Title

Latent Class Analysis of Maternal Depression From Pregnancy Through Early Childhood: Differences in Children's Executive Functions

Journal

Developmental Psychology, 59(8)

ISSN

0012-1649

Authors

Choe, Daniel Ewon
Deer, LillyBelle K
Hastings, Paul D

Publication Date

2023-08-01

DOI

10.1037/dev0001540

Latent Class Analysis of Maternal Depression from Pregnancy through Early Childhood: Differences in Children's Executive Functions

Daniel Ewon Choe [1] *, LillyBelle K. Deer [2], and Paul D. Hastings [3]

[1] Department of Human Ecology, University of California, Davis

[2] Department of Psychology, University of Denver

[3] Department of Psychology, University of California, Davis

*Corresponding author

THIS PREPRINT IS ACCEPTED BY *DEVELOPMENTAL PSYCHOLOGY*, HAS YET TO UNDERGO COPYEDITING, AND MAY DIFFER FROM ITS PUBLISHED VERSION.

Author Note

The UK Medical Research Council and Wellcome (Grant ref: 217065/Z/19/Z) and the University of Bristol provide core support for ALSPAC. This publication is the work of the authors who serve as guarantors for the contents of this paper. A comprehensive list of grants funding is available on the ALSPAC website (http://www.bristol.ac.uk/alspac/external/documents/grant-acknowledgements.pdf); This manuscript was prepared with support from the Center for Poverty and Inequality Research at University of California, Davis and funding provided by NSF Grant 1327768, as well as a Hellman Fellowship awarded to the first author.

Abstract

Prenatal and postpartum depression are highly prevalent worldwide, and emerging evidence suggests they contribute to impairments in children's executive functions. Studies of maternal depression, however, have focused on the postpartum and postnatal periods with relatively less consideration of prenatal influences on child development. This study of the large population-based Avon Longitudinal Study of Parents and Children (ALSPAC) U.K. cohort estimates latent classes of maternal depression across the prenatal, postpartum, *and* postnatal periods to capture heterogeneity in the developmental timing and length of maternal depression, as well as to test whether latent classes differ in children's executive function impairments in middle childhood. Repeated measures latent class analysis yielded five groups demonstrating unique patterns of change in maternal depression from pregnancy through early childhood (N = 13,624). Latent classes differed in executive functions at age 8 among a subsample of children (N = 6,870). Children exposed to chronic maternal depression beginning in utero showed the most impairments in inhibitory control, while accounting for child sex, verbal IQ, parents' highest education level, and average family income in childhood. The critical roles of the timing and length of children's exposure to maternal depression are discussed in relation to executive function development, prevention, and intervention.

Keywords: ALSPAC, maternal depression, prenatal, postpartum, postnatal, executive functions

Public Significance Statement: Results of this longitudinal birth cohort study suggest that children exposed to maternal depression persistently through pregnancy and early childhood are less able to control their thoughts and memories than children exposed to maternal depression temporarily after birth or not at all. The results add to the growing number of studies that have linked early exposure to maternal depression to impairments in children's executive functions.

Latent Class Analysis of Maternal Depression from Pregnancy through Early Childhood: Differences in Children's Executive Functions

Prenatal and postpartum depression are highly prevalent worldwide (Gelaye et al., 2016). Studies of maternal depression and child development have largely focused on the postpartum and postnatal periods, which we differentiate as the first year (O'Hara & McCabe, 2013) vs. the second and third years after birth, respectively (Choe et al., 2020). Exposure to maternal depression in infancy and early childhood is related to children's heightened externalizing (Shaw et al., 2003) and internalizing problems (Choe et al., 2020), poor language development (McManus & Poehlmann, 2012; Sohr-Preston & Scaramella, 2006), suboptimal self-regulation (Choe et al., 2013, 2014), and executive function impairments (Baker & Kuhn, 2017; Hughes et al., 2013). Prenatal depression is also strongly related to child outcomes, but fewer studies have examined its effects on children as opposed to postpartum depression, and only more recently have the potential consequences of prenatal depression garnered more research focus (Field, 2011). One topic of this focus has been children's executive functions (EFs), the top-down control processes that emerge early in life as a unidimensional construct but gradually differentiate with age into working memory, inhibitory control, and cognitive flexibility; skills that promote goal-directed behavior and self-regulation (Best & Miller, 2010; Diamond, 2013; Park et al., 2018). A recent meta-analysis of 16 studies linking perinatal depression, covering conception through one year after birth, to child EFs reported a negligible difference between prenatal and postpartum depressions' modest effect sizes (Power et al., 2021). Hence, despite prenatal depression being the focus of less research, it may convey comparable risk for children's EF development as exposure to maternal depression after birth.

Depression is a cyclical disorder with varying trajectories of severity. Patterns of change

in mothers' depressive symptoms are related to individual differences in children's cognitive and socioemotional functioning (Campbell et al., 2009; Park et al., 2018). At least one population-level longitudinal study has reported distinct patterns of change in maternal depression across the prenatal, postpartum, *and* postnatal periods (Cents et al., 2013), but it remains unclear whether population-level heterogeneity in maternal depression across this developmental span is related to impairments in children's EFs. Attention to this matter is warranted, as the optimal development of EFs promotes adaptive functioning across a myriad of domains, such as self-regulation, school readiness, and mental health (Blair & Raver, 2015). For example, a recent meta-analysis found poor EFs in childhood predict future externalizing and internalizing problems (Yang et al., 2022). Diamond (2013) argues that EFs and their neural underpinnings serve as an "early warning system" when life goes awry, as they show some of the earliest signs of harm when people are stressed, sad, lonely, sleep deprived, or physically unhealthy. We therefore focus on impairments in children's EFs as they are implicated in a wide range of cognitive and socioemotional problems. This study leverages data from the large population-based Avon Longitudinal Study of Parents and Children (ALSPAC), which targeted all pregnant women in the former county of Avon in the southwest of England (Fraser et al., 2013). We estimate latent classes of change in maternal depression throughout pregnancy and early childhood and use these latent classes to determine whether the developmental timing and length of exposure to maternal depression have implications for children's EFs.

Prenatal Depression and Child Executive Functions

Prevalence estimates suggest 10% to 30% of women experience prenatal depression, or antenatal depression, as it is termed in many countries (Gelaye et al., 2016; Mukherjee et al., 2016). Prenatal depression increases mothers' risk of re-experiencing depression after pregnancy,

as experiencing a depressive episode increases the odds of future episodes (Field, 2011; O'Hara & McCabe, 2013). Chronic exposure to maternal depression beginning early in childhood is more strongly related to school-age internalizing and externalizing problems compared to intermittent exposure or exposure starting later in childhood (Campbell et al., 2007; Goodman et al., 2011), which suggests the timing and length of exposure to maternal depression are critical factors that determine the extent of depression's impact on child outcomes. We use the terms *maternal depression* and *maternal depressive symptoms* interchangeably because both mothers' diagnosed depression and high symptom ratings predict children's suboptimal outcomes across development (Meaney, 2018). Thus, children exposed to chronic maternal depression from the prenatal period through early childhood are expected to show more impaired EFs than children exposed to maternal depression intermittently or only later in development.

Although some studies have found postpartum depression predicts child behavior problems more strongly than does prenatal depression (Bagner et al., 2010; van der Waerden et al., 2015a), research with the ALSPAC sample has shown prenatal depression predicts some child outcomes more strongly than postnatal depression. Compared to other longitudinal studies, many of which collected limited prenatal data, sometimes with retrospective reports or short-term follow-up data in childhood (Campbell et al., 2007), the ALSPAC study collected extensive perinatal and postnatal data prospectively. Prior ALSPAC studies found that relative to postnatal depression, prenatal depression more powerfully predicts developmental delays at 18 months old (Deave et al., 2008), emotional–behavioral dysregulation from 2 to 7 years old (Pina-Camacho et al., 2014), and cognitive impairments at 8 years old (Barker et al., 2013). Therefore, prior studies of the ALSPAC sample suggest that children exposed to only prenatal depression may be more likely to show EF impairments than children exposed to maternal depression only after birth.

The *developmental origins of health and disease* framework posits that the fetus prepares for the postnatal environment by adapting to cues in the prenatal environment (e.g., maternal stress and nutrition) to optimize survival during both developmental periods (Camerota & Willoughby, 2021). Prenatal stress alters fetal brain development, as well as autonomic nervous system, hypothalamus-pituitary-adrenal (HPA) axis, and immune functioning in utero (Davis et al., 2018; Van den Bergh et al., 2020) with long-term effects on neurodevelopment and cognitive functioning (Robinson et al., 2019). In support of Diamond's (2013) "early warning system" argument, prenatal depression and stress are associated with functional and structural changes in fetal brain development in neural substrates of EFs and the regulation of HPA-axis functioning, specifically the prefrontal cortex and amygdala (Davis et al., 2018; Robinson et al., 2018; Van den Bergh et al., 2020). Prenatal alterations in stress reactivity or regulation may in turn increase offspring vulnerability to the effects of postpartum and postnatal depression, resulting in a wider range of cognitive and socioemotional problems. Prenatal depression may alter EF development, but the limited empirical research has yielded inconsistent evidence as to whether it predicts specific or global impairments in EFs (Robinson et al., 2019; Van den Bergh et al., 2020).

Postpartum and Postnatal Depression

A meta-analysis of postpartum depression reported a worldwide prevalence rate of 17% of healthy mothers with no prior history of depression (Shorey et al., 2018). Children in the U.K. are most often exposed to maternal mental illness in the first three months postpartum, and depression is the most common mental illness mothers experience in the U.K. (Abel et al., 2019). Exposure to postpartum depression, in turn, predicts children's poor language and cognitive skills, including IQ scores, mediated in part by compromised parenting (O'Hara & McCabe, 2013; Sohr-Preston & Scaramella, 2006). Recent studies have shown young children's EFs,

language skills, and general cognitive ability are separable but interrelated constructs (Gooch et al., 2016; Hughes & Devine, 2019), so impairments in one domain related to exposure to postpartum depression may generalize to other domains. Evidence of the enduring impact of early exposure to maternal depression on children's EFs is slowly emerging in the literature.

Prior research with the ALSPAC sample found mothers' postpartum depressive symptoms, averaged across 2 and 8 months, were negatively related to 8-year-old's attentional control and attentional switching but not their working memory or selective attention (Pearson et al., 2016). A dose–response relation showed corresponding increases in the severity of mothers' postpartum symptoms and children's impairments. This study, however, accounted for prenatal anxiety, as opposed to prenatal depression, to capture distinct pathways of biological risk during pregnancy, as it was noted that prenatal and postnatal depressive symptoms were highly intercorrelated ($r > .70$). Prenatal depression is a stronger predictor of children's socioemotional problems than prenatal anxiety (Madigan et al., 2018), but whether prenatal depression predicts child EFs independently of, or incrementally over, postpartum depression remains untested. Furthermore, no studies of the ALSPAC sample have examined longitudinal patterns of change in maternal depression from the prenatal period through early childhood in relation to child EFs.

Early childhood is a period of development during which children are believed to be highly vulnerable to the negative effects of maternal depression, partly because of the substantial time young children spend with their parents and their heavy dependence on them for physical and emotional care and stimulation (Bagner et al., 2010; Choe et al., 2013, 2014). Exposure to postnatal depression during early childhood is negatively associated with children's performance on EF tasks (Baker & Kuhn, 2017; Park et al., 2018). For example, higher initial levels of mothers' postnatal depressive symptoms and smaller decreases in depression across early

childhood predict children's poorer EFs at 6 years old (Hughes et al., 2013). No study that we know of has examined heterogeneity in prenatal, postpartum, *and* postnatal symptoms in a population-based sample to clarify the roles of developmental timing and duration of exposure to maternal depression in its longitudinal relations with child EFs.

Heterogeneity in Maternal Depressive Symptoms

Longitudinal studies have frequently used group-based trajectory modeling, such as latent class growth analysis or growth mixture modeling, to estimate discrete trajectory classes of growth in maternal depressive symptoms (e.g., Campbell et al., 2007; Cents et al., 2013). This approach delineates heterogeneity in the timing, severity, and course of maternal depression, and thus variation in initial levels of symptoms and their functional forms of change across time. At least six studies in western Europe (Cents et al., 2013; Fredriksen et al., 2017; Luoma et al., 2015; Sutter-Dallay et al., 2012; van der Waerden et al., 2015a, 2015b) and six studies in North America (Campbell et al., 2007; Choe et al., 2020; Gross et al., 2009; McCall-Hosenfeld et al., 2016; Mora et al., 2009; Park et al., 2018) estimated growth in maternal depression during pregnancy, infancy, and/or childhood using group-based trajectory modeling. Overall, studies found separate trajectory classes of maternal depression illustrating prototypical patterns of continuity and change: some classes had chronically high, moderate, or low levels of symptoms that were stable across time, while others had intermittent, increasing, or decreasing trajectories of symptoms that were only elevated in the prenatal, postpartum, or postnatal period.

A few of these studies spanned the prenatal, postpartum, and postnatal periods, but many did not examine trajectory class differences in child outcomes, and only one examined EFs. A Canadian study found that children of mothers with increasing depressive symptoms from pregnancy to 3 years of age showed poorer EFs at age 6 than children of mothers with few

symptoms (Park et al., 2018). This study's small sample yielded a low trajectory class with 105 mothers, an increasing trajectory with 27 mothers, and a decreasing trajectory with only 15 mothers. Mothers at high risk for mental illness were also oversampled, which likely influenced their estimated trajectory classes and limited their generalizability. Thus, a population-level study of maternal depression covering this same developmental span is needed.

Latent class analysis (LCA) is another person-centered latent variable modeling approach to estimating patterns of change in longitudinal data and population heterogeneity on a set of behaviors (Lanza & Cooper, 2016). Repeated measures LCA (RMLCA) estimates latent classes that reflect different patterns of categorical change over three or more assessments. A key difference between RMLCA and group-based trajectory modeling is the latter estimates change as a function of time whereas RMLCA does not impose functional forms of growth (e.g., quadratic) to identify naturally occurring changes across time in each latent class (Collins & Lanza, 2010). In this study, mothers' ratings of their depressive symptoms are dichotomized by whether they exceed the depression measure's clinical cutoff, so RMLCA can estimate patterns of change in mothers' risk of probable depression from pregnancy through early childhood.

To our knowledge, only Campbell and colleagues (2009) used LCA to estimate patterns of change in maternal depression across 10 assessments from ages 1 month to 12 years in a large U.S. sample. A small class of mothers (4.7%) reported chronically high symptoms exceeding the clinical cutoff throughout the 12 years. A small class of mothers (5.1%) reported high symptoms exceeding the clinical cutoff in the postpartum period that declined at 24 months to moderate subclinical levels. A larger class of mothers (10.9%) reported moderate symptoms that dipped below the clinical cutoff at 6 and 15 months but exceeded the clinical cutoff at all other ages. Almost 80% of mothers followed one of two trajectories with low symptom levels throughout

childhood. Children of mothers with the fewest symptoms had lower levels of risky behavior and behavior problems at age 15, but they did not differ from children of mothers with early and decreasing symptoms (Campbell et al., 2009). We extend this study, which did not assess EFs or prenatal depression, by examining latent classes of change in mothers' prenatal, postpartum, and postnatal depression and their differences in child EFs.

The Current Study

This longitudinal study of ALSPAC families tests two research questions: 1) Are there separable latent classes of mothers who meet clinical cutoffs for depression during the prenatal, postpartum, or postnatal periods? and 2) Exposure to which class of maternal depression is most detrimental to children's school-age EFs? H1: We hypothesize finding five latent classes of mothers with elevated risk of: chronic depression, only prenatal depression, only postpartum depression, only postnatal depression, or no depression throughout the prenatal to postnatal periods. Studies show early and chronic exposure to maternal depression most strongly predicts child maladjustment, and prior ALSPAC studies show prenatal depression predicts child outcomes more strongly than postpartum depression. H2: We hypothesize finding a gradient of EF impairments across latent classes based on the timing and duration of depression, such that children of mothers at high risk for chronic depression will show the most impairments at 8 years old, while children of mothers at low risk for depression across time will show the fewest impairments. Between these two classes, more EF impairments are expected for children of mothers at high risk for only prenatal depression compared to children of mothers at high risk for only postpartum or postnatal depression, and more for children of mothers in the postpartum group than the postnatal group. Lastly, in exploratory tests of whether exposure to maternal depression is related to specific or global EF impairments, we examine latent class differences in

three facets of inhibitory control: selective attention and divided attention (also subcomponents of executive attention) and cognitive inhibition (Diamond, 2013).

Methods

Participants and Procedure

The sample consists of participants from ALSPAC, an ongoing longitudinal birth cohort study of more than 14,000 participants from pregnancy to adulthood conducted to understand genetic and environmental factors in a wide range of developmental and health outcomes. Pregnant women in the former county of Avon in the U.K. were recruited if they had an expected delivery date between April 1, 1991 and December 31, 1992. An initial sample of 14,541 pregnant women were recruited, resulting in 14,676 fetuses, 14,062 live births, and 13,988 children alive at one year of age. When the oldest children were approximately 7 years old, an attempt was made to add to the initial sample eligible families who initially declined to participate or did not respond to earlier recruitment calls, resulting in a total sample size of 15,454 pregnancies and 15,589 fetuses. Of this sample, 14,901 children were alive at age one (49% female, 2% non-White mothers, 79% married couples). Notably, this sample includes mothers who experienced pregnancy and birth complications, as well as mental health problems that likely impact the current study's focal variables. This rich dataset includes many waves of data collection, including questionnaires completed by children, parents, and teachers; administrative records; observational data; clinical assessments; and biological samples. The study website contains details of all available data through a fully searchable data dictionary and variable search tool: http://www.bristol.ac.uk/alspac/researchers/our-data/. For further information regarding sample enrollment, participant characteristics, and general study methodology, we refer the reader to publications from the ALSPAC team that have profiled this

cohort (Boyd et al., 2013; Fraser et al., 2013; Golding et al., 2001). Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee, but the current study was exempt from local institutional review because it was a secondary data analysis. Informed consent for the use of the data collected via questionnaires and clinics was obtained from participants following recommendations of the ALSPAC Ethics and Law Committee at the time.

The current study's LCA analyses, addressing hypothesis 1, included 13,624 mothers who reported their depressive symptoms at least once across all assessments: 4.5 and 8 months in the prenatal period, 2 and 8 months in the postpartum period, and 21 and 33 months in the postnatal period. Parents also completed demographic questionnaires throughout childhood, which yielded data on childhood covariates. When children were approximately 8 years old, a subsample of children were invited to attend clinic visits for assessments of their intellectual and executive functioning. This subsample of 6,870 children with EF data were included in analyses replicating LCA results and testing mean differences among latent classes for hypothesis 2. Only 5,095 children possessed data for all childhood covariates, so we imputed missing data in childhood covariates to include all 6,870 children with EF data in tests of hypothesis 2.

Measures

Maternal depressive symptoms. Mothers reported their symptoms on the Edinburgh Postnatal Depression Scale (EPDS), a 10-item questionnaire validated for use during and outside the postnatal period as well as with community samples (Murray & Carothers, 1990), on a scale from 0 to 3 with higher values indicating greater agreement to statements, such as "I have blamed myself unnecessarily when things went wrong" (Cox et al., 1987). Cronbach alphas for the EPDS scores ranged from .85 to .88 across assessments (Paul & Pearson, 2020). Scores over 12 are highly sensitive and specific in predicting clinical diagnoses of major depressive disorder

(Hewitt et al., 2009; Gaynes et al., 2005). Recent work shows a clinical cutoff of 13 distinguishes between pregnant or postpartum women with probable depression and those with subthreshold symptoms (Levis et al., 2020). We coded mothers who scored at or above the clinical cutoff of 13 as "1" for probable depression and those who scored below as "0" for non-depressed. At 4.5 and 8 months in pregnancy, 12.4% and 13.5% of women met risk criteria for probable depression, respectively. At 2 and 8 months postpartum, 8.7% and 7.2% of mothers met criteria for probable depression, respectively. At 21 and 33 months in the postnatal period, 7.4% and 8.7% of mothers met criteria for probable depression, respectively.

Child executive functions. Executive functions were assessed when children were 8 years old using three subtests of the Test of Everyday Attention for Children: Sky Search, Dual Attention, and Opposite Worlds (Manly et al., 1998). The Sky Search task assesses a child's ability to focus on relevant stimuli and measures selective attention. The Dual Attention task builds on Sky Search and measures the ability to divide attention, as it requires children to multi-task. The Opposite Worlds task is a Stroop-like task used to measure cognitive inhibition. Items for the tasks were aggregated into task-specific normative scores with high test-retest reliability (Sky Search r = .90, Dual Attention r = .81, Opposite Worlds r = .92; Manly et al., 2001).

Covariates. Main analyses included childhood correlates of children's performance on EF tasks at age 8. We included weekly family income in childhood (averaged across ages 33 months, 47 months, 7 years 1 month, and 8 years 1 month, alpha = .90), the highest level of education among either parent at child age 8, biological sex of the child (coded as -1 = male, 1 = female), and child verbal IQ scores assessed at age 8 with the following five subtests of the widely-used Wechsler Intelligence Scale for Children (WISC-III^{UK}): information, similarities, arithmetic, vocabulary, and comprehension (Wechsler, 1991). We used covariates assessed at the

same age as children's EFs to limit the impact of potential confounds when examining mean differences among latent classes of maternal depression.

Data Analysis Plan

We examined descriptive statistics, correlations, missing data, and attrition in SPSS 27. We tested study hypotheses using LCA in M*plus* 8.7 (Muthén & Muthén, 1998–2021). The goal of LCA is to identify latent classes that represent subgroups of individuals with the same pattern of responses on a set of categorical variables, prevalence estimates for each latent class, and each variable's amount of error in measuring latent classes (Collins & Lanza, 2010; Lanza & Cooper, 2016). We used the full LCA sample of mothers (n = 13,624) to test Hypothesis 1. We estimated two through six latent classes in a series of RMLCA to identify patterns of change in mothers meeting the clinical cutoff for probable depression from the prenatal through postnatal periods. We compared model fit indices to identify a solution with the best balance of model fit and parsimony and conceptually meaningful latent classes. Best model fit was decided by the lowest Akaike's information criterion (AIC) and Bayesian information criterion (BIC), entropy close to 1.0, class membership posterior probabilities close to 1.0, and significant Lo-Mendell-Rubin adjusted likelihood ratio test (LMR-LRT) and bootstrap likelihood ratio test (BLRT), which indicate improvements over simpler solutions with one fewer class. After finding the best solution, to test Hypothesis 2, we examined mean differences among latent classes on Sky Search, Dual Attention, and Opposite Worlds scores at age 8 in a series of models wherein covariances among the three dependent variables were held constant across latent classes. We used a manual Bolck, Croon, and Hagenaar (BCH) method to test mean differences in EFs with z-tests while accounting for uncertainty in latent class membership, without altering latent class composition (Asparouhov & Muthén, 2021), and childhood covariates to reduce bias from non-

random missing data and attrition in the 6,870 children with imputed missing data for childhood covariates. In sensitivity analyses, we examined mean differences in child EFs and childhood covariates with the full LCA sample (n = 13,624) using Wald chi-square (χ^2) tests and an automatic BCH method that accounts for class uncertainty (Asparouhov & Muthén, 2021). The automatic BCH method does not allow one to account for covariates, so these sensitivity analyses were used to replicate tests of mean differences in study variables without altering latent class composition or using imputed data. We calculated Cohen's *d* effect sizes (J. Cohen, 1988) post hoc with an online calculator (Lenhard & Lenhard, 2016) for all latent class differences.

Results

Preliminary Results

Supplemental Table 1 shows descriptive statistics and intercorrelations of study variables. Absolute skewness and kurtosis values (1.94–2.92 and 1.75–6.51, respectively) were within acceptable ranges for maternal depression, covariates, and child variables. Only distributional values for Opposite Worlds scores indicated non-normality (skewness = -3.52, kurtosis = 17.58), which supported use of maximum likelihood with robust standard errors to produce parameter estimates robust to non-normality (Kline, 2016). Little's (1988) missing completely at random (MCAR) test with expectation maximization, $\chi^2(166) = 1{,}380.33$, $p < .001$, indicated that missing data were related to measured variables warranting their inclusion as covariates in main analyses. Missing data and attrition analyses are reported in the Supplemental Appendix. We included weekly family income, parental education, child sex, and child verbal IQ as covariates in predictive models to account for their associations with nonrandom missing data and attrition.

Main Results

H1: We hypothesized finding five latent classes of change in mothers' risk of chronic

depression, only prenatal depression, only postpartum depression, only postnatal depression, or no depression throughout the prenatal to postnatal periods. As shown in Table 1, the 5-class solution yielded the best overall fit (i.e., second lowest AIC and BIC, second highest entropy), was a significant improvement over the 4-class solution, and its five classes mostly supported our first hypothesis. Figure 1 displays the 5-class solution's latent classes of mothers showing elevated risk of chronic depression (4.6% of LCA sample, .72 class membership posterior probability), prenatal-only depression (10.7%, .65 probability), postnatal-only depression (5.2%, .62 probability), and a large class of non-depressed mothers (76.8%, .92 probability). Our hypothesized "postpartum-only" class was not supported, but we found a postpartum class (2.6%, .71 probability) in which all mothers exceeded the clinical cutoff at 8 months postpartum and about 50% odds of exceeding the cutoff at 21 and 33 months. We chose a 5-class solution to test our second hypothesis, as it best fit the data when considering all indices, improved upon simpler solutions, and produced conceptually meaningful latent classes. We largely replicated these classes in the subsample with EF data (n = 6,870), but the postnatal-only class was smaller with a lower risk of depression and more mothers were assigned to the prenatal-only class.

H2: We hypothesized finding a gradient of EF impairments across latent classes based on the timing and duration of children's exposure to maternal depression. Table 2 shows means and standard errors for each EF task and latent class among the 6,870 children with EF data, with statistically significant mean differences indicated with super-scripted letters; only significant covariates are noted. While accounting for uncertainty in latent class membership and childhood covariates with imputed missing data, children of mothers with chronic depression scored lower in cognitive inhibition than children of mothers in the postpartum (z = -.77, p = .010, Cohen's d = -.24), postnatal-only (z = -.84, p = .003, Cohen's d = -.28), and non-depressed classes (z = -.53,

p = .028, Cohen's d = -.26), but they did not differ from the prenatal-only depression class.

We expected children of mothers in the prenatal-only depression class to demonstrate greater impairments in EFs than children of mothers in the postpartum, postnatal-only, and non-depressed classes. Table 2 shows that after accounting for uncertainty in latent class membership and childhood covariates, children of mothers in the prenatal-only depression class did not differ in EFs from children of mothers in any other class.

We expected children of mothers in the postpartum depression class to demonstrate greater impairments in EFs than children of mothers in the postnatal-only and non-depressed classes. Table 2 shows that when accounting for class uncertainty and childhood covariates, children of mothers in the postpartum class scored lower in divided attention than children of mothers in the non-depressed class (z = -2.09, p = .037, Cohen's d = .24).

We expected children of mothers in the postnatal-only depression class to demonstrate greater EF impairments than children of mothers in the non-depressed class. Unexpectedly, when accounting for class uncertainty and covariates, children of mothers in the postnatal-only class scored higher in cognitive inhibition than children of mothers in the non-depressed class (z = .32, p = .024, Cohen's d = -.17) and, as noted above, the chronic depression class (see Table 2).

Overall, no latent classes of maternal depression differed in children's selective attention. Children's selective attention and divided attention scores were unrelated to each other (r = .001, p = .952), but both selective (r = .20, p < .001) and divided attention (r = .17, p < .001) scores were positively correlated with cognitive inhibition scores. Children of mothers with chronic depression showed the most impairments in cognitive inhibition, consistently across all models. Unexpectedly, children of mothers with only postnatal depression performed best in cognitive inhibition. Children of mothers without depression performed better on divided attention than

children of mothers with postpartum depression. Cohen's *d* effect sizes were small to very small.

Sensitivity Analyses

Supplemental Table 2 shows results of sensitivity analyses with the full LCA sample (*n* = 13,624) testing mean differences on study variables using maximum likelihood estimation while accounting for uncertainty in latent class membership. These analyses more than doubled the analytic sample size to test hypothesis 2 and did not control for childhood covariates, so they had greater statistical power and no imputed missing data. Consistent with main results, children of mothers in the chronic class scored lower in cognitive inhibition than children of mothers in the postpartum, postnatal-only, and non-depressed classes, and children of mothers in the postpartum class scored lower in divided attention than children of mothers in the non-depressed class. Contrary to main results, children of mothers in the prenatal-only class scored lower in cognitive inhibition than children of mothers in the postpartum, postnatal-only, and non-depressed classes, children of mothers in the postnatal-only and non-depressed classes no longer scored differently in cognitive inhibition, and children of mothers in the prenatal-only class scored lower in cognitive inhibition than children of mothers in the non-depressed class. Sensitivity analyses mostly replicated findings from the main results, but they revealed more impairments in EFs for children of mothers with only prenatal depression that did not remain significant when only the 6,870 children with EF data were included in analyses that accounted for childhood covariates. The second row of Table 2 reporting main results shows percentages of each latent class found in replicated classes in sensitivity analyses with the full LCA sample.

Lastly, we examined mean differences on childhood covariates among the five latent classes in the full LCA sample (*n* = 13,624) to better understand whether differences in their family and child characteristics help explain their differences in child EFs. The bottom of

Supplemental Table 2 shows the non-depressed class had higher family incomes than the postnatal-only, postpartum, prenatal-only, and chronic classes. The postpartum class had higher family incomes than the postnatal-only, prenatal-only, and chronic classes. The non-depressed class had higher parent education than the prenatal-only and chronic classes, and the postnatal-only class had higher parent education than the chronic class. The non-depressed class had higher child verbal IQ than the postpartum and prenatal-only class, and the postnatal-only class had higher child verbal IQ than the prenatal-only class. Child sex did not differ among latent classes.

Discussion

To our knowledge this longitudinal study of the ALSPAC cohort is only the second study to use LCA to identify subgroups of mothers showing distinct patterns of naturally occurring change in maternal depression across development. It extends Campbell et al.'s (2009) study of U.S. families by identifying five latent classes of change in Avon mothers' risk of clinical depression beginning in the second trimester of pregnancy through the postpartum and postnatal periods. Latent classes differed in the timing and length of mothers' depression, as well as in children's cognitive inhibition when accounting for child and family characteristics in childhood, such as household income and parent education. In the following, we integrate our main findings with the broader literature on maternal depression including past ALSPAC studies.

Latent Classes of Maternal Depression from Pregnancy through Early Childhood

Our RMLCA findings reveal natural patterns of change in ALSPAC mothers' risk of clinical depression that largely support our first hypothesis and align with longitudinal studies from North America and Western Europe, and the only other population-based study to document discrete patterns of change in maternal depression from pregnancy through early childhood (Cents et al., 2013). The largest observed latent class included over 75% of mothers

with minuscule risk of meeting the clinical cutoff for probable depression, which aligns with past longitudinal evidence (e.g., Cents et al., 2013; Fredriksen et al., 2017; Park et al., 2018).

The next largest latent class included almost 11% of ALSPAC mothers with moderately high risk of prenatal depression that dropped to a slightly elevated risk after pregnancy, similar to other findings (Fredriksen et al., 2017; Mora et al., 2009; Park et al., 2018; van der Waerden et al., 2015a). In contrast, Sutter-Dallay and colleagues (2012) found 21% of French mothers had depressive symptoms that peaked prenatally and met clinical cutoffs from pregnancy through early childhood, but they excluded mothers with prior depressive episodes, which likely affected their trajectory class sizes, as studies generally show higher prevalence rates of prenatal than postpartum depression (Field, 2011). And indeed, our prenatal-only depression class included substantially more mothers than our postpartum class (2.6%), which showed increasing risk of depression from the second trimester until all mothers met the clinical cutoff at 8 months postpartum. Contrary to our hypothesis, the postpartum class' risk of depression remained at moderate levels after infancy. This was the smallest class, similar to the 2% of Norwegian mothers whose symptoms only exceeded the clinical cutoff at 1.5 months postpartum (Fredriksen et al., 2017). Other studies have found trajectories differing in size in which mothers' symptoms peaked after birth but were slightly elevated to moderately high in the prenatal or postnatal period (Campbell et al., 2009; McCall-Hosenfeld et al., 2016; Sutter-Dallay et al., 2012).

Almost 5% of ALSPAC mothers continually showed high risk of exceeding the clinical cutoff during pregnancy through early childhood. This chronic depression class was similar in size to the 4.7% of U.S. mothers reporting the most symptoms throughout childhood (Campbell et al., 2009), and trajectory classes spanning pregnancy through early childhood of chronically depressed mothers in the U.S. (Mora et al., 2009) and France (Sutter-Dallay et al., 2012; van der

Waerden et al., 2015a). Other studies have found smaller chronic trajectory classes among Dutch (Cents et al., 2013) and U.S. mothers (McCall-Hosenfeld et al., 2016), and studies with smaller samples have not found chronic trajectories (e.g., Choe et al., 2020; Luoma et al., 2015; Park et al., 2018).

About 5% of ALSPAC mothers were in the postnatal-only depression class, showing moderately low risk of depression in pregnancy and early infancy that increased from 8 months postpartum to similar risk levels as the chronic depression class at 33 months. Van der Waerden and associates (2015a) also found almost 5% of French mothers experienced severe depressive symptoms during the preschool years that worsened from ages 3 to 5. Mora et al. (2009) found 7% of U.S. mothers experienced worsening symptoms that peaked when children were 2 years old. These findings align with evidence that some mothers experience low to moderate levels of depressive symptoms that progressively worsen in severity across early childhood until exceeding clinical cutoffs (Campbell et al., 2009; Fredriksen et al., 2017; Park et al., 2018).

Latent Class Differences in Children's School-Age Executive Functions

Almost all differences between latent classes of maternal depression were in children's cognitive inhibition, a subcomponent of inhibitory control that involves the suppression of prepotent mental representations (Diamond, 2013). Cognitive inhibition supports working memory by helping children inhibit distracting thoughts and memories that can interfere with their learning and performance on goal-oriented tasks. The ability to inhibit attention to distractors also promotes selective and sustained attention, as well as flexibility in engaging in self-control (Diamond, 2006, 2013). Impairments in cognitive inhibition are positively associated with children's memory errors (Alexander et al., 2002) and hyperactivity (Burley et al., 2022), which may in part explain inhibitory control's positive association with academic skills (Allan et

al., 2014) and its negative association with externalizing problems (Choe et al., 2014). Notably, the Opposite Worlds task that assesses cognitive inhibition may have been more sensitive in detecting differences among latent classes than the other two tasks, as it is the most difficult.

Regardless of whether we accounted for childhood covariates, children of mothers in the chronic depression class continually showed poorer cognitive inhibition than children in the non-depressed, postpartum, and postnatal-only classes. These three classes had higher family incomes, parent education, and/or child verbal IQ than the chronic and prenatal-only classes, yet differences in cognitive inhibition remained even when accounting for covariates. Children in these three latent classes also showed better cognitive inhibition than children in the prenatal-only class, but not after accounting for covariates. These findings support the argument that chronic exposure to maternal depression beginning in utero is more detrimental to child development than intermittent or later exposure (Campbell et al., 2007; Goodman et al., 2011). They add to the growing evidence of negative associations between mothers' depression and children's EFs (Baker & Kuhn, 2017; Hughes et al., 2013; Park et al., 2018; Pearson et al., 2016) by showing chronic exposure to maternal depression from pregnancy through early childhood is associated with impairments in cognitive inhibition but not in selective attention, another subcomponent of inhibitory control that reflects inhibition at the level of attention rather than at the level of thoughts and memories (Diamond, 2013).

Executive functions emerge in rudimentary form in infancy, are first measured reliably between 8 and 12 months of age, and improve rapidly across early childhood and the early school years (Best & Miller, 2010; Diamond, 2006, 2013). Age-related improvements in EFs attributed to the continuing maturation of the prefrontal cortex promote school readiness, social and academic competencies, as well as optimal physical and mental health (Blair & Raver, 2015;

Diamond, 2013). The development of EFs and the prefrontal cortex are disrupted by early and chronic adversity (Blair, 2010), suggesting that exposure to maternal depression starting in utero is most harmful to children's EFs and their neural underpinnings when followed by exposure to maternal depression and its caregiving disruptions in infancy and early childhood (Baker & Kuhn, 2017; Gueron-Sela et al., 2018). This study's findings extend past ALSPAC evidence of greater deficits in children related to prenatal than postpartum depression by showing that the chronicity of maternal depression from pregnancy through early childhood is a key determinant of its impact on specific EFs (Barker et al., 2013; Deave et al., 2008; Pina-Camacho et al., 2014).

We found and replicated one other latent class difference in children's divided attention, a condition under which focused attention is needed for concurrent tasks, such as when multi-tasking (R. A. Cohen, 2014). Consistent with hypotheses, children of mothers in the postpartum depression class showed more divided attention impairments than children of non-depressed mothers. The postpartum depression class had lower family incomes and child verbal IQ than the non-depressed class, as well as a heightened risk of depression during the postnatal period, so children's suboptimal performance may have been related to intermittent exposure to maternal depression in both infancy and early childhood. Pearson and colleagues' (2016) prior study of ALSPAC families found greater postpartum depressive symptoms predicted 8 years old's greater impairments in cognitive inhibition and divided attention, which they referred to respectively as attentional control and attentional switching. Cognitive inhibition impairments in turn predicted lower academic achievement at 16 years old, thereby mediating effects of postpartum depression. Strong stability in depression across the perinatal period may explain our similar findings linking maternal depression with children's impairments in cognitive inhibition and divided attention.

Contrary to our hypothesis, children of mothers in the postnatal-only depression class

showed better cognitive inhibition than children of mothers in the non-depressed class. We know of no other study to document such an effect, but the late manifestation of depression for mothers in the postnatal-only class may not have affected the early development of inhibitory control and its neural underpinnings. This does not, however, explain why children exposed to maternal depression only in early childhood performed better than children never exposed to maternal depression when other studies have linked early childhood exposure to EF deficits (e.g., Baker & Kuhn, 2017; Hughes et al., 2013). We refrain from speculating as to why children exposed to only postnatal depression performed better than children of mothers at lowest risk for depression and consider this finding to be tentative pending independent replication.

Strengths, Limitations, and Future Directions

Strengths of this study include its large population-based sample, longitudinal design, multi-method data, and person-centered approach to estimating latent classes capturing heterogeneity in maternal depression across the prenatal, postpartum, and postnatal periods. The ethnically homogenous composition of our U.K. sample, however, limits to whom we can generalize our findings. Samples with greater racial-ethnic and cultural diversity are needed in future studies as there are clear racial-ethnic disparities in maternal depression that are not entirely explained by socioeconomic factors (Mukherjee et al., 2016). Mothers were retained in the sample regardless of their pregnancy or birth complications and psychiatric history, which all increase risk of depression and children's poor EFs (Field, 2011). We also dichotomized mothers' EPDS scores at the clinical cutoff rather than use their continuous scores; however, mothers' subclinical symptoms of depression also predict children's poor outcomes, so it is important for future studies to examine the severity of mothers' symptoms across a continuum (Meaney, 2018). Further, we lacked data on EFs in early childhood and adolescence as well as

mothers' depression and other mental health problems in middle childhood, so we do not know whether latent class differences in child EFs were present before or after middle childhood and whether they were a result of maternal depression or other mental illnesses at age 8. Most latent classes included about 50% of mothers from their replicated classes in sensitivity analyses with the full LCA sample, except for the prenatal-only (37.2%) and chronic (41.4%) depression classes which showed greater evidence of selective attrition than the other latent classes. Future studies that incorporate time-varying covariates will be better suited for examining the continuity of associations among our focal variables. Lastly, future studies of the prenatal and time-varying predictors of membership to latent classes of maternal depression, such as a history of anxiety or depression (Sutter-Dallay et al., 2012; van der Waerden et al., 2015b) or limited social support (Choe et al., 2020; McCall-Hosenfeld et al., 2016), can further clarify their antecedents and correlates across developmental periods, as well as inform prevention and intervention efforts.

Conclusion

Depression is the most common mental illness afflicting mothers in the U.K., with rates as high as 17.5% of mothers with young children and 22.2% with adolescents (Abel et al., 2019). This longitudinal investigation found that most mothers show the greatest risk of clinical depression during pregnancy, but impairments in EFs only consistently emerge for children exposed to maternal depression persistently throughout the perinatal period and early childhood. Perinatal depression is a major public health issue that creates large lifetime costs to families and society, for example in preterm births, productivity losses, and health services (Bauer et al., 2016). Among all live births in the U.K. in 2013, the total cost of perinatal depression and anxiety was approximately £6.6 billion ($10.3 billion USD), a majority of which was for child-related costs (Bauer et al., 2016). Exposure to maternal depression is related to children's

impaired EFs with severe repercussions for their health, well-being, and achievement (Baker & Kuhn, 2017; Hughes et al., 2013). As prenatal depression confers risk for postpartum and postnatal depression (Field, 2011; O'Hara & McCabe, 2013), intervening early to prevent pregnant women from developing prenatal depression may reduce impairments in their offspring, but prenatal depression in and of itself is not a robust predictor of children's EFs. The malleability of children's EFs through early childhood intervention (Diamond & Lee 2011) encourages its integration with treatments for maternal depression in two-generation programs.

References

Abel, K. M., Hope, H., Swift, E., Parisi, R., Ashcroft, D. M., Kosidou, K., Osam, C. S., Dalman, C., & Pierce, M. (2019). Prevalence of maternal mental illness among children and adolescents in the UK between 2005 and 2017: A national retrospective cohort analysis. *Lancet Public Health*, *4*(6), e291–e300. https://doi.org/10.1016/S2468-2667(19)30059-3

Alexander, K. W., Goodman, G. S., Schaaf, J. M., Edelstein, R. S., Quas, J. A., & Shaver, P. R. (2002). The role of attachement and cognitive inhibition in children's memory and suggestibility for a stressful event. *Journal of Experimental Child Psychology, 83*(4), 262–290. https://doi.org/10.1016/S0022-0965(02)00149-2

Allan, N. P., Hume, L. E., Allan, D.M., Farrington, A. L., & Lonigan, C. J. (2014). Relations between inhibitory control and the development of academic skills in preschool and kindergarten: A meta-analysis. *Developmental Psychology*, *50*(10),2368–2379. http://dx.doi.org/10.1037/a0037493

Asparouhov, T., & Muthén, B. (2021). Auxiliary variables in mixture modeling: Using the BCH method in Mplus to estimate a distal outcome model and an arbitrary secondary model. Mplus Web Notes: No. 21. May 14, 2014. Revised February 4, 2021.

Bagner, D. M., Pettit, J. W., Lewinsohn, P. M., & Seeley, J. R. (2010). Effect of maternal depression on child behavior: A sensitive period*? Journal of the American Academy of Child and Adolescent Psychiatry*, *49*, 699–707.

Baker, C., & Kuhn, L. (2017). Mediated pathways from maternal depression and early parenting to children’s executive function and externalizing behaviour problems. *Infant and Child Development*, *27*(1), e2052. https://doi.org/10.1002/icd.2052

Barker, E. D., Kirkham, N., Ng, J., & Jensen, S. K. G. (2013). Prenatal maternal depression

symptoms and nutrition, and child cognitive function. *The British Journal of Psychiatry*, *203*(6), 417–421. https://doi.org/10.1192/bjp.bp.113.129486

Bauer, A., Knapp, M., & Parsonage, M. (2016). Lifetime costs of perinatal anxiety and depression. *Journal of Affective Disorders*, *192*, 83–90. http://dx.doi.org/10.1016/j.jad.2015.12.005

Bell, A. F., Carter, C. S., Davis, J. M., Golding, J., Adejumo, O., Pyra, M., Connelly, J. J., & Rubin, L. H. (2016). Childbirth and symptoms of postpartum depression and anxiety: A prospective birth cohort study. *Archives of Women's Mental Health*, *19*, 219–227. https://doi.org/10.1007/s00737-015-0555-7

Best, J. R., & Miller, P. H. (2010). A developmental perspective on executive function. *Child Development*, *81*(6), 1641–1660. https://doi.org/10.1111/j.1467-8624.2010.01499.x

Blair, C. (2010). Stress and the development of self-regulation in context. *Child Development Perspectives*, *4*(3), 181–188. https://doi.org/10.1111/j.1750-8606.2010.00145.x

Blair, C., & Raver, C. C. (2015). School readiness and self-regulation: A developmental psychobiological approach. *Annual Review of Psychology*, *66*(1), 711–731. https://doi.org/10.1146/annurev-psych-010814-015221

Boyd A, Golding J, Macleod J, Lawlor DA, Fraser A, Henderson J, Molloy L, Ness A, Ring S, Davey Smith G. (2013). Cohort Profile: The 'Children of the 90s'; the index offspring of The Avon Longitudinal Study of Parents and Children (ALSPAC). *International Journal of Epidemiology*, *42*(1), 111–127. https://doi.org/10.1093/ije/dys064

Burley, D. T., Anning, K. L., & van Goozen, S. H. M. (2022). The association between hyperactive behaviour and cognitive inhibition impairments in young children. *Child Neuropsychology*, *28*(3), 302–317. https://doi.org/10.1080/09297049.2021.1976128

Camerota, M., & Willoughby, M. T. (2021). Applying interdisciplinary frameworks to study prenatal influences on child development. *Child Development Perspectives*, *15*(1), 23–30. https://doi.org/10.1111/cdep.12395

Campbell, S. B., Matestic, P., von Stauffenberg, C. V., Mohan, R., & Kirchner, T. (2007). Trajectories of maternal depressive symptoms, maternal sensitivity, and children's functioning at school entry. *Developmental Psychology*, *43*(5), 1202–1215. https://doi.org/10.1037/0012-1649.43.5.1202

Campbell, S. B., Morgan-Lopez, A. A., Cox, M. J., McLoyd, V. C., & National Institute of Child Health and Human Development Early Child Care Research Network. (2009). A latent class analysis of maternal depressive symptoms over 12 years and offspring adjustment in adolescence. *Journal of Abnormal Psychology, 118*(3), 479–493. https://doi.org/10.1037/a0015923

Casey, B. J., Tottenham, N., Liston, C., & Durston, S. (2005). Imaging the developing brain: What have we learned about cognitive development? *TRENDS in Cognitive Sciences*, *9*(3), 104–110. https://doi.org/10.1016/j.tics.2005.01.011

Cents, R. A. M., Diamantopoulou, S., Hudziak, J. J., Jaddoe, V. W. V., Hofman, A., Verhulst, F. C., Lambregtse-van den Berg, M. P., & Tiemeier, H. (2012). Trajectories of maternal depressive symptoms predict child problem behavior: The Generation R Study. *Psychological Medicine*, *43*(1), 13–25. https://doi.org/10.1017/S0033291712000657

Choe, D. E., McDonough, S., Sameroff, A. J., & Lawrence, A. C. (2020). Postnatal trajectories of maternal depressive symptoms: Postpartum antecedents and differences in toddler adjustment. *Infant Mental Health Journal*, *41*(2), 278–293. https://doi.org/10.1002/imhj.21843

Choe, D. E., Olson, S. L., & Sameroff, A. J. (2013). Effects of early maternal distress and parenting on the development of children's self-regulation and externalizing behavior. *Development and Psychopathology*, *25*(2), 437–453. https://doi.org/10.1017/S0954579412001162

Choe, D. E., Shaw, D. S., Brennan, L. M., Dishion, T. J., & Wilson, M. N. (2014). Inhibitory control as a mediator of bidirectional effects between early oppositional behavior and maternal depression. *Development and Psychopathology*, *26*(4.1), 1129–1147. https://doi.org/10.1017/S0954579414000613

Cohen, J. (1988). Statistical Power Analysis for the Behavioral Sciences (2nd ed.). Lawrence Erlbaum Associates, Inc.

Cohen, R. A. (2014). Attention. Encyclopedia of the Neurological Sciences, Volume 1 (2nd ed.). 303–313. https://doi.org/10.1016/B978-0-12-385157-4.00450-4

Collins, L. M., & Lanza, S. T. (2010). *Latent Class and Latent Transition Analysis: With Applications in the Social, Behavioral, and Health Sciences*. John Wiley and Sons Inc. https://doi.org/10.1002/9780470567333

Cox, J. L., Holden, J. M., & Sagovsky, R. (1987). Detection of postnatal depression: Development of the 10-item Edinburgh Postnatal Depression Scale. *The British Journal of Psychiatry*, *150*(6), 782–786. https://doi.org/10.1192/bjp.150.6.782

Davis, E. P., Hankin, B. L., Swales, D. A., Hoffman, M. C. (2018). An experimental test of the fetal programming hypothesis: Can we reduce child ontogenetic vulnerability to psychopathology by decreasing maternal depression. *Development and Psychopathology*, *30*(3), 787–806. https://doi.org/10.1017/S0954579418000470

Deave, T., Heron, J., Evans, J., & Emond, A. (2008). The impact of maternal depression in

pregnancy on early child development. *General Obstetrics*, *115*(8), 1043–1051. https://doi.org/10.1111/j.1471-0528.2008.01752.x

Diamond, A. (2006). The early development of executive function. In E. Bialystok & F. I. M. Craik (Eds.). In *Lifespan cognition: Mechanisms of change* (pp. 70–95). Oxford, UK: Oxford University Press. doi:10.1093/acprof:oso/9780195169539.003.0006

Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, *64*(1), 135–168. https://doi.org/10.1146/annurev-psych-113011-143750

Diamond, A., & Lee, K. (2011). Interventions shown to aid executive function development in children 4 to 12 years old. *Science*, *333*(6045), 959–964. http://dx.doi.org/10.1126/science.1204529

Durston, S., & Casey, B. J. (2006). What have we learned about cognitive development from neuroimaging? *Neuropsychologia*, *44*(11), 2149–2157. https://doi.org/10.1016/j.neuropsychologia.2005.10.010

Espy, K. A., Stalets, M. M., McDiarmid, M. M., Senn, T. E., Cwik, M. F., & Hamby, A. (2002). Executive functions in preschool children born preterm: Application of cognitive neuroscience paradigms. *Child Neuropsychology*, *8*(2), 83–92. https://doi.org/10.1076/chin.8.2.83.8723

Field, T. (2011). Prenatal depression effects on early development: A review. *Infant Behavior and Development*, *34*(1), 1–14. https://doi.org/10.1016/j.infbeh.2010.09.008

Fraser, A., Macdonald-Wallis, C., Tilling, K., Boyd, A., Golding, J., Smith, G. D., Henderson, J., Macleod, J., Molloy, L., Ness, A., Ring, S., Nelson, S. M., & Lawlor, D. A. (2013). Cohort profile: The Avon Longitudinal Study of Parents and Children: ALSPAC mothers cohort. *International Journal of Epidemiology*, *42*(1), 97–110.

https://doi.org/10.1093/ije/dys066

Fredriksen, E., von Soest, T., Smith, L., & Moe, V. (2017). Patterns of pregnancy and postpartum depressive symptoms: Latent class trajectories and predictors. *Journal of Abnormal Psychology*, *126*(2), 173–183. https://doi.org/10.1037/abn0000246

Gaynes, B. N., Gavin, N., Meltzer-Brody, S., Lohr, K. N., Swinson, T., Gartlehner, G., Brody, S., & Miller, W. C. (2005). Perinatal depression: Prevalence, screening accuracy, and screening outcomes. *Evidence Report Technology Assessment (Summary)*, *119*, 1-8. http://dx.doi.org/10.1037/e439372005-001

Gelaye, B., Rondon, M. B., Araya, R., & Williams, M. A. (2016). Epidemiology of maternal depression, risk factors, and child outcomes in low-income and middle-income countries. *Lancet Psychiatry*, *3*(10), 973–982. http://dx.doi.org/10.1016/S2215-0366(16)30284-X

Golding, J., Pembrey, M., Jones, R., & the ALSPAC Study Team (2001). ALSPAC–The Avon Longitudinal Study of Parents and Children I. Study methodology. *Paediatric and Perinatal Epidemoiology*, *15*(1), 74–87. https://doi.org/10.1046/j.1365-3016.2001.00325.x

Gooch, D., Thompson, P., Nash, H. M., Snowling, M. J., & Hulme, C. (2016). The development of executive function and language skills in the early school years. *Journal of Child Psychology and Psychiatry, 57*(2), 180–187. https://doi.org/10.1111/jcpp.12458

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: A meta-analytic review. *Clinical Child Family Psychology Review*, *14*, 1–27. https://doi.org/10.1007/s10567-010-0080-1

Gueron-Sela, N., Camerota, M., Willoughby, M. T., Vernon-Feagans, L., Cox, M. J., & The Family Life Project Key Investigators. (2018). Maternal depressive symptoms, mother-

child interactions, and children's executive function. *Developmental Psychology, 54*(1), 71–82. https://doi.org/10.1037/dev0000389

Gump, B. B., Reihman, J., Stewart, P., Lonky, E., Darvill, T., Granger, D. A., & Matthews, K. A. (2009). Trajectories of maternal depressive symptoms over her child's life span: Relation to adrenocortical, cardiovascular, and emotional functioning in children. *Development and Psychopathology*, *21*(1), 207–225. https://doi.org/10.1017/S0954579409000133

Gross, H., Shaw, D., Burwell, R., & Nagin, D. (2009). Transactional processes in child disruptive behavior and maternal depression: A longitudinal study from early childhood to adolescence. *Development and Psychopathology*, *21*(1), 139–156. https://doi.org/10.1017/S0954579409000091

Hewitt, C. E.., Gilbody, S. M., Brealey, S., Paulden, M., Palmer, S., Mann, R., Green, J., Morrell, J., Barkham, M., Light, K., & Richards, D. (2009). Methods to identify postnatal depression in primary care: An integrated evidence synthesis and value of information analysis. *Health Technology Assessment*, *13*(36), 1-145, 147-230. https://doi.org/10.3310/hta13360

Hughes, C., & Devine, R. T. (2019). For better or for worse? Positive and negative parental influences on young children's executive function. *Child Development, 90*(2), 593–609. https://doi.org/10.1111/cdev.12915

Hughes, C., Roman, G., Hart, M. J., & Ensor, R. (2013). Does maternal depression predict young children's executive function? A 4-year longitudinal study. *Journal of Child Psychology and Psychiatry*, *54*(2), 169–177. https://doi.org/10.1111/jcpp.12014

Kline, R. B. (2016). Principles and Practice of Structural Equation Modeling-Fourth Edition. Guilford Press.

Lanza, S. T., & Cooper, B. R. (2016). Latent class analysis for developmental research. *Child Development Perspectives*, *10*(1), 59–64. https://doi.org/10.1111/cdep.12163

Lenhard, W., & Lenhard, A. (2016). Computation of effect sizes. Retrieved from: https://www.psychometrica.de/effect_size.html. *Psychometrica*. http://dx.doi.org/10.13140/RG.2.2.17823.92329

Levis, B., Negeri, Z., Sun, Y., Benedetti, A., Thombs, B. D., & DEPRESsion Screening Data (DEPRESSD) EPDS Group (2020). Accuracy of the Edinburgh Postnatal Depression Scale (EPDS) for screening to detect major depression among pregnant and postpartum women: Systematic review and meta-analysis of individual participant data. *BMJ*, 371:m4022. https://doi.org/10.1136/bmj.m4022

Little, R. J. A. (1988). A test of missing completely at random for multivariate data with missing values. *Journal of the American Statistical Association*, *83*(404), 1198–1202. https://doi.org/10.1080/01621459.1988.10478722

Luoma, I., Korhonen, M., Salmelin, R. K., Helminen, K., & Tamminen, T. (2015). Long-term trajectories of maternal depressive symptoms and their antenatal predictors. *Journal of Affective Disorders*, *170*, 30–38. https://doi.org/10.1016/j.jad.2014.08.017

Madigan, S., Oatley, H., Racine, N., Fearon, R. M. P., Schumacher, L., Akbari, E., Cooke, J. E., & Tarabulsy, G. M. (2018). A meta-analysis of maternal prenatal depression and anxiety on child socioemotional development. *Journal of the American Academy of Child & Adolescent Psychiatry*, *57*(9), 645–657. https://doi.org/10.1016/j.jaac.2018.06.012

Manly, T., Anderson, V., Nimmo-Smith, I., Turner, A., Watson, P., & Robertson, I. H. (2001). The differential assessment of children's attention: The Test of Everyday Attention for Children (TEA-Ch), normative sample and ADHD performance. *Journal of Child*

Psychology and Psychiatry, 42(8), 1065–1081. https://doi.org/10.1111/1469-7610.00806

Manly, T., Robertson, I. H., Anderson, V., & Nimmo-Smith, I. (1998). Test of Everyday Attention for Children. Pearson.

McCall-Hosenfeld, J. S., Phiri, K., Schaefer, E., Zhu, J., & Kjerulff, K. (2016). Trajectories of depressive symptoms throughout the peri- and postpartum period: Results from the first baby study. *Journal of Women's Health*, *25*(11), 1112–1121. https://doi.org/10.1089/jwh.2015.5310

McManus, B. M., & Poehlmann, J. (2012). Parent–child interaction, maternal depressive symptoms and preterm infant cognitive function. *Infant Behavior and Development*, *35*(3), 489–498. https://doi.org/10.1016/j.infbeh.2012.04.005

Meaney, M. J. (2018). Perinatal maternal depressive symptoms as an issue for population health. *American Journal of Psychiatry*, *175*(11), 1084–1093. https://doi.org/10.1176/appi.ajp.2018.17091031

Mora, P. A., Bennett, I. M., Elo, I. T., Mathew, L., Coyne, J. C., & Culhane, J. F. (2009). Distinct trajectories of perinatal depressive symptomatology: Evidence from growth mixture modeling. *American Journal of Epidemiology*, *169*(1), 24–32. https://doi.org/10.1093/aje/kwn283

Mukherjee, S., Trepka, M. J., Pierre-Victor, D., Bahelah, R., & Avent, T. (2016). Racial/ethnic disparities in antenatal depression in the United States: A systematic review. *Maternal Child Health Journal*, *20*, 1780–1797. https://doi.org/10.1007/s10995-016-1989-x

Murray, L., & Carothers, A. D. (1990). The validation of the Edinburgh Post-natal Depression Scale on a community sample. *The British Journal of Psychiatry*, *157*(2), 288–290. https://doi.org/10.1192/bjp.157.2.288

Muthén, L. K., & Muthén, B. O. (1998–2021). Mplus User's Guide. Eighth Edition. Los Angeles, CA: Muthén & Muthén

O'Hara, M. W., & McCabe, J. E. (2013). Postpartum depression: Current status and future directions. *Annual Review of Clinical Psychology*, *9*, 379–407. https://doi.org/10.1146/annurev-clinpsy-050212-185612

Park, M., Brain, U., Grunau, R. E., Diamond, A., & Oberlander, T. F. (2018). Maternal depression trajectories from pregnancy to 3 years postpartum are associated with children's behavior and executive functions at 3 and 6 years. *Archives of Women's Mental Health*, *21*, 353–363. https://doi.org/10.1007/s00737-017-0803-0

Paul, E., & Pearson, R. M. (2020). Depressive symptoms measured using the Edinburgh Postnatal Depression Scale in mothers and partners in the ALSPAC Study: A data note. *Wellcome Open Research*, *5*(108). https://doi.org/10.12688/wellcomeopenres.15925.2

Pearson, R. M., Bornstein, M. H., Cordero, M., Scerif, G., Mahedy, L., Evans, J., Abioye, A., & Stein, A. (2016). Maternal perinatal mental health and offspring academic achievement at age 16: The mediating role of childhood executive function. *Journal of Child Psychology and Psychiatry*, *57*(4), 491–501. https://doi.org/10.1111/jcpp.12483

Pearson, R. M., Evans, J., Kounali, D., Lewis, G., Heron, J., Ramchandani, P. G., O'Connor, T. G., & Stein, A. (2013). Maternal depression during pregnancy and the postnatal period: Risks and possible mechanisms for offspring depression at age 18 years. *JAMA Psychiatry*, *70*(12), 1312–1319. doi:10.1001/jamapsychiatry.2013.2163

Pina-Camacho, L., Jensen, S. K., & Gaysina, D. (2014). Maternal depression symptoms, unhealthy diet and child emotional–behavioural dysregulation. *Psychological Medicine*, *45*(9), 1851–1860. https://doi.org/10.1017/S0033291714002955

Power, J., van IJzendoorn, M., Lewis, A. J., Chen, W., & Galbally, M. (2021). Maternal perinatal depression and child executive function: A systematic review and meta-analysis. *Journal of Affective Disorders*, *291*, 218–234. https://doi.org/10.1016/j.jad.2021.05.003

Robinson, R., Lahti-Pulkkinen, M., Heinonen, K., Reynolds, R. M., & Räikkönen, K. (2019). Fetal programming of neuropsychiatric disorders by maternal pregnancy depression: A systematic mini review. *Pediatric Research*, *85***,** 134–145. https://doi.org/10.1038/s41390-018-0173-y

Shaw, D. S., Gilliom, M., Ingoldsby, E. M., & Nagin, D. S. (2003). Trajectories leading to school-age conduct problems. *Developmental Psychology*, *39*(2), 189–200. https://doi.org/10.1037/0012-1649.39.2.189

Shorey, S., Chee, C. Y. I., Ng, E. D., Chan, Y. H., Tam, W. W. S., & Chong, Y. S. (2018). Prevalence and incidence of postpartum depression among healthy mothers: A systematic review and meta-analysis. *Journal of Psychiatric Research*, *104*, 235–248. https://doi.org/10.1016/j.jpsychires.2018.08.001

Sohr-Preston, S. L., & Scaramella, L. V. (2006). Implications of timing of maternal depressive symptoms for early cognitive and language development. *Clinical Child and Family Psychology Review*, *9*, 65–83. https://doi.org/10.1007/s10567-006-0004-2

Sutter-Dallay, A.-L., Cosnefroy, O., Glatigny-Dallay, E., Verdoux, H., & Rascle, N. (2012). Evolution of perinatal depressive symptoms from pregnancy to two years postpartum in a low-risk sample: The MATQUID cohort. *Journal of Affective Disorder*, *139*(1), 23–29. https://doi.org/10.1016/j.jad.2011.08.018

van der Waerden, J., Galéra, C., Larroque, B., Saurel-Cubizolles, M.-J., Sutter-Dallay, A.-L., & Melchior, M. (2015a). Maternal depression trajectories and children's behavior at age

five years. *Journal of Pediatrics*, *166*(6), 1440–1448. https://doi.org/10.1016/j.jpeds.2015.03.002

van der Waerden, J., Galéra, C., Saurel-Cubizolles, M.-J., 2015, Sutter-Dallay, A.-L., Melchior, M., & the EDEN Mother–Child Cohort Study Group (2015b). Predictors of persistent maternal depression trajectories in early childhood: Results from the EDEN mother–child cohort study in France. *Psychological Medicine*, *45*(9), 1999–2012. https://doi.org/10.1017/S003329171500015X

Van den Bergh, B. R. H., van den Heuvel, M. I., Lahti, M., Braeken, M., de Rooij, S. R., Entringer, S., Hoyer, D., Roseboom, T., Räikkönen, K., King, S., & Schwab, M. (2020). Prenatal developmental origins of behavior and mental health: The influence of maternal stress in pregnancy. *Neuroscience & Biobehavioral Reviews*, *117*, 26–64. https://doi.org/10.1016/j.neubiorev.2017.07.003

Wechsler, D. (1991). Wechsler Intelligence Scale for Children-Third Edition (WISC-III): Manual. Psychological Corporation.

Yang, Y., Shields, G. S., Zhang, Y., Wu, H., Chen, H., & Romer, A. L. (2022). Child executive function and future externalizing and internalizing problems: A meta-analysis of prospective longitudinal studies. *Clinical Psychology Review*, *97*, 102194. https://doi.org/10.1016/j.cpr.2022.102194

Table 1.

Comparison of Fit Among Latent Class Analysis Solutions for Maternal Depressive Symptoms (N = 13,624)

Fit Statistic	2-Class	3-Class	4-Class	5-Class	6-Class
1. AIC	41,397.57	41,117.88	40,990.77	40,979.56	**40,975.56**
2. BIC	41,495.33	41,268.27	**41,193.80**	41,235.23	41,283.86
3. Entropy	**.79**	.70	.73	.78	.71
4. Percentage of Sample in Each Class Based on Posterior Probabilities	18.1%, 81.9%	5.7%, 20.9%, 73.4%	4.9%, 7.9%, 11.1%, 76.1%	2.6%, 4.6%, 5.2%, 10.7%, 76.8%	2.2%, 2.4% 4.3%, 7.0%, 16.1%, 68.1%
5. Probability of Class Membership	.87 – .96	.72 – .91	.62 – .91	.62 – .92	.61 – .88
6. LMR-LRT	6,694.66, $p < .001$	289.35, $p < .001$	139.02, $p < .001$	**24.83, $p = .004$**	17.74, $p = .175$
7. BLRT	-24,083.35 (7), $p < .001$	-20,685.79 (7), $p < .001$	-20,538.94 (7), $p < .001$	**-20,468.39 (7), $p < .001$**	-20,455.78 (7), $p = .061$

Note. Bold text indicates best fit indices among all possible solutions. AIC = Akaike's Information Criterion. BIC = Bayesian Information Criterion. LMR-LRT = Lo-Mendell-Rubin Likelihood Ratio Test. BLRT = Bootstrap Likelihood Ratio Test.

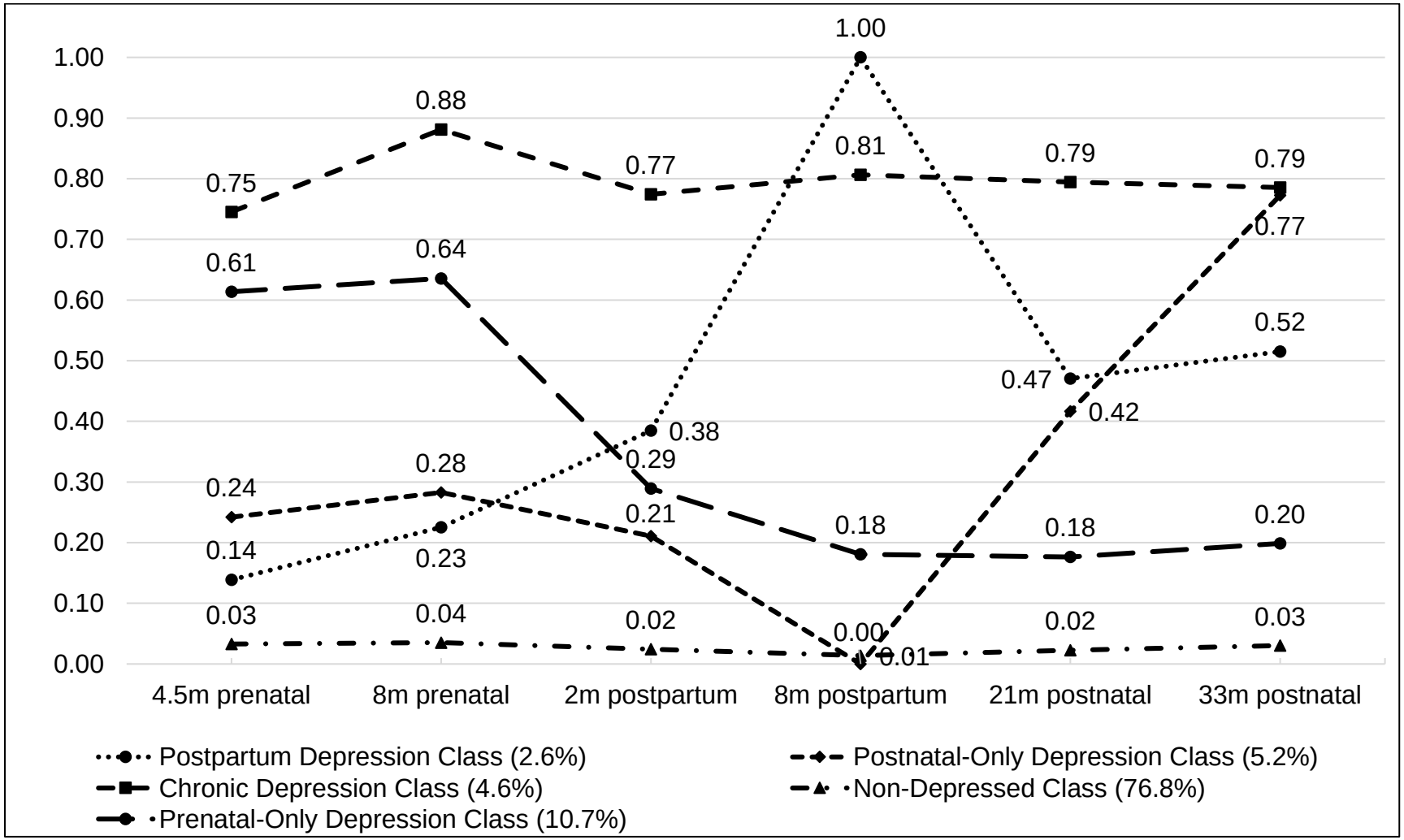
Table 2.

Executive Function Differences Between Latent Classes of Maternal Depression: Manual BCH with Imputed Covariates (N = 6,870)

	Latent Classes of Maternal Depressive Symptoms				
Dependent Variables	*Non-Depressed* n = 5,867 (52.2%)	*Postnatal-Only* n = 199 (50.8%)	*Postpartum* n = 128 (52.5%)	*Prenatal-Only* n = 438 (37.2%)	*Chronic Depression* n = 238 (41.4%)
"Selective Attention" Sky Search	M = 8.78 [a] SE = 0.04 R^2 = .01 VIQ (β = .11, p < .001)	M = 8.87 [a] SE = 0.24 R^2 = .14	M = 8.83 [a] SE = 0.27 R^2 = .10 PE (β = -.26, p = .047)	M = 8.44 [a] SE = 0.37 R^2 = .03	M = 8.36 [a] SE = 0.26 R^2 = .20 VIQ (β = .48, p < .001)
"Divided Attention" Dual Attention	M = 7.59 [a] SE = 0.06 R^2 = .07 Sex (β = -.13, p < .001); VIQ (β = .21, p < .001)	M = 7.49 [a,b] SE = 0.41 R^2 = .11 VIQ (β = .33, p = .009)	M = 6.57 [b] SE = 0.48 R^2 = .09	M = 7.53 [a,b] SE = 0.59 R^2 = .06	M = 7.51 [a,b] SE = 0.45 R^2 = .11 VIQ (β = .35, p = .005)
"Cognitive Inhibition" Opposite Worlds	M = 18.27 [a] SE = 0.03 R^2 = .06 VIQ (β = .25, p < .001)	M = 18.58 [b] SE = 0.13 R^2 = .06	M = 18.50 [a,b] SE = 0.14 R^2 = .11 PE (β = -.34, p = .031); Income (β = .36, p = .020)	M = 18.12 [a,b,c] SE = 0.20 R^2 = .14	M = 17.74 [c] SE = 0.24 R^2 = .33 VIQ (β = .61, p = .001)

Note. Means in a row not sharing a common super-scripted letter differ at p < .05. Only significant covariates are shown. Latent class sizes based on most likely membership. Percentages next to latent class sizes reflect percentages of replicated classes in sensitivity analyses with the full LCA sample. VIQ = Verbal IQ. PE = Parental Education. Sex coded -1 = males, 1 = females.

Figure 1. Final Latent Class Analysis Solution for Maternal Depressive Symptoms (N = 13,624)



Note. The y-axis represents the estimated likelihood that members within a latent class scored above the clinical cutoff of the Edinburgh Postnatal Depression Scale. m = months.

Supplemental Table 1.

Descriptive Statistics and Intercorrelations of Study Variables

Variable	1	2	3	4	5	6
1. EPDS 4.5m g	—					
2. EPDS 8m g	.64	—				
3. EPDS 2m	.53	.58	—			
4. EPDS 8m	.50	.55	.61	—		
5. EPDS 21m	.49	.52	.56	.61	—	
6. EPDS 33m	.47	.50	.52	.58	.62	—
M	6.98	7.07	6.06	5.41	5.72	6.26
SD	4.87	5.08	4.79	4.68	4.79	5.04
N	11,995	11,949	11,570	11,179	10,277	9,476

Variable	7.	8.	9.	10.	11.	12.
7. Family Income	—					
8. Parent Education	.38	—				
9. Verbal IQ	.28	.27	—			
10. Selective Attention	.04	.04	.14	—		
11. Divided Attention	.10	.08	.24	.05	—	
12. Cognitive Inhibition	.11	.05	.26	.23	.22	—
M	3.58	8.12	107.20	8.75	7.58	18.26
SD	1.16	4.08	16.75	2.38	3.79	1.68
N	10,449	6,881	6,947	6,865	6,634	6,772

Note. All correlation coefficients are statistically significant ($p < .01$). EPDS = Edinburgh Postnatal Depression Scores. m = month. g = gestation (prenatal).

Supplemental Table 2.

Executive Function and Childhood Covariate Differences Between Latent Classes: Automatic BCH Method (N = 13,624)

Dependent Variables	Latent Classes of Maternal Depressive Symptoms				
	Non-Depressed n = 11,234	*Postnatal-Only* n = 392	*Postpartum* n = 244	*Prenatal-Only* n = 1,179	*Chronic Depression* n = 575
"Selective Attention" Sky Search Overall test: $\chi^2(4) = 8.20$, $p = .084$	$M = 8.79$ $SE = 0.04$	$M = 8.69$ $SE = 0.24$	$M = 8.80$ $SE = 0.27$	$M = 8.49$ $SE = 0.26$	$M = 8.33$ $SE = 0.24$
"Divided Attention" Dual Attention Overall test: $\chi^2(4) = 11.52$, $p = .021$	$M = 7.67$ $SE = 0.06$ **Non-Depressed > Postpartum, $p = .014$.**	$M = 7.46$ $SE = 0.40$	$M = 6.49$ $SE = 0.47$	$M = 6.97$ $SE = 0.43$	$M = 7.70$ $SE = 0.41$
"Cognitive Inhibition" Opposite Worlds Overall test: $\chi^2(4) = 16.64$, $p = .002$	$M = 18.29$ $SE = 0.02$ **Non-Depressed > Chronic Depression, $p = .031$, Prenatal-Only, $p = .012$.**	$M = 18.50$ $SE = 0.15$ **Postnatal-Only > Prenatal-Only, $p = .012$.**	$M = 18.49$ $SE = 0.16$ **Postpartum > Chronic Depression, $p = .023$.**	$M = 17.79$ $SE = 0.19$ **Prenatal-Only < Postpartum, $p = .008$.**	$M = 17.82$ $SE = 0.22$ **Chronic Depression < Postnatal-Only, $p = .011$.**
Family Income (weekly average) Overall test: $\chi^2(4) = 325.08$, $p < .001$.	$M = 3.73$ $SE = 0.01$ **Non-Depressed > Postnatal-Only, $p < .001$, Postpartum, $p = .005$, Prenatal-Only, $p < .001$, Chronic, $p < .001$.**	$M = 3.06$ $SE = 0.09$	$M = 3.41$ $SE = 0.11$ **Postpartum > Postnatal-Only, $p = .015$, Prenatal-Only, $p = .006$, Chronic, $p = .003$.**	$M = 2.97$ $SE = 0.10$	$M = 2.92$ $SE = 0.09$
Parent Education (highest attainment level of either parent) Overall test: $\chi^2(4) = 27.74$, $p < .001$.	$M = 8.24$ $SE = 0.06$ **Non-Depressed > Prenatal-Only, $p = .029$, Chronic, p**	$M = 8.23$ $SE = 0.39$ **Postnatal-Only > Chronic, $p = .015$.**	$M = 7.94$ $SE = 0.45$	$M = 7.21$ $SE = 0.46$	$M = 6.78$ $SE = 0.44$

	= .001.				
Child Biological Sex (1 = male, 2 = female) Overall test: $\chi^2(4) = 2.83$, $p = .586$.	*M* = -0.04 *SE* = 0.01	*M* = -0.08 *SE* = 0.09	*M* = 0.09 *SE* = 0.09	*M* = 0.00 *SE* = 0.05	*M* = -0.10 *SE* = 0.06
Child Verbal IQ Overall test: $\chi^2(4) = 42.48$, $p < .001$.	*M* = 108.01 *SE* = 0.24 **Non-Depressed > Postpartum, *p* = .034, Prenatal-Only, *p* < .001.**	*M* = 106.76 *SE* = 1.77 **Postnatal-Only > Prenatal-Only, *p* = .023.**	*M* = 103.76 *SE* = 1.98	*M* = 100.19 *SE* = 1.73	*M* = 105.59 *SE* = 1.83

Note. Bold text indicates significant mean differences between latent classes. Latent class sizes based on most likely membership.

Loglikelihood = -20,455.78. Bayesian information criterion = 41,235.23. Entropy = .78.

Supplemental Appendix

Among the LCA sample, 12,174 mothers (89.4%) reported their depressive symptoms at 4.5 months gestation, 12,044 mothers (88.4%) reported symptoms at 8 months gestation, 11,690 mothers (85.8%) reported symptoms when children were 2 months old, 11,179 mothers (82.1%) reported symptoms at 8 months old, 10,277 mothers (75.4%) reported symptoms at 21 months old, and 9,563 mothers (70.2%) reported symptoms at 33 months old. Mothers missing depression data at 4.5 months gestation reported lower weekly family income (M = 3.26, SD = 1.31) than mothers with these data (M = 3.61, SD = 1.14), $t(1059.79) = 7.85$, $p < .001$. Mothers missing depression data at 8 months gestation reported lower weekly family income (missing M = 3.09, SD = 1.32; not missing M = 3.61, SD = 1.14), $t(717.36) = 9.78$, $p < .001$, parent education (missing M = 7.41, SD = 4.15; not missing M = 8.15, SD = 4.07), $t(6879) = 3.16$, $p = .002$, and their children had lower verbal IQ scores (missing M = 103.18, SD = 16.86; not missing M = 107.42, SD = 16.72), $t(6945) = 4.67$, $p < .001$.

Mothers missing depression data at 2 months old reported lower weekly family income (missing M = 2.95, SD = 1.31; not missing M = 3.62, SD = 1.14), $t(638.86) = 12.02$, $p < .001$, parent education (missing M = 7.52, SD = 4.00; not missing M = 8.14, SD = 4.08), $t(6879) = 2.31$, $p = .021$, and their children had lower verbal IQ (missing M = 102.59, SD = 16.37; not missing M = 107.43, SD = 16.74), $t(6945) = 5.09$, $p < .001$, Dual Attention (missing M = 6.79, SD = 4.02; not missing M = 7.61, SD = 3.77), $t(331.52) = 3.50$, $p < .001$, and Opposite World scores (missing M = 17.83, SD = 2.15; not missing M = 18.28, SD = 1.65), $t(335.67) = 3.67$, $p < .001$.

Mothers missing depression data at 8 months old reported lower weekly family income (missing M = 3.05, SD = 1.31; not missing M = 3.62, SD = 1.14), $t(774.43) = 11.08$, $p < .001$,

parent education (missing $M = 7.33$, $SD = 4.01$; not missing $M = 8.15$, $SD = 4.08$), $t(6879) = 3.37$, $p < .001$, and their children had lower verbal IQ (missing $M = 102.74$, $SD = 16.32$; not missing $M = 107.49$, $SD = 16.74$), $t(6945) = 5.63$, $p < .001$, Dual Attention (missing $M = 6.84$, $SD = 3.94$; not missing $M = 7.62$, $SD = 3.78$), $t(447.31) = 3.86$, $p < .001$, and Opposite World scores (missing $M = 17.95$, $SD = 2.09$; not missing $M = 18.28$, $SD = 1.64$), $t(448.04) = 3.19$, $p = .002$.

Mothers missing depression data at 21 months old reported lower weekly family income (missing $M = 3.19$, $SD = 1.29$; not missing $M = 3.62$, $SD = 1.14$), $t(1208.73) = 10.40$, $p < .001$, parent education (missing $M = 7.33$, $SD = 4.09$; not missing $M = 8.17$, $SD = 4.07$), $t(6879) = 4.20$, $p < .001$, and their children had lower verbal IQ (missing $M = 102.42$, $SD = 16.38$; not missing $M = 107.69$, $SD = 16.71$), $t(6945) = 7.62$, $p < .001$, Dual Attention (missing $M = 7.04$, $SD = 3.94$; not missing $M = 7.63$, $SD = 3.77$), $t(726.44) = 3.56$, $p < .001$, and Opposite World scores (missing $M = 17.94$, $SD = 2.02$; not missing $M = 18.29$, $SD = 1.64$), $t(717.61) = 4.26$, $p < .001$.

Mothers missing depression data at 33 months old reported lower weekly family income (missing $M = 3.36$, $SD = 1.31$; not missing $M = 3.61$, $SD = 1.14$), $t(1370.23) = 6.21$, $p < .001$, parent education (missing $M = 7.48$, $SD = 4.10$; not missing $M = 8.17$, $SD = 4.07$), $t(6879) = 3.90$, $p < .001$, and their children had lower verbal IQ (missing $M = 103.72$, $SD = 16.34$; not missing $M = 107.68$, $SD = 16.75$), $t(6945) = 6.43$, $p < .001$, Dual Attention (missing $M = 7.21$, $SD = 3.88$; not missing $M = 7.63$, $SD = 3.77$), $t(1014.27) = 2.87$, $p = .004$, and Opposite World scores (missing $M = 18.09$, $SD = 1.80$; not missing $M = 18.28$, $SD = 1.66$), $t(1017.85) = 2.99$, $p = .003$.

Age 8 clinic data were available for only a subset of children. The most data were

available for verbal IQ scores (n = 6,947 children, 51.0% of LCA sample, M = 107.20, SD = 16.75), Sky Search (n = 6,865, 50.4% of LCA sample, M = 8.75, SD = 2.38), Opposite Worlds (n = 6,772, 49.7% of LCA sample, M = 18.26, SD = 1.68), and then Dual Attention scores (n = 6,634, 48.7% of LCA sample, M = 7.58, SD = 3.79). Children missing verbal IQ data had lower weekly family income (missing M = 3.23, SD = 1.25; not missing M = 3.79, SD = 1.05), $t(7126.76) = 23.41$, $p < .001$, parent education (missing M = 7.50, SD = 4.12; not missing M = 8.30, SD = 4.05), $t(6879) = 6.82$, $p < .001$, and Sky Search scores (missing M = 7.95, SD = 2.96; not missing M = 8.76, SD = 2.37), $t(6863) = 3.11$, $p = .002$, than children with verbal IQ data.

Compared to children with Sky Search data, children missing Sky Search data had higher verbal IQ scores (missing M = 109.75, SD = 15.99; not missing M = 107.14, SD = 16.76), $t(6945) = -1.99$, $p = .046$, but lower levels of parental education (missing M = 7.54, SD = 4.13; not missing M = 8.30, SD = 4.05), $t(6879) = 6.56$, $p < .001$, and weekly family income (missing M = 3.24, SD = 1.25; not missing M = 3.79, SD = 1.05), $t(7377.64) = 22.78$, $p < .001$.

Compared to children with Dual Attention data, children missing Dual Attention data had higher Sky Search scores (missing M = 9.41, SD = 4.31; not missing M = 8.73, SD = 2.28), $t(234.51) = -2.39$, $p = .018$, but lower parental education (missing M = 7.63, SD = 4.12; not missing M = 8.29, SD = 4.05), $t(6879) = 5.85$, $p < .001$, weekly family income (missing M = 3.26, SD = 1.25; not missing M = 3.79, SD = 1.05), $t(7925.77) = 22.71$, $p < .001$, verbal IQ (missing M = 102.84, SD = 19.54; not missing M = 107.45, SD = 16.54), $t(411.04) = 4.51$, $p < .001$, and Opposite Worlds scores (missing M = 17.33, SD = 2.94; not missing M = 18.29, SD = 1.61), $t(209.91) = 4.65$, $p < .001$.

Compared to children with Opposite Worlds data, children missing Opposite Worlds data had lower parental education (missing M = 7.58, SD = 4.13; not missing M = 8.29, SD = 4.05),

$t(6879) = 6.26$, $p < .001$, and weekly family income (missing $M = 3.25$, $SD = 1.25$; not missing $M = 3.79$, $SD = 1.05$), $t(7606.65) = 22.84$, $p < .001$.

A subset of participants had childhood covariate data for parental education ($n = 6,881$, 50.5% of LCA sample, $M = 8.12$, $SD = 4.08$), weekly family income ($n = 10,449$, 76.7% of LCA sample, $M = 3.58$, $SD = 1.16$), and child sex ($n = 13,552$, 99.5% of LCA sample, 51.7% male). Compared to children with parental education data, children missing education data had lower scores for verbal IQ (missing $M = 104.79$, $SD = 17.28$; not missing $M = 107.94$, $SD = 16.52$), $t(2588.07) = 6.48$, $p < .001$, Sky Search (missing $M = 8.50$, $SD = 2.55$; not missing $M = 8.83$, $SD = 2.32$), $t(2482.18) = 4.56$, $p < .001$, Dual Attention (missing $M = 7.30$, $SD = 3.85$; not missing $M = 7.66$, $SD = 3.77$), $t(2500.50) = 3.18$, $p < .001$, Opposite Worlds (missing $M = 18.13$, $SD = 1.75$; not missing $M = 18.30$, $SD = 1.65$), $t(2492.05) = 3.44$, $p < .001$, and weekly family income (missing $M = 3.22$, $SD = 1.28$; not missing $M = 3.78$, $SD = 1.04$), $t(6445.03) = 22.74$, $p < .001$.

Compared to children with weekly family income data, children missing income data had lower scores for verbal IQ (missing $M = 103.02$, $SD = 16.33$; not missing $M = 107.46$, $SD = 16.74$), $t(6945) = 5.14$, $p < .001$, Sky Search (missing $M = 8.48$, $SD = 2.77$; not missing $M = 8.77$, $SD = 2.36$), $t(444.94) = 2.02$, $p = .044$, Dual Attention (missing $M = 6.97$, $SD = 3.86$; not missing $M = 7.61$, $SD = 3.78$), $t(6632) = 3.25$, $p = .001$, and Opposite Worlds (missing $M = 17.93$, $SD = 2.06$; not missing $M = 18.28$, $SD = 1.65$), $t(431.83) = 3.32$, $p < .001$.

We found sex differences for age 8 assessment scores of verbal IQ, $t(6890.43) = 2.66$, $p = .008$, Sky Search, $t(6863) = 2.28$, $p = .023$, Dual Attention, $t(6394.05) = 10.12$, $p < .001$, and Opposite Worlds, $t(6770) = 2.11$, $p = .035$. Males (verbal IQ $M = 107.74$, $SD = 17.44$; Sky Search $M = 8.82$, $SD = 2.50$; Dual Attention $M = 8.04$, $SD = 4.10$; Opposite Worlds $M = 18.30$, $SD = 1.65$) scored higher than females on all tasks (verbal IQ $M = 106.67$, $SD = 16.01$; Sky

Search $M = 8.68$, $SD = 2.26$; Dual Attention $M = 7.11$, $SD = 3.39$; Opposite Worlds $M = 18.22$, $SD = 1.70$). We included weekly family income, parental education, child sex, and verbal IQ as covariates in predictive models to account for their associations with nonrandom missing data and attrition.