Racial/Ethnic Differences in Early-Life Risk Factors for Childhood Obesity

abstract

OBJECTIVE: By the preschool years, racial/ethnic disparities in obesity prevalence are already present. The objective of this study was to examine racial/ethnic differences in early-life risk factors for childhood obesity.

METHODS: A total of 1343 white, 355 black, and 128 Hispanic mother–child pairs were studied in a prospective study. Mother’s reported child’s race/ethnicity. The main outcome measures were risk factors from the prenatal period through 4 years old that are known to be associated with child obesity.

RESULTS: In multivariable models, compared with their white counterparts, black and Hispanic children exhibited a range of risk factors related to child obesity. In pregnancy, these included higher rates of maternal depression (odds ratio [OR]: 1.55 for black, 1.89 for Hispanic); in infancy more rapid weight gain (OR: 2.01 for black, 1.75 for Hispanic), more likely to introduce solid foods before 4 months of age (OR: 1.91 for black, 2.04 for Hispanic), and higher rates of maternal restrictive feeding practices (OR: 2.59 for black, 3.35 for Hispanic); and after 2 years old, more televisions in their bedrooms (OR: 7.65 for black, 7.99 for Hispanic), higher intake of sugar-sweetened beverages (OR: 4.11 for black, 2.48 for Hispanic), and higher intake of fast food (OR: 1.65 for black, 3.14 for Hispanic). Black and Hispanic children also had lower rates of exclusive breastfeeding and were less likely to sleep at least 12 hours/day in infancy.


WHAT’S KNOWN ON THIS SUBJECT: By the preschool years, racial/ethnic disparities in obesity prevalence are already present.

WHAT THIS STUDY ADDS: Racial/ethnic differences in risk factors for obesity exist prenatally and in early childhood. Racial/ethnic disparities in childhood obesity may be determined by factors that operate at the earliest stages of life.
By the preschool years, racial/ethnic disparities in obesity prevalence are already present. According to the most recent National Health and Nutrition Examination Survey (2003–2006), the prevalence of children who are aged 2 through 5 years and have a BMI ≥95th percentile was 10.7% among non-Hispanic white children, 14.9% among non-Hispanic black children, and 16.7% among Mexican American children. These data suggest that racial/ethnic disparities in childhood obesity prevalence have their origins in the earliest stages of life.

The life-course approach to chronic disease prevention posits that factors may act in the prenatal period into infancy, childhood, and beyond to determine risk for chronic disease. These factors can range from the social/built environment through behavior, physiology, and genetics. Factors interact with each other over the life course, with various determinants being more or less important at various life stages. Today we recognize that the prenatal, infancy, and early childhood periods are likely to be key to the development—and thus prevention—of obesity and its consequences in children.

In recent years, investigators have identified several factors in the prenatal, infancy, and early childhood periods that increase the risk for obesity in childhood. Few studies, however, have examined racial/ethnic differences in the prevalence of obesity-related risk factors during these periods. Racial/ethnic differences in early-life risk factors for obesity might contribute to the high prevalence of obesity among minority preschool-aged children and beyond. Understanding these differences may help inform the design of clinical and public health interventions and policies to reduce disparities in childhood obesity and its complications.

The purpose of this study was to examine racial/ethnic differences in early-life risk factors for childhood obesity. We hypothesized that compared with white children, black and Hispanic children would have higher rates of obesity-related risk factors in early life.

METHODS

Subjects/Study Design

Study subjects were participants in Project Viva, a prospective, prebirth cohort study. Details of recruitment and retention procedures are available elsewhere. Of the 2128 women who delivered a live infant, 2109 had information on our main exposure, child race/ethnicity. For this analysis, we excluded participants who were Asian (n = 94) or multiracial/other (n = 189) to examine risk factor differences among white children and children of racial/ethnic groups that are known to have a high prevalence of childhood obesity. Sample sizes for each time point were 1826 for prenatal and birth measures, 1517 for 6-month measures, 1420 for 1-year measures, 1212 for 2-year measures, 1086 for 3-year measures, and 1058 for 4-year measures.

After obtaining informed consent, we performed in-person study visits with the mother at the end of the first and second trimesters of pregnancy, in the first few days after delivery, and at 6 months and 3 years after birth. Mothers completed mailed questionnaires at 1, 2, and 4 years after birth. Human subjects committees of Harvard Pilgrim Health Care, Brigham and Women’s Hospital, and Beth Israel Deaconess Medical Center approved the study protocols.

Measurements

Main Exposure

At the 3-year interview, research assistants asked mothers the question, “Which of the following best describes your child’s race or ethnicity?” Mothers had a choice of ≥1 of the following racial/ethnic groups: Hispanic or Latina, white or Caucasian, black or African American, Asian or Pacific Islander, American Indian or Alaskan Native, and other (please specify). For the participants who chose the “other” race/ethnicity, we compared the specified responses with US census definitions for the other 5 races and ethnicities and reclassified them when appropriate.

Outcome Measures

The main outcomes were risk factors during the prenatal, infancy, and early childhood periods that are associated with childhood obesity in the medical literature.

Gestational Weight Gain

We calculated total gestational weight gain (GWG) by subtracting prepregnancy weight from the last prenatal weight. Studies have shown that excessive weight gain during pregnancy is associated with increased risk for offspring obesity in childhood and adolescence. In this analysis, we expressed GWG in categories on the basis of the recommendations of the Institute of Medicine.

Gestational Diabetes

 Mothers in the study were routinely screened for gestational diabetes at 26 to 28 weeks of gestation with a nonfasting oral glucose challenge test. We categorized women with ≥2 abnormal fasting glucose tolerance test results as having gestational diabetes on the basis of published criteria. Previous studies have found that gestational diabetes exposure is associated with higher adiposity among children.

Smoking During Pregnancy

We asked mothers at both first- and second-trimester visits about their cigarette smoking habits before and during pregnancy. A recent meta-
analysis of 14 studies showed that smoking during pregnancy is associated with a 50% increased odds of childhood obesity.14

Maternal Depression
Mothers completed during mid-pregnancy a validated 10-item Edinburgh Postpartum Depression Scale that queried history of depression and current depressive symptoms.15 We used the cut point of ≥13 to indicate probable depression during pregnancy.16 Previous studies found that maternal stressors are associated with childhood obesity.17,18

Fetal Growth and Rapid Infant Weight Gain
We calculated birth weight for gestational age z score as a measure of fetal growth.19 As a measure of infant weight gain, we calculated the difference between weight-for-age z scores at 6 months and at birth. Both fetal growth and rapid early infancy weight gain are associated with later BMI in childhood or adulthood.20–25

Cord Blood Adipokines
We collected cord blood samples from the umbilical vein after delivery of the infant and measured concentrations of leptin and adiponectin, as described previously.24,25 In Project Viva, lower cord blood leptin levels were found to be associated with higher BMI at 3 years old, whereas higher cord blood adiponectin levels predicted an increase in central adiposity.26

Infant Feeding
Research assistants asked mothers whether they had initiated breastfeeding. At 6 and 12 months, we asked mothers whether they were exclusively breastfeeding, mixed breast and formula feeding, weaned, or were formula feeding only. When they had stopped breastfeeding, we asked them the children’s age at cessation. A recent meta-analysis that examined duration of breastfeeding demonstrated a decrease in odds of later obesity for each additional month of breastfeeding.27

At 6 months, we also measured timing of introduction of solids (<4 months, 4–5 months, and ≥6 months). Previous studies found that introduction of solids at <4 months is a risk factor for increased infant weight gain.28,29

Maternal Control of Infant Feeding
At 1 year, we measured mothers’ reports of restricting their children’s food intake and pressuring their children to eat more food by using a modified Child Feeding Questionnaire.30 Restriction was measured by the question, “I have to be careful not to feed my child too much.” We derived a score of continuous pressure to eat from 5 questions of the modified Child Feeding Questionnaire. We previously reported the internal reliability of this score.31 Maternal feeding restriction is associated with greater odds of childhood obesity at age 3.32 In some studies, mothers’ pressure on their children to eat has been associated with disinhibited eating33 and increased child energy intake and body weight.34,35

Daily Sleep During Infancy
At 6 months, 1 year, and 2 years postpartum, we asked mothers to quantify the average amount of daily sleep that their children obtained during the past month. We calculated a weighted average of sleep duration from 6 months to 2 years. We recently showed in the Project Viva cohort that average daily sleep duration from 6 months to 2 years is inversely associated with obesity at 3 years.36

Daily Television Viewing During Infancy
At 6 months, 1 year, and 2 years postpartum, we asked mothers to report the number of hours their chil-
children watched television/videos on an average weekday and weekend day in the past month. We calculated a weighted average of television/video viewing from 6 months to 2 years. At 4 years old, we also asked mothers whether their child had a television in his or her bedroom. Previous studies have found that greater hours spent watching television at 3 years old was associated with obesity at 7 years old and that having a television in a child’s bedroom is a risk factor for child obesity.

Sugar-Sweetened Beverages, Fast Food, and Family Dinner

We used a validated semiquantitative child food frequency questionnaire completed by each mother when the child was 2 years old to estimate daily sugar-sweetened beverage intake. We defined a sugar-sweetened beverage as soda (not sugar-free), flavored milks, and fruit drinks (eg, Hi-C, Kool-Aid, lemonade). Previous studies have found that consumption of sugar-sweetened beverages is associated with childhood obesity, although the relationship between 100% fruit juice and obesity is still controversial; therefore, we excluded orange juice and other 100% fruit juice intake. At 3 years old, we asked mothers to report their child’s weekly servings of fast food by using a question adapted from a longitudinal study of adults. A similar, modified question was validated by association with childhood obesity in a study of 9- to 14-year-olds. Greater intake of fast food has been found to be associated with poorer diet quality among children and with higher BMI among adolescents. At 4 years old, we asked mothers to report how often their child ate supper or dinner together with family members. Eating dinner with family members was associated with healthful dietary intake patterns and with BMI in cross-sectional analyses of 9- to 14-year-olds.

Other Measures

We measured length/height and weight of children by using a calibrated stadiometer and scale. We calculated age- and gender-specific anthropometric measures by using US national reference data. Mothers reported their prepregnancy weight and height as well as fathers’ weight and height, from which we calculated their BMIs. We also collected information about maternal age, education, parity, marital status, immigration status, and household income.

Statistical Analysis

We first examined bivariate relationships of child race/ethnicity with other covariates and our main outcomes. We then used multivariable linear and logistic regression models to assess the independent effects of child race/ethnicity on our main outcomes. Our first model was adjusted for child gender only. We then additionally adjusted the multivariable models for demographic and socioeconomic factors including maternal age, education, and parity and household income. Because maternal prepregnancy BMI and paternal BMI could be confounders of the relationship between race/ethnicity and obesity-related risk factors, we additionally adjusted the multivariable models for these variables. It is also possible that adjustment for parental BMI may be overadjustment given shared family behaviors. Thus, we separately present models with and without such adjustment. We report re-

### TABLE 2

<table>
<thead>
<tr>
<th>Early-Life Risk Factors for Childhood Obesity Overall and According to Child Race/Ethnicity</th>
<th>Overall</th>
<th>White, Non-Hispanic</th>
<th>Black, Non-Hispanic</th>
<th>Hispanic or Latino</th>
</tr>
</thead>
<tbody>
<tr>
<td>Institute of Medicine GWG category, %</td>
<td>Adequate</td>
<td>34</td>
<td>34</td>
<td>33</td>
</tr>
<tr>
<td>Inadequate</td>
<td>15</td>
<td>13</td>
<td>20</td>
<td>18</td>
</tr>
<tr>
<td>Excessive</td>
<td>51</td>
<td>53</td>
<td>47</td>
<td>44</td>
</tr>
<tr>
<td>Gestational diabetes</td>
<td>5</td>
<td>5</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td>Mother smoked during early pregnancy</td>
<td>13</td>
<td>12</td>
<td>14</td>
<td>22</td>
</tr>
<tr>
<td>Maternal antenatal depression</td>
<td>9</td>
<td>7</td>
<td>15</td>
<td>18</td>
</tr>
<tr>
<td>Fetal growth, %</td>
<td>Large for gestational age</td>
<td>14</td>
<td>16</td>
<td>9</td>
</tr>
<tr>
<td>Small for gestational age</td>
<td>6</td>
<td>4</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>Cord blood adipokines, mean ± SD</td>
<td>Leptin, ng/mL</td>
<td>9.0 ± 6.7</td>
<td>8.7 ± 6.4</td>
<td>10.8 ± 8.1</td>
</tr>
<tr>
<td>Adiponectin, µg/mL</td>
<td>28.7 ± 6.8</td>
<td>28.8 ± 6.6</td>
<td>28.1 ± 7.7</td>
<td>28.5 ± 6.4</td>
</tr>
<tr>
<td>Rapid infant weight gain, %</td>
<td>Highest quartile of change in weight-for-age 0 to 6 mo</td>
<td>25</td>
<td>22</td>
<td>39</td>
</tr>
<tr>
<td>Infant feeding</td>
<td>Did not initiate breastfeeding, %</td>
<td>14</td>
<td>14</td>
<td>13</td>
</tr>
<tr>
<td>Not exclusively breastfed through 6 mo, %</td>
<td>79</td>
<td>76</td>
<td>88</td>
<td>90</td>
</tr>
<tr>
<td>Total duration of breastfeeding, mean ± SD, mo</td>
<td>5.7 ± 4.6</td>
<td>6.0 ± 4.6</td>
<td>4.6 ± 4.2</td>
<td>4.6 ± 4.2</td>
</tr>
<tr>
<td>Introduction of solids at &lt;4 mo, %</td>
<td>17</td>
<td>14</td>
<td>32</td>
<td>36</td>
</tr>
<tr>
<td>Maternal restriction of child feeding at 1 y, %</td>
<td>13</td>
<td>9</td>
<td>29</td>
<td>38</td>
</tr>
<tr>
<td>Highest quartile of pressure to eat score at 1 y, %</td>
<td>22</td>
<td>19</td>
<td>33</td>
<td>40</td>
</tr>
<tr>
<td>Infant sleep, %</td>
<td>Average daily sleep duration 6 mo to 2 y, &lt;12 h</td>
<td>34</td>
<td>30</td>
<td>62</td>
</tr>
<tr>
<td>Television viewing, %</td>
<td>Average daily television viewing 6 mo to 2 y, ≥2 h</td>
<td>17</td>
<td>15</td>
<td>32</td>
</tr>
<tr>
<td>Television in room where child sleeps at 4 y</td>
<td>15</td>
<td>7</td>
<td>51</td>
<td>56</td>
</tr>
<tr>
<td>Diet patterns</td>
<td>Any (vs none) sugar-sweetened beverage intake at 2 y, %</td>
<td>51</td>
<td>45</td>
<td>82</td>
</tr>
<tr>
<td>Any (vs none) fast food intake at 3 y, %</td>
<td>70</td>
<td>66</td>
<td>83</td>
<td>88</td>
</tr>
<tr>
<td>Family meal consumption per day at 4 y, mean ± SD</td>
<td>0.82 ± 0.26</td>
<td>0.83 ± 0.25</td>
<td>0.75 ± 0.31</td>
<td>0.83 ± 0.26</td>
</tr>
</tbody>
</table>
gression estimates (H9252) or odds ratios and 95% confidence intervals for child race/ethnicity. In the regression models, the referent group was white children. We performed data analyses with SAS 9.1 (SAS Institute, Cary, NC).

RESULTS

Table 1 shows characteristics of the 1026 mother–child pairs from Project Viva included in our analyses. Consistent with national statistics, we found that compared with white children, black and Hispanic children had lower birth weights for gestational age but higher BMI z scores and higher prevalence of obesity at 3 years old (Table 1).

Table 2 shows the overall and race/ethnicity-specific prevalence of obesity-related risk factors.

In crude models adjusted only for child gender, we observed racial/ethnic differences in almost every risk factor examined (Tables 3 and 4). Socioeconomic factors confounded the observed relationships between race/ethnicity and obesity-related risk factors. Adjustment for socioeconomic factors minimally (≤10%) attenuated our observed crude estimates for some risk factors (eg, GWG, birth weight for gestational age, infant weight gain), modestly (10%–30%) attenuated the estimates for other risk factors, particularly those in early childhood such as television viewing and dietary practices, and substantially (≥30%) attenuated others, including maternal smoking during pregnancy and total duration of breastfeeding.

TABLE 3

<table>
<thead>
<tr>
<th>Risk Factors for Childhood Obesity</th>
<th>Black, Non-Hispanic, Effect Estimate (95% CI)</th>
<th>Hispanic or Latino, Effect Estimate (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetal growth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birth weight-for-gestational age z score</td>
<td>-0.38 (-0.49 to -0.27)</td>
<td>-0.36 (-0.49 to -0.24)</td>
</tr>
<tr>
<td>Cord blood adipokines*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leptin (per 10-ng/mL increment)</td>
<td>0.30 (0.18 to 0.43)</td>
<td>0.33 (0.20 to 0.47)</td>
</tr>
<tr>
<td>Adiponectin (per 10-μg/mL increment)</td>
<td>-0.050 (-0.180 to 0.080)</td>
<td>-0.120 (-0.260 to 0.020)</td>
</tr>
<tr>
<td>Infant feeding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total duration of breastfeeding, mo</td>
<td>-1.41 (-2.05 to -0.77)</td>
<td>-0.48 (-1.19 to -0.23)</td>
</tr>
<tr>
<td>Diet patterns</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family meal consumption per day at 4 y</td>
<td>-0.070 (-0.120 to -0.030)</td>
<td>-0.080 (-0.130 to -0.030)</td>
</tr>
</tbody>
</table>

Crude models are adjusted for child gender only. SES models are additionally adjusted for maternal age, education, and parity and household income. Parental BMI models are adjusted for maternal and paternal prepregnancy BMI. Reference group for all models is white children. CI indicates confidence interval.

* Models that predicted cord blood adipokines are additionally adjusted for birth weight-for-gestational age z score.
feeding duration disappeared after adjustment for maternal and paternal BMI (Table 3).

In fully adjusted multivariable models, we observed several differences between black and Hispanic children compared with white children in a range of risk factors related to childhood obesity. Black and Hispanic children had lower fetal growth indices but grew very rapidly in the first 6 months of life (Tables 3 and 4). Although black and Hispanic mothers were more likely to initiate breastfeeding, they were less likely to breastfeed their infants exclusively to 6 months of age and were more likely to introduce solid foods before 4 months of age (Table 4, Fig 1). Black and Hispanic mothers were also more likely to exert greater control over their infants’ feeding by restricting and pressuring their children to eat (Table 4). Between 6 months and 2 years of age, black and Hispanic children were sleeping less than their white counterparts. After 2 years old, black and Hispanic children were much more likely to have a television in their bedroom and had higher consumption of sugar-sweetened beverages and fast food (Table 4, Fig 1).

Some risk factors were associated with only 1 minority group. Hispanic but not Black mothers had elevated odds of gestational diabetes (Table 4). Black children had higher cord blood leptin levels, viewed greater hours of television from 6 months to 2 years of age, and consumed fewer meals together with their families. In contrast to these risk factors that might raise the risk for obesity, black and Hispanic women had lower odds of excess GWG. Black women also had lower odds of smoking during pregnancy (Table 4, Fig 1).

DISCUSSION

In this prebirth, prospective cohort of mother–child pairs, we found racial/ethnic differences in many early-life risk factors for childhood obesity. Although adjustment for socioeconomic

### TABLE 4

<table>
<thead>
<tr>
<th>Risk Factors for Childhood Obesity</th>
<th>Excessive GWG*</th>
<th>Gestational diabetes</th>
<th>Mother smoked during early pregnancy</th>
<th>Maternal antenatal depression</th>
<th>Fetal growth</th>
<th>Infant feeding</th>
<th>Parental BMI</th>
<th>SES Adjusted</th>
<th>Parental BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black, Non-Hispanic, OR (95% CI)</td>
<td>0.85 (0.67–1.08)</td>
<td>1.18 (0.70–1.99)</td>
<td>1.15 (0.79–1.66)</td>
<td>2.31 (1.47–3.62)</td>
<td>0.51 (0.34–0.76)</td>
<td>2.20 (1.42–3.42)</td>
<td>2.35 (1.83–3.39)</td>
<td>0.93 (0.66–1.31)</td>
<td>2.35 (1.57–3.52)</td>
</tr>
<tr>
<td>Hispanic or Latino, OR (95% CI)</td>
<td>0.76 (0.52–1.10)</td>
<td>2.57 (1.49–4.66)</td>
<td>2.08 (1.29–3.34)</td>
<td>2.96 (1.55–5.62)</td>
<td>0.40 (0.20–0.81)</td>
<td>2.05 (1.07–3.93)</td>
<td>2.14 (1.50–3.44)</td>
<td>0.88 (0.51–1.51)</td>
<td>3.80 (1.98–7.32)</td>
</tr>
</tbody>
</table>

Crude models are adjusted for child gender only. SES models are additionally adjusted for maternal age, education, and parity and household income. Parental BMI models are additionally adjusted for maternal and paternal prepregnancy BMI. Reference group for all models is white children. Models that predicted maternal risk factors are not adjusted for paternal BMI. OR indicates odds ratio; CI, confidence interval.

*Models that predicted excessive GWG are additionally adjusted for total length of gestation.
status (SES) and maternal and paternal obesity attenuated many of the observed relationships, independent of SES and parental obesity, black and Hispanic children had increased odds of rapid infant weight gain, greater maternal control of infant feeding, shorter sleep duration during infancy, more televisions in bedrooms, higher sugar-sweetened beverage intake, and higher intake of fast food compared with white children.

Our findings are consistent with previous studies of racial/ethnic differences in pregnancy-related risk factors for childhood obesity.\textsuperscript{16,47–49} Consistent with a national study by Chu et al,\textsuperscript{47} black and Hispanic women in our study were more likely than white women to begin their pregnancies already overweight or obese and gained less weight during pregnancy. Similar to a previous study in New York City,\textsuperscript{48} we found that Hispanic women had a higher risk for gestational diabetes. In contrast to other studies, however, we did not find that black women had an increased risk for gestational diabetes.\textsuperscript{50} Few studies have examined racial/ethnic differences in smoking during pregnancy\textsuperscript{43} and in maternal antenatal depression.\textsuperscript{16} In our study, the observed racial/ethnic differences in both smoking during pregnancy and antenatal depression were largely explained by socioeconomic factors, including maternal education and household income.

Our findings extend to infancy and early childhood previous studies of older children that have found higher prevalence of obesity-related risk factors among racial/ethnic minorities. Previous studies have found higher levels of television viewing and more televisions in bedrooms,\textsuperscript{9} higher consumption of sugar-sweetened beverages,\textsuperscript{51} increased fast food consumption,\textsuperscript{52} and lower levels of physical activity among black and Hispanic youth compared with white youth.\textsuperscript{53,54} In addition, previous studies have observed racial/ethnic differences in maternal feeding practices and beliefs.\textsuperscript{55,56} To our knowledge, no published studies have examined racial/ethnic differences in rapid infant weight gain, sleep duration in infancy, or fast food consumption in early childhood.

Overall, our findings suggest that racial/ethnic disparities in childhood obesity may be determined by factors that operate in pregnancy, infancy, and early childhood. These factors may include differences in behaviors such as diet patterns and physical activity or differences in access to and use of pregnancy- or infancy-related health care. It is also possible that cumulative disadvantage, or “weathering,”\textsuperscript{57} even before conception, among racial/ethnic minority mother–child pairs may explain the observed disparities. Additional studies should examine these possibilities. Our study also suggests that interventions to modify early-life risk factors may have a substantial impact on reducing disparities in childhood obesity prevalence and its re-
Strengths of our study included having prospectively collected data on a wide range of risk factors that extend from pregnancy to early childhood and the ability to adjust for several important confounding factors, including parental obesity. The study also had several potential limitations. First, most of our measures were from self-report, including prepregnancy maternal weight, smoking, breastfeeding, and infant sleep, and loss to follow-up was not random. These factors could have introduced bias toward the null. Second, the educational and income levels of our study population were relatively high. Because some of the associations between race/ethnicity and obesity risk factors seemed to depend on SES, our results may not be generalizable to more socioeconomically disadvantaged populations. Although we studied many risk factors for childhood obesity that had plausible hypotheses, we did not measure others, such as lifestyle or cultural determinants of dietary and sedentary practices. Third, we did not have enough power to examine potentially important interactions between race/ethnicity and SES.

CONCLUSIONS

We found that from pregnancy to the preschool period, many risk factors for child obesity are more prevalent among black and Hispanic children than among white children. These differences may very well explain the observed racial/ethnic differences in prevalence of obesity in young children. Although more studies are needed to understand the underlying reasons for the development of these disparities, our findings provide a strong rationale for testing comprehensive interventions in early life to reduce disparities in obesity prevalence.

ACKNOWLEDGMENTS

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*Teen Use of Online Devices: Can It Grow More Than It Already Has?:* If you thought six and a half hours a day of smart phone use, watching videos, listening to music, texting, and surfing the internet is already a shocking amount of time for teens to be using technology on a daily basis, think again. According to a study from the Kaiser Family Foundation, as reported in The New York Times (Lewin T, January 20, 2010), teens 8 to 18 are now spending more than 7 1/2 hours a day interfacing with technology, up by an hour since the same survey was conducted five years ago. In addition, if you take into account the multitasking of listening to music while also using the internet, this number may be as high as 11 hours of media content being crammed into the 7 1/2 hours. Whether you believe this much technology use is good or bad for our teens (and studies argue both sides of this question), according to Dr. Michael Rich, director of the Center on Media and Child Health at Children’s Hospital Boston, the use of media is so ubiquitous, we just need to accept that it is occurring to our children and teens “like the air they breathe, the water they drink and the food they eat.”

Noted by JFL, MD
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