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Cumulative social risk exposure, infant birthweight, and cognitive delay in infancy

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Abstract

Objective—To determine: (1) the effect of exposure to multiple social risks on cognitive delay at nine-months of age; and (2) whether obstetric factors mediate the relationship between cumulative social risk and cognitive delay.

Methods: Data were from 8,950 mother-child dyads participating in the first wave of the *Early Childhood Longitudinal Study, Birth Cohort*. Cognitive delay was defined as falling in the lowest 10% of mental scale scores from the *Bayley Short Form-Research Edition*. Five social risk factors were combined and categorized into a "social risk index." Staged multivariable logistic regressions were used to investigate whether obstetric factors mediated the impact of social risk on the odds of cognitive delay.

Results—Infants with cognitive delay were more likely to live with social risks than infants without cognitive delay. The percentage of infants with cognitive delay increased with the number of social risks. In adjusted analyses, exposure to multiple social risk factors was associated with higher odds of cognitive delay at nine-months of age (adjusted odds ratio 2.11; 95% confidence interval: 1.18-3.78 for four or more risks versus no risks). Accounting for birthweight attenuated this relationship (p<0.001).

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Conclusions—This is the first population-based study to investigate the independent and cumulative effects of social risk factors on cognitive delay in infancy. Findings show a significant cumulative relationship between exposure to social risk and cognitive delay, which is partly mediated by birthweight. Programs that address the social context of US infants are needed to improve their developmental trajectories.

What's New—National data show a significant cumulative relationship between exposure to social risks and cognitive delay in infancy, which is mediated by infant birthweight. Programs that address the social context of US infants are needed to improve their developmental trajectories.

Keywords

Child development; infancy; social environment; birthweight; lifecourse

INTRODUCTION

Social risk factors including poverty,[1] single-parent families,[2] and low parental education[3] are consistently among the strongest correlates of poor cognitive and developmental outcomes among children. However, despite the well-established relationship between social risk factors and children's cognitive outcomes and academic achievement,[4-8] comparatively little work has investigated the relationship between social risk factors and cognitive delay in infancy. This represents a vital gap in the literature as infancy marks a critical period with significant implications for children's cognitive development. In fact, the prevalence of cognitive delay has been shown to be highly dynamic in early childhood, whereby a substantial proportion of infants with cognitive delays at nine months of age display normal cognitive development by 24 months (Cheng ER et al, under review). Nevertheless, for some children, early cognitive delays persist over time[4] threatening their school-readiness[9] and long-term health and functioning.[10] Improving our understanding of the relationship between social risk factors and cognitive delay during this critical stage in the lifespan may therefore have substantial impacts on prevention, treatment, and rehabilitation strategies.

The relationship between social risk factors and early cognitive outcomes may be best explored from the lifecourse perspective, which conceptualizes child health as arising from cumulative effects of events and exposures at different life periods, as well as from intergenerational effects.[11-15] For example, social risk factors like race, poverty, and education are known to influence the risk of adverse obstetric outcomes,[16-20] and obstetric outcomes have been associated with cognitive delay at school-age.[21-23] A linked pathway may therefore exist among maternal social risk factors, adverse obstetric outcomes, and early child cognition; as such exposure to social risk factors increases women’s risk of having poor obstetric outcomes, and obstetric outcomes in turn contribute to early cognitive delay. A cumulative risk model would suggest that social risk factors influence early childhood cognition via an “additive effect,” in which there is an accumulation of risk caused by individual negative exposures.[15] However while several studies have demonstrated that poor child health and developmental outcomes are accelerated by exposure to multiple risks relative to singular risk exposure,[24-28] it is unknown whether social risk factors influence cognition in infancy via the same cumulative mechanism.

In this study, we investigated the relationship among social risks, including family demographic and socioeconomic factors, and cognitive delay among infants participating in the *Early Childhood Longitudinal Study, Birth Cohort* (ECLS-B). We examined the independent and cumulative effects of social risk factors on cognitive delay, and explored whether these relationships were mediated by obstetric factors. We hypothesized that social risks would be associated with early cognitive delay in a cumulative manner, but that this risk would be partially attributable to social disparities in obstetric outcomes. To our knowledge, this is the first nationally representative lifecourse study in the US to examine the cumulative influence of social risk factors on cognitive delay in infancy.

METHODS

Data Source

Data were drawn from the first wave of the ECLS-B, a nationally representative, population-based cohort study of nearly 11,000 US children from birth through kindergarten. A detailed discussion of the ECLS-B's data collection procedures and methodology is available elsewhere.[22] Briefly, the ECLS-B selected a nationally representative probability sample of the approximately four million children born in 2001, with oversampling of children from minority groups, twins, and children born at low and very low birthweights, based on registered births from the National Center for Health Statistics vital statistics system.[29] Children born to mothers under the age of 15 and those who were adopted or died before nine months of age were excluded from the sampling frame.[30] The first data collection wave occurred in 2001 when the children were approximately nine months old

Restricted data for this study were obtained by approval from the Institute for Education Sciences Data Security Office of the US Department of Education, National Center for Education Statistics (NCES). In accordance with NCES guidelines, all reported unweighted sample sizes are rounded to the nearest 50.[29] The University of Wisconsin-Madison Health Sciences Institutional Review Board considered this study exempt from review.

Participants were eligible for the current study if the biological mother was the main survey respondent and if the infant participated in the cognitive assessment (n=10,000); we excluded 250 infants with missing birth certificate information. The ECLS-B dataset included individual records for each child sampled as a twin; for these families, a random number generator was used to randomly select one child from each pair to remain in the sample, leaving 8,950 participants.

Measures

Cognitive Delay—Cognitive delay was determined using the mental scale of the *Bayley Short Form Research Edition* (BSF-R).[29] The BSF-R comprised a subset of items from the revised *Bayley Scales of Infant Development* (BSID-II), a standardized screening instrument of developmental status designed for children ages birth to 42 months.[31] The NCES recorded age-normed mental scale scores for the BSF-R (mean=50, standard deviation=10); the age at administration for children who were born preterm was recorded as their chronological age minus the number of weeks preterm. We considered children falling within lowest 10th percentile of the age-normed BSF-R mental scale (i.e., those in the lowest 10% of ability relative to other children of the same age in our sample) to have a cognitive delay, similar to previous research.[32-34] We also tested alternative specifications of cognitive delay using the 5th and 15th percentile of the BSF-R as cutoffs and found consistent results with those using the 10th percentile delineation.

Social Risk Index—We examined five social risks assessed during the nine-month interview: family income below 100% of the Federal poverty level (FPL); single-parent household status; maternal black (non-Hispanic) race; maternal education less than high school; and three or more children other than the index child living in the household. These factors were selected on the basis of their individual relationships with child health in existing research.[35,36] Bivariate analyses revealed that each factor was significantly associated with cognitive delay (Table 1).

To address our hypothesis that these risks would be associated with cognitive delay in a cumulative manner, we generated a social risk index by giving infants one point for the presence of each social risk. This risk-index approach, developed by Sameroff et al,[37] has been used in numerous studies of children's social, behavioral, and health development.[24-28] Scores ranged from 0 to 5, with higher scores reflecting more social risk (Cronbach's α=0.53).

Covariates—Birth certificates provided obstetric factors: (1) birthweight in grams (<1,500 [very low]; 1,500-2,499 [low]; 2,500-3,999 [normal]; and ≤4,000 [high]); (2) pregnancy complications (flag for: anemia, diabetes, (oligo) hydramnios, lung disease, hypertension during pregnancy, cardiac disease, eclampsia, hemoglobinopathy, incompetent cervix, Rh sensitization, uterine bleeding, renal disease, genital herpes, or other medical risk factors); (3) labor and/or delivery complications (flag for: febrile, moderate/heavy meconium, premature rupture of membranes, abruptio placenta, placenta previa, excessive bleeding, seizures during labor, precipitous or prolonged labor, dysfunctional labor, breech/ malpresentation, cephalopelvic disproportion, cord prolapse, anesthetic complications, fetal distress, the use of forceps or vacuum, or any other complication); and (4) congenital anomalies (e.g., Down's syndrome, spina bifida).

Additional covariates included the infant's gender and plurality status (e.g., singleton versus twin or triplet).

Analytic Approach

Analyses were conducted using SAS v9.2. Means and percentages were obtained to describe the sample characteristics using appropriate weights to account for ECLS-B's complex sampling design. Chi-square tests were used to determine the statistical significance of differences in social risk and obstetric factors by cognitive delay status.

Multivariable logistic regression was used to examine associations between each social risk factor and levels of cumulative social risk with cognitive delay. Staged multivariable logistic regression analyses were used to determine whether the association between categorical social risk (with 0 risks as the reference category) and cognitive delay was mediated by obstetric factors: first pregnancy complications, labor/delivery complications and congenital anomalies; then infant birthweight. The relationship between social risk and cognitive delay was determined to be mediated by obstetric factors if the regression coefficient for cognitive delay was attenuated. The statistical significance of any mediating effects was formally tested using Imai et al.'s *mediation* package in R.[38] All models controlled for infant gender and plurality.

Complementary analyses evaluated the relationship between social risk and continuous BSF-R scores using weighted linear regression analysis, adjusted first for infant gender and plurality and then obstetric factors. These analyses sought to further evaluate the cumulative effect of social risk and to quantify the magnitude of the change in BSR-F scores associated with increasing levels of social risk.

RESULTS

Sample Description

Sample characteristics are presented in Table 1. In this national sample, a substantial proportion of infants lived with social risks: approximately 24% lived in poverty, nearly 20% lived with mothers who had less than a high school education, 14.7% had black (non-Hispanic) mothers, and 19.8% lived in single-parent households. 24.3% of infants had two or more social risk factors and 3.1% had four or more.

Independent Associations of Social Risk Factors with Cognitive Delay

In logistic regression analyses adjusting for infant gender and plurality, every social risk factor independently increased the odds of cognitive delay at nine months of age (Table 2, Model 1). However, only single-parent household status (adjusted odds ratio [AOR] 1.34; 95% confidence interval [CI]: 1.05-1.71) and having three or more siblings (AOR 1.43; 95% CI: 1.05-1.96) had significant independent associations with cognitive delay controlling for all of these risks simultaneously (Table 2, Model 2).

Social Risk Index

There was a significant increasing association of social risk on cognitive delay (Figure 1), such that the percentage of infants with indications of cognitive delay increased with the number of social risks (p-value for trend ≤0.001). 5.4% of infants with no social risks had indications of cognitive delay compared to nearly 12.0% of infants with four or more risks.

In adjusted analyses, the association between social risk and the odds of cognitive delay increased with the number of social risk factors (Table 3, Model 1 and Figure 2), such that the relative odds of cognitive delay was over two-fold higher for infants living with four or more social risk than infants with no risks (AOR 2.11; 95% CI 1.18-3.78). Infants with one risk factor were not statistically different from those with zero risk factors. These associations were largely unaffected by adjustment for pregnancy complications, labor/delivery complications, and congenital anomalies (Table 3, Model 2). However, when infant birthweight was added to the model (Model 3), the social risk coefficients attenuated for infants living with three (AOR from 1.62 to 1.46; 95% CI: 1.04-2.04) and four or more risks

(AOR from 2.13 to 1.85; 95% CI: 1.04-3.32. The inclusion of infant birthweight completely mediated the association between social risk and cognitive delay for children living with two risks (AOR from 1.52 to 1.41; 95% CI: 0.99-1.98). The overall mediation effect of social risk on cognitive delay by birthweight was statistically significant, as verified by Imai's approach ($p_{mediation} \leq 0.001$).

Complementary analyses confirmed an increasing effect of social risk on mean infant cognition scores, such that each social risk exerted an additional negative effect on continuous (standardized) BSF-R scores (Beta coefficient= -0.90, $p \leq 0.001$; adjusted for gender and plurality). When birthweight was added to the model, the effect of social risk attenuated (B=-0.62; $p \leq 0.001$). This mediation effect was highly significant, with 42% of the association between social risk and BSF-R scores acting via mediation by birthweight ($p_{mediation} \leq 0.001$; data not shown).

DISCUSSION

This nationally representative, population-based study investigated the relationship between social risk factors and cognitive delay in infancy. Our results contribute three important findings to the literature. First, we found significant associations between exposure to social risk factors and cognitive delay at nine months of age, extending to infancy previous studies illustrating the strong relationship between social disadvantage and children's cognitive and intellectual achievement.[1,7,39-41] Second, in addition to demonstrating independent associations of each social risk factor, we observed a significant cumulative relationship between exposure to these social risks and the likelihood of cognitive delay, informing the mechanism of the effects of social risk on early cognitive delay. Third, the relationship between exposure to multiple social risks and cognitive delay was mediated by birthweight, elucidating an important target for preventive interventions. These findings are discussed with respect to their implications for pediatric practice, public health policy, and future research.

Our findings suggest that disparities in the prevalence of early cognitive delay may reflect a cumulative effect of multiple social risks. In our national sample, social risks, including living in poverty, in a single-parent household, with three or more siblings, or having a mother of black (non-Hispanic) race or with less than a high school education, was associated with cognitive delay at nine months of age. However, when risks were combined into a single social risk index, large cumulative associations were observed, with over two-fold higher odds of cognitive delay among infants with four or more risk factors compared to those with no risks. This overall pattern is consistent with previous research on the importance of the total number of risk factors for child developmental outcomes.[24-28] For example, Sameroff et al found that the cumulative effect of ten risk factors had a stronger impact on cognitive functioning and social- emotional development of four-year-old children than the effect of any individual risk factor.[24,37,42] Larson et al recently reported that eight social risk factors cumulatively impacted children's global, dental, socio-emotional, and physical health.[25]

Our mediation analysis suggests that the association between exposure to multiple social risks and early cognitive delay may be partly attributable the increased likelihood of low birthweight among those living with social risk. In the empirical literature, African American race, social disadvantage, and poverty are consistently associated with low birthweight and prematurity;[16-20] by school age, these social risk factors are also associated with lower levels of IQ, academic achievement, and school performance.[5,7,8] The exact timing of these effects is not well understood, although it has been proposed that social disparities in child cognition *develop* during early childhood through pathways like inadequate nutrition, poor access to healthcare, or low quality child care.[33] Using a lifecourse approach,[11,13,15] we suggest that the relationship between social risk factors and cognitive delay in infancy may additionally operate through a linked health trajectory of children and their mothers, whereby social risk contributes to disparities in birthweight, which, in turn, contribute to differences in the prevalence of cognitive delay in infancy. However, this finding should be viewed as only descriptive and exploratory in nature. Our cross-sectional data could not establish temporality; moreover, the relationship between social risk and cognitive delay was not mediated by pregnancy or labor/delivery complications, which are indicative of maternal health status. Further, our data did not account for the dynamics of the constituents of the social environment, such as the length of exposure to or number of episodes of poverty both for infants and mothers over their lifetimes. For example, the number of years of family poverty has been shown to be more strongly associated with adverse childhood outcomes than the family's current economic status.[1] Additional research that links maternal lifetime exposure to social risk with her children's health outcomes over time is needed to test directionality and clarify the intergenerational relationships among the constructs we considered in this study.

To our knowledge, only one other nationally representative study has investigated risk factors for cognitive delay in infancy. Consistent with our findings, Hillemeier et al[33] demonstrated that obstetric factors, including low birthweight, were significantly associated with cognitive delay at nine months of age; however, unlike our study, socioeconomic status was not. Hillemeier's study, however, examined a composite socioeconomic status measure as an independent variable, which did not account for the cumulative effect of multiple clusters of social risk factors to which children are often exposed. Social risks likely impact early cognition through dynamic, multifaceted interactions with environmental, psychological, and biologic systems over time. Additional research is needed to elucidate the influence of these multiple dimensions to understand the pathways and patterns by which social disadvantage impacts cognition in infancy. Understanding these mechanisms will facilitate the design of effective interventions and improve our understanding of periods when such interventions would have the greatest impact on improving outcomes.

Several limitations to this study should be noted. First, even though our social risk indicator encompassed several factors, it was not comprehensive of all social risks associated with child health. Further, our use of an index to assess social risk has some limitations, including the assignment of equal weight to each factor.[43] Future research could consider alternate approaches to social risk assessment (e.g., factor analysis)[43] that may more accurately capture the relative importance of individual risks. Such work could also identify specific combinations of risk factors most pertinent to outcomes. Second, the present study is cross-sectional, limiting the interpretation of our mediation analyses and the causal inference of our results. Similarly, we did not examine whether cumulative social risk relates to health outcomes over time. We are following children over multiple ECLS-B waves to examine such relationships. Third, there may be overlap between our definition of cognitive delay, based on the cognitive component of the BSF-R, and other developmental delays to executive or sensorimotor functions. Fourth, due to small numbers, we operationalized obstetric outcomes as dichotomous variables and therefore did not determine independent or cumulative impacts of such events. Findings may also be subject to survival bias, as the ECLS-B did not include infants who died within nine months of their birth. Finally, birth certificate data may under or incorrectly report some information.[44]

This study also has several notable strengths, including the use of a large, national database which allowed us to examine multiple social and health factors in one model. Further, the high quality measurements provided in the ECLS-B allowed us to objectively assess cognitive delay, avoiding misclassification error and biases associated with parental report of childhood conditions.[45,46] Finally, evaluating these associations from a theoretical perspective enhanced our understanding of the mechanisms and pathways by which social risk factors contribute to early cognitive delay.

There are several implications of our findings. Clinically, our results suggest that approaches to identify young children with or at risk for cognitive delay may be most effective if they incorporate multiple biologic and social risk criteria. While these factors are not measures of cognitive delay themselves, they are associated with higher rates of cognitive delay and thus can be regarded as important risk factors. Pediatricians should be aware social risk factors may have additive effects on cognitive delay in infancy and focus identification and referral efforts to those living with two or more risk factors. In the absence of standard well-child practices to assess social risks in children, our findings support recommendations[25] to develop a social risk assessment screening tool that pediatricians can administer early in children's lives.

Our findings also suggest that efforts need to be made to improve birth outcomes as they are clearly tied to early cognitive delay. The linkages between women's obstetric health and infant cognition observed in this study acknowledge the importance of intergenerational factors for health development[47] and advocate for increased attention to more upstream factors (e.g., women's health) in order to combat downstream outcomes such as early childhood cognition. Future work should test whether preconceptional and prenatal interventions improve cognitive outcomes for children, bearing in mind that multifaceted interventions that address social and biologic risks over prolonged periods may have the greatest chance at success.

The high proportion of infants in our sample living with least one social risk (nearly 50%) and the cumulative association between these social risk factors and cognitive delay underscores the need for programs and policies to address the social context of US infants in an effort to improve their developmental trajectories. The independent associations between living in a single-parent household or with three or more siblings and cognitive delay provide some guidance regarding potential starting points for intervention. While we did not investigate the mechanisms of these associations, these variables may reflect constrained economic and other family resources.[45] However, our findings also suggest that the accumulated burden of risk, rather than any particular risk factor, is most important. Therefore, interventions aimed at high-risk groups (i.e., infants with multiple risks and/or with these particular risk factors), may ultimately be necessary to improve outcomes.

Finally, this work supports state efforts to extend eligibility under Part C of IDEA beyond diagnoses of developmental delay to include biologic, social, and environmental risk criteria. Early intervention and other support systems not based on qualification due to developmental delay, including the newly-funded Maternal, Infant Early Childhood Home Visiting (MIECHV) program, likely offer excellent opportunities and resources for infants and families living with multiple social risks associated with cognitive delays and other poor developmental outcomes. These new investments in maternal and child health may be critical to ensure specialized services are serving a broader community of children who need them.

CONCLUSION

In this nationally representative, population-based study, we noted associations between the accumulation of social risk factors and cognitive delay among infants, which was partly mediated by birthweight. Our findings support a lifecourse approach and indicate that programs to address the social context of US infants are needed to improve their developmental trajectories.

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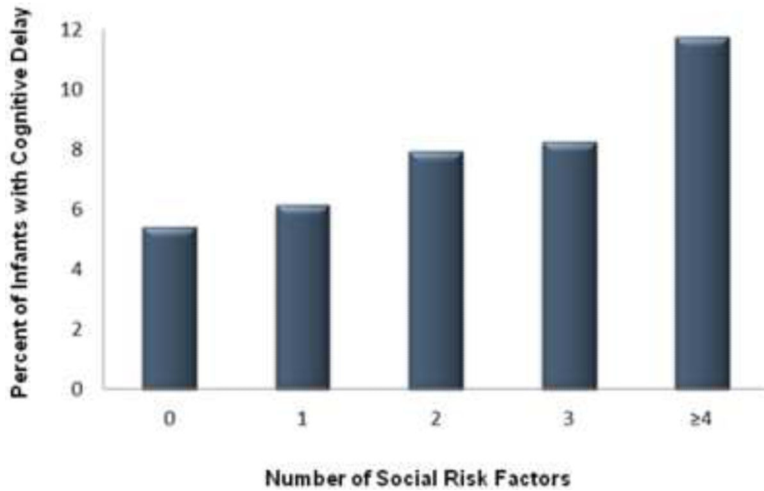

Figure 1. Percentage of nine month old infants with cognitive delay by number of social risk factors, national estimates from the 2001 ECLS-B

Figure 1 presents the unadjusted prevalence of cognitive delay at nine-months of age (y-axis), by the number social risk factors (x-axis). Social risk factors include: family income below 100% of the Federal poverty level, single-parent household status, maternal black (non-Hispanic) race, maternal education less than high school, and three or more children other than the index child living in the household. Prevalence estimates account for the complex sampling design of the ECLS-B.

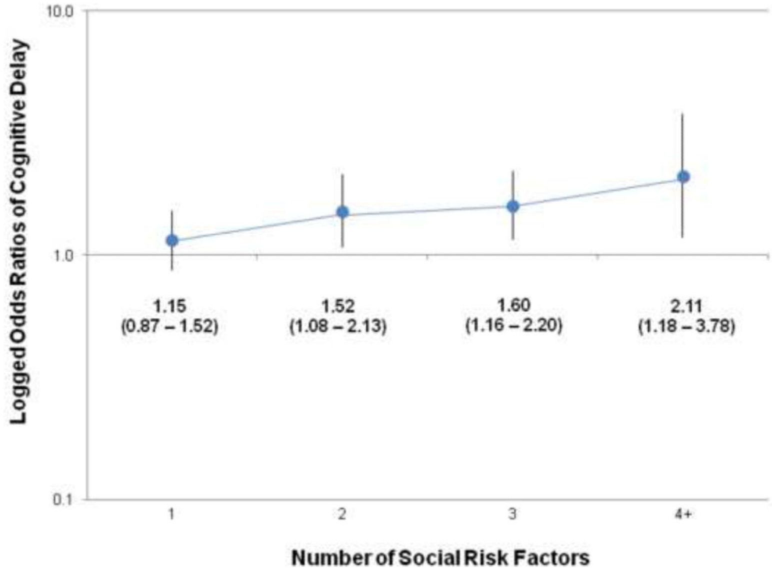

Figure 2. Adjusted odds ratios of cognitive delay at nine-months of age by exposure to social risk, national estimates from the 2001 ECLS-B

Figure 2 presents adjusted odds ratios and 95% confidence intervals for cognitive delay at nine-months of age associated with exposure to social risk, adjusted for child gender and plurality. The reference category for all estimates is zero risk factors. Social risk factors include: family income below 100% of the Federal poverty level, single-parent household status, maternal black (non-Hispanic) race, maternal education less than high school, and three or more children other than the index child living in the household. These odds ratios were generated accounting for the complex sampling design of the ECLS-B.

Table 1

Characteristics of nine-month-old infants with and without cognitive delay and their families, national estimates from the ECLS-B nine month data wave

		Cognitive Delay at Nine Months of Age		
	TOTAL	**Yes**	**No**	**p-value**
TOTAL (unweighted)	8,950	900	8,050	
Social Risk Factors	%	%	%	
Family Income ≤ 100% of the FPL				0.02
Yes	24.2	30.8	23.8	
No	75.8	69.2	76.2	
Maternal Race/ethnicity				0.16
White (non-Hispanic)	60.1	56.1	60.4	***
Black (non-Hispanic)	14.7	17.4	14.5	*
Other (non-Hispanic)	4.2	4.7	4.1	
Hispanic	21.0	21.7	21.0	
Maternal Education				0.19
No or Some High School	19.7	23.3	19.5	*
High School Graduate	28.6	30.5	28.5	
Some College	27.6	26.0	27.7	
Bachelor's Degree or Higher	24.1	20.2	24.4	
Family Structure				0.003
Single-Parent Household	19.8	26.0	19.3	
Two-Parent Household	80.2	74.0	80.7	
Number of Other Children in the Household				≤.0001
0	41.1	30.3	41.8	*
1	33.7	38.9	33.4	**
2	16.6	18.4	16.5	**
3+	8.6	12.4	8.3	**
Number of Social Risk Factors				0.02
0	52.0	44.5	52.6	**
1	23.7	23.0	23.7	
2	13.4	16.8	13.1	**
3	7.8	10.2	7.7	***
4+	3.1	5.5	2.9	***
Obstetric Factors				
Pregnancy Complications				0.01
Yes	27.7	35.5	27.3	
No	73.3	66.5	72.7	
Labor and Delivery Complications				0.002

		Cognitive Delay at Nine Months of Age		
	TOTAL	**Yes**	**No**	**p-value**
Yes	35.8	44.1	35.2	
No	64.2	55.9	64.8	
Congenital Anomaly				0.05
Yes	1.2	2.9	1.1	
No	98.8	97.1	98.9	
Birthweight Status				≤.0001
Very Low	1.2	5.7	0.9	***
Low	5.6	15.4	4.9	***
Normal	83.8	74.7	84.4	***
High	9.5	4.2	9.8	***
Infant Characteristics				
Gender				0.02
Male	**51.0**	**55.7**	50.**7**	
Female	49.0	44.3	49.3	
Plurality				≤.0001
Singleton Birth	98.3	95.3	98.5	
Twin or Triplet	1.7	4.7	1.5	

Notes. Weighted estimates. Percentages may not sum to 100 due to rounding. Unweighted sample sizes were rounded to the nearest 50 in accordance with NCES guidelines. Data were extracted from the ECLS-B 9-month data collection wave.

Abbreviations: *OR,* Odds Ratio; *CI,* Confidence Interval; *FPL,* Federal Poverty Level.

[*] p<0.05

[**] p<0.01

[***] p<0.0001

Table 2

Adjusted logistic regression model of cognitive delay at nine months of age, national estimates from the ECLS-B

	Cognitive Delay at Nine-Months of Age			
	Model 1		Model 2	
	AOR	95% CI	AOR	95% CI
Social Risk Factor				
Single-Parent Household	1.47	1.18 – 1.82	1.34	1.05 – 1.71
Family Income ≤ 100% of the FPL	1.45	1.16 – 1.82	1.22	0.94 – 1.59
Maternal Black (non-Hispanic) Race	1.30	1.02 – 1.66	1.00	0.76 – 1.31
Maternal Education Less than High School	1.49	1.06 – 2.10	1.09	0.81 – 1.47
Three or More Other Children in the Household	1.52	1.09 – 2.10	1.43	1.05 – 1.96

Notes. Weighted analyses. Data were extracted from the ECLS-B 9-month data wave. All models control for infant gender and plurality. Model 1 examines each social risk factor separately; Model 2 mutually adjusts for the social risk factors.

Abbreviations: AOR, Adjusted Odds Ratio; CI, Confidence Interval; FPL, Federal Poverty Level

Table 3

Staged multivariable logistic regression of the mediating role of obstetric factors on the relationship between categorical social risk and the odds of cognitive delay at nine months of age, national estimates from the ECLS-B

	Model 1		Model 2		Model 3	
	AOR	95% CI	AOR	95% CI	AOR	95% CI
Number of Social Risk Factors[a]						
0	1.00	Reference	1.00	Reference	1.00	Reference
1	1.15	0.87 – 1.52	1.16	0.88 – 1.53	1.11[¥]	0.83 – 1.47
2	1.52	1.08 – 2.13	1.52	1.09 – 2.13	1.41[¥]	0.99 – 1.98
3	1.60	1.16 – 2.20	1.62	1.17 – 2.24	1.46[¥]	1.04 – 2.04
4+	2.11	1.18 – 3.78	2.13	1.20 – 3.80	1.85[¥]	1.04 – 3.32
Pregnancy Complications						
Any			1.20	0.98 – 1.45	1.08	0.88 – 1.33
None			1.00	Reference	1.00	Reference
Labor or Delivery Complications						
Any			1.43	1.14 – 1.78	1.33	1.06 – 1.67
None			1.00	Reference	1.00	Reference
Congenital Anomaly						
Any			1.98	0.95 – 4.15	1.98	0.95 – 4.15
None			1.00	Reference	1.00	Reference
Birthweight Status						
Very Low					5.69	4.57 – 7.09
Low					3.10	2.55 – 3.77
Normal					1.00	Reference
High					0.47	0.30 – 0.74

Notes. Weighted analyses. Data were extracted from the ECLS-B 9-month data wave. All models control for infant gender and plurality.

Abbreviations: AOR, Adjusted odds ratio; CI, Confidence interval.

[a]Social risk factors include: family income below 100% of the Federal poverty level, single-parent household status, maternal black (non-Hispanic) race, maternal education less than high school, and three or more children other than the index child living in the household.

[¥]Denotes statistical significance of the overall mediation effect at $p<.0001$. Mediational analyses were conducted using Imai et al.'s *mediation* package in R. Significance levels are based upon two-tailed hypothesis tests.