLoS One. 2011; 6:e25533. [PubMed: 21980486]

Cnattingius S, Villamor E, Lagerros YT, Wikstrom AK, Granath F. High birth weight and obesity--a vicious circle across generations. Int J Obes (Lond). 2012; 36:1320–1324. [PubMed: 22158263]

Cochran WG. The comparison of percentages in matched samples. Biometrika. 1950; 37:256–266. [PubMed: 14801052]

Corvalan C, Kain J, Weisstaub G, Uauy R. Impact of growth patterns and early diet on obesity and cardiovascular risk factors in young children from developing countries. Proc Nutr Soc. 2009; 68:327–337. [PubMed: 19400973]

de Onis M, Garza C, Onyango AW, Borghi E. Comparison of the WHO child growth standards and the CDC 2000 growth charts. J Nutr. 2007a; 137:144–148. [PubMed: 17182816]

de Onis M, Onyango A, Borghi E, Siyam A, Blossner M, Lutter C. WHO Multicentre Growth Reference Study Group. Worldwide implementation of the WHO Child Growth Standards. Public Health Nutr. 2012; 15:1603–1610. [PubMed: 22717390]

de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. Bull World Health Organ. 2007b; 85:660–667. [PubMed: 18026621]

de Onis M, Siyam A, Borghi E, Onyango AW, Piwoz E, Garza C. Comparison of the World Health Organization growth velocity standards with existing US reference data. Pediatrics. 2011; 128:e18–26. [PubMed: 21708799]

Dennison BA, Edmunds LS, Stratton HH, Pruzek RM. Rapid infant weight gain predicts childhood overweight. Obesity (Silver Spring). 2006; 14:491–499. [PubMed: 16648621]

Dewey KG, Peerson JM, Brown KH, Krebs NF, Michaelsen KF, Persson LA, Salmenpera L, Whitehead RG, Yeung DL. Growth of breast-fed infants deviates from current reference data: a pooled analysis of US, Canadian, and European data sets. World Health Organization Working Group on Infant Growth. Pediatrics. 1995; 96:495–503. [PubMed: 7651784]

Druet C, Ong KK. Early childhood predictors of adult body composition. Best Pract Res Clin Endocrinol Metab. 2008; 22:489–502. [PubMed: 18538288]

Druet C, Stettler N, Sharp S, Simmons RK, Cooper C, Smith GD, Ekelund U, Levy-Marchal C, Jarvelin MR, Kuh D, Ong KK. Prediction of childhood obesity by infancy weight gain: an individual-level meta-analysis. Paediatr Perinat Epidemiol. 2012; 26:19–26. [PubMed: 22150704]

Dyer JS, Rosenfeld CR. Metabolic imprinting by prenatal, perinatal, and postnatal overnutrition: a review. Semin Reprod Med. 2011; 29:266–276. [PubMed: 21769766]

Gillman M, Rich-Edwards J, Huh S, Majzoub J, Oken E, Taveras E, Rifas-Shiman S. Maternal corticotropin-releasing hormone levels during pregnancy and offspring adiposity. Obesity (Silver Spring). 2006; 14:1647–1653. [PubMed: 17030976]

Gillman MW. Developmental origins of health and disease. N Engl J Med. 2005; 353:1848–1850. [PubMed: 16251542]

Gluckman PD, Hanson MA. Developmental and epigenetic pathways to obesity: an evolutionary-developmental perspective. Int J Obes (Lond). 2008; 32(Suppl 7):S62–71. [PubMed: 19136993]

Gluckman PD, Hanson MA, Low FM. The role of developmental plasticity and epigenetics in human health. Birth Defects Res C Embryo Today. 2011; 93:12–18. [PubMed: 21425438]

Goodell LS, Wakefield DB, Ferris AM. Rapid weight gain during the first year of life predicts obesity in 2–3 year olds from a low-income, minority population. J Community Health. 2009; 34:370–375. [PubMed: 19526332]

Gross RS, Velazco NK, Briggs RD, Racine AD. Maternal depressive symptoms and child obesity in low-income urban families. Acad Pediatr. 2013; 13:356–363. [PubMed: 23830021]

Grummer-Strawn LM, Reinold C, Krebs NF. Use of World Health Organization and CDC growth charts for children aged 0–59 months in the United States. MMWR Recomm Rep. 2010; 59:1–15. [PubMed: 20829749]

Hanson MA, Gluckman PD. Developmental origins of health and disease: new insights. Basic Clin Pharmacol Toxicol. 2008; 102:90–93. [PubMed: 18226060]

Hediger ML, Overpeck MD, Ruan WJ, Troendle JF. Early infant feeding and growth status of US-born infants and children aged 4–71 mo: analyses from the third National Health and Nutrition Examination Survey, 1988–1994. Am J Clin Nutr. 2000; 72:159–167. [PubMed: 10871575]

Hui LL, Schooling CM, Leung SS, Mak KH, Ho LM, Lam TH, Leung GM. Birth weight, infant growth, and childhood body mass index: Hong Kong's children of 1997 birth cohort. Arch Pediatr Adolesc Med. 2008; 162:212–218. [PubMed: 18316657]

IOM. Weight Gain During Pregnancy: Reexamining the Guidelines. Washington, DC: The National Academies Press; 2009.

Karaolis-Danckert N, Buyken AE, Kulig M, Kroke A, Forster J, Kamin W, Schuster A, Hornberg C, Keil T, Bergmann RL, Wahn U, Lau S. How pre- and postnatal risk factors modify the effect of rapid weight gain in infancy and early childhood on subsequent fat mass development: results from the Multicenter Allergy Study 90. Am J Clin Nutr. 2008; 87:1356–1364. [PubMed: 18469259]

Karp RJ, Winkfield-Royster T, Weedon J. Relation of growth rate from birth to three months and four to six months to body mass index at ages four to six years. J Nutr Metab. 2012; 2012:158643. [PubMed: 22474576]

Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, Wei R, Curtin LR, Roche AF, Johnson CL. 2000 CDC Growth Charts for the United States: methods and development. Vital Health Stat. 2002; 11:1–190.

Lachenbruch PA, Lynch CJ. Assessing screening tests: extensions of McNemar's test. Stat Med. 1998; 17:2207–2217. [PubMed: 9802179]

Lamb MM, Dabelea D, Yin X, Ogden LG, Klingensmith GJ, Rewers M, Norris JM. Early-life predictors of higher body mass index in healthy children. Ann Nutr Metab. 2010; 56:16–22. [PubMed: 19940472]

Maalouf-Manasseh Z, Metallinos-Katsaras E, Dewey KG. Obesity in preschool children is more prevalent and identified at a younger age when WHO growth charts are used compared with CDC charts. J Nutr. 2011; 141:1154–8. [PubMed: 21525264]

McNemar Q. Note on the sampling error of the difference between correlated proportions or percentages. Psychometrika. 1947; 12:153–157. [PubMed: 20254758]

Mei Z, Grummer-Strawn LM. Comparison of changes in growth percentiles of US children on CDC 2000 growth charts with corresponding changes on WHO 2006 growth charts. Clin Pediatr (Phila). 2011; 50:402–407. [PubMed: 21242198]

Mei Z, Ogden CL, Flegal KM, Grummer-Strawn LM. Comparison of the prevalence of shortness, underweight, and overweight among US children aged 0 to 59 months by using the CDC 2000 and the WHO 2006 growth charts. J Pediatr. 2008; 153:622–628. [PubMed: 18619613]

Monteiro P, Victora C. Rapid growth in infancy and childhood and obesity in later life--a systematic review. Obes Rev. 2005; 6:143–154. [PubMed: 15836465]

Morrissey TW, Dagher RK. A longitudinal analysis of maternal depressive symptoms and children's food consumption and weight outcomes. Public Health Nutr. 2014:1–10. [PubMed: 24650538]

Moss BG, Yeaton WH. U.S. children's preschool weight status trajectories: patterns from 9-month, 2-year, and 4-year Early Childhood Longitudinal Study-Birth cohort data. Am J Health Promot. 2012; 26:172–175. [PubMed: 22208415]

Mushtaq MU, Gull S, Mushtaq K, Abdullah HM, Khurshid U, Shahid U, Shad MA, Akram J. Height, weight and BMI percentiles and nutritional status relative to the international growth references among Pakistani school-aged children. BMC Pediatr. 2012; 12:31. [PubMed: 22429910]

Ong K, Loos R. Rapid infancy weight gain and subsequent obesity: systematic reviews and hopeful suggestions. Acta Paediatr. 2006a; 95:904–908. [PubMed: 16882560]

Ong KK, Ahmed ML, Emmett PM, Preece MA, Dunger DB. Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. BMJ. 2000; 320:967–971. [PubMed: 10753147]

Ong KK, Loos RJ. Rapid infancy weight gain and subsequent obesity: systematic reviews and hopeful suggestions. Acta Paediatr. 2006b; 95:904–908. [PubMed: 16882560]

Onyike CU, Crum RM, Lee HB, Lyketsos CG, Eaton WW. Is obesity associated with major depression? Results from the Third National Health and Nutrition Examination Survey. Am J Epidemiol. 2003; 158:1139–1147. [PubMed: 14652298]

Padula G, Seoane AI, Salceda SA. Variations in estimates of underweight, stunting, wasting, overweight and obesity in children from Argentina comparing three growth charts. Public Health Nutr. 2012; 15:2086–2090. [PubMed: 22444072]

Rifas-Shiman SL, Gillman MW, Oken E, Kleinman K, Taveras EM. Similarity of the CDC and WHO Weight-for-Length Growth Charts in Predicting Risk of Obesity at Age 5 Years. Obesity (Silver Spring). 2011; 20:1261–1265. [PubMed: 22158005]

Rifas-Shiman SL, Rich-Edwards JW, Scanlon KS, Kleinman KP, Gillman MW. Misdiagnosis of overweight and underweight children younger than 2 years of age due to length measurement bias. MedGenMed. 2005; 7:56. [PubMed: 16614678]

Roberts RE, Deleger S, Strawbridge WJ, Kaplan GA. Prospective association between obesity and depression: evidence from the Alameda County Study. Int J Obes Relat Metab Disord. 2003; 27:514–521. [PubMed: 12664085]

Shehadeh N, Weitzer-Kish H, Shamir R, Shihab S, Weiss R. Impact of early postnatal weight gain and feeding patterns on body mass index in adolescence. J Pediatr Endocrinol Metab. 2008; 21:9–15. [PubMed: 18404968]

Sheskin, D. Handbook of parametric and nonparametric statistical procedures. Boca Raton, FL: CLC Press; 2004.

Sibley CP, Brownbill P, Dilworth M, Glazier JD. Review: Adaptation in placental nutrient supply to meet fetal growth demand: implications for programming. Placenta. 2010; 31(Suppl):S70–S74. [PubMed: 20060581]

Siegel, SCNJ. Nonparametric statistics for the behavioral sciences. New York, NY: McGraw-Hill; 1988.

Sinclair KD, Lea RG, Rees WD, Young LE. The developmental origins of health and disease: current theories and epigenetic mechanisms. Soc Reprod Fertil Suppl. 2007; 64:425–443. [PubMed: 17491163]

Singh AS, Mulder C, Twisk JW, Van Mechelen W, Chinapaw MJ. Tracking of childhood overweight into adulthood: a systematic review of the literature. Obes Rev. 2008; 9:474–488. [PubMed: 18331423]

Stettler N. Nature and strength of epidemiological evidence for origins of childhood and adulthood obesity in the first year of life. Int J Obes (Lond). 2007; 31:1035–1043. [PubMed: 17589539]

Stettler N, Iotova V. Early growth patterns and long-term obesity risk. Curr Opin Clin Nutr Metab Care. 2010; 13:294–299. [PubMed: 20179588]

Stettler N, Kumanyika SK, Katz SH, Zemel BS, Stallings VA. Rapid weight gain during infancy and obesity in young adulthood in a cohort of African Americans. Am J Clin Nutr. 2003; 77:1374–1378. [PubMed: 12791612]

Stettler N, Zemel BS, Kumanyika S, Stallings VA. Infant weight gain and childhood overweight status in a multicenter, cohort study. Pediatrics. 2002; 109:194–199. [PubMed: 11826195]

Sullivan EL, Grove KL. Metabolic imprinting in obesity. Forum Nutr. 2010; 63:186–194. [PubMed: 19955786]

Taveras E, Rifas-Shiman S, Belfort M, Kleinman K, Oken E, Gillman M. Weight status in the first 6 months of life and obesity at 3 years of age. Pediatrics. 2009; 123:1177–1183. [PubMed: 19336378]

Taveras EM, Rifas-Shiman SL, Sherry B, Oken E, Haines J, Kleinman K, Rich-Edwards JW, Gillman MW. Crossing growth percentiles in infancy and risk of obesity in childhood. Arch Pediatr Adolesc Med. 2011; 165:993–998. [PubMed: 22065180]

Twells LK, Newhook LA. Obesity prevalence estimates in a Canadian regional population of preschool children using variant growth references. BMC Pediatr. 2011; 11:21. [PubMed: 21356057]

Wang L, Anderson JL, Dalton WT III, Wu T, Liu X, Zheng S, Liu X. Maternal depressive symptoms and the risk of overweight in their children. Matern Child Health J. 2013; 17:940–948. [PubMed: 22833333]

Wells JC, Chomtho S, Fewtrell MS. Programming of body composition by early growth and nutrition. Proc Nutr Soc. 2007; 66:423–434. [PubMed: 17637095]

WHO. Report of a WHO consultation. Geneva, Switzerland: World Health Organization; 2000. Obesity: preventing and managing the global epidemic. World Health Organ Tech Rep Ser 894

WHO. Global strategy for infant and young child feeding. Geneva, Switzerland: World Health Organization; 2003.

Wisner KL, Sit DK, Hanusa BH, Moses-Kolko EL, Bogen DL, Hunker DF, Perel JM, Jones-Ivy S, Bodnar LM, Singer LT. Major depression and antidepressant treatment: impact on pregnancy and neonatal outcomes. Am J Psychiatry. 2009; 166:557–566. [PubMed: 19289451]

Yu Z, Han S, Zhu J, Sun X, Ji C, Guo X. Pre-pregnancy body mass index in relation to infant birth weight and offspring overweight/obesity: a systematic review and meta-analysis. PLoS One. 2013; 8:e61627. [PubMed: 23613888]

Table I

Proportion of infants with RWG within each age interval by growth reference

	Proportion categorized as RWG				
	Both references	**CDC reference only**	**WHO standards only**	**n***	**p-value†**
0–3 months	16 (9.9%)	23 (14.3%)	0	161	<0.0001
0–6 months	17 (13.2%)	5 (3.9%)	0	129	0.0625
6–12 months	5 (5.2%)	0	9 (9.3%)	97	0.0039
0–12 months	14 (10.5%)	0	7 (5.2%)	134	0.0156

*Denominator for each age interval, reflecting attrition and availability of longitudinal data.

†In the case of the 0–3 month interval, the p-value for McNemar's chi-square is reported. For all other intervals, the p-values shown are the Exact McNemar's significance probabilities because the number of discordant pairs for each of these intervals is <10.

Table II

Proportion of infants with RWG within each growth reference by age interval*

	0–3 months	0–6 months	6–12 months	0–12 months	p-value for Cochran's Q
CDC references	16 (22.2%)	14 (19.4%)	3 (4.2%)	8 (11.1%)	0.0002†
WHO standards	6 (8.3%)	12 (16.7%)	9 (12.5%)	12 (16.7%)	0.2424

*n=72 for this analysis, the analytic sample without any missing RWG measures across all age intervals.

†McNemar's paired chi-square tests revealed that all age intervals were statistically significantly different from each other ($p<0.05$) except for the 0–3 month v. 0–6 month interval comparison, and the 6–12 month v. 0–12 month comparison.