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Maternal depressive symptoms predict general liability in child psychopathology

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Abstract

Objective: The current study examines how maternal depressive symptoms relate to child psychopathology when structured via the latent bifactor model of psychopathology, a new organizational structure of psychopathological symptoms consisting of a general common psychopathology factor (p-factor) and internalizing- and externalizing-specific risk.

Method: Maternal report of depressive symptoms (Beck Depression Inventory - II) and child psychopathological symptoms (Child Behavior Checklist and Children's Behavior Questionnaire) were provided by 554 mother-child pairs. Children in the sample were 7.7 years old on average ($SD = 1.35$, range = 5–11 years), and were 49.8% female, 46% Latinx, and 67% White, 6% Black, 5% Asian/Pacific Islander, and 21% multiracial.

Results: Maternal depressive symptoms were positively associated with the child p-factor but not with the internalizing- or externalizing-specific factors. We did not find evidence of sex/gender or race/ethnicity moderation when using latent factors of psychopathology. Consistent with past research, maternal depressive symptoms were positively associated with internalizing and externalizing composite scores on the Child Behavior Checklist.

Conclusions: Findings suggest that maternal depressive symptoms are associated with transdiagnostic risk for broad child psychopathology (p-factor). Whereas the traditional Achenbach-style approach of psychopathological assessment suggests that maternal depressive symptoms are associated with both child internalizing and externalizing problems, the latent bifactor model suggests that these associations may be accounted for by risk pathways related to the p-factor rather than internalizing or externalizing specific risk. We discuss clinical and research implications of using a latent bifactor structure of psychopathology to understand how maternal depression may impact children's mental health.

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Keywords

P-factor; Bifactor latent models; Maternal depression; Child psychopathology; Risk

Background

Maternal depression is one of the strongest risk factors for poor mental health outcomes in offspring, including the development of major depressive disorder and other mood related disorders throughout the lifespan (Downey & Coyne, 1990; Goodman et al., 2011). However, the impact of maternal depression is not specific to depressive disorders among offspring, or even to the internalizing domain of psychopathology. Rather, maternal depression relates to a wide range of offspring psychopathologies and problem behaviors, such as increased anxiety, conduct problems, negative affect, and school problems, as well as lower self-esteem, social competency, and positive affect (Goodman & Gotlib, 1999; Goodman et al., 2011). Meta-analytic data suggest that children of mothers with depression are at a higher risk of having both internalizing ($k = 121$, $r = 0.23$) and externalizing ($k = 111$, $r = 0.21$) problems than children whose mothers are not depressed, with similar effect sizes across these psychopathology dimensions (Goodman et al., 2011). Maternal depression thus is not associated with the development of a single psychiatric diagnosis, but rather has been characterized as a non-specific risk factor for psychopathology broadly.

Not only are both internalizing and externalizing psychiatric outcomes observed in children of depressed mothers, but diagnoses often overlap, reflecting commonly observed patterns of comorbidity which are pervasive across psychiatric nosologies such as the Diagnostic and Statistical Manual of Mental Disorders (DSM) and International Classification of Diseases (ICD) (Kessler, Chiu, Demler, & Walters, 2005; Merikangas et al., 2010). Comorbidity among common psychopathologies is highly prevalent, as 45% of individuals who meet criteria for one psychiatric disorder will be diagnosed with a second (Kessler, Berglund, et al., 2005). Given the critiques of the co-occurrence among discrete psychopathological disorders (e.g., Krueger & Markon, 2006; Rutter & Uher, 2012), more recent dimensional models of psychopathology have been proposed and evaluated as one alternative approach to organize symptoms and syndromes in psychopathology, including bifactor models (e.g., Caspi et al., 2014; Hankin et al., 2016; Lahey, Krueger, Rathouz, Waldman, & Zald, 2017; Snyder, Young, & Hankin, 2017b). In bifactor models of psychopathology (e.g., the p-factor model), shared symptom covariance across traditional psychopathological diagnoses and syndromes are organized together in a single general, latent factor of psychopathology, similar to the g factor of intelligence (Caspi et al., 2014). Remaining variance in manifest symptoms or syndromes is further partitioned into internalizing and externalizing-specific latent factors, such that each latent factor accounts for independent and unique variance in psychopathological symptoms. Thus, what is common and shared across psychopathology is grouped together and represented in the p-factor as a general latent dimension, and the specific latent dimensions of internalizing and externalizing represent unique symptoms related to emotional, internalizing symptoms and behavioral, externalizing problems, respectively. This latent bifactor model of psychopathology is a promising approach, because it provides a more parsimonious organization of psychopathological symptoms,

allows for dimensional symptom variance, and may map on to the observed general risk for many manifestations of DSM-oriented psychopathologies among children of depressed mothers.

Recent research in neuroscience, genetics, temperament, and environmental risk has shown that latent dimensions of the p-factor model directly map on to key risk pathways hypothesized to underlie the emergence of psychopathology. For example, brain structure (grey matter volume) in the prefrontal cortex relates to the p-factor, whereas specific internalizing problems relate to limbic areas (Snyder, Hankin, Sandman, Head, & Davis, 2017). Psychiatric genetic research also increasingly shows that all psychopathological disorders are highly polygenic and do not demonstrate unique links between disorder and genotype, and that polygenic risk scores may broadly relate to general psychopathology as well as unique internalizing and externalizing problems (Caspi & Moffitt, 2018; Consortium, 2013; Neumann et al., 2016; Smoller et al., 2018; Tackett et al., 2013; Waldman, Poore, van Hulle, Rathouz, & Lahey, 2016). Well-established personality and temperament risks, such as negative emotionality, positive emotionality, and cognitive (effortful) control, also have been shown to associate with both the p-factor and specific internalizing and externalizing dimensions in theoretically coherent and predicted ways (Caspi et al., 2014; Hankin et al., 2017; Olino, Dougherty, Bufferd, Carlson, & Klein, 2014). Last, chronic stress and childhood adversity predict, and are predicted by, the p-factor as well as the unique externalizing dimension (Schaefer et al., 2018; Snyder, Young, & Hankin, 2017a). Recent work also suggests that a latent factor approach not only relates to key risk pathways, but may yield a more parsimonious link to these underlying neurological structures and biophysiological processes than traditional, DSM-based diagnoses (for recent reviews, see Beauchaine & McNulty, 2013; Caspi & Moffitt, 2018; Hankin et al., 2016). Although additional work is needed to compare these approaches, these prior studies suggest that additional knowledge to advance understanding of risk for psychopathology may be gained with latent models of psychopathology as opposed to just utilizing traditional, singular diagnostic categories and syndromes of psychopathology, often with high degrees of comorbidity.

A few investigations have examined relations between maternal and offspring psychopathology when studied with latent psychopathology models. Both Martel and colleagues (Martel et al., 2017) and Michelini and colleagues (2019) found that the maternal p-factor was positively associated with the p-factor in the offspring, demonstrating that a general risk for psychopathology may be conferred from parent onto child (or vice versa, or due to shared risk factors such as genetic risk). Martel and colleagues (2017) also examined the relation between maternal p-factor and child specific latent factors of psychopathology and found that heightened maternal p-factor predicted specific fear and distress, but not a specific child externalizing factor, in a community sample of families in Brazil. Similarly, Starr and colleagues (Starr, Conway, Hammen, & Brennan, 2014) assessed a singular latent factor of internalizing psychopathology and found that the internalizing factor in both the mother and child were significantly related within a large, predominantly Caucasian, birth cohort of mother child dyads in Australia. Waldman and colleagues (Waldman et al., 2016) also found that the p-factor is heritable, linking maternal diagnostic history of depression in addition to other parental psychopathology factors to the child p-factor in a predominantly

Caucasian sample in the United States. However, additional work is needed to further evaluate how current maternal depressive symptoms relate to child psychopathology within a p-factor framework. This is an important contribution, because although maternal psychopathology is similarly not bound by traditional diagnostic categories, promoting a better understanding of the relation between maternal depressive symptoms and child transdiagnostic factors of psychopathology may provide important research and clinical implications. Specifically, application of a transdiagnostic framework to assess child outcomes provides useful context when interpreting the robust literature linking maternal depression to child mental health disorders. Additionally, maternal depressive symptoms are currently an easily measurable and identifiable risk factor that can be used for screening in clinical settings.

Although a well-established literature has reported children of depressed mothers and mothers with a history of depression to be at an elevated risk for mental health disorders in both the internalizing and externalizing domains, it remains unclear whether these associations are explained by what is common across internalizing and externalizing (i.e., a general risk for psychopathology as captured by the p-factor), or if maternal depression is separately associated with unique risk for internalizing and externalizing problems. Organizing offspring psychopathological outcomes within a latent bifactor model may therefore address this gap in the depression literature and could carry important clinical implications. First, exploring latent factors of psychopathology in children of mothers with elevated depressive symptoms may facilitate a new and informative characterization of child risk for psychopathology. Second, a latent structure approach to characterizing offspring psychopathology, rather than traditionally defined symptom categories, produces constructs which may more closely align with risk mechanisms (Hankin et al., 2016; Neumann et al., 2016; Waldman et al., 2016), and could facilitate greater understanding of the structure and etiology of psychopathology.

The current study utilizes a bifactor model to evaluate whether maternal depressive symptoms relate to both general (p-factor) and specific (internalizing and externalizing-specific) latent factors of psychopathology in children. We emphasize that we have chosen a bifactor model not on the basis of model fit, but rather based on its utility for addressing the study aims (e.g., Greene et al., 2019). Specifically, as reviewed above maternal depression is associated with both internalizing and externalizing symptoms in offspring, a pattern that could arise in one of two ways: via what shared between these symptom dimensions (i.e., general psychopathology), or separately for each (e.g., via different mediating mechanisms or genetic risks). A correlated factor model or manifest scale scores cannot differentiate between these possibilities, as each factor includes both shared and dimension-specific variance, either or both of which could drive associations with maternal depressive symptoms. The bifactor model disentangles these possibilities to clarify whether associations with maternal depressive symptoms are driven by what is shared across child internalizing and externalizing (p factor) and/or what is unique to each (specific factors).

Because this method of partitioning variance in psychopathology differs from traditional measurement scales of externalizing and internalizing psychopathology, such as in the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001), we also will replicate prior work

showing that maternal depressive symptoms are associated with greater total internalizing and externalizing composite scores on the CBCL (e.g., Tompson et al., 2010) and contrast these associations to those of the bifactor model. Finally, prior studies have reported sex differences in psychopathology and underlying etiological pathways (Rutter, Caspi, & Moffitt, 2003; Sutherland & Brunwasser, 2018) as well as gender differences in the mental health of children of depressed mothers (Goodman & Gotlib, 1999, Goodman et al., 2011; Sheeber et al. 2002). Additionally, the majority of prior study samples are predominantly Caucasian, and few have considered key sociodemographic variables (e.g., race/ethnicity) when assessing these relations between maternal depression and child mental health (Goodman et al., 2011). We therefore assess whether child sex/gender and race/ethnicity moderate the relation between maternal depressive symptoms and child latent factors of psychopathology within a sample of women and children from diverse backgrounds.

Method

Participants

Participants included 554 child-mother pairs. Participants were initially recruited through hospitals in the greater Los Angeles area, as part of two studies. Recruitment procedures for these cohorts are described elsewhere in detail (e.g., Davis et al., 2013; Glynn et al., 2018; Glynn et al., 2019; Sandman, Davis, Buss, & Glynn, 2012). For the present investigation, participants from these cohorts were administered measures of maternal depressive symptoms and child psychopathology following the same protocol and data was combined for the current study analyses (see Hankin et al., 2017). Two-hundred and seventy-five mother-child dyads from the first cohort and 279 mothers-child dyads from the second cohort were included in current study analyses. No additional exclusion criteria were applied.

In the current study sample, children on average were 7.7 years of age ($SD = 1.35$, range = 5–11 years). Parents identified their child's ethnicity as 46% Latinx and identified their child's race as 67% White, 6% Black, 5% Asian/Pacific Islander, and 21% multiracial; 49.8% of youth in the sample were female. Median annual family income was \$75,000. Rates of clinically elevated symptoms on the Child Behavior Checklist DSM-oriented scales ranged from 4% for affective problems and ADHD to 9% for anxiety, consistent with epidemiological studies (Costello, Copeland, & Angold, 2016).

Child sex was determined via medical record abstraction at birth, and parents reported on the gender of their child at the time of assessment. Biological sex and parent report of child gender were consistent for all participants. We therefore refer to "sex/gender" (Fausto-Sterling, 2012) throughout this manuscript because prior studies suggest that pathways related to both child gender (e.g., cultural and societal factor related to gender expression) and biological sex (e.g., biological factors related to child sex) are implicated in the intergenerational transmission of psychopathological risk (Altemus, 2006; Goodman & Gotlib, 1999; Goodman et al., 2011; Hyde, Mezulis, Abramson, 2008; Rutter, Caspi, & Moffitt, 2003; Sheeber, Davis, & Hops, 2002; Sutherland & Brunwasser, 2018). Using this criterion, children within the study sample were 50.2% female ($n = 278$) and 49.8% male ($n = 276$).

All procedures were approved by the University of California, Irvine and the Long Beach Memorial Medical Center Institutional Review Boards. Mothers provided informed, written consent for both themselves and their children. Children provided verbal assent.

Measures

Maternal depressive symptoms—Maternal depressive symptoms were assessed using the 21-item Beck Depression Inventory – II (BDI-II; Beck, Steer, & Brown, 1996). Participants provided ratings of how often they experienced a symptom of depression in the past week, on a 4-point Likert scale with a range of 0 to 3 (e.g., “*I do not feel sad*” is a 0 whereas “*I am so unhappy that I can’t stand it*” is a 3). Final sum scores could range from 0 to 63, with higher scores indicating more symptoms of depression. The BDI-II demonstrated excellent internal consistency within the study sample ($\alpha = .92$) and has been shown in prior work to be a well validated measure of depression (Beck, Steer, & Brown, 1996; Dozois, Dobson, & Ahnberg, 1998).

Child psychopathology

Child Behavior Checklist.: Child psychopathology was measured using the parent report form of the Child Behavior Checklist (Achenbach & Rescorla, 2001) from the Achenbach System of Empirically Based Assessment. The CBCL is a widely used measure of youth mental health and behavioral problems, and demonstrates good test-retest reliability, and discriminant, convergent and predictive validity with other measures of psychopathology, including DSM clinical diagnoses (Achenbach & Rescorla, 2001). Both the internalizing and externalizing composite scores on the CBCL demonstrated good internal consistency within the current study sample ($\alpha = .84$ and $.89$ respectively).

The CBCL contains items representing a broad scope of behaviors. Responses were made by mothers on a 3- point Likert scale ranging from 0 (*not true*) to 2 (*very true*). The CBCL includes the following subscales: Aggressive Behavior (18 items, e.g., “Gets in many fights”, “Cruelty, bullying or meanness to others”), Anxious/Depressed (13 items, e.g., “Worries”, “Cries a lot”), Attention Problems (10 items, e.g., “Can’t concentrate, can’t pay attention for long”, “Can’t sit still, restless or hyperactive”), Rule-Breaking Behavior (17 items, e.g., “Breaks rules and home, school or elsewhere” “Doesn’t seem to feel guilty after misbehaving”), Thought Problems (15 items, e.g., “Can’t get his/her mind of certain thoughts/obsessions”, “Strange behaviors”), Somatic Complaints (11 items, e.g., “Stomachaches”, “Headaches”), Social Problems (11 items, e.g., “Doesn’t get along with other kids”, “Complains of loneliness”), and Withdrawn/Depressed (8 items, e.g., “There is very little he/she enjoys”, “Unhappy, sad or depressed”).

In the CBCL scoring system, the Anxious/Depressed, Withdrawn/Depressed and Somatic Complaints subscales are classified as assessing an internalizing dimension and the Aggressive Behavior and Rule-Breaking Behavior as assessing an externalizing dimension, yielding internalizing and externalizing composite scores (i.e., raw sum scores for internalizing and externalizing subscales respectively.). The remaining subscales (Attention, Social and Thought Problems) are not assigned to either the internalizing or externalizing scales but are included in the total problem measure.

Children's Behavior Questionnaire: Two subscales from Children's Behavior Questionnaire (CBQ; Rothbart, Ahadi, Hershey, & Fisher, 2001) were also included to improve coverage of child psychopathology, incorporate symptoms not fully assessed in the CBCL, and increase the number of indicators for latent variable modeling. Selected CBQ subscales include Anger/Frustration (13 items, e.g., "Easily gets irritated when he/she has trouble with some task", "Gets mad when provoked by other children") and Fear (12 items, e.g., "Is afraid of the dark", "Is afraid of loud noises"). Responses were made by mothers on a 7-point Likert scale ranging from 1 (extremely untrue of your child) to 7 (extremely true of your child). The CBQ is widely used in research with children and has good test-retest and inter-rater reliability and convergent validity (e.g., Rothbart et al., 2001; Rothbart, Ahadi, Hershey, & Fisher, 2001). Internal consistency of the Anger/Frustration and Fear subscales of the CBQ ranged from acceptable to good within the current study sample ($\alpha = .82$ and $.75$ respectively).

Statistical analysis

P factor measurement model—Structural equation modeling (SEM) was conducted in Mplus (Muthén & Muthén, 2012) using full information maximum likelihood with robust standard errors (MLR, which does not assume normal distributions) estimation to handle missing data. Missing data rates for all measures administered were low (4%). For all models, we considered various factors to evaluate best fitting models, including parsimony and conceptual consistency, but also conservative "rules of thumb" in which good fit was defined as root mean square error of approximation < 0.06 , comparative fit index > 0.95 , Tucker–Lewis index > 0.95 , and standardized root mean square residual < 0.08 (Hu & Bentler, 1999). Each individual fit index has strengths and limitations; no consensus has been reached on a single fit index to evaluate model fit (Loehlin, 2004).

A bifactor confirmatory factor analysis (CFA) of the psychopathology measures was identical to that in a previous study with a subset of these participants (Hankin et al., 2017). Specifically, we followed the standard scoring of the CBCL into internalizing (Anxious/Depressed, Withdrawn/Depressed and Somatic Complaints), externalizing (Rule Breaking Behavior, Aggressive Behavior) and other (Social Problems, Thought Problems, Attention Problems) subscales (Achenbach & Rescorla, 2001). The CBQ Fear and Anger/Frustration subscales were included as additional internalizing and externalizing measures respectively. All raw-score measures were loaded onto the p-factor. Internalizing measures were additionally loaded onto an internalizing-specific factor and externalizing measures onto an externalizing-specific factor, capturing unique variance not accounted for by the p-factor. Factors were constrained not to correlate because what is shared between factors is already captured by the common factor (e.g., Chen, Hayes, Carver, Laurenceau, & Zhang, 2012). Modification indices were inspected and residual correlations were added between the Thought Problems subscale and the Somatic Complaints and Anxious/Depressed subscales. There is clear overlap between some items on the Thought Problems and Anxious/Depressed subscales (e.g., "deliberately harms self or attempts suicide" from Thought Problems and "talks about killing self" from Anxious/Depressed, which likely accounts for their residual correlation. Thought Problems also includes somatic behaviors (e.g., "picks skin") which are likely related to Somatic Complaints items (e.g., "skin problems"), again likely accounting

for the residual correlation. Factor loadings were quite similar across the models, with a mean absolute difference of .021, and a maximum absolute different of .052. SEM results were nearly identical for models with and without the residual correlations.

SEM models with latent factors of child psychopathology—Structural equation models tested the association of maternal depressive symptoms (i.e., BDI score) with each child psychopathology factor (p-factor, internalizing-specific, externalizing-specific), adjusting for child age and sex/gender. Models were tested using the residual method (Koch, Holtman, Bohn, & Eid, 2018), which provides unbiased estimates of the relation between the predictor and the general factor, free of influences of the specific factors, and vice versa. Specifically, effects are tested in two models. In the first model, the explanatory variable (in the current study, maternal BDI) is regressed on the latent specific factors (in the current study, internalizing- and externalizing-specific factors), and the residual of this latent regression is defined as latent variables (residual maternal BDI); the general factor (p-factor) is then regressed on the latent residual variable, and the partial regression weight is the unbiased effect of the predictor free of specific factor influences (Koch et al., 2018). In the second model, the reverse is done, with the explanatory variable regressed on the latent general factor, the residual of this latent regression is defined as a latent variable, and the latent specific factors then regressed on this latent residual variable (Koch et al., 2018). Finally, we also tested for possible moderation by sex/gender and race/ethnicity, by running multi-group models of the above SEMs. For race/ethnicity, the two groups most represented within our sample include children identifying as non-Latinx White ($n = 189$, 34%) and Latinx ($n = 255$, 46%). We therefore assessed race/ethnicity moderation two ways, non-Latinx White vs. all others and Latinx vs. non-Latinx. In moderation analyses, the psychopathology measurement model factor loadings and intercepts were constrained to be the same across groups, but the regressions were freely estimated in each group, and group differences in the association of maternal BDI with child psychopathology were tested with Wald tests.

Regression models with traditional, CBCL composite scores of child psychopathology—Regressions were also conducted using SPSS to test the association of maternal depressive symptoms and child internalizing and externalizing total composite scores on the CBCL, adjusting for child age and sex/gender. Internalizing and externalizing symptoms were tested in separate regressions, in parallel to the SEM models. Finally, we also tested for possible moderation by sex/gender and by race/ethnicity (i.e., non-Latinx White vs. all others and Latinx vs. non-Latinx) by running the above regressions with child sex/gender x maternal BDI interactions and then with race/ethnicity x maternal BDI interactions.

Results

P factor measurement model

The p-factor measurement model achieved excellent fit (CFI = .988, TLI=.980, RMSEA=.038 (90% CI = .020–.055, probability of close fit = .864), SRMR=.027), and all indicators loaded significantly on their specified factors (Table 1; Figure 1).

SEM with latent factors of child psychopathology

Results are reported in Table 2. All analyses controlled for child age and sex/gender. There was an unbiased (i.e., free of specific-factor influences) association between elevated maternal depressive symptoms and higher p-factor in the child ($\beta = 0.526$, $SE = 0.048$, $z = 10.92$, $p < .001$; $R^2 = .285$). There was no unbiased association between maternal depressive symptoms and the child internalizing-specific factor ($\beta = -0.005$, $SE = 0.106$, $z = -0.04$, $p = .966$), or externalizing-specific factor ($\beta = -0.07$, $SE = 0.062$, $z = -1.27$, $p = .204$).

Sex/Gender and Race/Ethnicity—The p factor was significantly higher in boys than girls, but with a very small effect size ($\beta = -0.099$, $SE = 0.046$, $z = -2.13$, $p = .033$; $R^2 = .010$). There was no difference in the specific factors ($ps > .30$), or in maternal depression levels ($p = .362$) between boys and girls. There were no differences in the unbiased associations between maternal depression and the p factor (Wald test = 1.93, $p = .165$), internalizing-specific factor (Wald test = 1.73, $p = .189$), or externalizing-specific factor (Wald test = 0.15, $p = .701$; Table S3).

Child race/ethnicity, whether defined as non-Latinx White vs. all others or Latinx vs. non-Latinx, was not associated with any child psychopathology factor ($ps > .10$). However, maternal BDI scores were higher for Latinx ($M = 8.70$, $SD = 9.61$) than non-Latinx ($M = 6.48$, $SD = 7.06$; $t(539) = -3.01$, $p = .003$) participants, and were lower for non-Latinx White participants ($M = 6.47$, $SD = 7.00$) in comparison to all others ($M = 8.03$, $SD = 9.01$; $t(539) = -2.22$, $p = .027$). There was no difference in the unbiased associations between maternal depression for non-Latinx White vs. all other children (p factor: Wald test = 0.04, $p = .844$; internalizing-specific factor Wald test = 1.23, $p = .268$; externalizing-specific factor: Wald test = 0.28, $p = .597$; Table S5) or Latinx vs. non-Latinx children (p factor: Wald test = 3.49, $p = .062$; internalizing-specific factor Wald test = 1.13, $p = .288$; externalizing-specific factor: Wald test: 0.03, $p = .865$; Table S4). Maternal depression was associated with the p factor in all groups, and associations with the specific factors were non-significant in all groups (Tables S4–S5).

Regression analyses with traditional CBCL composite scores of child psychopathology

Results are reported in Table 3. Maternal depressive symptoms were positively associated with the internalizing composite score ($\beta = 0.38$, $SE = 0.04$, $t = 9.59$, $p < .001$) and the externalizing composite score on the CBCL ($\beta = 0.38$, $SE = 0.04$, $t = 9.70$, $p < .001$). Results from SEM models with correlated latent internalizing and externalizing factors were similar to results from these analyses with CBCL composite scores (Table S7).

Sex/Gender and Race/Ethnicity—Child sex/gender did not moderate the relation between maternal depressive symptoms and the internalizing composite score ($\beta = 0.02$, $t = .32$, $p = .752$) or externalizing composite score ($\beta = -0.10$, $t = -1.80$, $p = .073$).

Child race/ethnicity, whether defined as non-Latinx White vs. all others or Latinx vs. non-Latinx, was not associated with either internalizing or externalizing composite score ($ps > .10$). Additionally, child race/ethnicity did not moderate the relation between maternal depressive symptoms and internalizing composite scores (non-Latinx White vs. all others: β

= $-.20$, $t = -1.11$, $p = .267$; Latinx vs. non-Latinx: $\beta = -.05$, $t = -.69$, $p = .490$). Child race/ethnicity also did not moderate the relation between maternal depression and externalizing composite score when child race/ethnicity was coded as non-Latinx White vs. all others ($\beta = -.08$, $t = -.46$, $p = .648$; Latinx vs. non-Latinx: $\beta = .055$, $t = -.69$, $p = .490$), however moderation was significant when child race/ethnicity was coded as Latinx vs. non-Latinx ($\beta = -.21$, $t = -2.77$, $p = .006$). Follow up regression analyses (split by Latinx vs. non-Latinx) revealed that maternal depressive symptoms were positive associated with the externalizing composite for both Latinx ($\beta = .36$, $t = 6.03$, $p < .001$) and non-Latinx children ($\beta = .41$, $t = 7.79$, $p < .001$), however this association was slightly weaker amongst Latinx children.

Discussion

Maternal depression has been established as a robust predictor of many child psychopathologies, spanning across both internalizing and externalizing domains of functioning (Goodman et al., 2011). Although these associations have been well documented, how maternal depression relates to transdiagnostic and specific risk for child psychopathologies remains less understood. The findings of the current study expand our understanding of maternal depression and child mental health by exploring these associations within a latent bifactor model. Using this alternative model of psychopathology, we find that maternal depressive symptoms are associated with the p-factor in the child, reflecting a general liability for broad, co-occurring psychopathology (i.e., what is shared across all domains of psychopathological symptoms). After broad co-occurring psychopathology is accounted for, maternal depressive symptoms were not associated with the unique variance specific to internalizing or externalizing psychopathology in the offspring. To further explore the utility of the latent bifactor approach, findings were contrasted with more traditional approaches of assessing internalizing and externalizing psychopathological symptoms (using CBCL composite scores and correlated factors models) (Goodman et al., 2011). Specifically, maternal depressive symptoms were shown to be associated with both elevated internalizing as well as externalizing composite scores on the CBCL and factors in the correlated factor model, replicating prior findings that children of mothers with elevated depressive symptoms are more likely to exhibit symptoms for both internalizing and externalizing psychopathological disorders (Goodman et al., 2011). Taken together, results from the latent bifactor approach suggest that the observed elevations in total child internalizing and externalizing problems may be a product of pathways related to broad, transdiagnostic risk for general psychopathology (i.e., the p-factor) rather than specific risk for the aspects of internalizing and externalizing symptoms that are unique to each. Findings therefore demonstrate how this latent model of psychopathology may shed new light on our understanding of how maternal depression relates to child psychopathological outcomes.

These findings are consistent with the limited prior work linking maternal psychopathology to child latent factors of psychopathy. Specifically, prior studies observed positive associations relating the maternal p-factor and history of a maternal diagnosis of depression to the child p-factor (Martel et al., 2017). The current study is also consistent with the robust literature linking maternal depression to broad and co-occurring child psychopathologies, including both internalizing and externalizing symptoms and disorders (Goodman et al.,

2011). Given the prevalence of depression and well-established literature linking maternal depression to child mental health problems, these findings fill an important gap by characterizing how children of mothers with elevated depressive symptoms are at risk for psychopathology.

We found that neither child sex/gender nor race/ethnicity moderated the association between maternal depressive symptoms and latent factors of psychopathology. The current findings suggest that although the p factor is slightly higher in boys than girls, the relation between maternal depression and child psychopathology does not differ on the basis of sex/gender. These results are consistent with prior studies reporting that the p-factor is higher in boys than girls (Huan, Shapiro, Galloway-Long, & Weigard, 2017), as well as meta-analytic evidence reporting that maternal depression is equally associated with general psychopathology in boys and girls (Goodman et al., 2011). Although we do not replicate prior findings of sex/gender differences in the relation between maternal depression and internalizing symptoms (Goodman et al., 2011), past work suggests that sex/gender differences in internalizing disorders and the internalizing-specific factor may not emerge until later in adolescence (Angold, Erkanli, Silberg, Eaves, & Costello, 2002; Hayward & Sanborn, 2002; Huang-Pollock, Shapiro, Galloway-Long, & Weigard, 2017). Future work is needed to continue to explore potential sex/gender-based differences in vulnerability to maternal depression across development. Similarly, although rates of maternal depression are higher amongst mothers of racial or ethnic minority children, child race/ethnicity did not moderate the relation between maternal depression and child latent factors of child psychopathology. Findings were comparable when child psychopathology was assessed via internalizing and externalizing composite scores on the CBCL, although the relation between maternal depressive symptoms and child externalizing scores was slightly weaker but also significant for Latinx children. Although prior work has suggested that maternal depression is a stronger predictor of internalizing and externalizing problems among ethnic minority women and children, this work has been quite limited and findings have been mixed (Goodman et al., 2011). Future studies could expand upon this literature by more directly assessing the processes underlying these potential risk pathways, such as ethnic minority stress, acculturation, and enculturation. These processes and experiences may also vary across different communities and individuals. Replication in other diverse samples is therefore critical.

The current study has several strengths. First, a large sample of mother-child dyads participated ($N= 554$). Second, we utilized both a traditional CBCL approach to evaluate child psychopathological symptoms, as well as a new, reorganized latent bifactor approach. Utilizing both approaches within the same study sample provides compelling evidence that updating the way in which we structure and characterize child psychopathology may shed new light on our understanding of how maternal depression relates to child mental health outcomes. Third, the study sample included women and children from diverse racial and ethnic backgrounds, with almost half of the sample identifying as Latinx and only one third of the sample identifying as non-Latinx White. This racial/ethnic diversity of the study sample supports the generalizability of study findings to a more inclusive range of women and children.

There are several limitations in this study which can be addressed in future research. First, data were collected at a single time point. Although establishing these associations and their magnitude are an important first step, future studies may utilize longitudinal study designs to assess the temporal emergence of latent psychopathological factors and to identify periods of increased vulnerability to maternal depression, as well as examine the potential effects of child psychopathology on maternal depressive symptoms over time. Second, mothers reported on both their own depressive symptoms as well as the psychopathology symptoms in their child. Using a maternal report measure offers many advantages, because the mother has the opportunity to observe and report on the child's socioemotional and behavioral symptoms across a variety of contexts. However, maternal report could also be confounded by symptoms of maternal depression and other psychopathologies, as elevated maternal depressive symptoms have a small yet significant impact on reporting biases (Youngstrom, Izard, & Ackerman, 1999). Future studies may therefore utilize a combination of multi-rater reporting, structured interviewing, and self-report to assess and minimize reporter biases. Third, latent bifactor models of psychopathology, while statistically valid, may sacrifice specificity of diagnostic categories. However, this approach still offers promise as it could help to further elucidate mechanisms of psychopathological risk, which are likely not bound by traditional diagnostic categories. Fourth, latent bifactor models of psychopathology have been critiqued because traditional criteria for model fit favors a bifactor modeling approach (e.g., Bonifay, Lane, & Reise, 2017; Morgan, Hodge, Wells, & Watkins, 2015; Watts, Poore, & Waldman, 2019). It is therefore important to continue to emphasize not just model fit, but whether a latent bifactor approach provides additional validity in the characterization of psychopathological symptoms (Hankin, 2019).

The current findings have implications for clinical practice and research. Ongoing discussion of optimal diagnostic classification systems and organizational structures will continue. As new approaches continue to emerge, future work should continue to evaluate the relation between maternal depression and child psychopathology from these differing perspectives (e.g. Michelini et al., 2019). An increasing number of studies are employing dimensional and latent factor approaches to characterize psychopathology and are demonstrating its potential utility in research designs. The latent bifactor model follows this line of scientific inquiry by demonstrating that a latent structure of organizing psychopathological symptoms provides a unique perspective when applied to characterize child mental health and its relation to maternal depression, particularly when contrasted to traditional, Achenbach style approaches. The current findings also reinforce that children of depressed mothers should be screened for broad psychopathological risk, not just depression or internalizing disorders.

Future work also may explore the etiological pathways leading to the emergence of these psychopathological traits. Plausible mechanisms proposed to underlie the relation between maternal depression and offspring general psychopathology liability include but are not limited to genetic heritability, shared environment, fetal vulnerability to maternal depression, and maladaptive parenting (Hankin et al., 2016; Snyder, Young, et al., 2017b). Although further work is needed to explore these hypothetical risk pathways, this line of scientific inquiry may facilitate the development of efficacious early screenings and targeted interventions for these at-risk mother-child pairs.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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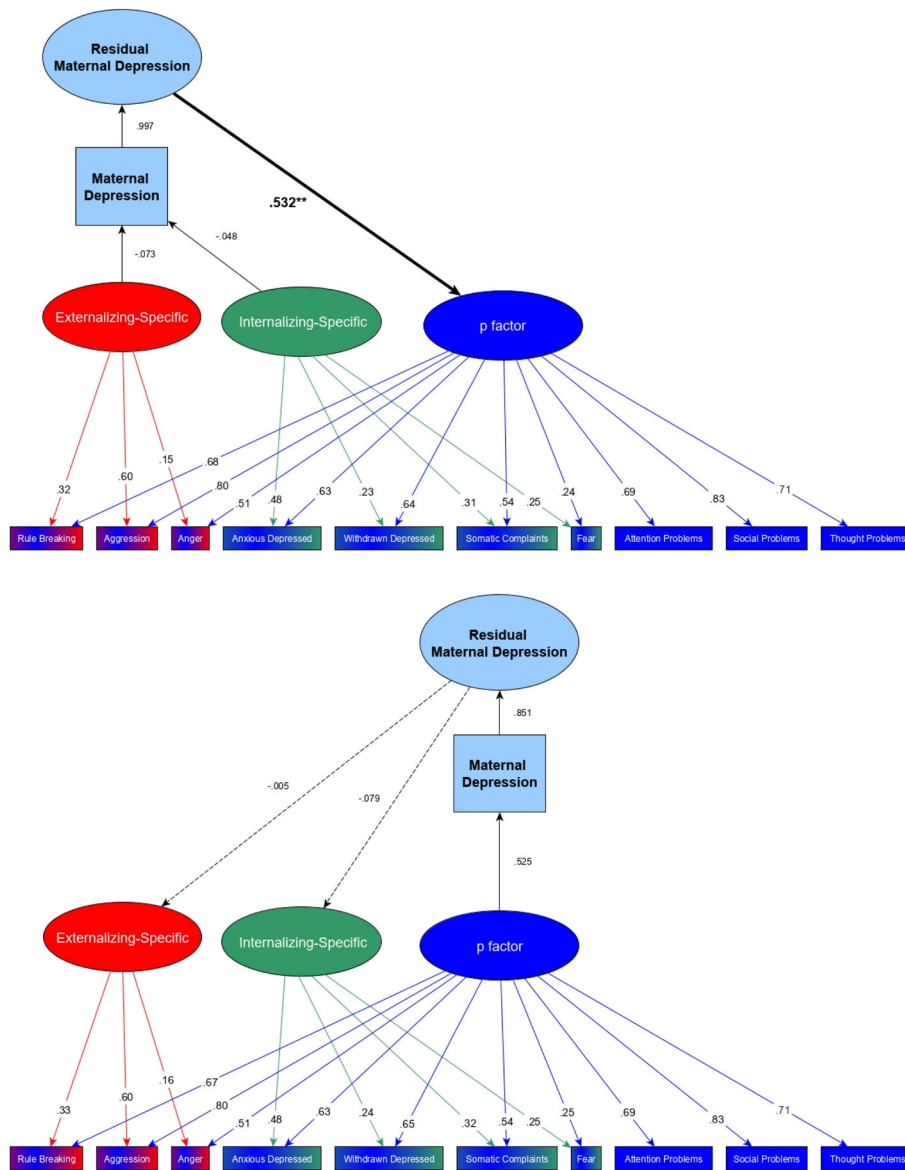


Figure 1. Maternal depressive symptoms and latent factors of child psychopathology.

Table 1.

Descriptive Statistics and Standardized Factor Loadings for Child Psychopathological Symptoms.

Measure	Mean	SD	n	Standardized Factor Loadings		
				p-factor	Internalizing-specific	Externalizing-specific
CBCL Anxious/Depressed	3.17	3.00	553	.632	.460	-
CBCL Withdrawn/Depressed	1.32	1.81	553	.634	.241	-
CBCL Somatic Complaints	1.77	2.11	548	.533	.321	-
CBQ Fear	3.98	1.00	540	.245	.248	-
CBCL Rule Breaking	1.77	2.01	553	.672	-	.331
CBCL Aggressive Behavior	4.75	5.00	553	.803	-	.596
CBQ Anger/Frustration	4.10	0.88	540	.509	-	.160
CBCL Attention Problems	3.62	3.31	553	.695	-	-
CBCL Social Problems	2.53	2.63	553	.825	-	-
CBCL Thought Problems	2.09	2.55	553	.714	-	-
Maternal BDI	7.50	8.40	541	-	-	-

Note. All factor loadings are significant $p < .01$. CBCL = Child Behavior Checklist. CBQ = Children's Behavior Questionnaire. BDI = Beck's Depression Inventory.

Table 2.

SEM Models of Maternal Depressive Symptoms and Latent Dimensions of Child Psychopathology from Bifactor Modeling.

Model	Predictors	β	SE	z	p
Model 1: Unbiased associations with p-factor (CFI=.964, TLI=.948, RMSEA=.051, SRMR=.037)					
p-factor	Maternal BDI	.526	.048	10.92	< .001**
	Child age	-.048	.046	-1.06	.290
	Child sex/gender	-.074	.039	-1.87	.061
Model 2: Unbiased associations with specific factors (CFI=.964, TLI=.947, RMSEA=.051, SRMR=.040)					
Internalizing-specific	Maternal BDI	-.005	.106	-0.04	.966
	Child age	.037	.086	0.44	.662
	Child sex/gender	.053	.093	0.57	.566
Externalizing-specific	Maternal BDI	-.079	.062	-1.27	.204
	Child age	-.127	.044	-2.85	.004*
	Child sex/gender	-.053	.051	-1.05	.293

Note. Child sex/gender coded -1 males, 1 females. CBCL = Child Behavior Checklist. CBQ = Children's Behavior Questionnaire. BDI = Beck's Depression Inventory.

*
 $p < .05$

**
 $p < .001$

Table 3.

Regression Models of Maternal Depressive Symptoms and Internalizing and Externalizing Composite Scores on the CBCL.

Outcome	Predictors	β	SE	<i>t</i>	<i>p</i>
Internalizing Symptoms	Maternal BDI	.382	.039	9.59	<.001***
	Child age	.026	.039	.65	.517
	Child sex/gender	-.025	.039	-.64	.525
Externalizing Symptoms	Maternal BDI	.384	.039	9.70	<.001***
	Child age	-.087	.039	-2.21	.028**
	Child sex/gender	-.084	.039	-2.13	.033*

Note. Child sex/gender coded -1 males, 1 females. CBCL = Child Behavior Checklist. BDI = Beck's Depression Inventory.

*
p < .05

**
p < .001