EXPOSURE TO MATERNAL DEPRESSION DURING EARLY CHILDHOOD AND RISK FOR CHILDHOOD OVERWEIGHT

by

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Abstract

**Background:** Childhood obesity has tripled in the United States between 1963 and 2008 and is an important public health issue. Recently maternal depression has been explored as a possible risk factor for childhood obesity, but the results have been inconsistent. These inconsistencies might be related to methodological issues, specifically, the use of one measurement of depression.

**Objective:** To evaluate the relationship between multiple exposures to maternal depression from age 3 to age 5 and a child’s risk for being overweight at age 7.

**Methods:** We used data from the Early Steps Project, a longitudinal study exploring the impact of the Family Check Up on a child’s risk for behavior problems. Maternal depression was assessed at age 3, 4, and 5 with the Center for Epidemiologic Studies Depression Scale (CES-D). Elevated maternal symptomology was determined by a CESD score > 27. Childhood BMI was measured at age 7. Childhood overweight was determined by a BMI z score >= 85%. Logistic regression was used to explore the relationship between the total number of exposures between age 3 and 5 and risk for childhood overweight at age 7.
**Results:** 442 children had consistent female caregivers at all assessment and BMI assessed at age 7 and were included in the study. 68.6% of the children had no exposures to maternal depression, 17.2% had 1 exposure, 9.7% had 2 exposures, and 4.5% had 3 exposures. 34.0% of those with no exposures were overweight, 23.7% of those with 1 exposure were overweight, 39.5% of those with 2 exposures were overweight, and 55.0% of those with 3 exposures where overweight. Logistic regression was used to explore the relationship between multiple exposures (ages 3-5) to maternal depression and childhood overweight at age 7. Only those exposed 3 times were at a greater risk childhood overweight when compared to children with no exposures (OR = 2.58 95% CI = 1.01-6.61).

**Conclusions:** Exposure to maternal depression is related to an increase in risk for childhood overweight, but only when it is chronic. Interventions should incorporate maternal mental health when dealing with childhood obesity.
LIST OF TABLES

Table 1. Sample characteristics and relationships to outcome and exposure variables 19
Table 2. Difference in recruitment site between samples selected and not selected…20
Table 3. Means and prevalence of maternal depressive symptoms…………………….21
Table 4. Number of exposures to elevated maternal depressive symptoms from ages 3 to 5 and prevalence of child being overweight at age 7.................................................................22
Table 5. Association of the number of exposures to elevated maternal depressive symptoms (CES-D >= 27) and childhood overweight.................................................................23
INTRODUCTION

Childhood obesity in the United States is on the rise. Between 1971-1974 and 2007-2008 the rate of childhood obesity tripled. In children 2 to 5 years old, it rose from 5.0% to 10.4%, in children 6 to 11 it rose from 4.0% to 19.6%, and among adolescents 12 to 19 it rose from 6.1% to 18.1%\textsuperscript{1}. It has become so prevalent that in 2002 the U.S. Surgeon General declared childhood obesity to be a national epidemic. If obesity is left unchecked, some have predicted life expectancy in the US could drop for the first time in generations\textsuperscript{2}. While some have dismissed this pessimistic view as “scaremongering”\textsuperscript{3}, and projections still predict increasing life spans for those born until 2020\textsuperscript{4}, the idea highlights a very real issue. Childhood obesity is a problem the United States needs to address.

Childhood obesity can have psychological, physical, and social ramifications. Obese children are more at risk for orthopedic, neurological, gastroenterological, and endocrine conditions\textsuperscript{5}. They are more likely to experience type 2 diabetes and have more cardiovascular risk factors (e.g., high blood pressure) than their normal weight peers\textsuperscript{6}.

Psychologically, children who are obese report more negative self-perceptions, lower self-worth\textsuperscript{7}, and higher dissatisfaction with their physical appearance\textsuperscript{8} relative to
children who are not obese. Obesity has also been associated with elevated rates of behavioral and emotional problems (such as aggressive and anxious/depressed behavior)\textsuperscript{8-9} and having lower grades and higher rates of absenteeism in school\textsuperscript{10-14}.

Children who are obese are also more likely to become obese adults, with the risk growing linearly with the child’s obesity\textsuperscript{15}. People who are chronically overweight from a young age are more likely not to pursue further education after high school, to be single, and to receive unemployment or welfare at age 40 than those who gain weight later in life\textsuperscript{16}.

With childhood obesity becoming such a problem, it is important that we understand its causes to inform both prevention and early interventions before obesity occurs. Once an obese person achieves a healthy weight, it is a life long struggle to maintain it, most often with lackluster outcomes\textsuperscript{17}. A summary of the Consensus Development Conference sponsored by the NIH Nutrition Coordinating Committee found that after 1 year, one third to two thirds of those who lost the weight on their own will gain it back. After 5 years, almost all will have regained the weight\textsuperscript{18}.

Weight gain is caused by an imbalance in calorie intake and expenditure\textsuperscript{19}. Children who are more sedentary and who consume nutrient-poor energy are at increased risk for obesity\textsuperscript{19-25}.

Many factors can influence a child's weight. The prevalence of obesity in the period 2007-2008 among minorities was higher than non-Hispanic Caucasians\textsuperscript{1}. A study using 8270 children from the National Longitudinal Survey of Youth found that between 1986 and 1998 childhood overweight (a BMI greater than the 85\textsuperscript{th} percentile) increased in African Americans and Hispanic children but not among Caucasian children\textsuperscript{26}.
However, this disparity could be diminishing because of a rapid increase in obesity in Caucasian children in the early half of the 2000s\textsuperscript{27}. The higher prevalence of obesity in African American and Hispanic children could be caused by a mix of genetic, socioeconomic, environmental, and cultural differences\textsuperscript{27-28}.

Socioeconomic disparities have a more complex relationship with childhood obesity. In the US, without stratifying for race, low-income children are at a higher risk for obesity compared to those living in higher income homes\textsuperscript{29-30}. But when stratified by race, higher income may be a protective factor for Caucasians while increasing the risk in African Americans\textsuperscript{26,31}. Other studies have found contrasting and mixed results, associating low income with childhood obesity only in white children, while finding no relationship between household income and childhood obesity for African Americans and Hispanics\textsuperscript{32}. Education, as an indicator of SES, has a more consistent relationship with childhood obesity. In an analysis of 20 cross-sectional studies 75\% of the studies showed an inverse relationship between a parent’s education and childhood obesity\textsuperscript{33}. Freedman et al. found similar results for parental education and suggested education is more stable than income as a marker for SES\textsuperscript{31}. Some of the underlying reasons for this relationship involve children in low SES households having less access to physical activities and healthier foods, and having fewer resources to devote to healthier foods\textsuperscript{28,32-33}.

Recent research has evaluated maternal depression as another possible risk factor for childhood overweight and obesity with varying results. Some studies have found that maternal depression is associated with a child’s risk of obesity\textsuperscript{36-37}. For example, a cross-sectional study of 589 mother-child dyads found that children ages 18
to 24 months with depressed mothers are 2.3 times as likely to be obese when compared to those whose mothers are not depressed\textsuperscript{38}. However some studies have found no association between maternal depression and childhood obesity\textsuperscript{39-41}. Interestingly, other studies have even found an opposite effect: maternal depression was associated with children being underweight\textsuperscript{42-43}. Surprisingly, sometimes all three results were observed in the same study. In a sample with age periods comparable to those in the current study, reported that children in kindergarten with depressed mothers had different outcomes based on age of BMI assessment and gender\textsuperscript{44}. In girls, exposure to maternal depression while in kindergarten was related to lower BMI in third grade and to an increase in BMI when in the fifth grade. For boys, exposure to maternal depression in kindergarten was related to higher BMI in fifth grade, but had no relation to BMI in third grade. When maternal depression was measured again in 3\textsuperscript{rd} grade, there was a positive relationship with BMI in girls, but no relation in boys. A review of 5 studies exploring maternal depression and childhood obesity found similar mixed results\textsuperscript{46}.

The inconsistencies in these results could be due to study limitations and methodological differences. Most studies only used one measurement of maternal depression in their analyses, which does not take into account the impact of duration or recurrence of depression on the risk for obesity in children. Depression varies greatly from person to person\textsuperscript{46-50}. Mothers classified as depressed by a single measurement might be in their first and only depressive episode or they might be in the midst of a five year struggle with major depressive disorder.
Little research has focused on the relationship between multiple exposures to maternal depression and childhood overweight and obesity, and the existing results are conflicting. Wang et. al. found children having three exposures to previous maternal depression (at ages 1 month, 24 months, and 36 months) were at greater risk for later childhood overweight (OR =1.88 at grade 1 and 3, OR = 2.01 at grade 6) than those with no exposures. Santos et. al. reported the number of exposures to maternal depression (at ages 3, 12, 24, and 48 months) was not associated with the risk for childhood obesity at 48 months.

The aim of this study is to examine the relationship between early childhood exposure to maternal depression and later childhood overweight and whether the association varies by the number of exposures. It is hypothesized that children whose mothers demonstrate elevated levels of depressive symptoms at multiple time points during early childhood are at higher risk to be overweight later in childhood than children whose mothers did not have elevated depressive symptoms. We hypothesize also a dose-response relationship between number of depressive episodes and childhood overweight that will be independent of measured confounders.
METHODS

Participants and Procedures

The Early Steps Project is a longitudinal study exploring the effectiveness of the Family Check-Up, a family based and ecological preventive intervention for children at risk for problem behavior. 731 mother-child dyads were recruited in Pittsburgh, PA; Eugene, OR; and Charlottesville, VA from 2002-2003. One thousand six hundred sixty-six potential participants were approached in Women, Infant, and Children (WIC) sites if they had a child between 2 years old and 2 years and 11 months old. Participants were eligible if they met the risk requirements for 2 of the 3 domains for future child behavioral problems: sociodemographic risk factors (low family income and education), family risk factors (maternal depression, daily family challenges, substance use problems, teen parent status), and/or child risk factors (conduct problems, high conflict relationship with adults). Risk was determined by having 1 SD or above the normative averages for continuous measures (e.g., maternal depression measured with the Center of Epidemiological Studies on Depression scale) or the presence of a risk factor (e.g., being a teen parent) for at least one measure for a given domain. 879 families met the eligibility requirements with 731 (83.2%) agreeing to participate. 272 (37.2%) were recruited in Pittsburgh, 271 (37.1%) in Eugene, and 188 (25.7%) in Charlottesville.
Data were gathered by research assistants in home assessments at ages 2, 3, 4, 5, and 7. At these assessments, primary caregivers (those who had legal custody of the child), and, if available, alternate caregivers (those who the primary caregivers indicated assisted in raising the child) participated in interactive tasks with the child and completed questionnaires. All families were randomly assigned to either the treatment group, where they received the opportunity to participate in the Family Check-Up, or the control group.

Of the original 731 families, 659 (90.2%) participated in the age 3 visit, 619 (84.7) participated in the age 4 visit, 622 (85.1%) children participated in the age 5 visit, and 568 (77.7%) participated in the age 7 visit. Of those who participated in the age 7 visit, 516 (70.6%) children had BMI measured. For this study, maternal caregivers were defined as females who had primary custody of the child at home assessments. Eligibility for the current analyses included having a consistent maternal caregiver at the study assessments with depressive symptomology measured at a minimum of twice from age 3 to 5 and having child BMI assessed at age 7. Four hundred fifty two (60.5%) families met these requirements and were included in the analyses (436 biological mothers and 6 other female caregivers).

Measures
Exposure Variables

Maternal depressive symptomology (MatD) was assessed using the Center of Epidemiological Studies on Depression (CES-D) a widely used self-administered measure that has been shown to be a reliable indicator of possible clinical depression.
Devin et al. found the CES-D to have an internal consistency ranging from 0.63 to 0.93 and a test-retest reliability of 0.61 after 3 months across varied populations. This measure has twenty items with values ranging from 0 (0-1 days a week) to 3 (5+ days a week). Total scores range from 0 to 60. MatD was assessed at ages 2, 3, 4, 5 and 7.

Two cut offs were considered to indicate elevated MatD in the analyses: 16, the more frequently used cutoff and a more stringent cutoff of 27 (~1 SD above the mean CESD score at study entry). This second cutoff was used to account for the larger proportion of participants with a score of 16 or more than in the general population, as elevated maternal depressive symptoms were a possible eligibility requirement. The number of exposures to previous MatD was the sum of exposures from age 3 to 5, ranging from 0 to 3. Age 2 MatD was excluded in the sum of total exposures because of the small proportion of children with 4 exposures. Selecting only three measures of MatD, as opposed to combining exposure levels, retained the chronic aspect of being in the highest exposure group.

Outcome Variables

Child’s height and weight were measured by trained research assistants at the age 7 assessment with a scale and stadiometer. BMI scores were calculated as weight in kilograms divided by height in meters squared. BMI z scores were determined with the CDC BMI-for-age growth charts for the United States using child age in months during the age 7 assessment. Childhood overweight (childhood overweight) at age 7 was defined as a BMI z-score >= 85th percentile.
Covariates

Child’s gender and race were reported by primary caregivers at study entry. Race was coded into 3 groups (Non-Hispanic Caucasian, Non-Hispanic African American, and other). Household income, maternal education, maternal marital status, and child’s age in months were assessed at the age 7 assessment. Family income was coded into 10,000 dollar increments from 0 to 29,999 and 30,000+ dollars per year. Maternal education was coded into 3 groups (Less than high school completed, high school completed/GED, and post high school training or schooling). Marital status was coded into married or single. Whether or not the families were assigned to the treatment group was coded dichotomously (yes/no) based on group assignment regardless if treatment occurred.

Statistical Analyses

Simple statistics (means, standard deviations, and frequencies) were used to describe MatD, childhood overweight, and covariates. Chi-square and student t-tests as appropriate were used to compare the characteristics of subjects included in the analyses (n=442) and those not (n=289).

Seven measures of MatD were missing at age 3, 11 at age 4, and 10 at age 5. The mean of available assessments of MatD were substituted for the missing data points. Two measures of household income were missing and replaced with household income assessed at age 5.

Logistic and ordinal regression were used to explore the univariate relationships between covariates and both exposure and outcome variables.
Child race, child gender, child age in months, SES, and maternal marital status were selected for the adjusted model a priori, based on previous literature. Because of the small sample size and the desire for a parsimonious model, only household income was selected as the indicator of SES because of its relationship with MatD in our sample. Recruitment site and tx group were added to the adjusted model as possible confounders.

Logistic regression was used to estimate the unadjusted and adjusted relationships between individual exposures to MatD (at ages 3, 4, 5 and 7) and childhood overweight, as well as the number of exposures to MatD from age 3 to 5 and childhood overweight.
RESULTS

Sample Characteristics

In the sample included in the analyses, the average child age was 91.3 months (SD = 2.18) at BMI measurement. The participating children were predominantly non-Hispanic Caucasians (44.8%) and about half were male. The sample was mostly socioeconomically disadvantaged families, with less than 40% of the household incomes being above thirty thousand dollars per year and half of the maternal caregivers having only a high school education or less. Less than half of the maternal caregivers were married. Of the sample, 40.5% was recruited in Eugene, OR while only 23.3% was recruited in Charlottesville, VA. See Table 1. There were no statistical differences between the sample used (n= 442) and those not used (N = 289) except for recruitment site $X^2 (2, N = 731) = 6.38, p = 0.04$. See Table 2.

Table 3 shows the mean number of depressive symptoms for each assessment age and the prevalence of scores 16 or higher and 27 or higher. The mean CESD score declined year to year from 15.99 (SD = 11.00) at age 3 to 14.74 (SD = 11.07) at age 7. The proportion of scores $\geq 16$ ranged from 43.2% at age 3 to 37.6% at age 7, while the proportion of scores $\geq 27$ ranged from 18.1% at age 3 to 15.6% at age 7.

Table 4 shows the proportion of children at each MatD exposure level and the prevalence of childhood overweight. Using the 16 cutoff, 37.3% of the children were not exposed, 23.8% had 1 exposure, 18.8% had 2, and 20.1% had 3. The prevalence of
childhood overweight at age 7 was fairly consistent among the exposure levels, ranging from 29.5% for 1 exposure to 35.8% for 0 exposures. Using the 27 cutoff, 68.6% were not exposed to MatD, 17.2% were exposed 1 time, 9.7% were exposed 2 times, and 4.5% were exposed 3 times. 34.0% of children not exposed to MatD were overweight at age 7, 23.7% of children exposed 1 time, 39.5% of children exposed 2 times, and 55.0% of children exposed 3 times.

Analyses

Because no relationships were found between exposures to MatD and childhood overweight using a CESD cutoff score of 16, for the remainder of this paper MatD will only be referring to a cutoff of 27.

In the unadjusted models, no single measure of MatD (at ages 2, 3, 4, 5, and 7) was associated with childhood overweight at age 7. Additionally, there was not a significant relationship between the number of exposures to MatD (age 3 – 5) and childhood overweight.

In the adjusted models, single measures of MatD continued to have no relationship with childhood overweight. Children with 3 exposures to MatD were 2.58 times as likely to be overweight (OR = 2.58, 95% CI = 1.01 – 6.61) than children with no exposures, while having 1 or 2 exposures was not associated with the risk for childhood overweight. Females were 1.75 times as likely to be overweight, (OR = 1.75, 95% CI = 1.16-2.65). Child’s age in months was positively related to risk for overweight (OR = 1.10, 95% CI = 1.00-1.20). See Table 6.
DISCUSSION

We found that children who were exposed to elevated maternal depressive symptoms 3 times at ages 3, 4, and 5 were at a higher risk for being overweight at age 7 compared to those never exposed. Contrary to our hypothesis, there was not a dose-response relationship. Additionally, no single exposure to MatD from age 3 to 7 was significantly related to childhood overweight.

These results provide evidence that exposure to maternal depressive symptoms in early childhood might be an important factor in a child’s risk for overweight at age 7, but only when it is persistent, supporting the findings of Wang et al. However, the study results are counter to those found by Santos et al. discussed earlier. The observed differences could be related to the age of the children during the BMI assessment. The prevalence of childhood obesity is significantly higher in children and adolescents ages 6 to 19 than children 2 to 5. Wang et al.’s and our study assessed BMI during ages with a higher risk of childhood obesity, while Santos et al. assessed child’s BMI at age 4, when obesity is less prevalent. There could also be a delay in the relationship between multiple exposures to MatD and a child’s risk of being overweight or obese. Santos et al. measured child weight concurrently with the last measure of MatD while both Wang’s and our study assessed child weight years later. Considering both of these possibilities, had BMI been assessed again when the children were older, Santos et al. might have found results similar to ours.

It should be noted that this relationship was found using a CES-D cutoff of 27, but not a cutoff of 16. This suggests that the relationship between MatD and childhood
overweight may be stronger with higher levels of depressive symptomology. Using a higher cut off is not without precedent. Schulberg et al. found a score of 27 to be an effective cut off for Muscular Sclerosis patients with a sensitivity of 89% and a specificity of 70%\textsuperscript{59}, while other researchers have suggested using higher cutoffs to reduce false positives\textsuperscript{60-61}.

There are several possible ways in which MatD may influence childhood overweight. Depression can have profound effects on a person’s ability to parent, including how involved they are in their children’s lives. Mothers with depression are less engaged with and less responsive to their children than mothers who are not depressed\textsuperscript{62-64}. Their children also tend to participate in more sedentary activities, participate in fewer organized activities\textsuperscript{65-68}, and be fed less healthy foods\textsuperscript{69-71}. Parenting quality and it’s relation to activity levels and food consumption could be a mechanism by which MatD influences a child’s risk for being overweight or obese. McConley et al. explored this possible pathway and showed that depressed mothers were more likely to report lower parenting quality, which was related to lower activity levels, more sedentary behavior, and less healthy food consumption in their children. Activity levels and sedentary behavior were then found to be associated with higher BMI, accounting for the relationship between MatD and BMI\textsuperscript{36}. In contrast, unhealthy food was not. Similarly, Duarte et al. explored physical activity, TV watching, and healthy eating as possible pathways between MatD and a child’s BMI. Results indicated TV viewing and low levels of physical activities explained the relationship between MatD and BMI in girls, while unhealthy eating habits explained the relationship in boys\textsuperscript{44}. But
no study has explored such mediators using multiple exposures to MatD as a single factor and child BMI; future studies need to address this.

In the current study, we did not have data on childhood behaviors that might explain the extent to which persistent or recurrent maternal depression is associated with childhood overweight. We also lacked information on parental BMI, an established risk factor for childhood obesity. Many studies have shown a strong relationship between parent and child weight, particularly maternal BMI. These variables are of crucial importance because of their association with depression in addition to childhood obesity. Women who are depressed are at a higher risk of being obese. Maternal BMI could, at least partially, explain the relationship between maternal depression and a child's weight.

Several other limitations are noteworthy. As discussed earlier, SES (e.g. household income and maternal education) has been found to be associated with a child's weight, but wasn't significantly related to childhood overweight in our sample. Because our participants were predominantly of low SES, we may not have had the variation in household income and maternal education to assess these potential associations. Therefore, we can only generalize results to lower SES populations.

Our study had important strengths. It prospectively obtained data on MatD, including MatD concurrent with BMI assessment. While research exploring the relationship between current MatD and childhood obesity has yielded inconsistent results, MatD has not been ruled out as a possible risk factor. If current MatD does have a relationship with a child's risk for obesity, repeated exposures to past MatD could merely be predicting risk for current MatD and its relation to childhood obesity. By
exploring the relationship between current MatD and childhood overweight in addition to the number of exposures to past MatD, we have more confidence in our results.

The inclusion of region as a possible confounder is unique to our study. The Early Steps Project collected data from three geographically diverse areas (Pittsburgh, PA; Eugene, OR; and Charlottesville, VA), and research has found the risk for childhood obesity differs regionally across the United States\textsuperscript{78}.

While having a sample consisting predominantly of low SES children has limited our generalizability, it has honed in on a population vulnerable for childhood obesity. As previously discussed, lower household income and maternal education have been associated with higher risk for childhood obesity.
CONCLUSION

Maternal depression may play a part in a child’s risk of being overweight, but only when the exposure is chronic. Further research is needed, particularly studies including important variables such as parental BMI, and child food consumption, activity levels, and sedentary behavior. Regardless, these results suggest that maternal mental health should be considered when developing interventions to prevent childhood obesity.
APPENDIX A

TABLES REFERENCED
<table>
<thead>
<tr>
<th>Characteristic</th>
<th># of MATD exposures</th>
<th>COW</th>
<th>OR (95% CI)</th>
<th>OR (95% CI)</th>
</tr>
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<tbody>
<tr>
<td>Child age in months (SD)</td>
<td></td>
<td></td>
<td>1.09 (1.01-1.19)$^a$</td>
<td>1.03 (0.95-1.13)</td>
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<tr>
<td>Child race (%)</td>
<td></td>
<td></td>
<td>1.60 (1.01-2.56)$^a$</td>
<td>0.68 (0.48-1.15)</td>
</tr>
<tr>
<td>Non-Hispanic Caucasian</td>
<td>198 (44.8)</td>
<td></td>
<td>1 (referent)</td>
<td>1 (referent)</td>
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<tr>
<td>Non-Hispanic African American</td>
<td>122 (27.6)</td>
<td></td>
<td>1.47 (0.92-2.37)</td>
<td>0.67 (0.41-1.09)$^b$</td>
</tr>
<tr>
<td>Other</td>
<td>122 (27.6)</td>
<td></td>
<td>1.47 (0.92-2.37)</td>
<td>0.67 (0.41-1.09)$^b$</td>
</tr>
<tr>
<td>Child gender (%)</td>
<td></td>
<td></td>
<td>1.83 (1.23-2.71)$^a$</td>
<td>1.22 (0.83-1.80)</td>
</tr>
<tr>
<td>Male</td>
<td>220 (49.8)</td>
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<td>1 (referent)</td>
</tr>
<tr>
<td>Female</td>
<td>222 (50.2)</td>
<td></td>
<td>1.83 (1.23-2.71)$^a$</td>
<td>1.22 (0.83-1.80)</td>
</tr>
<tr>
<td>Household income per year (%)</td>
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<td></td>
<td>0.77 (0.41-1.48)</td>
<td>0.64 (0.34-1.20)</td>
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<tr>
<td>&lt;10k</td>
<td>60 (13.6)</td>
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<td>10 – 19.9k</td>
<td>113 (25.6)</td>
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<td>0.85 (0.48-1.64)</td>
<td>0.42 (0.22-0.79)$^a$</td>
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<td>20 – 29.9k</td>
<td>100 (22.6)</td>
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<td>0.85 (0.48-1.64)</td>
<td>0.42 (0.22-0.79)$^a$</td>
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<tr>
<td>30 – 39.9k</td>
<td>169 (38.2)</td>
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<td>0.69 (0.37-1.49)</td>
<td>0.50 (0.26-0.98)$^a$</td>
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<td>Maternal marital status (%)</td>
<td></td>
<td></td>
<td>0.75 (0.7-0.99)$^a$</td>
<td>1.30 (0.99-1.70)$^b$</td>
</tr>
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<td>Single</td>
<td>250 (56.6)</td>
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<td>1 (referent)</td>
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<td>Married</td>
<td>192 (43.4)</td>
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<td>0.75 (0.7-0.99)$^a$</td>
<td>1.30 (0.99-1.70)$^b$</td>
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<tr>
<td>Maternal Education (%)</td>
<td></td>
<td></td>
<td>1.08 (0.60-1.95)</td>
<td>1.04 (0.62-1.73)</td>
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<td>&lt; HS</td>
<td>74 (16.7)</td>
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<td>1.08 (0.60-1.95)</td>
<td>1.04 (0.62-1.73)</td>
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<td>&gt;HS</td>
<td>223 (50.5)</td>
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<td>0.99 (0.57-1.73)</td>
<td>0.95 (0.55-1.64)</td>
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<td>Site (%)</td>
<td></td>
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<td>1 (referent)</td>
<td>1 (referent)</td>
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<td>Eugene, OR</td>
<td>179 (40.5)</td>
<td></td>
<td>1 (referent)</td>
<td>1 (referent)</td>
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<td>Charlottesville, VA</td>
<td>103 (23.3)</td>
<td></td>
<td>1.42 (0.86-2.35)</td>
<td>1.07 (0.63-1.82)</td>
</tr>
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<td>Pittsburgh, PA</td>
<td>160 (36.2)</td>
<td></td>
<td>1.42 (0.86-2.35)</td>
<td>1.07 (0.63-1.82)</td>
</tr>
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<td>Group (%)</td>
<td></td>
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<td>1 (referent)</td>
<td>1 (referent)</td>
</tr>
<tr>
<td>Control</td>
<td>225 (50.9)</td>
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<td>1 (referent)</td>
<td>1 (referent)</td>
</tr>
<tr>
<td>Treatment</td>
<td>217 (49.1)</td>
<td></td>
<td>0.97 (0.64-1.41)</td>
<td>1.01 (0.68-1.49)</td>
</tr>
</tbody>
</table>

$^a$ Significant at a p value of 0.05

$^b$ Marginally significant at a p value of 0.10
<table>
<thead>
<tr>
<th>Recruitment Site</th>
<th>Sample selected (N = 442)</th>
<th>Sample not selected (N = 289)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eugene, OR</td>
<td>179 (40.4)</td>
<td>92 (31.8)</td>
<td>0.04</td>
</tr>
<tr>
<td>Charlottesville, VA</td>
<td>103 (23.3)</td>
<td>85 (29.4)</td>
<td></td>
</tr>
<tr>
<td>Pittsburgh, PA</td>
<td>160 (36.2)</td>
<td>112 (38.8)</td>
<td></td>
</tr>
</tbody>
</table>
Table 3. Means and prevalence of maternal depressive symptoms

<table>
<thead>
<tr>
<th>Age</th>
<th>Mean (SD)</th>
<th>N &gt;=27 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age 3</td>
<td>15.99 (11.00)</td>
<td>80 (18.1)</td>
</tr>
<tr>
<td>Age 4</td>
<td>15.27 (11.01)</td>
<td>72 (16.3)</td>
</tr>
<tr>
<td>Age 5</td>
<td>14.81 (11.19)</td>
<td>70 (15.8)</td>
</tr>
<tr>
<td>Age 7</td>
<td>14.74 (11.07)</td>
<td>69 (15.6)</td>
</tr>
</tbody>
</table>
Table 4. Number of exposures to elevated maternal depressive symptoms from ages 3 to 5 and prevalence of child being overweight at age 7

<table>
<thead>
<tr>
<th># of exposures</th>
<th>N   (% )</th>
<th>Prevalence of childhood overweight (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>303 (68.6)</td>
<td>103 (34.0)</td>
</tr>
<tr>
<td>1</td>
<td>76 (17.2)</td>
<td>18 (23.7)</td>
</tr>
<tr>
<td>2</td>
<td>43 (9.7)</td>
<td>17 (39.5)</td>
</tr>
<tr>
<td>3</td>
<td>20 (4.5)</td>
<td>11 (55.0)</td>
</tr>
<tr>
<td></td>
<td>Unadjusted Model</td>
<td>Adjusted Model</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>------------------</td>
<td>----------------</td>
</tr>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>OR (95% CI)</td>
</tr>
<tr>
<td><strong># of exposures (ages 3-5)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>1 (referent)</td>
<td>1 (referent)</td>
</tr>
<tr>
<td>1</td>
<td>0.60 (0.34-1.08)</td>
<td>0.58 (0.32-1.05)</td>
</tr>
<tr>
<td>2</td>
<td>1.27 (0.66-2.45)</td>
<td>1.29 (0.62-2.48)</td>
</tr>
<tr>
<td>3</td>
<td>2.37 (0.95-5.91)</td>
<td>2.58 (1.01-6.61)</td>
</tr>
<tr>
<td>Child age in months</td>
<td>*</td>
<td>1.10 (1.00-1.20)</td>
</tr>
<tr>
<td>Child gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>*</td>
<td>1 (referent)</td>
</tr>
<tr>
<td>Female</td>
<td>*</td>
<td>1.75 (1.16-2.65)</td>
</tr>
<tr>
<td>Child race</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic Caucasian</td>
<td>*</td>
<td>1 (referent)</td>
</tr>
<tr>
<td>Non-Hispanic African American</td>
<td>*</td>
<td>1.62 (0.88-2.97)</td>
</tr>
<tr>
<td>Other</td>
<td>*</td>
<td>1.41 (0.85-2.35)</td>
</tr>
<tr>
<td>Maternal marital status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>*</td>
<td>1 (referent)</td>
</tr>
<tr>
<td>Married</td>
<td>*</td>
<td>1.25 (0.77-2.02)</td>
</tr>
<tr>
<td>Annual household income</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;10,000$</td>
<td>*</td>
<td>1 (referent)</td>
</tr>
<tr>
<td>10,000 – 19,999</td>
<td>*</td>
<td>0.93 (0.46-1.86)</td>
</tr>
<tr>
<td>20,000 – 29,999</td>
<td>*</td>
<td>1.06 (0.51-2.20)</td>
</tr>
<tr>
<td>30,000+</td>
<td>*</td>
<td>0.98 (0.49-1.96)</td>
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<tr>
<td>Recruitment site</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eugene, OR</td>
<td>*</td>
<td>1 (referent)</td>
</tr>
<tr>
<td>Charlottesville, VA</td>
<td>*</td>
<td>1.23 (0.70-2.14)</td>
</tr>
<tr>
<td>Pittsburgh, PA</td>
<td>*</td>
<td>1.09 (0.62-1.90)</td>
</tr>
<tr>
<td>Treatment Assignment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>*</td>
<td>1 (referent)</td>
</tr>
<tr>
<td>Treatment</td>
<td>*</td>
<td>0.95 (0.63-1.43)</td>
</tr>
</tbody>
</table>
BIBLIOGRAPHY


