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Do stressed mothers have heavier children?: A meta-analysis on the relationship between maternal stress and child body mass index

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Abstract

Child obesity continues to be a prevalent public health issue. This meta-analysis synthesized 17 studies investigating the association between levels of psychological stress experienced by mothers and the body mass index of their children. The overall standardized mean difference effect size was positive and significantly different from zero in cross-sectional $d = 0.20$ [$k = 14$, 95% Confidence Interval (CI): 0.06, 0.34] and longitudinal studies $d = 0.18$ ($k = 5$, 95% CI: 0.00, 0.351), and had significant heterogeneity in both [cross-sectional, $Q(13) = 193.00$, $p < 0.001$; longitudinal, $Q(4) = 29.46$, $p < 0.001$]. In longitudinal studies, effect sizes were larger when children also would have experienced the stressor, $Q(6) = 4.68$, $p < 0.05$, for toddlers than infants, $Q(4) = 5.04$, $p < 0.05$, and in higher quality studies, $Q(4) = 14.58$, $p < 0.05$. Results highlight the potential benefits of including a parent stress management component in childhood obesity prevention programs.

Keywords

maternal stress; child obesity; body mass index; stress

In 2007–2008, 9.5% of infants and toddlers in the United States and 16.9% of children aged 6 to 19 years were estimated to be obese [1]. Child obesity prevention programs typically emphasize making changes to children's diet and physical activity behaviors, and such programs have shown moderate success [2, 3]. Although including child caretakers has been recommended for boosting obesity prevention program success, [4] targeting specific changes in mothers' behaviors has not often been a focus of childhood obesity prevention programs. Programs that have targeted parenting practices, such as child-feeding strategies, rarely address issues such as parents' depression, emotional distress, or anxiety [5]. Yet parents of overweight and obese children report higher rates of negative emotions [6, 7]. Growing evidence suggests that mothers' psychological stress is associated with children's decreased

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consumption of fruits, vegetables and high-calcium foods [8], as well as lower physical activity parenting, perceived lower importance of child physical activity, and higher child sedentary behavior [9, 10]. These behaviors increase children's risk for obesity or compromised nutritional status [5, 7, 11–17]. Studies examining different facets of parental stress such as social, financial, and parenting stress show associations with increased rates of obesity in children [15, 17, 18]. However, not all studies find a significant relationship between parental stress and child obesity risk.[19–21] Conflicting findings underscore the need for conclusive, summative evidence.

Untested moderators such as type of maternal stress, child's own experience of stress, and child age may account for the observed inconsistencies in the size and direction of the association between maternal stress and child obesity. In particular, mothers' stress that is specifically tied to parenting may be associated with child obesity risk in ways that differ from other types of stress. Parenting stress is characterized by feelings of distress combined with a dysfunctional parent-child relationship and a difficult child. [22, 23] Parenting stress has been shown to interact with parenting behaviors, [24] but whether its effect on child obesity risk is stronger than that of generalized stress remains an open question. In addition, children's experience of stress may exacerbate the effects of maternal stress on children's obesity risk. In children, chronic stress repeatedly activates the hypothalamic-pituitary-adrenal axis (HPA) and release of stress hormones (e.g., glucocorticoids), which are associated with subsequent metabolic syndrome, including visceral adiposity, with particularly damaging effects during crucial stages of brain development [25–27]. Stressors experienced by the child, such as maltreatment and abuse, have been associated with higher rates of concurrent and subsequent obesity [28, 29]. Finally, child age may moderate the relationship between maternal stress and child obesity, with a smaller effect for older children. Although parent feeding practices play an important role in child eating behaviors, childcare and school settings have an increasingly strong influence as children spend more time away from home with age [30]. Peers and media also contribute to child dietary consumption patterns [31], and media exposure may increase as children age. For infants, the introduction of solid foods creates variability in their diet, and consistent provision of healthy foods in appropriate quantities could be disrupted by maternal stress. Child age could be a proxy for these types of changes, reducing the effect of maternal stress on the incidence of child obesity risk for older children.

The current meta-analysis synthesized data from cross-sectional and longitudinal studies measuring the association between maternal stress and child obesity in order to estimate an overall effect size and test moderators of the effect. Data from cross-sectional studies were expected to provide evidence about whether there are concurrent, but not necessarily directional, associations between stress and obesity. Results from longitudinal studies were expected to shed light on whether maternal stress, especially coupled with children's stress or present during critical periods of development, increased children's subsequent obesity risk. Based on the existing evidence, we hypothesized that the effect size for maternal stress and child BMI would be (a) positive, (b) larger when children would have also experienced the stressor, and (c) smaller for older children.

Method

Sample of studies and inclusion criteria

Literature searches were conducted between February 5 and June 25 of 2014 using Google Scholar, Web of Science, PyscINFO, PsycEXTRA, ProQuest. Search terms and logic relations were: (*maternal* OR *mother*) AND (*stress* OR *allostatic load* OR *stressor*) AND "*child BMI*" OR "*obesity*" NOT *prenatal* NOT *rat* NOT "*in utero*"). Ancestral searches were conducted from reference sections of articles retrieved via database searches. Author websites were searched for unpublished or additional published results on the topic, and requests for unpublished data were sent to the listservs of the American Public Health Association and the Society for Behavioral Medicine. To be included in this review, studies had to (a) be conducted on a human population, (b) include mother-child dyads in which children were 18 years of age or younger, (c) measure stress that mothers experienced after the child's birth, (d) report the relationship between maternal stress and a measure of child obesity (*e.g.*, BMI z-score, BMI > 95th percentile, waist circumference), and (e) be a quantitative (vs. qualitative) study. Studies not reported in English were excluded.

Database, ancestry searches, and personal communications returned 142 studies after removal of duplicates. Listserv requests returned zero. One study met screening criteria but was not included because it reported findings by categorizing children into "overweight" vs. "not overweight" based on adjusted relative weight over the 75th percentile according to the Department of Health Education and Welfare norms in 1979, which could not be compared to current BMI over the 95th percentile [32]. Only one study was excluded because it used a measure of obesity that was not based on BMI or BMI z-score (*i.e.*, waist-to-hip ratio)[7]. Two longitudinal studies used the same dataset (Fragile Families and Child Well-being Study; [17, 33] but calculated maternal stress exposure differently (exposure to intimate partner violence only *vs.* a composite score that included intimate partner violence, food insecurity, housing insecurity, maternal depressive symptoms, maternal substance use, and father's incarceration). Effect sizes from those two studies were combined in the analysis. Two longitudinal studies used two different measures of "overall stress" (*ex.*, composite stressors and perceived stress), [19, 34] which were combined to form one "overall stress" effect size per study.

Effect size estimation procedures

Odds ratios (OR), correlation coefficients, and regression coefficients were reported and retained for the meta-analysis. Odds ratios (OR) were retrieved directly from study results for both cross-sectional and longitudinal studies. ORs were converted into effect sizes and standard errors using ln-transformations, resulting in effect sizes comparable to standardized mean difference scores (*d*) [35]. This method is provided in the *Cochrane Handbook for Systematic Reviews of Interventions* [36]. A positive *d* indicated that higher maternal stress was associated with increased child obesity risk, whereas a negative *d* indicated that higher maternal stress was associated with reduced obesity risk. Effect sizes for correlations and regression coefficients, *d*s, and variance of *d*s were calculated according to the procedures outlined in Borenstein et al. [37].

Decision rules for lifting effect size information from study reports were as follows: When both Centers for Disease Control (CDC) and International Obesity Task Force (IOTF) cut-points were used to determine obesity status, CDC scores were retained to allow comparability with other studies [13]. When ORs were given only for sub-groups (*ex.*, gender), a summary OR was calculated using the overall summary effect procedures for fixed effects outlined in Borenstein (2011) [17]. When ORs were presented at different levels of stress, we retained the comparison between high/chronic stress with low/no stress [17, 33]. When a correlation or OR was tested but not reported as significant, it was assumed to be non-significant, and an effect size of 0 was imputed [38]. In those cases, confidence intervals were calculated based on the assumption that equal numbers of participants would have fallen into each of the four cells of a 2×2 odds ratio table. Summary effect sizes were calculated with and without the imputed zero values, and the results are described below [38, 39]. Studies were coded for the percentage of obese children in the sample and the presence of seven of the most common covariates reported by the included studies: race, marital status, age, socioeconomic status, education, general health, maternal obesity.

Coding for moderators

All coding and rating were performed by two authors independently, and initial agreement for each item ranged from 70% to 100%. All discrepancies were resolved through discussion. First, a code was created to reflect whether the maternal stress measure captured parenting stress specifically (Parenting Stress Index or subscale [14, 40–42], Swedish Parenting Stress questionnaire [18, 34]) vs. an overall/composite measure of stress (cumulative maternal stressor index [11], cumulative social risk summary [17], Depression Anxiety Stress Scale [43, 44], General health questionnaire [19], Holmes and Raye Life Change Questionnaire [32, 39], intimate partner violence [33], Life event checklist + Swedish Parenting Stress Questionnaire + Quality of social support + Parental worries [34], Perceived Stress Scale [38, 45], Self-esteem + Brief symptom inventory + work stress + overall health + social support [13], Symptom Distress Checklist [19], Symptom Distress Checklist + Global Severity Index [46]). Second, studies were coded for whether or not the child would most likely have experienced the same stress or stressor as the one captured by the maternal stress measure. Codes were assigned to indicate that children would have likely experienced the stress (cumulative maternal stressor, cumulative social risk index, Holmes and Raye Life Change questionnaire, intimate partner violence, Life event + SPSQ + quality of social support + parental worries, Parenting Stress Index, Swedish Parenting Stress Questionnaire). Studies that either controlled for child stress separately or used a maternal stress measure that reflected stress that the mother *could* have experienced outside the child's realm were coded to reflect this (Depression Anxiety Stress Scale, General Health Questionnaire, Perceived Stress Scale, Self-esteem + Brief symptom inventory + work stress + overall health + social support, Symptom Distress Checklist, Symptom Checklist + Global Severity Index). Third, average child age when maternal stress was measured ("baseline" in longitudinal studies) was used as the indicator of child age.

Quality assessment

The Tool to Assess Risk of Bias in Cohort Studies was modified and used to evaluate study quality [47] (see supplement). One item assessed population representativeness, two items

assessed risk of bias in stress and obesity measures, a fourth assessed self-selection bias, and a fifth assessed adjustment for baseline level of child weight/obesity (longitudinal only). Scoring options for each item ranged from *1 = High risk of bias* to *4 = Low risk of bias*. Higher scores indicated higher study quality.

Data analytic plan

Overall effect size, confidence intervals, and *Q* (standardized measure of heterogeneity within effect sizes) were calculated separately for cross-sectional and longitudinal studies using mixed-effects models. Mixed-effects models were used in the analysis. Comparisons across subgroups (e.g., parenting stress vs. overall stress) assumed a fixed variance across studies, indicating that the categories had a common meaning across studies. The variance within subgroups (e.g., parenting stress) was modeled as a random effect, indicating that effect sizes were randomly selected from multiple populations and did not necessarily reflect a single underlying mean value [37]. Continuous moderators (i.e., child age, study quality) were tested using mixed-effects Method of Moments regression models. Analyses were conducted using Comprehensive Meta-Analysis Version 2 (2005) (http://www.meta-analysis.com/index.html).

Publication bias—Two techniques were used to assess potential publication bias. First, Egger's test provided a significance value for the test of asymmetry of funnel plots; two-tailed tests are reported [48]. Asymmetrical funnel plots suggest publication bias especially if less precise, small-sample studies are more likely to show effects in the predicted direction than more precise, large-sample studies. Second, Duval and Tweedie's Trim & Fill (random effects) procedure was used to estimate the number of missing studies that would need to be added to create a symmetrical distribution around the observed overall effect size [49, 50].

Results

Descriptive statistics

The search process resulted in 17 studies with 21 effect sizes (15 cross-sectional, 6 longitudinal) (see Figure 1). Most studies adjusted for marital status (59%), child age (65%) (by study design or statistical adjustment), SES (71%), maternal education (82%), and maternal obesity (88%). Effect sizes from studies that adjusted for covariates did not differ significantly from those that did not. Thus, adjusted and unadjusted effect sizes within studies were pooled, leaving a total of 19 independent effect sizes (14 cross-sectional, 5 longitudinal). The percent of child obesity in the cross-sectional and longitudinal samples was not significantly associated with the effect size.

Tables 1 and 2 show study characteristics and descriptive statistics. Children in the cross-sectional studies were elementary school age (M = 6.36 yrs., SD = 3.23) but were younger at baseline in longitudinal studies (M = 1.68 yrs., SD = 1.38), $p < 0.01$. Average rates of child obesity were 17.6% (SD = 18.27%) for children and 7.9% (SD = 7.47%) for infants and toddlers, comparable to U.S. estimates [1]. Studies were relatively balanced on child gender and were conducted in 5 different countries (*i.e.*, Australia, Brazil, Denmark, Sweden, and

United States). Sample size ranged from 110 mother-child dyads to 21,121, with a mean of 2,462 (*SD* = 4,980).

Overall Effect Size for Association between Maternal Stress and Child Obesity

For cross-sectional studies, the overall effect size between maternal stress and child obesity was positive and significantly different from zero, $d = 0.20$ ($k = 14$, 95% CI: 0.06, 0.34) and had significant heterogeneity, $Q(13) = 193.00$, $p < .001$ (see Table 3). The overall effect size was not changed by excluding the imputed effect size of 0 for one study [38], $d = 0.20$ ($k = 13$, 95% CI: 0.06, 0.35, $p = 0.006$, $Q(12) = 192.99$, $p < .001$. For longitudinal studies, the overall effect size between maternal stress and child obesity was marginally significant, $d = 0.18$ ($k = 5$, 95% CI: 0.00, 0.35, $p = 0.05$) and had significant heterogeneity, $Q(4) = 29.46$, $p < .001$.

Putative moderators of the association between maternal stress and child obesity

The comparison between types of stress (parenting versus overall) did not reach significance for the cross-sectional, $Q(1) = 0.02$, $p = .881$, or longitudinal studies, $Q(1) = 0.026$, $p = .871$.

For cross-sectional studies, the comparison between children who would have experienced stress versus those who would not was not significant, $Q(1) = 0.74$, $k = 14$, $p = .785$, indicating that the relationship between maternal stress and child obesity is relatively constant across levels of child stress. However, the comparison was significant in longitudinal studies, $Q(1) = 4.68$, $p = 0.031$ reflecting that the effect size was larger for children who would have experienced stress, $d = 0.24$, $k = 4$, 95% CI: 0.02, 0.45, than those who would not, $d = 0.00$, $k = 3$, 95% CI: −0.02, 0.02.

In cross-sectional studies, child age at maternal stress assessment was not significantly associated with effect size ($B = -0.03$, $k = 11$, 95% CI: −0.10, 0.03), $Q(1) = 1.16$, $p = 0.281$, suggesting that the effect size was relatively constant for children aged 3 – 13 years (3 studies did not provide sufficient information). However, in longitudinal studies, which had an age range of 0 – 3 years at baseline, older age at baseline was associated with a larger effect size ($B = 0.11$, $k = 5$, 95% CI: 0.01, 0.21), $Q(1) = 5.04$, $p = 0.025$ (see Figure 3).

Study quality

Study quality was not significantly associated with effect size in cross-sectional studies ($B = -0.09$, $k = 14$, 95 % CI: −0.46, 0.28), $Q(1) = 0.22$, $p = 0.636$ but was associated with a larger effect size in longitudinal studies ($B = 0.86$, $k = 5$, 95% CI: 0.42, 1.30), $Q(1) = 14.58$, $p < 0.001$ (see Figure 3), indicating that higher quality studies had larger effect sizes.

Publication Bias

For cross-sectional studies, the Egger's regression test was not significant, $t(12) = 1.72$, $p = 0.112$, suggesting a symmetrical funnel plot and no publication bias. Similarly, Duval and Tweedie's technique estimated that no missing studies would be required to create a symmetrical distribution, also indicating no bias. The pattern was similar for longitudinal studies. Neither Egger's test, $t(3) = 2.38$, $p = 0.097$, nor Duval and Tweedie's technique indicated publication bias.

Discussion

As child obesity continues to be a public health problem, discovering parental factors that influence obesity risk provides new targets for intervention and prevention. Results of this meta-analysis indicate that maternal stress may be one such factor. Across study designs, children were at greater risk of obesity when mothers experienced stress. For longitudinal studies, higher study quality was associated with larger effect sizes, supporting the validity of the results.

Our results support theoretical predictions that maternal stress may reduce proactive obesity-related parenting practices [13, 51, 52] or affect weight gain through changes in maternal or child mental health. According to household production theories and family stress theory, maternal stress could change parenting behavior, such as meal preparation or transportation to organized sports, in ways that ultimately increase obesity risk. [8–10, 13, 51] Maternal stress may also reduce parent sensitivity or disrupt bonds of secure attachment formation, decreasing children's ability to learn self-regulation skills, such as control of eating behavior. [52, 53] While no other studies, to our knowledge, have systematically examined maternal stress in this way, one review found that chronic maternal depression was linked to greater risk for child overweight.[54] Thus, our study extends previous work to paint a fuller picture of the "family ecology"[54] that might identify higher risk children. Obesity prevention programs and policies aimed at parents and families may want to consider the important influence of maternal stress.

Children's own experience of stress seemed to exacerbate the association between maternal stress and subsequent higher child obesity risk. This finding fits with previous research confirming a positive association between chronic stress and obesity in children, although studies have been mixed regarding longitudinal effects [55]. Children's experience of chronic stress could increase energy intake, decrease physical activity, or stimulate visceral fat accumulation through elevated cortisol secretion [55, 56]. Among adolescents, stress-related eating has been shown to co-occur with other unhealthful habits, such as inadequate sleep or high-calorie/low-nutrient food consumption [57]. Child stress may interact with other stressors to decrease physical activity, such as family financial strain [10] or natural disasters [58]. This finding brings to light the possibility that maternal stress is only a risk factor for subsequent child obesity risk *when the child also experiences stress*. The two main types of child stress that appeared in the current review were environmental/external stressors and mother-child dysfunctional relationship. Stress that mothers experienced outside of those domains, such as work-related difficulties, did not heighten child risk of obesity. However, in the cross-sectional studies, this was not the case. On a cross-sectional basis, child risk was equally high whether or not the children would have experienced the stress(or). Combined, these findings indicate that maternal stress identifies concurrent child obesity risk but not subsequent risk, unless the stressor also affects the child. Combined, these findings indicate that maternal stress identifies concurrent child obesity risk but not subsequent risk, unless the stressor also affects the child. This discovery should be tested in subsequent studies but suggests that policy-makers and program designers focused on primary prevention consider addressing both mother's and children's stress.

This meta-analysis also revealed that toddlers may be more susceptible to the effects of maternal stress on obesity than infants. Results from longitudinal studies indicated that the effect of maternal stress on child obesity was stronger when the stressor occurred during the toddler years than infancy. Yet across the ages of 3 – 13 years, as found in the cross-sectional studies, the effect size was relatively consistent. Different age ranges in the cross-sectional and longitudinal studies likely account for these different results. Maternal stress may have little effect on the composition of the child's nutritional intake if it is experienced before children have transitioned to solid foods only; but the wider variety of foods and greater time and preparation required for feeding solid foods may introduce variability into feeding practices that could be disrupted by stress and lead to excess weight gain. For older children, the impact of maternal stress may be diluted by a larger network of social influences and food environments.

Previous theory and research suggested a link between *prenatal* maternal stress and subsequent child obesity risk[59, 60] but this meta-analysis is, to our knowledge, the first to synthesize results from maternal stress during infancy and childhood, finding higher risk from toddlerhood through adolescence. Given that eating preferences and self-regulatory ability can be established in childhood,[61, 62] and that weight tends to track across development, childhood is an important target for prevention.[63] Infancy may be a relatively lower-risk time because the content of the child's diet is consistent (i.e., breast milk or formula). Preventing and treating maternal stress when children are toddler-age may be the most strategic intervention point for maternal stress-based policies and programs.

Various mechanisms may help explain the effect of maternal stress on child obesity, such as changes in obesity-related parenting practices, child internalizing symptoms, or increased maternal depression.[9, 51, 64–66] Stressed mothers may be more likely to disengage from proactive physical activity parenting or healthy meal planning, resulting in higher fast-food consumption or altered feeding practices [9, 51, 15, 64]. Parenting stress might also increase child internalizing symptoms (*ex.*, depression, anxiety) [66], which have been associated with adolescent obesity [67, 68], weight gain as children age [69], and impaired adolescent weight loss [70] possibly due to changes in health behaviors, psychiatric medication, or sociobiological factors affecting both symptoms and weight [68]. Chronic maternal stress could precipitate or exacerbate maternal depression, interact with parenting styles and increase child overweight [65, 71–73, 74]. However, a post-hoc analysis conducted with the current data did not find a significant difference between measures of maternal stress that included mental health ratings, such as depression or anxiety, and those without (results not reported). Future research is needed to investigate how mental health may influence stress, interact with parenting practices or affect child internalizing symptomatology.

One limitation of the present research is that most of our studies were conducted in developed countries, potentially limiting global generalizability. One study conducted in a developing country found that maternal stress was associated with *lower* child obesity risk [45], and another found that the effect of poverty – a stressor – on child obesity risk differed by country [75]. Possibly, maternal stress lowers mothers' ability to protect children from environmental pressures, such as the marketing of unhealthy food products,[76] pushing children toward obesity in countries where food is abundant but toward malnutrition in

nutritionally impoverished areas. A post-hoc analysis with the current cross-sectional data suggested that SES may moderate the effect, with no effect among low-income populations (in developed countries) and a positive effect in the general population. However, the trend did not reach significance ($p = 0.09$, $k = 14$, results not reported), and there were a small number of effect sizes from low-income populations ($n = 3$). Finally, the relatively small number of studies included in the overall meta-analysis, especially longitudinal, underscores the need for additional research.

Conclusions and future research

Across study designs, children whose mothers who experienced stress were at greater risk for obesity. Longitudinal studies are needed to test changes over time, the mechanisms of transmission, establish the temporal order of the effect (i.e., maternal stress leads to child obesity), and assess whether reciprocal relationships occur across time. Some studies have begun to test mediators, such as child feeding practices [43], but effects on child weight are not consistent [20], and additional work is needed to clarify disparate findings. Intervention studies are needed to examine potential causal effects of maternal stress reduction on decreasing child obesity risk. Many studies, including this one, operate under the hypothesis that parenting behaviors increase child risk, but the causal arrow could run in the opposite direction, with the diagnosis of an obese child heightening parent stress or altering parenting behaviors. Additional research is needed that uses consistent measures of maternal stress (*e.g.*, Cohen's Perceived Stress scale) and alternative methods of assessment such as ecological momentary assessment[77], salivary cortisol[78], waist-to-height ratios[79], or dual energy X-ray absorptiometry (DEXA)[80] scans to clarify whether differences exist that are not identified using less sophisticated technology. In conclusion, mothers who experience higher perceived stress or are exposed to more stressors may have children at greater risk for obesity. Although additional basic and intervention research is needed, the potential remains that child obesity prevention programs may be improved by minimizing the negative effects of maternal stress on child obesity risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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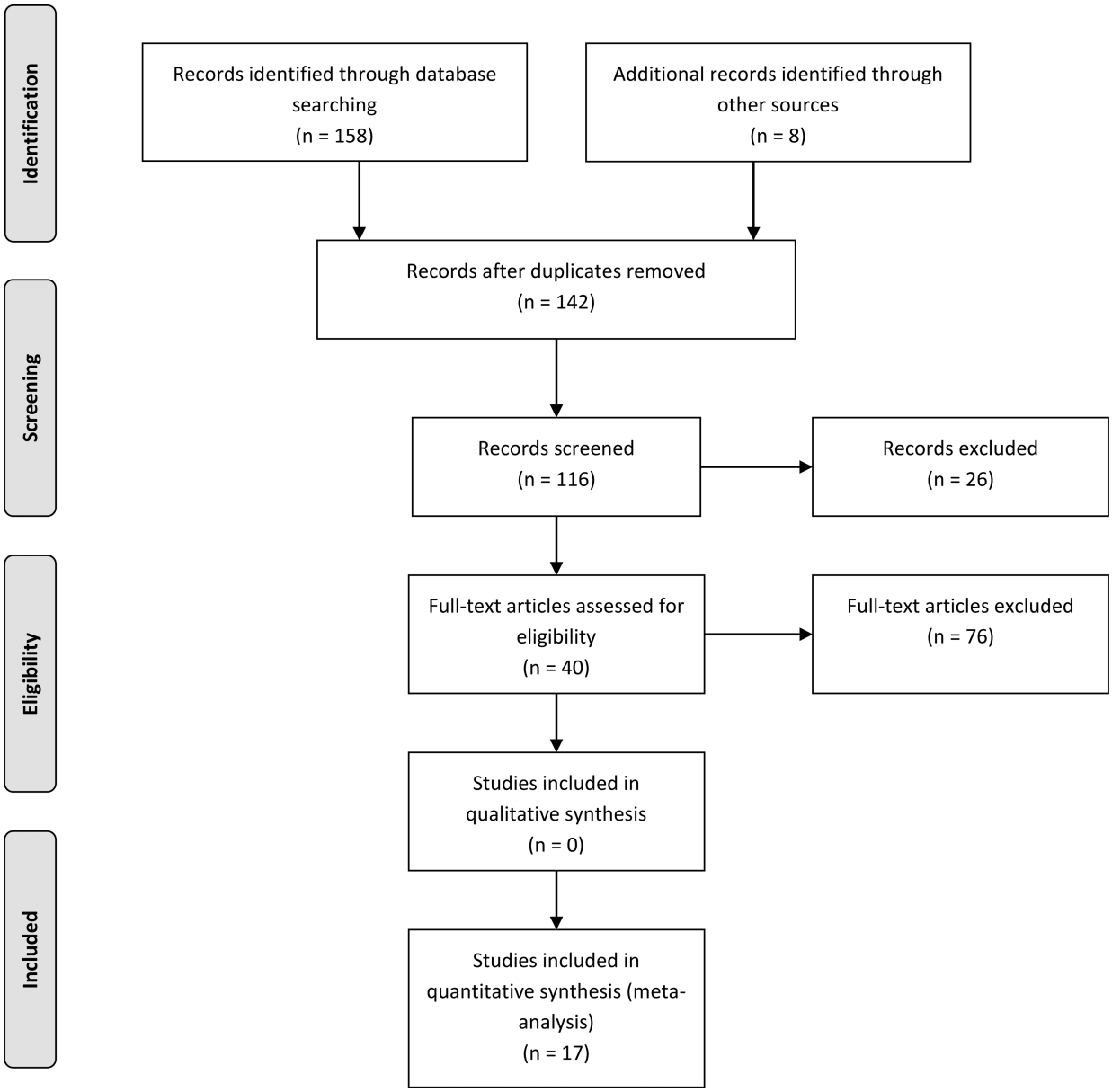

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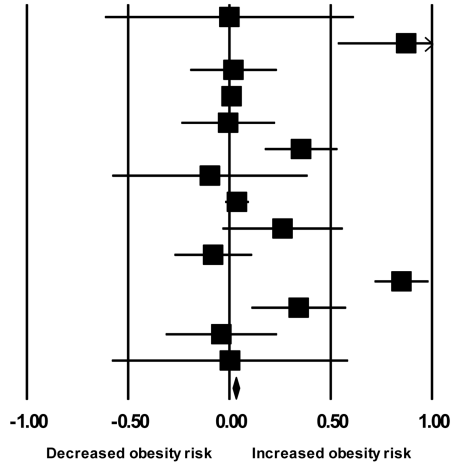
For more information, visit www.prisma-statement.org.

Figure 1.
PRISMA 2009 Flow Diagram

Overall Effect Size in Cross-sectional Studies

Study name	Point estimate	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
(Burdette et al., 2001)	0.000	0.315	0.099	-0.617	0.617	0.000	1.000
(Gemmill et al., 2013)	0.873	0.174	0.030	0.533	1.213	5.028	0.000
(Gibson et al., 2007)	0.020	0.110	0.012	-0.196	0.236	0.181	0.856
(Gunderson et al., 2008)	0.011	0.008	0.000	-0.005	0.027	1.304	0.192
(Koch et al., 2008)a	-0.006	0.119	0.014	-0.240	0.227	-0.052	0.958
(Koch et al., 2008)b	0.354	0.093	0.009	0.172	0.536	3.819	0.000
(Levers-Landis et al., 2008)	-0.096	0.247	0.061	-0.581	0.388	-0.389	0.697
(Lohman et al., 2009)	0.037	0.031	0.001	-0.024	0.098	1.202	0.229
(McPhie et al., 2011)	0.262	0.153	0.023	-0.037	0.562	1.715	0.086
(Rondó et al., 2013)	-0.080	0.099	0.010	-0.274	0.114	-0.808	0.419
(Stenhammar et al., 2010)	0.850	0.069	0.005	0.715	0.985	12.313	0.000
(Zeller et al., 2007)	0.342	0.120	0.015	0.106	0.578	2.840	0.005
(Bergmeier et al., 2014)	-0.040	0.141	0.020	-0.317	0.237	-0.283	0.777
(Walton et al., 2014)	0.003	0.298	0.089	-0.582	0.588	0.009	0.993



Overall Effect Size in Longitudinal Studies

Study name	Point estimate	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
(Ajslev et al., 2010)	-0.002	0.010	0.000	-0.022	0.018	-0.193	0.847
(Boynton-Jarrett & Suglia, combined)	0.266	0.084	0.007	0.102	0.431	3.173	0.002
(Koch et al., 2008)	0.523	0.124	0.015	0.279	0.766	4.212	0.000
(Sowan & Stember, 2000)	0.000	0.080	0.006	-0.156	0.156	0.000	1.000
(Bergmeier et al., 2014)	0.201	0.142	0.020	-0.078	0.480	1.414	0.157

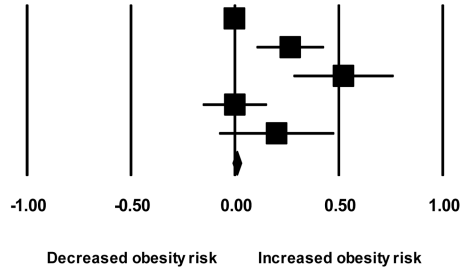


Figure 2.
Overall Effect Size Estimates for Cross-sectional and Longitudinal Studies

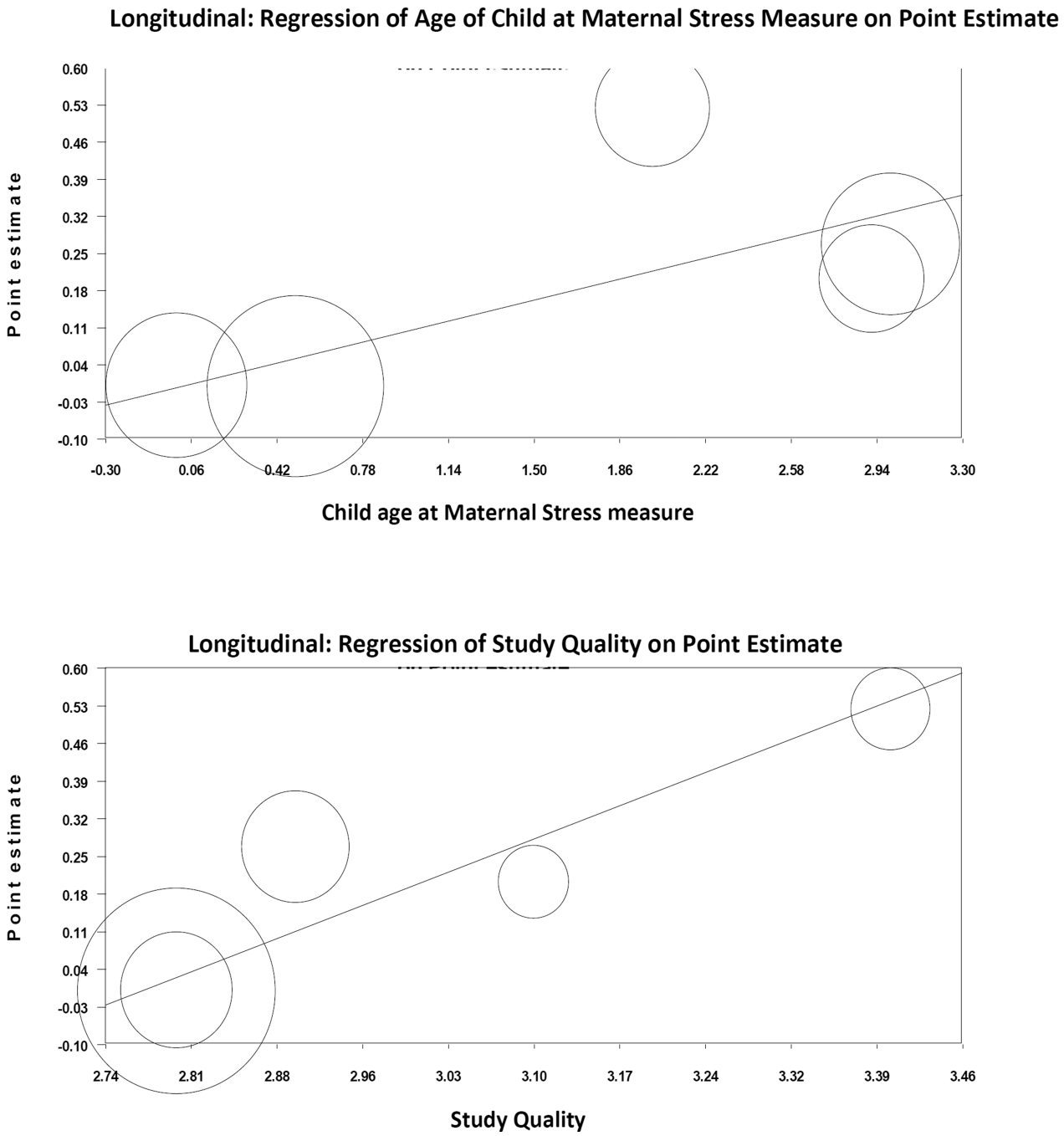


Figure 3.

Regression of Effect Size on Age of Child at Maternal Stress Measure and Study Quality in Longitudinal Studies

Table 1

Study characteristics

Study name	Dataset	d	SE	Maternal stress	Published	Sample size	Country	Avg. child age	Avg. mat. age	Child Stress	% Child obesity	BMI method	% Male	Study quality
Longitudinal														
(Ajslev et al., 2010)	Danish National Birth Cohort	−0.002	0.010	Stress	Yes	21121	Denmark	7.00	30.69	No	--	Reported	--	2.800
(Bergmeier et al., 2014)	Australian Research Council Discovery	0.201	0.142	Parenting stress	Yes	201	Australia	3.92	36.11	Yes	3.0%	Assessed	42.3%	3.100
(Boynton-Jarrett & Suglia, combined)	Fragile Families and Child Well-being Study	0.266	0.084	Stress	Yes	1595	USA	5.00		Yes	16.5%	Assessed	51.2%	2.900
(Koch et al., 2008)	All Babies in Southeast Sweden-project	0.523	0.124	Stress	Yes	5302	Sweden	5.00		Yes	4.2%	Assessed	52.1%	3.400
(Sowan & Stember, 2000)	Infant Growth Study	0.000	0.080	Stress	Yes	630	USA	0.58		Yes	0.0%	Assessed	54.0%	2.800
Cross-sectional														
(Bergmeier et al., 2014)	Australian Research Council Discovery	−0.040	0.141	Parenting stress	Yes	201	Australia	2.92	36.11	Yes	3.0%	Assessed	42.3%	3.125
(Burdette et al., 2001)	Vermont Special Supplemental Nutrition Program	0.000	0.315	Stress	No	150	USA			No	--	Assessed	--	3.375
(Gemmill et al., 2013)	National Postnatal Depression Program	0.873	0.174	Stress	Yes	159	Australia	4.82	37.00	No	10.1%	Reported	45.3%	2.625
(Gibson et al., 2007)	Childhood Growth and Development Study	0.020	0.110	Stress	Yes	329	Australia	9.51		No	12.2%	Assessed	--	3.750
(Gunderson et al., 2008)	National Health and Nutrition Examination Survey	0.011	0.008	Stress	Yes	841	USA	8.30	32.32	Yes	--	Assessed	54.4%	3.000
(Koch et al., 2008)	All Babies in Southeast Sweden-project	−0.006	0.119	Parenting stress	Yes	6603	Sweden	5.00		Yes	4.2%	Assessed	52.1%	3.500
(Koch et al., 2008)	All Babies in Southeast Sweden-project	0.354	0.093	Stress	Yes	6106	Sweden	5.00		Yes	4.2%	Assessed	52.1%	3.625
(Ievers-Landis et al., 2008)	Cleveland Children's Sleep and Health Study	−0.096	0.247	Parenting stress	Yes	819	USA	9.50		Yes	16.9%	Unknown	51.0%	3.000

Study name	Dataset	d	SE	Maternal stress	Published	Sample size	Country	Avg. child age	Avg. mat. age	Child Stress	% Child obesity	BMI method	% Male	Study quality
(Lohman et al., 2009)	Welfare, Children, and Families: A Three-City Study	0.037	0.031	Stress	Yes	1011	USA		38.32	Yes	20.0%	Assessed	48.0%	3.000
(McPhie et al., 2011)	Recruited for this study	0.262	0.153	Parenting stress	Yes	175	Australia	2.83	35.69	Yes	--	Reported	46.3%	2.875
(Rondó et al., 2013)	National Health Service (Sistema Unico de Saude)	−0.080	0.099	Stress	Yes	409	Brazil	6.50	30.70	No	4.2%	Assessed	44.0%	3.500
(Stenhammar et al., 2010)	Child Public Health Unit in Uppsala County, Sweden	0.850	0.069	Parenting stress	Yes	865	Sweden	3.00		Yes	--	Assessed	--	3.625
(Walton et al., 2014)	Parents and Tots Together	0.003	0.298	Parenting stress	No	110	USA			Yes	48.5%	Unknown	--	3.750
(Zeller et al., 2007)	Recruited for this study	0.342	0.120	Stress	Yes	159	USA	12.59	40.53	No	52.3%	Assessed	43.9%	2.833

Table 2

Descriptive Statistics for Cross-sectional and Longitudinal Studies

Item	Cross-sectional M (SD) or N (%)		Longitudinal M (SD) or N (%)		t-test or χ^2
Stress					
Stress measure					--
Parenting stress	6	42.86	1	20.00	
Stress	8	57.14	4	80.00	
Child stress					--
No	5	35.71	1	20.00	
Yes	9	64.29	4	80.00	
Avg. child age at mat. stress measure	6.36	3.23	1.68	1.38	4.06**
Sample characteristics					
Percent child obesity	17.55	18.27	5.93	7.27	1.70
Percent male	47.94	4.20	49.90	5.20	−0.67
Mother's average age	35.81	3.38	33.40	3.83	0.80
Avg. age child at obesity measure	6.36	3.23	4.30	2.36	1.44
Country of origin					--
Australia	4	28.57	1	20.00	
Brazil	1	7.14	0	0.00	
Denmark	0	0	1	20.00	
Sweden	3	21.43	1	20.00	
United States	6	42.86	2	40.00	
Methodological features					
Outcome Assessment type					--
Assessed	10	71.43	4	80.00	
Self-report	2	14.29	1	20.00	
Unknown	2	14.29	0	0.00	
Sample size	1281.20	2174.70	5769.80	8813.90	−1.13
Publication year	2009.40	3.52	2008.00	5.89	0.44

Item	Cross-sectional M (SD) or N (%)		Longitudinal M (SD) or N (%)		t-test or χ^2
Study quality	3.26	0.38	3.00	0.26	1.69

** < 0.01

-- too few studies to estimate significance values

Note: Satterthwaite t-test results are reported, which assume unequal variances

Table 3

Effect Sizes by Moderator for Cross-sectional and Longitudinal Studies

			Cross-sectional Effect size estimate			Longitudinal Effect size estimate
Moderator	***k***	***Q***	***d* or *slope* (95% CI)**	***k***	**Q**	***d* or *slope* (95% CI)**
Overall effect	14	193.00**	0.196** (0.056 / 0.337)	5	29.461**	0.176‡ (0.000 / 0.351)
Stress measure		0.022			0.026	
Parenting stress	6		0.181 (−0.236 / 0.598)	1		0.201 (−0.078 / 0.480)
Overall stress	8		0.148** (0.038 / 0.258)	4		0.173 (−0.026 / 0.371)
Child stress		0.74			4.679*	
No	5		0.232 (−0.086 / 0.550)	3		−0.002 (−0.022 / 0.018)
Yes	9		0.182* (0.008 / 0.355)	4		0.237* (0.021 / 0.453)
Child age^	11	1.162	−0.034 (−0.096 / 0.028)	5	5.037*	0.110* (0.014 / 0.206)
Study quality	14	0.224	−0.090 (−0.461 / 0.282)	5	14.581*	0.858** (0.418 / 1.298)

** p < 0.01,

* p < 0.05,

‡ p = 0.05

^ Child age at maternal stress measure